PREP XI RR

STUDY NOTES

MEDICINE

10
VIDEOS

211
PAGES

PREMIUM STUDY MATERIAL

PREP XI RR

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■ RR Reignite

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4. RHEUMATOLOGY

BIG QUESTION 1: What is Systemic Lupus Erythematosus (SLE), how is it diagnosed, and what are its key systemic manifestations?

Broad Answer:

- SLE is a chronic multisystem autoimmune disease where the immune system attacks healthy tissues, mediated by autoantibodies and immune complexes.
- It typically affects women of reproductive age and can involve skin (malar rash), joints (non-erosive polyarthritis), kidneys (lupus nephritis), CNS (neuropsychiatric symptoms), serosa (pleuritis, pericarditis), and hematological systems (AIHA, thrombocytopenia).
- The SLICC 2012 criteria and ACR/EULAR 2019 classification guide diagnosis, requiring ≥4 criteria (with at least 1 clinical and 1 immunological feature).
- Key antibodies include ANA, anti-dsDNA, anti-Smith, and antiphospholipid antibodies. Renal biopsy showing the full house effect confirms lupus nephritis.
- Photosensitive rashes, oral ulcers, and systemic features are hallmarks, and immunofluorescence studies help guide disease severity and treatment.

DETAILED QUESTIONS

PYQ: FMGE 2020,2021

Q1.1: What are the distinguishing features of a malar rash in SLE?



Answer:

- There is a characteristic butterfly rash with sparing of the nasolabial fold.
  - The severity of the rash will worsen on exposure to sunlight.

Q1.2: What autoimmune disease is associated with a malar rash?

Answer:

- Systemic Lupus Erythematosus (SLE)

Q1.3: What cardiac condition presents with a malar flush?

Answer:

- Mitral stenosis

Q1.4: A 35-year-old woman presents with a violaceous rash over her nose and cheeks, shortness of breath, and bilateral hilar lymphadenopathy. What is the most likely diagnosis and its key features?

Answer:

- The clinical presentation is consistent with lupus pernio, a cutaneous manifestation of sarcoidosis.
- Lupus pernio presents as a violaceous, indurated rash commonly affecting the nose, cheeks, ears, and lips, often associated with systemic involvement.
- A chest X-ray showing bilateral hilar lymphadenopathy is characteristic of sarcoidosis, which is marked by non-caseating granulomas on histology.

- It is important to differentiate from lupus vulgaris, a cutaneous tuberculosis form, which shows caseating granulomas.



Q1.5: A woman presents with shortness of breath while climbing stairs, a heliotrope rash on her eyelids, and symmetrical proximal muscle weakness. Muscle biopsy shows perifascicular atrophy, and CK-MM is elevated. What is the diagnosis, and what are its key features?

Answer:

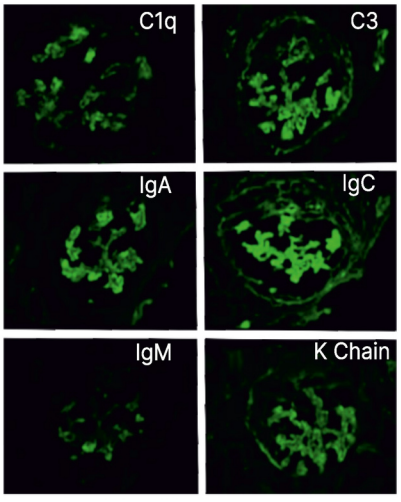


- Rash is present in the upper and lower eyelids
- On muscle biopsy: Perifascicular atrophy, myositis.
- CK-MM values are elevated
- Probable diagnosis: Dermatomyositis

Q1.6: What does the Full House pattern indicate, and which antibodies are involved?

Answer:

- The Full House pattern on immunofluorescence indicates **lupus nephritis**. It is characterized by the simultaneous deposition of:
  - IgG
  - IgA
  - IgM
  - C3
  - C1q
- This reflects widespread immune complex formation and activation of the classical complement pathway, a key mechanism in SLE-related tissue damage.





Full house effect

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Q1.7: Describe the 2012 SLICC criteria for diagnosis of SLE

Answer:

- ≥4 criteria out of 17 must be positive
- At least 1 clinical feature should be present
- At least 1 immunological feature should be present, e.g., Antinuclear Antibody positive, C3 levels, or C4 levels are lower because it is an immunologically mediated disease.
- Musculoskeletal presentation is the most common presentation for SLE, not dermatological presentation
- In 95% of cases, joint pain, frank synovitis, or intermittent polyarthritis, along with fever, can be present

Q1.8: Describe the 2019 ACR/EULAR classification

Answer:

- Entry point: Antinuclear Ab (ANA) should be positive >1:80 by the Immunofluorescent study.
- Clinical parameters
  - Musculoskeletal features: Joint pain
  - Dermatological feature: Malar rash
  - Haematological manifestation: Autoimmune hemolytic anemia (Antibodies causing damage here), so a coombs positive hemolytic anemia
- Cumulative score: ≥ 10 with one clinical feature

Q1.9: Explain the cutaneous manifestations of SLE

Answer:

- Skin manifestation: Acute and subacute lupus erythematosus
  - Malar rash
  - Photosensitivity: Rash tends to worsen in sunlight exposure (patient complains of skin burning in sunlight)
    → Hydroxychloroquine is given
  - Chronic subacute lupus erythematosus: Discoid lupus, subcutaneous fat involvement, i.e., panniculitis
  - Oral ulcers: Oral aphthous ulcers can be painless or painful

Q1.10: Describe the musculoskeletal manifestations of SLE

Answer:

- Synovitis: Involvement of ≥ 2 joints
  - Intermittent polyarthritis: Intermittent joint pain (Pain will be unilateral/ bilateral on alternate days).
  - It is non-erosive in a majority of cases (no deformities)
  - In rheumatoid arthritis, osteoarthritis is always erosive

Q1.11: Which is the commonest lung involvement in SLE?

Answer:

- Pleuritis

Q1.12: Which is the commonest cardiac involvement in SLE?

Answer:

- Pericarditis

Q1.13: How can the Auscultatory findings of pleural rub and pericardial friction rub be differentiated?

Answer:

- Both can be differentiated by breath-holding
  - o If sound disappears on holding breath: Pleural rub
  - o If the sound persists despite holding breath: Pericardial friction rub

Q1.14: What is the nature of vegetation in Libman-Sacks endocarditis?

Answer:

- The vegetation is sterile and non-infective.

Q1.15: Which part of the heart valve is primarily affected in Libman-Sacks endocarditis?

Answer:

- The inferior surface, especially the chordae tendineae.

Q1.16: What is the common valvular complication in Libman-Sacks endocarditis?

Answer:

- Mitral regurgitation.

Q1.17: What is the management of Libman-Sacks endocarditis if the patient is asymptomatic and has mild MR?

Answer:

- No treatment is required.

Q1.18: What is the approach if the patient becomes symptomatic?

Answer:

- Treatment is required, based on severity.

Q1.19: Are steroids used in the treatment of Libman-Sacks valvular lesion?

Answer:

- No, steroids are not given for valvular involvement.

Q1.20: What is the treatment for severe mitral regurgitation in Libman-Sacks endocarditis?

Answer:

- Valvuloplasty is performed if MR is severe.

Q1.21: What part of the valve is involved in rheumatic endocarditis?

Answer:

- The commissural ends, specifically the tip of the valve

BIG QUESTION 2: What are the systemic manifestations of SLE, and how are they evaluated and managed?

Broad Answer:

- SLE is a chronic autoimmune disease with multi-organ involvement. Oral ulcers can be painless or painful.
- Non-scarring alopecia is common.
- Neuropsychiatric manifestations include lupus cerebritis, seizures, psychosis, and myelitis due to antibody-mediated vascular damage; steroids are given cautiously.

- Hematological features include normocytic normochromic anemia (chronic disease), autoimmune hemolytic anemia (AIHA), lymphopenia, and thrombocytopenia.
- Renal involvement includes lupus nephritis, typically proteinuria, and RBC casts.
- Class IV diffuse proliferative lupus nephritis is the most severe and common subtype.
- Diagnosis uses urine protein: creatinine ratio, renal biopsy, and immunofluorescence showing full-house pattern.

DETAILED QUESTIONS

Q2.1: Which type of alopecia is seen in SLE?

Answer:

- Non-scarring alopecia

Q2.2: What is the neuropsychiatric manifestation of SLE?

Answer:

- Can cause lupus cerebritis; seizures can occur
- Steroids can be given
- Antibody-mediated damage causes cerebral edema
- Steroids are given with caution, as psychosis can develop in these patients as a psychiatric manifestation
- Depression
- SLE can affect blood vessels supplying the spinal cord, causing myelitis and cranial nerve involvement

Q2.3: Describe the hematological manifestations of Systemic Lupus Erythematosus (SLE).

Answer:

- SLE is a chronic disease
  - Anemia of chronic disease - Normocytic Normochromic anemia (Nonspecific)
    → Not a part of the diagnostic criteria
  - AIHA (Autoimmune hemolytic anemia)
    → Antibodies attack self-RBCs; there would be anti-erythrocyte antibodies.
    → Causes recurrent infections
  - Antibodies attack self-WBCs, leading to lymphopenia
  - Thrombocytopenia

Q2.4: Describe the renal involvement in Systemic Lupus Erythematosus (SLE).

Answer:

- Lupus nephritis, Proteinuria
- Urinary protein: Urinary creatinine ratio: >0.5
- RBC casts are present due to damage to the glomerulus
- Kidney biopsy (To gauge the severity of disease): Wire loop lesions
  - Histopathological subtypes: 6 classes of lupus nephritis by ISN (International Society of Nephrology)
  - Class 4: Diffuse lupus nephritis - Most common and most severe
- Immunofluorescence staining (IF)
  - The full house effect is seen

Q2.5: Which antibodies are involved in the Immunological Features of SLICC 2012 Criteria? PYQ: NEET PG 2022

Answer:

- Antinuclear antibody (ANA) should be positive
  - ANA > Reference negative value
- Anti-ds DNA: It helps chart out the progression of the disease
  - As the disease becomes more severe, anti-dsDNA ds DNA > 2 times the reference value
- Anti-Smith Ab - most specific
- Anti-phospholipid Ab - This antibody can cause uterine artery or venous thrombosis

Q2.6: What is the mechanism of recurrent abortions in patients with anti-phospholipid syndrome?

Answer:

- Anti-phospholipid Ab - This antibody can cause uterine artery or venous thrombosis
- It can trigger recurrent abortions
- ELISA test: Done for Anti Cardiolipin Ab, anti β2 glycolipid
  - These two antibodies will trigger the intrinsic system of clotting to go into automatic mode
  - In these patients, there is no trauma. Without trauma, the intrinsic system of clotting is activated because of these antibodies
  - Therefore, clots can develop in the uterine circulation which can cause recurrent abortion

Q2.7: Describe the rapid test for syphilis

Answer:

- False positive RPR (Rapid Plasma Reagin) - it is a rapid test for syphilis
  - The antigen used in this kit cross-reacts with these antibodies
  - Recurrent abortions can occur

Q2.8: Which complement components are reduced in active SLE?

Answer:

- **C3 and C4** are decreased due to classical complement pathway activation.

Q2.9: Which diagnostic test confirms Autoimmune Hemolytic Anemia in SLE?

Answer:

- Positive Coombs' test

Q2.10: What are the acceptable combinations of features to diagnose SLE?

Answer:

- For diagnosis of SLE: Total required is ≥ 4 features out of 17
  - 3 clinical features and 1 immunological Feature

    Or
  - 1 clinical feature and 3 immunological features

Q2.11: Specify Entry criteria and Additive ACR/EULAR 2019 criteria

Answer:

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Entry criterion

↓

Antinuclear antibodies (ANA) at a titer of → 1:80 on Hep-2 cells or an equivalent positive test

↓

If absent, do not classify as SLE. If present, apply additive criteria.

↓

Additive criteria

- Do not count a criterion if there is a more likely explanation than SLE.
- The occurrence of a criterion on at least one occasion is sufficient
- SLE classification requires at least one clinical criterion and > 10 points.
- Criteria need not occur simultaneously.
- Within each domain, only the highest weighted criterion is counted toward the total scores.

Q2.12: Describe ACR/EULAR: 2019 criteria for SLE.

Answer:

Clinical domains and criteria	Weight	Immunology domains and criteria	Weight
Constitutional		**Antiphospholipid antibodies**	
Fever	2	Anti-cardiolipin antibodies Or Anti-BETA 2GP1 antibodies, OR Lupus anticoagulant	2
Hematologic			
Leukopenia	3	**Complement proteins**	
Thrombocytopenia	4	Low C3 OR	3
Autoimmune hemolysis	4	low C4 Low C3 and low C4	4
Neuropsychiatric (Anti - Ribosomal - P antibody)		**SLE-specific antibodies**	
Delirium			
Psychosis	2	Anti-dsDNA	6
Seizure	3	antibody*DR	
	5	Anti-smith antibody	
Mucocutaneous			
Non-scarring alopecia	2		
Oral ulcers	2		
Subacute cutaneous OR discoid lupus	4		
Acute cutaneous lupus (photosensitive malar rash)	6		
Serosal			
Pleural or pericardial effusion	5		
Acute pericarditis	6		
Musculoskeletal			
Joint involvement - most common (Intermittent polyarthritis)	6		
Renal			
Proteinuria > 0.5g/24 h	4		
Renal biopsy Class II or V Lupus nephritis	8		
Renal biopsy Class III or IV Lupus nephritis	10		

Total score:

↓

Classify as systemic Lupus Erythematosus with a score of 10 or more if the entry criterion is fulfilled.

Q2.13: What can be the cause of death in SLE if not diagnosed?

Answer:

- Cause of death: Can be lupus nephritis if not diagnosed

Q2.14: What are the diagnostic prerequisites for systemic lupus erythematosus (SLE)?

Answer:

- 3 prerequisites for diagnosis-
  - ANA >1/80 by IF
  - At least 1 clinical feature should be present
  - At least 1 immunological/ laboratory feature should be present

Q2.15: Why is lupus nephritis considered a major cause of mortality in systemic lupus erythematosus (SLE), and what are the current treatment strategies?

Answer:

- Lupus nephritis: High chances of mortality.
  - Patient progresses to end-stage renal disease, whether the patient requires hemodialysis every other day, i.e., 3 times/week, or kidney transplantation
  - With hemodialysis, the chances of cardiovascular mortality increase
  - Kidney transplantation is recommended
  - With advancement, the prognosis is improving

BIG QUESTION 3: What are the key autoantibodies associated with Systemic Lupus Erythematosus (SLE), and what clinical manifestations or disorders are they linked to?

Broad Answer: PYQ: INICET 2021

Antibody	SLE
ANA (highly sensitive)	Best screening test, Immunofluorescence report > 1:80
Anti-ds DNA	Correlate with disease activity (nephritis)
Anti-Smith Ab	Most specific
Anti C1q Antibody (part of complement system)	63% of cases of lupus nephritis Determines severity of disease
Anti Ribo nucleoprotein antibody	Nonspecific, Mixed Connective Tissue Disorder
Anti-Ro Antibody (Anti SS-AAntibody)	Predispose to photosensitivity, causes congenital heart block in fetus
Anti La/SS-B	Nonspecific for SLE
Anti-Histone	Drug-induced lupus erythematosus
Anti-phospholipid	ELISA cardiolipin, ELISA β_2 glycoprotein, Dilute Russel Viper Venom Time Test
Anti-erythrocyte antibody	AIHA
Anti-platelet	Petechiae, thrombocytopenia
Anti-neuronal antibody/anti-glutamate receptor 2 antibody	Lupus cerebritis, seizures
Anti-Ribosomal-P	Depression, psychosis

Q3.1: What is the diagnostic utility of anti-dsDNA in SLE?

Answer:

- Correlates with disease activity, i.e., if the disease is severe enough and has contributed to the development of nephritis or severe vasculitis, then the value of Anti-ds DNA will be higher.

Q3.2: How can the Severity of lupus nephritis in SLE be determined?

Answer:

- Kidney biopsy, i.e., Class IV (commonest and most severe), there are a total of 6 classes, but it is invasive
- Anti-C1q antibody titres will tell the progression and severity of disease in comparison to previous years

Q3.3: Discuss the clinical implications of Anti-Ro antibody in SLE and other autoimmune disorders.

Answer:

- Responsible for photosensitivity
- Anti-Ro Ab can also be seen in SICCA/Sjogren's syndrome
- SICCA/ Sjogren's syndrome: A Condition with dry eyes, dry mouth, and dry vagina
- Photosensitivity should not be treated with steroids

Q3.4: Explain the role of Anti-Ro (SS-A) antibodies in fetal complications associated with SLE.

Answer:

- If a lady is having SLE, then this antibody can cross (Anti Ro Antibody/ Anti SS-A Antibody) over from the mother to the fetus via the placenta
- This antibody will damage the AV node in the child
- The conduction is hampered, and the baby born to this lady can have a complete heart block

Q3.5: Discuss the role of Anti-Histone antibodies in lupus. Which drugs are commonly implicated in drug-induced lupus?

Answer:

- Anti-histone is seen in drug-induced lupus erythematosus
  - Drugs: S.H.I.P
    - → S - Sulfonamide
    - → H - Hydralazine
    - → I - Isoniazid
    - → P- Procainamide

Q3.6: Which antibody correlates with lupus nephritis?

Answer:

- Two antibodies: Anti C1q> Anti ds DNA

Q3.7: Explain the clinical significance of Anti-phospholipid antibodies.

Answer:

- It predisposes to recurrent abortions
- Antigens present in the body of the patient are triggering the intrinsic system of clotting without any trauma
- Usually, there should be some trauma to the body to trigger the clot formation
- DRVVT (Dilute Russel Viper Venom Time Test) shows derangement with respect to autoactivation

- The intrinsic system of clotting is autoactivated, resulting in recurrent fetal loss
- An important cause of fetal loss can be syphilis, and there is a high probability for a syphilis test to be a false positive in this condition

Q3.8: What is the confirmatory test for AIHA?

Answer:

- Direct Coombs (Direct Antiglobulin) Test - detects antibodies or complement bound to RBCs.

Q3.9: What is the characteristic RBC shape in AIHA?

Answer:

- Spherocytes - small, dense RBCs without central pallor.

Q3.10: Which pediatric condition also shows spherocytes?

Answer:

- Hereditary spherocytosis.

Q3.11: How to differentiate AIHA from hereditary spherocytosis in the lab?

Answer:

- AIHA: Coombs test positive; Hereditary spherocytosis: Coombs test negative, osmotic fragility positive.

Q3.12: What stain is used in routine peripheral smear examination for AIHA?

Answer:

- Hematoxylin and eosin (H&E) or Romanowsky stains (Leishman/Giemsa).

Q3.13: What does the presence of anti-glutamate receptor 2 antibodies indicate?

Answer:

- Breach of the BBB with immune-mediated neuronal damage.

BIG QUESTION 4: Describe systemic sclerosis (scleroderma) with its key features, antibody profile, and complications.

Broad Answer:

- Systemic sclerosis is an autoimmune connective tissue disease marked by excessive collagen deposition, causing skin thickening, vasculopathy, and internal organ fibrosis.
- Diffuse type is associated with anti-Scl-70 and has widespread involvement; limited type presents as CREST syndrome with anti-centromere antibodies.
- The early sign is the Raynaud phenomenon (white→blue→red color change in digits).
- Skin changes include leather-like texture, salt-and-pepper pigmentation, and microstomia.
- Internal involvement includes esophageal dysmotility (↓LES tone), interstitial lung disease, and pulmonary artery hypertension (leading cause of death).
- Renal crisis with malignant hypertension is managed with ACE inhibitors.

DETAILED QUESTIONS

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Q4.1: Specify the antibodies of Scleroderma/Systemic Sclerosis

Answer:

- Anti-topoisomerase antibody present
- Anti-SCL-70 present

Q4.2: What are the clinical features of Scleroderma/Systemic sclerosis?

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Answer:

- Fibrosis around blood vessels in the tips of the fingers ,because of whichthe autoregulation of blood supply will be hampered

Q4.3: Explain the mechanism of the Raynaud phenomenon and its sequence of color change.

Answer:

- A lady in a cold environment, there could be a change in the color of the fingertips of this patient in the order:

Exaggerated vasoconstriction - White fingertips

↓

Due to vasoconstriction, there is less blood supply, and the concentration of deoxygenated Hb will rise - Cyanosis/ blue fingertips

↓

Exaggerated vasodilation

- Initially exaggerated vasoconstriction, then exaggerated vasodilation: Raynaud phenomenon



Q4.4: What are the early and characteristic skin changes in diffuse systemic sclerosis?

Answer:

- Early feature: Raynaud phenomenon
- Leather-like skin: Fibrosis under the skin
  - Salt and pepper appearance of skin

Q4.5: Explain esophageal involvement in systemic sclerosis

Answer:

- Microstomia: Fibrosis can also occur in the mouth of these patients, so there may be difficulty in opening the mouth
- Oesophageal dysmotility: Fibrosis can occur in the esophagus, causing dysphagia
  - In women, dysphagia is commonly seen with achalasia cardia
  - To differentiate achalasia cardia from scleroderma, LES tone is checked
  - Achalasia cardia: Loss of inhibitory control, so LES (Lower oesophageal sphincter) tone increases
- In scleroderma, Fibrosis in the LES, so the tone will be relatively less
  - As a result, reflux oesophagitis can occur

Q4.6: Describe the major pulmonary complications of systemic sclerosis

Answer:

- Interstitial lung disease: Fibrosis in the lungs
- Pulmonary artery hypertension: Fibrosis in the pulmonary artery
  - PAH: Loud P2
  - DLCO (Diffusion in lung for carbon monoxide) values lesser: Fibrosis in the wall of the blood vessel

Q4.7: Describe the Scleroderma crisis and its management.

Answer:

- Scleroderma crisis: There will be a progressive decrease in the size of the kidneys
  - GFR will be lower
  - RAAS will be activated, causing hypertension
  - Hypertensive crisis develops
- Management: ACE inhibitors (oral)

Q4.8: What is the leading cause of mortality in Scleroderma?

Answer:

- Leading cause of mortality in scleroderma: PAH

Q4.9: When is a heart-lung transplant preferred over a lung transplant in systemic sclerosis?

Answer:

- When **severe PAH (>60 mmHg)** is present

Q4.10: Describe the clinical features and management of CREST syndrome

Answer:

C	• Calcinosis
R	Raynaud's phenomenon: • DOC: Ca channel blockers like amlodipine • (Start with the lowest possible dose to prevent postural hypertension)
E	• Esophageal dysmotility - Dysphagia
S	Sclerodactyly • Presence of pitting scars in the volar aspect of fingers due to fibrosis under the skin
T	Telangiectasia- Dilated cutaneous blood vessels

Q4.11: Which antibody is most commonly associated with CREST syndrome?

Answer:

- **Anti-centromere antibody** – highly specific

BIG QUESTION 5: Describe the clinical features, investigation, and treatment of Granulomatosis with Polyangiitis.

Broad Answer: Granulomatosis with Polyangiitis is a necrotizing vasculitis with granuloma formation involving small-to-medium vessels.

- URT involvement: Recurrent sinusitis, nasal crusting, epistaxis.

LRT involvement: Hemoptysis, multiple small lung cavitations (unlike TB, which usually has a single upper-lobe cavity).

- Kidney involvement: Hematuria due to rapidly progressive glomerulonephritis.
- Differential: Goodpasture syndrome also causes hemoptysis + hematuria, but lacks URT symptoms and cavitations.
- Investigations: Positive c-ANCA (anti-proteinase 3); p-ANCA (anti-MPO) seen in microscopic polyangiitis.
- Treatment: Cyclophosphamide with corticosteroids.

Q5.1: Describe the respiratory involvement in Granulomatosis with angiitis / Wegener's granulomatosis

Answer:

- Upper respiratory tract involvement (URT): Recurrent sinusitis, epistaxis
- Lower respiratory tract involvement (LRT): Hemoptysis, Cavitations (due to damage to the blood supply of the lungs)
  - In tuberculosis, there is a cavity in the upper lobe of the lungs
  - In this condition, there could be multiple small cavities seen secondary to necrotizing vasculitis

Q5.2: Explain the renal involvement of Wegener's granulomatosis.

Answer:

- Kidney: Hematuria
  - Good pasture syndrome: There is a combination of hemoptysis and hematuria, damage to the basement membrane of the lung blood vessels, and kidney blood vessels
  - But in this condition, there is lung cavitation and URT involvement

Q5.3: Describe the workup of Wegener's granulomatosis.

Answer:

Work Up

- c-ANCA, this antibody is against the cytoplasmic antigen anti-proteinase 3
- p-ANCA: Seen with microscopic polyangiitis, the antibody is directed against the perinuclear antigen myeloperoxidase

Q5.4: What is the treatment for Wegener's granulomatosis?

Answer:

- Treatment: Cyclophosphamide

Q5.5: A 4-year-old child is having ECG findings of MI, strawberry tongue, and periungual desquamation. What is the diagnosis?

Answer:

- Diagnosis - Kawasaki disease:
  - Presence of coronary artery vasculitis

Q5.6: What is the management of Kawasaki disease?

Answer:

- IVIG: As vasculitis is very aggressive, Aspirin (low dose)

Q5.7: What are the features of Kawasaki disease?

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Answer:

- Initial presentation: Fever for > 5 days
- Physical examination shows unilateral cervical lymphadenopathy
- Strawberry tongue
- Other features seen:
  - Rash, periungual/ perianal desquamation, bulbar congestion (non-purulent)

Q5.8: What are the Echocardiography findings of Kawasaki disease?

Answer:

- Echocardiography: Demonstrates a coronary artery aneurysm > 1cm

Q5.9: When is the highest risk of MI in Kawasaki disease?

Answer:

- If Kawasaki disease is not identified in the child, it can lead to MI

Q5.10: Is Behçet's disease limited to small vessel vasculitis?

Answer:

- No, it can involve small, medium, and large vessels.

Q5.11: What is the classical triad of Behçet's disease?

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Answer:

- Features: Oro-oculo-genital syndrome

Q5.12: Explain the spectrum of blood vessel involvement in the disease that causes oropharyngeal, ocular, and genital manifestations.

Answer:

Recurrent Oral Ulceration	**Major, minor, or herpetiform observed by physician or patient recurrence at least thrice in any 12 months.**
PLUS TWO OF:	
Recurrent genital ulceration	Aphthous or scarring observed by physician or patient
Eye lesions	Anterior uveitis Posterior uveitis Cells in vitreous on slit lamp examination, OR Retinal vasculitis observed by ophthalmologist
Skin lesions	Erythema nodosum observed by physician or patient pseudofolliculitis or papulopustular lesions OR Acneform nodules observed by physician in post-adolescent patients not on corticosteroid treatment
Positive pathergy test	Read by the physician at 24-48 hours

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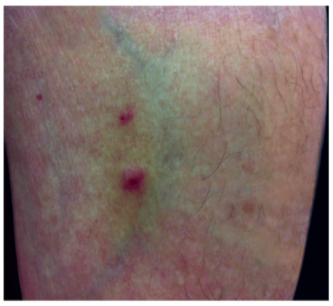
Q5.13: Describe the procedure of the Pathergy test. What is its diagnostic significance?

Answer:

Pathergy test:

- Take a sterile needle and make a puncture mark with a hypodermic cutting edge on the forearm of the patient
- In a healthy person, if there is some injury to the dermis and epidermis with the pointed edge of the needle, it will cause bleeding, clotting and then the mark disappears in 2-3 days
- In this patient, after 48hours, There is the formation of sa terile pustule (which does not happen in normal people)
- Most common diagnostic feature of Behcet's - Recurrent oral ulceration

Positive Pathergy test



Q5.14: In female patients with Behçet's disease, where are ulcers commonly seen?

Answer:

- Vulva.

Q5.15: In male patients with Behçet's disease, where are ulcers commonly seen?

Answer:

- Scrotum.

Q5.16: Are the genital ulcers of Behçet's disease the same as those seen in STDs like Haemophilus ducreyi or syphilis?

Answer:

- No, they are not penile ulcers; they are typically scrotal or vulvar ulcers.

Q5.17: Which vascular complication can develop in Behçet's disease?

Answer:

- Pulmonary artery aneurysm.

Q5.18: Is the pulmonary artery involved in Polyarteritis Nodosa (PAN)?

Answer:

- No — pulmonary artery involvement is absent in PAN.

Q5.19: What is a lethal complication of pulmonary artery aneurysm in Behçet's disease?

Answer:

- Rupture of the aneurysm.

Q5.20: How is ocular management done in Behçet's Disease?

Answer:

- Ocular management: Azathioprine

BIG QUESTION 6: Compare Sjögren's syndrome, Mixed Connective Tissue Disease (MCTD), and Rheumatoid Arthritis (RA) in terms of hallmark features, key investigations, and important complications.

Broad Answer:

- Sjögren's syndrome presents with dry eyes (positive Schirmer's test), dry mouth, and anti-Ro/SSA, anti-La/SSB antibodies; complications include corneal ulcers, dental caries, and increased lymphoma risk in secondary cases.
- MCTD combines features of SLE, scleroderma, polymyositis, and RA, often with Raynaud's phenomenon, proximal muscle weakness, synovitis, and positive anti-U1-RNP antibody.
- RA primarily affects small joints (PIP, MCP, wrist) bilaterally, causing deformities like Z-thumb, swan neck, and boutonnière, with anti-CCP positivity indicating erosive disease.
- RA management uses aggressive DMARD therapy (MTX ± hydroxychloroquine, sulfasalazine) to prevent functional disability.

DETAILED QUESTIONS

PYQ: FMGE 2021,2022
PYQ: NEET PG 2022

Q6.1: What are the features of Sjögren's syndrome?

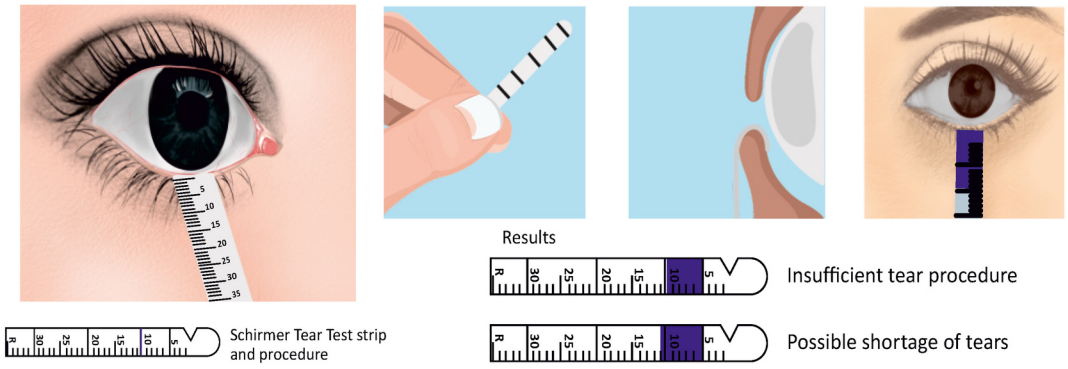
Answer:

- Features: Dry eyes, dry mouth

Q6.2: Discuss the role of Schirmer's test in evaluating lacrimal gland function.

Answer:

- Schirmer's test: Demonstrates the function of the 7 cranial nerves
  - It is done to pick up tearing in the eye
  - Tear production can be quantified
- Once tear production decreases, exposure to keratitis and corneal ulceration occurs.



Q6.3: Which antibody is seen in Sjogren syndrome?

Answer:

- Anti-Ro antibody (SS-A)

Q6.4: What happens to a baby born to a mother with Sjogren syndrome?

Answer:

- A baby born to a mother with Sjogren syndrome can have a complete heart block
  - A pacemaker (dual chamber) is required for the correction of complete heart block

Q6.5: Which antibody is highly specific for Sjögren syndrome?

Answer:

- Anti-La (SS-B) antibody

Q6.6: What is the IOC for Sjögren syndrome?

Answer:

- Salivary gland biopsy

Q6.7: What eye symptoms are characteristic of Sjögren syndrome?

Answer:

- Sand-like or grain-like sensation in the eye due to dryness.

Q6.8: Which dye is used to detect a corneal ulcer in Sjögren syndrome?

Answer:

- Fluorescein dye.

Q6.9: Most common oral complication of Sjögren syndrome?

Answer:

- Dental caries due to *Streptococcus mutans*.

Q6.10: Name a drug that stimulates salivary secretion in Sjögren syndrome.

Answer:

- Cevimeline.

Q6.11: What is the major malignancy risk in secondary Sjögren syndrome?

Answer:

- Lymphoma

Q6.12: What is Mixed Connective Tissue Disorder (M.C.T.D)?

Answer:

- Combo lesion of:
  - SLE
  - Scleroderma
  - Polymyositis
  - Rheumatoid arthritis

Q6.13: How does Mixed Connective Tissue Disorder (M.C.T.D) present?

Answer:

- A female patient having a malar rash, musculoskeletal complaints, synovitis, Raynaud's phenomenon, leather-like skin, difficulty in getting up from the toilet seat (due to proximal hip joint muscle involvement), morning stiffness, PIP, MCP, wrist joint red and inflamed. She is not able to use her toothbrush in the morning.

Q6.14: Which antibody is present in Mixed Connective Tissue Disorder (M.C.T.D)?

Answer:

- Anti U1 R.N.P (Ribonucleoprotein) antibody present

BIG QUESTION 7: Describe the hand deformities seen in rheumatoid arthritis and differentiate them from other conditions.

Broad Answer:

- Z-thumb (Hitchhiker thumb): Hyperextension at the interphalangeal joint with flexion at the metacarpophalangeal joint.
- Z-line deformity: Ulnar deviation at the wrist with radial deviation of fingers.
- Swan-neck deformity: Extension at the PIP and flexion at the DIP joint.
- Boutonniere deformity: Flexion at the PIP and extension at the DIP joint. Mallet finger: Flexion at DIP without PIP deformity; caused by extensor tendon injury, e.g., wicket-keeper injury – not a feature of RA.

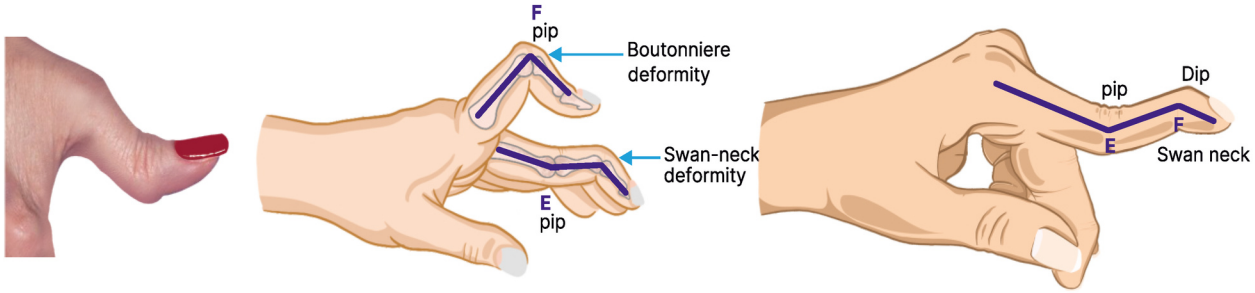
DETAILED QUESTIONS

PYQ: FMGE 2020,2021
PYQ: NEET PG 2021,2022

Q7.1: Describe the thumb deformity in Rheumatoid Arthritis.

Answer:

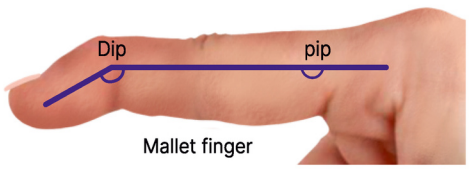
- Thumb deformity: Z - thumb deformity/ Hitch Hiker thumb deformity
  - Z - thumb deformity is different than Z-line deformity (ulnar deviation)
  - In ulnar deviation of the wrist joint, Fingers will move towards the radial side, and the wrist will have ulnar deviation
  - In the middle finger, at the DIP, there is a flexion present, but at PIP (proximal interphalangeal joint), there is an extension: Swan neck deformity



Q7.2: Describe Boutonniere Deformity.

Answer:

- In Boutonniere Deformity, there is Flexion at the PIP joint, but Extension at the DIP joint.
  - There is no deformation in the proximal interphalangeal joint, but there is flexion at the level of DIP: Mallet finger
  - It can be present in a wicket-keeper who's facing very fast balls, at the speed of 150miles/ hour; there could be an extension, and there could be an injury to the extensor tendon
  - There could be avulsion of the extensor tendon, which could result in Mallet finger (Not a component of rheumatoid arthritis)



Q7.3: What are the Clinical Features of Rheumatoid Arthritis?

Answers:

- This disease involves the small joints of the hand, like PIP, MCP, and wrist joint; in rheumatic, Large joints are involved, like the ankle joint, knee joint

YOURWISH

- Involvement can be bilateral
- When patients come to the hospital, there could be that only the wrist joint can have redness and inflammation; inflammation of
- PIP may not be noticed

Q7.4: Describe the investigations done for RA.

Answer:

- Monitor these patients for the next 6 weeks
- RA factor test should be done, but because RA factor is an IgM class antibody, it can be biologically false-positive in 5% of the population; even in sore throat, RA factor can turn out positive
- Anti C.C.P (cyclic citrulline peptide) antibody test is done
  - Once this test is concluded, positive- It is erosive arthritis

Q7.5: How is erosive arthritis managed?

Answer:

- NSAIDs are not recommended
- Triple therapy
  - In RA, it is disease-modifying drugs that should be used
  - Start with Methotrexate (MTX). If patients respond, it is good.
  - If not, do not let the patient deteriorate because the deformity contributes to difficulty in doing tasks. In Z thumb deformity, a person cannot do any pinching action. Eating with a spoon can be difficult; dressing is difficult
  - Then add Hydroxychloroquine to MTX and Sulfasalazine

Q7.6: Describe the clinical response monitoring protocol for Rheumatoid Arthritis (RA).

Answer:

- Protocol: First, start with MTX
  - If we get a good response, Morning stiffness is reduced along with subjective redness and inflammation of the hand joints of patients
  - In ankylosing spondylitis, as the person does an activity and as the day progresses, the stiffness will reduce
  - But in RA, the morning stiffness would be so much that the person complains of always getting late for the office; even brushing teeth would be difficult

Q7.7: What are rheumatoid nodules, and where are they typically found?

Answer:

- Rheumatoid nodules: The most common extra-articular manifestation of RA
  - These are extensors in distribution like over olecranon process or occiput of patient or tips of spine or dorsum of hand of patient

Q7.8: What is the eye manifestation in Rheumatoid Arthritis?

Answer:

- Eye manifestations: Keratoconjunctivitis Sicca

Q7.9: Why should RA patients avoid certain yoga poses and sports activities?

Answer:

- Patients are advised never to do yoga, which involves movement of the spine like neck rotation or doing a headstand because most of these patients would have weakness of the cervical spine
- If the person is doing yoga in RA, there are chances of atlantoaxial subluxation, and because of this, there can be compression of the spinal cord and quadriplegia
- The spinal cord is highly vulnerable in these patients, so these patients should not participate in sports-related activities

Q7.10: What is the cardiovascular manifestation of Rheumatoid Arthritis?

Answer:

- Rheumatoid nodules can not only be present in the skin. They can also be present in the heart
  - In rheumatic fever, there are Aschoff nodules in the heart
  - If rheumatoid nodules are present between the SA node and the AV node - Causing 1st-degree heart block
  - This rheumatoid nodule can cause damage to the myocardium
  - Studies have shown that accelerated atherosclerosis can be seen in patients with RA - One of the important reasons for death

Q7.11: What are the respiratory manifestations of Rheumatoid Arthritis?

Answer:

- Respiratory manifestation: Rheumatoid nodules can be present in the lungs
  - Pleuritis/ pleural effusion
  - Exudative pleural effusion: Lights criteria
    - → Protein in pleural fluid will be elevated; it could be > 0.5 times the serum protein value because it is exudate, and exudates are inflammatory
    - → Pleural fluid LDH > 2/3 of upper reference limit because of inflammation
    - → Light's criteria include pleural fluid protein and LDH values; it does not include pleural fluid sugar

Q7.12: Which type of anemia is seen in RA?

Answer:

- Anemia of chronic disease: Normocytic normochromic anemia& Microcytic hypochromic anemia

Q7.13: What is the Peripheral nervous system manifestation in RA?

Answer:

- Peripheral nervous system: multiple peripheral nerves are involved, which is called mono neuritis multiplex

BIG QUESTION 8: Explain the key clinical features, pathophysiology, and diagnosis of sarcoidosis.

Broad Answer:

- Sarcoidosis is a multisystem granulomatous disease characterized by non-caseating granulomas that can synthesize vitamin D, leading to hypercalcemia, which is steroid-responsive.
- Commonly, it presents with bilateral hilar lymphadenopathy on chest X-ray and parenchymal lesions on HRCT showing the galaxy sign; eggshell calcification may be seen later.
- It can affect multiple organs—lungs (dyspnea, airway compression), eyes (uveitis, retinitis), CNS (lymphocytic meningitis, diabetes insipidus), parotid gland (facial diplegia), and heart (heart block, restrictive cardiomyopathy).

- Diagnosis is confirmed by bronchoscopy-guided biopsy showing non-caseating granulomas, with supportive tests such as bronchoalveolar lavage showing CD4/CD8 ratio >3.5:1

DETAILED QUESTIONS

Q8.1: What is characteristic histopathology of Sarcoidosis?

Answer:

- It is characterized by the presence of non-caseating granulomas
- These non-caseating granulomas can synthesize Vitamin D leading to an increase in serum calcium levels

Q8.2: What are the main causes of hypercalcemia and how does sarcoidosis fit in?

Answer:

Cause of Hypercalcemia

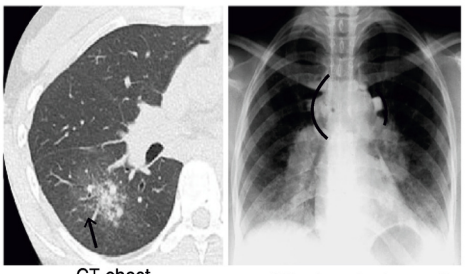
- Malignancy: Lung cancer i.e., squamous cell cancer producing PTHrP (Parathormone releasing peptide)
- Primary hyperparathyroidism (e.g. Parathyroid adenoma)
- Sarcoidosis (via increased Vitamin D synthesis in granulomas)
- In sarcoidosis, hypercalcemia improves with corticosteroid therapy

PYQ: FMGE 2022
PYQ: INICET 2023
PYQ: NEET PG 2022

Q8.3: What are the CT chest and Chest X-ray findings of Sarcoidosis?

Answer:

- In CT chest: Lesions are seen in lung parenchyma, and this is called galaxy sign
- Chest X-ray: Mediastinal mass present, i.e., hilar lymphadenopathy, and in later phases of the disease, there could be calcification around lymph node also
- Egg shell calcification is seen in silicosis, eggshell calcification also seen in sarcoidosis



Q8.4: Why is there shortness of breath in Sarcoidosis patients ?

Answer:

- The enlarged lymph nodes can press on the trachea, and that explains the shortness of breath in these patients

Q8.5: Describe the Clinical presentation of Sarcoidosis.

Answer:

- Clinical presentation: The female comes with complaints of a progressive increase in shortness of breath
  - On chest X-ray: Hilar lymphadenopathy and Eggshell calcification
  - Eggshell calcification appears later, so the majority of cases show hilar lymphadenopathy

Q8.6: Describe the CNS manifestations in Sarcoidosis.

Answer:

YOURWISH

- Lymphocytic meningitis (lymphocyte count increased), Non-Caseating granuloma can damage the posterior pituitary, which will hamper the release of ADH, causing nephrogenic diabetes insipidus

Q8.7: What are the eye manifestations in sarcoidosis?

Answer:

- Eye manifestations: Uveitis, Retinitis, Pars planitis

Q8.8: What is the parotid gland manifestation in Sarcoidosis?

Answer:

- Parotid gland enlargement - presses on VII CN causing bilateral VII palsy (seen with Guillain barre syndrome, Sarcoidosis- cause of facial diplegia)

Q8.9: What are the Hilar lymph node manifestations in sarcoidosis?

Answer:

- Hilar lymph node enlargement presses on airways, causing either partial collapse of the airway or segmental collapse of the lungs
  - Initially, these lymph nodes pressing on the airway - cause shortness of breath
  - Part of the lung involved: Mainly the upper lobe of the lung
  - Steroids are given for LN enlargement

Q8.10: Describe the cardiac involvement in Sarcoidosis.

Answer:

- Cardiac involvement - non-caseating granulomas in the conductive system of the heart - Heart block
  - These non-caseating granulomas present in the myocardium cause restrictive cardiomyopathy
  - Any cardiomyopathy can trigger the development of tachyarrhythmias
  - In ankylosing spondylosis, the cardiac lesion is Aortic Regurgitation

Q8.11: What heart sound finding may indicate pulmonary artery hypertension in sarcoidosis?

Answer:

- Loud P2

Q8.12: What is the investigation of choice for confirming sarcoidosis diagnosis?

Answer:

- Bronchoscopy-guided biopsy showing non-caseating granulomas.

Q8.13: What are the investigations done for Sarcoidosis?

Answer:

- Investigations
  - Chest X-ray: Hilar lymphadenopathy and Eggshell calcification
  - HRCT chest: Galaxy sign
  - Gallium scan (not done currently): Panda sign signifies parotid gland, lacrimal gland involvement
    → Lambda sign: Hilar lymphadenopathy
  - Kveim test (not useful): Spleen of expired patient of sarcoidosis is used
    → Antigen is injected into another person where sarcoidosis is suspected

YOURWISH

→ Because of the incidence of prion disease and Creutzfeldt-Jakob disease, this test is not done

- Bronchoalveolar lavage - Shows lymphocytosis
  → CD4/CD8 ratio > 3.5:1 (normal - 2:1)

Q8.14: What is the drug of choice for sarcoidosis with organ involvement?

Answer:

- Corticosteroids

BIG QUESTION 9: What is the significance of the Schober test in diagnosing ankylosing spondylitis and how does the disease typically present?

Broad Answer

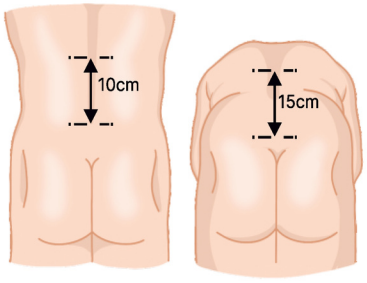
- The Schober test assesses lumbar spine flexion by measuring the increase in distance between two points marked 10 cm apart over the lower back; in ankylosing spondylitis, this increase is less than 5 cm due to spinal stiffness.
- The disease commonly presents in young males with chronic low back pain, morning stiffness, and sacroiliitis visible on X-ray.
- Progressive ligament calcification leads to "bamboo spine." Enthesopathy and extra-articular features like aortic regurgitation and costochondral joint stiffness are common.

DETAILED QUESTIONS

Q9.1: Describe Schober test

Answer:

- Schober test: Reveals involvement of spine because spine flexion would not be possible
  - In a normal person, if asked to bend forward - It is possible because the spine is flexible
  - Markings: Put first mark in respect to posterior superior iliac spine and 2nd mark at a distance of 10 cm above
  - In a normal person, when asked to bend forward - there would be stretching, and this gap would increase to 15 cm
  - The Schober test is done to check for the ability or normal flexibility of the spine or to evaluate the degree of spine flexion possible
  - But in this patient, flexion is at a limit of < 5cm

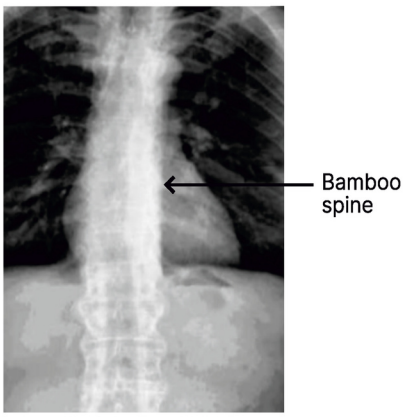


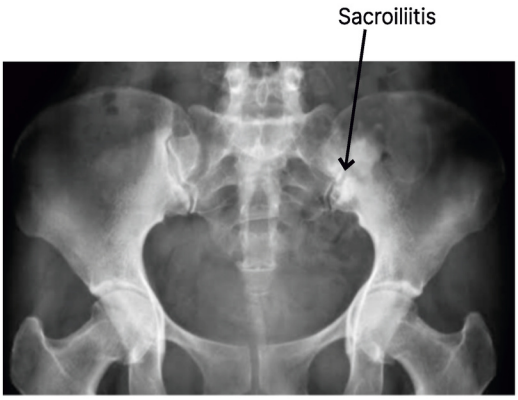
Q9.2: Describe the presentation of Ankylosing Spondylitis

PYQ: FMGE 2019

Answer:

- Presentation: A young male patient presents with low backache.
  - X-ray of lumbosacral area is done - it shows sacroiliitis
  - If the disease is progressive, it shows a large number of osteophytes present,
  - Calcification present in paravertebral ligaments, which results in the presentation of bamboo spine
  - In this patient, the complaint would be that when he wakes up in the morning, he has pain in his back, and when he stands or puts his foot on the ground, there is pain in the foot or sole





Q9.3: What is enthesopathy and how does it present in conditions like ankylosing spondylitis?

Answer:

- Enthesopathy: Tendons like the Achilles tendon can be involved so that tendinitis can be a presentation, and due to stretching of the tendon on standing, there could be complaints of heel pain

Q9.4: What is the cardiovascular manifestation of Ankylosing Spondylitis?

Answer:

- Valvular aortic regurgitation

Q9.5: What are the lung manifestations of Ankylosing Spondylitis?

Answer:

- Lung manifestations: There is stiffness of costochondral joint
  - Extra parenchymal restrictive lung disease
  - Because of this, the ability of patients to exhale air out of their lungs would be lesser because chest compliance is affected so residual volume is increased
  - In contrast, in parenchymal restrictive lung disease, there is fibrosis of the lung

Q9.6: What is the significance of a positive HLA-B27 test and in which conditions is it commonly found?

Answer: PYQ: INICET 2022

- HLA-B27 test is positive in Ankylosing spondylitis
  - It is also positive in
    - → Juvenile rheumatoid arthritis,
    - → Ulcerative colitis
    - → Reiter syndrome/Reactive arthritis

Q9.7: What imaging modality is done in Ankylosing Spondylitis?

Answer:

- MRI pelvis - Shows evidence of bone marrow edema

Q9.8: What is the treatment for ankylosing spondylitis?

Answer:

- Treatment: Infliximab - Monoclonal antibody TNF α agonist

Q9.9: What are the causes of low backache in older men?

Answer:

- Malignancy
- multiple myeloma
- prostate cancer-causing damage to the lumbosacral spine

Q9.10: What does low backache at young, which restricts his lifestyle, takes painkillers on a regular basis, morning stiffness', enthesopathy and tendinitis are indicative of ?

Answer:

- Ankylosing spondylitis

BIG QUESTION 10: Explain the pathophysiology, clinical features, diagnosis, and management of gout.

Broad Answer:

- Gout is caused by deposition of monosodium urate crystals in joints due to hyperuricemia, commonly affecting the 1st metatarsophalangeal joint (podagra). Repeated flare-ups lead to inflammation and characteristic punched-out "rat-bite" lesions seen on X-ray.
- Diagnosis is confirmed by joint aspiration showing turbid fluid with negatively birefringent needle-shaped crystals under polarized microscopy.
- Acute attacks are treated with NSAIDs like indomethacin, while aspirin is avoided as it worsens hyperuricemia.
- Chronic gout requires urate-lowering therapy such as allopurinol.
- Differentiation from pseudogout is crucial, where calcium pyrophosphate crystals with positive birefringence are seen.

DETAILED QUESTIONS

Q10.1: What is podagra?

Answer:

- Acute gouty arthritis involving the 1st metatarsophalangeal joint.

Q10.2: What radiological sign is characteristic of gout at the 1st MTP joint?

Answer:

- Punched-out "rat-bite" lesions or Martel sign.



Q10.3: Which crystal shows negative birefringence under polarized light in joint fluid?

Answer:

- Monosodium urate crystals in gout.

Q10.4: Which crystal shows positive birefringence and is seen in pseudogout?

Answer:

- Calcium pyrophosphate crystals.

Q10.5: What is the preferred initial treatment for acute gout?

Answer:

- Indomethacin (NSAID)
  - Aspirin is not used as it increases uric acid crystallization and worsens the attack.
  - If there is an Indomethacin (COX-1 inhibitor) allergy - Steroids are given
  - Allopurinol is not used in acute gout; it is useful in chronic gout

Q10.6: Why is aspirin avoided in acute gout?

PYQ: FMGE 2020,2021,2022
PYQ: NEET PG 2022,2023
PYQ: INICET 2021

Answer:

- Aspirin increases uric acid crystallization, worsening gout.

Q10.7: What test is diagnostic for gout?

Answer:

- Investigation of choice: Joint aspiration
  - On joint aspiration: Fluid-Turbid
  - Negative birefringence crystals in polarised microscopy present
  - Positive birefringence crystals in polarised microscopy are seen in Pseudo gout (Ca pyrophosphate), which involves the knee joint

Q10.8: What differentiates an overproducer and an underexcretor in gout?

Answer:

- 24-hour urinary uric acid excretion; overproducers have high excretion, underexcretors have low.

Q10.9: What tests are done in case acute gout progresses to chronic gout?

Answer:

- Tests done are:
  - Serum uric acid: Elevated
  - 24-hour urinary uric acid

Q10.10: Describe the underexcretor and overproducer in gout.

Answer:

- Under excreter: Uric acid is excreted by the kidney tubules; if excretion does not occur, the uric acid levels will rise
  - Serum uric acid levels are elevated in both overproducers as well as in underexcreters
  - Urinary uric acid levels will differentiate
- Overproducer: Avoid a non-vegetarian diet; certain vegetarian foods should also be avoided; if kidney function is normal, allopurinol is given; if not, febuxostat is given
  - For under excreters: Probenecid is given

BIG QUESTION 11: What are the key cutaneous features, muscle involvement, antibody associations, and related conditions in idiopathic inflammatory myopathies (dermatomyositis, polymyositis, inclusion body myositis, and antisynthetase syndrome)?

Broad Answer:

- Idiopathic inflammatory myopathies are a group of autoimmune disorders primarily affecting skeletal muscle and sometimes skin.

YOURWISH

- Dermatomyositis is recognized by characteristic rashes such as heliotrope rash and Gottron papules, along with symmetrical proximal muscle weakness, especially at the hips and shoulders.
- It has important associations with malignancy (notably ovarian cancer) and distinct nailfold changes.
- Polymyositis presents with similar proximal muscle weakness but lacks cutaneous manifestations and has a moderate cancer risk. Inclusion body myositis differs by presenting with asymmetrical, proximal, or distal weakness, often affecting men more than women, and is associated with characteristic biopsy findings and unique antibodies.
- Antisynthetase syndrome combines myositis with systemic features such as Raynaud's phenomenon, interstitial lung disease, and mechanic's hands, strongly linked to anti-Jo-1 and other tRNA synthetase antibodies.
- Across these conditions, multiple myositis-specific antibodies (e.g., Anti-Mi-2, Anti-MDA-5, Anti-TIF1, Anti-Jo-1, anti-cytosolic 5′ nucleotidase) aid diagnosis and stratify risk.
- Diagnostic confirmation is typically made with MRI-guided muscle biopsy, showing features such as perifascicular atrophy in dermatomyositis.

Q11.1: What is a heliotrope rash?

Answer:

- A violet or purplish rash around the upper eyelids, characteristic of dermatomyositis.



Heliotrope rash

Q11.2: Where are Gottron papules typically found?

Answer:

- Over the knuckles and interphalangeal joints.



Gottron papules

Q11.3: How does myositis present?

Answer:

- Myositis presentation - Mainly hip joint muscle weakness (symmetrical proximal muscle weakness)
  - o Perifascicular atrophy

Q11.4: What are the usual complaints in patients with myositis?

Answer:

- Usual complaints in these patients are difficulty in standing up from a squatting position

Q11.5: What type of muscle weakness is most common in dermatomyositis?

Answer:

- Symmetrical proximal muscle weakness, mainly affecting hip muscles.

Q11.6: What cutaneous manifestation appears on the nail beds in dermatomyositis?

Answer:

- Nail bed telangiectasia.

Q11.7: Which cancer is most commonly associated with dermatomyositis?

Answer:

- Ovarian cancer.

Q11.8: What is the Work up for Dermato-Myositis?

PYQ: FMGE 2023

Answer:

- CK MM levels - elevated
- Anti-MDA-5 (melanoma differentiation antigen) antibody present
- Anti-TIF1 (Transcription inhibitory factor) antibody
- Anti Mi-2
  - o These 3 antibodies are not confirmatory

Q11.9: What is the IOC for Dermato-Myositis?

Answer:

- MRI-guided muscle biopsy: Perifascicular atrophy

Q11.10: Describe Polymyositis

PYQ: FMGE 2023

Answer:

- Skin manifestations are not present in polymyositis
- Presentation: A lady who is having difficulty getting up due to symmetrical proximal muscle weakness
- Cancer risk: Increased but lesser with respect to polymyositis
- Antibodies are the same as those of dermatomyositis

Q11.12: What are the features of Inclusion Body Myositis?

Answer:

- Asymmetrical muscle weakness
- It could be either proximal or distal,
- Biopsy: to identify myositis and inclusion bodies
- Most rheumatological disorders are common in female patients, but in inclusion body myositis, Male > Female
- Antibody targeting against cytosolic 5' nucleotidase

Q11.13: Describe the features of Anti-Synthetase Syndrome

Answer:

- It would be having Myositis: Proximal muscle weakness
- Raynaud phenomenon: Feature of scleroderma
- Specific features: Interstitial lung disease & Mechanicl hand (cracks in the tips of fingers of the patient)
- Antibody present - Anti aminoacyl - t - RNA synthetase
- Anti-Jo-1 antibody increases the risk of the development of ILD

Q11.14: Which antibody is associated with Anti-synthetase syndrome?

Answer:

- Anti-aminoacyl-tRNA synthetase antibodies.

Q11.15: Name three antibodies associated with dermatomyositis.

Answer:

- Anti-MDA-5, Anti-Mi-2, and Anti-TIF1 antibodies.

Q11.16: Which antibody is linked to inclusion body myositis?

Answer:

- Anti-cytosolic 5' nucleotidase antibody.

8. NEPHROLOGY

BIG QUESTION 1: What are the different types of Dyselectrolytemias?

Broad answer: Dyselectrolytemias are imbalances of sodium, potassium, and magnesium. Hyponatremia can be hypovolemic or euvolemic, treated with fluids or restriction. Hypernatremia, often from dehydration, needs slow correction with hypotonic fluids. Hypokalemia causes ECG changes like ST depression and U waves, treated with potassium. Hyperkalemia and magnesium imbalances require urgent correction to prevent cardiac complications.

DETAILED QUESTIONS

Q.1.1: What is the formula for calculating total sodium deficit?

Answer:

- **Female**: Weight × 0.5 ´[Desired value - Actual value]
- **Male:** Weight ´0.6 ´[Desirable value - Actual value]

Q1.2: What are the key features of Hypovolemic Hyponatremia?

PYQ: FMGE 2022

PYQ: INICET 2022

Answer:

- TBS: Decreased
- TBW: Decreased
- **Causes:**
  - Diarrhea
  - Vomiting
  - Cerebral salt wasting syndrome
  - Addison's Disease
- **Treatment:** · ORS and IVF

Q1.3: What are the key features of Euvolemic Hyponatremia?

Answer:

- TBW: Increased
- TBS: Normal
- **Causes:**
  - SIADH: Causes of SIADH include **Cerebral toxoplasmosis and Oat cell lung cancer**
  - Postoperative: Non-judicious use of IVF (overcorrection of IVF)
  - Endurance sports
  - Hypothyroidism: Thyroid hormones play an indirect role in the regulation of antidiuretic hormone
- **Treatment:** Fluid restriction and Vaptans (V2 receptor blocker)

Q1.4: What are the key features of Hypervolemic Hyponatremia?

Answer:

- TBW: Increased (water >> salt)

- TBS: Increased
- **Causes:**
  - CHF
  - Cirrhosis
  - Chronic Kidney Disease
- **Treatment:** · Diuretics

Q1.5: What are the key features of Hyponatremia?

Answer:

- Dangerous Hyponatremia: **Any value < 125 meq/L**
- Fast correction of Hyponatremia will lead to **Osmotic demyelination syndrome**
  - Manifestations that can occur in this patient: **Stroke-like features**
- Fluid of choice for correction of severe hyponatremia: **3% saline/ Hypertonic saline**
  - Value of Na+ in **3% saline** = 514 meq/L
  - Value of Na+ in **0.9 % saline** = 154 meq/L

Q1.6: What are the key features of Hypernatremia?

Answer:

- When the Na+ value is **> 158meq,** it is called dangerous hypernatremia (causes a shift of fluids across the brain, leading to seizures)
- Leading cause of Hypernatremia: Debility in old age (low water intake)
- Causes for the development of Hypernatremia (Mnemonic: **MODEL**)
  - **M**edication - **Lithium** can contribute to nephrogenic diabetes insipidus, which causes loss of water from the body, and if this loss is not compensated, it will lead to Hypernatremia
  - **O**smotic diuresis
  - **D**iabetic Insipidus (Central diabetic insipidus and Nephrogenic diabetic insipidus)
  - **E**xcessive H2O loss
  - **L**ow H2O intake
- **Note:** An Important cause of Hypernatremia in pediatrics is due to improperly diluted ORS, which causes doughy skin on examination

Q1.7: What are the clinical features and management of Hypernatremia?

Answer:

- **Clinical features** (Mnemonic:) **SALT**
  - **S**kin: Flushed
  - **A**gitation
  - **L**ow-grade fever
  - Increased **t**hirst
- **Investigation: Urine osmolarity**
  - **<250 mosm:** Diabetic insipidus
  - **> 400 mosm**: Lactulose osmotic diarrhea
- Formula for correction in a symptomatic patient: **TBW x (Actual Na - 140)/140**
- Correction fluid: **5% Dextrose**
- Asymptomatic Hypernatremia: Liberal intake of H2O

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Q1.8: What are the key features of Hypokalemia?

Answer:

- **Clinical features:**
  - Cramps
  - Ileus
  - **Flaccid paralysis:** if weakness persists in spite of potassium correction, then the electrolyte imbalance is **Hypomagnesemia**
- Cause of death: **Diaphragmatic paralysis**
- CVS arrhythmia: **Torsades De pointes** → Both hypokalemia and hypomagnesemia can trigger torsades de pointes
- **Trans tubular potassium gradient**- if value is **> 4**, it indicates renal wasting → Two important causes of renal wasting are Gitelman syndrome (problem will be in DCT) and Bartter syndrome (Problem in thick ascending loop of Henle)

Q1.9: What is Pseudo-P-Pulmonale?

Answer:

- P-pulmonale is a p-wave more than 2.5mm in the presence of pulmonary artery HTN. Here, there is no Pulmonary artery HTN, so we call it Pseudo-P-Pulmonale
- Whenever potassium goes down, ST ↓, T-wave inversion, QU interval prolongation, and Prominent U wave occurs.

Q1.10: What is the Correction rule for Hyokalemia?

Answer:

- For value 3.0- 3.5 meq/L: ORAL
- For value <3.0 meq/L: IV
- 1ml KCl=2meq
- 1ampoule=10ml KCl contains 20 meq
- 20 meq causes potassium in the blood to rise by 0.25 meq

Q1.11: What are the ECG Findings of Hyperkalemia?

Answer:

- Tall, tented T-waves
- ST elevation

Q1.12: What is the Treatment of Hyperkalemia?

Answer:

- **Mnemonic: CABG-D2**
  - **C**alcium gluconate: Antagonizes the action of potassium on the heart
  - **A**lbuterol nebulization
  - **B**icarbonate: It is not used routinely; used when a patient is having metabolic acidosis concomitantly
  - **G**lucose + Insulin: The objective of giving insulin to the patient is to send potassium inside the cell. Insulin drip is one of the most effective drugs to treat dangerous hyperkalemia. The magnitude of fall is **0.5-1 meq /hr**
  - **D**iuretic: Loop diuretics cause i.e., urinary loss of potassium, Kaliuria
  - Most effective method: **Dialysis**
- **For chronic Hyperkalemia**
  - Patiromer + Sodium Polystyrene sulfonate (K+ Bind)

Q1.13: What are the causes of Hypomagnesemia?

PYQ: NEET PG 2021

Answer:

- Hypomagnesemia will be present in Chronic diarrhea, Alcoholics, Use of Thiazides (because of action on DCT), and Gitelman Syndrome
  - TRPM6 is a receptor that helps in the reabsorption of Mg. Thiazides will block this receptor and congenital defect in Gitelman syndrome
  - TRPM6 will not be working as a result, both thiazides and Gitelman syndrome cause magnesium wasting
- Magnesium **antagonizes** the effect of **intracellular calcium**.

Q1.14: What are the side effects of Hypomagnesemia?

Answer:

- Muscle cramps
- HTN: As Mg antagonizes the action of intracellular calcium, low magnesium levels will lead to increased calcium activity, i.e., Vasoconstrictio,n and cause HTN
- Increase in HR
- Risk of development of Torsades-de-pointes: Hypokalemia, hypomagnesemia, and Hypocalcemia can cause TDP
- Increased neuromuscular excitability can cause tremors, Nystagmus, and Athetosis

Q1.15: What is the Treatment of Hypomagnesemia?

Answer:

- **Magnesium sulfate** (depending on the deficit, it can be given parenterally/IM/IV)
- Oral: Magnesium oxide

Q1.16: What are the key features of Hypermagnesemia?

PYQ: NEET PG 2021

PYQ: FMGE 2022

Answer:

- Normal values: **1.3-2.1** meq/L
- Death: If value goes > 10 meq/L, it can cause Asystole
- **Causes:**
  - CKD
  - Eclampsia: $MgSO_4$ overdose
  - Antacid, Laxative abuse
- **Clinical features:**
  - Shock, which is not responding to IV fluids, vasopressor (earliest feature)
  - DTR will decrease
  - Urine output will decrease
  - Respiratory rate will decrease

Q1.17: What is the treatment of Hypermagnesemia?

Answer:

- **Vigorous Hydration**
- **DOC: Calcium gluconate**
  - Calcium gluconate is used in the treatment of Tetany, acute hyperkalemia, and acute hypermagnesemia.
- Hemodialysis

Q1.18: What are the causes of death and Treatment in different Dyselectrolytemias?

Answer:

	Cause of death	Treatment
Sodium <125 meq	Seizure	3% saline
Sodium > 158 meq	Seizure	5% dextrose
Potassium > 8.0 meq	Diastolic arrest	Calcium gluconate
Potassium <2.5 meq	Diastolic paralysis	KCL + IVF
Calcium > 13 mg%	Systolic arrest	Ibandronate
Calcium < 7 mg%	Laryngospasm	Calcium gluconate
Magnesium > 10 meq	Asystole	Calcium gluconate
Magnesium < 1.0 meq	V. Arrhythmia	Magnesium sulfate

Q1.19: What is the difference between Urinary anion gap and Anion Gap?

PYQ: FMGE 2021 ,2023

Answer:

Urinary anion gap	Anion Gap
• Formula = $(Na^+ + K^+) - (Cl^-)$ • Normal value: 0 • In any patient of diarrhea where severe dehydration occurs, the perfusion of tissues will be hampered. When perfusion of tissue is less, they switch to anaerobic mode of respiration which results in generation of lactic acid and protons as a result the patient will end up in metabolic acidosis • To neutralize this proton, Ringer lactate can be administered (lactate will produce bicarbonate and this bicarbonate will neutralize the H^+ ion) • H^+ loss in urine: Kidney will be in Electron neutrality and kidney will also lose Cl^- • Negative: Negative urinary anion gap in diarrhea • Positive: Urinary anion gap is positive in Renal tubular acidosis (RTA will be characterized by impaired H^+ excretion)	• Formula = $(Na^+) - (Cl^- + HCO_3^-)$ • Normal value = 12 meq • Anion gap values are affected by serum albumin values: Require normal serum albumin • Albumin decrease of 1g/dl causes anion gap to fall by 2.5 meq. • NAGMA (normal anion gap metabolic acidosis): Seen in (Mnemonic: **DR. FUse)** o Diarrhea o RTA o Fistula: Enteric fistula, pancreatic fistula, Enterocutaneous fistula o Ureterosigmoidostomy • HAGMA (High Anion gap metabolic acidosis): Seen in (Mnemonic: **KULT)** o Ketoacidosis: Alcohol, starvation o Uremia: AKI, CKD o Lactic acidosis: Type A and Type B o Toxins → **Methyl alcohol poisoning: Antidote is Fomepizole** → **Ethylene Glycol poisoning: Antidote is Fomepizole** o Toluene: glue sniffing

Q1.20: What are the key features of Lactic Acidosis?

Answer:

- **Normal Levels:** 2-4 mmol/L
- **Type A:** Hypoperfusion/shock
- **Type B:**

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- Diabetes mellitus
- Phenformin
- Metformin can cause lactic acidosis if it is given to a patient whose eGFR is < 30ml/min
- Liver disease
- Malignancy
- TPN

BIG QUESTION 2: What are the key features, causes, diagnosis, and treatment of nephrotic syndrome?

Broad Answer: Nephrotic syndrome features massive proteinuria, hypoalbuminemia, edema, hyperlipidemia, lipiduria, and hypercoagulability. Primary causes include FSGS, membranous nephropathy, minimal change disease, and Finnish type; secondary causes include diabetes, amyloidosis, and lupus. Diagnosis uses urine protein quantification, microscopy, complement levels, and biopsy. Steroid-responsive cases get corticosteroids; resistant forms require cyclosporine/tacrolimus, with cyclophosphamide or levamisole for toxicity. Edema is managed with diuretics, and early therapy prevents complications.

DETAILED QUESTIONS

Q2.1: What are the key features of Nephrotic syndrome?

Answer: **Features**

- **Massive proteinuria:** > 3.5 g of protein present in urine in a 24 hr period (> 3.5 g / day)
  - To prevent under-collection/over-collection of urine, we do a spot sample where we check for protein
  - In spot sample > 2gm of protein per gram of urinary creatinine
- **Hypoalbuminemia:** This will reduce the oncotic pressure
- **Oedema**- usually begins in the periorbital area, and this edema tends to reduce as the day progresses, and later appears around the genitals, and subsequently the edema will migrate to the feet.
  - In a girl with minimal change disease, there will be vulvar edema
- **Hyperlipidemia:** Accelerated Atherosclerosis
  - Accelerated atherosclerosis can be seen in DM, hypothyroidism, metabolic syndrome, SLE, and Rheumatoid arthritis
- Lipiduria (oval fat bodies): Other conditions where lipiduria is present include Fat embolism syndrome, Chyluria
- **Hypercoagulable state:** Loss of ceruloplasmin, ferritin, Protein C,S & Antithrombin III.
  - Fibrinogen levels can be elevated

Q2.2: What is the difference between Primary and Secondary Nephrotic Syndrome?

Answer:

Primary nephrotic syndrome- causes include FSGS, MGN

- FSGS is the most common cause of Nephrotic syndrome in the adult population
- MGN is the leading cause of primary nephrotic syndrome in the geriatric population after 65 years of age

Secondary nephrotic syndrome- causes include DM, Amyloidosis, Lupus nephritis

Q2.3: What are the key features of Focal Segmental Glomerular Sclerosis?

Answer:

- In FSGS, as the disease is concentrated at the corticomedullary junction, there is an increased chance of misdiagnosing the disease when a superficial biopsy is taken.

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- The most common cause of nephrotic syndrome in adults
- Important causes:
  - **Primary cause:** Idiopathic
  - **Secondary cause:**
    → HIV associated nephropathy (most important cause)
    → HBV
    → Human Parvovirus B19
    → Heroine, Pamidronate, Lithium
    → Reflux Nephropathy
    → Hypertensive Nephrosclerosis
    → Alport syndrome
    → Sickle cell anemia

Q2.4: What are the key features of Membranous Glomerulopathy?

Answer:

Causes:

- **Primary cause:** Anti-PLA2 receptor antibody
- **Secondary Causes:**
  - Infection: HBV, HCV
  - Autoimmune: Graves/ Hashimoto disease
  - Connective tissue disorders
  - Solid organ tumors
  - Drugs: D-Penicillamine, Captopril

Histopathological examination:

- **Sub-epithelial** deposits with increasing matrix spike reaction

Q2.5: What are the key features of Minimal Change Disease?

Answer:

- Aka Nil Lesion/ Lipoid Nephrosis
- Cause: Idiopathic, NSAIDS, Hodgkin lymphoma
- Clinical presentation:
  - Pediatric age group patients present with Puffy eyes.
  - Genital edema, which subsequently migrates to the feet (pedal edema): Vulval edema in girls
  - Weight gain: Reduction in oncotic pressure causes oedema, which contributes to weight gain
  - Shortness of Breath: Due to pleural effusion (transudative)
  - Ascites: Ascites seen with Nephrotic syndrome will have a SAAG value <1.1g/dl → Ascites will contribute to an increase in the abdominal girth of the child

Q2.6: What do we do in the workup of Minimal Change Disease?

Answer:

1. KFT: Normal
2. Urine -
   - Routine microscopic examination: **oval fat bodies**
   - Dipstick: 4+

- Spot sample: Protein will be increased.
- Selective proteinuria: Mainly, low molecular weight proteins will be present

3. C3 levels: Normal
4. Kidney biopsy
   - Light microscopy: Normal
   - Electron microscopy: **Foot process effacement**

Q2.7: What is the treatment of Minimal Change Disease?

Answer: Treatment: As this condition responds very well to steroids, it is also known as **Steroid-responsive nephrotic syndrome.**

- Steroids- 8 weeks: Initially, steroids are given daily and later tapered
- For periorbital edema/ Weight gain/ Ascites: Spironolactone

Q2.8: What are the key features of Steroid Dependent Nephrotic Syndrome?

Answer:

- Treatment: Steroids
- Within a few days of starting steroids in this patient, there will be remission
  - If the urine of this patient is evaluated by dipstick, the urine albumin value will come as trace or nil
  - We need 3 consecutive nil reports of the early morning sample should be present to say the child of minimal change disease has gone into remission
- Features of steroid toxicity:
  - Cataract
  - Hirsutism
  - Hypertension
  - Myopathy
  - Weight gain
- Treatment of steroid toxicity: Taper steroids, Cyclophosphamide, and Levamisole
- Relapse: urine of the patient is showing 3+ / 4+ proteinuria, > 40 mg of protein / sq.meter 3 days early morning urine sample

Q2.9: What are the key features of Finnish Variety of Nephrotic Syndrome?

Answer:

- Neonate with congenital nephrotic syndrome
- Reason:
  - Defect in **NPHS 1 gene: Nephrin**
  - Defect in **NPHS 2 gene: Podocin** → Defect in Nephrin and Podocin will result in a defect in the filtration slit
- Clinical features:
  - Non-immunogenic hydrops fetalis
  - ANASARCA +
  - Foamy urine: Proteinuria
- Workup:
  - 24 hr urinary protein is increased (> 40 mg/m2)
  - Spot sample: 2-3 mg albumin/mg of Urine Creatinine

Q2.10: What are the varieties of nephrotic syndrome, the Age groups affected, and the findings?

Answer:

Verity of nephrotic syndrome	Age groups	Findings
FSGS	Adults	Hyaline deposits, foot process obliteration
MGN	> 65 yrs	Subepithelial deposits + Spikes
MCD	Child	IGM deposits, foot process effacement
Finnish variety	Neonate	Non-immunogenic hydrops fetalis, NPHS-2 has a poor prognosis
Relapse	Urine protein excretion > 40mg/m^2/h or nil or trace by urine dipstick on spot sample for 3 consecutive days	
Infrequent relapse	**One relapse within 6 months** of initial response, or **1-3 relapses in any 12-month period**	
Frequent relapse nephrotic syndrome	**Two or more relapses within 6 months** of initial response, or **four or more relapses in any 12- month period**	
Steroid-dependent nephrotic syndrome	The occurrence of 2 consecutive relapses during corticosteroid therapy or within 2 weeks of its cessation in the absence of infection	
Steroid-resistant nephrotic syndrome	Failure to achieve complete remission after 4 weeks of daily therapy with oral prednisolone of 2 mg/kg/day	

BIG QUESTION 3: Summarize the causes, key features, and diagnostic clues of nephritic syndrome, with emphasis on PSGN and IgA nephropathy.

Broad Answer: Nephritic syndrome is commonly caused by IgA nephropathy (Berger's disease) and post-streptococcal glomerulonephritis (PSGN). IgA nephropathy follows an upper respiratory infection with cola-colored urine appearing within 48 hours, normal C3, and normal BP. PSGN is a type III hypersensitivity reaction occurring 1-3 weeks after pharyngitis or 2-6 weeks after impetigo from nephritogenic Group A streptococcal strains; it presents with hematuria (smoky/cola urine), hypertension, oliguria, and low C3. Diagnosis of PSGN is supported by Anti-DNase B or anti-hyaluronidase antibodies and biopsy showing "starry-sky" immunofluorescence. Chronic kidney disease most often results from diabetic nephropathy, with cardiovascular disease as the leading cause of death.

DETAILED QUESTIONS

Q3.1: What are the key features of PSGN?

Answer:

- PSGN is an example of a **type III** hypersensitivity reaction.
- Occurs after infection with Group A streptococcus
- Some of the strains of Group A streptococcus are nephritogenic strains
- The surface of this bacteria contains **SP-EB** (Streptococcal pyogenic exotoxin- B) antigen, which results in antibody production, resulting in the formation of immune complexes. These immune complexes are distributed across the entire glomerulus, which, on immunofluorescence, shows a starry sky appearance
- **Nephritogenic strains** include: 1, 2, 3, 4, 25, 47, 49, 50, 55, 60

Q3.2: What are the renal Manifestations of PSGN?

Answer: **Renal Manifestations:**

1. Hematuria: Smoky urine, Cola color urine
2. Hypertension: due to activation of the RAAS system
   - Causes headaches
   - Hypertensive encephalopathy
   - Pulmonary edema: shortness of breath
3. Oliguria, Uremia present with or without chest pain.
   - Antibodies that aid in the diagnosis of this condition are: **Anti-DNAse antibody, Anti-hyaluronidase antibody**

Q3.3: What is the difference between Berger disease and PSGN?

Answer:

1. Berger disease PYQ: FMGE 2021	2. PSGN
• Presentation include: o URTI o cola color urine (appear as early as 48 hrs) o C3 values: Normal o BP will be normal	• Presentation include: o Impetigo or Pharyngitis o Hematuria appears within weeks o C3 values: Low o BP: Elevated o Antibodies +

Q3.4: How to calculate eGFR?

Answer:

$$eGFR = \frac{K \times height}{Serum\ creatinine}$$

Q3.5: What is the difference between the (MDRD) Study equation and the (CKD-EPI) equation?

Answer:

(MDRD) Study equation	(CKD-EPI) equation
GFR (mL/min/1.73m^2) = 175 ´ $(S_{Cr})^{-1.154}$ ´ $(Age)^{-0.203}$ ´ 0.742 (if female) ´ 1.12 (if African American) • It is used to estimate GFR in adult patients with chronic kidney disease • It isn't valid in many population groups including children, the elderly (>70 yr) and patients with serious comorbid conditions. • The equation was found to underestimate measured GFR at higher levels (>60 mL/min/1.73m^2)	GFR (mL/min/1.73m^2) = 141´ min $(S_{Cr}/K,1)^a$ ´ max$(S_{Cr}/K,1)^{-1.209}$ ´ 0.993^{Age} ´ 1.018 (if female) ´ 1.159 (if African American) Where, • **SCr** is serum creatinine in mg/dL, • **K** is 0.7 for females and 0.9 for males • **a** is -0.329 for females and -0.411 for males • **min** indicates the minimum of SCr/K or 1 • **max** indicates the maximum of SCr/K or 1 • It is used to estimate GFR in adult patients with chronic kidney disease • Considered more accurate than the MDRD Study equation for a GFR > 60 mL/min/1.73m^2.

Q3.6: 40-year-old female weighting 40 kg, with type 2 DM, on annual medical checkup, her S. creatinine was 1 mg/dl. Calculate the eGFR by the Cockcroft-Gault formula?

Answer:

- eGFR = 140 - age X weight / 72 X Serum creatinine

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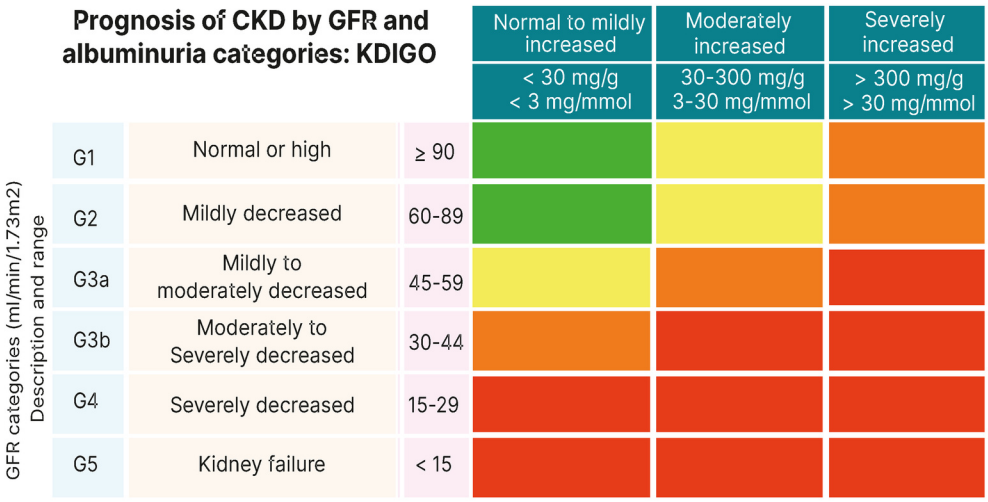
- (140 - 40) X 40 / 72 X 1 = 55
- The Cockcroft-Gault formula tends to underestimate the GFR of the patient, especially in patients with low body weight
- This formula is valid for male patients; for female the formula will be 0.85 X (140 - 40) X 40 / 72 X 1

Q3.7: What is the prognosis of CKD by GFR and albuminuria categories: KDIGO?

Answer:



Q3.8: What are the key features of Chronic Kidney Disease?

Answer:

- The leading cause of CKD is diabetic nephropathy
- Leading cause of death: Cardiovascular diseases
- Kidney transplantation or Hemodialysis should be done at Grade 4
- Target BP in a CKD patient: **<130/80 mm Hg.**
- CKD patients can present with edema. Pedal edema can be managed by thiazides.
- Another diuretic that can be used in a CKD patient is metolazone. Used when the eGFR of the patient is low (i.e., if the eGFR is < 30 ml)
- Once the patient goes into kidney failure/ ESRD, a-blockers can be used
- ACE inhibitors/ARBS are useful initially, but discontinued if the eGFR falls / potassium↑
  - Can start KALIURETICS: Furosemide + Metolazone or Patiromer
- Uremic pericarditis is an absolute indication for dialysis
- In CKD patients, there will be elevated values of Phosphate and decreased calcium values

Q3.9: What are the causes of Elevated phosphate, and how is it managed?

Answer:

- In CKD patients, there will be elevated values of Phosphate and decreased calcium values
  - An elevated phosphate value is due to defective excretion
  - Low calcium value is a result of low vitamin D3 synthesis by the kidney
  - Because of Low calcium values, these patients will have elevated values of PTH, i.e., all patients with CKD will have Secondary Hyperparathyroidism
    - → The most common cause of **Secondary Hyperparathyroidism** in **CKD**.
    - → The most common cause of **Primary Hyperparathyroidism** is **Parathyroid Adenoma**. Most commonly, the inferior parathyroid gland will be involved.

→ A special scan done in this condition is a **Sestamibi Scan**

- Elevated phosphate is managed by:

1. **Phosphate Binders** (taken with meal): Calcium Acetate, Calcium Carbonate, Sevelamer, Lanthanum
   - Calcium acetate and calcium carbonate can cause a disproportionate rise in calcium. Once calcium values increase, they can contribute to the **calcification of blood vessels** in this patient
   - **Sevelamer** and **lanthanum carbonate** are the preferred drugs as they do not cause calcification of blood vessels.
2. Tenapanor: It inhibits the absorption of phosphate from the GI tract
3. Calcitriol: Counter-regulatory hormone of PTH. A disadvantage of calcitriol is **hypercalcemia**

Q3.10: What is the approach used for the management of any patient with CKD?

Answer:

- A - HbA1C ↓ 7%: Adjust insulin dosage (give only 80% of calculated dose)
- B - BP control: Initially, ACE inhibitors can be used, watch for K+ ↑. If Potassium is rising despite Kaliuretic / Patiromer, switch to CCBs
- C - Cholesterol ↑: Statins
- D - Vitamin D3 ↓ : Vitamin D3 supplementation,
- E-EPO↓: Iron supplementation + Erythropoietin manufactured by DNA recombinant technology
- P - Phosphate ↑: Sevelamer
- K - K+ ↑: Patiromer

Q3.11: What are the Opportunistic Infections Post Kidney Transplant?

PYQ: INICET 2021

Answer:

Peri transplant: < 1 month	Early: 1-6 months	Late: >6 months
• Wound infection - MRSA • Herpes Simplex • Oral candidiasis • UTI	• P. Jirovecii • **CMV** o On urine microscopic examination, presence of basophilic inclusion bodies in cytoplasm and nucleus noted **(Owl Eye inclusions)** • HBV • HCV • Legionella	• Aspergillus • Nocardia • Polyoma (BK Virus) infection: special microscopic finding is **decoy cells** • HBV • HCV • Herpes zoster

Q3.12: What are Novel Biomarkers in AKI?

Answer:

1. **KIM-1**: It is expressed in the PCT. The values of KIM-1 will be elevated in urine and plasma
2. **NGAL** (Neutrophil gelatinase-associated lipocalin)/ Lipocalin/ Siderocalin- expressed in the PCT
3. **suPAR**: Soluble urokinase plasminogen activator receptor
   - BUN and creatinine are functional markers and are slow to rise
   - To confirm oliguric AKI: Give an IV bolus of furosemide 1.5mg/kg. If the urine output is < 200 ml at 2 hrs, even after givinga bolus of Furosemide, the patient is at risk of progression to severe AKI

Q3.13: What is the AKIN Criteria?

PYQ: FMGE 2023

Answer:

Stage	Serum Creatinine criteria	Urine Output Criteria
1	Increase of ≥ 3 mg/dL (≥26.4 µmol/L) or 1.5 - 2 fold increase from baseline	<0.5 ml/kg/h for >6h
2	> 2-to-3-fold increase from baseline	<0.5 ml/kg/h for >12h
3	> 3 fold increase from baseline or increase of ≥ 4 mg/dL (≥35.4µmol/L) with an acute increase of at least 0.5 mg/ dl (44 µmol/L)	<0.3 ml/kg/h for 24h or anuria for 12 h

Acute kidney injury (acute renal failure)			
Variable	Prerenal	Intrinsic Renal	Postrenal
Urine osmolality (mOsm/kg)	> 500	< 350	< 350
Urine Na^+ (mEq/L)	< 20	> 40	> 40
FENa	< 1%	> 2%	> 1% (mild) > 2% (severe)
Serum BUN/Cr	> 20	< 15	Varies
Cr = creatinine: FENa = fractional excretion of sodium			

Q 3.14: What is the most common variety of AKI & where are AKI with eosinophiluria seen?

Answer:

- **Most common variety of AKI: Prerenal variety**
- Test to differentiate Prerenal and renal variety of AKI
  - Example of prerenal variety of AKI is diarrhea with severe dehydration. Due to dehydration, most of the ultrafiltrate will be reabsorbed as a result the urinary sodium will be relatively less
  - In renal variety due to damage to PCT the SGLT2 will not work, and the salt will not be reabsorbed, hence the urinary sodium of the patient will be elevated
- Important cause for **post renal** variety of AKI: **BPH**

AKI with Eosinophiluria: Two possibilities

- Allergic interstitial nephritis seen with Antibiotics
- Atheroembolic kidney disease: H/o angiography will be given (KFT vales are normal before angiography but grossly elevated after angiography)

Q 3.15 What are the conditions where AKI with WBC casts are seen?

Answer:

- Pyelonephritis
- Interstitial nephritis
- Glomerulonephritis
- Allograft rejection
- Malignant infection of Kidney

Q3.16: What are the key features of Tumor Lysis Syndrome?

PYQ: FMGE 2019

Answer:

- Burkitt lymphoma has the highest chance of developing tumor lysis syndrome
  - Phosphate ↑: When cancer cells die, the ATP present in the cell will produce phosphate
  - Calcium ↓
  - Potassium ↑

- Uric acid ↑: Uric acid crystals can block the tubule and cause uric acid nephropathy
- Adequate hydration should be done in any patient of chemotherapy to prevent Tumor lysis syndrome
- The choice of Hypouricemic therapy depends on the risk assessment
  - **Allopurinol** is the agent of choice in intermediate-risk patients
  - **Rasburicase** is recommended for high-risk patients

Q3.17: What is the Patient Stratification risk in Tumor Lysis Syndrome?

Answer:

	Risk		
Type of Cancer	High	Intermediate	Low
NHL	Burkitt's, lymphoblastic, B-ALL	DLBCL	Indolent NHL
ALL	WBC ≥ 100,000	WBC 50,000-100,000	WBC ≤50,000
AML	WBC ≥ 50,000, monoblastic	WBC 10,000-50,000	WBC ≤10,000
CLL	-	WBC 10,000-100,000 Tx w/ fludarabine	WBC ≤10,000
Other hematologic malignancies (including CML and multiple myeloma) and solid tumors	-	Rapid proliferation with expected rapid response to therapy	Remainder of patients

Q3.18: What are the therapies for severe hypercalcemia?

Answer:

Therapies for Severe Hypercalcemia				
Treatment	Onset of action	Duration of action	Advantages	Disadvantages
Most Useful Therapies				
Hydration with normal saline	Hours	During infusion	Rehydration invariably needed	Volume overload
Forced diuresis; normal saline plus loop diuretic	Hours	During treatment	Rapid action	Volume overload, cardiac decompensation, intensive monitoring, electrolyte disturbance, inconvenience
Pamidronate	1-2 days	10-14 days to weeks	High potency; intermediate onset of action	Fever in 20%, hypophosphatemia, hypocalcemia, hypomagnesemia, rarely jaw necrosis
Zoledronate	1-2 days	>3 weeks	Same as for pamidronate (lasts longer)	Same as pamidronate above
Denosumab	1-2 days	>3 weeks	Strongest antiresorptive	Occasional severe hypocalcemia, rarely jaw necrosis, skin infections

YOURWISH

Q3.19: What are the special use therapies used in severe hypercalcemia?

Answer:

Special Use Therapies				
Calcitonin	Hours	1-2 days	Rapid onset of action; useful as adjunct in severe hypercalcemia	Rapid tachyphylaxis
Phosphate oral	24 h	During use	Chronic management (with hypophosphatemia); low toxicity if P<4 mg / dL	Limited use except as adjuvant or chronic therapy
Glucocorticoids	Days	Days, weeks	Oral therapy, antitumor agent	Active only in certain malignancies, vitamin D excess, and sarcoidosis; glucocorticoid side effects
Dialysis	Hours	During use and 24-48 h afterward	Useful in renal failure; onset of effect in hours; can immediately reverse life-threatening hypercalcemia	Complex procedure, reserved for extreme or special circumstances

Q3.20: What are the key features in the treatment of Hypercalcemia?

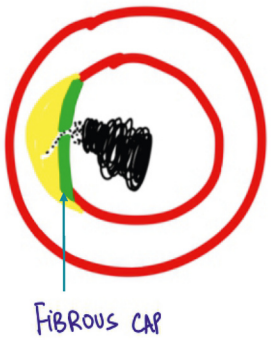
Answer:

- **First-line** (non-life-threatening): Hydration with normal saline to reduce calcium levels.
- **Drug of choice (DOC):** Bisphosphonates - pamidronate, zoledronate.
- Alternatives:
  - Calcitonin - rapid onset, short duration.
  - Oral phosphate.
  - Glucocorticoids - effective in *sarcoidosis, lymphoma, and multiple myeloma*.
  - Dialysis - indicated if K^+ is elevated with Ca^{2+} >14 mg/dL.
- **Severe hypercalcemia therapy:**
  - Bisphosphonates act in 1-2 days, lasting weeks.
  - Denosumab - most potent.
  - Calcitonin - works in hours; tachyphylaxis develops quickly.
- Furosemide caution: Avoid bolus; use only with IV fluids to prevent dehydration and worsening hypercalcemia.
- The **steroid-responsive hypercalcemia** conditions are sarcoidosis, lymphoma, and multiple myeloma

1. CARDIOLOGY PART-1

BIG QUESTION 1: What are the key events and ECG features associated with Myocardial Infarction (MI)?

Broad Answer: Coronary artery disease contributes to the maximum amount of mortality in India (the diabetic capital of the world). Myocardial infarction (MI) results from atherosclerotic plaque rupture in the coronary artery, leading to thrombus formation and complete lumen occlusion, which causes myocardial necrosis. Cut section of the coronary artery with atherosclerosis shows: Yellow: Fat deposition (lipid core in the tunica intima) and Green (The fibrous cap). ECG findings include hyperacute T waves, ST elevation, T wave inversion, and Pathological Q waves.



DETAILED QUESTIONS

Q1.1: What is the pathogenesis of myocardial infarction due to atherosclerosis?

Answer:

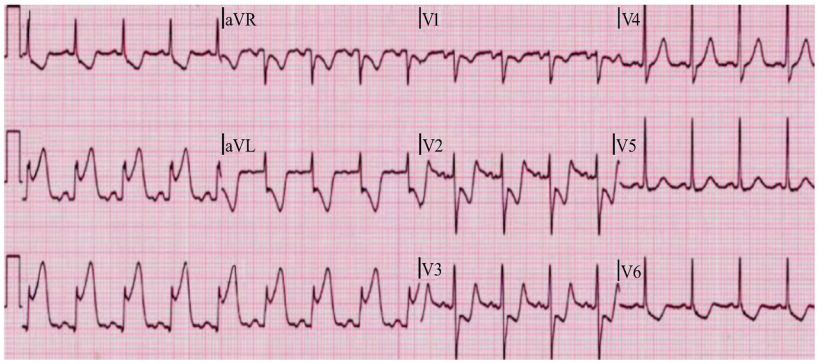
Atherosclerosis
↓
Blood flowing at a fixed pressure through the coronary artery
↓
Plaque fissure
↓
Exposure of collagen
↓
Platelet plug formation
↓
Clot/Thrombus formation
↓
Occlude the entire lumen
↓
Muscle death/Myocardial necrosis

Q1.2: What are the ECG findings in Inferior wall MI?

PYQ: FMGE 2019

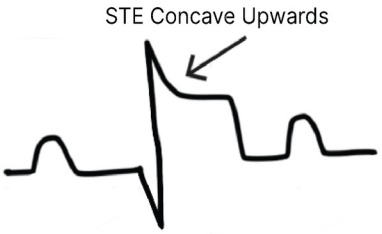
Answer:

- ST elevation in leads II, III, and aVF
- Reciprocal ST depression in V1-V4
- P wave present in Lead II, no Q wave
- Tombstone pattern: Convex ST elevation in the upward direction
- Most commonly due to RCA thrombus



Q1.3: How can you differentiate acute pericarditis from MI on ECG?

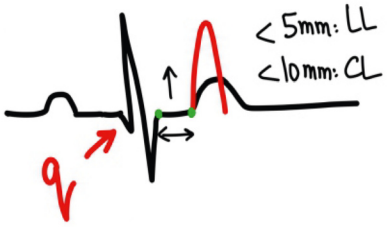
Answer:



- Acute pericarditis (Inflammation of the outermost layer of the heart) shows concave ST elevation
- MI shows a convex (tombstone) ST elevation
- Cardiac biomarkers (Troponin I and T, CPK-MB) are grossly elevated in MI, but normal in pericarditis

Q1.4: What is the earliest ECG change seen in MI, and what does it indicate?

Answer:



- Hyperacute T waves are the earliest changes
  - **Taller than normal T waves: >5 mm in limb leads, >10 mm in chest leads**
  - Indicates myocardial ischemia
  - Appears within seconds of infarction

Q1.5: What does ST elevation indicate, and what are its criteria?

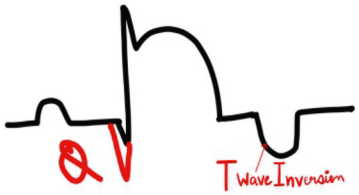
Answer:



- Indicates myocardial injury (current of injury)
- Appears within minutes
- Significant if:
  - ≥2 mm in men
  - ≥1.5 mm in women
- Myocardial injury is best confirmed with cardiac biomarkers

Q1.6: What is the significance of the T wave and Q wave in MI?

Answer:



- T wave inversion: Follows ST elevation
- In a normal individual, the Q wave is within one small square
- Pathological Q waves:
  - Develop after 1 hour of infarction
  - Indicate cell death or old MI
  - Deep Q waves persist lifelong

BIG QUESTION 2: How is myocardial injury and infarction defined, diagnosed, and managed?

Broad Answer: Myocardial injury is diagnosed when cardiac troponin (cTn) exceeds the 99th percentile upper reference limit, and it is considered acute if there is a rise or fall in cTn values. Myocardial infarction (MI) is diagnosed when there is evidence of acute myocardial ischemia, accompanied by elevated cardiac biomarkers and supporting clinical, ECG, or imaging findings. STEMI is best managed with PCI or thrombolysis, depending on the facility, while NSTEMI is treated with antiplatelet agents.

DETAILED QUESTIONS

Q2.1: What defines myocardial injury, and how is it detected?

Answer:

- Myocardial injury: Evidence of cTn levels with at least one value elevated above the 99th percentile URL
- Acute if there's a rise and/or fall in cTn

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- Normal Troponin I: 0.04 ng/dl
- Troponin I doubles → Confirms MI with myocardial necrosis
- **Troponin I rises in 3-4 hrs,** whereas CPKMB rises in 4-6 hrs
- ECG is done first, but ECG alone is not diagnostic or confirmatory. Cardiac biomarkers should also be checked
- Troponin I is more specific, and serial Troponin I is done if the patient presents early with definitive necrosis in the heart. The values are repeated, but the treatment is still initiated.

Q2.2: What is the clinical definition and diagnostic criteria for myocardial infarction (MI)?

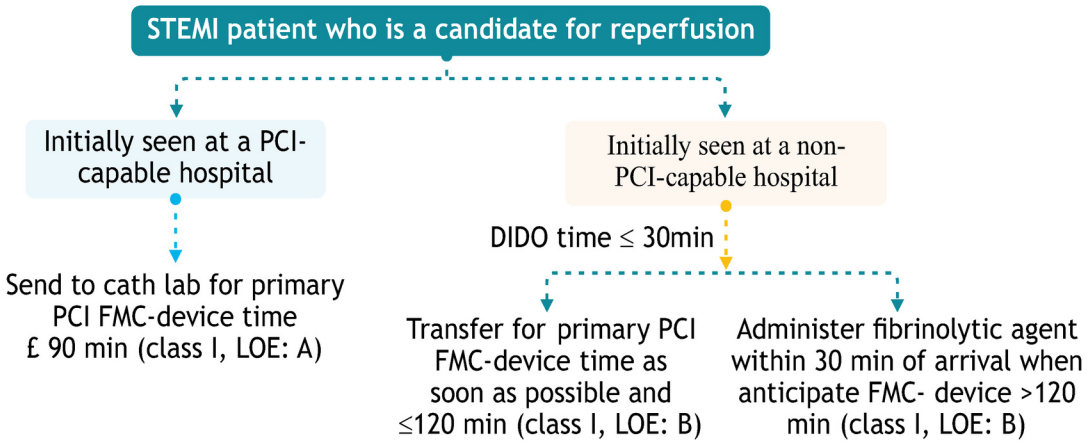
Answer:

- The term myocardial infarction (MI) should be used when there is acute myocardial injury with clinical evidence of acute myocardial ischemia and with detection of a rise and/or fall of cTn values with at least one value above the 99th percentile URL and at least one of the following:
  - Symptoms of myocardial ischemia
  - New ischemic ECG changes
  - Development of pathological Q waves
  - Imaging shows a new loss of viable myocardium or a new regional wall motion abnormality in a pattern consistent with an ischemic etiology
  - Coronary thrombus detected by angiography/autopsy (except in type 2 or 3 MI)
- **Levine sign:** Diffuse chest pain > 20 minutes, presenting with a clenched fist over the chest
- **ECHO:** Shows regional hypokinesia /stunned myocardium (Sudden decrease in contractility of the heart)
- Best diagnostic tool: Elevated biomarkers with one of the 5 above points

Q2.3: How is STEMI managed in PCI-capable hospitals?

PYQ: FMGE 2018

Answer:



- PCI-capable hospital with CATH lab facility:
  - Coronary angiography is done by using femoral or radial artery access (preferred)
  - Guidewire is sent retrogradely towards the heart
  - However, the contrast is unable to flow forward because of the coronary artery occlusion
  - In such cases, a balloon angioplasty/primary percutaneous intervention (PCI) is done
  - Done **<90 mins** of a patient coming in contact with medical services - **Door to balloon time/FMC (First medical contact) device time**

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Q2.4: How is STEMI managed in non-PCI-capable hospitals?

Answer:

- Non-PCI hospital:
  - Do thrombolysis in the shortest possible time
  - **Door-to-needle time: <30 minutes**
  - Initially, the patient is given **Dual Antiplatelet Therapy (DAPT): Aspirin + Ticagrelor**
  - Anti-ischemia measure: **Sublingual Nitroglycerine** (SLNTG)
    → Avoid if SBP is <90 mmHg - because nitrates are vasodilators and a further decrease in BP will compromise the coronary blood flow and renal blood flow
  - **Morphine**: Used when nitrates are given 3 times and there is no improvement
  - **O_2**: If SPO_2 < 93%
    → Example: Pulmonary edema seen in LV involvement
  - **Metoprolol** (ß-blocker): Reduces O_2 consumption by decreasing heart rate and thus reduces the size of the infarct
    → Avoided in bradycardia as it leads to hypotension

Q2.5: What is the leading cause of death in patients with diabetes mellitus?

Answer:

- Leading cause of death in DM - Increased incidence of cardiovascular mortality
- **In DM due to neuropathy, there is a Silent MI**

Q2.6: What drugs are used in STEMI vs. NSTEMI and why?

Answer:

- STEMI:
  - Fibrin & RBC-rich clot → use Fibrinolytics (Streptokinase, Alteplase, Reteplase, Tenecteplase)
  - Primary PCI = Treatment of Choice
- NSTEMI:
  - Platelet-rich clot → use Antiplatelet/Antithrombotic therapy
  - **Thrombolysis is contraindicated**

Q2.7: What is the time window for thrombolysis?

Answer:

- Upper limit for thrombolysis: <12 hours
  - >12 hrs side effects like bleeding are more than the benefits
- Ischemic stroke thrombolysis window: <4.5 hrs
  - Contraindicated in hemorrhagic stroke

Q2.8: What is the antidote to Streptokinase toxicity?

Answer: Antidote of streptokinase toxicity: Epsilon Amino Caproic Acid (E.A.C.A)

BIG QUESTION 3: How is the site of myocardial infarction localized using ECG, and what are the clinical implications of different infarct locations?

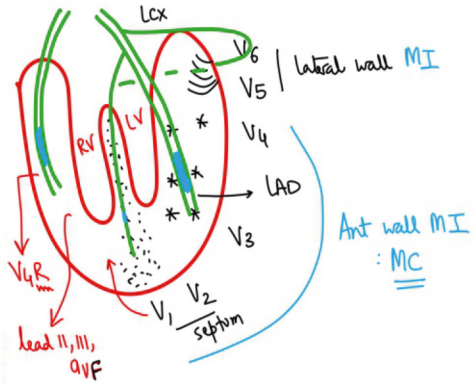
Broad Answer: ECG leads correspond to specific regions of the heart, helping identify the location of a myocardial infarction. Anterior, lateral, septal, inferior, and posterior infarcts are linked to specific coronary arteries. Extensive anterior MI due to LAD or left main artery involvement carries a high risk of cardiogenic shock and sudden cardiac death.

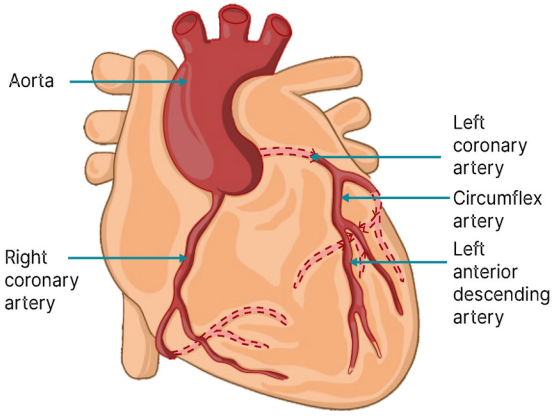
DETAILED QUESTIONS

Q3.1: Which ECG leads correspond to different anatomical regions of the heart?

Answer:

- **V1, V2** → Septum and part of LV
- **V3, V4** → Major part of LV (Anterior wall). Anterior wall MI is most common because the commonest blood vessel of the heart involved in atherosclerosis is the LAD
- **V5, V6** → Lateral wall (some part of LV facing the axilla)
- **V1** → Some part of RV
- **V4R** → Chest lead for RV (not routinely used)
- **II, III, aVF** → RV





Q3.2: Which coronary arteries are associated with infarcts in various ECG leads?

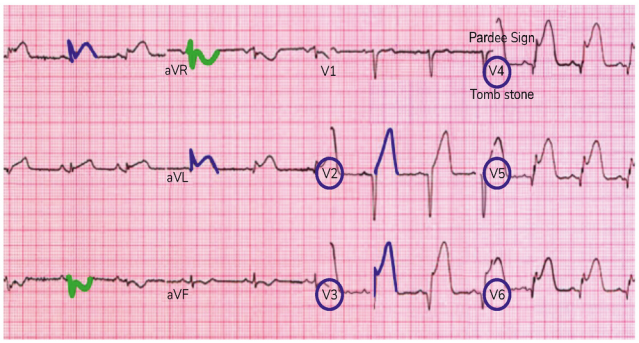
Answer:

Blood Vessel	Surface	Leads
Left circumflex artery	Lateral wall MI	I, aVL, V5, V6
Septal branch artery	Septal MI	I, aVL, V1, V2
Left anterior descending	Anterior wall MI	I, aVL, V1-V4
Left main coronary artery	Extensive anterior wall MI	I, aVL, V1-V6
Posterior descending artery	Posterior wall MI	V1-V4= STD V7,V8,V9= STE
Left anterior descending	NSTEMI anterior wall	V1-V4= STD
Right coronary artery	Inferior wall MI	Lead II, III, avF

Q3.3: What ECG findings indicate an extensive anterior wall MI?

Answer:

- ST elevation in V2-V6, I, aVL
- Reciprocal ST elevation in III, aVR
- Tombstone pattern/Pardee sign
  - Pardee waves refer to the symmetric inversion of T waves during an acute coronary syndrome or myocardial ischemia



Q3.4: What are the complications of extensive anterior wall MI?

Answer:

- Cardiogenic shock
- Pulmonary edema
- Sudden cardiac death (SCD)
  - Death within 1 hour of being seen/heard
  - It could be ventricular fibrillation or pulseless ventricular tachycardia (PVT)
  - Requires defibrillation

BIG QUESTION 4: What are the clinical features, pathophysiology, and treatment principles of Non-ST Elevation Acute Coronary Syndrome?

Broad Answer: NSTE-ACS includes unstable angina and NSTEMI, caused by plaque rupture and micro-embolization of thrombus fragments. Unlike STEMI, there is no full-thickness myocardial necrosis or ST elevation. Diagnosis relies on ECG and biomarkers. Thrombolysis is contraindicated, and management focuses on anti-ischemic therapy, antiplatelets, and anticoagulation.

DETAILED QUESTIONS

Q4.1: What is the pathophysiology behind Unstable Angina and NSTEMI?

Answer:

- Atheroembolism is seen in NSTEMI
- Pathophysiology:

Unstable angina: Plaque fissure leading to thrombus formation

↓

Inbuilt anticlotting system (Protein C, Protein S, Antithrombin III)

↓

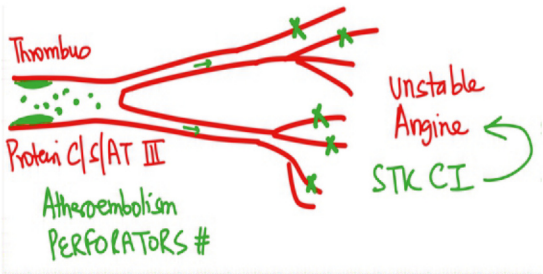
Breaks the clot into fragments

↓

Fragments block the perforators

↓

Chest pain



Q4.2: What are the key ECG and troponin findings that differentiate ACS subtypes?

Answer:

ACS Type	ECG Findings	Troponin I
STEMI	ST Elevation	Elevated ×2
NSTEMI	ST Normal or ↓	Elevated ×2
Unstable Angina	ST Normal or ↓	Normal

Q4.3: Which drugs are used in the management of NSTEMI and unstable angina?

Answer:

- **Anti-Ischemia measures:**
  - Sublingual nitrates or buccal spray
  - ß-blocker (Metoprolol): Decreases O_2 consumption of the heart, decreases HR, and reduces hypoxia

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- Morphine: Given if there is no improvement with nitrates.
  - → Morphine also reduces pulmonary edema
  - → Disadvantage: Stimulates the vagus nerve, and a person can have a vomiting sensation
- **Dual Antiplatelet drugs:** Aspirin + Clopidogrel/Prasugrel/Ticagrelor
  - Clopidogrel may not show good results, especially in Indian patients
  - P2Y12 inhibitors: Prasugrel / Ticagrelor
  - Commonly used nowadays: Aspirin + Ticagrelor
- IV Antiplatelet Drugs (at the time of PCI): Cangrelor, Eptifibatide, Tirofiban
- Parenteral anticoagulation: Low molecular weight heparin - Enoxaparin

Q4.4: Why is thrombolysis/streptokinase therapy contraindicated in NSTEMI and Unstable Angina?

Answer: Thrombolysis would further disseminate the fragments and worsen the symptoms

Q4.5: What is the difference between Acute chest syndrome and Acute coronary syndrome?

Answer:

- **Acute chest syndrome:** Seen in sickle cell anemia patients during sickling episodes → RBCs become sticky and occlude the coronary micro-circulation, leading to chest pain.
- **Acute coronary syndrome (ACS):** The problem is atherosclerosis

BIG QUESTION 5: What are the features of Prinzmetal angina?

Broad Answer: Prinzmetal (variant) angina is caused by coronary vasospasm, presents with chest pain at rest, and ST elevation without myocardial necrosis or elevated troponins. It is not considered a type of acute coronary syndrome (ACS).

DETAILED QUESTIONS

Q5.1: What are the key clinical and diagnostic features of Prinzmetal angina?

Answer:

- Chest pain
  - More in morning hours/winter season because Prinzmetal angina is related to **vasospasm**
- More common in **females**
- Past medical history of **Raynaud's phenomenon** (fingers changing white → blue →red)
- ECG showing ST elevation: It is the **only angina with ST elevation**
  - Also known as Variant Angina
- **Troponin I is normal** (vasospasm does not cause necrosis/death of cardiac muscles)

Q5.2: How is Prinzmetal angina managed?

Answer:

- Acute attack: Sublingual nitrates
- Prevention: Calcium channel blockers like **Amlodipine** (prevent vasospasm)

Q5.3: What are the features of type-1 MI?

Answer:

- Type-1 MI: Spontaneous MI as a result of intraluminal thrombus as a consequence of either atherosclerotic disease or dissection

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Q5.4: What is the type-2 MI, and what is the pathogenesis behind it?

Answer:

- Type-2: Secondary to ischemic imbalance
- An imbalance between myocardial oxygen supply and demand that is not precipitated by coronary artery disease (but can occur in the presence of stable coronary artery disease)
  - Example: Cocaine overdosage, Severe Anemia

Q5.5: What are the characteristic features of type-3 MI?

Answer:

- Type - 3: Resulting in death before Biomarker values
- Death occurring before biomarkers can be taken, where the clinical presentation and ECG changes support a diagnosis of MI
- Sudden death is due to tachyarrhythmias (V fibrillation, PVT) and Mobitz II heart block

Q5.6: What is the type-4A MI related to?

Answer:

- Type 4A: Related to percutaneous coronary intervention
- A rise of cTn greater than 5 times the upper limit of normal in association with any of the symptoms of myocardial ischemia; new ECG changes; angiographic loss of a coronary artery or persistent slow/no-flow; new regional wall motion abnormality demonstrated on imaging

Q5.7: What is type-4B MI related to?

Answer:

- Type 4B: Related to stent thrombosis
- Detected at either angiography or autopsy with an associated biomarker rise
- This is Latent stent thrombosis

Q5.8: What is the typ-4C MI related to?

Answer:

- Type 4C: Related to re-stenosis
- MI in the context of angiographically significant re-stenosis within the infarct artery

Q5.9: What is type-5 MI associated with?

Answer:

- Type 5: Related to coronary artery bypass grafting (CABG)
- Associated with CABG and a biomarker rise 10 times the upper limit of normal in association with any of: new pathological Q waves or LBBB; new graft or coronary artery occlusion; new regional wall motion abnormality demonstrated on imaging.

Q5.10: What is Mobitz Type II heart block, and how is it managed post-MI?

Answer:

- **Sudden death due to bradyarrhythmia after MI**
- Give **Atropine** 1 mg thrice as per the AHA 2020 guideline for management
- Definitive Management: Trans cutaneous pacemaker

BIG QUESTION 6: How is heart rate calculated on ECG, and what are the features of sinus arrhythmia?

Broad Answer: Heart rate on ECG is determined using the R-R interval and the number of large squares between two QRS complexes. As the number of large squares increases, the heart rate decreases. Lead used to calculate HR in a 12-lead ECG - Lead II/ Rhythm strip. Sinus arrhythmia is a normal physiological variation where the R-R interval varies with respiration due to vagal tone changes, and it should not be confused with pathological tachyarrhythmias.

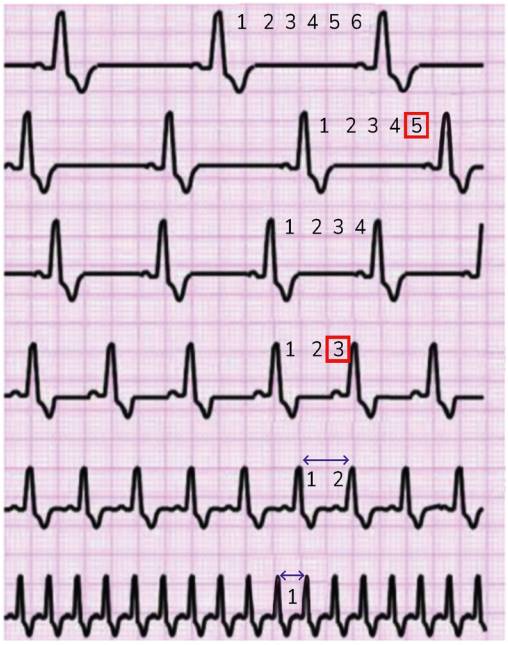
DETAILED QUESTIONS

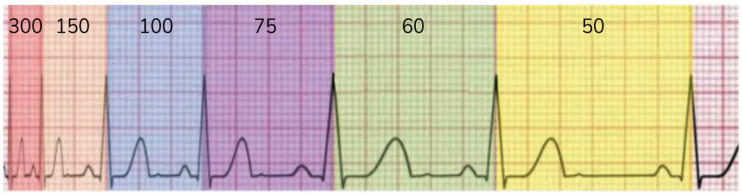
Q6.1: How do you calculate heart rate using ECG and R-R interval?

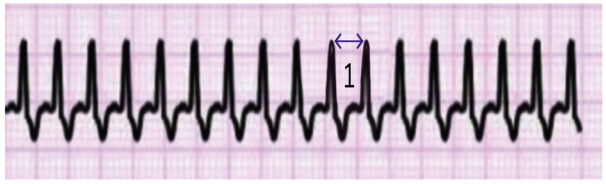
Answer: Heart Rate (HR) = $\frac{300}{\text{R-R interval}}$

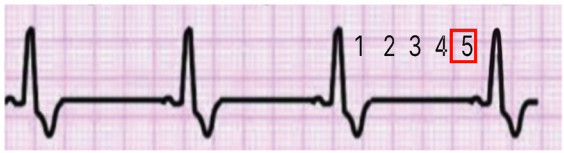
- R-R interval = Number of large squares
- Therefore, when the number of large squares increases, the Heart rate decreases

Example:





	• R-R interval: 1 large square • HR= 300/1 = 300.
	• R-R interval is composed only of 2 large squares • HR = 300/2 = 150 bpm. • Indicates Tachycardia.
	· R-R interval is composed of 3 large squares · HR = 300/3 = 100 bpm

	• R-R interval is composed of 4 large squares • HR = 300/4 = 75 bpm.
	• R-R interval is composed of 5 large squares • HR = 300/5 = 60 bpm.
	• R-R interval is composed of 6 large squares • HR = 300/6 = 50 bpm • Indicates Bradycardia. • A fit individual with bradycardia - High vagal tone might be the cause.

No. of Large Squares	Heart Rate
<3	>100 bpm (Tachycardia)
>5	<60 bpm (Bradycardia)

Q6.2: What is sinus arrhythmia, and what causes it?

Answer:

- Normal sinus rhythm with variable R-R interval that is not tachyarrhythmia is known as Sinus arrhythmia.
- Caused by vagal tone variation during respiratory phases



Q6.3: How do you identify sinus arrhythmia on ECG?

Answer:

- **P wave followed by QRS complex**
- Normal sinus rhythm
- **Varying R-R intervals**
- Heart rate changes between **100 and 75 bpm** (but not >100 bpm)

Q6.4: An 18-year-old female presents with recurrent respiratory tract infections. His ECG is given below. What is the most likely diagnosis?

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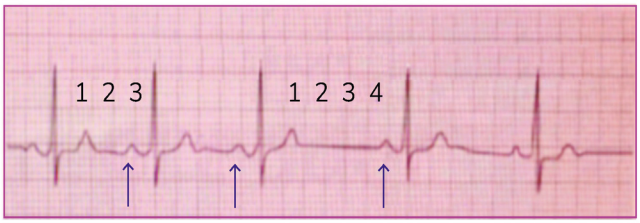
A. Wolff-Parkinson-White Syndrome

B. Atrial flutter

C. Atrial fibrillation

D. Sinus arrhythmia



Answer: D. Sinus arrhythmia

- ECG shows variable RR intervals, like in the above figure.
- Here HR is varies from 100 to 75bpm (this is not tachyarrhythmia: HR>100bpm)
- P wave with QRS complex present - normal sinus rhythm.
- Normal sinus rhythm + Varying HR = Sinus arrhythmia.

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Q6.5: What are the key features of Inferior wall MI?

Answer:

- The right ventricle is involved
- Inferior wall MI with bradycardia:
  - The vagus nerve innervates the wall of the RV along with the conduction system
  - Damage to the RV wall → Irritation of the vagus nerve → Bradycardia, Vomiting, hiccups
- The only type of MI that can have a history of significant gastrointestinal complaints.
- The only type of MI in which IV fluids are administered to maintain BP in case of bradycardia with hypotension.

BIG QUESTION 7: What are the clinical features, complications, and management of atrial fibrillation (AF)?

Broad Answer: Atrial fibrillation is the most common sustained arrhythmia and is characterized by the absence of P waves, irregularly irregular pulse, and variable R-R intervals. It may result in atrial thrombus formation, especially in the left atrial appendage, leading to embolic events like stroke and mesenteric ischemia. Management includes the RACE approach.

DETAILED QUESTIONS:

Q7.1: What are the ECG findings and clinical features of atrial fibrillation?

Answer:



- **ECG:**
  - Increasing and decreasing variations of HR
  - No P waves
  - Variable R-R interval
  - Twitching baseline
- Pulse: Irregularly irregular
- The most common sustained arrhythmia is:
  - Hypertrophic cardiomyopathy
  - Hypertension
  - >65 years age group

Q7.2: What complications are associated with atrial fibrillation?

Answer:

- Clot formation in the left atrial appendage due to atrial twitching
  - May embolize to the brain → Stroke
  - May embolize to mesenteric vessels → infarction of bowel with lower GI bleeding presenting as very severe abdominal pain, and if not identified, will lead to gangrene of the intestine
- Holiday Heart Syndrome:
  - Sudden cardiac death in alcoholics post-binge drinking
  - Post-binge drinking → inhibition of vagus nerve → Ectopic foci in AF increase HR (pathological tachycardia) → ↓ CO → Sudden cardiac death

Q7.3: What is pathological tachycardia?

Answer: All tachyarrhythmias decrease blood pressure

Heart beating disproportionately fast
↓
↓ Time for relaxation of heart/ Diastole
↓
↓Filling of heart
↓
↓EDV
↓
↓SV

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Q7.4: How is atrial fibrillation managed using the RACE protocol?

Answer: The order of treatment should not be bypassed

R	Rate control	• First step • Most commonly used drugs - Esmolol (short-acting ß-blocker)/ Verapamil (CCB)
A	Anticoagulants	• **Rheumatic Heart Disease/ Mitral Stenosis/ Left Atrial appendage dilatation: Warfarin is used** • Non-rheumatic AF: Newer Oral Anticoagulants (N.O.A.C)
C	Chemical cardioversion	• Use of certain drugs like amiodarone/ Ibutilide to shut down the ectopic focus in the left atrium
E	Electrical cardioversion	• Only used in case of failure of chemical cardioversion. • 200 joules biphasic of DC shock will be deployed

Q7.5: What is the CHA_2DS_2-VASc score, and how does it guide anticoagulation?

Answer: The CHA_2DS_2-VASc score is a clinical prediction rule for estimating the risk of stroke in people with non-rheumatic atrial fibrillation (AF)

Letter	Component	Score
C	Congestive Heart Failure	1
H	Hypertension	1
A_2	Age ≥ 75 years	2
D	Diabetes	1
S_2	Stroke	2
V	Vascular disease	1
A	Age ≥ 65 years	1
Sc	Sex category, female	1

- Maximum total score = 9
- Score = 0 - no therapy is preferred
- Score = 1 - Aspirin or oral anticoagulation
- Score ≥ 2 - Oral anti-coagulation

Q7.6: What are the anticoagulation recommendations based on the CHA_2DS_2-VASc score?

Answer:

Condition	Recommendation	
Nonvalvular atrial fibrillation	CHA_2DS_2-VASc score of 0	• Calculate CHA_2DS_2-VASc score • Aspirin or no antithrombotic
	CHA_2DS_2-VASc score of 1	Aspirin or OAC
	CHA_2DS_2-VASc score of ≥2	OAC
Rheumatic mitral valve disease	With atrial fibrillation, previous embolization, or atrial appendage thrombus, or left atrial diameter > 55 mm	OAC
	Embolization or appendage clot despite OAC	OAC plus aspirin despite OAC

Mitral valve prolapse	Asymptomatic	No therapy
	With otherwise cryptogenic stroke or TIA	Aspirin
	Atrial fibrillation	OAC

BIG QUESTION 8: How do you identify and manage supraventricular tachyarrhythmias?

Broad Answer: Supraventricular tachyarrhythmias include distinct ECG patterns and require targeted treatment. Multifocal atrial tachycardia (MAT) is seen in COPD patients and characterized by ≥3 different P wave morphologies. Atrial flutter shows a saw-tooth pattern and arises from macro re-entry in the right atrium. Paroxysmal supraventricular tachycardia (PSVT), especially AVNRT, presents with narrow QRS, merged P/T waves, and is managed with adenosine if the patient is hemodynamically stable.

DETAILED QUESTIONS

Q8.1: What are the features and treatment of Multifocal Atrial Tachycardia (MAT)?

Answer:



- Common in COPD patients with recurrent episodes of palpitations and dizzy spells
- ECG features:
  - o **P waves of ≥3 different morphologies/varieties**
  - o Variable R-R interval
  - o Heart rate >100 bpm
- DOC: Verapamil
- **DC shock is contraindicated**

Q8.2: What are the ECG features and management of Atrial Flutter?

Answer:



- A variant of atrial fibrillation, but with a different anatomical origin
- ECG feature: **Saw Tooth Pattern** & constant R-R interval

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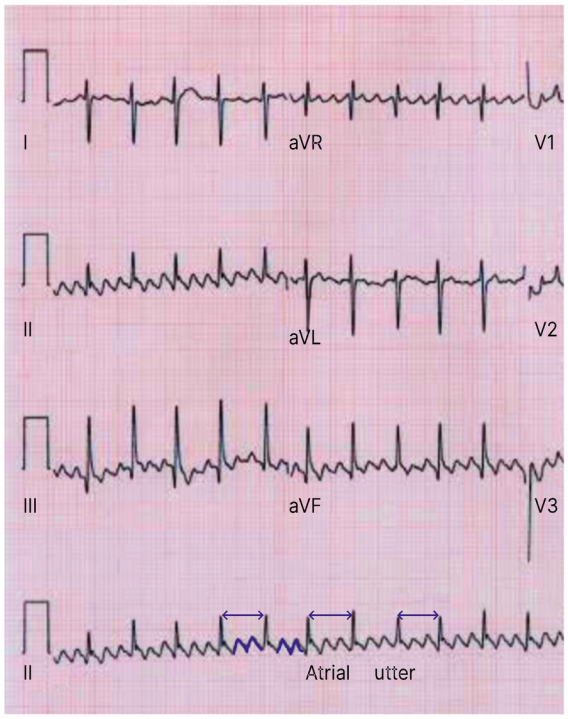
- Caused by macro re-entry around the cavotricuspid isthmus (right atrium)
- Management:
  - o Rate control: Esmolol
  - o Anticoagulation
  - o Ibutilide (chemical cardioversion)
    → **If fails → Low-intensity DC shock (25-50 J)**

Q8.3: Identify the findings seen in the given ECG.

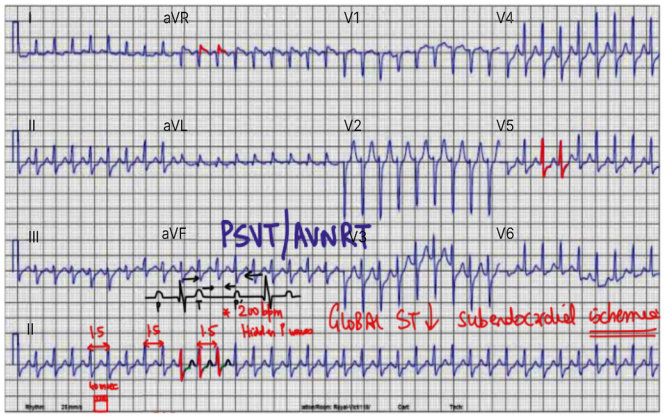
Answer: R-R interval is not changing, and a saw tooth pattern is seen, indicating Atrial flutter



Q8.4: What are the ECG features of Paroxysmal Supraventricular Tachycardia (PSVT)?

Answer:

- Also called **AV nodal re-entrant tachycardia** (AVNRT)
- ECG findings:
  - Tachyarrhythmia (Increased HR in lead 2)
  - Narrow QRS complex
  - Constant R-R interval
  - Merged P and T waves (**hidden P waves**)
  - Global ST depression → indicates subendocardial ischemia PYQ: NEET PG 2019



Q8.5: What are the management strategies used for PSVT?

Answer:

Crashing Patient	SBP>90mmHg	For Prevention
Synchronized DC shock (Cardioversion)	• Carotid sinus massage or Facial ice pack (child) • Adenosine (S/E: Transient bronchospasm) • Synchronized DC shock: 120-200 J (is done only when adenosine fails to work)	Verapamil

Q8.6: Differentiate between cardioversion and defibrillation in terms of shock synchronization and indications.

Answer:

- Cardioversion: Uses synchronized DC shock.
- Defibrillation: Uses non-synchronized DC shock, used for V. Fib and PVT

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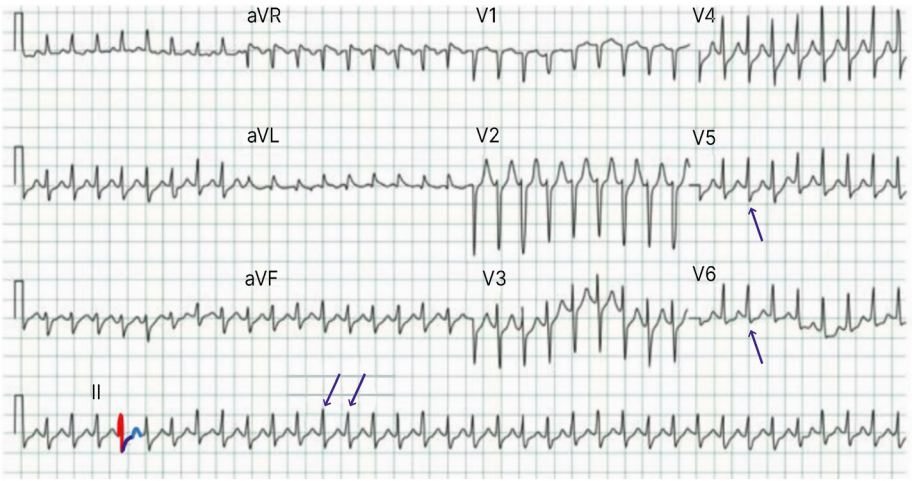
Q8.7: A diabetic patient presents with palpitations. His BP is 122/80 mmHg. His ECG is given below. What is the correct intervention?



A. Adenosine
B. Cardioversion
C. Amiodarone
D. PCI

Answer: A. Adenosine

- In the given ECG, a narrow QRS complex and ST depression, along with a merged P and T wave, are seen, indicating PSVT.
- As the BP indicates that the patient is stable, adenosine is preferred

BIG QUESTION 9: What are the features, ECG findings, and management of pre-excitation syndromes?

Broad Answer: Pre-excitation syndromes are cardiac conduction disorders where accessory pathways bypass the AV node, leading to early activation of the ventricles. **Wolff-Parkinson-White** (WPW) **Syndrome** involves the Bundle of Kent and shows delta waves with a short PR interval. **Lown-Ganong-Levine** (LGL) **Syndrome** involves the James bundle and is characterized by a short PR interval with normal QRS. Both can present with palpitations and syncope, and are managed with antiarrhythmic drugs acutely, and ablation for definitive cure.

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DETAILED QUESTIONS:

Q9.1: What is the pathophysiology of WPW Syndrome?

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Answer:

- There are two conduction pathways in the heart:
  - Normal conduction system
  - Abnormal conduction pathway - Bundle of Kent



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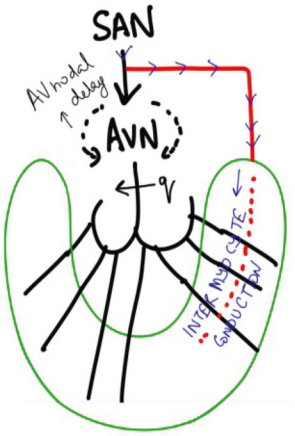
- There is a change in the slope of the upswing of the R wave - Delta waves.
- In this figure, the black line indicates the normal conduction pathway, and the red line indicates the pathological pathway
- In the normal conduction pathway:
  - Current goes from the SA node to the AV node, and there is an AV nodal delay
  - This pathway has its own self-limiting mechanisms
- In the accessory pathway:
  - Fast conduction: It bypasses the normal pathway, and the impulse from the SA node travels via the abnormal pathway/ bundle of Kent
  - This pathway does not have any decremental mechanisms

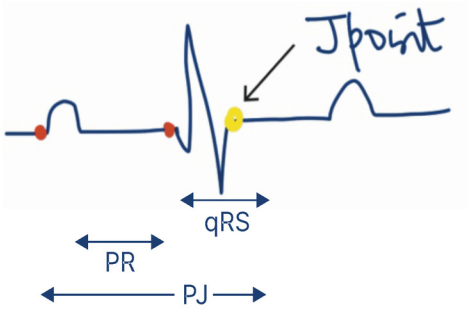


Q9.2: What are the clinical features of WPW Syndrome?

Answer:

- Clinical features:
  - Palpitations
  - Recurrent syncopal attacks
  - Family history of sudden cardiac death (especially in siblings)

Q9.3: What are the ECG findings in WPW syndrome?



Answer:

- Short PR interval because of fast conduction in the heart (PR interval is
- inversely related to HR)
- 'q' wave is absent as the current is not involving the AV nodal pathway and bundle of HIS
- Change in the upswing of the R wave: **Delta waves**
- Broader RS complex:
  - The delta wave contributes to a broader RS complex
  - Intermyocyte conduction: The current travels through the myocytes (slower) instead of the Purkinje fibers (Fastest conducting fiber in the heart)
- **PJ interval remains normal**
  - PJ= (↓) PR+ (↑) QRS
  - The amount of PR reduction is equal to the increase in the RS complex

Q9.4: Which EEG findings can be easily confused with these ECG findings?

Answer:

- Saw Tooth Waves in EEG are seen in REM sleep
- Delta waves (the slowest wave) in the EEG are seen in Stage 3 of NREM sleep

Q9.5: What are the ECG findings in Lown-Ganong-Levine (LGL) Syndrome?

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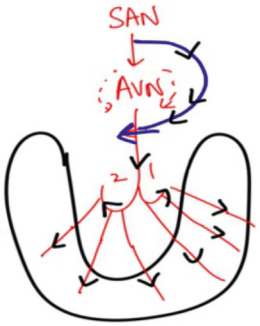
Answer:

- **Pre-excitation syndrome** of the heart
- Alternate conduction system: James bundle
  - Current from the SA node bypasses the AV node and reaches the bundle of His and Purkinje fibres

- ECG findings:
  - Short PR interval
  - 'q' wave is present
  - Normal QRS complexes
  - PJ interval is short
  - PJ= (↓) PR+ (n) QRS



Q9.6: What are the treatments for WPW and LGL syndromes?

Answer:

	Treatment
Emergency DOC	• Procainamide
Long-term prevention DOC	• Oral Flecainide
Definitive TOC	• Radiofrequency ablation of the accessory pathway

BIG QUESTION 10: What are the different QRS abnormalities on ECG?

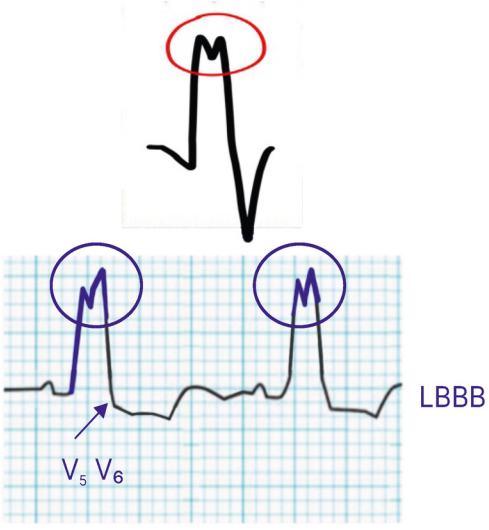
Broad Answer: QRS abnormalities help localize cardiac conduction disturbances. A narrow QRS suggests supraventricular origin, while a wide QRS indicates ventricular tachycardia.

DETAILED QUESTIONS

Q10.1: What are the common QRS complex abnormalities on ECG?

Answer:

QRS Pattern	Clinical Correlation
Narrow QRS tachycardia	Supraventricular tachycardia
Wide QRS tachycardia	Ventricular tachycardia / Polymorphic VT
Osborn wave	Hypothermia (core temperature <35°C)
Rabbit ear / M/W pattern in the peak of qRS	Bundle branch block • In LBBB: Seen in V5, V6 • In RBBB: Seen in V1, V2



Q10.2: How is the core body temperature graded?

Answer: Core body temperature grading:

Temperature (°C)	Grade
32-35°C	Mild hypothermia
28-32°C	Moderate hypothermia
<28°C	Severe hypothermia

Q10.3: What are the preferred sites for monitoring core body temperature?

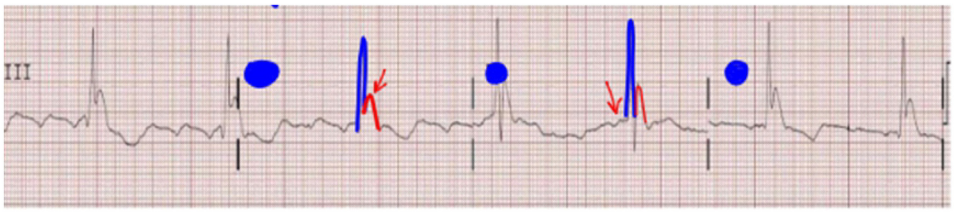
Answer:

- Ideal core temperature site: Pulmonary artery (invasive)
- Preferred non-invasive site: Lower esophagus

Q10.4: What are the ECG findings in hypothermia?

Answer:

- ECG findings:
  - Prolonged PR interval due to slowing of the heart in hypothermia
  - Inverted P wave (retrograde conduction)
  - Osborn wave: Notch in the downslope of the R wave



Q10.5: Which of the following is not an adaptation at high altitude?

A. Pulmonary vasoconstriction
B. Increased heart rate
C. Respiratory acidosis
D. Increased cerebral blood flow

Answer: C. Respiratory acidosis

- At high altitudes:
  - **Low PO_2** → Pulmonary **vasoconstriction** & **Cerebral vasodilation**
    → Hypoxia causes dilatation of all blood vessels in the body except the pulmonary artery
  - Cerebral vasodilatation leads to headache
  - Hypoxia leads to tachycardia
  - Since high altitude results in rapid respiration → CO_2 washout → it will cause respiratory alkalosis, not acidosis

Q10.6: Which of the following is not associated with central cyanosis?

A. Methemoglobinemia
B. Hypothermia
C. High altitude
D. Pulmonary AV malformation

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Answer: **B. Hypothermia**

- Meth hemoglobinemia: oxidized Hb ($Fe^{2+} \rightarrow Fe^{3+}$) leads to anemic hypoxia and causes central cyanosis
- Hypothermia causes localized vasoconstriction that is **associated with peripheral cyanosis,** not central cyanosis
- High altitude leads to hypoxic hypoxia and causes central cyanosis
- Pulmonary AV malformation: extracardiac shunt leads to mixing of arterial & venous blood and causes central cyanosis

BIG QUESTION 11: What is axis deviation on ECG, how is it interpreted, and what are its clinical implications?

Broad Answer: The heart's electrical axis can be estimated using lead I (X-axis) and lead aVF (Y-axis) on the ECG. The normal axis ranges from -30° to +90°. Deviation from this range is clinically significant.

DETAILED QUESTIONS

Q11.1: What is the normal range of the electrical axis of the heart?

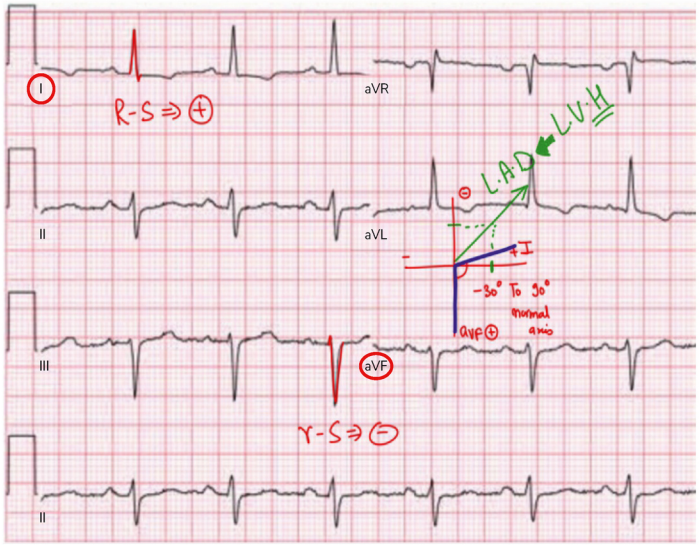
Answer:

- Normal axis of the heart: -30° to +90°
- Assessed using:
  - Lead I (X-axis)
  - Lead aVF (Y-axis)

Q11.2: How do you identify Left Axis Deviation (LAD) on ECG?

Answer:

- R wave is bigger than S wave; therefore, R wave - S wave gives a +ve value in Lead I
- R wave is smaller than S wave; therefore, R wave - S wave gives a -ve value in Lead aVF
- On vector analysis, it shows left-axis deviation
- **Seen in: Left Ventricular Hypertrophy (LVH)**



Q11.3: How do you identify Right Axis Deviation (RAD) on ECG?

Answer:

- R wave is smaller than S wave; therefore, R wave - S wave gives a -ve value in Lead I
- R wave is bigger than S wave; therefore, R wave - S wave gives a +ve value in Lead aVF
- On vector analysis: Right Axis Deviation
- **Seen in: Right Ventricular Hypertrophy (RVH)**

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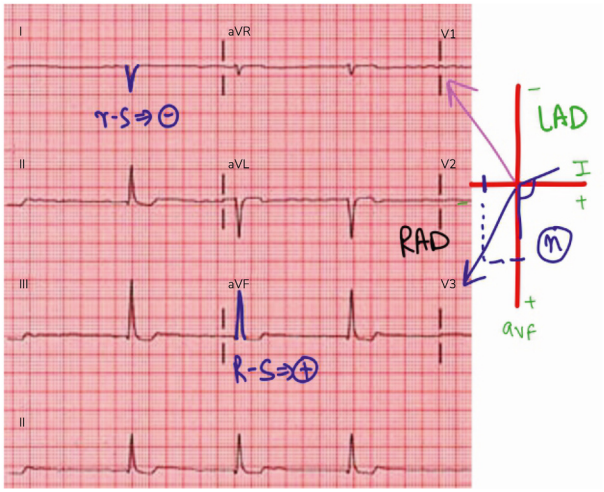


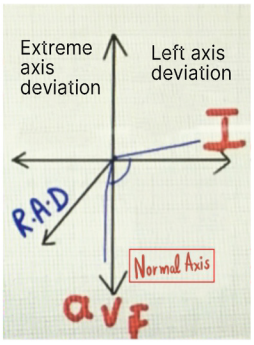
Q11.4: What is Extreme Axis Deviation and its implications?

Answer:

- If in ECG, the R-S wave is negative in both Lead I and Lead aVF, it indicates extreme axis deviation
- It is seen as Ventricular Tachycardia

Q11.5: An MBBS student was asked to diagrammatically make the axis deviation of a patient. The following schemata represents:

A. Right ventricular hypertrophy
B. Left ventricular hypertrophy
C. Biventricular hypertrophy
D. Normal axis in an obese individual



Answer: A. Right ventricular hypertrophy

- Negative R-S in Lead I + Positive R-S in Lead aVF = Right Axis Deviation → Seen in RVH

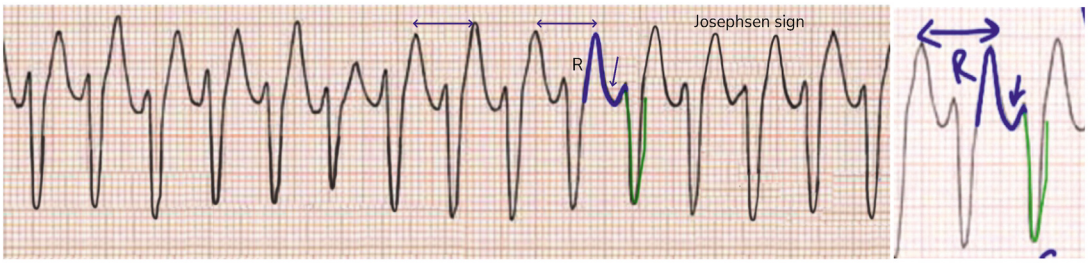
BIG QUESTION 12: What are the types of ventricular arrhythmias and their ECG features?

Broad Answer: Ventricular arrhythmias include Monomorphic VT, Polymorphic VT (Torsades de Pointes), and Electrical Alternans. These are identified by broad QRS complexes and distinct patterns on the ECG. Management depends on hemodynamic status.

DETAILED QUESTIONS

Q12.1: What are the key ECG findings of Monomorphic Ventricular Tachycardia (VT)?

Answer:



- HR ≃ 200 bpm
- Shortened R-R interval
- Broad QRS complex (>100 ms)

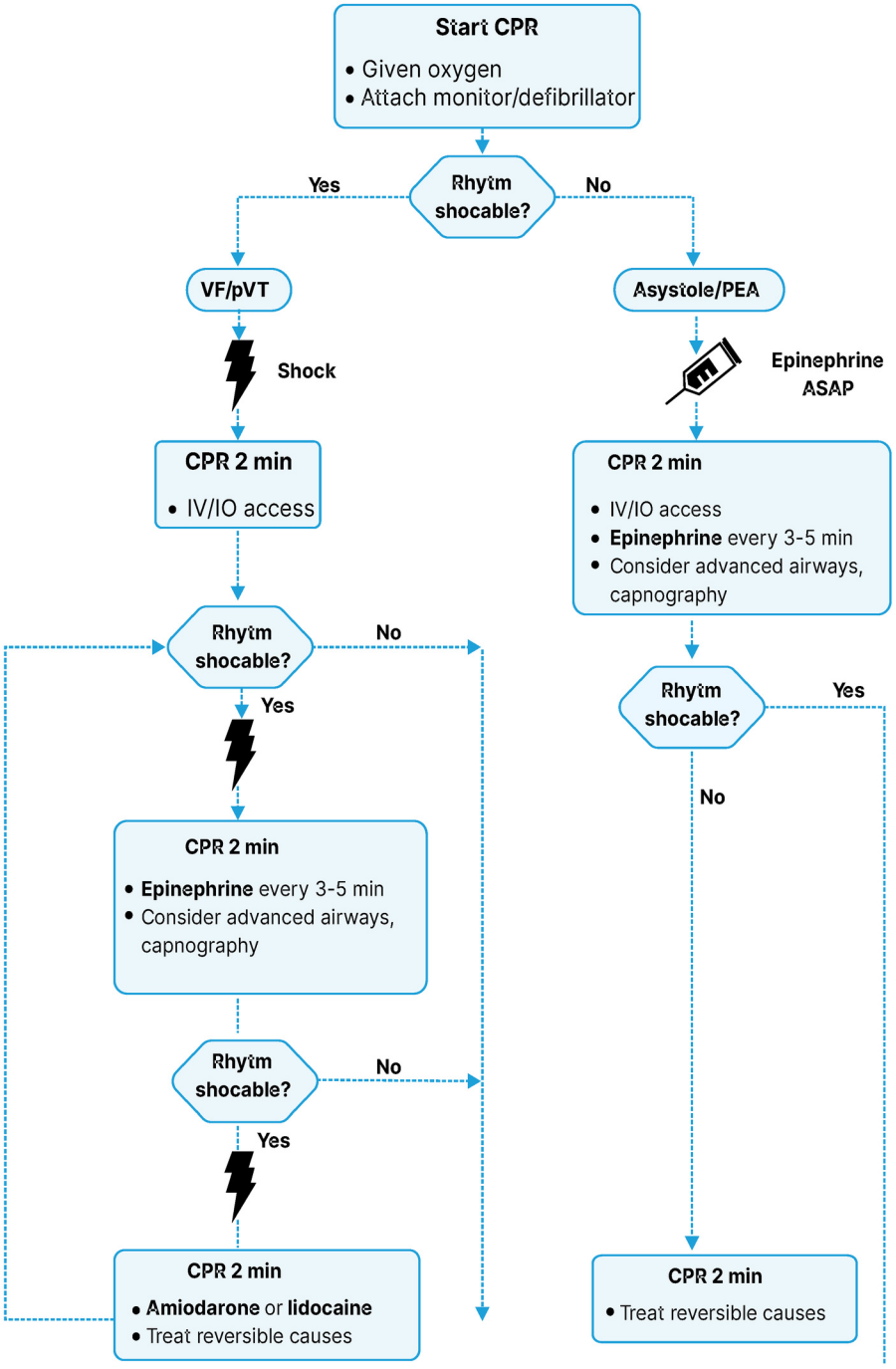
- Normal qRS complex is 2-2.5 small squares, i.e., 80 to 100 milliseconds
- All broad QRS complexes are monomorphic VT
- **Josephson sign: A Slight notch on QRS seen specifically in VT**

Q12.2: How is Monomorphic VT managed in stable vs unstable patients?

Answer:

Condition	Management
Hemodynamically stable VT	IV Amiodarone
Hemodynamically unstable VT	DC Shock (200 J biphasic) + CPR

PYQ: INICET 2021, 22, 23



Shock

- Push haed (at least 2 inches (5cm) and fast (100-120)/min) and allow complete chest recoil.
- Minimize interruptions in compressions.
- Avoid excessive ventilation.
- Change compressor every 2 minutes, or sooner if fatigued.
- If no advanced airway, 30:2 compression-ventilation ratio.
- Quantitative waveform capnography
  - If $PETCO_2$ is low or decreasing. reassess CPR quality

Shock Energy for Defibrillation

- **Biphasic**: Manufacturer recommendation (eg, intial dose of 120-200 J); if unknown, use maximum available. Second and subsequent doses should be equivalent doses should be equivalet, and higher doses may be considered
- **Monophasic**: 360J

Drug Therapy

- **Epinephrine IV/IO dose:** 1 mg every 3-5 minutes
- **Amiodarone IV/IO dose:** First dose : 300 mg bolus. Second dose: 150 mg. or
- **Lidocaine IV/IO dose:** First dose:1-1.5 mg/kg. Second dose: 0.5-0.75 mg/Kg.

Advanced Airway

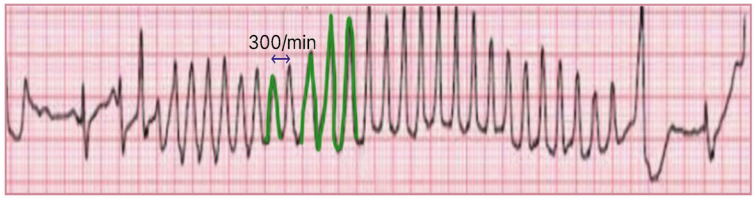
- Endotracheal intubation or supraglottic advanced airway
- Waveform capnography or capnometry to confirm and monitor ET tube placement
- Once advanced airway in place give 1 breath every 6 seconds (10 breath/nmin) with continuous chest compressions

Return of Spontaneous Circulation (ROSC)

- Pulse and blood pressure
- Abrupt sustained increases in $PETCO_2$ (typically > 40 mmHg)
- Spontaneous arteial pressure wave

Q12.3: What is Torsades de Pointes (Polymorphic VT) and how is it treated?

Answer:

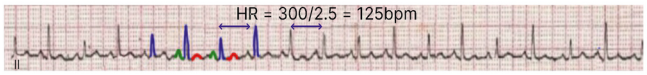


- HR ≃ 300 bpm
- Variation in the size of the broad QRS complex
- Causes:
  - o ↓Magnesium
  - o ↓Potassium
  - o Anti-arrhythmics: Class Ia, Ic, III
- Drug of choice: IV **$MgSO_4$**

PYQ: FMGE 2021

Q12.4: What is Electrical Alternans, and what condition does it indicate?

Answer:



- HR ≃ 125 bpm
- Variable QRS complex amplitude
- Normal sinus rhythm with slight tachycardia
- Seen in Pericardial effusion
- Mechanism: Swinging of the heart in a bag of fluid surrounding the heart

Q12.5 In which conditions is blood pressure not recordable?

Answer:

- Cardiac Tamponade: Breath sounds present
- Tension Pneumothorax: Breath sounds absent

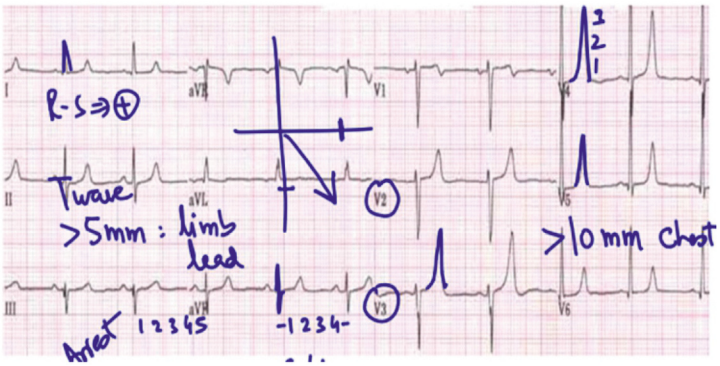
BIG QUESTION 13: What are the ECG changes and management principles in hyperkalemia and hypokalemia?

Broad Answer: Hyperkalemia and hypokalemia both manifest with characteristic ECG changes and can be life-threatening if not corrected promptly. Hyperkalemia presents with tall, tented T waves, a prolonged PR interval, and a sine wave pattern in its late stages. At the same time, hypokalemia exhibits T wave inversion, U waves, and pseudo-P pulmonale. ECG is supportive; serum potassium levels guide management.

DETAILED QUESTIONS

Q13.1: What are the ECG findings in hyperkalemia?

Answer:



- HR >60 bpm
- **Tall-tented T waves** (>10 mm in chest leads, >5 mm in limb leads)
- Increased R-R interval (K+ slows the heart)

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- **ST elevation**
- The amplitude of the P wave decreases and gradually disappears
- PR interval increases and broad QRS complex merges with T wave: **Sine wave pattern**
  - Occurs just before diastolic arrest
- ECG is not reliable alone; It is just supportive evidence.
- Serum electrolytes are more reliable
- Hyperkalemia can lead to death by causing diastolic arrest (K^+ slows the heart)

Q13.2: What are the ECG findings in hypokalemia?

Answer:



- **T wave inversion** (absent in severe cases)
  - Characteristic finding
- ST depression
- **Prominent U wave**
- **Pseudo-P pulmonale** (vertical height of P wave >2.5 mm in the absence of pulmonary artery HTN)

Q13.3: How is potassium corrected in hypokalemia?

Answer:

- **To raise K^+ by 1 mEq/L → 200 mEq of KCl is needed**
- Normal lower limit: 3.5 mEq/L
- Threshold: 3.0 mEq/L
- From 3.0 → 3.5 mEq/L: Oral potassium (Oral potchlor mixed with water/coconut water/banana)
- Below 3.0 mEq/L: IV KCl in fluids

Q13.4: How do you calculate potassium correction?

Answer:

- Example: A patient on Amphotericin-B has K^+ = 2.3 mEq/L
- To reach the threshold (3.0mEq/L), an increment of 0.7mEq/L is needed **(3.0 - 2.3 = 0.7 mEq/L)**
- Correction = **0.7 × 200 = 140 mEq KCl over 24 hours**
  - Note: Amphotericin-B is nephrotoxic and causes hypokalemia

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2. CARDIOLOGY PART-2

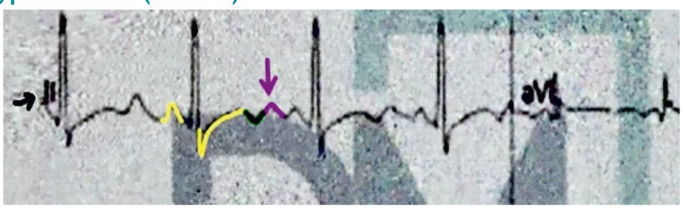
BIG QUESTION 1: What are the ECG changes, complications, and management principles of potassium imbalance?

Broad Answer: Potassium imbalance affects cardiac electrophysiology, producing characteristic ECG changes, arrhythmias, and potentially fatal complications. Hypokalemia is marked by tall P waves, ST depression, T wave inversion, and U waves, while hyperkalemia often causes bradycardia, diastolic arrest, or ventricular fibrillation. Management involves correcting potassium levels, stabilizing the myocardium, and preventing life-threatening arrhythmias.

DETAILED QUESTIONS

Q1.1: What are the characteristic ECG changes seen in hypokalemia (Lead II)?

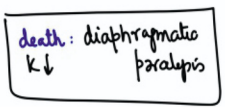
Answer:

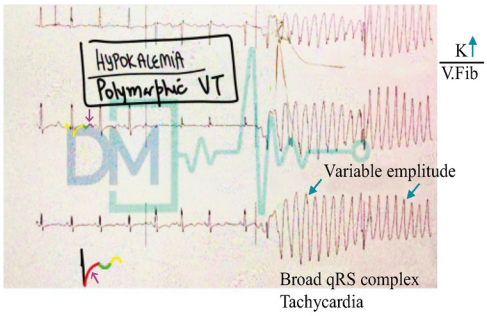


- P wave is slightly taller than normal
- Visible ST-segment depression
- T wave inversion
- Prominent U wave

Q1.2: What arrhythmias can occur in hypokalemia?

Answer:





- Polymorphic VT:
  - Broad QRS complexes
  - Reduced R-R interval
  - Varying QRS amplitudes
- Torsades de pointes (managed with $MgSO_4$)

Q1.3: What is the leading cause of death in hypokalemia?

Answer: Diaphragmatic paralysis

Q1.4: Can U waves be seen in normal individuals? If yes, why?

Answer: Yes, due to delayed papillary muscle repolarization.

Q1.5: What are the main cardiac effects of hyperkalemia?

Answer:

- Bradycardia
- Diastolic arrest
- Ventricular fibrillation (tachyarrhythmia)

Q1.6: Which drugs are used in the management of hyperkalemia?

Answer:

Chronic	Acute onset K↑
• Like in the case of grade 4 CKD 1. Patiromer 2. Sodium Zirconate 3. K bind - Sodium Polystrene sulphonate resin (oral or enema) o Side effect (Rare)-Colonic necrosis post enema component	1. Calcium gluconate or Calcium chloride (more efficacious) given IV 2. **Insulin** drip (Causes a redistribution of potassium) o **Most effective drug** o Supplemented with - Salbutamol / Albuterol (causes endogenous increase in insulin) 3. Furosemide - Kaliuria (Cause urinary loss of potassium) 4. **Hemodialysis - Most effective method**

- Calcium contracts so it antagonizes the effect of potassium, which relaxes the heart
- Calcium carbonate (oral salt) is not used

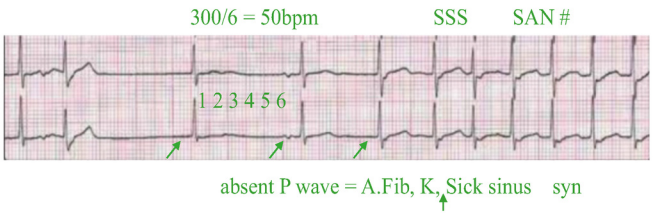
BIG QUESTION 2: What are the types, ECG features, and management principles of bradyarrhythmia?

Broad Answer: Bradyarrhythmias are rhythm disturbances characterized by abnormally slow heart rates due to sinus node or atrioventricular (AV) conduction defects. Sick sinus syndrome involves erratic SA node activity, while AV blocks (Mobitz I, Mobitz II, 2:1 block, third-degree block) vary in site and pattern of conduction delay or failure. Management ranges from observation to pacemaker implantation depending on symptoms and severity.

DETAILED QUESTIONS

Q2.1: What are the ECG features and clinical presentation of sick sinus syndrome?

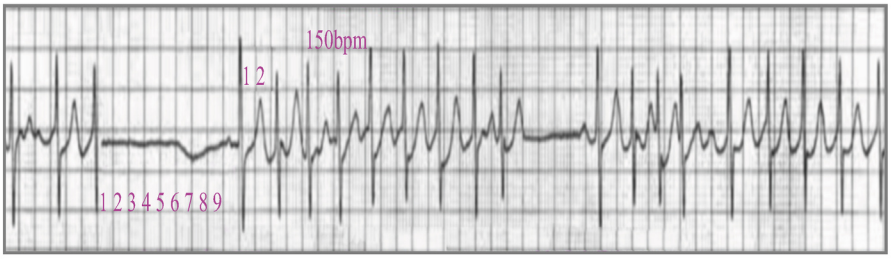
Answer:



- ECG: P wave absent or inverted, HR ≈ 50 bpm (300/60)
- SA node fires slower than normal (60-100 bpm)
- On exertion: Inadequate cardiac output → syncope
- Bradycardia-tachycardia syndrome: Alternating long and short R-R intervals due to erratic SA node firing
- No. of squares between R-R interval increases
- P wave is not deciphered, or it is inverted, which indicates malfunctioning of the SA node
- Heart rate = 300/60 = 50bpm
- SA node is not functioning properly (SA node normally fires 60-100 bpm)
- On exertion, the heart will not be able to generate the required amount of cardiac output, leading to a syncopal episode

Q2.2: What is Bradycardia-Tachycardia syndrome and how is it managed?

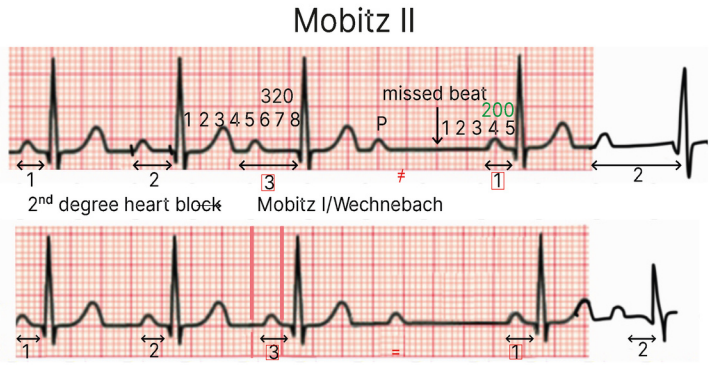
Answer:



- R-R interval of the patient is initially increased & then reduced
- A combination of tachycardia and bradycardia
- A peculiar situation is seen in sick sinus syndrome
- There is erratic behavior of the SA node - Either it is firing slower or faster, or totally shutting down
- Management
  - As the Heart rate is down to 30 bpm, Syncopal attacks may occur
  - A pacemaker is required in symptomatic patients, only if it interferes with the quality of life
  - A detailed Holter study has to be done

Q2.3: What are the key ECG features of Mobitz I (Wenckebach) second-degree AV block?

Answer:

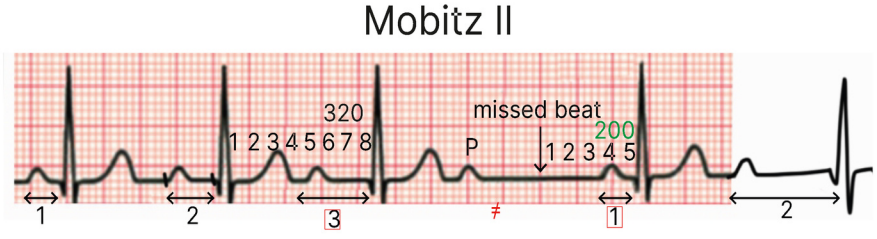


- Increasing trend of the PR interval (starting from P to the starting of the Q wave, it is not till the R wave)
- There is a P wave but no qRS complex
- There is a missed beat experienced by a person who feels their own pulse
- Subsequently, there is a reset in heart rhythm and a serial prolongation of PR interval, followed by a missed beat
- There is slow conduction via the AV node
- Diagnosis:
  - Whenever there is a missed beat, look for the PR interval after the missed beat and compare it with the PR interval before the missed beat
  - It will show a marked variation
  - PR interval before & after shall be unequal

Q2.4: What are the key ECG features of Mobitz II second-degree AV block?

Answer:

- PR interval for 1st, 2nd and 3rd time is constant
- P wave is followed by a missed beat, then again rhythm is reset
- PR interval that comes subsequently will be almost the same size
- Diagnosis:
  - Look for PR interval before and after the missed beat
  - It will always be the same
  - Multiple conductions followed by a drop



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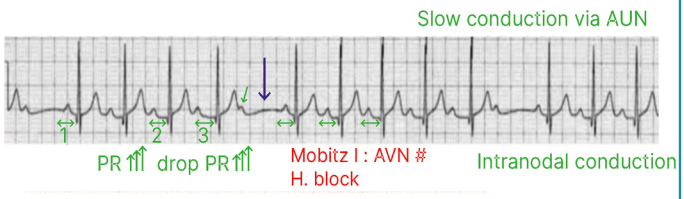
Q2.5: How does a 2:1 block differ from Mobitz type 1 and 2?

Answer:

ECG	Interpretation
	• For every one conducted P wave, another one is not conducted • **Interpretation -** It is called 2:1 Block • The PR interval after and before the missed beat is equal - can be confused with Mobitz type II heart block • **In 2:1 block- For every conduction, there is a drop**
	• There is a gradual increase in the PR interval, then there is a drop (marked by an arrow), and then again a progressive increase in the PR interval • Interpretation - **Mobitz Type I heart block**
	• There is a missed beat • PR interval after and before the missed beat is almost equal • Interpretation - **Mobitz Type II heart block**

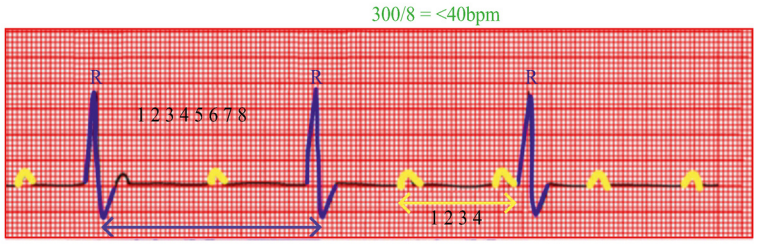
Q2.6: What are the site of conduction defects in Mobitz I vs Mobitz II?

Answer:

- In Mobitz Type I heart block
  - Problem is in the AV node, and it fails to conduct: Intranodal conduction defect
  - Therefore, after P, there is no QRS complex
- In Type II heart block
  - Problem is in the Bundle of HIS below the AV node: Infranodal conduction defect
- When there is damage to the left or right fascicle of the main central bundle of His it is called Bundle branch block (BBB)

Q2.7: What are the ECG and clinical features of third-degree heart block (Stokes-Adams syndrome)?

Answer:



- R-R interval here is increased
- Heart rate < 40 bpm
- QRS complex is relatively broad
- A:V dissociation: No. of R waves and P waves do not match
- Stokes-Adams syndrome leads to a hampered quality of life
- Dual chamber/dual lead pacemaker is used on a priority basis
- Patients could have 'a' wave in JVP or frog waves in the JVP
- No conduction via AV node

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BIG QUESTION 3: What are the essential ECG patterns, their causes, and associated clinical correlations?

Broad Answer: ECG interpretation involves recognizing changes in wave morphology, intervals, and segments that correlate with specific cardiac, metabolic, and systemic conditions. Key patterns include P wave abnormalities, PR interval changes, pathological Q waves, QRS morphology variations, ST-T changes, and QT interval alterations.

DETAILED QUESTIONS

Q3.1: What are the important P wave abnormalities and their causes?

Answer:

P. mitrale	• Duration > 120 msec, or it might have a notch in between • Increased duration is due to enlargement of left atrium • Example: Mitral stenosis • Since the orifice here is narrow, the left atrium will dilate; therefore, current will take more time to go across dilated left atria
P. Pulmonale	• Increase in vertical height of P wave (height >2.5 mm while evaluating in the limb leads of the patient) • Occurs in the Pulmonary artery hypertension • Height of the P wave is >2.5 mm in case of Pseudo-P-pulmonale occurring in the absence of PAH due to hypokalemia
Absent P wave	Atrial fibrillation Hyperkalemia Sick Sinus Syndrome
Varying amplitude of P wave and tachycardia	• COPD • Multifocal atrial tachycardia (MAT)
Retrograde P wave	• P wave is always a positive wave; only the lead where it is negative is aVR, and where it is biphasic is V1 • It is on the negative side when the impulses that are depolarising the atria start from AV node (causing retrograde activation of atria) • Seen in Junctional Tachycardia (JT)

Q3.2: What conditions cause short and prolonged PR intervals?

Answer:

- **Short PR:** Wolf Parkinson White syndrome (delta wave, normal PJ), Lown - Ganong - Levine syndrome
  - o In both these syndromes → Accessory pathway and faster conduction
- **Prolonged PR:** First-degree heart block (Aschoff nodules), reduced exercise capacity

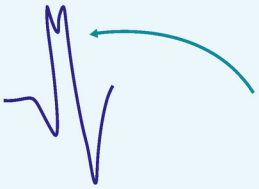
Q3.3: What are the causes of pathological Q waves?

Answer:

- Myocardial infarction
- Hypertrophic obstructive cardiomyopathy (HOCM)

Q3.4: How do QRS morphology and width help in diagnosis?

Answer:

Narrow qRS tachycardia	• Supraventricular tachycardia, like PSVT, Atrial fibrillation, or Atrial flutter
Broad qRS tachycardia	• Monomorphic VT - If the amplitude is equal o Stable BP - Amiodarone o Pulseless VT - Defibrillation right away • Polymorphic VT/ Torsades de pointes o Most of the time, the patient would be pulseless and require defibrillation o If hemodynamically stable and BP > 90/60mmHg - $MgSO_4$
Notching of qRS complex/Rabbit ear appearance 	At the peak of the arc, there could be an appearance like a rabbit's ear In V5 and V6, it could be suggestive of Bundle branch block, which could be either right or left Left bundle branch block is always pathological and is seen with MI Right bundle branch block can be physiological as well
Osborn wave	Encountered in a patient with hypothermia Core temperature - best evaluated in lower esophagus Only history will be of sudden death in the family
Delta wave 	• Encountered in a patient of Wolff Parkinson White syndrome
Epsilon wave 	• Appears like an Osborn wave • Seen in ARVD/cardiomyopathy and contributes to sudden cardiac death • ARVD - Arrhythmogenic right ventricular dysplasia • Fibrofatty deposition (instead of muscle) just below the endocardium/ more towards pericardium • This can lead to enhanced automaticity, resulting in VT or polymorphic VT • Setting for hypothermia, like myxoedema coma, or a soldier brought down from Siachen glacier to base hospital

Q3.5: What are the causes of ST-segment elevation (Mnemonic: ELEVATION AB)?

Answer:

- Electrolyte: Hyperkalemia
  - o Tall tented T wave (if T wave goes up, ST will go up simultaneously)
- Left bundle branch block
- Early repolarisation variant
  - o ST elevation in ECG in a few leads
  - o It will not follow a pattern like in MI, where there is V1-V4 or II, III, aVF
- Ventricular aneurysm seen in MI
  - o In anterior wall or extensive anterior wall MI, structural damage to the heart results in a ventricular aneurysm, which could trigger ventricular tachycardia

- Trauma due to
  - Road traffic accident
  - Punch in the chest
- Ischemia - Myocardial Ischemia
  - A person would have ST elevation convex in the upper direction, called as **Pardee sign**



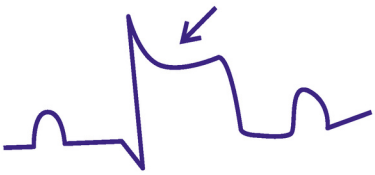
  - Osborn wave - Hypothermia setting because Osborn wave also occurs right at J point



  - In a soldier with hypothermia ECG - P wave might be smaller, the SA node is not firing due to hypothermia, the PR interval might be substantially increased, and a broad qRS complex
  - An abnormal wave different from T wave is picked up on ECG



- Non-occlusive vasospasm - All angina would have ST depression except Prinzmetal angina (PA)
- Acute Pericarditis - ST elevation is concave upward



- Brugada: It is a Sodium channel 5A defect (SCN5A defect)
  - Sudden cardiac death could be occurring
  - ST elevation with T wave inversion is called the COVE pattern



Q3.6: What are the causes of ST depression?

Answer:

- Unstable angina
- NSTEMI
- Hypokalemia
- Digoxin effect

Q3.7: What are the main T-wave abnormalities and their causes?

Answer:

- Tall tented: Hyperkalemia
- Hyperacute: Myocardial Ischaemia, predominantly ST elevation MI
- Inverted: Unstable angina, MI, hypokalemia

Q3.8: What is the clinical significance of a prominent U wave?

Answer: Seen in hypokalemia

Q3.9: How do calcium and magnesium affect the QT interval?

Answer:

- Calcium and magnesium have an inverse relationship with the QT interval
- Tetany - Chvostek sign, Trousseau's sign, and QT prolongation

$$QT_C = \frac{QT}{\sqrt{R-R\ internal}}$$

$$\uparrow QT_C\ corrected$$

- QTC is one of the diagnostic criteria for polymorphic ventricular tachycardia/ Torsades de pointes

Q3.10: What is the "$S_1Q_3T_3$" pattern and its significance?

Answer:

- Deep S in lead I
- Deep Q in lead III
- Inverted T in lead III
- Seen in massive pulmonary embolism

Q3.11: What is the relationship between digoxin toxicity and ECG findings?

Answer:

- Can cause ventricular bigeminy (alternating sinus and PVCs)
- Discontinue digoxin before treadmill testing

BIG QUESTION 4: How are dual-chamber pacemakers used in cardiac disease?

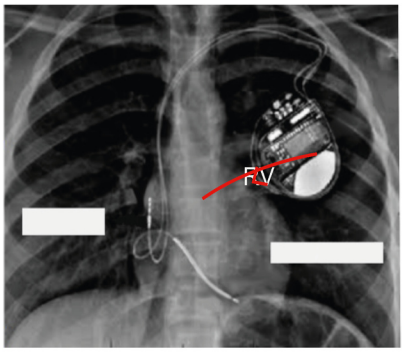
Broad Answer: A dual chamber pacemaker paces both the right atrium and right ventricle, restoring AV synchrony in complete heart block (Stokes-Adams syndrome).

DETAILED QUESTIONS

Q4.1: What is a dual chamber pacemaker, and when is it indicated?

Answer:

- Structure: Two leads -one in right atrium, one in right ventricle
- Function: Controls atrial and ventricular contractions
- Indication: Complete heart block with AV dissociation (Stokes-Adams syndrome)
- Triple chamber pacemaker: Third lead placed in coronary sinus for LV pacing in cardiac resynchronization therapy



Q4.2: What is the acute management of pulmonary edema due to CHF?

Answer:

- Mnemonic: LMNOP
  - L: Lasix (furosemide) - diuresis
  - M: Morphine - reduces preload and anxiety
  - N: NTG - reduces preload and afterload
  - O: Oxygen - improves oxygenation
  - P: Position - propped up to reduce venous return

Q4.3: What is the mainstay of chronic CHF treatment?

Answer:

- A: ACE inhibitors (or ARB) - afterload reduction

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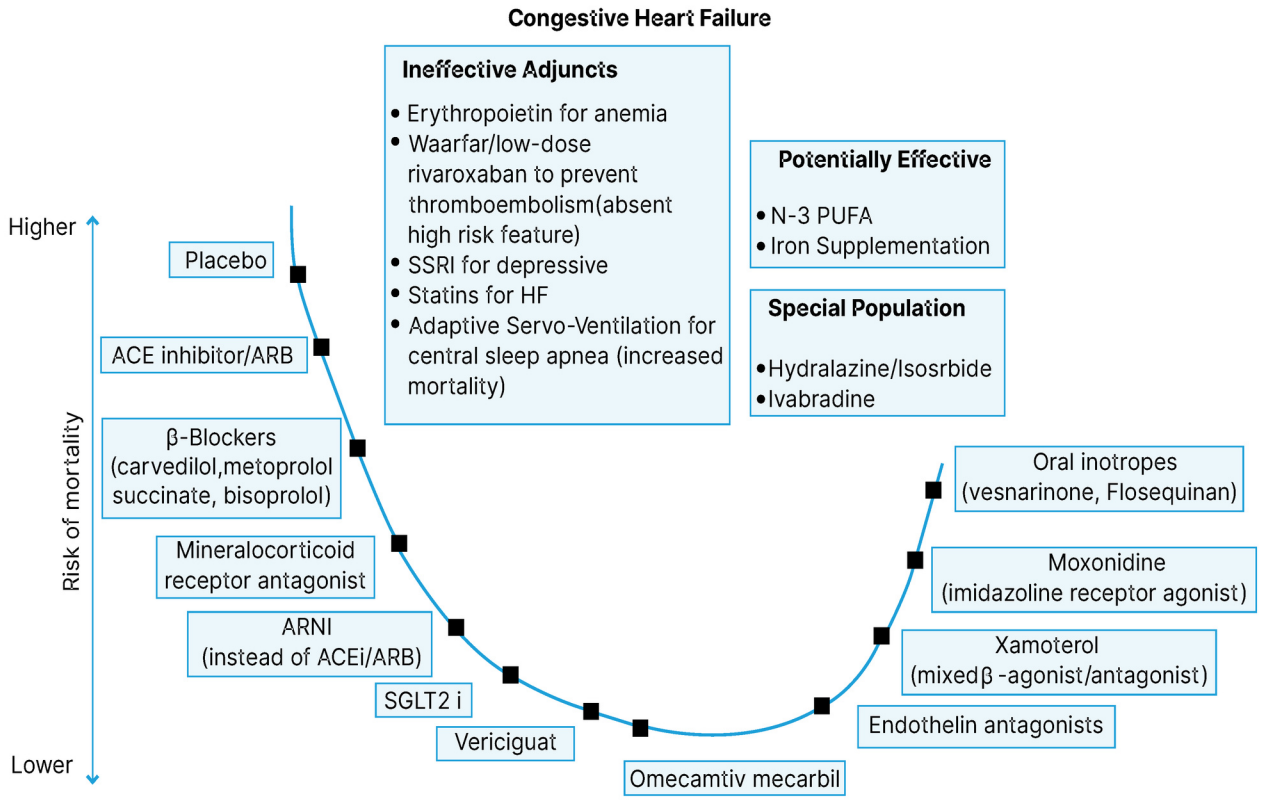
- B: ß-blockers (metoprolol, carvedilol, bisoprolol) - HR control, mortality benefit
- Aldosterone antagonist: Spironolactone - reduces edema, mortality
- Alternatives:
  - ARNI (sacubitril/valsartan) - enhances natriuresis via BNP
  - SGLT2 inhibitors (empagliflozin, canagliflozin) - cardioprotective, mortality reduction, especially in diabetics
  - Vericiguat, omecamtiv mecarbil - newer mortality-reducing agents in select cases



Q4.4: Which of the following statements is wrong about heart failure?

A. Non-cardiovascular mortality in HFrEF is 40% while non-cardiovascular mortality in HFpEF is 15%

B. 5-year death rate is 60%

C. Non-rheumatic atrial fibrillation is the leading cause of embolic stroke

D. ACEI leads to angioedema in 1% and dry cough in 10-15% cases

Answer:

- Non-cardiovascular mortality in HFrEF and HRpEF has been shown to be equal
- Cardiovascular mortality will be higher in heart failure with reduced ejection fraction, as low cardiac output will involve the kidney and lead to cardio-renal syndrome

Q4.5: Which antidiabetic drug is potent in reducing cardiovascular mortality?

Answer: $SGLT_2$ inhibitors like Canagliflozin

Q4.6: What is the best imaging and histopathology-based method to diagnose myocarditis?

Answer:

- Imaging: Cardiac MRI (Lake Louise criteria - 2009 original vs revised for edema + inflammatory injury)
- Histopathology: Dallas criteria - biopsy showing histologic or immunohistologic evidence of inflammation

Q4.7: What are the common causes of myocarditis?

Answer:

- Viral: Parvovirus > Coxsackie B
- COVID-19 delta strain (In pediatrics: Multiple-Inflammatory Syndrome in Children)
- Rheumatic fever (Aschoff nodules affecting endocardium)

Q4.8: How is myocarditis diagnosed?

Answer:

- According to radiology, the best way to diagnose myocarditis is a Cardiac MRI
- Imaging can identify the changes in contractility of the heart and late contrast enhancement.
- Radiological criteria - Lake Louise Criteria - For cardiac MRI
- Histopathological criteria - Dallas Criteria are used
- "**Definite myocarditis** is diagnosed when there is histologic or immunohistologic evidence of inflammation on endomyocardial biopsy and does not require any other laboratory or clinical criteria." Magnetic resonance imaging is increasingly employed early in the evaluation for possible myocarditis. With the original 2009 Lake Louise criteria for myocarditis, a positive study required any two of three findings: Abnormal T2- T2-weighted imaging or early or late gadolinium enhancement. Revised criteria for specificity require both a T2-weighted criterion indicating edema and one T1-based criterion consistent with inflammatory injury, although more liberal diagnostic criteria allowing for the presence of either one yield higher sensitivity. The presence of pericardial effusion supports the diagnosis of inflammation, although it is not specific.

BIG QUESTION 5: How do pericardial diseases differ in pathophysiology, ECG findings, clinical features, and exam relevance?

Broad Answer: Normal pericardial fluid is 20-50 ml; more is pericardial effusion. When intrapericardial pressure exceeds LV end-diastolic pressure cardiac tamponade. Low-pressure tamponade has a weak but present pulse, no pulsus paradoxus; acute tamponade has an absent pulse on inspiration and pulsus paradoxus > 12 mm Hg. Pericarditis shows diffuse concave ST elevation + PR depression; pericardial effusion shows electrical alternans and low voltage; constrictive pericarditis has no fluid but calcification, pericardial knock, and Kussmaul sign.

DETAILED QUESTIONS

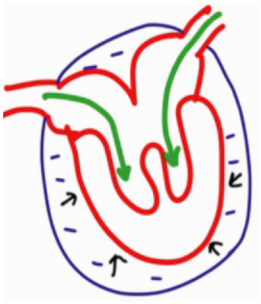
Q5.1: What is the normal volume of pericardial fluid, and what defines pericardial effusion?

Answer:

- Normal: 20-50 ml
- Pericardial effusion: Fluid > 50 ml in the pericardial sac

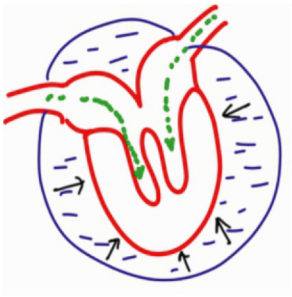
Q5.2: What is cardiac tamponade, and how does it differ from low-pressure tamponade?

Answer:



Low-pressure cardiac tamponade

- The amount of fluid has increased and puts pressure on the heart from the outside
- Since the pressure is limited, it does not hamper the filling of ventricles
- Pulse is still appreciated but it is weak
- **Pulsus Paradoxsus - absent**



Cardiac tamponade

- The amount of fluid increases in a short period of time and pressure on the heart increases dramatically
- The input into the ventricles decreases and the output is also hampered
- Pulse - disappear in the phase of inspiration
- **Pulsus Paradoxsus - present**
- Clinical presentations:
  - Driver without seatbelt slamming the chest on the steering wheel
  - Patients of tuberculosis who have not taken treatment
- Rx: **Urgent Echocardiography guided pericardiocentesis with Xiphisternal approach**

Q5.3: What is pulsus paradoxus, and how is it measured?

Answer:

- Definition: Fall in systolic BP > 12 mm Hg during inspiration
- Measurement:
  - Patient breathes normally
  - Record SBP during inspiration and expiration
  - Calculate difference (> 12 mm Hg = pulsus paradoxus)
- Seen in:
  - Cardiac tamponade
  - Pregnancy

Q5.4: What is Beck's triad in cardiac tamponade?

Answer:

- Hypotension (obstructive shock)
- Muffled heart sounds
- Congested neck veins
  - Non-pulsatile elevation of JVP is seen in cardiac tamponade

Q5.5: How do pericarditis, pericardial effusion, tamponade, and constrictive pericarditis differ?

Pericarditis	Pericardial effusion	Cardiac tamponade	Constrictive pericarditis
• ST segment elevation on ECG • It will be concave in an upward direction • PR segment depression	• Electrical alternans • Variation in amplitude of qRS complex but no tachycardia • Low voltage complex • Causes - TB, malignancy, kidney failure, uremic pericardial effusion	• Electrical alternans, severity will increase • Presence of pulsus paradoxsus (not present in pericardial effusion) • i pulse absent i.e., inspiratory disappearance of pulse of the patient	• No Electrical alternans because there is calcification outside the heart, there is no fluid • Low voltage ECG & pulsus paradoxsus • Auscultatory finding - Pericardial shudder/knock/shock • Kussmaul sign: Paradoxical rise of JVP • Usually seen after cases where a person had tubercular pericarditis & received ATT • Diagnosis: CT chest, Cardiac MRI

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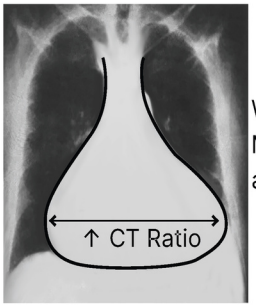
Q5.6: What is the Kussmaul sign, and where is it seen?

Answer:

- Definition: Paradoxical rise in JVP during inspiration
- Seen in:
  - Constrictive pericarditis
  - Restrictive cardiomyopathy
  - Right heart failure (e.g., RV infarct, PE, cor pulmonale)
- Not seen in: Cardiac tamponade

Q5.7: What are the classical radiologic signs in large pericardial effusion?

Answer: Chest X-ray: **"Water bottle" or "money bag"** heart silhouette



Water bottle/
Money bag
appearance

BIG QUESTION 6: How do hypertrophic, dilated, and restrictive cardiomyopathies differ in cause, presentation, diagnosis, and exam features?

Broad Answer: Hypertrophic cardiomyopathy (HCM) is the most common cardiomyopathy (1:500 incidence), usually autosomal dominant, commonly caused by a MYH7 mutation, and can cause sudden death in young athletes via ischemic VF. DCM is a flabby heart with systolic dysfunction; RCM is a stiff heart with diastolic dysfunction due to amyloid, sarcoid, radiation fibrosis, or endomyocardial fibroelastosis, often showing the Kussmaul sign.

DETAILED QUESTIONS

Q6.1: What is the incidence and most common type of cardiomyopathy?

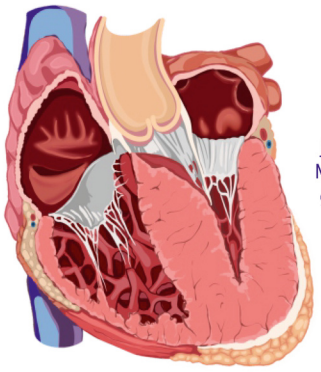
Answer:

- HCM: Incidence ≈ 1:500
- Most common cardiomyopathy overall: Hypertrophic (especially HOCM subtype)

Q6.2: Which gene is most often mutated in HCM, and what is the inheritance?

Answer:

- Gene: **MYH7** (β-myosin heavy chain)
- Inheritance: Autosomal dominant



HCM
MYH7
gene

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Q6.3: What is the mechanism of sudden death in HCM?

Answer: Ischemic ventricular fibrillation from myocardial ischemia + arrhythmogenic substrate in hypertrophied myocardium

Q6.4: How do you differentiate causes of sudden death in athletes?

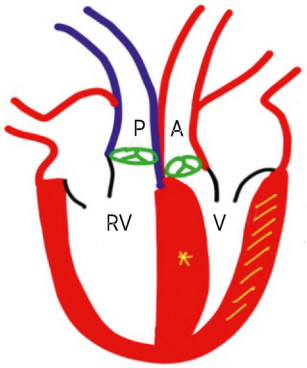
Answer:

Condition	Mechanism	Features
Athlete's heart	Physiologic LVH ± dilated great vessels → coronary kinking	Extreme training, enlarged heart
Commotio cordis	Blunt chest trauma during vulnerable T-wave phase	Often martial arts/baseball punch
ARVD	Fibrofatty infiltration of RV → arrhythmia	RV failure/VT ECG: Epsilon wave
HCM	LVH ± outflow obstruction → VF	Septal hypertrophy, murmur ↑ with standing ECG: LVH pattern

Q6.5: What are the cardinal echo & anatomic features of HCM?

Answer:

- Primary defect - Diastolic malfunction
- Septal hypertrophy (LV free wall ~3.0 cm)
- Banana-shaped LV cavity
- SAM (Systolic Anterior Motion of mitral valve)
- Subvalvular aortic stenosis
- Mitral regurgitation in some cases



Q6.6: What are the key physical signs in HCM?

Answer:

- Pulse: Pulsus bisfiriens (jerky upstroke), double apical impulse (LVH + LAH)
- Heart sounds: S4 (atrial gallop)
- Murmur: Ejection systolic murmur ↑ with standing, Valsalva, amyl nitrate (↓ preload → ↑ obstruction)

Q6.7: What is the investigation of choice and special echo finding in HCM?

Answer:

- IOC: Transesophageal echocardiography
- Special finding: SAM of the mitral valve

Q6.8: What is the treatment of HCM?

Answer:

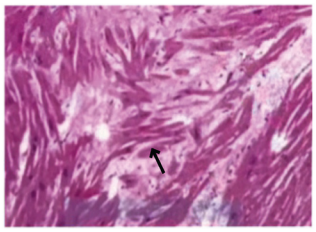
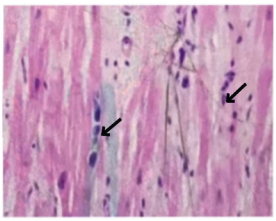
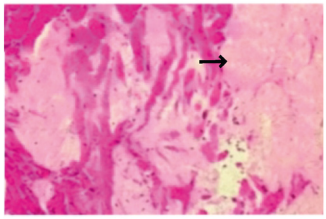
- First-line: Propranolol (nonselective ß-blocker)
- Antiarrhythmic: Disopyramide
- TOC for high-risk/symptomatic: ICD
- Surgical: Alcohol septal ablation/myomectomy
- Avoid: Digoxin, nitrates, ACE inhibitors, calcium channel blockers (↓ preload → ↑ obstruction)

Q6.9: How do murmurs change with maneuvers in HCM compared to most other murmurs?

Answer:

- Most murmurs ↓ with standing/Valsalva/amyl nitrate
- Exception: HOCM murmur ↑ with these maneuvers (↓ LV volume → ↑ turbulence)
- MVP murmur also ↑/lengthens with these maneuvers

Q6.10: How do HCM, DCM, and RCM differ histologically and clinically?

Hypertrophic Cardiomyopathy (HCM)	Dilated Cardiomyopathy (DCM)	Restrictive Cardiomyopathy (RCM)
		
• Disarray of arrangement of myocytes or whorled pattern.	• Damage to myocytes manifests as a fragmented nucleus so multinucleated myocytes are present.	• Rare type of cardiomyopathy • Pink amyloid material which is present in between the cardiac myocytes makes the heart wall stiffer & therefore output from the heart will be lesser
• Special pulse - Pulsus bisfiriens (Jerky pulse)	• Special pulse-Dicrotic pulse o Twice beating pulse felt in both systole & diastole	• Cause: o Amyloidosis o Sarcoidosis o Radiation to chest o Endomyocardial fibroelastosis - causes fibrosis to the conduction system and damage to myocytes resulting in a stiffer heart • Kussmaul sign is seen
• Clinical features: o Ejection systolic murmur o S4 +	• Clinical features: o Functional tricuspid/mitral regurgitation o Pansystolic murmur	• Clinical features: o S4 +

Q6.11 How do you differentiate between Constrictive pericarditis & Restrictive Cardiomyopathy?

Answer:

- Endomyocardial biopsy - gold standard
  - o Amyloid deposition in the heart or fibrosis due to radiation
  - o Non-caseating granulomas cause damage to cardiac myocytes.
- Cardiac MRI - an emerging technique
  - o RCM - Fibrosis
  - o DCM - Flabby heart
  - o HCM - Hypertrophy

BIG QUESTION 7: What are the types, clinical features, and causes of abnormal arterial pulses?

Broad Answer: Abnormal arterial pulses can be classified by waveform characteristics (e.g., slow-rising, jerky, collapsing, dicrotic, alternating, irregular) and are often associated with specific cardiovascular pathologies.

DETAILED QUESTIONS

Q7.1: What are the ABCD patterns of pulse and their clinical associations?

Answer:

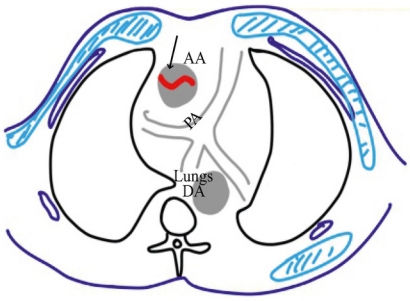
- **A: Anacrotic pulse / Pulsus Tardus et Parvus**
  - Slow-rising, decreased amplitude
  - Seen in aortic stenosis (elderly with calcified valves or pediatric rheumatic origin)
- **B: Pulsus Bisfiriens (Jerky pulse)**
  - Two systolic peaks, slow-rising
  - Seen in Hypertrophic Obstructive Cardiomyopathy (HOCM) due to dynamic LVOT obstruction
- **C: Collapsing pulse / Corrigan's pulse**
  - Bounding upstroke, rapid collapse
  - Seen in aortic regurgitation, accentuated by raising the patient's arm above the head
- **D: Dicrotic pulse**
  - One palpable component in systole, one in diastole
  - Seen in low cardiac output states

Q7.2: What are other notable pulse abnormalities and their significance?

Answer:

- Pulsus alternans: Alternating strong and weak beats: Left ventricular failure
- Irregularly irregular pulse: Atrial fibrillation; often with pulse deficit

Q7.3: What is aortic dissection, and what causes it?



Answer:

- Definition: Intimal tear of the aorta → false lumen creation
- Common cause: Uncontrolled hypertension (most frequent)
- Other causes: Marfan syndrome, cystic medial necrosis
- May propagate retrograde into coronary arteries

Q7.4: How is aortic dissection diagnosed in stable vs unstable patients?

Answer:

- Unstable: Transesophageal echocardiography (TOE)
- Stable: CT chest (tennis-ball appearance) or MRI

Q7.5: How is aortic dissection classified?

Answer: Stanford classification:

Type A	Type B
• Intimal tear in ascending aorta	• Intimal tear in descending aorta
• Rx: Surgery is required	• Rx: o If BP is maintained: short-acting ß-blocker (esmolol) → To control HR and prevent the propagation of the tear o If BP crashes: Surgery is required

YOURWISH

- DeBakey classification:

Type I	Originates in the ascending aorta, propagates at least to the aortic arch and often beyond it distantly
Type II	Originates in and as confined to the ascending aorta.
Type III	Originates in the descending aorta and extends distally down the aorta or, rarely retrograde into the aortic arch and ascending aorta
Stanford	
Type A	All dissections involving the ascending aorta, regardless of the site of origin
Type B	All dissections not involving the ascending aorta

BIG QUESTION 8: How is infective endocarditis diagnosed, what are its major microbiological patterns, and when is prophylaxis indicated?

Broad Answer: Infective endocarditis (IE) is diagnosed using the Modified Duke Criteria (major + minor criteria). Causative organisms vary depending on presentation (acute, subacute, IV drug user, prosthetic valve). Prophylaxis is indicated for certain high-risk cardiac lesions before dental procedures, but not for low-pressure shunts like ASD.

DETAILED QUESTIONS

Q8.1: What are the Modified Duke Criteria for IE?

Answer:

- **Major criteria:**
  - Positive blood culture with typical IE organisms (Viridans strep, Strep bovis, HACEK, Staph aureus, Enterococci without other focus) from ≥2 separate cultures; persistent positivity (2 drawn >12 h apart or majority of ≥4 with 1 h gap); or single positive for Coxiella burnetii / high IgG phase I titer (>1:800).
  - Evidence of endocardial involvement:
  - Echo: Vegetation, abscess, prosthetic valve dehiscence
  - New valvular regurgitation (change in murmur alone not enough)
- **Minor criteria:**
  - Predisposition (heart disease or IV drug use)
  - Fever >38 °C
  - Vascular phenomena (emboli, septic pulmonary infarcts, mycotic aneurysm, intracranial hemorrhage, conjunctival hemorrhage, Janeway lesions)
  - Immunologic phenomena (GN, Osler nodes, Roth's spots, rheumatoid factor)
  - Positive blood culture not meeting major criteria
- **Diagnosis:**
  - Definite IE: 2 major OR 1 major + 3 minor OR 5 minor
  - Possible IE: 1 major + 1 minor OR 3 minor

Q8.2: What are the common causative organisms in different scenarios?

Answer:

- Acute bacterial IE: Staph aureus, Streptococcus (community-acquired)
- Subacute bacterial IE: Strep viridans
- IV drug abuser (right-sided IE): Staph aureus
- Prosthetic valve IE: Coagulase-negative Staphylococcus

YOURWISH

Q8.3: How should blood cultures be taken in suspected IE?

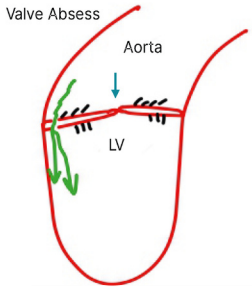
Answer:

- At least 3-4 cultures, 1-2 h apart, before starting antibiotics
- Timing gap helps prove the heart is the bacterial source
- Coxiella burnetii (Q fever) may show only 1 positive culture out of 3

Q8.4: What are the echocardiographic findings in IE progression?

Answer:

- Vegetations → Valve Abscess → Leaflet Perforation → Regurgitation → New Murmur



Q8.5: When is antibiotic prophylaxis indicated for dental procedures?

Answer:

- Indicated in:
  - o Prosthetic heart valve
  - o LV assist device
  - o Prior endocarditis
  - o Certain congenital heart diseases (unrepaired, repaired <6 months, or repaired with residual defects)
  - o Transcatheter pulmonic valve
  - o Valvulopathy after cardiac transplantation
- Not indicated in:
  - o Low-pressure shunts like ASD (LA-RA gradient ~4 mmHg)
    - → Example: Ampicillin 1 h before procedure in indicated cases

Q8.6: A 25-year-old patient with dental caries is undergoing dental extraction. Which of the following does not need prophylaxis for endocarditis?

A. Previous h/o endocarditis
B. Artificial heart valve
C. Untreated cyanotic heart disease
D. ASD

Answer: D

- ASD: Low-pressure shunt
- It operates at a pressure difference of only 4mmHg, LA-RA = 4mm Hg
- So antibiotic prophylaxis would not be required

BIG QUESTION 9: What are the causes, diagnostic criteria, manifestations, and management of Rheumatic Fever?

Broad Answer: Rheumatic fever is a type II hypersensitivity reaction caused by group A β-hemolytic streptococcus. Diagnosis is based on the 2015 Modified Jones criteria. Management involves aspirin for arthritis, steroids for refractory inflammation or pericarditis, and long-term benzathine penicillin for secondary prophylaxis.

DETAILED QUESTIONS

Q9.1: What is the causative organism and type of hypersensitivity reaction in Rheumatic Fever?

Answer:

- Caused by group A β-hemolytic streptococcus
- Type II hypersensitivity reaction

Q9.2: What are the 2015 Modified Jones criteria for diagnosis in low- and high-risk populations?

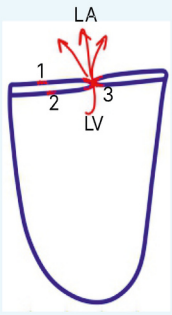
Answer:

A. For all patient populations with Evidence of Preceding Group A Streptococcal infection	
Diagnosis: Initial ARF	2 major manifestations or 1 major plus 2 minor manifestations
Diagnosis: Recurrent ARF	2 major or 1 major and 2 minor or 3 minor
B. Major Criteria	
Low-risk populations	Moderate - and high-risk population
Carditis: Clinical and/or subclinical	Carditis: Clinical and/or sub-clinical
Arthritis • Polyarthritis only	Arthritis • **Monoarthritis or polyarthritis** • Polyarthralgia
Chorea	Chorea
Erythema marginatum	Erythema marginatum
SC nodules	SC nodules
C. Minor Criteria	
Low-risk populations	Moderate - and high-risk population
Polyarthralgia	Monoarthralgia
Fever (≥38.5°C)	Fever (≥38°C)
ESR ≥60 mm in the first hour and/or CRP ≥3.0 mg/dL	ESR ≥30 mm/h and/or CRP ≥3.0 mg/dL
Prolonged PR interval, after accounting for age variability (unless carditis is a major criterion)	Prolonged PR interval, after accounting for age variability (unless carditis is a major criterion)

Q9.3: Which cardiac findings are characteristic of RHD?

Answer:

Pericarditis	• Aschoff nodule causes irritation of the outermost surface of the heart, leading to Chest pain
	• Auscultatory finding - Pericardial friction rub
	• Associated with an ECG finding of ST elevation concave in an upward direction
	• Differentiating between pericardial friction rub and pleural rub
	  o Ask the patient to take a deep breath & hold it
	  o Auscultatory finding disappearing on holding breath - Pleural rub
	• Sound is persisting despite holding breath - Pericardial friction Rub
	• Child with chest pain - Managed by steroids

Myocarditis	• More severe manifestations • Aschoff nodule is causing inflammatory changes in the muscles of the heart, leading to the development of heart failure • Heart failure contributes to the dilation of the mitral valve/tricuspid valve annulus, resulting in pulmonary edema. • In the early phase of the disease, MR can occur without actual damage to the mitral valve leaflet because there is dilation of the mitral annulus due to myocarditis, where the heart is dilated
Endocarditis 	• Commonest valvular lesion - Endocarditis • In adult patients - Mitral stenosis • In a child, Mitral regurgitation (mitral stenosis takes time to develop) • Commissural end involvement occurs in mitral valve cardiac involvement • Aschoff nodule could be present on the surface of the heart valve (superior surface or inferior surface) • It is present in higher concentration in the area where valve cusps are touching each other. i.e., commissural end involvement in rheumatic heart disease • There could be a leakage of blood from the left ventricle to the left atrium during systole, resulting in the development of a murmur

- Characteristic murmur of Rheumatic heart disease - Carey Coombs murmur
  - o The amount of blood leaking from the LV to the LA is much less
  - o Once blood becomes substantial in the lumen of the LA, the LA will dilate, and this blood now comes back to the LV. This is a flow murmur or diastolic murmur
- Mitral valve - Most common valve involved
- Pulmonic valve involvement is rare

Q9.4: What are the key features of arthritis in Rheumatic Fever?

Answer:

- Traditionally, migratory polyarthritis (large joints)
- In high-risk populations (e.g., India), monoarthritis is also diagnostic
- Arthritis is an early feature (2-3 weeks post-sore throat)
- Pain may occur without redness/swelling

Q9.5: What are the neurological and skin manifestations of Rheumatic Fever?

Answer:

- Sydenham chorea: Late feature, antibodies attack the caudate nucleus → fast distal involuntary movements
- Erythema marginatum: Rare in the Indian population due to skin color
- Subcutaneous nodules: Found on extensor surfaces

Q9.6: How is Sydenham chorea managed?

Answer:

- Usually self-limiting in 3-6 months
- If disabling: Valproate or Haloperidol (side effects possible)
- Steroids if drugs fail; IVIG if refractory

YOURWISH

Q9.7: What is the role of aspirin and steroids in Rheumatic Fever?

Answer:

- Aspirin: For arthritis; avoid in viral illness to prevent Reye syndrome
- Steroids: For arthritis unresponsive to aspirin, or pericarditis; avoid in pulmonary edema

Q9.8: How is secondary prophylaxis done for Rheumatic Fever?

Answer:

- Benzathine Penicillin G (test dose before administration)
- ≥27 kg: 1.2 MU IM every 3 weeks
- <27 kg: 0.6 MU IM every 3 weeks
- Duration:
- RHD with MR: Until 40 yrs or 10 yrs from onset (whichever longer)
- Rheumatic carditis: Until 21 yrs or 10 yrs from onset
- RF without carditis: Until 21 yrs or 5 yrs from onset

Q9.9: A 11-year-old child with manifestations of rheumatic fever is prescribed 0.6 MU i.m. Benzathine penicillin. Which of the following is correct for secondary prevention?

A. Lifelong every 3 weeks
B. Only once a single dose
C. Till 5 years, 3 weekly
D. Till 10 years, 3 weekly

Answer: **C** Till 5 years, 3 weekly

- Till 5 years, 3 weekly (21 years of age or 5 years from onset, whichever is longer, for RF without carditis)

BIG QUESTION 10: How is congenital heart disease diagnosed, and what are its key features?

Broad Answer: Congenital heart disease (CHD) can be diagnosed using the NADAS criteria, requiring either 2 major or 1 major + 2 minor features. Major signs include cyanosis, high-grade murmurs, diastolic murmurs, and abnormal second heart sounds. CHD can present with abnormal ECG patterns, characteristic chest X-ray appearances, or abnormal blood pressure patterns.

DETAILED QUESTIONS

Q10.1: What are the NADAS criteria for diagnosis of congenital heart disease?

Answer:

Major criteria	Minor criteria
Systolic murmur grade 3 or more • Tetralogy of Fallot - Ejection systolic murmur • VSD - Pan systolic murmur	Systolic murmur less than grade 3 (intensity would be lesser)
Diastolic murmur • ASD - Mid diastolic murmur	Abnormal second sound
Cyanosis • In right to left shunt - there could be central cyanosis and Tet spells	Abnormal ECG

YOURWISH

CHF • In left to right shunt - due to volume overload	Abnormal Chest X ray
	Abnormal B.P.
For diagnosis of CHD: 2 Major or 1 Major + 2 Minor	

Q10.2: What are ECG, BP, and CXR findings suggestive of CHD?

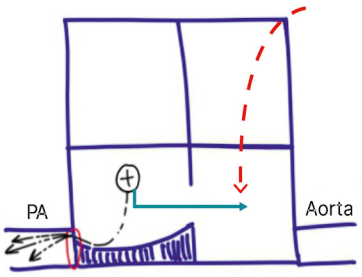
Answer:

- ECG:
  - o Chamber enlargement
  - o Ostium secundum ASD → right axis deviation
  - o Ostium primum ASD → left axis deviation (due to cleft mitral valve → LA enlargement)
- Chest X-ray:
  - o Boot-shaped heart (TOF)
  - o Egg-on-side (TGA)
  - o Figure of 8/ Snowman's heart (supracardiac TAPVC)
- Abnormal blood pressure
  - o Abnormal B.P. values are valid for a defect in the aorta - coarctation of aorta
    - → In coarctation of aorta, as less blood reaches the legs, the BP of the lower extremities will be relatively less
    - → In contrast, the BP in the upper limbs will be elevated

Q10.3: What are the four defects in Tetralogy of Fallot, and which one determines cyanosis?

Answer:

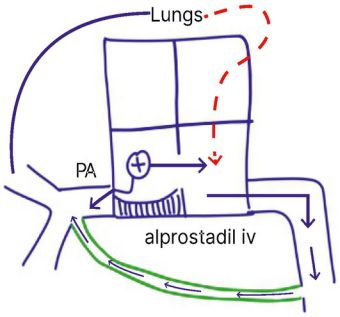
- Ventricular septal defect (VSD)
- Subpulmonic stenosis - major determinant of cyanosis
- Overriding of the aorta (receives blood from both ventricles)
- Concentric right ventricular hypertrophy (present from birth)



Q10.4: Why does cyanosis in TOF usually appear after day 7 of life?

Answer:

- Before day 7, the ductus arteriosus allows blood from the aorta to reach the pulmonary artery for oxygenation
- After functional closure by day 7, this extra flow stops → reduced oxygenated blood → cyanosis develops



Q10.5: What is a Tet spell, and how is it recognized?

Answer:

- Hypercyanotic episode in TOF, often during crying
- Child becomes blue or blackish, lips darken
- Murmur becomes softer due to decreased RV output to the lungs
- Cyanosis worsens due to:
- Increased R→L shunting
- Reduced pulmonary blood flow

Q10.6: How are Tet spells managed acutely?

Answer:

- First step: Knee-chest position (supine or prone with knees flexed to chest)
- Drugs:
  - Propranolol (non-selective ß-blocker)
  - Vasopressors (reduce R→L shunt)
  - Morphine

Q10.7: What are the definitive and palliative treatments for TOF?

Answer:

- Neonates: Alprostadil - keeps the ductus arteriosus patent, preventing early cyanosis
- Palliative surgery: Blalock-Taussig shunt - subclavian artery connected to pulmonary artery to increase pulmonary blood flow
- Definitive correction: Done after 2 years of age

Q10.8: What is Pentalogy of Fallot?

Answer: TOF defects plus an ASD

Q10.9: Which conditions present with central cyanosis on day 0?

Answer:

- Day 0 cyanosis: Transposition of great arteries (TGA)
- Cyanosis ↑ with crying: Congenital diaphragmatic hernia (Bochdalek)
- Cyanosis ↓ with crying: Choanal atresia

Q10.10: Why is the second heart sound (S2) often described as a "single S2" in Tetralogy of Fallot?

Answer:

- In Tetralogy of Fallot, the pulmonary valve is narrowed due to subpulmonic stenosis, causing reduced blood flow to the lungs.
- As a result, the pulmonary component (P2) of the second heart sound is very faint or absent, making the second heart sound (S2) appear as a single sound (only the aortic component, A2, is heard).

3. CARDIOLOGY PART -3

BIG QUESTION 1: What are the important auscultatory areas, heart sounds, and their clinical significance?

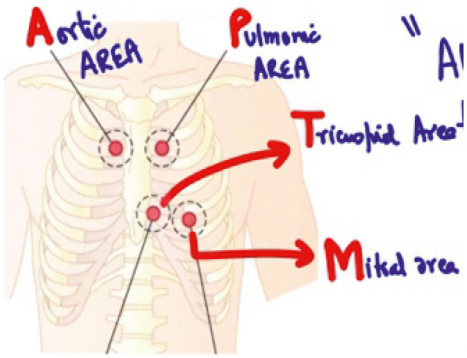
Broad Answer: Cardiac auscultation involves listening at specific anatomical points-Aortic, Pulmonic, Erb's point, Tricuspid, and Mitral areas-to assess heart sounds (S1-S4), splits, clicks, snaps, and murmurs. Variations in heart sounds and splitting patterns can indicate underlying cardiac pathologies such as valve lesions, heart failure, conduction defects, and structural abnormalities.

DETAILED QUESTIONS

Q1.1: What are the different auscultatory areas for the heart?

PYQ: FMGE 2021

Answer:



Aortic area	• A_2 component of the 2nd heart sound • 2nd intercostal space, Parasternal line
Pulmonic area	• P_2 component of the heart sound
Tricuspid area	• M_1 and T_1 components of the heart sound • Parasternal line on left side, 4th-5th intercostal space
Mitral area	• 2 cm medial to the mid-clavicular line • M_1 and T_1 components of the heart sound

Q1.2: What are the normal components of the first and second heart sounds?

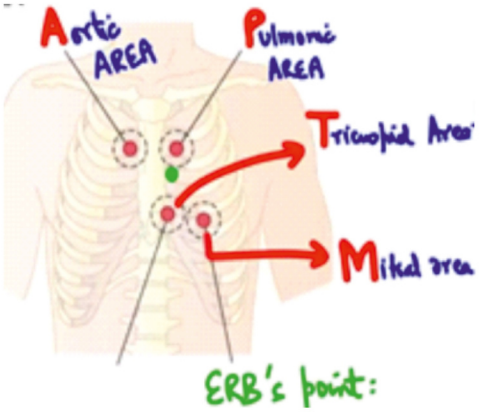
Answer:

- **Mnemonic: "All physicians must take money."**
  - $S^1 \rightarrow M^1$ (Mitral closure), T^1 (Tricuspid closure)
  - $S^2 \rightarrow A^2$ (Aortic closure), P^2 (Pulmonic closure)

Q1.3: What are the key features of the ERB's point?

Answer: The Green point shows the location of Erb's point.

- 3rd Intercostal space parasternally on the left-hand side
- Splitting of S_2 is checked.
- Best heard during inspiration.



Q1.4: What is the splitting of the second heart sound?

Answer: The time lag between the closure of aortic and pulmonic valve, which is about 30 milliseconds, is called the splitting of the second heart sound.

YOURWISH

Q1.5: In what conditions are narrow split S_2 and wide split S_2 heard?

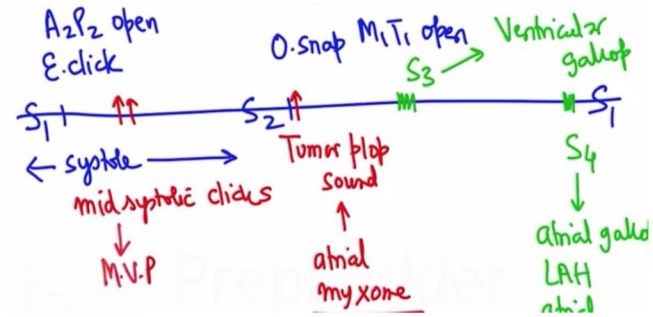
Answer:

- Narrow split S_2 is heard in left ventricular failure.
- Wide split S_2 is heard in right ventricular failure.

Q1.6: Why is the distance between S1 and S2 shorter compared to the distance between S2 and S1?

Answer:

- The distance between S1 and S2 is relatively shorter compared to S2 and S1 because the diastole duration is longer.
  - S1-S2: Systole
  - S2-S1: Diastole



Q1.7: What is the ejection click?

Answer:

- Ejection click is the heart sound heard after S_1 with a phonocardiogram.
- Ejection click means the opening of the aorta (A_2) and the Pulmonary valves (P_2)

Q1.8: Write briefly regarding the opening snap.

Answer:

- The opening snap is heard after S_2, which is due to the mitral (M_1) and tricuspid valve (T_1) opening.
- A loud opening snap is heard in Mitral stenosis.
- Plain opening snap - Not heard by stethoscope.
- Duration between S_1 and S_2 is **systole**.
- Duration between S_2 and opening snap is isovolumetric relaxation followed by rapid ventricular filling

Q1.9: What are the key features of S_3 Heart sound?

Answer:

- During rapid ventricular filling, S3 is heard due to **turbulence**.
- S_3 is also called the Ventricular gallop rhythm
- Usually seen in Congestive heart failure patients

Q1.10: What are the key features of S_4 Heart sound?

Answer:

- S_4, also called **the Atrial gallop, is heard before S_1**
- It is heard due to atrial hypertrophy.
- Uncontrolled, undiagnosed hypertension in a person causes left ventricular hypertrophy (LVH).
- First there is compensatory Left ventricular hypertrophy and then Left atria will become bulky and generate more force causing an atrial kick.
- S_3 and S_4 are low pitched heart sounds heard through the bell of the stethoscope.
- S_1, S_2 , Ejection click and Opening snap are heard through the diaphragm of the stethoscope.

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Q1.11: What are low-pitched heart sounds?

Answer:

- S3
- S4
- Tumor plop sound
- Mitral stenosis murmur- Mid-diastolic murmur

Q1.12: What is Mid systolic click?

Answer:

- Mid systolic click, heard in lacks and loose chordae tendineae condition.
  - Heard between S_1 and S_2
  - Turbulence is created by loose chordae tendineae
  - The condition is known as Floppy valve syndrome or Mitral valve prolapse.

Q1.13: What is the Tumor plop sound?

Answer:

- Tumor plop sound is heard just after the opening snap in conditions like.
  - **Atrial myxoma**
  - A tumor on the left atrium obstructs the mitral valve leaflets and behaves like **mitral stenosis.** This limits the amount of blood going into the left ventricle, causing effort intolerance
  - **Pulmonary edema** and breathlessness worsen in the sitting position

Q1.14: What is the difference between Orthopnea and Platypnea?

Answer: Orthopnea

- Dyspnea worsens with the supine position, seen in Congestive Heart Failure.

Platypnea

- Dyspnea worsens in sitting position
- Physical obstruction of blood flow in left atria (Atrial myxoma)
- Congestion in left atria causes congestion in pulmonary veins
- Increases hydrostatic pressure
- Transudation of fluid into alveoli: Pulmonary edema

Q1.15: Write the key features of Pericardial shock.

Answer:

- It is heard at S_3
- Also known as Pericardial knock or shudder
- A person who underwent Antitubercular treatment for 6 months presents with calcification outside the heart
- Complication of extrapulmonary tuberculosis-constrictive pericarditis
- Due to calcification, the ventricles are non-compliant
- Pericardial shudder is a finding of constrictive pericarditis.

Q1.16: What are the key features of the S_2 Heart sound?

Answer:

- Narrow split

- Left ventricular failure
- Wide split
  - Right ventricular failure
- The normal time lag between A_2 and P_2 is in case of left ventricular failure the aortic valve closes late **30 milliseconds**, reducing the time lag between A_2 and P_2.

Q1.17: What is the difference between a Narrow and a wide split?

PYQ: FMGE 2023 PYQ: INICET 2022,23

Answer:

Narrow split	Wide split

• Left ventricular failure
  o Severe Anemia
  o Beri Beri (wet)
  o Anterior wall MI
• LBBB
• Aortic stenosis
  o Valvular lesion in geriatric patients
• Hypertrophic Obstructive Cardiomyopathy (HCOM)
	Variable split
	• The pulmonary valve closure varies with inspiration
	• Pansystolic murmur
	• VSD (Ventricular septal defect)- Blood from left ventricle to right ventricle
	• MR (Mitral regurgitation) in adults- Blood from left ventricle to left atria
	• RBBB
	Fixed split
	• ASD (Atrial Septal Defect)
	• Holt Oram syndrome
	• Down syndrome
	  o Endocardial cushion defect

Q1.18: What are the conditions in which soft S_1 is heard?

Answer:

- Bradycardia
- Prolonged P-R interval
- Hypothyroidism
- Heart block
- Mitral regurgitation
- Bradycardia

Q1.19: In what conditions is loud S_1 heard?

Answer: Loud S1

- Mitral stenosis
- Tachycardia

S1 is Loud with respect to Tachycardia

- Short P-R interval (<120 millisecond)
- Anemia o Beri Beri
- CHF
- Mitral stenosis: Increased Transvalvular gradient

Q1.20: How does mitral stenosis affect the characteristics of the first heart sound (S_1) and splitting?

Answer:

- In untreated patient with Mitral stenosis elastic recoiling will be lesser due to development of calcification causing a soft S_1

- Due to a smaller opening, it takes more time for the blood to go across, so the mitral valve will close later. The time lag between M_1 and T_1 reduces, causing a narrow split S_1 to be seen in Mitral stenosis
- If M_1 gets close to T_1 , Single S_1 may be heard
- In CHD, Single S_2 is a feature of Tetralogy of Fallot
- If Mitral stenosis is still untreated, Mitral valve closure will overshoot tricuspid closure
- This contributes to Reverse splitting of S_1 i.e. T_1 and M_1.

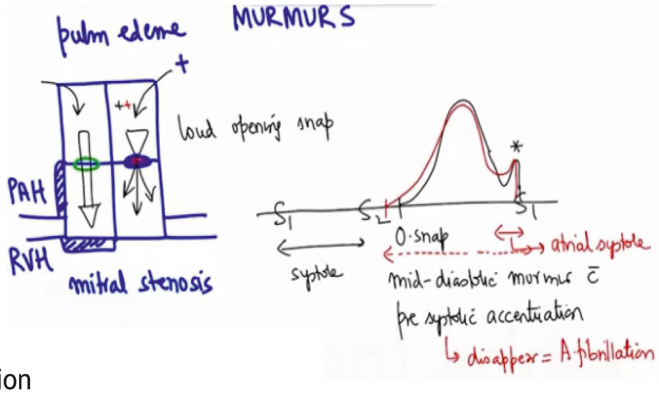
BIG QUESTION 2: What are the key features, types, and clinical associations of cardiac murmurs, along with their distinguishing characteristics?

Broad Answer: Cardiac murmurs are abnormal heart sounds produced by turbulent blood flow through the heart or great vessels. They are classified into diastolic, systolic, and continuous murmurs, each with characteristic timing, pitch, causes, and clinical associations. Examples include the low-pitched mid-diastolic murmur of mitral stenosis, the pansystolic murmur of mitral regurgitation, the crescendo-decrescendo ejection systolic murmur of aortic stenosis, and the machinery-like continuous murmur of patent ductus arteriosus. Murmur severity can be determined by specific clinical signs, and certain congenital and acquired diseases show characteristic murmur patterns.

DETAILED QUESTIONS

Q2.1: Describe the characteristic murmur and auscultatory findings of mitral stenosis.

Answer:



- Loud opening snap: Caused by high left atrial pressure forcing open the mitral and tricuspid valves
- Diastolic murmur begins with the opening snap
- **Pre-systolic accentuation** (small spike before S_1) is present if atrial systole is intact (disappears in atrial fibrillation)
- Low pitch, best heard with the bell of the stethoscope
- Mid-diastolic murmur (MDM) with pre-systolic accentuation
- Smaller valve opening → earlier opening snap, longer murmur
- **Severity determined by duration of murmur**
- Associated with pulmonary artery hypertension

Q2.2: The severity of Mitral stenosis is determined in:

A. Intensity of murmur

B. Loud P2

C. Loud S1

D. Duration or length of murmur

Answer: D. Duration or length of murmur

- The murmur of mitral stenosis is a low-pitched, rumbling sound that occurs during diastole (the period when the heart relaxes and fills with blood).

Q2.3: Which cardiac event corresponds with the terminal part of diastole?

Answer: The terminal part of diastole corresponds with atrial systole.

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Q2.4: What happens to pre-systolic accentuation in atrial fibrillation?

Answer:

- Pre-systolic accentuation disappears in the absence of atrial systole.
- In atrial fibrillation, there is no secondary spike since atrial twitching does not generate enough force.

Q2.5: How do Mitral stenosis patients develop Pulmonary Artery Hypertension?

Answer:

Back pressure changes
↓
Congestion in pulmonary veins causes pulmonary edema.
↓
Hypoxia causes vasoconstriction in the pulmonary artery.
↓
Increases pulmonary Hypertension.
↓
Hypertrophy of Right ventricle

Q2.6: What is the difference between mid and early diastolic murmur?

Answer:

Mid Diastolic Murmur (Mnemonic: CAM)	Early Diastolic Murmur (Mnemonic: GAP)
• Carey Coombs murmur o Characteristic of Rheumatic etiology o Flow murmur • Austin flint murmur- seen in severe aortic regurgitation • Mitral stenosis	• Noise occur in early part of diastole and disappears • Graham Steel Murmur o Seen in severe PAH o Normal mean pressure in pulmonary artery is 15 mmHg o PAH: >25 mmHg o In severe PAH is the pressure is 4 times the normal pressure leading to a leak of blood from pulmonary artery into right ventricle causing a murmur • Aortic regurgitation, Mild • Pulmonary regurgitation, mild

Q2.7: Name the drugs used in treating PAH.

Answer:

- Bosentan
- Ambrisentan
- PDE-5 Inhibitors: Sildenafil

Q2.8: What are the characteristic features of a Pansystolic murmur?

PYQ: FMGE 2022

Answer:

- Systolic murmur
- Intensity of murmur remains the same throughout the systole.
- Mitral regurgitation
- VSD

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Q2.9: What are the key features of an Ejection systolic murmur?

Answer:

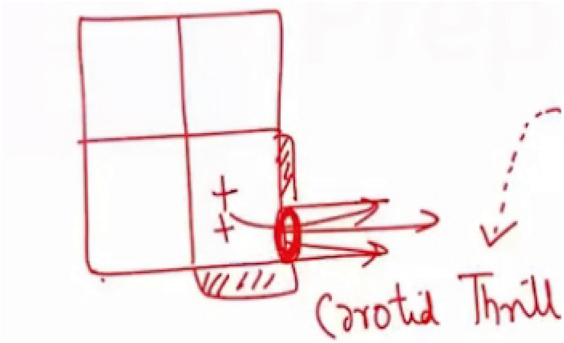
- Intensity of noise increases in the middle and then decreases.
- Crescendo- Decrescendo murmur
- Mnemonic: **PASS**
  - **PS** (Pulmonic stenosis)
  - **AS** (Aortic stenosis)
    - → Age: >65 years
    - → Syncope on exertion
    - → Angina
    - → Dyspnea
    - → Leading cause, senile calcification
  - HOCM

PYQ: FMGE 2022

Q2.10: What are the hemodynamic and clinical findings in aortic stenosis?

Answer:

- In Aortic stenosis, Narrowing increases turbulence.
- Normal orifice of
  - 2 Aortic valve: 2.5 - 4 cm^2
  - 2 Mitral valve: 4-6 cm^2
- Left ventricle generated more force and turbulence
- Carotid thrill, intensity of noise is felt by finger placed on carotid artery



Q2.11: What are the murmur characteristics in Patent Ductus Arteriosus (PDA)?

Answer:

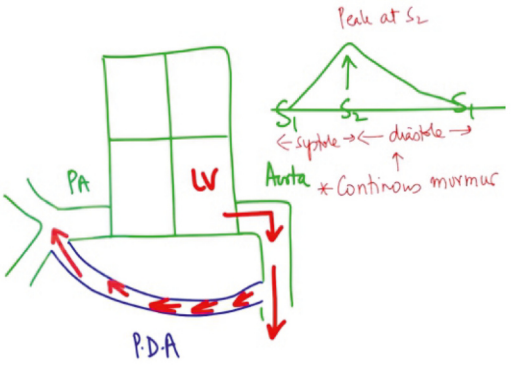
- In PDA, due to the pressure difference, the blood from the left ventricle to the aorta enters into Ductus arteriosus into the pulmonary artery
- Pressure of the aorta is always more than pulmonary artery
- Murmur is heard both in systole and diastole, but the murmur peaks at S_2.

PYQ: FMGE 2021

Q2.12: What are the Conditions presenting with a Continuous murmur?

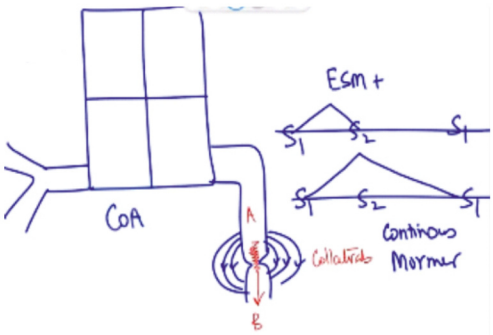
Answer:

- Pediatric disease: Congenital Rubella syndrome (PDA)
- Coarctation of aorta is common in Turner's syndrome
- Peripheral pulmonic stenosis
  - Blood flows continuously in pulmonary artery
  - Narrowing may cause turbulence
- Mammary souffle (Auscultatory finding) due to increased blood supply to the internal thoracic artery.
- Rupture of the sinus of Valsalva - develops communication between the aorta and the Right atrium.



Q2.13: What are the murmur characteristics in coarctation of the aorta?

Answer:



- Usually occurs at the aortic isthmus (beginning of descending aorta).
- Collateral formation → turbulence → systolic murmur.
- Blood travels through collaterals in both systole and diastole.
- Initially: ejection systolic murmur (due to blood flow across the narrowed segment).
- Later: continuous murmur (as collaterals develop).
- Main collateral vessels: intercostal arteries.

Q2.14: How can coarctation of the aorta be clinically identified?

Answer:

- Coarctation of aorta can be easily be identified by:
  - o Radio femoral delay
  - o Blood pressure
    - → Decrease in lower limbs
    - → Increased in upper limbs
    - → Unequal BP between left and right arms
    - → Differential cyanosis

Q2.15: What is the investigation of choice in diagnosing Coarctation of the aorta?

Answer: Investigation of choice for Coarctation of the aorta is MRI of the aorta

Q2.16: Which congenital heart diseases are commonly associated with Down syndrome and Turner's syndrome?

Answer:

- Down syndrome → Atrial Septal Defect (ASD)
- Turner's syndrome →
  - o Bicuspid Aortic Valve (BAV) (most common)
  - o Coarctation of Aorta

Q2.17: Unequal BP between left and right arms is seen in?

Answer:

- Coarctation of the aorta
- Takayasu arteritis
- Aortic dissection

Q2.18: What are the types of coarctation of the aorta seen in pediatrics?

Answer:

- **Pre-ductal variety**
- **Post-ductal variety**

Q2.19: What are the conditions in which large 'a' waves are seen?

Answer: Seen in

- **Seen in Tricuspid stenosis**
- It is not in Mitral stenosis due to the development of PAH

- The right side of the heart should generate more force
- a' wave is increased on long-standing

- Long-standing Mitral stenosis
- **PAH**
  - Eisenmenger complex
    - → Shunt reversal from right to left
    - → The right side of the heart should generate more force, increasing 'a' waves
  - Drugs: Fenfluramine

Q2.20: In what conditions are cannon or giant 'a' waves seen?

PYQ: FMGE 2023

PYQ: INICET 2022,23

Answer:

- Super large a waves
- **Frog sign**
- Due to a lack of coordination between the atria and the ventricles
- Generally, atria should contract first. If atria and ventricles contract together, the power amplified cause Super large a waves
- A-V dissociation
  - Ventricular tachycardia
  - Complete Heart block- Bradyarrhythmia
- Simultaneous contraction causes fluttering waves in neck- Frog sign

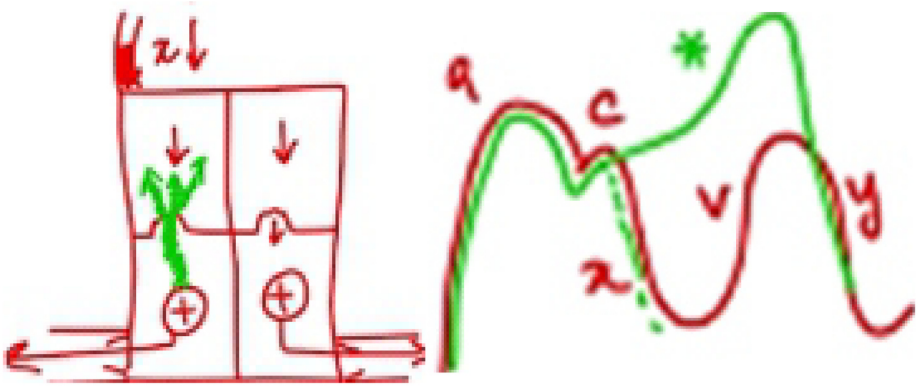
Q2.21: What condition presents with absent 'a' waves?

Answer: Atrial fibrillation

Q2.22: What is the significance of the x descent in JVP, and how is it affected in tricuspid regurgitation?

Answer:

- X descent is a negative vector
  - Atrial relaxation/ventricular systole
- Ventricular systole, the force generated, opens the aortic and pulmonary valves
- Blood races into the aorta and pulmonary artery
- Both valves come back to their normal position
- Downward arrow-ventricles decompress, and mitral and tricuspid valves come back to their normal position
- In tricuspid regurgitation, the opposite vectors cancel each other, giving rise to Absent 'x' descent
- Increased severity of tricuspid regurgitation, x will become absent. o
  - After the 'c' wave, there is no 'x' descent
  - Cv' wave is produced, giving rise Steep 'y' descent



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Q2.23: Absent 'x', 'cv' wave and steep 'y' found in:
Answer. Severe Tricuspid regurgitation

Q2.24: Absent 'y' descent is found in:
Answer: Cardiac tamponade

Q2.25: Blunted 'y' wave is found in:
Answer: Tricuspid stenosis (large 'a' wave is also seen)

Q2.26: Steep 'x', Absent 'y' wave is found in:
Answer: Cardiac tamponade

Q2.27: Steep 'x' and steep 'y' descent is found in:
Answer: Pericardial shudder, constrictive pericarditis

Q2.28: What is the Kussmaul sign?
Answer: Rise in JVP with inspiration

- Constrictive pericarditis due to calcification around heart
- Restrictive cardiomyopathy due to Fibrosis
- Right sided CHF due to reduced compliance of the heart

BIG QUESTION 3: What is metabolic syndrome, and why is it important?

Answer: Metabolic syndrome (Syndrome X) is a cluster of conditions that occur together, significantly increasing your risk of developing serious health problems like heart disease, stroke, and type 2 diabetes. It's often linked to insulin resistance, where your body's cells don't respond effectively to insulin, a hormone that helps regulate blood sugar.

Q3.1: What are the components of Metabolic syndrome?
Answer:

Component	Clinical Cutoff Values
Waist Circumference	≥ 102 cm in men ≥ 88 cm in women
Triglycerides	≥ 150 mg / dL
HDL Cholesterol	< 40 mg/dL in men < 50 mg/dL in women
Blood Pressure (BP)	≥ 130 mmHg Systolic BP or ≥ 85 mmHg diastolic BP
Fasting Glucose	≥ 100 mg/dL
Diagnosis	Any 3 of the 5 features above

Q3.2: What is Coronary Syndrome X, and how is it diagnosed and managed?
Answer:

- Caused by narrowing of perforator vessels myocardial ischemia.
- Epicardial coronary arteries are normal.

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- Clinical presentation mimics chronic stable angina.
- Coronary angiography → gold standard test:
  - Determines % blockage.
  - Normal in Coronary Syndrome X → hence diagnosis of exclusion.
- Treatment:
  - Nitrates → vasodilation
  - Beta blockers

Q3.3: What are the waist circumference cut-offs for Indians in diagnosing central obesity?

Answer :

- **Males:** > 90 cm
- **Females:** > 80 cm

Q3.4: What are the risk factors and recommendations in Metabolic Syndrome?

Answer :

- Centripetal obesity → ↑ incidence of CAD.
- Lipid profile abnormalities:
  - Hypertriglyceridemia
  - Low HDL →↑ risk of CAD & atherosclerosis
- Hypertension cutoff (AHA): ≥ 130/80 mmHg
- Obesity effects:
  - ↑ Peripheral resistance →↑ BP
    → Impaired glucose tolerance
- Recommendations to reduce risk:
  - Aggressive use of statins
  - Control BP
  - Reduce weight

Q3.5: What is Syndrome Z?

Answer: A patient with metabolic syndrome X associated with obstructive sleep apnea is Syndrome-Z

5. NEUROLOGY PART -1

BIG QUESTION 1: How does EEG aid in diagnosing seizures and sleep stages, and what is its role in epilepsy management?

Broad Answer: EEG detects abnormal brain activity using standardized electrode placement. It differentiates seizure types (generalized vs focal) and sleep stages through characteristic waveforms. EEG guides epilepsy treatment, helps distinguish seizures from syncope, and influences drug choice in children, pregnancy, and the elderly.

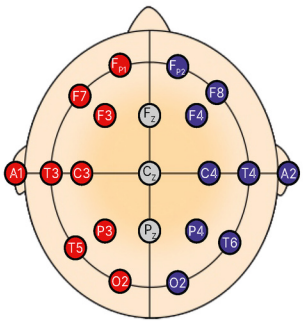
DETAILED QUESTIONS

Q1.1: Who invented the EEG?

Answer: Hans Berger

Q1.2: How are the EEG electrodes numbered, and why is it done?

Answer:



- Electrodes placed on the left side of the head are given odd numbers.
- Electrodes placed on the right side of the head are given even numbers.
- This numbering is important as it helps to identify diffuse disease

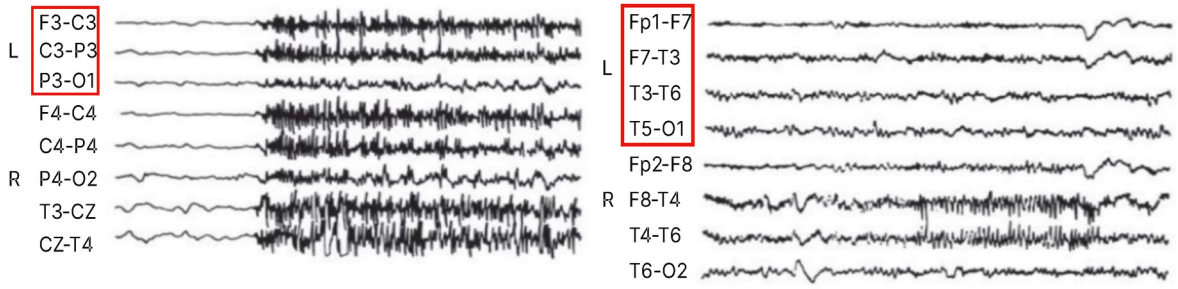
Q1.3: What is an EEG recording called?

Answer: Montage

Q1.4: How can EEG patterns help differentiate between GTCS and focal seizures?

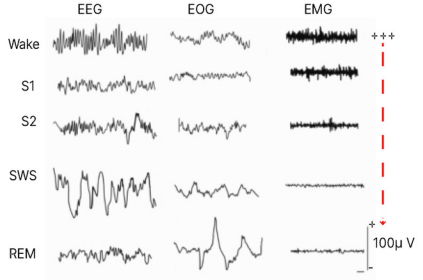
Answer:

- The discharge in the first EEG shows polyspike waves in both the left and right sides of the brain, which is seen in GTCS.
- In the second EEG, the discharge is localized to only a few electrodes on the right side, indicating focal seizures



Q1.5: How do EMG findings differ between wakefulness and sleep?

Answer: In the wake state, the muscles have a particular tone due to spontaneous electrical activity, but as we sleep, the tone of the muscles is reduced and lowest in a state of deep sleep



Q 1.6: How do EOG findings change from wakefulness to sleep?

Answer: At the wake state, the waves are coarse, and as we fall asleep, the waves become finer with a bigger amplitude

Q1.7: What are the characteristic EEG wave patterns and associated EMG and EOG findings during different stages of sleep and wakefulness?

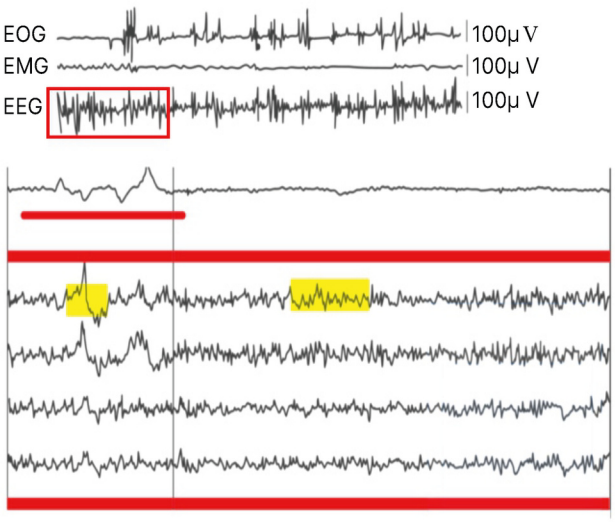
Answer:

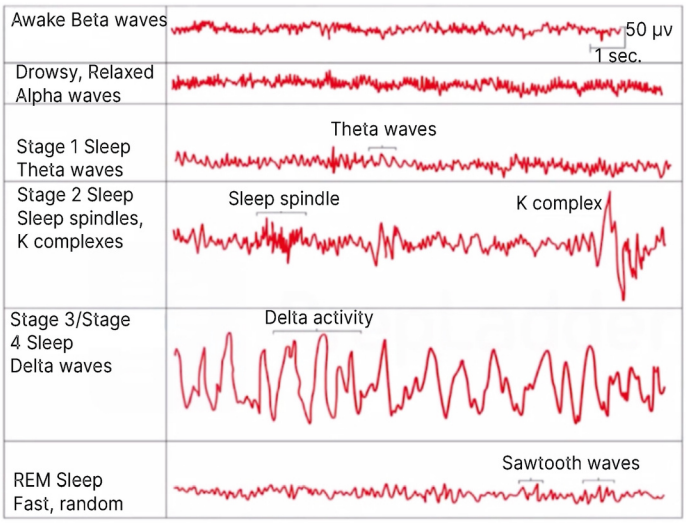
Sleep	Waves	Frequency
AWAKE (Eyes Open)	b wave	8-13 hertz (by beta waves)
NREM 1	q wave	4- 7 hertz (by theta waves)
NREM 2		Sleep spindles (in the middle amplitude is bigger) and K-complexes are seen.
NREM 3 (slow wave sleep)	d waves	0.5-4 hertz (least frequency, but the amplitude of delta waves is the maximum)
REM PHASE	Sawtooth waves	• EMG finding shows minimal activity because muscles are relaxed. • EOG shows a spike due to rapid eye movement. EOG is pointed and fine (not coarse) during REM sleep.

Q1.8: Which phase of sleep is shown in the EEG pattern shown below?

A. REM
B. NREM 1
C. NREM 2
D. NREM 3

Answer: **A.** REM





BIG QUESTION 2: How are seizures, epilepsy, and convulsions defined?

Broad Answer:

- Seizure: Abnormal electrical activity due to some metabolic cause.
- Epilepsy is > 2 unprovoked episodes of seizures.
- Convulsions are motor manifestations of a seizure.

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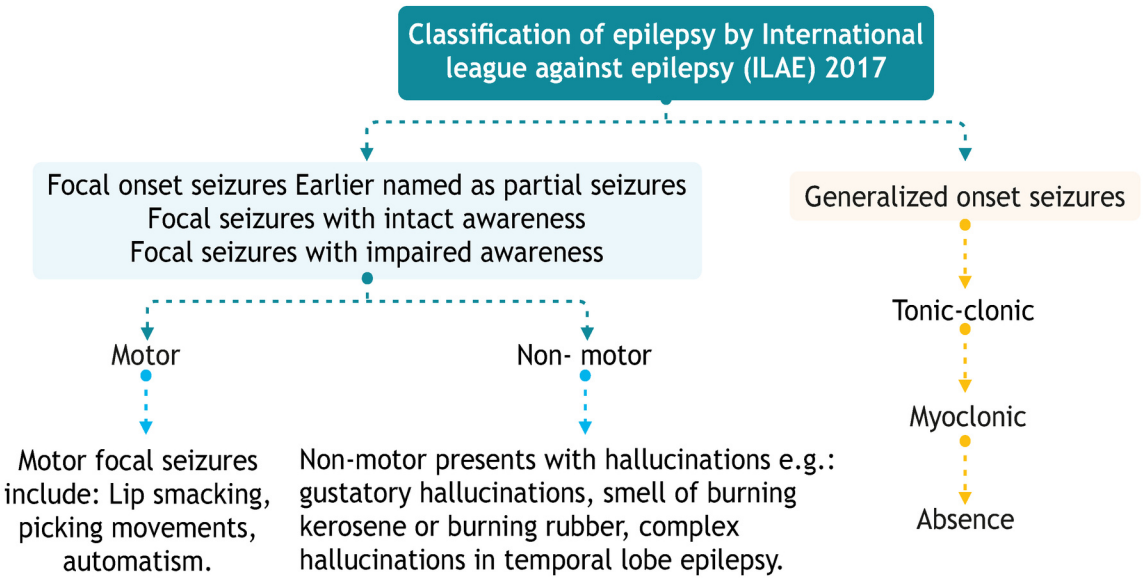
DETAILED QUESTIONS

Q2.1: How does the International League Against Epilepsy (ILAE) 2017 classification categorize different types of seizures?

Answer:



Q2.2: What are the characteristics of the tonic and clonus phases in generalized tonic-clonic seizures (GTCS)?

Answer:

- Tonic phase- Increased tone, ictal cry, and then the patient falls.
- Clonus phase- Initial stiffness followed by jerking movements and intermittent relaxation of the muscles.

Q2.3: What is the recommended approach to managing a patient during GTCS?

Answer:

- Do not restrain the patient during GTCS, as it may cause soft tissue injuries in this patient.
- Most GTCS episodes terminate by themselves within one minute

Q2.4: What is myoclonus?

Answer: Involuntary, sudden, and jerky movement of limbs

Q2.5: What are the key features of absence seizures?

Answer:

- Absence seizures are characterized by vacant staring spells followed by blinking episodes.
- No tonus or clonus will be present.

Q2.6: What is the incidence of seizures and epilepsy in the general population?

Answer:

- 5-10 % of the normal population will have at least one seizure, with the highest incidence in childhood/late adulthood.
- Overall incidence of epilepsy: 0.3-0.5%

Q2.7: What are the main differences between seizures and syncope?

Answer:

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	Seizures	Syncope/vasovagal
Features	AURA (visual blurring) is seen in focal seizures (and migraine), but not seen in GTCS Prodrome is seen in GTCS	Triggered by the sight of blood, or an extreme amount of physical pain
Loss of consciousness	Minutes to hours	Few seconds to minutes.
Tonic clonic movements	30-60 seconds	<15 seconds.
Facial appearance	Perioral cyanosis	Circum-oral paleness
Tongue bite	Sometimes	Rarely
Disorientation	++	+
Urinary incontinence	++	+
Headache	Sometimes	Rarely
Aching muscles	++	-

Q2.8: What happens to serum prolactin levels after a convulsion?

Answer: Serum PROLACTIN level rises after an episode of convulsion.

BIG QUESTION 3: What are the goals in treating epilepsy? Name some of the drugs used.

Broad Answer: The treatment of epilepsy is highly individualized, depending on factors such as the type of seizures, their frequency and severity, the person's age, and overall health. The primary goal is to control seizures and improve quality of life with as few side effects as possible. Some of the drugs include: Lamotrigine, Carbamazepine, Oxcarbazepine, Eslicarbazepine, Phenytoin, Levetiracetam, etc.

PYQ: NEET PG 2021, 22, 23 PYQ: INICET 2020, 21, 22, 23
PYQ: NEET PG 2021, 22, 23 PYQ: FMGE 2019, 20, 21, 22, 23

DETAILED QUESTIONS

Q3.1: What are the first-line drugs used to treat GTCS?

Answer:

- Lamotrigine
- Valproic acid

Q3.2: Name the 1st line drugs used in treating focal seizures.

Answer:

- Lamotrigine
- Carbamazepine
- Oxcarbazepine
- Eslicarbazepine
- Phenytoin
- Levetiracetam

Q3.3: Name the 1st line drugs used in treating typical absence seizures.

Answer:

- Valproic acid
- Ethosuximide
- Lamotrigine

Q3.4: Name the 1st line drugs used in treating atypical absence seizures, myoclonic, and atonic seizures.

Answer:

- Valproic acid
- Lamotrigine
- Topiramate

Q3.5: What are the side effects of and precautions taken while using lamotrigine?

Answer:

- Steven-Johnson syndrome
- Hence, lamotrigine is started at the lowest possible dose. Upon the development of a skin rash, lamotrigine should be stopped immediately

Q3.6: Why is valproate avoided in pregnancy? Name the alternate drug used in pregnancy for treating seizures

Answer:

- Valproate is highly teratogenic (7-20%) and can cause neural tube defects.
- In pregnancy, with GTCS, lamotrigine or levetiracetam are used, provided the patient is not pregnant as of now

Q3.7: What are the side effects of carbamazepine?

Answer: Leucopenia, aplastic anemia, hepatotoxicity

Q3.8: Which drug is preferred over carbamazepine in patients with liver damage?

Answer: Oxcarbazepine.

Q3.9: Which antiepileptic is given once daily and is a safer alternative to carbamazepine?

Answer: Eslicarbazepine

Q3.10: Why is phenytoin less preferred for long-term use?

Answer: It has nonlinear pharmacokinetics, leading to rapid toxicity.

Q3.11: Which antiepileptic is preferred in elderly patients due to minimal drug interactions?

Answer: Levetiracetam.

Q3.12: What is the first-line drug for absence, atonic, and myoclonic seizures?

Answer: Valproate.

Q3.13: Which drug is preferred over valproate in children under 5 years?

Answer: Ethosuximide

Q3.14: When can epilepsy medication be safely stopped?

Answer:

- Complete medical control for 1-5 years
- Single seizure type
- Normal CNS examination, including intelligence
- No family history
- Normal EEG

- Reasonable to attempt withdrawal after 2 years if all the above are satisfied
- Sudden stoppage of Anti-epilepsy medications can lead to rebound epilepsy
  → The frequency, severity, and duration of epilepsy can increase.

Q3.15: What are the key features of Lennox-Gastaut syndrome in children?

Answers:

- The child presents with multiple types of seizures, which can be like atypical absence seizures, atonic seizures, or GTCS.
- These children also have low IQ

Q3.16: Which antiepileptic drug is the most teratogenic?

Answer: All anti-epileptic drugs are teratogenic; the most teratogenic drug is Valproate (7-20% adverse fetal outcome).

Q3.17: Which antiepileptic drugs reduce vitamin K-dependent clotting factors, increasing the risk of hemorrhagic disease in newborns?

Answer: Phenytoin, Carbamazepine, and Oxcarbazepine

Q3.18: Which antiepileptic drugs are considered safest during pregnancy?

Answer:

- Levetiracetam and Lamotrigine

$$\frac{\text{Drug in breast milk}}{\text{Drug in serum}}$$

- For Valproate, it is 5%
- Levetiracetam: 300%

Q3.19: Should antiepileptic drugs be continued during breastfeeding?

Answer: Yes, but the baby should be monitored for excessive drowsiness or lethargy. If it is excessive, then decrease/modulate the dose

Q3.20: What should be done if a pregnant woman on antiepileptics presents in the 2nd or 3rd trimester?

Answer: Continue the same antiepileptic drug she is already taking

BIG QUESTION 4: Give a brief description of absence seizures, subtle seizures, and febrile seizures.

Broad Answer: Absence seizures, formerly known as "petit mal" seizures, are a type of generalized seizure that causes a brief, sudden lapse of consciousness. They are more common in children than in adults and are often mistaken for daydreaming or inattention. Subtle seizures are more common in neonates, and febrile seizures are the most common type of seizures in children

DETAILED QUESTIONS

Q4.1: In which age group are absence seizures most commonly seen?

Answer: Pediatric age group

Q4.2: Do absence seizures have tonic or clonic phases?

Answer: No, they do not

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Q4.3: What are the clinical features of absence seizures in children?

Answer:

- Vacant staring spells are seen in children (The child is called a daydreamer/absent-minded by parents)
- Blinking of the eyes is seen during the episode
- Seizure episodes can occur several times a day
- No post-ictal deficit is seen
- The child is unresponsive during the episode, but the postural tone is maintained



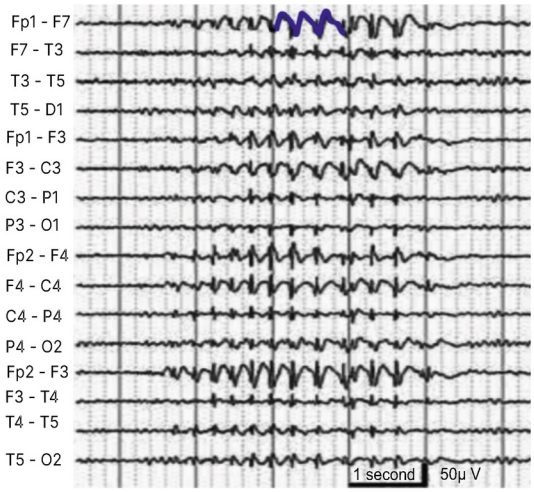
"Vacant Staring Spells"

Q4.4: What can trigger absence seizures?

Answer: Hyperventilation or bright light (photic stimulation).

Q4.5: What is the characteristic EEG finding in absence seizures?

Answer: 3 per second spike-and-slow wave pattern



Q4.6: What is the first-line treatment for absence seizures?

Answer: Valproate

Q4.7: Which drug is preferred for absence seizures in children under 5 years old, and why?

Answer: Ethosuximide, because valproate may cause hepatotoxicity in young children

Q4.8: What is the most common type of seizure in children?

Answer: Febrile seizures.

Q4.9: What is the most common type of epilepsy in children?

Answer: Benign Rolandic Epilepsy.

Q4.10: What is the most common type of epilepsy in adults?

Answer: Generalized tonic-clonic seizures (GTCS)

Q4.11: What is the most common type of seizure in neonates?

Answer:

- Subtle seizures
- There will be no convulsions; only jitteriness is seen, like tremors of the lips and fingers.

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Q4.12: Why are convulsions typically absent in neonatal seizures?
Answer: Because the central nervous system is not fully developed in neonates

Q4.13: Is a febrile seizure considered epilepsy? Give a reason for the same.
Answer: No, because it is provoked by high-grade fever.

Q4.14: What is a key feature after a febrile seizure episode?
Answer:

- The child has a fever, lips will turn blue, but no post-ictal confusion is seen.
- The child becomes completely normal after the episode

Q4.15: What is the acute treatment for febrile seizures?
Answer: Intranasal midazolam or rectal diazepam.

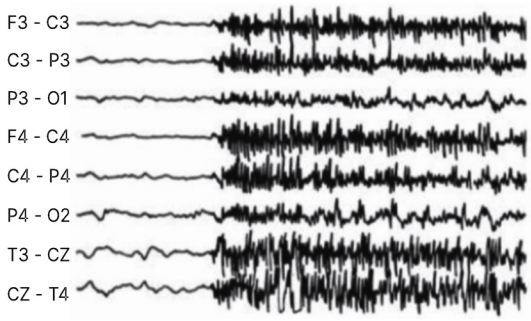
Q4.16: What medications are used for the prevention of febrile seizures?
Answer: Oral diazepam or oral clobazam

Q4.17: What type of seizure is seen in Benign Rolandic Epilepsy?
Answer: Focal seizure.

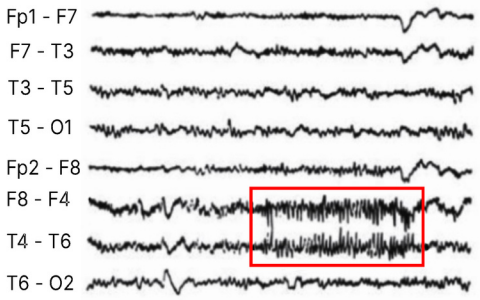
Q4.18: What are common symptoms of Benign Rolandic Epilepsy?
Answer: An uncontrollable twitching on one side of the face and drooling of saliva occur

Q4.19: How might a parent recognize nocturnal episodes of Benign Rolandic Epilepsy?
Answer: By noticing saliva stains on the pillow in the morning.

Q4.20: What does the following EEG indicate?
Answer: The EEG pattern shows low voltage, fast activity, followed by polyspike high amplitude waves in odd and even leads. This implies that it is a case of GTCS



Q4.21: What does the following EEG suggest?
Answer: EEG shows abnormality is localized to the even leads or the temporal lobes. This suggests a Focal seizure



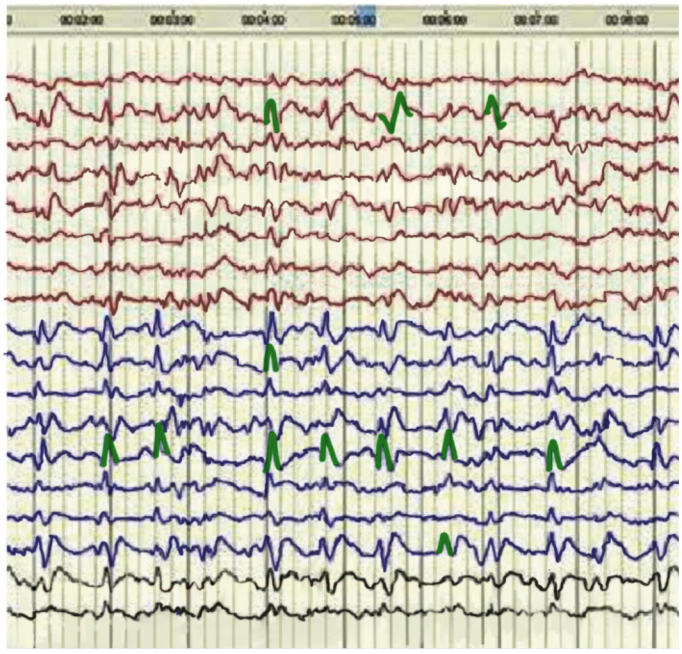
BIG QUESTION 5: What is Variant Creutzfeldt-Jakob Disease (vCJD) and what is it associated with?

Broad Answer: Variant Creutzfeldt-Jakob Disease (vCJD) is a rare and fatal neurodegenerative disease that is part of a family of diseases known as transmissible spongiform encephalopathies (TSEs). It is a human form of a prion disease, and it is strongly linked to bovine spongiform encephalopathy (BSE), commonly known as "mad cow disease."

DETAILED QUESTIONS

Q5.1: A 30-year-old man presented with complaints of myoclonic jerks and dementia. History suggests the consumption of poor-quality beef.

Answer: EEG shows periodic sharp wave complexes & is a keyword in MCQ, to identify VCJD.



Q5.2: Explain the pathogenesis of Variant Creutzfeldt-Jakob Disease (vCJD).

Answer: Neurodegeneration also results in dementia.

Tainted beef has prion particles which enter the GIT and after that crosses gut wall to enter blood stream. → These prion particles can cross the blood brain barrier. Blood brain barrier is composed of foot process of astrocytes. → After crossing the BBB these prions start accumulating in the cytoplasm of neurons and cause neurodegeneration. → This results in abnormal firing of neurons which results in myoclonic jerks.

Q5.3: In which condition is periodic lateralized epileptiform discharge (PLED) seen on EEG?

Answer: HSV encephalitis

BIG QUESTION 6: How does the presentation and etiology of myoclonic jerks vary across different age groups, and what are the associated EEG patterns and management strategies?

Broad Answer:

- Myoclonic jerks vary by age. West syndrome in infants shows hypsarrhythmia on EEG and is treated with ACTH or vigabatrin. Around 8 years SSPE follows measles infection and causes cognitive decline.
- Juvenile Myoclonic Epilepsy (10–19 years) presents with hand jerks and shows 4–6 Hz polyspikes on EEG, treated with valproate. In adults (~35 years), vCJD from prion-infected beef causes startle myoclonus and dementia, with periodic sharp waves on EEG.

DETAILED QUESTIONS

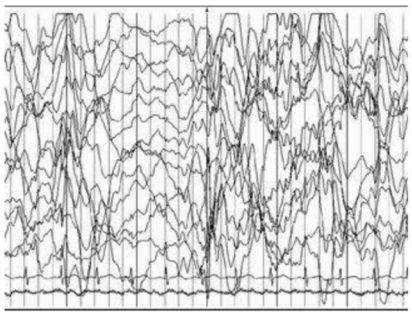
Q6.1: Which age-related conditions are commonly associated with myoclonic jerks?

Answer:

- <1 year: West syndrome/ Infantile spasm/salaam seizures
- 8 years: SSPE
- 10- 19 years: JANZ syndrome/juvenile myoclonic epilepsy
- 35 years: Variant Creutzfeldt-Jakob disease/prion disease

Q6.2: What are the key features, EEG findings of the West Syndrome/ Infantile Spasm/ Salaam Seizures?

Answer:



- It is seen in infants (<1 year age)
- EEG findings
  - Hypsarrhythmia: A Chaotic pattern is seen
  - High-amplitude polyspikes are seen
  - Frequency is low (delta grade)
- CRH is incriminated in causing the peculiar condition

Q6.3: What is the management of West Syndrome?

Answer:

- Inj. ACTH (it downgrades the CRH)
- VIGABATRIN: Tuberous sclerosis

Q6.4: What is SSPE?

PYQ: FMGE 2019

Answer: It is a rare complication of the measles virus.

Q6.5: At what age does SSPE usually occur?

Answer: It usually occurs around 8 years of age.

Q6.6: Which group of children is more likely to develop SSPE?

Answer: Children who are not immunized against measles.

Q6.7: What happens to a child's school performance with SSPE?

Answer:

- Scholastic performance of the child keeps decreasing, and the child is not able to understand things taught in school.
- Previously, the child was completely normal.

Q6.8: How is SSPE diagnosed?

Answer: By checking for measles IgG antibodies in the cerebrospinal fluid (CSF)

Q6.9: What age group is affected by JANZ syndrome (Juvenile Myoclonic Epilepsy)?

Answer: Adolescents aged 10-19 years.

Q6.10: What symptom do parents often notice in children with JANZ syndrome?

Answer: Recurrent slipping of things from the child's hands.

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Q6.11: What is the typical EEG pattern seen in JANZ syndrome?
Answer: A 4-6 Hz polyspike pattern.

Q6.12: What seizure type may develop later in children with juvenile myoclonic epilepsy?
Answer: Generalized Tonic-Clonic Seizures (GTCS).

Q6.13: What is the treatment for JANZ syndrome?
Answer: Sodium valproate.

Q6.14: What causes Variant Creutzfeldt-Jakob disease?
Answer: Eating tainted or infected beef containing prions.

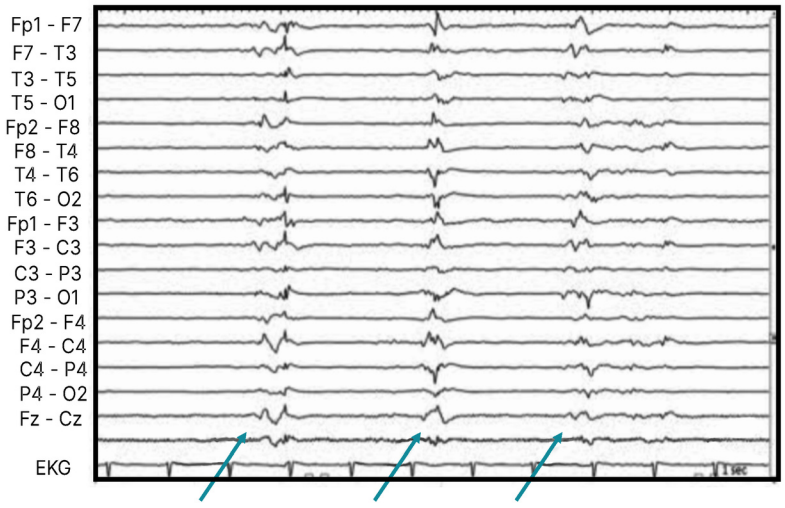
Q6.15: What are the main symptoms of this disease?
Answer: Myoclonic jerks and dementia.

Q6.16: What is startle myoclonus?
Answer: Myoclonic jerks triggered by loud sounds.

Q6.17: What protein is found in the CSF in Variant Creutzfeldt-Jakob disease?
Answer: Protein 14.3.3.

Q6.18: What EEG pattern is characteristic of Variant Creutzfeldt-Jakob disease?
Answer: Periodic sharp wave complexes

Q6.19: What does the following EEG pattern suggest?
Answer: EEG shows a Burst suppression pattern, which is seen in comatose patients. The reason for the coma in this patient is drug overdosage and severe hypothermia.



Q6.20: What does the following EEG pattern indicate?
Answer:

- EEG shows Triphasic waves.
- In a patient with hepatic encephalopathy
- It is called so because a Negative wave will be present before and after every positive wave

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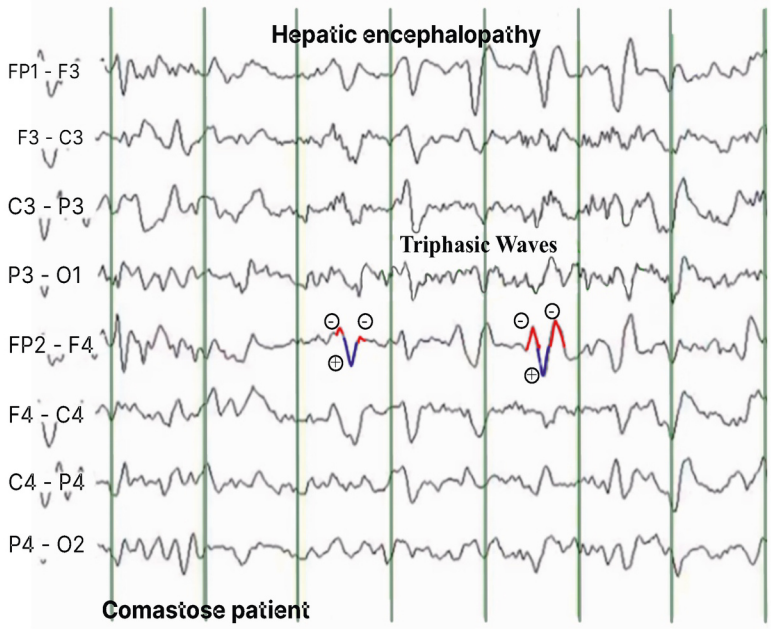


BIG QUESTION 7: What is Generalized Convulsive Status Epilepticus (GCSE)?

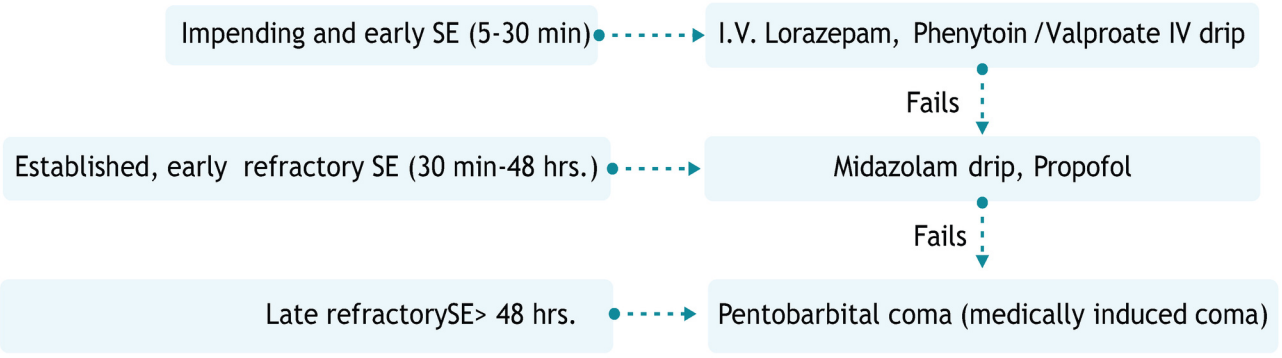
Broad Answer:

- GCSE is defined as a convulsion for > 5 minutes
- Earlier, the term Status epilepticus was used instead of GCSE, and the cut-off duration for status epilepticus was 30 minutes

DETAILED QUESTIONS

Q7.1: What is the treatment of GCSE?

Answer:



BIG QUESTION 8: What is a Subarachnoid Hemorrhage (SAH)?

Broad Answer: SAH is bleeding into the subarachnoid space (between arachnoid and pia mater). Causes include: Rupture of a berry aneurysm (usually at the circle of Willis bifurcations), Arteriovenous malformations (AVMs), and Trauma

DETAILED QUESTIONS

Q8.1: What are the causes of Subarachnoid Hemorrhage (SAH)?

Answer: The most common cause of subarachnoid hemorrhage is trauma > rupture of a berry aneurysm/mycotic aneurysm/ rupture of a Charcot Bouchard aneurysm

Q8.2: What is a mycotic aneurysm, and what causes it?

Answer:

- The causative agent of mycotic aneurysm is Staphylococcus aureus. Mycotic aneurysm is a complication of Infective Endocarditis
- The infection from the heart reaches the brain's blood vessels

Q8.3: What are the clinical features of Subarachnoid Hemorrhage (SAH)?

Answer:

- Thunderclap headache/worst headache of life
- As blood will spill into the meninges, it will cause nuchal rigidity

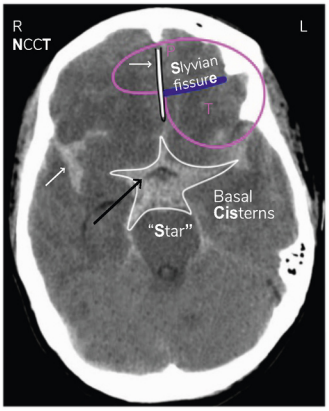
Q8.4: What is the CSF finding of SAH?

Answer: Bloody CSF/Xanthochromic CSF (RBCs in CSF break down to bilirubin, which will result in a yellowish appearance of CSF)

Q8.5: What does NCCT show in subarachnoid hemorrhage related to the Sylvian fissure?

Answer:

- NCCT: Shows blood in the sylvian fissure, which comes from the Middle cerebral artery (MCA)
- NCCT showing whiteness in the Sylvian fissure.
- The Sylvian fissure anatomically separates the temporal and parietal lobes
- Spillage of blood in the Basal cistern (star appearance)



Q8.6: What are common causes of bloody CSF seen on lumbar puncture?

Answer: Trauma, HSV-1 encephalitis, and subarachnoid hemorrhage.

Q8.7: Is fever commonly seen in subarachnoid hemorrhage?

Answer: No, fever is not typically seen, there is nuchal rigidity

Q8.8: How does elevated BNP contribute to seizures in subarachnoid hemorrhage?

Answer: Elevated BNP- BNP causes natriuresis, which results in a decrease in the sodium levels of the body. When the serum sodium level becomes <125 mEq, resulting in seizures.

Q8.9: What ECG changes may be seen in subarachnoid hemorrhage and why?

Answer:

- The intensity of pain is very high in SAH, so patients may develop MI due to catecholamine excess.
- ECG shows ST depression and T wave inversion

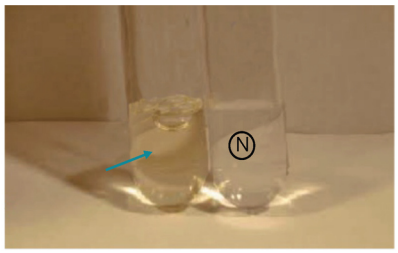
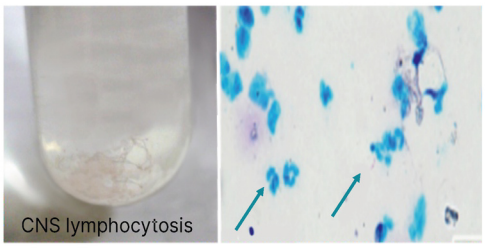
Q8.10: What are the typical NCCT and CSF findings in tubercular meningitis?

Answer:

- Basal exudates on NCCT are seen in tubercular meningitis
- Cobweb coagulum is seen in the CSF sample of TB meningitis

Q8.11: What is seen in the following images

Answer:

Xanthochromic CSF 	
• Xanthochromic CSF • It takes 24 hours to appear	• Cobweb coagulum • CNS lymphocytosis is seen in the CSF of TB meningitis.

Q8.12: What are the WFNS and Hunt and Hess scales used for in subarachnoid hemorrhage?

Answer:

SAH Grade	World Federation of neurological Surgeons Scale	Hunt and hess scale
1 (good)	GCS 15, no motor deficit	Asymptomatic or mild headache and slight nuchal rigidity
2 (good)	GCS 14-13, no motor deficit	Moderate to severe headache, nuchal rigidity, no focal neurologic deficit other than cranial nerve palsy
3 (good)	GCS 14-13, motor deficit	Confusion, lethargy, or mild focal neurologic deficit Other than cranial nerve palsy
4 (poor)	GCS 12-7, with or without motor deficit	Stupor or moderate-to-severe hemiparesis
5 (poor)	GCS 6-3, with or without motor deficit	Coma, extensor posturing, moribund appearance
GCS: Glasgow coma scale.		

Q8.13: What is the treatment of SAH?

Answer:

- When the berry ruptures, the blood vessels in the surrounding area exhibit a protective spasm of blood vessels.
- But this mechanism acts as a double insult as it causes cerebral infarction.
- So, as a damage control measure, use
  - Nimodipine (a calcium channel blocker)
  - With strict complete bed rest advised

Q8.14: What are the causes of death in SAH?

Answer:

- Vasospasm leading to cerebral infarction (the most common cause of death)

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- Rebleeding (rebleeding can occur from the same or a different site)
- Hydrocephalus
- Seizures: when Na^+ <125 mEq

BIG QUESTION 9: What is a berry aneurysm (saccular aneurysm)?

Broad Answer: A berry aneurysm, also known as a saccular aneurysm, is a common type of brain aneurysm. It's a localized, sac-like bulge or ballooning in the wall of a blood vessel in the brain. The name "berry aneurysm" comes from its appearance, which resembles a small berry hanging from a stem.

Q9.1: Which cranial nerve is most commonly involved in an unruptured Berry aneurysm?

Answer: The 3rd cranial nerve.

Q9.2: What symptom is caused by 3rd cranial nerve involvement in a Berry aneurysm?

Answer: Ptosis (drooping of the eyelid).

Q9.3: Where is the most common location of an unruptured Berry aneurysm causing 3rd nerve palsy?

Answer: Posterior communicating artery.

Q9.4: Which is the most common cranial nerve involved in a ruptured berry aneurysm?

Answer: 3rd cranial nerve (not 6th cranial nerve)

Q9.5: Which layer of the blood vessel is affected in a Berry aneurysm?

Answer: Internal elastic lamina.

Q9.6: What is the most common site of a Berry aneurysm?

Answer: Anterior communicating artery.

Q9.7: Where is the most common site of rupture for a Berry aneurysm larger than 8 mm?

Answer: Top of the Basilar artery.

Q9.8: What is the rarest location for a Berry aneurysm?

Answer: Vertebral artery.

Q9.9: What are the causes of 3rd nerve palsy?

Answer:

- Mid-brain lesions: Weber/Nothnagel/Benedict syndrome
- Miller Fisher syndrome
- Unruptured berry aneurysm.

BIG QUESTION 10: What are the common types and causes of headaches, their key clinical features, and how can they be differentiated in diagnosis and management?

Broad Answer:

- Headaches include primary types like tension (band-like pain), migraine (unilateral, pulsating), and cluster (severe retro-orbital pain with tearing).
- Secondary headaches result from causes like giant cell arteritis, subarachnoid hemorrhage, or brain tumors. Diagnosis relies on symptoms and imaging, and treatment varies—NSAIDs or triptans for migraines, steroids for arteritis, and urgent care for serious causes

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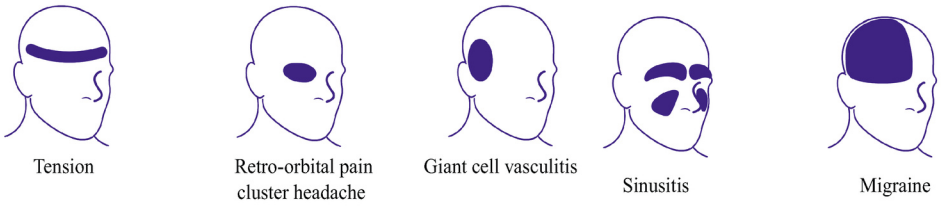
DETAILED QUESTION

Q10.1: What are the different types of headaches based on their location and underlying cause, and how do they clinically present?

Answer:

- Tension headache: pain in the band-like region
- Retro-orbital pain in cluster headache: deep boring pain
- Temporal headache-giant cell vasculitis (the only extracranial cause of headache)
- Sinusitis headache
- Migraine: Unilateral headache (Calcitonin gene-related peptide causes activation of nociceptive receptors



Q10.2: What are the most and least common causes of headache?

Answer:

	Primary headache	Secondary headache
Most common cause of headache	Tension headache: It is always a diagnosis of exclusion	Systemic infection
Least common	Cluster Headache Treatment: o 1st line: O_2 @ 6-12 L for 15 minutes o Inj. Sumatriptan SC	Brain tumor

Q10.3: What is a thunderclap headache?

Answer: It means headache intensity will peak in 60 seconds.

Q10.4: What are the causes of thunderclap headache?

Answer:

- SAH
- Intracranial hypotension
- Raised intracranial pressure
- PRES: Posterior Reversible Encephalopathy Syndrome
- Migraine
- Cluster headache: Lithium can be used
- Pituitary apoplexy- arteriovenous thrombosis/ Sheehan syndrome
- Cerebral venous thrombosis

Q10.5: What is the clinical presentation and imaging finding of Posterior Reversible Encephalopathy Syndrome (PRES)?

Answer:

- Clinical presentation: Hypertension, Headache, Visual blurring, Seizures
- MRI head shows cerebral edema in the occipital lobes and the posterior part of the parietal lobe (Vasogenic cerebral edema)

Q10.6: Which condition presents post-delivery with neurological features and shows the Empty Delta Sign on CT head?

Answer:

- **Condition:** Cerebral Venous Sinus Thrombosis (CVST)
- **Clinical features:** Common in the postpartum period, Headache, altered mental status, and Seizures / Coma (in severe cases)
- **Imaging (CT/MRV):** Empty Delta Sign → seen in superior sagittal sinus thrombosis

Q10.7: What does imaging show in intraparenchymal hemorrhage?

Answer: In an intraparenchymal hemorrhage, there will be hyperintensity and spilling of blood in the brain parenchyma

Q10.8: What are the red flags that indicate a serious cause of headache?

Answer:

- Headache increases on sitting, Valsalva maneuver (seen in Arnold Chiari malformation type 2)
- Papilledema
- Headache associated with a focal neurological deficit
- Sudden onset headache and nuchal rigidity in a known hypertensive patient raise the suspicion of Subarachnoid hemorrhage

Q10.9: What are the types of Arnold-Chiari malformations and their clinical associations?

Answer:

- There are 4 types of Arnold Chiari malformations- types 3 and 4 are not compatible with life.
- Arnold Chiari malformation type 1 leads to syringomyelia, a disease related to the spinal cord, where the patient experiences painless burns in the hand.

BIG QUESTION 11: Describe Giant Cell Arteritis (GCA), including the vessels it affects and its potential complications.

Broad Answer: Giant cell arteritis (GCA), also known as temporal arteritis, is a type of vasculitis—an inflammation of the blood vessels. This condition primarily affects medium- and large-sized arteries, most often those in the head, especially the temporal arteries located at the temples. This inflammation causes the arteries to swell and narrow, restricting blood flow and potentially leading to serious complications if not treated promptly.

Q11.1: In which age group is Giant Cell Arteritis most commonly seen?

Answer: Most commonly seen in the older (60 and above) population, in a physically active person with a complaint of temporal headache.

Q11.2: What are the hallmark symptoms of Giant Cell Arteritis?

Answer:

- It is not relieved by taking NSAIDs
- The patient is normotensive and not taking any other medications
- The patient also has LG fever, off & on, for the past few weeks
- Other features: Jaw claudication, blindness (Monocular)

Q11.3: What clinical finding may be observed on palpation near the temporomandibular joint?

Answer: A cord-like structure may be felt over the temporomandibular joint

Q11.4: Which laboratory marker is characteristically elevated in temporal arteritis?

Answer: ESR (Erythrocyte Sedimentation Rate) is markedly elevated, often >100 mm/hr.

Q11.5: Which artery is primarily involved in Giant Cell Arteritis?

Answer: The superficial temporal artery, which supplies the skin of the scalp and the TM joint.

Q11.6: What are the biopsy findings in Giant Cell Arteritis?

Answer: Granulomatous vasculitis with the presence of giant cells.

Q11.7: What other arteries can be involved in Giant Cell Arteritis?

Answer:

- Branches of the external carotid artery
- Branches of the internal carotid artery
- Ophthalmic artery

Q11.8: Which serious ocular complication can result if the ophthalmic artery is involved?

Answer: Monocular or binocular blindness due to ischemia.

Q11.9: What is the treatment of choice for Giant Cell Arteritis?

Answer: Steroids (to prevent complications like blindness).

BIG QUESTION 12: What are the typical features of migraine, who is most commonly affected, and what are the main treatment options?

Broad Answer: A migraine is a neurological condition most common in young women, characterized by a debilitating, pulsating, unilateral headache. It's often accompanied by nausea, photophobia (light sensitivity), and phonophobia (sound sensitivity), and sometimes a visual aura. Treatment includes triptans for acute attacks and medications like propranolol for prevention.

Q12.1: What are the clinical features of migraine?

Answer: Most commonly seen in young females (25 years)

- Pulsatile/pounding unilateral headache
- Usually, the headache disappears within 24 hours after taking rest or a painkiller
- Nausea is present in some patients
- Aura/visual blurring is seen (aura is seen in migraine and focal seizures)
- Migraine is disabling in nature- due to PHOTOPHOBIA and PHONOPHOBIA

Q12.2: What are the key features of migraine?

Answer:

- Mnemonic: **POUND**
  - **P** - Pulsating
  - **O** - One day illness
  - **U** - Unilateral
  - **N** - Nausea
  - **D** - Disabling in character

Q12.3: What tool is used to assess the severity of migraine?

Answer: MIDAS (Migraine Disability Assessment Score)

Q12.4: What are the treatment options for acute migraine attacks?

Answer:

- TRIPTANS subcutaneously/ nasal spray/ mouth dissolving tablet PYQ: FMGE 2020
  - o Zolmitriptan
  - o Rizatriptan
- In case of a mild attack of migraine: NSAIDS

Q12.5: What are the prophylactic medications used in migraine management?

Answer: Propranolol, Flunarizine, and Valproate PYQ: FMGE 2020

BIG QUESTION 13: What are cluster headaches? Who are they most commonly seen in, and how do they typically present?

Broad Answer: Cluster headaches are a rare but extremely painful type of primary headache disorder. They are more common in young males, typically presenting with severe, unilateral pain located around or behind one eye.

Q13.1: What is the most commonly affected age and gender group in cluster headaches? PYQ: NEET PG 2020

Answer: Most commonly seen in young males (20 years)

Q13.2: What are the key symptoms of a cluster headache?

Answer:

- Retro orbital pain. (Deep boring pain)
- Epiphora (tear production)
- Nasal stuffiness
- Bulbar congestion
- Forehead sweating/flushing
- Clustering of headaches in a particular season is seen.

Q13.3: What is the typical duration of a cluster headache episode?

Answer: 15-180 minutes

Q13.4: What treatments are used during an acute cluster headache attack?

Answer:

- Oxygen therapy at 6-12 L/min
- Lignocaine jelly in the nostrils of the patient
- Triptans SC inj
- Lithium may be used: Lithium is given if self-mutilation symptoms are seen
- Steroids
- Occipital nerve blocks.
- Monoclonal antibodies- GALCANEZUMAB: It blocks CGRP (Calcitonin gene-related peptide)

Q13.5: Which drugs are used for prophylaxis and advanced treatment of cluster headaches?

Answer: Verapamil

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Q13.6: What is the difference between cluster headache, paroxysmal hemicrania, and SUNCT?

Answer:

	Cluster Headache	Paroxysmal Hemicrania	SUNCT: Short lasting unilateral Neuralgiform headache attack with conjunctival injection and tearing
Prevalence	0.06-0.3%	0.02%	Very rare
Sex ratio F/M	1:3	2.4:1	1:1.3-2
Mean age at onset	29	37	48
Attack duration (mean)	15-180 min (70-160 min)	2-30 min (17 min)	5s to 4 min (58 s)
Attack frequency (mean)	1-8 day (5)	5-40 day (11)	3-200 day (59)
Chronic/episodic	Episodic (85%)	Chronic (80%)	Chronic (70%)
Pain quality	Boring, pressing, burning	Sharp, stabbing, throbbing	Stabbing, electric shock, sharp
Pain location	Retro-orbital temporal	Temporal, orbital	Eye, retro-orbital
Triggers	Alcohol, nitroglycerin	Usually not triggered	Cutaneous stimuli
Autonomic features	Yes	Yes	Yes (Cl & T)
Migrainers features	Yes	Yes	No
Indomethacin effects	-	++	-
Abortive treatment	Oxygen, Sumatriptan SC	Indomethacin	Lidocaine I.V.
Prophylactic treatment	Verapamil/ lithium	-	Lidocaine I.V

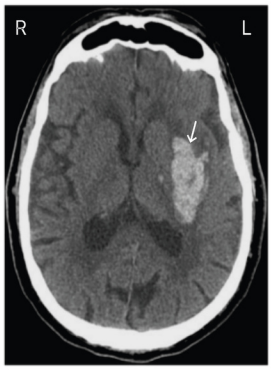
BIG QUESTION 14: Write briefly about intracerebral hemorrhage (ICH), its common causes and symptoms, and management.

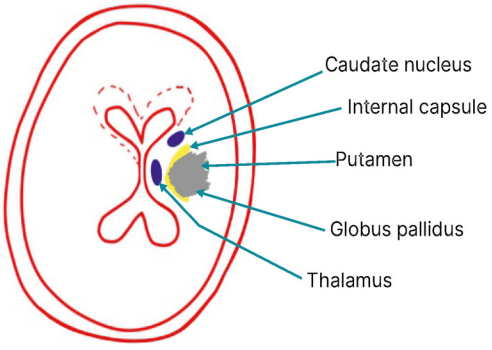
Broad Answer: Intracerebral hemorrhage (ICH) is a severe type of stroke caused by a ruptured blood vessel and bleeding inside the brain, often due to uncontrolled high blood pressure. Symptoms appear suddenly and include severe headache, one-sided weakness, and altered consciousness. It's a medical emergency requiring immediate stabilization and potential surgery to remove the blood clot and relieve pressure.

Q14.1: What is the most common site, and which populations have a higher incidence of intracerebral hemorrhage?

Answer:

- 35-45% die by 1 month due to complications
- More incidence in asians and blacks
- The most common site of intraparenchymal hemorrhage is the putamen





Q14.2: What are the main causes of intraparenchymal hemorrhage?

Answer:

- Hypertension
- Coagulopathy
- Cocaine and methamphetamine cause vasoconstriction in cerebral blood vessels, leading to increased pressure and rupture
- Cerebral amyloid angiopathy (apolipoprotein E gene: E2, E4)- there occurs weakening of blood vessels in the brain.
  - Cerebral amyloid angiopathy is the cause of intracerebral hemorrhage in non-diabetic and non-hypertensive patients.

Q14.3: What are the common causes of coagulopathy and their respective antidotes?

Answer: Warfarin toxicity-treatment: Prothrombin complex concentrate and vitamin K

- Dabigatran toxicity-Antidote: IDARUCIZUMAB
- Apixaban toxicity-Antidote: Andexanet alpha

Q14.4: What is the most common site of hypertensive intracerebral hemorrhage?

Answer:

- Putamen
- When the bleed is extended thalamus, cerebellum, and pons are also involved due to the rupture of penetrating arteries

Q14.5: What complication can occur if a cerebellar hamartoma is >3 cm, and how is it managed?

Answer: Obstructive hydrocephalus requiring a surgical intervention.

Q14.6: How soon do clinical features typically develop after an intracerebral hemorrhage, and what are some common symptoms?

Answer:

- Clinical features develop over 30-90 minutes after the bleed
- Features include
  - Contralateral sagging of the face
  - Slurring of speech
  - Arm weakness
  - Eye deviation (away from the site of the lesion)
  - In a cortical stroke, motor areas are involved- the eyes deviate to the side of the stroke.
  - In the subcortical stroke, the eye looks away from the stroke.
  - Irregularly irregular breathing (Biot's breathing) due to raised ICP
  - Biot's breathing indicates a bad prognosis
  - Coma
  - Decerebrate posturing/Rigidity

Q14.7: What are the key clinical features of thalamic hemorrhage?

Answer:

- Contralateral hemiplegia, hemianesthesia
- Chronic debilitating contralateral pain (Dejerine-Roussy syndrome)

Q14.8: What are the characteristic clinical signs and symptoms of pontine hemorrhage?

Answer:

- Deep coma
- Quadriplegia
- Pinpoint pupil (~1 mm)
- Hypertension, increased respiratory rate, hyperhidrosis
- Hyperthermia

Q14.9: What are the key clinical features of a cerebellar hematoma, and when is neurosurgical intervention indicated?

Answer:

- Occipital headache
- Vomiting
- Ataxia
- Hematoma > 3 cm needs a neurosurgical intervention

Q14.10: What is an important differential diagnosis to consider in a patient with intracerebral hemorrhage presenting with raised intracranial pressure?

Answer: Posterior reversible leukoencephalopathy

Q14.11: What are the common clinical features of Posterior Reversible Leukoencephalopathy Syndrome (PRES)?

Answer:

- Clinical presentation includes
  - Headache
  - Vomiting
  - Retinal hemorrhage on fundus examination
  - Convulsions
  - Stupor and coma

Q14.12: Which MRI finding is characteristic of PRES?

Answer: Vasogenic cerebral oedema in the occipital region

Q14.13: What underlying conditions can cause hypertension leading to PRES?

Answer:

- Patient is a known case of hypertension with features of raised ICP
- The cause of hypertension could be any: acute glomerulonephritis, CKD, toxemia of pregnancy

Q14.14: What is the primary treatment approach for Posterior Reversible Leukoencephalopathy Syndrome?

Answer: Reduce blood pressure

Q14.15: What factors are included in the ICH score used to assess the severity and prognosis of intracerebral hemorrhage?

Answer:

- ICH score Mnemonic: **AHI$_2$G**
  - **A** - Age
  - **H** - Hematoma volume

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- o **I** - Infratentorial location of bleed (infratentorially located in 4 ventricle, and there are high chances of developing obstructive hydrocephalus. Infratentorial bleed has a worse prognosis as compared to supratentorial bleed)
- o **I** - Intraventricular hemorrhage (intraventricular hemorrhage in pediatrics is usually common after birth trauma)
- o **G** - GCS

Q14.16: What are the common causes, clinical presentations, and diagnostic methods for intraventricular hemorrhage in neonates?

Answer:

- Intraventricular hemorrhage is usually common after birth trauma, e.g., faulty forceps delivery.
- Presentation in a neonate would be
  - o Shrill cry
  - o Bulging anterior fontanelle
  - o Pallor
- The IOC USG skull shows Intraventricular hemorrhage

Q14.17: What is the specific management of intracranial hemorrhage?

Answer:

- Airway protection- with intubation
- BP control: Nicardipine is used
  - o Sometimes, Nicardipine can cause reflex tachycardia.
  - o Esmolol is used to neutralize tachycardia in such patients.
  - o Target blood pressure is <140 mmHg (page 3349 Harrison)
- Treatment of coagulopathy
- Midline shift/obtundation in the patient- osmotic diuretics are used
  - o Mannitol is used in 3% concentration.
  - o 5% dextrose is never used in management.
- If Mannitol fails, we can perform: Ventriculostomy- A drain is placed in the lateral horn of the ventricle
- If the pressure is still rising, then neurosurgical decompression has to be done based on the ICH score

BIG QUESTION 15: What is an acute ischemic stroke, and what happens in the brain when it occurs?

Broad Answer: An acute ischemic stroke is a medical emergency that occurs when blood flow to part of the brain is suddenly blocked, typically by a blood clot. This deprives brain tissue of oxygen and nutrients, causing brain cells to die within minutes.

Q15.1: What percentage of total stroke cases does Acute Ischemic Stroke (AIS) account for?

Answer: Incidence of A.I.S is 85% of total cases of stroke. The other 15% is hemorrhagic

Q15.2: What is the most common source of AIS?

Answer: Cardioembolic

Q15.3: What are the common causes of AIS?

Answer:

- Atrial fibrillation (the commonest cause of cardioembolic clot formation)
- Atherosclerosis of the internal carotid artery
- Rare cause: (Cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy).
  - o It has a genetic basis
  - o The gene involved is NOTCH3.

YOURWISH

Q15.4: What is the typical duration of neurological symptoms in a Transient Ischemic Attack, and how soon do they usually resolve?

Answer:

- Sudden onset of neurological symptoms that resolve within 24 hours.
- In the majority of cases, spontaneous resolution occurs within 1 hour
- CT head is normal in TIA/ischemic stroke
- The highest chance of stroke after TIA is within 48 hours

Q15.5: What scoring system is used to predict the risk of stroke after a TIA within the next 3 months?

Answer: $ABCD_2$ score

PYQ: NEET PG 2019

Q15.6: What are the parameters of the $ABCD_2$ score?

Answer:

- Maximum score is 7
- If the score is 7, then the chances of the development of stroke within 3 months would be 22%

Parameter	Score
A: Age >60	1
B: BP >140/90 mmHg	1
C: Clinical features: Unilateral weakness Speech disturbance without weakness.	1+1
D: Duration > 60 minutes 10-59 minutes Diabetes mellitus	2 1 1

Q15.7: What is the management of TIA?

Answer:

- Dual antiplatelet therapy: Drugs used are Aspirin and Ticagrelor
  - Note: Dual antiplatelet therapy is also given in MI (both STEMI and NSTEMI), in a patient undergoing PCI with stenting in a CABG patient.
  - Ticagrelor is a P Y inhibitor. 2 12
  - Clopidogrel is not preferred in Asians as it is not metabolized in an active form
- Clopidogrel exhibits CYP2C19 polymorphism
- Statins: Used because most patients with TIA have hypercholesterolemia
- Control hypertension
  - Long-term target of BP in TIA < 120 mmHg (SPRINT TRIAL)
  - Immediate target of BP < 130/80 mmHg

Q15.8: In a patient with rheumatic heart disease, mitral stenosis, and atrial fibrillation who has experienced TIA episodes, which anticoagulant is used to reduce the risk of further TIA?

Answer: Warfarin

6. NEUROLOGY PART -2

BIG QUESTION 1: What is the approach to thrombolysis in the acute management of ischemic stroke?

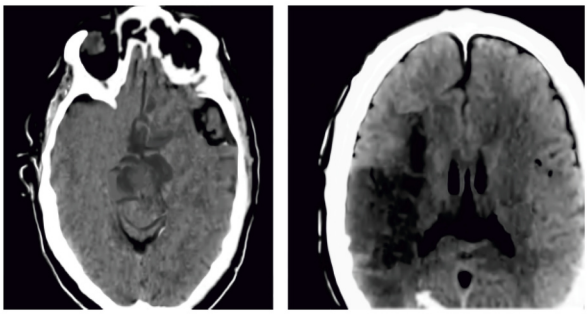
Broad Answer: Thrombolysis in acute ischemic stroke is performed to restore cerebral blood flow within a specific time window, aiming to salvage brain tissue. Beyond this window, mechanical thrombectomy is considered up to 24 hours. Management includes airway stabilization, BP control, NIHSS-based severity assessment, antiplatelet and anticoagulant therapy, statins, and consideration of carotid endarterectomy if indicated.

DETAILED QUESTIONS

Q1.1: What are the key radiological findings in acute ischemic stroke before thrombolysis?

Answer:

- Non-contrast CT head
  - Normal in the majority
  - Done initially to rule out haemorrhagic stroke
  - One of the side effects seen with thrombolysis is an increase in the incidence of bleeding.
- Hyperdense MCA sign: Early finding; not seen in most cases.
- Hypodensity: Develops later with ischemic damage.



Q1.2: What is the recommended time window for thrombolysis in ischemic stroke?

Answer:

- General recommendation: Up to 4.5 hours
- USA: Up to 3 hours
- Canada & UK: Up to 4.5 hours
- Harrison's latest edition: Benefit possible up to 6 hours
  - So, thrombolysis can be done up to: 4.5-6 hours

Q1.3: What are the indications and contraindications for thrombolysis?

Answer:

Indications	Contraindications
Clinical diagnosis of stroke	Sustained BP > 185/110 mmHg despite treatment (labetalol or nicardipine)
Onset of symptoms to time of drug administration ≤ 4.5 hours	Bleeding diathesis (INR > 1.7)
CT scan showing no hemorrhage or edema > 1/3 of the MCA territory Age ≥ 18 years	Recent head injury or intracerebral hemorrhage
	Major surgery in preceding 14 days
	GI bleeding in preceding 21 days
	Recent myocardial infarction

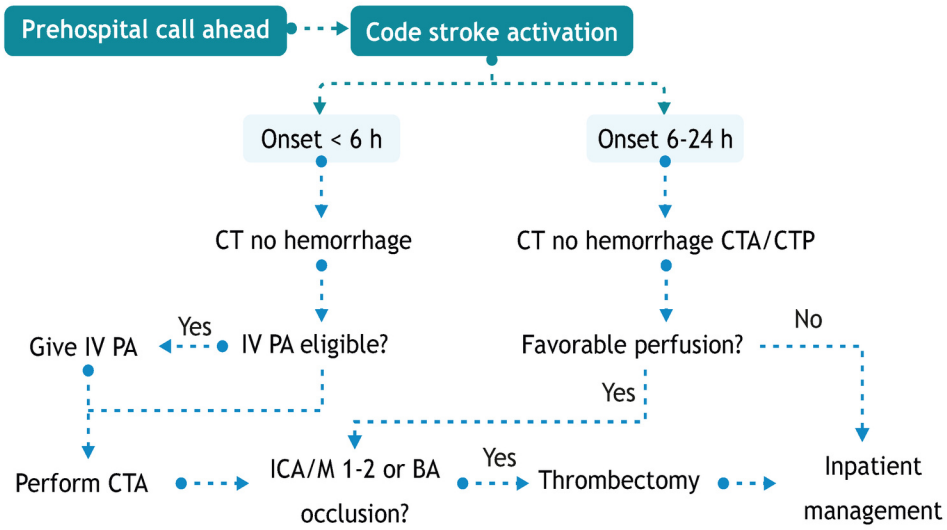
Q1.4: What are the drugs used in thrombolysis and their administration method?

Answer:

- **Alteplase:** Bolus followed by infusion
- **Tenecteplase:** Single bolus, can be given inside a CT scan after ruling out hemorrhage

Q1.5: What is the management pathway for acute ischemic stroke?

Answer:



Q1.6: Which patients are eligible candidates for thrombolysis within the first 6 hours?

Answer:

- Age > 18 years
- No evidence of hemorrhage with respect to CT head
- Onset of symptoms (earlier detected/intervened, more brain tissue can be saved)

Q1.7: In which clinical scenario is Tenecteplase given to a stroke patient?

- When the patient has clinical features suggestive of stroke
- The CT scan is normal
- Evidence of hemorrhage is ruled out

Q1.8: What are the steps in the management of acute ischemic stroke?

Answer:

1. Assess airway
2. BP control
3. Time last seen normally
4. Evaluate the severity of stroke by NIHSS (National Institute of Health Stroke Severity)
5. If NIHSS > 5
   - Thrombolysis
   - Endovascular therapy: MERCI
6. Once an acute ischemic stroke is handled
   - Then aspirin can be given after the first 24 hours, as it will prevent the redevelopment of stroke and reduce current mortality
   - Ticagrelor: Loading dose 180 mg and subsequently 90 mg twice a day

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- After 1 month: Ticagrelor will be discontinued and aspirin will be continued at a low dose for the whole life of this patient, 75- 81 mg, whichever is available (In Harrison: 81 mg)
- D.A.P.T.: Aspirin + Ticagrelor should be continued for 1 month, as it reduces the chances of reinfarction in these patients

7. Statins
8. Anticoagulant agents: Apixaban - Atrial fibrillation
   - If it is rheumatic heart disease or mitral stenosis, then warfarin is given
9. If there is atherosclerotic narrowing of the internal carotid artery, then either in the same hospitalization or maybe on a later admission, carotid endarterectomy can be done
10. Achieve target systolic blood pressure
    - Initially, SBP can be 130/140 to ensure cerebral perfusion
    - Cerebral perfusion = Mean arterial pressure - Intracranial pressure
    - But trials have shown that if a person has suffered from a stroke & once he is on the road to recovery/ handled the acute phase, then subsequently the target of BP in the range of 120 or less should be achieved

BIG QUESTION 2: What are the various sources of bleeding in different types of intracranial hemorrhage?

Broad Answer:

- Intracranial hemorrhage can present at various sites.
- Acute and chronic subdural hemorrhages appear concavo-convex on CT and arise from cortical bridging veins.
- Extradural hemorrhage is typically biconvex, often from middle meningeal artery rupture with a lucid interval.
- Intraparenchymal hemorrhage is often due to hypertensive crisis, causing lenticulostriate artery rupture.
- Intraventricular hemorrhage occurs as an extension of parenchymal bleed in adults or due to traumatic instrumentation in neonates.
- Subarachnoid hemorrhage presents with the "worst headache of life," blood in basal cisterns, and xanthochromic CSF on delayed LP.

DETAILED QUESTIONS

Q2.1: What are the imaging features and causes of acute subdural hemorrhage?

Answer:

PYQ: NEET PG 2022 PYQ: FMGE 2023

Q. Case scenario: A 70-year-old woman is a known case of Type II DM is suffering from diabetic neuropathy. She fell in bathroom last week and after 1 week she is having neurological symptoms  Acute Subdural hemorrhage	• Reason for neurological symptom: CNS bleed which can be seen in NCCT head and is **concavo-convex** in appearance causing pressure on brain parenchyma therefore focal neurological deficit occurring in the patient o Focal neurological deficit would be gradual in onset • Whether it is acute subdural hemorrhage or chronic subdural hemorrhage, it depends upon whether this concavo-convex bleed present is white or black • If it is black - Chronic subdural hemorrhage o Source of bleed in SDH: Cortical bridging veins • But if this is white - Acute subdural hemorrhage • Source of bleeding in SDH: Cortical bridging veins.

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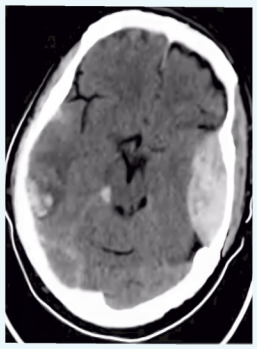
Q2.2: What is intraventricular hemorrhage, and how does it present in adults vs. pediatrics?

Answer:

- Adults: Extension of intracerebral/intraparenchymal hemorrhage.
- Pediatrics: Due to trauma from instrumentation (e.g., faulty forceps)
  - o Manifestations: Shrill cry, pallor, bulging anterior fontanelle
  - o Complication: Obstructive hydrocephalus → requires ventriculoperitoneal shunt

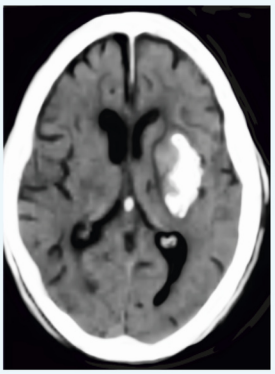
Q2.3: What are the key features of extradural hemorrhage (EDH)?

Answer:

Q. Case scenario: Biking accident without use of helmet.  Extradural hemorrhage	• Appearance of bleed present in this patient is **Biconvex** PYQ: NEET PG 2021 PYQ: INICET 2022 PYQ: FMGE 2021 • In any RTA - CNS bleed is suspected and CT scan is done because lots of time there is extradural hemorrhage & patient who deteriorate suddenly • Lucid interval would be seen • Source of bleeding: Middle meningeal artery

Q2.4: What are the imaging features of intraparenchymal hemorrhage?

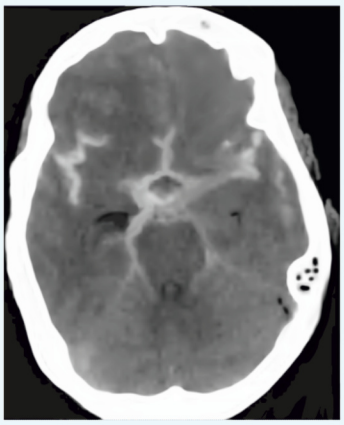
Answer:

Q. Case scenario: A guy is a known case of hypertension (hypertensive crisis) results in the rupture of lenticulostriate artery (Branch of middle cerebral artery).  Intra parenchymal hemorrhage	• First CT: shows left sided intra- parenchymal hemorrhage or intra -cerebral hemorrhage

Q2.5: What are the imaging features of subarachnoid hemorrhage (SAH)?

Answer:

Q. Case scenario: Patient came with the worst headache of his life PYQ: FMGE 2022	• In NCCT Head: Blood accumulation into the basal cisterns (Star of death) o Keywords will be patient will experience the worst headache of his life


Sub arachnoid hemorrhage

- Diagnosis: **Subarachnoid hemorrhage**
- Source of bleed: Middle cerebral artery (CT shows bleeding in the sylvian fissure)
- In these patients if lumbar puncture is usually not done because of raised ICP
- Lumbar puncture: Bloody CSF
- Work up
  - o NCCT
  - o CT scan not available: lower pressure in brain by mannitol and lumbar puncture shows evidence of bloody CSF
- If CT scan is done after 24-48 hours, then RBCs will break down to produce bilirubin and appearance of CSF will be slightly yellowish i.e., **Xanthochromic CSF**

BIG QUESTION 3: What is the anatomy of the Circle of Willis?

Broad Answer:

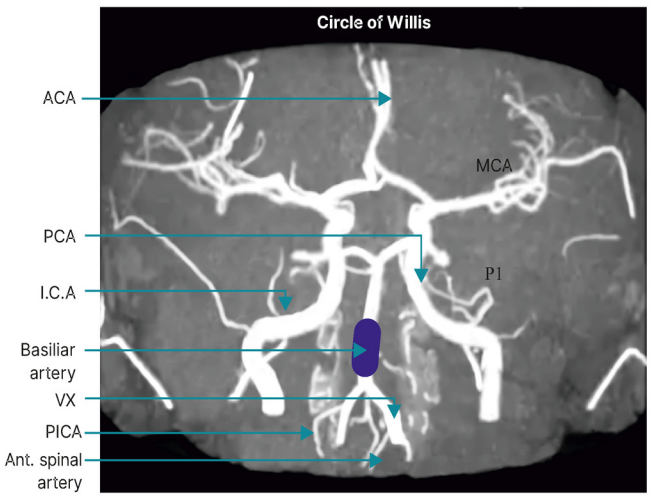
- The Circle of Willis is formed by the internal carotid arteries, vertebral arteries, and basilar artery with their branches.
- Key branches include the posterior cerebral artery (PCA), middle cerebral artery (MCA), posterior inferior cerebellar artery (PICA), anterior spinal artery, and lenticulostriate arteries.

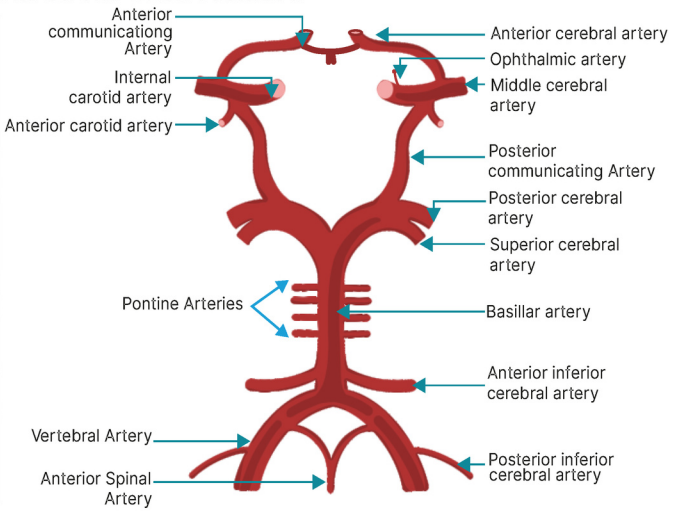
DETAILED QUESTIONS

Q3.1: What are the main vessels forming and supplying the Circle of Willis?

Answer:

- Internal carotid artery: Major blood supply to the brain
- Basilar artery: Supplies pons; formed by the union of vertebral arteries
- Posterior cerebral artery (PCA): Originates from the basilar artery
  - o P1 segment: Extends posteriorly to form P2; supplies the occipital lobe
- Vertebral artery branches:
  - o Lateral branch: PICA → involved in Wallenberg syndrome
  - o Medial branch: Anterior spinal artery → involved in medial medullary syndrome
- MCA branches: Lenticulostriate arteries → cause lacunar stroke when occluded





Q3.2: What is the clinical presentation of P1 PCA involvement?

Answer:

- I/L 3rd nerve palsy
- Crus cerebri involvement → C/L hemiplegia
- Results in crossed manifestations (cranial nerve palsy on one side, motor deficit on the opposite side)
  - Crossed Hemiplegia: characteristic manifestation of brainstem stroke

Q3.3: What is Weber syndrome?

Answer:

- Lesion: Midbrain (P1 PCA territory)
- Features: I/L 3rd nerve palsy + C/L hemiplegia (crossed hemiplegia)

Q3.4: What is Millard-Gubler syndrome and its vascular cause?

Answer:

- Lesion: Pons (basilar artery, pontine perforators)
- Features: I/L 6th and 7th nerve palsy + C/L hemiplegia

Q3.5: What is locked-in syndrome and its manifestations?

Answer:

- Cause: **Thrombus** in the main stem of the basilar artery **due to atrial fibrillation**.
- Manifestations:
  - Quadriplegia
  - Anarthria
  - Bilateral Babinski sign / Bilateral long tract signs
  - **Consciousness preserved**

Q3.6: What is Wallenberg (lateral medullary) syndrome, and how does it present?

Answer:

- Cause: Involvement of the **vertebral artery or PICA**
- Features:
  - Sensory nucleus of trigeminal nerve → Loss of facial sensations (I/L)
  - Lateral spinothalamic pathway → Loss of pain & temperature (C/L)
  - This combination = Crossed hemianesthesia
  - Other CNs involved: 5, 7, 8, 9, 10, 11
  - Nerve not involved: 12th CN (since its nucleus is medial)
  - All involved nerves originate from the lateral brainstem

Q3.7: What is medial medullary syndrome and its presentation?

Answer:

- Cause: Anterior spinal artery occlusion.
- Features: I/L 12th nerve palsy + C/L hemiplegia.
- Sign: Tongue deviates toward the side of the lesion on protrusion

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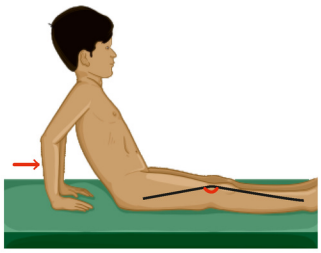
BIG QUESTION 4: What are the key clinical features, causes, investigations, and treatments of CNS infections?

Broad Answer: CNS infections include meningitis (bacterial, viral, tubercular, fungal, aseptic), neurocysticercosis, viral encephalitis (including Nipah virus), amoebic meningoencephalitis, and rare variants like Mollaret meningitis. They present with fever, headache, nuchal rigidity, altered sensorium, seizures, or focal neurological deficits. Diagnosis is made with clinical signs, CSF analysis, blood cultures, and neuroimaging. Treatment varies with etiology, including antimicrobials, antifungals, antivirals, steroids, and supportive care.

DETAILED QUESTIONS

Q4.1: What are the features and causes of aseptic meningitis?

Answer:



- Clinical sign: AMOSS sign/Tripod sign
  - On asking the child to sit up:
    - → Slight flexion at knee & hip joints
    - → Uses arms to prop body
    - → Associated with slight spinal cord flexion
- Causes:
  - Viral: Enterovirus
  - Rheumatological diseases - Sjogren syndrome
  - Drugs: Ibuprofen, Trimethoprim, Sulfamethoxazole, Ciprofloxacin
- Investigations:
  - Gram stain: Negative
  - Culture: Negative

Q4.2: What is the causative organism and mode of transmission in neurocysticercosis?

Answer:

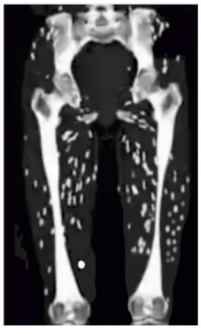
- Caused by *Taenia solium* (pork tapeworm)
- Ingestion of pork (larvae): Causes intestinal tapeworm
- Ingestion of vegetables: Grown on fecally contaminated soil leads to ingestion of eggs and development of NCC

Q4.3: What are the clinical and examination findings in neurocysticercosis?

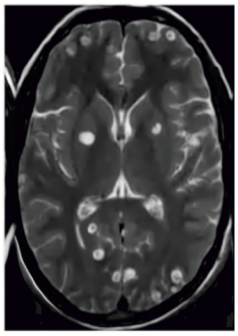
PYQ: NEET PG 2019

Answer:

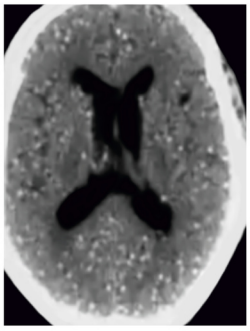
- Low socioeconomic status
- Focal seizures: Multiple episodes
- O/E: Multiple subcutaneous lumps with limited mobility
- Work-up
  - Initial investigation: Lumbar puncture (guarded) for CSF immunoblot for NCC antigen (CSF ELISA is for cryptococcal antigen)
  - Imaging modality of choice: Gadolinium enhanced MRI



Cysticercosis: Rice grain calcification (dead larvae)



Scolex of developing worm or larvae in brain



NCCT head: Starry sky appearance

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→ Evidence of vasogenic cerebral edema
→ MRI head of patient: Lesion having white dot in black circle
→ Vasogenic cerebral edema → irritates neurons → focal seizures
- CT: Calcification is not an early presentation of neurocysticercosis

Q4.4: What are the radiological stages of lesion in neurocysticercosis?

Answer:

- Vesicular stage
- Colloidal vesicular stage
- Nodular vesicular stage
- Nodular calcified stage

Q4.5. What are the differential diagnoses for a ring-enhancing lesion in the brain, and how can Magnetic Resonance Spectroscopy (MRS) help in differentiating between them?

Answer:

- The main differential diagnoses for a ring-enhancing lesion are **neurocysticercosis** and **tuberculoma**.
- **MRS (Magnetic Resonance Spectroscopy)** is useful in differentiation, as it evaluates the metabolic profile:
  - **Neurocysticercosis** shows **multiple amino acid peaks** (due to parasite metabolism).
  - **Tuberculoma** shows a **single lipid peak**, as seen in tubercular lesions.
- Additionally, **tuberculoma** is often associated with **tubercular meningitis (TBM)**, where **basal exudates** (pus in basal cisterns) may be observed.

Q4.6: What are the distinguishing features between NCC and tuberculoma of the brain?

Answer:

Features	Neurocysticercosis	Tuberculoma of the brain
Lesions	Smaller (< 20mm), single or multiple	Larger (>20 mm), often multiple, conglomerated
Associated meningitis	Absent	Present (associated findings like nuchal rigidity, Kernig sign or Brudzinski sign)
Location	Junction of gray-white matter	Common in posterior fossa
MR spectroscopy	Multiple amino acid peaks	Lipid peaks (with respect to M. Tuberculosis metabolism)

Q4.7: What is the treatment protocol for cysticercosis and neurocysticercosis?

Answer:

- Cysticercosis:
  - Steroids and Praziquantel
- Neurocysticercosis
  - Initial treatment: Dexamethasone for 48 hrs (reduce cerebral edema)
  - Anti-parasitic: Albendazole (given after steroids to avoid inflammatory reaction)
  - For Focal Seizure: Carbamazepine/Lamotrigine

Q4.8: What are the etiology and clinical features of cryptococcal meningitis?

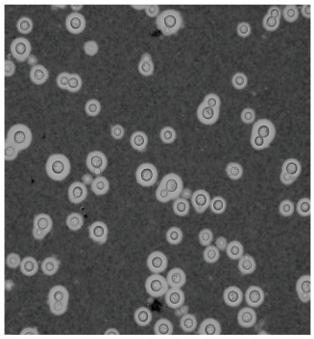
Answer:

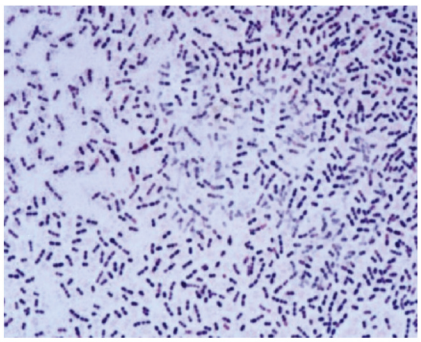
- Seen in: Immunocompromised patients (e.g., AIDS positive truck driver)
- Clinical features:
  - o Fever, headache, photophobia, vomiting, nuchal rigidity

Q4.9: What is the workup to be done in a suspected case of Cryptococcal meningitis?

Answer:

- First investigation: Blood culture, start on empirical antibiotics within 1 hour on admission of patient
- Empirical antibiotics: Ceftriaxone + Vancomycin (do not cover cryptococcus)
- NCCT head: for evidence of raised ICP
- Lumbar puncture: CSF for cell counts
  1. Lymphocytic pleocytosis
  2. Sugars slightly reduced, proteins increased in fungal meningitis (normal protein value: 15 - 45 mg %)
  3. Gram staining: No organism demonstrated
  4. India ink preparation: Negative stain (not reliable)
  5. CSF ELISA for Cryptococcal Antigen: IOC for cryptococcal meningitis
     - → Cryptococcus is excreted in pigeon droppings, so it is present in dust and it can cause cryptococcomas in lungs





     - → In healthy person due to good immunity the infection never reaches the bloodstream and brain and therefore do not develop meningitis
     - → In a person with low CD4 count, he would susceptible to cryptococcomas not only in lungs but a hematogenous dissemination, it reaches brain and cause cryptococcal meningitis
  6. Lateral flow assay Cryptococcal Antigen: Can be done on serum/plasma/CSF
  7. CD4 count should be <100 to cause manifestations of cryptococcal meningitis

Q4.10: What is the treatment of choice for cryptococcal meningitis?

Answer: The treatment of choice is **Liposomal Amphotericin B + Fluconazole**.

Q4.11: An AIDS-positive truck driver with a Gram stain showing Gram-positive diplococcus. What is the diagnosis?

Answer: Pneumococcal meningitis.

Q4.12: Which organism, besides Cryptococcus, is also positive with India ink preparation?

Answer: **Pneumococcus** can also be positive with India ink preparation.

Q4.13: What is the treatment for pneumococcal meningitis?

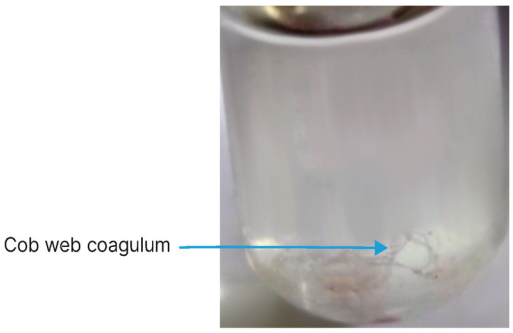
Answer: Antibiotics

Q4.14: What are the CSF findings in tubercular meningitis?

Answer:

- Straw-colored CSF with >100 lymphocytes/mm^3

- ↓ Decreased sugar
- ↑↑ protein
- Cobweb coagulum



Q4.15: What is the typical clinical history of tubercular meningitis?

Answer:

- Longer history (for a couple of weeks)
- Gradual onset of neurological deficit
- In pediatrics, it can be associated with hydrocephalus

Q4.16: How do CSF findings differ in pyogenic (bacterial) meningitis?

Answer: Predominant cells: Neutrophils

Q4.17: What is seen in acute bacterial meningitis regarding CSF sugar?

Answer:

- Marked reduction in sugar value
- A condition termed hypoglycorrhachia

Q4.18: What is the normal CSF protein value, and how does it change in tubercular meningitis?

Answer:

- Normal: 15 - 45 mg%
- In TB meningitis, 10-20 times higher than normal

Q4.19: What is the most common organism in acute bacterial meningitis in adults and children?

Answer: Streptococcus pneumoniae (Pneumococcus)

Q4.20: Which meningitis shows albuminocytological dissociation in CSF?

Answer:

- Guillain-Barré Syndrome (GBS)
  - Ascending Symmetrical flaccid paralysis is present
  - Commonest CN involved: 7th cranial nerve
  - One of the most typical forms of GBS: A.I.D.P. (Acute Inflammatory Demyelinating Polyneuropathy)
  - Treatment of GBS: IV IG

Q4.21: What are the normal CSF protein and sugar values?

Answer: Protein: 15-45 mg%; Sugar: > 2/3 of blood sugar

Q4.22: What is the difference between various types of meningitis?

Answer: PYQ: NEET PG 2021, 2022 PYQ: INICET 2022, 2024 PYQ: FMGE 2020, 2021, 2022

Opening pressure	Appearance	Cells	Sugar	Protein	Diagnosis	Organism
↑	Turbid	>1000 PMN (Normal - 0-4 lymphocyte in CSF)	↓= 0 (Hypoglycorrhachia)	↑	ABM (Acute Bacterial Meningitis)	Pneumococcus in both adult and pediatrics.
↑	Straw coloured	>100 lymphocytes /mm^3	↓	↑↑ Cobweb coagulum	TBM (Tuberculous Meningitis)	
↑	Clear	>20 lymphocytes	Normal	↑	Viral meningitis	Enterovirus
Normal	Clear	0-4 lymphocytes	Normal	↑ Albumino-cytological dissociation	GBS (Guillain Barre Syndrome)	

Q4.23: What are the leading causes of viral encephalitis in India and other specific scenarios, along with their transmission?

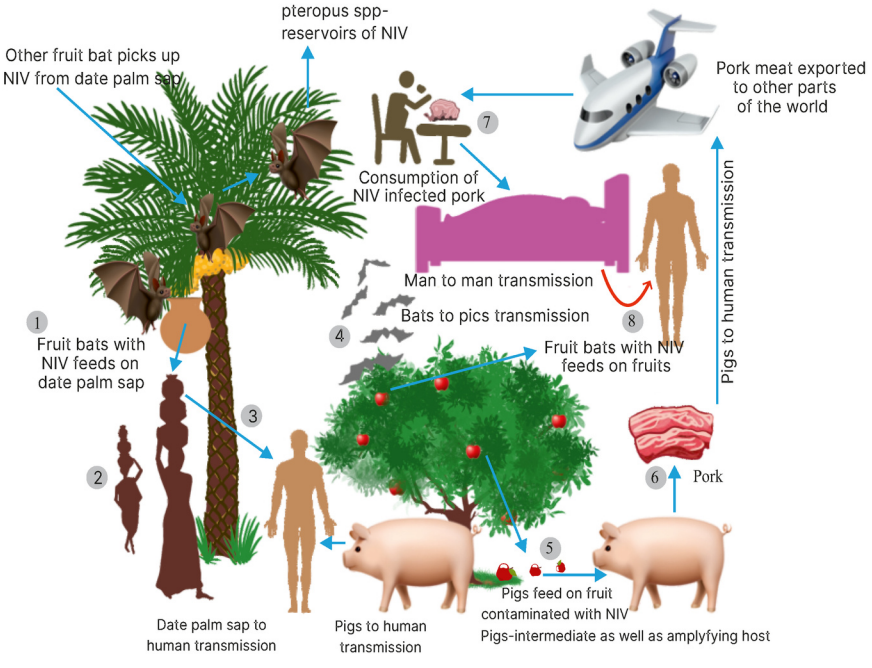
Answer:

- The leading cause of viral encephalitis in India is Japanese B encephalitis.
- The overall most common cause worldwide is Herpes Simplex Virus-1 (HSV-1).
- In Kerala outbreaks, the cause is the Nipah Virus (NiV).
- Transmission of NiV occurs through human-to-human spread
- Organism is present in human secretions

Q4.24: What was the cause of the Kerala outbreak of encephalitis, and how is it transmitted?

Answer:

- Causative factor: Nipah virus
- Reservoir: *Pteropus* bats
- Humans can get infected in 2 ways:
  - o A bat sitting on a date palm; there is contamination of date palm sap by the saliva or feces of the bat
  - o Fruit consumption - Fruits might fall from a tree and be consumed by a pig, which gets infected, and subsequently, people who eat pork might get infected. If this pork is exported to a different country, then the people there might also get infected



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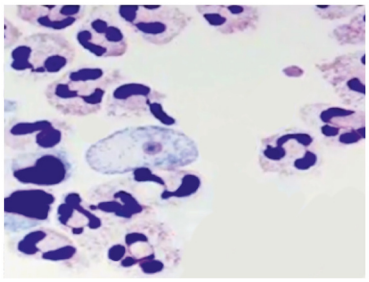
Q4.25: What are the clinical features and diagnostic methods for Nipah virus infection?

Answer:

- Encephalitis ± ARDS
- Diagnosis by PCR of the CSF sample
- No specific treatment

Q4.26: What are the features of primary amoebic meningoencephalitis (PAM)?

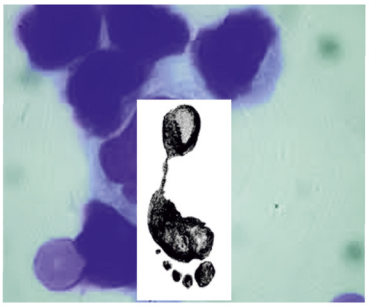
Answer:



- Organism: Naegleria fowleri (nasal route)
- Risk: Swimming in stagnant water ((like pond), rarely swimming pools
- Clinical: Meningitis + encephalitis
- CSF: Neutrophils (multilobed), trophozoites swimming
- Treatment:
  - Liposomal Amphotericin B
  - Rifampicin
  - Corifungin (new drug)

Q4.27: What is Mollaret meningitis and its cause?

Answer:



- It is a variant of aseptic meningitis
- CSF shows: Monocytes
  - Nucleus of monocyte has clefts present, which looks like toes - Mollaret cells
- Mollaret meningitis is caused by HSV-2
- HSV-2 is responsible for the development of recurrent aseptic meningitis

Q4.28: What are the common clinical features of meningitis?

Answer:

- Fever
- Headache
- Nuchal rigidity
- Vomiting (due to raised ICP)
- Sleepiness
- Irritability
- Delirium (if diagnosis is delayed)

Q4.29: What are the important examination signs for meningitis?

Answer:

- For demonstration of nuchal rigidity
  - Patient should be cooperative, ask the patient to touch his chin with his chest
- Kernig sign (knee extension):
  - Patient is lying supine on a hospital bed, put one hand just below the ankle of the patient and the second hand just adjacent to the patella, there is slight extension of the leg
  - On examination - With slight extension of the leg upward, the patient complains of pain with respect to the hamstring muscle or discomfort with respect to the back

- Brudzinski sign:
  - Ask the patient to flex his neck. When there is flexion of the neck, there would be flexion at the hip joint and the knee joint
  - Passive flexion of the neck triggers passive flexion of the hip and knee joint -
- AMOSS sign/Tripod sign
- Jolt accentuation test:
  - Ask patient to turn head left to right 2-3 times/sec or faster, it will be excruciatingly painful
  - False positive - In people with less tolerance for pain, like with headaches



Kernig's sign



Brudzinski sign

Q4.30: What is the workup in suspected meningitis?

Answer:

- Blood culture: Detects hematogenous spread; antibiotics should be started within 1 hour.
- NCCT head: To rule out raised ICP/midline shift before lumbar puncture.
- Lumbar puncture:
  - Site for L.P:
    → The Doctor would evaluate both sites L3-L4 and L4-L5, whichever is bigger, wider, and gives better access
  - Quincke needle (with pointed edge, traumatic needle)
    → Tear in dura mater; therefore, post-lumbar puncture headache is a complication
  - Sprotte needle (atraumatic needle)
    → Used with experience, and it has a blunted cutting edge; the tear of the fibers of the dura mater is relatively less
  - Advance the needle slightly cephalad (with bevel upwards)
  - On obtaining CSF, replace the stylet of the lumbar puncture needle halfway to prevent sudden escape of CSF to reduce the chance of development of trans tentorial herniation

Q4.31: What are the uses of the different tubes in CSF analysis?

Answer:

- Tube 1: Glucose & protein (biochemistry).
- Tube 2: Cell count & differential (hematology).
- Tube 3: Gram stain, culture, latex agglutination (immunology).
- Tube 4: Reserved for repeat or additional studies.

Q4.32: What are the common causative organisms of acute bacterial meningitis by age group?

Answer:

- Neonates:
  - Group B streptococcus/ Streptococcus agalactiae (most common)
  - *Listeria monocytogenes* (tumbling motility, PALCAM media, treated with ampicillin).
- Children:
  - *Streptococcus pneumoniae*
  - *Haemophilus influenzae* (can cause post-meningitis deafness in unvaccinated children).

BIG QUESTION 5: Describe neurological conditions with special radiological features and their key clinical associations.

Broad Answer: Certain neurological disorders have characteristic MRI or imaging appearances that aid in diagnosis, often correlating with specific pathological processes such as metal deposition, demyelination, or structural atrophy.

DETAILED QUESTIONS

Q5.1: What is Wilson's disease and its characteristic MRI finding?

Answer:

- Caused by copper toxicity
- It is autosomal recessive, with a defective ATP7B gene
- Excess copper is deposited in the basal ganglia and is therefore also called hepatolenticular degeneration
- Basal ganglia: Lenticular nucleus
- MRI head: Giant face of Panda
  - Panda sign is seen in sarcoidosis with Gallium-67 scan
- Neurological feature:
  - All excess copper deposited in the liver will be transported through RBCs to the brain and basal ganglia
  - Dystonia, tremors, rigidity
- Management
  - Copper chelators: Trientine, D-Penicillamine
  - DOC (in maintenance phase): Zinc acetate
  - DOC for neurological features: Tetrathiomolybdate

Q5.2. What is NBIA, and what is its MRI hallmark feature?

Answer:

- Neurodegeneration with Brain Iron Accumulation (Hallervorden-Spatz disease)
- Pathology: Iron deposition in the globus pallidus in the basal ganglia
- MRI feature: “Eye of the tiger” sign

Q5.3. Which disorder shows “Tigroid appearance” on MRI?

Answer:

- Condition: Pelizaeus-Merzbacher syndrome
- Pathology: Leukodystrophy with hypomyelination of the brain white matter
- MRI: Tigroid pattern due to alternating stripes of normal and abnormal myelination

Q5.4. What pathological change occurs in the brain in multiple sclerosis?

Answer: There is **plaque formation** in the brain, also called **Dawson's finger**.

Q5.5. Which cranial nerve is commonly involved in Multiple Sclerosis?

Answer: The **optic nerve** is involved.

Q5.6. What is the typical presentation of a patient with Multiple Sclerosis?

Answer:

- Usually a **female patient**, mainly of **Caucasian origin / Northern Europe / British women**, presenting with:

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- One-sided decreased visual acuity
- Relative afferent pupillary defect (RAPD)
- Optic neuritis

Q5.7. What type of disorder is Multiple Sclerosis?
Answer: It is a **demyelinating disorder of the brain.**

Q5.8. What are the MRI findings in Multiple Sclerosis?
Answer:

- On **MRI head with Gadolinium contrast** (due to BBB damage):
  - Dawson's fingers
  - Periventricular lesions

Q5.9. What is the difference in optic neuritis between NMOSD and MS?
Answer:

- **NMOSD (Neuromyelitis Optica Spectrum Disorder):** Bilateral optic neuritis, sudden onset
- **MS:** Unilateral optic neuritis, gradual, slow process → seen in **Secondary Progressive MS (SPMS)**

Q5.10. What is the treatment for an acute episode/first presentation of MS (e.g., visual blurring, decreased color vision)?
Answer: Methylprednisolone

Q5.11. What are the drugs used for prevention in MS?
Answer:

- ß-Interferon
- Glatiramer
- Monoclonal antibodies (worst cases), e.g., Natalizumab

Q5.12. What is the treatment of Primary Progressive Multiple Sclerosis (PPMS)?
Answer: Ocrelizumab

Q5.13. What is the treatment of Secondary Progressive Multiple Sclerosis (SPMS)?
Answer: Siponimod

Q5.14. What is Progressive Supranuclear Gaze Palsy (PSP) and its MRI finding?
Answer:

- Seen in Atypical Parkinsonism
- Presentation: Patient having rigidity or bradykinesia
  - Vertical gaze palsy: Difficulty in looking downwards: Recurrent falls in the backward direction
  - In a typical Parkinsonian person, they walk slowly, they will not be able to lift their foot over an obstacle, and might hit against a stone/brick, topple over, and fall forward
- Electrooculography finding: Square wave jerks
- Brain area involved: Basal ganglia and superior colliculus
- MRI head: Hummingbird appearance
- No drug of choice for management: Poor prognosis

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Q5.15. Which neurotransmitters are affected in caudate nucleus atrophy-related disorders?

Answer:

- **Dopamine:** Increased
- **GABA:** Decreased

Q5.16. How do dopamine levels differ between Huntington's chorea and Parkinsonism?

Answer:

- **Huntington's chorea:** Dopamine ↑
- **Parkinsonism (typical or atypical):** Dopamine ↓.

Q5.17. What is the typical age of presentation in Huntington's chorea?

Answer: Huntington's chorea typically presents in adults between **50-60 years of age**.

Q5.18. How does Sydenham chorea differ from Huntington's chorea in presentation?

Answer:

- **Huntington's chorea:** Adult onset (50-60 years).
- **Sydenham chorea:** Pediatric presentation, associated with **rheumatic fever**.

Q5.19. What are the MRI head findings in caudate nucleus atrophy and related conditions?

Answer:

- **Caudate nucleus atrophy:** Boxcar ventricle
- **Callosal dysgenesis:** Racing car appearance

Q5.20: Which cardiac catheterization sign is seen in constrictive pericarditis?

Answer: **Finding:** Square root wave sign.

BIG QUESTION 6: Discuss hyperkinetic movement disorders, their causes, clinical features, and examples.

Broad Answer: Hyperkinetic movement disorders involve involuntary excessive movements without weakness, often due to basal ganglia lesions, and include conditions like chorea, athetosis, and hemiballismus, with varied causes such as genetic mutations, rheumatic fever, or metabolic derangements.

DETAILED QUESTIONS

Q6.1. Differentiate between chorea, athetosis, and hemiballismus.

Answer:

Chorea	Athetosis	Hemiballismus
• It is defined as fast distal involuntary movement found in hands of the patient • This would be purposeless or semi purposive	• Athetoid cerebral palsy - Complication of kernicterus • Basal ganglia involved: Globus pallidus • Athetosis: Slow, distal, writhing involuntary movement in hands	• There are wide flinging movements of arms • Seen with patients of nonketotic hyperosmolar coma

• Coordination with respect to handwriting would be difficult • Falling school grades especially in pediatric presentation in Sydenham chorea with rheumatic fever • Lesion is present in caudate nucleus	• In athetoid cerebral palsy there will be intellectual deterioration subsequently	• Hyperosmolar coma is complication more common with Type II Diabetes mellitus • In Hemiballismus: Involuntary Movement will be present and is both proximal and distal

Q6.2. Differentiate Huntington's chorea and Sydenham's chorea.

Answer:

Huntington chorea	Sydenham chorea
Autosomal dominant, have anticipation (severity of the disease increases with every successive generation)	Manifestation of rheumatic fever i.e Type II hypersensitivity reaction
Huntington gene, Chromosome 4	Recurrent sore throat
Dopamine , GABA level^{-}	Elevated ASO Titres
Adult > 50 years, Presentation o Dementia o Chorea	Child: Falling school grades Classical manifestation • Darting tongue • Milk-Maid grip
Treatment: Tetrabenazine (dopamine depletor)	Self-limiting disease If disabling, affect quality of life- Give Valproate, Steroids

Q6.3. Explain the paradox of hyperkinetic features in Parkinson's disease.

Answer:

- **Hyperkinetic movement disorders**
  - Characterized by **involuntary movements unaccompanied by weakness.**
  - The term is somewhat arbitrary and potentially misleading, as **hypokinetic disorders (e.g., Parkinson's disease)** are often accompanied by **tremor**, which is a hyperkinetic feature.
- **Hypokinesia**
  - A diagnostic feature of **Parkinsonism.**
  - Progresses from **bradykinesia** → **akinesia** → **wheelchair bound.**
- **Tremors in Parkinsonism**
  - Although Parkinsonism is a **hypokinetic disorder**, tremors are a **hyperkinetic feature** seen in it.

Q6.4. Describe the hung-up reflexes and ankle jerk

Answer:

- Hung up reflexes: Feature of chorea
- Hung up ankle jerk
  - Seen with a patient having myxoedema of the Achilles tendon
  - The relaxation phase of the reflex is slow
  - Seen in hypothyroidism

Q6.5. What is the defective gene in Huntington's chorea?

Answer: The defective gene is the **Huntington gene,** which contains **trinucleotide repeats.**

Q6.6. Which neurodegenerative disorder is associated with hexanucleotide repeats?

Answer: **Amyotrophic lateral sclerosis (ALS)** is associated with a **hexanucleotide repeat disorder.**

Q6.7. Huntington's chorea is an example of which type of condition?

Answer: **Huntington's chorea** is an example of a **neurodegenerative condition.**

Q6.8. What is the role of IVIG in chorea?

Answer: **IVIG (Intravenous Immunoglobulin)** is useful in cases of **medically refractory chorea.**

BIG QUESTION 7: Outline the types, common sites, clinical features, and key imaging findings of brain tumors in adults and children.

Broad Answer:

- Brain tumors may be secondary (most common overall) or primary, with meningioma being the most frequent primary tumor in adults and cerebellar astrocytoma being common in children.
- Tumor location often differs by age, with adult tumors usually supratentorial and pediatric tumors infratentorial.
- Diagnosis relies on gadolinium-enhanced MRI, and prognosis varies widely depending on tumor type.

DETAILED QUESTIONS

Q7.1. What is the most common brain tumor overall and by category?

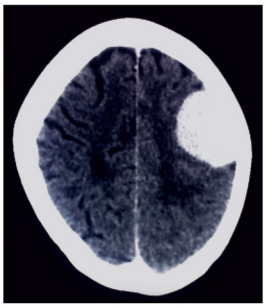
Answer:

- Overall, the most common: 2° / Metastasis
  - Oat cell cancer of the lung / Small cell cancer - Micrometastasis
  - Breast cancer
  - Malignant melanoma
- Primary most common: Meningioma (34%)
- Sequence: 2° > Meningioma > Glioma
- Gadolinium MRI head (IOC): Multiple sclerosis, Brain tumors (to demonstrate cerebral edema)
- Pediatric brain tumor commonly seen: Cerebellar astrocytoma
  - Most of the tumors seen in Pediatrics are infratentorial UYGVKI C CFTR
- In adults, 2/3rd of tumors are supratentorial

Q7.2. Describe the origin, imaging features, and histology of meningioma.

Answer:

- Cell of origin for meningioma: Arachnoid capsules
- NCCT intracranial calcification
- It is an extra-axial brain tumor
- There is hyperostosis of calvarium / Lytic lesions (It can grow from inside & cause erosion of calvarium)
- Histopathology: Presence of Psammoma bodies
- Radiological finding: Dural tail sign and Sunburst appearance of blood vessels (contrast)
- Meningioma can arise as a complication of therapeutic radiation



Q7.3. How do brain tumors typically present clinically?

Answer:

- Headache (worse in the morning, with Valsalva, bending forward, or rising from lying position)
- Altered mentation
- Focal seizures
- Speech defects
- Gait abnormalities, head tilt, gait ataxia
- Gaze abnormalities
- Visual field defects – Bitemporal hemianopia

Q7.4. Which brain tumors have the worst prognosis?

Answer:

- Pediatric: Brainstem glioma, medulloblastoma, cerebellar astrocytoma (pilocytic – prognosis better than medulloblastoma).
- Adult: Grade IV glioma (glioblastoma multiforme).

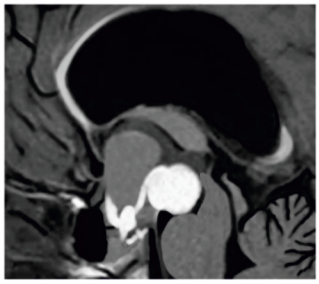
Q7.5. What are leptomeningeal metastases, and how are they diagnosed?

Answer:

- Cause: Commonly from breast cancer.
- Diagnosis:
  - Gadolinium-enhanced MRI
  - lumbar puncture with CSF cytology showing malignant cells.

Q7.6. Describe craniopharyngioma and its key associations.

Answer:

Craniopharyngioma	
	• MRI head: A cystic tumor with machinery oil consistency along with intracranial calcification PYQ: NEET PG 2019 • Craniopharyngioma: Supratentorial tumor • Clinical presentation 1. Short stature 2. Central diabetes insipidus: Polyuria, polydipsia 3. Visual deficit • Craniopharyngiomas: Related to Rathke's pouch and are situated near pituitary • It is a supratentorial pediatric brain tumor but is rare

Q7.7. In which conditions can psammoma bodies be seen?

Answer:

- Papillary carcinoma of the kidney and thyroid
- Serous cyst adenoma related to the ovary
- Meningioma

Q7.8. In which conditions can intracranial calcifications be seen?

Answer:

- Neurocysticercosis

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- TORCH (in Pediatrics)
- Meningioma
- Craniopharyngioma

Q7.9. What is the overall most common tumor in children?

Answer: Acute lymphoblastic leukemia (ALL) is the most common tumor in children, followed by brain tumors.

Q7.10. What is the most common infratentorial tumor in children?

Answer: The most common infratentorial tumor in children is cerebellar astrocytoma (pilocytic type).

Q7.11. What is the most common malignant brain tumor in children (infratentorial)?

Answer: The most common malignant brain tumor in children (infratentorial) is medulloblastoma.

Q7.12. Why does medulloblastoma have a bad prognosis?

Answer:

- Medulloblastoma can spread from the brain to the spinal cord (cranio-spinal axis spread).
- This spread can cause neurological deterioration or the development of obstructive hydrocephalus.

Q7.13. What is the IOC diagnosis of brain tumors?

Answer: Gadolinium-enhanced MRI

BIG QUESTION 8: What are the major neurocutaneous disorders, and what are their key clinical and treatment aspects?

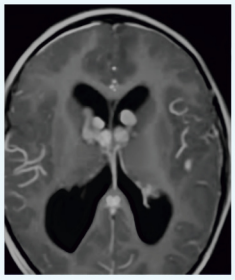
Answer:

- Neurocutaneous disorders include tuberous sclerosis, Sturge-Weber syndrome, Neurofibromatosis, Ataxia-telangiectasia, and von Hippel-Lindau disease.
- Infantile spasms are treated with ACTH, except in tuberous sclerosis, where vigabatrin is preferred.

DETAILED QUESTIONS

Q8.1: What are the cutaneous lesions in tuberous sclerosis?

Answer:

Tuberous Sclerosis	
	• Autosomal dominant inheritance • Gene defect seen: o TSC 1 gene: Chromosome 9 o TSC 2 gene: Chromosome 16 • Ashleaf macule: One of the earliest cutaneous manifestations • Neurological manifestations: Epilepsy • Presentation: epileptic + cutaneous lesion o This cutaneous lesion - Acneiform lesion with butterfly distribution usually appears at age of puberty is **Adenoma sebaceum**


Ashleaf macule


Adenoma Sebaceum

- Shagreen patch: Found on trunk of patients
  - o Orange peel consistency irregular consistency
- General physical examination
  - o Examination of foot: Fleshy growths at the root of nail
    - → This is subungual fibroma originating from below the nail causing nail to crack and called KOENEN tumor
- CNS manifestations
  - o In first year of life: Infantile spasm
    - → Management: Vigabatrin
  - o > 1 year: Focal seizures
    - → Management: Carbamazepine (lifelong)
  - o Child has epilepsy since birth so would develop mental retardation
- MRI Brain shows Subependymal astrocytoma (looks like potatoes growing in the brain responsible for causing focal seizures)

Q8.2: What are the cutaneous lesions seen in Sturge-Weber syndrome?

Answer:

Sturge Weber Syndrome

- S - Seizures (Focal): Due to vascular malformation in brain i.e., called as **Cavernous Angioma**
  - o Cutaneous manifestation: Port wine stain around eyes
- T - Trigeminal nerve distribution: **Port wine stain**
- U - Unilateral weakness: Due to tumor compressing the corticospinal pathway
- R - Mental Retardation PYQ: FMGE 2022
- G - Glaucoma
- E - Buphthalmos
- CT head: Tram track appearance (also seen in membranoproliferative glomerulonephritis, bronchiectasis) or rail road calcification

Q8.3: What are the key features seen in NF1 and Nf2?

Answer:

Neurofibromatosis

- Two types
  - o NF 1
  - o NF 2
- Important features of NF 1
  - o **Hypertension** because of association with pheochromocytoma
  - o It is autosomal dominant disorder with defect on chromosome 17
  - o Neurological manifestation: **Optic glioma**
- For NF 2: **Acoustic neuroma**
- Cutaneous manifestation
  - o ≥ 6 C.A.L.M (Café-au-lait macules), Size: ≥ 5mm
  - o Freckles in axilla or inguinal area of patient
  - o Pulsation of cavernous sinus: thinning of sphenoid bone
  - o Lisch nodules: Iris hamartoma

Q8.4: Which immune deficiency is seen in ataxia-telangiectasia?

Answer:

Ataxia-Telangiectasia	• Telangiectasia: Dilated blood vessels • Ataxia: Spinocerebellar pathway is involved • Deficiency of IgA • Increased incidence of blood cancer

Q8.5: Which chromosome is affected in von Hippel-Lindau disease, and what is its major malignancy risk?

Answer:

V.H.L (von Hippel-Lindau)	• Cutaneous manifestations: C.A.L.M (Café-au-lait macules) • Defect in chromosome 3, VHL gene involved • Development of malignancy like renal cell carcinoma • CNS manifestation o Cerebellar hemangioblastoma: Tumor is originating from blood vessels of cerebellum o Retinal hemangioblastoma: Tumor originating from blood vessels related to retina → Lesions can bleed causing vision loss o Spinal cord hemangioblastoma - Vascular lesion of spinal cord

BIG QUESTION 9: Discuss Neuromyelitis Optica Spectrum Disorder (NMOSD) — its etiology, clinical features, diagnosis, and management.

Broad Answer:

- NMOSD is a female-predominant autoimmune astrocytopathy targeting aquaporin-4 water channels, leading to optic neuritis, longitudinally extensive transverse myelitis, and other CNS syndromes.
- Diagnosis is based on clinical core features with antibody or MRI confirmation, and treatment includes steroids, plasmapheresis, and immunosuppressants.

DETAILED QUESTIONS

Q9.1 What is the female-to-male ratio in NMOSD?

Answer: 3:1

Q9.2 Which cells are primarily affected in NMOSD, and what is the consequence?

Answer:

- Astrocytes: Damage to their foot processes disrupts the blood-brain barrier, causing cerebral edema and possible death
- The disease caused: Astrocytopathy

Q9.3 What is the cause of death in NMOSD?

Answer:

- Death occurs due to involvement of the spinal cord at C3, C4, and C5
  - The phrenic nerve supply to the diaphragm is affected, resulting in respiratory failure
  - Most of the time, in neurological illnesses like Parkinsonism or Alzheimer's
- Death is caused by pneumonia as patients become bedridden, which increases the chances of aspiration

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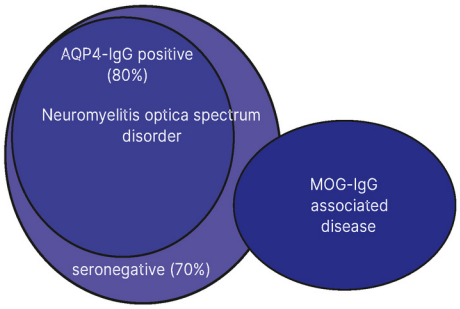
Q9.4 Name the two antibodies associated with NMOSD.

Answer: Anti-aquaporin-4 antibody and anti-MOG (myelin oligodendrocyte glycoprotein) antibody



Q9.5 List the core clinical features of NMOSD.

Answer:

- Core clinical features of NMOSD
  - Optic neuritis
  - Longitudinally extensive transverse myelitis
  - Manifestations of spectrum disorder

Q9.6 Give examples of NMOSD spectrum disorder manifestations.

Answer:

- Area postrema syndrome (vomiting)
- Brainstem syndrome (cranial nerve palsy)
- Cerebral syndrome (seizures)
- Diencephalic syndrome (weight loss, anorexia)
- Narcolepsy (due to RAS damage)

Q9.7 What is the MRI finding in NMOSD?

Answer:

- Optic neuritis
- MRI spine: > 3 segments in the spinal cord involved should be demonstrated
- Cloud-like lesions were demonstrated in the brain
  - It can be in the parenchyma or the brain stem
  - Brain stem involvement can cause cranial nerve palsy

Q9.8 State the diagnostic criteria for NMOSD.

Answer:

- ≥ 1 core clinical feature + anti-aquaporin-4 antibody, OR
- If antibody negative: ≥ 2 core clinical features + gadolinium-enhanced MRI evidence.

Q9.9 Which drugs are contraindicated in NMOSD?

Answer: ß-interferon and glatiramer acetate

Q9.10 How is acute NMOSD treated?

Answer: High-dose IV methylprednisolone; if no improvement, plasmapheresis

Q9.11 Name the immunomodulators used for NMOSD prevention.

Answer: Mycophenolate mofetil, azathioprine (combined with steroids)

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Q9.12 Therapeutic plasmapheresis is indicated in which condition?

Answer:

- Hemolytic uremic syndrome
- Thrombotic thrombocytopenic purpura
- NMOSD

Q9.13: What is the ocular manifestation of NMOSD?

Answer: Optic neuritis.

Q9.14: How is optic neuritis different in Multiple Sclerosis compared to NMOSD?

Answer:

- In **Multiple Sclerosis**, the course is **relapsing and remitting**, with mostly **unilateral ocular deficit**.
- In NMOSD, blindness can be **sudden and bilateral**.

Q9.15: Which cranial nerve is involved in NMOSD presenting with sudden blindness?

Answer: The **Optic nerve (Cranial nerve II)**.

Q9.16: What is Longitudinal Extensive Transverse Myelitis (L.E.T.M.), and how is it defined?

Answer: L.E.T.M. is defined as **involvement of ≥ 3 consecutive spinal cord segments**.

Q9.17: What are the typical symptoms associated with L.E.T.M. in NMOSD?

Answer:

- Pin and needle sensation in extremities
- Root pain
- Radicular pain below a particular level

BIG QUESTION 10: What are the causes, clinical features, diagnostic criteria and management of Guillain-Barré Syndrome (GBS)?

Broad Answer:

- Guillain-Barré Syndrome is an acute demyelinating polyneuropathy affecting the peripheral nervous system, most often occurring 2-3 weeks after a respiratory or gastrointestinal infection.
- The antibody responsible is the Anti-GM1 antibody.
- It presents with bilateral ascending symmetrical flaccid paralysis, quadriplegia, truncal weakness, neck floppiness, and sometimes facial diplegia due to 7th nerve palsy.
- The diagnostic criteria used to identify GBS are the Brighton criteria.

DETAILED QUESTIONS

Q10.1: What are the clinical manifestations of GBS?

Answer:

- GBS usually appears 2-3 weeks after a respiratory/ digestive tract infection
- Presentation:
  - Bilateral ascending symmetrical flaccid paralysis

- Equal paralysis of left & right leg- Quadriplegia
- Truncal paralysis
- Demyelination of the cervical spine (respiratory paralysis)
- Neck floppiness
- Facial nerve involvement leads to 7th nerve palsy - Facial diplegia

Q10.2: Which antibody is associated with Miller Fisher syndrome?

Answer: Anti-GQ1 antibody

Q10.3: What are the causes of facial diplegia?

Answer:

- Sarcoidosis
- Melkersson-Rosenthal syndrome
- Guillain-Barré syndrome

Q10.4: Differentiate GBS from transverse myelitis.

Answer:

- GBS: Bladder and bowel function spared
- Transverse myelitis: Bladder and bowel involved
  - Root pain
  - Urinary incontinence

Q10.5: What are the lumbar puncture findings seen in GBS?

Answer:

- Albumino-cytological dissociation
- CSF opening pressure is normal
- Cells: normal
- Sugar: normal
- Color: normal
- Protein: ↑
- The earliest manifestation of GBS is distal areflexia

Q10.6: What is the Brighton Criteria for GBS?

Answer:

- Bilateral and flaccid weakness of limbs
- Decreased or absent deep tendon reflexes in weak limbs
- Monophasic course and time between onset-nadir = 12 hours to 28 days
- Absence of an alternative diagnosis for weakness
- CSF cell count <50/ml
- CSF protein concentration >60 mg/dL
- Nerve conduction study findings consistent with one of the subtypes of GBS

Q10.7: What is the management of GBS?

Answer:

- IV Immunoglobulins (DOC)
- Plasmapheresis
- The efficacy of both IV immunoglobulin and Plasmapheresis is equal

Q10.8: What is the difference between GBS, transverse myelitis, Miller-Fisher syndrome, and NMOSD?

Answer:

GBS (Guillain Barre Syndrome)	Transverse Myelitis	Miller Fisher Syndrome	Neuromyelitis optica spectrum disorders (NMOSD)
Ascending Symmetrical flaccid paralysis	Ascending Symmetrical flaccid paralysis	Antibody: anti GQ1 Antibody	Antibody: anti Aquaporin 4 Antibody
Diaphragmatic paralysis Autonomic insufficiency	Diaphragmatic paralysis, Autonomic insufficiency	Triad: OAA • Ophthalmoplegia (3rd nerve palsy) • Areflexia • Ataxia	Optic Neuritis + Longitudinal extensive transverse myelitis
Sensory: Paresthesia	Sensory: Root pain	-	Earlier, NMOSD was known as Devic's disease
Bladder/bowel spared	Bladder/bowel involved	-	-
Test: Nerve conduction test / Albumino cytological dissociation	MRI spine	-	-
Rx: IV IG	Rx: IV Methylprednisolone	Rx: IV IG	Rx: IV Methylprednisolone

- In Transverse myelitis, initially, features of spinal shock (areflexia) are present.
- Once the disease is established, whichever level the spinal cord is involved (e.g., T6), at the level of T6, features of the lower motor and above T6 features of the upper motor nervous system are present.
- These manifestations take time to develop.

Q10.9: Which criteria are used for the identification of Multiple Sclerosis?

Answer: The revised McDonald criteria are used for the identification of Multiple Sclerosis

BIG QUESTION 11: Discuss the clinical features, diagnostic workup, and pharmacological management of Chronic Inflammatory Demyelinating Polyneuropathy (CIDP)?

Broad Answer: CIDP is a chronic demyelinating neuropathy mimicking GBS but with >9 weeks progression, managed with steroids. Key CNS drugs include tetrabenazine for Huntington's, trihexyphenidyl for drug-induced Parkinsonism, and methylprednisolone for acute demyelinating conditions.

DETAILED QUESTIONS

Q11.1: What are the clinical features of CIDP?

Answer:

- CIDP presents with symmetrical flaccid paralysis
- Progression lasts > 9 weeks (GBS is < 4 weeks)

- Mimics GBS in the early phase, but is chronic
- Managed with IV methylprednisolone

Q11.2: What are the first-line drugs used in specific neurological disorders?

Answer:

- Huntington's disease → Tetrabenazine
- Wilson's disease (CNS features) → Tetrathiomolybdate
- Drug-induced Parkinsonism → Trihexyphenidyl / Benzhexol
- Guillain-Barré syndrome → IV Immunoglobulin
- Transverse myelitis / NMOSD / CIDP → Methylprednisolone
- Multiple sclerosis
  - Acute attack → Methylprednisolone
  - Prevention → ß-interferon
- Parkinson's disease → Levodopa + Carbidopa

Q11.3: What is the etiology of Variant Creutzfeldt-Jakob disease?

Answer:

- VCJD is a Prion disease (prion particle - the smallest living infectious particle)
- It has only a protein coat (no DNA/RNA)
- Resistant to heat at 121 °C
- After consumption of tainted beef (bovine spongiform encephalopathy), prion particles in beef cross the blood-brain barrier
- PrPSC is the abnormal protein involved in the disease

Q11.4: What are the key clinical manifestations of VCJD?

Answer:

1. Myoclonic jerks (Startle myoclonus)
2. Dementia
3. Gait ataxia
4. Progressive incapacitation → bedridden → death (often due to pneumonia).

Q11.5. What are the diagnostic findings in VCJD?

Answer:

- CSF: Elevated 14-3-3 protein
- MRI brain: Cortical ribboning / Hockey stick sign
- EEG: Periodic sharp wave complexes

Hockey stick sign

- In ECG: Digoxin
- In Echo: Mitral stenosis

Q11.6. What are the diseases caused by prions?

Answer:

- Variant Creutzfeldt-Jakob disease
- Classical Creutzfeldt-Jakob disease

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- Kuru disease (cannibalism)
- Familial fatal insomnia - chromosome 20 defect
- Gerstmann-Sträussler-Scheinker syndrome - chromosome 20 defect

BIG QUESTION 12: Discuss the genetic basis, clinical features, diagnosis, and management of different muscular dystrophies.

Broad Answer:

- Duchenne muscular dystrophy is an X-linked recessive dystrophin gene defect causing progressive muscle weakness, wheelchair dependence by adolescence, and death from heart failure.
- Charcot-Marie-Tooth is a hereditary neuropathy with distal muscle wasting and onion-skin nerve changes.
- Myasthenia gravis is an autoimmune post-synaptic NMJ disorder with diurnal ptosis and diplopia, linked to thymic abnormalities, diagnosed with antibody testing and EMG, and treated with pyridostigmine or thymectomy.
- Lambert-Eaton syndrome is a presynaptic NMJ disorder, often paraneoplastic, presenting with proximal weakness and incremental EMG response.
- Motor neuron disease involves progressive UMN and LMN degeneration, with ALS as the mixed form, leading to respiratory failure.

DETAILED QUESTIONS

Q12.1: What is the genetic defect in Duchenne muscular dystrophy (DMD), and how does it differ from Becker's?

Answer:

- **Gene:** Dystrophin gene (X-linked recessive)
- **DMD:** Complete absence of dystrophin → severe, early onset
- **Becker's:** Reduced dystrophin → milder, later onset

Q12.2: What are the hallmark features of DMD?

Answer:

Duchenne Muscular Dystrophy	• It is a X-linked Recessive disorder: Mainly affects boys • Due to defect in Dystrophin gene PYQ: FMGE 2021 PYQ: INICET 2022 PYQ: NEET PG 2023 o If Dystrophin protein is less, the disease is known as Becker's muscular dystrophy (milder variant) o If Dystrophin protein is totally absent in Duchenne muscular dystrophy- Progressive increase in muscle weakness. • Defect in dystrophin gene leads to inhibition of calcium uniport in mitochondria. • Duchenne muscular dystrophy children presents with recurrent falls while walking, waddling gait, GOWER sign (difficulty in standing from sitting in cross legged position is due to weakness in the hip joint muscle), pseudohypertrophy of calf muscles and brachioradialis (floppy) • Because of these symptoms, most of the children become Wheelchair bound at 10-12 years of age, subsequently leading to death. • Main cause of death in Duchenne muscular dystrophy is Congestive Heart Failure. • Investigation of Choice: PCR for dystrophin gene.

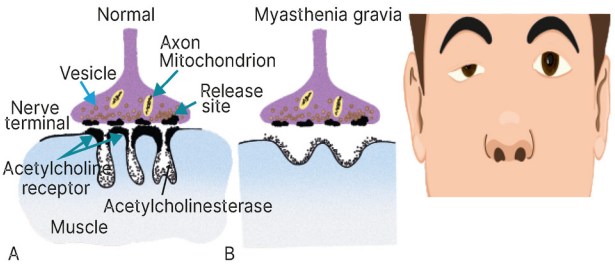
Q12.3: What is Charcot-Marie-Tooth disease and its classic signs?

Answer:

Charcot Marie Tooth Disease	• Patients presents with history of Recurrent falls • It is a Hereditary sensorimotor neuropathy • Nerve biopsy shows Onion Skinning appearance • Clinical features: 1. Stork leg appearance (peroneal muscle atrophy) 2. Muscle wasting 3. Thickened nerves • No definitive treatment

Q12.4 What is the pathophysiology and clinical features of Myasthenia gravis (MG)?

Answer:

Myasthenia Gravis  PYQ: INICET 2021, 2022 PYQ: NEET PG 2022 PYQ: FMGE 2021, 2022	• Myasthenia gravis occurs in young females, also occurs in male of age group > 50 years • It has auto antibodies which affects Neuromuscular junction • Due to changes in NMJ, receptors are blocked called Post-junctional defects. • Auto antibodies formed are Anti Ach receptor blocking antibody • Among 75% of Myasthenia gravis patient have a Thymus abnormality (Thymoma or Thymic hyperplasia) • Earliest manifestation of MG: Asymmetrical ptosis • Clinical features 1. Asymmetrical ptosis- Diurnal variations of symptoms (doesn't present in morning hours, progresses through the day) 2. Diplopia 3. Jaw muscle weakness (Chewing complaints) 4. Dysarthria 5. Shoulder Girdle muscle weakness

Q12.5: How is Myasthenia gravis diagnosed?

Answer:

- Screening
  - Anti-ACh receptor blocking antibody titer
  - Anti MuSK antibody (Muscle-specific kinase) - Ocular myasthenia gravis
- IOC
  - Single fiber Electromyography (SF EMG)
  - Repetitive nerve (+) test (decremental response)
- Tensilon test
  - In MG, Tensilon tests are not done nowadays due to the cholinergic crisis, which causes hypersalivation
  - If Edrophonium injection is given to myasthenia gravis patients, it will reduce ptosis but cause a cholinergic crisis
- Nowadays, the ICE PACK test is done

- The sensitivity of both the tensilon test and the ice pack test is equal
- Deep tendon reflex in Myasthenia gravis is normal

Q12.6: How is Myasthenia gravis treated?

Answer:

- Pyridostigmine (adults)
- Neostigmine (neonatal MG)
- Thymectomy for generalized MG
- Steroids/azathioprine for ocular MG

Q12.7: What is Lambert-Eaton syndrome?

Answer:

Lambert Eaton Syndrome	• It is a Paraneoplastic manifestation causing decreased release of acetylcholine at the neuromuscular junction known as Pre-Junctional defect
	• Antibody responsible is anti P/Q antibody
	  o Receptors are normal
	• In Repetitive nerve stimulation test: Incremental response occur whereas weakness begins from shoulder girdle muscle
	• Age group: >50 years of age
	• This syndrome also occurs in patient who is diagnosed with oat cell cancer lung
	• Treatment
	  o Pyridostigmine is used
	  o DOC: 3,4 aminopyridine

Q12.8: What are the three main manifestations of Motor Neuron Disease (MND)?

Answer:

- MND can present as:
  - o Lower motor neuron type (flaccid weakness with absent reflex)
  - o Upper motor neuron type (spasticity with brisk reflex)
  - o Combination of LMN + UMN lesions





Q12.9. What is Progressive Muscular Atrophy in Motor Neuron Disease?

Answer:

- Progressive muscular atrophy is a **lower motor neuron type** presentation characterized by:
  - o Limb weakness with areflexia
  - o Site of lesion: **Anterior horn cells of the spinal cord**
  - o Due to the lesion, current will not flow, leading to an LMN lesion

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Q12.10. What is Progressive Bulbar Palsy in Motor Neuron Disease?

Answer:

- Progressive bulbar palsy is another LMN-type presentation, with:
  - Loss of **gag reflex**
  - Increased risk of **aspiration**

Q12.11. What is Primary Lateral Sclerosis in Motor Neuron Disease?

Answer:

- Primary lateral sclerosis is an **upper motor neuron type** presentation characterized by:
  - Spasticity
  - Brisk reflexes
  - Involvement of the **corticospinal pathway**

Q12.12. What is Amyotrophic Lateral Sclerosis (ALS) in Motor Neuron Disease?

Answer:

- ALS is a **combination of lower motor neuron and upper motor neuron lesions**, characterized by:
  - Gradual onset of weakness over the span of years
  - Asymmetrical pattern of weakness
  - Knee jerk and ankle jerk on the **left side may be brisk**
  - Biceps jerk on the **right side may be absent**
  - Death is usually due to **respiratory failure (pneumonia)**

Q12.13. What are the management options for Motor Neuron Disease?

Answer:

- Management includes: (Both drugs increase the longevity of patients)
  - **Riluzole**
  - **Edaravone**

Q12.14. In which conditions can a combination of UMN and LMN features (absent ankle jerk with extensor plantars) be seen?

Answer:

- Vitamin B12 deficiency
- Friedreich ataxia
- Amyotrophic lateral sclerosis (ALS)

Q12.15: How is CPK-MM used to monitor disease progression?

Answer:

- Elevated in age by 4 years
- In 10-12 years, muscle wasting occurs, so the CPK-MM level may be falsely normal
- In Duchenne muscular dystrophy, CPK-MM levels will fluctuate at different ages, so diagnosis doesn't depend upon CPK-MM

BIG QUESTION 13: Discuss the types of dementia, their distinguishing features, and the role of proteinopathies like tauopathy in neurodegenerative diseases.

Broad Answer: Dementia can be broadly classified into cortical and subcortical types based on the site of brain involvement, clinical features, and associated protein abnormalities. Tauopathies and other proteinopathies contribute to their pathogenesis, with conditions like Alzheimer's disease, Parkinson's disease, and others showing characteristic protein deposits and genetic associations.

DETAILED QUESTIONS

Q13.1: What are the main differences between cortical and subcortical dementia?

Answer:

Features	Cortical Dementia	Subcortical Dementia
Site of brain	Outer cortex	Subcortical gray matter
Examples	Alzheimer's disease, **Pick's disease**	Huntington's disease, **Parkinson disease (most common),** Westphal variant of HD, Progressive supranuclear gaze palsy, HIV D
Motor symptoms	Rare	Usual
Memory symptoms	Common	Less marked
Language	Aphasia present, Dysarthria absent	Aphasia absent, Dysarthria present
Calculation	Acalculia (+)	Acalculia (-)
Coordination	Preserved	Impaired
Posture	Upright	Bowed or Extended

Q13.2: What is tauopathy, and which conditions are associated with it?

Answer:

- Tau protein is a cytosolic microtubule-binding protein that stabilizes the neuronal cytoskeleton.
- Defects lead to cortical loss and dementia.
- Causes:
  - Alzheimer's disease / Down syndrome (presenile dementia ~25 years in Down's)
  - Corticobasal degeneration
  - Progressive supranuclear gaze palsy (hummingbird sign)
  - Pick's disease
- Mnemonic: ACP (Alzheimer's, Corticobasal degeneration, Pick's disease)

Q13.3: What are the protein and genetic associations of Alzheimer's disease?

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Answer:

- Proteins: Tau protein, Hirano bodies
- Genes:
  - Chromosome 1 → Presenilin 2
  - Chromosome 14 → Presenilin 1
  - Chromosome 19 → APOE4
  - Chromosome 21 → APP (Amyloid precursor protein)

Q13.4. How is Alzheimer's disease diagnosed and managed?

Answer:

- Diagnosis (4A's): Amnesia, Apraxia, Agnosia, Aphasia (parietal & temporal lobe involvement)
- Investigation of choice: Functional MRI → Hypometabolism in parietal & temporal lobes
- Screening: MMSE (<24/30 suggests dementia; false positives in depression)
- Treatment: Donepezil (acetylcholinesterase inhibitor)

Q13.5. What proteinopathy is seen in Parkinson's disease, and what test can detect apraxia?

Answer:

- Parkinson's disease: α-synuclein protein forming Lewy bodies
- Cognition defect/apraxia detection: Clock face test

7. ENDOCRINOLOGY

BIG QUESTION 1: What is Acromegaly and how is it diagnosed and managed?

Broad Answer: Acromegaly is a hormonal disorder caused by excessive growth hormone secretion, usually due to a pituitary **adenoma.** Since it develops after puberty, patients do not become taller but instead show enlargement of extremities and characteristic facial changes. Diagnosis relies on biochemical tests such as IGF-1 and glucose suppression test, supported by imaging. Treatment includes surgery, medical therapy, and newer receptor blockers.

DETAILED QUESTIONS

Q1.1: What is the cause of acromegaly?

Answer: Pituitary adenoma causing excessive GH production after puberty.

Q1.2: Why does acromegaly not increase height?

Answer: Occurs after epiphyseal plate closure post-puberty → no longitudinal bone growth.

Q1.3: What are the clinical features of acromegaly?

Answer:

- Spade-like hands (increase in hand size)
- Prognathism
- Increased foot size
- Increased heel pad thickness
- Galactorrhea

Q1.4: Which imaging is used for assessing heel pad thickness?

Answer: X-ray of foot.

Q1.5: Which anterior pituitary cells are most abundant and involved?

Answer:

- Somatotrophs (main)
- Lactotrophs (also involved)

Q1.6: What is the screening test for acromegaly?

Answer: IGF-1 levels (elevated).

Q1.7: What is the investigational gold standard for confirming acromegaly?

Answer:

- Glucose suppression test:
  - Normally, 100 g glucose suppresses GH.
  - In acromegaly, GH fails to suppress → GH >1 ng/mL.

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Q1.8: Which imaging is used for pituitary adenoma?
Answer: MRI of the head.

Q1.9: What are the treatment options for acromegaly?
Answer:
- Lanreotide (long-acting somatostatin analog)
- Trans-sphenoidal surgery
- Pegvisomant (GH receptor antagonist for recurrence)

BIG QUESTION 2: What are the recommended screening and confirmatory investigations for common endocrine disorders?
Broad Answer: Endocrine disorders are investigated using screening tests and investigation of choice (IOC)/confirmatory tests specific to each condition. Proper interpretation requires understanding of hormone levels, feedback mechanisms, and functional testing.

DETAILED QUESTIONS

Q2.1: What is the investigation of choice (IOC) for Diabetes Insipidus?
Answer: Water deprivation test

Q2.2: What is the investigation of choice for SIADH?
Answer: Water loading test

Q2.3: What is the IOC for Conn syndrome (adrenal adenoma)?
Answer: Saline infusion test

Q2.4: What is the confirmatory test for Addison disease?
Answer: ACTH stimulation test / Cosyntropin test

Q2.5: What is the screening and IOC for pheochromocytoma?
Answer:
- Screening: Plasma free metanephrine levels
- Other test: 24-hour urinary VMA
- IOC: Plasma free metanephrine

Q2.6: What is the test of choice for Carcinoid syndrome?
Answer: Urine 5-HIAA

Q2.7: What is the test of choice for Acromegaly?
Answer: Glucose challenge test

Q2.8: What is the screening and IOC for Prolactinoma?
Answer: Serum prolactin levels + MRI head

Q2.9: What is the screening and confirmatory test for Cushing's syndrome?

Answer:

- Screening: Low-dose dexamethasone suppression test
- IOC: Overnight dexamethasone suppression test / Low-dose DST

Q2.10: Which imaging is used for parathyroid adenoma?

Answer: Sestamibi scan

BIG QUESTION 3: How do water balance disorders such as Diabetes Insipidus, SIADH, Psychogenic Polydipsia, and Adipsic Hypernatremia present, and how are they diagnosed and managed?

Broad Answer: Water balance disorders result from abnormalities in antidiuretic hormone (ADH) secretion/action or thirst mechanism. Key disorders include Diabetes Insipidus (DI), Syndrome of Inappropriate ADH Secretion (SIADH), Psychogenic Polydipsia, and Adipsic Hypernatremia. Diagnosis involves assessment of urine and plasma osmolality, serum sodium, and functional tests. Management is disorder-specific.

DETAILED QUESTIONS

Q3.1: What is the difference between Urine osmolality, Plasma osmolality, and Na+ concentration in different endocrine disorders?

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Answer:

	Diabetes insipidus (loss of water)	**SIADH** (gain of water)	**Psychogenic polydipsia**	**Adipsic hypernatremia** (due to defect in thirst center: **Organum vasculosum laminae terminalis**[OVLT])
Urine osmolality	Dilute ↓	Concentration ↑	↓	↑
Plasma osmolality	Concentration ↑	Dilute ↓	↓	↑
Na^+ concentration	↑ (Hypernatremia)	↓ (Dilutional hyponatremia/Euvolemic hyponatremia)	↓	↑

Q3.2: How is hyponatremia classified based on TBS and TBW?

Answer:

- Euvolemic hyponatremia: Normal TBS, ↑ TBW → Seen in SIADH → V2 receptor activity ↑ → Vaptans block activity.
- Hypervolemic hyponatremia: ↑ TBS, ↑ TBW → Caused by Aldosterone → Treated with Spironolactone / Eplerenone.

Q3.3: How is Nephrogenic Diabetes Insipidus treated?

Answer:

- Thiazide diuretics → Cause diuresis but suppress osmoreceptor activity at OVLT → Reduce polydipsia.
- Drug causing nephrogenic DI: Lithium

BIG QUESTION 4: What are the types of Diabetes Mellitus, their pathophysiology, methods of insulin delivery, types of insulin, and acute and long-term complications?

Broad Answer: Diabetes mellitus (DM) is a metabolic disorder characterized by hyperglycemia due to defective insulin secretion, action, or both. Types include Type 1, Type 1.5 (LADA), Type 2, MODY. Management involves insulin therapy, oral hypoglycemic agents, and lifestyle modification. Complications include acute emergencies (DKA, HHS) and long-term microvascular and macrovascular complications.

DETAILED QUESTIONS

Q4.1: What are the different types of Diabetes Mellitus?

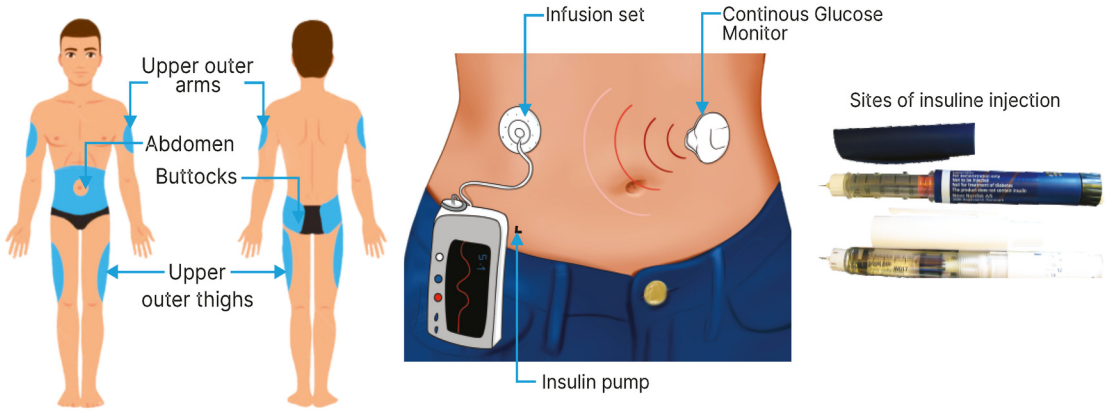
Answer: PYQ: NEET PG 2022, 2023 PYQ: FMGE 2022, 2021, 2022 PYQ: INICET 2022, 2023

	RIA Insulin (Radioimmunoassay)	Beta cell mass	Cause (Why?)	
Type 1 DM	↓	Reduced	Autoimmune, Fulminant diabetes caused by virus- Coxsackie, Rubella	Pediatric onset
Type 1.5 DM aka LADA (Latent autoimmune diabetes in adults)	↓	Reduced	Anti GAD, Anti ICA	Adult
Type 2 DM (Most common)	/ (less due to beta cell exhaustion)	Normal	**Polygenetic** • High levels of Adipokines promotes weight gain • Decreased levels of Adiponectin: Guardian angel against obesity	Adult
MODY (Maturity onset diabetes in young)**:** Rarest	↓	Normal	• Autosomal dominant (more than 2 consecutive generations getting affected) • Subtypes of MODY 1,2,3,4,5,6. • Among these, **MODY-3** is most common in India/USA • In MODY-3, chromosome involved is Ch 12, gene involved is HNF1A gene	• Adult (peak age of onset:25 years of age- Initially asymptomatic

Q4.2: What are the methods of insulin delivery, their key features, and important considerations in diabetes management?

Answer:

- **CSII (Continuous subcutaneous insulin infusion) / Insulin Pump**: Most preferred method
- Insulin pen (can adjust the dose itself)
- Multi-dose vial
- Pre-filled syringes



- Insulin pump (Best method)
  - o Mimics an artificial pancreas
  - o Continuous SC insulin infusion
  - o Continuously releases basal insulin into the body
  - o Bolus insulin delivery at mealtimes is also programmed proportionately to carbohydrate intake
- Insulin injection at the same site often has the risk of developing lipoatrophy
- The drug given in Type 1 DM is Insulin
- Initially, the drug given in Type 2 DM is OHA, but after beta cell exhaustion, Insulin is administered.
- MC side effect is Hypoglycemia
- The most preferred site is the Anterior abdominal wall (2cm away from the umbilicus)
- **Drug effective in both Type 1 and Type 2 DM is Pramlintide**
  - o Pramlintide mainly affects the stomach motility. It regulates the amount of sugar entering the small intestine.
  - o Post-prandial blood sugar is controlled

Q4.3: What are the different types of Insulin?

PYQ: INICET 2023 PYQ: FMGE 2021 ,2022

Answer:

Type of Insulin	Appearance	Action times after injection (in hours)	Comments
Rapid-acting • Lispro (Humalog) • Glulisine (Apidra) • Aspart (NovoRapid)	Clear	• Onset: 10 to 15 min • Peak: 1 to 2 hours • Duration: 3 to 5 hours	Can be injected at the start of a meal
Intermediate-acting • NPH (Humulin-N, Novolin-NPH) • Isophane or zinc insulin	Cloudy	• Onset: 1 to 3 hours • Peak: 5 to 8 hours • Duration: Upto 18 hours	Used to control glucose levels between meals. May be combined with short- acting insulin
Slow or long- acting/ Bedtime insulin- AKA Peakless insulin • Glargine (Lantus)- 24hours • Detemir (Levemir) • Long acting (42 hours)- Degludec	Clear	• Onset: 90 minutes • Peak: None • Duration: Upto 24 hours	Usually taken once daily

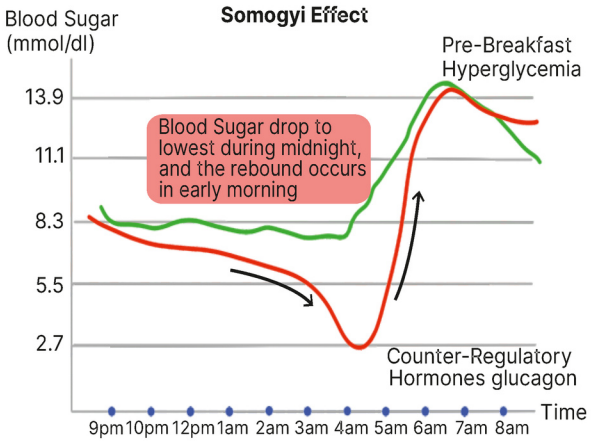
YOURWISH

Short-acting (soluble/ regular insulin)		• Onset: 30 minutes • Peak: 1-3 hours • Duration: 4-8 hours	• Usually injected 15-30 minutes before a meal. Clear solution • Advantage of regular insulin: It can also be given intravenously. • Given as IV in emergencies like Diabetic ketoacidosis, hyperosmolar coma, dangerous hyperkalemia.

Q4.4: What is the Somogyi effect?

Answer:

- Early morning hypoglycemia due to an excess dose of insulin at bedtime / less intake of required food
- At 4 A.M, blood sugar will fall.
- This will cause the release of Counter-Regulatory hormone (Glucagon) in circulation, leading to Pre-breakfast hyperglycemia
- Thus, the main manifestation of the Somogyi phenomenon is Early Morning Hypoglycemia.
- Red- Shows the insulin levels in the Somogyi effect.
- Green- Shows the insulin levels in the Dawn phenomenon.



Q4.5: What is the Dawn phenomenon?

Answer:

- In a diabetic patient, the insulin receptors show decreased sensitivity between 4 AM - 7 AM in the morning.
- 4 AM: Blood sugar begins to rise due to downregulation of GLUT-4 receptors in muscle in T2DM
- 7 AM: Pre-breakfast hyperglycemia
- Dawn phenomenon manifests with early morning hyperglycemia

Q4.6: What is the difference between Diabetic ketoacidosis and Hyperosmolar coma?

PYQ: FMGE 2019

Answer:

Diabetic ketoacidosis	Hyperosmolar coma
• This complication is more common in Type 1 DM >Type 2 DM. (due to negligence) • Insulinopenia will cause burning of fat in the body. • Burning of fat will cause generation of free fatty acid and also cause more production of ketones • 3 main ketones: o β-Hydroxybutyrate: Most acidic o Acetoacetate: Falsely normal o Acetone: Fruity odor breath	• More common in Type 2 DM • Indolent course (i.e. the person may not be diagnosed for type 2 DM until the patient landed in coma) • Blood sugar level rises >600mg% • Patient is brought in **unconscious** & **unresponsive** state o Even though the patient is not diagnosed as DM, if brought in unconscious state, capillary blood glucose must be tested

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• Investigation of choice for DKA is plasma ß-Hydroxybutyrate level • Manifestations o Pneumonia, UTI o MI, Stroke • These illnesses will cause increased energy requirement in the body	**Investigations** • Capillary blood glucose level is very high (>600mg%) • VBG and RBS has to be taken • Values: o pH: 7.40 o HCO_3: 24 meq o Urine dipstick: Ketones negative o RBS: 600mg% (causing neuronal damage)

Q4.7: The severity of Diabetic ketoacidosis is determined by?

Answer:

- **Bicarbonate levels**
- Severity of DM is determined by HbA1c levels (More elevated levels of HbA1c → More severe complications)
- Normal bicarbonate levels: 22-26 meq
- In metabolic acidosis: ABG
  - o pH ↓
  - o HCO 3 ↓
  - o pCO 2 ↓

Q4.8. A patient with Diabetic ketoacidosis is brought to casualty with dehydration. His BP was low. What is the first line intervention?

Answer:

1. Start with Normal saline followed by insulin drip
2. Before giving insulin drip, watch for potassium levels. If K is <3.5 meq/l at first potassium correction has to be done. (insulin + will send K into cells, if hypokalemia is not corrected before insulin drip, it may cause Respiratory failure, Torsades de pointes)
3. If potassium levels are normal, insulin drip can be started.
4. Insulin drip: Treatment of choice

Q4.9: What are the long-term complications of diabetes in Diabetic Retinopathy?

Answer:

- Earliest fundus finding: **Microaneurysm in the inner nuclear layer of the Retina**
- Retinopathy manifestations occur in:
  - o Type 1 DM: 5 years
  - o Type 2 DM: 15-20 years
- Gross findings
  - o Non-proliferative retinopathy: **Macular edema**
  - o Proliferative retinopathy: **Neovascularization** (may cause retinal detachment → blindness)
- To prevent blindness, PRP (Panretinal Photocoagulation) using Nd YAG Laser is done.
- Also, intravitreal injection (Bevacizumab) was given. Ideally, blood sugar control is also needed.
  - o Diabetes mellitus with HbA1c >7% (poor sugar control) progresses to Diabetic Retinopathy/ Diabetic Nephropathy.

Q4.10: What are the long-term complications of diabetes in Diabetic Nephropathy?

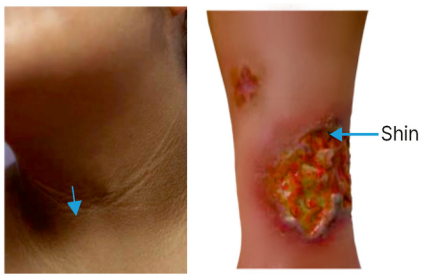
Answer:

- Diabetic Nephropathy is the leading cause of Chronic kidney disease. If not treated, it may lead to end-stage renal disease (GFR <15 ml/min)
- Progression speed is the same as Diabetic retinopathy
- To monitor the Progression of Diabetic nephropathy
  - Homocysteine levels ↑→ Homocysteine levels also contribute to Premature atherosclerosis, Stroke
  - Creatinine clearance ↓
  - Serum creatinine ↑
- Screening test: Urine albumin/Urine creatinine Ratio(UAC) or Albumin excretion rate: >300mg/g of urinary creatinine
  - Normal Albumin excretion rate: <30mg/g of urinary creatinine.
- Treatment: Reduce the progression towards ESRD by giving low-dose ACE inhibitors (also increases survival)
- **The most common HPE finding of Diabetic nephropathy is Diffuse glomerulosclerosis**

Q4.11: What are the long-term complications of diabetes in Diabetic Dermopathy?

Answer:

- **The most common manifestation of Diabetic dermopathy is delayed wound repair (Non-healing ulcer)**
- Acanthosis nigricans: Type 2 DM + hyperpigmentation (At extensor aspects like back of neck, etc.)
  - Seen in Diabetic dermopathy, PCOD, Metabolic syndrome (Impaired glucose tolerance), and Ca Pancreas/stomach
- Non-healing ulcer on the sole of the foot is due to neuropathy and Angiopathy component.
- Skin manifestation of Glucagonoma: Necrolytic migratory erythema ·
- Necrobiosis Lipoidica Diabeticorum: **Ulcer seen in the shin of a patient (Type 1 DM-Females)**



Q4.12: What is the most common long term complication of DM?

Answer: Diabetic neuropathy

Q4.13: What is the most common HPE finding of HIV associated nephropathy?

Answer: Focal segmental glomerulosclerosis

Q4.14: What are the different lesions found on shin?

Answer:

- **Lesions found on Shin (anterior aspect of leg)**
  - Necrobiosis lipoidica diabeticorum
  - Pyoderma gangrenosum: Inflammatory bowel disease/crohn's disease
  - Pretibial myxedema: Grave's disease
  - Martorell ulcer: HTN

BIG QUESTION 5: What are the causes, clinical features, investigations, and treatment strategies for adrenal disorders including Conn's Syndrome, Addison Disease, Liddle Syndrome, Cushing's Syndrome, and Pheochromocytoma?

Broad Answer: Adrenal disorders result from hormonal excess or deficiency affecting aldosterone, cortisol, catecholamines, and sex steroids. Key disorders include Conn's syndrome (hyperaldosteronism), Addison disease (adrenal insufficiency), Liddle syndrome (low-renin hypertension), Cushing's syndrome, and Pheochromocytoma. Diagnosis involves hormonal assays, imaging, and functional tests. Treatment is tailored to the specific disorder.

DETAILED QUESTIONS

Q5.1: What is the difference between Conn's Syndrome and Addison's Disease?

Answer: PYQ: FMGE 2023 PYQ: NEET PG 2022, 2023

	Conn's syndrome	Addison disease
Cause	Tumor (Adrenal adenoma) • Aldosterone ↑	Autoimmune destruction • Aldosterone ↓ • Cortisol ↓ • DHEAS ↓
Clinical features	• HTN • Hypokalemic alkalosis (urinary loss of potassium and hydrogen)	• Salt wasting • Postural hypotension: o Fall in Systolic BP & Diastolic BP: Test to be done is Tilt table test or Sudden standing from sitting or supine position (wait 3 minutes for compensatory mechanism to act) o Even after 3 minutes, if it falls in SBP >20mmHg and DBP >10mmHg, then it is known as Postural hypotension. • Hypoglycemia- due to cortisol deficiency • Hyperpigmentation- due to cortisol deficiency → Elevated ACTH levels • Loss of pubic hair (due to deficiency of sex steroid)
Screening	• ARR(Aldosterone renin ratio)- Low renin hypertension • Aldosterone ↑ /Renin ↓	• Basal plasma ACTH and basal serum cortisol, glucose, urea and electrolytes • High ACTH and Low cortisol
IOC	Saline infusion test	ACTH stimulation test/Cosyntropin test: No response
Treatment	Spironolactone	• Hydrocortisone • Management postural hypotension (Mineralocorticoid deficiency) o Fludrocortisone • Management of Hypoglycemia (Glucocorticoid deficiency) o Dexamethasone

Q5.2: What are the key points differentiating Primary and Secondary Addison Disease?

Answer:

- Primary: Adrenal defect → ACTH ↑ → Hyperpigmentation
- Secondary: Pituitary defect (Sheehan syndrome) → ACTH ↓ → Hypopigmentation

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Q5.3: What are the key features of Liddle Syndrome?

Answer:

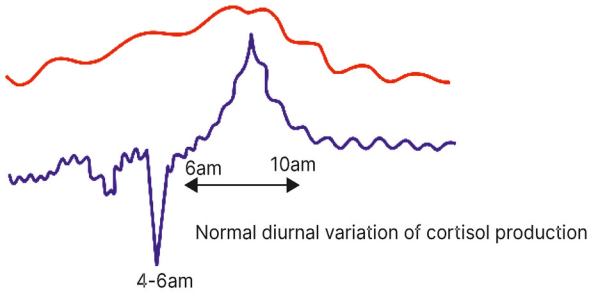
- **Cause of Low-renin hypertension**
- Autosomal dominant
- Exhibits gain-of-function of ENaC
- **Hypertension + Hypokalemic alkalosis**
- In **Liddle syndrome,** aldosterone levels are decreased due toa negative feedback mechanism, but hypertension occurs due to + excess salt & water in the body secondary to excessive activity ofthe epithelial sodium channel.
  → exaggerated urinary loss of H + and K+
  ⊖ RAAS: ↓ and Renin ↓ Aldosterone
- Imaging shows Normal Adrenals
- Treatment: Amiloride (DOC)

Q5.4: What are the key features of Cushing's Syndrome?

PYQ: INICET 2023

Answer:

- In a normal individual, cortisol values keep fluctuating (nadir or high) - Less between 4-6 am, and in the mornin,g cortisol levels begin to rise (cortisol-stress hormone).
  - o But in Cushing's syndrome patients, diurnal variation is lost & overall cortisol level is higher.
  - o **Earliest manifestation: Loss of diurnal variation of cortisol production**



Normal diurnal variation of cortisol production

- Clinical features
  - o Centripetal obesity, a lemon on stick appearance
  - o Moon facies (big puffy face)
  - o Hirsutism
- Increased cortisol, blood sugar levels lead to impaired glucose tolerance
  - o FBS & 2 hr value elevated ,leading to Secondary diabetes
- Most common cause: Iatrogenic steroids (Exogenous): Excess cortisol
  - o Cortisol↑ and ACTH ↓
- **Endogenous cause: Pituitary adenoma, aka Cushing's disease**
  - o Cortisol ACTH ↑ and ↑
- Adrenal adenoma (cortisol increased)
  - o Cortisol ACTH ↑ and ↓
- Oat cell cancer lung
  - o Cortisol ACTH ↑ and ↑

Q5.5: What are the screening tests for Cushing's Syndrome?

Answer:

- 24-hour urinary cortisol: ↑
- Followed by an overnight dexamethasone suppression test
  - o 3 type dexamethasone suppression test
    - → Overnight dexamethasone suppression test
    - → Low-dose dexamethasone suppression test

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→ High-dose dexamethasone suppression test

- IOC: Low-dose dexamethasone suppression test

Q5.6: What is the high-dose dexamethasone suppression test?

Answer:

- Used in etiological identification
- ACTH-dependent Cushing's (ACTH normal or high)
  - o Pituitary adenoma
  - o Oat cell cancer lung
- ACTH-independent Cushing's (ACTH value low)
  - o Adrenal adenoma
- MRI pituitary is done to differentiate Cushing's disease from Cushing syndrome caused by an adrenal adenoma
- The high-dose dexamethasone test is used to differentiate between pituitary adenoma and oat cell cancer lung

Q5.7: What are the key features of Pheochromocytoma?

PYQ: NEET PG 2021 PYQ: INICET 2021

Answer:

- According to the Harrison guidelines
  - o Screening test: 24-hour urinary fractionated metanephrine levels
  - o IOC: Plasma free metanephrine levels
  - o 24-hour urinary catecholamines
- Test done is 24-hour urinary VMA (Vanillylmandelic acid) level: Increased (According to Robbins pathology)
- Mostly benign (90%)
- 10% pheochromocytoma can be malignant
- 10% can be extra-adrenal.
  - o Both Extra-adrenal and Malignant can be identified by MRI
- 10% of pheochromocytoma can have a Familial basis (Ret proto-oncogene involvement)
- **Pheochromocytoma is associated with MEN-2A (Multiple Endocrine Neoplasia), aka Sipple Syndrome**
- Paraganglioma: Extra-adrenal pheochromocytoma with sympathetic chain

Q5.8: What are the Clinical features of Pheochromocytoma?

Answer:

Clinical features

1. Palpitation: HR ↑
2. Headache (due to hypertension-Paroxysmal/episodic): Vasoconstriction
3. Diaphoresis: ++ sweating
   - Mnemonic: **PHD**
   - Misdiagnosis: Anxiety neurosis
4. Weight loss
5. Nausea, Vomiting, Abdominal pain
6. Tremors
7. Impaired glucose tolerance (FBS and 2 hr value elevated)
8. In later stages, due to constricted intravascular volume, results in Orthostatic hypotension (postural BP low)

BIG QUESTION 6: How are thyroid disorders and Multiple Endocrine Neoplasia (MEN) syndromes diagnosed, interpreted, and managed, including their clinical features, scans, antibodies, genetic mutations, and associated tumors?

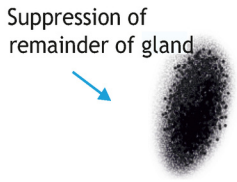
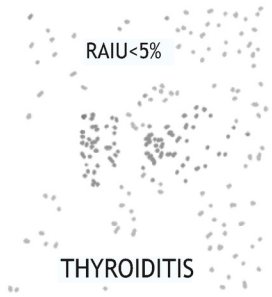
Broad Answer:

- Thyroid disorders involve altered hormone production (T3, T4) and can be autoimmune, neoplastic, or functional. Diagnosis uses thyroid function tests, antibody assays, and thyroid scans to classify nodules as hot, cold, or warm, or diffuse uptake patterns. Treatment depends on the underlying pathology.
- Multiple Endocrine Neoplasia (MEN) syndromes are autosomal dominant disorders with tumors in multiple endocrine glands. MEN 1, MEN 2A (Sipple Syndrome), and MEN 2B (MEN 3) have specific tumor associations, clinical features, and genetic mutations. Early diagnosis via screening, scans, and genetic testing is essential for timely intervention and preventing complications.

Q6.1: What are the thyroid scan findings in different thyroid disorders?

Answer:

 COLD NODULE	• **Cold nodule:** Suspect malignancy o MC: Papillary carcinoma thyroid associated with radiation exposure
Pyramidal lode  GRAVE DISEASE	• **Diffuse increase uptake:** Grave's disease PYQ: FMGE 2020, 2021 • In Grave's disease : Thyroid stimulating antibody/ LATS (long acting thyroid stimulating antibody) • T3,T4 are high and TSH can't be detectable
Hot and Cold nodules  TOXIC MULTINODULAR	• **Asymmetrical uptake/Hot & Cold nodule present:** Toxic nodular goiter
 HOT NODULE	• Bit of black are with remaining gland visible (grayish) Hot nodule

 AUTONOMOUS NODULE	• Complete suppression of gland with few part left Single black area : **Autonomous nodule**
 THYROIDITIS	• **Diffuse decrease uptake: Hashimoto's thyroiditis** o Antibody responsible for Hashimoto thyroiditis is anti TPO antibody (thyroid peroxidase)/anti microsomal antibody

Q6.2: What are the biochemical features of hyperthyroidism and hypothyroidism?

Answer:

- Hyperthyroidism: ↑T3, ↑T4, resting tachycardia, increased sleeping pulse rate
- Hypothyroidism: ↓T3, ↓T4; reliable sign is *hung-up ankle jerk* (also seen in Huntington's chorea)

Q6.3: What is the life-threatening complication of hyperthyroidism and its treatment?

Answer:

- Condition: Myxedema coma (core temperature falls, triggered by intercurrent illness)
- Treatment:
  - o IV Liothyronine
  - o IV Hydrocortisone (given in all endocrine emergencies)

Q6.4: What are the different types of Multiple Endocrine Neoplasia (MEN) and their key features?

Answer: PYQ: FMGE 2021, 2022 PYQ: INICET 2021, 2022

	Clinical manifestations
MEN 1	• Pituitary adenoma (Prolactinoma) • Parathyroid adenoma (MC) • Pancreatic adenoma (Zollinger ellison syndrome)
MEN 2/ Sipple Syndrome/MEN 2A · Mutation in RET gene	• Parathyroid Hyperplasia/Adenoma • Pheochromocytoma • Medullary carcinoma thyroid
MEN 2B/MEN 3	• Marfanoid habitus (arms are longer than height of person) • Mucosal neuroma (bumpy lips & tongue) • Medullary carcinoma thyroid • Pheochromocytoma

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9. RESPIRATORY SYSTEM

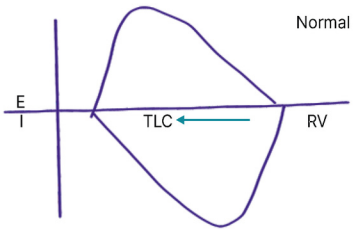
BIG QUESTION 1: What are the different types of flow-volume curve patterns and their clinical significance?

Broad Answer: Flow-volume curves are essential tools in pulmonary function testing to differentiate between obstructive, restrictive, fixed, and variable airway diseases, as well as neuromuscular disorders. Each pattern has characteristic features in the inspiratory and expiratory limbs, total lung capacity (TLC), residual volume (RV), and reversibility after bronchodilator use, which help guide diagnosis and management.

DETAILED QUESTIONS

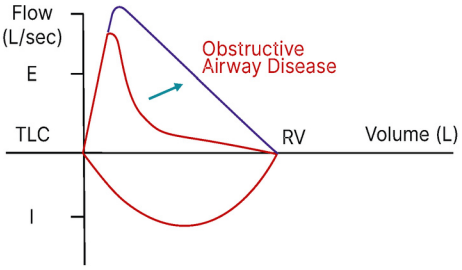
Q1.1: What is the normal Flow Volume Curve?

Answer:



Q1.2: What are the changes in the flow volume curve in asthma?

Answer:

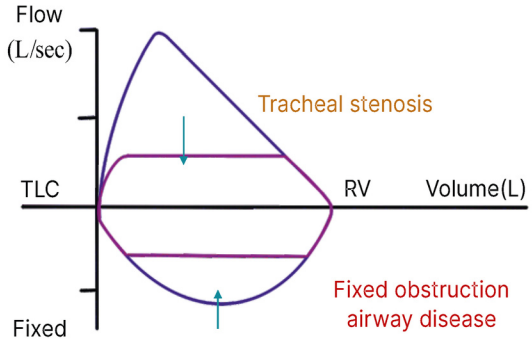


- It is a disorder of expiration
- In the flow volume curve: The inspiration part is the same or superimposed with the original curve, and there will be resistance to the expiratory component leading to concavity in the expiration part of the curve, suggesting of **obstructive airway disease.**
- To differentiate between Asthma and COPD, **bronchodilators** are given
  - The test is repeated after 15 minutes of bronchodilator administration. **Concavity will reduce** significantly and go towards normal, indicating Bronchial Asthma (reversible airflow obstruction)
  - Curve remains the same, and **no change in curve indicates COPD** (because it is a non-reversible airflow obstruction)

Q1.3: What are the key features of Fixed Obstructive Airway Disease?

PYQ: FMGE 2023

Answer:



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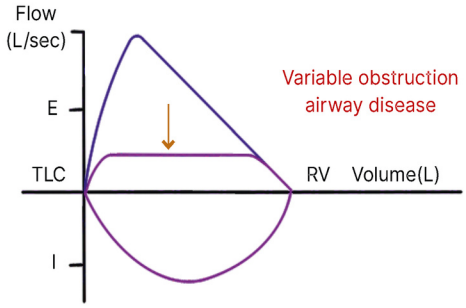
- In this image, there is universal flattening of the entire curve. This is due to **tracheal stenosis.**
- E.g., the patient developed a delta variant of COVID-19, and he has been intubated for 2 weeks. Because of prolonged intubation patient developed tracheal stenosis
- In tracheal stenosis, there is obstruction in both the expiration and inspiration phases.
- This curve shows fixed obstructive airway disease. Here **problem lies with the trachea,** not in the parenchyma of the lung, and there is no role for a bronchodilator.

Q1.4: What are the key features of Variable Obstructive Airway Disease?

Answer:

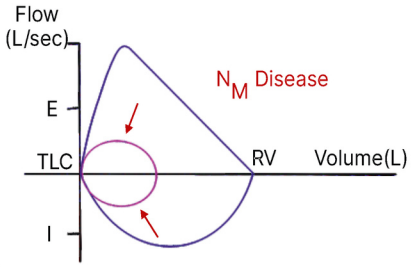
- Flattening is present only in the expiratory part
- It is called a variable because the problem is present in the expiratory phase of respiration
- In this image, the flattening is because there is an airway mass that is hampering the expiratory phase of respiration



Q1.5: What are the changes in the flow volume curve in Neuromuscular Disease?

Answer:

- In this image, the curve becomes a small circle
- In neuromuscular disease like generalised myasthenia gravis, there is diaphragmatic/ respiratory muscle weakness, so the entire curve becomes relatively small.

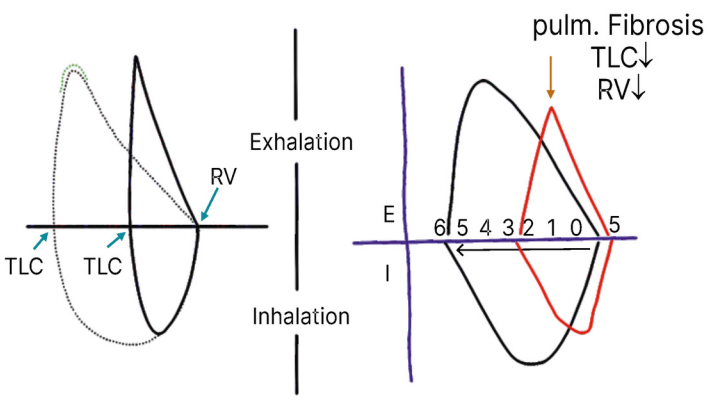


Q1.6: What are the changes in the flow volume curve in Restrictive Lung Disease?

Answer:

- Restrictive airway disorder presents with pulmonary fibrosis
- In Pulmonary fibrosis, the TLC is decreased, i.e., less than 6L, because of a shrunken lung
- In this condition, the curve won't end at 6L; instead, it ends earlier because of the shrunken lung
- Shrunken lung results in decreased TLC and decreased RV
- Therefore, RV starts a bit earlier (in 400ml) and ends earlier (in 3L)
- In this curve, black represents normal and red represents Restrictive Lung Disease
- In the expiratory part, the tip of the curve looks pointed, known as the **Beanie cap** appearance
- Examples of Restrictive Lung Disease: Asbestosis, idiopathic pulmonary fibrosis (IPF is a histopathological diagnosis)
- The curve appears shifted, and the Beanie cap appearance S/O Restrictive Lung Disease

PYQ: FMGE 2022



Restriction lung disease

Q1.7. A patient presents with cough and SOB episodes for the last 2 years, for which he has taken treatment, but has no records. He has no occupational risk factors, but reports growth of molds in his house due to leakage. CT chest reveals basilar emphysematous changes. Which test would help in diagnosis?

A. Serum precipitins against Aspergillus species

B. Flow volume curve with non-reversible airflow obstruction

C. Skin prick test for Aspergillus

D. Serum A1 antitrypsin levels

Answer. **D - Serum al antitrypsin levels**

Explanation:

The patient has a history of episodic shortness of breath (SOB) and cough, features consistent with asthma or COPD exacerbations.

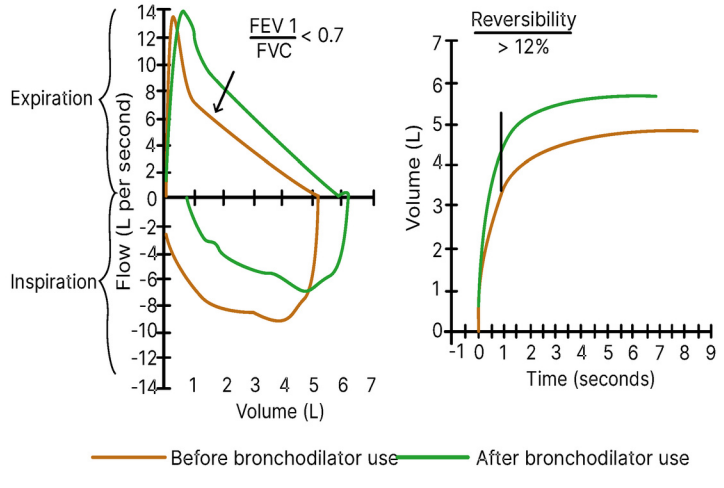
There are no occupational risk factors, but there is reported mold growth in the house, which could suggest an allergic component.

The key finding is **basilar emphysematous changes** on CT chest.

- **Option A (Serum precipitins against Aspergillus species)** and **Option C (Skin prick test for Aspergillus)** are tests for Allergic Bronchopulmonary Aspergillosis (ABPA). However, ABPA typically presents with **central bronchiectasis** on HRCT, not basilar emphysema. Therefore, these options are incorrect.
- **Option B (Flow volume curve with non-reversible airflow obstruction)** would confirm obstructive lung disease (e.g., emphysema) but does not help identify the underlying aetiology. Since emphysema is already suggested by CT, this test does not provide additional diagnostic value for the cause.
- **Option D (Serum al antitrypsin levels)** is the correct choice. **Basilar emphysema** is a classic feature of **alpha-1 antitrypsin deficiency**. This test would help diagnose this genetic condition as the underlying cause of the emphysematous changes. Confirmation may involve a liver biopsy showing PAS-positive, diastase-resistant inclusions in hepatocytes, especially if there is concomitant liver disease.

Q1.8. A 40-year-old smoker has recently joined a construction company and presents to you with worsening dyspnea. His flow volume curve is shown with the following results. What is the correct interpretation?

	Prebronchodilators				Postbronchodilators		
Spirometry	Predicted	LLN	Actual	% of Predicted	Actual	% of predicted	% change
FVC(L)	5.20	4.34	5.18	99	6.06	116	+16
FEV1(L)	4.37	3.64	3.55	81	4.64	106	+30
FEV,FVC(%)	84	75	68	81	77	91	+11
FEF (L per second)	4.74	3.11	2.41	50	3.84	80	+59



A. Restrictive aetiology with response to bronchodilator
B. Restrictive aetiology with no response to bronchodilator
C. Obstructive aetiology with response to bronchodilator
D. Obstructive aetiology with no response to bronchodilator

Answer: **C - Obstructive aetiology with response to bronchodilator**
Explanation:

- The key spirometry parameters are:
  - **Pre-bronchodilator FEV1/FVC ratio**: The actual value is 68% (which is below the Lower Limit of Normal, LLN, of 75%), indicating an **obstructive defect**.
  - **Post-bronchodilator FEV1/FVC ratio**: Improves to 77% but remains below the predicted norm, confirming persistent obstruction.
  - **Reversibility**: There is a significant response to bronchodilators:
    - → FEV1 increases by **30%** (well above the 12% threshold for significant reversibility).
    - → FVC increases by 16%.
    - → FEF (forced expiratory flow) increases by 59%.
- The flow-volume curve (not fully depicted but implied) typically shows a scooped expiratory limb in obstruction, which improves post-bronchodilator.
- There is **no evidence of restriction**:
  - Pre-bronchodilator FVC is 99% of predicted, which is normal.
  - Post-bronchodilator FVC is 116% of predicted, arguing against a restrictive defect.
- Therefore, the findings are consistent with an **obstructive aetiology (e.g., asthma or COPD) with a significant response to bronchodilators**.

BIG QUESTION 2: What are the common causes, diagnostic clues, and treatment strategies for pneumonia in different clinical contexts?

Broad Answer: Pneumonia is most often caused by **Streptococcus pneumoniae** in both community-acquired and AIDS patients. The most common organism for acute bacterial Pneumonia in the adult and pediatric population is Pneumococcus. Special organisms are associated with particular risk factors, such as Pneumocystis jiroveci in AIDS with CD4 <200, Klebsiella pneumoniae in alcoholics, and Legionella pneumophila in hotel or cruise exposures. Diagnosis relies on clinical features, imaging, and in some cases, bronchoalveolar lavage (BAL) with special stains. Severity can be assessed with the **CURB-65** score to guide outpatient, inpatient, or ICU management.

DETAILED QUESTIONS

PYQ: NEET PG 2020, 21, 22 PYQ: FMGE 2020, 21, 22
PYQ: AIIMS 2018 PYQ: INICET 2022, 2023

Q2.1: What are the key features of Pneumonia with AIDS?

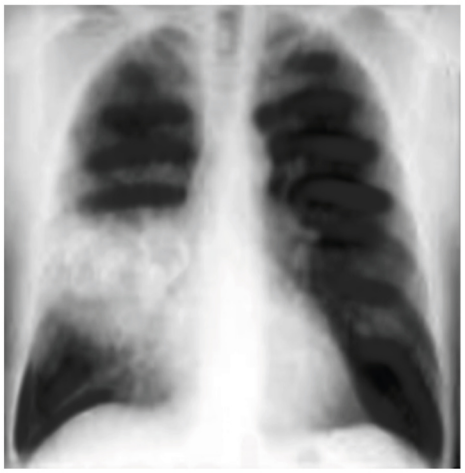
Answer:

- The commonest organism for acute bacterial Pneumonia in the adult and pediatric population is Pneumococcus.
- The commonest cause of pneumonia in AIDS AIDS-positive patient is Pneumococcus, not Pneumocystis jiroveci.
- Pneumonia in an AIDS +ve patient, low CD4 count of <200 cells / cu.mm & paucity of examination findings is an Opportunistic infection in an AIDS +ve patient is Pneumocystis jiroveci
- Lobar consolidation and bronchial breathing are absent in Pneumocystis jiroveci.
- If bronchial breathing and Lobar consolidation are given in question, then the answer is Pneumococcus. Because the leading cause of Pneumonia in AIDS positive patients is Pneumococcus.
- Low CD4 count is s/o Pneumocystis jiroveci, but not confirmatory.

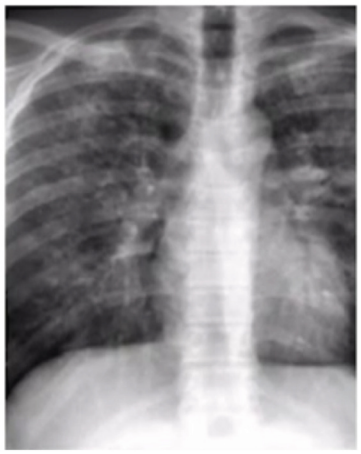
- Below X-ray shows Lobar consolidation in the Left upper lobe.



- Below X-ray shows Lobar consolidation in the Right middle zone.



Q2.2: What are the key features of Pneumocystis jiroveci



Answer:

- Pneumocystis jiroveci causes interstitial pneumonia.
- Chest is clear initially, but later develops diffuse rales
  - Chest X-ray: Bilaterally symmetrical perihilar reticular interstitial changes are seen
- HRCT chest: **Diffuse ground glass opacities**
- LDH increases.
- Increase A - a gradient.
- Confirmatory test: **Bronchoalveolar Lavage** stained with Gomori methenamine silver stain, which demonstrates the cysts.
- Rx: Trimethoprim - **Sulfamethoxazole** (DOC for treatment and prevention)

Q2.3: Which organisms are associated with specific patient groups or exposures in pneumonia

Answer:

- Alcoholics: S. pneumoniae, Klebsiella pneumoniae (necrotising, "red currant jelly" sputum), oral anaerobes (lung abscess, air-fluid level).
- Impaired airway defence is seen in alcohol/drug intoxication, dementia, stroke, etc. The cause of pneumonia is **Oral anaerobes** - treatment is **clindamycin.**
- COPD: Haemophilus influenzae
- Hotel/cruise: Legionella pneumophila
- Birds: Histoplasma capsulatum, Chlamydia psittaci
- Rabbits: Francisella tularensis (ulceroglandular fever with eschar & lymphadenopathy)
  - Tularemia is caused by the bacterium Francisella tularensis.
  - Hunter, after killing a rabbit, develops an Eschar/ ulcer and regional lymph node swelling.
  - Ulceroglandular fever: The most common form of tularemia, which usually occurs following a tick bite or after handling an infected animal. The ulcer is accompanied by swelling of regional lymph glands, usually in the armpit or groin.
- Sheep: **Coxiella burnetii** (Q fever → atypical pneumonia, endocarditis; Treatment - **macrolides**)
- CA-MRSA: Post-viral or secondary infections.

YOURWISH

YOURWISH

Q2.4: What is the CURB-65 score

Answer:

CURB-65 score PYQ: NEET PG 2021, 22	Points
Confusion (defined as a Mental Test Score of ≤8, or disorientation in person, place, or time)	1
Uremia: BUN >7 mmol/L (~19 mg / dL)	1
Respiratory rate: ≥30 breaths / minute	1
Blood pressure: systolic <90 mm Hg or diastolic ≤60 mm Hg	1
Age ≥65 years	1
Total points	

Q2.5: What are the Treatment options based on the CURB-65 Score

Answer:

Treatment options based on CURB-65 Score		
Score	**Group**	**Treatment Options**
0	Group 1; mortality low (1.5%)	Low risk; consider home treatment
1-2	Group 2; mortality intermediate (9.2%)	Consider hospital-supervised treatment (either short - stay inpatient or hospital - supervised outpatient)
3	Group 3; mortality high (22%)	Manage in hospital as severe pneumonia; consider admission to

- For group 1: OPD management is done.
- For group 2: IPD management is done. E.g., 70-year-old patient with elevated BUN RX: IV Augmentin with macrolides
- For group 3: ICU management is done.
  - o If only age is >/= 65 and the rest are normal, treatment is based on the health condition of the patient, and the treating doctor can decide the type of management.
  - o If age, along with another deranged parameter, is given. Then admit the patient and give parenteral antibiotics.

Q2.6: What is The Treatment Strategy in C.A.P. Outpatient?

PYQ: INICET 2021, 23

Answer:

Treatment Strategy: C.A.P: Outpatient	
No comorbidity	Comorbidity or Received antibiotics in the last 3 months
1. Amoxicillin + Macrolide/Doxycycline OR 2. Doxycycline OR 3. Macrolide (if local prevalence of pneumococcal Resistance <25%)	1. Amoxicillin Clavulanate +Macrolide +Doxycycline 2. Levofloxacin, Moxifloxacin, Gemifloxacin (Respiratory fluoroquinolones)

Q2.7: What is the treatment Strategy in the C.A.P. inpatient?

Answer:

- For Inpatient Management · β lactam + macrolide or respiratory fluroquinolones (Levofloxacin, Moxifloxacin, Gemifloxacin)
- If recent hospitalisation/ respiratory isolation
  - o Add coverage for MRSA: Vancomycin/ linezolid & for Pseudomonas: Piperacillin-Tazobactam.

Q2.8: What are the Commonest opportunistic infections based on CD4 count in AIDS patients?

Answer:

- Normal CD4 COUNT is 1000 on average
- <500: TB
- <200: *P. jiroveci* → cotrimoxazole
- <100: Toxoplasmosis (seizures)
- <50: CMV retinitis ("pizza pie" retinopathy), Mycobacterium avium complex (MAC)
- Note: Cryptococcus prophylaxis is NOT started at CD4 <150; it's treated only after infection, and chemoprophylaxis may be considered recurrent until CD4 >100.
- A patient with AIDS develops cryptococcal meningitis in 2021 & develops the same infection in 2022. Treat the current infection with Liposomal amphotericin B + Fluconazole & start the patient on chemoprophylaxis. Stop chemoprophylaxis if CD4 count is > 100
- Stop prophylaxis if the count is >350 for two consecutive times at an interval of 6 months.
- Once a day pill in AIDS management consists of Dolutegravir, emtricitabine and tenofovir.

Q2.9: How do you diagnose metabolic acidosis with respiratory acidosis using Winter's formula

Answer: In metabolic acidosis, the expected compensation is a decrease in pCO_2, calculated using Winter's formula:

- Standard: Expected $pCO_2 = (1.5 \times HCO_3^-) + 8 \pm 2$
- Modified: Expected $pCO_2 = HCO_3^- + 15$

If the measured pCO_2 is higher than expected, an additional respiratory acidosis is present, indicating a mixed disorder.

Example: pH = 7.0, HCO_3^- = 10 mEq/L, pCO_2 = 30 mmHg.

- Expected pCO_2 (modified) = 10 + 15 = 25 mmHg.
- Measured pCO_2 is 30 mmHg (↑) → inadequate compensation → metabolic acidosis with respiratory acidosis. If the values matched, it would be a simple metabolic acidosis; if pCO_2 was lower, there would be a coexisting respiratory alkalosis.

Q2.10. Which of the following statements is incorrect with regard to prophylaxis in AIDS?

A. Start PCP prophylaxis when CD4 count is <200

B. Stop if the count is >350 for two consecutive times at an interval of 6 months

C. MAC prophylaxis started when the CD4 count is less than 50

D. Cryptococcus prophylaxis started when the CD4 count is less than 150

Answer:

PYQ: NEET PG 2021
PYQ: FMGE 2020, 21

D - Cryptococcus prophylaxis started when the CD4 count is less than 150

Explanation:

- Normal CD4 count is around 1000 on average.
- Common opportunistic infections based on CD4 count:
  - <500: Tuberculosis (TB)
  - <200: *Pneumocystis jirovecii* pneumonia (PCP). Drug of choice (DOC) for both treatment and prophylaxis is Cotrimoxazole.
  - <100: Toxoplasmosis (presents with seizures)
  - <50: CMV retinitis (presents with "pizza pie" retinopathy) or *Mycobacterium avium-intracellulare* (MAC)
- **Option A is correct:** PCP prophylaxis is started when the CD4 count is <200.
- **Option B is correct:** Prophylaxis (e.g., for PCP) is stopped if CD4 count is >350 for two consecutive measurements at an interval of 6 months.

- **Option C is correct:** MAC prophylaxis is started when the CD4 count is <50.
- **Option D is incorrect: Cryptococcus prophylaxis is not routinely recommended for all patients with a CD4 count <150.** Instead, it is considered only for certain high-risk patients (e.g., those with a prior history of cryptococcal meningitis). In such cases, prophylaxis is stopped if the CD4 count rises to >100 for at least 3 months. Therefore, the statement that Cryptococcus prophylaxis is started universally when the CD4 count is <150 is inaccurate.

Additional note: Once-daily pill for AIDS management often consists of Dolutegravir, Emtricitabine, and Tenofovir.

Q2.11. pH = 7, HCO_3 = 10 meq, pCO_2 = 30 mmHg. Which is correct?

A. Metabolic acidosis with respiratory alkalosis
B. Metabolic acidosis with respiratory acidosis
C. Respiratory acidosis with metabolic alkalosis
D. Metabolic acidosis with metabolic alkalosis

Answer.

B - Metabolic acidosis with respiratory acidosis

Explanation:

- The given values are: pH = 7 (acidemia), HCO_3 = 10 meq (low), pCO_2 = 30 mmHg (low). All values are low, indicating **metabolic acidosis**.
- For **respiratory acidosis**, pCO_2 should be high (not low), so option C is incorrect.
- To assess compensation, we use **Winter's formula** (or modified Winter's formula) for metabolic acidosis:
  - Expected $pCO_2 = (1.5 \times HCO_3) + 8 \pm 2$

    OR
  - Modified Winter's formula: Expected $pCO_2 = HCO_3 + 15$
- Applying the modified formula: Expected pCO_2 = 10 + 15 = **25 mmHg**.
- The actual pCO_2 is **30 mmHg**, which is higher than expected. This indicates that the respiratory compensation is inadequate (the pCO_2 should be lower to compensate for the metabolic acidosis). Instead, the higher pCO_2 suggests a concomitant **respiratory acidosis**.
- Therefore, this is a **mixed disorder: metabolic acidosis with respiratory acidosis**.
- Option A (metabolic acidosis with respiratory alkalosis) is incorrect because respiratory alkalosis would involve even lower pCO_2 (e.g., <25 mmHg).
- Option D (metabolic acidosis with metabolic alkalosis) requires calculation of the anion gap and delta gap, which is not provided here. However, the respiratory component (pCO_2) is abnormal, so this is not purely metabolic.
- Thus, the correct interpretation is **metabolic acidosis with respiratory acidosis** (Option B).

Q2.12: What are the common HRCT findings and lab markers in COVID-19 pneumonia, the confirmatory diagnostic test, and a shared treatment approach with Pneumocystis jiroveci pneumonia

Answer: In COVID-19 pneumonia, HRCT typically shows ground-glass opacities, and D-dimer levels are increased. The confirmatory test for COVID-19 is RT-PCR. Steroids are used in the treatment of both COVID-19 pneumonia and Pneumocystis jiroveci pneumonia, which also causes interstitial pneumonia.

BIG QUESTION 3: What are the key diagnostic features, classifications, and management strategies for major respiratory disorders, including COPD, pleural effusion, asthma, pulmonary embolism, pulmonary hypertension, and hemoptysis?

Broad Answer: Respiratory disorders like COPD, pleural effusion, asthma, pulmonary embolism (PE), pulmonary arterial hypertension (PAH), and hemoptysis have distinct diagnostic criteria, grading systems, and management plans. COPD is diagnosed via spirometry with reduced FEV_1/FVC and classified into GOLD stages; pleural effusion is differentiated as transudate or exudate by Light's criteria; asthma is reversible airway obstruction often guided by stepwise ICS/formoterol therapy; PE requires imaging confirmation and urgent anticoagulation; PAH is diagnosed via hemodynamic monitoring and treated with vasodilators; hemoptysis management depends on severity, etiology, and includes bronchial artery embolization for massive bleeding.

DETAILED QUESTIONS

Q3.1: What is the classification of airflow limitation severity in COPD (Based on Post-Bronchodilator FEV1)

Answer:

- FEV/FVC ratio is a timed vital capacity, this is reduced in obstructive airway disorder and normal to elevated in restrictive 1 airway disorder
- In mild, moderate, severe & very severe, no improvement is seen in timed vital capacity even after giving bronchodilators

Classification of Airflow limitation severity in COPD (Based on Post-Bronchodilator FEV_1)		
In patients with FEV1/FVC < 0.70: Timed Vital Capacity		
GOLD 1	Mild	$FEV_1 \geq 80\%$ predicted
GOLD 2	Moderate	$50\% \leq FEV_1 < 80\%$ predicted
GOLD 3	Severe	$30\% \leq FEV_1 < 50\%$ predicted
GOLD 4	Very Severe	$FEV_1 < 30\%$ predicted

Q3.2: What is the grading of dyspnea

Answer: Modified Medical Research Council (mMRC) Dyspnea Scale

- mMRC Grade 1: Hurrying on the level or walking up a slight hill.
- mMRC Grade 2: Walk slower than people of the same age on the level because of breathlessness or must stop for breath while walking
- mMRC Grade 3: Stop for breath after walking about 100 meters or after a few minutes on the level
- mMRC Grade 4: Too breathless to leave the house or breathless while dressing or undressing.

Q3.3: What is COPD Management

PYQ: NEET PG 2023

Answer: 2021 UPDATE: Initial Pharmacological Treatment

>/=2 moderate exacerbations or >/= 1 leading to hospitalization	**Group C:** LAMA	**Group D:** LAMA or LAMA + LABA* or ICS + LABA** *Consider if highly symptomatic (e.g.CAT > 20) **Consider if eos >/= 300
0 or 1 moderate exacerbations (not leading to hospital admission)	**Group A:** A Bronchodilator on need basis	**Group B:** A Long-Acting Bronchodilator (LABA or LAMA) - on continuous basis
	mMRC 0-1 CAT <10	**mMRC 2 CAT >10**

- CAT: COPD Assessment Test
- Mortality in COPD patient is reduced by Low flow O and drugs like LAMA

YOURWISH

Q3.4: What are the key features of Pleural Effusion

PYQ: NEET PG 2019
PYQ: FMGE 2019, 20

Answer:



- Minimum fluid for clinical detection of pleural effusion: **300 ml**
- Obliteration of the hemidiaphragm on chest X-ray: **500 ml**
- The above chest X-ray shows blunting of the costophrenic angle and Ellis curve
- Best test to diagnose Minimal pleural effusion: **CT chest** > USG chest
- Minimum fluid for clinical detection of ascites: 1.5 to 2 L
- Puddle sign in ascites can detect 100 ml
- In CSF normal cells: 0-4 lymphocytes/ cu.mm
- CSF sugar is 2/3 of plasma blood sugar

Q3.5: What are the key features of Light's Criteria

Answer: Light's Criteria

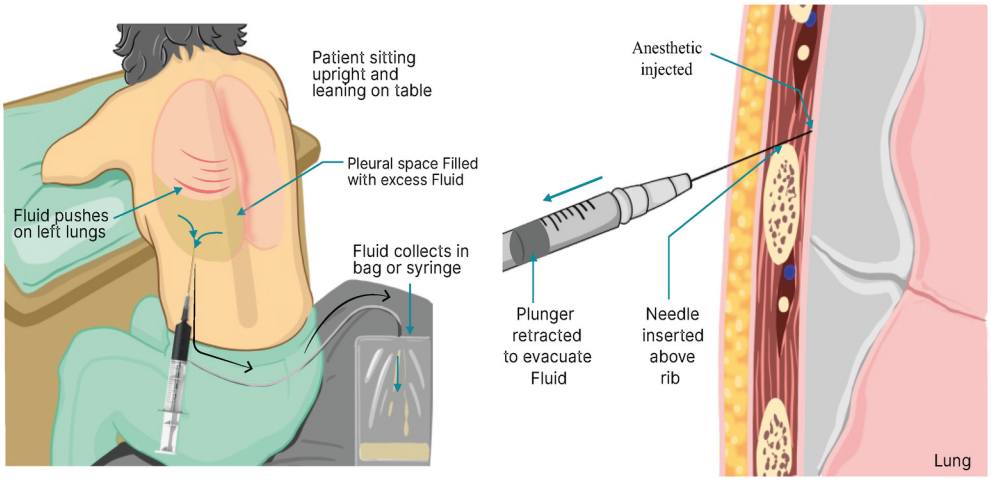
- It helps to D/B Exudate and Transudate
  - o Pleural fluid protein/serum protein > 0.5
  - o Pleural fluid LDH/serum LDH > 0.6 or Pleural fluid LDH more than two-thirds the normal upper limit for Serum
- If the values are more than this, it is exudate, and if less than this, it is transudate.
- Light's criteria are not accurate in (25% cases, there can be misdiagnosis)

Q3.6: What are the key features of Thoracocentesis

Answer:

- The preferred site is decided by the USG
- On percussion, stony dullness is the characteristic feature noted on the side of the pleural effusion.
- The ideal site is decided based on USG.
- If USG is not mentioned in the option. Then the site to insert the needle is medial to the scapular line and lateral to the posterior axillary line at the 8th ICS.
- The needle is inserted at the upper border of the lower rib or the lower part of the ICS to prevent damage to the neurovascular bundle.

PYQ: FMGE 2021



Q3.7: Indications for ICD tube

Answer:

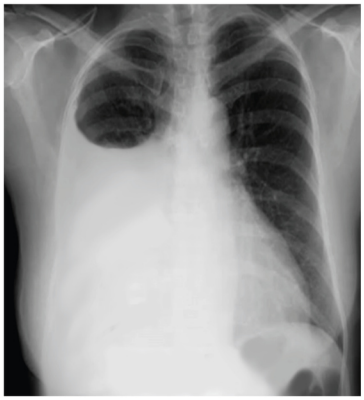
Indications for the ICD tube

- Loculated pleural fluid/ empyema (i.e., pus in the pleural cavity)
  - o Empyema can be without loculations or with loculations

- Pleural fluid pH <7.20
- Pleural fluid glucose <3.3 mmol/L (<60 mg/dL)
- Positive Gram stain or culture of the pleural fluid
- Presence of gross pus in the pleural space

Q3.8: What are the radiological findings in Pleural effusion/ hydrothorax? PYQ: INICET 2020

Answer: In the chest X-ray below, blunting of the CP angle and Ellis curve is present S/O Pleural effusion/ hydrothorax.



Q3.9: What are the radiological findings in Hydro-pneumo thorax?

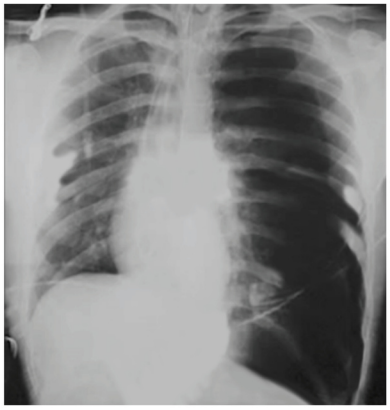
Answer: In the chest X-ray below, the blunting of the CP angle and straight line is present S/O Hydro-pneumo thorax (both water and air present simultaneously).



Q3.10: What are the radiological findings and treatment of Pneumothorax?

Answer:

- In the chest X-ray below deep sulcus sign and absence of vascular markings in present S/O Pneumothorax
- Treatment - Tension pneumothorax: Lung decompression by **wide bore needle at 5th ICS in mid axillary line (according to ATLS guidelines) & subsequent ICD tube insertion with underwater seal is done.**



Q3.11: What are the key features of Asthma?

Answer:

- Reversible airway disorder (R.A.D), FEV1 increased ≥ 12% or 200ml after giving Bronchodilator
- SABA, repeated use indicates poor control and↑ mortality
- Formoterol is used as a reliever
- ICS (inhaled corticosteroids) ↓ Mortality

Q3.12: What is the pathophysiology of asthma?

Answer:

- **Type 2 inflammation:** associated with the Type 2 subset of CD4 T helper cells. Which produce IL-4, IL-5, IL-13 (If the patient is not responding to conventional drugs, then anti-IL-5 drugs: Mepolizumab, Benralizumab are used)
  - Reliever in all steps of Asthma severity ICS/ formoterol
  - Formoterol acts rapidly, and ICS reduces the airway inflammation
- **Controller medication** PYQ: FMGE 2019
  - Step 1 = None, on a need basis, like ICS/ formoterol
  - Step 2 = Low-dose ICS
  - Step 3 = Low-dose ICS/ Formoterol
  - Step 4 = Medium dose ICS/ Formoterol
  - Step 5 = Medium-High dose steroids/ LABA + LAMA (Ipratropium or Tiotropium)
  - Step 6 = Anti IgE (omalizumab) or anti IL-5 or anti IL-4 receptor antagonist

Q3.13: What are the Goals of Asthma Therapy in Asthma?

Answer: Goals of Asthma Therapy

1. Symptoms < 2 Times/week
2. Night-time awakening: <2 Times/month
3. Use of Reliever <2 Times/week
4. No more than 1 exacerbation/year
5. Can do daily activities
   - If children: Inhaler (teach the mother about how to use the inhaler)
   - If child <7 years: Spacer is attached
   - If infant: Spacer with facemask is attached
   - Otherwise, the drug delivery gets affected
   - Before upgrading from low to high dose steroids, check the compliance of the patient & inhaler techniques.

Q3.14: What is the Dosing for Asthma in patients ≥ 12 years old?

Answer:

Dosing for Asthma in patients ≥ 12 years old				
DRUG		Daily Dose (mcg)		
Genetic	Brand	Low	Medium	High
Beclomethasone	Qvar	100-200	>200-400	>400
Budesonide	Pulmicort	200-400	>400-800	>800
Ciclesonide	Alvesco	80-160	>160-320	>320
Fluticasone furoate	Arnuity Ellipta	100	N/A	200
Fluticasone dipropionate	Flovent	100-250	>250-500	>500
Mometasone furoate	Asmanex	110-220	>220-440	>440

YOURWISH

Q3.15: What are the key features of Allergic Broncho Pulmonary Aspergillosis?

Answer:

- ABPA is a hypersensitivity reaction to A. fumigatus
- Common in Asthma & Cystic Fibrosis patients
- Clinical features:
  - Coughing Bouts: Episodes of Bronchial obstruction with Brown Colour mucus plugs
  - Thick tenacious sputum
  - Shortness Of Breath
- Work-up
  - Spirometry/ flow volume curve: Obstructive pattern with FEV1/FVC ratio < 0.7
  - Eosinophilia
  - HRCT: Central Bronchiectasis
- Treatment of ABPA: **Antifungals** (Itraconazole, Voriconazole) + **Oral steroids** (prednisolone)
- ABPA exacerbation: Steroids

Q3.16: What are the important diagnostic criteria for Allergic Broncho Pulmonary Aspergillosis?

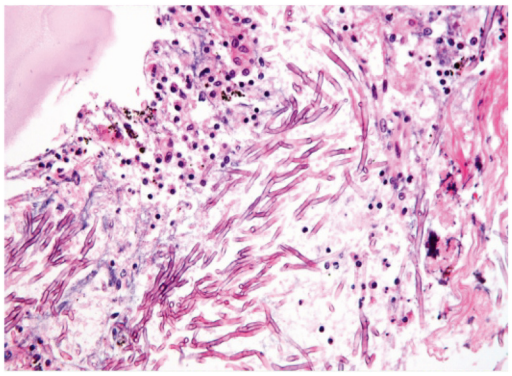
Answer: Important diagnostic criteria are:

- IgE for Aspergillus: Cardinal Diagnostic Test
- Total IgE > 1000 IU/mL
- Positive skin prick test
- If all 3 are present, then it is Fungal Asthma
- If any 1 or 2 are present, then it is Severe Asthma with Fungal Sensitisation (SAFS)
- SAFS is a variant of ABPA
- Fungal asthma is an infection with a fungus. Whereas ABPA is a hypersensitivity reaction to a fungus

PYQ: FMGE 2019

Q3.17: What are the key features of Aspergillus?

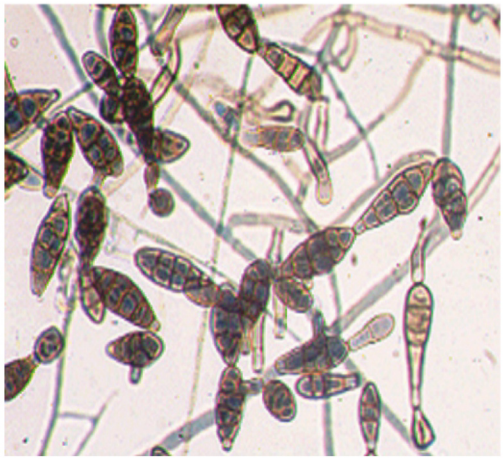
Answer:



- The image given shows Aspergillus species having thinner septate hyphae with regular branching, and they branch at acute angles (45° as opposed to 90°).
- Aspergillus is wrongly named as the yellow fungus.
- Aspergillus species: Aspergillus niger, Aspergillus fumigatus
- Aspergillus niger causes invasive aspergilloma
- DOC invasive aspergillosis: **Voriconazole**

Q3.18: What are the key features of Dematiaceous Fungi?

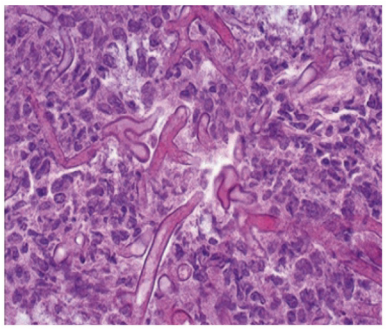
Answer:



- In the image given, a high concentration of melanin is present
- Dematiaceous fungi have a high concentration of melanin and melanin-like pigment in the cell wall, causing a dark pigment
- Black fungus is the for mucor mycosis

YOURWISH

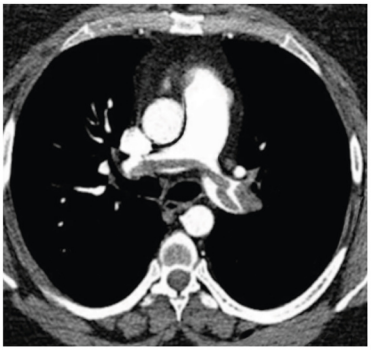
Q3.19: What are the key features of Mucor Mycosis?



Answer:

- The image given is of mucor mycosis because there are thick aseptate hyphae with variable branching.
- Test to identify Mucor Mycosis**: Nasal Endoscopy + Biopsy**
- Culture media: **SDA**
- For early diagnosis, the slide is examined on the same day because the disease can spread to the brain.

Q3.20: What are the CT angiography findings of Pulmonary Embolism?



Answer: CT Angiography

PYQ: FMGE 2021

PYQ: NEET PG 2022

- Saddle embolus in the pulmonary artery
- Leads to massive pulmonary embolism and RV failure

Q3.21: What are the causes of a Hypercoagulable state?

Answer:

- Malignancy
- Post-op THR (clots break from the femoral vein and can reach the pulmonary artery. Clots can be large or multiple small clots resulting in sub-massive pulmonary embolism)
- Factor V Leiden mutation: Defective binding between protein C and factor V. Activity of factor V is not under control → clot formation
- Antiphospholipid Antibody Syndrome: Activation of the intrinsic system of clotting → clot formation
- Prolonged immobilization
- Nephrotic syndrome: loss of protein C, protein S and antithrombin III leads to a hypercoagulable state
- COVID-19 delta variant

Q3.22: What is the workup for Pulmonary Embolism?

Answer:

- D- Dimer Assay: Screening test
- ECG: MC sinus Tachycardia, characteristic S1 Q3 T3 (deep S wave in lead I and deep Q wave in lead III and T wave inversion in Lead III)
- CXR: Hampton hump, **Palla sign** (due to obstruction of right descending Pulmonary Artery): massive Pulmonary Embolism
- CXR of sub-massive Pulmonary Embolism looks normal

Q3.23: What is the management of Pulmonary Embolism?

Answer:

Management:

- SBP <90mmHg thrombolytics are indicated
- Massive PE: ALTEPLASE + LMW HEPARIN
- Sub-massive PE: LMW HEPARIN (thrombolysis not done)
- Echo: Mc conell sign is seen
- Prevention of DVT: Compression stockings & Heparin (given post-op for Gynae, Pelvic and Ortho Surgery)

Q3.24: What are the key features of fat embolism syndrome?

Answer:

- Usual presentation - A patient with a fracture of a long bone following a bike accident develops respiratory distress after 24 to 48 hrs and goes into a coma.
- Diagnosis is fat embolism syndrome.
- Criteria for fat embolism is **GURD WILSON CRITERIA**
- Mortality is high in fat embolism syndrome

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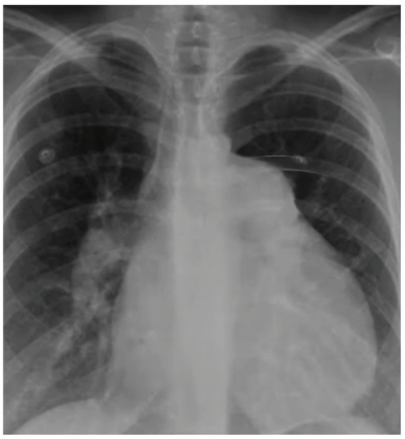
PYQ: NEET PG 2021

Q3.25: What are the key features of Pulmonary Artery Hypertension?

Answer:



- Chest X-ray shows **pulmonary artery prominence, Right Ventricular Hypertrophy.**
- Normal pulmonary artery pressure is 25/10mmHg
- Mean pulmonary artery pressure is 15 mmHg
- PAH is when (Harrison 21st update)
  - **Mean > 20mmHg**
  - **PA systolic pressure >35 mmHg**
- Clinical features:
  - Exercise intolerance
  - Presyncope
  - Chest pain

Q3.26: What are the Auscultation findings of Pulmonary Artery Hypertension?

Answer:

On Auscultation:

- Loud P_2 (due to high pressure in the pulmonary artery)
- Narrow split S_2
- Right-sided S3/S4 (due to RV decompensation and RA hypertrophy)
- RV Failure → Functional Tricuspid Regurgitation (tricuspid valve annulus dilated) → Leads to Pan-systolic murmur

Q3.27: What are the causes of RVEDP?

Answer:

- RA pressure ↑
- Epicardial venous pressure ↑
- Pericardial effusion due to increased pressure
- In some cases, scleroderma and CREST syndrome are also seen
- Clubbing

Q3.28: What is the workup of Pulmonary artery hypertension?

Answer: Work Up

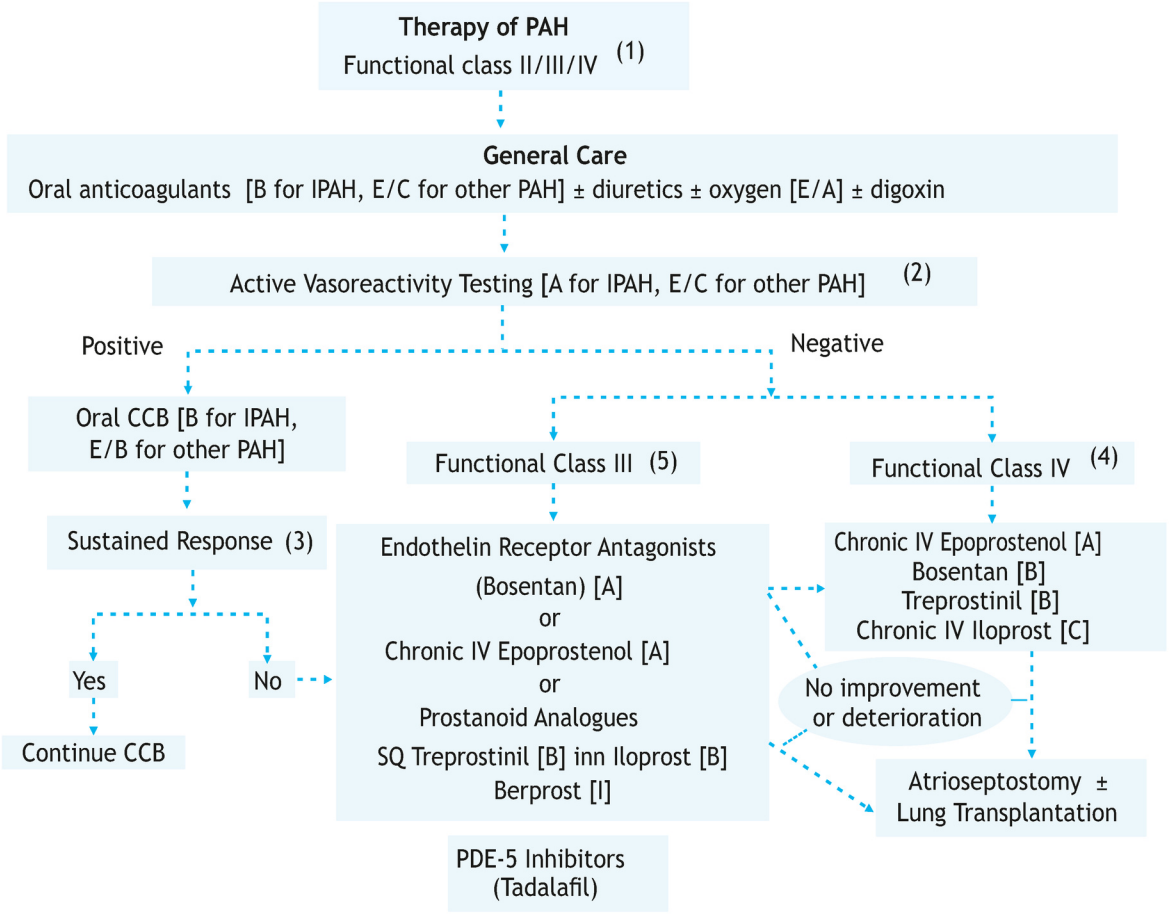
- CXR: Prominent PA, RVH, obliteration of the retrosternal space
- **Echo (Bubble contrast): Screening**
- 6 Min Walk Distance test: ask patient to check SPO_2 before and after 6 min walk
- Cardiopulmonary Exercise Test (CPET): Gives Peak volume of O_2 Consumption

- **Invasive hemodynamic monitoring, like cardiac catheterisation gold standard (do vasoreactivity test by giving inhaled NO: If result is positive, patient will respond to calcium channel blockers, and if response is poor, drugs other than CCBs are given)**
- Diffusion capacity of lungs for Carbon monoxide (DLCO): Decreases (because of plexiform arteriopathy, i.e., fibrosis in the inner lining of the PA and its branches, which leads to a decrease in the exchange of gases)
- HRCT chest: Enlargement of main PA - Prior to the block, PA gets dilated, and peripheral pruning of PA branches
- Nocturnal oximetry: nocturnal desaturation occurs

Q3.29: What is the treatment of Pulmonary artery hypertension?

Answer:

- Ambrisentan + Tadalafil = 50% mortality reduction
- IV epoprostenol + Bosentan + Sildenafil = 100%, mortality reduction (cons: small sample size)



Q3.30: What are the Determinants of the prognosis of Pulmonary artery hypertension?

Answer:

Determinants of prognosis [estimated 1 year mortality]	Low risk <5%	Intermediate risk 5-10%	High risk >10%
Clinical signs of right heart failure	Absent	Absent	Present
Progression of symptoms	No	Slow	Rapid
Syncope	No	Occasional syncope	Repeated syncope
WHO functional class	I, II	III	IV
6MWD m	>440	165-440	<165

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Cardiopulmonary exercise testing	Peak Vo_2 >15 mL min - kg^{-1} (>65% pred) Vt / Vco_2 slope <36	Peak Vo_2 11-15 mL min- kg^{-1} (35-65% pred) Vt / Vco_2 slope 36-44.9	Peak Vo_2 <11 mL min - kg^{-1} (<35% pred) Vt / Vco_2 slope >45
NT- preBNP plasma leves $ng\text{-}L^{-1}$	BNP <50 NT proBNP <300	BNP 50-300 NT proBNP 300- 1400	BNP >300 NT proBNP >1400
Imaging (echocardiography, CMR imaging)	RA area <18 cm^2 no Pericardileffusion	RA area 18-26 cm^2 no or minimal pericardial effusion	RA area <26 cm^2 pericardial effusion
Haemodynamics	RAP <8 mm Hg CI >2.5 L min^{-1} m^{-2} Svo2 >65%	RAP 8-14 mm Hg CI 2.0-2.4 L min^{-1} m^{-2} Svo2 60-65%	RAP >14 mm Hg CI <2.0 L min^{-1} m^{-2} Svo2 <60%

Q3.31: What is the WHO Classification of Various groups of Pulmonary artery hypertension?

Answer:

Group 1	• Group 1 - Pulmonary arterial hypertension (PAH) → No other cause detected then it is group 1. Due to Pulmonary Artery precapillary remodeling o Because of intimal fibrosis, classic plexiform lesions seen in pathology
Group 2	• Pulmonary hypertension due to left-sided heart disease • Group 2 examples: o Child with untreated VSD → blood goes from LV to RV (L→R shunt) → dilates the pulmonary artery → intimal fibrosis in PA → PAH develops. Shunt reversal occurs if PA pressure is more than LV pressure → Eisenmenger complex o Scleroderma is a connective tissue disorder & patient develops pulmonary artery fibrosis and Pulmonary artery Hypertension o COPD: Hypoxia cause vasodilation of all blood vessel except pulmonary artery (vasoconstriction occurs). Because of vasoconstriction in PA leads to PAH & later develops RVF K/a Cor pulmonale
Group 3	• Pulmonary hypertension due to lung diseases and/or hypoxia
Group 4	• Chronic thromboembolic pulmonary hypertension (CTEPH)
Group 5	• Pulmonary hypertension with unclear or multifactorial etiologies, including hematologic disorders (e.g., Myeloproliferative disorders), systemic disorders (e.g., Sarcoidosis, pulmonary Langerhans cell histiocytosis, lymphangioleiomyomatosis, neurofibromatosis, vasculitis), metabolic disorders (e.g., Glycogen storage disease, Gaucher disease, thyroid disorders), and miscellaneous conditions (e.g., Tumor obstruction, mediastinal fibrosis, chronic renal failure on dialysis)

Q3.32: What are the key features of Hemoptysis?

Answer:

- **Life-threatening haemoptysis**
  - **> 400 ml/ day**
  - **> 100-150 ml: Single sitting**
- **The most common cause of small volume haemoptysis is viral bronchitis > chronic bronchitis.**
- Most common cause of massive haemoptysis: **Bronchiectasis**, TB
- The leading cause of death in bronchiectasis is life-threatening haemoptysis

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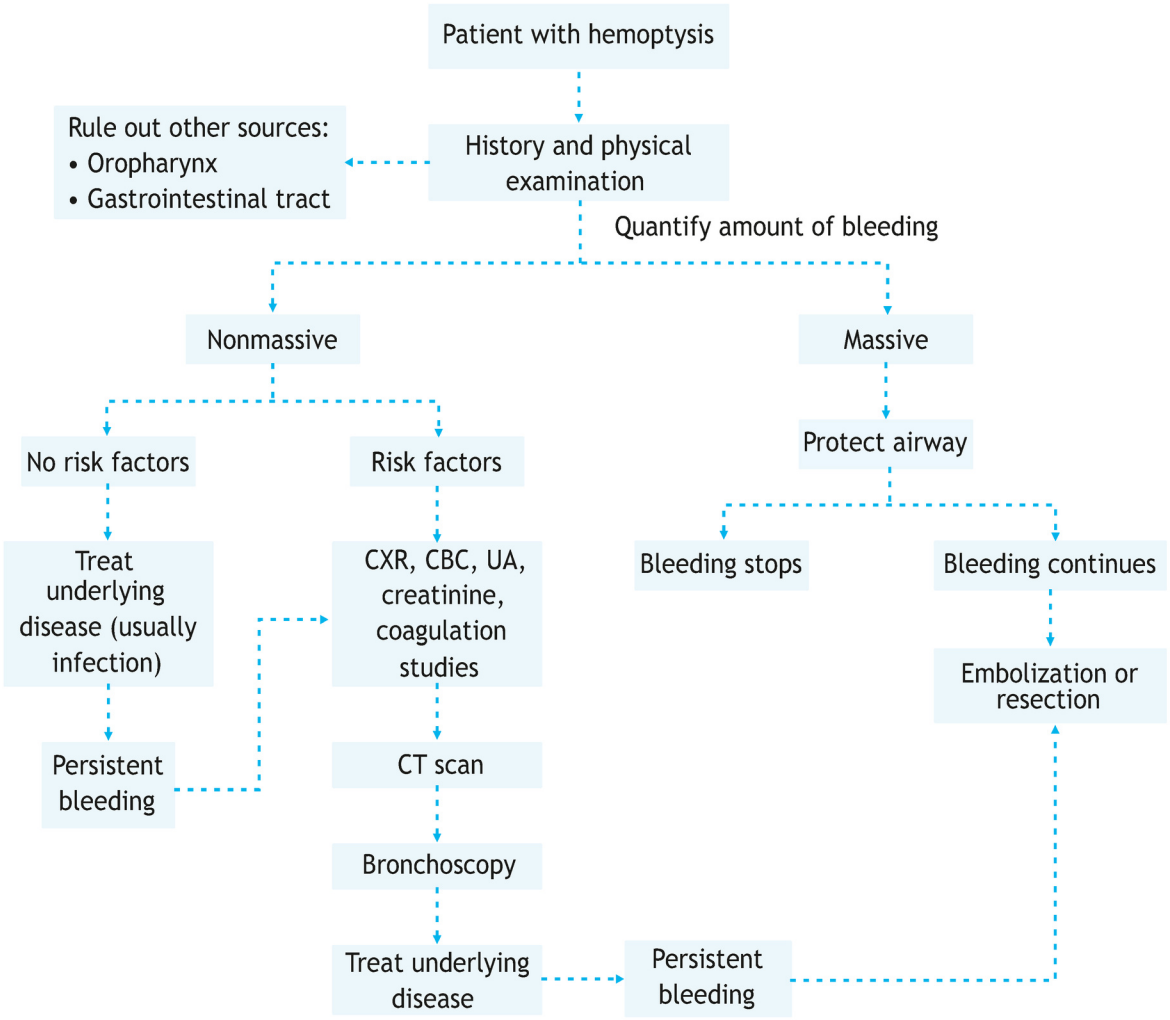
- Invasive aspergilloma is caused by Aspergillus Niger, leading to cavity formation in the lungs in diabetic patients or immunocompromised patients or COVID-19 patients on steroids
- **TB is the commonest cause of massive haemoptysis in the Indian population**
- The artery affected in TB is the bronchial artery (thin-walled); rupture leads to massive haemoptysis
- In TB, rupture of the pulmonary artery is less common (Since it has more connective tissue) and leads to RASMUSSEN ANEURYSM
- Endemic haemoptysis: P.westermani (lung fluke). Source: uncooked seafood

Q3.33: What is the treatment of Hemoptysis?

Answer:



- Lie with bleeding side dependent
- Avoid ET if cough reflex is present; If required, selective intubation ofthe non-bleeding lung
- CTA: to locate the site
- Flexible Bronchoscopy: Suction clot, Balloon Catheter, tamponade effect OR Rigid Bronchoscopy: Photo coagulation
- TOC for massive haemoptysis is Bronchial Artery Embolisation
- S/E: Paraplegia due to Anterior Spinal Artery embolisation
- >40 years old smoker with non-massive haemoptysis, do CT scan > CXR
- If high-risk features are present, do CT scan
- Life-threatening haemoptysis
  - o > 400 ml/ day
  - o > 100-150 ml: Single sitting

Q3.34: What are the causes of Hemoptysis?

Answer:

- Mitral stenosis: Source of bleeding - Bronchial veins
- LVF
- PE: source - Pulmonary artery and its branches
- Goodpasture: Anti-basement membrane antibody
- Diffuse Alveolar Haemorrhage: SLE, Cocaine
- Pulmonary - Renal syndrome: Granulomatosis Polyangiitis (GPA)
- Vaping-induced lung injury
- Malignancy - Bronchogenic Carcinoma: Small cell carcinoma/SCC & metastasis
- ILD associated with smoking: Desquamative interstitial pneumonitis (DIP), Respiratory Bronchiolitis-ILD, Pulmonary Langerhans cell histiocytosis (PLCH)

Q3.35: What are common causes of pericardial effusion

Answer: Pericardial effusion is seen in infections like tuberculosis (TB) and malignancies such as lung cancer.

Q3.36: What is the normal volume and function of pericardial fluid

Answer: Normal pericardial fluid volume is 20 to 50 ml, and it serves to lubricate the heart.

Q3.37: What determines the production of pericardial fluid, and what happens when pressure increases

Answer: Pericardial fluid is produced by the epicardial venous plexus. If the pressure in the epicardial venous plexus increases, it can lead to pericardial effusion.

Q3.38: What are the features of CREST syndrome?

Answer: CREST syndrome includes:

- Calcinosis cutis
- Raynaud's phenomenon
- Esophageal dysmotility
- Sclerodactyly
- Telangiectasia

Q3.39: Which diagnostic tool is used in hepatopulmonary syndrome to detect shunts

Answer: Bubble contrast echocardiography is used in hepatopulmonary syndrome.

Q3.40: In which conditions is DLCO (Diffusion Capacity of the Lung for Carbon Monoxide) decreased?

Answer: DLCO is decreased in:

- Interstitial lung disease (ILD)
- Pulmonary fibrosis
- Idiopathic pulmonary fibrosis
- Respiratory bronchiolitis

Q3.41: What tests are used to diagnose Obstructive Sleep Apnea (OSA)

Answer: Polysomnography and nocturnal oximetry are used to diagnose Obstructive Sleep Apnea (OSA).

10.LIVER, GIT & HEMATOLOGY

BIG QUESTION 1: Describe the scoring systems used in hepatology to assess liver disease severity and guide treatment decisions.

Broad Answer: Scores to assess liver disease severity

MELD Score	• It is a metric which helps us calculate 3-month mortality for any newly diagnosed case of end stage liver disease • The minimum score possible is 6 and maximum is 40, depending on how high or how low the score is we can also prioritize the allocation of liver transplantation • International body that validates this algorithm is UNOS (United Network of Organ Sharing) • A patient is enlisted for Orthotopic liver transplantation if the MELD score is > 17
PELD Score	• Calculates the pediatric version of the MELD score for liver cirrhosis severity and transplant planning. • Pediatric version of MELD score
Child Pugh Score	• It helps to differentiate whether the patient is having compensated Cirrhosis or Decompensated cirrhosis. The development of portal HTN indicates that the person has decompensated cirrhosis • A patient is enlisted for Orthotopic liver transplantation if the Child Pugh score is ³ 7 o 7-9: Decompensated o < 7: Compensated
Nazer's Index	• It is used in patients of Wilson's disease • It helps determine the prognosis of Wilson's disease • Also helps to determine if the patient has to be treated medically or by orthotopic liver transplantation (OLT) o Medical therapy: With Zinc and subsequently Copper chelators like penicillamine, trientine and dimercaprol.
Maddrey Discriminant Score	• Used in patients with alcoholic hepatitis • This is mainly to guide the treatment for Alcoholic hepatitis • DOC for the management of Alcoholic hepatitis is Prednisolone

DETAILED QUESTION

Q1.1: What are the key parameters used in hepatology to assess liver disease severity?

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Answer: Parameters to assess liver disease severity:

MELD- Na^+ Score	PELD Score	Nazer Index	Child Pugh Score	Discriminant Score
• MELD score will be having 3 parameters: o Bilirubin o International normalized ratio (INR),	• Parameters of PLED score: o Bilirubin o INR o Serum Albumin	• Bilirubin • INR • SGOT	• Parameters include: o Bilirubin o INR	• Parameters include: o Bilirubin o INR

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o Serum creatinine • Minimum Score is 6 and maximum is 40 • A person with a higher MELD score will have a higher mortality	o Serum Albumin o Age o Growth failure parameters		o Serum Albumin o Ascites o Asterixis (Flapping tremors)	

Q1.2: What is the most accurate test for determining the severity of chronic liver disease (CLD)?

Answer:

- The most accurate test is a **liver biopsy**
- Prior to the procedure, a patient's coagulogram must be checked because liver damage often leads to low levels of Factor VII

Q1.3: What are the non-invasive tests for CLD?

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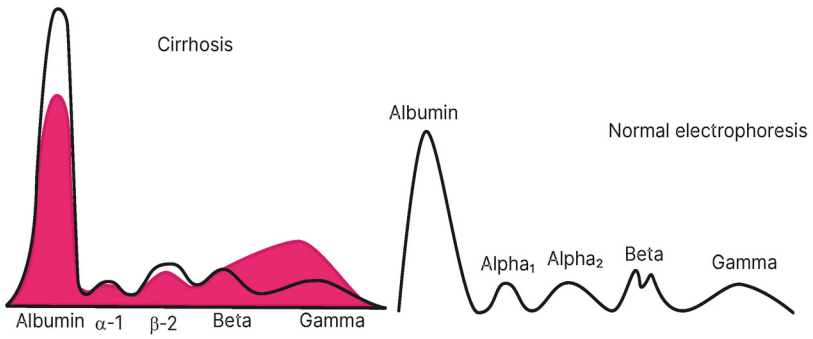
Answer:

- The two non-invasive tests are:
  - o Transient Elastography
  - o FibroTest
    - → It uses parameters such as bilirubin, gamma-GGT, haptoglobin, apolipoprotein-A, and alpha-2 macroglobulin
  - o Albumin-Globulin ratio:
    - → Albumin values will be less, and gamma globulin values will be increased (globulin is elevated because catabolism of globulin occurs in the liver)
    - → In patients with nephrotic syndrome, both albumin and globulin values decrease as there is urinary loss of globulin



Q1.4: What are the scores to assess CLD by a biopsy?

Answer:

- Metavir score (F0-F4) and
- Ishak score (0-6)

Q1.5: What is the most specific liver function test (LFT)?

Answer: SGPT

Q1.6: What is the most specific test for alcoholic hepatitis?

Answer:

- SGOT
- SGOT/SGPT ratio > 1

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Q1.7: What is the most specific test for alcoholism?

Answer: Gamma-GGT

Q1.8: What is the best test for obstructive jaundice, and how does it present?

Answer:

- Serum alkaline phosphatase (SAP)
  - Fourfold elevation
    - → In bone-specific SAP, we disproportionate rise is seen in Paget's Disease
    - → Bone-specific SAP will be normal in: Osteoporosis, Multiple myeloma

Q1.9: What is the most common cause of cirrhosis and cirrhosis requiring an orthotopic liver transplant (OLT)?

Answer: Non-alcoholic Fatty Liver Disease (NAFLD)

BIG QUESTION 2: What are the common causes of cirrhosis?

Broad Answer:

- ALD
- Hepatitis B/C/D
  - Hepatitis C is considered more dangerous than Hepatitis B because it can cause the gradual destruction of the liver, which can lead to hepatitis-induced cirrhosis
  - DOC for management of Hep B: Tenofovir/ Entecavir
- Autoimmune hepatitis
  - Histopathology of AIH shows Interface Hepatitis, and Rosette formation
- Primary Biliary cirrhosis/ Primary sclerosing cholangitis
- Non-Alcoholic Steatohepatitis (NASH)
- Wilson disease/hemochromatosis
- α-1 antitrypsin deficiency: ZZ phenotype present
- Cardiac cirrhosis
- Cryptogenic cirrhosis/ idiopathic

Q2.1: What is the difference between Type-1 and Type-2 AIH?

Answer:

Type 1 Autoimmune hepatitis	Type-2 Autoimmune Hepatitis
Important antibodies include: • Anti-Smooth Muscle Antibody (SMA) • Anti-Soluble Liver Antigen antibody (SLA) • Atypical P-ANCA / X-ANCA • ANA +ve	Important antibodies include: • Anti- LKM1 (Liver Kidney Microsomal1) Antibody • ANA -ve • Anti-Liver cytosol

Q2.2: How do Primary Biliary Cirrhosis (PBC) and Primary Sclerosing Cholangitis (PSC) differ, and what are their specific antibodies?

Answer:

- PBC is a pure intrahepatic problem, and its important antibody is the Anti-mitochondrial antibody
- PSC is an extrahepatic problem with fibrosis in the bile duct, and its important antibody is P-ANCA

Q2.3: What are the causes of primary and secondary hemochromatosis?

Answer:

- Primary hemochromatosis is caused by a defective HFE gene
- Secondary hemochromatosis is caused by packed RBC transfusions

Q2.4: Which conditions are associated with anti-LKM antibodies?

Answer:

- Anti-LKM1 antibody is associated with: AIH Type-2, HCV
- Anti-LKM2 antibody is associated with: Drug-induced hepatitis
- Anti-LKM3 antibody is associated with: HDV

BIG QUESTION 3: Discuss the pathophysiology, clinical manifestations, diagnosis, and management of portal hypertension.

Broad Answer:

- Portal hypertension is a condition where the hepatic venous pressure gradient (HVPG) is greater than 5 mmHg.
- The key clinical signs are splenomegaly, ascites, and the formation of portosystemic collaterals at various sites, including esophageal varices, rectal hemorrhoids, and the caput medusae around the umbilicus.
- The initial diagnosis of portal hypertension is made with a Doppler ultrasound.
- The management of non-bleeding esophageal varices involves medications like nadolol or propranolol.

DETAILED QUESTIONS

Q3.1: At what hepatic venous pressure gradient (HVPG) is portal hypertension considered to have started, and at what pressure do esophageal varices occur?

Answer:

- Portal hypertension: HVPG is > 5 mmHg
- Esophageal varices: HVPG > 12 mmHg

Q3.2: What are the key clinical manifestations and diagnostic tools for portal hypertension?

Answer:

- Splenomegaly
  - It is an early and reliable finding
  - As the pressure in the portal vein will rise due to progressive fibrosis occurring in the liver, the congestion will be transmitted to the splenic vein; therefore, the spleen of the patient will become enlarged
- Ascites
  - Puddle sign
- Portosystemic collaterals
- The investigation of choice for portal hypertension is a doppler ultrasound

Q3.3: List the sites of portosystemic collaterals.

Answer:

- Esophageal varices
- Rectal hemorrhoids
- Bare area of the liver

- Caput medusae: Dilated vessels around the umbilicus
- Omental Veins
- Retroperitoneal veins
- Lumbar vein

Q3.4: How to differentiate between caput medusae and superior/inferior vena cava thrombosis?

Answer:

- Dilated vessels around the umbilicus and flow of blood is away from the umbilicus: Caput medusae
- Dilated vessels above the umbilicus and flow of blood is towards the umbilicus: Superior vena cava thrombosis
- Dilated vessels below the umbilicus and flow is towards the umbilicus: Inferior vena cava thrombosis
- Spider Angiomas: Seen in cirrhosis, Pregnancy, and Rheumatoid arthritis. Spider angiomas are arterioles that are seen in the Superior vena cava distribution (face, neck, upper part of the trunk, arms, and fingers).

Q3.5: How is portal hypertension classified?

Answer:

Prehepatic	Hepatic	Post-Hepatic
• Portal vein thrombosis • Splenic vein thrombosis • BANTI Syndrome	• Sinusoidal: Cirrhosis • Pre-Sinusoidal: Schistosomiasis • Post Sinusoidal: Radiation, Herbal tea, Endemic Ascites	• Budd Chiari Syndrome • IVC thrombosis • Cardiac Cirrhosis

Q3.6: What are BANTI syndrome and BANTU disease?

Answer:

- BANTI syndrome refers to idiopathic portal hypertension
- BANTU disease is an iron-related liver damage seen in African populations

Q3,7: What is the most common cause of hematemesis? How does the presence of splenomegaly aid in differential diagnosis?

Answer:

- The most common cause of hematemesis is peptic ulcer disease
- The presence of splenomegaly along with hematemesis is a key clinical pointer toward portal hypertension

Q3.8: How are non-bleeding esophageal varices and bleeding esophageal varices managed differently?

Answer:

Non-Bleeding Oesophageal Varices	Bleeding Oesophageal Varices with very low BP
• Goal: Prevent first episode of variceal bleed (primary prophylaxis) • Drug of Choice (DOC): o Non-selective ß-blockers Propranolol or Nadolol • Propranolol - Other Important Uses: o Hypertrophic Obstructive Cardiomyopathy (HOCM) o Migraine prophylaxis o Thyrotoxicosis - controls sympathomimetic symptoms	• The initial aim is to stabilize the patient (volume resuscitation): Securing a grey cannula and starting IV fluids, followed by massive • Blood transfusion o IV fluid for compensated shock: Ringer's lactate o IV fluid for decompensated shock / Crashing patient: Normal saline o Preferred method of volume resuscitation: Massive blood transfusion

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o Primary prophylaxis in non-bleeding oesophageal varices	• IV Octreotide • Once the bleeding is reduced and BP is gradually returning to normal, then do a Upper GI Endoscopy and Endoscopic Variceal ligation or Sclerotherapy

Q3.9: What is the management plan for recurrent bleeding from esophageal varices?

Answer:

- Assuming the patient is hemodynamically stable, repeated endoscopic variceal ligation is performed
- If the bleeding persists, a TIPS (Transjugular Intrahepatic Portosystemic Shunt) procedure is preferred if the patient's Child-Pugh score is Class-A (5-6)
- If the score is ≥7, an evaluation for a liver transplant is performed

Q3.10: What is portal gastropathy, and what are its endoscopic appearance and treatment?

Answer:

- Portal gastropathy is a condition where increased pressure in the gastric veins leads to a continuous oozing of blood into the stomach
- On endoscopy, it presents as a "mosaic appearance" of blood vessels in the stomach mucosa
- Progressive anemia, despite endoscopic variceal ligation, is a strong indicator.
- The treatment is a TIPS procedure

BIG QUESTION 4: Discuss ascites and its complications, including their clinical features, diagnosis, and treatment.

Broad Answer:

- Ascites is the accumulation of fluid in the peritoneal cavity. While >100 mL of fluid can be detected by a puddle sign, larger volumes (>500 mL) present with shifting dullness, a fluid wave sign, and an everted umbilicus.
- The primary treatment is a salt-restricted diet, combined with diuretics like spironolactone and furosemide. In refractory cases, medications like midodrine or a large volume paracentesis (LVP) may be used, with albumin administration to prevent dehydration.

Q4.1: How is ascites detected clinically, and what are the signs of significant fluid accumulation?

Answer:

- >100 ml of fluid in the peritoneal cavity: Puddle sign
- When the fluid amount is >500 ml:
  - o Shifting dullness can be elicited
  - o Fluid wave sign:
    - → It can be a false positive in a person having a pendulous abdomen, like in a Grand multipara or in patients with centripetal obesity due to metabolic syndrome
  - o Superficial veins of the anterior abdominal wall will become prominent (tortuous)
  - o Everted umbilicus
  - o Hepatic Hydrothorax
  - o Abdominal compartment syndrome:
    - → Manifests when the pressure is > 25 mmHg
- Minimum amount of ascitic fluid that can be clinically detected: 1500-2000ml

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Q4.2: What is the treatment for ascites, including medications and invasive procedures?

Answer:

- Salt-restricted diet
- Spironolactone + Furosemide:
  - Eplerenone or amiodarone can be used instead of spironolactone because spironolactone can cause gynecomastia as S/E
  - Dose can be increased up to 400 mg of Spironolactone and 160 mg of Furosemide per day
- Midodrine / Clonidine: Constricts the Splanchnic blood vessels, which will reduce ascites
- Large volume paracentesis (LVP):
  - Risk of dehydration, causing an increase in NH_3 concentration, leading to Asterixis
  - It can be prevented by giving salt-free albumin that will increase the oncotic pressure resulting in an increase in circulating fluid volume and thereby decreasing the magnitude of dehydration
- TIPS:
  - Superior treatment to prevent re-accumulation
  - S/E: Hepatic encephalopathy
- ß-Blockers tend to increase mortality whenever the patient has gone into refractory ascites

Q4.3: Describe the SAAG (serum albumin ascites gradient)

Answer:

SAAG (serum albumin ascites gradient)

SAAG value > 1.1g/dl:
Ascitic Fluid is checked for protein

- Amount of protein in Ascitic Fluid is **< 2.5g/dl:**
  - Late Budd Chiari syndrome: due to scarring the movement of protein from the sinusoid could be affected
  - Cirrhosis
  - Massive liver metastasis
- Amount of protein in Ascitic Fluid is **>2.5g/dl:**
  - CHF: In any patient of CHF, there could be increased pressure in the portal vein. Higher pressure will cause albumin to move out into Ascitic Fluid hence Ascitic Fluid albumin value will be more than 2.5g/dl
  - Early Budd Chiari syndrome
  - IVC Obstruction
  - Sinusoidal Obstruction

SAAG value < 1.1g/dl: Condition associated with it include (Mnemonic: BNP2T)

B - Bile leak
N - Nephrotic syndrome: Nephrotic syndrome causes transudative pleural effusion but here serum albumin and ascitic Fluid protein will become less due to urinary loss, So the SAAG value will become < 1.1g/dl
P - Peritoneal Carcinomatosis: this include Mesothelioma, Ca Ovary, Ca Stomach
P - Pancreatitis
T - Tuberculosis

Q4.4: A patient with refractory Ascites started having fever, there is an increase in the girth of the abdomen, and simultaneously, encephalopathy has been noted. Why is he having this fever, and why is there a sudden worsening of this patient?

Answer:

- His condition is caused by Spontaneous Bacterial Peritonitis (SBP)

- In this patient, bowel loops are surrounded by ascitic fluid
- E. coli present inside the bowel loop transmigrates to the lymph nodes as a result, neutrophils are activated, and they enter the peritoneal fluid
- Once the patient develops peritonitis, it will cause fever, an increase in abdominal girth, and later progress to septic shock
- An increase in the ammonia concentration in the body leads to hepatic encephalopathy

Q4.5: How is SBP diagnosed and treated?

Answer:

- Diagnosis is done by abdominal paracentesis
  - Send for Gram stain, cytology, and culture
  - Cytology: Microscopic examination shows > 250 neutrophils present per cubic mm
- Treatment: IV Ceftriaxone
- Prevention::
  - Early large-volume paracentesis, i.e., inthe first 12 hrs of admission
  - Norfloxacin

Q4.6: What is hepatorenal syndrome, and what is its treatment?

Answer:

- HRS is the development of acute kidney injury (AKI) in a patient with refractory ascites, where the patient's urine output decreases
- The treatment is a combination of salt-free albumin and octreotide/midodrine

Q4.7: What is hepatopulmonary syndrome, and how is it diagnosed?

Answer:

- HPS is a condition characterised by:
  - Platypnea / Orthodeoxia:
    - → Platypnea is shortness of breath that is worsened by being upright (standing or sitting) and improves when lying supine (flat)
    - → Orthodeoxia: Fall in SpO_2 values by 5% / PO_2 > 4mmHg / PaO_2 <80mmHg, when changing from supine to sitting position
  - Cirrhosis (decompensated)
  - Intrapulmonary shunting
    - → IOC: Bubble contrast echocardiogram

Q4.8: What are the early clinical manifestations of hepatic encephalopathy?

Answer:

- Early manifestations include
  - Confusion
  - Changes in personality
  - Alteration in the sleep-wake cycle
  - Number connection test: Apraxia
  - Anomic aphasia
  - Asterixis (Negative myoclonus)

- Stupor
- Coma

Q4.9: How is hepatic encephalopathy investigated and treated?

Answer:

- Investigation
  - Blood ammonia levels: Elevated
  - EEG: Triphasic waves are present
- Treatment
  - Lactulose (ammonia binder) is administered via NG tube
  - Rifaximin/ Neomycin:
    → Bacteriostatic antibiotics help in reducing bacterial load
  - LOLA (L-Ornithine and L-Aspartate): Ornithine and Aspartate help in clearing ammonia from the body
    → Site of ammonia production in the body: Gut bacteria
  - Fresh frozen plasma

Q4.10: What are the key triggers for hepatic encephalopathy?

Answer:

- Excessive use of diuretics
- Internal GI bleed
- Large volume Paracentesis

BIG QUESTION 5: Discuss acute viral hepatitis, its common causes, and the diagnostic workup.

Broad Answer:

- Most common cause of **acute viral hepatitis**: **Hepatitis E**
- Clinical presentation:
- Fever
- Nausea/vomiting
- Tender hepatomegaly
- Diagnostic workup:
  - IgM Anti-HAV (Hepatitis A)
  - HBsAg and IgM Anti-HBc (Hepatitis B)
  - Anti-HCV (Hepatitis C)

DETAILED QUESTIONS

Q5.1: How do various causes of acute viral hepatitis present on work-up?

Answer:

HBsAg	IgM Anti HAV	IgM Anti HBc	Anti HCV	Diagnosis
+ve	-ve	+ve	-ve	Acute Hep-B
-ve	+ve	-ve	-ve	Acute Hep-A
-ve	-ve	-ve	+ve	Acute Hep-C
-ve	-ve	-ve	-ve	Non A, Non B, Non C

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Q5.2: What is the diagnostic workup for Hepatitis A, B, and C?

Answer: PYQ: NEET PG 2019, 2020, 2021, 2022 | PYQ: INICET 2022, 2023 | PYQ: FMGE 2020, 2021, 2022

HBsAg	Anti HBs	Anti HBc	HBeAg	Diagnosis
+	-	IgM	+	Acute Hep-B with high infectivity
+	-	IgG	+	Chronic Hep-B
-	+	IgG	-	Recovery
-	+	-	-	Vaccine
-	-	IgM	-	Gap period
-	-	IgG	-	Hep- B remote infection in the past / Low level carrier

Q5.3: What are the routes of transmission of Hepatitis B?

Answer:

- Leading cause of transfusion-associated hepatitis: Hepatitis B
- MC route: Percutaneous route (sexual contact)
- Transmission of hepatitis B in accidental needle stick injury: 30%

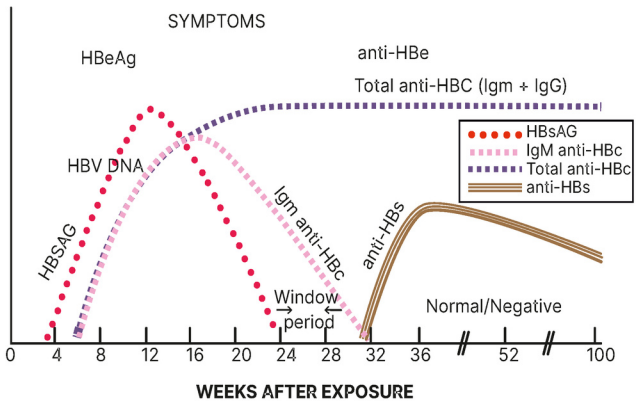
Q5.4: What is the protective antibody for Hepatitis B, and what titer indicates safety in a vaccinated person?

Answer:

- Protective antibody: Anti-HBs
- In a vaccinated person, the titer of anti-HBs is > 10 IU, then the person is safe
- If the titer of anti-HBs is < 10 IU, then a booster dose of Hepatitis B vaccine is given

Q5.5: What is the window period in Hepatitis B infection?

Answer: The window period is the time between the disappearance of HBsAg after exposure and the appearance of protective antibodies (Anti-HBs)



Q5.6: What is the treatment for acute Hepatitis B, and what are the criteria for initiating treatment in chronic Hepatitis B?

Answer:

- Treatment of choice: Entecavir or Tenofovir
- Conservative management
- Recovery rate is 90%
- In some patients, it may be converted to chronic hepatitis B
  - o Treatment for chronic Hepatitis B is initiated when HBV DNA is >2×104 IU/mL and the SGPT values have doubled

YOURWISH

Q5.7: What is the treatment for Hepatitis C and Hepatitis D?

Answer:

- Hepatitis C: Sofosbuvir and Velpatasvir
- Hepatitis D: α-Interferon

Q5.8: What is the major route of transmission of Hepatitis D, and what is its role in fulminant hepatic failure?

Answer:

- Major route of transmission for Hepatitis D: Parenteral
- A co-infection with Hepatitis B and D can lead to fulminant hepatic failure, with Hepatitis D being one of its leading causes.

Q5.9: What is the leading cause of fulminant hepatic failure during pregnancy?

Answer: The leading cause of fulminant hepatic failure during pregnancy is **Hepatitis E**.

BIG QUESTION 6: Detail the radiological and clinical features of various esophageal and gastric pathologies.

Broad Answer:

- **Zenker's diverticulum** is a pulsion diverticulum characterized by dysphagia and regurgitation of undigested food.
- **Esophageal candidiasis**, often seen in patients on inhaled corticosteroids, shows **shaggy margins on a barium swallow**.
- **Achalasia cardia** presents with a **"bird beak" appearance** on barium swallow due to an increased tone of the lower esophageal sphincter (LES).
- **Esophageal carcinoma** shows an **irregular filling defect or "shouldering sign"** on barium swallow.
- Other conditions, like **diffuse esophageal spasm,** show a **"corkscrew" appearance** on barium swallow, while a **nutcracker esophagus** is diagnosed by manometry, showing increased contraction amplitude.

DETAILED QUESTIONS

Q6.1: A patient presents with odynophagia for several weeks, with no improvement despite multiple medical consultations. The patient has a history of asthma and is on Foracort (inhaled corticosteroids). His barium swallow is given below. What is the most likely diagnosis?



A. Zenker Diverticulum

B. Achalasia

C. Esophageal Angina

D. Esophageal Candidiasis

Answer: D. Esophageal Candidiasis

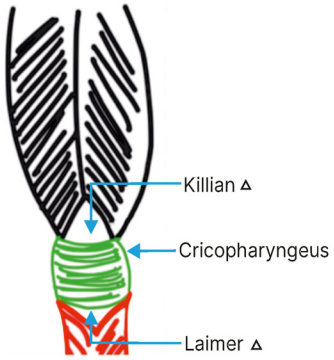
- Barium swallow shows shaggy margins of the esophagus
- The patient has developed esophageal candidiasis
  - Corticosteroids act as immunosuppressants
- In Zenker's diverticulum, there will be dysphagia, but odynophagia will not be present
- In Esophageal angina, the patient will have chest pain after eating food

Q6.2: What are the key clinical and radiological features of Zenker's diverticulum?

Answer:

- It is a pulsion diverticulum
- There is a defect present above the cricopharyngeus muscle, k/a Killian triangle, and a defect below the cricopharyngeus, k/a Laimer's triangle and Zenker diverticulum can occur in either of the two defects
- Diagnosis: Barium swallow





Q6.3: What are the key clinical features of Zenker's diverticulum?

Answer:

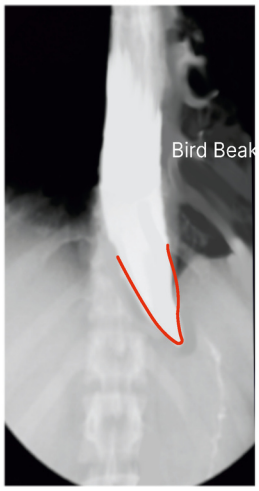
- Dysphagia
  - Most patients (98%) present with some degree of dysphagia
- Regurgitation of undigested food hours after eating
- Sensation of food sticking in the throat
- Special maneuvers to dislodge food
- Coughing after eating
- Aspiration of organic material
- Unexplained weight loss
- Fetor-ex-ore (halitosis)
- Borborygmi in the neck

Q6.4: What is the characteristic barium swallow finding for achalasia cardia?

PYQ: FMGE 2021

Answer:

- Barium swallow shows a bird beak-like appearance
- The leading cause of Achalasia cardia is Autoimmune
  - It can be caused by the paraneoplastic manifestation of lung cancer
- In Achalasia cardia, the tone of the lower esophageal sphincter will be increased
  - Normal tone of LES: 15-25mmHg
- D/D: Scleroderma
  - Scleroderma also presents in a female patient with dysphagia, reflux esophagitis, decreased LES, and Raynaud's phenomenon

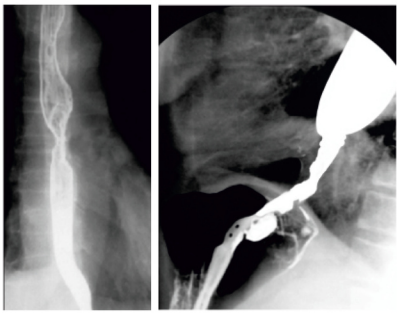


Q6.5: What are the radiological findings for esophageal carcinoma, and what are its clinical presentations?

PYQ: FMGE 2020 PYQ: INICET 2020

Answer:

- A barium swallow shows an irregular filling defect: Shouldering sign
- Clinical presentation includes progressive dysphagia in a person over 55, halitosis, and regurgitation
- Dysphagia typically occurs when more than two-thirds of the esophageal lumen is obstructed.

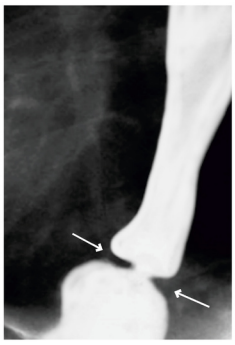


Q6.6: What is a Schatzki ring?

PYQ: FMGE 2022 PYQ: INICET 2021 PYQ: NEET PG 2022

Answer:

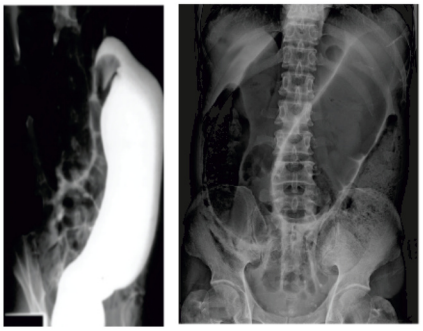
- A Schatzki ring is a circular, membranous structure of mucosa and submucosa at the squamocolumnar junction of the distal esophagus
- Schatzki ring appears as thin membranous structures that do not contain any muscularis propria
- Food impaction/Meat impaction occur in these patients



Q6.7: What are the radiological features and management of sigmoid volvulus?

Answer:

- Radiological features:
  - Coffee bean sign in the X-ray abdomen
  - Bird beak appearance in barium enema
- Treatment: Endoscopic detorsion



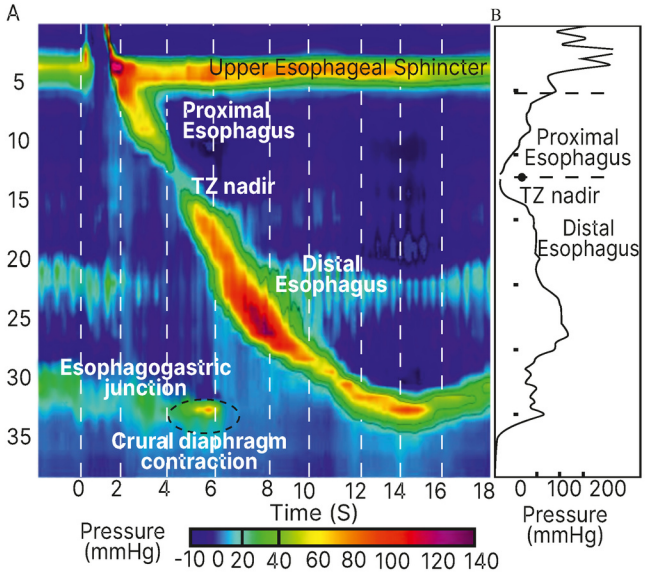
Barium enema "Bird beak" Sigmoid volvulus

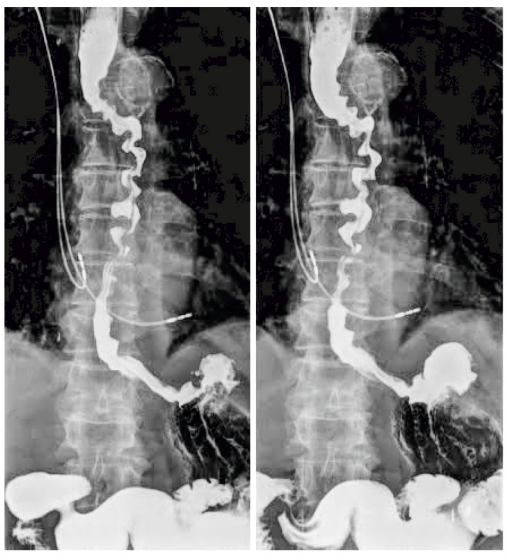
Q6.8: What is the difference between Diffuse Esophageal Spasm and Nutcracker Esophagus?

Answer:

	Diffuse Esophageal Spasm	Nutcracker Esophagus
Manometry Findings	• Normal contraction amplitude • Uncoordinated contractions o >20% simultaneous contractions (>30 mmHg) in distal 3/5 of esophagus o Repetitive LES contractions o Aperistalsis in 30% of wet swallows	• Increased contraction amplitude (> systolic perfusion pressure) • Coordinated contractions

PYQ: FMGE 2021 PYQ: NEET PG 2021





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Q6.9: How is DES diagnosed?

Answer:

- Barium swallow: Corkscrew appearance/Segmentation appearance/Rosary bead appearance
- IOC: High-resolution manometry
  - > 20% of contractions being simultaneous with amplitude >30 mmHg in distal 3/5th of the esophagus
  - Repetitive contractions of the LES
  - Aperistalsis in 30% of wet swallow

Q6.10: What are the clinical symptoms and management for DES?

Answer:

- Clinical presentation:
  - Dysphagia for both solids and liquids
  - Chest pain on eating quickly or on consuming hot or cold beverages
- Management:
  - Anxiolytics
  - Nitrates
  - CCBs
  - Not highly efficacious, it helps in relieving symptoms

Q6.11: What are some important upper GI endoscopic findings seen in various conditions?

Answer: PYQ: NEET PG 2022 PYQ: FMGE 2019, 2020 PYQ: INICET 2021

1. Feline Esophagus	• Found in **Eosinophilic esophagitis** • Feline esophagus is associated with GERD and Asthma • Biopsy is done to confirm the diagnosis of Eosinophilic esophagitis • In biopsy there should be demonstration of > 15 eosinophils/HPF
2. Serpiginous ulcer	• Esophageal ulcer in CMV infection
3. Volcano Ulcer	• Esophageal ulcer in HSV infection
4. Cerebriform appearance of stomach mucosa	• Menetrier's disease • Foveolar cells + • Rx: Cetuximab
5. Watermelon Stomach	• Gastric antral vascular ectasia
6. Cracked mud appearance (fissure, mosaic pattern)	• Oral cavity: **Leukoplakia** • Duodenum: **Crohn Disease, Celiac sprue**

BIG QUESTION 7: Discuss Peptic Ulcer Disease (PUD), including its causes and clinical presentation.

Broad Answer: Peptic Ulcer Disease (PUD) is most commonly caused by H. pylori and is a leading cause of both perforation and peritonitis, and upper gastrointestinal bleeding (hematemesis and melena). Diagnosis of PUD can be aided by an X-ray showing gas under the diaphragm in the case of perforation.

DETAILED QUESTIONS

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Q7.1: What is the leading cause of peptic ulcer disease, and what is its most common complication?

PYQ: FMGE 2022, 2022

Answer:



- Leading cause of PUD: H. pylori
- Leading cause of perforation peritonitis: PUD
- Leading cause of hematemesis and melena: PUD
- MC gastric ulcer: Type 1
- X-ray: Gas under diaphragm
- IOC for melena: Upper GI endoscopy

Q7.4: How are gastric ulcers classified according to the Johnson Classification?

Answer:

Type	Location	Acid Hypersecretion	Complications	Incidence
I	Gastric body, Lower curvature	No	Bleeding Uncommon	55%
II	Body of stomach + duodenal ulcer	Yes	Bleeding perforation, Obstruction	20%
III	Prepyloric	Yes	Bleeding perforation	20%
IV	High on lesser curvature	No	Bleeding	<5%
V	Anywhere (Medication induced)	No	Bleeding perforation	<5%

BIG QUESTION 8: Discuss Inflammatory Bowel Disease (IBD), differentiating between Crohn's disease and Ulcerative Colitis.

Broad Answer:

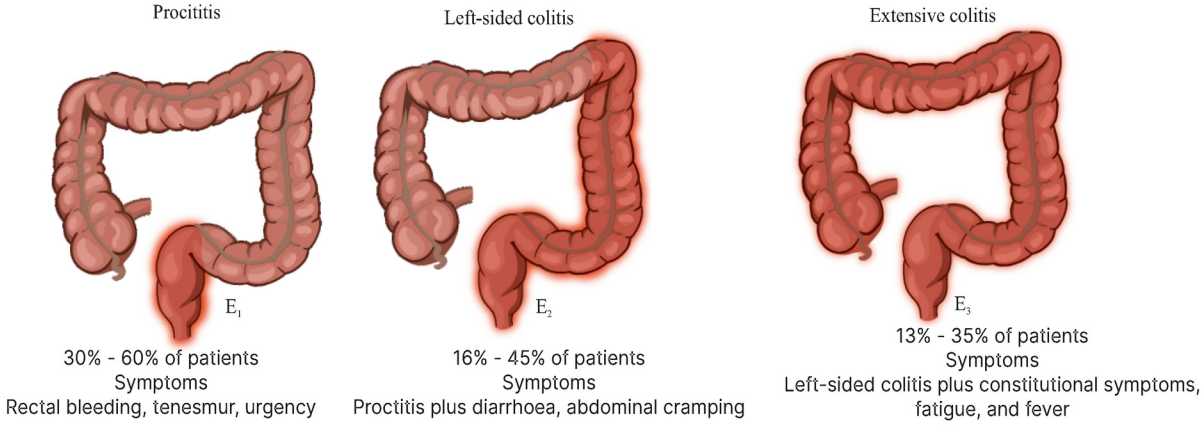
	Crohn's disease	Ulcerative colitis
Common site	Terminal ileum	Rectum
Distribution	Mouth to anus	Rectum
Spread	Discontinuous	Continuous
Gross feature	Focal aphthous ulcer with intervening normal mucosa, linear fissures, cobblestone appearance, thickened bowel wall, creeping fat.	Extensive ulceration pseudopolyps.
Microscopy	Non-caseating granulomas	Crypt abscesses
Inflammation	Transmural	Limited to mucosa and submucosa
Complications	Strictures, string sign on barium studies, abscesses, sinus tract, obstruction, fistulas	Toxic megacolon
Extraintestinal manifestations	Uncommon	Arthritis, spondylitis, primary sclerosing cholangitis, erythema nodosum, pyoderma gangrenosum
Cancer risk	1-3%	5-25%
Barium meal follow through	Hose Pipe colon, String sign of Kantor	Lead pipe Colon

DETAILED QUESTION

Q8.1: How is Ulcerative Colitis classified by its extent?

Answer:

- The Montreal classification for the extent of UC is:
  - o E1: Proctitis (inflammation limited to the rectum)
  - o E2: Left-sided colitis (inflammation extending from the rectum up to the splenic flexure)
  - o E3: Pan colitis (inflammation extending beyond the splenic flexure, including the entire colon)



Q8.2: How is the severity of Ulcerative Colitis classified?

Answer:

Montreal Classification of severity of ulcerative colitis (UC)		
Severity		**Definition**
S0	Clinical remission	Asymptomatic
S1	Mild UC	Passage of four or fewer stools/day (with or without blood), absence of any systemic illness, and normal inflammatory markers (ESR)
S2	Moderate UC	Passage of more than four stools per day but with minimal signs of systemic toxicity
S3	Severe UC	Passage of at least six bloody stools daily, pulse rate of at least 90 beats per minute, temperature of at least 37.5^{0}C, hemoglobin of less than 10.5g/100 ml, and ESR of at least 30 mm/h

Q8.3: What are the key differences in the clinical features of Crohn's disease and Ulcerative Colitis?

Answer:

Feature		Crohn's disease	Ulcerative colitis
Symptoms	Fever	Some people may feel generally unwell, feverish, with raised temperature	
		Suggests severe disease with systemic toxicity	Temperature >37.8^{0}C indicative of severe UC
	Diarrhea	Sometimes mixed with mucus, pus, or blood	Often with blood and mucus, and an urgent need to rush to the toilet
	Abdominal pain	Common	Cramping pain, often before passing a stool

	Abdominal mass	Common	Absent
	Tachycardia	Suggests severe disease with systemic toxicity	Pulse rate > 90 bpm suggests severe UC
	Other	Hair loss (owing to nutritional deficiencies e.g. B12 and iron), mouth ulcers (50% CD), fatigue, anemia, weight loss, loss of appetite, and growth impairment in children and young people	

Q8.4: How do the microscopic findings of Crohn's disease and Ulcerative Colitis differ?

Answer:

- Crohn's disease: Shows transmural inflammation and non-caseating granulomas
- Ulcerative Colitis: Inflammation is limited to the mucosa and submucosa, and microscopy shows crypt abscesses
- Toxic megacolon: Diameter of colon is > 6 cm, seen in UC

Q8.5: What are the complications of Crohn's disease and Ulcerative Colitis?

Answer:

	Crohn's disease	Ulcerative colitis
Fistulae	Often perianal, occurs in around 25-33% of cases	Rarely, more likely in those who have had pouch surgery
Strictures	As a result of scar tissue or inflammation	Unusual in UC, sometimes a sign of bowel cancer
Fissure	Complication of perianal disease	Absent

Q8.6: What are the drugs of choice for managing Crohn's disease and Ulcerative Colitis?

Answer: PYQ: NEET PG 2022, 2023 PYQ: INICET 2022, 2023 PYQ: FMGE 2020, 2021

- DOC Crohn's disease: Steroids
- DOCUlcerative Colitis: Sulfasalazine
  - In case of exacerbation of Ulcerative Colitis, budesonide enema is used

Q8.7: What are the extraintestinal manifestations of IBD?

Answer:

- The manifestations can affect various systems:
  - Metabolic bone disorders (40%): Osteoporosis, Osteonecrosis
  - Hepatobiliary (10-35%): Fatty liver, Cholelithiasis, PSC
  - Rheumatological (5-20%): Peripheral arthritis, Sacroiliitis, Ankylosing spondylitis
  - Urologic: Nephrolithiasis, especially in patients with CD, following small bowel resection, there is a higher incidence of Ca Oxalate stone formation
  - Dermatologic: Erythema nodosum, Pyoderma gangrenosum (ulcerative lesion on shin)
  - Ocular: Uveitis, Episcleritis

BIG QUESTION 9: Discuss the causes and diagnostic methods for malabsorption syndrome.

Broad Answer: Causes of malabsorption syndrome: PYQ: NEET PG 2021 PYQ: FMGE 2021

Celiac Sprue	• Cereals which are not given: barley, rye, oats, and wheat • Cereals which can be given maize, quinoa • Celiac sprue is characterized by autoantibodies which are damaging the microvilli of the intestine. This leads to decrease in surface area of intestine, as a result sugar will not be absorbed, this contribute to osmotic diarrhea • Antibody causing damage is **Anti TTG** • Anti ETG: Cause of dermatitis herpetiformis • IOC: **Small intestinal mucosal biopsy** • Leading cause of death: Malignancy (lymphoma)
Whipple disease	• IOC: **Small intestinal mucosal biopsy** • Biopsy will show PAS positive inclusions. • These PAS +ve inclusions are Tropheryma whipplei (Intracellular bacteria)
SIBO	• Large intestinal bacteria are migrating via incompetent ileocecal valve into small bowel • IOC: Jejunal aspirate + Culture • SIBO is confirmed if culture count is > 10^{11} • Rx: Metronidazole • Vit B12 deficiency will be more common

DETAILED QUESTIONS

Q9.1: What is the results of diagnostic studies in different causes of steatorrhea?

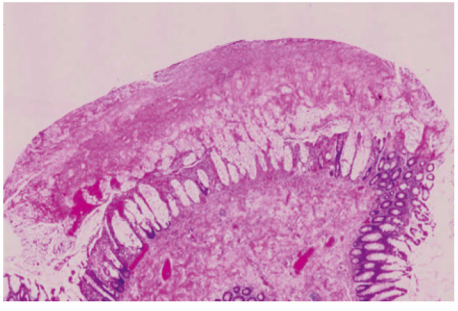
Answer:

	D-Xylose Test	Schilling Test	Duodenal Mucosal biopsy
Chronic pancreatitis	Normal	50% abnormal; if abnormal, normal with pancreatic enzymes	Normal
Bacterial overgrowth syndrome	Normal or only moderately abnormal	Often abnormal; if abnormal, normal after antibiotics	Usually normal
Ileal Disease	Normal	Abnormal	Normal
Celiac Sprue	Decreased	Normal	Abnormal: Probably flat
Intestinal lymphangiectasia	Normal	Normal	Abnormal: Dilated

Q9.2: What is the primary cause of Pseudomembranous Colitis, and what are the most common antibiotic triggers?

Answer:

- PMC is caused by the overgrowth of *Clostridium difficile*, which releases a strong toxin that causes inflammation and bleeding
- MC antibiotic triggers: Cephalosporins > Clindamycin



Q9.3: What are the diagnostic tests for PMC?

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Answer:

- Stool culture: Most sensitive test, but the turnaround time is too slow for practical use
- NAAT for C. difficile toxin A or B gene in stool: Best way for identification after stool culture. NAAT is the test for diagnosis in the early phase of the disease
- EIA for toxins A and B in stool
- EIA for C. difficile common antigen in stool

Q9.4: Describe the recommended treatment for C. difficile infection based on severity.

Answer: PYQ: FMGE 2022

Recommendations for the Treatment of Clostridium difficile Infection	
Clinical Setting	**Treatment (S)**
Initial episode, mild to moderate	Fidaxomicin (200 mg bid × 10 d) or Oral Vancomycin (125 mg qid × 10 d)
Initial episode, severe	Oral Vancomycin (125 mg qid × 10 d) or Fidaxomicin (200 mg bid × 10 d)
Initial episode, Fulminant	Vancomycin (500 mg PO or via nasogastric tube) plus metronidazole (500 mg IV q8h) plus consider Rectal instillation of vancomycin (500 mg in 100 mL of normal saline as retention enema q6 - 8h)
First recurrence	Fidaxomicin (200 mg bid × 10 d) or Oral Vancomycin (125 mg qid × 10 d) or Oral Vancomycin followed by taper- and- pulse regimen
Multiple recurrences	Oral vancomycin treatment followed by a taper-and- pulse regimen or Fidaxomicin (200 mg bid × 10 d or 200 bid × 5 f followed by every other day × 20 d) or Vancomycin (125 mg qid × 10 d), then stop vancomycin and start rifaximin (400 mg bid × 2 weeks) or Fecal microbiota transplantation (FMT)
Patients at high risk of recurrent CDI who are receiving vancomycin, fidaxomicin, or metronidazole	Bexlotoxumab 10 mg/kg given IV

BIG QUESTION 10: Discuss the key complications of blood transfusion.

Broad Answer: The leading cause of death after a blood transfusion is Transfusion-Associated Circulatory Overload (TACO), followed by Transfusion-Related Acute Lung Injury (TRALI). The most common complication, however, is a Febrile Non-hemolytic Transfusion Reaction (FNHTR). TACO is a cardiogenic pulmonary edema resulting from volume overload, leading to elevated BNP and blood pressure. Conversely, TRALI causes non-cardiogenic pulmonary edema, where both BNP and blood pressure remain normal.

DETAILED QUESTIONS

Q10.1: What is the leading cause of death after a blood transfusion, and what is the most common complication overall?

Answer:

- The leading cause of death is TACO (Transfusion-Associated Circulatory Overload), followed by TRALI (Transfusion-related Acute Lung Injury).
- The most common complication is a Febrile Non-hemolytic Transfusion Reaction (FNHTR)

Q10.2: How can TACO be differentiated from TRALI?

Answer:

- TACO is caused by cardiogenic pulmonary edema due to volume overload. This leads to an elevated BNP and increased blood pressure.
- TRALI, on the other hand, is a non-cardiogenic pulmonary edema, so BNP values and blood pressure remain normal.

Q10.3: What is the role of citrate in a blood transfusion, and what are its potential side effects?

Answer:

- Citrate is added to blood products to prevent coagulation. Excess citrate can cause two main issues:
  - Metabolic alkalosis: The liver metabolizes citrate into bicarbonate, which causes an increase in blood pH.
  - Hypocalcemia: Citrate binds to ionized calcium, causing its levels to fall, which can trigger carpopedal spasms and tetany

Q10.4: How can a blood transfusion cause both hyperkalemia and hypokalemia?

Answer:

- Hyperkalemia can occur when blood is transfused near its expiry date. The red blood cells' sodium-potassium pumps fail, causing potassium to leak out of the cells.
- Hypokalemia can be caused by the metabolic alkalosis from citrate. As pH is inversely proportional to potassium levels, the high pH drives potassium into the cells, leading to a decrease in serum potassium.

BIG QUESTION 11: Describe the types of hematological malignancies.

Broad Answer:

- Chronic Leukemias
  - Chronic Lymphocytic Leukemia (CLL) is the most common leukemia in adults
  - Chronic Myeloid Leukemia (CML) is caused by the t(9;22) translocation (Philadelphia chromosome), which creates the BCR-ABL gene..
- Acute Leukemias are either AML or ALL.
  - AML is more common in adults and presents with granulocytic sarcoma and Auer rods, while ALL is more common in children, presenting with CNS leukemia and sternal tenderness.

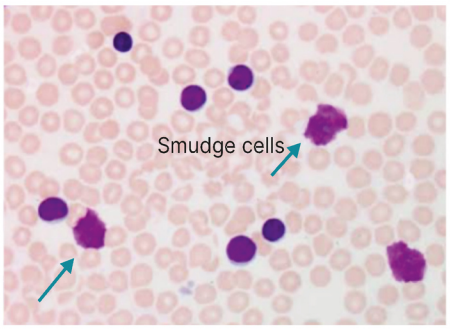
DETAILED QUESTIONS

Q11.1: What is the most common leukemia in adults, and what are its key clinical and laboratory findings?

Answer: PYQ: NEET PG 2021, 2023 PYQ: INICET 2021

- Chronic Lymphocytic Leukemia (CLL) is the most common leukemia in the adult population
- Age group > 70 years
- Clinical features
  - Fatigue, involuntary weight loss, night sweats
  - Progressive Cervical Lymphadenopathy

- Infections: Recurrent pneumonia, Herpes zoster: Grouped vesicles in dermatomal distribution:
- Herpes zoster: Grouped vesicles in genitals: Herpes simplex
- Anemia: Autoimmune hemolytic anemia
- Hepatosplenomegaly



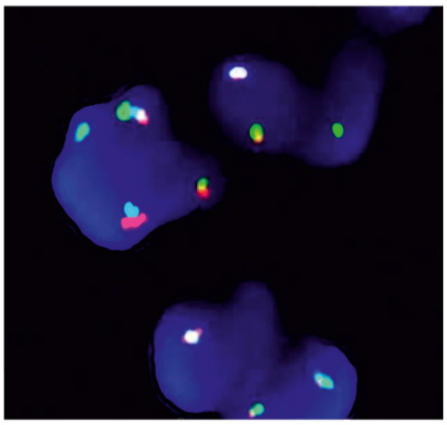
- Work up
  - Peripheral smear shows smudge cells
  - CBC shows a Decrease in Hb and Platelets, TLC ↑
  - IOC: flow cytometry (CD19, CD20, CD23, CD5+ve)

Q11.2: What is the genetic basis of Chronic Myeloid Leukemia (CML), and what are its key clinical diagnostic features?

PYQ: INICET 2022

Answer:

- Pathogenesis
  - t(9;22) translocation → BCR-ABL fusion gene →↑ tyrosine kinase activity → uncontrolled myeloid proliferation.
  - No maturational arrest → all precursor stages present in blood (*shift to left*).



- Epidemiology
  - Age: 55-65 years (peak incidence).
- Clinical Features
- Constitutional: Weight loss (>5% in 6 months), fatigue, night sweats.
- GI-related: Early satiety (splenomegaly), constipation, diarrhea, flushing, pruritus. Massive splenomegaly (hallmark).
- Thrombotic events: Priapism, leukostasis, MI, CVA, respiratory distress, blindness.
- Late stage (blast crisis): Thrombocytopenia.

Q11.3: Describe the laboratory findings and diagnosis of CML.

Answer:

- NAP score ↓ (defective neutrophil killing → recurrent infections).
- Vitamin B12 ↑ (due to ↑ transcobalamin production)

Investigation	Finding
Peripheral smear	Basophilia, marked leukocytosis, shift to left (myelocytes, metamyelocytes, promyelocytes)
Bone marrow aspirate	Hypercellular, M:E ratio 15-20:1 (normal 3:1), ± reticulin fibrosis (Snook silver stain)
NAP score	Decreased
B12 levels	Elevated
Gold standard (IOC)	FISH - quantifies Ph+ cells
PCR for BCR-ABL	Used for monitoring; not IOC (false ± possible)

Q11.4: What are the treatment strategies for CLL and CML?

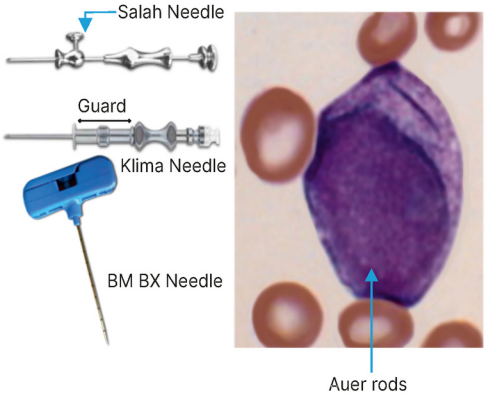
Answer:

Feature	CLL	CML
Typical Age Group	>60 years (elderly)	40-60 years (middle-aged)
Pathology	B-cell malignancy	Myeloproliferative neoplasm with BCR-ABL fusion (Philadelphia chromosome)
First-Line Treatment	• <65 yrs, no comorbidity: FCR regimen: • F - Fludarabine (DOC) • C - Cyclophosphamide • R - Rituximab • >65 yrs/comorbidity: Chlorambucil + Obinutuzumab	TKI therapy: • Imatinib • Dasatinib • Nilotinib • Bosutinib (*for imatinib-resistant CML*)
Additional Drugs	• Venetoclax - BCL2 inhibitor → pro-apoptotic protein release • Ibrutinib - BTK inhibitor	Ponatinib - only TKI effective against T315I mutation (resistant to Imatinib, Dasatinib, Nilotinib, Bosutinib)
Treatment Goal	Symptom control, delay progression	Molecular remission: <0.1% BCR-ABL transcripts (3-log reduction)
Mutation Notes	No specific signature mutation like CML	T315I mutation → resistant to most TKIs except Ponatinib

Q11.5: What are the clinical and diagnostic differences between Acute Myeloid Leukemia (AML) and Acute Lymphoblastic Leukemia (ALL)? PYQ: FMGE 2023

Answer:

- AML is more common in adults and presents with granulocytic sarcoma, gingival hyperplasia, and DIC (in t(15:17))
- Its hallmark is the presence of Auer rods.
- ALL is more common in children and presents with sternal tenderness, CNS leukemia, and testicular lumps.
- The definitive investigation for both is a bone marrow aspiration using a Salah or Klima needle.
  - o Thrombocytopenia is not a contraindication for bone marrow biopsy



Q11.6: What are the treatments for AML and ALL? What is a significant side effect of a drug used for AML?

Answer:

- The standard treatment for AML is Cytarabine and Daunorubicin. For the M3 variety (APML), All-trans retinoic acid (ATRA) is used, which can cause differentiation syndrome due to cells adhering to the lung endothelium, leading to ventilation-perfusion imbalance.
- This is treated with steroids.
- The treatment for ALL includes steroids and vincristine, along with other drugs.

Q11.7: Describe the characteristic skull X-ray findings for Multiple Myeloma, Thalassemia, and Paget's Disease.

Answer:

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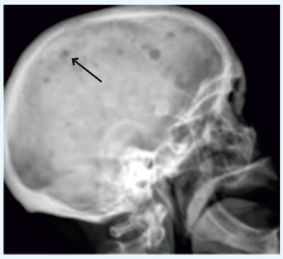
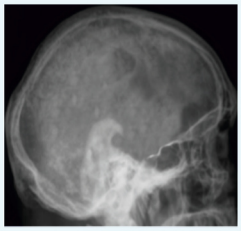
Multiple Myeloma PYQ: FMGE 2020 	• Image shows raindrop configuration and lytic lesions points towards Multiple myeloma • For diagnosis 1. Plasma count in the BM should be > 10% 2. M-Component in serum electrophoresis 3. CRAB: → Hypercalcemia, SAP normal → Renal failure → Anemia → Bony lytic lesions
Thalassemia PYQ: NEET PG 2022 	• Image shows Hair on end appearance • Diagnosis o b globin gene estimation: IOC o HPLC: High performance liquid Chromatography • TOC: Allogeneic stem cell transplantation • Earliest radiological finding: Squaring of metacarpals • Chipmunk facies, Anemia and splenomegaly are features of these patients
Pagets Disease 	• Multiple lytic lesions associated with extreme whiteness of skull (like Ivory bone) • Disproportionate rise is SAP is seen • Skull deformity, sensorineural deafness anemia, and heart failure can be features of these patients

- X-ray skull in a child, similar to Paget's lytic lesions: Geographical skull seen in Histiocytosis X