MEDICINE AIIMS 2018

1.

A 40-year-old male chronic smoker presents to the AIIMS OPD with fever, fatigue, yellow-colored urine, and clay-colored stools. For the past few days, he has developed an aversion to cigarette smoking. On examination icterus was present. What investigations would you advise to rule out acute viral hepatitis? Liver function test results are given below:

- Total Bilirubin - 18.5
- Direct Bilirubin - 7.5
- SGOT - 723
- SGPT 812

✓AntiHAV, HBsAg, IgM antiHBc, AntiHCV

•HBsAg, IgM antiHBC, AntiHCV, AntiHEV

•HBsAg, IgM antiHBc, AntiHDV, AntiHCV, Anti HEV

•AntiHAV, IgM antiHBc, AntiHCV, AntiHEV

The given clinical scenario is suggestive of Jaundice and tests to Rule Out Acute Viral Hepatitis:

1. Anti-HAV (Hepatitis A Virus Antibody)
2. IgM anti-HBc (IgM Antibody to Hepatitis B Core Antigen)
3. Anti-HCV (Hepatitis C Virus Antibody)

Suspected Hepatitis Conditions and Targeted Tests

Condition	Targeted Tests Performed
Acute Hepatitis B	HBsAg and IgM Anti-HBc
	(both can be positive)
	IgM Anti-HBc
	(positive with negative HBsAg)
Acute Hepatitis C	Anti-HCV
Chronic Hepatitis B	HBsAg
Chronic Hepatitis C	Anti-HCV
	HCV RNA (PCR)
Hepatitis A	Anti-HAV IgM
Hepatitis D	Anti-HDV
	HDV RNA (PCR)
Alcoholic Hepatitis	AST (SGOT)
	ALT (SGPT)
	GGT
	MCV
	AST/ALT ratio

Suspected Non-Hepatitis Conditions and Targeted Tests

Condition	Targeted Tests Performed
Non-Alcoholic Fatty Liver Disease (NAFLD)	Liver function tests (LFTs)
	Liver ultrasound
	Hepatic steatosis markers (e.g., Fatty Liver Index)
Cirrhosis	Liver function tests (LFTs)
	Liver biopsy
	Imaging studies (CT, MRI)
	FibroScan or other liver stiffness measurement
Liver Cancer (Hepatocellular Carcinoma)	Alpha-fetoprotein (AFP)
	Imaging studies (CT, MRI, ultrasound)
	Liver biopsy
Autoimmune Hepatitis	Liver function tests (LFTs)
	Anti-nuclear antibodies (ANA)
	Anti-smooth muscle antibodies (ASMA)
	Liver biopsy

Hepatitis D (HDV) Testing:

- Hepatitis D occurs only concurrently with Hepatitis B, either as a co-infection (indistinguishable from HBV infection) or superimposed on chronic HBV infection.
- ***Presentation:*** In the latter scenario, it causes a sudden worsening of the patient's condition.
- ***Diagnostic Tests:*** When hepatitis D is suspected, the following tests are useful for diagnosis:
    1. IgM anti-HDV (IgM Antibody to Hepatitis D Virus)
    2. IgG anti-HDV (IgG Antibody to Hepatitis D Virus)
    3. HDV RNA assays (Detection of Hepatitis D Virus RNA)

Hepatitis E (HEV) Testing:

- Hepatitis E is not routinely tested due to the spontaneous clearance of the virus in the majority of patients who acquire HEV infection.

- **High-Risk Groups:** Acute hepatic failure is more likely in pregnant individuals and those who are malnourished or have preexisting liver disease. Chronic HEV is mainly seen in immunocompromised hosts, such as organ transplant recipients.
- ***Diagnosis:*** The diagnosis of acute HEV is typically based on the detection of IgM antibodies to HEV.
- ***Reliability:*** Available assays for anti-HEV antibodies may yield both false positives and negatives, making them less reliable as screening tests.
- ***Confirmation:*** Additional serologic testing or HEV RNA testing should be performed to confirm the diagnosis.

2.

AKIN and RIFLE criteria are used to classify

✓Acute kidney injury

•Acute glomerulonephritis

•Chronic renal failure

•Nephrotic syndrome

AKIN and RIFLE criteria are used to classify **Acute kidney injury**

Classification of Acute Kidney Injury (AKI)

1. **RIFLE Criteria**

- **Risk:** Represents a stage where there is a risk of kidney dysfunction, indicated by an increase in serum creatinine or reduction in urine output.
- **Injury:** This stage signifies the development of mild kidney injury, characterized by a more significant increase in serum creatinine or reduction in urine output.
- **Failure:** In this stage, there is a further worsening of kidney function, denoted by a substantial increase in serum creatinine or severe reduction in urine output.
- **Loss:** This stage indicates a persistent loss of kidney function, lasting for a specific period.
- **End-stage:** At this critical stage, there is complete loss of kidney function, requiring renal replacement therapy or kidney transplantation.

2. **AKIN (AKI Network) Classification**

- **Stage 1:** In this initial stage, there is a slight increase in serum creatinine or reduction in urine output within 48 hours.
- **Stage 2:** The second stage denotes a moderate increase in serum creatinine occurring within 48 hours.
- **Stage 3:** At this severe stage, there is a substantial increase in serum creatinine or a significant reduction in urine output within 48 hours or the need for renal replacement therapy.

3. **KDIGO (Kidney Disease Improving Global Outcomes) Classification**

- **Stage 1:** Represents a mild AKI stage with an increase in serum creatinine by 1.5 to 1.9 times the baseline or a reduction in urine output <0.5 mL/kg/h for 6-12 hours.
- **Stage 2:** Denotes a moderate AKI stage with an increase in serum creatinine by 2.0 to 2.9 times the baseline or a reduction in urine output <0.5 mL/kg/h for ≥12 hours.
- **Stage 3:** This is the most severe AKI stage with an increase in serum creatinine ≥3.0 times the baseline or an absolute increase in serum creatinine to ≥4.0 mg/dL or a reduction in urine output <0.3 mL/kg/h for ≥24 hours or anuria for ≥12 hours.

3.

A 22-year-old man presented with diarrhea and intolerance to dairy products. On investigation, he was found to have a lactase deficiency. Which of the following agents is least likely to cause symptoms of lactose intolerance?

•Condensed Milk

•Skimmed Milk

✓Yogurt

•Ice Cream

Yogurt is **unlikely to cause symptoms of lactose intolerance because it contains lactobacillus**, which produces lactase. This enzyme helps in the digestion

of lactose, making yogurt generally well-tolerated by individuals with lactose intolerance.

Lactose Intolerance and Yogurt

- Yogurt is the Least Likely Cause of Symptoms
- Presence of Lactobacillus in Yogurt
- Lactobacillus Produces Lactase
- Generally Well Tolerated

Lactase: The Key Enzyme

- Lactase is a Brush Border Enzyme
- Function: Hydrolyzes Lactose into Glucose and Galactose

Clinical Symptoms in Lactase Deficiency

- Varied Clinical Symptoms
- Dependence on Severity of Lactase Deficiency
- Influence of Lactose Ingestion Amount

Treatment Goal for Isolated Lactase Deficiency

- Achieving Patient Comfort
- Elimination of Dietary Lactose

Lactose Content in Foods

- Foods High in Lactose:
    - Milk: 12g/cup
    - Ice Cream: 9g/cup
    - Cottage Cheese: 8g/cup
- Aged Cheese Contains Lower Lactose
- Tolerance to Lactose Ingestion:
    - Most People: Up to 12g/day
    - Without Significant Symptoms

4.

An 18-year-old girl with the diagnosis of acute promyelocytic leukemia was treated medically. She developed fever and tachypnea and a chest X-ray showed pulmonary infiltrates. What drug should she be given next?

✓Dexamethasone

•Cytarabine

•Doxorubicin

•Methotrexate

The clinical scenario describes a patient of APL (acute promyelocytic leukemia) developing fever, tachypnea, and pulmonary infiltrates while on medical treatment This is suggestive of APL differentiation syndrome, an adverse effect of all-trans-retinoic acid (ATRA). The management involves glucocorticoids like dexamethasone.

Management:

- Glucocorticoids (e.g., dexamethasone)
- Chemotherapy for cytoreduction
- Supportive measures

Severe Cases Management:

- Temporary discontinuation of ATRA may be necessary

ATRA Mechanism:

- Oral drug-inducing differentiation of leukemic cells bearing the t(15;17) chromosomal translocation
- Reduces the incidence of DIC associated with cytarabine and daunorubicin

Cause of APL Differentiation Syndrome:

- Adhesion of differentiated neoplastic cells to the pulmonary vasculature endothelium

Treatment Approach for Low-Risk APL Patients with Low Leukocyte Count:

- Combination of ATRA and arsenic trioxide (ATO)
- Found to be superior and current standard of care

5.

The most common bleeding manifestation seen in severe hemophilia is:

•Recurrent hematomas

✓Recurrent hemarthrosis

•Intracranial hemorrhage

•Hematuria

Recurrent hemarthrosis is the **most common bleeding manifestation of severe hemophilia**. Hemophilia is said to be severe when the residual activity of **factor VIII is**

- Hemophilia is an X-linked recessive hemorrhagic disorder caused by mutations in the F8 gene (hemophilia A) or the FS gene (hemophilia B).
- It is impossible to clinically distinguish between hemophilia A and hemophilia B as they present with similar symptoms.
- The disorder can be classified based on residual activity of FVIII or FIX as severe (<1%), moderate (1—5%), or mild (6—30%).

Clinical Presentation

- Hemophilia presents early in life, with bleeding episodes commonly occurring after circumcision or when the child begins to walk or crawl.

- **Acute hemarthrosis:**
  - Local pain, swelling, and erythema are observed.
  - Muscle contractures may develop due to fixed positioning.
- **Chronic hemarthrosis:**
  - Synovial thickening is observed.
  - Synovitis occurs due to the accumulation of blood within the joint.
- The knee joint is the most commonly affected joint, and one joint is typically more affected than others, referred to as the "target joint."
- Muscle hematomas can lead to compartment syndromes.
- Hematuria is common but self-limited.
- Life-threatening bleeds may occur in oropharyngeal spaces, the central nervous system (CNS), or the retroperitoneum.

Treatment

- Without treatment, **severe hemophilia may limit life expectancy.**
- The mainstay of **therapy for hemophilia** is factor replacement.
- **FVIII dose calculation:** FVIII dose (IU) = Target FVIII levels - FVIII baseline levels x body weight (kg) x 0.5 unit/kg.
- **FIX dose calculation:** FIX dose (IU) = Target FIX levels - FIX baseline levels x body weight (kg) x 1 unit/kg.
- The half-life of **FVIII is 8—12 hour**s, requiring twice-daily injections to maintain therapeutic levels.
- The **half-life of FIX is longer, approximately 24 hours**, making once-a-day injections sufficient for treatment.

6.

What is the most likely causative organism of ventilator-associated pneumonia among the following?

•Klebsiella

•Clostridium

•Hemophilus

✓Acinetobacter

Acinetobacter species is the most commonly isolated organism in cases of ventilator-associated pneumonia (VAP). VAP develops in a mechanically ventilated patient >48 hours after endotracheal intubation.

Ventilator-Associated Pneumonia (VAP)

Most Commonly Isolated Organism: Acinetobacter species

- VAP develops in a mechanically ventilated patient >48 hours after endotracheal intubation.

Organisms Frequently Causing VAP in Hospital Settings:

- Staphylococcus aureus
- Pseudomonas aeruginosa
- Acinetobacter sp.
- Stenotropnomonas maltophilia
- Gram-negative rods (Enterobacter species, Klebsiella pneumoniae, and Escherichia coli)

Differences Between Nosocomial Pneumonia and Community-Acquired Pneumonia:

- Different infectious agent
- Different antibiotic susceptibility patterns, specifically, a higher incidence of drug resistance
- Poorer underlying health status of patients putting them at risk for more severe infections

Colonization and Risk Factors:

- Within 48 hours of admission, 75% of seriously ill hospitalized patients have their upper airway colonized with organisms from the hospital environment.

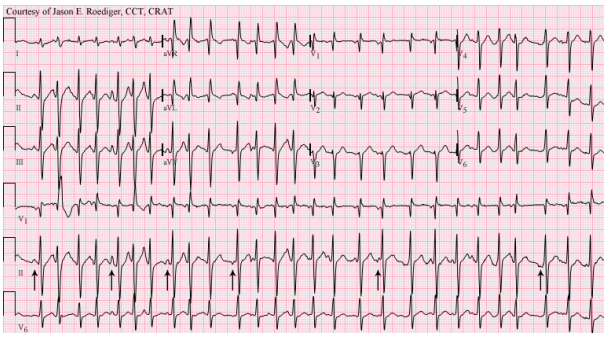
- Impaired cellular and mechanical defense mechanisms in the lungs of hospitalized patients raise the risk of infection after aspiration has occurred.

Other Potential Pathogens:

- VAP patients may also be infected with Stenotrophomonas maltophilia.
- Anaerobic organisms (Bacteroides, anaerobic streptococci, Fusobacterium) may also cause pneumonia in hospitalized patients.

7.

A 50-year-old man was brought to the emergency in an unconscious state. He had a fever for the past two days and is a known case of severe COPD. His ECG is given below. What is the most likely diagnosis?



✓Multifocal atrial tachycardia

•Atrial tachycardia

•Ventricular tachycardia

•Atrial fibrillation

The given Clinical scenario of **chronic pulmonary disease** and the ECG showing different p-wave morphologies are suggestive of multifocal atrial tachycardia

Multifocal Atrial Tachycardia (MAT)

- Impulses arise from multiple abnormal foci in the atria
- Leads to supraventricular tachycardia with p waves and QRS complexes of different morphologies
- Commonly encountered in patients with chronic pulmonary disease and acute illness

Diagnosis:

- ECG shows narrow QRS complexes with irregular RR intervals
- Differential diagnosis between atrial fibrillation and multifocal atrial tachycardia

Differentiating Factors:

- Multifocal Atrial Tachycardia (MAT)
  - Characterized by at least 3 distinct P-wave morphologies
  - P-waves represented by arrows in the image
  - Presence of identifiable P-waves with different shapes
- Atrial Fibrillation
  - No distinct P-waves observed
  - Absence of identifiable P-waves

Treatment Approach:

- Directed at treating the underlying disease
- Correcting any metabolic abnormalities

Additional Treatment Modalities:

- **Calcium-Channel Blockers**
  - Used as a therapeutic option for **MAT**
  - Helps in controlling heart rate and rhythm
- **Radiofrequency Ablation of the AV Node**
  - Considered as another treatment modality for MAT
  - Involves ablation of abnormal electrical pathways in the heart

8.

A patient with chronic kidney disease came with complaints of vomiting and diarrhea. His blood gas reports show pH = 7.40, HCO3-= 23 mEq/L, Na+ = 145 mEq/L, Cl-= 100 mEq/L. What is your probable diagnosis?

•No acid base abnormality

•Metabolic alkalosis

•Respiratory acidosis

✓High anion gap metabolic acidosis with metabolic alkalosis

The most probable diagnosis is High anion gap metabolic acidosis with metabolic alkalosis

Clinical Interpretation of ABG:

Patient's ABG results:

1. pH: 7.4 (within normal limits)
2. Plasma HCO3-: 23 mEq/L (within normal limits)

Step 1: Assessing Acid-Base Disorder

- pH within normal limits (7.4): Rules out simple acid-base disorder (options A and C eliminated).

Step 2: Calculating Anion Gap

- Anion gap = [Na+] - ([HCO3-])
- [Na+] (Sodium): 145 mEq/L
- [HCO3-] (Bicarbonate): 100 + 23 = 123 mEq/L
- Anion gap = 145 - 123 = 22 mEq/L

Step 3: Identifying Acid-Base Imbalance

- High anion gap (22 mEq/L) suggests the presence of an acid-base disorder, ruling out option A.

- High anion gap indicates metabolic acidosis. However, normal HCO3- levels suggest coexisting metabolic alkalosis.

Calculating Delta Gap:

- Delta gap = Anion Gap - Change in HCO3-
- Anion Gap (AAG): 22 mEq/L
- Change in HCO3- (AHCOS): 24 - 23 = 1 mEq/L
- Delta gap = (22 - 1) - (24 - 23) = 11 mEq/L

Delta gap > +6, indicating the presence of metabolic alkalosis along with high anion gap metabolic acidosis (HAGMA).

Calculating Delta Ratio:

- Delta ratio = Increase in Anion Gap / Decrease in HCO3-
- Delta ratio = 12 / 1 = 12

Delta ratio > 2, confirming HAGMA with coexisting metabolic alkalosis.

9.

A patient presents with left-sided facial paralysis and left-sided limb weakness for the past 1 hour. Her blood pressure is 160/100 mm Hg and her CT appears normal. What would be the next step of management?

•Nothing, since CT was normal

✓Intravenous thrombolysis

•Start on aspirin + clopidogrel

•Advice BP control

The patient is experiencing **sudden onset hemiparesis**, which is a weakness on one side of the body, and the CT scan appears normal. This combination of symptoms and **imaging findings suggests an ischemic stroke.**

In such cases, it is crucial to **promptly administer intravenous thrombolysis with alteplase,**

a medication that helps dissolve blood clots.

This **treatment should be given immediately to patients who arrive within the window period, which is within 3-4.5 hours of the onset of stroke symptoms**. Early administration of alteplase can significantly improve the chances of a positive outcome for the patient.

Clinical use of Aspirin + Clopidogrel in Secondary Prevention of Stroke and Prevention following TIA

Transient Ischemic Attack (TIA)

- Brief episodes of stroke symptoms last less than 24 hours, with most lasting less than 1 hour.
- Can result from emboli to the brain or in situ thrombosis of an intracranial vessel.
- Occluded blood vessel reopens, restoring neurologic function.
- If a brain infarction is identified on imaging, it is classified as a stroke regardless of symptom duration.

Risk of Stroke after TIA

- Approximately 10-15% risk in the first three months, with the highest risk in the first two days, **ABCD2 Score is** used to reliably estimate the risk of stroke following a TIA.

Clinical Indications for **Aspirin + Clopidogrel**

- **Secondary Prevention of Stroke;** Indicated for patients with a history of stroke to prevent recurrent strokes.
- **Prevention following TIA:** Used after a TIA to reduce the risk of subsequent stroke.

CT Imaging

- Helpful for ruling out hemorrhagic stroke.
- Ischemic stroke changes may take time to appear on non-contrast CT (NCCT).

Blood Pressure Control

- Initiated when blood pressure exceeds 220/120 mmHg.

- Also, required in cases of malignant hypertension or concomitant myocardial ischemia.
- Blood pressure control is necessary if BP is >185/110 mmHg and thrombolytic therapy is anticipated.

10.

The arterial blood gas findings of a patient are pH = 7.12, pCO2 = 50, HCO3 = 28. What is the diagnosis?

✓Respiratory acidosis with metabolic compensation

•Respiratory alkalosis with metabolic compensation

•Metabolic acidosis with respiratory compensation

•Metabolic alkalosis with respiratory compensation

The above findings give the diagnosis of respiratory acidosis with metabolic compensation

Interpretation of the ABG:

Step 1: pH is reduced (7.12), hence acidosis.
Step 2: Since pC02 is raised (50 mmHg), it is respiratory acidosis.
Step 3: HC03 is raised (28 mEq/L) suggesting there is metabolic compensation.

Component	Normal Range	Optimal Value
pH (Arterial blood)	7.35 - 7.45	7.40
pCO2 (Partial pressure of carbon dioxide)	35 - 45 mmHg	40 mmHg
HCO3- (Bicarbonate)	22 - 28 mmol/L	24 mmol/L
pO2 (Partial pressure of oxygen)	80 - 100 mmHg	90 mmHg
SaO2 (Arterial oxygen saturation)	95 - 100%	98%
Base Excess (BE)	-2 to +2 mmol/L	0 mmol/L
Anion Gap	8 - 16 mmol/L	12 mmol/L
Lactate	0.5 - 1.6 mmol/L	<1.0 mmol/L

11.

Which of the following is not done before drawing blood for arterial blood gas analysis?

•Rinse syringe with heparin

•Allen's test

✓Flexion of wrist

•Placing needle at 45 degree angle

Flexion of wrist is not done before drawing blood for arterial blood gas analysis

The ideal position for drawing arterial blood from the radial artery is to maintain the wrist in extension. This stabilizes the artery and prevents it from slipping away during the puncture. The skin is pierced at a 45-degree angle to draw blood for ABG analysis.

12.

Relative bradycardia occurs in:

•Leptospirosis

•Q fever

•Typhoid fever

✓All of the above

Relative bradycardia occurs in **Leptospirosis, Q fever and Typhoid fever**

Relative bradycardia, also known as **Faget's sign, is a clinical finding observed in patients who exhibit a lower heart rate** than expected in relation to an increase in body temperature. This phenomenon is commonly seen in various **infectious and non-infectious conditions.**

Infectious Causes:

1. **Salmonella typhi:** Known for causing typhoid fever, this bacterium can lead to relative bradycardia as the body temperature rises.

2. **Francisella tularensis:** This bacterium is responsible for tularemia, a zoonotic infection that can be associated with relative bradycardia.

3. **Coxiella burnetii (Q fever):** Q fever, caused by Coxiella burnetii, is another infectious agent that may lead to relative bradycardia.

4. **Leptospira interrogans:** Leptospirosis, caused by Leptospira interrogans, can be associated with this pulse-temperature dissociation.

5. **Legionella pneumophila:** Legionnaires' disease, caused by Legionella pneumophila, is also among the infectious causes of relative bradycardia.

6. **Rickettsia spp.:** Various Rickettsia species can cause diseases like Rocky Mountain spotted fever, potentially leading to relative bradycardia.

7. **Orientia tsutsugamushi (scrub typhus):** Scrub typhus, caused by Orientia tsutsugamushi, is associated with this clinical finding.

8. **Corynebacterium diphtheriae:** Diphtheria, caused by Corynebacterium diphtheriae, can cause relative bradycardia.

9. **Plasmodium spp. (malaria):** Certain species of Plasmodium, which cause malaria, can lead to a decrease in heart rate relative to an increase in body temperature.

Non-infectious Causes:

1. **Drug Fever:** Certain medications can cause fevers, and when these drugs affect heart rate, relative bradycardia can be observed.

2. **Beta-blocker Use:** Beta-blockers, commonly used to manage various conditions, can also lead to a decreased heart rate in response to increased body temperature.

3. **Central Nervous System Lesions:** Conditions affecting the central nervous system can disrupt the autonomic control of heart rate, resulting in relative bradycardia.

4. **Malignant Lymphoma:** Malignant lymphomas and other neoplastic conditions can occasionally present with relative bradycardia.

5. **Factitious Fever:** Factitious fever, where patients intentionally fake symptoms of fever, can also lead to relative bradycardia as part of the overall clinical presentation.

Clinical Utility: While relative bradycardia is not highly sensitive or specific for establishing a definitive diagnosis, it can be a helpful clinical sign, especially in resource-limited settings.

13.

Which of the following drugs is not used in rheumatoid arthritis?

•Leflunomide

•Etanercept

•Methotrexate

✓Febuxostat

Febuxostat is not used in rheumatoid arthritis

Drugs used in Rheumatoid Arthritis

1) NSAIDs - Nonsteroidal Anti-Inflammatory Drugs

- NSAIDs are used to relieve pain and reduce inflammation in various conditions.
- Clinical Use: They are employed as adjunctive therapy for managing uncontrollable symptoms and acute flares.
- Examples: Common NSAIDs include ibuprofen, naproxen, and diclofenac.

2) Glucocorticoids

- Glucocorticoids are potent anti-inflammatory drugs.
- Clinical Use: They are used for rapid control of acute flares and as initial therapy before the onset of action of DMARDs (Disease-Modifying Anti-Rheumatic Drugs). In some cases, chronic low-dose administration is necessary for patients with inadequate response to DMARDs.
- Prednisolone: Prednisolone is administered orally.
- Triamcinolone: Triamcinolone is used for intraarticular injections.

3) DMARDs - Disease-Modifying Anti-Rheumatic Drugs

DMARDs are used to slow down the progression of rheumatoid arthritis and modify the disease course.

Conventional DMARDs:

- Methotrexate (MTX): An antifolate drug, considered the first-line DMARD of choice and the anchor drug for combination therapy. Regular liver function test (LFT) monitoring is necessary.
- Hydroxychloroquine: Used as monotherapy for early and mild disease, and in combination with other DMARDs for severe cases.
- Sulfasalazine: A salicylate, it reduces the radiographic progression of the disease.
- Leflunomide: A pyrimidine synthase inhibitor used as an alternative to MTX.

Biologicals:

- TNF-a Antagonists: Adalimumab, golimumab, etanercept, infliximab, and certolizumab are TNF-alpha inhibitors used in combination with MTX. They carry a risk of serious bacterial infections, opportunistic fungal infection, and reactivation of latent TB.
- Abatacept: A T-cell costimulation inhibitor, used in combination with MTX, leflunomide, etc.
- Rituximab: An anti-CD20 antibody, used in refractory disease in combination with MTX.
- Anakinra: An IL-1 receptor antagonist, not to be combined with anti-TNF-alpha drugs.
- IL-6 Receptor Antagonists: Tocilizumab and sarilumab used as monotherapy or in combination therapy for moderate to severe disease.

Small Molecules:

- Janus Kinase Inhibitors: Tofacitinib, baricitinib, and upadacitinib used as monotherapy or in combination therapy.

14.

Anti-nuclear antibodies are required for the diagnosis of which of the following?

•Scleroderma

•Rheumatoid arthritis

✓Systemic lupus erythematosus

•Sjogren's syndrome

Anti-nuclear antibodies are required for the diagnosis of Systemic lupus erythematosus

2019 EULAR/ACR Classification Criteria for Systemic Lupus Erythematosus (SLE)

1. Obligatory Entry Criterion: Positive Antinuclear Antibody (ANA) Test

- ANAs are autoantibodies that bind to the contents of the cell nucleus.
- They are present in > 95% of patients with SLE.
- ANA test titer should be at least 1 for this criterion.

2. Additive Criteria in Clinical Domains The presence of at least one of the following criteria is required for classification as SLE:

Musculoskeletal Involvement

- Arthritis: Non-erosive arthritis involving two or more peripheral joints.
- Myalgia: Muscle pain or tenderness not related to physical activity.

Mucocutaneous Involvement

- Malar Rash: Fixed erythema, flat or raised, over the malar eminences, sparing the nasolabial folds.
- Discoid Rash: Erythematous raised patches with adherent keratotic scaling and follicular plugging.
- Photosensitivity: Skin rash as a result of unusual reaction to sunlight.

Renal Involvement

- Proteinuria: Urine protein-to-creatinine ratio (or 24-hour urine collection) ≥ 0.5 g/day or ≥ 3+ on urine dipstick testing.
- Cellular Casts: Presence of red blood cell casts or hematuria casts in the urine.

Neurologic Involvement

- Seizures: Unprovoked seizures or seizures not explained by a known medical condition.
- Psychosis: Delusions, hallucinations, or thought disorders.

Hematologic Involvement

- Hemolytic Anemia: Abnormal blood tests indicative of hemolysis (e.g., low haptoglobin, elevated lactate dehydrogenase, and/or elevated indirect bilirubin).
- Leukopenia: White blood cell count < 4,000/mm³ on at least one occasion.
- Lymphopenia: Absolute lymphocyte count < 1,000/mm³ on at least one occasion.
- Thrombocytopenia: Platelet count < 100,000/mm³ in the absence of drugs or offending agents known to cause thrombocytopenia.

3. Additive Criteria in Immunologic Domains The presence of at least one of the following criteria is required for classification as SLE:

ANA Subtypes

- Anti-dsDNA: Specific to SLE, but less sensitive than ANA.
- Anti-Sm (Smith): Specific to SLE and highly specific for the disease.
- Anti-phospholipid Antibodies: Presence of lupus anticoagulant and/or anticardiolipin antibodies.

Complement Proteins

- Low Complement: Decreased levels of C3, C4, or CH50 in the blood.

Other Immunologic Criteria

- Direct Coombs Test: Positive result in the absence of hemolytic anemia.
- Antinuclear Antibodies (ANA) by Immunofluorescence: A positive result with a titer of at least 1:80 in the absence of drugs known to induce ANAs.

15.

A middle-aged woman with a history of constipation, dry skin and menorrhagia presents to the ER with altered sensorium, non-pitting edema, hypothermia, bradycardia, and hypotension. What is the most likely diagnosis?

•Cardiogenic shock

✓Myxedema coma

•Hypoglycemia

•Septic shock

The given clinical scenario is suggestive of a case of hypothyroidism and the diagnosis is **myxedema coma, which is a serious complication of hypothyroidism**

Myxedema coma can lead to a **myxedema crisis**. Myxedema crisis refers to severe, life-threatening manifestations of hypothyroidism. It is often precipitated by the administration of sedatives, antidepressants, hypnotics, anesthetics, or opioids.

Clinical Presentation of Myxedema Crisis:

1. Impaired Cognition:
   - Confusion
   - Somnolence
   - Coma (myxedema coma)
   - Convulsions and abnormal CNS signs
2. Profound Hypothermia:
   - Temperature can reach 23°C (73.4°F)
3. Respiratory Symptoms:
   - Hypoventilation
4. Metabolic Abnormalities:
   - Hyponatremia
   - Hypoglycemia
   - Hypoxemia
   - Hypercapnia
   - Hypotension
5. Muscle and Kidney Complications:
   - Rhabdomyolysis

- Acute Kidney Injury

6. High Mortality Rate:

Option A: Cardiogenic shock presents with symptoms of left heart failure, such as pitting pedal edema, abdominal distension, prominence of neck veins, and dyspnea.

Option B: Hypoglycemia usually presents with sweating, palpitations, and tachycardia.

Option D: Septic shock is unlikely when there is no history of or focus of infection.

16.

Which of the following is the most specific marker for alcoholism?

•ALT

•ALP

✓GGT

•LDH

GGT (Gamma Glutamyl Transferase) is the most specific marker of alcoholism

Clinical Markers for Heavy Alcohol Use

1. Gamma-Glutamyl Transferase (GGT)

- Sensitivity: >60%
- Specificity: >60%
- Cutoff Value: >35 u
- **Explanation:** GGT is a reliable marker for heavy alcohol use. Elevated GGT levels (>35 u) in the serum indicate possible alcohol abuse or alcoholic liver disease. When isolated GGT elevation or GGT elevation out of proportion to other liver enzymes (such as ALT and ALP) is observed, it further supports the indication of alcohol-related conditions.

2. Carbohydrate-Deficient Transferrin (COT)

- Sensitivity: >60%
- Specificity: >60%
- **Explanation:** COT is another useful marker for heavy alcohol consumption. It is particularly effective when combined with GGT to increase accuracy in detecting alcohol abuse. Elevated levels of COT indicate a higher probability of heavy alcohol use.

3. Combination of GGT and COT

- Explanation: When both GGT and COT are used together, their combined sensitivity and specificity enhance the accuracy of diagnosing heavy alcohol use. The simultaneous elevation of GGT and COT levels strengthens the suspicion of alcohol abuse or alcoholic liver disease.

Clinical Indicators of Alcohol-Related Liver Disease

1. Alanine Aminotransferase (ALT)

- Explanation: ALT is a specific marker of liver injury and is predominantly found in the liver. However, in alcoholic liver disease, ALT levels may often appear normal. Therefore, while elevated ALT levels usually indicate acute hepatocellular conditions like hepatitis, in the context of suspected alcoholic liver disease, normal ALT levels do not rule out the possibility of liver damage due to alcohol abuse.

2. AST/ALT Ratio

- Explanation: An AST/ALT ratio greater than 2.1 indicates the possibility of alcoholic liver disease. A higher ratio is highly indicative of this condition. Monitoring the AST/ALT ratio can provide valuable insights into the likelihood of alcohol-related liver damage.

Markers Not Specific for Alcohol-Related Conditions

1. Alkaline Phosphatase (ALP)

- Explanation: ALP is elevated in cholestasis and during periods of rapid bone growth, such as in children and adolescents. Additionally, normal pregnancies can also lead to increased ALP levels due to placental alkaline phosphatase activity. However, ALP elevation is not specific to heavy alcohol use or alcoholic

liver disease.

2. Lactate Dehydrogenase (LDH)

- Explanation: LDH elevation is a non-specific marker and does not specifically indicate heavy alcohol use or alcoholic liver disease. Elevated LDH levels may be observed in various conditions, and it does not provide targeted information for diagnosing alcohol-related issues.

17.

An elderly diabetic and hypertensive patient was carried to the emergency room in a comatose state. On examination, the blood pressure was 170/100 mmHg and pulse rate was >100/min. Plantar reflex was bilateral extensor. What is the next step to do?

•Give antihypertensive

✓Check blood sugar level

•CT brain

•IV mannitol

The **initial step in evaluating this comatose patient** involves excluding extreme glycemic conditions since most cases of coma are metabolic in nature and can be fully resolved with the right treatment.

Given that the **patient is elderly and lacks any other significant medical history**, the most probable causes of the coma include

diabetic ketoacidosis, hyperglycemic hyperosmolar state, or possibly hypoglycemia, which might present with hypertension. Identifying and addressing these metabolic issues promptly can lead to a complete reversal of the coma.

Patient Presentation:

- Tachycardia is present, not a sign of raised intracranial pressure
- No bradycardia, altered respiratory pattern, or raised systolic blood pressure

Initial Steps:

1. **Exclude Intracranial Space-Occupying Lesion**: CT scan not required at this point.
2. **Administer IV Mannitol**: To reduce intracranial pressure.
3. **Address Hypertension**:

- **Known case of hypertension**, which may cause hypertensive encephalopathy and cerebral edema.
- **Antihypertensive medications** will treat the condition, but not the first step. Metabolic causes should be addressed first.

Coma is a deep sleep-like state with no responsiveness.

Causes of Coma:

1. **Metabolic and Toxic Encephalopathies**:
    - Brain imaging will be normal.
2. **Meningitis Syndromes**.
3. **Diseases with Prominent Focal Signs**:
    - E.g. Stroke or Hemorrhage.
    - Urgent intervention is needed to prevent further brain damage.

Immediate Actions:

1. **Supplemental Oxygen**.

2. **Intravenous Thiamine** (At least 100 mg).
3. **Intravenous 50% Dextrose in Water** (25 g) Empirically:
    - Measure baseline serum glucose levels before administering glucose.

Initial Examination:

- General Appearance Assessment.
- Vital Signs Evaluation.
- Breath Sounds Examination.
- Responsiveness to Stimulation.
- Pupil Size and Responsiveness.
- Check for Abnormal Posturing or Movements.

In Trauma Cases:

- Stabilize the **neck until ruling out cervical spine fracture.**

Necessary Interventions:

1. **Hypotension**: Immediate therapeutic intervention needed.
2. **Significant Hypertension**: Immediate therapeutic intervention needed.
3. **Bradycardia**: Immediate therapeutic intervention needed.
4. **Arrhythmias Affecting Blood Pressure**: Immediate therapeutic intervention needed.
5. **Marked Hyperthermia**: Immediate therapeutic intervention needed.

Signs Indicating Urgent Lumbar Puncture: Meningeal irritation or fever.

Diagnostic Tests:

1. **Fundus Examination for Papilledema**.
2. **CT Scan of the Brain**:
    - Performed prior to lumbar puncture in comatose patients.

18.

Infertility in Kartagener's syndrome is due to which of the following?

✓Asthenospermia

•Oligospermia

•Undescended testes

•Epididymis obstruction

Infertility in Kartagener's syndrome is due to Kartagener's syndrome is a type of **primary ciliary dyskinesia**, also known as immotile cilia syndrome.

It is an autosomal recessive disorder that affects ciliary motility due to ultrastructural defects in the dynein arm of cilia.

The syndrome is characterized by the classical triad of symptoms, including:

- **Situs inversus:** Organs in the chest and abdomen are reversed from their normal positions.
- **Chronic sinusitis:** Persistent inflammation of the sinuses.
- **Bronchiectasis:** Abnormal widening and scarring of the airways in the lungs.

Infertility in Kartagener's Syndrome:

- Infertility in **Kartagener's syndrome** is primarily caused by asthenospermia, which refers to immotile spermatozoa due to impaired sperm flagella function.
- **Unlike azoospermia** (complete absence of spermatozoa in ejaculate), some sperm are present but unable to move properly due to the dysfunctional flagella.
- **This impaired sperm motility** leads to difficulties in fertilizing an egg, resulting in infertility.

Pathophysiology:

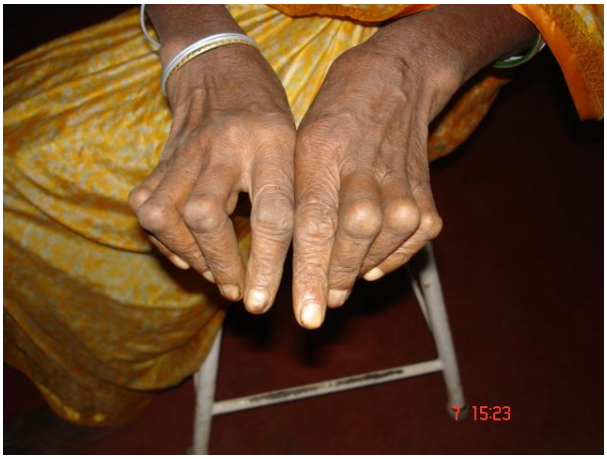
- Kartagener's syndrome is a result of the defective ciliary function, leading to a failed mucociliary clearance mechanism.
- The impaired ciliary motility causes the retention of secretions, leading to chronic sinusitis and recurrent infections.
- Over time, these persistent infections can cause bronchiectasis, a condition where the airways become dilated and scarred.

Clinical Presentation:

- Patients with Kartagener's syndrome may present with the following clinical features:
    - Chronic respiratory symptoms, such as a persistent cough, wheezing, and recurrent chest infections.
    - Chronic sinusitis, with symptoms like nasal congestion, facial pain, and nasal discharge.
    - Male infertility due to asthenospermia, which may lead to difficulties in conceiving a child.
    - Situs inversus, where the positions of organs in the chest and abdomen are reversed from the normal arrangement. This condition is seen in approximately half of the patients with primary ciliary dyskinesia.

19.

An elderly woman presents with a chronic history of pain in the small joints of hands with stiffness of joints in the early hours of the day. The image of the patient's hands is given below. What is the most likely diagnosis?



✓Rheumatoid Arthritis

•Complex Regional Pain Syndrome

•Osteoarthritis

•Villonodular synovitis

The image shows flexion at the **PIP joint and extension** in **the DIP joints** which is known as **boutonniere deformity** which is seen in rheumatoid arthritis(RA).

Clinical Presentation of Rheumatoid Arthritis

1. Early Morning Joint Symptoms

- The patient reports experiencing pain and stiffness in the joints, primarily in the early hours of the day.
- These symptoms tend to decrease as the day progresses.

2. Predominant Involvement of Smaller Joints

- RA typically affects smaller joints, such as those in the hands, wrists, and feet.

3. Mechanism

- The condition is characterized by the accumulation of inflammatory mediators in the affected joints.
- During increased joint mobility, these inflammatory mediators are flushed out due to enhanced blood supply, leading to a temporary reduction in symptoms.

Clinical Presentation of Osteoarthritis (OA)

1. Gradual Worsening of Joint Symptoms Throughout the Day

- Patients with OA experience pain and stiffness that progressively worsens as the day goes on.
- Increased joint activity contributes to the aggravation of symptoms.

2. Nodules Formation

- OA is associated with the development of nodules around the distal interphalangeal joint (Heberden nodes) and the proximal interphalangeal joint (Bouchard nodes).

20.

Calculate the GCS Score in an intubated patient, with findings of eye movements to pain and abnormal flexion?

•E2V1M3

•E2VTM3

•E2VTM4

✓E2VNTM3

Given findings points towards GCS Score: E2VNTM3

- Eye Opening (E): 2
- Verbal Response (V): NT (Not Testable)
- Motor Response (M): 3

Explanation:

- **Eye Opening (E): 2**

    - The patient's eye-opening response is scored as 2, indicating that they have spontaneous eye opening in response to pain stimuli.
- **Verbal Response (V): NT (Not Testable)**
    - The verbal component of the GCS score is denoted as 'Not Testable' in this case, which means that the patient's ability to produce a verbal response cannot be assessed. This could be due to endotracheal intubation or tracheostomy.
- **Motor Response (M): 3**
    - The patient's motor response is scored as 3, indicating abnormal flexion. The patient displays decerebrate posturing in response to painful stimuli.

Component	Score	Description
Eye Response	4	Spontaneously opens eyes
	3	Opens eyes to speech
	2	Opens eyes to pain
	1	Does not open eyes
Verbal Response	5	Oriented and converses coherently
	4	Confused, but able to answer questions
	3	Inappropriate responses or words
	2	Incomprehensible sounds
	1	No verbal response
Motor Response	6	Obeys commands appropriately
	5	Localizes pain (purposeful movement to pain)
	4	Withdraws from pain (pulls away)
	3	Abnormal flexion to pain (decorticate response)
	2	Extends in response to pain (decerebrate response)
	1	No motor response

Glasgow Coma Scale (GCS) Explanation:

Eyes Opening:

- Spontaneously: The patient's eyes open without any external stimulation.
- To verbal command: The patient's eyes open in response to a verbal command.

- To painful stimulus: The patient's eyes open in response to a painful stimulus.
- Do not open: The patient's eyes do not open even with any stimulation.
- Non-testable: The eye-opening response cannot be assessed.

Verbal Response:

- Normal-oriented conversation: The patient responds appropriately and coherently to verbal questions and conversation.
- Confused: The patient's verbal response is confused and disoriented.
- Inappropriate words only: The patient uses words, but they are inappropriate and do not make sense.
- Sounds only: The patient only produces sounds without forming words.
- No sounds: The patient does not produce any verbal sounds.
- Non-testable: The verbal response cannot be assessed.

Motor Response:

- Obeys commands: The patient follows commands and performs specific movements upon request.
- Localizes to pain: The patient localizes the source of pain and withdraws from it.
- Withdrawal/flexion: The patient withdraws or exhibits flexion in response to painful stimuli.
- Abnormal flexion/Decorticate posturing: The patient shows abnormal flexion posture in response to pain.
- Extension/Decerebrate posturing: The patient exhibits extension or decerebrate posturing in response to pain.
- No motor response: The patient does not display any motor response to stimuli.
- Non-testable: The motor response cannot be assessed.

GCS Score Interpretation:

- The GCS score ranges from **3 (minimum) to 15 (maximum).**
- A total score is not recorded if any component is non-testable (NT).
- In case of endotracheal intubation or tracheostomy, the verbal component is denoted as NT.
- A score of 'I' is not used for missing components; instead, NT is used to indicate non-testable components.
- The GCS, especially the motor score, is a valuable predictor of neurological outcome.

- Non-testable components may be left out to avoid confusion and misinterpretation by medical colleagues.
- A low GCS score may suggest a more severe condition than the patient actually has.

21.

A 60-year-old patient came with a history of hyponatremia. He was treated by a large volume of hypertonic fluids over 24 hours following which the patient developed quadriparesis. What is the most likely cause for this patient's condition?

✓Central Pontine Myelinolysis

•Brain Infarct

•Brainstem Injury

•Hypernatremia Rare Cause

The given clinical scenario is suggestive of **Central Pontine Myelinolysis a complication associated with hyponatremia**

Central Pontine Myelinolysis, also known as **Osmotic Demyelination Syndrome**, is a neurological disorder that occurs due to rapid correction of chronic hyponatremia (low sodium levels in the blood persisting for more than 48 hours).

It leads to the **efflux of organic osmolytes from brain cells, causing a reduction in intracellular osmolality** and osmotic gradient, leading to the entry of water into the cells.

Pathophysiology:

- Rapid correction of hyponatremia (>8-10 mM in 24 hours or 18 mM in 48 hours) induces hypertonic stress in brain astrocytes, resulting in demyelination without inflammation at the base of the pons while sparing axons and nerve cells.

Clinical Presentation: Patients typically present with one or more of the following symptoms one or more days after overcorrection of hyponatremia:

- Paraparesis or quadriparesis (weakness in the lower or all four limbs)
- Diplopia (double vision)
- Locked-in syndrome (a state of complete paralysis, except for eye movements and preserved consciousness)
- Dysphagia (difficulty in swallowing)
- Dysarthria (difficulty in speaking) with or without loss of consciousness

Diagnostic Imaging: MRI (Magnetic Resonance Imaging) is a valuable tool for diagnosing central pontine myelinolysis. It can also identify partial forms of the condition, presenting as confusion, dysarthria, and/or disturbances of conjugate gaze without paraplegia.

Preventive Measures: Prevention of central pontine myelinolysis involves gradual restoration of severe hyponatremia, with a recommended increase of 10 mmol/L (10 mEq/L) within 24 hours and 20 mmol/L (20 mEq/L) within 48 hours. This slower correction helps to reduce the risk of osmotic demyelination.

22.

Two brothers were arguing over a property dispute when the elder of the two complained of chest pain and collapsed and was later declared brought dead by the hospital. His family says he was previously healthy and there was no similar disease in the family members. What is the likely diagnosis?

•Acute myocardial infarction

•Infective cardiomyopathy

✓Takotsubo cardiomyopathy

•Hypertrophic cardiomyopathy

The given clinical scenario of sudden heart failure without any previous history is suggestive of Takotsubo cardiomyopathy

Takotsubo cardiomyopathy, also known as broken heart syndrome, apical ballooning syndrome, or stress-induced cardiomyopathy, is a transient myocardial stunning caused by microcirculation spasms due to a surge in catecholamines during periods of intense stress.

Epidemiology:

- Predominantly affects middle-aged women.

Clinical Presentation:

- Symptoms: Chest pain, hypotension, and pulmonary edema.

Echocardiography (ECHO):

- Findings: Apical dilatation with basal hypercontraction of the heart, resembling a Takotsubo (pot for catching octopus).

Electrocardiogram (ECG):

- Changes: ECG changes may mimic myocardial infarction, but the changes extend beyond a specific coronary arterial territory.

Diagnostic Evaluation:

- Coronary angiography may be required to rule out coronary occlusion.

Prognosis:

- Takotsubo cardiomyopathy is a self-limiting disorder and typically resolves within days to weeks.
- The prognosis is generally good.

Infective Cardiomyopathy

Definition: Infective cardiomyopathy is characterized by inflammation of the heart muscle primarily caused by viral infections and the protozoan Trypanosoma cruzi, which is associated with Chagas' disease.

Etiology:

Most common causes: Viral infections and Trypanosoma cruzi (Chagas' disease).

Clinical Presentation of Acute Viral Myocarditis:

Symptoms: Symptoms and signs of heart failure, chest pain, and ECG changes resembling pericarditis or acute myocardial infarction.

Chagas' Disease:

Abnormalities in the Conduction System: Dysfunction of the sinus node and AV node.

Right Bundle Branch Block: May be observed in Chagas' disease.

Acute Myocardial Infarction (MI)

Clinical Presentation:

Symptoms: Substernal chest pain persisting for minutes, and diaphoresis.

Sudden Cardiac Death: Arrhythmias are the leading cause of sudden cardiac death in acute myocardial infarction.

Hypertrophic Cardiomyopathy (HCM)

Definition: Hypertrophic cardiomyopathy is the most common genetic cardiovascular disorder and a significant cause of sudden death before the age of 35 years.

Risk Factors for Sudden Death in HCM:

- Young Age
- Nonsustained Ventricular Tachycardia
- Failure of Blood Pressure to Increase During Exercise
- Recent (Within 6 Months) Syncope
- Ventricular Wall Thickness > 3 cm
- Possibly the Severity of LV Outflow Obstruction

Sudden Death Mechanism in HCM:

Polymorphic Ventricular Tachycardia/Fibrillation: Responsible for sudden death in hypertrophic cardiomyopathy.

23.

Which of the following is a differentiating feature between cardiac tamponade and tension pneumothorax?

✓Breath sounds

•Increased heart rate

•Muffled heart sounds

•Raised JVP

The differentiating feature between cardiac tamponade and tension pneumothorax is **breath sounds.** The breath sounds are absent in tension pneumothorax while they are present in cardiac tamponade.

Clinical Features of Cardiac Tamponade	Clinical Features of Tension Pneumothorax
- Breath Sounds: Present	- Breath Sounds: Absent
- Hypotension: Yes	- Hypotension: Yes
- Distended Jugular Veins: Yes	- Distended Jugular Veins: Yes
- Heart Sounds: Muffled (Beck's triad)	- Heart Sounds: Muffled (Beck's triad)
- Additional Signs: None provided	- Additional Signs:
	- Hyperresonance to percussion
	- Mediastinal shift to the contralateral side

24.

Which of the following is used in the treatment of late cardiovascular syphilis?

•Benzathine penicillin 2.4 million units as single dose

✓Benzathine penicillin 2.4 million units weekly for three weeks

•Benzathine penicillin 12-24 million units for 21 days

•Tetracycline 2g daily

Benzathine penicillin 2.4 million units weekly for three weeks is the treatment of late cardiovascular syphilis.

Management

Patients allergic to penicillin: In **primary, secondary and early latent syphilis**, these patients should be given tetracycline or doxycycline for 2 weeks, and in late latent syphilis it should be given for 4 weeks.

Ceftriaxone: It may be given in the dose of 1 g once a day for 10–14 days.

Azithromycin: Single dose of 2 g is also effective except in homosexual men and during pregnancy.

Response to treatment is considered as satisfactory if there is a four-fold decrease in **non-treponemal titers at 3 to 6 months in all stages of syphilis.**

Neurosyphilis: It should be followed up with lumbar puncture and evaluation of CSF every 6 months until the cell count is normal. If after 6 months the cell count is not decreased, retreatment should be given.

Treatment reactions

Anaphylaxis: Penicillin is a common cause. Jarisch-Herxheimer reaction:

Treatment of syphilitic patients having a high bacterial load, by antibiotics can cause a massive release of endotoxins, and cytokine causing Jarisch-Herxheimer reaction.

The jarisch-Herxheimer

reaction is an acute febrile reaction that follows (about 8 hours after the first injection) any therapy for syphilis. It is characterized by headache, myalgia, malaise, mild fever, rigors, and other symptoms that usually resolve within the first 24 hours.

It is common in early syphilis and rare in late syphilis.

It may induce fetal distress or premature labor in pregnancy. This concern should not prevent or delay therapy. It may be severe and worsen the clinical manifestations in cardiovascular or neurosyphilis.

Penicillin should not be withheld because of the Jarisch–Herxheimer reaction. Because it is not dose-dependent and there is no point in giving a smaller dose.

Prednisolone 10–20 mg orally three times daily for 3 days given for 24 hours prior to therapy may prevent the reaction.

Stage of syphilis	*Drug*	*Regimen*
Primary	Procaine penicillin	6,00,000 units IM/day for 12 days
	Oxytetracycline	500 mg orally four times/day for 15 days
	Doxycycline	100 mg orally two times/day for 15 days
	Benzathine penicillin	2.4 mega (million) units IM single dose (1.2 million units in each buttock)
Secondary	Procaine penicillin	6,00,000 units IM once daily for 15 days
	Benzathine penicillin	2.4 million units IM single dose
Early latent	Benzathine penicillin	2.4 million units IM single dose
Late latent/tertiary and cardiovascular syphilis	Benzathine penicillin	2.4 million units IM weekly for 3 weeks
Neurosyphilis	Crystalline penicillin	18–24 million units/day for 10–14 days
	Procaine penicillin PLUS probenecid	2.4 million units/day IM for 10–14 day + 500 mg QID for 10–14 day

25.

A patient presented with chronic diarrhea and steatorrhea. D-xylose test was normal and the Schilling test was abnormal. A duodenal biopsy was normal. What is the most likely diagnosis?

•Celiac disease

•Ulcerative colitis

•Intestinal lymphangiectasia

✓Pancreatic insufficiency

In this clinical scenario, the **patient presents with malabsorption syndrome, characterized by chronic diarrhea and steatorrhea**. xylose test and a duodenal

biopsy, both were conducted which yielded normal results. However, the**Schilling test, which is used to assess vitamin B12 absorption**, showed abnormal findings, suggesting **pancreatic insufficiency.**

Clinical Presentation: Small Intestinal Pathologies

1. Celiac Disease:

- Normal xylose test and normal duodenal biopsy.
- The small intestine is affected due to the immune response to gluten.
- Autoimmune damage to the intestinal villi leads to malabsorption.

2. Intestinal Lymphangiectasia:

- Normal xylose test and normal duodenal biopsy.
- Disorder of lymphatic vessels in the intestinal wall.
- Impairs fat absorption, leading to edema and fat in the stools.

3. Ulcerative Colitis:

- Presence of steatorrhea rules out ulcerative colitis.
- Ulcerative colitis primarily affects the large bowel.
- Fat absorption occurs mainly in the small intestine.

Diagnosis: Ileal Disease

Clinical Features:

- Normal xylose test and normal duodenal biopsy.
- Presence of steatorrhea.
- Abnormal Schilling test.

Pancreatic insufficiency is characterized by the **deficiency of exocrine pancreatic enzymes, leading to maldigestion and malabsorption.**

Supporting Features:

1. **Steatorrhea:** Deficiency of pancreatic lipases results in undigested fat in stools.
2. **D-xylose Test:** D-xylose, a monosaccharide, is directly absorbed by the intestinal mucosa without undergoing any enzymatic action. A normal D-xylose test denotes an intact mucosa.
3. **Schilling Test:** The Schilling test is a**bnormal as pancreatic enzymes are required for the absorption of vitamin B12.**

26.

A triad of skin lesions, asymmetric mononeuritis multiplex and eosinophilia is seen in which of the following conditions?

•Polyarteritis nodosa

✓Churg Strauss syndrome

•Giant cell arteritis

•Cryoglobulinemic vasculitis

A triad of skin lesions, **asymmetric mono neuritis multiplex**, and eosinophilia is seen in Churg Strauss syndrome

Churg Strauss syndrome

Diagnosis Criteria:

- Evidence of asthma
- Peripheral blood and tissue eosinophilia (>1000 cells/µL)
- Extravascular granuloma formation
- Clinical features consistent with vasculitis

Common Presenting Symptoms:

- Severe attacks of asthma
- Allergic rhinitis, sinusitis, and nasal polyps (upper respiratory tract symptoms)
- Mono-neuritis multiplex (peripheral neuropathy affecting at least two nerves)
    - Complaints of pain, numbness, or weakness in the affected areas
- Skin lesions: palpable purpura and subcutaneous nodules

Renal Involvement: Less common and less severe than other small vessel vasculitis

Laboratory Finding: P-ANCA antibodies are present in Churg-Strauss syndrome

Treatment:

- Glucocorticoids: Effective in managing the condition

- Other drugs: Mepolizumab and rituximab have been tried

For Fulminant Multisystem Disease:

- Daily cyclophosphamide with prednisolone
- Followed by azathioprine or methotrexate

Differential Diagnosis:

Cryoglobulinemic Vasculitis

- Presents with palpable purpura, arthralgia, neuropathy, and glomerulonephritis
- Most commonly associated with Hepatitis C

Polyarteritis Nodosa

- May present with mononeuritis multiplex and skin lesions
- Does not have eosinophilia; predominantly neutrophilia is observed

Giant Cell Arteritis

- Manifests with a cardinal symptom of localized headache (temporal region)
- Common symptoms include headache, polymyalgia rheumatica, jaw claudication, fever, and weight loss
- Laboratory results show increased ESR, CRP levels, and normochromic/mildly hypochromic anemia

27.

Which of the following drugs is commonly used for treating community-acquired pneumonia in OPD?

•Streptomycin

✓Azithromycin

•Ceftriaxone

•Vancomycin

Azithromycin is drug of choice used for treating community-acquired pneumonia in OPD

CLINICAL GUIDELINES FOR OPD TREATMENT OF COMMUNITY-ACQUIRED PNEUMONIA

Previously Healthy Patients (No antibiotics within the past 3 months):

Treatment Options:

a) Azithromycin:

First dose: 500 mg orally
Subsequent dosing: 250 mg orally daily for 4 days, or 500 mg orally daily for 3 days

b) Clarithromycin:

Dosage: 500 mg orally twice a day

c) Doxycycline:

Dosage: 100 mg orally twice a day

Patients with Comorbid Medical Conditions (Chronic heart, lung, liver, or kidney disease, diabetes mellitus, alcoholism, malignancy, asplenia, immunosuppressant conditions, or recent antibiotic use within 3 months):

Treatment Options:

a) Respiratory Fluoroquinolone (choose one):

- Moxifloxacin: 400 mg orally daily
- Gemifloxacin: 320 mg orally daily
- Levofloxacin: 750 mg orally daily

b) Macrolide (choose one) + Beta-lactam (choose one):

Macrolide

- *Amoxicillin: 1 g orally three times a day*
- *Amoxicillin-clavulanate: 2 g orally twice a day*
- *Cefuroxime: 500 mg orally twice a day*

High Macrolide-Resistant Streptococcus pneumoniae (>25% infection rate with MIC ≥ 16 mcg/mL) in the region:

Consider alternative agents listed in (2) for patients with comorbidities.

Other Parenteral Options (not for outpatient management):

- Vancomycin
- Ceftriaxone
- Streptomycin

Management of Community-Acquired Pneumonia (CAP)

Risk Assessment: CAP severity can be assessed using the CURB-65 scoring system. Patients are classified based on their scores:

- Score 0: Treat as an outpatient
- Score 1 to 2: Admit to the hospital
- Score ≥3: Admission to the Intensive Care Unit (ICU) is recommended

Treatment Guidelines:

Score 0: Drug of choice: **Macrolides**

- Azithromycin 500mg once daily (OD) or
- Clarithromycin 500mg single dose (BD) or
- Doxycycline 100mg three times daily (BD)

Score I: Treatment:

- **Azithromycin 500mg** once daily (OD)
- **With Ceftriaxone 2gm once** daily (OD)

Note: **Respiratory quinolones** (e.g., Levofloxacin) are not preferred.

Score >2: Treatment:

- **Azithromycin 500mg** once daily (OD)
- With **Ceftriaxone 2gm** intravenous once daily **(IV OD)**
- Specific management according to the severity

Important Note: In a modified version of CURB-65, the criteria for blood urea nitrogen have been removed. This eliminates the need for lab testing.

28.

A patient walks with a stomping gait. When asked to close his eyes and walk, he is unable to do so. Which of the following tracts is probably affected?

✓Posterior column tract

•Spinocerebellar tract

•Rubrospinal tract

•Vestibulospinal tract

The given **clinical scenario is suggestive of the involvement of the Posterior column tract**

Proprioception, carried by **the posterior column of the spinal cord, is crucial for coordinating motor movements**. When there is a loss of proprioception, it can result in a stomping gait. This type of **gait abnormality is observed in conditions such as vitamin B12 deficiency**, which can lead to subacute combined degeneration, tabes dorsalis, or diabetic neuropathy.

29.

Which among the following is wrong about DIC?

•Increased schistocytes

✓Increased fibrinogen

•Increased PT

•Increased FDPs

In **Disseminated Intravascular Coagulation (DIC)** fibrinogen level decreases to bleeding

DIC is an **acquired syndrome characterized by widespread activation of blood clotting factors within blood vessels, leading to a loss of clotting control.**

Primary Mechanism The main mechanism of DIC involves the uncontrolled generation of thrombin through multiple pathways, resulting in sustained coagulation activation, formation of fibrin, and consumption of clotting factors and platelets.

Causes Infections are the **most frequent cause of DIC.**

Laboratory Investigations DIC can be diagnosed based on the following laboratory findings:

1. Decreased platelet count
2. Prolonged Prothrombin Time (PT)
3. Prolonged Activated Partial Thromboplastin Time (aPTT)
4. Decreased fibrinogen
5. Elevated fibrin degradation products (D-dimers)

Peripheral Smear Microangiopathic hemolytic anemia with schistocytes (fragmented red blood cells) may be observed in DIC.

Treatment The treatment of DIC involves the following approaches:

1. Platelet transfusion when platelet counts are below normal.
2. Replacement of fibrinogen and coagulation factors using Fresh Frozen Plasma (FFP) along with cryoprecipitate or fibrinogen concentrate.

Specific Situations In low-grade DIC associated with solid tumors or acute promyelocytic leukemia, low doses of continuous-infusion heparin have shown effectiveness.

30.

Which of the following is not a component of Syndrome Z?

•Obstructive sleep apnea

•Fasting triglyceride more than 150mg/dl

•Blood pressure more than 130/85 mmHg

✓LDL more than 100mg/dl

Syndrome Z is a characterized by the coexistence of obstructive sleep apnea syndrome (OSAS) with metabolic syndrome, LDL more than 100mg/dl is not a component of Syndrome Z

Metabolic Syndrome Diagnosis - NCEP: ATP III Criteria

To diagnose metabolic syndrome according to the National Cholesterol Education Program: Adult Treatment Panel III (NCEP: ATP III) criteria, three or more of the following criteria must be present:

1. **Central Obesity:**
    - Men: Waist circumference >102 cm

    - Women: Waist circumference >88 cm
2. **Hypertriglyceridemia:**
    - Fasting triglyceride level ≥150 mg/dL
    - Or on specific triglyceride-lowering medication
3. **Low HDL Cholesterol:**
    - Men: HDL cholesterol <40 mg/dL
    - Women: HDL cholesterol <50 mg/dL
    - Or on specific HDL-raising medication
4. **Hypertension:**
    - Systolic blood pressure ≥130 mmHg
    - Or diastolic blood pressure ≥85 mmHg
    - Or on specific antihypertensive medication
5. **Elevated Fasting Plasma Glucose:**
    - Fasting plasma glucose level ≥100 mg/dL
    - Or on specific glucose-lowering medication
    - Or previously diagnosed type 2 diabetes

Clinical Significance: Syndrome Z represents a significant health concern due to its combination of obstructive sleep apnea and metabolic disturbances. Metabolic syndrome is known to increase the risk of cardiovascular disease, type 2 diabetes, and other related complications.

Early ***diagnosis and management of this syndrome*** are crucial in preventing further health complications and improving overall well-being.

Clinicians should be **vigilant in assessing patients with symptoms suggestive of Syndrome Z** and consider appropriate interventions and treatment strategies to manage its components effectively.

31.

A patient with thalassemia has a history of multiple blood transfusions, iron overload, and cardiac arrhythmia. She has now come for blood transfusion and during the process, complains of backache and looks very anxious. What would you do next?

•Stop the transfusion. Wait for patient to become normal and then start it again

✓Stop the blood transfusion

•Continue the transfusion but do an ECG

•Do clerical check and get ECG

The clinical scenario where the patient is being transfused blood and has complaints of backache, this is **suggestive of an acute hemolytic transfusion reaction.**

Acute hemolytic transfusion reactions occur within 24 hours following a blood transfusion, primarily due to ABO incompatibility.

Presentation:

- Backache
- Hypotension
- Tachypnea
- Tachycardia
- Fever (1-2°C increase in temperature)
- Chills
- Chest and back pain
- Hemoglobinuria
- Hemoglobinemia
- Severe cases may lead to disseminated intravascular coagulation (DIC), acute renal failure, shock, and death.

Management:

- **Step 1: Stop Transfusion Immediately:** As soon as an acute hemolytic transfusion reaction is suspected, discontinue the blood transfusion promptly.

- **Step 2: Collect Blood Samples for Testing:** Obtain blood samples for further investigation and identification of the underlying cause.
- **Supportive Care:** Provide supportive care to the patient.
- **Hydration:** Administer vigorous hydration with isotonic saline to maintain adequate fluid balance.
- **Diuretics:** Use diuretics to promote urine output and ensure renal function.
- **Monitor Vital Signs:** Continuously monitor the patient's vital signs, including blood pressure, respiratory rate, heart rate, and temperature.
- **Treat Complications:** Address any complications that arise, such as DIC or acute renal failure, with appropriate medical interventions.
- **Preventive Measures:** To prevent future occurrences, ensure rigorous pre-transfusion testing to confirm ABO compatibility.

Note: Acute hemolytic transfusion reactions can be life-threatening, and immediate recognition and appropriate management are crucial to optimize patient outcomes.

32.

A 16-year-old girl, who is taking antiepileptics, has had a seizure-free period of 6 months. She has no family history of epilepsy. Her EEG is now normal and she has a normal neurological exam and intelligence. What would your advice be?

•Stop the treatment and follow up treatment

✓Continue treatment for another 18 months

•Gradually taper the drug and stop

•Continue lifelong treatment with antiepileptics

Clinical Scenario:

- Female patient
- Has been on antiepileptic drugs for 6 months
- No further seizures during this period
- No family history of epilepsy
- Normal EEG results
- Normal neurological examination

- Normal intelligence

Based on the history the patient should be advised to continue treatment for another 18 months

Treatment Plan:

- Continue antiepileptic drugs for another 18 months (totaling 2 years) to prevent seizure recurrence

Explanation:

1. **Treatment Duration:** The patient has been on antiepileptic drugs for 6 months with no seizure recurrence during this period. It is recommended to continue the medication for a total duration of 2 years to reduce the risk of seizure recurrence.

2. **Reasoning: Studies have shown that maintaining treatment with antiepileptic drugs for at least 2 years after the last seizure helps prevent seizure recurrence**. Since the patient has already completed 6 months of treatment, continuing for another 18 months will fulfill the minimum 2-year duration.

3. **Importance of Continued Treatment:** Abruptly stopping antiepileptic drugs can lead to a higher risk of seizure recurrence. To ensure optimal management, it is essential to follow the prescribed treatment plan and gradually reduce the dosage if necessary, under medical supervision.

Option A: Sudden Cessation of Drug Therapy: Stopping the medication suddenly is not advised as it may trigger seizures to recur.

Option C: Ineligibility for Medication Withdrawal: The patient has only experienced a seizure-free interval of 6 months, which is not sufficient to consider medication withdrawal.

Option D: Lifelong Treatment Not Necessary: Considering the patient's current clinical status, drug withdrawal is a viable option after completing 2 years of seizure freedom, as the patient meets the criteria for withdrawal.

33.

A patient with a history of a backache for 10 days, has now presented with sudden onset difficulty in micturition and defaecation. There was no history of a cough or fever previously. What is the diagnosis?

•Pott's Spine

•Guillain Barre Syndrome

✓Cauda Equina Syndrome

•Multiple Sclerosis

The clinical scenario suggests **cauda equina syndrome**

Injury of multiple **lumbosacral nerve roots within the spinal canal distal to the termination of the spinal cord at LI-L2.**

Causes:

- Ruptured lumbosacral intervertebral disk
- Lumbosacral spine fracture
- Hematoma within the spinal canal
- Compressive tumor
- Other mass lesions

Symptoms:

- Low back pain
- Weakness and areflexia in the legs
- Saddle anesthesia
- Loss of bladder function

Treatment options:

- Surgical decompression (sometimes urgent) to restore or preserve motor or sphincter function
- Radiotherapy for metastatic tumors.