

# NEET SS 2024 Diploma Paediatrics Question Paper 3 with Solutions

Time Allowed :3 Hours	Maximum Marks :100	Total Questions :10
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## General Instructions

Read the following instructions very carefully and strictly follow them:

1. The test is of 3 hours duration.
2. The question paper consists of 10 questions. The maximum marks are 100.
3. Each Question is of 10 marks.

### Q1. (a) Clinical features of Juvenile Idiopathic Arthritis.

#### Solution:

Juvenile Idiopathic Arthritis (JIA) is the most common type of arthritis in children and involves chronic inflammation of the joints. The clinical features can vary depending on the subtype of JIA, but common features include:

#### 1. Joint Involvement:

- Asymmetric or symmetric joint involvement, often affecting the knee, hip, and wrist.
- Swelling, pain, and stiffness are common in the affected joints.
- Morning stiffness and difficulty in moving joints after rest are typical symptoms.

#### 2. Systemic Features:

- Some children may experience fever, rash, or swollen lymph nodes, particularly in the systemic onset type (Still's disease).
- Fatigue and growth delays are often seen in children with chronic JIA.

#### 3. Age of Onset:

- JIA typically affects children under the age of 16 and often begins between 1-5 years of age.

#### 4. Duration:

- The condition typically lasts for more than six weeks. Chronicity and relapse are common.

#### Quick Tip

The diagnosis of JIA is based on the clinical presentation, and the symptoms may vary according to the type of JIA.

### Q1. (b) Investigation in Juvenile Idiopathic Arthritis.

#### Solution:

Investigating Juvenile Idiopathic Arthritis (JIA) involves both clinical and laboratory tests to confirm the diagnosis, identify the subtype, and exclude other conditions. Common investigations include:

1. Blood Tests:

- C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR): These are markers of inflammation and are often elevated in active disease.
- Complete blood count (CBC): Often shows anemia (due to chronic disease) and leukocytosis (in the systemic type).
- Rheumatoid factor (RF) and anti-cyclic citrullinated peptide (anti-CCP): These tests are typically negative in JIA but may be positive in some cases (especially in polyarticular JIA).
- Antinuclear antibody (ANA): Commonly positive in children with oligoarticular JIA, and it can be associated with an increased risk of uveitis.

2. Imaging:

- X-rays: Used to assess joint damage, erosion, and inflammation. In early stages, X-rays may be normal.
- Ultrasound: Can detect early joint inflammation, synovial fluid accumulation, and soft tissue changes.
- MRI: Useful in evaluating early joint changes, especially in the spine and hip.

3. Ophthalmologic Examination:

- Slit-lamp examination: Regular eye exams are essential to monitor for uveitis, a common complication of JIA, particularly in those with ANA-positive oligoarticular JIA.

Quick Tip

Regular ophthalmologic screening is crucial in JIA, especially in ANA-positive patients, as uveitis is a potentially serious complication.

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## Q1. (c) Treatment of Juvenile Idiopathic Arthritis.

### Solution:

The treatment of Juvenile Idiopathic Arthritis (JIA) aims to reduce inflammation, relieve symptoms, and prevent long-term joint damage. The approach is multidisciplinary and often involves pediatric rheumatologists, physiotherapists, and ophthalmologists. Treatment modalities include:

1. Nonsteroidal Anti-inflammatory Drugs (NSAIDs):

- Indications: First-line treatment for pain and inflammation.
- Common Medications: Ibuprofen, naproxen.
- Mechanism: NSAIDs work by reducing prostaglandin synthesis and have anti-inflammatory, analgesic, and antipyretic effects.
- Side Effects: Gastrointestinal upset, renal issues, and increased risk of cardiovascular events

with long-term use.

2. Disease-modifying Antirheumatic Drugs (DMARDs):

- Methotrexate: The mainstay of treatment for most JIA subtypes. It reduces inflammation and prevents joint damage by modulating the immune system.
- Sulfasalazine and leflunomide: Alternatives in some cases.
- Side Effects: Hepatotoxicity, gastrointestinal distress, and blood dyscrasias.

3. Biologic Agents:

- Tumor Necrosis Factor (TNF)-inhibitors (e.g., etanercept, adalimumab): Used when disease is resistant to conventional DMARDs.
- Interleukin-6 (IL-6) inhibitors (e.g., tocilizumab): Used in systemic JIA or when other biologics are ineffective.
- Indications: Reserved for moderate to severe disease that is unresponsive to DMARDs.
- Side Effects: Risk of infections, malignancies, and immune reactions.

4. Corticosteroids:

- Indications: Used in severe, active disease or during flare-ups to rapidly control inflammation.
- Routes of Administration: Oral, intra-articular, or intravenous depending on disease severity.
- Side Effects: Long-term use can cause growth retardation, osteoporosis, and cataracts.

5. Physical and Occupational Therapy:

- A critical component of treatment to maintain joint mobility, muscle strength, and overall functionality. Regular physiotherapy helps prevent deformities and enhances quality of life.

6. Surgical Treatment:

- In severe cases with significant joint damage or deformities, surgical interventions such as synovectomy or joint replacement may be required.

**Step 2: Management of Complications.**

- Uveitis: Regular ophthalmologic monitoring is essential. Treatment may include topical corticosteroids or systemic immunosuppressive therapy if uveitis develops.
- Growth and Development Issues: Long-term monitoring is needed to manage growth issues due to disease activity or medication side effects.

**Quick Tip**

The goal of JIA treatment is to control inflammation early to prevent joint damage and promote normal growth and development in children.

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**Q2. (a) Diagnostic criteria for Chronic Liver Disease.**

**Solution:**

Chronic liver disease (CLD) refers to long-term liver dysfunction that can lead to cirrhosis and liver failure. The diagnostic criteria for CLD generally include the following:

1. History:

- Risk factors: A history of alcohol abuse, viral hepatitis (hepatitis B or C), autoimmune diseases, or metabolic disorders (e.g., non-alcoholic fatty liver disease).
- Symptoms: Symptoms may include fatigue, jaundice, abdominal pain, and signs of portal hypertension (e.g., varices, ascites).

2. Laboratory Tests:

- Elevated liver enzymes (AST, ALT): Mild to moderate increases may be seen in liver inflammation.
- Alkaline phosphatase (ALP): Elevation suggests cholestasis or biliary obstruction.
- Bilirubin levels: Jaundice or elevated bilirubin levels may suggest liver dysfunction.

3. Imaging:

- Ultrasound: Detects liver texture changes, ascites, or hepatomegaly. It may show signs of cirrhosis such as a nodular liver surface.
- CT or MRI: Used to assess liver morphology, complications like varices, and liver fibrosis.
- Liver biopsy: The gold standard for assessing liver damage, fibrosis, and cirrhosis.

**Quick Tip**

Chronic liver disease is diagnosed based on clinical history, laboratory tests, imaging studies, and, when necessary, a liver biopsy.

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**Q2. (b) Laboratory diagnosis of Chronic Liver Disease.**

**Solution:**

The laboratory diagnosis of chronic liver disease (CLD) involves a combination of tests to assess liver function, identify the cause of the disease, and assess the degree of liver damage.

1. Liver Function Tests (LFTs):

- AST (Aspartate Aminotransferase) and ALT (Alanine Aminotransferase): These enzymes are released into the bloodstream when liver cells are damaged. ALT is more specific to the liver, while AST may also be elevated in conditions involving other organs.
- Alkaline Phosphatase (ALP): Elevated in cholestatic liver disease, biliary obstruction, or cirrhosis.
- Bilirubin: Elevated levels of total bilirubin may indicate liver dysfunction, as seen in cirrhosis or hepatocellular injury.
- Albumin: Decreased levels of albumin are seen in advanced liver disease, as the liver's ability to synthesize proteins is impaired.

## 2. Viral Hepatitis Markers:

- Hepatitis B (HBV): Presence of HBsAg (Hepatitis B surface antigen) indicates an ongoing infection, while anti-HBc (Hepatitis B core antibody) and anti-HBs (Hepatitis B surface antibody) help assess immunity or past infection.
- Hepatitis C (HCV): Detection of anti-HCV antibodies and HCV RNA indicates an active or past infection. The HCV genotype helps guide treatment decisions.

## 3. Autoimmune Markers:

- Anti-nuclear antibodies (ANA): Positive in autoimmune liver diseases like autoimmune hepatitis.
- Anti-smooth muscle antibodies (ASMA): Associated with autoimmune hepatitis.
- Immunoglobulin levels: Elevated immunoglobulin G (IgG) levels are common in autoimmune hepatitis.

## 4. Alpha-1 Antitrypsin Levels:

- Alpha-1 antitrypsin deficiency can cause cirrhosis in children and adults. Low levels of this enzyme are associated with liver damage.

## 5. Serum Ferritin and Transferrin Saturation:

- Elevated serum ferritin and transferrin saturation can indicate hemochromatosis, a condition causing iron overload in the liver.

## 6. Prothrombin Time (PT):

- Prolonged PT or INR (international normalized ratio) indicates impaired liver function and synthetic capacity, often seen in cirrhosis.

### Quick Tip

Liver function tests, viral hepatitis markers, and autoimmune markers are critical for diagnosing and identifying the etiology of chronic liver disease.

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## Q2. (c) Management of Chronic Liver Disease.

### Solution:

The management of chronic liver disease (CLD) focuses on treating the underlying cause, managing symptoms, preventing complications, and improving the patient's quality of life.

#### 1. General Measures:

- Lifestyle modifications: Patients should avoid alcohol, follow a balanced diet, and engage in regular exercise.
- Vaccination: Vaccination against hepatitis A and B is recommended, especially in patients with cirrhosis, to prevent additional liver injury.
- Management of comorbidities: Controlling associated conditions like diabetes, hypertension,

and obesity is important for preventing further liver damage.

## 2. Medical Therapy:

- Antiviral therapy: For patients with hepatitis B or C, antiviral medications like tenofovir (HBV) or sofosbuvir (HCV) can help suppress viral replication and reduce liver inflammation.
- Immunosuppressive therapy: In cases of autoimmune liver diseases like autoimmune hepatitis, prednisolone or azathioprine may be used to suppress the immune system and reduce inflammation.
- Chelation therapy: For patients with hemochromatosis, chelating agents like deferasirox may be used to reduce iron overload.

## 3. Management of Complications:

- Ascites management: Diuretics (spironolactone, furosemide) are used to control fluid retention. In refractory cases, paracentesis (removal of fluid from the abdomen) may be necessary.
- Variceal bleeding: Endoscopic band ligation or TIPS (transjugular intrahepatic portosystemic shunt) can be used to treat varices and reduce portal pressure.
- Hepatic encephalopathy: Lactulose is used to reduce ammonia levels in the blood, improving cognitive function.

## 4. Surgical Options:

- Liver transplantation: In cases of end-stage liver disease or cirrhosis, liver transplantation may be the only option. It is reserved for patients who do not respond to medical therapy and have a poor prognosis.

### **Step 2: Hepatocellular Carcinoma (HCC) Screening.**

- Surveillance: Patients with chronic liver disease, particularly those with cirrhosis, should undergo regular screening for hepatocellular carcinoma (HCC) with ultrasound and serum alpha-fetoprotein (AFP) testing.

#### Quick Tip

Management of chronic liver disease is multifaceted, involving antiviral or immunosuppressive therapy, lifestyle changes, and treatment of complications like ascites and variceal bleeding.

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### **Q3. (a) Prevention of Hepatitis C.**

#### **Solution:**

Prevention of Hepatitis C (HCV) focuses on reducing the risk of transmission, as there is currently no vaccine available for HCV. The main preventive measures include:

#### 1. Avoiding Blood-to-Blood Contact:

- Safe needle practices: Avoid sharing needles or other equipment for injecting drugs. Safe needle disposal and needle exchange programs can reduce transmission among intravenous drug users.

- Screening blood donations: All donated blood should be screened for HCV to avoid transfusion-related transmission.

2. Safe Medical Practices:

- Sterilization of medical equipment: Medical tools (e.g., syringes, dental equipment) should be properly sterilized to prevent transmission during medical procedures.

- Prevention of transmission in healthcare settings: Adherence to universal precautions and appropriate use of personal protective equipment (PPE) for healthcare workers.

3. Education and Awareness:

- Educating high-risk populations, such as people with multiple sexual partners or those undergoing hemodialysis, on safe practices.

Quick Tip

Avoiding blood-to-blood contact and safe medical practices are key in preventing the spread of Hepatitis C, as there is no vaccine yet.

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**Q3. (b) Mother to Child Transmission of Hepatitis C.**

**Solution:**

Mother-to-child transmission of Hepatitis C (HCV) occurs when the virus is transmitted from an infected mother to her baby during childbirth. The key points related to mother-to-child transmission include:

1. Risk Factors for Transmission:

- Maternal HCV Viral Load: Higher maternal HCV viral load, especially during pregnancy, increases the risk of vertical transmission.

- Coinfection with HIV: Mothers who are coinfecting with HIV have a higher risk of transmitting HCV to the infant.

- Mode of Delivery: Cesarean delivery does not significantly reduce the risk of transmission, as the primary risk is during the passage through the birth canal.

2. Incidence of Transmission:

- The estimated rate of mother-to-child transmission of HCV is approximately 5-10%.

- The risk is generally lower in mothers with low or undetectable HCV RNA levels.

3. Prevention Strategies:

- No routine screening for HCV in pregnant women: HCV screening is not universally recommended for all pregnant women, but it should be considered in high-risk groups (e.g., women with a history of injection drug use).

- Antiviral Treatment during Pregnancy: There is currently no approved antiviral therapy during pregnancy to reduce the risk of HCV transmission, but treating mothers with HIV can reduce the overall transmission risk.

#### 4. Postpartum Care:

- **Infant Testing:** Infants born to HCV-positive mothers should be tested for HCV. Initial testing is done after 18 months of age due to the possibility of the presence of maternal antibodies in the newborn.

#### Quick Tip

Mother-to-child transmission of HCV is most common during delivery, but the risk can be minimized by managing maternal HCV viral load and treating coinfection with HIV.

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### Q3. (c) Management of a child with Hepatitis C.

#### **Solution:**

The management of a child with Hepatitis C (HCV) includes antiviral therapy, monitoring liver function, and preventing complications. The treatment strategy is dependent on the child's age, HCV genotype, and the presence of any liver damage.

#### 1. Initial Assessment:

- **Liver Function Tests (LFTs):** Monitoring levels of AST, ALT, and bilirubin to assess liver involvement.
- **HCV RNA Test:** Quantitative PCR test to measure the viral load and determine the amount of virus in the blood.
- **Genotype Testing:** To determine the most effective antiviral regimen, particularly in determining if the child has genotype 1 or another subtype.
- **Liver Biopsy or Elastography:** In some cases, a liver biopsy or non-invasive imaging (e.g., elastography) may be required to assess liver fibrosis or cirrhosis.

#### 2. Antiviral Therapy:

- **Direct-acting antivirals (DAAs):** The mainstay of treatment for children over the age of 3 years with chronic HCV. Commonly used DAAs include sofosbuvir, ledipasvir, glecaprevir/pibrentasvir, and velpatasvir.
- **Treatment Duration:** Depending on the child's genotype, treatment may last between 8-12 weeks. The goal is to achieve sustained virologic response (SVR), meaning undetectable HCV RNA 12 weeks after completing therapy.

#### 3. Monitoring:

- **Follow-up testing:** Regular follow-up with liver function tests and HCV RNA testing during and after treatment to assess treatment response.
- **Screening for Liver Damage:** Regular screening for liver damage (including cirrhosis and hepatocellular carcinoma) if the child has chronic HCV.

#### 4. Management of Complications:

- **Liver transplantation:** In severe cases with cirrhosis or liver failure, liver transplantation may be necessary.
- **Supportive care:** Symptom management for fatigue or pruritus and maintaining a healthy

lifestyle with balanced nutrition.

5. Prevention:

- Vaccination: Vaccination against Hepatitis A and B is recommended to prevent further liver damage in children with chronic HCV.
- Psychosocial Support: Counseling and support for the child and family, particularly regarding the chronic nature of the disease and the treatment process.

**Quick Tip**

DAAAs are the cornerstone of HCV treatment in children and offer high cure rates with minimal side effects.

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**Q4. (a) Clinical features of Scrub Typhus.**

**Solution:**

Scrub typhus is a rickettsial infection caused by \*Orientia tsutsugamushi\*, transmitted through the bite of infected chiggers (larvae of mites). The clinical features include:

1. Fever: Sudden onset of high fever, often with chills.
2. Rash: A maculopapular or petechial rash may develop after 3-5 days of fever, often starting on the trunk.
3. Eschar: A characteristic painless, necrotic ulcer (eschar) at the site of the mite bite is present in many patients, though not all.
4. Lymphadenopathy: Swollen lymph nodes near the site of infection.
5. Headache and muscle pain: Generalized symptoms like headache, myalgia, and malaise.
6. Other symptoms: Nausea, vomiting, and abdominal pain may also be present in some cases.

**Quick Tip**

The presence of an eschar at the site of a mite bite is highly suggestive of scrub typhus, especially in endemic areas.

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**Q4. (b) Diagnosis of Scrub Typhus.**

**Solution:**

Diagnosis of scrub typhus is made based on clinical features, patient history, and laboratory tests. The key diagnostic approaches include:

1. Serology:
  - Indirect Immunofluorescence Assay (IFA): The most reliable test for diagnosing scrub typhus.

Detection of IgM and IgG antibodies against \*Orientia tsutsugamushi\*.

- Enzyme-linked immunosorbent assay (ELISA): Detects antibodies to the rickettsial antigen.
- PCR (Polymerase Chain Reaction): Used to detect \*Orientia tsutsugamushi\* DNA in blood or tissue samples. It is highly sensitive during the acute phase.

2. Blood Culture:

- Blood culture can isolate the causative organism, but it is less commonly performed due to difficulty and time involved.

3. Rapid Diagnostic Tests (RDTs):

- RDTs based on detecting IgM antibodies or antigen can provide quick results in endemic regions.

#### Quick Tip

PCR is the most sensitive method for early diagnosis of scrub typhus, especially during the acute phase.

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#### Q4. (c) Treatment of Scrub Typhus.

##### **Solution:**

The treatment of scrub typhus is primarily antibiotic therapy, which should be started promptly to reduce morbidity and mortality. The treatment approach includes:

1. First-Line Antibiotics:

- Doxycycline: The drug of choice for adults and children over 8 years old. It is effective against \*Orientia tsutsugamushi\* and should be administered for at least 7-10 days.
- Alternatives:
  - Chloramphenicol is an alternative for children under 8 years of age or those who cannot tolerate doxycycline.
  - Azithromycin can also be used in pregnant women or children under 8 years old.

2. Supportive Care:

- Hydration: Oral or intravenous fluids should be administered to prevent dehydration.
- Fever Management: Antipyretics like acetaminophen can be used to control fever.
- Monitoring: Continuous monitoring for complications such as organ failure, particularly in severe cases.

3. Prognosis:

- With prompt antibiotic treatment, the prognosis is generally good, and most patients recover fully. However, delay in treatment can lead to severe complications, including pneumonia, hepatic failure, encephalitis, and acute renal failure.

#### 4. Prevention:

- Personal Protection: Use of insect repellents, wearing protective clothing, and avoiding exposure to areas with high mite infestations can help prevent infection.
- Vector Control: Controlling the populations of infected mites is crucial in endemic regions.

#### Quick Tip

Starting treatment with doxycycline as early as possible is critical for preventing severe complications of scrub typhus.

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### Q5. (a) Etiology of Acute Flaccid Paralysis.

#### Solution:

Acute Flaccid Paralysis (AFP) is characterized by sudden-onset weakness or paralysis, usually affecting the limbs, with associated reduced or absent reflexes. The common etiologies of AFP include:

#### 1. Viral Infections:

- Poliovirus: Historically the most common cause of AFP, particularly in unvaccinated populations.
- Enterovirus (EV71): This virus is also a significant cause, often associated with outbreaks of hand, foot, and mouth disease.
- Other viruses: Adenoviruses, herpesviruses (like Herpes simplex virus), and Zika virus have also been associated with AFP.

#### 2. Bacterial Infections:

- Guillain-Barré syndrome (GBS): An autoimmune disease triggered by bacterial or viral infections, particularly *Campylobacter jejuni*, leading to ascending paralysis.
- Tetanus: Though rare, *Clostridium tetani* infection can cause AFP through neurotoxin release.

#### 3. Toxic Causes:

- Botulism: Caused by *Clostridium botulinum* neurotoxin, it results in descending flaccid paralysis and is a critical differential diagnosis for AFP.

#### 4. Other Causes:

- Neuromuscular disorders: Disorders like myasthenia gravis or spinal cord trauma can mimic AFP but are less common.
- Metabolic conditions: Some metabolic conditions (e.g., hypokalemia) can cause flaccid paralysis.

#### Quick Tip

Poliovirus and enterovirus infections remain the leading causes of acute flaccid paralysis, particularly in regions with low vaccination rates.

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**Q5. (b) Laboratory evaluation of Acute Flaccid Paralysis.****Solution:**

Laboratory evaluation is essential to confirm the diagnosis, identify the causative agent, and rule out other possible causes of paralysis. The key laboratory tests include:

## 1. Viral Studies:

- Throat and stool samples: These are collected early (within 14 days of onset) and sent for viral culture, polymerase chain reaction (PCR), and sequencing to detect poliovirus or other enteroviruses.
- Cerebrospinal fluid (CSF) analysis: CSF should be collected to assess for signs of inflammation (pleocytosis) and rule out meningitis. PCR can be used to detect viral DNA or RNA, confirming viral etiology.

## 2. Blood Tests:

- Complete blood count (CBC): May show signs of infection or inflammation.
- Serological tests: Tests for antibodies to viruses (like enteroviruses or herpesviruses) can be performed to identify past infections or current viral replication.

## 3. Electromyography (EMG):

- Motor nerve conduction studies can assess the extent of motor nerve damage and aid in confirming the diagnosis of neuropathic vs. myopathic conditions.

## 4. Toxin Detection:

- In cases of suspected botulism, serum and stool samples should be tested for the botulinum toxin using toxicity assays.

## 5. Spinal Imaging:

- In cases of suspected spinal cord injury or neurological diseases, MRI of the spinal cord may be performed to rule out structural causes like tumors or infections.

**Quick Tip**

Early stool and throat samples are crucial for detecting viral causes like poliovirus or enterovirus, especially within the first two weeks of symptom onset.

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**Q5. (c) Management of Acute Flaccid Paralysis.****Solution:**

The management of Acute Flaccid Paralysis (AFP) is multidisciplinary, focusing on the identification of the underlying cause, supportive care, and preventing complications. Key aspects of management include:

### 1. Supportive Care:

- Airway management: Some patients may require ventilatory support if respiratory muscles are involved.
- Pain management: Analgesics and muscle relaxants are used to control pain and spasticity.
- Physical therapy: Early rehabilitation is essential to prevent muscle atrophy, improve function, and enhance recovery.

### 2. Antiviral Treatment:

- For enteroviral infections, there is no specific antiviral therapy, but supportive care and hydration are essential.
- For botulism, botulinum antitoxin should be administered as soon as possible to prevent further toxin effects. Antibiotics may also be given if a bacterial source is identified.
- In Guillain-Barré syndrome (GBS), intravenous immunoglobulin (IVIg) or plasmapheresis can be used to modulate the immune response and prevent further nerve damage.

### 3. Botulism Management:

- In cases of botulism, besides antitoxin therapy, wound care and supportive treatments such as respiratory support and nutritional management are essential.

### 4. Monitoring and Prognosis:

- Patients with AFP should be monitored for complications such as respiratory failure or autonomic instability. The prognosis depends on the underlying cause—patients with viral or toxic causes may recover fully, while those with more severe etiologies, like GBS, may require prolonged rehabilitation.

### 5. Vaccination:

- For poliovirus-related AFP, vaccination is critical for prevention. Children should receive the full course of the polio vaccine to prevent future outbreaks.

#### Quick Tip

Early supportive care, including pain management and physical therapy, is critical to prevent complications and improve recovery outcomes in AFP.

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## Q6. (a) Clinical manifestations of Myocarditis.

### Solution:

Myocarditis is an inflammation of the heart muscle (myocardium) caused by infection, autoimmune diseases, or other factors. The clinical manifestations of myocarditis can vary depending on the severity of the inflammation and the underlying cause. Common clinical features include:

#### 1. Chest Pain:

- The most common symptom in myocarditis, often described as a sharp, stabbing pain. It

may mimic that of a myocardial infarction (heart attack).

2. Fatigue and Weakness:

- Due to decreased cardiac output, patients may experience fatigue, weakness, and general malaise.

3. Heart Failure Symptoms:

- Shortness of breath (dyspnea), especially on exertion, and swelling in the legs or abdomen due to fluid retention.

4. Arrhythmias:

- Palpitations, tachycardia, or irregular heartbeats can occur due to the disruption of the normal electrical activity of the heart.

5. Fever:

- Mild to moderate fever is common, especially in viral myocarditis, and is usually associated with other systemic symptoms like chills and muscle aches.

6. Syncope or Lightheadedness:

- This can occur if the heart's pumping ability is severely compromised.

7. Signs of Systemic Inflammation:

- In viral or autoimmune myocarditis, symptoms such as joint pain, rashes, and enlarged lymph nodes may be present.

Quick Tip

Myocarditis can present with symptoms that mimic a heart attack, so it is essential to differentiate between the two, especially in young patients with no prior heart disease.

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## Q6. (b) Diagnosis of Myocarditis.

### Solution:

The diagnosis of myocarditis involves a combination of clinical evaluation, laboratory tests, imaging, and biopsy. The key diagnostic steps include:

1. Clinical History:

- Detailed patient history to identify risk factors like recent viral infections (e.g., influenza, COVID-19) or autoimmune diseases.

2. Electrocardiogram (ECG):

- Myocarditis often shows nonspecific ST-segment and T-wave changes, which may mimic acute

coronary syndrome. In severe cases, arrhythmias like atrial fibrillation or ventricular tachycardia can occur.

### 3. Blood Tests:

- Troponin levels: Elevated in myocarditis due to myocardial injury.
- C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR): Markers of inflammation, which may be elevated in inflammatory myocarditis.
- Viral PCR or antibody tests: To detect viruses that can cause myocarditis, such as enteroviruses or adenoviruses.

### 4. Echocardiography:

- This imaging technique assesses heart function and identifies signs of heart failure. It can reveal left ventricular dysfunction and reduced ejection fraction.

### 5. Cardiac Magnetic Resonance Imaging (MRI):

- The gold standard for imaging in suspected myocarditis. MRI shows late gadolinium enhancement in areas of myocardial injury and inflammation.

### 6. Endomyocardial Biopsy:

- This is the definitive diagnostic test for myocarditis, though it is rarely performed. It involves taking a small sample of heart tissue for histological examination to look for evidence of inflammation and viral particles.

#### Quick Tip

Cardiac MRI is highly sensitive for diagnosing myocarditis and can detect myocardial inflammation even before changes in heart function occur.

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## Q6. (c) Treatment of Myocarditis.

### Solution:

The treatment of myocarditis is focused on managing the underlying cause, relieving symptoms, and preventing complications like heart failure and arrhythmias. The main treatment strategies include:

#### 1. Supportive Care:

- Rest: Bed rest and avoidance of strenuous physical activity are recommended to reduce the strain on the heart.
- Fluid management: Diuretics may be used to manage fluid retention in cases of heart failure.

#### 2. Antiviral Therapy:

- If a viral cause is identified, specific antiviral treatments may be administered, although there are no FDA-approved antivirals for most viral myocarditis. Treatment focuses on supportive

care while the body fights off the infection.

3. Immunosuppressive Therapy:

- Corticosteroids or immunosuppressive drugs may be used in cases of autoimmune myocarditis, such as that caused by conditions like lupus or sarcoidosis.

4. Heart Failure Management:

- ACE inhibitors, beta-blockers, and diuretics are used to manage heart failure symptoms and improve heart function.

- Inotropic agents may be used in severe cases to improve cardiac output.

5. Arrhythmia Management:

- Antiarrhythmic drugs or implantable cardioverter-defibrillators (ICDs) may be required if life-threatening arrhythmias develop.

6. Hospitalization and Advanced Care:

- In severe cases of myocarditis with acute heart failure or shock, patients may require mechanical circulatory support (e.g., ventricular assist devices) or heart transplantation in cases of refractory disease.

7. Vaccination:

- Vaccination against preventable infections like influenza and COVID-19 can reduce the risk of viral infections that can lead to myocarditis.

**Quick Tip**

Early treatment with heart failure medications, rest, and careful monitoring can significantly improve outcomes in myocarditis patients.

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**Q7. (a) Genetics of Cystic Fibrosis.**

**Solution:**

Cystic fibrosis (CF) is an autosomal recessive genetic disorder caused by mutations in the CFTR gene (Cystic Fibrosis Transmembrane Conductance Regulator), located on chromosome 7. The CFTR gene encodes a protein that regulates the movement of chloride ions across cell membranes, affecting the lungs, pancreas, liver, and intestines.

1. Inheritance:

- CF is inherited in an autosomal recessive manner, meaning a child must inherit one defective gene from each parent to develop the disease. Parents who are carriers (have one defective gene) do not show symptoms of CF but can pass the defective gene to their offspring.

2. Mutation:

- Over 2,000 different mutations in the CFTR gene have been identified, but the most common mutation is F508del (a deletion of three nucleotides resulting in the loss of phenylalanine at

position 508 of the protein).

### 3. Pathophysiology:

- The CFTR protein dysfunction leads to thick, sticky mucus in various organs, causing blockages and damage, particularly in the lungs and digestive system.

#### Quick Tip

Cystic fibrosis is inherited in an autosomal recessive pattern, meaning both parents must carry a copy of the defective CFTR gene for a child to develop the disease.

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## Q7. (b) Clinical Features of Cystic Fibrosis.

### Solution:

Cystic fibrosis affects multiple organ systems, and its clinical features can vary in severity. The most common systems involved are the respiratory and gastrointestinal systems.

#### 1. Respiratory System:

- Chronic cough and sputum production: Due to thick mucus accumulation in the lungs.
- Recurrent respiratory infections: Caused by bacterial colonization of the lungs (e.g., \*Pseudomonas aeruginosa\*, \*Staphylococcus aureus\*).
- Wheezing and shortness of breath: Resulting from airway obstruction and inflammation.
- Clubbing of the fingers and toes: Often seen in advanced disease due to chronic hypoxia.

#### 2. Gastrointestinal System:

- Pancreatic insufficiency: Most patients with CF have difficulty digesting food due to a lack of enzymes produced by the pancreas, leading to malabsorption, poor growth, and steatorrhea (fatty stools).
- Meconium ileus: A condition where the first stool is abnormally thick and can cause intestinal obstruction in newborns.
- Chronic diarrhea: Due to malabsorption and bile salt deficiency.
- Liver disease: Progressive liver damage and cirrhosis can occur in some patients.

#### 3. Reproductive System:

- Infertility in males: A majority of men with CF are infertile due to congenital absence of the vas deferens.
- Reduced fertility in females: Due to thick cervical mucus.

#### 4. Other Features:

- Salty sweat: Increased sodium and chloride concentrations in sweat are a hallmark of CF and can be detected by a sweat chloride test.

### Quick Tip

A salty taste to the skin is a characteristic sign of cystic fibrosis due to increased chloride concentrations in sweat.

## Q7. (c) Management of Cystic Fibrosis.

### Solution:

The management of cystic fibrosis (CF) is multidisciplinary and aims to improve the quality of life, slow disease progression, and manage complications. Key components of management include:

#### 1. Respiratory Management:

- Airway clearance: Techniques like chest physiotherapy (e.g., postural drainage and percussion), high-frequency chest wall oscillation (HFCWO), and inhaled therapies help clear mucus from the lungs.
- Inhaled medications:
  - Bronchodilators (e.g., albuterol) help open the airways.
  - Mucolytics (e.g., dornase alfa) break down mucus, making it easier to clear.
  - Hypertonic saline: Helps to hydrate the airway surface and loosen mucus.
  - Inhaled antibiotics: To prevent and treat lung infections, particularly with \*Pseudomonas aeruginosa\*.

#### 2. Gastrointestinal Management:

- Pancreatic enzyme replacement: Enzyme supplements (e.g., Creon) are necessary for patients with pancreatic insufficiency to aid digestion and nutrient absorption.
- Nutritional support: High-calorie, high-fat diet, and fat-soluble vitamin supplementation (A, D, E, K).
- Treating liver disease: Ursodeoxycholic acid may be used for liver disease, and in some cases, liver transplantation may be necessary.

#### 3. Endocrine Management:

- Diabetes management: Many patients with CF develop cystic fibrosis-related diabetes (CFRD), requiring insulin therapy for glucose control.

#### 4. Gene Therapy and CFTR Modulators:

- CFTR modulators (e.g., ivacaftor, lumacaftor) help correct the defective CFTR protein in some patients, improving lung function and other symptoms.
- Gene therapy: Still under research, but holds promise for future CF treatments by targeting the genetic defect.

#### 5. Transplantation:

- Lung transplantation is considered in patients with end-stage respiratory failure, and liver

transplantation may be required for patients with cirrhosis.

6. Psychosocial Support:

- Psychological counseling, support groups, and education are important for patients and families to manage the chronic nature of CF.

Quick Tip

Gene therapy and CFTR modulators have transformed the treatment of cystic fibrosis, especially in patients with specific genetic mutations.

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**Q8. (a) Causes of sudden onset breathlessness in a 3-year-old child.**

**Solution:**

Sudden onset breathlessness in a 3-year-old child can be caused by a variety of conditions, including:

1. Asthma:

- Asthma is a common cause of acute breathlessness in young children, often triggered by viral infections, allergens, or environmental factors. Children with asthma may present with wheezing, cough, and difficulty breathing.

2. Respiratory Infections:

- Viral infections, such as respiratory syncytial virus (RSV) or parainfluenza virus, can cause acute bronchiolitis or pneumonia, leading to respiratory distress, wheezing, and difficulty breathing.  
- Bacterial pneumonia can also lead to sudden breathlessness with associated fever, cough, and chest pain.

3. Foreign Body Aspiration:

- A child may inhale a small object, such as a peanut or toy part, leading to obstruction of the airway. Sudden onset wheezing, coughing, and cyanosis may occur.

4. Croup:

- Croup, often caused by the parainfluenza virus, is characterized by a "barking" cough and stridor, typically worsening at night. It can lead to significant breathing difficulty due to upper airway obstruction.

5. Allergic Reactions:

- Anaphylaxis due to food, insect stings, or other allergens can cause sudden swelling of the airways, leading to difficulty breathing, wheezing, and hypotension.

6. Congenital Heart Disease:

- Conditions like tetralogy of Fallot or ventricular septal defect can present with acute breath-

lessness if there is a sudden worsening of heart function or if the child develops respiratory infections.

#### Quick Tip

Asthma and respiratory infections are the most common causes of sudden onset breathlessness in young children.

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### Q8. (b) Diagnosis and management of sudden onset breathlessness in a 3-year-old child.

#### Solution:

The diagnosis and management of sudden onset breathlessness in a 3-year-old child depend on identifying the underlying cause. The steps include:

#### 1. Diagnosis:

- Clinical History: Detailed history regarding onset, progression, associated symptoms (e.g., cough, fever, wheezing), and possible triggers (e.g., foreign body aspiration, asthma, viral infections).
- Physical Examination: Checking for signs of respiratory distress (tachypnea, use of accessory muscles, cyanosis), auscultating for wheezing or crackles, and palpating for signs of a foreign body.
- Imaging: A chest X-ray can help rule out pneumonia, foreign body aspiration, and other structural causes of breathlessness.
- Pulse Oximetry: Measuring oxygen saturation to assess the degree of hypoxia.
- Blood Tests: Complete blood count (CBC) to check for infection, and in some cases, arterial blood gases (ABG) to evaluate respiratory acidosis in severe cases.

#### 2. Management:

- Airway Management: Administer oxygen to maintain adequate oxygen saturation if hypoxic. In cases of foreign body aspiration, prompt removal by a healthcare provider is essential.
- Nebulized Bronchodilators: For conditions like asthma, albuterol nebulization helps relieve bronchospasm.
- Steroids: In cases of croup or severe wheezing, dexamethasone or prednisolone can help reduce inflammation in the airways.
- Antibiotics: For bacterial causes like pneumonia, empiric antibiotics (e.g., amoxicillin or cephalosporins) are administered.
- Epinephrine: In cases of anaphylaxis, immediate administration of epinephrine is crucial to reverse airway swelling and hypotension.
- Close Monitoring: In the case of severe or persistent breathlessness, hospitalization and close monitoring in an intensive care setting may be required.

### Quick Tip

A detailed clinical evaluation is key to diagnosing the underlying cause of sudden onset breathlessness in children, and early intervention with bronchodilators or corticosteroids may be crucial.

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## Q9. (a) Etiopathogenesis of Chronic Diarrhoea.

### Solution:

Chronic diarrhoea is defined as the passage of loose or watery stools for more than 4 weeks. It can result from a variety of underlying causes, including infections, inflammatory conditions, and metabolic disorders.

#### 1. Infectious Causes:

- Parasitic Infections: \*Giardia\*, \*Entamoeba histolytica\*, and \*Strongyloides\* are common parasitic infections that lead to chronic diarrhoea.
- Bacterial Infections: Infections with \*Salmonella\*, \*Campylobacter\*, \*Shigella\*, and \*Clostridium difficile\* can lead to prolonged diarrhoea, especially in immunocompromised individuals.
- Viral Infections: Some viruses, like rotavirus, can cause prolonged diarrhoea, particularly in children.

#### 2. Inflammatory Causes:

- Inflammatory Bowel Disease (IBD): Includes Crohn's disease and ulcerative colitis, which are chronic inflammatory conditions of the gastrointestinal tract that cause diarrhoea, along with abdominal pain and weight loss.
- Celiac Disease: An autoimmune disorder triggered by gluten that causes chronic diarrhoea, bloating, and malabsorption.
- Microscopic Colitis: A form of IBD that can cause chronic watery diarrhoea, often seen in older adults.

#### 3. Malabsorptive Disorders:

- Lactose Intolerance: Inability to digest lactose leads to diarrhoea after the consumption of dairy products.
- Pancreatic Insufficiency: In conditions like chronic pancreatitis, the lack of pancreatic enzymes results in malabsorption and chronic diarrhoea.

#### 4. Endocrine Causes:

- Hyperthyroidism: Increased thyroid hormone levels can cause increased gut motility, resulting in chronic diarrhoea.
- Diabetes Mellitus: Diabetic patients may suffer from autonomic neuropathy, which affects gut motility and leads to diarrhoea.

#### 5. Medications and Toxins:

- Antibiotics: Broad-spectrum antibiotics can disrupt the normal gut flora, leading to antibiotic-

associated diarrhoea or Clostridium difficile infections.

- Chemotherapy: Diarrhoea can be a side effect of chemotherapy.

6. Functional Disorders:

- Irritable Bowel Syndrome (IBS): This functional gastrointestinal disorder can cause chronic diarrhoea, often associated with abdominal cramping and bloating.

#### Quick Tip

Chronic diarrhoea requires a thorough workup to differentiate between infectious, inflammatory, malabsorptive, and functional causes.

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### Q9. (b) Management of Chronic Diarrhoea.

#### Solution:

The management of chronic diarrhoea depends on the underlying cause and aims to control symptoms, address the root cause, and improve the patient's quality of life.

1. General Measures:

- Dietary Modifications: A low-fat, easily digestible diet may be recommended for patients with malabsorption or inflammatory conditions. For lactose intolerance, a lactose-free diet should be initiated.
- Rehydration: Oral rehydration solutions (ORS) or intravenous fluids are essential to prevent dehydration, particularly in children and the elderly.

2. Pharmacological Treatment:

- Antidiarrhoeal Medications:
  - Loperamide: Reduces motility in cases of functional diarrhoea like IBS.
  - Bismuth subsalicylate: Can be used for symptomatic relief in cases of infection or irritation.
  - Octreotide: In cases of diarrhoea due to neuroendocrine tumors or short bowel syndrome, octreotide can reduce secretory diarrhoea.

3. Specific Treatment Based on Etiology:

- Infectious Causes:

- Antibiotics: If a bacterial infection like Salmonella or Shigella is identified, appropriate antibiotics (e.g., ciprofloxacin or azithromycin) should be prescribed.
- Antiparasitic Treatment: If parasitic infections such as Giardia are diagnosed, drugs like metronidazole or tinidazole are used.
- Antivirals: In viral cases like rotavirus or norovirus, supportive care and hydration are typically the mainstay of treatment.

- Inflammatory Causes:

- Corticosteroids: For Crohn's disease or ulcerative colitis, corticosteroids like prednisolone can reduce inflammation and control diarrhoea.

- Immunosuppressive Agents: Drugs like azathioprine, methotrexate, and biologics (e.g., TNF inhibitors) may be used for refractory inflammatory bowel disease.

- Pancreatic Insufficiency:

- Pancreatic Enzyme Replacement Therapy (PERT): For patients with chronic pancreatitis, enzyme supplements (e.g., Creon) are necessary to aid digestion and reduce diarrhoea.

4. Management of Complications:

- Electrolyte Imbalance: Monitoring and correcting electrolyte imbalances like hypokalemia or hyponatremia is important in chronic diarrhoea.

- Anemia: Iron supplementation may be necessary if chronic diarrhoea leads to malabsorption of nutrients.

5. Surgical Treatment:

- In severe cases where conservative management fails, especially in conditions like short bowel syndrome or Crohn's disease, surgical interventions like bowel resection or ileostomy may be required.

6. Psychosocial Support:

- Cognitive-behavioral therapy (CBT): For functional disorders like IBS, psychological support can help manage the stress and anxiety that contribute to diarrhoea symptoms.

#### Quick Tip

The management of chronic diarrhoea is multifaceted and depends on addressing the underlying cause, rehydration, and symptom control.

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### Q10. (a) Etiopathogenesis and types of Periodic Paralysis.

#### Solution:

Periodic paralysis is a group of disorders characterized by recurrent episodes of muscle weakness or paralysis, often triggered by specific factors such as potassium fluctuations, stress, or exercise. The key points about the etiopathogenesis and types of periodic paralysis are:

1. Etiopathogenesis:

- Genetic mutations: Periodic paralysis is primarily caused by autosomal dominant genetic mutations that affect the ion channels, leading to improper ion transport across muscle cell membranes. The most common mutations are found in the SCN4A gene, which encodes the sodium channel, and the CACNA1S gene, which encodes the calcium channel.

- Impaired ion exchange: Disruption in the movement of sodium, potassium, or calcium ions leads to improper muscle depolarization, resulting in episodes of weakness or paralysis.

- Electrolyte disturbances: Changes in the levels of potassium, sodium, or calcium in the blood are major triggers for attacks.

## 2. Types of Periodic Paralysis:

- Hypokalemic Periodic Paralysis: Characterized by episodes of muscle weakness associated with low levels of potassium in the blood. Attacks can be triggered by stress, exercise, or high carbohydrate meals.
- Hyperkalemic Periodic Paralysis: Characterized by episodes of muscle weakness or paralysis due to high potassium levels in the blood. These attacks can occur spontaneously or be triggered by factors like rest after exercise.
- Thyrotoxic Periodic Paralysis: A form of periodic paralysis that occurs in patients with hyperthyroidism, often presenting as sudden-onset paralysis associated with low potassium levels.

### Quick Tip

Periodic paralysis is often triggered by changes in potassium levels or thyroid dysfunction. Genetic testing can help confirm the diagnosis.

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## Q10. (b) Clinical features of Periodic Paralysis.

### Solution:

The clinical features of periodic paralysis include:

#### 1. Muscle Weakness:

- The hallmark of periodic paralysis is muscle weakness or paralysis that comes on suddenly, typically lasting from hours to days. It is often bilateral, affecting both the arms and legs.
- Weakness may start in one area (e.g., the legs) and then progress to involve other muscle groups.

#### 2. Triggers:

- Attacks are often triggered by certain factors, including:
- Exercise: After strenuous physical activity, especially in the morning after sleep.
- Dietary factors: Consumption of large meals, especially those high in carbohydrates, can trigger episodes in some individuals.
- Stress: Physical or emotional stress can be a common trigger.
- Cold temperatures: Exposure to cold weather can provoke episodes of weakness.

#### 3. Duration and Frequency:

- The episodes can last from a few hours to several days, with some individuals experiencing attacks multiple times a week, while others may only have them infrequently.

#### 4. Potassium Levels:

- Hypokalemic Periodic Paralysis: Associated with low potassium levels during attacks. Serum potassium levels may fall to dangerously low levels.
- Hyperkalemic Periodic Paralysis: Associated with high potassium levels during attacks. Potas-

sium levels may rise above normal ranges, often precipitating weakness.

5. Recovery:

- Symptoms of paralysis improve with rest, and potassium supplementation or intravenous potassium may accelerate recovery during hypokalemic attacks.

**Quick Tip**

Periodic paralysis can present with sudden, reversible episodes of muscle weakness. Potassium levels are often altered during attacks, making it important to monitor and correct them.

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**Q10. (c) Management of Periodic Paralysis.**

**Solution:**

Management of periodic paralysis aims to prevent and treat attacks, as well as manage the underlying condition. The approach includes:

1. Pharmacological Treatment:

- Potassium Supplements: In hypokalemic periodic paralysis, potassium supplementation during an attack can help restore muscle function. Oral potassium chloride or intravenous potassium can be used.
- Acetazolamide: This carbonic anhydrase inhibitor is sometimes used to prevent attacks, as it helps to stabilize potassium levels.
- Bicarbonates: In cases of hyperkalemic paralysis, sodium bicarbonate can be administered to help lower potassium levels.
- Thyroid Management: If thyrotoxic periodic paralysis is diagnosed, controlling the thyroid disorder with antithyroid medications (e.g., methimazole) or iodine therapy is essential.

2. Lifestyle Modifications:

- Avoiding Triggers: Identifying and avoiding known triggers, such as strenuous exercise, stress, or large carbohydrate meals, can help prevent attacks.
- Dietary Changes: A low-carbohydrate diet may be recommended in some cases to prevent episodes. Potassium-rich foods should be included for individuals with hypokalemic periodic paralysis.

3. Genetic Counseling:

- Genetic testing can identify specific mutations (e.g., SCN4A gene) that cause periodic paralysis, allowing for better management and familial screening. Genetic counseling is important for affected individuals and their families.

4. Emergency Management:

- In severe cases, particularly with hyperkalemic periodic paralysis, urgent medical intervention

may be required to stabilize potassium levels and prevent life-threatening complications such as cardiac arrhythmias.

**Quick Tip**

Management of periodic paralysis requires addressing the underlying cause, correcting potassium imbalances, and lifestyle modifications to reduce triggers.

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