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OFFICIAL JOURNAL OF THE PAEDIATRIC ASSOCIATION OF NIGERIA



CLINICAL QUIZ

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Clinical Case Scenario

A 12-year-old Nigerian girl presented at the Paediatric Gastroenterology Clinic following two episodes of haematemesis and persistent melaena. She had an upper GI endoscopy procedure and thereafter did not return to the hospital for further care due to financial handicap. She presented nine months later with recurrent haematemesis, easy fatigability, and lower limb swelling over the preceding three months. There was associated progressive exertional dyspnoea of recent onset. On physical examination, she was pale, lethargic, and had bilateral pitting pedal oedema up to the upper third of the legs. The pulse rate was 100 bpm, the respiratory rate was 34 cycles per minute, and there was a loud pulmonic component of the second heart sound. The finding on upper GI endoscopy is shown below:



Part 1: Multiple-Choice Questions (Best-of-Four)

- Which of the following will not explain the underlying cause of the findings in upper GI endoscopy?
 - Peptic ulcer disease
 - Umbilical vein catheterisation
 - Chronic liver diseases
 - Protein C deficiency
- Based on the findings from the child's second presentation to the hospital, what is the most likely unifying cause of her condition?
 - Congenital heart disease
 - Hepato-renal syndrome
 - Portopulmonary hypertension
 - Pulmonary embolism
- If this child presented with splenomegaly without hepatomegaly, jaundice, signs of chronic liver disease, or ascites, along with entirely normal serum albumin, total bilirubin, and prothrombin time (INR: 1.0), which aetiological factor will explain the clinical scenario?
 - Biliary atresia
 - Idiopathic neonatal hepatitis
 - Budd-Chiari syndrome
 - Portal vein thrombosis
- In the index case presenting with haematemesis, which intravenous medication should be administered immediately to control the active bleeding?
 - Ranitidine
 - Octreotide
 - Omeprazole
 - Propranolol

5. What is the standard first-line management for the findings identified in this patient?
- Endoscopic band ligation
 - Surgical portocaval shunt
 - Liver transplant
 - Immediate splenectomy

Answer key and detailed explanations

Question 1

Correct answer: A

It is important to understand that the findings from endoscopy indicate the presence of oesophageal varices. In a child with endoscopic evidence of oesophageal varices, upper gastrointestinal bleeding is attributed to these varices rather than to peptic ulcer disease. Bleeding from varices is characterised as a high-volume, low-pressure venous haemorrhage, which occurs due to elevated pressure in the portal vein (portal hypertension). This condition can arise from umbilical catheterisation, chronic liver disease, or protein C deficiency.

Umbilical catheterisation is a common cause of extrahepatic portal vein obstruction (EHPVO) in children. If a neonatal umbilical catheter is left in place for too long, is improperly positioned, or becomes infected (resulting in omphalitis or sepsis), it can lead to mechanical endothelial injury and trigger localised clot formation. This results in portal vein thrombosis (PVT), which blocks blood flow to the liver. Over time, the body develops a network of bypass vessels (known as a portal cavernoma). However, the increased pressure can cause backflow into the splenic and oesophageal veins, leading to the formation of large, fragile varices that may present as silent gastrointestinal bleeding during later childhood.

In cases of chronic liver disease, such as biliary atresia, scar tissue can compress the normal hepatic sinusoids, increasing resistance to portal blood flow. This situation diverts blood away from the liver and into systemic circulation through porto-systemic collateral networks. This results in the dilation of the oesophageal venous plexus.

Protein C is an essential natural anticoagulant that inactivates clotting factors Va and VIIIa. It requires Protein S as a cofactor to neutralise these factors effectively. A deficiency of these proteins, whether congenital or acquired, can cause the body to enter a hypercoagulable state, increasing the risk of spontaneous thrombosis within major visceral vessels,

particularly leading to thrombus formation in the main portal vein trunk.

Question 2

Correct answer: C

The child was initially scheduled for banding of oesophageal varices but missed the appointment due to financial reasons. Upon her subsequent presentation, she exhibited signs of right ventricular heart failure, which included easy fatigue, lower extremity oedema, exertional dyspnoea, and a pronounced pulmonic component of the second heart sound. The elevated pressure in the portal venous system triggers high blood pressure in the arteries of the lungs. Right heart failure is due to strain on the right ventricle due to increased pulmonary pressure. Given her history of oesophageal varices, combined with a recent upper gastrointestinal bleed and indications of pulmonary hypertension, the most likely diagnosis is Portopulmonary Hypertension (PoPH).

Hepatorenal syndrome (HRS) is a life-threatening complication associated with advanced liver disease. The portal hypertension stemming from liver disease leads to the release of vasodilators such as nitric oxide. This causes blood to pool in the gut circulation, reducing blood flow to the kidneys. In response, vasoconstriction occurs in the kidneys' circulation, resulting in functional renal failure without a primary kidney disease. The symptoms of HRS can include decreased urine output and mental confusion.

Pulmonary embolism typically occurs suddenly and is characterised by shortness of breath, pleuritic chest pain, and haemoptysis. It occurs when a blood clot or other material blocks a major artery in the lungs, usually after dislodgement from the deep veins (deep vein thrombosis) and travelling to the heart and lungs.

Question 3

Correct answer: D

PVT can cause EHPVO, which aligns perfectly with the clinical presentation in this child. When a blood clot blocks the extrahepatic portal vein, it causes presinusoidal portal hypertension. Since the obstruction occurs before the blood enters the liver, the liver's architecture and hepatocytes remain healthy and functional. This upstream backup of venous pressure congests the splenic vein, leading to an enlargement of the spleen.

In cases of extrahepatic PVT, the high pressure is strictly presinusoidal, and albumin levels remain normal, making ascites highly unlikely. In contrast, conditions such as biliary atresia and neonatal hepatitis can progress to severe intrahepatic biliary cirrhosis, resulting in high sinusoidal pressure and low serum albumin levels. Budd-Chiari syndrome occurs when thrombi or structural webs block blood flow exiting the liver, causing blood to pool back into the liver tissue. This pooling creates massive congestion, cellular ischemia, and post-sinusoidal portal hypertension.

Question 4

Correct answer: B

The first-line intravenous medication in a case of active variceal bleeding is Octreotide. Octreotide is a synthetic analogue of somatostatin that selectively constricts the splanchnic (mesenteric) arterial bed. This action significantly reduces blood flow into the portal venous system, which helps to lower the pressure in the oesophageal varices and control active bleeding.

Propranolol has no role in the acute phase of active variceal bleeding and is contraindicated during an acute haemorrhage. However, it is used for long-term prevention of variceal bleeding in adults. While not officially approved for this purpose in children, paediatric gastroenterologists often prescribe propranolol empirically for secondary prophylaxis against variceal bleeding.

Omeprazole, a proton pump inhibitor, is also an important secondary supportive medication in the management of variceal bleeding, but it cannot stop acute variceal bleeding on its own. Ranitidine, like omeprazole, suppresses acid secretion but is also unable to alter portal vein pressures or stop active variceal bleeding independently.

Question 5

Correct answer: A

The standard first-line management for oesophageal varices depends on whether the patient is experiencing an acute haemorrhagic emergency or requires long-

term prevention (prophylaxis). For a patient actively vomiting blood or who is hemodynamically unstable due to ruptured varices, immediate management involves a three-part approach: resuscitation, vasoactive medications, and endoscopic intervention. Endoscopic variceal ligation (EVL) is the gold-standard first-line mechanical treatment for bleeding vessels. A surgical portocaval shunt is a definitive but highly invasive procedure that should not be included in the immediate first-line management of acute variceal bleeding. Additionally, performing an immediate splenectomy is strongly contraindicated and should never be done as an emergency treatment for acute variceal bleeding. Splenomegaly is a symptom of portal hypertension, not its cause, and removing the spleen does nothing to reduce the dangerously high blood pressure within the portal vein or to stop the bleeding of oesophageal varices.

While a liver transplant is a definitive cure for oesophageal varices, it is reserved strictly for patients with advanced, end-stage liver cirrhosis or acute liver failure.

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