

Adolescent-Onset Extrahepatic Portal Vein Obstruction with Gamna-Gandy Bodies: A Case Report

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Abstract

Extra hepatic portal vein obstruction is the leading cause of portal hypertension in children in India and presents usually within first ten years of life. The presentation in adolescence is often less described. This is a case of a 14-year-old adolescent with no risk factors presenting with flank pain and hematemesis whose examination showed pallor and massive splenomegaly with no features of chronic liver disease. Investigations showed severe anaemia requiring transfusion, thrombocytopenia consistent with hypersplenism. Upper gastroscopy revealed oesophageal varices requiring banding. An adolescent with haematemesis and a large spleen needs evaluation for portal hypertension even without a risk factor.

Keywords: Extrahepatic portal vein obstruction; hypersplenism; haematemesis; adolescent

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Introduction

Extrahepatic portal vein obstruction is a vascular disease of liver where portal vein is blocked with or without involvement of hepatic branches, splenic or mesenteric branches. With time the occluded segment is bypassed by collateral channels, the portal cavernoma [1]. It is the most common cause of portal hypertension in India, whereas it is cirrhosis in western countries [2].

Typically, a child presents with bleeding within 10 years of age with umbilical catheterisation, neonatal sepsis, dehydration as triggers [3]. The distortion of the bile ducts and formation of collaterals in their walls named portal cavernoma cholangiopathy [4], and leave small iron-laden scars in the congested spleen, the Gamna-Gandy bodies, which look distinctive on MRI [5]. This is a case of a 14-year-old female diagnosed at adolescent age with no predisposing risk factors.

Case Presentation

History

A 14-year-old girl, second order birth to non-

consanguineous parents, was admitted with 5–6 days of dull, intermittent pain over both flanks, worse on the left and immediately after eating, which had blunted her appetite. She had one episode of massive hematemesis on the day of admission. She had one day of low-grade fever the week before. She had similar complaints six months ago which resolved spontaneously after symptomatic treatment. There was no melaena, pale stool, itching, weight loss, swelling, abdominal distension or bleeding elsewhere, and no drug exposure. Yellow eyes and dark urine appeared only after admission. She was born at term by vaginal delivery weighing 3.5 kg and needed no neonatal intensive care, so umbilical catheterisation had not occurred. Development, schooling and immunisation up to five years were appropriate. Menarche was at 14 years with normal cycles. Her daily intake was about 1500 kcal and 34 g of protein, well below requirement.

Examination

She was alert and haemodynamically stable (pulse 92/min, blood pressure 106/64 mmHg, SpO₂ 96% on room air). Height (160.5 cm) and weight (50 kg) lay between the 50th and 90th centiles, with a BMI of 19.4 kg/m². She was markedly pale and mildly icteric, with knuckle pigmentation, but had no clubbing, oedema, spider naevi, palmar erythema, flap or Kayser–Fleischer rings.

The abdomen was not distended with no visible veins. A firm, smooth, non-tender liver was just palpable (span 7 cm) and the spleen reached the umbilicus. There was no ascites or bruit. Other systems were normal, and sexual maturity rating was 5.

Investigations

Admission haemoglobin was 6.4 g/dL later falling to 5.2 g/dL requiring transfusion; leucocytes dropped from 4,790 to 2,380/μL and platelets stayed between 52,000 and 82,000/μL (Table 1). Iron studies confirmed deficiency (transferrin saturation 10.3%, ferritin 23.4 ng/mL) with normal vitamin B12. INR was 1.33 and albumin 3.4 g/dL. Repeat liver tests showed bilirubin 1.9 mg/dL (direct 1.0), ALT 103 U/L and alkaline phosphatase 267 U/L. Screens for malaria, dengue, HIV, hepatitis B and hepatitis C were negative.

FibroScan was done showing a liver stiffness (fibrosis) of 8.0 kPa and a controlled attenuation parameter of 246 dB/m showing grade 1 fatty liver. Endoscopy showed grade 2 varices in oesophagus (Figure 1) requiring banding; Other parameters were normal. MRCP showed absent portal flow voids, with collaterals at the porta, bile duct and gallbladder, splenic hilum and gastro-oesophageal junction. The common hepatic (4.7 mm) and common bile (5.2 mm) showed extrinsic narrowing with mild intrahepatic dilatation. The gallbladder wall was oedematous without stones. The spleen was enlarged with T2 dark foci.

Diagnosis, management and follow-up

EHPVO was diagnosed with portal cavernoma, hypersplenism, Gamna–Gandy bodies, portal cavernoma cholangiopathy and iron-deficiency anaemia; preserved synthesis, absent ascites and low stiffness argued against cirrhosis. She was admitted to paediatric intensive care unit where she received an octreotide infusion, intravenous antibiotics, a proton-pump inhibitor and 2 units of packed red cells. Once stable, two bands were applied with a multiband ligator. She resumed liquids four hours later and shifted to ward on antibiotics, pantoprazole 40 mg twice daily, sucralfate 10 mL four times daily and carvedilol. She had no episodes of hematemesis further and was discharged on iron and folate supplements and being followed by pediatric gastroenterologist.

Table 1. Laboratory findings

Parameter	Result	Reference range

Haemoglobin (g/dL)	6.4	12–15
Haematocrit (%)	20.3	36–46
MCV (fL) / MCH (pg)	55 / 17.3	78–95 / 26–32
RDW (%)	21.6	11.5–14.5
Total leucocyte count (/μL)	4,790 (lowest 2,380)	4,000–11,000
Platelets (/μL)	62,000 (range 52,000–82,000)	150,000–450,000
Reticulocyte count (%)	2.3	0.5–2.5
Serum iron (μg/dL) / TIBC (μg/dL)	26 / 252	50–170 / 250–450
Transferrin saturation (%)	10.3	20–50
Serum ferritin (ng/mL)	23.4	[lab range]
Vitamin B12 (pg/mL)	683	200–900
PT (s) / INR	15.6 / 1.33	[lab range] / 0.8–1.2
Total protein / albumin (g/dL)	6.1 / 3.4	6.4–8.3 / 3.5–5.2
Bilirubin total / direct (mg/dL) — admission	1.3 / 0.5	0.2–1.2 / 0.1–1.0
Bilirubin total / direct (mg/dL) — repeat	1.9 / 1.0	0.2–1.2 / 0.1–1.0
AST / ALT (U/L) — admission	32 / 26	8–48 / 7–55

AST / ALT (U/L) — repeat	69 / 103	8–48 / 7–55
Alkaline phosphatase (U/L) — admission / repeat	91 / 267	[age-specific range]
GGT (U/L)	11	[lab range]
Urea / creatinine (mg/dL)	18 / 0.5	8–23 / 0.6–1.35
C-reactive protein (mg/L)	4.04	<5
Liver stiffness (kPa) / CAP (dB/m)	8.0 / 246	<7.0 / <240
Malaria, dengue, HIV, HBsAg, anti-HCV	Negative	—

MCV, mean corpuscular volume; MCH, mean corpuscular haemoglobin; RDW, red cell distribution width; TIBC, total iron-binding capacity; PT, prothrombin time; INR, international normalised ratio; AST, aspartate aminotransferase; ALT, alanine aminotransferase; GGT, gamma-glutamyl transferase; CAP, controlled attenuation parameter.

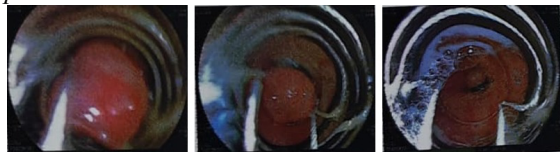


Figure 1. Endoscopic variceal ligation. Oesophageal varices with red colour signs drawn into the ligator cap (left), and ligated varices with bands deployed (centre and right). [Replace with original high-resolution endoscopic images before submission.]

Discussion

This case is prompted for discussion emphasising that she had no risk factors, was physiologically well with near normal liver function tests which could easily be misdiagnosed as gastritis. Doppler and endoscopy are helpful in differentiating the portal hypertension [7]. Diagnosing and separating extrahepatic portal vein obstruction and cirrhosis helps in prognosis and treatment of the patient [8] which is seen in fibroscan of the liver; The collaterals around bile duct explains duct being narrowed. The thick gallbladder wall most likely represents congestion rather than cholecystitis.

Gamna–Gandy bodies indicate prolonged splenic congestion.

Her anaemia combined blood loss, hypersplenism and poor intake, and the bulky spleen pressing on the stomach probably caused the post-meal pain.

The red colour signs made these varices high risk. Banding is preferred to sclerotherapy in children with EHPVO [9], but it does not relieve hypersplenism or biliopathy. A meso-Rex bypass, or a distal splenorenal shunt when the Rex recess is unsuitable, offers definitive decompression [10]. Her massive bleeding, large spleen and early biliopathy justify early surgical referral.

Conclusion

This case emphasises on the recognition of extra hepatic portal vein obstruction declaring in adolescents without any risk factors which are under described. It even documents long standing features like Gamna-Gandy bodies which reveals late diagnosis of the patient. The reports of fibroscan and MRCP add to the diagnosis and help avoid common misdiagnoses. The above case is worth mentioning since adolescent reports regarding EHPVO are rarely mentioned.

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