Mesenteric ischemia and portal hypertension caused by splenic arteriovenous fistula

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Splenic arteriovenous fistula is rare and usually presents with features of established portal hypertension (PHT). Presentation as acute mesenteric ischemia with features of acute PHT is uncommon. We report a 35-year-old lady who presented with severe abdominal pain, diarrhea and ascites, which was found to result from mesenteric ischemia and acute PHT secondary to splenic arteriovenous fistula. She underwent resection of fistula, which resulted in complete symptom relief. [Indian J Gastroenterol 2004;23:184-185]

Key words: Ascites

Splenic arteriovenous fistula (SAVF) is a rare cause of forward portal hypertension (PHT). Its presentation as mesenteric ischemia is unusual.

A 35-year-old lady presented with abdominal pain and diarrhea followed by frank lower gastrointestinal bleeding and abdominal distension. The pain was insidious in onset, diffuse, severe, and progressively increasing over 2 weeks. Repeated intake of non-steroidal anti-inflammatory drugs (NSAIDs) did not bring relief. Diarrhea was 8-10 times a day, initially watery stool followed by frank bloody stools. She had lost 10% of her body weight in 1 month. There was no history of anorexia, liver disease, embolic phenomenon or a hypercoagulable state. She was multiparous and had had 5 full-term vaginal deliveries, the last one being 14 months back.

On examination, she was poorly nourished, with tachycardia and blood pressure of 120/80 mmHg. There was moderate pallor but no signs of liver disease. The abdomen was distended, tense and tender with no guarding. There were prominent upper abdominal pulsations, ascites and sluggish bowel sounds. Rest of the examination was unremarkable.

Investigations: hemoglobin 6.8 g/dL, leukocyte count 7000/ mm³ and platelet count 3x10⁴/mm³. Liver and renal function tests, and screening coagulogram were normal. Ascitic fluid had high serum-ascitic fluid albumin gradient (2 g), with 100 polymorphs/mm³, but was sterile. Abdominal X-ray showed distended and thickened bowel loops with air-fluid levels. Endoscopy showed gastric erosions but no varices. Ultrasonography revealed dilated portal vein (25 mm at porta) and splenic vein (16 mm) and ascites. Spleen and liver were normal. CT scan confirmed these findings, showed splenic artery calcification, and a normal superior mesenteric artery and vein. Bowel loops were distended and thickened. During CT scan early opacification of the portal vein was noticed even before enhancement of spleen. This provided a clue to the existence of an arteriovenous fistula in the portal circuit. Re-evaluation of the patient revealed a machinery bruit in the left upper abdomen. Angiography revealed a dilated tortuous splenic artery with a high-flow SAVF (Fig) with evidence of mesenteric steal phenomenon.

Balloon occlusion was unsuccessful due to a high-flow state. At laparotomy, there was ascites, congested ischemic bowel, dilated splenic artery and vein (5 cm), a palpable bruit in the splenic artery, atrophic pancreas and an SAVF at the hilum. The bowel perfusion improved dramatically on clamping the splenic artery. The spleen with AV fistula were resected. Histology confirmed the presence of an AV fistula, with intimal and medial myxoid degeneration and calcification of the splenic artery with a fibrocongestive spleen. Postoperative recovery was uneventful except for transient elevation of transaminases due to hepatic ischemia secondary to sudden drastic reduction in portal blood flow. She is asymptomatic and has gained 10 Kg weight over the 2 months post-surgery.

SAVF can be congenital or acquired. Congenital aneurysms that occur in the Osler-Weber-Rendu and Ehlers-Danlos syndrome are intraspinal, multiple and intractable. Acquired fistulas are secondary to trauma, aneurysmal rupture or rarely spontaneous. Splenic aneurysms are especially common among multiparous women due to myxoid degeneration of the artery in the presence of a hyperdynamic portal circulation during pregnancies. Increased intra-abdominal pressure during delivery promotes their rupture into the free peritoneum, lesser sac, adjacent viscera or rarely into the splenic artery.

PHT in SAVF is secondary to hyperdynamic circulation and is associated with a hepatopetal flow. These patients manifest with varices, splenomegaly, ascites and diarrhea. As PHT was acute in our patient, varices were absent. Diarrhea is of secretory nature. It occurs due to a combination of acute PHT and mesenteric ischemia. Compensation occurs later by increasing lymphatic flow and reflux arteriolar vasoconstriction.

In our patient, mucosal ischemia due to steal phenomenon manifested as pain, bleeding, diarrhea and weight loss. It was accentuated by the use of NSAIDs, which inhibited the mucosal vasodilatory prostaglandins. NSAIDs-induced platelet dysfunction may have accentuated the bleed.

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Case Snippets

Intestinal metastases from lung cancers are uncommon and duodenal metastases are rare. The commonest manifestations of intestinal metastases are perforation or obstruction; frank bleeding due to metastases has been reported only occasionally. Squamous cell cancer of the lung causing biliary as well as duodenal obstruction due to metastatic deposits in the ampullary region and its endoscopic management has not been reported earlier.

A 65-year-old gentleman was admitted with history of tarry stools for one day, followed by hematemesis. Five months earlier, he was diagnosed to have squamous cell carcinoma of the lung and had received chemotherapy. The patient was pale, pulse was 96/min, blood pressure was normal. There was no icterus or edema. Hemoglobin was 9 g/dL and PCV was 27.

Serum urea, bilirubin, transaminases, alkaline phosphatase, amylase were normal. Upper GI endoscopy revealed blood in the stomach. There was a bulky fleshy mass in the distal first part of the duodenum with fresh bleeding from its superior aspect. Injection of 1:10,000 epinephrine resulted in stoppage of the active bleeding. Re-look endoscopy on the next day did not show any bleeding. Biopsies were obtained from the mass. Histological examination of the tissue revealed distorted duodenal crypts with erosion of the surface epithelium at places. Small clumps of malignant squamous epithelial cells were observed in lymphatic and vascular channels (Fig).

A month later, he was readmitted with jaundice and itching since ten days. Examination revealed slight wasting with pallor and icterus. Scratch marks were seen all over the body. There was no lymphadenopathy. The gall bladder was palpable and was cystic in consistency. Hemoglobin was 9.5 g/dL, serum bilirubin 188.1 mmol/L, alkaline phosphatase 664 U/L, AST and prothrombin time were normal. Ultrasonography revealed dilatation of the intrahepatic biliary radicals and the common bile duct (10 mm at the lower end). The pancreatic duct measured 7 mm. No calculus was seen could be appreciated in the bile duct, parenchyma of the pancreas or the duodenum.

At ERCP, the metastatic mass in the duodenum had grown and was now also occupying the area in and around the papilla of Vater. The patient could not afford a metal stent and therefore two 10 Fr straight Cotton Leung plastic stents (Wilson Cook, Winston Salem, NC) were placed in the common bile duct. The patient became asymptomatic in three weeks. Clinically icterus was not appreciable and the gall bladder was no more palpable. Serum bilirubin was 27.4 mmol/L and alkaline phosphatase 122 U/L.

Fig: Histology of duodenal metastases. Note malignant squamous cells in lymphatic channels and capillaries in lamina propria. Crypts of mucosa are distorted, with erosion of surface epithelium (H & E, 80 x)

Duodenal metastases from squamous cell carcinoma of the lung: endoscopic management of bleeding and biliary and duodenal obstruction

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Intestinal metastases from cancer of the lung are rare. We report a 65-year-old man with duodenal metastases from squamous cell cancer causing GI bleeding and biliary and duodenal obstruction; these were managed endoscopically. [Indian J Gastroenterol 2004;23:185-186]

Key words: Gastrointestinal bleed, lung cancer, metastasis