Reversible portal hypertension due to tuberculosis

USHA DUTTA, VIKAS BHUTANI, BIRENDER NAGI, KARTAR SINGH

Department of Gastroenterology, Postgraduate Institute of Medical Education and Research, Chandigarh 160 012

Presentation of abdominal tuberculosis with portal hypertension is rare. We report a 25-year-old man with portal hypertension due to compression of the portal vein by tuberculous lymph nodes at the hepatic hilum. After antitubercular therapy, features of portal hypertension disappeared as the nodes regressed. [Indian J Gastroenterol 2000;19:136-137]

Key words: Liver; hilum, lymph nodes

Presentation of abdominal tuberculosis as portal hypertension is rare. We report a patient with abdominal tuberculosis who had portal hypertension due to lymph nodes at the porta compressing the porta vein.

A 25-year-old man presented with a 3-year-long history of intermittent fever, and epigastric pain, anorexia, weight loss and increasing abdominal distension for three months. There was no history of jaundice or pedal edema. On examination, he was febrile. There was no icterus, peripheral lymphadenopathy or evidence of chronic liver disease. Abdominal examination revealed splenomegaly of 3 cm and moderate ascites. The liver span was 14 cm.

Investigations: Hemoglobin 10.5 g/dL, leukocyte count 8400/mm³, serum bilirubin 0.7 mg/dL, AST 15 IU/L and ALT 9 IU/L, serum alkaline phosphatase 12 KAU, total serum proteins 5.7 g/dL (albumin 3.6). Coagulation parameters and platelet counts were normal. Ascitic fluid analysis revealed proteins 2.6 g/dL (albumin 1.6), serum ascitic fluid albumin gradient 1.2, leukocyte count 150 cells/mm³ with >90% lymphocytes. The ascitic fluid adenosine deaminase level was 37 units/L (normal <32).

Ultrasoundography and computerized tomography revealed dilated (13 mm) portal vein, splenomegaly, ascites, minimal left-sided pleural effusion and thickening of the greater omentum. Multiple lymph nodes with central hypodense areas suggestive of necrosis were seen at the porta, subpyloric and peripancreatic regions, lower retroperitoneum and mesentery. The portal vein was compressed by lymph nodes at the porta; there was no portal vein thrombosis. Upper gastrointestinal endoscopy revealed 3 columns of grade II esophageal varices, gastric varices and a duodenal varix. Ultrasound-guided fine-needle aspiration cytology from the retroperitoneal lymph nodes revealed epithelioid cell granulomas, positive for acid-fast bacilli. Markers for hepatitis B, hepatitis C, Wilson’s disease and autoimmune liver disease were negative.

The patient was started on antitubercular therapy (isoniazid, rifampicin, ethambutol and pyrazinamide) for 2 months followed by isoniazid and rifampicin for another 9 months. He showed clinical improvement in the form of weight gain (4 kg), improved appetite and regression of ascites and splenomegaly. Monthly ultrasonography over the first three months of therapy showed regresssing splenomegaly and decreasing portal vein dimension; there was decrease in size, followed by disappearance of the lymph nodes. Endoscopy showed complete disappearance of varices by 4 months. Three months after completion of therapy, the patient is asymptomatic; ultrasonography and endoscopy are normal.

The common lymph node groups involved in abdominal tuberculosis are the mesenteric, followed by the ileocecal and pyloroduodenal groups. Involvement of lymph nodes at the porta hepatis is exceptional.

Our patient had features of portal hypertension due to compression of the portal vein by enlarged lymph nodes at the hepatic hilum. The suggested mechanisms of portal hypertension in tuberculosis are: splenic vein thrombosis due to pancreatic tuberculosis or retroperitoneal tubercular abscess, portal vein thrombosis secondary to lymph nodes, hepatic tuberculosis causing sinusoidal compression, and hepatic outflow obstruction due to tuberculotic constrictive pericarditis.

There are only two case reports of lymph node compression at the porta causing portal hypertension. Interestingly, all features of portal hypertension regressed completely with antitubercular drugs; this has not been reported earlier.

Thus, tuberculous lymph nodes at the hepatic hilum may be a cause of portal hypertension.

References

5. Diab S, Abu Nema T, Abu Zidan F. Portal hypertension
Primary hypertrophic colonopathy

M JANAKI, K P A CHANDRASEKHAR, G MALLIKARJUNA RAO,* S V RANGA REDDY*

Departments of Pathology and Surgery, Kurnool Medical College/ Govt General Hospital, Kurnool 518 002, AP

We report a 45-year-old man and a 60-year-old woman who presented with features of intermittent intestinal obstruction. Barium enema revealed narrowing at the pelvic-rectal junction in the man, and from the pelvic colon to the anal verge in the woman. Histology of the resected sections showed marked hypertrophy of the muscularis propria in both cases, with normal mucosa, submucosa and myenteric plexus. Both patients are asymptomatic at 4 years' and 2 years' follow up. This entity of primary hypertrophic colonopathy may be a variant of primary visceral myopathy. [Indian J Gastroenterol 2000; 19:137]

Key words: Chronic idiopathic intestinal pseudo-obstruction, colonic myopathy

Chronic intestinal pseudo-obstruction, usually occurs due to involvement of the small bowel. The esophagus and colon are rarely involved in this primary myopathy. We report a rare entity of primary hypertrophic colonopathy as a cause of pseudo-obstruction.

A 45-year-old man presented with pain and distention of the abdomen since 4 days. He had recurrent attacks of pain and distention since 4 years. Systemic examination was normal. Emergency laparotomy revealed massive distension of the colon with an area of narrowing at the pelvis-rectal junction. Proximal colostomy was done. Barium enema examination revealed narrowing at the pelvis-rectal junction with apple-core sign. Local resection of the constricted portion with end-to-end anastomosis of the colon was done. The proximal colostomy was closed in due course. Follow up for 4 years was uneventful. The resected specimen, measuring 2 cm x 2 cm with a button-hole narrow lumen, was submitted for histopathological examination.

A 60-year-old woman presented with difficulty in passing stools since 1 year. Past history and family history were not significant. Systemic examination was normal. Per rectal examination revealed an annular firm mass at the ano-rectal junction, admitting the tip of the index finger. Edge biopsy of the mass was inconclusive. Barium enema examination revealed narrowing of the lumen from the pelvic colon to the anal verge.

with proximal dilatation. Abdomino-perineal resection with end colostomy was done. Follow up for 2 years was uneventful. The resected specimen, measuring 30 cm from the distal part of the descending colon, sigmoid colon, rectum and the anal canal, showing narrowed lumen with proximal dilated thin wall of colon, was submitted for histological examination.

Histology of both the specimens revealed normal colonic mucosa and submucosa. The muscle coat showed marked hypertrophy (Fig). The myenteric plexus was normal. There was no evidence of malignancy or any other pathological changes.

Primary hypertrophic colonic stenosis, with marked hypertrophy of the muscle coat of the colon causing intestinal pseudo-obstruction, is very rare. Myopathies of the gastro-intestinal tract usually produce atrophy and fibrosis, and rarely hypertrophy of the muscle coat. In most cases with muscular hypertrophy, an abnormal myenteric plexus leads to disordered muscle function and smooth muscle hypertrophy. Usually, the small bowel is involved, though the esophagus and colon may also be affected.

We report two patients with hypertrophic colonopathy causing colonic pseudo-obstruction, where no cause was found for the muscular hypertrophy.

References