Case Snippets

Fig: Colonoscopy showing hepatic flexure varices

edema, and denied ingestion of non-steroidal anti-inflammatory
drugs. On examination he was pale, but hemodynamically
stable. There were no stigmata of chronic liver disease. Mild
hepatom egaly was noticed.

Laboratory parameters showed hemoglobin of 7.9 g/dL.
Liver and renal biochemistry, and coagulation parameters were
normal. Ultrasonography of abdomen was normal. Upper gas-
trointestinal endoscopy showed small gastric fundal arterio-
venous malformation without active bleeding; there were no
esophageal or gastric varices or ulcers. The day after ad-
mission, the patient had another episode of melena. Colonoscopy
showed large varices in the hepatic flexure of the colon (Fig);
the rest of the colon was normal. No endoscopic intervention
was done. Assuming the source of bleeding was the gastric
arteriovenous malformation (there were no signs of recent hem-
orrhage on the varices or the gastric lesions), sclerosing agent
was injected into it. Color Doppler study of hepatic veins, portal
vein and inferior vena cava revealed normal flow pattern.
Liver biopsy showed fatty liver. There was no further
bleeding after follow up of 8 months.

The prevalence of colonic varices, mainly rectal
varices, in patients with portal hypertension varies from
4%-89%.1,2 In the absence of portal hypertension and other
obvious causative factors, colonic varices are classified as 'idiopathic'. Six of the reported patients
with idiopathic colonic varices in literature had similar
varices in other family members.3,4 Our patient had small
fundal arteriovenous malformation. Colonic varices may
be part of the spectrum of congenital vascular abnor-
malities.5 Recognition of this abnormality is important
because colonic varices may be the cause of recurrent,
frequently massive lower gastrointestinal hemorrhage.
Our patient did not have recurrence of bleeding after
follow up for eight months. Whether surgical interven-
tion or endoscopic therapy is effective for idiopathic
colonic variceal bleeding is not known at present.

References
1. Rabinovitz M, Schade RR, Dindzans VI, Belle SH, Van
Tiel DH, Gavaler JS. Colonic disease in cirrhosis: an endo-
soscopic evaluation in 412 patients. *Gastroenterology* 1990;
2. Ganguly S, Sarin SK, Bharia V, Lahoti D. The prevalence
and spectrum of colonic lesions in patients with cirrhotic
and non-cirrhotic portal hypertension. *Hepatology*
and other mucosal changes in patients with portal
and idiopathic colonic varices: an unusual cause of lower
5. Lopez-Cepero Andrade JM, Lopez Silva M, Ferre Alamo
A, Sahdeo Puentes M, Benitez Roldan A. Familial colonic
varices: report of two cases. *Gastroenterol Hepatol*

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Pancreatrico-pericardial fistula: a rare
complication of chronic pancreatitis

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A 16-year-old boy presented with pericardial effusion,
bilateral pleural effusion and mediastinal fluid collection.
CT scan of abdomen revealed pancreatic calcification
and a fistulous tract from a pseudocyst going along the
inferior vena cava wall up to the pericardial cavity.
After initial pericardiocentesis and pleurocentesis,
lateral pancreatoco-cardiomyotomy with Roux-en-Y loop
was performed. The patient is well at 6 months follow
up. [Indian J Gastroenterol 2004;23:31-32]

Key words: Tropical calcific pancreatitis

Pleural effusion due to internal pancreatic fistula is a
known complication of chronic pancreatitis.1 However,
periardical effusion is rare and reported mainly in patients
with alcoholic pancreatitis.2,3 To our knowledge, this is
the first such report in a patient with tropical pancreatitis.

A 16-year-old boy presented to his local physician with
complaints of right-sided pleuritic chest pain and dyspnea
of five days’ duration. He was found to have right-sided pleural
effusion and was started on anti-inflammatory therapy based on
pleural fluid examination. Three months later he was referred
to our center because of recurrence of dyspnea and retrosternal
chest discomfort despite regular anti-tuberculous treatment.
Physical examination revealed pericardial and left-sided pleu-
ral effusion.

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leaking pancreatic pseudocyst/ fluid collection communicates through the esophageal or aortic hiatus into the mediastinum.\textsuperscript{5} Schoonjans \textit{et al.}\textsuperscript{6} reported that only 15 cases of symptomatic pericardial effusion secondary to chronic pancreatitis had been documented prior to 1996. Most (12/15) were related to chronic alcoholism and only 6 had documented fistulous communication to the pericardium. We did not find a report of pancreatico-pericardial fistula due to tropical calcific pancreatitis in literature.

Our patient presented with only chest symptoms and no abdominal complaints. This misled the treating physician to prescribe anti-tuberculous therapy. In pancreatic duct disruptions that occur posteriorly, the seeretions in the retroperitoneum reach the mediastinum through the aortic or esophageal hiatus.\textsuperscript{1,2} In our patient the fistula was seen to track along the IVC wall to reach the pericardial cavity where the pericardium attaches to the IVC, this has not been reported so far.

The initial management of pancreatic fistulas is conservative and includes intercostal tube drainage, somatostatin, parenteral nutrition or jejunal feeding.\textsuperscript{3,5} Medical therapy is successful only in few cases because it does not affect the ductal morphological changes.\textsuperscript{3} Endoscopic transpapillary drainage of the pseudocyst or stenting of the disrupted duct is an option in patients with internal pancreatic fistulas.\textsuperscript{5} If the fistula does not close within 2-4 weeks or there is infection, surgery is performed. The choice of operation depends on the site of leak and the presence of any associated ductal pathology.

References

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