ach. She underwent surgical exploration, which revealed a locally advanced growth with a large common hepatic artery node. The disease was considered inoperable; antecolic gastrojejunostomy was performed. Biopsy of the mass revealed non-Hodgkins lymphoma (NHL), diffuse large B-cell type (Fig). Immunohistochemistry was positive for LCA and CD 20; there was no evidence of MALT or H. pylori.

At our hospital, after confirming the histology and completing the staging work-up, a final diagnosis of stage II E B NHL of stomach was made. She was treated with six cycles of CHOP followed by involved-field radiotherapy (50 Gy/28 fractions/45 days) to the upper abdomen.

Six-monthly follow-up endoscopy showed no disease visually or on histology. Five years later she developed dyspeptic symptoms again. UGIE revealed thickened gastric mucosal folds with poor distensibility in the antrum and pylorus; the stomach proximal to the gastrojejunostomy was normal. Biopsy of the thickened fold in the antrum revealed adenocarcinoma. CT scan showed circumferential wall thickening of the distal stomach.

At exploration, there was no obvious tumor on palpation. Distal gastrectomy with D2 lymphadenectomy was performed without disturbing the previous gastrojejunosotomy. The postoperative period was uneventful. The final histology revealed a small area of ulceration in the antrum with features of adenocarcinoma (Fig) focally invading the submucosa; distal and proximal margins were free of tumor, and all lymph nodes sampled were negative. Rest of the resected stomach showed chronic atrophic gastritis with extensive intestinal metaplasia. There was no evidence of lymphoma, a few H. pylori could be seen. She is disease-free over the last 8 months.

Our patient developed gastric adenocarcinoma 5 years following treatment of gastric DLBL. The postoperative histology report of the resected specimen did not reveal any features of MALT. H. pylori can lead to MALT, which can progress to DLBL.2 This could be a hypothesis for the development of metachronous adenocarcinoma following treatment of gastric DLBL in our patient, H. pylori being an etiologic factor for both MALT and adenocarcinoma of stomach.3 The presence of chronic atrophic gastritis and intestinal metaplasia may reflect the evolution of gastric adenocarcinoma; however, only an occasional H. pylori was seen in the resected specimen. Use of alkylating agent and radiotherapy could also contribute to the development of gastric adenocarcinoma.4

References

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Primary small gut volvulus in adult presenting with hematemesis

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Small gut volvulus is rare in adults and usually presents with acute intestinal obstruction. We report a 25-year-old man with primary small gut volvulus who presented with hematemesis. The gangrenous gut was resected at laparotomy. He is well 3 months later. [Indian J Gastroenterol 2006;25:262-263]

Small gut volvulus is an uncommon cause of acute intestinal obstruction in adults. Most of the reported cases are secondary to pathology; primary small gut volvulus in adults is extremely rare.1,2

A 25-year-old man was admitted with one-day history of blood in the vomitus and continuous, severe, non-radiating upper abdominal pain. There was no significant past medical history. On examination, the patient was pale, dehydrated and febrile (38.0°C) and had tachycardia (100/
Spontaneous bacterial peritonitis in isolated splenic vein thrombosis with portal hypertension

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Spontaneous bacterial peritonitis (SBP) is a relatively common complication of chronic liver disease and rarely of some non-cirrhotic disorders.\(^1,2,3\) Lower ascitic fluid protein levels and diminished phagocytic property of neutrophils in patients with advanced liver disease predisposes to SBP.

A 50-year-old woman presented with upper GI bleed. After stabilization, endoscopic band ligation was done on bleeding grade 4 esophageal varices. Examination revealed normal liver span, and 6-cm splenomegaly without ascites. Investigations showed normal hemogram, liver enzymes, alkaline phosphatase and serum protein levels. Ultrasonographic Doppler study and MR venogram helped in making the diagnosis of isolated splenic vein thrombosis. Liver biopsy showed normal liver parenchyma and portal tracts. The patient declined further investigations and was discharged on beta-blocker therapy.

After seven months she presented with increasing ascites for one month, melena for 2 days, hematemesis for 1 day, and pain in abdomen and fever for 2 days. The patient was managed with blood transfusion, octreotide and repeat band ligation. Ascitic fluid analysis revealed high serum-ascitic albumin gradient (2.8 g/dL), ascitic fluid leukocyte count 1300/mm\(^3\) with 91% polymorphonuclear cells, and ascitic fluid protein 1.1 g/dL. The patient was started on intravenous antibiotics. Liver and coagulation profile were again normal. Culture of ascitic fluid grew E. coli; blood and urine cultures were sterile. Repeat ascitic fluid analysis 48 hours later showed 420 leukocytes/mm\(^3\) with 20% polymorphs. Tests for thrombophilia for finding the cause of splenic vein thrombosis were negative.

To the best of our knowledge, SBP has not been documented previously in pre-hepatic portal hypertension. The reasons are many. First, ascites, a sine qua non for SBP, is uncommon and transient in these patients; Sarin et al\(^4\) reported ascites in only 13% of patients with extrahepatic portal vein obstruction. Second, in patients with normal liver function, the reticuloendothelial system (RES) clears bacteria from circulation, thus decreasing bacteremia. Third, high protein content of ascitic fluid has good opsonic activity, which prevents SBP.

The reasons for development of E. coli-related SBP in our patient are unclear. She had pre-existing ascites, probably related to the recent GI bleed. It

**References**


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**Case Snippets**

**Spontaneous bacterial peritonitis** is a known complication of ascites due to cirrhosis; it has also been reported in some non-cirrhotic conditions with ascites. We report a 50-year-old lady with isolated splenic vein thrombosis who developed SBP due to *E. coli*. [Indian J Gastroenterol 2006;25:263-264]