To conclude, although the diagnosis of Meckel's diverticulum is not easy, if the complications are managed promptly and appropriately, the condition carries a good prognosis.

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Implantation of Mousseau-Barbin tube for 13 years

Mousseau-Barbin tube is a straight, semi-flexible, non-collapsible tube made of plastic. It has been used in the past for palliation of dysphagia due to carcinoma esophagus or benign stricture of the esophagus. This tube has also been used in the management of esophageal tuberculosis associated with esophageal stricture or tracheo-esophageal fistula. The long-term outcome of such a tube placement has not been reported.

A 40-year-old lady was admitted in 1979 with absolute dysphagia, cachexia and dehydration. Rapid esophagoscopy showed a reddish mass occluding the esophageal lumen at 34 cm from the incisors; biopsy was taken. With a suspicion of carcinoma esophagus, a 12-mm diameter Mousseau-Barbin tube was implanted by passing an esophageal bougie retrograde through a left upper paramedian laparotomy and gastroscopy and retrieving it through the mouth. The tube was tied to the bougie and pulled into the stomach until the funnel rested firmly above the lesion. The lower end was stitched to the stomach. Histology of the biopsy tissue showed tuberculosis.

She was asymptomatic after antitubercular treatment for 18 months, when removal of the tube was advised; however, since she was asymptomatic, she did not follow this advice. Thirteen years later, she reported with complaint of a hard linear mass in the left hypochondrium and epigastrium. On palpation, it appeared that the Mousseau-Barbin tube was lying in the stomach. The tube was removed through a gastrotomy. Esophagoscopy showed normal esophagus.

Mousseau-Barbin has been reported to be well tolerated in the short run although a number of complications have been reported. Long-term implantation of this tube has not been reported. In this patient, the tube was tolerated well for 13 years.

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Hepatic vein thrombosis with ulcerative colitis

In reference to the recent report on sagittal sinus thrombosis in a patient with ulcerative colitis, we wish to report hepatic vein thrombosis leading to the Budd-Chiari syndrome in a patient with ulcerative colitis.

An 11-year-old girl was referred for investigation of jaundice and hepatosplenomegaly. She had a history of intermittent bloody diarrhea and iron-deficiency anemia for 5 years. At the age of 7 years she had received 3 units of blood transfusion. She had been on oral iron and folic acid supplementation, but the anemia persisted. She developed jaundice and abdominal distension for three weeks prior to admission. She had no fever or abdominal pain and no past history of jaundice. Other symptoms included episodic ill-defined lower abdominal discomfort, and arthralgia in the large joints, especially the knees and ankles. There was no family history of gastrointestinal disease. Her appetite was good and weight was stable.

On examination, she was pale and had mild icterus. Her weight was 36 Kg and height, 138 cm. The vital parameters were normal. Cardiovascular and respiratory systems were normal. Her abdomen was soft and non-tender, with dilated veins over the flanks and back, hepatosplenomegaly, and free fluid. There were no focal neurological abnormalities.

Investigations: Microcytic hypochromic anemia (hemoglobin 8 g/dL); WBC 10.9×10^9/L (18% polymorphs), platelet count 290×10^9/L; ESR 13 mm in first hour. Asciotic fluid analysis revealed wide albumin gradient with no evidence of infection. Upper gastrointestinal endoscopy revealed one grade 1 varix. Duodenal biopsy was normal. Barium meal follow-through was normal. Colonoscopy documented active colitis with confluent erythema, ulceration and friability, extending from the rectum to the midtransverse colon. Colonic biopsies revealed diffuse mucosal inflammation with heavy infiltrate of acute and chronic inflammatory cells, crypt abscesses and ulceration of surface epithelium.

Renal and liver parameters were normal. Viral serology (HBsAg, anti-HCV by EIA) was negative. Her 24-hour urinary copper and serum ceruloplasmin levels were normal. Antinuclear
antibody and lupus anticoagulant were negative. There was no evidence of disseminated intravascular coagulation. Protein C, protein S and antithrombin III levels were normal. The international normalized ratio and activated partial thromboplastin time ratios were both abnormal. Markers for autoimmune liver disease (antimitochondrial antibody, smooth muscle antibody, anti-LKM1) were negative. Ultrasound and doppler sonography failed to pick up normal phase forward flow in hepatic veins, but showed comma-shaped intrahepatic collateral vessels, normal splenorenal venous axis and free fluid. Dynamic CT scan revealed global liver enlargement with patchy caudate lobe enhancement with radiographic contrast.

Inflammatory bowel disease is known to be associated with microvascular and macrovascular thrombosis. This has been reported to involve the pelvic plexus, lungs, portal-mesenteric systems, cerebral veins and peripheral veins.1,2 The coexistence of these two conditions, i.e., hepatic vein thrombosis leading to Budd-Chiari syndrome and ulcerative colitis in our case may be either a coincidence or an association. As thromboembolic phenomena are well documented in inflammatory bowel disease, the latter seems more likely. This has been reported in only one case previously.4

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Hepatocellular carcinoma presenting as soft tissue mass in gluteal region

Metastasis in hepatocellular carcinoma usually occurs late in the clinical course1 and is rarely discovered before autopsy. We report a patient with hepatocellular carcinoma with soft tissue metastasis in the gluteal region.

A 38-year-old man with hepatitis B virus-related cirrhosis, portal hypertension and ascites, presented with rapid distension of the abdomen, unresponsive to diuretics, along with a small painless lump of recent onset in the right gluteal region. On clinical examination, the patient had firm, nodular hepatomegaly, 4 cm below the costal margin, along with massive splenomegaly and tense ascites. The lump in the gluteal region was 2 cm x 2 cm in size, non-tender, with a smooth surface and well-defined edge, firm in consistency and mobile in all directions. The lump was increasing in size rapidly; a month later it had increased to about 5 cm x 5 cm, was fixed to the underlying structures, and the overlying skin was stretched and glossy with engorged veins.

Investigations: AST 102 U/L (N <40), ALT 78 U/L (N <40), alkaline phosphatase 15 KAU/dL (N <130), serum bilirubin 2 mg/dL (direct 1.4). Ascitic fluid was transudative, without malignant cells. He was HBSAg positive, and upper gastrointestinal endoscopy revealed three columns of grade II varices. Sonography revealed a nodular liver with diffusely altered echotexture, splenomegaly, and retroperitoneal, mesenterial and short gastric collaterals. CT scan revealed a nodular liver with multiple hypodense lesions in both lobes, with a large soft tissue mass in the muscles to the right of the erector spine muscle (Fig): this mass was destroying the underlying posterior aspect of iliac blades and sacrum. Alpha-fetoprotein levels were >500 IU/L (N <10). Fine-needle aspiration from the lump revealed it to be a metastatic lesion from adenosquamous, possibly hepatocellular carcinoma. Needle aspiration from the hypoechoic hepatic lesion was suggestive of hepatocellular carcinoma. The patient died after a rapid deteriorating course.

Most metastases from hepatocellular carcinoma are encountered at autopsy. Metastasis to soft tissues is rare; these patients have a poor prognosis. Studies in patients with bony metastasis have revealed that aggressive surgery and local radiotherapy can prolong survival; this treatment can be tried in patients with soft tissue metastases.2 Some studies have shown that octreotide administration significantly improves survival and can be a valuable alternative in the treatment of inoperable cases.3

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