Colonoscopy was normal.

Hookworms develop into adults in the small intestine and anchor themselves to the mucosa. Very rarely they have been recovered from the gastric antrum and cecum. The ectopic localization in the antrum has been attributed to jejuno-duodeno-gastric reflux. The recovery of hookworms from the cecum has been attributed to bowel preparation that may have washed the worms downstream. In our patient the duodenum and ileum were normal and worms were seen on initial sigmoidoscopy itself. Moreover the hookworms were seen anchored to the mucosa with evidence of oozing of blood from punctate erosions in the colon. To the best of our knowledge this is the first report of symptomatic hookworm infestation of the colon.

Unusual cases of hookworm infection of proximal jejunum causing intestinal bleeding diagnosed by enteroscopy have been reported. Recently the dog hookworm Ancylostoma caninum has been found in adult form in the human small intestine and has been implicated in cases of eosinophilic enteritis. Diagnosis of hookworm infection relies on the identification of ova in the feces but differentiation between species based on morphology of ova is extremely difficult. In some instances of infestation by male hookworms only, stool examination will be negative for ova. As colonoscopy is indicated in most patients with iron-deficiency anemia and positive stool occult blood test, the physician who performs endoscopies on patients from endemic regions should recognize these helminths.

References

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Fig: Colonoscopy showing multiple punctate erosions and hookworms (Inset: worm with one end buried in mucosa, with blood in its gut)

Laparoscopic control of spontaneous external hemorrhage from umbilical varix

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Spontaneous external hemorrhage from an umbilical varix is rare. We describe a 40-year-old man with cirrhosis and portal hypertension, who presented with recurrent external bleeding from an umbilical varix. The first episode was controlled by transfixation of the vein under local anesthesia. Contrast-enhanced CT scan demonstrated a hugely distended recanalized umbilical vein arising from the left branch of the portal vein and ending in the umbilical cicatrix. Recurrent bleeding necessitated laparoscopy and in-situ clipping of the bleeding vein in the falciform ligament. At six months' follow up the patient has no further bleeding. [Indian J Gastroenterol 2006;25:211-212]

Symptomatic ectopic varices in portal hypertension are unusual. The detection of a recanalized umbilical vein has been an incidental ultrasound finding in these patients. External hemorrhage from rupture of an umbilical varix is rare. The two cases reported in literature were managed by exomphalectomy.

A 40-year-old man presented to the emergency medical services with massive hemorrhage from his umbilicus following trivial blunt trauma to the abdomen when he slipped and fell. He was a chronic alcoholic, receiving treatment for diabetes mellitus and essential hypertension for the past 3 years. On examination, he was pale, anicteric, with blood pressure 90/60 mmHg and pulse rate 110/min. There was active bleeding from an umbilical varix. There was no visible caput medusae or periumbilical venous hum. The spleen was enlarged and there was no ascites. The patient was resuscitated and the umbilicus was explored under local anesthesia and a dilated umbilical vein was isolated and transfixed with silk sutures.

Investigations: Hemoglobin 7.5 g/dL, total leukocyte count 3,200/mm³, platelet count 59,000/mm³, blood sugar
17 mmol/L, bilirubin 51 μmol/L (direct 15), AST 90 IU/L, ALT 45 IU/L, alkaline phosphatase 369 IU/L, GGT 1549 U/L, prothrombin time within 1 second of control. Serological tests were negative for HBsAg and anti-HBs.

Ultrasoundography revealed coarse echopattern of a normal-sized liver, enlarged spleen of 16.5 cm in the longitudinal axis, portal vein diameter of 14 mm with left branch more dilated than the right, and no ascites. Duplex ultrasound revealed a 0.9-cm umbilical vein with flow away from the liver and arising from the left branch of the portal vein, with multiple collateral vessels in the splenic hilum, spleno-renal ligament and retroperitoneum. Gastro-duodenoscopy revealed two columns of esophageal varices and mild portal congestive gastropathy. The varices were prophyllactically injected with six mL polidocanol. Contrast-enhanced CT scan revealed a nodular shrunken liver with mild hypertrophy of left lobe. Portal vein measured 18 mm and had a patent lumen. The spleen was enlarged, with splenic vein 14 mm. Multiple splenic hilar, spleno-renal, mesenteric and anterior abdominal wall collaterals with a 1-cm umbilical vein were seen.

Five days following the first bleed he had recurrence of bleeding (about 100 mL) from the umbilical varix, following a bout of cough in the night, which was controlled with external pressure alone. In view of recurrent bleeding, laparoscopic proximal clipping of the umbilical vein was suggested. At laparoscopy under general anesthesia, a Veress needle was inserted in the left lumbar area away from the anterior abdominal wall collaterals and a closed pneumoperitoneum was established. A left lumbar port was introduced, followed by introduction of a left hypochondrial port under direct vision of the laparoscope, avoiding the other abdominal wall collaterals. Laparoscopy revealed a macronodular cirrhotic liver with a 1-cm dilated umbilical vein in the falciform ligament. In-situ clipping of the umbilical vein was done using a 10-mm clip applicator with large titanium clips (LT 400 Ligaclip® Extra; Ethicon Endo-Surgery, Cincinnati, OH, USA) (Fig). The operating time was less than half an hour and the patient did not receive blood transfusion.

Postoperative duplex ultrasound revealed no flow in the umbilical vein distal to the site of clip application. The patient was discharged with propranolol and his oral hypoglycemic and antihypertensive medications. There was no further episode of bleed attributable to portal hypertension at six months of follow up. Gastro-duodenoscopy revealed obliterated esophageal varices.

Rupture of a recanalized umbilical vein leading to hemodynamic instability is exceedingly rare. Rupture usually results in hemoperitoneum. Treatment involves urgent laparotomy and ligation of the bleeding varices. One patient died of esophageal variceal bleed following ligation of all the anterior abdominal wall collaterals. We clipped only the bleeding recanalized umbilical vein in our patient and did not obliterate the other abdominal wall varices.

The success of minimal-access clipping of the umbilical varix is due to the precise mapping of the abdominal wall collaterals on pre-operative imaging. As a result, safe placement of the Veress needle with minimal blood loss could be achieved during the procedure. Laparoscopy is considered safe in Child’s class A and B patients.

Only two cases of spontaneous umbilical hemorrhage from umbilical varices have been reported, to the best of our knowledge. Both had favorable outcome following variceal ligation and excophagectomy. Our patient had re-bleeding following umbilical exploration and variceal ligation, necessitating proximal clipping of the vein laparoscopically.

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