Portal hypertensive enteropathy; an unusual cause of gastrointestinal bleeding in cirrhosis: or is it?

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ABSTRACT We present a patient with liver cirrhosis, and portal hypertensive gastropathy who was transfusion-dependent due to chronic gastrointestinal blood loss. Wireless capsule endoscopy revealed bleeding from extensive vascular ectatic lesions in the small bowel and right colon as well as from the stomach. We discuss briefly the recent literature relating to chronic gastrointestinal bleeding due to portal hypertensive enteropathy, the diagnosis of this condition, and the use of TIPSS in treatment.

KEYWORDS Cirrhosis, gastrointestinal bleeding, portal hypertensive enteropathy

LIST OF ABBREVIATIONS Argon plasma coagulation (APC), portal hypertensive enteropathy (PHE), portal hypertensive gastropathy (PHG), transjugular intrahepatic porto-systemic shunt (TIPSS), wireless capsule endoscopy (WCE)

DECLARATION OF INTERESTS No conflict of interests declared.

CASE REPORT

A fifty-one-year-old man with cryptogenic cirrhosis became blood transfusion-dependent for two years due to continued gastrointestinal blood loss. He required up to six units of blood per week.

Continued melaena suggested a source of bleeding in the upper gastrointestinal tract. Upper gastrointestinal endoscopies had revealed florid PHG and well-covered gastric varices without stigmata of bleeding. Vascular ectatic lesions were also present in the first and second part of the duodenum. A colonoscopy was reported as normal. He had other signs of portal hypertension including splenomegaly and thrombocytopenia but normal coagulation. Despite treatment with beta-blockers, he remained transfusion-dependent. Endoscopic APC of the PHG lesions led to endoscopic improvement, but he still required up to three units of blood every two weeks.

In view of continued bleeding, WCE (Pillcam™, Given Imaging Ltd.) was carried out. This confirmed the previous gastric antral (see Figures 1 A–B) and proximal duodenal vascular ectatic lesions. Furthermore, mucosal oedema and vascular ectatic lesions (some actively bleeding during the examination) were visible along the jejunum and down to the ileum (see Figures 1 C–E), in keeping with a diagnosis of PHE. Wireless capsule endoscopy also allowed vascular ectatic lesions to be seen in the right colon (see Figure 1 F).

Widespread lesions along the totality of the small bowel made endoscopic treatment through an enteroscope impracticable, so a decision was taken to treat the patient with a TIPSS. Following successful TIPSS insertion the portal pressure gradient was reduced from 15 to 3 mmHg. He suffered a short-lasting episode of hepatic encephalopathy. His transfusion requirements dramatically improved, and three months after TIPSS insertion he no longer required any blood transfusions.

DISCUSSION

Portal hypertensive enteropathy is a recently recognised manifestation of portal hypertensive intestinal vasculopathy. Endoscopic findings include bleeding, vascular ectasias, ulceration and small bowel varices. In a study of 44 patients, ileal varices were present in 18% of patients with cirrhosis, especially in patients with concomitant colopathy.1 A case control study in similar patients demonstrated jejunal lesions, and all patients had portal gastropathy.3 Previous case reports have demonstrated the efficacy of push enteroscopy in the initial diagnostic work-up of these cases, as well as proposing retrograde ileoscopy and WCE as alternative modalities.3

In a comparative study of 37 cirrhotic cases vs controls using WCE, mucosal changes were found to be significantly more common in the former (67.5% vs 0,
Portal hypertensive enteropathy

FIGURE 1 A–B Wireless capsule endoscopy images. Portal hypertensive gastropathy (antrum) with active bleeding (B).

FIGURE 1 C–D Wireless capsule endoscopy images. Portal hypertensive enteropathy with lesions in the jejunum.

FIGURE 1 E Wireless capsule endoscopy image. Portal hypertensive enteropathy with lesions in distal ileum.

FIGURE 1 F Wireless capsule endoscopy image. Portal hypertensive colopathy with lesions in the ceacum.
p<0·001). Grade 2+ or larger oesophageal varices, portal gastropathy and colopathy and Childs–Pugh class C cirrhosis were found to be significantly associated with PHE, mirroring findings of previous studies. Underlying cause of cirrhosis, gender, or prior history of variceal bleeding was not related to the presence of PHE.

Wireless capsule endoscopy is now firmly established as an adjunct investigation in identifying sources of obscure gastrointestinal bleeding. A recent study comparing WCE and enteroscopy showed high detection rates of occult bleeds with both. Although the detection rate of WCE was superior, their results indicate that the procedures are complementary; an initial diagnostic imaging employing WCE might be followed by therapeutic enteroscopy in some cases. Over 170,000 WCE examinations have been carried out to date, with very few clinical risks or technical problems, which can often be prevented by careful case selection.

This case also elegantly demonstrates the therapeutic potential of TIPSS in the context of severe chronic blood loss due to portal hypertensive intestinal vasculopathy. Wireless capsule endoscopy imaged most of potential bleeding sites, not only in the stomach and colon, but also throughout the small intestine. Small bowel enteropathy correlates with portal hypertensive gastropathy, and colopathy, for which TIPSS is a recognised treatment modality. Wireless capsule endoscopy is a very useful new modality of investigating such complex cases.

REFERENCES