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Case report

Upper gastrointestinal bleeding due to ectopic varices in a patient with alcoholic cirrhosis

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SUMMARY

We report a case of upper gastrointestinal bleeding due to ectopic varices located in the duodenum in a patient with alcoholic cirrhosis. Ectopic varices are a rare cause of intestinal hemorrhage in patients with portal hypertension, whether they suffer from cirrhosis or not. Hemorrhage can be severe and high mortality rates have been reported. The treatment of varices is challenging and has been debated in the literature. In the case presented, we used injection sclerotherapy with ethanolamine oleate, to obliterate varices and control bleeding. A short review on the etiology pathogenesis and management of ectopic varices is presented.

Key words: ectopic varices, cirrhosis, gastrointestinal bleeding, portal hypertension, injection sclerotherapy

INTRODUCTION

Duodenal varices are ectopic varices commonly found in patients with portal hypertension. Although they may be found angiographically in up to 40% of these patients, they rarely bleed. However hemorrhage can be severe, difficult to treat and therefore related to poor prognosis.

We report a case of upper gastrointestinal (GI) bleeding due to duodenal varices in a patient with alcoholic cirrhosis, who was successfully treated with injection sclerotherapy.

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CASE REPORT

A 57-year-old patient, with a history of decompensated alcoholic cirrhosis, was admitted to the hospital because of melena, which had occurred 12 hours before admission. His history included diabetes mellitus, treated with insulin, and cholelithiasis. Although he reported two previous episodes of GI bleeding, no Endoscopy had been performed and propranolol was prescribed for suggested portal hypertension. In admission he was pale, well orientated, with a blood pressure of 80/40 mmHg and a rare of 66b/min. After volume replacement and stabilization he underwent emergency upper GI Endoscopy. No esophageal varices were detected (Figure 1). No fresh blood or coffee-ground material were found in the stomach. The duodenal bulb was normal. In the second part of the duodenum, we noticed three large, confluent varices with stigmata ("white nipple") in the confluence, but no active bleeding (Figures 2, 3). Three hours later the patient vomited fresh blood. A second Endoscopy was performed and oozing hemorrhage at the confluence of the varices was noticed (Figure 4). We performed injection sclerotherapy with 9 cc of ethanolamine oleate and the bleeding stopped. In the next 7 days no recurrence of the hemorrhage occurred and the patient recovered uneventfully. He didn't consent to undergo angiography, but Doppler-ultrasound examination identified the ectopic varices in the second part of the duodenum (Figure 5).

Gastroscopy was performed on the day before discharge and was repeated three weeks later while the patient was receiving treatment with beta-blockers. Varices were present in the 2nd part of the duodenum, with no stigmata, and no endoscopic therapy was performed.

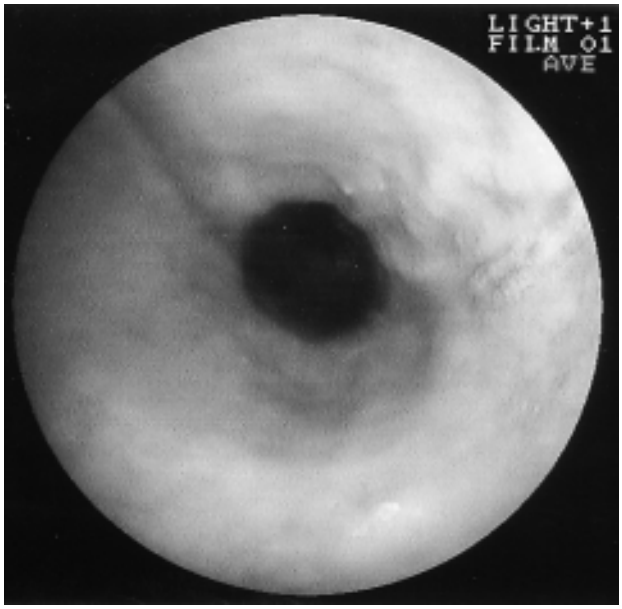
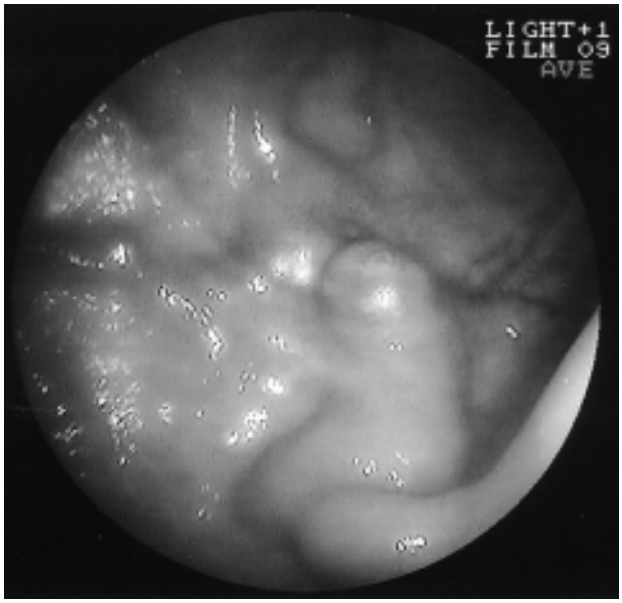
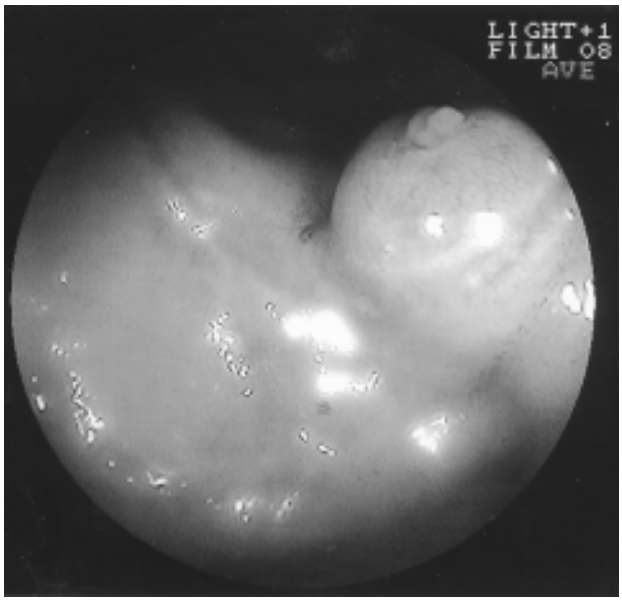


Figure 1. Esophagus; no varices are present.





Figures 2, 3. Varices in the second part of the duodenum; a stigma (white-nipple) is seen in the confluence of the varices.

DISCUSSION

Ectopic varices are "natural large portosystemic venous collaterals which appear apart from the gastroesophageal region anywhere in the abdomen".[1] They are usually found in patients with portal hypertension although familial occurrence in the absence of portal hypertension has been reported.

The prevalence of ectopic varices depends on the technique used for diagnosis and on the etiology of portal hypertension. It's reported to appear in 1% to 5% of cirrhotic patients and up to 20% to 30% of patients with extrahepatic portal hypertension.[2,3] The location of the varices also depends on the cause of portal hypertension.[2] Duodenal varices are found by angiography in more than 40% of patients with extrahepatic portal hypertension (due to obstruction of the portal or splenic vein). Varices in other sites of the small or large intestine are commonly found in patients with cirrhosis, especially in those with history of abdominal surgery, stomas etc. (e.g. patients with primary sclerosing cholangitis who have undergone colectomy and ileostomy for underlying inflammatory bowel disease).

Although commonly present, duodenal varices rarely bleed. The first report of bleeding from duodenal varices was presented by Alberti et al in 1931.[4] Bleeding can be fatal and mortality rates may reach 35% to 40%.[5-7]

The duodenal bulb is the most common location of duodenal varices. Their frequency decreases at the distal duodenum.[8] They are usually located in the deeper layers of the duodenal wall, in contrast to the submucosal position of the esophageal varices. If they are not endoscopically seen, they have no clinical value, since they never bleed.

The afferent vessel of the varix is usually the superior or inferior pancreaticoduodenal vein, the superior or inferior mesenteric vein and sometimes the gastroduodenal or pyloric veins.[9] The efferent vein drains into the

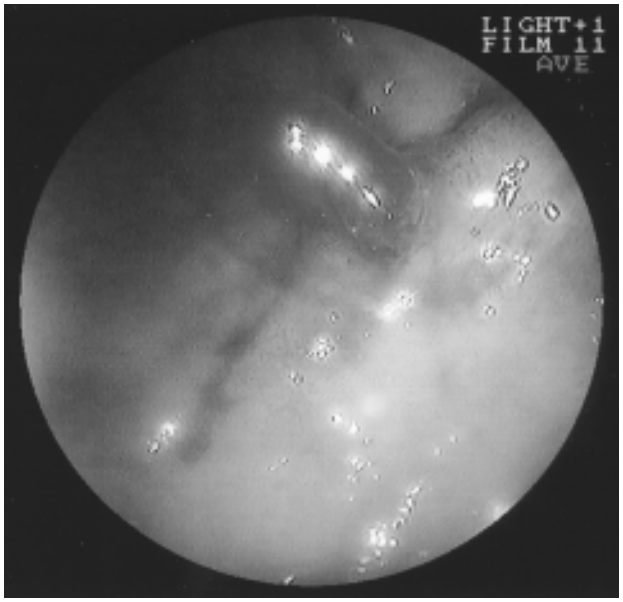


Figure 4. Oozing hemorrhage at the confluence of the duodenal varices.

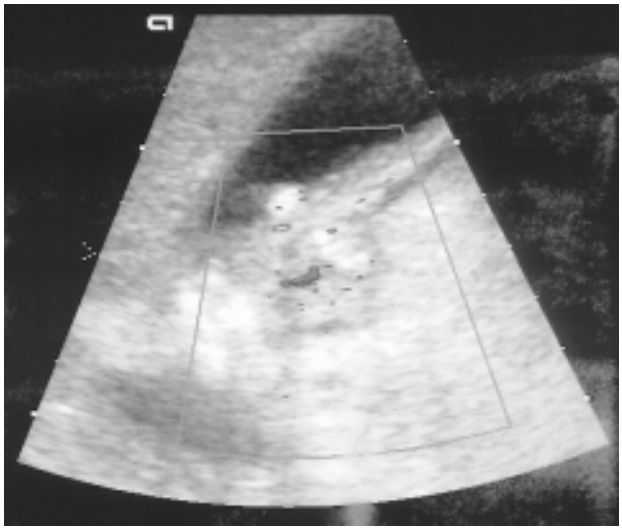


Figure 5. On Doppler-ultrasound examination the ectopic varices are seen in the wall of the duodenum.

inferior vena cava either directly or through the retroperitoneal veins. Duodenal varices seem to have smaller diameter and shorter length than esophageal varices. Wall tension (depending on the vessel size and the portal pressure) seems to be the major determinant of risk of rupture.[10]

The formation of duodenal varices depends on the etiology of portal hypertension. In patients with extrahepatic portal hypertension varices spring from portal-to-portal anastomoses connecting afferent branches of the portal vein, upstream of the obstruction. In patients with previous abdominal surgery, adhesions can be formed between the bowel and the abdominal wall or between other abdominal structures drained by the systemic venous circulation. Collaterals, within the wall of the duodenum may open up. Finally, there have been reports of formation of duodenal varices after injection slcerotherapy or ligation of esophageal or gastric varices.[11] This is probably due to post-treatment alterations in the hemodynamics of portal flow.

Management of bleeding duodenal varices is difficult and depends on local expertise and the cause of portal hypertension. The optimal therapy has been debated in the literature. There are reports of treatment with injection sclerotherapy with different types of sclerosant agents such as ethanolamine, polidocanol, dextrose 50% solution with 3% sodium tetradecylsulfate, polidocanol/thrombin.[12,13] Emergency sclerotherapy has been shown to be useful as a first-line therapeutic measure in the treatment of bleeding duodenal varices.

Endoscopic variceal ligation of ectopic varices has been reported,[14] but some authors believe that the banding technique is unsafe for large ectopic varices, since the entire varix cannot be banded and there is also a risk of causing a wide defect in the varix after sloughing off the band.[1]

Embolization therapy using radiological techniques is an alternative in the short term management of bleeding ectopic varices and controls bleeding in up to 94% of cases.[15,16] However rebleeding rates over 1 year are high. If the patient continues to bleed, options include either using a TIPS or proceeding with surgery. Surgery is preferred in patients with Child-Pugh class A and in patients with extrahepatic portal vein thrombosis. In the review of Khouqeer[6] on the surgical treatment of duodenal varices, the portocaval shunt was the most effective method of preventing recurrent hemorrhage.

TIPS are effective in controlling bleeding in the acute phase but up to 50% of TIPS will stenose in 6 months and long-term mortality is high, due mainly to poor liver function.[17,18] Thus TIPS can be used in patients with Child-Pugh class B or C, who are candidates for liver transplantation, if endoscopic or radiological embolization therapy fail.

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