Duodenal Varices: A Novel Treatment and Literature Review

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Hemorrhage associated with duodenal varices is an uncommon but often fatal manifestation of portal hypertension. We report a case of duodenal varices, review the literature, and present a new treatment modality. A 63-year-old man presented with hematemesis and hematochezia. An upper gastrointestinal endoscopy revealed hemorrhage from the duodenal varices that was initially controlled with injections of epinephrine. However, this was only partially successful, as the patient had repeated episodes of bleeding that was not amenable to injection sclerotherapy. The patient was taken emergently to the operating room after endoscopy failed to control the hemorrhage. The bleeding was controlled with simple oversewing of the duodenal varices through a duodenotomy. Three years later the patient remains symptom free. We propose that simple oversewing of duodenal variceal veins combined with a beta-blocker is an effective treatment for duodenal variceal hemorrhage.

DUODENAL VARICES ARE an uncommon endoscopic finding and an unusual surgical problem. The causes for duodenal varices are numerous; however, treatments for this problem are limited. In this paper we present a review of the literature and a case report of duodenal varices along with a novel treatment.

Case Report

A 63-year-old man presented to the VA hospital on December 8, 1997 with hematemesis, hematochezia, and orthostatic hypotension. The patient had an extensive past surgical history beginning in May 1981 when he underwent cholycystectomy for acute cholecystitis. After the operation he became septic with a bile leak and a subdiaphragmatic abscess. He underwent a second surgical procedure for drainage of the abscess with T-tube placement. He was discharged a month later on June 2, 1981.

He presented to the Mayo Clinic 8 days later and was found to have an enterocutaneous fistula, pancreatitis with large cysts, and an abscess formation causing a partial obstruction of the duodenum. A fistulagram through the skin incision revealed the large previously evacuated abscess cavity with communication to several loops of small bowel. The T-tube cholangiogram was normal. On June 12, 1981 he underwent another surgery for evacuation of a pancreatic abscess. He did well postoperatively and was discharged.

For the next 2 years it was noted that the patient had intermittent abdominal pain and sequential CT scans that revealed a persistent inflammatory mass in and around the pancreatic head.

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In October 1988 the patient presented again to the Mayo Clinic with intermittent pain, increasing abdominal girth, and an abdominal wall hernia. A CT scan again revealed an enlarged pancreatic head without change from previous studies and a large amount of ascitic fluid. Diagnostic paracentesis produced chylous fluid with negative cultures and cytology. An esophagoduodenogastroscopy was normal.

In November 1988 he had a reaccumulation of ascites and an exploratory laparotomy was done attempting to identify the cause of ascites, to repair the abdominal wall hernia, and for possible placement of a LeVeen shunt. At laparotomy 8 liters of ascites were removed, the left side of the abdomen was normal but "the right side was completely obliterated with agglutinated bowel and adhesions." Multiple biopsies were taken of this mass/adhesions at the head of the pancreas; all biopsies showed inflammatory granulation tissue. A follow-up CT in October 1989 looked similar to previous CT scans.

The patient did not have further surgical problems until he presented to our institution with massive upper gastrointestinal (GI) bleeding. Three endoscopies on sequential days revealed blood in the stomach and duodenum without an identified source of the bleeding. It was noted that the patient had thickened folds in the duodenum; on the third endoscopy a biopsy was taken of the enlarged duodenal folds (Fig. 1). There was an immediate large hemorrhage from the biopsy site, which was stopped with an injection of epinephrine. It then was apparent that these were duodenal varices. The patient did not have any history of alcohol use or liver disease and a transfer to a local tertiary-care center was arranged for a possible transjugular intrahepatic portosystemic shunt (TIPS) procedure.

An angiogram revealed a normal portal vein with normal splenic venous return. The superior mesenteric vein was

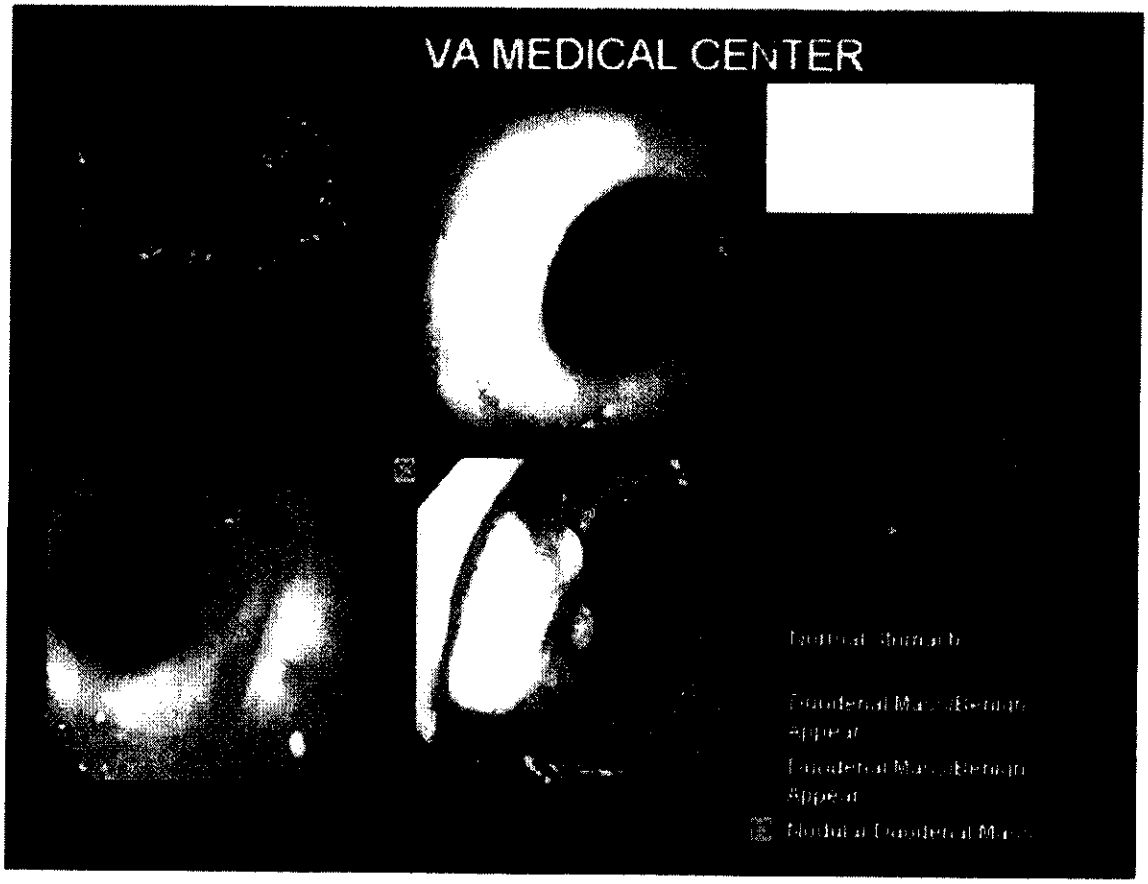

FIG. 1. *Left to right:* A normal-appearing gastroesophageal junction; duodenal bulb showing varices laterally; other varices in the duodenal bulb; more duodenal varices in the second portion of the duodenum.

replaced by a large mass of tangled vessels, which surrounded the second, third, and fourth portion of the duodenum. After the angiogram the patient again had massive bleeding. A repeat endoscopy identified the duodenal varices as a source of the bleeding, but it could not be stopped with injection therapy, and the patient was taken emergently for operation where the bleeding was stopped by oversewing the duodenal varices through a duodenotomy. Further surgical therapy for bleeding was not feasible secondary to extensive unresectable adhesions in the right upper quadrant. He was transferred back to the VA hospital with the institution of beta-blocker therapy in an attempt to reduce venous portal hypertension through vasodilatory effects.

Evaluation of recurrent vomiting discovered a fistula between the third portion of the duodenum and the peritoneal cavity. The patient was placed on total parenteral nutrition until June 15, 1998 when it was proven that the fistula had closed. However, the patient continued to have nausea and vomiting and oral intake was not possible. A repeat endoscopy was performed which showed stricture at the site of the oversewing. Endoscopic balloon dilation was performed on three separate occasions over the course of a month. This relieved all symptoms of nausea and vomiting. Total parenteral nutrition was stopped on July 15, 1998. A repeat CT was performed that was unchanged from those done at the Mayo Clinic in the 1980s. Three years later the patient has suffered no further nausea, vomiting or upper GI bleeding.

Literature Review

Duodenal varices can be classified as hepatic (two-thirds of cases) or extrahepatic (remaining one-third).[1] Cirrhosis is the most common hepatic cause and accounts for 30 per cent of all cases of duodenal varices.[2, 3] These percentages vary geographically as evidenced by Al-Mofarreh's series of 13 patient with schistosomiasis.[4] The extrahepatic causes are many and result from occlusion of one of the major vessels in the area: the splenic, portal, or superior mesenteric vein. Thrombotic disorders are one of the first causes that should be investigated.[1, 2, 5, 6] Surgery is an undocumented cause of duodenal varices and Khouqeer et al. state they have not been associated with surgical adhesions.[7] Obstructive tumors and severe pancreatitis have also been noted to cause duodenal varices.[1, 4, 8]

Diagnosis of duodenal varices is almost always made during endoscopy for investigation of GI bleeding.[9] The diagnosis has also been made on barium studies, splenoportography, angiography and laparotomy. It is not unusual to use more than one of these modalities to confirm the diagnosis. When duodenal varices are diagnosed by endoscopy 60 per cent have varices elsewhere in the GI tract.[1] Fifty per cent of

these are gastroesphageal.[8] Forty per cent of patients with portal hypertension have duodenal varices at angiography, but they are rarely clinically significant because they often do not penetrate the submucosa.[10]

The treatment is as varied as the causes with success depending on the clinical situation. For acute bleeding there are many reports of successful injection sclerotherapy using a variety of solutions.[11] Portocaval shunts are occasionally appropriate.[11–13] When possible gastroduodenectomy and duodenectomy has been effective. Simple surgical ligation of affected vessels has worked.[14, 15] Radiological embolization has recently been gaining acceptance as well.[10] Long-term outcome is regarded as grim by all authors; however, no long-term series are available to substantiate this assumption.

In summary it can be said that duodenal varices are rare, have many etiologies, and are often fatal. Our case was unique in that bleeding was controlled with simple oversewing and medical management, and the etiology was due to previous surgical trauma with adhesions.

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