Duodenal Varices: A Rare Manifestation of Portal Hypertension

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Background
Duodenal varices are a rare but potentially serious consequence of portal hypertension in the event of a bleed. The etiology of duodenal varices can be classified into hepatic (e.g. cirrhosis) or extra hepatic (e.g. portal, splenic or superior mesenteric vein thrombosis). Endoscopic injection sclerotherapy (EIS) and endoscopic variceal ligation (EVL) are widely accepted as primary therapies for esophageal variceal bleeding whereas bleeding gastric fundal varices are usually treated with cyanoacrylate injection or shunt procedures. However there is no widely accepted treatment modality for duodenal varices.

Method
We report two patients with duodenal varices secondary to portal hypertension and briefly discuss the different treatment modalities reported in treatment of duodenal varices.

Results
Case 1
The first patient is a 19-year-old female with significant portal hypertension and cavernous transformation of the portal vein secondary to portal vein thrombosis acquired as a child following umbilical vein cannulation. Grade II and III esophageal varices, a small gastric fundal varix and a solitary post-bulbar duodenal varix approximately 2cm in diameter were noted at screening endoscopy (Figure 1& 2).

The esophageal varices were successfully obliterated by EVL after a total of seven sessions and she remains on a beta-blocker. The size of duodenal varix was unchanged at follow up endoscopies. An abdominal CT also demonstrated several large intra-abdominal varices (mesenteric and splanchnic) not visible at endoscopy. This patient is currently being managed conservatively with beta-blockade only. She has not had a variceal bleed to date.

Case 2
The second patient was a 56-year-old male with a long standing history of diabetes and severe coronary artery disease as well as decompensated liver disease secondary to alcoholic cirrhosis with portal and splenic vein thrombosis. He presented to hospital with a myocardial infarction and gave a history of melena. An esophagastroduodenoscopy was performed which did not identify a source of bleeding. No varices were documented at this time. He represented to hospital two weeks later with an episode of severe upper gastrointestinal bleeding and was found at repeat endoscopy
to have large varices in the duodenal bulb. The varices were not amenable to band ligation due to their extent and he underwent a mesocaval shunt which successfully controlled the bleeding. However, he died several days post-operatively due to a second myocardial infarction leading to asystolic cardiac arrest.

**DISCUSSION**

Duodenal varices are rare, occurring in 0.4% of patients with portal hypertension. The commonest site is the duodenal bulb followed by the second part of the duodenum. The diagnosis is frequently made at endoscopy or by other means e.g. barium studies, angiography or laparotomy/laparoscopy. Two-thirds of all reported cases of duodenal varices have portal venous hypertension caused by hepatic cirrhosis. In the remaining one-third, pre-hepatic portal hypertension as a consequence of either a compromised portal venous circulation (portal vein thrombosis) or a primary hematological disease was the underlying cause. The fact that the pancreaticoduodenal venous communication with the systemic venous system via the veins of Retzius is one of the four major portosystemic communications, splanchnic hypertension would result in variceal dilatation at the duodenum. There is currently no consensus with regards to best treatment option and very little evidence on which to base clinical decision-making.

Isolated case reports have reported treatment of active hemorrhage with variceal obliteration (i.e. injection of liquid tissue adhesive, sclerosant and/or band ligation) whereas others have reported treatment with decompressive shunting procedures e.g. transjugular intrahepatic porto-systemic shunt (TIPSS) and portocaval shunting. There are also several reports of successful variceal obliteration using balloon-occluded retrograde transvenous obliteration (BRTO) and surgical procedures like oversewing/ligation of varices, duodenal dearterialization and stapling, duodenectomy or gastroduodenectomy. Blood flow in duodenal varices is frequently high and results in profuse bleeding. Prognosis is poor with mortality rates as high as 40%. We note that the literature on prophylaxis of patients who have yet had a first bleed is scant.

**CONCLUSION**

Duodenal varices are rare but can be very difficult to treat successfully, particularly as there is no consensus as to the best treatment option. This is highlighted in the two cases presented here. Further data are needed to determine the efficacy and safety of endoscopic therapies but shunt therapy is probably still the preferred option, especially as non-intervention is a risky strategy with such a significant mortality from bleeding.

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