SCLEROTHERAPY OF DUODENAL VARICES WITH HISTOACRYL – A CASE REPORT

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Bleeding from duodenal varices, although clinically not so common as esophageal and gastric varices, could be severe and fatal. We now report such a case: A 43 y/o male patient, used to be a victim of liver cirrhosis and esophageal varices bleeding, was admitted for hematemesis and tarry stool while panendoscopy revealed a blood-coated varices in the duodenal bull. Undergoing sclerotherapy with Histoacryl injection and observing for 2 more days without evidences of bleeding any longer, he was permitted to leave and keep in resting state for weeks at home. But, he came back for re-bleeding of the duodenal varices, that was stopped with another endoscopic sclerotherapy of same procedure mentioned above. We hereupon discuss the etiology, clinical presentation, radiological finding, endoscopic picture, and therapeutic methods of this disorder.

Keywords: **duodenal varices, sclerotherapy, Histoacryl.**

Upper gastrointestinal (UGI) bleeding from the esophageal or gastric varices is not uncommon, but variceal bleeding from other parts of the GI tract is rare. Duodenal varices was first described by Alberti in 1931 [1]. Since then about 100 cases were reported [2]. Bleeding from duodenal varices could be severe and fatal[3]. Various therapies have been reported to have effects on duodenal varices bleeding, including endoscopic injection sclerotherapy, transjugular intrahepatic portosystemic shunt (TIPS), variceal resection, duodenectomy, and shunt surgery [2,4-10]. We report a case of portal hypertension resulting in duodenal varices bleeding, that was successfully treated with endoscopic sclerotherapy.

CASE REPORT

A 43-year-old male patient was admitted to our hospital because of tarry stool and hematemesis off and on for 7 days. He had alcoholic liver cirrhosis with several episodes of varices bleeding and DM for decades. On admission, his blood pressure was 100/54 mmHg, heart rate 114/min, and respiratory rate 20/min; the conjunctiva was pale, sclera mild icteric, and abdomen soft and not tender. Laboratory data were the following: hemoglobin was 2.9 mg/dl, Hct 9.2%, platelet count 136000/ul, prothrombin time11.2 second, partial prothrombin time 25.0 second, albumin 2.2 g/dl, total bilirubin 1.6 mg/dl, direct bilirubin 0.6 mg/dl, AST 27 U/L, ALT 15 U/L, HbsAg(–), anti-HCV(–), and AFP 2.9 ng/dl.

Emergency panendoscopy revealed F1 esophageal and gastric varices without evidence of

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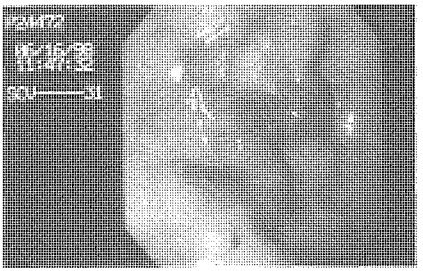

Fig 1. A snake-like varix over anterior wall of duodenal bulb. A suspected hemorrhagic point is seen over the varix (arrow).

bleeding, but 1 snake-like cherry-red varix with a suspected hemorrhagic spot on the anterior wall of duodenal bulb(Fig 1). We injected 1 ml Histoacryl (1:1 diluted with normal saline to 2ml) into the varix at a site near the hemorrhagic spot . Mild oozing was noted in the distal part of the varix, but it spontaneously stopped under observation. On the 2n day, a follow-up panendoscopy demonstrated smooth varix surface except for blood clots coating the distal area. To prevent recurrent bleeding, another 1ml Histoacryl was injected into the blood coated area. He then experienced no more tarry stool passage and left for home several days later. About 2 weeks later, he came back because of another episode of tarry stool . Panendoscopy this time revealed bleeding recurred through the same duodenal varix , and sclerotherapy with Histoacryl was performed again. The following days, his clinical condition was quite smooth, and he was then discharged.

During hospitalization, abdominal sonography revealed liver cirrhosis, and dilatation of common bile duct and pancreatic duct. Abdominal computed tomography(CT) showed a heterogeneous mass (about 5 cm in diameter) in the pancreatic head. Subsequently, celiac and superior mesenteric artery angiography demonstrated gastroduodenal artery and splenic vein occlusion, gastric varices and collateral venous circulation, but no definite tumor stain found.

DISCUSSION

Duodenal varices may lead to upper gastrointestinal bleeding, as esophageal and gastric varices do. Its occurrence could be underrated clinically. Esophageal varices lie usually under the submucosal layer and drain into azygous vein, but duodenal varices may arise from the afferent branches of portal vein via retroperitoneal collaterals and drain into the inferior vena cava. They are usually located in a deeper layer, mainly in the serosa [2].

The causes of duodenal varices include intrahepatic portal hypertension (mainly liver cirrhosis, comprising 34% of cases), extrahepatic portal vein hypertension, splenic vein obstruction, and localized portal hypertension (as in a fistula between aneurysm of hepatic artery and portal vein) [2,3]. In cases of intrahepatic portal hypertension secondary to liver cirrhosis, the collateral veins drain blood from the liver and anastomose with systemic veins, producing hepatofugal flow; duodenal varices therefore coexist with esophageal varices. In cases of extrahepatic portal vein obstruction, when intravenous pressure elevated distal to the obstruction site, the pressure gradient actually induced hepatopedal flow; thus, isolated duodenal varices may be present[2,11].

Duodenal varices mostly occur in the bulb, rather in the second portion, that is the next most common site[3]. However, varices in other portions of the duodenum were also reported[2,3]. Their presentation under endoscopy has been described in the literature as tortuous bluish columns or a cherry-red mucosal protuberance[4,6,12]. Differential diagnosis of duodenal varices include duodenal neoplasm, adenomatous polyp, bulb deformity with ulcer, and AV malformation[2,3]. Gastrointestinal series x-ray may demonstrate varices as band-like or circoid, horseshoe filling defects within the lumen [1,11]. Endoscopic ultrasound (EUS) is undoubtedly a good diagnostic tool, that reveals varix appears as a rounded, echo free structure usually. Besides, EUS allows visualization of a large part of the portal system and its collaterals[13]. Color doppler sonography can also detect duodenal varices, revealing a thickened duodenal wall containing slow blood flow , possibly with a concomitant portal thrombus[14]. Selective mesenteric angiography or splenoportography may

also be used as a diagnostic method, showing large collaterals in duodenal area; but with a lower sensitivity of about 50%[11].

Management of duodednal varices bleeding remains controversial. Systemic vasopressin and somatostatin can be tried, but have only little effect. Endoscopic ligation inevitably leads to rebleeding, so it is not currently suggested. Endoscopic sclerotherapy with thrombin, polidocanol, ethanolamine oleate, sodium tetradecyl sulfate, or histoacryl has been successfully used[4-6,12]. Sclerotherapy becomes the first line therapy nowadays; however, it is not without risk. Because of the thin duodenal wall, perforation could happen by accident. Systemic embolism could also happen, since the varices flow directly drains into the inferior vana cava. Percutaneous transhepatic portal obliteration has been used successfully as an alternate, though it carries the risk of distant embolism [7]. Transjugular intrahepatic portosystemic shunt (TIPS) can be used if endoscopic therapy failed[8,9]; but, the complication of cerebral embolism has been reported[15]. The more definite therapy is surgical intervention, including direct ligation of the varix, varix resection, and portosystemic shunt, which can be performed in certain patients [10].

In conclusion, duodenal varices bleeding is a potentially fatal disease. In patients with portal hypertension and UGI bleeding, if no bleeding point was found in the stomach or esophagus, the duodenum should be examined carefully for duodenal varices. Sclerotherapy with Histoacryl appears to be an effective therapy to stop duodenal varices bleeding through our experience in the present case, though its long term hemostasis remains further observation.

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以Histoacryl治療十二指腸靜脈瘤出血一病例報告

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臨床上，十二指腸靜脈瘤出血遠較食道及胃靜脈瘤少見，但可能造成大量出血，並且危及生命。我們報告一個病例：43歲男性病人，曾因酒精性肝硬化及食道靜脈瘤出血住院治療；此次因為吐血及黑便而至急診求診，經內視鏡檢查診斷為十二指腸球部靜脈瘤出血。經由內視鏡注射Histoacryl之後止血成功，而出院。兩週後，再度出血而住院；再一次實施內視鏡注射Histoacryl止血。之後追蹤至今，未再復發。我們於此討論十二指腸靜脈瘤的成因，臨床上、放射學上，以及內視鏡上的表現，與其治療方式。

關鍵詞：十二指腸靜脈瘤、硬化治療、Histoacryl