

Ruptured duodenal varices arising from the main portal vein successfully treated with endoscopic injection sclerotherapy: a case report

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Duodenal varices result from retroperitoneal portosystemic shunts that usually come from the pancreaticoduodenal vein and drain into the inferior vena cava. Because they are a rare but fatal cause of gastrointestinal bleeding, a prompt hemostatic intervention is mandatory. A 62-year-old man who had a history of excessive alcohol consumption presented with massive hematemesis and melena. Emergent endoscopy revealed ruptured varices with an adhering whitish fibrin clot on the postbulbar portion of the duodenum. Abdominal computed tomography demonstrated a cirrhotic liver with venous collaterals around the duodenum and extravasated contrast in the second and third portions. The collaterals originated from the main portal vein and drained via the right renal vein into the inferior vena cava. Endoscopic injection sclerotherapy with cyanoacrylate was successful in achieving hemostasis, and resulted in the near eradication of duodenal varices at a 6-month follow-up. ([Korean J Hepatol 2011;17:152-156](#))

Keywords: Duodenal varices; Endoscopic injection sclerotherapy; Portal hypertension; Cirrhosis

INTRODUCTION

Duodenal varices represent an ectopic portosystemic shunt, usually originating from pancreaticoduodenal vein.¹ Although duodenal varices are a rare cause of gastrointestinal bleeding, a greater than 40% mortality rate has been reported after the initial bleeding episode.² For bleeding control there are several treatment modalities including endoscopic treatments, radiologic interventions and surgical procedures. However, it is still unclear which option would be the best for treatment of bleeding duodenal varices. Here we report a case of massive bleeding from the ruptured duodenal varices, directly arising from main portal vein, which was successfully treated with endoscopic injection sclerotherapy (EIS).

CASE REPORT

A 62-year old man who had a history of excessive alcohol consumption presented with massive hematemesis and melena. He had no medical history of other systemic disease. On arrival at emergency room, his systolic blood pressure was about 45 mmHg. Melena was seen on digital rectal examination and fresh blood was drained through L-tube during gastric irrigation. Complete blood counts showed hemoglobin of 3.6 g/dL and platelet count of 68,000/mm³. Other laboratory tests were followings; total bilirubin 0.9 mg/dL, albumin 1.5 g/dL, prothrombin time 18.8 seconds. Child-Pugh's classification was B. Hepatitis B surface antigen, anti-hepatitis C virus antibody and anti-human immunodeficiency virus antibody were all seronegative.

An emergent upper endoscopy was immediately performed

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Abbreviations: BRTO, balloon-occluded retrograde transvenous obliteration; CT, computed tomography; EIS, endoscopic injection sclerotherapy; EVL, endoscopic variceal ligation; TIPS, transjugular intrahepatic portosystemic shunt

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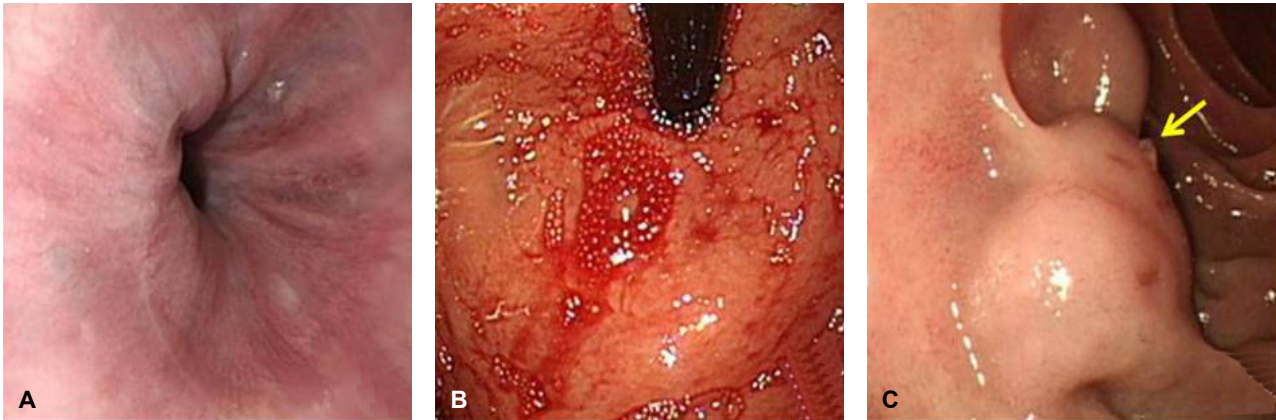


Figure 1. Initial endoscopy results. (A) Minimal esophageal varices without stigmata of recent hemorrhage. (B) Blood-stained cardiac and fundal gastric mucosae without evidence of varices. (C) Dumbbell-shaped varices with an adhering whitish fibrin clot (arrow) on the postbulbar portion of the duodenum.

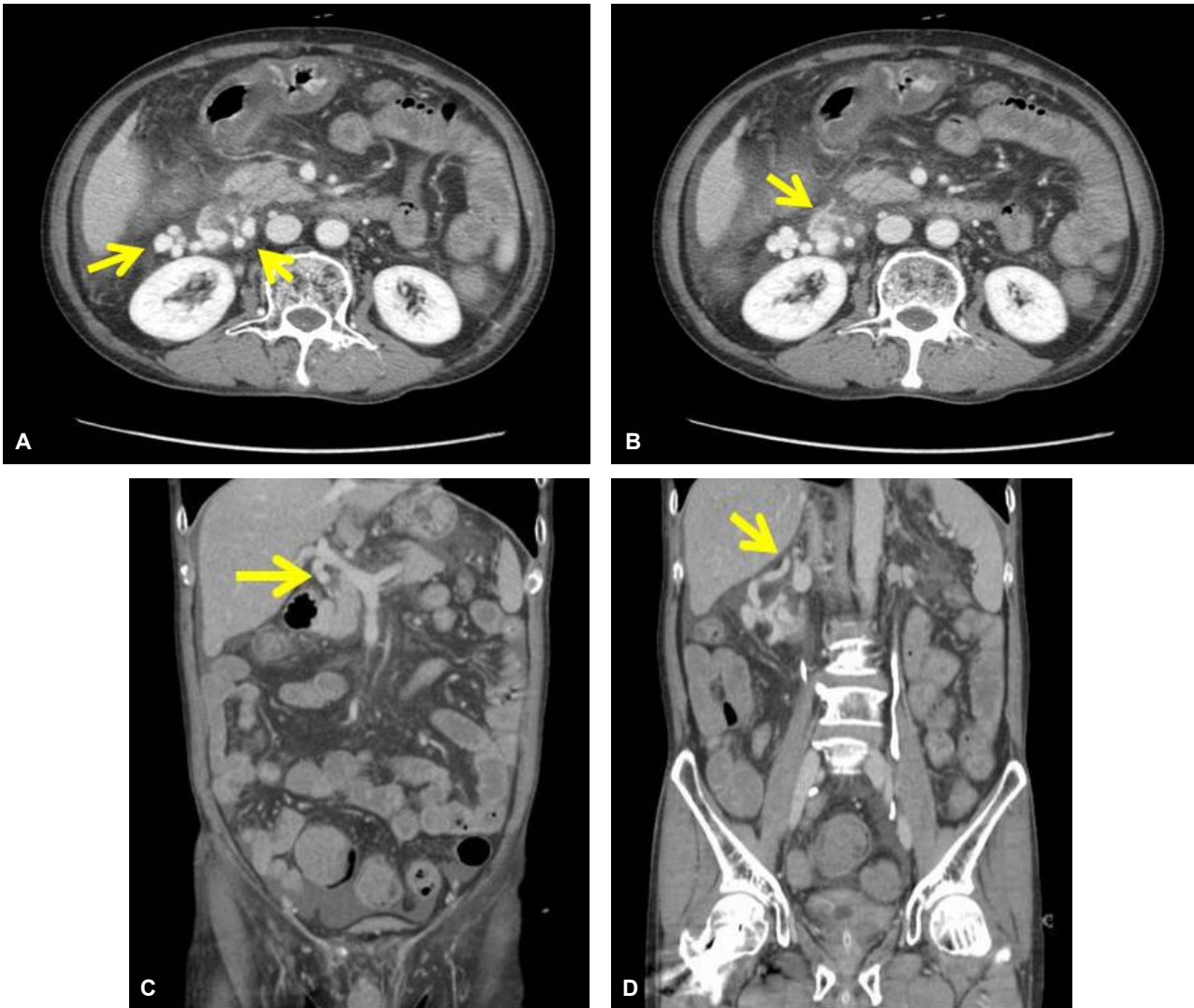


Figure 2. Initial abdominal CT results. (A) Venous collaterals (arrow) around the duodenum. (B) Extravasated contrast (arrow) in the second and third portions of the duodenum. (C) The afferent collateral vessel (arrow) originated directly from the main portal vein. (D) The efferent collateral vessel (arrow) drained throughout the inferior vena cava via the right renal vein.

following fluid resuscitation with blood transfusion. It revealed minimal esophageal varices without a stigmata of hemorrhage (Fig. 1A) and blood-stained gastric mucosae with no evidence of the cardiac or fundal varices (Fig. 1B). Duodenal ulcer scar with marked luminal narrowing was noted on the bulb, however, the endoscope can pass through the narrow lumen. Dumbbell-shaped ruptured varices with an adhering whitish fibrin clot were seen on the postbulbar portion of the duodenum (Fig. 1C). The duodenal varices were collapsed during air inflation and engorged with suction of the air. Abdominal computed tomography (CT) demonstrated liver cirrhosis with splenomegaly and moderate amount of ascites. Venous collaterals around the duodenum with extravasated contrast in the second and third portion were seen and the collaterals originated from main portal vein and drained throughout the inferior vena cava via the right renal vein (Fig. 2A-D).

For hemostasis, we intravariceally injected 1.0 mL of cyanoacrylate into the duodenal varices. After EIS, the bulging varices were not collapsed during luminal aeration. The patient was getting hemodynamically stable and hemoglobin level was recovered up to 8.9 g/dL with transfusion of the packed red blood cells.

An endoscopy after 5 days showed a tubular-shaped venous bulging, by previous injection of cyanoacrylate,

with a hard consistency and hyperemic covering mucosa. Endoscopic biopsy was done at the ulcer scar on the bulb, showing a chronic inflammation with granulation tissue microscopically. After 2 weeks, an endoscopy revealed post-EIS ulceration with yellow plaque and venous bulging.

Six months later, a follow-up endoscopy revealed much collapsed duodenal varices with small yellow plaque on the surface (Fig. 3A). Abdominal CT disclosed the collapsed and much decreased portosystemic venous collaterals in the retroperitoneum of the paraduodenal space (Fig. 3B). No bleeding has been detected by this time as of 9 months after the initial bleeding episode.

DISCUSSION

Portosystemic communications in splanchnic hypertension occur through the several venous routes, and the duodenal varices are one of ectopic varices resulting from retroperitoneal portosystemic shunts. These are caused by increased hepatofugal blood flow through cystic branches of superior mesenteric vein, superior and inferior pancreaticoduodenal veins, gastroduodenal veins, and pyloric veins.¹ In ectopic duodenal varices of this present patient, the afferent vessel originated from the main portal vein and the efferent vein drained into the inferior vena cava via the right

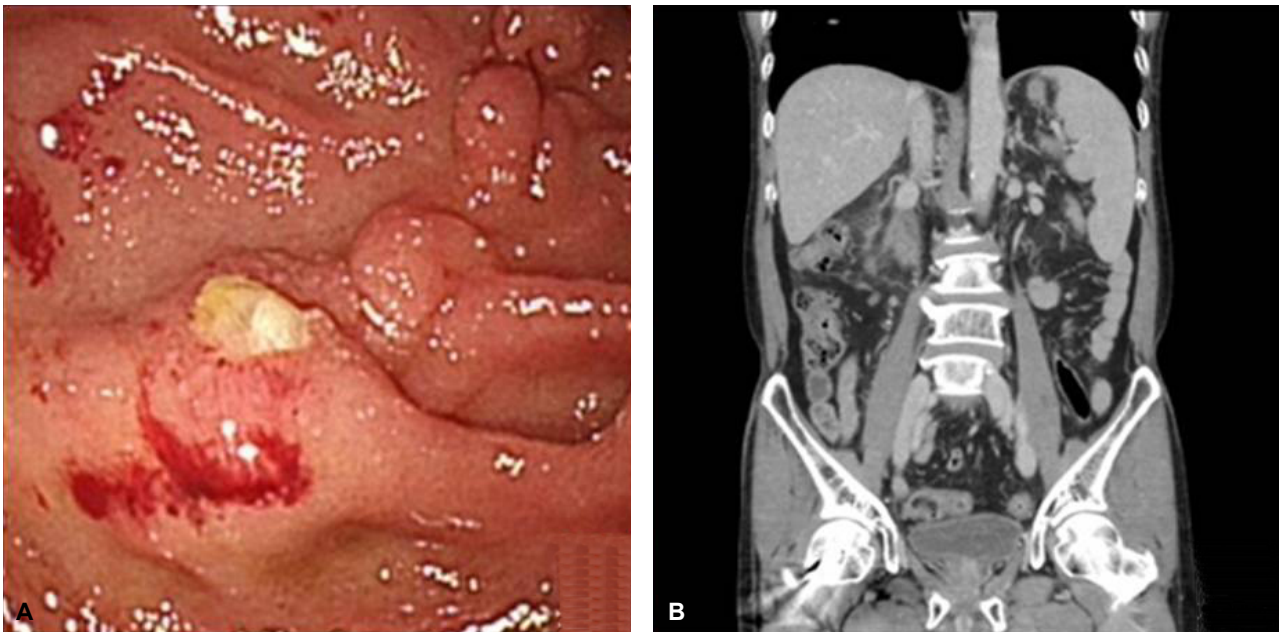


Figure 3. Results of follow-up endoscopy and CT performed 6 months after EIS. (A) Endoscopy revealed more collapsed varices with small yellow plaques on the postbulbar portion of the duodenum. (B) Abdominal CT disclosed collapsed and greatly decreased portosystemic venous collaterals in the retroperitoneum of the paraduodenal space.

renal vein. To our knowledge this is the first case of duodenal varices directly arising from the main portal vein, confirmed by radiologic imaging.

The most common cause of duodenal varices is portal hypertension in cirrhotic liver and the others are obstruction of splenic vein, portal vein, superior mesenteric vein or inferior vena cava.³ Biliary cirrhosis can also rarely cause duodenal varices by the same mechanism in cirrhotic patients from other causes.⁴ Lebrec and Behamou have reported that an extrahepatic etiology of portal hypertension accounted for 20-30% of the patients with duodenal varices.⁵ In the present case, the patient had laboratory (thrombocytopenia, hypoalbuminemia and prolongation of prothrombin time), and radiologic findings (ascites and splenomegaly) corresponding to liver cirrhosis with obvious portal hypertension.

In review by Amin et al, most of the duodenal varices are located in the duodenal bulb and they may also occur in the second and third portions of the duodenum.⁶ Liu et al reported, however, they could see almost all of duodenal varices in the descending part of the duodenum.⁷ The discrepancy of occurrence site was thought to be attributable to the ethnicity of the patients and/or the experience of the endoscopists.⁷ Nineteen of the 21 cases with duodenal varices in Korea were located in the second portion of the duodenum,⁸ and the varices in this present case was also located in the postbulbar portion of the duodenum. Approximately 50-60% of the patients with duodenal varices have concomitant gastroesophageal varices.⁹

Various treatment modalities have been reported to be available for bleeding from duodenal varices, including endoscopic variceal ligation (EVL), EIS, transjugular intrahepatic portosystemic shunt (TIPS), balloon-occluded retrograde transvenous obliteration (BRTO), beta-blocker therapy, and resection of a segment of bleeding site.¹⁰⁻¹⁴ Although TIPS or BRTO can be performed in the hemodynamically unstable patients whose endoscopic procedure cannot secure a clear view because of massive hemorrhage, these radiologic interventions have some shortcomings to perform; a significant mortality rate, shunt occlusion, hepatic encephalopathy, renal failure or worsening of esophageal varices. Also these procedures should be performed by well-trained radiologists. Therefore, over the last few years, it seems that endoscopic interventions have been more frequently performed, because it is less invasive, easier and

faster to perform, and more effort-effective than other treatment options. Several reports suggest that EVL is effective for treatment of bleeding from duodenal varices.¹⁵⁻¹⁷ However, it can be difficult to perform in some location, and rebleeding can occur by post-EVL ulcer.¹⁶ And also it is difficult to achieve complete long-term eradication of varices with EVL alone, because of the insufficient effect of EVL on the feeding collateral vessels.¹⁸ EIS has been performed for treatment of bleeding duodenal varices and suggested as a practical and effective measure.^{7,13} However, it carries a risk of perforation, tissue injury, and embolism. Among several sclerosants such as cyanoacrylate, ethanolamine oleate, polidocanol, sodium tetradecyl sulfate, and fibrin,¹³ adopted for EIS, cyanoacrylate and ethanolamine oleate are frequently used. Ethanolamine oleate disturbs the endothelial cells, induces a coagulation necrosis, and directly promotes thrombus formation. However, this compound can lead to penetration or perforation of the thin duodenal wall.¹⁹

Accordingly, some investigators prefer cyanoacrylate which causes less tissue injury. Lee et al reported that a combination therapy of the EVL and EIS was applied to all of 4 cases resulting in the successful hemostasis and eradication of duodenal varices.⁸ Currently, no best treatment option for controlling duodenal varices has been proposed and verified. In the maelstrom of treatment debate, we chose a cyanoacrylate as a sclerosant for EIS, performed bleeding control successfully without any complication, and eradicated the duodenal varices radiologically and endoscopically.

We report a case of endoscopic treatment in a patient with a massive bleeding from duodenal varices directly arising from the main portal vein. We recommend EIS with cyanoacrylate as a first line of treatment for bleeding duodenal varices.

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