CASE REPORT

Balloon occluded retrograde transvenous obliteration and percutaneous transhepatic obliteration for ruptured duodenal varices after operation for rectal cancer with multiple liver metastasis : report of a case

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Abstract : We report a patient with duodenal varices oozing blood who had undergone low anterior resection of the rectum and resection of the liver tumor because of multiple liver metastasis from rectal cancer 80 months previously. Although endoscopic variceal ligation (EVL) was carried out for the ruptured duodenal varices, their bleeding persisted and hepatic encephalopathy also appeared. Finally, balloon occluded retrograde transvenous obliteration (BRTO) with percutaneous transhepatic obliteration (PTO) was carried out for the duodenal varices. Percutaneous transhepatic portography revealed detailed hemodynamics. Following PTO, the duodenal varices were stagnated by BRTO, and no complications were recognized. No re-bleeding episode has been observed since the treatment. In addition, the hepatic encephalopathy was also improved. J. Med. Invest. 52 : 212-217, August, 2005

Keywords **:** *duodenal varices, portal hypertension, balloon occluded retrograde transvenous obliteration, percutaneous transhepatic obliteration*

INTRODUCTION

Duodenal variceal bleeding is very rare in patients with portal hypertension. Though re-bleeding or complications are reported concerning non-surgical treatments for ruptured duodenal varices (1-3), there appears to be only been one reported balloon occluded retrograde transvenous obliteration (BRTO) with percutaneous transhepatic obliteration (PTO) for a duodenal varix suggestive of bleeding with liver cirrhosis due to hepatitis C virus infection (4).

In this report, we describe a case with ruptured duodenal varices, which appeared postoperatively following multiple liver metastasis from rectal cancer, that was successfully treated by BRTO combined with PTO.

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CASE REPORT

A 39-year-old woman was admitted to our hospital for melena and hematemesis. At age of 33, because of rectal cancer with multiple liver metastasis she had undergone low anterior resection of the rectum and resection of the right hepatic lobe ; the upper part of the medial segment and the superior area of lateral

segment (Figure 1). The patient received oral tegafur/uracil, and mitomycin-C via the hepatic artery as postoperative adjuvant chemotherapy. She was relatively well until the current hospitalization.

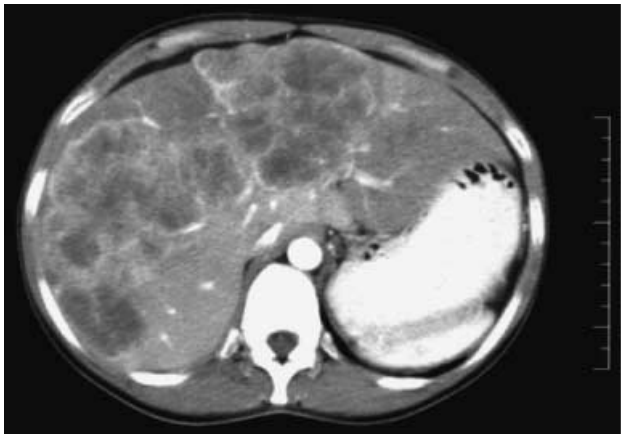

Fig.1. Preoperative computed tomography scan of the abdomen reveals multiple liver metastasis, which occupy the greater part of the liver.

At admission, blood pressure was 88/50 mm Hg and heart rate 106/min. There was no scleral jaundice. Cardiopulmonary examination was unremarkable except for tachycardia. Melena was evident on rectal examination. The initial hemoglobin was 7.7 g/dl, platelet count 68,000/mm^3, albumin 3.0 g/dl, total bilirubin 0.8 mg/dl, aspartate aminotransferase 52 U/L, alanine aminotransferase 23 U/L, blood urea nitrogen 24 mg/dl, and creatinine 0.42 mg/dl. The patient had no viral hepatitis or immunoserological markers, nor any history of alcohol consumption.

Emergent esophagogastro-duodenoscopy revealed no esophageal varices. A small amount of pooled blood was recognized in the stomach ; being superficial erosive gastritis with oozing blood. Furthermore, duodenal varices in the second portion were detected (Figure 2A). A computed tomography examination showed a hypertrophyic remnant lobe (Figure 3A) and marked duodenal varices were located in the lumen of the duodenum (Figure 3B). No metastatic nodules were revealed in the remnant liver.

Anemia progressed after admission, and esophagogastro-duodenoscopy revealed duodenal varices oozing blood. Endoscopic variceal ligation (EVL) was performed for the duodenal varices. Hematemesis and melena appeared 13 days after the EVL, and a second EVL was carried out for the varices in the duodenal second portion because of re-bleeding. No hemorrhage from the duodenal varices was recognized for 33 days after the second EVL,

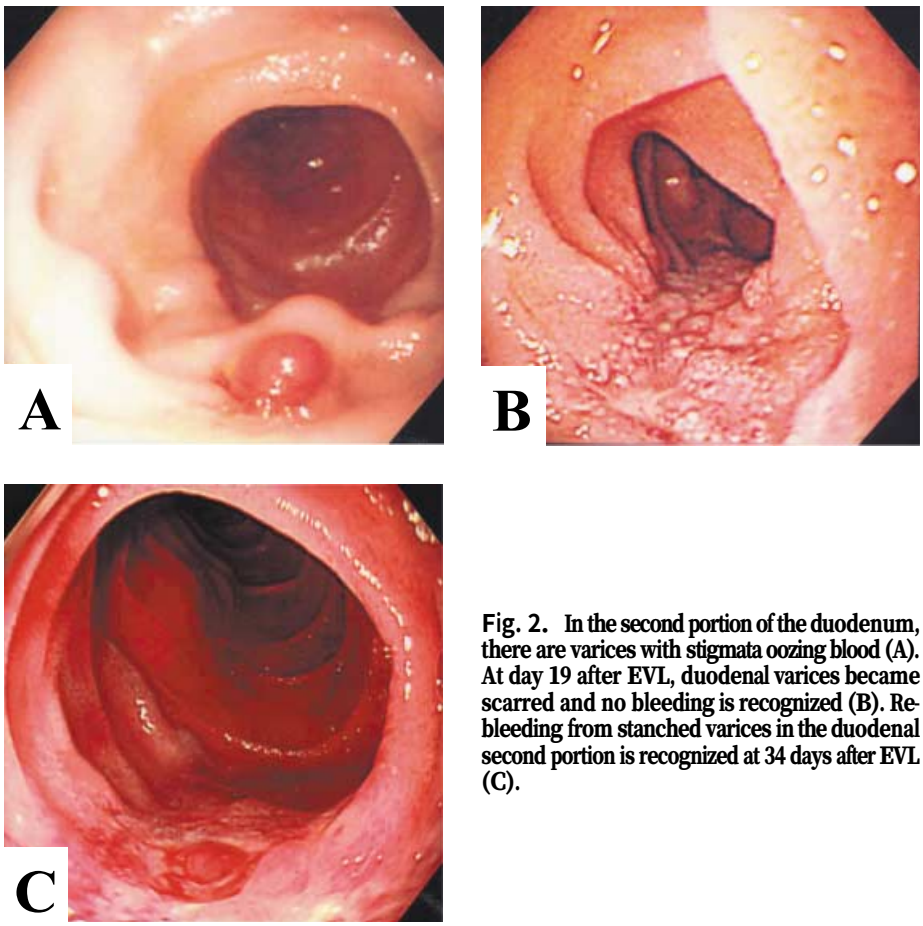

Fig. 2. In the second portion of the duodenum, there are varices with stigmata oozing blood (A). At day 19 after EVL, duodenal varices became scarred and no bleeding is recognized (B). Re-bleeding from stanched varices in the duodenal second portion is recognized at 34 days after EVL (C).

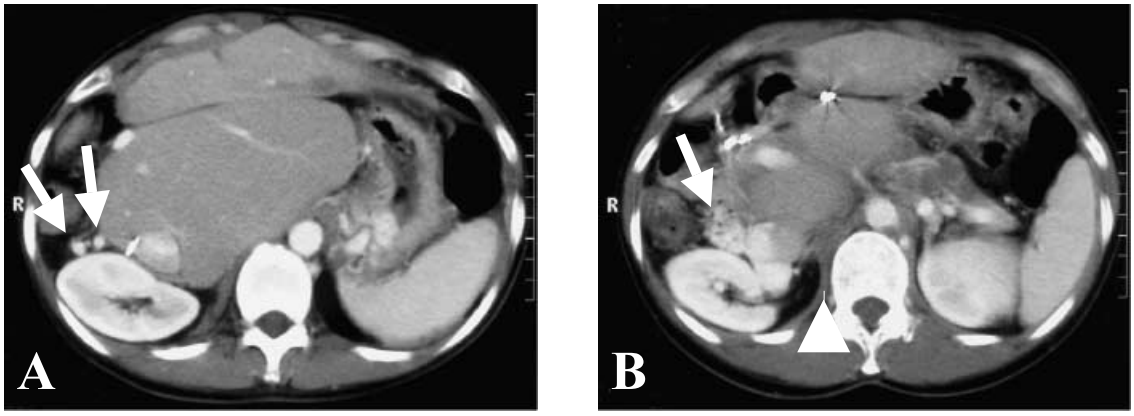

Fig. 3. Computed tomography scan of the abdomen reveals markedly hypertrophic remnant liver (the caudate and part of left lobes), a low density area as a postoperative change in the lateral segment, and the inferior vena cava displaced on the right side. Collateral vessels and portosystemic shunt via the duodenal varices are observed (white arrows) (A). Contrast enhanced duodenal varices are located in the lumen of the duodenum (white arrow) (B).

and the duodenal wall became scarred (Figure 2B). Hemorrhage from the stanched varices in the duodenal second portion was recognized at the 34th day after EVL (Figure 2C). Although EVL was carried out again for the duodenal varices, hemostasis was unsuccessful because of the hard scar tissue formation after the series of EVLs ; as well, hepatic encephalopathy appeared.

Because the hemodynamics around the duodenal varices were unclear using transarterial portography and a sclerosing agent might have leaked into the systemic circulation or the portal vein via the multiple drainage or feeding vessels of the duodenal varices, percutaneous transhepatic portography (PTP) was performed via the portal branch of the hypertrophied caudate lobe using an abdominal ultrasonographic guide. The duodenal varices originated in the portal vein via some afferent vessels and drained into the efferent vein (Figure 4A). There was neither oppressive, stenotic, no blocking image in the portal vein. Portal vein pressure was 30 cm H_2O. A 5-Fr balloon catheter (MOIYAN, GOODTEC, Gifu, Japan) was inserted into the drainage vein through the right femoral vein according to the Seldinger technique. The efferent vein (diameter 6 mm) drained directly into the inferior vena cava, located a little to the cranial side of a right renal vein (Figure 4B). Using retrograde transvenous venography, the efferent vessels were revealed in more detail than with PTP. However, retrograde transvenous venography did not reveal the afferent vessels of the duodenal varices. To effectively pool the sclerosing agent in

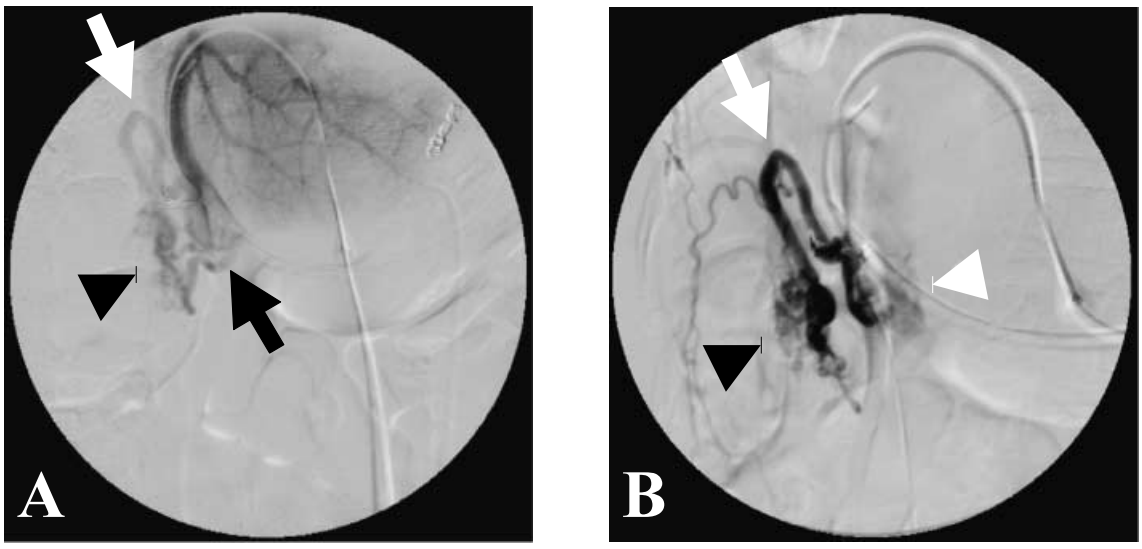

Fig. 4. Percutaneous transhepatic portography (PTP) was performed via the portal vein branch of the caudate lobe, which was obviously hypertrophied due to an operation. The afferent vessels (black arrow) from the portal vein form varices (black arrow head), and drain into the efferent vein (white arrow) (A). Using retrograde transvenous venography, the duodenal varices (black arrow head) and the efferent vein (white arrow) are shown to directly drain into the inferior vena cava (white arrow head) ; and in more detail than using PTP (B).

the entire portosystemic shunt, PTO was initially carried out. A microcatheter (Progreat Micro Catheter, TERUMO, Tokyo, Japan) was advanced into these afferent vessels to perform microcoil occlusion. Microcoils (TORNADO EMBOLIZATION MICROCOIL, COOK, Bloomington, Minnesota, USA) were placed in the afferent vessels (Figure 5A). Next, balloon occluded retrograde transvenous venography was performed to confirm that the contrast medium had not leaked out of the estimated area and to better ascertain the right amount of sclerosing agent to use for obliteration. Using a microcatheter, 5% ethanolamine oleate with iopamidol (total volume, 4.0 ml) was injected into the portosystemic shunt. The sclerosing agent had not leaked out of the estimated area (Figure 5A), and the balloon was inflated for 24h to completely pool the sclerosing agent. Twenty-four hours after treatment, balloon occluded retrograde transvenous venography was performed to confirm complete hemostasis and to assess whether there were any new efferent vessels ; this showed that the contrast medium had not completely flowed into the vessels. By retrograde transvenous venography (collapsed balloon), only part of the afferent vessels were imaged retrogradely (Figure 5B). Laboratory data on the 4th post-treatment day indicated that the range of the ammonia was lowered from 161 μg/dl to 48 μg/dl (normal range, 12-66 μg/dl). Grade II hepatic encephalopathy was also disappeared. No rebleeding episode has been observed since treatment. However, the patient died of a metastatic brain tumor 11 weeks after BRTO with PTO for the duodenal varices.

DISCUSSION

The etiology of the duodenal varices is mostly portal hypertension due to cirrhosis, idiopathic portal hypertension, or extra hepatic portal obstruction (5). Although temporary portal hypertension after hepatectomy is reported (6), no report on permanent portal hypertension was found. After surgery for rectal cancer with hepatic metastasis, our patient received 400 mg of oral UFT® daily and administration of 6 mg of mitomycin-C 6 weekly via the hepatic artery as postoperative adjuvant chemotherapy. Chronic active hepatitis and liver cirrhosis induced by the combination of tegafur and tamoxifen (7) or by UFT® alone (8, 9) have been reported. Functional abnormalities of the liver have occasionally been reported following normal doses of mitomycin-C; and large doses of mitomycin-C induce veno-occlusive disease or hepatitis (10). Harmful side effects can result in liver cirrhosis ; which might occur in the period in which there is cell division after hepatectomy using anticancer drugs.

There are case reports concerning ruptured duodenal varix successfully managed by EVL (11) and successful BRTO for re-bleeding from duodenal varices after EVL (2). Endoscopic variceal sclerotherapy (EIS) with thrombin, polidocanol, ethanolamine oleate, sodium tetradecyl sulfate, and n-butyl-2-cyanoacrylate, and α-cyanoacrylate as primary care in the hemorrhage from duodenal varices is reported (12-14). For bleeding esophageal varices, a few randomized controlled trials that compared EIS with EVL, have favored EVL for

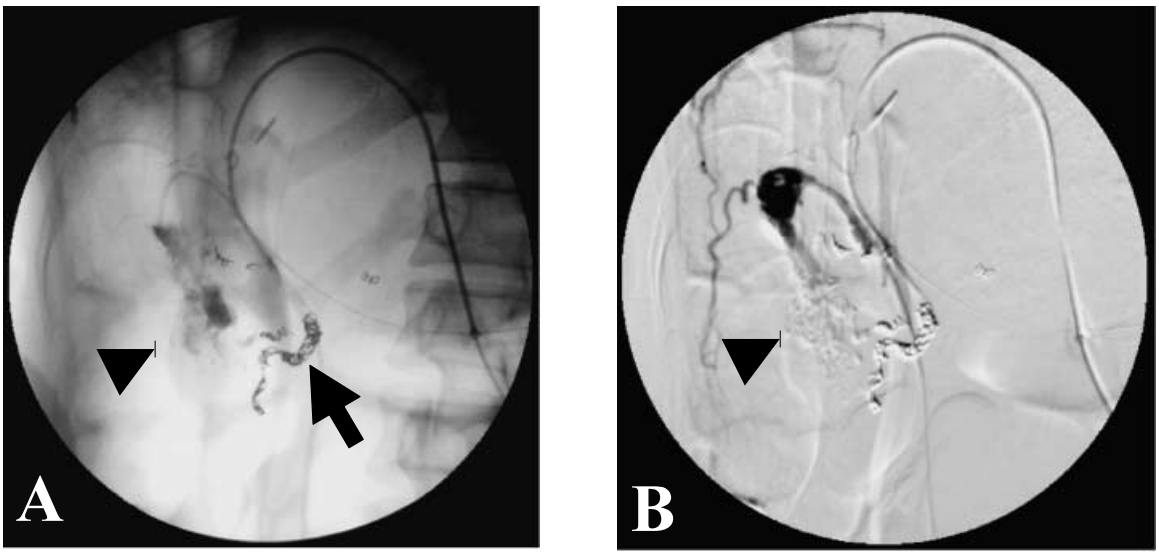

Fig. 5. Microcoils were placed in the afferent vessels (black arrows) via the efferent vein, 4 ml of 5% ethanolamine oleate with iopamidol was injected under superselective catheterization into duodenal varices (black arrow head) (A). Retrograde transvenous venography at 24 h after treatment revealed that duodenal varices were not imaged (black arrow head) and no newly efferent vessels were found (B).

decreasing the risk of re-bleeding with fewer complications (15 -18). Further, urgent endoscopic examination is indispensable when upper gastrointestinal bleeding is suspected. Therefore, EVL seems to be a first choice, but a randomized controlled trial would be necessary to establish a therapy for duodenal varices. For repeated hemorrhage in spite of a series of EVL, another treatment should be chosen.

While carrying out this procedure, there is some possibility of complications, such as renal dysfunction, pulmonary embolism, pleural effusion, pulmonary edema, hypertensitivity reaction, and pyrexia. It is important to prevent the sclerosing agent had not leaked out of the estimate area. Ethanolamine oleate promptly binds with albumin in the blood, thus becoming inactive, and decomposes. It is necessary to revise hypoalbuminemia in advance. Ethanolamine oleate also causes hemolysis of the blood vessels, leading free hemoglobin, which may give rise renal dysfunction. Intravenous administration of haptoglobin, which combines with free hemoglobin prevents renal dysfunction associated with hemolysis.

PTP was performed because the hemodynamics around the duodenal varices were unclear from transarterial portography, and it is possible that the sclerosing agent might have leaked into the systemic circulation or a portal vein via the multiple drainage or feeding vessels of the duodenal varices. In this case, PTP could reveal the detailed hemodynamics. Following PTO, the sclerosing agent had not leaked into the systemic circulation or portal vein via the multiple drainage or feeding vessels of the duodenal varices, which appeared stagnated by BRTO. Hepatic encephalopathy also improved. There were no complications after PTO, and BRTO. For duodenal varices, this method by which afferent and efferent vessels including varices are embolized, is an excellent therapy from the viewpoints of safety and certainty.

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