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Retrograde Transvenous Obliteration of Gastric Varices[1]

PURPOSE: To evaluate the clinical efficacy, techniques, and complications associated with balloon-occluded retrograde transvenous obliteration of gastric varices.

MATERIALS AND METHODS: Between December 1994 and November 1997, balloon-occluded retrograde transvenous obliteration was performed on 20 patients with gastric varices in danger of rupture and with gastrorenal shunts; three patients also had hepatic encephalopathy. The sclerosant was injected into the gastric varices during balloon occlusion. The degree of progression of the gastric varices and of collateral veins was classified into five grades, with grade 1 being least progression and grade 5 most progression; collateral veins that had developed were treated with embolization. Follow-up consisted of fiberoptic endoscopy and computed tomography.

RESULTS: Technical success was achieved in all patients. Occlusion of collateral veins was essential for the occlusion of gastric varices with a grade greater than grade 2. The clinical symptoms of hepatic encephalopathy in the three patients improved remarkably. Follow-up endoscopy 3 months after the procedure revealed the disappearance of gastric varices in 15 patients and reduced variceal size in five. During the follow-up period, 19 patients had no recurrence of gastric varices; three patients had aggravation of the esophageal varices.

CONCLUSION: Balloon-occluded retrograde transvenous obliteration is a feasible alternative to a transjugular intrahepatic portosystemic shunt for patients with large gastrorenal shunts or hepatic encephalopathy (or both).

Recent advances in interventional radiology for esophageal varices in patients with portal hypertension have introduced new concepts such as the transjugular intrahepatic portosystemic shunt (1), while endoscopic injection sclerotherapy has become the treatment of choice for the management of esophageal varices and is now widely performed. However, gastric fundal varices located in the fornix cannot be treated effectively with endoscopic injection sclerotherapy (2–5) because of a rapid loss of the sclerosing agent as a result of the fast blood flow in these varices, and such varices are associated with a mortality rate as high as 45% (3) when they rupture (6,7).

For this reason, surgery or a transjugular intrahepatic portosystemic shunt procedure has been performed for gastric fundal varices. However, poor hepatic functional reserve is a contraindication for surgery, and a transjugular intrahepatic portosystemic shunt does not always result in regression of gastric fundal varices, as evidenced by a reported success rate of 50% (8). In addition, patients with severe hepatic encephalopathy are not always recommended for a transjugular intrahepatic portosystemic shunt procedure because of possible aggravation of the encephalopathy (9–11).

For these reasons, a minimally invasive interventional radiologic procedure to occlude gastric varices through a gastrorenal shunt, known as balloon-occluded retrograde transvenous obliteration of varices, was introduced by Kanagawa et al (12), and satisfactory results were reported for patients with gastric varices or hepatic encephalopathy (or both) (13). The present study was performed to evaluate the clinical efficacy, technique, and complications associated with balloon-occluded retrograde transvenous obliteration of gastric varices.

MATERIALS AND METHODS

Patients

Between December 1994 and November 1997 in our department, balloon-occluded retrograde transvenous obliteration was performed on 20 patients (15 men and five

women; age range, 46–68 years; mean age, 59.5 years) with gastric varices in danger of rupture as shown by endoscopic findings, with or without a history of bleeding gastric varices, and the presence of dilatation of the gastrorenal shunt or moderate to severe hepatic encephalopathy (or both). Patients who had splenorenal shunts connecting with the left adrenal vein and had coronary vein opacification seen at adrenal venography during balloon occlusion were excluded from this procedure because of the risk of thrombus formation in the portal vein caused by overflow of the sclerosing agent. Informed consent was obtained from all patients.

The gastric varices were classified according to criteria based on gastroendoscopic findings in Japan (14,15), as follows: Lg-c, adjacent to the cardiac ring; Lg-f, separated from the cardiac ring; and Lg-cf, continuing from cardiac ring to gastric fundus. Seven patients had gastric varices that were classified as Lg-f, and 13 patients had varices classified as Lg-cf.

Eleven patients had esophageal varices that had been treated with endoscopic injection sclerotherapy, and five patients had a history of rupture. Sixteen patients had viral hepatitis, one patient had primary biliary cirrhosis, and three patients had alcoholic hepatitis. The Child classification of hepatic function was used to identify four patients with class A, 11 with class B, and five with class C. Four patients had complicating hepatocellular carcinoma, which had been treated and effectively controlled with transcatheter arterial embolization, one patient had undergone resection of an esophageal cancer, and three patients had a history of hepatic encephalopathy.

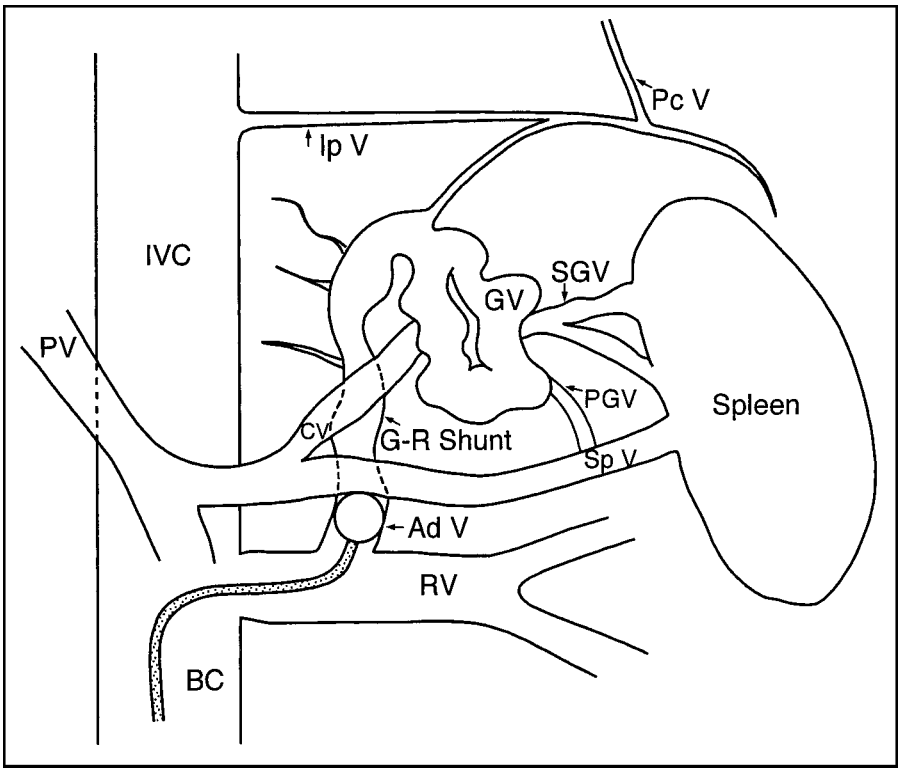


Figure 1. Diagram of the hemodynamics of the gastric fundal varices and collateral veins. *Ad V* = adrenal vein, *BC* = balloon catheter, *CV* = coronary vein, *Ip V* = inferior phrenic vein, *IVC* = inferior vena cava, *G-R* = gastrorenal, *GV* = gastric varices, *Pc V* = pericardiacophrenic vein, *PGV* = posterior gastric vein, *PV* = portal vein, *RV* = renal vein, *SGV* = short gastric vein, *Sp V* = splenic vein.

Procedure

The procedure to be followed for connecting the gastric varices to the left adrenal vein through a gastrorenal shunt and the diameter of the shunt were investigated before surgery with contrast medium–enhanced spiral computed tomographic (CT) scanning in the portal phase; the contrast medium used was iopamidol (Iopamiron; Schering Japan, Osaka, Japan). The contrast-enhanced spiral CT scan revealed that one of the varicose veins around the gastric fornix ran posteriorly and caudally to empty into the inferior phrenic vein, which in turn connected to the left adrenal vein.

The sclerosing agent consisted of a mixture of 10% ethanolamine oleate (Odamin; Mochida Pharmaceutical, Tokyo, Japan) and the same dose of a nonionic contrast medium (350 mg of iodine; iopamidol) and produced radiopacity. Intravenous administration of 4,000 units of human haptoglobin (Green Cross, Osaka, Japan), 1 unit of which binds 1 mg of hemoglobin, was performed during the procedure to prevent hemolysis and subsequent renal failure, which may be induced by ethanolamine oleate (16).

A 6-F balloon catheter with a balloon 20 mm in diameter (Clinical Supply, Gifu, Japan) was inserted from either the right femoral or the right internal jugular vein and was wedged into the left adrenal vein. Then left adrenal venography was performed with the balloon inflated. Sometimes, the gastric varices were not opacified because of blood flow from the gastric varices to the dilated collateral veins, such as the inferior phrenic or pericardiacophrenic vein (Fig 1).

We classified the degree of progression of the gastric varices and collateral veins into five grades (Fig 2) according to the results of adrenal venography during balloon occlusion: grade 1, gastric varices were well opacified without evidence of collateral veins (Fig 3); grade 2, collateral veins were small and few in number, and the contrast medium remained in the gastric varices for 3 minutes or more (Fig 4); grade 3, collateral veins were medium to large, there were few veins, and the contrast medium filled the gastric varices only partially and disappeared within 3 minutes (Fig 5); grade 4, there were many large collateral veins, and the gastric varices were not opacified (Fig 6); and grade 5, the left adrenal vein could not be occluded with the balloon catheter because of a very large gastrorenal shunt with rapid blood flow.

For patients with grade 2 progression of gastric varices and collateral veins, we could occlude the collateral veins with only injection of the ethanolamine oleate–iopamidol mixture through a 6-F balloon catheter during balloon occlusion of the left adrenal vein. For patients with a collateral vein grade of 3 or higher, we occluded these veins with an injection of the ethanolamine oleate–iopamidol mixture or ethanol or with spring coils (Tornado; Cook, Bloomington, Ind) inserted through a 2.9-F microcatheter with selective catheterization. Ethanol was injected through a 5-F balloon catheter directly

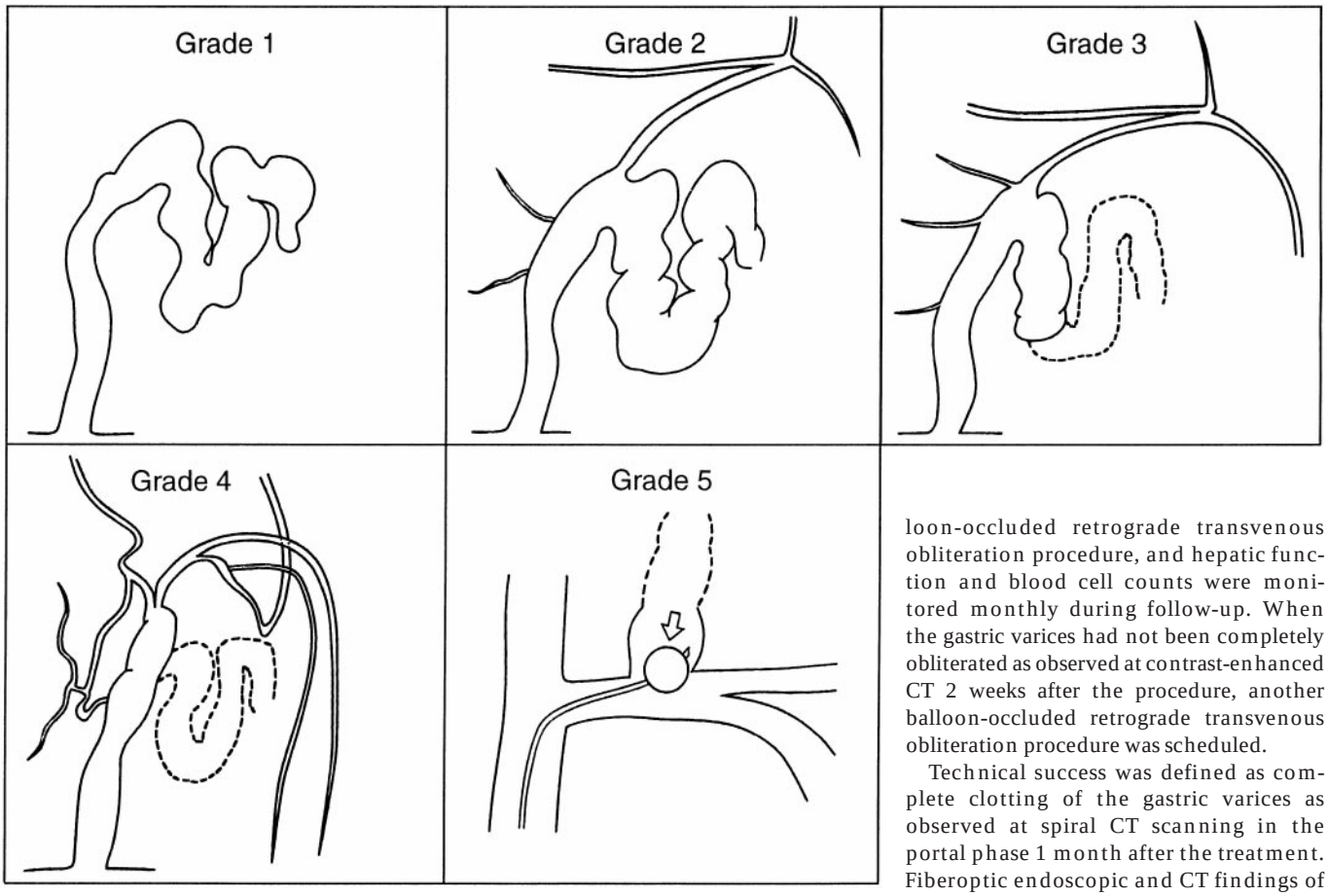


Figure 2. Diagram of the grades of progression of gastric varices and collateral veins: grade 1, gastric varices were well opacified without evidence of collateral veins; grade 2, collateral veins were small and few in number, and the contrast medium remained in the gastric varices for 3 minutes or more; grade 3, collateral veins were medium to large, there were few veins, and the contrast medium filled the gastric varices only partially and disappeared within 3 minutes; grade 4, there were many large collateral veins, and the gastric varices were not opacified; and grade 5, the left adrenal vein could not be occluded with the balloon catheter (arrow) because of a very large gastrorenal shunt with rapid blood flow.

into the left inferior phrenic vein in one patient. The contrast medium (iopamidol) was flushed during balloon occlusion to confirm that it filled the gastric varices and to measure the volume injected.

The sclerosing agent (ethanolamine oleate–iopamidol) was injected slowly with fluoroscopic monitoring until the gastric fundal varices had been completely filled. In one patient, 20 mL of 50% glucose was injected before the injection of the ethanolamine oleate–iopamidol mixture to reduce the dose of the mixture. The maximum dose of the ethanolamine oleate–iopamidol mixture used at one time was 30 mL, and the injected ethanolamine oleate–iopamidol mixture remained in the varices for 1–3 hours during balloon occlusion. After this, as much of the ethanolamine oleate–iopamidol mixture as possible was withdrawn under fluoroscopic observation.

The procedure was terminated when test injection of the contrast medium (iopamidol) showed that clots had formed in the varices. During the procedure, the blood pressure, pulse rate, electrocardiogram, arterial oxygen saturation (pulse oximeter), and volume of urine were monitored. Also, the serum NH_3 level was measured both before and after the procedure in the three patients with hepatic encephalopathy.

Follow-up

Fiberoptic endoscopy, follow-up CT, or endoscopic ultrasonography (US) (or some combination of these) was performed at 2 weeks, 1 month, 3 months, and then every 3 months after the balloon-occluded retrograde transvenous obliteration procedure, and hepatic function and blood cell counts were monitored monthly during follow-up. When the gastric varices had not been completely obliterated as observed at contrast-enhanced CT 2 weeks after the procedure, another balloon-occluded retrograde transvenous obliteration procedure was scheduled.

Technical success was defined as complete clotting of the gastric varices as observed at spiral CT scanning in the portal phase 1 month after the treatment. Fiberoptic endoscopic and CT findings of gastric varices, hepatic function, and symptoms were evaluated after the treatment. Techniques and complications of embolization of collateral veins are also reported.

RESULTS

Balloon-occluded retrograde transvenous obliteration was performed once on 14 patients, twice on five patients, and three times on one patient with large gastric varices and dilated collateral veins. With regard to collateral veins, inferior phrenic veins were found in 15 patients, and dilatation of the pericardiacophrenic vein was found in two patients.

The progression of the gastric varices and collateral veins in four patients was classified as grade 1, in five patients as grade 2, in six patients as grade 3, in four patients as grade 4, and in one patient as grade 5. To occlude the collateral veins, we used the ethanolamine oleate–iopamidol mixture for five patients with grade 2 progression and for two patients with grade 3 progression, we used ethanol for four patients with grade 3 progression and for two patients with grade 4 progres-

sion, and we used balloon occlusion and spring coils for one patient each with grade 4 progression. For the patient with grade 5 progression, partial splenic embolization was performed; 2 weeks later, blockage of the blood flow of the coronary vein with a transhepatically inserted balloon catheter was necessary to occlude a very large adrenal vein. There was no need to occlude the collateral veins in the four patients with grade 1 progression.

Obliteration of the collateral veins with ethanol and the ethanolamine oleate–iopamidol mixture during the initial procedure was unsuccessful in two patients. Therefore, coil embolization of the large collateral veins was needed for one patient, and for the other patient, ethanol embolization under balloon occlusion of the inferior phrenic vein was performed with direct insertion of a 5-F balloon catheter from the inferior vena cava.

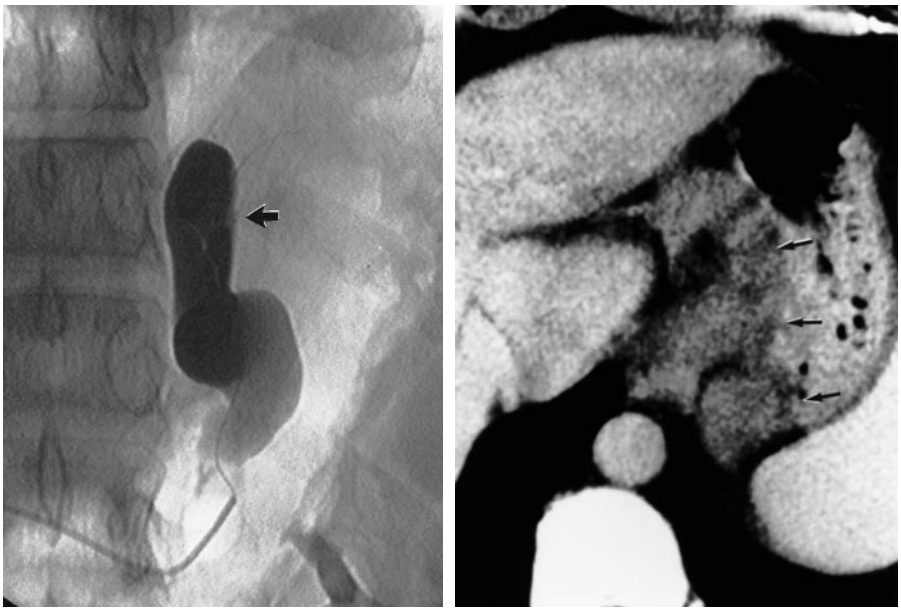
With regard to the relationship between the grades of collateral development and the mean number of balloon-occluded retrograde transvenous obliteration procedures, grade 1 was associated with 1.0 procedure, grade 2 with 1.2 procedures, grade 3 with 1.6 procedures, and grade 4 with 2.0 procedures. Patients with grade 5 progression required other procedures such as partial splenic embolization and percutaneous transhepatic obstruction of coronary veins by balloon occlusion.

During the procedure, 18 of the patients complained of slight pain and hot sensations in the epigastric region, but these symptoms had almost disappeared by the end of the procedure. No hepatic toxic effects were observed after the procedure.

Complications occurred during the procedure in three patients. One patient, who had shown no allergy to the iodinated contrast medium, developed cardiogenic shock resulting from an anaphylactic reaction to ethanolamine oleate. Emergency treatment was needed, including cardiac massage and administration of catecholamine drugs.

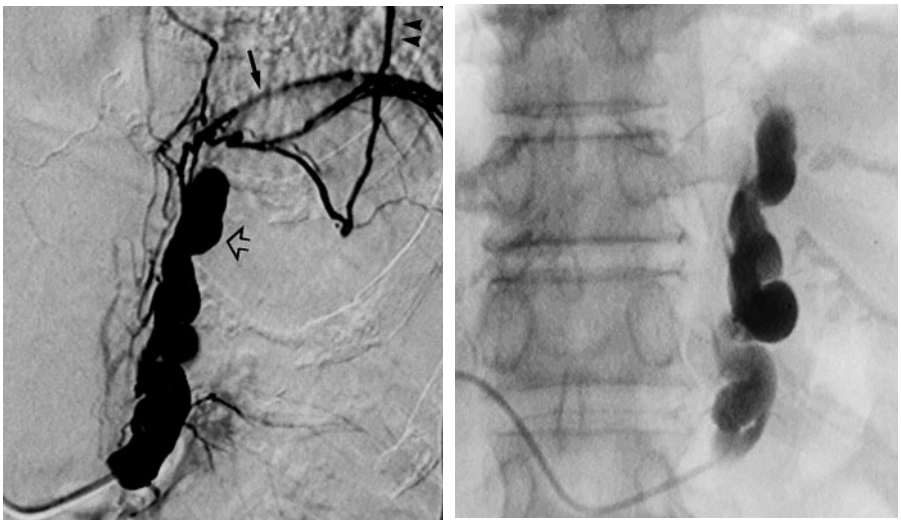
In another patient, a spring coil 3 mm in diameter, which was used for embolization of the inferior phrenic vein, migrated to the proximal portion of the adrenal vein because of a change in the blood flow direction and stopped at the balloon. A Bird's Nest inferior vena cava filter (Cook, Bloomington, Ind) was placed at the suprarenal position, which resulted in successful entrapment of the coil with the filter.

In the third patient, the balloon part of the catheter slipped down to the renal vein, which resulted in partial obstruction of the renal vein. Macrohematuria caused by the increase in renal vein pressure was observed during the proce-



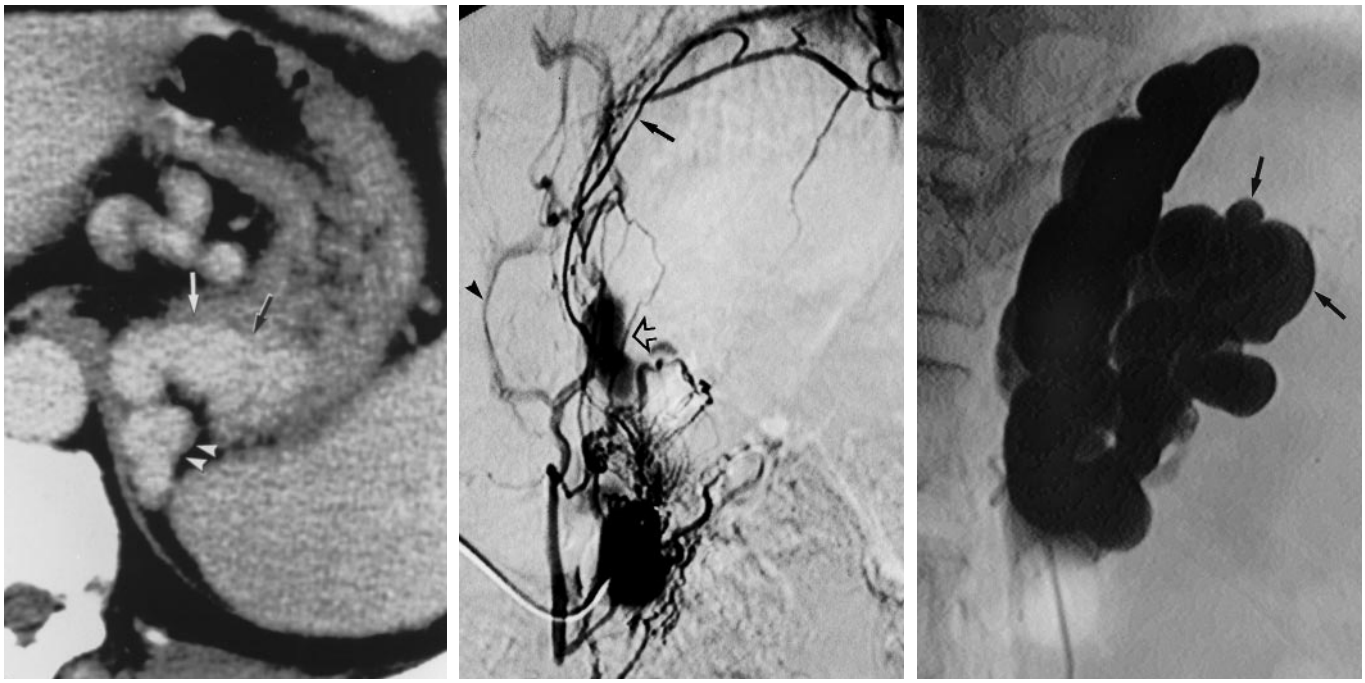
a. **b.**

Figure 3. Grade 1 gastric varices and collateral veins in a 48-year-old man with a history of two episodes of esophageal variceal bleeding. **(a)** Left adrenal venogram shows large gastric fundal varices. Because no collateral veins were seen on the balloon-occluded left adrenal venogram, balloon-occluded retrograde transvenous obliteration was performed with 18 mL of the ethanolamine oleate–iopamidol mixture. The ethanolamine oleate–iopamidol mixture filled the gastric varices (arrow). **(b)** CT scan obtained 1 month after balloon-occluded retrograde transvenous obliteration shows gastric varices (arrows) with very low attenuation, suggesting complete coagulation.



a. **b.**

Figure 4. Grade 2 gastric varices and collateral veins in a 69-year-old woman with a history of bleeding from esophageal varices. **(a)** Angiogram obtained during balloon occlusion of the proximal part of the left adrenal vein shows dilatation of the inferior phrenic (solid arrow) and pericardiacophrenic (arrowheads) veins. Because the gastric varices (open arrow) could be visualized in their entirety, however, we judged this case to be one of grade 2 collateral veins. **(b)** Angiogram obtained during the procedure. Injection of 5 mL of ethanol caused occlusion of the collateral veins, after which balloon-occluded retrograde transvenous obliteration with 17 mL of the ethanolamine oleate–iopamidol mixture was performed. Gastric varices are filled with the ethanolamine oleate–iopamidol mixture.



a. **b.** **c.**

Figure 5. Grade 3 gastric varices and collateral veins in a 62-year-old man who had huge gastric fundal varices, 8 cm in diameter according to endoscopic measurement. **(a)** CT scan shows marked dilatation of gastric varices (arrows), which are connected with the dilated gastrorenal shunt (arrowheads). **(b)** Left adrenal angiogram obtained during balloon occlusion shows the inferior phrenic vein (solid arrow) and dilated paravertebral veins (arrowhead), but gastric varices (open arrow) are only partially visible. Injection of 30 mL of the ethanolamine oleate–iopamidol mixture into the veins terminated the first balloon-occluded retrograde transvenous obliteration procedure. **(c)** Angiogram from second angiographic study performed 2 weeks after the first shows complete depiction of the gastric varices (arrows) and occlusion of the collateral veins; 30 mL of the ethanolamine oleate–iopamidol mixture was injected with balloon occlusion. One month after the second balloon-occluded retrograde transvenous obliteration procedure, the gastric varices were completely obliterated.

dure, but no damage to the kidney was found.

Follow-up contrast-enhanced CT at 1 month after the procedure showed gastric varices as areas of nonenhancement, meaning clot formation, in all cases. The endoscopic appearance of the varices 2 weeks after balloon-occluded retrograde transvenous obliteration did not show any change, but endoscopic US, which was performed before and 2 weeks after the treatment in seven patients, showed filling with an echogenic mass and stagnation of the blood flow in the gastric varices, compared with anechoic findings before the treatment. This change indicated clot formation in the gastric varices after the treatment. After 3 months, the varices of 15 patients had completely disappeared, and endoscopic examination showed that the varices of five patients had decreased remarkably in size.

Follow-up results (follow-up period, 3–36 months; mean, 16.6 months) showed that 19 patients had survived without recurrence or bleeding from the gastric varices, with the longest survival being 3 years after the procedure. One patient could not be followed up, and three of the 19 patients showed aggravation of the esophageal varices and were treated with endoscopic injection sclerotherapy, after which they were followed up every 3 months.

The clinical symptoms of the three patients with encephalopathy improved remarkably. The serum NH_3 levels of the three patients, measured both before and after the procedure, changed from 124 to 71 mg/100 mL, from 260 to 78 mg/100 mL, and from 132 to 45 mg/100 mL. Newly detected hepatocellular carcinoma during the follow-up period had to be treated with arterial embolization in two patients.

DISCUSSION

The prevalence of gastric varices in patients with portal hypertension is approximately 30% (17–19), lower than that of esophageal varices. However, the more often esophageal varices are controlled with endoscopic injection sclerotherapy, the more often gastric varices develop because of portal pressure exerted on the perigastric vein (12).

Gastric varices, especially those classified as Lg-cf and Lg-f, develop because of dilatation of the short and posterior gastric veins or because of the generation of direct anastomotic veins between the gastric and retroperitoneal veins; gastric varices classified as Lg-c feature a direct continuity with the coronary and esophageal veins. Watanabe et al (17) reported that 100% (eight varices) of eight Lg-f varices, 80% (12 varices) of 15 Lg-cf varices, and 17% (17 varices) of 99 esophageal varices in their study featured a gastrorenal shunt. Previous studies showed hemodynamic differences between esophageal and gastric varices.

The bleeding rate for gastric varices has been reported as 78% (seven of nine varices) for Lg-f, 63% (10 of 16 varices) for Lg-cf, and 43% (32 of 74 varices) for Lg-c by Obara and Kasukawa (18) and as 78% (seven of nine varices) for Lg-cf and 12% (10 of 85 varices) for Lg-c by Sarin et al (19). In addition, the mortality rate for bleeding gastric varices is much higher than that for bleeding esophageal varices (6,7).

Treating gastric fundal varices (Lg-f and Lg-cf) with endoscopic injection sclerotherapy is very difficult because the high blood flow volume through gastrorenal or splenorenal shunts results in a rapid loss of the sclerosing agent into the systemic circulation during endoscopic injection sclerotherapy. Furthermore, gastric varices are too large to ligate endoscopically.

However, balloon-occluded retrograde transvenous obliteration effectively causes the gastric fundal varices to coagulate because of stoppage of the voluminous blood flow in the varices by retrograde balloon occlusion. The results of our study show a very high initial success rate (100%) of obliteration of gastric varices, while portosystemic encephalopathy was well controlled in three patients after balloon-occluded retrograde transvenous obliteration. Moreover, the midterm results showed that the effect persisted for 1 year or more without aggravation.

Collateral veins such as the inferior phrenic, hemiazygos, or pericardial veins develop in accordance with the blood flow from the gastric varices. Occlusion of these collateral veins is essential for success with obliteration of the gastric varices. However, Kanagawa et al (12) did not mention the need for occlusion of the collateral veins in 32 patients treated with balloon-occluded retrograde transvenous obliteration, but 16 of our 20 patients (80%) had developed small to large collateral veins.

To determine the appropriate technique for balloon-occluded retrograde transvenous obliteration, we needed to determine the blood flow and the volume of the gastric varices and collateral veins. Hence, we classified the degree of progression of the gastric varices and collateral veins into five grades according to the results of adrenal venography during balloon occlusion. In patients with grade 1 progression, we could easily occlude the gastric varices because of the absence of collateral veins. In patients with grade 2 progression, injection of less than 10 mL of the ethanolamine oleate–iopamidol mixture into the collateral veins resulted in occlusion 15–30 minutes after injection. The gastric varices can then be completely opacified and obliterated.

In patients with grade 3 progression, we often needed to use ethanol to occlude the collateral veins, and for patients with grade 4 progression, injection of ethanol and of about 20 mL of the ethanolamine oleate–iopamidol mixture into the collateral veins was necessary. However, when obliteration of the collateral veins with ethanol and the ethanolamine oleate–iopamidol mixture was unsuccessful during the initial procedure, coil embolization or balloon occlusion of the inferior phrenic vein with a directly inserted catheter was helpful.

Even when the collateral veins had been occluded, however, the very large gastric varices of six patients could not be obliterated, even with injection of 20 mL of the ethanolamine oleate–iopamidol mixture; in these patients, a second balloon-occluded retrograde transvenous obliteration procedure was necessary 1 or 2 weeks after the initial one. The gastric varices of five of these patients were obliterated with the second procedure because the gastric varices had shrunk, but for one patient with extremely large varices, three balloon-occluded retrograde transvenous obliteration procedures were needed.

For the patient with grade 5 progression, which features voluminous blood flow into the varices, partial splenic embolization and occlusion of the coronary vein with a transhepatically inserted catheter were necessary to reduce the blood flow into the varices sufficiently for occlusion of the gastrorenal shunt with the balloon catheter. The grading system of the gastric varices and collateral veins is therefore thought to be very useful for selecting the optimum technique for balloon-occluded retrograde transvenous obliteration.

We decided that the maximum injection of the ethanolamine oleate–iopamidol mixture was 30 mL at one time. Although the maximum dose of the ethanolamine oleate–iopamidol mixture is 20 mL during endoscopic injection sclerotherapy, we were able to inject 30 mL because residual ethanolamine oleate–iopamidol mixture could be retrieved during the final step of the procedure.

Ethanolamine oleate, an anionic surfactant (20), infiltrates and destroys the cell membrane of endothelial cells. When the gastric varices are very large, however, ethanolamine oleate is diverted to the destruction of red blood cell membranes, thus causing insufficient destruction of the endothelium of the varices (20–24). For this reason, in one patient, we injected 20 mL of a 50% glucose solution into the gastric varices to replace the red blood cells (25) before injection of the ethanolamine oleate–iopamidol mixture. The result was a successful injection that filled the gastric varices, with the ethanolamine oleate–iopamidol mixture attaching to the endothelial but not the red blood cells. In addition, the 50% glucose solution had a high osmolarity that injured the endothelial cells, so that this proved to be an easy and effective supplementary technique.

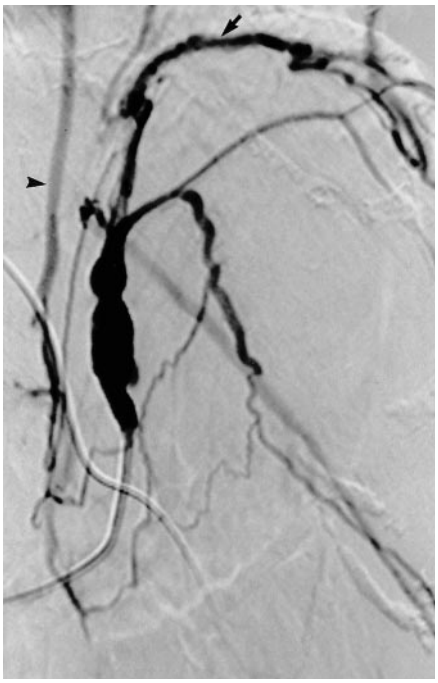


Figure 6. Grade 4 gastric varices and collateral veins in a 64-year-old woman who had a history of bleeding from esophageal varices. Left adrenal angiogram obtained during balloon occlusion shows dilatation of the inferior phrenic (arrow) and ascending lumbar (arrowhead) veins, but the gastric varices are not visible, which led to a classification of grade 4.

Among sclerosing agents, ethanol is an agent with a strongly destructive effect on endothelial cells, and ethanol was used by us for embolization of collateral veins. However, severe acute alcohol intoxication causes coma (blood alcohol concentration, 300–400 mg/100 mL) and stoppage of respiration (blood alcohol concentration, >400 mg/100 mL). Short-term injection of about 15 g (19.2 mL; 0.23 g/kg) of ethanol into the systemic vein may also induce coma, so one should be very cautious about injecting ethanol into the venous system and should determine carefully the dose injected.

Furthermore, 85% of Japanese have an atypical alcohol dehydrogenase and an unusual acetaldehyde dehydrogenase, which promptly metabolize alcohol to acetaldehyde but take much more time to metabolize acetaldehyde to acetate (26). The resultant high blood level of acetaldehyde causes sympathetic symptoms and high sensitivity to alcohol. This phenomenon causes us to use ethanol as little as possible. Furthermore, we suspect that

dilution of ethanol with contrast medium weakens its effect. These are the reasons we used the ethanolamine oleate–iopamidol mixture to obliterate gastric varices, but the combined use of ethanol and the ethanolamine oleate–iopamidol mixture warrants further study.

Surgical procedures or transjugular intrahepatic portosystemic shunts are also used to treat gastric varices. Surgery, especially the Hassab (27) operation, which consists of devascularization of the upper half of the stomach and esophagus accompanied by splenectomy, can eliminate gastric varices, but the operation has some risks, and patients must be in good condition and able to tolerate surgery and anesthesia.

Gastric varices in the fundus frequently failed to disappear with treatment with a transjugular intrahepatic portosystemic shunt; the reported success rate is only 50% (8). Sanyal (8) observed that these varices were often associated with spontaneous splenorenal collateral vessels that fed the gastric varices or were associated with massive splenomegaly, and blood continued to flow through the short gastric vessels despite a decrease in the portosystemic gradient to less than 12 mm Hg.

With regard to the relationship between the shunt volume and the hepatic encephalopathy after a transjugular intrahepatic portosystemic shunt procedure, Haskal and Ring (28) reported their experience with implanting second stents in 10 patients in whom the first stent had only partially corrected the portal hypertension. In two of these patients, portosystemic encephalopathy appeared for the first time promptly after the insertion of the second shunt. Thus, the increment in portal-systemic shunting caused by the addition of a second shunt, 8 mm in diameter, to a preexisting 10-mm shunt, a procedure that increases the blood flow by about 40%, eliminated the portal hypertension and greatly increased the amount of comagenic substances shunted into the systemic circulation.

Furthermore, Kerlan et al (9) reported that a transjugular intrahepatic portosystemic shunt may induce or aggravate hepatic encephalopathy, as with surgical shunts. Brown and Lake (10) and Somberg (11) added that severe hepatic encephalopathy is a relative contraindication to transjugular intrahepatic portosystemic shunt. We therefore believed that occlusion of the gastrorenal shunt was preferable to new shunt formation with a transjugular intrahepatic portosystemic shunt in selected cases of severe portosystemic encephalopathy caused by a gastrorenal shunt.

The balloon-occluded retrograde transvenous obliteration procedure, on the other hand, is less invasive and provides blockage of the gastric variceal blood flow with a very high rate of success. In fact, because this technique is safe and effective, its prophylactic use may well be warranted. We therefore recommend this treatment for patients who have gastric varices in danger of rupture and in association with a gastrorenal shunt or hepatic encephalopathy (or both).

One of our patients developed cardiogenic shock immediately after injection of 10 mL of the ethanolamine oleate–iopamidol mixture. Once an anaphylactic reaction to ethanolamine oleate occurs, it can become a major complication, so the injection of the ethanolamine oleate–iopamidol mixture should be done very carefully. First, a small amount of the ethanolamine oleate–iopamidol mixture should be injected slowly, and the patient should be observed for a while to confirm that there is no allergic reaction. Although there have been some reports of complications caused by ethanolamine oleate—namely, pulmonary edema (29,30), hemothorax (31), and disseminated intravascular coagulation (32)—to our knowledge, cardiogenic shock has not been reported as a complication of intravenous administration of the ethanolamine oleate–iopamidol mixture.

One of the technical complications we encountered was migration of the embolic coil that was released at the inferior phrenic vein. This migration was caused by a reversal of the direction of the blood flow, which resulted from the obliteration of the collateral veins. Careful attention should thus be paid to the effects of such a reversal. The use of greatly oversized coils is important to prevent retrograde movement. The other complication consisted of the balloon catheter slipping down into the left renal vein during balloon-occluded retrograde transvenous obliteration, indicating that the catheter position should be frequently checked fluoroscopically during the procedure.

A gastrorenal shunt functions to decrease portal venous pressure. Obliteration of the gastrorenal shunt may, therefore, cause portal venous pressure to increase and new collateral veins to the esophageal varices to develop. In fact, three of our patients experienced exacerbation of their esophageal varices after balloon-occluded retrograde transvenous obliteration. However, these esophageal varices could be largely controlled with endoscopic injection sclerotherapy.

In conclusion, balloon-occluded retrograde transvenous obliteration was found to be a feasible alternative to a transjugular intrahepatic portosystemic shunt procedure in patients with a large gastrorenal shunt or hepatic encephalopathy (or both).

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