J Gastroenterol 2000; 35:290–295

Journal of Gastroenterology

Case report

Gastric varices with splenic vein occlusion treated by splenic arterial embolization

Takahiro Sato, Katsu Yamazaki, Jouji Toyota, Yoshiyasu Karino, Takumi Ohmura, and Toshihiro Suga

Department of Gastroenterology, Sapporo Kosei General Hospital, Kita 3 Higashi 8, Chuo-ku, Sapporo 060-0033, Japan

Abstract: A 53-year-old man was admitted to our hospital in August 1997 with enlarged gastric varices. Computed tomography (CT) showed splenic vein occlusion, gastric varices, and extra-gastric wall collateral veins. Color flow images of gastric varices were clearly visualized, and the velocity in the gastric varices was 19.6 cm/s via endoscopic color Doppler ultrasonography (ECDUS). The patient was diagnosed with gastric varices according to angiographic findings of splenic vein occlusion, and splenic arterial embolization was performed. Two weeks after the splenic arterial embolization, CT showed peripheral areas of low attenuation in the spleen, due to splenic infarction, with 70% of the spleen volume showing low attenuation. Eight months after the splenic arterial embolization, ECDUS revealed a decrease in gastric variceal color flow images, with the velocity in the gastric varices being 10.3 cm/s.

Key words: gastric varices, splenic vein occlusion, splenic arterial embolization

Introduction

Gastric variceal hemorrhaging is a common complication of portal hypertension. Recent technical advances have offered clinicians increasingly greater clarity in visualizing gastric varices. Hemorrhaging from gastric varices is associated with higher morbidity and mortality rates than hemorrhage from esophageal varices.[1] Hemodynamic studies of gastric varices are employed worldwide.[2,3] Splenic vein occlusion, however, is commonly silent clinically. This condition may cause hypersplenism or gastrointestinal hemorrhaging due to gastric varices.[4–7]

Received: April 26, 1999 / Accepted: August 27, 1999
Reprint requests to: T. Sato

We describe a patient with gastric varices caused by splenic vein occlusion associated with chronic pancreatitis, who was successfully treated by splenic arterial embolization.

Case report

A 53-year-old asymptomatic man was admitted to our hospital in August 1997 with enlarged gastric varices. He had no particular family history of illness. At age 43 years, he had been diagnosed with acute alcoholic pancreatitis, and he had been followed-up subsequently for chronic pancreatitis. At age 50 years, fibergastroscopic examination had revealed a giant fold from the fornix to the curvatura ventriculi major of the gastric body.

At the time of admission to our hospital, he was 170 cm tall and weighed 75.6 kg. His blood pressure was 140/80 mmHg, pulse, 80/min and regular; and body temperature, 36.0°C. Neither jaundice nor anemia was observed. The abdomen was soft and flat, and there were no abdominal masses or hepatomegaly. Splenomegaly was recognized. Peripheral blood examinations revealed a decreased platelet level. Results of biochemical examinations were all within normal limits, except for an elevated triglyceride level (Table 1).

Fiberscopic examination on admission revealed gastric varices (assessed according to the new classification system proposed by the Japanese Research Society for Portal Hypertension[8]) located from the fornix (classified as Lg-f) to the curvatura ventriculi major of the gastric body (Fig. 1). According to this system, varices are grouped as largest size (F_3), blue varices (C_B), and red color-sign-negative varices (RC[-]). Esophageal varices were not detected on the endoscopic examination.

The splenic vein was not detected by computed tomography (CT) (Fig. 2a), while gastric varices and

Table 1. Laboratory data on admission

Peripheral blood count	
WBC	5100
RBC	446×10^4
Hb	14.2 g/dl
Ht	41.1%
Plt	11.3×10^4
Blood chemistry	
TP	7.2 g/dl
Alb	4.6 g/dl
T.bil	0.7 mg/dl
GOT	19 IU/I
GPT	16 IU/I
ALP	132 IU/I
LDH	353 IU/I
ZTT	7.0 KU
ChE	187 IU/I
BUN	17.4 mg/dl
Cr	0.9 mg/dl
FBS	105 mg/dl
Tcho	189 mg/dl
TG	231 mg/dl
Serological tests	
HB_sAg	(−)
HB_sAb	(−)
HCVAb	(−)
CRP	0.1 mg/dl
Tumor markers	
AFP	3.4 ng/ml
CEA	1.1 ng/ml
Urinalysis	
Protein	(−)
Sugar	(−)
Bilirubin	(−)

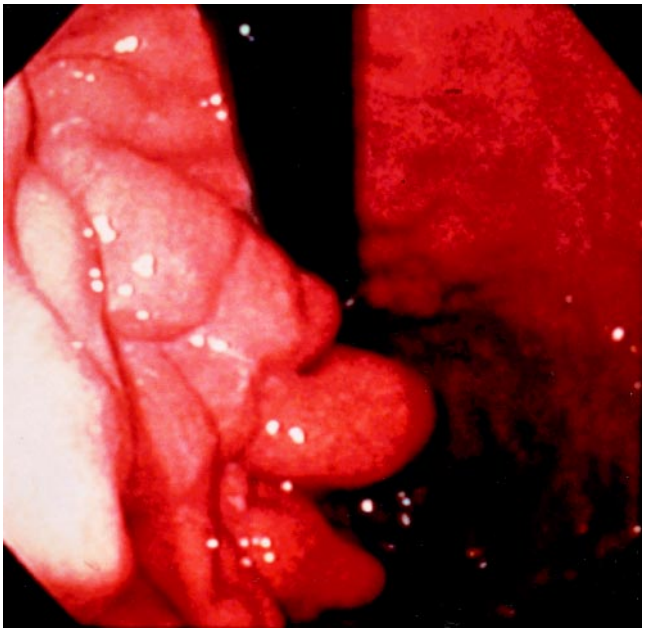

Fig. 1. Endoscopic appearance consistent with F_3 type varices (according to the classification system of the Japanese Research Society for Portal Hypertension[8]) located from the fornix to the curvatura ventriculi major of the gastric body

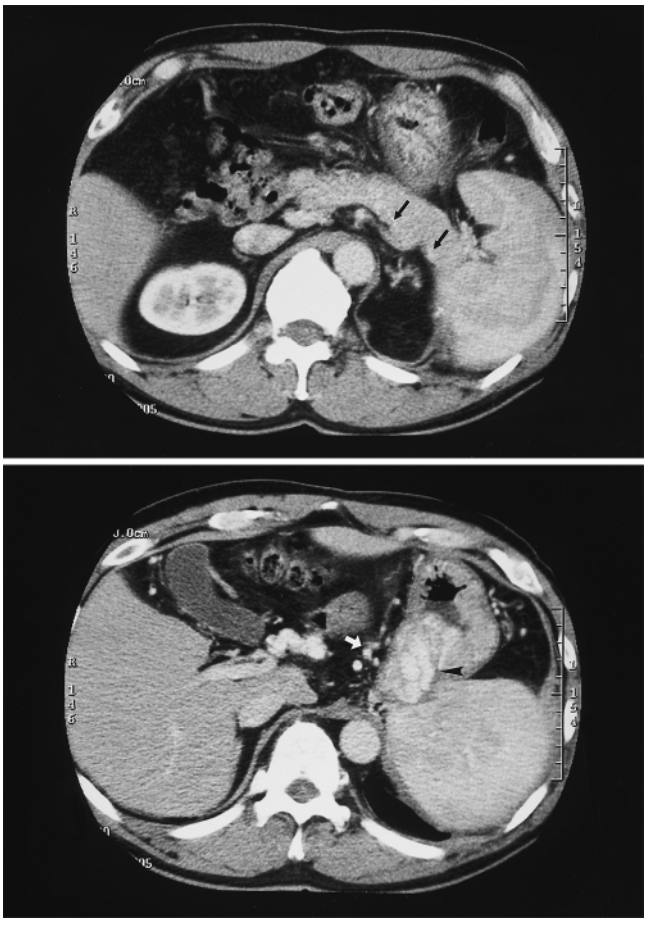

Fig. 2. **a** Splenic vein occlusion was visualized on computed tomography (CT) scan (*arrows*). **b** Gastric varices (*arrowhead*) and other collateral veins (*arrow*) were demonstrated by CT

extra-gastric wall collateral veins were revealed (Fig. 2b). On CT, no pancreatic mass was seen, and neither hepatomegaly nor lymphadenopathy was evident. Endoscopic retrograde pancreatography (ERP) indicated pancreatic divisum and irregularity of the dorsal pancreatic duct (Fig. 3). The ERP findings were found to be consistent with chronic pancreatitis. Color flow images of gastric varices were clearly visualized via endoscopic color Doppler ultrasonography (ECDUS) (Fig. 4). The blood flow in the intramural gastric varices showed a continuous wave under fast-Fourier transform (FFT) analysis, and the velocity in the gastric varices was 19.6 cm/s. The patient was diagnosed with gastric varices caused by splenic vein occlusion associated with chronic pancreatitis.

Next, angiography was performed, by the Seldinger technique for femoral artery catheterization, and a splenic arteriogram was obtained with venous phase follow-up, which demonstrated completely occluded splenic vein in the hilus of the spleen. Gastric varices

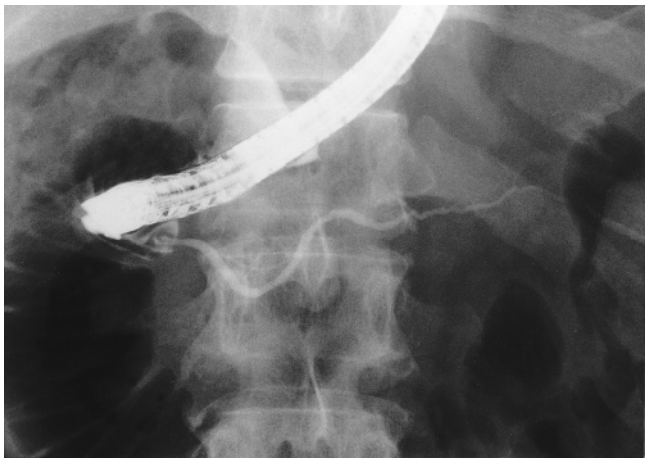

Fig. 3. Irregularity of dorsal pancreatic duct was visualized on endoscopic retrograde pancreatography (ERP)

and left gastric vein were demonstrated by angiography (Fig. 5). The short gastric vein and gastroepiploic vein were both dilated, and were draining into the proximal splenic vein, while the portal vein was patent. The patient was diagnosed as having a high risk of rupture of the gastric varices. Splenectomy was not performed in light of the difficulty of operating because of the adhesion of the spleen and pancreas to the diaphragm. Splenic arterial embolization was therefore performed, on September 30, 1997. A guide-wire was directed into the splenic artery, and a wedge balloon catheter was passed over the guide wire. Five milliliters of contrast agent mixed with aminocaproic acid and absorbable gelatin sponge particles was then injected into the splenic artery. Additional splenic arterial embolization was performed on October 20, and again on November 11, 1997. After splenic arterial embolization, the patient had a slight fever and abdominal pain, and showed mild elevation of leukocyte levels on laboratory findings.

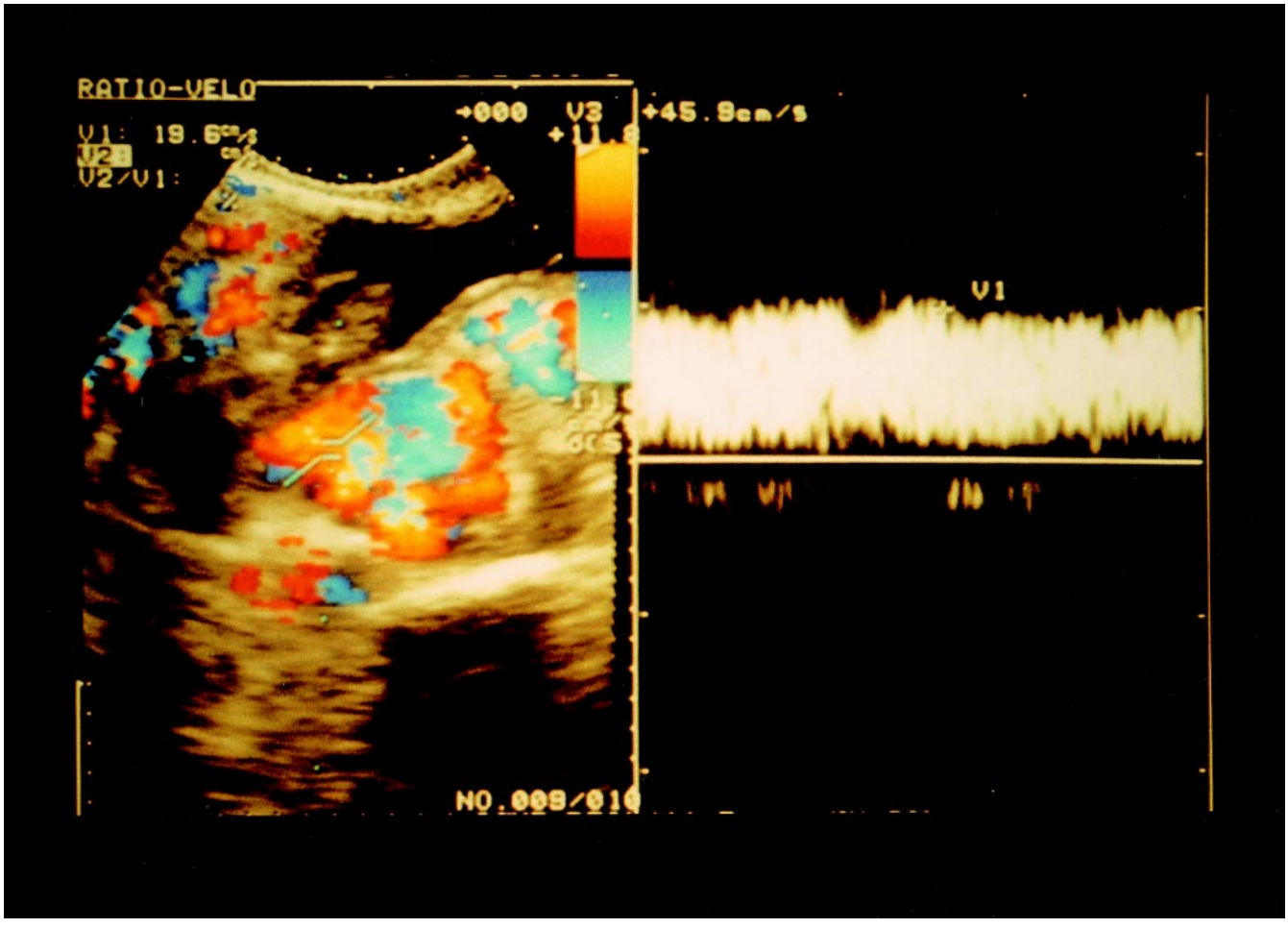

Fig. 4. Color flow images of gastric varices were visualized by endoscopic color Doppler ultrasonography, (ECDUS), and fast-Fourier transform analysis indicated a continuous wave

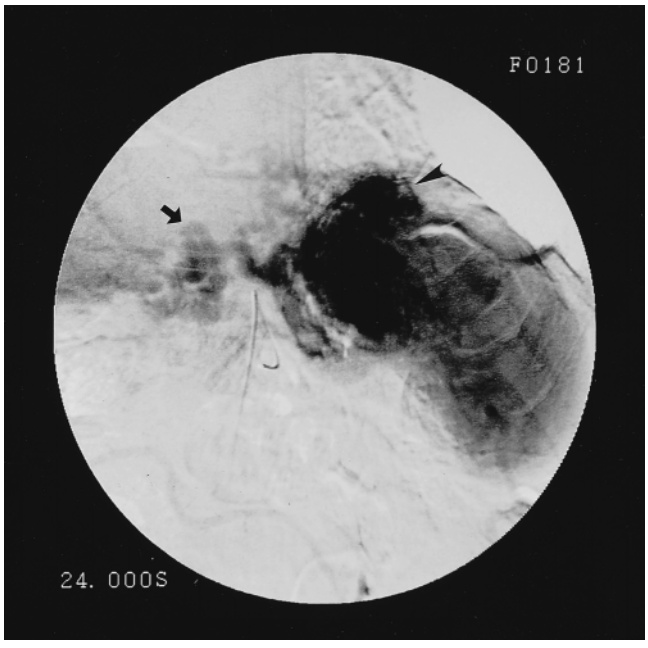

Fig. 5. Completely occluded splenic vein was demonstrated in the hilus of the spleen, and gastric varices (*arrowhead*) and left gastric vein (*arrow*) were demonstrated by angiography

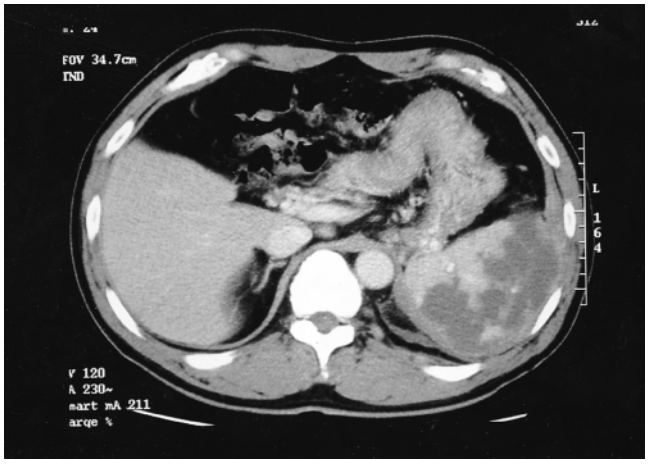

Fig. 6. CT scan 2 weeks after splenic arterial embolization; 70% of the spleen volume showed low attenuation, indicating infarction

Two weeks after the final splenic arterial embolization, a CT scan of the abdomen showed peripheral areas of low attenuation in the spleen, thought to caused by splenic infarction; 70% of the spleen showed low attenuation (Fig. 6). Eight months after the final splenic arterial embolization, ECDUS findings revealed a decrease in gastric variceal color flow images, with the velocity in gastric varices 10.3 cm/s (Fig. 7).

Discussion

Splenic vein occlusion is characterized by gastric varices, splenomegaly, and normal liver function, and results in left-sided portal hypertension,[9–11] which is secondary to pancreatic inflammation or neoplasm.[12,13] The tail of the pancreas is enveloped with splenic vessels and the splenorenal ligament at the hilus of the spleen, and this anatomical proximity results in intimal damage of the splenic vein. Splenic vein occlusion creates an outflow block and prevents the circulation of blood from the spleen. Splenic blood, which is unable to drain through the occluded splenic vein, must then flow into collaterals. Collaterals (the short gastric vein and left gastroepiploic veins) occur from the splenic hilus through the gastric wall, and increased flow in the stomach wall dilates the submucosal veins, producing gastric varices. Because blood drainage is diverted by the coronary vein into the patent portal system, the presence of gastric varices without esophageal varices is a very specific sign of splenic vein occlusion.

Although splenic vein occlusion is commonly silent clinically, this condition may cause hypersplenism or gastrointestinal hemorrhage due to gastric varices. Sutton et al.[9] found 53 cases of such occlusion, and reported a 64% incidence of upper gastrointestinal bleeding. Itzchak and Glickman[14] noted gastrointestinal hemorrhaging in only 3 of 19 patients with splenic vein occlusion. Sarin et al.[15] studied the prevalence of gastric varices in 568 patients with portal hypertension. They reported that 7 of 9 patients (78%) with gastric varices due to splenic vein occlusion had a history of previous variceal bleeding.

Patients with bleeding gastric varices secondary to splenic vein occlusion can be successfully treated by splenectomy.[9,11,16] The logic behind splenectomy is that it decompresses the short gastric veins by cutting off inflow. However, it is often difficult to determine whether surgical therapy should be undertaken in patients with advanced disease such as pancreatic carcinoma. Endoscopic injection sclerotherapy (EIS) is more difficult to perform for gastric varices than for esophageal varices, and EIS has had varying success rates.[17–19] EIS, using Histoacryl, is useful in the treatment of bleeding gastric varices. However, the sustained increased pressure on the stomach wall may cause the formation of new collateral veins after EIS. As patients with splenic vein occlusion have normal portal pressure and normal hepatic function, portal systemic shunting is not indicated. Splenic arterial embolization has been used in the treatment of hypersplenism. An alternative treatment, in patients with gastric varices due to splenic vein occlusion, is splenic arterial embolization, which reduces blood flow through the splenic parenchyma. Splenic arterial embolization

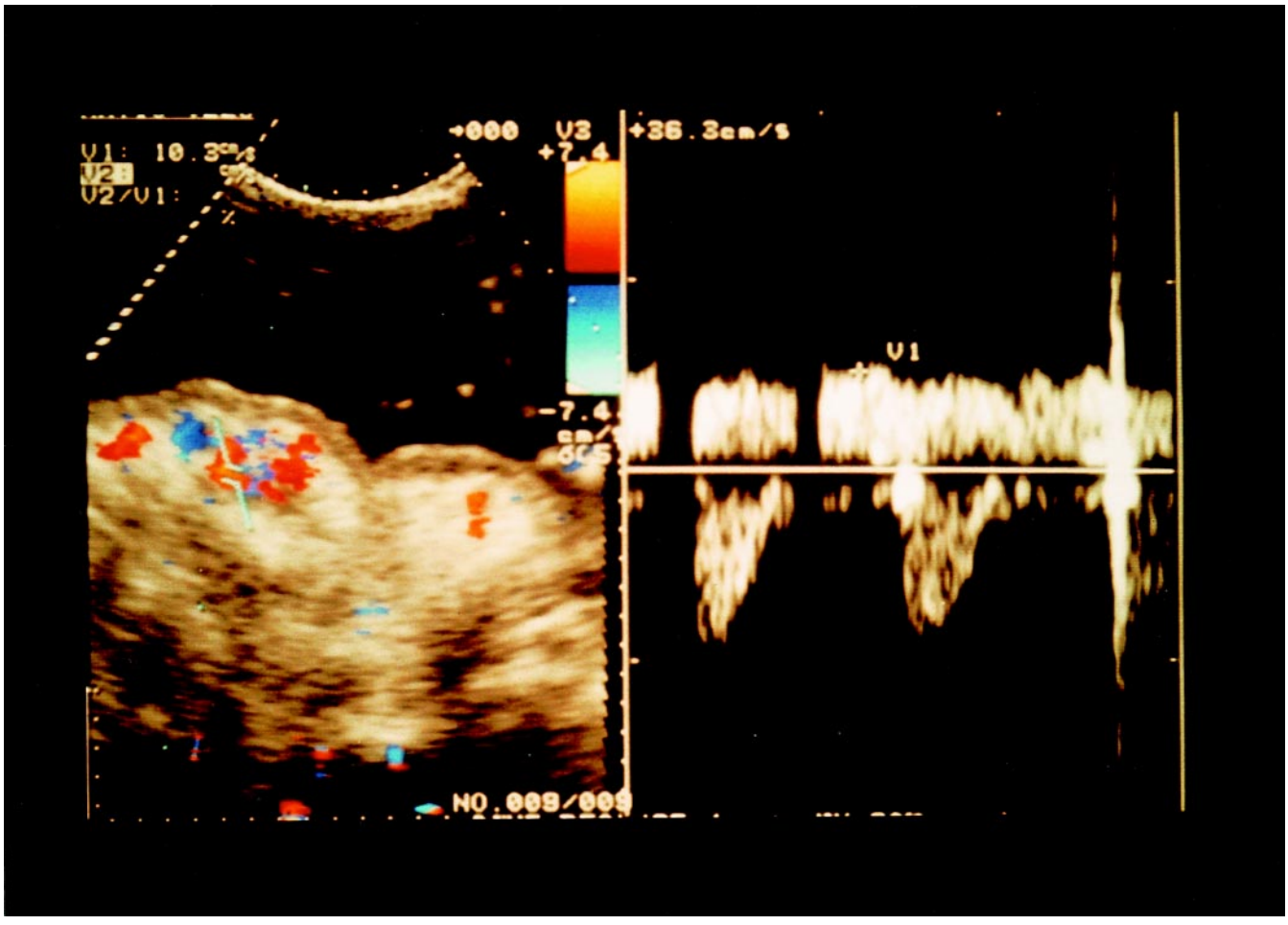

Fig. 7. Eight months after the splenic arterial embolization, ECDUS revealed a decrease in gastric variceal color flow images

with Bucrylate (isobutyl 2-cyanoacrylate) was done successfull for bleeding gastric varices secondary to splenic vein occlusion.[20] Mcdermott et al.[21] reported a patient with bleeding gastric varices secondary to splenic vein occlusion who was successfully treated by splenic arterial embolization, using Gianturco coils. Adams et al.[22] have described the usefulness of preoperative control of splenic artery inflow in patients with splenic vein occlusion. Embolization using particulate matter, such as gelatin sponge, in the splenic artery (end-artery) produces extensive splenic infarction and abscess. Splenic arterial embolization was performed as the definitive therapy for our patient because of the high risk involved with operation, and no episodes of side effects have occurred. Immediately after splenic arterial embolization, color Doppler flow indicated a decrease in the blood flow in the short gastric vein. Eight months after splenic arterial embolization, endoscopic findings revealed a decrease in the form of the gastric varices, and ECDUS indicated a decrease in the blood flow in the gastric varices. In our patient, splenic arterial embolization was an effective treatment for gastric varices caused by splenic vein occlusion.

In conclusion, splenic arterial embolization is an attractive alternative treatment for gastric varices associated with splenic vein occlusion in patients at high surgical risk.

References

1. Trudeau W, Prindiville T. Endoscopic injection sclerosis in bleeding gastric varices. Gastrointest Endosc 1986;32:264–8.
2. Sato T, Higashino K, Murashima Y, Suga T, Yaosaka T, Imamura A, et al. An application of endoscopic color Doppler ultrasonography (ECDUS) in the diagnosis of hemodynamics of gastric varices, and the therapeutic effect of endoscopic therapy. Dig Endosc 1994;6:326–33.
3. Sato T, Yamazaki K, Toyota J, Karino Y, Omura T, Suga T. Use of the ultrasonic microprobe to evaluate gastric varices—comparison with endoscopic color Doppler ultrasonography. Dig Endosc 1997;9:189–93.
4. Marks LJ, Weingarten B, Gerst GR. Carcinoma of the tail of the pancreas associated with bleeding gastric varices and hypersplenism. Ann Intern Med 1952;37:1077–84.

5. Goldstein GB. Splenic vein thrombosis causing gastric varices and bleeding. Am J Gastroenterol 1972;58:319–25.
6. Moosa AR, Gadd MA. Isolated splenic vein thrombosis. World J Surg 1985;9:384–90.
7. Madsen M, Petersen TH, Sommer H. Segmental portal hypertension. Ann Surg 1986;204:72–7.
8. Japanese Research Society for Portal Hypertension. The general rules for recording endoscopic findings of esophago-gastric varices—revised edition (in Japanese). Kanzou (Acta Hepatol Jpn) 1991;33:277–81.
9. Sutton JP, Yarborough DY, Richards JT. Isolated splenic vein occlusion. Arch Surg 1970;100:623–6.
10. Babb RR. Splenic vein obstruction: a curable cause of variceal bleeding. Dig Dis 1976;21:512–13.
11. Muhletaler C, Gerlock AJ, Goncharenko V, Avant GR, Flexner JM. Gastric varices secondary to splenic vein occlusion: radiographic diagnosis and clinical significance. Dig Radiol 1979;132: 593–8.
12. Salam AA, Warren WD, Tyras DH. Splenic vein thrombosis: a diagnosable and curable form of portal hypertension. Surgery 1973;74:961–72.
13. Cho KJ, Reuter SR. Angiographic assessment of pancreatic pseudocyst: a reappraisal. J Can Assoc Radiol 1976;27:193–5.
14. Itzchak W, Glickman M. Splenic vein thrombosis in patients with a normal size spleen. Invest Radiol 1977;12:158–63.
15. Sarin SK, Lahoti D, Saxena SP, Murthy NS, Makwana UK. Prevalence, classification and natural history of gastric varices: a long-term follow-up study in 568 portal hypertension patients. Hepatology 1992;16:1343–9.
16. Greenwald HM, Wasch MG. The roentgenologic demonstration of esophageal varices as a diagnostic aid in chronic obstruction of the splenic vein. J Pediatr 1939;14:57–65.
17. Sato T, Aso K, Higashino K, Kato S, Toyota J, Karino Y, et al. Gastric varices due to splenic vein occlusion with left renal cell carcinoma. Dig Endosc 1996;8:166–9.
18. Heseltine D, Bramble M, Cole A. Weber-Christian disease producing splenic vein occlusion and bleeding gastric varices: successful treatment with sclerotherapy. Postgrad Med J 1990;66:321–35.
19. Evans GRD, Yellin AE, Weaver FA, Stain SC. Sinistral (left-sided) portal hypertension. Am Surg 1990;56:758–63.
20. Goldman ML, Philip PK, Sarrafizadeh MS, Sarfeh IJ, Salam AA, Galambos JT, et al. Intra-arterial tissue adhesive for medical splenectomy in humans. Radiology 1981;140:341–9.
21. Mcdermott VG, England RE, Newman GE. Case report: bleeding gastric varices secondary to splenic vein thrombosis successfully treated by splenic artery embolization. Br J Radiol 1995;68: 928–30.
22. Adams DB, Mauterer DJ, Vujic IJ, Anderson MC. Preoperative control of splenic artery inflow in patients with splenic venous occlusion. South Med J 1990;83:1021–4.