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Abstract

Obliteration for gastric or duodenal variceal hemorrhage was performed via transileocoecal or transhepatic portal catheterization in 8 patients with portal hypertension. The patients were 6 men and 2 women, whose average age was 59 years. All of the patients had cirrhosis of the liver. The obliteration was performed as an emergency procedure in 6 cases, and 2 patients were electively treated. Transileocoecal obliteration (TIO) and transhepatic obliteration (PTO) were selected for 6, and 2 patients, respectively. Variceal bleeding was successfully controlled in all patients after completion of the therapy. One patient died after 3 months when duodenal variceal bleeding recurred. Elective surgical operations were performed on 2 patients have survived to date without bleeding. Transient oliguria and jaundice after the therapy were noticed in 2 patients. Histological examination revealed cast formation of polymerized cyanoacrylate in the obliterated gastric varices of 2 patients. TIO and PTO seem to be safe, effective procedures to stop bleeding from ectopic varices, gastric or duodenal. This therapy is useful either to obtain accurate information about the varices or to obliterate the collateral veins in patients with ruptured ectopic varices.

KEYWORDS: cardial varices, duodenal varices, portal hypertension, variceal obliteration, cyanoacrylate

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Obliteration for gastric or duodenal variceal hemorrhage was performed via transileocoecal or transhepatic portal catheterization in 8 patients with portal hypertension. The patients were 6 men and 2 women, whose average age was 59 years. All of the patients had cirrhosis of the liver. The obliteration was performed as an emergency procedure in 6 cases, and 2 patients were electively treated. Transileocoecal obliteration (TIO) and trenshepatic obliteration (PTO) were selected for 6, and 2 patients, respectively. Variceal bleeding was successfully controlled in all patients after completion of the therapy. One patient died after 3 months when duodenal variceal bleeding recurred. Elective surgical operations were performed on 2 patients after the initial therapy, because the vein feeding toward the varices remained. Six of the patients have survived to date without bleeding. Transient oliguria and jaundice after the therapy were noticed in 2 patients. Histological examination revealed cast formation of polymerized cyanoacrylate in the obliterated gastric varices of 2 patients. TIO and PTO seem to be safe, effective procedures to stop bleeding from ectopic varices, gastric or duodenal. This therapy is useful either to obtain accurate information about the varices or to obliterate the collateral veins in patients with ruptured ectopic varices.

Key words : cardial varices, duodenal varices, portal hypertension, variceal obliteration, cyanoacrylate

The prognosis for patients with portal hypertension and esophageal varices is improving through the development of management for bleeding esophageal varices. The development of endoscopic injection sclerotherapy (EIS) has been particularly useful. Gastric varices, however, have been reported to be difficult to control by EIS (1). Several reports described the formation of new gastric varices after the elimination of esophageal varices by EIS (2, 3). In this paper, hemostatic efficacy of transileocoecal obliteration (TIO) or transhepatic obliteration (PTO) for bleeding gastric and duodenal varices are reported.

Subjects and Methods

Seven patients with gastric variceal hemorrhage and 1 patient with duodenal variceal hemorrhage were treated by TIO or PTO in the period from January 1988 to May 1991 in our hospital and affiliated hospitals. The ages of the 8 patients ranged from 30-74 years (average, 59 years). The common background disease for the 6 men and 2 women was cirrhosis of the liver (Table 1). Primary liver cancer accompanied liver cirrhosis in 2 patients. The sites of variceal hemorrhage were determined endoscopically to be the gastric cardial varices in 4 patients, the gastric fundal varices in 1 patient, both cardial and fundal varices in 2 patients, and the duodenal varices in 1 patient. EIS for esophageal variceal hemorrhage had been previously performed on 3 patients; 2 years before in Case 2, 3 years before in Case 3, 3 months before in Case 6, respectively. The emergency EIS was not effective for gastric variceal bleeding in 2 patients (Cases 1, 6). Apparent jaundice was recognized in 2 patients, and 5 patients had ascites. According to Child's classification, 1 patient was grade A, 3 patients were grade

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ł	Site of varices	Clinical diagnosis	Child-Pugh score	Ascites	T. Bil (mg/dl)	Age (sex)	Case
f Em	Lgc, Lgf	LC	13	+	21.5	30 (F)	1
m Em	Duodenum	LC + HCC	9	+	1.9	61 (F)	2
Elec	Lgc	LC	7	_	0.9	63 (M)	3
Em	Lgc	LC	12	++	0.9	74 (M)	4
Em	Lgf	LC	7	—	1.1	56 (M)	5
Eme	Lgc	LC + HCC	11	++	2.1	57 (M)	6
Elec	Lgc	LC	5	_	0.4	66 (M)	7
f Eme	Lgc, Lgf	LC	11	+	4.2	65 (M)	8

Table	1 (linical	diagnosis	site o	of varices	severity	of liver	disease	and	procedure
raoic	1 0	/mnca	ulagilosis,	SILC (л vances,	Sevency	OI IIVEI	uisease,	anu	procedure

LC: Liver cirrhosis; HCC: Hepatocellular carcinoma; Lgc: Cardia varices; Lgf: Fundus varices; TIO: Transileocecal obliteration; PTO: Percutaneous transhepatic obliteration.

B, and 4 patients were grade C. The esophageal varices were mild in all patients; F1 form in 4 patients, F2 form in 1 patient according to the criteria of the Japan Gastroenterological Endoscopy Society, and 3 patients showed no esophageal varices endoscopically. The red color sign was observed in 5 out of 7 gastric varices.

TIO was selected for 6 patients and PTO was selected for 2 patients. The catheter was inserted into the ileocoecal vein, then proceeded toward the superior mesenteric vein through a small incision of the right lower abdomen under general anesthesia in TIO. The catheter for PTO was inserted into the intrahepatic portal vein, then proceeded toward the main portal vein according to the US guided puncture procedure, which was conducted under local anesthesia. The catheter was selectively inserted into the vein which connected to the ruptured varices, and embolic material was injected. Ethyl-alfa-cyanoacrylate (CA) was used in 6 patients, a combination of stainless steal coil and CA was used in 1 patient, and absolute ethanol was used in 1 patient. The obliteration was repeatedly performed in all veins flowing into the ruptured varices until the outflow toward the varices disappeared. Portal vein pressure was measured in the main portal vein before and after the obliteration.

Results

Portal vein pressure, veins supplying to the varices, and communications to the esophageal varices. The feeding veins which flowed into the ruptured varices were, gastric coronary veins, short gastric veins, and posterior gastric veins (Table 2). Only one connection between the esohageal varices and gastric varices was observed (Case 3). Large portal systemic shunts via the gastrorenal or the inferior vena cava were observed in 7 of 8 cases.

Changes of portal vein pressure. Portal vein pres-

sure was measured before and after the obliteration of the varices. Portal vein pressure ranged from 250 to 510 mmH₂O (mean, 318 mmH₂O). After the obliteration, pressure ranged from 285 to $580 \text{ mmH}_2\text{O}$ (mean rise in pressure, $42 \text{ mmH}_2\text{O}$) (Fig. 1).

Table 2 Portal vein pressure and abnormal portal circulation

Case Portal vein (mmH ₂ O)		Feeding veins to varices	Communication to esophageal varices	Portal systemic shunt		
1	400	GCV, SGV	_	GRS		
2	280	IPDV	—	IVC		
3	250	GCV, SGV, PGV	Partial	GRS		
4	340	GCV, SGV	_	GRS		
5	305	SGV, PGV, GCV		GRS		
6	510	GCV, SGV	—	GRS		
7	290	PGV	—	_		
8	285	PGV, GCV, SGV	—	GRS		

GCV: Gastric coronary vein; SGV: Short gastric vein; IPDV: Inferior pancreatic duodenal vein; PGV: Posterior gastric vein; GRS: Gastrorenal shunt; IVC: Inferior vena cava.

Table 3 Success rate and completion of obliteration.

Case	Hemostasis	Degree of obliteration	Aditional therapy	Prognosis (Month)
1	Success	Complete		Died of pneumonia (1M)
2	Success	Complete	_	Died of bleeding (3M)
3	Success	Complete	_	Alive (20M)
4	Success	Complete		Alive (18M)
5	Success	Incomplete ^a	Operation	Alive (15M)
6	Success	Incomplete ^a	Operation	' Alive (11M)
7	Success	Complete	_	Alive (11M)
8	Success	Complete	_	Alive (9M)

a: Remaining small veins feeding varices.

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Complications, additional therapy, and prognosis. Transient oliguria and jaundice were observed in 2 patients after the obliteration, but these symptoms improved quickly. Surgical operations were performed on 2

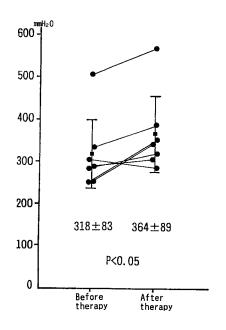


Fig. 1 Elevation of portal vein pressure after obliteration of the varices. After the obliteration, portal vein pressure elevated significantly compared to the pressure before therapy (t test, p < 0.05).

patients, because obliteration by TIO was incomplete (Table 3). Variceal hemorrhage was controlled by the obliteration in all patients. One patient died of pneumonia 1 month after the therapy, but re-bleeding did not occur. Another patient with duodenal varices and primary liver cancer manifested re-bleeding 30 days after the initial therapy, and she died of liver failure after 3 months. The other 6 patients have survived to date without bleeding, and the survival term ranged from 9 to 20 months.

Histology of the obliterated varices. Histological examination of material from the obliterated varices of 2 patients, 1 an autopsy and 1 from a surgical biopsy showed few changes in the shape of obliterated varices. Solid cast-like materials were observed inside the varices. Amorphous materials observed microscopically were apparently polymerized cyanoacrylate (Fig. 2).

Discussion

Bleeding from gastric varices often poses serious clinical problems, because gastric variceal bleeding is more difficult to control than bleeding from the esophageal varices, and the incidence of gastric variceal rupture is not rare (4, 5). Hemostasis of ruptured esophageal varices is not difficult to achieve in most of the patients, given the development of the EIS technique. After EIS for eso-

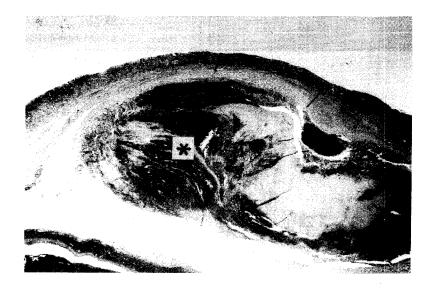


Fig. 2 Histology of the varices surgically resected in patient 5. The vein was occluded by Cyanoacrylate polymer (*).

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phageal varices, observations of gastric variceal deterioration (6), or the appearance of new gastric varices (2) have been reported. When portal blood flow toward the esophageal varices is intercepted by EIS, new gastric varices appear alongside the gastric veins because of elevation of the portal vein pressure. In the present group of cases, 3 patients had a history of EIS for esophageal varices.

Successful EIS for gastric varices has been exceptionally reported (7), but in most of the cases, gastric varices are resistant to EIS, and the hemostatic effect of EIS has been poor, especially in patients with severe gastric varices (1). The insufficient efficacy of EIS for gastric varices is supposed to be due to the large hepatofugal blood flow toward the gastric varices. Large gastrorenal shunts were often observed in patients with ruptured gastric varices (8). The use of a large dose of injection materials such as ethanolamine oleate is avoided to prevent the outflow into the systemic circulation, because it is difficult to intercept the blood flow in the gastrorenal shunt by means of a balloon occlusion. It is also difficult to clarify the abnormal portal circulation by endoscopical observation alone. Fortunately, portal vein catheterization in TIO and PTO facilitates the selection of the best therapy by providing a clear portal venogram.

PTO was reported to be an effective hemostatic procedure for ruptured esophageal varices by Lunderquist and Vang (9) in 1974, and the same results have been described elsewhere (10–12). Recently, PTO is often performed to stop the gastric variceal hemorrhage because of its strong hemostatic effect in large varices. It is important to embolize all the collateral veins that flow into varices to obtain a long-term hemostatic effect (10). In the present study, complete obliteration was not achieved in 2 patients, where the remaining small veins which fed to varices remained. Those veins were completely removed by elective surgical operation.

Cyanoacrylate was used in 7 patients as an embolization material. This material immediately intercepts the blood flow mechanically in large vessels by polymerization *in situ* after the injection. Immediate polymerization and obliteration is suitable for use in large gastric varices with a high blood flow, but it is important to select peripheral vessels as a site for obliteration, because the obliteration occurs just at the injection site of the cyanoacrylate. Absolute ethanol appeared to have an excellent hemostatic effect in patient with a poor gastro-renal shunt.

Recanalization of obliterated veins or the appearance of new collaterals which feed varices after PTO or TIO have been reported (13, 14). Two patients of our series underwent surgical operations, because TIO was thought to be insufficient. Another patient (Case 8) showed new cardial varices 9 months after the initial therapy and follow-up portography demonstrated a new formation of collateral vessels. In this case, a second PTO was performed and the new collateral veins were completely obliterated. It seems to be important to follow the patient's progress carefully after the initial therapy, and to repeat the obliteration when new varices develop, to obtain long-term hemostatic effect. EIS after initial PTO was attempted recently to achieve more complete obliteration (12, 16).

In conclusion, hemostasis of ectopic variceal hemorrage such as gastric or duodenal varices by EIS is difficult, and therefore, urgent hemostasis is necessary. TIO and PTO is an excellent method to obtain the most accurate information about the varices. It is also a safe, reliable hemostatic procedure for the urgent control of gastric variceal hemorrhage.

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