Abdom Imaging 25:567–571 (2000)
DOI: 10.1007/s002610000046

Abdominal Imaging

Transjugular retrograde obliteration for chronic portosystemic encephalopathy

F. Chikamori,[1] **N. Kuniyoshi,**[2] **S. Shibuya,**[3] **Y. Takase**[3]

[1]Department of Surgery, Kuniyoshi Hospital, 1-3-4 Kamimachi, Kochi City, Kochi 780-0901, Japan
[2]Department of Internal Medicine, Kuniyoshi Hospital, 1-3-4 Kamimachi, Kochi City, Kochi 780-0901, Japan
[3]Department of Surgery, Tsukuba Soai Hospital, Japan

Received: 27 January 2000/Accepted: 23 February 2000

Abstract
Chronic portosystemic encephalopathy (CPSE) is uncommon, and its management has yet to be determined. We have been able to control five cases of CPSE using transjugular retrograde obliteration (TJO), and we report our clinical results with this technique. All of the five patients were suffering from cirrhosis and had gastric varices and large gastrorenal shunts. According to Sherlock's classification, the grade of encephalopathy was II in two patients, III in two, and IV in one. According to Child's classification, one had class B and four had class C cirrhosis. TJO was performed using a 6-F angiographic catheter with an occlusive balloon 20 mm in diameter. Absolute ethanol and 5% ethanolamine oleate with iopamidol were used to obliterate the gastrorenal shunt. The gastrorenal shunt was successfully obliterated, and the encephalopathy improved to grade 0 after TJO in all cases. The portal flow volume increased significantly from 542 ± 189 to 992 ± 139 mL/min ($p < 0.01$). The plasma ammonia levels before and after TJO were 189 ± 40 and 51 ± 23 μg/dL, and the indocyanine green retention rates at 15 min were 44 ± 13% and 27 ± 12%, with both changes being significant ($p < 0.01$). Minor complications observed were fever of over 38°C and tarry stools due to hemorrhagic gastritis in one patient, which was being controlled conservatively. One patient died of hepatocellular carcinoma 27 months after TJO. The other four patients survived without recurrence of CPSE 17–74 months (44 ± 24 months) after TJO. We conclude that TJO can be adopted as a safe and effective treatment for CPSE.

Correspondence to: F. Chikamori

Key words: Transjugular retrograde obliteration—Chronic portosystemic encephalopathy—Gastrorenal shunt.

Chronic encephalopathy due to spontaneous large portosystemic shunt is uncommon [1, 2]. Chronic portosystemic encephalopathy (CPSE) is sometimes associated with large gastric varices [3]. Transcatheter retrograde obliteration has been reported to be effective for the treatment of gastric varices [4, 5], but its effectiveness for managing CPSE has yet to be determined. We have been able to control five cases of CPSE using transjugular retrograde obliteration (TJO), and we report the clinical results obtained with this technique.

Patients and methods

Between June 1992 and April 1998, five patients with CPSE presented at our hospital. All of them had a repeated episode of mental confusion, disorientation, flapping tremor, and hyperammonemia and required prolonged protein restriction, lactulose, and branched-chain amino acid treatment. According to Sherlock's classification [6], grade of encephalopathy was II in two patients, III in two, and IV in one. All had liver

Table 1. Patients with chronic portosystemic encephalopathy

Case	Age (years)	Sex	Primary disease	Child's classification	Grade of encephalopathy
1	65	F	LC	C	II
2	51	F	LC with HCC	C	III
3	62	F	LC	C	III
4	66	M	LC with HCC	C	IV
5	57	M	LC	B	II

LC, liver cirrhosis; HCC, hepatocellular carcinoma

Table 2. Blood laboratory examinations before and after transjugular retrograde obliteration for chronic portosystemic encephalopathy

Case	Total bilirubin		Serum albumin		NH_3[a]		ICG15[a]	
	Before	After	Before	After	Before	After	Before	After
1	1.5	1.8	2.8	3.3	149	68	32	17
2	2.2	2.0	2.9	2.9	210	60	57	40
3	1.0	1.0	2.6	2.8	243	75	46	31
4	1.6	1.1	2.8	3.5	194	27	54	34
5	2.0	1.3	3.2	3.0	150	26	29	11
Mean	1.7	1.4	2.9	3.1	189	51	44	27

ICG15, indocyanine green retention rate at 15 min
[a] $p < 0.01$

Table 3. Plasma ammonia and total bile acid levels before transjugular retrograde obliteration

Blood sample	Ammonia (μg/dL)	Total bile acid (μM/L)
Superior vena cava	156	62.5
Hepatic vein	44	24.7
Inferior vena cava	186	67.0
Left renal vein	201	67.1
Gastrorenal shunt	261	87.7

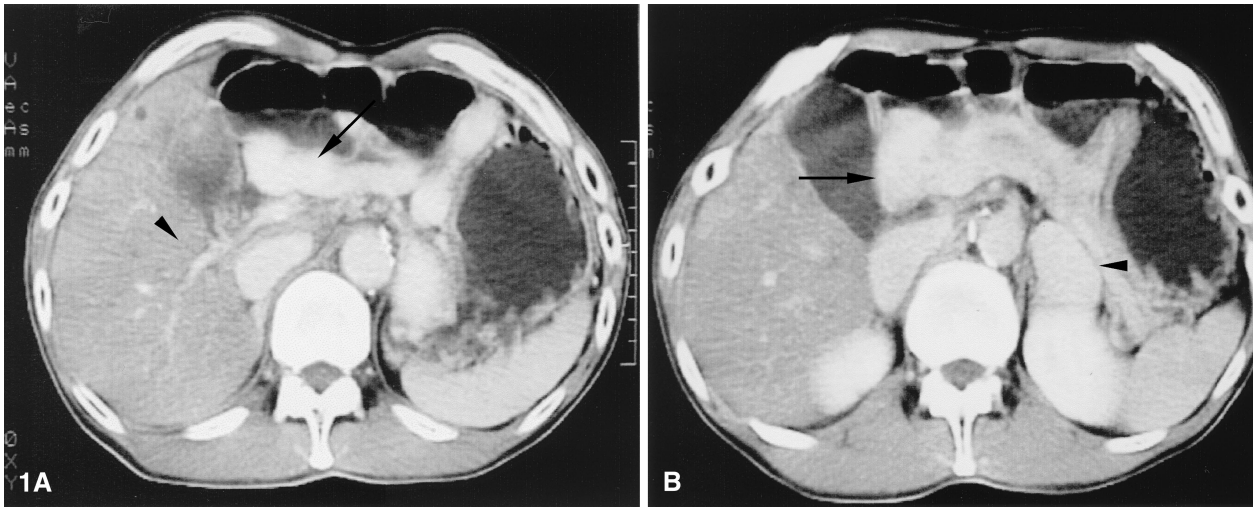
cirrhosis, and two had hepatocellular carcinoma. The cause of cirrhosis was hepatitis C in two, alcohol abuse in two, and unknown in one. The patients ranged in age from 51 to 66 years; two were men, and three were women. According to Child's classification, one had class B and four had class C cirrhosis (Table 1). None had ascites or jaundice. Although none had a history of variceal bleeding, endoscopy showed gastric varices in all cases. The gastric varices were located in the fundus (Lg-f) in four patients and in both the cardia and the fundus (Lg-cf) in one patients; the forms of these varices were tortuous (F1) in one, nodular (F2) in three, and tumorous (F3) in one. Three patients had undergone endoscopic injection sclerotherapy for esophageal varices. The portosystemic shunt was examined by splenic and superior mesenteric arterial portography. The portosystemic shunt in all five cases was gastrorenal. Superior mesenteric arterial portography showed that a large volume of superior mesenteric venous blood drained hepatofugally into the systemic circulation through this shunt, and the intrahepatic portal vein was poorly opacified.

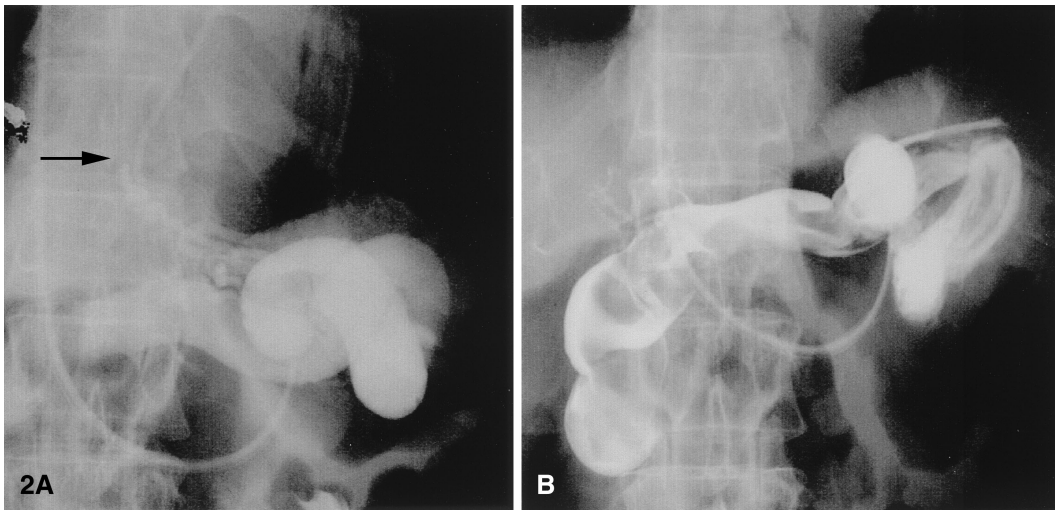
TJO was performed using a 6-F angiographic catheter with an occlusive balloon 20 mm in diameter. Through the right internal jugular vein, this catheter was inserted into the gastrorenal shunt through the superior vena cava, the inferior vena cava, and the left renal vein. When the top of the catheter was inserted into the shunt beyond the bifurcation of the inferior phrenic vein, we called it *superselective access*. After occlusion of the shunt, absolute ethanol was injected to obliterate the other blood-draining routes of the shunt such as retroperitoneal or azygos veins. Then 5% ethanolamine oleate with iopamidol (5%EOI) was injected into the shunt. The catheter was left for 24 h and removed after confirming the obliteration of the shunt with thrombi under shunt venography. The technical details of this method have been reported elsewhere [4].

The wedged hepatic venous pressure (WHVP) was examined immediately before TJO and the day after. The portal blood flow was examined by pulsed Doppler flowmetry 1 week before and 1 week after TJO by the same investigator. The device used was an ultrasonic duplex system composed of a real-time electronic convex-type scanner and a pulsed Doppler flowmeter (Hitachi Medical, EUB-655, Tokyo, Japan). The portal venous flow was measured in the supine position after an overnight fast. The sample point was set at the central portion of the portal vein trunk, at the midpoint between the confluence of the superior mesenteric and splenic veins, and at the branching portion of the intrahepatic portal branches. The Doppler angle was less than 60°. Blood laboratory parameters were evaluated 1 week before and 1 week after the operation. The five patients who underwent this procedure were followed up for 17–74 months.

Results

In three of these patients, the gastrorenal shunt was obliterated superselectively and the inferior phrenic vein was unaffected after TJO. In the other patients, the top of the catheter could not be inserted beyond the bifurcation of the inferior phrenic vein, so the gastrorenal shunt and the inferior phrenic vein were selectively obliterated. The volumes of sclerosant required for TJO were 5.0 ± 3.7 mL absolute ethanol and 16.4 ± 13.5 mL 5%EOI. The gastrorenal shunt was successfully obliterated, and the encephalopathy improved to grade 0 after TJO in all cases. The WHVP before and after TJO was 249 ± 33 and 298 ± 32 mmH_2O, respectively; the change was significant ($p < 0.01$). The portal flow velocity before and after TJO was 13 ± 4 and 17 ± 2 cm/s, respectively, without a significant difference, although the portal cross-sectional area increased significantly from 0.70 ± 0.14 to 1.01 ± 0.22 cm^2 and the portal flow volume increased significantly from 542 ± 189 to 992 ± 139 mL/min ($p < 0.05$ and 0.01, respectively). The serum albumin levels before and after TJO were 2.9 ± 0.2 and 3.1 ± 0.3 g/dL, respectively, and the total bilirubin levels were 1.7 ± 0.5 and 1.4 ± 0.4 mg/dL, respectively; neither level changed significantly. The plasma ammonia levels before and after TJO were 189 ± 40 and 51 ± 23 μg/dL, respectively, and the indocyanine green retention rates at 15 min were 44 ± 13 and 27 ± 12%, with both showing a significant change ($p < 0.01$; Table 2).





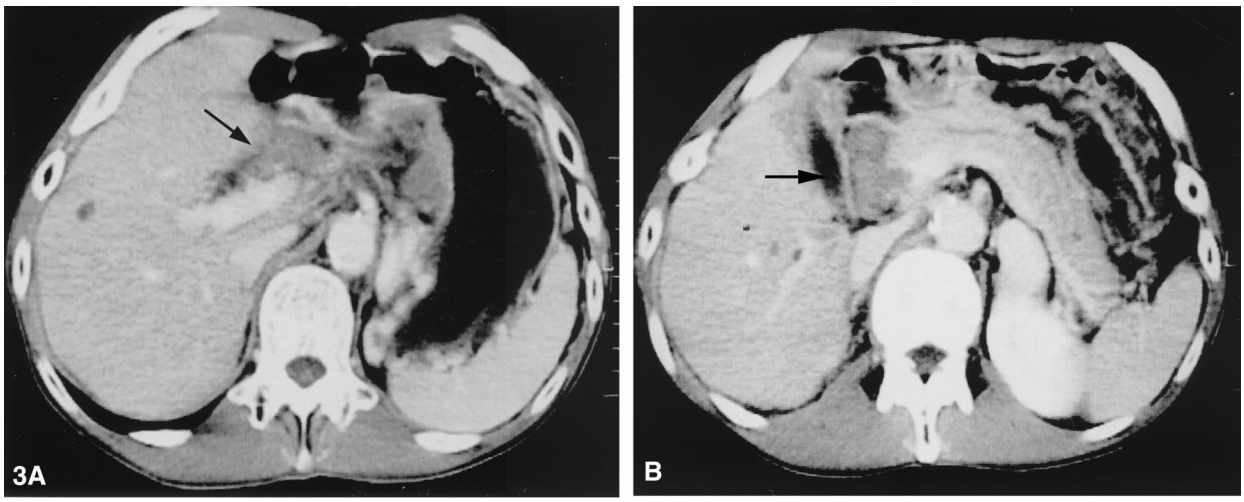


Fig. 1. A CT shows the large gastrorenal shunt (*arrow*) and the narrowed portal vein (*arrowhead*). **B** CT shows that the root of the left gastric vein (*arrow*) and the gastrorenal shunt (*arrowhead*) are almost the same size as that of the inferior vena cava.

Fig. 2. A Retrograde shunt venogram shows the gastrorenal shunt and communicating vessels (*arrow*) to the azygos vein. **B** Retrograde shunt venogram performed the day after shows thrombi in the gastrorenal shunt.

Fig. 3. A CT 1 week after TJO shows that the gastrorenal shunt has been completely obliterated by thrombi (*arrow*) and that the portal vein has enlarged. **B** CT 1 week after TJO shows that the root of the left gastric vein has been completely obliterated by thrombi (*arrow*).

Minor complications observed were fever of over 38°C and tarry stools due to hemorrhagic gastritis in one patient (Case 5), although the problems were controlled conservatively. The duration of the hospital stay after TJO was 6–28 (14 ± 9) days. During the follow-up period, there was no recurrence of CPSE. Esophageal varices occurred in three patients (nos. 1–3) who had undergone endoscopic injection sclerotherapy. One patient (no. 2) died of hepatocellular carcinoma 27 months after TJO. The other four patients survived 17–74 months (44 ± 24) after the procedure.

Case report (patient 5)

A 57-year-old man presented at our hospital in a confused, apathetic, tremulous, and forgetful condition. The grade of encephalopathy was II. The plasma ammonia and total bile acid levels were abnormally elevated to 230 μg/dL and 59.5 μM/L, respectively. Ultrasonography and computed tomography (CT) showed a large gastrorenal shunt and a narrowed portal vein (Fig. 1A,B). Superior mesenteric arterial portography showed that most of the superior mesenteric venous blood was draining into the large gastrorenal shunt through the left gastric vein, and the main portal vein was not opacified.

The plasma ammonia and total bile acid levels before TJO were lowest in the hepatic vein and highest in the gastrorenal shunt (Table 3). Retrograde shunt venography visualized the gastrorenal shunt and communicating vessels to the azygos vein (Fig. 2A). A 6-F occlusive balloon catheter was inserted superselectively into the gastrorenal shunt. After obliteration of the communicating vessels to the azygos vein with 5 mL absolute ethanol, 13 mL 5%EOI was injected into the gastrorenal shunt as far as the root of the left gastric vein.

Retrograde shunt venography was performed the next day, showing thrombi in the gastrorenal shunt (Fig. 2B). CT 1 week after TJO showed that the gastrorenal shunt had been completely obliterated by the thrombi and that the portal vein had enlarged (Fig. 3A,B). The WHVP increased from 290 to 340 mmH_2O and the portal flow volume increased from 210 to 1010 mL/min.

The encephalopathy improved to grade 0 after TJO, and the plasma ammonia and total bile acid levels were reduced to 31 μg/dL and 10.5 μM/L, respectively. Tarry stools due to hemorrhagic gastritis occurred 2 days after TJO, but this was treated conservatively without blood transfusion. The patient was discharged on day 13 after TJO. Follow-up examinations 23 months after TJO have indicated no recurrence of encephalopathy, and there has been no need for protein restriction or administration of lactulose and branched-chain amino acid. The plasma ammonia and total bile acid levels continue to be normal.

Discussion

CPSE resulting from a large collateral shunt is less common than hepatic encephalopathy resulting from the terminal stage of decompensated cirrhosis. The frequency of CPSE is 1.6% (7/445) in patients with cirrhosis [2] and 15% (5/33) in patients with severe gastric varices [3]. Up to now, we have seen five cases (11%) of CPSE among 45 patients with gastric varices treated by TJO.

It has been suggested that CPSE results from a shunt of a large proportion of the superior mesenteric venous blood into the systemic circulation [2, 7]. The pathogenesis of CPSE is like that which occurs after a nonselective mesocaval shunt operation [8]. Surgical ligation of the shunt is an effective method for CPSE, but it is invasive and associated with high mortality [9]. Percutaneous transhepatic obliteration is one of the nonsurgical treatments of CPSE [10] but has certain complications such as intraperitoneal bleeding. Therefore, the most appropriate treatment of CPSE has yet to be established.

The use of transcatheter retrograde obliteration techniques, such as TJO, for isolated gastric varices has been increasing recently in Japan [4, 5] because such techniques enable gastric varices to be eradicated easily and are less invasive than other methods. However, the effectiveness of TJO for CPSE has yet to be determined. Kawanaka et al. [11] reported the first case of successful reversion of CPSE by transcatheter retrograde obliteration through a femoral vein approach using 33 mL 5%EOI. In June 1992, we began to use TJO for treatment of CPSE. TJO has an advantage over the femoral approach in being able to obliterate the shunt superselectively. The quantity of 5%EOI administered should be less than 0.5 mL/kg because an excess volume can cause renal injury or lung congestion. Superselective embolization can reduce the dose of sclerosant required for shunt obliteration [4].

As a complication, one patient suffered tarry stools due to hemorrhagic gastritis, which may have been due to congestion of the portal system after TJO. After TJO, it is necessary to prescribe drugs such as diuretics, H_2 receptor antagonists, and antacids. Endoscopic follow-up at periodic intervals is also required because treatment of CPSE may encourage the occurrence of esophageal varices. Because shunt occlusion may carry a risk of development of refractory ascites or hepatic failure, it should be applied carefully. The ratio of plasma ammonia in the hepatic vein to that in the shunt may become an index of the degree of success of shunt obliteration for CPSE. Further investigations of this index and the effects of TJO on CPSE are needed. We conclude that TJO can be adopted as a safe and effective treatment for CPSE.

References

1. Sherlock S, Summerskill WHJ, et al. Portal-systemic encephalopathy. Neurological complications of liver disease. *Lancet* 1954;2: 453–457
2. Takashi M, Igarashi M, Hino S, et al. Portal hemodynamics in chronic portal-systemic encephalopathy. Angiographic study in seven cases. *J Hepatol* 1985;1:467–476
3. Watanabe K, Kimura K, Matsutani S, et al. Portal hemodynamics in patients with gastric varices, a study in 230 patients with esophageal and/or gastric varices using portal vein catheterization. *Gastroenterology* 1988;95:434–440
4. Chikamori F, Shibuya S, Takase Y, et al. Transjugular retrograde obliteration for gastric varices. *Abdom Imaging* 1996;21:299–303
5. Kanagawa H, Mima S, Kouyama H, et al. Treatment of gastric fundal varices by balloon-occluded retrograde transvenous obliteration. *Gastroenterol Hepatol* 1996;11:51–58

6. Sherlock S. Hepatic coma. *Gastroenterology* 1968;54:754–757
7. Smith-Laing G, Camilo ME, Dick R, et al. Percutaneous transhepatic portography in the assessment of portal hypertension. Clinical correlations and comparison of radiographic techniques. *Gastroenterology* 1980;78:197–205
8. Rikkers LF. Portal hemodynamics, intestinal absorption and postshunt encephalopathy. *Surgery* 1983;94:126–133
9. Hana SS, Smith RS, Henderson JM, et al. Reversal of hepatic encephalopathy after occlusion of total portasystemic shunts. *Am J Surg* 1981;142:285–289
10. Uflacker R, de O e Silva A, Carneiro d Albuquerque LA, et al. Chronic portosystemic encephalopathy: embolization of portosystemic shunts. *Radiology* 1987;165:721–729
11. Kawanaka H, Ohta M, Hashizume M, et al. Portosystemic encephalopathy treated with balloon-occluded retrograde transvenous obliteration. *Am J Gastroenterol* 1995;90:508–510