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Masazumi Fujimoto
Fuminori Moriyasu
Hitoshi Someda
Takayuki Nada
Minoru Okuma
Shinji Uemoto
Yukihiro Inomata
Koichi Tanaka
Yoshio Yamaoka
Kazue Ozawa

Recovery of graft circulation following percutaneous transluminal angioplasty for stenotic venous complications in pediatric liver transplantation: assessment with Doppler ultrasound

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M. Fujimoto (✉) · F. Moriyasu
H. Someda · T. Nada · M. Okuma
First Department of Internal Medicine,
Kyoto University School of Medicine,
54 Shogoin, Kawaharacho, Sakyo-ku,
Kyoto 606-01, Japan
Fax: +81 75 751 3201

S. Uemoto · Y. Inomata · K. Tanaka
Y. Yamaoka
Second Department of Surgery,
Kyoto University School of Medicine,
54 Shogoin, Kawaharacho, Sakyo-ku,
Kyoto 606-01, Japan

K. Ozawa
Shiga Prefectural Medical College,
Setatsukinowa-cho, Otsu-city, Shiga
520-21, Japan

Abstract Percutaneous transluminal angioplasty was performed for venous stenosis after living related liver transplantation in three children. Two of them had hepatic vein stenosis and one had stenosis of both the hepatic and portal veins. Progressive development of ascites and deterioration of liver function were found in all cases. Serial Doppler ultrasound studies showed that the flow velocity in the hepatic vein gradually decreased with a flattened velocity waveform, followed by a decrease in portal blood flow. After a successful hepatic vein angioplasty, the velocity in the hepatic and portal veins increased and the Doppler waveform in the hepatic vein became pulsatile in two cases. In the remaining case, a remarkable recovery of both graft perfusion and clinical findings was achieved via combined hepatic vein and portal vein angioplasty. We conclude that balloon angioplasty is an effective alternative to surgery for post-transplant vascular stenosis and that Doppler ultrasound is useful in monitoring graft circulation.

Key words Liver transplantation, portal vein stenosis, percutaneous transluminal angioplasty · Portal vein stenosis, liver transplantation, percutaneous transluminal angioplasty · Percutaneous transluminal angioplasty, portal vein stenosis, liver transplantation

Introduction

After orthotopic liver transplantation, stenotic vascular complications related to the portal or hepatic vein occur less frequently than those of the hepatic artery or biliary tract [4, 13, 15]. To our knowledge, no pediatric cases with postoperative hepatic vein stenosis have been reported in detail in partial liver transplantation from living donors. Yet, Zajko et al. have reported two cases of obstruction to hepatic venous drainage due to stenosis of the suprahepatic inferior vena caval anastomosis after liver transplantation from cadavers [16]. The few cases with portal vein stenosis were mainly treated by surgical reconstruction [12, 13, 15]. In this article, we describe three patients in whom percutaneous transluminal angioplasty (PTA) for hepatic vein stenosis or portal vein stenosis was successfully performed, and we discuss the usefulness of Doppler ultrasound (US) in examining hemodynamic states during such stenotic vascular complications, as well as in monitoring the therapeutic effects of PTA.

Patients and methods

Between June 1990 and December 1993, we performed a series of 83 partial liver transplantations from living related donors on 82 children with end-stage liver disease (31 males and 51 females, mean age 4.2 years) at the Second Department of Surgery, Kyoto University Hospital [8]. Doppler US examinations were carried out on all recipients to estimate vascular anastomosis intraoperatively and for postoperative follow-up monitoring of graft perfusion. A color Doppler US system (SSA-270A, Toshiba Corporation, Tokyo, Japan), equipped with a phased array scanner operating at 3.75 MHz in imaging mode and 3.0 MHz in Doppler mode,

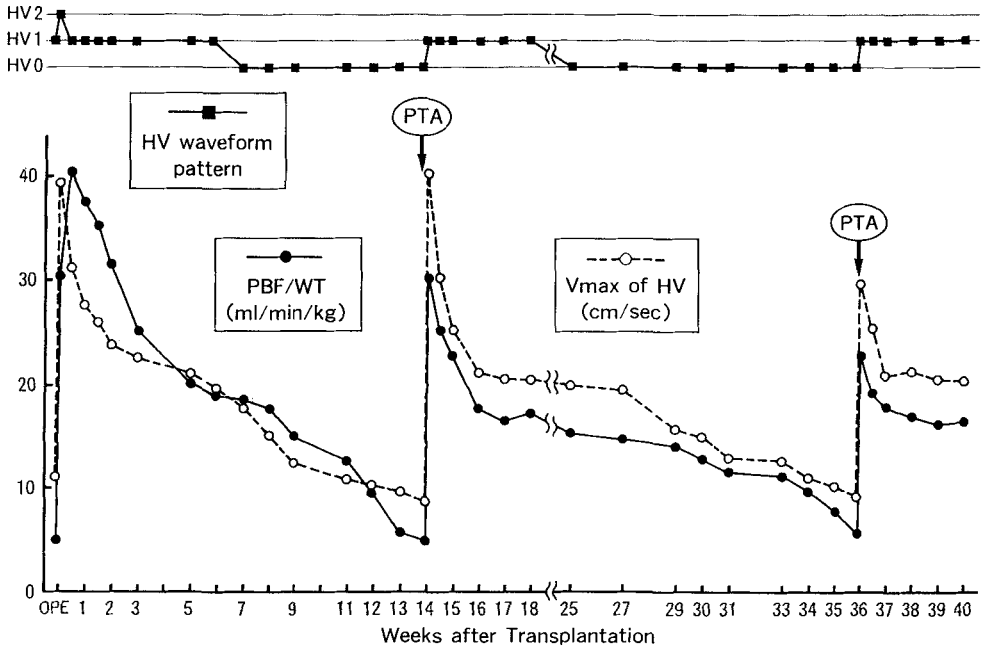

Fig. 1 Serial determinations of portal blood flow and hepatic venous blood flow during the follow-up period in case 1. (*PBF/WT* portal blood flow volume per kg body weight, V_{max} *of HV* maximum flow velocity of hepatic vein, *HV* waveform pattern: *HV0* completely flat waveform without any cardiac oscillations, *HV1* biphasic pattern without the short phase of reversed flow, *HV2* normal triphasic waveform)

was used. The blood flow in the reconstructed portal vein, hepatic vein, and hepatic artery were evaluated daily during the 2-week period following surgery. After postoperative day (POD) 14, the frequency of follow-up scans was determined by the clinical findings in each patient. Our examinations focused on (1) the peak systolic velocity in the hepatic artery at the proximal site of the intrahepatic branch along the umbilical portion of the portal vein, (2) the portal blood flow volume, which is calculated by multiplying the angle-corrected mean portal venous velocity and the cross-sectional area of the portal trunk [7], and (3) the pulsatility of the velocity waveform and the maximum velocity in the hepatic vein at a distance of 0.5–2 cm from the anastomotic site.

After transplantation, three patients developed hepatic vein stenosis and one of the three also developed portal vein stenosis, although the vascular anastomosis had been judged adequate at surgery. All three patients were female and all developed secondary biliary cirrhosis after Kasai surgery for congenital biliary atresia; the left lateral hepatic segment of their mothers' livers had been harvested at 11, 14, and 23 months, respectively, after birth. Clinical symptoms due to the vascular complications gradually became manifest after about 2 months postoperatively. Liver biopsy findings showed no evidence of rejection in the three cases.

PTA was performed transhepatically under general anesthesia. The graft liver was usually located in the epigastric region, and the left hepatic vein or the lateral segmental branch of the portal vein was punctured via an epigastric approach under ultrasonographic guidance. Then, a 7.5-Fr sheath-introducer system was inserted under fluoroscopic guidance. Hepatic venography or portography confirmed that stenosis had developed at the site of anastomosis between the donor and recipient vessels. A 0.035-inch guide wire was inserted through the stenotic portion and a dilating balloon catheter (balloon length 3 cm, inflated diameter 10 mm; Medi-tech/Boston Scientific, Watertown, Mass., USA) was advanced over the guide wire through the stenosis. The balloon was inflated three or four times for 5 min each time. After the procedures, subsequent venography was performed to confirm the therapeutic effects. Then, the sheath was withdrawn and hemostasis was maintained by manual compression. Using Doppler US, we also examined graft circulation by follow-up monitoring after PTA.

The present series of living related liver transplantations and PTA were approved by the Ethics Committee of Kyoto University and carried out after informed consent was given by the patients' parents.

Case reports

Case 1

A 14-month-old female with biliary atresia received the left lateral hepatic segment from her mother. The recipient hepatic veins were too small and sclerotic to be used for anastomosis, so the hepatic vein of the graft liver was anastomosed to a new aperture made on the recipient inferior vena cava. After POD 50, the flow velocity in the hepatic vein gradually decreased with a loss of pulsatility in the Doppler waveform, followed by a decrease in portal blood flow volume and an increase in hepatic arterial flow velocity (Fig. 1). Liver function parameters gradually deteriorated and arterial ketone body ratio (AKBR), which reflects the liver's functional reserve [2], became critical – less than 0.7 – on POD 92. The patient presented with massive ascites and intermittent gastrointestinal bleeding. We assumed that the hepatic venous outflow disturbance had caused portal hypertension, leading to a decreased portal blood flow and ascites. Percutaneous transhepatic balloon dilation was performed on POD 95. Just before angioplasty, the flow velocity in the hepatic vein was 10 cm/s with a flattened waveform; portal blood flow volume had decreased to approximately 5 ml/min per kilogram body weight. Hepatic venography prior to angioplasty revealed a severe stenosis of the hepatic vein anastomosis (Fig. 2). On manometry, free hepatic vein pressure was 16 mm Hg.

Balloon dilation resulted in relief of the anastomotic stenosis of the hepatic vein and a decrease in free hepatic venous pressure to 4 mm Hg. Doppler examinations showed that the velocity in the

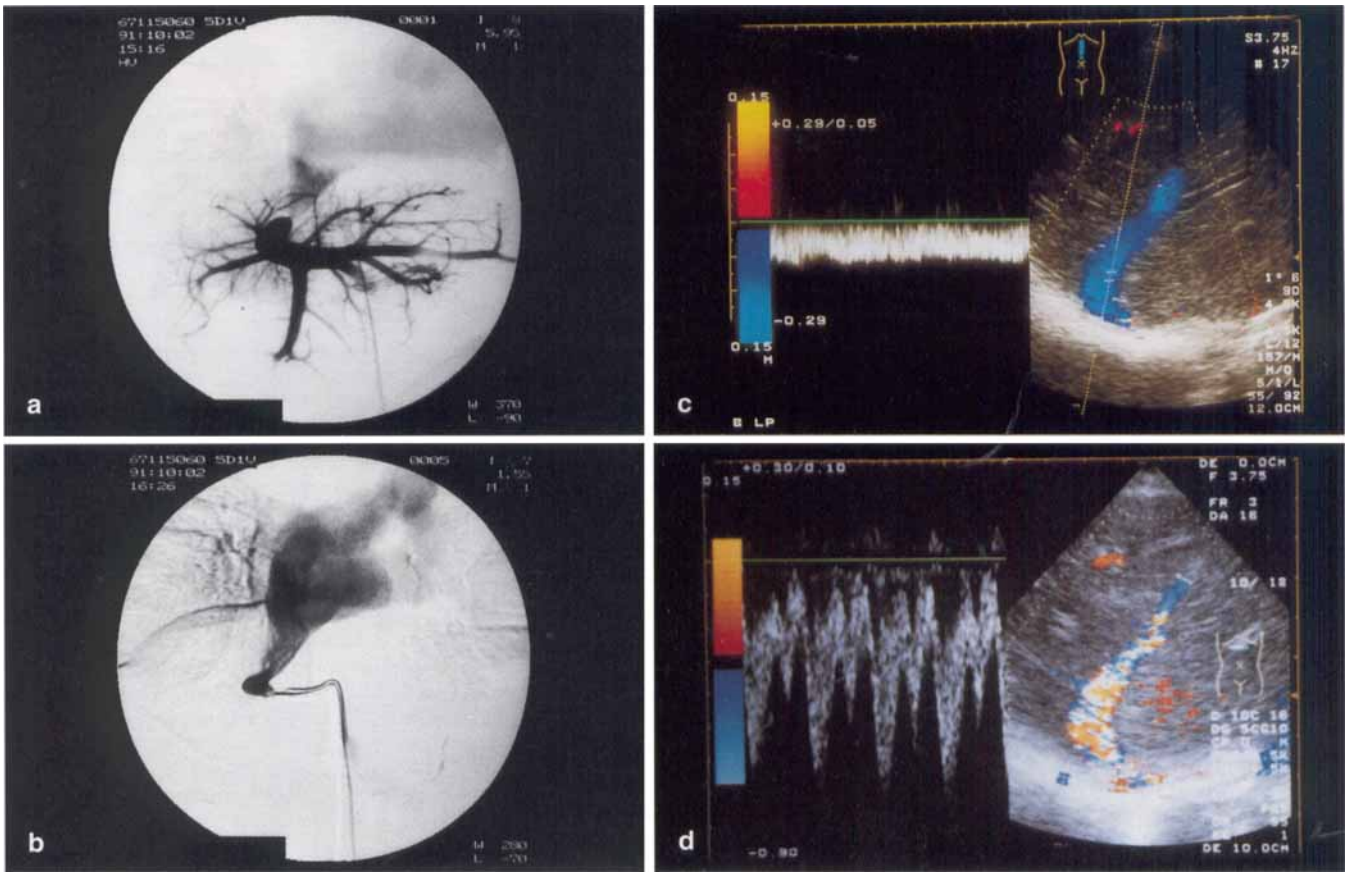

Fig. 2 a–d Radiographs of angioplasty for hepatic vein stenosis and Doppler US findings of the hepatic vein in case 1: **a** Hepatic venography shows severe stenosis of the hepatic vein anastomosis before angioplasty. The flow of contrast medium toward the right atrium is delayed and peripheral branches of the hepatic vein are enhanced; **b** PTA relieves the anastomotic stenosis and flow of contrast medium toward the right atrium is clearly obtained; **c** Before PTA, the flow velocity in the hepatic vein is decreased with a loss of pulsatility in the Doppler waveform; **d** Doppler spectrum of the hepatic vein after PTA shows a biphasic waveform pattern with a remarkable increase in the flow velocity

hepatic vein increased (V_{max} = 40 cm/s) and that the waveform became pulsatile just after the angioplasty (Fig. 2). At the same time, the portal blood flow increased remarkably (Table 1). On the following day, ascites and melena disappeared and liver function tests improved. However, blood flow in the hepatic and portal veins began to decrease again 3.5 months later. Clinical symptoms, such as ascites, also developed gradually. PTA for anastomotic stenosis of the hepatic vein was performed again on POD 252, and this resulted in recovery of graft perfusion and improved clinical symptoms. We were able to predict the recurrence of hepatic vein stenosis by follow-up monitoring of graft circulation with Doppler US (Fig. 1). Thirty-one months after the second PTA, this patient presents with no clinical problems.

Case 2

An 11-month-old female with biliary atresia underwent partial liver transplantation using the lateral segment of the left hepatic lobe from her mother. The left hepatic vein of the graft was anastomosed to the confluence of the middle and left hepatic veins of the recipient in an end-to-end fashion. Arterial reconstruction was performed by end-to-end anastomosis of the left hepatic arteries of the graft and recipient. Vascular anastomoses were judged to be adequate by intraoperative Doppler US studies. On POD 1 we could not detect any arterial flow signals from the common hepatic artery to the entire liver on color Doppler imaging, and the AKBR had decreased to 0.7. This was diagnosed as hepatic artery thrombosis and confirmed by repeated studies. Emergency revision was performed and the graft artery was successfully anastomosed to the common hepatic artery after complete thrombectomy. Thereafter, the patient was clinically well for a while. Doppler US studies showed that flow velocity in the hepatic vein was 18 cm/s with a mild decrease in pulsatility of the velocity waveform on POD 57. After that, the velocity in the hepatic vein gradually decreased and the Doppler waveform became flattened. After POD 70, portal blood flow decreased progressively and its Doppler waveform finally showed a "to-and-fro" pattern on POD 102, which indicated the existence of a hepatofugal blood flow through the portal trunk without an effective portal blood flow toward the graft. The maximum flow velocity in the hepatic vein had decreased remarkably to less than 5 cm/s on POD 104. Intrahepatic arterial color flow images became prominent with peak systolic velocity above 70 cm/s, and portal collateral pathways developed from the splenic hilum toward the abdominal wall on Doppler

Table 1 Changes in hepatic blood flow and clinical data after percutaneous transluminal angioplasty (PTA). (*GOT* glutamic oxalacetic transaminase, *AKBR* arterial ketone body ratio)

		Case 1				Case 2		Case 3			
		Before 1st PTA[a]	After 1st PTA	Before 2nd PTA[a]	After 2nd PTA	Before PTA[a]	After PTA	Before 1st PTA[a]	After 1st PTA	Before 2nd PTA[b]	After 2nd PTA
Hepatic vein	Flow velocity (cm/s)	9	40	10	30	3	27	13	24	10	35
	Waveform	Flat	Pulsatile	Flat	Pulsatile	Flat	Pulsatile	Flat	Pulsatile	Pulsatile	Pulsatile
Portal blood flow volume (ml/min per kg body weight)		5	30	6	23	To-and-fro	18	5	8	4	21
GOT (U/l)[c]		184	42	192	40	168	32	311	45	200	36
Total bilirubin (mg/dl)		2.4	1.0	2.5	0.9	1.6	1.0	1.7	1.0	1.4	0.9
AKBR		0.62	0.95	0.58	1.02	0.56	1.12	0.45	0.85	0.68	1.23

[a] PTA for hepatic vein stenosis, [b] PTA for portal vein stenosis, [c] The values of GOT, total bilirubin, and AKBR after PTA are those measured at 2 weeks after PTA

US. Liver function tests also became worse. Clinically, ascites and leg edema developed progressively around POD 85, showing a poor response to diuretics. No signs of congestive heart failure were observed in cardiac US or chest X-ray photos. A hepatic venous outflow disturbance was considered to be the cause of the decreased portal blood flow, leading to graft failure.

We performed PTA to treat the hepatic venous outflow disturbance on POD 108. The pressure gradient between the hepatic vein and the right atrium decreased from 17 mm Hg to 2 mm Hg by dilating the stenotic lesions. Doppler US findings of both the hepatic and portal veins improved immediately after angioplasty (Table 1). On the following day, the hepatic arterial flow velocity gradually decreased to the normal level and portosystemic collaterals disappeared on Doppler US. Twenty-eight months later, the patient is clinically well.

Case 3

A 23-month-old girl with biliary atresia received the lateral segment of the left hepatic lobe from her mother. The left hepatic vein of the graft liver was anastomosed end-to-end to the recipient left hepatic vein. Portal vein reconstruction was performed in an end-to-end fashion between the recipient portal trunk and left portal vein of the graft liver. On POD 1, blood pressure decreased to less than 60 mm Hg with progressive development of anemia. Blood pressure was maintained by transfusion, but the hemoglobin value did not increase to more than 6.5 g/dl despite the blood transfusion. US examination showed fluid collection and a large hematoma near the cutting surface of the graft. Emergency laparotomy confirmed bleeding from a small artery in the hepatoduodenal ligament of the graft, and ligation of the vessel, as well as removal of the hematoma, were performed. At that time, there was no bleeding from the arterial anastomosis. On POD 3, this patient had an episode similar to the one seen on POD 1, and laparotomy was performed to control the bleeding. Thereafter, intraperitoneal bleeding never occurred. No abnormal findings were obtained in Doppler US studies or laboratory data on POD 61. However, the blood flow in both the portal and hepatic veins decreased concomitantly after POD 80. On POD 112, Doppler US showed that the portal blood flow had decreased to 5 ml/min per kilogram body weight and was associated with a hepatofugal blood flow of the splenic vein. Collateral pathways from the splenic hilum to the retroperitoneum were also detected. Ascites was observed and liver function parameters had, by that time, also deteriorated. Judging from our earlier experiences, a hepatic vein stenosis seemed possible, and PTA was attempted on POD 123. Hepatic venography prior to the angioplasty confirmed anastomotic stenosis of the hepatic vein; however, it was not as severe as that seen in the previous two cases (cases 1 and 2). Balloon dilation of the stenosis reduced the pressure gradient between the hepatic vein and the right atrium from 8 mm Hg to 1 mm Hg. Postoperative Doppler US demonstrated that the flow velocity in the hepatic vein had increased and that the velocity waveform had changed from flat to pulsatile (Table 1). The splenic vein began to flow hepatopetally. Ascites decreased and liver function tests improved transiently. However, portal blood flow did not increase as much (approximately 8 ml/min per kilogram body weight). Subsequently, in the outpatient department, the blood flow in both the portal and hepatic veins decreased again, although the Doppler waveform of the hepatic vein remained pulsatile. The caliber of the portal trunk gradually decreased, and reversed flow in both the left gastric vein and splenic vein was detected on color Doppler US. Clinically, ascites developed and liver function deteriorated, as before the hepatic vein angioplasty. Portal vein stenosis was suspected and the appropriate interventional procedures were performed on POD 213. Using a transhepatic approach, a 7-Fr catheter was inserted into the junction of the portal and splenic veins under fluoroscopic guidance, where the pressure was 20 mm Hg. Portography showed an approximately 3-cm-long stenosis in the portal vein anastomosis (Fig. 3). For technical reasons, the pressure gradient across the stenosis was not measured.

The stenosis was completely resolved by balloon dilation. Immediately after the procedure, the portal blood flow increased remarkably (Fig. 3) and the splenic vein flowed hepatopetally on Doppler studies. The portal pressure was reduced to 9 mm Hg. During the postoperative course, ascites disappeared and liver function parameters returned to normal. This patient has now been followed up for 14 months since PTA for portal vein stenosis. Portal blood flow appears to remain adequate on follow-up Doppler US. She presents no clinical symptoms and blood examinations are normal.

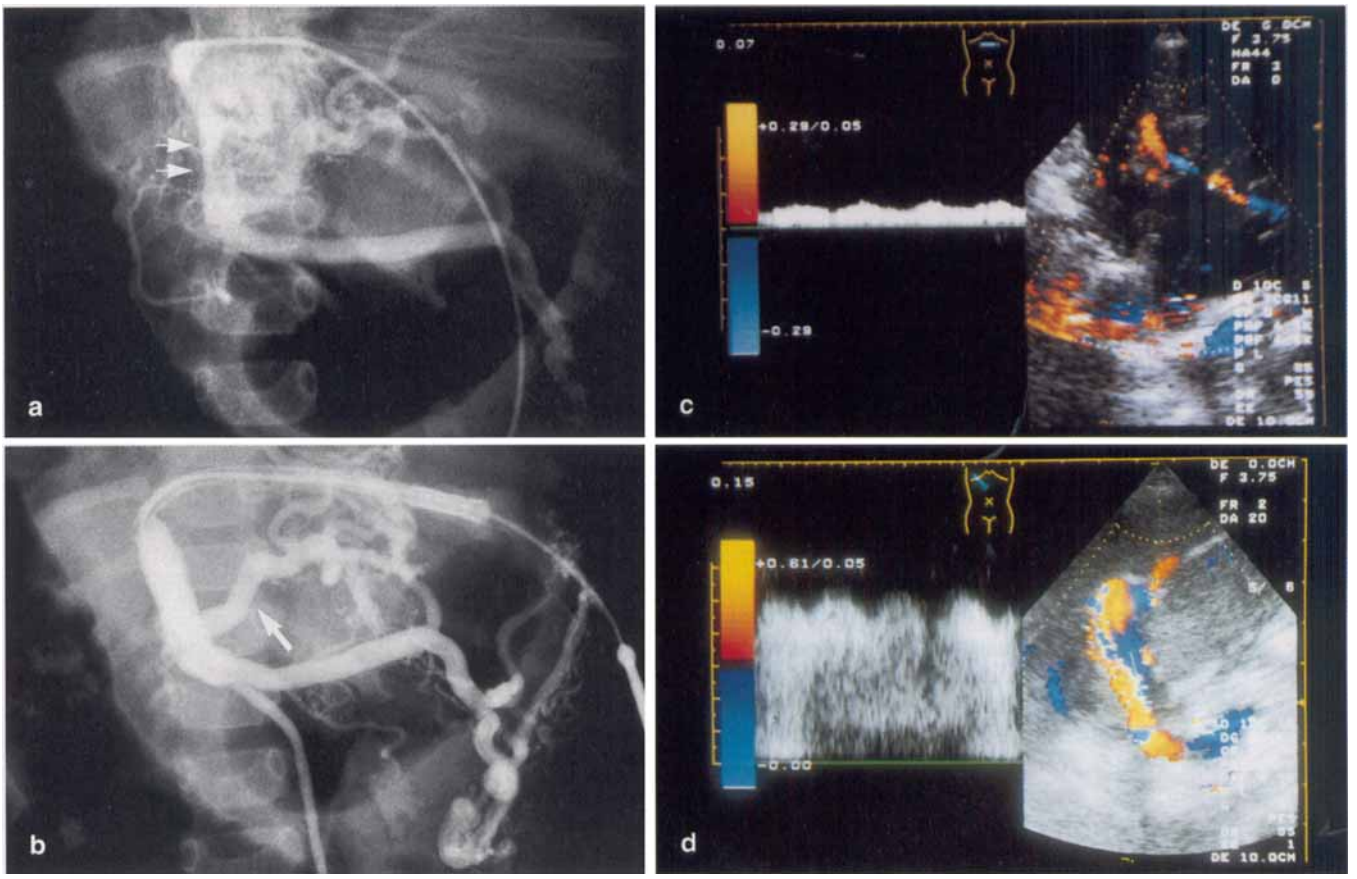

Fig. 3a–d. Radiographs of angioplasty for portal vein stenosis and Doppler US findings of the portal vein in case 3: **a** Transhepatic portography prior to PTA demonstrates a stenosis about 3 cm long in the portal vein anastomosis *(arrows);* **b** Subsequent portography reveals complete resolution of the stenosis. A dilated left gastric vein is also found *(arrow);* **c** Before PTA, the flow velocity in the intrahepatic portal vein is decreased. The sample volume is positioned at the umbilical portion; **d** After PTA, color flow images of the portal vein become prominent and the flow velocity increases significantly

Discussion

Stenosis of the hepatic or portal vein is not frequently seen but can be a devastating complication in pediatric liver transplantation. It may lead to portal hypertension and finally result in graft failure. In living related partial liver transplantation performed in our hospital, the inferior vena cava of the donor was maintained intact to minimize surgical invasiveness for the living donor, and hepatic vein reconstruction was often performed in an end-to-end fashion in the left hepatic veins of the recipient and the donor [8]. This type of reconstruction can be achieved by side-clamping the recipient left hepatic vein to maintain vena cava blood flow, but it may be prone to kinking or compression, compared with end-to-side implantation of the left hepatic vein onto the recipient vena cava described by Ringe et al. [10] or Broelsch et al. [3]. In order to prevent such complications, we made the extrahepatic segment of the hepatic vein as short as sound anastomosis would allow. Furthermore, as Broelsch et al. proposed a wide anastomosis [3], we recently created a common anastomotic orifice by incising the septum between the middle and left hepatic vein of the recipient, and we added an incision in the inferior vena cava wall, as needed, to widen the orifice according to the size of the graft hepatic vein.

Stenosis of the hepatic vein anastomosis can come about in several ways. Aside from technical failure, twisting or stretching of the vessel due to the inappropriate positioning of the graft may cause stenosis. In all of our 82 cases, Doppler US studies were performed before abdominal closure to decide graft positioning so as to obtain adequate graft perfusion. At that time, we also checked the hepatic venous blood flow carefully. No abnormal Doppler findings were obtained just after abdominal closure in the three cases presented. Shrinkage of the anastomotic scar might have been one cause of the delayed stenosis of the hepatic vein anastomosis in our patients since the vein narrowed sharply and smoothly at the stenotic portion on the venogram. Yet,

the stenotic lesion of the portal vein was about 3 cm long in case 3. Such a long stenotic lesion would seem different from simple anastomotic scarring. A decreased portal blood flow due to anastomotic scarring might cause thrombotic changes in that site. In this patient, stenosis of both the hepatic and portal veins complicated matters, and portal blood flow did not significantly increase after hepatic vein angioplasty alone, even though Doppler US findings of the hepatic vein had improved. Clinical findings improved temporarily, but portal hypertension remained. Overall, portal vein angioplasty brought about a remarkable recovery of graft perfusion. Since both hepatic venous outflow obstruction and portal vein stenosis can finally diminish the blood flow in the portal and hepatic veins. Doppler US examinations of those vessels are considered useful in detecting the primary cause of the disturbance in graft perfusion.

Using Doppler US, we can assess blood flow under physiological conditions noninvasively. In our cases, findings obtained with this method correlated well to those of angiography, which is the "gold standard" for diagnosing vascular complications [4]. Therefore, we feel that careful follow-up study with Doppler US can provide important information on these postoperative complications. Under free respiratory conditions, the Doppler waveform of the normal hepatic vein shows a triphasic pattern, consisting of one small retrograde wave during atrial systole and two antegrade waves during ventricular systole and atrial diastole [1, 14]. This pattern in the hepatic venous waveform is the final consequence of changes in right atrial pressure. When the blood flow from the hepatic vein to the right atrium is disturbed, the waveform of the hepatic vein decreases in pulsatility, changing to a flat pattern. In our series of 82 liver transplant recipients, the triphasic waveform of the reconstructed hepatic vein was found in only 6 cases after surgery. A biphasic waveform pattern, which lacks the short phase of reversed flow during atrial systole, was seen in 65 cases, and 11 cases showed a nearly flattened waveform without decreased flow velocity in the hepatic vein. Thus, the normal waveform is often not seen after surgery in the hepatic vein. This does not always indicate a pathological situation requiring treatment because the hepatic vein is reconstructed in an end-to-end fashion and is easily compressible. With our patients, during the first 2 weeks after transplantation, the pulsatility of the waveform and the flow velocity in the hepatic vein decreased slightly, but the velocity was usually maintained at more than 20 cm/s and the pulsatile waveform did not flatten out in any of the cases. However, when hepatic vein stenosis develops progressively, hepatic circulation is disturbed and Budd-Chiari syndrome develops. Serial Doppler US studies provide information regarding hemodynamic changes during hepatic venous outflow obstruction. Initially, the hepatic vein shows a nonpulsatile velocity waveform associated with a decrease in flow velocity. Then, the portal blood flow gradually decreases and the hepatic arterial flow increases reciprocally. When the Doppler spectrogram of the portal vein shows a "to-and-fro" pattern, the graft liver is fed by hepatic arterial flow alone. Clinical symptoms due to portal hypertension, such as ascites or gastrointestinal bleeding, become manifest. Laboratory data also indicate liver dysfunction in this condition, and treatment is required. After PTA, therapeutic effects must be confirmed repeatedly and noninvasively with Doppler US, and the recurrence of the vascular complications can be predicted, as shown in case 1.

PTA has recently been used to treat membraneous webs of the hepatic veins or the hepatic segment of the inferior vena cava in Budd-Chiari syndrome [5, 6]. We consider this method an effective alternative to surgical reconstruction for anastomotic stenosis of the hepatic or portal vein in the late phase after transplantation. Prior to angioplasty, angiographic studies are required to confirm the exact location of the stenotic region. PTA for portal vein stenosis can usually be carried out using a transhepatic approach. However, there are three ways to perform the hepatic vein angioplasty; transjugularly, transfemorally, and transhepatically. We selected the transhepatic approach because we felt this approach enabled us to insert the guide wire across the severe stenosis of the hepatic vein anastomosis with a fair degree of certainty. The hepatic vein can be accurately punctured under US guidance, but we should avoid inadvertent puncture of other intrahepatic vessels. Color Doppler US examinations prior to the puncture are useful in avoiding such a complication because they provide more information about the blood flow in the intrahepatic vessels than conventional B-mode does. Since the graft liver is usually located in the epigastric region in pediatric partial liver transplantation, we have easier access to the hepatic vein by going through the epigastrium, without disturbing the intestinal tract located near the cutting surface of the graft, than with an approach via the right intercostal space. Postoperative bleeding may be a possible complication in transhepatic angioplasty, though we have never experienced it. The graft liver is thought to adhere tightly to the abdominal wall in the epigastric region, and so we think hemostasis can be performed only by manual compression unless severe coagulopathy exists. In our series, we did not use gelatin sponge pledgets for embolization of the tract on removal of the sheath in order to avoid the risk of thrombosis due to the inadvertent release of thrombogenic material into the treated vein. In fact, no postoperative bleeding was ever clinically manifested.

Transjugular intrahepatic portosystemic shunt (TIPS) has recently been performed in the treatment of

recurrent variceal bleeding or refractory ascites in chronic liver disease [9, 11]. This technique can decompress portal hypertension with a relatively low rate of procedure-related mortality, rebleeding, or hepatic encephalopathy [11]. However, we did not consider stenosis of the portal or hepatic vein in our cases as a good indication for TIPS because this intervention decreases the effective portal blood flow and may cause liver function to deteriorate in the long run. TIPS for hepatic vein stenosis may reverse portal blood flow and increase the risk of variceal bleeding without correcting the hepatic venous outflow disturbance. For portal vein stenosis as seen in case 3, we can hardly expect effective decompression of portal hypertension by TIPS since the portal inflow remains disturbed. We firmly believe that balloon angioplasty is the treatment of choice for such venous complications.

In conclusion, percutaneous transluminal angioplasty is an effective method of treating hepatic and portal vein stenosis following pediatric liver transplantation from living related donors. Moreover, Doppler US is a useful technique not only for diagnosing abnormal graft circulation, but also for monitoring the therapeu-

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