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Interventional Therapeutic Techniques in Budd-Chiari Syndrome

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Abstract
Purpose: To analyze the results obtained with percutaneous therapeutic procedures in patients with Budd-Chiari syndrome (BCHS).
Methods: Between August 1991 and April 1993, seven patients with BCHS were treated in our hospital. Three presented with a congenital web; in another three cases the hepatic veins and/or the inferior vena cava (IVC) were compromised after major hepatic surgery; one patient presented with a severe stenosis of the intrahepatic IVC due to hepatomegaly.
Results: One of the patients with congenital web has required several new dilatations due to restenosis; one patient required a transjugular intrahepatic portosystemic shunt procedure while awaiting a liver transplantation. The two postsurgical patients with stenosed hepatic veins did not require any new procedure after the placement of metallic endoprostheses. However, the patient with liver transplantation presented IVC restenosis after balloon angioplasty that required the deployment of metallic endoprostheses. In the patient with hepatomegaly a self-expandable prosthesis was placed in the intrahepatic portion of the IVC before (4 months) a liver transplantation.
Conclusion: Interventional therapeutic techniques offer a wide variety of possibilities for the treatment of patients with BCHS. For IVC stenoses, the results obtained with balloon angioplasty are at least as good as those obtained with surgery.

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Key words: Budd-Chiari syndrome—Stents and prostheses—Transluminal angioplasty—Venae cavae, stenosis, or obstruction

Budd-Chiari syndrome (BCHS) is caused by obstruction of the hepatic venous flow and characterized by the presence of ascites, hepatomegaly, and abdominal pain. Clinically indistinguishable from hepatic veno-occlusive disease, in which progressive occlusion of the small postsinusoidal hepatic venules occurs, BCHS is secondary to an obstruction of the major hepatic veins or even the inferior vena cava (IVC) [1].

Obstruction in BCHS can be secondary to multiple situations: coagulation abnormalities (polycythemia vera, paroxysmal nocturnal hemoglobinuria, etc.), surgery [orthotopic liver transplantation (OLT), high-risk hepatic surgery, etc.), tumor extension, or congenital conditions (obstructing membranes, stenoses, coarctations, etc.) [2].

Different surgical procedures have been described to improve the decompression of the portal system when medical treatment becomes ineffective: mesocaval or mesoatrial shunts, or membranotomy of the congenital web [3, 4]. Recently, OLT has been advocated as the ideal treatment in patients with end-stage liver function and BCHS.

In recent years several percutaneous techniques have been reported for treating BCHS including angioplasty, fibrinolysis, stenting with endoprostheses, transjugular intrahepatic portosystemic shunt (TIPS). The long-term reported results with such techniques are at least as good as those obtained with surgery [5–7].

Table 1. Etiology, characteristics of the lesion, technique, and results of the first procedure

Patients (no./sex/ age, years)	Localization of the stenosis	Etiology	Approach	Gradient pre-procedure (mmHg)	Balloon diameter (mm)	Prosthesis diameter (mm)	Gradient post-procedure (mmHg)	Associated procedure	Follow-up (months)
1/F/47	IVC	Web type III	Femoral	18[a]	23	—	7[a]	—	5[c] 37[d]
2/M/26	IVC	Web type Ia	Femoral; jugular	25[a]	30	—	5[a]	—	38
3/F/45	IVC RHV	Web type Ib	Femoral; transhepatic	36[b] 16[a]	8 + 20	—	1[b] 1[a]	—	49
4/M/32	IVC	Post-OLT	Femoral	13[a]	30	—	3[a]	Dilatation of the portal vein	33
5/F/57	RHV	Postsurgery	Jugular	19[b]	10	9	4[b]	Hepatic abscess drainage	23[e]
6/M/38	RHV	Postsurgery	Transhepatic	16[b]	8	9	2[b]	Biliary recanalization; abscess drainage	45
7/F/31	IVC	Hepatic vein thrombosis; hepatomegaly	Femoral	NA	—	16	NA	—	1[c] 42[d]

IVC = inferior vena cava; RHV = right hepatic vein; NA = not available
[a] Gradient between the IVC and the right atrium
[b] Gradient between the hepatic vein and the right atrium
[c] Time between dilatation and liver transplantation
[d] Follow-up after liver transplantation
[e] Dead

This paper describes different percutaneous procedures in seven patients with BCHS secondary to congenital membranous obstructions, surgery, and extrinsic compression of the IVC.

Materials and Methods

Between August 1991 and April 1993, seven patients with BCHS were treated in our hospital. Four were female and three were male, and their mean age was 39.4 years (range 26–57 years). Obstruction of hepatic venous outflow was secondary to a web or coarctation in three patients, complications after major hepatic surgery in two cases, IVC narrowing after OLT in one patient, and IVC compression due to hypertrophy of the caudate lobe in another patient (Table 1).

Web or Coarctation

According to Hirooka and Kimura [8] the stenoses in these three patients were classified as Ia, Ib, and III. All patients presented with moderate to severe hepatomegaly. Liver function was partially preserved in patient 2, moderately impaired in patient 3, and markedly abnormal in patient 1.

The vascular approach used for therapeutic purposes varied according to the cause and location of the stenoses. IVC stenosis in patient 1 was dilated with an 18-mm-diameter valvuloplasty balloon catheter (William Cook Europe, Bjaeverskov, Denmark) via a transfemoral approach (Fig. 1A, B).

In patient 2, the complete membranous obstruction of the IVC was traversed, from a transjugular approach, with the heavy tip of an Amplatz super-stiff guidewire (Meditech, Boston Scientific, Watertown, MA, USA) (Fig. 2A). Once the wire had crossed the web, a 5 Fr straight catheter was advanced and then the super-stiff wire was exchanged for a 260-cm-long, 0.035-inch J-guidewire (Bard, Galway, Ireland). Using a femoral approach, the J-wire was grasped and extracted with a wire loop. The web was finally dilated from the transfemoral approach with a 25-mm balloon angioplasty catheter (Balt, Montmorency, France) (Fig. 2B).

Patient 3, who presented with severe narrowing of the IVC that was also compromising the outlet of the right hepatic vein (RHV) [the only one detected at ultrasound (US)], was treated using a double approach (transfemoral and transhepatic). Direct transhepatic catheterization of the RHV was performed under US guidance and the stenosis was traversed with a 0.035-inch hydrophilic guidewire (Terumo, Tokyo, Japan). Once the guidewire was in the right atrium (RA), a 5 Fr, 8-mm balloon angioplasty catheter (Schneider, Bülach, Switzerland) was placed in the hepatic vein stenosis. By using a transfemoral approach, a 25-mm balloon angioplasty catheter (Balt) was placed in the stenosis of the IVC. The two balloons were simultaneously inflated and the waists disappeared in a few seconds.

IVC Narrowing After OLT

Patient 4 was a 32-year-old man referred to our hospital with acute liver failure due to complete thrombosis of the hepatic veins related to the presence of α1-antitrypsin deficiency. An emergent OLT was performed but the graft was rejected. A new liver was placed but rejected again. Finally, a third graft was placed 22 months after his first admission.

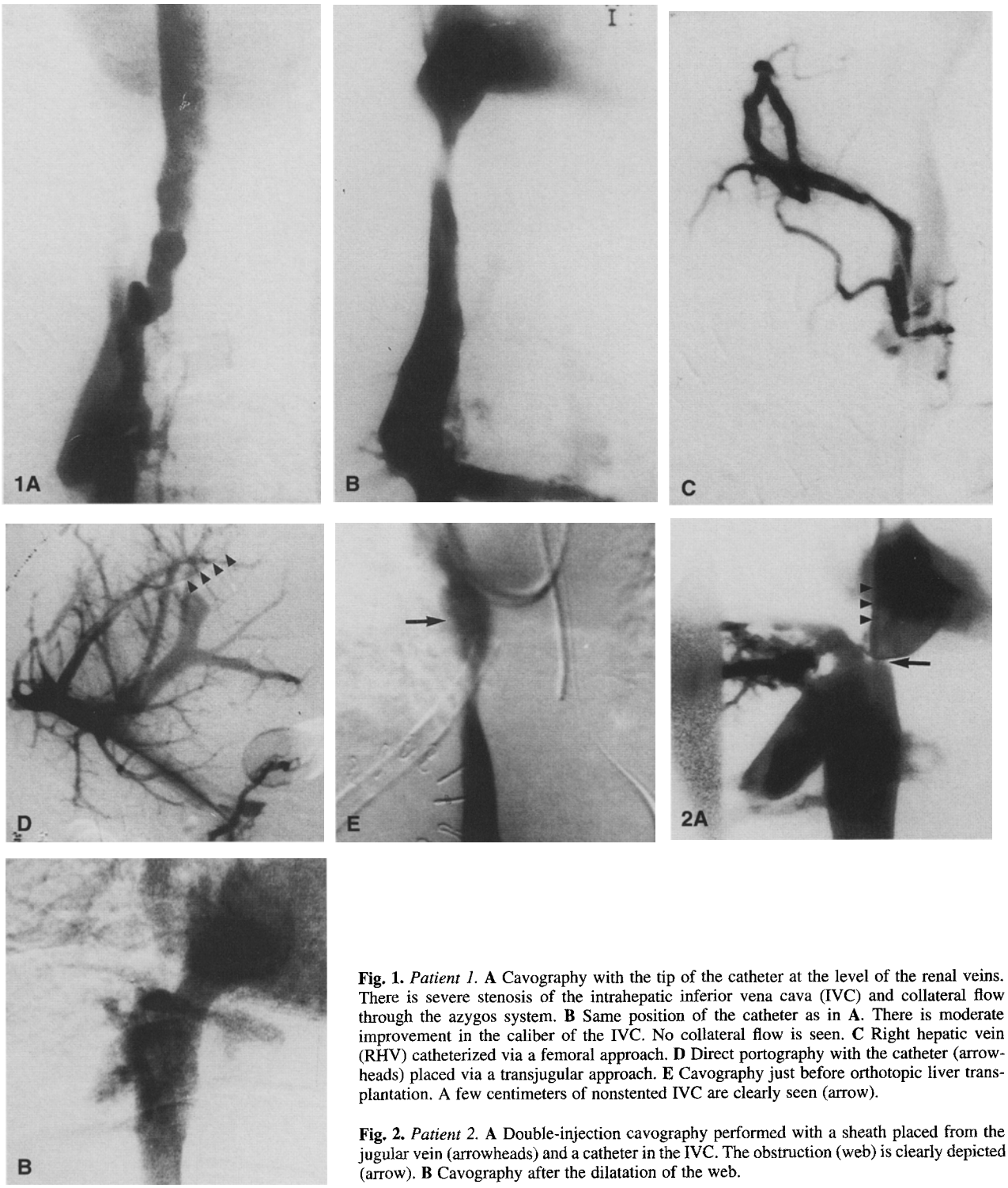


Fig. 1. *Patient 1.* **A** Cavography with the tip of the catheter at the level of the renal veins. There is severe stenosis of the intrahepatic inferior vena cava (IVC) and collateral flow through the azygos system. **B** Same position of the catheter as in **A**. There is moderate improvement in the caliber of the IVC. No collateral flow is seen. **C** Right hepatic vein (RHV) catheterized via a femoral approach. **D** Direct portography with the catheter (arrowheads) placed via a transjugular approach. **E** Cavography just before orthotopic liver transplantation. A few centimeters of nonstented IVC are clearly seen (arrow).

Fig. 2. *Patient 2.* **A** Double-injection cavography performed with a sheath placed from the jugular vein (arrowheads) and a catheter in the IVC. The obstruction (web) is clearly depicted (arrow). **B** Cavography after the dilatation of the web.

The patient remained asymptomatic for 5 months but liver function parameters deteriorated again and a moderate hepatomegaly was detected. Color Doppler US showed marked narrowing in the juxtacardial IVC as well as in the surgical connection of the portal vein (PV) (Fig. 3A, B). The stenosis of the IVC was dilated via a transfemoral approach with a 30-mm-diameter valvuloplasty balloon catheter (Schneider) (Fig. 3C, D). During the same procedure, the PV was transhepatically catheterized and subsequently dilated with a 15-mm-diameter balloon catheter (Balt).

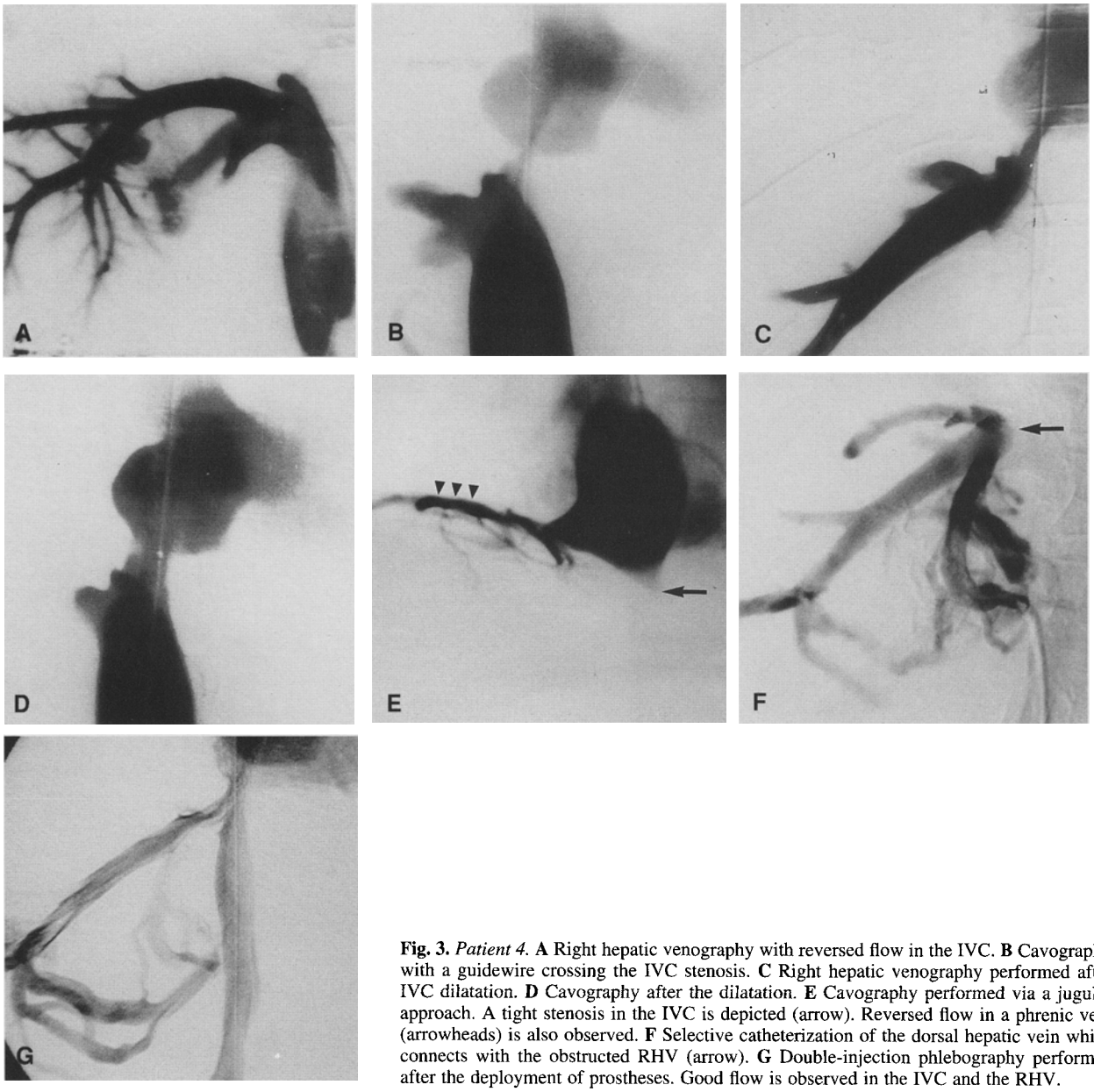


Fig. 3. *Patient 4.* **A** Right hepatic venography with reversed flow in the IVC. **B** Cavography with a guidewire crossing the IVC stenosis. **C** Right hepatic venography performed after IVC dilatation. **D** Cavography after the dilatation. **E** Cavography performed via a jugular approach. A tight stenosis in the IVC is depicted (arrow). Reversed flow in a phrenic vein (arrowheads) is also observed. **F** Selective catheterization of the dorsal hepatic vein which connects with the obstructed RHV (arrow). **G** Double-injection phlebography performed after the deployment of prostheses. Good flow is observed in the IVC and the RHV.

Venous Stenoses After Hepatic Surgery

Patient 5 presented a past history of hepatic hydatid cyst which had required surgery 12 years previously. The surgical procedure consisted of partial extraction of the cyst and connection of the remaining cavity with the main bile ducts. Although there was an initial improvement, in the last 5 years she suffered repeated episodes of cholangitis with progressive splenomegaly and portal hypertension. At the time of the study, CT and US demonstrated the presence of an intrahepatic fluid collection adjacent to the RHV. An hepatic venogram, from a transfemoral approach, showed a marked venous stenosis which provoked a gradient of 19 mmHg with the RA (Fig. 4A). The stenosis was dilated with a 10-mm-diameter balloon catheter (Cordis, Roden, The Netherlands) but immediately relapsed. A Wallstent prosthesis (9 mm wide; Schneider) was then placed, re-establishing an adequate venous caliber, and gradients normalized.

Patient 6 suffered an acute occlusion of the RHV after a major hepatectomy consisting of an atypical trisegmentectomy to remove two huge hydatid cysts. The hepatic vein was transhepatically punctured under US guidance. A venogram confirmed the presence of a segmentary obstruction of the vein with a wide net of intrahepatic collaterals. The obstruction was traversed with a hydrophilic guidewire (Terumo) and dilated with an 8-mm-diameter balloon catheter (Schneider); angiographic results obtained after angioplasty were poor. Two 9-mm-wide Wallstent prostheses (Schneider) were then placed, achieving good flow as well as normalization of the pressure gradients.

IVC Extrinsic Compression Due to Hepatomegaly

Patient 7 was a 31-year-old woman with end-stage liver insufficiency that required OLT due to the presence of massive regenerative liver

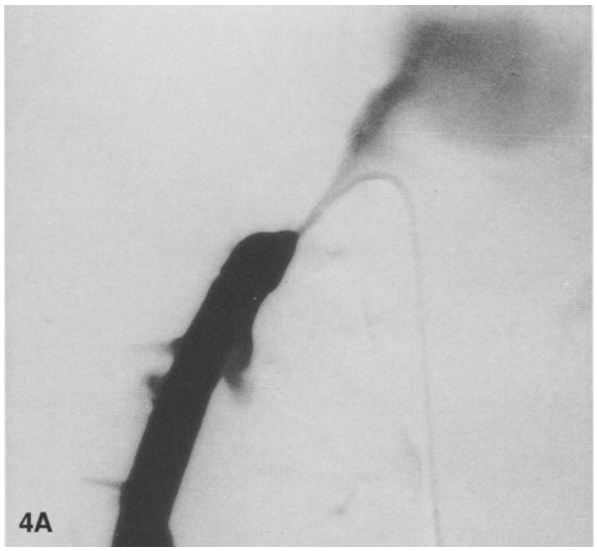
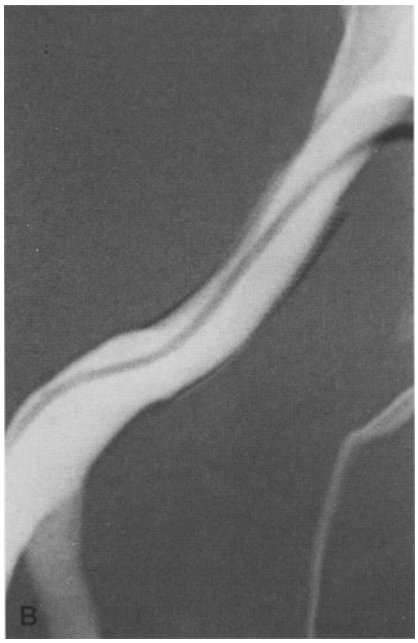
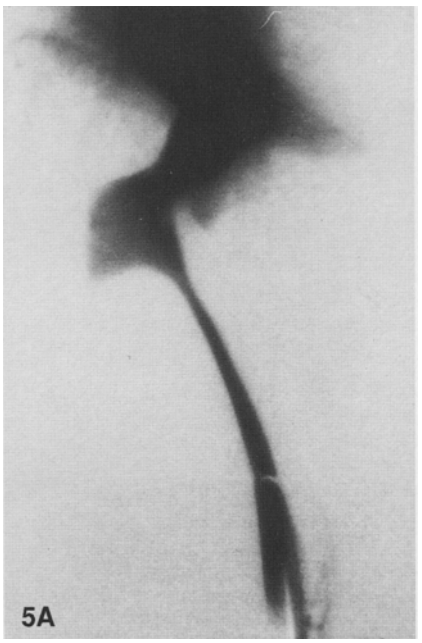
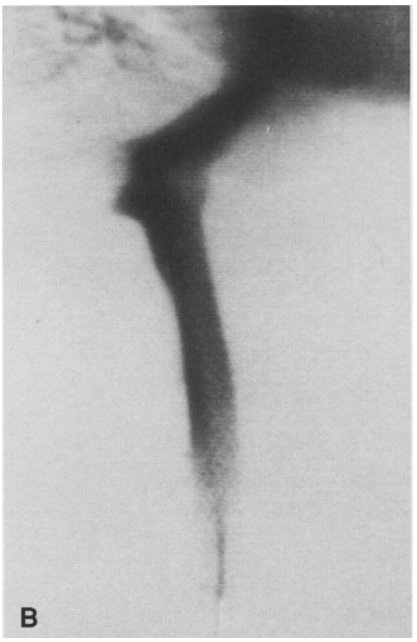


Fig. 4. *Patient 5.* **A** Phlebography of the RHV that shows a tight stenosis. **B** Follow-up phlebography performed 20 months after the first procedure.

Fig. 5. *Patient 7.* **A** Severe stenosis of the intrahepatic IVC. **B** Good caliber of the IVC after the placement of the prosthesis.







nodular hyperplasia. Hepatomegaly caused severe narrowing of the intrahepatic IVC (Fig. 5A) which provoked renal insufficiency and lower extremity edema. While the patient was awaiting liver transplantation, the IVC was stented with a 16-mm-wide Wallstent prosthesis (Schneider) carefully placed between the outlet of the main hepatic veins and the renal veins (Fig. 5B), with restoration of the normal flow parameters at Doppler US studies.

Results

All patients presented marked improvement in their clinical status after the recanalization, dilatation, or stenting of the venous stenoses. One patient (no. 5) died 23 months after the procedure due to repetitive episodes of cholangitis and progressive liver failure. The other patients are in good clinical condition with a mean follow-up of 42.1 months (range 33–51 months). No major complications appeared during or after the procedure, but some patients required new interventions during their follow-up due to restenoses or the development of new vascular and nonvascular complications (Table 2).

Web or Coarctation

A significant decrease in the venous gradient was obtained after the recanalization, dilatation, or stenting of

Table 2. Therapeutic procedures performed during follow-up

Patient	Procedure	Length of follow-up (months)	Approach	Gradient pre-procedure (mmHg)	Balloon diameter (mm)	Prosthesis diameter (mm)	Gradient postprocedure (mmHg)	Associated procedure
1/F	TIPS	4	Jugular	31[a]	8	9	17[a]	—
2/M	Dilatation	3	Femoral	11[b]	30	—	5[b]	—
	Dilatation	7	Femoral	8[b]	28	—	3[b]	—
	Dilatation	14	Femoral	12[b]	28	—	3[b]	—
	Dilatation	27	Femoral	11[b]	28	—	3[b]	—
4/M	Dilatation	5	Jugular	10[b]	10 + 10 30	—	1[b]	—
	Stenting	20	Jugular; femoral	15[b]	25	9 + 16	4[b]	PTA of the hepatic artery

TIPS = transjugular intrahepatic portosystemic shunt; PTA = percutaneous transluminal angioplasty
[a] Portosystemic gradient
[b] Gradient between the IVC and the right atrium

the venous stenoses in the three patients. The mean gradient before the procedure was 26.3 mmHg, which decreased to 4.3 mmHg.

The greatest decrease was obtained in patient 3, with a drop of 35 mmHg between pressures measured in the RHV and the RA. In this patient, the gradient between the IVC and the RA also decreased from 16 to 1 mmHg. Follow-up cavograms at 3, 12, and 19 months revealed a wide and patent IVC with maintained gradients between the IVC and the RA of 4–5 mmHg. The patient currently remains asymptomatic and follow-up Doppler US studies reveal good flow in the IVC and the RHV.

Patient 2 showed a marked improvement in his clinical status after the recanalization and dilatation of the obstruction. Unfortunately, Doppler US at follow-up revealed a decrease in flow parameters that required angiographic studies with subsequent redilatation at 3, 7, 14, and 27 months. The gradient between the IVC and the RA increased to 10.5 mmHg (mean values), with a decrease to 3.5 mmHg after new dilatations. The patient now remains asymptomatic.

Renal function showed a moderate improvement after the dilatation of the IVC stenosis in patient 1. However, ascites was still present and hepatic function progressively deteriorated. OLT was deemed necessary and, while awaiting the graft, a TIPS procedure was performed with the aim of improving her clinical status. Due to the absence of hepatic veins (Fig. 1C), the right portal vein was punctured directly from the IVC (Fig. 1D). The portosystemic gradient was 30 mmHg which, after the placement of three Wallstent prostheses (Schneider), dropped to 17 mmHg (Fig. 1E). The patient underwent OLT 5 weeks later and the stent inside the IVC did not cause any major surgical problem. At 37 months after OLT, the patient remains clinically asymptomatic.

IVC Narrowing After OLT

Follow-up Doppler US studies have not detected any restenosis in the PV. However, 5 months after the initial dilatation, a critical restenosis was detected affecting the IVC and also the outlet of the RHV. Both stenoses were approached from a transjugular approach and simultaneously dilated with two 10-mm-diameter balloon angioplasty catheters (Cordis). The IVC was posteriorly dilated with a 30-mm-diameter valvuloplasty balloon catheter. Unfortunately, 15 months later a marked restenosis appeared in the IVC and the RHV. The IVC stenosis was easily traversed from a femoral approach but the RHV could not be catheterized from either the femoral or the jugular vein (Fig. 3E). Transfemoral catheterization of the dorsal hepatic vein showed a direct intrahepatic communication with the RHV (Fig. 3F); the guidewire was then easily placed in this vein and later in the RA, where it was placed and extracted from the jugular approach. Finally, a 9-mm-wide Wallstent prosthesis (Schneider) was placed (from the jugular vein) at the origin of the RHV and, simultaneously, a 16-mm-wide Wallstent prosthesis (Schneider) (from a femoral approach) in the stenosed IVC (Fig. 3G). In addition, the hepatic artery (connected directly to the aorta) showed a marked narrowing that was dilated with a 5-mm-diameter balloon angioplasty catheter (Schneider). Fourteen months later, the patient remains asymptomatic and Doppler US studies have not demonstrated any restenosis; there are normal flow parameters in the IVC, the RHV, the PV, and the hepatic artery.

Venous Stenosis After Hepatic Surgery

After the procedure, patient 5 showed mild clinical improvement and a significant increase in the platelet

count. On the other hand, cholangitis requiring medical treatment and intrahepatic collection requiring percutaneous drainage recurred. The patient died of liver failure. The gradient was always less than 5 mmHg and no hepatic vein restenosis was detected (Fig. 4B).

Patient 6 has markedly improved clinically. Color Doppler follow-up shows a wide open hepatic vein without any change in the venous flow pattern.

IVC Extrinsic Compression Due to Hepatomegaly

One month after stent placement the patient underwent an OLT. The prosthesis did not provoke any additional difficulty during the surgical procedure as it was completely within the intrahepatic portion of the IVC. Histological examination showed partial incorporation of the stent into the wall of the IVC with patency of the veins draining the caudate lobe. Fifty-one months after the stenting the patient remains asymptomatic.

Discussion

BCHS is a rare condition caused by obstruction of the hepatic veins and/or the IVC; a wide variety of etiologies has been reported which differ among countries. In the Far East, 30% of such patients present a membranous obstruction of the IVC caused by a web, probably congenital, in the outlet of the hepatic veins and/or the suprahepatic portion of the IVC. The classic treatment of these patients was a "membranotomy" performed with the surgical exposure of the IVC or the RA. The membranotomy could also be performed with cutting devices introduced surgically or through a femoral venotomy [4]. In the early 1980s, two reports from Yamada et al. [9] and Uflacker et al. [10] suggested the use of balloon angioplasty catheters to perform the same procedure (membranotomy) but via a percutaneous transvascular approach. Since then several series have been published reporting excellent results with balloon dilatation of the congenital obstructing webs of the IVC [5, 6].

In most cases, recanalization of the obstructed segment can be performed with wires or intravascular needles [6], and only in a very few patients are other devices, such as a laser, needed to recanalize the web [11]. In patients with long obstructed segments of the IVC, mechanical thrombectomy or fibrinolysis can be performed before the dilatation to avoid a pulmonary embolism. There is no general agreement concerning the use of only one balloon or two or even three simultaneously placed in the stenosis [9]; moreover, the balloon can either be inflated in the lesion or pulled through the stenosis once inflated near the web [12].

The reported success rate with PTA in congenital stenoses is as high as 81%; unfortunately, the primary permeability rate decreases to 50% in noncongenital IVC obstructions [12]. In patients with restenosis, a new balloon dilatation must be performed, but only in cases with a 75% reduction in the caval lumen or a gradient of at least 20 mmHg [13]. The number of redilatation procedures must be reduced as much as possible since angioplasty stimulates the hyperplasia of the intima, sometimes increasing the risk of restenosis. For such problematic patients, the use of metallic stents has been advocated by Yamada et al. [6], with the theoretical purpose of obtaining a stable diameter of the IVC. However, according to Park et al. [5], a hyperplastic neointima that grows around the wires reduces the diameter of the stenosed IVC, usually by 2–5 mm. In some cases even, a complete thrombosis of the stent has been reported. There are two additional drawbacks with the use of metallic stents. First, if the stent crosses the outlet of the hepatic vein, the continuous growth of the neointima and the increase in turbulent flow can lead to thrombosis of the vein, which would provoke an undesired worsening of the clinical situation. Second, if an OLT has to be carried out, the surgical technique would be markedly hindered by the presence of the metallic wires [2].

It is our opinion that metallic stents within the hepatic IVC should be placed in only very selected cases [14], as was done in patient 5, in whom a fourth OLT was deemed technically very difficult and the function of the original liver needed to be maintained at all costs. In this case, as recommended by Venbrux et al. [15], the stents were placed in both the IVC and the RHV with the aim of securing the venous outflow of the liver. Another possibility is to place the stent (patient 7) within the margins of the hepatectomy when the suprahepatic portion of the IVC is preserved.

Stenoses within the hepatic veins are a different problem. In such cases, after a failed percutaneous transluminal angioplasty, a stent can easily be placed via a transjugular, transfemoral, or transhepatic approach [16]. Unlike in TIPS, the stents rarely provoke intimal hyperplasia and an adequate caliber of the vein can be ensured [17].

In patients with a deteriorated liver function and symptomatic portal hypertension, a TIPS procedure can be performed, instead of surgical derivations, to improve the clinical condition while waiting for OLT. In a recent report, Blum et al. [7] demonstrated the usefulness of TIPS for the treatment of patients with subacute or chronic BCHS; in their series, 83% (10/12) of patients showed a marked improvement of the ascites. An additional difficulty for TIPS in BCHS is the ab-

sence or at least the distortion of the hepatic veins; in such cases, the prostheses can be placed directly from the IVC but ensuring enough nonstented venous lumen to allow the surgical technique.

In conclusion, interventional therapeutic techniques offer a wide range of possibilities for the treatment of patients with BCHS. When a congenital web is present, a percutaneous "membranotomy" can be performed with a result at least similar to that obtained with surgery. However, when restenoses appear, careful and individualized treatment is needed, and only in very selected cases should metallic endoprostheses be placed. Finally, TIPS allows an efficient portosystemic circulation in patients with BCHS who are waiting for liver transplantation.

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