New approach to dilation of stenotic lesions through the accessory hepatic vein in Budd-Chiari syndrome

Zhanguo Sun, MD, Jingjun Jiang, MD, Wei Li, PhD, Xuemin Zhang, MD, Xiaoming Zhang, MD, and **Changshun He, PhD,** *Beijing, China*

We investigated a new approach to dilation of stenotic lesions through the femoral vein-accessory hepatic vein-intrahepatic communicating branched vein-hepatic vein-inferior vena cava loop in two cases of Budd-Chiari syndrome. For some selected patients, this approach represents an additional method to increase technical success and to decrease complications. (J Vasc Surg Cases 2015;1:42-5.)

The most commonly used approaches for the endovascular treatment of Budd-Chiari syndrome (BCS) are percutaneous transhepatic, femoral vein-inferior vena cava (IVC)-hepatic vein (HV), and jugular vein-IVC-HV. We report an alternative approach through the femoral vein-accessory hepatic vein (AHV)-intrahepatic communicating branched vein-HV-IVC loop in two cases of BCS. Both patients provided consent to publish this case report.

CASE REPORT

Case 1. A 46-year-old woman complained of abdominal distention, shortness of breath after exercise, and a 15-year history of varicose superficial epigastric veins. Physical examination showed that the flow direction of the varicose superficial epigastric veins was cephalad. The liver was enlarged, with its lower edge 1 cm below the right costal margin. Ultrasound (US) examination of the abdomen revealed hepatomegaly and obstruction of the retrohepatic segment of the IVC. The blood flow was hepatopedal. No other imaging modalities were used. The preoperative diagnosis was BCS, and no concomitant diseases that may have been risk factors for or causes of the development of BCS were found. After puncture of the right femoral vein under local anesthesia, a pigtail catheter was placed in the IVC; angiography showed a long, obstructive lesion of the hepatic segment of the IVC, AHV compensatory enlargement, and dilation of the abundant intrahepatic communicating branched veins (Fig 1, *A*). Subsequent angiography with a pigtail catheter after catheterization of the retrohepatic segment of the IVC through the AHV-intrahepatic communicating branched vein-HV demonstrated severe stenosis of the suprahepatic IVC (Fig 1, *B*). The blood flow of the HV was static, and the pressure was 25 cm H_2O. A 0.035-inch angled hydrophilic guidewire was passed through the stenosis of the suprahepatic IVC, and the lesion was subjected to balloon angioplasty (diameters of 8 mm and 14 mm successively) (Fig 1, *C* and *D*). Repeated angiography showed 0% residual stenosis (Fig 1, *E*). The blood flow to the heart through the HV returned to normal (Fig 1, *F*), and the pressure in the HV decreased to 14 cm H_2O. After the procedure, the varicose superficial epigastric vein was no longer visualized, and the liver was shown to have decreased in size on abdominal US. Warfarin was used for long-term anticoagulation. The patient was observed with US examination at 3 months, 6 months, and 12 months after the operation and yearly thereafter. The US examination 3 months after the procedure showed a normal-size liver and patency of the suprahepatic IVC.

Case 2. A 28-year-old man complained of a 1-year history of abdominal distention. No concomitant diseases that may have been risk factors for or causes of the development of BCS were found. Varicose superficial veins were visible in the midsection, and the flow direction was cephalad. The liver was enlarged, with its lower edge 3 cm below the right costal margin. Physical examination revealed pitting edema of the lower extremities. Abdominal US revealed hepatomegaly and obstruction of both the HV and the retrohepatic segment of the IVC. The blood flow was hepatofugal. No other imaging modalities were used. Angiography after puncture of the right femoral vein under local anesthesia revealed severe stenosis of the IVC and stenosis of the opening of the AHV (Fig 2, *A*). Subsequent angiography after catheterization of the AHV with a JR4 catheter revealed dilation of the abundant intrahepatic communicating branched veins, stasis of the HV, and obstruction at the junction of the right HV and the IVC (Fig 2, *B*). The pressure in the HV was 27 cm H_2O. A 0.035-inch angled hydrophilic guidewire was passed through the occlusive segment of the HV to the suprahepatic IVC by the new approach under the support of a 55-cm-long 6F introducer sheath. The lesions of the AHV and the right HV were free from residual stenosis after balloon angioplasty (10 mm in diameter) (Fig 2, *C-E*). Angioplasty of the stenosed IVC was then performed with a 25-mm balloon, and the stenosis was subsequently resolved (Fig 2, *F*). The pressure

From the Department of Vascular Surgery, Peking University People's Hospital.

Author conflict of interest: none.

Reprint requests: Jingjun Jiang, MD, Department of Vascular Surgery, Peking University People's Hospital, 11 Xizhimen South St, West District, Beijing 100044, China (e-mail: jjjdoctor@163.com).

The editors and reviewers of this article have no relevant financial relationships to disclose per the Journal policy that requires reviewers to decline review of any manuscript for which they may have a conflict of interest.

2352-667X

http://dx.doi.org/10.1016/j.jvsc.2014.10.005

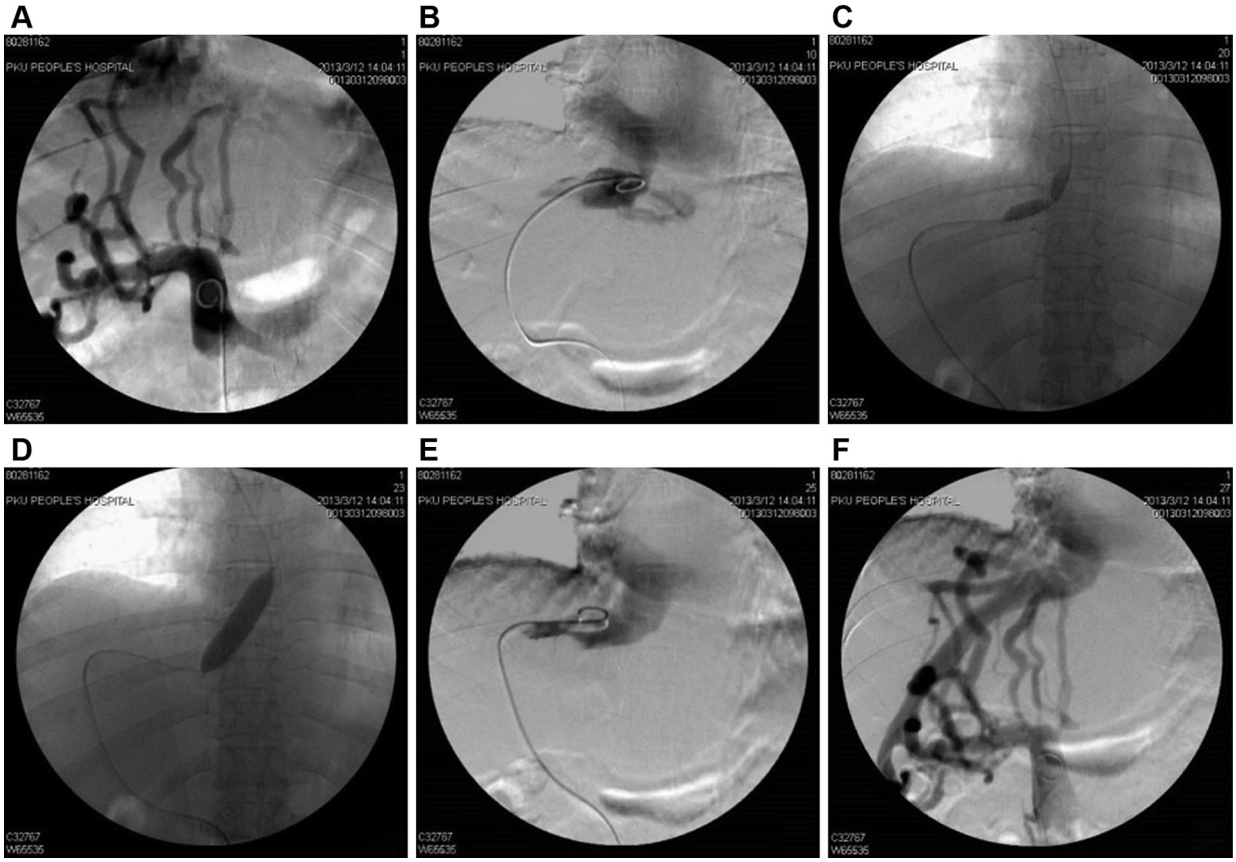

Fig 1. A, Long obstructive lesion of the hepatic segment of the inferior vena cava (IVC), accessory hepatic vein (AHV) compensatory enlargement, and dilation of the abundant intrahepatic communicating branched veins. **B,** Severe stenosis of the IVC. **C** and **D,** The lesion was subjected to balloon angioplasty (diameters of 8 mm and 14 mm successively). **E,** The stenosis had disappeared. **F,** The blood flow to the heart through the hepatic vein (HV) returned to normal.

in the HV fell to 15 cm H_2O. The abdominal distention and lower extremity edema also resolved within 1 week. Warfarin was used for long-term anticoagulation. The follow-up plan was the same as in the first case. The US examination 12 months after the procedure showed a normal-size liver and patency of the HV and the suprahepatic IVC.

DISCUSSION

BCS is defined as hepatic venous outflow obstruction at any level from the small HVs to the junction of the IVC and the right atrium.[1] The goal of therapy is to relieve hepatic venous outflow tract obstruction. Treatments of BCS include radical resection of lesions, liver transplantation, surgical shunts, and endovascular techniques including angioplasty with or without stent placement. Open surgeries that were previously used as a standard treatment always have a high mortality rate.[2] Endovascular treatment, with its minimally invasive and reproducible features, can shorten the hospital stay and reduce the mortality rate.[3-5] It has become the primary treatment of choice for BCS, especially for those with short-length HV or IVC stenosis or occlusion.[6] The Achilles heel of endovascular treatment is the high rate of restenosis.[7]

The AHV was first observed by Elias and Petty in 1952.[8] The angle between the AHV and the IVC is approximately 90 degrees.[9] In patients with BCS, the abundant intrahepatic communicating branched veins dilate (because of HV stenosis or occlusion), and the AHV compensates for hepatic venous drainage. At this time, signs and symptoms of posthepatic portal hypertension may not appear. Catheterization of the HV through the femoral vein-IVC route is usually difficult because of the acute angle between the HV and IVC. Catheterization of the HV through the jugular vein-IVC route can usually be attained, but internal jugular vein puncture subjects the patient to the risk of carotid artery injury or hemothorax or pneumothorax. Percutaneous transhepatic access is technically difficult, and the incidence of complications such as HV thrombosis, biliary tract injuries, and hemorrhage is high.[10] Catheterization of the AHV is more easily achieved than catheterization of the HV because of the larger angle between the AHV and the IVC. The abundant intrahepatic communicating branched veins often dilate in BCS because of the peripheral HV stenosis or occlusion, thereby allowing easier access and cannulation of the HV (Fig 3). Between June 2001 and December 2013, 236 patients with

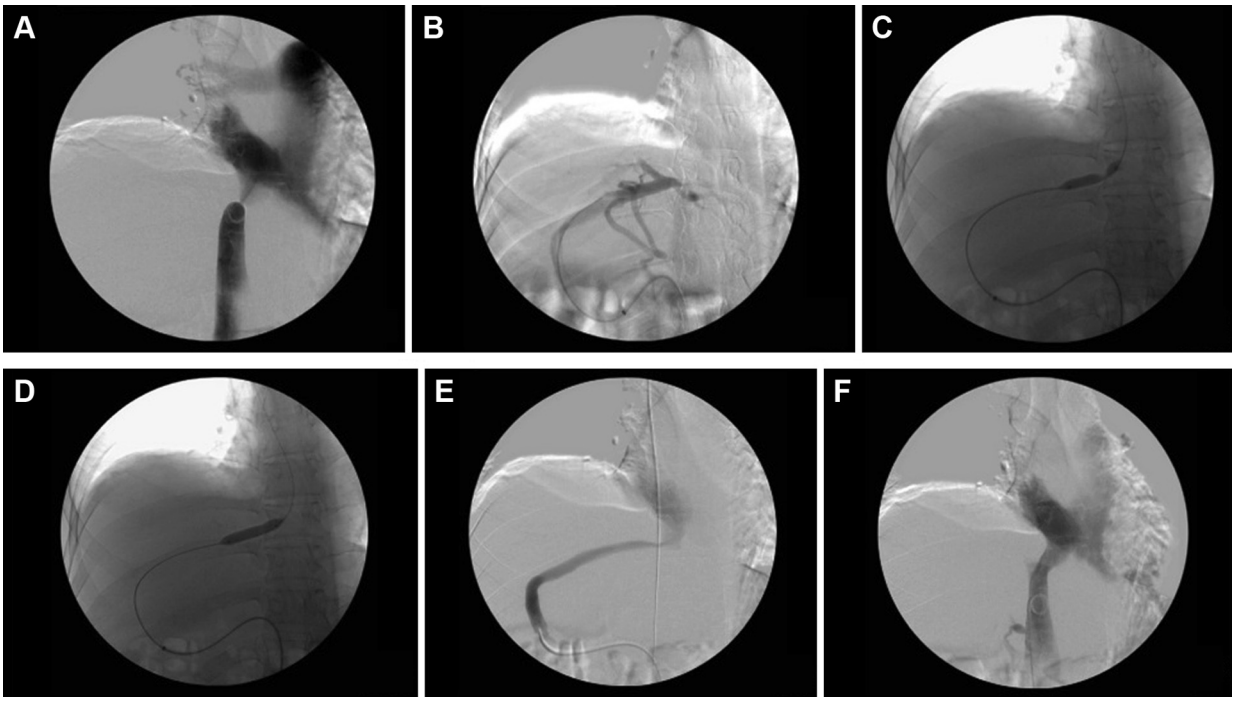

Fig 2. A, Severe stenosis of the inferior vena cava (IVC) and stenosis of the opening of the accessory hepatic vein (AHV). **B,** Obstruction at the junction of the right hepatic vein (HV) and the IVC. **C** and **D,** Angioplasty with a balloon 10 mm in diameter. **E,** The stenosis of the HV had disappeared. **F,** The stenosis of the IVC had disappeared.

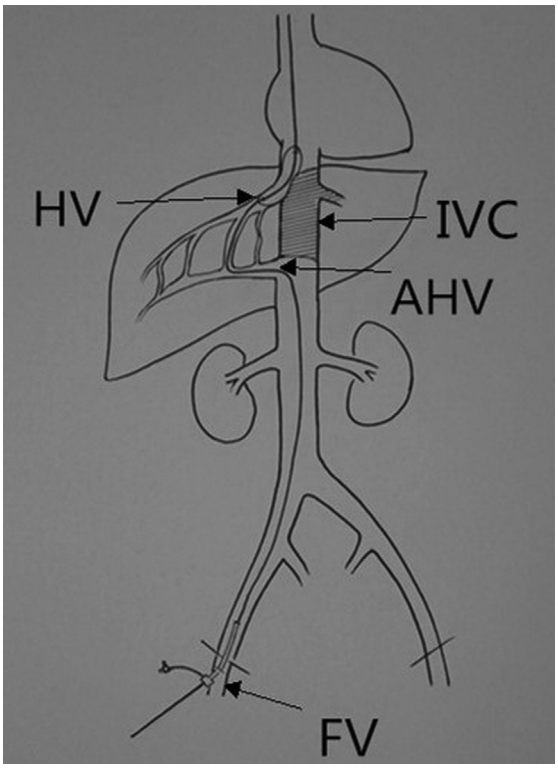

Fig 3. The schematic drawing of this new approach. *AHV,* Accessory hepatic vein; *FV,* femoral vein; *HV,* hepatic vein; *IVC,* inferior vena cava.

BCS were treated in our hospital. Isolated HV block accounted for 18% of the cases. Combined IVC and HV occlusion accounted for 67% of the cases, whereas 15% of the patients had pure IVC occlusion. Of these patients, 106 were treated by radical resection surgery, 119 were treated by angioplasty with or without stent placement, 7 were treated with thrombolysis, and 4 were treated with surgical shunts. In addition to these two patients, we unsuccessfully attempted this approach in three other patients and thought that we failed because the occluded segment of the HV or IVC was too long. We believe that the approach described in this report is likely to be successful for short-segment stenosis or occlusion of the HV or suprahepatic IVC. To the best of our knowledge, this is the first report describing the femoral vein-AHV-intrahepatic communicating branched vein-HV-IVC loop approach.

CONCLUSIONS

Catheterization of the AHV, intrahepatic communicating branches, and HV to reach the IVC is a new and novel approach in the endovascular treatment of BCS and can be used in selected patients.

REFERENCES

1. Janssen HL, Garcia-Pagan JC, Elias E, Mentha G, Hadenque A, Valla D, et al. Budd-Chiari syndrome: a review by an expert panel. J Hepatol 2003;38:364-71.
2. Slakey DP, Klein AS, Venbrux AC, Cameron JL. Budd-Chiari syndrome: current management options. Ann Surg 2001;233:522-7.

3. Beckett D, Olliff S. Interventional radiology in the management of Budd-Chiari syndrome. Cardiovasc Intervent Radiol 2008;31:839-47.
4. Plessier A, Sibert A, Consigny Y, Hakime A, Zappa M, Denninger MH, et al. Aiming at minimal invasiveness as a therapeutic strategy for Budd-Chiari syndrome. Hepatology 2006;44:1308-16.
5. Kwasniewska-Rutczynska A, Bakon L, Januszewicz M, Wroblewski T, Krawczyk M, Rowinski O. Budd-Chiari syndrome: current options in interventional radiology treatment exemplified by three selected cases. Med Sci Monit 2006;12:CS4-12.
6. Meng QY, Sun NF, Wang JX, Wang RH, Liu ZX. Endovascular treatment of Budd-Chiari syndrome. Chin Med J (Engl) 2011;124:89-92.
7. Fisher NC, McCafferty I, Dolapci M, Wali M, Buckels JA, Olliff SP, et al. Managing Budd-Chiari syndrome: a retrospective review of percutaneous hepatic vein angioplasty and surgical shunting. Gut 1999;44:568-74.
8. Elias H, Petty D. Gross anatomy of the blood vessels and ducts within the human liver. Am J Anat 1952;90:59-111.
9. Williams PL, Warwick R, Dyson M. Gray's anatomy. 37th edition. London: Churchill Livingstone; 1995. p. 1602-6.
10. Li T, Zhai S, Pang Z, Ma X, Cao H, Bai W, et al. Feasibility and midterm outcomes of percutaneous transhepatic balloon angioplasty for symptomatic Budd-Chiari syndrome secondary to hepatic venous obstruction. J Vasc Surg 2009;50:1079-84.

Submitted Jul 3, 2014; accepted Oct 25, 2014.