

Use of Accessory Hepatic Vein Intervention in the Treatment of Budd–Chiari Syndrome

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Abstract

Purpose To evaluate the clinical value of accessory hepatic vein (AHV) intervention in the treatment of Budd–Chiari syndrome (BCS).

Patients and Methods From August 2008 to July 2014, consecutive patients with BCS caused by obstruction of three hepatic veins (HVs) with or without obstruction of inferior vena cava (IVC) were treated by recanalization or transjugular intrahepatic portosystemic shunt in our center. Patients who had the compensatory AHV and successfully underwent recanalization of AHV outflow were enrolled in

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this retrospective study. The clinical response to AHV drainage was analyzed.

Results Compensatory AHV was found in 69 of 97 (71.1 %) patients, and 66 patients successfully underwent recanalization of AHV outflow (IVC recanalization, n = 49; AHV recanalization, n = 15; both, n = 2). In total, 78 AHVs were used instead of HV as the hepatic drainage vein after treatment. Fifty-five patients had one AHV, 10 patients had two AHVs, and 1 patient had three AHVs. The average diameter of all AHV stems was 8.0 ± 2.6 mm (range 5–21 mm). Clinical response to AHV drainage was positive in all patients. Patients' symptoms and liver function improved progressively after treatment. During the follow-up of 3–74 months (average 39.4 \pm 11.0 months), 11 patients experienced reobstruction at 6 to 36 months (average 16.8 \pm 9.8 months) after treatment.

Conclusion Compensatory AHV can be effectively used instead of HV for drainage of hepatic blood in patients with BCS. AHV intervention can help to simplify the BCS treatment procedure.

Keywords Budd–Chiari syndrome · Accessory hepatic vein · Intervention treatment

Introduction

Budd–Chiari syndrome (BCS) is a rare disorder consisting of hepatic venous outflow obstruction at any level of hepatic vein (HV) and/or inferior vena cava (IVC) resulting in portal hypertension [1–3]. Intervention treatment, including recanalization and transjugular intrahepatic portosystemic shunt (TIPS), has been widely used to treat BCS. In Western countries, anticoagulation and TIPS provide excellent outcomes in most patients [4–6]. In Asia, recanalization is used as the first line treatment [7–9].

To date, most studies concentrated on TIPS or HV recanalization in the management of BCS [4–9]. In addition to three main HVs, some patients with BCS may have the compensatory accessory hepatic vein (AHV) that connects to the IVC and constitutes the drainage vessel of the liver [10–12]. A retrospective study of AHV recanalization in 20 patients with BCS proved that AHV recanalization is an effective method for the treatment of patients with BCS who had a compensatory AHV [10]. However, large sample studies are still lacking. In this study, we aimed to evaluate the clinical value of AHV intervention in the treatment of BCS.

Patients and Methods

Study Design

This is a retrospective, single-center study. Our Institutional Review Board approved this study. Written informed consent was obtained from each patient.

From August 2008 to July 2014, consecutive patients with symptomatic BCS caused by the obstruction of three main HVs with or without obstruction of IVC were treated by recanalization or TIPS in our center. Patients who had the compensatory AHV and successfully underwent recanalization of AHV outflow were enrolled in this study. Patients were excluded if they had BCS secondary to malignancy, had no compensatory AHV, underwent HV recanalization or TIPS, or experienced technical failure of recanalization. Baseline data of these patients included age, gender, patients' history, risk factors, clinical presentations, imaging examination, laboratory examination, Child-Pugh grade, Child-Pugh score, BCS-TIPS score, Rotterdam score, and New Clichy score.

Patients were followed-up with abdominal Doppler ultrasound and physical examination at 7 days, 1, 3 months, and then every 6 months after recanalization of AHV outflow.

Diagnosis and Definition

Diagnosis of BCS was established by reviewing the patients' history as well as the results of abdominal Doppler ultrasound and magnetic resonance angiography (MRA). Computed tomography angiography (CTA) was performed if the patients had previously undergone the placement of metal item.

An AHV was defined as an intrahepatic vein with the ostium at the third hepatic hilus [11]. A compensatory AHV was defined as an AHV with its stem $\geq 5 \text{ mm}$ [11–13].

Recanalization of AHV outflow referred to as recanalization of AHV outflow obstruction at the level of AHV and/or IVC, while the three HVs were still obstructed after recanalization. In other words, the AHV was used instead of HV to drain the hepatic blood. Technical success of recanalization was defined as elimination of the venous obstruction as determined by venography with disappearance of the collateral vessels. Clinical response to AHV drainage was considered positive if patient's symptoms and liver function test improved after technically successful recanalization of AHV outflow. Reobstruction was suspected if no flow or retrograde flow was present within the lumen or if the degree of lumen obstruction was more than 30 % with intrahepatic collateral vessels on ultrasound examination [8].

Assessment of AHV

Patients with the compensatory AHV were chosen based on the preoperative ultrasound and MRA/CTA results. The number of compensatory AHVs in each patient was determined. The diameter of each AHV stem was measured from the results of MRA/CTA. The value of AHV drainage was accessed by comparing patients' symptoms and liver function indices before and after recanalization.

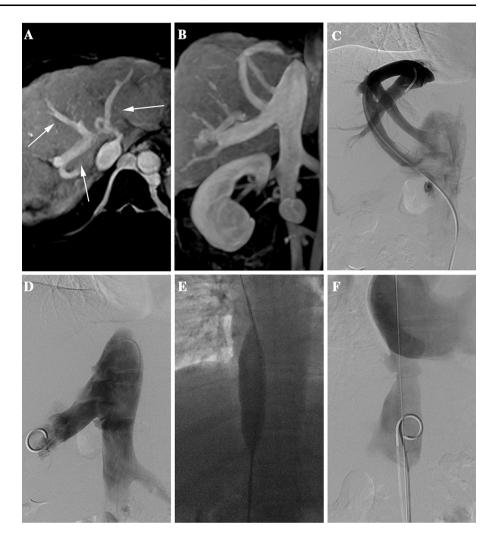
Recanalization of AHV Outflow

All procedures were performed by three interventional radiologists under fluoroscopic guidance. Each patient was placed in the supine position. The blood pressure, heart rate, arterial oxygen saturation, and respiratory rate were monitored throughout the treatment.

If the patient had the obstructed IVC and patent AHV, IVC balloon dilation or stent insertion was performed (Fig. 1). If the patient had the patent IVC and obstructed AHV, AHV balloon dilation or stent insertion was performed (Fig. 2). If the patient had both the obstructed IVC and AHV, both IVC and AHV recanalization were performed. AHV recanalization was performed if the obstruction length of AHV was shorter than that of each of the three main HVs. Stent insertion was required if more than 30 % residual stenosis was present after balloon dilation [7].

IVC recanalization was performed from the femoral vein approach. The approach to AHV recanalization depended on the angle between the ostium of AHV and the distal side of IVC. The femoral vein approach was used if the angle was obtuse or right. Otherwise, the jugular vein approach was used.

After treatment, all patients received subcutaneous lowmolecular-weight heparin (5000 IU, twice a day) for 3 days, followed by warfarin sodium for 12 months. The international normalized ratio was maintained at 2–3. Fig. 1 A 45-year-old patient with the obstructed IVC and three patent AHVs underwent IVC recanalization. The AHVs were used as the hepatic drainage veins. A MRA displayed the obstruction of all three HVs (*arrow*). B MRA displayed the obstructed IVC and three patent AHVs. C, D Venography confirmed the three patent AHVs. E, F IVC was patent after IVC balloon dilation, and the obstructed HV was not managed



Statistical Analysis

Continuous variables are summarized as mean \pm standard deviation. The paired samples *t* test was performed to compare variables before and after treatment. Categorical variables were compared by χ^2 test or Fisher exact test. Cumulative recanalization patency was calculated using Kaplan–Meier curves. A *p* value <0.05 was considered statistically significant. All statistical calculations were performed using SPSS 16.0 (Chicago, IL, USA).

Results

Patients

During the enrollment period, 97 patients with BCS caused by the obstruction of three main HVs with (n = 52) or without (n = 45) IVC obstruction were treated by recanalization or TIPS in our center. We first excluded 28 patients with no compensatory AHV. Among them, 19 patients were treated by HV recanalization, 8 patients were treated by TIPS, and 1 patient was treated by IVC and HV recanalization. Thus, the remaining 69 patients had the compensatory AHV. We further excluded 3 patients. Among them, 2 patients were treated by HV recanalization, and 1 patient failed to undergo HV and AHV recanalization due to long segmental obstruction of three HVs and AHV. This patient underwent TIPS. Thus, 66 patients successfully underwent recanalization of AHV outflow. The baseline data of the 66 patients are shown in Table 1.

Baseline Data of AHV

In total, 78 compensatory AHVs (average, 1.2 AHVs per patient) were found among all 66 patients (Table 2). All 78 AHVs were used as the hepatic drainage vein instead of HV after recanalization in these 66 patients. There were 61 patent AHVs and 17 obstructed AHVs. Fifty-five patients had one AHV, 10 patients had two AHVs, and 1 patient had three AHVs. Among the 55 patients with one AHV, 38 patients had a patent AHV, and 17 patients had an

Table 1Baseline data of the 66patients with BCS whounderwent recanalization ofAHV outflow	Variable	Value	
	Sex (male/female)	34/32	
	Age (years)	$46.4 \pm 12.7 \ (21-79)$	
	Duration (months)	$14.1 \pm 8.3 \ (1-38)$	
	Risk factors		
	JAK2 mutation	0	
	Protein C deficiency	0	
	Protein S deficiency	0	
	Factor V Leiden mutation	0	
	Imaging findings		
	Patients with IVC obstruction	49	
	Patients with AHV obstruction	15	
	Patients with both IVC and AHV obstruction	2	
	Laboratory tests		
	Prothrombin time (PT) (s)	$15.0 \pm 2.6 \ (11.6 - 25.9)$	
	International normalized ratio (INR)	$1.2 \pm 0.2 \ (0.86 - 2.10)$	
	Aspartate aminotransferase (AST) (U/L)	33.8 ± 27.9 (10–136)	
	Alanine aminotransaminase (ALT) (U/L)	35.4 ± 17.8 (16–97)	
	Total bilirubin (TBIL) (µmol/L)	$36.8 \pm 19.1 \ (12.7 - 107.8)$	
	Albumin (g/L)	$38.3 \pm 5.8 \ (25.1 - 48.4)$	
	Creatinine (µmol/L)	$59.3 \pm 18.0 \; (34122)$	
	Alpha-fetoprotein (AFP) (µg/L)	$7.2 \pm 9.9 \ (0.65 - 48.38)$	
	Carcinoembryonic antigen (CEA) (ng/ml)	$2.4 \pm 1.6 \ (0.18 - 11.7)$	
	Cancer antigen 125 (CA125) (U/ml)	$121.5 \pm 200.4 \ (7.5 - 1480.5)$	
	Cancer antigen 19-9 (CA19-9) (U/ml)	$12.3 \pm 8.0 \ (0.1-39.73)$	
	Child–Pugh grade (A/B/C)	22/37/7	
	Child–Pugh score	$7.4 \pm 1.6 (5-12)$	
	BCS-TIPS score	$10.2 \pm 3.3 \; (6.31 24.05)$	
	Rotterdam score	$1.1 \pm 0.4 \ (0.05 - 1.47)$	
	New Clichy score	$4.6 \pm 1.4 \ (2.35 - 9.28)$	

BCS Budd-Chiari syndrome, IVC inferior vena cava, HV hepatic vein, AHV accessory HV

Table 2 Baseline data of the AHV AHV	Variable	Value
	Patients number	66
	Total number of AHVs	78
	Patent AHV	61
	Obstructed AHV	17
	Patient with one AHV	55
	Patient with two AHVs	10
	Patient with three AHVs	1
	Average diameter of total AHVs (mm)	$8.0 \pm 2.6 (5-21)$
	Average diameter of AHVs in patient who had one AHV (mm)	8.2 ± 2.3 (6–21)

AHV accessory hepatic vein

obstructed AHV. All AHVs in the patients with two and three AHVs were patent. The diameter of the AHV stem was measured by MRA in 63 patients and CTA in 3 patients. The average diameter of all AHV stems was 8.0 ± 2.6 mm (range 5–21 mm). The average diameter of the AHV stems in the 55 patients with one AHV was 8.2 ± 2.3 mm (range 6–21 mm). The average diameters of the two AHV stems in the 10 patients with two AHVs were

Table 3 Improvements insymptoms before and afterrecanalization of AHV outflow

	Before (no.)	1 month after treatment (no.)		3 months after treatment (no.)	
		Disappear	Improve	Disappear	Improve
Abdominal distension	63	52	11	63	0
Abdominal pain	16	16	0	16	0
Ascites	58	42	16	55	3
Hepatomegaly	50	32	18	41	9
Splenomegaly	43	28	15	36	7
Gastrointestinal bleeding	10	10	0	10	0

AHV accessory hepatic vein

Table 4 Number of patientswith abnormal and normal liverfunction indices before and afterrecanalization of AHV outflow

	Before (no.)		1 month after treatment (no.)		3 months after treatment (no.)	
	Abnormal	Normal	Abnormal	Normal	Abnormal	Normal
TBIL	57	9	41	25	17	49
Albumin	17	49	13	53	2	64
AST	16	50	10	56	3	63
ALT	20	46	12	54	2	64

AHV accessory hepatic vein, TBIL total bilirubin, AST aspartate aminotransferase, ALT alanine transaminase

 8.1 ± 2.3 mm (range 6–13 mm) and 6.3 ± 1.3 mm (range 5–7 mm), respectively. The diameters of the three AHV stems in the patient with three AHVs were 20, 6, and 5 mm, respectively. There were also many intrahepatic collateral circulations.

Treatment

Among the 66 patients, 49 patients had the obstructed IVC and patent AHV and underwent IVC balloon dilation (n = 35) or stent insertion (n = 14), 15 patients had the patent IVC and obstructed AHV and underwent AHV balloon dilation (n = 12) or stent insertion (n = 3), and 2 patients had both obstructed IVC and AHV and underwent IVC and AHV balloon dilation (n = 1) or IVC stent insertion and AHV balloon dilation (n = 1). Four patients experienced hematoma in right groin. These patients were managed by local compression.

All IVC stents were Z-type stents with a diameter of 28–30 mm and a length of 70–90 mm (Yongtong, Shenyang, China). The AHV stents included Zilver stent (Cook, Bloomington, Indiana, USA) and Luminexx stent (Bard, Murray Hill, New Jersey, USA) with a diameter of 10–12 mm and a length of 40–60 mm.

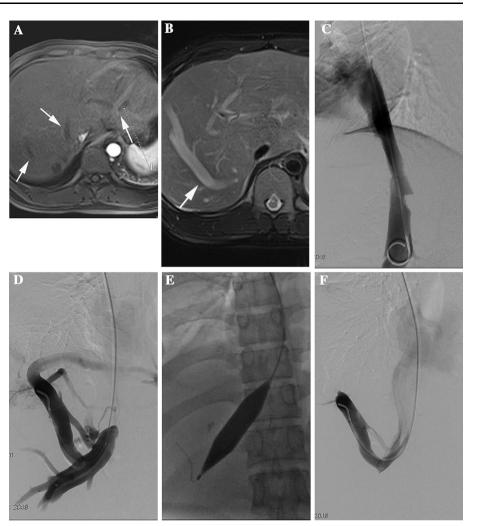
Follow-Up

During the follow-up of 3–74 months (average 39.4 \pm 11.0 months), 11 patients experienced reobstruction of IVC

(n = 8) or AHV (n = 3) at 6 to 36 months (average 16.8 \pm 9.8 months) after treatment. There was no significant difference between reobstruction of IVC and AHV (8/51 vs. 3/17, p = 0.557). All of these patients were revised by subsequent IVC balloon dilation (n = 6), IVC stent insertion (n = 2), or AHV balloon dilation (n = 3). All patients were alive at the time of this report. The cumulative 1-, 2-, and 4-year primary patency rates were 90.7, 83.9, and 80.2 %, respectively. The cumulative 1-, 2-, and 4-year secondary patency rates were 100, 98.3, and 90.8 %, respectively.

Clinical response to AHV drainage after treatment was positive in all patients. Patients' symptoms improved progressively from 1 to 3 months after treatment (Table 3). Patients' liver function also improved progressively from 1 to 3 months after treatment. The preoperative levels of total bilirubin (TBIL) (normal range 1.7-20 µmol/L), albumin (normal range 35-55 g/L), aspartate aminotransferase (AST) (normal range 0-40 U/L), and alanine transaminase (ALT) (normal range 0-40 U/L) were abnormal in 57, 17, 16, and 20 patients, respectively. The number of patients with abnormal TBIL, albumin, AST, and ALT decreased progressively from 1 to 3 months after treatment (Table 4). The average value of abnormal preoperative TBIL, albumin, AST, and ALT improved from $40.0 \pm 18.6 \ \mu mol/L$, $30.5 \pm 2.8 \ g/L$, $71.2 \pm 35.3 \ U/L$, and 56.5 \pm 18.0 U/L to 31.6 \pm 16.0 μ mol/L (t = 10.669, p < 0.001), 33.5 \pm 2.3 g/L (t = 7.278, p < 0.001), 54.1 \pm 26.1 U/L (t = 6.119, p < 0.001), and 45.3 ± 14.3 U/L

Fig. 2 A 30-year-old patient with the patent IVC and obstructed AHV underwent AHV recanalization. A MRA displayed the obstructions of all three HVs (arrow). B MRA displayed a compensatory but obstructed AHV (arrow). C Venography showed the patent IVC. D Venography showed the obstructed AHV and intrahepatic collateral vessels. E, F The AHV was used instead of the HV as the hepatic drainage vein after AHV balloon dilation



(t = 6.936, p < 0.001) 1 month after treatment, respectively. The normal preoperative TBIL, albumin, AST, and ALT levels were still within the normal range 1 month after treatment. The average value of abnormal TBIL, albumin, AST, and ALT at 1 month after treatment improved from 36.6 ± 16.3 µmol/L, 32.6 ± 1.9 g/L, 66.8 ± 25.4 U/L, and 51.7 ± 15.2 U/L to 20.2 ± 5.6 µmol/L (t = 8.800, p < 0.001), 38.8 ± 3.4 g/L (t = 9.818, p < 0.001), 39.5 ± 13.0 U/L (t = 5.165, p = 0.001), and 34.5 ± 5.7 U/L (t = 5.086, p < 0.001) 3 months after treatment, respectively. The normal 1-month postoperative TBIL, albumin, AST, and ALT levels were still within the normal range 3 months after treatment.

Discussion

This study evaluated the clinical value of AHV intervention in the treatment of BCS. Initial results were positive. The clinical response to AHV drainage was positive in all 66 patients with BCS caused by the obstruction of three HVs who successfully underwent recanalization of AHV outflow.

In healthy people, AHV is usually thin and is not the main hepatic drainage vein [12]. Therefore, AHV is usually not given enough attention. However, AHV can become compensatorily dilated in some patients with BCS, because the increased hepatic pressure can cause the hepatic blood to flow into the AHV via the intrahepatic collateral circulations [10–12].

MRA or CTA can provide a good evaluation of AHV [11, 12]. MRA shows a high accuracy in the detection and grading of vascular disease [14]. There is no significant difference in the measurement of vascular length or diameter between MRA and digital subtraction angiography [14]. In this study, the compensatory AHV was found in 71.1 % (69/97) of patients with BCS. This rate is comparable with that (64 %) in an MRA study of AHVs in patients with BCS [12]. The average diameter of AHV stems in our patients was 8.0 ± 2.6 mm, which is also comparable with that (7.3 ± 3.9 mm) in the MRA study [12].

The purpose of management of BCS is improving patients' symptoms and liver function [5–8]. Most previous studies have demonstrated that HV recanalization is suitable for patients with short segmental obstruction of HV [7–9]. However, HV recanalization is difficult with a high failure rate in patients with long segmental obstruction or diffuse obstruction of HV [7–9]. TIPS is considered to be the first choice for BCS secondary to long segmental obstruction or diffuse obstruction of HV [4–6]. Although TIPS can effectively decrease the pressure of portal vein and significantly improve patients' symptoms, hepatic encephalopathy occurred in 17 % of patients who underwent TIPS [6].

In this study, we used the compensatory AHV instead of HV as the hepatic drainage vein. Patients' symptoms and liver function improved after recanalization of AHV outflow. Fifty-five of 66 (83.3 %) patients had only one compensatory AHV. This finding may indicate that single compensatory AHV is enough for patients with BCS. This finding is also similar to the finding that recanalization of single HV can allow for drainage of the entire liver [7-9]. Most AHVs (61/78, 78 %) were patent in this study, thus, there was no need to manage the obstructed HV or perform TIPS. There were also 17 (12 %) obstructed AHVs in this study. This obstruction occurred because the ostium of the AHV is restricted by the IVC wall and does not dilate along with the AHV stem dilation [15]. Under this condition, recanalization of the obstructed AHV is also a simple and effective method for management of BCS [10]. Compared with TIPS, AHV recanalization is performed in reference to the physiological anatomy, and there is no portacaval shunt placement after treatment [10]. Therefore, before TIPS is planned for a patient with BCS, it is important to confirm whether the patient has the compensatory AHV.

There are some limitations in this study. First, the patients were from a single center. Second, there was no control group. Third, most but not all patients had the compensatory AHV, therefore, use of the AHV was only suitable for these selected patients.

In conclusion, our study indicates that the compensatory AHV appears in most patients with BCS and that it can effectively drain the hepatic blood instead of HV when meeting the following criteria: (1) more than one compensatory AHV; (2) single compensatory AHV with its stem diameter at least 6 mm. If the compensatory AHV is patent, there is no need to manage the obstructed HVs. If the compensatory AHV is obstructed, AHV recanalization is also an effective treatment option.

Conflict of interest All authors including Yu-Fei Fu, Ning Wei, Qian Wu, Qing-Qiao Zhang, Yan-Feng Cui, and Hao Xu of this study declare that they have no conflicts of interest.

References

- Qi X, Zhang C, Han G, et al. Prevalence of the JAK2V617F mutation in Chinese patients with Budd–Chiari syndrome and portal vein thrombosis: a prospective study. J Gastroenterol Hepatol. 2012;27(6):1036–43.
- Wang H, Sun G, Zhang P, et al. JAK2 V617F mutation and 46/1 haplotype in Chinese Budd–Chiari syndrome patients. J Gastroenterol Hepatol. 2014;29(1):208–14.
- Qi X, De Stefano V, Wang J, et al. Prevalence of inherited antithrombin, protein C, and protein S deficiencies in portal vein system thrombosis and Budd–Chiari syndrome: a systematic review and meta-analysis of observational studies. J Gastroenterol Hepatol. 2013;28(3):432–42.
- Tripathi D, Macnicholas R, Kothari C, et al. Good clinical outcomes following transjugular intrahepatic portosystemic stentshunts in Budd–Chiari syndrome. Aliment Pharmacol Ther. 2014;39(8):864–72.
- Fitsiori K, Tsitskari M, Kelekis A, Filippiadis D, Triantafyllou K, Brountzos E. Transjugular intrahepatic portosystemic shunt for the treatment of Budd–Chiari syndrome patients: results from a single center. Cardiovasc Intervent Radiol. 2014;37(3):691–7.
- Eapen CE, Velissaris D, Heydtmann M, Gunson B, Olliff S, Elias E. Favourable medium term outcome following hepatic vein recanalisation and/or transjugular intrahepatic portosystemic shunt for Budd Chiari syndrome. Gut. 2006;55(6):878–84.
- Gao Y, Chen S, Yu C. Applicability of different endovascular methods for treatment of refractory Budd–Chiari syndrome. Cell Biochem Biophys. 2011;61(2):453–60.
- Zhang CQ, Fu LN, Xu L, et al. Long-term effect of stent placement in 115 patients with Budd–Chiari syndrome. World J Gastroenterol. 2003;9(11):2587–91.
- Cheng D, Xu H, Lu ZJ, et al. Clinical features and etiology of Budd–Chiari syndrome in Chinese patients: a single-center study. J Gastroenterol Hepatol. 2013;28(6):1061–7.
- Fu YF, Xu H, Zhang K, Zhang QQ, Wei N. Accessory hepatic vein recanalization for treatment of Budd-Chiari syndrome due to long-segment obstruction of the hepatic vein: initial clinical experience. Diagn Interv Radiol. 2015;21(2):148–53.
- Hanaoka J, Shimada M, Uchiyama H, et al. A simple formula to calculate the liver drainage volume of the accessory right hepatic vein using its diameter alone. Surgery. 2009;146(2):264–8.
- Tang W, Zhang XM, Yang L, Mitchell DG, Zeng NL, Zhai ZH. Hepatic caudate vein in Budd–Chiari syndrome: depiction by using magnetic resonance imaging. Eur J Radiol. 2011;77(1): 143–8.
- Buhe S, Miyaki T, Saito T, et al. A study of the accessory hepatic vein to segments VI and VII with a morphological reconsideration of the human liver. Surg Radiol Anat. 2008;30(3):201–7.
- Loewe C, Schillinger M, Haumer M, et al. MRA versus DSA in the assessment of occlusive disease in the aortic arch vessels: accuracy in detecting the severity, number, and length of stenoses. J Endovasc Ther. 2004;11(2):152–60.
- Xu H, Zu MH, Li GJ, et al. Interventional therapy of Budd–Chiari syndrome with hepatic vein obstruction. Chin J Med Imaging Technol. 2004;20(10):1588–91 (in chinese).