

Percutaneous recanalization for hepatic vein-type Budd-Chiari syndrome: long-term patency and survival

Yan-Feng Cui¹ · Yu-Fei Fu² · De-Chun Li² · Hao Xu¹

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Abstract

Objective To determine the long-term patency and survival of percutaneous recanalization for hepatic vein (HV)-type Budd-Chiari syndrome (BCS).

Methods From March 2009 to November 2014, consecutive symptomatic HV-type BCS patients were treated by percutaneous recanalization in our centers. These patients underwent main HV (MHV) or accessory HV (AHV) recanalization. Data on patient characteristics, technical success, clinical success, long-term patency, and survival were collected and analyzed.

Results During the enrolled periods, a total of 143 symptomatic HV-type BCS patients were treated by percutaneous recanalization in our centers. Technical success was achieved in 140 of 143 patients. One hundred eleven patients underwent MHV recanalization, and 29 underwent AHV recanalization. Clinical success was achieved in 136 of 140 patients. The mean MHV/AHV pressure decreased from 33.5 ± 4.1 mmHg before treatment to 12.5 ± 3.1 mmHg after treatment ($p = 0.000$). The 136 patients were followed for 7–75 months (mean 33.9 ± 15.3 months). Twenty-eight patients experienced re-obstruction of MHV ($n = 24$) or AHV ($n = 4$) at 3 to 36 months (mean 18.0 ± 11.5 months) after treatment. The cumulative 1-, 3-, and 6-year primary patency rates

were 91.1, 77.4, and 74.0 %, respectively. The cumulative 1-, 3-, and 6-year secondary patency rates were 97.0, 92.4, and 88.8 %, respectively. The cumulative 1-, 3-, and 6-year survival rates were 97.7, 92.2, and 90.0 %, respectively.

Conclusion Percutaneous recanalization can provide good long-term patency and survival in HV-type BCS patients.

Keywords Hepatic vein · Budd-Chiari syndrome · Percutaneous recanalization · Long-term outcomes

Introduction

Budd-Chiari syndrome (BCS) is a rare disease characterized by hepatic venous outflow obstruction [1–11]. According to the different obstructed sites, the BCS is divided into three types: (1) inferior vena cava (IVC)-type BCS is defined as IVC obstruction with at least one patent main hepatic vein (MHV); (2) hepatic vein (HV)-type BCS is defined as obstruction of the three MHVs; (3) combined-type BCS is defined as obstruction of both the IVC and three MHVs [1]. IVC-type BCS can easily be treated by IVC recanalization [2–4]. Approximately 86–89 % of combined-type BCS patients have a compensatory and patent accessory HV (AHV); therefore, single IVC recanalization is suitable for most combined-type BCS patients [5, 6].

The strategy for treating HV-type BCS patients is relatively complex. MHV recanalization is suitable for most HV-type BCS patients [7]. If MHV recanalization fails, a transjugular intrahepatic portosystemic shunt (TIPS) should be considered [8]. However, along with the in-depth research involving the AHV in BCS, AHV recanalization can be an effective method for HV-type BCS patients with a compensatory AHV [9–11]. Recently, the long-term

Y.-F. Cui and Y.-F. Fu contributed equally to this work.

✉ Hao Xu
xuhao587@163.com

¹ Department of Interventional Radiology, The Affiliated Hospital of Xuzhou Medical College, 99 West Huai-hai Road, Xuzhou 221006, China

² Department of Radiology, Xuzhou Central Hospital, 199 South Jie-fang Road, Xuzhou 221009, China

outcome of percutaneous recanalization for HV-type BCS was still not known. In this study, we determined the long-term patency and survival of percutaneous recanalization for HV-type BCS.

Materials and methods

Patients selection

From March 2009 to November 2014, consecutive symptomatic HV-type BCS patients were treated by percutaneous recanalization in our centers. Patients were excluded if they had BCS secondary to a malignant tumor, asymptomatic BCS due to well-established intrahepatic collateral vessels, achieved clinical success of medical treatment only (anticoagulation and diuresis), or underwent TIPS, a surgical shunt, or liver transplant. Patients' baseline data before treatment included age, sex, symptoms, imaging findings, and laboratory examination findings.

Diagnosis and preoperative evaluation

Diagnosis of HV-type BCS was established by reviewing patients' history, abdominal ultrasound findings, and abdominal magnetic resonance angiography (MRA)/computed tomography angiography (CTA) findings. All patients' blood samples were collected to check for BCS risk factors (JAK2 V167F mutation, protein C deficiency, protein S deficiency, and factor V Leiden mutation). Symptomatic BCS is defined as a BCS patient who has any one of the following clinical manifestations: abdominal pain, abdominal distention, jaundice, ascites, variceal bleeding, or encephalopathy [5].

Before treatment, the obstruction length of three MHVs was measured by MRA/CTA. Confirmation of the compensatory AHV by ultrasound and MRA/CTA was made before treatment. The AHV stem diameter was measured from the results of MRA/CTA. A compensatory AHV is defined as an AHV with its stem ≥ 5 mm [9]. If the patient had a compensatory but obstructed AHV, the obstruction length of the AHV was also measured by MRA/CTA. The membranous obstruction of MHV/AHV is defined as an obstruction length ≤ 1 cm, and segmental obstruction of MHV/AHV is defined as an obstruction length >1 cm [6]. The total obstruction of an MHV is defined as no visualization of MHV on MRA/CTA.

Confirmation of the target vein

If the patient had no compensatory AHV, the target vein was the MHV. The target MHV was chosen with the one MHV with the shortest obstruction length. If the patient

had a compensatory but obstructed AHV, we compared the obstruction length between three MHVs and the AHV, and the target vein was chosen with the one MHV or AHV with the shortest obstruction length.

MHV and AHV recanalization

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

If the target vein was the MHV, MHV recanalization was performed. MHV recanalization was routinely performed from the transjugular approach. If the transjugular MHV recanalization failed, the ultrasound-guided percutaneous transhepatic route would be used to access the MHV, and the MHV recanalization was performed via the combined transhepatic and transjugular approaches.

If the target vein was the AHV, AHV recanalization was performed. The approach to AHV recanalization depended on the angle between the ostium of the AHV and distal side of the IVC. The femoral vein approach was used if the angle was obtuse or right. Otherwise, the jugular vein approach was used. If the transjugular or transfemoral AHV recanalization failed, the ultrasound-guided percutaneous transhepatic route was used to access the AHV and the AHV recanalization performed via the combined transhepatic and transjugular/transfemoral approaches.

Percutaneous recanalization was performed with the balloon or stent. Stent insertion was performed if there was $>30\%$ residual stenosis after balloon dilation. MHV or AHV pressure was measured by a piezometer tube before and after recanalization. After treatment, all patients received subcutaneous low-molecular-weight heparin (5000 IU, twice a day) for 3 days, followed by oral warfarin for 12 months. The dose of warfarin was adjusted to maintain the international normal ratio of 2–3.

Definitions and endpoints

Technical success of percutaneous recanalization was defined as the MHV/AHV being restored at venography with the disappearance of intrahepatic collateral vessels. Clinical success was defined as the symptoms and liver function tests improving after technical success of percutaneous recanalization [5]. Re-obstruction was defined as no or retrograde flow being present in the lumen or if the degree of lumen obstruction was $>30\%$ with intrahepatic collateral vessels on ultrasound examination [5]. Re-obstruction was suspected if the BCS-related symptoms reappeared.

All patients underwent abdominal ultrasound and clinical examination 7 days, 1, 3, 6, and then every 6 months after treatment to confirm the long-term patency. The primary endpoint was re-obstruction of the target vein. The secondary endpoints included anticoagulation-related bleeding and death. The follow-up ended at the patients' death, the point of undergoing TIPS, surgical shunt, or liver transplant, the point of being lost to follow-up, or the point of setting this study (June 2015).

Statistical analysis

Continuous variables are summarized as the mean \pm standard deviation. The paired samples *t* test was performed to compare variables before and after treatment. Categorical variables are compared by the chi-square test or Fisher's exact test. Cumulative patency and survival rates were calculated by using Kaplan-Meier curves. The predictors of re-obstruction were determined using univariate and multivariate Cox regression analysis. The covariates incorporated into the multivariate analysis were the variables with $p < 0.1$ on univariate analysis. A p value < 0.05 was considered statistically significant. All statistical calculations were performed using SPSS 16.0 (SPSS, Chicago, IL, USA).

Results

Patients

During the enrolled periods, 143 symptomatic HV-type BCS patients were treated by percutaneous recanalization in our centers. Three patients had hepatic cellular carcinoma (HCC). However, the HCC was not the cause of BCS, so they were not excluded. All patients received medical treatment (anticoagulation and diuresis) for 1 week before percutaneous recanalization, but no patient responded to medical treatment.

Technical success

Technical success was achieved in 140 (97 %) of 143 patients. Four patients experienced mild hematoma at the right jugular region, and they were treated with local pressure. Three patients failed to undergo MHV recanalization because of total obstruction of three MHVs. They were treated with TIPS insertion. One hundred eleven patients underwent MHV recanalization, and 29 underwent AHV recanalization. Among the 29 patients who underwent AHV recanalization, 11 had the total obstruction of three MHVs. One hundred twenty-four patients underwent balloon dilation, and 16 underwent stent insertion. The balloons were

10–14 mm in diameter and 40 mm in length (Cook, Bloomington, IN, USA, or Bard, Murray Hill, NJ, USA). The stents were Zilver stents (Cook) or Luminexx stents (Bard) with a diameter of 10–14 mm and length of 40–60 mm.

Clinical success

Clinical success was achieved in 136 (97 %) of 140 patients. The mean MHV/AHV pressure decreased from 33.5 ± 4.1 mmHg before treatment to 12.5 ± 3.1 mmHg after treatment ($p = 0.000$). Four patients (3 with MHV recanalization and 1 with AHV recanalization) experienced clinical failure due to the decompensated liver cirrhosis, and they were treated with TIPS insertion. The baseline data and treatment details of these 136 patients are demonstrated in Tables 1 and 2, respectively.

Patency

The 136 patients were followed for 7–75 months (mean 33.9 ± 15.3 months). No patient was lost to follow-up. Twenty-eight patients experienced re-obstruction of the MHV ($n = 24$) or AHV ($n = 4$) 3–36 months (mean 18.0 ± 11.5 months) after treatment. There was no significant difference in re-obstruction between patients who underwent MHV and AHV recanalization (24/124 vs. 4/28, $p = 0.532$). There was no significant difference in re-obstruction between patients with and without stent insertion (6/16 vs. 22/120, $p = 0.075$). Among the 28 patients, 26 underwent repeat percutaneous recanalization (balloon dilation: 18; stent: 8); the remaining 2 underwent TIPS insertion because of the refractory gastrointestinal bleeding. The cumulative 1-, 3-, and 6-year primary patency rates were 91.1, 77.4, and 74.0 %, respectively. The cumulative 1-, 3-, and 6-year secondary patency rates were 97.0, 92.4, and 88.8 %, respectively (Fig. 1).

At univariate analysis, the predictors of re-obstruction were preoperative gastrointestinal bleeding, segmental obstruction of the target vein, a higher alkaline phosphatase level, lower albumin level, higher creatinine level, and higher cancer antigen 125 level. At multivariate analysis, the independent predictor of re-obstruction was segmental obstruction of the target vein (hazard ratio: 2.557, 95 % confidence interval: 1.092–5.986, $p = 0.031$, Table 3).

Survival

Nine patients died 8–38 months (median 15 months) after treatment. The causes of death included hepatic failure ($n = 4$), HCC ($n = 3$), and gastrointestinal hemorrhage ($n = 2$). The hepatic failure and gastrointestinal hemorrhage occurred after the second re-obstruction of MHV ($n = 5$) or AHV ($n = 1$). The cumulative 1-, 3-, and 6-year

Table 1 Baseline data of the 136 patients with technical and clinical success

	Values
Age (years)	32.6 ± 10.7 (14–72)
Sex (male/female)	58/78
Duration of symptoms (months)	13.1 ± 8.4 (1–36)
Risk factors	
JAK2 V167F mutation	5
Protein C deficiency	0
Protein S deficiency	0
Factor V Leiden mutation	0
Clinical manifestations	
Abdominal distention	136
Abdominal pain	46
Ascites	130
Jaundice	16
Gastrointestinal bleeding	12
Liver cirrhosis	18
HBV/HCV/HIV infection	3/0/0
Imaging finding	
Combined compensatory AHV	28
Right MHV obstruction (MO/SO)	38/98
Middle MHV obstruction (MO/SO)	34/102
Left MHV obstruction (MO/SO)	14/122
AHV obstruction (MO/SO)	28/0
Laboratory tests	
Prothrombin time (PT, s)	16.4 ± 3.0 (11.8–28.9)
International normalized ratio (INR)	1.3 ± 0.3 (0.9–2.2)
Aspartate aminotransferase (AST, U/l)	40.3 ± 59.3 (6–315)
Alanine aminotransaminase (ALT, U/l)	45.5 ± 59.7 (11–349)
Alkaline phosphatase (ALP, U/l)	130.5 ± 45.4 (48–293)
Total bilirubin (TBIL, μmol/l)	41.3 ± 36.8 (16.8–280.2)
Albumin (g/l)	36.3 ± 6.4 (22.6–47.3)
Creatinine (μmol/l)	60.6 ± 19.5 (34–122)
Alpha fetoprotein (AFP, μg/l)	5.1 ± 10.0 (0.6–100.2)
Carcinoembryonic antigen (CEA, ng/ml)	1.9 ± 1.2 (0.3–6.7)
Cancer antigen 125 (CA125, U/ml)	140.6 ± 106.7 (11.4–487.1)
Cancer antigen 19-9 (CA19-9, U/ml)	14.9 ± 10.0 (0.1–68.9)
Child-Pugh score	8.1 ± 1.9 (5–12)
BCS-TIPS score	10.1 ± 5.9 (5.7–48.0)
Rotterdam score	1.0 ± 0.5 (0.1–2.2)
New Clichy score	4.8 ± 2.0 (2.4–8.2)

AHV accessory hepatic vein, MHV main hepatic vein, MO membranous obstruction, SO segmental obstruction

survival rates were 97.7, 92.2, and 90.0 %, respectively (Fig. 2).

Discussion

This study demonstrated our clinical results of percutaneous recanalization for HV-type BCS patients. The technical and clinical success rates (97 and 97 %, respectively)

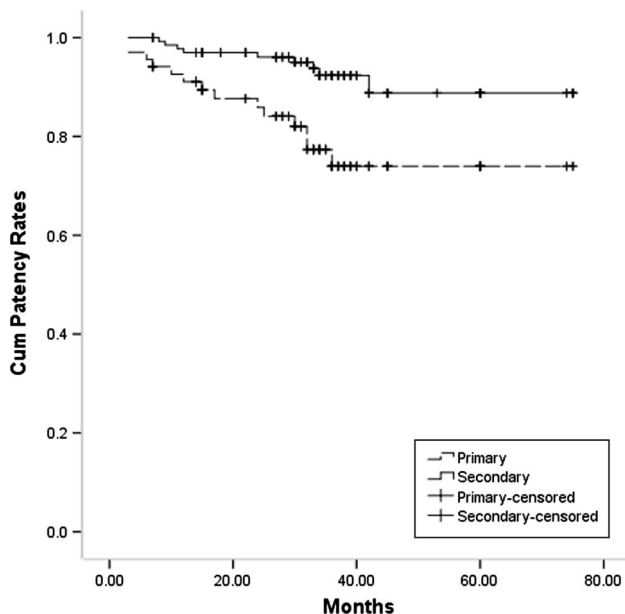
were comparable to previous studies involving percutaneous recanalization for HV-type BCS patients [12, 13]. These results may indicate that percutaneous recanalization is suitable for most HV-type BCS patients.

In the West, a majority of BCS patients are HV-type BCS patients [14–16]. While in Asia, approximately 11–28 % of BCS patients are HV-type BCS patients [1, 5]. The purpose of MHV/AHV recanalization is to relieve

Table 2 Details of treatment procedures

	MHV recanalization group (<i>n</i> = 108)	AHV recanalization group (<i>n</i> = 28)
Balloon dilation	97	23
Stent insertion	11	5
Target MHV		
Right	57	–
Middle	34	–
Left	17	–
Treatment approach		
Transjugular	82	17
Transfemoral	–	8
Combined transhepatic and transjugular	26	2
Combined transhepatic and transfemoral	–	1
Nature of obstruction of target vein		
Membranous obstruction	88	28
Segmental obstruction	20	0

MHV main hepatic vein, AHV accessory hepatic vein

**Fig. 1** Primary and secondary patency rates after treatment

liver congestion, relieve patients' symptoms, and improve liver functions [7–13]. In this study, we performed single MHV/AHV recanalization for our patients. Single MHV/AHV can afford draining the entire liver because of the well-established intrahepatic collateral vessels in the liver of BCS patients [1, 9–11].

Compensatory AHV is a compensatory mechanism in BCS patients [9–11]. Approximately 71 % of BCS patients have compensatory AHV, and approximately 78 % of AHVs are patent [9]. In this study, we found all of the AHV obstruction was membranous obstruction. This result may be attributed to the AHV obstruction occurring because the ostium of the AHV is restricted by the IVC wall and does not dilate along with the AHV stem dilation [9]. If the BCS patients have the segmental obstruction of three MHVs but a compensatory AHV, AHV recanalization can help the patients to avoid TIPS insertion [9–11].

Qi et al. [17] reported the use of TIPS for BCS in Chinese patients. In this study, TIPS insertion was performed for seven patients who experienced technical failure (*n* = 3) or clinical failure (*n* = 4) of percutaneous recanalization. The main indications of TIPS insertions for HV-type BCS were unsuccessful and ineffective percutaneous recanalization.

The cumulative 1-, 3-, and 6-year primary patency rates were 91.1, 77.4, and 74.0 %, respectively. These rates are comparable to previous studies involving percutaneous recanalization for HV-type BCS patients [12, 13]. We further found an excellent cumulative 6-year secondary patency rate of 88.8 %, which supports percutaneous recanalization being well repeatable. We also found that the independent predictor of re-obstruction of HV/AHV was segmental obstruction of the target vein. This risk factor is similar to re-obstruction of IVC in a study involving percutaneous recanalization for IVC-type BCS [2].

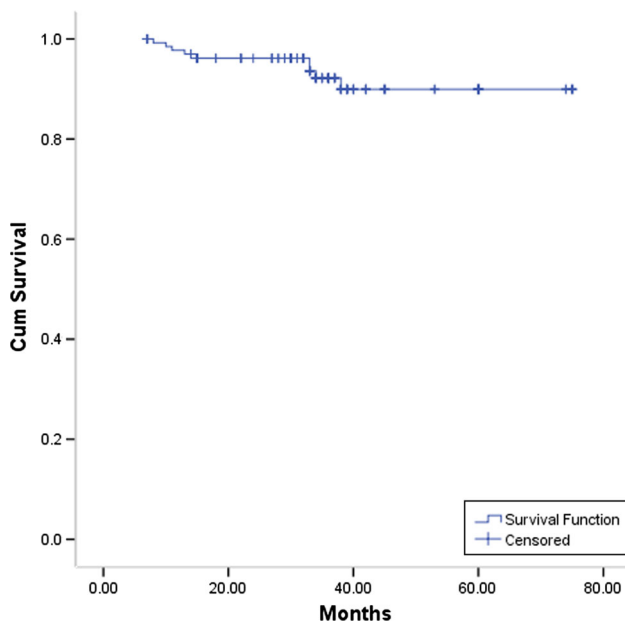
The cumulative 1-, 3-, and 6-year survival rates were 97.7, 92.2, and 90.0 %, respectively. Re-obstruction is considered to be the risk factor of death in BCS patients after percutaneous recanalization [5]. BCS patients are in need of regular follow-up after percutaneous recanalization. If the patients experience re-obstruction, re-intervention should be performed in a timely fashion to decrease the mortality.

In this study, we found that percutaneous recanalization was suitable for 95 % (136 of 143) of HV-type BCS patients. A retrospective study involving interventional treatment demonstrated that percutaneous recanalization was only suitable for 51 % (31 of 61) of BCS patients in the West [18]. The main indication of percutaneous recanalization of BCS is membranous or short length obstruction of MHV or AHV [9, 18]. In Asia, most BCS patients have membranous obstruction of the MHV [19]. However, membranous obstruction or short length obstruction of MHV was only identified in 29–41 % of BCS patients in the West [18, 20]. This phenomenon may explain the different applicability rates of percutaneous recanalization for BCS between the present study and studies of BCS patients in the West.

Table 3 Univariate and multivariate analysis for re-obstruction

	Univariate analysis			Multivariate analysis		
	Hazard ratio	95 % CI	<i>p</i> value	Hazard ratio	95 % CI	<i>p</i> value
Age	0.654	0.301–1.419	0.283			
Sex	0.970	0.933–1.009	0.130			
Duration	0.988	0.943–1.036	0.619			
Gastrointestinal bleeding	2.408	0.974–5.959	0.057	1.882	0.714–4.960	0.201
Segmental obstruction	3.938	1.811–8.562	0.001	2.557	1.092–5.986	0.031
Prothrombin time	1.067	0.950–1.200	0.273			
International normalized ratio	1.670	0.483–5.766	0.418			
Aspartate aminotransferase	1.001	0.996–1.006	0.631			
Alanine aminotransaminase	0.999	0.992–1.006	0.751			
Alkaline phosphatase	1.006	0.999–1.013	0.099	1.007	0.999–1.014	0.071
Total bilirubin	0.981	0.956–1.006	0.134			
Albumin	0.902	0.848–0.960	0.001	0.941	0.878–1.007	0.080
Creatinine	1.108	1.004–1.033	0.012	1.012	0.995–1.029	0.162
Alpha fetoprotein	0.983	0.921–1.049	0.600			
Carcinoembryonic antigen	0.932	0.665–1.305	0.681			
Cancer antigen 125	1.006	1.002–1.009	0.001	1.003	1.000–1.007	0.075
Cancer antigen 19-9	1.012	0.979–1.047	0.476			
Stent	2.140	0.865–5.291	0.100			
Child-Pugh score	1.133	0.973–1.370	0.199			
BCS-TIPS score	0.942	0.841–1.056	0.307			
Rotterdam score	1.462	0.627–3.412	0.380			
New Clichy score	1.140	0.947–1.372	0.165			

CI confidence interval

**Fig. 2** Survival rates after treatment

This study has some limitations. First, the biggest limitation is its retrospective nature. Further randomized controlled trials should be performed. Second, there is no control group in this study. However, we aimed to evaluate the long-term outcomes of percutaneous recanalization for HV-type BCS patients. Third, the sample size is not large.

In conclusion, although further randomized controlled trials are needed, our results demonstrated that percutaneous recanalization can provide good long-term patency and survival in HV-type BCS patients.

Compliance with ethical standards

Conflicts of interest Yan-Feng Cui, Yu-Fei Fu, De-Chun Li, and Hao Xu declare that they have no conflict of interest.

Ethical approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. For this type of study formal consent is not required. This article does not contain any studies with animals performed by any of

the authors. This was a retrospective study approved by our Institutional Review Board. Each patient received the details about percutaneous recanalization and provided written informed consent for percutaneous recanalization before treatment.

Informed consent Informed consent was obtained from all individual participants included in the study.

References

- Zhang CQ, Fu LN, Xu L, Zhang GQ, Jia T, Liu JY, et al. Long-term effect of stent placement in 115 patients with Budd-Chiari syndrome. *World J Gastroenterol*. 2003;9:2587–91.
- Zhang QQ, Xu H, Zu MH, Gu YM, Shen B, Wei N, et al. Strategy and long-term outcomes of endovascular treatment for Budd-Chiari syndrome complicated by inferior vena caval thrombosis. *Eur J Vasc Endovasc Surg*. 2014;47:550–7.
- Sun J, Zhang Q, Xu H, Huang Q, Shen B, Zu M, et al. Clinical outcomes of warfarin anticoagulation after balloon dilation alone for the treatment of Budd-Chiari syndrome complicated by old inferior vena cava thrombosis. *Ann Vasc Surg*. 2014;28:1862–8.
- Fu YF, Xu H, Wu Q, Zhang QQ, Cui YF, Wei N. Combined thrombus aspiration and recanalization in treating Budd-Chiari syndrome with inferior vena cava thrombosis. *Radiol Med*. 2015;. doi:10.1007/s11547-015-0554-9 (Epub 2015 May 24).
- Han G, Qi X, Zhang W, He C, Yin Z, Wang J, et al. Percutaneous recanalization for Budd-Chiari syndrome: an 11-year retrospective study on patency and survival in 177 Chinese patients from a single center. *Radiology*. 2011;266:657–67.
- Fu YF, Li Y, Cui YF, Wei N, Li DC, Xu H. Percutaneous recanalization for combined-type Budd-Chiari syndrome: strategy and long-term outcome. *Abdom Imaging*. 2015;. doi:10.1007/s00261-015-0496-7 (Epub 2015 Jun 30).
- Cui YF, Fu YF, Wei N, Zhu HC, Xu H. Retrograde puncture assisted hepatic vein recanalization in treating Budd-Chiari syndrome with segmental obstruction of hepatic vein. *Radiol Med*. 2015;. doi:10.1007/s11547-015-0557-6 (Epub 2015 Jun 7).
- Tripathi D, Macnicholas R, Kothari C, Sunderraj L, Al-Hilou H, Rangarajan B, et al. Good clinical outcomes following transjugular intrahepatic portosystemic stent-shunts in Budd-Chiari syndrome. *Aliment Pharmacol Ther*. 2014;39:864–72.
- Fu YF, Wei N, Wu Q, Zhang QQ, Cui YF, Xu H. Use of accessory hepatic vein intervention in the treatment of Budd-Chiari syndrome. *Cardiovasc Intervent Radiol*. 2015;. doi:10.1007/s00270-015-1105-4 (Epub 2015 Apr 23).
- 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;77:143–8.
- Mammen T, Keshava S, Eapen CE, Moses V, Babu NR, Kurien G, et al. Intrahepatic collateral recanalization in symptomatic Budd-Chiari syndrome: a single-center experience. *J Vasc Interv Radiol*. 2010;21:1119–24.
- Sang HF, Li XQ. Endovascular treatment of Budd-Chiari syndrome with hepatic vein obstruction in China. *J Laparoendosc Adv Surg Tech A*. 2014;24:846–51.
- Ding PX, Zhang SJ, Li Z, Fu MT, Hua ZH, Zhang WG. Long-term safety and outcome of percutaneous transhepatic venous balloon angioplasty for Budd-Chiari syndrome. *J Gastroenterol Hepatol*. 2015;. doi:10.1111/jgh.13025 (Epub 2015 Jun 23).
- Zahn A, Gotthardt D, Weiss KH, Richter G, Schmidt J, Stremmel W, et al. Budd-Chiari Syndrome: Long term success via hepatic decompression using transjugular intrahepatic porto-systemic shunt. *BMC Gastroenterol*. 2010;10:25.
- Darwish Murad S, Plessier A, Hernandez-Guerra M, Fabris F, Eapen CE, Bahr MJ, et al. Etiology, management, and outcome of the Budd-Chiari syndrome. *Ann Intern Med*. 2009;151:167–75.
- Seijo S, Plessier A, Hoekstra J, Dell'era A, Mandair D, Rifai K, et al. Good long-term outcome of Budd-Chiari syndrome with a step-wise management. *Hepatology*. 2013;57:1962–8.
- Qi X, Guo W, He C, Zhang W, Wu F, Yin Z, et al. Transjugular intrahepatic portosystemic shunt for Budd-Chiari syndrome: techniques, indications and results on 51 Chinese patients from a single centre. *Liver Int*. 2014;34:1164–75.
- 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;55:878–84.
- Cheng D, Xu H, Lu ZJ, Hua R, Qiu H, Du H, Xu X, et al. Clinical features and etiology of Budd-Chiari syndrome in Chinese patients: a single-center study. *J Gastroenterol Hepatol*. 2013;28:1061–7.
- Valla D, Hadengue A, el Younsi M, Azar N, Zeitoun G, Boudet MJ, et al. Hepatic venous outflow block caused by short-length hepatic vein stenoses. *Hepatology*. 1997;25:814–9.