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Hemodialysis-related portosystemic encephalopathy after transjugular intrahepatic portosystemic shunt: A case report and literature review

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Case Report

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Abstract

BACKGROUND

Hemodialysis-related portosystemic encephalopathy (HRPSE) represents a form of dialysis-induced hepatic encephalopathy. We present a rare case of the portosystemic shunt tract being opened by transjugular intrahepatic portosystemic shunt (TIPS) in a cirrhotic patient to alleviate the symptoms of refractory ascites, in which encephalopathy symptoms recurred during the maintenance hemodialysis for this patient's chronic renal failure.

CASE PRESENTATION

A 71-year-old female with cirrhosis and stage G5 chronic kidney disease (CKD) was admitted. A TIPS stent was placed because of portal hypertension with refractory ascites. Due to preexisting CKD requiring maintenance hemodialysis, hemodialysis continued post-TIPS. During the latter half of dialysis sessions at 2 weeks post-TIPS, the patient exhibited recurrent hepatic encephalopathy symptoms, including sluggish responses, incoherent answers, and progressive consciousness impairment. After excluding other neurological disorders, HRPSE was diagnosed. Ammonia-lowering interventions and dialysis prescription modification reduced encephalopathy incidence.

CONCLUSION

In cirrhotic patients with concurrent CKD, TIPS placement requires careful prior evaluation, and clinicians should remain alert for the development of HRPSE following the procedure

INTRODUCTION

Refractory ascites in hepatic cirrhosis constitutes a principal indication for transjugular intrahepatic portosystemic shunt (TIPS) [1–4], Ascites clearance with TIPS is associated with improved quality of life, better nitrogen balance, significant muscle gain, and improved survival[2]. While with concomitant chronic renal failure does not represent an absolute contraindication for TIPS intervention[5, 6], patients with cirrhosis and chronic renal failure may develop hemodialysis-related portosystemic encephalopathy (HRPSE) during hemodialysis. HRPSE is a clinical phenomenon in which portosystemic encephalopathy typically develops because of hemodialysis-induced portosystemic hemodynamic changes. Essentially, HRPSE represents a form of dialysis-induced HE; portosystemic shunts and hemodialysis are key distinguishing features, and most cases involve spontaneous portosystemic shunts (SPSSs) in vivo[7]. TIPS-created shunts constitute an uncommon cause of HRPSE. In this case, we present a 71-year-old female with cirrhosis and stage G5 chronic kidney disease (CKD) who underwent TIPS for alleviating the symptoms of refractory ascites, after which encephalopathy symptoms recurred during maintenance hemodialysis for CKD. This HRPSE diagnosis serves as a reminder to carefully evaluate TIPS indications and contraindications in such patients.

CASE PRESENTATION

History of present illness

A 71-year-old female cirrhotic patient with concomitant CKD stage G5 was admitted due to consciousness disturbance during the latter half of hemodialysis sessions. Two weeks prior, the patient underwent TIPS treatment for refractory ascites secondary to hepatic cirrhosis. Indications and contraindications were rigorously evaluated, confirming ascites etiology as portal hypertension (non-renal/non-hypoalbuminemic). The patient reported significant ascites-related symptoms and strong motivation to improve her condition. Conservative management yielded poor response. Consequently, a TIPS stent was placed via the jugular vein after an extensive discussion with the patient and multidisciplinary interaction with other specialists. During the operation, an 8-mm-diameter TIPS-specific stent-graft system was used to establish a right hepatic vein to right portal vein shunt (Figs. 1 and 2). The portal caval pressure gradient (PCG) was reduced from 20 mmHg to 7.5 mmHg. The patient recovered well post-TIPS and reported significant improvement in ascites symptoms versus pre-TIPS.

Due to preexisting CKD requiring maintenance hemodialysis, thrice-weekly 4-hour sessions continued postoperatively. During the latter half of a hemodialysis session (4 hours, ultrafiltration volumes 2.5L) at 2 weeks post-TIPS, the patient developed sluggish responses, incoherent answers, and progressive consciousness impairment. She was transferred to our emergency department post-dialysis. The patient underwent comprehensive examinations, including emergency cranial magnetic resonance imaging (MRI) and blood ammonia testing. After excluding cerebrovascular accidents and uremic encephalopathy, hepatic encephalopathy (HE) grade 3 was diagnosed based on TIPS history and neurological symptoms. Following treatment with lactulose, rifaximin, and ornithine aspartate, the patient's consciousness gradually recovered the next day, and encephalopathy symptoms resolved. The patient resumed dialysis post-discharge; however, HE symptoms recurred during the latter half of a hemodialysis session (4 hours, ultrafiltration volumes 2.0L) at 1 month post-TIPS.

History of past illness

The patient had a 10-year history of diabetes managed with premixed insulin. Four years ago, oliguria led to a diagnosis of nephrotic syndrome; renal biopsy confirmed diabetic nephropathy with CKD stage G5 (serum creatinine 533 μmol/L, eGFR < 15 mL/min/1.73m²), necessitating thrice-weekly hemodialysis via a radiocephalic arteriovenous fistula (AVF).

The patient also had a > 40-year history of schistosomiasis, resulting in decompensated cirrhosis (present for > 1 year) with ascites and splenomegaly-induced pancytopenia. There was no history of HE, epilepsy, stroke, or brain trauma. During neurological symptom onset, no gastrointestinal bleeding (hematemesis, melena, or hematochezia) or other encephalopathy-precipitating factors (e.g., constipation) were reported.

The patient denied a history of HBV or HCV infection, tuberculosis, or sarcoidosis, as well as right-sided heart failure, constrictive pericarditis, or Budd–Chiari syndrome. Neuroactive medications (sedatives, gabapentinoids, opioids) were not used, and there was no family history of genetic diseases. The patient denied any history of tobacco use or alcohol consumption.

Physical examination

Post-hemodialysis, while experiencing HE symptoms, the patient presented with delirium and consciousness impairment, and was admitted with a low (8/15) Glasgow Coma Scale (GCS) score, and disorientation. Asterixis, hyperreflexia, and hypertonia were detected. The patient had no signs of intracranial infection, including meningeal irritation, increased intracranial pressure, parenchymal damage, or systemic toxicity. Abdominal examination was unremarkable except for ascites, with normoactive bowel sounds and no tenderness, organomegaly, or palpable masses. No signs of peritoneal irritation or abdominal wall abnormalities were detected. The abdominal circumference had decreased by 8 cm compared with pre-TIPS measurements (98 − 90 cm) over 2 weeks. No facial or pedal edema was documented; physical examination revealed no stigmata of chronic liver disease including caput medusae, spider angiomata, or scleral icterus.

Laboratory examinations

During the post-hemodialysis HE episode occurring at 2 weeks post-TIPS, clinical laboratory examination showed the following: venous serum ammonia 71.0 μmol/L (reference 18–72), hemoglobin 8.8 g/dL (11.5–15.0), white blood cell counts (WBC) count 2850/μL (3500–9500), platelet count 93,000/μL (125,000-350,000), albumin 33.0 g/L (35–55), arterial blood gas pH 7.59 (7.35–7.45), pO_2 114 mmHg (80–100), pCO_2 25 mmHg (35–45), HCO_3^- 24 mmol/L (21.4–27.3), Na^+ 137.0 mmol/L (135–145), and K^+ 4.3 mmol/L (3.5–5.1). Liver enzymes, bilirubin, C-reactive protein, serum procalcitonin, prothrombin time, and international normalized ratio (INR) results were normal. There were no obvious abnormalities in hepatitis B virus (five-item) and hepatitis C antibody tests. The Child-Pugh score was 8. Serum ammonia and liver function trends are presented in Table 1.

Table 1
Changes in blood ammonia, potassium and liver functions of the patient.

	Pre-TIPS	Post-TIPS immediately	2 weeks post-TIPS		1 month post-TIPS		HD Adjustment Period	2 months post-TIPS		Reference values
Status			**HRPSE began**	**HRPSE end**	**HRPSE began**	**HRPSE end**	**Symptom-Free Period**	**HRPSE began**	**HRPSE end**	
HD plan	3×/week, 4 h/session						4×/week, 2-3h/session	3×/week, 4 h/session		
Glasgow Coma Scale	15/15	15/15	8/15	15/15	8/15	15/15	-	8/15	15/15	-
Blood ammonia (μmol/L)	-	31	71	29	87	24	-	101	34	18–72
Potassium (mmol/L)	3.9	4.8	4.3	4.3	3.5	4.2	-	3.8	4.2	3.5–5.1
ALT (U/L)	28	23	21	15	42	14	-	37	22	7–40
AST (U/L)	49	44	43	38	86	37	-	78	51	13–35
Serum albumin (g/L)	33	31	33	33	38	35	-	34	31	35–55
Child-Pugh score	8	8	10	7	9	7	-	10	7	

HRPSE: Hemodialysis-related portosystemic encephalopathy; HD: Hemodialysis; ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; TIPS: Transjugular intrahepatic portosystemic shunt.

Imaging examinations

Pre-TIPS contrast-enhanced abdominal computed tomography (CT) demonstrated typical cirrhotic ascites manifestations (Figs. 3), with splenomegaly noted. No signs of esophageal or gastric varices were identified. No signs of portal vein thrombosis were found. Preoperative ultrasound showed no sign of portal vein thrombosis either. Pre-TIPS Gastroscopy showed no significant esophageal or gastric varices. During the post-hemodialysis HE episode occurring at 2 weeks post-TIPS, cranial MRI ruled out cerebrovascular accidents. The MRI results demonstrated a few ischemic foci in the bilateral frontal and parietal lobes; diffusion imaging did not reveal any significant acute infarct foci. Mild demyelinating changes in the white matter of the brain were detected. No evidence of acute hemorrhage or territorial infarction was observed.

Diagnosis and treatment

HRPSE was diagnosed after excluding other neurological causes, based on the temporal link between TIPS, dialysis, and encephalopathy episodes. Considering that this patient's encephalopathy was caused by relatively elevated circulating ammonia levels and hemodynamic changes during dialysis, the management comprised two aspects. 1) Ammonia-lowering therapy with intravenous L-ornithine L-aspartate (20 g/day) acutely, followed by a low-protein diet (1.2–1.5 g/kg/day), rifaximin (600 mg TID), and lactulose (15 mL TID); 2) Dialysis prescription modification, increasing frequency to four sessions weekly while shortening each session.

Outcome and follow-up

The patient was deeply distressed by the recurrent encephalopathy symptoms, so initially she actively cooperated with our treatment. The described interventions reduced the patient’s HE incidence, with no events occurring during a 1-month period. However, the

patient found it difficult to adhere to stringent lifestyle interventions and subsequently reduced the frequency of hemodialysis sessions while increasing the dialysis duration. Subsequently at 2 months post-TIPS, the patient developed recurrent HE episodes that demonstrated a significant association with hemodialysis, with symptom onset predominantly occurring at the end of each session. However, due to economic considerations and the patient's preference, planned TIPS shunt occlusion or reduction was not performed. Unfortunately, the patient succumbed to multiorgan failure at 7 months post-TIPS.

DISCUSSION

Hepatic cirrhosis with refractory ascites is an important indication for TIPS[1–4], with concomitant chronic renal failure not representing an absolute contraindication for TIPS intervention[5, 6]. TIPS is effective in controlling ascites in approximately 80% of patients[2]. HE is a common complication after TIPS surgery, with an incidence rate of approximately 30%–50%[8, 9]. It is noteworthy that the onset of encephalopathy in our case occurred after dialysis. The authors believe that l factors potentially inducing HE appeared during the process of hemodialysis, although the exact mechanisms of hemodialysis-induced encephalopathy are not yet fully understood. Thus we reviewed the relevant literatures and found that Ubara *et al.* [10] proposed the concept of HRPSE in 2004. Since HRPSE is quite rare, Takashima *et al.* [7] summarized 15 cases and proposed five diagnostic criteria: a. The patient is on hemodialysis. b. The patient presents with neuropsychiatric symptoms suspected to be HE. c. Regardless of the presence of chronic liver disease (except for decompensated cirrhosis), laboratory tests do not reveal any severe condition other than hyperammonemic hepatic dysfunction. d. Imaging (ultrasound, CT, MRI, etc.) reveals the presence of a portosystemic shunt > 10 mm in diameter; or occlusion of the shunt tract by surgical ligation or interventional embolization improves hyperammonemia and neuropsychiatric symptoms. e. Other neuropsychiatric disorders are ruled out. Twelve cases of HRPSE, including our case (Table 2) have been reported. Most HRPSE cases involve SPSSs in vivo[7] ; however, in this case, a TIPS-created shunt induced recurrent HE during maintenance hemodialysis for renal failure. Geerinckx *et al.* [11] reported a similar case: a 22-year-old male with congenital hepatic fibrosis and autosomal recessive polycystic kidney disease developed portal hypertension with intractable ascites during long-term cirrhosis management, underwent TIPS, and experienced recurrent HE during postoperative hemodialysis.

Table 2
Cases of hemodialysis-related portosystemic encephalopathy

Case age/sex	Cause of CKD	Dialysis and duration	Shunt	Therapy	Ref.
1 57/M	DN	HD 1 mo.	SMV – LGV-LRV	Surgical ligation	Shimono *et* al[22], 1998
2 68/M	FSGS	HD 1 mo.	LGV – LRV	Surgical ligation	Ubara *et al*[10], 2004
3 75/M	IgAN	PD 15 mo.	LGV – AV – SVC	Surgical ligation	Paraíso *et al*[23], 2007
4 79/F	DN	HD 15 year.	SV – LRV	BRTO	Yasukawa *et al*[18], 2013
5 32/F	CIN	HD 1 year.	Intrahepatic vein	BRTO	Yang *et al*[21], 2013
6 75/F	DN	HD 5 year.	SV – LRV	BRTO	Kondo *et al*[24], 2015
7 55/M	DN	HD 1 year.	SV – LRV	BRTO	Oi *et al*[17]. 2015
8 22/M	ARPKD	HD 7 mo.	TIPS	Relocation dialysis catheter	Geerinckx *et al*[11]. 2019
9 50/M	DN and IRGN	HD 2 mo.	SV – LRV	Vascular plug embolism	Aboobacker *et al*[25]. 2022
10 78/F	DN	HD 1 mo.	SV – LRV	BRTO	Taniguchi *et al*[20], 2023
11 74/F	nephrosclerosis	HD 4 year.	LGV – LRV	Ammonia-lowering intervention and dissolving thrombosis around hemodialysis catheter	Yoshida *et al*[26]. 2025
12 71/F	DN	HD 3 year.	TIPS	Ammonia-lowering interventions and dialysis prescription modification	Our case

CKD: Chronic kidney disease; DN: Diabetic nephropathy; FSGS: Focal segmental glomerulosclerosis; IgAN: Immunoglobulin A nephropathy; CIN: Chronic interstitial nephritis; HD: Hemodialysis; ARPKD: Autosomal recessive polycystic kidney disease; IRGN: Infection related glomerulonephritis; SMV: Superior mesenteric vein; LGV: Left gastric vein; LRV: Left renal vein; AV: Azygos vein; SVC: Superior vena cava; SV: Splenic vein, TIPS: Transjugular intrahepatic portosystemic shunt; BRTO: Balloon-occluded retrograde transvenous obliteration.

Through a literature review, the authors propose a hypothesis for the pathogenesis of HRPSE involving two synergistic pathophysiological mechanisms. First, hemodialysis-induced hemodynamic changes increase the inferior vena cava (IVC)- portal vein (PV) pressure gradient across TIPSs, redistribute liver perfusion, attenuate hepatic function, and promote endotoxemia[12]. As a critical barrier between the portal and systemic circulations, the liver metabolizes gut-derived uremic toxins. Hemodialysis reduces gut blood flow, yet the liver—with its dual circulation—maintains perfusion via portal inflow that is rich in uremic toxins and endotoxins but low in oxygen. This process impairs excretory function and increases systemic toxin translocation[13]. This cascade is exacerbated in patients with portosystemic shunts, whether congenital or iatrogenic. These shunts create low-resistance pathways that divert toxin-laden portal blood directly into the systemic circulation, bypassing hepatic detoxification, increasing intestinal ammonia bioavailability, and ultimately triggering HE episodes. Second, patients with cirrhosis are prone to electrolyte disorders such as hypokalemia[14].

Hemodialysis frequently induces hypokalemic states[15] while concurrently generating a persistent metabolic alkalosis. Such alkalosis is common in hemodialysis patients and persists due to decreased protein catabolic rates and increased dialysis doses[16]. Available evidence indicates that hypokalemia and alkalemia act synergistically to promote hyperammonemia, thereby increasing the risk of HE.

As HRPSE represents hemodialysis-induced encephalopathy, there are many aspects requiring exploration in treatment. We list reported treatment methods and explain the rationale for our approach. First, HRPSE is essentially hemodialysis-induced HE; therefore, blood ammonia-lowering therapy is standard. L-ornithine L-aspartate, rifaximin, and lactulose are recognized treatments for HE. Second, most prior HRPSE reports involve SPSSs, where shunt occlusion (via balloon-occluded retrograde transvenous obliteration, vascular plug embolism or surgical ligation) is effective[10, 17, 18]. However, occlusion of TIPS causing artificial shunt may compromise TIPS' therapeutic effect (though it is an option if patients request it). Reducing TIPS stent diameter represents a theoretical alternative. A recent study confirmed a lower HE incidence (27% vs. 54%) in patients with stents under-dilated to 6 mm compared to 8–10 mm dilation [19]. Therefore, either under-dilation or smaller-diameter stents may reduce the likelihood of post-TIPS HE. Last, dialysis prescription adjustments were used in a few documented cases to address hemodynamic alterations in HRPSE. Its mechanism remains unclear but may involve stabilization of metabolic derangements and reduced IVC-PV pressure gradients. As noted, hemodialysis rapidly clears fluid within 3–4 hours, increasing the IVC-PV pressure gradient and portosystemic shunt flow[10, 12]. Taniguchi *et al* showed that Doppler ultrasound before and after dialysis shows that the blood flow velocity of portosystemic shunt increases due to dialysis[20]. Ammonia-rich portal blood entering systemic circulation may cause neurotoxicity. Additionally, hypokalemia and metabolic alkalosis during dialysis may contribute to increased ammonia levels[15, 16, 21]. Shortening dialysis duration while increasing frequency reduces per-session dehydration, mitigating systemic venous pressure drops and stabilizing hemodynamics/electrolytes. These hypotheses require further validation through clinical trials. Another hemodynamic modification, relocating hemodialysis catheters from the superior vena cava to the femoral vein—achieved symptom resolution in a similar TIPS case[11], However, liver transplantation in that patient precluded long-term efficacy assessment.

CONCLUSION

The coexistence of dialysis-induced metabolic disturbances and shunt-mediated circulatory bypass establishes a self-reinforcing cycle of neurotoxin accumulation, ultimately manifesting as HRPSE. This case, diagnosed as HRPSE, serves as a reminder to more carefully evaluate TIPS indications and contraindications in such patients. This approach is crucial to avoid dialysis-induced HRPSE after TIPS placement.

Declarations

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Authors’ contributions

All authors reviewed and approved the final manuscript and agree to be accountable for all aspects of the work. B.P. and Y.R. wrote the main manuscript text and B.P. prepared tables1-2 and figures 1-3. All authors reviewed the manuscript.

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Data availability

This study is a case report. The data utilized primarily consist of the de-identified clinical records, radiological images, and laboratory test results of the patient. All materials supporting the findings of this study have been included within the manuscript. The raw data involving patient privacy are safeguarded by legal and ethical regulations and will not be publicly shared. However, they may be made available upon reasonable request and with the approval of the relevant Ethics Committee.

Ethics approval and consent to participate

Ethical approval for this case report was obtained from the Institutional Review Board of Chengdu Second People's Hospital. This study complies with the ethical standards of the Declaration of Helsinki. In accordance with ethical requirements and the protection of minors, written informed consent was obtained from both the patient and his legal guardian prior to submission.

Clinical trial number

Not applicable. This is a single case report and did not involve a clinical trial. Therefore, a clinical trial number is not applicable.

Consent for publication

Patient’s guardian gave written informed consent for their personal or clinical details along with any identifying images to be published in this study.

Competing interests

The authors declare no competing interests.

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Figures

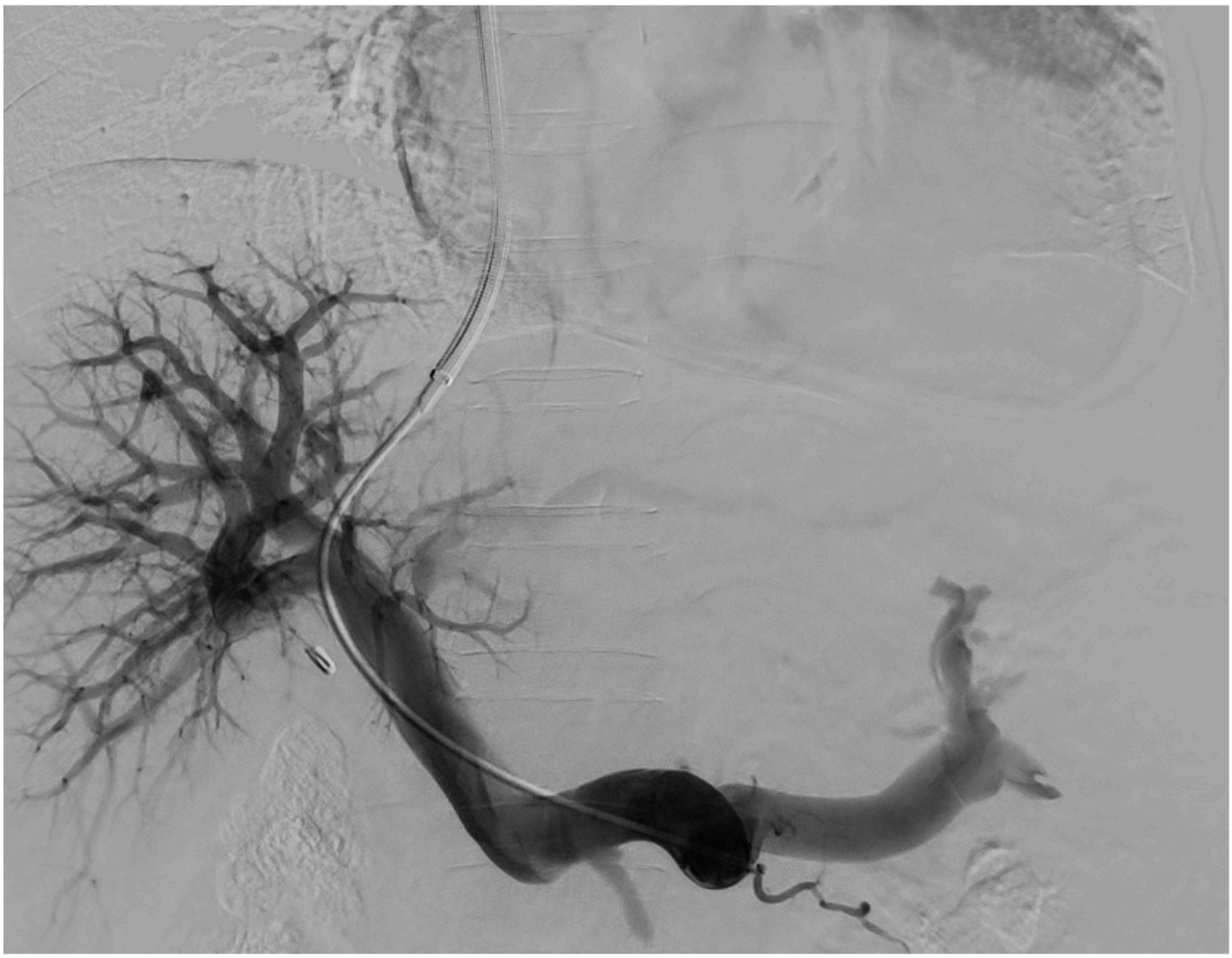


Figure 1

Establishment of right hepatic vein to right portal vein shunt after puncture of right portal vein branch during the transjugular intrahepatic portosystemic shunt procedure.

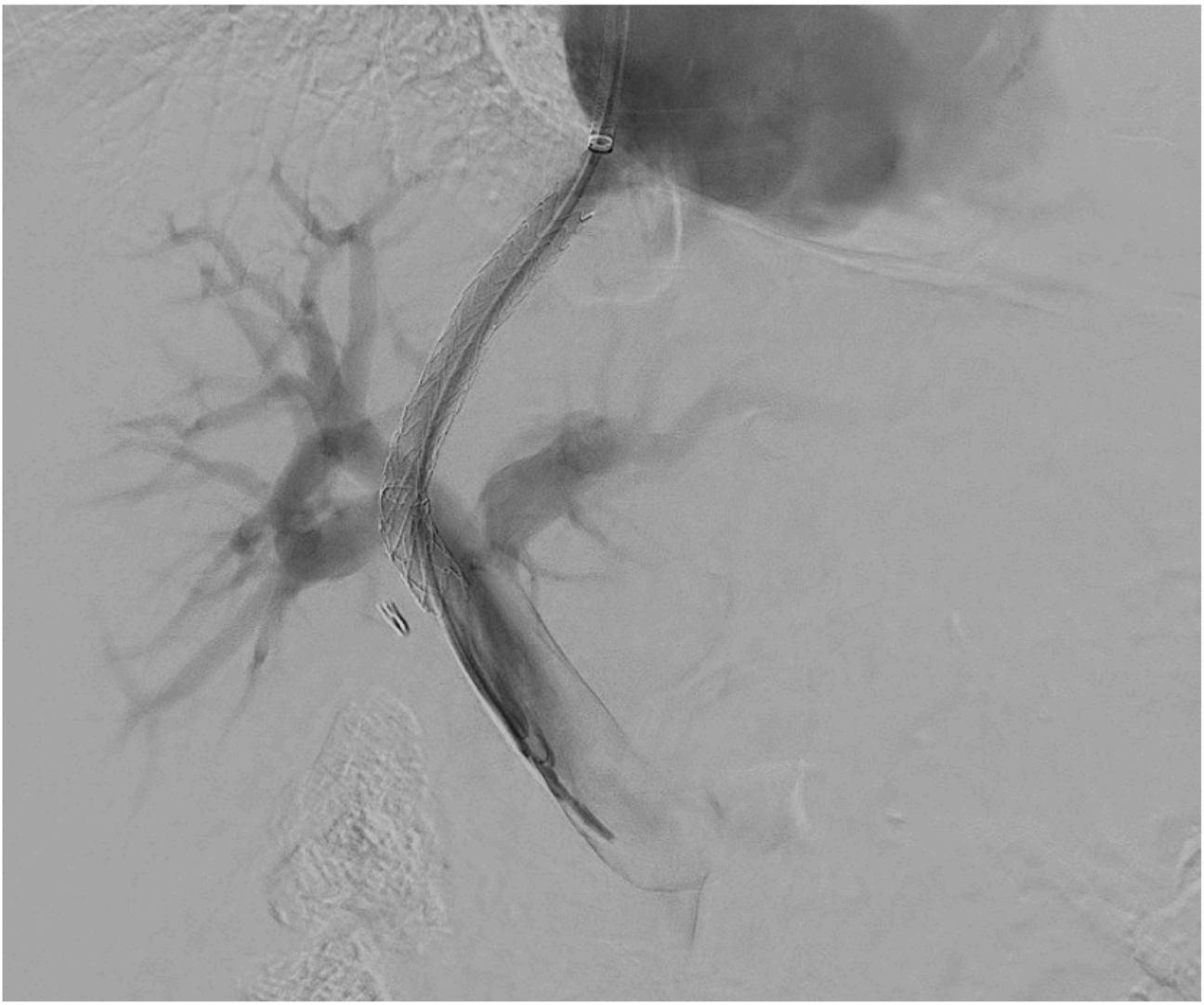


Figure 2

An 8-mm-diameter transjugular intrahepatic portosystemic shunt-specific stent-graft system was placed.

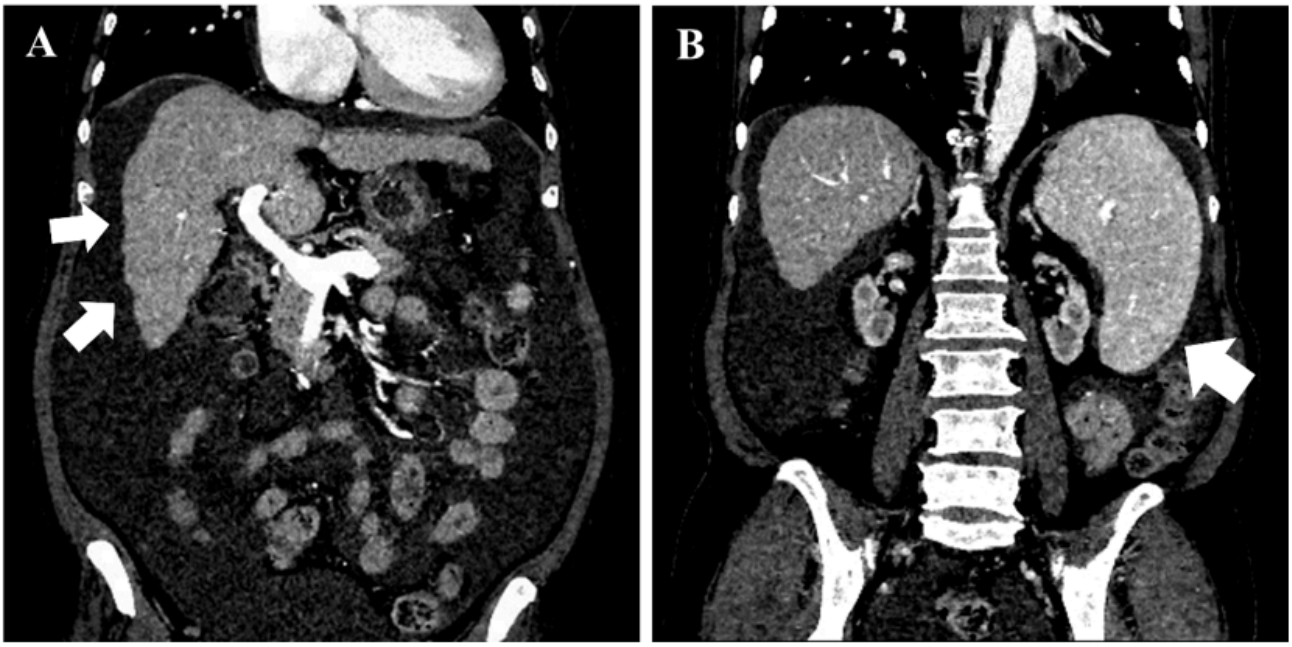


Figure 3

Pre-transjugular intrahepatic portosystemic shunt contrast-enhanced abdominal computed tomography of the patient. A: Computed tomography (CT) indicated typical signs of hepatic cirrhosis (arrow) and ascites. No signs of esophageal or gastric varices were identified. No signs of portal vein thrombosis were found. B: CT showing the splenomegaly (arrow).