

Acute TIPS occlusion due to iatrogenic arteriovenous shunt in a cirrhotic patient with total portal vein thrombosis

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Abstract: A 69-year-old man with portal hypertension was admitted with decompensated alcoholic cirrhosis and diuretic resistant ascites. Ultrasound revealed partial portal thrombosis. Due to diuretic intolerance, transjugular intrahepatic portosystemic shunt (TIPS) was decided during which a hepatic arterial branch was inadvertently catheterized. Finally, TIPS was created, but the patient continued gaining weight. Color-Doppler ultrasonography (CDUS) showed upper stent part patency with absence of flow in lower stent portion. Twenty-five days later, the patient presented melena. Endoscopy revealed blood emerging from the Vater papilla. Hepatic angiography revealed arteriovenous shunt between a hepatic arterial branch and the proximal part of the TIPS shunt. Covered stent placement restored sufficient TIPS flow. The patient deteriorated and died 1 month later. We found out that our major technical drawback was that we did not inject a small amount of contrast after puncturing the supposed portal vein, in order to confirm correct position of the needle.

Keywords: TIPS, complications, arteriovenous shunt, portal vein thrombosis, interventional radiology

Introduction

Transjugular intrahepatic portosystemic shunt (TIPS) is indicated for portal hypertension decompression and ascites management in selected patients, if standard medical therapy fails [1–3]. Total portal vein occlusion is considered as a relative contraindication for TIPS, but the procedure is potentially possible [2, 4]. During the intrahepatic punctures, accidental punctures of hepatic arterial branches are usually without sequelae, but they may induce major complications like intra-abdominal hemorrhage or hemobilia, which might necessitate re-intervention for intravascular treatment [2, 5]. Additionally, TIPS occlusion due to stent thrombosis is reported [2, 3]. Acute or late TIPS occlusion causes include thrombophilia, biliovenous shunts, or mechanical dysfunction with slow blood flow, causing subsequent stent

thrombosis [6, 7]. This can be percutaneously managed by balloon dilatation and mechanical thrombectomy [6, 7]. We report a case of TIPS occlusion, due to an iatrogenic arterial-venous shunt, which kept the upper stent part patent but caused thrombosis of the lower stent portion, and also present the management of this complication.

Case Report

A 69-year-old man with a history of diabetes mellitus, minor beta thalassemia, and alcoholic cirrhosis was admitted with abdominal distention and lower extremity oedema. Clinical examination revealed signs of chronic liver disease and portal hypertension. Viral and autoimmune markers were negative. He had grade III ascites

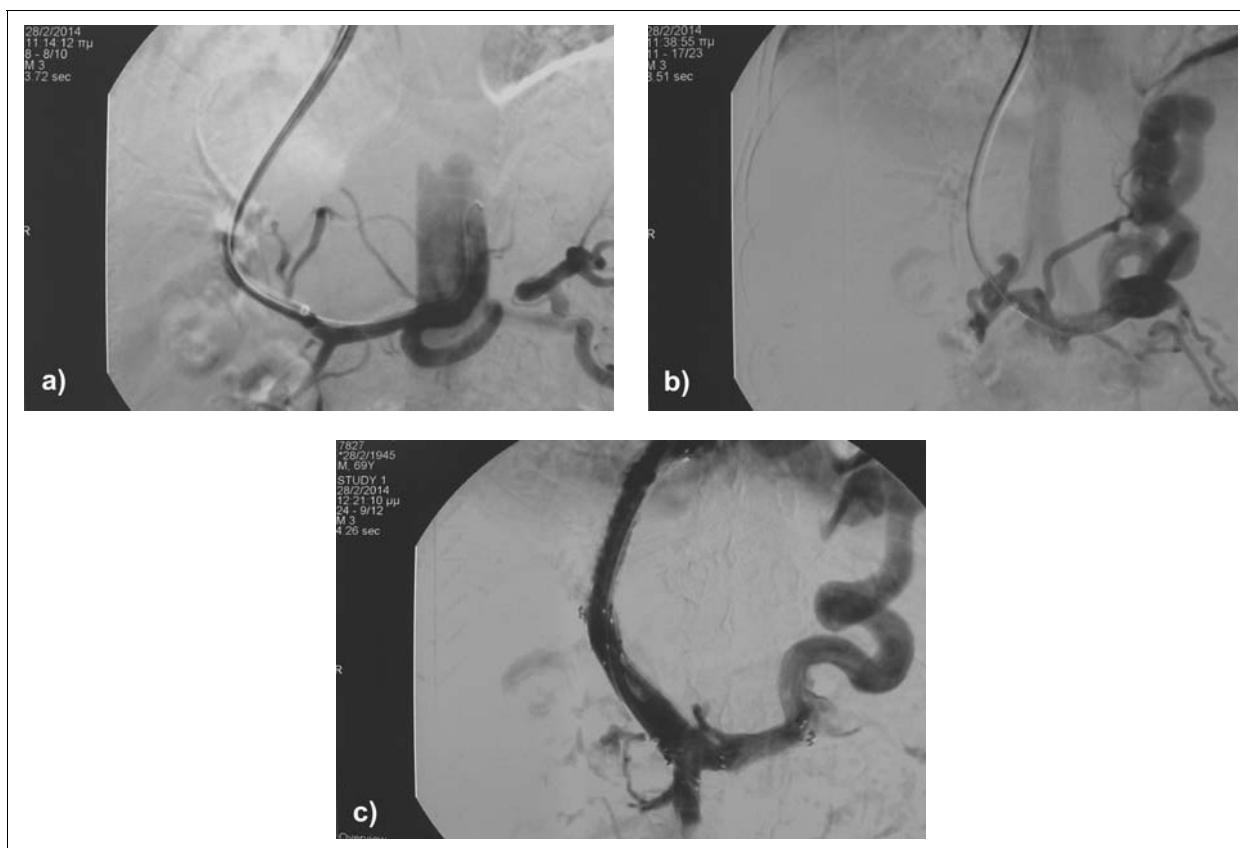


Fig. 1. Initial TIPS procedure in the thrombosed portal system. (a) Angiographic imaging through the 9 Fr TIPS sheath accidentally advanced into the enlarged common hepatic artery. Notice that the catheter and guidewire direction is similar to that of the portal vein course. (b) Angiographic imaging through the 9 Fr TIPS sheath after correct placement into the splenic vein. Large splenic varices and splenorenal shunt are seen on delayed images. Complete thrombosis of the main portal vein and the intrahepatic branches distal to the superior mesenteric junction is seen. (c) Angiographic imaging through the 9 Fr TIPS sheath after TIPS creation with three overlapping stents deployed, one intrahepatically, one in the main portal, and one in the splenic vein. Sufficient flow towards the right atrium is seen

without evidence of spontaneous bacterial peritonitis. Upper GI endoscopy demonstrated grade II esophageal varices and portal hypertensive gastropathy. The clinical diagnosis was decompensated alcoholic cirrhosis.

The patient was commenced on diuretics and propranolol with initial response to treatment. However, a few days after his first discharge, the patient was readmitted due to syncope, acute renal failure, and hyperkalemia and was successfully resuscitated. During the following months, he was hospitalized three additional times for grade I hepatic encephalopathy in the context of spontaneous bacterial peritonitis, for renal impairment (diuretic induced) and for cellulitis. Upper abdominal color-Doppler ultrasonography (CDUS) revealed cirrhotic liver appearance, massive ascites, and partial portal vein thrombosis. Topographic relation of right hepatic vein to portal bifurcation was convenient for TIPS. One month after sonography, a TIPS procedure was decided due to diuretic intolerance, resistant ascites, and ineffective large volume paracenteses. At this time, his Child-Pugh was B and the MELD score was 13.

Right internal jugular puncture with catheterization of the right hepatic vein was easily accomplished. Multiple unsuccessful intrahepatic punctures with a Roesch-Uchida needle (Transjugular Liver Access Set, Cook Medical, Bjaeverskov, Denmark) were attempted. Twice, the left biliary duct system was opacified. Finally, a blood vessel was punctured and the guidewire was advanced in the direction of the portal vein system. The catheter-sheath system was advanced over the wire, but although the sheath's direction was similar to the portal system direction, angiography showed that the sheath was in the right hepatic artery (*Fig. 1a*). Subsequently, sheath was retrieved, and after several attempts, the guidewire was advanced in a position which proved to be the fully thrombosed portal vein (*Fig. 1b*). Angiographic imaging also showed large splenic varices with a splenorenal shunt on delayed images. Complete thrombosis of the intrahepatic portal branches and the main portal vein distal to the superior mesenteric junction, probably occurred during the last 4 weeks between CDUS and TIPS. We decided to stent the entire portal vein and create a TIPS

shunt between the splenic-superior mesenteric vein junction. We thought that a covered stent in the main portal vein would be more suitable for compressing thrombus against the vessel wall. Unfortunately, at that time, only one covered stent was available in our unit. In total, three stents were used for this purpose, including one covered stent of 13.5 × 80 mm in the main portal vein (Fluency, Bard peripheral vascular, Tempe, AZ, USA), one bare 10 × 80 mm for stenting of the intrahepatic tract (eLuminexx, Bard peripheral vascular, Tempe, AZ, USA), and another same type bare stent of 10 × 60 mm in the distal splenic vein for straightening of the portal stent towards the splenic axis. Five thousand IU of hepa-

rin was intravenously administered. Angiographic imaging after TIPS creation showed sufficient flow towards the right atrium (*Fig. 1c*).

To our disappointment, the patient did not improve clinically and repeated large volume paracenteses were still required. Three days after TIPS, a CDUS showed patent stent with sufficient intraluminal blood flow. Twenty-five days after TIPS, the patient presented with melena appeared and an emergency upper GI endoscopy revealed blood in the second duodenal segment, emerging from the ampula of Vater.

The patient underwent hepatic angiography to identify the bleeding source. Hepatic angiography re-



Fig. 2. TIPS revision and arteriovenous shunt cessation. (a) Angiographic imaging through a 5 Fr Cobra catheter placed in the right hepatic artery reveals extravasation of the contrast medium through a hepatic tract towards the distal portion of the TIPS-stent in the form of arteriovenous shunt. The intrahepatic tract corresponds to the path of the mistakenly placed sheath during TIPS creation. The middle and distal stent portion proved to be thrombosed. (b) Angiographic imaging through a 5 Fr catheter after TIPS revision and placement of a covered stent over the intrahepatic tract. Blood flow is established from the superior mesenteric vein towards the TIPS and the right atrium but not from the splenic vein. (c) Angiographic imaging through a 5 Fr Cobra catheter placed in the right hepatic artery shows occlusion of the arteriovenous shunt

vealed an arteriovenous shunt between a hepatic arterial branch and the upper part of the TIPS shunt, which was still patent (*Fig. 2a*). Probably, this was the part of the TIPS stent diagnosed as patent in CDUS. The lower part of the TIPS was considered thrombosed, and new catheterization for TIPS revision followed. This was easily performed revealing lower shunt thrombosis and subsequent thrombosis of the main portal vein stent. An Angiojet rheolytic thrombectomy catheter (DVX type, Possis Medical, USA) was advanced over the wire, and multiple passes of this device into the thrombus were performed using a standard catheter aspiration thrombectomy technique without injection of any thrombolytic agent. During each pass, the probe was slowly moved in and out, more than 30 s each time. Adjunctive thrombus disruption with an inflated 10 × 40 mm PTA balloon (Juturna-V, TsunaMED, Germany) was performed due to residual thrombus. Additional dilatation of the stents with the same balloon followed, providing complete stent patency. Additionally, 5.000 IU of heparin was intravenously administered. Finally, two covered stents (Fluency, Bard peripheral vascular, Tempe, AZ, USA) were placed, one 12 × 80 mm in the intrahepatic part of the TIPS shunt, so that the arteriovenous shunt could be sealed and another one 12 × 60 mm in the portal vein, inside the previously placed fluency stent. Total portal vein thrombosis precluded hepatic arterial branch embolization. The final result was sufficient with blood flow established from the superior mesenteric vein towards the TIPS and the right atrium but not from the splenic vein (*Fig. 2b*). Repeat hepatic angiography revealed the immediate cessation of the arteriovenous shunt (*Fig. 2c*).

Post procedure, the patient developed macroscopic hematuria without hemodynamic instability that gradually settled, due to the mechanical thrombus manipulation. It has been our experience that use of the Angiojet catheter is occasionally followed by macroscopic hematuria, as a result of non-immune-mediated intravascular hemolysis. Low-molecular heparin was readministered within 12 h, and repeated US-Doppler scans confirmed patency during the next days and weeks. Despite the good angiographic result, the patient became profoundly anemic during the following days. Laboratory studies revealed a non-immune-mediated hemolytic anemia with a negative direct Coombs reaction test, a decreased haptoglobin level, and elevated levels of LDH, findings that were attributed to mechanical hemolysis (acanthocytes identified on the peripheral blood smear). He gradually deteriorated presenting excessive edema and worsening liver function, and he was further complicated by staphylococcal septicemia followed by multi-organ failure and succumbed 1 month later. Until the end of his life, TIPS remained patent in color Doppler examinations.

Discussion

Liver cirrhosis is frequently complicated by significant portal hepatic hypertension, and concomitant portal vein thrombosis may worsen patient's condition and increase mortality rate [8]. Medical treatment represents the initial approach, but if the patient deteriorates, decompressive portosystemic shunting or liver transplantation may be considered [3].

TIPS is nowadays an established percutaneous intervention for creation of artificial communication between portal venous system and the systemic circulation for the management of portal hypertension complications [1–3]. It avoids major surgical shunt operation and is successfully applied in critically ill patients irrespective of inferior vena cava obstruction [9].

Retrograde puncture of the liver is usually achieved through a large hepatic vein, aiming a central portal branch. Creating TIPS in a patient with partial portal vein thrombosis can be a demanding procedure. Technical precautions can be made, so such complications shall be avoided. For example, wedged carbon dioxide portography or wedged portography using contrast media can be performed to delineate the portal vein itself. Also, after puncture of the supposed portal vein, a small amount of contrast media should be injected via the puncture needle to confirm the puncture of a portal branch.

In patients with total portal vein occlusion, usually due to hemodynamic factors [3], TIPS, although potentially possible, cannot be easily applied and is considered as a relative contraindication [2, 4]. Successful insertion of TIPS not only recanalizes the thrombosed portal vein but also relieves symptomatic portal hypertension. However, the technical difficulty of TIPS potentially limits its widespread application, and the risk and benefits should be fully balanced [4].

Accidental puncture of the biliary system or a hepatic arterial branch during the intrahepatic passages is usually without clinical consequences. Nevertheless, occasionally, it could induce major iatrogenic complications like intra-abdominal hemorrhage or hemobilia which might need immediate interventions for intravascular treatment [2, 5, 6].

TIPS was associated in the past with a high reintervention rate of 88–96% due to stenosis or occlusion of the stent [6]. Primary shunt patency rates are found 45.4% and 26.0% at 1 and 2 years, respectively, while the overall secondary assisted patency rate can reach 72.2% [2]. With use of new stents with ePTFE-FEP coverage, the need for reintervention has strongly decreased, but unfortunately, such stents were not available in our institution at that time. Acute or late causes for TIPS occlusion might be thrombophilia, biliovenous shunts, or mechanical dysfunction with slow blood flow, causing subsequent stent thrombosis, complications which

should be managed by means of balloon dilatation and percutaneous mechanical thrombectomy [6, 7, 10].

In unselected patients, post-TIPS, overall mortality rate can be as high as 60%, largely resulting from liver failure, so that careful patient selection is very important [2]. Procedure-related mortality, according to Petersen et al., is about 2%, due to 30 different fatal complications [5]. One such fatal case was due to inadvertent arterial puncture with placement of a 9 Fr sheath inside the hepatic artery that was believed to be a high-flow portal vein branch. In our case, the location of the guide wire was thought to be in the portal vein system, so we decided to advance the catheter–sheath system without any contrast injection. In Petersen's complication, the patient developed tense abdominal distention with severe hypotension and bleeding was controlled by means of endovascular coil embolization of the entire right hepatic artery. After successful embolization and TIPS procedure, the patient died 7 days later due to hepatic infarction and ARDS [5]. The author's conclusion was that patients with limited hepatic reserve and portal blood flow might not survive arterial embolization. Perhaps, a more distal targeted arterial branch embolization could prevent patient's death. In our patient's case, we decided not to embolize the arterial branch associated with the arterial-venous shunt and closed with placement of a covered stent, a solution that was judged safer. Despite the effective angiographic management, our patient deteriorated clinically due to sepsis and worsening liver failure and died.

Conclusions

Technical precautions during TIPS procedure can be made, so that such complications can be avoided. We found out that our major technical drawback was that we did not inject a small amount of contrast after puncturing the supposed portal vein, in order to confirm correct position of the needle. Despite that, various rare complications related to TIPS placement, such as A-V shunt in this case, can be managed by means of interventional radiology. Appropriate preprocedural patient selection

and early postprocedural recognition of the potential complications are critical for the outcome.

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Conflict of interest: None.

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