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Case Report

Acute liver failure and infarction complicating TIPS placement

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ABSTRACT

Here in we report a case of acute liver failure with hepatic infarction after transjugular intrahepatic portosystemic shunt (TIPS). An upper gastrointestinal hemorrhage patient with a medical history of alcoholic cirrhosis underwent a TIPS procedure. One day after TIPS, his alanine aminotransferase and aspartate aminotransferase levels increased to 1214 U/L and 1511 U/L, respectively. Two days after TIPS, they peaked at alanine aminotransferase 8389 U/L and aspartate aminotransferase >7500 U/L, respectively. An emergent stent occlusion was performed on the second day. Portography showed that there were no portal vein branches or parenchymal stains on the edge of the right liver lobe. A CT scan demonstrated diffuse hepatic parenchyma, homogeneous hypodense lesion, and bilateral pleural effusion. The patient died of liver failure and multiple organ dysfunction syndrome 6 hours after the stent occlusion.

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Introduction

Transjugular intrahepatic portosystemic shunt (TIPS) has been widely employed for the management of recurrent or refractory variceal bleeding and refractory ascites. Its use has also been advocated for hepatic hydrothorax, hepatorenal syndromes, portal vein thrombosis, and portal hypertensive gastropathy [1,2]. Various complications, such as abdominal hemorrhage, encephalopathy, and TIPS dysfunction, can occur after TIPS placement.

Hepatic infarction combined with acute liver failure post-TIPS is a rare but severe complication with a poor progn-

sis. TIPS-induced liver failure was frequently reported in the previous literature [3]. But very few cases had been proven to be liver infarction. We describe herein a case of TIPS-induced acute liver failure and hepatic infarction that was identified using computer tomography (CT) and digital subtraction angiography (DSA).

Case report

A 69-year-old male with a medical history of alcoholic cirrhosis presented with repeated hematemesis and melena. After

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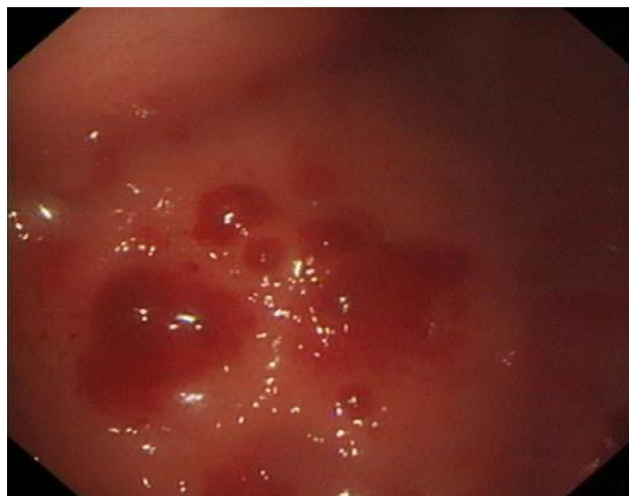


Fig. 1 – An endoscopy revealed multiple submucosal hemorrhages at the gastric antrum pre-TIPS.



Fig. 2 – During the percutaneous transhepatic portal vein cannulation-assisted TIPS procedure, portography showed the dense portal vein branches.

Table 1 – Hepatic function and coagulation values pre-TIPS and post-TIPS.

	2 days pre-TIPS	1 days post-TIPS	2 days post-TIPS
ALT (u/L)	18	1214	8389
AST (u/L)	25	1511	>7500
TBIL (μ mol/L)	10.2	36.7	79.9
PT (s)	12.8	20.8	54.8
INR	1.17	1.94	5.31
APTT (s)	29.5	29.8	48.7
DD (mg/L)	1.78	21.47	>40
FIB (mg/dL)	169	148	<40

he was admitted, an endoscopy examination found gastroesophageal varices and diffuse gastric hemorrhage due to portal hypertensive gastropathy (Fig. 1). An unsuccessful hepatic venous pressure gradient measurement attributed to incomplete occlusion of the hepatic vein during wedged hepatic venous pressure measurement had a value of 11 mm Hg. An abdominal enhanced CT scan showed cirrhosis, portal hypertension, portal venous collateral pathway establishment, hypersplenotrophy, and bilateral pleural effusion. The Child-Pugh score was 8. Laboratory evaluation of liver function and blood coagulate function is shown in Table 1.

The TIPS procedure was performed under local anesthesia. The right branch of the portal vein was punctured with an ultrasound-guided 21G Chiba needle. A 4F pigtail catheter was introduced into the superior mesenteric vein via a 6F sheath. Portography was performed, and the port vein pressure was 22 mm Hg. Then a RUPS-100 liver access set was inserted via the right jugular vein into the right hepatic vein; the right atrium pressure was 2 mm Hg. Access to the right branch of the portal vein was gained at the first pass guided by the pigtail catheter, and a shunt was created with 2 overlapping stents (bare stent: 8 mm \times 10 cm Bard E-Luminexx; covered stent: 8 mm \times 5 cm GORE Viabahn). The portal vein and right atrium

pressure changed to 14 mm Hg and 6 mm Hg, respectively. Finally, a Nester coil (3 mm \times 14 cm Cook Medical) was used to block the liver puncture passage (Fig. 2).

Seven hours after the procedure, the patient developed vomiting after eating, and his stomach contents were vomitus. One day after the procedure, laboratory results showed evidence of acute liver failure and severe coagulopathy. Alanine aminotransferase (ALT) and aspartate aminotransferase (AST) increased to 1214 U/L and 1511 U/L, respectively. Total bilirubin increased to 36.7 μ mol/L. The prothrombin time (PT) was 20.8 seconds, international normalized ratio was 1.94, activated partial thromboplastin time was 29.8 seconds, D-dimer was 21.47 mg/L, and fibrinogen was 148 mg/dl. An abdominal-enhanced CT was performed on the second day after TIPS demonstrating diffuse hepatic parenchyma and homogeneous hypodense lesion. In the meantime, although the patient underwent liver protection, plasma transfusion, and hemostasis therapy, his liver function and coagulation continually deteriorated as follows: ALT = 8389U/L, AST > 7500 U/L, Total bilirubin = 79.9 μ mol/L, PT = 54.8 seconds, international normalized ratio = 5.31, activated partial thromboplastin time = 48.7 seconds, D-dimer > 40 mg/L, and fibrinogen < 40 mg/dl.

An emergent stent occlusion was performed immediately after a blood test on the second day. Urgent occlusion of the intrahepatic shunt was achieved by inflation of a Fogarty balloon that was introduced through the right internal jugular vein. The balloon was kept inflated. The pressure in the the portal pressure and right atrium were 14 mm Hg and 10 mm Hg, respectively, before stent occlusion, and were 7 mm Hg and 26 mm Hg, respectively, after stent occlusion. Portography showed that there were no portal vein branches or parenchymal stains on the edge of the right liver lobe (Figs. 3 and 4). Despite active treatment, the patient died of liver failure and multiple organ dysfunction syndrome 6 hours after stent occlusion.

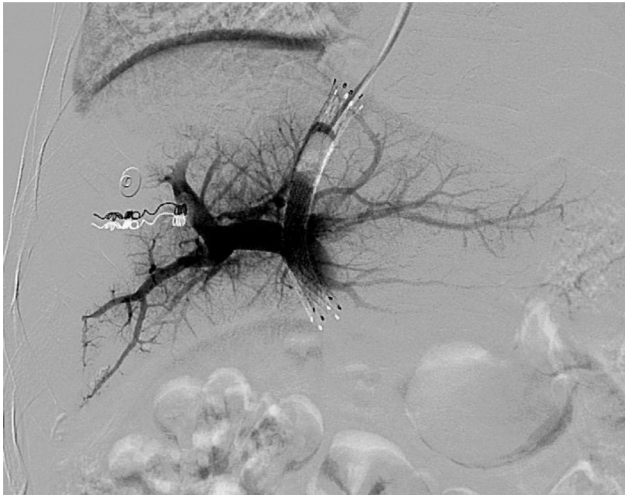


Fig. 3 – After stent occlusion, portography showed that there were no portal vein branches or parenchymal stains on the edge of the right liver lobe.



Fig. 4 – A CT scan of a patient who presented with acute liver failure demonstrated hepatic diffuse parenchyma, homogeneous hypodense lesions, and bilateral pleural effusion.

Discussion

Liver infarction rarely occurs in the normal population. It occurs only during various hepatic artery occlusions, such as surgical ligature, bland embolism, local lesion of the small vessels in the liver, and thrombosis [4]. However, for patients with portal hypertension who undergo TIPS, a diversion of portal flow and insufficient compensation of the hepatic arterial blood flow may lead to hepatic ischemia, which can induce liver failure and even liver infarction.

This patient differed from those reported in the literature. Hepatic transaminase increased rapidly and extremely after TIPS. ALT and AST reached peaks of 8389 U/L and >7500 U/L,

respectively. PT was prolonged to 54.8 seconds. A CT scan demonstrated a homogeneous region of low attenuation in the hepatic parenchyma, the branch of the portal vein and hepatic vein reduction, and bubbles appeared in the liver parenchyma. The exact evidence of hepatic infarction was provided by DSA after the occluded stent, which showed that the fourth-grade branches and distal of the right portal vein had disappeared, and there was a band-shaped unstained liver parenchyma around the right lobe of the liver. The DSA findings of a massive band-shaped liver infarction have not been described previously. Wedge-shaped liver infarctions are commonly reported in the literature [5–10]. Liver infarction is usually evaluated by ultrasonography and CT but rarely by DSA. DSA examination can uncover the degree of liver infarction. Patients with TIPS-induced liver failure, especially those with extremely high ALT and AST values, should undergo DSA. For post-TIPS patients, liver failure may indicate a liver infarction.

The pathogenesis of acute liver failure and hepatic infarction post-TIPS includes occlusion of the hepatic artery, hepatic vein, or low portal pressure gradient [11]. Most cases reported in the literature were induced by hepatic artery injury. Liver sinusoidal perfusion in cirrhotic patients was significantly reduced compared to healthy patients [12]. Therefore, hepatic artery perfusion is very important in patients with cirrhosis who undergo TIPS [13]. Hepatic artery injury during TIPS can lead to partial liver parenchyma; ischemia, and even infarction. Furthermore, in a minority of patients, the covered stent can partially or completely occlude the hepatic venous outflow. Some of these patients may develop hepatic venous thrombosis, which results in liver parenchymal congestion, hypoxia, and infarction [7,10]. The reduction of portal liver perfusion post-TIPS may not be compensated sufficiently by the increased hepatic arterial perfusion. In addition, the artery flow is diverted away from periportal areas with high resistance to hepatic parenchyma. It can also lead to liver parenchymal ischemia and infarction.

In the present case, a post-TIPS CT scan showed no evidence of hepatic artery injury and arteriovenous fistula. The covered stent was implanted in the right hepatic vein and did not block its outflow. Nevertheless, before stent occlusion, the right atrium pressure and portal pressure was 10 mm Hg and 14 mm Hg, respectively. The portosystemic pressure gradient was 4 mm Hg, which can be diagnosed as a medically uncontrolled low pressure gradient that may lead to the deterioration of hepatic function [14]. It is likely the main cause of liver infarction. In our patient, the portal vein pressure and right atrial pressure immediately after TIPS were 14 mm Hg and 6 mm Hg, respectively, different from 2 days after TIPS. It is notable that the portal pressure gradient 2 days post-TIPS was not in accordance with the pressure immediately post-TIPS.

Emergent stent occlusion and reducing stent implantation can reduce the mortality of post-TIPS acute liver failure [15,16]. In this case, stent occlusion did not prevent liver failure and subsequent mortality. It is possible that an earlier stent occlusion would have led to a different outcome.

In conclusion, acute liver failure with hepatic infarction post-TIPS is a rare but potentially devastating complication. DSA demonstrating a lack of band-shaped vein branches and a parenchymal stain area on the edge of the liver lobe was a

definitive sign of low portal pressure gradient-induced liver infarction. Patients with acute liver failure post-TIPS should be monitored closely and intervened earlier to prevent fatal liver infarction.

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