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CASE REPORT

Acute Liver Failure After a Late TIPSS Revision

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Abstract We report a rare case of late transjugular intrahepatic portosystemic stent shunt (TIPSS) occlusion due to progressive stent protrusion into the periportal liver parenchyma, which was a result of delayed liver shrinkage 2 years after TIPSS. The initial TIPSS procedure had been carried out in a 52-year-old man as a bridge for liver transplantation because of post-alcoholic liver cirrhosis. We describe the applied TIPSS recanalization and revision technique. Immediately after TIPSS revision acute liver failure developed, which required emergency liver transplantation.

Keywords Liver failure · Liver transplantation · Portal hypertension · Stent · Transjugular intrahepatic portosystemic stent shunt · TIPSS

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Introduction

The minimally invasive transjugular intrahepatic portosystemic stent shunt (TIPSS) is a well-accepted method in the management of liver cirrhosis-associated variceal bleeding, ascites, and Budd-Chiari syndrome [1, 2]. Despite its therapeutic success, TIPSS can be associated with clinical and technical complications including mechanical stent-specific problems such as stent fracture, protrusion, and migration [3]. Even after a technically successful TIPSS procedure, up to 33–50% patients still require percutaneous interventions for shunt malfunction [3–6].

A clinically relevant worsening of liver function after TIPSS procedure or revision has been reported infrequently [6–9]. In TIPSS revision, to the best of our knowledge, such an emergency has not previously been reported. If TIPSS-associated liver failure occurs, the morbidity and mortality of this complication are very high: 95% of these patients die or require liver transplantation within 90 days after TIPSS creation [10, 11].

Case Report

The 52-year-old man presented here had been listed for liver transplantation since January 2004 because of post-alcoholic liver cirrhosis Child B complicated by therapy-refractory ascites (Fig. 1) but without variceal bleeding episodes.

A TIPSS procedure was performed in October 2004 using the standard technique [1, 8]. The initial portosystemic gradient measured 25 mmHg and decreased after implantation of a 10/60 mm Palmaz Genesis stent (Cordis Europe, The Netherlands) to 8 mmHg (Fig. 2).

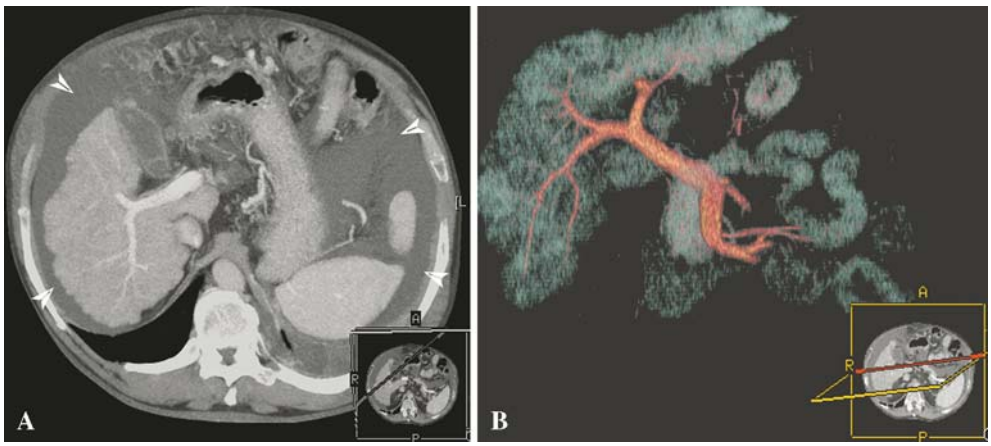

Fig. 1 A, B. Before TIPSS placement in 2004, the preinterventional baseline CT scan (venous phase) showed typical signs of liver cirrhosis with ascites (arrowheads), relative atrophy of the right lobe, and a nodular liver contour. We found a technically simple anatomic situation with a prominent right portal vein. (**A**) MIP projection created from axial contrast-enhanced venous slices and (**B**) a three-dimensional volume-rendered reconstruction

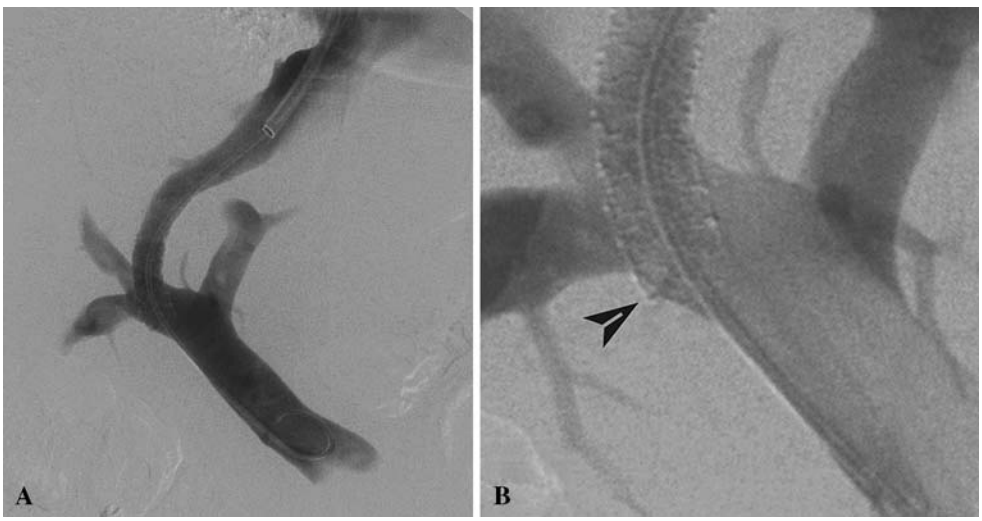

Fig. 2 (**A**) Control angiography of the portal vein after TIPSS placement in October 2004 (10/60 mm Genesis stent) with a final portosystemic gradient of 8 mmHg. (**B**) The caudal portion of the stent dug into the portal vein wall slightly (arrowhead)

Postinterventional total heparinization for 48 hr was carried out. Liver-specific laboratory parameters remained normal after TIPSS (Table 1) and ascites decreased shortly after TIPSS creation.

In January 2005, the patient reported increasing abdominal width. Instead of scheduled routine invasive TIPSS controls in our institution, which he refused, ultrasonography was performed in a peripheral hospital over a period of 2 years, which was always considered normal.

In September 2006, the patient—still listed for liver transplantation and showing normal liver-specific laboratory tests (Table 1)—was referred to our institution for an emergency umbilical hernia operation. Because the periumbilical skin was so malperfused, complete excision of the navel had to be performed. During postoperative follow-up, ascites was detected and punctured daily. Ultrasound findings in our institution were interpreted as a stenosis of the TIPSS. Invasive portography was performed using a transjugular approach. Despite the easy introduction of a 7 Fr guiding catheter (Angiomed Bard, Germany) and a 5 Fr vertebralis catheter (Terumo Europe, Belgium) into the TIPSS outflow tract, it was not possible to catheterize the proximal TIPSS and reach the portal vein. Pushing the catheter tip against the proximal portion of the stent, portography was carried out (Fig. 3A).This revealed almost complete TIPSS tract occlusion without regular venous outflow. The proximal stent portion was protruding out of the lacerated portal vessel wall into the periportal liver parenchyma. However, contrast extravasation was not present around the stent (Fig. 3A).

Table 1 Serum blood parameters during follow-up

Parameter	Normal range of parameters	Day of TIPSS Oct 24, 2004	3 days after TIPSS Oct 27, 2004	5 days after TIPSS Oct 29, 2004	1 day before herniotomy Sept 22, 2006	6 days after herniotomy Sept 28, 2006	Day of TIPSS-recanalization Oct 2, 2006	1 days after TIPSS-recanalization Oct 3, 2006	Before LTX Oct 4, 2006	2 days after LTX Oct 6, 2006	2 months after LTX Dec 2006
Albumin	30–50 g/dl	35.4	34.7	33.4	41.9	38.5	37.2	36.5	35.4	35.6	33
AP	40–130 U/l	134	118	11.5	93	78	87	110	131	105	413
Bilirubin	≤1 mg/dl	1.0	1.4	0.6	0.9	1.1	0.8	2.9	4.1	4.79	0.6
CHE	5.3–12.9 kU/l	3.56	3.2	1	5.84	4.99	5.17	5.42	4,87	7.21	7.3
GGT	≤60 U/l	164	143	144	263	210	264	283	291	207	729
GOT	≤50 U/l	37	59	55	32	27	27	1080	5197	2671	31
GPT	≤50 U/l	27	44	44	25	20	27	802	3107	2088	71
Thrombocytes	150–350 × 109/l	56	91	127	113	95	97	126	62	54	259
Quick	70–130%	86	80	84	86.3	76.4	86.3	61	40,9	30	107.3
INR	≤1.2	1	1.1	1.1	1.05	1.1	1.05	1.22	1.54	1.93	0.96
Krea	≤0.86 mg/dl	0.91	0.73	0.89	0.93	0.86	1.02	0.8	1.23	2.1	0.77
MELD		6	9	7	7	8	7	14	19	27	6

LTX, liver transplantation; MELD, model for end-stage liver disease [12]

Because direct shunt revascularization was impossible, it was decided to perform a TIPSS obliteration by creating an end-to-side shunt through a stent mesh into the portal vein to maintain normal anatomy. The portal vein was reached through a stent mesh using a Terumo wire and a vertebralis catheter (Fig. 3B). Subsequently, the stent mesh was dilated with a 10/40 mm balloon dilatation catheter (Powerflex, Cordis Europe, The Netherlands; Fig. 3C). After pushing a 10 Fr sheath (Superflex, Arrow International, USA) through the widened stent mesh, a 10/68 mm self-expanding Wallstent-uni endoprosthesis (Boston Scientific, USA) was implanted (Fig. 3D). Despite postdilatation of the entire neo-tract with a 12/40 mm balloon catheter, the portosystemic pressure gradient was 17 mmHg (Fig. 3F). It was found that the TIPSS outflow (Fig. 3F) into the liver vein was another problem. The shunt tract was further extended into the liver vein by a balloon-expandable 10/40 mm Genesis Stent (Cordis Europe, The Netherlands) which was dilated up to 12 mm diameter. Control portography documented proper stent positions without stenosis or thrombosis. The final portosystemic pressure gradient measured 9 mmHg (Fig. 3G). The intervention was performed under analgosedation with 1.25 mg midazolam (Hoffmann–La Roche, Grenzach-Wyhlen, Germany) and 75 mg pethidine (Aventis Pharma, Bad Soden, Germany). For peri-interventional anticoagulation, a total of 8,000 IU heparin was given.

One day later, massive elevation of liver enzymes (GPT, >800 U/l; GOT, >1000 U/l) was recognized (Table 1) and the MELD (model for end-stage liver disease) score [12] rose from 7 to 14 points. A contrast-enhanced (150 ml Imeron 300, Altana Pharma, Germany) multiphase (native, arterial phase, portocaval phase, late phase) CT scan (Somatom 4, Siemens, Germany) was carried out. This detected no abnormalities in either the shunt tract (Fig. 4B) or the liver arteries. However, multiple small filling defects in the superior mesenteric vein were identified suggesting partial thrombosis, which was previously unknown (Fig. 4C). Pre-TIPSS and post-TIPSS liver volumes and cranio-caudal diameters were compared. For that purpose, the data of the baseline and post-TIPSS revision CT studies were analyzed by a three-dimensional liver volumetry program (Volume Viewer Plus, AW Suite 5.5.3b, General Electric, USA).

Because this situation was summarized as acute liver failure, emergency liver transplantation was performed 1 day later, without complications and using a modified piggy-back technique according to Belghiti [13]. Pathologic examination of the explanted liver showed complete cirrhotic conversion with distinct perisinusoidal fibrosis due to the known post-alcoholic liver cirrhosis without signs of necrotic liver parenchyma as a possible embolic

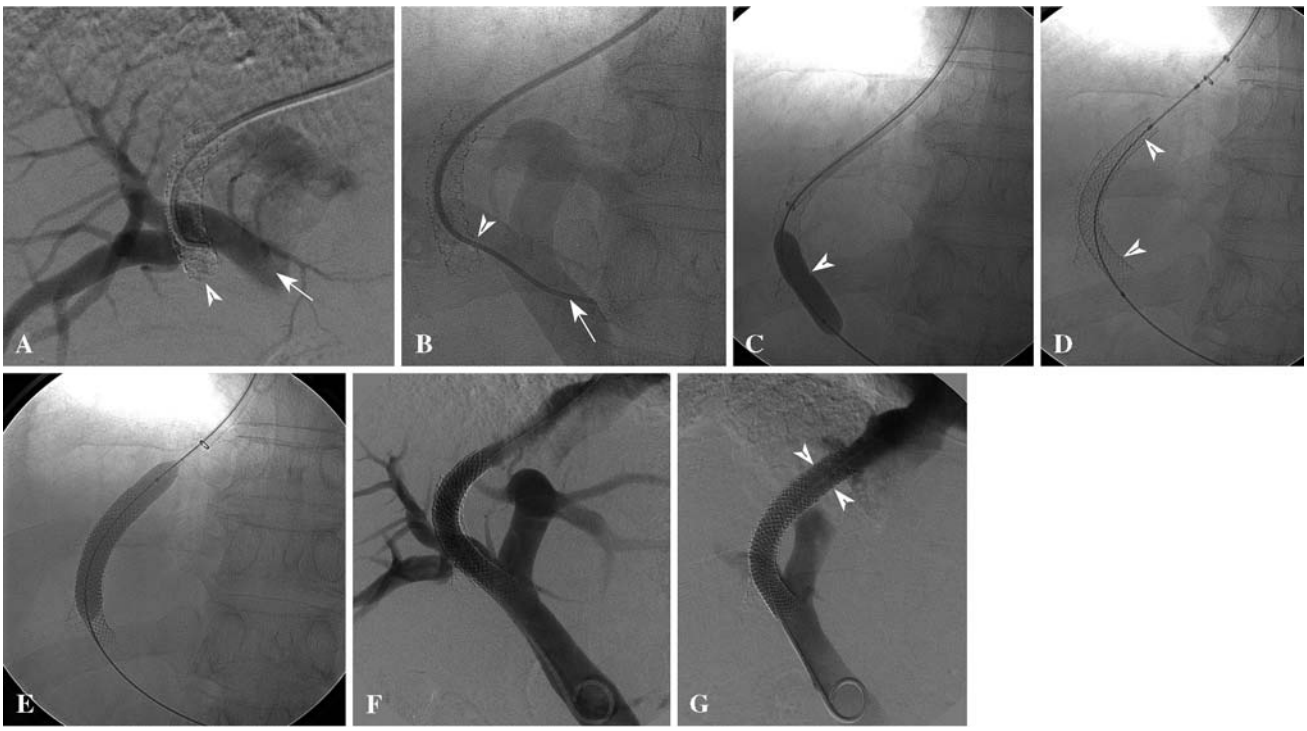

Fig. 3 (**A**) Portography showed protrusion of the proximal stent ending into the periportal liver parenchyma (arrowhead) without contrast extravasation around the portal vein (arrow). (**B**) The vertebralis catheter (arrow) positioned in the main trunk of the portal vein through a TIPSS mesh (arrowhead). (**C**) Dilatation of the TIPSS mesh (arrowhead) with a 10/40 mm balloon. (**D**) Creation of a TIPSS neo-tract with a 10/68 mm self-expanding Wallstent (arrowheads). (**E, F**) After postdilatation with a 12/40 mm balloon dilatation catheter there was a normal angiographic appearance of the TIPSS tract but still a portosystemic pressure gradient of 17 mmHg. (**G**) Extension of the TIPSS tract into the liver vein using a balloon-expanding stent (arrowheads). Portography demonstrated a regular TIPSS tract with a portosystemic pressure gradient of 9 mmHg

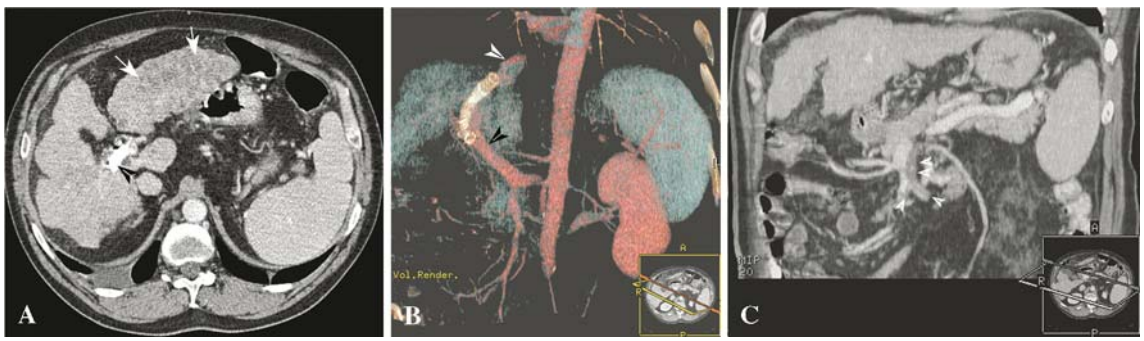

Fig. 4 (**A**) CT after TIPSS revision showed that the TIPSS tract is perfused (black arrowhead); the liver is cirrhotic, and in the venous phase slightly inhomogeneous (white arrows). (**B**) The three-dimensional volume-rendered reconstruction showed a standard location and perfusion of the TIPSS tract (black arrowhead marks inflow, white arrowhead marks outflow). (**C**) Coronally orientated MIP projection demonstrated partial thrombosis of the superior mesenteric vein (white arrowheads) as a complication of the recent herniotomy

complication of the partial thrombosis of the superior mesenteric vein. Furthermore, the partial thrombosis of the superior mesenteric vein was removed during the transplantation using a Fogarty maneuver.

After an uneventful postoperative follow-up with normalization of the liver function, the patient was transferred from the intensive care unit to a regular ward 10 days after transplantation. He died 4 months later due to a massive pulmonary embolism.

Discussion

Liver Failure after Portal Decompression

Even after the primary use of covered stent-grafts for TIPSS, occlusions (2–6%) and hemodynamically significant stenoses (4.5–11%) can occur [5, 14]. Therefore, up to 33–50% patients still require percutaneous shunt revision in the first year after TIPSS [3–6].

Although TIPSS revisions are routine procedures, intervention-associated acute liver failure is a rare complication [7–9]. Neither liver-specific laboratory parameters (Table 1) nor the portosystemic pressure gradient, both comparable after primary TIPSS and after TIPSS revision, served as predictive factors for the acute onset of liver failure in our patient. However, the three-dimensional volumetric analysis demonstrated liver shrinkage from 2,130 ml before TIPSS creation to 2,018 ml before TIPSS revision (–5.3%) and a decrease of around 5.1% in the craniocaudal diameter as a distinct sign of progression of liver cirrhosis. Diversion of portal flow away from the hepatic sinusoids [15] and insufficient compensation by the hepatic arteries, both due to cirrhotic progression [16], might lead to reduced sinusoidal perfusion with hepatic ischemia resulting in acute liver failure.

Harrod-Kim et al. retrospectively reviewed TIPSS procedures carried out for the treatment of refractory ascites. Sixteen of 99 patients died shortly after TIPSS creation (mean survival of 1.9 months). During follow-up, these patients showed a significant increase in their MELD scores and a significant decrease in their portosystemic pressure gradients after TIPSS creation in comparison with the survivors [17]. The authors concluded that an excessive reduction of the portosystemic pressure gradient due to TIPSS in patients with severe liver dysfunction is associated with increased mortality.

The newly diagnosed partial thrombosis of the superior mesenteric vein in our patient was attributed to the recent herniotomy following a perioperative thrombosis of the umbilical vein. Postinterventionally, the patient was anticoagulated with intravenous heparin and there were no clinical symptoms or signs of bowel edema. After liver transplantation, histopathologic examination showed no signs of necrotic liver parenchyma due to possible portal embolization.

Stent Protrusion

Because of the concerns of our transplantation surgeons, and in contrast to Maleux et al. [18], we do not use covered stent-grafts such as the Viatorr endoprosthesis in patients receiving TIPSS as a bridge for planned liver transplantation. Major reasons for stent protrusion out of the portal vein into liver parenchyma could be the combination of residual rigid strength of the implanted Genesis stent, its relatively short portion within the portal vein, and the progression of liver shrinkage since TIPSS creation.

Therapeutic Alternatives

Interventional techniques to reduce the blood flow through a TIPSS tract have been described in a number of patients suffering from TIPSS-induced hepatic encephalopathy and TIPSS-induced liver failure [19–22]. Wolf et al. performed therapeutic shunt occlusion in 7 cases of acute liver failure in the early post-TIPSS period [23]. In these cases the TIPSS tract was occluded using a 10 mm balloon catheter, which was kept inflated in the TIPSS until permanent shunt occlusion was obtained. Applying this technique, Wolf et al. were able to reverse the liver failure, with survival of 3 patients.

In the patient presented in this report, liver transplantation was regarded as the only reasonable therapeutic option because there were serious concerns about severe liver necrosis suggested by the rapid increase in both bilirubin (from 0.8 to 4.2 mg/dl) and liver transaminases (Table 1). Our appraisal is endorsed by the report by Rouillard et al. [10], who demonstrated, that severe hyperbilirubinemia occurring after TIPSS creation is associated with progressive hepatic deterioration as well as high mortality with the need for liver transplantation [10]. Ninety-five percent of the patients who developed severe hyperbilirubinemia within 1 month after TIPSS placement, died or required liver transplantation within 90 days.

In conclusion, acute liver failure following TIPSS revision is an extremely rare but potentially disastrous complication. Retrospectively, we were unable to identify any prognostic parameters predicting its onset. The diagnosis of acute liver failure after either a primary TIPSS procedure or a TIPSS revision must occur as soon as possible to allow proper emergency salvage treatment.

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