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REVIEW

Transjugular intrahepatic portosystemic shunt (TIPS) complications: what diagnostic radiologists should know

Giuseppe Mamone[1] · Mariapina Milazzo[1] · Ambra Di Piazza[1] · Settimo Caruso[1] · Vincenzo Carollo[1] · Giovanni Gentile[1] · Francesca Crinò[1] · Gianluca Marrone[1] · Gianvincenzo Sparacia[1] · Luigi Maruzzelli[1] · Roberto Miraglia[1]

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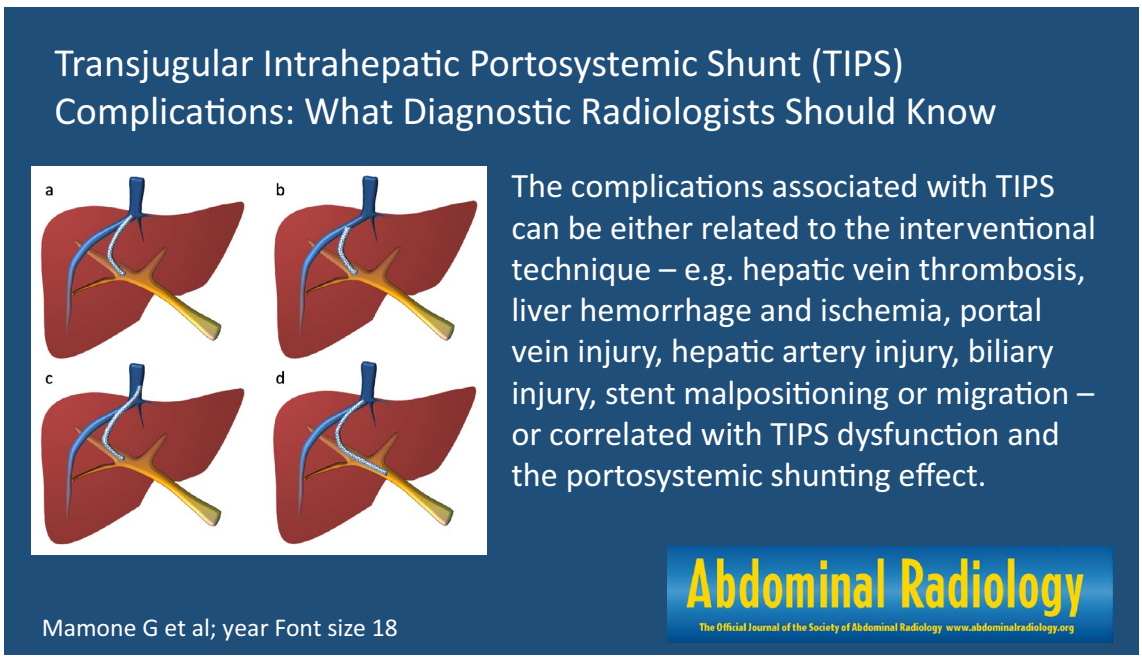
Abstract

Transjugular intrahepatic portosystemic shunt (TIPS) is an effective therapy for portal hypertension complications and can successfully treat variceal bleeding and refractory ascites. Although TIPS is relatively safe, procedural- or shunt-related morbidity can reach 20%, and procedural complications have a fatality rate of 2%. Delayed recognition and treatment of TIPS complications can lead to life-threatening clinical scenarios. Complications can vary from stent migration or malpositioning to nontarget organ injury, TIPS dysfunction, encephalopathy, or liver failure. This review aims to outline the role of diagnostic radiology in assessing post-TIPS complications.

Graphical abstract



Keywords Transjugular intrahepatic portosystemic shunt · TIPS · Imaging · Complications

✉ Giuseppe Mamone
gmamone@ismett.edu

Extended author information available on the last page of the article

Introduction

Transjugular intrahepatic portosystemic shunt (TIPS) is an effective treatment for complications of portal hypertension such as variceal bleeding (unresponsive to medical and endoscopic management) and refractory ascites [1]. Randomized clinical trials support these indications [1, 2], yet broader indications have emerged, including Budd-Chiari syndrome, refractory hydrothorax type 2, hepatorenal syndrome, and portal vein thrombosis. Contraindications to TIPS creation are severe liver failure, severe organic renal failure, heart failure, severe pulmonary arterial hypertension, recurrent or persistent overt hepatic encephalopathy, biliary obstruction, and uncontrolled sepsis [1, 2].

The creation of a TIPS consists of building a channel within the liver between the hepatic venous drainage and the portal supply to reduce portal pressure. Though TIPS is a relatively safe procedure, cases of direct procedure-related morbidity can reach 20% [3]. The reported risk for minor complications is 4%, and for major complications 3% [4, 5, 6]. The polytetrafluoroethylene (ePTFE)-covered stent is associated with a significantly lower rate of TIPS dysfunction, but with comparable efficacy, making it the standard of care [6]. Acute, life-threatening complications account for less than 2% of cases, but shunt dysfunction can occur in 15 to 43.9% of cases, and post-intervention hepatic encephalopathy in up to 53.9% of cases [7]. Other complications include: migration, biliary fistula, segmental intrahepatic cholestasis, hemorrhage, and liver infarction, as well as injury to other organs [7]. This review aims to describe the potential complications that can arise following TIPS placement and their diagnostic imaging in an attempt to aid radiologists in arriving at a correct diagnosis.

Pre-Tips imaging evaluation

Careful pre-procedural image evaluation is essential in assessing candidacy for the procedure and technical feasibility [3, 4, 5, 8]. Pre-procedure computed tomography (CT) and portal venous phase magnetic resonance imaging (MRI) are useful in verifying hepatic and portal vein patency. Very small portal venous branches may be seen in some cases of advanced portal hypertension, while in Budd-Chiari syndrome, the hepatic veins can be small or even absent. It is important to check for dangerous anatomic variations such as extrahepatic portal vein bifurcation.

The presence of multiple hepatic cysts and solid masses should be reported, as these may interfere with the technical success of the procedure and pose a risk of hemorrhage. Obstructed bile ducts require decompression before the procedure and should also be identified on pre-procedure imaging. If cross-sectional imaging has not been performed within one month prior to TIPS placement, preoperative assessment of the hepatic vasculature by Doppler ultrasound should be obtained.

Conventional technique for TIPS creation

TIPS creation involves several steps: the procedure typically begins with percutaneous access via the right internal jugular vein with ultrasound (US) guidance [3, 4]. The right hepatic vein is then catheterized before puncturing the right portal vein. This is the simplest approach as the right hepatic vein usually lies directly posterior to the right portal vein [3, 4, 5, 6].

Once transportal access is secured, a portogram is obtained and the portosystemic pressure gradient (PSG), defined as the difference between the portal pressure and inferior vena cava pressure, is measured. Subsequently, balloon catheter dilation of the intrahepatic parenchymal tract allows stent deployment, followed by further dilatation as needed. A final venogram with pressure measurements is obtained to ensure portal decompression; post-TIPS reduction of the PSG below 12 mmHg typically represents hemodynamic success.

Compared to bare-metal (BM) stents, (ePTFE)-covered stent-grafts have been shown to improve TIPS patency and, nowadays, they represent the standard of care [9]. The (ePTFE)-covered Viatorr endoprosthesis (GORE, Flagstaff, AZ, USA), developed specifically for TIPS creation, is now in common use. The first 2 cm along the portal side of the Viatorr stent is uncovered, whereas the more distal part of the shunt, which can vary in length, is protected by PTFE [9]. The device length is dependent on the length of the hepatic parenchymal tract.

For optimal positioning, the stent should extend through the whole tract right up to the hepatic-caval junction; a gap at the distal hepatic venous portion of the stent may predispose the patient to intimal hyperplasia and stenosis [5]. If the distal portion of the Viatorr stent falls short of the junction between the hepatic vein and inferior vena cava (IVC), an additional Viatorr can be used to extend the stent at the discretion of the primary operator.

Embolization of gastroesophageal varices or other portosystemic venous collateral channels may be performed after TIPS insertion based on the clinical scenario, degree of portosystemic pressure gradient reduction after TIPS, number and size of varices, and presence and degree of variceal filling at post-TIPS portography [3, 4, 5, 6]. Ideally, the portosystemic pressure gradient should be less than 12 mmHg for patients with variceal hemorrhage and

less than 8 mmHg for patients with intractable ascites [3]. Although TIPS are usually performed from the right hepatic to the right portal vein, placement from the middle hepatic vein or left hepatic vein to the left portal venous system is possible [6, 10]. Placement of TIPS to the left portal vein, while less common, may be indicated in some cases of advanced cirrhosis where the right lobe is highly atrophic. Furthermore, patients with Budd-Chiari syndrome may necessitate a direct intrahepatic portacaval shunt when hepatic veins are unsuitable or inaccessible [11].

Complications

The TIPS procedure can be difficult for interventional radiologists to perform. However, it is technically successful in more than 95% of cases, and major complications tend to happen in fewer than 5% of cases [3, 4, 5, 6, 12]. The complications associated with TIPS can be either related to the interventional technique—e.g., hepatic vein thrombosis, liver hemorrhage and ischemia, portal vein injury, hepatic artery injury, biliary injury, stent malpositioning or migration—or correlated with TIPS dysfunction and the portosystemic shunting effect.

Stent malpositioning

Correct stent positioning is mandatory in order to achieve proper functioning and durable patency (Fig. 1). The correct stent position—edge location, in particular—can be assessed by US, CT and MR imaging. Considering the proximal end, the covered portion of the stent should start at the confluence of the portal vein and the liver parenchymal tract. The insertion of a covered stent within the portal vein can cause problems in the perfusion of the intrahepatic portal venous branches [3, 5].

Shunt outflow stenosis and dysfunction can be caused by incorrect stent positioning (Fig. 2a, b): the distal end of the Viatorr needs to reach (or arrive within 1 cm of) the hepatic vein–IVC confluence [3, 5]. In particular, a shunt that is too long with the stent extending into the hepatic level IVC or right atrium (Fig. 2c, d) can make the upcoming liver transplantation more complicated by leaving little room for caval cross-clamping [3, 5].

In order to avoid stent malpositioning, the practitioner should concentrate on taking an accurate shunt length measurement and making a successful Viatorr deployment. The best way to measure the length of the shunt is to use simultaneous portal and hepatic venography, measuring the length of the parenchymal tract of the liver through the use of a catheter with radiopaque markers. In order to

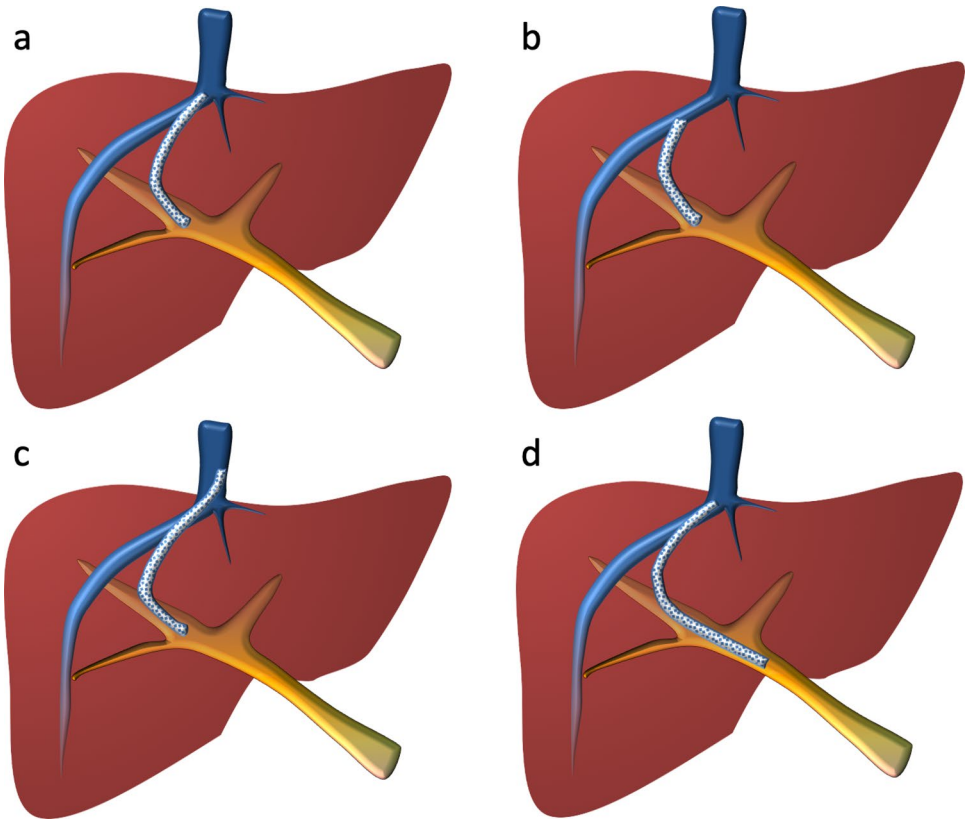


Fig. 1 TIPS positioning. Illustration of transjugular intrahepatic portosystemic shunt (TIPS) positioning. TIPS is usually performed from the right hepatic to the right portal vein. The optimal positioning of the stent (**a**) is to cover the hepatic vein up to the hepatic-caval junction; for the proximal edge of the TIPS, the covered portion of the stent should begin at the junction of the right portal vein and liver parenchymal tract. A gap at the distal hepatic venous portion of the stent (**b**) may predispose a patient to intimal hyperplasia and stenosis. A shunt that is too long, with stent extension into the inferior vena cava (IVC) or right atrium (**c**), may complicate a subsequent liver transplantation, leaving inadequate room for caval cross-clamping at the time of surgery. Deployment of a covered stent within the portal vein (**d**) may limit perfusion of the intrahepatic portal venous branches

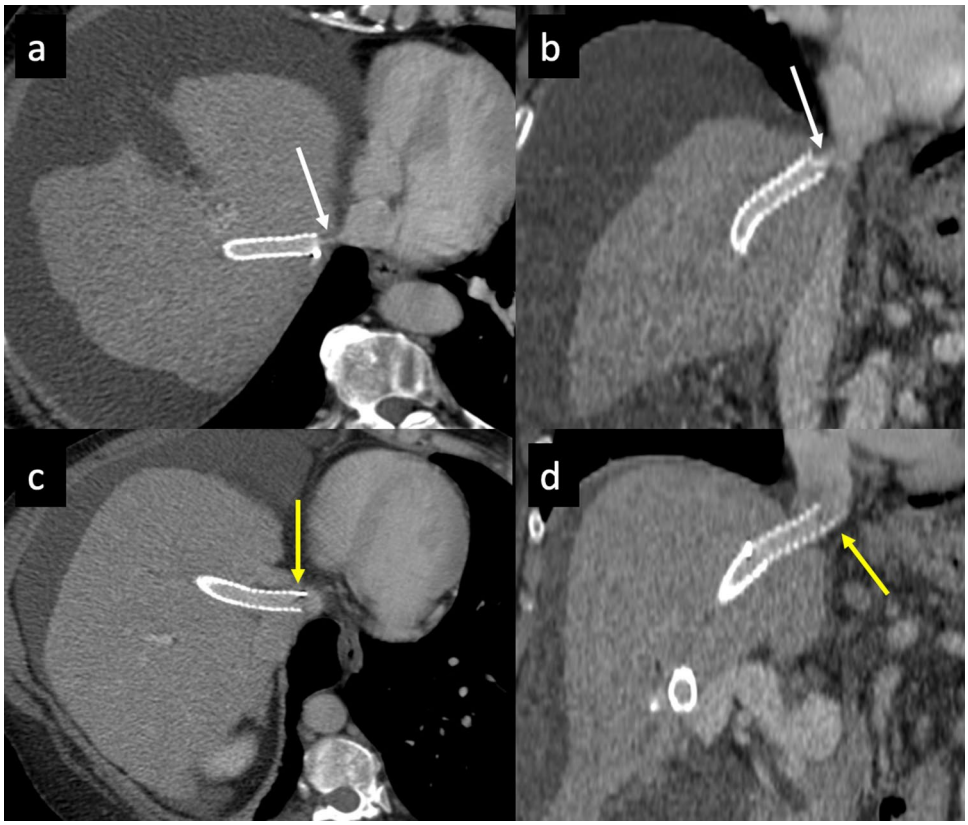


Fig. 2 Stent malposition. Axial (**a**) and coronal (**b**) contrast-enhanced CT images show a stent with the distal edge too short in the right hepatic vein, leaving a gap that led to stenosis (white arrows). Contrast-enhanced CT multiplanar reconstruction (MPR) images (**c**, **d**) show a stent with a proximal edge that is too long extending into the IVC (yellow arrows)

account for the curved nature of the tract, many operators use the "fudge factor", that is they add 1 cm to the length of the hepatic parenchymal tract measured venographically [3]. Shunt extension or revision with stent deployment to the hepatic vein–IVC confluence with a bare-metal (BM) stent may be used.

Stent migration

Stent migration is another complication of TIPS. Covered stents have better long-term TIPS patency but, compared with BM stents, the tendency for migration is higher due to their relatively slippery outer covering [3].

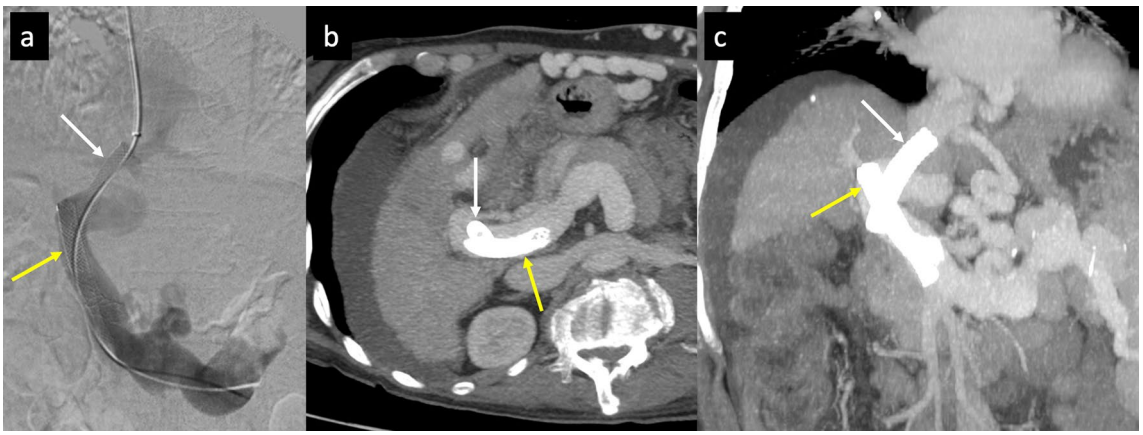


Fig. 3 Stent migration. Angiographic (**a**) and CT maximum intensity projection (MIP) reconstruction (**b**, **c**) images in a patient with multiple stents. The first stent migrated inferiorly into the main portal vein (yellow arrows) and was not removed during the TIPS procedure because the attempted repositioning was unsuccessful. The last stent (white arrows) was correctly positioned

Cardiovascular fistula formation, cardiac arrhythmia, IVC thrombosis, valvular injury, and cardiac rupture with hemopericardium can all be caused by cranial TIPS stent migration [3, 13].

Figure 3 can make a subsequent liver transplantation more complicated [14]. Repositioning can be attempted through balloons or snares, although the definitive treatment option is surgical removal of the migrated stents [3]. If the stent is not removed during the TIPS procedure, cross sectional imaging can help to identify it and to evaluate the findings.

It is also necessary to understand whether variceal embolization happens after TIPS insertion since the recently placed shunt can form a conduit for migration of coils into the pulmonary circulation. Should this happen, CT images can indicate coil numbers and location, and show probable vascular complications, such as occlusion and thrombosis.

Hepatic artery injury

During TIPS insertion, accidental perforation of the hepatic artery or its branches occurs in 1 to 6% of patients [3, 4, 5, 12, 15]. Generally, the symptomatic arterial injury rate is less than 2%, with low clinical significance [3]. Multiphase contrast-enhanced CT can be utilized to detect potential complications of hepatic arterial puncture, including pseudoaneurysm formation, vascular dissection or occlusion, and arterioportal fistula hemorrhage.

The arterioportal fistula may worsen pre-existing portal hypertension. In contrast-enhanced CT imaging, the fistula looks like a communication between the intrahepatic artery and a portal branch, and/or early portal branch opacification (Fig. 4). In addition, Doppler ultrasound shows arterialized flow into the portal venous branch.

Portal vein injury

Since the puncture of the portal vein may result in massive bleeding, it is considered the riskiest step in performing a TIPS procedure [12]. This complication can be avoided with a clear understanding of the vascular anatomy of the liver [12], making the planning of the procedure particularly important in order to assess possible portal venous variants. An extrahepatic puncture when the location of the bifurcation of the MPV is completely extrahepatic (seen in 47% of the population) leads to a risk of bleeding. Three other major variants at risk are trifurcation of the MPV, the right posterior segmental branch arising as the first branch from the MPV, and the right anterior segmental branch arising from the left portal vein [12, 16, 17]. In such cases, the target portal vein branch could be minor in caliber compared to usual. Portal vein perforation occurring during the procedure is treated by the interventional radiologist. In this case, CT or MR are useful for visualizing the residual perihepatic hemorrhage after the procedure. This hemorrhagic collection will be hyperdense in unenhanced CT images and hyperintense in T1-weighted MR sequence images.

During diagnostic imaging studies, another portal vein complication that is frequently found is portal thrombosis. Thrombosis of PV branches could be caused by needle punctures, by the presence of the covered portion of the stent blocking flow to the branches, by the alteration of flow dynamics related to flow diversion, or by a combination of these and other unknown factors [18].

Since imaging is not usually acquired for most patients, it is not possible to estimate the true incidence of segmental portal thrombosis. The thrombosis appears as a non-enhanced vessel on contrast-enhanced CT or MR images (Figs. 5, 6) with the absence of flow at color-Doppler

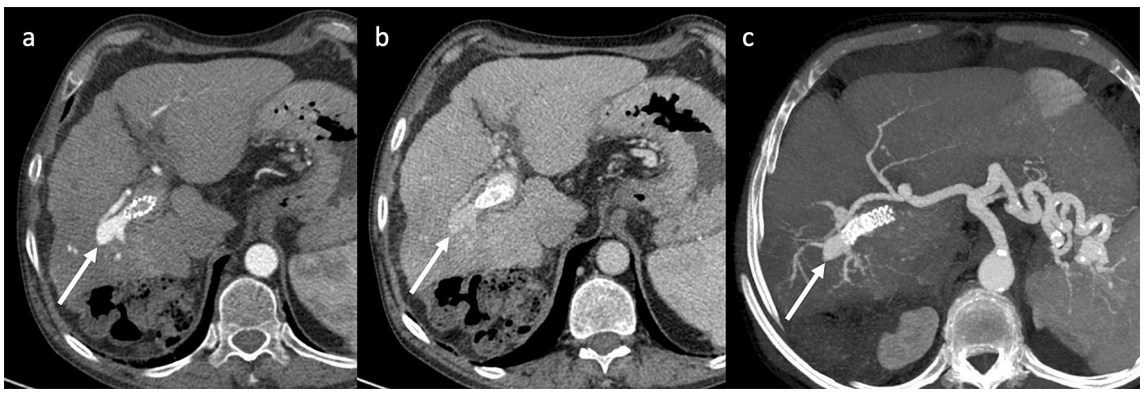


Fig. 4 Arterioportal fistula. Contrast-enhanced CT axial images in the arterial phase (**a**) portal venous phase (**b**), and arterial MIP reconstruction (**c**), exhibit an arterioportal fistula (arrows) close to the TIPS, appearing as a communication between the right hepatic artery and the right portal vein with opacification of early portal branches

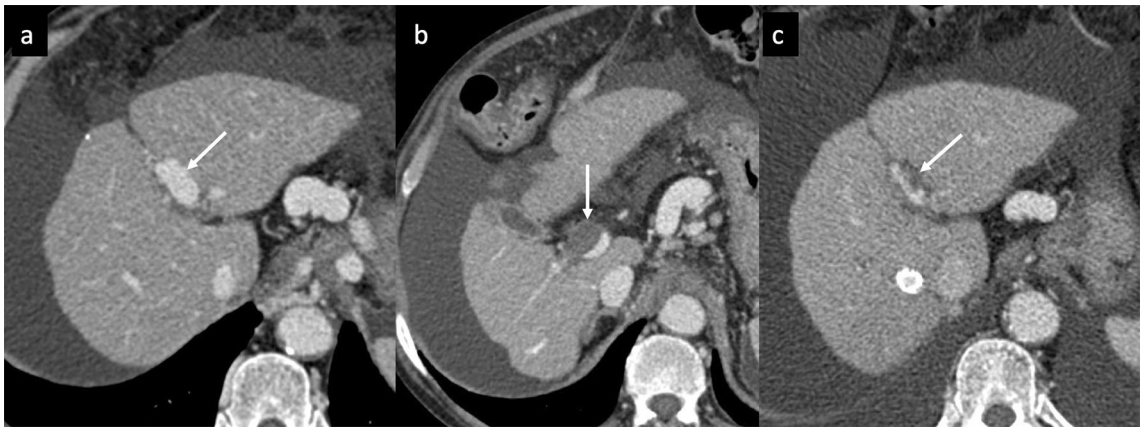


Fig. 5 Portal vein thrombosis. Pre-procedure CT images in the portal venous phase (**a**, **b**) show left portal vein patency and partial thrombosis of the main portal vein (arrows). On the contrast-enhanced CT performed after the TIPS procedure (**c**), the clot moved from the main portal vein to the left portal vein (arrow)

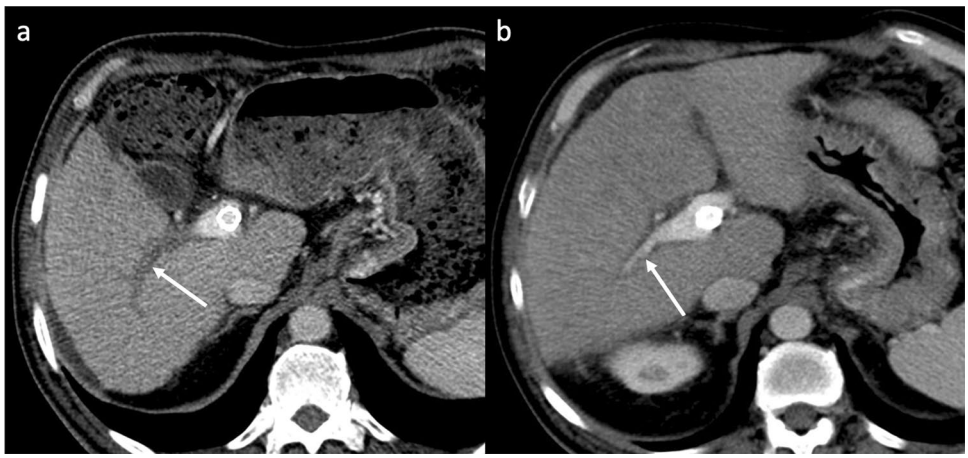


Fig. 6 Portal vein thrombosis. A portal venous phase contrast-enhanced CT image (**a**) shows right portal vein thrombosis occurred after the TIPS procedure which disappeared on the CT examination performed after anticoagulant therapy (**b**)

examination. In order to interpret the data correctly, radiologists should be aware that to-and-fro or stagnant portal flow at color-Doppler could be misinterpreted as portal thrombosis.

Portal vein dissection and venous pseudoaneurysm represent further, but extremely rare, portal complications.

Hepatic vein thrombosis

Hepatic vein thrombosis at the site of stent access, frequently the right or middle hepatic vein, in patients undergoing a TIPS procedure is another vascular complication that can occur during diagnostic imaging studies [18]. Thrombosis of the hepatic vein has been seen after the use of covered stents [19]: the obstruction of the hepatic vein by the covered stent or the slow flow into the vein can give rise to thrombosis in about 5% of patients. Usually, clinical consequences are not present [19]. However, a Budd-Chiari-type hepatic ischemia and acute hepatic failure can be the result of an occlusion of one or more hepatic veins—particularly when there are shared origins—due to the covered portion of the TIPS [5]. The thrombosis appears as a non-enhanced hepatic vein on contrast-enhanced CT or MR images (Fig. 7), with the absence of flow at color-Doppler examination.

In order to interpret the data correctly, radiologists should be aware that stagnant hepatic vein flow at color-Doppler could be misinterpreted as hepatic vein thrombosis.

After a TIPS procedure, thrombosis can also happen in the inferior vena cava (Fig. 8).

Biliary injury and dilatation

During TIPS insertion, the stent can transect a bile duct causing a biliary injury. Intrahepatic biliary duct puncture is reported in up to 5% of cases [3, 4, 20].

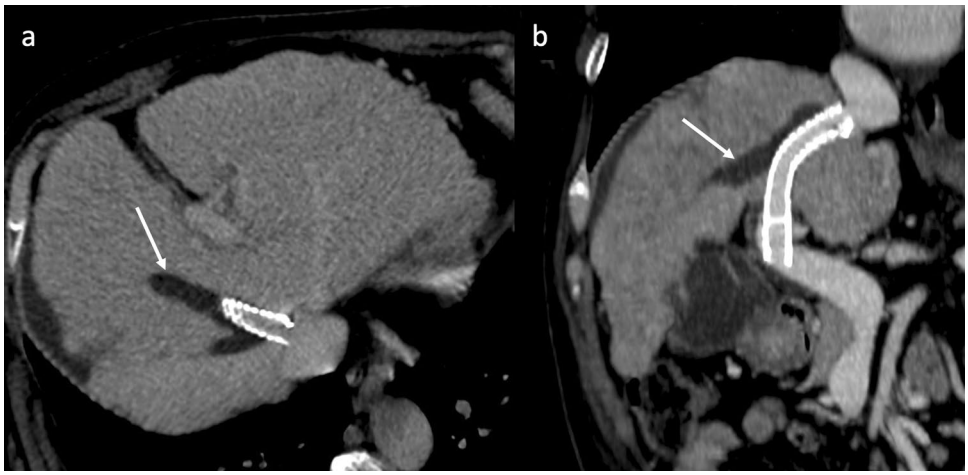


Fig. 7 Hepatic vein thrombosis. MPR CT images (**a**, **b**) show complete middle hepatic vein thrombosis (arrows) up to the site of stent access, appearing as hypodensity in a non-enhanced vessel

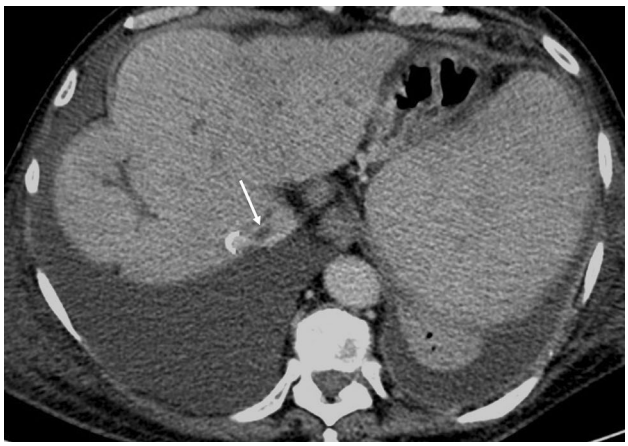


Fig. 8 IVC thrombosis. Axial contrast-enhanced CT image on 3 min delayed phase shows a thrombus in IVC close to the distal edge of the TIPS in the right hepatic vein

In the era of uncovered stents, the treatment of bile duct fistulas was quite complex since they generated marked pseudointimal hyperplasia and secondary stent occlusion, with frequent propagation of thrombosis into native portal systems [4, 5]. This fistulous communication between biliary and vascular systems can cause sepsis, stent infection, hemobilia and cholangitis [3]. The covered stent should prevent the formation of such fistulas [5]; indeed they are now reported less frequently. TIPS stents can also generate bilomas and/or biliary occlusion [5, 7, 21]. Rarely, rapid hepatic decompensation can occur, speeding up the need for orthotopic liver transplantation [21].

The mechanical obstruction of intrahepatic biliary ducts by the stent-graft after the TIPS procedure can cause segmental or sectorial intrahepatic biliary dilatation, defined as segmental cholestasis [7]. Mechanical compression and ischemia through compression of the segmental artery for stent placement through the segmental bile duct are assumed to play a combined role [7]. Significant congestion of the biliary system proximal to the obstructed intrahepatic bile duct can be caused by this segmental biliary dilatation. A segmental intrahepatic cholestasis event may be avoided by TIPS placement from the medial hepatic vein to the right portal vein [7]. US, CT and MR imaging after TIPS positioning shows a segmental or sectorial biliary duct dilatation finishing at the level of the stent (Fig. 9). Other possible aspects that may cause a segmental or sectorial biliary dilatation, such as a tumor, portal hypertensive biliopathy or cavernoma, should be excluded using CT and MRI. Segmental intrahepatic biliary dilatation could be the cause of cholangitis and biloma (Fig. 9).

Ascending cholangitis, caused by reduced biliary excretion, could necessitate urgent biliary decompression. MR imaging with MRCP is the best way to evaluate cholangitis since it can show intrahepatic biliary dilation through ductal wall thickening and enhancement. These events can be linked with parenchymal inflammatory changes, including patchy or peribiliary parenchymal enhancement (in particular in the arterial phase) and wedge-shaped T2-hyperintensity in the segments involved in biliary dilatation. The appearance of material inside the dilated biliary ducts can also be identified. Multiphasic contrast-enhanced CT and MR imaging shows a biloma as a hypodense/hypointense round or oval lesion. Should the infection advance, an intrahepatic abscess (Fig. 10) is shown as a uni- or multiloculated hypodense/hypointense lesion with peripheral enhancement by multiphasic contrast-enhanced CT and MR imaging.

Classic findings of a hepatic abscess are the double target sign and the cluster sign. The double target sign is caused by a central low attenuation lesion (fluid or necrosis) on contrast-enhanced CT imaging, surrounded by an enhancing inner rim and a low attenuation outer ring (edema in the surrounding liver parenchyma) [22]. The cluster sign arises from the aggregation of multiple low

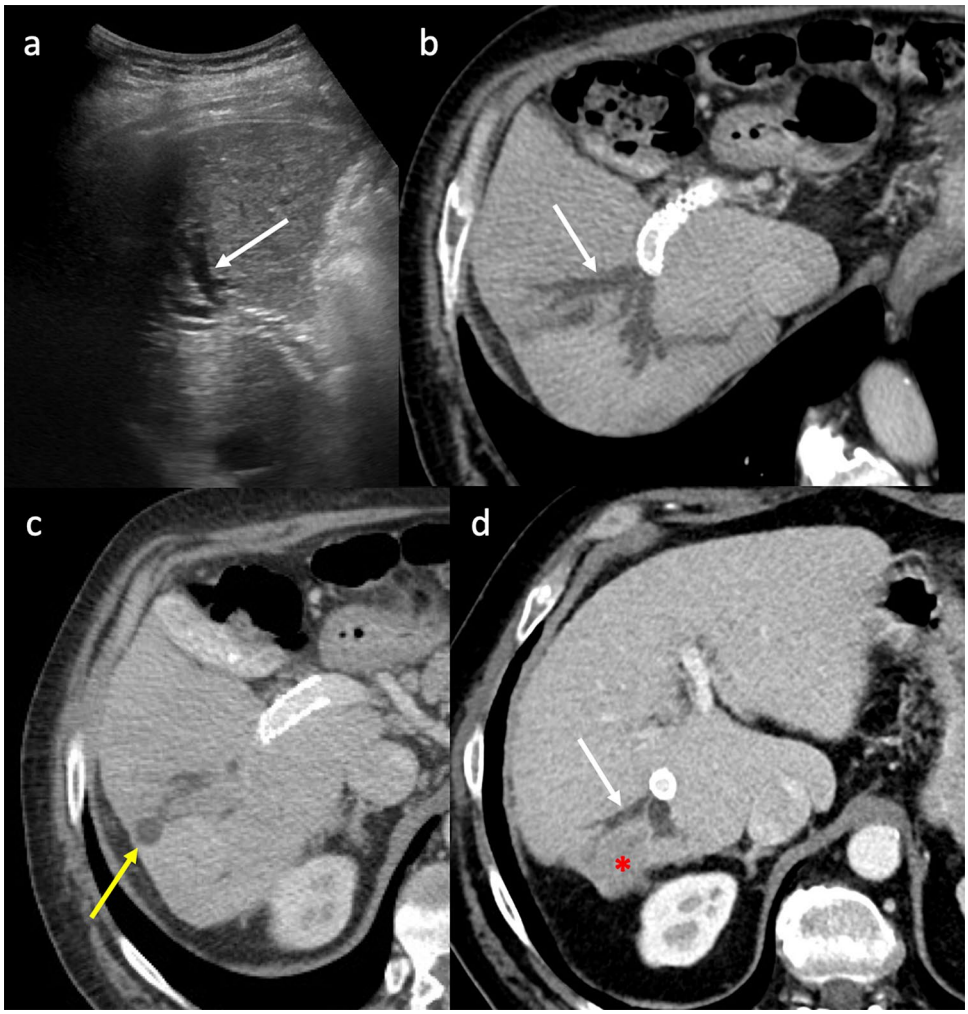


Fig. 9 Biliary dilatation and biloma. Liver ultrasound (**a**) shows biliary dilatation (white arrow) which stops at the level of the stent (arrow), confirmed on contrast-enhanced CT images (**b**, **c**). Notice the presence of a small biloma (yellow arrow) as a round fluid-like hypodensity. Contrast-enhanced CT imaging performed one month later (**d**) showed reduction of biliary dilatation and posterior sectorial atrophy (asterisk)

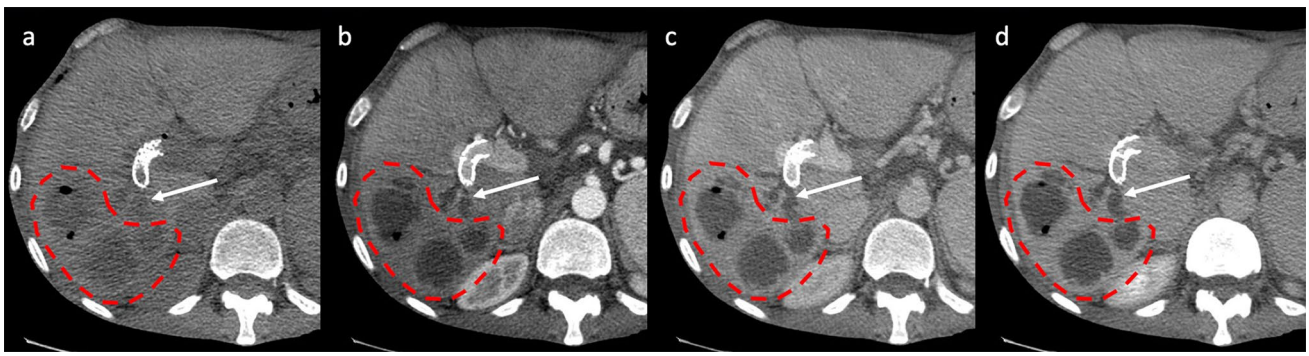


Fig. 10 Biliary dilatation and hepatic abscess. Axial unenhanced (**a**) and contrast-enhanced CT imaging in the arterial (**b**), portal venous (**c**), and 3 min late (**d**) phases show a loculated hepatic abscess (red line) as a hypoattenuating lesion containing gas bubbles. This lesion is associated with biliary dilatation (arrows) which stops at the level of the stent

attenuation liver lesions to form a larger solitary abscess with multilocularity; this sign is typical of pyogenic hepatic abscesses [23]. Segmental, wedge-shaped or circumferential perfusion abnormalities with early enhancement are often linked with hepatic abscesses. Sometimes, bubbles or air-fluid levels can be seen inside the lesion (Fig. 10).

Liver ischemia

Liver ischemia and infarction sometimes complicate the TIPS procedure [18].

The true incidence of liver ischemia after TIPS creation is undetermined since contrast-enhanced CT or MR imaging is often performed only when complications occur.

Hepatic infarcts are extremely rare because of the dual blood supply to the liver (70% portal flow and 30% hepatic arterial flow, with most arterial blood feeding the biliary tree) [24]. Shunting of the already compromised portal flow from the portal vein into the systemic venous circulation results in reduced sinusoidal flow which in turn is believed to cause infarction [3]. The increase in liver arterial blood flow creates a buffering effect, capable of buffering 25%-60% of the decreased portal flow. This arterial buffer reserve negatively correlates with the Child–Pugh score [15]. Vizzutti et al. [4] also propose thrombosis of the portal vein as a potential contributing factor to liver infarcts [25], and other studies show that stent compression of the hepatic artery can cause hepatic ischemia or infarction [3]. Moreover, liver infarcts secondary to thrombosis of the hepatic vein have been reported in up to 5% of cases after using a covered stent [18] in the right or middle hepatic venous territory. This complication, which can sometimes lead to hepatic failure, occurs more frequently with covered stents [25]. When imaging is obtained from patients suffering from right upper quadrant pain and/or having a marked increase in liver enzyme levels or hepatic encephalopathy after a TIPS procedure, ischemia is often found to be the cause [18]. Hepatic ischemia has been found incidentally during follow-up imaging even in asymptomatic patients [18]. MRI and CT show the parenchymal ischemic injury as a hypodense/hypointense non-enhancing area on contrast-enhanced images without mass-effect on adjacent structures (Fig. 11). Hepatic ischemia and infarct are often peripheral and wedge-shaped but can show a round or irregular shape. On MRI imaging, hepatic ischemia or infarction appears as hypointense area on T1 imaging, with hyperintensity on T2 images. Ischemia is a condition of inadequate blood supply, while infarction is a localized area of ischemic necrosis. The extent of damage can be assessed by observing the distribution of segmental or sectorial ischemia. Potential sequelae of hepatic infarction include bile duct necrosis and biloma formation, abscess, and parenchymal atrophy and scarring of the affected segments (Fig. 11).

Liver hematoma

Intrahepatic hematoma is a sporadic complication of the TIPS procedure. It can originate from injury to the hepatic artery or other hepatic vessels with intrahepatic bleeding. Hepatic hematoma appears as a hyperdense lesion on unenhanced CT images and a hyperintense lesion on T1-weighted MRI sequences (Figs. 12, 13); enhancement is not visible on contrast-enhanced CT or MR images. A large hematoma can lead to compression of the hepatic vessel or the biliary duct and biliary dilatation (Fig. 13).

Nontarget puncture

Rarely during the transhepatic needle puncture phase of the TIPS procedure, a nontarget organ injury can occur [3, 4, 5]. Transgression of the liver capsule with the needle/catheter combination during TIPS can happen in 33% of cases, with intraperitoneal hemorrhage occurring in 1 to 2% of cases [5].

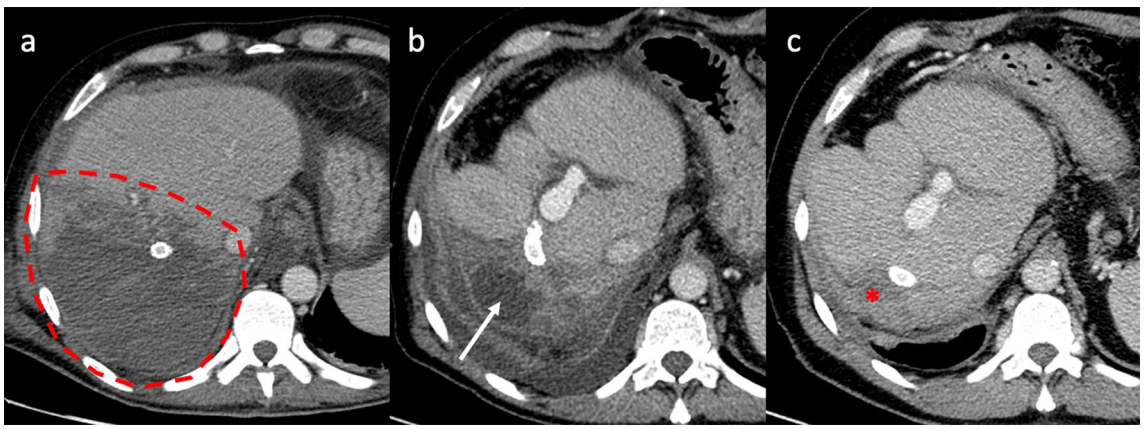


Fig. 11 Liver ischemia. Axial contrast-enhanced CT images (**a**, **b**), exhibit a large hypodense hepatic area of infarct (red line) complicated by abscess (arrow). Contrast-enhanced CT imaging performed some months later (**c**) showed sectorial atrophy associated with a fibrotic area at the site of a previous abscess (asterisk)

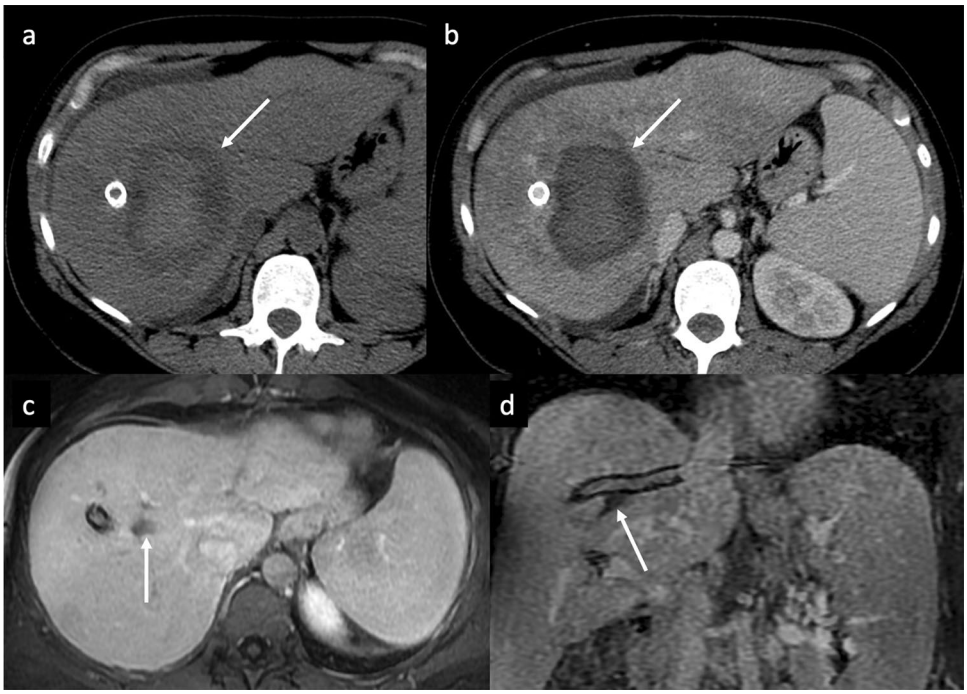


Fig. 12 Liver hematoma. TIPS procedure complicated by large intrahepatic hematoma (arrows) near the stent, which appears as a hyperdense lesion on unenhanced CT image (**a**) without contrast enhancement in a portal venous phase image (**b**) Some months later, axial (**c**) and coronal (**d**) contrast-enhanced MR imaging shows hematoma regression with a non-enhancing fibrotic area left (arrows).

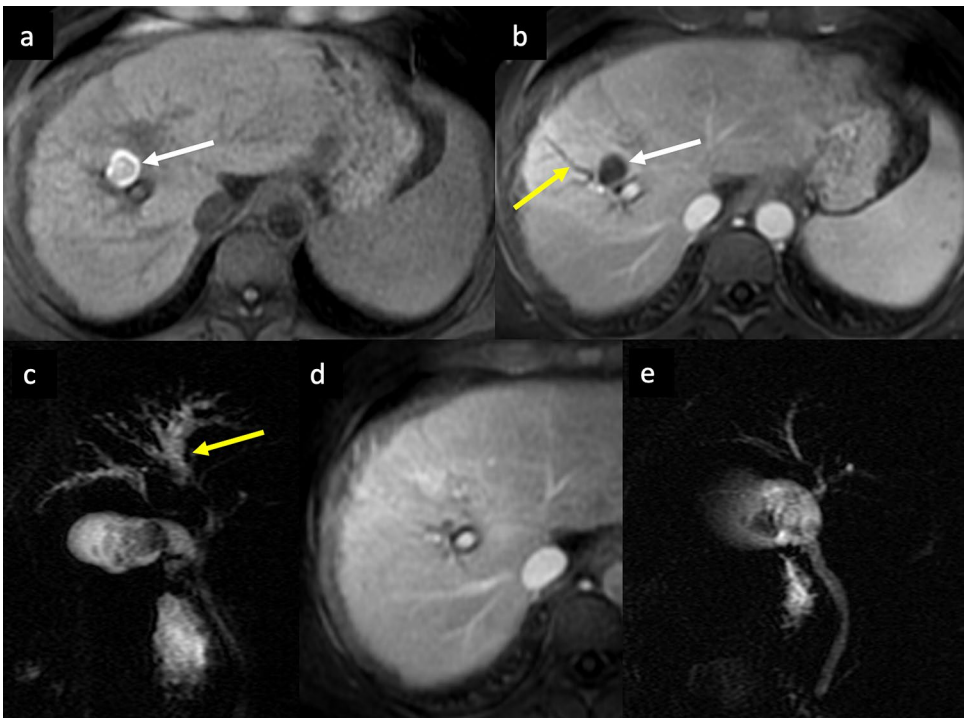


Fig. 13 Liver hematoma. MR imaging shows a hepatic hematoma (white arrows) hyperintense on a T1-weighted image (**a**) and non-enhancing in a contrast-enhanced portal venous phase image (**b**). The hematoma causes biliary dilatation (yellow arrows), better visualized on a coronal MRCP image (**c**). Some months later, axial contrast-enhanced MR (**d**) and coronal MRCP (**e**) images show hematoma regression with a reduction in biliary dilatation

Nontarget organs that are at risk of injury are the right kidney, gallbladder, colonic hepatic flexure and duodenum. The most commonly injured organ is the gallbladder [3]. Signs and symptoms of gallbladder injury include hemobilia, cholangitis, and intrabiliary blood clots [3]. Ultrasound and contrast-enhanced CT or MR imaging shows laceration on the gallbladder wall (Fig. 14) with peripheral fluid (leaking of bile and blood). Hematuria is the most frequent finding of kidney injury, although the theoretical risk of kidney laceration and hemorrhage exists [3]. Mostly, nontarget organ injuries are well-tolerated by patients. The most important means of avoiding nontarget puncture is a deep knowledge of the anatomic relationship between the hepatic veins and portal veins, in order to correctly direct needle throws [3].

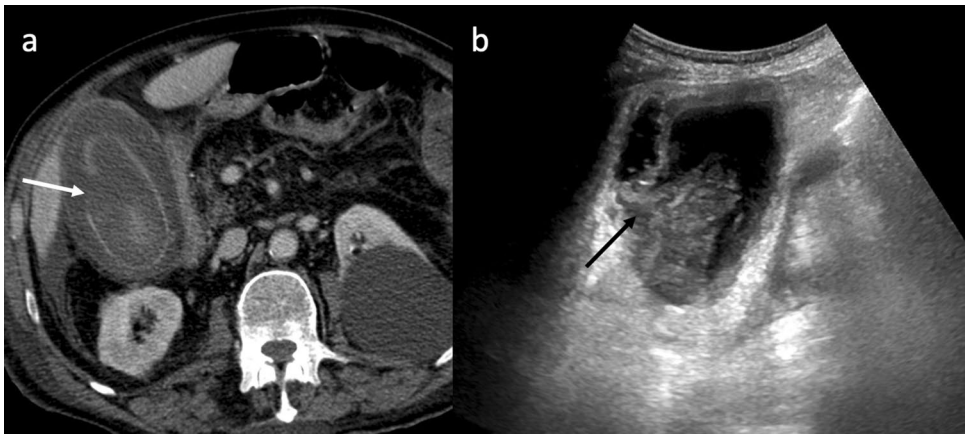


Fig. 14 Nontarget puncture. TIPS procedure complicated by gallbladder puncture. Axial portal venous phase contrast-enhanced CT image (**a**) shows laceration on the gallbladder wall (arrow) with peripheral fluid. The ultrasound (**b**) also confirmed the perforation of the gallbladder wall (arrow) with leaking of bile and blood

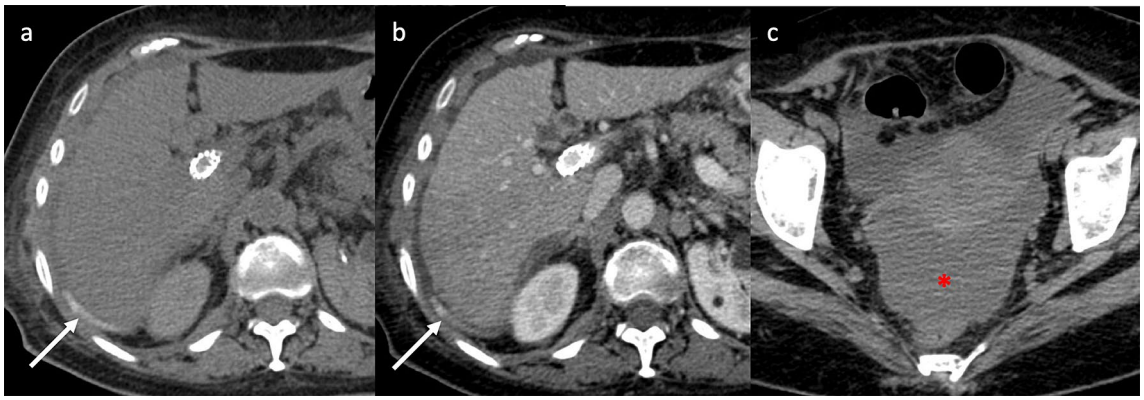


Fig. 15 Nontarget puncture. TIPS procedure complicated by transgression of the liver capsule during the transhepatic needle puncture phase, with intraperitoneal hemorrhage. Unenhanced (**a**, **c**) and contrast-enhanced (**b**) CT images show hyperdense perihepatic (arrows) and pelvic (asterisk) hemorrhagic fluid

Careful pre-procedural planning with cross-sectional imaging analysis is also essential [3].

The use of real-time US guidance during the intrahepatic portion of the right PV puncture may reduce the occurrence of these complications. CT or MRI imaging performed early after the procedure can help to show signs of nontarget organ injury, such as bleeding or perivisceral fluid/air. In the case of intraperitoneal hemorrhage, CT and MR imaging shows abdominal fluid, with typical hyper attenuation and T1-hyperintensity, respectively (Fig. 15).

TIPS dysfunction

TIPS dysfunction, defined as a reduction in portal venous decompression due to occlusion or stenosis of the shunt, can be difficult to detect since signs and symptoms of device malfunction are usually equivocal or absent. In order to detect TIPS dysfunction accurately, shunt venography is generally required, which despite allowing simultaneous TIPS revision when stenosis or occlusion is found, is invasive and expensive. Thus, follow-up of TIPS by Doppler ultrasound has become a common and crucial method of identifying shunt dysfunction. Nonetheless, during the first few days after covered stent implantation, an acoustic barrier can prevent the examination of the shunt lumen with Doppler ultrasound: the acoustic barrier seems to be caused by air bubbles trapped inside the graft and usually resolves spontaneously during the first week after TIPS creation [6]. For this reason, initial baseline examinations in patients with a PTFE-covered stent should be performed 7–14 days post-procedure.

TIPS stenosis and occlusion are the most frequent complications related to shunt procedures. In the era of BM stents, several studies reported an incidence of 60–80% TIPS dysfunction at 2 years [26]. With the introduction of the Viatorr (ePTFE)-covered stent, the long-term patency of shunts has significantly increased with a subsequent large decrease in the incidence of TIPS dysfunction at 2 years (20–30%) [26]. Whereas dysfunction in covered stents tends to be related to intrastent stenosis and pseudointimal hyperplasia, in BM stents it is associated with stenosis and occlusion through the

creation of biliary-TIPS fistulas. Indeed, a proinflammatory and thrombogenic environment (granulomatous inflammatory response) may be caused by the content of bile acids, salts, cholesterol and phospholipids. Doppler ultrasound evaluation of the TIPS can show velocity and flow changes that suggest stent dysfunction.

TIPS occlusion

It is reported that less than 5% of TIPS procedures result in acute shunt thrombosis [5].

Diagnosis of TIPS occlusion with Doppler ultrasonography is simple and has very high sensitivity and specificity (100%) (it is redundant) as long as neither color nor duplex signals are present within the shunt lumen. Contrast-enhanced CT and MR images show stent thrombosis as an absence of lumen opacification (Fig. 16) and reveal any eventual stent malpositioning. Early acute TIPS thrombosis rarely occurs with Viatorr stents.

It is more common to find suboptimal TIPS positioning or configuration causing structural obstruction of flow leading to thrombosis and this may occur due to the following:

(1) The cranial stent end of the TIPS lies within the parenchymal tract.
(2) The cranial end of the TIPS is directed perpendicularly toward the superior wall of the hepatic vein.
(3) The cranial end of the TIPS projects excessively into the IVC with the stent-graft abutting the IVC wall.

In all three scenarios, flow through the TIPS is blocked, potentially resulting in thrombosis.

In addition, stasis and shunt thrombosis may result from extrahepatic or hemodynamic causes of TIPS dysfunction, such as flow theft from varices or mesocaval shunts.

Acute TIPS thrombosis can be managed with mechanical thrombectomy or catheter-directed thrombolysis [5]. In the cases of suboptimal configurations (1) and (2) above, attempts can be made to perform TIPS cannulation and extend it with another stent [5]. If this is not achievable, a new TIPS needs to be created.

TIPS stenosis

Shunt stenosis is a serious problem associated with TIPS. Even with the introduction of the PTFE-covered stent significantly improving patency, the stenosis rate remains relatively high.

Recurrence of portal hypertension, due to shunt failure, can lead to a re-accumulation of ascites or repeated variceal bleeding. Therefore, during TIPS follow-up, shunt dysfunction is commonly recognized by Doppler ultrasound. Shunt stenosis can happen anywhere along the created portosystemic tract; that is, at the proximal segment (shunt-portal vein junction), mid-segment, or along the distal segment (shunt-hepatic vein junction) [6].

The development of pseudointimal hyperplasia, specifically along the hepatic venous outflow, is a common cause of TIPS dysfunction [27]. The distal edge of the stent should lie in the hepatic vein-vena cava junction to avoid hepatic vein stenosis from pseudointimal hyperplasia. Some authors reported that the patency rate of covered TIPS with an optimal stent length was 91% at 1 year, but only 80% with a lack of full-tract coverage [28]. Non-bile-related dysfunction appears due to the differentiation of hepatic fibroblasts into myofibroblasts, migrating into the TIPS lumen from the hepatic parenchyma and causing tissue overgrowth (fibrotic healing response). The gold standard for TIPS evaluation continues to be trans-shunt venography (shunt portography) with a portosystemic pressure gradient (PSG) measurement.

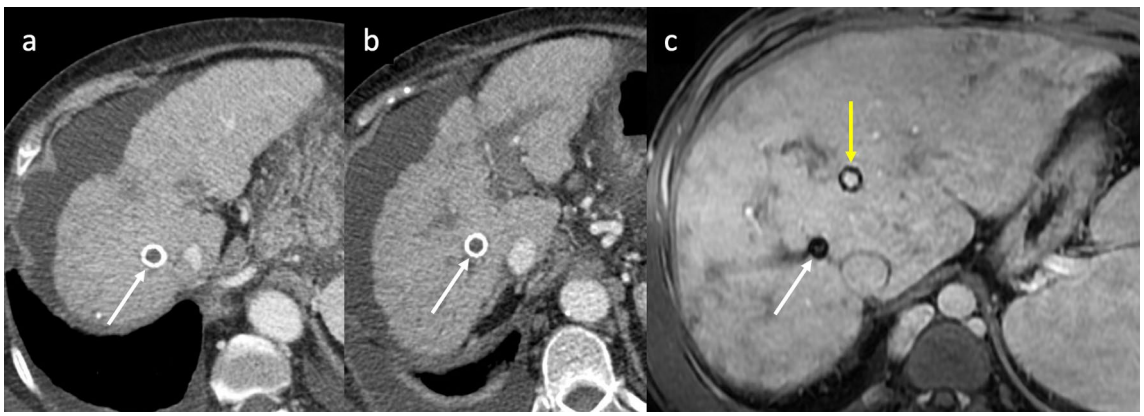


Fig. 16 TIPS dysfunction. Axial contrast-enhanced CT images (**a**, **b**) in a cirrhotic patient show TIPS occlusion as hypodensity in the stent lumen (arrows). Axial contrast-enhanced MR image (**c**) shows stent occlusion (white arrow) in patient with Budd-Chiari syndrome. The patient underwent a second TIPS procedure with a new patent stent (yellow arrow)

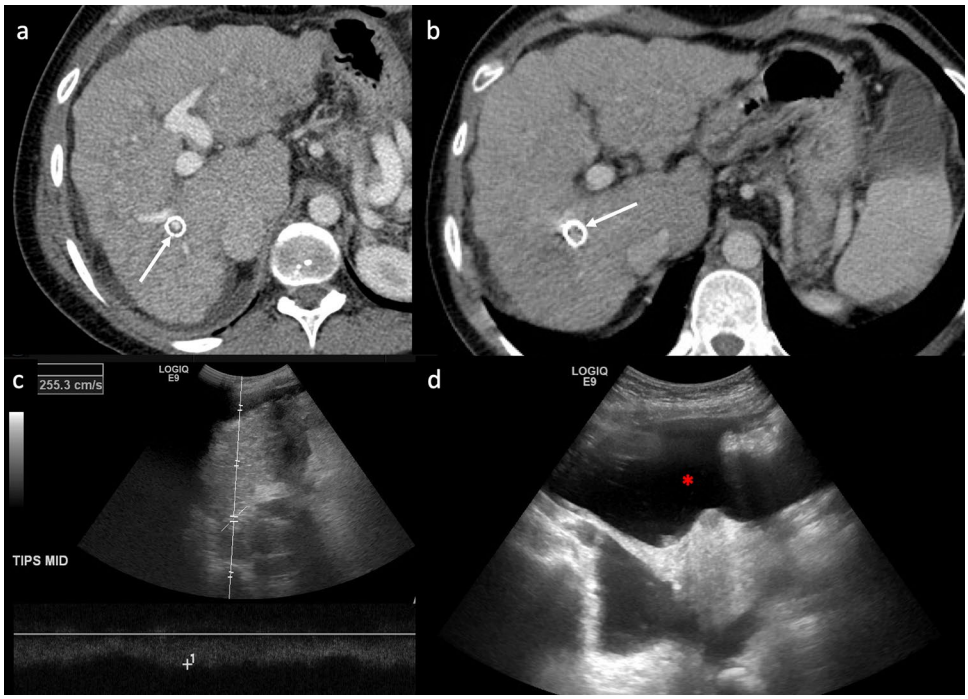


Fig. 17 TIPS dysfunction. Axial contrast-enhanced CT images (**a**, **b**) show a filling defect in the stent lumen (arrows) caused by thrombosis or intimal hyperplasia. Doppler ultrasound shows high flow velocity in the medium part of the stent (**c**) associated with recurrence of ascites (red asterisk) (**d**), suggesting a TIPS dysfunction

Although various venography criteria of TIPS dysfunction are identified in the literature, they have been most commonly defined as a PSG greater than 12 mmHg or a luminal narrowing greater than 50% [6]. Multidetector CT (MDCT) has also been defined as a tool in the follow-up of patients with PTFE-covered stent-grafts [6, 29], but to date few studies have been published on the topic. CT can be used to identify filling defects in the shunt caused by thrombosis or pseudointimal hyperplasia, but it does not provide functional information about flow or pressure. In addition, this technique cannot be used as a screening tool because of the cost and the administration of radiation dose and contrast medium. Nonetheless, CT can have a role in patients for whom ultrasound is invalidated for poor acoustic windows or in early course post-TIPS procedures with PTFE-covered stent-grafts when trapped gas can make visualization by Doppler ultrasound difficult.

MR imaging is a limited tool for the detection of intraluminal defects due to the fact that steel stents give rise to a large number of susceptibility artifacts. There are newer stents, causing minimal susceptibility artifacts, which allow for better visualization of the stent lumen. An alternative to ultrasound for measuring flow velocities is four-dimensional phase-contrast MRI [30]. In this case, MR can provide both functional and anatomical information. However, only a few studies have been published on this topic.

In conclusion, TIPS patency is commonly evaluated using Doppler ultrasonography. While being the best screening tool for identifying early shunt dysfunction, many studies show conflicting results regarding its performance.

First, ultrasonographic criteria were developed from studies in the era of BM stents when data on covered stents were more limited [26]. Second, several criteria have been suggested, but there is no consensus regarding the best ones. Third, many of the ultrasound studies are methodologically incorrect since sonographic criteria of shunt dysfunction were used to trigger TIPS venography; therefore, they lack proof of shunt patency when sonography suggested no shunt dysfunction and venography was not performed. Fourth, some old studies applied a cut-off of 15 mmHg for the PSG to identify dysfunction, whereas a cut-off of 12 mmHg has been more typically used since 2000. Since PFTE-covered stents have shown improved long-term patency, a follow-up is needed every 3–6 months; indeed, frequent routine surveillance may not be cost-effective for this type of stent.

The main Doppler criteria used to identify a TIPS dysfunction are [6, 7]:

- Reversal of flow in portal branches (from hepatofugal or stagnant to hepatopetal) (high specificity: 81–100%)
- Reversed hepatic vein flow (low sensitivity: 29–33%)
- Shunt velocities of ≤ 60 cm/s or ≥ 200 cm/s
- Velocity in the shunt to the proximal segment of < 50 cm/s (nearly 100% sensitivity and 93% specificity)
- Temporal changes in shunt velocities of > 50 cm/s
- Main portal venous velocity of < 30–31 cm/s (63% sensitive and 68% specific)

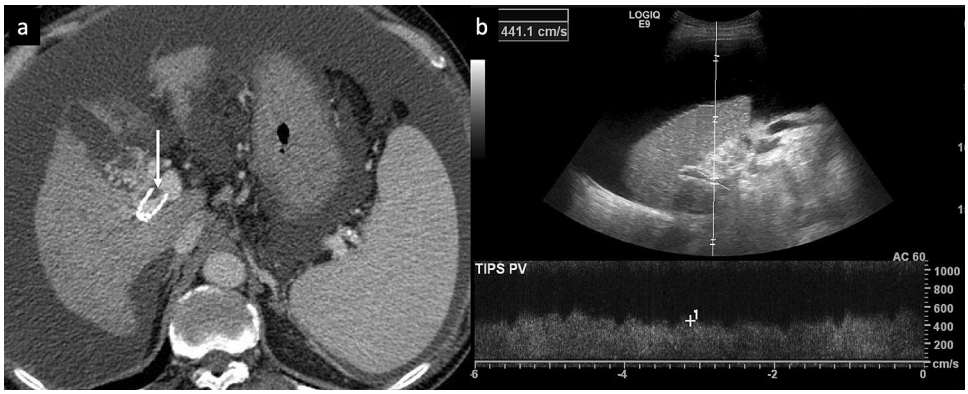


Fig. 18 TIPS dysfunction. Axial contrast-enhanced CT (**a**) and Doppler ultrasound (**b**) imaging shows a filling defect in the proximal edge of the stent (arrow) caused by thrombosis or intimal hyperplasia, associated with high flow velocity and recurrence of ascites, suggesting a TIPS dysfunction

– Difference between two points in the device (gradient) of over 100 cm/s (56% sensitive and 78% specific)

  For instance, there are typical cases where low velocity was seen in the shunt lumen but with a focal velocity elevation that correlates with a site of stenosis (Figs. 17, 18).

  Doppler ultrasound also has technical challenges. It is operator-dependent, and it needs highly accurate angle-corrected velocities throughout all parts of the stent. Two general approaches are used to improve the accuracy of ultrasound for predicting dysfunction:
– Combining several sonographic velocity criteria in the hope of improving the sensitivity and specificity of detecting shunt dysfunction
– Focusing on velocity changes compared with a baseline examination

Few studies have examined how to apply Doppler ultrasound optimally, in particular to covered stents.

The performance of Doppler ultrasound was studied in a multicenter prospective randomized trial involving 80 patients focused on comparing bare and covered stents [6, 31]. Shunt dysfunction correlated significantly with low velocity in the MPV, and the optimal cut-off value was found to be 31 cm/s. One study showed that, despite having low accuracy, Doppler ultrasound can avoid unnecessary venograms by excluding TIPS dysfunction in many patients. In more recent studies where venography was not routinely performed in the follow-up, the false-negative rate from a normal Doppler ultrasound examination was uncertain. Routine Doppler ultrasound surveillance in covered stent-graft patients was found to change medical management in only a small percentage of cases since many symptomatic patients with TIPS dysfunction go straight to venography. Similar results were obtained by Pan et al., who compared bare and covered stents in 128 patients and did not find abnormal Doppler ultrasound to be highly predictive of dysfunction at venography [32]. The number of patients with covered stents who had abnormal Doppler ultrasound results requiring venography was too low to be statistically relevant. Overall findings suggest that protocols requiring frequent ultrasound may be unnecessary in asymptomatic patients and that a routine Doppler ultrasound follow-up should be performed together with tumor surveillance only in cirrhotic patients. It is clear that more studies are needed to evaluate the performance of Doppler ultrasound in the detection of TIPS stenosis with PFTE-covered stents.

Liver failure and acute hepatic encephalopathy

Liver failure and hepatic encephalopathy represent other complications associated with TIPS and are also considered contraindications to TIPS placement.

Hepatic failure after TIPS placement is a rare but serious complication with a poor prognosis. Patients typically have a marked elevation in liver function test values, severe coagulopathy, and severe hepatic encephalopathy [5]. Contrast-enhanced CT or MR do not allow for an immediate diagnosis but can help find out the underlying etiology of acute hepatic failure. In fact, if no evidence of vessel occlusion or thrombosis is present, the etiology may be most likely related to the changes in portal venous flow.

Encephalopathy after TIPS is probably the most frequent complication related to the procedure; it occurs in 5 to 35% of cases [12] due to a diminished metabolic filtering effect of the liver parenchyma associated with the diversion of portal venous flow through the shunt. Severe incapacitating encephalopathy can occur in 1 to 3% of TIPS patients [5].

At the moment, the diagnosis of encephalopathy is based on clinical data not on diagnostic imaging, although

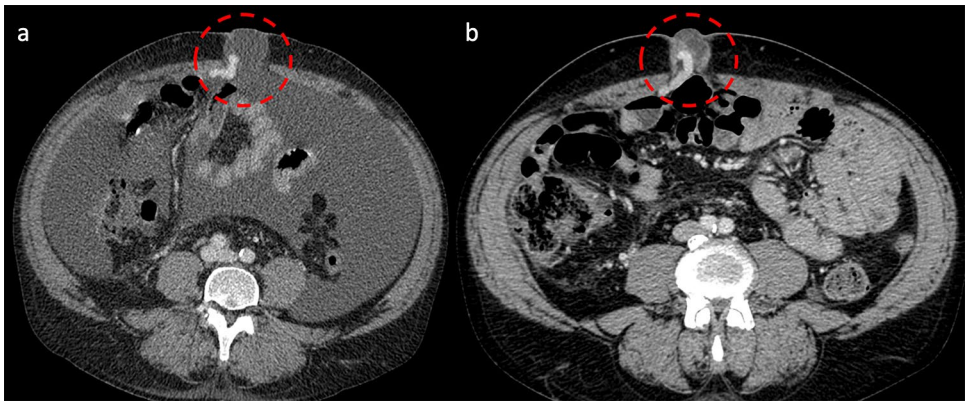


Fig. 19 Hernia incarceration. Axial contrast-enhanced CT images before (**a**) and after (**b**) TIPS procedure for refractory ascites show umbilical hernia (red line). The resolution of massive ascites after TIPS insertion left abdominal fat and vessels entrapped in the hernia with a risk of ischemia or necrosis

functional brain MR imaging seems to have a role in showing abnormalities in neuronal connectivity.

For patients with persistent severe encephalopathy or acute liver failure, TIPS occlusion should be considered. Another choice to mitigate encephalopathy, in place of TIPS occlusion, is the reduction of the stent-graft diameter [5, 33, 34].

TIPSITIS

Tipsitis or endotipsitis is a rare expression of a TIPS stent infection [5, 35, 36, 37]. Some authors reported an incidence of 1% [37]. This complication should be suspected in a patient experiencing sustained, unexplained bacteremia after a TIPS procedure. A wide range of infecting organisms have been isolated, such as *Enterococcus faecalis*, *Staphylococcus aureus, Klebsiella pneumonia*, *Escherichia coli*, *Candida albicans*, *Candida parapsilosis*, *Lactobacillus rhamnosus*, and *Enterobacter cloacae* [5, 37]. Tipsitis can occur early or late after TIPS positioning and is often associated with biliary venous fistulas and cholangitis. Diagnosis can be difficult since no standard criteria exist.

Contrast-enhanced CT or MR do not make a direct diagnosis possible but can help exclude other etiologies of infection. In any case, partial or complete TIPS occlusion with thrombus or vegetations can be identified. If no evidence of infective focus is present on imaging or in clinical data, the etiology is most likely related to tipsitis.

Hernia incarceration

Patients with abdominal and inguinal hernias, who undergo TIPS procedures for refractory ascites, are at increased risk of hernia complications up to 25% [5, 38]. The resolution of massive ascites after TIPS placement can change the intra-abdominal configuration, leaving the bowel entrapped in hernias [5], especially when the hernia sac has a narrow neck. Incarceration refers to an irreducible hernia and is clinically diagnosed when it is not possible to reduce a hernia manually. Detection is important since it predisposes the patient to complications such as obstruction, inflammation, or ischemia (strangulation).

In patients with hernia complications, emergent surgery can be required, including bowel resection for necrosis [38]. CT and MR imaging guides toward an evaluation of the presence of a hernia, its size, location and contents (Fig. 19). Signs of bowel ischemia, caused by a compromised blood supply, are dilated, fluid-filled loops of bowel entrapped within the hernia sac and proximal obstruction, free fluid within the hernia sac, wall thickening, wall enhancement, mesenteric vessel engorgement, mesenteric haziness, and ascites [39].

Conclusions

TIPS is a relatively safe and established procedure for the treatment of portal hypertension complications. However, the morbidity rates can be as high as 20% due to the technical complexity of this intervention. A clear understanding of the procedure and a knowledge of potential complications helps the diagnostic radiologist to reach a prompt and correct diagnosis.

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Declarations

Conflict of interest We declare no conflict of interest.

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Authors and Affiliations

Giuseppe Mamone[1] · Mariapina Milazzo[1] · Ambra Di Piazza[1] · Settimo Caruso[1] · Vincenzo Carollo[1] · Giovanni Gentile[1] · Francesca Crinò[1] · Gianluca Marrone[1] · Gianvincenzo Sparacia[1] · Luigi Maruzzelli[1] · Roberto Miraglia[1]

[1] Radiology Unit, Department of Diagnostic and Therapeutic Services, IRCCS ISMETT (Mediterranean Institute for Transplantation and Advanced Specialized Therapies), via Tricomi 5, 90127 Palermo, Italy