Chapter 8
Liver Transplant Interventions

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Introduction

Initial efforts at human liver transplantation in the early 1960s were marred by extremely poor postoperative survival, with the earliest organ recipients surviving for 0–23 days postoperatively [1]. Following improvements in surgical technique, the focus shifted from postoperative patient survival to survival of the graft, and efforts were concentrated on optimizing antirejection regimens [2]. In the current era, patients undergoing liver transplantation have survival rates of 82%, 70%, and 65% at 1, 5, and 10 years, respectively [3]. Despite significant improvements in survival following liver transplantation, posttransplant complications are not uncommon, and image-guided interventional strategies have emerged as a reasonable alternative to open or laparoscopic reoperation in the management of many of these complications.

Postoperative complications following liver transplantation can be divided into three broad categories: arterial, venous, and nonvascular.

Arterial Complications

Hepatic Artery Thrombosis

Hepatic artery thrombosis (HAT) is the most common arterial complication following liver transplantation, historically affecting 2–12% of liver transplant recipients [4–7]. More recent studies suggest a drop in incidence to 3–5% [8]. HAT is a

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leading cause of graft loss, accounting for up to 53% of cases of liver graft failure and carrying a mortality rate of greater than 80% when untreated [9, 10]. Even with intervention, this complication is fatal in approximately 1/3 of affected recipients [11, 12]. Apart from inherent risks related to surgical technique (e.g., kinking at sites of anastomosis), risk factors for the development of HAT include donor age > 60 years, back-table arterial reconstruction, use of an arterial conduit, cytomegalovirus infection, donor death secondary to cerebrovascular accident, increased cold ischemia time, rejection, ABO blood group incompatibility, and primary sclerosing cholangitis [12–15]. HAT is subdivided into early and late forms, with early HAT typically defined as occurring within 30 days of transplantation. The distinction is an important one as the clinical presentation, urgency of intervention, and impact on graft outcome are different.

The incidence of early HAT in adults ranges from 2.6 to 9% [16]. Early HAT typically occurs prior to the development of any meaningful collateral arterial vasculature, although collateral vessels have been documented angiographically as early as 2 weeks following transplantation [11]. Collateral vessels are largely derived from the phrenic vessels and typically develop 2–4 months following transplantation [16, 17], which may explain the less severe clinical course of late HAT. Early loss of arterial flow to the graft can result in acute fulminant hepatic failure, as well as necrosis of the arterial-dependent biliary ducts and uncontrollable sepsis in the immunocompromised patient [18].

The incidence of late HAT ranges from 1 to 25% [19]. Late HAT typically presents with biliary tract complications with or without fever [4] with a median time to presentation of 6 months [19].

The standard treatment of HAT is liver retransplantation; however, a shortage of available donor organs as well as the associated high mortality with retransplantation has led to a pursuit of alternate treatment options, including surgical thrombectomy (with or without anastomotic revision) and endovascular intervention in the form of intra-arterial thrombolysis (IAT), percutaneous transluminal angioplasty (PTA), and PTA with stent placement [8]. In a study by Duffy et al. [13] evaluating outcomes of vascular complications in 4200 liver transplant recipients, 65% of patients with HAT underwent surgical exploration and thrombectomy with or without anastomotic revision, 17% required retransplantation, and 3% underwent catheter-directed thrombolysis. Despite attempts at revascularization, up to 75% of liver transplant recipients who develop HAT will ultimately require retransplantation [13].

Murata et al. [18] reviewed outcomes of 120 consecutive adult patients who underwent living donor liver transplant. A total of nine patients (7.5%) developed HAT and underwent endovascular treatment with an overall reported technical success rate of 78% (7/9 patients). Intra-arterial thrombolysis (IAT) alone was successful in one patient, six patients were treated with both IAT and percutaneous transluminal angioplasty (PTA), and two patients underwent IAT with PTA and stenting. The two patients failing endovascular management were observed and did not require further intervention. Complications included hepatic arterial rupture ($n = 1$) and arterioportal shunt formation ($n = 1$).

A review by Singhal et al. [20] examined 69 cases of endovascular treatment of HAT collected from 16 separate published studies. Thrombolysis was found to be successful in 68% of cases (47/69). Among the patients successfully treated with thrombolysis, 62% ($n = 29$) required further intervention in the form of PTA alone ($n = 4$), stent placement alone ($n = 20$), and PTA with stent placement ($n = 5$). The most common reported complication was hemorrhage ($n = 18$; 38%), with three cases proving fatal.

Failed endovascular intervention may also complicate subsequent surgical intervention (either in the form or revascularization/anastomotic revision or retransplant) in the early period due to possible catheter-related intimal injury to the artery [18] or at a later period due to the presence of stents at desired anastomotic sites [21].

Sheiner et al. [22] found that graft salvage outcomes were different among patients with HAT who were asymptomatic at time of presentation versus those who were symptomatic (elevated liver function tests, bile leak, sepsis): asymptomatic patients had a graft salvage rate of 81.8% vs. only 40% among those patients with symptomatic HAT. This difference in outcomes led the authors to conclude that attempts at emergent revascularization be reserved for patients with asymptomatic HAT, while those with symptomatic HAT should undergo retransplantation.

Hepatic Artery Stenosis

Unlike HAT, hepatic artery stenosis (HAS) has a more insidious presentation with more gradual graft dysfunction (Fig. 8.1) [23, 24]. The incidence of HAS ranges from 5 to 15% [25] and typically presents within 3 months of transplant. Risk factors for HAS are similar to those of HAT, with the added risk factor of intraoperative clamp injury [26]. An untreated stenosis can progress to HAT in more than half of the patients, and many stenoses are therefore diagnosed concurrently with HAT [13, 23, 27]. Historically, these lesions were treated surgically with resection of the stenotic segment and reanastomosis with or without the use of an aortic conduit graft, interposition vein/artery graft, or vein patch angioplasty [23]. Endovascular treatment with PTA with or without stent placement has since replaced surgical intervention as the treatment of choice for HAS, except in cases of early (≤7 days posttransplant) thrombosis or stenosis where surgical revascularization remains the standard of care [25]. PTA is successful in 85–100% of patients with HAS and has a 1-year reported patency rate ranging from 50 to 90% [13, 27–32].

In a series of 870 patients who underwent liver transplantation, a total of 30 patients (3.4%) developed critical HAS, defined as >50% luminal narrowing, and were treated with endovascular intervention [25]. Patients were not candidates for endovascular intervention within the first 21 days following transplant due to risk of arterial anastomotic rupture and hemorrhage; these patients were therefore managed with systemic anticoagulation and/or antiplatelet agents until 21 days postoperatively and underwent PTA at that time. PTA was successfully completed in 27/30 patients (90%), with 23 patients undergoing PTA with stent placement and 4

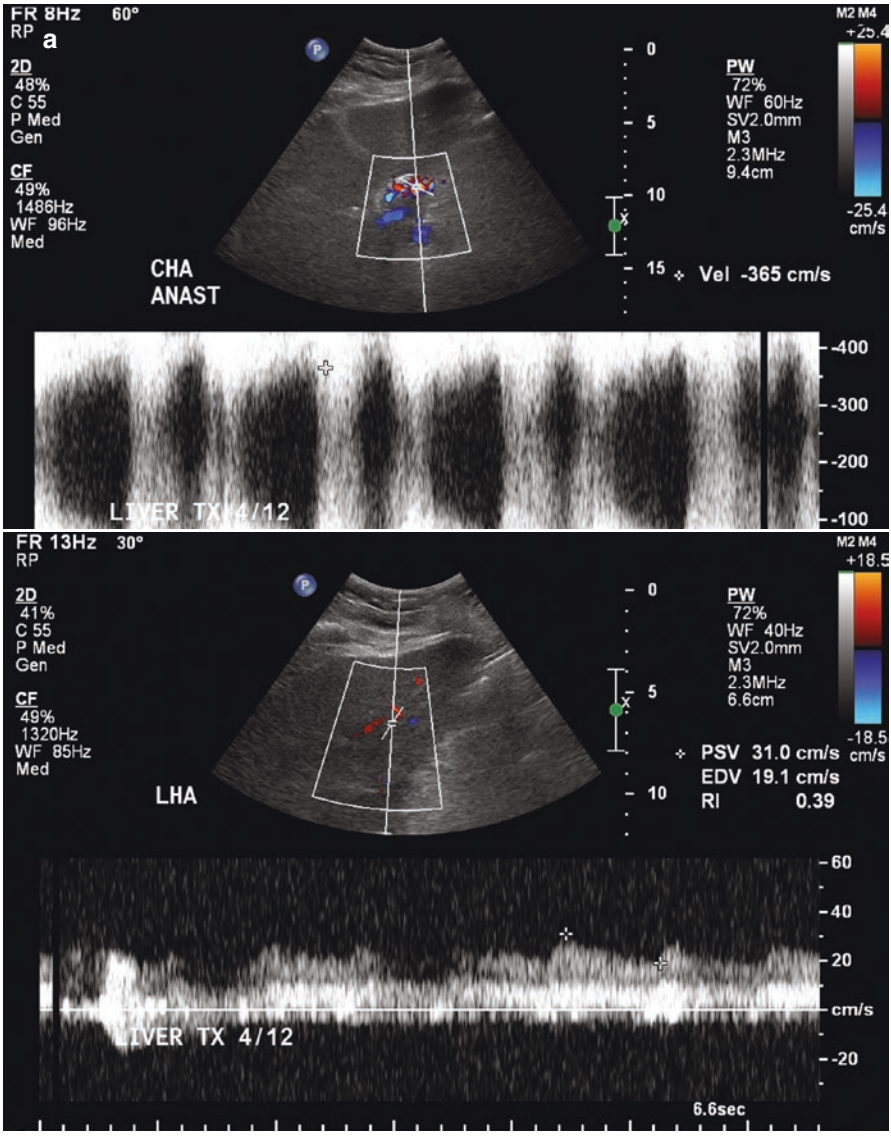

Fig. 8.1 Hepatic arterial stenosis: (**a**) Color doppler images of the transplant arterial vasculature demonstrating findings consistent with high-grade anastomotic stenosis. (**b**) Reconstructed CTA confirming the sonographic findings. (**c**) Pretreatment DSA of the high-grade stenosis. (**d**) Balloon angioplasty of the stenotic segment. (**e**) Post-angioplasty DSA demonstrating improved stenosis

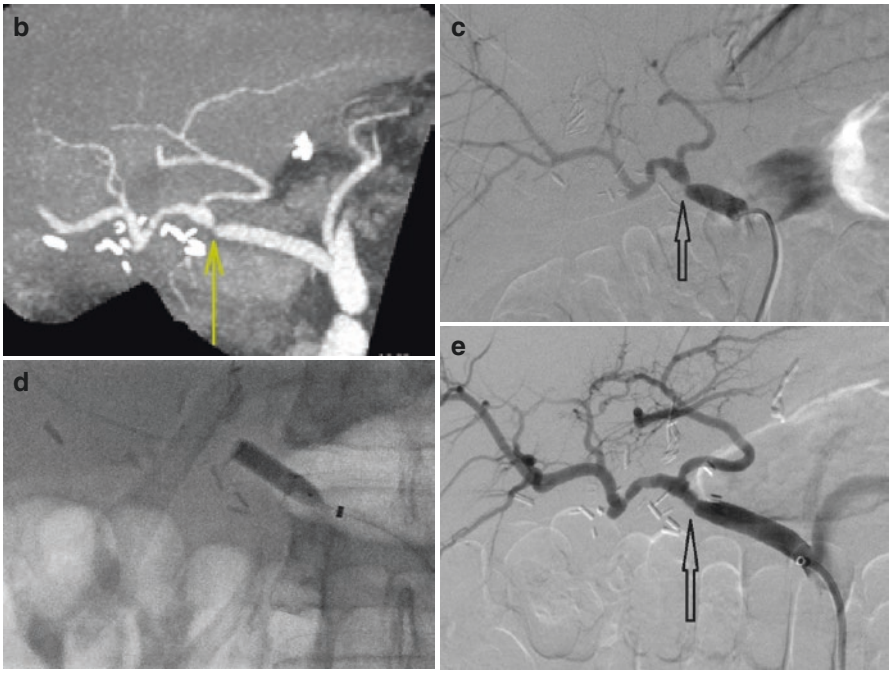

Fig. 8.1 (continued)

undergoing PTA alone. Failed intervention was secondary to an inaccessible hepatic artery in two patients and arterial dissection in one patient. A total of ten patients developed restenosis (33%)—four were restented, three were transplanted due to ischemic cholangitis, and three did not require further intervention. Eight patients were retransplanted following PTA, largely owing to symptomatic ischemic cholangitis (7/8 patients). At 1, 3, and 5 years, overall patency rates were 68%, 62.8%, and 62.8%, respectively.

A meta-analysis comparing treatment outcomes in patients with HAS undergoing either stent placement or PTA alone found similar procedural success rates (98% vs. 89%) and rates of arterial patency at ≥6 months (68% vs. 76%) [31]. There were no significant differences in complication rate or requirement for re-intervention or retransplantation.

Hepatic Artery Pseudoaneurysm

Hepatic artery pseudoaneurysms (HAP) are rare, occurring in 1–2% of liver transplant recipients (Fig. 8.2) [33]. HAP can be subclassified as extrahepatic and intrahepatic pseudoaneurysms. Historically, extrahepatic pseudoaneurysms were thought to be more spontaneous in nature, while iatrogenic pseudoaneurysms were localized to the intrahepatic hepatic arteries [33, 34].

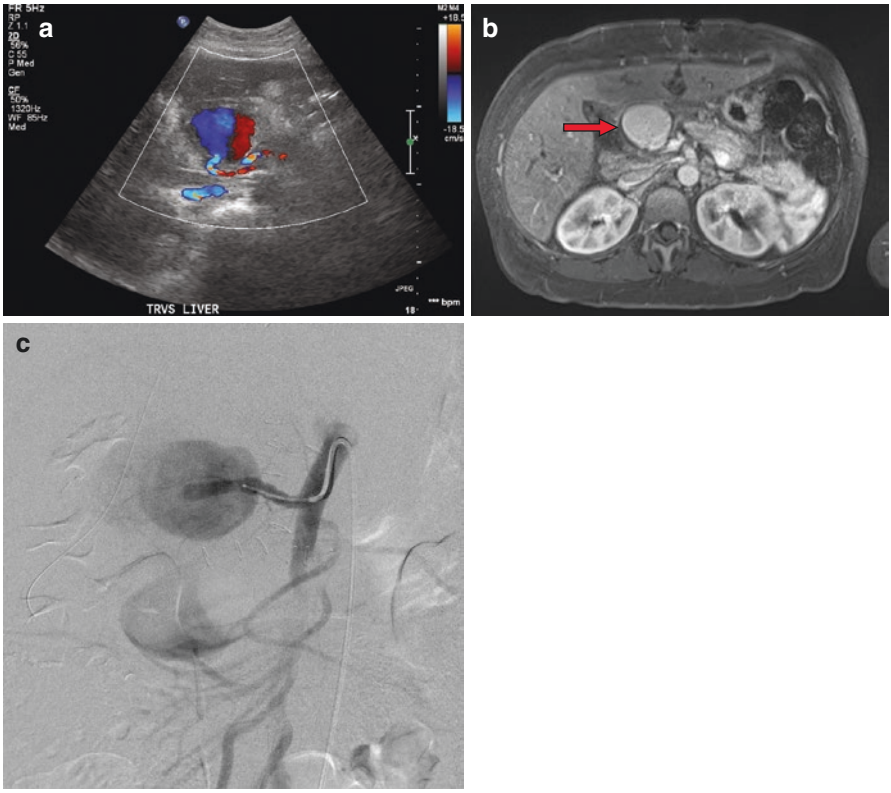

Fig. 8.2 Extrahepatic hepatic artery pseudoaneurysm: (**a**) Sonographic images demonstrating a "yin-yang" sign consistent with a pseudoaneurysm. (**b**) Post-contrast MRI shows the presence of an extrahepatic pseudoaneurysm. (**c**) DSA confirms the presence of the pseudoaneurysm

Extrahepatic pseudoaneurysms typically arise within 30–60 days posttransplant and are generally infectious (mycotic infection, postoperative infectious arteritis) or technical (dissection) in etiology [33, 35–37]. They have been reported to occur more frequently in patients with biliary-enteric anastomoses [33, 34]. Ruptured extrahepatic pseudoaneurysms typically present with hemodynamic instability (50–67%) or gastrointestinal bleeding and hemobilia (22–25%) [33, 38, 39]. Other nonspecific signs may include anemia, elevated liver function tests, and infection/sepsis.

Intrahepatic pseudoaneurysms are often iatrogenic in nature and arise either during or shortly after an interventional procedure (e.g., PTAs, percutaneous transhepatic cholangiography, biopsy) within the intrahepatic hepatic arteries [5, 6, 40]. With the advent of minimally invasive management of both HAT and HAS, an increasing number of extrahepatic pseudoaneurysms may now be classified as iatrogenic in etiology [41].

The standard treatment of HAP is surgical resection with arterial reconstruction, particularly in cases of mycotic infection where interventional efforts may provide a nidus for continued infection [42]. HAPs may be asymptomatic or can rupture into

the GI tract, peritoneum, or biliary system. In cases of active hemorrhage, emergent occlusion of the hepatic artery may be necessary. For non-mycotic extrahepatic HAPs, coil embolization and stent-graft placement are potential endovascular treatment options [5, 6, 33, 40]. In cases of extremely tortuous arterial vasculature, direct percutaneous access may be necessary for embolization and thrombin injection [37].

In a retrospective study by Saad et al. [41], a total of 20 pseudoaneurysms were identified out of 1857 liver transplants (1.1%). Of these 20, 9 were spontaneous pseudoaneurysms (extrahepatic), while the remaining 11 were iatrogenic (intrahepatic; 4 secondary to angioplasty and 7 after biliary intervention). Endovascular management was attempted in 12 patients with a technical success rate of 83% (10/12). In one patient, attempted selective embolization of the HAP resulted in thrombosis of the entire hepatic artery. Unfortunately, despite the relatively high technical success rate, the overall 1-, 3-, and 6-month graft survival rates were 70%, 40%, and 35%, respectively.

Pseudoaneurysms typically present within the first 60 days after transplant, prior to the development of significant arterial collateral vessels, when preserved arterial flow to the graft is critical [34, 36, 39, 43–45]. Incidentally discovered HAPs must be treated due to the high risk of rupture and resultant life-threatening bleeding [35]. During active hemorrhage, occlusion of the hepatic artery, either with surgical ligation or via endovascular intervention, may be necessary, with resultant graft loss [33, 35]. Independent of intervention, HAP confers a high rate of retransplantation (33–45%) and mortality (33–78%) [33, 35, 38, 39, 45–47]. Although surgical resection/revision remains the mainstay for definitive management of HAP, there may be a role for endovascular management in stabilizing or temporizing unstable patients either as a bridge to retransplantation or to allow them to undergo definitive treatment in a more elective setting [41].

Splenic Artery Steal Syndrome

In patients with longstanding liver disease and portal hypertension, splanchnic blood flow can preferentially shunt to regions of lower resistance, namely, the splenic vascular bed, with subsequent development of splenomegaly and enlargement of the splenic artery. When the degree of blood flow shunted away from the high-resistance liver parenchyma becomes clinically significant, this is termed "splenic artery steal syndrome (SASS)" and may result in hepatic failure and/or biliary injury. The diversion of blood flow away from the hepatic vascular bed can persist even after liver transplantation and may even be further augmented by graft injury, rejection, or hepatitis [48]. Proposed treatment options include splenectomy, splenic artery ligation/embolization, or the use of an interposition graft between the common hepatic artery and aorta to drive blood flow back toward the liver [49].

In a series of 350 patients who underwent orthotopic liver transplantation, Uflacker et al. [48] reported a total of 11 patients with liver ischemia secondary to SASS (3.2%). All 11 patients were treated with splenic artery coil embolization, and all demonstrated improvement in both LFTs and clinical parameters within 24 h.

One patient went on to develop hepatic artery thrombosis 24 h after embolization and required surgical intervention with an interposition graft following unsuccessful hepatic artery recanalization.

A larger retrospective series by Nüssler et al. [50] included 1250 consecutive patients who underwent orthotopic liver transplantation and reported a total of 69 cases (5.9%) of SASS via the splenic or gastroduodenal artery. Twenty-five patients were classified as having SASS prior to undergoing liver transplantation, while the remaining 44 patients were diagnosed after transplant. Of the 69 total patients with SASS, 18 were treated with splenectomy, 29 with coil embolization of the splenic or gastroduodenal arteries, and 9 with splenic artery banding.

The highest complication rates were observed in patients diagnosed with SASS after liver transplantation who were subsequently treated with coil embolization. Half of these patients developed local or systemic septic complications, 8 required secondary splenectomy, 7 required retransplantation, and 5 of the 29 patients who underwent coil embolization died (four from graft failure and one from sepsis and multiorgan failure). This high complication rate was attributed to distal placement of embolization coils in the first 15 patients with resultant splenic infarction and abscess. No complications were reported after more proximal placement of embolization coils.

Of the patients treated with splenectomy ($n = 18$), two patients died from biliary complications despite retransplantation. No complications were reported in the remaining 16 patients. Patients treated with splenic artery banding demonstrated normalization of graft perfusion without any reported associated complications.

Thirteen patients had mild symptoms of SASS and were untreated; however, three of these patients subsequently required retransplantation for biliary ischemia.

Technical Management of Arterial Complications

Hepatic Angiography

Initial angiograms are usually performed through a standard trans-femoral approach, although a trans-brachial or trans-radial approach can also be used. A 5-Fr catheter such as a C-2 Glidecath (Terumo, Tokyo, Japan) is used to select the celiac axis. Initial digital subtraction angiograms (DSA) are performed. Further DSA images may be obtained in various obliquities depending on patient anatomy. A cone-beam computed tomography (CBCT) may be of benefit to better identify sites of potential pathology.

Arterial Thrombolysis

If HAT is identified during initial angiography, mechanical and/or pharmacologic thrombolysis can be performed. Depending on the size of the occluded vessel, a 2.4- or 2.8-Fr microcatheter or a 4- or 5-Fr catheter can be advanced over a 0.018″

or 0.035″ guidewire, respectively, into the thrombus. Angiograms performed from this point may allow for evaluation of the extent of the thrombus.

Mechanical thrombolysis can be performed using various techniques including balloon maceration or suction thrombectomy. Pharmacologic thrombolysis is often performed in concert with mechanical thrombolysis. A multi-sidehole infusion catheter is positioned in the thrombus with infusion of a thrombolytic agent such as recombinant tissue plasminogen activator (r-tPA, Alteplase; Genentech, San Francisco, CA) over 12–48 h. Alteplase is infused at a rate of 0.5–1.0 μg/h often in conjunction with a basal rate (250–500 U) of heparin delivered through the sheath. Patients are often admitted to a monitored unit for serial neurologic evaluation. Serial lab values including fibrinogen, PTT and PT/INR, and hemoglobin are also assessed in regular intervals.

Arterial Angioplasty

A long vascular sheath such as a Flexor Ansel Guiding Sheath (Cook Medical, Bloomington, IN) is introduced over a 0.035″ or 0.038″ wire and seated at the celiac artery origin to provide additional support for tracking of an angioplasty balloon. The sheath is sized to accommodate the angioplasty balloon, usually 6- to 8-Fr in size. Initial angiograms can be used to determine the appropriate size of the balloon for angioplasty. Intravenous heparin (3000–5000 U bolus or 50–80 U/kg) should be delivered prior to crossing the HAS/HAT. HAS can be crossed with a 0.035″ hydrophilic guidewire such as a Glidewire (Terumo, Tokyo, Japan) or a 0.010–0.018″ microwire. An over-the-wire or monorail balloon system can be used for angioplasty once the HAS is crossed. Often, serial upsizing of the balloon is performed to reduce potential risks of arterial perforation. If suboptimal results are seen following balloon angioplasty, a scoring or cutting balloon may be utilized followed by a high-pressure balloon. These balloons should be used with caution as they may confer a higher rupture rate.

Arterial Stenting

For inadequate results following angioplasty, or complications such as vessel rupture, a stent may be placed. Given the relative rigidity of stents, a guide catheter should be utilized for delivery. If possible, the guide catheter should be advanced into the common hepatic artery to provide additional support during stent delivery and placement. Bare metal or covered stents can be utilized for persistent HAS; covered stents should be used for pseudoaneurysm or rupture. Self-expanding stents may be utilized for HAS but many prefer to use balloon-mounted stents for more accurate positioning. If a self-expanding stent is used, the stent should be upsized

10–20% above the size of the treatment vessel. Although practice varies, patients are routinely maintained on antiplatelet agents post-stent placement, especially for a covered stent.

Splenic Artery Embolization

Splenic artery embolization is performed after a standard angiogram to delineate the splenic and hepatic arteries. The splenic artery is subselected with a 3–5 Fr catheter. After sizing the vessel, embolization can be performed in the proximal splenic artery. Fibered coils are most often utilized for this purpose; however, vascular plugs can also be used. Care should be taken to perform embolization in the proximal splenic artery as distal embolization can lead to increased complications such as splenic infarction and infection.

Venous Complications

Portal Vein Thrombosis

The incidence of posttransplant portal vein thrombosis (PVT) ranges from 3 to 10.6% and occurs more frequently in pediatric population as well as in recipients with pre-existing portal hypertension, hypoplastic portal veins, donor-recipient vessel size mismatch, graft edema, significant portosystemic collaterals, prior treatment for portal hypertension (e.g., TIPS), intimal injury at time of surgery, and technical problems with the anastomosis (e.g., kinking/twisting, stenosis) [13, 51–55]. The presence of pre-transplant PVT has also historically been associated with a higher incidence of posttransplant PVT and patients with intrahepatic extension of PVT were previously ineligible for transplant [56]. The data supporting the exclusion of these patients is inconsistent, however, with one published report citing no significantly increased risk of posttransplant portal vein rethrombosis relative to controls [57] and others reporting a highly variable incidence of posttransplant rethrombosis ranging from as low as 5% to as high as 40% in some series [56, 58, 59]. Sharma et al. [60] demonstrated that patients with pre-existing extensive PVT ($n = 78$) had a significantly higher rate of non-thrombosis-related graft failure (9.0% vs. 1.3%) and retransplantation (17.9% vs. 7.7%) when compared with controls ($n = 78$). More recently, the decision to offer liver transplant to this patient population has been left to the discretion of the transplant surgical team [61].

PVT typically presents within the first month following liver transplantation (“early”), but may present later (“late”). PVT manifests clinically with symptoms of portal hypertension (e.g., ascites, splenomegaly, variceal bleeding) and with elevation of liver enzymes [21, 62]. Early PVT has been associated with a 100% rate of

graft loss [62]. Endovascular treatment options include catheter-directed thrombolysis (via percutaneous or transcatheter approach) followed by angioplasty with or without stent placement and TIPS creation [8, 62].

Limited data is available on long-term patency rates following interventional treatment of PVT; however, data is available on long-term patency rates for angioplasty with or without stent placement for portal vein stenosis and will be discussed in the following section.

For patients who are not candidates for endovascular intervention, surgical thrombectomy, anastomotic revision, or retransplantation may be necessary [5, 6, 40, 63].

Portal Vein Stenosis

Portal vein stenosis (PVS) affects approximately 5% of liver transplants and almost always occurs at the site of anastomosis [41, 62]. The majority present more than 6 months after liver transplant and manifest clinically with symptoms of portal hypertension (including ascites, splenomegaly) and may progress to PVT without treatment [8, 21, 42]. PVS is more common with split grafts, especially pediatric split grafts where the incidence has been reported to be as high as 27% [62, 64–66], and is attributed to the relative size mismatch between the recipient portal vein and the graft portal vein. Prior splenectomy has also been associated with a fivefold higher rate of PVS [65].

PVS is treated with balloon angioplasty with technical success rates ranging from 36 to 71% at 2–3 years [62, 67]. In cases of concomitant portal vein thrombus, thrombolysis may be performed for 12–48 h prior to angioplasty. If angioplasty is unsuccessful (pressure gradient >5 mmHg across the stenosis), or if there is recoil stenosis or residual stenosis greater than 30%, then a stent may be placed [8] with long-term patency rates reported to be nearly 100% at 3–5 years [62, 68] (Fig. 8.3).

Hepatic Venous Outflow Stenosis and Thrombosis

Venous outflow stenosis or occlusion involves the hepatic veins (HV) or inferior vena cava (IVC) at rates of 4–5% and 1%, respectively [69, 70]. It is more commonly seen in livers that are transplanted in piggyback fashion [71]. Patients with Suprahepatic IVC stenosis may present with ascites, pleural effusion, and/or Budd-Chiari-type symptoms, while stenoses involving the infrahepatic IVC manifest with lower extremity edema. Outflow stenosis or obstruction of the hepatic veins, on the other hand, results in vascular engorgement and passive congestion of the transplant liver. Early complications are usually related to technical issues during

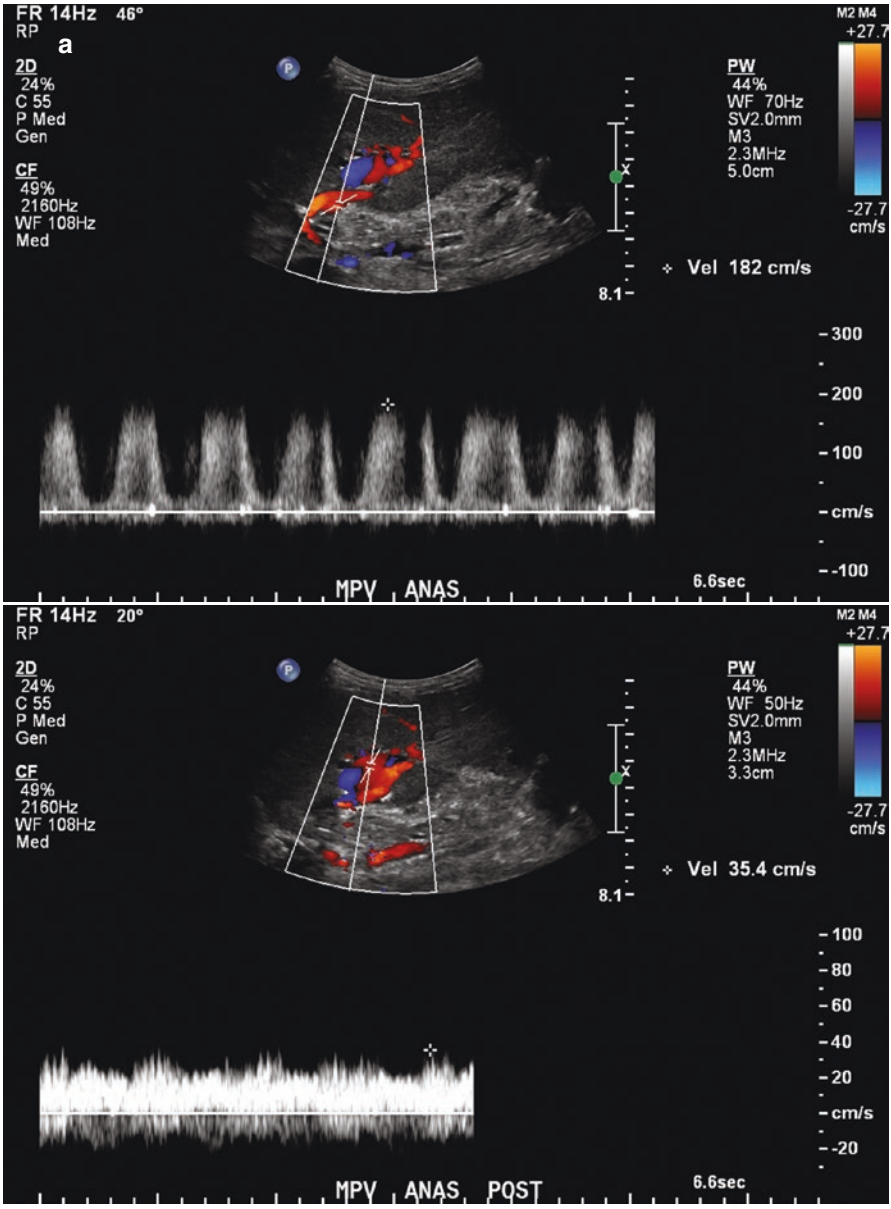

Fig. 8.3 Portal vein stenosis: (**a**) ultrasound demonstrates elevated velocities at the portal vein anastomosis. (**b**) Digital subtraction venography (DSV) of the left portal vein demonstrates stenosis of the main portal vein at the anastomosis (arrow). (**c**) Balloon angioplasty of the portal vein stenosis. (**d**) Post-venoplasty DSV demonstrates marked improvement of the stenosis

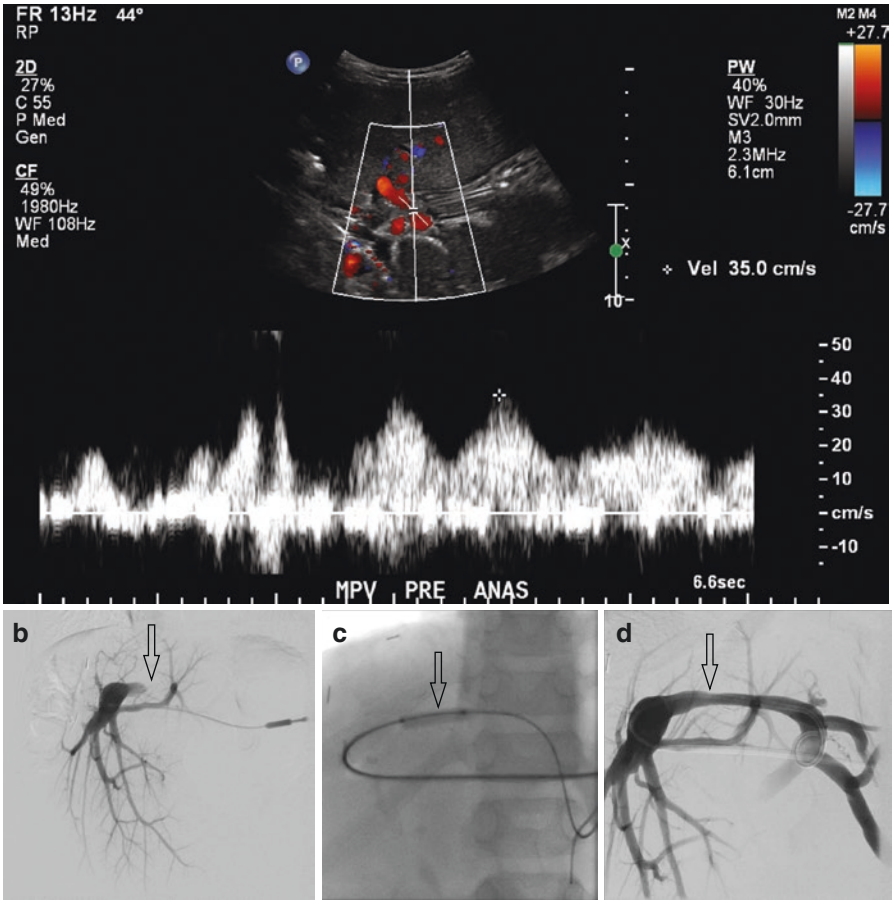

Fig. 8.3 (continued)

transplantation, while late complications are more often attributed to intimal hyperplasia and/or fibrosis at the anastomosis [72] (Fig. 8.4).

The treatment of venous outflow stenosis or thrombosis is similar to PVT/PVS and includes thrombolysis (12–48 h prior to angioplasty, if applicable) and balloon angioplasty with or without stent placement depending on the degree of residual stenosis following balloon dilation [8]. Technical success is defined as a decrease in the trans-anastomotic gradient to less than 3–5 mmHg [72, 73] for both the IVC and HV. The reported patency rate following HV angioplasty is 60% at 1 year, with an increase to nearly 100% with assisted angioplasty [74]. For IVC stenosis/occlusion, patency rates are only 40% at 1 year with angioplasty alone, with an increase to 91% following stent placement [72, 73, 75]. Due to the inferior patency rates with

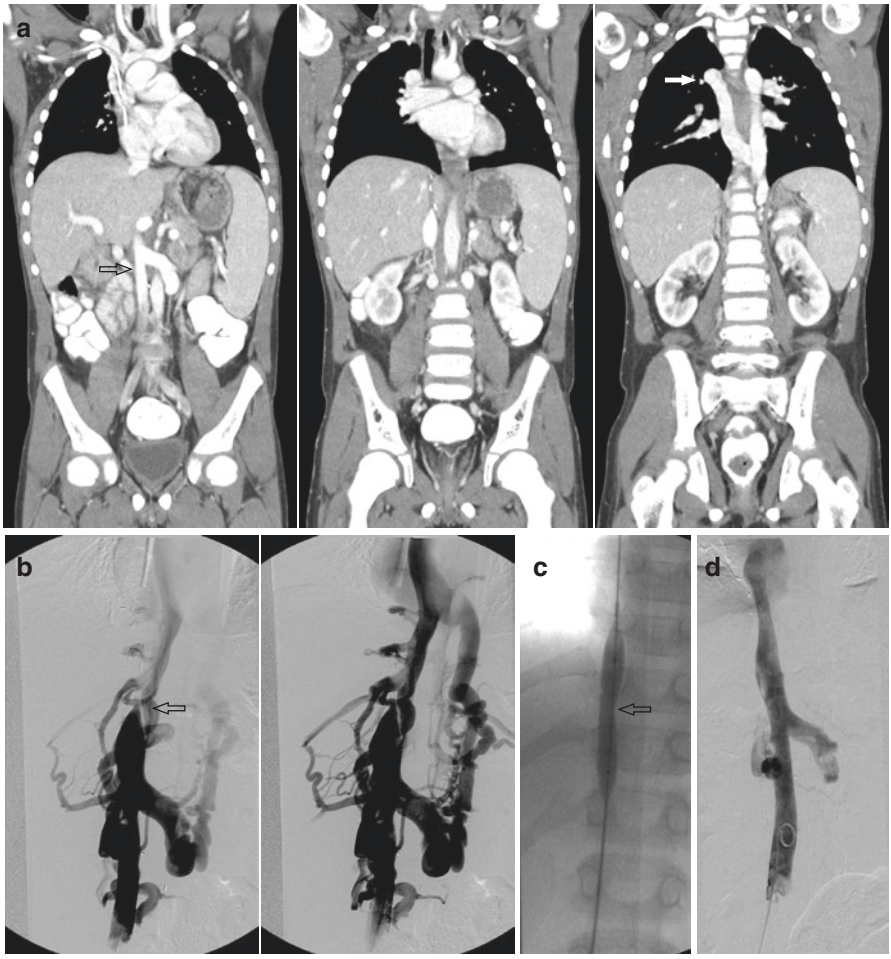

Fig. 8.4 IVC stenosis: (**a**) CT venogram demonstrates a high-grade stenosis of the intrahepatic IVC. (**b**) Conventional venacavagram demonstrates complete occlusion of the IVC and development of extensive collateral veins. (**c**) Venoplasty of the stenotic segment. (**d**) Post-venoplasty image demonstrates no residual stenosis or collateral venous flow

angioplasty alone, primary stenting is recommended for treatment of both HV and IVC stenosis/occlusion [8].

Portal Vein Thrombolysis/Venoplasty

Direct portal vein access is obtained with an 18 or 21 gauge needle under direct sonographic guidance. A trans-splenic route may be used if portal access cannot be obtained due to thrombus. A 0.035″ guidewire is advanced (through a transition set if a 21 gauge needle is used). A 6–8 Fr sheath is advanced over the guidewire. For thrombolysis, a multi-sidehole infusion catheter can be placed to perform a prolonged thrombolysis as previously described. For portal vein stenosis, an angled 5-Fr catheter is advanced through the sheath and, along with a hydrophilic wire, is traversed through the stenotic lesion. The 5 Fr catheter can be exchanged for a pigtail catheter to perform a direct portal venogram and to measure the portal venous pressure gradient. After appropriate sizing of the vessel, a prolonged balloon venoplasty is performed. Intravenous bolus of heparin may be delivered prior to venoplasty. For stenosis that is resistant to balloon venoplasty, an intraluminal stent can be placed as described in the arterial intervention section.

Transjugular Intrahepatic Portal-Systemic Shunt (TIPS) Placement

A TIPS shunt may be created to augment portal venous flow prior to thrombolysis. After obtaining jugular access, a 9 Fr sheath is advanced into the SVC. An angled 5 Fr catheter is used to select the right hepatic vein. Over a stiff wire, a TIPS access needle such as the Colapinto needle (Cook Medical, Bloomington, IN) is advanced. The needle is advanced anteriorly in attempt to select a branch of the portal vein. A partially contrast-filled syringe is attached to the back of the needle, and the needle is retracted while negative syringe pressure is applied. Once blood is aspirated, contrast is injected to confirm positioning of the needle within the portal vein. A wire is advanced through the needle into the portal venous system, and the needle is exchanged for a marking pigtail catheter. Here, direct portal venous pressures are obtained and the portal-systemic pressure gradient is calculated. Simultaneous venograms are performed through the sheath (positioned in the hepatic vein) and the pigtail catheter to measure for the appropriate stent length. The liver tract is balloon dilated—we prefer with an 8 mm diameter balloon—followed by stent deployment. A partially covered stent (Viatorr, Gore Medical, Flagstaff, AZ) is preferred due to superior patency rates over bare metal stents. The transjugular route through the TIPS can then be used to position the multi-sidehole catheter in the portal vein thrombus for thrombolysis.

Nonvascular Complications

Biliary Stricture (Anastomotic and Non-anastomotic)

The overall incidence of biliary stricture following liver transplantation is approximately 13%, as reported by a recent large meta-analysis including 14,000 patients [76]. Symptoms of biliary stricture include jaundice, abdominal pain, cholangitis, and increased liver function tests; some patients, however, may be asymptomatic [8]. Magnetic resonance cholangio-pancreatography (MRCP) can aid in the diagnosis of biliary stricture, as well as delineate the affected areas within the biliary system. Owing to the reliance of the biliary system on hepatic arterial blood supply, patients with known or suspected biliary stricture(s) should be further evaluated for arterial complications with either Doppler ultrasound or CT angiography.

The majority (75%) of biliary strictures occur at the anastomosis and may be attributed to surgical error or technique (e.g., anastomotic breakdown, local tissue ischemia, fibrosis) [77–79]. Patients with early postoperative bile leak have also been shown to be at increased risk for developing anastomotic stricture [80]. Non-anastomotic strictures occur in the setting of graft arterial compromise (hepatic artery thrombosis/stenosis) or other ischemic cholangiopathy (e.g., prolonged cold ischemia time) [81] and are often hilar in location, although they may be present intrahepatically in a diffuse manner.

Endoscopic retrograde cholangio-pancreatography (ERCP) is often attempted initially with cholangioplasty and stent placement [8]. Although treatment success rates can be as high as 90%, multiple treatment sessions are generally needed [77, 78, 82]. For patients with endoscopically inaccessible biliary systems, e.g., those with biliary-enteric anastomoses, percutaneous intervention with transhepatic biliary drain placement and cholangioplasty may be performed. Similar to ERCP intervention, sequential dilatation with or without concomitant catheter drainage is necessary for achieving the best clinical outcome. Patency rates have been reported to be close to 90% at 5 years following transplantation [83, 84] in patients amenable to percutaneous intervention. For the small subset of patients who cannot be managed endoscopically or via percutaneous methods, surgical revision of the anastomosis may be necessary.

Regarding patients with non-anastomotic strictures, treatment success is dependent on the number, location, and severity of strictures [81], with extrahepatic strictures being more likely to respond to therapy. Patients who fail treatment or who develop secondary biliary cirrhosis, recurrent cholangitis, or progressive cholestasis may require retransplantation.

Biliary Leak

Bile leaks have been reported in up to 25% of patients undergoing liver transplantation and can be classified as either early (within 4 weeks of transplant) or late (more than 4 weeks after transplant) [80, 85–87]. They generally occur at the anastomosis and may be secondary to T-tube removal, ischemia, or downstream obstruction (including sphincter of Oddi dysfunction) [88] or can occur at the cystic duct stump or along the cut edge of the graft in living donor liver transplant [89]. Patients with bile leak typically present with abdominal pain, fever, and signs of peritonitis, although these symptoms may be masked by corticosteroid administration.

Biliary leaks, including non-anastomotic leaks and communicating bilomas, may be successfully treated with diversion, either via ERCP with sphincterotomy and stent placement or with percutaneous transhepatic cholangiography and stent placement in patients not amenable to ERCP due to anatomic considerations. These interventions have been reported to be successful in 85–100% of cases [90].

Non-communicating bilomas may be treated with antibiotics with or without accompanying percutaneous drainage [76, 77]. Finally, biliary leaks occurring secondary to ischemia are more difficult to treat as the underlying cause may not be amenable to endoscopic or percutaneous intervention; in these cases, surgical intervention may be necessary for definitive management [80].

Percutaneous Transhepatic Cholangiogram (PTC) and Cholangioplasty

Pre-procedural antibiotics should be administered to reduce the risk of cholangitis. PTC can be performed through the left or the right lobe of the liver. Direct access to the biliary tree can be obtained with a 21 gauge needle under sonographic guidance. If the bile duct is not visible, the needle can be advanced under fluoroscopic guidance especially if accessing the right liver lobe. For a right liver lobe access, the

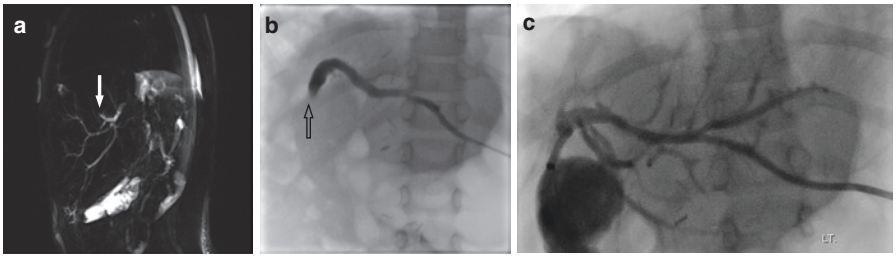

Fig. 8.5 Bile duct stricture: (**a**) MRCP demonstrating bile duct stricture with more proximal dilatation of the intrahepatic bile duct. (**b**) This finding was confirmed on PTC. (**c**) Biliary drain placed for decompression and dilation of stricture with contrast flowing through the bile duct into the duodenum

access site should be below the tenth intercostal space and anterior to the mid-axillary line to reduce the risk of pneumothorax. The needle is then slowly retracted with contrast administration under fluoroscopic guidance to demonstrate filling of the biliary tree. Once the needle tip is confirmed to be within the biliary tree, a 0.018″ wire is advanced into the biliary system. A transition set is then advanced over the wire. We prefer to perform a complete cholangiogram through the outer catheter of the transition set to assess areas of stricture or leak. A combination of an angled catheter and hydrophilic wire can be used to cross areas of stricture or leak. For lesions which are difficult to traverse, an external drain may be placed for decompression with a reattempt at crossing the lesion in 1–2 weeks. Once the stricture is crossed, an internal/external biliary drain is placed (Fig. 8.5).

For bile leaks, repeat cholangiograms may be performed every 6–12 weeks to assess for sealing of the leak. For biliary stenosis, serial cholangioplasties may be performed every 4–6 weeks. With each cholangioplasty, the drain may be exchanged for a larger diameter catheter. This may be a prolonged process, and patients should expect to have a drain in place for weeks to months. Once there is evidence of resolution of the stricture, the internal/external biliary drain is exchanged for an external biliary drain. The patient is then reevaluated in 1–2 weeks to ensure resolution of the stricture at which point the drain may be removed.

References

1. Starzl TE, Iwatsuki S, Van Thiel DH, Carlton Gartner J, Zitelli B, Malatack J, et al. Evolution of liver transplantation. Hepatology. 1982;2:614–36.
2. Starzl TE, Klintmalm GB, Porter K, Iwatsuki S, Schroeter G. Liver transplantation with use of cyclosporin a and prednisone. N Engl J Med. 1981;305:266–9.
3. Agopian VG, Petrowsky H, Kaldas FM, Zarrinpar A, Farmer DG, Yersiz H, et al. The evolution of liver transplantation during 3 decades: analysis of 5347 consecutive liver transplants at a single center. Ann Surg. 2013;258:409–21.
4. Pareja E, Cortes M, Navarro R, Sanjuan F, López R, Mir J. Vascular complications after orthotopic liver transplantation: hepatic artery thrombosis. Transplant Proc. 2010;42(8):2970–2.
5. Bhargava P, Vaidya S, Dick AAS, Dighe M. Imaging of orthotopic liver transplantation: self-assessment module. Am J Roentgenol. 2011;196(3 Suppl):S35–8.
6. Singh AK, Nachiappan AC, Verma HA, Uppot RN, Blake MA, Saini S, et al. Postoperative imaging in liver transplantation: what radiologists should know. Radiographics. 2010;30:339.
7. Crossin JD, Muradali D, Wilson SR. US of liver transplants: normal and abnormal. Radiographics. 2003;23:1093.
8. Ingraham CR, Montenovo M. Interventional and surgical techniques in solid organ transplantation. Radiol Clin North Am. 2016;54(2):267–80.
9. Tzakis AG. The dearterialized liver graft. Semin Liver Dis. 1985;5(4):375–6.
10. Langnas AN, Marujo W, Stratta RJ, Wood RP, Li SJ, Shaw BW. Hepatic allograft rescue following arterial thrombosis. Role of urgent revascularization. Transplantation. 1991;51(1):86–90.
11. Wozney P, Zajko AB, Bron KM, Point S, Starzl TE. Vascular complications after liver transplantation: a 5-year experience. AJR Am J Roentgenol. 1986;147(4):657–63.
12. Bekker J, Ploem S, De Jong KP. Early hepatic artery thrombosis after liver transplantation: a systematic review of the incidence, outcome and risk factors. Am J Transplant. 2009;9(4):746–57.

13. Duffy JP, Hong JC, Farmer DG, Ghobrial RM, Yersiz H, Hiatt JR, et al. Vascular complications of orthotopic liver transplantation: experience in more than 4,200 patients. J Am Coll Surg. 2009;208(5):896–903.
14. López-Andújar R, Moya A, Montalvá E, Berenguer M, De Juan M, San Juan F, et al. Lessons learned from anatomic variants of the hepatic artery in 1,081 transplanted livers. Liver Transpl. 2007;13(10):1401–4.
15. Oh CK, Pelletier SJ, Sawyer RG, Dacus AR, McCullough CS, Pruett TL, et al. Uni- and multivariate analysis of risk factors for early and late hepatic artery thrombosis after liver transplantation. Transplantation. 2001;71(6):767–72.
16. Stange BJ, Glanemann M, Nuessler NC, Settmacher U, Steinmüller T, Neuhaus P. Hepatic artery thrombosis after adult liver transplantation. Liver Transpl. 2003;9(6):612–20.
17. Heffron TG, Pillen T, Welch D, Smallwood GA, Redd D, Romero R. Hepatic artery thrombosis in pediatric liver transplantation. Transplant Proc. 2003;35(4):1447–8.
18. Murata Y, Mizuno S, Kato H, Tanemura A, Kuriyama N, Azumi Y, et al. Technical feasibility and clinical outcomes of interventional endovascular treatment for hepatic artery thrombosis after living-donor liver transplantation. Transplant Proc. 2016;48:1142–8.
19. Gunsar F, Rolando N, Pastacaldi S, Patch D, Raimondo ML, Davidson B, et al. Late hepatic artery thrombosis after orthotopic liver transplantation. Liver Transpl. 2003;9(6):605–11.
20. Singhal A, Stokes K, Sebastian A, Wright HI, Kohli V. Endovascular treatment of hepatic artery thrombosis following liver transplantation. Transpl Int. 2010;23:245–56.
21. Amesur NB, Zajko AB. Interventional radiology in liver transplantation. Liver Transpl. 2006;12:330–51.
22. Sheiner PA, Varma CV, Guarrera JV, Cooper J, Garatti M, Emre S, et al. Selective revascularization of hepatic artery thromboses after liver transplantation improves patient and graft survival. Transplantation. 1997;64(9):1295–9.
23. Abbasoglu O, Levy MF, Vodapally MS, Goldstein RM, Husberg BS, Gonwa TA, et al. Hepatic artery stenosis after liver transplantation—incidence, presentation, treatment, and long term outcome. Transplantation. 1997;63:250–5.
24. Gordon S, Carmody A. Arterial complications after liver transplantation. Transplant. Liver. Philadelphia: Elsevier; 2005. p. 953–61.
25. Rajakunnu M, Awad S, Ciacio O, Pittau G, Adam R, Cunha A, et al. Intention-to-treat analysis of percutaneous endovascular treatment of hepatic artery stenosis after orthotopic liver transplantation. Liver Transpl. 2016;22:923–33.
26. Frongillo F, Lirosi MC, Nure E, Inchingolo R, Bianco G, Silvestrini N, et al. Diagnosis and management of hepatic artery complications after liver transplantation. Transplant Proc. 2015;47(7):2150–5.
27. Saad WEA, Davies MG, Sahler L, Lee DE, Patel NC, Kitanosono T, et al. Hepatic artery stenosis in liver transplant recipients: primary treatment with percutaneous transluminal angioplasty. J Vasc Interv Radiol. 2005;16(6):795–805.
28. Laštovičková J, Peregrin J. Percutaneous transluminal angioplasty of hepatic artery stenosis in patients after orthotopic liver transplantation: mid-term results. Cardiovasc Intervent Radiol. 2011;34(6):1165–71.
29. Maruzzelli L, Miraglia R, Caruso S, Milazzo M, Mamone G, Gruttadauria S, et al. Percutaneous endovascular treatment of hepatic artery stenosis in adult and pediatric patients after liver transplantation. Cardiovasc Intervent Radiol. 2010;33(6):1111–9.
30. Sommacale D, Aoyagi T, Dondero F, Sibert A, Bruno O, Fteriche S, et al. Repeat endovascular treatment of recurring hepatic artery stenoses in orthotopic liver transplantation. Transpl Int. 2013;26(6):608–15.
31. Rostambeigi N, Hunter D, Duval S, Chinnakotla S, Golzarian J. Stent placement versus angioplasty for hepatic artery stenosis after liver transplant: a meta-analysis of case series. Eur Radiol. 2013;23(5):1323–34.

32. Huang M, Shan H, Jiang Z, Li Z, Zhu K, Guan S, et al. The use of coronary stent in hepatic artery stenosis after orthotopic liver transplantation. Eur J Radiol. 2006;60(3):425–30.
33. Marshall MM, Muiesan P, Srinivasan P, Kane PA, Rela M, Heaton ND, et al. Hepatic artery pseudoaneurysms following liver transplantation: incidence, presenting features and management. Clin Radiol. 2001;56(7):579–87.
34. Saad WEA. Management of nonocclusive hepatic artery complications after liver transplantation. Tech Vasc Interv Radiol. 2007;10(3):221–32.
35. Álamo JM, Gómez MA, Tamayo MJ, Socas M, Valera Z, Robles JA, et al. Mycotic pseudoaneurysms after liver transplantation. Transplant Proc. 2005;37(3):1512–4.
36. Bonham C, Kapur S, Geller D, Fung J, Pinna A. Excision and immediate revascularization for hepatic artery pseudoaneurysm following liver transplantation. Transplant Proc. 1999; 31:443.
37. Patel JV, Weston MJ, Kessel DO, Prasad R, Toogood GJ, Robertson I. Hepatic artery pseudoaneurysm after liver transplantation: treatment with percutaneous thrombin injection. Transplantation. 2003;75(10):1755–7.
38. Fistouris J, Herlenius G, Bäckman L, Olausson M, Rizell M, Mjörnstedt L, et al. Pseudoaneurysm of the hepatic artery following liver transplantation. Transplant Proc. 2006;38(8):2679–82.
39. Leelaudomlipi S, Bramhall SR, Gunson BK, Candinas D, Buckels JAC, McMaster P, et al. Hepatic-artery aneurysm in adult liver transplantation. Transpl Int. 2003;16(4):257–61.
40. Caiado AHM, Blasbalg R, Marcelino ASZ, da Cunha Pinho M, Chammas MC, da Costa Leite C, et al. Complications of liver transplantation: multimodality imaging approach. Radiographics. 2007;27:1401.
41. Saad WEA. Liver transplant-related vascular disease. In: Dake M, Geshwind J-F, editors. Abrams angiography: interventional radiology. 3rd ed. Philadelphia: Lippincott Williams & Wilkins; 2012.
42. Baheti AD, Sanyal R, Heller MT, Bhargava P. Surgical techniques and imaging complications of liver transplant. Radiol Clin North Am. 2016;54(2):199–215.
43. Finley DS, Hinojosa MW, Paya M, Imagawa DK. Hepatic artery pseudoaneurysm: a report of seven cases and a review of the literature. Surg Today. 2005;35(7):543–7.
44. Lowell JA, Coopersmith CM, Shenoy S, Howard TK. Unusual presentations of nonmycotic hepatic artery pseudoaneurysms after liver transplantation. Liver Transpl Surg. 1999;5(3):200–3.
45. Stange B, Settmacher U, Glanemann M, Nuessler N, Bechstein W, Neuhaus P. Aneurysms of the hepatic artery after liver transplantation. Transplant Proc. 2000;32:533–4.
46. Houssin D, Ortega D, Richardson A, Ozier Y, Stephan H, Soffer M, et al. Mycotic aneurysm of the hepatic artery complicating human liver transplantation. Transplantation. 1988;46: 469–72.
47. Jones VS, Chennapragada MS, Lord DJE, Stormon M, Shun A. Post-liver transplant mycotic aneurysm of the hepatic artery. J Pediatr Surg. 2008;43(3):555–8.
48. Uflacker R, Selby JB, Chavin K, Rogers J, Baliga P. Transcatheter splenic artery occlusion for treatment of splenic artery steal syndrome after orthotopic liver transplantation. Cardiovasc Intervent Radiol. 2002;25(4):300–6.
49. De Carlis L, Sansalone C, Rondinara G, Belli L, Rimoldi P, Romani F, et al. Splenic artery steal syndrome after orthotopic liver transplantation: diagnosis and treatment. Transplant Proc. 1993;25:2594–6.
50. Nüssler NC, Settmacher U, Haase R, Stange B, Heise M, Neuhaus P. Diagnosis and treatment of arterial steal syndromes in liver transplant recipients. Liver Transpl. 2003;9(6):596–602.
51. Yerdel MA, Gunson B, Mirza D, Karayalçin K, Olliff S, Buckels J, et al. Portal vein thrombosis in adults undergoing liver transplantation: risk factors, screening, management, and outcome. Transplantation. 2000;69(9):1873–81.
52. Jia Y-P, Lu Q, Gong S, Ma B-Y, Wen X-R, Peng Y-L, et al. Postoperative complications in patients with portal vein thrombosis after liver transplantation: evaluation with Doppler ultrasonography. World J Gastroenterol. 2007;13(34):4636–40.

53. Ueda M, Egawa H, Ogawa K, Uryuhara K, Fujimoto Y, Kasahara M, et al. Portal vein complications in the long-term course after pediatric living donor liver transplantation. Transplant Proc. 2005;37(2):1138–40.
54. Woo DH, LaBerge JM, Gordon RL, Wilson MW, Kerlan RK. Management of portal venous complications after liver transplantation. Tech Vasc Interv Radiol. 2007;10(3):233–9.
55. Lladó L, Fabregat J, Castellote J, Ramos E, Torras J, Jorba R, et al. Management of portal vein thrombosis in liver transplantation: influence on morbidity and mortality. Clin Transplant. 2007;21(6):716–21.
56. Manzanet G, Sanjuán F, Orbis P, López R, Moya A, Juan M, et al. Liver transplantation in patients with portal vein thrombosis. Liver Transpl. 2001;7(2):125–31.
57. Enestvedt BK, Enestvedt CK, Diggs B, Orloff SL. Hypercoagulability in liver transplant recipients: does portal vein thrombosis predict postoperative thrombotic complications? Open Journal of Organ Transplant and Surgery. 2011;1:1–7.
58. Tao Y-F, Teng F, Wang Z-X, Guo W-Y, Shi X-M, Wang G-H, et al. Liver transplant recipients with portal vein thrombosis: a single center retrospective study. Hepatobiliary Pancreat Dis Int. 2009;8(1):34–9.
59. Gimeno FA, Calvo J, Loinaz C, Meneu JC, Pérez B, Gomez R, et al. Comparative analysis of the results of orthotopic liver transplantation in patients with and without portal vein thrombosis. Transplant Proc. 2005;37(9):3899–903.
60. Sharma R, Kashyap R, Jain A, Safadjou S, Graham M, Dwivedi AK, et al. Surgical complications following liver transplantation in patients with portal vein thrombosis—a single-center perspective. J Gastrointest Surg. 2010;14(3):520–7.
61. Feltracco P, Barbieri S, Cillo U, Zanus G, Senzolo M, Ori C. Perioperative thrombotic complications in liver transplantation. World J Gastroenterol. 2015;21(26):8004–13.
62. Saad WEA. Portal interventions in liver transplant recipients. Semin Intervent Radiol. 2012;29(2):99–104.
63. Quiroga S, Sebastià MC, Margarit C, Castells L, Boyé R, Alvarez-Castells A. Complications of orthotopic liver transplantation: spectrum of findings with helical CT. Radiographics. 2001;21:1085.
64. Lerut J, Tzakis AG, Bron K, Gordon RD, Iwatsuki S, Esquivel CO, et al. Complications of venous reconstruction in human orthotopic liver transplantation. Ann Surg. 1987;205(4):404–14.
65. Settmacher U, Nuessler N, Glanemann M, Haase R, Heise M, Bechstein W, et al. Venous complications after orthotopic liver transplantation. Clin Transplant. 2000;14:235–41.
66. Buell JF, Funaki B, Cronin DC, Yoshida A, Perlman MK, Lorenz J, et al. Long-term venous complications after full-size and segmental pediatric liver transplantation. Ann Surg. 2002;236(5):658–66.
67. Funaki B, Rosenblum JD, Leef JA, Zaleski GX, Farrell T, Lorenz J, et al. Percutaneous treatment of portal venous stenosis in children and adolescents with segmental hepatic transplants: long-term results. Radiology. 2000;215(1):147–51.
68. Ko GY, Sung KB, Yoon HK, Lee SG. Early posttransplantation portal vein stenosis following living donor liver transplantation: percutaneous transhepatic primary stent placement. Liver Transpl. 2007;13(4):530–6.
69. Brouwers MA, de Jong KP, Peeters PM, Bijleveld CM, Klompmaker IJ, Slooff MJ. Inferior vena cava obstruction after orthotopic liver transplantation. Clin Transplant. 1994;8(1):19–22.
70. Cheng YF, Chen CL, Huang TL, Chen TY, Chen YS, Wang CC, et al. Angioplasty treatment of hepatic vein stenosis in pediatric liver transplants: long-term results. Transpl Int. 2005;18(5):556–61.
71. Navarro F, Le Moine MC, Fabre JM, Belghiti J, Cherqui D, Adam R, et al. Specific vascular complications of orthotopic liver transplantation with preservation of the retrohepatic vena cava: review of 1361 cases. Transplantation. 1999;68(5):646–50.
72. Darcy MD. Management of venous outflow complications after liver transplantation. Tech Vasc Interv Radiol. 2007;10(3):240–5.

73. Wang SL, Sze DY, Busque S, Razavi MK, Kee ST, Frisoli JK, et al. Treatment of hepatic venous outflow obstruction after piggyback liver transplantation. Radiology. 2005;236(1):352–9.
74. Kubo T, Shibata T, Itoh K, Maetani Y, Isoda H, Hiraoka M, et al. Outcome of percutaneous transhepatic venoplasty for hepatic venous outflow obstruction after living donor liver transplantation. Radiology. 2006;239(1):285–90.
75. Weeks SM, Gerber DA, Jaques PF, Sandhu J, Johnson MW, Fair JH, et al. Primary Gianturco stent placement for inferior vena cava abnormalities following liver transplantation. J Vasc Interv Radiol. 2000;11(2 Pt 1):177–87.
76. Akamatsu N, Sugawara Y, Hashimoto D. Biliary reconstruction, its complications and management of biliary complications after adult liver transplantation: a systematic review of the incidence, risk factors and outcome. Transpl Int. 2011;24(4):379–92.
77. Atwal T, Pastrana M, Sandhu B. Post-liver transplant biliary complications. J Clin Exp Hepatol. 2012;2(1):81–5.
78. Kothary N, Patel AA, Shlansky-Goldberg RD. Interventional radiology: management of biliary complications of liver transplantation. Semin Intervent Radiol. 2004;21(4):297–308.
79. Verdonk RC, Buis CI, Porte RJ, van der Jagt EJ, Limburg AJ, van den Berg AP, et al. Anastomotic biliary strictures after liver transplantation: causes and consequences. Liver Transpl. 2006;12(5):726–35.
80. Londoño MC, Balderramo D, Cárdenas A. Management of biliary complications after orthotopic liver transplantation: the role of endoscopy. World J Gastroenterol. 2008;14(4):493–7.
81. Kochhar G, Parungao JM, Hanouneh IA, Parsi MA. Biliary complications following liver transplantation. World J Gastroenterol. 2013;19(19):2841–6.
82. Rerknimitr R, Sherman S, Fogel EL, Kalayci C, Lumeng L, Chalasani N, et al. Biliary tract complications after orthotopic liver transplantation with choledochocholedochostomy anastomosis: endoscopic findings and results of therapy. Gastrointest Endosc. 2002;55(2):224–31.
83. Sung RS, Campbell DA, Rudich SM, Punch JD, Shieck VL, Armstrong JM, et al. Long-term follow-up of percutaneous transhepatic balloon cholangioplasty in the management of biliary strictures after liver transplantation. Transplantation. 2004;77(1):110–5.
84. Roumilhac D, Poyet G, Sergent G, Declerck N, Karoui M, Mathurin P, et al. Long-term results of percutaneous management for anastomotic biliary stricture after orthotopic liver transplantation. Liver Transpl. 2003;9(4):394–400.
85. Thuluvath PJ, Pfau PR, Kimmey MB, Ginsberg GG. Biliary complications after liver transplantation: the role of endoscopy. Endoscopy. 2005;37(9):857–63.
86. Stratta RJ, Wood RP, Langnas AN, Hollins RR, Bruder KJ, Donovan JP, et al. Diagnosis and treatment of biliary tract complications after orthotopic liver transplantation. Surgery. 1989;106(4):675–83; discussion 683–4.
87. Greif F, Bronsther OL, Van Thiel DH, Casavilla A, Iwatsuki S, Tzakis A, et al. The incidence, timing, and management of biliary tract complications after orthotopic liver transplantation. Ann Surg. 1994;219(1):40–5.
88. Scanga AE, Kowdley KV. Management of biliary complications following orthotopic liver transplantation. Curr Gastroenterol Rep. 2007;9(1):31–8.
89. Macías-Gómez C, Dumonceau J-M. Endoscopic management of biliary complications after liver transplantation: an evidence-based review. World J Gastrointest Endosc. 2015;7(6):606–16.
90. Morelli J, Mulcahy HE, Willner IR, Cunningham JT, Draganov P. Long-term outcomes for patients with post-liver transplant anastomotic biliary strictures treated by endoscopic stent placement. Gastrointest Endosc. 2003;58(3):374–9.