Liver transplantation is now frequently used in the treatment of end-stage liver disease. Therefore, it is important that radiologists be aware of common anastomotic techniques and expected postoperative imaging findings. Imaging is most useful in evaluating for posttransplantation complications, which are broadly classified into vascular, biliary, and other complications. Hepatic artery thrombosis is the most significant complication and is often associated with graft failure. Radiologists have multiple modalities at their disposal for optimal evaluation. Doppler ultrasonography (US) is the preliminary imaging modality for gross evaluation of the liver parenchyma, biliary tree, and vasculature for abnormalities. When US findings are indeterminate or there is persistent clinical suspicion for an abnormality, computed tomography (CT) is often performed. The major indications for CT are detection of bile leak, hemorrhage, and abscess, but CT is also useful in the assessment of the vasculature. T-tube cholangiography and magnetic resonance cholangiopancreatography are the best noninvasive imaging tools for evaluating for biliary stricture. Some investigators would argue that endoscopic retrograde cholangiopancreatography (ERCP) is a better diagnostic imaging modality; however, ERCP is invasive. Hepatobiliary scintigraphy is optimal for the evaluation of biliary leakage. Early detection of posttransplantation complications will help lower morbidity rates and will likely allow graft salvage in selected cases.

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Abbreviations: CBD = common bile duct, ERCP = endoscopic retrograde cholangiopancreatography, IVC = inferior vena cava

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Introduction

The world’s first successful liver transplantation was performed by Dr. Thomas Starzl in the 1960s (1). Since then, liver transplantation has been performed in an increasing number of patients with liver dysfunction due to chronic liver disease and acute liver failure (2). Liver transplant recipients are followed up closely after surgery to evaluate for acute rejection and other complications. Acute rejection has a nonspecific clinical manifestation and can be definitively diagnosed only with graft biopsy (3). Therefore, the role of noninvasive imaging is to exclude other complications that can clinically mimic acute rejection.

To understand these complications, it is first important to know that orthotopic liver transplantation requires donor-to-recipient surgical anastomosis of the hepatic artery, portal vein, inferior vena cava (IVC), and bile duct (Figs 1, 2). Dysfunction at these anastomotic sites often results in transplant dysfunction. It is also important to recognize the normal posttransplantation imaging findings, including right-sided pleural effusion, minimal ascites, perihepatic hematoma, and periportal edema, all of which should resolve within a few weeks (4).

Posttransplantation complications are classified into vascular, biliary, and other complications (Fig 3). Vascular complications include hepatic artery thrombosis-stenosis, pseudoaneurysm, hepatic infarct, portal vein thrombosis-stenosis, and IVC or hepatic vein stenosis-thrombosis. After rejection, vascular complications are the second most common cause of graft failure and should be considered in patients with liver failure, bile leak, abdominal bleeding, or septicemia (5). Biliary complications include bile duct obstruction, anastomotic stenosis, bile duct stricture, stone formation, bile leak, biloma, biliary necrosis, and cholangitis. Other complications of liver transplantation include hematoma, abscess, infection, recurrent hepatitis, portal hypertension, splenic infarct, recurrent malignancy, and posttransplantation lymphoproliferative disorder.

In this article, we depict the spectrum of findings seen at ultrasonography (US), CT, MR imaging, cholangiography, angiography, and scintigraphy in patients with posttransplantation complications involving the hepatic artery, portal vein, IVC, and hepatic vein, as well as biliary complications (biliary obstruction, choledocholithiasis, bile duct stricture, bile duct leak) and other complications. In addition, we provide a brief overview of percutaneous interventional procedures that can be used in this setting (Table).
Figure 2. (a) Computed tomographic (CT) angiogram shows a normal anastomosis of the donor celiac artery (curved arrow) to the recipient common hepatic artery (straight arrow) at the takeoff of the gastroduodenal artery. (b) Magnetic resonance (MR) angiogram shows a normal anastomosis of the right hepatic vein to the IVC (arrow). (c) Endoscopic retrograde cholangiopancreatographic (ERCP) image shows a biliary anastomosis (arrow) in the midportion of the common bile duct (CBD). (d) Spectral Doppler ultrasonographic (US) image shows normal portal venous flow with minimal pulsatility (arrow).

Figure 3. Chart illustrates the classification of postsurgical complications of liver transplantation.
Hepatic Artery Complications

There are several anastomotic possibilities involving the hepatic artery. In orthotopic liver transplantation, the donor celiac axis is anastomosed to the recipient hepatic artery at either the bifurcation into left and right hepatic arteries or the takeoff of the gastroduodenal artery. In patients with a small or diseased hepatic artery, a donor iliac artery interposition graft may be anastomosed directly to the recipient aorta (6). Knowledge of the type of anastomosis is important because stenosis frequently occurs at this site. Hepatic artery complications include thrombosis, stenosis, and pseudoaneurysm. Hepatic artery thrombosis and stenosis can lead to biliary ischemia, since the hepatic artery is the only source of vascular supply to the bile ducts (7). Biliary ischemia may in turn lead to a nonanastomotic biliary stricture or a biloma, which are often associated with hepatic artery complications.

Thrombosis

Hepatic artery thrombosis is the single most common vascular complication of orthotopic liver transplantation, occurring in 2%–12% of cases (8–10), and has been reported to occur between 15 and 132 days following transplantation (6). Risk factors include allograft rejection, end-to-end anastomosis, short warm ischemia time, and pediatric transplantation (11). Postoperative Doppler US has a high sensitivity and specificity for the detection of hepatic artery thrombosis, whose presence may be indicated by nonvisualization of the hepatic artery; however, the inability to depict flow in a patent hepatic artery remains a substantial problem despite improvements in Doppler technology. Reduced flow, whether secondary to spasm or to low cardiac output, can also cause nonvisualization of flow at Doppler US. Moreover, Doppler US in the immediate postoperative period is limited by technical factors such as surgical dressing material. Microbubble contrast material–enhanced US may help improve flow visualization in the hepatic artery (12). In patients in whom no flow is identified in the hepatic artery, CT angiography or MR angiography is usually required to obtain a definitive answer about thrombosis. MR angiography is equivalent to US in terms of diagnostic accuracy (13), whereas CT angiography has been shown to have a diagnostic accuracy equivalent to (14) or better than (15) that of US. CT may demonstrate low-attenuation foci in the liver parenchyma representing liver infarction (Fig 4). Treatment usually consists of emergent thrombectomy or retransplantation.

Stenosis

Hepatic artery stenosis occurs in 2%–11% of transplantations (16–18). The median time from transplantation to the diagnosis of hepatic artery stenosis is 100 days (19). Risk factors for hepatic artery stenosis include allograft rejection, poor surgical technique, and clamp injury (19). Doppler US findings include a low resistive index (<0.5), a long systolic acceleration time (>0.08 seconds), and a tardus-parvus waveform distal to the stenosis, with increased peak systolic velocity (>200 cm/sec) at the stenosis (1,13,20). However, in the very early postoperative period (<72 hours after transplantation), increased hepatic artery resistance (resistive index >0.8) is commonly seen, although resistance usually returns to normal within a few days (21). Increased hepatic artery resistance is associated with older donor age and a prolonged period of ischemia (21). It is important to remember that Doppler US of a tortuous hepatic artery can yield false high-velocity measurements due to incorrect alignment of the sample volume angle. Three-dimensional reconstruction of CT angiographic data allows reliable identification of hepatic artery stenosis as a focal narrowing (22). MR angiography is a developing technique that is limited by a relatively high false-positive rate (13). Hepatic artery stenosis may be treated with percutaneous angioplasty or surgical intervention (19).
Figure 4. Hepatic artery occlusion in a 49-year-old woman. Axial contrast-enhanced CT scan shows low-attenuation areas in the liver (arrows) representing infarction and necrosis.

Figure 5. Hepatic artery aneurysm in a 64-year-old woman. (a) Noncontrast CT scan shows a large lesion representing a hepatic artery aneurysm and outlined by atherosclerotic calcification (arrowheads). (b) Doppler US image shows a fusiform anechoic structure at the porta hepatis with central flow (arrow) representing an aneurysm.

**Pseudoaneurysm**
Hepatic artery pseudoaneurysm is an uncommon complication and can be classified as either extrahepatic or intrahepatic. Extrahepatic pseudoaneurysm most commonly occurs at the arterial anastomosis or arises as a complication of angioplasty, whereas intrahepatic pseudoaneurysm may result from percutaneous biopsy, biliary procedures, or infection (23,24). Doppler US reveals a cystic structure with a disorganized arterial flow pattern or characteristic bidirectional flow (25). US depiction of a fluid collection near the arterial anastomosis requires further evaluation with pulsed Doppler US to rule out pseudoaneurysm. Contrast-enhanced CT demonstrates a focal lesion with central enhancement that follows arterial blood-pool attenuation. Treatment consists of coil embolization for both types of aneurysms, as well as stent placement or surgical resection for an extrahepatic pseudoaneurysm (24,25). A ruptured intrahepatic pseudoaneurysm can lead to a portal or biliary fistula.

Intrahepatic arterioportal fistula can also occur secondary to liver biopsy. Arterioporal fistula may be seen at up to 50% of posttransplantation biopsies performed the first week, decreasing to 10% of biopsies performed in subsequent weeks (22). CT findings include early arterial phase enhancement of peripheral portal veins and of the corresponding wedge-shaped region of liver parenchyma that is supplied. Hepatic artery aneurysm is another complication of liver transplantation (Fig 5).

**Portal Vein Complications**
In orthotopic liver transplantation, portal vein anastomosis is most often end to end between the donor and recipient portal veins. However, in patients with extensive thrombosis involving the portal and superior mesenteric veins, a venous jump graft may be inserted between the donor portal vein and the recipient superior mesenteric vein with use of a free segment of the donor iliac vein.
Figure 6. Portal vein stenosis and occlusion in a 6-year-old boy. Contrast-enhanced CT scan shows long-segment stenosis of the portal vein (arrows).

Figure 7. Acute portal vein thrombosis in a 54-year-old man. (a) Color Doppler US image shows lack of flow in the main portal vein and an echogenic clot (arrowheads). (b) Gadolinium-enhanced MR venogram shows a hypointense thrombus in the main portal vein (arrow) cranial to the confluence of the superior mesenteric vein and the splenic vein.

Figure 8. IVC thrombosis in a 45-year-old woman. MR venogram obtained after liver transplantation shows a clot in the infrarenal IVC (arrow).

(5). Alternatively, these patients may undergo arterialization of the portal vein, performed by anastomosing the donor portal vein to the recipient hepatic artery (22). Knowledge of the location of the anastomosis is important because stenosis may occur at this site. Portal vein complications are less common and occur in 1%–13% of transplantsations (5). Causes include faulty surgical technique, discrepancy between the calibers of the donor and recipient portal veins, hypercoagulable state, and history of prior thrombus (5).

Stenosis
Portal vein stenosis usually occurs at the anastomosis (22). Long-segment stenosis of the portal vein may also be seen (Fig 6). US findings include peak anastomotic velocity greater than 125 cm/sec or an anastomotic-to-preanastomotic velocity ratio of 3:1 (26). An apparent anastomotic narrowing may in fact simply represent a size discrepancy between the recipient and donor portal veins. Under these circumstances, transhepatic direct portography can be used to evaluate for a pressure gradient greater than 5 mm, a finding that indicates significant stenosis (23).
The anastomotic narrowing can also be depicted with CT angiography and MR angiography (4). Treatment options include angioplasty, stent placement, and resection.

**Thrombosis**

Portal vein thrombosis occurs in 3% of liver transplantations (3) and often involves the main extrahepatic segment. Doppler US demonstrates no blood flow, whereas CT and MR imaging demonstrate a filling defect in the portal vein (Fig 7). Conventional angiography with splenic, mesenteric, transjugular, or transhepatic portography approaches will also demonstrate a filling defect. Treatment options include angioplasty, stent placement, surgical thrombectomy, thrombolysis, and resection.

**IVC and Hepatic Vein Complications**

There are several anastomotic options for the IVC in orthotopic liver transplantation. Anastomosis of the donor and recipient IVCs can be end to end, with resection of the recipient retrohepatic IVC and anastomosis of the donor IVC superiorly and inferiorly to the recipient IVC. Another technique, known as the “piggyback” technique, involves anastomosis of the donor IVC to the stump of the recipient hepatic vein without resection of the retrohepatic IVC.

With living donor liver transplantation, the donor hepatic vein is anastomosed to the recipient IVC. It is helpful to know the type of anastomosis because stenosis may occur at the anastomotic site. Complications include IVC stenosis and thrombosis, as well as hepatic vein stenosis and thrombosis, which altogether occur in only 1%-2% of transplantations (1).

**IVC Stenosis and Thrombosis**

IVC stenosis is caused by anastomotic narrowing (25) and extrinsic compression secondary to graft swelling (22), fluid, or hematoma. US demonstrates a three- to fourfold increase in velocity compared with the unaffected IVC, and associated color Doppler aliasing. Indirect findings include distention of the hepatic veins with dampening and loss of phasicity of the hepatic venous Doppler waveform. CT venography and MR venography demonstrate focal narrowing of the IVC, and there may be imaging features of Budd-Chiari syndrome or portal hypertension. Angioplasty and stent placement have reportedly been used in the treatment of IVC stenosis (5).

IVC thrombosis is caused by surgical factors and a hypercoagulable state (22). In addition to the US findings discussed earlier, intraluminal thrombus is seen. Contrast-enhanced CT and MR imaging demonstrate an intraluminal filling defect (Fig 8). Coronal imaging is useful for determining the extent of IVC thrombus (4).

**Hepatic Vein Stenosis and Thrombosis**

Hepatic vein stenosis occurs mostly in living donor liver transplantation. US findings of a venous pulsatility index of less than 0.45 and monophasic waveforms in the hepatic veins are indicative of hepatic vein stenosis (26,27). Treatment options include balloon angioplasty of the stenosis (Fig 9).
Hepatic vein thrombosis manifests as an intraluminal filling defect and a lack of blood flow.

**Biliary Complications**

A biliary anastomosis is usually between the donor CBD and the recipient common hepatic duct, and a T tube is left in place for 6 weeks postoperatively. In patients with sclerosing cholangitis, the donor CBD is anastomosed directly to the recipient jejunum. Again, knowledge of the anastomotic site is important because stenosis may occur at this site. If there is a T tube in situ, T-tube cholangiography is preferable to MR cholangiopancreatography because the distention of the bile ducts with contrast material permits better stricture analysis and functional assessment. If there is no T tube in situ, MR cholangiopancreatography is the optimal noninvasive imaging study, since it permits evaluation of the biliary tree without the complications associated with invasive percutaneous transhepatic cholangiography or endoscopic retrograde cholangiography. US has a lower sensitivity (54%) for detecting biliary complications (28).

Biliary complications occur in 5%–15% of hepatic transplantations (3) and are usually seen in the early postoperative period (<3 months after surgery). These complications include bile duct obstruction, anastomotic stenosis, bile duct stricture, stone formation, bile leak, biloma, biliary necrosis (Fig 10), and cholangitis. In a large study of 1792 orthotopic liver transplant recipients, biliary stricture occurred in 5% of cases, bile leaks in 3%, ampullary dysfunction in 2%, and biliary obstruction in 1.6% (29). Biliary complications are more common with right-lobe liver donor transplantation, which is more technically challenging (3). Complications following choledochocholedochal anastomosis are often managed with ERCP, whereas complications following Roux-en-Y choledochojejunostomy often require surgical correction (30).

**Biliary Obstruction**

Biliary obstruction is frequently secondary to anastomotic stricture, but it may also be secondary to choledocholithiasis. There is postobstructive dilatation of the donor CBD (31). It is important to recognize that some patients demonstrate nonobstructive dilatation of the extrahepatic donor and recipient ducts without intrahepatic biliary dilatation. Nonobstructive biliary dilatation may be secondary to papillary dyskinesia or may be clinically insignificant. Dilatation of the CBD may also be attributed to a discrepancy between the calibers of the donor
and recipient ducts. MR cholangiopancreatography is useful in the evaluation of biliary obstruction (31). US is less reliable, particularly in the detection of mild intrahepatic biliary obstruction and choledocholithiasis (28).

**Choledocholithiasis**
Biliary filling defects including stones, sludge, and necrotic debris occur in 5.7% of hepatic transplants (32). Alteration in bile composition in transplant recipients may be a predisposing factor in biliary stone and sludge formation (30). The well-defined margin of stones at MR cholangiopancreatography helps distinguish them from the castlike appearance of sludge or debris. Pneumobilia is a mimic of stones at MR cholangiopancreatography, but the nondependent location of the air bubbles usually helps distinguish them from stones. US is not reliable in the detection of choledocholithiasis (28), which is usually treated with endoscopic sphincterotomy and stone removal (30).

**Bile Duct Stricture**
Most bile duct strictures are extrahepatic and occur at the anastomosis secondary to scar formation (Fig 11). Nonanastomotic biliary stricture occurs secondary to ischemia (often as a result of hepatic artery thrombosis or stenosis), infectious cholangitis (Fig 12), or pretransplantation sclerosing cholangitis (31). Sometimes a biliary stricture is suspected even if CT demonstrates no biliary dilatation. In such cases, MR cholangiopancreatography, ERCP, or transhepatic cholangiography should be performed to identify the stricture, since liver transplants may not develop biliary dilatation despite severe stenosis. Extrahepatic anastomotic biliary strictures are initially treated with angioplasty (with or without stent placement), whereas intrahepatic ischemic biliary strictures are treated with percutaneous transhepatic biliary drainage (30).

**Bile Duct Leakage**
Bile duct leaks occur with a prevalence of 4.3% after hepatic transplantation, most commonly at the biliary anastomosis or the T-tube exit site (31). At direct cholangiography, bile leaks appear as extravasation of contrast material from the T-tube site into the peritoneal cavity, or as single or multiple bilomas (33). Bilomas are contained bile leaks that may be caused by anastomotic

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**Figure 11.** Biliary stricture. (a) Thick-slab MR cholangiopancreatogram obtained in a 12-year-old boy shows a nonanastomotic stricture (arrow) at the confluence of the right and left hepatic ducts. (b) ERCP image obtained in a 45-year-old woman shows an anastomotic stricture at the choledochocholedochal anastomosis (arrow).

**Figure 12.** Biliary complications of cholangitis and biliary stenosis in a 51-year-old man. ERCP image shows a nonanastomotic intrahepatic biliary stricture in the right hepatic duct (arrow). Note also the irregularity of the left lobe biliary ducts from cholangitis.
Figure 13. Intrahepatic biloma in a 53-year-old man. Axial noncontrast CT scan shows a rounded fluid collection in segment VI of the liver (arrow). Aspiration and laboratory analysis helped confirm the diagnosis of an intrahepatic biloma.

Figure 14. (a) Cholescintigrams obtained in a healthy 58-year-old man show normal flow of radiotracer through the CBD (arrow) into the small bowel. (b) Postoperative bile leak in a 55-year-old man. Cholescintigram obtained 1 hour after radiotracer injection shows bile leakage into the peritoneal cavity in the right subphrenic space, right paracolic region, and pelvis (arrows).

dehiscence secondary to ischemia. They are seen as discrete, rounded, hypoechoic (US) or hypodense (CT) fluid collections (Fig 13). Cholangiography is the most precise modality for the detection of bile leak; however, cholescintigraphy (hepatobiliary nuclear imaging) is a sensitive and specific noninvasive test for biliary leakage (34). US may not be able to help differentiate bile leaks from nonbiliary postoperative fluid collections such as ascites, abscess, and hematoma (34). The progressive accumulation in the abdomen of radiotracer that does not conform to the morphologic characteristics of bowel indicates a bile leak (Fig 14). However, a localized bile leak (biloma) can be difficult to distinguish from bowel. Another diagnostic pitfall can occur in patients who undergo hepaticojejunostomy with a Roux-en-Y limb, in whom normal radiotracer accumulation in the blind end of the limb can be mistaken for a bile leak (35). US can help detect a localized biloma; however, a more dispersed bile leak is usually not clearly depicted. Bile duct leaks are treated with stent placement across the leakage site, whereas bilomas are treated with percutaneous drainage (30).

Other Complications
Other complications of liver transplantation include hematoma, abscess, infection, recurrent hepatitis, splenic infarct, recurrent malignancy, and posttransplantation lymphoproliferative disorder (2,4,36).

Hematoma usually manifests within 2 weeks after transplantation and occurs near the vascular anastomosis, as well as in the perihepatic space and lesser sac (22). The hematoma is echogenic
at US, hyperattenuating at CT (Fig 15), and hypointense at T2-weighted MR imaging. Most hematomas will resolve spontaneously within a few weeks, but in some cases superimposed infection may require catheter drainage or aspiration.

Intrahepatic abscess is often secondary to liver infarction (4). Predisposing factors include biliary stricture, arterial insufficiency, and immunosuppressive medications. The presence of a complex fluid collection with a possible air-fluid level at US and CT suggests an abscess (Fig 16). Treatment consists of catheter drainage (2). Liver transplant recipients are often immunocompromised and susceptible to lung infection (2).

Another complication is tumor recurrence in the transplant following liver transplantation for neoplasm such as hepatocellular carcinoma or hepatic metastases from a neuroendocrine tumor (22). Liver transplant recipients are also at increased risk for non-Hodgkin lymphoma, Kaposi sarcoma, and squamous cell skin cancer due to their immunosuppressed state (22). A further complication seen in patients with hepatitis C or hepatitis B viral infection is persistent viremia in the absence of effective prophylaxis. In these patients, reinfection of the liver with resultant liver cirrhosis is likely to occur (22). Recurrent liver cirrhosis is often accompanied by sequelae of portal hypertension, portal colopathy, secondary neoplasm, and variceal hemorrhage. Splenic infarction may occur but is not clinically significant if there is no infection. Intestinal perforation may occur due to (a) deserosalization of the bowel during a difficult hepatectomy, or (b) an intestinal leak at the jejunostomy site (2).

**Overview of Percutaneous Interventional Procedures**

Biliary interventional procedures include angioplasty (with or without stent placement) across extrahepatic strictures, transhepatic biliary drain placement across intrahepatic strictures, stent placement across bile leakage sites, catheter drainage of bilomas, and removal of occluded biliary stents. Vascular interventional procedures are less common and include angioplasty for hepatic artery stenosis, coil embolization of or stent placement for pseudoaneurysms, and angioplasty or

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**Figure 15.** Hematoma in a 52-year-old man. Noncontrast CT scan obtained after liver transplantation shows a hyperattenuating fluid collection in the Morison pouch (arrow), a finding that represents a hematoma.

**Figure 16.** Intrahepatic abscess in a 64-year-old woman. (a) US image shows a complex intrahepatic fluid collection in the right lobe (arrows). (b) Axial noncontrast CT scan shows an intrahepatic abscess (arrow) containing an air-fluid level. An internal drainage catheter is also seen.
stent placement for all venous stenoses. Catheter drainage is effective for diagnostic and therapeutic purposes in fluid collections such as abscesses.

Conclusions
Multiple complications can be observed after liver transplantation. US is the main initial imaging modality for the evaluation of liver transplant dysfunction due to its easy availability and high sensitivity in the detection of vascular as well as biliary complications. CT is complementary to US and is often used as a problem-solving modality, being reserved for second-line investigation when US findings are indeterminate or inconclusive. Cholescintigraphy remains the most sensitive noninvasive modality for the detection of bile leak. MR cholangiopancreatography is very useful in the evaluation of bile duct dilatation and obstruction. Imaging is useful for the detection of early and late complications, as well as for long-term follow-up to assess transplant viability. An understanding of potential posttransplantation complications and of the strengths and weaknesses of each imaging modality will aid in early diagnosis and promote timely therapy.

References


Hepatic artery thrombosis and stenosis can lead to biliary ischemia, since the hepatic artery is the only source of vascular supply to the bile ducts.

US depiction of a fluid collection near the arterial anastomosis requires further evaluation with pulsed Doppler US to rule out pseudoaneurysm.

Another technique, known as the “piggy-back” technique, involves anastomosis of the donor IVC to the stump of the recipient hepatic vein without resection of the retrohepatic IVC.

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