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Biliary complications after liver transplantations: A ²² computed tomography and magnetic resonance imaging pictorial review

Vernuccio ³⁵ F *et al.* Imaging of biliary complications after liver transplantation

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INTRODUCTION

²⁹ Computed tomography (CT) and magnetic resonance imaging (MRI) are the cornerstones for the postoperative assessment of patients after liver transplantation^[1]. Complications may be distinguished by cause in surgical, graft-related, immunologic, infectious, and neoplastic and by the time of occurrence in early if they occur within six months from surgery and late if they occur after six months (Table 1). Surgical complications are mainly distinguished in vascular, ⁴⁴ biliary, and parenchymal. Biliary complications are the ⁴ most common ones, being a major source of morbidity and mortality in liver transplantation recipients, with an incidence of 10%-32%^[2-4]. Biliary complications after liver transplantation ¹² include anastomotic strictures, non-anastomotic strictures, bile leaks, bile casts, bilomas, sphincter of Oddi dysfunction, and ⁹ mucocele of the cystic duct remnant^[4-9]. Biliary complications have a significant negative impact on patient survival and may lead to the need for re-transplantation^[3,6]. ⁸ Magnetic resonance cholangiopancreatography (MRCP) is nowadays ⁶ the gold standard for the diagnosis of intra- and extrahepatic biliary complications, while invasive cholangiography should be restricted for therapeutic uses or when MRCP is equivocal^[10].

¹⁹ The aim of this pictorial review is to illustrate CT and MRI findings of biliary complications after liver transplantation related to surgery based on their usual timing of appearance and frequency. The knowledge of surgical techniques is of key importance to understand postoperative anatomic changes and imaging evaluation. Therefore, we will first provide a short summary of the main techniques of liver transplantation with a focus

on biliary anastomosis. Then, we will discuss imaging tips and tricks for the prompt diagnosis of biliary complications on CT and MRI.

SURGICAL TECHNIQUE

Most liver transplantations are performed with orthotopic implantation of deceased donor whole liver graft that may be performed with a conventional technique or with piggyback technique; other options include split or segmental liver transplantation (Figure 1)^[7]. The split liver procedure may be performed with two approaches: in the first and most common one, the liver is divided into a left lateral segment graft (II e III ± IV segments) to be transplanted to a child and a right extended liver lobe graft (I + V-VIII ± IV segments) for an adult recipient; in a technically more challenging variant of this procedure, the liver is split into two hemigrafts and the left side (I-IV) is used for a small adult or a teenager and the right side (V-VIII) for a medium-sized adult patient. In patients with prior biliary disease or re-transplantation, different than usual biliary anastomosis may be performed^[7]. Liver transplantation is a multi-step surgery. After skin preparation and incision, the surgeon checks if there is any undiagnosed malignancy or anatomic variant and, then, dissects the recipient's liver and gallbladder. The donor's liver, without the gallbladder, is then implanted in the recipient with the anastomoses between recipient and donor being performed in the following order: Systemic venous outflow (inferior cava vein-hepatic veins), portal venous inflow, hepatic arterial inflow, and, finally, biliary anastomosis. The types of anastomosis will depend on the donor's and recipient's anatomy and surgeon's preference. Finally, when the surgical field is dry, the abdomen will be closed. Each of the above-mentioned steps is critical and complications may be directly or indirectly related to failure of one or more of these steps^[11,12].

Biliary anastomosis is known as the "Achilles tendon" of liver transplantation. The most common form of biliary reconstruction is choledocho-choledochostomy (duct-to-duct anastomosis)^[7], which may be performed in an end-to-end or end-to-side fashion. Choledocho-choledochostomy can be performed either with a T-tube, which allows rapid

decompression of the biliary tree if needed and reduces the risk of anastomotic stricture formation but may lead to biliary leakage and cholangitis at the time of removal, or without a T-tube^[13]. Choledocho-jejunostomy to a Roux-en-Y defunctionalized intestinal loop (i.e., the connection of the bile duct to jejunum loop) (Figure 1F) is the second most common type of biliary reconstruction techniques, usually preferred in patients with pre-existing biliary disease, in case of size mismatch between donor and recipient ducts, retransplantation or previous biliary surgery^[7]. Potential complications of choledochojejunostomy include stricture, leakage, and bleeding at the jejuno-jejunostomy site. Choledocho-choledochostomy is preferred over choledocho-jejunostomy due to shorter operation time, lower septic complications, preserved sphincter of Oddi, better physiologic enteric function, and easier endoscopic access to the biliary tree for future needs.

CT/MRI OF BILIARY COMPLICATIONS

MRI has sensitivity and specificity of 98%-99% and 94%-96%, respectively, for the diagnosis of biliary complications after liver transplantation^[14,15]. MRI protocol includes 2D-MRCP and 3D-MRCP and an unenhanced T1-weighted sequence, while gadoxetate disodium hepatobiliary MRI is performed in selected cases^[16]. Ultrasound is usually performed as the first imaging technique and may help identify some features that suggest the presence of complications. Despite not being as comprehensive as MRI, CT may be helpful for the assessment of biliary complications and it is often used due to its higher availability as compared to MRI. Table 2 summarizes biliary complications related to surgery with frequency, time of occurrence, and treatment^[4-10].

Biliary strictures

Biliary strictures are distinguished in anastomotic and non-anastomotic. Anastomotic strictures (Figure 2) account for about 47% of biliary complications, being slightly more frequent after choledocho-choledochostomy rather than choledochojejunostomy, and may occur also after split liver donation^[3]. Nowadays, percutaneous biliary techniques

are considered effective treatment options with good outcome in liver transplant with anastomotic biliary strictures^[17]. Non-anastomotic strictures account for about 23% of all biliary complications, being slightly more frequent after choledochojejunostomy rather than choledocho-choledochostomy^[3]. Non-anastomotic strictures (Figure 3) typical include ischemic-type biliary lesions in the early period after transplant while they are mostly related to recurrence of the primary biliary disease, chronic rejection, or secondary sclerosing cholangitis in the late period.

Biliary strictures are one of the most critical complications in ABO-incompatible living donor liver transplantation recipients and may occur as perihilar or diffuse, with the latter having worse clinical outcome^[18].

MRI will demonstrate the stenosis at the level of the stricture and the upstream irregular dilation of the biliary system; typically, the change in duct caliber at the level of the stricture is abrupt. Anastomotic strictures tend to be single and short in length and occur at the level of anastomosis, usually in the late period after transplantation. Non-anastomotic strictures are frequently multiple, long and hilar in location, tend to occur early after transplantation and may result in graft loss. Radiologists should write in their report the level of the biliary injury and the length of the obstruction. Although not routinely recommended for the diagnosis of biliary strictures, MRCP with hepatobiliary contrast agent may allow the assessment of the severity of bile duct obstruction based on the degree of hepatobiliary contrast agent filling distal to the stricture. Complete obstruction of the biliary tree is demonstrated in case of absence of contrast agent distal to the stricture, while the obstruction is partial if there is limited passage of contrast agent beyond the stricture. In case of complete obstruction, hepatic function may be impaired with elevated bilirubin level, which may hamper the excretion of hepatobiliary contrast agents^[19]. Biliary strictures must be differentiated on MRI from their mimickers including size mismatch of the donor and recipient common ducts, which appears on MRI as gradual tapering of the bile duct lumen at the anastomosis, and from postoperative oedema, which may cause extrinsic compression at the level of the anastomosis with tapered “hour-glass” appearance. Other potential mimickers of biliary strictures on MRI

include pneumobilia, which may occur normally if a choledochojejunostomy anastomosis has been performed, and MRI susceptibility artifacts caused by nearby surgical clips. CT may help identifying the inadvertent placement of metallic surgical clips. In ABO-incompatible living donor liver transplantation recipients, imaging and clinical follow-up is recommended if post-transplantation CT at one month after surgery demonstrates subtle intrahepatic duct dilatation with perihilar abnormality for the possible occurrence of diffuse intrahepatic duct dilatation strictures in the future^[18].

Biliary leak and biloma

Biliary leaks account for approximately 23% of all biliary complications^[3]. They may be anastomotic or non-anastomotic (Figure 4) and are more common after choledochocholedochostomy rather than choledochojejunostomy. Leaks at the biliary anastomosis are the most common ones^[20]. Non-anastomotic leaks may occur at the level of T-tube insertion, cystic duct or the cut surface of partial liver grafts. The use of a T-tube seems to be a risk factor for biliary leaks, most commonly after its removal^[21]. However, there are still discordant data about the causative mechanism of T-tube on the occurrence of biliary leaks^[22,23]. Non-anastomotic biliary leaks may be cut-surface leaks, such as those originating from small bile ducts that are transected perioperatively during hepatic resection or from the cystic duct stump, or may be caused by bile duct necrosis in patients with hepatic artery occlusion. Biliary leaks may result in the development of bilomas. Bilomas may be intra- or extra- hepatic depending on the source of origin of the bile leakage, although they most commonly occur in the perihepatic space. Bilomas may superinfect and potentially lead to sepsis; another potential serious complication of bilomas is erosion of the adjacent hepatic artery. Ultrasound and CT are most commonly performed as first-line techniques due to their wide availability. On ultrasound and CT, biliary leaks and biloma are demonstrated as free fluid or fluid collection usually in the perihepatic and subhepatic space, mostly anechoic on US and hypoattenuating with fluid density on CT. On MRI, biliary leaks and biloma will be hypointense on T1-weighted sequence and hyperintense on T2-weighted sequence, with the former appearing as free

fluid and the latter as a fluid collection (Figure 5). However, these findings are non-specific and biliary leaks and bilomas are virtually indistinguishable from other types of fluid collection and ascites. In case of biliary leak occurring after bile duct necrosis in patients with hepatic artery occlusion, there may be the development of intrahepatic bilomas or bile lakes with a characteristic imaging appearance as cystic or linear dilatations of the intrahepatic bile ducts (Figure 6) on CT and MRI in the early period after transplantation. MRCP with hepatobiliary contrast has 100% sensitivity and 98% specificity in the diagnosis of bile leaks^[24,25]. MRCP with hepatobiliary contrast allows to demonstrate the level and the entity of biliary leakage showing contrast agent extravasation into bilomas in case of active leakage. However, small bilomas are oftentimes self-limiting and active extravasation may not be demonstrated. The demonstration of the lack of active bile leak into the bilomas is highly relevant from a clinical standpoint, as it may help in choosing a conservative management. However, it is important to highlight that the diagnostic accuracy of MRCP with hepatobiliary contrast depends on the timing of acquisition of the hepatobiliary phase. When conventional acquisition at 20 min only is adopted, sensitivity may be as low as 42.9%^[26], while the acquisition at 60-90 min, 150-180 min, or even 210-240 min to 390 min increases the sensitivity^[26,27]. The reason behind the lower sensitivity of the 20-min hepatobiliary phase compared to acquisitions at later times may be two-fold. On one hand the increased bilirubin in these patients may result in low uptake of hepatobiliary contrast by the hepatocytes at 20 min; indeed, bilirubin is taken up at the hepatocyte level by the same family of organic anion transport proteins of gadoxetate disodium (Figure 7). On the other hand, bile duct obstruction may result in the up-regulation of a multidrug resistance protein, which could reduce the excretion of gadoxetate disodium in the blood mainstream, delaying or preventing the visualization of bile ducts and bile leakage^[28,29]. For this reason, based on consensus reports for liver MRI, an elevated bilirubin level is considered a relative contraindication to injection of gadoxetate disodium at some centers, with threshold bilirubin levels from 2.0-5.0 mg/dL; delayed acquisitions prove to be helpful for the diagnosis of biliary leaks^[29,30].

Biliary casts, stones and sludge

Biliary casts, stones and sludge account for about 6% of all biliary complications^[3], and usually complicate biliary strictures. These concretions may occur at both the intrahepatic and extrahepatic bile ducts as the consequence of bile stasis and may lead to cholangitis, graft failure or need for re-transplantation^[31]. Biliary concretions after liver transplantation are related to an inhomogeneous group of lithogenic conditions mostly due to bile tract damage in the presence of a multifactorial, complex physiopathology environment^[32]. In regard to biliary casts, they complicate about 4.5% of liver transplantations, may recur and may lead to biliary strictures in 85.0% of patients on follow-up^[4]. Morphologically, biliary casts after liver transplantation may have a cordlike, columnar, dendritic shape within the biliary tree^[33]. The prompt identification of biliary casts is of utmost importance as patients with biliary casts syndrome have lower overall and graft survival compared with non-anastomotic and anastomotic strictures^[4]. MRCP has very good sensitivity in the identification of biliary concretions as filling defects surrounded by a thin film of hyperintense bile (Figure 8). Importantly, sensitivity for biliary casts detection increases when using T1-weighted images compared to T2-weighted MRCP; unenhanced T1-weighted images show hyperintensities in the bile ducts (Figure 8C), leading to the correct diagnosis of biliary cast^[34]. As recently pointed out, intraductal hyperintense filling material on T1-weighted MRI is a sensitive sign for biliary casts and intraductal filling defect on T2-weighted MRI with the duct-in-a-duct feature is a specific sign, which probably reflects biliary mucosal detachment^[4].

Sphincter of Oddi dysfunction

Sphincter of Oddi dysfunction (SOD) comprises functional or mechanical obstruction of the sphincter of Oddi and involves the biliary sphincter and/or the pancreatic sphincter. Biliary and pancreatic SOD have been each sub-classified for years into three types on the basis of symptoms, laboratory tests and imaging (common bile duct diameter of at least 12 mm): Type I with biliary pain, abnormal liver enzymes and dilatation of the common

bile duct; type II with biliary pain and either abnormal liver enzymes or dilatation of the common bile duct; and type III with biliary pain and no objective criteria^[35,36]. More recently the Rome IV consensus has proposed a new classification, as most type I patients present a papillary stenosis rather than a functional disorder and have an excellent response after sphincterotomy, type II has now been renamed as suspected functional biliary sphincter disorder, and type III patients have no response to sphincterotomy^[37,38]. SOD after liver transplantation has been reported in about 2%-5% of patients, with papillary stenosis (*i.e.*, SOD type I) accounting for about 1% of cases and suspected functional biliary sphincter disorder (*i.e.*, SOD type II) for about 1% as well^[39]. The pathogenesis of biliary sphincter disorder in liver transplantation recipients is poorly understood; some predisposing factors may be the use of a T-tube, the presence of opportunistic infections, and post-surgical edema^[40,41]. Patients with functional biliary sphincter disorder after liver transplantation may be asymptomatic due to hepatic denervation after the surgery and immunosuppression, thus making the diagnosis more difficult^[41]. Therefore, suspicion of a SOD after liver transplantation should be raised when cholestasis and/or dilation of bile ducts appear in the absence of bile stones or other structural abnormalities. Sphincter of Oddi manometry has been the gold standard for years, although it is invasive, patient- and operator- dependent, and may lead to post-procedure pancreatitis; therefore, it is no longer routinely used in all patients with suspected SOD and its utility has been questioned^[39-41]. Hepatobiliary scintigraphy can demonstrate structural or functional partial biliary obstruction showing increased time to hepatic peak, delayed biliary visualization, delayed clearance of the radiotracer from the dilated bile ducts, and prolonged biliary to bowel transit^[42-44]. MRCP may be used to exclude biliary lithiasis and other structural abnormalities, and may show an enlarged papilla in some cause of papillary stenosis (Figure 9). Secretin-MRCP may suggest the diagnosis of SOD, showing stenosis of the sphincter and lack of relaxation of the main pancreatic duct after secretin injection, increased prominence of pancreatic duct side branches and/or acinarization^[45]. Secretin-MRCP seems more useful for SOD type II with a diagnostic accuracy of 73%, rather than for SOD type III for which accuracy drops to

only 46%^[46]. Given the low accuracy, the cost of secretin, and the acquisition time of at least 15 minutes, secretin-MRCP for SOD should be considered only in few selected cases (e.g., noninvasive evaluation is preferred or when endoscopic evaluation is not available or impractical)^[45]. Gadoxetate disodium-enhanced MRCP may help in ruling out SOD in case of normal passage of hepatobiliary contrast in the duodenum at 20-30 min and in suggesting the diagnosis in case of delayed or no passage of bile through the ampulla of Vater after 30 min-1 h^[47]. Interestingly, the diagnostic accuracy of gadoxetate disodium-enhanced MRCP for SOD has not been investigated yet, and represents an area of interest particularly when invasive evaluation is not indicated.

¹⁷ *Mucocele of the cystic duct remnant*

⁴⁸
The mucocele of the cystic duct remnant, whether recipient or donor in origin, ⁴⁵is an extremely rare complication after liver transplantation^[48-51]. It is characterized by an abnormally ³¹dilated cystic duct remnant with flattening of the walls of the residual cystic duct to form a collection of mucous from cells lining the cystic duct remnant. ²⁵The causative mechanism of the mucocele is still unclear. Lack of nervous regulation of the biliary tract after a liver transplant may probably affect bile secretion and outflow. The differential diagnosis include abscess, biloma, hemobilia, tumor, or aneurysm. If left untreated, the enlarged mucocele may cause chronic mechanical compression of the biliary system; however, it may also remain stable in size^[50]. Ultrasound and CT can demonstrate the presence of a collection at the level of the hepatic hilum. MRCP will demonstrate a rounded and well-delimited collection adjacent to ⁴⁷the common hepatic duct, and lack of any other cause of obstruction (Figure 10).

Bile duct redundancy

¹⁴
Bile duct redundancy is described as a surgically reconstructed donor-recipient extrahepatic bile duct that creates a looped, sigmoid-shaped appearance in the absence of any anastomotic stricture^[8]. A redundant bile duct occurs when the donor or the recipient bile duct is too long and may lead ¹⁴to delayed bile flow into the duodenum,

functionally translating into cholestasis, abnormal liver laboratory tests and cholangitis, or may predispose to kinking of the redundant bile duct and, therefore, obstruction^[8,52,53]. 2D and 3D MRCP images may demonstrate the abnormal long-constructed donor-recipient extrahepatic bile duct, its shape as well as its kinking, if present.

²³ *Vanishing bile duct syndrome*

The vanishing bile duct syndrome is a very rare biliary complication occurring after liver transplantation, characterized by progressive destruction and disappearance of the intrahepatic bile ducts in the portal area leading to cholestasis^[54,55]. It is caused by an acute or chronic T-cell-mediated rejection of the allograft^[54,55]. The diagnosis of vanishing bile duct syndrome is suspected in a patient with liver biochemical abnormalities consistent with cholestasis in the absence of other conditions associated with cholestasis^[54-56]. Histologic examination through liver biopsy is needed for the diagnosis and MRI may help in excluding other causes of cholestasis^[54,56].

CONCLUSION

In conclusion, biliary complications represent a clinically relevant problem after liver transplantation occurring in up to one third of liver transplantation recipients. Radiologists need to be aware of surgical techniques and post-surgical anatomy as well as clinical information for comprehensive image interpretation. MRCP is an established non-invasive procedure for the diagnosis of post-transplantation biliary complications. In selected cases, gadoxetate disodium enhanced MRCP is needed for improving diagnostic accuracy of biliary complications and its protocol must be tailored based on the clinical suspicion.

Figure 1 Schematic representation of surgical techniques for liver transplantation. A: Normal anatomy of the liver; B: Conventional technique for liver transplantation; C: Piggyback technique; D: Split liver technique in adults; E: Living donor right lobe liver

transplantation; F: Conventional (top row) and piggyback (bottom row) techniques with choledochojejunostomy.

Figure 2 Anastomotic biliary strictures. A: Anastomotic stricture of choledocho-choledochostomy after 3 mo from liver transplantation. Endoscopic retrograde cholangiopancreatography was performed with balloon dilatation of the stricture and stent positioning. Magnetic resonance cholangiopancreatography (MRCP) maximum intensity projection (MIP) image demonstrates anastomotic biliary stricture (arrowhead) with marked upstream biliary dilatation; B: Contrast enhanced computed tomography in the coronal plane shows in the same patient the stent in the biliary tree (arrow) and normal biliary tree caliber; C: Anastomotic stricture of choledocho-jejunostomy after 6 mo from liver transplantation. MRCP MIP image demonstrates anastomotic biliary stricture (arrowhead) with marked upstream biliary dilatation; D: Anastomotic stricture of end-to-end biliary anastomosis in after split liver transplantation with right split lobe. MRCP MIP image demonstrates anastomotic biliary stricture (arrowhead) with marked upstream biliary dilatation of the right split transplanted lobe.

Figure 3 Non-anastomotic biliary strictures. A: Non-anastomotic strictures in a patient with chronic rejection demonstrated at biopsy eleven years after transplant and then retransplanted. Magnetic resonance cholangiopancreatography (MRCP) maximum intensity projection (MIP) image demonstrates multiple non-anastomotic biliary strictures (arrowheads); B: Non-anastomotic strictures in a patient with recurrent secondary cholangitis four years after liver transplantation. MRCP MIP image demonstrates multiple non-anastomotic biliary strictures (arrowheads) with upstream biliary dilatation.

Figure 4 Schematic representation of biliary leaks. A: Anastomotic leak at the level of choledochocholedochostomy; B: Anastomotic leak at the level of choledochojejunostomy; C: Non anastomotic leak at the level of the cystic duct stump; D: Non anastomotic leak at

the level of T-tube removal; E: Non anastomotic leak from small bile ducts that are transected perioperatively during hepatic resection.

Figure 5 Bilomas and biliary leak after liver transplantation. A: Patient with bilomas occurring after nine months from liver transplantation. T2-weighted axial image shows a collection (arrow) in the right hepatic lobe with internal inhomogeneous signal intensities. Percutaneous drainage of the collection was performed demonstrating superinfected biloma; B: Patient with biliary leak occurring after liver transplantation. T2-weighted coronal magnetic resonance imaging image shows an intrahepatic fluid collection (arrowhead) consistent with bilomas; C: Cholangiographic image in the same patient demonstrated the biliary leak (circle) causing an intrahepatic biloma.

Figure 6 Biliary lakes occurring after liver transplantation. A: Contrast-enhanced computed tomography (CT) images in the axial plane in the portal venous phase demonstrates biliary lakes (arrowheads) adjacent to the portal vein branches; B: Contrast-enhanced CT image in the arterial phase demonstrates lack of enhancement of the hepatic artery (arrow) caused by the adjacent surgical clip.

Figure 7 Lack of excretion of hepatobiliary contrast after liver transplantation in a patient with increased serum bilirubin of 2.2 mg/dL. Gadoxetate disodium enhanced magnetic resonance imaging in the hepatobiliary phase acquired at 20 min is inadequate as demonstrated by hypointensity of the liver parenchyma compared to hepatic vessels and lack of contrast in the biliary tree.

Figure 8 Biliary sludge and biliary cast. A: Patient with biliary sludge and anastomotic stricture three months after liver retransplantation. Magnetic resonance cholangiopancreatography (MRCP) maximum intensity projection image demonstrates anastomotic biliary stricture (arrowhead) with marked upstream biliary dilatation; B: 3D-MRCP image in the coronal plane in the same patient demonstrates biliary sludge (arrow)

in the dilated hepatic duct extending into the left and right ducts. C: Patient with biliary cast after two years from liver transplantation. Unenhanced T1-weighted gradient-recalled image shows hyperintense content (arrowhead) in the left biliary duct, consistent with biliary cast.

Figure 9 Enlarged ampullary papilla occurring years after liver transplantation and causing minimal cholestasis. T2-weighted image in the coronal plane demonstrates an enlarged ampullary papilla (arrowhead) protruding in the duodenal lumen (arrow). Ultrasonography-endoscopy confirmed the enlarged ampullary papilla and biopsy was performed which excluded malignancy and confirmed the diagnosis of papillary stenosis (*i.e.*, Sphincter of Oddi dysfunction); sphincterotomy was then performed.

Figure 10 Mucocele occurring years after liver transplantation. Magnetic resonance cholangiopancreatography maximum intensity projection image shows the fluid collection in communication with biliary tree. Biliary content was confirmed with percutaneous drainage.

Table 1 Main post-transplant complication ³⁷ with the relevance of computed tomography and magnetic resonance imaging for their diagnosis

Time of onset	Type of complications	Relevance of CT/MRI for diagnosis
Early (< 6 mo)	Surgical	++++
	Graft-related	++
	Immunologic	+
	Infectious	++
Late (> 6 mo)	Surgical	++++
	Graft-related	++
	Immunologic	+
	Infectious	+++
	Neoplastic	++++
	Disease recurrence	++

++++: Highly relevant, often mandatory; +++: Very useful; ++: Useful; + sometimes helpful, but clinical diagnosis is usually very relevant. ³⁸ CT: Computed tomography; MRI: Magnetic resonance imaging.

Table 2 Post-transplant biliary complications related to surgery based on their frequency, onset and management

Type of complication	Frequency	Timing of onset	Common treatment
Biliary stricture	5%-15% (up to 30 in LDLT)	Early late	Re-fashioning after stenting
Biliary leak	2%-25%	Early	ERCP and stenting if anastomotic
Biloma/Biliary lakes	2.6%-11.5%	Early	Percutaneous drainage and antibiotics if large
Bile duct filling defects	3%-6%	Early late	ERCP/percutaneous drainage
Sphincter of Oddi dysfunction	2%-5%	Late	¹² ERCP with sphincterotomy and consideration of stent placement
Redundant common bile duct	Rare	Late	Stent
Mucocele of bile duct remnant	Rare	Late	Surgery if causing compression

³⁹ LDLT: Living donor liver transplantation; ERCP: Endoscopic retrograde cholangiopancreatography.

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