

Biliary complications

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The biliary complications usually present as bile leak, biliary anastomotic stricture (AS), biliary non-anastomotic stricture (NAS), bile duct sludge/stone/casts, biloma and duct loss (ductopenia) in patients with chronic rejection. Biliary complications following LT can be caused by the vulnerable vascular supply to the biliary tree (supplied by hepatic artery alone), the biliary epithelium being more liable to ischaemic injury than hepatocytes, suboptimal preservation of the peribiliary plexus - Summary box 89.6 - Complications after LT

Early complications (within 6 months) Graft Primary non-function Delayed graft function Surgical Bleeding Hepatic artery thrombosis Portal vein thrombosis Hepatic venous outflow obstruction Bile leak Biliary anastomotic stricture Medical Infections (bacterial, viral, fungal) Rejections Acute kidney injury Late complications (after 6 months) Graft Ischaemic cholangiopathy (non-anastomotic biliary strictures) Surgical Vascular stenosis (hepatic artery, portal vein or hepatic vein) Late HAT Biliary AS Incisional hernia Medical Infections (bacterial, viral, fungal) Rejections/chronic rejection leading to graft failure Renal impairment Disease recurrence Cardiovascular disease Metabolic and bone diseases Malignancy

The incidence of biliary complications is higher in LDLT and other anatomical variant grafts than in whole-graft LT. This is because of the small size of the ducts, multiple duct anastomoses and also cut surface leaks. Due to the morbidity involved with biliary complications, bile duct anastomosis is considered the Achilles' heel of LT. The management of bile leaks usually involves bile duct reconstruction with Roux-en-Y hepaticojejunostomy in the immediate post-transplant period or endoscopic decompression of the bile duct and percutaneous drain insertion. For AS, the management involves endoscopic dilatation, stent insertion or surgical revision. The NAS or ischaemic-type biliary lesions (ITBL) are the most severe form of biliary strictures, where there is widespread desquamation of biliary epithelial cells with formation of biliary casts, multiple segmental stenosis and a picture similar to PSC. This can happen within a few weeks, months or years after transplantation and the incidence is 25–30% with DCD grafts but is less common in DBD grafts. The management will include imaging to rule out HAT or stenosis, dilatation of dominant strictures, ursodeoxycholic acid to increase bile flow and lower the lithogenicity and antibiotic maintenance therapy to prevent recurrent episodes of cholangitis. Eventually most patients will require retransplantation.

Revision #1

Created 2025-12-31 15:31:48 UTC by Omar Ayman

Updated 2025-12-31 15:31:48 UTC by Omar Ayman