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# Imaging of the post –transplant liver: an overview

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## Abstract

Liver transplant is the ultimate treatment available for end-stage chronic liver disease, secondary to the high prevalence of chronic hepatitis. The liver donated for transplant may either be obtained from a living or deceased donor. Although the surgical anastomotic points for both are different owing to the necessary differences of tissues taken from the deceased and the living, the general complications remain the same with broad categorisation into parenchymal, vascular and biliary complications apart from the general surgical complications inherent to any surgery. This makes it relatively easier to screen the sites for focussed post-transplant imaging. As hepatic imaging plays a crucial role in the evaluation of hepatic parenchyma and its vascular and biliary connections, the current overview was planned to summarise the key imaging features to be looked for in the evaluation of a transplanted liver. Ultrasound, including Doppler scan, remains the main investigation with additional information from contrast-enhanced computed tomography, magnetic resonance imaging or angiography for detailed hepatic arterial, vena cava and/or portal vein complications.

**Key Words:** Liver transplant, Living donor, Deceased donor, Anastomoses, Post-transplant imaging.

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## Introduction

Liver transplant is now considered the definitive treatment of pathologies causing end-stage liver failure. An orthotopic allograft taken from a deceased donor is the most common procedure in which the donated liver is transplanted to replace either the whole or part of the diseased liver.[1, 2] In case of living donor, the left lobe of donor, from segments II and III, are harvested into the recipient.[1, 2]

Although many customised variations to the basic surgical technique exist, the transplanted liver, on the similar physiological principles as a de novo liver, requires oxygenated and nutritionally rich arterial blood supply, venous drainage for the chemical metabolic output, and drainage of the synthesised bile. This necessitates connecting the donor portal vein, hepatic artery, inferior vena cava (IVC), and the bile duct to that of the recipient, making four important sites of post-transplant complications. Native anatomic variations also require innovative adaptions in the surgical techniques.[2]

Certain physiological changes are expected in the early postoperative period (Table 1), which subside after about 10 days to three weeks. Familiarity with these findings are essential for both the reporting radiologist as well as the surgical specialist so as to avoid misinterpretation of normal appearances into abnormal findings, and missing the early warning signs of impending complications.

Because of its ability to visualise the anatomy of the living without incising the body, and including structure, drainage and vascular flow pathways, and sometimes even the physiology, radiological imaging plays a key role in the evaluation of the harvested liver and its vascular and biliary anastomoses. Among the available diagnostic armamentarium, ultrasound remains the first line of assessment, providing comprehensive assessment of parenchymal, vascular (with Doppler) and biliary morphology. It is economical, swift and readily available even by the bedside, with the only limitation being operator dependence.[1-5]

The current overview was planned to summarise the key imaging features to be looked for in the evaluation of a transplanted liver.

**Normal post-transplant ultrasound features in the early postoperative period (7-10 days):**Heterogeneous or slightly stormy-sky appearance due to reperfusion oedema in the early postoperative period is seen. Also, free fluid is commonly seen in the peri-hepatic space, but quite likely extend to the sub-phrenic space (Figure 1). Pleural effusion (PE) is extremely common, but resolves over time. Further, foci of increased echogenicity are seen due to haemorrhage. These resolve with time usually by 3 weeks, when they liquify, lose the echogenicity, and finally resolve in about 2 months' time. Lastly,

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pneumobilia is commonly seen with entero-biliary anastomosis, giving a starry-sky appearance1-4. (Figure 1)

**Doppler scan**

With respect to arterial evaluation, the hepatic artery shows a peak systolic velocity (PSV) usually <200cm/sec, which may increase transiently with prolonged systemic acceleration. The resistive index (RI) ranges 0.55-0.8, but may rise to 1.0 with absent forward diastolic flow in the immediate postoperative period. The portal vein (PV) shows aliasing and increased PSV (>85cm/sec) in the early postoperative period which returns to biphasic hepatopetal flow without aliasing. A Doppler scan also encompasses IVC.

**Potential complications**

The most common reasons for suspecting complications are a worsening liver function profile which necessitates establishing the patency of all the vascular connections to rule out parenchymal infarction secondary to thrombosis or stenosis. The other suspicious clinical scenarios are hypotension, abdominal distension, and fever with worsening hypochondrial pain which suggests leakage, and infection either within the hepatic parenchyma or of the collections forming in and around the transplanted liver (Table 1).

**Vascular complications**

The hepatic artery is the most commonly involved vessel with a reported frequency of 4-12% thrombosis and 5-11% stenosis, followed by PV complications (1-2%).[1] All three vessels are likely to develop stenosis and thrombosis, and are best evaluated with Doppler scan. Their consequences are best evaluated with contrast-enhanced computed tomography (CECT) scan, or less commonly with magnetic resonance imaging (MRI). Catheter angiography is better for therapeutic purposes, while contrast-enhanced sonography and multi-detector CT angiography (CTA) improve the non-invasive diagnosis in dubious cases.

**Hepatic arterial complications**

Hepatic artery anastomosis is checked at the porta hepatis, the anastomotic site and at the bifurcation of the main right and left hepatic arteries. The normal anastomotic appearance is often called a 'fish-mouth' appearance, and should not be mistaken for a pseudoaneurysm.

**Hepatic arterial**

Thrombosis starts manifesting as absent diastolic flow, with systolic dampening becoming completely absent finally. This is often referred to as the 'syndrome of impending thrombosis', with 92% diagnostic accuracy.[1]

**Table-1:** Summary of normal post-transplant findings and complications expected on hepatic imaging.

| | Normal post-transplant liver appearances | Complications to be looked for | Optimal imaging modality |
|---|---|---|---|
| General | Right sided pleural effusion | Collections with or without superimposed infection | Ultrasound |
| Hepatic Parenchyma | • Periportal oedema with starry sky appearance on ultrasound, and lucency on CT scan<br>• Normal enhancement | • Focal abscesses<br>• Recurrent HCC or PTLD (after 5 years | • Ultrasound<br>• Contrast enhanced CT for hepatic infarction |
| Hepatic artery | • Low resistance hepatic arterial flow- RI 0.5-0.7<br>• Hepatic arterial flow velocity 30 cm/sec or less | • Low RI (<0.5)<br>• Turbulent high velocity flow at the anastomotic site (>30 cm/sec) in case of stenosis<br>• No flow if thrombosed<br>• Pseudoaneurysm<br>• Haemorrhage<br>• Arteriovenous fistula | • Ultrasound with Doppler<br>• Contrast enhanced CT for hepatic infarction |
| Portal vein | Continuous hepatoportal flow with patent lumen | • Thrombosis<br>• Turbulent flow due to anastomotic stenosis<br>• Arteriovenous fistula | • Ultrasound with Doppler |
| IVC and hepatic veins | • Pulsed waveform conforming to cardiac pulsations, with patent lumen and hepatofugal flow | • anastomotic stenosis<br>• phasic flow variations | • Ultrasound with Doppler |
| Biliary tract | • Mild biliary narrowing at the anastomotic site | • Biloma due to leak from anastomosis or intrahepatic ducts<br>• Anastomotic stricture/ obstruction<br>• choledocholithiasis | • Ultrasound<br>• MRCP |

CT: Computed tomography, IVC: Inferior vena cava, PTLD: Post transplant liver disease , MRCP: Magnetic Resonance Cholangiopancreatography .

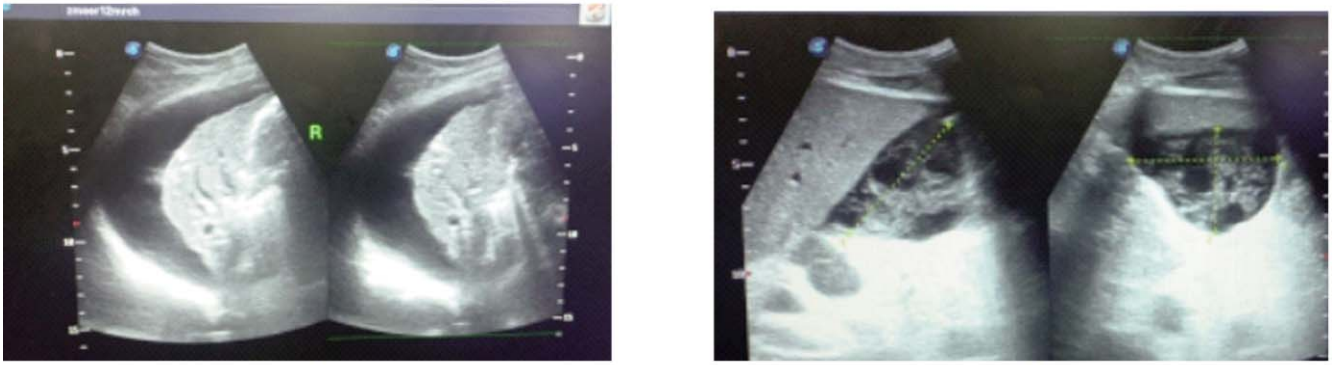


**Figure-1:** Greyscale ultrasound images showing perihepatic fluid collections, organised haematoma, intrahepatic focal abnormalities and echogenic sutures.

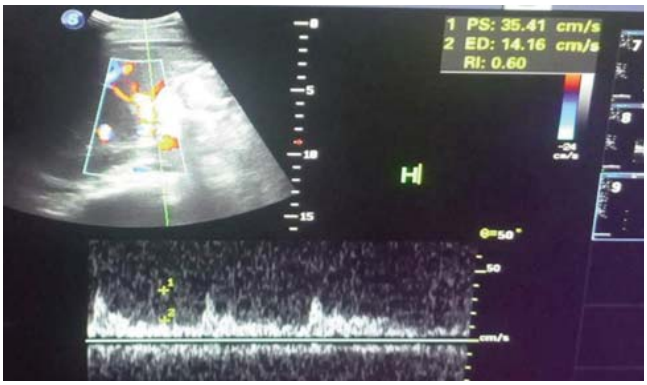


**Figure-2:** Hepatic arterial insufficiency in a post-transplant, normotensive, 35-year-old male.

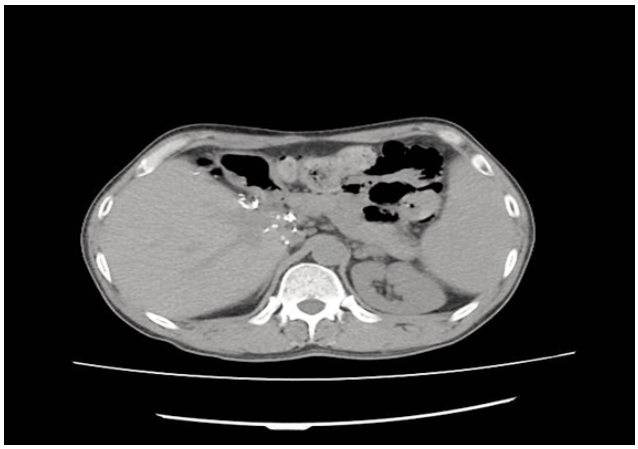

**Figure-3:** Axial plain computed tomography (CT) scan showing biliary, portal and arterial vascular anastomoses marked by hyper-dense sutures.

Care should be taken not to over-diagnose it in the presence of high- grade stenosis, or hepatic oedema; or miss it in case of collateral formation. Hepatic arterial stenosis is manifested as low RI (<0.6) with 100% sensitivity and 80% specificity (Figure 2). Diagnosis is augmented by using ultrasound contrast agents, while catheter angiography remains the gold standard for diagnostic-cum-therapeutic approach. The multi-detector CT (MDCT) and catheter angiography signs for stenosis and thrombosis are tapered narrowing and abrupt cut-off at the anastomotic site, respectively (Figure 2).

Pseudoaneurysm is an uncommon complication of infection or intervention at the anastomotic site, and can lead to rupture, fistula formation and massive haemorrhage. The imaging features include a new-onset peri-portal cystic lesion or arterial outpouching showing turbulent flow, and swirling on colour Doppler (ying-yang sign). Differential diagnosis includes venous varices.

**Portal venous complications**

Although less common than arterial ones, thrombosis presenting with portal hypertension and oedema is part of the complications, with PV getting anastomosed at the main portal vein site (Figure 3).

Flow velocity should be measured across the anastomotic site, and a three times increased velocity gradient across the anastomosis suggests significant vascular abnormality (Figure 4A-B).

Thrombosis appears as the universal echogenic lumen with absent flow in acute complete thrombosis, and cavernoma and collateral venous channels in chronic thrombosis. The stated sensitivity and specificity for sonographic and Doppler signs are 73% and 95% for a peak anastomotic velocity >125cm/sec, and 73% and 100% for anastomotic-to-preanstomotic ratio of 1.3.[6] It is easily visualised on CT or MR angiography, and also on direct and much more invasive percutaneous trans-hepatic portography, all of which have been eclipsed by the ease and accuracy of Doppler sonography for the

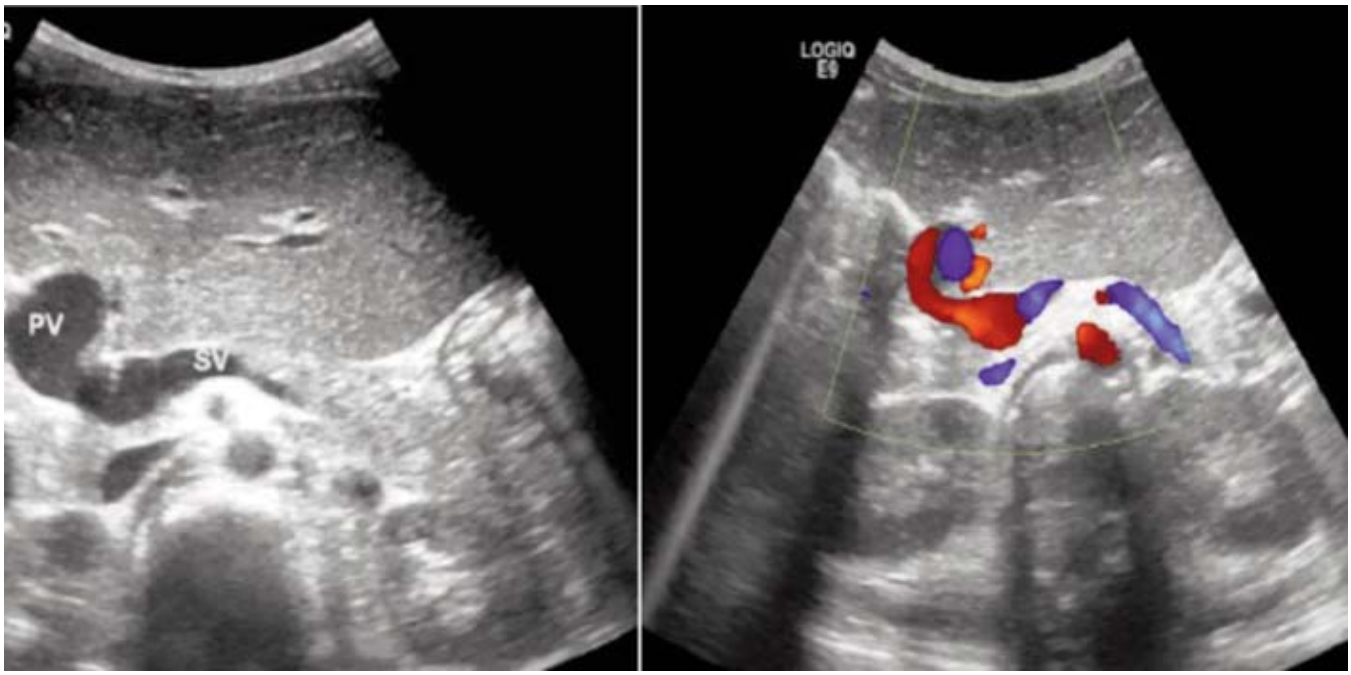


**Figure-4-A:** The anastomosed portal vein and how it should be evaluated across the anastomosis .

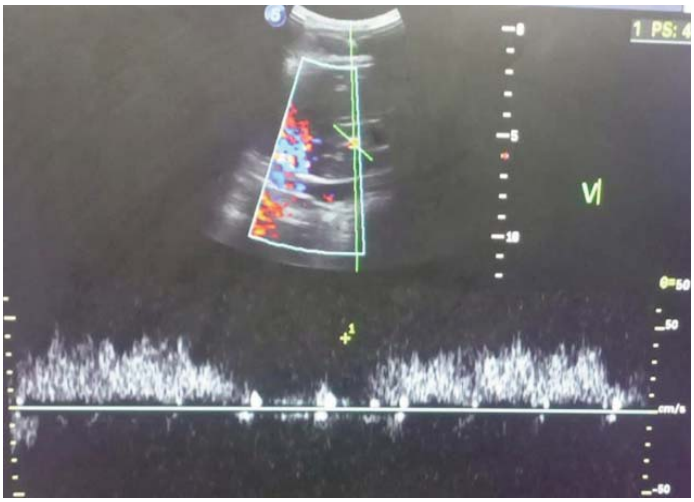

**Figure-4-B:** Normal hepatopetal portal vein flow with respiratory phasic variation in a post-transplant patient. .

diagnosis of PV complications.[7]

**IVC and hepatic complications**

IVC and hepatic complications also occur at the anastomotic site and are very rare being <1%.1 The IVCs of the recipient and the donor are anastomosed after retro-hepatic recipient IVC resection (Figure 5). The underlying pathogenesis entails iatrogenic perioperative injury, cavo-caval dehiscence and suboptimal venous drainage, causing Budd-Chiari syndrome. Screening ultrasound shows enlarged liver with pleuro-peritoneal fluid. Doppler sign of persistent monophasic flow, as against the normal triphasic or biphasic flow (Figure 6), narrow venous calibre, absent respiratory or cardiac, and a prognostic index (PI) value <0.45,6 are all fairly diagnostic. CECT scan shows mosaic enhancement in case of hepatic venous thrombosis.

**Biliary complications**

Last, but certainly not least, biliary complications occur with a frequency of 10-32%,[8] and usually take three weeks to three months to develop. They are said to be the result of ischaemic-reperfusion injury, causing sub-cellular mitochondrial damage.[8] This leads to cholangiopathy and strictures with all the consequent stasis, and its attendant complications.[9] Another suggested mechanism is vasculopathic or neuropathic dysfunction of the ampulla of Vater. Mucocoele of the cystic duct remnant, redundant bile ducts and Sphincter of Oddi dysfunction are all likely to occur rather late after surgery.[10]

CBD (common bile duct) anastomotic stricture occurs at a staggering frequency of 47% among the biliary complications,[9] and may show thickening of walls due to sclerosing cholangitis secondary to mitochondrial damage, being the most marked in the biliary system. This also causes intraluminal sludge, which may lead to calculi formation over due course of time.

Bilomas are early complications formed by leaks (frequency being 23%)10 adjacent to anastomosis, exert extrinsic pressure and may cause effects on viscera. These

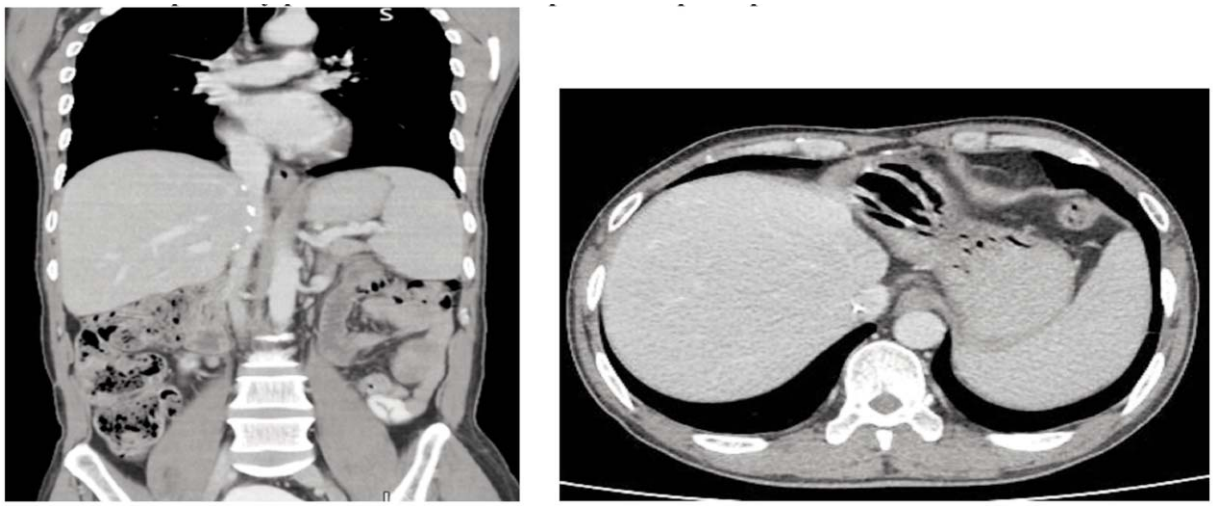

**Figure-5:** Contrast-enhanced computed tomography (CT) scan showing the retro-hepatic inferior vena cava anastomosis marked by the hyper-dense asterisk sign in the axial section, produced by the metallic suture.

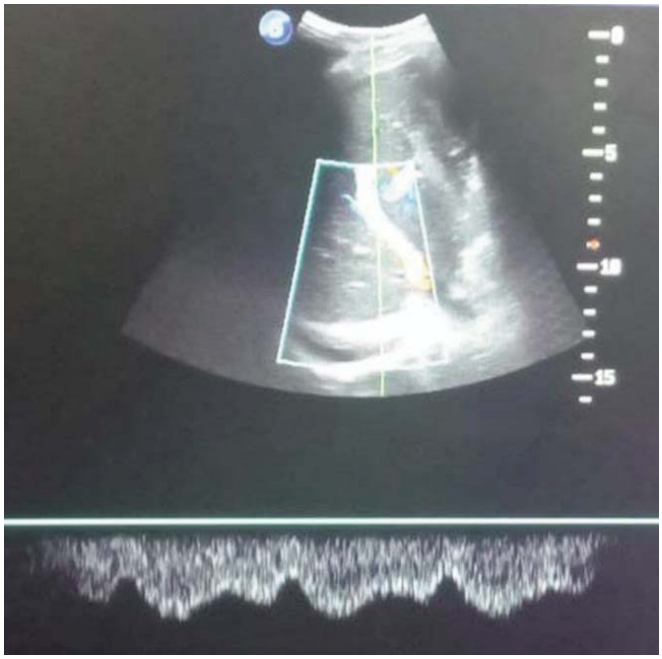

**Figure-6:** Monophasic flow in the right hepatic vein in a post-transplant patient suggestive of inferior vena cava (IVC) abnormality.

are well-documented by CT scan, while MRCP (Magnetic resonance cholangiopancreatography) and ERCP (Endoscopic retrograde cholangiopancreatography) can both localise and visualise the exact site of leakage.[10]

Biliary casts, sludge and calculi occur in about 6% hepatic transplant patients,[9] and they are diagnosed with ultrasound and MRCP. Cast is usually seen as a T1-hyperintense intra-ductal signal on unenhanced MRI; calculi produce signal dropout on heavily T2-weighted MRCP, and duct-within-duct sign is diagnostic of biliary mucosal sloughing.[11]

While ultrasound is the universal screening modality, MRCP is the gold standard for biliary tract pathologies, and the protocol is tailored to detect casts and mucosal detachments. CT is only used where MR is either not available or contraindicated due to some magnetisable implant.

'Vanishing bile duct syndrome' is a rarely encountered pathology with focal destruction of bile duct leading to foci of bile stasis without actually centripetally draining intrahepatic bile ducts visualised on imaging. It is actually an immune-mediated rejection and a histological diagnosis of exclusion.[12]

**Hepatic parenchymal complications**

Infarction and abscesses are two most common complications. While the vascular cause is evaluated with Doppler scan, the hepatic parenchyma is best evaluated with CT scan, which detects micro-abscesses as well as maps out the infarction as non-enhancing geographic areas of low density; abscesses are hypo-dense peripherally enhancing lesions.

Post-transplant fatty liver disease takes around five years to develop, and is more frequent if fatty liver was the indication for transplant.[13] It may occur in as many as 78-88% cases, progressing to steatohepatitis in as many as 41%.[13] Compared to the gold standard biopsy, many non-invasive imaging modalities, like ultrasound, MRI, elastography and CT scanning, may be used with multiple parameters. While MR is said to be highly accurate for hepatic fat detection, software availability remains a limitation.

Likewise, there may be recurrence of chronic liver disease

malignancy for which transplant was initially indicated. In addition, immunosuppression or immune deficiency may lead to new-onset malignancies, like lymphomas, all which have the same features as in the general population, and require regular imaging surveillance.

## Conclusion

Biliary and hepatic arterial complications are extremely common after liver transplant. These require regular surveillance, and radiological imaging provides reliable non-invasive monitoring. Ultrasound with Doppler is the mainstay of radiological surveillance with other modalities contributing effectively when judiciously used.

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