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Liver Transplant Complications—A Pictorial Review

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Abstract

Liver transplantation is the most definitive treatment for decompensated chronic liver diseases, acute hepatic failure, and localized hepatocellular carcinomas. With the widespread use of extensive preop evaluation and advanced surgical techniques, the success rate of liver transplantation has dramatically risen over the decade. In a world of increasing demand for liver donors from brain dead donors, a rising trend is seen in the acceptance of living donor liver transplantation procedures. However, postoperative surveillance needs a lower threshold for early imaging to identify the most dreaded complications for salvaging the graft. This pictorial essay aims to categorize the postliver transplantation complications and various imaging findings to diagnose them. The complications can be broadly classified into vascular and nonvascular complications. The most important concern among vascular complications is hepatic artery thrombosis, which frequently results in graft failure. The nonvascular complication comprises biliary origin, infection, and immune response. Multimodality imaging is the need of the hour, which includes ultrasound as the primary tool for gross evaluation, followed by triphasic computed tomography and magnetic resonance imaging (MRI). Newer techniques in MRI, like diffusion-weighted imaging, arterial spin labeling, diffusion kurtosis imaging, blood oxygenation-level dependent, and magnetic resonance elastography, can also be used to diagnose these complications. Interventional management through minimal access has become the first line of management in certain vascular and nonvascular complications, which can salvage the graft.

Keywords
- liver transplantation
- thrombosis
- biliary strictures
- interventional radiology

Introduction

The world's first successful liver transplant was performed by Dr. Thomas Starzl in the 1960s.[1] In the current era, an increasing number of surgeries are performed for various liver pathologies which improve life expectancy. Liver transplant, being the last resort for the treatment of end-stage chronic liver disease as well as acute liver failure, requires strict vigilance for assessing long-term graft survival. Since

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acute rejections are diagnosed only by liver biopsy, noninvasive imaging techniques are used to rule out other vascular and nonvascular complications. After rejection, vascular complications are the second most common cause of graft dysfunction.[2] Therefore, imaging plays an important role in the surveillance of post-liver transplantation (LT). Posttransplantation complications are classified as vascular and nonvascular complications, which in turn are again divided into biliary, intraparenchymal, and intra-abdominal complications. In this pictorial essay, we demonstrate a spectrum of findings using various imaging techniques like B-mode ultrasound (USG), Doppler imaging, computed tomography (CT), and magnetic resonance imaging (MRI) for diagnosing these complications. Tremendous improvement and wide acceptance of interventional management using minimally invasive procedures is the current trend in treating various vascular complications.

Surgical Techniques

While deceased donor LT utilizes the whole liver graft from the donor, in live donor transplantation most commonly right liver lobe from the donor is implanted in the recipient. In right lobe donor hepatectomy, the middle hepatic vein is left with remnant left lobe in the donor. In smaller adults and older children, left lobe of the liver with middle and left hepatic vein drainage can be utilized as a viable graft .The most frequent procedure is a right hepatectomy with a resection plane just to the right (about 1 cm) of the middle hepatic vein.[3] The alternate method is a left hepatectomy, in which the liver is often divided into two parts in a similar plane which is reserved for those where the small-sized liver graft is adequate. For smaller pediatric patients, left lateral segmentectomy is specifically used.[4] The most common hepatic arterial anastomosis is end-to-end between donor common hepatic artery and recipient proper hepatic artery in deceased donor transplantations[5] and donor right (or left) hepatic artery and the recipient right/left hepatic arteries in case of live donor LTs. Hepatic vein anastomosis may be performed by either end-to-side cavo-caval "piggyback technique" or side-to-side cavo-caval anastomosis as per surgeon's preference. Portal vein is also anastomosed with end-to-end technique between the donor and recipient vessel. Biliary anastomosis is usually performed end-to-end between the donor and recipient common bile ducts.

Imaging Modalities

Ultrasound

This is the first-line imaging modality used to assess postliver transplant patients in the immediate postoperative period.[6] USG is a widely available noninvasive imaging modality for quick evaluation of the liver parenchyma. B-mode imaging, color Doppler, spectral evaluation, and elastography are the diverse techniques to assess the graft function. Being a nonionizing and cheaply available modality, it can be repeated multiple times in appropriate clinical settings at the bedside without the need for radiating or shifting the patients. It is done daily during the first week of the postoperative period and as and when required in the rest of the postoperative period. B-mode is usually done to screen the hepatic parenchymal echotexture and look for any obvious intra- and perihepatic collections, biliary radicle dilatation, or periportal cuffing (►**Fig. 1**). Color Doppler is used to determine the presence of blood flow and its direction with respect to the liver. Any absence of flow warrants immediate further assessment by triple-phase CT to exclude thrombosis of the culprit vessel. Spectral waveform acquisition gives information about the peak systolic velocity (PSV), resistive index (RI), acceleration time, and acceleration index. Hepatic artery is usually assessed intrahepatically which shows rapid upstroke with an acceleration time of less than 0.08 seconds. The presence of a forward diastolic component with an RI value of 0.55 to 0.7 in hepatic arteries

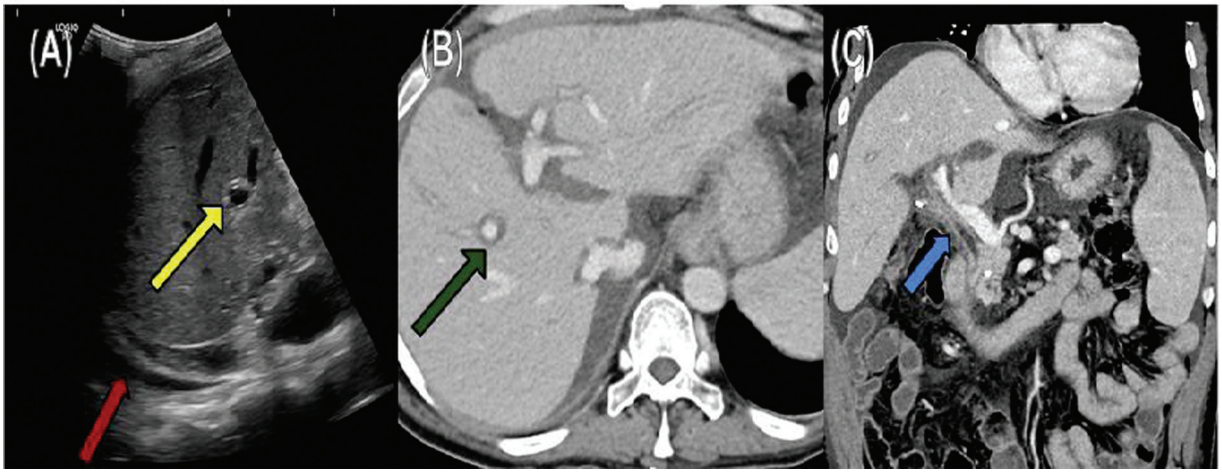

Fig. 1 A 41-year-old male patient who underwent liver transplant 2 weeks back, presented with history of fever and elevated bilirubin levels with deranged liver function test (LFT). Ultrasound (USG) abdomen of the liver showed diffuse periportal hyper echogenicity (**A**, yellow arrow) with mild right pleural effusion (red arrow). Contrast-enhanced computed tomography (CECT) abdomen axial (**B**) and coronal (**C**) image of the upper abdomen depicted diffuse intrahepatic periportal hypodensity (green arrow) with diffuse common hepatic duct enhancement (blue arrow) suggestive of periportal edema with cholangitis.

is essential for adequate perfusion of the graft. RI value of more than 0.8 is a nonspecific finding which indirectly indicates various causes of graft dysfunction as well as could also be a normal finding in the immediate postoperative period in view of graft edema. The PSV is usually within 200 cm/s at the anastomotic site and less than 103 cm/s in the intrahepatic branches. Portal vein is assessed extrahepatically which demonstrates hepatopetal, monophasic, and continuous phasic spectrum. Sainz-Barriga et al reported that portal flow volume of less than 180 mL/min per 100 g liver weight (LW) showed poor survival rates.[7] Asencio et al proposed that portal flow volume exceeding 250 mL/min per 100 g LW is indicated for controlling portal venous pressure.[8] Normal hepatic veins show a normal triphasic pattern of flow which can be demonstrated in the inferior vena cava (IVC) also.[9] Using newer techniques like shear wave elastography, the parenchymal stiffness can be evaluated for follow-up patients. Limitations of USG include poor acoustic window secondary to postoperative scars and dressing and it is operator-dependent.

Computed Tomography

CT remains the confirmatory and complementary noninvasive imaging modality because of its rapid acquisition, and excellent spatial and temporal resolution. Noncontrast CT is essential to identify the graft and its bed as well as to identify any hyperdense hematoma. Fluid density collections can also be identified in the perigraft regions which can extend to the pelvic cavity. The gross evaluation of the abdominal cavity is also looked upon to screen for other associated pathologies. Triple-phase contrast-enhanced study is performed mainly to assess the vascular status of the graft. Any abrupt change in RI value in the Doppler scan should raise concern for hepatic artery thrombosis (HAT)/stenosis (HAS), which is evaluated in the arterial phase. Similarly, portal and venous phases are used to analyze the portal vein and hepatic veins draining to the IVC. Multiplanar reconstruction can be used to visualize coronal and sagittal planes for optimal assessment of small vessels in all directions. The virtual reconstruction technique depicts three-dimensional images of complex anatomic structures.

Magnetic Resonance Imaging

MRI is used only for the assessment of certain pathologies which are inconclusive by the above-mentioned imaging modalities. It is limited by longer acquisition time, lack of accessibility, poor patient factors, and lower spatial resolution than CT. Noncontrast MR techniques can be used to assess the hepatic vessels; however, contrast administration is necessary for precise evaluation. A noninvasive comprehensive evaluation of the biliary system is possible only by magnetic resonance cholangiopancreatography (MRCP). The likelihood of biliary complications in the graft may be increased as a result of variant anatomy, which may necessitate two separate biliary-enteric anastomoses or ductoplasty, with the creation of a common ductal opening.[10] Fluid characterizations to differentiate abscess, hematoma, and biloma can be done using advanced imaging modalities, likely Dixon sequences, diffusion imaging, and cholangiography technique.

Digital Subtraction Angiography

Digital subtraction angiography (DSA) can be used as a diagnostic as well as a therapeutic tool. It has a superior temporal resolution as compared to CT or MRI and thus it helps in assessment of flow characteristics in obstructive lesions to guide further management. Definite diagnosis of vascular pathologies is made with the help of the DSA, particularly arterial stenosis or thrombosis, with the added advantage of treating the pathologies in the same sitting. Postbiopsy or infective pseudoaneurysm (PSA) is also confirmed by DSA which warrants interventional management. Unique advantages include the elimination of motion artifacts and providing a superselective angiogram for guiding interventional treatment.

Vascular Complications (►Table 1)

Hepatic Artery

Exclusive knowledge about the vascular anastomosis is necessary to optimally assess the vessel status. In orthotopic LT, the donor coeliac artery is anastomosed with the right or left hepatic artery. In the case of a diseased hepatic artery, a

Table 1 Vascular complications following liver transplantation

Vascular complications	Clinical features/lab parameters	Imaging modality of choice
Hepatic artery thrombosis	• Early postoperative period • May present with fulminant liver failure • Elevated liver enzymes, biliary leaks, rarely septicemia	Contrast-enhanced CT
Hepatic artery stenosis	• Insidious course with vague abdominal discomfort • Elevated liver enzymes	
Portal vein thrombosis/stenosis	• Deranged LFT in late postoperative period • Signs of portal hypertension (ascites, gastrointestinal bleeding)	
Hepatic vein/IVC stenosis/thrombosis	• Signs of Budd–Chiari syndrome – abdominal distension/pain, ascites, anterior abdominal wall collaterals, splenomegaly	

Abbreviations: CT, computed tomography; IVC, inferior vena cava; LFT, liver function test.

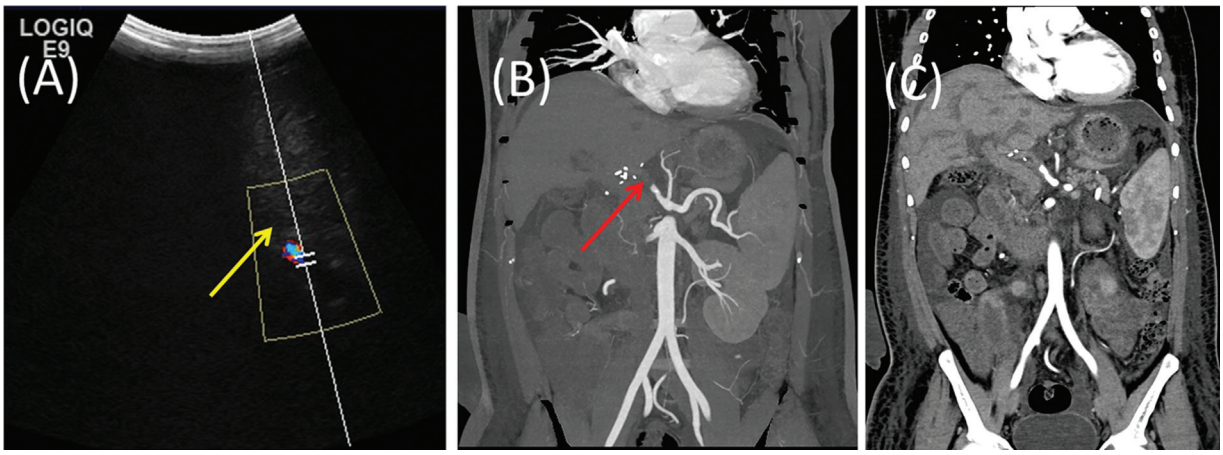

Fig. 2 A 37-year-old male patient post DDLT (deceased donor liver transplantation) - postoperative day (POD) 4. Routine ultrasound (USG) Doppler surveillance (**A**) showed absent color flow (yellow arrow) in the intrahepatic portions of hepatic artery, raising the possibility of thrombosis. Contrast-enhanced computed tomography (CECT) abdomen coronal maximum intensity projection image (**B**) shows non-opacification of hepatic artery (red arrow) consistent with vascular thrombosis. CECT coronal multiplanar reconstruction images (**C**) show diffuse body wall edema with mild ascites.

graft interposition is done between the anastomosis. The hepatic artery being the sole blood supply for biliary ducts, vigilant surveillance is necessary to rule out biloma or biliary strictures which could indirectly represent HAS/occlusion.

(1) Hepatic artery thrombosis

The most common and most dangerous vascular complication following LT is HAT which usually occurs between 2nd and 15th weeks.[9] It can occur in 4.8 to 9% of patients postliver transplant patients. Risk factors include prolonged cold ischemia time, graft rejection, ABO incompatibility, and pediatric population.[11] Hepatic artery is vital as it is the sole supply to the biliary tree. Untreated HAT will lead on to biliary ischemia and strictures with adverse outcomes. USG is the initial modality of choice which has high sensitivity and specificity for HAT. It is identified as the absence of flow in the visualized hepatic arteries in the Doppler study (►**Fig. 2**). Reduced diastolic component and peak systolic velocities are predictors of imminent HAT. False positive findings may be secondary to diffuse severe spasm of the hepatic arteries or due to reduced cardiac output.[9] The absence of flow in Doppler evaluation warrants contrast-enhanced CT (CECT) which can accurately determine the length and extent of thrombosis of hepatic arteries. MR angiography can also depict similar findings in cooperative patients. Most patients need surgical exploration or endovascular thrombectomy in the event of an early posttransplant HAT. In case the attempts to salvage the hepatic artery fail, retransplantation is indicated. Delayed HAT is usually managed conservatively.[12]

(2) Hepatic artery stenosis

Stenosis of the hepatic arteries usually occurs within the first 3 months of surgery with a median time of 100 days.[2,13] The incidence is around 4 to 11%. It usually occurs due to clamping injury, graft rejection, and vasa vasorum disruption. Increased velocity of more than 200 cm/s at the site of suspected stenosis or anastomosis with aliasing in color Doppler suggests HAS. Indirect findings distal to the stenosis are an increase in diastolic component with reduced resistance index to less than 0.55, a delayed acceleration peak (more than 80 ms) together producing the characteristic parvus et tardus pattern of flow.[14,15] Other ancillary findings in Doppler evaluation include an anastomotic to the pre-anastomotic ratio of 3:1. Three-dimensional reconstruction of CECT depicts focal stenosis of the hepatic artery and its branches (►**Figs. 3** and **4**). HAS can predispose to thrombosis and thereby pose a risk to the liver. As mentioned in the previous section, HAS can impair blood supply to the biliary tree resulting in strictures and ischemic cholangiopathy. Early posttransplant HAS is usually related to a surgical issue and surgical correction is preferred; angioplasty is likely to pose a risk of rupture at the anastomotic site. An interventional procedure like percutaneous transluminal balloon angioplasty is the treatment of choice in late HAS, occurring

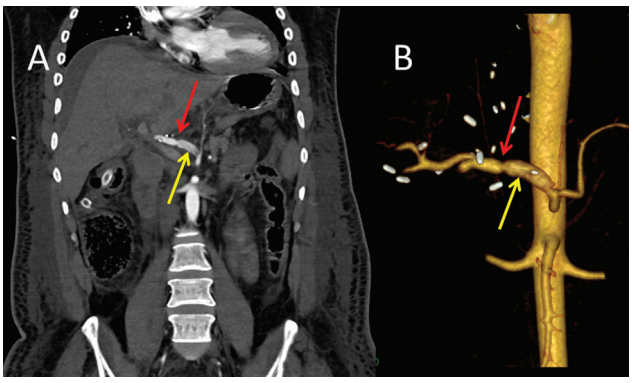

Fig. 3 A 44-year-old male patient post deceased donor liver transplantation (DDLT) - postoperative day (POD) 5. Coronal contrast-enhanced computed tomography (CECT) image (**A**) shows linear filling defect (yellow arrow) in the proximal hepatic artery with luminal irregularity s/o dissection in the recipient common hepatic artery and focal narrowing at the anastomotic site (red arrow). Virtual reconstructed image (**B**) demonstrates the same findings (yellow and red arrows).

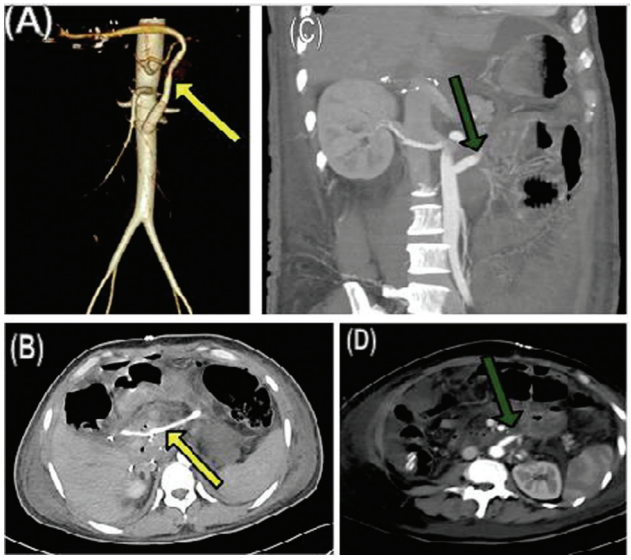

Fig. 4 In the same patient, virtual reconstructed image (**A**) shows infrarenal aorto-hepatic jump graft (yellow arrow) after ligating the native hepatic artery. Contrast-enhanced computed tomography (CECT) axial sections (**B**) of the upper abdomen show linear contrast opacification of aorto-hepatic jump graft (yellow arrow). Mild ascites with mesenteric haziness is also noted. Following deranged lab parameters repeat CECT images in coronal and axial planes (**C** and **D**) showed nonopacification of the jump graft (green arrow) consistent with complete thrombosis of the extrahepatic portions of jump graft.

after the first 30 days of LT. In cases where there is recoil or flow limiting dissection, stent placement may be considered.

(.) Hepatic artery pseudoaneurysm

PSAs are abnormal focal outpouching of the arteries which are lined only by the tunica adventitia. It can occur extrahepatically at the vascular anastomotic site or intrahepatically secondary to needle biopsy or local infection. Though PSAs are silent, intrahepatic rupture or erosion into the biliary tree are dreaded complications of hepatic PSAs. B-mode USG shows anechoic focus along the course of the hepatic artery which on color Doppler shows a characteristic bidirectional yin-yang pattern (to and fro). A bidirectional or sometimes slow monophasic pattern of flow is seen in spectral Doppler.[16] In CT/MRI there is contrast distribution within the lesion similar to that of the arterial vessels. Peripheral PSAs can be managed with superselective embolization of the feeding artery close to the PSA with coils/glue. More central PSAs arising in nonexpendable vessels can be managed with stent graft placement if feasible.

Portal Vein

Portal vein complications are usually less frequent as compared to those in the hepatic artery with a prevalence of 1 to 2%. Early-onset portal vein thrombosis (PVT) is often due to rapidly progressing graft dysfunction whereas late-onset PVT is due to chronic graft rejection. Clinical features range from abdominal pain, ascites, splenomegaly (signs of portal hypertension), varices, or liver failure.

(1) Portal vein thrombosis

PVT may occur at the site of anastomosis (extrahepatically) or intrahepatically. Various risk factors include previous PVT, small-sized portal vein (< 5 mm), hypercoagulable states, prior portal vein surgery (transjugular intrahepatic portosystemic shunt), and interposition of grafts for portal vein reconstruction.[13,17] B-mode USG shows an echogenic thrombus within the portal vein reflecting chronic thrombosis whereas acute thrombosis is seen as anechoic intraluminal focus. Lack of venous flow in the Doppler imaging and focal filling defect at the anastomotic site or beyond in triple-phase CT is the corresponding finding (►**Fig. 5**). Partial

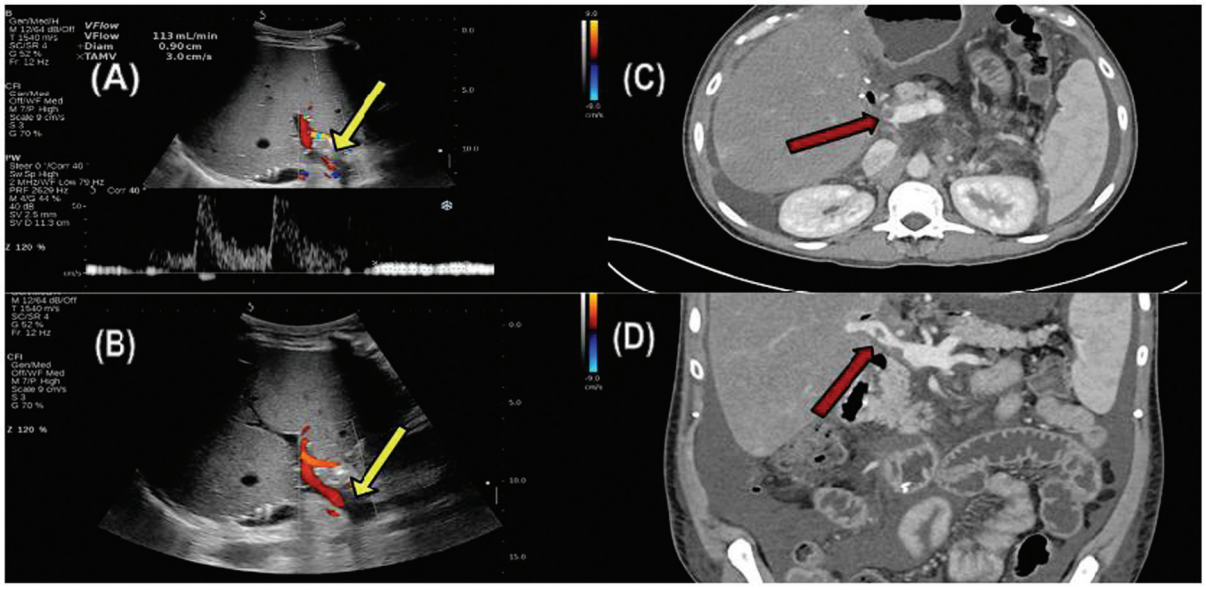

Fig. 5 A 44-year-old male with living donor liver transplantation (LDLT) 1 year back. Color Doppler images (**A** and **B**) show absence of wall-to-wall color in the main portal vein (yellow arrow) secondary to eccentric echogenic thrombus. Reduced flow volume in main portal vein was documented with spectral analysis. Contrast-enhanced computed tomography (CECT) axial and coronal images (**C** and **D**) depict partial eccentric hypodense thrombus in the extrahepatic portions of main portal vein (red arrow), respectively.

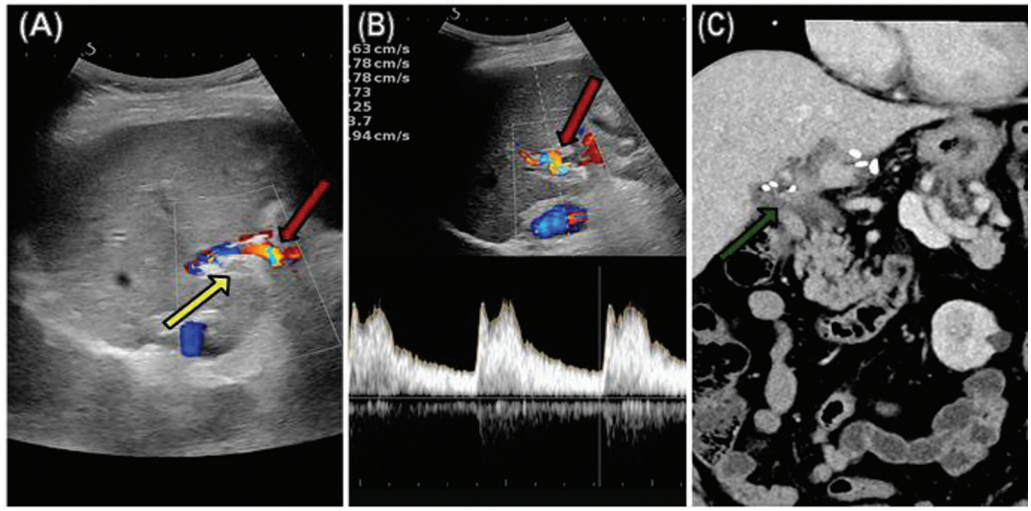

Fig. 6 A 63-year-old male patient with orthotopic liver transplant after 5 months. Ultrasound (USG) images of the porta hepatis shows absent color uptake in the main portal vein (**A**, yellow arrow) with hypertrophied and tortuous hepatic artery (**B**, red arrow)—consistent with thrombosis in main portal vein and compensatory increase in hepatic artery flow. Contrast-enhanced computed tomography (CECT) coronal image (**C**) of the upper abdomen shows nonopacification of main portal vein (green arrow), s/o thrombosis of main portal vein.

filling defect can later evolve to complete chronic occlusion (►**Fig. 6**). PVT can occur in the perioperative period (< 72 hours after transplant), early (< 30 days), or late (> 30 days) periods after LT. Perioperative PVT is associated with a high rate of graft loss (75%) irrespective of treatment. Early PVT also has a poor prognosis and must be managed aggressively. Catheter-directed thrombolysis combined with endovascular thrombectomy or surgical thrombectomy are the treatment options. For thrombolysis, the portal vein can be accessed through transhepatic, transjugular, or transsplenic routes. Late PVT can be managed conservatively with anticoagulation if asymptomatic.[12]

(2) Portal vein stenosis

Stenosis of the portal vein usually occurs at the site of the vascular anastomosis with an incidence of less than 1%.[13] Early portal vein stenosis within the first 6 months is usually due to poor iatrogenic anastomosis whereas late stenosis occurs secondary to neointimal hyperplasia. Abrupt focal narrowing is seen with PSV of > 125 cm/s. The anastomotic to preanastomotic PSV ratio is 3:1.[18] CT/MR depicts similar imaging findings in the form of focal significant narrowing at the anastomotic site. Mild "waisting" of the portal vein is a common finding which should not be mistaken for stenosis. This often occurs due to discrepancies in the size of the donor and recipient vein anastomosis. Invasive diagnostic modalities likely transhepatic portography can also be used to diagnose portal vein stenosis with a cutoff of > 5 mm Hg gradient.[19,20] CT/MR angiography can also be used to identify flow-limiting stenosis in the extra- and intrahepatic portal veins. Endovascular treatment in the form of a percutaneous transhepatic portal vein angioplasty with or without stenting is the mainstay of treatment. Primary stent placement is favored due to the increased risk of hemorrhagic complications with repeated transhepatic access.

Hepatic Veins and IVC

Several surgical techniques can be used to anastomose donor IVC to the recipient. End-to-end anastomosis of donor and recipient IVC following resection of the recipient retrohepatic IVC is the most commonly used technique. Anastomosis of the donor IVC to the stump of the recipient hepatic vein without resection of retrohepatic IVC is called the "piggyback" technique. The usual site of stenosis occurs at the anastomotic site and hence the technique of surgical anastomosis must be known prior.

(1) IVC stenosis/thrombosis

Just like the other vessels, the most common site for stenosis is the anastomotic junction. They are rare complications which usually occur late (more than 6 months). Various risk factors include size discrepancy between donor and recipient IVC, suprahepatic IVC kinking from organ rotation, intimal hyperplasia or fibrosis compression by graft edema, or the adjacent collection and hypercoagulable states.[5,13] Doppler parameters show an increased velocity at the site of stenosis with focal aliasing. CT/MR venography demonstrates focal narrowing/stenosis of the IVC with background features of Budd–Chiari syndrome (hepatomegaly, ascites, pleural effusion) (►**Fig. 7**). Percutaneous angioplasty and stenting have become the preferred treatment providing symptom relief and early clinical outcomes.

(2) Hepatic vein stenosis/thrombosis

Hepatic vein thrombosis may occur in the early postoperative period secondary to twisting of the veins, tight anastomosis, and size discrepancy; however, it occurs late in the course of the postoperative period due to intimal hyperplasia and perivascular fibrosis. Echogenic thrombus (►**Fig. 8**) with loss of triphasicity and decrease in velocity of less than 10 cm/s confirms hepatic vein stenosis. A pulsatility index of less than 0.45 is also indicative of hepatic vein stenosis.[2,21,22]

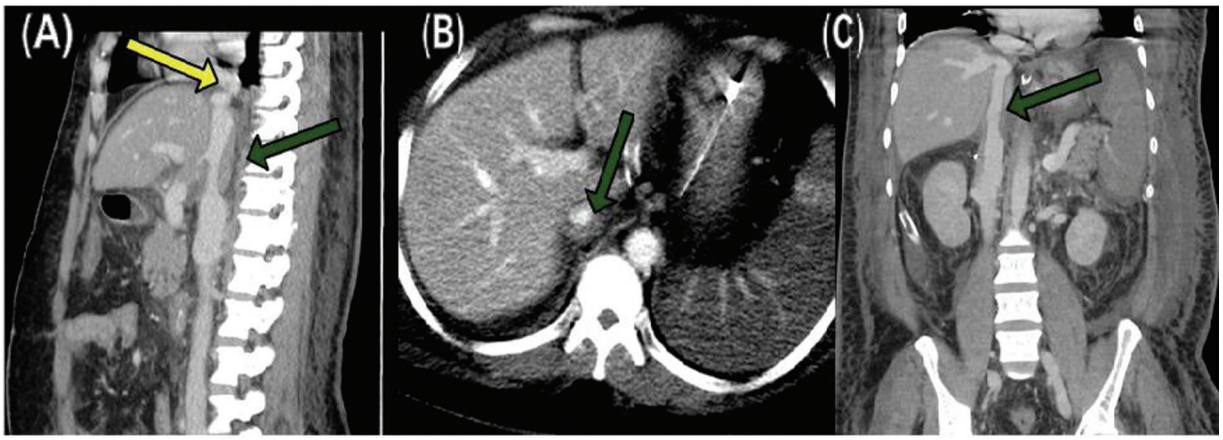

Fig. 7 A 48-year-old male patient who underwent orthotopic liver transplantation had high urine output with deranged renal parameters on 6th postoperative day (POD). Contrast-enhanced computed tomography (CECT) sagittal images of the upper abdomen shows focal critical stenosis (**A**, yellow arrow) in the superior aspect of inferior vena cava (IVC). Axial and coronal images of the same patient show eccentric hypodense nonocclusive filling defect in IVC (**B** and **C**, green arrow)—consistent with partial IVC thrombosis.

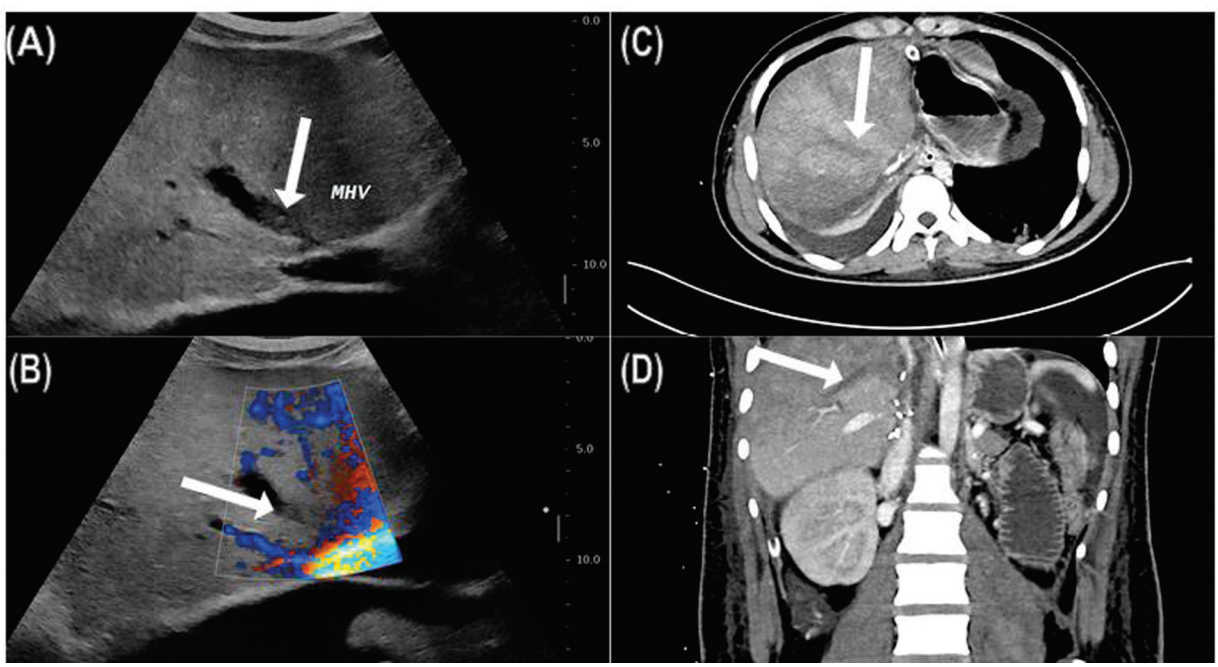

Fig. 8 A 62-year-old male patient—post-deceased donor liver transplantation (DDLT) on 6th postoperative day (POD) routine Doppler surveillance. Ultrasound (USG) B-mode image (**A**) shows iso- to hyperechoic intraluminal thrombus (white arrow) within the right hepatic vein with no color uptake in Doppler image (**B**). Contrast-enhanced computed tomography (CECT) axial and coronal sections (**C** and **D**) of the upper abdomen of the same patient shows nonopacification in the right hepatic vein (white arrow) in the venous phase.

Transstenotic pressure gradient of more than 5 mm Hg warrants treatment. CT/MR venography can also be used to diagnose hepatic vein stenosis/thrombosis. Early presentation often requires a surgical revision of the anastomosis while late presentation is usually managed with percutaneous transhepatic/transjugular angioplasty with or without stenting.[12]

Nonvascular Complications

Biliary Complications

The second most common complication after graft dysfunction is biliary complication.[23] This can be due to biliary leak, biliary anastomotic strictures, stone casts, and sludge (►**Table 2**).

(1) Bile leak

Biliary leaks usually occur in the early postsurgical period (within first month) commonly associated with HAS/HAT which is typically seen involving the distal biliary radicles. The bile leak is most commonly from the biliary anastomosis[13]; other possible sites are bile leaks from the cut surface of the graft and caudate lobe biliary radicals which are left unsutured. Other rare causes include immunological and cytotoxic injury. Bile can leak into the peritoneal cavity or form an organized collection in the

Table 2 Biliary complications following liver transplantation

Biliary complications	Clinical features/lab parameters	Imaging modality of choice
Biliary stricture	• Abdominal pain, yellowish discoloration, elevated bilirubin levels	MRCP
Biliary sludge/calculus	• Obstructive jaundice • Increase in alkaline phosphatase, SGOT, and SGPT	
Biloma/biliary abscess	• Fever, abdominal pain, raised total counts	

Abbreviations: MRCP, magnetic resonance cholangiopancreatography; SGOT, serum glutamic-oxaloacetic transaminase; SGPT, serum glutamic pyruvic transaminase.

form of a biloma. Intrahepatic anechoic cystic focus with no evidence of internal vascularity could represent biloma. Hepatobiliary-specific contrast-enhanced MRCP can demonstrate the exact anatomical site as well as active leakage of contrast into the collections.[24,25] Nonanastomotic leaks secondary to HAT usually require retransplantation. Cholescintigraphy (hepatobiliary nuclear imaging) is a sensitive and specific test for biliary leakage. A bile leak is indicated by the gradual accumulation of radiotracer in the abdomen that does not match the morphologic features of the intestine.[26] A diagnostic pitfall that can happen in patients who have a Roux-en-Y limb placed after hepaticojejunostomy is when normal radiotracer build-up in the limb's blind end is confused with a bile leak.[27] Bile duct leaks are usually treated by placing stents across the site of leakage.

(2) Biliary strictures

Biliary obstruction is the most common nonvascular complication occurring mostly as anastomotic site structures. It is due to intimal hyperplasia and scarring of perianastomotic tissues. Nonanastomotic sites occurring intrahepatically should raise concern for HAT or HAS. Other causes of nonanastomotic strictures include pretransplant biliary diseases (primary sclerosing cholangitis) and focal infections.[13] Biliary radicle dilatation caused by strictures is suspected when bilirubin levels are elevated. However, in a few cases, CT may not demonstrate dilatation of bile ducts. In such a case, MRCP or endoscopic retrograde cholangiopancreatography or transhepatic cholangiography should be performed to determine strictures since even in severe stenosis biliary radicles may not be dilated (►**Fig. 9**).

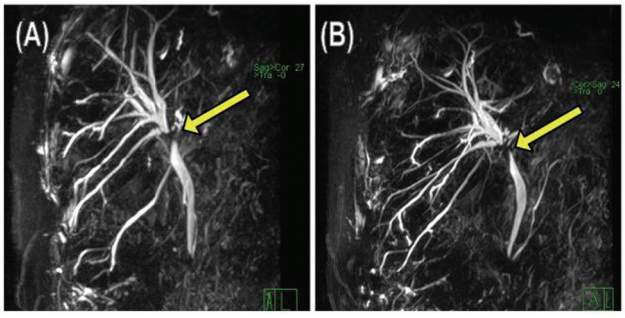

Fig. 9 A 47-year-old male patient, living donor liver transplantation (LDLT) 4 years ago came with h/o elevated bilirubin levels during routine yearly follow-up. Maximum intensity projections of biliary system shows focal short segment tight stricture (**A** and **B**, yellow arrow) involving the anastomotic site.

Extrahepatic biliary strictures may be treated with simple dilatation or rendezvous procedure whereas intrahepatic strictures are usually treated by percutaneous transhepatic biliary drainage. Serial dilatation with periodic upsizing of the drainage tube may be needed for opening the stricture.

(3) Biliary sludge, stones, and casts

Casts and sludge are usually seen within 1 year of transplantation but choledocholithiasis is seen after 1 year. It usually occurs due to biliary stasis from preexisting chronic strictures or alteration of the composition of bile following transplantation. On MRCP this is usually seen as a well-defined T2 hypointense filling defect in the case of stones but shows irregular margins for biliary casts (►**Fig. 10**). This is typically seen in donation after cardiac death cases where significant warm ischemic times might lead to ischemic necrosis of the intrahepatic biliary tree. The occurrence of biliary cast syndrome, which is defined as the presence of hard, black lithogenic material inside the biliary system, is reported to range from 4 to 18% in the literature.[28]

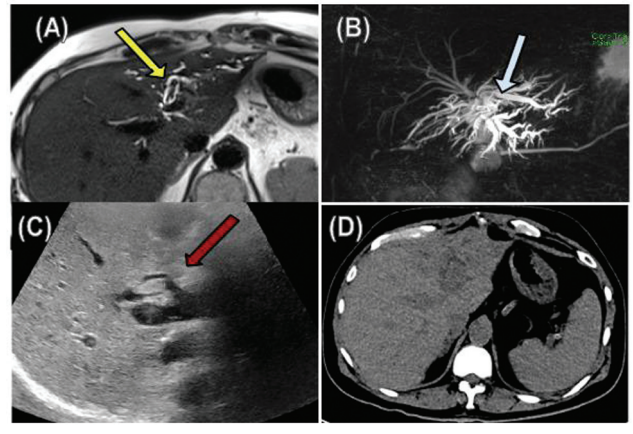

Fig. 10 A 62-year-old male patient living donor liver transplantation (LDLT) 2 years back with elevated bilirubin. T2 HASTE (Half fourier Single-shot Turbo spin-Echo) axial image showed focal hypointense filling defect (**A**, yellow arrow) in the proximal left hepatic duct—may represent calculous/dense sludge. Maximum intensity projections of biliary system (**B**, white arrow) show multiple dilated intrahepatic biliary radicles predominantly on the left side (blue arrow), secondary to short segment stricture involving the junctions of the left and right hepatic duct confluence. Ultrasound (USG) correlation image revealed echogenic focus (**C**, red arrow) with no posterior acoustic shadowing. Corresponding axial computed tomography (CT) image (**D**) showed no e/o radiodense focus within the left hepatic duct, s/o dense biliary sludge/cast.

Table 3 Other complications following liver transplantation

Complications	Clinical features/lab parameters	Imaging modality of choice
Hematoma	• Right upper quadrant pain • Massive bleed leads to acute collapse	Contrast-enhanced CT
Abscess	• Fever, abdominal pain, raised total counts	Contrast-enhanced CT
Graft rejection	• Deranged LFT, hepatic failure, ascites, encephalopathy	Imaging – nonspecific Biopsy – gold standard

Abbreviations: CT, computed tomography; LFT, liver function test.

Intra-Abdominal Fluid Collections (►Table 3)

(1) Hematomas

Perihepatic hematomas are inadvertent complications in the immediate and early postoperative period. Acute hematomas are echogenic in USG with internal echoes on USG. Subacute to late hematomas usually appear as less echogenic collections in the perihepatic and along the paracolic gutters. In the case of acute large hematomas with a significant drop in hemoglobin, CECT should be done to rule out active extravasation. Any hyperdense collections ($>$ 40 HU) in CT in the intra- or perihepatic regions with postcontrast increase in the density confirms recent hematoma with active bleeding (►**Fig. 11**). MRI typically shows T1 shortening seen along the hepatic surface, which can produce a mass effect over the parenchyma in case of very large hematomas. Catheter-directed embolization of the culprit vessel can be attempted to arrest the bleeding.

(2) Abscess

An abscess can occur due to bacteremia or superadded infection of preexisting collection or infarcted/ischemic tissue. Since bile acts as an excellent growing medium for bacteria, superadded infections are much more common in intrahepatic bilomas. It is seen as thick irregular peripheral rim enhancement on imaging with intrinsic air pockets and central diffusion restriction on MRI. Any abscess intrahepatically must be treated with sensitive antibiotics and percutaneously drained to prevent further graft dysfunction.[29]

(3) Seroma

The expected complication following immediate postsurgery is perihepatic seroma which may be ill-defined and/or localized with thin perceptible walls. USG shows an anechoic collection with/without septations. It consists of simple liquid with fluid attenuation ($\sim$10 HU) in CT and T1 hypo- and T2 hyperintensity in MRI. With time seroma generally resolves and hence follow-up USGs are generally not necessary.

Intraparenchymal Complications

(1) Graft dysfunction/rejection

An overall most common complication is graft rejection; however, imaging plays a limited role in its diagnosis. It is divided into acute cellular rejection and chronic ductopenic rejection. Three weeks after the transplant, cellular rejections can occur, and chronic rejection starts 6 weeks to 6 months later. CT images show nonspecific findings which include nonhomogeneity of the liver parenchyma, periportal edema, and differential parenchymal enhancement. Liver biopsy is the gold standard diagnostic test for identifying acute/chronic rejection.[30]

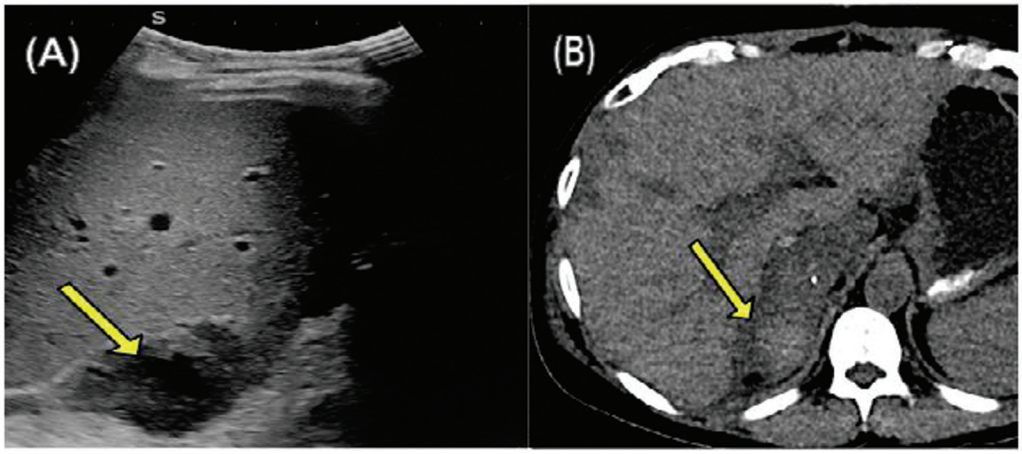

Fig. 11 A 41-year-old post-cadaveric liver transplant status (postoperative day [POD] 20) under routine weekly surveillance. Ultrasound (USG) showed heteroechoic hematoma with central hypoechogenicity (A, yellow arrow) corresponding to the hyperdensity in computed tomography (CT) axial images (**B**).

(2) Neoplasms

Any LT is an immunodeficient state due to the administration of immunosuppressive drugs which leads to posttransplant lymphoproliferative disorder (PTLD). The liver is the most common abdominal organ to manifest as PTLD; however, it may affect any organ with a wide range of spectrum. In the liver, these are seen as hypoechoic nodules in the liver which appear hypodense on CT. They are T1 and T2 hypointense lesions without postcontrast enhancement. This may be accompanied by enlarged lymph nodes in the periportal and para-aortic regions.[31] Neoplasms following LT may be due to the recurrence of primary hepatic malignancy or metastatic disease from a separate primary malignancy. Transplanted patients are at high risk for the development of de novo malignancies, Kaposi's sarcoma, skin, cervical, and breast malignancies. Hepatocellular carcinoma most commonly recurs as lung metastases or as multiple lesions within the liver graft.[32] Proven tumor-related risk factors for hepatocellular carcinoma recurrence after LT include elevated alpha-fetoprotein, tumor grade/stage, and vascular invasion.[33,34]

Splenic Artery Steal Syndrome

Splenic artery steal syndrome is a very rare entity which occurs in the immediate postoperative period.[28] On Doppler evaluation there are increased RI values with a reduced diastolic component in the intra- and extrahepatic arteries accompanied by increased PSV in the portal and splenic vein (►**Fig. 12**). In Angiogram, when the hepatic artery is patent and shows poor sluggish flow with delayed contrast filling in comparison to the rest of the celiac trunk branches, Splenic artery steal syndrome is diagnosed. The diagnostic criteria are splenic artery diameter > 4 mm or 150% of the hepatic artery, enlarged gastroduodenal artery, and sluggish hepatic artery flow.[35–37] No consensus treatment protocol is there in the literature, however, transcatheter proximal splenic artery embolization using coils is the most acceptable treatment followed in many institutions.[38,39] Other management options include splenic artery ligation and splenectomy. Redemonstration of normal hepatic artery flow with an RI value of less than 0.8 indicates successful management.

Conclusion

LT is the definite and final resort for decompensated chronic parenchymal liver disease. The imaging plays a pivotal role in the prompt diagnosis of various postoperative complications. USG is the initial modality of choice for screening the hepatic parenchyma and vasculature, where the alarming findings are reassessed with CT which is more accurate and has a high spatial resolution. Understanding potential posttransplant complications as well as the benefits and drawbacks of each imaging technique may help with early detection and prompt treatment.

Teaching Points

(1) Complications are divided into vascular and nonvascular complications which in turn are divided into biliary, infections, and immune response. Hepatic artery thrombosis is the most common vascular thrombosis.
(2) Hepatic artery thrombosis occurs usually between 2nd to 15th week. Untreated hepatic artery thrombosis will lead on to biliary ischemia and strictures with adverse outcomes. Most patients need surgical exploration or endovascular thrombectomy in the event of an early posttransplant HAT.
(3) Hepatic artery stenosis occurs within the first 3 months with median time of 100 days. PSV of more than 200 cm/s is highly suggestive whereas other indirect findings include increase in diastolic component with reduced resistance index to less than 0.55, a delayed acceleration peak (more than 80 ms) together producing the characteristic parvus et tardus pattern of flow.
(4) Biliary strictures are the most nonvascular complications which usually occur at the anastomotic site. It occurs due to intimal hyperplasia and perianastomotic site scarring. Nonanastomotic site strictures should raise the suspicion for hepatic artery thrombosis.
(5) Splenic artery steal syndrome is a rare complication that occurs in the immediate postoperative period.

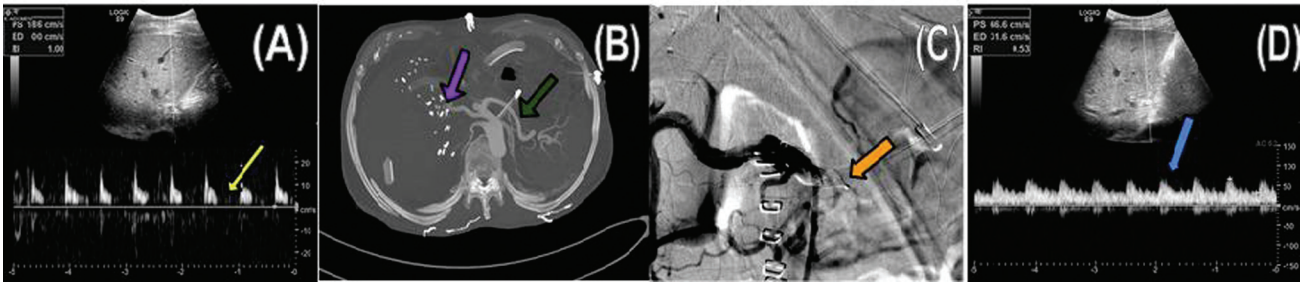

Fig. 12 A 45-year-old male patient with living donor liver transplantation (LDLT) had increased drain output on 3rd postoperative day (POD). Spectral Doppler image (**A**) shows increased resistive index (RI) values in the hepatic artery with absent diastolic flow (yellow arrow). Maximum intensity projection showed poor opacification of the hepatic artery (**B**, violet arrow) with hypertrophied splenic artery (green arrow) in the arterial phase. Digital subtraction angiography shows proximal splenic artery embolization (**C**, orange arrow). Immediate postprocedural Doppler image showed increase in hepatic artery flow (**D**, blue arrow).

Splenic artery diameter of more than 4 mm or more than 150% of hepatic artery enlarged gastroduodenal artery and sluggish hepatic artery. Transcatheter proximal splenic artery embolization (SAE) using coils is the most acceptable treatment followed in many institutions.

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Conflict of Interest
None declared.

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