Postoperative doppler evaluation of liver transplants

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Abstract

Doppler ultrasound plays an important role in the postoperative management of hepatic transplantation, by enabling early detection and treatment of various vascular complications. This article describes the normal Doppler findings following liver transplantation and reviews the imaging appearances of various vascular complications associated with it. The article also discusses transient waveform abnormalities, often seen on a post-transplant Doppler examination, and the importance of differentiating them from findings suggestive of ominous vascular complications.

Key words: Doppler; hepatic artery stenosis; hepatic artery thrombosis; liver transplant

Postoperative Doppler Evaluation of Liver Transplants

Liver transplantation is the only curative treatment for end-stage liver disease. The common indications for liver transplantation include cirrhosis secondary to alcoholic liver disease, hepatitis, hepatocellular carcinoma, non-alcoholic steatohepatitis, cholestatic and metabolic diseases, and fulminant hepatic failure. Over the last few decades, advancement in surgical techniques and perioperative management has greatly improved the outcomes of liver transplantation. Early detection of vascular complications by postoperative Doppler imaging has played a vital role in decreasing the incidence of graft failure. Here, we discuss the color Doppler imaging findings after liver transplantation for accurate and early detection of vascular complications.

Surgical Anatomy

The allograft for liver transplantation can be obtained either from a deceased donor or from a living donor after performing a partial hepatectomy. Deceased donor liver transplantation is by far the most prevalent method of transplantation in much of the western world. For example, approximately 300 living donor liver transplants (LDLT) are performed every year in the US compared to 6000 cadaveric donor transplants. In countries where cadaveric organ procurement is difficult, LDLT is more common than cadaveric donor transplants. Although the preoperative workup and technique of cadaveric donor and LDLT are different, the principles of postoperative Doppler imaging, performed to evaluate the vascular patency, are similar.

During liver transplantation surgery, inferior vena cava (IVC) anastomosis is followed by portal venous, arterial, and biliary anastomoses. For deceased donors, IVC anastomosis can be performed using the 'standard' or 'piggyback' techniques. In the standard technique, the recipient liver is removed along with the retrohepatic IVC. End-to-end anastomoses, between the donor and recipient IVCs, are performed above and below the allograft. In the piggyback technique, the donor hepatic venous confluence is anastomosed with the recipient IVC. Piggyback anastomosis

helps maintain hemodynamic stability during surgery. The piggyback technique is used for all LDLTs.

Portal venous and hepatic arterial anastomoses are performed after caval anastomosis. The portal venous anastomosis is an end-to-end anastomosis. For cadaveric donors, the donor hepatic artery is harvested at the level of the celiac axis with a patch of the aorta. The aortic patch is then anastomosed to the recipient hepatic artery near the gastroduodenal artery take-off. For living donors, the arterial anastomosis is to the right, left or proper hepatic artery.

Role of Doppler Ultrasound in Postoperative Evaluation

Vascular complications are a common cause for allograft failure after hepatic transplantation.[1] Early detection and treatment of vascular complications help reduce the incidence of graft failure. Doppler ultrasound is a noninvasive test that allows real time dynamic evaluation of the allograft vasculature and is extensively used in the postoperative evaluation of allografts.

Gray-scale ultrasound of the right upper quadrant precedes Doppler interrogation of the allograft vasculature. Along with evaluation of the hepatic parenchyma, attention is paid to the presence any perihepatic fluid collection. Perihepatic fluid collections may represent postoperative hematomas, seromas or bilomas. Superinfection of any fluid collection can cause abscess formation. Pneumobilia can be present in patients with bilioenteric anastomosis. Presence of biliary dilation may indicate development of biliary stenosis.

Doppler examination of the liver transplant involves interrogation of the main hepatic artery and its intrahepatic branches, main portal vein and its branches, hepatic veins, and the IVC.

The techniques to optimize color Doppler image acquisition in liver transplant patients are discussed in Table 1.

Normal Postoperative Doppler Appearance

The first post-transplant Doppler examination is often performed within the first 24 hours of surgery, with follow-up exams, as clinically indicated. The main, right, and left hepatic arteries are evaluated and should have a similar appearance. When examining the hepatic arterial waveform, attention is directed to the systolic upstroke, peak systolic velocity, and resistive index (RI) [Figure 1].

The normal hepatic arterial waveform has a rapid systolic upstroke. The systolic acceleration time (SAT) - the interval from end diastole to the first systolic peak - is a measure of the rapidity of the upstroke. Dodd *et al.*[2] have suggested that

a SAT greater than 80 ms is abnormal in a post-transplant patient. However, Paulson *et al.*[3] have shown that there is a significant variability in the measurement of SAT, a

Table 1: Optimizing color doppler image acquisition

Transducer selection

    Curved or sector transducer for liver transplant evaluation

    Highest frequency allowing adequate penetration chosen

    Focal zone at region of interest

Grayscale image optimization

    Image depth adjusted so that area of interest takes up nearly the entire image

    Grayscale gain optimized. Too low a gain causes hypoechoic thrombus to be missed and too high a gain results in spurious echoes, mimicking a thrombus

    Harmonic imaging, real-time compounding may be used to decrease image noise and increase anatomic detail

Color Doppler optimization

    Color Doppler angle less than 60 degrees achieved. Color detection will be poor if the vessel is perpendicular to the ultrasound waves

    Color gain optimized by increasing the gain till color speckles are seen in the soft tissues adjacent to the vessel and then decreasing it till these speckles disappear

    Color scale pulse repetition frequency is optimized by selecting the PRF, so that little or no aliasing is seen in the normal vessel. After color scale optimization, aliasing will suggest the presence of stenosis. Too low PRF causes a 'color bleed' and may result in a non-occlusive thrombus being overlooked. Too high a PRF will cause the color to not fill up the normal lumen

Spectral Doppler optimization

    Scale (PRF) and baseline optimized such that the waveform fills approximately two-thirds of the window. If the waveform overlaps the spectral window, aliasing is present and the PRF needs to be increased until the spectral waveform is contained within the spectral window

Additional points

    Color Doppler interrogation of cystic structures to exclude pseudoaneurysms

    Hepatic arterial kinks cause spuriously increased velocities and grayscale examination reveals the vascular tortuosity

    Power Doppler detects a very slow flow, and is used to verify when there is no flow seen on the color Doppler
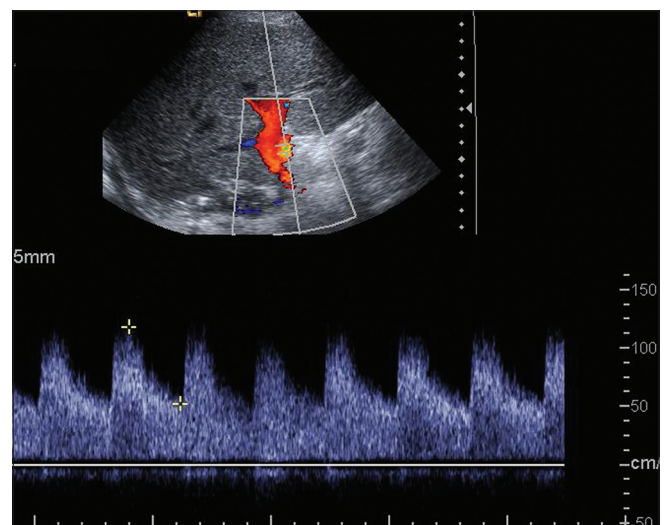
PRF: Pulse repetition frequency

Figure 1: Normal main hepatic arterial waveform. Doppler image of the main hepatic artery post liver transplant shows continuous antegrade flow with rapid systolic upstroke, RI of 0.60 and PSV of 110 cm/s

fact that needs to be kept in mind when interpreting small changes in its value.

The hepatic arterial resistive index (RI) is an important tool to assess the hepatic arterial waveform. RI is determined by the following formula: (Peak systolic velocity-end diastolic velocity)/Peak systolic velocity. The RI increases as the diastolic flow decreases in the hepatic artery and absent or reversed diastolic flow corresponds to an RI of 1.0. In a post-transplant patient, the normal hepatic arterial RI ranges from 0.55 to 0.80.[4,5]

The mean hepatic arterial peak systolic velocity (PSV) is 103 cm/s.[6] However, a wide variability in PSV may be noted in the immediate postoperative period in the absence of any hepatic arterial complication.

Variability in the Immediate Postoperative Period

On account of the hemodynamic changes associated with transplantation, the normal hepatic arterial waveform described earlier, is often not present in the immediate postoperative Doppler ultrasound. The hepatic arterial abnormalities seen in the immediate postoperative Doppler are often transient and resolve on follow-up scans.[7,8] The first postoperative Doppler serves as baseline for future Doppler examinations.

The most common transient hepatic arterial waveform abnormality seen in the immediate postoperative period is increased hepatic arterial RI, due to decreased diastolic flow. Increased RI (RI > 0.8, absent or even reversed diastolic flow) is seen in almost half the patients immediately after liver transplantation. This can involve the main hepatic artery as well as its intrahepatic branches. The RI usually normalizes within 7-15 days and is not associated with poor graft function.[8] This transient elevation of RI is likely secondary to allograft edema, increased cold ischemia time, increased portal flow or vessel spasm [Figure 2].[9]

A decrease in hepatic arterial RI (RI < 0.55) due to increased diastolic flow is a more ominous finding than increased RI, and is usually of concern for arterial complications. Decreased RI may occasionally be a transient finding on an immediate postoperative scan, which resolves in a few days. This is probably due to anastomotic edema in the postoperative period.

Vascular kinks due to vessel redundancy can lead to spuriously elevated values of peak systolic velocity. Correction of the angle of interrogation can help differentiate this from true stenosis. Anastomotic edema may also occasionally cause temporarily increased hepatic arterial velocity in the immediate postoperative scans.[6]
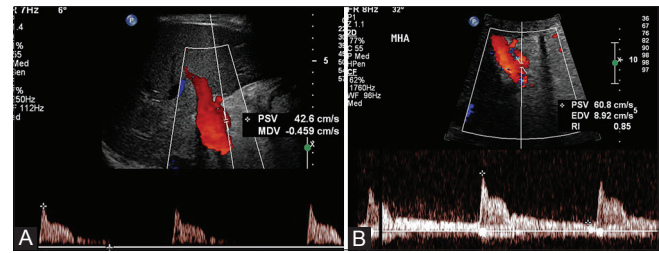

Figure 2 (A and B): (A) Transient increased hepatic arterial RI. Doppler ultrasound performed on the first postoperative day after liver transplantation shows absent diastolic flow (RI = 1) in the main hepatic artery (B) Transient increased hepatic arterial RI. Follow-up Doppler ultrasound performed after three days shows improvement of the waveform with increase in diastolic flow (RI 0.85). Increased RI is a common finding in the immediate postoperative period, which usually resolves on follow-up imaging

In view of the above mentioned variability in the immediate postoperative period, other than in cases of absent arterial flow, the diagnosis of a hepatic arterial complication is seldom established on the basis of a single immediate postoperative Doppler ultrasound. Arterial waveform abnormalities on the immediate postoperative scans should be followed and correlated with the patient's clinical findings including liver function tests. Transient waveform changes usually resolve in 7-15 days. Arterial complications are detected by persistence of abnormalities or further deterioration of the waveform, on follow-up Doppler studies.

An important point to note while interpreting post-transplant Doppler studies is that deterioration of a waveform on a follow-up study is highly suggestive of an arterial complication. The first postoperative Doppler serves as a baseline and any worsening of the hepatic arterial waveform on follow-up is always due to an arterial complication.

The reduction in portal venous resistance in the immediate post-transplant period can cause increased portal venous flow. This manifests as a high portal venous velocity, which gradually normalizes in the postoperative period, as the body adapts to the new hemodynamics. Compression of the portal vein by transient postoperative collections can also cause temporarily increased portal venous velocity, which normalizes as the postoperative fluid resolves. A transient increase in portal venous velocity should not be misdiagnosed as portal venous stenosis, which is a rather rare complication [Figure 3A and B]. Hepatic veins normally have a triphasic waveform, but monophasic or biphasic waveforms are commonly seen in the postoperative period, secondary to graft edema or compression by the adjacent fluid collection. This usually normalizes on follow-up studies.

Hepatic Arterial Complications

Hepatic arterial complications include hepatic arterial thrombosis (HAT), hepatic arterial stenosis (HAS), and

pseudoaneurysm formation. Hepatic arterial complications are particularly significant in post liver transplant patients. The biliary tree derives its blood supply solely from the hepatic arteries after transplantation. Any hepatic arterial complication can cause biliary ischemia, resulting in biliary strictures, necrosis, abscesses, and allograft failure. Biliary strictures secondary to arterial complications are usually non-anastomotic and typically involve the hilum, but can be multifocal and intrahepatic. Biliary changes usually take time to develop and are usually not evident on ultrasound in the early stages. The usual timeline of presentation of various vascular complications after liver transplantation is provided in Table 2.

Hepatic Arterial Thrombosis

HAT is the most common and devastating complication after liver transplantation, which results in high mortality.[5,10] Early HAT occurs in the first few weeks and presents with rapid clinical deterioration.[11] Thrombosis occurring after the first month has a more insidious clinical course. HAT requires re-transplantation in majority of the cases.[12] Surgical revascularization may be attempted when the diagnosis is established early in the postoperative course.

Ultrasound is diagnostic in 92% of cases of HAT.[13] On Doppler ultrasound, HAT is diagnosed by the absence of flow in the hepatic arteries. As the allograft edema in the postoperative period can make visualization of the hepatic arteries difficult, it is important to ensure that the gain settings are optimized and a thorough search for hepatic arteries is performed to prevent a false positive diagnosis. A high resistance waveform (RI = 1) may be obtained in the hepatic artery if it is sampled proximal to the site of the thrombosis. On account of the catastrophic nature of this complication, computed tomography (CT) or digital subtraction angiography (DSA) angiography is usually performed to confirm the diagnosis.
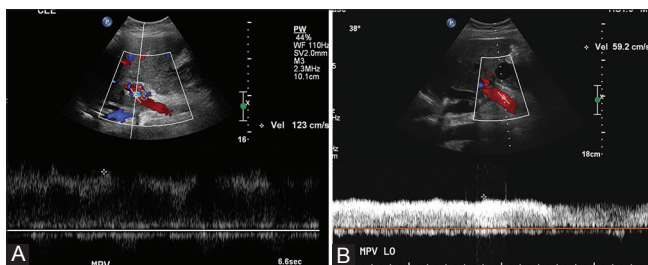
Hepatic arterial collaterals can develop after HAT, particularly in the late cases.[14] These collaterals can cause tardus parvus waveforms in the right and left hepatic arteries [Figure 4A and B]. Demonstration of such waveforms in the downstream hepatic arteries must not be considered evidence of patency of the more proximal hepatic artery. The tardus parvus waveform does not exclude HAT, but rather indicates the presence of either upstream stenosis or thrombosis.

Hepatic Arterial Stenosis

HAS can cause complications similar to HAT, but it has a more insidious course with symptoms developing over days to weeks after transplantation. HAS usually occurs at the anastomosis, but can occur anywhere in the graft artery. Patients usually present with abnormal liver enzymes or non-anastomotic biliary strictures.[15] Timely detection of HAS on a postoperative Doppler is extremely important. Unlike HAT, this condition is treatable by percutaneous angioplasty and stenting.[16,17] HAS itself causes graft ischemia and untreated stenosis can progress to the even more devastating HAT.

On Doppler ultrasound, HAS can be diagnosed by direct visualization of the stenotic segment or by identification of the characteristic downstream changes in the distal arteries. The stenotic segment is identified on color Doppler as an area of turbulence and aliasing. The PSV at the stenotic segment is usually elevated, greater than 200 cm/s.[2]

Table 2: Time of presentation of vascular complications after liver transplants

Complication	Time of presentation after transplantation
Hepatic artery thrombosis	First two weeks (Late HAT occurs, but is less devastating)
Hepatic artery stenosis	Few weeks to several months
Portal venous thrombosis	Majority within one month
Portal venous stenosis	Usually late (>6 months)
Hepatic venous stenosis	Usually late (>6 months)

HAT: Hepatic artery thrombosis

Figure 3 (A and B): (A) Transient increase in portal venous velocity. Doppler ultrasound performed on the first postoperative day after liver transplantation shows increased main portal venous velocity (123 cm/s) (B) Transient increase in portal venous velocity. Follow-up Doppler ultrasound performed five days later shows decrease in the main portal venous velocity (59 cm/s). Increased portal venous velocity is a common transient finding after liver transplantation, which normalizes on follow-up imaging
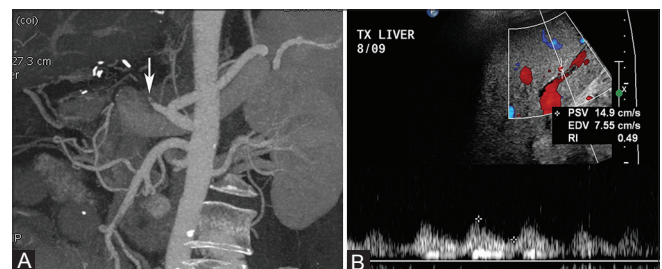

Figure 4 (A and B): (A) Hepatic artery thrombosis. CT angiogram performed six months after liver transplantation shows thrombosis of the main hepatic artery (arrow) (B) Hepatic artery thrombosis. Doppler ultrasound of the same patient shows tardus parvus waveform (delayed upstroke with increased diastolic flow) in the left hepatic artery, due to formation of collaterals following hepatic arterial thrombosis. Detection of flow in the left hepatic artery does not exclude HAT

Secondary changes seen downstream to the stenosis are also very useful in establishing the diagnosis of HAS. There is decreased resistance of the arterial tree downstream to the stenosis. This results in an increased diastolic flow and a corresponding decrease in RI, to less than 0.55. The usually rapid hepatic arterial systolic upstroke gets delayed (systolic acceleration time greater than 80 ms). The sensitivity and specificity of the secondary findings range from 73-83% and 60-73%.[2,4] A tardus parvus waveform may be seen distal to the stenotic segment.

Even as direct demonstration of the stenotic segment clinches the diagnosis of HAS, the stenotic segment is usually short, and direct visualization may not be possible. The presence of downstream changes must prompt a close scrutiny of the proximal hepatic artery for any stenotic segment. If the stenotic segment cannot be demonstrated, one must keep in mind that collateralization after HAT can mimic the downstream changes of HAS. CT or DSA angiography can be performed to confirm the diagnosis. Usually DSA is preferred, as therapeutic angioplasty/stenting can be performed in the same setting [Figure 5A-D].

Hepatic Artery Pseudoaneurysm

Hepatic arterial pseudoaneurysm formation is a very rare complication. Pseudoaneurysm formation can be due to infection or can be iatrogenic, secondary to a biopsy or angioplasty. Doppler interrogation of any cystic structure encountered near the hepatic hilum is performed to exclude a pseudoaneurysm. On Doppler ultrasound, a

Figure 5 (A-D): (A) Hepatic artery stenosis. Doppler ultrasound of the right hepatic artery shows increased diastolic flow with an abnormally low RI of 0.48 (B) Hepatic artery stenosis. Doppler ultrasound of the proximal main hepatic artery near the anastomosis shows focal area of aliasing and increased peak systolic velocity (>350 cm/s) consistent with focal stenosis (C) Hepatic artery stenosis. CT angiogram shows a focal severely stenotic segment in the main hepatic artery (arrow) (D) Hepatic artery stenosis. Percutaneous stenting of the stenotic segment was performed. DSA angiogram image shows a patent stent (arrowheads) across the previously stenotic segment

pseudoaneurysm is identified as a cystic structure along the hepatic artery, with a disorganized flow within it.

Splenic Artery Steal Syndrome

In some patients with pre-existing portal hypertension and splenomegaly, following liver transplantation, the hypertrophied splenic artery may shift the blood flow away from the liver toward the spleen, resulting in hepatic hypoperfusion. This is known as the splenic artery steal syndrome. On Doppler, this manifests as a high-resistance waveform in the main, left, and right hepatic arteries. Increased portal vein velocity and hyperdynamic splenic arterial flow are present. The imaging findings are not specific, as these may be present in normal patients in the immediate postoperative period and usually normalize on follow-up imaging. However, if these findings persist on follow-up examinations and patients have graft dysfunction, the presence of a splenic artery steal needs to be considered. Angiography is performed to confirm the diagnosis and the condition is treated by splenic arterial embolization.[18]

Portal Venous Complications

After transplantation, the normal portal vein has antegrade flow. The portal venous velocity is variable and tends to decrease on serial examinations after transplantation. Portal vein thrombosis is the most common portal venous complication after surgery.[19] The thrombus is identified as either anechoic or echogenic material within the portal vein, on gray-scale ultrasound, and no flow is identified on Doppler interrogation [Figure 6]. The portal vein should be interrogated with power Doppler so that the slow flow is not mistaken as thrombosis. In a majority of the patients, portal venous thrombosis is diagnosed within one month of transplantation. It may present with signs of portal hypertension or nonspecific abnormalities of liver function tests. The treatment options in acute cases include catheter-guided thrombolysis and thrombectomy, as well as, anastomotic revision and re-transplantation.

Portal vein stenosis is another complication occasionally seen after transplantation. Clinically significant portal vein stenosis after transplantation is rare and usually occurs in the pediatric and living donor population, due to the small graft vein size.[20] Portal vein stenosis presenting within six months is likely due to technical reasons and a more delayed presentation is due to neointimal hyperplasia.[21] The presentation is similar to portal vein thrombosis with portal hypertension or hepatic failure. Aliasing and a three-to-four fold increase in velocity is noted at the stenotic segment of the portal vein.[19,22] A venogram, with measurement of the pressure gradient across the stenotic segment can be performed to assess the functional significance of the narrowing [Figure 7A-C]. Portal venous stenosis is
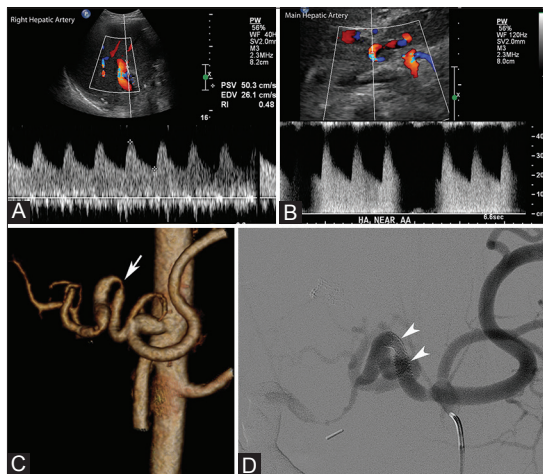
treated with catheter-guided angioplasty, and if necessary stenting.[23] Portal vein thrombosis can be a complication of portal vein angioplasty or stenting.[21]

Hepatic Venous and Inferior Vena Cava Complications

Thrombosis and stenosis are two rare complications of the IVC and hepatic veins, after hepatic transplantation. The
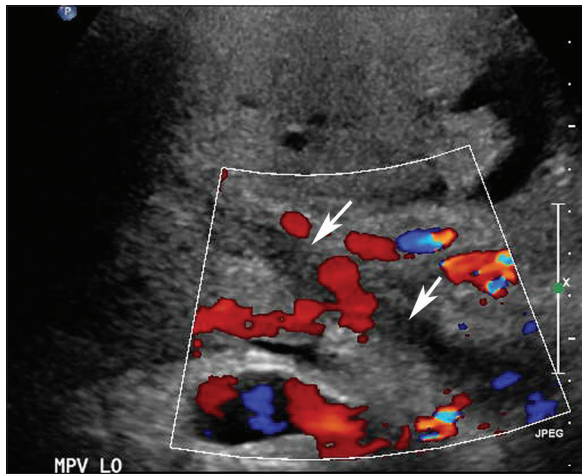

Figure 6: Portal vein thrombosis. Doppler ultrasound of the main portal vein following liver transplant shows echogenic thrombus within the main portal vein with lack of color-filling, consistent with portal vein thrombosis (arrows)
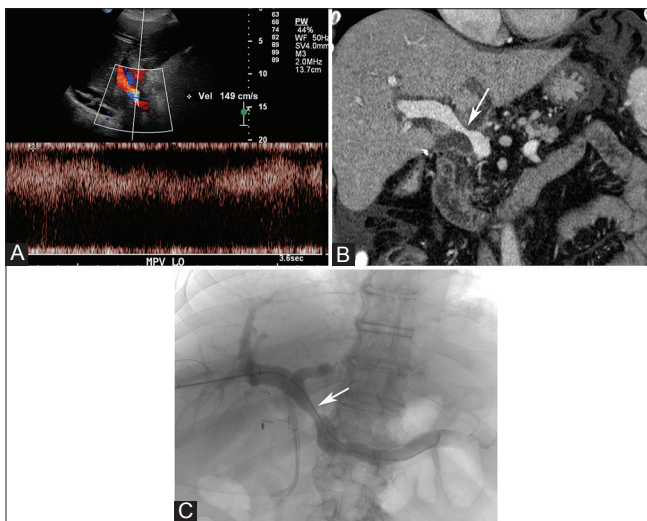

Figure 7 (A-C): (A) Portal vein stenosis. Doppler ultrasound of the main portal vein following liver transplant shows a focal area of aliasing and increased velocity (149 cm/s). The adjacent segment of the portal vein has a velocity of 40 cm/s. This was considered suspicious for main portal vein stenosis (B) Portal vein stenosis. CT scan confirmed moderately severe narrowing of the main portal vein (arrow) (C) Portal vein stenosis. Patient subsequently underwent a portal venogram with pressure gradient measurement. Although the portal vein narrowing was re-demonstrated (arrow), there was a negligible pressure gradient across the stenotic segment. There was no evidence of portal hypertension. The stenosis was deemed not to be functionally significant and no therapeutic intervention was performed

venous complications tend to occur at the anastomosis and knowledge of the surgical anatomy is useful. The incidence of hepatic venous outflow stenosis is higher in living donors and split graft liver pediatric grafts than in whole liver grafts, likely due to the size mismatch and small anastomosis.[24] Most hepatic venous stenosis present late, many months to years after transplantation.[25]

Normal hepatic veins have a triphasic waveform due to transmission of cardiac pulsations from the heart. Hepatic venous stenosis prevents transmission of the cardiac pulsations, resulting in loss of triphasicity.[26,27] However, loss of triphasicity is a very nonspecific finding and is often seen in normal postoperative patients. Therefore, while the presence of hepatic venous triphasicity can be used to exclude hepatic venous stenosis, loss of triphasicity does not imply the presence of a hepatic venous complication. Clinically significant hepatic venous outflow stenosis can be treated with venous angioplasty and stenosis.[25]

Overall, portal venous and hepatic venous outflow stenoses are rare. Ultrasound findings suggestive of venous stenosis should be correlated with the patients' clinical presentation [Figure 8].

Conclusion

Doppler ultrasound is the modality of choice for evaluating post liver transplant patients for vascular complications. Awareness of the normal postoperative Doppler findings and timely identification of various
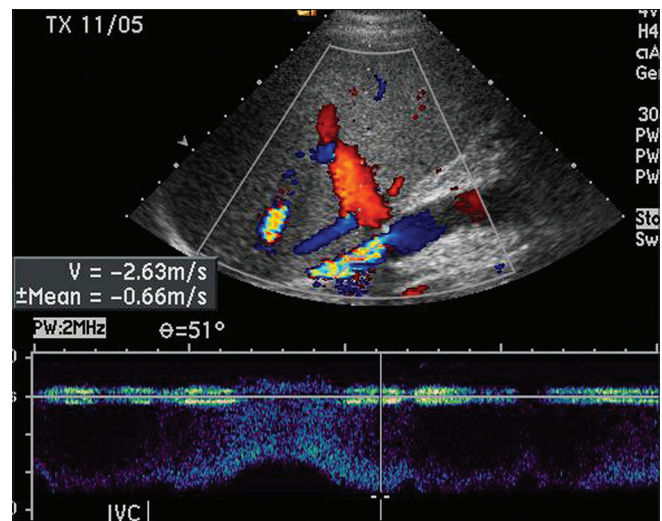

Figure 8: IVC stenosis. Doppler ultrasound performed seven months after a cadaveric donor liver transplantation demonstrates aliasing and increased velocity (upto 263 cm/s) at the caudal IVC anastomosis, consistent with stenosis. IVC anastomoses had been performed using the standard technique, in which two end-to-end anastomoses were performed. Although the IVC was stenotic on imaging at the caudal anastomosis, the patient remained asymptomatic and no intervention was carried out

vascular complications is essential for improving outcome of liver transplantation.

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