Review Article

The Role of Ultrasonography in Portal Hypertension

Nizar A. Al-Nakshabandi

Department of Radiology, Faculty of Medicine, King Khalid University Hospital, King Saud University, Riyadh, Saudi Arabia

Address:
Nizar A. Al-Nakshabandi, Department of Radiology, Faculty of Medicine, King Khalid University Hospital, King Saud University, Riyadh, Saudi Arabia.
E-mail: nizarn@ksu.edu.sa

ABSTRACT

Portal hypertension is a commonly encountered clinical condition with multiple causes and several squeal. Ultrasound is an accurate non-invasive means of assessing its aetiology, severity and complications. I will review the role of ultrasonography in portal hypertension. The ultrasonic features that help identify its aetiology will be discussed as will the criteria that allow an assessment of disease severity and its complications.

Key Words: Portal hypertension, ultrasound

Received 07.06.2006, Accepted 20.07.2006
The Saudi Journal of Gastroenterology 2006 12(3):111-117

Portal hypertension is a common clinical syndrome, characterised by an increase in portal venous pressure. It is variously defined as a wedged hepatic vein pressure or direct portal vein pressure of more than 5 mm Hg greater than inferior vena cava pressure, a splenic vein pressure of greater than 15 mm Hg or portal vein pressure at surgery of more than 30 cm H20. However, actual direct measurements of portal pressures are obtained in only a small minority of patients. Consequently, non-invasive imaging modalities and in particular ultrasound, play a crucial role in the diagnosis and management of portal hypertension.

The goals of ultrasonic assessment should be threefold, namely to:

- Make the diagnosis
- Establish the cause
- Evaluate the risk of complications

Making the diagnosis

Multiple gray scale and doppler findings have been proposed as markers of underlying portal hypertension and the merits and limitations of these are discussed below.

Portal vein (PV)

Traditionally, enlargement of the portal vein has been considered a sign of portal hypertension. However, studies have shown that threshold PV diameters of greater than 13 or 15 mm have a sensitivity for diagnosing portal hypertension of only 40 and 12.5 % respectively.[1,2] In fact, it has been noted angiographically that the diameter of the portal vein does not increase with the porto-hepatic venous pressure gradient and may even tend to decrease depending on the severity of the hypertension.[3] Furthermore, with the development of reversed portal vein flow (hepatofugal flow) and/or portosystemic shunts the portal vein calibre will decrease. Although the absolute size of the portal vein may not be a reliable indicator of portal hypertension, its relative change in size with respiration is a more sensitive, if somewhat rarely assessed, finding. An increase of less than 20% in the diameter of the PV with deep inspiration indicates portal hypertension with a sensitivity of 80% and specificity of 100%.[4]

Normally, portal blood flows towards the liver, hepatopetal flow, [Figure 1a] throughout systole and diastole with only mild superimposed respiratory phasicity and cardiac periodicity. In most cases of portal hypertension the flow is still hepatopetal but spectral doppler may demonstrate loss of respiratory phasicity and more pronounced cardiac periodicity which can progress to an absence of end-diastolic flow, arterialized flow or bidirectional "to-and-fro" flow. Rarely, with increasing hepatic parenchymal scarring and fibrosis, the pathway of least resistance for the hepatic arterial inflow becomes the portal vein resulting in reversed portal vein flow. Studies have demonstrated that it is possible for patients with portal hypertension to have hepatofugal flow on one day and normal hepatopetal flow on another.[5]

In normal patients the mean PV flow rate is 13 to 23 cm/sec but in patients with portal hypertension, the mean PV velocity may vary depending on the presence and location of spontaneous shunts. The velocity tends to increase in the presence of a patent paraumbilical vein and decrease in the presence of splenorenal collaterals.

The "congestion index" is the ratio of PV cross-sectional area (cm 2) to mean PV flow velocity (cm/sec), thereby taking into

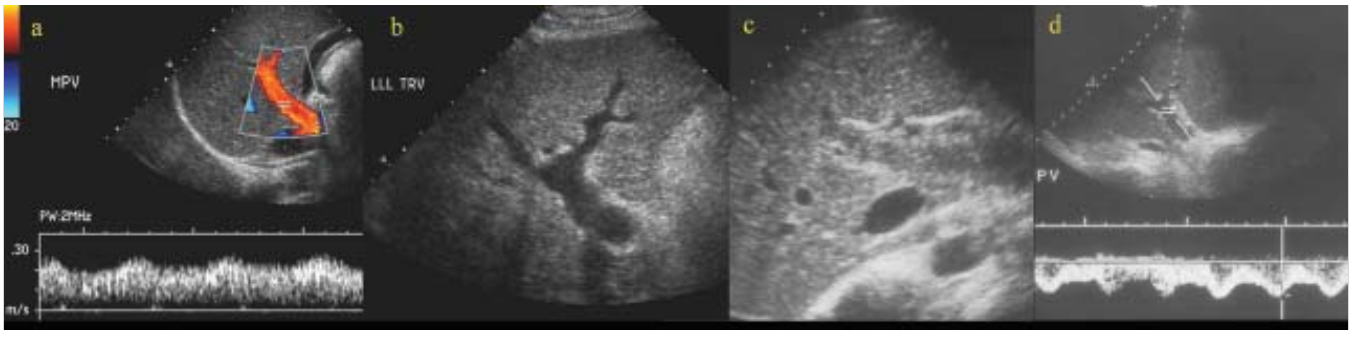

Figure 1: CDI in 33 year old patient (a). Enlarged (17 mm) portal vein in 67 year old with portal hypertension (b). Completely occluded PV lumen in 45 year old cirrhotic patient on gray scale imaging (c). Reversed flow seen on spectral doppler (d) in 56 year old cirrhotic, note the arterialised waveform reflecting hepatic artery-portovenous shunting.

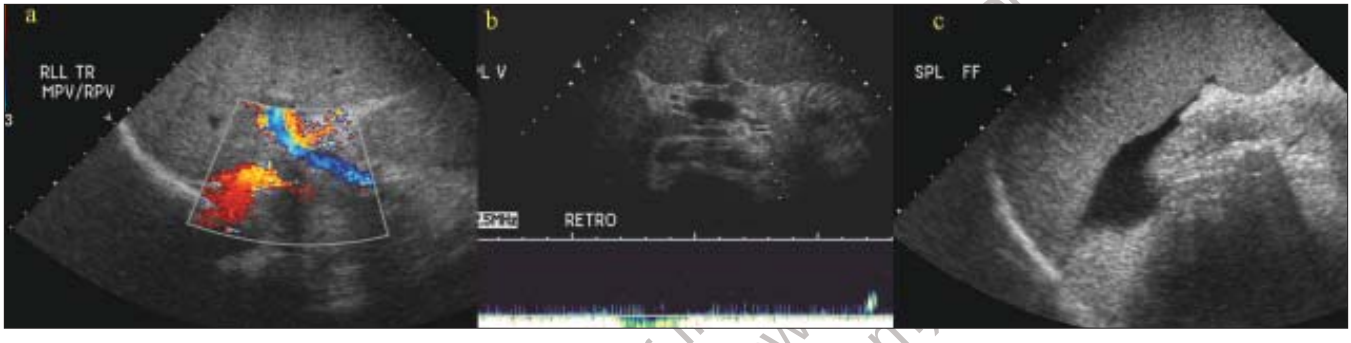

Figure 2: Reversed portal and splenic vein flow. 37 year old with cirrhosis and evidence of retrograde (hepatofugal) flow in portal vein on CDI (a) and splenic vein on spectral doppler (b) as well as splenomegaly and ascites (c).

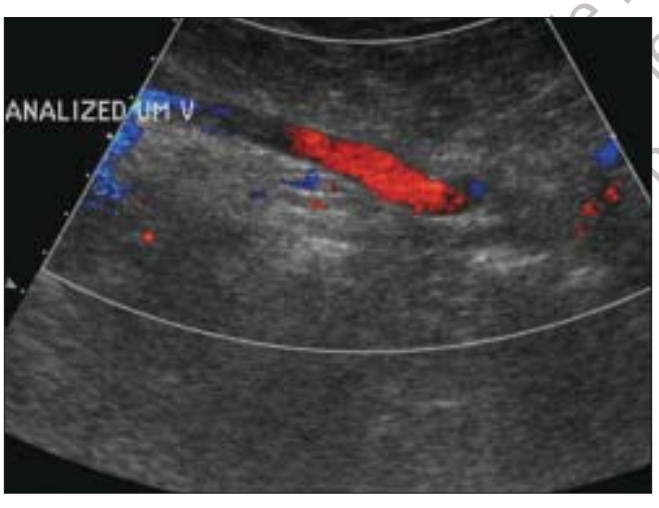

Figure 3: Recanalised paraumbilical veins. Cirrhotic patient with recanalised paraumbilical veins and ascites

account portal vein dilatation and decreased flow velocity, two physiological changes associated with portal hypertension. In normal subjects this ratio is approximately 0.07[6] and a value above 0.1 suggests the diagnosis of portal hypertension with a 95% sensitivity and specificity[7] [Figure 1].

Hepatic artery (HA)

In normal subjects the HA possesses forward flow in diastole due to a low-resistance peripheral vascular bed and the normal resistance index (RI) ranges from 0.5 to 0.7. Although, the changes in hepatic artery resistance have not been extensively investigated most studies have demonstrated that the arterial resistance index increases in cirrhosis. This finding is complicated somewhat by the observation in some studies that when portal vein thrombosis occurs the resistance index may actually fall below 0.5.[8] On their own the alterations in resistance in the HA are not sufficiently specific or sensitive to allow a diagnosis of portal hypertension.

Hepatic veins (HVs)

The Doppler signal of the normal hepatic veins shows that net flow is towards the right atrium but that there are wide variations in flow velocity and direction. These variations are the result of transmitted cardiac pulsations and reflux of blood from the right atrium into the veins during systole. Apart from the changes of hepatic vein thrombosis which is discussed below, two main types of alteration of hepatic vein flow profile can be observed in portal hypertension, especially when due to cirrhosis. The first is regional flow acceleration resulting from focal compression by regenerative nodules. The second is dampening of the normal pulsatile flow pattern secondary to non-compliance caused by fibrous tissue. In one study of cirrhotic patients a normal flow profile was found in 50% of cases, a dampened flow profile in 30% and completely flattened flow in 20%.[9]

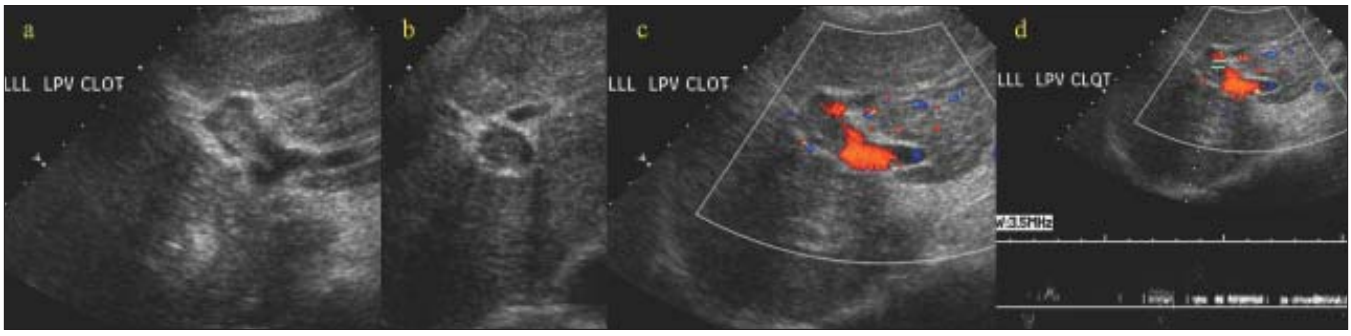

Figure 4: Non-occlusive portal vein thrombus. 49 year old male with hepatitis B and long-standing cirrhotic portal hypertension complicated by multicentric hepatoma. There is non-occlusive thrombus in the left portal vein which is well seen on these gray scale ((a) and (b)) and CDI ((c) and (d)) images. It was felt to be benign in view of the absence of any pulsatile flow on spectral doppler examination and it was distant from any primary tumour mass.

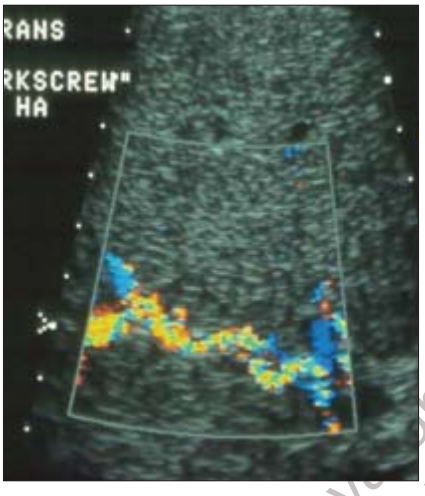

Figure 5: CDI demonstrating tortuous "corkscrew"-like intrahepatic course of hepatic artery branch, which is typical of cirrhosis

Splanchnic veins

Enlarged splanchnic veins (e.g., a superior mesenteric vein (SMV) and splenic vein (SV) diameter of more than 1 cm) are suggestive of portal hypertension. Several studies have shown that the diameters of the SMV and SV are statistically different in control subjects and patients with cirrhosis with the expiration measurements being the most discriminating.[10]

A lack of calibre variation of the splanchnic vessels during breathing was originally thought to be highly sensitive (80%) and specific for portal hypertension[11] and some workers have suggested that an increase in diameter during inspiration of less than or equal to 10% is a marker of portal hypertension.[12] However, these findings were not confirmed by another study which found that absent calibre variation with respiration had a sensitivity of only 42% for portal hypertension.[2]

Reversed flow may be detected in the SMV or SV at Doppler sonography [Figure 2], however this is a relatively rare finding, seen in less than 5% of cases of portal hypertension. Although reversed splanchnic vein flow is not related to the aetiology of portal hypertension, it is seen more frequently in patients classified as Child's B and C than in those classified as Child's A [Figure 2].

Splanchnic arteries

Several studies have shown that blood flow increases and the resistance index falls in the superior mesenteric and splenic arteries in the setting of portal hypertension. After a meal splanchnic artery RIs are reduced in all patients but the reduction is less in patients with cirrhosis. However, among

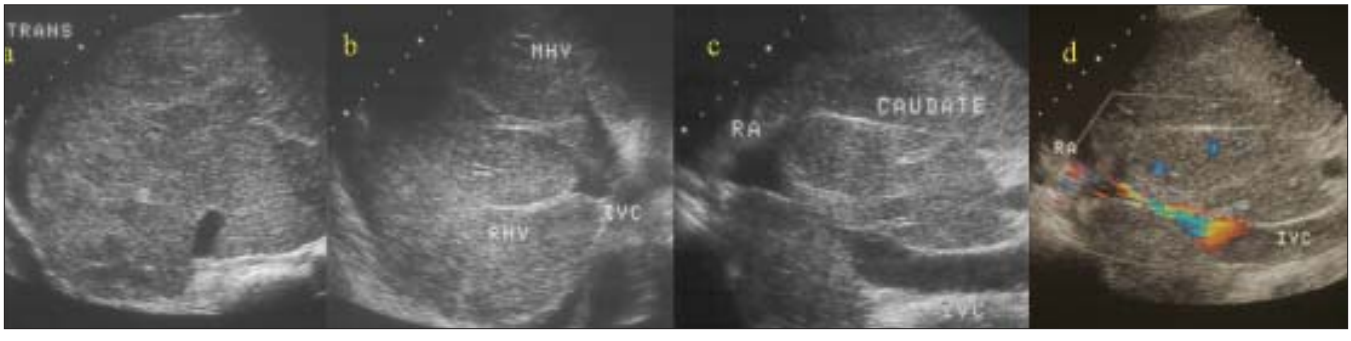

Figure 6: Budd-Chiari syndrome. Heterogeneous liver parenchyma with ascites and hypertrophied caudate lobe (a). The hepatic veins are thrombosed (b) and the enlarged caudate lobe compresses the IVC on gray scale

patients with portal hypertension there is no correlation between the magnitude of the post-prandial decrease of the RI and the severity of the underlying disease.[13]

Portosystemic collateral vessels

Portosystemic collaterals form when the resistance to blood flow in the portal vessels exceeds the resistance to flow in the small communicating channels between the portal and systemic circulations. Several major sites of portosystemic venous collaterals occur with the first three being the most commonly identified on ultrasound.

Gastro-oesophageal Junction: Collaterals in this region run between the coronary and short gastric veins and the systemic oesophageal veins. They are best visualised ultrasonically through the left lobe of the liver. These vessels are of particular importance as they may lead to life threatening variceal haemorrhage. The normal coronary vein measures up to 6mm in diameter and has hepatopetal flow, whereas in patients with portal hypertension, dilatation above this size is seen in 26% of cases and hepatofugal flow is seen in 78%.[14] One interpretation of these findings is that dilatation of the coronary vein does not occur in the majority of patients with portal hypertension.

Paraumbilical vein: This collateral originates from the left portal vein and connects with the superior and inferior epigastric veins, of the systemic circulation, around the umbilicus. It runs in the recanalised ligamentum teres of the falciform ligament [Figure 3] as a tubular structure measuring more than 3 mm. Slow flow (up to 5 cm/sec) in either direction may be detected in the ligamentum teres of normal subjects but flow does not extend anterior to the liver surface. Hepatofugal venous flow in the ligamentum teres with a velocity greater than 5 cm/sec or visualised anterior to the liver's surface is highly specific (100% in one series) for the presence of portal hypertension.[15] As epigastric vessel collaterals run just deep to the rectus muscles they are not apparent clinically but are easily identified by colour doppler.

Splenorenal and gastrorenal: Tortuous vessels may be seen in the hilar region of the spleen and left kidney representing collaterals between the splenic, coronary and short gastric veins and the systemic left adrenal or renal veins. These are best visualised using the enlarged spleen as an ultrasonic window. Their existence may be inferred by asymmetrical enlargement of the left renal vein, although this can also be seen with renal arteriovenous fistula and renal tumours.

Intestinal: These collateral pathways occur in areas where the gastrointestinal tract is retroperitoneal (e.g., ascending and descending colon and duodenum) and they connect pancreaticoduodenal, retroperitoneal and omental veins with renal, phrenic and lumbar veins. The ease with which these vessels are identified on ultrasound depends to a large degree on the amount of air in the bowel at the time of the study as well as the size of the collateral vessels.

Haemorrhoids: Collaterals in this region connect the inferior mesenteric veins, via the superior rectal veins, with the middle and inferior rectal veins of the systemic circulation. Usually, conventional transabdominal sonography cannot detect rectal/ pararectal varices but these can be seen using transvaginal scanning.

Gallbladder (GB): These varices are sometimes observed in portal hypertension and they reflect shunting of blood from the cystic vein to the anterior abdominal wall or to patent PV branches in the liver. On ultrasound they appear as cystic or tubular areas in the GB wall. Colour doppler is useful to show flow and differentiate these collaterals from Rokitansky-Aschoff sinuses of hyperplastic cholecystosis.

Other less common portosystemic pathways include the the iliolumbar, gonadal and ascending retrosternal veins. It is worth noting that almost any vein in the abdomen may serve as a potential collateral to the systemic circulation.

Detection of abnormal collateral vessels appears to be one of the most sensitive (70-83%) and specific sonographic signs for the diagnosis of portal hypertension.[2] The more severe the portal hypertension the higher the number of portosystemic pathways. There appears to be no relationship between the collateral pathway location and the cause of portal hypertension except for the paraumbilical vein which is only observed in patients with sinusoidal or post-sinusoidal subtypes of portal hypertension.[16] On doppler sonography collateral vessels demonstrate continuous flow similar to that of the PV. Unfortunately, collaterals can be missed by doppler ultrasound due to obesity or bowel gas.

Splenomegaly

Splenomegaly (>12 cm in longest axis) is often seen in portal hypertension. It is usually only mild to moderate in degree and may be the only evidence of elevated portal pressures. Conversely, the absence of splenomegaly does not rule out portal hypertension. The presence in the splenic parenchyma of small echogenic parallel lines (termed "reflective channels") containing blood flow on colour doppler, has been proposed as a means of differentiating splenomegaly due to portal hypertension from that due to other causes.[17] These structures are felt to reflect dilated sinusoidal veins which have an increased amount of collagen in their walls- a process known to occur in portal hypertension. This observation has a sensitivity, specificity and accuracy for predicting portal hypertension of 0.85, 0.77 and 0.80 respectively.[17] The only other ultrasonic finding that would suggest that splenomegaly is due to portal hypertension is siderotic gamma-gandy bodies

which are present in approximately 13% of cases and on ultrasound appear as multiple hyperechoic foci.

Ascites

This is a non-specific finding but is frequently seen in portal hypertension.

Establishing the cause

Portal hypertension can result from a variety of insults to the hepatobiliary system and these can be divided into pre-sinusoidal (extra- and intra-hepatic), sinusoidal and post-sinusoidal sub-types. In North America and Europe, 90% of cases of portal hypertension are due to cirrhosis whilst in Asia and South America, non-cirrhotic portal fibrosis, hepato-splenic schistosomiasis and extrahepatic portal vein thrombosis are much more common. Although it is not always possible on ultrasound to establish the exact aetiology of portal hypertension it may be possible to determine whether it is pre-sinusoidal, sinusoidal or post-sinusoidal.

Pre-sinusoidal extrahepatic

Causes: Portal or splenic vein thrombosis. This sub-type should be suspected if there are secondary signs of portal hypertension (e.g. splenomegaly, ascites and portosystemic collaterals) but the liver is normal in appearance ultrasonically.

Portal vein thrombosis: This can be seen in children secondary to umbilical vein catheterization or sepsis and in adults due to pancreatitis, pancreatic carcinoma, trauma, sepsis, hepatocellular carcinoma, cirrhosis, portacaval shunts and hypercoaguable states. It can occur at any point along the course of the vein. The sonographic findings of PV thrombosis include echogenic thrombus within the lumen of the vein [Figure 4], expansion of the calibre of the vein, absent doppler signal and carvernous transformation. Acute thrombus may appear relatively anechoic and can be overlooked unless colour Doppler interrogation is performed. Doppler is also useful in distinguishing between benign and malignant PV thrombi. Both kinds of thrombi may demonstrate continuous blood flow but pulsatile flow is 95% specific for the diagnosis of malignant thrombus.[18] The sensitivity is only 60% as many malignant thrombi are hypovascular. Other features suggesting malignant portal vein thrombus are a portal vein calibre of greater than or equal to 23 mm and evidence of thrombus continuity with an extraluminal mass.[19] Cavernous transformation refers to the development of numerous worm-like periportal collateral vessels at the porta hepatis after chronic PV thrombosis. It requires up to 12 months to develop and is therefore more likely to be seen with benign causes of portal hypertension. As the cause is usually pre-sinusoidal, flow within the collateral vessels is hepatopetal, unlike the hepatofugal flow seen in cirrhosis. It is seldom reliably visualised on gray-scale imaging alone because of the small vessel size but colour doppler reveals a web of numerous serpiginous small veins which on spectral doppler demonstrate hepatopetal portal flow.

Pre-sinusoidal Intrahepatic

Causes: Diseases affecting portal tracts (notably primary biliary cirrhosis, schistosomiasis, malaria, sarcoid, congenital hepatic fibrosis and secondary to hepatotoxins such as polyvinyl chloride and methotrexate).

Primary or secondary biliary cirrhosis: Primary biliary cirrhosis is a disease of unknown cause in which intra-hepatic bile ducts are progressively destroyed. Secondary biliary cirrhosis is a consequence of chronic intrahepatic bile duct obstruction. These two disease processes are mostly diagnosed on clinical, immunological, cholangiographic and histological findings.

Hepatosplenic schistosomiasis: This results from infection with *Schistosoma Mansoni* and occurs predominantly in South America, the Middle East and Africa. Portal hypertension is observed in 5-10% of those infected. The adult *S. Mansoni* worms live in the small mesenteric veins and release ova, some of which pass up the portal vein into the liver where they elicit an intense periportal granulomatous and fibrotic reaction. This process leads to peripheral portal venous occlusion and ultimately to presinusoidal portal hypertension. Splenomegaly is seen in all, GB thickening in over 80% and splenic and portal vein enlargement in over half of patients. Different grades of liver involvement have been proposed on ultrasound. In grade 1 disease there are hyperechoic bands accompanying the portal vein which are most readily visible in the area of the portal vein bifurcation and GB neck. In grade 2, the hyperechoic bands extend from the portal bifurcation along major portal vein branches towards the liver surface. Grade 3 includes all of the features of grades 1 and 2. The significance of this grading system is as a predictor of complications. In one study[20] none of the patients with grade 1 disease had a history of GI bleeding whilst a significant proportion of patients with grades 2 and 3 disease had already bled from oesophageal varices. Schistosoma japonicum can also involve the liver and is characterised by marked periportal and pericapsular calcification which is induced by the dead schistosoma eggs.

Congenital hepatic fibrosis: Portal hypertension is common in this condition which is caused by broad densely collagenous fibrous bands surrounding otherwise normal hepatic lobules and compressing the portal vein radicles. It can be associated with Caroli's disease. Ultrasound findings include; atrophy of the right lobe as well as secondary signs of portal hypertension.

Sinusoidal

Causes: Parenchymal liver disease (cirrhosis of whatever aetiology, is far and away the commonest cause, accounting for over 90% of cases, in the western hemisphere but it can

also result from extensive liver metastases).

Cirrhosis: This is characterised by the presence of parenchymal fibrosis and regenerating nodules and at ultrasonography the liver has a coarsened and heterogeneous echo pattern. There is usually an increase in parenchymal echogenicity, increased sound attenuation, tortuosity of intrahepatic vessels and nodularity of the liver surface. Whilst parenchymal heterogeneity and coarsening with changes in sound attenuation are not specific, surface nodularity is a more specific sonographic sign of cirrhosis. This is more easily identified with high frequency ultrasound probes or when ascites is present. As well as the parenchymal changes, the liver morphology is frequently altered because of atrophy of the right lobe (segments 5, 6, 7 and 8) and medial segment of the left lobe (4), with hypertrophy of the lateral segment of the left lobe (2 and 3) and caudate lobe (1) [Figure 5]. These changes in morphology may be assessed by calculating the size of the right lobe in comparison with that of the caudate lobe or by measuring the size of segment 4. When this latter measurement extends from the left wall of the gallbladder to the left portal vein where it gives rise to the segment 4 branch, a value of less than 30mm is a highly specific sign of a cirrhotic liver, whatever the cause.[21]

Post-sinusoidal

Causes: Budd Chiari syndrome, hepatic, veno-occlusive disease and cardiac insufficiency

Budd-Chiari syndrome: This is a relatively rare syndrome characterised by hepatic venous outflow obstruction which usually results from thrombosis of the main hepatic veins. It is frequently due to hypercoaguable states but can also result from tumour thrombosis and obstructing membranes. Ultrasound can play a crucial role in demonstrating both direct and indirect features of hepatic venous outflow obstruction. Direct features include; partial or complete inability to see the hepatic veins, enlargement of the hepatic veins proximal to a stenosis, intraluminal echogenicity, complete echogenic replacement of a hepatic vein and thickened hepatic vein walls. Direct signs on Duplex doppler and colour or power doppler include [Figure 6]; absence of hepatic vein flow, flow reversal or continuous flow (the pseudoportal doppler signal) upstream of a stenosis. Indirect signs include the presence of intrahepatic collaterals, ascites (which is almost always present) and hepatomegaly with heterogeneous parenchyma due to haemorrhagic infarction. The intrahepatic collaterals are tortuous vessels better seen on colour or power doppler than on gray-scale imaging. They are located predominantly in the upper or subcapsular parts of the liver. The caudate lobe emissary veins drain directly into the IVC at a lower level than the involved hepatic veins and consequently this lobe is either unaffected or enlarged (seen in about 50% of cases). Membranous webs may be identified as echogenic or focal obliterations of the hepatic vein or caval lumens and they are particularly well seen on doppler sonography.

Hepatic Veno-occlusive disease: This is a serious early complication of bone marrow transplantation, intensive chemotherapy or radiation therapy but can also be seen secondary to alkaloid toxicity (e.g., from Jamaican bush tea). It results from occlusion of multiple small hepatic venules. Although it is often clinically indistinguishable from Budd-Chiari syndrome, doppler sonography will demonstrate normal calibre, patency and phasic forward flow in the larger hepatic veins and IVC. Flow in the PV may be abnormal showing either reduced, reversed or "to and fro" flow and the hepatic artery flow typically demonstrates increased resistance.

Cardiac insufficiency: This can result from a variety of cardiac problems including constrictive pericarditis, ischaemic heart disease and pericardial effusion. Ultrasound may suggest the diagnosis of hepatic congestion due to cardiac insufficiency by demonstrating prominence of the hepatic veins and inferior vena cava with absence or reduction in the normal collapse of the IVC during inspiration. Hepatomegaly may also be seen, but the liver parenchyma is of normal echo texture. Doppler studies demonstrate an increase of the normal retrograde hepatic vein flow wave during atrial contraction and associated pulsatile antegrade and retrograde flow may be observed in the portal vein in advanced cases. Ultrasound may also demonstrate ascites, poor cardiac contractility and pericardial and pleural effusions.

Evaluating the risk of complications

The two most frequent complications of portal hypertension are gastrointestinal bleeding and hepatic encephalopathy.

Gastrointestinal bleeding

The higher the congestive index the higher the likelihood of a first variceal bleed. Splenic venous flow exceeding portal venous flow may be observed in patients with an increased prevalence of varices which tend to be larger. There is a weak but positive correlation between the size of the coronary vein (> 6 mm) and the risk of subsequent variceal bleeding. In one study preservation of hepatopetal coronary vein flow was associated with a lower risk of variceal haemorrhage.[14] None of the patients with portal hypertension and hepatopetal flow in the coronary vein had a history of variceal hemorrhage, whereas 40% of those with hepatofugal flow had had variceal hemorrhage.[14] Conversely, high hepatofugal flow in a patent paraumbilical vein exceeding hepatopetal flow in the PV may protect against oesophageal varices. The demonstration of para-oesophageal varices increases the risk of future variceal bleeds.

CONCLUSION

Portal hypertension is a commonly encountered clinical

condition with multiple causes and several sequelae. Ultrasound is an accurate non-invasive means of assessing its aetiology, severity and complications but in order to maximise diagnostic accuracy a systematic and thorough approach is advised involving assessment of the liver, spleen, portal and hepatic circulations. All radiologists involved in abdominal imaging should be familiar with the spectrum of gray-scale, colour and spectral doppler appearances.

REFERENCES

1. Bolondi L, Gandolfi L, Arienti V, Caletti GC, Corcioni E, Gasbarrini G, *et al*. Ultrasonography in the diagnosis of portal hypertension: Diminished response of portal vessels to respiration. Radiology 1982;142:167-72.
2. Vilgrain V, Lebrec D, Menu Y, Scherrer A, Nahum H. Comparison between ultrasonographic signs and the degree of portal hypertension in patients with cirrhosis. Gastrointest Radiol 1990;15:218-22.
3. Lafortune M, Marleau D, Breton G, Viallet A, Lavoie P, Huet PM. Portal venous system measurements in portal hypertension. Radiology 1984;151:27-30.
4. Bolondi L, Mazziotti A, Arienti V, Casanova P, Gasbarrini G, Cavallari A, *et al*. Ultrasonographic study of portal venous system in portal hypertension and after portosystemic shunt operations. Surgery 1984;95:261-9.
5. Ralls PW. Color Doppler sonography of the hepatic artery and portal venous system. AJR Am J Roentgenol 1990;155:517-25.
6. Moriyasu F, Nishida O, Ban N, Nakamura T, Sakai M, Miyake T, *et al*. "Congestion index" of the portal vein. AJR Am J Roentgenol 1986;146:735-9.
7. Haag K, Rossle M, Ochs A, Huber M, Siegerstetter V, Olschewski M, *et al*. Correlation of duplex sonography findings and portal pressure in 375 patients with portal hypertension. AJR Am J Roentgenol 1999;172:631-5.
8. Platt JF, Rubin JM, Ellis JH. Hepatic artery resistance changes in portal vein thrombosis. Radiology 1995;196:95-8.
9. Kok T, van der Jagt EJ, Haagsma EB, Bijleveld CM, Jansen PL, Boeve WJ. The value of Doppler ultrasound in cirrhosis and portal hypertension. Scand J Gastroenterol Suppl 1999;230:82-8.
10. Zoli M, Dondi C, Marchesini G, Cordiani MR, Melli A, Pisi E. Splanchnic vein measurements in patients with liver cirrhosis: A case-control study. J Ultrasound Med 1985;4:641-6.
11. Bolondi L, Gaiani S, Gebel M. Portohepatic vascular pathology and liver disease: diagnosis and monitoring. Eur J Ultrasound 1998;7: S41-52.
12. Rector WG Jr, Campra J, Ralls PW, Charms M. Utility and limitations of splanchnic venous ultrasonography in diagnosis of portal hypertension. J Clin Ultrasound 1986;14:689-96.
13. Taourel P, Perney P, Dauzat M, Gallix B, Pradel J, Blanc F, *et al*. Doppler study of fasting and postprandial resistance indices in the superior mesenteric artery in healthy subjects and patients with cirrhosis. J Clin Ultrasound 1998;26:131-6.
14. Wachsberg RH, Simmons MZ. Coronary vein diameter and flow direction in patients with portal hypertension: Evaluation with duplex sonography and correlation with variceal bleeding. AJR Am J Roentgenol 1994;162:637-41.
15. Wachsberg RH, Obolevich AT. Blood flow characteristics of vessels in the ligamentum teres fissure at color Doppler sonography: Findings in healthy volunteers and in patients with portal hypertension. AJR Am J Roentgenol 1995;164:1403-5.
16. Subramanyam BR, Balthazar EJ, Madamba MR, Raghavendra BN, Horii SC, Lefleur RS. Sonography of portosystemic venous collaterals in portal hypertension. Radiology 1983;146:161-6.
17. Kedar RP, Merchant SA, Malde HH, Patel VH. Multiple reflective channels in the spleen: A sonographic sign of portal hypertension. Abdom Imaging 1994;19:453-8.
18. Dodd GD 3rd, Memel DS, Baron RL, Eichner L, Santiguida LA. Portal vein thrombosis in patients with cirrhosis: Does sonographic detection of intrathrombus flow allow differentiation of benign and malignant thrombus? AJR Am J Roentgenol 1995;165:573-7.
19. Tublin ME, Dodd GD 3rd, Baron RL. Benign and malignant portal vein thrombosis: Differentiation by CT characteristics. AJR Am J Roentgenol 1997;168:719-23.
20. Richter J, Zwingenberger K, Ali QM, Lima Wde M, Dacal AR, de Siqueira GV, *et al*. Hepatosplenic schistosomiasis: Comparison of sonographic findings in Brazilian and Sudanese patients—correlation of sonographic findings with clinical symptoms. Radiology 1992;184:711-6.
21. Lafortune M, Matricardi L, Denys A, Favret M, Dery R, Pomier-Layrargues G. Segment 4 (the quadrate lobe): A barometer of cirrhotic liver disease at US. Radiology 1998;206:157-60.

Source of Support: Nil, **Conflict of Interest:** None declared.