Section: Radiodiagnosis

Original Research Article

DIAGNOSTIC ROLE OF ULTRASOUND IMAGING IN PORTAL HYPERTENSION

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ABSTRACT

Background: Portal hypertension (PHT) is a severe, debilitating disease. It is caused by many reasons, but cirrhosis is the most common. Colour Doppler ultrasonography, which is noninvasive, accurate, and readily available, is the first-line tool for the assessment and diagnosis of PHT, determining the aetiology and complications. This study aimed to assess the role of colour Doppler ultrasonography in the diagnosis and characterisation of PHT.

Materials and Methods: This cross-sectional study included 50 patients with PHT referred for radiodiagnosis. Ultrasonography of the abdomen using a curvilinear probe of 3.5-5.0 MHZ coupled with colour Doppler equipment was used for assessment. Data was presented using frequency and percentage.

Result: Of the 50 patients with PHT, most were men (74%), and the affected age group was 51–65 years (42%). Cirrhosis was the most common cause (70%), followed by portal vein occlusion (10%). Portal vein (PV) diameter >13 mm was observed in 60% of patients. Splenomegaly and ascites were observed in 78% and 86% of patients, respectively. Hepatopetal flow was the most prevalent pattern in the portal (74%), splenic (88%), and superior mesenteric veins (92%). Thrombosis was frequent in the PV (28%), and collateral veins mostly appeared in the splenorenal region (76%). 20% of the patients had >20% increase in PV diameter induced by inspiration.

Conclusion: Cirrhosis is the most common cause of PHT, and its sonographic findings include splenomegaly, ascites, and PV dilatation. Although severe, PHT is usually accompanied by preserved hepatopetal flow. These results emphasise the value of ultrasound in early detection, disease monitoring, and guiding clinical management without invasive intervention.

Keywords: Portal hypertension, Colour Doppler ultrasonography, Noninvasive, Cirrhosis, Hepatopetal flow.

INTRODUCTION

Portal hypertension (PHT) is a term used to describe the abnormal elevation of venous pressure in the portal system.[1] PHT is characterised by a hepatic venous pressure gradient (HVPG) of >5 mm Hg, but the risk of developing clinical complications of decompensated chronic liver disease, such as ascites, variceal bleeding, and hepatic encephalopathy (HE), exists only when it is ≥10 mm Hg. HVPG ≥10 mmHg is then referred to as clinically significant portal hypertension (CSPH).[2]

PHT arises due to elevated intrahepatic vascular resistance, usually secondary to cirrhosis, resulting in structural distortion by fibrosis, microvascular thrombosis, dysfunction of sinusoidal endothelial cells of the liver (LSECs), and activation of hepatic stellate cells (HSCs).[3] A second mechanism that controls PHT is a heightened splanchnic blood flow due to some extent of mesenteric arteriolar vasodilatation and reduced vascular responsiveness to endogenous vasoconstrictors.[4]

Early detection of PHT is of paramount significance to hepatologists because life-threatening complications, such as variceal bleeding and acute or chronic hepatic encephalopathy, can be avoided. Liver biopsy and HVPG measurement are the gold standards for assessing PHT; however, these

assessments are invasive and require specialised expertise. Therefore, their application in routine clinical practice and beyond third-level centres is restricted. Over the past decade, strong evidence favours the application of Noninvasive methods (NITs), like liver stiffness, to characterise PHT and direct surveillance for varices in need of treatment in patients with advanced chronic liver disease (ACLD).[5]

Colour Doppler ultrasonography, as an NIT, is safe, simple to perform, cost-effective, and provides numerical and precise results. Colour Doppler imaging aids in the evaluation of the size of the liver and spleen, bluntness of the liver edge, nodularity of the liver surface, and coarseness of the liver parenchyma, which are helpful parameters for hepatic fibrosis or PHT. Hence, the colour Doppler imaging is typically recommended as a promising marker for the diagnosis of cirrhosis and PHT.[6]

The Doppler method provides real-time visualisation of blood flow with qualitative and quantitative evaluation, and the use of microbubble-based contrast agents has enhanced the visibility of peripheral blood flow. Such an advantage of real-time visualisation is beneficial in the assessment of the severity of liver disease and PHT.[7] With ultrasonic Doppler, portal vein velocity, flow metric changes, and portosystemic collaterals can be accurately evaluated. The complicated haemodynamics of PHT in cirrhosis can be adequately detected and characterised using colour Doppler, and they correlate with the clinical stage of the disease. Knowledge of these different patterns of flow provides additional information that can aid in the diagnosis of cirrhosis, staging, and provide prognostic information for determining the course of therapy.[8]

With the limitations of invasive diagnostics, such as HVPG, more importance is now being placed on noninvasive techniques for CSPH assessment. Colour Doppler ultrasonography is more useful because of its capability to visualise haemodynamic alterations and vascular changes. This study aims to emphasise the utility of the US in the detection of CSPH, enabling earlier diagnosis and improved clinical decision-making.

Objectives

This study aimed to assess the range of colour Doppler sonographic features and changes in flow measures in patients with PHT, determine the presence of different portosystemic collaterals, and compare the findings with those of published literature.

MATERIALS AND METHODS

This 16-month cross-sectional study was conducted on 50 patients diagnosed with PHT who were referred to the Department of Radio-Diagnosis at Sree Balaji Medical College and Hospital, Chennai.

Inclusion and exclusion criteria

Adults diagnosed with PHT and aged 20–65 years were included, while paediatric patients, pregnant patients, and those with trauma were excluded.

Methods

All patients included in the study underwent abdominal ultrasonography using a 3.5–5.0 MHz curvilinear probe coupled with colour Doppler imaging. The examinations were performed using Siemens Acuson X300, Siemens Acuson S2000, and Mindray DC-7 ultrasound machines. Data are presented as frequencies and percentages.

RESULTS

The study involved 50 adults (13 females and 37 males), with the highest prevalence of PHT in the 51–65 years age group (42%), followed by the 36–50 years (40%) and 20–35 years (18%) age groups. The most frequent cause of PHT was cirrhosis (alcoholic, viral, and other causes), which accounted for 70% of the patients. Benign portal vein occlusion was seen in 10%, malignancy-associated venous occlusion and sinistral PHT in 6% and 4%, respectively. Other infrequent causes were observed in 10% of patients.

The majority of patients (60%) had a large portal vein diameter (> 13 mm). Among individuals with PHT, 78% had splenomegaly (> 13 cm). Ascites was observed in 86% of patients. [Table 1]

Table 1: Portal vein diameter, Splenomegaly and Ascites

Clinical Feature	Category	Count (%)
Diameter	<13 mm	20(40%)
	>13 mm	30(60%)
Splenomegaly	Present	39(78%)
	Absent	11(22%)
Ascites	Present	43(86%)
	Absent	7(14%)

Approximately 74% of patients showed centripetal flow, 6% showed centrifugal flow, 2% showed to-and-fro flow, and 18% had no flow. The value range was similar to that of the splenic vein flow, with 88% for centripetal flow and 8% for no flow. However, hepatopetal remained the most frequent in the superior mesenteric vein (SMV) at 92%, with no flow at 4%, and the others at 2% each. [Table 2]

Table 2: Flow in the portal vein, splenic vein and direction of flow SMV

Flow site	Flow direction	Count (%)
Flow in the portal vein	Petal	37(74%)
	To and fro	1(2%)
	Fugal	3(6%)
	No flow	9(18%)
Flow in the splenic vein	Petal	44(88%)
	To and fro	1(2%)
	Fugal	1(2%)
	No flow	4(8%)
Flow in the SMV	Petal	46(92%)
	To and fro	1(2%)
	Fugal	1(2%)
	No flow	2(4%)

Thrombosis was most commonly observed in the portal vein (28%), followed by the splenic (8%) and superior mesenteric veins (4%). Among portosystemic collaterals, splenorenal shunts were the most frequent (76%), followed by the gastroesophageal junction (44%) and paraumbilical collaterals (40%). Additionally, 80% of the patients showed a < 20% increase in the portal vein diameter with deep inspiration. [Table 3]

Table 3: Thrombosis in veins, Collaterals and Variation in PV diameter

Clinical feature	Category	Count (%)
Thrombosis in Veins	Portal vein	14(28%)
	Splenic vein	4(8%)
	Superior mesentric	2(4%)
Collaterals	Gastroesophageal junction (GEJ)	22(44%)
	PU	20(40%)
	SR	38(76%)
	Cavernoma	3(6%)
	GB	5(10%)
Increase in PV Diameter	>20%	10(20%)
	<20%	40(80%)

DISCUSSION

PHT is the most significant complication of cirrhosis and is characterised by HVPG of >5 mmHg. Clinically significant PHT is characterised by HVPG ≥ 10 mmHg. Our study was designed to assess the utility of colour Doppler ultrasonography in the evaluation of PHT and its haemodynamic alterations. Through the analysis of flow metric parameters, identification of portosystemic collaterals, and correlation of sonographic with clinical findings, this study demonstrated the value of Doppler imaging as a noninvasive, accessible, and useful tool.

In our study, the majority of patients with PHT were male (74%), with the highest incidence observed in the 51–65 years age group (42%). A total of 70% of patients had cirrhosis (alcoholic, viral, and other types) as the common aetiology of PHT. Tan et al. had similar findings and reported that males have a high incidence of cirrhosis (mainly alcohol), which ultimately led to PHT or malignancy.[9] Cokkinos et al. emphasised that, while the normal PV diameter is 13 mm, 60-70% of patients with PHT had a PV diameter >13 mm. These reports correlate with our study, as 60% of the studied cases had a portal vein diameter above 13mm.[10]

Splenomegaly, anaemia, leukopenia, thrombocytopenia, oesophageal and fundal varices, and variceal bleeding are among the most common signs and symptoms of PHT, as mentioned in the study by Ozturk et al,[11] Our findings also support this study, as 78% of the admitted PHT cases had splenomegaly. Another study on 111 patients by Gibson et al. states that 70-80% of their cases had reports of splenomegaly, which further strengthens our study.[12]

Burcharth et al. studied 108 patients and found that the direction of portal vein flow was towards the liver, with 65% hepatopetal flow. They also found that 3% of cases had bidirectional flow in the portal vein.[13] Our study has similar findings, as we have reported 74% for hepatopetal flow and 2% for bidirectional flow. In another study by Ditchfield et al. on 118 PHT cases, 5.3% of cases had reversed portal vein flow,[14] whereas our study had 6%, which is very close.

Our study reported various collaterals, such as splenorenal, GEJ, and GB, in 76%, 44%, and 10% of patients, respectively. These findings are reflected in the results of two studies, one by Chawla et al. on 102 patients with GB collateral between 13-24% and another on 40 patients by Subrananyam et al. with reporting 88% and 64% of cases on splenorenal and GEJ collaterals, respectively.[15,16]

In our study, 80% of patients showed a < 20% increase in diameter with deep inspiration, and only 20% of patients had an increased diameter of > 20%. These results mirror the study by Chakenahalli et al., where 87.9% of the 63 patients didn't show any increase in the PV diameter of > 20%.[17]

Our study highlights the value of colour Doppler ultrasonography in the diagnosis of PHT. Its capacity to identify flow metric alterations and image portosystemic collaterals justifies its routine use in clinical practice, especially in cases where invasive techniques are impractical. In the future, improvements in Doppler technology can generate even more precise imaging and assist in the early detection and monitoring of clinically relevant PHT, potentially revolutionising patient care and outcomes in chronic liver disease.

Limitations

This study is limited by its small sample size, single-centre design, and absence of a gold-standard reference such as HVPG for comparison. Future research with larger cohorts and invasive correlation is warranted.

CONCLUSION

This study confirms that colour Doppler ultrasonography is an effective and noninvasive tool for evaluating portal hypertension, particularly in cirrhotic patients. It effectively detects significant haemodynamic alterations, including the reversed direction of portal vein flow, decreased respiratory variation in diameter, and the presence of portosystemic collaterals, such as splenorenal and GEJ collaterals. These observations are consistent with the existing literature, supporting the diagnostic role of Doppler echocardiography. With continuing constraints in invasive techniques, technological advances in Doppler technology are urgently required to enhance the precision of imaging, early detection, staging, and control of PHT.

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