

To Study the Role of Duplex Sonography in Cases of Portal Hypertension

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ABSTRACT

BACKGROUND

New techniques like elastography and hepatic vein transit time (HVTT) for diagnosing portal hypertension are costly, invasive, time-consuming and not easily available. CT/MRI are now easily available modalities, but they cannot establish flow pattern in portal venous system. Ultrasound (grey scale and colour doppler) though widely available are still not accepted as a definite tool to establish early PHT. We wanted to determine the role of ultrasound (greyscale and colour Doppler) as a definitive modality to diagnose early PHT, thus preventing absolute liver damage by fibrosis.

METHODS

This is a prospective cross-sectional study undertaken in the Department of Radiodiagnosis, Government Medical College, Amritsar, between February 2018 to May 2018, conducted among 50 patients clinically diagnosed/suspected as PHT. All these patients were given a sonographic examination. Sonographic findings including colour Doppler of blood flow pattern were studied. In addition, clinical and biochemical findings were also correlated. Findings were tabulated, subjected to statistical analysis and inferences were drawn.

RESULTS

Our study found a good correlation with the assessment of spleen size (≥ 13 cms) showing a p-value of 0.003. p-value of ascites is 0.008 while diameter of portal vein (p-value 0.005) also show strong correlation. Splenic vein was found to be highly specific (77.4%) but not sensitive. Decrease in velocity of portal vein correlated significantly, more with portal hypertension, as compared to increase in splenic vein velocity. Various collaterals showed 100% sensitivity with p-values for coronary collaterals of 0.001 and paraumbilical collaterals to be of 0.000.

CONCLUSIONS

Colour Doppler is still the best modality of choice for diagnosing portal hypertension at very early stages. Doppler helps by diagnosing direction of flow as well as velocity of portal venous system. It can also help in evaluating collaterals and portosystemic shunts. Other information provided by pulsed Doppler includes detection of veno-occlusive disease.

KEY WORDS

Portal Hypertension (PHT), Colour Doppler, Ultrasound, Liver, Spleen, Portal Vein, Splenic Vein, Collaterals, Ascites

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BACKGROUND

Portal hypertension (PH) is defined as an increase of the hydrostatic pressure within the portal vein or its tributaries. It represents an increase in the pressure gradient between the portal vein and hepatic veins or the inferior vena cava (IVC). The most common cause of portal hypertension is cirrhosis, with portal hypertension developing as a result of a progressive increase in resistance to portal vein blood flow.

Doppler ultrasound is the accepted gold standard for assessing direction of flow in the portal vein (PV).¹ Grey-scale imaging may demonstrate changes, such as volume redistribution, capsule nodularity, parenchymal nodularity, and echotexture changes. The Doppler findings in the hepatic and portal veins, hepatic artery, and varices allow assessment of liver cirrhosis. There are other flow patterns besides, reversal of the portal venous flow. These flow patterns, although not as easily understood, may play an important role in assessing the disease status. Duplex sonography is a sensitive technique for detection of flow patterns and other associated findings in a patient of portal hypertension. Duplex sonography has marked potential in evaluating liver, spleen, and portal venous system, including normal and congenitally malformed anatomy, besides various pathologies.

Colour Doppler is an excellent noninvasive tool for preoperative as well as postoperative evaluation of patients with PHT. It also helps in diagnosis of patients with unexplained jaundice or upper GI bleed. Sonography is a reliable and reproducible diagnostic technique in patients with PHT

Newer techniques such as elastography and hepatic vein transit time (HVTT) have the potential to exclude patients without significant fibrosis or cirrhosis; however, they are operator dependent and require specific software.² Angiographic techniques such as transhepatic portography and wedge hepatic venography are invasive and time consuming. Modalities like CT/MRI are noninvasive but are relatively costly and cannot establish flow patterns in portal venous system.

We wanted to re-establish the role of ultrasound (greyscale and colour Doppler) as a definitive modality to diagnose early PHT, thus helping in preventing absolute liver damage by fibrosis.

METHODS

Fifty consecutive patients with clinical signs (e.g. jaundice, ascites, muscle wasting, cutaneous spider angiomas, ecchymosis, palmar erythema and flapping tremors; etc) or biochemical findings (decreased serum albumin, increased prothrombin time, increased serum bilirubin, etc) suggestive of portal hypertension were selected. After noting all relevant information like history, clinical findings, biochemical investigations, ultrasound was done using sector probe (2-5 Mhz) and where required linear probe (7-12 Mhz).

Examination was done in grey scale as well as with colour doppler mapping and relevant ultrasonographic findings were noted. Patients were required to come after overnight fasting of minimum eight hours. Coupling agent in the form of jelly was used for proper contact between probe and skin.

Examination was done with the patient lying in supine position in B-scale followed by examination with colour-doppler mapping. The doppler study was performed using intercostal or subcostal approach, by positioning the sample volume in at least 1/3rd of vessel diameter in the centre, to reduce the possible influence of peripheral changes in vessel wall. The angle of insonation of Doppler US beam was maintained at ≤ 60 degree and the spectral wave form was recorded for at least 4-6 secs. Patient was instructed to hold breath during the examination. Following parameters were studied and recorded systematically-

1. Liver and spleen size with sonographic signs of cirrhosis.
2. PV and SV diameter and size.
3. PV and SV flow velocity.
4. Direction of flow and spectral pattern of PV.
5. Various Collaterals.
6. Ascites.

Statistical Analysis

All findings were tabulated, and inferences drawn. Statistical analysis was done using ANOVA test to calculate Pearson's Correlation factor, odds ratio, specificity and sensitivity as required.

RESULTS

Chief Complaints

The chief complaint in reported cases of PHT in our study group included hematemesis (32%), jaundice (20%), and alcoholic liver disease (22%). Clinical examination showed ascites in 14% and splenomegaly in 24% cases.

Age and Sex Distribution

The mean age of the patients in our study was 41.52 ±11.88 years. PHT occurred more frequently in males 38 (76%) in the age group 51-65 years compared to females 12 (24%) in the age group of 20-30 years. The male to female ratio is 3:1. Age range in our study compared with other studies.

Biochemical Parameters

In our study 72% of the patients had raised serum bilirubin and 42% had abnormal SGOT/SGPT levels. Viral markers were positive in 12% of patients. Since large number of patients who were sonographically confirmed as that of portal hypertension had abnormal liver function tests and viral markers, our objective of taking biochemical parameters in our study was vindicated.

Investigation	No. of Pts.	% of Pts.
S. Bilirubin (≥1.5 mg %)	36	72%
SGOT/SGPT(≥20 IU)	21	42%
PTI (≤80%)	18	36%
Alk. Phosphates(≥144 IU)	14	28%
Viral markers	06	12%

Table 1. Biochemical Parameters

Sl. No.	B-Scale Findings	No. of Pts.	% of Pts.
1	Liver size(≥15 cm)	09	18
2	Spleen size (≥13 cm)	26	52
3	PV diameter(≥13 mm)	22	44
4	SV diameter(≥10 mm)	18	36
5	Ascites	18	36

Table 2. B-Scale Findings

Liver Size

Liver was found to be enlarged or shrunken according to the different stages of PHT. A shrunken liver was seen in only advanced stages in 17 patients. Hepatomegaly with size more than 15 cm was seen in 9 patients. Rest of the patients had normal size. In our study, mean size of liver in patients with PHT was 12.43 ± 2.52 cm and it was 14.3 ± 2.31 cm in patients without PHT.

Spleen Size

Splenomegaly (size more than 13 cm) was seen in 52% patients having PHT. Splenomegaly was less common in patients with alcoholic liver disease (13.5 cm) compared to non-alcoholic (15.6 cm) or patients with vascular occlusion disease (16.52 cm). In our study spleen size correlated strongly with presence of PHT with p value of 0.003 and odds ratio estimate of 8.500 (sensitivity of 89.47% and specificity of 50%).

Ascites

In our study group 45% (18 out of 40) of PHT patients had ascites. Ascites was seen in 70% cases of cirrhosis and 60% cases of extrahepatic portal vein obstruction. Presence of ascites correlated strongly with PHT with p value of 0.008 and positive predictive value of 45%. It is 100% sensitive and 31.25% specific.

Portal Vein Diameter

Mean PV diameter in our study was 9.48 ± 1.78 mm in normal subjects and 13.28 ± 4.39 mm in PHT patients. PV diameter of >13 mm is seen in 22 patients out of 40 with sensitivity of 62%. Patients having PV diameter >15 mm with collaterals showed specificity of 100%. PV diameter >17 mm has predictive value of 100% for PHT. Correlation factor for PHT is 0.005 with odds ratio estimate of 13.5.

Splenic Vein Diameter

Mean splenic vein diameter in normal subjects was 5.48 ± 1.38 mm. In patients with PHT, the mean splenic vein diameter is 10.35 ± 2.24 mm. Increase in splenic vein diameter was seen in 36% patients, which is a specific sign (77.4%) but it has low sensitivity (13.33%). Odds ratio estimate was less than 1 (0.519).

Portal and Splenic Vein Velocity

Portal Vein Flow Velocity- The fasting mean flow velocity in patients in our study was 12-18 (mean 16.38 ± 2.5) cm/sec. It showed variation on respiration i.e. decreased on inspiration and increased on expiration. Velocity less than 12 cm/sec was found to be highly suggestive of PHT, according to our study. Velocity of ≤ 15 cm/sec could be taken as absolute cut-off for detection of PHT. Our study had 42% patients with PV velocity of <15 cm/sec and there was strong correlation of PV velocity less than 15 cm/sec with presence of PHT. p value was 0.001 and odds ratio estimate was 13.778.

Splenic Vein Velocity

During our study we found that in cirrhotics, splenic vein flow was significantly faster than in the portal vein. On the contrary, in healthy controls, splenic vein flow was significantly slower

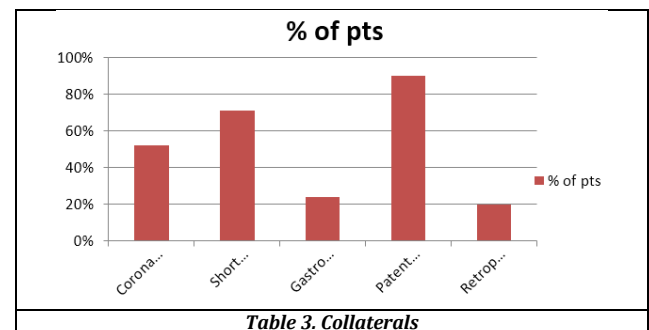
than in the portal vein. Mean SV velocity in normal patients was 13.0 ± 0.5 cm/sec and in cirrhotics was 12.2 ± 0.6 cm/sec.

Direction of Flow in PV

Using colour Doppler studies, we can easily establish the direction of flow of blood in PV. Flow in PV is normally directed towards liver i.e., hepatopetal flow (seen in 48% of our patients) with variations secondary to respiration. Helical flow is present in some normal patients also, mimicking bidirectional flow. But in the patients with portal hypertension, flow may be hepatopetal, hepatopetal with loss of respiratory phasicity, or reversed flow (hepatofugal). To rule out reversal in otherwise normal patients, the flow has to be assessed throughout the portal system. In our study 48% had hepatopetal flow while 24% had hepatofugal flow and bidirectional flow was seen in 4 patients. But there was no correlation of direction of flow, with PHT.

Porto-Systemic Collaterals

Various collaterals assessed in our study subjects included: Left gastric (coronary vein), paraumbilical, splenocaval, splenoportal, retroperitoneal, and collaterals in the wall of GB. Short gastric vein is seen between upper pole of spleen and gastric wall and are the commonest collaterals seen (71% in our study).



Final Sonographic Diagnosis

Intrahepatic Causes

Most important intrahepatic cause of PHT is cirrhosis. Most common cause of cirrhosis particularly in males is alcoholic liver disease followed by post infective cirrhosis. Less common intrahepatic causes include noncirrhotic portal vein fibrosis and veno-occlusive disease.

Cirrhotic Changes Seen on B-Scale in Liver

1. Coarse echotexture with altered echogenicity
2. Micro- or macro nodularity of liver surface.
3. Presence of degenerating nodule
4. Reduced PV and Hepatic vein radicles.
5. Reduced liver size
6. Enlarged caudate lobe (caudate to right lobe ratio of more than 0.65)
7. Reduced transverse diameter of segment 4 i.e., less than 30 mm
8. Noncirrhotic portal vein fibrosis include thickening of intrahepatic branches of portal walls seen on u/s as increased periportal echogenicity and subcapsular atrophy. Heterogeneously decreased portal perfusion,

particularly in periphery with increased hepatic arterial perfusion helps to diagnose it.

Extrahepatic Causes

The three common cause were- Thrombosis of portal vein with or without portal vein formation, Budd-Chiari syndrome and post CHF syndrome. Portal vein occlusion can occur due to thrombus, tumour and post inflammatory fibrosis like pancreatitis, peritonitis etc. PV occlusion is usually permanent and is followed by formation of multiple dilated vascular channels called cavernous transformation of PV. The portal vein thrombus is usually hypoechoic and can be difficult to discern on grayscale imaging alone. Therefore, evaluation of the main portal vein on both grayscale and colour flow Doppler is necessary. The portal vein thrombosis is associated with the presence of periportal collaterals, and sometime cavernous formation but flow remained in hepatopetal direction.

Hyperkinetic PHT

Was diagnosed in patients with massive splenomegaly with hepatopetal flow and high PV velocity

Sl. No.	Final Sonographic Diagnosis	No. of Pts.	% of Pts.
1.	ALD with cirrhosis	17	34
2.	PV thrombosis	06	12
3.	Non cirrhotic PV fibrosis	06	12
4.	Veno occlusive disease	01	02
5.	Post infective Cirrhosis	05	10
6.	Post CHF PHT(Extrahepatic)	01	02
7.	Budd Chiari syndrome	02	04
8.	Alcoholic hepatitis/ pancreatitis/fatty liver etc.	10	20
9.	Cavernoma formation	02	04
10.	Hyperdynamic PHT	02	04

Table 5. Final Sonographic Diagnosis

DISCUSSION

Age and Sex Distribution

The mean age of the patients in our study was 41.52±11.88 years. PHT occurred more frequently in males 38 (76%) in the age group 51-65 years compared to females 12 (24%) in the age group of 20-30 years. The male to female ratio is 3:1 which compared well with similar studies done earlier. In a similar study by Mittal et al the mean age of patients was 45 years¹. The mean age was 50.9±17.6 years (Range 13-85) in a study done by Shateria et al.³ The 14 studies included in their analysis by Kim G. et al included 827 patients with cirrhosis. The average age of the patients was 53.2±9.6 years old.⁴

Liver Size

No correlation was found between liver size and PHT (0.053 correlation factor by Pearson Chi-square test) in our study. Liver size was found to be nonspecific for PHT (specificity of 13.5 % with sensitivity of 61.5%). In comparison, Perisic et al found significant correlation between diameters of right liver lobe and portal vein.⁵

Splenomegaly

In our study spleen size correlated strongly with presence of PHT with p value of 0.003 and odds ratio estimate of 8.500

(sensitivity of 89.47% and 50% specificity). According to Bao-Min Shi et al splenomegaly is a cardinal feature of hepatic cirrhosis complicated by portal hypertension. The prevalence of splenomegaly in cirrhosis varies between 36-92%.⁶ Perisic et al found significant correlation between diameters of spleen and splenic vein.⁵ 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.⁷ Splenomegaly with a length ≥ 11 cm is another valuable diagnostic sign.⁸

Ascites

Presence of ascites correlated strongly with PHT in our study with p value of 0.008 and positive predictive value of 45%. It is 100% sensitive and 31.25% specific. Bao-Min Shi et al demonstrated that cirrhotics with ascites had a significantly lower portal flow velocity and volume compared to those without ascites, which confirms that ascites is a sign of liver function decompensation.⁶ Perisic et al found significantly lower portal flow velocity in patients with ascites as compared to those without ascites.⁵ This is a non-specific finding but is frequently seen in portal hypertension.⁷

Portal Vein Diameter

Patients having PV diameter >15 mm with collaterals showed specificity of 100%. PV diameter >17 mm has predictive value of 100% for PHT. Correlation factor for PHT is 0.005 with odds ratio estimate of 13.5. 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%.⁷ Chawla et al found no difference in the maximum inner diameter of portal vein in cirrhotics and controls.⁹ Bao-Min Shi et al also did not find significant difference in portal vein diameter in their study.⁶ El-Shabrawi et al found significant correlation of portal vein diameter and cirrhosis in their study on children with chronic liver disease.¹⁰ Perisic et al also found significant increase in portal vein diameter in patients with hepatic encephalopathy.⁵ Shateria et al concluded that sonographic portal vein parameters cannot be a substitute for clinical grading and staging of cirrhosis.³

Splenic Vein Diameter

Was found to be a specific sign (77.4%) but has low sensitivity (13.33%) in our study. Odds ratio estimate was less than 1(0.519). Bao-Min Shi et al found significant differences in splenic vein diameter in their study.⁶ Perisic et al did not find statistically significant difference in splenic vein diameter in patients with and without hepatic encephalopathy.⁵

Portal Vein Flow Velocity

Normal Portal vein velocity (PSV) ranges between 20 cm/sec and 40 cm/sec. Our study had 42% patients with PV velocity of <15 cm/sec and there was strong correlation of PV velocity less than 15 cm/sec with presence of PHT. P value was 0.001 and odds ratio estimate was 13.778. The portal flow velocity and volume were significantly lower in patients with cirrhosis compared to controls according to Bao-Min Shi et al.⁶ According to Perisic M et al portal flow velocity shows linear

decrease, related to the increase of the liver damage.⁵ In patients with alcoholic cirrhosis, PV blood velocity and flow are correlated to the severity of portal hypertension and to the severity of liver failure.¹¹ A low flow velocity of <16 cm/sec in addition to a caliber increase in the MPV are diagnostic features of portal hypertension.¹² Decrease in portal flow velocity has been found to be significant by other authors also.^{3,13}

Splenic Flow Velocity

Increased splenic velocity was found to be significant in cirrhotics compared to healthy controls. Odds ratio estimate of splenic velocity for PHT was 3.955 Mean SV velocity in normal patients is 13.0±0.5 cm/sec and in cirrhotics was 12.2± 0.6 cm/sec. Splenic flow velocity did not show significant increase according to Perisic et al.¹⁴

Direction of Flow in Portal Vein

In our study 48% had hepatopetal flow while 24% had hepatofugal flow and bidirectional flow was seen in 4 patients. But there was no significant correlation of PHT with direction of flow. According to Iranpour et al, a normal portal venous flow is hepatopetal. A flow reversal (or a hepatofugal flow) is seen in the case of portal hypertension. In particular, in patients with cirrhosis, obstruction of the hepatic venules and sinusoids by fibrosis, substantiated by arteriportal and portosystemic shunting, eventually leads to flow reversal.¹² In the study by Mittal et al overall six patients (12%) among a total of fifty had non-hepatopetal flow (hepatofugal/bidirectional), four of them (8%) showed continuous hepatofugal flow and two patients (4%) showed bidirectional flow.¹ The presence of a non-hepatopetal flow pattern implicates an increased risk of oesophageal varices bleeding.¹¹

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.⁷

Collaterals

Left gastric (coronary vein): Normal diameter of coronary vein is 4 mm while >7 mm diameter is s/o PHT. Short gastric vein is seen between upper pole of spleen and gastric wall and are the usually the commonest collaterals seen (71% in our study). Short gastric vein and left gastric vein have flow directed towards oesophagus in cases of PHT and patients presenting with hematemesis. Paraumbilical vein runs in ligamentum teres in left lobe of liver and is collapsed normally. Recanalization of paraumbilical vein is a very specific and highly sensitive sign of PHT. It usually measures 3 mm and shows hepatofugal flow towards umbilicus in PHT. Splenorenal collaterals are seen between spleen and upper pole of left kidney. Splenorenal and paraumbilical collaterals decrease the flow towards oesophagus and reduce the chances of hematemesis. But the risk of hepatic encephalopathy is increased. Other collaterals include splenocaval, splenoportal,

retroperitoneal, and collaterals in the wall of GB. In our study the commonest collaterals seen (71%) are mentioned in collaterals section of results. Our study shows 100% sensitivity for presence of collaterals in patients of PHT. p-value for various collaterals shows strong correlation. Coronary vein with p-value of 0.001, and paraumbilical collaterals with p-value of 0.000 shows strongest correlation. Collaterals are never seen in normal patients. Cirrhotics with the lowest portal velocity have oesophageal and gastric varices.¹¹ 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. According to Nakshabandi et al, the three most commonly identified collaterals include gastroesophageal junction, paraumbilical vein, splenorenal and gastrosplenic collaterals.⁷ 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. 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 sub-types of portal hypertension. 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.⁷

Limitations

Assessment of portal flow using Doppler can be ambiguous or spuriously reversed due to technical errors like-

1. Improper pulse frequency settings.
2. Angle of insonation >60°
3. Improper positioning and size of Doppler gate.
4. Gas in abdomen obscuring the structures.
5. Massive ascites.
6. Obesity.

CONCLUSIONS

Sonography helps in establishing PHT in all cases, where it is present. Ultrasound and Colour Doppler are non-invasive, cheap and easily accessible modalities for evaluating patients with PHT. Sonography has predominant role in establishing the diagnosis of PHT in conjunction with clinical and biochemical findings. It also spares patients from invasive and costly techniques, thus, in the process, avoiding excruciating pain as well as financial burden to the family members.

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