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Original Article

Radiology Section

Diagnostic Value of Doppler Ultrasonography in Non-invasive Diagnosis of Chronic Liver Disease and Portal Hypertension

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ABSTRACT

Introduction: A number of disorders can affect the hepatic parenchyma, leading to chronic liver disease (CLD). CLD and cirrhosis are a significant cause of morbidity and mortality in developing nations and ultimately lead to the development of portal hypertension (PHT).

Aim: To assess the portal and hepatic vein hemodynamics in patients of CLD and PHT using Doppler ultrasonography (USG).

Materials and Methods: This prospective study was carried out in 50 biopsy and/or biochemically proven cases of CLD. Doppler USG was performed and flow hemodynamics in hepatic veins and portal vein was assessed.

Results: Portal vein diameter >13mm was seen in 22.2% cases of CLD without PHT whereas, PV diameter >13mm was seen in 56.2% cases with PHT. Also dilatation of the splenic vein (> 10mm) was observed in 46.9% patients of CLD with PHT. Reduced mean peak portal vein velocity (PVV) was observed in patients with CLD (14.2cm/sec) and CLD with PHT (12.3cm/sec). Altered hepatic vein morphology was seen in 74% cases of CLD.

Conclusion: Doppler USG evaluation of hepatic and portal vein hemodynamics is a very useful tool in the non-invasive diagnosis of CLD and can be reliably used to distinguish patients of CLD with and without PHT.

Keywords: Cirrhosis, Endoscopy, Hepatic vein, Portal vein

INTRODUCTION

CLD is caused by various insults to hepatocytes. These in turn lead to liver fibrosis and finally to cirrhosis where nodules replace the normal architecture of liver. Morphologically, liver cirrhosis can be defined as a pathological condition characterized by diffuse pseudonodules formation throughout the entire liver. Hepatic necrosis, increased connective tissue and regeneration of hepatocytes are the fundamental pathogenetic changes in the development of cirrhosis [1].

Cirrhosis of liver has been classified into micronodular and macronodular types. In micronodular cirrhosis, nodules are less than 3mm in diameter while macronodular cirrhosis is characterized by nodules of varying size more than 3mm. Alcohol consumption is the most important cause of micronodular cirrhosis and chronic viral hepatitis is the most frequent cause of macronodular cirrhosis [2].

The classic clinical presentation in CLD and cirrhosis is hepatomegaly, jaundice and ascites. However, these classical signs and symptoms of liver disease are seen only in 60% of patients. When incidental screening tests such as liver transaminases or radiologic findings suggest the presence of liver disease, the diagnosis of asymptomatic cirrhosis is usually made and patients undergo further evaluation and liver biopsy to confirm the diagnosis.

Hemodynamic abnormality frequently associated with CLD is portal hypertension (PHT). Gilbert A et al., [3] first coined the term Portal Hypertension (PHT), recognizing the association of ascites, cirrhosis, splenomegaly and variceal bleeding. Relative or absolute obstruction to the splanchnic blood flow or less commonly increased portal blood flow contributes to the development of PHT.

PHT is defined as a wedged hepatic vein pressure or direct portal vein pressure of more than 5 mmHg greater than inferior vena cava pressure, a splenic vein pressure of greater than 15mmHg or portal vein pressure at surgery of more than 30 cm H_2O [4]. Normal Portal pressure is 5-10mmHg.

Conventional USG is considered a first-line imaging technique for the initial assessment of patients with suspected or

established liver disease, and/or the monitoring of diffuse liver disease and its complications [5]. However, Doppler provides information regarding the presence or absence of flow and the direction and velocity of the flow rapidly and relatively inexpensively [6] and thus enables detection of abnormalities of the hepatic and portal venous system.

In the evaluation of a case of CLD and PHT, abdominal B-mode USG, liver biopsy, upper Gastro-Intestinal (GI) endoscopy for gastro-esophageal varices, and direct measurement of portal pressure have traditionally been used. B-mode USG has low sensitivity and specificity. The latter three including liver biopsy is an invasive and painful procedure, and associated with low, but a definite risk of patient morbidity and mortality. Therefore, it is important to use non-invasive methods in diagnosis of CLD. Doppler USG provides a quantitative measurement of blood flow to the liver and can evaluate hemodynamic state, so it is theoretically superior to B-mode ultrasound in sensitivity and specificity. Hence, the present study will be done to evaluate the role of Doppler USG in the evaluation of CLD and PHT.

MATERIALS AND METHODS

This prospective study was conducted in the Department of Radio-Diagnosis and Imaging, ASCOMS Hospital, Sidhra Jammu and Kashmir, India from October 2012 to October 2013. The study group comprised of 50 biopsy and/or biochemically proven cases of CLD. Patients with evidence of cardiac or respiratory failure, paediatric cases, patients with Transjugular intrahepatic shunts, pregnant women and traumatic cases were excluded from the study. Ethical committee of the institute had approved the study and verbal consent was obtained from all the patients. A detailed clinical history was elicited from all the patients. All patients were subjected to upper GI endoscopy and relevant laboratory investigations. Liver biopsy was performed wherever possible. Diagnosis of CLD and cirrhosis was made on liver biopsy and/or persistently raised liver enzymes. The presence of gastropathy and/or gastro-esophageal varices on endoscopy was taken as evidence of PHT.

Patients were advised to come empty stomach for USG and informed consent was obtained from all the patients. Sonographic examination was performed using “Logiq 500 PRO Series GE” and “Logiq C5 Premium GE” Doppler USG machines. Imaging was carried out on the patients in quiet respiration. Doppler frequency of 3.5MHZ was used for evaluating the portal and hepatic veins. Doppler angle was kept between 45-60 degrees for optimal detection and filter settings were kept at the lowest possible setting. Sample volume, gate position, pulse repetition frequency, colour base and gain settings were kept at optimum level for optimal signal detection. Peak Portal vein velocity (PVV) was evaluated in all patients from the hilar segment. Also, Waveform of hepatic vein was evaluated in right and middle hepatic vein, about 3-6 cm from the confluence of the hepatic vein and inferior vena cava at the end of normal inspiration. Upper GI endoscopy and relevant laboratory investigations including liver function tests were performed in all cases. After obtaining the full data of the study group, the collected data was presented in the form of tables and analyzed using appropriate statistical methods.

RESULTS

Fifty cases of CLD formed the material of present study. The age of patients varied from 22 years to 78 years. The majority of the patients (16/50, 32%) belonged to 6th decade [Table/Fig-1]. Out of 50 patients, 33 (66%) were males and 17 (34%) were females with a male to female ratio of 1.9 [Table/Fig-1]. Of these 50 cases, 18 cases had CLD only while 32 cases had CLD with PHT as observed on upper GI endoscopy. Jaundice was the most common presenting complaint in patients with CLD alone (75%) while upper GI bleed was the most common clinical manifestation in patients with PHT (72.7%).

Portal vein (PV) diameter was evaluated in all the patients. In patients with CLD only, PV diameter >13mm was seen in 22.2% cases (4/18), whereas PV diameter >13mm was seen in 56.25% cases (18/32) with PHT [Table/Fig-2]. Also dilatation of the splenic vein (>10mm) was observed in 46.9% (15/32) patients with PHT while no case of CLD without PHT had SV diameter >10 mm [Table/Fig-2].

Doppler findings were also evaluated in all the cases. In patients with CLD only, mean peak PVV was 14.2cm/sec while in patients of CLD with PHT it was 12.3cm/sec [Table/Fig-2]. Hepatic vein waveform was also evaluated in all the patients. Abnormal hepatic vein (HV1 and HV2) waveform was detected in 74% (37/50) cases of CLD. Normal triphasic waveform i.e. HV0 pattern was seen in 26% (13/50) cases only [Table/Fig-3].

Age groups (years)	No. of males	No. of females	Total
21-30	2	0	2
31-40	3	1	4
41-50	7	4	11
51-60	10	6	16
61-70	8	4	12
> 70	3	2	5
Total	33	17	50

[Table/Fig-1]: Age & sex distribution of patients (n=50).

Finding	CLD only (n=18)	CLD with PHT (n=32)
PV Diameter > 13 mm	4	18
SV Diameter >10 mm	0	15
Mean PVV (cm/sec)	14.2	12.3

[Table/Fig-2]: PV and SV diameter and mean PVV in cases of CLD (n=50).

Hepatic Vein Waveform	CLD only	CLD with PHT	Total
Normal (HV0)	4	9	13
Abnormal (HV1&HV2)	14	23	37
Total	18	32	50

[Table/Fig-3]: Hepatic vein waveform on Doppler USG (n=50).

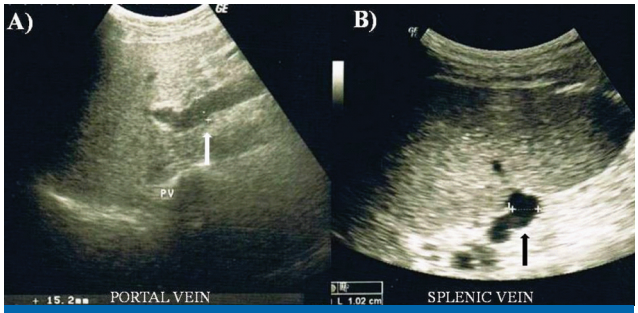
DISCUSSION

Cirrhosis of liver is the final outcome of various insults to the liver parenchyma and represents the most common cause of portal hypertension. Early diagnosis of cirrhosis and PHT is critical to prevent the development of life threatening complications. Study of hepatic hemodynamics provides an insight into the pathophysiology of the cirrhosis and PHT and helps determine newer therapeutic alternatives. Direct measurement of the portal venous pressure is an accurate method of diagnosing PHT. But is an invasive procedure and has a definite risk of patient morbidity and mortality. Doppler USG is being increasingly used as a non-invasive method for the evaluation of portal hemodynamics. It is a safe, painless and repeatable method and well accepted by the patients. It provides a quantitative assessment of hepatic and portal blood flow and thus can be used as an inexpensive and non-invasive method in evaluation of CLD and PHT. It can also be used as the initial imaging technique for confirming suspected portal hypertension in patients of hepatic cirrhosis. So the study was carried out to evaluate the role of Doppler USG in the evaluation of CLD and PHT.

Our study comprised of 50 cases of CLD and portal hypertension. In our study, majority of cases were seen in 6th decade of life with a male preponderance, similar to the previously published studies [7]. Jaundice was the most common clinical feature in cases of CLD without PHT, seen in 75% cases similar to observations of Onyekwere CA et al.,[8] where jaundice was seen in nearly 80% cases. In cases of CLD with portal hypertension, the most common clinical feature was upper GI bleed which was seen in 72.7% cases.

PV diameter >13mm [Table/Fig-4a] was seen in 56.25 % cases of PHT. Our results are in agreement with Bolondi L et al., [9] who evaluated 160 patients with portal hypertension. 73 of 129 patients in their study had portal vein dilatation of more than 1.3cm. Splenic vein diameter >10mm [Table/Fig-4b] was seen in 46.9% cases of PHT, similar to the results of Bolondi L et al., [10] where it was seen in 49% cases.

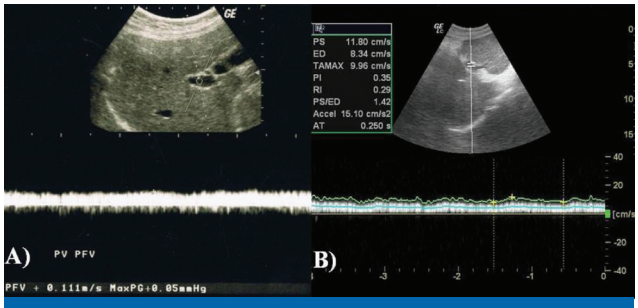
PPV was evaluated from hilar segment of portal vein in all the cases [11]. Tomic D et al., [12] and Iwao T et al., [13] suggested that decrease of the portal blood flow velocity below 12cm/s is a reliable indicator of the portal hypertension. In our study, peak PVV was observed to be 14.2cm/sec and 12.3cm/sec in patients of CLD only and CLD with PHT respectively, thus,



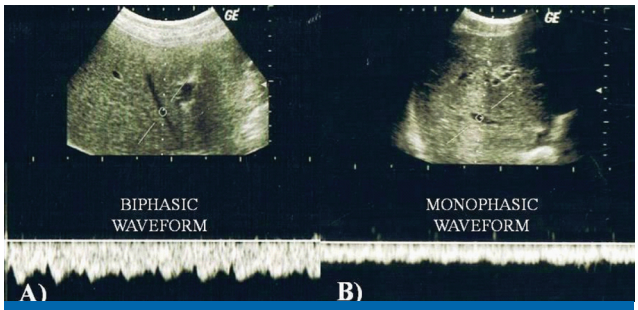
[Table/Fig-4a,4b]: (a) Ultrasound images showing increased Portal vein and (b) Splenic vein diameter in cases of CLD with PHT.

confirming with previously described studies [Table/Fig-5a,5b]. Kuo CH et al., [14] also concluded that decreased portal vein velocity may reflect the severity of clinical portal hypertension in cirrhotic patients and could be a prognostic factor in cirrhotic patients.

Hepatic vein waveform on Doppler USG has been categorised in to 3 groups by Bolondi L et al., [15] HV0, HV1, and HV2. HV0 represents normal triphasic waveform consisting of two negative waves and one positive wave. HV1 and HV2 represent abnormal waveforms (without the reversed phase). In our study of 50 patients, hepatic vein waveform was evaluated in right and middle hepatic vein, about 3-6 cm from the confluence of the hepatic vein and inferior vena cava [11]. Abnormal hepatic vein waveform (HV1 and HV2) [Table/Fig-6a,6b] was seen in 74% patients while the rest 26% patients

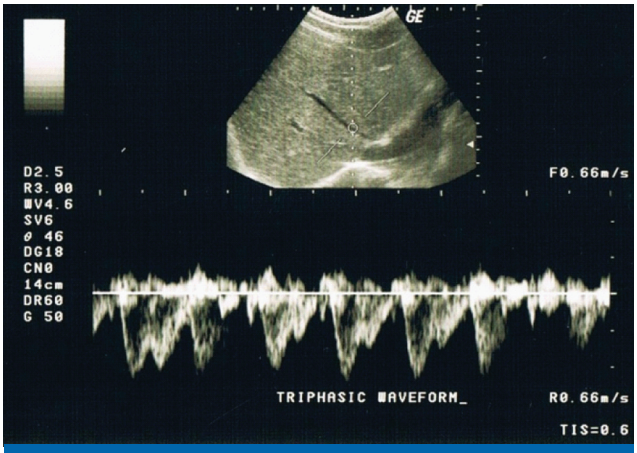


[Table/Fig-5a,5b]: Doppler ultrasound images showing significantly reduced peak portal vein velocity in cases of CLD with PHT.



[Table/Fig-6a,6b]: Doppler ultrasound images showing HV1 and HV2 waveforms in hepatic vein in cases of CLD.

showed normal triphasic wave forms [Table/Fig-7]. Our findings are consistent with the observations of Colli A et al., [16] who observed abnormal hepatic vein waveforms in 75% cases of cirrhosis. Similarly, according to other few studies, flattening of the hepatic waveform can be used as diagnostic tool for chronic parenchymal liver disease [15-17]. According to Bolondi L et al., [15], the mechanism of the change in the HV waveform may be related to liver fibrosis, which progressively reduces phasic oscillation in HVs. But according to a recent study by KCS et al., [18], HV waveforms are independent of liver functions and the changes appearing in cirrhotic livers is presumably due to the change in hepatic hemodynamics. So, changes in hepatic blood flow with progressive degree of cirrhosis alter the HV waveforms on Doppler evaluation.



[Table/Fig-7]: Doppler ultrasound image showing normal triphasic waveform in hepatic vein.

The hepatic artery (HA) hemodynamics is also altered in cases of cirrhosis. HA shows increased resistance in chronic liver disease and cirrhosis [19]. Normal values are < 0.66 for the HA resistance index and <1.1 on the pulsatility index [19].

LIMITATIONS

One of the limitations was that the diagnosis of portal hypertension was made entirely on the basis upper GI endoscopy findings only. In no case, hepatic venous pressure gradient was calculated. Also in some cases, diagnosis of cirrhosis was based on the combination of clinical and laboratory findings and this could lead to excluding patients with early disease and those with atypical findings.

CONCLUSION

Doppler USG is an excellent modality for evaluating the flow hemodynamics in portal and hepatic circulation and can be used for non-invasive diagnosis in suspected cases of CLD and PHT with great reliability. It can be reliably used to distinguish patients of CLD with and without PHT and also aids in long-term management and follow up of patients with CLD.

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