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Role of Colour Doppler in Diagnosis of Portal Hypertension Among Cirrhotic Patients

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Abstract: ***Introduction: Portal hypertension is one of the most important causes of morbidity and mortality in cirrhotic patients. A colour Doppler evaluation has proven utility in the prediction of portal hypertension. Therefore, we aimed to determine the role of colour doppler in the diagnosis of Portal Hypertension among cirrhotic patients. Aim: To determine the role of colour doppler in the diagnosis of Portal Hypertension among cirrhotic patients. Methodology: In this cross - sectional study, sixty patients with a history of chronic alcoholism, features of cirrhosis and haematemesis and Melena were included in the study. A detailed clinico - demographical parameters were recorded. Liver and spleen were measured along with portal vein parameters. Modified child Pugh classification was used to classify the patient with respect to the severity of the liver disease. Results: Of 60 patients, most of the patients were males (73.3%) with a presenting symptom of volume shift (90%).93.3% of the patients had irregular liver surfaces observed on USG.81.7% had collaterals, and 46.7% of them were splenorenal. The majority of the patients had splenomegaly (78.3%). Portal vein parameters showed that most of the patients had portal vein diameter of >13mm, Hepatopetal direction, <12 cm/sec velocity, and congestion index of >0.1. Hepatic Vein Waveform was Biphasic in 40%, monophasic in 26.7%, and Triphasic in 11.7%. According to the child Pugh classification, 48.3% of the patients belonged to Grade A, 31.7% to Grade B and 20% to Grade C classification. A significant difference was noted in a hepatic artery (p<0.001*), splenic artery (p=0.024*), splenic vein diameter (p=0.042*) and spleen size (p=0.046*) with the severity of Cirrhosis. Conclusion: From the study, it can be concluded that Doppler Sonography is a reliable, non - invasive, and rapid diagnostic technique in portal hypertensive patients.***

Keywords: Portal Hypertension, Colour Doppler USG, Child - Pugh, Cirrhosis

1. Introduction

Portal hypertension is one of the common medical problems in India. It may remain silent in early stages or manifest as a dramatic, life - threatening emergency in the form of bleeding oesophagal varices and hence assume clinical significance. Untreated patients surviving a variceal haemorrhage have a risk of rebleeding within 1 - to 2 years and a mortality rate of about 40% to 50%. [1] Portal hypertensive patients can be investigated with colour doppler ultrasonography, upper gastrointestinal endoscopy, computed tomography and magnetic resonance imaging. Endoscopy is the most accurate procedure for evaluating varies but is somewhat inconvenient for patients and requires sedation with benzodiazepines which can significantly exaggerate already existing hepatic encephalopathy. [2, 3] CT plays a vital role in patients with suspicion of portal venous obstruction with doubtful Doppler results. The main limitation of CT is its inability to demonstrate the direction of blood flow. Magnetic Resonance Imaging (MRI) has now challenged sonography, as it is the only other non - invasive test requiring no injection of contrast for the visualization of blood vessels. The common cause of death in portal hypertension is due to life - threatening haemorrhage from ruptured varices and liver failure. [4] Colour Doppler sonography can predict the early recurrence of Esophageal varices, after eradication of varices by endoscopic sclerotherapy or endoscopic variceal ligation, proving to be useful in the follow - up of such patients. Hence this study was conducted to determine the role of colour doppler in the diagnosis of Portal Hypertension among cases of suspected portal hypertension at our territory care centre.

2. Material and Methods

This cross - sectional study was conducted at the Department of Radiodiagnosis, Al - Ameen medical college and Hospital, from November 2020 to October 2022. After obtaining ethical clearance and informed consent, sixty patients were included in the study. Pregnant women, the patient presenting with trauma and those who were unwilling to undergo ultrasound scans were excluded. A pre - structured proforma was used for the collection of clinical data. A high - resolution Duplex Doppler sonography study using GE LOGIQ P9 ultrasound machines with 5 - 13MHz linear transducer was done. History was taken from the patients focusing on risk factors, signs, and symptoms of the hepatocellular disease. Previous medical history relating to hepatocellular disease was noted. First grey scale parameters were recorded, such as liver and spleen size, shape, surface and echotexture with the presence or absence of ascites. Vascular parameters were diameter, patency, phasicity, velocity, flow direction, congestion index, collaterals presence or absence, and spectral widening. Criteria included to diagnose portal hypertensionwere: Portal vein diameter > 13mm, partially patent or complete obliteration of portal vein lumen, Loss of cardiac and respiratory phasicity, Velocity <12cm/sec, markedly pulsatile flow, Absent flow, To &Fro flow or Hepatofugal flow, Congestion index >0.1, Presence of collaterals and Complete portal spectral widening. Modified child Pugh classification was used to classify the patient with respect to the severity of the liver disease. [5, 6] Patient with cirrhosis and portal hypertension were divided into class A, class B & class C as per Child - Pugh classification. Portal vein plurality index and spectral widening were calculated. After the grey scale examination, a colour doppler examination was done.

Statistical Analysis:
Data were entered into Microsoft Excel data sheet and wereanalyzed using SPSS 22 version software. Categorical data was represented in the form of Frequencies and proportions. The chi - square test was used as a test of significance for qualitative data. Continuous data wererepresented as mean and standard deviation. Normality of the continuous data was tested by Kolmogorov–Smirnov test and the Shapiro–Wilk test. ANOVA (Analysis of Variance) was the test of significance to identify the mean difference between more than two groups for quantitative data. A p - value of <0.05 was considered statistically significant after assuming all the rules of statistical tests.

3. Results

The mean age of subjects was 48.90 ± 10.587 years. Majority of subjects were in the age group 41 to 50 years (36.7%), where 73.3% were males and 26.7% were females. Most of the patients were alcoholic (66.7%). Most common symptoms were volume shift, splenomegaly, ascites, jaundice and fever. On USG, the liver surface was irregular in 93.3% and smooth in 6.7%. Other USG findings showed that 81.7% of the patients also had collaterals, and the most prevalent collateral was splenorenal in 46.7%. [Figure - 1] A large number of patients had splenomegaly (78.3%). [Figure - 2] Portal vein parameters showed that the congestion index was >0.1 in 78.3% of the patients. [Table - 1] Hepatic Vein Waveform was Biphasic in 40%, monophasic in 26.7%, and Triphasic in 11.7%. [Figure - 3] In our study, 80% had Hepatopetal flow. [Figure - 4] According to the child Pugh classification, 48.3% belonged to Grade A, 31.7% to Grade B and 20% to Grade C. [Table - 2] Mean Hepatic artery PI in Grade A was 0.702 ± 0.170, in Grade B was 1.006 ± 0.267 and in Grade C was 1.5000 ± 0.361. [Table - 3] Mean Splenic artery RI in Grade A was 0.750 ± 0.165, in Grade B was 0.668 ± 0.218 and in Grade C was 0.5817 ± 0.136. [Table - 4] Mean Splenic Vein diameter in Grade A was 9.86 ± 3.971 mm, in Grade B, it was 11.68 ± 2.926 mm and in Grade C was 12.42 ± 0.669. [Table - 5] Mean Spleen size in Grade A was 14.69 ± 2.647 cm. In Grade B, it was 15.63±2.166 and in Grade C was 16.67±1.497 cm. [Table - 6] A significant difference was noted in the hepatic artery (p<0.001*), splenic artery (p=0.024*), splenic vein diameter (p=0.042*) and spleen size (p=0.046*) with the severity of Cirrhosis.

4. Discussion

Alcoholic cirrhosis was found to be the leading cause of portal hypertension in our study, contributing to 66.7, followed by hepatitis - induced cirrhosis. In Western countries, cirrhosis of the liver accounts for more than 90% of cases of portal hypertension. [7, 8]In our study, Portal hypertension was predominantly seen in Males, which can also be explained by the causal relationship of alcoholism with portal hypertension. Portal vein diameter of > 13 mm was found in 58.3%, while a diameter <13 mm was found in 41.7%. Similar findings were reported by **Weinreb et al.** [9], **Bolondi et al.** [10], **Ditchfield et al.** [11]. The reason for the low sensitivity of portal vein caliberis that collateral formation decompresses the portal vein, which causes a reduction in its size. [10]In our study, we had 81.7% of cases which showed a diameter change of less than 20%. Our study correlates with other studies. [10, 12 - 14] In addition, this clearly states that caliber variation with respiration is a more sensitive finding of portal hypertension as compared to increased portal vein diameter which was found in only 58.3% patients in our study. **Ditchfield MR et al.** [11]found that sonographically only 52% of their patients had large spleen. They concluded that splenomegaly is an insensitive sign of portal hypertension and that the degree of splenic enlargement had no correlation to the severity of portal hypertension. On the other hand, **Berzigotti et al.,** found that not only was splenomegaly the most common and sensitive sign of portal hypertension, but it also is an independent predictor of esophageal varices, [15] which was also supported by **Talwalkar et al.** [16] In our study, we had 78.3% cases showing splenomegaly which makes it a very common finding in portal hypertension. Mean Spleen size in Grade A was 14.69 ± 2.647 cm, in Grade B, was 15.63±2.166 and in Grade C was 16.67±1.497 cm. There was a significant increase in Spleen size with respect to Child Pugh Class. In our study, 80% had Hepatopetal flow, 5% had To and Fro flow, 3.3% had Hepatofugal Flow, and 11.7% had Absent Flow. The low incidence of hepatofugal flow in our study could be explained by a low incidence of hepatofugal flow (1 - 8%) in portal hypertensive patients. [17]The finding of a reversed flow in the portal venous system is important forunderstanding the clinical picture of a cirrhotic patient since hepatic encephalopathy may be explained based on large hepatofugal collaterals. Portal hypertension leads to pathological fibrotic changes in the liver, which decreases the transmission of right atrial pressure changes through the hepatic veins, eventually causing a decrease in the subtle pulsatility of portal vein waveform. In fact, this finding occurs earlier than PV diameter change as per study by Barakat. [18]**Ditchfield et al.** [11] found that reversed flow in portal veins was seen in 3.4 – 5.3% cases. **Alexandra von et al.** and **others**reported that the direction of portal vein flow was normal in 73%, hepatofugal in 9% and bidirectional in 6% patients. [19, 20]These studies showed that the prevalence of hepatofugal flow in portal hypertension varies between 3 % to 23%. Differences between series were attributed to the differences in the severity of the disease and to whether the flow was evaluated only in the main portal vein or its main tributaries and branches as well.3.3% of our study showed hepatofugal flow, which is concordant with the previous studies. The mean portal vein velocity (PVV) in cirrhotic patients is relatively low compared with that in healthy subjects because of increased intrahepatic vascular resistance. **Zironi**and **colleagues** reported that the cut - off value of 15 cm/s showed a sensitivity and specificity of 88 and 96%, respectively. However, as portal hypertensive patients have various portosystemic shunts, which leads to decompression of the congestion, this group may have PVV in a normal to high range, like normal subjects. [21] In our study, more than 55 % of cases had a velocity greater than 12cm/sec. This shows that this parameter is not very reliable in the identification of portal hypertensive patients. In our study, congestive index of more than 0.1 was seen in 78%. These findings are similar to the findings of **Moriyasu F et al**. [22] They showed that there was a statistically significant difference between congestive indices from the normal subject group and indices obtained from patients with portal

hypertension. It was also seen that CI was high in patients with low portal venous velocity, with is self - explanatory. In our study, 11 patients had no collaterals. In the remaining 49 patients, the most common collateral was the splenorenal collateral which was present 46.7%. Studies have shown that large spontaneous splenorenal shunts are usually not associated with gastrointestinal bleeding due to spontaneous decompression of portal pressure. [21, 23]Gallbladder varices developed as venous collateral because of extrahepatic portal vein occlusion in these patients. Gall bladder (GB) varices were seen in 18.3% patients in our study. [16, 24]Hepatic waveforms were difficult to obtain in portal hypertensive patients due to ascites and non - cooperation during breath holding. In the present study, 40% showed a biphasic waveform, 26.79% showed a monophasic waveform and 11.7% showed a triphasic waveform. [25]**Barkat M et al.,** calculated doppler signals from the main portal vein of 36 healthy adults and 52 cirrhotic patients with portal hypertension and graded the severity of liver disease using modified Child - Pugh classification. The mean pulsatility index value in the control group was 0.37+0.10, and in cirrhotic patients was 0.17+0.03. The difference between the control and cirrhosis group, as well as the difference within different Child classes, were statistically significant ($p<0.05$). None of the patients in the control group had complete spectral widening, while 76.92% of cirrhotic patients had complete spectral widening (28.5% of Child A, 66.6% of Child B and 100% of Child C). [18] In the present study, 48.3% had Grade A, 31.7% had Grade B, and 20% had Grade C Child - Pugh classification. Splenic artery RI (SARI) is potentially an excellent non - invasive measurement method for diagnosing clinically significant portal hypertension (CSPH), especially those without splenomegaly. In our study, mean Splenic artery RI in Grade A was 0.750 ± 0.165, in Grade B was 0.668 ± 0.218 and in Grade C was 0.5817 ± 0.136. There was a significant decrease in Splenic artery RI with respect to Child Pugh Class. In our study, mean splenic vein diameter in Grade A was 9.86 ± 3.971 mm, in Grade B was 11.68 ± 2.926 mm and in Grade C was 12.42 ± 0.669. There was a significant increase in Splenic Vein diameter with respect to Child - Pugh Class. In cirrhotic patients, due to portal outflow obstruction (i. e., elevated intrahepatic portal vascular resistance) and increasing pressure of the portal venous system, the diameters of the PV and SV may initially enlarge. When the diameters of the PV and SV dilate to a peak point with a concomitant increase of the portal venous system pressure, the common collaterals (esophageal and gastric fundic varices) send blood flow from their originating veins to the collaterals, which, in turn, results in a decrease in diameter. [26]Because of the significant difference in SV diameter between patients with and without esophageal and gastric fundic varices, the SV diameter measurements can be used as criteria to predict the presence of varices. [27]Studies have proved the high reliability and accuracy of ultrasound in the measurement of the diameter of the portal vein and the size of the spleen. The size of the spleen may provide information on the diagnosis and prognosis of disease courses. Spleen size correlates positively with Portal vein diameter in Cirrhosis subjects. With the increase in Portal Vein diameter, there was an increase in Spleen size. **Zaman S et al.,** observed similar findings of an increase in spleen size with increase in portal vein diameter among cirrhotic patients. [28] **O'Donohue et al.,** Observed that the mean spleen size was 16.0 cm in patients with chronic liver parenchymal disease, which is significantly larger than in normal individuals. [29] The mean portal vein diameter in cirrhotic patients was 10.8 mm. They concluded that spleen size and portal vein diameter increase with liver congestion. Subash **Bhattarai, et al.** observed mean portal vein diameter of 10.800 mm among subjects without varices, and 13.731 mm subjects with varices. [30] Mean spleen size of patients with no varices was 12.67 cm and 15.367 cm in participant with varices. There was 92.72 % sensitivity and 90 % specificity for the portal vein diameter of 12.25 mm, and 94.5 % sensitivity, 75% specificity for the spleen size of 13.9 cm, to predict gastro - esophageal varices.

5. Conclusion

From the study it can be concluded that Doppler Sonography is a reliable, non - invasive, and rapid diagnostic technique in portal hypertensive patients. Greyscale findings, together with colour Doppler characteristics, help in the qualitative as well as quantitative evaluation of the portal venous system. Colour Doppler ultrasonography is an excellent non - invasive investigation tool which shows various spectrums of findings, flow metric changes and collaterals accurately in portal hypertension.

Conflict of Interest - All authors declare no conflict of interest.

Source of Funding - None

Consent: As per international standards or university standards written participant consent has been collected and preserved by the authors.

Ethical Approval: As per international standards or university standards written ethical permission has been collected and preserved by the author (s).

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Tables and Figures

Table 1: Clinico - demographic parameters ofenrolled patients (n=60)

Demographic parameters			Count	%
Age	30 to 40 years		15	25.00%
	41 to 50 years		22	36.70%
	51 to 60 years		14	23.30%
	61 to 70 years		9	15.00%
Gender	Female		16	26.70%
	Male		44	73.30%
	Total		60	100.00%
Etiology	Alcohol		40	66.70%
	Hepatitis		19	31.70%
Signs and symptoms	Ascites		44	73.30%
	Fever		14	23.30%
	Jaundice		40	66.70%
	Splenomegaly		49	81.70%
	Volume shift		54	90%
USG and Doppler Findings	Liver surface Irregular		56	93.30%
	Liver surface Smooth		4	6.70%
	Fatty Liver		23	38.30%
	Spectral Widening		39	65.00%
	Portal Vein Thrombosis		2	3.30%
	Collaterals		49	81.70%
Portal vein	Portal Vein	>13mm	35	58.30%

parameters	Diameter	≤13mm	25	41.70%
	Portal Vein Direction	Absent	2	3.30%
		Hepatopetal	53	88.30%
		Hepatofugal	1	1.70%
		To &Fro	4	6.70%
	Portal Vein Velocity	<12 cm/sec	33	55.00%
		>12 m/sec	27	45.00%
	Portal Vein Phasicity	<20% increase in diameter	49	81.70%
		>20% increase in diameter	11	18.30%
	Congestion Index	>0.1	47	78.30%
		<0.1	13	21.70%

Table 2: Child Pugh Class distribution

		Count	%
Child Pugh Class	Grade A	29	48.3%
	Grade B	19	31.7%
	Grade C	12	20.0%
	Total	60	100.0%

Table 3: Hepatic artery PI with respect to Child Pugh Class

Hepatic artery PI	N	Mean	SD	95% Confidence Interval for Mean		Minimum	Maximum	p value
				Lower Bound	Upper Bound			
Grade A	29	0.7021	0.17047	0.6372	0.7669	0.50	1.10	**F = 43.99, p <0.001***
Grade B	19	1.0068	0.26767	0.8778	1.1359	0.50	1.40	
Grade C	12	1.5000	0.36181	1.2701	1.7299	0.80	1.80	
Total	60	0.9582	0.39086	0.8572	1.0591	0.50	1.80	

Table 4: Splenic artery RI with respect to Child Pugh Class

Splenic artery RI	N	Mean	SD	95% Confidence Interval for Mean		Minimum	Maximum	p value
				Lower Bound	Upper Bound			
Grade A	29	0.7507	0.16540	0.6878	0.8136	0.40	0.98	**F = 3.984, p = 0.024***
Grade B	19	0.6689	0.21876	0.5635	0.7744	0.40	0.98	
Grade C	12	0.5817	0.13624	0.4951	0.6682	0.46	0.96	
Total	60	0.6910	0.18810	0.6424	0.7396	0.40	0.98	

Table 5: Splenic Vein diameter with respect to Child Pugh Class

Splenic Vein diameter (mm)	N	Mean	SD	95% Confidence Interval for Mean		Minimum	Maximum	**p value**
				Lower Bound	Upper Bound			
Grade A	29	9.86	3.971	8.35	11.37	3	18	**F = 3.340, p = 0.042***
Grade B	19	11.68	2.926	10.27	13.09	5	17	
Grade C	12	12.42	0.669	11.99	12.84	12	14	
Total	60	10.95	3.372	10.08	11.82	3	18	

Table 6: Spleen size with respect to Child Pugh Class.

Spleen size	N	Mean	SD	95% Confidence Interval for Mean		Minimum	Maximum	p value
				Lower Bound	Upper Bound			
Grade A	29	14.69	2.647	13.68	15.70	9	18	**F =3.257, p = 0.046***
Grade B	19	15.63	2.166	14.59	16.68	11	18	
Grade C	12	16.67	1.497	15.72	17.62	14	18	
Total	60	15.38	2.401	14.76	16.00	9	18	

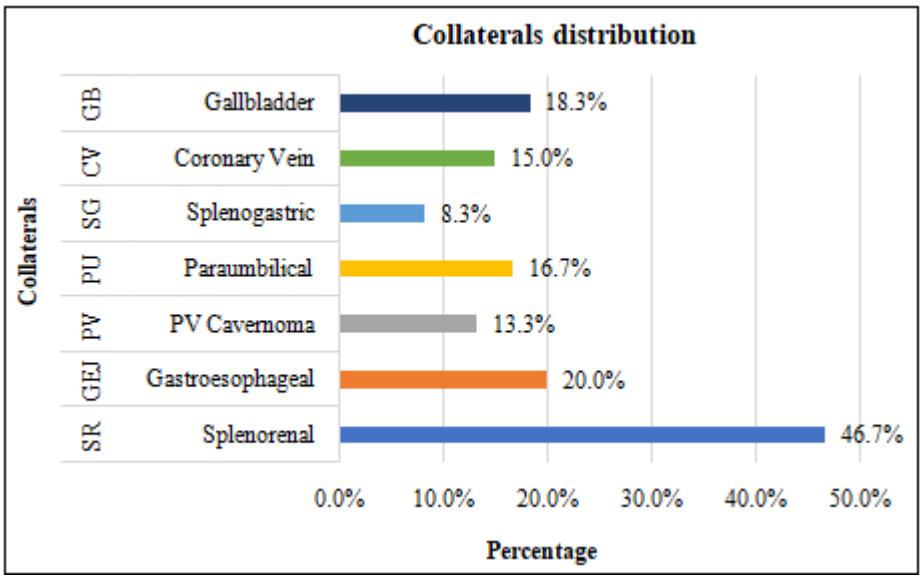

Figure 1: Collaterals distribution among enrolled patients

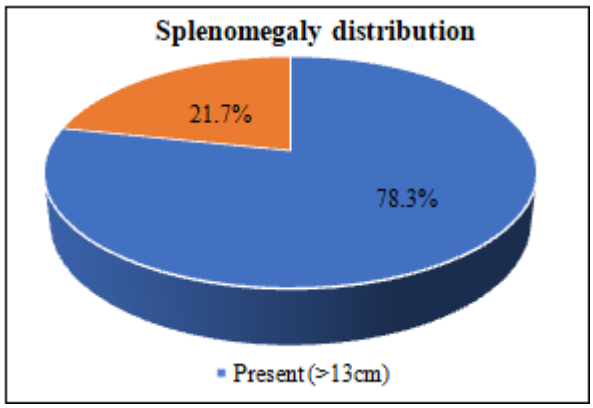

Figure 2: Splenomegaly distributionamong enrolled patients

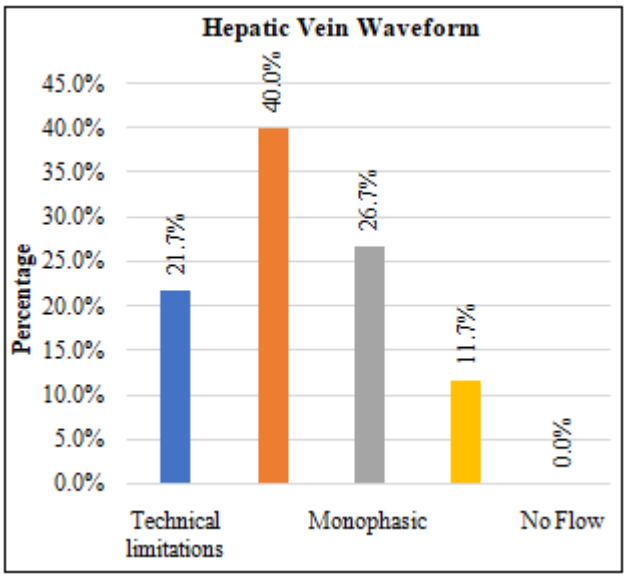

Figure 3: Hepatic Vein Waveformamong enrolled patients

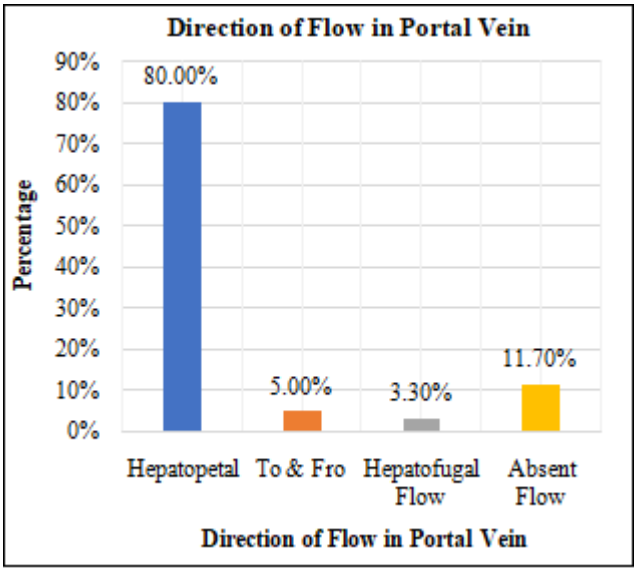

Figure 4: Direction of Flow in Portal Veinamong enrolled patients