Original Research Article

A study of incidence of doppler criteria for ultrasound diagnosis of portal hypertension in cirrhosis

Uttareshvar M Dhumal[1*], Sudam Mehatre[2]

[1]Associate Professor, [2]Assistant Professor, Department of Radiology, MIMSR Medical College Latur, INDIA.
Email: opglakshmi@gmail.com

Abstract

Background: Portal hypertension is defined as an increase in pressure gradient between the portal vein and the hepatic veins or IVC (Bosch, 1992). It represents an increase of the hydrostatic pressure within the portal vein or its tributaries. **Aims and Objectives:** To study of incidence of Doppler criteria for Ultrasound diagnosis of portal hypertension in Cirrhosis. **Methodology:** The cases selected for the present study were the patients from the MIMSR Medical College, Latur, during the period from November 2018 to October 2019. All patients were scanned at Department of Radiology, MIMSR Medical College, Latur. The healthy control population (25 controls) was also selected randomly with respect to particular age group and included 13 men and 12 women with an age range of 6-70 years (mean ± standard deviation (SD): 35 ± 17 years). The statistical analysis was done by Epi-Info Version 6.04d. The tests which were used were χ^2 and standard error of mean (SE). **Result:** Out of total cases 15 cases (31.25%) showed the portal vein diameter of above 16 mm. the sensitivity is thus 31.25% and specificity 100%. In patients with Portal Vein Thrombosis the Hepatic Arterial Resistive Index was significantly higher (0.821±0.67) as compared to the patients without portal vein thrombosis and as compared to the controls (0.624±0.071). In patients with patent Portal Vein Thrombosis the oesophageal varices were seen in 6 patients out of total of 15. In patients with portal vein thrombosis (15) the Gall Bladder Varices were present in 11 (73.33%) patients. The Portal Vein Cavernoma (PVC) was found in 6 (12.85%) patients with portal hypertension. Of the six patients with Portal Vein Cavernoma four patients (66.66%) were less than 20 years of age. In majority of the eases the Paraumbilical Vein was massively enlarged. Suggesting that, the patients present late in the course of the illness. All patients with Patent Paraumbilical Vein had Portal Hypertension. The mean portal venous flow velocity (12.341 ± 6.481 cm/s) and the flow volume (2.239 ± 1.445 l/min) were significantly higher in patients with paraumbilical vein patency. **Conclusion:** Portal vein diameter, Portal Vein Thrombosis the Hepatic Arterial Resistive Index, Paraumbilical Vein, Patent Paraumbilical etc. parameters were very important in the diagnosis of portal hypertension.

Keywords: Portal hypertension, Portal vein diameter, Portal Vein Thrombosis the Hepatic Arterial Resistive Index.

[*]**Address for Correspondence:**
Dr Uttareshvar M Dhumal, Associate Professor, Department of Radiology, MIMSR Medical College Latur, INDIA.
Email: opglakshmi@gmail.com

Received Date: 06/11/2020 Revised Date: 13/12/2020 Accepted Date: 08/01/2021
DOI: https://doi.org/10.26611/10131738

Access this article online

Quick Response Code:

Website: www.medpulse.in

Accessed Date: 27 March 2021

INTRODUCTION

In cirrhotics with portal hypertension, portal venous system has the features of elevated vascular resistance and hyperdynamics, and the latter mainly results from increased blood flow in SV. Esophageal variceal bleeding (EVB) score may become a valuable parameter in predicting occurrence of EVB.[6] In a study including 1123 patients without varices when first diagnosed, it was reported a 10-year cumulative incidence of varices of 44%[1]. Improvement in liver function and abstinence from alcohol may result in a decrease or even disappearance of varices[2]. Esophageal varices have been observed only in patients with portal pressure gradient (the difference between portal pressure and inferior venacava pressure, HVPG) above

How to cite this article: Uttareshvar M Dhumal, Sudam Mehatre. A study of incidence of doppler criteria for ultrasound diagnosis of portal hypertension in cirrhosis. *MedPulse International Journal of Radiology.* March 2021; 17(3): 78-84.
http://www.medpulse.in/Radio%20Diagnosis/

the threshold value of 10 mmHg (i.e. more than double the normal value). However, not all patients with HVPG above this level have esophageal varices[3]. Once varices develop, they tend to increase in size before they eventually rupture and bleed. Increase in the size of varices from "small" to "medium" or "large" occurs in 10-20%, 1-2 years after their first observation[4, 5 and 6] and is related to the severity of liver disease. The risk of bleeding increases with worsening liver function, being approx. 4/100 patients per year in Child - Pugh Class A patients and 8/100 in Class B or C and significantly lower in patients without, than in those with ascites independent of the variceal size[1]. Variceal bleeding occurs only in patients with HVGP above or equal to the threshold value of 12 mmHg[3] with a higher risk in patients with higher baseline HVPG.

When esophageal varices are discovered, they are graded according to their size, as follows:

Grade 1: Small straight varices.

Grade 2: Enlarged tortuous varices occupying less than one third of the lumen.

Grade 3: Large coil-shaped-varices occupying more than one third of the lumen.

Acute Variceal Bleeding: It has been suggested that the-risk for prevention of rebleeding is higher in patients in Child-Pugh class C than in those in class A or B. Continued alcohol abuse and hepatocellular carcinoma are also associated with higher incidence of rebleeding.[3]

Ascites

Reliable indicators of the risk of developing ascites have not been identified. Age over 63, albumin <3.4 g/dl, prothrombin time <63% and platelet <102x10^9/L have been recently found to be related to the development of overt decompensation of cirrhosis by invariable analysis but their prognostic role way not confirmed in a multivariable model. Once developed, ascites marks the last stage of the disease. The median survival time after appearance of ascites is consistently reported to be between 1 and 2 years[1, 13 and14], whereas the mortality while free of ascites is 2% per year in a study after more than 10 years of follow-up[1].

Portal Hypertensive Gastropathy (PHG)

Patients with severe liver dysfunction and large esophageal varices are at higher risk of developing PHG[1]; whereas large fundal varices may have a protective role, particularly when they are associated with spontaneous gastro-renal shunt. β-blockers significantly reduce the rebleeding risk in patients who bled from PHG[7].

The Outcome of Patients with Portal Hypertension: This classification was obtained by combining data from two prospective studies of the natural history of cirrhosis, including a total of 1649 patients[1].

Stage-0 : It is characterized by the absence of esophageal varices and ascites. In this stage, i.e. in the varices and ascites-free period, the mortality rate is as low as 1% per year.

Stage-1: It is characterized by the presence of esophageal varices without ascites and without bleeding. At this stage, the mortality rate is 4% per year.

Stage-2: It is characterized by ascites and esophageal varices without bleeding. The mortality rate is 12.6% per year.

Stage-3: It is characterized by ascites and a previous bleed. The mortality rate is 15 per year. As the incidence portal hypertension is very common among the patients with liver disease and ultra-sonography is very important in the diagnosis of it so we have studied incidence of Doppler criteria for Ultrasound diagnosis of portal hypertension in Cirrhosis

METHODOLOGY

The cases selected for the present study were the patients from the Department of Radiology, MIMSR Medical College, Latur, during the period from November 2018 to October 2019. All patients were scanned at Department of Radiology, MIMSR Medical College, Latur. No selection bias was exercised in terms of patient age, sex or pathology. The study included 50 patients with Portal Hypertension (PHT); 25 men and 25 women with an age range of 5-75 years (mean $\pm$ standard deviation (SD): 37$\pm$17 years). The healthy control population (25 controls) was also selected randomly with respect to particular age group and included 13 men and 12 women with an age range of 6-70 years (mean $\pm$ standard deviation (SD): 35 $\pm$ 17 years). Portal hypertension was considered to be present when any of the following previously demonstrated to be specific for portal hypertension, were present: a dilated portal vein, collateral vessels, abnormal flow in portal vein, an enlarged and/or patent para-umbilical vein, portal vein obstruction or hepatofugal flow in the portal vein. The diagnosis of PHT was based on a combination of clinical data, e.g. jaundice, ascites, muscle wasting, cutaneous spider angiomas, ecchymosis, palmar erythema and flapping tremors, laboratory data e.g. decreased serum albumin and prolonged prothrombin time and ultrasound data e.g. coarsened bright liver echopattern and nodular liver surface, endoscopic varices in addition to liver biopsy whenever possible. Doppler ultrasound examination was performed for all patients and healthy adults using (TOSHIBA Nemio-30 and TOSHIBA Just Vision 400 US Scanner) a sector transducer operating at 3.5 MHz and high-resolution superficial probe operating at 7.5 MHz. The Doppler angle (between the axis of the Doppler beam and that of the vein examined) was always < 60^o. The sample volume was adjusted to include as much of the lumen as possible without including the vessel wall. All examinations were performed on fasting subjects and during suspended expiration. The Portal Vein flow waveform was recorded at a point midway between the

confluence of the splenic and superior mesenteric veins and the bifurcation of the portal vein with the transducer oriented along the longitudinal axis of the main portal vein through a paramedian or slightly oblique plane, where the portal vein crosses the inferior vena cava. The portal vein waveform was described as PV_o when the wave was slightly pulsatile (i.e. showing variation of the peak velocity with time along the wave envelope), or PV_1 when the wave envelope was almost flat. The Main Portal Vein and Right Branch of Portal Vein and its branches are best studied through a right intercostal approach. The Left Branches of Portal Vein, its branches and the hepatic veins are best seen through an oblique subcostal approach.

Statistical analysis: The statistical analysis was done by Epi-Info Version 6.04d. The tests which were used were χ^2 and standard error of mean (SE). Probability values (*p-Value*) < 0.05 were considered significant.

RESULT

Table 1: Age distribution in Cases and Controls

Age Distribution	Cases				Controls			
	Male		Females		Male		Females	
	N	%	N	%	N	%	N	%
1-15	4	16	2	8	2	15.4	2	16.6
16-30	6	24	6	24	4	30.8	2	16.6
31-45	8	32	11	44	5	38.5	4	33.3
46-60	4	16	3	12	2	15.4	3	25
> 60	3	12	3	12	1	7.7	1	8.3
Total	**25**		**25**		**13**		**12**	
	X2- 1.28, DF- 4, p- Value- 0.864				**X^2- 1.74, DF- 4, p- Value- 0.7833**			

The age distribution in cases was matched between the males and females and with the number of cases in each group to avoid selection bias. The age distribution in controls was matched between the males and females and with the number of cases in each group to avoid selection bias. The p- Value in the cases was found to be 0.864 i.e. not significant. The p- Value in the cases was found to be 0.7833 i.e. not significant. Non- Significant p- Value suggests that there is no selection bias exercised.

Table 2: Mean diameter of Portal Vein in Cases and Controls

Portal Vein	Cases			Controls		
	No.	%	Mean ±SD	No.	%	Mean ±SD
8-10	6	12.5	8.198±1.33	7	28	9.357±0.683
10-12	5	10.42	10.92±0.61	15	60	11.187±0.567
12-14	9	18.75	13.067±0.642	1	4	14.1±0
16-18	3	6.25	16.8±0.5	0	0	-
>18	12	25	21.541±2.012	0	0	-
Not Applicable	2	4.16				-
Overall	**50**			**25**		-

The mean diameter of portal vein was higher in-patient population than in controls. Out of total cases 15 cases (31.25%) showed the portal vein diameter of above 16 mm. the sensitivity is thus 31.25% and specificity 100%. The sensitivity of the test can be increased to 58.33% if we take the cut off value to be 14 mm, but then we will have to compromise on the specificity. In the patient population 47 (94%) patient had loss of respiratory variation. Thus suggesting very high sensitivity of this finding in the prediction of Portal Hypertension. All the control population had respiratory variation present (> 20% increase in diameter during deep inspiration from the expiratory diameter).

Table 3: Incidence of Portal Vein Thrombosis in Cases and Controls

Portal Vein Thrombosis	Cases (N=50)	Controls (N=25)
Present	15	0
Absent	35	25

The Portal Vein Thrombosis (PVT), whether partial or complete, was present in fourteen cases (30%) with portal hypertension. In patients with Portal Vein Thrombosis the Hepatic Arterial Resistive Index was significantly higher (0.821±0.67) as compared to the patients without portal vein thrombosis and as compared to the controls (0.624±0.071). In patients with patent Portal Vein Thrombosis the oesophageal varices were seen in 6 patients out of total of 15. In patients with portal vein thrombosis (15) the Gall Bladder Varices were present in 11 (73.33%) patients. Thus a high incidence of GB Varices is seen in patients with Portal Vein thrombosis. All the Cases were of Benign Thrombus. No Case of Malignant Thrombus was found in our study.

Table 4: Incidence of Portal Vein Cavernoma in Cases and Controls

Portal Vein Cavernoma	Cases (N=50)	Controls (N=25)
Present	6 (12.85%)	0
Absent	43	25

The Portal Vein Cavernoma (PVC) was found in 6 (12.85%) patients with portal hypertension. Of the six patients with Portal Vein Cavernoma four patients (66.66%) were less than 20 years of age. The splenomegaly was present in five (83.33%) patients. One patient had been splenectomized earlier. Gall Bladder Varices were present in two (33.33%) out of seven patients.

Table 5: Incidence of Recanalized Para Umbilical Vein in Cases and Controls

Para Umbilical Vein (mm)	Cases		Controls	
	N	%	Mean±SD	N
<1	00	-	-	0
1-2	02	04	1.4±0.566	0
2-3	03	06	2.333±0.306	0
3-5	03	06	3.632±0.584	0
>5	13	26	7.756±2.901	0
Not - Visualized	29	58	-	25

In patients with portal hypertension the enlarged Paraumbilical Vein was visualized in 21 (42%) out of 50 cases. In majority of the eases the Paraumbilical Vein was massively enlarged. Suggesting that, the patients present late in the course of the illness. All patients with Patent Paraumbilical Vein had Portal Hypertension. The mean portal venous flow velocity (12.341±6.481 cm/s) and the flow volume (2.239±1.445 l/min) were significantly higher in patients with paraumbilical vein patency. The mean portal venous flow velocity and the flow volume in patients without paraumbilical shunts were 8.863±7.772 cm/s and 1.467±1.653 l/min. respectively. The prevalence of shunts of the paraumbilical vein in cirrhotics was significantly lower in category Child A (43.33%) than in Child B (66.66%) and Child C (80.00%).

DISCUSSION

In the evaluation of portal hypertension

Transparietal Ultrasonography

Signs of portal hypertension include:

a. Dilatation of the portal vein, which presents a maximal diameter greater than 12.5-13 mm.
b. The attenuation or disappearance of the kinetic wall motion of the SMV and splenic vein, which have a tendency of increasing in diameter during inspiration
c. The presence of portosystemic collaterals.[9,10]

Certain studies have demonstrated that a portal vein diameter greater than 13 mm has a sensitivity of 40% for the detection of portal hypertension while a diameter greater than 15 mm has a sensitivity of only 12%.[11] Thus it is better, to use the kinetic motion of the splenic vein and SMV instead of the portal vein diameter, since these have higher sensitivity and specificity values. In particular if one observes the diminished response of the portal vessels to respiration, this finding presents a sensitivity and specificity for the diagnosis of portal hypertension of 80% and 100% respectively.[9]

At the level of the portal vein itself, a to-and-fro venous flow pattern may be observed.[12] This type of blood flow remains antegrade during inspiration and becomes retrograde or hepatofugal during expiration. This is due to a high peripheral portal resistance which will slow the overall portal venous blood flow and stop or even render it retrograde when the central venous pressure increases. Flow reversal pattern can also be observed in the afferent veins of the portal system. With Doppler imaging techniques, this flow reversal is especially noted in the splenic vein and more rarely, in the SMV[13]. With portal hypertension, the Coronary Vein presents a diameter >6 mm in only 26% of cases, while it will contain a hepatofugal flow in up to 78%.[14] Doppler duplex detection of portosystemic collaterals is also a sensitive sign for the qualitative diagnosis of portal hypertension. While the portal blood flow and portal vein caliber increase after a meal in normal individuals associated with an increased resistive index of the hepatic artery, these values demonstrate little or no modulation in patients with portal hypertension.

The three most frequently visualized collaterals include:

a. The gastroesophageal veins
b. The paraumbilical veins
c. The splenorenal veins.

By taking the ratio of the cross-sectional area of the portal vein to the portal flow velocity, one may obtain the Congestion Index of the portal vein. Since the portal blood flow will decrease with portal hypertension, this index will increase. In the presence of a patent paraumbilical plexus, portal blood flow and resistance will be modified by the hepato - hepatic shunting, thus rendering portal vein velocity and congestion index measurements of limited value. In the presence of fibrotic changes of the hepatic

parenchyma, the peripheral resistance of the hepatic arterial bed will increase and thus render evaluation of arterial flow patterns difficult, this increased fibrosis will also cause a flattening of the hepatic venous flow since the liver will lose part of its elasticity.

Mean diameter of Portal Vein in Cases and Controls

In normal individuals the Portal Vein diameter does not exceed 13 mm in quiet respiration and 16 mm in deep inspiration, as measured where the Portal Vein crosses the IVC.[1-3] A Portal Vein diameter exceeding 13 mm indicates portal hypertension with high degree of specificity (100%) but low sensitivity (45-50%). Sensitivity is increased by evaluating the respiratory variation.[1,2] In normal individuals the diameters of Portal Vein, Splenic Vein and Superior Mesenteric Vein increases by 20-100% from quiet to deep inspiration. An increase of less than 20% indicates Portal Hypertension with sensitivity of 81% and specificity of 100% [2]. In our study the mean diameter of the Portal Vein was higher in-patient population than in controls. Out of total cases 15 cases (31.25%) showed the portal vein diameter of above 16 mm. If we take 16 mm as the cut off the sensitivity of the criteria of diameter of the Portal Vein was found to be low (31.25%) with a specificity of 100%. The sensitivity of the test can be increased to 58.33% if we take the cut off value to be 14mm, but then we will have to compromise on the specificity. Thus the parameter of diameter of Portal Vein was not sensitive enough to diagnose Portal Hypertension but has a high specificity at high values. All the patients except three had loss of respiration (94%). Thus loss of respiratory variation has a very high sensitivity for the prediction of Portal Hypertension. In our study the there is no significant difference between the diameters of the right and the left Portal Veins in Cases. There is however reduction in the diameter of the right and the left Portal Veins with increase in the degree of cirrhosis. thus, reflecting an active process of shrinkage of liver. Nishihara *et al.* 1994[15], reported results supporting the regional differences in portal blood flow. They reported significant decrease in blood flow in the anterior branch of the right Portal Vein in patients compared to normal subjects; however, they did not find significant difference in the posterior branch of right Portal Vein or Main Portal Vein. The results of our study are in accordance with a study by Kutlu R *et al.* 2002.[16] There is no significant difference between the diameters of the right and the left Portal Veins. However, they also found reduction in the diameters of right and left Portal Veins in accordance with increase in the degree of cirrhosis. In a study by Goyal AK *et al.* 1990[17], investigating 100 healthy subjects and 50 patients with portal hypertension, the upper normal limits of portal, splenic and superior mesenteric vein diameters were reported as 16, 12 and 11 mm, respectively and the dimensions above these values provided an overall sensitivity of 72%, an accuracy of 91%, and a specificity of 100% in diagnosing the patients with suspected portal hypertension. The high sensitivity could not be achieved possibly due to the fact that small study sample is taken.

Incidence of Portal Vein Thrombosis in Cases and Controls

Liver cirrhosis is an established cause of PVT and accounts for approximately 11.2%[2]-21.5%[3] of cases. In cirrhosis both benign and malignant portal vein thrombosis develops. The benign portal vein thrombosis occurs because of development of portal hypertension and malignant portal vein thrombosis occurs by direct invasion of portal vein by hepatocelluluar carcinoma. Etiological factors in non-cirrhotic PVT patients are prothrombotic states (i.e. hypercoagulability, myeloproliferative disorders) in 40-70% and local factors (i.e. intraabdominal inflammatory conditions, surgery, cancer) in 10-50%.[3,4] Frequently more than one factor is present. In our study the Portal Vein Thrombosis (PVT), whether partial or complete, was present in fourteen cases (30%) with portal hypertension. In patients with Portal Vein Thrombosis the Hepatic Arterial Resistive Index was significantly higher (0.821 ± 0.67) as compared to the patients without portal vein thrombosis and as compared to the controls (0.624 ± 0.071). In patients with patent Portal Vein Thrombosis the esophageal varices were seen in 6 patients out of total of 15. Thus there was not much correlation with incidence of esophageal varices. In patients with portal vein thrombosis[15], the Gall Bladder Varices were present in 11 (75.33%) patients. Thus a high incidence of GB Varices is seen in patients with Portal Vein Thrombosis. All the Cases were of Benign or Bland Thrombus. No Case of Malignant Thrombus was found in our study. In a study by Kocher G *et al.* 2005[18], analyzing a cohort of 20 noncirrhotic patients with portal vein thrombosis (PVT) seen in a general internal medicine setting with a median age at the time of diagnosis of 50.5 years, with ages ranging from 17-83 years, the causative factors included prothrombotic sates (9 patients, 45%) and/or local factors (5 patients, 25%). Complications from portal hypertension (15 patients, 75%), which was associated with variceal bleeding in 6 patients (30%). Bowel ischaemia (5 patients, 25%) and bowel infarction (2 patients) were less frequent. The most common causes in our study were Cirrhosis and Pancreatitis.

Incidence of Portal Vein Cavernoma in Cases and Controls

Cavernous deformity of the portal vein has been described as an uncommon cause of portal hypertension in children and adults. It can occur either as an idiopathic disorder or after recanalization of the portal vein with development of periportal collateral channels in cases of portal vein obstruction. Such an obstruction can result from many causes e.g., omphalitis, umbilical vein catheterization, pancreatitis, liver cirrhosis, and tumor invasion. Color

Doppler examination can show the vascular nature when these spaces light up instantaneously with color, and further confirmation can be made with the characteristic venous waveform shown on spectral analysis. In our study the Portal Vein Cavernoma was found in 6 (12.85%) patients with portal hypertension. Of the seven patients with PVC four patients (66.66%) were less than 20 years of age. Splenomegaly was present in five (83.33%) of the patients. One patient had been splenectomised earlier. Although the main right and left portal vein branches could not be identified, some smaller intrahepatic portal vein branches were shown in all patients and had normal flow direction inside. Also, the hepatic veins were patent in all, with normal flow direction draining into the inferior vena cava. Abdominal collaterals vessels were found in all of the patients. Gall Bladder Varices were present in two (33.33%) of the seven patients. This high prevalence could be explained by the meticulous examination with color Doppler imaging, which facilitated identification of such collaterals and differentiation of them from other causes of gallbladder wall thickening (e.g., mucosal edema and, less commonly, mucous retention cysts and heterotropic pancreas). In adults the cause of the PVC was either Cirrhosis or Pancreatitis. In a study by Barakat M *et al.* 2002[19], investigating 12 children (age 4-10 years) with PVC, Doppler sonography confirmed the venous flow waveform in the cavernous portal vein in all children with normal flow direction in the few intrahepatic portal vein branches and also in the intrahepatic veins. Splenomegaly was present in all (100%). Gallbladder varices were shown in four (33.33%) patients and perisplenic collaterals were shown in 3(30%). Our study showed similar results with splenomegaly in five (83.33%) of the patients and Gall Bladder Varices were present in two (28.57%) of the seven patients. The finding of a patent paraumbilical vein in cirrhosis of liver with clinical manifestations of caput medusae and a venous murmur in the epigastric region is called Cruveilhier - Baumgarten Syndrome. The patent paraumbilical vein runs in the faciform ligament from ligament from the left branch of the portal vein to the anterior abdominal wall, connected to the superior or inferior epigastric veins. It may be found in 6-30% of patients with cirrhosis. The sonomorphologic appearance has been described as "Bull's eye" falciform ligament. Their study showed that patent paraurnbhcal vein with a width of >3mm and hepatofugal flow suggests portal hypertension. In our study the patent Paraumbilicai vein was seen in 42% of the cases with cirrhosis. In majority of the cases the Paraumbilical Vein was massively enlarged. Suggesting that, the patients present late during the course of the illness. In 50 patients (Child A: 30; Child B; 15; Child C: 5) with cirrhosis of different etiologies the portal venous flow velocity and the diameter of the portal vein were examined by Duplex sonography. The mean portal venous flow velocity (12.341±6.481 cm/p) was significantly higher in patients with paraumbilical vein patency than in patients without paraumbilical shunts (8.863±7.772 cm/s). The prevalence of shunts of the paraumbilical vein in cirrhotics was significantly lower in category Child A (43.33%) than in Child B (66.66%) and Child C (80.00%). In study by Domland M *et al.* 2000 [20], investigating 70 patients with portal hypertension. 16 patients had a patent paraumbilical vein. The mean portal venous flow velocity (19.2±7.8 cm/s) and the flow volume (1.29±0.50 1/min) were significantly higher in patients with paraumbilical vein patency than in patients without paraumbillcal shunts (14.4 ± 4.6 cm/s; p = 0.029 and 0.88±0.34 1/min; p = 0.007 respectively). The prevalence of shunts of the paraumblhcal vein in cirrhotics was significantly lower in category Child A (6.3%) than in Child 13 (25.9%: p = 0.011) and Child C (33.3%: p = 0.006). With an increase in the severity of liver cirrhosis the incidence of paraumbilical vein patency rises. Our study showed incidence of patent paraumbihcal vein to be 34 %. This higher incidence could be attributed to small sample volume and selection bias.

CONCLUSION

Portal vein diameter, Portal Vein Thrombosis the Hepatic Arterial Resistive Index, Paraumbilical Vein, Patent Paraumbilical etc. parameters were very important in the diagnosis of portal hypertension.

REFERENCES

1. Lautt WW (1985) Mechanism and role of intrinsic regulation of hepatic arterial blood flow : the hapatic buffer response. Am J Physiol 249: G549-G556.
2. Gatta A II cuore nella cirrosi epatica. Proceedings 93rd Congresso Societa Italiana Medicina Interna, vol II. Edizioni Pozzi. Roma, 1992: pp 303-326.
3. Bernardi M. Trevisani F (1997) Systemic and regional hemodynamics in pre-ascitic cirrhosis) Hepatol 27: 588-591.
4. Epstein M, Berk DP, Hollenberg NK, Adams DF, Chalmers TC, Abrams HL, Merrill JP. Renal failure in a patient with cirrhosis. The role of active vasoconstriction. 1970 Am J Med; 49: 175-189.
5. Merkel C, Gatta A, Milani L, Amodio P, Zuin R. Intrarenal blood flow, circulation time and cortical vascular volume in a patient with cirrhosis. Scand J Gastroenterol 1981; 16: 775-780.
6. Gatta A. Merkel C, Gassetto M, Milani L. Zuin R, Ruol A. enhanced renal symphathetic tone in liver cirrhosis: evaluation by intrarenal administration of dihydroergocristine. Nephron 1982; 30: 364-367.
7. Sacerdoti D, Bolognesi M, Merkel C, Angeli P. Gatta A. Renal vasoconistriction in cirrhosis evaluated by duplex doppler ultrasonography. Hepatology 1993; 17: 219-224.
8. Forrest EH, Jalan R, Hayes PC (1996) Review article: renal circulatory changes in cirrhosis - pathogenesis and therapeutic prospects. Ailment Pharmacol Ther 10: 219-231.

9. Larsen FS, Olsen KS, Ejlersen E, Hansen BA. Paulson OB, Kundsen GM (1995) Cerebral blood flow autoregulation and transcranial Doppler sonography in patients with cirrhosis. Hepatology 22: 730-736.
10. Dam M, Burra P, Tedeschi U, Cagnin A. Chierichetti F, Ermani M, Ferlin G, Naccarato R, Pizzolato G (1998) Regional cerebral blood flow changes in patients with cirrhosis assessed with 99m TC-HM-PAO single-photon emission computed tomography: effect of liver transplantation. J Hepatol 29: 78-84.
11. Miller VE. Borland LL (1985) Pulsed Doppler duplex sonography and CT of portal vein thrombosis, AJR 145:73.
12. Alpern MB. Rubin JM. \Yilliams DM *et al.*. (1987) Artery hepatic: duplex Doppler US with angiographic correlation. Radiology 162:53-56.
13. Scoutt LM, Zawin ML, Taylor KJW (1990) Doppler US, part II Clinical applications. Radiology 174:309.
14. Gatani S, Bolondi L. l.i Bassi S, Zironi G. Barbara L (199l)Prevalence of spontaneous hepatofugal portal flow in liver cirrhosis. Clinical and endoscopic correlation in 228 patients. Gastroenterology 100 : 160
15. Nishihara K, Sakata K. Yagyu T, *et al.* Relationship between peripheral portal blood flow and liver function in patients with liver cirrhosis. Pulsed Doppler ultra sonographic study. Scand J Gastroenterol 1994; 29:859.
16. Kutlu R. Karaman I, Akbulut A, Baysal T. Sigirci A, Alkan A, Aladag M, Seckin Y, Sarac K. Quantitative Doppler evaluation of the splenoportal venous system in various stages of cirrhosis: differences between right and left portal veins. J Clin Ultrasound. 2002;30(9):537-43.
17. Goyal AK, Pokharna DR and Sharma SK. Ultrasonic measurements of portal vasculature in diagnosis of portal hypertension. *A* controversial subject reviewed. J Ultrasound Med; 19(1):45-48.
18. Gregor Kocher, Andreas Himmelmann Portal vein thrombosis (PVT): a study of 20 non-, cirrhotic cases. Swiss Med Weekly 2005: 135:372-376.
19. Barakat M. Doppler sonographic findings in children with idiopathic portal vein cavernous deformity and variceal hemorrhage. J Ultrasound Med 2002;21: 825-830.
20. Domland M. Gebel M. Casehtz M. Bleck J, Manns MP. Comparison of portal venouf| flow in cirrhotic patients with and without paraumbilical vein patency using duplex-sonography. Ultraschall Med. 2000:21(4):165-9.

Source of Support: None Declared
Conflict of Interest: None Declared