

Original Article

Frequency of Increased Portal Vein Size in Cirrhotic Patients with Esophageal Varices

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Abstract

Objective: To determine the frequency of portal vein size of more than 13 mm in patients with cirrhosis with oesophageal varices.

Methods: This cross-sectional descriptive study was conducted in the Medical OPD, Department of Medicine, from July 2021 to December 2021. The study duration was 6 months from 1-07-21 to 31-12-21. After reviewing the Upper GI report by the author and confirming the presence of varices in the oesophagus, patient demographics were obtained and the patients were referred to the Radiology OPD, where they were asked to undergo ultrasonography (USG) of the abdomen to measure the long axis of the portal vein diameter from the Radiology OPD. They were reassessed again in the Medical OPD with the USG abdomen report and portal vein diameter with spleen size in millimetres.

Results: Out of 60 patients with cirrhosis and oesophageal varices, 52 (86.7%) were found to have a portal vein diameter of equal to or more than 13 mm.

Conclusion: In our setting, there is a high frequency of portal vein diameter equal to or more than 13 mm in patients with varices.

Keywords: Portal vein size, Cirrhosis, Varices, Ultrasonography, Chronic Liver Disease.

Introduction

Cirrhosis represents the late stage of progressive hepatic fibrosis, which is characterised by distortion of the hepatic architecture and the formation of regenerative nodules. It is considered to be irreversible in its most advanced stages, at which point the only option may be liver transplantation. The commonest causes of cirrhosis are alcohol, especially in Western countries, whereas Hepatitis B virus (HBV) and Hepatitis C (HCV) are the prevailing causes in the eastern world. Evidence of HCV, HBV, and co-infection is found in the majority of patients (90%) with chronic liver diseases, and cirrhosis is found in 74 % of patients.¹ Hepatitis C virus is known to cause cirrhosis more commonly than hepatitis B virus after acute infection due to its potential to remain dormant and not be detected by the human immune system. Non-alcoholic fatty liver disease (NAFLD), which is now known as Metabolically Associated Steatotic Liver Disease (MASLD), is rapidly becoming a leading cause of cirrhosis due to the prevalence of obesity in the population. Statistics suggest that MASLD is overcoming HBV and HCV-related cirrhosis in western countries owing to the obesity epidemic and prevalence of a sedentary lifestyle. Since HBV and HCV-related liver diseases are silent diseases having almost no symptoms, the development of decompensation remains unnoticed until examination is done, which is characterised by the appearance of spider angiomas, flapping tremors, clubbing, palmar erythema, ascites, reduced liver span, splenomegaly, gynecomastia, and testicular atrophy in males. Investigations, including blood test and ultrasonography of the abdomen to confirm the diagnosis of cirrhosis in most cases. Till now, the only primary prophylaxis for cirrhosis is abstaining from alcohol, watching your weight and health, and vaccinating against the hepatitis B virus. The period between compensated and decompensated cirrhosis, which is considered the time when treatment can halt the progression of development of cirrhosis, can only be identified by carrying out screening tests for HBsAg and Anti-HCV antibody. Some patients present with incidental findings of positive tests for HBV and HCV, who can then be offered treatment with antiviral therapy, but mostly, patients are identified once decompensated cirrhosis

Contributions:

SA AR MJ NA - Conception, Design
SI NR - Acquisition, Analysis, Interpretation
AR SI - Drafting
SA AR NR MJ NA - Critical Review

All authors approved the final version to be published & agreed to be accountable for all aspects of the work.

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Data Availability Statement: The data supporting the findings of this study are available from the corresponding author upon reasonable request.

Institutional Review Board

Approval

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settles in, and only then can supportive treatment be offered to the patient with close follow-up for identifying the development of complications. Liver transplantation is the only modality that can offer complete resolution of all complications associated with cirrhosis. One complication of cirrhosis is the formation of varices, which have serious implications in undiagnosed cases of decompensated cirrhosis. Other complications associated with decompensated cirrhosis are hepatic encephalopathy, ascites with spontaneous bacterial peritonitis, increased risk of peptic ulcer, and hepatocellular carcinoma. Non-interventional methods such as ultrasonography (USG) of the abdomen have been recognised as an easy way of identifying the portal vein size, which correlates well with the formation of varices as well as their size. Other modalities that can be used include computed tomography (CT) scan and magnetic resonance imaging (MRI).

The portal vein is unique. The portal vein drains blood from the capillaries of the intestinal wall and spleen to the capillaries of the hepatic sinusoids.² The major abnormality of the portal venous system in CLD is portal hypertension, which is caused by increased resistance to portal venous flow due to the enlargement of the intra- and extrahepatic portal vein and subsequent development of portosystemic collaterals.

Studies have shown that continuous dilatation of the portal vein in cirrhosis leads to endothelial cell damage and stasis, and this is one of the major factors for thrombosis. Portal vein dilatation also increases the risk of thrombosis and is considered one of the complications that can occur in patients with decompensated cirrhosis. If association could be proved, patients could also be picked early for monitoring and prophylaxis or early treatment.³ It is a fact that portal vein diameter is usually increased in cirrhosis of the liver with portal hypertension, and the spleen is also enlarged in size. Previous studies reported have shown that there is a definite correlation between portal vein diameter and the presence of gastro-oesophageal varices.⁴ World Journal of Medical Sciences in 2009 concluded that patients who had a portal vein size of more than 14 mm were at a significant risk of bleeding from oesophageal varices.⁵ This cross-sectional study aimed to establish the frequency of portal vein size of more than 13 mm in cirrhotic patients with oesophageal varices in Pakistan.

Materials And Methods

This cross-sectional descriptive study focused on patients diagnosed with liver cirrhosis. The research was carried out at the Outpatient Department of Medicine from July 2021 to December 2021. This study was conducted in accordance with the ethical standards and principles. Institutional Review Board (IRB) or Ethics Committee approval was obtained before the commencement of the research (Reference number: IRB/POF/02-05/10-2025). Informed consent was obtained from all participants involved in the study. The research was conducted with respect to participant privacy, confidentiality, and autonomy. The sample size was calculated using the WHO sample size calculator by taking the Population Proportion of 81.5% with an absolute precision of 10%. A desired confidence level of 95% ($Z = 1.96$) and a specified margin of error were considered. A sample size of 60 patients was determined and collected through a consecutive non-probability sampling technique. Patients with previously or newly diagnosed cirrhotic liver disease with varices of any grade of either sex between 18 and 70 years of age were included in the study. However, patients with Grade IV Hepatic Encephalopathy and those on beta-blockers were excluded.

Upper GI Endoscopy was performed in patients in the Department of Gastroenterology in patients included for study, to look for esophageal varices. Ultrasonography was performed in all cases, and the diameter of the portal vein in mm was recorded along the long axis of the portal vein using a Toshiba Nemio MX machine with a curved array probe of 3.5Mhz from the Radiology OPD. The Radiologist was blinded and was unaware of the Upper GI endoscopy report and the presence or absence of varices.

Data were entered and analysed using SPSS version 21.0. The mean and standard deviation were calculated for quantitative variables such as age. Frequency and percentages were calculated for qualitative data, that is, portal vein diameter and oesophageal varices grade I, II, III, and IV. Effect modifiers such as age, sex, and grade of varices were controlled by stratification. Post-stratification chi-square test was applied. A P-value of ≤ 0.05 was considered significant.

Results

The mean age of the patients was 58.65 ± 7.190 , with a mean portal vein diameter of 14.03 ± 1.507 .

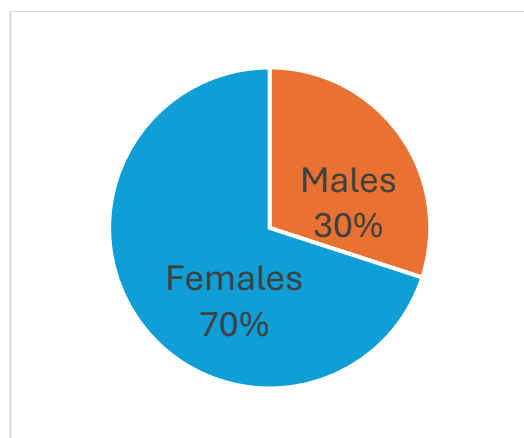


Figure 1: Distribution of male and female patients

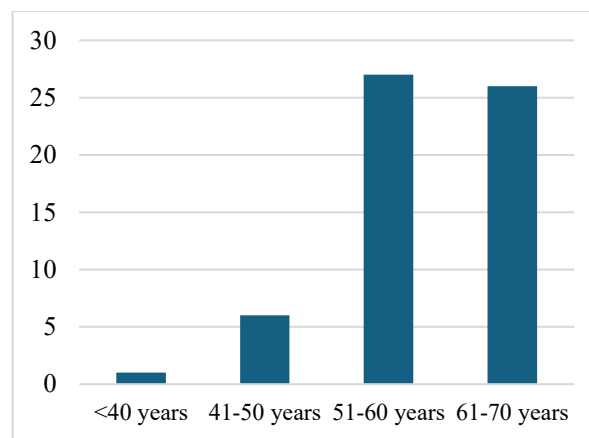


Figure 2: Number of Patients in various age groups

Patients were labelled as having a portal vein less than 13 mm or equal to or more than 13 mm based on the ultrasonography report of the abdomen. In my study, the mean portal vein diameter was 14.03 ± 1.507 . Of the 60 patients, 8(13.3%) had portal vein diameters of less than 13 mm, and 52(86.7%) had diameters of equal to or more than 13 mm.

Table 1: Crosstabulation of Portal Vein Diameter with Gender.

	Portal Vein Diameter >13mm		Total N	P-Value
	Yes n(%)	No n(%)		
Gender				
Male	14(77.8%)	4(22.2%)	18	0.225
Female	38(90.5%)	4(9.5%)	42	

Table 2: Crosstabulation of Portal Vein Diameter with Age.

	Portal Vein Diameter >13mm		Total N	P-Value
	Yes n(%)	No n (%)		
Age of Patients				
20-50	6(85.7%)	1(14.3%)	7	0.937
51-70	46(86.8%)	7(13.2%)	53	

As shown in the demographic analysis (Table 1), sex did not significantly affect the vein diameter. Age also showed no correlation (Table 2)..

Discussion

Bleeding from oesophageal varices is a major complication of cirrhosis and is a leading cause of mortality in these patients. Determining the presence and size of oesophageal varices requires OGD, which is invasive, expensive, and carries certain risks. Multiple national and international studies have been conducted in this regard, comparing different non-invasive methods and different portal vein sizes as predictors of the presence of oesophageal varices. A study conducted by Mathur et al,⁶ showed that out of 65 patients, 53 had a portal vein diameter of more than 13 mm, and this measurement of portal vein diameter (> 13 mm) was 81.5%, thus concluding that ultrasonography is an independent non-invasive predictor of the presence of oesophageal varices in patients with cirrhosis with portal hypertension.⁷ A study conducted by Lin et al. evaluated patients and found that portal vein >13.9 mm has a remarkable diagnostic potential in detecting oesophageal varices. Ayele et al,⁸ found the findings consistent with our study, claiming that the portal vein size is significantly increased in patients with CLD compared to controls. This study stated that a portal vein diameter of >13 mm had a sensitivity of 71.5% in predicting the presence of oesophageal varices.⁹

Ultrasonographic imaging plays a vital role in the assessment of the portal vein diameter, flow rate, and peak velocity, which provides an accurate and reliable method for diagnosing liver conditions, including CLD. The clinical manifestations of several CLDs include

Portal hypertension is a direct consequence of portal hypertension, which is defined as an increase in portal venous pressure above the normal limit of 5–10 mmHg. This linear increase in the diameter of the portal vein with CLD was observed even in the study by Yunusa *et al.*, thus explaining that enlargement of the portal vein has been considered a sign of portal hypertension. In addition to CLD, age has a consistent relationship with an increase in portal vein diameter.¹⁰ The subsequent thickening of the vessel wall and narrowing of the lumen of the vessel lead to (partial) obstruction of blood flow. The development of intimal hyperplasia often leads to an increase in portal pressure and diameter, leading to a compensatory increase in diameter. The progression of portal hypertension in patients with chronic liver disease is an important risk factor for the development of major complications, such as gastroesophageal varices, ascites, hepatic encephalopathy, and spontaneous bacterial peritonitis (SBP).¹¹ When the hepatic venous pressure gradient reaches a threshold value, there is a substantial risk of variceal bleeding, which is associated with increased mortality. A study conducted in Nigeria showed that ultrasonography was sensitive in gauging the right, left, and main portal vein diameters and was hence associated with an increased risk of cirrhosis-related complications.¹¹ One study showed that the greater the portal vein diameter, the greater the chances of having oesophageal varices, and diameters > 30 mm were found to be more significantly associated with the development of oesophageal varices.¹² Similar findings were found in a study by Khan *et al.* Similar findings were found in a study by Khan *et al.*,¹³ and Lv *et al.*,¹⁴ that portal vein diameter is greater with larger oesophageal varices. Katwal *et al.*, noted that the splenic index was directly proportional and portal venous velocity was indirectly proportional to variceal development, and ultrasonography was sensitive in patients with large varices and increased portal vein diameter.¹⁵ Nouh *et al.*, concluded that the association between portal vein diameter and oesophageal varices development may also help to restrict the need for unnecessary screening endoscopy.¹⁶ Song *et al.*, found that computed tomography is a safe and alternative method of predicting the risk of oesophageal bleeding in cirrhotic patients with portal hypertension, which shows that other modalities can be used alongside ultrasonography in predicting the risk of oesophageal bleeding.¹⁷

The results of our study and other studies suggest that the measurement of portal vein size by ultrasonography in patients with cirrhosis is a good guide for predicting the presence of oesophageal varices. It can help initiate beta-blocker therapy for these patients and is cost-effective, especially in Pakistan, where facilities to diagnose varices based on upper GI endoscopy are not available everywhere. This is also useful for patients who are unwilling or not fit enough for invasive procedures in areas where upper GI endoscopy facilities are available. Such patients can undergo timely referrals to hospitals with facilities where endoscopy and banding can be performed, which is a mode of treatment for patients at risk of developing oesophageal bleeding in the near future. Since variceal bleeding is a common complication in patients with cirrhosis and a significant cause of morbidity and mortality, it is prudent to detect it early so that intervention can help combat this complication.

Conclusions

There is a high frequency of portal vein diameters equal to or greater than 13 mm in patients with cirrhosis with varices.

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