Original Article

Portal Vein Diameter as a Single Parameter in Diagnosing Esophageal Varices - A Tertiary Care Hospital Experience

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Abstract

Esophageal varices are considered as one of the major complications of Decompensated Chronic liver Disease. The gold standard for diagnosis has been Esophagogastroduodenoscopy. Early diagnosis of esophageal varices is vital as dreadful complications like bleeding from the varices can be avoided with prophylactic medications.

Aim: This study is aimed at evaluating Portal Vein Diameter using Ultrasound Abdomen which is more widely available, as a tool to predict the presence of esophageal varices.

Methods: The study is a cross sectional study of 50 newly diagnosed patients with features of decompensated liver disease admitted at Chettinad Hospital, Kanchipuram. The portal vein diameter whether dilated or normal is compared with Esophagogastroduodenoscopy for the presence of varices in all patients with chronic liver disease.

Results: Out of the 50 patients with chronic liver disease 40 patients had esophageal varices. We observed that the portal vein diameter significantly correlated with esophageal varices. The odds of esophageal varices was 12.33 (OR=12.33, 95% CI 2.23 to 68.12, p-value <0.001) times higher in people with portal vein diameter > 13 mm, when compared to people with portal vein diameter < 13 mm.

Conclusion: Ultrasound Abdomen with measurement of portal vein diameter should be the first approach to screen any patient with Chronic Liver disease however portal vein diameter alone is not sufficient enough to predict esophageal varices and those patients who has an increased portal vein diameter should be planned for an esophago gastroduodenoscopy at the earliest.

Key Words: Portal vein Diameter, Esophageal Varices, Chronic Liver Disease

Introduction

An inevitable complication of most chronic liver disease is the development of portal hypertension. The reason behind its widespread complications of portal hypertension is due to the formation of Porto-systemic collaterals which results in the formation of various other complications in the form of gastresophageal varices, with or without variceal hemorrhage, ascites, renal dysfunction etc. Gastresophageal varices are seen in about 50% of patients with cirrhosis and they invariably correlates with the stage of liver disease. Around 40% patients of Child A liver disease have varices but they are present in almost 85% patients with Child class C[1,2] . The esophageal or gastric varices progress at a rate of 5% per year, which are even more prevalent in patients who continue to consume alcohol or with a patient whose liver functions keep deteriorating[3]. An HVPG of at least 10-12 mm Hg is needed for the esophageal varices to form[4,5]. Early diagnosis and treatment of gastresophageal varices prevent the further complications. The most vital part is if the varices can be diagnosed early and much before the varices bleed initially. It's essential because the studies of primary prophylaxis clearly show that the risk of variceal hemorrhage can be reduced significantly from 50% chances for bleeding to about or even less than 15% for large esophageal varices[6]. Endoscopy is regarded as a reliable method to assess the presence of gastresophageal varices and allows assessing risk factors if any such like any varices which are large, (more than 5mm in diameter) and those with presence of red color signs, (venules or any formation of red spots on varices); these factors if seen has a high propensity for bleeding[7,8]. The available guidelines recommend that all patients with cirrhosis of liver should be screened for any gastresophageal varices at the time of diagnosis and also these patients should be followed up regularly, if possible at every 2- 3 years in patients without varices (in regard with the severity of liver disease) and follow up every 1-2 years in patients with small varices, to assess for enlargement of varices and to decide on the need for prophylactic treatment and every 1 year for those with decompensated disease with or without varices[9]. Progression of a small varix to a large varix can happen due to several factors and they usually progress at a rate of 5 to 6% per year[10].

Non-invasive measures are used to predict esophageal varices especially in areas where routine endoscopies cannot be done. There are several non-invasive tests but an ideal test would be a one which is simple but quick, easily available with no or very low inter observational variations and which could be easily reproducible with a very low cost.

Doppler ultrasound is an effective modality to assess portal system as it provides a real-time picture of the portal system and allows estimation of both arterial and venous flow at a very affordable rate and hence is considered as the initial imaging modality in patients with chronic liver disease. Measurement of the Portal vein diameter, the portal flow velocity, the measurement of the congestion index, spleen size, the flow pattern in the hepatic veins and the presence of abdominal Porto systemic collaterals are all ultrasound parameters with a reliable prognostic significance. The factors related to the presence of varices are not well-defined. Therefore, in our study we have tried to determine the association of the portal vein diameter using ultrasound abdomen in predicting the existence of esophageal varices.

Materials And Methods

In order to avoid false negative results only new patients with features of chronic liver disease admitted at Chettinad Hospital, Kanchipuram was included in the study. An informed consent was obtained from all the patients prior to the study. The study was carried out from September 2015 to August 2016. The study was a cross sectional study and Fisher t- test was used for statistical analysis.

Inclusion Criteria: All patients with no previous documented medical history of liver disease and also those who were not on any medications for liver disease were included in the study. Patients with chronic liver disease evidenced by routine ultrasound abdomen with the age under 65 and above 18 were selected for the study.

Exclusion Criteria: Patients who presented with complications of chronic liver disease like Hepatorenal syndrome, Hepatocellular Carcinoma and those patients in shock were excluded from the study. Patients on beta blockers or patients with previous history of TIPS, Variceal band ligation, sclerotherapy were also excluded from the study.

Doppler Ultrasound

The patients in the study group were kept under overnight fasting. The Doppler ultrasound was done with the patient in the supine position during quiet respiration. The Portal vein diameter was measured and a diameter of more than 13mm was considered as elevated.

Endoscopic Features

All the patients were subjected to upper G.I endoscopy after an overnight fasting. Esophageal varices were graded as small if they are less than 5 mm and large if they are greater than 5 mm. Gastric varices if present, were typed according to their position and graded as small if less than 10 mm, medium if size is between 10 to 20 mm and large if greater than 20 mm. Portal hypertensive gastropathy was graded as mild and severe.

A total of 50 adult patients with history of chronic liver disease were included in the study. Complete history and physical examination along with complete blood count, viral hepatitis panel, erythrocyte sedimentation rate (ESR) and prothrombin time (PT/INR), liver function test, blood urea and serum creatinine was done for all the study participants. All patients were then planned for abdominal ultrasound and upper G.I endoscopy. Ultrasound evaluation was performed during inspiration; liver span along with echogenicity, nodularity of surface and size of the spleen were noted. Splenomegaly was defined as spleen size > 12 cm along the long axis. Dilated Portal vein is defined as a Portal vein diameter > 13 mm.

Their sensitivity, specificity and predictive values were calculated using upper G.I endoscopy as a gold standard. Written consent was obtained for upper G.I endoscopy from the patient in a language familiar to them.

Statistical analysis

Esophageal varices were considered as primary outcome variable. Portal vein diameter was considered as explanatory variable. The socio demographic variables, etiology and other clinical parameters were considered as other explanatory variables. Descriptive analysis of all the variables was done using mean and standard deviation for quantitative variables, frequency and percentage for categorical variables. The association between portal vein diameter and esophageal varices were assessed by cross tabulation. Odds ratio and it's 95% CI were calculated and chi square test was used to assess the statistical significance of association. The mean values of quantitative variables like MELD and INR were compared between people with and without esophageal varices using independent sample t test. P value less than 0.05 was considered statistically significant. IBM SPSS version 21 was used for statistical analysis.

Results

A total of 50 participants were included in the final analysis.

Table-1: General Profile of patients with esophageal varices

		Upper GI Endoscopy							
		Grade 1		Grade 2		Grade 3		No Varices	
		N	%	n	%	n	%	n	%
Etiology	Alcohol	18	90	7	95	9	75.00	9	90
	Cryptogenic	2	10	1	5	3	25.00	1	10
Ascites	Yes	1	5	5	63	12	100	4	40
	No	19	95	3	17	0	0	6	60
Splenomegaly	Yes	9	45	8	100	10	83	2	20
	No	11	55	0	.00	2	17	8	80
Child-Pugh Class	A	7	53.00	0	.00	0	.00	6	47.00
	B	12	63.015	3	15.78	0	.00	4	21.00
	C	1	5.5	5	27.77	12	66.66	0	.00

Alcohol was the major cause for most of the chronic liver disease patients (92%). When compared with Child Pugh Class with esophageal varices, among all the patients with Grade I varices 53% were Class A patients but no patients in class A had grade 3 varices. Similarly only 5% (n=1) with Child class C had Grade 1 varices but around 66.6% (n=12) had grade 3 varices. (Table 1)

Table-2: Comparison of esophageal varices with general parameters

	Upper GI Endoscopy							
	Grade 1		Grade 2		Grade 3		No Varices	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Hb	12.16	2.15	11.54	2.00	9.33	2.51	11.35	2.95
Platelet count (in Lakhs)	1.23	.50	1.48	.63	.80	.30	1.75	.71
Serum Bilirubin	1.99	1.70	3.19	2.00	5.42	4.36	1.78	1.44
Serum Albumin	3.26	.39	2.96	.37	2.70	.51	3.15	.74
Creatinine	1.02	.19	1.24	.31	1.64	.32	.98	.11
Prothrombin Time	14.26	2.89	16.06	3.20	16.48	1.30	13.77	1.46
INR	1.26	.20	1.36	.30	1.50	.20	1.12	.13
Portal Vein Diameter (in mm)	14.17	.89	15.44	.82	16.63	.88	12.12	1.10
Meld Score	12.17	5.73	15.27	4.02	20.53	4.75	9.31	2.05

Patients with Grade I esophageal varices the mean Portal vein diameter was 14.17mm with a standard deviation of 0.89, similarly Grade 3 varices had a mean portal vein diameter of 16.63 with a standard deviation of 0.88 and in patients with no varices at all the mean diameter was 12.12mm with a standard deviation of 1.10. (Table 2)

Table-3: Comparison of liver function parameters between people with and without EV (N=50)

Groups	Mean	Mean difference	P value	95% CI	
				Lower	Upper
I. EV –Serum Bilirubin					
Yes	3.25	1.47	0.15	1.47	1.01
No	1.78				
II. EV –Serum Albumin					
Yes	3.03	0.12	0.53	0.19	0.12
No	3.15				
III. EV–Creatinine					
Yes	1.25	0.276	0.025	0.03	0.51
No	0.97				
IV. EV– PROTHROMBIN TIME					
Yes	13.77	1.51	0.09	0.29	3.32
No	15.28				

The mean Serum bilirubin was 3.25 in people having esophageal varices and 1.78 in people without esophageal varices with a mean difference of 1.47 (95% CI 01.47 to 1.01) which was statistically not significant. (P value 0.15). (Table 3)

Table-4: Association of MELD score with EV

Meld Score				
	Upper GI Endoscopy	N	Subset for alpha = 0.05	
			1	2
Tukey B	Grade 1	20	12.1725	
	Grade 2	8	15.2713	
	Grade 3	12		20.5342

Grade 3 EV cases have the highest Meld score values and Grade 1 EV have lowest Meld score. (Table 4) The mean Meld score was 15.30 in people having esophageal varices and 9.30 in people without esophageal varices with a mean difference of 0.20 (95% CI 1.95 to 10.02) which was statistically significant. (P value 0.004).

Note: 1. One way Analysis of Variance (ANOVA) is used to test whether the groups compared have the same mean values or not. It is hypothesized (null hypothesis) that the groups compared have the same mean values against (the alternative) that at least one group has a different mean value. Fisher's F test is used to test the null hypothesis. If the Significance (Sig. also known as P value) is less than or equal to 0.05, the null hypothesis gets rejected and post hoc tests are used to find out homogeneous subsets having similar mean values among the groups compared. On the other hand, if the P value is more than 0.05, it is inferred that there is no reason to reject the null hypothesis.

For each one of the parameters considered in the present study, ANOVA is carried out and the results are summarized in the following table.

Table- 5: Association of Esophageal varices with Portal vein diameter in study population (N=50)

Portal Vein Diameter	EV		Odds ratio	P-value	95% CI	
	Yes	No			Lower	Upper
High	37	5	12.33	<0.001	8.239	1005.038
	88.1%	11.9%				
Low	3	5				
	37.5%	62.5%				

Portal Vein Diameter		EV		Chi-Square Test		Odds Ratio and 95% CI			
		Yes	No	Value	Exact Sig.	OR	P (OR)	95% CI	
								LL	UL
High	Count	39	3	27.121	< 0.001	91.000	< 0.001	8.239	1005.038
	Row %	92.86	7.14						
Low	Count	1	7						
	Row %	12.50	87.50						

The odds of esophageal varices was 12.33 (OR=12.33, 95% CI 2.23 to 68.12, p-value <0.001) times higher in people with portal vein diameter > 13 mm, when compared to people with portal vein diameter < 13 mm. The association between the esophageal varices with portal vein diameter was statistically significant. (Table 5)

Discussion

The progression of liver disease to chronic liver disease or cirrhosis of liver and then to the complication of liver disease are mainly dependent on the duration of the disease and due to the etiological nature of the disease11. Portal Hypertension is regarded as one of the major complication which develops as the liver disease progresses which results in an increased resistance for the portal blood flow[12]. And as a result, portal hypertension leads to the development of esophageal and gastric varices. The other ectopic regions where the varices can rarely form are the colonic and enteric varices 13. As a general rule the usual presentation of portal hypertension can be manifested by esophageal varices, gastric varices in the form of fundic varices or as portal hypertensive gastropathy or duodenopathy, splenomegaly, ascites, and lower limb edema[14,15,16].

Our study was done on 50 patients with features of chronic liver disease. Doppler ultrasound was used to measure the portal vein diameter.

The advantage of using Doppler study as a non invasive marker to predict the flow and its various characteristics in the venous system of a patient with portal hypertension was explained by Barakat[17]. Our study was centered on this basis to evaluate the portal vein diameter to predict esophageal varices using Doppler ultrasound.

We observed that the portal vein diameter significantly correlated with esophageal varices. The odds of esophageal varices was 12.33 (OR=12.33, 95% CI 2.23 to 68.12, p-value <0.001) times higher in people with portal vein diameter > 13 mm, when compared to people with portal vein diameter < 13 mm. A study done by Sarwar et al reported that a portal vein diameter of even greater than 11mm (which is 2mm less than the 13mm cut off taken in our study) had high risk for high grade varices and variceal bleed. Similar result was observed by Nicolau et al [18] who concluded that there is a significant association between portal vein diameter and formation of esophageal varices in cirrhotic patients with portal hypertension. Hagen-Ansert [19], in his study report had similar finding as ours. He stated that a portal vein diameter >13mm is associated with gastresophageal varices. Study by Jaheen[20] had a contradictory view and he stated that portal vein dilatation is very much insensitive indicator for portal hypertension and can be false positive in response to massive splenomegaly or acute PV thrombosis Bolondi et al, in his study concluded that Doppler study of portal vein was able to predict esophageal varices in only about 42% of patients with endoscopy proven esophageal varices and he added that portal vein diameter, portal vein velocity or even congestion index were not sensitive enough to predict esophageal varices[21]. It was contradictory to the study done by S.Plestina et al[22] who stated that portal vein size on Doppler scan can predicit esophageal bleeding varices. This was supported by the study done by Prihatini et al,[23] who in his study found that portal vein size of more than 12mm can directly give evidence of portal hypertension and esophageal varices.

Conclusion

The study for non-invasive marker to predict esophageal varices have been going since long. Ultra sound abdomen with Doppler study is an effective and reliable method to assess portal pressure. Measurement of Portal vein diameter is an alternative and non-invasive method in prediction of the presence of portal hypertension. However portal vein diameter alone is not sufficient enough to predict esophageal varices and those patients who have an increased portal vein diameter should be planned for an esophago gastroduodenoscopy at the earliest.

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Keep The Blood flowing to Remain Sane

Atrial fibrillation (A-fib) is the most common form of cardiac arrhythmia and it affects nearly 2.7 million adults in United States alone. As it sometimes predisposes to more serious complications like thrombosis, heart failure or stroke,the affected patients are often prescribed anticoagulants. A new large scale research done at Intermountain Medical Center Heart Institute in Salt Lake City, UT, suggests a possible link between delay in the start of anticoagulant therapy and occurrence of dementia. In this study, the researchers followed up 76000 A-fib patients without dementia on anticoagulant therapy for more than a year. They found that the risk of dementia significantly increased in those patients in whom the initiation of treatment was delayed after diagnosis. Even patients with low risk for stroke had 30% greater chance of developing dementia if the treatment was delayed by more than 30 days after diagnosis. Not surprisingly, that figure went up to 136% in those with high risk for stroke. So, when treating A-fib patients, it is prudent to remember to start the treatment as soon as possible after the diagnosis, provided you want your patient to remain sane!

(Presented at the Heart Rhythm Society's 38th annual Scientific Sessions in Chicago, 2017)

- Dr. K. Ramesh Rao