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Prediction Of Esophageal Varices In Chronic Liver Disease Patients Using A Combination Of Radiological And Biochemical Markers

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Abstract

Background: Portal hypertension is the main causative factor for most of the complications of cirrhosis and esophageal varices is the most dreaded complication. About 40-50% of patients presenting with decompensated cirrhosis are reported to have varices. In cirrhosis patients without varices, there is a chance of the development of varices at 5-8% per year. Variceal bleeding is a medical emergency. The overall mortality in six weeks is around 15-20% in variceal hemorrhage. The mortality rate is much higher in decompensated cases. Early diagnosis of varices before the first bleed is important as studies of primary prophylaxis clearly show that the risk of variceal hemorrhage can be reduced by 50% in small varices to about 15% in large esophageal varices.

Aim & Objective: The present study was conducted to investigate non-invasive biochemical and radiological markers for the prediction of esophageal varices in chronic liver disease patients.

Methods: In this observational study of 75 patients, conducted in the General Medicine Department of Kanyakumari Government Medical College, newly diagnosed patients with a chronic liver disease without a history of previous gastrointestinal bleeding were included between April 2019 and April 2020. Relevant clinical parameters were assessed which included physical examination, complete hemogram, liver function tests, renal function tests, prothrombin time, ascitic fluid analysis, liver stiffness measurement using fibroscan USG measurement of spleen diameter, OGD scopy, platelet count/ spleen diameter ratio.

Results: About 90% of cases had esophageal varices as detected by endoscopy. Patients with esophageal varices were found to have thrombocytopenia. Elevated prothrombin time and hypoalbuminemia were also found to be good predictors of esophageal varices. Patients with grade 2 and 3 varices were found to have spleen diameters more than 12 cm and were found to have a significant p-value. Also, patients with all grades of esophageal varices were found to have liver stiffness > 20Kpa. The sensitivity and specificity of liver stiffness in detecting the presence of esophageal varices are 61% and 91 % respectively with a cut-off of 20 Kpa. PC/SD ratio<909 was found to have a sensitivity and specificity of 81% and 85.7% in predicting esophageal varies in our study.

Conclusion: Non-invasive markers like platelet count, prothrombin time, spleen diameter, PC/SD ratio, and liver stiffness were found to have a significant role in predicting the presence of esophageal varices in cirrhotic patients. Patients with esophageal varices were found to have thrombocytopenia, elevated prothrombin time, large spleen diameter, and increased liver stiffness.

Keywords: Cirrhosis, esophageal varices, platelet count, liver stiffness, spleen diameter, endoscopy

Introduction

Cirrhosis is a condition that is defined histopathologically by the development of fibrosis to the point that there is architectural loss with the formation of regenerative nodules. The induction of fibrosis occurs with the activation of hepatic stellate cells, resulting in the formation of increased amounts of collagen and other components of the extracellular matrix.[1] Cirrhosis has many complications like portal hypertension and its sequelae of gastroesophageal variceal bleeding, splenomegaly, ascites, spontaneous bacterial peritonitis, hepatorenal syndrome, hepatopulmonary syndrome, and hepatocellular carcinoma. The most common and deadly complication of chronic liver disease is portal hypertension.[2] Portal hypertension is an abnormal increase in blood pressure defined clinically as a hepatic venous pressure gradient > 5 mmHg in the hepatic portal vein system. Portal pressure is a function of hepatic resistance and portal blood flow. In cirrhosis portal hypertension occurs due to structural changes due to fibrosis, notably progressive intrahepatic vascular remodeling with capillarization of sinusoids, fibrogenesis, neoangiogenesis, and development of intrahepatic shunts that lead to increased hepatic resistance. [3] This eventually produces an increase in portal pressures and a decrease in effective hepatocyte perfusion. It is responsible for the development of ascites and bleeding from esophagogastric varices, two complications that signify decompensated cirrhosis. The prevalence of any size varices in patients with compensated cirrhosis is approximately 40-50% and large varices are 5-10% respectively. [4] The incidence of esophageal varices (EVs) increases by nearly 5% per year, and the rate of progression from small to large varices is approximately 5 to 10 % per year small varices bleed at a rate of 5% per year, and large varices at 15% per year. It has been shown that the risk of variceal bleeding is related to the size of esophageal varices, with large esophageal varices being at greater risk; this is possibly due to a higher variceal wall tension in large esophageal varices.[5] The risk of variceal hemorrhage is also dependent on the severity of liver dysfunction and also the presence of red wale signs on endoscopy. Variceal bleeding is a medical emergency associated with a mortality that, despite recent progress, is still in the order of 10–20% at 6 weeks. Due to the increasing prevalence of chronic liver diseases, variceal hemorrhage is associated with significant morbidity, mortality, and healthcare costs. Early diagnosis of varices before the first bleed is essential as studies of primary prophylaxis clearly show that the risk of variceal hemorrhage can be reduced significantly. [6] Current Baveno VI consensus emphasizes the use of noninvasive markers like transient elastography for measuring liver stiffness and platelet counts. It also has put forward the criteria for screening endoscopy and surveillance endoscopy which will be discussed later. Endoscopies are the main diagnostic tool for the detection of esophageal varices in cirrhotic patients as they can correctly detect the presence, and location as well as detects at-risk varices.[7] But the main disadvantages of endoscopies are that they are invasive and many patients find them uncomfortable. Also, the number of endoscopy units in our country is not adequate to screen the cirrhotic population of our country.[8] Also, many patients with cirrhosis at the time of diagnosis wouldn't have developed varices. So subjecting them to endoscopy will not be of no use. So my study aims at using noninvasive criteria like platelet count, prothrombin time, spleen size, and fibroscan to predict the prevalence of esophagogastric varices in newly diagnosed cirrhotic patients.[9,10]

Methods: In this observational study of 75 patients, conducted in the General Medicine Department of Kanyakumari Government Medical College, newly diagnosed patients with a chronic liver disease without a history of previous gastrointestinal bleeding were included between April 2019 and April 2020. Relevant clinical parameters were assessed which included physical examination, complete hemogram, liver function tests, renal function tests, prothrombin time, ascitic fluid analysis, liver stiffness measurement using fibroscan USG measurement of spleen diameter, OGD scopy, platelet count/ spleen diameter ratio. Inclusion criteria: Age>18-60years, Newly diagnosed chronic liver disease patients.Exclusion criteria: Patients with unstable vitals, History of prior treatment, History of band ligation of varices/sclerosis /tips, History of bleeding disorders, History of liver transplantation. Relevant clinical history with particular attention to the history of hematemesis, melena, jaundice, abdominal distension, altered sensorium, alcohol intake, blood transfusion, intake of hepatotoxic drugs, exposure to sexually transmitted diseases, iv drug

Volume 5, Issue 6; November-December 2022; Page No 628-639

abuse, etc to find out the etiology of cirrhosis were noted. Physical examination included features of liver cell failure, jaundice, anemia, ascites, splenomegaly, hepatomegaly, and abdominal vein collaterals were recorded. All patients underwent biochemical tests, such as complete hemogram, liver function test, renal function tests, and prothrombin time. All patients were screened for hepatitis B surface antigens and antibodies to hepatitis C. In patients with ascites, ascitic fluid was tapped under aseptic precautions and ascitic fluid albumin and serum-ascites albumin gradients were measured. All patients underwent ultrasonography of the abdomen and details like liver size, spleen diameter, portal vein diameter, ascites, and presence of collaterals were noted. A fibro scan was done to assess the stiffness of the liver and the value was expressed in kPa. Upper GI endoscopy was carried out in all patients and the presence or absence of esophageal veins was recorded and grading of esophageal veins was done.

Stastical Methods: Data were analyzed using the SPSS software version 25 and data were significantly described in terms of mean $\pm$ standard deviation. For comparing categorical data Chi-square test was used. Kruskal Wallis test was used for comparing more than 2 groups. Differences were considered statistically significant if the two-tailed p-value was less than 0.05.

Results

The mean age of the study participants was 52±11.6 years. In table -1, 61.3% of the study participants were between 41-60 years of age followed by 22.7% who were above more than 61 years of age. The majority of the study participants were males.

Table-1:Distribution of demographic details among the study participants

S. No	Variables	Frequency	Percentage
1.	**Age**		
	<20	1	1.3
	21-40	11	14.7
	41-60	46	61.3
	>61	17	22.7
2.	**Sex**		
	Male	66	88.0
	Female	9	12.0

Table -2: Distribution of etiologies among the study participants

S. No	Etiologies	Frequency	Percentage
1.	AIH	3	4.0

Volume 5, Issue 6; November-December 2022; Page No 628-639

2.	Alcohol	47	62.7
3.	Cryptogenic	2	2.7
4.	Hep B	11	14.7
5.	Hep C	5	6.7
6.	NASH	1	1.3
7.	PSC	1	1.3
8.	Wilsons	5	6.7

Among the study participants, alcoholism (62.7%) is the most common cause of chronic liver disease followed by Hepatitis B disease (14.7%). About 6.7% of chronic liver pathology was caused by Wilsons (6.7%).

Table -3: Distribution of presenting symptoms among the study participants

S. No	Presenting symptoms	Frequency	Percentage
	Jaundice		
1.	Present	43	57.3
	Absent	32	42.3
	Encephalopathy		
2.	Present	6	8
	Absent	69	92
	Ascites		
3.	Present	46	61.3
	Absent	29	38.7

Among the study participants, 61.3 % of cases presented with ascites while 57.3% had jaundice and 8 % had encephalopathy.

Table -4: Mean distribution of the blood parameters among the study participants

Volume 5, Issue 6; November-December 2022; Page No 628-639

S. No	Blood parameters (N=75)	Mean	Standard deviation
1.	Hemoglobin (g/dl)	10.6	2.61
2.	Platelet counts (L/ml)	148773.3	63581.7
3.	Bilirubin (mg/dl)	2.78	1.9
4.	SGPT (units/L)	107.27	142.2
5.	SGOT (units/L)	142.67	188.19
6.	Serum Albumin (g/dl)	2.93	0.87
7.	Ascitic albumin (g/dl)	1.36	0.96
8.	SAAG (g/dl)	1.32	12.3
9.	PT-INR	1.58	0.962

Table -5: Distribution of the blood parameters among the study participants

S. No	Blood parameters (N=75)	Frequency	Percentage
	Hemoglobin (g/dl)		
1.	<9.1	19	25.3
	>9.1	56	74.7
	Platelet count (Lakh/mL)		
2.	<1.5	39	52.0
	>1.5	36	48.0
	Bilirubin(mg/dL)		
3.	>1.5	42	56.0
	<1.5	33	44.0
	SGPT (units per liter)		

Volume 5, Issue 6; November-December 2022; Page No 628-639

4.	>40	55	73.3
	<40	20	26.7
	SGOT (units per liter)		
5.	>40	68	90.7
	<40	7	9.3
	Serum albumin (g/dL)		
6.	<3	44	58.7
	>3	31	41.3
	PT/INR		
7.	>1.5	25	33.3
	<1.5	50	66.7
	PC/SD ratio		
8.	<909	24	32
	>909	51	68

In Table 5, 25.3% of the study participants had hemoglobin less than 9.1gm/dl. About 52% of them had platelets less than 1.5lakh/mm3.56% of them had bilirubin above 1.5mg/dl. 73.3% and 90% of patients had increased SGPT and SGOT respectively. About 58.7% of the study participants had serum albumin less than 3g/dl. 33.3% had elevated PT/INR. Among the study participants, 32% of them had a platelet count- spleen diameter ratio of less than 909.

Table 6: Distribution of Liver stiffness among the study participants

S. No	Variable (N=75)	Frequency	Percentage
	Liver stiffness (kPa)		
1.	>20	64	85.3
	<20	11	14.7

In Table 6, 85.3% of the study participants had liver stiffness of more than 20Kpa.

Table 7: Distribution of grading of esophageal varices among the study participants

S. No	Grading of esophageal varices (N=75)	Frequency	Percentage
	Grade -0	7	9.3

Volume 5, Issue 6; November-December 2022; Page No 628-639

1.	Grade -1	38	50.7
	Grade -2	18	24.0
	Grade-3	12	16.0

TABLE:7 Among the study participants, 90.7 had esophageal varices.9.3% had no varices. 50.7% of participants had grade-I and 24% of them had grade II followed by 16% grade-III esophageal varices during the *Chi-square test was applied. P<0.05 was considered significant. the majority of the study participants in 41-60 years of age had grade II to III esophageal varices and the majority in the age group above 61 years had grade II varices and the difference was found not statistically significant. The four grades of varices were found higher in males when compared to females.

Table 8: Association between platelet count and grading of esophageal varices among the study participants

S.no	Platelet count (Lakhs/ml)	Grading				P value
		0	1	2	3	
1.	<1.5	1	13	13	12	
	>1.5	6	25	5	0	**0.000***

Chi-square test was applied. P<0.05 was considered significant. In table 8, the study participants with grade II and grade III esophageal varices had a platelet count of fewer than 1.5 lakhs/mm3 which was found statistically significant with a p-value of 0.000.

Table 9: Association between bilirubin and grading of esophageal varices among the study participants

S.no	Bilirubin (mg/dl)	Grading				P value
		0	**1**	**2**	**3**	
1.	>1.5	5	18	10	9	
	<1.5	2	20	8	3	0.310

Chi-square test was applied. P<0.05 was considered significant. In table -13, the study participants with bilirubin levels above 1.5, g/dl had esophageal varices grade I-III which was almost equal to the participants with bilirubin less than 1.5mg/dl, and the difference was found not statistically significant.

Table 10: Association between serum albumin and grading of esophageal varices among the study participants

	Serum	Grading	

Volume 5, Issue 6; November-December 2022; Page No 628-639

S.no	albumin(g/dl)	0	1	2	3	P value
1,	<3	2	17	13	12	**0.001***
	>3	5	21	5	0	

In table-10*Chi-square test was applied. P<0.05 was considered significant, the majority of the study participants with a serum albumin less than 3gm/dl presented with esophageal varices of grades I to III when compared to study participants with albumin above 3gm/dl and it was found statistically significant.

Table 11: Association between PT INR and grading of esophageal varices among the study participants

		Grading				
S.no	**PT/INR**	**0**	**1**	**2**	**3**	**P value**
	>1.5	2	6	7	10	
1						**0.000***
	<1.5	5	32	11	2	

*Chi-square test was applied. P<0.05 was considered significant. In table-11, the majority of the study participants with PT/INR more than 1.5 presented with esophageal varices of grades III when compared to other groups and it was found statistically significant.

Table 12: Association between Spleen size and grading of esophageal varices among the study participants

S.no	Spleen size (cm)	Grading				P value
		0	1	2	3	
1.	>12	2	11	11	12	
	<12	5	27	7	0	**0.000***

*Chi-square test was applied. P<0.05 was considered significant. Spleen size of more than 12 cm had a sensitivity of 65% and specificity of 71.4% in predicting the presence of varices in cirrhosis patients. In table-15, the study participants with esophageal grades II and III had spleen sizes more than 12 cm when compared to participants with spleens less than 12 cm and it was found statistically significant.

Table-13: Association between PC/SD ratio and presence of esophageal varices among the study participants

		Esophageal varices	P value

Volume 5, Issue 6; November-December 2022; Page No 628-639

Sl.no	PC/SD	Present	Absent	
1.	<909	45	6	
	>909	23	1	**0.003***

*Chi-square test was applied. P<0. between platelet count and spleen size with less than 909 had a stronger association with esophageal varices (p=0.003). The PC/SD had a sensitivity of 81% and specificity of 85.7% in detecting esophageal varices in the study.

Table -14: Association between the mean blood parameters and grading of esophageal varices among the study participants.

S. No	Blood parameters	Grading				P value
		0	1	2	3	
1.	Hemoglobin Mean SD	11.2 2.5	11.4 1.6	10.17 2.11	8.39 1.7	**0.036***
2.	Platelet count Mean SD	228285.7 95257.04	163157.89 33780.313	75166.67 22254.043	148773.33 63581.736	**0.000***
3.	Bilirubin Mean SD	3.1 1.5	2.4 1.8	2.8 1.9	3.6 2.07	0.205*
4.	Serum albumin Mean SD	3.35 .86	3.18 0.89	2.5 0.8	2.41 0.42	**0.009***
5.	PT-INR Mean SD	1.58 0.9	1.142 0.422	1.717 1.09	2.80 1.03	**0.000***
	Ascitic					

Volume 5, Issue 6; November-December 2022; Page No 628-639

6.	albumin Mean SD	3.45 1.90	1.616 0.8	0.87 0.43	1.13 0.89	**0.014***
7.	SAAG Mean SD	1.85 1.06	1.14 0.3	1.300 0.34	1.55 0.4	**0.049***

*Kruskal Wally test was applied. P<0.05 was considered significant.

From the table-14, the mean and SD were found to exceed normal limits in esophageal varices II to III and the difference was found statistically significant.

Discussion

Variceal bleeding is the most important complication of portal hypertension. So we need to screen patients with cirrhosis for the presence of esophageal varices. Upper GI scopy is the gold standard for screening for varices. But since it is an invasive procedure patient compliance will not be good enough. In addition to that many patients with initial, stages of cirrhosis won't have developed varices, and performing endoscopy won't be of much use in such patients. [11]Prediction of the presence of varices by non-invasive modes would allow us to minimize unnecessary endoscopies as well as for early detection of varices. Our study consisted of seventy-five patients of whom 9 were female and 66 were male. Males constitute the majority of 88% of the total sample population.61.3 % of the study participants were in the 41-60 years of age group. In our study, the main etiology of cirrhosis was found to be alcoholic liver disease (62.7%) followed by Hepatitis B (14.7%). 6.7% of the cases were due to Wilson's disease. [12]Ascites were found in 46 patients in our study constituting around 61.3% of the total. Encephalopathy was present in 6 of our patients constituting about 8 % of the total. 69 patients in our study had esophageal varices. Of this 50.7% had grade 1 varices, and 24% and 16% had grade II and grade III varices respectively. Relationships between several blood parameters and the presence of varices were studied. [13]Blood parameters included hemoglobin, bilirubin, SGOT, SGPT, serum albumin, ascitic albumin, and prothrombin time. Among these only platelet count (p0.000), prothrombin time (p 0.000), and serum albumin (0.001) had significant associations. [14] In a study by Fernandez M, et al. platelet count < 1 lakhs /mm3 had a sensitivity of 96% and specificity of 46% in predicting the presence of varices in cirrhosis patients. Radiological markers like spleen diameter and liver stiffness measured using fibroscan were used to predict the presence of esophageal varices in cirrhosis patients. Both of them were found to have significant associations. Splenomegaly with size > 12 mm was found to have a significant association (0.000) in predicting grade II and III varices while liver stiffness > 20 Kpa had an association for predicting all grades of varices.In our study splenomegaly had a sensitivity of 65% and a specificity of 71.4% respectively. [15]Mean liver stiffness in grade 0,1,2,3 varices was 11.8 Kpa, 23.7 Kpa, 51.3 Kpa, and 54.6 Kpa respectively. The sensitivity and specificity of liver stiffness in detecting the presence of esophageal varices are 61% and 91 % respectively with a cut-off of 20 Kpa. [16] A study by Sarin SK et al found that liver stiffness with cut-off 22.7Kpa had a sensitivity of 90% and specificity of 50% respectively. In a study by Vizzutti et al, a cut-off value of 17 kPa was used for the prediction of varices. A meta-analysis by Quet al found liver stiffness measurement by transient elastography as a good non-invasive tool for predicting esophageal varices in cirrhosis patients.[17] In a study by Henry Z et al association between platelet count < 88,000 and splenomegaly with the presence of varices was studied. It was found to have a sensitivity of 90% and a specificity of 50% respectively.[18] In our study platelet, count/spleen

Volume 5, Issue 6; November-December 2022; Page No 628-639

diameter ratio cut-off point was selected as 909 as done by Thuluvath et al. It was found that PC/SD < 909 had higher chances of esophageal varices. It had a significant assosciation (p =0.003). PC/SD ratio is a noninvasive and easy-to-obtain parameter. It had a sensitivity of 81% and specificity of 85.7% in detecting esophageal varices in our study.[19,20]

Conclusion

From our study, it was concluded that noninvasive parameters like low platelet count, elevated prothrombin time, hypoalbuminemia, splenomegaly, platelet count/spleen diameter ratio < 909, and increased liver stiffness can be used for predicting esophageal varices in cirrhosis patients. They can be used effectively to identify cirrhosis patients with a low probability of varices and avoid unnecessary endoscopies. These predictors show good efficacy in detecting the presence of esophageal varices.

References

1. Merli M, Nicolini G, Angeloni S, Rinaldi V, De Santis A, Merkel C, et al.Incidence and natural history of small esophageal varices in cirrhotic patients. J Hepatol 2003;38:266–272.
2. Reverter E, Tandon P, Augustin S, et al. A MELD-based model to determine the risk of mortality among patients with acute variceal bleeding. Gastroenterology 2014; 146(2): 412–419.e3.
3. de Franchis R; Baveno VI Faculty. Expanding consensus in portal hypertension: Report of the Baveno VI Consensus Workshop: Stratifying risk and individualizing care for portal hypertension. J Hepatol. 2015 Sep;63(3):743-52. doi: 10.1016/j.jhep.2015.05.022. Epub 2015 Jun 3. PMID: 26047908
4. North Italian Endoscopic Club for Study and Treatment of Esophageal Varices. Prediction of the first variceal hemorrhage in patients with cirrhosis of the liver and esophageal varices. A prospective multicenter study. N Engl J Med 1988; 319(15): 983–989.
5. Dooley., J., et al. (2018). Sherlock's diseases of the liver and biliary system. Hoboken, NJ, Wiley.
6. 6.. D'Amico G, Garcia-Tsao G, Pagliaro L. Natural history and prognostic indicators of survival in cirrhosis: a systematic review of 118 studies. Jhepatol 2006;44:217–231
7. Infante Rivard C, Esnaola S, Villeneuve JP. The clinical and statistical validity of conventional prognostic factors in predicting short□term survival among cirrhotics. Hepatology 1987; 7: 660–664.
8. Kim WR, Biggins SW, Kremers WK, et al. Hyponatremia and mortality among patients on the liver transplant waiting list. N Engl J Med 2008; 359: 1018–1026
9. Bosch J, Abraldes JG, Berzigotti A, Garcia Pagan JC. The clinical use of HVPG measurements in chronic liver disease. Nat Rev Gastroenterol Hepatol 2009; 6(10): 573–582
10. Bhathal PS, Grossman HJ. Reduction of the increased portal vascular resistance of the isolated perfused cirrhotic rat liver by vasodilators. J Hepatol 1985; 1(4): 325–337.
11. Iwakiri Y, Groszmann RJ. Vascular endothelial dysfunction in cirrhosis. J Hepatol 2007; 46(5): 927–934.
12. Gines P, Schrier RW. Renal failure in cirrhosis. N Engl J Med 2009; 361(13): 1279–1290.
13. Ripoll C, Groszmann R, Garcia Tsao G, et al. Hepatic venous pressure gradient predicts clinical decompensation in patients with compensated cirrhosis. Gastroenterology 2007; 133(2): 481–488.
14. Groszmann RJ, Garcia Tsao G, Bosch J, et al. Beta-blockers to prevent gastroesophageal varices in patients with cirrhosis. N Engl J Med 2005; 353(21): 2254–2261
15. Fernandez M, Semela D, Bruix J et al. Angiogenesis in liver disease. J Hepatol 2009; 50(3): 604–620
16. Hashizume M, Kitano S, Sugimachi K, Sueishi K. Three-dimensional view of the vascular structure of the lower esophagus in clinical portal hypertension. Hepatology 1988; 8(6): 1482–1487.
17. Sarin SK, Lahoti D, Saxena SP, et al. Prevalence, classification and natural history of gastric varices: a long term follows up study in 568 portal hypertension patients. Hepatology 1992; 16(6): 1343–1349.
18. Henry Z, Uppal D, Saad W, Caldwell S. Gastric and ectopic varices.
19. Clin Liver Dis 2014; 18(2): 371–388.
20. Merli M, Nicolini G, Angeloni S, Gentili F, Attili AF, Riggio O. The natural history of portal

Volume 5, Issue 6; November-December 2022; Page No 628-639

hypertensive gastropathy in patients with liver cirrhosis and mild portal hypertension. Am J Gastroenterol2004;99:1959–1965.

21. Berzigotti A, Garcia Criado A, Darnell A, Garcia Pagan JC. Imaging in clinical decision making for portal vein thrombosis. Nat Rev Gastroenterol Hepatol 2014; 11(5): 308–316.

Volume 5, Issue 6; November-December 2022; Page No 628-639