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An Observational Study of Predictors of Large Esophageal Varices in Cirrhotic Patients in a Tertiary Care Hospital

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Abstract: ***Introduction:*** *Cirrhosis of liver is a condition that has a variety of manifestation and complications, some of which can be life threatening (e.g. variceal bleeding). However it has become apparent that when the underlying insult (e.g. Chronic hepatitis C, hemochromatosis) has been removed, reversal of fibrosis also seen. Portal hypertension is a significant complication of cirrhosis and is responsible for development of esophageal varices.*

Materials and Methods: *This prospective observational study was performed between March 2017 and August 2018 at Department of Gastroenterology, Kurnool Medical College and Govt General Hospital, Kurnool. All patients with cirrhosis of liver presenting to the Department of Gastroenterology and General Medicine, who did not undergo endoscopy previously were included. Patients with prior treatment with β-blockers or endoscopic variceal ligation or endoscopic sclerotherapy, upper gastrointestinal hemorrhage before endoscopy, history of TIPS or Shunt surgery, patients with only gastric varices and patients with HCC were excluded from this study. Detailed history and symptoms and signs of cirrhosis were recorded in all patients. Ascites was graded as Grade I (Mild ascites only detectable by ultrasound), Grade II (Moderate ascites evident by moderate symmetrical distension of abdomen) or Grade III (Large or gross ascites with marked abdominal distension).[3] Hepatic encephalopathy was graded from grade 0 to IV as per the Conn's grading.[4] Diagnosis of cirrhosis was based on clinical, biochemical, and imaging findings.*

Results: *Fifty patients fulfilling inclusion criteria were enrolled for this study. Of these patients, 29 [58%] were male; 21 [42%] were female with a mean age of 47 years with a range of 18-56yrs.[Table 1]. Most common etiology was alcohol [n–19; 38%], followed by hepatitis B [n-8; 16%], hepatitis C [n-6; 12%], combined infection with hepatitis B and C [n-2; 4%], hepatitis B with alcohol [n-3; 6%], hepatitis C with alcohol [n-3; 6%], Wilson's disease [n-3; 6%], Budd Chiari syndrome [n-6; 12%], secondary biliary cirrhosis [n-1; 2%]. There were 12 patients in CTP class A; 21 patients in class B; 23 patients in class C.*

Conclusion: *Cirrhotic patients with a platelet count of less than 88,000, are more likely to have large esophageal varices on endoscopy than patients with a platelet count of more than 88,000. Platelet count has an inverse correlation with the presence of large varices. As the platelet count decreases, prevalence of large esophageal varices increases. Size of spleen has a direct correlation with size of esophageal varices. A spleen size of >16cm was associated with large varices whereas a spleen size of >14.5cm was associated with small esophageal varices.*

Key Words: *Cirrhosis, endoscopy, endoscopic sclerotherapy, Chronic hepatitis C*

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I. Introduction

Cirrhosis of liver is a condition that has a variety of manifestation and complications, some of which can be life threatening (e.g. variceal bleeding). However it has become apparent that when the underlying insult (e.g. Chronic hepatitis C, hemochromatosis) has been removed, reversal of fibrosis also seen. Portal hypertension is a significant complication of cirrhosis and is responsible for development of esophageal varices.[1]

Early detection of varices is important for treatment and prevention of progression. Over the last decade, it has become the common practice to screen known cirrhotics with endoscopy to look for esophageal varices. Several factors predict the risk of bleeding, including the severity of cirrhosis (Child's class, MELD score), the height wedged hepatic vein pressure, the size of varices, and some endoscopic stigmata, including red wale signs, haematocystic spots, diffuse

erythema, bluish colour, cherry red spots or white nipple spots.[2] There is no reliable noninvasive predictor for the presence or absence of esophageal varices that can be adopted in clinical practice. Various study show that transient elastogram and APRI for the assessment of oesophagealvarices in cirrhotic patient has a excellent accuracy.[3]

The parameter is chosen as it allows us to assess velocity of ultrasound wave through the fibrosed liver and another parameter like simple blood test aspartate aminotransferase and platelet count are easily obtainable and noninvasive and can be done as a routine liver function test. So, considering these results can be very helpful in predicting the esophageal varices.[4] In our country most of the patients with cirrhosis present at advanced decompensated stage. Endoscopic evaluation for esophageal varices is not always possible at most of the health care centers. Though endoscopy is gold standard to find varices, in order to relieve the patients from

Discomfort and the risk of rupture during endoscopy and to reduce costs, efforts to find non invasive methods like transient elastography and Aspartate aminotransferase-to-Platelet ratio index (APRI) are thought to be promising.

II. Materials And Methods

This prospective observational study was performed between March 2017 and August 2018 at Department of Gastroenterology, Kurnool Medical College and Govt General Hospital, Kurnool. All patients with cirrhosis of liver presenting to the Department of Gastroenterology and General Medicine, who did not undergo endoscopy previously were included. Patients with prior treatment with β-blockers or endoscopic variceal ligation or endoscopic sclerotherapy, upper gastrointestinal hemorrhage before endoscopy, history of TIPS or Shunt surgery, patients with only gastric varices and patients with HCC were excluded from this study. Detailed history and symptoms and signs of cirrhosis were recorded in all patients. Ascites was graded as Grade I (Mild ascites only detectable by ultrasound), Grade II (Moderate ascites evident by moderate symmetrical distension of abdomen) or Grade III (Large or gross ascites with marked abdominal distension).3 Hepatic encephalopathy was graded from grade 0 to IV as per the Conn's grading.4 Diagnosis of cirrhosis was based on clinical, biochemical, and imaging findings.

Blood investigation:

Complete blood picture with haemoglobin, total leukocyte count, platelet count, liver function tests, serum bilirubin (Total and conjugated), alanine aminotransferase and aspartate aminotransferase, protein, albumin, INR prothrombin time were done. Child-Pugh score was calculated for each patient.5Viral markers for HBsAg and antibodies to hepatitis C virus were done to determine the cause of cirrhosis. Other investigations like (Serum ceruloplasmin and slit lamp examination for Wilson's disease, tests for autoantibodies for autoimmune liver disease, iron studies for hemochromatosis) were carried out wherever indicated.

Ultrasound Abdomen with Doppler: Ultrasonography was done in all patients and the maximum vertical span of the liver, nodularity of liver surface, spleen size (Length of its longest axis), diameter of the portal and splenic veins, presence of portal-systemic collaterals, and presence of ascites were recorded.

Endoscopic Evaluation: All patients underwent endoscopy for assessment of esophageal and gastric varices within 1-2 days of admission. Esophageal varices were graded as small <5mm or large >5mm.6Gastric varices, portal hypertensive gastropathy, duodenopathy and rectal varices were recorded wherever present.

III. Results

Fifty patients fulfilling inclusion criteria were enrolled for this study. Of these patients, 29 [58%] were male; 21 [42%] were female with a mean age of 47 years with a range of 18-56yrs.[Table 1]. Most common etiology was alcohol [n–19; 38%], followed by hepatitis B [n-8; 16%], hepatitis C [n-6; 12%], combined infection with hepatitis B and C [n-2; 4%], hepatitis B with alcohol [n-3; 6%], hepatitis C with alcohol [n-3; 6%], Wilson's disease [n-3; 6%], Budd Chiari syndrome [n-6; 12%], secondary biliary cirrhosis [n-1; 2%]. There were 12 patients in CTP class A; 21 patients in class B; 23 patients in class C.

Overall, 29 patients had esophageal varices; 21 had no esophageal varices. In patients with esophageal varices, 14 had large varices and 15 had small varices. In our study, a platelet count of <100,000; spleen size of >14.5cm; platelet count/spleen diameter ratio <870; portal vein diameter >13mm; CTP class B/C were the best indicators of varices[Table 2]. These values represent median values and offered the best discrimination value on univariate analysis, variables significantly associated with presence of large varices were CTP class B/C, platelet count, spleen size, SAAG ratio, portal vein diameter and platelet count/spleen diameter ratio. On multivariate analysis, presence of large esophageal varices was significantly associated with a spleen size >16cm, platelet count < 88,000 and CTP class B/C [Table 3]

Table 1: Patient Demographics

S.No	Characteristics	Data
1	Total Number of patients	50
2	Male/Female	29/21
3	Mean age (years)	47 (Range, 18-56)
4	Etiology of liver disease	
5	Alcohol	19 (38)
6	Hepatitis B	8 (16)
7	Hepatitis C	6 (12)
8	Hepatitis B/Alcohol	3 (6)
9	Hepatitis C/Alcohol	3 (6)
10	Hepatitis B + C	2 (4)
11	Wilson's disease	3 (6)
12	Budd-Chiari syndrome	6 (12)
13	Secondary biliary cirrhosis	1(2)
14	Child–Pugh class	
15	A(5-6)	12 (24)
16	B(7-9)	21 (42)
17	C(10-15)	23 (46)
18	MELD (Mean ± SD)	18±6

Table 2: Relationship of Various Parameters with Presence or Absence of Large Esophageal Varices on Univariate Analysis

S.No	Variable	Varices Large N=14	Varices Small N=15
1	Age in years	42.16±6.13	45.13±8.53
2	Sex(M/F)	9/5	10/5
3	Ascites (%)	76%	78%
4	PALPABLE SPLENOMEGALY [%]	92.33%	76%
5	HEPATIC ENCEPHALOPATHY [%]	26.12%	26%
6	JAUNDICE [%]	94.13%	86.2%
7	PALLOR [%]	79%	75.34%
8	Hb [g/dl]	10.12±1.12	11.12±1.02
9	TOTAL COUNT	5813±1265	7125±1764
10	ALBUMIN [mg/dl]	1.86±0.075	2.15±0.35
11	BILIRUBIN [mg/dl]	3.02±0.43	2.65±0.76
12	AST [IU/L]	44.12±14.12	37.12±31.12
13	ALT [IU/L]	22.65±8.65	26.12±16.12
14	PT [sec]	18.54±3.22	17.12±1.62
15	INR	1.65±0.32	1.65±0.10
16	CREATININE [mg/dl]	1.22±0.47	1.08±0.21
17	PLATELET COUNT [n/mm^3]	88300±18560	102420±268435
18	PLC/SD RATIO	567.2±126.13	713.4±206.12
19	SAAG	1.65±0.13	1.17±0.65
20	PVD [in mm]	16.13±0.65	12.56±1.07
21	PVF HP HF OBS	 32.13% 60% 6.56%	 26% 65.24% 6.14%
22	COLLATERALS	42.6%	56.12%
23	USG ASCITES	67.10%	86.12%
24	USG SPLENOMEGALY [in cm]	16.14±4.13	12.62±1.65
25	MELD	19.65±4.13	17.13±3.05
26	CTP CLASS A B C	 0 3(12.12%) 12 (85.12%)	 2 (16.12%) 7 (42.15%) 6 (36.12%)

IV. Discussion

Endoscopy to detect varices is an invasive procedure and is not readily available in rural settings; hence, non-invasive parameters to detect large esophageal varices which are more likely to bleed are required. Several studies have evaluated possible non-invasive markers of esophageal varices. The aim of the present study is to identify clinical variables, lab parameters and imaging features that correlate with presence of large esophageal varices, so as to selectively refer patients for prophylactic esophageal band ligation.

Our data showed that 8 factors had predictive ability for the presence of esophageal varices on univariate analysis. However, on multivariate analysis only 5 of these 8 variables, namely a platelet count of <100,000; spleen size of >14.5cm; platelet count/spleen diameter ratio <870; portal vein diameter >13mm; CTP class B/C were the best indicators of varices. These values represent median values and offered the best discrimination value. Our data showed that 6 factors had predictive ability for the presence of large esophageal varices on univariate analysis. However, on multivariate analysis only 3 of these 6 variables, namely a platelet count of <88,000; spleen size of >16cm; CTP class B/C were the best indicators of large varices. These values represent median values and offered the best discrimination value.

The two parameters found to have independent predictive ability for large esophageal varices in our study namely, platelet count and splenomegaly have been the most consistently identified predictors of varices in a number of previous studies. Relationship of these two predictors to the presence of large esophageal varices may be explained, in that a palpable spleen as well as a low platelet count are both related to the presence of higher portal pressure.

V. Conclusion

Cirrhotic patients with a platelet count of less than 88,000, are more likely to have large esophageal varices on endoscopy than patients with a platelet count of more than 88,000. Platelet count has an inverse correlation with the presence of large varices. As the platelet count decreases, prevalence of large esophageal varices increases. Size of spleen has a direct correlation with size of esophageal varices. A spleen size of >16cm was associated with large varices whereas a spleen size of >14.5cm was associated with small esophageal varices. Size of varices was also related to the severity of liver disease. Proportion of patients with large varices was more in Child class B and C compared to Child class A

References

[1]. Merli M, , Nicolini G, , Angeloni S, , et al. . Incidence and natural history of small esophageal varices in cirrhotic patients. J Hepatol 2003;38(3):266–272.
[2]. Bosch J, , Berzigotti A, , Garcia-Pagan JC, , Abraldes JG. The management of portal hypertension: rational basis, available treatments and future options. J Hepatol 2008;48(Suppl 1):S68–S92.
[3]. National Guideline Clearinghouse . World Gastroenterology Organisation practice guideline: esophageal varices. Rockville, Md: Agency for Healthcare Research and Quality. http://www.guideline.gov/.
[4]. Liu CH, , Hsu SJ, , Liang CC, , et al. . Esophageal varices: noninvasive diagnosis with duplex Doppler US in patients with compensated cirrhosis. Radiology 2008;248(1):132–139.
[5]. D'Amico G, , De Franchis R; Cooperative Study Group. Upper digestive bleeding in cirrhosis. Post-therapeutic outcome and prognostic indicators. Hepatology 2003;38(3):599–612.
[6]. Chalasani N, , Kahi C, , Francois F, , et al. . Improved patient survival after acute variceal bleeding: a multicenter, cohort study. Am J Gastroenterol 2003;98(3):653–659.
[7]. North Italian Endoscopic Club for the 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.
[8]. Garcia-Tsao G, , Sanyal AJ, , Grace ND, , Carey W; Practice Guidelines Committee of the American Association for the Study of Liver Diseases; Practice Parameters Committee of the American College of Gastroenterology. Prevention and management of gastroesophageal varices and variceal hemorrhage in cirrhosis. Hepatology 2007;46(3):922–938.
[9]. de Franchis R. Evolving consensus in portal hypertension. Report of the Baveno IV consensus workshop on methodology of diagnosis and therapy in portal hypertension. J Hepatol 2005;43(1):167–176.
[10]. Chalasani N, , Imperiale TF, , Ismail A, , et al. . Predictors of large esophageal varices in patients with cirrhosis. Am J Gastroenterol 1999;94(11):3285–3291.

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