Sys Rev Pharm 2021; 12(4): 151-153
A multifaceted review journal in the field of pharmacy
E-ISSN 0976-2779 P-ISSN 0975-8453

Research Article

Correlation between AST Platelet Ratio and Severity of Espohageal Varices in Hepatitis B Induced Cirrhosis

Usama Riaz[1*], Amna Tariq[2], Uzma Yasmeen[3], Hafiza Sobia Ramzan[4]

[1]Department of Medicine, Medical Officer, Samundri Tehsil, Pakistan
[2]Department of Medicine, National Hospital and Medical Center, Lahore, Pakistan
[3]Department of Biochemistry, WMO, THQ hospital Bhowana, Punjab, Pakistan
[4]Department of Biochemistry, University of Lahore, Lahore, Pakistan

Article History: *Submitted: 03.05.2021* *Accepted: 10.05.2021* *Published: 17.05.2021*

ABSTRACT

Liver diseases, especially liver cirrhosis, are common in Pakistan due to the high prevalence of chronic viral hepatitis.

Objectives: The basic aim of the study is to analyse the correlation between AST platelet ratio and severity of oesophageal varices in hepatitis B induced cirrhosis. Setting: This cross sectional study was conducted in National Hospital Defence, Lahore during January 2019 to August 2019.

Material and methods: This study includes the patient who visited the OPD of the hospital regularly. All patients met the diagnostic criteria for cirrhosis for chronic hepatitis B prevention and treatment was included in this study. We recorded the age of the patients and the levels of AST, ALT, albumin, and PLT in peripheral blood. In addition, patients were divided into slight, moderate, and severe groups according to the morphology of oesophageal varices (EV) and the severity of haemorrhage under gastroscopy.

Results: The data were collected from 150 patients of both genders. There were 44 cirrhosis patients without oesophageal varices (nonoesophageal varices group), 43 with mild oesophageal varices (mild group), 31 with moderate oesophageal varices (moderate group), and 32 patients with severe oesophageal varices (severe group). There was no significant difference in age or gender between patients in each group (all p>0.05).

Conclusion: It is concluded that HBV infection is the leading aetiological factor underlying cirrhosis. Presentation for screening upper endoscopy is often late and with severe liver disease. Majority of cirrhotics have varices at endoscopy and with most being large varices.

Keywords: Liver; Viral; Chronic; Hepatitis

***Correspondence:**

Usama Riaz, Department of Medicine, Medical Officer, Samundri Tehsil, Pakistan, E-mail: Usamabutt1111@gmail.com

INTRODUCTION

Gastroesophageal varices are the most important clinical manifestation of portal hypertension. Oesophageal varices (OV) are present at diagnosis in approximately 50% of cirrhotic patients, being more common in Child-Pugh class C patients compared to Child-Pugh class A patients (85% versus 40%) (Gores GJ, *et al.*, 1989). De novo formation of varices occurs at a rate of 8% per year and the strongest predictor for development of varices in those with cirrhosis who have no varices at the time of initial endoscopic screening is an hepatic venous pressure gradient (HVPG)>10 mm Hg. Once varices form, they progress from small to large at a rate of 5-12% per year (D'Amico G and De Franchis R, 2003) (Table 1).

Esophageal varices (EV) are dilated collateral veins in the esophageal wall that project directly into the lumen. They are the second principle cause of death in cirrhotics and its prevalence in decompensated cirrhotics is 60% whereas in compensated cirrhotics which is 30% (Villanueva C, *et al.*, 2006). About 30% of these patients will experience an episode of variceal hemorrhage within a year of diagnosis. In patients with Child Class A and Child Class C disease, 6-week mortality for a single event of variceal bleed ranges from 0% to 30% respectively (Bosch J, *et al.*, 2008).

Liver cirrhosis, a common chronic liver disease, is caused by long-term or repeated damage to liver tissue due to one or more causes, leading to diffuse degeneration and necrosis of

Table 1: Demographic values of the patients

General information	no	mild	moderate	severe
Number of cases	44	42	31	32
Mean age	50.0 ± 12.6	47.8 ± 12.7	51.9 ± 11.3	46.5 ± 9.3
Male(cases)	25	31	19	24
Female(cases)	19	12	13	9
APRI	1.0(0.6,3.5)	1.7(1.1,2.9)	2.2(1.4,4.9)	2.7(1.9,4.2)
AAR	1.1(0.8,1.2)	1.0(0.8,1.4)	1.1(0.9,1.4)	1.2(0.9,1.4)
FIB-4	102(55.5,562.7)	196.3(104.9,392.2)	324.3(165.9,708.1)	244.0(153.3,518.1)
S index	0.3(0.1,1.0)	0.7(0.2,1.3)	0.9(0.4,1.9)	0.8(0.4,1.9)

hepatocytes, regenerative nodules, and fibrous tissue hyperplasia. Portal hypertension is observed in the advanced stage of cirrhosis (Smith JL and Graham DY, 1982). When the portal vein pressure increases to a certain degree, oesophageal varices can occur, while, in severe cases, oesophageal variceal bleeding will emerge, which is the most common and severe complication of cirrhosis and cirrhotic portal hypertension, as well as the most common cause of death for cirrhosis (Fugger R, *et al.*, 1992). In the 1990s, the mortality rate after the onset of oesophageal variceal bleeding was as high as 50%. With the development and advancements of medical technology and equipment, along with a further understanding of the disease, the mortality rate has decreased; however, new statistics indicate that the 6-week mortality rate is still as high as 20% (GraVeo M, *et al.*, 1994).

The basic aim of the study is to analyse the correlation between AST platelet ratio and severity of espohageal varices in hepatitis B induced cirrhosis.

MATERIALS AND METHODS

Settings

This cross sectional study was conducted in National Hospital Defence, Lahore during January 2019 to August 2019. This study was conducted with the permission of ethical committee of hospital.

Data collection

This study includes the patient who visited the OPD of the hospital regularly. All patients met the diagnostic criteria for cirrhosis for chronic hepatitis B prevention and treatment was included in this study. We recorded the age of the patients and the levels of AST, ALT, albumin, and PLT in peripheral blood. In addition, patients were divided into slight, moderate, and severe groups according to the morphology of oesophageal varices (EV) and the severity of haemorrhage under gastroscopy.

SPSS22.0 statistical software was used for all data processing and analysis. The chi-square test was used for enumeration data, the median (quartile) was used for normally distributed measurement data, and the rank sum test was employed for comparisons between groups. $p<0.05$ represented statistical significance.

RESULTS

The data were collected from 150 patients of both genders. There were 44 cirrhosis patients without oesophageal varices (nonoesophageal varices group), 43 with mild oesophageal varices (mild group), 31 with moderate oesophageal varices (moderate group), and 32 patients with severe oesophageal varices (severe group). There was no significant difference in age or gender between patients in each group (all $p>0.05$) (Table 2).

The following laboratory parameters; AST, ALP, GGT were found to be associated with presence of OV among the cirrhotic patients. However none of these were statistically significant on multivariate analysis.

DISCUSSION

This finding is in parallel with reports in the literature from West Africa and other hepatitis B endemic countries (Graham DY and Smith JL, 1981). Alcohol abuse was the second common cause of chronic liver disease in this study which implies that alcohol is a significant cause of liver cirrhosis in patients attending clinic at KBTH. This is a public health concern; therefore society should be educated on the harmful effect of alcohol abuse on the liver. A study by Abel *et al.* in Ethiopia, found HBV and HCV as the major causes of liver cirrhosis whiles in Sudan (Farooqi JI, *et al.*, 2007) alcohol abuse and HBV were the common causes (D'Amico G, *et al.*, 1999). However in Brazil alcohol was the commonest cause and in United States of America (Archampong TNA, *et al.*, 2015) HCV and alcohol were the major causes. The causes of liver cirrhosis are the same globally but the percentage of individual causes varies from one country to another country. NAFLD was not an important cause of cirrhosis in this study although (Mahassadi AK, *et al.*, 2012), globally; it is increasingly becoming an important aetiological factor (Pugh RN, *et al.*, 1973). The reasons for this difference may be due to increasing rate of obesity in the western countries and Asia compared to African countries (Valletpichard A, *et al.*, 2006).

Hepatic fibrosis is anticipated by APRI in a better way. Significant calculation errors are not seen in variables like platelet count and AST. Other existing possibilities of non-invasive measures to distinguish the existence of EV conversely have not yet been recognized to be used as an alternative to endoscopy (Sumon SM, *et al.*, 2013). It has been reported that APRI is correlated with histologic degree of liver fibrosis and cirrhosis (Perry IJ, *et al.*, 1998). APRI may well be interrelated to the existence of EV was postulated by innovators like Sanyal, *et al.* While potal hypertension and APRI were correlated by Berzigotti, *et al.* Various studies have proposed different cut off values for APRI like for Castéra, *et al.* and it was 1.3. The sensitivity, specificity, positive and negative predictive value of 68%, 64%, 51% and 78% respectively in their study. However as compared to our study these values were different (Mansoor S, *et al.*, 2015) (Figure 1).

Table 2: Multiple logistic regression model of independent risk factors of oesophageal varices

Independent risk factors	Adjusted Odds Ratio(OR)	Standard error	95% CI	p-value
Jaundice	1.58	1.28	0.32-7.75	0.57
Hematemesis	1.31	1.46	0.15-11.53	0.81
Mealena stools	38.95	82.15	0.62-2431.00	0.08
Weight loss	0.77	0.69	0.13-4.491	0.78
Anorexia	0.2	0.18	0.04-1.13	0.07
AST	1	0	0.99-1.01	0.31
ALP	1	0	0.99-1.00	0.27
GGT	1	0	0.99-1.01	0.1

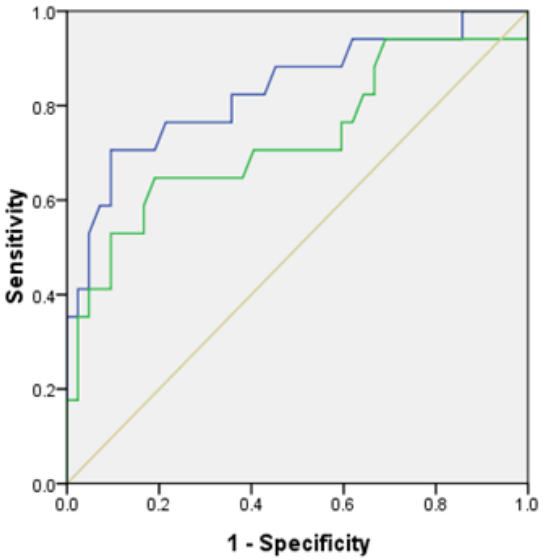

Figure 1: Receiver operating characteristic curve of APRI and S index in diagnosis of cirrhosis combined with severe oesophageal varices

CONCLUSION

It is concluded that HBV infection is the leading aetiological factor underlying cirrhosis. Presentation for screening upper endoscopy is often late and with severe liver disease. Majority of cirrhotics have varices at endoscopy and with most being large varices. Prophylactic treatment should be considered for all cirrhotics when upper GI endoscopy cannot be done immediately. Efforts at controlling HBV including universal vaccination at birth and access to testing and treatment should be intensified.

REFERENCES

1. Gores GJ, Wiesner RH, Dickson ER, Zinsmeister AR, Jorgensen RA, Langworthy A. Prospective evaluation of esophageal varices in primary biliary cirrhosis: development, natural history and influence on survival. Gastroenterology. 1989; 96(6):1552-1559.
2. D'Amico G, De Franchis R. A cooperative study group upper digestive bleeding in cirrhosis: Post-therapeutic outcome and prognostic indicators. Hepatology. 2003; 38(3):599-612.
3. Villanueva C, Piqueras M, Aracil C, Gomez C, Lopez-Balaguer JM, Gonzalez B, *et al.* A randomized controlled trial comparing ligation and sclerotherapy as emergency endoscopic treatment added to somatostatin in acute variceal bleeding. J Hepatol. 2006; 45(4): 5607-5617.
4. Bosch J, Thabut D, Albillos A, Carbonell N, Spicak J, Massard J, *et al.* Recombinant factor VIIA for variceal bleeding in patients with advanced cirrhosis: a randomized, controlled trial. Hepatology. 2008; 47(5): 1604-1614.
5. Smith JL, Graham DY. Variceal hemorrhage: A critical evaluation of survival analysis. Gastroenterology. 1982; 82(5): 968-973.
6. Fugger R, Herbst F, Mirza D. Combined sclerotherapy and operation for the treatment of bleeding oesophageal varices. Eur J Surg. 1992; 158(9): 485-489.
7. GraVeo M, BuVoli F, Lanzani G, Donato F, Cesari P, Benedini D, *et al.* Survival after endoscopic sclerotherapy for esophageal varices in cirrhotics. Am J Gastroenterol. 1994; 89(10): 1815-1822.
8. Graham DY, Smith JL. The course of patients after variceal hemorrhage. Gastroenterology. 1981; 80(4): 800-809.
9. Farooqi JI, Ahmed H, Ikramullah Q, Ahmed F, Masood-ur R. Predictors of esophageal varices in patients of liver cirrhosis. JPMI. 2007; 21(1): 60-64.
10. D'Amico G, Pagliaro L, Boch J, Patch D. Pharmacological treatment of portal hypertension: an evidence-based approach. Semin Liver Dis. 1999; 19(4): 475-505.
11. Archampong TNA, Tachi K, Agyei AA, Nkrumah KN. The significance of variceal haemorrhage in Ghana: a retrospective review. Ghana Med J. 2015; 49(4): 142-146.
12. Mahassadi AK, Bathaix FY, Wassi C, Bangoura AD, Allah-Kouadio EE, Kissi HY, *et al.* Usefulness of noninvasive predictors of oesophageal varices in black African cirrhotic patients in Cote d'Ivoire (West Africa). Gastroenterology Res Pract. 2012; 2012: 216390.
13. Pugh RN, Murray-Lyon IM, Dawson JL, Pietroni MC, William R. Transection of the oesophagus for the bleeding of the oesophageal varices. Br J Surg. 1973; 60(8): 646-469.
14. Valletpichard A, Mallet V, Pol S. FIB-4: A simple, inexpensive and accurate marker of fibrosis in HCV-infected patients. Hepatology. 2006; 44(3): 769-770.
15. Sumon SM, Sutradhar SR, Chowdhury M, Khan NA, Uddin MZ, Hasan MI, *et al.* Relation of different grades of esophageal varices with child-pugh classes in cirrhosis of liver. Mymensingh Med J. 2013; 22(1): 37-41.
16. Perry IJ, Wannamethee SG, Shaper AG. Prospective study of serum γ-glutamyltransferase and risk of NIDDM. Diabetes Care. 1998; 21(5): 732-737.
17. Mansoor S, Yerian L, Kohli R, Stavra Xanthakos, Paul Angulo, Simon Ling, *et al.* The evaluation of hepatic fibrosis scores in children with nonalcoholic fatty liver disease. Dig Dis Sci. 2015; 60(5): 1-8.