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Original Article

Frequency of Gastro Esophageal Varices in Cirrhotic Patients with Splenomegaly

Asma Abdul Razzak[1], Kamran Ali[1], Ayesha Zia[1], Abdul Lateef[2]
Department of Gastroenterology, Liaquat National Hospital[1], Karachi, Avicena Medical College[2], Lahore.

Abstract

Background: Patients with esophageal varices due to cirrhosis have a high propensity to bleed. Changes in hepatic structure result in higher resistance to portal blood flow which causes esophageal varices to form dilated portal veins and splenomegaly. Endoscopic procedures for diagnosing and treating varices have continued to evolve, with improved techniques and equipment for more effective treatment and reduced patient discomfort.
Objective: To determine the frequency of gastro-esophageal varices in cirrhotic patients with splenomegaly.
Study type, settings & duration: A cross-sectional study was conducted at the Department of Gastroenterology, Liaquat National Hospital, Karachi from September 2019 to February 2020.
Methodology: The sample size of 257 cirrhosis patients with splenomegaly with no prior history of variceal hemorrhage was taken. The patients with a history of hypertensive variceal bleeding, interferon therapy, and those who had received endoscopic or surgical treatment for portal hypertension were excluded from the study. The data of patients were collected by the author through a self-designed proforma and the demographics of those patients were noted.
Results: Out of 257 patients with cirrhosis, 157 (62%) were male and 100 (38%) were females. The mean age of these patients was 48.65±8.22 years. The frequency of gastro-esophageal varices in cirrhotic patients with splenomegaly was 62.65% (161/257).The prevalence of gastro-esophageal varices did not differ significantly by age group and gender.
Conclusion: The gastro-esophageal varices are frequently found in cirrhotic patients with splenomegaly. The chance of variceal bleeding in cirrhotic individuals can be expected by a variety of clinical and physiological parameters.
Key words: Gastro-esophageal varices, variceal hemorrhage, cirrhosis, splenomegaly, hepatitis.

Introduction

Esophageal varices can rupture or bleed which are the main problems of portal hypertension. Around 10-30% of all occurrences of upper gastrointestinal bleeding are caused by varices. It is the main factor in both morbidity and death in cirrhotic individuals.[1] Patients who do not already have gastro-esophageal varices experience an 8% annual growth rate.[2] After a year, 10–20 percent of instances of gastro-esophageal varices progress from minor to big varices.[3] First variceal hemorrhages occur at a rate of around 12% in the first year, and recurrences occur at a rate of about 60% in the first year. The presence of varices in cirrhotic patients with splenomegaly is an important clinical indicator of the severity of liver disease. Detecting varices in such patients helps healthcare providers monitor disease progression. It can indicate the need for more aggressive treatment and close follow-up.

When cirrhosis is diagnosed, it is advised that patients have endoscopic screening for gastro-esophageal varices.[2] Due to altered hepatic architecture, there is an increased resistance to portal blood flow which results in portal vein dilatation, splenomegaly, and the development of esophageal varices.[4] Patients with cirrhosis and splenomegaly may require regular screening and surveillance for varices to detect them at an early stage, allowing for timely intervention. Managing varices can improve the quality of life for these

Corresponding Author:
Asma Abdul Razzak
Department of Gastroenterology
Liaquat National Hospital, Karachi.
Email: dr.asmabofficial1@gmail.com

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Authors Contribution

AAR conceptualized the project. AL performed the statistical analysis. KA did the data collection and literature search. Drafting, revision & writing of manuscript were done by AZ.

patients. Preventing bleeding episodes and associated complications is essential for their well-being.

Gastro-esophageal varices in cirrhotic patients with splenomegaly are significant because they indicate advanced liver disease, carry a high risk of bleeding, and guide clinical decisions for management and prevention of complications. The rationale of this study was to determine the frequency of gastro-esophageal varices in cirrhotic patients with splenomegaly. This study would help to select patients for primary prophylaxis for gastro-esophageal varicesas local data are scarce on this topic. Several international studies are available but this study reports the findings of our population so that the current magnitude can be determined. The result of this study would help to improve the management of cirrhotic patients and in turn to reduce the morbidity of patients and overburdening of patients in hospital as it is focused on developing non-invasive methods for assessing the presence and severity of varices, such as using advanced imaging techniques and biomarkers, reducing the need for invasive endoscopic procedures.

Methodology

This cross-sectional study was held in the Department of Gastroenterology, Liaquat National Hospital, Karachi for a period of six months from September 2019 to February 2020. After obtaining approval from the ethical committee, 256 cirrhotic patients with splenomegaly of either gender without a previous history of variceal bleeding were taken in the study by non-probability consecutive sampling technique. A sample size of 257 patients was calculated by the WHO calculator keeping a 95% confidence interval considering a 60% prevalence of varices in patients with cirrhosis keeping a margin of error of 6%.[5]

The patients who had a history of portal hypertensive variceal bleeding, interferon therapy, and who were on treatment with β-Blockers, diuretics, or nitrates, and patients who had received endoscopic or surgical treatment for portal hypertension were excluded. Patients with a history of alcohol, diabetes, hypertension sepsis, or acute febrile illness based on systemic inflammatory response syndrome (SIRS) were also excluded from the study.

The data were analyzed using statistical software SPSS version 21. For qualitative variables like gender, child Pugh class (A/B/C), hepatitis B, hepatitis C, and esophageal varices, frequencies and percentages were calculated. Mean and standard deviations were calculated for quantitative data like age and duration of cirrhosis. Effect modifier like age, gender, and duration of cirrhosis, child Pugh class (A/B/C), hepatitis B, and hepatitis C was controlled through stratification and chi-square test was applied for their association. Independent sample t-test was used to check the mean difference between continuous variables. *p* values below 0.05 were considered significant.

The ethical approval was obtained from the Ethical Review Committee of Liaquat National Hospital, Karachi through reference number 0488-2019-I-NH-ERC.

Results

Two hundred and fifty-seven (n=257) patients with cirrhotic liver disease were enrolled. The mean age of these cirrhotic patients was 48.65±8.22 years. Most of the patients were 51 to 60 years of age 125 (48.6%) followed by 41 to 50 years 76 (29.5%) and 56 (22%) of age less than 40. There were 157 (61%) males and 100 (39%) females. Regarding hepatitis status, hepatitis-B was observed in 98 (38%) cases and hepatitis-C in202 (78.6%). According to Child-Pugh score, 25(9.7%) cases were in class A, 73 (28%) were in class B and 189 (61.8%) were in class C. The frequency of gastro-esophageal varices in cirrhotic patients with splenomegaly was 62.65% (161/257) (Table-1).

Table 1: Characteristics of patients with cirrhotic Disease. (n=257)

Variables	*Frequency (n)*	*Percentage (%)*
Gender		
Male	157	61
Female	100	39
Age		
< 40	56	22
41 – 50	76	29.5
51 – 60	125	48.6
Hepatitis status		
B	98	38
C	202	78.6
Child-pugh score		
Class – A	25	9.7
Class – B	73	28
Class – C	159	61.8

Table 2: Mean difference of age among child pugh score (CPS).

	Child Pugh Scoring	*Mean±SD*	*p-value*
Age	Class A	44.6±9.6	0.031*
	Class B	49.4±7.3	
	Class C	48.9±8.2	

A statistically significant difference was seen in the age of patients among the child Pugh

scoring (CPS) as the *p*-value was 0.031 with the increased mean age of 49±7 years in class B patients. Mean hemoglobin levels were 10.64±1.5 gm/dl and 10.32±1.6 gm/dl in patients with and without gastro-esophageal varices respectively (*p*-value >0.05) (Table-2). Mean leukocyte count and platelet counts in patients with gastro-esophageal varices were 8117.11±958.9/L and 116088.20±12806.1/L, respectively (Table-3). The prevalence of gastro esophageal varices was not significant among different age groups (*p* =0.071) as shown in Table-4. The rate of gastro-esophageal varices was not significant between males and females, and the rate of gastro-esophageal varices was not significant in those cases who had hepatitis B (*p* =0.48). At the same time, it was significantly high in those cases who had hepatitis C (*p* =0.019) respectively. The effect of duration of cirrhosis was also not statistically significant (*p*-value =0.88). Prevalence of gastro-esophageal varices was significantly high with Class B and C (*p* =0.0005).

Table 3: Mean difference among patients with and without gastroesophageal varices.

	With Gastro Esophageal Varices	*Without Gastro Esophageal Varices*	*p-value*
Hemoglobin	10.64±1.5	10.3±1.6	0.127
Leukocyte	8117.1±958.9	8174±903.6	0.637
Platelets	116088.2±12806.1	114713.2±12177.3	0.398

Discussion

In 2001, cirrhosis and chronic liver disease claimed nearly 27,000 lives annually, ranking as the 10th and 12th main causes of death for men and women, respectively.[6] Every year, 32,000 people die from cirrhosis, which affects 3.6 out of every 1000 individuals. It also results in more than a million lost work time. The occurrence of variceal bleeding, a direct result of portal hypertension, is a significant factor in cirrhosis-related morbidity and death.[7] Mortality is correlated with active variceal bleeding episodes and is approximately 30%.[8] Additionally, after a year of an episode of active bleeding, survivors are 70% more likely to experience another hemorrhage.[9]

There are a variety of liver conditions that can lead to cirrhosis by inducing cholestasis or persistent hepatic inflammation. Hepatitis C, alcoholic liver disease, and nonalcoholic liver disease are the three most frequent causes of cirrhosis in the West, collectively accounting for around 80% of people on the liver transplant waiting list.[10] The findings of this study correlate as out of two fifty-seven patients with cirrhosis liver disease Hepatitis-B was observed in 38.13% of cases and Hepatitis-C was 78.60%. Many other studies support these findings. According to estimates, HCV is responsible for 60 to 80 percent of cases of cirrhosis in Japan.[11,12] While the prevalence of HCV-associated cirrhosis ranges from 40 to 71 percent in Italy, it ranges from 27 to 58 percent in France.[13,14]

Numerous studies have shown that the male gender, drinking alcohol, and having HIV or HBsAg positive all increase the chance of developing cirrhosis and liver disease.[15,16] This study also reported male prevalence as there were 62% males and 39% females. In this study, most of the patients were 51 to 60 years of age. The patients were 48.65±8.22 years old on average. The correlation between age and cirrhosis liver disease is still unclear, with some research suggesting that the risk is higher among those under 50 [17] and others claiming that it is higher among people 40 years of age or older.[18]

Table 4: Frequency of gastro esophageal varices in cirrhotic patients with splenomegaly.

		Gastro Esophageal Varices		*p-value*
		Yes (N=161)	*No (N=96)*	
Age Groups (Years)	≤ 40 Years	35 (62.5%)	21 (37.5%)	0.071
	41 to 50 Years	40 (52.6%)	36 (47.4%)	
	51 to 60 Years	86 (68.8%)	39 (31.2%)	
Gender	Male	100 (63.7%)	57 (36.3%)	0.66
	Female	61 (61%)	39 (39%)	
Hepatitis B	Yes	64 (65.3%)	34 (34.7%)	0.48
	No	97 (61%)	62 (39%)	
Hepatitis C	Yes	134 (66.3%)	68 (33.7%)	0.019
	No	27 (49.1%)	28 (50.9%)	
Duration of Cirrhosis	6 to 9 months	74 (62.2%)	45 (37.8%)	0.88
	>9 months	87 (63%)	51 (37%)	
Different stages	Class A	7 (28%)	18 (72%)	0.0005
According to child	Class B	52 (71.2%)	21 (28.8%)	
Pugh's score	Class C	102 (64.2%)	57 (35.8%)	

The most common cause of morbidity and mortality in cirrhosis patients is variceal bleeding, which affects one-third of all patients with esophageal varices.[19] Around half of individuals with cirrhosis have this condition. About 50% of cirrhotic individuals experience the development of varices as a result of portal hypertension, and variceal bleeding happens at a rate of 5 to 15% each year.[20] According to a previous study, 25 to 40% of cirrhotic individuals experience variceal bleeding.[7] In this study, the frequency of gastroesophageal varices in cirrhotic patients with splenomegaly was 62.65%. Shin and group found that 38% of patients present with large esophageal varices had splenomegaly.[19]

This study concludes that gastro-esophageal varices are frequently found in cirrhotic patients with splenomegaly. The chance of variceal bleeding in cirrhotic individuals can be expected by a variety of clinical and physiological parameters.

Conflict of interest: None declared.

References

1. Hong W, Dong L, Jiang Z, Zhu Q, Jin S. Prediction of large esophageal varices in cirrhotic patients using classification and regression tree analysis Clinics (Sao Paulo) 2011; 66: 119-24.
2. Garcia-Tsao G, Sanyal AJ, Grace ND, Carey WD. Prevention and management of gastroesophageal varices and variceal hemorrhage in cirrhosis. Hepatology 2007; 102(9): 2086-102.
3. Giuffrè M, Macor D, Masutti F, Abazia C, Tinè F, Bedogni G, et al. Spleen Stiffness Probability Index (SSPI): A simple and accurate method to detect esophageal varices in patients with compensated liver cirrhosis. Ann Hepatol 2020; 19(1): 53-61.
4. Kamran A, Gilani SA, Bacha R, Shakeel S, Rashid R, Haider Z, et al. Correlation Between Diameter of Portal Vein and Length of Spleen in Patients with Chronic Liver Disease. Asian J Allied Health Sci 2020; 4(3): 4-8.
5. Sarangapani A, Shanmugam C, Kalyanasundaram M, Rangachari B, Thangavelu P, Subbarayan JK. Noninvasive prediction of large esophageal varices in chronic liver disease patients. Saudi J Gastroenterol 2010; 16(1): 38.
6. Anderson RN, Smith BL. Deaths: leading causes for 2001. Natl Vital Stat Rep 2003; 52(9): 1-85.
7. Garcia-Tsao G, Bosch J. Management of varices and variceal hemorrhage in cirrhosis. N Eng J Med 2010; 362(9): 823-32.
8. Sultan S, Altayar O, Siddique SM, Davitkov P, Feuerstein JD, Lim JK, et al. AGA institutes rapid review of the gastrointestinal and liver manifestations of COVID-19, meta-analysis of international data, and recommendations for the consultative management of patients with COVID-19. Gastroenterology 2020; 159(1): 320-334.e27.
9. Zanetto A, Garcia-Tsao GJF. Management of acute variceal hemorrhage. F1000Res 2019; 8: Rev-966.
10. Younossi Z, Stepanova M, Ong JP, Jacobson IM, Bugianesi E, Duseja A, et al. Nonalcoholic steatohepatitis is the fastest growing cause of hepatocellular carcinoma in liver transplant candidates. Clin Gastroenterol Hepatol 2019; 17(4): 748-55.
11. Toyoda H, Atsukawa M, Uojima H, Nozaki A, Tamai H, Takaguchi K, et al., editors. Trends and efficacy of interferon-free anti–hepatitis C virus therapy in the region of the high prevalence of elderly patients, cirrhosis, and hepatocellular carcinoma: A real-world, nationwide, multicenter study of 10 688 patients in Japan. Open forum infectious diseases, Oxford University Press US; 2019.
12. Rong D, Wu F, Lu C, Sun G, Shi X, Chen X, et al. m6A modification of circHPS5 and hepatocellular carcinoma progression through HMGA2 expression. Mol Ther Nucleic Acids 2021; 26: 637-48.
13. Buonomo AR, Scotto R, Coppola C, Pinchera B, Viceconte G, Rapillo CM et al. Direct acting antivirals treatment for hepatitis C virus infection does not increase the incidence of de novo hepatocellular carcinoma occurrence: Results from an Italian real-life cohort (LINA cohort). Medicine (Baltimore) 2020; 99(6): e18948..
14. Bertot LC, Adams LA Trends in hepatocellular carcinoma due to non-alcoholic fatty liver disease. Expert Rev Gastroenterol Hepatol 2019; 13(2): 179-87.
15. Sato S, Genda T, Ichida T, Murata A, Tsuzura H, Narita Y, et al. Impact of aldo-keto reductase family 1 member B10 on the risk of hepatitis C virus-related hepatocellular carcinoma. J Gastroenterol Hepatol 2016; 31(7): 1315-22.
16. Westbrook RH, Dusheiko G. Natural history of hepatitis C. J Hepatol 2014; 61(1) :S58-68.
17. Arora S, Thornton K, Murata G, Deming P, Kalishman S, Dion D, et al. Outcomes of treatment for hepatitis C virus infection by primary care providers. N Engl J Med 2011; 364(23): 2199-207.
18. Vergniol J, Boursier J, Coutzac C, Bertrais S, Foucher J, Angel C, et al. Evolution of noninvasive tests of liver fibrosis is associated with prognosis in patients with chronic hepatitis C. Hepatology 2014; 60(1): 65-76.
19. Shin SU, Lee J-M, Yu MH, Yoon JH, Han JK, Choi B-I, et al. Prediction of esophageal varices in patients with cirrhosis: usefulness of three-dimensional MR elastography with echo-planar imaging technique. Radiology 2014; 272(1): 143.
20. Seo YS. Prevention and management of gastroesophageal varices. Clin Mol Hepatol 2018; 24(1): 20-42.