ORIGINAL ARTICLE

Comparative Study of Serum Albumin and Platelet Count as Predictors of Esophageal Varices in Cirrhosis

ZAHID HUSSAIN SHAH, FARRUKH GHIAS, BEENISH SAFDAR, *IRSHAD HUSSAIN QURESHI, SAJID ABADULLAH*

ABSTRACT

Aim: To evaluate the predictive value of serum albumin and platelet count as predictors of esophageal varices in patients with cirrhosis and to screen patients for upper GI endoscopy.
Study design: This was a prospective & an analytical study.
Study place and duration: Gastroenterology and medical units at KEMU/ Mayo Hospital, Lahore. December 2007 - November 2009
Methodology: We studied 250 cirrhotic patients at Gastroenterology & medical Units of KEMU/ Mayo hospital, Lahore without previous history of upper esophageal varices bleeding were inducted in study and investigated by laboratory tests and abdominal ultrasound. Upper Gastro intestinal endoscopy was done to evaluate presence of gastro esophageal varices with endoscope GIF 150. Non endoscopic predictors of esophageal varices (platelet count& serum albumin) were identified & correlated by Univariate / Multivariate logistic Regression analysis. Variables mean values were measured by unpaired two tailed student t- test. Percentages were given for categorized variables. Chi square test was applied for categorical data. Quantitative variables were expressed as Mean± SD.
Results: Out of two hundred and fifty patients, one hundred and eighty patients (180) were found to have esophageal varices while seventy patients (70) patients had no varices. platelet count < 100 $x10^3$/ µL and serum albumin <2.5g/dl were significantly associated along with presence of Eosophageal varices.
Conclusion: platelet count < 100 $x10^3$/ µL& serum albumin < 2.5 g/dl were significantly associated with presence of esophageal varices.
Keywords: Esophageal varices, serum albumin, platelet count, cirrhosis.

INTRODUCTION

Cirrhosis accounts for the top most causes of death in our country. Upper GI bleed is due to rupture of varices which is usually a common mode of presentation in cirrhotic patients[2].

Esophageal varices bleed is the cause of death in one third of these patients. Re bleeding occurs in about 25 percent to 30 percent due to esophageal varices in a span of 2 to 3 years[4].

Cirrhotic patients have 5% incidence of esophageal varices / year[5].

AASLD recommends that upper GI endoscopy should be done in these patients for evaluation of eosophageal varices[6,11].

Different nonendoscopic parameters in the past are evaluated, such as platelet count, raised porta vein l pressure, red spots etc found on Endoscopy. There is a need of time to detect non endoscopic predictors to select group of patients who undergo such expensive procedure and therefore play a role of reasonable screening tool[11].

Department of Medicine, K. E. Medical University/Mayo Hospital, Lahore
Correspondence to Dr. Zahid Hussain Shah, Assistant Professor Medicine, email: zahidhamdani65@gmail.com

The objective of the study was to determine non-invasive parameters for example serum albumin & platelet count in patients with cirrhosis having no previous history of upper variceal GI bleeding and thus to find out patients for surveillance endoscopy.

METHODOLOGY

It was Cross sectional & analytical study conducted during December 2007 and October 2009. 250 patients were studied. Sample size was calculated by Raosoft calculator. For this study confidence interval was 92% and 8% was margin of error. Grades of esophageal varices were classified by PAQUET classification (1-1V). Grade I–II are further considered as small and Grade III-IV as large varices in this study[1,12,13]. Cirrhotic patients of either sex having no previous history of upper eosophageal variceal GI bleed were included in the study.

Exclusion criteria

1. Pharmacological or interventional therapy for esophageal varices in the past
2. History of active alcohol use < six months abstinence
3. History of upper variceal bleed

After taking proper consent from the patient. History and physical examination were done & registered in proforma after patient consent. Lab investigations were ordered e.g. hepatitis B and C viral markers, complete blood count, serum platelet count, liver function tests etc. Upper esophagogastroduodenoscopy was done in all these patients through Olympus Video Endoscope GIF 150 to detect esophageal varices[11]. Sysmex KX-21 hematology analyzer was used to calculate platelet count. Bromocresol Green method was used through Sturno-300 chemistry analyzer which was fully automatic and also used was Olympus AU-400 fully Automatic Chemistry Analyzer for measuring serum albumin.

Statistical analysis: All collected data was recorded on specified proforma. For analysis of data SPSS version 17.0 was used. Threshold of variables for diagnostic accuracy was calculated by using ROC curve .p- value less than (< 0.05) had significance in this study[11].

RESULTS

Mean age of patients was 51±10 years. Enlarged liver was found in 88(35.2%) patients and fifty six (22.4%) had spleen palpable. Forty (16%) patients had child class A, child class B was found in one hundred thirty (52%) and 80(32%) were detected with child class C. One hundred eighty (72%) patients had ascites while 70(28%) patients had no ascites. In one hundred eighty 180(72%) patients esophageal varices were seen. Hepatitis C virus was detected in one hundred sixty five (66%) patients while hepatitis B virus was found in twenty (8%). Different variables comparison in patients with and without varices.

Presence of varices was revealed by linear correlation. There was significant for Serum albumin (p - 0.010) &Platelet count (p - 0.097).

Identification of cutoff values for Serum Albumin as 2.5 g /dl and 100 x 10^3/ µL for Platelet count was done by ROC curve.

Different variables significant correlation for presence of varices was done for negative predictive value, specificity & sensitivity, odds ratio, positive predictive value, and receiver operating characteristic (ROC) curve, esophageal varices in a study by Brennan MRS et al[8].Results of this study are in accordance with it.

Chalasani et al[11] conducted a study on three hundred forty six patients and concluded that splenomegaly & platelet count < 88x10^3/µL were evaluated as independent risk factors for high grade eosophageal varices. Results of our study conclude that platelet count < 100 x10^3/ µL was found to be independent risk factor for development of eosophageal varices.

Raised prothrombin time, low platelet count, and spider naevi were detected as isolated risk factors for the development & progression of esophageal varices in a study by Pilette et al[13].

Development of esophageal varices had significant correlation with decreased serum albumin level and platelet count <150 x 10^3/ µL in a study by Zein et al [21].

Identification of eosophageal varices was significantly associated with platelet count less than 88x10^3/ µL in study conducted by Zaman et al[12]. These results match study results of us the decreased platelet count is concerned.

In a study done by Umar M et al[9,17] 73% patients were male and 27% patients were female.

Nadeem MA et al[16] study concluded that twenty three percent patients were found to be HBsAg +ve (positive), fifty percent patients were detected as Hepatitis C virus+ve while nine percent patients were found to be positive for both Hepatitis B & Hepatitis C viruses whereas thirteen percent patients were negative for both Hepatitis B and Hepatitis C virus. Our study concluded that, male patients had HCV.> HBV.

CONCLUSION

Platelet count < 100 x 10^3/ µL & Serum albumin < 2.5g /dl were found to be independent predictors for presence & development of eosophageal varices. In addition eosophageal varices were found to have a strong predictive value with serum albumin < 2.5g /dl as compared to platelet count<100 x 10^3/µL. Most probably there are some other factors besides portal hypertension which play an important role in the etiology of thrombocytopenia in cirrhotic patients like bone marrow suppression in chronic liver disease. Take home message of study is that Endoscopy should be performed in patients with CLD if either one predictor or both are detected in any patient to decrease mortality & morbidity of these patients.

REFERENCES

1. Merican I, Guan R, Amarapuka D, Alexander MJ, Chutaputti A, Chein RN. Chronic hepatitis B virus infection in Asian countries. J Gastroentrol Hepatol 2000; 15:1356-61
2. Iqbal F. Non-operative management of bleeding esophageal varices: endoscopic pharmacological, or both? J Coll Physcian Surg Pak 2002; 12: 2-3.
3. Chaudary ZI. Octeriotide in management of variceal bleeding and comparison with injection sclerothrerapy. Pak J Gastroentrol 2001; 15: 63-71.
4. Zoli M, Merkel C, Donatella M, Marchesini G, Gatta A, Pisi E. Evaluation of a new endoscopic index to predict

first bleeding from the upper gastrointestinal tract in patients with cirrhosis. Hepatology 1996; 24: 1047-52.
5. Grace ND, Groszmann RJ, Garcia-Tsao G. Portal hypertension and variceal bleeding: an AASLD single topic symposium. Hepatology 1998; 28: 868–80.
6. Merkel C, Bolognesi M, Sacerdoti D, et al: The hemodynamic response to medical treatment of portal hypertension as a predictor of clinical effectiveness in the primary prophylaxis of variceal bleeding in cirrhosis. Hepatology 32:930, 2000
7. Gartia-Tasao G. Current management of complications of cirrhosis and portal hypertension. Gastroentrol 2001;120:726-48.
8. Brennan MRS, Targownik L, Gareth SD, Hetal AK, Ian MG. Endoscopic screening for esophageal varices in cirrhosis: Is it ever cost effective? Hepatology 2003; 37: 366-77.
9. Umar M, Kanwal S,Chohan AR, Chohan S, Baqai HZ, Khaar HTB, et al. Pattern of eophageal vrices and portal gastropathy in HCV related cirrhosis. Pakistan J Gastroenterol Mar 2003; 17(1):20-3.
10. Macias RMA, Rendon UP, Navas RC, Tejada CM, Infantes HJM, Martin HL. Ultrasonography in patients with chronic liver disease: its usefulness in the diagnosis of cirrhosis. Rev Esp Enferm Dig. 2003 Apr; 95(4):258-64, 251-7.
11. Chalasani N, Imperiale T F, Ismail A, Sood G, Carey M, Wilcox CM, et al. Predictors of large esophageal varices in patients with cirrhosis. Am J Gastroenterol 1999; 94: 3285–91.
12. Zaman A, Hapke R, Flora K, Rosen HR, Benner K. Factors predicting the presence of esophageal varices or gastric varices in patients with advanced liver disease. Am J Gastroenterol 1999; 94: 3292–6.
13. Pilette C, Oberti F, Aube C, Rousselet MC, Bedossa P, Gallois Y, et al. Non-invasive diagnosis of esophageal varices in chronic liver disease. J Hepatol 1999; 31: 867–73.
14. Bernatik T, Strobel D, Hahn EG, Becker D. Doppler measurements: a surrogate marker of liver fibrosis? Eur J Gastroenterol Hepatol. 2002 Apr;14(4):383-7.
15. Younas BB, Khan GM, Akhtar P, Chaudhry A. Audit of patients of chronic liver disease. Ann King Edward Med Coll Mar 2001; 7(1):52-4.
16. Nadeem MA, Waseem T, Sheikh AM, Grumman N, Irfan K, Hasnain SS. Hepatitis C Virus: An alarmingly increasing cause of liver cirrhosis in Pakistan. Pakistan J Gastroenterol Mar 2002; 16(1):3-8.
17. Umar M, Khaar HTB, Anwar F, Ahmed S, Chohan A, Siddique K, et al. Evaluation of anti-HCV antibodies among family contacts of HCV related chronic liver disease patients. Pakistan J Gastroenterol Mar 2003; 17(1):27-9.
18. Khokhar N, Gill ML. Serological profile of incidentally detected asymptomatic HBsAg positive subjects (IDAHS). J Coll Physicians Surg Pak Apr 2004; 14(4):208-10.
19. Metz CE. Basic principles of ROC analysis. Semin Nucl Med 1978; 8: 283-98.
20. Hosmer DW, Lemeshow S. Applied logistic regression. New York: John Wiley & Sons; 1989.
21. Zein CO, Lindor KD, Angulo P. Prevalence and predictors of Esophageal varices in patients with primary sclerosing cholangitis. Hepatology 2004; 39: 204-10.