Non-Endoscopic Parameters for the Identification of Esophageal Varices in Patients with Chronic Hepatitis

Abstract

Objective: To develop non-invasive laboratory variables for the identification of esophageal varices in patients with cirrhosis at Digestive Disease Center, Shifa International Hospital, Islamabad.

Patients and Methods: All patients with chronic viral hepatitis who presented to the Gastroenterology Division between July 2002 to July 2003 were enrolled in the study. All patients with a diagnosis of Chronic Hepatitis who had platelet counts of 140,000, I.N.R of greater than 1.5 and portal vein (PV) diameter of 13mm or greater were included in the study. All patients underwent endoscopy to see the presence of varices which were graded following the standard criteria

Results: Of 140 patients, 100 (71%) were males and 40 (29%) females. Esophageal varices were present in 70% of the patients while 30% had no varices.

Conclusion: It was seen that using the standard criteria of a PV diameter 13 mm, I.N.R 1.5 and platelet counts 100,000 for the diagnosis of portal hypertension about 70% patients had endoscopic evidence of esophageal varices. As prophylactic beta blockers are recommended to reduce the chances of a bleed from esophageal varices therefore it is suggested that endoscopy should be done only in patients who fulfill the standard criteria of portal hypertension and those found to have varices should be put on beta blockers (JPMA 54:575;2004).

Introduction

Portal hypertension is a common complication of hepatic cirrhosis. Cirrhotic patients with portal hypertension develop esophageal varices and have a very high risk of variceal bleeding.1 The incidence of esophageal varices is approximately 5% per year in patients with cirrhosis2,3 and progression from small to large varices occurs in 20% after one year.4 In the two years after detection of esophageal varices, the risk of esophageal bleeding ranges from 20-30% and the mortality within one week is approximately 25-30%.2-4

In 1996, American Association for the Study of Liver Disease (AASLD) recommended that cirrhotic patients should undergo endoscopic screening for esophageal varices when there is clinical evidence of portal hypertension, i.e. platelet count=140,000 and portal vein diameter=13mm. By these parameters, we would be subjecting many patients to endoscopy.5 This becomes even more consequential in our setting due to socio economic restraints. In this background, we set to develop non-invasive tools for the detection of esophageal varices in our patient population.

Patients and Methods

This study was conducted at the Digestive Disease Center, Shifa International Hospital, Islamabad. All patients with chronic hepatitis who presented to the GI Division between July 2002 and July 2003 were enrolled in the study.

The patients were selected by the consultant gastroenterologist in the out patient department. After a detailed history and physical examination, laboratory data was collected to screen patients for GI endoscopy.

The inclusion criteria were patients with a diagnosis of chronic hepatitis with platelet counts of 140,000, I.N.R 1.5, PV diameter 13mm.

All patients with clinical ascites, diagnosis of carcinoma or documented esophageal varices were excluded from the study. Endoscopy was performed by consultant gastroenterologists and esophageal varices were graded according to the de Franchis classification defining F1 as small straight varices, F2 as enlarged tortuous varices occupying less than one third of the lumen and F3 as large coil-shaped varices occupying more than one third of the lumen.6

In order to identify the variables associated with the presence of esophageal varices, univariate analysis was performed using student's T test of independent variables. We sought the approval of the hospital ethical committee and institutional review board.

Results

There were a total of 140 patients. Of these, one hundred (71%) were males whereas 40 (29%) were females. Mean age was 45 ±8 years. Mean platelet count was 100 x 109 + 30. Mean albumin and bilirubin levels were 3.0 + 0.50 and 1.50 + 1.0 respectively. Mean portal vein diameter was 13.00 + 0.21 mm. (Table 1). Most of the patients had
hepatitis C (Table 2). Ninety eight (70%) patients had esophageal varices whereas 42 (30%) had no evidence of esophageal varices (Table 3). Sex, age and bilirubin were not associated with the presence of esophageal varies. Serum albumin, prothrombin time and platelet counts were statistically different between the two groups.

### Discussion

Severe upper gastrointestinal (UGI) bleeding as a complication of portal hypertension develops in about 30%-40% of cirrhocics. Despite significant improvements in the early diagnosis and treatment of esophagogastric variceal hemorrhage, the mortality rate of first variceal hemorrhage remains high (20%-35%).

We have demonstrated that non invasive variables can serve as reliable predictors of esophageal varices. These can be employed to screen cirrhotic patients who need to undergo endoscopic examination. This would help us evaluate the presence of esophageal varices in a selective group of patients who are candidates for primary prophylaxis.

1. Results from trials on primary prophylaxis for variceal bleeding have shown that there giving β-blocker therapy without documenting esophageal varices has no advantage. Patients who did not have esophageal varices and still were given β-blockers did not have any better outcome in terms of preventing bleeding. On the contrary, they reported more adverse events.

It has been documented that empiric beta-blocker therapy for the primary prophylaxis of variceal hemorrhage is a cost-effective measure, and the use of screening endoscopy to guide therapy adds significant cost with only marginal increase in effectiveness.

This added cost to screening endoscopy is presumably due only to the large number of unplanned endoscopies; where the endoscopic findings do not significantly alter the treatment plan.

In our study, we have shown that PV diameter of 13 mm, I.N.R. 1.5 and platelet counts 100,000 are reliable markers for predicting esophageal varices in cirrhotic patients.

Platelet count has not only been associated with the presence of cirrhosis but has also been correlated with the size of varices. In one study, the presence of any varices was associated with a platelet count of 90x103/µL whereas for large varices, a platelet count of 80x103/µL was independent risk factors associated with varices. In both cases, and advanced Child-Pugh class was an independent risk factor for the presence of varices.

Other than the platelet count, factors which have been associated with the presence of large esophageal varices are the size of spleen and presence of ascites by ultrasound. It has been shown that using mean values as cut-off points, only 12.8% with platelets 118x109/L, spleen length = 135 mm and no ascites had varices. Moreover, all these patients had small sized varices. On the other hand, 83.3% with a platelet count 118x109/L, spleen length 135 mm and ascites had varices. Moreover, 28.3% of those patients had large varices.

One might correctly conclude that low platelet count, spleen length and ascites give a fair estimate for the severity of liver disease. Therefore, the distribution of esophageal varices according to the Child-Turcotte-Pugh class has been found in Child's Class A, B and C as 35%, 60% and 69% respectively.

In conclusion, considering disease burden and economic cost of end stage liver disease treatment, it is prudent to screen patients who qualify the criteria of increased I.N.R, decreased albumin and platelet count. They shall be screened by endoscopy for the presence of esophageal varices. And if present, they shall be put on β blocker therapy. Further studies may be needed to validate our findings.

### References