Original Article

Correlation of HVPG Level with CTP Score, MELD Score, Ascites, Size of Varices, and Etiology in Cirrhotic Patients

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ABSTRACT

Background/Aim: This study intends to determine the correlation of a patient's hepatic venous pressure gradient (HVPG) measurement with six factors: Child–Turcotte–Pugh (CTP) score, model for end-stage liver disease (MELD) score, presence of ascites, size of varices, presence of variceal bleeding, and an etiology of cirrhosis. The study also aims to identify the predictors of higher HVPG measurements that can indirectly affect the prognosis of cirrhotic patients. **Patients and Methods:** Thirty patients diagnosed with cirrhosis were enrolled prospectively and each patient's HVPG level was measured by the transjugular catheterization of the right or middle hepatic vein. The wedged hepatic venous pressure (WHVP) and free hepatic venous pressure (FHVP) were measured using a 7F balloon catheter. The HVPG level was calculated as the difference between the WHVP and FHVP measurements. **Results:** The mean HVPG level was higher in alcoholic than in nonalcoholic cirrhosis (19.5 ± 7.3 vs 15.2 ± 4.5 mm Hg, $P = 0.13$). The mean HVPG was also higher in bleeders compared with nonbleeders (18.5 ± 5.3 vs 10.7 ± 3.1 mmHg, $P = 0.001$). Patients with varices had a higher mean HVPG level than those without varices (17.4 ± 5.8 vs 11.7 ± 3.9 mmHg, $P = 0.04$). The difference among the three categories of varices (small, large, and no varices) was statistically significant ($P = 0.03$). In addition, the mean HVPG level was higher in patients with ascites than in those without ascites (18.7 ± 4.7 vs 11 ± 5.3 mmHg, $P = 0.002$), and it was significantly higher in patients in CTP class C (21.8 ± 5.5 mmHg) as compared with those in CTP class B (16.9 ± 2.9 mmHg) and CTP class A (10.5 ± 4.1 mmHg; $P \leq 0.001$). **Conclusion:** HVPG levels were significantly higher in patients in CTP class C as compared with those in CTP classes A and B, thereby indicating that an HVPG measurement correlates with severity of liver disease. A high HVPG level signifies more severe liver disease and can predict the major complications of cirrhosis.

Key Words: Ascites, cirrhosis, Child–Turcotte–Pugh, hepatic venous pressure gradient, model for end-stage liver disease

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Portal hypertension in cirrhosis leads to many complications including upper gastrointestinal bleeding, hepatic encephalopathy, renal dysfunction, ascites, and peritonitis.[1] Portosystemic collaterals develop when the portal pressure gradient increases to 10 mmHg, whereas variceal bleeding occurs with a pressure gradient of more than 12 mmHg.[2,3] Many clinical and biochemical parameters indicate decompensation. Most important of these parameters are ascites, variceal hemorrhage, and hepatic encephalopathy.[4] The survival rates of patients presenting with compensated are different from those with decompensated cirrhosis.[4] The scoring systems of the model for end-stage liver disease (MELD) and Child–Turcotte–Pugh (CTP) assessments are the most common prognostic methods for the assessment of survival in cirrhotic patients. Although both scoring systems are good predictors of decompensation and survival in cirrhosis, they have a sensitivity and specificity of only 80% when used for prognostication. However, with the increasing incidence of liver transplantation, greater accuracy in prognosis assessment is necessary. Therefore, the need arises for new

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methods and variables to prognosticate cirrhotic patients. Because many of the complications in cirrhosis are due to increased portal pressure, it has been suggested that the assessment of portal hemodynamics could be helpful in predicting the course of cirrhosis. The direct measurement of portal venous pressure is invasive and inconvenient.[5] Portal venous pressure is indirectly measured by the hepatic venous pressure gradient (HVPG), which is the difference between portal vein pressure and intra-abdominal venacaval pressure.[6] Portal pressure in cirrhotic patients is clearly reflected by their HVPG levels.[5] The normal HVPG value lies between 1 and 5 mmHg. A gradient, regardless of clinical features, defines the presence of portal hypertension. Clinically significant portal hypertension (CSPH) is defined as HVPG ≥ 10 mm Hg.[7,8] We conducted this study to correlate HVPG measurements in cirrhotic patients with their CTP and MELD scores, presence and absence of ascites, size of varices, presence or absence of variceal bleeding, and the etiology of cirrhosis.

PATIENTS AND METHODS

This prospective study was approved by the institutional review board. Participants included 39 consecutive patients attending the hepatology clinic of a tertiary care institute in North India who were diagnosed with cirrhosis by clinical or biochemical, sonographic, and/or histologic findings. These patients were enrolled in the study for 12 months. Written informed consent was taken from all patients. A detailed clinical examination, baseline laboratory tests, endoscopy, and HVPG measurements were performed for all patients. Excluded from the study were patients presenting with a history of or diagnosed to have allergic reactions to contrast agents, underlying severe cardiac, respiratory or psychiatric illness, hepatocellular carcinoma, splenic or portal vein thrombosis, primary biliary cirrhosis, a prothrombin time index (PTI) of <60%, or a platelet count of <50,000 per cubic millimeter. Based on these findings, nine patients were excluded and 30 patients formed the final study group. A diagnosis of encephalopathy was made using the criteria of West Haven.[9] Varices were graded as small (≤5 mm) or large (>5 mm) according to the American Association for the Study of Liver Disease (AASLD) guidelines.[10]

Technique of HVPG measurement

Hepatic venous catheterization was done in the digital subtraction angiography (DSA) suite (Siemens, Germany or Philips, Netherlands) and pressure was measured with a Sirecust 1260 (Siemens) strain gauge transducer. The procedures were performed by authors SR, NKa, and AB. A 7F double-lumen balloon-tipped Swan–Ganz catheter was advanced into the right or middle hepatic vein through the percutaneous transjugular route. In difficult cases, a 5F multipurpose catheter first was used to cannulate the hepatic vein initially. The use of this catheter was followed by the placement of the balloon-tipped catheter over an exchange guidewire. With the balloon catheter advanced 2 cm into the right or middle hepatic vein, the free hepatic venous pressure (FHVP) was measured. With the continuous monitoring of pressure, the balloon was inflated by injecting air so as to wedge the hepatic vein and thereby measure the wedged hepatic venous pressure (WHVP). The wedged position was confirmed by gently injecting 2 mL of contrast agent through the catheter to demonstrate the retention of the contrast agent in the occluded portion of the hepatic vein. Three readings were taken for both FHVP and WHVP, and arithmetic mean was obtained. During the entire procedure, heart rate and blood pressure were constantly monitored. The HVPG level was calculated as the difference between the WHVP and FHVP readings.[11]

Statistical analysis

Statistical analysis was done using SPSS software (version 17.0; SPSS Inc., Chicago, IL, USA). Quantitative data was expressed as mean ± standard deviation and compared using Mann–Whitney and analysis of variance tests. Correlation between variables was analyzed using Spearman's correlation test. Univariate and multivariate Cox regression analyses were performed to assess the variables predicting a higher HVPG level. Receiver operating characteristics (ROC) curve was plotted to determine the optimum HVPG level predicting variceal bleeding. Statistical significance was defined as $P \leq 0.05$.

RESULTS

The study group included 25 male and 5 female patients in the age range of 18–67 years. The mean age of the study group was 46.1 years. Table 1 summarizes the demographic profile of the patients.

HVPG and etiology of cirrhosis

The mean HVPG level was slightly higher in alcoholic cirrhotics as compared with viral and other etiologies, although none of the higher HPVG levels were found to be statistically significant ($P = 0.07$). Furthermore, the mean HVPG level was higher in alcoholics (19.5 ± 7.3 mmHg) than in nonalcoholics (15.2 ± 4.5 mmHg). However, none of the higher HVPG levels were found to be statistically significant ($P = 0.13$).

HVPG and ascites

Ascites was seen in 22 (73.3%) of the 30 patients. Fourteen patients had a mild grade and eight had a severe grade of ascites. The mean HVPG level was significantly higher in patients with ascites than in those without ascites (18.7 ± 4.7 vs 11 ± 5.3 mmHg, $P = 0.002$). In addition,

the difference in the mean HVPG levels between patients with mild and severe grades of ascites was statistically significant ($P = 0.04$) [Figure 1].

HVPG and encephalopathy

The mean HVPG level in patients with mild encephalopathy was not significantly different from those without encephalopathy (17.3 ± 6.1 vs 15.8 ± 6.5 mmHg, $P = 0.8$).

HVPG and bleeder status

Out of the 30 patients, 23 (76.7%) had variceal bleeding. The mean HVPG level was higher in bleeders than in nonbleeders (18.4 ± 5.2 vs 10.7 ± 3.1 mmHg) and the difference was statistically significant ($P = 0.001$) [Figure 2].

HVPG and variceal size

Esophageal varices were present in 26 out of 30 (86.7%) patients. Of the 26 patients with esophageal varices, 18 had small varices and 8 had large varices. The measured mean HVPG level was higher in patients with large varices (20 ± 7 mmHg) than in patients with small varices (16.2 ± 4.9 mmHg) and those without varices (11.7 ± 3.8 mmHg) [Figure 3]. Overall, the difference among the three categories (small, large, and no varices) was statistically significant ($P = 0.03$). However, the difference between patients with small varices and those with large varices was found to be statistically insignificant ($P = 0.06$). When the mean HVPG level in patients with varices (17.4 ± 5.8) was compared with patients without varices (11.7 ± 3.8), the difference was statistically significant ($P = 0.04$).

Table 1: Demographic profile

Total number of patients	**30**
M:F	5:1
Etiology	
Alcoholic	10 (33.3)
HBV	7 (23.3)
HCV	5 (16.7)
Cryptogenic	6 (20.0)
Miscellaneous	2 (6.7)
Ascites, *n* (%)	22 (73.3%)
Encephalopathy, *n* (%)	7 (23.3%)
Variceal bleeding, *n* (%)	23 (76.7%)
CTP class	
A	8 (26.7%)
B	13 (43.3%)
C	9 (30%)
CTP score (mean)	8.0±2.1
MELD score(mean)	13.4±4.8
Varices	
None	4 (13.3)
Small	18 (60)
Large	8 (26.7)

CTP: Child-Turcotte-Pugh; HBV: Hepatitis B virus; HCV: hepatitis C virus; MELD: Model for end-stage liver disease

HVPG and CTP class

The mean HVPG level was significantly higher in patients with CTP class C (21.7 ± 5.4) than in those with CTP class B (16.9 ± 2.8) and CTP class A (10.5 ± 4.1). The difference was found to be highly significant ($P \leq 0.001$). When the CTP classes were compared in pairs, the difference between

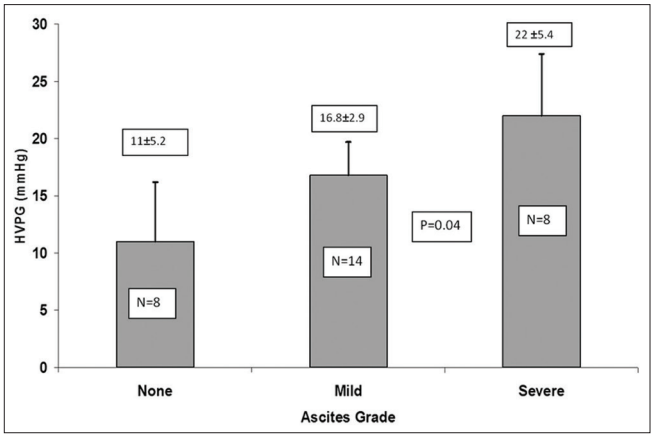

Figure 1: Mean hepatic venous pressure gradient levels (mmHg) in patients with mild, severe, and no ascites

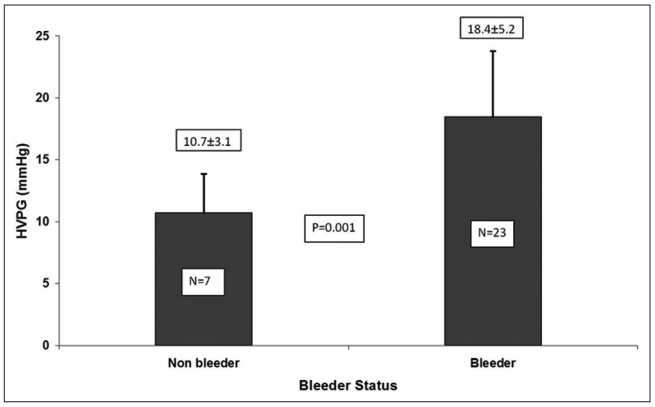

Figure 2: Mean hepatic venous pressure gradient levels (mmHg) in bleeders and nonbleeders

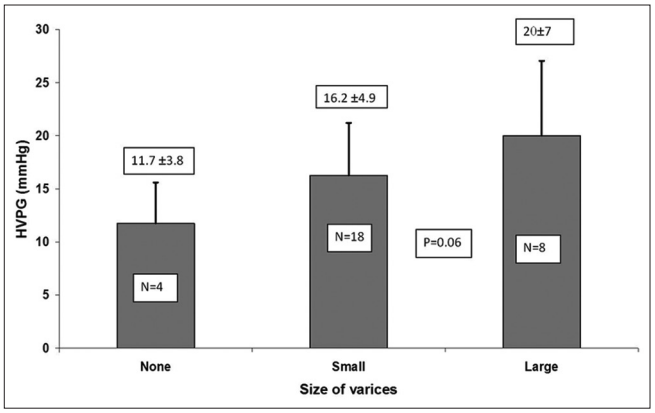

Figure 3: Mean hepatic venous pressure gradient levels (mmHg) in patients with small, large, and no varices

CTP classes A and B, and CTP classes B and C were also found to be significant statistically ($P = 0.002$ and 0.030, respectively) [Figure 4].

Variables predicting higher HVPG level: Univariate and multivariate analyses

To choose the variables that independently predict a higher HVPG level, a multivariate Cox regression analysis was applied using the variables from a univariate analysis that was shown to be significant in predicting raised HVPG levels ($P < 0.10$). The variables included etiology ($P = 0.076$), variceal bleeding ($P \leq 0.001$), variceal grade ($P = 0.099$), presence of varices ($P = 0.017$), presence of ascites ($P = 0.007$), ascites grade ($P = 0.002$), albumin ($P = 0.023$), international normalized ratio (INR, $P = 0.063$), CTP score ($P \leq 0.001$), MELD score ($P = 0.019$), and CTP class ($P \leq 0.001$). Out of the three varices-related variables, variceal bleeding was chosen because it is the most significant of the three variables. Ascites, albumin, and INR were not chosen because they are part of the CTP score and could create a confounding effect on the final model. Finally, two models were developed. Model 1 included etiology (alcoholic vs nonalcoholic), variceal bleeding, and CTP score; and model 2 included etiology (alcoholic vs nonalcoholic), variceal bleeding, and MELD score. Using this modeling strategy, variceal bleeding ($P = 0.006$) and CTP score ($P = 0.018$) in model 1 and variceal bleeding ($P \leq 0.001$) and MELD score ($P = 0.019$) in model 2 were found to be strong independent predictors of higher HVPG. Etiology was not found to be an independent predictor in either model [Tables 2 and 3].

Correlation of HVPG with various variables

Correlation between the HVPG level and each variable was assessed using Spearman's correlation test. A statistically significant strong positive correlation ($P < 0.05$) was found with variceal bleeding, presence of varices, varices grade, presence of ascites, ascites grade, INR, CTP score, CTP class, and MELD score. A strong negative correlation was seen with serum albumin [Table 4].

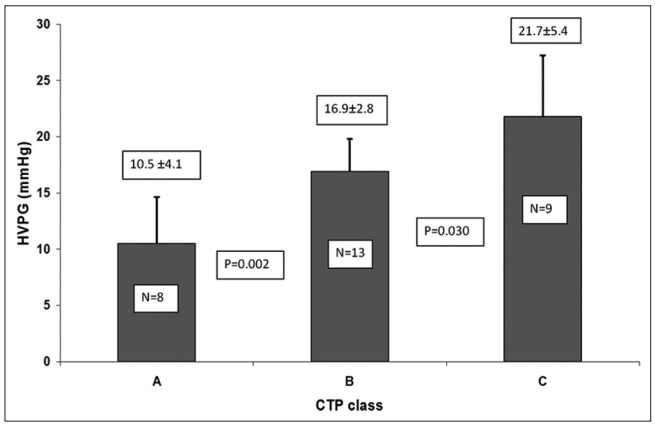

Figure 4: Mean hepatic venous pressure gradient (HVPG) levels (mmHg) in Child–Turcotte–Pugh (CTP) classes A, B, and C

ROC curve for optimum HVPG predicting variceal bleeding

ROC curves were plotted and the area under the curve (AUC) was calculated to establish the optimum HVPG level that predicts variceal bleeding. AUC was found to be 0.916 with c statistic of 0.91 which was highly significant ($P = 0.001$). The sensitivity and specificity of different HVPG values were assessed to choose the best cutoff level that could predict variceal bleeding. A cutoff value of 12.5 mmHg had a sensitivity of 87% but a specificity of only 67%. The most appropriate cutoff value was found to be 14.5 mmHg, which had a sensitivity of 83% and specificity of 100% [Figure 5].

Table 2: Multivariate analysis of variables in model 1

Variables	RR	95.0% CI		*P*
		Lower	Upper	
Variceal bleeding	0.15	0.04	0.59	0.006
Etiology	1.43	0.55	3.71	0.458
CTP score	0.74	0.58	0.95	0.018

CTP: Child-Turcotte-Pugh; RR: Relative risk

Table 3: Multivariate analysis of variables in model 2

Variable	RR	95.0% CI		*P*
		Lower	Upper	
Variceal bleeding	0.08	0.02	0.30	≤0.001
Etiology	1.72	0.69	4.29	0.243
MELD score	0.89	0.82	0.98	0.019

CI: Confidence interval; MELD: Model for end-stage liver disease; RR: Relative risk

Table 4: Spearman's correlation of variables with HVPG

Variable	Spearman's rho	*P*
Variceal bleeding	0.612	<0.001
Varices	0.369	0.022
Variceal grade	0.488	0.003
Ascites	0.576	<0.001
Ascites grade	0.648	<0.001
Encephalopathy	0.037	0.424
Albumin	−0.386	0.018
Bilirubin	0.305	0.050
INR	0.527	0.001
Creatinine	0.059	0.378
CTP score	0.666	<0.001
MELD score	0.504	0.002
CTP class	0.717	<0.001

HVPG: Hepatic venous pressure gradient; INR: International normalized ratio; CTP: Child-Turcotte-Pugh; MELD: Model for end-stage liver disease

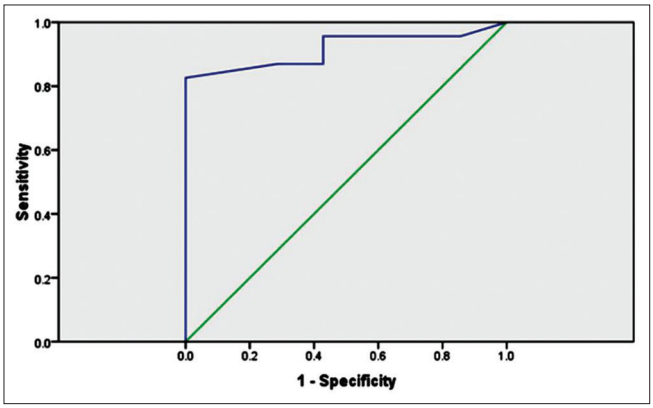

Figure 5: Receiver operating characteristics curve showing the sensitivity and specificity for hepatic venous pressure gradient >14.5 mmHg predicting variceal bleeding

DISCUSSION

HVPG measurement is done to assess the degree of portal hypertension, which is the difference between WHPG and FHVP. In clinical practice, the measurement of HVPG level is related to evaluation of the efficacy of treatment, assessment of the hemodynamic response to drug therapy, and to assess the risk of rebleeding from the periesophageal varices. Measurement of HVPG level is a reliable and a good predictive means in the management of patients with portal hypertension if performed in an appropriate manner.[8]

There is sufficient data in the literature concerning the importance of portal pressure reduction for the primary and secondary prophylaxis of bleeding varices.[12] However, the effect of HVPG values on various other factors determining the portal hypertension and chronic liver disease has not been evaluated well especially in a patient population with varying etiologies of cirrhosis.[13-15] Also, the data from the Indian subcontinent is scarce in spite of having a very high prevalence of cirrhosis in this population. We conducted our study in 30 cirrhotic patients of various etiologies. Majority of the patients (76.7%) had variceal bleeding either in the past or at the time of presentation during the study period.

The results of the present study demonstrate the importance of measuring HVPG in patients with a clinical diagnosis of cirrhosis. Compared with the patients in CTP class A, the HVPG level was significantly higher among patients in CTP classes B and C. This finding indicates that the severity of liver disease correlates with the rise in HVPG level. Patients of CTP class C had a higher HVPG level (21.8 ± 5.5 mmHg) than in patients of CTP class B (16.9 ± 2.9 mmHg), the difference being statistically significant. There was a very strong correlation between HVPG level and CTP score in our study. On univariate analysis, both CTP class and CTP score were found to be significant predictors of HVPG levels.

Conflicting data are available in the literature on portal pressure in alcoholic versus non-alcoholic cirrhosis.[16] The initial studies in alcoholic cirrhosis reported a higher portal pressure. However, this finding was subsequently refuted.[17] In our study, alcohol was the most common etiology of cirrhosis (33.3%), followed by HBV (23.3%), cryptogenic (20%), and HCV (16.7%). It was found that mean HVPG level was higher in alcoholic as compared with nonalcoholic cirrhosis. However, the difference was not found to be statistically significant. Similarly, the mean HVPG was slightly more in alcoholic as compared with viral and other etiologies. But none of them was found to be statistically significant. Hence, it may be concluded that the current consensus on the management of portal hypertension, which is mainly based on studies including patients with alcoholic cirrhosis, may also be applicable to patients with cirrhosis due to viral etiologies.

Variceal bleeding is the most important cause of morbidity and mortality in cirrhotic patients. Almost half of the cirrhotic patients develop esophageal varices, with the lifetime prevalence around 80%–90%. The most important role of HVPG level is in predicting the risk of variceal bleeding in cirrhotic patients. It is one of the areas where large number of studies have been done so far.[3,12,18-21] In our study, 26 (86.7%) patients had varices. The mean HVPG level in patients having varices (17.4 ± 5.8) was higher as compared with patients without varices (11.7 ± 3.8), the difference being statistically significant. Similarly, the mean HVPG level was found to be higher in bleeders than in nonbleeders. However, the difference between small (16.2 ± 4.9 mmHg) and large varices (20 ± 7 mmHg) was found to be statistically insignificant. It could be a type II error. Alternatively, it has been suggested that the tension in the variceal wall is the major factor, which determines the rupture of varices. The tension in the variceal wall is a combination of the variceal size and pressure. In this context, small varices may bleed at a higher pressure.[22] In our study, presence of varices and variceal bleeding were shown to predict HVPG levels in a statistically significant way on the univariate analysis. Furthermore, a statistically significant strong positive correlation was found between HVPG level with variceal bleeding and presence of varices. These observations support the basic concept that reduction in the portal pressure is required to prevent the growth of varices, and thereby the variceal bleeding in patients with portal hypertension. However, there would be little relevance to differentiate bleeders and nonbleeders in the noncirrhotic patient population, as only postsinusoidal pressure is measured by HVPG.

Another important area where the role of HVPG level has been studied thoroughly is the threshold level above which the variceal bleeding occurs. It is not practical

to perform sequential wedge pressure measurements in patients in non-specialized centers, especially if they have not bled. Therefore, there is a need for a "single shot" for prognostication purposes.[22] To develop an effective methodology, one needs threshold values, which may signify a worse prognosis such as variceal bleeding, and identify the patients who require an early intervention. Results of several randomized controlled trials, prospective cohort studies, and meta-analyses have favored the prognostic role of HVPG level for portal hypertensive bleeding showing that a reduction of HVPG level to <12 mmHg or a reduction by 20% of baseline value significantly reduces the risk of bleeding.[12,14,23] However, based on the individual needs and not on cohort observations, it may be the lowest common denominator that does not exclude other (higher) threshold gradients that might be essential to prevent the variceal bleeding in every patient. The patients who have initially very high HVPG level may respond to reduction therapy even though the threshold of 12 mmHg may not be reached. Similarly, those who have initially mildly elevated HVPG level and presenting with bleeding may not respond to treatment even when the HVPG level has been reduced to <12 mmHg.[24] In our study, the mean value of HVPG level was 16.7 mmHg, whereas the median value was 17 mmHg. We plotted ROC curves for choosing the cutoff point of HVPG level, which can predict variceal bleeding. HVPG level of >14.5 mmHg had a sensitivity of 83% and specificity of 100%. Various studies have previously reported the cutoff values in the range of 12–17 mmHg.[22,23]

One of the common causes of morbidity in the cirrhotic patients is ascites, which results due to elevation of the sinusoidal pressure. Mean HVPG level was higher in patients with ascites compared with patients without ascites. Our results show that the patients with severe ascites have a significantly higher portal pressure and HVPG level as compared with those patients without ascites, which is comparable to prior studies.[25,26] Moreover, on the univariate analysis presence as well as higher grade of ascites were shown to predict HVPG level.[26] We think that more studies evaluating the role of reduction of HVPG level in the reduction of ascites are required.

The role of HVPG level in predicting the prognosis in the cirrhotic patients is well established. Now it is one of the essential hemodynamic parameter in the evaluation of patients with cirrhosis.[14,19,27] However, there is a lack of sufficient data on the various factors independently predicting the HVPG level. In our study, two models of the different variables were developed to find the variables that can independently predict the HVPG level. In both the models, variceal bleeding was found to be a strong independent predictor of HVPG level, thus establishing the crucial relationship between the HVPG level and variceal bleeding. Both MELD and CTP scores were found to independently predict HVPG level in models 2 and 1, respectively. Our data is in accordance with the growing fact that HVPG level is a dynamic marker of the progression of the disease.[12]

Although there are a large number of studies comparing the HVPG level with CTP score, very few studies are available comparing HVPG level and MELD score.[28,29] MELD score correlates with the residual liver function and predicts the mortality across a broad continuum of liver diseases. It also has been shown to be associated with a better transplantation rate without an increase in the mortality rates for the patients waiting for transplantation. Although MELD score and HVPG level imitate the underlying severity of liver disease, the correlation between them has not been demonstrated adequately. We found that the MELD score has a positive and significant correlation with HVPG level, suggesting that both markers are substantially linked with the severity of cirrhosis. There was a strong positive correlation between HVPG level and MELD score in our study. On univariate analysis, MELD score was shown to be a statistically significant predictor of HVPG level. As of now, there is no study available that has directly compared the HVPG level and MELD scores. However, there are few studies available that have shown the role of HVPG level and MELD score in predicting hepatic decompensation and death in cirrhotic patients.[29] In our study, MELD score was found to be a significant predictor of HVPG level on univariate analysis as well as on multivariate analysis. MELD score was initially introduced as a stratifying score in the patients waiting for transplantation, and now it plays a dominant role in predicting the short-term mortality of patients with cirrhosis.[30] This further confirms the role of HVPG level in predicting the prognosis and survival in patients with cirrhosis.

Our study is not without limitations. The sample size of our study is small. Additionally, we performed a single shot baseline measurement of HVPG level, while ideally sequential measurement of HVPG level is required for the better assessment of its prognostic value. Finally, survival benefit of measuring HVPG level was not directly assessed as long-term follow up was not a part of our study.

CONCLUSION

To conclude, compared with patients in CTP classes A and B, HVPG level is significantly higher in patients in CTP class C. This indicates that the HVPG level correlates with the severity of liver disease. A higher HVPG level in patients with ascites, varices, and bleeders indicates that the HVPG level can predict the major complications of cirrhosis. MELD score is an independent predictor of HVPG level, thereby indirectly signifying the role of HVPG level in predicting

the short-term mortality. There exists no difference in the prognostic value of HVPG level between the alcohol-related and other etiologies of cirrhosis. Furthermore, large-sample studies are required to establish a more appropriate and uniform level of HVPG level to decide on the treatment options and establish a new scoring system based on HVPG level in the long term.

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