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New noninvasive method to predict the presence of Oesophageal Varices

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Abstract:

Background: Cirrhosis is characterized by tissue fibrosis and conversion of the normal liver architecture into structurally abnormal nodules. The major morbidity from liver cirrhosis is due to portal hypertension.

Variceal bleeding is one of the most serious complications of portal hypertension. UGI endoscopy is the gold-standard technique for the identification of esophageal varices, though it is invasive. Efforts were made to find a non-invasive, easily available tool to predict the presence of esophageal varices.

Methods: UGI endoscopy was done to look for esophageal varices. Patients are divided into two groups: one group with varices and the other without varices. The diameters of Portal vein and Splenic vein were measured by using ultrasound, then the Venous Diameter Ratio (VDR) was calculated.

Venous Diameter Ratio=Portal Vein Diameter ÷Spenic Vein Diameter

Further, the Venous Diameter Gradient (VDG) was also calculated.

Venous Diameter Gradient=Portal Vein Diameter-Splenic Vein Diameter

Finally, the VDG and the VDR were compared between the two groups.

Results: The mean value of VDR in patients of cirrhosis with varices was 1.33 ± 0.16 and in patients without varices was 1.73 ± 0.09. The mean value of VDG in patients with varices was 3.13 ± 1.38 and in patients without varices was 5.78 ± 0.7. The difference between the two was found to be statistically significant.

Conclusions: By measuring the diameter of portal and splenic vein by simple ultrasound, we successfully calculated the Venous Diameter Ratio and Venous Diameter Gradient. These two parameters can be used as a useful noninvasive tool to anticipate the presence of esophageal varices in liver cirrhosis.

Keywords: **ss**Portal hypertension, portal vein diameter, splenic vein diameter, Venous Diameter Ratio (VDR), Venous Diameter Gradient (VDG).

Introduction

The formation of structurally abnormal regenerative nodules leads to development of cirrhosis with a 10–year

mortality of 34–66%. [1] Due to structural and dynamic changes there is increased resistance to portal blood flow, resulting in portal hypertension with formation of venous collaterals and circulatory as well as vascular abnormalities.[2]

Portal hypertension is responsible for many complications, with variceal bleeding being one of the most severe complications of the same. [3] In compensated cirrhosis, esophageal varices are seen in 30 – 40% of the patients and in the case of decompensated cirrhosis, esophageal varies are seen in 60% - 85% of the patients.[4] The rate of development of new varices in patients with cirrhosis is approximately 8% per year. Variceal bleeding is one of the most frequent causes of death in patients with cirrhosis and portal hypertension. Approximately 30–50% of cirrhotic patients die within six weeks of the first variceal bleed [5] with an additional one-third patients dying within a year. Thus, there is a need for screening for varices in all patients with cirrhosis once the diagnosis is made. The presence of varices can be ascertained with 100% surety only through upper GI endoscopy.

In case of the absence of varices on screening, follow up endoscopy may be done in intervals of 2-3 years. The intervals can be shortened for patients with an HVPG >10 mmHg who are at a higher risk. [6,7,8,] UGI endoscopy being an invasive procedure requires trained endoscopists. Limited availability of endoscopy centers in rural areas highlights the need to find alternative easily available tools to anticipate the presence of esophageal varices in patients of cirrhosis of liver.

Methods

This prospective observational cross-sectional study was conducted in the Department of Medicine, Vardhman Mahavir Medical College and Safdarjung Hospital, New Delhi, over a period of 18 months. The study included 130 patients of cirrhosis above the age of 18 years who had given written consent. Patients who were on beta blockers or had portal vein thrombosis or extrinsic compression of portal vein were excluded from the study. After capturing the brief history & clinical examination, all the patients were subjected to UGI endoscopy for the presence of esophageal varices.

The patients were then subjected to ultrasonographic examination of abdomen. Portal vein and Splenic vein diameter were measured within 5mm of the splenomesentric confluence.

Both the VDR and the VDG were calculated.

The patients of cirrhosis were divided into two groups:

Group I **Group II**

Patients with varices Patients without varices

The ratio and gradient data measured by ultrasound were then compared between the two groups.

UGI ENDOSCOPY was done by single endoscopist by OLYMPUS GIF-H170 VIDEO ENDOSCOPE and varices were classified according to Beppu classification. USG abdomen was done by single radiologist by PHILIPS HD11XE Machine using convex 2-5 MHz probe. Patients underwent the procedure after 6 hours of fasting in supine position. Patients were sonographically evaluated on gray scale ultrasound for:

- Liver surface nodularity
- Overall course and heterogenous echo texture
- Splenic and portal vein diameter

Statistical Analysis

Categorical variables were presented in number and percentage (%), and continuous variables were presented as mean ± SD and median. The Kolmogorov-Smirnov test was used to test the normality of the data, and non-parametric tests were used if the normality was rejected. Diagnostic tests were used to calculate sensitivity and specificity of VDR and VDG to predict presence of

esophageal varices. The Receiver Operating Characteristics curve analysis was used to find out cut off points of VDR and VDG for predicting esophageal varices. A p-value of <0.05 was considered statistically significant.

Results

113 patients (86.92%) had varices (Group I) and 17 patients (13.07%) had no varices (Group II). Mean age of patients in group I & group II was 43.5 years and 41.24 years respectively. There was a male predominance in the study (80.77 % were males and 19.23% females). In our study there were 70% alcoholics, 5.38% were HbsAg positive,1.54% Anti HCV positive and none had KF ring.

Table 1: Baseline parameters in patients of cirrhosis of liver in study

	Sample size	Mean ± Stdev	Median	Min-Max	Inter quartile Range
Hb (g/dl)	130	7.5 ± 1.5	7.8	2.4-11.7	6.700 - 8.500
TLC (cu mm)	130	8357.69 ± 5377.89	6750	1200-34000	5400 – 9800
PLATELET (cu mm)	130	71610 ± 28226.84	68000	11000-146000	56000 – 89000
#S. BILIRUBIN (mg/dl)	130	1.87 ± 1.77	0.9	0.4-8.3	0.600 - 2.500
SGOT (IU/ml)	130	84.44 ± 86.23	56	22-699	43 – 89
SGPT(IU/ml)	130	69.45 ± 60.78	45	12-435	40 – 68
ALP (IU/ml)	130	125.62 ± 56.85	115.5	45-451	90 – 135
PT (sec)	130	16.39 ± 3.16	14.65	12.3-26	14.300 - 17.600
INR	130	1.5 ± 0.35	1.32	1-2.4	1.300 - 1.700
S. PROTEIN TOTAL (g/dl)	130	6.16 ± 0.66	6	4.1-8.65	5.800 - 6.800
S. ALBUMIN (g/dl)	130	3.08 ± 0.48	3.1	1.9-4.2	2.900 - 3.500
*B. UREA (mg/dl)	130	46.61 ± 11.48	45	32-94	38 – 56
S. CREATININE (mg/dl)	130	0.87 ± 0.3	0.8	0.5-2.1	0.600 – 1
Na (meq/l)	130	134.95 ± 8.4	135	112-156	132 – 142
K (meq/l)	130	3.94 ± 0.58	3.95	2.7-5.8	3.500 - 4.300
RBS (mg/dl)	130	118.78 ± 50.54	110	56-345	86 – 135

Mean value of portal vein diameter in Group I was 12.92 ± 2.17 and in Group II was 13.69 ± 1.5. The difference was not statistically significant.

The mean value of splenic vein diameter in Group I was 9.79 ± 1.86(mm) and in Group II was 7.91 ± 1.04(mm). The difference was statistically significant.

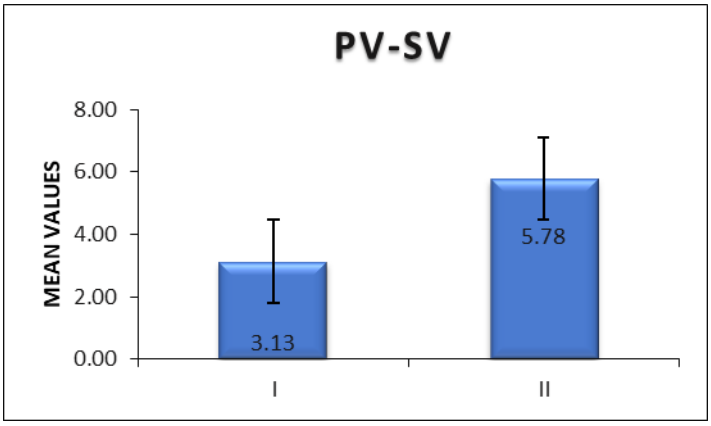

Figure 1: Graph showing comparison of mean value of portal vein and splenic vein diameter **gradient** in group I and group II.

The mean value of portal vein and splenic vein diameter gradient in Group I was 3.13 ± 1.38 and in Group II was 5.78 ± 0.7. The difference between the two was statistically significant.

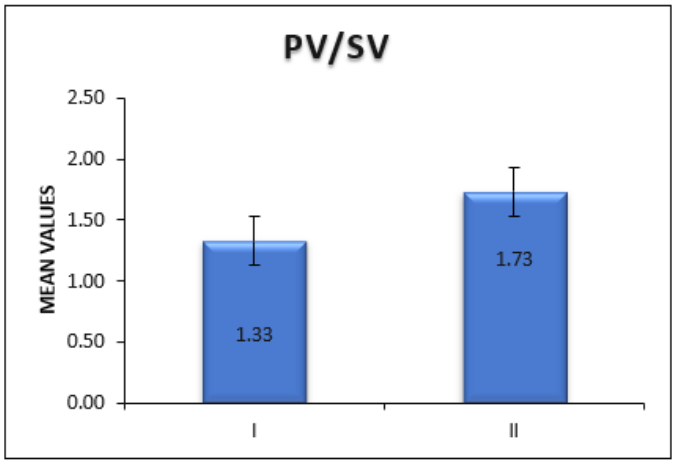

Figure 2: Graph showing comparison of portal vein diameter and splenic vein diameter ratio in group I and group II.

The mean value of portal vein diameter and splenic vein diameter ratio in Group I was 1.33 ± 0.16 & in Group II was 1.73 ± 0.09. The difference was statistically significant.

The sensitivity and specificity of PV/SV in predicting esophageal varices was 97.35% and 100% respectively. The sensitivity and specificity of PV-SV in predicting esophageal varices was 96.46% and 94.12% respectively.

Table 2: Sensitivity and specificity

	Sensitivity	Specificity	Positive predictive value	Negative predictive value
PV/SV	97.35%	100.00%	100.00%	85.00%
95% CI	92.44% to 99.45%	80.49% to 100.00%	96.70% to 100.00%	62.11% to 96.79%
PV-SV	96.46%	94.12%	99.09%	80.00%
95% CI	91.18% to 99.03%	71.31% to 99.85%	95.04% to 99.98%	56.34% to 94.27%

Table 3: Receiver operative characteristics curve analysis showing cutoff points of portal and splenic vein diameter ratio and gradient for predicting varices.

	Area under the ROC curve	Standard error	95% confidence interval	P value	Cut off point	Sensitivity	Specificity
PV/SV	0.98178	0.0125	0.941218 to 0.997167	<0.0001	≤1.58	97.35%	100%
PV-SV	0.966684	0.0161	0.919558 to 0.990224	<0.0001	≤4.8	96.46%	94.12%

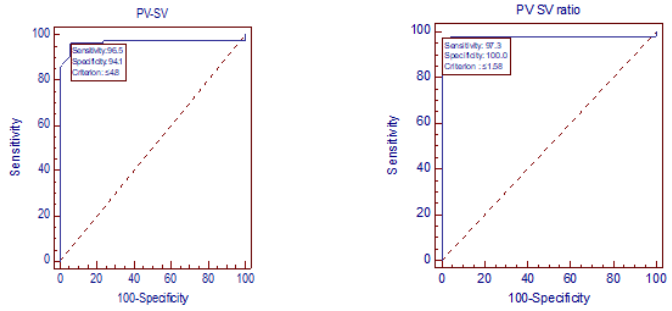

Figure 3: ROC curve

Discussion

Liver has got a dual blood supply by hepatic artery & portal vein. The portal vein carries blood from various organs like gall bladder, spleen, pancreas, esophagus, stomach, large and small intestine.

Portal hypertension is defined by an increase of HVPG of more than 6 mm Hg. The fibrosis of liver & regeneration of structurally abnormal liver nodule increases the resistance to portal blood flow. Portal hypertension may also occur due to an increase in vasoconstrictors as a result of dynamic changes.

As portal hypertension increases, the diameter of both portal & splenic vein increases. To decrease the portal pressure the collaterals between portal & systemic system open up[10] resulting in differential decompression of these veins. The decompression is more in portal vein as compared to splenic vein, resulting in decreased VDR and VDG. These two parameters can be used as tool to predict the presence of esophageal varices [10]

The results of the study collaborate to the sole study done on this subject by Vinaya Gadupati [10] et al which showed that portal and splenic vein diameter ratio as well gradient values were less in patients with varices as compared to those without varices.

However, Vinaya Gadupati used CT scan of abdomen for portal and splenic vein diameter, and we used USG abdomen. No studies based on USG abdomen could be

found to compare the results of our study.

Conclusions

The ultrasonographic evaluation of Portal vein and Splenic vein diameter gradient and ratio can be used as a non-invasive method to predict esophageal varices.

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