



A Noninvasive Panel for Diagnosis of Esophageal Varices in Patients with Compensated Cirrhosis

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Abstract

Background & Aim: Varices are present in 30-40 % of patients with compensated cirrhosis (Child-Pugh class A). Although screening endoscopy for esophageal varices (O.V.) is recommended to all patients with cirrhosis, this recommendation is not a result of evidence-based data. We studied the association of (platelet count / spleen diameter ratio, insulin resistance and splenoportal index) and the presence of O.V. in patients with compensated cirrhosis.

Patients and Methods: 124 patients with compensated liver cirrhosis due to chronic HCV were studied. After clinical, laboratory ultrasound examinations, all patients underwent screening endoscopy and O.V were reported as present or absent. According to presence or absence of varices; two groups were described. group I without varices and group II with varices.

Results: Among 124 patients with mean age of (51.81 ± 12.94) , 2 groups were described: group I (30 patients) and group II (94 patients) with a male majority (20 patients in group I and 66 patients in group II). In group I and group II: the mean platelet count/spleen diameter ratio was $(1022.6 \pm 73.36, 608.76 \pm 58.44)$ respectively, the mean insulin resistance value was $(2.426 \pm 0.618, 3.081 \pm 0.474)$ respectively. The mean splenoportal index (SPI) value was $(2.878 \pm 0.870, 6.349 \pm 0.514)$ respectively.

Conclusion: Low platelet count/spleen ratio and high SPI are very useful non invasive predictors for the presence of O.V. that could be used either separately or combined to decrease the number of upper GIT endoscopies needed in cirrhotic patients management, However insulin resistance as a non invasive predictor is still in need for further evaluation.

Keywords: Esophageal Varices, Cirrhosis, Spleno-portal index, Platelet count/spleen diameter, Insulin resistance.

Introduction

Liver cirrhosis is a condition prone to multiple complications because of portal hypertension and is the final evolutive stage of any chronic liver disease. A major complication is development of esophageal varices (O.V.) that may occur in up to 90% of cirrhotic patients. Variceal bleeding is a life-threatening event that has an incidence of 5% in patients with small O.V. and up to 15% in those with large O.V. Once bleeding occurs, mortality is around 10-20% and one-year survival is only 63% (Stefanescu et al., 2011).

Endoscopic screening of all cirrhotic patients for the presence of varices at the time of diagnosis is recommended by consensus based guidelines (Emam et al., 2009).

Approximately, 50% to 60% of cirrhotic patients presenting for initial screening upper endoscopy will have esophageal varices. Of these, only 9% to 36% of patients have been found to have varices that are large enough to warrant prophylactic β -blocker use for treatment. In light of the financial, social and medical resource burdens implicated by these recommendations, a number of attempts have been made to determine a noninvasive method of stratifying cirrhotic patients according to their risk of having varices (Schwarzenberger et al 2010). Investigators have attempted to identify characteristics that noninvasively predict the presence of varices. These studies have shown that clinical, biochemical and ultrasonographic parameters alone or together have good predictive power for noninvasively assessing the presence of O.V. (Garcia-Tsao et al., 2006 & Ismail et al., 2008).

Liu et al., 2008 noticed that, the predicted probability in patients with O.V. was a function of both increased splenic index (SI) and decreased mean portal vein velocity (PVV) and to amplify the opposite effects on which mean PVV and SI exerted, they proposed an index, the Spleno-portal index (SPI), obtained in centimeters times seconds with the following formula: $SPI = SI/PVV$ mean, where SI is in square centimeters and PVV mean is in centimeters per second (Liu et al., 2008).

Insulin resistance (IR) is exceedingly common in patients with hepatitis C virus (HCV)-related chronic liver disease and both experimental and clinical studies suggested that HCV *per se* is able to decrease insulin sensitivity. Although the underlying mechanisms linking hyperinsulinemia/IR to fibrosis are far from clear, IR has been systematically associated with advanced fibrosis and fibrosis progression in several reports. So, the quantitative measurement of IR might be a potential predictor of portal hypertension in early cirrhosis, alone or in combination with other clinical features (Camma` et al., 2009).

The aim of the study:

The present study was aimed to determine the association of three parameters (platelet count/spleen diameter ratio, insulin resistance & splenoportal index) and the presence of esophageal varices in patients with compensated cirrhosis.

Patients and Methods

This study was carried out on 124 patients who were recruited from the outpatient clinic of clinical hepato-gastroenterology department of National Liver Institute (NLI) hospital from June 2009 to October 2010. A written informed consent was obtained from all patients who presented with compensated liver cirrhosis due to chronic HCV {Child-Turcotte-Pugh (CTP) classification score: class A} and participated in the study.

Inclusion criteria: Patients were included if they had a diagnosis of HCV cirrhosis based on history, physical examination, radiological and biochemical parameters or liver biopsy.

Exclusion criteria: We excluded from our study patients with: advanced cirrhosis (CTP class B or C); other causes of liver disease or mixed causes (alcohol abuse, hepatitis B, autoimmune liver disease, Wilson's disease, hemochromatosis; α 1-antitrypsin deficiency); current or previous history of ascites or hepatic encephalopathy or portal hypertensive bleeding, hepatocellular carcinoma; portal vein thrombosis; current treatment with any

dosage of insulin or oral hypoglycemic drugs; previous or current treatment with beta-blockers, diuretics or other vasoactive drugs.

- All patients were subjected to a thorough history taking with particular attention to drug history and full clinical examination and investigations in the form of complete blood picture, liver function tests, renal function tests, fasting and 2 hours postprandial blood glucose levels and fasting serum insulin. HCV RNA was tested by qualitative polymerase chain reaction.

- Abdominal ultrasound was done and duplex Doppler examination to evaluate the (liver echotexture, portal vein flow and diameter, mean PVV, splenic vein flow, splenic dimensions; longitudinal and transverse and ascites) using the real-time ultrasound equipment TOSHIBA Xario and TOSHIBA Nemio XG with a 3.5 MHz convex array transducer which was made by TOSHIBA Corporation, Japan.

Methods

Insulin resistance (IR) was determined for every patient by the homeostasis model assessment (HOMA) method by using the following equation: Insulin resistance (HOMA-IR) = Fasting insulin ($\mu\text{U/mL}$) \times fasting glucose (mmol/L)/22.5.

The SI was calculated in square centimeters with the following formula:

$\text{SI} = a * b$, where a is the transverse diameter in centimeters and b is the vertical diameter in centimeters of the maximal cross-sectional images of the spleen.

The portal vein was longitudinally scanned. The Doppler sampling cursor was placed in the middle of the portal trunk with the width of approximately half the lumen. The mean PVV in centimeters per second was automatically measured by the machine with time-averaged velocity in two to three cardiac cycles and angle correction of less than 60° .

The SPI, obtained in centimeters times seconds with the following formula:

$\text{SPI} = \text{SI} / \text{PVV mean}$, where SI is in square centimeters and PVV mean is mean PVV in centimeters per second.

Platelet count/spleen diameter ratio was calculated in all patients as platelet count (N/mm^3)/spleen diameter (mm).

After the clinical, laboratory and US examinations, all patients underwent screening endoscopy and esophageal varices were reported as present or absent.

Both endoscopy and abdomen ultrasonography operators were blinded to the others' instrumental results and to the patients' biochemical data.

Statistical methods: The data were collected and statistically analyzed using SPSS computer program version 21. The data were expressed as mean \pm SD. P value is considered significant if <0.05 . Stepwise logistic regression analysis was performed to detect the variables that were significantly related to the presence of varices.

Results

Studying baseline clinical, biological and biochemical characteristics of the participants, different parameters and variables among 124 compensated cirrhotic patients (CTP class A) who agreed, met the inclusion and exclusion criteria and were randomly selected showed the following results:

The Age of the patients ranged from 23 to 60 years with a mean of (47.58 ± 8.4) . Our series included 38 females (30.6%) and 86 males (69.4%).

According to presence or absence of varices, two groups were described: group I (30 patients without varices) and group II (94 patients with varices) with a male majority (20 patients in group I and 66 patients in group II).

The mean value for platelet count in group I was $146.27 \pm 44.43 /\text{mm}^3$ and in group II was $73.45 \pm 16.59 /\text{mm}^3$. The mean value for fasting insulin in group I was $13.07 \pm 3.51 \mu\text{U/mL}$ and in group II was $15.67 \pm 3.49 \mu\text{U/mL}$ and that for fasting glucose in group I was $4.22 \pm 0.407 \text{ mmol/L}$ and in group II was $4.48 \pm 0.51 \text{ mmol/L}$.

The mean value for portal vein diameter in group I was $12.2 \pm 1.6 \text{ mm}$ and in group II was $13.9 \pm$

3.95 mm and that for mean portal vein velocity (MPVV) in group I was 19.65 ± 3.35 cm/sec and in group II was 14.34 ± 6.61 cm/sec. The findings of splenic dimensions revealed a mean value for transverse diameter in group I of 139.86 ± 17.64 mm and in group II of 165.40 ± 27.41 mm while that of the vertical diameter in group I was 46.8 ± 7.87 mm and in group II was 61.70 ± 14.35 mm.

We calculated the platelet count/spleen diameter ratio for all patients in both groups and the mean value in group I was 1022.6 ± 73.36 while in group II was 608.76 ± 58.44 (table 1). Using the splenic index in cm^2 and the MPVV in cm/sec, we calculated SPI for all patients in both groups and the mean value in group I was 2.878 ± 0.870 while in group II was 6.35 ± 0.51 (table 2).

With the exception of age, gender, laboratory findings as (AST, ALT, albumin, Bilirubin, Prothrombin concentration, Creatinine and fasting glucose), univariate and multivariate analysis of variables between group I and group II patients showed that higher fasting insulin, portal vein diameter, bisplenic dimensions, splenic index and Splenoportal index were found to be significantly

associated with the presence of varices ($P < 0.05$). Also, lower mean PVV, Platelets count and Platelets count/Spleen diameter ratio were found to be significantly associated with the presence of varices ($P < 0.05$).

Interestingly, multivariate logistic regression analysis showed that three features were independently linked to the presence of varices: a high SI and SPI, [odds ratio (OR), 1.800; 95% confidence interval (95%CI), 87.91-36.58; $P < 0.01$] and [OR, 3.376; 95%CI, 9.77-4.15; $P < 0.001$] respectively and a low Platelets count/Spleen diameter ratio, [OR, 3.429; 95%CI, 503.84-727.88; $P < 0.001$]. But this was not the case for IR that did not show any significant relationship [OR, 1.620; 95%CI, 2.35-1.39; $P < 0.325$].

According to the area under the receiver operating characteristic curve (AUROC); we found that cut-off value for Platelets count/Spleen diameter ratio 755, cut off value for insulin resistance 3.5 and cut-off value for SPI 3 were the best in the detection of the presence of O.V.

Table (1) The platelet count /spleen diameter ratio; Splenoportal Index; Insulin Resistance

		Patients Without Esophageal Varices (G I) n = 30	Patients With Esophageal Varices (G II) n=94
The platelet count /spleen diameter ratio (N/mm^3)/mm	Mean	1022.6	608.76
	\pm SD	73.36	18.44
	t. test	29.632	
	p. value	0.001**	
	Odds ratio	3.429	
	(95%CI)	503.8416-727.8845	
Splenoportal Index	Mean	2.878	6.349
	\pm SD	0.870	0.514
	t. test	8.526	
	p. value	0.001**	
	Odds ratio	3.376	
	(95%CI)	9.7738-4.1480	
Insulin Resistance	Mean	2.426	3.081
	\pm SD	0.618	0.474
	t. test	0.863	
	p. value	>0.05	
	Odds ratio	1.620	
	(95%CI)	2.3463-1.3945	

Table (2) the splenic index in cm²

Splenic index in cm ²	Patients Without Esophageal Varices (G I) n = 30	Patients With Esophageal Varices (G II) n=94
Mean	57.25	99.36
±SD	18.51	45.69
t. test	5.427	
p. value	0.01*	
Odds ratio (OR)	1.800	
95% confidence interval (95%CI)	87.9129-36.5826	

* Significant

Table (3) showed the sensitivity, specificity, positive predictive value, negative predictive value and accuracy of Platelet count /spleen diameter ratio, Insulin Resistance and Splenoportal index.

	Platelet count /spleen diameter ratio	Insulin Resistance	Splenoportal index
Sensitivity (%)	95.7	44.6	95.7
Specificity (%)	93.3	80	73.3
Positive Predictive Value (%)	97.8	87.5	91.8
Negative Predictive Value (%)	87.5	31.5	84.6
Accuracy (%)	96.7	53.2	90.3
P Value	0.001	0.325	0.001

Table (4) shows the sensitivity, specificity, positive predictive value, negative predictive value and accuracy when we combine (Platelet count /spleen diameter ratio & Splenoportal index), (Platelet count /spleen diameter ratio & IR) and (IR& Splenoportal index).

	Platelet count /spleen diameter ratio & Splenoportal index	Platelet count /spleen diameter ratio & IR	IR& Splenoportal index	Platelet count /spleen diameter ratio & IR& Splenoportal index
Sensitivity (%)	95.7	70.2	70.2	78.7
Specificity (%)	83.3	86.7	76.7	82.2
Positive Predictive Value (%)	94.7	94.3	90.4	93.2
Negative Predictive Value (%)	86.2	48.1	45.1	55.2
Accuracy (%)	92.7	74.1	71.7	79.5

Discussion

In our study, we had studied some biochemical and radiological parameters as well as some calculated indexes as predictors for the presence of O.V. in a series of 124 cirrhotic patients (child A) due to hepatitis C virus. Ninety-four from the 124 patients (75.8%) were proved by endoscopy to have varices.

Some other studies showed a significant relation between the presence of varices and liver profile parameters as Pilette et al.,1999, Schepis et al.,

2001, Bressler et al., 2005 and Berzigotti et al., 2008. However, all of these studies were quite heterogeneous, enrolling patients with cirrhosis of different causes (viral, alcoholic and mixed) and different disease severity (Child B or end-stage liver disease in pre-transplant series). But our patients were early cirrhotics (child A) without biochemical and clinical alterations caused by poor liver function, and all were due to HCV.

Our results showed significant inversed relation between platelet count and O.V. (P value < 0.01)

where the mean platelet count in the no varices group (I) was 146.266 /mm³ and in the varices group (II) was 73.446/mm³. These findings agreed with the findings reported by many other studies such as Chalasani et al., 1999 (346 patients) who found that a platelet count <88.000/ mm³ was an independent risk factor for the presence of large varices, that was confirmed by Sarwar et al., 2005 in their series. Zaman et al., 2001 reported that the group without varices had a higher mean platelet count (mean platelet count, 128.500/mm³) than the group with small varices (mean platelet count, 107.800/mm³) and platelet count of $\leq 90.000/\text{mm}^3$ increased the risk of having O.V. by nearly 2.5 folds. Also, Garcia-Tsao et al., 1997 (180 patients), Pilette et al., 1999 (116 patients) and Thomopoulos et al., 2003 (184 patients) reported a low platelet count to be an independent risk factor for the presence of varices.

Humera Khan and Noor ul Iman 2009, in their study, proved that thrombocytopenia is an important predictor of O.V. in patients with chronic liver disease due to infective hepatitis. Even more, they described three categories according to platelet count with a value attached and added to CPT class just like prothrombin time and international normalized ratio (platelet $\leq 150.000/\text{mm}^3 = 3$, platelet $150.000-200.000/\text{mm}^3 = 2$, platelet $>200.000/\text{mm}^3 = 1$). Logistic regression analysis of our radiological data proved a significant relation between varices and PV diameter, mean PVV and bisplenic dimensions ($P < 0.05$). The mean PV diameter in the varices group in our study was 13.9 mm while in the no varices, it was 12.2 mm. The same data were reported by Giannini et al., 2003, and Gill et al., 2004 where they reported that PV diameter of 13 mm is a reliable marker for predicting O.V. in cirrhotic patients. Also, Nashaat et al., 2010 reported the best cut-off value for PV diameter of 13.5 mm and Cherian et al., 2011 confirmed a PVD of >13 mm for small and > 14 mm for large O.V.

As regard the splenic dimensions, the mean value of bipolar (transverse) diameter in the no varices

group (I) was 139.8 mm and in the varices group (II) was 165.4 mm while the mean value of the vertical diameter was 46.8 mm in group (I) and 61.7 mm in group (II). These data were in agreement with Amarapurkar et al., 1994 who reported that splenomegaly alone was a significant predictor for the development of large O.V., Chalasani et al., 1999 who reported that splenomegaly is recognized as one of the diagnostic signs of cirrhosis and portal hypertension. Sharma et al., 2007 in a prospective study, observed that splenomegaly was the independent predictor for the presence of large varices, Nashaat et al., 2010 reported a best cut-off value for the transverse splenic diameter >145 mm and Cherian et al., 2011 reported a mean bipolar splenic dimension of >160 mm as an independent predictor of large O.V.

It was proved by Bolondi et al., 1991 and Aiello et al., 1993 that splenomegaly was found more frequently in post hepatitic cirrhosis than in alcoholic cirrhosis and this difference was more pronounced in patients with CPT class A or B.

We can explain the finding that splenic size in our study was larger than the above-mentioned studies because the etiology of cirrhosis in our series was HCV and all of our patients were CTP class (A) while the etiology was mixed in those studies with different disease stages. Agreeing with us in this finding was the series of Camma` et al., 2009 due to similarity in the etiology and disease stage.

In our study, univariate analysis showed a significant inversed relation between the mean PVV and presence of O.V. The mean value of mean PVV in the varices group (II) was 14.3 cm/sec and in the no varices group (I) was 19.6 cm/sec. These findings were in consistence with the findings of many other studies such as Korner 1996, Erdozain et al., 2000, Yin et al., 2001 and Liu et al., 2008, who all reported that mean PVV is inversely related to portal pressure and the presence of O.V.

Pathogenesis of thrombocytopenia includes productive, consumptive or distributional mechanisms. It is commonly believed to be due to

pooling and destruction of platelets in the spleen which may be mediated by platelet-associated IgG. Reduced levels of thrombopoietin either due to impaired production or rapid degradation may also add to thrombocytopenia. Thus, platelet count depends on multiple factors, not just portal hypertension (Thabut et al., 2003). The platelet count/spleen diameter ratio was proposed by Giannini et al., 2003 to be the appropriate parameter to be used as splenomegaly is implicated in thrombocytopenia of cirrhosis with spleen size being inversely correlated with platelet count. The use of this ratio normalizes platelet count to splenic sequestration since platelet count alone may be misleading and cannot be solely attributed to portal hypertension. In our study, the multivariate analysis confirmed the independent association of a lower platelet count/spleen ratio with the presence of O.V.

The platelet count/spleen ratio cut-off value with the best sensitivity and specificity for the diagnosis of O.V. identified by Giannini et al., 2003 & 2006 was 909. Agha et al., 2009 from Pakistan and Sarangapani et al., 2010 from India made identical observations. Also, Emam et al., 2009 reported a cut-off value of 900, Nashaat et al., 2010 reported a cut-off value of 820 and Esmat and Omran 2011 reported a cut-off value of 1326 due to the different disease stages and different etiologies of cirrhosis (mixed etiology). In our study, the best cut-off value was 755. Camma` et al., 2009 in their cohort, identified a value of 792 as the best cut-off. Also, Sen and Griffiths, 2008 found the platelet count/spleen diameter ratio of ≤ 650 as a sensitive non-invasive marker and Cherian et al., 2011 identified a value of ≤ 666 as best cut-off, Virus C hepatitis cirrhosis is associated with larger splenic size as proved before and this may explain the lower values of platelet count/spleen diameter ratio in these studies.

In our study, the mean value of the platelet count/spleen diameter ratio in group I was 1022.6 ± 73.36 while in group II was 608.76 ± 58.44 and we found the best cut-off value to be 755 and the

area under receiving operator curve (AUROC) was 0.938. As independent predictor for varices in our study, the platelet count/spleen diameter ratio revealed sensitivity of (95.7%), specificity of (93.3%), positive predictive value of (97.8%), negative predictive value of (87.5%) and accuracy (96.7%). This is in agreement with Giannini et al., 2003 and Wolf 2004 both reported that the platelet count/spleen diameter ratio has a diagnostic accuracy of 92% as a noninvasive parameter in detection of the presence of O.V. Sarangapani et al., 2010 reported sensitivity of (88.5%), specificity of (83%), positive predictive value of (83.5%), and negative predictive value of (90.5%) for the platelet count/spleen diameter ratio in the diagnosis of large O.V. Also, Emam et al., 2009 reported a diagnostic sensitivity of (98%) with specificity of (64%), positive predictive value of (83%), negative predictive value of (94%) and accuracy of (86%) for the predictor platelet count/spleen diameter ratio.

In this study, we did evaluate the predictor introduced by Camma` et al., 2009 which is the association of IR, regardless of diabetes, with the presence of O.V. None of our 124 patients was found to be diabetic. We calculated IR by the HOMA method for all patients in both groups and the mean value in group I was 2.426 ± 0.618 while in group II it was 3.081 ± 0.474 . Univariate and multivariate logistic regression analysis of our data did not show a significant relationship between IR and esophageal varices P value = 0.325 [odds ratio (OR), 1.620; 95% confidence interval (95%CI), 2.3463-1.3945;], the AUROC was 0.598. Camma` et al., 2009 identified an HOMA-IR score of greater than 3.5 as the cut-off value with the best sensitivity and specificity for predicting O.V. presence (sensitivity, 61%; specificity, 76%; positive likelihood ratio, 2.58; negative likelihood ratio, 0.51). In our study, we used the same cut-off value and we reported a sensitivity of (44.6%), specificity of (80%), positive predictive value of (83.5%), negative predictive value of (31.5%) and accuracy of (53.2%).

The difference between our study and that of Camma` et al., 2009 is that none of our patients were diabetics and whey the IR was higher in their study than ours may agree with the view of Dogru et al., 2009 in their comment on the study of Camma` where we noticed that most of the study participants were overweight and some of them were even obese. Obesity is a strong risk factor for DM and also for IGT (impaired glucose tolerance). Although it was stated in the article that 26% of the patients had diabetes at baseline, there is no information regarding the glucose tolerance status of the other subjects.

Our study and the study of Camma` are cross-sectional studies but not a longitudinal follow-up study and were limited by the variability and severity of the liver disease. Therefore, further studies are needed to evaluate in cirrhosis of different stages and etiologies the usefulness of IR and changes in IR over time in predicting not only the presence of varices but also the development of varices and in correlating them to changes in hepatic venous pressure gradient over time.

We also evaluated the predictor introduced by Liu et al., 2008 that is the SPI. Liu et al., on their study of 383 patients with compensated cirrhosis to evaluate the usefulness of Duplex Doppler ultrasonography in predicting the presence of varices noticed after logistic regression analysis of their data that the predicted probability in patients with O.V. was a function of both increased SI and decreased mean PVV and to amplify the opposite effects on which mean PVV and SI exerted, they proposed an index, the SPI, obtained in centimeters times seconds with the following formula: $SPI = SI/PVV$ mean.

In our study, SPI revealed AUROC of 0.865. However, multivariate logistic regression analysis for the SPI showed odds ratio (OR) of 3.376; (95%CI), 9.7738-4.1480 and P value of 0.001. In our series at a cut-off value of 3: the sensitivity was (95.7%), specificity (73.3%), positive predictive value (91.8%), negative predictive value (84.6%) and diagnostic accuracy was (90.3%). Our findings confirmed the findings of

Liu et al., 2008 where univariate and multivariate logistic regression analysis proved more significant association with varices in SPI ($P=0.001$) than in SI ($P=0.01$) and mean PVV ($P=0.04$). Also, the diagnostic accuracy of SPI in our series (90.3%) is comparable to that of Liu et al., 2008 (93%).

In conclusion of our study, we calculated the sensitivity, specificity, positive predictive value, negative predictive value and accuracy when we combined the main predictors in our series with one another or all of them as follow (platelet count /spleen diameter ratio & SPI), (platelet count /spleen diameter ratio& IR), (IR and SPI) and (the three combined) where we found that:

For platelet count/spleen diameter ratio & SPI, the sensitivity was (95.7%), specificity (83.3%), positive predictive value (94.7%), negative predictive value (86.2%) and accuracy (92.7%).

For platelet count/spleen diameter ratio & IR, the sensitivity was (70.2%), specificity (86.7%), positive predictive value (94.3%), negative predictive value (48.1%) and accuracy (74.1%).

For IR & SPI, the sensitivity was (70.2%), specificity (76.7%), positive predictive value (90.4%), negative predictive value (45.1%) and accuracy (71.7%).

For the three predictors combined, the sensitivity was (78.7%), specificity (82.2%), positive predictive value (93.2 %), negative predictive value (55.2%) and accuracy (79.5%).

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List of Abbreviations

SI	Splenic Index
AST	Aspartate Aminotransferase
ALT	Alanine Aminotransferase
γ -GT	Gamma-Glutamyl Transpeptidase
O.V.	Oesophageal Varices
SPI	Spleno-Portal Index
PVV	Portal Vein Velocity
HCV	Hepatitis C Virus
GIT	Gastro-Intestinal Tract
NLI	National Liver Institute
IR	Insulin Resistance
CTP	Child - Turcotte – Pugh
MELD	Model for End stage Liver Disease
AUROC	Area Under the Receiver Operating characteristic Curve

Key points:

- Approximately, 50%- 60% of cirrhotic patients presenting for initial screening upper endoscopy will have varices, only 9% to 36% of patients have large varices that need prophylactic β - blocker for treatment.
- In our series:
 1. For platelet count /spleen diameter ratio & SPI, the sensitivity was (95.7%), specificity (83.3%) and accuracy (92.7%).
 2. For platelet count/spleen diameter ratio & IR, the sensitivity was (70.2%), specificity (86.7%) and accuracy (74.1%).
 3. For IR & SPI, the sensitivity was (70.2%), specificity (76.7%) and accuracy (71.7%).
 4. For the three predictors combined, the sensitivity was (78.7%), specificity (82.2%) and accuracy (79.5%).