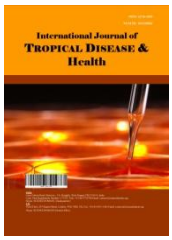

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Platelet Count/Spleen Diameter Ratio as a Non-invasive Parameter in the Prediction of Esophageal Varices in Patients with Liver Cirrhosis

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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ABSTRACT

Background: Esophagogastroduodenoscopy (EGD) is the gold standard for detecting oesophageal varices (OVs) in cirrhotic patients. However, due to the possible limitations of EGD, there has been much interest in the use of non-invasive techniques for this purpose. This study aimed to evaluate the use of platelet count/ spleen diameter ratio (PC/SD) in the prediction of the presence and grading of OVs in cirrhotic patients.

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Methods: One hundred cirrhotic patients were included in this cross-sectional study and subjected to EGD after informed consent. Either absence or the grade of OVs if existent was correlated with values of the PC/SD ratio. Univariate and multivariate analyses of data and areas under the receiver operating characteristic curve (AUC) were used.
Results: The PC/SD ratio was a good indicator in predicting the development of OVs (AUC of 0.897) with cut-off values of (987.28). Also, it correlated well with grades of oesophageal varices, a significant stepwise progressive decrease in PC/SD ratio was recorded through the grades of oesophageal varices as follows: Mean ± SD (882.59±390.43) (603.33±266.99) (503.76±190.80) (439.69±22.51) for grades I, II, III and IV respectively ($p < 0.002$),(AUC=0.688, 0.764, 0.795, 0.849) with a cut-off value of (784.37, 640.27, 597.50, 462.00) in grades I, II, III and IV respectively.
Conclusion: The PC/SD ratio could be considered a non-invasive method of choice for screening OVs, sparing EGD for patients in need of intervention.

Keywords: Esophagogastroduodenoscopy; oesophageal varices; cirrhotic patients; platelet count/ spleen diameter ratio.

1. INTRODUCTION

"Liver cirrhosis is a common consequence of the long clinical course of all chronic liver diseases and is characterized by tissue fibrosis and the conversion of normal liver architecture into structurally abnormal nodules" [1]. "Portal hypertension commonly accompanies the presence of liver cirrhosis, and the development of esophageal varices (OVs) is one of the major complications of portal hypertension" [2].

Fibrosis describes the encapsulation of injured hepatic tissue by a collagenous scar [3]. "While cirrhosis is an advanced stage of liver fibrosis and is accompanied by distortion of the hepatic vasculature. The resultant vascular distortion leads to the shunting of the portal and arterial blood supply directly into the hepatic outflow (central veins), compromising the exchange between hepatic sinusoids and the adjacent hepatocytes" [4]. "Moreover, liver cirrhosis is the major risk factor for the development of hepatocellular carcinoma (HCC), as more than 80% of HCCs develop on a fibrotic or cirrhotic background" [5].

Esophagogastroduodenoscopy (EGD) is the gold standard for the detection and grading of PH-related complications such as gastroesophageal varices GOVs, ectopic varices (EcV), and portal hypertensive gastropathy PHG as previously mentioned, and is used also for therapeutic intervention [6].

"During liver cirrhosis, splenomegaly and hypersplenism are relatively sub-fatal complications in the absence of bleeding varices. Splenic enlargement is one of the most palpable abnormalities accompanying liver cirrhosis and frequently occurs in parallel with hypersplenism, which is thought to be a major cause" [7].

"Thrombocytopenia is a frequent complication in patients with cirrhosis. As many as 84% of patients with cirrhosis have thrombocytopenia, and it is an independent variable indicative of advanced disease and poor prognosis" [8].

1.1 Aim of the Work

So, this study aimed to evaluate the value of platelet count/spleen diameter ratio as a non-invasive parameter to predict the presence of oesophageal varices in cirrhotic patients.

2. METERIALS AND METHODS

The present study is a cross-sectional study conducted on a total of one hundred patients diagnosed to have liver cirrhosis of different causes. The patients were recruited between" February 2021 to August 2021" from the outpatient clinic and internal ward of the Gastroenterology and Hepatology Unit, at Tanta University Hospitals.

2.1 Inclusion Criteria

Adult patients (more than 18 years) with cirrhotic liver whatever the etiology and divided into 2 groups (**group A: 63** cirrhotic patients with oesophageal varices and **group B: 37** cirrhotic patients without varices).

We excluded patients less than 18 years, patients with active upper GIT bleeding, patients with hepatic encephalopathy, patients are known to have OVs with previous endoscopy (either underwent band ligation or sclerotherapy),

patients with a history of partial splenic embolization or splenectomy, patients with HCC, patients have transjugular intrahepatic portosystemic shunt TIPS, patients with portal vein PV thrombosis confirmed by ultrasound US and color doppler study, patients with a history of any liver surgery, and patients on NSBBs.

2.2 Methods

All patients in this study were subjected to full history taking and full clinical examination. Whole blood samples were collected from all patients.

Routine Laboratory tests were done such as CBC, AST, ALT, T.bilirubin, serum albumin, and INR.

Pelvic-Abdominal ultrasonography and upper GIT endoscopy.

- Esophageal varices were graded as I-IV, using the Paquet grading system [9].

• **Grade 0:** No varices.
• **Grade I:** Varices, disappearing with air insufflation.
• **Grade II:** Larger, clearly visible, usually straight varices, not disappearing with air insufflation.
• **Grade III:** More prominent varices, locally coil-shaped and partly occupying the lumen.
• **Grade IV:** Tortuous, sometimes grape-like varices occupying the esophageal lumen.

- **Assessment Child-Pugh score:** Child A= 5-6 points, Child B= 7-9 points, Child C= 10-15 points [10].
- **MELD score (Model for End Stage Liver Disease):** The original MELD score is calculated using the following formula: MELD Score = 9.57 x Log_e (creatinine mg/dL) + 3.78 x Log_e(bilirubin mg/dL) + 11.2 x Log_e(INR) + 6.431 [11].
- **The FIB-4 index:** [age (years) × AST (U/L) / platelet (PLT) (109/L) × √ALT(U/L)] [12].
- **Aspartate-aminotransferase-to-platelet-ratio index (APRI):** [(AST/upper limit of the normal AST range) X 100]/Platelet Count [13].
- **Aspartate aminotransferase-to-alanine aminotransferase (AST-to-ALT) ratio (AAR) [14].**
- **Specific investigations:** Platelet-count-to-spleen-diameter (PC/SD) ratio=Platelet count (mm3)/ maximum bipolar diameter of the spleen (mm) [15].

2.3 Statistical Analysis of the Data

Data were fed to the computer and analyzed using IBM SPSS software package version 20.0. (Armonk, NY: IBM Corp) Qualitative data were described using numbers and percentages. The Kolmogorov-Smirnov test was used to verify the normality of distribution. Quantitative data were described using range (minimum and maximum), mean, and standard deviation. The significance of the obtained results was judged at the 5% level. The following tests were used:

Chi-square test: for categorical variables, to compare between different groups.
Fisher's Exact or Monte Carlo correction: correction for chi-square when more than 20% of the cells have an expected count of less than 5.
Standard student "t-test": a test of the significance of the difference between two means.
F-test (ANOVA): for normally distributed quantitative variables, to compare between more than two groups and Post-Hoc test (Tukey) for pairwise comparisons.
ROC-curve: Receiver Operating Characteristic curve analysis.
Regression analysis: by binary logistic regression models.

3. RESULTS

Laboratory and clinical investigations were shown in Tables 1, 2.

Table 1 showed that there was no statistically significant difference between the studied groups as regards age, sex, and etiological factors of liver cirrhosis. However, HCV infection was the most common cause of liver cirrhosis in the studied patients. There was a statistically significant difference in Clinical data between the two studied groups as regards ascites jaundice and LL edema.

Table 2 showed that hemoglobin and platelets were lower in group A with a statistically significant difference. Total bilirubin and INR were significantly higher in group A. While serum albumin was significantly lower in group A.

There was a significant difference between the studied groups as regards grading of ascites, portal vein diameter, and spleen diameter by ultrasound. Also, most cases of varices were in advanced Child grade.

Table 1. The clinicopathologic characteristics of patients

Groups / Variables	Group A (n= 63)	Group B (n= 37)	P-Value
Age (years)	56.37± 9.91	54.6 ± 6.35	0.34(a)
Sex	29 (46.03%)	15 (40.54%)	0.89(b)
Male	34 (53.97%)	22 (59.46%)	
Female			
HCV	51 (81%)	32 (86.5%)	0.477(b)
HBV	2 (3.2%)	1 (2.7%)	1.00(c)
NAFLD	2 (3.2%)	2 (5.4%)	0.658(c)
Mixed Bilharzial and HCV	6 (9.5%)	1 (2.7%)	0.812(c)
Autoimmune hepatitis (AIH)	2 (3.2%)	1 (2.7%)	0.651(c)
Ascites clinically	36 (57.14%)	9 (24.32%)	0.001*(b)
Palpable liver	0 (0%)	1 (2.7%)	0.370(c)
Palpable spleen	31 (49.2%)	16 (43.24%)	0.564(b)
Jaundice	23 (36.5%)	5 (13.51%)	0.013*(b)
LL edema	36 (57.14%)	0 (0%)	0.001*(b)

Group A: *cirrhotic patients with esophageal varices,* ***Group B:*** *cirrhotic patients with no varices,* ***HCV:*** *hepatitis C virus,*
HBV: *hepatitis B virus,* ***NAFLD:*** *non-alcoholic fatty liver disease,* ***AIH:*** *autoimmune hepatitis,* ***LL:*** *lower limb,*
(a): *student t-test,*
(b): *Chi-square test,* ***(c):*** *Fisher's Exact test,* **Statistically significant at p ≤ 0.05*

Table 2. The clinicopathologic characteristics of patients

Groups / Variables	Group A (n= 63)	Group B (n= 37)	P-Value
Hb (g/dl)	9.22 ± 1.67	11.50 ± 1.65	0.001 *(a)
TLC /(mm3)	4309.52 ± 1553.42	4913.51 ± 1622.78	0.067(a)
Platelets /(mm3)	100349.21 ± 35349.94	183348.35 ± 45931.14	0.001 *(a)
AST (U/L)	35.57 ± 13.78	33.43 ± 10.51	0.385(a)
ALT (U/L)	31.73 ± 13.48	31.35 ± 7.54	0.857(a)
T. Bilirubin (mg/dl)	2.40 ± 1.15	1.56 ± 0.87	0.001 *(a)
S. Albumin (g/dl)	2.81± 0.62	3.65 ± 0.51	0.001 *(a)
INR	1.58 ± 0.25	1.18 ± 0.22	0.001 *(a)
Ascites U/S			0.001 *(b)
No	15 (23.8%)	24 (64.9%)	
Mild	15 (23.8%)	2 (5.4%)	
Moderate	26 (41.3%)	11 (29.7%)	
Marked	7 (11.1%)	0 (0%)	
PV diameter (mm)	15.88 ± 1.45	11.95 ± 2.03	0.001 *(a)
Spleen diameter (mm)	168.84± 20.63	139.03 ± 19.08	0.001 *(a)
Child-Pugh score			0.001 *(b)
A	19 (30.2%)	24 (64.8%)	
B	26 (41.3%)	11 (29.7%)	
C	18 (28.5%)	2 (5.4%)	
MELD	16.21 ± 4.42	9.47 ± 4.12	0.001 *(a)
APRI	0.99 ± 0.66	0.50 ± 0.31	0.001 *(a)
AST/ALT	1.17 ±0.339	1.07±0.287	0.115(a)
FIB-4	3.85 ± 2.12	2.00 ± 1.47	0.000 *(a)
PC/SD	681.83± 341.00	1370.36 ± 452.61	0.010 *(a)

Group A: *cirrhotic patients with esophageal varices,* ***Group B:*** *cirrhotic patients with no varices,* ***Hb:*** *hemoglobin,* ***TLC:*** *total leucocytic count,* ***ALT:*** *alanine transaminase,* ***AST:*** *aspartate transaminase,* ***MELD:*** *model for end stage liver disease,*
APRI: *AST- to-Platelet-count ratio Index,* ***FIB-4:*** *Fibrosis-4,* ***PC/SD:*** *Platelet Count / Spleen diameter ratio,*
(a): *student t-test,* ***(b):*** *Chi-square test,* ***(c):*** *Fisher's Exact test,* **Statistically significant at p ≤ 0.05*

Table 3. Relation between esophageal varices grades and PC/SD in group A (n= 63)

PC/SD	Esophageal varices				P value
	I (n= 24)	II (n= 23)	III (n= 13)	IV (n= 3)	
Min. – Max.	280.00 – 1855.56	221.05–1133.33	103.13–781.25	421.05–464.71	0.002*
Mean ± SD	882.59± 390.43	603.33 ± 266.99	503.76± 190.80	439.69 ± 22.51	
P1		0.014*	0.004*	0.095	
P2			0.784	0.819	
P3				0.988	

*F: F for ANOVA test, P1 II: Group1 versus group 2, P1 III: Group1 versus group 3, P1 IV: Group1 versus group 4, P2 III: Group2 versus group 3, P2 IV: Group2 versus group 4, P3 IV: Group 3 versus group 4, P: Probability value for comparing the studied groups; *: Statistically significant at p ≤ 0.05*

Table 4. Probability value for sensitivity and specificity

	AUC	P	95% C.I	Cut off	Sensitivity	Specificity	PPV	NPV
MELD	0.861	0.001*	0.783 – 0.939	13.69	74.60	91.90	94.0	68.0
APRI	0.766	0.001*	0.671 – 0.862	0.77	61.90	94.60	95.10	59.30
FIB4	0.810	0.001*	0.723 – 0.898	2.09	77.60	81.10	87.50	68.20
AST/ALT	0.647	0.014*	0.539 - 0.755	1.06	61.90	59.90	72.20	47.80
PC/SD	0.897	0.000*	0.838– 0.957	987.28	83.80	81.0	72.10	89.50

***AUC:** Area Under a Curve; **P:** Probability value for comparing the studied groups' **CI:** Confidence Intervals **NPV:** Negative predictive value; **PPV:** Positive predictive value; *: Statistically significant at p ≤ 0.05*

Table 5. Roc curve value for the OVs

PC/SD	AUC	p	95% C. I	Cut off	Sensitivity	Specificity	PPV	NPV
grade I	0.688	0.859	0.616- 0.752	784.37	65.3	61.7	61.4	39.8
grade II	0.764	0.001*	0.667 - 0.862	640.27	68.8	60.9	85.5	36.8
grade III	0.795	0.001*	0.704 - 0.887	597.50	71.3	69.2	93.9	26.5
grade IV	0.849	0.040*	0.776 - 0.922	462.00	82.5	66.7	98.8	10.5

***AUC:** Area Under a Curve; **P:** Probability value for comparing the studied group's **CI:** Confidence Intervals **NPV:** Negative predictive value; **PPV:** Positive predictive value; *: Statistically significant at p ≤ 0.05*

A significant negative correlation was observed between PC/SD and esophageal varices in group A.

Table 3 showed that patients with higher grades of varices have a lower PC/SD ratio with statistical significance.

ROC curves analysis of the MELD score, APRI score, FIB4 score, and AST/ALT for detection of esophageal varices revealed an area under curve (AUC) of 0.861, 0.766, 0.810, and 0.647 respectively as well as a sensitivity of 74.6%, 61.9%, 77.6%, and 61.9% and specificity of 91.9%, 94.6%, 81.1%, and 59.9%.

While that of the PC/SD revealed an area under curve (AUC) of 0.897 as well as a sensitivity of 83.8% and specificity of 81%.

ROC curves analysis of the PC/SD to discriminate between different grades of esophageal varices revealed an area under curve (AUC) of 0.688, 0.764, 0.795, and 0.849 as well as a sensitivity of 65.3%, 68.8%, 71.3%, and 82.5% % and specificity of 61.7%m 60.9%, 69.2%, and 66.7% in grade I, II, III and IV respectively (Table 5).

4. DISCUSSION

"Variceal bleeding is the one of most dramatic complications of cirrhosis, with a mortality rate of

up to 20% in six weeks" [16]. "That is why it is mandatory to offer prophylactic measures against variceal rupture. To identify those patients at higher risk, it is traditionally recommended that every patient undergoes upper gastrointestinal endoscopy at the time of the diagnosis of cirrhosis. Although, patients with cirrhosis must have clinically significant portal hypertension before they develop(OVs)" [17].

"Bearing this in mind, it is obvious that a significant part of patients with a new diagnosis of cirrhosis will undergo endoscopy unnecessarily [18]. Moreover, endoscopy is an invasive procedure, associated with some risks (yet quite low), patient discomfort, and high costs" [19].

So, our study aimed to evaluate platelet count/spleen diameter ratio as a non-invasive predictor of esophageal varices in cirrhotic patients. This parameter was chosen as it allows us to identify the degree of thrombocytopenia which is most likely related to hypersplenism.

As shown in Table 1 as regards age and gender of this study, there was no statistically significant difference. This result came in agreement with [20] who documented no correlation between either age or gender and the presence of varices in cirrhotic patients.

In contrast to the study done by Yogananda et al. [21] among 50 patients with liver cirrhosis, 37 (74 %) had varices. Males predominance was noted [42 (84 %)].

In the present study, HCV infection was the most common cause of liver cirrhosis in the studied patients. This came in agreement with [22], who reported that "Egypt is enduring a large HCV disease burden, and is likely to be the most affected nation worldwide by this infection".

According to the clinical data in the present study, ascites, lower limb edema, and jaundice were prominent in group A (cirrhotic with varices) with statistical significance. This came in agreement with that of [23] who found that there was a significant statistical difference among studied groups regarding signs suggestive of hepatic decompensation such as jaundice, ascites, and lower limb edema. Patients with chronic liver disease (CLD) in group I with gastroesophageal and/or fundal varices had a higher incidence of these signs in comparison with patients with CLD without varices in group II.

As shown in Table 2 hemoglobin levels were significantly lower in cirrhotic patients with esophageal varices than in those without varices, this came in agreement with Gunda et al. [24] who documented a statistically significant relationship between lower hemoglobin levels and varices presence. But the study done by Sarangapani et al. [9] and Kumar et al. [25] found no statistically significant difference as regards HB levels between patients with large varices and those without varice.

There was no significance in total leucocytic count (TLC) between patients with varices and without varices, this came in agreement with the study done by Elatty et al. [26]. But in disagreement, the study done by Mahmood et al. [27] documented a statistically significant relationship between low WBC and varices presence.

In the present study, platelet count was significantly lower in patients with varices than those without varices. Our results were also in agreement with that reported by Abe et al. [28] who reported "platelet count to be an excellent parameter for detecting esophageal varices in patients with liver cirrhosis and portal hypertension. In addition, significant splenomegaly with low platelet count is considered a surrogate marker for portal hypertension".

This is also can be explained by Scharf., [29] who found that thrombocytopenia is one of the portal hypertension complications and caused mainly by splenic sequestration as a complication of portal hypertension-induced splenomegaly.

In the present study, there was no statistically significant difference between groups as regards ALTand AST. This came in agreement with [30], who documented that no significant difference was observed as regards the mean values of liver enzymes between patients with esophageal varices and those without esophageal varices.

Serum albumin was statistically significantly lower in group A than in group B this result is in agreement with [31] as serum albumin in a patient with varices had a mean value of (3.29±0.39) while those without varices had a mean value of (3.84±0.42).

In the current study, total bilirubin was found to be higher in patients with varices than in patients

without varices with statistical significance. This came in agreement with [32] who documented that a high total bilirubin level was found with varices presence. On the other hand, [33] showed no association between bilirubin level with esophageal varices.

As regards the international normalized ratio (INR), it was significantly higher in patients with varices (mean= 1.58±0.25) than without varices (mean= 1.18±0.22) p < 0.05). This can be explained by Bates et al. [34] who found that elevated INR in cirrhotic patients can be explained by the reduction of the nutritional status and impairment of fat-soluble vitamins absorption (A, D, E, K) resulting from poor appetite associated with cholestasis and portal hypertensive gastropathy. As a result, patients with cirrhosis and portal hypertension have reduced levels of vitamin K–dependent coagulation factors (II, VII, IX, and X). This finding is against that found by Chandail et al. [35] who studied non-invasive markers for the prediction of OVs including INR, and found no significant difference in INR for the prediction of small or large OVs. As this study failed to draw association between two groups of patients(patient with varices and those without) as regards with INR on multivariate analysis and found that only two variables namely portal vein size and spleen diameter were found to be independent predictors of esophageal varices with a significant association.

As shown in Table 2 the findings of the studied groups revealed that there was significantly higher (P- < 0.000) mean values of spleen diameter, portal vein diameter, and ascites between both groups A and B. This came in agreement with the study of Faheem et al. [32] who found that portal vein diameter and spleen diameter which are indirect predictors of portal hemodynamics can be used effectively as a screening test without subjecting patients to EGD.

The studies done by [36,37] documented that, “splanchnic vessels vasodilatation is promoted by local over-production of vasodilators, along with intrinsic vascular hypo-contractility allowing increased blood flow through the splanchnic vessels. So, splenomegaly in portal hypertension appears initially as venous congestion and structural hyperplasia with pooling of the blood and finally as an overflow related to the hyperdynamic circulation associated with portal hypertension”.

In the present study, ascites were predominant in cases with varices (63.33%) when compared to cases without varices (33.33%).

These results were in agreement with [38] who reported that; ascites were significantly increased in cases with varices when compared to cases without varices (p 0.008), and spleen size was significantly higher in cases with varices than those without (p 0.001).

~~C~~hild score which was significantly higher in patients with esophageal varices than those without esophageal varices. A similar finding was reported by Elsalakawy et al. [39] that showed a statistically significant difference between Child-Pugh classes as in class A, 91.7% showed no varices, Whereas, in class B, 41.9% showed grade II esophageal varices. In contrast, patients in class C showed grade IV in 57.8%. The variceal presence correlates with the severity of liver disease as stated by [9].

Results of the present study revealed that the platelets count/spleen diameter (PC/SD) ratio was lower in group A (patients with varices) than in group B (patients without varices) (681.83±341.00 VS 1370.36±452.61) with sensitivity and specificity in prediction of OVs (83.80% and 81.0% respectively), PPV 72.10%, NPV 89.50%, and proportion of AUC 89.7% at cut-off value (987.28). These results were in agreement with [40] who found that the cut-off point of (909) had 82.5% sensitivity and 92.6% specificity for the prediction of the presence of OVs, PPV86.3%, NPV90.4% and they also found that direct correlation between low platelet count/spleen diameter ratio and the grade of OVs with a high statistical significance (p 0.0001). Also, the study done by Kothari et al. [41] found that for the prediction of esophageal varices, the PC/SD ratio was significant and showed an area under the curve of 65.6% at a cut-off of <997.

“The high diagnostic accuracy of the PC/SD ratio for varices can be explained as follows: varices and hypersplenism are the results of portal hypertension. The platelet count can be influenced by many factors in cirrhotic patients other than hypersplenism. The decrease in thrombopoietin production may be the reason. Thrombopoietin is mainly produced by hepatocytes and the quantity can be largely reduced when the hepatocytes are damaged. In addition, the shortened platelet mean lifetime and myelotoxic effects of alcohol or hepatitis viruses lead to thrombocytopenia. Splenomegaly is the

clinical manifestation of hypersplenism. Thus, a combined index of platelet count and spleen diameter has much more relevance with portal hypertension and varices than the sole decreased platelet count" [42].

In the present study, a significant stepwise progressive decrease in PC/SD ratio was recorded through the increasing grades of esophageal varices mean ± SD (882.59±390.43) (603.33±266.99) (503.76± 190.80) (439.69±22.51) for grade I, II, III and IV respectively (p 0.002), (AUROC= 0.688, 0.764, 0.795, 0.849) with a cut-off value of (784.37, 640.27, 597.50, 462.00) in grade I, II, III and IV respectively. These results agreed with [38] who found that the mean ±SD of PC/SD ratio was(725.6 ± 273.5) (567.9 ± 280.2) (347.8 ± 162.6) (293.8 ± 91.8) in grades I, II, III and IV respectively as well (p 0.001).

In contrast, [21] concluded that the PC/SD ratio might not be accurate enough in predicting the presence of oesophageal varices. The evidence is not sufficient enough to replace endoscopy as a screening tool for oesophageal varices in all patients with portal hypertension. It is a useful tool for predicting the presence of oesophageal varices in patients with portal hypertension non-invasively when endoscopy facilities are unavailable [21] but this study was done only on 50 patients and only 37 patients had OVs, so this shortage in the number of cases limits its significance.

Also, [43] concluded that the PC/SD ratio was significantly associated with high-risk esophageal varices (HREV), but with suboptimal sensitivity and specificity. Therefore, the results of this study do not support the routine clinical use of the PC/SD ratio for screening HREV. The drawback of this study was due to it was only done on 67 patients for the detection of HREV as focusing on patients with large OVs, would miss an important subset of patients requiring medical treatment.

However, in this study the distribution of the study population was homogeneous and representative of the population of cirrhotic patients seen in clinical practice, thus biases caused by the selection of subgroups of patients were avoided. Diagnosis and classification of OVs were made in the same endoscopy center using a single classification (Paquet classification) and done by the same experienced operator. We focused on the presence of any OVs grade rather than on the presence of large OVs as this is the first step in the diagnostic/prognostic workup of the patients and allows decision-making processes (surveillance, repeat endoscopy at predetermined intervals, start therapy) while focusing on patients with large OVs would only miss an important subset of patients requiring medical counseling. Moreover, analysis of the presence/ absence of OVs prevents misinterpretation of data and allows generalization of the results.

The variabilities in the cut-off value of PC/SD ratio measurement between different studies may be due to equipment-related, intra-observer, and inter-observer variability or according to the etiology of liver disease. In this study, we tried to decrease the effect of these variabilities through the measurement of the maximum spleen diameter of all patients by a single highly-trained physician at the same time of the day before lunchtime, and by using a highly equipped instrument done by a single expert. Also, OVs detection is done by a single expert with the same highly equipped endoscopy. Also, the main etiological factor for liver cirrhosis in the present study was HCV infection.

In the present study, we found a significant correlation between the PC/SD ratio and Child-Pugh score classification, MELD, FIB-4 score, and APRI score, indicating that the PC/SD ratio is correlated to the severity of liver function decompensation in patients with cirrhosis. This also agreed with [41] who found "a significant correlation between the PC/SD ratio with the size of OVs, and the Child-Turcotte-Pugh classification".

There are some limitations in our study; the limited number of patients and a single-center study might affect results. The study participants were cirrhotic patients with different etiologies of decompensated liver cirrhosis.

5. CONCLUSION

PC/SD ratio is statistically significantly lower in cirrhotic patients with OVs. It is a good indicator in predicting the development and the degree of esophageal varices. It also correlates with the severity of liver cirrhosis assessed by FIB-4 score, and APRI score.Concerning previously proved differences in values of PC/SD ratio, further separate studies are needed to assess the relation of PC/SD ratio with the presence and grading of OVs for each etiology.

Future studies are needed to evaluate PC/SD ratio in isolated PHG and GVs in separate groups without the presence of OVs.

CONSENT AND ETHICAL APPROVAL

This study is in agreement with the ethical guidelines of the Declaration of Helsinki and it follows the ethical standards of the Tanta faculty of medicine approval code (34408/1/21) all patients were aware of the steps, and goal of the study, and they were included after obtaining written informed consent from them.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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