ORIGINAL ARTICLE ASIAN JOURNAL OF MEDICAL SCIENCES

Comparison of serum-ascites albumin gradient and platelet count/splenic diameter ratio in predicting esophageal varices in cirrhosis of liver

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Submission: 31-08-2025 Revision: 03-10-2025 Publication: 01-11-2025

ABSTRACT

Background: Prediction of esophageal varices (EV) in cirrhotic patients by non-invasive methods is of greatest importance as it may decrease medical and economic load related to screening. In this study, we aim to validate and compare the platelet count/spleen diameter ratio (PC/SD) and serum-ascites albumin gradient (SAAG) as predictors of EV in patients with cirrhosis of liver. **Aims and Objectives:** The study was conducted to determine, assess, and compare the efficacy of SAAG and PC/SD in predicting EV in patients with cirrhosis. **Materials and Methods:** The study was conducted on eighty patients with cirrhosis of liver at Kempegowda Institute of Medical Sciences, Department of General medicine, Bangalore, over a period of 2 years. All relevant clinical parameters including examination, laboratory tests, endoscopy, spleen measurement, and ascitic fluid analysis were done and documented. **Results:** The study predominantly included male patient's (90%) with mean age 48.84 ± 10.92 years. EV present in 87.5% of patient's, with 97.5% having high SAAG (>1.1). SAAG significantly correlated with variceal presence (P<0.001), while PC/SD showed no correlation (P=0.593). **Conclusion:** Elevated SAAG showed statistically significant correlation with EV in cirrhotic patient's and served as a better predictor in liver cirrhosis, while PC/SD showed no correlation. Therefore, SAAG is a better predictor for the presence of EV compared to PC/SD.

Key words: Endoscopy; Esophageal varices; Cirrhosis of liver; Platelet count-to-spleen diameter ratio; Thrombocytopenia; Serum-ascites albumin gradient

Access this article online

Website: https://ajmsjournal.info/index.php/AJMS/index

DOI: 10.71152/ajms.v16i11.4853

E-ISSN: 2091-0576

P-ISSN: 2467-9100

INTRODUCTION

Portal hypertension is the most common and major complication of chronic liver disease. It is responsible for the development of esophageal varices (EV), variceal hemorrhage, ascites, renal dysfunction, hepatic encephalopathy, hypersplenism, and hepatopulmonary syndrome. Among these complications, EV are a major cause of morbidity and mortality, with variceal hemorrhage accounting for approximately 70% of upper gastrointestinal (GI) bleeding episodes in cirrhotic patients and with mortality rate of 15–20% per bleeding episode.[1] The identification and risk stratification of patients with EV at the earliest is crucial for implementing appropriate prophylactic measures and reducing mortality associated with variceal bleeding.

Upper GI endoscopy remains the gold standard for diagnosing EV and assessing their bleeding risk. However, this invasive procedure is associated with patient discomfort, potential complications, healthcare costs, and limited availability in resource-limited settings. Current

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guidelines recommend endoscopic screening for all patients with newly diagnosed cirrhosis, followed by surveillance endoscopy every 2–3 years in patients without varices and every 1–2 years in those with small varices.[2] This approach has a substantial burden on healthcare systems and there is a necessity for the development of reliable non-invasive predictive markers that can identify high-risk patients requiring urgent endoscopic evaluation.

The hemodynamic alteration associated with portal hypertension is accompanied by characteristic laboratory and imaging findings that have been investigated as potential non-invasive predictors of varices. The serum-ascites albumin gradient (SAAG) has emerged as a valuable diagnostic tool that reflects the degree of portal hypertension by measuring the oncotic pressure difference between serum and ascitic fluid.[3] The SAAG reflects the colloid osmotic pressure gradient and the degree of portal hypertension, with values ≥1.1 g/dL indicates portal hypertension-related ascites.[4]

SAAG not only serves as a diagnostic marker for portal hypertension but also a predictor of complications of portal hypertension including EV. SAAG has been concluded in several studies as an oblique marker in predicting portal hypertension and its complications, which helps to forecast the occurrence of EV.[5] Hence, higher SAAG values reflects more severe portal hypertension, and increased likelihood of developing EV.

Simultaneously, the platelet count-to-spleen diameter ratio (PC/SD) is another non-invasive predictor of EV. This parameter is based on thrombocytopenia due to hypersplenism and splenomegaly resulting from increased portal pressure. The PC/SD ratio is found to be the most reliable marker to predict EV. It is easy to obtain and can be used with other markers to identify the high-risk patients for developing EV.[6] The ratio was first proposed as a non-invasive predictor of EV with various cutoff values reported across different populations, ranging from 500 to >2000.[7] PC/SD: A promising non-invasive tool for predicting EV in cirrhosis patients has been validated in multiple studies, with varying degrees of accuracy in a resource limited settings.[8]

PC/SD and SAAG are promising as predictors of EV and needs a proper validation. The objectives of our study were to validate and compare the PC/SD ratio and SAAG as predictors of the presence and grades of EV in patients with cirrhosis of liver.

Aims and objectives

To assess and compare the efficacy of Serum-Ascites Albumin Gradient (SAAG) and Platelet Count to Spleen Diameter (PC/SD) ratio as non-invasive markers for detecting oesophageal varices in patients with liver cirrhosis.

MATERIALS AND METHODS

The cross-sectional study conducted on 80 patients with cirrhosis of liver, attending to Department of General Medicine at Kempegowda Institute of Medical Sciences and Research Institute, Bangalore, over the period of 2 years from 2022 to 2024 was selected, based on inclusion and exclusion criteria.

All patients in the study underwent a full clinical evaluation. Clinical history and physical examination findings were recorded with particular attention to present or previous hematemesis, malena, bleeding per rectum, bleeding tendencies, alcoholism, blood transfusion, intake of hepatotoxic drugs, exposure to Sexually transmitted diseases, IV drug abuse, jaundice, anemia, edema, stigmata of chronic liver disease, dilated abdominal veins, ascites, splenomegaly, and encephalopathy.

All patients underwent biochemical tests, such as liver function tests, complete blood counts, renal function tests, prothrombin time, ultrasonography of the abdomen to confirm the presence of cirrhosis and to record the spleen bipolar diameter, portal vein size, ascites, and presence of collaterals. Child-Pugh score was calculated for all patients.

Diagnosis of cirrhosis was done based on a combination of history, clinical findings, impaired liver function tests, deranged coagulation profile, and abdominal ultrasound.

Ascitic fluid analysis was done and SAAG was calculated by subtracting ascitic fluid albumin from serum albumin.

The PC/SD was calculated for all patients in the study.

Upper GI endoscopy was done in all patients to confirm the presence of varices and also to grade them. All endoscopies were performed in a single endoscopy unit using a video endoscope.

SAAG and PC/SD was correlated with the presence of EV.

Statistical methods

Descriptive and inferential statistical analysis has been carried out in the present study. Results on continuous measurements are presented on Mean±SD (Min-Max) and results on categorical measurements are presented in Number (%). Significance is assessed at 5% level of significance.

Statistical software

Table 1: Age distribution of subjects studied

Age in years	No. of subjects	%
21–30	3	3.8
31–40	17	21.3
41–50	25	31.3
51–60	23	28.8
61–70	11	13.8
>70	1	1.3
Total	80	100.0

Table 2: Gender distribution of subjects studied

Gender	No. of subjects	%
Female	8	10.0
Male	72	90.0
Total	80	100.0

Table 3: Distribution of subjects based on presence or absence of esophageal varices

Esophageal varices	No of subjects	%
Negative	10	12.5
Positive	70	87.5
Total	80	100.0

Table 4: Distribution of subjects based on platelet count/spleen diameter ratio (PC/SD)

PC/SD ratio	No. of subjects	%
≤500	19	23.8
500–1000	37	46.3
1000–2000	18	22.5
>2000	6	7.5
Total	80	100.0

Table 5: Distribution of subjects based on SAAG

SAAG	No. of subjects	%
≤1.1	2	2.5
1.1–1.5	10	12.5
1.5–2	21	26.3
2–2.5	29	36.3
>2.5	18	22.5
Total	80	100.0

SAAG: Serum-ascites albumin gradient

Table 6: Correlation of PC/SD ratio with presence of esophageal varices

PC/SD	Varices (%)		Total (%)
	Present	Absent	
≤500	3 (30)	16 (22.9)	19 (23.8)
500–1000	3 (30)	34 (48.6)	37 (46.3)
1000–2000	3 (30)	15 (21.4)	18 (22.5)
>2000	1 (10)	5 (7.1)	6 (7.5)
Total	10 (100)	70 (100)	80 (100)

P=0.593, Not significant, PC/SD: Platelet count/spleen diameter ratio

Table 7: Correlation of SAAG with presence of esophageal varices

SAAG	Varices (%)		Total (%)
	Present	Absent	
≤1.1	2 (20)	0 (0)	2 (2.5)
1.1–1.5	6 (60)	4 (5.7)	10 (12.5)
1.5–2	2 (20)	19 (27.1)	21 (26.3)
2–2.5	0 (0)	29 (41.4)	29 (36.3)
>2.5	0 (0)	18 (25.7)	18 (22.5)
Total	10 (100)	70 (100)	80 (100)

P<0.001**, Significant, SAAG: Serum-ascites albumin gradient

The statistical software, namely, SAS 9.2, SPSS 15.0, Stata 10.1, MedCalc 9.0.1, Systat 12.0, and R environment ver.2.11.1 were used for the analysis of the data.

Chi-square/Fisher Exact test has been used to find the significance of study parameters on categorical scale between two or more groups.

Analysis of variance has been used to find the significance of study parameters between three or more groups of patients. Student t-test (two-tailed, independent) has been used to find the significance of study parameters on continuous scale between two groups (Intergroup analysis) on metric parameters.

Inclusion criteria

- Patients above 18 years of age with cirrhosis of liver, who gives consent to get involved in the study will be included.

Exclusion criteria

- Patients with present or previous history of variceal bleed
- Patients on previous/current treatment with beta-blockers, diuretics, or anti-platelet drugs
- Patients who have undergone sclerosis or band ligation of EV, TIPSS, or surgery for portal hypertension
- Patients having fever associated with thrombocytopenia in the past and history of fever in the past 15 days will be excluded
- Patients who are on drugs which are associated with thrombocytopenia will be excluded
- Patients with spontaneous bacterial peritonitis will be excluded.

RESULTS

The study revealed age distribution of 25 subjects (31.3%) between the age group of 41 and 50 years, 23 (28.8%)

Table 8: Comparison of PC/SD and SAAG in correlation with presence of esophageal varices

Variables	Varices		Total	P-value
	Present	Absent		
PC/SD ratio	1112.49±869.49	899.66±735.70	926.26±750.92	0.405
SAAG	1.29±0.22	2.21±0.39	2.09±0.48	<0.001**

SAAG: Serum-ascites albumin gradient, PC/SD: Platelet count/spleen diameter ratio

Table 9: ROC curve analysis of PC/SD ratio and SAAG to predict esophageal varices

Variables	ROC results to predict esophageal varices						Cut-off	AUROC	SE	P-value
	Sensitivity	Specificity	LR+	LR-	PPV	NPV				
PC/SD ratio	52.86	70.00	1.76	0.67	92.50	17.50	≤771.46	0.559	0.108	0.527
PC/SD ratio	62.8	40.0	1.05	0.93	88.0	13.3	≤909	-	-	0.861
SAAG	90.00	100.00	0.00	0.10	100.00	58.82	>1.6	0.974	0.016	<0.001**

SAAG: Serum-ascites albumin gradient, PC/SD: Platelet count/spleen diameter ratio, ROC: Receiver operating characteristic, AUROC: Area Under the Receiver Operating Characteristic Curve, LR+: Positive likelihood ratio, LR-: Negative likelihood ratio, PPV: Positive predictive value, NPV: Negative predictive value, SE: Standard error

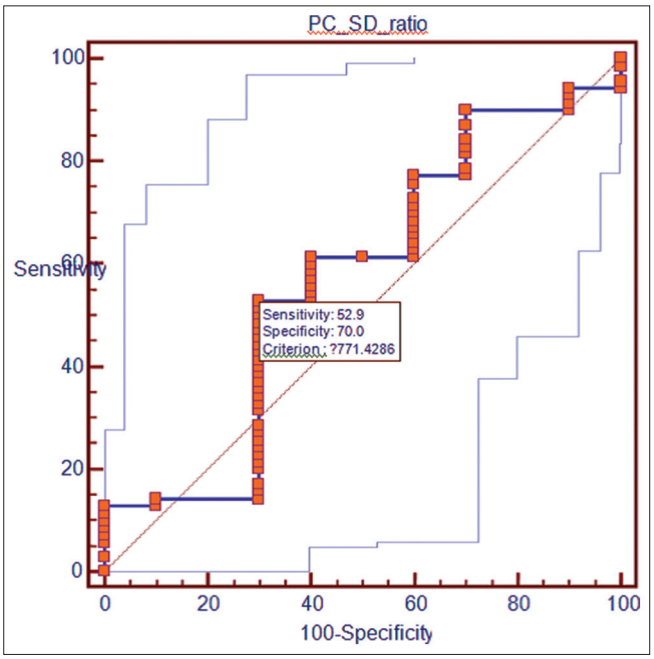

Figure 1: Receiver operating characteristic curve analysis of platelet count-spleen diameter ratio in predicting varices

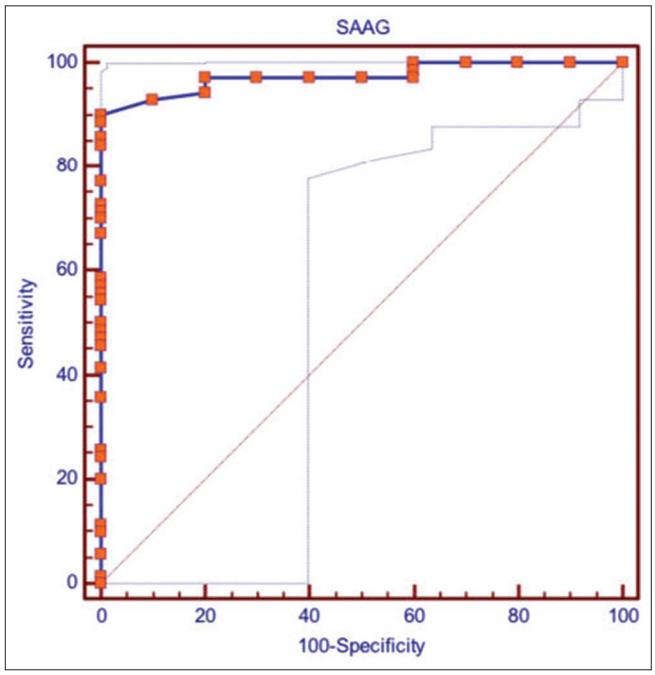

Figure 2: Receiver operating characteristic curve analysis of serum-ascites albumin gradient in predicting varices

between 51 and 60 years, 17 (21.3%) between 31 and 40 years, 11 (13.8%) between 61 and 70 years, 3 (3.8%) between 21 and 30 years, and 1 (1.3%) was above 70 years. Mean age was 48.84±10.92 years (Table 1 and Figure 1).

Among the subjects studied, males predominate the study population with 72 subjects (90%) and females 8 (10%) (Table 2).

Among 80 subjects studied, 70 (87.5%) were found to have EV and 10 (12.5%) did not have EV (Table 3).

In this study, 37 subjects (46.3%) had PC/SD ratio between 500 and 1000, 19 (23.8%) had PC/SD ratio <500, 18 (22.5%) had PC/SD ratio between 1000 and 2000, and 6 (7.5%) had PC/SD ratio more than 2000. (Table 4 and Figure 2).

Among the study population, 29 subjects (36.3%) had SAAG between 2 and 2.5, 21 (26.3%) had SAAG between 1.5 and 2, 18 (22.5%) had SAAG more than 2.5, 10 (12.5%) had SAAG between 1.1 and 1.5, and 2 (2.5%) had SAAG <1.1 (Table 5).

This study showed that out of 70 subjects with presence of EV, 16 had PC/SD ratio <500, 34 had PC/SD ratio between 500 and 1000, 15 had PC/SD ratio 1000–2000, and 5 had PC/SD ratio more than 2000. PC/SD ratio was not associated with presence of varices. Correlation of PC/

SD with presence of varices was not statistically significant (P=0.593) (Table 6).

In this study, out of 70 subjects with EV, 0 had SAAG <1.1, 4 had SAAG between 1.1 and 1.5, 19 had SAAG between 1.5 and 2, 29 had SAAG between 2 and 2.5, and 18 had SAAG more than 2.5. All patients with EV had high SAAG (>1.1). Correlation of SAAG with presence of EV was strongly significant (P≤0.001) (Table 7).

In this study, the mean PC/SD in subjects with absence of varices was 1112.49±869.49 and in subjects with presence of varices was 899.66±735.70. The mean SAAG in subjects with absence of varices was 1.29±0.22 and in subjects with presence of varices was 2.21±0.39. SAAG is strongly associated with presence of varices (P≤0.001), whereas PC/SD is not associated with presence of varices (P=0.405) (Table 8).

In predicting EV, PC/SD ratio cutoff of 771.46% had a sensitivity of 52.86%, specificity of 70%, positive predictive value of 92.50%, and negative predictive value of 17.50%, and with 909 cutoff, it had sensitivity of 62.8% and specificity of 40%. PC/SD was not associated with varices (P=0.527) (Table 9 and Figure 3).

SAAG ratio with a cutoff of 1.6 had sensitivity of 90% and specificity of 100%, positive predictive value of 100%, and negative predictive value of 58.82%. SAAG was strongly significant in predicting varices (P≤0.001) (Figure 4).

DISCUSSION

This study was conducted on 80 patients with cirrhosis of liver, presenting to Department of General Medicine, KIMS Hospital, Bangalore, over a period of 2 years. This study had a maximum distribution in the age group 41–50 years, accounting for 31.3%, male being 90%, and mean age was 48.84±10.92 which was comparable to a study by Liang et al.,[9] who had a mean age of 52±11 years and 80.9% were male.

In our study, 87.5% patients were found to have varices which are comparable to a study conducted by Liang et al.,[9] who had 87.1%% of subjects with varices.

In our study, majority of subjects belonged to the category of platelet count/spleen bipolar diameter ratio between 501 and 1000 accounting to 46.3%, followed by <500 constituting about 23.8% comparable with a study by Tiwari et al,[10] which had 59% cases with PC/SD ratio <1000.

In our study, PC/SD ratio was not associated with presence of varices. Correlation of PC/SD with presence of varices was not statistically significant (P=0.593). Study done by Liu et al., concluded that although the PC/SD is a predictor of the existence of EV, the relationship between it and the risk of variceal bleed is unknown.[11]

Considering the PC/SD ratio cutoff 909 as suggested by Giannini et al.,[12] (which yielded 100% sensitivity and 93% specificity), this study yielded a sensitivity of 62.8% and specificity of 40%. In this study, PC/SD ratio was not statistically associated with presence of varices (P=0.593) and PC/SD ratio was not statistically associated with grades of varices (P=0.157). As compared to this study, in a study by Mattos et al.,[13] PC/SD ratio cutoff 909 had 77% sensitivity and 45.5% specificity. In our study, PC/SD ratio cutoff 771.46 yielded a sensitivity of 53.86 and specificity of 70%, positive predictive value of 92.50% and negative predictive value of 17.50%.

In our study, out of 80 patients, 2.5% had low SAAG (SAAG <1.1) and 97.5% had high SAAG (SAAG >1.1). In our study, SAAG was strongly associated with the presence of varices. All patients with EV in this study had high SAAG (SAAG >1.1) and patients with low SAAG (SAAG <1.1) did not have varices, these results are comparable to a study by Kumar et al.,[14] which showed patients found to have SAAG value <1.10 and EV were absent in all of them.

In this study, SAAG with cutoff 1.6 had sensitivity of 90% and specificity of 100%. In a study by Sukhiranwat et al.,[15] showed that the cutoff point of SAAG >1.65, the sensitivity and specificity for diagnosis of EV were 96.4% and 71.4% and at the cutoff point of SAAG >2.05, the sensitivity and the specificity for diagnosing large EV were 87.5% and 66.7%, respectively.

In our study comparing SAAG and PC/SD ratio in predicting the presence of EV, PC/SD ratio was not statistically significant with P=0.593, whereas SAAG was found to be strongly associated with presence of varices with P≤0.001. Comparing SAAG and PC/SD ratio in correlation with grades of EV, PC/SD ratio was not statistically significant with P=0.157, whereas SAAG was found to be strongly associated with grades of varices with P≤0.001.

In our study, ROC curve analysis showed PC/SD ratio with cutoff 771.46 had sensitivity of 52.86% and specificity of 70%, SAAG with cutoff 1.6 had sensitivity of 90% and specificity of 100% in predicting varices.

CONCLUSION

PC/SD was not associated to the presence and grades of EV in subjects with cirrhosis of liver. SAAG was strongly

associated to the presence of EV and also to the grades of EV. Raised SAAG reflects great probability of presence of EV in the cirrhosis of liver patients. Furthermore, the presence of EV is observed to be directly concerned to the degree of SAAG.

Therefore, SAAG is a better predictor of presence and severity of EV compared to platelet spleen ratio.

ACKNOWLEDGMENTS

None.

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Authors' Contributions:
STA- Definition of intellectual content, literature survey, prepared first draft of manuscript, implementation of study protocol, data collection, data analysis, manuscript preparation and submission of article; **RH-** Concept, design, clinical protocol, manuscript preparation, editing, and manuscript revision; **AN-** Design of study, statistical analysis and interpretation.

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Source of Funding: None, **Conflicts of Interest:** None.