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Original Research Article

Correlation of serum-ascites albumin concentration gradient and endoscopic parameters of portal hypertension in chronic liver disease

Suresh I[1], Shiva Prasad Jagini[2]*

[1]Assistant Professor, Department of General Medicine, Kamineni Institute of Medical Sciences, Narketpally, Nalgonda Dist., Telangana, India
[2]Assistant Professor, Department of General Medicine, Kamineni Academy of Medical Sciences and Research Centre, LB Nagar, Hyderabad, Telangana, India

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***Correspondence:**
Dr. Shiva Prasad Jagini,
E-mail: drjaginishiva@gmail.com

ABSTRACT

Background: In many studies Serum ascitic albumin gradient (SAAG) was found to be an independent predictor of PHTN and EV especially in alcoholic cirrhosis. Objectives of this study was to study correlation of level of "Serum-Ascites Albumin Concentration Gradient" (SAAG) and complications of "Portal hypertension" (PHTN), manifested by "Esophageal Varices" (EV).
Methods: Present study was hospital based cross sectional study. The sample (100) was of patients with ascites. SAAG was measured in all subjects. EV was assessed by endoscopy in all. Data was analyzed using proportions and appropriate statistical tests.
Results: High SAAG value was seen in 79% of the patients. EV incidence was 84.5%. "child-pugh score" and size of the portal vein was found to be associated with EV. The incidence of EV among patients with high "SAAG value of 1.1 to 1.44 g/dl" was 50%. The size of the EV was found to be significantly associated with SAAG level.
Conclusions: Patients having ascites with EV were also having high levels of SAAG. Thus, we conclude that value of SAAG more than or equal to 1.2±0.05 g/dl can be used as a predictor of EV presence among ascites patients.

Keywords: Ascites, Cirrhosis of liver, SAAG

INTRODUCTION

Among the internal organs of the body, liver is the largest. It receives blood supply not only from portal vein but also from hepatic artery. Liver has many functions. These essential Functions become impaired when liver develops cirrhosis. "Chronic liver disease (CLD) including cirrhosis is currently twelfth leading cause of death in US", "ninth leading cause of death in India".[1,2] Cirrhosis of liver is mainly due to hepatitis C infection and alcoholism of long standing duration.[3] Chronicity of liver disease is determined either by duration of liver disease or by evidence of either severe liver disease or physical stigmata of chronic liver disease. Cirrhosis of liver is usually associated with portal hypertension. The most common complication of portal hypertension is esophageal varices with incidence of 60-80%.[4,5]

The death rate due to EV bleeding is 17-57%. These patients are at risk of bleeding or rupture which leads to death.[6,7]

"The American Association for the Study of Liver Disease single topic symposium 1996" guideline says that all patients with cirrhosis of liver and portal hypertesion should be screened for EV. "Baveno III

Consensus Conference on PHTN" recommended the same. Some authors suggest to carry out endoscopy at regular intervals.[8,9]

Endoscopic screening may take place under two circumstances: at the initial diagnosis of cirrhosis, since EV are an independent predictive factor and an early complication of cirrhosis, and during the follow-up of patients with cirrhosis without EV at risk of bleeding at first examination with or without decompensation.[8]

It has been estimated that it is only the large esophageal varices (LEV), which are associated with a substantially increased risk of variceal bleed. The reported incidence of LEV ranges from 9% to 49%. In a recent review, Boyer, using a prevalence of LEV of 20%, estimated that a 100 screening endoscopic examination need to be performed to prevent 1 to 2 cases of variceal bleeding.[4]

The SAAG has been defined as the serum albumin concentration minus the ascitic fluid albumin concentration. The SAAG is superior to the exudate transudate concept to classify ascites, being an exact PHTN marker.[6]

There are scant studies in the literature to evaluate SAAG and EV in the patients with non-alcoholic cirrhosis. When the value of SAAG was more than 2, strong correlation existed even in Non-alcoholic cirrhosis. Hence present study was carried out to identify "non-invasive parameters for prediction of esophageal varices" (EV) in newly diagnosed patients with cirrhosis.

METHODS

Type of Study: Cross sectional study. Sample size: 100 patients. Place of study: Department of General Medicine, Basaveshwar Teaching and General Hospital, Gulbarga attached to Mahadevappa Rampure Medical College. Duration of study: January 2011 to May 2012.

Inclusion Criteria

All newly diagnosed cases of chronic liver disease with Ascites and confirmed by physical examination, biochemical parameters, and Ultra sound Abdomen.

Exclusion Criteria

Patients with pregnancy, bleeding from existing portal hypertension, having AIDS, currently on beta blockers were excluded from the present study.

All patients in this study underwent a full clinical evaluation i.e., clinical history and physical examination and findings were recorded with particular attention to present or previous history of yellowish discoloration of sclera, abdominal swelling, swelling of both lower limbs, hematemesis, melena, 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.

Necessary investigations and endoscopy was carried out among the patients. Ultrasonographic features of cirrhosis include coarse echogenic echo texture, nodular surface and regenerating nodules surrounded by fibrous septae. The nodules are seen as iso to hypo-echoic lesions with echogenic borders corresponding to fibrous septa. The caudate lobe to right lobe of liver ratio of > 0.65 is highly specific to cirrhosis. Ultrasonographic features of portal hypertension is based on demonstration of underlying liver disease, splenomegaly, ascites, dilated portal vein (> 13 mm diameter) and presence of collaterals. Portal vein velocity is reduced in portal hypertension. Blood sample was collected as per the guidelines.[10]

Serum albumin as well as albumin in the ascitic fluid was determined with standard technique.[11] Endoscopy was performed with standard guidelines.[12]

TABLE 1: EV was graded as follows.[13]

Grading	Esophageal varices
I	Small and straight
II	Tortuous and occupying<1/3rd of the lumen
III	Large and occupying <1/3rd of the lumen

Statistical analysis

Data were collected in a predetermined proforma and results were analyzed using Software Statistical package student science (SPSS) version 14.0.

Continuous variables were analyzed using student unpaired t-test and categorical variables by Chi square test ($\chi 2$). Pearson Correlation was used to find correlation between two variables. "Receiver operating characteristic curve" was used to find the SAAG value with best sensitivity and specificity for predicting EV. "A p value of < 0.05 was considered statistically significant", p value of < 0.01 was considered highly significant and p value of < 0.001 was considered very highly significant.

RESULTS

Table 2: Distribution of cases based on etiology.

Etiology	Number of Cases
Alcoholic	75 (75%)
HBV	10 (10%)
HCV	14 (14%)
Wilson disease	1 (1%)
Total	100

In this study Maximum number of patients with cirrhosis were due to alcohol (75%), followed by HCV (14 %), HBV (10%), and 1 case of Wilsons disease.

Table 3: Distribution of cases based on grade of esophageal varices.

Grade of varices	Frequency
0	33 (33%)
I	18 (18%)
II	19 (19%)
III	30 (30%)
Total	100

Among 100 patients in this study 67 patients had esophageal varices. 30 % patients had grade III esophageal varices, 19% had grade II and 18 % had grade I esophageal varices respectively.

Table 4: Relation between hepatic encephalopathy and varices.

Grade of varices	Hepatic encephalopathy		Total
	Yes	No	
0	0	33	33
I	1	17	18
II	4	15	19
III	5	25	30
Total	10	90	100

$\chi 2$= 6.70; P=0.15 NS

No significant relation between presence of hepatic encephalopathy and grade of varices was found.

Table 5: Association between EV and SAAG values.

Esophageal varices	High SAAG (>1.1)	Low SAAG (<1.1)	Total
Presence	67	3	70
Absence	12	18	30
Total	79	21	100

$\chi 2$= 39.29; P < 0.001 S

84.5 % of the study population had varices when SAAG value was more than 1.1 g/dl (P < 0.001) and there is very highly significant relation found between high SAAG and presence of esophageal varices.

Table 6: Relationship between child Pugh score and varices.

Grade of varices	CP grade			Total
	A	B	C	
0	3	21	9	33
I	5	12	1	18
II	4	8	7	19
III	5	16	9	30
Total	17	57	26	100

$\chi 2$= 31.5 P=0.01 S

Patients were grouped according to Child Pugh Classification of Cirrhosis. The relationship between Child Pugh Grade and the grade of varices was studied and significant correlation noted (P = 0.01).

Table 7: Relationship between portal vein size and presence of Varices.

Parameter	Varices	Number of patient
Portal vein size (> 13 mm)	Present	67
	Absent	33

t=2.03, P<0.05 S

Significance was noted between portal vein size (mm) and presence of varices in the study group.

Table 8: Distribution based on SAAG values.

SAAG	Frequency
< 1 .1	21
1.1 – 1.44	30
1.45-1.99	20
> 2	29
Total	100

In this study 21% of study population had SAAG < 1.1, and 79 % had SAAG > 1.1, among high SAAG 30 (37.6%) had SAAG value between 1.1 - 1.44, 20 (25.3%) had SAAG value between 1.45 – 1.99 and 29 (36.3) had SAAG value >2.

Table 9: Relationship between SAAG and grade of varices.

SAAG	Grade of varices			Total
	I	II	III	
<1.1	3	0	0	3
1.1 – 1.44	4	4	7	15
1.45 – 1.99	5	5	10	20
>2	6	10	13	29
Total	18	19	30	67

Correlation Co-efficient (r) = 0.607, p < 0.01 S

When the Value of SAAG was < 1.1 g/dl it was noted that Grade II and III varices were absent. When the SAAG values increased more than 1.1 g/dl, there was considerable increase in grading of varices. There was highly significant association found between SAAG and grade of varices.

Table 10: Role of SAAG in predicting varices in cirrhotic patients with ascites.

	Esophageal varices		Total
	Present	Absent	
SAAG <1.1	3	18	21
SAAG >1.1	67	12	79
Total	70	30	100

It has been observed from above table that when SAAG value was more than 1.1 then the possibility of having

esophageal varices was more than with value of less than 1.1.

Table 11: Diagnostic accuracy of SAAG in predicting presence of varices.

	Value in %	95 % CI
Sensitivity	81	62 – 94
Specificity	100	63 – 100
False positive	0	0 – 33
False negative	19	6 – 38
Positive predictive value	100	85 – 100
Negative predictive value	64	35 – 87

The sensitivity of SAAG >1.1 in predicting the presence of varices is 81% and its specificity and positive predictive value is 100%.

DISCUSSION

Our study sample consists of hundred patients of whom eighty were male and twenty were females. The mean age was 44.5 (SD = 11.8).

In present study Maximum number of patients with cirrhosis were due to alcohol (75 %), followed by HCV (14%), HBV (10%), and 1 case of Wilsons disease and these patients presented with symptoms of abdominal distension in all patients, 73% presented with yellowish discoloration of sclera, 54% presented with swelling of lower limbs, 29% presented with hematemesis, 28 % had malena and 10% presented with altered sensorium

All patients in the study group had ascites; other frequent signs elicited were visible abdominal veins (78%), Decreased axillary and pubic hairs (70%), splenomegaly (67%), spider nevi (35%), gynecomastia (35%), palmar erythema (10%), and flapping tremors (10%). Keiser Fleischer's ring was observed only in 1 patient.

We studied the frequency of distribution based on grading of varices and found that Grade I in 18%, grade II in 19%, grade III in 30% and 33% of study sample did not have EV.

10 patients in the study population presented with hepatic encephalopathy and present study did not find any association with varices.

Patients were grouped according to Child Pugh Classification of Cirrhosis. The relationship between Child Pugh Grade and the grade of varices was studied and significant correlation noted (P=0.001) Thus as patients progress to decompensate liver disease (CP Grade B & C) it is noted that the presence of varices increases. This is finding is consistent with study by Madhotra et al and Zaman et al.[14,15] Significance was noted between portal vein size (cm) and presence of varices. Similar results were obtained in study by D'Amico et al.[16]

We grouped patients based on range of SAAG values. Of the study sample SAAG was less than 1.1 in 21% of the patients and more than 1.1 in 79 %. 84.5 % of the study population had varices when SAAG value was more than 1.1. 14.28% of patients in whom varices were present had SAAG values less than 1.1. The two groups showed statistically significant difference (P=0.001) based on presence and absence of varices. This is consistent with studies by Torres et al, Gurubacharya et al, Kajani et al, Dittrich et al.[17-20]

We also assessed the correlation between grade of varices and SAAG values. When the Value of SAAG was < 1.1 it was noted that Grade II and III varices were absent. When the SAAG values increased more than 1.1, there was considerable increase in grading of varices.

In our study there was moderate correlation (r= 0.607) between SAAG and grade of varices. This finding is similar to the study by Torres et al, Gurubacharya et al and Demyrel et al.[17,18,21]

We found that when the cut off value of SAAG was taken as 1.20±0.05 gm/dl it was noted that the test achieved 100 % specificity and sensitivity ranging from 77.8 to 81.5%. But Torres et al stated that the SAAG value must be considered $\geq$ 1.435±0.015 gm/dl for predicting EV.

The sensitivity of SAAG > 1.1 g/dl in predicting the presence of varices in the subgroup of patients with ascites was 81 % (95% CI; 62-94) and its positive predictive value is 100 % (95% CI = 85-100)

Thus, use of SAAG may help to identify patients with a probability of esophageal varices that may not need endoscopy. This may help reduce costs and discomfort for these patients and the burden on endoscopy units.

CONCLUSION

In conclusion, we state that high SAAG values are seen among patients of EV with liver cirrhosis. SAAG degree is directly proportional to the size and presence of EV among these patients. We give value of SAAG as > 1.2± 0.05 g/dl which will help in predicting whether the portal hypertension is present or not.

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