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An Evaluation of the Significance of High SAAG Value (1.1 gm/dl) as a Predictor of Portal Hypertension and low SAAG Value as a Predictor of Absence of Portal Hypertension

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Abstract | **Original Research Article**

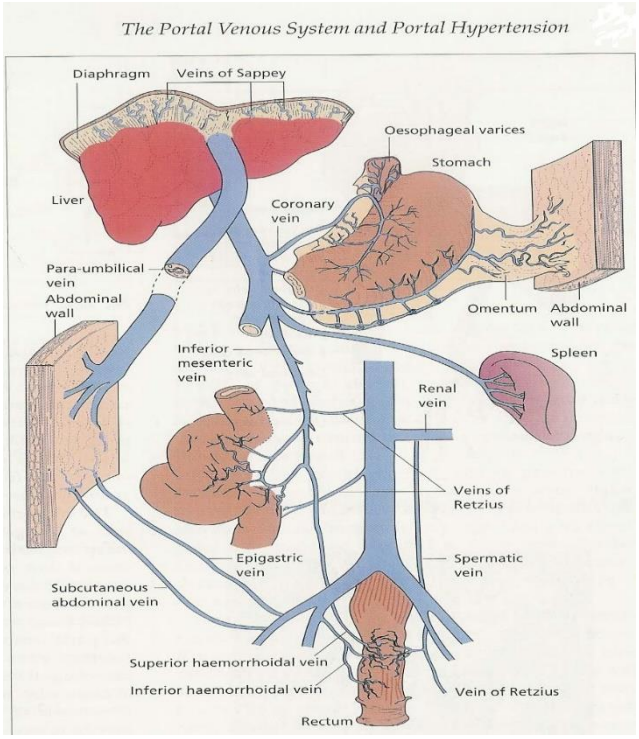
This cross sectional analytic study was conducted in the department of Pediatrics, Bangabandu Sheikh Mujib Medical University, Dhaka Medical College Hospital and Dhaka Shishu Hospital of Bangladesh for a period from January 2017 to June 2018. The main objective of the study was to evaluate the significance of high SAAG value as a predictor of portal hypertension, manifested by oesophageal varices and low SAAG value as a predictor of absence of portal hypertension. A total of 55 patients were studied. Among them, 31 were chronic liver disease patients with ascites and rest 24 patients were nephrotic syndrome patients with ascites. The mean age of the patients was 8.3±3.6 years. In the study 81% of the patients with chronic liver disease were found to have high SAAG (≥1.1gm/dl) and 100% of the nephrotic syndrome patients were found to have low SAAG (<1.1gm/dl). Mean SAAG value of patients with chronic liver disease was significantly higher than that of nephrotic syndrome patients. This finding indicates that portal hypertension is the cause of this difference among the two transudative causes of ascites. By upper gastrointestinal endoscopy, oesophageal varices were found in 81% of the high SAAG patients with chronic liver disease whereas oesophageal varices was found only in 17% of low SAAG patients with chronic liver disease. This finding indicates that oesophageal varices are associated with high SAAG value. The mean SAAG value of chronic liver disease patients with oesophageal varices were significantly higher than that of patients with nephrotic syndrome. But the mean SAAG value of both chronic liver disease patients and nephrotic syndrome patients without oesophageal varices were low and no significant difference of SAAG value was found among them. The SAAG value of ≥1.1 gm/dl will differentiate chronic liver disease with oesophageal varices from those without oesophageal varices. In this study predicting oesophageal variceal sensitivity (76%) was found reasonable, specificity (83%) good and high positive predictive value (95%) was found.

Key word: Hypertension, Transudative, Oesophageal, Serum Ascitic Albumin Gradient (SAAG).

INTRODUCTION

Ascites was classified as transudativeand exudative based on the total protein concentration of the aseitic fluid. Ascitic third in chronic liver disease is transudalive. The traditional concept of low protein aseities (<2.5 gm/dl) as transudative was questioned because: the normal peritoneal fluid protein concentration is sometimes 4 gm/dl [1]. The aseitic fluid protein concentration increases in cornice liver disease patients with darns [2] and albumin infusion [3]. Some transudative saluted like cardiac ascites have high protein concentration while some traditionally exudative ascites has low concentration of protein [4]. More over cirrhosis may be the most frequent cause of high protein ascites [5]. To overcome the short comings, ascites is now being clarified as “high gradient” and “low gradient [6]” When the difference between were serum albumin and ascetic fluid albumin is ≥ 1.1 gm/dl it is called high gradient ascites whereas if the difference is <1.1gm/dl it is termed as low gradient ascites [7-9] found that the SAAG is ≥ 1.1 in 58% cases of chronic liver disease patients with presumed portal hypertension and < 1.1 in 85% cases of nephrotic syndrome related ascites. According to the starling hypothesis, the fluid movement across a capillary membrane is controlled by the balance of hydrostatic and colloid osmotic force across the capillary wall. Forces tend to achieve a dynamic

equilibrium such that increased portal pressure is counter



Balanced by increased oncotic pressure gradient, the effective gradient between serum and interstitial or aseitic fluid absolute oncotic pressure. Studies in patients with cirrhosis and portal hypertension have confirmed the reality of this physiological event [10]. When portal pressure is not increased, ascites formation occurs in the presence of an oncotic gradient [11]. Since albumin is the main determinant of oncotic pressure [12], measured the serum-ascitis gradient of albumin concentration as a reflection of the oncotic pressure gradient for documenting presence or absence of portal hypertension in the genesis of ascites from various causes. This gradient should not depend on the protein content of the ascitic fluid, whether its source is hepatic or splanchnic. In either of these two sources it probably reflects the dynamic equilibrium between the opening forces acting at the site of splanchnic vascular bed. A serum ascites albumin gradient >1.1gm/dl suggests presence of portal hypertension not only in patients with a transudate is type of ascites but also in cases with a high protein concentration. Ascites high protein level in patients with liver disease is probably explained by a high total serum protein concentration or a rather low degree of portal hypertension or both [13] on the other hand a serum ascitis fluid albumin gradient <1.1gm/dl would suggest absence of significant portal hypertension in patients with an exudative type of ascites. Ascites associated with nephrotic syndrome show a narrow albumin gradient probably due to the absence of increased portal pressure in their genesis, while the cardiac failure case demonstrated an increased albumin gradient. This further reinforces the significance of serum-ascites albumin concentration gradient as a parameter reflecting the portal pressure. Wide serum-ascites albumin gradient in patients with liver disease is compatible with increased diffusion of fluid and protein as the predominant process of ascites formation in portal hypertension [14]while a narrow gradient in patients with ascites suggests abnormal capillary permeability. A large hydrostatic pressure gradient between the portal capillaries and the peritoneal cavity characterizes all transudative causes of ascites except nephrotic syndrome, generating a large blood-to-ascites oncotic pressure difference and consequently, a low ascites total protein concentration. The converse is theoretically true for all exudative causes of ascites. A relative high ascites protein concentration may be seen in patients with transudative ascites when blood oncotic pressure, determined chiefly by the albumin concentration, is relatively well preserved, as occurs in some patients with cirrhosis and most patients with congestive heart failure or constrictive pericarditis[4]. Conversely, a relatively low ascites protein concentration may be found in patients with exudative ascites if there is severe reduction of the serum albumin concentration. These relationships limit the diagnostic usefulness of the ascites total protein concentration. The serum-ascites albumin difference, an index of the oncotic pressure difference, is on the other hand, independent of the serum albumin concentration. The major findings of John cart hoofs was that (1) the serum to ascites albumin concentration gradient is proportional to the pressure gradient from the portal vein to the intra-abdominal cavity (or inferior vena cava) and (2) the variation in ascitic fluid protein concentration between

patients can be related to a large extend, to factors determine the serum oncotic pressure and hydrostatic pressures in blood and ascites [15] (i.e. serum protein concentrations and the portal pressure or albumin concentration gradient). The protein concentration in ascites fluid associated with chronic liver disease is typically less than 2.5mg/dl (transudate), although more recent studies have reported 20% of such patients having a higher concentration of ascitic fluid protein. The protein concentration is not fixed at a low level, since it increases subsequently during albumin infusion [11]. During albumin infusion and discuss. Albumin concentration gradient and oncotic pressure difference on simultaneously obtained samples of ascites and serum show these to be virtually constant for a given patient [15]. These gradients change minimally during ingestion, albumin infusion and diuresis [3, 11]. In an individual patient, the ascitic fluid oncotic pressure or protein concentration is the resultant of the relatively constant effective portal pressure and serum oncotic pressure [10, 11]. The inference that the albumin or oncotic pressure gradient is determined by portal pressure [2]. It is supported by a wide albumin concentration gradient in patients with chronic liver disease. Quantitatively at least 80% of an increase in the capillary to tissue hydrostatic gradient is transmitted to the serum to tissue oncotic pressure gradient, since decrease in tissue pressure gradient, since decrease in tissue oncotic pressure and increase in the tissue hydrostatic pressure are the major buffering mechanisms. The close relationship of the portal pressure gradient to the serum to ascitic fluid albumin concentration gradient indicates that the major factors of buffering increase the hydrostatic gradient induced by portal pressure elevation are dilution of ascites with a decreased ascitic fluid oncotic pressure and increase in the intra-abdominal pressure. Due to high compliance of the peritoneal cavity, a large increase in peritoneal fluid volume is required for a relatively small increase in intra-abdominal pressure. [11]. Thus sharp increase in capillary hydrostatic pressure are buffered primarily by dilution of ascites. Fluid formation may buffer as much as 20% of an increase in the hydrostatic gradient from blood to ascites and therefore may influence dilution of protein in the peritoneal cavity.

OBJECTIVES

General objective

- To evaluated the significance of high SAAG Value (1.1 gm/dl) as a predictor of portal hypertension and low SAAG Value as a predictor of absence of portal hypertension.

Specific Objectives

- To determine the serum ascitic fluid albumin concentration gradient in children with chronic liver disease and nephrotic syndrome.

METHODOLOGY AND MATERIALS

This cross sectional analytic study was conducted in the Department of Pediatrics, Bangabandu Sheikh Mujib Medical University, Dhaka Medical College Hospital and Dhaka Shishu Hospital for a period from January 2017 to June 2018. A total of 55 patients were studied. Among them 31 were chronic liver disease patients with ascites and rest 24 patients were nephrotic syndrome patients with ascites. The mean age of the children was 8.3±3.6 years. In the study 81% of the patients with chronic liver disease were found to have high SAAG (≥1.1gm/dl) and 100% of the nephrotic syndrome patients were found to have low SAAG (<1.1gm/dl). Mean SAAG value of patients with chronic liver disease was significantly higher than that of nephrotic syndrome patients.

Inclusion Criteria

- Age: 1-15 years.
- Sex: Both sex.
- Sample: Accordingly to group criteria.

Exclusion Criteria

- Age <1 year > 15 years.
- Hemodynamicinstability: Severe bleeding manifestation hepatic come.
- Medical contra indication to perform endoscopy.
- Acute infection: Spontaneous bacterial peritonitis.

RESULTS

A total number of 55 children were studied. Among them, 31 children with chronic liver disease with ascites were designated as Group I and rest 24 nephrotic syndrome children with ascites were designated as group II. The mean age of the children was 8.3±3.6 years ranging from 1.0-15.0 years. The mean age of the male patient was 8.0±3.6 years and that of female patients was 9.3±3.5 years. No statistically significant mean age difference was found between male and female patients ($p>0.05$), although female patients had a bit higher mean age than male patients (Table I). The mean age of the group I patients was 9.2±3.7 years ranging from 1.0-14.0 years and group II patients was 7.2±3.2 ranging from 2.0 to 15.0 years and the mean age difference between two groups patients was statistically significant ($p<0.05$) indicated that patients with chronic liver disease had higher age than patients with nephrotic syndrome (Table II). Figure I show the sex distribution of the study subjects. Among the studied patients, 43(78.2%) were male and 12(21.8%) were female. It was observed that among the group I patients, 22(71.0%) were male and 9(29.0%) were female, whereas among the group II patients, 21(87.5%) were male and 3(12.5%) were female. No sex difference was found between two groups of patients (p>0.05), but male dominance was found in both groups. Figure II shows the correlation of serum albumin with albumin in ascitic fluid and serum

albumin with SAAG. Analysis revealed significant positive correlation between serum albumin with albumin in ascitic fluid (*p<0.001*). Similarly, positive correlation was also found between serum albumin with SAAG (*p<0.001*). It was evident that among the low SAAG, 6(20.0%) were group I patients i.e. chronic liver disease and 24(80.0%) group II patients i.e. nephrotic syndrome. But among the high SAAG patients, about same percent were group I patients i.e. chronic liver disease and the difference was statistically significant (*p<0.001*) (Table III). (Table IV) shows the distribution of oesophageal varices among the low and high SAAG patients. It was observed that, among the low SAAG patients, only one (16.7%) had esophageal varices and 5(83.3%) had none, whereas among the high SAAG patients, 19(76.0%) had esophageal varices i.e. 8(32.0%) grade I, 7(28.0%) grade II and 4(16.0%) grade III esophageal varices and 6(24.0%) had no varices and the difference was statistically significant (*p<0.05*) indicated that esophageal varices were associated with high SAAG. In the present study, the serum-ascites albumin gradient (SAAG) was divided into low SAAG (<1.1 gm/dl) and high SAAG (≥1.1 gm/dl). Analysis revealed that no statistically significant age difference was found between low and high SAAG groups of patients (*p>0.05*) (Table V). (Table VI) shows the distribution of SAAG by sex. It was evident that no statistically significant difference was found between low and high SAAG in the group I patients (*p>0.05*), although the proportion of female patients were higher among the high SAAG (32.0%) compared to low SAAG (16.7%). (Table VII) shows the mean distribution of SAAG patients with or without esophageal varices. Analysis revealed that a statistically significant mean SAAG difference was found between patients having EV in group I patients and group II patients (*p<0.05*) having no esophageal varices and no statistically significant mean SAAG difference was found between patients having no EV in group I and group II patients (*p>0.05*). (Table VIII) shows the sensitivity and specificity analysis of SAAG with oesophageal varices in group I patients. Analysis revealed that the sensitivity, specificity, positive predictive value, negative predictive value and predictive accuracy were 76.0%, 83.3%, 95.0%, 45.5% and 77.4% respectively.

Table-I: Age and sex distribution of the study subjects (n=55)

Age in years	Sex		Total (%)	p value
	Male (%)	Female (%)		
<5	9 (20.9)	1 (8.3)	10 (18.2)	0.266
5-9	17 (39.5)	4 (33.3)	21 (38.2)	
≥10	17 (39.5)	7 (58.3)	24 (43.6)	
Total	43 (78.2)	12 (21.8)	55 (100.0)	

P value reached from unpaired student's t test (*p>0.05*)

Table-II: Group-wise age distribution of the study subjects (n=55)

Age in years	Study subjects		Total (%)	p value
	Group I (%)	Group II (%)		
<5	4 (12.9)	6 (25.0)	10 (18.2)	0.036*
5-9	9 (29.0)	12 (50.0)	21 (38.2)	
≥10	18 (58.1)	6 (25.0)	24 (43.6)	
Total	31 (56.4)	24 (43.6)	55 (100.0)	

Group I= Patients with chronic liver disease,
Group II= patients with nephritic syndrome,
P value reached from unpaired student's t test (*p<0.05)*

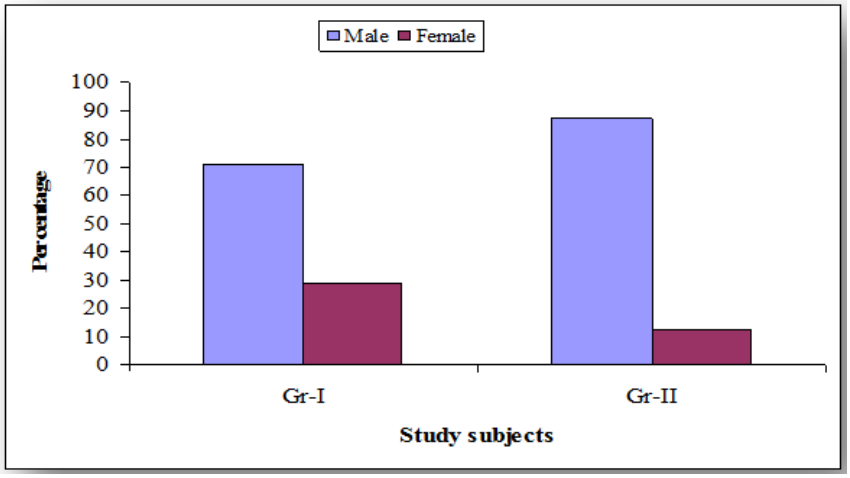


Fig-I: Sex Distribution of the Study Subjects (n=55)

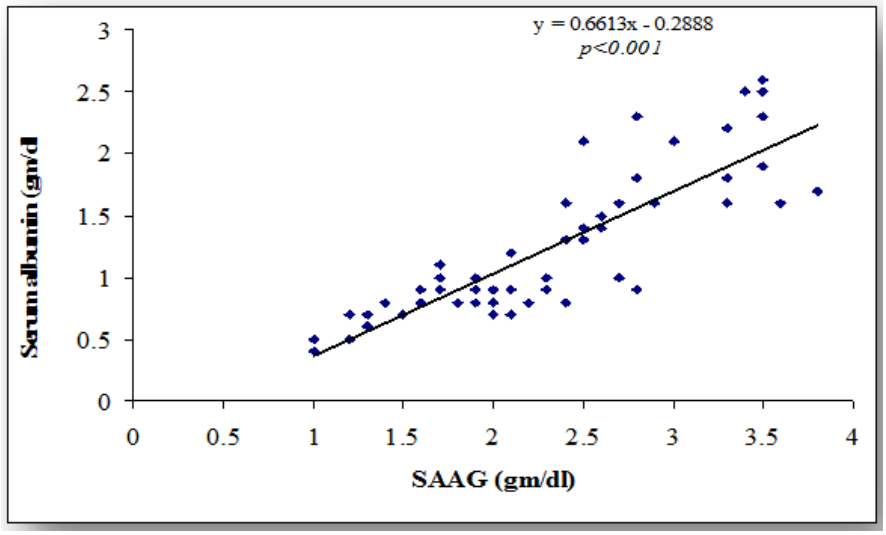


Fig-II: Correlation of serum albumin with SAAG (n=55)

Table-III: Distribution of study subjects by serum-ascites albumin gradient (n=55)

Study subjects	Serum-ascitic albumin gradient		Total (%)	p value
	Low SAAG (<1.1 gm/dl) %	High SAAG (≥1.1 gm/dl) %		
Group I	6 (20.0)	25 (100.0)	31 (56.4)	0.001***
Group II	24 (80.0)	0 (0.0)	24 (43.6)	
Total	30 (54.5)	25 (45.5)	55 (100.0)	

P value reached from chi square test (*p<0.001*)

Table-IV: Distribution of oesophageal varices by SAAG of the group I subjects (n=31)

Esophageal varices	**Serum-ascitic albumin gradient**		**Total (%)**	**p value**
	Low SAAG (<1.1 gm/dl) %	High SAAG (≥1.1 gm/dl) %		
Present	1 (16.7)	19 (76.0)	20 (64.5)	0.024*
Absent	5 (83.3)	6 (24.0)	11 (35.5)	
Total	6 (19.4)	25 (80.6)	31 (100.0)	

p value reached from chi square test with Yates's correction *$p<0.05$

Table-V: Age distribution of the group I subjects (n=31)

Age in years	Serum-ascitic albumin gradient		Total (%)	p value
	Low SAAG (<1.1 gm/dl) (%)	High SAAG (≥1.1 gm/dl) (%)		
<5	3 (50.0)	1 (4.0)	4 (12.9)	0.675
5-9	0 (0.0)	9 (36.0)	9 (29.0)	
≥10	3 (50.0)	15 (60.0)	18 (58.1)	
Total	6 (19.4)	25 (80.6)	31 (100.0)	

p value reached from fisher's exact test after collapsing two groups of age (<10 vs. ≥10) (*p>0.05*)

Table-VI: Sex Distribution of the Group I Subjects (n=31)

Sex	**Serum-ascitic albumin gradient**		**Total (%)**	**p value**
	Low SAAG (<1.1 gm/dl) %	High SAAG (≥1.1 gm/dl) %		
Male	5 (83.3)	17 (68.0)	22 (71.0)	0.642
Female	1 (16.7)	8 (32.0)	9 (29.0)	
Total	6 (19.4)	25 (80.6)	31 (100.0)	

Figure in parenthesis indicate percentage, p value reached from fisher's exact test (*p>0.05*)

Table-VII: Mean Ascites Albumin Gradient of subjects by Oesophageal Varices (n=55)

Oesophageal varices	Serum-ascitic albumin gradient			p value
	N	Mean±SD	Range	
Group I				
Present (a)	20	1.9±0.5	0.9-2.6	a vs c p<0.05
Absent (b)	11	1.2±0.3	0.7-1.6	b vs c p>0.05
group II (c)	24	0.8±0.2	0.4-1.0	

p value reached from unpaired student's t test

Table-VIII: Sensitivity and specificity analysis of SAAG with *oesophageal* varices in Gr. I patients (n=31)

Oesophageal varices	Serum-ascitic albumin gradient		Total (%)	p value
	High SAAG (≥1.1 gm/dl) (%)	Low SAAG (<1.1 gm/dl) (%)		
Present	19 (76.0)	1 (16.7)	20 (64.5)	0.024*
Absent	6 (24.0)	5 (83.3)	11 (35.5)	
Total	25 (80.6)	6 (19.4)	31 (100.0)	

Sensitivity	**76.1%**
Specificity	83.3%
Positive predictive value	95.0%
Negative predictive value	45.5%
Predictive accuracy	77.4%

P value reached from chi square test with Yates's correction *$p<0.05$*

DISCUSSION

According to equilibrium of starling law, whenever ascites is related to portal hypertension, increment of portal pressure should be counter balanced by an increased difference of osmotic forces and thus creating increased difference of albumin concentrations between serum and ascitic fluid. The serum-ascites albumin concentration gradient is an indicator of portal hypertension [16-21] and there is a direct relationship between SAAG and portal hypertension. Variceal bleeding, in the form of haematemesis and / or malaena occurs in approximately one third of patients with portal hypertension [22]. The high mortality rate associated with the first episode of gastrointestinal bleeding has led to the search for identification of a high risk group patient which can then be offered prophylactic therapy. Upper gastrointestinal endoscopy is recommended to screen patients at a high risk for GI haemorrhage. However, as the facility for endoscopy is not available everywhere in a developing country like Bangladesh, attempts have been made to identify easily obtainable clinical variables to which can provide the same information. This study was designed to determine the correlation and association between the level of SAAG and the features of portal hypertension, mainly the presence and grade of oesophageal varices found on upper gastrointestinal endoscopy. This would permit the use of SAAG as a preliminary indirect parameter that would indicate the presence of oesophageal varices as a manifestation of portal hypertension. The SAAG is the difference between serum albumin and ascitic fluid albumin concentration. It is a subtraction not a ratio. The SAAG is proportional to the presence of pressure gradient between portal vein and intra-abdominal cavity. The SAAG can reliably separate the aetiology of ascites into two categories: the high gradient (≥ 1.1 gm/dl) from portal hypertension and low gradient (<1.1 gm/dl) other than portal hypertension in 97% of patients (Rector et al., 1984). In the present study SAAG correctly classified ascites of chronic liver disease patients as high gradient in 81% cases and of nephrotic syndrome patients as low gradient in 100% cases. This finding is in consistent with the finding of [8, 9, 17]. Both chronic liver disease and nephrotic syndrome cause transudative ascites. But the mean SAAG value of chronic liver disease patients were significantly higher than that of nephrotic syndrome patients [12] also found similar result in their studies. This mean SAAG value difference indicates that portal hypertension is the cause of this difference. By upper gastrointestinal endoscopy oesophageal varices was found in 65% of patients with chronic liver disease. Among them 81% of the high SAAG patients had oesophageal varices. Previous researchers have reported similar endoscopic findings in the literature [23, 24]. These findings suggest high SAAG value significantly correlates with oesophageal varices. Similarly [20, 25] found that high SAAG value correlates with portal hypertension. Significant difference was present among the SAAG value of chronic liver disease with oesophageal varices patient and nephrotic syndrome patient. But statistically no significant difference was present among the SAAG value of nephritic syndrome patients and patients of chronic liver disease without oesophageal varices. These findings were consistent with the findings of [8, 9]. Therefore, high SAAG value correlates with presence of oesophageal varices and hence portal hypertension. The patients, who are found to have high SAAG value, should undergo endoscopy as soon as possible. This may lead to timely diagnosis and prevention of possible life threatening upper

gastrointestinal bleeding. In present study, oesophageal varices were found in only17% of chronic liver disease patients with low SAAG. It is consistent with the finding of [18] but differ from [8] who got slightly higher percentage of patients with oesophageal varices. This difference is possibly due to small sample size of the later author. So in case of low SAAG, there is less chance of presence of oesophageal varices and therefore endoscopy can be delayed. Of course, both groups of chronic liver disease patients with ascites should be transferred to a tertiary care facility, but having a SAAG values may help in terms of to take decision of doing endoscopy. In this study the SAAG strongly differentiated chronic liver disease patients with oesophageal varices from those without oesophageal varices. In the study sensitivity of predicting oesophageal varices (76%) was reasonable and specificity (83%) was good with a high positive predictive value (95%). These results are similar to earlier studies but predictive accuracy (77%) was slightly lower than that of previous studies [19]. These differences may be due to small sample size of the present study and difference in study population. It was found in the present study that as the SAAG values raised the number of presence of oesophageal varices also increased. It was found that oesophageal varices were present in 50% patients in the SAAG range of 1.10-1.49 mg/dl, 73% of patients in the SAAG range of 1.50-1.99 mg/dl and 100% when SAAG value was ≥2gm/ dl. These findings are consistent with the findings of [18]. These findings strongly supported the correlation between the SAAG and portal hypertension. Therefore, it can be said that when higher SAAG value is found chance of presence of portal hypertension is more.

LIMITATIONS OF THE STUDY

The sample size of this study was small. So further evaluation with large sample size is needed to come to a definite conclusion

CONCLUSION AND RECOMMENDATIONS

From the present study it may be concluded that, the serum-ascites albumin gradient (SAAG), the difference between serum albumin and ascitic fluid albumin concentration correlates directly with portal pressure and a high SAAG value indicates portal hypertension. High SAAG denotes higher chances of presence of oesophageal varices in children with ascites due to chronic liver disease. SAAG value of ≥1.1gm/dl is a useful indicator to predict the oesophageal varices in children with ascites and can guide the paediatricians to determine the urgency of care. Another aspect of studying the SAAG value to detect the grades of oesophageal varices is important. Because grading of oesophageal varices has prognostic value. Grades of oesophageal varices and upper gastrointestinal bleeding have a positive correlation with the high SAAG value.

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