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A Study on Correlation between Serum Ascites Albumin Gradient (SAAG) and Esophageal Varices in Patients with Liver Cirrhosis in a Tertiary Level Hospital

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Abstract

Portal hypertension in liver cirrhosis leads to ascites and esophageal varices, and serum–ascites albumin gradient (SAAG) may reflect the portal hypertensive state. The aim of this study was to evaluate the relationship between serum–ascites albumin gradient categories and endoscopic grades of esophageal varices in patients with liver cirrhosis. This hospital-based cross-sectional observational study was conducted among indoor patients admitted to the Department of Medicine and Hepatology, Mymensingh Medical College Hospital, Mymensingh, Bangladesh, from April 2019 to March 2020. Fifty adult patients with liver cirrhosis were enrolled consecutively. Serum and ascitic fluid albumin were measured to calculate serum–ascites albumin gradient, and esophageal varices were graded by upper gastrointestinal endoscopy (Grades I–IV). The mean age was 37.05 ± 9.32 years, and 34 (68.0%) participants were male. Abdominal distension was present in 50 (100.0%), and ascites was detected in 45 (90.0%) patients consecutively. Hepatitis B virus was the leading etiology, 37 (74.0%). Serum–ascites albumin gradient values were 1.1–1.49 g/dL in 8 (16.0%), 1.50–1.99 g/dL in 29 (58.0%), and ≥2.0 g/dL in 13 (26.0%) patients consecutively. Variceal grades were Grade I 9 (18.0%), Grade II 17 (34.0%), Grade III 14 (28.0%), and Grade IV 10 (20.0%) consecutively. A progressive shift toward higher variceal grades was observed with increasing serum-ascites albumin gradient (SAAG), with the ≥2.0 g/dL group showing predominance of Grade IV varices, 9 (90.0%). Higher serum–ascites albumin gradient values clustered with more advanced esophageal variceal grades in cirrhosis, supporting its potential role as an adjunctive marker for endoscopic risk stratification.

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Introduction

Liver cirrhosis remains a major cause of preventable morbidity and premature mortality worldwide, with substantial country-level variation over time that reflects underlying etiologic patterns and health system capacity.[1] Clinically, the transition from compensated disease to decompensation is largely mediated by portal hypertension, which drives hallmark complications including ascites, gastro esophageal varices, and variceal hemorrhage.[2,3] Esophageal varices represent a key portal-hypertension milestone because their presence and endoscopic features stratify bleeding risk, guide primary prophylaxis, and anchor surveillance strategies; in contemporary practice, screening and surveillance aim to identify varices at high risk of bleeding so that non-selective beta-blockers or endoscopic variceal ligation can be implemented in time.[2,4] Variceal bleeding remains a life-threatening

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emergency and continues to be a major contributor to cirrhosis-related death despite advances in acute care and secondary prevention, underscoring why earlier identification of clinically significant portal hypertension and high-risk varices is central to outcome-oriented cirrhosis management.[5,6] At the same time, universal endoscopic screening is constrained in many real-world settings by procedure availability, cost, invasiveness, and patient acceptability, which has fueled sustained. interest in non-invasive or minimally invasive predictors that can help triage who most needs endoscopy, particularly where endoscopy resources are limited.[2,7] Several non-invasive approaches have been evaluated, including platelet-based and spleen-based indices, with platelet count to bipolar spleen diameter ratio demonstrating utility for predicting the presence of esophageal varices in specific populations, providing proof-of-concept that portal hypertension complications can be partially risk-stratified using routine clinical parameters.[8] Ascites, meanwhile, is the most frequent decompensating event and a common reason for hospital admission in cirrhosis, and its evaluation is standardized in guideline-based care because ascitic fluid assessment helps define mechanism and informs downstream management decisions.[9] Within ascites work-up, the serum–ascites albumin gradient, calculated as serum albumin minus ascitic fluid albumin, is widely used because it reflects the oncotic and hydrostatic forces that accompany portal hypertension, and it provides clinically actionable discrimination between portal hypertension–related ascites and other causes.[10,11] This physiologic interpretation is particularly relevant to variceal disease because portal hypertension is the shared causal pathway for both high-gradient ascites and the development and progression of esophageal varices, suggesting that serum–ascites albumin gradient may serve as an accessible surrogate marker of portal hypertensive burden in routine inpatient practice.[3,12] However, existing evidence is mixed regarding how well the gradient tracks the extent of portal hypertensive changes, and its practical value may depend on study population, disease stage, and the specific portal hypertension outcome being predicted, which justifies continued evaluation in diverse clinical settings.[12] Importantly, some open-access clinical studies have directly explored the gradient in relation to esophageal varices, including work assessing the prediction of esophageal varices using serum–ascites albumin gradient in cirrhotic patients, supporting the plausibility of using a routinely measured parameter to anticipate endoscopic findings.[13] In Bangladesh, where cirrhosis frequently presents at a decompensated stage and endoscopy capacity can be variably distributed across facilities, a pragmatic marker that is already embedded in ascites evaluation may be particularly useful for risk stratification and rational endoscopy referral, while remaining aligned with guideline-concordant ascites assessment.[7,9] Therefore, the present study was undertaken to evaluate the relationship between serum–ascites albumin gradient categories and endoscopic grades of esophageal varices among patients with liver cirrhosis in a tertiary care hospital setting.

Methods

This hospital-based cross-sectional observational study was conducted among indoor patients admitted to the Department of Medicine and Hepatology, Mymensingh Medical College Hospital, Mymensingh, Bangladesh, from April 2019 to March 2020. Ethical approval for this study was obtained from the Institutional Review Board (IRB) of Mymensingh

Medical College. IRB Memo no. MMC/IRB/2019/105, Dated: 28/01/2019. Adult patients with liver cirrhosis admitted during the study period were screened, and eligible participants were enrolled using a non-probability & consecutive approach based on feasibility; a total of 50 patients were included.

These were the following criteria for eligibility as study participants:

Inclusion criteria: Adult patients with liver cirrhosis aged 18–74 years, of either sex, who were willing to participate.

Exclusion criteria: Patients age <18 years or >74 years, haemodynamic instability, active gastrointestinal bleeding at the time of enrollment, and/or prior endoscopic therapy (sclerotherapy or band ligation), receipt of prophylactic treatment for portal hypertension or interferon therapy, portal vein thrombosis or hepatoma on abdominal ultrasound, hypoalbuminaemia due to nephrotic syndrome, malnutrition or malabsorption, and unwillingness to participate.

Operational definitions: Chronic liver disease was defined as hepatic injury, inflammation and/or fibrosis persisting for more than 6 months, and cirrhosis diagnosis was based on clinical features with supportive biochemical and ultra sonographic findings. Esophageal varices were assessed by upper gastrointestinal endoscopy and graded using Paquet's grading system (Grades I–IV). Serum–ascites albumin gradient was defined as serum albumin concentration minus ascitic fluid albumin concentration; it was used as a marker related to portal hypertension physiology.

Data collection procedures: Data were collected using a structured questionnaire and a pre-structured case record form completed by the investigator, covering sociodemographic variables, clinical history, and examination findings. After enrolment and assignment of a unique ID, participants were briefed on the study objectives, risks and benefits, and confidentiality. As part of routine evaluation, 10 mL of blood was collected and sent to the clinical pathology and biochemistry laboratories; serum–ascites albumin gradient was then evaluated as per the operational definition.

Outcomes: The primary analytic outcome was the relationship between serum–ascites albumin gradient and endoscopic grade of esophageal varices.

Statistical analysis: Data were recorded on a predesigned proforma, managed in Microsoft Excel, and analyzed using SPSS version 22.0. Continuous variables were summarized using mean and standard deviation, categorical variables using frequencies and percentages, and results were presented in tables. A p value <0.05 was considered statistically significant.

Results

Table I: Socio demographic Characteristics of the Study Participants (n = 50)

Characteristic	n	%
Age group (years)		
18–30	8	16.0
31–45	22	44.0
46–60	15	30.0
>60	5	10.0
Sex		
Male	34	68.0
Female	16	32.0
Residence		
Rural	18	36.0
Urban	32	64.0
Occupation		
Service holder	6	12.0
Business	9	18.0
Worker	12	24.0
Housewife	13	26.0
Farmer	10	20.0
Socioeconomic class		
Poor	24	48.0
Middle class	17	34.0
Upper class	9	18.0

Table-I shows among the 50 participants, the largest age group was 31–45 years, 22 (44.0%), followed by 46–60 years, 15 (30.0%), 18–30 years, 8 (16.0%), and >60 years, 5 (10.0%). Males comprised 34 (68.0%) and females 16 (32.0%). Most participants were from urban areas, 32 (64.0%), while 18 (36.0%) were rural residents. Regarding occupation, housewives were the most frequent, 13 (26.0%), followed by workers, 12 (24.0%), farmers, 10 (20.0%), business, 9 (18.0%), and service holders, 6 (12.0%). Nearly half belonged to the poor socioeconomic class, 24 (48.0%), while 17 (34.0%) were middle class and 9 (18.0%) were upper class.

Table II: Clinical Presentation of Cirrhosis among Study Participants (n = 50)

Clinical feature	n	%
Symptoms		
Abdominal distension	50	100.0
Jaundice	41	82.0
Vague abdominal pain	38	76.0
Vomiting	22	44.0
Altered consciousness	15	30.0
Hematemesis	9	18.0
Respiratory distress	8	16.0
Melena	6	12.0
Scanty micturition	4	8.0
Clinical signs		
Ascites	45	90.0
Icterus	43	86.0
Loss of body hair	30	60.0
Spider naevi	24	48.0
Palmar erythema	7	14.0
Clubbing	5	10.0

Table-II shows abdominal distension was reported by all participants, 50 (100.0%), while jaundice was present in 41 (82.0%) and vague abdominal pain in 38 (76.0%). Vomiting occurred in 22 (44.0%), altered consciousness in 15 (30.0%), hematemesis in 9 (18.0%), respiratory distress in 8 (16.0%), melena in 6 (12.0%), and scanty micturition in 4 (8.0%). On examination, ascites was documented in 45 (90.0%) and icterus in 43 (86.0%), with additional stigmata including loss of body hair in 30 (60.0%), spider naevi in 24 (48.0%), palmar erythema in 7 (14.0%), and clubbing in 5 (10.0%).

Table III: Etiology of Cirrhosis in the Study Participants (n = 50).

Etiology	n	%
Hepatitis B virus	37	74.0
Hepatitis C virus	10	20.0
Alcohol-related	1	2.0
Others	2	4.0

Table-III shows Hepatitis B virus was the predominant etiology of cirrhosis, affecting 37 (74.0%) participants, followed by hepatitis C virus in 10 (20.0%). Alcohol-related cirrhosis was uncommon, identified in 1 (2.0%) participant, and other causes accounted for 2 (4.0%) participants.

Table IV: Distribution of Serum-Ascites Albumin Gradient Categories and Esophageal Variceal Grades (n = 50).

Variable	Category	n	%
Serum–ascites albumin gradient (g/dL)	1.1–1.49	8	16.0
	1.50–1.99	29	58.0
	≥2.0	13	26.0
Esophageal varices (endoscopic grade)	Grade I	9	18.0
	Grade II	17	34.0
	Grade III	14	28.0
	Grade IV	10	20.0

Table IV shows serum–ascites albumin gradient, most participants fell within 1.50–1.99 g/dL, 29 (58.0%), while 13 (26.0%) had values ≥2.0 g/dL and 8 (16.0%) had values 1.1–1.49 g/dL. Endoscopically, Grade II esophageal varices were most frequent, 17 (34.0%), followed by Grade III, 14 (28.0%), Grade IV, 10 (20.0%), and Grade I, 9 (18.0%) consecutively.

Table V: Association between Serum–Ascites Albumin Gradient Category and Esophageal Variceal Grade (n=50).

Serum–ascites albumin gradient (g/dL)	Grade I (n = 9)	Grade II (n = 17)	Grade III (n = 14)	Grade IV (n = 10)	Total (N = 50)
1.1–1.49 (n = 8)	7 (87.5)	1 (12.5)	0 (0.0)	0 (0.0)	8 (100.0)
1.50–1.99 (n = 29)	2 (6.9)	16 (55.2)	10 (34.5)	1 (3.4)	29 (100.0)
≥2.0 (n = 13)	0 (0.0)	0 (0.0)	4 (30.8)	9 (69.2)	13 (100.0)
Total	9 (18.0)	17 (34.0)	14 (28.0)	10 (20.0)	50 (100.0)

Table-V shows a stepwise shift toward higher variceal grades with increasing gradient: Grade I predominated at 1.1–1.49 g/dL (7/8, 87.5%), mixed Grade II–III predominated at 1.50–1.99 g/dL (16/29, 55.2%; 10/29, 34.5%), and Grade IV predominated at ≥2.0 g/dL (9/13, 69.2%).

Discussion

This present study evaluated the relationship between serum–ascites albumin gradient and esophageal variceal grade in patients with liver cirrhosis and demonstrated a clear gradient–severity pattern within a relatively young, predominantly male cohort. The mean age of 37.05 ± 9.32 years, with the largest group between 31–45 years (44.0%), reflects the pattern observed in several South Asian hospital-based studies, where cirrhosis frequently presents in early and middle adulthood, often related to chronic viral hepatitis.[14,15] Male predominance in the present study, 68.0%, is consistent with global and regional observations indicating higher cirrhosis burden among men, attributed to differential exposure to viral infection, behavioral risk factors, and possibly sex-based biological susceptibility.[1,16] The predominance of patients from urban areas (64.0%) and from lower socioeconomic strata (48.0%) may reflect referral dynamics to tertiary centers and the established association between socioeconomic disadvantage and advanced chronic liver disease.[17]

Clinically, abdominal distension was universal (100.0%) and ascites was detected in 90.0% of patients, indicating that most participants were in a decompensated stage at presentation. Ascites is widely recognized as the most common first decompensating event in cirrhosis and a marker of clinically significant portal hypertension.[3] The high frequency of jaundice (82.0%) and icterus (86.0%) further supports advanced disease status, comparable to reports from regional perspective cohorts where decompensated cirrhosis frequently presents with ascites and jaundice as dominant features.[18,19] Altered consciousness in 30.0% of patients suggests concurrent hepatic encephalopathy in a substantial proportion, consistent with established descriptions of neuropsychiatric manifestations in advanced chronic liver disease.[20] Upper gastrointestinal bleeding manifestations, hematemesis (18.0%) and melena (12.0%), were observed in a subset of patients, aligning with literature describing variceal hemorrhage as a major but not universal complication in cirrhotic populations.[6]

Regarding etiology, hepatitis B virus was the predominant cause, accounting for 74.0% of cases, followed by hepatitis C virus at 20.0%, while alcohol-related cirrhosis was uncommon (2.0%). This distribution mirrors earlier Bangladeshi data demonstrating hepatitis B as the leading cause of

chronic liver disease and cirrhosis in hospital-based settings.[21,22] In contrast to Western cohorts where alcohol-related cirrhosis contributes substantially to overall burden, viral hepatitis remains the principal driver in many South Asian contexts, consistent with regional epidemiological analyses.[1]

The distribution of serum–ascites albumin gradient values showed that 58.0% of patients had levels between 1.50–1.99 g/dL and 26.0% had values ≥2.0 g/dL, indicating that most cases had portal hypertension–related ascites. The clinical utility of a threshold ≥1.1 g/dL as a marker of portal hypertension is well established.[10] Moreover, higher gradient values have been associated with more pronounced portal hypertensive changes, providing physiological plausibility for exploring its relationship with variceal severity.[12]

Endoscopically, Grade II varices were most frequent (34.0%), followed by Grade III (28.0%) and Grade IV (20.0%), meaning that nearly half of patients (48.0%) had large varices. Similar distributions have been reported in decompensated cirrhosis cohorts where moderate-to-large varices predominate.[23] The principal finding of this study was the progressive shift toward higher variceal grades with increasing serum–ascites albumin gradient. Patients with gradient 1.1–1.49 g/dL predominantly had Grade I varices (77.8%), whereas those with ≥2.0 g/dL showed clustering of Grade III (28.5%) and especially Grade IV varices (90.0%), with absence of lower grades in this group. This pattern parallels findings from studies evaluating the predictive role of serum–ascites albumin gradient for esophageal varices, which reported higher gradients associated with large varices and advanced portal hypertension.[13] Given that variceal size correlates with bleeding risk and clinical outcomes, the observed gradient–severity relationship suggests that serum–ascites albumin gradient may serve as a simple adjunctive marker for risk stratification in resource-limited settings.[5]

Limitations of the study

This was a single-center, hospital-based cross-sectional observational study with a small sample size (n=50), which limits generalizability. In addition, portal pressure was not directly measured (for example, hepatic venous pressure gradient), and selection bias among admitted patients cannot be excluded.

Conclusion

In this tertiary-hospital cohort of liver cirrhosis patients, ascites and moderate-to-large esophageal varices were common. Higher serum–ascites albumin gradient (SAAG) values showed a consistent, stepwise clustering with more advanced esophageal variceal grades, with the highest gradient group predominantly demonstrating Grade IV varices, suggesting that the serum–ascites albumin gradient (SAAG) may be a useful adjunct marker for endoscopic risk stratification in decompensated cirrhosis patients.

Recommendation

CLD patients with ascites where endoscopy is not available, serum ascites albumin gradient (SAAG) should be routinely integrated and patients with high SAAG values should be started treatment immediately.

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