International Journal of Medicine
pISSN 1468-3814 | eISSN 2667-7008
https://theinternationalmedicine.com/

Original Researcher Article *DOI: 10.61336/im/26-09-132*

Comparative Evaluation of Serum-Ascites Albumin Gradient and Ascitic Fluid Total Protein in the Evaluation of Ascites in Chronic Liver Disease.

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Received: 04-08-2026
Revised: 24-08-2026
Accepted: 08-09-2026
Published: 25-09-2026

Abstract

Background: Ascites is a common complication of chronic liver disease. Serum-ascites albumin gradient (SAAG) and ascitic fluid total protein are used to help evaluate its cause and relationship to portal hypertension. Aim: To compare SAAG and ascitic fluid total protein in the evaluation of ascites in patients with chronic liver disease. ***Methods***: This hospital-based cross-sectional study included 92 patients with chronic liver disease and ascites. Paired serum and ascitic fluid measurements were used to calculate SAAG and determine ascitic fluid total protein. Diagnostic performance was assessed against the clinical classification of portal hypertension. ***Results***: Of 92 participants, 71 were classified as having portal hypertension and 21 as not having portal hypertension. High SAAG (≥1.1 g/dL) was observed in 73 (79.3%) participants and was more frequent in the portal hypertension group than in the comparison group (94.4% vs 28.6%; p<0.001). Mean SAAG was 1.78 (SD 0.42) g/dL versus 0.96 (SD 0.39) g/dL (p<0.001). Mean ascitic fluid total protein was 2.18 (SD 0.71) g/dL versus 2.73 (SD 0.76) g/dL (p=0.004); the proportion with protein <2.5 g/dL did not differ significantly (p=0.752). SAAG had higher estimated sensitivity and accuracy than AFTP <2.5 g/dL. The AFTP positive predictive value calculated from the displayed counts is 78.1%, not 87.7%. ***Conclusion***: In these illustrative data, SAAG showed higher sensitivity and overall accuracy than the protein cutoff for identifying portal hypertension. Both measures should be interpreted with clinical findings. These results should not be reported as study findings until verified against the original patient data.

Keywords: Serum-ascites albumin gradient; Ascitic fluid protein; Chronic liver disease.

INTRODUCTION

Ascites is the abnormal accumulation of fluid in the peritoneal cavity and is a frequent complication of chronic liver disease, particularly cirrhosis. Its development usually reflects portal hypertension and is associated with further complications, including spontaneous bacterial peritonitis, renal dysfunction and electrolyte imbalance. Because other conditions such as malignancy, tuberculosis, cardiac failure and pancreatic disease can also cause ascites, determining its underlying mechanism is important for appropriate treatment.[1] The initial assessment generally includes clinical evaluation, imaging and diagnostic paracentesis, with ascitic fluid analysis and a blood sample obtained at the same time.[1,2]

The serum-ascites albumin gradient (SAAG) is calculated by subtracting the ascitic fluid albumin concentration from the serum albumin concentration. A SAAG of ≥1.1 g/dL supports portal hypertension, while a value below this threshold suggests a non-portal hypertensive cause. In contrast, ascitic fluid total protein (AFTP) has traditionally been used to classify fluid as transudative or exudative, commonly using a threshold of 2.5 g/dL. However, protein concentration alone can be misleading: some patients with cirrhosis have higher-protein ascites, while diuretic treatment or infection may alter the fluid profile.[1,3]

Comparative studies have reported that SAAG may identify portal hypertension more sensitively than the transudate-exudate classification based on AFTP, though performance varies by population and reference standard. In one study, high SAAG had 94.3% sensitivity for portal hypertension, whereas low ascitic protein classification had lower sensitivity.[3] A more recent cohort also found that SAAG performance was not uniform across all causes of ascites, supporting

interpretation alongside clinical findings rather than in isolation.[4] Research comparing albumin and protein gradients continues to examine how these measurements can assist in differentiating ascites by cause.[5]

AIM

To compare serum-ascites albumin gradient and ascitic fluid total protein in the evaluation of ascites in patients with chronic liver disease.

OBJECTIVES

1. To estimate SAAG in patients with chronic liver disease and ascites.
2. To measure ascitic fluid total protein and describe its distribution in the study participants.
3. To compare SAAG and ascitic fluid total protein with the clinical assessment of portal hypertension and the cause of ascites.

MATERIALS AND METHODS

Source of Data

Data were obtained from eligible patients with chronic liver disease and clinically or radiologically confirmed ascites who attended or were admitted to the Department of Medicine during the study period. Information was collected from patient interviews, clinical examinations, laboratory investigations, imaging records and other relevant medical records.

Study Design

A hospital-based, observational cross-sectional study was conducted. Serum and ascitic fluid measurements were assessed during the same episode of evaluation.

Study Location

The study was conducted in the Department of Medicine, in collaboration with the hospital's clinical biochemistry laboratory.

Study Duration

The study was conducted over 12 months.

Sample Size

The sample size was estimated using the single-proportion formula, based on the reported sensitivity of high SAAG for identifying portal hypertension in an earlier study.[3]

$$n=(Z_{(1-\alpha/2)}^2 \text{ } \text{ } p(1-p))/d^2$$

Where:

- $Z_{(1-\alpha/2)}=1.96$ for a 95% confidence level
- $p=0.943$, the anticipated sensitivity of high SAAG
- $d=0.05$, the absolute precision

$$n=((1.96)^2\times0.943\times(1-0.943))/(0.05)^2 =82.7$$

Thus, 83 evaluable participants were required. Allowing approximately 10% for incomplete records or inadequate samples:

$$n_{\text{adjusted}} =83/(1-0.10)=92.2$$

The adjusted minimum sample size was 92 participants. Consecutive eligible patients were recruited until the required sample size was reached.

Inclusion Criteria

- Patients aged 18 years or older.
- Patients with chronic liver disease diagnosed on the basis of history, examination, laboratory findings and imaging, as applicable.
- Patients with ascites confirmed clinically or by ultrasonography.
- Patients who provided written informed consent.

Exclusion Criteria

- Patients with ascites primarily attributed to a non-hepatic cause, such as malignancy, tuberculosis, cardiac failure, nephrotic syndrome or pancreatic disease, based on clinical assessment and available investigations.
- Patients who had received albumin infusion or undergone large-volume paracentesis immediately before paired sample collection, if this was likely to affect the measurements.

- Patients with inadequate serum or ascitic fluid samples for analysis.
- Patients who declined consent.

Procedure and Methodology

After obtaining ethics committee approval and written informed consent, eligible patients were assessed using a structured case record form. A detailed history was recorded, including presenting symptoms, duration of abdominal distension, relevant liver disease history, alcohol use where appropriate, previous treatment and use of diuretics. A clinical examination was performed, including assessment for ascites and signs of chronic liver disease or portal hypertension.

Relevant investigations were reviewed or performed as clinically indicated. These included liver and renal function tests, complete blood count, coagulation profile and abdominal ultrasonography. Diagnostic paracentesis was performed under aseptic precautions by a trained clinician when clinically indicated. Paired venous blood and ascitic fluid samples were obtained as close together in time as practicable, preferably before administration of albumin or other interventions that could alter the measurements. Diagnostic evaluation of ascites commonly includes serum albumin, ascitic fluid albumin and total protein.[1,2]

Sample Processing

Blood was collected in an appropriate plain tube and allowed to clot. Serum was separated by centrifugation and analyzed for albumin using the hospital laboratory's validated method. Ascitic fluid was collected in a sterile container and transported promptly to the laboratory. Ascitic fluid albumin and total protein were measured using the laboratory's routine validated biochemical methods. Where clinically indicated, fluid cell count, differential count, culture or other investigations were performed as part of patient care.

SAAG was calculated for each patient as:

$$\text{SAAG (g/dL)} = \text{Serum albumin (g/dL)} - \text{Ascitic fluid albumin (g/dL)}$$

SAAG was classified as high at ≥1.1 g/dL and low at <1.1 g/dL. Ascitic fluid total protein was categorized using 2.5 g/dL as the conventional threshold; results were also retained as continuous measurements for analysis.

Statistical Methods

Data were entered into a spreadsheet and analyzed using SPSS 28.0 version. Continuous variables were summarized as mean and standard deviation when approximately normally distributed, or median and interquartile range when skewed. Categorical variables were summarized as frequencies and percentages.

The distribution of SAAG and ascitic fluid total protein was described, and their relationship was assessed using Pearson's or Spearman's correlation, as appropriate. Categorical comparisons were assessed using the chi-square test or Fisher's exact test. If a suitable clinical reference classification for portal hypertension was available, sensitivity, specificity, positive predictive value, negative predictive value and accuracy were calculated for the predefined SAAG and AFTP thresholds, with 95% confidence intervals. Receiver operating characteristic analysis was used where appropriate. A two-sided p value <0.05 was considered statistically significant.

Data Collection

Data were collected prospectively using a pretested structured case record form. Each participant was assigned a study identification number, and identifying information was kept separate from the analysis dataset. The form captured demographic details, relevant clinical history, examination findings, chronic liver disease profile, ascites assessment, relevant investigation results, serum albumin, ascitic fluid albumin, ascitic fluid total protein and calculated SAAG. The completed records were checked for completeness before data entry.

RESULTS

Table 1. Sociodemographic and clinical profile by clinical assessment of portal hypertension (N=92)

Characteristic	Portal hypertension (n=71)	No portal hypertension (n=21)	Effect estimate (95% CI)	Test statistic	p value
Age, years, mean (SD)	52.4 (11.8)	48.1 (13.2)	Mean difference: 4.3 (−2.3 to 10.9)	t=1.34	0.184
Male sex, n (%)	47 (66.2)	11 (52.4)	OR: 1.78 (0.66-4.78)	χ^2=1.33	0.249
Alcohol-related CLD, n (%)	33 (46.5)	7 (33.3)	OR: 1.74 (0.63-4.82)	χ^2=1.11	0.292
Child-Pugh class C, n (%)	29 (40.8)	5 (23.8)	OR: 2.21 (0.73-6.71)	χ^2=1.96	0.161
Serum albumin, g/dL, mean (SD)	2.63 (0.52)	2.98 (0.61)	Mean difference: −0.35 (−0.64 to −0.06)	t=−2.39	0.019

SAAG, g/dL, mean (SD)	1.78 (0.42)	0.96 (0.39)	Mean difference: 0.82 (0.62-1.02)	t=8.31	<0.001
Ascitic fluid total protein, g/dL, mean (SD)	2.18 (0.71)	2.73 (0.76)	Mean difference: −0.55 (−0.92 to −0.18)	t=−2.94	0.004

Among the 92 participants, 71 had clinical evidence of portal hypertension and 21 did not. Mean age was 52.4 years in the portal hypertension group and 48.1 years in the comparison group; this difference was not statistically significant (p=0.184). Male sex, alcohol-related chronic liver disease and Child-Pugh class C were also more frequent in the portal hypertension group, but none of these differences reached statistical significance. Mean serum albumin was lower in participants with portal hypertension (2.63 vs 2.98 g/dL; mean difference −0.35, 95% CI −0.64 to −0.06; p=0.019). Their mean SAAG was higher (1.78 vs 0.96 g/dL; p<0.001), while mean ascitic fluid total protein was lower (2.18 vs 2.73 g/dL; p=0.004).

Table 2. SAAG distribution and comparison with clinical assessment of portal hypertension (N=92)

SAAG measure	Overall (N=92)	Portal hypertension (n=71)	No portal hypertension (n=21)	Effect estimate (95% CI)	Test statistic	p value
SAAG, g/dL, mean (SD)	1.59 (0.56)	1.78 (0.42)	0.96 (0.39)	Mean difference: 0.82 (0.62-1.02)	t=8.31	<0.001
High SAAG (≥1.1 g/dL), n (%)	73 (79.3)	67 (94.4)	6 (28.6)	OR: 41.88 (10.72-163.54)	χ^2=39.20	<0.001
Low SAAG (<1.1 g/dL), n (%)	19 (20.7)	4 (5.6)	15 (71.4)			

The overall mean SAAG was 1.59 (SD 0.56) g/dL. Mean SAAG was higher among participants with portal hypertension than among those without it (1.78 vs 0.96 g/dL; mean difference 0.82, 95% CI 0.62-1.02; p<0.001). Overall, 73 participants (79.3%) had a high SAAG (≥1.1 g/dL). High SAAG was present in 94.4% of the portal hypertension group compared with 28.6% of those without portal hypertension; the association was statistically significant (OR 41.88, 95% CI 10.72-163.54; p<0.001).

Table 3. Ascitic fluid total protein distribution and comparison with clinical assessment of portal hypertension (N=92)

Ascitic fluid total protein measure	Overall (N=92)	Portal hypertension (n=71)	No portal hypertension (n=21)	Effect estimate (95% CI)	Test statistic	p value
Total protein, g/dL, mean (SD)	2.31 (0.74)	2.18 (0.71)	2.73 (0.76)	Mean difference: −0.55 (−0.92 to −0.18)	t=−2.94	0.004
Low protein (<2.5 g/dL), n (%)	64 (69.6)	50 (70.4)	14 (66.7)	OR: 1.19 (0.43-3.30)	χ^2=0.10	0.752
High protein (≥2.5 g/dL), n (%)	28 (30.4)	21 (29.6)	7 (33.3)			

The overall mean ascitic fluid total protein was 2.31 (SD 0.74) g/dL. Mean protein was lower in participants with portal hypertension than in those without it (2.18 vs 2.73 g/dL; mean difference −0.55, 95% CI −0.92 to −0.18; p=0.004). Low protein (<2.5 g/dL) was observed in 64 participants (69.6%), with similar proportions in the portal hypertension and non-portal hypertension groups (70.4% and 66.7%, respectively; OR 1.19, 95% CI 0.43-3.30; p=0.752). Thus, the difference in mean protein levels was significant, while the categorical comparison at the 2.5 g/dL cutoff was not.

Table 4. Diagnostic performance of SAAG and ascitic fluid total protein against clinical assessment of portal hypertension (N=92)

Measure	SAAG ≥1.1 g/dL	Ascitic fluid total protein <2.5 g/dL	Difference or comparison	Test statistic	p value
Sensitivity, % (95% CI)	94.4 (86.4-98.5)	70.4 (58.7-80.1)	Difference: 24.0 percentage points	McNemar χ^2=13.47	<0.001
Specificity, % (95% CI)	71.4 (47.8-88.7)	66.7 (43.0-85.4)	Difference: 4.7 percentage points	McNemar χ^2=0.11	0.739

Positive predictive value, % (95% CI)	91.8 (83.0-96.1)	87.7 (76.8-93.9)			
Negative predictive value, % (95% CI)	78.9 (56.7-91.5)	40.0 (28.0-53.3)			
Overall accuracy, % (95% CI)	89.1 (81.1-94.5)	69.6 (59.5-78.1)	Difference: 19.6 percentage points	McNemar χ^2=9.14	0.003

Using clinical assessment of portal hypertension as the reference, SAAG ≥1.1 g/dL had higher sensitivity than ascitic fluid total protein <2.5 g/dL (94.4% vs 70.4%; p<0.001). Specificity was similar for the two measures (71.4% vs 66.7%; p=0.739). SAAG also showed higher positive predictive value (91.8% vs 87.7%), negative predictive value (78.9% vs 40.0%) and overall accuracy (89.1% vs 69.6%). The difference in overall accuracy was statistically significant (19.6 percentage points; p=0.003). In this sample, SAAG therefore showed better sensitivity and overall accuracy, while specificity was comparable between the measures.

DISCUSSION

Sociodemographic and clinical profile

In the supplied table, age, male sex, alcohol-related chronic liver disease and Child-Pugh class C did not differ significantly between participants classified with and without portal hypertension. The confidence intervals for these comparisons include the null value, so the results do not establish a difference between the groups. This may reflect limited precision, particularly in the smaller group without portal hypertension (n=21). Nakhale et al.(2016)[1] reported a varied adult ascites cohort in Maharashtra in which cirrhosis was the most frequent cause. Shahed et al.(2016)[2] studied patients with cirrhotic ascites and examined SAAG in relation to portal hypertensive changes; they reported only a modest relationship with the extent of those changes. Thus, the group differences in the present tables should be interpreted as biochemical differences by clinical classification, not evidence that demographics or SAAG measure portal hypertension severity.

Mean serum albumin was lower in the portal hypertension group, while mean SAAG was higher and mean ascitic fluid total protein was lower. The higher SAAG is consistent with its use in identifying ascites associated with portal hypertension. The lower ascitic fluid protein is also compatible with the protein-poor ascitic fluid often found in cirrhosis. However, these patterns are not universal: fluid protein may vary with the cause of ascites, concurrent disease and treatment. Fortune and Cardenas(2017)[6] reviewed the pathophysiology of cirrhotic ascites, while current guidance recommends interpreting SAAG and ascitic fluid protein alongside the broader clinical evaluation.[10-12]

SAAG distribution

High SAAG (≥1.1 g/dL) was present in 79.3% of the illustrative sample and was more frequent among those classified as having portal hypertension than among those without it (94.4% vs 28.6%). The mean SAAG was also higher in the portal hypertension group. These findings are consistent with the established interpretation of a SAAG ≥1.1 g/dL as suggestive of portal hypertension; the AASLD and Korean Association for the Study of the Liver guidance include SAAG in the diagnostic evaluation of ascites.[10,12]

Several comparative studies have similarly reported stronger diagnostic performance for SAAG than for the traditional protein-based transudate-exudate classification. Suman et al.(2017)[5] reported higher sensitivity and specificity for SAAG than for ascitic fluid total protein in identifying portal hypertension. Tiu et al.(2017)[4] and Gopi and Hanifah(2019)[7] also compared these measures in hospital-based ascites cohorts. Results have not been uniform, however: Subhani et al.(2022)[13] reported SAAG sensitivity of 85.5% and specificity of 60.6% in a contemporary adult cohort, while Cervantes Pérez et al.(2020)[9] reported a different balance of sensitivity and specificity in their Mexican cohort. These differences underline that test performance may vary with case mix and how portal hypertension is established.

Ascitic fluid total protein distribution

Mean ascitic fluid total protein was higher in the group without clinically assessed portal hypertension, but the proportion with low protein (<2.5 g/dL) did not differ significantly between groups. This suggests that the continuous protein measurement distinguished the groups more clearly than the single cutoff in these illustrative figures. Categorizing a continuous value can reduce information and, in a modest sample, may obscure a difference. AFTP also serves purposes beyond classifying portal hypertension: for example, high ascitic fluid protein in a patient with high SAAG may prompt consideration of a cardiac contribution. Angeleri et al.(2016)[3] described limitations of classifying ascites using protein alone, and Prabhu et al.(2019)[8] evaluated protein-related measures alongside SAAG in a cohort with multiple ascites etiologies.[10-12]

Diagnostic performance of SAAG and total protein

Against the clinical classification used in Table 4, SAAG ≥1.1 g/dL had higher sensitivity and overall accuracy than AFTP <2.5 g/dL, while the estimated specificities were similar. This direction is consistent with the comparative results reported

by Suman et al.(2017)[5] and Gopi and Hanifah(2019)[7], and with the AASLD recommendation to use SAAG in ascites evaluation.[7,12] Nevertheless, the sensitivity and specificity reported in other cohorts vary. Subhani et al.(2022)[13] found lower sensitivity and specificity than the illustrative values here, while Cervantes Pérez et al.(2020)[9] found that SAAG performance differed in their population. Vadlapudi et al.(2024)[14] reported high diagnostic accuracy in children, but their pediatric findings should not be directly applied to an adult CLD cohort.

Predictive values depend on the prevalence of portal hypertension in the sampled population; therefore, the illustrative PPV and NPV should not be generalized to other settings. The clinical reference classification also needs a clearly defined, consistently applied standard. If the test results themselves informed that classification, diagnostic performance could be overestimated. These considerations are especially important before interpreting the apparently higher accuracy of SAAG as definitive evidence of superiority.

CONCLUSION

In the supplied illustrative figures, participants classified as having portal hypertension had higher mean SAAG, lower mean ascitic fluid total protein and lower serum albumin than those without portal hypertension. SAAG ≥1.1 g/dL showed higher sensitivity and overall accuracy than AFTP <2.5 g/dL, while specificity was similar. These patterns support assessing SAAG alongside ascitic fluid total protein and the clinical evaluation of ascites. The conclusion must be confirmed using the actual study data and a clearly defined reference standard.

LIMITATIONS OF STUDY

- The tables contain illustrative values rather than verified observations; they cannot support a final scientific conclusion until recalculated from the original dataset.
- The sample was small, particularly the group without portal hypertension, resulting in wide confidence intervals and limited precision.
- The diagnostic estimates depend on the clinical reference classification. If this classification was not defined consistently or incorporated the index test results, misclassification or incorporation bias may have affected the estimates.
- The single-centre design, if applicable, may limit generalizability to other patient populations and clinical settings.
- Predictive values depend on the prevalence of portal hypertension in the study sample and may differ elsewhere.
- Dichotomizing SAAG and total protein at fixed cutoffs may lose information from their continuous measurements.
- Mixed causes of ascites, diuretic use, infection, albumin infusion and timing of paired serum and ascitic fluid sampling may affect measured values and diagnostic performance.
- Cross-sectional measurements do not establish the temporal relationship between biochemical markers and progression or complications of chronic liver disease.

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