

Comparison of the Diagnostic Accuracy of Serum Ascites Albumin Gradients (SAAG) With the Traditional Marker-Ascitic Fluid Total Protein

Aravindan R, KH Noorul Ameen*

Department of General Medicine, Sree Balaji Medical College and Hospital Affiliated to Bharath Institute of Higher Education and Research, Chennai, Tamil Nadu, India

ABSTRACT

To determine the sensitivity and specificity of serum ascites albumin gradient (SAAG) and that of ascitic fluid total protein, in identifying the etiology of ascites. To compare the diagnostic accuracy of serum ascites albumin. A total of 100 adult patients with ascites, admitted to the Department of General Medicine, Sree Balaji medical college & hospital, Chennai within approved period, whose etiological diagnosis had not been known previously were studied prospectively. The protocol was approved by the hospital's ethical committee and an informed consent was obtained from all patients. On entry, a detailed history and clinical examination were conducted. The 100 patients who satisfied the set criteria were included in the study. The sensitivity and specificity of SAAG in the differentiation of different types of ascites are 94% and 91% respectively. The accuracy of SAAG in the etiological diagnosis is 94%. The serum ascites albumin gradient (SAAG) is superior to ascitic fluid total protein (AFTP) in the differential diagnosis of ascites and it is statistically significant.

Key words: Failure, Ephrotic syndrome, peritoneal tuberculosis, Disseminated carcinomatosis, Pancreatitis, Myxedema.

HOW TO CITE THIS ARTICLE: Aravindan R, KH Noorul Ameen, Comparison of the Diagnostic Accuracy of Serum Ascites Albumin Gradients (SAAG) With the Traditional Marker-Ascitic Fluid Total Protein, J Res Med Dent Sci, 2021, 9 (5):271-275.

Corresponding author: KH Noorul Ameen

e-mail: noorulameen@bharathuniv.ac.in

Received: 24/04/2021

Accepted: 24/05/2021

INTRODUCTION

Ascites is defined as an accumulation of fluid within the peritoneal cavity. It complicates a variety of disorders [1] which include cirrhosis, decompensated heart failure, Ephrotic syndrome, peritoneal tuberculosis, disseminated carcinomatosis, pancreatitis, myxedema etc. In these conditions, ascites develops only because of the underlying illness. So, the evaluation of the patients with ascites requires, so that the cause of ascites could be established. A proper diagnosis is an important tool for the successful management of these patients. Diagnostic ascitic fluid aspiration is the most rapid and cost-effective test for identifying the basic disease process [2]. Previously the ascitic fluid total protein [AFTP] concentration was used to classify ascites as either exudative [AFTP \geq 2.5 g/dl] or transudative [AFTP $<$ 2.5g /dl] [3]. This

classification is unable to correctly identify the etiological factors responsible for the ascites. Hence this antiquated system of ascitic fluid is classified.

Giving false results and it offers only a little insight to the pathophysiology of the ascitic fluid formation. Further, these drawbacks led to the development of a new approach to classify ascites, based on the difference between the serum and ascitic fluid albumin concentration [Serum Ascites Albumin Gradient-SAAG]. A special technique was followed to prevent the leakage of fluid after the needle was withdrawn. This technique of needle insertion (Z tract) was accomplished by displacing the skin approximately 2 mm downward and then slowly inserting the paracentesis needle mounted on the syringe held the other hand. The paracentesis needle is a steel 22-gauge needle about 1.5 inch in length. The hand holding the syringes was used to stabilize the syringes and to retract the plunger simultaneously. The skin was released only after the needle had penetrated the peritoneum. When the needle was ultimately

removed, the skin resumed the original position and sealed the needle pathway. The needle was advanced slowly through the abdominal wall. Slow insertion helped to allow the bowel to move away from the needle thereby avoiding bowel puncture.

This newer concept classified ascites into two categories-High SAAG ascites with SAAG ≥ 1.1 g/dl in cases with portal hypertension and Low SAAG ascites with SAAG < 1.1 g/dl in cases with ascites, unrelated to portal hypertension [4-7]. The serum ascites albumin gradient [SAAG] has been proved in multiple studies to category. 1z e ascites better than either the ascitic fluid total protein or other parameters in ascitic fluid analysis. In view of the above, the present study is undertaken among the in patients, admitted with ascites in the medical wards of Sree Balaji medical college, to evaluate the value of SAAG in the etiological diagnosis of ascites and also to compare its sensitivity and diagnostic accuracy with that of ascitic fluid total protein [AFTP] [8-11].

MATERIALS AND METHODS

A total of 100 adult patients with ascites, admitted to the Department of General Medicine, Sree Balaji medical college & hospital, Chennai within approved period, whose etiological diagnosis had not been known previously were studied prospectively. The protocol was approved by the hospital's ethical committee and an informed consent was obtained from all patients. On entry, a detailed history and clinical examination were conducted. The 100 patients who satisfied the set criteria were included in the study. Paired ascitic fluid and serum samples were collected from them simultaneously and were examined for ascitic fluid albumin, ascitic fluid total protein and serum albumin with established methods of estimation.

Inclusion criteria

All patients with ascites due to any cause with normal coagulation profile.

Exclusion criteria

Ascitic patients with severe coagulopathy or disseminated intravascular coagulation (DIC).

Informed consent

Obtained.

Abdominal paracentesis

After obtaining informed consent from the patient and relatives, diagnostic abdominal paracentesis was done. The patients were asked to empty the bladder, prior to the procedure. The skin of the abdominal wall was disinfected with an iodine solution. The skin and subcutaneous tissue were infiltrated with a local anaesthetic. The needle was inserted into the left lower quadrant rather than the right lower quadrant because the caecum may be distended with gas from lactulose therapy. In the presence of a surgical scar, the needle was placed several centimeters from the scar. The ascitic fluid collected was sent for cell count in an EDTA bottle, biochemical analysis including total protein and albumin in a plain bottle- and for culture in a blood culture bottle. Simultaneously blood samples were collected from the patients and were sent for the estimation of serum albumin to the laboratory.

Calculation of SAAG

The serum ascites albumin gradient was calculated after measuring the serum and ascitic fluid albumin concentrations and simply subtracting the ascitic fluid value from the serum value. To increase the accuracy of SAAG, specimens of serum and ascitic fluid were obtained simultaneously.

Correction of SAAG

To correct the SAAG in the setting of a high serum globulin level the following formula was used. Corrected SAAG = Uncorrected SAAG $\times 0.16 \times (\text{Serum globulin} \pm 2.5)$. Serum hyperglobulinemia (Serum globulin > 5 g/dl) leads to a high ascitic fluid globulin concentration and can narrow the albumin gradient by contributing to the oncotic forces. A narrow gradient caused by high globulin levels occurs in one percent of ascitic fluid specimens.

Albumin estimation

BCG method and protein in biuret method.

RESULTS AND DISCUSSION

Majority of the patients belonged to the 31-50 years age class intervals (n=29, 53.70%) in 70 years age class intervals (n=21, 45.65%) in exudative group. Among the study patients, there was no statistically significant difference in relation to age distribution between transudative group (mean=50.19, SD=1 1.25) and exudative

group (mean=52.89, SD=12.88) with a p value of >0.05 as per unpaired t test. Therefore, we fail to reject the null hypothesis that there is no difference in age distribution between the study groups (Table 1 and Table 2) [12].

Majority of the patients belonged to male gender both in transudative group (n=51, 94.44%) and exudative group (n=43, 93.48%). Among the study patients, there was no statistically significant difference in relation to gender status between transudative group and exudative group with a p value of >0.05 as per Fisher's exact test. Therefore, we fail to reject the null hypothesis that there is no difference between gender status between the study groups (Figure 1) [13].

Majority of the patients belonged to the < 3.5 g/dl serum albumin levels class intervals (n=48, 88.89%) in transudative group and <3.5 g/dl serum albumin levels class intervals (n=40, 86.96%) in exudative group. Among the study

patients, there was no statistically significant difference in relation to serum albumin levels distribution between transudative group (mean=2.71, SD=0.62) and exudative group (mean=2.70, SD=0.66) with a p value of >0.05 as per unpaired t test. Therefore, we fail to reject the null hypothesis that there is no difference in serum albumin levels distribution between the study groups (Figure 2 and Figure 3) [14].

Majority of the patients belonged to the 1.01-2.00 g/dl ascitic fluid albumin levels class intervals (n=27, 50.00%) in transudative group and 1.01-2.00 g/dl ascitic fluid albumin levels class intervals (n=27, 58.70%) in exudative group. Among the study patients, there was no statistically significant difference in relation to ascitic fluid albumin levels distribution between transudative group (mean=1.27, SD=0.58) and exudative group (mean=1.35, SD=0.66) with a p value of >0.05 as per unpaired t test. Therefore, we fail to reject the null hypothesis

Table 1: Groups.

Groups	Definition of Subjects	Number
Transudative Ascites	Ascitic Fluid Total Protein < 2.5g/dl	54
Exudative Ascites	Ascitic Fluid Total Protein ≥ 2.5 g/dl	46

Table 2: Null hypothesis.

Null Hypothesis: H ₀	Transudative group equal in effect compared to Exudative group
Alternate Hypothesis: H ₁	Exudative group hazardous in effect compared to Transudative group

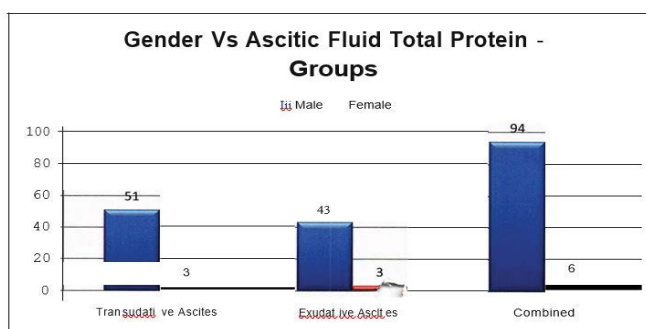


Figure 1: Gender Vs. ascitic fluid total protein.

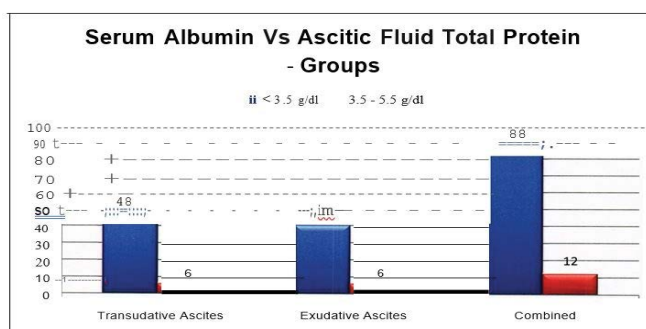


Figure 2: Serum albumin vs. ascitic fluid total protein.

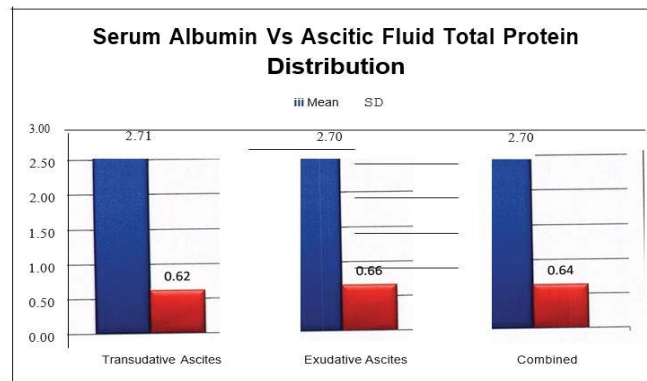


Figure 3: Serum Albumin Vs. ascitic fluid total protein distribution.

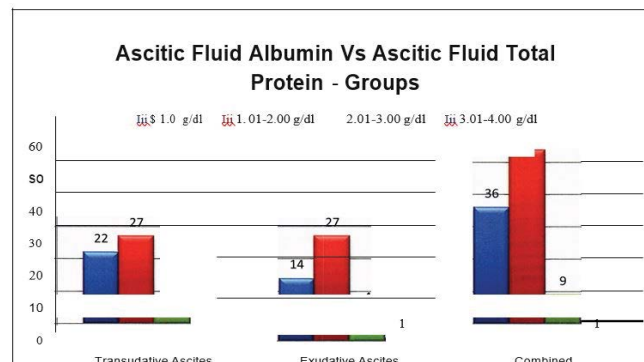


Figure 4: Ascitic fluid albumin vs. ascitic fluid total protein.

that there is no difference in ascitic fluid albumin levels distribution between the study groups. The percentage of the patients who had TB as diagnosis in transudative group was 0.00% (n=0) and exudative group was 4.35% (n=2). Among the study patients, there was no statistically significant difference in relation to TB as diagnosis between transudative group and exudative group with a p value of >0.05 as per fisher’s exact test. Therefore, we fail to reject the null hypothesis that there is no difference in TB as diagnosis between the study groups (Figure 4) [15-18].

CONCLUSION

The study "Serum ascites albumin gradient in the etiological diagnosis of ascites" conducted among the hundred in-patients with ascites, in the wards of the Department of General Medicine, at Sree Balaji Medical College Hospital has concluded that. The sensitivity and specificity of SAAG in the differentiation of different types of ascites are 94% and 91% respectively. The accuracy of SAAG in the etiological diagnosis is 94%. The serum ascites albumin gradient (SAAG) is superior to ascitic fluid total protein

(AFTP) in the differential diagnosis of ascites and it is statistically significant.

FUNDING

No funding sources.

ETHICAL APPROVAL

The study was approved by the Institutional Ethics Committee

CONFLICT OF INTEREST

The authors declare no conflict of interest.

ACKNOWLEDGMENTS

The encouragement and support from Bharath University, Chennai, is gratefully acknowledged. For provided the laboratory facilities to carry out the research work.

REFERENCES

1. Sleisenger, Fordtran. Gastrointestinal and Liver disease, Pathophysiology/ Diagnosis /Management. 8th Edn.
2. <https://emedicine.medscape.com/article/170907-overview#:~:text=The%20word%20ascites%20is%20of,phase%20of%20their%20menstrual%20cycle.>

3. https://www.palliativedrugs.com/download/08_0203_Guidelines%20for%20the%20Management%20of%20Malignant%20Ascites%20%20283%2029%5B1%5D.pdf
4. Rössle M, Ochs A, Gülberg V, et al. A comparison of paracentesis and trans jugular intrahepatic portosystemic shunting in patients with ascites. *New England J Med* 2000; 342:1701-1707.
5. Yachha SK, Khanna V. Ascites in childhood liver disease. *Indian J Pediatr* 2006; 73:819-824.
6. Levy M. Pathophysiology of ascites formation. *Kidney Liver Dis* 1982; 245-80.
7. Ginès P, Cárdenas A, Arroyo V, et al. Management of cirrhosis and ascites. *New England J Med* 2004; 350:1646-1654.
8. Peter L, Dadhich SK, Yachha SK. Clinical and laboratory differentiation of cirrhosis and extrahepatic portal venous obstruction in children. *J Gastroenterol Hepatol* 2003; 18:185-189.
9. Kuntz E, Kuntz HT. Oedema and ascites Hepatic biology, Principles and Practice. Springer-Verlag Berlin, Heidelberg 2002; 266-290.
10. Rimola A, García-Tsao G, Navasa M, et al. Diagnosis, treatment and prophylaxis of spontaneous bacterial peritonitis: A consensus document. *J Hepatol* 2000; 32:142-153.
11. McHutchison JG. Differential diagnosis of ascites. In *Seminars in liver disease* 1997; 17:191-202.
12. Mauer K, Manzione NC. Usefulness of serum-ascites albumin difference in separating transudative from exudative ascites. *Digestive Diseases Sci* 1988; 33:1208-1212.
13. Albillos A, Cuervas-Mons V, Millan I, et al. Ascitic fluid polymorphonuclear cell count and serum to ascites albumin gradient in the diagnosis of bacterial peritonitis. *Gastroenterol* 1990; 98:134-140.
14. Arroyo V, Ginés P. Arteriolar vasodilation, and the pathogenesis of the hyperdynamic circulation and renal sodium and water retention in cirrhosis. *Gastroenterol* 1992; 102:1077-1079.
15. Lipsky MS, Sternbach MR. Evaluation and initial management of patients with ascites. *Am Family Physician* 1996; 54:1327-1333.
16. Pinto PC, Amerian J, Reynolds TB. Large-volume paracentesis in nonedematous patients with tense ascites: Its effect on intravascular volume. *Hepatology* 1988; 8:207-210.
17. Valdivia RM, Llanos CA, Zapata SC, et al. The validity of the protein's concentrations in the ascitic liquid and serum for the differential diagnosis of the ascitis. *Rev Gastroenterol Peru* 2002; 22:279-286.
18. Harjai KJ, Kamble MS, Ashar VJ, et al. Portal venous pressure and the serum-ascites albumin concentration gradient. *Cleveland Clin J Med* 1995; 62:62-67.