

Serum albumin gradient and total protein in ascitic fluid of hospitalised patients with hepatic and non-hepatic ascites compared

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Abstract

Introduction: Analysis of ascitic fluid is useful for diagnosing ascites, one of the most prevalent issues a doctor faces in practice. Traditionally, ascites has been divided into two types exudative and transudative based on an estimate of the AFTP.

Methods: 50 patients admitted to the medical ward of the General Medicine, Kamineni Academy of Medical Sciences and Research Centre, Hyderabad, participated in a study comparing the serum ascitic fluid albumin gradient with the ascitic fluid total protein in hepatic and non-hepatic causes of ascites. The research period covered the years of June, 2021 to May 2022.

Results: The diagnostic accuracy of SAAG against AFTP was evaluated for determining the etiological causes of ascites in patients, and the results showed that SAAG -Serum Ascitic Fluid Albumin Gradient was superior to AFTP.

Conclusion: In determining the cause of ascites, the SAAG is preferred over the previously used AFTP classification system. The ascites is divided into two categories, high SAAG and low SAAG, according on the SAAG value.

Keywords: Serum albumin, ascitic fluid, hepatic, non-hepatic ascites

Introduction

Ascitic fluid analysis can aid in the diagnosis of ascites, one of the most common medical issues faced by doctors. The condition is commonly classified as either exudative or transudative ascites^[1, 2]. If you have exudative ascites, the total protein concentration in your ascitic fluid is higher than 2.5 g/dl, while if you have transudative ascites, it's lower than 2.5 g/dl. Its use in ordinary clinical practise is dangerous due to its unreliability in diagnosing the aetiology of ascitic fluid^[3, 4]. Cancer cells can be present in the ascitic fluid of people with normal cirrhosis, most notably in the cases of cardiac ascites, cirrhotic patients on prolonged diuretic therapy, and roughly one-third of patients with malignant ascites.

As a solution, we developed a SAAG-based ascites categorization system. There are two distinct forms of ascitic fluid, distinguished by their albumin levels in contrast to serum^[5, 6]. Portal hypertension was associated with a gradient of 1.1 g/dl, while ascites from other sources also showed a gradient of 1.1 g/dl. Among the SAAG's shortcomings is its inability to distinguish between ascites due to alcoholic cirrhosis and those due to malignancy or tuberculosis^[7, 8]. Studies comparing the diagnostic accuracy of SAAG and AFTP in identifying the pathophysiology underlying ascites^[9, 11] are needed to elucidate the etiological origin of ascites in a variety of Hepatic and Non Hepatic disorders. Both approaches suffer from significant flaws. The albumin concentration of the ascitic fluid is used to classify the severity of the ascites^[12]. Both SAAG and AFTP were evaluated for their diagnostic utility in determining the cause of ascites. Studying how SAAG stacks up against the established diagnostic marker AFTP is the focus of this investigation.

Methods

Serum ascitic fluid albumin gradient and total protein in hepatic and non-hepatic causes of ascites were studied in 50 patients in the medical ward under General Medicine, Kamineni Academy of Medical Sciences and Research Centre, Hyderabad. Amongst the June, 2021 to May 2022.

Inclusion criteria

- Severe coagulopathy patients.
- Patients with ascites and blunt abdominal injuries

Exclusion criteria

- Sufferers of severe coagulopathy.
- Blunt abdominal damage in ascitic individuals.
- Patients with ascites who have hepatic encephalopathy
- Patients with ascites receiving diuretic medication prior to ascites fluid analysis.

All patients in the study had a comprehensive history taken and underwent a detailed physical examination before they were even considered for enrollment. The origin of the ascites was determined after further diagnostic testing was performed on all 50 patients, including ultra sound imaging and CT scans. Next, using the established diagnosis, the diagnostic accuracies of AFTP and SAAG were determined and compared.

Results

Ascites was more common in males than in females, with males accounting for of all patients investigated and females for the remaining. This study's gender breakdown of ascitic patients.

Table 1: SAAG and AFTP concentrations as a scatter plot

| | High | Low | | P value |
|------|-------------|------------|----|----------------|
| High | 25 | 16 | 41 | 0.002 |
| Low | 05 | 04 | 09 | |
| | 30 | 20 | 50 | |

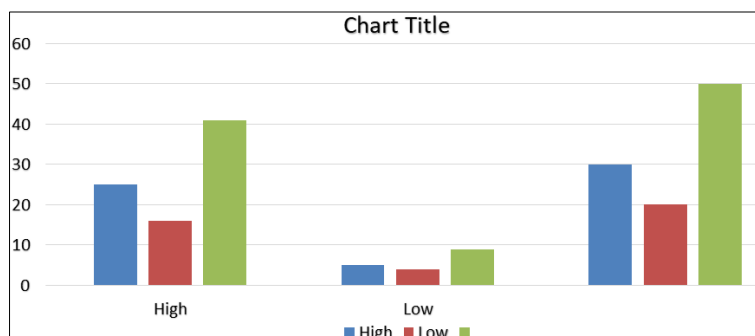


Fig 1: SAAG and AFTP concentrations as a scatter plot

Table 2: Among the investigated patients, AFTP

| AFTP | patients | % |
|-------------|-----------------|----------|
| <2.5 | 30 | 60.0 |
| ≥2.5 | 20 | 40.0 |
| Total | 50 | 100.0 |

As you can see from the table below, 60% of the patient population investigated had an AFTP

score of 1.0 or lower.

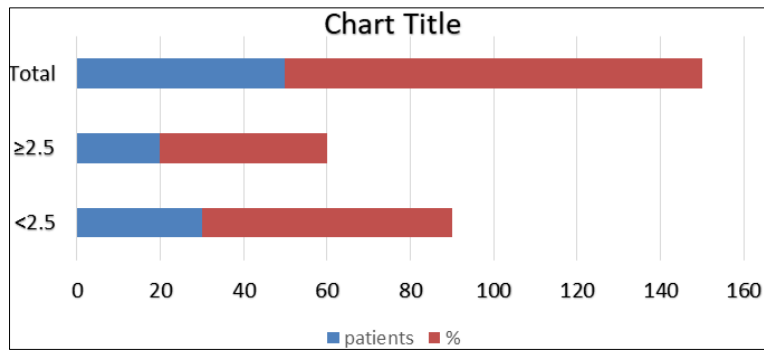


Fig 2: Among the investigated patients, AFP

Table 3: Among the patients studied, SAAG

| SAAG | patients | % |
|-------|----------|-------|
| <1.1 | 25 | 50.0 |
| ≥1.1 | 25 | 50.0 |
| Total | 50 | 100.0 |

As can be seen from the table, half of the patients in the study had a SAAG value.

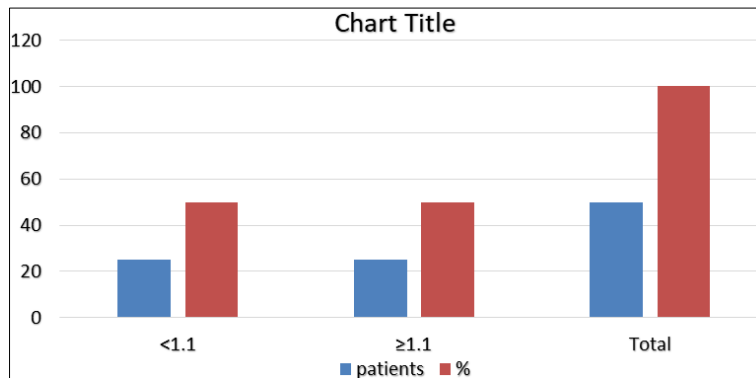


Fig 3: Among the patients studied, SAAG

Table 4: Etiology at the SAAG Level

| Sr. No. | Etiology | SAAG<1.1 | SAAG≥1.1 |
|---------|--------------------|----------|----------|
| 1. | Cirrhosis | 7 | 4 |
| 2. | CCF | 0 | 6 |
| 3. | TB ascites | 13 | 2 |
| 4. | Nephrotic syndrome | 4 | 2 |
| 5. | Carcinomatosis | 6 | 2 |
| 6. | Hypothyroidism | - | 0 |
| 7. | Liver metastasis | - | 2 |
| 8. | AN & HTN | 0 | - |

Based on the aetiology of the condition, ascites can be divided into two groups, low SAAG and high SAAG, as shown in the table below.

Table 5: AFP and SAAG specificity in ascites diagnosis

| Sr. No. | Etiology | SAAG | AFP |
|---------|------------|-------|-------|
| 1. | Cirrhosis | 85.58 | 66 |
| 2. | CCF | 91.12 | 39.56 |
| 3. | TB ascites | 93.54 | 68.98 |

| | | | |
|----|---------------------------|-------|-------|
| 4. | Nephrotic syndrome | 25 | 25 |
| 5. | Peritoneal Carcinomatosis | 84.74 | 82.21 |
| 6. | Miscellaneous | 100 | 100 |

Below is a table summarizing the diagnostic efficacy of SAAG and AFTP for ascites resulting from various sources.

Table 6: Mean, SD, and SE for AFTP and SAAG

| Sr. No. | Parameters | SAAG | AFTP |
|---------|------------------------|-------|--------|
| 1. | Mean | 1.65 | 2.314 |
| 2. | Standard deviation | 0.058 | 0.0782 |
| 3. | Standard error of mean | 0.512 | 0.8145 |

There is a table showing SAAG and AFTP averages, standard deviations, and mean errors.

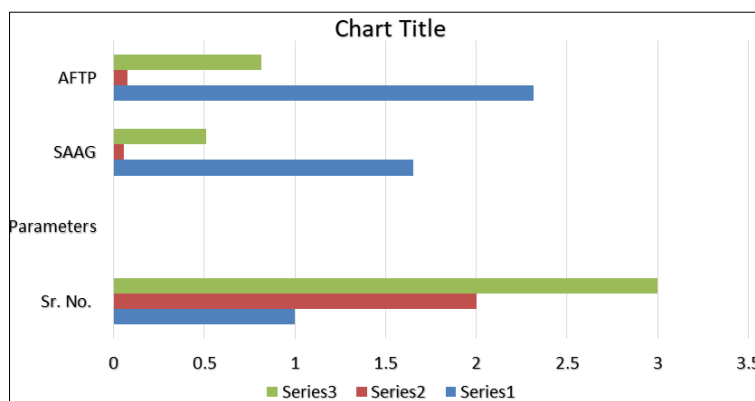


Fig 4: Mean, SD, and SE for AFTP and SAAG

Discussion

The diagnostic accuracy of SAAG -Serum Ascitic Fluid Albumin Gradient against AFTP for identifying the underlying aetiology of ascites in patients was compared. Goyal AK *et al.*, Beg *et al.*, Runyon *et al.*, and Das BB *et al.* all found essentially the same results in their studies [11-13].

A comparison of AFTP and SAAG revealed that the former was only sensitive while the latter was 87.01 percent sensitive. Younas *et al.*, research backs up these conclusions. The sensitivity of AFTP was 95%, while that of SAAG was only 71%, according to the research of Das *et al.* [14, 15]. Researchers have found that SAAG and AFTP had a specificity of 86.96% and 60.26%, respectively. The findings of the studies by Das *et al.* and younas *et al.* were consistent with one another. However, a study by Rana SV *et al* contradicts this conclusion by showing that AFTP is more specific than SAAG [16, 17]. Specificity was higher (88%) with AFTP than with SAAG (84%). One possible explanation for the increased specificity of AFTP compared to SAAG is that the cut off value of AFTP in the aforementioned study was 3, rather than the 2.5 used here. The PPV analysis showed that SAAG (95.71%) was more accurate than AFTP (89.03%). Research by Khan Fy *et al.* and Younas *et al.* [15-17] reach the same conclusion that SAAG is more accurate in estimating the test's positive prediction, therefore these results are consistent.

The NPV of SAAG was determined to be 66.67, whereas the NPV of AFTP was 85.45%. When comparing SAAG with AFTP, Das *et al.* found that the former had a negative predictive value of 85% and the latter of 92%. Younas *et al.* discovered that SAAG had a greater negative predictive value, hence these results contradict their conclusions. Only 5 of 30 people with Low SAAG in the study had an actual diagnosis of aftp, hence the negative predictive value of SAAG to aftp was rather low. Ten individuals had ascites due to portal hypertension,

whereas the underlying reason in the remaining 20 was a mystery. Cirrhosis is the true cause of ascites 90% of the time, with the heart being the incorrectly diagnosed reason 10% of the time. In 6 of the 8 false negative instances, the ascites characteristic of cirrhosis is not caused by alcohol. This result is consistent with the one found by Kajani *et al.*, who found that the SAAG value associated well with the portal venous pressure in cases of cirrhosis caused by alcohol-induced liver disease, but not in cases of cirrhosis produced by nonalcoholic causes of liver disease. Ascitic fluid accumulation is far more common in males than in females. Multiple investigations, including those by Jiang *et al.* and Khan *et al.* [18-21], indicated that males constituted the majority of their participants.

Almost 60% of the population suffers from ascites between the ages of 41 and 60. The mean age of the participants was calculated to be 49 years, 5 months, and 11.37 days [21, 22]. So, it's consistent with the results of previous research by Khan FY *Et al.* and Jiang *et al.* The results of this study agree with those of others, such as Younas *et al.* While some studies have linked ascites to tuberculosis and heart failure, others have linked it to carcinomatosis. According to research by Beg M. *et al.*, tuberculous ascites is the second most common cause of ascites. Nonetheless, the etiological classification only shows the tip of the iceberg found in the general population because it is based on hospital-based investigations. This research indicated that the average amount of albumin in the blood was 2.91 0.596 g/dL. The average serum albumin of people with ascites was 2.87 0.34, which agrees with the results of Santhosh Kumar *et al.* Serum albumin levels can be lowered by the following causes [23-25].

Cirrhosis was found to be the most common cause of ascites in people with high SAAG, followed by heart failure. Cirrhosis is the primary cause of ascites in high SAAG ascites, which is in agreement with the results found by Khan *et al.* According to Khan *et al.*, large hepatic metastases are the most common cause of high SAAG ascites, although cardiac failure was found to be the second most common cause. As an indicator of portal hypertension, the SAAG has been studied for its possible connection to the development of esophageal varices and other symptoms of the disease. Animal models can be used to learn more about how elevated SAAG levels may or may not serve as a signal for portal hypertension. Non-alcoholic cirrhosis is a different disorder for which more research is needed into SAAG's diagnostic value [25-27].

Conclusion

In determining the cause of ascites, the SAAG is preferred over the previously used AFTP classification system. The ascites is divided into two categories, high SAAG and low SAAG, according on the SAAG value. SAAG was found to have higher sensitivity and specificity than AFTP in detecting the pathophysiology of ascites, according to a study comparing the two. SAAG was found to be statistically significantly better than AFTP in terms of diagnostic accuracy.

Conflict of Interest

None

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References

1. Kajani MA, Yoo YK, Alexander JA, Gavalier JS, Stauber RE, Dindzans VJ, *et al.* Serum-ascites albumin gradients in nonalcoholic liver disease. *Dig Dis Sci.* 1990 Jan;35(1):33-7.
2. Alba D, Torres E, Vázquez JJ. [Sero-ascitic gradient of albumin: usefulness and diagnostic limitations]. *An Med Interna Madr Spain* 1984. 1995 Aug;12(8):404-7.
3. Hou W, Sanyal AJ. Ascites: diagnosis and management. *Med Clin North Am.* 2009

Jul;93(4):801-817.

4. Khan FY. Ascites in the state of Qatar: aetiology and diagnostic value of ascitic fluid analysis. *Singapore Med J.* 2007 May;48(5):434-9.
5. Moore CM, Van Thiel DH. Cirrhotic ascites review: Pathophysiology, diagnosis and management. *World J Hepatol.* 2013 May 27;5(5):251-63.
6. Healy JC, Reznick RH. The peritoneum, mesenteries and omenta: normal anatomy and pathological processes. *Eur Radiol.* 1998;8(6):886-900.
7. Standring Susan. *Gray's Anatomy: The Anatomical Basis of Clinical Practice.* 40th ed. Philadelphia: Churchill Livingstone/Elsevier; 2008.
8. Taylor AE. Capillary fluid filtration. Starling forces and lymph flow. *Circ Res.* 1981 Sep;49(3):557-75.
9. Schrier RW. Pathogenesis of Sodium and Water Retention in High-Output and Low-Output Cardiac Failure, Nephrotic Syndrome, Cirrhosis, and Pregnancy. *N Engl J Med.* 1988;319(17):1127-34.
10. Lieberman FL, Denison EK, Reynolds TB. The Relationship of Plasma Volume, Portal Hypertension, Ascites, and Renal Sodium Retention in Cirrhosis: The Overflow Theory of Ascites Formation. *Ann N Y Acad Sci.* 1970 Jul 1;170(1):202-12.
11. Lieberman FL, Reynolds TB. Plasma Volume in Cirrhosis of the Liver: Its Relation of Portal Hypertension, Ascites, and Renal Failure*. *J Clin Invest.* 1967 Aug 1;46(8):1297-308.
12. Schrier RW, Arroyo V, Bernardi M, Epstein M, Henriksen JH, Rodés J. Peripheral arterial vasodilation hypothesis: a proposal for the initiation of renal sodium and water retention in cirrhosis. *Hepatol Baltim Md.* 1988 Oct;8(5):1151-7.
13. Levy M, Wexler MJ. Renal sodium retention and ascites formation in dogs with experimental cirrhosis but without portal hypertension or increased splanchnic vascular capacity. *J Lab Clin Med.* 1978 Mar;91(3):520-36.
14. Levy M, Allotey JB. Temporal relationships between urinary salt retention and altered systemic hemodynamics in dogs with experimental cirrhosis. *J Lab Clin Med.* 1978 Oct;92(4):560-9.
15. Hoefs JC. Serum protein concentration and portal pressure determine the ascitic fluid protein concentration in patients with chronic liver disease. *J Lab Clin Med.* 1983 Aug;102(2):260-73.
16. Runyon BA, Montano AA, Akriviadis EA, Antillon MR, Irving MA, McHutchison JG. The serum-ascites albumin gradient is superior to the exudate-transudate concept in the differential diagnosis of ascites. *Ann Intern Med.* 1992 Aug 1;117(3):215-20.
17. Hoefs JC. Globulin correction of the albumin gradient: correlation with measured serum to ascites colloid osmotic pressure gradients. *Hepatol Baltim Md.* 1992 Aug;16(2):396-403.
18. Al-Knawy BA. Etiology of ascites and the diagnostic value of serum-ascites albumin gradient in non-alcohol liver disease. *Ann Saudi Med.* 1997 Jan;17(1):26-8.
19. Younas M, Sattar A, Hashim R, Ijaz A, Dilawar M, Manzoor SM, *et al.* Role of serum-ascites albumin gradient in differential diagnosis of ascites. *J Ayub Med Coll Abbottabad JAMC.* 2012 Dec;24(3-4):97-9.
20. Valdivia RM, Llanos CA, Zapata SC, Muñoz ON. [The validity of the proteins concentrations in the ascitic liquid and serum for the differential diagnosis of the ascitis]. *Rev Gastroenterol Perú Órgano of Soc Gastroenterol Perú.* 2002 Dec;22(4):279-86.
21. Shaikh MA, Khan J, Almani S, Dur-e -Yakta null, Shaikh D. Frequency of causes of ascites in patients admitted at medical unit of a tertiary medical care facility. *J Ayub Med Coll Abbottabad JAMC.* 2010 Jun;22(2):88-92.
22. Santhosh kumar, Iqbal Ahmed Memon, Mohammad Kaleem, Suhail A. Alamani. Prediction of Esophageal Varices in Cirrhotic Patients with Serum - Ascites Albumin Gradient. *JLUMHS.* 2013;12(03):167-71.
23. Greenblatt DJ. Reduced serum albumin concentration in the elderly: a report from the Boston Collaborative Drug Surveillance Program. *J Am Geriatr Soc.* 1979 Jan;27(1):20-2.
24. Gatta A, Verardo A, Bolognesi M. Hypoalbuminemia. *Intern Emerg Med.* 2012 Oct;7

Suppl 3:S193-199.

25. Maskey R, Karki P, Ahmed SV, Manandhar DN. Clinical profile of patients with cirrhosis of liver in a tertiary care hospital, Dharan, Nepal. *Nepal Med Coll J NMCJ*. 2011 Jun;13(2):115-8.
26. Perz JF, Armstrong GL, Farrington LA, Hutin YJF, Bell BP. The contributions of hepatitis B virus and hepatitis C virus infections to cirrhosis and primary liver cancer worldwide. *J Hepatol*. 2006 Oct;45(4):529-38.
27. Jauhar P, Watson AS. Severity of alcohol dependence in the East End of Glasgow. *Alcohol Alcohol Oxf Oxf*. 1995 Jan;30(1):67-70.