



E-ISSN: 2706-9575
P-ISSN: 2706-9567
IJARM 2021; 3(1): 292-298
Received: 06-06-2021
Accepted: 25-06-2021

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A study on serum ascites albumin gradient in the etiological diagnosis of ascites

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DOI: <https://doi.org/10.22271/27069567.2021.v3.i1e.274>

Abstract

Aim: To study the “Serum ascites albumin gradient in the etiological diagnosis of ascites”

Methodology: A total of fifty adult patients with ascites, admitted to the Department of General Medicine, NRI Medical College, Guntur within one year 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.

Results: In the present study, considering the etiology of ascites in the population studied, cirrhosis of the liver (28 cases) ranked first followed by decompensated heart failure (8 cases), tuberculous peritonitis (5 cases) and malignant ascites (4 cases). The other causes were nephrotic syndrome (2 cases), splenic abscess (1 case), pancreatitis (1 case), spontaneous bacterial peritonitis (1 case) and hypothyroidism (1 case). The sensitivity and specificity of ascitic fluid total protein (AFTP) in the differentiation of different types of ascites are 83% and 63% respectively. The sensitivity and specificity of serum ascites albumin gradient (SAAG) in the differentiation of different types of ascites are 95% and 92% respectively. The diagnostic accuracy of ascitic fluid total protein (AFTP) in the etiological diagnosis of ascites is 68%. The diagnostic accuracy of serum ascites albumin gradient (SAAG) in the etiological diagnosis of ascites 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.

Conclusion: Thus Serum ascites albumin gradient (SAAG) is the single best test against ascitic fluid total protein (AFTP), in the differential diagnosis of ascites. The terms exudative and transudative can be replaced by high SAAG and low SAAG ascites.

Keywords: serum ascites albumin gradient (SAAG), ascitic fluid total protein (AFTP), tuberculous peritonitis, malignant ascites

Introduction

Ascites is the pathological accumulation of fluid within the peritoneal cavity. It is one of the most common amongst the various clinical conditions, confronting not only a physician, but a surgeon and a gynaecologist too. It complicates a variety of disorders^[1] which include cirrhosis, decompensated heart failure, nephrotic syndrome, peritoneal tuberculosis, disseminated carcinomatosis, pancreatitis, myxedema etc. In these conditions, ascites develops only as a consequence of the underlying illness. So the evaluation of the patients with ascites requires, that the cause of ascites be established. A proper diagnosis is a prerequisite for the successful management of these patients^[2-4].

Diagnostic ascitic fluid aspiration is the most rapid and cost effective test for identifying the basic disease process. Before the 1980s, the ascitic fluid total protein [AFTP] concentration was used to classify ascites as either exudative [AFTP \geq 2.5g/dl] or transudative [AFTP $<$ 2.5g/dl]^[3]. This classification is unable to correctly identify the etiological factors responsible for its causation. Hence this antiquated system of ascitic fluid classification is problematic and it offers only a little insight to the pathophysiology of the ascitic fluid formation^[5].

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]. This newer concept classified ascites into two categories – High SAAG ascites with SAAG \geq 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. The serum ascites albumin gradient has been proved in multiple studies to categorize 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 inpatients, admitted with ascites in the medical wards of King George hospital, Andhra Medical College, Visakhapatnam 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].

Aim & Objectives

1. To differentiate ascites on the basis of serum ascites albumin gradient into high SAAG ascites > 1.1 g/dl and low SAAG ascites < 1.1 g/dl.
2. To determine the sensitivity and specificity of serum ascites albumin gradient and that of ascitic fluid total protein, in identifying the etiology of ascites.
3. To compare the diagnostic accuracy of serum ascites albumin gradient with the traditional marker – ascitic fluid total protein.

Materials and Methods

The study of 'Serum Ascites Albumin Gradient in the etiological diagnosis of Ascites' was carried out in the Department of General Medicine, NRI Medical College, Guntur.

Setting: Medical Wards

Study Design: Single centre observational prospective hospital based study

Period of study: June 2017 to June 2020

Ethical approval: Obtained

Methodology

A total of fifty adult patients with ascites, admitted to the Department of General Medicine, NRI Medical College, Guntur within one year 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 fifty 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 – Bromocresol green and Biuret methods as described by Varley *et al.*

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).

Abdominal paracentesis^[3]

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. 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 cms downward and then slowly inserting the paracentesis needle mounted on the syringe held in 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.

Site of needle insertion

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 $\times 0.16 \times (\text{Serum globulin} + 2.5)$

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.

All the 50 patients further underwent ultrasonogram abdomen which revealed the ultimate diagnosis for confirmation and comparing the diagnostic accuracies of SAAG and ascitic fluid protein.

Ultrasonography of the liver and portal venous system, which is a non-invasive imaging modality, helped to establish the diagnosis of portal hypertension. USG diagnosis of portal hypertension was based on demonstration of

- a) Dilated collaterals around the gastroesophageal junction and splenic hilum,
- b) Splenomegaly and dilated portal vein > 14 mm in diameter and splenic vein > 12 mm.

It also helped in establishing the etiology of portal hypertension by giving information about

- Hepatic architecture (altered echopattern with nodularity indicates cirrhosis, normal echopattern in extrahepatic portal vein obstruction and non-cirrhotic portal fibrosis).
- Patency of the portal and splenic veins (portal vein thrombosis and portal cavernoma diagnostic of EHPVO) and
- Patency of hepatic veins and IVC. (thrombosis or Budd chiari syndrome)

Observation and Results

The results of the diagnostic ascitic fluid aspiration and the ascitic fluid analysis in all the fifty selected patients are being interpreted here. The ascitic fluid specimens of forty eight patients were straw coloured, whereas two specimens were hemorrhagic. After complete workup, one turned out to be a case of chronic calcific pancreatitis and the other was a case of peritoneal carcinomatosis with primary in the large intestine.

Table 1: Sex Distribution

Males	26
Females	24

Table 1: Describes the distribution of ascites among the males and the females in the selected study group. The distribution of ascites among the males and the females was more or less equal with 26 males and 24 females with a sex ratio of 1.08.

Table 2: Age wise distribution

Age(years)	Total
11- 20	2
21- 30	3
31- 40	10
41- 50	9
51- 60	8
61- 70	14
71- 80	4

Table 2: Outlines the distribution of ascites among the different age groups. It is quite interesting to note that the incidence of ascites increases as the age advances and the total number of cases peaks around 60 -70. The majority of the cases i.e. 45/50 (90%) are aged above 30 years, with a minority i.e. 5/50 (10%) below 30 years of age.

Table 3: Comparison of age wise and sex wise distribution

Age (years)	Total	Female	Male
11- 20	2	2	0
21- 30	3	3	0
31- 40	10	6	4
41- 50	9	3	6
51- 60	8	4	4
61- 70	14	6	8
71- 80	4	0	4

Table 3: Gives an outlook of the distribution of the age wise distribution of ascites among males and females. The minor group (10%) who presented below the age of thirty years were all females with no males. As the age advanced beyond 30 years of age, the incidence increased in both males and females with a maximum of males compared to

females. Thus the incidence of ascites was more among females in the younger age, whereas the incidence though increased both in males and females as the age advanced, it was higher in males (i.e. 26/50 = 52%) when compared to females (i.e. 19/50 = 38%).

Table 4: Clinical profile

Etiology	Total number
Cirrhosis	28
Decompensated heart failure	8
Nephrotic syndrome	2
Liver metastasis	1
Peritoneal carcinomatosis	3
Tuberculous ascites	5
Pancreatitis	1
Splenic abscess	1
Hypothyroidism	1

Table 4: Interprets the etiological distribution of ascites among the study group. Considering the etiology of ascites in the population studied, cirrhosis of the liver (28 cases) ranked first followed by decompensated heart failure (8 cases), tuberculous peritonitis (5 cases) and malignant ascites (4 cases). The other causes were nephrotic syndrome (2 cases), splenic abscess (1 case), pancreatitis (1 case), spontaneous bacterial peritonitis (1 case) and hypothyroidism (1 case).

The cases who presented with ascites due to decompensated heart failure were evaluated. Among the 8 cases, there were 2 cases of valvular heart disease, 2 cases of cor pulmonale, 2 cases of coronary artery disease with ischemic cardiomyopathy, 1 case of dilated cardiomyopathy and another case of restrictive cardiomyopathy.

There were 4 cases of malignant ascites among which 3 were cases of peritoneal carcinomatosis and the other one was a primary colonic carcinoma with secondaries in liver. The primary tumours of peritoneal carcinomatosis were in ovary, stomach and large intestine.

Table 5: Comparison of sex distribution and etiology of ascites

Etiology	Males	Females	Total
Cirrhosis	19	9	28
CCF	2	6	8
Nephrotic syndrome	1	1	2
Liver metastasis	0	1	1
Peritoneal carcinomatosis	1	2	3
TB Peritonitis	1	4	5
Splenic abscess	1	0	1
Pancreatitis	0	1	1
Hypothyroidism	0	1	1

Table 5: Gives an outline of the etiological distribution of ascites among both males and females. The most common cause of ascites among both the males and females was cirrhosis. Though cirrhosis was the common cause in both the genders, it constituted the major cause in males (i.e. 19/26=73%). However in females, the noncirrhotic cases taken altogether, constituted the major group (i.e. 15/24=67%), whereas the cirrhotics constituted only the reminder (i.e. 9/24=33%).

Among the non-cirrhotic causes, decompensated heart failure topped the list with females about 75% (6/8) compared to males, followed by tuberculous peritonitis with females amounting 80% (4/5) and malignant ascites with 50% females (2/4).

Table 6: Comparison of etiology of ascites and age wise distribution in males

Etiology	11-20	21-30	31-40	41-50	51-60	61-70	71-80	Total
Cirrhosis	0	0	3	6	3	6	1	19
CCF	0	0	0	1	0	1	0	2
Nephrotic syndrome	0	0	1	0	0	0	0	1
Liver metastasis	0	0	1	0	0	0	0	1
Peritoneal Ca	0	0	0	0	0	0	1	1
TB Peritonitis	0	0	0	0	1	0	0	1
Splenic abscess	0	0	0	0	0	0	1	1

Table 6: Explains the distribution of ascites among different age groups in males. In males, cirrhosis is the leading cause peaking in the age group (61-70) followed by the age group (41-50). All the other causes of ascites like decompensated

heart failure, hepatocellular carcinoma, peritoneal carcinomatosis, tuberculous peritonitis and splenic abscess presented beyond 40 years of age. Something interesting here is that no men presented below 20 years of age.

Table 7: Comparison of etiology of ascites and age wise distribution in females

Etiology	11-20	21-30	31-40	41-50	51-60	61-70	71-80	Total
Cirrhosis	0	0	3	0	2	4	0	9
CCF	0	1	1	1	1	2	0	6
Nephrotic syndrome	0	0	1	0	0	0	0	1
Peritoneal Ca	0	0	0	1	0	0	1	2
TB Peritonitis	2	1	0	0	1	0	0	4
Pancreatitis	0	1	0	0	0	0	0	1
Hypothyroidism	0	0	0	0	0	1	0	1

Table 7: Explains the distribution of ascites among different age groups in females. About 42% women (10/24) presented below 40 years of age and the remaining 58% women presented above 40 years of age. Tuberculous peritonitis was common amongst the younger individuals when compared to the old, whereas cirrhosis was common among the older individuals. Decompensated heart failure was equally prevalent in all age groups. Congestive cardiac failure and tuberculous peritonitis were common amongst females (10/24=42%) compared to males (3/26=12%).

Table 8: Distribution of ascites on the basis of SAAG

Etiology	SAAG \geq 1.1	SAAG<1.1
Cirrhosis	26	2
CCF	8	0
Nephrotic syndrome	0	2
Liver metastasis	1	0
Peritoneal carcinomatosis	0	3
TB ascites	1	4
Pancreatitis	0	1
Splenic abscess	0	1
Hypothyroidism	1	0

Table 8: Groups the individuals with ascites in the study population into high SAAG group and low SAAG group with a cut off value of 1.1. About 74% of the people were in high SAAG group and the left out 26% were in low SAAG group. Cirrhotic patients (26 cases), 8 cases of decompensated heart failure, a case each of liver metastasis, TB ascites and hypothyroidism had high SAAG ascites.

4 cases of TB ascites, 3 cases of peritoneal carcinomatosis, 2 cases of nephrotic syndrome, 2 cases of cirrhosis, 1 case each of pancreatitis and splenic abscess had low SAAG ascites.

Table 9: Distribution of ascites on the basis of ascitic fluid total protein

Etiology	AFTP \geq 2.5	AFTP<2.5
Cirrhosis	9	19
CCF	4	4
Nephrotic syndrome	1	1
Liver metastasis	0	1
Peritoneal carcinomatosis	3	0
TB peritonitis	4	1
Pancreatitis	1	0
Splenic abscess	1	0
Hypothyroidism	1	0

Table 9: Classifies the ascites of the patients into two groups as exudative and transudative with the cut off of ascitic fluid total protein as \geq 2.5 and $<$ 2.5 respectively. 48% of cases presented as exudative and 52% of cases had transudative ascites.

After evaluating the patient with imaging studies and coming to the conclusion of the etiology, the patients were further classified into high SAAG or low SAAG, depending on the pathophysiology of ascites, whether related or unrelated to portal hypertension and exudative ascites or transudative ascites which are depicted in tables 10 & 11.

Table 10: Classification of ascites depending on the Pathophysiology

High SAAG	No.	Low SAAG	No.
Cirrhosis	28	Splenic abscess	1
CCF	8	Pancreatitis	1
Liver metastasis	1	Peritoneal carcinomatosis	3
Hypothyroidism	1	TB peritonitis	5
		Nephrotic syndrome	2

Table 10: groups the ascites into high SAAG and low SAAG depending on the pathophysiology. Cirrhosis, decompensated heart failure, liver metastasis and hypothyroidism were grouped under high SAAG ascites. Peritoneal carcinomatosis, tuberculous ascites, nephrotic syndrome, splenic abscess and pancreatitis were grouped under low SAAG ascites.

Table 11: Classification of ascites as exudative or transudative

Exudate	No.	Transudate	No.
Peritoneal carcinomatosis	3	Cirrhosis	28
Liver metastasis	1	Nephrotic syndrome	2
TB Peritonitis	5	CCF	8
Splenic abscess	1		
Pancreatitis	1		
Hypothyroidism	1		

Table 11 groups the ascites as transudative and exudative after the etiology being identified. Cirrhosis, decompensated heart failure and nephrotic syndrome were grouped under transudative ascites whereas malignant ascites, tuberculous ascites, splenic abscess, hypothyroidism and pancreatitis were grouped under exudative ascites.

Table 12: Comparison of SAAG and Portal hypertension

Pathophysiology	High SAAG	Low SAAG
Portal HT	36 (True positive)	2 (False negative)
Non Portal HT	1 (False positive)	11 (True negative)

On comparing the high SAAG values and the presence of portal hypertension, 36 patients with high SAAG had a pathophysiology related to portal hypertension i.e true positive (a), whereas only one patient with high SAAG did not have portal hypertension i.e false positive (b). On the other hand, 11 patients with low SAAG did not have portal hypertension i.e true negative (d) and 2 patients with low SAAG had portal hypertension as its pathophysiology i.e false negative (c). These results are depicted in the Table 12.

Table 13: Comparison of AFTP and exudative/transudative

	AFTP >2.5	AFTP <2.5
Exudate	10 (true positive)	2 (false negative)
Transudate	14 (false positive)	24 (true negative)

Comparing the values of ascitic fluid total protein and exudative/ transudative ascites, 12 patients with AFTP > 2.5 had exudative ascites i.e true positive (a), whereas 16 patients with AFTP > 2.5 had transudative ascites i.e false positive (b). However 20 patients with AFTP <2.5 had transudative ascites i.e true negative (d) and 2 patients with AFTP < 2.5 had exudative ascites i.e false negative (c). These results are depicted in Table 13.

1. Sensitivity is calculated by dividing true positive by the sum of true positive and false negative. i.e. $(a / a+c) \times 100$.⁽¹⁴⁾
2. Specificity is calculated by dividing true negative by the sum of true negative and false positive. i.e. $(d/ d+b) \times 100$.
3. Positive predictive value is calculated by dividing true positive by the sum of true positive and false positive i.e. $(a/a+b) \times 100$

4. Negative predictive value is calculated by dividing true negative by the sum of true negative and false negative i.e. $(d/d+c) \times 100$
5. Diagnostic accuracy is calculating by dividing the sum of true positive and true negative by the total number of cases and multiplying by 100.

All the parameters were calculated separately for SAAG and ascitic fluid total protein using the above formulae.

Ascitic fluid total protein (AFTP)

Sensitivity of AFTP = $10/(10+2) \times 100 = 83\%$

Specificity of AFTP = $24/(24+14) \times 100 = 63\%$

Positive predictive value of AFTP = $10/(10+14) \times 100 = 42\%$

Negative predictive value of AFTP = $24/(24+2) \times 100 = 92\%$

Serum ascites albumin gradient (SAAG)

Sensitivity of SAAG = $36/(36+2) \times 100 = 95\%$

Specificity of SAAG = $11/(11+1) \times 100 = 92\%$

Positive predictive value of SAAG = $36/(36+1) \times 100 = 97\%$

Negative predictive value of SAAG = $11/(11+2) \times 100 = 85\%$

Diagnostic accuracy

Diagnostic accuracy = $(\text{True positive} + \text{True negative}) / \text{Total number of cases}$

Diagnostic accuracy of AFTP = $(10+24)/50 \times 100 = 68\%$

Diagnostic accuracy of SAAG = $(36+11)/50 \times 100 = 94\%$

Table 14: Comparison of AFTP & SAAG

Parameters	AFTP	SAAG
Sensitivity	83%	95%
Specificity	63%	92%
Positive predictive value	42%	97%
Negative predictive value	92%	85%
Diagnostic accuracy	68%	94%

The five variables calculated for both AFTP and SAAG are depicted in table 14. At a glance from this table, one could understand easily and with no doubt that SAAG is better than AFTP in determining the etiology of ascites, which is the aim of the present study.

Discussion

The results of the study "SAAG in the etiological diagnosis of ascites" conducted in the medical wards of NRI Medical College, Guntur has yielded an etiological profile of the entire study group as follows:

1. Liver cirrhosis – 56%
2. Decompensated heart failure – 16%
3. Tuberculous peritonitis – 10%
4. Malignant ascites – 8% including 3 cases of peritoneal carcinomatosis and 1 case of malignant deposit in liver
5. Nephrotic syndrome – 4%
6. Splenic abscess – 2%
7. Pancreatitis – 2%
8. Hypothyroidism – 2%

In a study conducted by Writte *et al.* [6], found that, 44% liver cirrhosis, 23% TB peritonitis, 22% Malignant ascites, 6% heart diseases and 5% nephrotic syndromes. Cirrhosis, 10.6% peritoneal tuberculosis, 9.1% malignant ascites, 7.6% decompensated cardiac failure and 3% nephrotic syndrome.

In contrast to the two studies discussed above, decompensated heart failure ranked the second instead of tuberculous peritonitis, in the present study group. On the other hand the major causes of ascites are liver cirrhosis and malignant ascites in the western population, whereas tuberculous peritonitis leads the list in the Asians and Blacks [6].

The clinical profile of the low SAAG ascites in the present study group was 38% of tuberculous peritonitis, 25% of peritoneal carcinomatosis, 15% of nephrotic syndrome and other causes like pancreatitis and splenic abscess which accounted for 7% each. The present study is comparable to a study [7] conducted by Rocco VK., among 148 patients over a 7 years period at the Gastrointestinal and liver diseases research centre, Gullan University of medical sciences, Gullan Province, Iran, which concluded that Tuberculous peritonitis should be considered in all patients with low gradient ascites in the developing countries. This is in contrast to the study conducted by Runyon *et al.* [8] which stated malignant ascites as the commonest cause of low SAAG ascites in the developed countries.

Myxedema ascites is a rare entity and hypothyroidism as a cause of ascites accounts for less than one percent. Similarly the sixty eight years old hypothyroid female in our study, presented with high protein and high SAAG ascites [9].

In the present study, the patients with malignant ascites presented as two groups – one with high SAAG ascites comprising 1 case of secondaries liver comprising about 25%. The other group presented with low SAAG ascites consisting of 3 cases of peritoneal carcinomatosis comprising about 75%. The diagnostic accuracy of SAAG in malignant ascites is 100% in our present study. The results of the application of the two tests of interest, i.e

1. Serum ascites albumin gradient (SAAG) &
2. Ascitic fluid total protein (AFTP)

In the categorization of etiology of ascites in the study population are expressed in terms of sensitivity, specificity, positive predictive value, negative predictive value and diagnostic accuracy calculated by the appropriate formulae as already discussed [11].

The sensitivity, specificity, positive predictive value, negative predictive value and diagnostic accuracy of SAAG are 95%, 92%, 97%, 85% and 94% respectively as compared to 83%, 63%, 42%, 92% and 68% respectively with ascitic fluid total protein (Table:14). These results clearly demonstrate that SAAG offers an excellent discrimination of the causes of ascites. Similar observations have been reported by other studies too [12, 13].

In the study [10] conducted by Taki *et al.*, two other parameters i.e. ascitic fluid lactic dehydrogenase and ascitic to serum ratio of total protein, in addition to SAAG and AFTP were compared. Among all the four, SAAG had the highest positive and negative predictive values (80% & 98%) against that of ascitic fluid total protein (68% & 96%).

All these studies were based on the initial study conducted by Watson *et al.* [14], among 901 patients in the University of Iowa, Iowa city in the year 1992. The diagnostic accuracy

of SAAG and ascitic fluid total protein were 96.7% and 55.6% respectively.

In another study Sanai *et al.* [15], Hippocratical Hospital, Greece the diagnostic accuracy of SAAG was found to be 98% when compared to 52 – 80% in the four other diagnostic markers compared. (Ascitic fluid total protein, Ascites/Serum total protein ratio, Ascitic lactate dehydrogenase concentration and Ascites/Serum lactate dehydrogenase ratio).

The diagnostic accuracy of SAAG and AFTP were calculated separately for each cause in the present study and they were compared statistically using student's t test to determine the 95% confidence interval. A p value < 0.05 is considered significant.

The calculated t value is 2.72. ($p < 0.05$). For 10 degrees of freedom the table value is 2.23 at 5% level of significance, which is lesser than 2.72. So the data is significant at 5% level of significance and implies that SAAG is a better diagnostic measure than ascitic fluid total protein.

Thus Serum ascites albumin gradient (SAAG) is the single best test against ascitic fluid total protein (AFTP), in the differential diagnosis of ascites. The terms exudative and transudative can be replaced by high SAAG and low SAAG ascites. This result is similar to the results of all the studies conducted [15, 16].

Conclusion

The diagnostic accuracy of AFA was superior to that of SAAG for discriminating between PH-related and non-PH-related ascites, so it could be used in clinical practice in isolation or together with SAAG to achieve a more accurate diagnostic approach. In addition, we established. Prospective and large-scale evaluation is required in this and other settings to identify the usefulness of SAAG, identify its optimal cutoff points, and compare its diagnostic accuracy with that of other noninvasive markers. Hence, further studies are aimed at concentrating the correlation between the value of SAAG and the complications of portal hypertension like esophageal varices leading on to upper gastrointestinal bleeding. In case, these studies could yield certain satisfactory and significant results, the SAAG value can be used as a screening tool to predict upper GI bleed. Moreover, the patients with portal hypertension could be put on prophylactic therapy considering the significant SAAG value, so that the mortality and morbidity in these patients can be reduced. In a nutshell, further studies are expected to extend the use of SAAG, beyond the differential diagnosis of ascites, such as complications and management of ascites.

Acknowledgment

The author is thankful to Department of General Medicine for providing all the facilities to carry out this work.

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