



Estimation Of Serum Ascitic Albumin Gradient (SAAG) and Serum Ascitic Cholesterol Gradient (SACG) in different causes of ascites

Investigation of Self-Effectiveness and Self-Efficacy Levels of Nursing Students

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ABSTRACT

Background: The aim of present study was to differentiate the portal hypertension as well as malignant causes of ascites by estimating Serum Ascites Albumin Gradient (SAAG) and Serum Ascites Cholesterol Gradient(SACG) respectively.

Material and method: A total of 130 patients having ascites were included in the study. Serum and Ascitic albumin and cholesterol was measured. SAAG and SACG were calculated by subtracting ascitic values from the respective serum values.

Results: There was significant difference in SAAG in portal hypertension cases of ascites as compared to non-portal hypertension cases of ascites. The efficacy of SAAG in the present study to classify

portal hypertension and non-portal hypertension etiology was 98.4%. In malignant cases the SACG was found significantly lower than non-malignant cases.

SACG was able to accurately differentiate the malignant and non-malignant cases in 99.2% of cases at cut off value of 53mg/dl. **Conclusion:** In view of the good diagnostic efficacy, easy availability and cost-effectiveness, serum ascitic albumin and cholesterol gradient can be an excellent parameter for the diagnosis of portal hypertension and malignant ascites respectively.

Keywords: Ascites, Malignant, Albumin, Cholesterol

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INTRODUCTION

Ascites is defined as accumulation of fluid within the peritoneal cavity. The etiological spectrum of ascites is vast and practically includes pathology of all the systems. Careful history taking and clinical examination can provide clue to the etiology of ascites. But paracentesis remains the main stay of the investigation of ascites.

In most cases ascites appears as a part of a well-recognized illness i.e. cirrhosis, tuberculosis, congestive heart failure, nephrosis or disseminated carcinomatosis. Few patients have more than one cause of ascites formation. 84% of cases of ascites are due to portal hypertension; mainly as a result of cirrhosis.¹ Other subset of cause includes pathology of peritoneum, which are not related to portal hypertension. This classification is important because the mode of evaluation and management is different for these two groups.

In past portal hypertension ascites was distinguished from the non-portal hypertension causes by determining whether the fluid is transudate or exudate. This concept assumed that in portal hypertension, protein poor ascitic fluid transudates from the normal peritoneal surface, whereas in ascites associated with peritoneal diseases protein rich ascitic fluid exudates from the peritoneal surface. Ascitic fluid is termed transudate if AFTP (ascitic fluid total protein) is less than 2.5g/dl. Ascitic fluid is termed exudate if AFTP > 2.5g/dl. Currently many problems and exceptions have been noted with this concept. Many infected and malignancy related samples have been reported to have transudative fluid and many samples obtained from patients with cirrhosis or heart failure had exudative ascitic fluid.²

Serum - ascites albumin gradient (SAAG) is derived by subtracting ascitic fluid albumin from serum albumin. This method is physiologically based and is a parameter of oncotic pressure gradient reflecting presence or absence of portal hypertension.^{3,4} increased

ascites of portal hypertensive etiology and decreased in ascites of non-portal hypertensive etiology.⁵ The SAAG is accurate in 96.7% cases even in the presence of diuresis, intravenous infusions of albumin. However it is inaccurate in cases of mixed ascites.⁶

In a study cholesterol concentrations in the ascitic fluid provided the best diagnostic accuracy in the classification of ascitic fluids into exudates and transudates, with a sensitivity of 64.2% and a specificity of 86.7% as compared to SAAG with sensitivity of 86.8% and specificity of 40%.⁷

The ascitic fluid cholesterol level is sensitive in diagnosing malignancy related ascites. The high cholesterol level in malignancy related ascites is due to obstruction in lymph flow causing a rupture of lymphatic channel, which leads to secretion of chyle into the peritoneal cavity. Thus there is increased level of cholesterol in ascitic fluid. The other source of ascitic fluid cholesterol is cell membrane of malignant cells, so when the fragile malignant cells break down in ascitic fluid, cholesterol from the cell membrane is released into the ascitic fluid. Several studies have proved an elevated ascitic fluid cholesterol levels in patients with malignant ascites. Along with it, serum ascites cholesterol gradient (SACG) too aids in differential diagnosis of ascites⁸⁻¹⁰.

Therefore aim of present study was to differentiate the portal and non-portal hypertensive causes of ascites with the help of serum ascitic albumin gradient (SAAG) and to differentiate malignant and non-malignant causes of ascites with the help of serum ascitic cholesterol gradient (SACG).

Material and Method: one hundred and thirty patients with ascites were taken for the study irrespective of the etiology of ascites during time period from November 2013 to April 2015. All patients were subjected to detailed history and thorough clinical examination.

Serum and ascitic albumin was measured by BCG(Bromocresol green) method using

colorimetry. Serum and ascitic cholesterol was measured by enzymatic method using colorimetry.

Diagnosis of portal hypertension was established by ultrasonography of abdomen and portal venous system. Ultrasonogram diagnosis of portal hypertension was based on demonstration of dilated portal vein (> 13 mm diameter) with or without splenomegaly. Ultrasound evidence of altered hepatic echotexture with nodularity in presence of portal hypertension, was considered as cirrhosis of liver. Cardiac cause of ascites was diagnosed on the basis of history, clinical examination and echocardiographic evidence of cardiac failure. Malignancy related ascites was diagnosed with cytological examination of ascitic fluid revealing malignant cells or by imaging techniques or by relevant histopathological examination. Nephrogenic cause was considered in patients with albuminuria >3gm in 24 hours and by clinical assessment after ruling out tuberculosis, cirrhosis and cardiac failure. Tuberculosis abdomen was considered if history and clinical features are suggestive of tuberculosis and also if ascitic fluid shows elevated lymphocyte count &/or if Ascitic fluid adenosine deaminase >30IU/L.

Statistical analysis: Unpaired student's 't' test for 2 sample mean was applied to compare the mean values of two groups. Receiver operating characteristic curve was plotted to determine the sensitivity and specificity. Statistical software SPSS was used for statistical analysis.

Results:

The mean age of patients was 46.2 ± 14.2 years. Out of the 130 cases studied 99 were due to cirrhosis, 10 due to tuberculosis abdomen, 9 due to malignancy, 7 due to pancreatitis, 3 due to dilated cardiomyopathy (DCMP), 1 due to hypoproteinemia with severe anaemia and 1 due to nephrotic syndrome. Portal hypertension was recorded in 104 cases.

The most common presenting symptom was abdominal distension, pain in abdomen and loss of appetite. On clinical evaluation, 28 patients had icterus and 17 were pallor. Splenomegaly was observed in 19 cases whereas 13 patients have hepatomegaly. Out of 130 patients; 95 patients were alcoholic. Table 1 shows the values of serum and ascitic albumin and cholesterol in different groups. Serum and ascitic albumin was highest in DCMP group whereas serum and ascitic cholesterol was highest in malignant group.

Serum and ascitic albumin, cholesterol levels, serum ascitic albumin gradient (SAAG) and serum ascitic cholesterol gradient (SACG) were compared in malignant and non-malignant group and we observed significant higher ascitic albumin and cholesterol and significantly lower SACG and SAAG in malignant group as compared to non-malignant group. Significant lower ascitic albumin and cholesterol, and higher SACG and SAAG were observed in portal hypertensive group as compared to non-portal hypertension group (Table 2).

Table 1: Serum and Ascitic Albumin and Cholesterol level in Different Groups

Diagnosis		Serum Albumin (mg/dl)	Serum Cholesterol (mg/dl)	Ascitic Fluid Albumin (mg/dl)	Ascitic Fluid Cholesterol(mg/dl)
Cirrhosis	(n=99)	2.425±0.202	114.74775±8.1548	1.003±0.029	36.4108±5.5327
Tuberculosis	(n=10)	2.7878±0.688	123.20±8.30	2.068±0.5788	57.30±6.29
Malignancy	(n=9)	2.61762±0.419	132.67±8.44	1.853±0.495	93.11±9.20
Pancreatitis	(n=7)	1.900±0.105	126.00±6.14	1.042±0.113	55.86±7.67
DCMP	(n=3)	3.667±0.202	112.33±10.41	2.160±0.040	43.00±2.65

Table 2: Albumin and cholesterols in Serum and Ascitic Fluid

	Serum Albumin(mg/dl)	Ascitic Fluid Albumin(mg/dl)	SAAG(mg/dl)	Serum Cholesterol(mg/dl)	Ascitic Fluid Cholesterol(mg/dl)	SACG(mg/dl)
Malignant cause	2.61±0.41	1.85±0.49	0.76±0.241	132.66±0.44	93.11±9.19	39.55±10.48
Non Malignant causes	2.44±0.38	1.11±0.38	1.32±0.31	118.43±31.28	40.63±14.85	77.65±20.68
P value	0.193	≤0.0001	<0.0001	0.177	<0.0001	<0.0001
Portal Hypertensive causes	2.44±0.31	1.03±0.20	1.41±0.22	117.47±33.54	38.0±14.15	79.39±21.69
Non Portal hypertension causes	2.40±0.60	1.71±0.62	0.76±0.19	127.23±0.61	69.30±19.17	57.53±15.45
P value	0.193	<0.0001	<0.0001	0.145	<0.0001	<0.0001

The sensitivity and specificity of SAAG for portal hypertensive causes of ascites was found to be 99.00% and 96.2% respectively at cut off value of ≥ 1.03 gm/dl (Figure 1 and table 3).

For malignant cause of ascites; the sensitivity and specificity of SACG was observed as 100% and 99.2% respectively at cut off value ≤ 53 mg/dl (Table 3 and figure 2).

Table 3: Sensitivity, Specificity, Positive and Negative Predictive values, Efficacy of SAAG & SACG

Parameter	SAAG (%)	SACG (%)
Sensitivity	99.03	100
Specificity	96.15	99.15
Positive predictive value	99.03	90
Negative predictive value	96.15	100
Efficacy	98.4	99.2

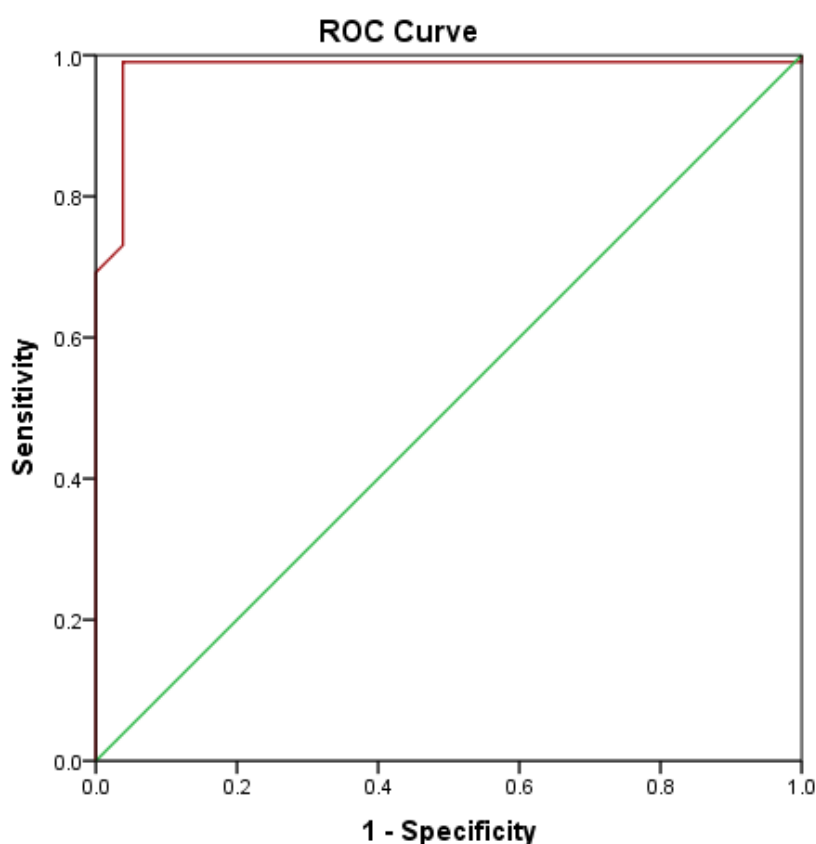


Figure 1: Receiver operating characteristics (ROC) plots for Serum Ascites Albumin Gradient for differentiating portal hypertensive causes of ascites

DISCUSSION

The differential diagnosis of ascites remains a clinical problem unless a positive diagnosis of malignancy or infection is confirmed by cytology or culture. Such a definite cause cannot be firmly established by conventional analysis of ascitic fluid. Moreover these possibilities may be suspected inappropriately in patients with ascites related to liver diseases. The earlier approach used in the differential diagnosis consisted of separating ascitic fluid based on the concentration of protein. However classification has been challenged in different clinical conditions^{2,11}.

The present study was undertaken to evaluate the reliability of SAAG, a parameter reflecting the oncotic pressure gradient between the vascular bed and the interstitial splanchnic or ascitic fluid. According to Starling hypothesis the fluid movement across the capillaries is controlled by the balance of hydrostatic and colloidal osmotic forces across the capillary wall. These forces tend to achieve a dynamic equilibrium so that the increased portal pressure is counter balanced by increased oncotic pressure gradient across the capillary membrane. This physiological event is the basis for postulated serum - ascites albumin gradient as the true indicator of presence or absence of increased portal pressure.¹²

The SAAG correctly differentiated ascites of portal hypertension and non-portal hypertension causes in 99.03% of the cases. The efficacy of SAAG in the present study to classify portal hypertension and non-portal hypertension etiology is 98.4%. These values are comparable to the results obtained by Younas et al et al (96%)¹³ and Runyon et al (96.7%).²

In ascites of liver disease, 96 out of 99 patients of liver diseases serum - ascites albumin gradient was increased, i.e. in portal hypertensive range. This correlated well with the previous studies by Pierre pare et al¹⁵ who studied 51 patients with ascites in which 29 patients had liver disease out of which serum - ascites albumin gradient was in the predicted range in 28 patients.

In the present study, in all the 3 patients of DCMP, serum - ascites albumin gradient is in the portal hypertension range i.e. >1.03 gm %. Pierre et al¹⁴ reported 100% efficacy of serum - ascites albumin gradient in cardiac failure patients.

In tuberculosis serum - ascites albumin gradient placed it under non-portal hypertension etiology in all 10 patients. Marshall et al¹⁵ reported SAAG < 1.1 gm/dl in the non-portal hypertension range in all the patients they studied.

In the present study in malignancy related ascites, serum - ascites albumin gradient is in the non-portal hypertensive range in all 9 patients studied. In Pierre et al¹⁴ study serum - ascites albumin gradient retained accuracy in 14 out of 15 patients (93.3%) with malignancy related ascites. Runyon¹⁶ reported efficacy of 96.7% for serum - ascites albumin gradient in malignancy related ascites. This study further substantiates that SAAG can be used classify ascites of portal and non-portal hypertensive causes. In this study SACG was able to differentiate ascites into malignant and non-malignant etiology in 99.2% of cases. Similar results were found by Sharatchandra et al⁹ in which the SACG values in cirrhosis, tuberculosis and malignancy were 99.2 ± 27.8 mg/dl, 54.16 ± 36.26 mg/dl and 50 ± 23 mg/dl respectively with a sensitivity of 80%. In view of the good diagnostic efficacy, easy availability and cost-effectiveness, ascitic fluid cholesterol is also an excellent parameter for the diagnosis of malignant ascites.

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In conclusion SAAG and SACG is a cost effect way and rapid method that can be effectively used in differentiating the portal hypertensive causes and malignant causes of ascites respectively.

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