Diagnostic Value of Serum Ascites Lipid Gradients in Patients with Ascites

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ABSTRACT

Introduction: Ascites is a common presentation in clinical practice. Less expensive biochemical techniques are required to differentiate ascites with unknown etiology. The purpose of this study was to analyse serum ascites lipid gradient (SALG) as a potential diagnostic test.

Material and methods: The study was conducted on patients admitted in the Department of Medicine at Gandhi Medical College and Hamidia Hospital, Bhopal. The study included 100 patients (71 with cirrhosis, 17 with tuberculosis and 12 with malignant ascites). Clinical evaluation, abdominal USG, and laboratory investigations including SAAG (Serum Ascites Albumin Gradient), serum lipid profile and SALG including total cholesterol, Triglycerides, HDL Cholesterol, LDL Cholesterol and VLDL Cholesterol were done.

Results: SAAG value in cirrhosis were 1.62±0.390 (>1.1), tuberculosis were 0.82±0.0(<1.1) and malignancy were 0.78±0.426 (<1.1). The SALG levels for differentiating high SAAG from low SAAG were 99.24 ± 10.51 versus 49.24 ± 21.9 and 56.08± 10.82 for SALG - total cholesterol, 70.56 ± 5.04 versus 61.29 ± 24.11 and 56.42 ± 10.71 for SALG - triglyceride, 22.44 ± 3.55 versus 16.29 ± 6.18 and 16.75 ± 5.93 for SALG - HDL cholesterol, 57.42± 5.68 versus 28.12 ± 12.22 and 26.75 ± 5.69 for SALG LDL cholesterol and 1.73 ± 1.04 versus 8.35 ± 2.57 and 9 ± 3.41 for SALG - VLDL Cholesterol respectively. These values are significantly higher in cirrhosis than tuberculosis or malignancy expects SALG -VLDL Cholesterol.

Conclusion: SALG can differentiate cirrhotic ascites from tuberculosis or malignant ascites but not tuberculous ascites from malignant ascites.

Keywords: Ascites, Cirrhosis, Malignancy, Serum Ascites Albumin Gradient (SAAG), Serum Ascites Lipid Gradient (SALG), Tuberculosis.

INTRODUCTION

Ascites is common clinical complication of different diseases. Liver cirrhosis (80%), followed by peritoneal malignancy (10%), tuberculous peritonitis (2%), congestive cardiac failure, nephrotic syndrome are the common cause.[1]

Various parameters like ascitic fluid analysis, cell count, total protein conc., SAAG, cytology, cholesterol, amylase, lactic acid dehydrogenase, adenosine deaminase, and fibronectin level have been used to differentiate exudative (Ascitic fluid total protein >2.5) and transudative (Ascitic fluid total protein ≤2.5) ascites of different etiologies.[2-6]

Physiologically approach to classify ascites by SAAG has been completely replaced the traditional way of classification as transudate and exudates.[3,7,8] High albumin gradient (>1.1%) is usually associated with increased portal hypertension in cirrhosis and low (<1.1%), in conditions where ascites is not related to portal hypertension.[9-12] In patients with low albumin gradient the major differential is between malignant ascites and other etiologies. Though ascitic fluid cytology is a gold standard for malignancy, its diagnostic sensitivity is 64%.[13-16]

Some studies have demonstrated an increased ascitic fluid cholesterol level in patients with malignant ascites. Serum ascites cholesterol gradient (SACG) is helpful in differential diagnosis of ascites.[17-19]

Few studies have related the Serum and Ascitic fluid- Total protein, Albumin, Cholesterol, and their gradients in differential diagnosis of ascites. SAAG has been suggested of categories ascites better than total protein concentration or other parameters. Serum ascites lipid gradient (SALG) is a subtraction of ascitic fluid values of lipid fraction from serum value of lipid fraction, but only few studies have been published on the significance of SALG in differential diagnosis of ascites.

This study was conducted to estimate the Ascitic Fluid Lipid Gradient (SALG) in Alcoholic liver cirrhosis, tubercular and malignant ascites and compared to the SAAG (Serum Ascites Albumin Gradient) in patients with Ascites.

MATERIAL AND METHODS

This was hospital based, prospective, observational cross sectional study. The present study was carried out on patients with ascites admitted in Medical and Radiotherapy Department in Hamidia Hospital Bhopal during the period December 2014 to December 2015. This study included 100 patients with ascites of different etiologies-71 patients with liver cirrhosis, 17 patients with tuberculosis and 12 patients with malignant ascites.

Patients having hepatocellular carcinoma with cirrhosis, spontaneous bacterial peritonitis, ascites due to other etiologies, mixed causes of ascites (cirrhosis with tuberculosis, cirrhosis with malignancy) were excluded from the study.

Clinical evaluation (medical history and physical examination), abdominal ultasonographic examination, liver function tests (serum level of total and direct bilirubin, albumin, alanine aminotransferase, aspartate aminotransferase), prothrombin time, serum lipid profile, ascitic lipid profile and ascitic fluid albumin, complete blood count were done.

Twelve hours fasting blood and ascitic fluid samples were simultaneously collected under aseptic technique from all patients.

Blood samples were collected in plain tubes and centrifuged for 10 min at 3000 rpm within 1 hour of collection. Serum and ascitic fluid samples were stored immediately at - 20^{0}c till analysis was

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done within a period of 48 hours. Ascitic fluid samples were obtained by paracentesis, and collected in sterile containers. Ascitic fluid centrifuged and supernatants were separated into new plain tubes. Albumin, cholesterol, triglycerides, HDL level were measured in serum and ascitic fluid supernatants by using fully automated analyzer.

LDL and VLDL values were calculated by using Friedewaled formula.

LDL Cholesterol = total cholesterol-HDL Cholesterol-TG/5.

VLDL Cholesterol= Triglyceride Cholesterol/5.

Serum-Ascites X Gradient = X concentration in serum- X concentration in ascitic fluid. (where X refer to the substance of interest i.e. albumin and lipids.)

The study was done after obtaining permission of the institutional ethics committee. A written Informed consent was taken from patients or from patients relative.

STATISTICAL ANALYSIS

Statistical analysis was done using computer software (SPSS version 20). The qualitative data were expressed in proportion and percentages and the quantitative data expressed as mean and standard deviations. The difference in proportion was analyzed by using chi square test and the difference in means were analyzed by using student T Test [unpaired]. Significance level for tests were 95% (P< 0.05).

RESULTS

Demographic, clinical, and laboratory characteristics of patients with ascites are shown in Table-1. The study included 100 patients of ascites of different etiologies 71 patients with liver cirrhosis (59 male and 12 female), age 24-65 years, mean age was 40.62 ± 10.98 years. 17 patients had tuberculosis (12 male and 5 female), age 16-60 years and mean age 39.35 ± 15.89 years. 12 patients had malignant ascites (6 male and 6 female), age 50-80 years and mean age 64.17±10.3 years with etiologic causes including ovarian carcinoma, pancreatic carcinoma, carcinoma breast, carcinoma stomach, carcinoma colon.

Serum albumin levels were significantly low in cirrhotic patients (2.06 ± 0.232gm%) when compared with tuberculous (3.12±0.487gm%) and malignant ascites (3.50 ± 0.522gm%) (p<0.001, p<0.001 respectively). There was no statistically significant difference between tuberculosis and malignant ascites (p =0.06)

Ascitic fluid albumin levels were significantly low in cirrhosis group (1.00±0.169gm%) when compared with tuberculous and malignant groups (p <0.001; p <0.001). The difference between tuberculosis (2.47±0.624gm%) and malignant (2.42±0.515gm %) groups was not significant (p =0.81).

The difference in the SAAG was significantly higher in cirrhotic group (1.62±0.390gm %) when compared with tuberculosis and malignant ascites (p =0.002, p =0.018 respectively) where as the difference between tuberculosis (0.82±0.00 gm%) and malignancy (0.78±0.426gm%) was not statistically significant (p =0.48;).

Serum cholesterol was significantly higher in patients with malignant ascites (155.33±9.71 mg%) and tubercular ascites (150.18±12.92 mg%) compared to cirrhotic (132.62±6.42 smg%) and (p<0.001 and <0.001 respectively); whereas the difference between malignant and tuberculosis groups was not statistically significant (p =0.25)

The ascitic fluid cholesterol was significantly elevated in malignant ascites (99.25±5.12 mg%) and tubercular ascites (99.76±20.55 mg%) when compared with cirrhosis (31.92±8.37 mg%) (p<0.0001 each).

The cirrhotic group had highest SACG (31.92±8.37 mg%) when compared with tubercular group (49.24±21.9 mg%) and malignant group (56.08±10.82 mg%). The difference between cirrhotic group and non cirrhotic group (tubercular or malignant is significant (p<0.001, p<0.001 respectively). But the difference between tubercular and malignant groups is not significant (p=0.33 table-2). At cut off value of SACG 68 mg%, the sensitivity is 94%, specificity is 87%, positive predictive value is 96% and negative predictive value is 80% and diagnostic value is 92%.

Serum Triglyceride was significantly higher in patients with malignant ascites (131.67±10 mg%) and tubercular ascites (123±17 mg%) compared to cirrhotic (100.31±7.22 mg%) and

Variables	Cirrhosis [n=71]		TB [n=17]		Malignancy [n=12]	
	Mean	± SD	Mean	± SD	Mean	± SD
Age	40.62	10.98	39.35	15.89	64.17	10.3
BMI	23.82	1.68	24.06	1.3	20.83	1.99
Jaundice	45		2		5	
	62.00%		11.80%		41.70%	
Hepatomegally	26		0		2	
	36.60%		0.00%		16.70%	
Spleenomegally	13		0		0	
	16.90%		0.00%		0.00%	
ABD Mass	1		0		2	
	1.40%		0.00%		16.70%	
Laboratory data						
Hb	11.2	1.59	11.18	1.63	11.42	1.68
Total bilirubin	3.03	1.5	1.24	0.66	2.25	1.14
ALT	54.24	11.6	31.88	5	40	6.9
AST	53.15	13.3	33.35	7.45	38.5	8.83
PT	16.51	1.16	12.41	0.87	13.33	1.07
Sr. Creat	2.17	1	1.24	0.44	2.42	1.31

Table-1: Demographic, clinical and laboratory distribution of patient characteristics

(p<0.001 and <0.001 respectively); whereas the difference between malignant and tuberculosis groups was not statistically significant (p =0.34).

The ascitic fluid Triglyceride was significantly elevated in malignant ascites (75.25±3.84 mg %) and tubercular ascites (69.71±14.15 mg%) when compared with cirrhosis (29.69±5.75 mg %) (p<0.0001 each) whereas the difference between malignant and tuberculosis groups was not statistically significant (p =0.34).

The cirrhotic group had highest SATGG (70.56±5.04 mg%) when compared with tubercular group (61.29±24.11 mg%) and malignant group (56.42±10.71 mg%). The difference between cirrhotic group and non cirrhotic group (tubercular or malignant is significant (p<0.001, p<0.003 respectively). But the difference between tubercular and malignant groups is not significant (p=0.52 table-2). At cut off value of SATGG 68 mg%, the sensitivity is 66%, specificity is 61%, positive predictive value is 85% and negative predictive value is 32% and diagnostic value is 60% (table-4).

Serum HDL Cholesterol was significantly higher in patients with malignant ascites (42.42±4.27 mg%) and tubercular ascites (42.53±5 mg%) as compared to cirrhotic (30.89±3.18 mg%) and (p<0.001 and <0.001 respectively); whereas the difference between malignant and tuberculosis groups was not statistically significant (p =0.95).

The ascitic fluid HDL Cholesterol was significantly elevated in malignant ascites (25.67±4.72 mg%) and tubercular ascites (26.82±6.62 mg%) when compared with cirrhosis (8.45±2.85 mg%) (p<0.0001 each). whereas the difference between malignant and tuberculosis groups was not statistically significant (p =0.61)

The cirrhotic group had highest Serum-Ascites HDL Gradient (22.44±3.55 mg%) when compared with tubercular group (16.29±6.18 mg%) and malignant group (16.75±5.93 mg%). The difference between cirrhotic group and non cirrhotic group (tubercular or malignant is significant (p<0.001, p<0.001 respectively). But the difference between tubercular and malignant groups is not significant (p=0.84 table-2). At cut off value of Serum-Ascites HDL Gradient 19 mg%, the sensitivity is 84%, specificity is 74%, positive predictive value is 91% and negative predictive value is 52% and diagnostic value is 78% (table-4).

Serum LDL Cholesterol was significantly higher in patients with malignant ascites (90±5.33 mg%) and tubercular ascites (90.06±6.06 mg%) compared to cirrhotic (77.86±4.32 mg%) and (p<0.001 and <0.001 respectively); whereas the difference between malignant and tuberculosis groups was not statistically significant (p =0.97).

The ascitic fluid LDL Cholesterol was significantly elevated in malignant ascites (63.25±6.69 mg %) and tubercular ascites (61.94±13.68 mg%) when compared with cirrhosis (20.44±6.38 mg %) (p<0.001 each). whereas the difference between malignant and tuberculosis groups was not statistically significant (p =0.76)

The cirrhotic group had highest Serum-Ascites LDL Gradient (57.42±5.68 mg%) when compared with tubercular group (28.12±12.22 mg%) and malignant group (26.75±5.69 mg%). The difference between cirrhotic group and non cirrhotic group (tubercular or malignant is significant (p<0.001, p<0.001 respectively). But the difference between tubercular and malignant groups is not significant (p=0.72 table-2). At cut off value of Serum-Ascites LDL Gradient 43 mg%, the sensitivity is 92%, specificity is 91%, positive predictive value is 89% and negative predictive value is 83% and diagnostic value is 94% (table-4).

Serum VLDL Cholesterol was significantly higher in patients with malignant ascites (25.58±1.98 mg%) and tubercular ascites (21.18±2.65 mg%) compared to cirrhotic (9.54±2.03 mg%) and (p<0.001 and <0.001 respectively); whereas the difference between malignant and tuberculosis groups was not statistically significant (p =0.25).

The ascitic fluid VLDL Cholesterol was significantly elevated in malignant ascites (16.5±2.47 mg %) and tubercular ascites (14.3±1.07 mg%) when compared with cirrhosis (7.8±1.9 mg%) (p<0.001each).

The cirrhotic group had lowest Serum-Ascites VLDL Gradient (1.73±1.04 mg%) when compared with tubercular group (8.35±2.57 mg%) and malignant group (9±3.41 mg%). The difference between cirrhotic group and non cirrhotic group (tubercular or malignant is significant (p<0.001, p<0.001

Gradient	Cirrhosis [n=71]		TB [n=17]		Malignancy [n=12]		P value		
	Mean	± Sd	Mean	± Sd	Mean	± Sd	Malignancy Vs Cirrhosis	Cirrhosis Vs TB	Malignancy Vs TB
CHOLE Gradient	99.24	10.51	49.24	21.9	56.08	10.82	<0.001 *	<0.001 *	0.33
TG Gradient	70.56	5.04	61.29	24.11	56.42	10.71	<0.001 *	0.003 *	0.52
HDL Gradient	22.44	3.55	16.29	6.18	16.75	5.93	<0.001 *	<0.001 *	0.84
LDL Gradient	57.42	5.68	28.12	12.22	26.75	5.69	<0.001 *	<0.001 *	0.72
VLDL Gradient	1.73	1.04	8.35	2.57	9	3.41	<0.001 *	<0.001 *	0.56

Table-2: Serum Ascites Lipid Gradient among patients with ascites

	Child Grd A[9]		Child Grd B[27]		Child Grd C[35]		p value
	Mean	± SD	Mean	± SD	Mean	± SD	
CHOLE Gradient	101.78	3.56	98.48	14.60	99.17	7.70	0.81
TG Gradient	70.33	5.24	71.30	5.99	70.06	4.20	0.34
HDL Gradient	20.44	2.35	22.59	3.74	22.83	3.57	0.82
LDL Gradient	56.33	4.66	56.85	7.43	58.14	4.26	0.39
VLDL Gradient	1.78	0.83	1.96	1.34	1.54	0.78	0.13

Table-3: Correlation between Serum Ascites Lipid Gradient levels and severity of cirrhosis

respectively). But the difference between tubercular and malignant groups is not significant (p=0.56). At cut off value of Serum-Ascites VLDL Gradient 5 mg%, the sensitivity is 9%, specificity is 13%, positive predictive value is 23% and negative predictive value is 4% and diagnostic value is 9% (table-4). The level of SALG correlate with severity of cirrhosis (Child grade) but there is no significant difference shown (table-3).

DISCUSSION

Many diseases are complicated by ascites. The most common cause of ascites is portal hypertension secondary to liver cirrhosis, but in about 20% of cases there is an extra hepatic cause and 5% have more than one cause of ascites (mixed)-usually cirrhosis with either tuberculosis or malignancy. It's appropriate treatment depend on proper diagnosis.[20]

This study is focused to evaluate the diagnostic sensitivity of various diagnostic parameters to differentiate Cirrhotic, Tuberculous and Malignant Ascites from each other and also to assess Ascitic Fluid Lipids and Serum Ascites Lipid Gradients.

Serum-Ascites Albumin Gradient (SAAG) was adopted as a newer and more physiological approach to classify ascites on the basis of presence or absence of portal hypertension.[8,21]

Hoefs et al.[22] established a cut off value of 1.1gm/dl, it was supported by various other studies.[8,21] SAAG ≥1.1 gm/dl can differentiate cirrhotic from non cirrhotic ascites. Similar results were observed in our study, with critical value of ≥1.1gm/dl. SAAG differentiate cirrhotic from non cirrhotic ascites. If the SAAG is ≥1.1gm/dl, the patient is considered to have portal hypertension. Conversely if the SSAG is <1.1gm/dl, the patient is unlikely to have portal hypertension like tuberculosis or malignant. The study by Lu CW et al[23] shows that SAAG was not as useful.

Presently SAAG is included in the guideline of investigations recommended on the management of ascites in cirrhosis by American Association of the study of Liver Disease (AASLD) and British Society of Gastroenterology.

Our study showed that the serum lipid profile (T. Cholesterol, TG, HDL, LDL and VLDL) decreases significantly in cirrhotic patients with a ascites. The results of our study is similar with previous study by Ghadir and colleagues.[24] Serum lipid profile was significantly lower in cirrhotic patients than the control group owing to decreased liver synthesis. Also, the present study is similar to studies from western world which documented that all the lipid profile in cirrhotic patients are lower than the non-cirrhotic patients.[25] Sharatchandra et al[18] and Khairy H Morsy et al[29] reported that serum lipid profile was significantly lower in cirrhotic patients than tuberculous and malignant patient and our study show the similar results.

Gupta R et al[26] have reported on SALG on cholesterol only. They found the SALG (Cholesterol) in cirrhotic, malignant and tubercular group's as 118.3±1.9, 88.6±3.6 and 56.5±2.6 respectively. We found SALG (Cholesterol) value in cirrhotic, tubercular and malignant group as 99.24±10.51, 49.24±21.9 and 56.08±10.82 respectively with a cut off value of 68mg%. There was a clear difference between cirrhosis, tubercular and malignant ascites (p<0.05). The difference was significantly higher in cirrhotic ascites compared to malignant and tubercular ascites, the cut off SALG values being 68 mg%, 68 mg%, 19 mg%, 43 mg% and 5 mg% in Total Cholesterol, TG, HDL, LDL and VLDL Cholesterol respectively in differentiating high albumin gradient ascites from low albumin gradient ascites. Positive and negative predictivity values on cut off values for SALG in Total Cholesterol, TG, HDL, LDL and VLDL Cholesterol in differentiating high albumin gradient ascites from low albumin gradient ascites were 96% and 80%, 85% and 32%, 91% and 52%, 89% and 83%, 23% and 4% respectively. This PPV and NPV compared with study by Sharatchandra et al[18] are similar except for LDL and VLDL.

Ascitic fluid cholesterol and SALG are better marker to differentiate malignant ascites from cirrhotic and tubercular ascites this is contrary to Dharwadkar and Bijoor study who were documented that SACG is not a good marker to differentiate tuberculous ascites and cirrhotic ascites.[27.]

Prieto et al[21] showed that ascitic fluid cholesterol concentration were significantly higher in patients with peritoneal metastasis and was better than ascitic fluid total protein, lactate dehydrogenase and SAAG for differentiating ascites from that due to liver disease. No difference was noted in ascitic fluid cholesterol between those with superimposed hepatocellular carcinoma. The etiology for the elevated cholesterol level in malignancy is due to the increased vascular permeability increased cholesterol synthesis and release from malignant cell implanted on peritoneum.[23,32,34] In our study ascitic fluid cholesterol concentration were significantly elevated in malignant and tubercular ascitic fluid when compared to cirrhotic ascites. With a critical value of 68 mg%, the diagnostic accuracy was 92%. This is supported by the fact that these results were consistent with the study of Sharatchandra et al.[18] and Khairy H Morsy et al.[29]

Variation in cut off value for ascitic fluid cholesterol was observed in different studies. Satya et al[23] (70 mg%) had diagnostic accuracy of 94%, Sharatchandra et al[18] (67 mg%) had a diagnostic accuracy of 96%, R. Gupta et al[26] (55 mg%) had a diagnostic accuracy of 94%. These variations in the cut off level could be attributed to the selection of patient, serum cholesterol level and to the extent of peritoneal implants. In our study critical value of SACG is 68 mg% with its diagnostic accuracy is 92%.

SALG	Cutoff	Sensitivity %	Specificity %	PPV %	NPV %	Accuracy %
Cholesterol Gradient	68	94	87	96	80	92
TG Gradient	68	66	61	85	32	60
HDL Gradient	19	84	74	91	52	78
LDL Gradient	43	92	91	89	83	94
VLDL Gradient	5	9	13	23	4	9
SAAG	1.1	100	100	100	100	100

Table-4: Cut off value of salg values and their sensitivity, specificity, positive predictivity, negative predictivity, and its diagnostic accuracy values

In our study, the lipid gradient was significantly higher in cirrhotic ascites compared to tuberculous and malignant ascites, in triglyceride (70.56±5.04 versus 61.29 ± 24.11 and 56.42 ± 10.71), HDL cholesterol (22.44 ± 3.55 versus 16.29 ± 6.18 and 16.75 ± 5.93), and LDL cholesterol (57.42 ± 5.68 versus28.12 ± 12.22 and 26.75 ± 5.69) respectively in differentiating high albumin gradient ascites from low albumin gradient ascites. These results were consistent with the study of Sharatchandra et al.[18] SALG was not superior to SAAG.

Portal hypertension has been described to be related to many theories – having a high hydrostatic pressure gradient between the portal bed and ascitic compartment. Hypercholesterolaemia has been described in alcoholic liver cirrhosis.[28] It is hypothesized that there might be factors related to lipid gradients which indirectly reflect the abnormally high hydrostatic pressure gradient between the portal system and peritoneal compartment. Chylous ascites on the other hand, is often the result of lymphatic obstruction from trauma, tumor, tuberculosis, filariasis, congenital abnormalities, or nephrotic syndrome. Our study showed significantly higher value of SALG for the portal hypertension group ($p<0.05$). Tubercular ascites had the lowest gradient among the three groups studied, for all parameters except for TG and LDL. The cut-off SALG values being 68 mg%, 68 mg%, 19 mg%, 43 mg% and 5 mg% in cholesterol, triglyceride, HDL cholesterol, LDL cholesterol and VLDL cholesterol respectively in differentiating cirrhotic ascites from tuberculosis or malignant ascites and these results were near the study of Sharatchandra et al.[18] and Khairy H Morsy et al[29]

In our study, A close relationship between the levels of SALG and severity of cirrhosis is found but it is not significant and to the best of our knowledge this was supported by Khairy H Morsy et al[29]

CONCLUSION

SAAG remains the best marker to differentiate cirrhotic ascites from tuberculous or malignant ascites. So SALG can be used as a screening test in ascitic patients as it may give clue to the possible etiology, and help in planning further investigative modalities in ascitic patients. In this study SALG can be differentiate cirrhotic from tuberculous and malignant ascites.

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