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Original research article

Clinical Study of Ascites with Special Reference to Serum – Ascites Albumin Gradient

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ABSTRACT

Background: The Serum – Ascites Albumin Gradient (SAAG) defined as serum albumin concentration minus ascitic fluid albumin concentration. SAAG has been proposed a physiologically based alternative criterion in the classification of ascites.

Aims & Objective: The present study was designed to differentiate ascites based on serum/ascites albumin gradient and comparing transudate and exudate concept using biochemical parameters. The study also designed to know various aetiologies of ascites.

Material and Methods: This cross sectional study was done among 60 patients with ascites in Sri Krishna Medical College and Hospital, Muzaffarpur, Bihar from June 2020 to May 2021. Ascitic fluid and blood samples were sent for various investigations depending on the presentation of the patient to hospital.

Results: In our study Cirrhosis of liver was the most common cause of ascites (78%) followed by Tubercular peritonitis (8%) and alcohol was the commonest cause for cirrhosis of liver (85%) followed by Hepatitis B virus infection. Cirrhosis of liver showed high SAAG compared with tubercular peritonitis and malignant ascites which showed low SAAG. Among High SAAG patients 96% had portal hypertension. Transudative ascites observed in 72.5% of cirrhosis patients whereas tubercular peritonitis showed exudative type in60% of cases.

Conclusion: Cirrhosis liver was the most common cause of Ascites and alcohol was the commonest cause for cirrhosis. SAAG is superior to transudate exudate concept in differentiating the causes of ascites. High SAAG indicates presence of portal hypertension and low SAAG indicates absence of portal hypertension.

Key-Words: Serum-Ascites Albumin Gradient; Exudate; Transudate; Portal Hypertension; Cirrhosis

INTRODUCTION

Ascites is the pathological accumulation of fluid in the peritoneal cavity.[1]The patients who suffer from ascites present a diagnostic and therapeutic problem. Abdominal paracentesis with careful analysis of ascitic fluid should be a very early step in evaluating patients with ascites. It is the most rapid and most effective method in the diagnosis of ascites. The traditional classification of ascites into exudative and Transudative involves estimation of ascitic fluid total protein (AFTP), which is high > 2.5 gm/dl in exudative and < 2.5 mg/dl in transudate.[2]This classification however, is unable to correctly identify the etiological factors responsible for its causation.[3,4]In contrast the Serum – Ascites Albumin Gradient (SAAG)-(defined as serum albumin concentration minus ascitic fluid albumin concentration) has been proposed a physiologically based alternative criterion in the classification of ascites. In case of portal hypertension, oncotic pressure gradient between plasma and ascitic fluid has to be raised, to counter balance the high hydrostatic pressure driving the fluid to the intraperitoneal cavity.[5]The difference between the serum and ascitic albumin concentration was used to differentiate ascitic fluid into gradient > 1.1 gm/dl in case with portal hypertension and < 1.1 gm/dl in ascites unrelated to portal hypertension5. Various studies have shown superiority of SAAG in classifying ascites compared to transudate-exudate concept.[6-8]The present study was undertaken to evaluate the value of SAAG in the differential diagnosis of ascites and also to compare its diagnostic accuracy with that of transudate and exudate concept.

MATERIAL AND METHODS

This study was done among 60 patients with ascites in Sri Krishna Medical College and Hospital, Muzaffarpur, Bihar from June 2020 to May 2021. Patients with a diagnosis of ascites clinically and confirmed by ultrasound examination of abdomen for evidence of free fluid in the peritoneal cavity were taken up for the study. Patients who were receiving long term diuretics and Patients with very low serum albumin (< 1.5 gm/dl) were excluded.

The detailed history and physical examination was done on every patient as per the proforma. The patients were subjected for detailed investigations like blood for total WBC count, differential count, ESR, haemoglobin percentage etc. Blood was drawn for various investigations like random blood sugar, blood urea, serum creatinine and liver function tests which included serum bilirubin, serum total protein, serum albumin, SGOT, SGPT and alkaline phosphatase. The abdominal paracentesis was done in all patients with full aseptic precaution. The location on either flanks two finger breadths cephalad and two finger breadth medial to anterior superior iliac spine was selected for abdominal paracentesis. 10 ml of ascitic fluid was drawn and was sent in two separate bottles for the ascitic fluid analysis. The samples were collected, handled and transported to the Pathological laboratory ofSKMCH.[13,14]25 ml was taken if the bacterial culture was done in patients who were suspected to be having SBP.

The ascitic fluid albumin estimation was done in all patients. SAAG is calculated by subtracting ascitic fluid albumin from serum albumin. Ascitic fluid cell count and differential count was done in all patients. The ascitic fluid cytology for malignant cells done in patients suspected to have malignancy related ascites. The other investigations were done when required to establish a definite diagnosis included ECG in all 12 leads, Chest X-ray PA view, Echocardiography, serum HBsAg, ascitic ADA level. The cirrhosis of liver was diagnosed based on clinical signs of portal hypertension, Laboratory and ultrasonographic evidence.

Classification of patients: Patients of each etiology were classified into exudative and transudative groups. Those patients with Ascitic Fluid Total Protein (AFTP) < 2.5 gm/dl were

taken to transudative group and > 2.5 gm/dl of AFTP were taken to exudative group. The same patients of all the etiologic groups were divided into patients with portal hypertension and patients without portal hypertension. The SAAG value distribution in those two groups was also studied. The presence of portal hypertension was said to be present if, (i) Presence of portosystemic collaterals, splenomegaly by clinical examination; (ii) Presence of esophageal varices proved by upper GI endoscopy; (iii) Unequivocal ultrasound evidence of portal hypertension like diffuse liver involvement, enlargement of portal vein > 13 mm and splenomegaly.

RESULTS

In our study among 60 patients with ascites were analysed and the following results were observed. The common age group where incidence of ascites was high between 40 to 50 years (33.3%) and was more among male patients 85% (Table-1). Transudative ascites was common (66.7%) in our study (Table-2). Cirrhosis of liver (78%) was the common cause of ascites followed by hepatitis B virus infection (Table-3). Among 40 patients with cirrhosis transudative ascites was observed in 29 patients. Exudative ascites was in 33.3% and high SAAG >1.1 was observed in 85% of patients. In this study all patients with cirrhosis of liver had high SAAG (Table-4). Whereas all patients with tubercular peritonitis and malignant ascites had low SAAG. Among 51 patients with high SAAG portal hypertension was seen in 48 patients (96%) (Table-5).

Table 1 : Age and sex distribution of study subjects

Characteristics		No. of Patients (%)
Age in years	20-30	6(10%)
	30-40	15(25%)
	40-50	20(33.3%)
	50-60	14(23.3%)
	>60	5(8.3%)
Sex	Male	9(15%)
	Female	51(85%)
Total		60(100%)

Table 2 : Study subjects showing pattern of transudate and exudates ascites

Ascitic Fluid Protein	No. of Patients
<2.5	40(66.7%)
>2.5	20(33.3%)

Table 3 : Pattern of ascitic fluid protein in various etiologies

Aetiology	Fluid Protein		Total
	<2.5	>2.5	
BCS	1(100%)	0(0)	1(100%)
CL	29(72.5%)	11(27.5%)	40(100%)
HBV	6(100%)	0(0)	6(100%)
HCV	0(0)	1(100%)	1(100%)
MA	1(25%)	3(75%)	4(100%)
SBP	1(33.3%)	2(66.7%)	3(100%)
TP	2(40%)	3(60%)	5(100%)
Total	40(66.7%)	20(33.3%)	60(100%)

BCS: Budd Chiari Syndrome; CL: Cirrhosis of liver; HBC: Hepatitis B Virus; HCV: Hepatitis C virus; MA: Malignant ascites; TP: Tubercular peritonitis; SBP: Sub acute bacterial peritonitis

Table 4 : Pattern of SAAG in various etiologies

Aetiology	SAAG		Total
	<1.1	≥1.1	
BCS	0(0)	1(100%)	1(100%)
CL	0(0)	40(100%)	40(100%)
HBV	0(0)	6(100%)	6(100%)
HCV	0(0)	1(100%)	1(100%)
MA	4(100%)	0(0)	4(100%)
SBP	0(0)	3(100%)	3(100%)
TP	5(100%)	0(0)	5(100%)
Total	9(15%)	51(85%)	60(100%)

BCS: Budd Chiari Syndrome; CL: Cirrhosis of liver; HBC: Hepatitis B Virus; HCV: Hepatitis C virus; MA: Malignant ascites; TP: Tubercular peritonitis; SBP: Sub acute bacterial peritonitis

Table 5 : Relationship of SAAG with USG abdomen findings

Procedure	Diseases	SAAG		Total
		<1.1	≥1.1	
USG Abdomen	Ascites	4(57.1%)	3(42.9%)	7(100%)
	Ca. Ovary	1(100%)	0(0)	1(100%)
	Ca. Pancreas	1(100%)	0(0)	1(100%)
	CLPH	2(4%)	48(96%)	50(100%)
	Secondary in liver	1(100%)	0(0)	1(100%)
Total		9	51	60

DISCUSSION

The results of present study shows that the serum ascitic albumin gradient is a useful marker for the diagnosis of ascites, as it has diagnostic accuracy of 100%.[14]Similar observations have also been reported by other studies such as Runyon BA[6], Gopal AK et al.[15]and Gupta R et al. If the gradient is > 1.1g/dl, the underlying cause is almost always related to portal hypertension. The application of albumin gradient disregards the concept of transudate versus exudate as it provides a more rational approach separating ascitic fluid into two categories on the basis of presence or absence of portal hypertension.

The albumin gradient retains its ability even in infected ascites, which is considered exudate according to traditional concept, although it usually develops in patients of cirrhosis which owing to the low ascitic fluid total protein concentration, is traditionally labelled as transudative ascites infact. The results of the present study reinforce the conclusions of reports which showed that albumin gradient is superior to transudate exudate concept in differentiating the causes of ascites.

The utility of albumin gradient in non-alcoholic liver disease has been debated.[15]However, in the present study the test was found to have significant diagnostic accuracy in ascites caused by both alcoholic and non-alcoholic liver disease. The results of the present study show that the serum ascites albumin gradient is a test with significant accuracy in separating ascites related to portal hypertension from the forms of ascites caused by mechanisms unrelated to portal hypertension. It does not provide the exact cause of ascites. The presence of high albumin gradient only means, the presence of portal hypertension. It is superior to previously proposed transudate exudate concept, because it provides a better approach to pathogenesis of ascites. The transudative exudative ascites should be replaced with ascites related to portal hypertension (high SAAG) and ascites not related to portal hypertension (low SAAG).

CONCLUSION

We conclude that SAAG is superior to transudate exudate concept in differentiating the causes of ascites. High SAAG indicates presence of portal hypertension and low SAAG indicates absence of portal hypertension.

REFERENCES

1. Runyon BA. Approach to the patient with ascites. In: Yamada T, Alpers DH, Kaplowitz N, editors. Textbook of Gastroenterology. Vol. 1. Philadelphia: Lippincott, Williams and Wilkins; 2003. pp. 948–72.
2. Rovelstad RA, Bartholomew LG, Cain JC, Mckenzie BF, Soule EH. The value of examination of ascitic fluid and blood for lipids and for proteins by electrophoresis. Gastroenterology 1958; 34: 436-50.
3. Sampliner RE, lber FL. High protein ascites in patients with uncomplicated hepatic cirrhosis. Am J Med Sci. 1974; 267: 275-9.
4. Rector WG, Reynolds TB. Superiority of the serum-ascites albumin difference over the ascites total protein concentration in separation of ‘transudative’ and ‘exudative’ ascites. Am J Med 1984; 77: 83-5.
5. Pare P, Talbot J, Hoefs JC. Serum-ascites albumin concentration gradient: A physiologic approach to the differential diagnosis of ascites. Gastroenterology. 1983; 85: 240-4.
6. 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; 117: 215-20.
7. Goyal AK, Goyal SK, Pokharna DS, Sharma SK. Differential diagnosis of ascitic fluid: Evaluation and comparison of various biochemical criteria with a special reference to serum ascites albumin concentration gradient and its relation to portal pressure. Trop Gastroenterol. 1989;10(1):51-5.
8. Gupta R, Misra SP, Dwivedi M, Misra V, Kumar S, Gupta SC. Diagnostic ascites: Value of total protien albumin, cholesterol their ratios serum ascites albumin and cholesterol gradient. J GastroenterolHepatol. 1995;10(3):295-9.
9. Arroyo V, Gines P, Planas R. Pathogenesis, diagnosis, and treatment of ascites in cirrhosis. In: Bircher J, Benhamou J-P, McIntyre N, eds. Oxford textbook of clinical hepatology. New York: Oxford University Press, Inc, 1999. 697–731.731.
10. Runyon BA. Ascites and spontaneous bacterial peritonitis. In: Feldman M, Friedman LS, Sleisenger MH, eds. Sleisenger&Fordtran's Gastrointestinal and Liver Disease. 9th ed. Philadelphia, Pa: Saunders Elsevier; 2010.

11. Ginés P, Quintero E, Arroyo V, Terés J, Bruguera M, Rimola A, et al. Compensated cirrhosis: Natural history and prognostic factors. Hepatology. 1987;7(1):122-8.
12. Saunders JB, Walters JR, Davices J. A 20-years prospective study of cirrhosis. BMJ. 1981; 282: 263-266.
13. Jain AP, Chandra LS, Gupta S, Gupta OP, Jajoo UN, Kalantri SP. Spontaneous bacterial peritonitis in liver cirrhosis with ascites. J Assoc Physicians India. 1999;47(6):619-21.
14. Jenkinson F, Murphy MJ. Biochemical analysis of pleural and ascitic fluid. Ann clinBiochem. 2007; 44:471-473.
15. Kajani MA, Yoo YK, Alexander JA, Gavaler JS, Stauber RE, Dindzans VJ, et al. Serum-ascites albumin gradients in nonalcoholic liver disease. Dig Dis Sci 1990; 35: 33-7.

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