ACCURACY OF SERUM - ASCITES ALBUMIN GRADIENT IN THE AETIOLOGICAL DIAGNOSIS OF ASCITES

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ABSTRACT

50 adults with ascites admitted to our hospital were studied. Simultaneous samples of ascitic fluid and blood were collected and subjected to analysis including ascitic fluid total protein and serum ascites albumin gradient. The cut off value of serum-ascites albumin gradient for differentiating between high and low gradient was taken as 1.1 gm% and of ascitic fluid protein for differentiating exudate and transudate as 2.5 gm%. The sensitivity, specificity, positive predictive value and negative predictive value of high gradient and transudative ascites in diagnosing portal hypertension were 94.3%, 60%, 84.6%, 81.8% and 62.9%, 13.3%, 91.7% and 50% respectively. High gradient ascites is a sensitive test in the diagnosis of portal hypertension as a cause of ascites. The exudate-transudate approach has severe limitations in the differential diagnosis of ascites.

MJAFI 2002; 58 : 124-126

KEY WORDS:Albumin Gradient; Ascites; High Gradient Ascites;Serum-Ascites.

Introduction

Abdominal paracentesis with appropriate ascitic fluid analysis is a rapid and cost effective method of diagnosing the cause of ascites. Conventionally, ascites has been classified as transudate or exudate with a cut-off value of ascitic fluid total protein (AFTP) of 2.5gm%. In patients with cirrhosis of liver, the ascites is usually a transudate. However, upto 20% of these patients may have exudative ascites thus complicating decision making [1].

Portal hypertension (PHT) results in a high hydrostatic pressure gradient between the portal bed and ascitic fluid. As a compensatory mechanism, a difference develops between the ascitic fluid and intravascular oncotic forces [2]. Amongst all proteins, albumin exerts the maximum oncotic force per gram. Accordingly, the Serum-Ascites Albumin Gradient (SAAG), i.e. serum albumin minus ascitic albumin, reflects the changes in oncotic forces due to PHT and thus provides a useful tool in the differential diagnosis of ascites [3]. The present study was carried out to ascertain the accuracy of SAAG in predicting the aetiology in patients with ascites.

Material and Methods

50 consecutive adults with ascites admitted to our hospital were studied. Simultaneous samples of blood and ascitic fluid were collected prior to administration of diuretics and subjected to analysis including AFTP and SAAG. Paracentesis was performed by means of a right iliac fossa puncture with a 22 gauge needle

and 50 ml of ascitic fluid was obtained under aseptic conditions. Investigations like abdominal ultrasound, CT scan, upper gastrointestinal endoscopy, liver biopsy and guided fine needle aspiration cytology were carried out as deemed necessary to arrive at a final diagnosis.

Patients with SAAG value of more than 1.1 gm% were considered to have high gradient ascites (HGA) and those with AFTP value less than 2.5 gm%, transudative ascites. The sensitivity, specificity, positive predictive value (PPV) and negative predictive value (NPV) of HGA and transudative ascites in diagnosing PHT were calculated.

Results

There were 34 (68%) males and 16 (32%) females with a mean age of 33.5 (range 15 to 73) years. The aetiology of ascites in these 50 patients is shown in Table-1.

TABLE 1

Causes of ascites with distribution of patients based on ascitic fluid total protein and serum-ascites albumin gradient

Diagnosis	Number	Transudate	Exudate	High gradient	Low gradient
Cirrhosis liver	35	22	13	33	2
Disseminated malignancy	9	1	8	5	4
Peritoneal tuberculosis	2	—	2	—	2
Pancreatitis	1	—	1	—	1
Biliary peritonitis	1	—	1	—	1
Cardiac failure	1	1	—	1	—
Nephrotic syndrome	1	1	—	—	1
Total	50	25	25	39	11

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TABLE 2

Accuracy of high gradient and transudative ascites in diagnosis of portal hypertension

Parameter	Portal hypertension	No portal hypertension	Sensitivity	Specificity	Positive predictive value	Negative predictive value
High SAAG	33	6	94.3%	60%	84.6%	81.8%
Low SAAG	2	9	—	—	—	—
Transudate	22	3	62.9%	13.3%	91.7%	50%
Exudate	13	12	—	—	—	—

Out of 35 patients with cirrhosis of liver, HGA was present in 33 (94.3%) and low gradient ascites in 2 (5.7%), as summarised in Table-1. 22 (68.8%) of these patients had transudative ascites while in 13 (37%) the ascites was exudative. The sensitivity, specificity, PPV and NPV of HGA and transudative ascites in predicting the diagnosis of PHT is shown in Table-2. In 2 patients with cirrhosis of liver with spontaneous bacterial peritonitis, ascitic fluid was exudative with high SAAG.

Out of 9 patients with disseminated malignancy, ascitic fluid was exudative in 8 and transudative in 1. The SAAG was high in 5 and low in 4. In all patients with peritoneal tuberculosis, pancreatic ascites and biliary peritonitis, the ascitic fluid was exudative with low SAAG. In congestive cardiac failure, the ascitic fluid was transudative with high SAAG while in nephrotic syndrome it was transudative with low SAAG.

Discussion

Most patients with cirrhosis of liver and ascites have AFTP less than 2.5 gm%. However, ascites may be exudative in uncomplicated cirrhotics and those with peritonitis, peritoneal tuberculosis or on diuretic therapy [4]. In our study only 63.8% of patients with uncomplicated cirrhosis had transudative ascites. The sensitivity of transudative ascites in diagnosing PHT was only 62.9%. On the contrary, HGA could predict the presence of PHT with a high sensitivity of 94.3%. The specificity of HGA for diagnosis of PHT was 60%. This was because 6 patients had HGA in the absence of demonstrable PHT. The cause of ascites in them was disseminated malignancy in 5 and congestive cardiac failure in 1. Runyon et al found that the accuracy of HGA in predicting PHT approached 97% [5]. It has also been shown that the value of SAAG correlates with the portal pressure [6]. Portal decompression by TIPS resulted in reduction of SAAG from mean of 1.9-0.5gm% to 1.7-0.5gm% within 6 hours, thus indicating that the value of SAAG reflects the portal pressure [7]. Also it has been shown that a value of SAAG greater than 1.435 is invariably associated with the presence of esophageal varices [8]. While transudative ascites in a cirrhotic may get converted into an exudate with diuretic therapy or in the presence of peritonitis and peritoneal tuberculosis, the ascites remains high gradient [9]. However, it is impor-

tant to remember that the value of SAAG may be falsely low in patients with cirrhotic ascites if there is marked hypoalbuminaemia, hypergammaglobulinaemia (more than 5gm%), hypotension or chylous ascites.

Patients with malignant ascites usually have exudative ascites due to peritoneal carcinomatosis or lymphatic blockage. However, if the serum proteins are markedly diminished due to malnutrition, the AFTP may be less than 2.5 gm%. Low SAAG is not a sensitive predictor of malignant ascites. Upto 38% of patients with malignant ascites could have HGA because of PHT as a result of massive hepatic metastasis or portal vein thrombosis [10].

Patients with peritoneal tuberculosis, pancreatitis and biliary ascites have low gradient ascites which is exudative. SAAG has superior discriminating power as compared to the transudate-exudate concept in the diagnosis of ascites due to PHT. At the same time, due to the fallacies associated with SAAG in the diagnosis of malignant ascites and some patients with PHT, the classification of ascites based on AFTP should not be abandoned.

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