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Spontaneous Bacterial Peritonitis: Diagnostic Importance of Ascitic Fluid Polymorphonuclear Cell Count, Biochemical and Microbiological Analysis

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Abstract

Introduction: Ascites represents the pathological collection of fluid in the peritoneal cavity. Spontaneous bacterial peritonitis is the infection of the ascitic fluid. For its diagnosis, the number of polymorphonuclear cell count from the ascitic fluid obtained by diagnostic abdominal paracentesis should equal or exceed 250 cells/cu.mm and from bacteriological cultures only one type of organism must be isolated. Biochemical analysis is also recommended. It is the most rapid and cost effective method for diagnosing cause of ascites. These patients must be treated with antibiotics aggressively as they have poor prognosis and high mortality if not treated early. *Materials and Methods:* Study was conducted at a tertiary care hospital, Haryana. All patients under went paracentesis within 24 hours of admission and were diagnosed before giving any antibiotics. Ascitic fluid was submitted in the Central Clinical Laboratory for total and differential leucocyte count, culture and biochemical investigations. *Results:* Present study included 38 patients of spontaneous bacterial peritonitis. Various clinical signs and symptoms were studied and followed by comparison statistics for all the variants. Statistical study was done to find the etiology for cirrhosis. Culture study revealed various organisms responsible for spontaneous bacterial peritonitis. *Conclusion:* Polymorphonuclear cell count in ascitic fluid without the need of positive culture is helpful in diagnosing spontaneous bacterial peritonitis. Mortality has decreased because of early diagnosis and effective treatment.

Keywords: Spontaneous Bacterial Peritonitis; Polymorphonuclear Cell Count; Serum/Ascites Albumin Gradient; Culture.

Introduction

The word ascites is of Greek origin (askos) and means bag or sac. Ascites represents the pathological collection of fluid in the peritoneal cavity. Common causes include liver cirrhosis, malignancy, congestive heart failure, tuberculosis, nephritic syndrome, pancreatic disease and dialysis. History and clinical examination provide clues to the possible aetiology of the ascitic fluid formation. Abdominal radiological and ultrasound studies can help detect small volumes of peritoneal fluid as well as assess the possible etiology of ascites. However, diagnostic abdominal paracentesis is recommended in patients presenting with new onset ascites, requiring hospitalization due to the presence of ascites. Abdominal paracentesis with appropriate ascitic fluid biochemical analysis is considered the most rapid and cost effective method for diagnosing the cause of ascites.

Spontaneous bacterial peritonitis (SBP) was first described in 1964 by Professor Harold O Conn [1]. SBP is the infection of the ascitic fluid that occurs in the absence of a visceral perforation and in the absence of intra-abdominal inflammatory focus such as abscess, acute pancreatitis or cholecystitis. SBP is a common complication of decompensated liver disease in North India and is associated with significant in-hospital mortality [2]. Sign and symptoms of SBP include fever, chills, nausea, vomiting, abdominal tenderness, general malaise and hepatic encephalopathy. In about 10% of SBP patients no signs or symptoms are present [3].

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For SBP diagnosis, the number of Polymorphonuclear (PMN) cell count from the ascitic fluid obtained by paracentesis should equal or exceed 250 cells/cu.mm and from bacteriological cultures only one type of organism must be isolated. These patients should be treated with antibiotics aggressively as they have poor prognosis and high chances of mortality if not treated early. All patients need investigations for the cause of ascites even when cirrhosis is suspected. Ascitic fluid should be sent for the determination of albumin or protein concentration, cytology and culture.

The difference between the serum and ascitic albumin concentration (serum/ascites albumin gradient - SAAG) was used to differentiate ascitic fluid into two categories: high gradient ≥1.1 g/dl in cases with portal hypertension and low gradient < 1.1 g/dl in ascites unrelated to portal hypertension. Both SAAG and ascitic fluid protein concentration are recommended for the initial evaluation of ascitic fluid in cirrhosis, proposed by the American Association for the Study of Liver Disease (AASLD) and British Society of Gastroenterology. Decreased ascites protein concentrations are associated with high risk for developing SBP. After differentiation of ascites into two broad categories, specific biochemical analyses can be useful for further evaluation of ascites etiology. All ascitic fluid samples should be screened for the development of SBP.

Based on the results of absolute white cell count and culture of the ascitic fluid, five variants of ascitic fluid infection have been recognised [4,5]. These are:

1. Classical SBP (CSBP): Defined as an ascitic fluid (AF) infection associated with a positive bacterial culture showing a single type of bacteria and an ascitic fluid PMNL cell count of ≥ 250 cells/cu. mm in the absence of a surgically treatable intra-abdominal source of infection.
2. Culture-negative neutrocytic ascites (CNNA): Diagnosed when cultures of ascitic fluid is negative, PMNL cell count is >250 cells/cu. mm.
3. Monomicrobial nonneutrocytic bacterial ascites (MNB): Characterized by the isolation of only one type of bacteria in cultures of ascitic fluid and PMNL cell count of <250 cells/cu. mm.
4. Secondary bacterial peritonitis: Defined as an ascitic fluid infection associated with a positive bacterial culture showing polymicrobial and an ascitic fluid PMNL cell count of ≥250 cells/ cu. mm in the presence of a surgically treatable intra-abdominal source of infection.
5. Ascitic Fluid infection associated with a positive bacterial culture showing polymicrobial and an ascitic fluid PMNL cell count of < 250 cells/cu. mm.

Clinical signs and symptoms do not distinguish secondary bacterial peritonitis from SBP. However, the ascitic fluid analysis is helpful in this regard. Ascitic fluid in secondary bacterial peritonitis usually meets at least 2 of the following criteria: Total protein content of <1 g/dl, glucose concentration of <50mg/dl, and lactate dehydrogenase (LDH) level of >225 U/L (or higher than the upper limit of normal for serum). A high index of suspicion followed by analysis of ascitic fluid for evidence of infection is helpful in making an early diagnosis of SBP and is today considered the standard of care in patient with cirrhosis and symptoms. The International Ascites Club recommends mandatory analysis of ascitic fluid in all cases of new onset of ascites, worsening of ascites and all the other cases whenever there is a suspicion of SBP [4].

The recommendations of the American Association for the study of Liver Disease (AASLD) differ from those of the International Ascites Club. The AASLD thus recommends testing of ascitic fluid for cell count and differential count, but not necessarily culture, for patients undergoing serial outpatient therapeutic paracentesis, each time the fluid is removed [6].

PMN cell count in ascitic fluid ≥250 cells/cu. mm is the gold standard criterion for SBP diagnosis. Although not specific, it is a highly sensitive indicator of SBP. PMN cell count in ascitic fluid ≥ 500 cells/cu. mm is considered specific for the diagnosis of SBP [6,7].

Because of poor prognosis, antibiotic treatment must be instituted immediately in all patients with suspected SBP without waiting for microbiology test results [4].

Materials and Methods

Study was conducted in the Central Clinical Laboratory in collaboration with clinical department at a tertiary care hospital, Haryana. It was a prospective analytic study. The patients included in the study were subjected to detailed work up including history of present and past illness along with all the required investigations. All patients under went paracentesis within 24 hours of admission and were diagnosed before giving any antibiotics. About 30 ml of ascitic fluid was tapped in each patient with all the aseptic precautions.

1. 10 ml of ascitic fluid was immediately inoculated into the blood culture bottle at the bed side for

microbiological analysis.

2. 10 ml of ascitic fluid was sent for biochemical examination.
3. 10 ml of ascitic fluid was sent for cytological examination [Total leucocyte count (TLC), Differential leucocyte count (DLC), and malignant cells].

Data collected was analysed on Microsoft Excel sheet and all the patients were compared with each other regarding age, sex, type and duration of symptoms. Detail history of alcohol addiction, intravenous drug intake, blood transfusion, and jaundice was taken for each patient. Cause of ascites and various radiological, biochemical, cytological and microbiological reports of ascitic fluid were studied.

Aims and Objectives

To study the profile of SBP according to ascitic fluid culture and neutrophil count.

Inclusion Criteria

All patients were above 18 years of age.

All indoor patients of ascites with high gradient (High SAAG = 1.1 or > 1.1) and low protein (ascitic fluid protein < 2.5 gm/dl) were included.

Exclusion Criteria

Known case of malignancy, tuberculosis, congestive heart failure, patients already on antibiotics, and patients with secondary peritonitis.

Results

A total of 117 patients with ascites were analysed. A total of 90 patients were studied thoroughly with regard to history and clinical examination, haematological, cytological, microbiological, and biochemical tests and had high gradient and low protein ascites. Rest 52 were excluded due to exclusion criteria. The study included 38 patients in SBP. Out of these 38 cases of SBP, 19 patients were of CSBP, 16 of CNNB, and 3 of MNB (Table 1).

Considering the age maximum numbers of patients with Classical SBP were found in the age group of 40-49 years. Patients with CNNA were in the age group of 40-49 years. Maximum numbers of MNB patients were in the age group of 30-59 years (Table 2).

According to sex distribution, amongst 38 patients with high gradient and low protein ascites 34 (89.47%) were male and 4 (10.52%) were female. Male and female ratio was 8.5:1. Amongst the total 38 patients of SBP, CSBP was present in 19 (50%) patients of these 17 (89.47%) were male and 2 (10.52%) were female. CNNB was present in 16 (42.10%) patients of these 15 (93.75%) were male and 1 (6.25%) was female. MNB was present in 3 (7.89%) patients of these 2 (66.66%) were male and 1 (33.33%) were female. (Table 3)

Considering the clinical signs and symptoms, all the 38 patients studied in this series had moderate to severe ascites. Flank dullness and shifting dullness was present in 38 (100%) patients. Icterus was observed in 30 (78.94%) of cases. Fever was present in 22 (57.89%). Pedal edema was seen in 14 (36.84%) cases. Hepatomegaly was seen in 3 (7.89%) of cases. Abdominal tenderness in 12 (31.57%), encephalopathy in 7 (18.42%) and upper gastrointestinal bleeding was seen in 10 (26.31%) patients.

Amongst the 38 patients of SBP, total patients in CSBP group were 19 and all 19 (100%) of them presented with abdominal distension, icterus, flank dullness, and shifting dullness. Pedal edema was present in 9 (47.36%), fever in 14 (73.68%), abdominal tenderness in 4 (21.05%), upper gastrointestinal bleeding in 4 (21.05%), hepatomegaly in 1 (5.26%) and encephalopathy was present in 4 (21.05%).

Total patients in CNNA group were 16 and all 16 (100%) of them presented with abdominal distension, flank dullness, and shifting dullness. Icterus was observed in 9 (56.25%), fever was present in 7 (43.75%), pedal edema was present in 5 (31.25%), abdominal tenderness in 8 (50%). Upper gastrointestinal bleeding was present in 5 (31.25%), hepatomegaly in 2 (12.5%) and encephalopathy was present in 3 (18.75%).

Total patients in MNB group were 3 and all 3 (100%) of them presented with abdominal distension, flank dullness, and shifting dullness. Icterus was observed in 2 (66.66%). Upper gastrointestinal bleeding was present in 1 (33.33%). Fever, pedal edema, hepatomegaly, abdominal tenderness and encephalopathy were not present in all the 3 cases (Table 4).

Considering the investigations, in SBP the mean haemoglobin and TLC values were 8.01 gm/dl and 10,850 cells/cu.mm respectively. Total bilirubin , serum protein and serum albumin were 5.69 mg/dl, 5.37 gm/dl, 2.13 mg/dl respectively. Mean of SGOT, SGPT and serum creatinine were 170 IU/L, 127.84 IU/L and 1.65 mg/dl respectively.

On analysis of ascitic fluid, the mean ascitic fluid cell count (PMN cell count), total protein, prothrombin time (PT) and INR were 974 cells/cu.mm, 1.27 gm/dl,

19.89 seconds and 1.84 respectively.

Mean of ascitic fluid cell count (PMN count) in CSBP, CNNA, and MNB were 1,353 cells /cu.mm, 734 cells/ cu.mm, and 244 cells/cu.mm respectively.

Aetiology for cirrhosis in all the 38 SBP patients with ascites, was mainly alcoholic liver disease 35 (92.10%) followed by HBV alone 1 (2.63%), HCV alone 1 (2.63%) and combined HCV and alcoholic liver disease 1 (2.63%).

Out of total 19 patients of CSBP, 17 (89.47%) were due to alcoholic liver disease. Remaining 2 cases, 1 (5.26%) each occurred due to HBV and HCV alone. Out of total 16 patients of CNNA, 15 (93.75%) were due to alcoholic liver disease. One (6.25%) was due to alcoholic liver disease and HCV. Out of 3 patients of MNB, 3 (100%) were due to alcoholic liver disease (Table 5).

Out of 38 patients of SBP, 16 (42.10%) had sterile ascitic fluid but had PMNL counts >250 cells/cu mm, 18 (47.36%) patients had Escherichia coli on culture of ascitic fluid. Two (5.26%) patients had Staphylococcus aureus. One (2.63%) patient culture revealed Klebsiella. Pseudomonas was isolated in 1 (2.63%) patient. Out of 19 patients of CSBP 17 (89.47%) had Escherichia coli on culture. One (5.26%) Staphylococcus aureus, and 1 (5.26%) Klebsiella. Out of 16 patients of CNNA, all 16 (100%) were sterile on culture. Out of 3 patients of MNB, 1 (33.33%) each

Table 1: Variant of SBP based on PMN cell count (cut of 250 cells/cu.mm) and culture result

PMN cell count (Cut of 250/cu.mm)	Culture	Total (N=38)	Variant of SBP
≥ 250	Positive	19	Classical Spontaneous bacterial peritonitis (CSBP)
>250	Negative	16	Culture negative neutrocytic ascites (CNNB)
<250	Positive	3	Monomicrobial non neutrocytic bacterial ascites (MNB)

Table 2: Age distribution of SBP patients in its 3 variants

Age (years)	SBP N (%)	CSBP N (%)	CNNA N (%)	MNB N (%)
20-29	0	0	0	0
30-39	6 (15.79)	2 (10.53)	3 (18.75)	1 (33.33)
40-49	14 (36.84)	7 (36.84)	6 (37.5)	1 (33.33)
50-59	7 (18.42)	4 (21.05)	2 (12.5)	1 (33.33)
60-69	8 (21.05)	4 (21.05)	4 (25)	0
70-79	1 (2.63)	1 (5.26)	0	0
80-89	2 (5.26)	1 (5.26)	1 (6.25)	0
Total	38 (100)	19	16	3

Table 3: Sex distribution of patients

Sex	SBP N (%)	CSBP N (%)	CNNA N (%)	MNB N (%)
Male	34 (89.47)	17 (89.47)	15 (93.75)	2 (66.66)
Female	4 (10.52)	2 (10.52)	1 (6.25)	1 (33.33)
Total	38 (100)	19 (50)	16 (42.10)	3 (7.89)

Table 4: Clinical signs and symptoms

Sign	SBP=38 N (%)	CSBP=19 N (%)	CNNA=16 N (%)	MNB=3 Nn (%)
Ascites	38 (100)	19 (100)	16 (100%)	3 (100)
Flank Dullness	38 (100)	19 (100)	16 (100%)	3 (100)
Shifting Dullness	38 (100)	19 (100)	16 (100%)	3 (100)
Icterus	30 (78.95)	19 (100)	9 (56.25%)	2 (66.67)
Fever	22 (57.89)	14 (73.68)	7 (43.75%)	0
Pedal oedema	14 (36.84)	9 (47.37)	5 (31.25%)	0
Upper gastrointestinal Bleeding	10 (26.31)	4 (21.05)	5 (31.25)	1 (33.33)
Hepatomegaly	3 (7.89)	1 (5.26)	2 (12.5)	0
Abdominal tenderness	12 (31.58)	4 (21.05)	8 (50)	0
Encephalopathy	7 (18.42)	4 (21.05)	3 (18.75)	0

Table 5: Cause of Cirrhosis

Causes	SBP=38 N (%)	CSBP=19 N (%)	CNNA=16 N (%)	MNB=3 N (%)
Alcoholic Liver Disease	35 (92.11)	17 (89.47)	15 (93.75)	3 (100)
Alcoholic liver disease + Hepatitis B virus (HBV)	0	0	0	0
Alcoholic liver disease + Hepatitis C virus (HCV)	1 (2.63)	0	1 (6.25)	0
Hepatitis B virus (HBV)	1 (2.63)	1 (5.26)	0	0
Hepatitis C virus (HCV)	1 (2.63)	1 (5.26)	0	0

revealed Escherichia coli, Staphylococcus aureus and Pseudomonas growth.

Discussion

Ascites is a common clinical problem and is one of the cardinal manifestations of many diseases. Most common cause of ascites is cirrhosis of liver. The most frequent infectious complication in patients with cirrhosis of the liver is SBP, occurring with a prevalence of 10–35%, followed by urinary tract infection, pneumonia and bacteremia [8].

Cause of ascites can be suspected from history and examination, but ascitic fluid analysis is an important investigation for the diagnosis of its aetiology. The present study revealed that in our region cirrhosis of liver is the commonest cause of ascites and alcoholism (92.11%) is most common cause of cirrhosis of liver. Incidence of Hepatitis B and Hepatitis C was low because intravenous drug abuse and high risk behaviour is possibly less in our region. It is comparable to the study done by R Maskey et al [9]. Nepal, in which most common cause was alcohol related cirrhosis. In a study by Mumtaz Ali Sheikh et al. [10] in Lakana, Pakistan, out of 128 (85.33%) patients of high SAAG, 122 (81.33%) were of viral hepatitis B, C and combined 105 (70%), alcoholic 7 (4.66%), cryptogenic 10 (6.66%).

Age distribution in our study varied from 30-85 years, SBP was seen predominantly in age group 40-49 year, and mean age at the time of diagnosis was 57.5 years. Mean age at the time of diagnosis in a study by Filik L et al [11] was 49.9 years and 39 years in MK Bhatnagar [12] series.

In our study 34 (89.47%) patients were males and 4 (10.52%) were females, with a male and female ratio was 8.5:1. It was comparable to the study by Syed et al [13] in Nepal. Dilshad Muhammad et al. [14] studied 50 patiens of cirrhosis wih ascites of which 27 (54%) were males and 23 (46%) were females. Males were affected more possibly due to more consumption of alcohol in the male counterpart.

Common mode of presentation of SBP in our study was jaundice 30/38 (78.94%), fever in 22/38 (57.89%), abdominal pain was present in 12/38 (31.57%) and encephalopathy was present in 7/38(18.42%), which is at variance from the study by Mihas AA et al [15] in which fever was found in 69%, abdominal pain in 59%, abdominal tenderness in 49% and encephalopathy was seen in 54%.

PMN cell count in ascitic fluid ≥ 250 cells/cu. mm is the gold standard criterion for SBP diagnosis. Although not specific, it is a highly sensitive indicator of SBP [7]. PMN cell count in ascitic fluid ≥ 500 cells/cu. mm is considered specific for the diagnosis of SBP. According to Moore et al [16] due to poor prognosis and high mortality, aggressive antibiotic treatment must be instituted immediately for all the patients with suspected SBP without waiting for microbiology test results [6,17]. Earlier studies [18] showed 80-100% mortality from SBP, later studies [19] had significantly reduced mortality 10% and the present study had no mortality. Mortality has decreased because of the early diagnosis and effective treatment. Most common organisms implicated in SBP are Escherichia coli. Escherichia coli and Staphylococcus aureus were the two common organisms isolated in our setup similar to Lata J et al [7] According to Runyon BA et al [20] Escherichia coli was responsible for 27.3% of cases of SBP and Staphylococcus aureus for 6.8%. While Wilcox et al [21] demonstrated Escherichia coli as responsible in 45% and Staphylococcus in 12% cases.

The patients should be put on empirical therapy pending the results of fluid analysis to reduce the mortality. Patients with sterile ascitic fluid were 42.10% but had PMN cell counts >250 cells/cu. mm. A positive culture is not necessary for diagnosing SBP and diagnosis can be made based upon the cell count alone even if the culture is negative.

Conclusion

PMN cell count in ascitic fluid ≥250 cells/cu. mm is the gold standard criterion for SBP diagnosis. A positive culture is not necessary for diagnosing SBP

and diagnosis can be made based upon the PMN cell count alone even if the culture is negative. Mortality has decreased because of the rapid and early diagnosis along with an effective treatment.

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