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YIELD OF ASCITIC FLUID CULTURES IN SPONTANEOUS BACTERIAL PERITONITIS IN CIRRHOSIS

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ABSTRACT

Objectives: To see the bacterial yield of ascitic fluid cultures in spontaneous bacterial peritonitis in cirrhosis liver patients with ascites and to know about the most common organism causing spontaneous bacterial peritonitis.

Material and Methods: This study was conducted in the department of medicine Khyber Teaching Hospital, Peshawar between July 2000 and June 2001. Fifty patients with cirrhosis liver having ascites and suspected spontaneous bacterial peritonitis (SBP) were included in this study. Apart from the baseline tests, diagnostic paracentesis was performed in all patient at admission before starting antibiotics. Ascitic fluid (10 ml) was inoculated in culture bottles followed by Sub cultures, identification of bacteria and antibiotic sensitivity.

Results: Out of 50 patients, 20(40%) were Hepatitis B surface antigen (HBs Ag) positive, 28(58%) were positive for anti hepatitis C antibodies and 2(4%) were positive for both. Twenty-nine (58%) cases were found to have bacterial peritonitis. Nineteen (65.51%) patients were anti HCV positive, 8(27.58%) were HBs Ag positive and 2(6.89%) were having both. Patients were divided into three groups. Eleven (37.93%) patients had classical SBP, 16(55.17%) had Culture Negative Neutrocytic Ascites and 2(6.89%) had bacterascites. Cultures were positive in 13(44.82%) patients. Escherichia coli(61.55%) was the commonest organism isolated, streptococcus in 2(15.38%) cases and one(7.69%) each was Staphylococcus, Klebsiella and Acinetobacter.

Conclusion: Spontaneous bacterial peritonitis is a common complication of cirrhosis liver with ascites. It is easily overlooked and needs high clinical suspicion for diagnosis. Ascitic fluid culture is the best method for diagnosis of SBP but is positive in less than half of cases.

Key words: Spontaneous bacterial peritonitis, cirrhosis liver, ascitic fluid, cultures.

INTRODUCTION

Cirrhosis liver is a final common pathway of various types of advanced chronic liver injury. It is mostly post hepatitic in our region due to high prevalence of chronic hepatitis B and C viral infections in contrast to western populations where it is mostly alcoholic and cryptogenic[1,2,3]. Spontaneous bacterial peritonitis (SBP) is a frequent and severe complication of cirrhosis liver with ascites[4,5,6]. Recent studies have shown that SBP is more common than previously thought among patients admitted to the hospital with cirrhotic ascites[7]. Caroli reported first case of SBP in 1958[8]. The prevalence of SBP in Pakistan has been reported to be 32-64%[9]. SBP is defined as bacterial peritonitis that occurs in cirrhotic patients with ascites in the absence of any recognized intra-abdominal surgically treatable source of infection[10]. SBP may present as sudden onset of fever with chills, diffuse abdominal pain, rebound tenderness and absent bowel sounds. In some patients the symptoms may be minimal and the presenting features in these patients may be jaundice, encephalopathy or hypotension without localized abdominal signs. SBP most commonly is the main consequence of bacterial translocation across the intestinal mucosal barrier leading to bacteremia and ascitic fluid inoculation. There are some proposed mechanisms which explain bacterial translocation in cirrhosis: the intestinal bacterial overgrowth, the structural and functional alterations of the intestinal mucosal barrier and the defiencies of the local immune response[11,12]. Low ascitic fluid albumin and other proteins impair the opsonising activity and thus decrease the bactericidal activity of ascitic fluid[13,14]. In about 90% of cases the infection is monomicrobial and organisms are usually of intestinal origin[15]. Aerobic Gram-negative organisms are the predominant bugs but 25% of infections are due to Gram-positive organisms. The purpose of this study was to see firstly the yield of ascitic fluid cultures in SBP in cirrhosis liver patients with ascites and secondly to know about the most common organism causing SBP.

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MATERIAL AND METHODS

This study was conducted in the department of medicine Khyber Teaching Hospital (KTH), Peshawar

from July 2000 to June 2001. It included 50 patients of cirrhosis liver having ascites with a clinical suspicion of SBP admitted to medical wards of KTH. Patients who fulfilled the following criteria were included in the study:

1. Patients presenting with fever, chills, abdominal pain, recent increase in abdominal distension, confusion or coma, rebound tenderness or signs of hepatic encephalopathy.
2. Ascitic fluid polymorphonuclear (PMN) cells count greater than 250 cells/mm^3.
3. Ascitic fluid total leukocytic count greater than 500 cells/cmm.
4. Ascitic fluid PMN cells count less than 250 cells/cmm but having clinical features suggestive of SBP.

Those patients who had non-cirrhotic ascites or had received antibiotics in the last 72 hrs, and those patients who had an infection of any other system were excluded from the study. Detailed history was obtained from each patient and a thorough clinical examination was performed. Diagnosis of cirrhosis liver with ascites was clinical supported by appropriate investigations. Histological confirmation for the presence of cirrhosis was not considered essential for inclusion in this study. Particular emphasis was laid upon fever, chills, abdominal pain and tenderness, deepening of jaundice, hypotension, edema, collateral vessels, rebound tenderness, hepatosplenomegaly, hepatic flap and confusion or coma. Diagnostic paracentesis was performed on every patient on admission to medical unit before starting them on antibiotics. This procedure was done using "Z" approach under aseptic conditions. About 10 ml of the ascitic fluid was immediately inoculated into a commercially available blood broth bottle (Biphasic Culture System) at bedside for bacterial culture and sensitivity and 10 ml was sent for routine biochemical and cytological examination and Gram staining. Sub-cultures were made from the sample for antibiotic sensitivity after identification of bacteria. Data collected was analyzed using SPSS version 10.

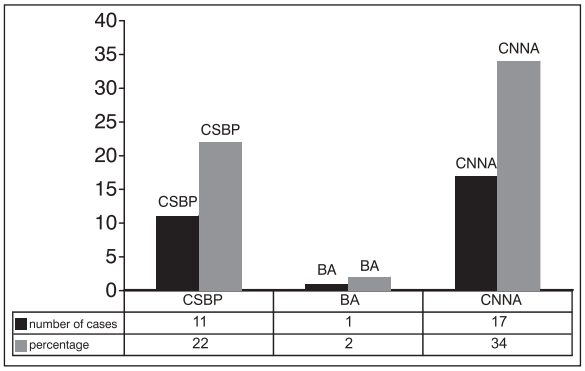
RESULTS

Among the 50 patients who fulfilled the criteria for inclusion, 27(54%) were male and 23 (46%) female. Most of them were in the age group of 40-60 years. Twenty (40%) patients were positive for HBs Ag, 28(56%) had positive anti HCV antibodies and 02(4%) patients were positive for both. SBP was found to be present in 29(58%) cases. Of these patients, 08(22.58%) were HBs Ag positive, 19(65.51%) were anti HCV antibody positive and 2(6.89%) had evidence of both hepatitis B and C infection. Patients having SBP were divided into different sub groups that are shown in Fig. 1. The commonest signs and symptoms in patients with SBP were fever, abdominal pain, abdominal tenderness and jaundice (Fig. 2). Biochemical examination data of patients with and without SBP are shown in Table 1. The total leukocyte count in SBP group was significantly increased as compared to non SBP group (p-value 0.000764). Same was the case with total leukocyte and polymorphonuclear cells count in the ascitic fluid (p-value 0.000063). ESR in patients with SBP was 12 mm/1^{st} hour higher than the other group making it a significant (p-value 0.0028) marker for detecting SBP in patients with cirrhosis liver and ascites. Prothrombin time was also significantly prolonged in patients having SBP as compared to the non SBP group (p-value 0.0303). Ascitic fluid cultures were positive in 13(44.82%) patients out of 29 cases of SBP. Eleven (37.93%) patients were having classical SBP, 02(6.89%) were in the bacterascites group while

Table 1: Biochemical data in SBP and non-SBP patients

Variable	SBP	Non-SBP	P value
Total Leucocyte Count	10510.341±1336.39/mm^3	8952.38±1136/mm^3	0.0000764*
ESR	55.44±16.23mm/1^{st}hr	43.85±5.16mm/1^{st}hr	0.0028*
Serum Bilirubin	5.00±1.44mg/dl	3.2±1.31mg/dl	0.00004*
Serum Protein	5.4±0.97gm/dl	5.68±1.21gm/dl	0.3615
Serum Albumin	2.39±0.65gm/dl	2.71±0.69gm/dl	0.1049
Ascitic Fluid Albumin	1.00±0.34gm/dl	1.51±0.52gm/dl	0.000157*
Ascitic FluidTLC	2587.89±85.18/cmm	106.8±58.39/cmm	0.0000631*
Ascitic FluidPMN Count	2052.41±206.83/cmm	25.83±36.27/cmm	0.000044**
ProthrombinTime (in Seconds)	15.24±14.77 seconds	7.52±6.63 seconds	0.0303*

Data Expressed as Mean ± SD, *Statistically Significant, TLC = Total Leucocytic Count, PMN =Polymorphonuclear, ALT = Alanine Aminotransferase



CSBP = Classical Spontaneous Bacterial Peritonitis.

BA = Bacterascites CNNA, = Culture Negative Neutrocytic Ascites

Fig. 1: Subgroups of patients with SBP.

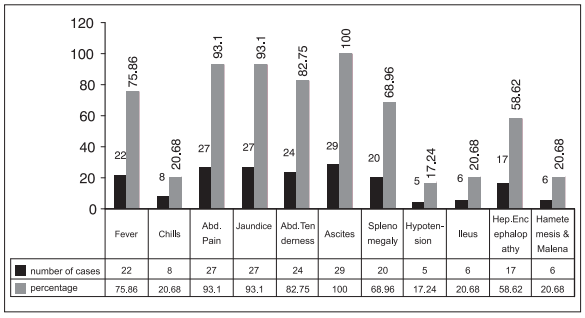


Fig. 2: Clinical presentation of patients with SBP.

16(55.17%) patients were of Culture Negative Neutrocytic Ascites. Escherichia coli (E. coli) was the predominant organism isolated which accounted for 61.55% of cases. Streptococcus was isolated in 02(15.38%) cases and staphylococcus, Klebsiella and acinetobacter each in one (7.69%) case. All the organisms recovered were sensitive to Quinolones and 3rd generation cephalosporins while some of the organisms were also sensitive to other antibiotics like amoxicillin, sulphonamides, piperacillin, gentamycin and tobramycin. Six (20.68%) patients having SBP died during hospitalization and all of them were in Child grade C. Four (13.79%) of them had hematemesis and melena and 02(6.89%) had developed hepato-renal syndrome.

DISCUSSION

SBP is a common and potentially fatal complication of cirrhosis liver[4,5,6]. It is commonly overlooked in seriously ill patients leading to high mortality in patients with cirrhosis liver, therefore needs high index of suspicion for early diagnosis and treatment[6]. Cirrhosis liver is a common problem in our set-up due to high prevalence of hepatitis B and C virus infections[1, 2, 3]. In this study majority of cases were due to viral hepatitis with Hepatitis C accounting for 56% of cases. Studies by Hamzullah Khan et al[1], Shah HA et al[16] and Khan TS et al[17] have also reported similar findings. Other studies have reported hepatitis B as the most common cause of cirrhosis liver[18]. The SBP rate of this study (58%) correlates with the study conducted by Munib S (58%) and Memon AQ et al[19] (64%), but is higher than in the studies conducted abroad by Rimola A et al[20,] Coral G et al[21] and Strauss E et al[22], who have reported SBP rate of 10-30%. The reasons for increased frequency of SBP in this study may be due to the low socio-economic conditions, malnutrition, high prevalence of infectious diseases and above all the technique of bedside ascitic fluid culture collection in commercially available blood culture bottles (Biphasic culture System). This culture system has been reported by Runyon to have a sensitivity of 91%[23]. Munib S study shows a sensitivity of 34.5% and Memon AQ Et al study shows 81.25%[18,19]. Cultures were positive in 13 (44.82%) out of 29 cases of SBP. Eleven (37.93%) cases were of classical SBP group and two (6.89%) cases were of mono microbial non-neutrocytic bacterascites group. Sixteen (55.17%) cases were those of Culture negative neutrocytic ascites (CNNA). The frequency of bacterascites and CNNA by Memon AQ et al[19] has been reported as 25% and 18.75% respectively. Rajput MR et al [24] has reported bacterascites in 3.5% and CNNA in 62% cases. Munib S[18] has reported it in 3.44% and 66.6% cases respectively. The prevalence of bacterascites has been reported in 8% of SBP patients by Qurban Hussain et al[25]. The low culture positivity in our study may be because of small sample size, improper collection technique or not using the more recent and improved method of (Bact Alert test)[26]. The differences can also be attributed to host factors like the general health status and causative organisms. The most common organism found was E coli (61.53%).Runyon BA[27] has reported E-Coli in 60% cases. Streptococcus was found in (15.38%) and staphylococcus, Klebsiella and acinetobacter (each in 7.69% cases). Memon AQ et al[19] and Rajput MR et al[24] studies show almost the same results. Gines P et al[28] and Thalheimer U et al[29] have reported G-positive Cocci in 25% cases. Portal hypertension, impaired gastrointestinal transit with bacterial overgrowth, functional mucosal damage and increased gut permeability to bacteria all lead to increased bacterial translocation to the lymphatic system and portal vein[11,12]. This is coupled by the impaired activity of reticuloendothelial system, decreased complement levels and diminished opsonic activity of ascitic fluid[13,14]. Ascitic fluid polymorphonuclear cell count is an easy and single best test in establishing the diagnosis of SBP regardless of the fact whether a discriminative value of 250 or 500/cmm is used[30]. It is a more reliable index of SBP than the ascitic fluid pH, lactate or their blood-ascitic fluid gradient[31]. Other studies[32] have also shown significant increase in the PMN cell count as was the case in our study. Though ascitic fluid culture is the gold standard method for the diagnosis of SBP but we should not ignore the value of ascitic fluid PMN cell count as we may miss all cases of CNNA.

CONCLUSION

High clinical suspicion is needed for the diagnosis of SBP as it can easily be missed in seriously ill patients. Ascitic fluid culture is the best method for diagnosis of SBP. Keeping in view the lower rate of culture positivity the ascitic fluid PMN cell count should be used as a primary tool for the diagnosis of SBP so that we are able to detect cases of culture negative neutrocytic ascites (CNNA).

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