# Frequency Of Spontaneous Bacterial Peritonitis in Chronic Liver Disease

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**Background:** Spontaneous bacterial peritonitis (SBP) is defined as infected ascites in the absence of any recognizable secondary cause of infection.

**Objectives:** To find out the percentage of SBP in patients of chronic liver disease with ascites, its clinical and laboratory characteristics.

Place and Duration of Study: Medical ward II, Jinnah Hospital lahore and duration of study was six months.

**Study Design:** Descriptive/ Observational study.

**Subjects and Method:** One hundred patients of chronic liver disease with ascities were included in this study. Diagnostic paracentesis was performed immediately upon admission and the bacterial cultures and biochemical analysis was done on sample.

**Results:** Ascitic fluid examination was the main basis for establishing the diagnosis of SBP. It was found that SBP was present in 22 patients, out of which 11 were culture positive and 11 were culture negative, 35 patients were HBsAg positive and 65 patients were anti HCV positive. SBP was found in 22 patients out of whom 10 were males and 12 females. Ascites was present in all the patients (100%). Shifting dullness was present in 76 (76%) cases, fluid thrill in 24 cases (24%) and tenderness in 47 patients (47%).

**Conclusion:** Frequency of SBP is quite high in patients with chronic liver disease with ascites. SBP should be suspected in all such cases presenting with typical or atypical features.

**Key Words:** Spontaneous bacterial Peritonitis (SBP), Ascites, Cirrhosis.

# Introduction

Ascites is the most frequent complication of cirrhosis and is associated with increased susceptibility to infections and poor prognosis. Spontaneous bacterial peritonitis (SBP) is characterized by spontaneous infection of ascitic fluid in the absence of any intra abdominal source of infection. Its prevalence ranges between 10 to 30 percent of patients. Most organism causing SBP are gram negative bacteria of enteric orgin.Out of which 50.0% are E.coli and 1/5<sup>th</sup> are aerobic gram positive organisms (19%). The criteria for making the diagonosis of SBP are:

- 1. Classical SBP:Patients with typical signs and symptoms, ascitic fluid polymorphonuclear count (AFPMN) > 250 cells/mm and positive gram stain and culture.
- Culture negative neutrocytic ascites (CNNA): Patients with minimum clinical symptoms but AFPMN count > 500 cells / mm and culture is negative plus negative gram staining.
- 3. Bacterascites: Patients with few clinical symptoms, AFPMN cell count < 250 / mm gram stain plus bacterial culture positive. Since SBP is associated with poor prognosis in patients with chronic liver disease. Owing to the morbidity of the condition we wanted to find out through our study frequency of SBP different modes of its presentation.

# **Material and Methods**

After informed consent the subjects were recruited upon

there presentation to outpatient or emergency department. Patients of all ages with chronic liver disease were included. Those having had a peritoneal paracentesis within last 2 weeks cardiac reasons and secondary peritonitis were excluded.

# **Procedure:**

After taking the informed consent diagonstic paracentesis was performed during first 24 hrs of hospitalization and 50cc of ascitic fluid was obtained under aseptic condition. Ten ml of fluid was injected directly into blood culture bottles and subjected to culture studies rest of the sample was subjected to the tests including TLC, DLC, biochemical analysis, cytology, Zeil Nelson staining and gram staining.

# Study design and statistical methods:

It was a prospective, observational and descriptive study of clinical cases. All the case records were collected in Microsoft excel. The statistical analysis was done with the help of SPSS 10.0 for Windows.

#### Results

It was a study of 100 patients of chronic liver disease with ascites. Out of these 100 patients 44 (44%) were males, 56 (56%) were females, 35 (35%) were HBs Ag positive and 65 (65%) were anti-HCV positive.SBP was found in 22 patients out of them 10 were males and 12 females (Table 1).

The patients showed clinical signs of anemia 84 (84%), 55 (55%) had jaundice, 9 (9%) had spider naevi, 13 (13%) had gynaecomastia, 2 (2%) had loss of chest hair, 2 (2%) had clubbing and 66 (66%) had palmer erythema.

Ascites was present in all the patients (100%). Shifting dullness was present in 76 (76%) cases, 24% had fluid thrill and tenderness was present in 17% patients. Splenomegaly was found to be present in 73 (73%), oedema feet in 71 (71%) and hepatic encephalopathy in 40 (40%) patient (Table 2). The ascitic fluid

Table 2: Clinical signs.

Signs	No.	Percentage
Anemia	84	84.0
Jaundice	55	55.0
Spider naevi	9	9.0
Gynaecomastia	13	13.0
Loss of chest hair	2	2.0
Clubbing	2	2.0
Palmer erythema	66	66.0
Ascites	100	100
Fluid thrill	24	24.0
Shifting dullness	76	76.0
Tenderness	17	17.0
Rebound tenderness	11	11.0
Splenomegaly	73	73.0
Hepatomegaly	-	-
Oedema	71	71.0
Hepatic encephalopathy	40	40.0

examination was the main basis for establishing the diagnosis of SBP. Twenty two patients were found to have SBP out of which 11 were culture positive and 11 were culture negative (Table 3). The cytological stu-dies revealed the following results. Among the patients with positive bacterial culture, ascitic fluid polymorphnuclear cell count was more than 250 mm<sup>3</sup> in all the cases. There was no case of bacterascites. Among the patients with CN-NA AFP-MNC were 700 – 1000 mm<sub>3</sub> in 4 patients (Table 3). The

Table 1:

Patient characteristics	Non SBP n = 78	SBP n = 22
Age	45 – 69 yrs	42 – 70 yrs
Sex	Male = 34 (43.89%) Female = 44 (56.41%)	Male = 10 (45.45%) Female = 12 (54.55%)
HBs Ag Anti-HCV	19 (24.35) 59 (75.64)	16 (72.73%) 06 (27.27%)
Child class	Child class A = 6 (07.69%) Child class B = 18 (23.07%) Child class C = 54 (69.23%)	Child class A = 0 (0%) Child class B = 6 (27.27%) Child class C = 16 (72.73%)

results of bacterial culture in 11 patients with SBP showed that there were 2 patients (18.18%) with Klebsiella,1 patient (9.09%) with streptococcus faecalis and 1 patients (9.09%) with a mixed growth of E coli and Klebsiella. The mean albumin concentration of ascitic fluid in patients with SBP was 1.7 g/dl. The mean serum albumin concentration in these patients was 3.2 g/dl and mean serum ascitic albumin gradient was 1.5 (Table 3). The glucose concentration in ascitic fluid of patients with SBP was 70-90 mg/dl and in patients with sterile cirrhotic ascites the glucose concentration was 110-13 mg/dl. The liver function test were abnormal in these patients. Prothombin Time and Activated Prothombin Time was prolonged in all the cases. Most of the patients were anemic and Hb was 7-9 g/dl.

Table 3: Ascitic fluid examination.

	Number	Percentage
Culture positive	11	11.0%
Culture negative	11	11.0%
CNNA Cell Count	7 (700 – 1000) 4 (1000 – 1500)	63.64% 36.36%
Serum albumin g/dl Ascitic fluid g/dl Gradient	3.2 1.7 3.2 – 1.7 = 1.5	

#### **Discussion**

SBP is defined as an infection of a previously sterile ascitic fluid without any apparent intra abdominal source of infection in patients with ascites caused by chronic liver disease. The diagnosis of SBP in this study was based upon the following two criteria.

The criterion recommended by Hoefs and Runyon.<sup>6</sup> Patients with clinical symptoms and signs suggestive of peritonitis, AFPMN cell count greater than 250/mm<sup>3</sup> and a positive bacterial culture.<sup>1</sup> The other criterion used is the one recommended by Conn and Atterbury <sup>7</sup>. Diagnosis of SBP is secure if AFPMN count is greater than 500 cells/

mm<sup>3</sup> and neutrophil count > 250 with minimal clinical features and negative bacterial culture (CNNA).

Our study included 100 patients of chronic liver disease with ascites using above criteria, a total of 22 patients were found to have SBP with a frequency of 22%. According to various studies carried out prevalence of SBP is 10 - 30%, which is well comparable with this study.<sup>2</sup>

The criterion used for making the diagnosis of CNNA were the absence of bacterial growth in ascitic fluid culture in the setting of portal hypertention and liver disease, while ascitic fluid neutrophil count was > 250 cells/mm3.8 In this study those patients who had culture negative neutrocytic ascites had few clinical features suggestive of peritonitis but when ascitic fluid was examined it had AFPMN count between 100-1500 mm³ in 36%,with neutrophil count more than 50% and when fluid was cultured techniques it did not yield any growth of microorganism.

In classical SBP patients presents with clinical features suggestive of peritonitis with AFPMN count >250 mm3 and bacterial culture positive. Same is the case in our study patients with classical SBP presented with fever in 100% cases, abdominal pain in 96%, abdominal distention in 100% cases. vomiting in 75% patients and abdominal tenderness in 96% patients. Ascitic fluid yielded AFPMN count > 250 mm<sup>3</sup> and a bacterial positive culture.

Bacterial culture results revealed growth of enteric organisms. Three fourth were gram negative bacteria and one fourth were gram positive. The isolated organisms included E.coli, klebsiella, acinetobacter and streptococcus faecalis. Our results were comparable with other international studies. 4,9 The isolated organisms were E.coli, streptococcus faecalis and klebsiella. These organisms were same except acineto bacter. Another international study revealed gram negative enteric bacteria in 76% cases. The prediction of these organisms as a cause of SBP is well recognized in most of the series. The possible reason is that although normal peritoneal fluid has antimicrobial activity for a number of organisms, ascitic fluid from cirrhosis has been shown to have impaired or deficient antimicrobial activity for E.coli, streptococcus faecalis and klebsiella. None of the organisms grew anaerobes. Chaudhry ZI, Khokhar MS 1999 have achived similar findings.<sup>4</sup> The anaerobes are rare because there is relatively high PO2 of the ascitic fluid therefore, creating a hostlile environment for their proliferation. 10

The albumin estimation revealed interesting results. The average albumin concentration in ascitic fluid of SBP patients was lower 1.7 g/dl as compared to average albumin concentration The serum albumin concentration were identical in the 2 groups i.e. 3.2g/dl. The SAAG in patients with SBP was 1.5 and was higher than in sterile ascites (1.2 g/dl) these results are identical to those of other studies. <sup>11</sup> These studies also revealed the same results with lower albumin concentration in the ascitic fluid of the patient with SBP predisposing the fluid to develop bacterial colonization. <sup>12</sup>

The protein concentration tends to be lower in SBP as

compared to sterile ascites. The low protein concentration of sterile ascites has believed to be a predisposing factor for the SBP because the opsonic activity correlates closely with fluids protein concentration, when the fluid protein levels are  $< 1 \, \text{g/dl}$  the opsonic activity is virtually non. It predisposes the fluid to bacterial colonization.

The ascitic fluid glucose concentration were lower in SBP then that in sterile ascites 90mg/dl and 110mg/dl respectively. Same is the case in the local study done by Chaudhry ZI, Khokhar MS in which patients with SBP had average glucose concentration in their ascitic fluid of 90 mg/dl<sup>4</sup>. Ascites was present in 100% cases and volume was larger as compared to the patients with sterile ascites. Presence of large amount of ascitic fluid probably predisposed to the infection. Presence of ascites is considerd to be a prerequisite of SBP. Conn and Atterbury postulate that ascites appear to be the most important factor of this syndrome, large amount of fluid reduces the chances of contact between phagocytic cells and bacteria. The case of the study of the study of the syndrome, large amount of fluid reduces the chances of contact between phagocytic cells and bacteria.

Cirrhotic ascitic fluid is also deficient in bacterial and opsonic activity compared with normal peritoneal fluid and non-cirrhotic fliud. LFTs were abnormal in all the cases. Serum bilirubin (80%), serum alkaline phosphatase (88%) and SGPT (70%).

However liver function tests are not related to SBP but rather to the underlying hepatic pathology. <sup>10,13</sup> In patients with SBP 45% were male and 54% were female while a study carried out abroad in which 214 patients were taken to evaluate the clinical and laboratory features of SBP, 69% were males and 30% were females. The current study has revealed disease is more prevalent in females in contrary to the foreign study in which percentage of male population was more. <sup>9</sup> In our study there was no patient with child class A,27% case were child class B,and 72% cases were child class C. In a local study where 50 patients were included none was child class A, 12% were Class B and 88% were class C. <sup>1</sup> So most of our results are comparable with other studies.

# Conclusion

It is concluded from this study that percentage of SBP is quite high in patients with chronic liver disease and ascites. Therefore, SBP most be suspected in all such presenting with typical or atypical features. A diagnostic paracentesis must be performed quickly within 24 hours and sample must be sent for culture and complete examination. The main idea is to start the treatment early in order to reduce the mortality rate in these patients.

# References

- Chaudry ZI, Nisar S. Ahmed U, Ali M. Short course of antibiotic treatment in SBP.J coll. physicians Surg pak 200; 10284-8.
- Such J, Review, definition, variant form, pathogensis, clinical features, diagnosis treatment, prevention and prognosis. CT infect Dis 1998; 27: 669.

- 3. Rimola A, Gracia-Tsao G Navasa M. Diagnosis, treatment and prophylaxis of spontaneous bacteria peritonitis, a consensus document.j Hepatol 2000; 32: 142-53.
- 4. Chaudhry ZI, Khokhar MS. Spontaneous bacterial peritonitis.pak Med sci 1993; 9: 277-89.
- Guarner C, Sola R, Soriano G. Risk of first community acquired spontaneous bacterial peritonitis with low ascitic fluid protein levels. Gastroenterology 199; 117: 717-9.
- 6. Hoefs JC, Runyon BA. Spontaneous bacterial peritonitis. Dis Mon 1985; 31: 9.
- Conn HO, Atterbury CE. cirrhosis management of major complication. In: Schiff ER, editprs. Diseases of the liver. 6<sup>th</sup> edition Philadelphia: JB Liponcott; 1987: 529-39.

- 8. Runyon BA, Hoefs TC. Culture negative neutrocutic ascites. A variant of spontaneous bacterial peritonitis. Hepatology 1984; 6: 1209-11.
- Filik L, Unsal S. clinical and laboratory features of spontaneous bacterial peritonitis. Afr Med J 2004; 81: 474-9.
- 10. Hallak A.SBP clinical review.Am J Gastroenterology 1989; 84:345-50.
- 11. Albillos A Cuervasmens V, Million J. Ascitic Fluid polys. cell count to serum to ascites albumin gradient in the diadnosis of bacterial peritonitis. Gastroenterol 1990; 98: 134-40.
- 12. Fleig WE Grothe W, Lotterer E, Behl S. Spontaneous bacterial peritonitis. Dtsch Med Wochenscher.
- 13. Garcia Tsao G. Spontaneous bacterial peritonitis : a historical perspective. J Hepatol 2004; 41: 522-7.