

## Clinical Profile of Spontaneous Bacterial Peritonitis in Cases of Liver Cirrhosis

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Received: 25-09-2022 / Revised: 20-10-2022 / Accepted: 07-11-2022

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Conflict of interest: Nil

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### Abstract

**Background:** As a consequence of decompensated liver cirrhosis, spontaneous bacterial peritonitis is infamous for having a high recurrence rate and is usually linked to both short- and long-term mortality in the afflicted individuals. To evaluate the prognostic indicators for the development of spontaneous bacterial peritonitis (SBP) or its variations and afterward associate the in-hospital mortality in these patients with several potential metrics, patients with SBP or its variants were compared to patients with Non-SBP Ascites.

**Methods:** Patients with CLD and ascites were gathered from Prathima Institute of Medical Sciences, Naganoor, Karimnagar. Patients' demographics, symptoms, and clinical indications were noted. Patients who had recently used antibiotics or had an infection from an intra-abdominal source were excluded. Diagnostic paracentesis was performed for the detailed report (D/R) and culture of ascitic fluid. Total leukocyte count, serum protein, and bilirubin levels were measured in blood samples.

**Results:** The predominant complaint/symptoms reported by the cases in the study included Abdominal distension in 92% of the cases followed by abdominal pain in 64% of cases, and swelling of the feet bilaterally was reported in 68% of the cases. *E. coli* was the predominant organism found in 5/10(50%) of the culture-positive SBP cases followed by *Enterococcus* species in 2/10(20%) cases, followed by *Klebsiella*, *Pseudomonas*, *CoNs* in 1/10 (10%) cases each. Ceftriaxone was the initial antibiotic given in 40% of cases followed by Cefotaxime in 18% of cases and Norfloxacin in 20% of cases.

**Conclusion:** Our research aimed to examine the prevalence of individuals with SBP or its variations as well as their diverse clinical and bacteriological profiles. Diagnostic paracentesis for D/R and C/S is crucial since it is difficult to diagnose SBP based solely on clinical signs. *Escherichia coli* (*E. coli*) is the most frequently isolated pathogen. Prompt broad-spectrum antibiotic therapy is the recommended course of treatment for SBP.

**Keywords:** Spontaneous Bacterial Peritonitis, Liver cirrhosis, Ascites, *Escherichia Coli*, Hepatorenal syndrome

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### Introduction

In a period of fewer than 10 years after receiving a cirrhosis diagnosis, ascites strike more than 50% of patients. [1, 2] One common and serious consequence in

such individuals with liver disease and ascites is spontaneous bacterial peritonitis (SBP), which is common in these patients. SBP can occur anywhere from 7% to 30% of the time in ascites patients. [3] Since SBP was initially described, a considerable drop in mortality has been seen, from 90% to 20%, as a result of improvements in diagnosis and treatment. [4] SBP has a highly varied and non-specific clinical appearance. [1] About 10% to 30% of people with SBP may even be entirely asymptomatic, which is a considerable number. [5, 6] Fever, diarrhea, gastrointestinal hemorrhage, discomfort in the abdomen, vomiting, diarrhea, hepatic encephalopathy, and other common symptoms and signs have been linked to SBP. [7,8] A positive ascitic fluid culture and a neutrophil count of more than 240/mm<sup>3</sup> are used to diagnose a classic instance of SBP. Based on the results of the ascitic fluid analysis (cell count and C/S), two variations of SBP have been identified: Culture Negative Neutrocytic Ascites (CNNA) and Bacterascites (BA). While the ascites fluid culture in bacterascites is positive but the neutrophil count is lower than 240/mm<sup>3</sup>, CNNA has a negative culture with a higher neutrophil count (> 240/mm<sup>3</sup>). [9]

Different biochemical assays, such as serum proteins, albumin, Serum Ascites Albumin Gradient (SAAG), ascitic fluid proteins/albumin, and ascitic fluid glucose levels, have also been proven to predict or imply the existence of SBP in cirrhosis in addition to the symptoms or ascitic fluid cell count. Gram-negative aerobic bacteria from the family Enterobacteriaceae are described as the leading cause of SBP (60%) because bacterial translocation from the intestinal lumen is primarily thought to be the antecedent factor for developing SBP. [9] *Streptococcus pneumoniae*, a non-enterococcal species, is the second most frequent bacterial pathogen identified from ascitic fluid (35%). However, SBP episodes brought on by gram-positive bacteria are now more often observed. [10-

13] The indiscriminate use of antibiotics, an increase in invasive procedures, and hospitalization in intensive care units are thought to be the causes of these changes in the bacteriological spectrum. This suggests the need for ongoing evaluation of common bacterial pathogens and their antibiogram to inform the empirical treatment of SBP patients. The purpose of this study was to determine the prevalence of SBP or its variations in CLD patients and the correlation between various indications, symptoms, or laboratory results and SBP.

### Material and Methods

This cross-sectional study was done in the Department of General Surgery, Prathima Institute of Medical Sciences, Naganoor, Karimnagar. Institutional Ethical approval was obtained for the study. Written consent was obtained from the participants of the study after explaining the nature of the study in the local language.

### Inclusion criteria

1. Ascites due to liver cirrhosis of any etiology.
2. Males and Females
3. Aged 20 – 80 years
4. Voluntarily willing to participate in the study

### Exclusion criteria

1. Non-cirrhotic ascites
2. Secondary bacterial peritonitis due to any cause
3. Ruptured intra-abdominal abscess
4. Intestinal perforations

Based on the inclusion and exclusion criteria n=50 cases were included in the study during the period of the study. The demographic profile of the patients was noted. A thorough history was obtained. The results of a thorough general physical examination were sought after, and symptoms of hepatocellular failure were observed. An abdominal ultrasound was used to confirm the clinical diagnosis of cirrhosis and ascites. Regular tests

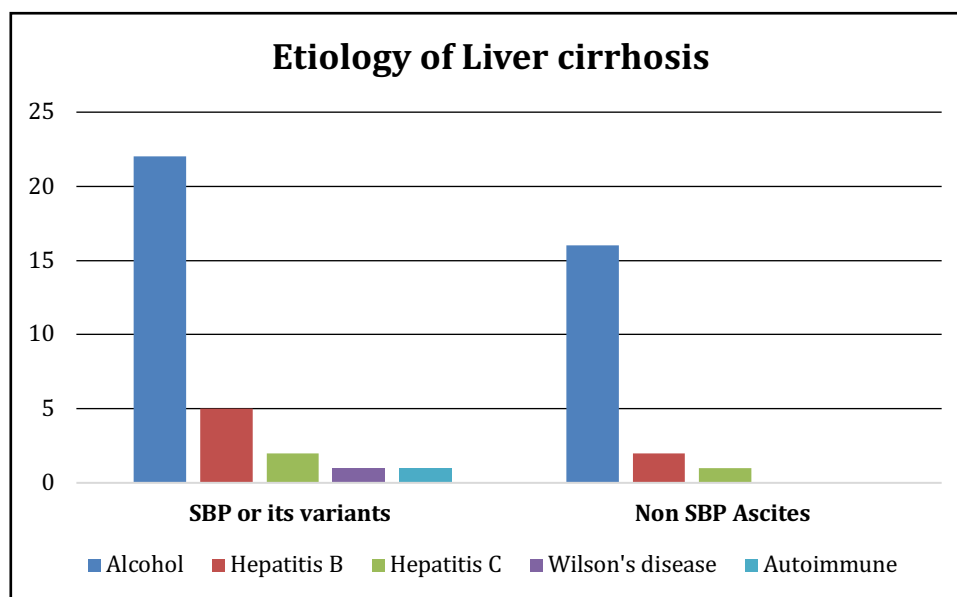
including CBC, LFT, RFT, serum electrolytes, regular urine collection, microscopy, and PT INR were all approximated. Before administering antibiotics for either preventative or therapeutic purposes, a diagnostic abdominal paracentesis was carried out in all patients who were suspected of having spontaneous bacterial peritonitis or one of its variations.

After that, ascitic fluid was sent for routine, microscopy, and culture inspection. At the time of paracentesis, patients were also assessed to obtain their Child-Pugh score, which measures the severity of the liver disease. Additionally, to the grading described above, each patient's MELD Na (Model for End Stage Liver Disease - Sodium) score was computed using an online calculator. [14]

**Statistical analysis:** The data was collected and uploaded on an MS Excel spreadsheet and analyzed by SPSS version 22 (Chicago, IL, USA). Quantitative variables were expressed on mean and standard deviations and qualitative variables were expressed in proportions and percentages. The Chi-square test has been used to find the difference between two proportions.

## Results

The age range of the group of cases in the study was from 35 – 75 years majority of the patients 44% belong to the age group 41 – 50 years. The mean age of the cases in the study was  $47.66 \pm 9.57$  years. The predominant etiological factor for liver cirrhosis was Alcoholism in 76% of all cases followed by Hepatitis B in 14% of cases and other causes depicted in Figure 1.



**Figure 1: Etiology of liver cirrhosis in SBP and Non-SBP ascites**

The predominant complaint/symptoms reported by the cases in the study included Abdominal distension in 92% of the cases followed by abdominal pain in 64% of cases, and swelling of the feet bilaterally was reported in 68% of the cases. Other complaints were fever in 36% of cases, jaundice in 26% of cases, altered sensorium and GI bleeding manifestation

found in 12% of cases, and oliguria in 8% of cases the detailed distribution of the symptoms in SBP and non-SBP cases are depicted in table 1. When the comparison of symptoms in SBP and non-SBP cases were analyzed the p values were found to be ( $<0.05$ ) for abdominal pain, fever, and swelling of feet.

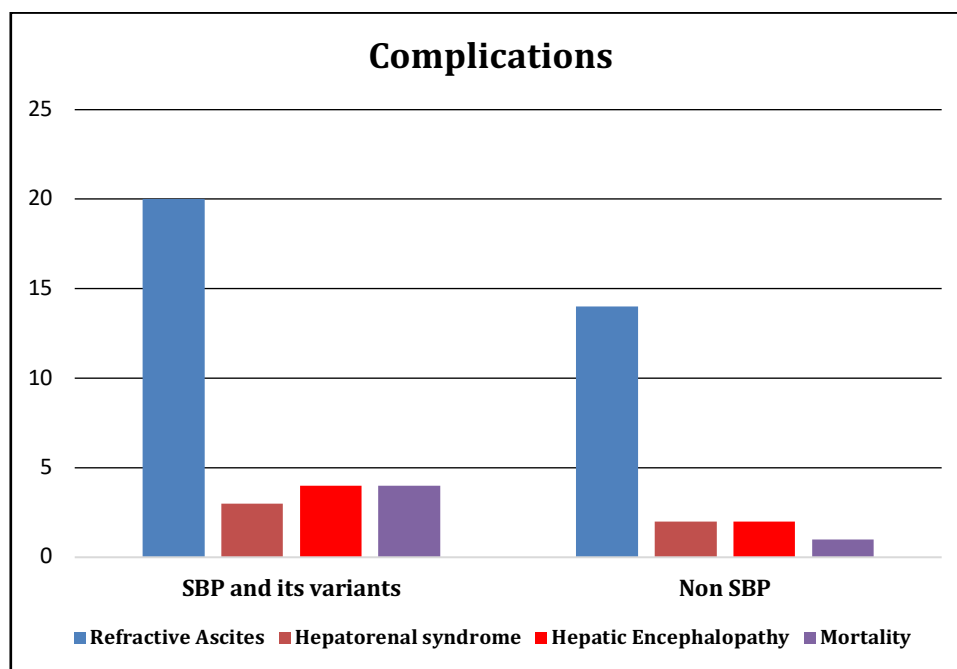
**Table 1: Complains and symptoms in the cases of the study**

Complaints/symptoms	SBP or its variants	Non-SBP or its variants
Abdominal distension	30	16
Abdominal pain	21	11
Fever	15	3
Jaundice	8	5
Altered Sensorium	5	1
GI Bleeding	4	2
Swelling of feet	20	9
Oliguria	3	1
Other	10	4

\* Note: Patients presenting with more than one complaint simultaneously

Cases of mortality occurred in n=5(10%) of all cases of which n=4/31(12.9%) occurred in SBP and its variants cases and n=1/19(5.26%) in non-SBP and its variant cases. Out of these cases of mortality culture, positive bacterial peritonitis was in 60% of cases. N=1 case was due to culture-negative neurocytic ascites.

Mortality due to non-SBP ascites was found in the n=1 case. Refractive ascites was found in n=20/31(64.51%) SBP cases and n=14/19(73.68%) non-SBP cases. Hepatic Encephalopathy was found in n=4/31 and n=2/19 non-SBP cases details depicted in figure 2.



**Figure 2: Complications in SBP ascites and non-SBP ascites cases in the study**

The Laboratory parameters of the patients with SBP ascites and Non-SBP ascites were estimated out of all the parameters the WBC counts were higher in non-SBP ascites as compared to SBP ascites, however serum total bilirubin levels were

found to be high in SBP ascites as compared to non-SBP ascites although none of the parameters was found to be significant the details have been depicted in table 2.

**Table 2: Laboratory findings in the cases of the study**

Lab findings	SBP and its variants	Non-SBP	p-value
Ascitic fluid			
Glucose (mg/dl)	110	105.33	0.512
Total Proteins (mg/dl)	1.88	1.920	0.448
Albumin (g/dl)	0.83	0.924	0.325
Blood			
WBC X 10 <sup>3</sup> /mm <sup>3</sup>	9.28	11.57	0.123
Total Proteins (g/dl)	6.71	6.37	0.335
Albumin (g/dl)	2.66	2.61	0.327
Total Bilirubin (mg/dl)	4.22	2.63	0.147
Serum Ascites Albumin Gradient (SAAG) (gm/dl)	1.88	1.47	0.067

*E. coli* was the predominant organism found in 5/10(50%) of the culture-positive SBP cases followed by *Enterococcus* species in 2/10(20%) cases, followed by *Klebsiella*, *Pseudomonas*, CoNs in 1/10 (10%) cases each. Ceftriaxone was the initial antibiotic given in 40% of cases followed by Cefotaxime in 18% of cases and Norfloxacin in 20% of cases. Depending on the culture report and patients' progress the other antibiotics they were used included piperacillin-tazobactam, cefoperazone + sulbactam in 4% of patients each and Ticarcillin + Clavulanate in 2% of cases. Mortality was significantly lower in patients with a MELD Na score < 30 as compared to patients with a score > 30.

### Discussion

The chronic form of liver disease called cirrhosis is often identified histologically by the emergence of regenerating nodules encircled by fibrous bands. According to the WHO, cirrhosis causes 1.1% of all fatalities. One of the most common and dangerous symptoms of decompensated liver cirrhosis is spontaneous bacterial peritonitis (SBP). [15] Since its first description in 1964, research has changed spontaneous bacterial peritonitis (SBP) from a dreaded condition (with a reported mortality of 90%) to a curable consequence of decompensated cirrhosis, despite its constant incidence and high

recurrence rate. [16] Blood-borne infections like spontaneous bacterial peritonitis can occur. The majority of the time, gut-derived bacteria are involved. Extraintestinal bacteria from the epidermis, urogenital tract, or respiratory system are far less usually implicated. Another potential cause of infection is the use of catheters and other intrusive devices. [17] Pathogenesis of spontaneous bacterial peritonitis is primarily characterized by four factors that include; an overgrowth of tiny intestinal bacteria, a rise in intestinal permeability, bacterial translocation, and immunosuppression. These essential components are related rather than distinct. In individuals with spontaneous bacterial peritonitis, pyrexia, increased disorientation, diffuse abdominal pain, expanding abdominal circumference, vomiting, and decreased urine output or ileus are the most typical symptoms and indicators. However, the clinical picture remains vague. The infection is mostly asymptomatic in most cases. [18] It is now well acknowledged that analyzing a sample of ascitic fluid is the sole approach to identifying a spontaneous bacterial peritonitis event. In patients with liver cirrhosis with ascites, diagnostic paracentesis performed within 12 hours of hospitalization is now advised because it is linked to earlier detection of spontaneous bacterial peritonitis and a better outcome or short-term survival because treatment

can then be started right away without delay. [19] The diagnosis of spontaneous bacterial peritonitis or one of its subtypes, such as bacterascites or culture-negative neutrocytic ascites, can be determined based on the ascitic fluid report. When bacteria isolated from a culture colonize ascitic fluid and the ascitic fluid polymorphonuclear count (PMNL) rises to 250/mm<sup>3</sup>, the condition is referred to as spontaneous bacterial peritonitis.

In the lack of an inflammatory response in the bacterial fluid, i.e., PMNL count is 250/mm<sup>3</sup>, the colonization of ascitic fluid by bacteria observed on culture is referred to as bacterascites (monomicrobial non-neutrocytic bacterascites). [20] When the ascitic PMNL count is 250/mm<sup>3</sup> but cultures are unable to support bacterial growth, the clinical condition is known as culture-negative neutrocytic ascites (CNNA) which is referred to as such. [21] Aggressive antibiotic therapy should be administered to patients with ascitic fluid PMN counts more than or equal to 250 cells/mm<sup>3</sup> in a clinical environment conducive to ascitic fluid infection. Patients with suspected ascitic fluid infection should get relatively broad-spectrum medication while waiting for the results of susceptibility testing. The preferred course of therapy for suspected SBP appears to be cefotaxime or a comparable third-generation cephalosporin, which protects against 95% of the flora, including the three most prevalent isolates of *Escherichia coli*, *Klebsiella pneumoniae*, and *Streptococcal pneumoniae*. The coverage spectrum may often be narrowed after sensitivities are identified. Our research has certain similarities with those of Kavita Paul et al., [22] Nakul Kadam et al., [23] and C Kumar Bal et al., [24] Similar to the study by Nakul Kadam et al., [23] (17%), our investigation showed an in-hospital death rate of 21.67% for all patients and 20% for those with SBP or its variations. In a study by C Kumar Bal et al., [24] the death rate was greater (43%). AKI, hepatic

encephalopathy, refractory Ascites, hepatorenal Syndrome, Child-Pugh Grade C, MELD Na score > 30, anemia, leukocytosis, coagulopathy or deranged PT INR, hyperbilirubinemia, hyponatremia, leukocytosis, coagulopathy or deranged PT INR, leukocytosis, and absence of albumin transfusion during hospitalization were all associated with increased mortality.

### Conclusion

Our research aimed to examine the prevalence of individuals with SBP or its variations as well as their diverse clinical and bacteriological profiles. Within the limitations of the current research, we found diagnostic paracentesis for D/R and C/S is crucial since it is difficult to diagnose SBP based solely on clinical signs. *Escherichia coli* (*E. coli*) is the most frequently isolated pathogen. Prompt broad-spectrum antibiotic therapy is the recommended course of treatment for SBP and should be adjusted based on resistance profiles. Supplementing with albumin is also advantageous, particularly for people with renal impairment (RI).

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