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ORIGINAL RESEARCH ARTICLE

Clinical Spectrum, Microbiological Profile, and Complications of Spontaneous Bacterial Peritonitis in Ascitic Cirrhosis: An 18-Month Cross-Sectional Study

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ABSTRACT

Introduction: Ascites, a common complication of liver cirrhosis, often lead to spontaneous bacterial peritonitis (SBP), a severe infection of ascitic fluid. SBP contributes to high morbidity and mortality in cirrhotic patients. This study aimed to evaluate the prevalence, risk factors, clinical spectrum, and microbiological and biochemical profiles of SBP in patients with ascites.

Material and methods: An 18-month cross-sectional observational study included 84 cirrhotic patients with ascites, diagnosed clinically and radiologically. SBP was diagnosed based on ascitic fluid analysis (polymorphonuclear count > 250 cells/mm³ or positive ascitic fluid culture). Exclusion criteria included age under 18, prior antibiotics use, and secondary causes of ascites. Data were analyzed using SPSS, with chi-square and t-tests for categorical and continuous variables, respectively. Ethical approval was obtained.

Results: SBP patients had significantly higher neutrophil counts (292.3 ± 31.9 *vs.* 126.5 ± 15.0, $p < 0.001$). The microbiological culture revealed *E. coli* (60%) as the predominant pathogen. SBP patients also had higher rates of UGI bleeding (48.0 vs. 10.2%, $p = 0.001$) and worse severity scores (CTP Class C: 48.0%, $p = 0.0017$). Hepatorenal syndrome (HRS) was significantly more common in SBP patients (28.0 *vs.* 5.1%, $p = 0.003$), and mortality was higher (16.0 *vs.* 3.4%, $p = 0.04$).

Conclusion: SBP in cirrhotic patients is associated with increased neutrophil counts, complications such as UGI bleeding and HRS, and higher mortality. Early detection and appropriate management are crucial for improving patient outcomes.

Keywords: Ascites, Spontaneous bacterial peritonitis, Liver cirrhosis, Neutrophil count, Hepatorenal syndrome, Mortality, Microbiological profile, Clinical outcomes

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INTRODUCTION

Ascites, a frequent complication of liver cirrhosis, result from fluid accumulation in the peritoneal cavity and affect up to 80% of cirrhotic patients during their illness. Among its complications, spontaneous bacterial peritonitis (SBP) is particularly life-threatening, characterized by infection of ascitic fluid without an evident intra-abdominal source, and associated with significant morbidity and mortality.[1]

SBP commonly arises in cirrhotic patients due to bacterial translocation from the gut to the ascitic fluid, facilitated by impaired immune defenses and nutrient-rich ascitic fluid. Clinical manifestations range from subtle symptoms like abdominal discomfort to severe presentations such as systemic inflammatory response syndrome.[2]

Diagnosis is based on ascitic fluid analysis, with a polymorphonuclear leukocyte count ≥250 cells/mm³ being diagnostic.[2] Empirical antibiotic therapy, often using third-generation cephalosporins, is the mainstay of treatment; however, the emergence of multidrug-resistant organisms has posed significant challenges.[3] Adjunctive therapies such as albumin infusion are sometimes employed to mitigate renal complications. Despite advancements, gaps remain in understanding SBP's pathogenesis, optimal therapy duration, and preventive strategies.[4,5]

This study aims to evaluate the prevalence, risk factors, clinical spectrum, and microbiological and biochemical profiles of SBP in patients with ascites. Insights from this research could improve early detection, management, and outcomes in these patients.

MATERIAL AND METHODS

This study was designed as a cross-sectional observational study conducted over 18 months. The study included 84 patients admitted to the medicine department with confirmed hepatic cirrhosis and ascites, diagnosed through clinical and radiological methods. These patients were screened for spontaneous bacterial peritonitis

(SBP) using clinical history, physical examination, and cytological, microbiological, and biochemical tests.

The study population comprised cases of chronic liver disease presenting with ascites. Inclusion criteria were patients with ascites and evidence of chronic liver disease diagnosed based on a medical history of more than six months supported by radiological findings. Patients were included if they met the diagnostic criteria for SBP, characterized by an ascitic fluid polymorphonuclear count >250 cells/mm^3 or a positive ascitic fluid culture without a primary intra-abdominal source of infection. Exclusion criteria included patients under 18 years of age, those who received antibiotics within three weeks before admission, and cases of secondary peritonitis, tuberculosis-associated ascites, malignant ascites, or non-cirrhotic portal hypertension.

SBP diagnosis was based on the following criteria: an ascitic fluid neutrophil count greater than 250 cells/mm^3, a positive culture of ascitic fluid, and no primary source of infection in the abdomen. Ascitic fluid was collected under aseptic precautions immediately after patient admission and before the administration of antibiotics.

A comprehensive clinical evaluation was conducted, including detailed patient history and physical examination. Ascites severity was graded according to the International Ascites Club criteria, while hepatic encephalopathy severity was classified using the West Haven grading system. All patients underwent biochemical, microbiological, and radiological investigations, along with endoscopy, as required. Laboratory evaluations included complete blood counts, liver function tests, renal function tests, viral markers (HBsAg and anti-HCV), coagulation profiles, and classification using the Child-Pugh-Turcotte score. Additional tests like chest X-rays, ECG, plain abdominal X-rays, and upper gastrointestinal endoscopy were performed based on clinical necessity.

Table 1: Demographic and clinical characteristics of study participants

Characteristic	*Spontaneous Bacterial Peritonitis*	*p-value*
Age (Mean ± SD)	Present: 42.0 ± 9.9, Absent: 42.1 ± 12.0:	0.74
Gender	Male: 17 (68.0%)/ Female: 8 (32.0%)	Chi-square: 0.37, p = 0.54

The sample size for the study was calculated to be 84, based on a 28% prevalence of SBP, with a 90% confidence interval and a 10% margin of error. Data analysis was conducted using SPSS version 25 and MS Excel. Qualitative data were analyzed using the chi-square test, while quantitative data were assessed with t-tests and ANOVA.

Ethical approval for the study was obtained from the institutional ethics committee, ensuring compliance with all ethical standards.

RESULTS

The study revealed significant differences between patients with spontaneous bacterial peritonitis (SBP) and those without, particularly regarding clinical characteristics, laboratory parameters, complications, and overall outcomes. Table 1 showed no significant differences in age or gender between the two groups ($p > 0.05$).

Table 2 showed that patients with SBP had a significantly higher neutrophil count (292.3 ± 31.9 cells/mm^3) compared to those without SBP (126.5 ± 15.0 cells/mm^3) ($p < 0.001$), indicating a marked inflammatory response. Other laboratory parameters, including bilirubin, albumin, PT, INR, and creatinine, did not show significant differences ($p > 0.05$).

Table 3 revealed that UGI bleeding was more prevalent in the SBP group (48.0%) compared to the non-SBP group

Table 2: Comparison of laboratory parameters in patients with and without spontaneous bacterial peritonitis.

Laboratory parameters	*Spontaneous bacterial peritonitis*			*p-value*
	Present (Mean ± SD)	*Absent (Mean ± SD)*	*Total (Mean ± SD)*	
AF neutrophil count (cells/mm^3)	292.3 ± 31.9	126.5 ± 15.0	175.9 ± 79.1	<0.001
Bilirubin	5.0 ± 6.8	4.5 ± 4.6	4.6 ± 5.3	0.67
Albumin	3.2 ± 0.7	3.2 ± 0.6	3.2 ± 0.6	0.8
PT	20.1 ± 5.1	19.9 ± 4.8	20.0 ± 4.9	0.89
INR	1.7 ± 0.5	1.7 ± 0.6	1.7 ± 0.6	0.74
Creatinine	0.9 ± 0.6	1.0 ± 0.8	0.9 ± 0.6	0.49
Cholesterol	135.7 ± 23.5	136.7 ± 23.9	136.0 ± 23.5	0.85
TG	119.4 ± 26.7	119.4 ± 27.8	119.4 ± 26.9	0.99
HDL	34.0 ± 6.6	34.5 ± 6.2	34.2 ± 6.5	0.76
LDL	78.3 ± 11.8	78.4 ± 12.9	78.3 ± 12.1	0.95

Table 3: UGI bleedingand international ascites club grading

Variable	*SBP present (%)*	*SBP absent (%)*	*Total (%)*	*p-value*
UGI bleeding				
Present	12 (48.0%)	6 (10.2%)	18 (21.4%)	0.001*
Absent	13 (52.0%)	53 (89.8%)	66 (78.6%)	
Ascites grade				
Grade 1	4 (16.0%)	20 (33.9%)	24 (28.6%)	
Grade 2	16 (64.0%)	31 (52.5%)	47 (56.0%)	0.24
Grade 3	5 (20.0%)	8 (13.6%)	13 (15.5%)	

Table 4: Severity scores and clinical outcomes

Severity variables	*SBP present (%)*	*SBP absent (%)*	*Total (%)*	*p-value*
CTP class				
A	4 (16.0%)	21 (35.6%)	25 (29.8%)	
B	9 (36.0%)	27 (45.8%)	36 (42.9%)	0.0017*
C	12 (48.0%)	11 (18.6%)	23 (27.4%)	
West Haven criteria				
0	3 (12.0%)	22 (37.3%)	25 (29.8%)	
1	7 (28.0%)	19 (32.2%)	26 (31.0%)	0.03*
2	2 (8.0%)	5 (8.5%)	7 (8.3%)	
3	13 (52.0%)	13 (22.0%)	26 (31.0%)	

Table 5: Complications associated with SBP

Complication	*SBP present (%)*	*SBP absent (%)*	*Total (%)*	*Chi-square/p-value*
HRS	7 (28.0%)	3 (5.1%)	5 (6.0%)	Chi-square: 8.7, p = 0.003*
HPS	1 (4.0%)	0 (0.0%)	1 (1.2%)	Fisher's Exact: 2.34, $p = 0.30$
Encephalopathy	7 (28.0%)	16 (27.1%)	23 (27.4%)	Chi-square: 0.007, $p = 0.94$

Table 6: Overall outcome

Outcome	*SBP present (%)*	*SBP absent (%)*	*Total (%)*	*Chi-square/p-value*
Survived	21 (84.0%)	57 (96.6%)	78 (92.9%)	Chi-square: 4.2, $p = 0.04$*
Died	4 (16.0%)	2 (3.4%)	6 (7.1%)	

(10.2%) ($p = 0.001$). Ascites grade distribution did not show a significant difference between the two groups ($p > 0.05$).

Table 4 shows the SBP group had significantly worse outcomes. A higher proportion of SBP patients were classified as CTP Class C (48.0%) compared to the non-SBP group (18.6%) ($p = 0.0017$). Additionally, the West Haven criteria indicated that 52.0% of SBP patients had severe hepatic encephalopathy (Grade 3), compared to 22.0% in the non-SBP group ($p = 0.03$).

Table 5 showed that patients with SBP had a significantly higher incidence of hepatorenal syndrome (HRS) (28.0 *vs.* 5.1%, $p = 0.003$), although hepatic encephalopathy was similarly prevalent in both groups (28.0 *vs.* 27.1%, $p = 0.94$). Hepatopulmonary syndrome (HPS) was rare and not significantly different between the two groups ($p = 0.30$). Table 6 demonstrated that mortality was significantly higher in the SBP group (16.0%) compared to the non-SBP group (3.4%) ($p = 0.04$). The survival rate was lower in SBP patients, with 84.0% surviving compared to 96.6% in the non-SBP group.

Micro-organisms

identified in SBP cultures were predominantly *E. coli* (60%), followed by *Klebsiella* and *Staphylococcus pneumoniae* (20% each).

DISCUSSION

In this study on spontaneous bacterial peritonitis (SBP) among 84 ascitic patients, individuals aged 31 to 50 predominated, with a male-to-female ratio of 53:31. SBP was diagnosed in 25 patients (29.8%), with a mean age of 42.0 ± 9.9 years, while the overall sample mean age was 42.1 ± 10.5 years. SBP prevalence was higher in males (68%) than females (32%). The association between upper gastrointestinal (UGI) bleeding and SBP was significant ($\chi^2 = 14.9$, $p = 0.001$), with SBP patients showing lower

survival (84.0%) compared to those without SBP (96.6%), also statistically significant ($\chi^2 = 4.2$, $p = 0.04$).

Comparatively, Duah *et al*. reported a mean age of 44.7 ± 13.2 years in 140 ascitic patients, with a 21.43% prevalence of SBP and *E. coli* (41.7%) as the predominant pathogen. Differences in demographics and methodologies highlight variations in findings.[2] Similarly, Oladimeji *et al*. found a 67% culture-positive SBP prevalence among 31 patients, with *E. coli* (70%) as the most common organism. Poor prognostic indicators included low ascitic protein, coagulopathy, and renal dysfunction ($p < 0.05$). [6]

In relation to UGI bleeding, Shih H. *et al*. noted that bacteremia significantly impacts mortality in cirrhotic patients, underscoring differences in focus between studies. [7]

The findings of the present study indicate a significant association between the presence of spontaneous bacterial peritonitis (SBP) and mortality. Among those with SBP, 84% survived, while 16% succumbed to the condition. In contrast, the survival rate among individuals without SBP was higher at 96.6%, with only 3.4% mortality. This difference was statistically significant ($\chi2 = 4.2$, $p = 0.04$), reinforcing the impact of SBP on survival outcomes. Similarly, the present study observed a notable association between SBP and hepatorenal syndrome (HRS), with a significant proportion of individuals with HRS also presenting with SBP ($\chi2 = 8.7$, $p = 0.003$). This suggests a correlation between these two conditions, as previously discussed by Baraldi *et al*., who identified SBP as a trigger for HRS in a substantial subset of patients. [8]

However, the observed correlation between HRS and SBP in this study deviates from expectations. Typically, ascites, which is a common feature of HRS, are linked to a higher incidence of SBP. In contrast, this study revealed a relatively lower occurrence of SBP in individuals with HRS, which may suggest variations in the disease presentation or other unmeasured factors influencing the incidence of SBP. This contrasts with the findings by Gines *et al*., who highlighted the increased risk of SBP among patients with decompensated liver disease. [4,9]

Furthermore, the study found a significant association between Child-Turcotte-Pugh (CTP) scores and SBP. Higher CTP scores correlated with a greater occurrence of SBP, particularly in individuals classified as CTP-C. This result aligns with previous studies, such as Miozzo *et al*., who observed that the severity of liver disease, as indicated by CTP scores, plays a critical role in the development of SBP.[10] Similarly, Elzouki *et al*. identified the CTP score and other variables like acute kidney injury as significant predictors of mortality in SBP patients.[87] This is consistent with Paul *et al*.'s findings that CTP class C patients had the highest incidence of SBP, further supporting the relationship between liver dysfunction severity and SBP occurrence. [12]

The findings from the current study suggest a lack of statistically significant correlation between the severity of hepatic encephalopathy (as assessed by WHC scores) and the occurrence of spontaneous bacterial peritonitis (SBP). This aligns with the study by Paul *et al*., who identified multiple predictors for SBP in cirrhotic patients, including fever, abdominal pain, jaundice, renal failure, encephalopathy, and a MELD score greater than 14.[11] However, the association of WHC scores with SBP was not emphasized in their findings. The study by Huang *et al*. introduced a seven-stage model that included both hepatic encephalopathy and SBP, demonstrating improved prognostic accuracy for mortality in cirrhosis compared to simpler models, although the prognostic impact of hepatic encephalopathy alone was not distinctly differentiated from SBP. [13]

Interestingly, in contrast to the current study, Oladimeji *et al*. found that older age (mean 62 years) was significantly associated with SBP, with a predominance of gram-negative bacteria, especially *E. coli*, in culture-positive cases. They also identified several laboratory and clinical factors (such as low platelet count, high INR, renal dysfunction, and leukocytosis) that were significantly linked with SBP, which the current study did not find. This highlights the complex nature of SBP development, which may be influenced by various factors beyond encephalopathy.[6]

One of the more compelling findings from the present study is the significant increase in neutrophil count in ascitic fluid among SBP patients. The mean neutrophil count was considerably higher in those with SBP (292.3 ± 31.9 cells/mm^3) compared to those without SBP (126.5 ± 15.0 cells/mm^3), which was statistically significant ($p < 0.001$). This underscores the diagnostic potential of neutrophil count in ascitic fluid for SBP, corroborating findings from Sheta *et al*., who also emphasized the importance of absolute neutrophil count (ANC) in diagnosing SBP. Their study found that an ANC cutoff value of >2.804 showed strong sensitivity and specificity for detecting SBP, highlighting its potential clinical utility. While the current study aligned with this finding, it did not find other laboratory parameters (such as WBC count or CRP) to be significantly different between the SBP and non-SBP groups, unlike Sheta *et al*.'s study.[14]

The study by Victor *et al*. also highlighted the importance of leukocyte count, particularly neutrophil count, in the ascitic fluid for diagnosing SBP, though it did not report significant differences in leukocyte counts between SBP and non-SBP groups. This contrasts with the

current study's more robust findings on neutrophil count, indicating that while neutrophil count is a promising diagnostic marker, its utility might vary depending on the specific patient population and study design.[15]

Moreover, laboratory parameters like bilirubin, albumin, creatinine, PT, INR, and others did not show statistically significant differences between SBP and non-SBP groups in the current study, which contrasts with studies such as Metwali *et al*., where significant differences in these parameters were found, particularly with variables like CRP, which was shown to be a useful predictor of SBP in patients with hepatitis C-related cirrhosis.[16] This difference in findings could be due to the specific etiology of liver disease in the populations studied, as the current study did not focus on a single liver disease etiology.

Lastly, studies like those by Tay Lin *et al*. and Nguyen *et al*. provide broader insights into the global and microbiological characteristics of SBP. Tay Lin *et al*.'s meta-analysis revealed a global SBP prevalence of 17.12%, with the highest prevalence in Africa, suggesting geographical variability in SBP incidence and outcomes.[17] Nguyen *et al*.'s findings underscored the challenges in treating SBP, with a significant proportion of cases being culture-negative and highlighting the importance of antibiotic resistance, particularly to commonly prescribed fluoroquinolones.[18]

While the current study did not find significant associations between hepatic encephalopathy severity and SBP, it emphasized the utility of neutrophil count in diagnosing SBP. The findings complement those from other studies that highlight the complex etiology, clinical presentation, and diagnostic markers of SBP, suggesting the need for comprehensive diagnostic approaches in managing SBP in cirrhotic patients.

CONCLUSION

Patients with SBP exhibited significantly higher neutrophil counts, a higher incidence of UGI bleeding, and worse severity scores. They also experienced more complications, particularly hepatorenal syndrome (HRS). Mortality was notably higher in the SBP group, emphasizing the need for early detection and management of SBP in cirrhotic patients.

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