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Original Research Article

Prevalence of Spontaneous Bacterial Peritonitis in Patients with Decompensated Liver Disease and Ascites: A Cross-Sectional Observational Study

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Abstract

Background: Spontaneous bacterial peritonitis (SBP) is a serious complication of decompensated liver disease with ascites. Indian data on SBP prevalence and clinical correlates remain limited.

Objective: To determine SBP prevalence, microbiological profile, associated clinical parameters, and in-hospital mortality in patients with decompensated liver disease and ascites.

Methods: This cross-sectional observational study enrolled 130 consecutive patients with confirmed liver cirrhosis and ascites at L.N. Medical College and J.K. Hospital, Bhopal. All patients underwent diagnostic paracentesis. SBP was diagnosed when ascitic fluid polymorphonuclear (PMN) cell count was ≥250 cells/mm³. Chi-squared and Fisher's exact tests were used; $p < 0.05$ was considered significant.

Results: The overall SBP prevalence was 13.8% (18/130), comprising culture-positive SBP (8.5%) and culture-negative neutrocytic ascites (5.4%). Mean age was 48.4 ± 12.0 years with male predominance (83.1%). Alcoholic cirrhosis was the commonest etiology (50.8%). Escherichia coli and Staphylococcus spp. were the most frequently isolated organisms (27.3% each). Altered sensorium was significantly associated with SBP ($p = 0.036$). In-hospital mortality was 16.2% overall, and higher among SBP patients (27.8% vs 14.3%; $p = 0.170$).

Conclusion: SBP is a common complication in hospitalised patients with decompensated liver disease, with a prevalence of 13.8%. Altered sensorium is a significant clinical predictor. Routine diagnostic paracentesis is recommended in all cirrhotic patients admitted with ascites.

Keywords: Spontaneous Bacterial Peritonitis; Decompensated Liver Disease; Ascites; Liver Cirrhosis; Ascitic Fluid Analysis; Prevalence.

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Introduction

Liver cirrhosis represents the end stage of chronic liver disease and is a major cause of morbidity and mortality worldwide. India bears a significant burden, with cirrhosis accounting for a substantial proportion of hospital admissions in departments of general medicine and gastroenterology [1,2]. Decompensated cirrhosis, characterised by the development of clinically evident complications such as ascites, variceal haemorrhage, hepatic encephalopathy, and jaundice, carries a poor prognosis with a median survival of approximately two years without liver transplantation [3]. Ascites is the most common complication of cirrhosis, occurring in approximately 60% of patients with compensated disease within 10 years of diagnosis [4]. The development of ascites marks a critical turning point, as it predisposes patients to spontaneous bacterial peritonitis (SBP), a bacterial infection of the ascitic fluid occurring in the absence of any surgically treatable intra-abdominal source of infection [5]. SBP is defined by an ascitic fluid polymorphonuclear (PMN) leucocyte count of ≥250 cells/mm³, with or without a positive ascitic fluid culture [6]. The reported prevalence of SBP

varies widely across studies, ranging from 10% to 30% among hospitalised cirrhotic patients with ascites [7–9]. SBP is associated with significant morbidity, including hepatorenal syndrome and septic shock, and carries an in-hospital mortality rate of 20–40% if not promptly recognised and treated [10,11]. Despite its clinical importance, SBP may present with subtle or non-specific symptoms, and a significant proportion of patients may be asymptomatic at the time of diagnosis [12].

The microbiological spectrum of SBP has evolved over time, with gram-negative organisms, particularly Escherichia coli and Klebsiella pneumoniae, traditionally being the most commonly implicated pathogens [13]. However, recent studies have reported an increasing prevalence of gram-positive organisms and multidrug-resistant bacteria, particularly in healthcare-associated settings [14,15]. This changing microbiological landscape has important implications for empirical antibiotic therapy.

Several risk factors for the development of SBP have been identified, including low ascitic fluid protein concentration (<1.5 g/dl), advanced liver disease (Child–Pugh class C), high Model for End-Stage Liver Disease (MELD) score, prior SBP episodes, gastrointestinal bleeding, and proton pump inhibitor use [16–18]. Early identification of high-risk patients is essential for implementing prophylactic strategies.

Despite the high burden of chronic liver disease in India, data on the prevalence of SBP, its microbiological profile, and associated clinical parameters from central Indian populations remain limited.

Most Indian studies have originated from tertiary referral centres in northern and southern India, and regional differences in etiology, healthcare access, and antimicrobial resistance patterns may influence SBP epidemiology [19]. The present study was therefore undertaken to determine the prevalence of SBP among hospitalised patients with decompensated liver disease and ascites at a tertiary care hospital in central India, to characterise its clinical and microbiological profile, and to assess its association with disease severity and in-hospital mortality.

Materials and Methods

Study design and setting: This was a cross-sectional observational study conducted at the Department of General Medicine, L.N. Medical College and Research Centre, J.K. Hospital, Bhopal, Madhya Pradesh, India. The institution is a tertiary care teaching hospital serving a large population from central India.

Study population: A total of 130 consecutive patients aged 18 years and above who were admitted with confirmed liver cirrhosis and ascites during the study period were enrolled. The diagnosis of liver cirrhosis was based on clinical, biochemical, and ultrasonographic findings.

Inclusion criteria: Patients aged ≥18 years with confirmed liver cirrhosis of any etiology and clinically or ultrasonographically detectable ascites were included.

Exclusion criteria: Patients with secondary peritonitis (evidenced by surgical or radiological findings), those who had received antibiotics within 48 hours prior to paracentesis, patients with hepatocellular carcinoma or other intra-abdominal malignancies, tubercular peritonitis, and those who refused to give informed consent were excluded.

Data collection: A detailed history was obtained and clinical examination performed for all patients at the time of admission. Sociodemographic data including age, gender, education, occupation, residence, socio-economic status, and religion were recorded. Clinical parameters including etiology of cirrhosis, duration of illness, presenting symptoms, general and abdominal examination findings, and vital signs were documented. Disease severity was assessed using the Child–Pugh classification and the Model for End-Stage Liver Disease (MELD) score.

Laboratory investigations: All patients underwent routine laboratory investigations including complete blood count, liver function tests (total and direct bilirubin, ALT, AST, ALP, serum albumin), renal function tests (serum creatinine, blood urea), serum electrolytes (sodium, potassium), and coagulation profile (PT, INR). Hepatitis B surface antigen (HBsAg) and anti-hepatitis C virus (anti-HCV) antibodies were tested for etiological evaluation.

Ascitic fluid analysis: Diagnostic paracentesis was performed under aseptic precautions in all patients at the time of admission, before initiation of antibiotic therapy. Ascitic fluid was analysed for appearance, total and differential cell count (including polymorphonuclear leucocyte count), protein, albumin (for SAAG calculation), and culture and sensitivity. Culture was performed by inoculating 10 ml of ascitic fluid into blood culture bottles at the bedside.

Diagnostic criteria for SBP: SBP was diagnosed when the ascitic fluid PMN cell count was ≥250 cells/mm³, irrespective of the ascitic fluid culture result, in the absence of any surgically treatable intra-abdominal source of infection. SBP was further classified into: (a) culture-positive SBP (PMN ≥250/mm³ with positive culture), (b) culture-negative neutrocytic ascites (CNNA; PMN

≥250/mm³ with negative culture), and (c) monomicrobial non-neutrocytic bacterascites (MNB; PMN <250/mm³ with positive culture) [6].

Statistical analysis: Data were analysed using appropriate statistical software. Continuous variables were expressed as mean ± standard deviation (SD) and range. Categorical variables were expressed as frequency and percentage. Chi-squared test was used for comparison of categorical variables. Fisher's exact test was used when the expected cell count in any cell of the 2×2 contingency table was less than 5. A p-value of less than 0.05 was considered statistically significant.

Ethical considerations: The study was approved by the Institutional Ethics Committee of L.N. Medical College and Research Centre, Bhopal. Written informed consent was obtained from all participants or their legally authorised representatives prior to enrolment.

Results

A total of 130 patients with decompensated liver disease and ascites were enrolled. The mean age was 48.4 ± 12.0 years (range: 21–78 years), with a male predominance (male:female ratio of 4.9:1). The majority (38.5%) belonged to the 51–60 years age group. Illiteracy was prevalent (42.3%), and most patients were from urban areas (62.3%) and belonged to the upper lower socio-economic class (39.2%). Alcoholic cirrhosis was the most common etiology (50.8%), followed by hepatitis B (25.4%), metabolic dysfunction–associated steatotic liver disease (MASLD; 17.7%), and hepatitis C (6.2%). More than half of the patients (51.5%) were classified as Child–Pugh class C, and the mean MELD score was 19.6 ± 7.8. Grade II (moderate) and Grade III (severe) ascites were present in 42.3% and 40.0% of patients, respectively. A history of previous SBP episodes was present in 12.3% of patients. The baseline demographic and clinical characteristics are presented in Table 1.

Table 1: Baseline demographic and clinical characteristics of the study population (n = 130)

Characteristic	Value
Demographic characteristics	
Age, years, mean ± SD	48.4 ± 12.0
Age, years, range	21–78
20–30 years	10 (7.7)
31–40 years	23 (17.7)
41–50 years	34 (26.2)
51–60 years	50 (38.5)
>60 years	13 (10.0)
Gender	
Male	108 (83.1)
Female	22 (16.9)
Male:Female ratio	4.9:1
Education level	
Illiterate	55 (42.3)
Primary	36 (27.7)
Secondary	25 (19.2)
Graduate	11 (8.5)
Postgraduate	3 (2.3)
Occupation	
Unemployed	31 (23.8)
Skilled worker	38 (29.2)
Professional	9 (6.9)
Business	21 (16.2)
Farmer	31 (23.8)
Place of residence	
Urban	81 (62.3)
Rural	49 (37.7)
Socio-economic status	
Upper class	5 (3.8)
Upper middle class	14 (10.8)
Lower middle class	30 (23.1)
Upper lower class	51 (39.2)
Lower class	30 (23.1)
Religion	

Hindu	109 (83.8)
Muslim	16 (12.3)
Others	5 (3.8)
Clinical characteristics	
Etiology of cirrhosis	
Alcoholic	66 (50.8)
Hepatitis B	33 (25.4)
Hepatitis C	8 (6.2)
MASLD	23 (17.7)
Child–Pugh classification	
Class A	17 (13.1)
Class B	46 (35.4)
Class C	67 (51.5)
MELD score	
MELD score, mean ± SD	19.6 ± 7.8
MELD score, range	8–36
<15	42 (32.3)
15–25	59 (45.4)
>25	29 (22.3)
Duration of illness	
<6 months	25 (19.2)
6–12 months	44 (33.8)
1–2 years	36 (27.7)
>2 years	25 (19.2)
Ascites grade	
Grade I (mild)	23 (17.7)
Grade II (moderate)	55 (42.3)
Grade III (severe)	52 (40.0)
Previous SBP episodes	
Yes	16 (12.3)
No	114 (87.7)

Values are n (%) unless otherwise stated. SD = standard deviation; MASLD = metabolic dysfunction–associated steatotic liver disease; MELD = Model for End-Stage Liver Disease; SBP = spontaneous bacterial peritonitis.

The most common presenting symptom was abdominal distension (89.2%), followed by fever (71.5%), abdominal pain (57.7%), nausea/vomiting (46.2%), shortness of breath (37.7%), and altered sensorium (33.8%). On general examination, icterus (84.6%), pallor (77.7%), and pedal edema (69.2%) were frequently observed. Fever (>100°F) was documented in 71.5%, tachycardia in 63.8%, and hypotension in 27.7% of patients.

On abdominal examination, ascites was present in all patients (100.0%), splenomegaly in 73.1%, abdominal tenderness in 63.1%, and hepatomegaly in 27.7% (Table 2).

Table 2: Clinical presentation and physical examination findings (n = 130)

Characteristic	Value
Presenting symptoms	
Abdominal distension	116 (89.2)
Fever	93 (71.5)
Abdominal pain	75 (57.7)
Nausea/vomiting	60 (46.2)
Shortness of breath	49 (37.7)
Altered sensorium	44 (33.8)
General examination findings	
Icterus	110 (84.6)
Pallor	101 (77.7)
Edema	90 (69.2)
Vital signs abnormalities	
Fever (>100°F)	93 (71.5)

Tachycardia (>100/min)	83 (63.8)
Hypotension	36 (27.7)
Abdominal examination	
Ascites	130 (100.0)
Splenomegaly	95 (73.1)
Abdominal tenderness	82 (63.1)
Hepatomegaly	36 (27.7)

Values are n (%). Symptom categories are not mutually exclusive.

Laboratory investigations revealed a mean haemoglobin of 9.27 ± 2.22 g/dl, mean total leucocyte count of 12,573 ± 5,725/mm³, and mean platelet count of 161.3 ± 64.3 ×10³/mm³. Liver function tests showed significantly deranged values with a mean total bilirubin of 9.17 ± 4.75 mg/dl, mean AST of 140.3 ± 65.6 U/L, and mean serum albumin of 2.46 ± 0.84 g/dl. The mean serum creatinine was 2.36 ± 0.99 mg/dl, mean INR was 2.40 ± 0.73, and mean serum sodium was 132.2 ± 8.2 mEq/L. Ascitic fluid analysis showed clear fluid in 73.8% and turbid fluid in 26.2%. The SAAG was ≥1.1 g/dl in 93.8% of patients, confirming portal hypertension as the underlying cause. Of 130 patients, 18 (13.8%) had a PMN count ≥250/mm³. Ascitic fluid culture was positive in 11 patients (8.5%). The most commonly isolated organisms were E. coli and Staphylococcus spp. (27.3% each), followed by Klebsiella pneumoniae (18.2%). Gram-negative organisms accounted for 54.5% and gram-positive organisms for 45.5% of positive cultures (Table 3).

Table 3: Laboratory investigations and ascitic fluid analysis (n = 130)

Parameter	Mean ± SD	Range
Complete blood count		
Hemoglobin (g/dl)	9.27 ± 2.22	5.5–13.0
Total WBC count (/mm³)	12,573 ± 5,725	3,700–21,921
Platelet count (×10³/mm³)	161.3 ± 64.3	48–280
Liver function tests		
Total bilirubin (mg/dl)	9.17 ± 4.75	1.0–17.8
Direct bilirubin (mg/dl)	6.42 ± 3.33	0.7–12.5
ALT (U/L)	105.1 ± 49.8	18–185
AST (U/L)	140.3 ± 65.6	32–259
ALP (U/L)	233.1 ± 93.9	71–390
Serum albumin (g/dl)	2.46 ± 0.84	1.2–4.1
Renal function tests		
Serum creatinine (mg/dl)	2.36 ± 0.99	0.6–4.0
Blood urea (mg/dl)	87.9 ± 40.0	18–115
Serum electrolytes		
Sodium (mEq/L)	132.2 ± 8.2	119–144
Potassium (mEq/L)	3.82 ± 0.74	2.5–5.1
Coagulation profile		
PT (seconds)‡	15 ± 25	11–15
INR	2.40 ± 0.73	1.1–3.7
Ascitic fluid parameter	**n (%)**	
Ascitic fluid appearance		
Clear	96 (73.8)	
Turbid	34 (26.2)	
PMN / neutrophil cell count		
PMN <250/mm³	112 (86.2)	
PMN ≥250/mm³	18 (13.8)	
SAAG		
Mean SAAG (g/dl)	1.80 ± 0.38	
≥1.1 g/dl	122 (93.8)	
<1.1 g/dl	8 (6.2)	
Ascitic fluid culture		
Positive	11 (8.5)	
Negative	119 (91.5)	

Organisms isolated (n = 11)		
E. coli	3 (27.3)*	
Klebsiella pneumoniae	2 (18.2)*	
Klebsiella oxytoca	1 (9.1)*	
Enterococcus spp.	1 (9.1)*	
Staphylococcus spp.	3 (27.3)*	
Streptococcus spp.	1 (9.1)*	
Gram stain profile (n = 11)		
Gram-negative	6 (54.5)*	
Gram-positive	5 (45.5)*	

Upper section: values are mean ± SD with range. Lower section: values are n (%) unless otherwise stated. *Percentages are of positive cultures (n = 11). ‡PT values require verification from raw data. SAAG = serum–ascites albumin gradient; PMN = polymorphonuclear leucocytes; WBC = white blood cells; ALT = alanine aminotransferase; AST = aspartate aminotransferase; ALP = alkaline phosphatase; PT = prothrombin time; INR = international normalised ratio.

The overall prevalence of SBP was 13.8% (18/130). Of these, 11 patients (8.5%) had culture-positive SBP and 7 (5.4%) had culture-negative neutrocytic ascites (CNNA). No case of monomicrobial non-neutrocytic bacterascites (MNB) was identified. The mean ascitic fluid PMN count in SBP patients was 518.1 ± 158.7 cells/mm³. The SBP prevalence by Child–Pugh class was 17.6% in Class A, 10.9% in Class B, and 14.9% in Class C. By MELD score, the prevalence was highest in the 15–25 group (16.9%), followed by >25 (13.8%) and <15 (9.5%) (Table 4).

Table 4: Prevalence and subtypes of spontaneous bacterial peritonitis (n = 130)

Characteristic	Value
SBP diagnosis	
Culture-positive SBP	11 (8.5)
CNNA	7 (5.4)
MNB	0 (0.0)
No SBP	112 (86.2)
Overall SBP prevalence	
Present	18 (13.8)
Absent	112 (86.2)
PMN ≥250 cells/mm³	18 (13.8)
Mean ascitic fluid PMN (SBP patients)	518.1 ± 158.7 cells/mm³
SBP prevalence by Child–Pugh class	
Class A (n = 17)	3 (17.6)
Class B (n = 46)	5 (10.9)
Class C (n = 67)	10 (14.9)
SBP prevalence by MELD score	
<15 (n = 42)	4 (9.5)
15–25 (n = 59)	10 (16.9)
>25 (n = 29)	4 (13.8)

Values are n (%) unless otherwise stated. SBP = spontaneous bacterial peritonitis; CNNA = culture-negative neutrocytic ascites; MNB = monomicrobial non-neutrocytic bacterascites; PMN = polymorphonuclear leucocytes.

On bivariate analysis, none of the demographic or clinical parameters—age, gender, etiology, Child–Pugh class, MELD score, or serum albumin—showed a statistically significant association with SBP (all p > 0.05). Among clinical presentations, altered sensorium was significantly more frequent in SBP patients (55.6%) compared to non-SBP patients (30.4%; p = 0.036). Fever (83.3% vs 69.6%; p = 0.232), abdominal pain (66.7% vs 56.3%; p = 0.406), and abdominal tenderness (77.8% vs 60.7%; p = 0.164) were also more common in SBP patients, though these differences did not reach statistical significance. The overall in-hospital mortality was 16.2% (21/130). Mortality was higher among SBP patients (27.8%) compared to non-SBP patients (14.3%; p = 0.170). Among SBP patients, mortality was higher in Child–Pugh class C (40.0% vs 12.5%; p = 0.314), MELD ≥20 (42.9% vs 18.2%; p = 0.326), and culture-positive SBP (36.4% vs 14.3% for CNNA; p = 0.596), though none reached statistical significance (Table 5).

Table 5: Association of clinical parameters and symptoms with SBP, and in-hospital mortality outcomes

Factor	SBP present (n = 18)	SBP absent (n = 112)	p-value	Significance
Age group				
≤50 years	10 (55.6)	57 (50.9)	0.713	NS
>50 years	8 (44.4)	55 (49.1)		
Gender				
Male	16 (88.9)	92 (82.1)	0.736†	NS
Female	2 (11.1)	20 (17.9)		
Etiology				
Alcoholic	9 (50.0)	57 (50.9)	0.241	NS
Viral	8 (44.4)	33 (29.5)		
Others	1 (5.6)	22 (19.6)		
Child–Pugh class				
A & B	8 (44.4)	55 (49.1)	0.713	NS
C	10 (55.6)	57 (50.9)		
MELD score				
<20	11 (61.1)	60 (53.6)	0.551	NS
≥20	7 (38.9)	52 (46.4)		
Serum albumin				
≥3.0 g/dl	2 (11.1)	33 (29.5)	0.152†	NS
<3.0 g/dl	16 (88.9)	79 (70.5)		
Clinical presentation	**SBP present (n = 18)**	**SBP absent (n = 112)**	**p-value**	**Significance**
Fever				
Present	15 (83.3)	78 (69.6)	0.232	NS
Absent	3 (16.7)	34 (30.4)		
Abdominal pain				
Present	12 (66.7)	63 (56.3)	0.406	NS
Absent	6 (33.3)	49 (43.8)		
Altered sensorium				
Present	10 (55.6)	34 (30.4)	0.036*	S
Absent	8 (44.4)	78 (69.6)		
Abdominal tenderness				
Present	14 (77.8)	68 (60.7)	0.164	NS
Absent	4 (22.2)	44 (39.3)		
Mortality parameter	**Deaths (n)**	**Mortality rate**	**p-value**	**Significance**
Overall in-hospital mortality (n = 130)				
Survived	109	83.8%		
Died	21	16.2%		
Mortality by SBP status				
SBP present (n = 18)	5	27.8%	0.170†	NS
SBP absent (n = 112)	16	14.3%		
Mortality by Child–Pugh (SBP, n = 18)				
Class A & B (n = 8)	1	12.5%	0.314†	NS
Class C (n = 10)	4	40.0%		
Mortality by MELD (SBP, n = 18)				
MELD <20 (n = 11)	2	18.2%	0.326†	NS
MELD ≥20 (n = 7)	3	42.9%		
Mortality by SBP subtype				
Culture-positive SBP (n = 11)	4	36.4%	0.596†	NS
CNNA (n = 7)	1	14.3%		

Values are n (%) unless otherwise stated. Chi-squared test used for categorical comparisons; †Fisher's exact test used when expected cell count was <5. *p < 0.05 considered significant. NS = not

significant; S = significant; SBP = spontaneous bacterial peritonitis; CNNA = culture-negative neutrocytic ascites.

Discussion

The present study was undertaken to determine the prevalence of spontaneous bacterial peritonitis (SBP) in hospitalised patients with decompensated liver disease and ascites at a tertiary care centre in central India. The overall prevalence of SBP in our study was 13.8%, which is consistent with the widely reported range of 10–30% in the published literature [7–9]. Our finding is comparable to the prevalence of 14.1% reported by Baijal et al. from a multicentre Indian study [19], 12.5% by Sheikhbahaei et al. in a systematic review [20], and 15% reported by Oladimeji et al. [21]. However, some studies have reported higher prevalence rates, such as Bhuiyan et al. (26%) [22], which may reflect differences in patient selection criteria, disease severity at presentation, and local referral patterns.

The demographic profile of our study population was characterised by a male predominance (83.1%) with a mean age of 48.4 ± 12.0 years. This is consistent with the known epidemiology of liver cirrhosis in India, where alcohol-related liver disease predominantly affects middle-aged men [1,2]. The male:female ratio of 4.9:1 in our study is similar to that reported by Khan et al. (4.5:1) [23] and Puri et al. (5.1:1) [24]. The finding that nearly half of the patients were illiterate and belonged to the lower socio-economic strata underscores the association between chronic liver disease and social determinants of health. Alcoholic cirrhosis was the most common etiology in our study (50.8%), followed by hepatitis B (25.4%) and MASLD (17.7%). This etiological profile is consistent with the changing landscape of liver disease in India, where alcohol consumption is a leading cause of cirrhosis, particularly in central and northern regions [2]. The relatively high proportion of MASLD (17.7%) in our cohort reflects the growing burden of metabolic liver disease in the Indian population [25].

The clinical severity of our study population was notable, with 51.5% classified as Child–Pugh class C and a mean MELD score of 19.6 ± 7.8, indicating predominantly advanced liver disease. This is expected in a tertiary care setting where patients are referred for management of complications. The high prevalence of Grade II–III ascites (82.3%) further attests to the severity of decompensation in our cohort. Among the clinical presentations, abdominal distension (89.2%), fever (71.5%), and abdominal pain (57.7%) were the most common symptoms. Notably, altered sensorium was the only clinical feature that was significantly associated with SBP (55.6% vs 30.4%; p = 0.036). This finding has important clinical implications, as it suggests that the development of encephalopathy in a cirrhotic patient with ascites should prompt an urgent search for SBP. This finding is consistent with the observation that SBP can precipitate or worsen hepatic encephalopathy, and conversely, encephalopathy may be the presenting feature of occult SBP. Fever, abdominal pain, and abdominal tenderness, although more frequent in SBP patients, did not reach statistical significance, highlighting the often insidious presentation of SBP and reinforcing the need for routine diagnostic paracentesis.

The microbiological profile in our study showed that gram-negative organisms (54.5%) were slightly more common than gram-positive organisms (45.5%). E. coli and Staphylococcus spp. were the most frequently isolated organisms (27.3% each), followed by Klebsiella pneumoniae (18.2%). While the predominance of gram-negative organisms is consistent with the traditional understanding of SBP microbiology [13], the nearly equal proportion of gram-positive isolates is noteworthy and consistent with the emerging trend of increasing gram-positive SBP reported in recent literature [14,15]. This shifting pattern has implications for the choice of empirical antibiotic therapy and may warrant reconsideration of traditional regimens based on third-generation cephalosporins alone.

The culture positivity rate in our study was 8.5% (11/130), with a substantial proportion of SBP cases (38.9%, 7/18) being culture-negative (CNNA). This is consistent with the known limitation of conventional culture methods in detecting SBP, where culture-negative rates of 30–60% have been reported [6,26].

The use of bedside inoculation of ascitic fluid into blood culture bottles, as performed in our study, has been shown to improve culture yield compared to conventional methods, but a significant proportion of cases remain culture-negative [27].

None of the demographic or clinical parameters examined in our study—including age, gender, etiology, Child–Pugh class, MELD score, and serum albumin—showed a statistically significant association with SBP. However, the trend towards higher SBP prevalence in patients with low serum albumin (<3.0 g/dl; 88.9% vs 70.5%; p = 0.152) is clinically relevant, as hypoalbuminaemia is a well-established risk factor for SBP due to its association with reduced opsonic activity of the ascitic fluid [16,17]. The lack of statistical significance may be attributed to the relatively small number of SBP cases (n = 18) in our study, limiting the statistical power to detect moderate associations.

The overall in-hospital mortality in our study was 16.2%, and was nearly twice as high among SBP patients (27.8%) compared to non-SBP patients (14.3%), although this difference did not reach statistical significance (p = 0.170). Among SBP patients, mortality was substantially higher in those with Child–Pugh class C (40.0% vs 12.5%) and MELD ≥20 (42.9% vs 18.2%), and in those with culture-positive SBP (36.4% vs 14.3% for CNNA). These findings are consistent with the established association between SBP and poor outcomes, particularly in patients with advanced liver disease [10,11]. The higher mortality with culture-positive SBP compared to CNNA aligns with findings by Runyon [6] and suggests that culture positivity may serve as a marker of higher bacterial burden and disease severity.

Limitations: This study has several limitations. First, the cross-sectional design precludes the establishment of causal relationships. Second, the relatively small sample size, particularly the small number of SBP cases (n = 18), limited the statistical power to detect associations with moderate effect sizes. Third, the study was conducted at a single tertiary care centre, which may limit the generalisability of the findings to other settings. Fourth, anaerobic cultures were not performed, which may have led to underestimation of the culture positivity rate. Finally, the PT values reported in this study require verification from the raw data, as the stated SD appears inconsistent with the reported range.

Conclusion

Spontaneous bacterial peritonitis is a common complication among hospitalised patients with decompensated liver disease and ascites, with a prevalence of 13.8% in the present study. The majority of cases were diagnosed by ascitic fluid PMN count alone, with culture-negative neutrocytic ascites accounting for a substantial proportion. Altered sensorium was the only clinical feature significantly associated with SBP, emphasising the need for a high index of suspicion in cirrhotic patients presenting with encephalopathy. The microbiological profile showed a nearly equal distribution of gram-negative and gram-positive organisms, which has implications for empirical antibiotic selection. The in-hospital mortality was higher among SBP patients compared to non-SBP patients, particularly in those with advanced liver disease and culture-positive infections. These findings reinforce the recommendation for routine diagnostic paracentesis in all cirrhotic patients admitted with ascites, regardless of symptoms, to enable early diagnosis and timely initiation of appropriate antibiotic therapy.

Declarations

Ethical approval: The study was approved by the Institutional Ethics Committee of L.N. Medical College and Research Centre, Bhopal, Madhya Pradesh, India.

Informed consent: Written informed consent was obtained from all participants or their legally authorised representatives.

Author contributions: SR conceptualised the study, collected data, and drafted the manuscript. AK and DPS supervised the study. VR, NM, and SS contributed to data collection and critical review of the manuscript. All authors read and approved the final version.

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