Original Research Paper

Volume - 16 | Issue - 07 | July - 2026 | PRINT ISSN No. 2249 - 555X | DOI : 10.36106/ijar

General Medicine

TO STUDY PREVALENCE OF BACTERIAL INFECTION OF ASCITIC FLUID IN CIRRHOTIC PATIENTS

Dr. Palak R. Doshi*	Assistant Professor, Department of General Medicine, Lokmanya Tilak Municipal Medical College & General Hospital, Sion, Mumbai. Ex-Assistant Professor, Department of General Medicine, Smt. B. K. Shah Medical Institute and Research Centre, Sumandeep Vidyapeeth, Vadodara *Corresponding Author
Dr. Pramod R. Jha	Ex-Professor and Head of Department, Department of General Medicine, Smt. B. K. Shah Medical Institute and Research Centre, Sumandeep Vidyapeeth, Vadodara
Dr. Pooja Khamar	Junior Resident, Department of General Medicine, Smt. B. K. Shah Medical Institute and Research Centre, Sumandeep Vidyapeeth, Vadodara

ABSTRACT **Background:** Spontaneous ascitic fluid infection (SAI) is a serious complication of cirrhotic ascites with high morbidity and mortality. **Objectives:** To assess the prevalence, clinical profile, and microbiological characteristics of ascitic fluid infections in cirrhosis. **Methods:** This cross-sectional study included 80 adult cirrhotic patients with ascites. Clinical evaluation and ascitic fluid analysis (cell count, biochemistry, Gram stain, and culture) were performed. SAI was defined as polymorphonuclear leukocyte count ≥250 cells/mm³ and/or positive culture. **Results:** Mean age was 46.6 years with male predominance (81.25%). In this study the most prominent clinical feature was abdominal distension (92.50%). The other features which were commonly seen amongst the study patients included splenomegaly (40.00%), swelling of feet (38.75%), fever (37.50%), pain in abdomen (35.00%) and abdominal tenderness (32.50%). Ethanol induced cirrhosis of liver was the commonest etiology (66.25%), followed by Hepatitis B (13.75%), Hepatitis C (2.50%) and other causes of cirrhosis made up for 17.50%. SAI was present in 30% (24 out of 80) patients, with culture-negative neutrocytic ascites being most common. Only 9 out of the 24 SAI patients had ascitic fluid culture positive with Escherichia coli being the predominant organism in 5 cases, while Acinetobacter and Staphylococcus aureus grew in 2 cases each. Fever, abdominal pain, tenderness, altered sensorium, and asterixis were significantly associated with SAI. Higher leukocyte count, low ascitic fluid protein level, high ascitic fluid neutrophil count and advanced liver disease correlated with infection. **Conclusion:** Spontaneous ascitic fluid infection is a frequent and clinically significant complication in patients with cirrhotic ascites, with a prevalence of 30% (24 out of 80 patients) in the present study. Only 9 out of the 24 SAI patients had ascitic fluid culture positive. Fever, pain in abdomen, abdominal tenderness, altered sensorium, asterixis, a high total leucocyte count, low ascitic fluid protein level and high ascitic fluid neutrophil count were found to have significant correlation with the presence of SAI (p value ≤0.05). There was a trend of more frequent occurrence of SAI in patients with advanced liver disease (CTP class C and high MELD score). Recognition of these predictors and early diagnosis can guide appropriate treatment of SAI, and thus reduce the morbidity and mortality of such patients.

KEYWORDS : Cirrhosis, Spontaneous Ascitic Fluid Infection, Spontaneous Bacterial Peritonitis.

INTRODUCTION

Cirrhosis is the end stage of chronic liver disease, characterized by fibrosis, architectural distortion, and regenerative nodules. Diagnosis is based on clinical, laboratory, and imaging findings, with common causes including alcohol, viral hepatitis, and non-alcoholic steatohepatitis.[1]

Decompensated cirrhosis is marked by complications such as ascites, variceal bleeding, and encephalopathy, indicating poor prognosis.[2] Ascites, the most frequent complication, occurs predominantly due to cirrhosis and reflects disease progression.[3]

Spontaneous bacterial peritonitis (SBP) is a common and severe infection in cirrhotic patients with ascites, with a prevalence of 10–30% in hospitalized cases. Gram-negative organisms, especially *Escherichia coli* and *Klebsiella* species, predominate, though Gram-positive infections are increasingly observed.[4]

NEED FOR THE STUDY

Ascitic fluid infections increase the risk of complications such as encephalopathy, hepatorenal syndrome, and acute-on-chronic liver failure, leading to high morbidity and mortality.[5] Early diagnosis through paracentesis and prompt antibiotic therapy significantly improve outcomes.[6]

Studying the prevalence and microbiological profile of ascitic fluid infections is essential for guiding empirical therapy and reducing mortality in cirrhotic patients.

MATERIALS AND METHODS

This cross-sectional study was conducted at Dhiraj Hospital, Vadodara, Gujarat over 18 months. A sample size of 80 was calculated (95% confidence interval, 80% power, 10% expected prevalence). Institutional ethics committee permission was obtained (SVIEC/ON/Medi/BNPG27/018188).

Adult cirrhotic patients with ascites were included after informed consent. Patients with contraindications to paracentesis, prior antibiotic use, pregnancy, or non-cirrhotic ascites were excluded.

Clinical evaluation and baseline investigations (CBC, liver and renal function tests, electrolytes, coagulation profile, viral markers) were performed. Ultrasonography confirmed cirrhosis and ascites.

Diagnostic paracentesis was done under aseptic precautions before antibiotics. Ascitic fluid (AF) was analysed for cell counts, biochemistry, Gram stain, culture, and serum ascites albumin gradient (SAAG).

Spontaneous ascitic fluid infection (SAI) was defined as polymorphonuclear leucocyte (PMNL) ≥250 cells/mm³ and/or positive culture without secondary peritonitis. Spontaneous bacterial peritonitis (SBP) was characterised by ascitic fluid infection with PMNL count >250 cells/mm^3 with / without positive bacterial culture in the absence of any intraabdominal surgically treatable source of infection. Culture negative neutrocytic ascites (CNNA) was defined as a negative ascitic fluid culture with an ascitic fluid PMNL count of >250 cells/mm^3 with no other explanation for the elevated PMNL count forthcoming. Monomicrobial non-neutrocytic bacterascites (MNB) constituted a positive ascitic fluid culture for a single organism with a PMNL count of <250 cells/mm^3 with no intraabdominal surgically treatable cause of infection. Secondary bacterial peritonitis was identified with a positive ascitic fluid culture for more than one organism with PMNL count >250 cells/mm^3 and a surgically treatable source of infection. Polymicrobial bacterascites referred to ascitic fluid culture positive for several pathogens but PMNL count <250 cells/mm^3.

Data were analysed using appropriate statistical tests, with p ≤0.05 considered significant.

RESULTS

Table 1 – Patient profile

Age group (years)	No. of patients (n = 80)	%
≤20	1	1.25
21-40	25	31.25
41-60	47	58.75
>60	7	8.75
Gender	No. of patients (n = 80)	%
Female	15	18.75%
Male	65	81.25%
Clinical Features	No. of Patients (n = 80)	%
Fever	30	37.50%
Abdominal distension	74	92.50%
Pain in abdomen	28	35.00%
Abdominal Tenderness	26	32.50%
Swelling of feet	31	38.75%
Decreased urine output	18	22.50%
Breathlessness	16	20.00%
Malena	19	23.75%
Hematemesis	7	8.75%
Altered sensorium	22	27.50%
Asterixis	21	26.25%
Splenomegaly	32	40.00%

As seen in table 1, a total of 80 cirrhotic patients with ascites were studied, with a male predominance (81.25%) and mean age of 46.6 years; most patients (58.75%) were aged 41–60 years.

Abdominal distension (92.5%) was the most common presentation, followed by splenomegaly (40%), pedal edema (38.75%), fever (37.5%), pain in abdomen (35.00%) and abdominal tenderness (32.50%). Other features seen in the study patients included altered sensorium (27.5%), asterixis (26.25%), malena (23.75%), decreased urine output (22.5%), breathlessness (20%), and hematemesis (8.75%).

Ethanol was the leading etiology of cirrhosis (66.25%), followed by hepatitis B (13.75%), hepatitis C (2.50%) and other causes accounting for 17.50%.

Table 2 - Types of spontaneous ascitic fluid infection (SAI)

Types of SAI	Culture positive SBP	Culture negative neutrocytic ascites (CNNA)	Monomicrobial non-neutrocytic bacterascites (MNB)	Polymicrobial bacterascites	Secondary bacterial peritonitis
No. of patients (n = 24)	5	15	4	0	0
% (n = 24)	20.83%	62.50%	16.67%	0.00%	0.00%

As seen in table 2, Spontaneous ascitic fluid infection (SAI) was observed in 24 (30%) of patients. In these 24 patients with SAI, 62.5% patients were seen to have predominantly culture-negative neutrocytic ascites (CNNA), while culture-positive SBP and monomicrobial bacterascites accounted for 20.8% and 16.7%, respectively.

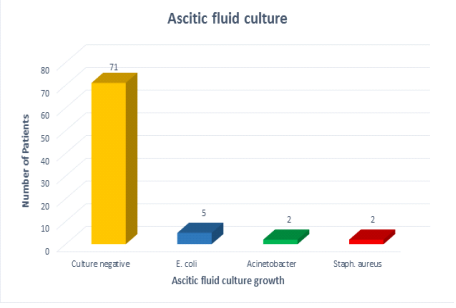

Figure 1 – Ascitic Fluid culture

As in figure 1, among the 80 patients in the study, only 9 patients had positive AF culture. E. coli was the most frequesnt isolate (5 patients), Acinetobacter and Staph. aureus were found in 2 of the patients each.

Table 3 - Comparison of clinical features and their correlation with SAI

Clinical Feature	Total no. of Patients (n = 80)	% (n = 80)	Patients with SAI (n = 24)	% (n = 24)	Patients without SAI (n = 56)	% (n = 56)	p value
Fever	30	37.50%	23	83.33%	7	17.85%	0.003
Abdominal distension	74	92.50%	22	91.67%	52	92.86%	NA
Pain in abdomen	28	35.00%	22	75.00%	6	17.85%	0.002
Abdominal Tenderness	26	32.50%	21	70.83%	5	16.07%	0.001
Swelling of feet	31	38.75%	9	37.50%	22	39.28%	0.464
Decreased urine output	18	22.50%	5	20.83%	13	23.21%	0.059
Breathlessness	16	20.00%	5	20.83%	11	19.64%	0.133
Melena	19	23.75%	6	25.00%	13	23.21%	0.108
Hematemesis	7	8.75%	2	8.33%	5	8.93%	NA
Altered sensorium	22	27.50%	19	62.50%	3	12.50%	0.006
Asterixis	21	26.25%	15	62.50%	6	10.71%	0.049
Splenomegaly	32	40.00%	9	37.50%	23	41.07%	0.808

Table 3 shows comparison of clinical features between patients with and without SAI. Fever, abdominal pain, tenderness, altered sensorium, and asterixis were significantly more common in SAI ($p<0.05$).

Table 4 – Comparison of laboratory parameters and their correlation with SAI

Laboratory Parameters	Patient with SAI -Mean	SD	Patient without SAI -	SD	p value
Hemoglobin	9.82	2.36	9.72	2.12	0.872
TLC (cells/cumm)	13795.00	7421.77	9905.00	5390.56	0.040
Platelets (lac	1.98	1.31	2.12	1.19	0.656
Sr. Bilirubin (mg/dl)	4.211	5.21	3.35	5.10	0.494
SGOT (units/liter)	58.70	46.99	55.25	48.25	0.768
SGPT	31.80	18.72	28.70	19.17	0.506
Urea (mg/dl)	43.50	47.08	41.17	21.78	0.764
Creatinine (mg/dl)	1.34	1.81	1.10	0.62	0.379
INR	1.37	0.33	1.31	0.31	0.500
Sr. Albumin (g/dl)	2.72	0.67	2.80	0.47	0.543

Table 4 shows comparison of laboratory parameters between patients with and without SAI. Total leukocyte count was significantly higher in patients with SAI ($p=0.04$), while biochemical parameters showed no significant difference.

Table 5 - Comparison of ascitic fluid parameters and their correlation with SAI

Laboratory Parameters	Patient with SAI -Mean	SD	Patient without SAI - Mean	SD	p value
AF ADA (units/liter)	16.55	21.91	16.09	13.45	0.924
AF Protein (g/dl)	1.40	0.25	1.71	0.12	0.01
AF Neutrophils (cells/cumm)	916.85	665.76	88.85	74.86	0.001

As seen in Table 5, ascitic fluid protein was significantly lower and neutrophil count significantly higher in patients with SAI (p=0.01 and p=0.001, respectively).

Table 6 – Comparison of Child Turcotte Pugh (CTP) Score and its correlation with SAI

CTP Score	Total No of Patients (n = 80)	% (n = 80)	Patients with SAI (n = 24)	% (n = 24)	Patients without SAI (n = 56)	% (n = 56)	Chi-square value (x2)	p value
Class A	12	15.00%	0	0.00 %	12	21.42 %	27.75	<0.001
Class B	46	57.50%	8	33.33 %	38	47.50 %		
Class C	22	27.50%	16	66.67 %	6	10.71 %		

Table 7 – Comparison of Model for End Stage Liver Disease (MELD) Score and its correlation with SAI

Score	Patient with SAI -Mean	SD	Patient without SAI - Mean	SD	p value
MELD Score	20.95	10.13	15.72	8.81	0.022

Table 6 & 7 shows, higher disease severity correlated with SAI, with most cases in Child-Pugh class C (p value <0.001) and higher mean MELD score (20.95 vs 15.72; p=0.022).

DISCUSSION

Spontaneous ascitic fluid infection (SAI) is a major complication of cirrhosis, contributing significantly to morbidity and mortality due to immune dysfunction and bacterial translocation. Recent studies show that infections markedly worsen outcomes in cirrhotic patients despite therapeutic advances.[7]

In the present study, male predominance and middle-aged distribution were observed, consistent with earlier reports, likely reflecting ethanol-related cirrhosis.[8–10] Ethanol was the leading etiology, in accordance with regional data, although global variation exists with viral hepatitis predominance in endemic regions.[10]

The prevalence of SAI (30%) is comparable to reported rates of 10–30% in hospitalized patients.[11] Culture-negative neutrocytic ascites was the most common subtype, as described in recent literature.[7,11] *Escherichia coli* remained the predominant organism, though changing microbiological patterns and rising resistance have been increasingly reported.[7,12]

Fever, abdominal pain, tenderness, and hepatic encephalopathy were significantly associated with SAI, supporting their diagnostic relevance. Elevated total leukocyte count further correlated with infection. Low AF protein and high AF neutrophil counts were strongly associated with SAI, reflecting impaired host defense mechanisms.[7,13]

No significant association was observed between etiology of cirrhosis and SAI. However, higher CTP and MELD scores showed strong correlation with SAI, indicating increased risk with advanced liver disease.[11,13]

Overall, early diagnostic paracentesis and prompt empirical therapy remain crucial for improving outcomes in cirrhotic patients with ascites.

CONCLUSION

Spontaneous ascitic fluid infection is a frequent and clinically significant complication in patients with cirrhotic ascites, with a prevalence of 30% (24 out of 80 patients) in the present study. Only 9 out of the 24 SAI patients had ascitic fluid culture positive. Fever, pain in abdomen, abdominal tenderness, altered sensorium, asterixis, a high total leucocyte count, low ascitic fluid protein level and high ascitic fluid neutrophil count were found to have significant correlation with the presence of SAI (p value ≤0.05). There was a trend of more frequent occurrence of SAI in patients with advanced liver disease (CTP class C and high MELD score). Recognition of these predictors and early diagnosis can guide appropriate treatment of SAI, and thus reduce the morbidity and mortality of such patients.

Declaration of conflicting interest

The authors declare that there is no conflict of interest.

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

REFERENCES :

1. Bosch J, Abraldes JG, Berzigotti A, García-Pagán JC. Semin Liver Dis. 2008;28(1):3–25.
2. D'Amico G, Garcia-Tsao G, Pagliaro L. J Hepatol. 2006;44(1):217–31.
3. Runyon BA. Hepatology. 2009;49(6):2087–107.
4. Fernández J, Acevedo J, Wiest R, et al. J Hepatol. 2012;56(1):S1–12.
5. Piano S, Singh V, Caraceni P, et al. J Hepatol. 2019;70(1):126–38.
6. Tandon P, Garcia-Tsao G. Hepatology. 2011;54(6):2083–92.
7. Piano S, Singh V, Caraceni P, et al. Epidemiology and effects of bacterial infections in cirrhosis. J Hepatol. 2019;70(1):126–38.
8. Kasztelan-Szczerbinska B, et al. Clin Exp Hepatol. 2015;1(1):1–7.
9. Mohan P, et al. J Clin Exp Hepatol. 2016;6(3):192–7.
10. Duah A, et al. Pan Afr Med J. 2019;33:213.
11. Fernández J, Acevedo J, Wiest R, et al. Bacterial infections in cirrhosis. J Hepatol. 2012;56(S1):S1–12.
12. BMC Infect Dis Study Group. Changing bacterial profile in SBP. BMC Infect Dis. 2024;24:528.
13. Sbeit W, et al. Advances in diagnosis of SBP. Clin Exp Med. 2024;24:25.