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# Profile of spontaneous bacterial peritonitis in non-alcoholic fatty liver disease cirrhosis

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## ABSTRACT

**Background:** Etiology of chronic liver disease (CLD) has an impact on the profile of spontaneous bacterial peritonitis (SBP) in terms of infection acquisition, resistance pattern, and treatment outcome. **Aims and Objectives:** This study aimed to study infection acquisition, antibiotic resistance pattern, and treatment outcome in patients with non-alcoholic fatty liver disease (NAFLD) cirrhosis with SBP. **Materials and Methods:** This observational prospective study was carried out in Government Medical College; Srinagar: over a period of 2 years from 2017 onward. We did analysis of patients with NAFLDs cirrhosis. We studied infection acquisition, antibiotic resistance pattern, and treatment outcome in this group of patients. **Results:** Over 2-year period, 246 patients were enrolled. The mean age of patients was 57.09 ± 13.90 years. Hepatitis B virus and NAFLD were the major etiological contributors to the burden of CLD amounting to 51.20% in Kashmir. SBP was present in 33/57 (57.90%) of NAFLD cirrhosis patients. Child-Turcotte-Pugh Class C was 63.60%. Patients with culture-positive SBP (CP-SBP) were 45.45%. In patients of SBP with etiology other than NAFLD, CP-SBP was 61% whereas culture-negative SBP was 38.77%. **Conclusion:** In our region, NAFLD is one of the leading contributors to CLD. In this study, the trend toward worse treatment outcome and mortality in the patients with NAFLD SBP was observed.

**Key words:** Chronic liver disease; Culture positive; Non-alcoholic fatty liver disease; Ascites and spontaneous bacterial peritonitis

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## INTRODUCTION

Spontaneous bacterial peritonitis (SBP) is an infection of the previously sterile ascitic fluid (AF), without any apparent intra-abdominal source of infection in patients with chronic liver disease (CLD).[1] It was first described by Conn and Fessel in patients with hepatic cirrhosis in 1906–1907.[2] The prevalence of SBP varies from 1.5% to 3.5% in outpatients and 10–30% in hospitalized patients.[3,4] Factors associated with SBP include age, history of SBP,[4] and gastrointestinal bleeding.[4,5] The severity of liver dysfunction scores including the Child-Turcotte-Pugh (CTP) score or model for end-stage liver disease (MELD) score, neutrophil count, low-protein concentration (<1.5 g/dL) in the AF, and long-term proton pump inhibitors (PPIs) use has been reported as a predictive factor[6-11] for SBP. In-hospital mortality for the first episode of SBP is 10–50%, depending on various risk factors.[12,13] Recurrence rates are high, more than 70% within 1 year.[14,15] In cirrhosis, disturbance in the microcirculation of intestinal mucosa, results in a reduction of mucosal blood flow, intestinal bacterial overgrowth, impaired mucosal integrity[16-18] and deficiencies in local host immune defenses are possible mechanisms for bacterial translocation.[19,20] Catheters and other equipment used during invasive procedures represent other possible sources of infection. The gold standard for diagnosis of SBP consists of count ≥250 cells/mm$^3$ and/or a positive AF culture without any evidence of intra-abdominal infectious source. Culture-negative SBP (CN-SBP) is defined as negative AF culture with a neutrophil count of ≥250 cells/mm$^3$ in AF.[21] Culture-positive SBP (CP-SBP) is seen in 35–65% of SBP patients.[22-26] Enteric bacteria

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are the most common etiological agent.[17] Frequency of multidrug resistance (MDR), extended drug resistance (XDR), and pan drug resistance (PDR) bacteria in hospital care associated SBP (HCA-SBP), hospital-acquired SBP (HA-SBP) is 20–35%[11,27] and 4–16% in community-acquired SBP (CA-SBP).[28]

### Aims and objectives

To study the infection acquisition, antibiotics resistance pattern, treatment success, and mortality in patients with non-alcoholic fatty liver disease (NAFLD)-related cirrhosis with SBP.

## MATERIALS AND METHODS

This prospective observational study was conducted in the department of Gastroenterology and Hepatology, Superspeciality Hospital, Shreenbagh, Srinagar. It is a 27 bedded department with round-the-clock gastroenterology services. All patients of cirrhosis and ascites with the possibility of SBP more than 10 years of age were recruited from outpatient Department of Gastroenterology and Hepatology and Medical Emergency of the Government Medical College, Srinagar over 1-year period. A pre-designed structured pro forma was used to record patient's demographics, clinical presentation, and laboratory results. We did subgroup analysis of patients with NAFLD-related cirrhosis.

### Exclusions

Etiology of ascites other than liver disease, recent antibiotics use (within 2 weeks), suspected or conformed intra-abdominal source of infections such as surgery or trauma, children under 10 years of age, and those who did not consented to participate.

Paracentesis (only diagnostic tap) was performed bedside with under mentioned protocol:

1. Performed using standard aseptic precaution for all study participants
2. Twenty milliliter syringes with 20 G (gauge) needle used for AF tap in left iliac fossa or midline below umbilicus at bedside
3. A total 20 mL AF was collected from each patient
4. 10 mL for AF detailed biochemical and cytological report
5. 10 mL of AF inoculated in blood culture bottles at the bedside using an aseptic technique and send for microbiology (for aerobic and anaerobic culture)
6. Blood sample (10 mL) was collected at the same time to perform serum/plasma-based blood workup as deemed necessary.

The severity of liver disease was assessed by CTP score. It depends on the sum of these five variables patients are divided into three classes; A (score of 5–6), B (score of 7–9), and C (score of 10–15). Class A has 1 year survival of 100% and 2 year survival of 90%. Class B has 1 year survival of 81% and 2 year survival of 57%. Class C has 1 year survival of 45 % and 2-year survival of 35% (Table 1).

Infections diagnosed on admission or within 2 days after admission were classified as HCA in patients with a prior contact with the healthcare environment (hospitalization or short-term admission for at least 2 days in the previous 90 days, residence in a nursing home or a long-term care facility or chronic hemodialysis). The infection was considered CA when present at the time of admission or developed within the first 2 days after hospitalization with no history as mentioned above in HCA and HA when the diagnosis was made thereafter.[29,30]

MDR was defined as non-susceptibility to at least one agent in 3 or more antimicrobial categories. XDR was defined as non-susceptibility to at least one agent in all but 2 or fewer antimicrobial categories and PDR as non-susceptibility to all currently available agents.[31]

### Data analysis

Statistical analysis was conducted using the Statistical Package for the Social Sciences (SPSS) version 16.0 for Windows (SPSS, Chicago, IL). Categorical variables were compared using the Chi-square or Fisher's exact test where appropriate. Continuous data were compared using the t-test or the Mann–Whitney test, the Kruskal–Wallis test was used for multiple comparisons, when appropriate. Quantitative variables with a normal distribution were expressed as mean values±standard deviation and those with a non-normal distribution as median values (range). The significance level was two sided and set to <0.05.

Informed consent was obtained from all participants or their attendants.

This study was cleared by institution's review board.

## RESULTS

The prevalence of SBP in CLD presenting at our center was 38.09%. The mean age of patients was 59.09±12.90 years

**Table 1: Child-Turcotte-Pugh score**

| Parameter | 1 | 2 | 3 |
|---|---|---|---|
| Encephalopathy | None | Stage 1–2 | Stage 3–4 |
| Ascites | None | controlled | Poor control |
| Serum bilirubin (mg/dL) | <2 | 2–3 | ≥3 |
| Serum albumin (g/dL) | >3.5 | 3–3.5 | <3 |
| Prothrombin time/INR | 0–4/<1.7 | 5–6/1.7–2.3 | >6/>2.3 |

INR: International normalized ratio

with a minimum of 20 and a maximum of 89. Males were 57.3% and females were 42.7%. The most common clinical presentations were ascites 100% and hepatic encephalopathy 89%. Our data shows an etiological profile different from rest of the India.

NAFLD is one of the major etiological contributors to the burden of CLD amounting to 57(23%) in Kashmir, that is, one-fourth of cases (Table 2).

We studied infection acquisition, antibiotic resistance pattern, treatment success, and mortality in NAFLD cirrhosis with SBP (Table 3).

Out of 57 with NAFLD cirrhosis 33 (58%) had SBP. CTP Class A, B, and C were 3.03%, 6.06%, and 63.60%, respectively. Patients with CP-SBP were 15 (45.45%) and CN-SBP was 18 (54.54%). In patients of SBP with etiology other than NAFLD, CP-SBP was 61% whereas CN-SBP was 38.77%. Culture positivity rates were higher in patients of SBP with etiology of CLD other than NAFLD.

CA-SBP, HCA-SBP, and HA-SBP were detected in 20 (60.60%), 11 (33.33%), and 2 (6.06%) of patients with NAFLD cirrhosis, respectively, whereas CA-SBP, HCA-SBP, and HA-SBP were detected in 35 (71.42%), 13 (26.53%), and 1 (2.02%) of patients with other etiology cirrhosis, respectively. There was an increased frequency of HA-SBP in patients with NAFLD cirrhosis.

Drug sensitive (DS)-SBP was seen in 6 (18.18%) and 17 (34.69%) of patients with NAFLD cirrhosis and other etiology cirrhosis, respectively. MDR-SBP was seen in 7 (21.21%) patients with NAFLD cirrhosis whereas 10 (20.40%) had MDR-SBP in other etiology cirrhosis, respectively. XDR-SBP was seen in 2 (6.06%) patients with NAFLD cirrhosis whereas 3 (6.12%) had XDR-SBP in other etiology cirrhosis, respectively. DS infections were more frequent in other etiology cirrhosis groups whereas drug resistance was more frequently encountered in patients with NAFLD cirrhosis. However, this difference was not statistically significant.

Cure rate of SBP in NAFLD cirrhosis was 24 (72.70%). Cure rate was 42 (85.70%) in patients with other etiology cirrhosis. Mortality was 9/33 (27%) in SBP patients with NAFLD cirrhosis whereas only 7 (15%) in patients with other etiology cirrhosis. In this study, mortality was higher in SBP patients with NAFLD cirrhosis.

**Table 2: Etiological profile of CLD in the study group**

| S. No. | Etiology | n | % |
|---|---|---|---|
| 1 | Chronic Hepatitis B | 69 | 28.04 |
| 2 | Non-alcoholic fatty liver disease | 57 | 23.17 |
| 3 | Chronic Hepatitis C | 42 | 17.03 |
| 4 | Cryptogenic liver disease | 33 | 13.41 |
| 5 | Recurrent pyogenic cholangitis | 21 | 8.53 |
| 6 | Non-cirrhotic portal fibrosis | 12 | 4.87 |
| 7 | Autoimmune hepatitis | 9 | 3.65 |
| 8 | Alcoholic liver disease | 3 | 1.21 |
| | | 246 | |

## DISCUSSION

Identifying and managing decompensating events in stable cirrhotic patients poses a perennial challenge. These events, including variceal bleeding, rising bilirubin levels, hepatic encephalopathy, development of ascites, and hepatorenal syndrome, mark significant milestones in disease progression.[32] SBP stands out as a dire complication within the continuum of decompensated liver disease, bearing high immediate, and 1-year mortality rates.[32,33] Consequently, liver transplantation becomes imperative,

**Table 3: SBP in NAFLD cirrhosis**

| Total SBP for analysis (n=82) | SBP with NAFLD cirrhosis (33) (%) | SBP with other etiology cirrhosis (49) (%) |
|---|---|---|
| Child Class A (9) | 1 (3.03) | 8 (16.32) |
| Child Class B (13) | 2 (6.06) | 11 (22.44) |
| Child Class C (60) | 21 (63.63) | 39 (79.59) |
| Culture positive (45) | 15 (45.45) | 30 (61.22) |
| Culture negative (37) | 18 (54.54) | 19 (38.77) |
| CA-SBP (55) | 20 (60.60) | 35 (71.42) |
| HCA-SBP (24) | 11 (33.33) | 13 (26.53) |
| HA-SBP (3) | 2 (6.06) | 1 (2.04) |
| DS-SBP (23) | 6 (18.18) | 17 (34.69) |
| MDR-SBP (17) | 7 (21.21) | 10 (20.40) |
| XDR-SBP (5) | 2 (6.06) | 3 (6.12) |
| Cured (66) | 24 (72.72) | 42 (85.71) |
| Death (16) | 9 (27.27) | 7 (14.82) |

CA: Community acquired, SBP: Spontaneous bacterial peritonitis, HCA: Hospital care associated, HA: Hospital acquired, DS: Drug sensitive, MDR: Multidrug resistant, XDR: Extended drug resistant, PDR: Pan drug resistant

contingent on adequate control of SBP. However, the treatment landscape for SBP has become increasingly complex in recent years, fraught with challenges stemming from drug-resistant and fungal infections.[34,35] The and antibiotic usage further underscores the need for a comprehensive evaluation of SBP patterns within this demographic. Our study reveals a lower incidence of culture-positive SBP in NAFLD compared to other NAFLD etiologies (45.45% vs 61.22%), alongside a higher prevalence of healthcare- multifaceted nature of this complication implicates a myriad of contributory factors, including antibiotic overuse, excessive health-care interactions, nutritional deficiencies, PPI overutilization, and advanced CTP score.[36,37] Yet, the literature remains ambiguous regarding the precise etiological underpinnings of SBP and its implications on culture positivity and treatment resistance. Notably, the correlation between etiology and SBP subtypes- CA-SBP, MDR, and XDR remains underexplored. NAFLD emerges as a prominent precursor to cirrhosis, often accompanied by a constellation of comorbidities such as diabetes mellitus, cardiovascular diseases, kidney disorders, and obstructive sleep apnoea.[38] The confluence of excessive hospitalizations associated SBP, MDR, and XDR SBP. SPB has been associated with poor prognosis[39] and despite the progress made in its management and prevention, the rate of mortality among hospitalized patients' was 37%,[14] with 1-year mortality rate estimated at up to 50% in some studies.[40] Several factors have been reported to impact mortality in this cohort of patients. These include nosocomial infections, sepsis and septic shock, acute kidney injury,[41] and diagnosis as well as need for hospitalization among others. The renal dysfunctions and the MELD are among the range of predictive algorithms that are suggested as predictors of mortality in hospitalized patients with cirrhosis and SBP.[24] In addition, a concerning trend toward decreased cure rates and elevated mortality rates is observed in NAFLD-related cirrhosis with SBP, warranting further investigation into optimal management strategies for this subset of patients.

### Limitations of the study

Sample size was small.

## CONCLUSION

The formidable challenges posed by resistance patterns such as MDR and XDR strains, coupled with their associated high mortality rates and low cure rates, underscore the urgent need for large-scale, meticulously designed studies focusing on these critical aspects of SBP in patients with NAFLD-related CLD.

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**MMM**, **ZAW**- Definition of intellectual content, literature survey, prepared the first draft of manuscript. **MMM, UNB, WIK**- Implemented of study protocol, data collection, data analysis, manuscript preparation, and submission of article; **ZAW, MMM, UNB, WIK**- Concept, design, clinical protocol, manuscript preparation, editing, and manuscript revision; **MMM, WIK, UNB, NHH**- Design of study, statistical analysis and Interpretation; **MMM, ZAW, NHH**- Review manuscript; **ZAW, NHH**- Review manuscript; **ZAW, UNB, MMM**- Literature survey and preparation of table; **ZAW, UNB, WIK, NHH**- Coordination, and manuscript revision.

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