ORIGINAL RESEARCH

A Study to Estimate Spontaneous Bacterial Peritonitis in Chronic Liver Disease

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ABSTRACT

Introduction: Spontaneous bacterial peritonitis (SBP) is an infection of initially sterile ascitic fluid without a detectable, surgically treatable source of infection. We analyzed the prevalence, clinical and laboratory features of SBP in 100 patients of chronic liver disease to identify risk factors for incidence and mortality. **Material and methods:** One hundred patients (mean age 46 years, 92% males) with chronic liver disease and ascites were studied in our prospective study during the period from October 2013 to November 2014 in Gajra Raja Medical College, Gwalior, Madhya Pradesh. Diagnosis of SBP was based on: An ascitic fluid polymorphonuclear leukocyte (PMN) count ≥250/mm^3 *and* ascitic fluid culture positive for single microorganism *With* An absence of source of infection in abdomen. Clinical features of the patients were studied on the basis of history and clinical examination. Relevant blood studies were sent as soon as possible and results analyzed. **Results:** Overall prevalence of SBP was found to be 22% in our study. Symptoms significantly associated with increased incidence of SBP were icterus (p value 0.036), altered sensorium (p value 0.012) and abdominal tenderness (p value 0.003). Higher Child-Pugh grade (Grade C 18.4%) and increased Model for End-stage Liver Disease (MELD) (p value 0.0009) score were associated with higher risk of developing SBP. Also, increasing MELD score was associated with a higher mortality (p value 0.032). SBP +ve group was associated with an increased mortality (p value 0.01) as compared to SBP -ve group. Mortality in SBP was strongly associated with higher serum creatinine level (p value 0.0006). **Conclusion:** Icterus, altered sensorium, abdominal tenderness, and raised creatinine were associated with increased risk of SBP. Child-Pugh grade and MELD score were associated with increased risk of SBP in cirrhosis in the present study. MELD score was found to be significantly associated with mortality in SBP.

Keywords: Chronic liver disease, spontaneous bacterial peritonitis, cirrhosis

Cirrhosis of liver is one of the most common conditions affecting the liver chronically and causing a very high mortality and morbidity, leading to a great burden affecting both quality of life and longevity[1]. It predisposes the patient to a number of complications, one of the most common being ascites[2,3]. Ascites itself has a number of complications, like ascitic fluid infection, cardiorespiratory embarrassment and umbilical hernia. One of the form of ascitic fluid infection is spontaneous bacterial peritonitis (SBP). SBP is an infection of initially sterile ascitic fluid without identifiable, surgically treatable source of infection[4]. This severe complication of cirrhotic ascites was first described in the mid-1960s[5]. Along with hepatorenal syndrome, it is stated as most common life-threatening complication in cirrhosis[6]. Culture-negative neutrocytic ascites (CNNA) - ascites is sterile, bacterial infection is not demonstrable by culturing; only an increased number of polymorphonuclear leukocytes (PMNs) above the limit of 250 cells/mm^3 is revealed. Monomicrobial non-neutrocytic bacterascites (or only bacterascites) has rarely been described. In this disorder, positive bacterial cultivation is presented without increased leukocytes.

SBP and CNNA are identical, both from the clinical perspective as well as therapeutic approach. The consensus conference of the International Ascites Club[7] thus recommends not to differentiate between these two entities. Even in case of CNNA, SBP is talked about, and a raised number of neutrophils in ascites is enough for the diagnosis. A spontaneous infection complicating

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ascites may appear even in malignant ascites[8]; however, it is found most often in cirrhotic ascites.

SBP can present as a full blown syndrome with fever, hypotension, abdominal pain, abdominal tenderness, altered mentation or one or more of its components may be missing[6,9-11]. So, all cirrhotic patients must be screened for SBP with ascitic fluid analysis, PMN count and culture of ascitic fluid[12-15]. Patients must be treated aggressively with antibiotics as they have poor prognosis and high mortality if not treated early.

Present study was undertaken to identify SBP in chronic liver disease patients with ascites with focus on prevalence, presenting features and laboratory findings to identify risk factors for SBP in cirrhotic patients and risk factors for mortality in SBP patients.

MATERIAL AND METHODS

One hundred patients with chronic liver disease and ascites were studied during the period from October 2013 to November 2014. The results obtained were subjected to standard statistical methods for analysis and relevant conclusions were drawn from them. Results were expressed as mean (± standard deviation [SD]). Chi-square test was used wherever applicable. P value ≤0.05 was considered statistically significant.

Inclusion Criteria

- All the cases of chronic liver disease with ascites on the basis of clinical, laboratory and radiological features suggestive of chronic liver disease.

Exclusion Criteria

- Patients found to have a secondary cause of peritonitis like ruptured liver abscess, perinephric abscess, etc.
- Patients found to have more than one organism in ascitic fluid culture.
- Patients who had an antibiotic treatment within last 10 days.
- Patients having a history of recent paracentesis within 10 days.

Diagnosis of SBP

Diagnosis of SBP was based on:

An ascitic fluid PMN count ≥250/mm^3
And
Ascitic fluid culture positive for single microorganism
With
An absence of source of infection in abdomen.

All patients underwent paracentesis within 24 hours of admission. About 30 mL of ascitic fluid was aspirated with aseptic precautions; 10 mL of fluid was sent to laboratory in sterile condition for conventional culture; 10 mL of ascitic fluid was utilized for biochemical and cytological examination.

Clinical features of the patients were studied on the basis of history and clinical examination. Relevant blood studies were sent as soon as possible and results analyzed.

RESULTS

Most of the patients studied were males (n = 92; Table 1).

Age of the patients studied ranged from 22 to 85 years, with the most number of patients in the age group of 40 to 49 years (n = 41); mean age of the patients studied was 46.81 ± 13.18 years (Table 2).

Among 100 patients of chronic liver disease studied, 22 patients showed >250 PMN/mm^3 of ascitic fluid (Table 3). Of these 22 patients, half of them (n = 11) showed ascitic fluid culture positive for single microorganism. Including its variants, overall prevalence of SBP was found to be 22%.

This makes up the prevalence of classical SBP to be 11% and the prevalence of CNNA to be 11% as well. None of the patients who had ascitic fluid PMN count <250/mm^3 showed positive ascitic fluid culture, so there

Table 1. Gender Distribution of Patients Studied

Sex	No. of cases	Percentage (%)
Female	8	8.0
Male	92	92.0
Total	**100**	**100.0**

Table 2. Age Distribution of Patients Studied

Age in years	No. of cases	Percentage (%)
20-29	6	6.0
30-39	18	18.0
40-49	41	41.0
50-59	15	15.0
60-69	9	9.0
70-79	9	9.0
>80	2	2.0
Total	**100**	**100.0**

Table 3. Prevalence of Spontaneous Bacterial Peritonitis

Total no. of patients (100)	Ascitic PMN count >250/mm^3		Ascitic PMN count <250/mm^3		Polymicrobial bacterascites
	Culture positive	Culture negative	Culture positive	Culture negative	
	SBP	CNNA	Bacterascites	Ascites	
100	11	11	0	78	0

was no patient with monomicrobial non-neutrocytic bacterascites. None of the patients showed ascitic fluid culture positive for more than one microorganism, so no patient of polymicrobial bacterascites was seen.

All of the patients (Table 4) who were positive for SBP (n = 11) belonged to either alcohol related (n = 9) or hepatitis B related (n = 2) chronic liver disease.

Most of the patients (Table 5) were having icterus (n = 62), of which 10 were positive for SBP; 30 patients had pedal edema, of which 2 were positive for SBP; 19 had fever, of which 4 were having SBP; 15 had abdominal tenderness, of which 5 were positive for SBP; 10 patients had flapping tremors, of which 2 were positive for SBP.

Coming to the mode of presentation, most of the patients presented with increased abdominal distension (n = 72), of which 6 were positive for SBP, followed by abdominal pain in 34 patients, of which 5 were positive for SBP. Nineteen patients presented with fever, of which 4 were positive for SBP; altered sensorium was evident in 18 patients, of which 5 were positive for SBP. Sixteen presented with gastrointestinal (GI) bleeding, of which 2 were positive for SBP; vomiting was evident in 10 patients, of which only 1 patient was positive for SBP and reduced urine output presented in 6 patients, of which 2 were positive for SBP (Table 6).

Icterus (p value 0.036), altered sensorium (p value 0.012) and abdominal tenderness (p value 0.003) were found to be significantly associated with SBP +ve group as compared to those in SBP -ve group.

Abdominal pain (p = 0.39), increased abdominal distension (p = 0.17), vomiting (p = 0.91), fever (p = 0.13), reduced urine output (p = 0.074), GI bleeding (p = 0.81), pedal edema (p = 0.365) and flapping tremors (p = 0.338) were not found to be of significant value.

Seventeen of the total 100 patients belonged to Child-Pugh Grade A (Table 7), out of which none of the patients had SBP. Thirty-four patients belonged to Child-Pugh Grade B, of which 2 patients were positive for SBP making 5.9% of the patients in Grade B. Most of the patients, i.e., 49, belonged to Grade C, of which 9 patients

Table 4. Etiology in Correlation with SBP

Etiology of CLD	Total No. of cases	Positive cases (SBP)	% positivity
Alcoholic liver disease	79	9	11.4
HBsAg positive	14	2	14.2
HCV related	2	0	0.0
NASH	4	0	0.0
NCPF	1	0	0.0
Total	**100**	**11**	**11**

CLD = Chronic liver disease; HBsAg = Hepatitis B surface antigen; HCV = Hepatitis C virus; NASH = Nonalcoholic steatohepatitis; NCPF = Noncirrhotic portal fibrosis.

Table 5. Clinical Signs in Correlation with SBP

Signs	No. of cases (n = 100)	Positive for SBP (n = 11)	Percentage (%)
Icterus	62	10	16.1
Fever	19	4	21.1
Abdominal tenderness	15	5	33.3
Flapping tremors	10	2	20.0
Pedal edema	30	2	6.7

Table 6. Mode of Presentation in Correlation with SBP

Mode of presentation	Total No. of cases (n = 100)	Positive for SBP (n = 11)	Percentage (%)
Abdominal pain	34	5	14.7
Increased abdominal distension	72	6	8.3
Fever	19	4	21.1
Vomiting	10	1	10.0
Altered sensorium	18	5	27.8
GI bleeding	16	2	12.5
Reduced urine output	6	2	33.3

had SBP, making 18.4% of the patients in Grade C. So, a higher Child-Pugh grade was found to be associated with higher percentage of patients having SBP.

Mean value of MELD (Model for End-stage Liver Disease) score (Table 8) in patients who were SBP +ve was 24.4 with a SD of 6.99 as compared to 16.6 in SBP -ve group with SD of 7.16 with a p value of 0.0009, suggesting that increasing MELD score is associated with a higher risk of developing SBP.

Comparing mean laboratory investigations (Table 9) between SBP +ve and SBP -ve groups, mean total bilirubin (p value 0.002) and INR (p < 0.001) were only significantly associated with SBP +ve group.

From among 100 patients selected, total 4 patients expired. Of SBP -ve group (n = 89), 2 patients expired while in SBP +ve group also, 2 patients expired (Table 10). P value came out to be 0.010 signifying

Table 7. Child-Pugh Classification and SBP

Child-Pugh grade	Total cases	SBP +ve	SBP -ve	% positivity
A	17	0	17	0.0
B	34	2	32	5.9
C	49	9	40	18.4
Total	**100**	**11**	**89**	**11.0**

Table 8. MELD Score in Correlation with SBP

	No. of patients	Mean MELD	SD
SBP -ve	89	16.6	7.16
SBP +ve	11	24.4	6.99

Unpaired *t*-test, p value 0.0009.

Table 9. Comparison of Mean Investigations for SBP +ve and -ve Groups

Investigations	SBP +ve		SBP -ve		P value
	Mean	SD	Mean	SD	
Hemoglobin (g/dL)	8.4	2.2	8.3	2.5	0.9
WBC count (/mm^3)	7215.4	3723.8	5763.6	2136.0	0.2
Total bilirubin	**3.1**	**3.8**	**7.4**	**6.8**	**0.002**
SGOT (IU/L)	73.7	63.4	95.6	53.0	0.274
SGPT (IU/L)	53.5	37.3	79.4	76.6	0.06
SAP (IU/L)	83.5	69.7	81.6	55.2	93
INR	**1.5**	**0.4**	**2.0**	**0.4**	**<0.001**
Blood urea (mg %)	46.6	39.2	53.8	26.3	0.55
S. creatinine (mg %)	2.3	6.2	1.6	0.6	0.69
S. protein (T) (g/dL)	3.8	0.6	3.9	0.5	0.79
S. albumin (g/dL)	2.3	0.7	1.9	0.7	0.11
Ascitic fluid total protein (g/dL)	1.9	0.4	1.9	0.4	0.68
Ascitic fluid albumin (g/dL)	0.5	0.1	0.4	0.2	0.55

Unpaired *t*-test

Table 10. Correlation of SBP with Mortality

	SBP +ve	SBP -ve
Survived	9	87
Expired	2	2
Total	**11**	**89**

Table 11. Laboratory Features as Mortality Predictors in SBP

Lab investigations	SBP +ve expired (2)		SBP +ve survived (9)		P value
	Mean	± SD	Mean	± SD	
Hemoglobin (g/dL)	9.1	2.3	8.2	2.6	0.68
WBC count (/mm^3)	4350.0	919.2	6077.8	2233.1	0.14
Total bilirubin (mg %)	6.4	1.7	7.7	7.6	0.65
SGOT (IU/L)	97.5	2.1	95.2	59.2	0.91
SGPT (IU/L)	59.0	24.0	83.9	84.4	0.46
SAP (IU/L)	105.5	6.4	76.3	60.3	0.19
INR	2.0	0.2	2.0	0.5	0.97
Blood urea (mg %)	55.5	17.7	53.4	28.7	0.87
S. creatinine (mg %)	2.3	0.6	1.4	0.5	0.0006
Ascitic fluid total protein (g/dL)	2.1	0.6	1.9	0.3	0.71

Unpaired *t*-test

Table 12. MELD Score in Correlation with Mortality in SBP

SBP +ve and outcome	N	Mean MELD score	SD
Expired	2	30.50	2.121
Survived	9	23.11	7.008

Unpaired *t*-test, p = 0.032.

that SBP +ve group was associated with an increased mortality as compared to SBP -ve group.

On comparing biochemical findings (Table 11), mortality in SBP was strongly associated with higher serum creatinine level in patients who were SBP +ve (p value 0.0006).

The other biochemical parameters, i.e., hemoglobin, white blood cell (WBC) count, serum glutamic oxaloacetic transaminase (SGOT), serum glutamic pyruvic transaminase (SGPT), serum alkaline phosphatase (SAP), serum bilirubin, international normalized ratio (INR), blood urea, and ascitic fluid total protein, were not found to be significantly different between survived and expired group.

Higher MELD score was found to be significantly associated with mortality group (n = 2), as compared to survived group (n = 9).

Thus, it can be said that higher MELD score is associated with increased mortality in SBP +ve patients as shown in Table 12.

DISCUSSION

The present study was conducted in 100 patients of chronic liver disease; 22 had ascitic fluid PMN count >250/mm^3 and 11 patients had ascitic fluid culture for single organism. So, prevalence of classical SBP was 11% and overall prevalence was 22%. Other studies have reported variable prevalence rate (24-30%)[16-21]. In studies done by Piroth et al[16] and Muhammad et al[17], prevalence of SBP was found to be 30%. In an Indian study by Agarwal et al[18], the prevalence of SBP was 34.14%, while in the study by Gill et al[19], the prevalence was 24%. In a study by Obstein et al[20], prevalence was 26%. In a study by Andreu et al[21], prevalence of SBP was found to be 25.45%.

In the present study, most of the patients of chronic liver disease had alcoholic liver disease (79%) followed by viral hepatitis B related (14%), HCV related (2%), nonalcoholic steatohepatitis (4%), noncirrhotic portal fibrosis (1%). In the study done by Campillo et al[22], out of 200 patients, 175 had alcoholic liver disease, 16 were hepatitis C related, 6 hepatitis B related, 1 case was of

hemochromatosis and 1 was cryptogenic cirrhosis. In an Indian study by Mohan and Venkataraman[23], alcoholism was seen in 55.5% and the cause was hepatitis B related in 21.8%.

SBP should be suspected when a patient with cirrhosis deteriorates, particularly with encephalopathy and/or jaundice. Patients with variceal bleeding or previous SBP are at particular risk. Clinical signs and symptoms such as fever and abdominal pain and systemic leukocytosis may be noted. In our study, icterus (p value 0.036), altered sensorium (p value 0.012) and abdominal tenderness (p value 0.003) were found to be significantly associated with SBP +ve group as compared to those in SBP -ve group.

Fever (p = 0.13), abdominal pain (p = 0.39), increased abdominal distension (p = 0.17), vomiting (p = 0.91), reduced urine output (p = 0.074), GI bleeding (p = 0.81), pedal edema (p = 0.365) and flapping tremors (p = 0.338) were not found to be of significant value.

Peripheral leukocytosis was found to be a strong indicator for presence of ascitic fluid infection. But these are also nonspecific markers for the presence of ascitic fluid infection and have not been of any statistical significance (p value 0.2) according to this study. Raised bilirubin levels (p value 0.002), and increased INR (p < 0.001) were only seen in most patients of SBP. Ascitic fluid protein levels did not significantly differ between SBP and non-SBP groups of patients and low ascitic protein was not associated with higher incidence of SBP in our study.

Advanced liver disease/Child-Pugh Class C had the highest incidence of spontaneous ascitic fluid infection among patients with cirrhosis. In the present study, 9 patients of 11 patients found to have SBP were present in Child-Pugh score Grade C while only 2 belong to Grade B, suggesting that 18.4% of Grade C patients had SBP while only 5.9% of Grade B had SBP. No patients of Grade A had SBP. In the study done by Campillo et al[22], Child-Pugh score was associated with higher incidence of SBP and mortality in SBP (0.0011). In the study by Puri et al[24], 95% of the patients who developed SBP belonged to Child-Pugh Grade C. In the study done by Agarwal et al[18], 12 of the 14 patients, i.e., 85.71% with SBP belonged to Class C and 2 (14.28%) patients were in Class B Child-Pugh score.

In the present study, mean MELD score of patients with SBP +ve was 24.4 ± 6.99 as compared to 16.6 ± 7.16 for those with SBP -ve, with p value of 0.0009, suggesting that increasing MELD score is associated with a higher risk of developing SBP. In the study by Obstein et al[20], mean MELD score for SBP +ve was 24 and without SBP was 18 (p = 0.0003). They concluded that MELD score is independently associated with a greater risk of SBP. In the study by Gill et al[19], mean MELD score for SBP +ve group was 19 ± 2.42 and 15 ± 3.93 for SBP -ve group.

Overall mortality in present study was 4% with SBP +ve group associated with an increased mortality as compared to SBP -ve group (p value 0.01). In the present study, we found serum creatinine as strong predictor for mortality in patients of SBP with chronic liver disease. In a study by Llovet et al[25], mortality was 17%. Factors associated with increased mortality were presence of upper GI bleeding at admission and PMN count in ascitic fluid. In another study by Rawat and Bhatnagar[26], mortality was 39%. Factors related were jaundice, hepatic encephalopathy, total leukocyte count, total bilirubin, hyponatremia, serum albumin, INR, blood urea and serum creatinine. Musskopf et al[27], in 2012, reported mortality of 40%. Factors associated with increased mortality were total bilirubin, serum creatinine and MELD score.

CONCLUSION

A large number of patients suffering from chronic liver disease with ascites were found to be suffering from SBP or its variant (22% in present study). This complication predisposes an individual to additional mortality risk.

Clinical features such as icterus, abdominal tenderness and altered sensorium were found to be associated with a higher risk of SBP. But none of the other clinical features was consistently associated with SBP in all the patients. None of the clinical features in expired group was found to be significantly associated with mortality in SBP. Higher serum creatinine was found to be associated significantly with expired group.

Child-Pugh grade and MELD score were associated with increased risk of SBP in chronic liver disease patients in the present study.

MELD score was found to be significantly associated with mortality in SBP.

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