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Original Article

Spontaneous bacterial peritonitis (SBP) in cirrhotic ascites: A prospective study in a tertiary care hospital, Nepal

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Abstract

Background: Spontaneous bacterial peritonitis (SBP) is one of the potentially lethal complications of cirrhosis and is defined as infected ascites in the absence of any recognizable secondary cause of infection. **Objective** was to study the occurrence of SBP, clinical and laboratory characteristics and the response to antibiotics. **Methods:** We had prospectively evaluated 81 cirrhotic patients with ascites during one-year period**.** All SBP patients were treated with cefotaxime, 2gm IV, every 12h for 5days.

Results: Of these 81 patients, 24.67% of patients (n=20) had SBP and its variants (classical SBP n= 4, CNNA n=13 and bacterascites n=3). There were thirteen males and 7 females in the study.85% of the cases had Child`s class C cirrhosis. UGI bleeding and abdominal pain were the most common presenting symptoms of SBP. Culture positives were 35% (n=7). The most frequent organisms were *Escherichia coli* (n=3) and S*treptococcus pneumoniae (*n=2). 94% of the patients responded to therapy after 48 hours of treatment. Total resolution after 5 days of therapy was 73% and in-hospital mortality was 15% (n=3).

Conclusion: SBP, if diagnosed early can be treated with very good success rate up to 73%. Appropriate treatment of SBP with cefotaxime can help in reducing mortality and morbidity in patients with chronic liver disease.

Key words: SBP, CNNA, Cirrhotic patients and Cefotaxime

Spontaneous bacterial peritonitis (SBP) is a potentially lethal complication of cirrhosis. It is probably the most characteristic infectious complication of cirrhosis. SBP is defined as the infected ascetic fluid in absence of any recognizable secondary cause of peritonitis. The occurrences of SBP are independent of the aetiology of liver diseases. It develops in 10-30% of hospitalized patients [1] and the mortality exceeded 90%, when it was first described. However, with the early recognition of disease and prompt and appropriate antibiotic treatment, the in-hospital mortality of an episode of SBP has been reduced to approximately 20% [2].

Spontaneous bacterial peritonitis is diagnosed when (a) the ascitic fluid culture grows pathogenic bacteria (almost always pure growth of a single type of organism), (b) the ascitic fluid neutrophils count ≥ 250 cells /mm^3 and (c) there is no evidence of surgically treatable intra abdominal sources of infection. Variants of SBP are – (i) Classic SBP: - ascitic fluid polymorphonuclear leukocyte (PMN) counts >250/mm^3 and positive culture. (ii) Culture negative neutrocytic ascitis (CNNA): - ascitic fluid PMN counts >250/mm^3 and culture negative (iii) Bacterascites: - a culture positive ascitic fluid in the presence of PMN counts <250/mm^3.

The infection of ascitic fluid leads to an elevation of polymorphonuclear leukocyte (PMN) count in ascitic fluid, which represents evidence of failure of the first line of defense, i.e. the peritoneal macrophages, to kill invading bacteria. Thus, elevation of PMN counts to more than 250/mm^3 in ascitic fluid has been adopted as a diagnostic criterion of SBP, without consideration of the detection of bacteria in ascitic fluid cultures. The infection of ascitic fluid in SBP is considered to be blood-borne and monomicrobial in 90% of patients [2.] Its occurrence is related to low protein levels and impaired opsonic activity in ascitic fluid. Most episodes of spontaneous bacterial peritonitis are monomicrobial and produced by enteric bacteria. Of such episodes, 67% involve gram- negative bacteria, *Escherichia coli* being the most frequently isolated organism.[3]

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Since clinical manifestations of spontaneous bacterial peritonitis are non-specific, the diagnosis of this complication is based on the analysis of ascitic fluid. Paracentesis is a safe and simple method for diagnosing spontaneous bacterial peritonitis without significant complications. The bacterial and PMN counts in ascites increase rapidly after infection unless antibiotic treatment is started. At the start of therapy, broad-spectrum antibiotics are warranted because bacteria range from gram-negative intestinal flora to *pnuemococcus.* However, it is not clear how many days of treatment are necessary to resolve the ascitic fluid infection. A recent consensus document about SBP has recommended a minimum duration of 5 days of antibiotic therapy with third generation cephalosporin [1]

Cefotaxime (CTX) third generation caphalosporin, has become one of the most widely used initial empiric therapies for SBP in cirrhosis. It has been shown to be safe, effective, and well tolerated in a wide range of doses and duration regimens. The antibacterial treatment should be continued for approximately 5 days, on the basis of a report that the PMN count in ascitic fluid decreases below 250/mm^3 in mean period of 5 days, the antibiotic treatment can then be safely discontinued [19]. It has been shown that 5 days treatment with cefotaxime is as effective as 10 days treatment.

High degree of suspicion, routine diagnostic paracentesis, standardization of diagnostic criteria of ascitic fluid infection and use of non-nephrotoxic antibiotics is essential for early diagnosis and management. Early diagnosed and treated SBP episodes resolved satisfactorily and improved short-term prognosis. The long-term prognosis continues to be extremely poor.

The profile of SBP may vary with different ethnic, geographic, social and etiological factors. Although there are many reports of SBP, from both developed and developing countries, there has not been any study conducted in Nepal to find out the occurrence of SBP We have conducted a prospective study to find out the occurrence of SBP in cirrhotic ascites and try to associate clinical, biochemical microbiological association and response to antibiotic therapy. The study has been conducted at B.P. Koirala Institute of Health Sciences (BPKIHS), a tertiary care hospital, in eastern part of Nepal.

Material and methods

This study was a hospital based descriptive study of one-year duration from March 2003 to February 2004.

The aims and objectives were to study the occurrence of SBP, its clinical and laboratory characteristics and response to antibiotics. This study had included 81 consecutive cases of cirrhosis of liver with ascites. The case of clinical cirrhosis of liver will be defined as a patient having at least one clinical sign of hepatocellular failure and one of portal hypertension along with at least three USG findings suggestive of cirrhosis of liver

Clinical signs of Hepatocellular Failure

1. Jaundice.
2. Hepatic encephalopathy.
3. Skin changes: spider angiomas, palmer erythema
4. Endocrine changes: breast atrophy, gynaecomastia, testicular atrophy.

Clinical Signs of Portal Hypertension

1. Gastroesopageal varices: haemetemesis, melaena or By upper gastrointestinal endoscopy
2. Splenomegaly

Findings of USG abdomen suggestive of cirrhosis of Liver

1. Coarse echotexture
2. Nodular Surface
3. Decreased caudate to right lobe (C/RL) ratio
4. Portal Hypertension: Ascites, splenomegaly, varices, dilatation of the portal vein (>13mm), dilatation of the splenic vein (>11mm).

Inclusion Criteria:

A case of clinical cirrhosis of liver with ascites

Exclusion Criteria:

I. Ascites due to renal, cardiac, tubercular, malignant pathology
II. Secondary peritonitis.

We studied all adult subjects with cirrhosis, admitted in the hospital. Cirrhosis was diagnosed clinically, by laboratory tests, sonological and/or histological methods. No selection was made on the basic of socio-demographic characteristics, education and clinical status. Patients were included irrespective of their viral status. Patients readmitted during the study period were included at first admission only. A case with ascites was labelled as having SBP and enrolled in the study if the ascitic fluid analysis had shown one or both the following:

- Total polymorphonuclear (PMN) count: >250 cells/mm3
- Ascitic fluid culture positive

All eligible cases were selected from the admitted patients in the medical wards of BPKIHS. Informed consent was taken from the patients or close relatives where relevant. With all aseptic precautions by inserting a needle of 22 or 18 gauge in the left iliac fossa or mid line just below the umbilicus, abdominal paracentesis was done and samples sent to the laboratory. Ascitic fluid (AF) was collected in an Ethylene diamine tetracetic acid (EDTA) tube for TLC, DLC, in a plain vial for protein and sugar and 10ml inoculated in a blood culture bottle at the bedside. The AF white blood cell count and PMN counts were determined by the total and differential counts on the basis of morphologic appearance in a manual counting chamber.

All the patients had simultaneous samples of AF and serum taken. Paracentesis was performed as early as possible after hospitalization, before starting antibiotics.

Liver function tests (S. Albumin, Total protein, Total Bilirubin, Conjugated Bilirubin, Prothrombin time), and USG abdomen was done in all patients. UGI endoscopy was done and gastroesopageal varices were graded (I-IV) accordingly. Antibiotic (Inj. Cefotaxime 2gm., 12 hourly, intravenously for five days) was started on those patients who had ascitic fluid PMN count of more than 250/mm^3. Patients were followed up daily and progress notes were recorded. A repeat ascitic fluid analysis was performed after 48 hrs of antibiotic initiation. Patients who did not show >25% decline in PMN count or exhibit clinical deterioration (e.g. worsening encephalopathy, renal failure, shock) were reassessed. These patients were considered as a case of treatment failure. Antibiotic therapy in these patients was modified empirically or on the basis of result of culture sensitivity (if available).

Each patient was classified according to Child Turcotte-Pugh (CTP) class[4], which is a scoring system to grade severity of chronic liver disease. The CTP score is calculated by assigning 1 point for any features in column 1, 2 points for any features in column 2 and 3 points for any features in column 3, class A ≤ 6, class B 7 – 9; class C ≥ 10.

Ethical clearance from the Institute was taken for this study.

Statistical analysis

All the case records were collated in Microsoft excel. The statistical analysis was done with the help of SPSS 10.0 for Windows. Results are expressed as mean ± SD. Comparison was made by using student`s t- test. Correlations were evaluated by Pearson's r test and the level of significance was set at <0.05.

Table 1: Child Turcotte-Pugh (CTP) classification of cirrhosis[4]

Factors	**1**	**2**	**3**
Encephalopathy	None	Stage I – II	Stage III – IV
Ascites	None	Easily controlled	Poorly controlled
Bilirubin (mg/dL)	<2	2– 3	>3
Albumin (gm/dL)	>3.5	3 -3.5	<3
Prothrombin time (Second Prolonged)	0 - 4	4 - 6	>6

Results

One hundred sixty-one patients were admitted with the diagnosis of chronic liver disease (Fig 1). Out of these, 81 patients who fulfilled the criteria for having cirrhotic ascites underwent successful diagnostic ascitic tapping, of which only 20 were diagnosed to have SBP or its variants.

Among the study population were 48 males and 33 females with mean age of 51.1±11.7 yrs as 51.34 ±13.01 and 49.3 ±14.06 amongst males and the females respectively. The age of the study population ranged between 23 yrs to 79 years.

With respect to the aetiology of chronic liver disease, most of them were having alcohol related cirrhosis whilst two of them were virus related. Twelve patients had past history of ascitic fluid paracentesis, but none of them had documented SBP and/or of receiving SBP prophylaxis.

The means of haemoglobin, TLC and platelet count were 9.46 ± 2.36gm/dl, 12306.24 ± 7271.9/mm^3, and 127839.5 ± 57823/mm^3 respectively (table 2). The mean of PT at the time of presentation was 19.72 ± 7.09 sec.

The mean total Bilirubin, total Protein, albumin, ALT, AST and Alkaline phosphatase were 7.04 ± 6.52 mg/dl, 6.9 ± 1.2 gm/dl, 2.28 ± .56 gm/dl, 55.44 ± 42.14U/L, 132.29 ± 147.9 U/L and 298.38 ± 144.88 U/L respectively. The means of urea and creatinine were 50.50 ± 43.53 mg/dl and 1.22 ± 0.97mg/dl respectively.

Ascitic fluid analysis at admission showed mean TLC, Polymorphonuclear (PMN), Protein and sugars as 903.34 ± 3342/mm^3, 411.62 ± 1109/mm^3, 1.18 ± 0.746gm/dl and 112 ± 38.19mg/dl respectively.

Ascitic fluid tapping was done in all cases and in only 3 cases procedure related complications occurred i.e. leakage from needle-prick site. The characteristics of non SBP patients (n = 61) is described in table 3.

Characteristics of SBP patients:

Of the 81 patients studied, 20 were diagnosed as having SBP or its variants of whom thirteen patients had CNNA. Classical SBP and Bacterascites were observed in 4 and 3 patients respectively. There were 13 males and 7 females. The mean age of these patients was 48.45 ± 12.68 years, ranging from 23 to 70 years. Six patients (30% cases) were less than 40 years of age.

Eighty five percent (n=17) cases were in Child`s class C and 15% (n=3) were in Child`s class B. All 7 female cases were in class C whereas 10 male cases out of 13 were in class C. The most common presenting symptoms were upper gastrointestinal bleeding and pain abdomen found in 75% and 65% cases respectively. Fever was present in 45% of cases. Asymptomatic patients, those without pain abdomen and fever comprised 30% of the cases (Fig 2). Clinical jaundice was found in 95% of cases. There was no significant difference in presenting complaints of Child Pugh class B and Child Pugh class C.

The mean of haemoglobin was 9.62 ± 2.56gm/dl. Mean of TLC and platelets was 12580±6864.4/mm^3 and 111950.00 ± 42992.0/mm^3. Mean of total bilirubin, albumin ALT, AST, ALK and prothromin time was 8.62 ± 6.9 mg/dl, 2.22 ± 0.41 gm/dl, 45.30 ± 28.46 U/L, 102.95 ± 78.8 U/L, 287.65± 136.99 U/L and 18.80 ± 2.67 sec respectively (table 4). The mean of S. urea and creatinine was 65.0 ± 60.4 mg/dl and 1.57 ± 1.36 mg/dl respectively. Serum Creatinine was found to be >1.4 in 8 cases.

On analysis of ascitic fluid's characteristics the mean TLC and neutrophils was found to be 3363.1 ± 6209.6 /mm^3 and 1510.2± 1866.7/mm^3. The mean protein and sugar was 1.106 ± 0.729 and 97±38 mg/dl. Liver function test, kidney function test and ascitic fluid characteristics at the beginning of episode did not differ according to Child Pugh class or presence or absence of symptoms.

Out of 20 cases of SBP, organisms were isolated from 7 cases (35%). Six cases were monobacterial and in 1 case 2 types of organisms were grown. The most common isolates were *Escherichia coli*, (n-=3), *Streptococcus pneumoniae* (n=2), *Klebsiella pneumoniae* (n=1), *Coagulase negative Staphylococcus* (n=1) and *Acinitobacter spp.* (n=1).We believe that the latter two isolates were not true infections, because they were not associated with neutrocytic ascites and their repeat cultures were sterile. *Escherichia coli* and *Klebseila pneumoniae* were isolated together in one case (table 5).

There was no difference between culture positive SBP and culture negative SBP in respect of their age, sex, child class, Liver function test and kidney function test**.** There was only one culture positive case out of 3 cases of Child Pugh class B, whereas 6 cases grew organisms of the 17 cases of Child Pugh class C.

Out of 20 cases, 17 were neutrocytic (≥ 250 PMN) ascites and 3 were non-neutrocytic (≤ 250 PMN) ascites (Fig 3). Of three non-neutrocytic ascites one patient developed neutrocytic ascites and was treated as neutrocytic ascites and repeat culture grown showed no organisms, so this patient was also treated as CNNA. Two cases of bacterascites were followed up with repeat culture in OPD which were culture were sterile, so treatment was not given to those patients.

Thus, a total of 18 cases were treated with injection Cefotaxime 2 gm IV, 12 hourly for five days. Repeat diagnostic tapping was done after 48 hours of antibiotic therapy. Out of 18 patients, 13 showed marked reduction in their TLC count and there was symptomatic improvement also. They responded to treatment and discharged after further 5 days of treatment with Cefotaxime.

Three patients died during the period of the study (Table 6). Two were males and one was female. Patients died on the 2^{nd}, 3^{rd} and 14^{th} day of admission. Two patients left against medical advice and hence were lost to follow up.

We looked for the association between clinical, biochemical and microbiological parameters. We found significant difference in SBP and non SBP group in fever (p=0.049), pain abdomen (0.001), UGI Bleeding (0.008) and ascitic fluid sugar. In other parameter no significant difference found. (Table 7)

Table 2: Characteristics of Total patients (n=81)

Parameter	Mean
No. of patients	81
Age	51.11 ± 11.80
Sex F/M	33 / 48
Jaundice	74
Fever	23
Hepatic Encephalopathy	46
Pain abdomen	27
Upper Gastrointestinal Bleeding	40
Haemoglobin (gm/dl)	9.46 ± 2.36
TLC	12306 ± 7271.9
Platelet count	127839.5 ± 57823
Prothrombin Time (Sec)	19.72 ± 7.09
T.Bilirubin (mg/dl)	7.04 ± 6.52
Conj. Bilirubin (mg/dl)	4.63± 4.80
T. protein (gm/dl)	6.9 ± 1.2
S. Albumin (gm/dl)	2.28 ± 0.56
AST (U/L)	132.92 ± 147.9
ALT (U/L)	55.44 ± 42.14
Alkaline Phosphate (U/L)	298.38 ± 144.88
Urea (mg/dl)	50.50 ± 43.53
Creatinine (mg/dl)	1.22 ± 0.97
Ascitic Fluid	
TLC	903.34 ± 3342
Neutrophils	411.62 ± 1109
Protein (gm/dl)	1.18 ± .746
Sugar (mg/dl)	112 ± 88.19
Child`s class B/C	24 / 57

Table3: Characteristics of Non-SBP patients (n=61)

Parameter	Mean
No. of patients	61
Age	51.98 ± 11.4
Sex F/M	27 / 34
Jaundice	55
Fever	14
Hepatic Encephalopathy	35
Pain abdomen	14
Upper Gastrointestinal Bleeding	25
Haemoglobin (gm/dl)	9.4 ± 2.3
TLC	12216 ± 7453
Platelet count	133049 ± 61327
Prothrombin Time	20.03 ± 8.02
T.Bilirubin (mg/dl)	6.53 ± 6.36
Conj. Bilirubin (mg/dl)	4.21 ± 4.33
T. protein (gm/dl)	6.21 ± 1.17
S. Albumin (gm/dl)	2.31 ± 0.6
AST (U/L)	141.92 ± 163.83
ALT (U/L)	58.77 ± 45.45
Alkaline Phosphate (U/L)	297.92 ± 148.39
Urea (mg/dl)	45.75 ± 35.75
Creatinine (mg/dl)	1.11 ± 0.79
Ascitic Fluid	
TLC	96.87 ± 91.23
Neutrophils	51.43 ± 59.65
Protein (gm/dl)	1.2 ± .75
Sugar (mg/dl)	117.5 ± 37.26
Child`s class	21 / 40

Table 4: Characteristics of SBP patients

Parameter	**Mean**
No. of patients	20
Age	48.45 ± 12.6
Sex M/ F	13 / 7
Jaundice	19
Fever	9
Hepatic Encephalopathy	11
Pain abdomen	13
Upper Gastrointestinal Bleeding	15
Hb% (gm/dl)	9.6 ± 2.5
TLC	12580 ± 6564
Platelet count	111950 ± 42992
PT	18.8 ± 2.6
T. Bilirubin (mg/dl)	8.62 ± 6.9
Conj.Bilirubin (mg/dl)	5.93 ± 3.96
T.protein (gm/dl)	6.18 ± 1.32
S Albumin (gm/dl)	2.22 ± 0.41
AST (U/L)	102 ± 78.8
ALT (U/L)	45.3 ± 28.4
Alkaline Phosphate (U/L)	287.6 ± 136.9
Urea (mg/dl)	65 ± 60.4
Creatinine (mg/dl)	1.57 ± 1.36
Ascitic fluid	
TLC	3363.1 ± 6209
Neutrophils	1510.2 ± 1866.7
Protein (gm/dl)	1.1 ± .72
Sugar (mg/dl)	97.65 ± 38
Child`s class	3 / 17
Response	72.2%
Culture positive	7

Table 5: Organisms in Culture Positive Cases

Bacteria	**No. of cases**	**Percentage**
Escherichia coli	3	42.8%
Streptococcus pneumoniae	2	28.57%
Klebsiella Pneumoniae	1	14.28%
Acinetobacter species	1	14.28%
Coagulase- negative Staphylococcus	1	14.28%

Table 6: SBP variants and their outcome

SBP	**Prevalence**	**Outcome**	
		Response	**No Response**
CSBP	4 / 81 (4.94 %)	3 (75%)	1 expired (25%)
CNNA	13 / 81 (16.05%)	9 (69.2%)	2 expired (15.38%)
BA	3 / 81 (3.7%)	3 (100%)	Not Applicable

Table7: Clinical and Laboratory variables with significant values

Parameters	**Non-SBP**	**SBP**	**P. Value**
No of patients	61	20	
Age	51.98 ± 11.4	48.45 ± 12.6	.247
Sex M / F	27 / 34	13 / 7	
Jaundice	55	19	0.5
Fever	14	9	0.049
HE	35	11	0.58
Pain abd	14	13	0.001
UGI Bleed	25	15	0.008
INVESTIGATION			
Hb	9.4 ± 2.3	9.6 ± 2.5	0.724
TLC	12216 ± 7453	12580 ± 6564	0.84
Platelet	133049 ± 61327	111950 ± 42992	0.15
PT	20.03 ± 8.02	18.8 ± 2.6	0.50
T.Billirubin	6.53 ± 6.36	8.62 ± 6.9	0.21
T.protein	6.21 ± 1.17	6.18 ± 1.32	0.92
Albumin	2.31 ± 0.6	2.22 ± 0.41	0.53
UREA	45.75 ± 35.75	65 ± 60.4	0.86
CREATININE	1.11 ± 0.79	1.57 ± 1.36	0.06
Ascitic fluid			
TLC	96.87 ± 91.23	3363.1 ± 6209	0.000
Neutrophil	51.43 ± 59.65	1510.2 ± 1866.7	0.01
PROTEIN	1.2 ± .75	1.1 ± .72	0.61
SUGAR	117.5 ± 37.26	97.65 ± 38	0.049
CHILD CLASS	21 / 40	3 / 17	0.117
RESPONSE		72.2%	
MORTALITY		15%	

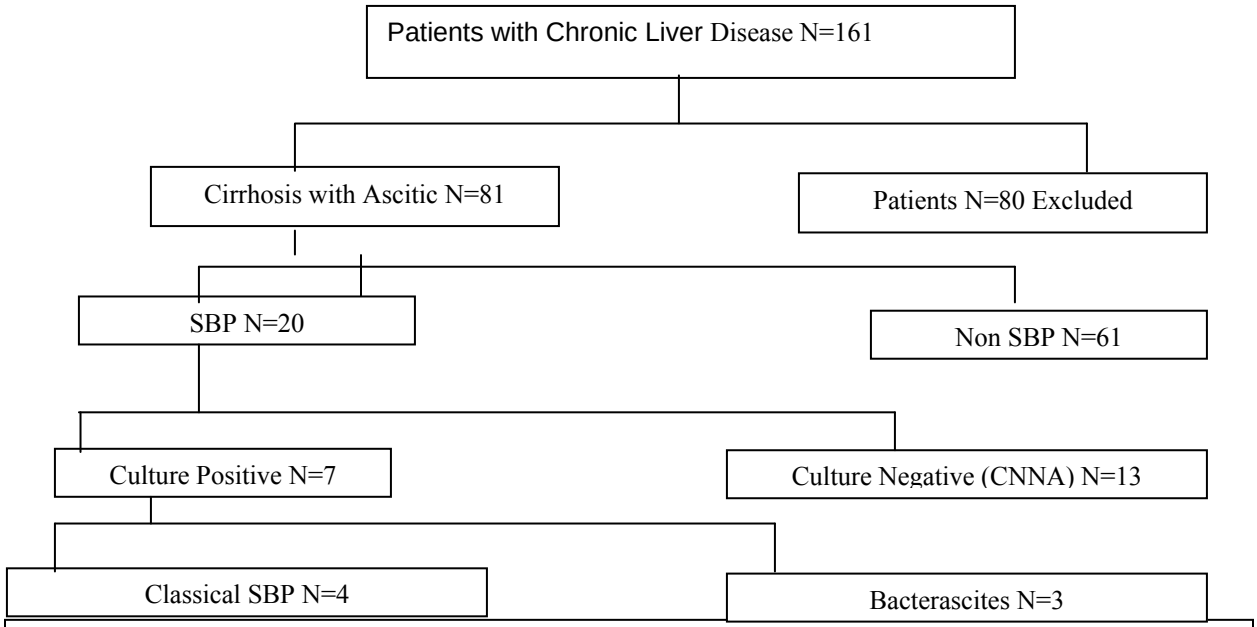

Fig 1: Study design and results. Consecutive inpatients with cirrhotic ascites that had undergone paracentesis were studied. SBP was diagnosed in 20 patients. 7 of the patient with SBP had culture positive and 13 had negative culture.

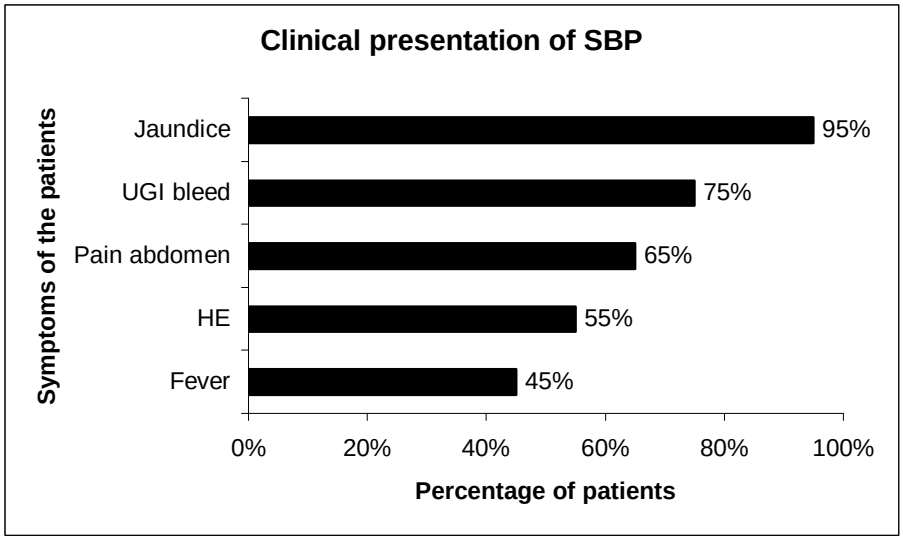

Fig: 2 Clinical presentation of SBP

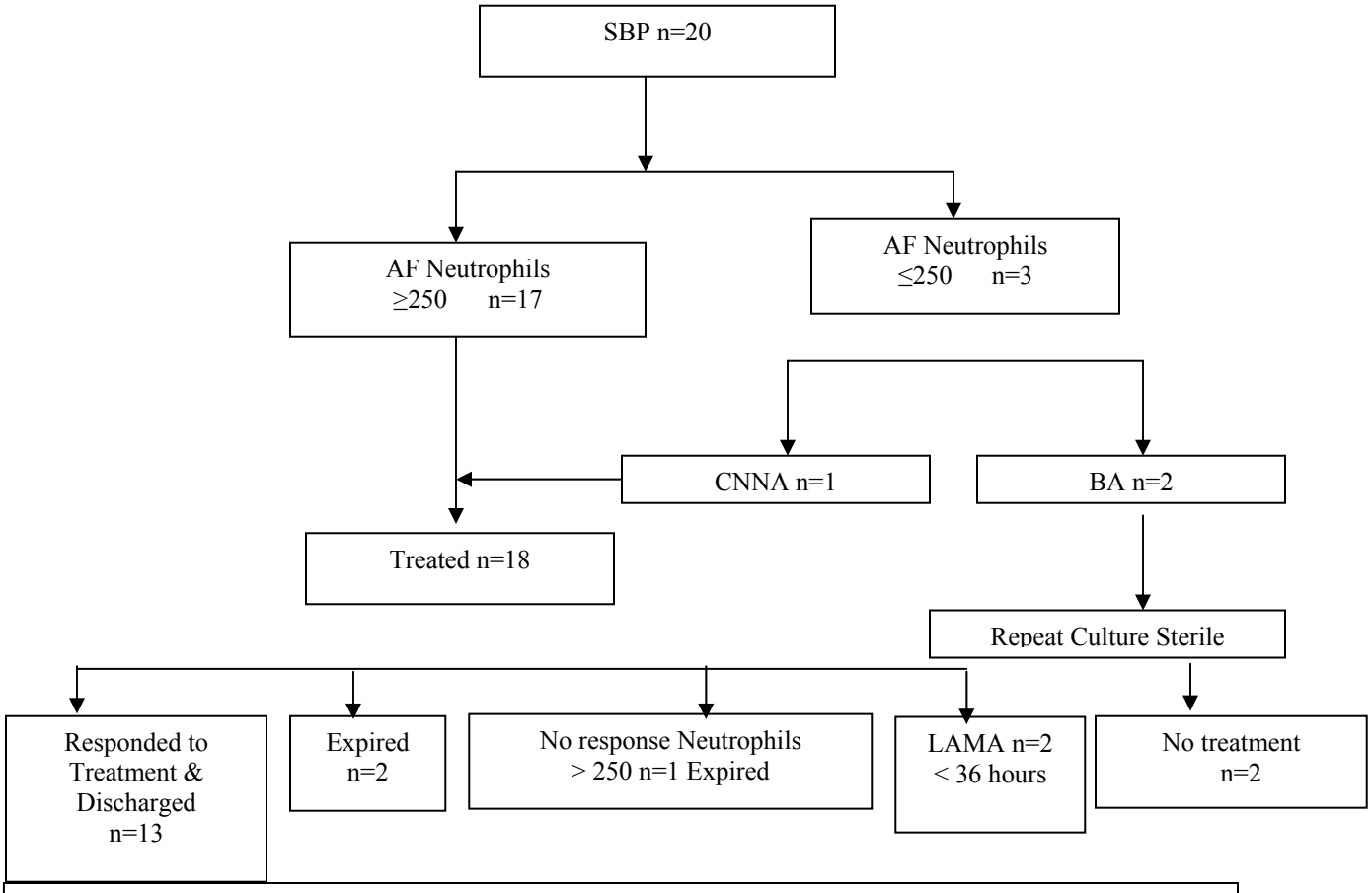

Fig 3: Summary of treated patients of SBP and their outcome
LAMA- Left against medical advice, AF- Ascitic fluid

Discussion

All cirrhotic patients with ascites can develop SBP. The prevalence of SBP in hospitalized patients ranges between 10%-30%[1].

The clinical manifestations of SBP have a broad range. Most patients of SBP have symptoms and/or signs clearly suggestive of peritoneal infection, especially pain abdomen, fever and altered gastrointestinal motility. Other patients may present with development of hepatic encephalopathy or renal failure which may be the predominant or only features.

Furthermore, SBP may be asymptomatic or have minor symptoms only. With the early diagnosis of the disease and prompt and appropriate antibiotic treatment, the in-patient mortality of an episode of SBP has been reduced to approximately 20%. [2]

In this study, the occurrence of SBP was 24.69% of the patients with cirrhotic ascites. All of them were diagnosed as SBP at the time of admission. In a study from India, Amarapurkar DN et al, reported similar prevalence of SBP as 22% in hospitalized patients.[5] The prevalence of SBP depends on severity of liver dysfunction, being higher in advanced liver disease. Jain et al, reported that the prevalence of SBP was 34.92% out of 63 patients. All patients who had SBP were in child class C.[6]

Puri AS et al, reported 21 out of 70 i.e.30% had SBP or its variants and 77% of their patients were in class C.[7] In our study, 85% of patients were Child Pugh class C .The reason for some lower incidence of SBP in comparison to Indian studies, is the strict criteria for diagnosing a case of clinical cirrhosis. Many patients with severe liver dysfunction had not been included in the study, because they expired within a very short period of hospital stay before the required investigations were performed.

Stogaard J S et al, in contrast to most other studies, diagnosed SBP only on the basis of ascitic fluid culture regardless of the number of WBCs. They found the incidence of SBP as 7.7%, which is much lower in comparison to our study. [8]

Liach et al , reported the occurrence of the first episode of SBP in cirrhotic patients with ascites followed for a long period of time was relatively low at 11% after one year and 15% after 3years of follow up.[9] The various reasons that could explain for this variation in comparison to our study are -that they have included patients with only moderately advance liver disease, as 80% of their patients were belonging to CTP class A and B in contrast to our study, in which 85% of patients were in CTP Class C. They have also included patients who were on oral non-absorbable antibiotics during UGI bleeding which on follow up may have reduced the risk of SBP in their patients.

Luke T Evan et al, reported the prevalence of SBP in the population of 427 cirrhotic outpatients was 3.5%. SBP in outpatients is less frequent; occurring in patients with less advanced liver disease and may have a better outcome than its counterpart in hospitalized patients with SBP.[10]

We found the mean age of patients as 48.5 years which is similar in the study by Denis R. et. al.[12] In our study most common presenting symptom were UGI bleeding (75%) followed by pain abdomen (65%). Fever was found in 45%. Great variation in symptoms and signs have been reported. Minhas et al, reported fever 54%, pain abdomen 57% and Hepatic encephalopathy 67%. [13] In other study, Pelletier et al , found 89% of patients were having fever, UGI bleed (42%), pain abdomen 53% and hepatic encephalopathy in 50% of cases.[16] Completely asymptomatic cases have been reported between 14% - 100%. [15,7]

In our study mean Hb% was found 9.6 gm/dl. It could be because 75% of our patients at presentation had UGI bleeding. Eighty five percent of cases were in child class C and or similar trend was found in other studies.[16, 18]

Most of the patients had features of liver dysfunction at admission the mean of total bilirubin, albumin and prothrombin time were 8.62 +/- 6.93 mg/dl, 2.22 +/- 0.41 gm/dl and 18.8 +/- 2.67 sec. Similar trend was noted in a study by Anastasia C T et al .[15]

In our study, out of 20 cases of SBP, organisms were isolated in 7 cases (35%). Most of them were gram negative, mainly *Escherichia coli* n=3 (42.59%), *Klebseilla Pnuemoniae* n=1 (14.28%) and *Acinetobacter Spp* (n=1 14.28%). Gram-positive organisms were *S. Pneumoniae* n=2 (28.4%) and *Coagulase. Negative Staphyloccus*. n=1 (14.28%).
E. Coli was found as most common organism in most of the other studies ranges approximately 60% of all positive culture.[14,19,21] The other common isolates were *Klebsiella Pneumoniae* and *Pnuemococcus.* Jain et al , found S*taphylococcus* as the most common organism[6].

Few of the studies have noted a predominance of gram-positive organisms in ascitic fluid cultures. David D et al ,in their study found that 53% of the organisms were Streptococcus.[20] Such an unusually high proportion might be due to the fact that they use a BACTEC plus aerobic and anaerobic bottles culture system.

We have found cultures positive only in 35% our cases. This low proportion of positive ascitic fluid is probably due to the relatively low concentration of bacteria in ascitic fluid.

The low rate of culture positivity can be attributed to prior antibiotics intake by the patients. Runyon BA et al , in spite of using sensitive methods of cultures, culture of ascitic fluid was negative in 40% of the cases with typical clinical feature of SBP and elevated ascitic PMN.[18]

Our culture positivity rate was not very different from that of Franca et al, who had obtained bacteriological diagnosis in only 47% of cases despite inoculation of ascitic fluid in blood culture bottles at the bedside. [19]

In our study, treatment was given to 18 patients with neutrocytic ascites. Injection cefotaxime 2gram iv bid for 5 days was used as per latest recommendation, [1] 93.3% of cases had responded to therapy, as confirmed by repeat ascitic tap after 48 hours. In spite of adequate treatment, two patients expired due to other complications of cirrhosis hence 72.22% of patients were discharged after complete resolution of SBP.

The treatment response to SBP with injection cefotaxime was 85% in a study by Navasa M et al.[21] Our study also showed a similar response rate.

Anastasia C T et al, showed that resolution of SBP was achieved in 90% of the patients with cefotaxime or quinolone.[15] In another study, Franca et al , found resolution rate on day 5 of treatment was 73%.[19] In our study the response rate at 48 hours was 93.3%.

Conclusion

In end stage liver disease due to cirrhosis with ascites, any triggering factor like SBP is sufficient to tilt the balance and herald the sequence of consequences leading to death. Patients with fever, UGI bleed and hepatic encephalopathy have increased risk for developing SBP. SBP can be diagnosed and treated easily and hence clinicians should have a high index of suspicion and low threshold for diagnosis. SBP if diagnosed early can be treated with very good success rate up to 73%. Gram-negative organism predominate most SBP infections in this part of the world. Culture positivity is low, so neutrophilia in ascitic fluid should be taken as sufficient criteria to treat the patient. Appropriate treatment of SBP with cefotaxime can help reduce morbidity and mortality in patients with chronic liver disease.

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