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Original Research Article

Prevalence of Spontaneous Bacterial Peritonitis in Cases of Ascites in a Tertiary Hospital

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Abstract

Objective: To study etiopathogenesis, prevalence, signs & symptoms, lab parameters of Spontaneous bacterial peritonitis (SBP) in cases of patients presenting with ascites of varied etiology.

Methods: The present study was conducted in 60 patients of ascites. Patients were selected randomly without any bias for age, sex or type of ascites. A detailed history was recorded with particular emphasis on symptoms of SBP like fever, pain in abdomen, sudden increase in abdominal distension, altered consciousness, hematemesis, malena, etc. Duration of ascites, alcoholism, past history of hematemesis, encephalopathy, jaundice was also noted. Bed side inoculation of ascitic fluid in blood culture bottles was done and growth noted.

Results: Fever (63.6%) and abdominal pain (45.45%) were common presenting symptoms Patients with severe liver disease expressed by presence of hepatic stigmata, hepatic encephalopathy, and abnormal liver function tests, had higher occurrence of SBP. Patients with ascitic fluid total protein less than 1 gm/dl had higher occurrence of SBP. Bed side inoculation of ascitic fluid in blood culture broth demonstrated organism in seven patients. In six patients E. Coli and in one patient Klebsiella was responsible for SBP.

Conclusion: Any patient of ascites, admitted with fever and abdominal pain. SBP should be considered. SBP is a common cause of hepatic encephalopathy. Thus all patients who are admitted with hepatic encephalopathy should be screened for SBP and treatment should be started as early as possible as their condition can be treated successfully with timely intervention.

Keywords: Monomicrobial Non-Neutrocytic Bacterasciten (MNBA), Culture Negative Neurocytic Anciton (CNNA) Spontaneous bacterial peritonitis (SBP).

Introduction

Ascites refers to collection of free fluid in peritoneal cavity. It is a common medical problem encountered in day-to-day practice. Ascites is usually a chronic disease. It is a distressing condition, having serious impact on patient's life. It reduces mobility of the patient, causes great discomfort, affects the productivity of the patient. Repeated hospitalization is required by the patient.[1]

Ascites is a disease of varied etiology. Cirrhosis, NCPH, Nephrotic syndrome,

CCF, are few common causes. Several complications may accompany ascites during the course of illness, like upper GI bleeding, hepatic encephalopathy, hepato-renal syndrome, pulmonary embolism, resulting in life threatening conditions. Spontaneous bacterial peritonitis is common and serious complication of ascites. SBP is defined as infection of pre-existing ascites without evidence of an intra-abdominal source for secondary infection. In hospital mortality rate of cirrhotic patients with SBP has been reported to range between 30-45%.[2-3]

Most episodes of SBP are caused by organism usually present in intestinal flora. The most accepted pathogenic mechanism of an in the passage of intestinal bacteria into general circulation and then into the ascitic fluid. 8.Coll and Klebsiella are the most common organism involved, other organism like proteus, *pseudomonas, anaerobes, atreptococcus, staphylococcus, listeria* are also isolated infrequently.[4]

Different workers have taken different criteria for the diagnosis of SBP. Most widely accepted criteria in a positive ascitic fluid culture and demonstration of more than 250 polymorpha/cumm of ascitic fluid. Two variants of the disease have been described. One in Bacterancitos (also called monomicrobial non-neutrocytic bacterasciten [MNBA] or asymptomatic bacterascites). It in defined an isolation of bacteria from ascitic fluid with neutrophils less than 250 cells per cubic mm of ascitic fluid. Second variant in Culture Negative Neurocytic Anciton (CNNA), which in diagnosed by demonstration of more than 500 polymorphs/cumn of ancitic fluid in the absence of a positive culture.[2-6]

Object of the study is to estimate prevalence of SBP in cases of ascites of varied etiology. Most of the cases studied were patients of cirrhosis of various etiology. A significant number of patients of nephrotic syndrome, CCF and NCPH were also included. An attempt is also made to correlate different clinical and laboratory data as predictive factors. Medicine is an ever advancing science. Each case heralds' new dimensions and is an incentive for new thoughts, experiment and derivations. The achievement we celebrate today is a step, an opening of opportunity, to greater horizons and achievements that await us. This study is also an effort to make some contribution to the existing cognizance on the subject.

Materials and Methods

The present study was conducted in 60 patients of ascites admitted in medical wards of the department of medicine. Patients studied in the study were having ascites of varied etiology on admission to the Hospital, selected randomly without any bias for age, sex or type of ascites. A detailed history was recorded with particular emphasis on symptoms of SBP like fever, pain in abdomen, sudden increase in abdominal distension, altered consciousness, haemetemesis, malena, etc. Duration of ascites, alcoholism, past history of hematemesis, encephalopathy, jaundice was also noted.

Exclusion Criteria:

Patients with history of ascitic tap, antibiotic treatment or upper GI endoscopy within last three weeks were excluded from the study. Patients with tubercular peritonitis ere also excluded

A thorough clinical examination with special reference to signs of hepatocellular failure, was done. Then all patients were examined in detail for signs of SBP viz body temperature, abdominal tenderness, encephalopathy. Relevant laboratory tests to establish cytological diagnosis of ascites and SBP were done viz CBP, Renal Function Tests, Liver Functions Tests, Ultrasonography of Abdomen, Urinalysis, Ascitic Fluid Analysis, Conventional Culture and Culture in Blood Culture broth.

Methods

Bed side inoculation of ascitic fluid in blood culture bottles.10 ml. ascitic fluid was drained under aseptic precaution, 4 ml.

was inoculated in blood culture broth immediately. 3 ml. was taken in conventional culture tube and 3 ml. was taken in plain vial for analysis. Blood culture broth was kept in incubator for 24 hours while sample taken in conventional culture tube was inoculated into glucose agar and MacConkey's media and kept in incubator for 24 hours.

Next day the plates were examined for any culture Blood culture broth was checked for any growth, material from blood culture broth was inoculated in MacConkey's medium a glucose agar plate and kept in incubator for bacterial growth. If colonies were found their macroscopic characteristics were noted and slides were prepared. Gram-stained slides were examined under microscope, organism was identified. Then appropriate biochemical tests were done to subclassify the bacteria.

Macroscopic Appearance of Colonies

E. Coli: large thick, greyish white, moist, smooth, opaque, partially translucent disc. On MacConkey's medium colonies are bright pink due to lactose fermentation.

Klebsiella: large, greyish white, moist discoid, lactose fermenting colonies.

Gram staining steps: Primary staining with methyl violet. Application of a dilute solution of iodine. Decolouration with ethanol counterstaining with carbol fuchsin.

Indole Test: Indole production is tested in peptone water culture.5 ml. of Kovac's reagent is added. A red colour indicates a positive reaction. Kovac's reagent consists of Paradimethylaminobenzaldehyde Amyl alcohol- Concentrated HC1.

SBP was diagnosed when a patient fulfilled following criteria. Having ascitic fluid polymorphonuclear count greater than 250/cumm. Having positive ascitic fluid culture. Having an absence of clinical, laboratory, radiological or ultrasonographical data suggesting secondary peritonitis. Variants of SBP were diagnosed by:

- Culture negative neutrophilic ascites.
- Ascitic fluid polymorphonuclear cell count more than 500 cells/cumm.
- Negative ascitic fluid culture. Monomicrobial non-neutrocytic bactericides.
- Positive ascitic fluid culture.
- Ascitic fluid polymorphonuclear cell count less than 250 cells/cumm.

A total of twelve variables were analysed possible predictors of SBP. For qualitative variables patients were grouped according to the presence or absence of variable. For quantitative variables patients were grouped according to the median value of each variable except in the case of ascitic fluid total protein. For this variable the cut off selected was 1 gm per dl because the risk of SBP during hospitalization of cirrhotic patients with ascites and the probability of SBP recurrence have been reported to be much greater in patients with an ascitic fluid protein content less than 1 gm/dl, then in those with higher ascites protein concentration. After grouping the patients, each variable is compared in SBP and Non-SBP groups.

Observation Chart

AGE AND SEX DISTRIBUTION OF PATIENTS WITH SPONTANEOUS BACTERIAL PERITONITIS

Age Group in Yrs.	Male	Female	Total	Percentage
15 - 30	00	01	01	09.09
31 - 45	04	01	05	45.45
More than 45	04	01	05	45.45
TOTAL PERCENT	08 72.72%	03 27.27%	11 100%	100

AGE AND SEX DISTRIBUTION OF PATIENTS WITHOUT SBP

Age Group in Yrs.	Male	Female	Total	Percentage
15 - 30	09	04	13	26.5
31 - 45	08	07	15	30.6
More than 46	17	04	21	42.85
TOTAL PERCENT	34 69.38%	15 30.6%	49 100%	100

DISTRIBUTION OF SYMPTOMS (FEVER & ABDOMINAL PAIN)

Symptoms	All Cases	Percen-tages	SBP	Percen-tage	Non-SBP	Percen-tage
Fever	13	21.6	07	63.6	06	12.24
Abdominal Pain	11	18.3	05	45.45	06	12.24

DISTRIBUTION OF CLINICAL SIGNS

Sign	Total Cases	Percen-tage	SBP	Percen-tage	Non-SBP	Percen-tage
Hepatic Stigmata	16	26.6	06	54.50	10	20.40
Hepatic Encephalopathy	11	18.3	05	45.45	04	08.16
Abdominal Tenderness	11	18.3	05	45.45	06	12.24

DISTRIBUTION OF RELEVENT LABORATORY DATA AMONGS THE PATIENTS

	Total Cases	Percentage	SBP	Percentage	Non-SBP	Percentage
SERUM BILIRUBIN						
<2	42	70	05	45.45	37	75.5
⩾2	18	30	06	54.5	12	24.48
SGOT						
<50 IU	44	43.3	06	54.5	38	77.55
⩾50 IU	16	26.6	05	45.45	11	22.44
SGPT						
<50 IU	47	78.3	06	54.5	41	83.67
⩾50 IU	13	21.6	05	45.45	08	16.32
ASCITIC FLUID TOTAL PROTEIN						
⩾1 gm/dl	55	91.6	08	72.72	47	95.9
<1 gm/dl	05	08.3	03	27.27	02	04.8

AETIOLOGICAL DISTRIBUTION OF CASES

Aetiology	Total Cases	Percentage	SBP	Percentage	Non-SBP	Percentage
CIRRHOSIS	37	61.66	10	90.9	27	55.1
Alcoholic	11	18.33	03	27.2	08	16.32
Postnecrotic	04	06.66	-	-	04	08.16
Cryptogenic	22	36.66	07	63.63	15	30.74
FULMINANT HEPATITIS	01	01.72	-	-	01	02.66
NCPH	06	10.00	-	-	06	12.29
LIVER MALIGNANCY	05	08.33	-	-	05	10.45
NEPHROTIC SYNDROME	09	15.0	-	-	09	18.58
CCF	03	05.0	01	09.09	02	04.08

WBC COUNT AND CULTURE RESULTS IN PATIENTS WITH SBP

Sl. No.	Ascitic Fluid Total Protein	Ascitic Fluid Cell Count	Culture Results	Diagnosis
1.	1.2 gm%	600 mostly Polymorphs	Indole +Ve E.Coli	SBP
2.	1.2 gm%	1200 Cells/cumm Mostly Polymorphs	Sterile	CNNA
3.	1.3 gm%	1200 Cells/cumm Mostly Polymorphs	Indole +Ve E.Coli	SBP
4.	0.6 gm%	2500 Cells/cumm Mostly Polymorphs	Sterile	CNNA
5.	2.5 gm%	3500 Cells/cumm Mostly Polymorphs	E.Coli	SBP
6.	1.2 gm%	2800 Cells/cumm Mostly Polymorphs	Klebsiella	SBP
7.	2.5 gm%	40 Cells/cumm Mixed	E.Coli	BA
8.	1.2 gm%	3600 Cells/cumm Mostly Polymorphs	Sterile	CNNA
9.	0.68 gm%	740 Cells/cumm Mostly Polymorphs	E.Coli	SBP
10.	1.0 gm%	400 Cells/cumm Mostly Polymorphs	E.Coli	SBP
11.	1.8 gm%	5400 cells/count Mostly Polymorphs	Sterile	CNNA

COMPARISON OF LABORATORY DATA AMONGS THE CIRRHOTIC PATIENTS

Laboratory Data	Total Patients With Cirrhosis		With S.B.P.		WITHOUT SBP	
	No.	Percentage	No.	Percentage	No.	Percentage
SERUM BILIRUBIN						
⩽2 mg%	23	62.16%	04	40%	19	70.3%
>2 mg%	14	37.83%	06	60%	08	29.6%
S.G.O.T.						
<50 IU	24	64.86%	05	50%	19	70.3%
⩾50 IU	13	35.1%	05	50%	08	29.6%
S.G.P.T.						
<50	27	72.97%	05	50%	22	81.4%
⩾50	10	27.02%	05	50%	05	18.5%
ASCITIC FLUID TOTAL PROTEIN						
>1 gm/dl	34	91.89%	07	70%	27	100%
⩽1 gm/dl	03	8.10%	03	30%	00	00

Results

In the present study we have included sixty patients of ascites admitted to medical wards of medical college. Study comprised of 42 males (70%) and 18 females (30%). 37 patients (61%) were cirrhotics (Alcoholic cirrhosis11, post necrotic 4, Cryptogenic 22), one patient was having fulminant hepatitis, six patients (10%) were having NCPH, five patients (8.3%) were having liver malignancy, nine patients (15%) were having nephrotic syndrome and three patients (5%) were having CHF.

Eleven patients (18.3%) had spontaneous bacterial peritonitis. Six patients had classical SBP, four had CNNA and one had BA. Out of 11 patients who had SBP 10 were cirrhotics while one was in CHF. None of patients of nephrotic syndrome, NCPH, liver malignancy and fulminant hepatitis had SBP. There was no significant difference in age and sex of the patient who developed SBP.

Fever (63.6%) and abdominal pain (45.45%) were common presenting symptoms. A significant number of patients were asymptomatic. Patients with severe liver disease expressed by presence of hepatic stigmata, hepatic encephalopathy, and abnormal liver function tests, had higher occurrence of SBP. Patients with ascitic fluid total protein less than 1 gm/dl had higher occurrence of SBP. Out of eleven patients of SBP, organism was isolated in seven patients. In six patients E. Coli and in one patient Klebsiella was responsible for SBP. Conventional culture of ascitic fluid found to have no value in isolating the organism. Bed side inoculation of ascitic fluid in blood culture broth demonstrated organism in seven patients.

Statistical Analysis

The collected data was summarized by using frequency, percentage, mean & S.D. To compare the qualitative outcome measures Chi-square test or Fisher's exact test was used. To compare the quantitative outcome measures independent t test was used. If data was not following normal distribution, Mann Whitney U test was used. SPSS version 22 software was used to analyse the collected data. p value of <0.05 was considered to be statistically significant.

Discussion

Numerous studies investigating the occurrence of SBP in cirrhotic patients hospitalized with ascites have reported that this complication occurs in approximately 20% of cases. In our series the occurrence of SBP was 18.33. In most studies only cirrhotics were taken for study, in our study cirrhotic patients were also included. [3-5]

In this study we have found that only one patient without cirrhosis developed S.B.P., rest of the patients who developed S.B.P. were cirrhotics. It indicates that patients with cirrhosis are at higher risk for development of SBP. This is because of reduced antimicrobial activity of ascitic fluid in patients with cirrhosis. None of patients with nephrotic syndrome developed SBP in our series. This clearly shows patients with nephrotic syndrome are at lower risk for development of SBP. We have tried to correlate symptoms, signs laboratory investigations to predict S.B.P. [6]

There was no significant difference in age and sex of the patients with SBP and without SBP. Nutritional status was also not significantly different in two groups. It was found that fever and abdominal pain was present in significantly higher number of cases with SBP, although few patients without SBP had fever and abdominal pain, which may be because of some other pathology. On the other hand, a significant number of patients with SBP were asymptomatic, thus abscence of symptom cannot rule out possibility of SBP in a patient with ascites. These findings correlate with previous studies. [7,8]

Clinical signs, abdominal tenderness, hepatic stigmata and hepatic encephalopathy were present in

significantly higher number of patients with SBP. Higher number of patients in SBP group had hepatic stigmata which reflects severity of hepatic disease. Severe the hepatic disease more are the chances of SBP. It has been mentioned in previous studies that severity of disease correlates with reduced opsonic actively of ascites fluid, this predisposes the patient for SBP.[9,10]

Amongst the patients who presented with hepatic encephalopathy 45.5% found to have SBP. Thus SBP is an important cause of hepatic encephalopathy in patients with ascites. In all patients who present with encephalopathy we strongly suspect SBP as a cause and treatment should be initiated as soon as possible, as SBP is a treatable cause of hepatic encephalopathy.

In the present study serum bilirubin, SGOT, SGPT ware rained in apparently higher number of patients with SBP. This again reflected severity of hepatic disease. Thus, it correlates with the findings of previous workers that patients with severe hepatic disease are prone to develop SBP. Ascitic fluid total protein has been shown to have predictive value for SBP by many workers. In our study 27% of patients with SBP had ascitic fluid total protein less than 1 gel while it was less than 1 gm in only 4% of patients without SBP. Thus, it is apparent that patients with ascitic fluid total protein less than 1 gm/dl are prone to develop SBP. This is because of low opsonic activity of ascitic fluid in such patients. [11-13]

Organism present in intestinal flora are usually involved in development of SBP. In our study in most of the cases E. Coli was isolated from ascitic fluid. In one case the organism involved was Klebsiella. It has been observed that conventional culture has no value in demonstrating organism in ascitic fluid. Culture in blood culture broth is now widely accepted method for isolation of organism from ascitic fluid as ascitic fluid has it's own inhibitory effect on organisms as of blood. In our study we have found 11 (18.3%) patients had SBP. Amongst the patients who developed SBP 6 (54.54%) had classical SBP, 4 (41.32%) had CNNA and 1 (9.09) had bacterascites. Biggins A described 105 cases of SBP, out of which 44 (41.9%) had CNNA. [14,15]

Conclusion

It is concluded that SBP is not an uncommon condition encountered in practice. It can be easily diagnosed by simple laboratory methods available in most of the hospitals in our country. High degree of suspicion is required to pick up the patients with this condition and timely institution of treatment can save many patients. Any patient of ascites, admitted with fever and abdominal pain. SBP should be considered. SBP is a common cause of hepatic encephalopathy. Thus all patients who are admitted with hepatic encephalopathy should be screened for SBP and treatment should be started as early as possible as their condition can be treated successfully with timely intervention.

Declarations: Funding: None.

Availability of data and material: Medical Wards, Department of Medicine, Medical College and associated Hospitals.

Code availability: Not applicable.

Consent to participate: Consent taken.

Ethical Consideration: There are no ethical conflicts related to this study.

Consent for publication: Consent taken

References

1. Garcia-Tsao G. Spontaneous bacterial peritonitis. Gastroenterology Clinics of North America. 1992 Mar 1;21(1):257-75.
2. Koulaouzidis A, Bhat S, Saeed AA. Spontaneous bacterial peritonitis. World journal of gastroenterology: WJG. 2009 Mar 3;15(9):1042.
3. Khan J, Pikkarainen P, Karvonen AL, Mäkelä T, Peräaho M, Pehkonen E, Collin P. Ascites: aetiology, mortality and the prevalence of spontaneous bacterial peritonitis. Scandinavian

journal of gastroenterology. 2009 Jan 1;44(8):970-4.
4. Andreu M, Sola R, Sitges-Serra A, Alia C, Gallen M, Vila MC, Coll S, Oliver MI. Risk factors for spontaneous bacterial peritonitis in cirrhotic patients with ascites. Gastroenterology. 1993 Apr 1;104(4):1133-8.
5. Filik L, Unal S. Clinical and laboratory features of spontaneous bacterial peritonitis. East African medical journal. 2004 Nov 17;81(9):474-9.
6. Paul K, Kaur J, Kazal HL. To study the incidence, predictive factors and clinical outcome of spontaneous bacterial peritonitis in patients of cirrhosis with ascites. Journal of clinical and diagnostic research: JCDR. 2015 Jul;9(7):OC09.
7. Castellote J, Xiol X, Verdaguer R, Ribes J, Guardiola J, Gimenez A, Casais L. Comparison of two ascitic fluid culture methods in cirrhotic patients with spontaneous bacterial peritonitis. American Journal of Gastroenterology (Springer Nature). 1990 Dec 1;85(12).
8. Lee JM, Han KH, Ahn SH. Ascites and spontaneous bacterial peritonitis: an Asian perspective. Journal of gastroenterology and hepatology. 2009 Sep;24(9):1494-503.
9. Runyon BA. Monomicrobial nonneutrocytic bacterascites: a variant of spontaneous bacterial peritonitis. Hepatology. 1990 Oct;12(4):710-5.
10. Llach J, Rimola A, Navasa M, Ginès P, Salmerón JM, Ginès A, Arroyo V, Rodés J. Incidence and predictive factors of first episode of spontaneous bacterial peritonitis in cirrhosis with ascites: relevance of ascitic fluid protein concentration. Hepatology. 1992 Sep;16(3):724-7.
11. Bobadilla MI, Sifuentes JO, Garcia-Tsao GU. Improved method for bacteriological diagnosis of spontaneous bacterial peritonitis. Journal of Clinical Microbiology. 1989 Oct;27(10):2145-7.
12. Pericleous M, Sarnowski A, Moore A, Fijten R, Zaman M. The clinical management of abdominal ascites, spontaneous bacterial peritonitis and hepatorenal syndrome: a review of current guidelines and recommendations. European journal of gastroenterology & hepatology. 2016 Mar 1;28(3):e10-8.
13. Sundaram V, Manne V, Al-Osaimi AM. Ascites and spontaneous bacterial peritonitis: recommendations from two United States centers. Saudi Journal of Gastroenterology: Official Journal of the Saudi Gastroenterology Association. 2014 Sep;20(5):279.
14. Wiest R, Krag A, Gerbes A. Spontaneous bacterial peritonitis: recent guidelines and beyond. Gut. 2012 Feb 1;61(2):297-310.
15. Biggins SW, Angeli P, Garcia-Tsao G, Ginès P, Ling SC, Nadim MK, Wong F, Kim WR. Diagnosis, evaluation, and management of ascites, spontaneous bacterial peritonitis and hepatorenal syndrome: 2021 practice guidance by the American Association for the Study of Liver Diseases. Hepatology. 2021 Aug 26;74(2):1014-48.