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Study of bacteriological profile in spontaneous bacterial peritonitis in cirrhotic patients at a tertiary teaching hospital in Northern India

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Abstract

Background: Spontaneous bacterial peritonitis (SBP) is the infection of ascitic fluid in the absence of any intra abdominal, surgically treatable source of infection in cirrhotic patients with ascites. The diagnosis is confirmed by neutrophil count more than 250/mm^3, associated with or without bacterial growth and culture in ascitic fluid.

Materials and Methods: In this observational study, the ascitic fluid analysis of 60 patients of cirrhosis of liver with spontaneous bacterial peritonitis was studied.

Results: Out of 60 patients, classic SBP was seen in 26 (43.3%) cases and culture negative neurocytic ascites (CNNA) in 34 (56.7%) cases. *E.coli* (57.7%) was the commonest organism cultured followed by Klebsiella (23.07%) and Streptococcus (19.23%). Mortality rate in Classic SBP was 30.76% while it was 11.76% in CNNA., Classic SBP was seen in 26 (43.3%) cases and CNNA in 34 (56.7%) cases. Gram negative organisms were 100% susceptible to imepenem. They were sensitive to third generation cephalosporins and fluoroquinolones. Streptococcus was 100% sensitive to linezolid and less sensitive to third generation cephalosporins.

Conclusion: Classic SBP was seen in 43.3% cases and CNNA in 56.7% cases. *E.coli* (57.7%) was the commonest organism cultured followed by Klebsiella (23.07%) and Streptococcus (19.23%).

Keywords: Spontaneous bacterial peritonitis, ascitic fluid analysis, bacteriological profile, *E. coli*

Introduction

Spontaneous bacterial peritonitis (SBP) is defined by the presence of ≥250 polymorphonuclear cells (PMN)/mm^3 in ascites in the absence of an intra-abdominal source of infection or malignancy. It is the most common bacterial infection in cirrhosis, accounting for 10%–30% of all reported bacterial infections in the patients admitted to hospital.[1-3] In outpatients without symptoms, the prevalence is low (3.5%)or lower, but in the nosocomial setting, the prevalence increases, ranging from 8% to 36%.[4-8]

Depending on the culture and cell count ascitic fluid results, SBP has been classified into two variants: [8]

Bacterascites (BA): It is defined as ascitic fluid leukocyte count <250/mm^3 with positive ascitic fluid culture.

Culture-negative neutrocytic ascites (CNNA): It is defined as ascitic fluid leukocyte count ≥500/mm3 or neutrophil count ≥250/mm^3 with negative ascitic fluid culture.

Current evidence suggests that the spontaneous ascitic fluid infections are due to translocation of the bacteria from the intestine to the mesenteric lymph nodes which results in spontaneous bacteremia and subsequent colonization of ascitic fluid.

It was proposed that intestinal bacterial overgrowth in patients with cirrhosis may be due to a combination of alteration in the local IgA immune response and delay in the intestinal transit.[9]Three main factors are found to be linked in the pathological bacterial translocation.[10]These include: alterations in the gut microbiota, increase in the intestinal permeability and impairment in the host defense.

Materials and Methods

The present study was carried out in 60 patients of cirrhosis with spontaneous bacterial peritonitis admitted in Medicine department in Guru Nanak Dev Hospital attached to Government Medical College, Amritsar. Patients with portal hypertension due to extra hepatic portal vein obstruction and patients treated with antibiotics in the last 2 weeks were excluded. On admission, detailed history and clinical examination of the patient was done. Under strict aseptic conditions diagnostic paracentesis was done for collection of ascitic fluid from all 60 patients. Ascitic fluid was sent to central lab attached to GMC Amritsar for cytology, biochemistry and ascitic fluid culture and sensitivity which was done as per standard biochemical and microbiological procedures Sunder aseptic precautions. Hematological tests included hemoglobin, white blood cell count, differential count, platelet count, erythrocyte sedimentation rate and blood culture. Biochemical tests included random blood sugar, blood urea, serum creatinine and liver function tests. Ultrasonography was done for all patients to find evidence for portal hypertension, size and echo-texture of liver, presence of ascites and splenomegaly. All patients were tested for hepatitis B and hepatitis C.

The data collected was analysed according to the appropriate statistical methods to reach a conclusion.

Results

The present study included 60 patients of cirrhosis with spontaneous bacterial peritonitis. This study was conducted to study the bacteriological profile in spontaneous bacterial peritonitis, antimicrobial susceptibility pattern of various bacterial isolates and to reduce morbidity & mortality in spontaneous bacterial peritonitis by prescribing appropriate antibiotics according to antibiotic sensitivity report of ascitic fluid.

The mean age in present study was 49.18 years. SBP is common in alcoholic cirrhotics. In present study, ethanol (81.7%)was the most common cause of cirrhosis followed by Hepatitis C (11.6%) and Hepatitis B (6.7%).86% cases were males as ethanol is the common etiology whereas 14% were females.70% were in Child Pugh Class C and 30% in Class B.

In present study, Classic SBP was seen in 26 (43.3%) cases and CNNA in 34 (56.7%) cases. *E.coli* (57.7%) was the commonest organism cultured followed by Klebsiella (23.07%) and streptococcus (19.23%). Low ascitic fluid protein levels, high serum bilirubin, high INR and Child class C are risk factors for SBP.

The mortality in patients with culture positive SBP (Classic SBP) is more frequent than with culture negative SBP (CNNA). In present study, mortality rate in Classic SBP was 30.76% while it was 11.76% in CNNA. Gram negative organisms were 100% susceptible to imepenem. They were sensitive to third generation cephalosporins and fluoroquinolones. Streptococcus was 100% sensitive to linezolid and less sensitive to third generation cephalosporins.

Bio chemical data in study group at presentation:

Investigations	Mean±S.D.
Hb	8.27±1.62
Serum Bilirubin	5.18±2.71
SGOT	94.47±58.5
SGPT	73.9±47.90
Serum Protein	6.32±0.75
Serum Albumin	2.53±0.25
Serum Creatinine	2.13±0.65
INR (International Normalized Ratio)	2.22±0.7
TLC (Total Leucocyte Count)	11538.51±3930.80

Ascitic fluid parameters:

Ascitic fluid parameters	AT Admission (MEAN±S.D.)	At Third day (Mean±S.D.)
Ascitic Polymorphonuclear cells	781.32±356.56	243.5±130.91
Ascitic Total Leucocyte Count	1014±389	543.43±263.67
Ascitic Protein	1.15±0.34	
Ascitic Albumin	0.79±0.3	

Distribution of organisms in culture positive cases:

Organism	No. of Culture Positive Cases (n=26) (Classic SBP)	Percentage
E.coli	15	57.7%
Klebsiella	6	23.07%
Streptococcus	5	19.23%

Percentage of mortality in classic SBP and CNNA patients:

Variants of SBP	No. of patients	Mortality	
		No. of patients	Percentage
Classic SBP	26	8	30.76%
CNNA	34	4	11.76%
Total	60	12	20

Table showing antibiotic sensitivity pattern:

	CEFO	CIP	IME	CEFT	LIN	A.CLAV
E.coli (n=15)	8(53.3%)	8(53.3%)	15(100%)	13(65%)	-	7(46.6%)
Klebsiella (n=6)	3(50%)	3(50%)	6(100%)	3(50%)	-	3(50%)
Streptococcus (n=5)	1(33.33%)	0(0%)	-	3(60%)	3(100%)	4(80%)

CEFO- cefotaxime, CIP- ciprofloxacin, IME- imepenem, CEFT- ceftriaxone, LIN- linezolid, A.CLAV- amoxyclav

Discussion

Maximum number of patients of SBP are found in age group of 40-69 years.[11] In this study the mean age was around 49 years which is comparable with other studies. Most of the patients were in fourth and fifth decade of life. The mean age of patients in study done by Fililk et al and Gill et al were 49.91±15.01 years and 49.06±11.35 respectively.[12-13]

The predominant etiology in patients with SBP in this study was found to be due to ethanol related cirrhosis (81.6%), followed by HCV (11.6%) and HBV (8.6%). All patients with alcohol related cirrhosis were males who frequently consume significant amounts of alcohol and present more frequently with advanced liver disease. This was in concordance with other study done by Lata et al and Paul et al.[14-15]In our study of 60 patients, 70% of the patients with SBP were in Child Pugh class C, which is consistent with study done by Amarapurkar DN et al and Jain et al.[16-17] The severity of the liver disease is the most important predisposing factor for the development of SBP.

In the present study, baseline investigations revealed that patients had a low haemoglobin value (mean of 8.27±1.62), high blood TLC (mean of 11538.5 ±3930.8), a high serum bilirubin (mean of 5.18±2.71), a high SGOT (mean of 94.7±58.5), a high INR value (mean of 2.22±0.77), high ascitic fluid TLC (mean of 1014±389), high ascitic fluid PMN count (mean of 781.32±356.56), low ascitic fluid albumin (mean of 0.79±0.3) and a low ascitic fluid protein (mean of 1.72±0.31). This might be due to its relation with the advanced stage of the liver disease. These findings are comparable with study done by Bankar et al.[18]In present study, 26 (43.3%) patients had classic SBP and 34 (56.7%) patients had CNNA variant of SBP. Similarly CNNA was most common variant of SBP in studies done by Bhat et al and Purohit et al.[19-20] However in study done by Jain et al and Archana et al, culture positive SBP was more common.[20-21]

In present study, the most frequent organism isolated in Classic SBP (n=26) was *E.coli* (n=15, 57.7%) followed by Klebsiella (n=6, 23.07%) and *Streptococcus pneumonia* (n=5, 19.23%). This study result was comparable to a similar study conducted by Doddamani et al which showed that E.coli (50%) was most frequently cultured organism followed by Klebsiella in 37% of cases.[22] In study done by Bankar et al also showed that *E.coli* (57.89%) was the commonest bacteria. In study done by Haider et al, streptococcus (16%) was most commonly cultured in gram positive organisms.[23] In study done by Jain et al, the commonest organism was coagulase positive *Staphylococcus aureus* 8 (44.44%) followed by *E.coli* (22.22%).[17]

The overall mortality in present study was 20%. In classic SBP mortality was 30.76% (n=8) and 11.76% (n=4) in CNNA. Pelletier et al also revealed a higher mortality with culture positive SBP patients as compared to patients with CNNA.[24] In study done by Bankar et al, overall mortality was 24% and more in CNNA group.[18]

Antibiotic susceptibility was done in present study. Both *E.coli* and Klebsiella were 100% sensitive to imepenem. E.coli was 53.3% sensitive to cefotaxime and ciprofloxacin while Klebsiella was 50% sensitive to both of them. Bankar et al showed 54.29% susceptibility of cefotaxime for gram negative bacilli which is comparable with our study. *E.coli* and Klebsiella were 60% and 50% sensitive to amoxyclav respectively and 65% sensitive to ceftriaxone. Streptococcus showed 100% sensitivity to Linezolid, 60% and 80% sensitivity to ceftriaxone and amoxyclav respectively. It showed less susceptibility to cefotaxime. Similar results were seen in antibiotic susceptibility tests done by Mirnejad et al and Nithya et al.[25-26]

Conclusion

In present study, Classic SBP was seen in 26 (43.3%) cases and CNNA in 34 (56.7%) cases. E.coli was the commonest organism cultured followed by Klebsiella and streptococcus. Low ascitic fluid protein levels, high serum bilirubin, high INR and Child class C are risk factors for SBP. The mortality in patients with culture positive SBP (Classic SBP) is more frequent than with culture negative SBP (CNNA). In present study, mortality rate in Classic SBP was 30.76% while it was 11.76% in CNNA. Gram negative organisms were 100% susceptible to imepenem. They were sensitive to third generation cephalosporins and fluoroquinolones. Streptococcus was 100% sensitive to linezolid and less sensitive to third generation cephalosporins.

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References

1. Fernandez J, Navasa M, Gomez J, Colmenero J, Vila J, Arroyo V, et al. Bacterial infections in cirrhosis: epidemiological changes with invasive procedures and norfloxacin prophylaxis. Hepatology 2002;35 (1):140–8.
2. Caly WR, Strauss E. A prospective study of bacterial infections in patients with cirrhosis. J Hepatol 1993;18:353–8.
3. Pinzello G, Simonetti RG, Craxi A, Di Pizza S, Spano C, Pagliaro L. Spontaneous bacterial peritonitis: a prospective investigation in predominantly nonalcoholic cirrhotic patients. Hepatology 1983;3:545–9.
4. Evans LT, Kim WR, Poterucha JJ, Kamath PS. Spontaneous bacterial peritonitis in asymptomatic outpatients with cirrhotic ascites. Hepatology 2003;37:897–901.
5. Castellote J, Girbau A, Maisterra S, Charhi N, Ballester R, Xiol X. Spontaneous bacterial peritonitis and bacterascites prevalence in asymptomatic cirrhotic outpatients undergoing large-volume paracentesis. J Gastroenterol Hepatol 2008;23:256–9.
6. Jeffries MA, Stern MA, Gunaratnam NT, Fontana RJ. Unsuspected infection is infrequent in asymptomatic outpatients with refractory ascites undergoing therapeutic paracentesis. Am J Gastroenterol 1999;94:2972–6.
7. Conn HO, Fessel JM. Spontaneous bacterial peritonitis in cirrhosis: variations on a theme. Medicine (Baltimore) 1971;50:161–97.
8. Rimola A, Garcia-Tsao G, Navasa M, Piddock LJ, Planas R, Bernad B, et al. Diagnosis, treatment and prophylaxis of spontaneous bacterial peritonitis: a consensus document. International Ascites Club. J Hepatol 2000;32:142–53.
9. Jose Such, Runyon BA. Spontaneous bacterial peritonitis. Clinical Infectious Diseases 1998; 27:669 – 76.
10. Wiest R, Garcia–Tsao G. Bacterial translocation in cirrhosis. Hepatology 2005;41:422- 33.
11. Sagar KV, Reddy PR, Chandrasekhar S, Harish D. A clinical study of spontaneous bacterial peritonitis in cirrhosis of liver with ascites in teritiary care hospital. J Evid Based Med Health. 2016;3 (1):36-41.
12. Filik L, Unal S. Clinical and laboratory features of spontaneous bacterial peritonitis. East African Medical Journal. 2004;81 (9):474-9.
13. Gills AS, Singh A, Matreja PS, Chinna RS, Mahajan R, Chhina DK. Spontaneous bacterial peritonitis in liver cirrhosis: An Indian perspective. Euroasian J Hepato-Gastroenterol. 2012;2 (1):14–19.
14. Lata J, Fejfar T, Krechler T, Musil T, Husová L, Šenkyrík M, Dolina J, Vanasek T. Spontaneous bacterial peritonitis in the Czech Republic: prevalence and aetiology. European journal of gastroenterology & hepatology. 2003 Jul 1;15(7):739-43.
15. 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;9 (7):09.
16. Amarapurkar DN, Viswanathan N, Parikh SS, Kalro RH, Desai HG. Prevalence of spontaneous bacterial peritonitis. J Assoc Physicians India 1992;40(4):236−8
17. Jain AP, Chandra LS, Gupta S, Gupta OP, Jajoo UN, Kalantri SP. Spontaneous bacterial peritonitis in liver cirrhosis with ascites. J Assoc Physicians India 1999;47 (6):619–21.
18. Bankar S, De A, Baveja S. Study of ascitic fluid for diagnosis of spontaneous bacterial peritonitis (SBP) in adult patients with cirrhosis. J. International Medical and applied. 2014;3 (9):768.
19. Bhat G, Vandana KE, Bhatia S, Suvarna D, Pai CG. Spontaneous ascitic fluid infection in liver cirrhosis: bacteriological profile and response to antibiotic therapy. Indian Journal of Gastroenterology. 2013 Sep 1;32 (5):297-301.
20. Purohit PH, Malek SS, Desai KJ, Sadadia M. A study of bacteriological profile of ascitic fluid in suspected clinical cases of spontaneous bacterial peritonitis at a tertiary care hospital in India. Inter J Med Sci Public Health. 2015;4:4.
21. Bhat A. A Study on Clinical and Laboratory Features of Spontaneous Bacterial Peritonitis in Chronic Liver Disease. Journal of Medical Science and Medical Research. 2017; 5 (4):21028-33.
22. Doddamani GB, Pujar S, Kora SA. Spontaneous Bacterial Peritonitis in Ascites: A prospective study in a tertiary care hospital. J Clin Diagn Res. 2010;4:2737-41.
23. Haider I, Ahmad I, Rashid A, Bashir H, Faheem M. Causative organisms and their drug sensitivity pattern in ascitic fluid of cirrhotic patients with spontaneous bacterial peritonitis. Journal of Postgraduate Medical Institute (Peshawar-Pakistan). 2011;22 (4):43-9.

24. Pelletier G, Salmon D, Ink O, Hannoun S, Attali P, Buffet C, Etienne JP. Culture-negative neutrocytic ascites: a less severe variant of spontaneous bacterial peritonitis. Journal of hepatology. 1990 May 1;10(3):327-31.
25. Mirnejad R, Jeddi F, Kiani J, Khoobdel M. Etiology of spontaneous bacterial peritonitis and determination of their antibiotic susceptibility patterns in Iran. Asian Pacific Journal of Tropical Disease. 2011;1 (2):116-8.
26. Nithya C, Rathnapriya N, Vasanthi S. A Study on Bacterial Isolates and Their Antibacterial Susceptibility Pattern in Patients with Spontaneous Bacterial Peritonitis in a Tertiary Care Hospital. Int J Curr Microbiol App Sci 2017;6 (9):3704-9.

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