ISSN: 0975-833X

Available online at http://www.journalcra.com

International Journal of Current Research
Vol. 10, Issue, 12, pp.76037-76039, December, 2018

DOI: https://doi.org/10.24941/ijcr.33259.12.2018

INTERNATIONAL JOURNAL OF CURRENT RESEARCH

RESEARCH ARTICLE

PREVALENCE OF SPONTANEOUS BACTERIAL PERITONITIS IN LIVER CIRRHOSIS WITH ASCITES IN KIMS HOSPITAL BENGALURU

[1]Dr. Keshava H. K., [2]Dr. Vinay Babu C.S., [2]Dr. Varun Manjunath, [2]Dr. Rajesh Kiran, K. V., [3]Dr. Sushmitha Anantha Murthy, [3]Dr. Vasudha K. V. and [3]Dr. Aisiri Anand

[1]Professor, Department of General Medicine, KIMS Hoapital, Bengaluru
[2]Junior Resident, Department of General Medicine, KIMS Hospital, Bengaluru
[3]Department of General Medicine, Kempegowda Institute of Medical Sciences and Research Centre, V. V. Puram, Bengaluru - 560004

ARTICLE INFO

Article History:
Received 13th September, 2018
Received in revised form
16th October, 2018
Accepted 17th November, 2018
Published online 29th December, 2018

Key Words:

Spontaneous,
KIMS,
SBP.

ABSTRACT

Aims and objectives: To determine the prevalence of spontaneous bacterial peritonitis in liver cirrhosis with ascites in KIMS hospital Bengaluru. **Materials and methods:** 58 patients with liver cirrhosis with ascites, who were admitted to KIMS hospital Bengaluru from March 2017 to August 2018) were studied retrospectively. The diagnosis of liver cirrhosis was established on the basis of clinical evaluation, biochemical parameters, abdominal ultrasound and viral markers. All the patients had abdominal paracentesis done within 48 hours of admission under aseptic condition and the data obtained were analysed. **Results:** The mean age of the studied population was 52±12 years (age range 32-66 years). Of the 58 patients 9 patients had SPB (15.5%), Among SBP patients culture is positive in 44.4%, while CNNA was found in 55.6% patients. The prevalence of MNB was 13.8% (8/58) in this study. Of those with SBP, 83.3% had monomicrobial infection with E.coli (60%) being the predominant organism followed by Klebsiella species (20%). Gram positive organisms Streptococcal species and Staphylococcus aureus both together contributing for 20 % of SBP. Patients with SBP had significantly lower platelet count when compared with those without SBP ($p < 0.05$). Also, international normalization ratio (INR) was significantly higher in those patients with SBP compared with those without SBP, ($p< 0.05$). **Conclusion:** Patients with cirrhosis of liver with ascites are at increased risk of developing SBP. Hence it is imperative to do diagnostic abdominal paracentesis in any cirrhotic patients with ascites and suggestive symptoms compatible or suggestive of SBP.

Citation: ***Dr. Keshava H. K., Dr. Vinay Babu C.S., Dr. Varun Manjunath, Dr. Rajesh Kirsn, K. V., Dr. Sushmitha Anantha Murthy, Dr. Vasudha K. V. and Dr. Aisiri Ananad*** **2018.** "Prevalence of spontaneous bacterial peritonitis in liver cirrhosis with ascites in kims Hospital Bengaluru", *International Journal of Current Research*, 10, (12), 76037-76039.

INTRODUCTION

One of the most common bacterial infections in cirrhosis is spontaneous bacterial peritonitis (SBP), an infection of ascites unique to patients with cirrhosis, particularly those with severely decompensated cirrhosis. Conn and Fessel first described a syndrome of infected ascitic fluid in patients with hepatic cirrhosis, which they named SBP (CONN, 1971). It is called spontaneous because it occurs in the absence of a contiguous source of infection (e.g. intestinal perforation, intra-abdominal abscess) and in the absence of an intra-abdominal inflammatory focus (e.g. abscess, acute pancreatitis, cholecystitis) (Fernandez, 2000 and Guadalupe Garcia, 2018). All patients with cirrhosis and ascites are at risk of SBP and the prevalence of SBP in outpatients is 1.5-3.5% and about 10%-30% in hospitalized patients (Rimola, 1984 and Fernandez, 2000). Half of the episodes of SBP are present at the time of hospital admission while the rest are acquired during hospitalization (Rimola, 2000). The diagnosis of SBP is established with a polymorphonuclear cell count of more than 250/mm3. Because ascites cultures are frequently negative (despite inoculation into blood culture bottles) and because isolating an organism is extremely useful for therapy, blood cultures should be obtained simultaneous to ascites cultures. SBP is typically a monobacterial infection. The most common infecting organisms are *Escherichia coli*, group D streptococci, or Klebsiella (Garcia, 1992 and Dever, 2015). Bacteria of gut origin are the most commonly isolated causative organisms. Therefore, migration of enteric bacteria across the intestinal mucosa to extraintestinal sites and the systemic circulation (bacterial translocation) has been implicated in its pathogenesis (Garcia, 1995). In cirrhosis, an overactive sympathetic nervous system slows gut motility and facilitates bacterial stasis and overgrowth, thereby facilitating bacterial translocation.

Corresponding author: **Dr. Keshava H. K.,**
Professor, Department of General Medicine, KIMS Hoapital, Bengaluru.

Persistence of bacteria in extraintestinal sites is favoured by impaired host defences. In cirrhosis, host defences are abnormal because of portosystemic shunting and impaired reticuloendothelial function. Neutrophils are abnormal in the alcoholic. Decreased synthesis of proteins, such as complement and fibronectin, result in diminished adhesiveness and decreased bacterial phagocytosis (Homann, 1997). Ascitic fluid favours bacterial growth and deficient ascitic opsonins lead to defective coating of bacteria which are indigestible by polymorphs. The opsonic activity of the ascitic fluid is proportional to protein concentration and SBP is more likely if ascetic fluid protein is less than 1 g/dL (Runyon, 1988). SBP should be suspected when a patient with cirrhosis deteriorates, particularly with encephalopathy, acute kidney injury and/or jaundice. Patients with variceal bleeding or with previous SBP are at particular risk. Pyrexia, local abdominal pain and tenderness, and systemic leukocytosis may be noted. These features, however, may be absent and the diagnosis is made following a high index of suspicion with examination of the ascetic fluid (cell count and cultures). The aim of this study was to determine the prevalence and characteristics of SBP among in-patients with cirrhosis and ascites seen at our facility and to compare the results with studies elsewhere.

MATERIAL AND METHODS

Fifty eight patients with liver cirrhosis and ascites who were admitted into the Medical ward of the Kempegowda Institute of Medical Sciences and Research Institute, Bengaluru from March 2017 to August 2018 were studied retrospectively. The diagnosis of liver cirrhosis was based on clinical, ultrasonographic, biochemical parameters and viral markers. Exclusion criteria were 1) clinical symptoms of infection (e.g. fever, chills, nonspecific abdominal pain or tenderness, impaired peristalsis), 2) developing de novo or worsening hepatic encephalopathy, 3) gastrointestinal bleeding within the last month, 4) worsening of renal function within last 30 days, 5) antibiotic treatment or norfloxacin prophylaxis at admission. Patients with non-cirrhotic ascites were not considered for evaluation. All the patients had abdominal paracentesis done within 48 hours of admission under aseptic condition. Ascitic fluid analysis was done that included absolute neutrophils count, culture sensitivity, ascitic albumin and cytology. The Child-Pugh classification was applied. The diagnosis of SBP was based upon criteria recommended by the International Ascites Club and published in 2000 (Rimola, 2000).

Five variants of ascitic fluid infection were defined as follows: 1) bacterial peritonitis (SBP) with an absolute neutrophil (PMN) count of ≥ 250 cells/mm3 associated with the presence of a single organism on culture; 2) monomicrobial nonneutrocytic bacterascites (MNB) with PMN < 250 cells/ mm3 and growth of a single organism on culture; 3) culturenegative neutrocytic ascites (CNNA) with PMN ≥ 250 cells/mm3 in the absence of culture growth; 4) secondary bacterial peritonitis defined as PMN ≥ 250 cells/mm3 in association with polymicrobial culture growth and surgically treatable source of infection (e.g. intestinal perforation, abscess); 5) polymicrobial bacterascites which presents with PMN < 250 cells/mm3 and polymicrobial growth on culture (in the rare case of perforation of the bowel by the paracentesis needle). An ethical clearance for this study was obtained from the Institutional Ethical committee, KIMS, Bangalore. Data obtained were analyzed using the statistical package for social sciences (SPSS) statistical software.

RESULTS

Our retrospective study included 58 patients, the mean age was 52±12 years (age range 32- 66 years). 52(89.6%) were males and 6 (10.4%) were females giving a male to female ratio of 8.6:1. Among 58 patients, 8 (13.8%) were less than 40 of age, while majority 38 (65.6) were between 40 to 60 and the remaining 12 (20.6%) were more than 60 age group (Table 1).

Table 1. Age and gender distribution of the study population

Age group (Yrs)	Male	Female	Total
< 40	08	-	08
40-60	34	4	38
>60	10	2	12
Total	52	6	58

SBP was diagnosed in 9 (15.5%) patients. Of the 9 who had SPB, culture positive SBP was found in 4 (44.4%), while CNNA was found in 5 (55.6%) patients. The prevalence of MNB was 13.8% (8/58) in this study (Table 2).

Table 2. Ascitic culture and neutrophil counts

Culture	Ascitic Neutrophils		TOTAL
	<250/mm^3	>250/mm^3	
Positive	8	4	12
Negative	41	5	46
Total	49	9	58

Among 12 patients with culture positivity, 10(83.3%) had monomicrobial infection of which 6 patients had E. coli (60%), 2(20%) patient had Klebsiella species, 1 patient(10%) had Staphylococcus aureus and 1(10%) patient had Streptococcal species. Two patients cultures were mixed growth (Table 3).

Table 3. Bacteriology

Bacteria	No. of patients	Percentage
E. coli	6	60%
Klebsiella species	2	20%
Streptococal species.	1	10%
Staphylococccus aureus	1	10%
Total	12	

Ascitic fluid protein was less than 1mg/dl in 89.6% the patients with SBP. Majority (75.8%) of the patients with SBP were in Child's grade C, while the remaining 24.2% were in Child's grade B. 62.1% had encephalopathy. Patients with SBP had significantly lower platelet count when compared with those without SBP ($p < 0.05$). Also, international normalization ratio (INR) was significantly higher in those patients with SBP compared with those without SBP ($p < 0.05$).

Table 4. Complications of cirrhosis

Sl. No.	Complications	Percentage
1	Portal Hypertension	86.2% (50)
2	Hepatic Encephalopathy	62.1%(36)
3	SBP	15.5% (9)
4	Hepatorenal syndrome Type 1 & 2	20.6%(12)
5	Coagulopathy (Thrombocytopenia)	77.5%(45)
6	Anemia	40%(40)

DISCUSSION

Spontaneous bacterial peritonitis (SBP) is a potentially life-threatening complication in patients with cirrhosis. The prevalence of SBP in outpatients is 1.5-3.5% and about 10%-30% in hospitalized patients (Rimola et al., 1984 and

Fernandez *et al.,* 2000). The prevalence of SPB in our study was found to be 15.5%. This prevalence is similar to 10-30% found by most studies done by Rimola A *et al.,* (1984), Fernandez *et al.* Gunjača *et al.* (2010).The prevalence of CNNA (a variant of SPB) in this study was 8.6% and this was similar to that reported by Runyon *et al.,* (Runyon, 1988). In our study among patients with culture positivity, 83.3% had monomicrobial infection with aerobic Gram-negative bacilli being responsible for more than two- third of all cases with Escherichial coli being the most common followed by Klebsiella species(20%), this microbial growth is similar to the study publishe by Runyon *et al.* (1990). Nearly 20% were caused by Gram-positive organisms with Streptococcal species and Staphylococcus aureus. Two patients had mixed growth. In the study conducted in Khyber Teaching Hospital, Peshawar 2003, showed E. coli was isolated in 58.13%, Streptococcus pneumoniae in 18.60%, Staphylococcus aureus in 9.13%, Klebsiella in 9.13% and Acinectobacter in 4.63% (Iqbal, 2004). Monomicrobial nonneutrocytic bacterascites (MNB) with PMN < 250 cells/ cu.mm and growth of a single organism on culture was found in 13.8% of patients. Anaerobes were not found in our study as a cause of SBP. The clinical presentations found in this study were; Abdomen distension (90%), Jaundice (74%), fever (70%), encephalopathy (62%), pain abdomen (42) and This was consistent with the findings of Runyon BA *et al.,* (Garcia-Tsao, 2005). Complications of cirrhosis of liver found in our study Portal Hypertension (86.2%), Hepatic Encephalopathy(62.1%), spontaneous bacterial peritonitis(15.5), Hepatorenal syndrome Type 1 & 2(20.6%), Coagulopathy (Thrombocytopenia-77.5%) and Anemia (40%).

Conclusion

Results of our retrospective study indicate that liver cirrhosis patients are more prone for SBP. We observed the trend towards more frequent occurrence of the infection in patients with advanced liver disease (Child-Pugh C group). It is imperative that awareness campaign be vigourously pursued as regards early presentation to reduce the morbidity and mortality in this group of patients. It is equally recommended that diagnostic abdominal paracentesis for cell count and culture in any patient with onset of ascites or cirrhotic patients with ascites and suggestive symptoms compatible or suggestive of SBP be carried out.

Acknowledgement

We extend our gratitude to all the faculty members of the Department of General Medicine and all other supporting departments and the management for giving their valuable opinion and suggestions during discussion of the case and also extending their help in working towards this article. We extend our gratefulness to all the patients and their family members. A special mention goes to Dr. R. Manjunath, Professor and HOD, Department of Medicine for giving his support in making this study a success.

REFERENCES

Conn HO, Fessel JM. 1971. Spontaneous bacterial peritonitis in cirrhosis: variations on a theme. *Medicine.,* 50:161- 97.

Dever, J.B., Sheikh, M.Y. 2015. Review article: spontaneous bacterial peritonitis–bacteriology, diagnosis, treatment, risk factors and prevention. *Aliment Pharmacol Ther*., 41(11): 1116–1131.

EASL clinical practice guidelines on the management of ascites, spontaneous bacterial peritonitis, and hepatorenal syndrome in cirrhosis. *J Hepatol*., 2010. 53(3): 397–417.

Fernandez, J., Bauer, T.M., Navasa, M., Rodes, J. 2000. Diagnosis, treatment, and prevention of spontaneous bacterial peritonitis. *Best Pract Res Clin Gastroenterol.,* 14(6):975-990. PubMed | Google Scholar

Fernandez, J., Navasa, M., Gomez, J. *et al.,* 2000. Bacterial infections in cirrhosis: epidemiological changes with invasive proceduresand norfloxacin prophylaxis. Hepatology., 35(1): 140-148.

Garcia-Tsao G. 1992. Spontaneous bacterial peritonitis. *Gastro Clin North Am.,* 21(1): 257–275.

Garcia-Tsao, G. 2005. Spontaneous bacterial peritonitis. Rev Gastroenterol Mex. 70 Suppl 3:66-8. PubMed | Google Scholar

Garcia-Tsao, G., Lee, F.Y, Barden, G.E. *et al.,* 1995. Bacterial translocation to mesenteric lymph nodes is increased in cirrhotic rats with ascites. *Gastroenterology.,* 108: 1835–1841.

Guadalupe Garcia. Ascites. In: James S. Dooley, Anna S. F. Lok (Eds). Sherlock's Diseases of the Liver and Biliary System. 13th ed. UK: John Wiley & Sons Ltd.; 2018: p 127-150

Gunjača I, Francetić I. Prevalence and clinical outcome of spontaneous bacterial peritonitis in hospitalized patients with liver cirrhosis: a prospective observational study in central part of Croatia. *Acta clinica Croatica.*, Mar 1;49(1):11-8.

Homann, C., Varming, K., Hogasen, K. *et al.,* 1997. Acquired C3 deficiency in patients with alcoholic cirrhosis predisposes to infection and increased mortality. Gut., 40: 544–549.

Iqbal S, Iman N, Alam N. Incidence of spontaneous bacterial peritonitis in liver cirrhosis, the causative organism and antibiotic sensitivity. *J Postgrad Med Inst.,* 2004. 18: 614-619. PubMed | Google Scholar

Rimola A, Garcia-Tsao G, Navasa M, Piddock LJ, Planas R, Bernard B, Inadomi JM. 2000. Diagnosis, treatment and prophylaxis of spontaneous bacterial peritonitis : a consensus document. *International Ascites Club. J Hepatol.* 32(1):142-53.

Rimola A, Gracia-Tsao G, Navasa M, *et al.,* 2000. Diagnosis, treatment and prophylaxis of spontaneous bacterial peritonitis: a consensus document. *International Ascites Club. J Hepatol.,* 32(1):142-153. PubMed | Google Scholar

Rimola A, Soto R, Bory F, Arroyo V, Piera C, Rodes J. 1984. Reticuloendothelial system phagocytic activity in cirrhosis and its relation to bacterial infections and prognosis. Hepatology., 4(1):53-58.

Runyon BA, Canawati HN, Akriviadis EA. 1988. Optimisation of ascitic fluid culture technique. *Gastroenterology.* 95(5): 1351-1355. PubMed | Google Scholar

Runyon BA. 1988. Patients with deficient ascitic fluid opsonic activity are predisposed to spontaneous bacterial peritonitis. *Hepatology.,* 8(3): 632–635.

Runyon BA. Monomicrobial nonneutrocytic bacterascites: A variant of spontaneous bacterial peritonitis. *Hepatology.* 1990. 12(4 pt. 1):710-715. PubMed | Google Scholar

****