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Frequency of Spontaneous Bacterial Peritonitis in Cirrhosis of Liver in Department of Medicine at People Medical College Hospital Nawabshah Pakistan

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Authors' contributions

This work was carried out in collaboration among all authors. Author JKD designed the study, performed the statistical analysis, wrote the protocol and wrote the first draft of the manuscript. Authors AS, SAPT, AAJ, AA and MSR managed the analyses of the study and managed the literature searches. All authors read and approved the final manuscript.

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ABSTRACT

Introduction: Bacterial Infections in cirrhosis of liver is one of the main complications with increase mortality rate. Infection of ascitic fluid in cirrhosis of liver was introduced by Conn and fessel [2], termed as spontaneous bacterial peritonitis.
Objective: To determine the frequency of Spontaneous Bacterial Peritonitis (SBP) in patients presenting with cirrhosis of liver.
Methodology: It is a descriptive cross-sectional study which was performed in the Medicine Ward

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at People's Medical College Hospital Nawabshah Pakistan from 1st June 2018 to 31 July 2020. Total 100 admitted patients were selected for this study, male patient were 58 and female patient were 42. Consent was taken from the patients and their relatives. Primary and secondary outcomes measured. All the patients with diagnosis of cirrhosis of liver were included for this study, ascites due to other causes and patients on antibiotic therapy were excluded from this study. Ascitic fluid aspirated for culture and DR. Blood CP, Urea, Creatinine, LFT and PT was performed.

Results: Among these 100 patients 13 were culture positive raised ascites PMN, 17 were culture negative raised ascites PMN. *E. coli* was detected in majority of the culture positive patients. 70 patients presented with sterile ascites. SBP patients presented with fever, abdominal pain, jaundice, Encephalopathy and raised prothrombin time.

Conclusion: SBP common complication of cirrhosis associated with high mortality. All cirrhotic Patients admitted in medical wards with history of abdominal pain, fever and Encephalopathy, diagnostic paracentesis is compulsory. Early management with intravascular expansion with albumin and antibiotics with good prognosis mortality can be reduced.

Keywords: Ascites; ascitic fluid culture; cirrhosis of liver; Spontaneous Bacterial Peritonitis (SBP).

1. INTRODUCTION

Bacterial Infections in cirrhosis of liver is one of the main complication [1], with increase mortality rate. Infection of ascitic fluid in cirrhosis of liver was introduced by conn and fessel [2], termed as spontaneous bacterial peritonitis. Cirrhosis of liver with Acute decompensation [3], associated with high morbidity and mortality 25% to 46% [4]. 10% to 30% ratio of seriously ill patients is a life threatening complication. Due to deficiency of paneth cell defensins [4], there is an increase prevalence of Enterobacteriaceae. Portal hypertension, reduction in pancreaticobilliary secretion and decreased motility of gut [5], resulting ascites. With increased portal pressure, splanchnic vasodilation and activation of renin angiotensin Sodium and water retention, with the result fluid in peritoneal cavity [6]. By the process of bacterial translocation mesenteric lymph nodes are infected when bacteria with their products cross the intestinal mucosal barrier, enter blood stream then ascitic fluid [7]. Transudate fluid of ascites is associated with poor opsonic activity of bacteria and provide favourable environment for growth of bacteria [8]. Frequent bacterial complication of cirrhosis of liver is sub acute bacterial peritonitis, followed by pneumonia, spontaneous bacteremia, urinary tract infection and skin and soft tissue infection [9]. During spontaneous bacterial peritonitis patient develop signs of decompensating, like hepatic encephalopathy, renal failure, gastrointestinal bleeding and progression of ascites [10]. Presentation of SBP are abdominal pain, encephalopathy, fever, diarrhea, ileus ,hypothermia and shock. Liver functions are severely impaired. Patients belong to child pugh class B or C [11]. Diagnosis of SBP is made by examination of ascitic fluid. DIC and fibrinolysis are contraindication for paracentesis [12]. Examination of ascitic fluid is necessary for all cirrhotic patients who develop abdominal pain, fever, change in mental status,septic shock,and during hematemesis before the adminsteration of antibiotics [13]. Single best investigation to diagnose infection in Ascitic fluid is PMN count > 250 cells/mm3. Diagnostic yield is increased from 40 to 80% if ascitic fluid 5-10 cc is inoculated in blood culture bottle. Blood culture is positive in 50% of patients presented with SBP, blood culture should be sent at the same time [14]. Circulating immune cells are decreased and decreased hepatic synthesis of immune molecules such as compliment system [15]. Low ascitic protein concentration (<1.5 g/dL) ,upper gastrointestinal bleeding and past history of SBP are the known risk factors for development SBP in Cirrhosis and ascites [16]. Treatment should be started immediately with cefotaxime 2 gm i/v 12 hourly, or ceftriaxone alternative piperacillin/tazobactam. Recurrence of SBP can be reduced with quinolones (Ciprofloxin or Norfloxin). In cirrhotic patients with past treatment of SBP chances of recurrence can be reduced with norfloxin 400 mg/day [17].

2. METHODOLOGY

Study was performed in the department of medicine at People Medical College Hospital Nawabshah from 1st June 2018 to 31 July 2020. 100 Patients were selected for this study. Out of 100 patients male were 58 and female were 42, age ranged from 35 to 65 years. Consent was taken from the patients or relatives. Performa was used for the Study. Diagnosis of cirrhosis was based on history of the patient, examination

of the patient, laboratory findings and ultrasound apearence. After aseptic measures ascitic fluid was collected by disposable syringe about 20-30 cc, 10cc of ascitic fluid was inoculated in blood culture bottle at the same time sent laboratory for culture of fluid and from remaining fluid detailed report. For Statistical Analysis SPSS 23 version was used.

- **Inclusion criteria:** All patients with cirrhosis of liver, ascites with fever tender abdomen, no any history of antibiotic.
- **Exclusion criteria:** Secondary peritonitis, and previous history of antibiotic.

3. RESULTS

Total 100 patients enrolled for this study. 65 patients were anti HCV + ve, 19 patients HBs Ag +ve and 16 patients were positive HBs Ag and anti HCV positive. 49 patients belong to class A, 16 patients were class in B and 35 patients in class C according to Childs Pughs Classifications. Among these 100 patients 13 were culture positive, 17 were culture negative with raised ascites PMN level. 70 patients were with sterile ascites. *E. coli* was detected in 8 patients, Klebsiela in 3 patients, Streptococcus in 1 patient and Staphylococcus in 1 patient. Severity of illness was more in culture positive and culture negative Neutocytic ascites. Serum Bilirubin, SGPT, WBC Count, Urea, Creatinine and P T was raised in this group of patients. Ascites PMN count was raised and ascites protein was decreased as mentioned in descriptive table.

4. DISCUSSION

One of the serious complications of cirrhosis is SBP. SBP needs early diagnosis, timely application of antibiotic therapy mortality can be reduced [18], to prevent patients from mortality. Frequency of SBP ranges from 32-64% reported from different studies in Pakistan [19]. Mortality rate associated with SBP 10-15% in a study [20]. SBP Patients can present with encephalopathy, acute variceal bleeding, abdominal pain, diarrhea and ileus during routine follow up [21]. Risk of SBP in cirrhosis of liver is increased in patients with raised PT indicating severity of liver disease; increased serum Bilirubin level and decreased ascitic fluid protein our findings are in line with reported data [22]. Ascitic fluid level of total protein is raised in cirrhotic patients on diuretic therapy, increases ascitic fluid level of total protein, opsonic activity and compliment level of ascetic fluid. It may prevent occurrence of SBP this is due to fluid loss in urine [23]. From stomach to colon 1000 or more trillion bacteria per gram of faeces in colon are present in healthy individual [24]. Deleterious effects due to normal intestinal flora mechanisms leading to SBP in advanced liver disease [25]. Immune dysregulation in cirrhotic patients intestinal permeability increased and pathological translocation of bacteria [26]. Most common organism for SBP are *E. coli* and *K. pneumonia* [27]. In recent years gram positive cocci are major pathogen compared to gram negative cocci in several studies [28]. In patients with community acquired infections gram negative bacteria were increased in number, and gram positive bacteria were predominant in noscomial infections, studies received from china [29]. Studies published between 1971 and 1991, 263 ascitic fluid culture organisms noted were Streptococcus 30%, *E. coli* were 46% and Klebsella were 9% [30]. Results in other studies 746 ascitic fluid culture 1992 *E. coli* 47%, Streptococcus 19% and Klebsella 13% [30]. Conn et al. [30] reported *E. coli* was major pathogen of SBP in literature review. Severity of illness is more in nosocomial SBP compared with community acquired SBP [31].

Table 1. Descriptive statistics of the variables (N=100)

Variables	N	Minimum	Maximum	Mean
SGPT	100	27.00	1211.00	94.03
Bilirubin	100	.90	9.00	3.08
PT	100	18.00	62.00	32.24
Hb	100	5.60	12.00	8.62
L.count	100	12000.00	28000.00	18896.70
Urea	100	27.00	320.00	58.32
Creatinine	100	.80	16.00	2.09
Asc.protein	100	1.50	2.30	1.95
Asc.PMN	100	61.00	1472.00	492.59

5. CONCLUSION

SBP common complication of cirrhosis associated with high mortality. All cirrhotic Patients admitted in medical wards with history of abdominal pain, fever and Encephalopathy diagnostic paracentesis is compulsory. Early management with intravascular expansion with albumin and antibiotics with good prognosis mortality can be reduced. In spite of management death can occur due to acute on chronic liver failure. Primary and secondary prevention with antibiotics have proven to be effective.

CONSENT

As per international standard or university standard, patients' written consent has been collected and preserved by the author(s).

ETHICAL APPROVAL

As per international standard or university standard written ethical approval has been collected and preserved by the author(s).

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Fernandez J, Gustot T. Management of bacterial infections in cirrhosis. J Heptol. 2012;56:S1-S12.
2. Conn HO, Fessel JM. Spontaneous bacterial peritonitis in cirrhosis: Variations on a theme. Medicine. 1971;50(3):161-97.
3. Conn HO. Spontaneous peritonitis and bacteremia in Laennec's cirrhosis caused by enteric organisms. A relatively common but rarely recognized syndrome. Ann Intern Med. 1964;60:568-80.
4. Teltschik Z. Wiest R, Beisner J, Nuding S, Hofmann C Schoelmcrich J, et al. Intestinal bacterial translocation in cirrhotic rats is related compromised Paneth cell antimicrobial host defence. Hepatology. 2012;55(4):1154-63.
5. Wiest R, Krag A, Gerbes A, Spontaneous bacterial peritonitis: Recent guidelines and beyond Gut. 2012;61:297-310.
6. Schrier RW, Arroyo V, Bernardi M, et al. Peripheral arterial vasodilation hypothesis: A proposal for the initiation of renal sodium and water retension in cirrhosis. Hepatology. 1988;8;1151-7.
7. Lata J, Stiburck O, Kapocova M. Spontaneous bacterial peritonitis: A severe complication of cirrhosis. Word J Gastroenterol. 2009;15;5505-10.
8. Runyon BA. Spontaneous bacterial peritonitis associated with cardiac ascites. Am J Gastroenterol. 1984;79:796.
9. Strauss E, Costa MF. The importance of bacterial infection as precipitating factor of chronic hepatic encephalopathy. Hepato-Gastroenterology. 1988;45:900-4.
10. Mumtaz K, Ahmed US, Abid S, Baig N, Hamid S, Jafri W. Preciptating factors and the outcome of hepatic encephalopathy. Hepatogastroenterology. J Coll Physicians Surg Pak. 2010;20:514-518.
11. Runyon BA. Ascites and spontaneous bacterial peritonitis. In: Feidman M, Friedman LS,Sliesenger MH. eds, Sliesenger and fordans gastrointestinal and liver disease 8th Philedelphia, Sunder. 2006;1935-64.
12. Gracia Tsao, Bacterial infection in cirrhosis treatment and prophylaxis. J Hepatology. 2005;42:585–92.
13. Arroyo V, Bataller R, Gines P. Spontaneous bacterial peritonitis In O grady IG Lake JR. Howdle PD eds. Comprehensive clinical Hepatology 1st Ed. Barcelona: Mosoby. 2009;153-169.
14. Gao B, Jeong WI, Tian Z. Liver an organ with predominant innate immunity. Hepatology. 2008;47(2):729-736.
15. De Franchis R, Faculty BVI, Baveno VI. Faculty Expanding in portal hypertension: Report of Baveno VI Consensus workshop: Stratifying risk and individualizing care for portal hypertension. J Heptol. 2015;63(3):743-752.
16. Piano S, Singh V, Garaceni P, et al. Adherence to EASL antibiotic treatment recommendations improves the outcomes of patients with cirrhosis and bacterial infections. Results from the ICA Global Study. 2018;68(Supplement 1):S46-S4.
17. European association for the study of the liver. EASL clinical practice guidelines on the management of ascites. Spontaneous bacterial peritonitis, and hepatorenal syndrome in cirrhosis. J Heptol. 2010; 53(3):397-417.
18. Memon Memon AQ, Khaskheli A. Spontaneous bacterial peritonitis in cirrhosis with ascites- An experience at

PMCH Nawabshah. Med Channel. 1999; 5(1):31-4.
19. Wiest R, Krag A, Gerbes A. Spontaneous Bacterial Peritonitis. Recent guidelines and beyond Gut. 2012;61(2):297-310.
20. European Association for the study of the Liver Electronic address: easloffice@ easloffice.eu, EASL clinical practice guidelines for the management of patients with decompensated cirrhosis. J Heptol. 2018;69(2):406-460.
21. Runyon BA. Low protein concentration in ascetic fluid predisposed to Spontaneous Bacterial Peritonitis. Gastroenterol. 1986; 91:1343-1346.
22. Runyan BA. Van epp De Diuresis of chirotic ascites increases its opsonic activity and may help prevent spontaneous bacterial peritonitis. Hepatology. 1986;92: 396-9.
23. Bauer TM, Steinbruckner B, Brinkman F, et al. Small intestinal bacterial overgrowth in patients with cirrhosis: Prevalence relation with spontaneous bacterial peritonitis. Am J Gastroenterol. 2001;96: 2962-7.
24. Baur TM, Steinbruckner B, Brinkman F, et al. Small intestinal bacterial overgrowth in patients with cirrhosis: Prevalence relation with spontaneous bacterial peritonitis. Am J Gastroenterol. 2001;96:2962-7.
25. Runyon BA. Squier S, Borzio M. Translocation of gut bacteria in rats with cirrhosis to mesenteric lymph nodes partially explains the pathogenesis of spontaneous bacterial peritonitis. J Heptol. 1994;21:792-6.
26. European Association for the study of the Liver. EASL clinical practice guidelines on the management of ascites, spontaneous bacterial peritonitis, and hepato renal syndrome in cirrhosis. J Heptol; 2010.
27. Alexopoulou A Papadopoulos N, Eliopoulos DG, Alexaki A, Toutouza M, Pectacides D. Increasing frequency of gram-positive cocci and gram-negative multidrug-resistant bacteria in spontaneous bacterial peritonitis. Liver Int. 2013;33(7):975-81.
28. Bhuva M, GangerD, Jensen D. Spontaneous bacterial peritonitis an update on evaluation, management and prevention. Am J Med. 1994;97:169-75.
29. Garcia-Tasao G. Spontaneous bacterial peritonitis. Gastroenterol Clin North Am. 1992;21:257-75.
30. Conn HO. Spontaneous bacterial peritonitis and bacteremia in Laennec's cirrhosis caused by enteric organisms. A relatively common but rarely recognized syndrome. Ann Intern Med. 1964;60:568-80.
31. Rimola A, Gracia-Tsao G, Navasa M, et al. Diagnosis, treatment and prophylaxis of spontaneous bacterial peritonitis: a consensus document. J Heptol 2000; 32:142-53. 2018;68(Supplement 1):S46-S4.

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