



SPONTANEOUS BACTERIAL PERITONITIS; PATIENTS OF LIVER CIRRHOSIS WITH LOW ASCITIC PROTEIN CONTENTS.

1. MBBS, FCPS
Senior Registrar
DHQ Hospital, Faisalabad.
2. MBBS, FCPS
Associate Professor
DHQ Hospital, Faisalabad.
3. MBBS
Medical Officer
Basic Health Unit.

Correspondence Address:

Dr. Noor Gul
Senior Registrar,
Medical Unit-V
DHQ Hospital Faisalabad.
naveed72020@gmail.com

Article received on:

23/08/2017

Accepted for publication:

15/12/2017

Received after proof reading:

31/01/2018

Noor Gul¹, Tahir Habib Rizvi², Memoona Alam³

ABSTRACT... Objectives: To record frequency of spontaneous bacterial peritonitis in patients of liver cirrhosis with low ascitic protein contents. **Methodology:** This study included 81 patients with liver cirrhosis with low ascitic proteins level. All the patients were evaluated for the presence of spontaneous bacterial peritonitis which was described as frequency distribution table. **Study Design:** Cross Sectional Study. **Setting:** Medical wards of DHQ and Allied Hospitals (Punjab Medical College) Faisalabad. **Duration of Study:** 11th May 2011 to 10th November 2011. **Results:** Spontaneous bacterial peritonitis was present among 29 (35.8%) patients and was not present among 52 (64.2%) patients. **Conclusion:** All the patients with low ascitic protein level should be evaluated for the presence of spontaneous bacterial peritonitis and antibiotic prophylaxis should be considered.

Key words: Live Cirrhosis; Ascitic Protein Level; Spontaneous Bacterial Peritonitis.

Article Citation: Gul N, Rizvi TH, Alam M. Spontaneous bacterial peritonitis; Patients of liver cirrhosis with low ascitic protein contents. Professional Med J 2018; 25(2):302-306. DOI:10.29309/TPMJ/18.4271

INTRODUCTION

Cirrhosis is the result of hepatocellular injury that leads to both fibrosis and nodular regeneration throughout the liver and is the twelfth leading cause of death in United States.¹ It was the 12th leading cause of death in United States in 2006.²

Ascites is the most common complication of cirrhosis, and > 60% of patients with compensated cirrhosis develops ascites within 10 years during the course of their disease. Ascites only occurs when portal hypertension has developed and is primarily related to an inability to excrete an adequate amount of sodium into urine, leading to a positive sodium balance.³

Patients with cirrhosis and ascites are more susceptible to bacterial infections, of which spontaneous bacterial peritonitis (SBP) is the most frequent and potentially life threatening. It has typically been described in hospitalized patients with cirrhotic ascites, with 7% to 27% of patients with cirrhotic ascites showing evidence of occult peritoneal fluid infection at the time of hospital admission.⁴ One-third of patients with

infected peritoneal fluid lack any overt signs or symptoms such as fever or abdominal pain at the time of initial presentation.⁵ SBP occurs in the absence of an apparent intra-abdominal source of infection.⁶ Spontaneous bacterial peritonitis is a serious infection that occurs frequently in patients with advanced cirrhosis. This condition has been defined as the infection of a previously sterile ascitic fluid, without any apparent intra-abdominal source of infection. Diagnosis of spontaneous bacterial peritonitis is based on ascitic fluid neutrophil count of $\geq 250/\text{cmm}$ (and/or leucocyte count of $\geq 500/\text{cmm}$).⁷ In the past, it was associated with a high mortality rate.⁸ Prevalence of spontaneous bacterial peritonitis is as high as 18% in cirrhotic patients with ascites and mortality associated with the complication ranges from 40-70%. During 1970s, mortality in patients hospitalized for spontaneous bacterial peritonitis was 80-90%.⁹

The common causative organisms of SBP include E. Coli (60%), Klebsiella (20%), proteus mirabilis and streptococcus (10%).¹⁰ The opsonin activity of ascitic fluid correlates with the concentration

of immunoglobulins, complement, fibronectin and total proteins in the ascitic fluid. Patients with a reduced total protein in the ascitic fluid are prone to the development of SBP.¹¹ In a lot of cases, SBP remains asymptomatic, common signs include sub febrile states, diffuse abdominal pain. They are frequently manifested only by the occurrence or deepening of symptoms that accompany the course of liver cirrhosis.

The prevalence of SBP was 5% to 10% in cirrhotic patients with ascites, but with newer diagnostic criteria and improved culture techniques Fernandez et al., (2002) have estimated a prevalence of 10% to 30% in cirrhotic patients with ascites admitted to hospitals.¹²

The prevalence of SBP is much lower (3.5%) in asymptomatic outpatients undergoing therapeutic paracentesis and its outcome seems to be better than SBP occurring in hospitalized patients.¹³

'Spontaneous bacterial peritonitis has a recurrence rate of 70% in 1 year. In addition, this infection determines a poor short and long-term prognosis.¹⁴

The SBP mortality rate ranges from 40-70% in adult patients with cirrhosis and is lower in children. Patients with concurrent renal insufficiency have been shown to be at a higher risk of mortality from SBP than those without concurrent renal insufficiency.¹⁵

Since SBP is associated with a relatively high in-hospital mortality rate (20 – 40%), prophylactic measures should be taken to prevent this infection.

The reason to conduct this study was to identify the frequency of SBP in patients of liver cirrhosis with low ascitic fluid protein so as to start prophylactic treatment for SBP and help to reduce the episodes of SBP.

MATERIAL AND METHODS

Study Design

Cross-sectional study

Setting

Medical wards of DHQ and Allied Hospital (Punjab Medical College) Faisalabad.

Sample Size

81 patients were evaluated with the prevalence 30 % (p), absolute precision (d) 10% and confidence level 95% (by using the WHO sample size calculator).

Duration & Dates

Six months from June 2006 to November 2006.

We included all cases of both sexes of any age admitted in medical wards through OPD and emergency having already diagnosed liver cirrhosis with low ascitic protein contents. We excluded all patients with prophylactic treatment for spontaneous bacterial peritonitis, secondary peritonitis or malignancy, and severe concomitant illness or immuno-compromised state. After approval by the ethical committee and Review Board Allied and DHQ hospitals Faisalabad, all consecutive subjects presenting in the medical ward with liver cirrhosis already diagnosed on ultrasound abdomen with ascites, were enrolled. An informed consent was obtained from the patient. After taking history and physical examination, baseline investigations i.e. complete blood picture, serum electrolytes, liver function tests and urine complete examination were sent to the pathology laboratory and were reported by the pathologist. Ultrasound abdomen was done by the radiologist. Peritoneal paracentesis was done by the researcher himself and ascitic fluid were checked for TLC, DLC and gram staining by the pathologist. Ascitic fluid was also checked for total proteins, total albumin, LDH and glucose. Serum proteins and albumin were also measured. The diagnosis of spontaneous bacterial peritonitis was made on basis of ascitic fluid polymorphonuclear cells (PMNS) > 250 cells/ UL.

RESULTS

The mean age of the patients was 36.61±12.19 years [range 15 - 65 years]. There was 1 (1.2%) patient of age < 15 years. There were 19 (23.5%) patients of age range of 21 - 30 years, 22 (27.2%) patients of age range of 31 – 40 years, 16 (19.7%)

patients of age range of 41 – 50 year, 18 (22.2%) patients of age range of 51 – 60 years and 5 (6.2%) patients of age > 60 years. (Table-I)

Age (Years)	No.	Percentage
< 20	01	1.2
21 – 30	19	23.5
31 - 40	22	27.2
41 – 50	16	19.7
51 – 60	18	22.2
> 60	5	6.2
Total	81	100

Table-I

Patients were also distributed according to sex. There were 32 (39.5%) female patients in the study, while 49 (60.5%) patients were male. Male to female ration was 1.53:1. (Figure-1)



Figure-1. Gender distribution

Distribution of patients by serum billurubin level:

The mean serum billirubin level was 4.28 + 1.38 mg/dL. (Table-II)

Laboratory finding	Mean	SD
Serum Billurubin level (mg/dL)	4.28	1.38

Table-II

Distribution of patients by presence of spontaneous bacterial peritonitis:

Spontaneous bacterial peritonitis was present among 29 (35.8%) patients and was not present among 52 (64.2%) patients. (Figure-2)

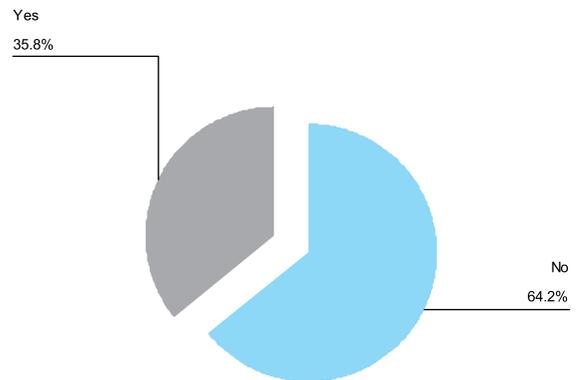


Figure-2. Distribution of patients by presence of spontaneous bacterial peritonitis (n = 81)

Distribution of patients by cause of hepatitis infection:

Among the 81 patients included in our study, Hepatitis B virus infection was present among 31 (38.3%) patients, Hepatitis C virus C infection was present among 46 (56.8%) patients and others were 4 (4.1%) patients. Of the four patients in others group, two patients had autoimmune hepatitis and the other two had hepatitis E virus infection. (Figure-3)

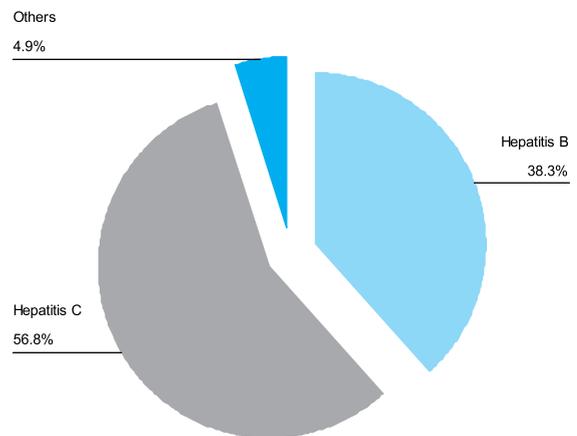


Figure-3. Distribution of patients by cause of hepatitis infection (n = 81)

DISCUSSION

The liver is the most important site of synthesis of the serum proteins. The plasma proteins produced by the hepatocyte are synthesized on polyribosomes bound to the rough endoplasmic reticulum, from which they are discharged into

plasma. The hepatocyte makes up albumin, fibrinogen, alpha1antitrypsin, heptaglobin, ceruloplasmin, transferrin and prothrombin. Fall in concentration usually reflects decreased hepatic synthesis.

SBP is a severe complication in cirrhotic patients with ascites. Early diagnosis and treatment are critical for improving prognosis. Not all patients with cirrhosis are equally susceptible to the development of SBP. However, patients with low ascetic fluid protein level may be prone to develop SBP. The results of this study which was conducted among 81 patients with liver cirrhosis, SBP was present among 35.8% patients which is quite a high frequency.

There are some other studies done in this regard to determine the frequency of SBP among patients with low ascetic proteins count. However, the results vary among different authors.

Runyon BA,¹⁶ conducted a study among 107 patients with low ascetic fluid count. The ascitic fluid protein concentration in the spontaneous peritonitis group (0.72 +/- 0.53 g/dl) was significantly lower ($p < 0.001$) than that in the group of patients with sterile portal hypertension-related ascites (1.36 +/- 0.89 g/dl). Of the patients whose initial sterile ascitic fluid protein concentration was less than or equal to 1.0 g/dl, 7 of 47 (15%) developed spontaneous peritonitis during their hospitalization; whereas only 1 of 65 (1.5%) patients who had an initial sterile ascitic fluid protein concentration greater than 1.0 g/dl developed spontaneous peritonitis. This difference in risk of development of peritonitis in relation to initial ascitic fluid protein concentration was also significant (p less than 0.01). They concluded that low-protein-concentration ascitic fluid predisposes to spontaneous bacterial peritonitis. The results of this study further validate our results that frequency of SBP was high among patients with low ascetic proteins contents.

In another study by Mostafa MG, et al.¹⁷ 29 patients with liver cirrhosis were enrolled. The patients were divided in two group; 14 patients who developed SBP and 15 patients with non SBP.

It was observed that mean ascitic fluid protein was 1.1 ± 0.3 g/dL in the cases of spontaneous bacterial peritonitis and 1.5 ± 0.5 g/dL in the nonspontaneous bacterial peritonitis group. The difference of findings between the two groups was highly significant ($p=0.008$). The protein level in the ascitic fluid of patients who developed spontaneous bacterial peritonitis (1.1 ± 0.3 g/dL) was significantly lower than the protein level in the ascitic fluid of those who did not develop spontaneous bacterial peritonitis (1.5 ± 0.5 g/dL).

In our study, the male dominated female population. There were 39.5% female patients and 60.5% patients were male. However, in study by Nouman S, et al.¹⁸ 44% patients were male and 56% patients were female.

In our study, the Hepatitis C virus 56.8 % infection was the most common cause of liver cirrhosis, followed by hepatitis B (38.3%) virus infection. Nouman S, et al.¹⁹ documented that hepatitis C virus infection was present among 65% patients and hepatitis B virus infection was present among 35% patients. This study has some limitation being non-blinded study.

CONCLUSION

Spontaneous bacterial peritonitis is frequently present among patients with liver cirrhosis with low ascitic proteins contents. So, it is recommended that every patient with liver cirrhosis with low protein count should be evaluated for the presence of SBP and prompt antibiotic prophylaxis should be started.

Copyright© 15 Dec, 2017.

REFERENCES

1. McPhee SJ, Papdakis MA. Cirrhosis. In: Lawrence M. Tierney, Jr, editor. **Current medical diagnosis and treatment**. New York: McGraw-Hill 2008; 584-588.
2. Heron M, Hoyert DL, Murphy SL, XuJ, Kochaneck KD, Tejada-Vera B. **Death: final data for 2006**: Natl Vital Stat Rep 2009;57:1-134.
3. Ripoll C, Groszmann R, Garcia-Tsao G. **Hepatic venous gradient predicts clinical decompensation in patients with compensated cirrhosis**. Gastroenterology 2007; 133:481-488.

4. Garcia-Tsao G. **Bacterial infections in cirrhosis: treatment and prophylaxis.** J Hepatol 2005; 42:585-592.
5. Runyon BA. **Management of adult patients with ascites due to cirrhosis: an update.** Hepatology 2009; 49:2087-2107.
6. Mcquaid KR. Gastrointestinal disorder. Inc: MCPhee SJ, Papadakis MA, editors. **Current medical diagnosis and treatment.** 49th ed. New York: Mc Graw Hill 2010; 526-527.
7. Thomas D, Boyer TD. **Diagnosis and management of cirrhotic ascites.** In: **Hepatology: A textbook of liver disease.** 4th ed. Philadelphia, Saunders 2003; 639.
8. Bass NM. **Intravenous albumin for spontaneous bacterial peritonitis in patients with cirrhosis.** New Engl J Med 1999; 341:443-444.
9. Sheer AT, Runyon AB. **Spontaneous bacterial peritonitis.** Digestive diseases 2005; 23:39-46.
10. Imran M, Hashmi SN, Altaf A, Rashid H, Hussain T. **Spontaneous bacterial peritonitis.** Professional Med J 2006; 13:201-205.
11. Ribeiro TCR, Chebli JMF, Konodo M, Gaburri PD, Chebil LA, Feldner ACA. **Spontaneous bacterial peritonitis; how to deal with this life-threatening cirrhosi complication?** Ther Clin Risk Manag 2008; 4:919-925.
12. Fernandez J, Navasa M, Gomez J. **Bacterial infections in cirrhosis: epidemiological changes with invasive procedures and norfloxacin prophylaxis.** Hepatology 2002; 35:140-148.
13. Evan LT, Kim WR, Poterucha JJ. **Spontaneous bacterial peritonitis in asymptomatic outpatients with cirrhotic ascites.** Hepatology 2003; 37:897-901.
14. Coral G, Mattos A, Valiatti F. **Bacterial infections in cirrhotic patients.** J. Hepatol 2002; 36:709.
15. Bandy SM. **Spontaneous bacterial peritonitis.** Emergency Medicine, Clinical Infectious Diseases 2005.
16. Runyon BA. **Low-protein-concentration ascitic fluid is predisposed to spontaneous bacterial peritonitis.** Gastroenterology 1986; 91:1343-1346.
17. Mustafa MG, Mamun MAA, Alam AKMAA. **Study on ascitic fluid protein level in cirrhotic patients with spontaneous bacterial peritonitis.** Bangladesh Med Res Counc Bull 2009; 35:41-43.
18. Zhang S, Ren W, Zhou K, Wang J, Zhu W. **The effect of prokinetic drug on small intestinal bacterial overgrowth and endotoxemia in cirrhosis.** J Gastroent Hepatol 2002; 17 Suppl: A12.
19. Nouman S, Hussain A, Hussain M, Ahmed M. **Frequency of Spontaneous Bacterial Peritonitis in Chronic Liver Disease.** ANNALS 2010; 16112-115.

AUTHORSHIP AND CONTRIBUTION DECLARATION

Sr. #	Author-s Full Name	Contribution to the paper	Author=s Signature
1	Noor Gul	Data collection	
2	Tahir Habib Rizvi	Topic selection, Statistical collection	
3	Memoona Alam	Article writing, Data collection	