



Spontaneous Bacterial Peritonitis as a Precipitating Factor of Hepatic Encephalopathy and Its Outcomes During Hospital Admission

Muntazir Mahdi, Maqsood Ahmad

Medical Unit IV, Allied Hospital-II/ District Headquarters Hospital, Faisalabad, Pakistan

ARTICLE INFO

Keywords

Hepatic Encephalopathy, Spontaneous Bacterial Peritonitis, Outcome.

Corresponding Author: Muntazir Mahdi, Medical Unit IV, Allied Hospital-II/ District Headquarters Hospital, Faisalabad, Pakistan.
Email: muntazir.khan5@gmail.com

Declaration

Authors' Contribution: All authors equally contributed to the study and approved the final manuscript.

Conflict of Interest: No conflict of interest.

Funding: No funding received by the authors.

Article History

Received: 13-02-2025, Revised: 01-03-2025

Accepted: 17-03-2025, Published: 24-03-2025

ABSTRACT

Introduction: Hepatic encephalopathy (HE) is a well-recognized clinical complication of liver cirrhosis and the presence and prompt identification of well-defined precipitating factors are extremely important in the diagnosis and treatment of this fatal condition. HE develops in 50% to 70% of patients with cirrhosis, and its occurrence is a poor prognostic indicator. **Objectives:** To determine the outcome (in terms of complete reversal of HE) of hepatic encephalopathy patients with Spontaneous bacterial peritonitis (SBP). **Materials & Methods:** 126 patients of hepatic encephalopathy and ascites with spontaneous bacterial peritonitis of age 20-60 years of either gender were enrolled. Patients with fulminant hepatic failure, non-cirrhotic portal hypertension, uremic, anoxic, cerebral and metabolic encephalopathy were excluded. All patients underwent standard management during their hospital stay. All the patients were assessed with these criteria and on daily basis for noting an improvement/deterioration in HE grades. Improvement of HE was defined as complete reversal of clinical symptoms on the basis of the West Haven criteria. Patients with HE of \geq grade 1 on discharge was considered as no improvement. **Results:** The study's age range was 20 to 60 years old, with a mean age of 42.15 ± 9.87 years. Majority of patients i.e. 62.70%, were between 41-60 years of age. Of these 126 patients, 65 (51.59%) were males and 61 (58.41%) were females with male to female ratio of 1.1:1. Mean duration of cirrhosis was 3.51 ± 1.16 years. Improved outcome (in terms of complete reversal of HE) of hepatic encephalopathy patients with Spontaneous bacterial peritonitis (SBP) was found in 111 (88.10%) patients. **Conclusion:** According to the study's findings, successful clinical outcomes can result from the early detection of hepatic encephalopathy's precipitant variables and subsequent care.

INTRODUCTION

Hepatic encephalopathy (HE) is a well-recognized clinical complication of liver cirrhosis and the presence and prompt identification of well-defined precipitating factors are extremely important in the diagnosis and treatment of this fatal condition. HE develops in 50% to 70% of patients with cirrhosis, and its occurrence is a poor prognostic indicator¹. Precipitating factors are represented mainly by infections, gastrointestinal bleeding, constipation, electrolyte disorders, renal dysfunction, hepatotoxic agents, and others². The most frequent and most severe one is spontaneous bacterial peritonitis (SBP). Prevalence of SBP in cirrhotic patients is as high as 18%, with 40–70% associated mortality³. Patient outcomes and survival depends on clinical presentation, identification of the precipitating factor, early management and treatment of complications^{4,5}.

Mumtaz et al conducted a cross-sectional study to determine precipitants of factors and the outcome of hepatic encephalopathy (HE) in liver cirrhosis patients.

The most common precipitant of HE was spontaneous bacterial peritonitis in 83 (20.5%), constipation in 74 (18.3%) and urinary tract infection in 62 (15.3%). Mean hospital stay was 4 ± 3 days. Complete reversal of HE was noted in 366 patients (91%) while the remaining had grade 1 HE on discharge. Nine (2.2%) patients died during the hospital stay. No mortality was noted in patients without precipitants⁶.

Early identification of precipitant factors of hepatic encephalopathy and its management can lead to good clinical outcomes. So, this research is being undertaken to study the clinical presentation of hepatic encephalopathy in patients with liver cirrhosis Spontaneous bacterial peritonitis (SBP) and to determine their clinical outcomes. The results will to formulate guidelines to treat hepatic encephalopathy patients with Spontaneous bacterial peritonitis that will help to reduce morbidity and mortality.

MATERIALS AND METHODS

After taking approval from hospital ethical committee,

this descriptive study was done from 13th August 2024 to 12th February 2024 at Department of Medicine, Allied Hospital, Faisalabad. Patients of hepatic encephalopathy and ascites with spontaneous bacterial peritonitis of age 20-60 years of either gender were enrolled through non-probability, consecutive sampling technique and informed consent was taken. The WHO single proportion calculator yields a sample size of 126 with a 95% confidence level, a 5% margin of error, and taking Outcome in HE (complete reversal of HE) as 91%⁶. Patients with fulminant hepatic failure, non-cirrhotic portal hypertension, uremic, anoxic, cerebral and metabolic encephalopathy were excluded. The diagnosis of acute HE being made on the basis of a detailed history, physical examination and West Haven criteria whereas Child Pugh Class were labeled according to Child Pugh Grading System. An ascitic tap was performed in all patients with ascites and sent for detailed report and culture in order to diagnose spontaneous bacterial peritonitis. All patients underwent standard management during their hospital stay. All the patients were assessed with these criteria and on daily basis for noting an improvement/deterioration in HE grades. Improvement of HE was defined as complete reversal of clinical symptoms on the basis of the West Haven criteria. Patients with HE of \geq grade 1 on discharge was considered as no improvement. All the data was recorded on specially designed pro-forma.

Data was analyzed using SPSS version 25.0. Quantitative variables like age and duration of liver cirrhosis were presented as mean \pm standard deviation. Categorical variables like gender, HE grades, Child Pugh class, SBP and outcome were presented in terms of frequencies and percentages. Chi square test was applied. Effect modifiers such as age, gender, duration of liver cirrhosis, child Pugh class were controlled by stratification. Post stratification chi-square test was applied to determine their effect on outcome and P value less than or equal to 0.05 was labeled as significant.

RESULTS

The study's age range was 20 to 60 years old, with a mean age of 42.15 ± 9.87 years. Majority of patients i.e. 62.70%, were between 41-60 years of age. Of these 126 patients, 65 (51.59%) were males and 61 (58.41%) were females with male to female ratio of 1.1:1. Mean duration of cirrhosis was 3.51 ± 1.16 years. Distribution of patients with variables is shown in Table I. Improved outcome (in terms of complete reversal of HE) of hepatic encephalopathy patients with Spontaneous bacterial peritonitis (SBP) was found in 111 (88.10%) patients (Figure I). Stratification of outcome with respect to age, gender, duration of liver cirrhosis and child Pugh class is shown in Table II.

Table 1

Distribution of patients with variables (n=126)

Variables	Frequency	%age
Age (years)	20-40	47
	41-60	79
Gender	Male	65
	Female	61
Duration of Cirrhosis (years)	≤ 3	68
	> 3	58
HE grade	1	10
	2	38
	3	55
	4	23
Child Pugh Class	A	21
	B	48
	C	57

Figure 1: Outcome (in terms of complete reversal of HE) of hepatic encephalopathy patients with Spontaneous bacterial peritonitis (SBP) (n=126).

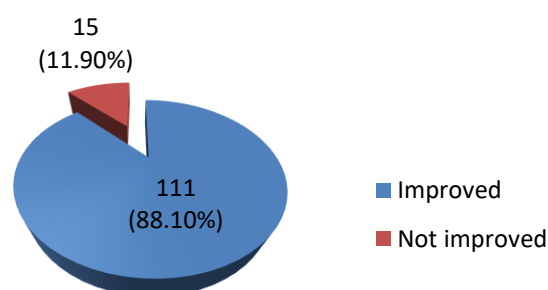
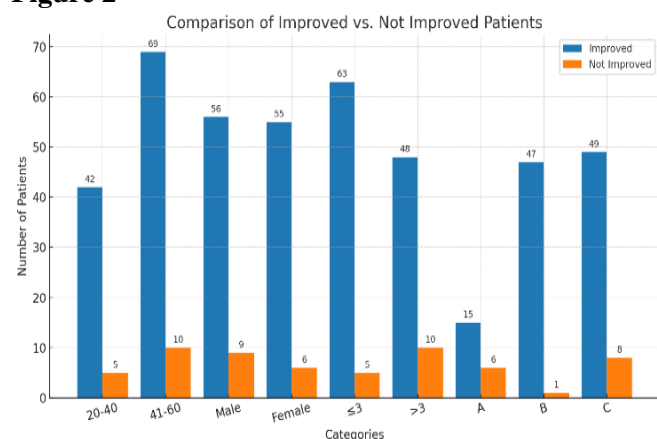


Table 2

Stratification of outcome with respect to age, gender, duration of liver cirrhosis and child Pugh class.

Variables	Improved (n=111)	Not improved (n=15)	P-value
Age (years)	20-40 42 (89.36%)	05 (10.64%)	0.735
	41-60 69 (87.34%)	10 (12.66%)	
Gender	Male 56 (86.15%)	09 (13.85%)	0.487
	Female 55 (90.16%)	06 (9.84%)	
Duration of Cirrhosis (years)	≤ 3 63 (92.65%)	05 (7.35%)	0.088
	> 3 48 (82.76%)	10 (17.24%)	
Child Pugh Class	A 15 (71.43%)	06 (28.57%)	0.006
	B 47 (97.92%)	01 (2.08%)	
	C 49 (85.96%)	08 (14.04%)	

Figure 2

DISCUSSION

For individuals with cirrhosis who have ascites, spontaneous bacterial peritonitis (SBP) is a serious consequence. Reducing mortality and morbidity in this patient group requires clinical awareness, early identification by ruling out subsequent bacterial peritonitis, and rapid treatment.⁷ Nevertheless, the advent of multidrug-resistant (MDR) microbes has altered our knowledge of the bacteriology and management of SBP. The best treatment for either nosocomial/healthcare-acquired or community-acquired SBP is antibiotic therapy, however liver transplantation is still the only option after SBP.⁸

Therefore, it is a crucial clinical concern to prevent SBP recurrence by administering antibiotic prophylaxis to patients awaiting liver transplantation. Further prospective research is necessary to determine whether the poorly absorbed antibiotic rifaximin is useful for primary and secondary SBP prophylaxis. It is also critically necessary to continue developing non-antibiotic tactics based on pathogenic processes.⁹ Future research should focus on blind trials that eliminate post-randomization dropout and take into account clinically important outcomes like mortality, health-related quality of life, and decompensation occurrences.¹⁰

SBP comes in three varieties. The most frequent cause of SBP is bacterial translocation from the GI tract. Thus, Gram-negative bacilli, nearly entirely Enterobacteriaceae, were responsible for two-thirds of SBP cases. The most often isolated pathogen is *Escherichia coli*, or *E. coli*.¹¹ Gram-positive cocci (GPC), including *Staphylococcus* and *Enterococcus*, as well as multi-resistant bacteria, have become common pathogens and have altered the traditional approach to treating SBP. However, a trend of GPC-associated SBP has been observed in recent years, representing a

changing paradigm in the known bacteriology of SBP, especially nosocomial SBP. Other sources, such as transient bacteremia due to invasive procedures, can also result in SBP.¹²

Increased awareness and consideration of alternate antibiotic coverage should be prompted by nosocomial and healthcare-associated SBP infections. In people who are at risk, beta-adrenergic antagonist and acid suppressive medications are closely linked to SBP.¹² Every patient with cirrhosis and ascites who needs hospitalization or emergency room care, who exhibits or reports the signs and symptoms listed above in the clinical presentations, or who has gastrointestinal bleeding should have a diagnostic paracentesis to confirm evidence of SBP.¹³ Because secondary bacterial peritonitis and SBP require different treatment approaches, it is crucial to distinguish between the two diseases. One-year overall death rates range from 53.9 to 78% since SBP may be considered the last clinical stage of liver cirrhosis for this condition.¹⁴

Therefore, SBP survivors who are suitable candidates for transplantation should give liver transplantation careful thought. Prompt broad-spectrum antibiotic therapy is the conventional treatment for SBP, and it should be customized based on hospital-acquired, CAP, or local resistance profiles.¹⁵ Supplementing with albumin is also advantageous, particularly for patients with renal impairment (RI). Selective intestinal decontamination (SID), also known as antibiotic prophylaxis, is not necessary for all individuals with cirrhosis and ascites. SID is linked to a lower risk of mortality and bacterial infection.¹⁶

Therefore, it is essential to identify precipitating causes early on, particularly in patients with severe liver disease, and to start appropriate treatment as soon as possible in order to treat and control them and, ultimately, ensure better results. In order to guarantee early presentation, diagnosis, and treatment of acute HE, it is also critical to inform patients and their families about these precipitants.

CONCLUSION

According to the study's findings, successful clinical outcomes can result from the early detection of hepatic encephalopathy's precipitant variables and subsequent care. The encephalopathy also usually goes away once the triggering disease is treated, and the patient returns to their pre-distress state. Poorer outcomes were linked to patients who had at least two triggering events and admission HE grades of 3 or 4.

REFERENCES

1. Poudyal NS, Chaudhary S, Sudhamshu KC, Paudel BN, Basnet BK, Mandal A, et al. Precipitating factors and treatment outcomes of hepatic encephalopathy in liver cirrhosis. *Cureus*. 2019; 11(4): 1-9. <https://doi.org/10.7759/cureus.4363>
2. Singeap AM, Cuciureanu T, Zenovia S, Girleanu I, Huiban L, Muzica CM, et al. Spectrum of Precipitating factors of Hepatic Encephalopathy in Patients with Liver Cirrhosis. *Med Surg J*. 2021; 125(4):492-501. <https://doi.org/10.22551/msj.2021.04.04>
3. Kamal SM, Abdelhakam SM, Massoud YM, Abd El Hafeez KA, Kamal HA. Clinical profile of patients with ascitic fluid infection at ain shams university hospitals. *Egypt J Hosp Med*. 2018; 72 (9): 5241-50. <https://doi.org/10.21608/ejhm.2018.10862>
4. Hadjihambi A, Arias N, Sheikh M, Jalan R. Hepatic encephalopathy: a critical current review. *Hepatology int*. 2018; 12(1): 135-47 <https://doi.org/10.1007/s12072-017-9812-3>
5. Ullah T, Iqbal M, Ali S, Ullah I. Precipitating Factors of Hepatic Encephalopathy in Patients With Liver Cirrhosis. *Med Forum*. 2020; 31(4). 54-57.
6. Mumtaz K, Ahmed US, Abid S, Baig N, Hamid S, Jafri W. Precipitating factors and the outcome of hepatic encephalopathy in liver cirrhosis. *J Coll Physician & Surgeons Pak*. 2010; 20(8):514-18.
7. Moreau R, Elkrief L, Bureau C. Effects of long-term norfloxacin therapy in patients with advanced cirrhosis. *Gastroenterology*. 2018 Dec; 155(6):1816–1827. <https://doi.org/10.1053/j.gastro.2018.08.026>
8. Tsung PC, Ryu SH, Cha IH. Predictive factors that influence the survival rates in liver cirrhosis patients with spontaneous bacterial peritonitis. *Clin Mol Hepatol*. 2013; 19(2):131–139. <https://doi.org/10.3350/cmh.2013.19.2.131>
9. Bal CK, Daman R, Bhatia V. Predictors of fifty days in-hospital mortality in decompensated cirrhosis patients with spontaneous bacterial peritonitis. *World J Hepatol*. 2016; 8(12):566–572. <https://doi.org/10.4254/wjh.v8.i12.566>
10. Cheong HS, Kang CI, Lee JA. Clinical significance and outcome of nosocomial acquisition of spontaneous bacterial peritonitis in patients with liver cirrhosis. *Clin Infect Dis*. 2009; 48(9):1230–1236. <https://doi.org/10.1086/597585>
11. D'Amico G, Garcia-Tsao G, Pagliaro L. Natural history and prognostic indicators of survival in cirrhosis: a systematic review of 118 studies. *J Hepatol*. 2006; 44(1):217–231. <https://doi.org/10.1016/j.jhep.2005.10.013>
12. Marciano S, Dirchwolf M, Bermudez CS. Spontaneous bacteremia and spontaneous bacterial peritonitis share similar prognosis in patients with cirrhosis: a cohort study. *Hepatol Int*. 2018; 12(2):181–190. <https://doi.org/10.1007/s12072-017-9837-7>
13. Ginés P, Rimola A, Planas R, et al. Norfloxacin prevents spontaneous bacterial peritonitis recurrence in cirrhosis: results of a double-blind, placebo-controlled trial. *Hepatology*. 1990; 12(4 Pt 1):716–724. <https://doi.org/10.1002/hep.1840120416>
14. Titó L, Rimola A, Ginès P, Llach J, Arroyo V, Rodés J. Recurrence of spontaneous bacterial peritonitis in cirrhosis: frequency and predictive factors. *Hepatology*. 1988; 8(1):27– 31. <https://doi.org/10.1002/hep.1840080107>
15. Fiore M, Maraolo AE, Gentile I. Nosocomial spontaneous bacterial peritonitis antibiotic treatment in the era of multi-drug resistance pathogens: a systematic review. *World J Gastroenterol*. 2017; 23(25): 4654– 4660. <https://doi.org/10.3748/wjg.v23.i25.4654>
16. Fernández J, Bert F, Nicolas-Chanoine MH. The challenges of multidrug-resistance in hepatology. *J Hepatol*. 2016; 65(5):1043–1054. <https://doi.org/10.1016/j.jhep.2016.08.006>