ORIGINAL ARTICLE

Retrospective Investigation of Factors Affecting Mortality in Spontaneous Bacterial Peritonitis

Tuğba Ocak[1], Macit Gülten[2]

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Abstract

Background: Spontaneous bacterial peritonitis (SBP) is an important reason for mortality in cirrhosis. This study aimed to identify the factors associated with mortality in SBP.

Materials and methods: A total of 69 patients with cirrhosis and 74 with SBP attacks that occurred in this group were assessed. Demographic data, symptoms at admission, comorbidities, laboratory parameters, treatment protocols, causes of cirrhosis, scoring characteristics, cirrhosis complications, and mortality were analyzed.

Results: Model for end-stage liver disease (MELD; $p = 0.001$), sodium-MELD ($p = 0.001$), and Child–Pugh–Turcotte (CTP) ($p < 0.001$) scores were correlated with mortality in patients with SBP episodes. Hepatorenal syndrome ($p = 0.001$) and esophageal variceal bleeding ($p < 0.001$) related to mortality. Serum lactate dehydrogenase (LDH) ($p = 0.007$), serum leukocyte ($p = 0.017$), and serum hemoglobin ($p = 0.010$) values had a statistically significant effect on mortality in multivariate regression analysis.

Discussion: The mortality rate can be reduced by identifying factors influencing death in patients with SBP episodes.

Keywords: Cirrhosis, Mortality, Peritonitis.

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Introduction

Ascites are a significant complication of liver cirrhosis and portal hypertension.[1] Approximately 10 years after the diagnosis of cirrhosis, ascites develop.[2] Ascites formation is a sign of poor prognosis in cirrhosis.[3] Although cirrhosis is a particularly prevalent cause of ascites, other significant causes include cancer, heart failure, tuberculosis, and similar conditions.[2,4,5]

Spontaneous bacterial peritonitis (SBP) is an issue with an increased death rate in patients with ascites.[6,7] Immune system disorders, intestinal dysmotility, microbiota alteration, higher permeability of the gut, and translocation of bacteria are risk factors for SBP.[8–11] Classic SBP is characterized by >250/mm^3 polymorphonuclear leukocytes (PMNL) in ascites and positive ascites culture.[12,13] In addition to the traditional SBP, three other types of SBP exist culture-negative neutrocytic acid (CNNA), monomicrobial bactericide (MMB), and polymicrobial bactericide (PMB).[14] Culture-negative neutrocytic acid is diagnosed with PMNL >250/mm^3 and negative acid fluid culture, MMB is diagnosed with PMNL <250/mm^3 and growth of a single microorganism in acid fluid culture, PMB is diagnosed with PMNL <250/mm^3 and growth of multiple microorganisms in acid fluid culture.[15]

Although the mortality rate of SBP was greater than 90% at the beginning, it decreased to 20% later on.[7] Our study focused on determining the variables related to mortality in SBP.

Materials and Methods

Seventy-four SBP episodes of 69 individuals with cirrhosis identified in the Division of Gastroenterology between 2013 and 2016 have been included in the study. Demographic characteristics, acid liquid laboratory characteristics [leukocytes, PMNL, total protein, albumin, lactate dehydrogenase (LDH), microorganisms grown], complete blood cell characteristics [leukocytes, neutrophils, hemoglobin (Hb), hematocrit (Hct), platelets], serum biochemistry characteristics [urea, creatinine, sodium (Na), potassium, calcium, aspartate aminotransferase, alanine aminotransferase, alkaline phosphatase, gamma-glutamyl transpeptidase, bilirubin, albumin, total protein, LDH], coagulation characteristic [international normalized ratio (INR)], positive acute phase reactants [sedimentation, procalcitonin, C-reactive protein (CRP)], scoring characteristics [model for end-stage liver disease (MELD), Na-MELD scores, Child–Pugh–Turcotte (CTP)], complications of cirrhosis (hepatorenal syndrome, esophageal variceal hemorrhage, hepatic encephalopathy, hepatocellular carcinoma), and treatment protocols in SBP were retrospectively analyzed.

Patients with infections other than SBP, ascites fluid due to causes other than portal hypertension, and patients under 18 years of age were excluded.

In our study, cefotaxime was used for empirical treatment in all patients. While a 25% decrease in the number of PMNLs in ascites at 48 hours of cefotaxime treatment was considered as antibiotic

[1]Department of Internal Medicine, Uludağ University Medical School, Bursa, Turkey

[2]Department of Gastroenterology, Uludağ University Medical School, Bursa, Turkey

Corresponding Author: Tuğba Ocak, Department of Internal Medicine, Uludağ University Medical School, Bursa, Turkey, Phone: +90 224 295 50 00, e-mail: tugba@uludag.edu.tr

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sensitive, absence of a 25% decrease was considered as antibiotic resistance.[16]

Statistical Analysis

The Shapiro–Wilk test was used to test whether the data had a normal distribution. Normal data distributions are presented as mean standard deviation. The median range (minimum to maximum) was used in descriptive statistics for non-normally distributed data. The frequency or proportion *n* (%) of categorical variables was provided. IBM SPSS Statistics 23.0 was used to conduct analyses with a significance level of 0.05 and a confidence level of 95%. SPSS 23.0 Windows statistical program was used for statistical evaluation of the findings. Mann–Whitney, Chi-square, and *t*-test were utilized to evaluate findings that did not have a normal distribution. To identify independent risk factors, a univariate logistic regression analysis was used. Consideration was given to including any univariate logistic regression variable with a *p*-value of 0.25 in the multivariate model. A *p*-value of 0.05 or less was regarded as statistically significant.

Results

The median age for all 69 patients was 63 (25–82) years. A total of 58.1% of the patients were male. Among 74 with SBP attacks, 24.3% were with classical SBP, 70.3% were CNNA, and 5.4% were MMB. Our study's total mortality rate in SBP patients was 37.8%. Five patients died during 18 classic SBP attacks and 23 patients died during 29 CNNA attacks. No statistically significant relationship was observed between the SBP subgroups and mortality ($p = 0.137$). The rate of mortality was 85.7% in community-acquired SBP and 14.3% in hospital-acquired SBP. The presenting symptoms were abdominal distension (37.8%), abdominal pain (33.7%), altered consciousness (14.9%), gastrointestinal bleeding (9.5%), and fever (4.1%). The causes of cirrhosis were hepatitis B virus (44.6%), cryptogenic (23%), hepatitis C virus (12.2%), alcohol (6.8%), autoimmune hepatitis (5.4%), nonalcoholic steatohepatitis (4.1%), Wilson's disease (1.3%), primary biliary cirrhosis (1.3%), and hemochromatosis (1.3%).

The relationship between laboratory values recorded for all patients with SBP episodes and mortality was evaluated. Complete blood cell characteristics [leukocytes ($p < 0.001$), neutrophils ($p < 0.001$), Hb ($p < 0.001$), Hct ($p = 0.001$)], serum biochemistry characteristics [urea ($p = 0.002$), creatinine ($p = 0.031$), total bilirubin ($p = 0,023$), direct bilirubin ($p = 0.014$), LDH ($p < 0.001$)], positive acute phase reactant [CRP ($p = 0.047$)], and ascites parameters [leukocytes ($p < 0.001$), PMNL ($p < 0.00$), LDH ($p = 0.024$)] were significantly associated with mortality. The laboratory values reported in patients with SBP episodes and their relationship with mortality are shown in Table 1. The relationship between MELD ($p = 0.001$), Na-MELD ($p = 0.001$), and CTP ($p < 0.001$) scores and mortality was statistically significant. Table 2 shows the relationship between scoring systems and mortality. Ascitic fluid cultures of all patients diagnosed with SBP were analyzed and growth was found in 29.7% of patients. The rate of growth was 37% in the alive patients and 17.9% in the dead ones. *Escherichia coli* was found in 54.5%, staphylococcus in 18%, enterococcus in 13.6%, streptococcus in 4.6%, and other microorganisms in 9.1% of the cases. There was no statistical significance between acid culture positivity ($p = 0.081$), antibiotic resistance ($p = 0.118$), and mortality. A statistically significant difference was found between mortality and infections other than SBP ($p = 0.004$). Esophageal variceal hemorrhage and hepatorenal syndrome were significantly related with mortality ($p = 0.001$ and 0.001, respectively) among the complications of cirrhosis. The association between complications of cirrhosis and mortality is shown in Table 3.

Serum leukocytes, serum neutrophils, serum Hb, serum total bilirubin, serum LDH, creatinine, INR, CRP, ascites leukocytes, and ascites PMNL with *p*-value of lesser than 0.25 were analyzed in multivariate regression model in univariate regression model evaluating the factors affecting mortality in cirrhotic patients diagnosed with SBP. In multivariate regression model, serum leukocytes ($p = 0.017$), serum Hb ($p = 0.010$), and serum LDH ($p = 0.007$) had statistically significant effects on mortality. Serum leukocytes (1-fold) and serum LDH (1.016-fold) posed a positive risk on mortality. Serum Hb posed a 0.197-fold negative risk on mortality. Logistic regression analysis between laboratory characteristics and mortality in SBP is shown in Table 4.

Discussion

This study showed that serum leukocytes and serum LDH positively and serum Hb negatively predicted mortality in multivariate regression analysis of laboratory characteristics in SBP. Laboratory characteristics of SBP patients are easily accessible in many centers without high economic costs. The mortality rate can be reduced by identifying patients in the high-risk category and determining variables that predict prognosis in the follow-up of this group of patients.

Serum leukocytes are the fighter cells of our defense system that protect the body against infections. Infections frequently result in an increase in the quantity of serum leukocytes. This investigation found that serum leukocyte counts statistically significantly affected mortality in cirrhotic patients with SBP. Bal et al.,[17] Popoiag et al.,[18] and Poca et al.[19] found that serum leukocyte count statistically significantly affected mortality, consistent with our results. Lactate dehydrogenase is an enzyme that contributes to the production of energy by converting lactate to pyruvate. It is found in the liver, heart, lungs, kidneys, muscles, and blood cells at high levels. Lactate dehydrogenase is a general indicator of tissue injury and a marker of inflammation.[20] When the relationship between LDH and infection is examined in the scientific literature, high LDH levels in COVID-19 pneumonia have been related to death.[21–23] In many studies, investigating the factors affecting mortality in patients with SBP serum LDH has not been evaluated. Our study found a statistically significant effect of serum LDH level on mortality.[17–19,24–27] Hemoglobin may consist of iron-carrying heme molecule and globin molecule which is a protein. Low Hb levels can be seen in chronic diseases and in cases of blood loss. According to a study by Gökdemir et al.,[26] Hb is a risk factor for mortality in SBP. In our study, multivariate analysis showed that the Hb value was a risk factor for mortality in SBP.

The CTP, MELD, and Na-MELD scoring systems use clinical and laboratory parameters to predict the prognosis of cirrhotic patients. Multiple studies have linked CTP scores,[19,25,26,28] MELD scores,[26,28–30] and Na-MELD scores[26,31–34] to mortality in patients with SBP. In this study, we determined whether CTP scoring, MELD scoring, and Na-MELD scoring have statistically significant effects on mortality in patients with SBP.

Several studies have investigated these complications' impact on SBP patients' mortality.[7,9,11,19,24–26,34] Tüzün et al.[25] and Gökdemir et al.[26] also showed the relationship between hepatorenal

Table 1: Laboratory characteristics of patients with spontaneous bacterial peritonitis and their relationship with mortality

	Survivors	*Deaths*	*p*
Hb (g/dL)	11.2 ± 1.6	9.8 ± 1.5	<**0.001**
Hct (%)	32.6 ± 4.8	29 ± 3.8	**0.001**
Serum leukocytes (K/µL)	7300 (1200–15600)	11700 (1610–43000)	<**0.001**
Serum neutrophils (K/µL)	5640 (540–11600)	9075 (770–38700)	<**0.001**
Platelets (K/µL)	112000 (10700–542000)	119500 (10300–859000)	0.551
INR	1.6 (0.8–2.8)	2 (1.7–5.8)	<**0.001**
Urea (mg/dl)	56 (15–180)	124 (17–259)	**0.002**
Creatinine (mg/dl)	1.1 (0.5–3.3)	1.7 (0.5–4.6)	**0.031**
AST (IU/L)	55 (11–541)	69 (16–432)	0.358
ALT (IU/L)	33 (6–248)	33 (6–261)	0.978
ALP (IU/L)	92 (41–877)	116 (48–1044)	0.138
GGT (IU/L)	36 (8–426)	62 (10–962)	0.099
LDH (IU/L)	216 (85–515)	397 (219–1383)	<**0.001**
T. Bilirubin (mg/dL)	3 (6–10)	4 (6–27)	**0.023**
D. Bilirubin (mg/dL)	1.6 (3–7.5)	2.5 (3–18)	**0.014**
Serum T. protein (g/dL)	5.6 (4.5–9.7)	5.3 (3.9–7.9)	0.161
Serum albumin (g/dL)	2.38 ± 0.46	2.3 ± 0.44	0.492
Na (mmol/L)	129.8 ± 7.1	129.7 ± 7.3	0.979
K (mmol/L)	4.05 ± 0.82	4.38 ± 0.74	0.088
Ca (mg/dL)	7.86 ± 0.64	8.15 ± 0.83	0.093
Ascites leukocytes (K/µL)	995 (280–2580)	2295 (470–13800)	<**0.001**
Acid PMNL (K/µL)	613.5 (7–2208)	1683 (300–12420)	<**0.001**
Acid T. protein (g/dL)	1.1 (0.8–3.6)	1.4 (0.8–4.4)	0.355
Acid albümin (g/dL)	0.5 (0.4–4)	0.6 (0.4–2.2)	0.635
Acid LDH (IU/L)	147 (38–2044)	283 (71–4500)	**0.024**
Sedimentation (mm/h)	18.5 (2–108)	16.5 (2–66)	0.955
CRP (mg/dL)	6.9 (0.3–22)	9.2 (1.2–34)	**0.047**
Procalcitonin (ng/mL)	0.8 (0.04–37.5)	0.85 (0.1–43)	0.307

Statistically significant values with a *p*-value < 0.05 are indicated in bold. Normally distributed data are given as mean ± standard deviation, and non-normally distributed data are provided as mean (minimum-maximum). ALP, alkaline phosphatase; ALT, alanine aminotransferase; AST, aspartate aminotransferase; Ca, calcium; CRP, c-reactive protein; D. Bilirubin, direct bilirubin; GGT, gamma-glutamyl transferase; Hb, hemoglobin; Hct, hematocrit; INR, international normalized ratio; K, potassium; LDH, lactate dehydrogenase; Na, sodium; PMNL, polymorphonuclear leucocytes; T. Bilirubin, total bilirubin; T. Protein, total protein

Table 2: Scoring characteristics of patients diagnosed with spontaneous bacterial peritonitis and association with mortality

Scorings	*Survivors*	*Deaths*	*p*
CTP	10 (7–12)	13 (10–15)	<**0.001**
MELD	18.3 ± 0.53	25 ± 8.63	**0.001**
Na-MELD	22.7 ± 6.49	28.5 ± 7.79	**0.001**

Normally distributed data are given as mean ± standard deviation, and non-normally distributed data are provided as mean (minimum-maximum). CTP, Child–Turcotte–Pugh; MELD, model for end-stage liver disease; Na, sodium

Table 3: Complications of cirrhosis and mortality

Complications	*Survivors*	*Deaths*	*p*
Esophageal variceal bleeding			
Present	0 (0%)	7 (25%)	<**0.001**
Absent	46 (100%)	21 (75%)	
Hepatic encephalopathy			
Present	11 (23.9%)	12 (42.9%)	0.088
Absent	35 (76.1%)	16 (57.1%)	
Hepatorenal syndrome			
Present	4 (8.7%)	12 (42.9%)	**0.001**
Absent	42 (91.3%)	16 (57.1%)	
Hepatocellular carcinoma			
Present	9 (19.6%)	6 (21.4%)	0.847
Absent	37 (80.4%)	22 (78.6%)	

syndrome and mortality in SBP. Tüzün et al. showed the relationship between esophageal variceal bleeding and mortality in SBP.[25] Our study determined a statistically significant effect of hepatorenal syndrome and esophageal variceal hemorrhage, which are complications of cirrhosis, on mortality in SBP.

Table 4: Logistic regression analysis of the relationship between laboratory characteristics and mortality in cirrhotic patients with spontaneous bacterial peritonitis

	Univariate analysis			*Multivariate analysis*		
Factors	*OR*	*95% CI*	*p*	*OR*	*95% CI*	*p*
Serum leukocytes (K/µL)	**1.000**	**1.000–1.000**	**0.001**	**1.000**	**1.000–1.001**	**0.017**
Serum neutrophils (K/µL)	**1.000**	**1.000–1.000**	**0.002**	0.999	0.996–1.003	0.719
Serum Hb (g/dL)	**0.503**	**0.333–0.761**	**0.001**	**0.197**	**0.057–0.675**	**0.010**
Serum PLT (K/µL)	1.000	1.000–1.000	0.283			
Total bilirubin (mg/dL)	1.269	1.049–1.536	0.014	1.302	0.786–2.157	0.305
Serum albumin (g/dL)	0.672	0.223–2.027	0.480			
Serum LDH (IU/L)	**1.017**	**1.008–1.025**	**0.000**	**1.016**	**1.004–1.027**	**0.007**
Creatine (mg/dL)	**2.146**	**1.212–3.801**	**0.009**	1.143	0.147–8.893	0.898
Na (mmol/L)	0.995	0.931–1.065	0.894			
INR	**63.423**	**7.418–542.265**	**0.000**	14.077	0.253–783.664	0.197
Sedimentation (mm/h)	0.996	0.973–1.021	0.776			
CRP (mg/dL)	**1.085**	**0.984–1.197**	**0.101**	1.184	0.762–1.842	0.453
Procalcitonin (ng/mL)	1.026	0.956–1.102	0.469			
Acid leukocytes (K/µL)	**1.001**	**1.000–1.002**	**0.002**	1.002	1.000–1.003	0.092
Acid PMNL (K/µL)	**1.001**	**1.000–1.002**	**0.003**	0.997	0.992–1.002	0.300
Acid albumin (g/dL)	1.028	0.435–2.430	0.950			
Acid LDH (IU/L)	1.000	1.000–1.001	0.335			

CI, confidence interval; CRP, c-reactive protein; Hb, hemoglobin; INR, international normalized ratio; LDH, lactate dehydrogenase; Na, sodium; OR, odds ratio; PLT, platelets; PMNL, polymorphonuclear leucocytes

The retrospective design of our study, the limited number of patients, and the evaluation of patient data according to the data written on the hospital electronic system are the main limitations of our study.

Conclusion

In our study, serum leukocyte, serum LDH, serum Hb values, CTP, MELD, and Na-MELD scoring systems, and hepatorenal syndrome and esophageal variceal bleeding were related to mortality in SBP. Mortality can be reduced by identifying the factors that affect death.

Ethical Statement

Ethics committee approval has been obtained from the Bursa Uludağ University Faculty of Medicine (Approval number: 2016-17/7).

Orcid

Tuğba Ocak https://orcid.org/0000-0002-4560-1569

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