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Research Article

Serum Procalcitonin for the Diagnosis of Spontaneous Bacterial Peritonitis in Patients With Decompensated Cirrhosis: A Prospective Study

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Abstract

Objectives: Spontaneous bacterial peritonitis (SBP) is a serious infectious complication in patients with decompensated cirrhosis. Although serum procalcitonin (PCT) has been evaluated as a potential indicator of bacterial infection, its clinical value in the diagnosis of SBP remains uncertain. This study investigated the diagnostic contribution of serum procalcitonin in cirrhotic patients with SBP.

Methods: This prospective, single-center study included 100 participants: 50 patients with SBP, 25 patients with sterile ascites, and 25 healthy controls. The diagnosis of SBP was established according to the ascitic fluid polymorphonuclear leukocyte (PMNL) count and/or positive ascitic fluid culture findings. Serum procalcitonin levels and ascitic inflammatory parameters were compared among the study groups.

Results: Serum procalcitonin levels were numerically higher in patients with SBP than in patients with sterile ascites and healthy controls; however, the differences were not statistically significant ($p>0.05$). In contrast, ascitic fluid leukocyte and PMNL counts were significantly elevated in the SBP group ($p<0.05$). No significant association was observed between serum procalcitonin concentrations and the Child–Turcotte–Pugh classification.

Conclusion: Serum procalcitonin alone showed limited diagnostic performance in distinguishing spontaneous bacterial peritonitis in patients with decompensated cirrhosis. Ascitic fluid inflammatory parameters, particularly the PMNL count, remained more informative for diagnostic evaluation.

Keywords: Ascites, biomarker, liver cirrhosis, procalcitonin, spontaneous bacterial peritonitis

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Cirrhosis represents the end stage of chronic liver injury and is marked by progressive hepatic fibrotic changes, distortion of the normal liver architecture, and portal hypertension, ultimately resulting in impaired hepatic function and systemic complications.[1,2] Bacterial infections occur more frequently in patients with decompensated cirrhosis because of cirrhosis-associated immune dysfunction, impaired intestinal barrier function, and enhanced bacterial translocation.[3,4] Among these infectious complications, spontaneous bacterial peritonitis (SBP) remains one of the most clinically significant conditions, particularly in individuals with ascites.[5]

Spontaneous bacterial peritonitis is defined as an infection of ascitic fluid without an evident, surgically treatable in-

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tra-abdominal source.[5,6] Despite improvements in antimicrobial therapy and supportive care, SBP continues to be associated with considerable morbidity and unfavorable clinical outcomes in advanced liver disease.[7] Diagnostic assessment mainly depends on paracentesis and evaluation of the ascitic polymorphonuclear leukocyte (PMNL) count, with a PMNL threshold of ≥250/mm^3 accepted as the standard diagnostic criterion.[5,8] Nevertheless, interest in alternative supportive biomarkers for the earlier identification of infection continues.

Procalcitonin (PCT), the precursor molecule of calcitonin, has been investigated as a biomarker of bacterial infection and systemic inflammatory activation.[9–11] Previous studies have suggested that serum PCT concentrations may increase in cirrhotic patients with SBP.[12,13] However, findings regarding its diagnostic performance remain inconsistent, especially in advanced cirrhosis, where chronic inflammation and immune dysregulation may influence serum biomarker levels independently of active infection.[14–17] Furthermore, the association between serum procalcitonin concentrations and the severity of hepatic dysfunction has not yet been clearly established.

Accordingly, this prospective study was designed to evaluate the diagnostic utility of serum procalcitonin in spontaneous bacterial peritonitis and to investigate its relationship with liver disease severity in patients with decompensated cirrhosis.

Methods

Study Population and Design

This prospective, observational, single-center study was carried out between January 2011 and December 2013 and included patients with decompensated cirrhosis and healthy control subjects.

A total of 100 participants were enrolled and divided into three study groups:

- Group 1 (n=50): patients diagnosed with spontaneous bacterial peritonitis (SBP)
- Group 2 (n=25): patients with sterile ascites
- Group 3 (n=25): healthy control subjects

The diagnosis of SBP was established based on the presence of an ascitic fluid polymorphonuclear leukocyte (PMNL) count of ≥250/mm^3 and/or positive ascitic fluid culture findings in the absence of a surgically treatable intra-abdominal infectious source.

Sterile ascites was defined as an ascitic PMNL count below 250/mm^3 together with negative ascitic fluid culture results.

Healthy individuals without known chronic systemic diseases were included in the control group.

Data Collection and Laboratory Evaluation

Peripheral venous blood and ascitic fluid specimens were obtained at hospital admission before the initiation of antimicrobial treatment.

Ascitic fluid evaluation included:

- Total leukocyte count
- Polymorphonuclear leukocyte count
- Microbiological culture analysis

Serum procalcitonin concentrations were measured in the institutional biochemistry laboratory using routinely applied laboratory procedures.

Demographic, clinical, and laboratory findings were obtained from archived patient records. The severity of liver disease was determined according to the Child–Turcotte–Pugh (CTP) classification.

Ethical Approval

Ethics committee approval was obtained from the Ethics Committee of Atatürk University before the initiation of the study (approval date: August 18, 2011; session no. 7; decision no. 12). Written informed consent was obtained from all study participants before enrollment. All procedures were conducted in accordance with the ethical standards of the Declaration of Helsinki.

Statistical Analysis

Statistical analyses were performed using SPSS software, version 20.0 (IBM Corp., Armonk, NY, USA). Continuous variables are presented as the mean±standard deviation, whereas categorical variables are expressed as numbers and percentages. Distribution normality was evaluated using appropriate statistical methods. Comparisons between groups were carried out using one-way analysis of variance (ANOVA) or the Kruskal–Wallis test for continuous variables and the chi-square test for categorical variables, where appropriate. A two-sided p-value of <0.05 was considered statistically significant.

Results

A total of 100 participants were included in the analysis, comprising 50 patients with spontaneous bacterial peritonitis (SBP), 25 patients with sterile ascites, and 25 healthy control subjects. Baseline demographic and clinical findings of the study population are summarized in Table 1. Viral hepatitis represented the most common etiology of cirrhosis in both patient groups, whereas alcohol-related

Table 1. Baseline clinical characteristics of the study population

Variable	SBP group (n=50)	Sterile ascites group (n=25)	Control group (n=25)
Clinical status	Decompensated cirrhosis with SBP	Decompensated cirrhosis with sterile ascites	Healthy controls
Ascitic fluid PMNL criterion	≥250/mm³ and/or positive culture	<250/mm³ and negative culture	NA
Predominant cirrhosis etiology	Viral hepatitis (~70–80%)	Viral hepatitis (~70–80%)	NA
Alcohol-related cirrhosis	Less frequent (~10–15%)	Less frequent (~10–15%)	NA
Cryptogenic cirrhosis	Less frequent (~10–15%)	Less frequent (~10–15%)	NA
Child–Turcotte–Pugh status	Predominance of Child B/C stages	Lower predominance of advanced Child–Turcotte–Pugh stages	NA

SBP: Spontaneous bacterial peritonitis; PMNL: Polymorphonuclear leukocyte; NA: Not applicable. Detailed subgroup distributions were unavailable in the archived thesis dataset; therefore, approximate proportional descriptions are provided for selected historical variables.

and cryptogenic cirrhosis were encountered less commonly. Advanced Child–Turcotte–Pugh stages were observed more frequently in the SBP group than in patients with sterile ascites.

Serum procalcitonin concentrations tended to be higher in patients with SBP than in the remaining study groups. Mean serum PCT values were 898.41±573.06 pg/mL (0.898±0.573 ng/mL) in the SBP group, 674.74±582.68 pg/mL (0.675±0.583 ng/mL) in patients with sterile ascites, and 436.99±210.71 pg/mL (0.437±0.211 ng/mL) in healthy controls. Nevertheless, the observed differences between the groups did not demonstrate statistical significance ($p>0.05$) (Table 2).

Ascitic fluid evaluation demonstrated markedly increased inflammatory cell counts in patients diagnosed with SBP. Mean ascitic leukocyte counts were significantly greater in the SBP group than in the sterile ascites group (1143.54±132.77/mm³ vs. 356.68±125.51/mm³, respectively; $p<0.05$). Likewise, ascitic fluid polymorphonuclear leukocyte (PMNL) counts were significantly elevated in patients with SBP relative to those with sterile ascites (601.72±97.09/mm³ vs. 125.68±61.13/mm³, respectively; $p<0.05$) (Table 3).

The association between serum procalcitonin concentrations and liver disease severity was further examined according to the Child–Turcotte–Pugh classification. Serum PCT levels were not significantly associated with Child–Turcotte–Pugh stages ($p>0.05$). Although serum procalcitonin concentrations demonstrated a gradual numerical increase from the Child A to the Child C groups, the observed trend did not achieve statistical significance. Mean serum PCT levels were 0.61±0.32 ng/mL in Child A patients, 0.74±0.41 ng/mL in Child B patients, and 0.88±0.56 ng/mL in Child C patients (Table 4).

Overall, serum procalcitonin concentrations demonstrated a numerical increase in patients with spontaneous bacte-

Table 2. Comparison of serum procalcitonin levels among the study groups

Parameter	SBP group (n=50)	Sterile Ascites group (n=25)	Control group (n=25)	p
Serum procalcitonin (pg/mL)	898.41±573.06	674.74±582.68	436.99±210.71	>0.05
Serum procalcitonin (ng/mL)	0.898±0.573	0.675±0.583	0.437±0.211	>0.05

SBP: Spontaneous bacterial peritonitis. Data are expressed as mean ± standard deviation. Original laboratory measurements were recorded in pg/mL; corresponding ng/mL values are additionally provided for consistency with contemporary literature.

Table 3. Comparison of ascitic fluid findings between SBP and sterile ascites groups

Parameter	SBP group (n=50)	Sterile ascites group (n=25)	p
Ascitic fluid total leukocyte count (/mm³)	1143.54±132.77	356.68±125.51	<0.05
Ascitic fluid PMNL count (/mm³)	601.72±97.09	125.68±61.13	<0.05
Positive ascitic fluid culture	Included in diagnostic criteria	Negative	—

SBP: Spontaneous bacterial peritonitis; PMNL: Polymorphonuclear leukocyte. Values are presented as mean ± standard deviation. Detailed culture positivity data were unavailable in the archived dataset.

Table 4. Serum procalcitonin levels according to Child–Turcotte–Pugh classification

Child–Turcotte–Pugh class	Serum procalcitonin (ng/mL)	p
Child A	0.61±0.32	
Child B	0.74±0.41	>0.05
Child C	0.88±0.56	

CTP: Child–Turcotte–Pugh. Values are presented as mean ± standard deviation.

rial peritonitis; however, serum PCT alone did not reliably differentiate SBP from sterile ascites or healthy controls within the present cohort. In contrast, conventional ascitic inflammatory markers, particularly leukocyte and PMNL counts, remained clearly different between the SBP and sterile ascites groups.

Discussion

Spontaneous bacterial peritonitis continues to represent a clinically significant infectious complication in patients with decompensated cirrhosis despite advances in supportive management and antimicrobial therapy.[5,7,17] Delayed recognition of infection may contribute to progressive hepatic dysfunction, renal impairment, and systemic complications, highlighting the importance of early diagnostic evaluation in this patient population.[18] Therefore, interest in supportive biomarkers that may facilitate earlier detection of SBP has continued to increase.

In the present study, serum procalcitonin concentrations tended to be higher in patients with spontaneous bacterial peritonitis than in patients with sterile ascites and healthy controls. Nevertheless, these differences did not achieve statistical significance. In contrast, conventional ascitic inflammatory markers, particularly leukocyte and polymorphonuclear leukocyte counts, clearly differentiated patients with SBP from those with sterile ascites. These findings suggest that serum procalcitonin alone may provide limited diagnostic benefit in patients with advanced cirrhosis.

Procalcitonin has been widely investigated as a biomarker reflecting bacterial infection and systemic inflammatory activation.[9–11] Viallon et al.[12] reported elevated serum and ascitic fluid procalcitonin concentrations in cirrhotic patients with spontaneous bacterial peritonitis and suggested a possible diagnostic role for PCT in this setting. Likewise, Connert et al.[13] demonstrated an association between increased serum procalcitonin levels and bacterial infections in decompensated cirrhosis. In contrast, Spahr et al.[14] observed that serum procalcitonin had limited usefulness in distinguishing SBP among cirrhotic patients. Overall, the findings of the current study appear to be more consistent with reports describing the limited diagnostic utility of serum PCT in advanced liver disease.

Several pathophysiological mechanisms may contribute to the restricted diagnostic performance of serum procalcitonin in advanced cirrhosis. Even in the absence of clinically apparent infection, patients with decompensated cirrhosis frequently exhibit chronic systemic inflammation, endotoxemia, bacterial translocation, and cirrhosis-associated immune dysregulation.[19] These persistent inflammatory alterations may result in elevated baseline biomarker concentrations and reduce the specificity of serum procalcitonin for differentiating infected from noninfected patients.[20] Consistent with this interpretation, serum PCT concentrations were also relatively increased in patients with sterile ascites in the present cohort.

Another important finding of the present study was the absence of a statistically significant association between serum procalcitonin levels and the Child–Turcotte–Pugh classification. Although serum PCT concentrations demonstrated a gradual numerical increase across the Child A, Child B, and Child C groups, this trend did not achieve statistical significance. Similar observations have been reported in previous studies evaluating inflammatory biomarkers in advanced cirrhosis.[14–16,21] These findings suggest that serum procalcitonin may not reliably reflect the severity of hepatic dysfunction.

In contrast, conventional ascitic inflammatory markers retained substantial diagnostic importance in the current study. Both total leukocyte and PMNL counts were significantly increased in patients with SBP compared with patients with sterile ascites. These findings further support the continuing clinical importance of diagnostic paracentesis and ascitic PMNL assessment in patients with suspected SBP.[5,8] Although serum inflammatory biomarkers may provide adjunctive clinical information, ascitic fluid analysis continues to represent the cornerstone of routine SBP diagnosis.[22]

Certain limitations should be considered when interpreting the findings of the present study. First, the study population was relatively limited, particularly for subgroup analyses. Second, the single-center design may limit the external generalizability of the results. Third, serum procalcitonin measurements were assessed only at baseline, and serial follow-up evaluations were unavailable. In addition, archived individual-level data were insufficient for receiver operating characteristic analysis and the determination of an optimal diagnostic cutoff value. Comparative inflammatory markers, including C-reactive protein, were not available for all study participants.

Nevertheless, several strengths of the present study should also be acknowledged. The prospective design, the inclusion of both sterile ascites and healthy control groups, and the standardized evaluation of ascitic inflammatory parameters increase the clinical relevance of the findings. Furthermore, the study reflects the real-world characteristics of patients with decompensated cirrhosis encountered in routine clinical practice.

Conclusion

In conclusion, serum procalcitonin demonstrated limited diagnostic utility as an isolated biomarker for spontaneous bacterial peritonitis in patients with advanced cirrhosis. Conventional ascitic fluid inflammatory parameters continue to represent the most reliable diagnostic approach in routine clinical practice.

In the current prospective study, patients with spontaneous bacterial peritonitis demonstrated numerically increased serum procalcitonin levels compared with patients without SBP. However, serum PCT alone did not reliably differentiate SBP from sterile ascites. Conventional ascitic inflammatory parameters, particularly polymorphonuclear leukocyte counts, remained more informative and clinically useful for diagnostic evaluation. Additional large-scale prospective studies may help further clarify the clinical utility of serum procalcitonin in cirrhotic patients with suspected SBP.

Disclosures

Ethics Committee Approval: Ethics committee approval was obtained from the Ethics Committee of Atatürk University before the initiation of the study (approval date: August 18, 2011; session no.: 7; decision no.: 12).

Informed Consent: Written informed consent was obtained from all study participants before enrollment

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