Clinical and laboratorial features of Spontaneous Bacterial Peritonitis in Southern Brazil

Características clínicas e laboratoriais da Peritonite Bacteriana Espontânea no Sul do Brasil

Gabriela Bicca Thiele, Otávio Marcos da Silva, Leonardo Fayad, César Lazzarotto, Mariana do Amaral Ferreira, Maíra Luciana Marconcini, Esther Buzaglo Dantas-Corrêa, Leonardo de Lucca Schiavon, Janaina Luz Narciso-Schiavon

1 Medical Student. Universidade Federal de Santa Catarina (UFSC), Florianópolis, Santa Catarina, Brazil.
2 Resident in Gastroenterology, Universidade Federal de Santa Catarina (UFSC), Florianópolis, Santa Catarina, Brazil.
3 MD, MSc. Núcleo de Estudos em Gastroenterologia e Hepatologia (NEGH), Universidade Federal de Santa Catarina (UFSC), Florianópolis, Santa Catarina, Brazil.
4 MD, PhD. II Adjunct Professor in Gastroenterology, Núcleo de Estudos em Gastroenterologia e Hepatologia (NEGH), Universidade Federal de Santa Catarina (UFSC), Florianópolis, Santa Catarina, Brazil.

Núcleo de Estudos em Gastroenterologia e Hepatologia (NEGH), Universidade Federal de Santa Catarina (UFSC), Santa Catarina, Brazil

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KEY WORDS:
Liver Cirrhosis.
Ascites.
Peritonitis.
Ascitic fluid.
Paracentesis.

ABSTRACT

CONTEXT AND OBJECTIVE: Spontaneous bacterial peritonitis (SBP) is a severe complication that occurs in 8-27% of hospitalized patients with cirrhosis and ascites, with high mortality rates. This study aims to identify clinical characteristics associated with SBP.

DESIGN AND SETTING: A cross-sectional study, conducted in a public university.

METHODS: The study included consecutive individuals with liver cirrhosis and ascites between September 2009 and March 2012. Forty-five patients were included with mean age of 53.2 ± 12.3 years, 82.2% male, 73.8% Caucasians, with mean Model of End-stage Liver Disease (MELD) of 19.5 ± 7.2, and 33.3 % with SBP. The subjects were divided into two groups: SBP and controls.

RESULTS: When individuals with SBP were compared to controls, they exhibited lower prothrombin activity (36.1 ± 16.0% versus 47.1 ± 17.2%, P = 0.044) and lower serum-ascites albumin gradient (SAAG) (1.2 versus 1.7, P = 0.045). There was a tendency of higher MELD among SBP group (22.2 ± 7.6 versus 17.9 ± 6.7, P = 0.067). There was a strong positive correlation between neutrophils count in ascitic fluid and serum leukocyte count (r = 0.501, P = 0.001) and negative correlation of neutrophils in ascitic fluid with prothrombin activity (r = -0.385, P = 0.011).

CONCLUSION: In conclusion, few characteristics are associated with the presence of SBP, especially liver dysfunction (prothrombin activity), SAAG and peripheral leukocytes count.
PALAVRAS-CHAVE:
Cirrose hepática.
Ascite.
Peritonite.
Líquido ascítico.
Paracentese.

RESUMO
CONTEXTO E OBJETIVO: Peritonite bacteriana espontânea (PBE) é uma complicaçãograve que ocorre em 8-27% dos pacientes hospitalizados com cirrose hepática (CH) e ascite, e apresenta altas taxas de mortalidade. O objetivo deste estudo é identificar as características clínicas associadas à PBE em portadores de CH descompensada em ascite.
TIPO DE ESTUDO E LOCAL: Estudo transversal, conduzido em uma universidade pública.
MÉTODOS: O estudo incluiu, consecutivamente, indivíduos com CH e ascite entre setembro/2009 e março/2012. Foram incluídos 45 indivíduos com média de idade de 53,2 ± 12,3 anos, sendo 82,2% homens, 73,8% brancos, com MELD (Modelo para Doença Hepática Terminal) de 19,5 ± 7,2, e 33,3% com PBE. Os indivíduos foram divididos em dois grupos: PBE e controles.
RESULTADOS: Quando se comparou os indivíduos com PBE aos controles, observou-se menor média de tempo de atividade da protrombina (TAP) (36,1 ± 16,0% versus 47,1 ± 17,2%; P = 0,044) e menor mediana de gradiente albumina soro-ascite (GASA) (1,2 versus 1,7; P = 0,045). Houve uma tendência ao grupo com PBE apresentar maior média de MELD (22,2 ± 7,6 versus 17,9 ± 6,7; P = 0,067). Foi observada forte correlação positiva entre neutrófilos do líquido ascítico e contagem sérica de leucócitos (r = 0,501; P = 0,001) e correlação negativa de neutrófilos do líquido ascítico com TAP (r = -0,385; P = 0,011).
CONCLUSÃO: Poucas características estão associadas à presença de PBE, em especial a disfunção hepática, o GASA e a leucocitose periférica.

INTRODUCTION
Spontaneous bacterial peritonitis (SBP) is found in 8% to 27% of the patients hospitalized with cirrhosis and ascites, and presents high taxes of intra-hospital mortality, between 20 and 40%. Studies suggest high recurrence rates, above 70% in one year.

In the great majority of the cases, the bacteria that cause SBP come from the digestive tract. Extra intestinal bacteria such as those from the respiratory and urogenital apparatus or the skin are much less frequent. Catheters and other equipment used during the invasive procedures represent another possible source of infection. Currently, the most accepted hypothesis on the pathogenesis of SBP is a bacteremia episode during fluids exchange between the peritoneal and the intravascular cavities, with consequent infection of the ascetic fluid. Aerobic gram negative bacteria (more frequently *Escherichia coli*) by means of translocation of the intestinal lumen are considered responsible by the majority of the SBP cases.

In fact, only few patients with SBP present suggestive typical symptoms of peritoneal infection as fever, abdominal pain and peripheral leukocytosis. SBP is more frequently suspected when the patient develops signals of hepatic encephalopathy, increase of the abdominal volume or renal disfunction without any apparent precipitating factor. In addition to this, in a significant part of the cases, SBP can be completely asymptomatic and the diagnosis can be done only by analyzing the paracentesis results. If ascitic fluid infection is suspected (fever, abdominal pain, unexplained encephalopathy, azotemia, acidosis, hypotension or hypothermia), it must be requested total and differential cellularity and ascitic fluid culture, with inoculation of the material in blood culture bottles at the bedside.

The SBP’s diagnosis consists in polymorphonuclear (PMN) counts ≥ 250 cells/mm³ and positive ascitic fluid culture without any evidence of external or intra-abdominal infectious source. Neutrophilic ascites, as well as the SBP, is defined by negative culture and also by PMN counts of the ascitic fluid higher than 250 cells/mm³. The presence of positive ascitic fluid culture with neutrophil count inferior than 250 cells/mm³ is diagnosed as bacterascites, and the conduct varies from patient to patient.

Given the scarcity of studies that assess the characteristics of patients with SBP in our country and also the existing regional differences, it is understood that this
infection should be evaluated in our midst. It is observing its behavior nowadays that we will be allowed to search new strategies that aim to improve its diagnostic.

OBJECTIVE

This study main objective is to identify the characteristics associated with the presence of SBP in individuals with decompensated cirrhosis with ascites, also describing the clinical profile of individuals with SBP.

METHODS

This cross-sectional analytic study, carried out through the revision of medical charts, evaluated individuals with decompensated cirrhosis with ascites that were admitted in the Gastroenterology infirmary of the Hospital Universitário Polydoro Ernani of São Thiago (HU), of the Federal University de Santa Catarina (Universidade Federal de Santa Catarina, UFSC), from September 2009 to March 2012. In the same period, we evaluated for inclusion in the study the results of ascitic fluid cultures made in the laboratory of the HU/UFSC. Among these, after an analysis of medical charts, we excluded the patients who didn’t have cirrhosis (ascites from other causes) and also those with insufficient register of clinical and laboratory data in their medical records.

The study protocol conformed to the ethical guidelines of the 1975 Helsinki Declaration and was approved by our institutional review board under the number (№: 885/10).

Clinical and laboratory variables of all the individuals with decompensated cirrhosis with ascites have been collected in the form of interview or from medical charts. The following clinical variables have been studied: SBP (which is defined by the neutrophils count of the ascitic fluid being higher than 250 cells/mm³ and/or positive culture); age; gender; color of the skin; jaundice; hepatic encephalopathy; upper gastrointestinal bleeding (UGIB) during the hospitalization; ascitic fluid culture; axillary temperature on maximum; abdominal pain; diarrhea; comorbidities: diabetes mellitus (DM), systemic arterial hypertension (SAH), dyslipidemia and HIV; etiology of the cirrhosis: alcohol, hepatitis C, hepatitis B; duration of prophylactic antibiotics in paracentesis. Among the laboratory variables, the following ones had been evaluated: Neutrophils count of the ascitic fluid; prothrombin activity (PA); albumin serum-ascites
albumin gradient (SAAG); hemoglobin; leukocytes; platelets count; aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (AP); gamma glutamyltransferase (GGT); albumin; total and direct bilirubin; creatinine; sodium; glucose; total protein of the ascitic fluid; albumin of the ascitic fluid. Liver biochemical tests AST; ALT; AP and GGT have been expressed in times the upper limit of normal (xULN). The other laboratory variables have been expressed as absolute values. Bilirubin tests, INR and creatinine have been used for MELD calculation (Model of End-stage Liver Disease). For analysis purposes, the patients were divided into two chronological groups: SBP and controls (cirrhotic individuals with ascites and no evidence of infection).

**Statistical analysis**

Continuous variables were compared using the Student t test or Mann-Whitney test when appropriate. Categorical variables were compared using the chi-square or Fisher exact test. P values less than 0.05 were considered statistically significant. The correlation between the neutrophils' count of the ascitic fluid and laboratory variables was assessed using the correlation coefficient of Pearson. All tests were executed by the Statistical Package for Social Science (SPSS, Chicago, IL, USA), version 17.0. Descriptive levels (P) lower than 0.05 were considered statistically significant.

**RESULTS**

From September 2009 to March 2012, 86 patients were evaluated for inclusion in the study because they presented decompensated cirrhosis with ascites and/or positive result of ascitic fluid culture in the laboratory. Three individuals were excluded from the study for not presenting neutrophils count of the ascitic fluid as well as other 38 patients that did not have cirrhosis (Figure 1).

We included in the study 45 patients with decompensated cirrhosis with ascites and 15 (33.3%) presented SBP (Table 1). The mean age was 53.2 ± 12.3 years, 82.2% of the patients were men, 73.8% of them were Caucasian. About 60% of the individuals were classified with Child-Pugh C and a mean MELD score of 19.5 ± 7.2. No individual was classified as Child-Pugh A and only one presented MELD score ≤ 10. Besides the SBP, we observed that 63.6% had jaundice, 35% hepatic encephalopathy
and 37.1% upper gastrointestinal bleeding. Only two individuals (6.5%) were using prophylactic antibiotics (norfloxacin) and none of them presented SBP.

With regard to the etiology of the cirrhosis, it was observed that 46.7% showed only alcoholism, 20% HCV and alcoholism, 20% hepatitis C, 6.7% only hepatitis B and 2.2% HBV and alcoholism. When the comorbidities presented in patients were evaluated, 37.9% presented diabetes, 42.9% hypertension and 4.3% dyslipidemia. Acquired immunodeficiency syndrome was found in 9.4% of the individuals, in co-infection with the HCV. The biochemical characteristics of individuals with decompensated cirrhosis with ascites are described in Table 2.

**Evaluation of the individuals included in the study in accordance with the presence of SBP**

Between the SBP group, the neutrophils count mean in the ascitic fluid was 1.393.5 ± 1.115.1 cells/mm³ and three patients (21.4%) presented positive ascitic fluid culture, two with Klebsiella pneumoniae and one with Streptococcus sp. The diagnostic paracentesis was performed, on average, with 3.6 ± 3.5 (median) days of hospitalization.

When the SBP was compared to controls (Tables 1 & 2), it was observed that the patients with SBP presented lower mean of PA (36.1 ± 16.0% versus 47.1 ± 17.2%; P = 0.044) and lower median of SAAG (1.2 versus 1.7; P = 0.045). There was a tendency of the SBP group to present higher MELD score mean (22.2 ± 7.6 versus 17.9 ± 6.7; P = 0.067). No difference was observed when the following clinical and laboratory variables were analyzed: age, gender, race, axillary temperature on maximum, abdominal pain, diarrhea, Child-Pugh, jaundice, presence of encephalopathy, upper gastrointestinal bleeding, death in the hospitalization, hemoglobin, leukocytes, platelets, AST, ALT, GGT, AP, albumin, total bilirubin, creatinine, sodium, glucose, proteins of the ascitic fluid and albumin of the ascitic fluid.

We observed a strong positive correlation between the neutrophils count of the ascitic fluid and the serum leukocytes count (r = 0.501; P = 0.001). It was also noted a negative correlation of the neutrophils count of the ascitic fluid in relation to the PA (r = -0.385; P = 0.011). Significant correlation with hemoglobin, platelets, AST, ALT, AP, GGT, serum albumin, INR, total bilirubin, creatinine, sodium, glucose, total proteins of
the ascitic fluid, albumin of the ascitic fluid, SAAG, and maximum axillary temperature has been observed.

DISCUSSION

SBP is described, more frequently, among individuals of the masculine sex, in percentages ranging from 72.8% to 83.7%, similar to what was found in this study.\textsuperscript{13-16} Despite the mean age observed between the individuals with SBP being somewhat lower than previously described by other authors, from 52.8 to 58.4 years,\textsuperscript{13-16} most patients were classified as Child-Pugh C (72.7%) with a high MELD score (22.2). Other authors noted prevalence of Child-Pugh Class C ranging from 50.9% to 77.7%\textsuperscript{14,16-18} and mean MELD score between 16.6 and 23.2, befitting what was found in this study.\textsuperscript{14,16,19}

With regard to the etiology of the cirrhosis, Heo et al. described a greater prevalence of cirrhosis by HBV (71.3%), followed by alcoholic cirrhosis (19.7%) and cirrhosis by HCV (6.4%),\textsuperscript{16} data that is consistent with the high prevalence of HBV in the Asian continent.\textsuperscript{20-22} In the North America, Heidelbaugh et al. reported alcohol as that the main cause of cirrhosis (60-70%), followed by viral hepatitis (10%) and non-alcoholic fatty liver disease (10%),\textsuperscript{23} similar to a Brazilian study, made in Rio de Janeiro, which showed 39.9% of cirrhosis of alcoholic etiology, 28.7% by virus,11.9% of mixed etiology (alcohol and virus) and 14.7% of varied etiologies.\textsuperscript{18} Alcoholism was described in Brazil in a prevalence range between 7.6% and 9.2%, what justifies the importance of this etiology as a cause of cirrhosis in our milieu.\textsuperscript{24}

Even though the diagnosis of SBP is made with an ascitic liquid neutrophil count \(\geq 250\) cells/mm\(^3\) of, cellularities of up to 8,000 neutrophils per mm\(^3\) have been described.\textsuperscript{25-26} Despite the high cellularity found in this study, only 21.4% of cases presented positive ascitic fluid culture, which is described in the literature in prevalence cases ranging from 12.6% and 68.4%.\textsuperscript{13,15,18,25,27,28}

When we evaluated the clinical and laboratory characteristics in relation to presence of SBP, the findings seen in the literature were controversial. The heterogeneity of the findings that are associated to the presence of SBP justifies the indication of paracentesis diagnostic in all patients with decompensated cirrhosis with ascites that were admitted in the hospital.\textsuperscript{13} Evans et al.\textsuperscript{29} assessed 427 patients with ascites and observed that 3.5% had SBP, but there were no significant differences in
relation to the serum albumin, serum bilirubin and INR between patients with and without SBP. Similarly to what was found in this study, it was described that individuals with moderate to high MELD score present a substantially greater risk for the development of SBP.\textsuperscript{19,28} At the same time, leukocytosis in the peripheral blood can help to predict the appearance of SBP in asymptomatic patients with ascites.\textsuperscript{14} Other variables that were described as predictors of SBP are: C-reactive protein, VHS (14), UGIB, hypoalbuminemia.\textsuperscript{18}

Figueiredo et al.\textsuperscript{18} evaluated 143 individuals with decompensated cirrhosis with ascites, among which, 20.3\% presented SBP diagnosis. About the analyzed variables, serum albumin (P < 0.001), C4 of ascitic fluid (P < 0.001) and UGIB in the previous week (P = 0.03) were identified as independent predictors for the diagnosis of SBP, and these combined variables might predict almost 97\% of episodes of ascitic fluid infection. Kim et al.\textsuperscript{30} assessed 188 patients with cirrhosis and showed that, when compared to patients with serum sodium ≥ 136 mmol/L, cirrhotic individuals with serum sodium concentration of ≤ 130 mmol/L present a significantly higher risk for the development of SBP (33.3\% versus 16.3\%; P = 0.037) Guarner et al.,\textsuperscript{31} evaluated 109 patients with ascites and cirrhosis, and discovered that 25.6\% had developed SBP. Of the 20 variables evaluated in this study, five presented positive value that predict the emergence of SBP: Child-Pugh score (P = 0.08), presence of encephalopathy (P = 0.06), serum bilirubin concentration (P = 0.007), total platelet count (P = 0.02) and total proteins in the ascitic fluid (P = 0.05). Only serum bilirubin count and platelet count presented independent correlation with the risk of SBP development.

Such et al.,\textsuperscript{32} evaluated 33 patients hospitalized with cirrhosis, of these, 21.2\% had SBP diagnosis. In SBP patients, the C3 concentration in the ascitic fluid was significantly lower when compared to patients who had not developed ascitic fluid infection (9.0 ± 2.67 versus 18.26 ± 8.11; P < 0.01). The C4 concentration did not show significant difference. Some serum markers were also indicated as predictors of SBP, they were: albumin (P < 0.05), bilirubin dosage (P < 0.05) and PA (P < 0.05). Girón-González et al.,\textsuperscript{33} studied 32 patients with cirrhosis and 20 of them had ascitic fluid infection. The study showed that SBP is significantly associated with high serum level of ICAM-I (P < 0.05), IL-8 (P < 0.01) and Gro-alpha (P <0.01) and also with high levels of ICAM-I in the ascitic fluid (P < 0.05). A positive correlation was detected between PMN count in the ascitic fluid and concentration of IL-8 (r = 0.65; P < 0.01).

Coskun et al.,\textsuperscript{34} studied 50 individuals with cirrhosis, being 20\% with SBP. In their
analysis, they demonstrated that nitrate levels are significantly higher in patients with SBP when compared with the patients without SBP (282.4 ± 111.3 versus 186.4 ± 87.6; P< 0.05). In the same way, they showed higher levels of nitrate in the ascitic fluid of patients with SBP (302.4 ± 66 versus 135.4 ± 65.8; P < 0.001).

Regarding the factors associated with SBP and looking at what has already been described in the literature, we perceived that these studies are discordant, which means that each one found different variables to predict the appearance of SBP. This study, as well as the others, identified some variables as factors of the existence of SBP, characteristics that confirm the superior prevalence of SBP between individuals with advanced liver disease.

This study reinforces the recommendation that a diagnostic paracentesis should be performed on hospital admission in all cirrhotic patients with ascites to investigate the presence of SBP, even in patients admitted for reasons other than ascites, as no clinical characteristics other than the gravity of liver disease can predict ascitic fluid infection.

**CONCLUSION**

In conclusion, few characteristics are associated with the presence of SBP, especially liver dysfunction (prothrombin activity), SAAG and peripheral leukocytes.

**REFERENCES**


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**Conflict of interest:** None  

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**Address for correspondence:**  
Janaína Luz Narciso-Schiavon  
Departamento de Clínica Médica  
Hospital Universitário Polydoro Ernani de São Thiago, 3º andar  
Universidade Federal de Santa Catarina (UFSC)  
Rua Professora Maria Flora Pausewang, s/n²  
Trindade — Florianópolis (SC) — Brasil  
CEP 88040-900  
Tel. (+55 48) 3721-9014  
E-mail: janaina.narciso@uol.com.br
Table 1. Comparative analysis of the clinical characteristics of 45 individuals with decompensated cirrhosis with ascites, in accordance with the presence of spontaneous bacterial peritonitis (SBP)

<table>
<thead>
<tr>
<th></th>
<th>Total</th>
<th>With SBP n = 15</th>
<th>Without SBP n = 30</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)*</td>
<td>53.2 ± 12.3</td>
<td>49.7 ± 13.0</td>
<td>55.0 ± 11.8</td>
<td>0.175 &lt;sup&gt;1&lt;/sup&gt;</td>
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<tr>
<td></td>
<td>(52.0)</td>
<td>(46.0)</td>
<td>(55.0)</td>
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<tr>
<td>Male gender, n (%)</td>
<td></td>
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<tr>
<td></td>
<td>37 (82.2)</td>
<td>12 (80.0)</td>
<td>25 (83.3)</td>
<td>1.000 &lt;sup&gt;1&lt;/sup&gt;</td>
</tr>
<tr>
<td>Caucasian, n (%)</td>
<td>31 (73.8)</td>
<td>10 (71.4)</td>
<td>21 (75.0)</td>
<td>1.000 &lt;sup&gt;1&lt;/sup&gt;</td>
</tr>
<tr>
<td>Child-Pugh C, n (%)</td>
<td>16 (59.3)</td>
<td>8 (72.7)</td>
<td>8 (50.0)</td>
<td>0.427 &lt;sup&gt;1&lt;/sup&gt;</td>
</tr>
<tr>
<td>MELD*</td>
<td>19.5 ± 7.2</td>
<td>22.2 ± 7.6</td>
<td>17.9 ± 6.7</td>
<td>0.067 &lt;sup&gt;1&lt;/sup&gt;</td>
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<tr>
<td></td>
<td>(19)</td>
<td>(20.0)</td>
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<tr>
<td>Complications:</td>
<td></td>
<td></td>
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<tr>
<td>. SBP, n (%)</td>
<td></td>
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<tr>
<td></td>
<td>15 (33.3)</td>
<td>15 (100.0)</td>
<td>0 (0.0)</td>
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<tr>
<td>. Jaundice, n (%)</td>
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<tr>
<td></td>
<td>28 (63.6)</td>
<td>11 (73.3)</td>
<td>17 (58.6)</td>
<td>0.336 &lt;sup&gt;4&lt;/sup&gt;</td>
</tr>
<tr>
<td>. Encephalopathy, n (%)</td>
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<tr>
<td></td>
<td>14 (35.0)</td>
<td>5 (41.7)</td>
<td>9 (32.1)</td>
<td>0.720 &lt;sup&gt;1&lt;/sup&gt;</td>
</tr>
<tr>
<td>. Upper gastrointestinal bleeding, n (%)</td>
<td>13 (37.1)</td>
<td>5 (41.7)</td>
<td>8 (34.8)</td>
<td>0.726 &lt;sup&gt;1&lt;/sup&gt;</td>
</tr>
<tr>
<td>Etiology of the cirrhosis:</td>
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<td>. Hepatitis B, n (%)</td>
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<tr>
<td></td>
<td>4 (12.1)</td>
<td>1 (10.0)</td>
<td>3 (13.0)</td>
<td>1.000 &lt;sup&gt;1&lt;/sup&gt;</td>
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<td>. Hepatitis C, n (%)</td>
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<tr>
<td></td>
<td>18 (47.4)</td>
<td>5 (38.5)</td>
<td>13 (52.0)</td>
<td>0.428 &lt;sup&gt;4&lt;/sup&gt;</td>
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<tr>
<td>. Alcohol, n (%)</td>
<td></td>
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<tr>
<td></td>
<td>31 (73.8)</td>
<td>12 (85.7)</td>
<td>19 (67.9)</td>
<td>0.283 &lt;sup&gt;1&lt;/sup&gt;</td>
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<tr>
<td>Comorbidities:</td>
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<tr>
<td>. Diabetes Mellitus, n (%)</td>
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<tr>
<td></td>
<td>11 (37.9)</td>
<td>2 (20.0)</td>
<td>9 (47.4)</td>
<td>0.234 &lt;sup&gt;1&lt;/sup&gt;</td>
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<td>. Hypertension, n (%)</td>
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<td></td>
<td>12 (42.9)</td>
<td>5 (50.0)</td>
<td>7 (38.9)</td>
<td>0.698 &lt;sup&gt;1&lt;/sup&gt;</td>
</tr>
<tr>
<td>. Dyslipidemia, n (%)</td>
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<tr>
<td></td>
<td>1 (4.3)</td>
<td>0 (0.0)</td>
<td>1 (6.3)</td>
<td>1.000 &lt;sup&gt;1&lt;/sup&gt;</td>
</tr>
<tr>
<td>. Aids, n (%)</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>3 (9.4)</td>
<td>11 (73.3)</td>
<td>17 (58.6)</td>
<td>0.336 &lt;sup&gt;4&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

MELD = Model of End Stage Liver Disease; Aids = acquired immunodeficiency syndrome. *Mean ± standard deviation (median).
Table 2. Comparative analysis of the laboratory characteristics of 45 individuals with decompensated cirrhosis with ascites, in accordance with the presence of spontaneous bacterial peritonitis (SBP)

<table>
<thead>
<tr>
<th></th>
<th>Total</th>
<th>With SBP n = 15</th>
<th>Without SBP n = 30</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin (g/dL)*</td>
<td>9.5 ± 2.4 (9.6)</td>
<td>9.1 ± 2.7 (9.1)</td>
<td>9.7 ± 2.3 (9.7)</td>
<td>0.385&lt;sup&gt;i&lt;/sup&gt;</td>
</tr>
<tr>
<td>Leukocytes (/mm&lt;sup&gt;3&lt;/sup&gt;)*</td>
<td>9.298.9 ± 6.7687 (8.550.0)</td>
<td>11.921.3 ± 9.5461 (8.880.0)</td>
<td>7.987.7 ± 4.4927 (8.000.0)</td>
<td>0.647&lt;sup&gt;i&lt;/sup&gt;</td>
</tr>
<tr>
<td>Platelets (/mm&lt;sup&gt;3&lt;/sup&gt;)*</td>
<td>134.266.7 ± 104.811.4 (109.000.0)</td>
<td>142.600.0 ± 102.083.2 (111.000.0)</td>
<td>130.100.0 ± 107.623.7 (76.500.0)</td>
<td>0.289&lt;sup&gt;m&lt;/sup&gt;</td>
</tr>
<tr>
<td>AST (U/L xULN)*</td>
<td>4.7 ± 7.7 (2.1)</td>
<td>7.8 ± 12.7 (2.2)</td>
<td>3.1 ± 2.2 (2.0)</td>
<td>0.804&lt;sup&gt;m&lt;/sup&gt;</td>
</tr>
<tr>
<td>ALT (U/L xULN)*</td>
<td>1.4 ± 1.6 (0.9)</td>
<td>1.7 ± 2.5 (0.6)</td>
<td>1.2 ± 0.8 (0.9)</td>
<td>0.144&lt;sup&gt;m&lt;/sup&gt;</td>
</tr>
<tr>
<td>AP (U/L xULN)*</td>
<td>1.0 ± 0.8 (0.9)</td>
<td>1.1 ± 0.6 (1.0)</td>
<td>1.0 ± 0.9 (0.6)</td>
<td>0.126&lt;sup&gt;m&lt;/sup&gt;</td>
</tr>
<tr>
<td>GGT (U/L xULN)*</td>
<td>3.6 ± 3.5 (2.3)</td>
<td>3.2 ± 2.7 (2.4)</td>
<td>3.8 ± 3.8 (2.2)</td>
<td>0.559&lt;sup&gt;i&lt;/sup&gt;</td>
</tr>
<tr>
<td>Albumin (g/dL)*</td>
<td>2.0 ± 0.6 (2.0)</td>
<td>1.9 ± 0.7 (1.9)</td>
<td>2.1 ± 0.6 (2.1)</td>
<td>0.466&lt;sup&gt;i&lt;/sup&gt;</td>
</tr>
<tr>
<td>PA (%)*</td>
<td>43.5 ± 17.4 (42.8)</td>
<td>36.1 ± 16.0 (35.4)</td>
<td>47.1 ± 17.2 (44.5)</td>
<td>0.044&lt;sup&gt;i&lt;/sup&gt;</td>
</tr>
<tr>
<td>INR*</td>
<td>1.7 ± 0.5 (1.6)</td>
<td>1.7 ± 0.6 (1.6)</td>
<td>1.6 ± 0.4 (1.6)</td>
<td>0.639&lt;sup&gt;m&lt;/sup&gt;</td>
</tr>
<tr>
<td>BRBT (mg/dL)*</td>
<td>5.5 ± 6.0 (2.8)</td>
<td>6.7 ± 5.9 (4.9)</td>
<td>4.8 ± 6.0 (2.0)</td>
<td>0.109&lt;sup&gt;m&lt;/sup&gt;</td>
</tr>
<tr>
<td>Creatinine (mg/dL)*</td>
<td>1.4 ± 0.7 (1.2)</td>
<td>1.6 ± 0.9 (1.4)</td>
<td>1.3 ± 0.5 (1.2)</td>
<td>0.303&lt;sup&gt;m&lt;/sup&gt;</td>
</tr>
<tr>
<td>Sodium (mEq/L)*</td>
<td>134.3 ± 5.9 (136.0)</td>
<td>133.1 ± 8.2 (133.0)</td>
<td>134.9 ± 4.3 (136.0)</td>
<td>0.245&lt;sup&gt;m&lt;/sup&gt;</td>
</tr>
<tr>
<td>Glucose (mg/dL)*</td>
<td>94.3 ± 23.4 (92.0)</td>
<td>91.4 ± 20.8 (91.0)</td>
<td>95.5 ± 24.8 (100.0)</td>
<td>0.524&lt;sup&gt;m&lt;/sup&gt;</td>
</tr>
<tr>
<td>Analysis of AF</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>. Proteins T (g/dL)*</td>
<td>1.1 ± 0.7 (0.9)</td>
<td>1.2 ± 0.5 (1.2)</td>
<td>1.0 ± 0.8 (0.9)</td>
<td>0.153&lt;sup&gt;m&lt;/sup&gt;</td>
</tr>
<tr>
<td>. Albumin (g/dL)*</td>
<td>0.3 ± 0.3 (0.3)</td>
<td>0.3 ± 0.2 (0.3)</td>
<td>0.4 ± 0.3 (0.3)</td>
<td>0.674&lt;sup&gt;m&lt;/sup&gt;</td>
</tr>
<tr>
<td>. SAAG (g/dL)*</td>
<td>1.6 ± 0.6 (1.6)</td>
<td>1.3 ± 0.4 (1.2)</td>
<td>1.7 ± 0.6 (1.7)</td>
<td>0.045&lt;sup&gt;m&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

AST = aspartate aminotransferase; ALT = alanine aminotransferase; AP = alkaline phosphatase; GGT = gamma glutamyl transferase; xULN = times the upper limit of normal; PA = prothrombin activity; INR = international normalized ratio; BRBT = total bilirubin; AF = ascitic fluid; T = total; SAAG = serum albumin ascitic gradient. *Mean ± standard deviation (median).
**Figure 1.** Flowchart of potential candidates for inclusion in the study, exclusion criteria and included individuals.

- Patients with cirrhosis and ascites
  - $n = 27$

- Positive ascitic fluid cultures
  - $n = 58$

Excluded:
- Without neutrophils $count = 3$
- Non-cirrhotic individuals $= 38$

- Individuals included in the study
  - $n = 45$