

Título: “Perfil microbiológico de la peritonitis bacteriana espontánea en una ciudad del sur de Brasil”

Title: “Microbiological profile of Spontaneous Bacterial Peritonitis in a Southern Brazilian City”

Título corrente: “Perfil microbiológico de la peritonitis bacteriana espontánea”

Running title: “Microbiological profile of Spontaneous Bacterial Peritonitis”

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SUMMARY

INTRODUCTION: The Spontaneous Bacterial Peritonitis (SBP) is one of the most frequent infectious complications that affect patients with decompensated cirrhosis and ascites, with high mortality. **OBJECTIVE:** To identify the main causing agents of SBP in a University Hospital between 2008 and 2011. **METHODS:** A cross-sectional study was carried out through positive results of ascitic fluid cultures. Clinical and laboratorial variables were extracted from the medical records. **RESULTS:** Were included 47 patients with positive ascitic fluid cultures, with 55.7 ± 15.5 years-old, from which 70.2% men and 53.6% presented cirrhosis. All cirrhotic patients presented GASA ≥ 1.1 and mean neutrophils' count in the ascitic fluid of $3,260.8 \pm 5,122.9$ cells. The most frequent germ found was *Escherichia coli* (25.5%), followed by *Klebsiella* (14.9%), *Enterococcus* (8.5%) and *Streptococcus* (8.5%). No significant differences were observed when cirrhotic were compared with non-cirrhotic patients regarding the prevalence of the *E.coli* (19.2% vs. 33.3%; P=0.270), *Klebsiella* (19.2% vs. 9.5%; P=0,436), *Enterococcus* (7.7% vs. 9.5%; P=1.000) and *Streptococcus* (15.4% vs. 0.0%; P=0.117). The presence of infection for two or more germs was more common among individuals without cirrhosis (11.5% vs. 38.1%; P=0,047). **CONCLUSION:** The microbiological profile of ascitic fluid cultures of this hospital are similar to other studies related to the SBP, with prevalence of gram-negative.

Keywords: Hepatic cirrhosis; Peritonitis; Ascitic fluid.

INTRODUCTION

Spontaneous Bacterial Peritonitis (SBP) is a frequent infectious complication that affects patients with decompensated cirrhosis and ascites¹. This disease is characterized by an ascitic fluid infection without evidence of a visceral perforation and an intra-abdominal inflammatory focus. For instance, there is acute pancreatitis or cholecystitis²⁻⁴. SBP normally occurs on the final stage of liver diseases and presents a high rate of recurrence – about 70% in a year⁵⁻⁷. Besides decompensated cirrhosis, other factors predispose the presence of SBP, including jaundice, malnutrition, and upper gastrointestinal bleeding⁸. Some studies also suggest high in-hospital mortality of patients with cirrhosis and ascites (20% to 40%)⁹⁻¹⁰. However, this rate has decreased in the last four decades because of early diagnosis and immediate use of proper antibiotherapy¹¹.

The criteria for the diagnosis of SBP require that paracentesis be collected and ascitic fluid be analyzed. The bacterial culture must be positive and/or the neutrophil (polymorphonuclears) count in this fluid must exceed 250 cells/mm³¹². The clinical manifestations are nonspecific. The most frequent signs and symptoms include: fever, abdominal pain, hepatic encephalopathy, pain or abrupt abdominal decompression, diarrhea, paralytic ileum and hypothermia. Normally, SBP is suspected when the patient begins to show signs of hepatic encephalopathy or drastic decrease of renal function, without any precipitating factor. Approximately 10% of patients with SBP have no signs or symptoms¹³⁻¹⁶.

The bacterial translocation through the intestinal cavity to mesenteric lymph nodes should be the main mechanism for developing the bacteremia, which precedes SBP manifestations. On the individuals with cirrhosis, there are three mechanisms involved on this infection's pathogenesis: deficient local immune response (decline of

the hepatic macrophages's phagocytic activity), bacterial overgrowth in the intestinal lumen, and functional and structural alterations on the intestinal mucosal barrier^{14,17}. Most of the microorganisms responsible for SBP derive from the intestinal flora, mainly the aerobic gram-negative bacteria. *Escherichia coli* and *Klebsiella pneumoniae* are the most frequently isolated agents¹⁸⁻²⁰. In approximately 25% of the cases, gram-positive bacteria, such as *Streptococcus* and *Enterococcus*, are found. *Streptococcus pneumoniae* is the most commonly found^{21,22}. On the other hand, anaerobic bacteria are never the cause of SBP, because of their inability to move on the intestinal mucosa, and due to the high levels of oxygen on the gut wall²³.

Based on the above considerations, this study has as its goal to identify the microorganisms found on ascitic fluid cultures and to describe the clinical characteristics related to the presence of ascitic fluid's infections on patients with cirrhosis.

METHODS

Casuistic

A cross-sectional study was carried out using positive results of ascitic fluid cultures in the microbiology laboratory of the Polydoro Ernani de São Thiago University Hospital at the Federal University of Santa Catarina (UFSC) from January 2008 to December 2011. Patients with missing data on their medical charts were excluded, and only the first culture of those that presented more than one positive result was included.

This study's protocol is in compliance with the ethical rules of the Helsinki Declaration and was approved by the UFSC Committee on the Ethics of Research on Human Beings (certificate N. 948).

Methods

Information on all individuals submitted to paracentesis, and had positive results on the ascitic fluid culture, was reviewed. Clinical, demographic, and laboratorial variables were gathered from medical reports. The following variables were examined: age (years); gender; length of stay (days); isolated germs on cultures; positive serologies for HBsAg, anti-HCV, and anti-HIV; creatinine; hemoglobin; platelet count, aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (AP), gamma glutamyltransferase (GGT), serum albumin, bilirubin, international normalization ratio (INR) and prothrombin activity (PA). The AST, ALT, FA and GGT hepatic biochemical tests were expressed on times the upper limit of normal (xULN). The other variables were expressed in absolute values. Bilirubin, INR, and creatinine were used to find the MELD (*Model for End-stage Liver Disease*)²⁴ on individuals with cirrhosis. Only laboratory tests performed within six months after the development of the culture were included in this study. These cultures were collected and plated at the bedside in vials of blood cultures (BacT/ALERT®; bioMérieux) or collected in dry tubes and plated at the laboratory on Blood Agar, MacConkey Agar and Thioglycollate Broth.

Static Analysis

Continuous variables were described with measures of central tendency and dispersion, while the categorical variables were described in absolute numbers and proportions. Continuous variables were compared using Student's *t*-test or Mann-Whitney as appropriate, and the categorical variables were assessed using chi-squared

test or Fisher exact test, as appropriate. The p values smaller than 0.05 were considered statically significant. All tests were two-tailed and carried out using the *Statistical Package for Social Science* software, version 17.0 (SPSS, Chicago, IL, USA).

RESULTS

3.1 Case-by-case examination

From January 2008 to December 2011, 660 ascitic fluid cultures were assessed, among which 53 (8.03%) were positive and were evaluated for inclusion in the study. Four cultures were excluded because they had insufficient medical records and two other for presenting repeatedly reactive results (Figure 1).

A total of 47 patients showing positive ascitic fluid cultures, with standard deviation of 55.7 ± 15.5 (55.0) mean years of age, from which 70.2% were male and 26 patients (55.3%) had cirrhosis.

Among the non-cirrhotic patients, acute appendicitis, dialytic chronic renal failure, and acute cholecystitis were the most commonly found pathologies.

Among the individuals with cirrhosis, the mean MELD score was 16.3 ± 9.3 and the GASA score was 1.6 ± 0.7 and all of them presented $GASA \geq 1.1$. Regarding the albumin and the neutrophils' count in this group's ascitic fluid, the mean, standard deviation, and median were respectively 0.3 ± 0.2 (0.3) g/dL and $3260,8 \pm 5122,9$ (892) cells. No differences were observed when comparing the neutrophil's median of the ascitic fluid of cirrhotic patients to infections by one or more germs, no differences were observed (892.0 vs. 322.5; $P= 0.407$).

3.2 Assessment of patients included according to the diagnosis of cirrhosis:

When comparing hepatic cirrhotic patients with others (Table 2), we noted a larger proportion of males (84.6% vs. 52.4%; $P = 0.016$); larger median of AST (2.0 vs. 1.0 xULN; $P = 0.021$) and bilirubin (2.7 vs. 0.7 g/dL; $P = 0.008$) and smaller median of platelets (1095000 vs. 2470000 /mm³; $P = 0.001$). The differences of age, length of hospital stay, infection by one or more germs, positive results of HBsAg, anti-HCV or anti-HIV were not found. Regarding the laboratorial variables, differences were not found for creatinine, hemoglobin, platelets, ALT, AP, GGT, albumin and PA values.

3.3 Assessment of culture results according to the presence of cirrhosis:

Escherichia coli (25.5%) was the most frequently found germ, followed by *Klebsiella* (14.9%), *Enterococcus* (8.5%) and *Streptococcus* (8.5%). The detailed microbial profile is described on Table 1. When comparing the frequency of these germs in the ascitic fluid cultures of cirrhotic patients to other germs (Figure 2), it was impossible to note great differences in the prevalence of *E.coli* (19.2% vs. 33.3%; $P = 0.270$), *Klebsiella* (19.2% vs. 9.5%; $P = 0.436$), *Enterococcus* (7.7% vs. 9.5%; $P = 1.000$) and *Streptococcus* (15.4% vs. 0.0%; $P = 0.117$). Infection by two or more germs was most commonly found among individuals with no cirrhosis ($P = 0.047$).

DISCUSSION

The described mean age of the individuals suffering from cirrhosis, with SBP, ranges between 54.3 ± 10 years-old and 58.3 ± 13.1 years-old²⁵⁻²⁷, quite similar to what was found in this study. Nonetheless, a smaller mean age was reported by other authors, ranging between 48.3 ± 1.8 and 49 years-old^{28,29}. Higher prevalence of males

has been reported by several authors. It can vary from 52.3 to 78.2%^{25,29}, and has been emphasized in up to 100% of the cases²⁹.

Due to the reduction of endotoxins and bacteria, liver insufficiency results in a greater susceptibility to infections, even causing immunosuppression in some patients. Although Shaw *et al.* have described that SBP is linked to HIV (*Human Immunodeficiency Virus*) infection on cirrhotic patients³⁰; in this study, none of the patients with cirrhosis and SBP were infected with HIV. Accordingly, among the non-cirrhotic patients, it was observed that 9% were HIV positive, possibly because the immunosuppression derived from the HIV is a risk factor for infection, regardless of the presence of cirrhosis.

This study shows that patients with cirrhosis and SBP presented liver dysfunction accompanied by low platelet count, low concentration of serum albumin, low PA and high MELD score, in addition to low albumin concentration in the ascitic fluid. All of these variables are associated with poor prognosis in patients with cirrhosis^{31,32}. It is clear that SBP generally occurs on patients with advanced cirrhosis. The higher the patient's MELD, the higher the risk to develop ascitic fluid infection³³. The mean MELD in this study was 16.3 ± 9.3 for cirrhotic patients, similar to Shi *et al.*²⁷ who described a mean MELD of 16.5 ± 5.1 , and inferior to that one presented by Desai *et al.*²⁶, of 23.8 ± 8.4 . In this study, low platelets count was evident, with an mean of $129,650.0 \pm 97,739.0/\text{mm}^3$ on the cirrhotic patients, higher than the mean found on other studies, which ranged from $77,960.0 \pm 48,370.0$ to $109,000.0 \pm 73,000.0/\text{mm}^3$ ^{27,34}. In this research, the average prothrombin activity on patients with cirrhosis was 42.7 ± 16.4 %, lower than that described by Shi *et al.*, of 55.4 ± 14.2 %²⁷. The mean of serum albumin for patients with cirrhosis ranges from 2.0 ± 0.4 to 2.8 ± 0.3 g/dL^{28,34-36}, similar to our study. Solá *et al.* acknowledged the tendency of a

higher incidence of SBP on individuals with low protein concentrations in the ascitic fluid³⁷. It was here detected an mean of $1,0 \pm 0,5$ g/dL of proteins in the ascitic fluid, similar to that found by Kim *et al.* of 1.2 ± 1.0 g/dL³⁵. The ascitic fluid albumin is described by values of 0.6 ± 0.3 g/dL and 1.0 ± 1.0 g/dL^{35,25}. The mean number of neutrophils in the ascitic fluid among patients with cirrhosis and SBP is compatible with the one described in the literature, with values that vary from 529 to 4,900 cells/mm³^{25,28,34,38}.

When using conventional techniques of microbiological diagnosis of SBP, the culture of ascitic fluid is negative in more than 60% of the cases, even in the presence of suggestive clinical manifestations (fever, abdominal pain, unexplained encephalopathy, acidosis, azotemia, hypotension or hypothermia). This occurs due to deficiency of the culture techniques. The inoculation of the ascitic fluid in vials of blood culture, by the bedside, with at least 10mL of fluid, makes it possible to increase to up to 90% the chances of obtaining positive culture. The transport of the fluid to the laboratory without using specific containers, such as syringe or tube, is also a factor that decreases the sensitivity of the test. In our milieu, we still used, in large part, the collection of ascitic fluid in a dry tube, fact that, perhaps in part, justifies why only 8% of the samples of ascitic fluid that was assessed in the laboratory showed positive results (although not knowing the cellularity of the ascitic fluid of these individuals, fact that was not assessed in this study)³⁹⁻⁴¹.

In the United States, Desai *et al.*²⁶ examined 55 patients with SBP and observed 40% of positive cultures, with 20% of gram-negatives and 5.5% of positive cultures for more than one microorganism. Singh *et al.*²⁹ assessed 61 individuals with SBP, with 42 positive cultures: 48% gram-positives, 40% gram-negatives and *Candida species* in 12% of the cases. With regard to the found bacteria, it was detected 21.4%

of *Escherichia coli*, 16.7% of *Enterococcus faecalis*, 14.3% of *Streptococcus viridans*, 11.9% of *Staphylococcus aureus*, 7.1% of *Klebsiella pneumoniae* and 4.7% of *Pseudomonas aeruginosa* and, additionally, one positive case of each one of the following germs *Streptococcus pneumoniae*, *Rhodococcus spp.*, *Klebsiella oxytoca*, *Enterobacter cloacae* and *Citrobacter freundii*.

In Mexico, Bobadilla *et al.*⁴² evaluated 31 cases of SBP with 14 positive cultures, being the *Escherichia coli* the most common germ (71.4%), then *Klebsiella pneumoniae* (14.2%), *Pseudomonas aeruginosa* (7.1%), *Streptococcus faecalis* (7.1%) and *Serratia marcescens* (7.1%). In Barcelona, Solà *et al.*³⁷ noticed, on 13 cases, 46.2% of *Escherichia coli*, 23.1% of *Pneumococcus*, 15.4% of *Klebsiella pneumoniae*, 7.7% of *Streptococcus viridans* and 7.7% of *Staphylococcus aureus*.

In a Pakistani research, carried out by Kamani *et al.*³⁴, among 44 individuals with SBP, 14.9% presented positive blood cultures and 23.5% presented positive cultures of ascitic fluid, and 72.7% of them gram-negative. *Escherichia coli* was the most commonly found microorganism (61.3%), followed by *Streptococcus pneumoniae* (11.3%), *Pseudomonas species* (9.0%), *Staphylococcus species* (6.8%), *Enterococcus species* (6.8%), *Bacillus species* (2.2%) and Group D *Streptococcus* (2.2%).

In Egipt, Abd Elaal *et al.*³⁶ evaluated 36 patients with SBP, among which 12 patients presented positive culture in the ascitic fluid. The most commonly found germs in the study were *Escherichia coli* (75.0%), *Streptococcus faecalis* (16.6%), and *Klebsiella pneumoniae* (8.3%).

In Korea, in a study conducted in the city of Seul, Kim *et al.*³⁵ evaluated 130 patients with diagnosis of SBP, among them, 37 (28.5%) presented positive culture of ascitic fluid. The majority of the samples collected were of enteric gram-negatives,

Escherichia Coli (62.1%). Other germs were also found, respectively: *Aeromonas* (13.5%), *Streptococcus* (10.8%), *Klebsiella pneumoniae* (8.1%) , and *Pseudomonas* in 5.4% of the positive samples. In another local study, Song *et al.*⁴³ compared the infections of ascitic fluid acquired in the community *versus* those acquired in the hospital. From October 1998 to August 2003, a total of 106 patients with positive cultures of ascitic fluid were studied, they discovered that 32 cases of SBP were caused by in-hospital focus and 74 were acquired in the community. Gram-negative bacilli, such as *Escherichia coli*, predominated in both groups (community and in-hospital). In 58.5% of the total samples, *Escherichia coli* was detected as agent causing the SBP, then *Klebsiella Pneumoniae* in 11.3% of the cases. Germs, such as *Streptococcus pneumoniae* (7.5%), others species of *Streptococcus* (7.5%), *Enterococco* (5.6%), *Pseudomonas aeruginosa* (1.9%), *Acinetobacter baumannii* (5.6%) and *Aeromonas hydrophila* (1.9%) were also isolated.

In the Dutch city of Rotterdam, from June 1987 to April 1991, Siersema *et al.*³⁸ compared two methods most often used for culture of ascitic fluid: vials of blood culture and conventional method of culture. In this period, 31 suspect cases of SBP were diagnosed on 28 patients. By employing the conventional method of culture, the samples of ascitic fluid showed positive results on 11 of the 31 cases of SBP (35%) against 26 of the 31 cases (84%) by using the vials for blood cultures. In every sample in which the bacterial growth didn't happen while using the conventional method, it also didn't happen while using the method of blood. From 26 positive cultures, the gram-negative bacilli were detected in 17 cases (65%) and the gram-positive cocci were detected in 9 cases (35%). The same study isolated in 38.5% of cultures *Escherichia coli*, *Klebsiella Pneumoniae* (7.7%), *Pseudomonas aeruginosa* (7.7%), *Enterobacter cloacae* (3.8%), *Acinetobacter sp.* (3.8%), not specified *Gram-negative*

(3.8%), *Streptococcus Alpha-hemolytic sp.*(7.7%), *Enterococcus faecalis* (7.7%), *Streptococcus pneumonia* (11.5%), *Staphylococcus aureus* (3.8%) and *Staphylococcus epidermidis* (3.8%).

During 20 year, in France, Dupeyron *et al.*⁴⁴ assessed a total of 240 cases of SBP in the period from April 1977 to April 1997 in order to verify changes in the microbiological profile of the agents that cause the SBP. At the end of the study, the majority of the ascitic fluid's infections were caused by enterobacteria, with no significant change in the percentage when compared to the initial period at the end of the study. The prevalence of *Escherichia Coli* in 43.3% of the cases was noted, as well as *Klebsiella pneumoniae* (10%), Groupe D *Estreptococcus* (7.6%), *Enterococcus faecalis* (6.7%), *Serratia marcescens* (4.6%), *Staphylococcus aureus* (4.6%), *Enterobacter cloacae* (2.5%), *Streptococcus pneumoniae* (3.3%), *Streptococcus* (2.6%), *Streptococcus pneumoniae* (3.3%), among other agents. We also found in smaller quantity germs such as: *Morganella morganii* (0.8%), *Citrobacter freundii* (0.8%), *Providencia stuartii* (0.4%), *Pseudomonas aeruginosa* (1.3%), *Streptococcus pyogenes*(0.8%) *Streptococcus agalatae* (0.8%), *Enterococcus avium* (0.4%), *Enterococcus faecium* (0.4%), *Staphylococcus coagulase negativo* (2.9%), *Listeria monocytogenes* (0.8%), *Bacterioides* (2.1%), *Clostridium* (1.7%), *Candida albicans* (0.4%), *Candida glabrata* (0.4%), *Fusobacterium* (0.4%), *Aerococcus viridans* (0.4%).

In São Paulo, Reginato *et al.*²⁵, 219 patients assessed with SBP and, among 123 individuals submitted to culture, 63 (33.8%) had positive culture results. As microbiological profile, it was described 31.7% of *Escherichia coli*, 7.9% of *Streptococcus pneumoniae*, 7.9% of *Staphylococcus aureus* and 7.9% of *Klebsiella pneumoniae*. Almeida *et al.*⁴⁵, in the state of Rio Grande do Sul, have evaluated

retrospectively cirrhotic individuals with SBP whose culture of ascitic fluid was positive, during two distinct periods: 1997-1998 and 2002-2003. In the first period (1997-1998) 33 cases were included, and 3 of them (9 %) were with polymicrobial infection. The most frequent bacteria were: *Escherichia coli* in 13 (36.1%) cases, Coagulase negative Staphylococci in 6 (16.7%), *Klebsiella pneumoniae* in 5 (13.9%), *Staphylococcus aureus* in 4 (11.1%) and *Streptococcus faecalis* in 3 (8.3%). From 2002 to 2003, there were 43 cases, and 2 (5.0 %) of them were with polymicrobial infection. The most frequent bacteria were: Coagulase negative Staphylococci in 16 (35.6%), *Staphylococcus aureus* in 8 (17.8%), *Escherichia coli* in 7 (15.6%) and *Klebsiella pneumoniae* in 3 (6.7%). There was a modification in the bacterial population that caused spontaneous bacterial peritonitis on the different periods analyzed, with predominance of gram-negative in the first period and gram-positive in the second.

The cultures of ascitic fluid that were positive for multiple germs may suggest, besides the contamination, the presence of secondary peritonitis to intestinal perforation. For diagnostic elucidation of these cases, evaluations with imaging tests are indicated. In this study, three cirrhotic individuals presented SBP by two germs, and in none of them secondary peritonitis was confirmed⁴⁶.

The samples of individuals with cirrhosis and spontaneous bacterial peritonitis observed in this study exhibits similar characteristics to those described in the literature, not only in relation to the clinical characteristics, but also of hepatic functions and microbial profile of the ascitic fluid. There was a higher prevalence of enterobacteria, fact that reflects the overall characteristics.

THERE ARE NO CONFLICTS OF INTEREST

REFERENCES

- 1- Garcia-Tsao G. Bacterial infections in cirrhosis: treatment and prophylaxis. *J Hepatol* 2005;42:85-92.
- 2- Berg RD. Mechanisms promoting bacterial translocation from the gastrointestinal tract. *Adv Exp Med Biol* 1999; 473:11-30.
- 3- Guarner C, Soriano G. Bacterial translocation and its consequences in patients with cirrhosis. *Eur J Gastroenterol Hepatol* 2005;17: 27-31.
- 4- Moore K. Spontaneous bacterial peritonitis SBP . In Warrel DA et al. *Oxford Textbook of Medicine*, 4th Edition, Oxford University Press 2003, Vol 2, sections 11-17, 739-41.
- 5- Runyon BA. Management of adult patients with ascites caused by cirrhosis. *Hepatology* 1998; 27:264-72.
- 6- Navasa M, Rodes J. Management of ascites in the patient with portal hypertension with emphasis on spontaneous bacterial peritonitis. *Semin Gastrointest Dis* 1997; 8:200-9.
- 7- Guarner C, Soriano G. Spontaneous bacterial peritonitis. *Semin Liver Dis* 1997; 1:203-17.
- 8- Arroyo V, Ginès P, Gerbes AL, Dudley FJ, Gentilini P, Laffig G, Reynolds TB, Ring-Larsen H, Scholmerich J. Definition and diagnosis criteria of refractory ascites and hepatorenal syndrome in cirrhosis. *Hepatology* 1996;23:164-75.
- 9- Sort P, Navasa M, Arroyo V, Aldeguer X, Planas R, Ruiz-del-Arbol L, Castells L, Vargas V, Soriano G, Guevara M, Ginès P, Rodés J. Effect of intravenous albumin on

renal impairment and mortality in patients with cirrhosis and spontaneous bacterial peritonitis. *N Engl J Med* 1999;341:403-9.

10- Toledo C, Salmeron JM, Rimola A, Navasa M, Arroyo V, Llach J, Ginès A, Ginès P, Rodés J. Spontaneous bacterial peritonitis in cirrhosis: Predictive factors of infection resolution and survival in patients treated with cefotaxime. *Hepatology* 1993;17:251-7.

11- Genuit T, Napolitano L. Peritonitis and abdominal sepsis. *EMedicine*, 2004.

12- Hoefs JC, Canawati HN, Sapico FL, Hopkins RR, Weiner J, Montgomerie JZ. Spontaneous bacterial peritonitis. *Hepatology* 1982;2:399-407.

13-Levison ME, Bush LM. Peritonitis and intraperitoneal abscesses. In Mandell GL, Bennett JE, Dolin R. *Principles and Practice of Infectious Diseases*. 6th Edition, Elsevier, Churchill Livingstone, Philadelphia, 2005, Vol 1, 927- 51.

14- Such J, Runyon BA. Spontaneous bacterial peritonitis. *Clin Infect Dis* 1998;27:669-74.

15- Parsi MA, Atreja A, Zein NN. Spontaneous bacterial peritonitis: recent data on incidence and treatment. *Cleve Clin J Med* 2004;71:565-69.

16- Angeloni S, Leboffe C, Parente A, Venditti M, Giordano A, Merli M, Riggio O. Efficacy of current guidelines for the treatment of spontaneous bacterial peritonitis in the clinical practice. *World J Gastroenterol* 2008;14:2757-62.

17- Guarner C, Soriano G. Bacterial translocation and its consequences in patients with cirrhosis. *Eur J Gastroenterol Hepatol* 2005;17:27-31.

18- Runyon BA. Management of adult patients with ascites due to cirrhosis. *Hepatology* 2004;39:841-56.

19-Wiest R, Garcia-Tsao G. Bacterial translocation in cirrhosis. *Hepatology* 2005;41:422-33.

20 – Strauss E, Caly WR. [Spontaneous bacterial peritonitis]. *Rev Soc Bras Med Trop* 2003;36(6):711-7.

21- Ginès P, Cárdenas A, Arroyo V, Rodés J. Management of cirrhosis and ascites. *N Engl J Med* 2004; 350: 1646-54.

22- Thalheimer U, Triantos CK, Samonakis DN, Patch D, Burroughs AK. Infection, coagulation and variceal bleeding in cirrhosis. *Recent Advances in Clinical Practice. Gut* 2005; 54: 556-63.

23- Fernandez J, Navasa J, Colmenero J, et al. Bacterial infections in cirrhosis: epidemiological changes with invasive procedures and norfloxacin prophylaxis. *Hepatology* 2002; 35:140-8.

24- Kamath PS, Wiesner RH, Malinchoc M, et al. A model to predict survival in patients with end-stage liver disease. *Hepatology* 2001;33:464-470.

25- Reginato TJ, Oliveira MJ, Moreira LC, Lamanna A, Acencio MM, Antonangelo L. Characteristics of ascitic fluid from patients with suspected spontaneous bacterial peritonitis in emergency units at a tertiary hospital. *Sao Paulo Med J.* 2011;129:315-9.

26- Desai AP, Reau N, Reddy KG, Te HS, Mohanty S, Satoskar R, Devoss A, Jensen D. Persistent spontaneous bacterial peritonitis: a common complication in patients with spontaneous bacterial peritonitis and a high score in the model for end-stage liver disease. *Therap Adv Gastroenterol.* 2012;5:275-83.

27- Shi KQ, Fan YC, Ying L, Lin XF, Song M, Li LF, Yu XY, Chen YP, Zheng MH. Risk stratification of spontaneous bacterial peritonitis in cirrhosis with ascites based on classification and regression tree analysis. *Mol Biol Rep.* 2012;39:6161-9.

28- Mohan P, Venkataraman J. Prevalence and risk factors for unsuspected spontaneous ascitic fluid infection in cirrhotics undergoing therapeutic paracentesis in an outpatient clinic. *Indian J Gastroenterol.* 2011;30:221-4.

- 29- Singh N, Wagener MM, Gayowski T. Changing epidemiology and predictors of mortality in patients with spontaneous bacterial peritonitis at a liver transplant unit. *Clin Microbiol Infect.* 2003;9:531-7.
- 30- Shaw E, Castellote J, Santín M, Xiol X, Euba G, Gudiol C, Lopez C, Ariza X, Gudiol F. Clinical features and outcome of spontaneous bacterial peritonitis in HIV-infected cirrhotic patients: a case-control study. *Eur J Clin Microbiol Infect Dis.* 2006; 25:291–298.
- 31- Deschenes M, Villeneuve JP. Risk factors for the development of bacterial infections in hospitalized patients with cirrhosis. *Am J Gastroenterol* 1999;94:2193–7.
- 32- Runyon BA. Low-protein-concentration ascitic fluid is predisposed to spontaneous bacterial peritonitis. *Gastroenterology* 1986;91:1343–6
- 33- Obstein KL, Campbell MS., Reddy KR, Yang YX. Association between model for end- stage liver disease and spontaneous bacterial peritonitis. *Am J Gastroenterol* 2007;102: 2732–6.
- 34- Kamani L, Mumtaz K, Ahmed US, Ali AW, Jafri W. Outcomes in culture positive and culture negative ascitic fluid infection in patients with viral cirrhosis: cohort study. *BMC Gastroenterol* 2008;8:59.
- 35- Kim SU, Chon YE, Lee CK, Park JY, Kim do Y, Han KH, Chon CY, Kim S, Jung KS, Ahn SH. Spontaneous bacterial peritonitis in patients with hepatitis B virus-related liver cirrhosis: community-acquired versus nosocomial. *Yonsei Med J* 2012;53:328-36.
- 36- Abd Elaal MM, Zaghloul SG, Bakr HG, Ashour MA, Abdel-Aziz-El-Hady H, Khalifa NA, Amr GE. Evaluation of different therapeutic approaches for spontaneous bacterial peritonitis. *Arab J Gastroenterol* 2012 ;13:65-70.

- 37- Solà R, Andreu M, Coll S, Vila MC, Oliver MI, Arroyo V. Spontaneous bacterial peritonitis in cirrhotic patients treated using paracentesis or diuretics: results of a randomized study. *Hepatology* 1995;21:340-4.
- 38- Siersema PD, de Marie S, van Zeijl JH, Bac DJ, Wilson JH. Blood culture bottles are superior to lysis-centrifugation tubes for bacteriological diagnosis of spontaneous bacterial peritonitis. *J Clin Microbiol* 1992;30:667-9.
- 39- Rimola A, Garcia-Tsao G, Navasa M, Piddock LJ, Planas R, Bernard B, Inadomi JM. Diagnosis, treatment and prophylaxis of spontaneous bacterial peritonitis: a consensus document. *J Hepatol* 2000;32:142-53.
- 40- Castellote J, López C, Gornals J, Tremosa G, Fariña ER, Baliellas C, Domingo A, Xiol X. Rapid diagnosis of spontaneous bacterial peritonitis by use of reagent strips. *Hepatology* 2003;37:893-6.
- 41- Runyon BA. Strips and tubes: refining the diagnosis of spontaneous bacterial peritonitis. *Hepatology* 2003;37:745-7.
- 42- Bobadilla M, Sifuentes J, Garcia-Tsao G. Improved method for bacteriological diagnosis of spontaneous bacterial peritonitis. *J Clin. Microbiol* 1989;27:2145-7.
- 43- Song JY, Jung SJ, Park CW, Sohn JW, Kim WJ, Kim MJ, Cheong HJ. Prognostic significance of infection acquisition sites in spontaneous bacterial peritonitis: nosocomial versus community acquired. *J Korean Med Sci* 2006;21:666-71.
- 44- Dupeyron C, Campillo B, Mangeney N, Richardet JP, Leluan G. Changes in nature and antibiotic resistance of bacteria causing peritonitis in cirrhotic patients over a 20 year period. *J Clin Pathol* 1998;51:614-6.
- 45- Almeida PR, Camargo NS, Arenz M, Tovo CV, Galperim B, Behar P. Spontaneous bacterial peritonitis: impact of microbiological changes. *Arq Gastroenterol* 2007;44:68-72.

46- Akriviadis EA, Runyon BA. The value of an algorithm in differentiating spontaneous from secondary bacterial peritonitis. *Gastroenterology* 1990;98:127–33.

Table 1. Microbial profile of 47 individuals with positive ascitic fluid cultures at the University Hospital Polydoro Ernani de São Thiago from January 2008 to December 2011 (P=0.326).

Germ	Total		Cirrhosis		Non Cirrhosis	
	n = 47		n = 26		n = 21	
	n	%	n	%	n	%
<i>Escherichia coli</i>	12	25.5	5	19.2	7	33.3
<i>Klebsiella pneumoniae</i>	7	14.9	5	19.2	2	9.5
<i>Enterococcus</i>	4	8.5	2	7.7	2	9.5
<i>Streptococcus</i>	4	8.5	4	15.4	0	0.0
<i>Staphylococcus aureus</i>	5	10.6	4	15.4	1	4.8
<i>Acinetobacter baumannii</i>	2	4.3	1	3.8	1	4.8
<i>Enterobacter cloacae</i>	1	2.1	1	3.8	0	0.0
<i>Serratia marcescens</i>	1	2.1	1	3.8	0	0.0
<i>E. coli</i> + <i>Enterococcus</i>	2	4.3	0	0.0	2	9.5
<i>E. coli</i> + <i>Klebsiella</i>	1	2.1	0	0.0	1	4.8
<i>E. coli</i> + <i>Acinetobacter baumannii</i>	1	2.1	1	3.8	0	0.0
<i>Enterobacter Clocae</i> + <i>Candida albicans</i>	1	2.1	0.0	0.0	1	4.8
<i>Enterobacter Clocae</i> + <i>Pseudomonas</i>	1	2.1	1	3.8	0	0.0
<i>Enterococcus faecalis</i> + <i>Pantora spp.</i>	1	2.1	0	0.0	1	4.8
<i>Enterococcus</i> + <i>Staphylococcus</i>	1	2.1	1	3.8	0	0.0
<i>Klebsiella</i> + <i>Enterococcus</i>	1	2.1	0	0.0	1	4.8
<i>Klebsiella</i> + <i>Pseudomonas</i>	1	2.1	0	0.0	1	4.8
<i>E. coli</i> + <i>Kleibisiella</i> + <i>Enterococcus</i>	1	2.1	0	0.0	1	4.8

Table 2. Distribution of clinical and laboratorial variables concerning 47 individuals with positive ascitic fluid cultures in relation to the presence of cirrhosis.

Characteristics	Total n = 47	Cirrhosis n = 26	Non Cirrhosis n = 21	P
Age (years) [§]	55.7 ± 15.5	56.6 ± 11.6	54.3 ± 16.8	0.380 ^t
Males (%)	70.2	84.6	52.4	0.016^q
Hospital Stay (d) [#]	15.0	15.0	15.0	0.323 ^m
Hospital Stay ≥ 48 h (%)	89.4	84.6	95.2	0.362 ^f
Two or more germs (%)	25.5	15.4	38.1	0.076 ^q
HbsAg-positive (%)	50.0	52.4	52.9	1.000 ^f
anti-HCV-positive (%)	46.7	56.5	14.3	0.086 ^f
anti-HIV-positive (%)	9.1	0.0	9.1	0.091 ^f
Creatinine (mg/dL) [#]	1.3	1.3	2.0	0.433 ^m
Hemoglobin (g/dL) [#]	10.2	10.5	9.5	0.864 ^m
Platelets (/mm ³) [#]	147,000.0	109,500.0	247,000.0	0.001^m
AST (xULN) [#]	1.8	2.0	1.0	0.021^m
ALT (xULN) [#]	0.8	0.9	0.7	0.390 ^m
AP (xULN) [#]	1.1	1.2	1.3	0.805 ^m
GGT (xULN) [#]	1.6	1.0	2.4	0.121 ^m
Serum albumin (g/dL) [§]	2.0 ± 0.7	2.1 ± 0.6	2.1 ± 0.8	0.900 ^t
Bilirubin (mg/dL) [#]	2.3	2.7	0.7	0.008^m
PA (%) [§]	46.2 ± 18.3	42.7 ± 16.4	50.9 ± 20.7	0.107 ^t

AST = aspartate aminotransferase; ALT = alanine aminotransferase; AP = alkaline phosphatase; GGT = gamma glutamil transferase; xULN = times upper limit of normal; PA = prothrombin activity; AF = ascitic fluid; HbsAg = Hepatitis B surface antigen; Anti-HCV = Antibodies to hepatitis C virus; Anti-HIV = Antibodies to human immunodeficiency virus; [§]Mean; ± standard deviation; [#]median; ^tStudent's *t* Test; ^mMann-Whitney, ^qChi-square test; ^fFisher's exact test.

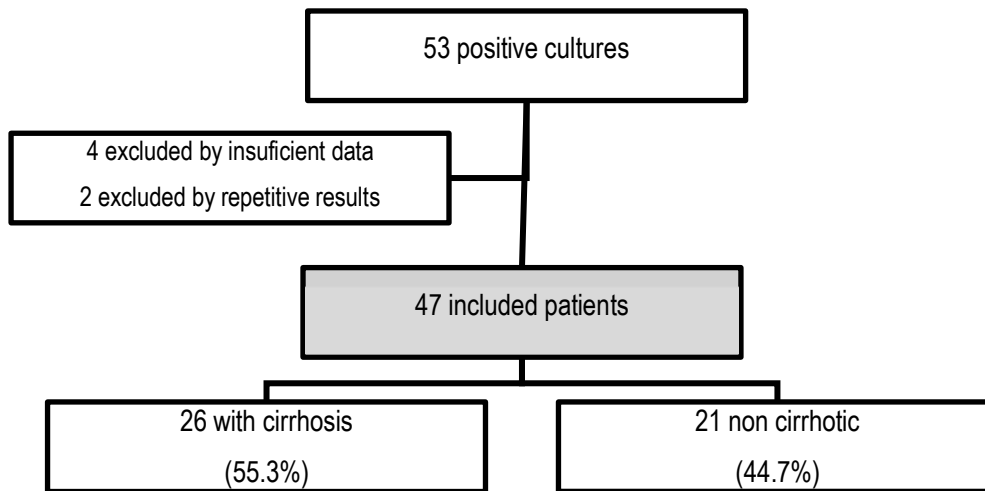


Figure 1. Distribution of patients assessed for this study

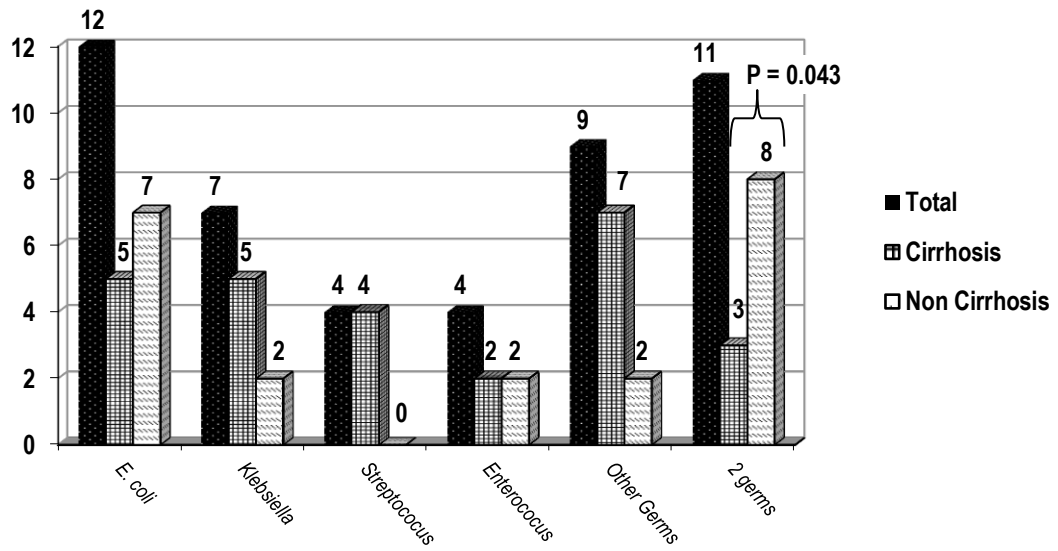


Figure 2. Distribution of germs that caused ascitic fluid infection on 47 individuals according to the occurrence of cirrhosis

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FINANCIAL DISCLOSURE

Nothing to report