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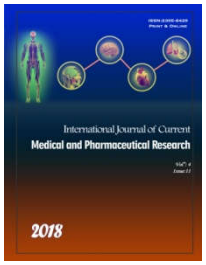

SPONTANEOUS BACTERIAL PERITONITIS: A REVIEW

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ABSTRACT

Liver cirrhosis is one of the commonest conditions which leads to a significant morbidity, multiple hospitalisation and mortality throughout the world. Spontaneous Bacterial Peritonitis (SBP) is one of the prevalent conditions associated with liver cirrhosis and as cites increasing its mortality several folds. These patients are prone to multiple infections owing to subdued immune response. This is due to deficient complement system, and diminished activity of neutrophilic and reticuloendothelial systems. This poor immune system leads to development of SBP and also responsible for its recurrence. The risk rises with previous episode of SBP, low ascitic fluid protein concentration (less than 1.5g/dL), high serum bilirubin (above 2.5 mg/dL) along with impaired renal functions and with gastrointestinal hemorrhage. The most common microbes found in the ascitic fluid are gram-negative aerobic bacteria and most common isolates are Escherichia coli (E. coli), Klebsiella pneumoniae and the pneumococci. Third generation cephalosporins are the first line agent to treat SBP. Other antimicrobial agents are amoxicillin/clavulanate, ciprofloxacin etc. Prophylactic therapy play a crucial role in decreasing the chances of infection and themorbidity and morbidity associated with this condition. The prophylactic antimicrobial therapy aims to decrease the bacterial contamination of the gut and prevent the seepage of bacteria from the gut to the ascitic fluid. Norfloxacin is most commonly prescribed antimicrobial therapy as lifelong prophylaxis in SBP. Trimethoprim-Sulfamethoxazole is yet another alternative. In view of such high recurrence and morbidity, early diagnosis, treatment and prophylaxis play a crucial role in determining the prognosis of SBP.

INTRODUCTION

Chronic liver diseases (CLD) cause is one of the rising morbid condition in the current scenario. It accounts for significant morbidity and mortality worldwide.[1]The mortality is more in the low and low-middle income countries of Asia and Africa. The data reported could be an underestimation of the disease burden as these nations have very deficient reporting systems.[2,3] India is one of the countries undergoing demographic and epidemiologic transition and has a huge burden of patients with liver disease. With the increased prevalence of the disease comes its complications. Spontaneous Bacterial peritonitis (SBP) is one of the commonest complications in patients of liver cirrhosis with ascites. SBP is defined as an infection of ascitic fluid without an evident detectable intra-abdominal surgically treatable source of infection.[4] It often develops insidiously and may remain unrecognized most of the times but with time gets quite evident with the deteriorating condition of the patient. In the patients with ascites, the probability of development of SBP ranges from 10% to 25% within a year.[5] The prevalence of SBP at hospital admission ranges from 10% to 27%.[6,7] The inpatient mortality rates are quite high and various studies have reported a range of 20% to 40%.[8] In view of such high mortality, the survival of a patient with SBP depends on an aggressive approach to diagnosis and treatment. After the first hospitalization of a patient, one-year and two-year mortality rates for those with SBP are approximately about 70% and 80% respectively which is due to the fact that, recurrence of this debilitating condition is very common ranging from 40-70% within the first year.[9] Mortality in western countries has been reported as 15.45% to 50% whereas, in India ranges from 27.2% to 43%.[10,11]In light of such a high recurrence rate and mortality, prophylaxis plays a paramount role in the prevention of recurrences and relapse of this highly morbid condition.

The following patients are identified to have a greater risk for SBP:[12,13]

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1) Any previous episode of SBP 2) A low ascitic fluid protein concentration (less than 1.5g/dL) 3) High serum bilirubin (above 2.5 mg/dL) along with impaired renal functions 4) Patients with gastrointestinal hemorrhage.

The cirrhotic patients with ascitic fluid protein concentrations below 1 g/dL are almost 10 times more prone to develop SBP than individuals with higher concentrations. It is thought that the antibacterial or opsonic activity of ascitic fluid is closely correlated with the ascitic fluid protein concentration.[14] On the contrary, patients with ascitic fluid of typically high protein content, such as patients with malignant ascites or congestive heart failure, are relatively resistant to SBP.[15,16] Literature search have shown the ascitic fluid protein concentration as the best and reliable predictor of the first episode of SBP.[6,16,17]Elevated serum bilirubin levels is also associated with high risk of SBP and conventionally it coexists with advanced stage of liver disease. As per Child-Pugh criteria, serum bilirubin is one of five markers used to map the severity of liver disease.[18] It is very evident from the literature that about 70% of the cases of SBP are seen in patients who are with Child-Pugh class C cirrhosis.[19] Patients suffering from severe acute or chronic liver disease are mostly deficient in complement and have a very subdued immunological system and perhaps also have suppressed neutrophilic and reticuloendothelial systems.[20,21,22]SBP is thought to be as a result from a plethora of factors responsible for cirrhosis and ascites, such as prolonged bacteremia secondary to compromised host defenses, intrahepatic shunting of colonized blood, and defective bactericidal activity within the ascitic fluid.[16,23] Bacteria those are responsible for SBP, mostly come from the digestive tract. Microbes responsible for extraintestinal infection such as those from the respiratory tract, urogenital tract or skin are comparatively less frequent.[24,25] Catheters, intravenous cannula and other equipment those are used during invasive procedures can be another plausible source of infection to the ascitic fluid. It is currently proposed that these enteric organisms cross the intestinal mucosal barrier and enters mesenteric lymph nodes. SBP follows an episode of bacteremia during which microbes enter the systemic bloodstream via the thoracic duct leading to bacteremia.[26,27] There is a constant exchange of fluids between the peritoneal and intravascular space, the ascitic fluid gets infected this translocation of bacteria leading to spontaneous bacterial peritonitis.[25,28]

Pathogenesis of SBP

Pathogenesis of SBP is hypothesized to be as follows:

The four key facets of SBP pathogenesis:

1. Bacterial small intestinal overgrowth,
2. Increased intestinal permeability,
3. Bacterial translocation, and
4. Immunosuppression.

These key elements above do not work independently but work in collaboration with each other to cause the condition.[27]

Small intestinal bacterial overgrowth- Liver cirrhosis is one of the conditions which are very commonly associated and culpable for small intestinal bacterial overgrowth. The cardinal reasons for Bacterial small intestinal overgrowth in patients affected by liver cirrhosis and ascites can be attributed to a reduced intestinal passage, an aberration in bile secretion, hypochlorhydria, an aberration in IgA production and malnutrition.[27,29] The gastrointestinal tract is an ocean of microorganism and among those innumerable bacterias found in, only a few of them habitually participate in the bacterial translocation. The most commonly associated bacteria are E. coli, Proteus spp., K. pneumoniae and other Enterobacteriaceae, Pseudomonas aeruginosa, Enterococci, Streptococci and Staphylococci i.e. these are the organisms which are mostly associated with infections in an immunocompromised host. It has been seen that there is an overgrowth of bacteria in the small intestine and this bacterial overgrowth nurture an environment conducive for translocation of the pathogenic bacteria.[27,30], On the contrary there is another school of thought which advocates bacterial overgrowth is mostly associated with the drugs those decrease the acid secretion in the stomach and it is hypothesized that this can amplify the risk of SBP.[31]

Increased intestinal permeability

In liver cirrhosis and ascites, the small intestinal motility is significantly impaired, which results into constipation culminating into bacterial overgrowth ultimately leading to translocation of microbes through the damaged mucosal barrier.[27] Portal hypertension results into dilatation of the vessels in the intestinal mucosa, edema of the lamina propria mucosae and degenerated intestinal mucosa. literature search has shown that increased intestinal permeability can be consistent with the degree of portal hypertension but lacks any direct linkage with the severity and etiology of liver disease.[32]

Bacterial translocation

Bacterial translocation is defined as migration of living microorganisms and their toxic products through the mucosal epithelial layer to the lamina propria mucosae by either active or passive means.[27,33] Microbes further migrate to mesenteric lymph nodes and other extraintestinal sites. The bacteria in lamina propria mucosae are swiftly killed by the competent immune system innormal healthy human and only possible if the bacterial count is high, up to 10^8 bacteria in 1 g of faeces.[33]If the microbes survive the complement system then it ultimately gets into the blood.

Immunosuppression

With the severity of the liver cirrhosis, the patients have a very subdued immunity with decreased phagocytic activity, dampened humoral immunity and opsonin activity of ascitic fluid.[34] Neutrophils and phagocytic cells in these patients have comparatively decreased chemotaxis.[35]The concentration of immunoglobulins, complement and total protein in ascitic fluid have a very strong correlation with the competency of ascitic fluid to avert infection and low ascitic fluid protein levels is an independent risk factor for the development of SBP.[14,27,36]

Types of SBP

SBP can be of different types like: [37,38]

1. Spontaneous bacterial peritonitis is defined as positive bacterial finding in ascites, with increased polymorphonuclear leukocytes in ascites (> 250 cells/mm3).[39] In this classic case of SBP, culpable bacteria are isolated in only 60%-70% of cases.
2. Culture-negative neutrocytic ascites (CNNA) -In this type, ascitic fluid seems to be sterile, and bacterial culture is negative. Whereas, polymorphonuclear leukocytes more than 250 cells/mm3 can be demonstrated.

3. Monomicrobial non-neutrocytic bacterascites- In this type positive bacterial culture without increased leukocytes is demonstrated.

Clinical Presentation

SBP has a very varied clinical presentation. It can be insidious or can rapidly present as a sepsis syndrome with a high mortality.[4]Patients with SBP may have local symptoms or signs of peritonitis like abdominal pain, abdominal tenderness, vomiting, diarrhea, ileus or can present with signs of systemic inflammation like hyper or hypothermia, chills, altered white blood cell count, tachycardia, and tachypnea with signs of worsening of liver function, hepatic encephalopathy, shock, renal failure, and gastrointestinal bleeding.[39,40]However, it is important to point out that SBP can be asymptomatic and it can manifest only by worsening of symptoms that accompany the course of liver cirrhosis such as an increase in ascites and failure of diuretic therapy, deteriorating encephalopathy, vomiting, etc.[27,41]

Diagnosis

The diagnosis of SBPis primely based on diagnostic paracentesis. Diagnostic paracentesis with leukocyte count isstrictly advocated in all the patients with ascites.

To confirm the diagnosis of SBP, the ascitic fluid absolute polymorphonuclear leukocyte (PMN) count of at least 250 cells/mm3 with a positive ascitic fluid bacterial culture.[42,43] The collection of ascitic fluid sample for diagnosis should be done before the first dose of antimicrobial administration to avert misdiagnosis.[44]

In SBP ascitic fluid culture is positive in about 40% of cases and the most common pathogens which are isolated include Gram-negative bacteria (GNB) like Escherichia coli (E coli)and Gram-positive cocci (mainly streptococcus species and enterococci).[39,45,46] The three most common isolates are Escherichia coli, Klebsiella pneumoniae and the pneumococci. E. coli is the most frequently isolated gram-negative bacteria.[45]

Management

Empirical antibiotic therapy must be initiated immediately after the diagnosis of SBP, without the results of the ascitic fluid culture.[39,46] European Association for the Study of the Liver (EASL) and American Association for the Study of Liver Diseases (AASLD) guidelines recommends cefotaxime, ceftriaxone or any other third-generation cephalosporin as a treatment of choice for suspected SBP.[37,43] Literature search has shown that it can cover up to 95% of the culpable flora and resolution of infection is obtained in about 77-98% of patients.[47,48] A dose of 4 g/day is as effective as a dose of 8 g/day and a 5-day therapy is as effective as a 10-day treatment.[47] After sensitivities are known, the spectrum of coverage can be narrowed down. Cefotaxime with a dosage of 2 g intravenously every 8 hours has been found to have magnificent result in controlling the infection.[43,49] Recently with the widespread use of quinolones as well as frequent hospitalizations and exposure to broad-spectrum antibiotics have led to a change in the spectrum of bacterial flora with more gram-positives and extended-spectrum β-lactamase producing Enterobacteriaceae in recent years.[50,51]Infections with these resistant organisms are associated with a higher mortality. Amoxicillin and clavulanic acid, initiated intravenously then shifted to oral therapy has shown similar results with respect to SBP resolution and mortality, when compared with cefotaxime and that too with a much lower cost.[52]Ciprofloxacin, given either for 7 days intravenously or for 2 days intravenously followed by 5 days orally, results in a similar SBP resolution rate and hospital survival compared with cefotaxime, but with a significantly higher cost. However, switch therapy (i.e., use of intravenous antibiotic initially, followed by oral step-down administration) with ciprofloxacin is more cost-effective than intravenous cefotaxime.[37] Ofloxacin, 400 mg bid for an average of eight days has shown similar results when compared to intravenous cefotaxime in uncomplicated SBP, without any renal compromise, hepatic encephalopathy, gastrointestinal bleeding or shock.[37,43,53] In SBP, a majority of patients suffering from renal failure which could further worsen the scenario with an increased renin angiotensin aldosterone system activity. Administration of intravenous albumin infusion in addition to cefotaxime has been proven to minimize the mortality in these cases.[37,54] It is recommended that albumin infusion should be given to patients with even clinical suspicion of SBP and serum creatinine > 1 mg/dL, blood urea nitrogen > 30 mg/dL, or total bilirubin > 4 mg/dL.[27,37]

Assessment of treatment response

The resolution of SBP is commonly associated with a rapid improvement in the patient's general condition. If there such rapid improvement is not seen, a follow-up paracentesis is highly recommended 48 h after commencement of antibiotics.[55] A fall in the ascitic PMN count of > 25% suggests that the choice of antimicrobial is appropriate.[43] If there is no fall in PMN count, alternative antimicrobial should be considered, either empirically or according to the sensitivity report and the possibility of secondary bacterial peritonitis should be excluded.[43,56]

Prevention of SBP

In view of the high mortality following an episode of SBP, prevention is extremely important. As there is a high chance of relapse of about 40-70 % with a year so prophylaxis has the utmost importance in its prevention and decreasing the mortality rates.[9]

Prophylaxis in SBP is of two types:[13,37,43,56]

1. Primary prophylaxis- the patients who are at a higher risk of SBP, like those with low ascitic fluid total protein (< 1 g/dl or 1.5 g/dl) or those with a gastrointestinal hemorrhage.
2. Secondary prophylaxis - given to patients who had an episode of SBP to prevent recurrence.

As per international Ascites club recommendations EASL[37] and AASLD[43] guidelines

1. Patients who have survived an episode of SBP should receive long-term prophylaxis with daily Norfloxacin 400mg or Trimethoprim-sulfamethoxazole (160/800)mg.
2. In patients with cirrhosis and ascites, long-term use of Norfloxacin or Trimethoprim-sulfamethoxazole can be justified if the ascitic fluid protein <1.5 g/dL. Both of these antimicrobials are to be given once daily for lifelong or continue until resolution of ascites, liver transplantation, or death.[8,39]

Norfloxacin is the most widely and commonly used antimicrobial used for long-term prophylaxis in this setting. Norfloxacin has been recommended for selective intestinal

decontamination because of its poor intestinal absorption and enhanced local activity. Apart from that, it has robust activity against gram-negative facultative aerobes and weak activity against anaerobes.[57]

CONCLUSION

SBP is a highly recurrent and morbid condition associated with patients with liver cirrhosis and ascites. Early diagnosis and treatment play a crucial role in the management and decreasing mortality. Prophylaxis with daily antimicrobial usage decreases the chances of recurrence and morbidity and should be taken without fail to improve the prognosis.

References

1. Schuppan D, Afdhal NH. Liver cirrhosis. Lancet 2008; 371:838-851.
2. Byass P. The global burden of liver disease: a challenge for methods and for public health. BMC medicine 2014;12:159 pmid:25286285
3. Gupta M, Rao C, Lakshmi PV, Prinja S, Kumar R. Estimating mortality using data from civil registration: a cross-sectional study in India. Bull World Health Organ 2016;94(1):10-21 pmid:26769992
4. Such J, Runyon A. Spontaneous bacterial peritonitis. Clin Infect Dis 1998; 27:669-676.
5. Dever JB, Sheikh MY. Review article: spontaneous bacterial peritonitis--bacteriology, diagnosis, treatment, risk factors and prevention. Aliment Pharmacol Ther. 2015;41:1116-31.
6. Andreu M, Sola R, Sitges SA, Alia C, Gallen M, Vila MC *et al*. Risk factors for spontaneous bacterial peritonitis in cirrhotic patients with ascites. Gastroenterology. 1993; 104: 1133-8.
7. Runyon A. Spontaneous bacterial peritonitis: An explosion of information. Hepatology.1988; 8: 171-5.
8. Alaniz C, Regal RE. Spontaneous bacterial peritonitis: a review of treatment options. P T. 2009; 34:204-10.
9. Tito L, Rimola A, Gines P. Recurrence of spontaneous bacterial peritonitis in cirrhosis: Frequency and predictive factors. Hepatology. 1988; 8: 27-31.
10. Lata J, Fejfar T, Krechler T, Musil T, Husova L, Senkyrik M *et al*. Spontaneous bacterial peritonitis in the Czech Republic: Prevalence and aetiology. *Eur J Gastroenterol Hepatol*.2003; 15: 739-43.
11. Jain PA, Chandra SL, Gupta S, Gupta OP, Jajoo NU, Kalantri PS. Spontaneous Bacterial Peritonitis in Liver Cirrhosis with ascites. *J Assoc Phys India*. 1999; 47: 619-21.
12. Andreu M, Sola R, Sitges SA, Alia C, Gallen M, Vila MC *et al*. Risk factors for spontaneous bacterial peritonitis in cirrhotic patients with ascites. Gastroenterology.1993; 104: 1133-8.
13. Runyon BA. Low-protein-concentration ascitic fluid is pre disposed to spontaneous bacterial peritonitis. Gastroenterology. 1986; 91: 1343-6.
14. Runyon BA. Patients with deficient ascitic fluid opsonic activity are predisposed to spontaneous bacterial peritonitis. Hepatology. 1988; 8: 632-5.
15. Kurtz RC, Bronzo RL. Does spontaneous bacterial peritonitis occur in malignant ascites? *Am J Gastroenterol*. 1982; 77: 146-8.
16. Runyon BA. Spontaneous bacterial peritonitis associated with cardiac ascites. *Am J Gastroenterol*. 1984; 79:796.
17. Llach J, Rimola A, Navasa M. Incidence and predictive factors of first episode of spontaneous bacterial peritonitis in cirrhosis with ascites: Relevance of ascitic fluid protein concentration. Hepatology. 1992; 16: 724-7.
18. Pugh RN, Murray-Lyon IM, Dawson JL. Transection of the esophagus for bleeding esophageal varices. Br J Surgery. 1973; 60: 646-9.
19. Green TE, Bandy SM, Tuttle A. Spontaneous bacterial peritonitis. [Internet] Updated on Jun 15, 2016 [Accessed on 15 Nov 2018] Available from: URL:http://emedicine.medscape.com/article/789105-clinical#b1.
20. Rimola A, Soto R, Bory F, Arroyo V, Piera C, Rodes J. Reticuloendothelial system phagocytic activity in cirrhosis and its relationship to bacterial infections and prognosis. Hepatology.1984; 4: 53-8.
21. Fox RA, Dudley FS, Sherlock S. The serum concentrations of the third component of complement in liver disease. Gut.1971; 12: 574-8.
22. Kourilsky O, LeRoy C, Peltier AP. Complement and liver cell function in 53 patients withliver disease. Am J Med. 1973;55: 78
23. Hoefs JC, Runyon BA. Culture-negative neutrocytic ascites: A variant of spontaneous bacterial peritonitis. Hepatology. 1984;4: 1209-11.
24. Garcia-Tsao G, Albillos A, Barden GE, West AB. Bacterial translocation in acute and chronic portal hypertension. Hepatology. 1993;17: 1081-5.
25. Llovet JM, Bartoli R, March F. Translocated intestinal bacteria cause spontaneous bacterial peritonitis in cirrhotic rats; molecular epidemiologic evidence. *J Hepatol*. 1998; 28:307-13.
26. Fernández J, Bauer TM, Navasa M, Rodés J. Diagnosis, treatment and prevention of spontaneous bacterial peritonitis. Baillieres Best Pract Res Clin Gastroenterol. 2000;14:975-90.
27. Lata J, Stiburek O, Kopacova M. Spontaneous bacterial peritonitis: A severe complication of liver cirrhosis. *World J Gastroenterol*. 2009; 15(44): 5505-10.
28. Runyon BA, Squier S, Borzio M. Translocation of gut bacteria in rats with cirrhosis to mesenteric lymph nodes partially explains the pathogenesis of spontaneous bacterial peritonitis. *J Hepatol*. 1994; 21: 792-6.
29. Ramachandran A, Balasubramanian KA. Intestinal dysfunction in liver cirrhosis: Its role in spontaneous bacterial peritonitis. *J Gastroenterol Hepatol*. 2001; 16: 607-12.
30. Dupeyron C, Mangeney N, Sedrati L, Campillo B, Fouet P. Rapid Emergence of Quinolone Resistance in Cirrhotic Patients Treated with Norfloxacin To Prevent Spontaneous Bacterial Peritonitis. Antimicrob Agents Chemother. 1994 Feb; 38(2): 340-4.
31. Bauer TM, Steinbrückner B, Brinkmann FE, Ditzen AK, Schwacha H, Aponte JJ, Pelz K, Kist M, Blum HE. Small intestinal bacterial overgrowth in patients with cirrhosis: prevalence and relation with spontaneous bacterial peritonitis. *Am J Gastroenterol* 2001; 96: 2962-2967
32. Ersöz G, Aydin A, Erdem S, Yüksel D, Akarca U, Kumanlioglu K. Intestinal permeability in liver cirrhosis. *Eur J Gastroenterol Hepatol* 1999; 11: 409-412

33. Guarner C, Runyon BA, Young S, Heck M, Sheikh MY. Intestinal bacterial overgrowth and bacterial translocation in cirrhotic rats with ascites. *J Hepatol* 1997; 26: 1372-1378
34. Cereto F, Molina I, González A, Del Valle O, Esteban R, Guardia J, Genescà J. Role of immunosuppression in the development of quinolone-resistant Escherichia coli spontaneous bacterial peritonitis and in the mortality of E. coli spontaneous bacterial peritonitis. Aliment Pharmacol Ther 2003; 17: 695-701
35. Fiuza C, Salcedo M, Clemente G, Tellado JM. Granulocyte colony-stimulating factor improves defiient in vitro neutrophil transendothelial migration in patients with advanced liver disease. Clin Diagn Lab Immunol 2002; 9: 433-439
36. Deschénes M, Villeneuve JP. Risk factors for the development of bacterial infections in hospitalized patients with cirrhosis. *Am J Gastroenterol* 1999; 94: 2193-2197
37. European Association for the Study of the Liver; EASL clinical practice guidelines on management of ascites, spontaneous bacterial peritonitis and hepatorenal syndrome in cirrhosis: Spontaneous bacterial peritonitis [Internet].2010. [Accessed on 20 Nov 2018] Available from: http://www.easl.eu /research/our-contributions/clinical-practice guidelines/detail/management-ofascites-spontaneous-bacterial-peritonitis-and-hepatorenal-syndrome-in-cirrhosis/report/4
38. Hoefs JC, Canawati HN, Sapico FL, Hopkins RR, Weiner J, Montgomerie JZ. Spontaneous bacterial peritonitis. Hepatology.1982; 2: 399-407.
39. Rimola A, García-Tsao G, Navasa M, Piddock LJ, Planas R, Bernard B. Diagnosis, treatment and prophylaxis of spontaneous bacterial peritonitis: a consensus document. International Ascites Club. *J Hepatol.* 2000; 32: 142-53.
40. Plessier A, Denninger MA, Consigny Y, Pessione F, Francoz C, Duran F, *et al*. Coagulation disorders in patients with cirrhosis and severe sepsis. Liver Int. 2003; 23:440-8.
41. Evans LT, Kim WR, Poter ucha JJ, Kamath PS. Spontaneous bacterial peritonitis in asymptomatic outpatients with cirrhotic ascites. Hepatology. 2003; 37: 897-901.
42. Dever JB, Sheikh MY. Review article: spontaneous bacterial peritonitis--bacteriology, diagnosis, treatment, risk factors and prevention. Aliment Pharmacol Ther. 2015; 41:1116- 31.
43. Runyon BA. American Association for the Study of Liver Diseases (AASLD) Practice Guidelines Committee. Management of Adult Patients with Ascites Due to Cirrhosis: Update 2012. [Internet].Accessed on 21 Nov 2018 from URL: https://www.aasld.org/sites/default/files/guideline_documents/AASLDPracticeGuidelineAsciteDuetoCirrhosisUpdate2012Edition4_.pdf
44. Recognition and Management of Spontaneous Bacterial Peritonitis: Management of Cirrhosis-Related Complications. Accessed on 21 Nov 2018 from URL: https://www.hepatitisc.uw.edu/go/management-cirrhosis-related-complications/spontaneousbacterial-peritonitis-recognition-management/core-concept/all.
45. Fernandez J, Navasa M, Gómez J, Colmenero J, Vila J, Arroyo V, Rodes J. Bacterial infections in cirrhosis: epidemiological changes with invasive procedures and norfloxacin prophylaxis. Hepatology. 2002; 35: 140-8.
46. Wong F, Bernardi M, Balk R, Christman B, Moreau R, Garcia-Tsao G, *et al*. Sepsis in cirrhosis: report on the 7th meeting of the International Ascites Club. Gut. 2005; 54: 718-25.
47. Rimola A, Salmerón JM, Clemente G, Rodrigo L, Obrador A, Miranda ML, *et al*. Two different dosages of cefotaxime in the treatment of spontaneous bacterial peritonitis in cirrhosis: results of a prospective, randomized, multicenter study. Hepatology. 1995;21: 674-9.
48. Navasa M, Follo A, Llovet JM, Clemente G, Vargas V, Rimola A, *et al*. Randomized, comparative study of oral ofloxacin versus intravenous cefotaxime in spontaneous bacterial peritonitis. Gastroenterology. 1996; 111: 1011-7.
49. Runyon BA, Akriviadis EA, Sattler FR, Cohen J. Ascitic fluid and serum cefotaxime and desacetyl cefotaxime levels in patients treated for bacterial peritonitis. Dig Dis Sci.1991; 36: 1782-6.
50. Fernandez J, Acevedo J, Castro M, Garcia O, Rodriguez de Lope C, Roca D *et al*. Prevalence and risk factors of infections by resistant bacteria in cirrhosis: a prospective study. Hepatology. 2012; 55: 1551-61.
51. Runyon BA. Changing flora of bacterial infections in patients with cirrhosis. Liver Internat. 2010; 30: 1245-6.
52. Ricart E, Soriano G, Novella MT, Ortiz J, Sàbat M, Kolle L, *et al*. Amoxicillin-clavulanic acid versus cefotaxime in the therapy of bacterial infections in cirrhotic patients. *J Hepatol*.2000; 32: 596-602.
53. Navasa M, Follo A, Llovet JM, Clemente G, Vargas V, Rimola A, *et al*. Randomized, comparative study of oral ofloxacin versus intravenous cefotaxime in spontaneous bacterial peritonitis. Gastroenterology. 1996; 111: 1011-7.
54. Sort P, Navasa M, Arroyo V, Aldeguer X, Planas R, Ruizdel-Arbol L, *et al*. 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.
55. Garcia-Tsao G. Current management of the complications of cirrhosis and portal hypertension: variceal hemorrhage, ascites, and spontaneous bacterial peritonitis. Gastroenterology. 2001; 120: 726-48.
56. Mowat C, Stanley AJ. Review article: Spontaneous bacterial peritonitis-diagnosis, treatment and prevention. Aliment Pharmacol Ther. 2001; 15: 1851 -9.
57. Wolfson, J S, Hooper DC. 1988. Norfloxacin: a new targeted fluoroquinolones antimicrobial agent. Ann. Intern. Med. 108: 238-51.

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