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Original Research

Associated Risk Factors and the Laboratory Parameters of Liver Cirrhosis after Spontaneous Bacterial Peritonitis (SBP)

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ABSTRACT:

Aim: To determine the associated risk factors and the laboratory parameters in spontaneous bacterial peritonitis (SBP). Materials and methods-All patients of cirrhosis with ascites underwent diagnostic paracentesis within 24 h of admission or whenever peritonitis is suspected. SBP was diagnosed in the presence of a polymorphonuclear (PMN) cell count ≥ 250 cells/mm3 in peritoneal fluid. SBP was considered as community-acquired when diagnosed at admission or within 48 h of admission. **Results-** All the patients included in our study presented clinically with fever (100%) followed by pain abdomen (95%), abdomen distention (61.66%), encephalopathy (38.33%), reduced urine output (33.33%), jaundice (30%), Haemetemesis (10%) and malena (6.66%).The mean value of PMN cell count in ascetic fluid was 394.63 cells/mm³, total bilirubin was 4.47 mg/dL, Serum Creatinine was 1.53mg/dL, Serum albumin was 2.47 g/dL, INR was 1.85 and Serum sodium was 132.83 mEq/L. The mean values of MELD and iMELD score were 20.63 and 46.28 respectively. **Conclusion-** total bilirubin, serum creatinine, serum sodium, INR, MELD score and iMELD score were found as prognostic factors for prediction of mortality in patients with SBP.

Keywords- Spontaneous Bacterial Peritonitis, Bacterascites, Serum Creatinine, Haemetemesis, Polymorphonuclear Cells.

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INTRODUCTION-

Spontaneous bacterial peritonitis (SBP) is defined by the presence of >250 polymorphonuclear cells (PMN)/mm3 (less accurately at the increase of all leukocytes over 400/mm3) in ascites in the absence of an intra-abdominal source of infection or malignancy.¹ The earliest description of Spontaneous bacterial peritonitis was in 1907-1908. It was first described by Conn and Fessel, as a syndrome of infected ascitic fluid in patients with hepatic cirrhosis.² SBP is the most frequent bacterial infection in cirrhosis, accounting for 10 to 30% of all reported bacterial infections in hospitalised patients.²⁻⁴ In outpatients without symptoms the prevalence is low, but the prevalence increases in the nosocomial setting, ranging from 8% to 36%.⁵ Bacterascites, defined as positive culture results but no increase in the polymorphonuclear cells count in the ascitic fluid, occurs with a prevalence of 2 - 3% in outpatients⁶⁻⁸ and in up to 11% in hospitalized patients.^{9,10} In-hospital mortality for the first episode of SBP ranges from 10% to 50%, depending on various risk factors.

Bacterial translocation is the most common cause of SBP.¹¹⁻¹³ Only a few intestinal bacteria are able to translocate into mesenteric lymph nodes, including Escherichia coli, Klebsiella pneumoniae and other Enterobacteriaceae.¹⁴ Interestingly, these species most frequently cause SBP, and DNA sequencing studies reveal genotypic identity of bacteria in mesenteric lymph nodes and ascites in the vast majority of cases.^{15,16} This suggests that pathological bacterial translocation is the underlying cause and source of SBP in cirrhosis and supports the view that

the route of pathological bacterial translocation leading to SBP is largely lymphatic. The development of SBP thus depends on the antibacterial capacity of ascitic fluid (the so-called opsonin activity) which is positively correlated to the content of the total protein in ascitic fluid and the immunocompetence of the patient. The objectives of the present study was to determine the associated risk factors and the laboratory parameters in spontaneous bacterial peritonitis (SBP).

MATERIALS AND METHODS-

The present study is a prospective observational study and was done in a period of two years. In the present study 60 adult patients both male and female having cirrhosis with Spontaneous bacterial peritonitis (SBP) admitted in the Department of Medicine, Govt. Medical College and Rajindra hospital Patiala were included. The patients were explained in their vernacular language about the procedures to be adopted in the study and their informed consent was taken. The study was approved by the local Ethics Committee, Govt. Medical College and Rajindra hospital Patiala. The diagnosis of cirrhosis was based on clinical, laboratory and imaging findings.

All patients of cirrhosis with ascites underwent diagnostic paracentesis within 24 h of admission or whenever peritonitis is suspected. SBP was diagnosed in the presence of a polymorphonuclear (PMN) cell count ≥ 250 cells/mm3 in peritoneal fluid. SBP was considered as community-acquired when diagnosed at admission or within 48 h of admission. Patients were treated with intravenous cefotaxime or amoxicillin-sulbactam. Piperacillin-tazobactam or carbapenems was used in patients with hospital-acquired SBP or receiving norfloxacin prophylaxis.

STATISTICAL ANALYSIS-

Continuous variables were expressed as mean \pm standard deviation (SD) and categorical variables as count and percentage. Comparisons between groups were performed using Student's t test or Mann-Whitney test for continuous variables and the chi-square test for categorical variables. were considered to be statistically significant if p-value was < 0.05.

RESULTS-

Maximum Number of patients was from age group 51-69 years (68.33%). The mean age of all the 60 patients included in the study was 54.93 years. The maximum and minimum age limit of patient included in our study was 80 years old and 36 years old respectively.(TABLE-1) The most common etiological factor was hepatitis C (50%) followed by Hepatitis C + alcohol (25%), Alcohol (11.6%), others (10%) like Non alcoholic fatty liver disease, and Autoimmune hepatitis Miscellaneous (Hemochromatosis, Wilson disease.

Idiopathic).(GRAPH-1)

All the patients included in our study presented clinically with fever (100%) followed by pain abdomen (95%), abdomen distention (61.66%), encephalopathy (38.33%), reduced urine output (33.33%), jaundice (30%), Haemetemesis (10%) and malena (6.66%).(GRAPH-2) The mean value of PMN cell count in ascetic fluid was 394.63 cells/mm³, total bilirubin was 4.47 mg/dL, Serum Creatinine was 1.53mg/dL, Serum albumin was 2.47 g/dL, INR was 1.85 and Serum sodium was 132.83 mEq/L.(GRAPH-3) The mean values of MELD and iMELD score were 20.63 and 46.28 respectively.(GRAPH-4) Maximum number of patients (45%) had MELD SCORE ranging in between 16 to 20.(GRAPH-5)

The mean age of patients admitted with cirrhosis with SBP who recovered from SBP and discharged was 54.7 ± 9.12 years and those who died was 55 ± 5.6 years. There was no statistically significant difference in patients who survived and died.(graph-6)

There were 24 male patients and 12 female patients admitted with cirrhosis with SBP who recovered from SBP and discharged. 17 male and 7 females died. There was no statistically significant difference in gender of patients who survived and died.(graph-7) The mean Ascitic fluid PMN cell count of patients admitted with cirrhosis with SBP who recovered from SBP and discharged was $340.44\pm$ 22.82 cells/mm³ and those who died was $475.91\pm$ 42.7 cells/mm³. There was statistically significant increase in Ascitic fluid PMN cell count in patients who died.(graph-8) The mean total bilirubin in patients admitted with cirrhosis with SBP who recovered from SBP and discharged was 3.16±2.48mg/dL and those who died was 6.42±5.3 mg/dL. The total serum bilirubin was significantly increased in patients who died as compared to survivors.(graph-9)

The mean creatinine in patients admitted with cirrhosis with SBP who recovered from SBP and discharged was 1.33±0.1 mg/dL and those who died was 1.84±0.25mg/dL. There is statistically significant difference in level of serum creatinine level in patients who died as compared to those who survived.(graph-10) The mean serum albumin in patients admitted with cirrhosis with SBP who recovered from SBP and discharged was 2.51±0.33 g/dL and those who died was 2.4±0.4 g/dL. There was no significant difference in Serum albumin amongst patients who died and who survived.(graph-11) The mean INR in patients admitted with cirrhosis with SBP who recovered from SBP and discharged was 1.76±0.31and those who died was 1.97±0.46. There was significant increase in INR in patients who died as compared to patients who survived.(graph-12)

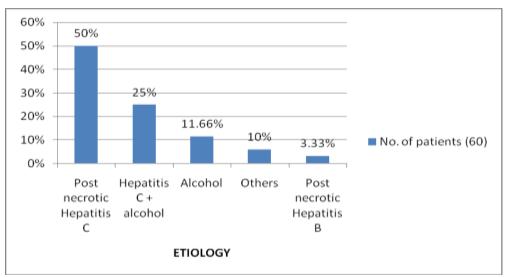
The mean serum sodium in patients admitted with cirrhosis with SBP who recovered

from SBP and discharged was 133.77 ± 3.12 mEq/L and those who died was 131 ± 3.44 mEq/L. There was significant decrease in serum sodium in patients who died as compared to patients who survived.(graph-13) The mean MELD score in patients admitted with cirrhosis with SBP who recovered from SBP and discharged was 17.88 ± 3.17 and those who died was 24.75 ± 4.6 .The patients who died had significantly higher MELD score as compared to patients who survived. (p value 0.0001).(graph-14) The mean iMELD score in patients admitted with cirrhosis with SBP who recovered from SBP and discharged was 44.86 ± 4.18 and those who died was 48.41 ± 4.18 . The patients who died had significantly higher iMELD score as compared to patients who survived (p value 0.0021).(graph-15)

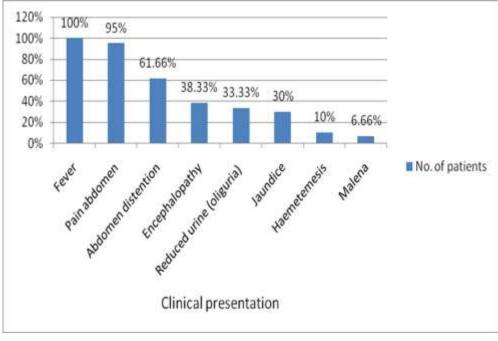
TABLE-1 AGE DISTRIBUTION

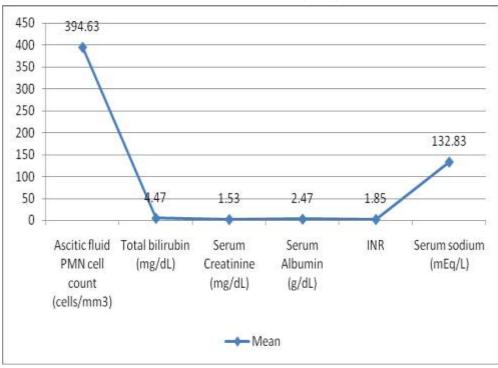
years)		U
20-50 17 (28.33%) 54.93±7.91 79 years 3	20-50	36 years

GRAPH-1 RISK FACTORS



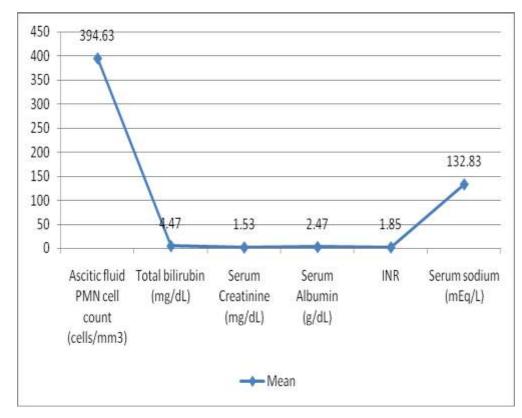
GRAPH 2: CLINICAL PRESENTATION

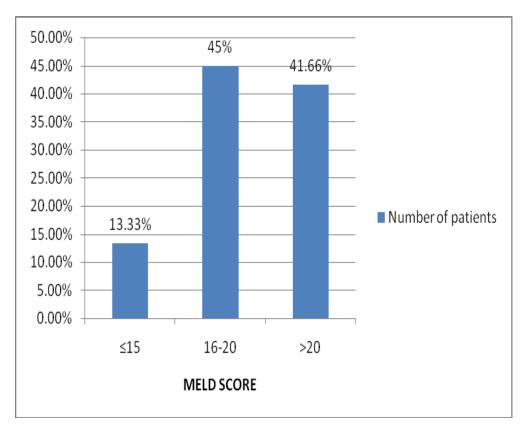




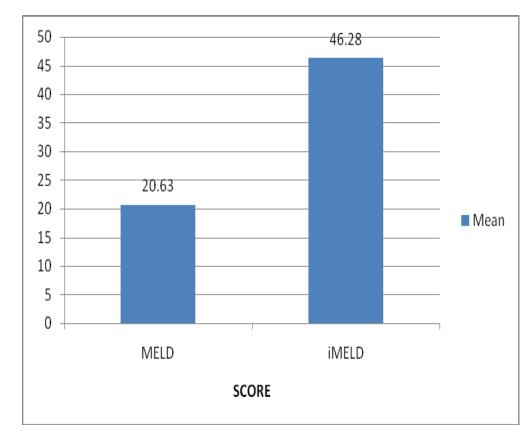
GRAPH 3: LABORATORY DATA OF ALL PATIENTS (N=60)

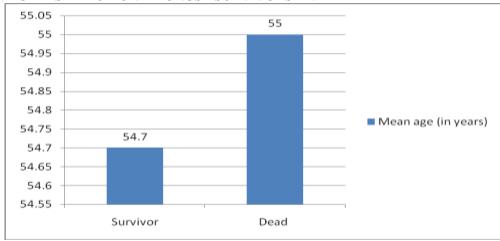
GRAPH 4 MEAN MELD AND iMELD SCORE (N = 60)





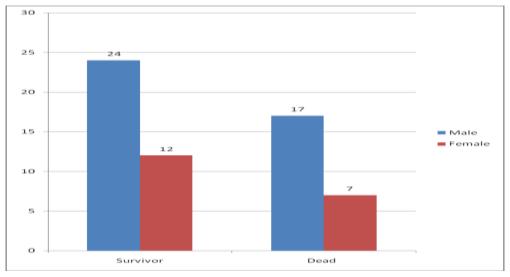
GRAPH 5 : DISTRIBUTION OF MELD SCORE



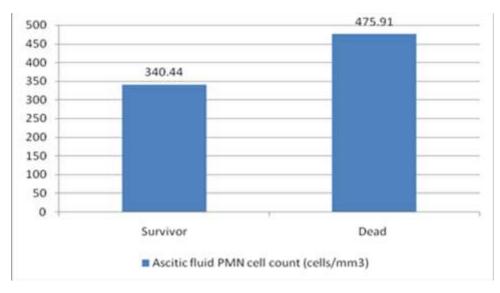


GRAPH 6: AGE DISTRIBUTION AMONGST SURVIVORS AND DEAD

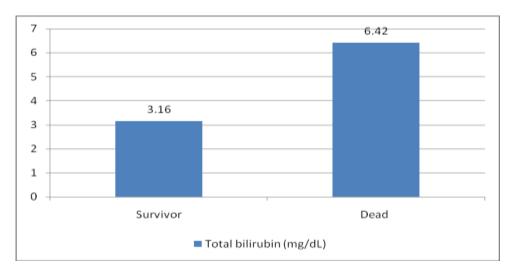




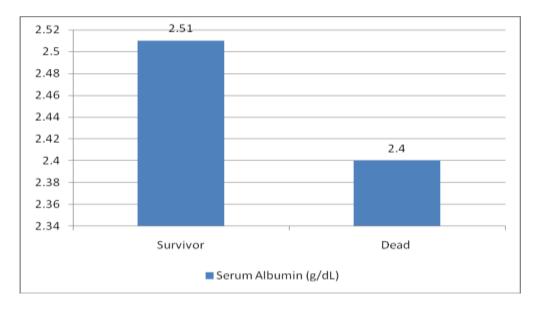




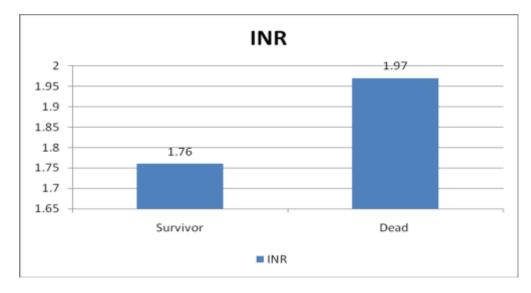
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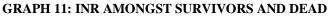


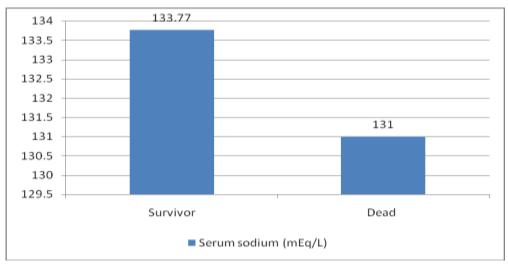




GRAPH 10 SERUM ALBUMIN AMONGST SURVIVORS AND DEAD



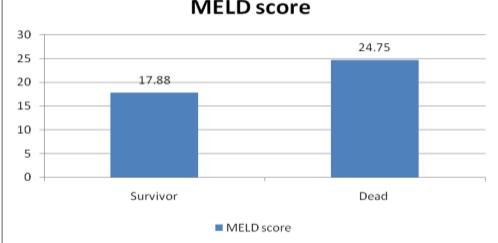




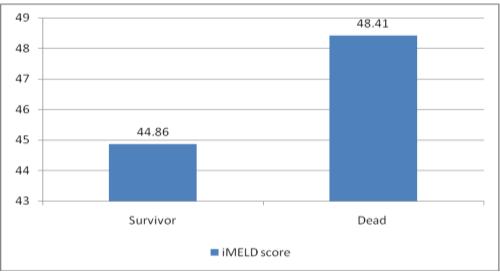
GRAPH 12: SERUM SODIUM AMONGST SURVIVORS AND DEAD



GRAPH 13: MELD SCORE AMONGST SURVIVORS AND DEAD







DISCUSSION-

The study design is a prospective observational study and was done in a period of two years. In the present study 60 adult patients both male and female having cirrhosis with Spontaneous bacterial peritonitis (SBP). In present study laboratory variables included were serum creatinine, total bilirubin, Prothrombin time by international normalized ratio (INR), serum albumin, serum sodium, and ascitic fluid PMN cell count. MELD score was calculated according to the United Network for Organ Sharing (UNOS) formula.¹⁷ For the iMELD score, MELD, age (years) and serum Na (mEq/L) were considered. ¹⁸ The mean age of patients included in our study was 54.93 years. The youngest patient in our study was 36 year old and oldest patient was 79 years old. Mean age at the time of diagnosis in the studies done by Filik L and Unal S¹⁹ was 49.9 years, 39 years in the study done by Rawat N et al²⁰ and 44 years in the study done by Mihas AA et al^{21} . Age distribution was almost similar in all the studies. Amongst 60 patients included in present study majority of patients were males (68.33%) and 31.66% of patients were females. Male to female ratio in our study is 2.15:1. The findings of our study are in concordance with the studies done by Musskopf MI et al²² who in their study of spontaneous bacterial peritonitis reported 65% males and 35% females. The Male to female ratio in their study was 1.8:1. Thilakavathi R et al²³ in their study of cirrhotic ascites reported that spontaneous bacterial peritonitis was present in 17 male patients (80.95%) and 4 female patients (19.05%). The Male to female ratio in their study was 4.2:1. Ding X et al^{24} in their study patients reported 270 males and 64 females. The Male to female ratio in their study was 4.2:1. Thus, there is male preponderance seen in all studies. In a study by Oladimeji AA et al²⁵ there was no gender difference in the occurrence of SBP. This difference in gender distribution could be due to alcoholism being more common in males than females. The most common risk factor in our study was Post necrotic hepatitis C (50%) followed by Hepatitis C + alcohol (25%), Alcohol (11.6%), others etiologies (10%) [like Non alcoholic fatty liver disease, Autoimmune hepatitis Miscellaneous (Hemochromatosis, Wilson and disease, Idiopathic)] and Post necrotic Hepatitis B (3.33%). Gayatri AA et al²⁶ reported that the etiology of liver cirrhosis with spontaneous bacterial peritonitis was chronic hepatitis B in 45.2% and hepatitis C in 23% of patients. Musskopf MI et al²² in their study of spontaneous bacterial peritonitis reported etiological factors as hepatitis C, Hepatitis C + alcohol, Alcohol and Hepatitis B. Kraja B et al²⁷ in their study reported etiological factors for Cirrhotic Patients with Spontaneous Bacterial Peritonitis as alcohol consumption (56.3%), HBV infection (23.4%), HCV infection (7.8%), alcohol + HBV (1.6%), and other etiologies (10.9%)[(like Non alcoholic fatty liver disease, Autoimmune hepatitis and Miscellaneous (Hemochromatosis, Wilson disease, Idiopathic)]

In our study the most common presentation was fever (100%), Pain abdomen (95%) and abdomen distention (61.66%). In the study done by Mihas AA and Toussaint J^{21} the common clinical features were fever (69%) followed by abdominal pain (59%), hepatic encephalopathy (54%), and abdominal tenderness (49%). Ding X et al^{24} in their study reported clinical signs such as fever, abdominal pain, renal failure and hepatocellular carcinoma. Runyon BA et al²⁸ in their study reported clinical signs like Fever, Chills, Abdominal pain, Abdominal tenderness, Rebound tenderness, Shock and Altered mental status. All the studies corroborate that most common presentation of SBP is fever, Pain abdomen followed by other presentation. Out of 60 patients included in our study 24 patients died that is 40%. The in hospital mortality rate is 40% in our study. Nobre SR et al²⁹ reported Inhospital mortality rate of 37%. Musskopf MI et al²² also reported In-hospital mortality mg/dL rate of 40%. Bal CK et al³⁰ in their study reported SBP related in hospital mortality rate of 43%.

Polymorphonuclear (PMN) cell count in the ascitic fluid is essential for the diagnosis and management of spontaneous bacterial peritonitis.³¹ In present study amongst all patients the mean PMN cell count in ascitic fluid was 394.63cells/mm³. In present study the mean Ascitic fluid PMN cell count in patients who survived was $340.44\pm$ cells/mm3 and patients who died was 475.91 ± 424.7 cells/mm3. The mean value of mean Ascitic fluid PMN cell count is significantly higher in patients who died. The findings of our study are in concordance with the studies done by Musskopf MI et al²², Kraja B et al²⁷ and Kawale JB and Rawat KJ ³². They documented evidence of SBP in the form of ascitic fluid PMN >250 cells /mm3.

In present study amongst all patients the mean total bilirubin was 4.47 mg/dL and mean Serum Creatinine was 1.53mg/dL. The mean total bilirubin and mean serum creatinine in patients who survived was 3.16±2.48 mg/dL and 1.33±0.1 mg/dL respectively. Mean total bilirubin and mean serum creatinine in patients who died was 6.42±5.3 mg/dL and 1.84±0.25 mg/dL respectively. On comparison of total bilirubin and serum creatinine between patients who survived and who died mean total bilirubin and serum creatinine were significantly higher in patients who died. Our study corroborates the value of total bilirubin and serum creatinine as predictors of death in patients with SPB as indicated by previous studies done by Terg R et al³³ and Kawale JB and Rawat KJ³². Terg R et al³³ reported that mortality was higher in patients with total bilirubin levels > 4 mg/dL or serum creatinine > 1 mg/dL. These patients are considered to be at high risk as compared to low risk patients. Kawale JB and Rawat KJ³² reported that the mean total bilirubin amongst the survived was 3.88 mg/dl and amongst the expired was 10.03 mg/dl. The mean value of serum creatinine amongst the survived was 0.86 mg% and amongst the expired was 2.50 mg%. A review by Tandon P et al³⁴ has shown that renal dysfunction, usually defined as serum creatinine > 1.5mg/dL, was the most robust predictor of death among prognostic parameters for in-hospital mortality in patients with SBP. That finding warrants the identification of patients at high risk of death and the intravenous administration of albumin to reduce renal impairment and improve survival.³² Kamath PS et al³⁵ in their study has questioned the value of creatinine level as a predictor of death. However, since most patients in that study did not receive intravenous albumin, it is possible that its conclusions could not be generalized. We identified MELD score as a predictor of death, which is in agreement with other studies done by Luca A¹⁸, Cho JH et al³⁶ and Nobre SR et al²⁹. Previous studies done by Kamath PS et al³⁵ have suggested that the ability of MELD score to predict prognosis should be validated in patients at high risk of death receiving albumin systematically.

CONCLUSION-

A high index of suspicion should exist for SBP in patients with cirrhosis and ascitis. PMN cell count in ascitic fluid, total bilirubin, serum creatinine, serum sodium, INR, MELD score and iMELD score were found as prognostic factors for prediction of mortality in patients with SBP. As albumin has ability to improve intravascular volume and bind inflammatory cytokines has led to use of albumin therapy in patients with SBP.

REFERENCES-

- Schiff L, Eugene R, Schiff M. Cirrhosis. In Raven, Editor. Disease of the Liver. (8th ed.). Philadelphia: Lippincott; 1999:20-725.
- Fernandez J, Navasa M, Gomez J, Colmenero J, Vila J, Arroyo V et al. Bacterial infections in cirrhosis: epidemiological changes with invasive procedures and norfloxacin prophylaxis. Hepatology 2002;35:140 - 8.
- 3. Caly WR, Strauss E. A prospective study of bacterial infections in patients with cirrhosis. J Hepatol 1993;18:353-8.
- Pinzello G, Simonetti RG, Craxi A, Di Piazza S, Spanò C, Pagliaro L. Spontaneous bacterial peritonitis: a prospective investigation in predominantly nonalcoholic cirrhotic patients. Hepatology 1983;3:545-9.
- 5. Wiest R, Krag A, Gerbes A. Spontanous bacterial peritonitis: recent guidelines and beyond. Gut 2012;61:297-310.
- Evans LT, Kim WR, Poterucha JJ, Kamath PS. Spontaneous bacterial peritonitis in asymptomatic outpatients with cirrhotic ascites. Hepatology 2003;37:897-901.
- Castellote J, Girbau A, Maisterra S, Charhi N, Ballester R, Xiol
- 8. X. Spontaneous bacterial peritonitis and bacterascites prevalence in asymptomatic cirrhotic outpatients undergoing large-volume paracentesis. J Gastroenterol Hepatol 2008;23:256 - 9.
- Jeffries MA, Stern MA, Gunaratnam NT, Fontana RJ. Unsuspected infection is infrequent in asymptomatic outpatients with refractory ascites undergoing therapeutic paracentesis. Am J Gastroenterol 1999;94:2972 - 6.

- Conn HO, Fessel JM. Spontaneous bacterial peritonitis in cirrhosis: variations on a theme. Medicine (Baltimore) 1971;50:161e97.
- 11. Chu CM, Chang KY, Liaw YF. Prevalence and prognostic significance of bacterascites in cirrhosis with ascites. Dig Dis Sci 1995;40:561e5.
- Arvaniti V, D'Amico G, Fede G, Manousou P, Tsochatzis E, Pleguezuelo M et al. Infections in patients with cirrhosis increase mortality four-fold and should be used in determining prognosis. Gastroenterology 2010;139:1246- 56.
- 13. Wiest R, Garcia-Tsao G. Bacterial translocation (BT) in cirrhosis. Hepatology 2005;41:422- 33.
- 14. Wiest R , Krag A, Gerbes A . Spontaneous bacterial peritonitis: recent guidelines and beyond. Gut. 2012 Feb;61(2):297-310.
- 15. Wells CL. Relationship between intestinal microecology and the translocation of intestinal bacteria. Antonie Van Leeuwenhoek 1990;58:87-93.
- 16. Guarner C, Gonzalez-Navajas JM, Sanchez E, Soriando G, Francés R, Chiva M, et al. The detection of bacterial DNA in blood of rats with CCl4-induced cirrhosis with ascites represents episodes of bacterial translocation. Hepatology 2006;44:633-9.
- Llovet JM, Bartoli R, March F, Planas R, Vinado B, Cabre E, et al. Translocated intestinal bacteria cause spontaneous bacterial peritonitis in cirrhotic rats: molecular epidemiologic evidence. J Hepatol. 1998; 28:307-13.
- Kamath PS, Wiesner RH, Malinchoc M, Kremers W, Therneau TM, Kosberg CL et al .A model to predict survival in patients with end-stage liver disease. Hepatology. 2001 Feb;33(2):464-70.
- Luca A, Angermayr B, Bertolini G, Koenig F, Vizzini G, Ploner M et al. An integrated MELD model including serum sodium and age improves the prediction of early mortality in patients with cirrhosis. Liver Transpl 2007; 13: 1174-8
- 20. Filik L, Unal S. SBP in cirrhosis, frequency and predictive factors. Hepatology, 1988;8(1):27-31.
- Rawat N, Bhatnagar MK. Study of Prognostic Factors in Spontaneous Bacterial Peritonitis in Indian Population; J Assoc Physicians India. 2006;53:350.
- 22. Mihas AA, Toussaint J, Hsu HS, Dotherow P, Achord JL. Spontaneous bacterial peritonitis in cirrhosis: clinical and laboratory features, survival, and prognostic indicators. Hepatogastroenterology 1992; 39:520–522.
- 23. Musskopf MI, Fonseca FP, Gass J, de Mattos AZ, John JA, de Mello Brandao AB .Prognostic factors associated with in-hospital mortality in patients with spontaneous bacterial peritonitis. Ann Hepatol. 2012 Nov-Dec;11(6):915-20.
- 24. Thilakavathi R, Prathiba P, Saiprashanth PR, Venkatachalam V
- 25. Analysis of prevalence of spontaneous bacterial peritonitis in cirrhotic ascites in a tertiary care centre in Northern Tamilnadu. J. Evolution Med. Dent. Sci. 2018;7(4):487-491.
- 26. Ding X, Yu Y, Chen M, Wang C, Kang Y, Lou J. Causative agents and outcome of spontaneous bacterial peritonitis in cirrhotic patients: community-acquired versus nosocomial infections. BMC Infectious Diseases 2019;19(1):463.
- 27. Oladmeji AA, Temi AP, Adekunle AE, Taiwo RH, Ayokunler DS. Prevalence of spontaneous bacterial peritonitis in liver cirrhosis with ascitis. Pan Afr Med J. 2013;15:128.

- 28. Gayatri AA, Suryadharma IG, Purwadi N, Wibawa ID. The relationship between a model of end stage liver disease score (MELD score) and the occurrence of spontaneous bacterial peritonitis in liver cirrhotic patients. Acta Med Indones. 2007 Apr- Jun;39(2):75-8.
- 29. Kraja B, Sina M, Mone I, Pupuleku F, Babameto A, Prifti S et al. Predictive Value of the Model of End-Stage Liver Disease in Cirrhotic Patients with and without Spontaneous Bacterial Peritonitis. Hindawi Publishing Corporation Gastroenterology Research and Practice Volume 2012, Article ID 539059, 5 pages doi:10.1155/2012/539059.
- 30. Runyon BA, Mchutchison JG, Antillon MR, Akriviadis EA, Montano AA. Short-Course Versus Long-Course Antibiotic Treatment of Spontaneous Bacterial Peritonitis A Randomized Controlled Study of 100 Patients. Liver Unit, University of Southern California. Gastroenterology 1991 Jun;100(6):1737-42.
- Nobre SR, Cabral JE, Gomes JJ, Leitao MC. In-hospital mortality in spontaneous bacterial peritonitis: a new predictive model. Eur J Gastroenterol Hepatol 2008; 20: 1176-81.
- Bal CK, Daman R, Bhatia V. Predictors of fifty days inhospital mortality in decompensated cirrhosis patients with spontaneous bacterial peritonitis. World J Hepatol. 2016 Apr 28;8(12):566-72.
- Riggio O, Angeloni S. Ascitic fluid analysis for diagnosis and monitoring of spontaneous bacterial peritonitis. World J Gastroenterol 2009 August 21; 15(31): 3845-3850.
- 34. Kawale JB, Rawat KJ. Study of etiology, clinical profile and predictive factors of spontaneous bacterial peritonitis in cirrhosis of liver. Int J Res Med Sci 2017;5:2326-30.
- 35. Terg R, Gadano A, Cartier M, Casciato P, Lucero R, Munoz A et al. Serum creatinine and bilirubin predict renal failure and mortality in patients with spontaneous bacterial peritonitis: a retrospective study. Liver Int 2009; 29: 415-9.
- 36. Tandon P, Garcia-Tsao G. Renal dysfunction is the most important independent predictor of mortality in cirrhotic patients with spontaneous bacterial peritonitis. Clin Gastroenterol Hepatol 2011; 9: 260-5.
- 37. Kamath PS, Wiesner RH, Malinchoc M, Kremers W, Therneau TM, Kosberg CL et al. A model to predict survival in patients with end-stage liver disease. Hepatology 2001; 33: 464-70.
- 38. Cho JH, Park KH, Kim SH, Bang JH, Park WB, Kim HB et al. Bacteremia is a prognostic factor for poor outcome in spontaneous bacterial peritonitis. Scand J Infect Dis. 2007; 39: 697-702.