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Urine Strips Test And High Sensitive C - Reactive Protein In Diagnosis Of Spontaneous Bacterial Peritonitis In Egyptian Cirrhotic Patients With Ascites.

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ABSTRACT

Ascetic fluid polymorphonuclear leukocyte count (PMNL) 250/mm3 is diagnostic for spontaneous bacterial peritonitis (SBP) but rapid and simple screening test is needed. The aim of our study is to compare leukocyte esterase reagent strips (LERS) and high sensitive c reactive protein (hs – CRP) as two rapid methods for diagnosis of SBP in Egyptian cirrhotic patients with ascites. Ascetic fluids of 100 patients with liver cirrhosis, ascites and clinical suspicion of SBP were tested by LERS, hs - CRP and PMNL counts. Patients were divided into 2 groups. Group 1: patients with PMNL count≥ 250 cells/ mm3 and group 2: patients PMNL count < 250 cells/ mm3. By LERS group 1 were all positive while in group 2 (5) patients were positive and 53 were negative. The cut-off point of LERS was > grade 2. Sensitivity, specificity, positive predictive value (PPV) and negative predictive value (NPV) of LERS were 100%, 91.38%, 89.4% and 100% with accuracy 95% area under the curve was 0.915-0.994. The sensitivity, specificity, PPV and NPV of hs-CRP for SBP diagnosis was 83.33%, 74.14%, 70% and 86% respectively with accuracy 0.78% and area under the curve was 0.814. LERS is rapid and bed side diagnosis for SBP and has higher accuracy than hs-CRP.

Keywords: SBP, LERS, Ascites, Cirrhosis and hs-CRP.

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INTRODUCTION

Spontaneous bacterial peritonitis is one of the most feared and potentially fatal complications of cirrhotic ascites, [1] it is a bacterial infection of ascites occurring in the absence of contagious source of infection with an absolute neutrophil count of more than 250 cells/ mm3 or a positive culture.[2] Its prevalence in hospitalized patients with liver cirrhosis and ascites ranges from 10% to 30%. And the hospital mortality rate of it is also very high (30%–50%).[3-4] Early diagnosis and treatment of SBP is necessary for preventing mortality.[5] It can be diagnosed by the clinical manifestations that require a high index of clinical suspicion as the symptoms of SBP are nonspecific.[6] An AF polymorphonuclear (PMN) leukocyte count 250/mm3 irrespective of the AF culture result, is universally accepted nowadays as the best surrogate marker for diagnosing SBP.[7-8] The ascetic fluid total leukocyte and PMN count are not always done stat, thereby delaying time to diagnosis. Therefore, a rapid, simple screening test is needed for the prompt diagnosis of SBP.[9] LERS which has been found to be a sensitive and accurate predictor for the presence of PMN in body fluids such as urine and can be used in the diagnosis of SBP and has shown promising results in rapid diagnosis of SBP.[10-12] hs-CRP is more sensitive than C-Reactive Protein (CRP) as an inflammatory marker. High sensitive CRP is elevated in collagen vascular diseases, inflammatory arthritis, infective hepatitis, alcoholic liver diseases, cirrhosis and SBP.[13] The aim of our study is to compare LESR and hs-CRP as two rapid methods for diagnosis of SBP in Egyptian cirrhotic patients with ascites.

PATIENTS AND METHODS

Our study was prospective case control study. With one hundred patients with liver cirrhosis, ascites and a clinical suspicion of SBP, randomly selected from chronic liver disease patients admitted to Tropical medicine, internal medicine departments, Minia University hospital, Egypt after providing written informed consent. None of all patients had received antibiotics 14 days prior to the hospital admission. The diagnosis of liver cirrhosis was made by using standard clinical, laboratory and radiological criteria. Ascites was confirmed clinically and by abdominal ultrasonography. Patients with any contiguous source of intra-abdominal infection, with or without a positive culture and other causes of elevated PMN count in ascetic fluid such as tuberculosis, peritoneal carcinomatosis, pancreatitis, collagen vascular diseases, and inflammatory arthritis were excluded from the study. Also patients with coronary artery diseases were excluded from the study. The patients were divided into two groups according to the results of ascetic fluid PMNL count as follows: Group 1: included 42 patients with infected ascetic fluid (SBP group) and Group 2: included 58 patients without ascetic fluid infection (Non SBP group). Ascetic fluid infection was diagnosed as the PMNL $\geq$ 250 cells/ mm3 (Thevenot et al., 2004) [14]. All patients were subjected to full history taking, clinical examination, complete blood picture, Liver function tests and abdominal ultrasonography. The ascetic fluid was aspirated under complete aseptic conditions. The needle was introduced in the midline between the umbilicus and the symphysis pubis in the area of maximum dullness to percussion. (Runyon, 1986) [15]. Aspiration of 40 cc ascetic fluid sample will be taken using 20 G spinal needles under aseptic condition. The ascetic fluid is first examined with naked eye for its color and turbidity. Immediately after the procedure, a 15 ml of ascetic fluid was sent to the laboratory in heparinized tube for determination of differential white blood cell counts. Another 15 ml of ascetic fluid was sent for the culture. Ten ml of ascetic fluid will be tested by the use of the reagent strips designed for the testing of urine (Aution Sticks 10 EA; Arkray, Kyoto, Japan) where the strip was immersed in the ascetic fluid, immediately removed and after 120 seconds (the required waiting period), the color of the different reagents was compared with the color chart on the bottle and accordingly colorimetric grading was done (scale from 0 to 4). A correlation between PMN and a 5-grade scale was suggested by the manufacturer, as follows: grade 0, 0 PMN mm3; grade 1, 25 PMN /mm3; grade 2, 75 PMN/ mm3; grade 3, 250 PMN /mm3; and grade 4, 500 PMN /mm3. hs - CRP was estimated in ascetic fluid by tubidimetry method which measures turbidity changes in the latex immunoagglutination reaction using a latex reagent containing an antibody or antigen conjugated to latex particles.

Statistical analysis of the data was performed by using SPSS_20 software package. Categorical data parameters were presented in the form of frequencies and percentages. Chi-square test was used to study significant association between two categorical variables. Quantitative data were expressed in the form of mean and SD. T-test was used to test the significance between groups for quantitative data (P-value) was assumed significant if less than 0.05 and confidence intervals were at 95% level. Sensitivity, specificity, positive predictive value (PPV) and negative predictive value (NPV) were performed using the ROC curve.

RESULTS

There were no statistical significant difference between the two studied groups as regards age and sex. Gastrointestinal bleeding, fever, abdominal pain, rebound tenderness and hepatic encephalopathy were significantly higher ($p< 0.05$) in group 1 than in group 2 (21.4%, 57.2%, 90.4%, 69.0%, 23.8% vs. 6.89%, 24.1%, 74.1%, 25.9%, 8.6%) respectively (Table 1).

There were statistical significant increase in serum creatinine in group1 than group2 (2.66±1.97 mg/dl vs. 1.74±2.17mg/dl) also total leucocyte count is significantly higher in group 1 than group 2 (5.5±1.4 thousands cells/mm^3 vs. 4.9 ± 1.4 thousands cells/mm^3) $P<0.05$. Hb level of group I decreased significantly that of group II (8.79±1.7 gm. /dl vs. 9.68±1.9 gm. /dl) $P<0.05$. As regards PMNL cell count of the ascetic fluid it was significantly higher in group 1 than group (2 687.6±457.8 cells/ mm^3 Vs 124.5±33.9 cells/ mm^3) $P<0.001$ (Table2).

By testing ascetic fluid samples by LE activity test there was 47 positive samples and 53 negative samples, These results compared with PMNL count indicated that all 42 patients with PMNL ≥250 cells/mm^3 were positive using the LE strips while in the rest 58 patients with PMNL <250 cells/mm^3 (53) of them were found negative by the use of LE strips meaning that LE test was false positive in 5 patients. The cut-off point reported in this study by ROC curve for leucocyte esterase was > grade 2. Fifteen cases were positive in Grade 3 and 32 cases were positive in grade 4. In non SBP group (58 cases) there were 5 cases had false positive results. Three of them had grade 3 positive scale and only 2 had grade 4 positive scale. The level of hs-CRP was significantly higher in group 1 than in group 2 (67.3±15.8 mg/ml vs. 48±14.6 mg/ml) with p value 0.001 (Table 3).

Leukocyte esterase reagent strips give results of sensitivity, specificity, positive predictive value (PPV) and negative predictive value (NPV) at > grade 2 cut-off scale to diagnose SBP (100%, 91.38%, 89.4% and 100%) respectively with accuracy 95% using the ROC curve and area under the curve was 0.915-0.994. The cut-off point reported in this study by ROC curve for hs-CRP was >50 mg/ml. The sensitivity, specificity, PPV and NPV of hs-CRPl for SBP diagnosis was 83.33% , 74.14%, 70% and 86% respectively with accuracy 0.78% using the ROC curve, area under the curve was 0.814(Table 4& figure 1) .

DISCUSSION

Spontaneous bacterial peritonitis is a consequence of ascites in cirrhotic patients with prevalence rate of 3.5% to 30%.[16] Over 60% of the SBP episodes are caused by Gram-negative enteric bacilli like E.coli and Klebsiella pneumonia. The key pathogenic mechanism initiating SBP is bacterial translocation, a process through which enteric bacteria cross the intestinal barrier and infect the mesenteric lymph nodes, thus entering the blood circulation and ascitic fluid. The high rate of bacterial translocation in cirrhosis is due to intestinal bacterial overgrowth, loss of integrity of intestinal mucosal barrier and local immune system.[17] The mortality of untreated SBP remains high (>80%), Patient course and clinical outcome is based on an aggressive approach aiming to rapid diagnosis and prompt initiation of antibiotic therapy.[12] The gold standard for diagnosis of SBP is AF culture. Also the PMNs ≥ 250 cell/µL is diagnostic.[18-19] The use of urine dipstick in the diagnosis of SBP has been studied and has shown promising results in rapid diagnosis of SBP. [12]

Ascetic fluid hs-CRP measures low levels of CRP using Laser nephelometry. This test gives immediate results within 25 minutes. It is elevated in chronic liver diseases and Spontaneous Bacterial Peritonitis (SBP). A study by Kadam et al., 2016 reported that hs-CRP is a prognostic marker in cirrhosis with spontaneous bacterial peritonitis with high sensitivity.[13]

The present study was designed to compare the diagnostic validity of LE urine strips as rapid test and ascetic fluid hs-crp for diagnosis of SBP in Egyptian cirrhotic patients with ascites. In the present study, the most important clinical and laboratory data in the two studied groups (SBP and non SBP groups) of ascetic patients were compared. Of the important clinical data in the present study that, fever, pallor, abdominal pain, rebound Tenderness, upper GIT bleeding and encephalopathy were significantly prevalent in SBP group than non-SBP group, While presentation of palmar erythema and lower limb oedema was parallel in both groups. This clinical presentation is similar to that of the study by Sheer and Runyon, 2005 who found that symptoms of infection occur in most patients with SBP, including fever, abdominal pain and tenderness.[7] On the other

hand, a study done by Mlwati, 2013 found that the most common presenting symptoms were upper gastrointestinal bleeding, abdominal pain and fever.[20] Jaundice and hepatic encephalopathy was found in only 5%, this could be due to the fact that Mlwati study screened large sample size and all patients at a specific time with portal hypertension and ascites due to any liver problems or periportal fibrosis. In contrast, our study had small sample size of cases with decompensated liver cirrhosis complicated with portal hypertension or not. In our study, results of liver function tests showed no statistical significant difference between the two studied groups. These results were parallel to that of Rizk et al., 2014 who found that there was no significant difference in ALT, serum bilirubin and serum albumin levels between SBP and non SBP groups. Serum albumin level in our study showed no statistical significant difference between the two studied groups.[21] On the other hand Mlwati, 2013 found that serum albumin and total protein concentrations decreased significantly in SBP patients than non SBP patients, this may be due to previous treatment of our studied groups with salt free human albumin after paracentesis.[20] In the present study, significant increase were observed between the 2 groups regarding serum creatinine, this is in accordance with Ruiz-Del-Arbol et al 2003, who found that patients with SBP frequently develop a rapidly progressive impairment in systemic hemodynamics, leading to severe Renal failure.[22] In our study, the total leukocytic count in the peripheral blood in group I (SBP group) was higher than in patients of group II (Non SBP), the difference was statistically significant, this coincides with Rizk et al., 2014. He reported significant increase in the peripheral WBC count and PMNL percentage in patients with SBP more than patients with sterile ascites and this returns to that ascetic fluid infection increases cytokine secretion that leads to increase the phagocytosis of leukocytes.[21] In contrast Mlwati, 2013 found that no significant difference between SBP and non SBP patients as regard the peripheral WBC count, stated that chronic liver disease patients were immunocompromised patients and this contributed to the greater risk of infections encountered in patients with liver cirrhosis.[20] No significant difference in platelet count noticed between the SBP group and the non-SBP group in our study. Gill et al., 2012 found that platelet count significantly decreased in SBP patients than those without SBP, stated that severe bacterial infections involving the blood (bacteraemia) cause suppression of the reticuloendothelial system.[23] The present results indicated that there was highly significant increase in ascitic PMNL count in SBP group than non SBP group (620.1 vs. 131.6 (cells/mm3) ($p < 0.01$), this agree with Runyon, 2009.[19] they documented that the ascetic fluid cell count has been used to diagnose SBP and the PMNs ≥ 250 cell/µL is diagnostic. For AF culture, patients with positive culture in SBP group was 22 cases (46.8%) in our study and no cases recorded in non SBP group this difference between both groups was highly significant ($p<0.01$) and this coincides with Fernández et al., 2000 who found significant increase of positive culture results in SBP patients than non SBP.[18] The present results showed that the sensitivity, specificity, PPV and NPV of leukocyte esterase reagent strip test for > grade 2 cut-off scales to diagnose SBP were 100%, 91.38%, 89.4%, 100%, respectively with an accuracy 95% and area under the curve 0.97%. These results coincides with the findings of Bafandeh et al., 2012 they found that the diagnostic value of leukocyte esterase reagent strip test is very high, as the sensitivity, specificity, PPV and NPV of leukocyte esterase reagent strip test for the 1+ and 2+ cut-off scales to diagnose SBP were 97.5%, 84.6%, 74%, 98.7%, and 87.8%, 96.7%, 92.3%, 94.6%, respectively.[24] Chugh, et al., 2015 evaluated the diagnostic utility of LE reagent strips for rapid diagnosis of SBP. They found that the sensitivity and specificity of the LE test for detecting SBP taking grade 2 as cut off were 95% and 96.4% respectively, with a positive predictive value of 86.4% and a negative predictive value of 98.8%. Diagnostic accuracy of LE test was 96.1%.[25] Ribeiro et al., 2007 evaluated the accuracy of a urine reagent dipstick to determine ascitic fluid leukocyte count, and found 86% sensitivity and 96% specificity with positive and negative predictive values of 60% and 99%, respectively and 95% diagnostic accuracy.[26] We also detected the diagnostic validity of ascetic fluid high sensitive c-reactive protein for rapid diagnosis of SBP and we found that at cutoff value >50 the sensitivity, specificity, positive predictive value, negative predictive value, accuracy and AUC of hs-CRP for SPB diagnosis was 83.33% , 74.14, 70%, 86%, 78% and 0.814 respectively. This means leukocyte esterase has high sensitivity, specificity, PPV and NPV than high sensitive C-reactive protein. Also the accuracy of leukocyte esterase is higher than hs-CRP and leukocyte esterase is rapid test while hs-CRP takes 25 minutes.

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Table (1) Comparison of the clinical data in the two studied groups

	Group I (n = 42)		Group II (n = 58)		P-value
	No.	%	No.	%	
Gastrointestinal bleeding	9	21.4	3	6.8	0.013
Past history of SBP	27	64.3	24	41.3	0.023
Fever	24	57.2	14	24.1	<0.001
Lower limb edema	37	88.1	51	87.9	0.980
Abdominal pain	38	90.4	43	74.1	0.039
Rebound tenderness	29	69.0	15	25.9	0.001
Hepatic encephalopathy	10	23.8	5	8.6	0.037

Table (2): Laboratory data in the 2 studied groups

	Group I	Group II	P-Value
ALT (IU/L)	88.2±66.3	81.2±34.9	0.294
AST (IU/L)	94.9±86.9	80.3±29.6	0.236
total bil.(mg/dl)	4.79±2.78	4.65±3.38	0.820
direct bil.(mg/dl)	2.66±2.14	2.65±2.29	0.994
Albumin (g/dl)	2.74±0.66	2.77±.055	0.820
INR	1.6±0.23	1.62±0.20	0.644
Creatinine (mg/dl)	2.66±1.97	1.74±2.17	0.032
HB gm/dL	8.7±1.7	9.6±1.9	0.017
WBCs ($x10^3$ / mm^3)	5.54±1.4	4.9±1.4	0.041
Platelet count ($x10^3$ / mm^3)	128±65.1	127±43.9	0.896
PMNL count of ascetic fluid (cells/mm)	687.6±457.8	124.5±33.9	<0.001

Table (3): LE activity and hs-CRPl in relation to PMNL count

		Group1	Group2	P value
		N=42	N=58	
hs-CRP	*Mean ± SD*	67.3±15.8mg/ml	48±14.6mg/ml	*<0.001**
LE activity	*Grade I* *Grade II* *Grade III* *Grade IV*	0(0%) 0(0%) 12(28.6%) 30(71.4%)	10(17.2%) 43(74.1%) 3(5.2%) 2(3.4%)	*<0.001**

Table (4): ROC curve analysis of LE activity and hs-CRPl for diagnosis of SBP

	Hs-CRP	LE activity
Cutoff point	>50mg/ml	> grade 2
AUC	0.814	0.970
95% CI	0.724-0.885	0.915-0.994
P value	*<0.001**	*<0.001**
Sensitivity	83.33	100
Specificity	74.14	91.38
PPV	70	89.4
NPV	86	100
Accuracy	78	95

AUC: area under curve; CI: Confidence interval; PPV: positive predictive value; NPV: negative predictive value

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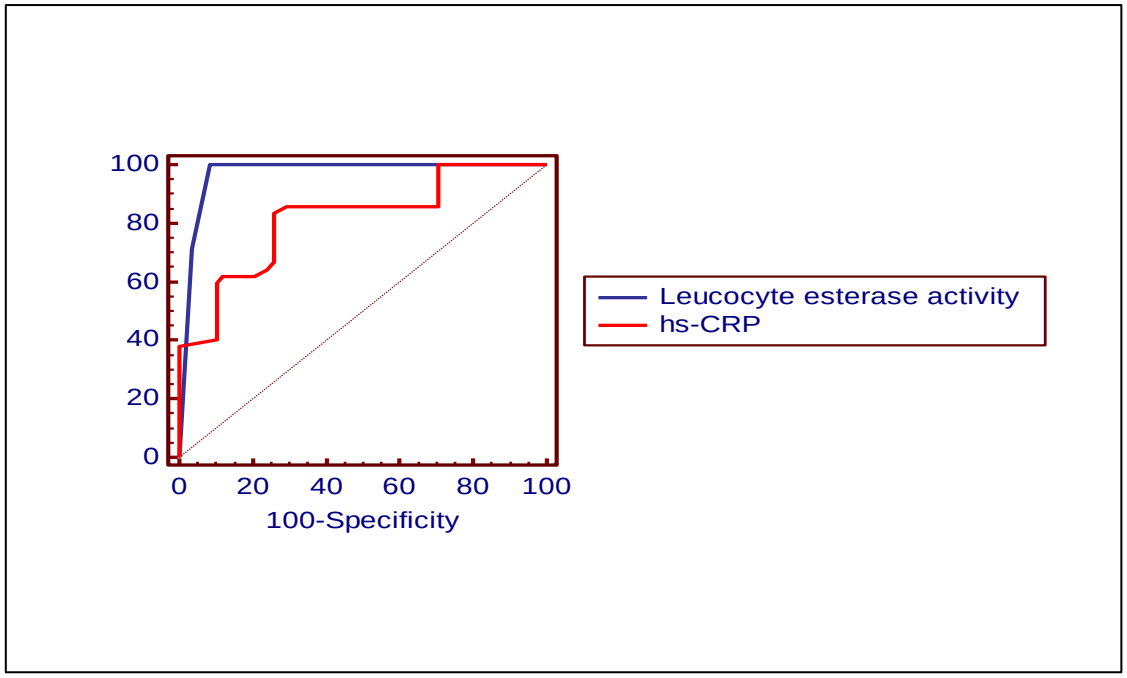

Figure (1): ROC curve of leucocyte esterase and Hs-CRPl in diagnosis of SBP

CONCLUSION

The reagent strip testing of ascetic fluid is a very sensitive and specific method for diagnosis of SBP in cirrhotic patients with ascites especially when the patient has manifestation suggestive of SBP. It can be used every-where at the patient's bedside as it is rapid, easy to use, inexpensive and good negative test. It is more sensitive, specific and accurate than hs-CRP. A positive result should be an indication for empirical antibiotic therapy. It may be useful as a screening test in cirrhotic patients with ascites or who suspected to have SBP for early detection of SBP. In patient with cirrhosis when there is fever, pallor, abdominal pain, rebound tenderness, bleeding tendency and renal impairment it is better to diagnose SBP by LES test as a rapid and bed side test and give empirical antibiotics.

Disclosure:

Ethics approval and consent to participate

The study was approved by Ethical Research Board of Minia School of Medicine, Egypt. Written informed consent was obtained from all patients participated in this study. The study was conducted in accordance with the guidelines of 1975 declaration of Helsinki.

Consent for publication: "Not applicable"

Availability of data and materials: Non applicable

Competing interests: All authors declare that they have no competing interest.

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Authors' contributions:

Elham Ahmed, Magdy fouad and Ali Hussien Aldahrouty gave the concept of the study. Elham Ahmed and Magdy fouad shared in study design and submitted of the manuscript, Wafaa A. Abdelghany: recruited the patient participated in the sequence alignment of the study. Alshymaa Ahmed Hassanin: shared in recruitment of the patient and edited the manuscript. Waleed Mahmoud Abdelhamid and Hend Mohammed Moness: performed the laboratory investigation.

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REFERENCES

[1] Amini M, Runyon BA: Alcoholic hepatitis: a clinician's guide to diagnosis and therapy. World J Gastroenterol. 2010 Oct 21; 16(39):4905-12.

[2] Gaya DR, David B Lyon T, Clarke J, Jamdar S, Inverarity D, Forrest EH, et al: Bedside leu-kocyte esterase reagent strips with spectrophotometric analysis to rapidly exclude spontaneous bacterial peritonitis: a pilot study. Eur J Gastroenterol Hepatol. 2007 Apr; 19(4):289-95.

[3] Fernández J, Navasa M, Gómez J, Colmenero J, Vila J, Arroyo V, et al: Bacterial infection in cirrhosis: Epidemiological changes with invasive procedures and norfloxacin prophylaxis Hepatology. 2002 Jan; 35(1):140-8.

[4] Ginès P , Cárdenas A, Arroyo V, Rodés J: Management of cirrhosis and ascites. N Engl J Med. 2004 Apr 15;350(16):1646-54.

[5] Jepsen P , Vilstrup H, Møller JK, Sørensen HT: Prognosis of patients with liver cirrhosis and spontaneous bacterial peritonitis. Hepatogastroenterology. 2003 Nov-Dec;50(54):2133-6.

[6] Rimola A , García-Tsao G, Navasa M, Piddock LJ, Planas R, Bernard B, et al: Diagnosis, Treatment and prophylaxis of spontaneous bacterial peritonitis: a consensus document J Hepatol. 2000 Jan;32(1):142-53.

[7] Sheer TA , Runyon BA: Spontaneous bacterial peritonitis. Dig Dis. 2005;23(1):39-46.

[8] Wong CL , Holroyd-Leduc J, Thorpe KE, Straus SE. Does this patient have bacterial peritonitis or portal hypertension? How do I perform a paracentesis and analyze the results? JAMA. 2008 Mar 12;299(10):1166-78. doi: 10.1001/jama.299.10.1166.

[9] Lejeune B , Baron R, Guillois B, Mayeux D: Evaluation of a screening test for detecting urinary tract infection in newborns and infants Clin Pathol. 1991 Dec;44(12):1029-30.

[10] Hiscoke C , Yoxall H, Greig D, Lightfoot NF: Validation of a method for the rapid diagnosis of urinary tract infection suitable for use in general practice. Br J Gen Pract. 1990 Oct;40(339):403-5.

[11] Castellote J , López C, Gornals J, Tremosa G, Fariña ER, Baliellas C, et al: Rapid diagnosis of spontaneous bacterial peritonitis by use of reagent strips. atology. 2003 Jun; 37(6):1

[12] Koulaouzidis A. Diagnosis of spontaneous bacterial peritonitis: An update on leucocyte esterase reagent strips. World J Gastroenterol. 2011 Mar 7;17(9):1091-4. doi: 10.3748/wjg.v17.i9.1091.

[13] Kadam N , Acharya S , Shukla S , Gupta K : Ascitic Fluid High Sensitive C-Reactive Protein (hs-CRP). A Prognostic Marker in Cirrhosis with Spontaneous Bacterial Peritonitis. J Clin Diagn Res. 2016 Apr;10(4):OC20-4. doi: 10.7860/JCDR/2016/17931.7610.

[14] Thévenot T , Cadranel JF, Nguyen-Khac E, Tilmant L, Tiry C, Welty S, et al: Diagnosis of spontaneous bacterial peritonitis in cirrhotic patients by use of two reagent strips, Eur J Gastroenterol Hepatol. 2004 Jun;16(6):579-83.

[15] Runyon, BA: Low-protein-concentration ascitic fluid is predisposed to spontaneous bacterial peritonitis. Gastroenterology. 1986 91(6): 1343-6.

[16] Farahmand F , Eshagh Roze M, Shams S, Ghajarzadeh M, Mohammadi B: Diagnosis of Spontaneous Bacterial Peritonitis in Children by Reagent Strips. Acta Med Iran. 2013 Mar 16;51(2):125-8.

[17] González Alonso R, González García M, Albillos Martínez A: Physiopathology of bacterial translocation and spontaneous bacterial peritonitis in cirrhosis. Gastroenterol Hepatol. 2007;30:78–84.

[18] Fernández J , Bauer TM, Navasa M, Rodés J: Diagnosis, Treatment and Prevention of spontaneous bacterial peritonitis. Baillieres Best Pract Res Clin Gastroenterol. 2000 Dec;14(6):975-990.

[19] Runyon BA (2009): Management of Adult Patients with Ascites Due to Cirrhosis : An Update. Hepatology. 46(6):2087–107.

[20] Mlwati A: Spontaneous bacterial peritonitis among patients with portal hypertension and ascitis attending Bugando Medical Centre. Tanzania. CUHAS MMed Dissertation: Mwanza; 2013.

[21] Rizk E, Elzehery R, Zakaria S, Abdel-Razik A, Elhammady D: Ascitic Fluid Calprotectin and Serum C-Reactive Protein as Diagnostic Markers for Spontaneous Bacterial Peritonitis Afro-Egypt J Infect Endem Dis 2014; 4(3): 117-125

[22] Ruiz-del-Arbol L, Urman J, Fernández J, González M, Navasa M, Monescillo A, et al: Hepatology. 2003 Nov;38(5):1210-8.PMID:14578859

[23] Gill AS, Singh A, Matreja PS, Chinna RS, Mahajan R, Chhina DK: Spontaneous Bacterial Peritonitis in Alcoholic Cirrhosis: An Indian Perspective. Euroasian J Hepato-Gastroenterol. 2012;2:14–19.

[24] Yousef B, Khodaei M: Evaluation of leukocyte esterase reagent strip test to detect spontaneous bacterial peritonitis in cirrhotic patients Gastroenterology Insights 2012 .; volume 4:e13. 52-55

[25] Chugh K , Agrawal Y , Goyal V , Khatri V , Kumar P : Diagnosing bacterial peritonitis made easy by use of leukocyte esterase dipsticks. Int J Crit Illn Inj Sci. 2015 Jan-Mar;5(1):32-7. doi: 10.4103/2229-5151.152337.

[26] Ribeiro TC , Kondo M, Amaral AC, Parise ER, Bragagnolo Júnior MA, Souza AF: Evaluation of Reagent Strips for Ascitic Fluid Leukocyte Determination: Is It a Possible Alternative for Spontaneous Bacterial Peritonitis Rapid Diagnosis? Braz J Infect Dis. 2007 Feb;11(1):70-4.