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Study of Serum and Ascitic Fluid Lipopolysaccharide Binding Protein as Potential Markers of Infection in Spontaneous Bacterial Peritonitis

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ABSTRACT

Background: Hemodynamic instability in cirrhotic individuals and the onset of bacterial infection are both linked to elevated levels of Lipopolysaccharide binding protein (LBP).

Objective: The aim of the current work was to evaluate the significance of lipopolysaccharide binding protein (LBP) level in serum and ascitic fluid in spontaneous bacterial peritonitis (SBP) patients as a marker for infection.

Patients and Methods:A total of 112 patients were enrolled in this case control study and were split into two categories: Group (A):consisted of 56 individuals with chronic liver disease (CLD) having ascites exacerbated by spontaneous bacterial peritonitis (SBP) through clinical and laboratory examinations. Group (B): consisted of 56 individuals with chronic liver disease (CLD) and ascites who had no detectable infection based on clinical and laboratory tests.

Results: In group A; significant positive correlations were found between serum LBP, HB, and total protein. Also, a negative remarkable correlation between serum LBP, INR, PTT, PT, serum creatinine, direct bilirubin, total bilirubin, PLT, and ascitic fluid LBP. In group (B);significant positive correlations were found between serum LBP, AST, and TLC. Also, a negative remarkable correlation between serum LBP, PTT, serum urea, serum creatinine, and total bilirubin.

Conclusion: It could be concluded that serum LBP demonstrated a highly significant difference between the two groups with a substantial difference as regard the diagnosis of spontaneous bacterial peritonitis, both in terms of sensitivity and specificity. Serum LBP may be considered as a diagnostic tool for SBP in cirrhotic patients with ascites.

Keywords: Lipopolysaccharide Binding Protein, Spontaneous Bacterial Peritonitis.

INTRODUCTION

Ascitic fluid becomes infected with bacteria (often a single species) when there is no clear indication of a peritoneal or other tissue source for the sepsis that results in peritonitis (also known as "spontaneous bacterial peritonitis" or "SBP"). Patients with cirrhosis are more likely to get SBP than urinary tract infections (UTIs), pneumonias, skin/soft tissue infections, or septicemia **(1).**

Diagnostic testing of ascitic fluid (AF) taken during abdominal paracentesis is used to identify cases of spontaneous bacterial peritonitis (SBP). Due to its great sensitivity, the polymorphonuclear leucocytic (PMNLs) count of 250 cells/mm^3 has long been used as the gold standard for SBP diagnosis. Translocation of bacteria seems to be the primary mechanism behind spontaneous bacterial peritonitis (SBP) **(2).**

This process, known as bacterial translocation (BT), involves the movement of bacteria or bacterial endotoxins over the intestinal mucosa and into the mesenteric lymph nodes and other extra intestinal locations. Patients with cirrhosis have an increased risk of death due to infections. Many researchers believe that bacterial translocation is the basic mechanism connected to infection development in cirrhosis. Serum LPS (Lipopolysaccharide) -LBP complex levels may rise in individuals with Spontaneous bacterial peritonitis because bacterial endotoxins stimulate LBP production **(3).**

Hepatocytes secrete a soluble acute phase protein called lipopolysaccharide-binding protein (LBP), which aids in the binding of bacterial lipopolysaccharide (LPS) to the cell membrane molecule CD14 and the Toll-like receptor 4, triggering a cascade that results in cytokine production and an inflammatory response**(4).**

The concentration of lipopolysaccharide binding protein (LBP) is thought to reflect chronic contact with bacteria and endotoxins. Patients with cirrhosis and ascites who have elevated levels of lipopolysaccharide binding protein (LBP) are at increased risk for developing life-threatening bacterial infections. The concentration of LBP in the peripheral blood has been utilized as a proxy for bacterial translocation. After an episode of bacteriemia, serum LBP levels remain elevated for an extended period of time and can be used as a reliable diagnostic for the diagnosis of bacterial translocation (BT)**(5)**.

Hemodynamic instability and the onset of bacterial infection are both linked to elevated LBP levels in cirrhotic patients. In liver cirrhosis, LBP remains a viable surrogate measure of BT. Patients who are free of infection at baseline but are at high risk for developing an infection during follow-up could be identified using LBP levels as a surrogate measure**(6).**

This study was aimed to evaluate the significance of lipopolysaccharide binding protein (LBP) level in serum and ascitic fluid in spontaneous bacterial peritonitis (SBP) patients as a marker for infection.

PATIENTS AND METHODS

This case control study included a total of 112 chronic liver disease (CLD) patients, attending at Out-Patient Clinics, Department of Internal Medicine, Zagazig University, during the period from January 2022 to August 2022. Research laboratory and immunology

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work was conducted at Clinical Pathology Department.

Patients' age ranged between 38 to 57 years. They were 78 males and 34 females.

The participants were divided between two groups; Group (A): consisted of 56 chronic liver disease (CLD) patients with ascites, complicated by spontaneous bacterial peritonitis (SBP) proved by clinical examination and laboratory investigations. **Group (B):** consisted of 56 chronic liver disease (CLD) patients with ascites without any evidence of infection proved by clinical examination and laboratory investigations.

Inclusion criteria: Patients with ascites due to chronic liver disease, both sexes, and age ≥ 18 years.
Exclusion criteria: Age ≤ 18 years. Patients with ascites due to other causes than chronic liver disease. Patients with an established illness (such as a chest infection or urinary tract infection) who had not developed spontaneous bacterial peritonitis. Patients with HIV, malignancy, auto immune diseases, or chronic renal failure. Patients who show hemodynamic instability.

All patients were subjected to

A. A comprehensive history taking (with special stress presence of on jaundice, pruritus, gastro-intestinal bleeding, coagulopathy, abdominal distension, abdominal pain, fever and altered mental status).
B. Full Clinical examination.
C. Lab investigations: Included any investigations that verify inclusion and exclusion criteria:
- Complete blood picture (CBC), plasma glucose concentration, liver &kidney function tests, coagulation profile: PT, INR, PTT, and inflammatory parameters: CRP and ESR.
- **Ascitic fluid samples** were taken and analyzed to confirm infection with spontaneous bacterial peritonitis.
- **Lipopolysaccharide binding protein** Serum and ascitic fluid samples from each patient were analyzed using a Human LBP (Lipopolysaccharide binding protein) ELISA kit (Fine Test ®) to determine LBP concentrations.

D. Radiological findings: Chest x-ray was done for exclusion of chest infection. Pelvi abdominal ultrasound was done to all patients to confirm liver cirrhosis and presence of ascites.

***Ethical Consideration*:**

This study was ethically approved by Zagazig University's Research Ethics Committee. and submitted them to Zagazig University (ZU-IRB##6612-22-12-2020).Written informed consent of all the participants was obtained. The study protocol conformed to the Helsinki Declaration, the ethical norm of the World Medical Association for human testing.

Statistical analysis:

In order to analyze the data acquired, Statistical Package of Social Services version 20 was used to execute it on a computer (SPSS). In order to convey the findings, tables and graphs were employed. The quantitative data was presented in the form of the mean, median, standard deviation, and confidence intervals. The information was presented using qualitative statistics such as frequency and percentage. The student's t test (T) is used to assess the data while dealing with quantitative independent variables. Pearson Chi-Square and Chi-Square for Linear Trend (X2) were used to assess qualitatively independent data. The significance of a P value of 0.05 or less was determined.

RESULTS

Table (1) shows that the average age was 48.96±5.24 in group (A) and 49.62±4.46 in group (B). Gender distributed as 26.8 % females, 73.2 % males in group (A) and 33.9 % females, 66.1 % males in group (B). Both groups were predominantly male, and there was no discernible age or sex difference between them.

Table (1): Demographics of studied groups according to age and gender distribution

			Group A	Group B	t/ X^2	P
Age (years**)**			**48.96±5.24**	**49.62±4.46**	**0.718**	**0.475**
Gender	**Female**	**N**	**15**	**19**		
		%	**26.8%**	**33.9%**		
	Male	**N**	**41**	**37**	**0.67**	**0.41**
		%	**73.2%**	**66.1%**		
Total		**N**	**56**	**56**		
		%	**100.0%**	**100.0%**		

Table (2) shows first & second hour ESR and CRP. Group A had significantly higher levels of all inflammatory markers compared to Group B.

Table (2): Inflammation markers (1st&2nd hours ESR, and CRP) in groups A and B.

	Group A	Group B	t	P
First hour ESR (mm/hr)	**15.39±3.08**	**10.83±2.41**	**5.282**	**0.000**
Second hour ESR (mm/hr)	**35.14±5.71**	**27.16±6.42**	**4.937**	**0.000**
CRP (mg/l)	**18.70±4.41**	**8.03±1.82**	**13.185**	**0.000**

Table (3) shows distribution of plasma glucose measured by mg/dl, serum total bilirubin and direct bilirubin measured by mg/dl, total protein and albumin measured by g/dl, ALT, AST and Alkaline phosphatase measured by IU/L, serum creatinine and serum urea measured by mg/dl, PT and PTT measured by seconds and INR. Serum Glucose, Total Protein, Serum Creatinine, Serum Urea, PT, PTT and INR were significantly higher among group A and of high significance.

Table (3): Plasma **glucose, liver and kidney function tests and bleeding profile distribution between group A and group B.**

	Group A	Group B	t	P
Plasma **Glucose** (mg/dl)	**99.05±0.87**	**92.37±0.25**	**2.720**	**0.008**
Total Bilirubin (mg/dl)	**1.34±0.29**	**1.32±0.28**	**0.408**	**0.684**
Direct Bilirubin (mg/dl)	**0.43±0.07**	**0.44±0.09**	**0.746**	**0.457**
Serum total protein (g/dl)	**6.42±0.50**	**6.17±0.32**	**3.100**	**0.002**
Albumin (g/dl)	**2.86±0.26**	**2.94±0.18**	**1.743**	**0.084**
ALT (IU/L)	**21.08±2.92**	**20.30±4.34**	**1.120**	**0.265**
AST (IU/L)	**44.19±5.91**	**42.51±7.03**	**1.374**	**0.172**
Alkaline Phos. (IU/L)	**73.19±8.97**	**71.87±5.41**	**0.943**	**0.348**
Serum creatinine (mg/dl)	**1.35±0.31**	**0.85±0.20**	**6.349**	**0.000**
Serum Urea (mg/dl)	**36.61±5.21**	**24.56±5.65**	**4.988**	**0.000**
PT (seconds)	**16.42±2.26**	**14.21±2.61**	**4.771**	**0.000**
PTT (seconds)	**45.41±9.56**	**41.23±10.13**	**2.080**	**0.040**
INR	**1.45±0.18**	**1.24±0.23**	**5.272**	**0.000**

Table (4) represents distribution of TLC (cells/µl) in ascitic fluid in groups A and B, serum and ascitic fluid LBP (µg/ml) in groups A and B. TLC in ascitic fluid, serum LBP and ascitic fluid LBP were higher in group A than group B.

Table (4): TLC in Ascitic fluid, Serum LBP and ascitic fluid LBP distribution between group A and group B

	Group A	Group B	t	P
TLC_in_Ascitic_fluid (cells/µl)	**4551.0±1110.1**	**384.16±23.64**	**28.082**	**0.000**
Serum_LBP (µg/ml)	**2161.59±85.26**	**1031.76±251.9**	**17.711**	**0.000**
Ascitic_fluid_LBP (µg/ml)	**59.81±12.54**	**13.35±3.28**	**5.367**	**0.000**

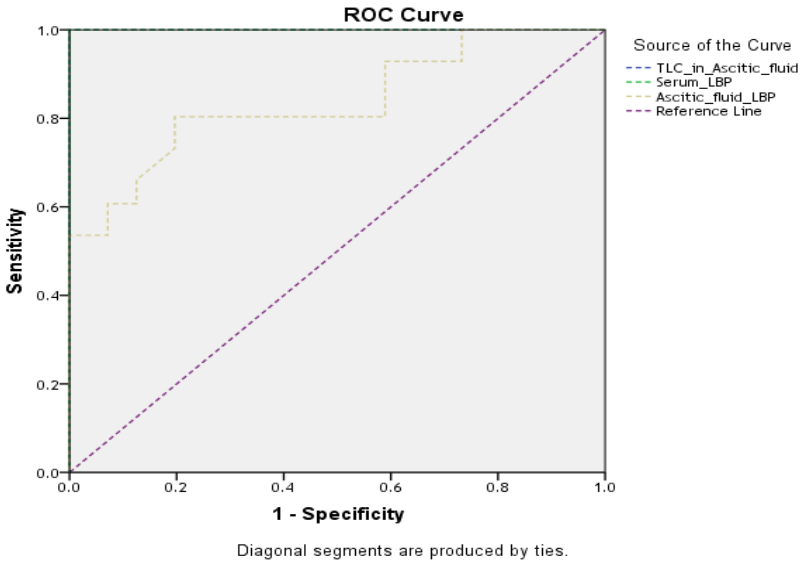

Fig. (1): ROC Curve for TLC in Ascetic fluid, Serum LBP and ascetic fluid LBP regard spontaneous infection.

All parameters were with significant AUC with cutoff >1836, >1817.9 and >18.2 with sensitivity 100.0%, 100.0% and 80.0% respectively and specificity were 100.0%, 100.0% and 79.0% respectively (Table 5).

Table (5): Area under curve (AUC) and validity

Test Result Variable(s)	Area	Cutoff	P	95% Confidence Interval		Sensitivity	Specificity
				Lower Bound	Upper Bound		
TLC in _ascitic fluid (cells/µl	**1.000**	**>1836**	**0.000**	**1.000**	**1.000**	**100.0%**	**100.0%**
Serum LBP (µg/ml**)**	**1.000**	**>1817.9**	**0.000**	**1.000**	**1.000**	**100.0%**	**100.0%**
ascitic fluid LBP (µg/ml**)**	**0.837**	**>18.2**	**0.000**	**0.762**	**0.911**	**80.0%**	**79.0%**

Regarding correlations in groups (A), there was statistically significant positive correlation between TLC in ascitic fluid, serum LBP, HB, and negative correlation between TLC in ascitic fluid, CRP, serum glucose and D. bilirubin. There was significant positive correlation between serum LBP, HB, and total protein. Also, a negative remarkable correlation between serum LBP, INR, PTT, PT, serum creatinine, direct bilirubin, total bilirubin, PLT, and ascitic fluid LBP. There was significant positive correlation between ascitic fluid LBP total bilirubin and AST, and negative correlation between ascitic fluid, PLT, and serum LBP (Table 6).

Table (6): Represents correlation between results in group A

Group			TLC in ascetic fluid	SerumLBP	Ascetic fluid LBP
	SerumLBP (µg/ml**)**	**r**	**0.569****	**1**	**-0.309-***
		P	**0.000**		**0.021**
	Ascitic fluidLBP (µg/ml**)**	**r**	**0.084**	**-.309-***	**1**
		P	**0.536**	**0.021**	
	TLC (x10^{3} cells/mm3)	**r**	**0.011**	**0.207**	**-0.123-**
		P	**0.938**	**0.125**	**0.366**
	Neutrophils (x10^{3} cells/mm3)	**r**	**-.112-**	**.173**	**-.113-**
		P	**.411**	**.202**	**.409**
	Lymphocytes (x10^{3} cells/mm3)	**r**	**.024**	**.038**	**-.031-**
		P	**.863**	**.780**	**.821**
	Hemoglobin(g/dl)	**r**	**.541****	**.342****	**-.247-**
		P	**.000**	**.010**	**.066**
	PLT (×10^{3} cells/mm3**)**	**r**	**-.121-**	**-.274-***	**-.310-***
		P	**.375**	**.041**	**.020**
	CRP(mg/l)	**r**	**-.352-****	**-.229-**	**-.058-**
		P	**.008**	**.089**	**.670**
	Serum Glucose (mg/dl**)**	**r**	**-.001-**	**-.025-**	**.212**
		P	**.994**	**.857**	**.116**
	Total Bilirubin (mg/dl**)**	**r**	**-.474-****	**-.478-****	**.365****
		P	**.000**	**.000**	**.006**
	Direct Bilirubin (mg/dl**)**	**r**	**-.453-****	**-.444-****	**.128**
		P	**.000**	**.001**	**.346**
	Total Proteins (g/dl**)**	**r**	**.175**	**.389****	**-.078-**
		P	**.198**	**.003**	**.567**
	Albumin (g/dl**)**	**r**	**.150**	**.089**	**.174**
		P	**.269**	**.515**	**.201**
	ALT (IU/L**)**	**r**	**-.171-**	**.019**	**-.062-**
		P	**.208**	**.892**	**.652**
	AST (IU/L**)**	**r**	**.151**	**-.175-**	**.512****
		P	**.265**	**.197**	**.000**
	Alkaline Phosphatase (IU/L**)**	**r**	**.043**	**-.051-**	**.039**
		P	**.752**	**.708**	**.778**
	Serum Creatinine (mg/dl**)**	**r**	**-.055-**	**-.457-****	**-.092-**
		P	**.686**	**.000**	**.498**
	Serum Urea (mg/dl**)**	**r**	**-.040-**	**-.043-**	**-.028-**
		P	**.772**	**.754**	**.836**
	PT(seconds)	**r**	**-.104-**	**-.401-****	**.082**
		P	**.445**	**.002**	**.549**
	PTT (seconds)	**r**	**-.101-**	**-.480-****	**.148**
		P	**.461**	**.000**	**.277**
	INR	**r**	**-.122-**	**-.375-****	**.054**
		P	**.370**	**.004**	**.690**

*, ** significant correlation ( ) or (-) correlation

Regarding correlations in groups (B), there was statistically significant positive correlation between TLC in ascitic fluid, PTT, serum creatinine, and AST, and negative correlation between TLC in ascitic fluid, Alb, PLT, and total bilirubin. There was significant positive correlation between serum LBP, AST and TLC. Also, a negative remarkable correlation between serum LBP, PTT, serum urea, serum creatinine, and total bilirubin. There was significant positive correlation between ascitic fluid LBP, PT, INR, AST, TLC, neutrophil, and serum LBP, and negative correlation between ascitic fluid LBP, serum creatinine, and serum urea (Table 7).

Table (7): represents correlation between results in group B

Group			TLCinAsciticfluid	SerumLBP	Ascitic fluid_LBP
	SerumLBP (µg/ml**)**	**r**	**-.073-**	**1**	**.606****
		P	**.594**		**.000**
	AsciticfluidLBP (µg/ml**)**	**r**	**-.221-**	**.606****	**1**
		P	**.102**	**.000**	
	TLC (x10^3 cells/mm3)	**r**	**.098**	**.312***	**.358****
		P	**.472**	**.019**	**.007**
	Neutrophils (x10^3 cells/mm3)	**r**	**.167**	**.362****	**.283***
		P	**.219**	**.006**	**.035**
	Lymphocytes (x10^3 cells/mm3)	**r**	**.078**	**.007**	**.049**
		P	**.568**	**.962**	**.721**
	Hemoglobin(g/dl)	**r**	**-.002-**	**.227**	**-.167-**
		P	**.988**	**.092**	**.220**
	PLT (×10^3 cells/mm3**)**	**r**	**-.419-****	**.154**	**.019**
		P	**.001**	**.258**	**.892**
	CRP(mg/l)	**r**	**.171**	**.060**	**-.080-**
		P	**.207**	**.659**	**.560**
	Serum Glucose (mg/dl**)**	**r**	**-.067-**	**-.176-**	**-.251-**
		P	**.622**	**.195**	**.062**
	Total Bilirubin (mg/dl**)**	**r**	**-.447-****	**-.333-***	**-.114-**
		P	**.001**	**.012**	**.405**
	Direct Bilirubin (mg/dl**)**	**r**	**.044**	**-.175-**	**.155**
		P	**.748**	**.197**	**.255**
	Total Proteins (g/dl**)**	**r**	**-.045-**	**-.256-**	**-.180-**
		P	**.743**	**.057**	**.184**
	Albumin (g/dl**)**	**r**	**-.288-***	**.159**	**-.056-**
		P	**.031**	**.242**	**.680**
	ALT (IU/L**)**	**r**	**-.126-**	**.061**	**-.042-**
		P	**.355**	**.657**	**.757**
	AST (IU/L**)**	**r**	**.466****	**.339***	**.336***
		P	**.000**	**.011**	**.011**
	Alkaline Phosphatase (IU/L**)**	**r**	**-.143-**	**-.047-**	**.075**
		P	**.294**	**.734**	**.584**
	Serum Creatinine (mg/dl**)**	**r**	**.394****	**-.602-****	**-.519-****
		P	**.003**	**.000**	**.000**
	Serum Urea (mg/dl**)**	**r**	**.216**	**-.568-****	**-.603-****
		P	**.110**	**.000**	**.000**
	PT(seconds)	**r**	**.239**	**.142**	**.382****
		P	**.076**	**.297**	**.004**
	PTT(seconds)	**r**	**.265***	**-.343-****	**-.186-**
		P	**.048**	**.010**	**.171**
	INR	**r**	**.252**	**.156**	**.390****
		P	**.061**	**.251**	**.003**

*, ** significant correlation ( ) or (-) correlation

DISCUSSION

SBP has a bad prognosis and outcome, hence early diagnosis is essential. To diagnosis SBP in patients with liver cirrhosis, it is necessary to find a polymorphonuclear (PMN) cell count in the ascitic fluid of 250 cells/mm3, have ascitic fluid cultures demonstrate just a single organism, and have ruled out other forms of peritonitis[7].

Hepatocytes secrete LBP, a soluble acute phase protein with a prolonged half-life that promotes the binding of bacterial lipopolysaccharide (LPS) to the cell membrane molecule CD14 and Toll-like receptor 4, thereby setting off a cascade that results in cytokine production and an inflammatory response. The concentration of LBP is thought to represent chronic exposure to endotoxins and bacteria. The concentration of LBP in the peripheral circulation has been utilized as a proxy for the dissemination of organisms [5].

The demographic data of the current study showed that the mean age was 48.96±5.24 years in group (A) and 49.62±4.46 years in group (B). Regarding gender, group (A) showed that 73.2% were males and 26.8% were females, while group (B) revealed that 66.1% were males and 33.9% were females. In terms of age and gender, there was no significant differences between both groups.

In agreement with the current results, **El Motasem *et al.*[8]**in their study on one hundred twenty Egyptian patients diagnosed with liver cirrhosis and ascites reported that regarding CLD without SBP group, the mean age of cases was 53.31 years old, 60% of cases were males, and 40% of cases were females. While the CLD with SBP group showed mean age of cases was 54.73 years, 51.4% were males, and 48.6% were females. Differences in age and gender distribution between the groups were not statistically significant.

In our study, Inflammatory markers (first hour ESR, second hour ESR and CRP) were significantly higher among group (A) than group (B) with (p< 0.001).The current study was in line with, **El Motasem *et al.*[8]**who found that regarding CLD without SBP group, the mean CRP was 8.64 mg/l. While the CLD with SBP group showed the mean CRP level was 22.2 mg/l. C-reactive protein levels were significantly different between the two groups, with the SBP group showing a marked increase in CLD.

Our results were in accordance with **Yildirim *et al.*[9]** who stated that the CRP levels of the SBP group were significantly higher than those of the non-SBP group in ascitic fluid and serum.

In our study, among group B; there were significant differences between groups regarding plasma glucose, total protein, serum creatinine, serum urea, PT, PTT and INR. These tests were significantly higher among group (A) with (p=0.008) for glucose, (p=0.002) for total protein, (p< 0.001) for serum creatinine, (p< 0.001) for serum urea, (p< 0.001) for PT, (p=0.04) for PTT and (p< 0.001) for INR. While there was no significant difference between groups regarding other parameters.

In accordance with the present findings, **Badawy *et al.*[10]** showed that regarding CLD with SBP group, the mean plasma glucose was 121.05 g/dl, the mean platelets count was 53.2×10^3 cells/mm^3 , the mean total protein was 3.89 g/dl, serum creatinine was 1.62 mg/dl, serum urea was 83.87 mg/dl, total bilirubin was 5.45 mg/dl, direct bilirubin was 3.43 mg/dl, and serum albumin was 2.16 g/dl.

In our study, there was significant difference between groups regarding TLC in ascitic fluid, serum LBP, and ascitic fluid LBP. TLC in ascitic fluid, Serum LBP and ascitic fluid LBP were significantly higher among group (A) with SBP than group (B) with non-SBP with (p< 0.001) for TLC in ascitic fluid, serum LBP and ascitic fluid LBP.

The present results were in line with **Yuan *et al.*[11]**who found that PMNLs count was highly significant in AF of patients with SBP more than the non-SBP group (p < 0.001).

The present results were disagreed with **Agiasotelli *et al.*[5]**as they stated that in CLD without SBP group the mean serum LBP was 13.13 µg/ml. While the CLD with SBP group showed the mean LBP was 13.99 µg/ml. Statistically, we found a major difference in serum LBP levels between the groups.

Our study reported that all parameters were with significant Area Under Curve (AUC) with cutoff >1836, >1817.9 and >18.2 with sensitivity 100.0%, 100.0% and 80.0% respectively and specificity were 100.0%, 100.0% and 79.0% respectively.

Our results showed higher specificity and sensitivity than reported by **Estakhri *et al.*[12]** who reported that ascitic fluid TLC was with significant AUC with cutoff point of 252 with sensitivity of 92.12%, and specificity were 78.57%, NPV of 89.8%, and PPV of 83.1%.

Regarding correlations in group (A), there was statistically significant positive correlation between TLC in ascitic fluid, serum LBP, HB, and negative correlation between TLC in ascitic fluid, CRP, serum glucose, D. bilirubin. There was significant positive correlation between serum LBP, HB, and total protein. Also, a negative remarkable correlation between serum LBP, INR, PTT, PT, serum creatinine, direct bilirubin, total bilirubin, PLT, and ascitic fluid LBP. There was significant positive correlation between ascitic fluid LBP, total bilirubin and AST, and negative correlation between PLT, and serum LBP

Agiasotelli *et al.*[5] agreed with our results when theyreported that leukocyte and neutrophil counts, C-reactive protein, and ascites LBP all increased in tandem with rising serum LBP. Serum C-reactive protein elevation, ascites leukocyte and neutrophil counts, and ascites LDH, albumin, and total protein values all increased considerably in correlation with ascites LBP levels. Furthermore, AST, ALT, total bilirubin, INR levels, MELD scores, and CTP scores were all negatively correlated with LBP in ascitic fluid.

Albillos *et al.*[13]Following 84 cirrhotic patients for a median of 46 weeks, the researchers prospectively

examined the presence of serious bacterial infection in ascitic fluid. In individuals with elevated LBP, the overall chance of infection was 32.4%, but in those with normal LBP, it was only 8% (p = 0.004). Therefore, keeping an eye on LBP may have added value in identifying which ascitic cirrhotic patients will benefit from antibiotic prophylaxis.

A number of research have investigated at how well LBP works as a diagnostic biomarker. Due to the limited size of the samples, the findings from these observational studies have been carefully evaluated. In order to distinguish between SIRS and a bacterial infection, LBP has been shown to be a sensitive and specific marker. A high LBP in the blood could be a great marker of bacterial infection. Elevated LBP concentrations are associated with the development of bacteremia or severe sepsis and septic shock in adult patients in intensive care units. In this investigation, LBP is employed not only for diagnosis but also as a prognostic indicator in determining the likelihood that a septic complication may occur[(14)].

CONCLUSION

It could be concluded that serum LBP demonstrated a highly significant difference between the two groups with a substantial difference as regard the diagnosis of spontaneous bacterial peritonitis, both in terms of sensitivity and specificity. Serum LBP may be considered as a diagnostic tool for SBP in cirrhotic patients with ascites.

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