Study the Diagnostic Performance of Interleukin 17 in Spontaneous Bacterial Peritonitis in Egyptian Patients with HCV-Related Liver Cirrhosis

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Abstract

Background

Spontaneous bacterial peritonitis (SBP) is a severe complication of cirrhosis that requires prompt diagnosis and treatment. Interleukin -17 (IL-17) has been suggested as a potential biomarker for SBP, but its diagnostic value in cirrhotic patients remains uncertain.

Objectives

The study aims to determine the diagnostic value of serum and ascetic IL-17 in SBP detection in Egyptian patients with hepatitis C virus (HCV)- related liver cirrhosis.

Patients and methods

This case-control study enrolled 60 patients with HCV-related liver cirrhosis complicated by ascites, who were divided into two groups based on the presence or absence of SBP. The study was conducted at the Gastroenterology and Hepatology Department of the Internal Medicine Department at Menoufia University Hospitals and Shebin El-Kom Teaching Hospital between May 2021 and July 2022. Ascitic fluid analysis and measurement of serum and ascitic IL-17 levels using ELISA kits were performed at baseline for both groups. After treatment, serum and ascitic IL-17 levels were measured again in the diseased group.

Results

Serum IL-17 exhibited excellent diagnostic performance with an AUC of 0.934, and the best cut-off criterion of > 89 Pg/mL was able to discriminate between patients with SBP and the non-SBP group with a sensitivity of 91.54% and specificity of 96.45%. Similarly, ascitic IL-17 demonstrated excellent diagnostic performance with an AUC of 0.876, and the best cut-off criterion of > 160 pg/mL was able to distinguish patients with SBP from the non-SBP group with a sensitivity of 94.85% and specificity of 95.69%. Moreover, a significant direct correlation was observed between ascitic IL-17 and AST (R=0.263, P=0.003), platelet count (R=0.256, P=0.001), ALT (R=0.627, P< 0.001), bilirubin (R=0.418, P< 0.001), INR (R=0.845, P <0.001), blood urea nitrogen (R=0.257, P=0.001), and creatinine (R=0.294, P< 0.001).

Conclusions

Serum and ascitic IL-17 had excellent diagnostic performance to differentiate SBP from the non-SBP group and in monitoring therapeutic response.

Keywords: Diagnostic, Hepatitis C virus, Interleukin, Liver Cirrhosis, Spontaneous Bacterial Peritonitis.

Introduction

Spontaneous bacterial peritonitis (SBP) is a life-threatening infection that is frequently encountered in cirrhotic patients with ascites, with over 50% of infections occurring in this population (1). In hospitalized patients, SBP has a prevalence as high as 12%, and it develops in up to 3.5% of patients who are treated as outpatients. Patients with SBP have a 10–20% mortality rate, with a high rate of recurrence and poor long-term prognosis, as evidenced by a one-year survival rate of 30-40% and a two-year survival rate of 20% following an episode of SBP (2). The measurement of SBP plays a vital role in monitoring the advancement of liver disease. It is essential to bear in mind this diagnosis whenever a cirrhotic patient experiences clinical decompensation. Early detection and effective treatment of SBP are of utmost importance (3). The pathogenesis of SBP involves immune dysfunction in decompensated cirrhotic patients (DCPs) and the increased susceptibility of the gut mucosa, which allows bacteria and bacterial endotoxins to migrate from the bowel lumen into the ascitic fluid (AF) (4). Additionally, SBP can contribute to various complications associated with cirrhosis, including deterioration of liver function, hepatic encephalopathy, exacerbation of coagulation abnormalities, variceal bleeding, renal failure, and potentially even mortality (5). In previous decades, the mortality rate associated with SBP exceeded 90%. However, thanks to advancements in early detection and appropriate treatment, the mortality rate has significantly reduced to approximately 20% (6). Normally, SBP manifests as abdominal pain accompanied by tenderness and fever. However, it can also exhibit additional symptoms and signs of peritonitis, such as vomiting and ileus. Other indications of systemic inflammation, including hypothermia, chills, rapid heart rate, rapid breathing, and shock, may be present. SBP can lead to a deterioration in liver or kidney function, as well as hepatic encephalopathy. It is worth noting that approximately 10% of cases may not display any symptoms (7).

While a positive AF culture for a specific pathogen is considered the definitive method for diagnosing SBP, approximately 60% of cases that exhibit clinical symptoms suggestive of SBP and an elevated polymorphonuclear leukocytic (PMNL) count in the AF yield negative culture results. As a result, an AF PMNL count of $\geq 250/\mu L$ is used as a diagnostic criterion for SBP, regardless of the culture findings (8). Diagnosing SBP can be difficult as some cases lack typical clinical characteristics. Therefore, it is advisable to pursue early non-invasive methods for diagnosing SBP in decompensated patients with liver disease, particularly in situations where clinical manifestations may not align, newly admitted patients, or individuals experiencing unexplained shock or deterioration in liver function (9).

Interleukin-17 (IL-17) is a pro-inflammatory cytokine involved in the body's defense against extracellular pathogens. Specifically, interleukin-17A, the primary member of the IL-17 family, regulates localized tissue inflammation by attracting neutrophils to sites of infection (10). Intestinal Paneth cells are responsible for producing and releasing a substantial amount of IL-17 during inflammatory processes. This cytokine, IL-17, has been associated with various chronic autoimmune diseases, such as psoriasis, multiple sclerosis, systemic sclerosis, as well as allergic rhinitis (11).

Aim of the work

The study aims to determine the diagnostic value of serum and ascetic IL-17 in SBP detection in Egyptian patients with HCV- related liver cirrhosis.

PATIENTS & METHODS

Study design and participants

This case-control study was done between May 2021 and July 2022 at the Gastroenterology and Hepatology Department of the Internal Medicine Department at Menoufia University Hospital and Shebin El-Kom Teaching Hospital. The study included 60 patients with HCV-related liver cirrhosis complicated by ascites, who were divided into two groups based on the presence or absence of SBP. Group I consisted of 30 patients without SBP, while Group II included 30 patients with SBP. AF analysis (WBCs, neutrophil count, RBCs, and albumin) and measurement of serum and ascitic IL-17 levels were performed at baseline for both groups. After treatment, IL-17 levels were measured again in the diseased group. Serum and ascitic Il-17 were measured using the ELISA kit with catalog no. EH3267 from Wuhan Fine Biotech Co., Ltd. in Hubei, China.

Inclusion and Exclusion Criteria

The study recruited patients who had liver cirrhosis associated with HCV and were also experiencing ascites as a complication. Exclusion criteria involved patients with ascites unrelated to portal hypertension, such as acute decompensated heart failure, pancreatic ascites, hemoperitoneum, peritoneal tuberculosis, hepatocellular carcinoma, patients with chylous ascites, and those who had undergone abdominal surgery within the past month. The diagnostic criteria for SBP included a confirmed history of hepatic disease, as well as investigations indicating the presence of ascites. Additionally, symptoms such as fever, chills, nausea, vomiting, abdominal pain and tenderness, general malaise, altered mental status, and worsening ascites were considered. Furthermore, an ascites polymorphonuclear (PMN) cell count of ≥ 250 PMNs/mm3, with or without a positive ascites culture, was also used as a diagnostic criterion (12).

Treatment for group II

Immediately after the diagnosis of SBP, intravenous cefotaxime 2 gm/8 hours was initiated empirically. A second paracentesis was performed 48 hours after the start of treatment to evaluate the effectiveness of the antibiotic therapy. If the antibiotic therapy was not effective, it was typically due to resistant bacteria, and the antibiotics were modified according to the in vitro susceptibility of the isolated organism or switched to an alternative empiric broad-spectrum antibiotic. The treatment was continued until the PMN count was <250/mm3 for at least 5 days. Albumin infusion was administered at a dose of 1.5mg/kg on the first day, followed by 1mg/kg on the third day, in order to reduce the risk of developing hepato-renal syndrome.

Data Collection and Investigations

Patients included in the study were subjected to a full history taking and thorough clinical examination. The laboratory investigations conducted in this study included liver function profiles, which comprised a comprehensive set of tests such as serum alanine aminotransferase, serum aspartate aminotransferase, total bilirubin, direct bilirubin, serum albumin, serum C-reactive protein (CRP), INR, and prothrombin time, analyzed using a Konelab 20i auto-analyzer from Thermo-electron incorporation in Finland. The complete blood count was also performed at baseline and the end of therapy using an automated cell counter Sysmex KX-21 N from TAO Medical incorporation in Japan, which measured hemoglobin, platelet, white blood cells, and neutrophil count. Additionally, hepatitis markers for screening, namely hepatitis B surface antigen (HBsAg) and hepatitis C antibody (HCV Ab), were evaluated using the Anti-HCV antibodies measurement enzyme immunoassay (EIA) kit from Sanofi Diagnostic Pasteur in France. HCV RNA levels were measured using quantitative (q) real-time (RT)-PCR, while INR and prothrombin time were assayed using Sysmex CS-1600 from Sysmex in Kobe, Japan. Moreover, the Child-Pugh-Turcot score, MELD, and MELD-NA score were calculated.

Radiological investigations were conducted using a General Electric ultrasound machine equipped with a transducer that had a frequency of 3.5 MHz. The purpose of these investigations was to determine the size and echogenicity of the liver, the presence of any hepatic focal lesions, the size of the spleen, the diameter of the portal vein, and the presence of ascites and turbidity.

Ethical considerations

The study procedures adhered to the guidelines set by the Menoufia University Ethical Committee, and written consent was obtained from all participants after explaining the purpose of the study. Informed consent was obtained from all eligible patients, and any unforeseen risks were immediately reported to both the Ethics Committee and the patients involved.

Statistical analysis

The data collected in the study were analyzed using IBM personal computer with Statistical Package of Social Science (SPSS) version 22. The statistical analysis included descriptive statistics and analytical statistics to find the association between the studied factors and the targeted disease. The descriptive statistics presented the quantitative data in the form of the mean ($\bar{X}$), standard deviation (SD), range, and qualitative data in numbers and percentages. The analytical statistics used tests of significance such as the Chi-square test ($\chi 2$) to study the association between two qualitative variables the and Student t-test for comparison between two groups having quantitative variables. A P-value of >0.05 was considered statistically non-significant, and a p-value of <0.05 was considered statistically significant

RESULTS

Among cirrhotic patients without SBP, 56.7% were in Child class B (n=17) and 43.3% were in Child class C (n=13). In contrast, among cirrhotic patients with SBP, 96.7% were in Child class C (n=29) while only 3.3% were in Child class B (n=1) (P=0.001) as shown in Table 1.

Table (1): Clinical features of cirrhotic patients with and without SBP:

Items	patients with SBP before treatment(n=30)	patients without SBP (n=30)	P value
Age (years):			**0.898**
[Mean± SD]	**61.63**±11.08	**60.63**±11.08	
Range	48-77	43-78	
Gender:			**0.553**
Males	20 (66.7)	19 (63.3)	
Females	10 (33.3)	11 (36.7)	
Ascites:			**0.102**
Mild	0(0)	0(0)	
Moderate	6(20.0)	9(30.0)	
Marked	24 (80.0) _	21(70.0)	
Child-Pugh class:			**0.001***
B	1 (3.3)	17(56.7)	
C	29 (96.7)	13 (43.3)	
MELD score:			**0.307**
[Mean± SD]	**17.7±3.08**	**13.66±6**	
Range	7-31	**7-28**	
MELD-NA score:			**0.493**
[Mean± SD]	25.26±5.98	21.93±3	
Range	15-34	15-29	

*Model for End-Stage Liver Disease (**MELD score**). *Model for End-Stage Liver disease-sodium (**MELD-NA score**).

Patients diagnosed with SBP exhibited significantly elevated levels of blood urea and serum creatinine in comparison to those without SBP (P=0.001 and 0.015, respectively). Moreover, patients with SBP had significantly lower levels of eGFR and platelet count compared to those without SBP (P=0.001 and 0.025, respectively). In addition, patients with SBP had significantly higher levels of CRP and serum IL-17 compared to those without SBP (P=0.004 and 0.001, respectively). However, there were no significant differences between patients with and without SBP regarding hemoglobin, INR, leukocytes, AST, ALT, albumin, bilirubin, serum sodium, and potassium (P> 0.005), as shown in Table 2.

Table (2): Laboratory investigations of cirrhotic patients with and without SBP:

Items	patients with SBP before treatment(n=30)	patients without SBP (n=30)	p-value
Hb (g/dl)	8.12±1.5	8.40±1.3	0.922
Leucocyte 10^9/L	6.31±2.1	4.79±2.2	0.518
Platelets 10^9/L	66.73±36.90	86.3±44.00	0.025*
AST (IU/L)	40.16±15.45	30.733±12.00	0.113
ALT (IU/L)	33.8±14.65	26.13±10	0.161
Albumin (g/dl)	2.17±0.51	2.196±0.5	0.986
bilirubin (mg/dl)	1.97±0.81	1.92±0.9	0.971
INR	1.42±0.60	1.44±0.4	0.986
S. Urea (mg/dl)	72.23±30	42.7±14	0.001*
Creatinine (mg/dl)	1.97±1	1.07±0.3	0.015*
eGFR mL/min/	40.32±23.4	69.66±29.3	0.001*
CRP mg/L	13.1±7.34	4.66±3	0.004*
Sr Sodium mg/L	123.03±9.34	126.33±7	0.767
Sr Potassium mg/L	3.98±1.6	3.38±0.7	0.754
serum IL17 pg/mL	618.83±247.72	52.076±22.54	0.001*

*Hemoglobin (Hb). *Aspartate aminotransferase (AST). *Alanine aminotransferase (ALT). * International normalized ratio (INR). *Estimated Glomerular Filtration Rate (eGFR). * C-reactive protein (CRP). * Serum interleukin 17(s.IL17).

Total leukocyte count and mean ascitic IL-17 were significantly higher in patients with SBP compared to patients without SBP with a P-value of 0.001 for both, as shown in Table 3.

Table (3): Laboratory measures of AF samples of cirrhotic patients with and without SBP:

Items	patients with SBP before treatment(n=30) Mean± SD	patients without SBP (n=30) Mean± SD	Mann-Whitney test	p-value
Total leucocyte count cells/µL	1377.666±314.54	221± 120	62.582	0.001*
mean ascitic IL17 pg/mL	639.748± 247.9	53.97± 28.94	57.524	0.001*

*Interleukin 17 (IL17).

MELD and MELD-NA scores were significantly improved after treatment of SBP (P= 0.017 and 0.036 respectively), (Table 4).

Table (4): Clinical characteristics of SBP patients concerning treatment:

Items	SBP patients		χ^2	p-value
	Before treatment (n=30) No (%)	After treatment (n=30) No (%)		
Ascites:				
Mild	0(0)	0(0)	2.666	0.102
Moderate	6(20.0)	9(30.0)		
Marked	24 (80.0) _	21(70.0)		
Child-Pugh class:				
B	1 (3.3)	0(0)	3.045	0.080
C	29 (96.7)	30 (100.0)		
MELD score				
[Mean± SD]	17.7±3.08	14.96±6	0.25	0.017*
range	7-31	7-28		
MELD-NA score:				
[Mean± SD]	25.26±5.98	22.76±5	0.382	0.036*
Range	15-34	11-33		

*Model for End-Stage Liver Disease (MELD **score**). *Model for End-Stage Liver disease-sodium (MELD-**NA score**).

After SBP treatment, there was a significant decrease in blood urea, CRP, and serum IL-17 levels with p-values of 0.001, 0.001, and 0.001 respectively, as shown in Table 5. Additionally, there was a significant reduction in total leukocyte count in ascitic fluid and mean ascitic IL-17 levels after SBP treatment, with p-values of 0.001 for both, as demonstrated in Table 6.

Table (5): Laboratory investigations of SBP patients before and after treatment:

Items	Before treatment (n=30)	After treatment (n=30)	t-test	p-value
Hb (g/dl)	8.12±1.5	8.70 ±0.7	0.041	0.838
Leucocyte 10^9/L	6.31±2.1	4.37 ±2.2	0.8	0.371
Platelets 10^9/L	66.73±36.90	66.56 ±34.00	0.004	0.983
AST (IU/L)	40.16±15.45	35.03 ±11.00	0.666	0.414
ALT(IU/L)	33.8±14.65	30.33 ±12	0.285	0.593
Albumin (g/dl)	2.17±0.51	2.21 ±0.4	0.007	0.978
Bilirubin (mg/dl)	1.97±0.81	1.65 ±0.7	0.056	0.812
INR	1.42±0.60	1.319 ±0.4	0.008	0.925
B.Urea (mg/dl)	72.23±30	48.6 ±25	9.6	0.001*
S.Creatinine (mg/dl)	1.97±1	1.66 ±0.6	0.052	0.818
eGFRmL/min/	40.32±23.4	46.87 ±23.1	0.837	0.360
CRP mg/L	13.1±7.34	3.96 ±2	9.8	0.001*
S.Sodium mg/L	123.03±9.34	125.8 ±7	0.032	0.857
S.Potassium mg/L	3.98±1.6	3.416 ±0.9	0.091	0.761
serum IL17 pg/mL	618.83±247.72	157.23 ±85.77	15.97	0.001*

*Hemoglobin (Hb). *Aspartate aminotransferase (AST). *Alanine aminotransferase (ALT). * International normalized ratio (INR). *Estimated Glomerular Filtration Rate (eGFR). *C-reactive protein (CRP). *Serum interleukin 17(s.IL17).

Table (6): Laboratory measures of AF samples of SBP patients concerning treatment:

Items	SBP patients		Mann-Whitney test	p-value
	Before treatment (n=30) Mean± SD	After treatment (n=30) Mean± SD		
Total leucocyte count cells/µL	1377.666±314.54	243.06±130	18.532	0.001*
mean ascitic IL17 pg/mL	639.748± 247.9	208.74± 77.47	16.425	0.001*

The diagnostic performance of serum IL-17 was excellent with an AUC of 0.934. A cut-off value of serum IL-17 > 89 Pg/mL was found to be the best criterion for discriminating between patients with SBP and the non-SBP group, with a sensitivity of 91.54% and specificity of 96.45%. Similarly, ascitic IL-17 had an excellent diagnostic performance with an AUC of 0.876. A cut-off value of ascitic IL-17 > 160 pg /mL was found to be the best criterion for discriminating between patients with SBP and the non-SBP group, with a sensitivity of 94.85% and specificity of 95.69% (Figure 1 & 2 and Table 7).

Table (7): ROC curve for serum IL17 and ascetic IL17 as a diagnostic test for SBP:

Cut off point	AUC	Sensitivity	Specificity	+PV	-PV	accuracy
Serum IL17						
>89	0.934	91.54	96.45	93.0	80.3	94.25
Ascetic IL17						
>160	0.876	94.85	95.69	86.2	81.1	95.00

AUC: area under the curve. +PV: positive predictive value. -PV: negative predictive value.

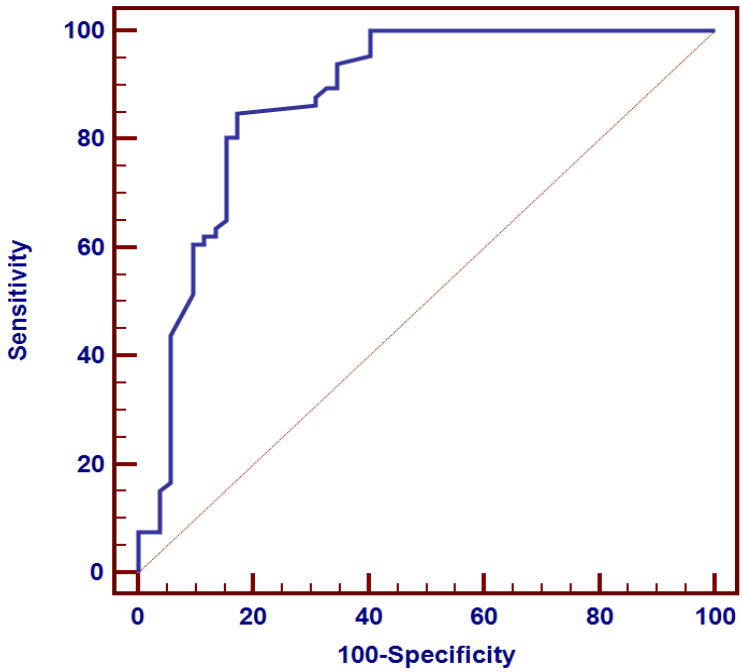

Figure (1): Sensitivity and specificity of serum IL-17 in the diagnosis of S.B.P.

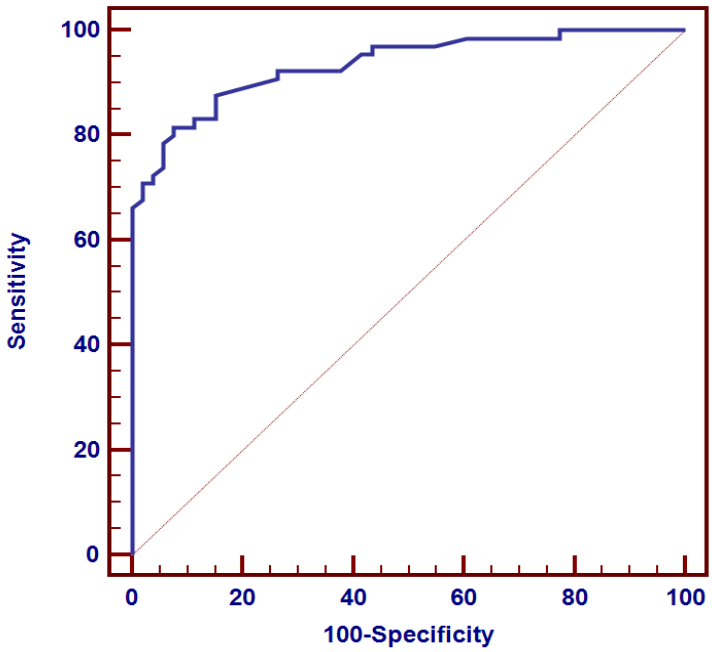

Figure (2): Sensitivity and specificity of total leucocyte count in AF and ascitic IL-17 in the diagnosis of S.B.P.

After conducting univariate analysis, it was found that higher levels of ascitic IL-17, serum IL-17, CRP, and Child-Pugh class were significant predictors of a poor outcome in patients with SBP, with p-values of 0.029, 0.001, 0.047, and 0.001, respectively. However, upon conducting multiple logistic regression analyses, only serum IL-17 was found to be a significant predictor of a bad outcome in cirrhotic patients with SBP with a p-value of 0.002, (Table 8).

Table (8): Univariate and multiple logistic regression analysis for predictors of outcome

		Univariate			
		B	**P-value**	**OR**	**95% CI**
Univariate analysis					
Child-Pugh class		0.275	0.001*	0.760	0.655-0.882
MELD score		0.085	0.997	0.551	0.424-0.623
mean ascitic IL17 pg/mL		0.044	0.029*	0.957	0.920-0.996
Total ascetic leucocyte count	cells/µL	0.009	0.786	0.991	0.932-1.05
serum IL17 pg/mL		1.48	0.001*	4.39	1.87-10.27
CRP mg/L		0.133	0.047*	1.01	0.995-1.01
Multiple analysis					
Child-Pugh class		0.566	0.997	0.123	0.112-0.23
MELD score		-----	-----	----	----
mean ascitic IL17 pg/mL		0.041	0.051	0.960	0.920-1.00
Total ascetic leucocyte count	cells/µL	-----	-----	----	----
serum IL17 pg/mL		1.38	0.002*	4	1.67-9.60
CRP mg/L		-----	-----	----	----

Table 9 shows a significant positive correlation between ascitic IL-17 and AST (R=0.263, P=0.003), platelet count (R=0.256, P=0.001), ALT (R=0.627, P<0.001), bilirubin (R=0.418, P<0.001), INR (R=0.845, P<0.001), blood urea (R=0.257, P=0.001), and serum creatinine (R=0.294, P<0.001). However, there was no correlation between ascitic IL-17 and age, hemoglobin, leukocytes, serum albumin, estimated glomerular filtration rate (eGFR), serum sodium, and potassium (P>0.005).

Table (9): Correlation between mean ascitic IL-17 pg/mL and different parameters in the SBP group before treatment

	R	P
Age (years)	0.188	0.428
Hb (g/dl)	-0.014	0.952
Leucocyte 10^9/L	**-0.134**	**0.353**
Platelets 10^9/L	0.256	0.001*
AST (IU/L)	0.263	0.003*
ALT (IU/L)	0.627**	< 0.001*
Albumin (g/dl)	0.333	0.152
bilirubin (mg/dl)	0.418	< 0.001*
INR	**0.845**	**<0.001***
B.Urea (mg/dl)	0.257	0.001*
S.Creatinine (mg/dl)	0.294	< 0.001*
eGFRmL/min/	0.019	0.920
CRP mg/L	0.307	0.099
S.Sodium mg/L	0.026	0.860
S.Potassium mg/L	-0.211	0.142

DISCUSSION

The mean age of the patients in the present study was consistent with previous studies (13). Male patients were predominant, which was in agreement with previous research (3). The development of SBP was observed only in patients with Child classes B and C, indicating that SBP develops more frequently in patients with advanced liver disease. Most of the studied cases were in the Child-Pugh C stage, which is consistent with previous studies (13,14,15). The severity of liver cirrhosis was related to an increased probability of SBP, which was consistent with the conclusions of previous studies (15,16,17).

The study found that the total leucocyte count did not significantly differ between SBP and non-SBP groups, which partially agrees with Keryakos et al.'s findings (18). However, platelet count was significantly lower in the SBP group, consistent with El Motasem et al.'s findings (14).

Within the scope of this study, the SBP group demonstrated notably higher levels of blood urea and serum creatinine in comparison to the non-SBP group. Additionally, the SBP group exhibited a significantly lower estimated glomerular filtration rate (eGFR), suggesting the presence of renal dysfunction. These outcomes align with prior research conducted by Keryakos et al (18), who observed that serum creatinine and blood urea were higher, and eGFR was lower in cirrhotic patients with SBP compared to those without SBP before treatment. The implementation of effective treatment resulted in a substantial decrease in serum creatinine and blood urea levels, as well as an improvement in eGFR. The occurrence of renal dysfunction in SBP has been extensively documented in the existing literature (19,20).

In the SBP group, the CRP level was elevated compared to the non-SBP group, aligning with previous research findings (15). SBP patients had a mean MELD score of 17.7±3.08, and each additional point on the MELD scale was associated with an 11% increased risk of SBP (21). Gram-negative bacteria were identified as the predominant causative agents of infection, with Escherichia coli being the most frequently encountered pathogen.

AF analysis and measurement of serum and ascitic IL-17 levels were performed at baseline for both groups. After treatment, IL-17 levels were measured again in the diseased group, we found that the levels of ascitic and serum IL-17 were significantly higher in the SBP group compared to the non-SBP group before treatment. However, after treatment, there was a significant decrease in the levels of both ascitic and serum IL-17 in the SBP group. These findings suggest that the IL-17 pathway may play a role in the pathogenesis of SBP and may serve as a potential therapeutic target. When comparing our findings with other studies, we found that our results are consistent with those of Keryakos et al. (18) who also found that serum IL-17 levels were significantly higher in patients with SBP compared to those without SBP. Moreover, they demonstrated that after effective treatment, the levels of IL-17 decreased significantly. These findings support the potential role of IL-17 as a therapeutic target in the management of SBP.

Finally, there are some limitations to this study that need to be acknowledged. Firstly, the sample size was relatively small, which could limit the generalizability of the findings to other populations. Secondly, the study was conducted in a single center, which may limit the generalizability of the results to other settings. Thirdly, this study only focused on IL-17 levels and did not investigate other cytokines or biomarkers that could potentially be associated with SBP. Lastly, the study did not evaluate the long-term outcomes of patients with SBP, such as the risk of developing hepatorenal syndrome or mortality.

Conclusion

The results of this study suggest that ascitic and serum IL-17 levels could be useful diagnostic markers for SBP. Both ascitic and serum IL-17 levels had good discriminatory power with high sensitivity and specificity values. The findings of this study also indicate that IL-17 levels decrease significantly after effective treatment for SBP. However, further studies are needed to validate these findings and to investigate the potential use of IL-17 as a prognostic marker for SBP.

LIST OF ABBREVIATIONS

SBP, Spontaneous bacterial peritonitis; PMN, Polymorphonuclears; DCPs, Decompensated cirrhotic patients; AF, Ascitic fluid; PMNL, Polymorphonuclear leukocytic; IL-17, Interleukin-17; BMI, Body mass index; CRP, C-reactive protein; HBsAg, hepatitis B surface antigen; HCV Ab, hepatitis C antibody; EIA, enzyme immunoassay.

DATA SHARING STATEMENT

All data and materials included in this work are available

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

Our local Ethics Committee approved our study and a written consent for participation was obtained from all patients.

CONSENT FOR PUBLICATION

Not applicable

HUMAN AND ANIMAL RIGHTS

No animals were used for studies that are the basis of this research. All the humans were used in accordance with the Helsinki Declaration of 1975.

AUTHORS' CONTRIBUTIONS

All authors played a substantial role in the research described, including contributions to the conception, study design, data acquisition, analysis, and interpretation. They were actively involved in drafting, revising, and critically reviewing the article. The final approval of the version to be published was granted by all authors. They have collectively agreed upon the choice of journal for submission and have accepted responsibility for all aspects of the work.

DISCLOSURE

The authors have disclosed no conflicts of interest regarding this study.

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