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Procalcitonin, Erythrocyte Sedimentation Rate and C- Reactive Protein Index as A Predictor for Spontaneous Bacterial Peritonitis in Decompensated Cirrhotic Patients

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ABSTRACT

Background: The most reliable way to diagnose Spontaneous bacterial peritonitis [SBP] is with a positive ascitic fluid culture for a pathogen. However, it is invasive method. Several studies had reported another noninvasive tool for the diagnosis of SBP.

The Aim of the work: the aim of the study is to evaluate the predictive value of PEC index in diagnosis of spontaneous Bacterial Peritonitis, which made by measurement of procalcitonin, ESR and CRP, which we called the PEC index as a predictive value in Decompensated liver disease ascites.

Patients and Methods: This study was carried on 120 patients with cirrhosis. All patients were selected from Internal Medicine Department at Al-Azhar University Hospital New Damietta. All patients were divided into the following groups: Group [A] include: Forty [40] patients with decompensated cirrhosis having SBP. Group [B] include: Forty [40] patients with decompensated cirrhosis having sterile ascites. Group [c] include: Forty [40] patients with compensated cirrhosis without ascites.

Results: We found that PCT in 0.48ng/mL cutoff level had sensitivity of 86% and specificity of 92%. ESR in 30.5 cutoff level had sensitivity of 84% and specificity of 64.4%. CRP in 23.5 cutoff level had sensitivity of 88% and specificity of 75.6%. PEC in 25 cutoff level had sensitivity of 92% and specificity of 97.8%.

Conclusion: The PEC index is effective in diagnosis of spontaneous Bacterial Peritonitis, which made by measurement of procalcitonin, ESR and CRP, which we called the PEC index as a predictive value in Decompensated liver disease ascites.

Keywords: Procalcitonin; Erythrocyte Sedimentation Rate; C- Reactive Protein; Liver Cirrhosis.

INTRODUCTION

Spontaneous bacterial peritonitis [SBP] represents the most prevalent and perilous infection observed in individuals affected with cirrhosis, specifically those who exhibit ascites. It is responsible for over 50% of all infections. The global prevalence of SBP was determined to be 17.12%. It was observed that the highest prevalence was recorded in the African region, with a rate of 68.20%. Conversely, the lowest prevalence was noted in North America, with a rate of 10.81% [1].

The immune dysfunction observed in individuals with decompensated cirrhosis, commonly referred to as DCPs, in conjunction with the susceptibility of the gut mucosa, plays a significant role in the development of SBP. This condition is characterized by the migration of bacteria and bacterial endotoxins from the intestinal lumen into the ascitic fluid [AF], thereby contributing to the underlying mechanisms involved in the pathogenesis of SBP [2].

The occurrence of SBP is known to trigger a cascade of additional complications associated with cirrhosis. These complications include but are not limited to hepatic impairment, hepatic encephalopathy, exacerbation of coagulopathy, variceal bleeding, renal failure, and potentially fatal outcomes [3].

It has been reported that SBP was linked to a mortality rate of over 90%, but with the advancement of timely diagnosis and effective treatment, that rate has now dropped to less than 20% [4]. SBP typically manifests as fever-related abdominal pain and tenderness. SBP may also be asymptomatic in 10% of cases. Nevertheless, it can also present with other local symptoms and signs of peritonitis, such as vomiting and ileus; other manifestations of systemic inflammation, such as hypothermia, chills, tachycardia, tachypnea, and shock; worsening of liver or kidney functions; or hepatic encephalopathy [5].

The most reliable way to diagnose SBP is with a positive ascitic fluid culture for a pathogen. However, this is not always the case; in fact, negative cultures are found in approximately 60% of cases with SBP-like symptoms and an elevated PMNL count in the ascitic fluid [6].

In numerous studies, non-invasive methods such as clinical scores, fecal calprotectin, and various serum inflammatory cytokines and chemokines like monocyte chemotactic protein-1, interleukin-10, human neutrophil peptide, platelet indices, macrophage inflammatory protein-1 beta, interferon-γ-induced protein-10, tumor necrosis factor-α, interleukin-6, and procalcitonin were attempted as alternatives to diagnostic para-centesis [7].

Procalcitonin [PCT] is a polypeptide consisting of 116 amino acids. It serves as a precursor to calcitonin and has a molecular weight of 13 KDa. PCT is synthesized by cells outside of the thyroid, such as monocytes. Highly cited studies have suggested that it could be a valuable serum biomarker for diagnosing bacterial infections in general, and specifically for SBP [8].

Typically, the PCT level is not measurable [< 0.01 ng/mL], but it quickly rises in the presence of an infection. Despite the relatively high reported average estimates of sensitivity and specificity [83% and 92%, respectively] for serum PCT in diagnosing SBP in various clinical trials, this level of performance was inadequate for achieving a reliable and accurate diagnosis [9].

So, the aim of the study is to evaluate the predictive value of PEC index in diagnosis of spontaneous Bacterial Peritonitis, which made by measurement of procalcitonin, ESR and CRP, which we called the PEC index as a predictive value in Decompensated liver disease ascites.

PATIENTS AND METHODS

This study was carried on 120 patients with cirrhosis. All patients were selected from Internal Medicine Department at Al-Azhar University Hospital New Damietta. All patients were divided into the following groups: Group [A] include: Forty [40] patients with decompensated cirrhosis having SBP. Group [B] include: Forty [40] patients with decompensated cirrhosis having sterile ascites. Group [c] include: Forty [40] patients with compensated cirrhosis without ascites. Our study approved from the ethical committee of our institution. Written consent was obtained from every patient.

The confirmed diagnosis of SBP was achieved by an AFPMNL count $\geq 250/mm^3$ with or without a positive culture of the ascitic fluid for pathogenic bacteria. Absence of both criteria meant the ascites was sterile.

Inclusion criteria were specific for each group as group A included patients with decompensated cirrhosis with confirmed diagnosis of SBP, while group B included patients with decompensated cirrhosis with sterile ascites and group C included patients with compensated cirrhosis and absent ascites. In addition, only adult patients [age > 18 years] were included.

The Exclusion criteria were: 1] Patients with infections other than those specifically affecting the Ascitic fluid. 2] Patients diagnosed with hepatocellular carcinoma [HCC] or any related pancreatic condition. 3] Patients who were administered antibiotics within 10 days before being admitted to the hospital. 4] Patients who have a positive culture of Ascitic fluid and a count of Ascitic fluid-PMNL less than 250/mm^3.

All patients were subjected to the following; full history taking and complete clinical examination. Laboratory investigations which included ALT [0-40 u/l], AST [0-40 u/l], total bilirubin [0.2-1.2 mg/dl], direct bilirubin [0-0.3 mg/dl] and albumin [3.5-5.2 g/dl], Prothrombin time [10-14 sec.], concentration and INR, Urea [10-50 mg/dl], creatinine [0.6-1.4 mg/dl] , CBC, ESR [first hour] [3-20 mm/h], CRP [0-6 mg/dl], viral markers [HCV ab, HBs Ag], Serum procalcitonin level [<0.05 ng/dl], peritoneal fluid examination: PMNL [cell/mm^3], protein, LDH [140-280 u/l]. PEC index: Procalcitonin [ESR+CRP] 4], and abdominal ultrasonography.

Peritoneal fluid examination: Sterile bedside diagnostic paracentesis was done. A 23-G needle was attached to a 20-cc syringe after application of local anesthesia. Then, aspirated ascitic fluid was collected into two tubes and analyzed within 2 h of aspiration. The first tube for culture and sensitivity and second tube with ethylene diamine tetraacetic acid were to be analyzed for biochemistry and leukocyte [PMNL] counts.

PCT measurement: This was done by the electrochemiluminescence immunoassay [ECLIA] [Cobas e 411 immunoassay analyzers, Roch, Germany], acting via the sandwich principle. It calculates the analyte concentration of each sample in ng/ml in an automatic fashion with a measuring range of 0.02 to 100 ng/ml and the coefficient of variability of 8%.

PEC index: This is a serum bioscore, calculated by the formula; PEC index = PCT × [ESR + CRP].

Statistical analysis: All data analysis was done using the SPSS version 25. Categorical data were presented as numbers and percentages and were compared using the Chi-Square Test. The normality of continuous data was initially checked by the Shapiro-Wilk test. All continuous data were not parametric, so we present it as Interquartile range and median [IQR]. Within-group comparison was done using the Kruskal-Wallis test, and every 2 groups were compared using the Mann-Whitney U-test.

RESULTS

In the current work, patients were mainly in the sixth decade of life and majority were males [85.3%]. Hypertension reported for 52% of all patients and diabetes for 47.5%. the causes of liver diseases were hepatitis C-virus infection, bilharziasis, concomitant HCV and bilharziasis, then NAFLD and finally hepatitis B and autoimmune diseases. Groups were comparable regarding patient demographics, except for significant differences regarding cause of liver disease [Table 1].

Table [2] presented the laboratory data among study groups. There were no significant differences between groups regarding liver enzymes [ALT and AST] and serum albumin. In addition, there was no significant differences were recorded regarding hemoglobin concentration. Otherwise, there were significant differences regarding INR, total and direct bilirubin, serum urea and creatinine, TLC, platelets, ESR, CP, PCT, PMNL and PEC. The PCT was significantly increased in group B than group A than group C [0.945 ± 0.732, 0.546 ± 0.650 and 0.126± 0.12, respectively]. The PMNL was absent in C-group and significantly increased in B than A groups [2185± 1850.8 vs 55± 49, respectively]. Finally, the PEC was significantly higher in B, than A and C groups [96± 32 vs 32.9± 18 and 2.4± 1.6, successively].

Table [3] presented comparison between groups A and C regarding laboratory investigations. Results showed that, both groups were comparable regarding serum albumin, INR, AST, ALT, TLC, hemoglobin, and platelet count. Otherwise, total and direct bilirubin, creatinine and urea, ESR, CRP, PCT, and PEC were significantly higher among A than C group.

In addition, there was a statistically significant increase of total and direct bilirubin, INR, AST, urea and creatinine, TLC, ESR, CRP, PCT and PEC, but significant decrease of platelets in B than C groups [Table 4].

Comparing A and B groups, revealed significant reduction of platelets and significant increase of total and direct bilirubin, INR, urea and creatinine, TLC, ESR, CRP, PCT, PMNL, and PEC in B than A group [Table 5].

The ascitic PMNL showed statistically significant, proportional correlation with TLC [r =0.724, p < 0.001] and PEC [r = 0.595, p = 0.005]. Otherwise, no significant correlation was recorded for PCT, CRP or ESR [Data not tabulated].

Table [6] and figure [1] revealed the sensitivity and best cutoff of different markers. For example, PCT at 0.48 had sensitivity of 86% and specificity of 92.20%. However, the best sensitivity was recorded for PEC [92.0%] followed by CRP [88.0%]. In addition, the best specificity was recorded for PEC [97.8%] followed by PCT [92.20%].

Table [1]: Demographic Data among study groups

	Variables	Total [n=120]	A[n=40]	B[n=40]	C [n=40]	P value
Age [years]	Mean ± SD	59 ± 9.7	62.6±7.5	55.9±7.3	58.4±5.4	**0.06**
Sex, n [%]	Male	70 [58.3]	23[32.8]	19[27.5]	28[40]	0.012
	Female	50 [41.7]	17[42.5]	21[52.5]	12[30]	
Comorbidities, n [%]	HTN	63[52]	15[23]	26[41]	22[34]	0.054
	DM	57[47.5]	24[42]	26[45]	7[12]	0.78
Cause of Liver disease, n [%]	Bilharziasis	18 [15]	6[33]	1[5]	11[66]	0.04*
	HCV	82[68.3]	31[37.8]	32[39]	19[23]	
	HCV and Bilharziasis	10 [8.3]	0[0]	6[60]	4[40]	
	HBV	2 [1.6]	0[0]	1 [50]	1[50]	
	Autoimmune	2 [1.6]	1 [50]	0 [0]	1 [50]	
	NAFLD	6 [5]	2 [33]	0 [0]	4 [66]	

NB: Percentages in groups were calculated from the total of each variable [not from the groups]. HTN: hypertension; DM: Diabetes Mellitus; HCV: Hepatitis -C virus; HBV: Hepatitis B virus; NAFLD: non-alcoholic fatty liver disease. * Indicate significant differences.

Table [2]: Results of laboratory investigations among study groups

	Total	A	B	C	P value
AST [u/l]	41.9± 34.5	46.7± 58.7	45.3± 8.5	34± 5.3	0.20
ALT[u/l]	39.3± 18.9	35± 23.7	32.5± 8.5	30 ± 10.9	0.20
Albumin [g/dl].	2.8± 0.6	2.8± 0.4	2.6± 0.4	3.5±0.3	0.07
INR	1.4± 0.4	1.2± 0.2	1.8±0.4	1.1±0.1	<0.001*
Total Bilirubin [mg/dl]	3.1± 3.1	2± 1.3	4± 2.5	0.8±0.4	<0.001*
Direct Bilirubin[mg/dl]	1.3± 1.5	0.9± 0.6	1.9± 0.5	0.2±0.1	<0.001*
Cr [mg/dl]	1.2± 1.02	1± 0.3	1.7± 1.6	0.7± 0.1	<0.001*
Ur [mg/dl]	48.9± 23.2	47± 12	71± 10	32 ±11	<0.001*
TLC	9.5± 3.9	7.3± 2.7	13.2± 3.3	7.9±2.6	<0.001*
HGB [g/dl]	10.2± 1.7	9.3± 2.1	8.7± 3	11.1± 1.8	0.44
PLT	110.9± 40.4	111.7±40.7	96.6± 49.1	134.5±22.9	0.01*
ESR [mm/h]	43.9± 25.5	45.6± 27.2	57.1± 20	20±13	0.01*
CRP [mg/dl]	31.6± 35.2	20.3± 16.7	50± 25.9	4± 3	<0.001*
PCT [ng/dl]	0.576± 0.670	0.546 ± 0.650	0.945 ± 0.732	0.126± 0.12	<0.001*
PMNL[cell/mm3]	2140.08±1644.2	55± 49	2185± 1850.8	0	<0.001*
PEC	44.1± 30	32.9± 18	96± 32	2.4± 1.6	<0.001*

AST: aspartate transaminase; ALT: Alanine transaminase; INR: International normalization ration; Cr: Creatinine; Ur: Urea; TLC: total leucocytic count; HGB: hemoglobin; PLT: platelets; ESR: Erythrocyte sedimentation rate; CRP: C-reactive protein; PCT: procalcitonin; PMNL: polymorphonuclear leucocytes; PEC: Procalcitonin, Erythrocyte Sedimentation Rate And C- Reactive Protein

Table [3]: Comparison of Laboratory test between Group C and A

	A	C	P value
Albumin[g/dl]	2.8± 0.4	3.5±0.3	0.09
Total bilirubin[mg/dl]	2± 1.3	0.8±0.4	0.01*
Direct bilirubin[mg/dl]	0.9± 0.6	0.2±0.1	0.01*
INR	1.2± 0.2	1.1±0.1	0.15
AST[u/l]	46.7± 58.7	34± 5.3	0.18
ALT[u/l]	35± 23.7	30± 10.9	0.20
Cr [mg/dl]	1± 0.3	0.7± 0.1	0.01*
Ur [mg/dl]	47± 12	32 ±11	0.01*
TLC	7.3± 2.7	7.9± 2.6	0.35
HG [g/dl]	9.3± 2.1	11.1± 1.8	0.65
PLT	111.7±40.7	134.5± 22.9	0.09
ESR [mm/h]	45.6± 27.2	20±13	**0.04***
CRP [mg/dl]	20.3± 16.7	4± 3	**0.02***
PCT [ng/dl]	0.546±0.650	0.126± 0.12	**0.01***
PMNL [cell/mm3]	55± 49	0	
PEC	32.9± 18	2.4± 1.6	**<0.001***

Table [4]: Comparison of Laboratory test between Group C and B

	C	B	P value
Albumin[g/dl]	3.5±0.3	2.6± 0.4	0.07
Total bilirubin[mg/dl]	0.8±0.4	4± 2.5	0.01*
Direct bilirubin[mg/dl]	0.2±0.1	1.9± 0.5	0.01*
INR	1.1±0.1	1.8±0.4	0.01*
AST[u/l]	34± 5.3	45.3± 8.5	0.07
ALT[u/l]	30.4± 10.9	32.5± 8.5	0.20
Cr [mg/dl]	0.7± 0.1	1.7± 1.6	0.01*
Ur [mg/dl]	32 ±11	71± 10	0.01*
TLC	7.9± 2.6	13.2± 3.3	0.01*
HG [g/dl]	11.1± 1.8	8.7± 3	0.25
PLT	134.5± 22.9	96.6± 49.1	0.002*
ESR [mm/h]	20±13	57.1± 20	0.01*
CRP [mg/dl]	4± 3	50± 25.9	0.02*
PCT [ng/dl]	0.126± 0.128	0.945± 0.732	0.01*
PMNL [cell/mm3]	0	2185± 1850.8	
PEC	2.4± 1.6	96± 32	<0.001*

Table [5]: Comparison of Laboratory test between Group A and B

	A	B	P value
Albumin[g/dl].	2.8± 0.4	2.6± 0.4	0.07
Total bilirubin[mg/dl]	2± 1.3	4± 2.5	**<0.001***
Direct bilirubin[mg/dl]	0.9± 0.6	1.9± 0.5	**<0.001***
INR	1.2± 0.2	1.8±0.4	**<0.001***
AST[u/l]	46.7± 58.7	45.3± 8.5	0.20
ALT[u/l]	35± 23.7	32.5± 8.5	0.20
Creatinine [mg/dl]	1± 0.3	1.7± 1.6	**<0.001***
Urea [mg/dl]	47± 12	71± 10	**<0.001***
TLC	7.3± 2.7	13.2± 3.3	**<0.001***
HG [g/dl]	9.3± 2.1	8.7± 3	0.44
PLT	111.7±40.7	96.6± 49.1	**0.01***
ESR [mm/h]	45.6± 27.2	57.1± 20	**0.01***
CRP [mg/dl]	20.3± 16.7	50± 25.9	**<0.001***
PCT [ng/dl]	0.546±0.650	0.945± 0.732	**<0.001***
PMNL [cell/mm3]	55± 49	2185± 1850.8	**<0.001***
PEC	32.9± 18	96± 32	**<0.001***

Table [6]: Cutoff Value of serum marker

	Cutoff	Sensitivity	Specificity	P value
PCT	0.48	86%	92.20%	0.001
ESR	30.5	84%	64.40%	0.001
CRP	23.5	88%	75.60%	0.001
PEC	25	92%	97.8%	0.001

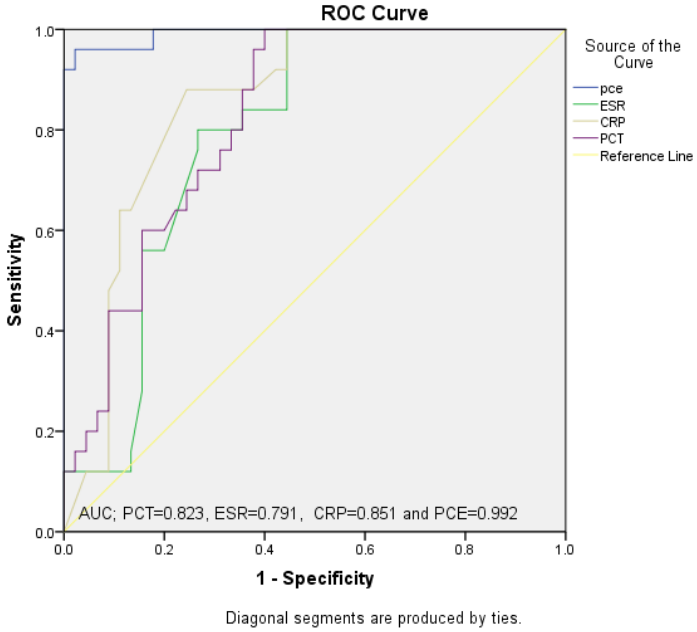

Figure 1: ROC curve of Sensitivity and specificity of serum marker cutoff point

DISCUSSION

In our study, we included 120 Egyptian patients with cirrhosis, 40 of them had an SBP, and other 40 had clear ascites and the last 40 are without ascites. The most prevalent cause of liver cirrhosis in our study was HCV, which represent 68% of the participants, followed by Bilharziasis [15%], followed by mixed HCV and Bilharziasis [8.3%], followed by non-alcoholic fatty liver disease [5%], followed by HBV [1.6%] and Autoimmune hepatitis [1.6%]. This is in agreement with the previous study done by **Elbahrawy *et al.*** [10], who reported that viral hepatitis [HCV] is the main cause of chronic hepatitis and liver cirrhosis in Egypt. The cause of liver cirrhosis was significantly different between the study groups [P value < 0.04].

In our study, the mean and SD age of the patients was around 60 years old, which is in line with the results of **Popoiag *et al.*** [11] who found that the mean and SD of the patients with SBP was 59.29±11.30.

As regard the age in our study there is no significant difference between the 3 groups.

In terms of the gender distribution of the participants, we found that male with SBP was more prevalent than females, which agreed with the findings of **Popoiag *et al.*** [11] who found that 66.7% of the patients with SBP were male. This male predominance may be explained by that, estrogen in females may have a protective role against fibrosis in chronic liver disease patients by inhibiting stellate cells, which are responsible for fibrogenesis in the liver [12].

Regarding Billirubin, we found that the total and direct billirubin were significantly higher in group the SBP patients [P value = 0.001] which is in line with **Elsadek *et al.*** [13].

In terms of renal functions, we found a significant elevation in the level of urea and creatinine in the group of SBP [group B] in comparison with group A and C. This renal impairment in patients with SBP occurred due to that, the bacterial endotoxins and inflammatory cytokines in patients with SBP will increase the nitric oxide production in the systemic circulation via bacterial translocation, impairing the hepatic clearance and portosystemic shunting. This arterial and splanchenic vasodilation,

causing decreased effective perfusion of the kidneys [14].

In terms of cytological examination of ascetic fluid aspiration, Patients in group B had ascetic fluid inflammatory response due to bacterial infection which was identified by AF PMNL count ≥ 250/HPF, which is similar to the results of **Elsadek *et al.*** [13].

Also, other serum levels of acute phase reactants; ESR, CRP, and PCT were significantly higher in group B than in groups A, and C [P value < 0.001 for all]. This is in line with previous studies [13, 15, 16]. This could be explained by SBP results in the activation of cytokine synthesis and innate immunity in response to circulating bacterial endotoxins [17].

As regard to PCT in our study there is significant increase in group B [0.945 ± 0.732] in comparison with group A [0.546 ± 0.650] and C [0.126± 0.12] with P value=0.00.

PCT elevation in group B confirms the fact that PCT is elevated in acute bacterial infections and reaches the highest serum level in severe infections and sepsis as it is enhanced by a systemic inflammatory response. However, is not elevated by a viral infection or autoimmune inflammation. So, it can be used for the differentiation between viral, and bacterial infections [18, 19].

Procalcitonin has been considered as a novel index of inflammation marker of bacterial infection [20].

In our study the area under the curve for procalcitonin in predicting SBP [0.82], are in accordance with the diagnostic capacity of procalcitonin reported in the cirrhotic population [AUC: 0.68-0.89] [21].

In **Yang *et al.*** [22]'s meta-analysis, procalcitonin was found to have a sensitivity of 0.82 and specificity of 0.86 with AUROC of 0.92 for diagnosis of SBP**,** which agree with our findings.

El Hasafy *et al.* [23] data showed a significantly raised PCT in cirrhotic patients with infected ascites than those with sterile ascites, which is in line with our study results. Despite that, other studies reported that the serum PCT was not a beneficial marker for predicting SBP [30% sensitivity] [24]. The small sample size [n = 32] of their study could explain this contradictory result.

As regards the CRP, our study showed that the CRP increased significantly in Group B in comparison with group A and C. This is in line with the results of two studies [13, 23] who showed that CRP levels in the patients with SBP were more elevated than those in sterile ascites.

In our study CRP at the optimal Cutoff was 23.5 mg/L and 88% sensitivity and 75.6% specificity, AUC [0.85], which agree with the findings of **Yuan *et al.*** [25], but disagree with **Papp *et al.*** [26] who reported a cutoff of 10 mg/L for CRP [AUC: 0.93] in a large study of patients with cirrhosis associated bacterial infections.

The diagnosis of SBP has been examined in several studies that investigated the combination of procalcitonin and CRP. These studies have been comprehensively summarized in the meta-analysis conducted by **Lin *et al.*** [27]. The pooled sensitivity and specificity for procalcitonin were reported to be 79% and 89% respectively. The combined sensitivity and specificity for CRP was 77% and 85%, respectively, which aligns with our findings.

Although CRP was more sensitive than PCT in this study [88%% vs. 86%], the sensitivity and specificity of serum PCT at a cutoff value of 0.48 for SBP diagnosis were relatively higher than those of ESR at a cutoff value of 30.5 mm/h [86% vs. 84%]. PCT appears to be more effective than other pro-inflammatory indicators like ESR and CRP for diagnosing septic illnesses because it rises more quickly and with more specificity than other markers. This is in line with previous studies [13, 19], and disagrees with the results of **Lesińska *et al.*** [24]**,** who reported that serum PCT could not distinguish patients with and without SBP. The small sample size [n = 32] of their study could explain this difference.

As regards the ESR, we found that it was significantly higher in group B than in groups A, and C [P value < 0.001]. This agree previous studies [13, 15, 16]. The ESR elevation in our patients could be explained by SBP results in the activation of cytokine synthesis and innate immunity in response to circulating bacterial endotoxins as reported by **Deschênes and Villeneuve** [17].

The current work reports the value of the PEC index in the diagnosis of spontaneous Bacterial Peritonitis, which is made by measurement of procalcitonin, ESR, and CRP. PEC index = [PCT × [ESR + CRP]].

As regards the PEC index, in our study the PEC index is significantly increase in group B [96± 32] in comparison with group A [32.9± 18] and group C [2.4± 1.6] with [P value < 0.001], and the sensitivity and specificity in diagnosis of SBP was [92% and 97.8%] at cut-off value 25. This was in agreement with **Elsadek *et al.*** [13], who found a cutoff value of 20, the sensitivity and specificity of serum PEC index for SBP diagnosis [98.33% and 96.67%, respectively]

Our study found that the peritoneal fluid epithelial cell [PEC] was more effective than the serum markers procalcitonin [PCT], erythrocyte sedimentation rate [ESR], and C-reactive protein [CRP] in diagnosing spontaneous bacterial peritonitis [SBP]. The PEC index is superior to any of its individual components for diagnosing SBP due to its ability to mathematically compensate for the varying degrees of sensitivity and specificity of each marker. This combination helps to minimize false positive and false negative results.

Conclusion: PEC index is effective in diagnosis of spontaneous Bacterial Peritonitis, which made by measurement of procalcitonin, ESR and CRP, which we called the PEC index as a predictive value in Decompensated liver disease ascites.

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REFERENCES

1. Liu YB, Chen MK. Epidemiology of liver cirrhosis and associated complications: Current knowledge and future directions. World J Gastroenterol. 2022 Nov 7;28[41]: 5910-5930. doi: 10.3748/wjg.v28.i41.5910.
2. Bonnel AR, Bunchorntavakul C, Reddy KR. Immune dysfunction and infections in patients with cirrhosis. Clin Gastroenterol Hepatol. 2011 Sep;9[9]:727-38. doi: 10.1016/j.cgh. 2011.02.031.
3. Garcia-Tsao G. Current management of the complications of cirrhosis and portal hypertension: variceal hemorrhage, ascites, and spontaneous bacterial peritonitis. Gastroenterology. 2001 Feb;120[3]:726-48. doi: 10. 1053/gast.2001.22580.
4. Ekpanyapong S, Reddy KR. Infections in Cirrhosis. Curr Treat Options Gastroenterol. 2019;17[2]:254-270. doi: 10.1007/s11938-019-00229-2.
5. European Association for the Study of the Liver. Electronic address: easloffice@easloffice.eu; European Association for the Study of the Liver. EASL Clinical Practice Guidelines: Management of hepatocellular carcinoma. J Hepatol. 2018 Jul;69[1]:182-236. doi: 10. 1016/j.jhep.2018.03.019.
6. Enomoto H, Inoue S, Matsuhisa A, Nishiguchi S. Diagnosis of spontaneous bacterial peritonitis and an in situ hybridization approach to detect an "unidentified" pathogen. Int J Hepatol. 2014;2014:634617. doi: 10.1155/2014/634617.
7. Fernandes SR, Santos P, Fatela N, Baldaia C, Tato Marinho R, Proença H, Ramalho F, Velosa J. Ascitic Calprotectin is a Novel and Accurate Marker for Spontaneous Bacterial Peritonitis. J Clin Lab Anal. 2016 Nov; 30[6]:1139-1145. doi: 10.1002/jcla.21994.
8. Maruna P, Nedelníková K, Gürlich R. Physiology and genetics of procalcitonin. Physiol Res. 2000;49 Suppl 1:S57-61.
9. Yang SK, Xiao L, Zhang H, Xu XX, Song PA, Liu FY, Sun L. Significance of serum procalcitonin as biomarker for detection of bacterial peritonitis: a systematic review and meta-analysis. BMC Infect Dis. 2014 Aug 22;14:452. doi: 10.1186/1471-2334-14-452.
10. Elbahrawy A, Ibrahim MK, Eliwa A, Alboraie M, Madian A, Aly HH. Current situation of viral hepatitis in Egypt. Microbiol Immunol. 2021 Sep;65[9]:352-372. doi: 10. 1111/1348-0421.12916.
11. Popoiag RE, Suceveanu AI, Suceveanu AP, Micu SI, Voinea F, Mazilu L, *et al*. Predictors of spontaneous bacterial peritonitis in Romanian adults with liver cirrhosis: Focus on the neutrophil-to-lymphocyte ratio. Exp Ther Med. 2021 Sep;22[3]:983. doi: 10.3892/ etm.2021.10415.
12. Guy J, Peters MG. Liver disease in women: the influence of gender on epidemiology, natural history, and patient outcomes. Gastroenterol Hepatol [N Y]. 2013 Oct;9[10]: 633-9.

13. Elsadek HM, Elhawari SA, Mokhtar A. A novel serum index for accurate diagnosis of spontaneous bacterial peritonitis in cirrhotic patients without other infections. Egypt Liver J. 2020 Dec; 10[2020]:1-8. doi: 10.1186/ s43066-020-0021-8.

14. Narula N, Tsoi K, Marshall JK. Should albumin be used in all patients with spontaneous bacterial peritonitis? Canad J Gastroenterol. 2011;25[7]:373–6. Doi: 10. 1155/2011/586702.

15. Wu H, Chen L, Sun Y, Meng C, Hou W. The role of serum procalcitonin and C-reactive protein levels in predicting spontaneous bacterial peritonitis in patients with advanced liver cirrhosis. Pak J Med Sci. 2016 Nov-Dec;32[6]:1484-1488. doi: 10. 12669/pjms.326.10995.

16. Viallon A, Zeni F, Pouzet V, Lambert C, Quenet S, Aubert G, Guyomarch S, Tardy B, Bertrand JC. Serum and ascitic procalcitonin levels in cirrhotic patients with spontaneous bacterial peritonitis: diagnostic value and relationship to pro-inflammatory cytokines. Intensive Care Med. 2000 Aug;26[8]:1082-8. doi: 10.1007/s001340051321.

17. Deschênes M, Villeneuve JP. Risk factors for the development of bacterial infections in hospitalized patients with cirrhosis. Am J Gastroenterol. 1999 Aug;94[8]:2193-7. doi: 10.1111/j.1572-0241.1999.01293.x.

18. Wacker C, Prkno A, Brunkhorst FM, Schlattmann P. Procalcitonin as a diagnostic marker for sepsis: a systematic review and meta-analysis. Lancet Infect Dis. 2013;13[5]: 426-35. doi: 10.1016/S1473-3099[12]70323-7.

19. Luzzani A, Polati E, Dorizzi R, Rungatscher A, Pavan R, Merlini A. Comparison of procalcitonin and C-reactive protein as markers of sepsis. Crit Care Med. 2003 Jun;31[6]:1737-41. doi: 10.1097/01.CCM. 0000063440.19188.ED.

20. Rule JA, Hynan LS, Attar N, Sanders C, Korzun WJ, Lee WM; Acute Liver Failure Study Group. Procalcitonin Identifies Cell Injury, Not Bacterial Infection, in Acute Liver Failure. PLoS One. 2015 Sep;10[9]:e0138566. doi: 10.1371/journal.pone.0138566.

21. Fernández J, Gustot T. Management of bacterial infections in cirrhosis. J Hepatol. 2012;56 Suppl 1:S1-12. doi: 10.1016/S0168-8278[12]60002-6.

22. Yang Y, Li L, Qu C, Zeng B, Liang S, Luo Z, Wang X, Zhong C. Diagnostic Accuracy of Serum Procalcitonin for Spontaneous Bacterial Peritonitis Due to End-stage Liver Disease: A Meta-analysis. Medicine [Baltimore]. 2015 Dec; 94[49]:e2077. doi: 10.1097/MD.0000000000-002077.

23. El Hasafy MY, El Kaffash DM, Shater MS, Attia NA. Diagnostic role of procalcitonin serum level in hepatitis C virus-infected patients with spontaneous bacterial peritonitis. Menouf Med J. 2022 Apr 1;35[2]:548-52. doi: 10.4103/mmj.mmj_430_20.

24. Lesińska M, Hartleb M, Gutkowski K, Nowakowska-Duława E. Procalcitonin and macrophage inflammatory protein-1 beta [MIP-1β] in serum and peritoneal fluid of patients with decompensated cirrhosis and spontaneous bacterial peritonitis. Adv Med Sci. 2014 Mar;59[1]:52-6. doi: 10.1016/j. advms.2013.07.006.

25. Yuan LY, Ke ZQ, Wang M, Li Y. Procalcitonin and C-reactive protein in the diagnosis and prediction of spontaneous bacterial peritonitis associated with chronic severe hepatitis B. Ann Lab Med. 2013 Nov; 33[6]:449-54. doi: 10.3343/alm.2013.33.6.449.

26. Papp M, Vitalis Z, Altorjay I, Tornai I, Udvardy M, Harsfalvi J, Vida A, Kappelmayer J, Lakatos PL, Antal-Szalmas P. Acute phase proteins in the diagnosis and prediction of cirrhosis associated bacterial infections. Liver Int. 2012 Apr;32[4]:603-11. doi: 10.1111/j. 1478-3231.2011.02689.x.

27. Lin KH, Wang FL, Wu MS, Jiang BY, Kao WL, Chao HY, Wu JY, Lee CC. Serum procalcitonin and C-reactive protein levels as markers of bacterial infection in patients with liver cirrhosis: a systematic review and meta-analysis. Diagn Microbiol Infect Dis. 2014 Sep;80[1]:72-8. doi: 10.1016/j.diagmicrobio. 2014.03.029.

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