

VALUE OF MACROPHAGE INFLAMMATORY PROTEIN-1 BETA IN DIAGNOSIS OF SPONTANEOUS BACTERIAL PERITONITIS IN CIRRHOTIC PATIENTS WITH ASCITES

MOSTAFA SOLIMAN EL-KADY*, MOHAMMED EL-SAYED EL-SHEWI*, MAHA ABD EL-MAWLA MOHAMMED*, SHUZAN ALI MOHAMMED**

**Hepatology, Gastroenterology and Infectious Diseases Department, **Medical Biochemistry Department, Faculty of Medicine - Benha University - Egypt*

Abstract:

Background: Spontaneous bacterial peritonitis (SBP) is a popular unique feature of cirrhosis, associated with systemic and local immune impairment with exaggerated stimulation of pro-inflammatory cytokines. Macrophage inflammatory protein-1beta (MIP-1 β) is produced by macrophages and interacts with chemokine C-C receptor 5 (CCR5). It is recognized for its chemotactic and pro-inflammatory influences.

Objective: to assess the value of MIP-1 β measurement in serum and ascitic fluid for SBP diagnosis in liver cirrhosis.

Subjects and methods: This study included 44 patients divided into 2 groups (22 each); SBP and non-SBP. They were subjected to full history taking, clinical examination, abdomino-pelvic ultrasonography and laboratory investigations including complete blood count, liver biochemical tests, renal function tests and viral markers. Abdominal paracentesis and ascitic fluid analysis were performed. MIP-1 β in serum and ascitic fluid was quantified by ELISA.

Results: This study showed significant increased number of patients suffering abdominal pain ($p=0.004$) and jaundice ($p=0.001$), or those who were Child-Pugh score class C and significant increased mean levels of TLC, serum bilirubin, creatinine and ascitic fluid TLC and PMN in SBP versus non-SBP. MIP-1 β was significantly elevated in SBP versus non-SBP both in serum and in ascitic fluid. The optimal cut-off point for MIP-1 β was 15.21 pg / ml in serum and 31.66 pg / ml in ascitic fluid. MIP-1 β in serum had 81.8% sensitivity and 72.7% specificity. MIP-1 β in ascitic fluid had 86.4% sensitivity and 81.8% specificity. Serum and ascitic fluid MIP-1 β were significantly positively correlated with each other both in SBP and non-SBP. In SBP, serum and ascitic MIP-1 β were positively correlated with serum creatinine and the ascitic MIP-1 β was positively correlated with ascitic TLC and ascitic PMN.

Conclusion: MIP-1 β could help diagnose SBP in cirrhotic patients. It might be of special importance in SBP patients with culture negative ascitic fluid.

Key Words: spontaneous bacterial peritonitis, MIP-1 β

Introduction:

Cirrhosis is a widespread process that may develop over weeks to years affecting the normal hepatic structure ⁽¹⁾. Cirrhosis is mostly complicated by ascites in more than 60% of compensated cirrhosis within 10 years of illness ⁽²⁾.

Cirrhosis patients are immune-deficient with exaggerated stimulation of pro-inflammatory cytokines, predisposing patients to infections, a condition known as cirrhosis-associated immune dysfunction syndrome. Almost all components of systemic immune response show significant dysregulation in cirrhosis, including reduced phagocyte activity, serum albumin, complement, protein C activities, and impaired opsonic activity both in serum and ascitic fluid ⁽³⁾.

Spontaneous bacterial peritonitis (SBP) is popular unique feature of cirrhosis. SBP is mainly due to bacterial translocation ^(4,5) induced by portal hypertension ⁽⁶⁾, systemic, and local immune impairment, especially the reduced opsonic performance in ascitic fluid ⁽⁷⁾. SBP traditional symptoms are fever, abdominal pain, and worsening of pre-existing ascites, despite absence of these symptoms in up to one-third of patients ⁽⁸⁾. SBP prevalence, in cirrhotic cases with ascites who are admitted to hospital, ranges from 10-30%; about 50% occur at the time of hospitalization and the other half takes place during hospitalization ⁽⁹⁾. The overall one-year mortality rate after the first episode ranges from 30% to 93% ⁽¹⁰⁾.

Macrophage Inflammatory Protein (MIP) is one of chemotactic cytokine family called chemokines. Two types do occur in the human body, MIP-1 α and MIP-1 β . Both are encoded by genes on human chromosome 17 ⁽¹¹⁾. MIP-1 β is known as chemokine C-C ligand 4 (CCL4)

that specifically bind chemokine C-C receptor 5 (CCR5) ⁽¹²⁾. MIP-1 β is produced by macrophages and it is critical for monocytes/macrophages (MF) recruitment⁽¹³⁾. It is best recognized for its chemotactic and pro-inflammatory influences and homeostasis promotion. MIP-1 β is expressed on different leukocyte types as macrophages, neutrophils, dendritic cells, and lymphocytes. It also activates human granulocytes that may cause acute neutrophilic inflammation. In addition, it attracts many immune cells as T lymphocytes, dendritic cells and natural killer cells ⁽¹⁴⁾. MIP-1 β enhances the synthesis and release of different pro-inflammatory cytokines as interleukin 1, IL6 and tumor necrosis factor α from fibroblasts and macrophages ⁽¹¹⁾. MIP-1 β is most effective at magnifying CD8 (+) T-cell adhesion to the cell adhesion molecules ⁽¹⁵⁾. A chemokines has a considerable shortened half-life than does a classical inflammatory biomarker that may trigger their appropriateness for diagnosis and monitoring ⁽¹⁶⁾. The MIP-1 β diagnostic importance for bacterial infection is fairly understood ⁽¹⁵⁾. So the current study aimed to assess the value of MIP-1 β measurement in serum and ascitic fluid in diagnosis of SBP in patients with liver cirrhosis.

Subjects and methods:

This cross-sectional case-control study was performed in cooperation between the Hepatology, Gastroenterology and Infectious Diseases Department, and the Medical Biochemistry Department, Faculty of Medicine, Benha University. Patients were among those admitted in the Department of Hepatology, Gastroenterology and Infectious Diseases, Benha University Hospitals in the period from May 2016 to May 2017.

Inclusion criteria

Patients with liver cirrhosis and ascites based on

clinical, biochemical and ultra-sonographic findings with no antibiotics received for 2 weeks prior to hospital admission.

Exclusion criteria

Patients with secondary bacterial peritonitis, current use of antibiotics, advanced hepatic encephalopathy (grades 3 and 4), current gastrointestinal bleeding, surgery within the last 6 months, comorbidities (e.g. heart failure, chronic renal disease, COPD), malignancy and bacterial infectious diseases other than SBP were excluded from the study.

Secondary bacterial peritonitis was defined as isolation of >1 organism from ascites or presence of at least 2 of the following criteria in ascitic fluid: glucose level <50 mg/ml, protein concentration >1 g/dl, and serum lactic dehydrogenase level above the upper limit normal)⁽¹⁷⁾.

Included patients were divided into 2 groups (22 patients each); group I: cirrhotic patients without SBP and group II: cirrhotic patients with SBP. SBP is defined as an ascitic fluid infection with a polymorphonuclear leucocyte (PMN) count $\geq 250/\text{mm}^3$ with or without positive ascitic culture and with no proved intra-abdominal surgically-curable cause⁽¹⁸⁾.

All patients were subjected to full history taking, clinical examination, abdominopelvic ultrasonography to exclude patients with ascites due to any cardiac cause and those with Budd-Chiari syndrome. Laboratory investigations including liver biochemical tests, kidney function tests, viral markers, serum and ascitic MIP-1 β were performed. Abdominal paracentesis under complete aseptic conditions was performed followed by ascitic fluid analysis for total leucocyte count (TLC) and PMN and total protein and albumin.

Sampling:

1. A blood sample:

A venous blood sample (7.5 ml) was taken from each patient using sterile syringes under aseptic conditions. The collected samples were sent immediately to the laboratory of Benha University Hospital and divided as follow; part 1: 1 ml on ethylene diamine tetra-acetic acid for complete blood count (CBC), part 2: 2 ml were put on 0.5 ml tri-sodium citrate (3.8%) to determine the erythrocyte sedimentation rate (ESR) (mm/hour). Part 3: 0.9 ml was put on 0.1 ml tri-sodium citrate solution (3.2%) in a ratio of 9:1 for determination of prothrombin time (seconds), concentration and International normalized ratio (INR) and part 4: ~ 3.5 ml were left to clot for half an hour and then centrifuged for 15 minutes at 1000 xg to separate serum. Hyperlipidemic and hemolyzed samples were excluded. Sera were used for estimation of liver function tests, kidney functions tests, viral markers and serum MIP-1 β .

2. Ascitic fluid sample:

Ascitic fluid sample (3 ml) was taken on EDTA via abdominal paracentesis under complete aseptic conditions. It was used for physical, cytological (TLC, PMN) and biochemical (total protein, albumin and ascitic MIP-1 β) findings.

Biochemical investigations

CBC was done automatically using hematology autoanalyzer Sysmex XS-1000i (Sysmex, Japan). Ascitic fluid analysis for TLC and PMN was performed using hemocytometer and microscopic examination. Biochemical assays for serum creatinine⁽¹⁹⁾, liver function tests including serum alanine amino transferase (ALT)⁽²⁰⁾, aspartate amino transferase (AST)⁽²⁰⁾, serum and ascitic total protein⁽²¹⁾, serum and ascitic albumin⁽²²⁾,

serum total bilirubin ⁽²³⁾ were performed **using Microtech spectrophotometer (Vital Scientific, Netherlands)**. Prothrombin time was determined using Behring Fibrin timer II (Behring, Germany) ⁽²⁴⁾. In addition, hepatitis markers were estimated by enzyme immunoassay for hepatitis B surface antigen ⁽²⁵⁾, HCV antibodies ⁽²⁶⁾. Serum ascites albumin gradient (SAAG) was estimated by the equation: SAAG = serum albumin concentration - ascitic fluid albumin concentration ⁽²⁷⁾.

Estimation of serum and ascitic MIP-1 β by enzyme-linked immune-sorbent assay (ELISA):

MIP-1 β in Serum and ascitic fluid was determined by an invitro double-antibody sandwich ELISA using a commercial kit (Shanghai Sunred Biological Technology, China). The assay ranged from 0.5 to 150 pg/ml and its was sensitive to a level of 0.432 pg/ml. Biotin-labelled antibody specific for MIP-1 β was used. After washing, streptavidin-conjugated Horseradish Peroxidase (HRP) was added to wells. After washing to remove any unbound avidin-enzyme reagent, a substrate solution was added and the color developed in proportion to MIP-1 β concentration in samples. The color development was stopped and the intensity of the color was measured at 450nm by TECAN Infinite F50 ELISA Reader (Singapore). A standard curve was created (figure 1) by Magellan Tracker software (Tecan Trading AG, Switzerland) to be used for determination of MIP-1 β concentration in samples.

Statistical analysis

Data were fed to the computer using SPSS software version 20. Quantitative data were described as mean and standard deviation (SD) for normally distributed data. Mann Whitney U test was used to compare between two groups of independent non-normally distributed data. The Pearson

correlation coefficient (r) was calculated to assess the relationship between various inflammatory markers. Receiver Operating Characteristics (ROC) curve was obtained to calculate the optimized cutoff value for MIP-1 β to predict SBP in cirrhosis. All tests were two sided with $p < 0.05$ was statistically significant.

Results:

There were significant increased number of patients suffering abdominal pain ($p=0.004$) and jaundice ($p=0.001$), or those who were Child-Pugh score class C ($p=0.003$) in the SBP versus the non-SBP patients (table 1).

Significant increased TLC ($p=0.00$), serum bilirubin ($p=0.01$), creatinine ($p=0.02$), TLC and PMN of ascitic fluid ($p=0.00$ for both) in SBP versus non-SBP cirrhosis were found (table 2).

There were also significantly elevated MIP-1 β in the SBP group versus the non-SBP group both in serum (figure 2-A) and in ascitic fluid (figure 2-B)

Table (3) and figure (3) showed the ROC analysis for serum and ascitic MIP-1 β levels. The optimal cut-off point was 15.21 pg / ml in serum and 31.66 pg / ml in ascitic fluid. Serum MIP-1 β had 81.8% sensitivity, 72.7% specificity, 75% PPV, 80% NPV, 77.3% accuracy and 0.81 for area under the curve (AUC). Ascitic fluid MIP-1 β had 86.4% sensitivity, 81.8% specificity, 82.6% PPV, 85.7% NPV, 84.1% accuracy and 0.89 for AUC.

Serum and ascitic fluid MIP-1 β levels were significantly positively correlated with each other both in non-SBP ($r=0.634$, $p=0.002$) (figure 4-A) and SBP ($r=0.660$, $p=0.001$) (figure 4-B).

In SBP, the serum and ascitic MIP-1 β were positively correlated with serum creatinine (figure 4-C; $r=0.752$, $p=0.00$ and, figure 4-D; $r=0.470$;

p=0.027, respectively) and the ascitic MIP-1 β was p=0.034) (figure 4-E) and with ascitic PMN positively correlated with ascitic TLC (r=0.454, (r=0.520, p=0.013) (figure 4-F).

Table (1): Demographic, clinical and ultrasonographic findings of the studied groups .

Variables	Group I (n.=22)	Group II (n.=22)	Test	p
	n.(%) or mean \pm SD			
Age (years)	58.82 \pm 9.11	56.09 \pm 9.81	t=0.34	0.561
Gender (σ / ρ)	14(63.6) / 8(36.4)	14(63.6) / 8(36.4)	X ² =0.0	1
Vomiting (N/Y)	17(77.3) / 5(22.7)	18(81.8) / 4 (18.2)	X ² =0.14	0.708
Diarrhea (N/Y)	22(100) / 0(0)	21(95.5) / 1(4.5)	X ² =1.02	0.312
Abdominal pain (N/Y)	21(95.5) / 1 (4.5)	13(59.1) / 9 (40.9)	X ² =8.28	0.004**
Hematemesis (N/Y)	15(68.2) / 7(31.8)	17(77.3) / 5 (22.7)	X ² =0.46	0.498
Sclerotherapy (N/Y)	19(86.4) / 3 (13.6)	21(95.5) / 1 (4.5)	X ² =1.1	0.294
Band ligation (N/Y)	9(40.9) / 13 (59.1)	12(54.5) / 10 (45.5)	X ² =0.82	0.365
History of tapping (N/Y)	13(59.1) / 9 (40.9)	15(68.2) / 7 (31.8)	X ² =0.39	0.531
Fever (N/Y)	21(95.5) / 1(4.5)	19(86.4) / 3(13.6)	X ² =1.1	0.294
Jaundice (N/Y)	11(50) / 11(50)	3(13.6) / 19(86.4)	X ² =6.71	0.001**
Pallor (N/Y)	21(95.5) / 1 (4.5)	22(100) / 0(0)	X ² =1.02	0.312
LL edema (N/Y)	2(9.1) / 20 (90.9)	3(13.6) / 19(86.4)	X ² =0.23	0.635
Palpable spleen (P/ PNP/SR)	1(4.5) / 20(90.9) / 1(4.5)	3(13.6) / 19(86.4) / 0(0)	X ² =2.023	0.363
Child-Pugh score (B / C)	11(50) /11(50)	2(9.1) / 20(90.9)	X ² =8.84	0.003**
PV diameter (US)	14.214 \pm 2.151	14.409 \pm 3.187	t=1.55	0.221

N: no, Y: yes, PNP: not palpable, P: palpable, SR: surgically removed, PV: portal vein, US: ultrasonography, "t": student t test, X²: Chi square test, **: high significant

Table (2): Laboratory investigations of the studied groups.

Variables	Group I (n.=22)	Group II (n.=22)	“t” test	p
Hemoglobin (mg/dl)	8.87 ± 1.83	10.13 ± 2.46	2.54	0.11
TLC (×10 ⁹ c /L)	6.47 ± 2.04	11.08 ± 5.45	34.71	0.00**
Platelets (×10 ⁹ c /L)	106.63 ± 45.36	97.22 ± 42.59	0.58	0.45
Serum AST (IU/L)	67.59 ± 28.55	79.55 ± 39.54	1.04	0.32
Serum ALT (IU/L)	65.81 ± 31.15	65.5 ± 36.13	0.21	0.65
Serum Albumin (g/dl)	2.85±0.33	2.67 ± 0.36	0.004	0.94
Serum Bilirubin (g/dl)	3.27 ± 3.46	6.70 ± 4.91	6.65	0.01*
Prothrombin time (seconds)	15.54 ± 2.4	16.65 ± 3.28	1.37	0.24
International normalized ratio	1.4 ± 0.35	1.55 ± 0.46	0.64	0.43
Serum creatinine (mg/dl)	1.42 ± 0.54	1.8 ± 1.28	5.41	0.02*
ESR (mm/hr)	52.72 ± 24.13	75.45 ± 29.99	1.41	0.24
TLC in ascitic fluid (×10 ⁹ c /L)	191.13 ± 116.75	1791.18 ± 1066.14	44.96	0.00**
PMN in ascitic fluid (×10 ⁹ c /L)	45.50 ± 20.61	1110.77 ± 490.41	41.55	0.00**
Ascitic albumin (g/dl)	0.7 ± 0.31	0.78 ± 0.37	1.24	0.27
Ascitic protein (g/dl)	1.59 ± 0.61	1.94 ± 0.78	2.65	0.11
SAAG (g/dl)	2.19 ± 0.48	1.93 ± 0.37	1.93	0.17

TLC: total leucocytic count, AST: aspartate transaminase, ALT: alanine transaminase, ESR: Erythrocyte sedimentation rate, PMN: polymorphonuclear leukocytes, SAAG: serum-ascites albumin gradient, “t”: student “t” test, *: significant, **: high significant

Table (3): Diagnostic performance of macrophage inflammatory protein-1 beta (MIP-1 β) for diagnosis of SBP in cirrhotic patients.

MIP-1 β	Cutoff level	Sens	Spec	PPV	NPV	Acc.	AUC	95% CI	p
Serum	15.21 pg/ml	81.8%	72.7%	75%	80%	77.3%	0.81	0.69-0.94	0.00**
Ascitic	31.66 pg/ml	86.4%	81.8%	82.6%	85.7%	84.1%	0.89	0.77-1.00	0.00**

Sens: Sensitivity, Spec: Specificity, PPV: positive predictive value, NPV: negative predictive value, Acc.: accuracy, AUC: area under the curve, CI: confidence interval, **: high significant

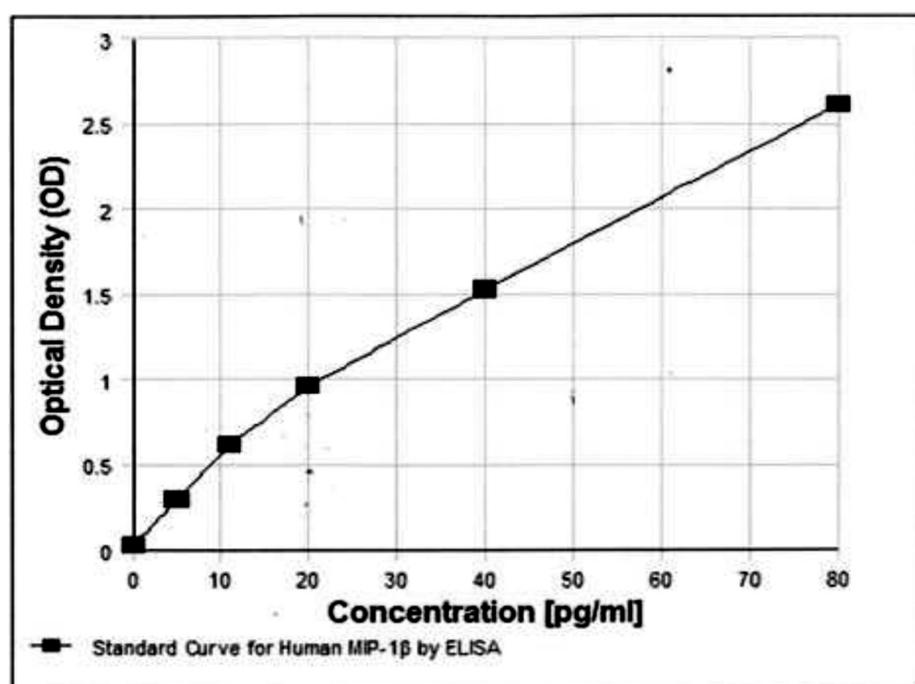


Figure (1): The standard curve for macrophage inflammatory protein-1 beta measured by ELISA

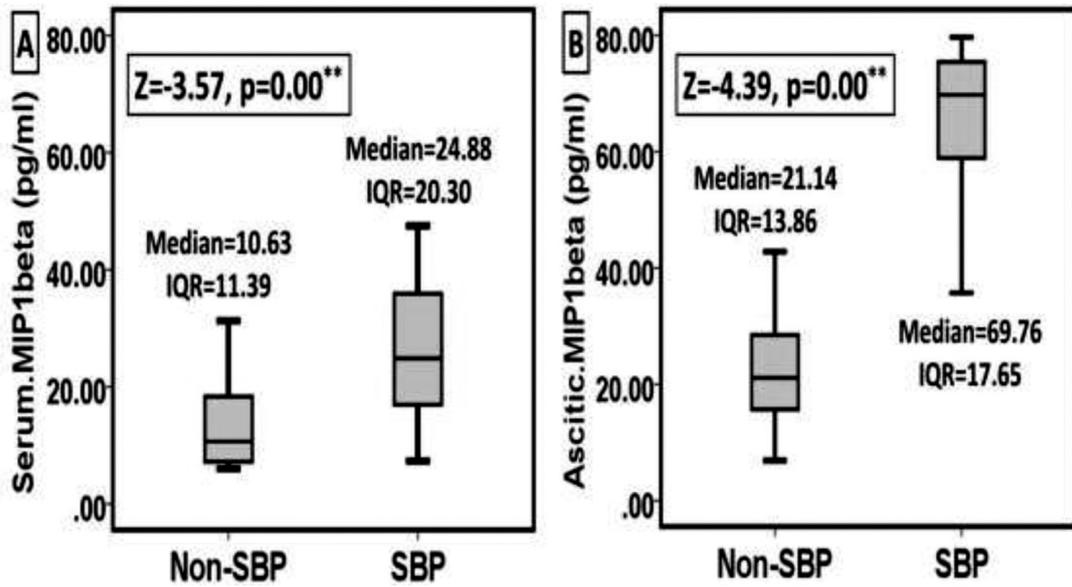


Figure (2): MIP-1 β in the study groups; A: serum, B: ascitic fluid

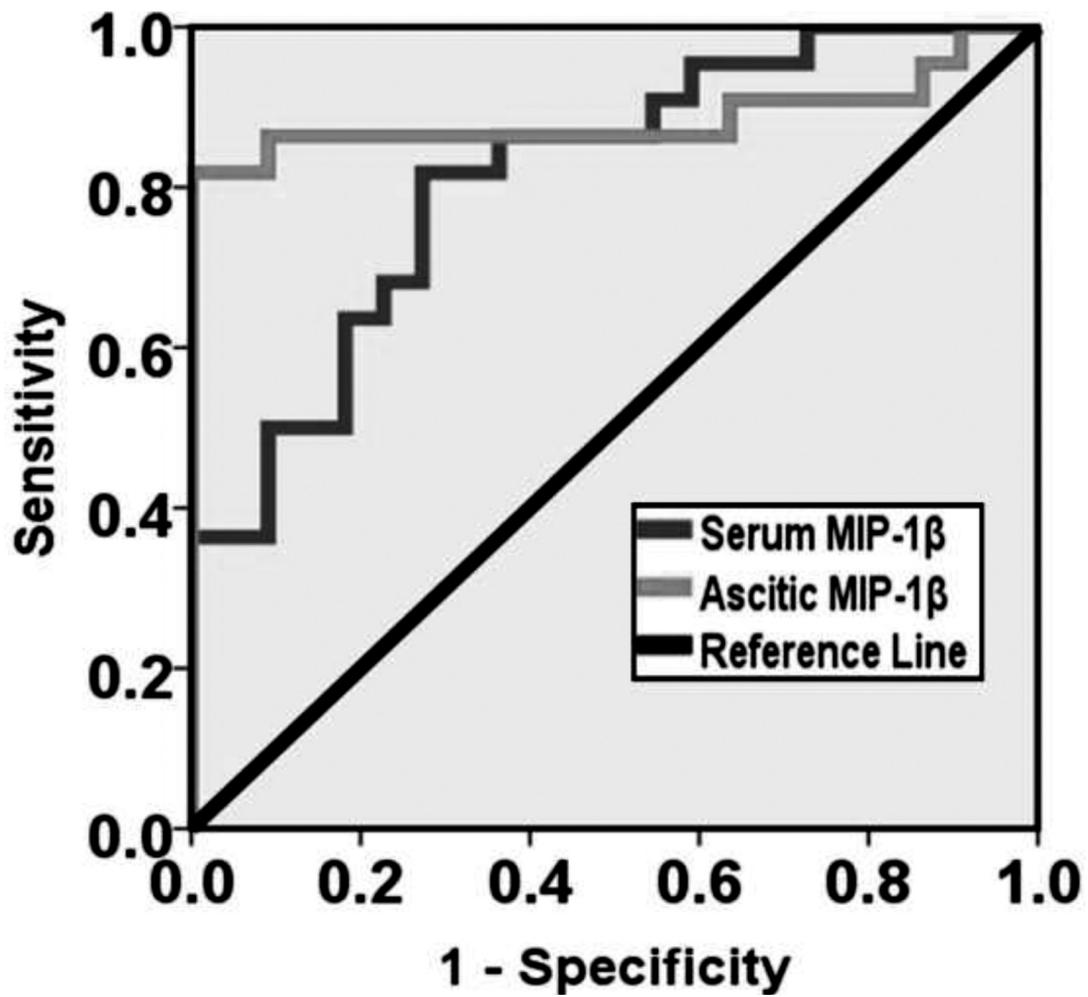


Figure (3): ROC curve of MIP-1 β for diagnosis of SBP in Cirrhosis

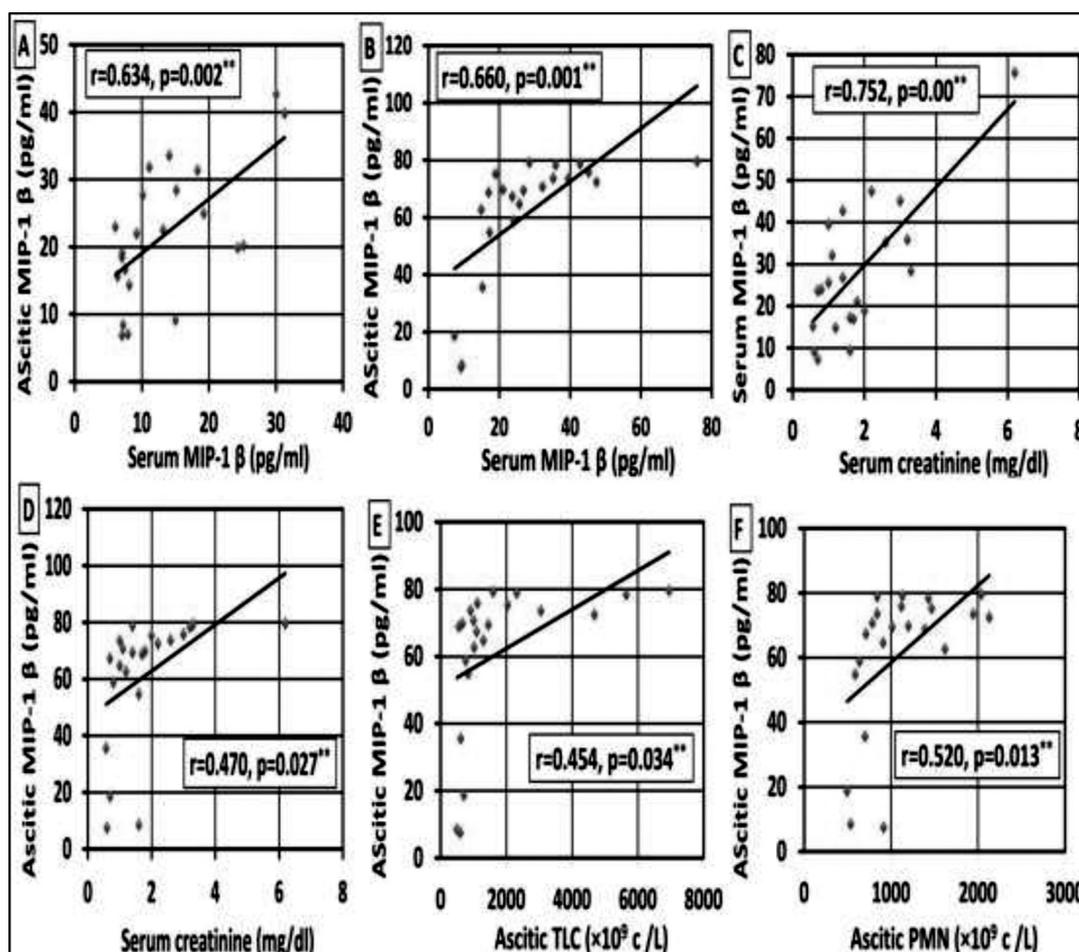


Figure (4): Correlation between MIP-1 β and some biochemical parameters; non-SBP (A: serum and ascitic MIP-1 β), SBP (B: serum and ascitic MIP-1 β , C: serum MIP-1 β and serum creatinine, D: ascitic MIP-1 β and serum creatinine, E: ascitic MIP-1 β and ascitic TLC, F: ascitic MIP-1 β and ascitic PMN)

Discussion:

Liver cirrhosis patients are at highly exposed to infection mainly bacteria because of subnormal phagocytic cell or opsonic activity in the liver reticuloendothelial system which enhance the bacterial entry into the general circulation via port-caval shunts⁽²⁸⁾. SBP is the most common and dangerous bacterial infection in hepatic cirrhosis due to associated immunocompromised state⁽²⁹⁾. Moreover, a delay in diagnosis of SBP is often fatal. Therefore, early and accurate diagnosis is particularly important⁽³⁰⁾.

In the present study, the clinical characteristics of the studied patients showed that abdominal pain was significantly higher in SBP patients than the non-SBP. In agreement, Aminiahidashti et al.⁽³¹⁾ reported that the majority of SBP cases had abdominal pain with 93.75% sensitivity for diagnosing SBP but with lower specificity (62.50%) and deduced that in cirrhotic cases who did not suffer abdominal pain, the **probability** of SBP is low.

The number of jaundiced patients in SBP was significantly higher than in the non-SBP. In accordance, several studies showed that jaundice

followed by fever were the most common symptoms of peritonitis in 60%-80% of patients ⁽³²⁾.

In this study, Child-Pugh score class C was significantly higher in patients with SBP than the non-SBP. Thiele et al. ⁽³³⁾ stated that the higher numbers of Child-Pugh rankings the higher the SBP risk, this help explain why 70% of SBP patients are seen in patients with Child-Pugh class C cirrhosis ⁽³²⁾ and those patients have poor long-term prognosis with ⁽¹⁰⁾.

As for laboratory investigations, the TLC and bilirubin were significantly higher in SBP than in non-SBP. Leukocytosis in peripheral blood could help predict the occurrence of SBP in asymptomatic ascites ⁽³⁴⁾. As for bilirubin, a higher serum level might be indirectly linked to a greater risk of having SBP as it usually **synchronizes** with an advanced stage of liver disease ⁽³⁵⁾.

Serum creatinine was significantly elevated in SBP versus the non-SBP. Our result could be explained by that bacterial infection in cirrhosis might be associated with organ failure (especially renal failure) ⁽³⁶⁾. Acute renal injury after infection occurs in 27-34% of advanced cirrhosis ⁽³⁾, and is a strong predictor of death (40%-50% mortality) ⁽³⁶⁾.

The current study showed that the ascitic fluid TLC and PMN were significantly higher in patients with SBP versus the non-SBP. The same results were found by some researchers ^(37, 38). In addition, PMN count starting at 250 or 500 cells / ml was considered valid marker for SBP ⁽³⁹⁾.

The present study showed significantly elevated serum and ascitic levels of MIP-1 β in SBP versus non-SBP. The optimal MIP-1 β cutoff value was 31.66 pg/ml in ascitic fluid and 15.21 pg/ml in serum with higher sensitivity, specificity and accuracy of ascitic fluid MIP-1 β (86.4%, 81.8% and 84.1%)

than serum MIP-1 β (81.8%, 72.7% and 77.3%), respectively. As an explanation, bacterial infection in cirrhosis is linked to dysregulated cytokine responsiveness that converts useful responses against infection into exaggerated, deleterious inflammation ⁽³⁾. In agreement, Khorshed et al. ⁽¹⁶⁾ found that significant elevated MIP-1 β in ascitic fluid ($p < 0.001$) but the rise was insignificant in serum ($p=0.054$) of SBP versus non-SBP. ROC curve analysis detected that the optimal cut-off level was; 121.9 pg/ml in ascites and 85.2 pg/ml in serum. The ascitic fluid MIP-1 β sensitivity and specificity were 76.1% and 100%, versus 76.1% and 100% for serum MIP-1 β , respectively. They reported that MIP-1 β estimation in both serum and ascitic fluid augments the sensitivity and specificity for SBP diagnosis up to 100%. Moreover, Lesińska et al. ⁽¹⁵⁾ found significant elevated MIP-1 β in ascitic fluid in SBP versus non-SBP. They reported that at 69.4 pg/ml cut-off level, the sensitivity was 80% and the specificity was 72.7% with 0.77 for AUC. They also reported that serum MIP-1 β had weak diagnostic role in SBP.

The ascitic MIP-1 β value in SBP was higher than that of serum; a finding which indicates that the peritoneal macrophages and neutrophils are the main origin for MIP-1 β liberation. In these conditions, the serum levels would only reflect the flood of MIP-1 β into systemic circulation; but still a fraction of MIP-1 β is generated by blood monocytes or macrophages ⁽¹⁵⁾. On the other side, our results were mismatched with Holub et al. ⁽⁴⁰⁾ as they reported increased serum MIP-1 β in community-acquired bacterial infection.

As for correlations, the current study agreed with Khorshed et al. ⁽¹⁶⁾ who found significant positive correlations between PMN in ascitic fluid and MIP-1 β in both serum and ascitic fluid ($r=0.997$ and

$r=0.928$, respectively, $p < 0.001$ for both) but they did not investigate the other correlations found in our study. However, our finding disagreed totally with Lesińska et al.⁽¹⁵⁾ who did not find any correlations between serum or ascitic MIP-1 β values and either clinical or routine laboratory parameters.

Conclusion:

MIP-1 β could help diagnose SBP in cirrhotic patients. It might be of special importance in SBP patients with culture negative ascitic fluid.

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تقييم كفاءة بروتين الالتهاب للخلايا الآكلة كبيرة الحجم من نوع بيتا 1 في تشخيص الالتهاب التلقائي للغشاء البريتوني في مرضى تآجر الكبد المصحوب بالاستسقاء

مصطفى سليمان القاضي*، محمد السيد الشيوبي*، مها عبد المولى محمد*،
شوزان على محمد**

* قسم أمراض الكبد والجهاز الهضمي والأمراض المعدية - كلية الطب - جامعة بنها - مصر

** قسم الكيمياء الحيوية الطبية - كلية الطب - جامعة بنها - مصر

يُعد الالتهاب التلقائي للغشاء البريتوني ملامح مميز ومنتشر في مرضى تآجر الكبد، وهو يرتبط باضطراب مناعي عام و موضعي مع المبالغة في خفيز السيتوكينات التي تفرز في بداية الالتهابات. تفرز الخلايا الآكلة كبيرة الحجم بروتينها من النوع بيتا والذي يتفاعل مع مستقبله «سي سي آر ٥». و يُعرف هذا البروتين بقدرته على الجذب و التأثير في بداية الالتهابات

الهدف: هدفت الدراسة إلى تقييم قياس مستوى بروتين الالتهاب للخلايا الآكلة كبيرة الحجم من نوع بيتا ١ في المصل و السائل البريتوني لمرضى الالتهاب التلقائي للغشاء البريتوني في مرضى تآجر الكبد المصحوب بالاستسقاء

الطريقة: ضمت الدراسة أربعة و أربعين مريضاً مصابين بتآجر الكبد- في مجموعتين- احتوت كل مجموعة على اثنين و عشرين مريضاً. المجموعة الأولى بدون الالتهاب التلقائي للغشاء البريتوني المصحوب بالاستسقاء و المجموعة الثانية بها هذا الالتهاب. تعرض المرضى لأخذ التاريخ الطبي الكامل، و الفحص الطبي الشامل. و عمل أشعة تليفزيونية على البطن و الحوض، و عمل التحاليل المعملية بما في ذلك صورة الدم الكاملة، و وظائف الكبد، و وظائف الكلى، و الدلالات الفيروسية. كما تم عمل بزل للسائل البريتوني و تحليله. كما تم قياس مستوى بروتين الالتهاب للخلايا الآكلة كبيرة الحجم من نوع بيتا ١ في كل من المصل و السائل البريتوني بالإليزا

النتائج: أظهرت الدراسة زيادات ذات جدوى إحصائية في أعداد المرضى الذين يعانون من ألم بالبطن أو من الصفراء، أو أولئك الذين كانوا ينتمون إلى الدرجة «سي» في تقسيم المرضى بطريقة « تشايلد بوف» و كذلك زيادات ذات جدوى إحصائية في العدد الكلي لخلايا الدم البيضاء، مستوى الصفراء و مستوى الكرياتينين في المصل. كما زاد العدد الكلي لخلايا الدم البيضاء و خلايا العدلات «نيوتروفيل» في السائل البريتوني في المجموعة الثانية «الالتهاب» مقارنة بالمجموعة الضابطة «بلا التهاب». كما زاد مستوى بروتين الالتهاب للخلايا الآكلة كبيرة الحجم من نوع بيتا-١ في المجموعة الثانية «الالتهاب» مقارنة بالمجموعة الضابطة أيضاً. و كان هذا في المصل و السائل البريتوني. أظهرت الدراسة أن المستوى الأمثل لهذا البروتين لتشخيص المجموعة الثانية «الالتهاب» في المصل ١٥,٢١ بيكوجرام/مل. و في السائل البريتوني ٣١,٦٦ بيكوجرام/مل. أعطى هذا البروتين في المصل حساسية اختبار ٨١,٨٪ و نوعية اختبار ٧٢,٧٪. بينما أعطى السائل البريتوني حساسية اختبار ٨٦,٤٪ و نوعية اختبار ٨١,٤٪. أما فيما يخص العلاقات الإحصائية، فقد تناسب مستويي هذا البروتين في كل من المصل و السائل البريتوني سوياً في كل مجموعة تناسباً طردياً. و في المجموعة الثانية «الالتهاب» تناسب ذات البروتين طردياً في كل من المصل و السائل البريتوني مع مستوى الكرياتينين في المصل. كما تناسب طردياً مستوى البروتين محل الدراسة في السائل البريتوني مع كل من العدد الكلي لخلايا الدم البيضاء و خلايا العدلات «نيوتروفيل» في السائل البريتوني

الخلاصة: خلصت الدراسة إلى أن بروتين الالتهاب للخلايا الآكلة كبيرة الحجم من نوع بيتا-١ قد يسهم في تشخيص الالتهاب التلقائي للغشاء البريتوني في مرضى تآجر الكبد المصحوب بالاستسقاء، خاصة في الحالات التي تعطي فيها مزرعة السائل البريتوني نتائج سلبية