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ORIGINAL ARTICLE

Diagnostic Value of Presepsin among Infected Adults in the Intensive Care Unit

Emad S. M. Bayad1 *, Neveen G. Elantouny2, Rehab H. El-sokkary3, Ghada M. Samir2
1Internal Medicine Department, AlAhrar Teaching Hospital, Zagazig, Egypt
2Internal Medicine Department, Faculty of Medicine, Zagazig University, Zagazig, Egypt
3Medical Microbiology and Immunology Department, Faculty of Medicine, Zagazig University, Zagazig, Egypt

Corresponding author:
Emad S M Bayad

E.mail:
emadsaid964@gmail.com

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ABSTRACT

Background: Infection is a challenge in intensive care units (ICUs), causing high mortality and morbidity. Early diagnosis and prediction is useful in improving management strategies of Adult patients. This study aimed at assessing the accuracy of presepsin for diagnosis of sepsis among adults.

Methods: This study was conducted on ninety-six patents within ICUs of Internal Medicine hospital Zagazig University from December 2019 and May 2020. All patients were assessed physically with quick SOFA score, laboratory investigations including serum presepsin level.

Results: Age of patients ranged from 19 to 60 years with median 56.5 years and male represented 54.2%. Positive blood culture was found in 75% of patients; out of them, 66.7%, 22.2% and 11.1% had infection, sepsis and septic shock respectively. About 57% and 24% had pneumonia and urinary tract infection respectively. Median C reactive protein (CRP) and procalcitonin were 62.5% and 1.7% respectively. Presepsin significantly positively correlated with procalcitonin and C reactive protein($p<0.05$). It significantly negatively correlated with platelet count($p<0.05$).There is significant association between serum presepsin and disease severity, with highest level in septic shock followed by sepsis($p<0.05$).The best cutoff of serum presepsin in diagnosis of infection was ≥1.95 mg/mL with sensitivity 80% and specificity 71% while at cutoff ≥2.4 mg/mL, it can predict sepsis, sensitivity 78.3% and specificity 69%.Presepsincutoff ≥7.9 mg/mL can predict septic shock with sensitivity 90% and specificity 92%.

Conclusions: Presepsin is a promising marker for diagnosis of infection and sepsis with capability for differentiating between sepsis severity groups.

Keywords: severity, accuracy, sepsis, shock, infection

INTRODUCTION

Infection and related sepsis are the leading cause of death in noncardiac ICUs, with mortality rates that reach 60% and account for approximately 40% of total ICU expenditures [1].

Complications can occur if infection is not early diagnosed or properly treated. Untreated and clinically significant bacteremia progresses to systemic inflammatory response syndrome (SIRS), sepsis, septic shock, and multiple organ dysfunction syndrome (MODS). These are serious, life-threatening conditions that need immediate treatment [2].

The definitions of sepsis and septic shock were updated in January 2016 with the goal of recognizing patients at higher risk of adverse outcomes, specifically those requiring ICU admission or with a high risk of death [3].

Numerous biomarkers have been conveyed valuable in sepsis diagnosis, such as procalcitonin and C-reactive protein (CRP). Though, these biomarkers may also be elevated in non-septic circumstances such as trauma, burn, and postoperative situations

and some are sluggish to increase after the occurrence of sepsis. It thus remains essential to find reliable biomarkers to replace or improve those that are currently available [4].

An emerging biomarker of infection is presepsin (PSP), newlylabelled as an early marker ofdiverse infections. Sepsis was the initial clinical setting where PSP was appraised as a biomarker [5].

Circulating presepsin levels can be perceived as a witness of triggered monocyte–macrophage in response to pathogens. Monocytes–macrophages are existing in the circulating compartment and baseline stimulation of these cells physiologically occurs. Therefore, discovery of presepsin is forecast even in healthy non-septic patients. This reason is vital in having a specific and sensitive method to measure PSP, in order to link the increase from the physiological cut-off value to the occurrence of a bacterial infection, and the quantity of this upsurge to the strength of the immune response, thus to infection severity. Presepsin (1) should be measurable in healthy individuals, (2) should upsurge early in the case of a bacterial infection, and (3) its upsurge should be reliant on strength of the innate immune response [6].

Presepsin levels are higher in patients with Gram-negative bacterial infections than patients with Gram-positive infections. In addition, patients with abdominal or urinary tract infections had higher baseline presepsin levels than patients with lung infections [7].

This study was conducted toassess the diagnostic accuracy of presepsin for diagnosis of sepsis in adults.

METHODS

This stud has been conducted according to the code of ethics of the world medical associations (Declaration of Helsniki) for studies involving humans. A written informed consent was gotten from the patients contributing in this research. Approval was obtained from departments of Internal Medicine and Medical microbiology, Zagazig University after approval from IRB.

Study design, study setting and study participants:

This cross-sectional study was performed in The Intensive Care Unit of Internal Medicine Department and medical microbiology and immunology department, Zagazig University Hospitals from the period between December 2019 and May 2020.

Sample size: Using OPEN_EPI software, the sample size was calculated to be 96 patients subdivided into three groups (Group “I” included 65 patients with infections, group “II” included 23 patients with sepsis and group “III” included 8 patient with septic shock) by using the most commonly used scoring system in medical ICU, quick SOFA (qSOFA) score on admission for Severity assessment after suspicion of infection; infection, sepsis and septic shock groupsincluding 65, 23 and 8 patients respectively.

Anysuspected case of infection among adults from 18 to 60 years old of both sexes admitted to the intensive care unit was included according to clinical examination and routine investigations.

Patients received any antibiotic within one week prior to this study, those received corticosteroids and other immunosuppressive drugs, those with renal dysfunction (serum creatinine more than 2.5), hemodialysis patients were excluded

All participants were submitted to the following:

Thorough history taking: Personal, past history of disease, drug, operation and present history of cough,fever,shortness of breath, dysuria,diarrhea,vomiting, abdominal pain,tachycardia,chest pain, seizures, confusion.

Full clinical examination including:

General examination: vital signs as temperature (fever, core temperature >38,Hypothermia, core temperature below 35),blood pressure especially hypotension(the mean arterial pressure is less than 70 mmHg or the systolic blood pressure (SBP) is less than 90 mmHg or falls by more than 40 mmHg from the baseline SBP)with Deceased capillary refill or mottling ,pulse (Heart rate > 90 beat per minute),respiratory rate (> 20 breath per minute)and hypergycemia ,and abnormal colors e.g jaundice, pallor, and petechiae.

Neurological assessment including level of consciousness,convulsions, signs of increased intracranial tension or lateralization, and neck rigidity.

Cardiovascular assessment including presence of heart failure or shock and tachycardia auscultation for pericardial rub and murmur.

Respiratory assessment including color of lips, chest expansion, auscultation for air entry, crepitations, wheezing and presence of respiratory distress signs (Grunting, Nose flaring, Retractions, sweating, wheezing, body position) or respiratory failure assessment especially with pulse oximetry and arterial blood gases.

Abdominal assessment including tenderness, rigidity, and herniation.
Skin and soft tissue assessment including skin ulcers, cellulitis, gangrene, and surgical wound infection.
Bone and joint assessment for osteomyelitis and septic arthritis.
Throat assessment for pharyngitis.
All the above with consideration of searching for focus infection.
Quick SOFA score (qSOFA score)is a simple score consisting of three items; presence of each gives score 1, respiratory rate≥22/minute (1 point), change in mental status (Glasgow Coma scale <15) (1 point), systolic blood pressure <100 mmHg). Scores ≥2 predicts mortality [8].
Definitions of SIRS ,sepsis ,severe sepis ,and septic shock [9].
SIRS: presence of temperature >38°, or <36°, heart rate>90 bpm, respiratory rate>20, PaCO2,32 mmHg, white blood cells>12000/cmm, <4000/cmm or >10% bands.
Sepsis: infection and two or more SIRS.
Severe sepsis: sepsis and end organ dysfunction defined as: sepsis-induced hypertension, lactate above upper limits of laboratory normal, urine output<0.5 ml/kg/hr * two hours, PaO2/FiO2 <250 in absence of pneumonia, PaO2/FiO2 <250 in presence of pneumonia, creatinine>2mg/dL, bilirubin>2 mg/dL, platelet count100.000/uL and INR>1.5.
Septic shock: sepsis and a SBP<90mmHg or reduction of 40mmHg from baseline or evidence of low perfusion after adequate fluid loss
Investigations:
Routine investigations including:
Complete blood count,searching for leukocytosis (TLC> 11,000), leucopenia (TLC<4000) or Normal white cell count with >10% immature forms, Renal, liver functions test, serum electrolytes and INR and Other routine investigations to determine focus of infection (eg. Chest X-ray ,abdominalultrasonagraphy ,urine analysis,……)
Special investigations including
Identification of micobes revealed from Blood culture at least two blood cultures from different sites, and other cultures from any suspected site of infection such as ; sputum ,wound ,urine,…….etc
Measurement of presepsin level
The blood sample was collected after skin sterilization with the ethyl alcohol swap. 3 ml from peripheral venous blood was withdrawn from each patient using a disposable syringe under complete aseptic conditions, and the tests were done in the laboratory of Medical Microbiology and Immunology department, Zagazig University.
The kit uses a double-antibody sandwich enzyme-linked immunosorbentassay(ELISA) to assay the level of Human presepsinin samples. We Add presepsinto monoclonal antibody Enzyme well which was pre-coated with Human presepsinmonoclonal antibody, incubation; then, add presepsinantibodies labeled with biotin, and combined with Streptavidin-HRP to form immune complex; then carrying out incubation andwashing again to remove the uncombined enzyme. Then adding Chromogen Solution A, B, the color of the liquid changes into the blue, And at the effect of acid, the color finally becomes yellow. The chroma of color and the concentration of the Human Substance presepsinof sample were positively correlated.
Sensitivity:0.024mg/L (The sensitivity of this assay,was defined as the lowest protein concentration that could be differentiated from zero.It was determined by sub tracting two standard deviations to the mean optical density value of twenty zero standard replicates and calculating the corresponding concentration.)
Assay range: 0.03mg/L→9mg/L

Statistical analysis
Data collected throughout history, basic clinical examination, laboratory investigations and outcome measures coded, entered and analyzed using Microsoft Excel software. Data were then imported into Statistical Package for the Social Sciences (SPSS version 20.0) **(**. According to the type of data qualitative represent as number and percentage, quantitative continues group represent by mean ± SD, median and range for non-parametric data. Kruskal Wallis test was used to compare more than two groups regarding continuous non-parametric variables.Mann Whitney test was used too compare between two groups regarding non-parametric continuous variable. Spearman's correlation test was used to assess strength and directions of correlation between two continuous non-parametric variables. ROC curve analysis was used to assess the best cutoff of certain parameter in diagnosis of health problem. The levelstatistical significance was set at $P<0.05$.

RESULTS

Baseline data of the studied patients:

We compared between the three studied groups regarding basic demographic, comorbidities, clinical, and laboratory data as shown in table (1) and our results showed that there was no significant difference between the 3 groups regarding age & gender distribution, comorbidities including hypertension & diabetes mellitus and history of smoking. Also we found that the most common source of infection was pneumonia followed by urinary tract infections in the three studied groups.

Regarding clinical and laboratory data, our results showed that there a statistical significant difference between the three studied groups regarding SBP, DBP, MAP, HR, TLC, INR, serum sodium, CRP, PCT and serum PSP levels. While no statistical significant difference was observed between them regarding other variables.

Regarding culture results, our results showed that 48 (73.8%), 16 (69.7%) and 8 (100.0%) were having positive culture results in group I, II and III respectively.

Association between serum presepsin level and spectrum of disease:

There was a statistical significant difference between serum PSP level in patients with positive culture in the three group of the study with highest level was observed in patent with septic shock "group III" (Table 2).There is significant association between serum presepsin and disease severity, on pairwise comparison, the difference is significant between each two individual groups with highest level was associated with septic shock (Table 3).

Correlation between serum presepsin and baseline data of the studied patients:

Presepsin significantly positively correlated with ALT,AST, Creatinine, C reactive protein andprocalcitoninmarker. It was significantly negatively correlated with platelet count (Table 4). Further statistical analysis was done by using linear regression analysis and results showed that there a significant association between PSP level and PCR, CRP and AST (table 5,figure 1).

Diagnostic performance of serum presepsin:

The best cutoff of serum presepsin in diagnosis of infection among the studied patients was ≥1.95 mg/mL with area under curve 0.692, sensitivity 80% and specificity 71% while at cutoff ≥2.4 mg/mL, it can identify presence of sepsis with area under curve 0.714, sensitivity 78.3% and specificity 69% (Table 6). Serum presepsin level ≥7.9 mg/mL can predict development of septic shock with area under curve 0.969, sensitivity 90% and specificity 92% (Table 5).

Table 1: Baseline of the studied patients (N=96)

Parameter	Group (I) N=65	Group (II) N=23	Group (III) N=8	X^2	p-value
Sex					
Female	32	9	3	0.942	0.624
Male	33	14	5		
HTN					
-VE	37	14	4	0.297	0.862
+VE	28	9	4		
DM					
-VE	49	15	6	0.908	0.635
+VE	16	8	2		
Smoking					
-VE	32	11	4	0.017	0.991
+VE	33	12	4		
Blood culture					
-VE	17	7	0	3.08	0.214
+VE	48	16	8		
Suggested sources of infection					
Pneumonia	40 (61.5%)	10(43.5%)	5 (62.5%)		
UTI	15 (23.1%)	7 (30.3%)	1 (12.5%)		
Dermal	3 (4.6%)	2 (8.7%)	1 (12.5%		
abdominal	2 (3.1%)	1 (4.4%)	1 (12.5%)		

endocarditis others	3 (4.6%) 2 (3.1%)	1 (4.4%) 2 (8.7%)	0 (0.0%) 0(0.0%)		
qSoFA score: 2-3 4-5 6-7 ≥8	 30 (46.1%) 31 (47.7) 4 (6.2) 0 (0.0%)	 0 (0.0%) 11 (47.8%) 10 (43.5%) 2 (8.7%)	 0 (0.0%) 0 (0.0%) 1 (12.5%) 7 (87.5%)		
Age Mean± SD	52.8 ± 10.5	51.4 ± 11.2	46.5 ± 11.3	***F*** 1.271	0.285
SBP Mean± SD	115.2 ±14.6	105.8 ± 13.7	85.8 ± 13.2	***F*** 16.71	<0.001
DBP Mean± SD	76.5 ±16.2	69.3 ± 12.4	56.3 ± 9.5	***F*** 7.466	0.001
MAP Mean± SD	87.4±12.4	75.6 ± 14.5	62.9 ± 11.6	***F*** 17.28	<0.001
HR Mean± SD	88.7 ±9.5	95.8 ± 7.0	101.4 ± 5.2	***F*** 11.348	<0.001
TLC Mean± SD	17.2 ± 8.9	16.3 ± 9.9	17.3 ± 4.2	***F*** 17.462	<0.001
Hemoglobin Mean± SD	10.5 ± 2.9	10.4 ± 2.5	12.6 ± 2.6	***F*** 2.089	0.129
Platelet count Mean± SD	242.4 ± 108.5	211.7 ± 103.0	286.5 ± 145.2	***KW*** 2.05	0.358
Serum creatinine Mean± SD	0.7 ± 0.3	0.8 ± 0.3	0.7 ± 0.3	***F*** 0.237	0.789
BUN Mean± SD	20.6 ± 8.3	33.2 ± 5.3	56.8 ± 6.0	***F*** 93.706	<0.001
ALT Mean± SD	31.3 ± 45.9	57.6 ± 89.7	65.3 ± 136.1	***KW*** 10.04	0.007
AST Mean± SD	46.8 ± 53.8	74.8 ± 88.4	70.0 ± 119.1	***KW*** 3.292	0.129
Serum bilirubin Mean± SD	0.7 ± 0.6	2.8 ± 5.3	1.1 ± 1.1	***KW*** 13.82	0.001
Serum albumin Mean± SD	3.1 ± 0.6	2.9 ± 0.7	3.2 ± 0.4	***F*** 1.858	0.162
Random blood sugar Mean± SD	153.2 ± 55.7	171.7 ± 81.7	187.1 ± 94.7	***KW*** 0.405	0.816
INR Mean± SD	0.99 ± 0.7	1.47 ± 0.93	1.7 ± 1.08	***KW*** 5.098	0.008
Serum sodium Mean± SD	141.5 ± 13.15	133.6± 15.37	131.7 ± 11.08	***F*** 3.934	0.023
Serum potassium Mean± SD	3.97 ±1.66	3.65±1.19	4.12±1.88	***F*** 0.428	0.653
CRP Mean± SD	83.9 ± 86.2	72.7 ± 63.4	188.8 ± 85.3	***KW*** 9.465	0.009
PCT Mean± SD	5.0 ± 10.4	16.3 ± 20.5	38.4 ± 25.1	***KW*** 23.08	<0.001
PSP Mean± SD	2.5 ± 2.2	4.0 ± 3.1	13.7 ± 5.8	***KW*** 24.73	<0.001

SBP: systolic blood pressure, DBP: diastolic blood pressure, MAP: mean arterial blood pressure,HR: heart rate BUN: blood urea nitrogen,cr:creatinine, RBS: random blood sugar TLC total leukocytic count, CRP C reactive protein PSP: presepsin, PCT: procalcitonin INR international normalized ratio, KW: Kruskal-Wallis Test, F: anova test, X^2: chi square test.

Table 2: Relation between serum presepsin and result of blood culture in the groups of the study:

Presepsin	**Group (I)**	**Group (II)**	**Group (III)**	***KW***	**p-value**
Positive Culture	2.7 ± 2.4	4.2 ± 2.9	13.7 ± 5.8	25.289	< 0.001
Negative Culture	2.1 ± 1.5	3.6 ± 3.8	-	0.170	0.679
KW	1.253	2.571			
p-value:	0.263	0.109			

PSP: presepsin, KW: Kruskal-Wallis Test,

Table 3: Relation between serum presepsin and spectrum of disease:

	Infection	**Sepsis**	**Septic shock**	**KW**	**P**
PSP	**2.0 (0.6-11.1)**	**3.05 (1.6-10.2)**	**12.65 (6.4-22.52)**	**45.756**	**<0.001***
Pairwise comparison	**P_1 0.027***	**P_2 0.046***	**P_3 0.001****		

KW Kruskal Wallis test *p<0.05 is statistically significant P1 the difference between infection and sepsis group P2 the difference between sepsis and septic shock groups P3 the difference between infection and septic shock groups

Table 4: Correlations between markers and other parameters

Variable		**PSP**
CRP	**r**	**0.235**
	p	**0.021***
PCT	**r**	**0.373**
	P	**<0.001***
Age	**r**	**-0.022**
	P	**0.831**
TLC	**r**	**-0.074-**
	P	**0.471**
Hemoglobin	**r**	**0.131**
	P	**0.205**
Platelet count	**r**	**-0.310**
	P	**0.008***
Creatinine	**r**	**0.254**
	P	**0.018***
BUN	**r**	**-.170-**
	P	**0.098**
ALT	**r**	**0.279**
	P	**0.013***
AST	**r**	**0.289**
	P	**0.011***
Bilirubin	**r**	**0.093**
	P	**0.365**
Albumin	**r**	**-.161-**
	P	**0.117**
RBS	**r**	**0.126**
	P	**0.220**

R Spearman rank correlation coefficient *p<0.05 is statistically significant BUN: blood urea nitrogen, ,RBS: random blood sugar PCT procalcitonin TLC total leucocytic count CRP C reactive protein INR international normalized ratio .

Table (5): Linear regression analysis of significant correlations

Variable	Coefficients (95.0% Confidence Interval)	p-value
PCT	0.200 (0.186 – 0.214)	0.000
CRP	0.004 (0.001 – 0.007)	0.006
PLT	0.000 (-0.002 – 0.001)	0.655
Cr	0.002 (-0.002 – 0.006)	0.368
ALT	.004 (-0.001 – 0.009)	0.096
AST	.005 (0.003 – 0.007)	0.000

Table 6: AUC, cutoff and validity of serum presepsin in diagnosis of positive culture, sepsis and septic shock:

	Area	Cutoff	P	95% Confidence Interval		Sensitivity	Specificity
				Lower	Upper		
Infection	0.692	≥1.95	0.012*	0.547	0.797	80.0%	71.0%
Sepsis	0.714	≥2.4	0.049*	0.505	0.782	78.3%	69.0%
Septic hock	**0.969**	**≥7.9**	**<0.001***	**0.929**	**1.000**	**90.0%**	**92.0%**

*p<0.05 is statistically significant

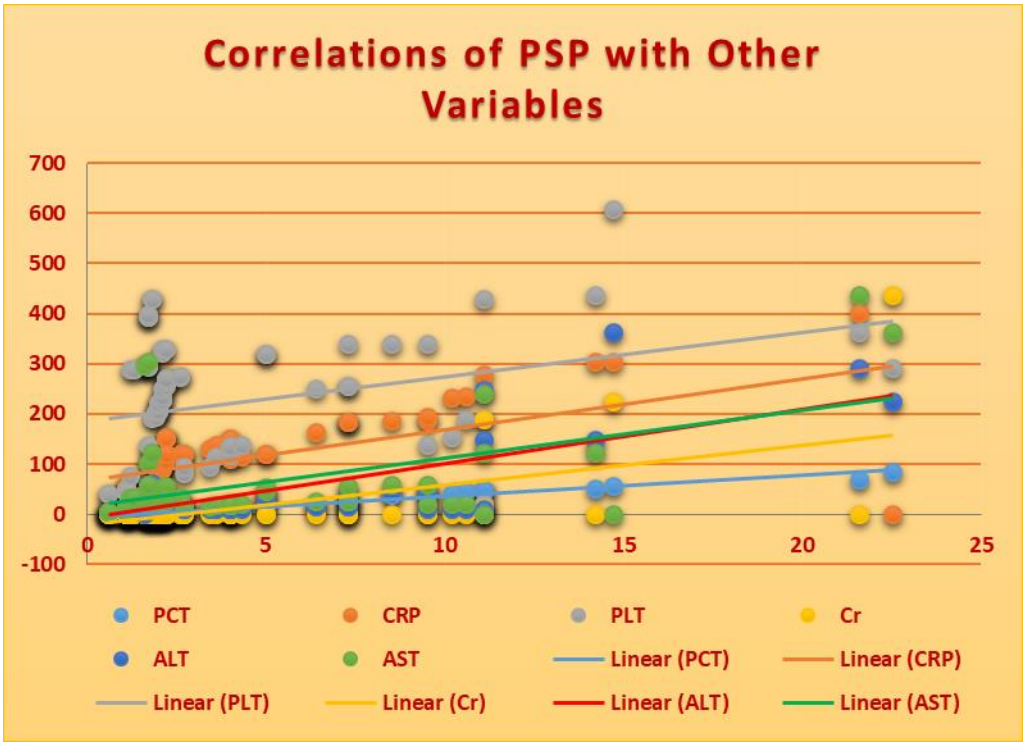

Figure 1: Significant correlation between PSP and different variables

DISCUSSION

Sepsis is a life-threatening ailment, with poor and highly diverse clinical displays, hard to detect. Sepsis persistedto be a diagnostic contest to clinicians, causing millions of deaths worldwide

each year. Hence, early recognition of sepsis is crucial for better disease outcome. [10]

Blood cultures are a gold standard for diagnosing sepsis. Despite the great advantages, blood cultures also have some limitations; they are often negative, especially in patients previously treated with antibiotics, and need several days to get the result. On waiting for that results, specific management of those critically ill patients with sepsis may be delayed with possible poor outcome. Among these biomarkers, procalcitonin (PCT) and C-reactive protein (CRP) had established rles for diagnosing infections yet lacking high specificity [11].

Soluble CD14 subtype (sCD14-ST), also known as presepsin, was first introduced in 2005 as a marker specifically upsurged in patients with sepsis on comparing it with level within healthy controls and those presenting with non-infectious systemic inflammatory response syndrome (SIRS). Presepsin specifically rises in patients with bacterial sepsis as the mechanism of its secretion linked to bacterial phagocytosis [12].

Positive blood culture was found in 75% of patients; out of them, 66.7%, 22.2% and 11.1% had infection, sepsis and septic shock respectively. About 57% and 24% had pneumonia and urinary tract infection respectively. Presepsin significantly positively correlated with procalcitonin and C reactive protein ($p<0.05$). It significantly negatively correlated with platelet count ($p<0.05$). There is significant association between serum presepsin and disease severity, with highest level in septic shock followed by sepsis ($p<0.05$). The best cutoff of serum presepsin in diagnosis of infection was ≥1.95 mg/mL with sensitivity 80% and specificity 71% while at cutoff ≥2.4 mg/mL, it can predict sepsis, sensitivity 78.3% and specificity 69%. Presepsin cutoff ≥7.9 mg/mL can predict septic shock with sensitivity 90% and specificity 92%.

Hypertension and diabetes were the commonest comorbidities prevailed among the studied patients. Those are among factors that enhance the risk of infection [13].

High INRencountered among the studied patients infers the ailment of coagulopathy associated with sepsis. This was in accordance with a previous study testified that Acute vascular endothelial dysfunction is a fundamental event in the pathogenesis of sepsis, increasing vascular permeability, encouraging activation of the coagulation cascade, tissue edema and compromising perfusion of vital organs [14].

Laboratory results of the studied patients settled results of leukocytosis, thrombocytopenia, impaired liver and kidney functions and hyperglyceamia that explain presence sepsis and septic cases associated with organ injury.

In agreement with the current study, renal and hepatic affection in infectious diseases may occur by a diversity of mechanisms: including the systemic release of endotoxin or other toxins with stimulation of the inflammatory cascade during septicemia or by ischemic hurt may result from insufficient perfusion brought by septic shock [15].

Also in agreement with the present results, hematologic deviations in septic patients are by leukocytosis (Infection stimulates the production of cytokines which trigger the release of granulocytes from the bone marrow) and thrombocytopenia (can either occur due to diminished platelet production or increased platelet turnover; Platelet activation diminishes platelet life span as activated platelets are rapidly cleared from the circulation) [16].

Also in harmony with the present results, hyperglycemia is regularly occurring in sepsis, even in non-diabetics. It is a consequence of inflammatory response and stress, so its occurrence is related to severity of illness. However, not all severely ill develop hyperglycemia and some do even in mild disease [17].

On comparison between positive and negative culture regard PSP, Positive cases were significantly higher PSP was 4.24±3.98. onthis regard to detection of positive culture, PSP significant area under curve and cutoffs(with Sensitivity 80%, Specificity 71%).

The results in previous paragraph were consistent with a study showed that Presepsin levels in patients with systemic bacterial infection and localized bacterial infection were significantly higher than in those with nonbacterial infections. The cutoff value of presepsin for discrimination of bacterial and nonbacterial infectious diseases was determined to be 600 pg/ml, of which the clinical sensitivity and specificity were 87.8 % and 81.4 %, respectively [18].

Positive result in culture was 75%.Bacteria are by far the most common causative microorganisms in sepsis, and cultures are positive in about 50% of cases It is known that cultures lack the sensitivity to identify all bacteria. Postulated reasons include prior antibiotic exposure, sampling error, insufficient volume for blood cultures, poor transport conditions, and slow-growing or fastidious bacteria. [19]

Another study showed the most common sites of infection were the lungs (31.0%), followed by intra-abdominal sites (26.3%), the urinary tract (18.4%),

and soft tissue (10.9%) in harmony to finding of the current result [20].

In the present study, septic shock group was significantly higher PSP than other group and sepsis group was significantly higher than infection but regard to sepsis, PSP was significant AUC and cutoff with sensitivity 78.3% and specificity 69.0% and regard to septic shock markers were significant. In agreement with our study, previous study stated thatpresepsin was found to have the highest diagnostic accuracy for discriminating non-sepsis from sepsis and septic shock group as well as between sepsis groups [21].

A previous Egyptian study reported that presepsin had acceptable ``diagnostic performance for bacterial infections in patients with decompensated cirrhosis [22].

In partial agreement, accordingly, when evaluating differences in presepsin concentrations between two severity groups of sepsis patients, ***Aliu-Bejta et al.*** found Presepsin concentrations were significantly higher on admission in patients with septic shock compared to patients with sepsis,their study revealed that presepsin concentrations had a good capacity for distinguishing disease severity. [11]

Also, Similar to our results, previous studiesreported significantly higher levels of presepsin in septic shock patients compared to septic patients without shock [7, 23-26].

The study had some limitations; including relatively simple size, being single center, and being cross-sectional study. So, large scale multicentric prospective studies to verify diagnostic performance of presepsin.

Recommendations:

In suspicion of infection and sepsis, we recommend routine presepsin assessment being involved in the early detection of sepsis severity groups.

History taking, Clinical and routine laboratory assessment of septic patients for Taking into account any abnormalities that disturb presepsin level measurement.

Larger study is needed as our study was little sample size due to high cost concerning estimation of PRESPSIN plusSerial estimation of the biomarkers is needed to identify the level after time of sepsis analysis.

CONCLUSIONS

Presepsin is a promising marker for diagnosis of infection and sepsis. It has capability for differentiating between sepsis severity groups**.**

CONFLICT OF INTEREST

None

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