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ORIGINAL ARTICLE

Role of Serum Tenascin-C in Sepsis

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ABSTRACT

Background: Sepsis can range in severity from infection to septic shock, and it can result in multiple organ dysfunction syndrome (MODS) and death. Tenascins are matrix glycoproteins located extracellular that are made during multicellular organism growth and involved in many pathological processes such as tissue damage, tumor angiogenesis metastasis, and inflammation.

Objectives: The aim of this study is to investigate the relationship between serum Tenascin- C levels and sepsis and disease severity in Intensive Care Unit (ICU) patients.

Methods: a case control study, selected participants included 9 apparently healthy subjects, 20 patients with sepsis in ICU and 10 diseased patients without sepsis in ICU. All patients were subjected to full clinical assessments of patients by SOFA score and Lab tests: (CBC, PCT, CRP, LFT&KFT). Tenascin C was measured by ELISA for all participants.

Results: The mean age for all groups is 21-70. There is a high significant increase in CRP, PCT, TLC, urea & creatinine, ALT, and AST in septic patients in relation to that of non-septic patients. Hemoglobin and albumin levels show a significant decrease in septic patients than that in non-septic patients. Sensitivity of tenascin to predict cases with sepsis vs those without sepsis was 75% and specificity was 100%.

Conclusion: In septic patients, the level of serum Tenascin-C can help with early sepsis diagnosis and severity assessment.

Key words: Sepsis; ICU; Tenascin–C.

INTRODUCTION

Sepsis can range in severity from infection to septic shock, and it can result in multiple organ dysfunction syndrome (MODS) and death. Since the early 1990s, the concepts of sepsis and septic shock have rapidly developed. Initial beliefs were established on the idea that sepsis was initiated by a host's systemic inflammatory response syndrome (SIRS) to infection. Severe sepsis refers to sepsis complicated by organ dysfunction which could progress to septic shock which is defined as sepsis induced hypotension persisting despite adequate fluid resuscitation [1]. The European Society of Intensive Care Medicine (ESICM) task force and the Society of Critical Care Medicine (SCCM) revised the definitions of sepsis, septic shock, and organ failure in 2016, after they had remained relatively unchanged for more than twenty years [2]. Sepsis is currently defined as life-threatening organ dysfunction caused by a dysregulated host response to infection. Organ dysfunction is known as an increase of 2 or more points in the Sequential Organ Failure Assessment (SOFA) score (table 1) [3]. SOFA score does not identify those patients' organ dysfunction is truly due to infection, but rather helps identify patients who potentially have a high risk of dying from infection [4].

Sepsis was identified as the presence of microbiological infections, systemic inflammatory response syndrome, acute organs failure and at least two of these parameters: temperature >37.9 °C or <35.9 °C; heart rate >95 beats min; PaCO2 < 32 mmHg or Respiratory rate >20 beats/min, white blood cell (WBC) count >11,000 or < 4000 cells/mm3, > 10% presence of immature forms [5].

A machine algorithm was validated recently for the prediction of sepsis by use of 6 vital signs: systolic and diastolic blood pressure, heart rate, respiratory

rate, peripheral capillary oxygen saturation and temperature [6].

The main lines of management of sepsis are securing airway, stabilizing respiration, establishment of venous access and restoring perfusion, Fluid resuscitation, use of vasopressors, control of septic focus, antimicrobial therapy, glucocorticoids, insulin and cooling [7,8].

After discharge from hospital, sepsis has an increased risk of death as well as an increased risk of another septic condition and hospital admissions (10 % are readmitted). Most of the deaths occur at the first six months but the risk is still high at 2 years [9].

SEPSIS BIOMARKERS

The availability of accurate sepsis biomarkers to facilitate diagnosis could be of use. This will enable timely appropriate treatment to be started, thus optimizing a patient's chances of survival [10,11].

CRP is synthesized by the liver. C-reactive protein was the first pattern recognition receptor (PRR) to be identified. Normal plasma levels of CRP are usually as less than 10 mg/L. Plasma levels increase within 4 to 6 h after initial tissue injury and continue to increase several hundred folds within 24 to 48 h [12,13].

Procalcitonin (PCT) is a aminoacid peptide precursor of the hormone calcitonin, which is thought to be a well-reliable diagnostic and prognostic marker of septic condition, which differentiates the inflammatory responses from the bacterial infections. In healthy persons, PCT is secreted only in the cells of the thyroid gland, but during an infection it is released up to a 1000-fold increase from all tissues and cells in the host [14,15].

In adults, Tenascin-C expression is confined to the site of tissue damage, which is typically transient, and Tenascin-C level expression returns to baseline once tissue recovery is completed. Tenascin-C high expression of is common in tissue remodeling, inflammation, and autoimmune diseases on the other hand. Injury and infection will trigger the make of Tenascin-C, which allows the body respond to bacterial lipopolysaccharide (LPS) with an efficient immune response. Tenascin-C stimulates proinflammatory cytokines synthesis in the macrophages which is activated by LPS through toll like receptor 4 (TLR4) and decreases the anti-inflammatory cytokines synthesis. So, Tenascin-C shares in regulating the inflammation axis in LPS-activated TLR signaling [16].

Serum Tenascin-C is also relevant to prognosis in septic patients as Tenascin-C serum levels are an independent prognostic factor and septic patients with Tenascin-C levels ≥ 56.8 pg/mL have a highly increased 30-day mortality rate [17,18].

Serum Tenascin-C levels were found to be associated with serum inflammatory factors such as CRP and IL-6 in sepsis patients. After induction of LPS, Tenascin-C was overexpressed by macrophages, which increases the make and secretion of proinflammatory cytokines by macrophages, promoting by this the inflammatory response of Toll-like receptor 4 (TLR4). Rapid early diagnosis and intervention are currently a major challenge for septic patients in the ICU. As a result, new biomarkers discovery is critical to make this aim, as well as introducing individualized care and enhancing septic patient prognosis [16,19].

So, we aimed to search the relationship between serum disease severity and serum Tenascin- C levels in patients with sepsis at ICU.

METHODS

This is a case control study conducted on patients admitted to (ICU) in Zagazig University Hospitals during the period from March 2019 till November 2019. A total of 39 participants were enrolled in this study and classified into three groups: group 1 included 9 apparently healthy subjects serving as control group. Group 2 included 20 patients with sepsis in ICU; group 3 included 10 diseased patients without sepsis in ICU. A written informed consent was obtained from all participants, the study was approved by the research ethical committee of Faculty of Medicine, Zagazig University. The study was done according to The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans. Inclusion criteria: Patients have sepsis diagnosed by laboratory investigations and clinical presentation older than 18 years. Criteria of exclusion: patients refusing to participate, causes of fever other than sepsis, causes of increase total leucocytic counts other than sepsis, tumor and autoimmune disease and participants < 18years old.

The patients were evaluated by: Full clinical assessment which includes complete history taking, clinical examination and SOFA score at time of admission. Routine laboratory testing including: - CRP, Procalcitonin, Complete blood count (CBC), Liver function tests, Kidney function tests, Blood cultures and sensitivity tests.

Specimen collection and storage: Three ml of venous blood by vein puncture were collected under

complete aseptic condition from all subjects in a sterile separator gel tube for serum isolation and left to clot. Centrifugation was done for 20-min at the speed of 2000 -3000 r.p.m. and supernatant removed and stored at (- 80) C till analysis.

Measurements of Tenascin – C: Tenascin-C was measured in serum samples by ELISA. Kit was provided from SunRed biotechnology company (China) Catalogue No. 201-12-1415 named Human Tenascin-C (TN-C) ELISA Kit. This ElISA kit is based on the principle of double antibody sandwich technique to detect human Tenascin-C.

STATISTICAL ANALYSIS

The data was analyzed by SPSS 19.0 statistical software. Categorical variables were shown as frequency (percentage) and continuous variables were shown as median (quartile). We did the Wilcoxon-Mann-Whitney test to compare continuous variables between patients who survived and patients who died like: SOFA score, age, CBC, LFT, KFT, CRP, ICU time, Procalcitonine, blood culture & sensitivity. The chi-square test was performed to compare categorical variables between survivors and nonsurvivors, including gender, site of infection, the presence of mechanical ventilation and septic shock. Spearman's rank sum test was performed to analyze the correlations between Tenascin-C and age, SOFA scores, ICU time, serum creatinine, WBC, CRP.

RESULTS

Demographic data: group 1: 5 males and 4 females aging from 29 to 67 years old serving as control group while in group 2: 14 males and 6 females aging from 21 to 71 years old and group 3: 7 males 3 females aging from 45 to 69 years old.

There was a highly significant increase in CRP, PCT as well as TLC in septic patients in relation to that of non-septic patients while haemoglobin level shows significant decrease in septic patients than that in non-septic patients. As regards platelets there was no significant difference between either group. A highly significant increase was observed in urea, creatinine, ALT & AST among diseased patients with sepsis in comparison with patients without sepsis. As regards serum albumin, it shows a significant decrease in septic group in comparison to non-septic group (table 2). It was revealed that of sepsis cases 14 cases died by a percent of 70%, 3 discharged by a percent of 15% and 3 still in ICU by a percent of 15%. Mean SOFA score was 6.75 ± 2.97. Regarding patients without sepsis who were admitted to ICU due to different causes, 4 of them had died representing 40 % of their number (table 3). There was significant increase in Tenascin-C level among patients with sepsis when compared with healthy and among patients with sepsis when compared with patients without sepsis, while no significant difference was obtained between non septic patients and healthy control (table 4). The sensitivity of tenascin to predict cases with sepsis vs control was 75%, specificity was 100%, PPV was 95% and NPV was 70% at a cut off >8100 with AUC of 0.878 (95% CI was 0.756 - 0.999) (Table 5) (figure 1), while the sensitivity of Tenascin-C to differentiate septic from non-septic cases was 75%, specificity was 100%, ppv was 95% and NPV was 42.1 % at a cut off >9000 (Table 6) with AUC of 0.865 (95 % CI 0.73-0.99) (figure 2).

Table 1: SOFA Score

System	Score				
	0	**1**	**2**	**3**	**4**
Respiration PaO_2/FiO_2 (mmHg)	≥400	<400	<300	<200 with respiratory support	<100 with respiratory support
Coagulation Platelets ($x10^3/\mu L$)	≥150	<150	<100	<50	<20
Liver Bilirubin (mg/dL)	<1.2	1.2-1.9	2.0-5.9	6.0-11.9	>12.0
CVS MAP or catecholamine administration (□g/kg/min)	MAP ≥70mmHg	MAP <70mmHg	Dopamine <5 **or**dobutamine (any dose)	Dopamine 5.1-15 **or** epinephrine ≤0.1 **or** nor-epinephrine ≤0.1	Dopamine >15 **or** epinephrine >0.1 **or** nor-epinephrine >0.1
CNS - GCS	15	13-14	10-12	6-9	<6
Renal Creatinine (mg/dL)	<1.2	1.2-1.9	2.0-3.4	3.5-4.9	>5.0
Urine output (mL/d)				<500	<200

PaO_2: Partial arterial pressure of oxygen, FiO_2: Fraction of inspired air oxygen, CVS: Cardiovascular System, MAP: Mean Arterial Pressure, CNS: Central Nervous System, GCS: Glasgow Coma Scale

Table 2: Comparison between diseased with Sepsis and diseased without sepsis according to CRP, PCT TLC, Hb, platelets, liver function tests & kidney function tests-:

	Diseased with Sepsis (n = 20)		**Diseased without sepsis (n = 10)**		**Test of sig.**	**P**
CRP						
Range	35.0 – 284.0		2.0 – 34.0		**MWW***	**<0.001***
PCT						
Range	1.0 – 110.0		0.04 – 0.50		**H=28.669***	**<0.001***
TLC						
Range	12.0 – 27.0		5.0 – 11.0		**MWW***	**<0.001***
HB						
Range	7.40 – 13.0		8.0 – 16.0		**MWW***	**0.003***
Platelet						
Range	75.0 – 400.0		20.0 – 440.0		**MWW**	**0.215**
Bilirubin mg/dl						
Range	0.30 – 3.2		0.2 – 3.2		**H=-0.946**	**0.352**
Albumin g/dl						
Range	1.70 – 3.80		2.10 – 4.50		**MWW***	**<0.001***
AST IU/L	**No.**	**%**	**No.**	**%**		
Normal	**11**	**55.0**	**5**	**50.0**	**χ^2= 7.183***	**MCp= 0.030***
Elevated	**9**	**45.0**	**5**	**50.0**		
ALT IU/L						
Normal	**11**	**55.0**	**5**	**50.0**	**χ^2= 7.183***	**MCp= 0.030***
Elevated	**9**	**45.0**	**5**	**50.0**		
Urea md/dl						
Range	10.0 – 130.0		12.0 – 68.0		**H=8.434***	**0.015***
Creatinine md/dl						
Range	0.40 – 8.0		0.40 – 6.50		**H= 5.330**	**0.070**

MWW: Mann-Whitney U test
H: H for Kruskal Wallis test,
χ^2: **Chi square test** MC: **Monte Carlo**

Table 3: Distribution of the studied diseased cases according to prognosis and SOFA score.

	With Sepsis (n=20)		**Without sepsis (n=10)**	
	No.	**%**	**No**	**%**
Prognosis				
Died	**14**	**70.0**	**4**	**40%**
Survival	**6**	**30.0**	**6**	**60%**
SOFA score				
Range	**3.0 –14.0**		**0.0- 2.0**	

Table 4: Comparison between the three studied groups according to Tenascin-C level

Tenascin-C	**Healthy control (n = 9)**	**Diseased with Sepsis (n = 20)**	**Diseased without sepsis (n = 10)**	**H**	**P**
Range ng/l	**950.0 –12000.0**	**2000.0 –82000.0**	**464.0 –50000.0**	**8.522***	**0.014***
IQR	**1410.0 –7000.0**	**8000.0 –22350.0**	**2800.0 –18000.0**		
Sig. bet. Groups.	**p_1=0.002*,p_2=0.005*,p_3=0.280**				

H: H for Kruskal Wallis test, pairwise comparison bet. each 2 groups were done using Post Hoc Test (Dunn's for multiple comparisons test)

IQR: Interquartile range
p: p value for comparing between the studied groups
p_1: p value for comparing between **Sepsis in ICU** and **diseased ICU not sepsis**
p_2: p value for comparing between **Sepsis in ICU** and **Healthy control**
p_3: p value for comparing between **diseased ICU not sepsis** and **Healthy control**
*: Statistically significant at $p \leq 0.05$

Table 5: Predictive value of Tenascin –C to detect sepsis in sepsis cases vs non sepsis cases

	AUC	95%CI	P value	Cutoff	Sensitivity	Specificity	PPV	NPV
Tenascin-C	**0.865**	**0.736 -0.994**	**0.001**	**>9000**	**75.0**	**100.0**	**95.0**	**42.1**

AUC: Area Under a Curve p value: Probability value
CI: Confidence Intervals
NPV: Negative predictive value PPV: Positive predictive value
*: Statistically significant at $p \leq 0.05$

Table 6: Predictive value of Tenascin –C to detect sepsis in sepsis cases vs control

	AUC	95%CI	P value	Cutoff	Sensitivity	Specificity	PPV	NPV
Tenascin-C	**0.878**	**0.756-0.999**	**0.001**	**>8100**	**75.0**	**100.0**	**95.0**	**70.0**

AUC: Area Under a Curve p value: Probability value
CI: Confidence Intervals
NPV: Negative predictive value PPV: Positive predictive value
*: Statistically significant at $p \leq 0.05$

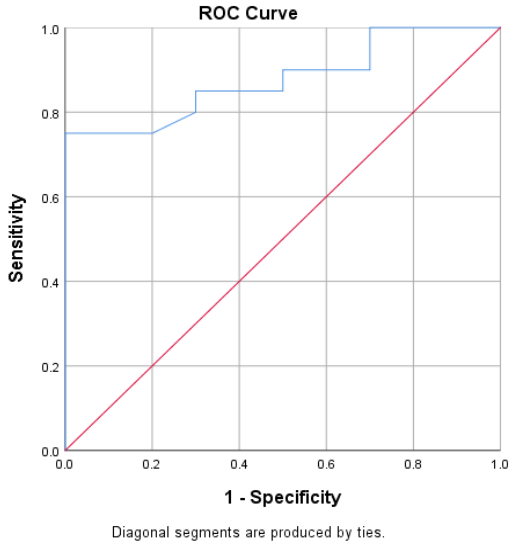

Figure 1: ROC curve for TN-C to predict sepsis cases vs control
The sensitivity of Tenascin-C to predict cases with sepsis vs control was 75%, specificity was 100%, a cut off > 8100 with AUC was .878

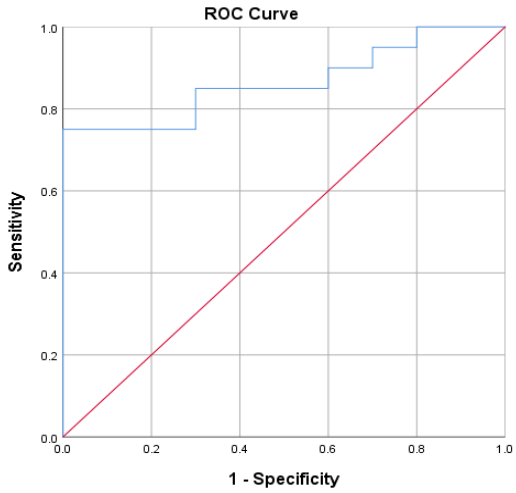

Figure 2:ROC curve for TN-C to predict sepsis cases vs non sepsis
The sensitivity of Tenascin-C to predict cases with sepsis vs that without sepsis was 75%, specificity was 100% at a cut off >9000 with AUC = .865

DISCUSSION

Tenascin-C expression in adults is restricted to the site of tissue damage and is transient, with Tenascin-C expression returning to normal once tissue repair is done. Inflammation, tissue remodelling, and autoimmune disease are all known to cause sustained high expression of Tenascin-C [20]. Tenascin-C is a broad inducer of inflammation and is involved in the pathogenesis of sepsis through a few mechanisms [21,22].

So, this study was designed to search the relationship between disease severity and serum Tenascin-C in patients with sepsis.

The result of this study revealed that there was statistically significant increase in CRP level in patients with sepsis when compared with patients without sepsis ($p< .001$). This result was in accordance with similar papers who reported that CRP exhibited a higher level in serum of sepsis group than the non-septic patients in ICU [23,24,25].

The present study revealed an elevated level in PCT among sepsis group when compared with non-septic group. This result came in accordance with that of Zhao et al [26]. The present study revealed that there is no significant difference in platelet count in sepsis compared with non-septic patients. This result was in disagree with that of Tambo et al who reported that platelet count was significantly lower in sepsis group than in non-septic group among patients with obstructive acute pyelonephritis [27]. The results for urea and creatinine revealed a significantly higher value in septic than nonseptic patients. These results are in accordance with Van massenhove et al who reported that serum creatinine in patients with sepsis represents renal dysfunction [28].

In the present study serum albumin level and aminotransferase activity were significantly reduced in sepsis than in non-septic patients, this result agrees with that of Czupryna et al who found that in septic patients, albumin concentration and aminotransferase activity were lower, and that this was even lower in patients with severe sepsis. In the present study (14/20) 70 % died and (6/20) 30 % survived, 15 % discharged from ICU and 15 % stayed in ICU. In the present study, 60 % of examined patients (12/20) with sepsis the blood cultures were positive. In both groups, gram positive bacteria dominated with 45 % (9/12) e.g: staph haemoliticus, staph epidermidis and staph hominis. This result agrees with that of Czupryna et al who reported that 55.1 % of their patients had positive blood cultures, but with staph. aureus as the most common pathogen [29].

The mean of SOFA score was 6.75 ± 2.97. The result of Czupryna et al reported that SOFA score mean was 2 while in sepsis group the score ranged from 0-9 points in severe sepsis. The same results were obtained by Su et al who stated that SOFA score was of diagnostic value for sepsis severity [8,13]. In this study, the Tenascin – C level revealed that mean ± SD in healthy control was 4906.7 ±3743ng/l, which is highly statistical significant increase in septic patients when compared with healthy control. The septic patients had higher level of Tenascin –C than non-septic patients, while there is no significant difference between non septic patients and healthy control. These results agree with that of Yuan et al who reported that in septic

patients' serum Tenascin – C levels were significantly higher in non-survivor compared to survivors [24,30].

CONCLUSIONS

Serum Tenascin-C concentration in septic patients can help in early sepsis diagnosis and assessment of severity, according to the findings of this study. Our findings indicate that Tenascin-C may play a role in sepsis pathogenesis and could be used as a biomarker and therapeutic target. Tenascin-C had a sensitivity of 75% and a specificity of 100% in predicting sepsis cases compared to controls.

DECLARATION OF INTEREST

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

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None declared

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