Submitted: 26 October, 2023 Accepted: 22 December, 2023 Published: 08 May, 2024 DOI:10.22514/sv.2024.056

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Signa Vitae

ORIGINAL RESEARCH

The role of Asprosin in the diagnosis of diabetic and non-diabetic patients with sepsis

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Abstract

Sepsis is among the major causes of mortality in the world due to its delayed diagnosis and inadequate treatment. Due to the high morbidity and mortality rates, a widely accepted biomarker is required for its diagnosis. Present study aimed to evaluate the serum asprosin levels in septic patients and determine its potential use as a biomarker for its diagnosis. A prospective cohort study was performed with total 81 participants. These were divided in two groups, *i.e.*, 54 septic patients and 27 healthy volunteers (control group). Among 54 septic participants, half (27) were diabetic, and half (27) were nondiabetic. Blood samples (3 mL) were collected from the control group and the patients who were diagnosed with sepsis and transferred to serum seperator tube. Samples were fractioned and stored at −80 °C for further evaluation. These samples were used for the measurement of serum asprosin levels. Demographic characteristics and laboratory data of the patients were recorded in the study form prepared previously. The serum asprosin levels in the control group were 4.1 $\pm$ 1.4 ng/mL, 32.8 $\pm$ 7 ng/mL in the non-diabetic septic patients and 90.7 $\pm$ 5.5 ng/mL in diabetic septic patients. The asprosin level in the non-diabetic group were higher as compared to the control group (p: 0.012), and it was statistically significantly higher in the diabetic group compared to the non-diabetic septic and control groups (both: $p < 0.001$). Serum asprosin levels were significantly higher in patients with sepsis specially having diabetes. Serum asprosin levels may be beneficial for the diagnosis of sepsis especially in diabetic patients along with the other laboratory parameters white blood cells (WBC), C-reactive protein (CRP), sequential organ failure assessment score (SOFA score) and fewer.

Keywords

Asprosin; Diabetes mellitus; Diagnosis; Sepsis

1. Introduction

Due to weak host response to infection, human body can face life-threatening organ dysfunction known as “sepsis” [1]. Sepsis can damage many tissues and organs systems costing billions of dollars every year for treatment [2]. The incidence of sepsis is increasing day by day, especially in elderly patients with comorbid diseases [3]. Mortality rates due to sepsis and septic shock are known to be at a very high rate up to 26%. To facilitate the diagnosis of sepsis and septic shock, the third surviving sepsis guideline was published in 2016 [4]. These guidelines suggest that it is more useful to use Quick Sequential Organ Failure Assessment (qSOFA) criteria instead of Systemic Inflammatory Response Syndrome (SIRS) criteria for the diagnosis of sepsis. Quick Sequential Organ Failure Assessment (qSOFA) score is calculated for the diagnosis of sepsis which including altered mental status, systolic blood pressure $<$100 mm and respiratory rate $>$22/minute [1]. One point is given for each positive parameter. If this score is $\geq$2, it is clinically significant, and the patient is treated as sepsis. Quick Sequential Organ Failure Assessment (qSOFA) score is also used for the assessment for mortality rate in septic patients. However, to calculate qSOFA score, many other clinical parameters such as PaO_2/FiO_2, platelets count, bilirubin level, mean arterial pressure, Glasgow Coma Scale score and creatinine level are also observed. Many other biomarkers such as C-reactive protein (CRP), procalcitonin, interleukin 6, complement 3a, elastase, lipopolysaccharide binding protein and cluster of differentiation 14 (CD14) are already being used for the diagnosis of sepsis. Based on the updated surviving sepsis guideline published in 2021, the use of qSOFA criteria alone is not sufficient for the diagnosis of sepsis. There is a great need for a new and more appropriate biomarker that can be used for the diagnosis of sepsis [5].

The incidence of comorbid diseases is very high in septic patients [6]. It is reported that nearly 21.8% of patients with sepsis had diabetes mellitus (DM) [7] and these diabetic patients with sepsis belonged to higher age group. In these patients, sepsis can develop even after a simple infection due to neutrophil dysfunction. The mortality rate in diabetic patients,

the increases due to delayed diagnosis of sepsis, and the duration of hospitalization is much longer than other patients [8]. In another study, in which the etiology of sepsis was investigated in diabetic patients, the most common reasons of sepsis in diabetic patients were urinary tract infections, lung infections and soft tissue infections [9]. Considering all these reasons, early diagnosis, and treatment of sepsis in diabetic patients is highly needed.

Asprosin is a protein-based hormone secreted from white adipose tissues and released as a product from the C-terminal part of profibrillin [10]. It is encoded by the gene fibrilline 1 (*FBN1*) which also encodes Fibrillin-1 [11]. Asprosin regulates hepatic glucose release with G protein- cyclic adenosine monophosphosphate- protein kinase (cAMP-PKA) pathway, especially during fasting. It has an ameliorative effect on chronic inflammation. Elevated asprosin level is a risk factor in the pathogenesis of Type-2 Diabetes Mellitus [12–14]. However, there is still no strong biomarker in the literature that can be used for the diagnosis of sepsis. Considering that the mortality rates of diabetic patients are much higher in sepsis, the importance of a biomarker for the early diagnosis of these patients increases much more.

This study aimed to investigate serum asprosin levels in diabetic and non-diabetic patients with sepsis to determine whether asprosin levels could be used as a biomarker for the diagnosis of sepsis in diabetic patients.

2. Material and methods

The study was conducted in Trakya University, Edirne, Turkey. A total 81 participants were selected. These were divided into two groups, *i.e.*, control and diseased. The diseased group comprised of 54 participants which were further subdivided into groups, *i.e.*, septic patients with diabetes mellitus (type-II) and septic patients without diabetes. Each group comprised of 27 participants. Patients with any other comorbid condition(s) were excluded from the study. Twenty-seven healthy individuals of same age and sex were recruited as control group. The study of Diao *et al.* [14] was followed for power analysis. Sample size for each group (diabetic sepsis, non-diabetic sepsis, and control) was calculated as 14 with an effect size of 0.552, an alpha level of 5%, and a power of 95%. To increase the power and validity of the study, 27 participants were included in each group. Asprosin levels were measured from the blood samples collected at the time of admission. Diagnosis of sepsis of all participants in the diabetic and non-diabetic groups were confirmed with bacteriological culture. Samples of blood, urine, sputum, bronchoalveolar lavage and soft tissue swab were used for bacterial culture. Blood samples (3 mL) were collected from the control group and septic group and transferred to a serum separator tube. These blood samples were kept at 4 °C for 2 hours for coagulation. The samples were then centrifuged at 4 °C at 1000 rpm for 15 minutes, serum fractions were collected with the help of a micropipette and transferred to a microcentrifuge tube. Serum samples were stored at −80 °C for further laboratory evaluation. Demographic characteristics and laboratory data of the patients were recorded in the study form previously prepared. After 24 hours, serum samples were brought to room temperature. Human asprosin ELISA kit (Catalog no: OKEH04875, Aviva Systems Biology, SanDiego, USA) was used for the measurement of serum asprosin levels. The measurement was carried out in accordance with the manufacturer's instructions. The sensitivity range of this human asprosin kit was between 1.56 to 100 ng/mL. Complete blood count, venous blood gas, serum urea, creatinin, serum electrolites, HbA1C and CRP levels of the patients were also measured at the time of admission. Simultaneously HbA1C level of the control group patients were also recorded. In-hospital mortality, defined as death occurring within 28 days of admission was recorded on the study form. Statistical evaluation was performed using IBM SPSS Statistics for Windows, Version 19.0 (IBM Corp., Armonk, NY. USA). The Shapiro-Wilk test was used for the assessment of normal distribution. For quantitative variables that did not conform to normal distribution, group comparisons were performed using the Mann-Whitney U test. The relationships between qualitative variables were investigated using Pearson's Chi-square test and Fisher's exact test (if the expected value of at least one of the eyes in the 2×2 tables was below 5 and the expected value of at least 20% of the eyes in the multi-eyed tables was below 5). For descriptive statistics, quantitative variables are given as mean $\pm$ standard deviation, and qualitative variables are given as number and percentage. The significance level was determined as 0.05 in all statistical analyses.

3. Results

The demographic and medical characteristics of the recruited participants are shown in Table 1. The mean age of the participants was 65.7 ± 10.9 (mean $\pm$ standard deviation (SD)) years in the group with diabetic sepsis, 63.6 ± 12.3 years in the non-diabetic sepsis group and 61.9 ± 7.8 years in the control group. Eleven (40.7%) of the diabetic sepsis group, 7 (25.9%) of the non-diabetic sepsis group and 8 (29.6%) of the control group were female. No statistically significant difference was observed among the groups in terms of age and gender (p: 0.632, p: 0.479, respectively). Similarly, no statistically significant difference was observed among the diabetic and non-diabetic groups for in-hospital mortality (p: 0.293). Due to the absence of patients in our diabetic group without comorbidities, we are unable to observe the potential predictive effect of comorbidity. Nevertheless, when the potential effects of dementia and hypertension on asprosin were excluded from the analysis based on the likelihood of being less than other comorbidities, there was no statistically significant difference in the presence of coronary artery disease (CAD), cerebrovascular disease (CVD) and chronic obstructive pulmonary disease (COPD) comorbidities between diabetic septic and non-diabetic septic groups (p: 0.096). It was shown as "Comorbid disease (re-categorized)" in Table 1.

The vital signs and laboratory findings of the various groups are shown in Table 2. The mean duration of diabetes in the diabetic sepsis group was 23.3 ± 3.4 years and HbA1C levels were $7.8 \pm 0.5\%$. The mean HbA1C level of the control group was $4.8 \pm 0.7\%$. The normal range of HbA1C is 4–

TABLE 1. Demographic and medical characteristics of diabetic and non-diabetic patients diagnosed with sepsis in the Trakya university emergency department in 2021.

Groups	Septic patients (Diabetic) n (%)	Septic patients (Non-diabetic) n (%)	Control n (%)	*p* value
Age (Mean ± SD)	65.7 ± 10.9	63.6 ± 12.3	61.9 ± 8.8	0.632
Gender (female)	11 (40.7%)	7 (25.9%)	8 (29.6%)	0.479
Complaint at time of admission				
Fever	4 (14.8%)	5 (18.5%)		
Weakness	6 (22.3%)	5 (18.5%)		
Deterioration in general condition	11 (40.7%)	13 (48.2%)		
SOB	6 (22.2%)	4 (14.8%)		
Comorbid disease				
CAD	9 (33.3%)	9 (33.3%)		
Hypertension	6 (22.2%)	1 (3.7%)		
CVD	7 (25.9%)	10 (37.0%)		
Dementia	5 (18.5%)	1 (3.7%)		
COPD	0 (0.0%)	6 (22.2%)		
Comorbid disease (re-categorized)				
CAD	9 (33.3%)	9 (33.3%)		
CVD	7 (25.9%)	10 (37.0%)		0.096*
COPD	0 (0.0%)	6 (22.2%)		
Diagnosis				
Urosepsis	11 (40.7%)	12 (44.1%)		
Pneumosepsis	8 (29.6%)	10 (37.0%)		
Sepsis related to soft tissue infection	8 (29.7%)	5 (18.5%)		
Clinical Outcome				
Exitus	5 (18.5%)	3 (11.1%)		0.063

*SOB: Shortness of breath; CAD: Coronary artery disease; CVD: Cerebrovascular disease; COPD: Chronic obstructive pulmonary disease; *Fisher-Freeman_Halton Test; SD: standard deviation.*

5.6%. The mean blood lactate level of the diabetic group was 18.9 ± 9.1 mg/dL, whereas this was 14.4 ± 4.2 mg/dL in the non-DM group. Lactate levels were significantly higher in diabetic group than non-diabetic group (*p*: 0.041). For glucose, C-reactive protein (CRP) and urea levels, there was a significantly higher difference among the groups as shown in Table 2. The groups did not differ statistically significant regarding other laboratory parameters. No correlation was observed between the qSOFA-SOFA scores and the asprosin levels.

The mean serum asprosin levels in the control group were 4.1 ± 1.4 ng/mL, 32.8 ± 7 ng/mL in the non-diabetic group and 90.7 ± 5.5 ng/min the diabetic group. Although the levels of asprosin in the non-DM group were significantly higher than the control group (*p*: 0.012), however, asprosin levels were statistically significantly higher in the diabetic group compared to the other two groups ($p < 0.001$).

4. Discussion

The main aim of the study was to evaluate the levels of asprosin which was significantly higher in diabetic and non-diabetic sepsis group than the control group. Asprosin levels were also higher in the group with diabetes, compared to non-diabetic group (mean values were 90.7 ± 5.5 ng/mL, 32.8 ± 7 ng/mL, respectively). Asprosin levels were statistically significantly higher in diabetic group compared to non-diabetic group ($p < 0.001$). The CRP levels above 100 mg/dL are associated with high mortality rate [8]. The CRP levels above 80 mg/dL and fever above 38.2 °C have a sensitivity between 67.6% and 93.4% and specificity between 61.3% and 86.3% in the diagnosis of sepsis [9]. In the present study, mean CRP levels were 21.7 ± 7.9 mg/dL in patients with diabetic group and 18.3 ± 5.2 mg/dL in non-diabetic group. These values, which are significantly low compared to the literature, suggest that sepsis

TABLE 2. Vital signs and laboratory parameters of the groups.

Parameter	Normal Range	DM group (Mean ± SD)	Non-DM group (Mean ± SD)	*p*	Control Group
Fever (°C)	36.4–37.2	38.1 ± 0.4	38.0 ± 0.5	0.251	38.1 ± 0.4
Pulse (beat/min)	60–100	119.8 ± 16.5	121.0 ± 16.4	0.687	78.0 ± 14.0
Respiratory rate (breath/min)	12–20	22.0 ± 2.4	21.8 ± 3.6	0.400	14.0 ± 2.0
Systolic blood pressure (mmHg)	≤120	95.1 ± 11.2	92.1 ± 8.7	0.270	112.0 ± 6.0
Diastolic blood pressure (mmHg)	≤80	59.0 ± 11.2	55.0 ± 8.8	0.154	72.0 ± 4.0
pH	7.4–7.5	7.3 ± 0.2	7.3 ± 0.1	0.163	7.4 ± 0.6
Glucose (mg/dL)	70–100	269.9 ± 86.5	149.9 ± 39.1	<0.001	86.0 ± 8.0
Lactate (mg/dL)	≤4.5	18.9 ± 9.1	14.4 ± 4.2	0.041	1.1 ± 0.3
HbA1C (%)	≤5.7	7.8 ± 0.5	5.8 ± 0.3	0.013	5.2 ± 0.3
WBC ($\times 10^3/\mu L$)	4.5–11.0	14.3 ± 4.4	15.4 ± 3.5	0.341	6.4 ± 2.2
Hemoglobin (g/dL)	11.5–17.0	12.7 ± 2.2	13.1 ± 1.9	0.923	13.2 ± 1.4
Plateletes ($\times 10^3/\mu L$)	150–450	187.0 ± 58.0	169.0 ± 43.0	0.598	180.0 ± 24.0
Natrium (mEq/L)	135–145	135.1 ± 7.4	137.6 ± 6.5	0.182	139.0 ± 3.0
Potassium (mEq/L)	3.6–5.2	4.7 ± 0.6	4.3 ± 0.5	0.714	4.1 ± 0.3
Chlor (mEq/L)	96–106	98.2 ± 4.8	95.2 ± 6.0	0.046	98.0 ± 2.0
Urea (mg/dL)	5–20	57.8 ± 18.2	47.1 ± 10.5	0.012	7.2 ± 1.4
Creatinine (mEq/L)	0.6–1.3	1.5 ± 0.7	1.3 ± 0.5	0.225	0.7 ± 0.2
CRP (mg/L)	0.3–1	21.7 ± 7.9	18.3 ± 5.2	0.423	0.5 ± 0.1
GCS	15	12.2 ± 1.1	13.9 ± 1.3	0.265	15
qSOFA score	<2	2.4 ± 0.5	2.2 ± 0.5	0.910	0
SOFA score	0	13.2 ± 2.8	12.9 ± 2.6	0.653	0
Asprosin (ng/mL)	1.6–5.0	90.7 ± 5.5	32.8 ± 7.0	<0.001	4.1 ± 1.4

WBC: White blood cell; CRP: C-reactive protein; GCS: Glasgow coma scale; qSOFA: Quick sequential organ failure assessment; SOFA: Sequential organ failure assessment; DM: diabetes mellitus; SD: standard deviation.

may develop even in the absence of significant elevation of CRP levels in diabetic patients. The lactate and urea levels of patients with DM and non-DM were significantly different for two groups. In this respect, the high blood lactate and urea levels in diabetic patients may be a promising risk factor for development of sepsis.

Despite its high mortality and morbidity rates a widely accepted biomarker is still needed for the diagnosis of sepsis [5]. Sepsis-3 consensus report mentions the inadequacy of diagnostic biomarkers for sepsis [1]. Additionally, the high incident rate of sepsis (30%) in hospitalized patients is a great economic burden for any country [15]. Sepsis development is much easier and faster in patients with diabetes mellitus than in patients without diabetes mellitus [16]. Owing to the high morbidity and mortality rate of sepsis, especially in patients with preexisting diabetes, a delay in diagnosis can be riskier and more fatal. So, it is very important to explore a new or valid biomarker for the diagnosis of sepsis. The reason for no correlation between SOFA score and asprosin levels is that the qSOFA score is insufficient to diagnose sepsis, as discussed earlier. We think that the sofa score is more effective in determining the prognosis than in the diagnosis of sepsis.

In a previous study, septic patients with DM and no-DM were examined and the rate of sepsis in female patients with diabetes and non-diabetes was 44.8% and 48.2% respectively [8]. But in the present study these were found to be 40.7% and 25.9% respectively. This low rate of female patients in the non-DM group may be related to the other comorbid conditions. The mean age was 72.4 ± 16.8 years in diabetic sepsis group and 76.8 ± 10.9 years in non-diabetic group in previous studies as mentioned above. In present study, these rates were 65.7 ± 10.9 and 63.6 ± 12.3 years, respectively. This difference in the rates may be attributed to the irregularity of diabetes treatment.

Ma *et al.* [17], investigated that the most common source of sepsis was abdominal infections with a rate of 25.5%. However, the most common source of sepsis in this present study was urinary tract infections with a rate of 42.4%. This variation may be due to the underlying diabetic condition which is the major cause of renal dysfunction. In the same study, it was also investigated that hypertension is the most commonly comorbid condition in septic patients (47.4%) [18]. But in the present study, the most common comorbid condition was coronary artery disease with a rate of 33.3%. This condition may be attributed to high frequency of coronary artery disease in our Turkey. The mortality rate of septic ambulatory patients with DM and no-DM was found to be 35.4% and 35.7%, respectively [19]. However, in the present study, the mortality rate of diabetic septic and non-diabetic septic group

was 18.5%, and 11.1% respectively which was not statistically significant (p: 0.063).

Acara and his coworkers reported that serum asprosin levels were effective in determining the severity of acute coronary syndrome in patients with unstable angina pectroris [20]. In the present study, it was found that the levels of asprosin were close to the upper limit of the measurement kit in septic patients, especially in diabetic patients who have a severe inflammatory process.

Diao *et al.* [14], reported that plasma asprosin levels increased in patients with Type-2 DM regardless of fasting glucose and triglyceride levels. Although the mechanism of this increase has not been clearly explained, plasma asprosin levels increased in patients with insulin resistance [21]. In the present study, serum asprosin level in diabetic septic group is an important factor to determine the underlying insulin resistance as compared non-diarrheal septic. Further the underlying insulin resistance may be effectively detected by high serum asprosin levels in patients with DM compared to the group without DM. Wang *et al.* [13] reported that plasma asprosin levels were associated with glucose dysregulation and insulin resistance. This mechanism is supposed to be effective to report an inflammatory process that plays a pivotal role in the pathology of DM [22, 23]. Therefore, the severe inflammatory process in sepsis may be detected by high serum asprosin as shown in the present study. Despite all these interesting results, there was no significant difference in serum asprosin levels between the diabetic and non-diabetic groups in terms of mortality. This may be due to other factors like co-morbid diseases, age, source of sepsis, *etc*. which affect the mortality rate in patients with sepsis.

The current study has some limitations. The study was performed in a single center, and the number of patients was limited. Fat tissue and body mass index, which were reported to be effective in the secretion of asprosin, were not measured in our study.

5. Conclusions

In conclusion, serum asprosin levels were significantly elevated in patients with sepsis. This elevation was much higher in diabetic septic patients than non-diabetic septic patients. Further the serum asprosin levels might be beneficial in the diagnosis of sepsis when evaluated together with the other criteria used in the diagnosis of sepsis, especially in diabetic patients.

AVAILABILITY OF DATA AND MATERIALS

The data used to support the findings of this study are available from the corresponding author upon request.

AUTHOR CONTRIBUTIONS

SAS—designed, collected and coordinated the data collection, interpreted the data, wrote and edited the manuscript; ÖS—supervised the study, edited and critically reviewed the manuscript for important intellectual content; NA—critically reviewed the manuscript for important intellectual content, and approved of the final version to be published; NF—performed the laboratory analysis of the blood samples and interpreted the results; NS—performed the biostatistical analysis of the results and interpreted them. All authors read and approved the final manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

This study was approved by the Trakya University Medical Faculty Scientific Researches Ethical Committee, Reference Number: TUTF-BAEK 2018/334. Informed consent forms were obtained from all participants.

ACKNOWLEDGMENT

Not applicable.

FUNDING

This research received no external funding.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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How to cite this article: Semra Aytürk Salt, Nurettin Aydoğdu, Ömer Salt, Nihayet Fırat, Necdet Süt. The role of Asprosin in the diagnosis of diabetic and non-diabetic patients with sepsis. Signa Vitae. 2024; 20(5): 40-45. doi: 10.22514/sv.2024.056.