Submitted: 20 February, 2024 Accepted: 10 May, 2024 Published: 08 December, 2024 DOI:10.22514/sv.2024.159

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Signa Vitae

ORIGINAL RESEARCH

Diagnostic value of asprosin for sepsis: a case-control study in the emergency department

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Abstract

Asprosin is a polypeptide with versatile metabolic roles, peripherally and centrally, secreted mainly by white adipose tissue during fasting. High asprosin levels may be a risk factor for conditions such as obesity, diabetic nephropathy, and retinopathy and are positively associated with inflammatory markers. Our study aimed to investigate the predictive value of asprosin in diagnosing sepsis. The prospective case-controlled study included 80 individuals with a 1:1 ratio of sepsis and healthy individuals. Receiver operating characteristic curve (ROC) analysis was used to evaluate the diagnostic impact of asprosin on sepsis patients. The sepsis patients were 24 women and 16 men, with a median age of 73 years (Interquartile Range (IQR) 63–79). The control group included 40 healthy individuals, 24 women and 16 men, with a median age of 73 (IQR 63–79). While the asprosin level of the control group was 0.29 ng/mL, that of the sepsis group was 0.77 ng/mL ($p < 0.001$). When the cut-off value was accepted as 0.452, the sensitivity and specificity of asprosin were 85% and 80%. The area under the curve (AUC) of asprosin was found to be 0.904 (95% Confidence Interval (CI): 0.841–0.967, $p < 0.001$). In conclusion, higher asprosin levels were found in sepsis than in control patients, and asprosin had a high discriminatory ability diagnosis for sepsis.

Keywords

FBN1 protein; Human; Sepsis; Biomarkers

1. Introduction

According to the current international consensus 2016, sepsis is defined as life-threatening organ dysfunction resulting from impaired host response to infection [1]. According to the World Health Organization global report, it is known that 27% of patients diagnosed with sepsis and followed up in the hospital, and 42% of patients followed up in the intensive care unit (ICU) may die due to sepsis [2]. Sepsis diagnosis and prognosis rely on various scores in emergency departments (EDs) and ICUs. Early detection using biomarkers like procalcitonin, interleukin 6, C-reactive protein, and white blood cells is crucial. However, the lack of a sepsis-specific biomarker complicates prompt treatment initiation [3–5].

Asprosin is a peptide hormone first identified by Romere *et al.* [6] in 2016. It is a glycogenic and orexigenic adipokine secreted from white adipose tissue in response to starvation. Asprosin is part of profibrillin encoded by the Fbn1 gene. Asprosin is encoded by the 65th and 66th exons of the Fbn1 gene and is a total polypeptide containing 140 amino acids. It is vital in regulating glucose and lipid metabolism homeostasis through the cyclic adenosine monophosphate-protein kinase A (cAMP-PKA) pathway. The organs, such as the heart, liver, pancreas, stomach, skeletal muscles, lungs, and brain, also produce asprosin. Asprosin has been shown to cross the blood-brain barrier and has also been reported to be associated with obesity, diabetes, and cardiovascular disease [7–11].

In a mouse study, Asprosin was shown to cause inflammation, cellular dysfunction, and apoptosis in β cells. In another study, Asprosin stimulation significantly increased the expression and secretion of the pro-inflammatory cytokines tumor necrosis factor α, interleukin-1β (IL-1β), IL-8 and IL-12 *in vitro* [12, 13].

In this study, we aimed to evaluate the level of asprosin in patients diagnosed with sepsis and to prospectively evaluate whether asprosin can be used as a new biomarker in diagnosing sepsis.

2. Material and methods

2.1 Study design

This study was conducted with local ethical approval on patients who applied to Erciyes University Faculty of Medicine Emergency Service and were diagnosed with sepsis. Patient selection and sample collection were done in the emergency department, and the samples were stored and analyzed in the medical biochemistry laboratory.

2.2 Participants

The age group was determined as 18–80, and those with a body mass index of less than 30 kg/m^2 were included in the study. Patients with advanced liver and kidney failure, end-stage cancer, recent major surgery and resuscitation, a severe trauma history, Human immunodeficiency virus (HIV) positivity, and absolute neutropenia were not included in the study. The control group was selected from the relatives of the patients who applied to the emergency department and healthy individuals who gave verbal and written consent by age and gender. Quick sequential organ failure assessment (qSOFA) score was used to screen patients with suspected sepsis. qSOFA score is scored according to (a) Glasgow Coma Scale $<$15, (b) systolic blood pressure $\leq$100 mmHg, and (c) respiratory rate $\geq$22/min, and patients with a score of 2 or more were considered at high risk for mortality [14]. Informed consent was obtained from patients diagnosed with sepsis. Twenty-eight patients were excluded from the study because they did not give consent. An Infectious Diseases and Clinical Microbiology specialist diagnosed sepsis according to the Sepsis 3 (2016) diagnostic criteria [14]. Patients with growth in blood and urine cultures were considered to have proven infection. Patients who required vasopressors for mean arterial pressure to be 65 mmHg or more and whose serum lactate level was above two mmol/L in the absence of hypovolemia were considered to have septic shock. qSOFA score and Systemic inflammatory response syndrome (SIRS) criteria were calculated for each patient diagnosed with sepsis. White blood count (WBC), C-reactive protein (CRP), procalcitonin, arterial blood gas, and routine biochemistry parameter tests were also performed on the patients.

2.3 Analysis of asprosin

In addition to routine complete blood count, CRP, procalcitonin, blood gas, and biochemistry tests, a 5 cc-blood sample was taken from each individual participating in the study into an aprotinin tube (which is a reliable option in polypeptide and enzyme analysis thanks to the anticoagulant and proteolytic inhibitor it contains) to measure blood asprosin level. The blood samples were centrifuged in a Nüve brand NF400 model centrifuge device at 4000 rpm for 10 minutes; their plasma was separated and placed in Eppendorf tubes and stored at −80 °C until the working day. Asprosin levels were measured using the ELISA (Enzyme-linked Immunosorbent Assay) kit offered by Cloud-Clone Corp (CCC, Katy, TX, USA, catalog no. CEA332Hu). The working range of the kit was 0.156–10 ng/mL (Intra-Assay: Coefficient of Variability (CV) $<$10%, Inter-Assay: CV $<$12%).

2.4 Statistical analysis

The histogram and q-q plots were evaluated, and the Shapiro-Wilk test was used to assess the normality of the data. Levene's test was used to test the homogeneity of variance. A two-sided independent samples *t*-test or Mann-Whitney U test was used to compare the distributions of continuous variables between patients and control groups. Pearson chi-square test was used to compare differences in categorical variables. Values are frequencies and percentages, mean and standard deviation, or median and 25th–75th. They were shown as percentages. Receiver operating characteristic curve (ROC) analysis was used to evaluate the diagnostic impact of asprosin on sepsis patients. The area under the ROC curve was calculated with a 95% confidence interval. The Youden index was used to determine the optimal cut-off value. Additionally, sensitivity, specificity, positive and negative predictive values, and positive and negative likelihood ratio statistics were calculated with 95% confidence intervals according to the determined cut-off values. Analyzes were performed using R 4.0.1 (www.r-project.org). A *p*-value below 5% was considered statistically significant.

3. Results

The study was conducted on 40 sepsis patients, 24 women, and 16 men, with a median age of 73 (IQR 63–79). The control group included 40 healthy individuals, 24 women and 16 men, with a median age of 73 (IQR 63–79). The median body mass index (BMI) value of patients was 24.3 kg/m^2 (IQR = 5.2). Albumin levels of sepsis patients measured within the first 24 hours of admission were recorded. The mean albumin level value was 3.01 $\pm$ 0.6 g/L (min: 1.7, max: 4.57). The clinical and demographic characteristics of the patients are shown in Table 1.

Asprosin level, heart rate, and respiration rate were significantly higher in patients with sepsis than in controls, and hemoglobin, platelet, Glasgow coma scale (GCS), and chloride parameters were significantly lower in patients with sepsis than in controls ($p < 0.001$). There were also significant differences in sodium, white blood cell count (WBC), blood urea nitrogen (BUN), and creatinine values between patients with sepsis and control subjects. Sodium (134.73 $\pm$ 5.56 *vs.* 137.89 $\pm$ 2.65, $p = 0.002$), WBC (10.93 (7.03–15.32) *vs.* 8.49 (7.36–9.45), $p = 0.033$); BUN (25.45 (16.55–52.48) *vs.* 17.95 (13.43–22.43), $p = 0.001$), creatinine (1.17 (0.92–2.04) *vs.* 0.84 (0.66–1.25), $p = 0.002$). There was no significant difference between the sepsis and control groups in terms of gender, age, systolic blood pressure, diastolic blood pressure, potassium, body temperature, aspartate aminotransferase and alanine aminotransferase ($p > 0.05$) (Table 2). The distribution of asprosin for the patient and control group is shown in Fig. 1.

In the ROC analysis, the decisive role of asprosin in determining the risk of sepsis was measured. When the cut-off value was accepted as 0.452 ng/mL, the sensitivity and specificity of asprosin were 85% and 80%. The area under the curve (AUC) of asprosin was found to be 0.904 (95% CI: 0.841–0.967, $p <$ 0.001) (Fig. 2). Other diagnostic measurements in the ROC analysis are shown in Table 3.

4. Discussion

Our study investigated the predictive value of asprosin in diagnosing sepsis. Asprosin level was statistically significantly higher in the sepsis group compared to the control group ($p <$ 0.001). While the asprosin level of the control group was 0.29 ng/mL, that of the sepsis group was 0.77 ng/mL. We found that asprosin had a statistically significant and high level (AUC =

TABLE 1. The characteristics of the patient group.

Variable	Statistics (n = 40) n (%)
Female	24 (60)
Age, yr, median (IQR)	73 (63–79)
Source of infection	
Respiratory	23 (57.5)
Urinary	7 (17.5)
Other*	10 (25.0)
Comorbidity	
Hypertension	18 (45.0)
Diabetes	11 (27.5)
COPD	3 (7.5)
Cerebrovascular Disease	5 (12.5)
CHF	5 (12.5)
CKD	4 (10.0)
CAD	4 (10.0)
SIRS	
2	20 (50.0)
3	18 (45.0)
4	2 (5.0)
qSOFA**	
0	3 (7.5)
1	10 (25.0)
2	24 (60.0)
3	3 (7.5)
SOFA score, median (IQR)	4.5 (3.0)
APACHE II score, median (IQR)	29 (6.5)
Mechanical ventilation support	4 (10.0)
Vasopressor support	11 (27.5)

Note: Values are expressed as n (%), mean ± SD, or median (1st–3rd quartiles). COPD: Chronic Obstructive Pulmonary Disease; CAD: Coronary Artery Disease; CHF: Congestive Heart Failure; CKD: Chronic Kidney Disease; IQR: Inter Quantile Range; SIRS: Systemic Inflammatory Response Syndrome; SOFA: Sequential Organ Failure Assessment; APACHE: Acute Physiologic Assessment and Chronic Health Evaluation.
**: Cellulitis, wound infection.*
***: The qSOFA score was calculated using the values at the time of visiting the ED.*

0.904) discriminatory ability in predicting sepsis ($p < 0.001$). When the cut-off value of asprosin was >0.452, its sensitivity was 85%, and specificity was 80%.

Although the clinical picture is sometimes evident in patients with sepsis, the findings may not be apparent, especially in the early stages of sepsis. Although fever, hypotension, and tachycardia are primarily expected in sepsis patients, they may be common symptoms and signs of many diseases. In our study, consistent with the literature, pulse and respiratory rates were significantly higher than those in the control group [15]. Although systolic and diastolic blood pressures were lower in the patient group, we did not detect any statistical difference. We used the Glasgow coma scale to evaluate the patients' consciousness status, and the consciousness status in sepsis patients was statistically significantly lower than in the control group.

No pathognomonic biomarker has yet been identified as a laboratory finding in sepsis. Complete blood count, serum electrolytes, kidney function tests, liver function tests, CRP, procalcitonin, *etc.*, are used to evaluate the patient's general hematological and metabolic status and to make a differential diagnosis. Tests are requested, but no single test is considered specific for sepsis [16].

Thrombocytopenia in sepsis patients, which is experienced

TABLE 2. Comparison of several parameters in patients and controls.

Variable	Group		Total Mean ± SD	*p*
	Control (n = 40) Mean ± SD	Patient (n = 40) Mean ± SD		
Female, n (%)	24 (60)	24 (60)	80 (100)	0.999
Pulse beats/minute	82.18 ± 8.33	106.93 ± 19.98	94.55 ± 19.66	<**0.001**
Systolic BP, mmHg	119.08 ± 7.59	117.20 ± 29.97	118.14 ± 21.74	0.703
Diastolic BP, mmHg	68.05 ± 6.95	63.13 ± 17.53	65.59 ± 13.48	0.105
Hemoglobin (g/dL)	13.23 ± 1.47	11.08 ± 3.32	12.15 ± 2.77	<**0.001**
Platelet, $\times 10^3/\mu L$	297.25 ± 45.28	187.85 ± 107.82	242.55 ± 98.90	<**0.001**
Sodium, mmol/L	137.89 ± 2.65	134.73 ± 5.56	136.31 ± 4.61	**0.002**
Potassium, mmol/L	4.28 ± 0.46	4.23 ± 1.05	4.26 ± 0.80	0.802
Respiration rate	16.13 ± 2.64	26.03 ± 5.11	21.08 ± 6.41	<**0.001**
	Control (n = 40) Median (IQR)	Patient (n = 40) Median (IQR)	Total Median (IQR)	
Age, yr	73 (63–79)	73 (63–79)	73 (63–79)	0.999
Asprosin, ng/mL	0.29 (0.15–0.44)	0.77 (0.53–0.98)	0.46 (0.29–0.80)	<**0.001**
Fever, °C	36.55 (36.40–36.70)	36.45 (36.00–37.18)	36.50 (36.00–36.80)	0.560
GCS	15 (14.99–15)	14 (13.00–15)	15 (14.00–15)	<**0.001**
WBC, $\times 10^3/\mu L$	8.49 (7.36–9.45)	10.93 (7.03–15.32)	8.80 (7.27–12.33)	**0.033**
BUN, mg/dL	17.95 (13.43–22.43)	25.45 (16.50–52.48)	20.00 (15.03–28.43)	**0.001**
Creatine, mg/dL	0.84 (0.66–1.25)	1.17 (0.92–2.04)	0.96 (0.74–1.56)	**0.002**
AST, U/L	26.40 (24.08–29.22)	41.75 (20.50–75.65)	26.90 (23.40–43.08)	0.099
ALT, U/L	26.50 (23.18–34.1)	21.15 (13.20–37.80)	26.35 (20.40–34.9)	0.107
Chloride, mmol/L	103.5 (100.7–106.10)	99.0 (95.2–102.75)	101.2 (97.6–105.30)	<**0.001**

Note: Values are expressed as n (%), mean ± SD, or median (1st–3rd quartiles), and significant values are indicated in bold. GCS: Glasgow Coma Scale; WBC: White Blood Cell; BUN: Blood Urea Nitrogen; AST: Aspartate Transferase; ALT: Alanine Amino Transferase; IQR: Interquartile range; BP: Blood Pressure; SD: Standard Deviation; IQR: Inter Quantile Range.

by 30% of sepsis patients and linked to poor prognosis, is believed to stem from intravascular coagulation [17]. Our study showed a statistically significant decrease in platelet count between sepsis patients and the control group. Furthermore, a statistically significant reduction in hemoglobin compared to the control group was revealed. This may be attributed to intravascular coagulation, but more studies with more patients are needed. Additionally, there was a statistically significant difference between the patient and control groups regarding high BUN and low creatinine. This can be attributed to the kidney damage that can be observed in sepsis.

Asprosin is a hormone activated during fasting and secreted from adipose tissue. It regulates glucose homeostasis by increasing hepatic glucose release via the G protein-cyclic adenosine monophosphate-protein kinase pathway. When circulating asprosin is reduced, glucose control is impaired, and proinflammatory cytokines increase, which may contribute to developing inflammatory diseases. These findings highlight the potential anti-inflammatory effects of asprosin and may contribute to wound healing by playing a role in the regulation of hepatic glucose production and glucose homeostasis [18]. A study on patients with familial Mediterranean fever (FMF) showed that serum asprosin levels of FMF patients with acute attacks were lower than those in attack-free periods and healthy controls [19]. On the other hand, asprosin has been shown to induce a pro-inflammatory response in THP-1 macrophages by significantly promoting the expression and secretion of essential pro-inflammatory mediators [13]. In our study, patients with sepsis had higher asprosin levels than the control group. There is little information about asprosin inducing inflammation and causing tissue damage by activating pro-inflammatory mechanisms. Based on data from animal studies, it has been argued that asprosin, a biomolecule released from the fat tissue of obese individuals, is an immunomodulator that increases inflammation [20].

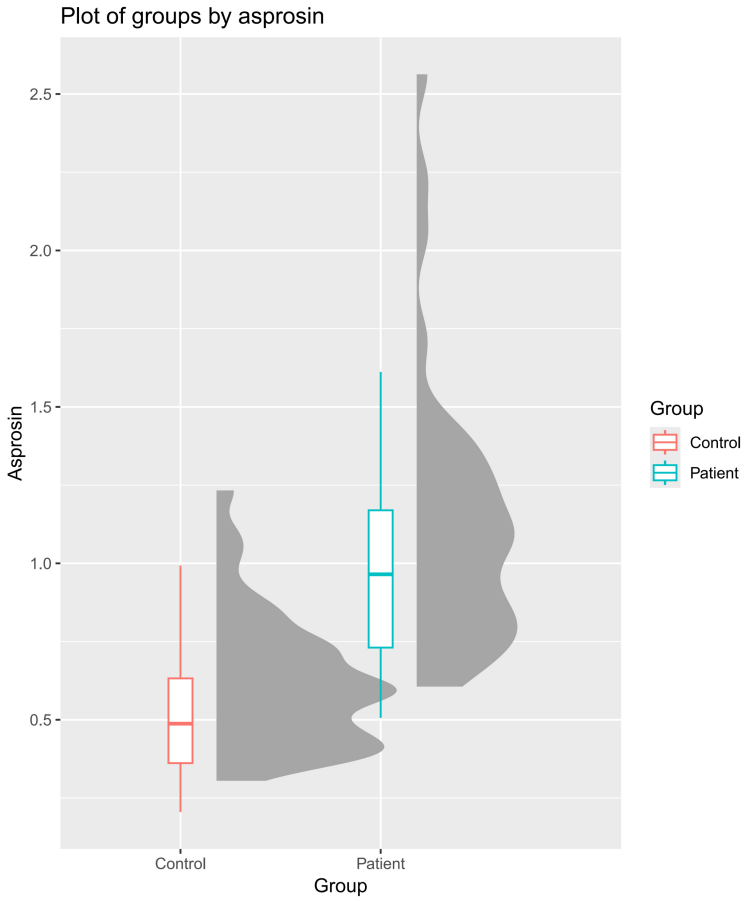

FIGURE 1. The distribution of Asprosin's level (ng/mL) for patient and control groups.

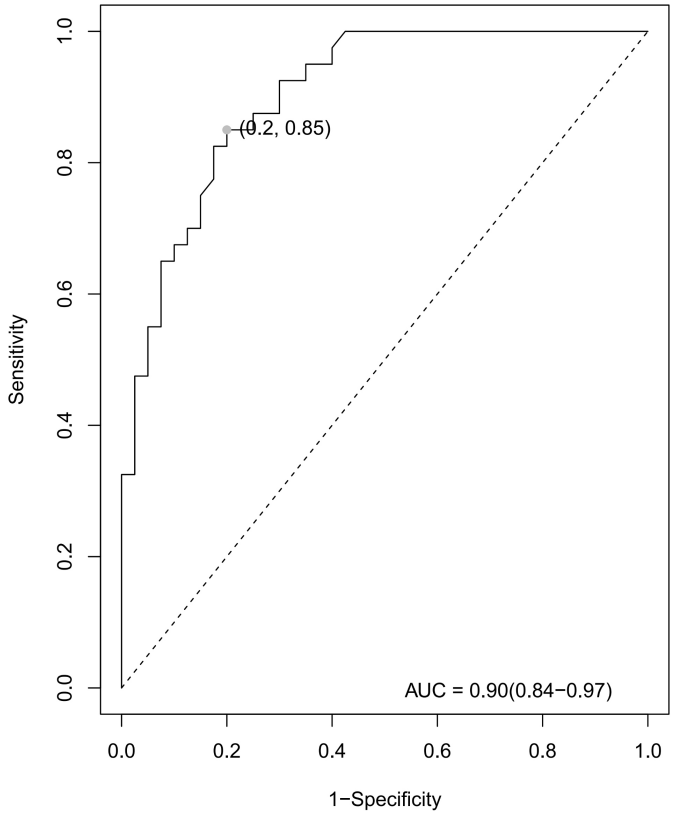

FIGURE 2. ROC curve of Asprosin (ng/mL). AUC: area under the curve.

TABLE 3. Statistical diagnostic measures calculated for asprosin.

Diagnostic measures	Asprosin (>0.452)
ROC curve statistics	
AUC (95% CI)	0.904 (0.841–0.967)
p	**<0.001**
Diagnostic statistics	
SEN (95% CI)	0.850 (0.739–0.961)
SPE (95% CI)	0.800 (0.676–0.924)
PPV (95% CI)	0.810 (0.691–0.928)
NPV (95% CI)	0.842 (0.726–0.958)
PLR (95% CI)	4.250 (2.256–8.006)
NLR (95% CI)	0.188 (0.088–0.398)

Note: ROC: Receiver Operating Characteristics; AUC: Area Under the Curve; SEN: Sensitivity; SPE: Specificity; PPV: Positive Predictive Value; NPV: Negative Predictive Value; PLR: Positive Likelihood Ratio; NLR: Negative Likelihood Ratio; CI: Confidence Interval. (Significant values are indicated in bold).

5. Limitations

The main limitations of our study are the relatively low number of patients, the asprosin level not being measured again after follow-up and treatment, and the type of microbiological agent not being included in the study.

6. Conclusions

Many markers have been studied in the literature for sepsis diagnosis, and no clear conclusion has been reached [21]. For this reason, we decided to use asprosin to diagnose sepsis. We found a statistically significant difference between the patient and control groups. When we performed ROC analysis, we found the sensitivity and specificity of asprosin to be 85% and 80%. We think that measuring asprosin levels in sepsis may be helpful in diagnosis.

AVAILABILITY OF DATA AND MATERIALS

The data presented in this study are available on reasonable request from the corresponding author.

AUTHOR CONTRIBUTIONS

EB and MAG—designed the research study. EB and AC—performed the research. İT, ZTY and SM—provided help and advice on manuscript preparation. AC and GZ—analyzed the data. EB, İT and SM—wrote the manuscript. All authors contributed to editorial changes in the manuscript. All authors read and approved the final manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

This study was conducted with local ethical approval from Erciyes University Clinical Research Ethics Board (Approval no: 2023/17, Approval date: 04 January 2023). For all patients participating in our study, written and verbal voluntary consent forms were obtained from themselves or their legal guardians.

ACKNOWLEDGMENT

We would like to thank the Proofreading & Editing Office of the Dean for Research at Erciyes University for the copyediting and proofreading service for this manuscript.

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: Emre Bülbül, İbrahim Toker, Ahu Cephe, Muhammed Ali Gençaslan, Zeynep Türe Yüce, Sabahattin Muhtaroğlu, *et al*. Diagnostic value of asprosin for sepsis: a case-control study in the emergency department. Signa Vitae. 2024; 20(12): 86-92. doi: 10.22514/sv.2024.159.