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# Investigation of the Relationship Between Serum Subfatin Levels and Clinical Outcome in Patients with Transient Ischemic Attack in the Emergency Department

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## Abstract

**Aim:** We hypothesized that lower serum subfatin levels at admission might be associated with a higher risk of subsequent stroke in transient ischemic attack (TIA) patients. Therefore, this study aimed to examine the relationship between baseline subfatin levels and the occurrence of new cerebrovascular events at 28 and 90 days.

**Materials and Methods:** Patients who were admitted to the emergency department and diagnosed with TIA between 01.04.2022 and 31.03.2023 were studied prospectively.

**Results:** A total of 141 volunteers (71 patients and 70 controls) who met the criteria were included in the study. The median subfatin level of the patients was 1.51 [interquartile range (IQR): 25-75: 1.27-1.71] and the median subfatin level of the control group was 1.62 (IQR: 25-75: 1.13-2.32). There was a statistically significant association between coronary artery disease (CAD) and stroke development at 28 and 90 days [($p<0.05$), ($p<0.05$)]. Median subfatin levels were numerically lower in patients who experienced stroke within 28 and 90 days compared to those without subsequent stroke; however, these differences did not reach statistical significance ($p>0.05$). Therefore, no definitive conclusion can be drawn regarding the prognostic role of subfatin in this cohort.

**Conclusion:** CAD was significantly associated with stroke occurrence at both 28 and 90 days, underscoring its role as a major predictor of adverse outcome in TIA patients. We also found that although subfatin levels were lower in patients who had a stroke within 28 and 90 days compared to patients who did not have a stroke, there was no statistically significant difference between them.

**Keywords:** Emergency department, transient ischemic attack, subfatin, stroke

## Introduction

Transient ischemic attack (TIA) is defined as a clinical picture of sudden onset and neurological deficits resulting from a transient decrease in blood flow to a specific area of the brain. It usually resolves spontaneously within 24 hours and is distinguished from stroke by these characteristics (1). However, the risk of stroke is significantly higher in individuals with TIA compared to the general population. The risk of stroke within the first 48 hours of TIA varies between 10-15% and this rate increases even more in the first week (2). Therefore, early evaluation, risk assessment and clinical follow-up of patients

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with TIA is critical to prevent more serious complications such as stroke (3).

In recent years, inflammation, oxidative stress and vascular dysfunction have been recognized to play important roles in the pathophysiology of TIA and stroke. In this context, the effects of adipokines (adipose tissue-derived hormones) on the central nervous and cardiovascular systems are of increasing interest (4). Adipokines are involved in various biological processes associated with cardiovascular diseases, obesity and metabolic syndrome. One adipokine, subfatin (C1q/tumor necrosis factor-related protein 13), is involved in many metabolic processes such as energy homeostasis, inflammation and insulin sensitivity. It has also been reported that subfatin levels may be associated with atherosclerosis and cardiovascular diseases (5). However, the relationship between subfatin levels and risk factors for TIA and stroke has not been fully elucidated and research on this subject is limited.

Investigating the possible effects of subfatin on the process of brain ischemia may contribute to the prediction of stroke risk in patients with TIA. It is thought that subfatin levels may affect neuroinflammation, oxidative stress, and endothelial dysfunction, processes that play critical roles in ischemic events (6). Therefore, examining the association of biomarkers such as subfatin with stroke risk may contribute to the identification of individuals at high risk after TIA and the development of strategies that can guide the management of these patients. This study aimed to investigate the relationship between subfatin levels and the clinical outcome of patients with TIA.

## Materials and Methods

### Place, Time and Type of Study

This study was conducted in the emergency department of University of Health Sciences Türkiye, Bursa Yüksek İhtisas Training and Research Hospital with the approval of the clinical research ethics committee with the (desicion number: 2011-KAEK-25 2022/03-08, date: 23.1.2011), patients with a final emergency department diagnosis of TIA were prospectively analyzed.

### Inclusion and Exclusion Criteria

### Inclusion Criteria

Patient group;

1. Patients over 18 years of age,
2. Patients with normal radiologic imaging results,
3. Patients who gave consent to the study by themselves or their relatives were included.

In the control group, patients over 40 years of age, who had no comorbidities, and gave consent to the study, were included.

### Exclusion Criteria in the Study

Patient group;

1. Patients younger than 18 years of age,
2. Patients who did not give consent to the study,
3. Patients with infarction detected on radiologic imaging,
4. Patients with bleeding detected on radiologic imaging,
5. Patients with a mass detected on radiologic imaging,
6. Pregnant patients,
7. Those with previous cerebrovascular disease were not included in the study.

In the control group, those under 40 years of age, pregnant women, and those with any comorbidities were excluded.

### Work Plan

A standardized study data entry form was created for the data of the patients included in the study. Demographic information (age, gender), height, weight, body mass index, date of presentation to the emergency department (ED), vital signs (fever, respiratory rate in minutes, fingertip oxygen saturation in room air and with oxygen supplementation, systolic blood pressure), data such as diastolic blood pressure, complaints at admission, chronic diseases, medications and radiologic imaging, and the patient's outcome in the ED (discharge, admission, intensive care unit admission, excitus, treatment refusal) were recorded. In addition, patients or their relatives were contacted on days 28 and 90 and asked whether they had a new stroke attack. After the study was completed, the data in the study forms obtained were saved in electronic format for statistical analysis.

### Laboratory Studies

Complete blood count, serum electrolytes (Na, Cl), renal function tests (blood urea nitrogen and creatinine), cardiac troponin, international normalized ratio levels were obtained from the patients participating in the study. To study METRNL (subfatin) level, 3 mL blood samples were collected into aprotinin tube (tube containing 500 KIU aprotinin to protect blood pretolysis).

Blood samples were allowed to clot for 30 minutes and then centrifuged at 3000 rpm for 10 minutes at room temperature. The serum obtained was portioned and stored at -80 degrees Celsius for subfatin level studies.

Human METRNL levels were determined by ELISA method in the biochemistry laboratory of S University of Health Sciences Türkiye, Bursa Yüksek İhtisas Training and Research Hospital. The ELISA kit used was Shanghai Biotechnology Co. Brand, produced in Yunnan, People's Republic of China, and the unit for Human METRNL is ng/mL. Baoshan, Romer, ChroMate 4300 ELISA reader, manufactured in Getzdersof /Austria, was used. ELISA method was performed according to the kit procedure as follows:

- Standards were prepared as recommended in the procedure. For this study, double standards and double blinds were used in each kit.
- 50 µL of the blind and standards were added to the appropriate wells. Then 40 µL of sample sera were added to the appropriate wells.
- Then 10 µL of biotin-METRNL antibody was added to the sample wells.
- 50 µL of Str-HRP-Conjugate Reagent was added to the standard and sample wells. No HRP solution was added to the blinds. The wells were then covered with a cover plate, shaken gently and incubated in an oven at 37 °C for 60 min.
- After removing from the oven, the sample was emptied. Then, 350 µL of 30 times diluted washing solution was added to each well and washed 5 times.
- After washing, 50 µL of cromogen A solution was added to each well. Then 50 µL of choromogen B solution was added to each well in dim light. The wells were covered with a cover plate, shaken gently and incubated in the dark at 37 °C for 10 min in an oven.
- The reaction was terminated by adding 50 µL of stop solution to each well. Measurement was performed at 450 nm on a ChroMate-4300 microplate reader.

### Statistical Analysis

IBM SPSS Statistics for Windows, Version 21.0 (IBM Corp. Armonk, NY: USA. Released 2012) package program was used for statistical analyses. Descriptive statistics were expressed as mean ± standard deviation, median and range and/or interquartile range (IQR) for numerical variables, while categorical variables were expressed as number of cases and (%). Kolmogorov-Smirnov test was used for normality distribution of the data. Levene's test was used to determine whether the assumption of homogeneity of variances was met. The significance of the difference between the groups in terms of continuous numerical variables for which parametric test statistical assumptions were met was examined by Student's t-test. Significance of the difference in terms of continuous numerical variables for which parametric test statistical assumptions were not met was evaluated by Mann-Whitney U test. Kruskal-Wallis test was used for comparisons of three or more groups. Pearson correlation analysis was used to evaluate the relationships between parametric distributed data and Spearman's correlation analysis was used to evaluate the relationships between non-parametric distributed data. Fisher's exact test was used to analyze whether there was a relationship between categorical variables. $P<0.05$ was considered statistically significant. Results were given at 95% confidence interval.

## Results

A total of 141 volunteers, including 71 patients and 70 controls, who met inclusion criteria were included in the study. The mean age of the patient group was 64.15±13.11 years and the mean age of the control group was 50.13±11.20 years. Forty-one (57.7%) of the patient group and 50 (71.4%) of the control group were male. It was found that 58 (81.7%) of the patients had comorbidities and the most common comorbidities were hypertension (n=35, 49.3%) and diabetes mellitus (n=26, 36.6%). Stroke developed within 28 days in 12 (16.9%) and within 90 days in 14 (19.7%) patients (Table 1).

The median subfatin level of the patients was 1.51 (IQR: 25-75: 1.27-1.71) and the median epicardial adipose tissue thickness was 0 mm (IQR: 25-75: 0-0.6), while the median subfatin level of the control group was 1.62 (IQR: 25-75: 1.13-2.32) and the median epicardial adipose tissue thickness was 0.3 mm (IQR: 25-75: 0.2-0.4).

While there was no statistically significant difference between the median subfatin levels of the patient group and the control group ($p>0.05$), the median epicardial adipose tissue thickness was found to be statistically significantly different ($p<0.001$) (Table 2).

In the analysis performed to determine the relationship between gender, comorbidities, medication use, electrocardiogram (ECG) findings, antiaggregant and anticoagulant use and stroke development in 28 days, a statistically significant relationship was found between coronary artery disease (CAD) and stroke development in 28 days ($p<0.05$). The stroke rate within 28 days was higher in patients with CAD (Table 3).

In the analysis performed to determine the relationship between gender, comorbidities, medication use, ECG findings, antiaggregant and anticoagulant use and stroke development in 90 days, a statistically significant relationship was found between CAD and stroke development in 90 days ($p=0.005$).

| Table 1. Clinical and demographic information | | |
|---|---|---|
| Patient group age (years)* | | 64.15±13.11 |
| Control group age (years)* | | 50.13±11.20 |
| Patient group gender# | Male | 41 (57.7) |
| | Female | 30 (42.3) |
| Control group gender# | Male | 50 (71.4) |
| | Female | 20 (28.6) |
| Comorbidities# | | 58 (81.7) |
| Comorbidities# | Hypertension | 35 (49.3) |
| | Diabetes mellitus | 26 (36.6) |
| | Coronary artery disease | 18 (25.4) |
| | Arrhytmia | 2 (2.8) |
| | Malignity | 2 (2.8) |
| | Valvular diease | 2 (2.8) |
| | Congestive heart failure | 1 (1.4) |
| | Asthma/COPD | 4 (5.6) |
| | Chronic kidney disease | 1 (1.4) |
| | Others | 16 (22.5) |
| Use of medication for comorbidity# | | 52 (73.2) |
| Antiaggregant drug use# | | 19 (26.8) |
| Anticoagulant drug use# | | 2 (2.8) |
| Electrocardiography findings # | Normal sinus rhythm | 52 (73.2) |
| | Atrial fibrillastion | 9 (12.7) |
| | Left bundle branch block | 6 (8.5) |
| | Others | 4 (5.6) |
| Emergency department outcome# | Admission | 67 (94.4) |
| | Others | 4 (5.6) |
| Stroke within 28 days | | 12 (16.9) |
| Stroke within 90 days | | 14 (19.7) |
| Total number of patients# | | 71 (100) |

#n (%), *Mean ± SD, COPD: Chronic obstructive pulmonary disease, SD: Standard deviation

The rate of stroke within 90 days was found to be higher in patients with CAD (Table 4).

In the analysis performed to investigate whether there was a difference between subfatin levels and stroke status of the patients within 28 and 90 days, no statistically significant difference was found between subfatin levels and stroke status of the patients within 28 and 90 days (Table 5).

## Discussion

Stroke is one of the leading causes of mortality and morbidity worldwide. TIA is a real neurological emergency due to the increased risk of stroke in patients after TIA. Many imaging methods and scoring systems are used to assess the risk of stroke after TIA. There are studies showing that these diagnostic methods are inadequate (2). In this study, we evaluated the effectiveness of serum subfatin level in determining the risk of stroke after TIA.

Hypertension is the most common comorbid disease in participants diagnosed with TIA. In our study, 81.7% of the patients had comorbid diseases and the most common comorbid disease was hypertension with a rate of 49.3%. In the study conducted by Wilson et al. (7) hypertension was found to be the most common comorbid disease with 73.5%. In a study by Kapral et al. (8) hypertension was found to be the most common comorbid disease with a rate of 66%. Our findings in this regard are consistent with other studies in the literature.

| Table 2. Clinical and laboratory data of participants | | | |
|---|---|---|---|
| **Variables** | **Patient group** | **Control group** | **p value** |
| Age, years[&] | 64.15±13.11 | 50.13±11.20 | **<0.001**[*] |
| Subfatin level[ε] | 1.51 (1.27-1.71) | 1.62 (1.13-2.32), | >0.05[#] |
| Height, cm[&] | 166.65±8.20 | 170.79±7.80 | **=0.002**[*] |
| Weight, kg[&] | 74.76±14.50 | 77.64±12.33 | >0.05[*] |
| BMI[&] | 26.85±4.33 | 26.55±4.37 | >0.05[*] |
| Fever, C[ε] | 36.2 (36.2-36.4) | 36.3 (36.2-36.5) | >0.05[#] |
| Pulse /mi [&] | 85.82±11.81 | 78.83±7.58 | **<0.001**[*] |
| SBP mm/Hg[&] | 145.48±22.55 | 127.30±6.20 | **<0.001**[*] |
| DBP mm/Hg[&] | 83.42±15.32 | 77.96±7.37 | **=0.008**[*] |
| Oxygen saturation[ε] | 97 (96-98) | 97 (96-98) | >0.05[#] |
| Respiratory rate /min[ε] | 14 (13-15) | 12 (12-13) | **<0.001**[#] |
| Epicardial adipose tissue thickness/mm | 0 (0-0.6) | 0.3 (0.2-0.4) | **<0.001**[#] |
| Left ventricle end – systolic diameter/cm | 3.68±0.70 | 2.80±0.28 | **<0.001**[*] |
| Left ventricle end – diastolic diameter/cm | 4.59±0.55 | 4.38±0.37 | **<0.05**[*] |
| Left atrium diameter/cm | 3.49±0.38 | 3.32±0.28 | **=0.003**[*] |
| Ascending aorta diameter/cm | 3.38±0.30 | 3.22±0.29 | **=0.002**[*] |
| Ejection fraction[ε] | 55 (50-60.0) | 60 (60-60) | **<0.001**[#] |
| $ABCD^2$ score[ε] | 4 (3-5) | | |
| Leukocyte count[&] | 8871.1±2649.9 | | |
| Neutrophil count[&] | 5649.0±2217.9 | | |
| Lymphocyte count[ε] | 2350 (1580-3140) | | |
| MPV[&] | 10.61±1.25 | | |
| Platelet count[&] | 244940±76574 | | |
| INR[ε] | 0.97 (0.89-1.08) | | |
| Glukose/mg/dL | 122 (103-143) | | |
| Sodium/mmol/L | 138.46±2.64 | | |
| Clor/mmol/L | 104,62 ±3.49 | | |
| BUN/mg/dL | 17 (13-23) | | |
| Kreatinin/U/L | 0.89 (0.68-1.07) | | |
| Troponin/ng/L | 4.80 (1.87-10.05) | | |

[ε]Median IQR (25-75), [&]Mean ± SD, [*]Student t-test, [#]Mann-Whitney U test, BMI: Body mass index, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, MPV: Mean platelet volume, BUN: Blood urea nitrogen, INR: International normalized ratio, SD: Standard deviation, IQR: Interquartile range

This may be due to the fact that hypertension disrupts the endothelial structure and leads to atherosclerosis formation.

In our study, there was a statistically significant association between CAD and stroke development at 28 and 90 days. In the study by Amarenco et al. (9) taking those without CAD as reference, the age- and sex-adjusted risk ratio of vascular events was 2.10 (0.63-6.96) for asymptomatic coronary stenosis <50%, 4.36 (1.35-14.12) for asymptomatic coronary stenosis ≥50% and 6.86 (2.15-21.31) for known CAD (9). Another study by Robinson et al. (10) also found an increased risk of stroke in patients with a history of CAD. The findings of our study are consistent with the literature.

In untreated TIAs, the risk of stroke at 3 months can reach up to 20%. Most of this risk occurs in the first 10 days, especially in the first 2 days (2,11,12). Observational data show that rapid clinical diagnosis and immediate preventive measures reduce the 3-month stroke risk by up to 80% (13). In a multicenter study in which patients with suspected TIA or mild stroke were rapidly triaged, evaluated and treated, the 3-month stroke risk was found to be approximately 5% (12). In some studies, the risk of

| Table 3. Fisher's exact test of variables with stroke within 28 days | | | | | | |
|---|---|---|---|---|---|---|
| **Variables** | | | **Stroke within 28 days** | | **Total** | **Fisher's exact test** |
| | | | **No** | **Yes** | | |
| Gender | Female | n (%) | 25 (83.3) | 5 (16.7) | 30 (100) | p>0.05 |
| | Male | n (%) | 34 (82.9) | 7 (17.1) | 41 (100) | |
| Accompanying disease | No | n (%) | 12 (92.3) | 1 (7.7) | 13 (100) | p>0.05 |
| | Yes | n (%) | 47 (81.0) | 11 (19.0) | 58 (100) | |
| Hypertension | No | n (%) | 30 (83.3) | 6 (16.7) | 36 (100) | p>0.05 |
| | Yes | n (%) | 29 (82.9) | 6 (16.1) | 35 (100) | |
| Coronary artery disease | No | n (%) | 47 (88.7) | 6 (11.3) | 53 (100) | p<0.05 |
| | Yes | n (%) | 12 (66.7) | 6 (33.3) | 18 (100) | |
| Diabetes mellitus | No | n (%) | 37 (82.2) | 8 (17.8) | 45 (100) | p>0.05 |
| | Yes | n (%) | 22 (84.6) | 4 (15.4) | 26 (100) | |
| Congestive heart failure | No | n (%) | 58 (82.9) | 12 (17.1) | 70 (100) | p>0.05 |
| | Yes | n (%) | 1 (100) | 0 | 1 (100) | |
| Arrhytmia | No | n (%) | 57 (82.6) | 12 (17.4) | 69 (100) | p>0.05 |
| | Yes | n (%) | 2 (100) | 0 | 2 (100) | |
| COPD/asthma | No | n (%) | 55 (82.1) | 12 (17.9) | 67 (100) | p>0.05 |
| | Yes | n (%) | 4 (100) | 0 | 4 (100) | |
| Malignity | No | n (%) | 57 (82.6) | 12 (17.4) | 69 (100) | p>0.05 |
| | Yes | n (%) | 2 (100) | 0 | 2 (100) | |
| Chronic renal failure | No | n (%) | 58 (82.9) | 12 (17.1) | 70 (100) | p>0.05 |
| | Yes | n (%) | 1 (100) | 0 | 1 (100) | |
| Valvular heart disease | No | n (%) | 57 (82.6) | 12 (17.4) | 69 (100) | p>0.05 |
| | Yes | n (%) | 2 (100) | 0 | 2 (100) | |
| Others | No | n (%) | 48 (87.3) | 7 (12.7) | 55 (100) | p>0.05 |
| | Yes | n (%) | 11 (68.8) | 5 (31.3) | 16 (100) | |
| Medication use | No | n (%) | 17 (89.5) | 2 (10.5) | 19 (100) | p>0.05 |
| | Yes | n (%) | 42 (80.8) | 10 (19.2) | 52 (100) | |
| Antiagregant | No | n (%) | 45 (86.5) | 7 (13.5) | 52 (100) | p>0.05 |
| | Yes | n (%) | 14 (73.7) | 5 (16.3) | 19 (100) | |
| Anticoagulant | No | n (%) | 57 (82.6) | 12 (17.4) | 69 (100) | p>0.05 |
| | Yes | n (%) | 2 (100) | 0 | 2 (100) | |
| Electrocardiography | Normal sinus rhythm | n (%) | 43 (82.7) | 9 (17.3) | 52 (100) | p>0.05 |
| | Atrial fibrillation | n (%) | 9 (100) | 0 | 9 (100) | |
| | Left bundle branch block | n (%) | 4 (66.7) | 2 (33.3) | 6 (100) | |
| | Others | n (%) | 3 (75.0) | 1 (25.0) | 4 (100) | |
| Total | | n (%) | 59 (83.1) | 12 (16.9) | 71 (100) | |

COPD: Chronic obstructive pulmonary disease

an acute ischemic stroke after TIA varies between 3.5%-10% in the first 2 days and 9.2%-17% in the first three months (14-17). In our study, the stroke rate was 16.9% within 28 days and 19.7% within 90 days. The reason why these rates were partially high may have been due to post-discharge treatment non-compliance and existing comorbidities in these patients.

Subfatin is an adipokine that has been discovered in recent years and is thought to play a role in various metabolic and inflammatory processes. This protein secreted by adipose tissue plays important roles in many disease processes such as obesity, diabetes and cardiovascular diseases. It has also been suggested that subfatin may be involved in vascular inflammation, endothelial dysfunction and atherosclerotic processes. In a study

| Table 4. Fisher's exact test of variables with stroke within 90 days | | | | | | |
|---|---|---|---|---|---|---|
| **Variables** | | | **Stroke within 90 days** | | **Total** | **Fisher's exact test** |
| | | | **No** | **Yes** | | |
| Gender | Women | n (%) | 24 (80.0) | 6 (20.0) | 30 (100) | p>0.05 |
| | Men | n (%) | 33 (80.5) | 8 (19.5) | 41 (100) | |
| Accompanying disease | No | n (%) | 12 (92.3) | 1 (7.7) | 13 (100) | p>0.05 |
| | Yes | n (%) | 45 (77.6) | 13 (22.4) | 58 (100) | |
| Hypertension | No | n (%) | 30 (83.3) | 6 (16.7) | 36 (100) | p>0.05 |
| | Yes | n (%) | 27 (77.1) | 8 (22.9) | 35 (100) | |
| Coronary artery disease | No | n (%) | 47 (88.7) | 6 (11.3) | 53 (100) | p<0.05 |
| | Yes | n (%) | 10 (55.6) | 8 (44.4) | 18 (100) | |
| Diabetes mellitus | No | n (%) | 36 (80.0) | 9 (20.0) | 45 (100) | p>0.05 |
| | Yes | n (%) | 21 (80.8) | 5 (19.2) | 26 (100) | |
| Congestive heart failure | No | n (%) | 56 (80.0) | 14 (20.0) | 70 (100) | p>0.05 |
| | Yes | n (%) | 1 (100) | 0 | 1 (100) | |
| Arrhytmia | No | n (%) | 55 (79.7) | 14 (20.3) | 69 (100) | p>0.05 |
| | Yes | n (%) | 2 (100) | 0 | 2 (100) | |
| COPD/asthma | No | n (%) | 53 (79.1) | 14 (20.9) | 67 (100) | p>0.05 |
| | Yes | n (%) | 4 (100) | 0 | 4 (100) | |
| Malignity | No | n (%) | 55 (79.7) | 14 (20.3) | 69 (100) | p>0.05 |
| | Yes | n (%) | 2 (100) | 0 | 2 (100) | |
| Chronic renal failure | No | n (%) | 56 (80.0) | 14 (20.0) | 70 (100) | p>0.05 |
| | Yes | n (%) | 1 (100) | 0 | 1 (100) | |
| Valvular heart disease | No | n (%) | 55 (79.7) | 14 (20.3) | 69 (100) | |
| | Yes | n (%) | 2 (100) | 0 | 2 (100) | p>0.05 |
| Others | No | n (%) | 47 (85.5) | 8 (14.5) | 55 (100) | p>0.05 |
| | Yes | n (%) | 10 (62.5) | 6 (37.5) | 16 (100) | |
| Medication use | No | n (%) | 17 (89.5) | 2 (10.5) | 19 (100) | p>0.05 |
| | Yes | n (%) | 40 (76.9) | 12 (23.1) | 52 (100) | |
| Antiagregant | No | n (%) | 44 (84.6) | 8 (15.4) | 52 (100) | p>0.05 |
| | Yes | n (%) | 13 (68.4) | 6 (31.6) | 19 (100) | |
| Anticoagulant | No | n (%) | 55 (79.7) | 14 (20.3) | 69 (100) | p>0.05 |
| | Yes | n (%) | 2 (100) | 0 | 2 (100) | |
| Electrocardiography | Normal sinus rhythm | n (%) | 42 (80.8) | 10 (19.2) | 52 (100) | p>0.05 |
| | Atrial fibrillation | n (%) | 8 (88.9) | 1 (11.1) | 9 (100) | |
| | Left bundle branch block | n (%) | 4 (66.7) | 2 (33.3) | 6 (100) | |
| | Others | n (%) | 3 (75.0) | 1 (25.0) | 4 (100) | |
| Total | | n (%) | 57 (80.3) | 14 (19.7) | 71 (100) | |

COPD: Chronic obstructive pulmonary disease

| Table 5. Analysis of the variables with subfatin levels | | | | |
|---|---|---|---|---|
| **Variables** | | **N** | **Subfatin level#** | **p value*** |
| Stroke within 28 days | No | 59 | 1.53 (1.23-1.73) | >0.05 |
| | Yes | 12 | 1.45 (1.33-1.58) | |
| Stroke within 90 days | No | 57 | 1.51 (1.22-1.72) | >0.05 |
| | Yes | 14 | 1.47 (1.38-1.61) | |

*Mann-Whitney U test, #Median interquartile range (25-75)

by Cavli et al. (18) investigating obesity/insulin resistance and subfatin levels, a significant relationship was found between low serum subfatin levels and obesity/insulin resistance. In another study by Köse et al. (19), the relationship between subfatin and preeclampsia was investigated and serum subfatin level was found to be significantly lower in preeclampsia patients compared to the control group. In the study by Demir et al. (20) investigating the relationship between ischemic stroke and serum subfatin level, no significant relationship was found. In the study by Yilmaz et al. (21) investigating subfatin levels in patients with acute coronary syndrome, subfatin levels were found to be lower in patients with non-ST elevation myocardial infarction and ST elevation myocardial infarction compared to the control group (21). In a study by Albayrak et al. (22), subfatin levels were found to be lower in patients with cerebral ischemia, intracerebral hemorrhage and subarachnoid hemorrhage compared to the control group.

In our study, although no significant difference was found between the plasma subfatin values of the patients and their stroke status on days 28 and 90, the mean serum subfatin values of the patients who had a stroke were lower than the mean serum subfatin values of the patients who did not have a stroke.

In conclusion, no significant relationship was found between serum subfatin levels and stroke in patients diagnosed with TIA in the ED. While a decrease in subfatin level poses a risk for conditions such as inflammation, endothelial damage and plaque formation, the lack of statistical difference in subfatin levels between the patient group and the control group in our study may be explained by the fact that no pathology was detected on imaging in the patient group diagnosed with TIA. The fact that serum subfatin levels were found to be low in clinical conditions such as obesity, insulin resistance and preeclampsia, which have been previously reported in the literature, suggests that this molecule may be used as a negative acute phase reactant in the future. More significant results may be obtained as the research on subfatin accumulates. Our study contributes to the literature as being the first study investigating the relationship between TIA and subfatin levels.

### Study Limitations

The single-center nature of our study restrained the number of patients included. In addition, the relatively short follow-up duration of the patients, such as stroke status on days 28 to 90, may have prevented a clear demonstration of the relationship between subfatin and TIA. As control group formed with healthy individuals without known comorbidities, mean age of the control group was significantly lower than that of patient group. This is also one of the limitations of our study.

## Conclusion

In our study, we found that CAD increased the risk of stroke in patients with TIA. We also observed that there was no significant difference between the subfatin levels of the patient and control groups. Finally, there was no significant difference between the subfatin levels measured at the time of admission in patients with TIA who had a stroke within 28 and 90 days and those who did not. Further research, conducted in a multi – center design, with larger patient groups and a longer follow – up period is needed on this inquiry.

### Ethics

**Ethics Committee Approval:** This study was conducted in the emergency department of University of Health Sciences Türkiye, Bursa Yüksek İhtisas Training and Research Hospital with the approval of the clinical research ethics committee with the (desicion number: 2011-KAEK-25 2022/03-08, date: 23.1.2011).

**Informed Consent:** Patients who gave consent to the study by themselves or their relatives were included.

### Footnotes

### Author Contributions

Surgical and Medical Practices: S.Ç., M.Y., Y.İ., O.Ö., Z.E., K.H., A.Z., Concept: S.Ç., M.Y., M.O.A., Y.İ., O.Ö., Ö.F.D., K.H., A.Z., Design: S.Ç., M.Y., Y.İ., H.K., O.Ö., Z.E., Ö.F.D., D.Y., A.Z., Data Collection or Processing: S.Ç., M.Y., M.O.A., Z.E., Ö.F.D., D.Y., A.Z., Analysis or Interpretation: S.Ç., M.Y., M.O.A., Y.İ., H.K., Ö.F.D., D.Y., Literature Search: S.Ç., M.Y., O.Ö., Z.E., D.Y., K.H., Writing: S.Ç., M.Y., M.O.A., Y.İ., Z.E., K.H., A.Z.

**Conflict of Interest:** The authors declare that they have no conflict of interest.

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