Available online at www.medicinescience.org

ORIGINAL ARTICLE

Medicine Science International Medical Journal

Medicine Science 2023;12(3):783-90

# Effects of serum vaspin level on infarct volume and severity in patients with acute ischemic stroke

**Filiz Demirdogen[1], Gokhan Ozdemir[2], Guven Akcay[3], Ibrahim Iyigun[4]**

*[1]Binali Yıldırım University, Mengücek Gazi Education and Research Hospital, Department of Neurology, Erzincan, Türkiye*
*[2]Selcuk University, Faculty of Medicine, Department of Neurology, Konya, Türkiye*
*[3]Hitit University, Faculty of Medicine, Department of Biophysics, Çorum, Türkiye*
*[4]Erzurum Atatürk University, Faculty of Medicine, Department of Neurology, Erzurum, Türkiye*

Received 22 March 2023; Accepted 06 July 2023
Available online 21.08.2023 with doi: 10.5455/medscience.2023.03.040



**Abstract**

Stroke has a major impact worldwide in terms of mortality and morbidity. The aim of our research was to determine the effect of serum Vaspin level on acute ischemic stroke and to investigate the relationship between serum Vaspin level, National Institutes of Health Stroke Scale (NIHSS) and infarct volume. This study was carried out in 100 patients hospitalized with acute ischemic stroke diagnosis in the Neurology Department of Yakutiye Research Hospital of Atatürk University Faculty of Medicine and 80 healthy controls. The age and sex of the patient and control groups were consistent. Patients who met the following criteria were selected to be included in the study. Exclusion criteria: patients with ischemic stroke presenting after the first 24 hours to overcome the potential bias associated with inflammatory conditions, patients diagnosed with rheumatic and autoimmune diseases, diseases that cause hypoxia and coronary artery disease. Vaspin levels were measured and compared in patients with acute ischemic stroke (AIS) and in the control group. NIHSS calculated. Infarct volume was also measured with Cavalieri's principle. The correlation between them was calculated statistically. Vaspin level was higher in patients with ischemic stroke compared to the control group (825 vs. 939; $p<0.001$). Vaspin levels were significantly higher in patients who had acute ischemic stroke. Since examination of Vaspin levels in patients presented with acute ischemic stroke suggested the severity of the stroke, it was found to be important in terms of mortality and morbidity affiliated with it.

**Keywords:** Ischemic stroke, infarct volume, NIHSS, vaspin

## Introduction

Stroke is one of the most important health problems all over the world. Stroke is a clinical neurological picture that usually occurs due to focal cerebrovascular disease, sudden onset, lasting longer than 24 hours, as a result of changes in the blood flowing through the blood vessels and/or vessels that blood supply the brain, blockage of the vessel or lack of blood supply. It is difficult to predict the cause of stroke before the age of forty-five. Ischaemic strokes account for 80-90% of all strokes [1]. Stroke has a major impact worldwide in terms of mortality and morbidity. The interaction of atherosclerosis and obesity with the visceral adipose tissue-derived serpin (vaspin) is indicated to be among the causes of stroke. Recent studies have shown increased serum vaspin levels in the acute phase of stroke. However, there is a lack of findings regarding the interaction between ischemic cerebrovascular incidents and vaspin. Neuron necrosis occurring in ischemic stroke is closely correlated to neuroinflammation [1-3]. This is due to the transport of numerous inflammatory mediators such as adipokines (adipocytokines), chemokines,

**CITATION**
Demirdogen F, Ozdemir G, Akcay G, Iyigun I. Effects of serum vaspin level on ınfarct volume and severity in patients with acute ıschemic stroke. Med Science. 2023;12(3):783-90.

**Corresponding Author:** Filiz Demirdogen, Binali Yıldırım University, Mengücek Gazi Education and Research Hospital, Department of Neurology, Erzincan, Türkiye
Email: fdemirdogen24@gmail.com

DOI: 10.5455/medscience.2023.03.040

leukocytes and adhesion molecules into and around ischemic tissue. Vaspin is a member of the Serine protease inhibitor family. It is a recently discovered adipocytokine secreted from visceral adipose tissue. In addition, some studies have shown that adipokines are secreted in atherosclerotic plaques and their local and endocrinal effects in the lesion were also shown [4]. Vaspin was first isolated from Otsuka Long-Evans Tokushima Fatty guinea pigs, which are animal models of type 2 Diabetes Mellitus (DM) characterized by dyslipidemia, abdominal obesity, hypertension and insulin resistance [5]. There are studies suggesting that Vaspin suppresses the expression of leptin, resistin and tumor necrosis factor-α (TNF-α) in obese individuals and stimulates the expression of decreasing adiponectin in obese individuals [6,7]. In connection with the studies in this direction, it is thought that Vaspin may be associated with obesity and metabolic syndrome [6]. In another study, they found out that serum Vaspin levels were associated with the insulin resistance in diabetic women and showed that serum Vaspin levels were positively correlated with hemoglobiini-A1C (HbA1C). In this study, Vaspin levels were lower in patients with microvascular complications such as neuropathy, retinopathy or nephropathy compared to those without microvascular complications [8]. The role of new members of adipokines in atherosclerosis-related cerebrovascular events has not been fully established. The interaction of SERPIN derived from visceral adipose tissue with atherosclerosis and obesity is shown. Data on the interaction between ischaemic cerebrovascular events and VASPIN are lacking. More clinical data are needed to elucidate the exact role of VASPIN in ischaemic cerebrovascular events. In our study, we aimed to investigate the correlation between serum Vaspin level and infarct volume and severity in acute ischemic stroke (AIS) patients.

## Material and Methods

This study was carried out in 100 patients hospitalized with AIS in the Neurology Department of Yakutiye Research Hospital of Atatürk University Faculty of Medicine and 80 healthy controls. The age and sex of the AIS patient and control groups were consistent. Patients who met the following criteria were selected to be included in the study.

### Inclusion criteria

- Patients with acute ischemic stroke between the ages of 18-80
- Volunteers will be included in the study

### Exclusion criteria

- Ischemic stroke patients presenting after the first 24 hours
- Patients with transient ischemic stroke
- Patients with brain tumor or systemic malignancy
- Patients with intracerebral and subarachnoid hemorrhage due to stroke
- Those with rheumatic and autoimmune diseases
- Those with acute head trauma
- Patients using anti-inflammatory drugs daily
- Patients with near-term and simultaneous myocardial infarction
- Patients with chronic obstructive pulmonary disease (COPD) and asthma

As for the medical histories of the patients included in the study; age, gender, concomitant findings and risk factors of ischemic cerebrovascular diseases in their personal and family histories were investigated. Systemic and neurological examinations were carried out for all the patients.

Peripheral venous blood samples were taken from the patients within the first 24 hours after stroke and at any time from the control group. The blood samples were centrifuged at +4°C degrees and 1500 rpm for 15 min., and the serum was separated. Small volumes were placed in eppendorf tubes and stored at -80°C until the analysis. When it is time to analyze, samples taken from -80°C were allowed to dissolve gradually after kept in -20°C overnight and to +4°C the next day, and then from +4°C to room temperature. Then the samples were mixed with the shaker. Homogeneous serum was obtained and the analyze was carried out using the homogeneous serum. Vaspin was measured using the commercially available RAYBIOTECH® Brand EIA-VAP-1 Human Vaspin EIA kit. Measurements were performed using the RAYBIOTECH® Brand EIA-VAP-1 Human VASPIN EIA kit protocol.

NIHSS was used to determine the severity and progression of stroke and the level of consciousness, conscious response to questions, response to orders, extraocular movements, visual field, facial paralysis, limb motor movements, extremity ataxia, sensation, aphasia, dysarthria and the degree of neglect were scored (Table 1). This scale was performed on the patients on the first day of stroke. NIHSS levels were divided into 3 groups. Patients with modified NIH stroke score between 0-6 were grouped as mild, those with a score between 7 and 15 were grouped as moderate, and those whose scores were 16 and above were grouped as severe [9].

### Calculation of infarct volume of patients imaging of patients' brains with magnetic resonance device

Magnetic Resonance Imaging (MRI) studies were performed with eco planar imaging system on Siemens® Sonata 1.5-T and GE-HD 3-T MR devices. The imaging protocol was determined as DWI-weighted images. Parameters used in imaging are: For 1.5 T MRI examination; TR/TE was 3300/84 ms, field of view (FOV) was 240 mm, section thickness was 5mm, and the cross-sectional gap was 1.5 mm. For 3 T MRI examination; TR/TE was 6000/80 ms, field of view (FOV) was 240 mm, section thickness was 5mm, and the cross-sectional gap was 1.5 mm.

DOI: 10.5455/medscience.2023.03.040

**Table 1.** NIH stroke scale

| | | |
|---|---|---|
| **Conscious level** | Awake | 0 |
| | Somnolence | 1 |
| | Strong stimulus required | 2 |
| | Reflex or autonomic response | 3 |
| **Conscious response to questions** | Responding accurately | 0 |
| | Rarely responding accurately | 1 |
| | Responding inaccurately or cannot speak | 2 |
| **Responding to orders** | Obeying accurately | 0 |
| | Occasionally obeying accurately | 1 |
| | Responding inaccurately | 2 |
| **Extraocular movements** | Normal | 0 |
| | Partial gaze palsy | 1 |
| | Deviation of eyes, total gaze palsy | 2 |
| **Visual field** | No loss of visual field | 0 |
| | Partial hemianopia | 1 |
| | Full hemianopia | 2 |
| **Facial paresis** | Normal | 0 |
| | Minimal | 1 |
| | Partial | 2 |
| | Full | 3 |
| **Arm, motor** | Holds his/her arm at 90° for 10 sec | 0 |
| | Holds his/her arm at less than 90° | 1 |
| | Cannot hold his/her arm at 90° | 2 |
| | Arm falls, cannot overcome the force of gravity | 3 |
| **Foot, motor** | Can hold his/her foot at 30° for 5 sec | 0 |
| | Holds his/her foot at 30° for less than 5 sec | 1 |
| | Cannot hold his/her foot at 30° | 2 |
| | Foot cannot overcome the force of gravity | 3 |
| **Extremity ataxia** | None | 0 |
| | Present in one extremity | 1 |
| | Present in two extremities | 2 |
| **Sensorial** | No sensation loss | 0 |
| | Moderate sensation loss | 1 |
| | Severe or complete loss of sensation | 2 |
| **Negligence** | None | 0 |
| | Visual, auditory and tactile extinction phenomenon | 1 |
| | Significant attention disorder | 2 |
| **Dysarthria** | Normal | 0 |
| | Moderate, but difficulty in understanding | 1 |
| | Severe, incomprehensible articulation | 2 |
| **Tongue** | Normal | 0 |
| | Moderate impairment in speech and paraphasia | 1 |
| | Severe Broca or Wernicke aphasia | 2 |
| | Mutism or global aphasia | 3 |

## Calculation of brain and infarct volume by stereological methods

All brain and infarct areas were calculated by Cavalieri method. The volumes of objects with a regular or symmetrical shape (such as cubes, prisms or cylinders) are easily calculated using the following mathematical formula. However, it may not be possible to use this method for biological objects most of the time. Especially when it is necessary to calculate the volume of any structure or organ in living things or to calculate volume ratios, different methods must be used.

$$\hat{V} = t \times a$$

(V) in the formula defines the volume of the object, (t) the height of the object and (a) the floor area of the object [10].

Cavalieri showed that the volumes of three-dimensional objects that do not have a regular geometric shape can be calculated by dividing them into parallel slices. With the Cavalieri method, it is possible to calculate the volume of an organ to be examined or a specific structure located within the organ. With Cavalieri method, the volume to be calculated is divided into slices, the sectional surface area of each slice is found, multiplied by the thickness of the section and thus the volume of the slice is calculated and finally the volumes of the slices are summed up to calculate the total volume of the respective structure.

In order to calculate the infarct volume, we need to calculate the surface area of the sections. In order to calculate the surface area of the sections and infarct areas, we used the image analysis system with the special software called Stereo Investigator software and the point grid method.

## Statistical method

Statistical analysis was performed with SPSS program. The Kolmogorov-Smirnov test was used to check whether the data is normally distributed. Among numerical variables, those who showed normal distribution were indicated as mean±standard deviation. And also those non-normally distributed were shown as median (min-max). T-test and Mann-Whitney U were used in independent samples to determine the factors associated with two-category risk groups. ANOVA test and Kruskall Wallis H test were used to determine the factors associated with three-category risk groups. Chi-square and Fisher's exact Chi-square test were used to compare categorical data. Correlation between numerical variables was analyzed by Pearson and Spearman correlation analysis. Stepwise multivariate linear regression analysis was used to determine the independent predictors of NIHHS score. Logarithmic transformation was performed since the NIHSS levels, infarct volume levels and Vaspin levels were not normally distributed prior to the regression analysis. Since there were NIHSS levels and Vaspin levels with zero value, "1" was added to the original values and then they were converted to normal distribution upon applying logarithmic transformation. Thus, linear regression assumptions were satisfied. Stepwise multivariate logistic regression analysis was used to predict the severe NIHSS classification. Predictive value of numerical independent predictors was evaluated with ROC Curve analysis, Youden index method.

## Results

### Demographic and clinical data of the research population

The research population consisted of 180 persons; 80 healthy individuals and 100 patients. 40% of the AIS patients were female and 60% were male. The age range of the patients was

DOI: 10.5455/medscience.2023.03.040

between 33-85 years and the mean age was 70.1±11.0 years, and the geriatric age rate was 74%. Demographic data of the patients are shown in Table 2. The Vaspin level of the patients was between 0-1000 ng/ml and the median was 879 ng/ml. NIH stroke scale of the patients was between 0-33 and the median was 11. 23% of the patients were in the NIHSS low-risk group 38% of the patients were in the moderate-risk group and 39% of the patients were in the severe-risk group.

The median infarct volume of the patients was 441.8 and ranged from 6 to 4545.8. Demographic and clinical findings of the AIS patients are presented in detail in Table 2.

**Table 2.** Demographic and clinical data of the patients

| Variables | All population (n=100) |
|---|---|
| **Age (years)** | 70.1±11.0 |
| **<65 years of age, n (%)** | 26 (26.0) |
| **≥65 years of age, n (%)** | 74 (74.0) |
| **Sex, n(%)** | |
| **Female** | 40 (40.0) |
| **Male** | 60 (60.0) |
| **DM, n (%)** | 22 (22.0) |
| **HT, n (%)** | 65 (65.0) |
| **CRF, n (%)** | 21 (21.0) |
| **AF, n (%)** | 36 (36.0) |
| **CAD, n (%)** | 24 (24.0) |
| **HL, n (%)** | 24 (24.0) |
| **CHF, n (%)** | 21 (21.0) |
| **Previous strokes, n (%)** | 12 (12.0) |
| **CVAS stenosis, n (%)** | |
| **<50%** | 89 (89.0) |
| **50-70%** | 7 (7.0) |
| **> 100%** | 4 (4.0) |
| **VASPIN, (ng/mL)** | 879 (0-1000) |
| **NIH stroke scale, (score)** | 11 (0-33) |
| **Low risk, n (%)** | 23 (23.0) |
| **Moderate risk, n (%)** | 38 (38.0) |
| **Severe risk, n (%)** | 39 (39.0) |
| **Infarct volume,** | 441.8 (6-4545.8) |

Numerical variables were indicated as mean±standard deviation or as median (min-max). Categorical variables were shown in figures (%). *p<0.05 indicates statistical significance.
DM: diabetes mellitus, HT: hypertension, CRF: chronic renal failure, AF: atrial fibrillation, CAD: coronary artery disease, HL: hyperlipidemia, CHF: congestive heart failure, CVAS: carotid vertebral artery stenosis

### Demographic and clinical data as per NIHSS classification results

According to the NIHSS classification, the mean age didn't differ significantly between the low, moderate and high risk groups (p=0.535). Coronear arter disease (CAD) rate increased as the NIHSS classification increased from low to severe (Low Risk: 30.4% versus Moderate risk: 7.9% versus High risk: 28.2%; p=0,335). The Vaspin level was higher in the severe NIHSS group compared to other risk groups (p=0.029).

As the NIHSS risk group increased, there also was an increase in the volume of the infarct (p=0.623) (Figure 1).

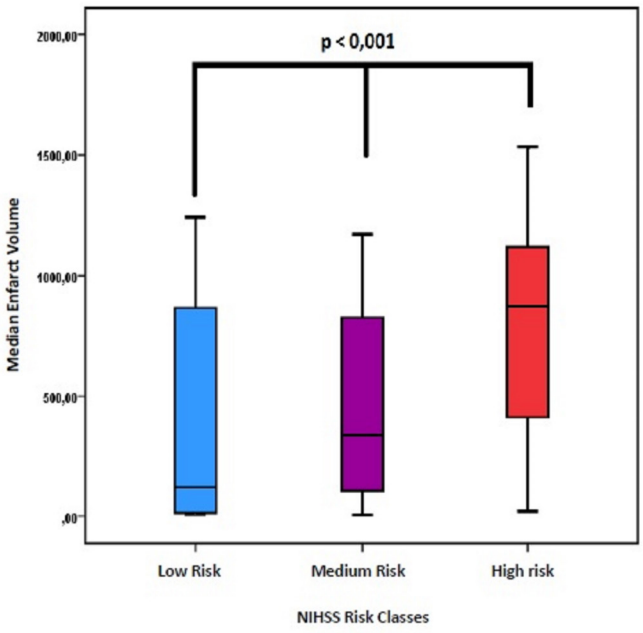


**Figure 1.** Average infarct volume levels based on NIHSS risk classes

### NIHSS level results

The NIH stroke scale was found to be higher in patients with coronary artery disease compared to those without coronary artery disease (10 versus 18; p=0.005).

### Vaspin level results

The Vaspin level was higher in AIS patients diagnosed compared to healthy individuals (825.0 versus 939.5; p=0.031). No significant difference was observed between Vaspin level and demographic findings (Table 3).

### Infarct volume level results

Infarct volume was higher in males than in females (296.4 versus 667.1; p=0,133). The infarct volume was higher in patients with chronic renal failure than those who did not have it (364.1 versus 1110.7; p=0,004). The median infarct volume was higher in patients with hyperlipidemia than those who did not have HL (362.4 versus 1063.4; p=0.0035).

### Correlation analysis

Although there was a positive correlation between NIHHS and infarct volume (r=0.374; p=0.0058), there was no correlation between NIHHS and age (p=0.067).

A positive correlation was found between NIHHS and infarct volume (Figure 2).

Positive correlation was detected between NIHHS and Vaspin levels (r=0.354; p=0.0048) (Figure 3) (Table 4).

DOI: 10.5455/medscience.2023.03.040

**Table 3.** Data regarding VASPİN levels

| | | VASPIN | | | |
|---|---|---|---|---|---|
| **Variables** | | **n** | **Median** | **min-max** | **P** |
| **Age** | <65 years old | 26 | 879.0 | 51.1-1000 | 0.877 |
| | ≥65 years old | 74 | 854.8 | 0-1000 | |
| **Gender** | Female | 40 | 879.0 | 0-1000 | 0.822 |
| | Male | 60 | 866.9 | 0-1000 | |
| **DM** | No | 78 | 854.8 | 0-1000 | 0.414 |
| | Yes | 22 | 903.2 | 0-1000 | |
| **HT** | No | 35 | 854.8 | 0-1000 | 0.634 |
| | Yes | 65 | 903.2 | 0-1000 | |
| **CRF** | No | 79 | 879.0 | 0.5-1000 | 0.157 |
| | Yes | 21 | 806.5 | 0-1000 | |
| **AF** | No | 64 | 879.0 | 0-1000 | 0.768 |
| | Yes | 36 | 854.8 | 0-1000 | |
| **CAD** | No | 76 | 879.0 | 0-1000 | 0.984 |
| | Yes | 24 | 842.7 | 0-1000 | |
| **HL** | No | 76 | 854.8 | 0-1000 | 0.495 |
| | Yes | 24 | 939.5 | 12.9-1000 | |
| **CHF** | No | 79 | 879.0 | 0-1000 | 0.684 |
| | Yes | 21 | 879.0 | 0-1000 | |
| **Previous strokes** | No | 88 | 854.8 | 0-1000 | 0.113 |
| | Yes | 12 | 963.7 | 637.1-1000 | |
| **CVAS stenosis** | <50% | 89 | 829.0 | 0-1000 | 0.753 |
| | 50-70% | 7 | 927.4 | 52-1000 | |
| | 100% | 4 | 661.3 | 39.9-1000 | |
| **CVAS stenosis** | Normal | 86 | 879.0 | 0-1000 | 0.347 |
| | Stenosis | 14 | 758.1 | 39.9-1000 | |

Numerical variables were indicated as mean±standard deviation or as median (min-max). Categorical variables were shown in figures (%). *p<0.05 indicates statistical significance.
DM: diabetes mellitus, HT: hypertension, CRF: chronic renal failure, AF: atrial fibrillation, CAD: coronary artery disease, HL: hyperlipidemia, CHF: congestive heart failure, CVAS: carotid vertebral artery stenosis

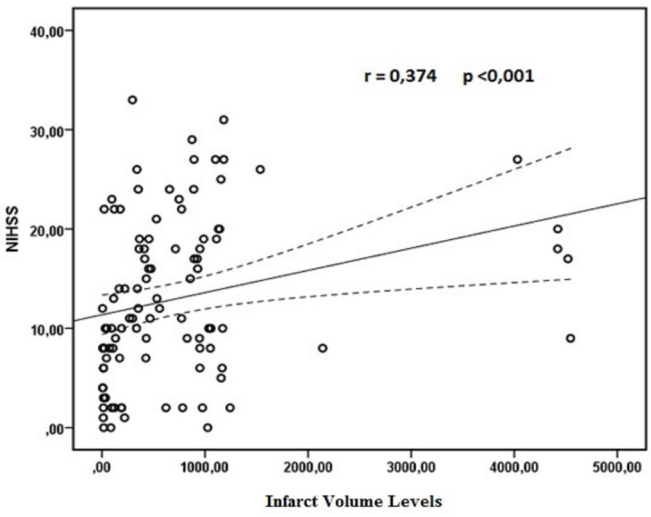


**Figure 2.** Correlation between NIHSS and infarct volume levels

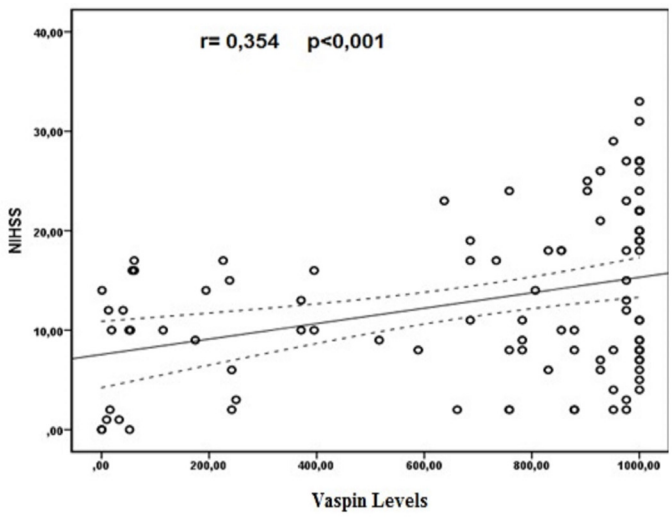


**Figure 3.** Correlation between NIHSS and Vaspin levels

DOI: 10.5455/medscience.2023.03.040

**Table 4.** Correlation analysis results

| Variables | NIHSS | | Infarct volume | | VASPIN | |
|---|---|---|---|---|---|---|
| | r | p | r | p | r | P |
| **Age** | 0.137 | 0.175 | 0.060 | 0.552 | 0.091 | 0.366 |
| **NIHSS** | - | - | 0.374 | <0.001* | 0.354 | <0.001* |
| **Infarct volume** | 0.374 | <0.001* | - | - | 0.128 | 0.204 |
| **VASPIN** | 0.354 | <0.001* | 0.128 | 0.204 | - | - |

*p<0.05 indicates statistical significance

## Discussion

Stroke has a significant impact on mortality and morbidity worldwide [11]. In inflammation-induced atherosclerosis, these inflammatory cells predispose to acute stroke by forming susceptible and ruptured atherosclerotic plaques in the relevant vascular beds via adhesion molecules and cytokines secreted by dysfunctional endothelium [12]. Adipose tissue produces a variety of cytokines and cytokines are known to play an important role in insulin sensitivity and pathogenesis. These cytokines can cause inflammation, diabetes, dyslipidemia, coronary artery disease and carotid atherosclerosis [13,14]. However, the role of these cytokines in atherosclerosis remains unclear. In addition to that, the role of atherosclerosis in stroke ranges from 27% to 43% [15,16].

NIHSS scoring system, which is used routinely because of its clinical practicality by neurologists in the follow-up and treatment of AIS, is superior to simple clinical stroke scales in clinical stroke researches [17-19]. It has been suggested to be a good indicator of prognosis after stroke [20,21]. Recent studies have shown that NIHSS levels are between 7.0-18.4 in patients with acute stroke [22-26]. In our research, the median value of NIHSS was 11 and the rate of the patients in the severe risk group was 39%.

The effects of adiponectins on acute stroke are one of the current topics that have been recently studied. Kadoglou et al. reported that Vaspin and Visfatin levels were high, Ghrelin levels were low and Apelin levels did not change in patients with AIS [27]. In the study carried out by Cura et al., Vaspin level was found to be high in AIS patients compared to the control group [28]. In our study, Vaspin levels were found to be higher in AIS patients compared to the control group. Cardiac outcome was not investigated in our study, but since NIHSS scoring is an independent predictor of cardiac outcome, independent predictors of NIH Stroke Scale were investigated and Vaspin level, CAD and infarct volume levels were determined as independent predictors.

According to our results, although the NIH stroke scale was higher in geriatric age and male gender, no statistically significant difference was observed. Similarly, in the severe NIHSS group, geriatric age and male sex ratio were higher compared to other NIHSS groups but no statistical difference was observed. However, these high rates observed in our study suggest that they might be significant in large populations in terms of NIHSS.

Another factor that supports this finding is that the infarct volume is significantly higher in male patients compared to women. Increased volume level was determined as a predictor of NIH Stroke scale and severity. In a study conducted by Tan et al., a positive correlation was found between infarct volume level and NIH Stroke Scale in patients who had AIS, and low NIHSS was understood to be an independent predictor of good functional outcome in MR imaging. In the literature it is stated that there is a positive correlation between NIHHS score and infarct volume [29-32]. Additionally, no relation was found between age and gender and Vaspin levels in our study and this result was in line with the literature [33]. In our study, the highest co-morbidity observed in patients with AIS was hypertension (65%). In the literature, the presence of hypertension is stated as a factor increasing the risk of stroke [34-38]. In the Aegean Stroke Database, among the studies carried out in our country, hypertension history was present in 63% of the patients [39]. The most important risk factor that can be corrected in stroke prophylaxis is hypertension. Vaspin level has a strong effect in the pathogenesis of hypertension. It is stated that the Vaspin level, with the antioxidative and anti-inflammatory mechanisms, can be effective in the development of hypertension by affecting the peripheral vascular resistance. Kameshima et al. showed that Vaspin levels were effective in inhibiting acetylcholine by endothelium-induced relaxation in isolated blood vessels [40]. In a study conducted by Esaki et al., no correlation was found between serum Vaspin levels and systolic and diastolic blood pressures, however, hypertension rates were significantly higher in patients with high Vaspin levels [33]. In this study, Vaspin levels were found to be higher in patients with hypertension but it was not statistically significant. In a study conducted by Tan et al., no significant correlation was found between infarct volume and hypertension [22]. No statistically significant difference was found between the presence of hypertension and infarct volume. Vaspin levels were reported to be higher in patients with type II diabetes compared to healthy control group. According to our results, there was no significant difference in Vaspin level in patients with AIS in terms of the presence of DM.

The Framingham study investigated the presence of coronary heart disease in 344 stroke cases based on their 24 years of follow-up. Risk of stroke is 3 times higher in patients with heart diseases. In a prospective study carried out by Davis et al. in Rochester, it was reported that coronary heart disease increased the risk of stroke by 2.2 times [37]. In our study, 24% of AIS patients had CAD and the CAD rate was higher in patients with severe NIHSS scores. No correlation was found between CAD and Vaspin and infarct volume levels.

## Conclusion

In this study, Vaspin levels were not found to be correlated with the presence of CAD, and although they were positively correlated with the infarct volume, the effect of this correlation can be eliminated in the regression analysis and since it is an independent predictor for NIH Stroke scale and severity, we think that it can be used as a marker to predict both the cardiac outcome and the presence and severity of AIS.

**Conflict of Interests**
*The authors declare that there is no conflict of interest in the study.*

**Financial Disclosure**
*The authors declare that they have received no financial support for the study.*

**Ethical Approval**
*The study was approved by the Ethics Committee of Erzurum Atatürk University Medicine Faculty, Turkey, and conformed to the ethical guidelines of the Declaration of Helsinki [approval number: meeting number: 1. Decision: 18 /28.01.2016].*

**Acknowledgments**
*The authors would like to thank all the healthcare providers at Ataturk University, Faculty of Medicine, Department of Neurology.*

## References

1. Clausen BH, Lambertsen KL, Babcock AA, et al. Interleukin-1beta and tumor necrosis factor-alpha are expressed by different subsets of microglia and macrophages after ischemic stroke in mice. J Neuroinflammation. 2008;5:46.

2. Downes CE, Crack PJ. Neural injury following stroke: are Toll-like receptors the link between the immune system and the CNS?. Br J Pharmacol. 2010;160:1872-88.

3. Coull BM. Inflammation and stroke: introduction. Stroke. 2007;38:631.

4. Tedgui A, Mallat Z. Cytokines in atherosclerosis: pathogenic and regulatory pathways. Physiol Rev. 2006;86:515-81.

5. Lago F, Dieguez C, Gómez-Reino J, Gualillo O. The emerging role of adipokines as mediators of inflammation and immune responses. Cytokine Growth Factor Rev. 2007;18:313-25.

6. Hida K, Wada J, Eguchi J, et al. Visceral adipose tissue-derived serine protease inhibitor: a unique insulin-sensitizing adipocytokine in obesity. Proc Natl Acad Sci U S A. 2005;102:10610-5.

7. Youn BS, Kloting N, Kratzsch J, et al. Serum vaspin concentrations in human obesity and type 2 diabetes. Diabetes. 2008;57:372-7.

8. Tan BK, Heutling D, Chen J, et al. Metformin decrreases the adipokine vaspin in overweight women with polycystic ovary syndrome concomitant with improvement in insulin sensitivity and a decrease in insülin resistance. Diabetes. 2008;57:1501-7.

9. NINDS rtPA Stroke Study Group; Lyden PD, Lu M, Levine SR, et al. A modified national institutes of health stroke scale for use in stroke clinical trials: preliminary reliability and validity. Stroke: Journal of the American Heart Association. 2001;32:1310-7.

10. Mayvev TM, Olsen DR. Magnetic resonanse imaging (MRI) and model freee stimates of brain volunes determinend using the cavalliere principle. J Anat. 1991;178:133-44.

11. Avgerinos ED, Kadoglou NP, Moulakakis KG, et al. Current role of biomarkers in carotid disease: a systematic review. Int J Stroke. 2011;6:337-45.

12. Kadoglou NP, Sailer N, Moumtzouoglou A, et al. Adipokines: a novel link between adiposity and carotid plaque vulnerability. Eur J Clin Invest. 2012;42:1278-86.

13. Nikolopoulou A, Kadoglou NP. Obesity and metabolic syndrome as related to cardiovascular disease. Expert Rev Cardiovasc Ther. 2012;10:933-9.

14. Michel P, Odier C, Rutgers M, et al. The acute strok registry and analysis of lausanne (ASTRAL): design and baseline analysis of an ischemic Strok registry including acute multimodal imaging. Stroke. 2010;41:2491-8.

15. Bogousslakarşıky J, Van Melle G, Regli F. The Lausanne Strok Registry: analysis of 1,000 consecutive patients with first Stroke. Stroke. 1988;19:1083-92.

16. Muir KW, Weir CJ, Murray GD, et al. Comparison of neurological scales and scoring systems for acute stroke prognosis. Stroke. 1996;27:1817-20.

17. Albers GW, Bates VE, Clark WM, et al. Intravenous tissue-type plasminogen activator for treatment of acute Stroke: The Standard Treatment with Alteplase to Reverse Stroke (STARS) study. JAMA. 2000;283:1145-50.

18. Grond M, Stenzel C, Schmulling S, et al. Early intravenous thrombolysis for acute ischemic Strok in a community-based approach. Stroke. 1998;29:1544-9.

19. Adams HP, Jr., Davis PH, Leira EC, et al. Baseline NIH strok scale score strongly predicts outcome after strok: a report of the Trial of Org 10172 in Acute Strok Treatment (TOAST). Neurology. 1999;53:126-31.

20. Frankel MR, Morgenstern LB, Kwiatkowski T, et al. Predicting prognosis after Strok: a placebo group analysis from the National Institute of Neurological Disorders and Strok rt-PA Strok Trial. Neurology. 2000;55:952-9.

21. Tan BY, Wan-Yee K, Paliwal P, et al. Good intracranial collaterals trump poor alberta strok program early CT score for intravenous thrombolysis in anterior circulation acute ischemic stroke. Stroke. 2016;47:2292-8.

22. Nezu T, Hosomi N, Lip GY, et al. Temporal trends in strok severity and prior antithrombotic use among acute ischemic strok patients in Japan. Circ Journal. 2016;80:2033-6.

23. Li YS, Chen W, Liu S, et al. Serum macrophage migration inhibitory factor levels are associated with infarct volumes and long-term outcomes in patients with acute ischemic Strok. Int J Neurosci 2017;127:539-46.

24. Khan MA, Baird GL, Miller D, et al. Endovascular treatment of acute ischemic Stroke in nonagenarians compared with younger patients in a multicenter cohort. J Neurointerv Surg. 2017;9:727-31.

25. Chen L, Fang J, Ma R, et al. Additional effects of acupuncture on early comprehensive rehabilitation in patients with mild to moderate acute ischemic Strok: a multicenter randomized controlled trial. BMC Complement Altern Med. 2016;16:226-34.

26. Kadoglou NP, Fotiadis G, Lambadiari V, et al. Serum levels of novel adipokines in patients with acute ischemic Strok: potential contribution to diagnosis and prognosis. Peptides. 2014;57:12-6.

27. Cura HS, Ozdemir HH, Demir CF, et al. Investigation of vaspin level in patients with acute ischemic stroke. J Stroke Cerebrovasc Dis. 2014;23:453-6.

28. Beaulieu C, de Crespigny A, Tong DC, et al. Longitudinal magnetic resonance imaging study of perfusion and diffusion in stroke: evolution of lesion volume and correlation with clinical outcome. Ann Neurol. 1999;46:568-78.

29. Baird AE, Lovblad KO, Dashe JF, et al. Clinical correlations of diffusion and perfusion lesion volumes in acute ischemic stroke. Cerebrovasc Dis. 2000;10:441-8.

30. Lovblad KO, Baird AE, Schlaug G, et al. Ischemic lesion volumes in acute Strok by diffusion-weighted magnetic resonance imaging correlate with clinical outcome. Ann Neurol. 1997;42:164-70.

31. Tong DC, Yenari MA, Albers GW, et al. Correlation of perfusion- and diffusion-weighted MRG with NIHSS score in acute (<6.5 hour) ischemic Strok. Neurology. 1998;50:864-70.

32. Esaki E, Adachi H, Hirai Y, et al. Serum VASPİN levels are positively associated with carotid atherosclerosis in a general population. Atherosclerosis. 2014;233:248-52.

DOI: 10.5455/medscience.2023.03.040

33. Kario K. Key points of the 2019 Japanese society of hypertension guidelines for the management of hypertension. Korean Circ J. 2019;49:1123-35.

34. Gürger M, Bozdemir MN, Yıldız M, et al. The value of cardiac markers in predicting the hospital mortality of ischemic stroke patients. Turk J Emerg Med. 2008;8:59-66.

35. Kannel WB, Wolf PA, McGee DL, et al. Systolic blood pressure, arterial rigidity, and risk of strok: the framingham study. JAMA. 1981;245:1225-9.

36. Davis PH, Dambrosia JM, Schoenberg BS, et al. Risk factors for ischemic stroke: a prospective study in Rochester, Minnesota. Ann Neurol. 1987;22:319-27.

37. Ringleb P, Schellinger P, Hacke W. Leitlinien zum Management von Patienten mit akutem Hirninfarkt oder TIA der Europäischen Schlaganfallorganisation 2008. Nervenarzt. 2008;79:936-957.

38. Kumral E, Özkaya B, Vardarlı E, et al. Ege stroke data bank: an analysis of 2000 patients with stroke. Turk J Neurol. 1997;1:3-12.

39. Kameshima S, Sakamoto Y, Okada M, Yamawaki H. Vaspin prevents elevation of blood pressure through inhibition of peripheral vascular remodelling in spontaneously hypertensive rats. Acta Physiol (Oxf). 2016;217:120-9.

40. Kameshima S, Yamada K, Morita T, et al. Visceral adipose tissue-derived serine protease inhibitor augments acetylcholine-induced relaxation via the inhibition of acetylcholine esterase activity in rat isolated mesenteric artery. Acta Physiol (Oxf). 2016;216:203-10.