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Original Article

The Effect of Uric Acid Levels on Mortality in Acute Ischemic Stroke Patients in the Emergency Department

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ABSTRACT

Objective: Uric acid (UA) is a molecule whose effect on cerebrovascular diseases, hypertension, and diabetes has been investigated. Conflicting results have been obtained in studies examining the relationship between ischemic stroke and UA. The previous studies have found that both high and low UA levels are associated with poor prognosis in stroke patients. Therefore, we investigated the effects of UA levels on mortality, stroke severity, and clinical outcome in ischemic stroke.

Methods: The patient demographics, chronic diseases, serum UA (SUA) levels, National Institutes of Health Stroke Scale scores, and modified Rankin scale (mRS) scores were recorded. The patients were divided into three groups based on SUA levels below 3.59 mg/dL, between 3.59 mg/dL and 8.5 mg/dL, and above 8.5 mg/dL. Pearson's Chi-square test was used to compare the data.

Results: A total of 820 patients were included in the study and 42.4% of them were women. The mean age was 68.53 years. SUA levels were lower in women. Moderate and moderate-to-severe strokes, a high mortality rate, poor neurological outcomes, and the need for intensive care and mechanical ventilation were most common in the patients with SUA levels above 8.5 mg/dL followed by the group with SUA levels below 3.59 mg/dL. The lowest mortality, best neurological outcomes, most cases of moderate stroke, and least need for intensive care and mechanical ventilation were in the group with SUA level between 3.59 mg/dL and 8.5 mg/dL.

Conclusion: UA has both oxidant and antioxidant properties and its effect is level dependent. The best prognosis was seen in the group with SUA levels of 3.59–8.5 mg/dL. Therefore, maintaining the UA levels within this range when following ischemic stroke patients might reduce mortality and morbidity.

INTRODUCTION

Ischemic stroke is the cessation of blood flow to a certain part of the brain for any reason, resulting in brain cell death. Atherosclerosis, hypertension (HTN), diabetes mellitus (DM), cardiovascular diseases and rhythm disorders, metabolic syndrome, and renal failure are risk factors for ischemic stroke.

Stroke is the fifth leading cause of death. According to 2019 data, 3.3 million people died due to ischemic stroke. It is the first among the causes of long-term disability.[1] Due to its high mortality and morbidity rates, stroke must be managed carefully. It is crucial to diagnose quickly and initiate appropriate treatment.

According to the current ischemic stroke guideline, brain computed tomography (CT) should be performed quickly in suspected cases of stroke. If bleeding is absent, the cases should be evaluated quickly in terms of thrombolytic and

thrombectomy indications. Thrombolytic therapy should be administered within the first 4.5 h for appropriate patient.[2] If there is an indication for thrombectomy, brain CT angiography should be performed. Although the guideline recommends thrombectomy for the first 6 h, there are studies suggesting that thrombectomy can be performed up to 24 h.[3] It is aimed to reduce mortality and morbidity with thrombolytic therapy and thrombectomy. These two treatments have replaced the previous treatments (such as acetylsalicylic acid and low-molecular-weight heparin) for appropriate patient groups.

High mortality and morbidity situations necessitated the investigation of various markers that can help clinicians in predicting and improving prognosis.[4] Uric acid (UA) is one of these markers. Serum UA (SUA) level, UA, and drugs that alter SUA levels have been used to predict prognosis, mortality, and stroke severity.[5–8]

UA is the end product of exogenous and endogenous purine metabolism. The normal range for UA is considered to be 3.0–6.8 mg/dL.[9] UA is a risk factor for insulin resistance, DM, atherosclerosis, coronary artery disease, vascular dementia, pre-eclampsia, renal diseases, HTN, and metabolic syndrome.[10] Some of these conditions are also risk factors for ischemic stroke.

The effect of UA on diseases is still not clearly understood. Stroke is one of these diseases. The effects of UA and of drugs altering SUA levels on the prognosis of stroke remain unclear. Studies have indicated that both low and high SUA levels have negative effects on the prognosis.[11–14] In some studies, no statistically significant relationship was found between stroke and SUA level.[15–17]

In this study, the effect of SUA levels on the severity, morbidity, and mortality of ischemic stroke was investigated.

MATERIALS AND METHODS

The study was conducted with 820 patients who applied to the emergency department (ED) of tertiary care hospital with the complaints of acute stroke and were hospitalized between 2013 and 2018. Patient data were analyzed retrospectively. Patients with acute stroke symptoms, aged 18 years and older, and whose SUA level was studied at the time of admission to the ED were included in the study. Patients younger than 18 years, pregnant patients, those who were not admitted to the hospital within the first 48 h, those diagnosed with hemorrhagic stroke, those who had had a previous stroke, and those whose clinical course could not be evaluated were excluded from the study.

Age, sex, and known chronic diseases (HTN, DM, chronic renal failure, atrial fibrillation, cancer, and chronic heart diseases) were recorded. The National Institutes of Health Stroke Scale (NIHSS) scores and modified Rankin scale (mRS) scores were calculated at the admission and discharge. Using the NIHSS, stroke severity was classified as minor (0–4), moderate (5–15), moderate-to-severe (16–20), and severe (21–42). Using the mRS, the neurological outcomes were evaluated as good (0–2), moderate (3–4), poor (5–6), and dead (6).

SUA levels were divided into three groups with levels below 3.59 mg/dL (SUA 1), between 3.59 mg/dL and 8.5 mg/dL (SUA 2), and above 8.5 mg/dL (SUA 3).

The statistical analysis was performed using IBM SPSS ver. 21. The data were analyzed using descriptive statistics (mean, standard deviation, and median) and Pearson's Chi-square test. The confidence interval was set to 95% and the level of significance was set at 0.05.

Ethics committee approval was obtained (Protocol No: 2018/646).

RESULTS

Of the 820 patients included in the study, 348 (42.4%) were women. The mean age of the patients was 68.53 years. The SUA levels were significantly lower in women than men (p=0.0001). No statistically significant correlation was found between age and SUA level (p=0.360).

The SUA level was between 3.59 and 8.5 mg/dL in 671 (81.3%) patients, below 3. 59 mg/dL in 81 (9.88%) patients, and above 8.5 mg/dL in 68 (8.29%) patients (Table 1). SUA levels were significantly higher in patients with HTN compared to those without HTN. SUA levels were significantly higher in patients with DM compared to those without DM. SUA levels were significantly higher in patients with atrial fibrillation compared to those without atrial fibrillation (all p<0.05). However, there was no significant difference in the SUA levels in patients with cancer or coronary artery disease (p>0.05).

NIHSS scores ranged from 0 to 19. An NIHSS score of 3 was the most common (15%, n=127), while a score of 19 was the least common (0.05%, n=4). Analyzing the SUA levels according to the NIHSS scores, there were significant differences between stroke severity and the SUA

Table 1. Baseline characteristics of patients according to serum uric acid levels

	n (%)	Serum uric acid 1 (%)	Serum uric acid 2 (%)	Serum uric acid 3 (%)	p-value
Sex					
Female	348 (42.4)	30 (44.1)	55 (67.9)	263 (39.2)	0.0001**
Male	472 (57.6)	38 (55.9)	26 (32.1)	408 (60.8)	

SUA: Serum uric acid. **P<0.05.

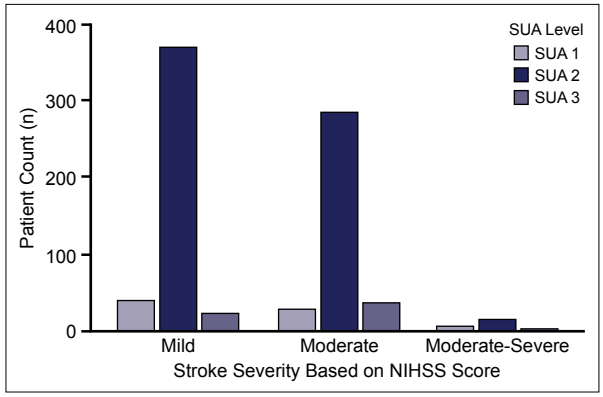

Figure 1. Relationship between SUA level and stroke severity.

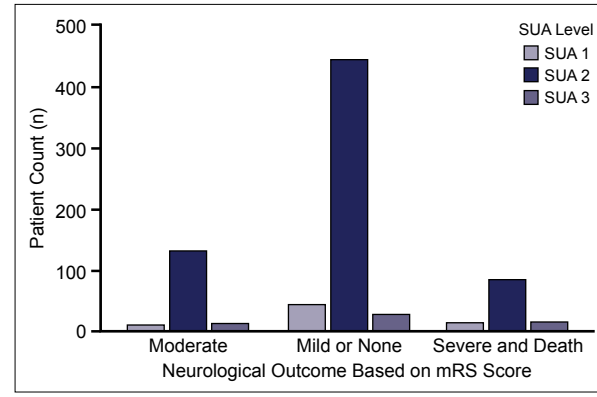

Figure 2. Relationship between SUA level and neurological outcome.

level groups (p=0.001). Moderate and moderate-to-severe strokes were more common in the group with SUA levels above 8.5 mg/dL, while moderate-to-severe strokes were more common in the group with SUA levels below 3.59 mg/dL. Minor strokes were more common in the group with SUA levels of between 3.59 and 8.5 mg/dL (Fig. 1 and Table 2).

Examining the mRS scores, the highest percentage of patients was discharged with an mRS score of 1 (28.4%, n=233), while the lowest percentage was discharged with an mRS score of 5 (5.2%, n=43). There was a significant relationship between the SUA level groups and mRS scores (p=0.002). The patients with SUA levels above 8.5 mg/dL had the worst neurological outcomes, and better neurological outcomes were observed in the group with SUA levels of between 3.59 and 8.5 mg/dL (Fig. 2 and Table 2).

The 1, 3, 6, 9, and 12 months mortality rates were significantly higher in the group with SUA levels above 8.5 mg/dL compared with the other groups (Table 3) (p<0.001). The patients with SUA levels below 3.59 mg/dL had the second highest mortality rates, while those with SUA levels of between 3.59 and 8.5 mg/dL had the lowest mortality rate. Therefore, SUA levels above 8.5 and below 3.59 mg/dL increased the mortality rates in ischemic stroke patients.

DISCUSSION

In this study, SUA level was found an independent risk factor in ischemic stroke patients. There was a positive correlation between the SUA levels in the range of 3.59–8.5

Table 2. Distribution of stroke severity and disability according to serum uric acid levels

Stroke severity	n (%)	SUA 1 (%)	SUA 2 (%)	SUA 3 (%)	p-value
(NIHSS score)					
Mild (0–4)	436 (53.2)	25 (36.8)	42 (51.9)	369 (55)	0.001**
Moderate (5–15)	355 (43.3)	39 (57.4)	31 (38.3)	285 (42.5)	
Moderate-severe (16–20)	29 (3.5)	4 (5.9)	8 (9.9)	17 (2.5)	
Disability					
(mRS Score)					
No disability-slight disability (0–2)	527 (64.3)	32 (47.1)	48 (59.3)	447 (66.6)	0.002**
Moderate disability (3–4)	168 (20.5)	17 (25)	15 (18.5)	136 (20.3)	
Severe disability and death (5–6)	125 (15.2)	19 (27.9)	18 (22.2)	88 (13.1)	

mRS: Modified Rankin scale; NIHSS: National Institutes of Health Stroke Scale; SUA: Serum uric acid. **P<0.05.

Table 3. Mortality and serum uric acid levels

Mortality	n (%)	SUA 1 (%)	SUA 2 (%)	SUA 3 (%)	p-value
1st month	71 (8.66)	19 (27.9)	8 (9.9)	44 (6.6)	0.0001**
3rd month	135 (16.5)	25 (36.8)	15 (18.5)	95 (14.2)	0.0001**
6th month	156 (19.0)	26 (38.2)	17 (21.0)	113 (16.8)	0.0001**
9th month	169 (20.6)	27 (39.7)	18 (22.2)	124 (18.5)	0.0001**
12th month	185 (22.2)	32 (47)	18 (22.2)	135 (20.1)	0.0001**

SUA: Serum uric acid. **P<0.05.

mg/dL and stroke severity, mortality, and good neurological outcomes in ischemic stroke patients.

UA is an end product of exogenous and endogenous purine metabolism. Yu et al.[10] in their review summarized the oxidant and antioxidant effects of UA. The antioxidant effects of UA are reacting with hydroxyl radicals, peroxynitrite, nitric oxide, and hydrogen peroxide to form stable intermediates, scavenging oxygen radicals with superoxide dismutase, providing chelation of metal ions, and preventing protein and lipid peroxidation and protein nitrification. The oxidant effects of UA are inhibiting nitric oxide formation and vasodilation, inducing chronic inflammatory response, inhibiting adiponectin synthesis, damaging tricarboxylic acid cycle, inhibiting fatty acid beta-oxidation, and increasing vascular smooth muscle cell proliferation. UA causes high levels of oxidative stress, although it eliminates free oxygen radicals.[18] The so-called UA paradox results from the antioxidant and pro-oxidant properties of UA.[19,20]

Different results have been obtained in the previous studies investigating the relationship between SUA level and ischemic stroke. Subramanyam et al.[11] found that low SUA level indicates good prognosis. Kawase et al.[12] found that low SUA level increased the severity of stroke. Wang et al.[14] found that SUA level above 6.22 mg/dL indicates good prognosis and SUA level below 3.98 mg/dL indicates poor prognosis. Sarfo et al.[21] found that high SUA levels increase the severity of stroke and cause poor neurological outcome. Mapoure et al.[22] found that SUA level was higher in those with 3-month mortality. On the other hand, Saadat et al.,[16] Miedema et al.,[17] and Kumar and Bandhavi[23] did not find a relationship between SUA level and stroke severity in their studies. The reason why different results were obtained in these studies may be the investigation of values below or above a certain reference value. UA has both oxidant and antioxidant effects. This may be the reason for the differences in studies.

Unlike these studies, Zhang et al.[24] found that SUA levels below 4.20 mg/dL and above 6.38 mg/dL were associated with a poor prognosis, while the best prognosis was seen with UA levels of 5.32–6.38 mg/dL. In our study, like this study, we found that in ischemic stroke, high UA levels (above 8.5 mg/dL) were associated with mortality and poor clinical outcomes due to the pro-oxidant effect of UA, and low UA levels (below 3.59 mg/dL) were associated with mortality and poor clinical outcome due to inadequate antioxidant defense.

In an experimental rats study, the administration of UA after thromboembolic stroke conferred neuroprotection, reduced infarct volume, improved neurological function, and promoted recombinant tissue plasminogen activator (rtPA).[5] Tian et al.[6] found that stroke patients given thrombolytics had higher rate of hemorrhagic transformation at low SUA levels. A double-blind placebo-controlled study with 411 patients (211 of them given UA and 200 of them given placebo) showed that the administration of UA 1000 mg in combination with alteplase within the first 4.5 h of stroke onset made a positive contribution to the prognosis compared with placebo. In the study was concluded, UA treatment could prevent early ischemic worsening and UA application could be neuroprotective.[7] In contrast to this study, in another double-blind randomized controlled study evaluating 70 ischemic stroke patients, allopurinol (200 mg/day) was given from the 1st day of stroke and the patient group given allopurinol had better neurological outcome than the group given placebo. However, there was no statistically difference in mortality between the two groups.[8] The reason for the differences between these studies may be the combination of oxidant and antioxidant effects of UA.

In this study, the best prognosis was in SUA levels of between 3.59 and 8.5 mg/dL in acute ischemic stroke patients. Keeping SUA levels within this range will contribute to reducing mortality and morbidity. Prospective multicenter studies with larger patient groups are needed to verify this.

Study limitations

The disadvantages of our study were that it was retrospective, single-centered, and relatively low number of patients with severe and poor neurological outcome.

Ethics Committee Approval

This study approved by the Haydarpaşa Numune Training and Research Hospital Clinical Research Ethics Committee (Date: 24.09.2018, Decision No: 2018/646).

Informed Consent

Retrospective study.

Peer-review

Externally peer-reviewed.

Authorship Contributions

Concept: S.Ç.; Design: B.B., B.G.Y., D.S.; Supervision: S.Ç.; Fundings: B.G.,Y., D.S., G.A.; Materials: B.B., B.G.Y., G.A.; Data: B.B., B.G.Y., D.S., G.A.; Analysis: B.B., B.G.Y., D.S.; Literature search: S.Ç., B.B., B.G.Y., D.S., G.A.; Writing: S.Ç., B.B., B.G.Y.; Critical revision: S.Ç., B.B., B.G.Y., D.S., G.A.

Conflict of Interest

None declared.

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Acil Servise Başvuran Akut İskemik İnme Hastalarında Ürik Asit Seviyesinin Mortaliteye Etkisi

Amaç: Ürik asit, serebrovasküler hastalıklar, hipertansiyon ve diyabet üzerine etkisi araştırılmış bir moleküldür. İskemik inme ve ürik asit arasındaki ilişkiyi inceleyen çalışmalarda çelişkili sonuçlar elde edilmiştir. Daha önce yapılan çalışmalarda hem yüksek hem de düşük ürik asit düzeylerinin inme hastalarında kötü prognoz ile ilişkili olduğunu bulmuştur. Bu nedenle, iskemik inmede ürik asit düzeylerinin mortalite, inme şiddeti ve klinik sonlanım üzerindeki etkilerini araştırdık.

Gereç ve Yöntem: Hastaların demografik özellikleri, kronik hastalıkları, serum ürik asit (SUA) düzeyleri, Ulusal Sağlık Enstitüsü İnme Skalası ve modifiye Rankin Skalası skorları kayıt altına alındı. Hastalar SUA düzeyleri 3.59 mg/dL'nin altında, 3.59–8.5 mg/dL arasında ve 8.5 mg/dL'nin üzerindeki hastalar olmak üzere olmak üzere üç gruba ayrıldı. Verilerin karşılaştırılmasında Pearson ki-kare testi kullanıldı.

Bulgular: Çalışmaya %42.4'ü kadın olmak üzere 820 hasta dahil edildi. Ortalama yaş 68.53 yıldı. SUA düzeyleri kadınlarda daha düşük bulundu. Orta ve orta-şiddetli inmeler, yüksek mortalite oranı, kötü nörolojik sonlanım ve yoğun bakım ve mekanik ventilasyon ihtiyacı en yaygın olarak SUA düzeyi 8.5 mg/dL'nin üzerinde olan hastalarda görüldü, bunu SUA düzeyi 3.59 mg/dL'nin altında olan hasta grubu izledi. En düşük mortalite, en iyi nörolojik sonlanım, en az yoğun bakım ve mekanik ventilasyon ihtiyacı ise bu iki SUA düzeyi arasındaki gruptaydı.

Sonuç: Ürik asit hem oksidan hem de antioksidan özelliklere sahiptir ve etkisi düzey bağımlıdır. En iyi prognoz SUA düzeyi 3.59-8.5 mg/dL arasında olan grupta görüldü. Bu nedenle iskemik inme hastalarını takip ederken ürik asit düzeylerini bu aralıkta tutmak mortalite ve morbiditeyi azaltabilir.

Anahtar Sözcükler: Acil servis; inme; mortalite; ürik asit.