The Clinical Importance of the Plasma Atherogenic Index, Other Lipid Indexes, and Urinary Sodium and Potassium Excretion in Patients with Stroke

Tuba Tulay Koca[1], Cemile Buket Tugan[2], Muhammet Seyithanoglu[3], Burhan Fatih Kocyigit[1]

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ORCID IDs of the authors:
T.T.K. 0000-0002-4596-858X
C.B.T. 0000-0001-6783-2336
M.S. 0000-0002-8027-7549
B.F.K. 0000-0002-6065-8002

[1]Department of Physical Medicine and Rehabilitation, Sütçü İmam University, Kahramanmaras, Turkey
[2]Department of Neurology, Sütçü İmam University School of Medicine, Kahramanmaras, Turkey
[3]Department of Clinic Biochemistry, Sütçü İmam University School of Medicine, Kahramanmaras, Turkey

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Correspondence to: Tuba Tulay Koca
E-mail: tuba_baglan@yahoo.com

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ABSTRACT

Objective: Cardiovascular complications are still the primary reason for high mortality rates worldwide. The determination of risk factors is important to prevent stroke. The aim of the present study was to analyze the importance of serum lipid indexes and urinary sodium (Na)/potassium (K) excretion in patients with stroke together with sex differences.

Materials and Methods: A total of 50 (28 male and 22 female, mean age 65.9±14.6 years) patients with acute stroke were included in the study group, and 32 body mass index-matched healthy subjects were included in the control group. Lipid profiles [(cholesterol, triglyceride, very low-density lipoprotein, low-density lipoprotein, and high-density lipoprotein (HDL)], serum creatinine (Cre), erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), and Na, K, and Cre excretion in spot urine samples of the patients were recorded.

Results: Systolic blood pressure (p=0.021), ESR (p=0.044), and CRP (p=0.042) were significantly higher in all patients in the stroke group; urinary Tanaka (K) (p=0.033), Kawazaki (K) (p=0.028), urinary spot Cre (p=0.012), and Na excretion (p=0.036) levels were found to be significantly lower in only male patients with stroke. The mean plasma atherogenic indexes were 0.57±0.24 in the study (stroke) group and 0.54±0.22 in the control group (p=0.61). Other lipid indexes, such as Castelli's risk index (CRI)-I (p=0.29), CRI-II (p=0.24), atherogenic coefficient (p=0.29), and non-HDL cholesterol (p=0.69), were not statistically different from the controls.

Conclusion: Urinary Na, K, and Cre excretion was significantly lower in male patients with stroke, and acute phase reactants were significantly higher in the entire stroke group than in controls. These parameters can be used as auxiliary biomarkers in the risk assessment of stroke.

Keywords: Atherosclerosis, hypertension, lipid indexes, plasma atherogenic index, stroke

Introduction

Owing to the aging population in the developed world, aging is a growing worldwide problem that has been reported to reduce the quality of life with high morbidity and mortality. Progression cannot be remediated; therefore, optimal management of aging should include an assessment of risk factors related with morbidity and mortality. Cardiovascular (CV) complications are the most important cause of mortality and morbidity worldwide. Stroke is the third most common cause of mortality and one of the main causes of physical disability of adults. It is caused by blocking of blood vessels that carry oxygen and nutrients to the brain. As a result, the brain needs blood, it cannot receive oxygen, and brain cells die [1]. Therefore, a good identification of risk factors for CV complications and prior knowledge of high-risk individuals are important for primary prevention. Biomarkers are needed to better define risk factors and guide us in clinical practice. A biomarker comprises small samples from the patient, such as blood, urine, and saliva, obtained through non-invasive techniques. The use of biomarkers in clinical practice is becoming more widespread and provides valuable information [2, 3].

Various lipid indexes show the risk of mortality rather than lipid levels in advanced age populations. Additionally, serum lipid abnormalities are considered as a risk factor for stroke development. However, a few studies reported some inconsistencies in the relationship between lipid profiles and the risk of stroke [3, 4]. The plasma atherogenic index (PAI) emerged as a valuable marker in determining the risk of CV diseases [3]. Recently, high levels of sodium (Na) and low levels of potassium (K) intake were shown to be associated with high blood pressure and

increased CV complications. The structure and level of this relationship varied according to the characteristics of the participants and the way in which the acquired samples were assessed [5]. The aim of the present study was to analyze the clinical significance of PAI and other lipid indexes [Castelli's risk index (CRI) I and II, atherogenic coefficient (AC), non-HDL cholesterol (NHC)] and 24-hour urinary Na and K excretion in patients with stroke.

Materials and Methods

This was a case-control study. A total of 50 (28 male and 22 female) patients with acute stroke comprised the study group, and 32 (13 male and 19 female) body mass index (BMI)-matched healthy subjects were included in the control group. The age, height, weight, existence of hypertension, use of antihypertensive medication, systolic/diastolic blood pressure, plasma lipids [cholesterol, triglyceride (TG), very low-density lipoprotein (VLDL), low-density lipoprotein (LDL), and high-density lipoprotein (HDL)], serum creatinine (Cre), erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), and Na, K, and Cre excretion in spot urine samples of the participants were recorded. Biochemical analysis was done by the device, ADVIA 1800 Clinical Chemistry System, Siemens, Erlangen, Germany. The lipids, lipid indexes, and other biochemical parameters of the patients in the study and control groups were examined using appropriate statistical tests.

PAI, which is a lipid index, is measured as the ratio of the logarithmic value of TG to high-density lipoprotein cholesterol (HDLc). The following formulas were used for other lipid indexes:

CRI-I: total cholesterol/HDLc,

CRI-II: low-density lipoprotein cholesterol (LDLc)/HDLc,

AC: (total cholesterol−HDLc)/HDLc,

Non-HDLc: total cholesterol−HDL [3].

The formulas developed by Kawasaki et al. [6] and Tanaka et al. [7] were used to predict the mean urinary Na and K excretion levels in the spot urine samples obtained in 24 h. The 24-hour urinary Na and K excretion was not affected by recall errors and represented all sources of uptake [6, 7].

Patients with a history of chronic renal failure, hepatic dysfunction, atrial fibrillation, congestive cardiac failure, acute or chronic infection, chronic obstructive pulmonary disease, and inflammatory rheumatologic diseases were excluded from the study.

Table 1 . Comparison of demographic and laboratory data for the groups

	Stroke (N=50) Mean±SD/median (min-max)	Control (N=32) Mean±SD/median (min-max)	p
Age (year)	65.9±14.6	60.9±14.1	0.13
Gender (M/F)	28/22	13/19	0.17
Systolic tension* (mm Hg)	131.5±24 (↑)	120.6±11.5	0.021
Diastolic tension (mm Hg)	75 (59-90)	80 (60-90)	0.208
Triglycerides (mg/dL)	164.6±82.1	161.3±72.9	0.85
LDL (mg/dL)	145.9±163.1	123.4±28.2	0.45
HDL (mg/dL)	39.4±8.6	42.6±9.5	0.14
VLDL (mg/dL)	26 (13-75)	31.4 (12-59)	0.601
Total cholesterol (mg/dL)	176.4±39.8	187.5±39.9	0.23
PAI	0.57±0.24	0.54±0.22	0.61
CRI-I	4.7±1.3	4.4±0.91	0.29
CRI-II	4.3±6.5	2.9±0.72	0.24
AC	3.7±36.6	3.4±0.91	0.29
NHC	141.4±36.6	144.8±36.1	0.69
BMI (kg/m2)	29.2±5.5	28.8±4.2	0.72
Urinary Na/K ratio	2.9±2.7	2±1.3	0.103
ESR (mm/h)*	22.9±15.1 (↑)	17.3±8.9	0.044
CRP (mg/dL)*	5.8 (1.3-177) (↑)	3.1 (2-28)	0.042

LDL: low-density lipoprotein; HDL: high-density lipoprotein; VLDL: very low-density lipoprotein; PAI: plasma atherogenic index; CRI: Castelli's risk index; AC: atherogenic coefficient; NHC: non-HDL cholesterol; BMI: body mass index; ESR: erythrocyte sedimentation rate; CRP: C-reactive protein.
Lipid measurements were given in mg/dL.
*Statistically different, p<0.05.

Statistical Analysis

All statistical analyses were performed by using the IBM SPSS version 19 statistics software package (IBM Corp., Armonk, NY, USA). Descriptive data are presented as mean±standard deviation (SD) or median scores according to their categories and distribution. The Kolmogorov-Smirnov test was used to analyze the coherence of variables to normal contribution (normality) because the number of patients in the study group was >30. Histograms were also used for evaluation of normal distribution. Spearman's correlation analysis was used to analyze the level of the correlation between the variables.

Categorical data are presented as percentages (%). The chi-square test was used for comparison of categorical data. Continuous data are presented as mean with SD or median with minimum and maximum. Continuous data were compared using parametric/non-parametric tests according to their normal or non-normal distribution. The Student's t-test and Mann Whitney U test were used for comparison of obtained values. A p<0.05 was considered as statistically significant. The study was approved by the Regional Committee for Ethics (date: 01/172018; approval no.: 06/protocol no.: 08) in accordance with the criteria of the Medical Research and Declaration of Helsinki. Informed consent was obtained from all participants.

Results

The study group consisted of 50 (28 male and 22 female) patients. The mean age of the study group was 65.9±14.6 years. The control group included 32 (13 male and 19 female) subjects. The mean age of the control group was 60.9±14.1 years. Both groups were similar with regard to sex (p=0.17), age (p=0.13), and BMI (p=0.72). The demographic and laboratory data of both groups are summarized in Table 1.

Overall, 52% of the study group and 46.9% of the control group were using antihypertensive medication (p=0.59) (Table 2). The Na and K excretion levels in the spot and 24-hour urine samples in male patients are given in Table 3. Systolic blood pressure (p=0.021), ESR (p=0.044), and CRP (p=0.042) were significantly higher in all patients in the stroke group; urinary Tanaka (K) (p=0.033), urinary Kawasaki (K) (p=0.028), Cre (p=0.012),

Table 2. The use of antihypertensive medications according to the groups

	Stroke group (N=50/%)	Control group (N=32/%)	p
ACE inhibitor	17/34	10/31.3	0.762
Calcium channel blocker	11/22	3/9.4	0.142
Beta blocker	4/8	4/12.5	0.509
Diuretic	8/16	7/21.9	0.508
Others (alpha blocker, etc.)	2/4	1/3.1	0.839
*p=0.59			

*Chi-square test: statistically different, p<0.05.

Table 3. Comparison of urine data for the groups in male gender

	Stroke (N=28) Mean±SD/median (min-max)	Control (N=13) Mean±SD/median (min-max)	p
Kawazaki (Na), mEq/L	174.98±11.2	139.1±85.5	0.56
Tanaka (K)*, mEq/L	67.8±24.3 (↓)	83.3±83.9	0.028
Tanaka (Na), mEq/L	179.57±117.74	153.2±118	0.67
Kawazaki (K)*, mEq/L	57.7±28.3 (↓)	76±38.1	0.033
Urinary spot Na*, mEq/L	72.2±47 (↓)	91.2±45.7	0.036
Urinary spot K, mEq/L	67.2±52.9	84.2±48.7	0.17
Urinary Na/K ratio	3.8±3.3	2±0.9	0.08
Urinary spot Cre*, g	102±71.2 (↓)	126±69.8	0.012
Serum Cre, mg/dL	0.9 (0.5–0.7)	0.8 (0.4–1.8)	0.544

*Independent samples t-test: statistically different, p<0.05

and Na excretion in the spot urine (p=0.036) were significantly low in only male patients in the study group. The urinary Na/K ratios were 2.9±2.7 in the study group and 2.0±1.3 in the control group (p=0.103). Moreover, the urinary Na (Kawasaki) was positively correlated with systolic blood pressure (p=0.001; r=0.381).

The mean PAIs were 0.57±0.24 in the study group and 0.54±0.22 in the control group (p=0.61). The PAI was positively correlated with urinary K excretion (Kawasaki) (p=0.014; r=0.315). ESR was significantly high in female patients in the hypertensive (HT) group (p=0.008). Lipid indexes, such as CRI-I (p=0.29), CRI-II (p=0.24), AC (p=0.29), and NHC (p=0.69), were not statistically different from the controls in patients with stroke (Table 1).

When we divided all the groups with regard to sex, female patients had higher BMI (p<0.001), HDL (p=0.017), and VLDL (p=0.022), and male patients had higher CRI-I (p=0.034), AC (p=0.034), Kawasaki Na (p=0.026), urinary spot Na (p=0.001), urinary Na/K ratio (p=0.018), and urinary spot Cre (p=0.008) levels (Table 4).

Discussion

The study and control groups were the same with regard to age, sex, and BMI. According to the results, all lipid indexes including PAI, CRI-I, CRI-II, AC, and NHC were the same as the controls in patients with stroke. Acute phase reactants, which show inflammation and systolic tension, were higher in patients with stroke than in controls. Urinary K, Na, and Cre excretion was found to be lower in male patients with stroke than in controls. There was no difference with regard to antihypertension medications. We also find differences in some lipid indexes (HDL, CRI-I, and AC against male sex), urinary Na and Cre excretion, and urinary Na/K ratio with regard to sex.

Stroke is the leading cause of inevitable deaths. The high mortality and morbidity rates of this disease can be precluded by knowing the possible risk factors that lead to this disease. Age, sex, race, and family history of stroke development are modifiable risk factors, and hypertension, diabetes, CV diseases, smoking, alcohol, obesity, dyslipidemia, physical inactivity, inflammation, hypercoagulability, and medication are unmodifiable risk factors [3-5, 8, 9].

Concentrations of lipid biomarkers in circulation have been found to be associated with CV disease risk [10, 11]. In particular, intraindividual variabilities in TG, LDLc, and HDLc levels should be considered [12]. Sujatha et al. [3] suggested that lipid parameters and indexes are significantly higher in patients with stroke. Uslu et al. [5] found that PAI is an independent risk factor in the diagnosis of atherosclerosis in patients with systemic lupus erythematosus. In the present study, no significant difference was found with regard to lipid indexes and PAI between the stroke and control groups. Bendzala et al. [11] revealed that PAI is positively correlated with age, comorbidity, statin use, and mortality risk regardless of smoking in patients with hypertension aged >60 years. Cai et al. [13] suggested that PAI can be a strong marker for predicting risk for CV diseases. Ersoy et al. [14] found that HDL, LDL, and PAI levels are negative factors for osteoporosis. Individuals with low physical activity were found to have high PAI values, causing an accelerated atherogenic process and CV diseases in the long term [15]. Gunay et al. [16] found that atherogenic indices (PAI, cardiogenic risk ratio, and AC) can be considered as a useful predictor for atherosclerosis and CV diseases in stable patients with chronic obstructive pulmonary diseases.

Hypertension control in patients with stroke was a key factor for preventing the disease [17]. Blood pressure control is important after stroke [18, 19]. In the present study, the prevalence of hypertension was found to be similar in both groups, but systolic blood pressure was significantly higher in the stroke group. Thrombosis is the most common cause of mortality in various CV events. Oxidized lipoproteins were the most common triggering factors in chronic inflammatory diseases associated with a high risk of thrombosis. Thromboinflammation can affect venous thrombosis [20]. In patients with stroke after cerebral ischemia, the presence of inflammatory cells (macrophages, neutrophils, and monocytes) at the site of injury supports the role of inflammation in the pathogenesis of stroke. With developed techniques, many inflammatory mediators, such as cytokines, chemokines, leukocytes, and adhesion molecules, have been shown in ischemic tissue and the environment. In ischemic brain injury, inflammation is important both in disease pathogenesis and in clinical course and prognosis [21]. In the present study, the inflammation parameters were found to be significantly higher in the stroke group, supporting this hypothesis.

The relationship between Na and K intake is still unclear in patients with CV diseases. The levels of Na and K intake in different populations are not known clearly. However, long-term studies using 24-hour urine samples and accurate estimation of daily K intake are scarce. The urinary excretion of >7 g of Na/day increases the risk

Table 4. Comparison of demographic and laboratory data according to gender in all groups

	Male (N=41)	Female (N=41)	p
Age (year)	66.9±15.3	61.1±13.2	0.076
Systolic tension	127.7±23	127±18.6	0.876
Diastolic tension	74.2±8.4	76.2±7.9	0.259
Triglycerides	129.5 (59–373)	159 (75–324)	0.47
LDL	124 (44–873)	128 (60–899)	0.935
HDL*	38.1±8	43.1±9.5 (↑)	0.017
VLDL*	25 (12–75)	33 (15–65) (↑)	0.022
Total cholesterol	178.8±38.4	182.6±41.8	0.678
PAI	0.54±0.24	0.58±0.21	0.453
CRI-I*	4.9±1.3 (↑)	4.3±1.0	0.034
CRI-II	4.3±6.5	3.2±3.0	0.357
AC*	3.9±1.3 (↑)	3.3±1.0	0.034
NHC	144.6±34	141.2±38.5	0.698
BMI (kg/m^2)*	26.9±3.6	31.2±5.3 (↑)	0.000
ESR (mm/h)	20.6±3.6	20.6±12.8	0.995
CRP (mg/dL)	3.6 (1.3–177)	4.3 (2–135)	0.824
Kawazaki (Na), mEq/L*	171.8±110.4 (↑)	121.8±65.4	0.026
Tanaka (K), mEq/L	52.5 (32.7–194.6)	123 (25.9–361.4)	0.062
Tanaka (Na), mEq/L	119.1 (29–499)	144.5±83.2	0.256
Kawazaki (K), mEq/L	57.7 (32.4–146.8)	53.9 (32.4–190.5)	0.961
Urinary spot Na, mEq/L*	98.4±51.5 (↑)	61.1±33.8	0.001
Urinary spot K, mEq/L	76.5±49.7	71.8±54.1	0.700
Urinary Na/K ratio*	3.2±2.8 (↑)	1.9±1.2	0.018
Urinary spot Cre*, g	133.4±77.9 (↑)	89±55.2	0.008
Serum Cre, mg/dL	0.94±0.21	0.95±1.1	0.942

LDL: low-density lipoprotein; HDL: high-density lipoprotein; VLDL: very low-density lipoprotein; PAI: plasma atherogenic index; CRI: Castelli's risk index; AC: atherogenic coefficient; NHC: non-HDL cholesterol; BMI: body mass index; ESR: erythrocyte sedimentation rate; CRP: C-reactive protein. Lipid measurements were given in mg/dL.
*Statistically different, $p<0.05$.

for all patients with CV disease. Excretion of <3 g/day of Na was also associated with increased CV disease mortality. Moreover, high K excretion was associated with decreased stroke risk [22]. Compatible with the literature, in our study, urinary Na and K excretion was found to be lower in male patients with stroke than in controls. Increased dietary Na and Na/K ratios were associated with increased blood pressure and prehypertension. In addition, diet was seen as a preventive method to inhibit unnecessary Na intake [23]. The Na/K ratios were similar in the groups independent of sex, and no relationship between systolic or diastolic tension and urinary Na/K ratio was found.

Prentice et al. [19] found a positive correlation between HT incidence and Na intake and urinary Na/K ratio in 40 postmenopausal women. They also found a positive correlation between Na/K ratio and CV diseases, such ischemic stroke, and an inverse correlation with hemorrhagic stroke. A poor correlation was found between observational studies of dietary K intake and CV disease risk. We did not investigate dietary intake. Kieneker et al. [24] found that urinary K excretion is not an independent risk factor for CV disease including stroke. Mentee et al. [25] found that urinary K excretion in older patients with hypertension is negatively associated with systolic blood pressure. Jackson et al. [8] found a strong positive correlation of hypertension with urinary Na excretion and a negative correlation with K excretion. We found a positive correlation between urinary Na excretion and systolic blood pressure. Therefore, free radicals were thought to increase K excretion in patients with acute stroke, and K deficiency caused various undesirable cardiac reactions [26]. The present study also found that urinary K and Na excretion was significantly lower in male patients with stroke than in controls. When we divided all the groups according to gender, although the BMI of the female patients was higher, some lipids, urinary Na and Cre excretion, and urinary Na/K ratio were higher in male patients. This result supports the high risk of atherosclerotic diseases in men. Sex differences should be considered separately when evaluating CV risk factors.

Our study has some limitations. Patients with stroke in all etiologies (ischemic, hemorrhagic, and subarachnoid hemorrhage) were included in the present study. Different etiologies may have affected the laboratory outcomes of the patients. The fact that the study data belonged to one local center limits the generalization of the results. When we calculated the laboratory data, these patients were in acute period stroke, and medical treatment had already started. These treatments may affect urine results. Although there were no differences in the medications taken, it would be more useful to obtain laboratory data before a stroke event occurs.

Some practical biomarkers are needed to determine the risk of stroke in the elderly. We found that urinary Na, K, and Cre excretion was low in only male patients with stroke, and that inflammation parameters were higher in the entire stroke group than in controls. Additionally, the lipid indexes of patients with stroke were the same as the controls. When we divided all the groups according to sex, although female patients had higher BMIs than male patients, some lipid indexes (CRI-I and AC), urinary Na and Cre excretion, and Na/K ratio were higher in male patients.

Ethics Committee Approval: Ethics committee approval was received for this study from the ethics committee of Kahramanmaraş Sütçü İmam University School of Medicine.

Informed Consent: An informed consent form was taken from the participants.

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