CORRELATION BETWEEN SERUM URIC ACID LEVEL AND BLOOD PRESSURE, BLOOD LIPID, BLOOD GLUCOSE AND CRP IN PATIENTS WITH ACUTE CEREBRAL INFARCTION

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ABSTRACT

Objective: *This study aimed to analyze the correlation between serum uric acid (SUA) level and blood pressure, blood lipids, blood glucose, and C-reactive protein (CRP) in patients with acute cerebral infarction.*

Methods: *We selected 113 patients with cerebral infarction admitted to the Department of Neurology of our hospital from December 2017 to December 2020 as the observation group, while 65 healthy people who came to our hospital for a normal physical examination in the same time frame were selected as the control group. SUA, total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and systolic blood pressure (SBP) were detected by automatic biochemical analyzer. Changes in SBP, DBP, FBG, and CRP were analyzed using Pearson's correlation.*

Results: *The levels of TC, TG, LDL-C, SBP, DBP, FBG, SUA, and CRP in the observation group were significantly higher than those in the control group, while the observation group's HDL-C level was significantly lower than that in the control group (P<0.05). The SUA level in members of the moderate and severe groups was significantly higher than that of the mild group (P<0.05); moreover, the value for the severe group was significantly higher than that of the moderate group (P<0.05). The levels of TC, TG, LDL-C, SBP, DBP, FBG, and CRP in hyperuricemia patients were significantly higher than those in the control group, while the former's HDL-C levels were significantly lower than those in the non-hyperuricemia patients (P<0.05). Pearson correlation analysis showed that SUA was positively correlated with TC, TG, LDL-C, SBP, DBP, FBG, and CRP levels but negatively correlated with HDL-C (P<0.05).*

Conclusion: *An increase in SUA level is closely related to the incidence of acute cerebral infarction. Moreover, the level is related to the severity of cerebral infarction, blood pressure, blood lipids, blood glucose, and CRP levels. Thus, controlling these risk factors for cardiovascular and cerebrovascular diseases is vital for the clinical treatment of cerebral infarction.*

Keywords: *Acute cerebral infarction, blood uric acid, blood pressure, blood lipids, blood glucose, CRP, correlation.*

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Introduction

Cerebral infarction, also known as ischemic stroke, is a dangerous disease, causing disability and death in humans. According to epidemiological data, this malady accounts for 67.5% of all cerebrovascular diseases. Researchers have identified a variety of causes of cerebral infarction, which involves an obstruction of the regional blood supply in the brain tissue, resulting in stenosis or even occlusion of the cerebral arterial lumen and leading to ischemia and hypoxia of the brain tissue and necrosis[1]. Some patients with cerebral infarction experience dizziness, temporary limb numbness, weakness, and other manifestations, most of which occur during rest or sleep. Incidence rates have increased annually, imposing a heavy burden on society as a whole as well as patients' families[2]. Currently, the identified risk factors for cerebral infarction include high blood pressure, elevated blood lipids, diabetes, smoking, and other factors. In recent years, some researchers have found atherosclerotic disease to be closely related to hyperuricemia[3]. Serum uric acid (SUA) is the final product of human purine

nucleotide metabolism. It exists in cells and all body fluids and can remove hydroxyl groups, hydrogen peroxide, and perssad produced by the degradation of peroxynitrite, etc., protecting the function of blood vessels and endothelial cells[4].

The occurrence of a cerebral infarction leads to a significant decrease in the level of SUA in patients. An associated decrease in the body's antioxidant capacity is related to the scope of the nerve damage in patients. Relevant data show that hyperuricemia is a common complication of hypertension, and SUA has also been found to be associated with insulin resistance. Clinical experimental studies have demonstrated that hyperlipidemia is often complicated by dyslipidemia[5]. Therefore, this study aimed to analyze the correlation between SUA and blood pressure, blood lipids, blood glucose, and CRP in patients with acute cerebral infarction.

Methods

General information

For this study, we selected 113 patients with cerebral infarction admitted to our hospital's Department of Neurology from December 2017 to December 2020 as the observation group. This study was approved by the hospital ethics committee.

The inclusion criteria for all study participants were as follows:

• Must meet the diagnostic criteria for cerebral infarction established by the Fourth National Cerebrovascular Disease of the Chinese Medical Association[6];

• Diagnosed by imaging examinations such as head CT or MRI;

• Stable vital signs;

• Disease course did not exceed 1 week at the time of admission for patients with acute cerebral infarction, and complete clinical relevant medical records.

Furthermore, patients and their families were informed of the study details and agreed to participate in this study.

The following exclusion criteria were applied:

• Multiple episodes of cerebral infarction;

• Neural impairment time ≤24h;

• Communication and cognitive impairment;

• Recent infection, fever, pregnancy and other diseases;

• Cerebral infarction secondary to cerebral hemorrhage, trauma, or cerebral infarction after hematopathy;

• Presence of subarachnoid hemorrhage and affected by SUA drugs.

Among the included patients, 54 were male and 59 were female. The participants ranged in age from 41 to 92 years old, with an average age of (73.91±3.47) years. The time from onset to admission was 1~24h, with an average of (8.34±1.25) hours.

There were 17 cases diagnosed with acute cerebral infarction on the right side, 18 cases with cerebral infarction on the left side, and 78 other cases. In terms of comorbidities, the largest number of cases (69) also revealed hypertension, while other cases involved diabetes (19), coronary heart disease (25), or atrial fibrillation (14). According to neurological impairment scores, 48 cases were divided into the mild group, with a score from 0 to 15, while 36 cases were divided into the moderate group, scoring from 16 to 30, and 29 cases were divided into the severe group, with scores from 31 to 45. For our control group, we selected 65 healthy people who came to our hospital for a routine physical examination during the same period. The control group included 37 males and 28 females who were aged 40 to 90 years old, with an average age of (72.84±3.25) years.

The results of statistical testing revealed no statistically significant difference in the age or gender of subjects between the two groups (P>0.05). According to the diagnostic criteria for hyperuricemia (male SUA levels >417μmol/L; females >357μmol/L), subjects in the two groups were divided into a hyperuricemia group (22 cases) and a non-hyperuricemia group (156 cases).

Method

In the patients with cerebral infarction, 5 ml of fasting venous blood was drawn in the morning within 24 hours of admission. In contrast, venous blood was collected from the participants in the control group during their physical examinations. The supernatant was taken after centrifugation. Changes in SUA, total cholesterol (TC), triglycine (TG), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL), lipoprotein cholesterol (HDL-C), systolic pressure (SBP), diastolic pressure (DBP), fasting blood glucose (FBG), and C-reactive protein (CRP) were detected using an automatic biochemical analyzer.

Statistical methods

The study data were analyzed using the SPSS version 21.0 software package, and the measurement data are all expressed using ($\bar{x}\pm s$). The data from the

observation and control groups were compared by t test. Data were compared between multiple groups using variance analysis, and all enumeration data were expressed as (n(%)). Data between the two groups were also compared using a chi-squared test. The correlation between the indicators was analyzed by Pearson correlation analysis. P<0.05 was regarded as statistical difference.

Results

Comparison of SUA, blood pressure, blood lipids, blood glucose, and CRP levels between patients with cerebral infarction and normal population

The levels of TC, TG, LDL-C, SBP, DBP, FBG, SUA, and CRP in the observation group were significantly higher than those in the control group, and the levels of HDL-C were significantly lower than those in the control group.

The difference was statistically significant (P<0.05). See Tables 1 and 2.

Group	TC (mmol/L)	TG (mmol/L)	LDL-C (mmol/L)	HDL-C (mmol/L)
Observation	6.19±1.10	1.67±0.89	2.41±0.57	1.08±0.21
Control	4.76±1.01	1.06±0.46	1.92±0.70	1.21±0.24
t	7.720	5.516	2.322	3.287
P	<0.001	<0.001	0.022	0.001

Table 1: Comparison of blood lipid levels between patients with cerebral infarction and normal individuals.

Group	Case	SBP (mmHg)	DBP (mmHg)	FBG (mmol/L)	SUA (μmol/L)	CRP (mg/L)
Observation	113	154.37±21.74	87.48±16.65	6.44±1.58	339.24±70.74	4.92±1.23
Control	65	131.02±12.15	78.23±9.41	5.60±1.35	313.25±52.54	3.14±1.17
t		5.600	5.860	3.963	2.378	4.970
P		<0.001	<0.001	0.001	0.019	<0.001

Table 2: Comparison of SUA, blood pressure, blood glucose, and CRP levels between patients with cerebral infarction and normal population.

Comparison of SUA levels in patients with cerebral infarction at different severity levels

The SUA level of patients in the moderate and severe groups was significantly higher than that of the mild group, and the difference was statistically significant (P<0.05).

The SUA level of patients in the severe group was significantly higher than that of the moderate group, and the difference was statistically significant (P<0.05); see Table 3.

Group	Case	SUA (μmol/L)
The mild group	48	316.12±60.21
The moderate group	36	362.02±70.74*
The severe group	29	427.01±101.02*#

Table 3: Comparison of SUA levels in patients with cerebral infarction in different severity.
*Note: Compared with mild group *P<0.05; compared with moderate group #P<0.05.*

Comparison of blood pressure, blood lipids, blood glucose, and CRP levels in patients with hyperuricemia and non-hyperuricemia

The levels of TC, TG, LDL-C, SBP, DBP, FBG, and CRP in patients with hyperuricemia were significantly higher than those in the control group. In contrast, the levels of HDL-C were significantly lower than those in non-hyperuricemia patients, revealing a statistically significant difference (P<0.05)). See Tables 4 and 5.

Group	TC (mmol/L)	TG (mmol/L)	LDL-C (mmol/L)	HDL-C (mmol/L)
Hyperuricemia	5.87±1.30	2.63±0.84	3.35±0.62	1.02±0.18
Non-hyperuricemia	5.01±1.20	2.05±0.78	3.01±0.58	1.20±0.23
t	2.805	2.913	2.299	3.174
P	0.006	0.004	0.023	0.002

Table 4: Comparison of blood lipid levels between hyperuricemia and non-hyperuricemia patients.

Group	Case	SBP (mmHg)	DBP (mmHg)	FBG (mmol/L)	CRP (mg/L)
Hyperuricemia	22	147.02±21.05	90.10±17.65	7.06±2.67	5.53±1.41
Non-hyperuricemia	156	134.25±18.13	82.23±14.52	6.02±1.52	4.40±1.84
t		2.728	2.083	2.411	2.495
P		0.007	0.039	0.017	0.014

Table 5: Comparison of blood pressure, blood glucose, and CRP levels between hyperuricemia and non-hyperuricemia patients.

Correlation analysis

Pearson correlation analysis results showed that SUA was significantly positively correlated with TC, TG, LDL-C, SBP, DBP, FBG, and CRP levels, and was significantly negatively correlated with HDL-C levels (P<0.05), as shown in Table 6.

	r	P
TC	0.533	0.001
TG	0.321	0.015
LDL-C	0.513	0.004
HDL-C	-0.563	0.002
SBP	0.156	<0.001
DBP	0.070	<0.001
FBG	0.438	0.015
CRP	0.568	0.023

Table 6: Correlation analysis of various indicators.

Discussion

Acute cerebral infarction is a common, frequently-occurring disease in neurology. The aging of the population and changes in people's lifestyles have increased the possibility for this disease to seriously endanger human health. Cerebral infarctions, 80% of which are ischemic cerebral infarctions, are currently the greatest cause of death in China and feature relatively high morbidity, recurrence rate, and disability rate[7]. At present, no effective treatment method is available, making prevention particularly important. Thus, understanding the risk factors of cerebral infarction is necessary for effective prevention. These risk factors can be divided into two categories, depending on whether they are susceptible to intervention. Non-intervention factors include age, gender, genetics, etc. Meanwhile, factors that can be affected by intervention include high blood pressure, diabetes, high blood lipids, high CRP, etc. Successful, effective prevention relies on discovering and controlling all important risk factors of cerebral infarction to be able to significantly reduce its incidence[8]. For example, in recent years, the prevalence of hyperuricemia has increased significantly, and its relationship with cardiovascular and cerebrovascular diseases has attracted wide attention from clinicians.

SUA is the final product of exogenous and endogenous purine metabolism in the human body. The average uric acid pool in a normal human body is 1200 mg. In the body, 70% of the uric acid is excreted through the kidneys, and the rest is excreted through the biliary tract and intestinal tract. The level of SUA in the body is primarily affected in two ways: The intake of a high porphyrin or protein diet increases uric acid production, while a decreased glomerular filtration rate and an increase in renal tubular reabsorption leads to decreased secretion of uric acid. The regulatory disorder related to these two aspects can lead to an increase in the plasma uric acid level[9]. Some researchers have found that reduced renal blood flow in patients with hypertension can lead to an increase in plasma uric acid levels; meanwhile, hypertension can induce microvascular disease. ATP, which produces adenine and xanthine during ischemia, increases the content of xanthine oxidase while also significantly increasing the level of uric acid[10]. The current understanding of the possible mechanism by which SUA causes cerebral infarction involves damage to the vascular endothelial function, promoting platelet adhesion and inducing thrombosis, increasing oxidative stress, promoting proliferation, and promoting inflammation. Vascular endothelial function plays a key role in maintaining cardiovascular homeostasis. Although SUA is an antioxidant, high uric acid levels can stimulate the adhesion of white blood cells to endothelial cells, thereby damaging vascular endothelial function[11]. SUA promotes proliferation and inflammation through organic anion transporters entering smooth muscle cells, regulating intracellular redox, and activating mitogen-activated protein kinase, cyclooxygenase-2, and platelet-derived growth factor[12]. Numerous studies have found SUA to be associated with many traditional cardiovascular factors, such as hypertension, hyperglycemia, and hyperlipidemia[13-14]. Some foreign scholars have reported a significant increase in the level of SUA in patients with ischemic stroke, although the adverse effect on the prognosis of patients was independent of the severity of the stroke and other prognostic factors[15].

The results of this study showed that the SUA level in patients with cerebral infarction was significantly higher than that of the normal population. Comparison within the observation group revealed that the SUA levels of patients with a moderate or severe cerebral infarction were significantly higher than those characterizing the mild group ($P<0.05$). Moreover, the SUA level in patients with severe cerebral infarction was significantly higher than that of the moderate group ($P<0.05$), suggesting that SUA is highly expressed in patients with cerebral infarction and is significantly related to the severity of the disease.

Further analysis showed that the levels of TC, TG, LDL-C, SBP, DBP, FBG, and CRP in patients with hyperuricemia were significantly higher than those in the control group, while the levels of HDL-C were significantly lower than those in non-hyperuricemia patients, suggesting that the increased level of each index is the aggravating factor of acute cerebral infarction. Pearson correlation analysis showed that SUA was significantly positively correlated with TC, TG, LDL-C, SBP, DBP, FBG, and CRP levels and was significantly negatively correlated with HDL-C levels ($P<0.05$). Clearly, SUA and TC, TG, LDL-C, SBP, DBP, FBG, and CRP levels are significantly negatively correlated ($P<0.05$), thus clarifying the relationship between SUA and the various factors and further providing a method for controlling the demonstrable risk factors of cerebrovascular disease.

In summary, an increase in blood uric acid levels is closely correlated with the onset of acute cerebral infarction. Moreover, this increase is correlated with the severity of the cerebral infarction, blood pressure, blood lipids, blood glucose, and CRP levels. Thus, controlling these risk factors related to cardiovascular and cerebrovascular diseases is of great significance for the clinical treatment of cerebral infarction.

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