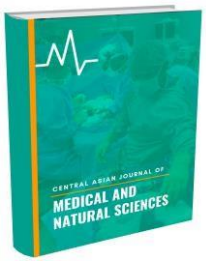

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Article

The Importance of Uric Acid in the Development of Cardiovascular Diseases

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Abstract: Cardiovascular disease (CVD) is a common name for diseases of the heart and blood vessels. Currently, these diseases are one of the main causes of death and disability worldwide. Currently, several studies have emphasized hyperuricemia as an independent risk factor for CKD [1]. According to the literature, hyperuricemia occurs in 2% of the US population, 17% in France, 7% in Spain, and 19.3% in Russia [2, 3]. This article provides information on the importance of uric acid in the development of cardiovascular diseases.

Keywords: comorbid conditions, cardiovascular diseases, anxiety-depressive disorders, uric acid, obesity, metabolic syndrome

1. Introduction

Changes in the amount of uric acid (UC) occupy the main place among the many factors affecting the MS, ENT system. Hyperuricemia has been proven in many studies to be the main predictor of the origin and complications of CKD, chronic and acute heart failure (CHF), AG and MS [4], [5], [6]. Gertler et al, in their mid-19th century studies, found that SK levels were associated with MI. Since then, many epidemiologic studies have proven that an increase in the amount of SK increases the risk of not only coronary diseases in the general population, but also complications in hypertensive patients [6].

Several studies have shown that increasing the amount of SC increases the oxygenation of low-density lipoproteins, leading to the formation of lipid peroxygenation. An increase in the amount of SK is associated with an increase in the production of free oxygen radicals. Oxidant stress and increased oxygenation of low-density lipoproteins lead to the development of atherosclerosis in the vessel wall. SK is involved in platelet aggregation and adhesion. These factors suggest an increased risk of coronary thrombosis in patients with hyperuricemic coronary disease [7].

SC is thought to reflect endothelial damage [8]. Endothelial dysfunction is manifested by the decrease in endothelium-dependent vascular relaxation under the influence of NO and the development of atherosclerosis in patients with QD and AG [8].

In endothelial cells, xanthine oxide serves as a generator of free oxygen radicals. Increased amounts of SC and xanthine oxide are more common in vessels damaged by atherosclerosis compared to undamaged vascular tissues. This suggests that SK plays a direct role in the development of atherosclerosis [9].

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The reason for the development of AG against the background of persistent hyperuricemia is related to preglomerular arteriopathy and tubulointerstitial injury. An increase in the amount of SK increases the activation of the renin-angiotensin system and worsens endothelial dysfunction. Endothelial dysfunction occurs due to a decrease in NO synthesis in hyperuricemia. This feature of SK shows its prooxidant appearance in conditions of metabolic changes [10].

When patients with type 2 QD were examined, an increase in the incidence of stroke was found to be associated with an increase in the amount of SK. This relationship has been shown to be superior to other cardiovascular risk factors [3].

Questions such as whether a high level of SK increases the risk of developing coronary diseases or is a marker of vascular degenerative diseases, or whether it is overestimated in cardiovascular diseases, have not been fully answered. Therefore, the amount of SK is AG, QD.

Type 2, insulin resistance, obesity, and vascular disease are pressing issues and suggest further study is warranted.

The purpose of this work is to study the importance of uric acid in the comorbid course of TDB with UCTK.

2. Materials and Methods

The study included patients with and without comorbidity of TDB with UQTK. During the research, questionnaires, clinical, laboratory, biochemical, instrumental and statistical analysis methods were used in accordance with the defined tasks.

3. Results

As a result of our research, it was found that the increase in SK in the comorbid course of TDB with UIC is higher than in the comparison groups, but these differences are statistically less reliable. The average amount of SK in the main group was 475±12.6 mmol/l, and in the control group it was 402±11.4 mmol/l (p=0.08). The level of SC in patients undergoing TDB without UICs was 416±10.1 mmol/l and could not reliably differ from the main group (p=0.09).

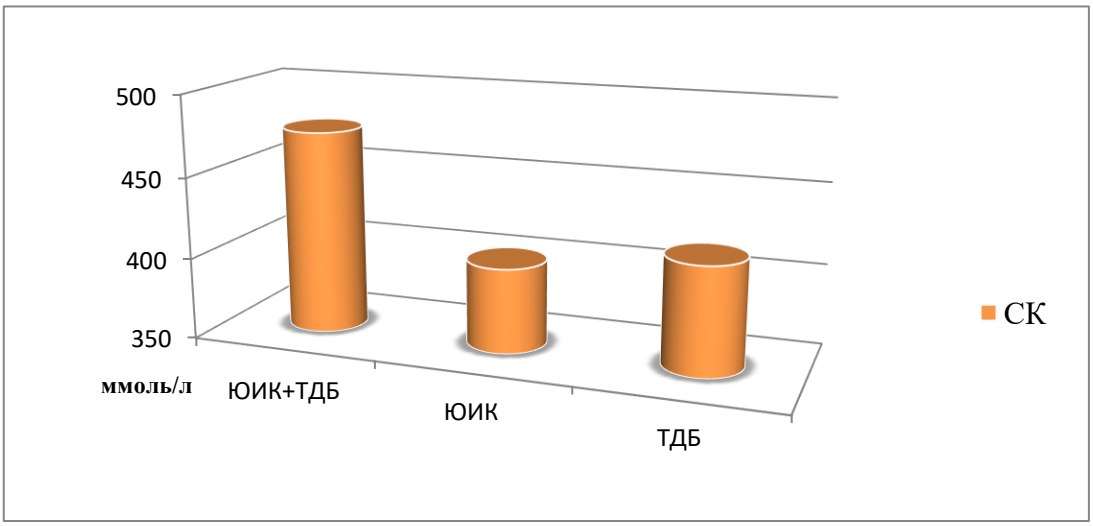

Figure 1. Status of uric acid in groups in comorbidities of TDB with UIC

However, in the comorbid condition of TDB with UIC and MS, there was a tendency to increase uric acid levels in affective disorders compared to the group without UIC (Table 1).

Table 1. The amount of UA (mmol/l) when comparing the comorbid course of TDB with UIC and MS and the course of TDB without UIC

Group	YIK+T	YIK+D	YIK+TDB+MS	TDB	T	D	P
Indicator	447,6±9,3	438,3±7,2	475,4±12,6	416±10,1	417,2±7,1	421±6,3	0,01

In addition, in the group with comorbidity of TDB with UIC, the amount of SC was 416.5 mmol/l. In patients with UICs without a history of TDB episodes, the amount of SC was 327.3 mmol/l ($p<0.05$) (Table 2).

Table 2. State of inter-group SC in patients with comorbidity of TDB with UIC and patients without affective disorders of UIC (mmol/l)

Group	YIK+TDB *(has an anamnesis)*	YIK+TDB (no history)	P
Indicator	416,5	327,3	p<0,05

In addition, the study found that uric acid and insulin have a positive correlation ($r<0.001$) (Figure 2).

Figure 2. Correlation of insulin levels with UA in the comorbidity of TDB with UIK

The obtained results show that hyperuricemia and IR can be laboratory predictors of the development of TDB comorbidity with IUD.

Diurnal excretion of uric acid in the urine of patients with TDB comorbidity with UIC. Daily excretion of SC in urine was studied. The concentration of uric acid in urine was 5.9–7.2 mmol/24h in patients with TDB comorbidity with UIC (n=52); 5.2–6.4 mmol/24h in the group of patients without mood disorders; determined in the range.

The average significance of these indicators is not statistically reliable between the compared groups: in the group of patients with comorbidity of IUD and TDB, the average amount of SC in urine is 6.4 ± 0.8; 5.8–1.1 mmol/24 h in the group of patients without affective disorders of the IUD; The results are presented in Figure 3.

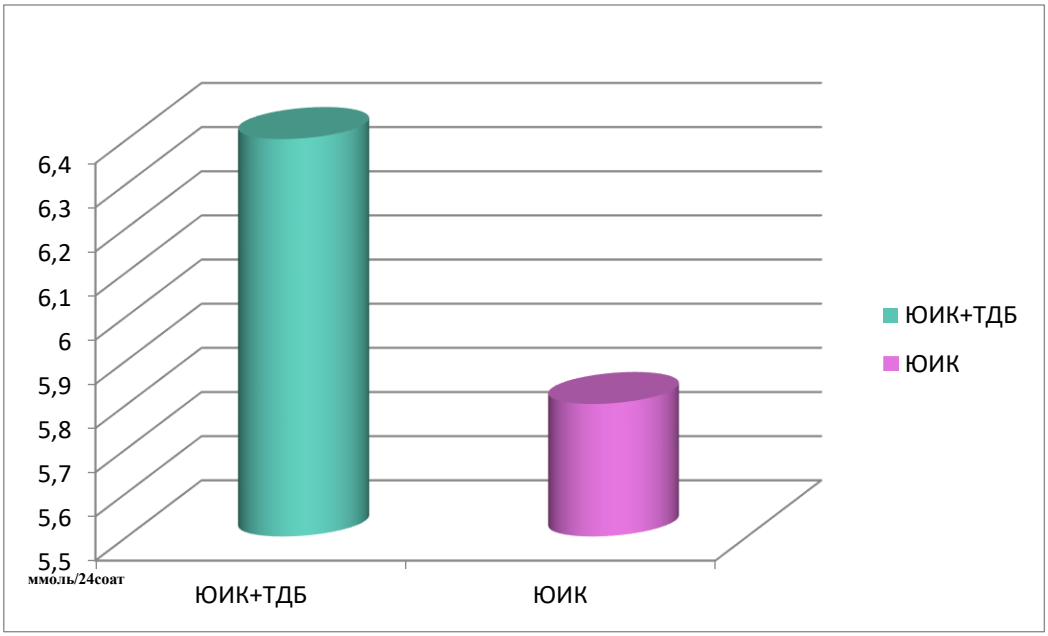

Figure 3. Comorbidity of TDB with UIC and daily excretion of SK in urine in patients in the comparison group

The amount of SC did not reliably differ compared to passing with the priority of TDBs, but in patients with the priority of the anxiety component in the comorbidity of TDB with YuIK, the level of SC excretion in urine was found to be high (Table 3).

Table 3. Excretion of SK in urine depending on the priority of the component of affective disorders in patients suffering from TDB comorbidity with UIC

Group	CUD+depression	CUD+ worry	CUD+TDB	CUD
Indicator	5,1±0,9	6,9±1,2	6,6±1,7	5,9±1,3

4. Discussion

In our study, it was found that there is an organic relationship between the increase in the amount of SC in patients with AG. In the analysis, it was found that the level of SK is correlated with CKD diseases with and without kidney diseases.

The amount of SC can be seen as a risk factor for the development of STDs. It was observed that an increase in SC level by 1.45 mg/dL (0.086 mmol/l) leads to aggravation of CKD and a 26% increase in the incidence of CKD diseases.

In patients with AG, an increase in the amount of SC to 59.9 mmol/l was observed, an increase of 18% in the comorbidity of TDB with YuIK. In the subjects, hyperuricemia increased SAB and DAB levels (average SAB 136±10.2 mm.sm.sg., DAB 87±6.2 mm.sm.sg.) and TVI increased by 27% in patients with UIK and TDS comorbidity compared to control patients (r=0.08) was found to be different.

As a result of the correlation analysis, the amount of SK in the blood serum revealed an organic relationship with the components of MS. According to him, with an increase in the amount of SK, high indicators of TVI and BA/SA ($r<0.01$), basal insulin ($r<0.01$), AG ($r<0.05$) have a reliable correlation.

It was found that the increase in the amount of SK is related to IR and TVO.

Increased uric acid level in patients with cardiovascular diseases occurs due to decreased tubular secretion of uric acid due to impaired renal excretion. Decreased secretion

may increase tubular reabsorption at the expense of IR. IR has been proven to exist in AG patients.

5. Conclusion

In comorbid conditions with high and often unpredictable variability, when cardiovascular diseases and metabolic disorders meet together, it is possible to diagnose hemostasis dysfunction, blood sugar levels, and diagnostic markers before the appearance of TDB disease symptoms in obesity, early detection and prediction of negative consequences of cardiovascular diseases. events are counted.

Based on the analyzes obtained from the study, it is possible to predict the development of CKD, the clinical course of ischemic heart dysfunction, the imbalance of the hemostasis system and metabolism.

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