

Important Principles of Diagnosis in Diabetic Nephropathy

*Bukhara State Medical Institute
Department of Endocrinology
Shakhlo Malikovna Axmedova*

Annotation: Hyperuricemia is the result of metabolic diseases and is one of the most common problematic diseases in recent years. The most common manifestations of this phenomenon are gouty arthritis, the appearance and formation of urate kidney stones. The problem of hyperuricemia and gout is a classic model of this trend, only going beyond articular syndrome. The current concept of gout nephropathy combines different clinical manifestations of uric acid metabolism: from direct damaging effects on joints and kidney tissue to indirect lipid and carbohydrate metabolism defects (by including them in clinical syndromes).

Key words: gout, hyperuricemia, uric acid, arterial hypertension, microalbuminuria, diabetes, metabolic syndrome

Uric acid has been considered metabolically inert for many years and as a result gout and hyperuricemia have been ignored by the scientific community for many years. After uric acid was identified as an etiological factor in the development of kidney disease, arterial hypertension, the medical community drew attention to this problem, after which it is perceived as a complex of diseases associated with reversibility of a large percentage of chain reactions and processes.

Hyperuricemia is the result of metabolic diseases and is one of the most common problematic diseases in recent years. The most common manifestations of this phenomenon are gouty arthritis, the appearance and formation of urate kidney stones. The problem of hyperuricemia and gout is a classic model of this trend, only going beyond articular syndrome. The current concept of gout nephropathy combines different clinical manifestations of uric acid metabolism: from direct damaging effects on joints and kidney tissue to indirect lipid and carbohydrate metabolism defects (by including them in clinical syndromes). The association of gout nephropathy with conditions such as obesity, arterial hypertension, type 2 diabetes is now associated with the term "metabolic syndrome", which is associated with dyslipidemia, early atherosclerosis, ischemic heart disease, hemostasis, microalbuminuria. [4].

In recent years, the study of uric acid and its role in the pathological development of various organ systems is increasing. One of the main challenges of research in this area is to eliminate the risk factors that contribute to the development of arterial hypertension and renal pathology. [3].

Arterial hypertension (25-50%), kidney disease (20-60%) and various cardiovascular diseases (90%) are more common among patients with hyperuricemia than in the general population. [5].

In recent years, secondary hyperuricemia caused by excessive consumption of various drugs (thiazide diuretics, salicylates, cytostatics) and alcohol has become more common. Therefore, it is advisable to correct hyperuricemia as early as possible to prevent additional kidney damage to uric acid salts [6].

The relevance of new research in this area is that, despite the fact that the gout clinic is characterized by a number of obvious manifestations, it is possible to doubt the diagnosis even with the development of the first classic gouty arthritis.

The prevalence of the disease increases with age; men get sick 3-4 times more often than women [10,12,13], but this imbalance decreases with age, in part due to a decrease in estrogen levels in women. The overall increase in the prevalence of gout nephropathy is undoubtedly associated with an aging population, an increase in the number of overweight people, and a standard diet [12,13].

Acquired and hereditary factors also play a role in the development of gout. The role of malnutrition in conjunction with physical inactivity is particularly great. Over the past 20 years, gout-related obesity, nephrolithiasis, and non-insulin-dependent diabetes mellitus in Central Asia have increased several times in parallel with the epidemic. [12]. Gout nephropathy is common, especially in countries that consume a lot of meat products.

It has been known for many years that there is a link between kidney disease and an increase in urate levels, but the fact that urate is the direct cause of kidney dysfunction is often overlooked by scientists. Modern epidemiological studies, including human and animal models with mild hyperuricemia, suggest that this metabolic disorder leads to microvascular changes and adductor of renal arterioles. This discovery shed light on the possible role of the bladder in the development of chronic kidney disease. [3].

Kidney damage develops in 30–50% [14], and according to some data, up to 75% of patients with gouty nephropathy [15] (10–25% die from this disease) [12]. The use of radioisotope radiography reveals impaired renal function in 89% of patients. With a steady increase in blood uric acid levels > 7.8 mg / dl, the risk of further development of chronic renal failure (CRF) increases by 2–10-fold. One in four patients with gout nephropathy develops chronic renal failure [16].

Significant decline in renal function is observed in approximately 38% of patients with gout. However, among elderly patients, renal failure causes 20-25% of deaths in patients with gout. However, over the years, elevated urate levels have been ruled out as a possible cause of kidney disease because arterial hypertension, diabetes, excessive alcohol consumption, overuse of nonsteroidal anti-inflammatory drugs, and lead poisoning can lead to the development of kidney disease [16].

There are two main mechanisms by which uric acid affects kidney function. Basically, hyperuricemia leads to damage and inflammation of the endothelium. Protein-1 is one of the key chemicals involved in the development of atherosclerosis and chronic kidney disease. And, secondly, hyperuricemia leads to a violation of glomerular hemodynamics. In experiments on mice, vasoconstriction of the cortex and increased renin expression were noted [12]. It has also been found that hyperuricemia resolves the nitric

oxide system in the renal apparatus and increases endothelin-1 levels, which induces vasoconstriction and exacerbates ischemia [14].

Uric acid itself leads to urate damage to the interstitial tissue of the kidney with the development of chronic tubulointerstitial nephritis, as well as acute renal failure due to intratubular obstruction through uric acid crystals (acute uric acid nephropathy) [11].

In addition, increased uric acid in the urine is a factor in the development of another variant of kidney damage - urate nephrolithiasis. Almost half of patients with gout nephropathy have urate stones. Ultrasound methods of kidney examination can significantly improve the detection of urate stones, including small and “asymptomatic” stones. A certain role in the occurrence of uric acid nephrolithiasis belongs to local renal factors, for example, impaired renal hemodynamics due to joint interstitial nephritis [14].

Hyperuricemia is also associated with metabolic syndrome. Metabolic syndrome can be an intermediate joint with many manifestations or can even lead to the development of kidney damage, including chronic inflammatory reaction, insulin resistance, and endothelial dysfunction [16]. It is also known that a fructose-rich diet is one of the factors that predispose to the development of metabolic syndrome. Because glucose consumption and high levels of hyperinsulinemia contribute to the development of hyperuricemia, some scientists believe that fructose itself can trigger or accelerate the development of renal nephropathy. Fructose itself can cause an inflammatory reaction, which has been shown in animal experiments to induce the expression of leukocyte adhesion factor, monocyte chymotactic factor-1, and cell adhesion-1 molecules. Thus, in the context of metabolic syndrome, it is more accurate to assume that only uric acid contributes to the development of nephropathy. [17].

Conclusion:

Currently, gout is an important common medical problem associated not only with an increase in disease prevalence, but also with data on the effects of hyperuricemia on triglyceridemia, insulin resistance, and metabolic syndrome. To date, many large epidemiologically promising studies have been conducted on this problem. The high frequency of hyperuricemia detected in cardiovascular disease has helped to study the role of these pathologies in the development and progression, so the detection and treatment of gout nephropathy, hyperuricemia an

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