

ARTERIAL HYPERTENSION IN PATIENTS WITH GOUT AND SYMPTOMATIC HYPERURICEMIA

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Currently, gout is a serious medical problem, and the number of patients with gouty arthritis is constantly growing in all countries with a high standard of living. Asymptomatic hyperuricemia and the high prevalence of gout (1-3%) among the population are important risk factors for cardiovascular diseases. The aim of the study is to assess the association of hyperuricemia with lipid profile disorders in patients with arterial hypertension, gout, and asymptomatic hyperuricemia. Nevertheless, the problem of using antihypertensive drugs in gout in Uzbekistan remains open. Thus, the analysis of the latest literature shows that at present, the problem of early diagnosis and modern treatment of arterial hypertension in patients with gout and asymptomatic hyperuricemia remains open.

Keywords: *gouty arthritis, asymptomatic hyperuricemia, uric acid, arterial hypertension.*

Gout is a systemic disease characterized by the deposition of sodium monourate crystals in various tissues and inflammation in people with hyperuricemia (GU) due to environmental or genetic factors [1, 14]. The first big toe joint is usually affected (50-60%) with the development of acute monoarthritis. In 15-20% of cases, gout begins with damage to other joints of the feet: II-IV fingers, ankles, knees and, exceptionally, the joints of the hands. In 5% of cases, the onset of the disease is observed in polyarticular joints. A number of studies have shown that at least 1-3% of the population suffers from gout. The incidence of gout has doubled over the past 10–20 years and continues to grow steadily: in various populations, it ranges from 5 to 50 per 1000 men and 0.2 per 1000 women [3, 11, 19, 22]. Asymptomatic elevations of uric acid (UA) occur in

5–20% of the population, of whom only 5–20% develop gout. According to the authors, asymptomatic hyperuricemia is observed in approximately 2–5% of men in the USA, 17% of the French population, 7% in Spain, and 19.3% in Russia [14, 16]. A direct relationship has been established between the level of uricemia and the likelihood of developing gout attacks.

Blood uric acid levels above 8.0 mg/dl (707 $\mu\text{mol/l}$) are considered a high-risk group for the development of gout, occurring in 5 to 12% of cases. At the same time, it has been found that GU occurs 10 times more often than gouty arthritis [5]. The prevalence of gout increases in direct proportion to age and is more than 3% in people over 75 years of age [2, 21]. The baseline level of gout has been shown to be significantly related to the level of uric acid. For example, the annual incidence was 4.5% for uric acid levels > 540 $\mu\text{mol/l}$ and only 0.1% for levels below 420 $\mu\text{mol/l}$. The 5-year cumulative incidence of gout with uric acid levels > 540 $\mu\text{mol/l}$ was 22% [6]. There is evidence that the onset of gout is younger [7, 24]. This suggests a close relationship between gout and hyperuricemia [9]. Several studies have shown that women and familial gout, nephrolithiasis, and some comorbid conditions common to gout patients, such as obesity, hypertriglyceridemia, and insulin resistance, are more likely to develop the disease [16, 19, 25]. The level of CK is higher in postmenopausal women than in premenopausal women and in urban areas than in rural areas.

Hyperuricemia has recently been considered as a risk factor for cardiovascular complications. It has been found that a large proportion (about 2/3) of patients with gout die from cardiovascular disease related to atherosclerosis, and only less than a quarter die from chronic renal failure [23]. According to the results of various studies, the frequency of arterial hypertension (AH) in patients with gout ranges from 25 to 52%, with an average of 36–41%, and increases to 72% in patients with metabolic syndrome [8, 15]. According to R. J. Johnson and co-authors, with an increase in SC by 1 mg/dl, systolic blood pressure (SBP) increases by 30 mm Hg and glomerular hypertrophy develops. The specific features of the diurnal blood pressure profile in young people with Gout are the

tendency of the nocturnal decrease in diastolic arterial pressure (DAP) to invert the diurnal DAP rhythm, as well as an increase in nocturnal blood pressure variability with an increase in blood pressure. According to D.G. Shonicheva, the frequency of systolic and diastolic hypertension in young patients with impaired purine metabolism is 50%. It is currently believed that more than 10% of patients with gout are true SK hyperproducers [10, 21]. These include people with various enzyme disorders, neoplastic diseases, and people taking cytostatics [12, 20]. In the majority of patients with gout (90%) renal excretion of SK is reduced (less than 750 mg per day) [18]. Uricosuric renal function decline can be due to various causes, including hypertension, chronic renal failure (CRF), organic kidney damage (polycystic, etc.), and drug exposure.

Epidemiological studies have shown that 15-20% of patients with gout develop urolithiasis (UT). In patients with primary gout, nephrolithiasis is 1000 times more common than in the general population [13, 25]. Urolithiasis is detected in 20% of cases with gout, which is hundreds of times more common than in patients without gout, and often (40%) precedes the articular manifestations of gout. The causes of the formation of urate stones are hyperuricemia, increased urine density, and a decrease in the fraction of excreted uric acid [14]. It is now known that gout occurs under the influence of a complex of etiological factors, which are influenced by environmental and genetic factors. Today, most researchers consider gout to be a multi-organ disease. In addition to damage to the joints and kidneys, the heart and nervous system can be involved in this process; hypertension, obesity, dermatoses, migraine diseases, that is, the process affects many organs at the same time and has a systemic nature [4, 16, 19]. According to modern data, there are 3 main phases in the pathogenesis of the disease: 1) hyperuricemia and accumulation of urate in the body, 2) urate deposition in the tissues, 3) acute gouty inflammation. Internal factors are of primary pathological importance. Increased uric acid and impaired excretion can be the result of a combination of individual disorders. In almost 90% of cases, persistent hyperuricemia is associated with reduced excretion by the kidneys, and

only a small proportion of patients with gout develop the disease solely as a result of excessive uric acid formation. Increased urate synthesis may be associated with a primary genetic defect, and sometimes with an increase in purines, a number of diseases (lymphoproliferative, hemolysis, diabetic ketoacidosis, pernicious anemia, sarcoidosis, hyperparathyroidism, hypothyroidism, psoriasis)

Food purines (beer, meat, bitter tea, caviar, legumes, coffee, cocoa, chocolate), drugs, in particular nicotinic acid, thiazide and loop diuretics, low-dose salicylates, are observed in healthy people, and also lead to short-term GU due to the rapid clearance of SK. In addition, a high-purine diet leads to a slight increase in SK - approximately 60-129 $\mu\text{mol} / \text{l}$. and a similar decrease in SK levels with a low-purine diet leads to chronic GU when conditions arise for a constant increase in SK.

The following question is currently becoming increasingly relevant:

How “harmless” is asymptomatic SG? Some researchers believe that an increase in SG can have a beneficial effect on the body. Some literature has noted that SG can improve mental and physical performance [13]. Numerous studies in the 1960s and 1970s have shown that people with SG are more intelligent and sensitive [7, 20].

A number of authors have suggested that elevated SG may be one of the main plasma antioxidants and prevent oxidative stress associated with aging, thereby helping to prolong life.

Along with data on some positive effects of SG, many studies have linked SG to cardiovascular disease and kidney damage.

One of the main visceral manifestations of gout is gouty nephropathy, which occupies one of the leading places among the extraarticular manifestations of gout. The concept of gouty nephropathy includes various forms of kidney damage caused by disorders of purine metabolism and other metabolic and vascular changes characteristic of gout. Kidney damage develops in 30-50% of patients with gout [16]. The harmful effect of Gout on the kidneys has been proven experimentally, therefore the term "asymptomatic Gout" reflects the absence of

one of the manifestations of gout - the articular syndrome, but does not exclude the development of urate nephropathy [18]. Various authors distinguish the following clinical and morphological variants of kidney damage with impaired uric acid metabolism: acute uric acid nephropathy (AUN), urate nephrolithiasis, urate nephropathy.

Acute uric acid nephropathy is common but not always diagnosed. Predisposing factors for UAN include increased urinary uric acid concentration, decreased urine pH, and decreased extracellular fluid volume. Acute UAN leads to diffuse intrarenal deposition of uric acid in the distal tubules and collecting ducts, where crystal deposition and intrarenal obstruction occur. Urinary tract obstruction develops as a result of uric acid crystallization in the distal tubules and collecting ducts, in the renal pelvis, and sometimes in the ureters. The most common manifestation of acute uric acid nephropathy is the development of acute renal failure, a characteristic feature of which is hyperuricosuria [11, 18, 24]. Urate nephrolithiasis According to the literature, 10-20% of patients with primary gout have a history of urolithiasis. The formation of stones in gout is facilitated by uricosuria of more than 700 mg per day, a decrease in urine pH, oliguria, and risk factors for the development of uric acid stones are age over 60 years and increased body weight [8, 15]. According to the literature, attention is paid to the fact that urate nephrolithiasis in patients with gout may precede articular syndrome. In 40% of patients with gout, the disease begins with urate nephrolithiasis and is subsequently accompanied by joint damage [3]. Urate nephropathy is a variant of chronic metabolic tubulointerstitial nephritis (TIN), characterized by the accumulation of SK crystals in the interstitium with the development of a secondary inflammatory process [6, 15, 21]. The main manifestations of urate nephropathy include urinary syndrome (leukocyturia, moderate proteinuria, microhematuria, hypostenuria), changes in renal tubular function [22]. According to some authors, with a persistent increase in SK levels $> 470 \mu\text{mol/l}$, up to $600 \mu\text{mol/l}$ for women and $780 \mu\text{mol/l}$ for men, the risk of further development of renal failure increases by 3-10 times, and every fourth

patient with gout develops end-stage chronic kidney disease. [1, 13]. Therefore, timely diagnosis of kidney damage in gout is an important prognostic factor.

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