ISSN 3060-4567

METHODS AND RISK FACTORS FOR EXAMINING PATIENTS WITH GOUT

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Abstract: *Gout is a crystal-deposition disease that results from chronic elevation of uric acid levels above the saturation point for monosodium urate (MSU) crystal formation. Initial presentation is mainly severely painful episodes of peripheral joint synovitis (acute self-limiting 'attacks') but joint damage and deformity, chronic usage-related pain and subcutaneous tophus deposition can eventually develop. The global burden of gout is substantial and seems to be increasing in many parts of the world over the past 50 years. However, methodological differences impair the comparison of gout epidemiology between countries. In this comprehensive Review, data from epidemiological studies from diverse regions of the world are synthesized to depict the geographic variation in gout prevalence and incidence. Key advances in the understanding of factors associated with increased risk of gout are also summarized. The collected data indicate that the distribution of gout is uneven across the globe, with prevalence being highest in Pacific countries. Developed countries tend to have a higher burden of gout than developing countries, and seem to have increasing prevalence and incidence of the disease. Some ethnic groups are particularly susceptible to gout, supporting the importance of genetic predisposition. Socioeconomic and dietary factors, as well as comorbidities and medications that can influence uric acid levels and/or facilitate MSU crystal formation, are also important in determining the risk of developing clinically evident gout.*

Keywords: *Gender; Gout; Risk factors; Systematic review*

Independent risk factors for gout. Obesity is one of the main components associated with gout in the long term . Obesity is a well-established risk factor for the

ISSN 3060-4567

development of diabetes, dyslipidemia, cardiovascular disease, hypertension, osteoarthritis, and gout. Epidemiological studies have shown that body mass index is independently associated with increased uric acid levels [18, 20]. Many clinical and epidemiological studies have been conducted on the association of hyperuricemia with various cardiovascular diseases. Some authors consider hyperuricemia to be an independent risk factor for the development of cardiovascular disease and an increased risk of mortality in patients . There are many studies in the literature, including epidemiological studies on the combination and interaction of hypertension and hyperuricemia.

Increased blood uric acid levels are considered by many authors as a predictor of the development of hypertension. Studies conducted in sufficiently large cohorts of patients have revealed an increased risk of hypertension even in patients with relatively low blood uric acid levels (300-420 μmol/l). Hypertension developing in patients with hyperuricemia and gout can be considered secondary. This is explained by a number of factors: the deposition of uric acid crystals in the interstitial tissues leads to the formation of a giant cell reaction with excessive production of cytokines, followed by fibrosis of the interstitial tissues of the kidneys and the development of tubulointerstitial nephritis. The development of tubulointerstitial kidney damage leads to increased sodium reabsorption in the ascending limb of the loop of Henle and increased sensitivity to natriuretic hormone, leading to water and sodium retention, increased circulating blood volume, total peripheral vascular resistance, activation of the sympathetic division, the nervous system, and ultimately to the development of hypertension. In addition, glomerular hyalinosis and intimal hypertrophy may develop. Another reason for the development of hypertension against the background of hyperuricemia may be a decrease in the response of the renin-angiotensin system to angiotensin II due to a decrease in the sensitivity of angiotensin II receptors in patients .

Left ventricular hypertrophy is common in patients with hypertension and hyperuricemia. Increased left ventricular myocardial mass index in patients with hypertension and established myocardial hypertrophy is closely related to uricemia

ISSN 3060-4567

[16]. Many studies have shown that the prooxidant effect of SC on lipid oxidation, particularly low-density lipoprotein, is an assessment of oxidative stress in patients with acute myocardial infarction and acute heart failure [3, 14]. The prooxidant effect of SC has also been found to be associated with cardiovascular disease, atherosclerosis, diabetes, lipid peroxidation, and increased endothelial dysfunction products. The effect of SC on endothelial dysfunction is associated with the development of hypertension by the expression of adhesion molecules on the surface of endothelial cells under the influence of the pro-inflammatory cytokines interleukin-1 (IL-1) and tumor necrosis factor. In these patients, impaired nitrogen release from endothelial cells and an increase in C-reactive protein are observed . The relationship between hyperuricemia and AG is still unclear. The presence of hypertension itself may be the cause of the formation of GU. If we take into account that AG is formed as a result of the action of uric acid, AG itself may contribute to the subsequent development of hyperuricemia. This depends on a number of factors. Hypertension causes microvascular damage, which in turn leads to tissue ischemia and the breakdown of ATP into adenine and xanthine, increased production of xanthine oxidase, and, as a result, the formation of purines from nucleosides due to hyperuricemia. Oxygen radicals are used in the xanthine oxidase reaction, which, in turn, activates lipid peroxidation . Thus, timely identification of risk factors is the basis for the prevention of cardiovascular events caused by atherosclerosis. It is of great importance to study renal function and the state of the lipid profile in patients with gout, especially in combination with arterial hypertension.

Methods of diagnosing gout

Despite the clear pathogenesis of the disease and the availability of effective therapy, gout is often not diagnosed or is diagnosed late. According to V.A. Nasonova et al. (2004), gout is diagnosed only in the 8th year of the disease.

For the diagnosis of gout, only the diagnostic criteria of the Wallace S.L. classification (1997) and the classification criteria approved by WHO in 2000 are currently considered to be the only ones. The criteria consist of three blocks: I (A) - the presence of characteristic crystalline urates in the joint fluid or II (B) - the presence

ISSN 3060-4567

of tophi containing crystalline urates confirmed by chemical or polarizing microscopy or III (C) - the presence of 6 of the following 12 signs.

1. multiple arthritis attacks in the anamnesis;
2. maximum inflammation on the first day;
3. monoarticular nature of arthritis;
4. hyperemia of the skin over the affected joint;
5. swelling and pain in the first big toe joint;
6. unilateral damage to the first big toe joint;
7. unilateral damage to the joints of the arch of the foot;
8. tophi;
9. hyperuricemia;
10. asymmetric swelling of the affected joint;
11. subcortical cysts without erosion on radiographs;
12. Lack of flora in the synovial fluid during synovial fluid culture.

In the absence of a polarizing microscope, the diagnosis of gout can be made according to clinical criteria III (B) (the presence of 6 of the 12 above signs), but in 100% of cases, a reliable diagnosis can be made only by the presence of characteristic crystalline urates in the synovial fluid and / or the presence of a tophi containing crystalline urates confirmed by chemical or polarizing microscopy. The diagnosis of gout seems simple even without microscopy. The nature of acute gouty arthritis is very characteristic: acute pain, reaching the culmination of arthritis within 12 hours, involvement of the first metatarsal phalanx (MTFB) joint, asymmetry. However, the presence of MTFB I is highly sensitive (0.98; 95% confidence interval 0.95–1.02) but has low specificity (0.23; 95% CI 0.10–0.35). Acute monoarthritis, including MTFB I, is also seen in other diseases [12]. In this regard, the European League Against Rheumatism (EULAR) expert panel recommends that physicians perform joint puncture in patients suspected of having gout, as well as in any other undifferentiated arthritis [8]. Several studies have shown that polarizing microscopy has a high specificity for detecting sodium monourate crystals (SMC) in synovial fluid or in tophus, but there is interlaboratory variability. This is likely due to the fact that SMC

ISSN 3060-4567

crystals cannot be detected in all patients with significant gout in synovial fluid samples. This depends on the stage of the disease, allopurinol therapy, and a number of other factors.

Hyperuricemia is the most important risk factor for gout. However, uric acid levels may decrease during a gout attack, so some time should elapse after the arthritis has resolved to check the level of uric acid in the blood. However, in a patient with gout, even with the treatment of arthritis, it is not necessary to expect a normal level of uric acid without antihyperuricemic therapy, that is, with normal values of this indicator, the presence of gout is doubtful . Of particular interest in the framework of the problem discussed is the study of inflammatory markers. On the one hand, uric acid is a “predictor” of cardiovascular events in the population, and its level is associated with the development of hypertension, insulin resistance and other classic cardiovascular risk factors . On the other hand, uric acid levels reflect the chronic inflammatory process in rheumatic diseases, including gout. X-ray examination of the joints. This helps to establish a differential diagnosis and identify typical signs of chronic gout. Radiologically diagnosed changes develop in 10-40% of cases after repeated attacks of arthritis and serve as one of the late manifestations of the disease. A characteristic radiological sign for late gout is the "shock" sign.

A number of authors have found that, as a rule, well-differentiated "perforated" periarticular erosions are detected radiographically 6-12 years after the first acute attack of gout . Ultrasound in the diagnosis of gout. Ultrasound examination of joints (UTT) in gout is a promising direction. A characteristic ultrasound symptom in gout is the "double contour" symptom [14]. The "double contour" sign is associated with the ability of crystals to accumulate on the articular surface, which is detected by ultrasound as an additional bright line parallel to the line of passage of the subchondral bone. Another pathological finding in gout that can be detected by ultrasound is tophus. Detection of tophus structures facilitates early therapeutic measures. The determination of renal excretion of uric acid, in particular the ratio of uric acid to creatinine, can be used to identify hyperproducers and to decide whether to prescribe uricosuric drugs. Magnetic resonance imaging (MRI) is particularly

useful in gout for the detection of tophus masses, since synovitis, synovial fluid, periarticular tissue damage, and edema are not characteristic of gout. Several authors have shown that tophus formation can occur early in the disease and even before the appearance of typical signs of arthritis. MRI can be informative in the differential diagnosis. Thus, the use of MRI in the differential diagnosis of tophus and neoplasms and infectious processes has been described In addition, MRI can be a good monitoring tool during antihyperuricemic therapy due to its ability to assess the size and number of tophus over time. Computed tomography (CT) is another potentially useful method for imaging tophus. The method allows to detect tophus localized both in the bone and in the tendon and soft tissue area . So far, the method has not found widespread application, primarily due to its high cost. Thus, a review of the recent 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.

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