RESEARCH JOURNAL OF TRAUMA AND DISABILITY STUDIES

Volume: 02 Issue: 11 Nov– 2023 ISSN: 2720-6866
http://journals.academiczone.net/index.php/rjtds

Results of a Clinical Approach to Gouty Nephropathy

Shakhlo Malikovna Axmedova

Bukhara State Medical Institute, Department of Endocrinology

Abstract 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.

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

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

Volume: 02 Issue: 11 Nov– 2023 ISSN: 2720-6866
http://journals.academiczone.net/index.php/rjtds

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].

Volume: 02 Issue: 11 Nov– 2023 ISSN: 2720-6866
http://journals.academiczone.net/index.php/rjtds

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 [16]. 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 [17]. 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 chymotractic 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. [18].

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

Volume: 02 Issue: 11 Nov– 2023 ISSN: 2720-6866
http://journals.academiczone.net/index.php/rjtds

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

Literature:

1. Shostak N.A., Anichkov D.A. Metabolic syndrome: diagnostic criteria and the possibility of hypertensive therapy // Russian Medical Journal. 2002. № 27. pages 1258-1261
2. Kanbay M., Ozkara A., Selsoki U. et al. Effects of treatment of hyperuricemia with allopurinol.
3. Hozla U.M., Jarikov S., Finchi J.L., Nokagawa T., Ronkal C., Mu V., Krotova K., Blosk E.R., Prabhakar S., Johnson R.J. Hyperuricemia causes endothelial dysfunction // Renal internation. 2005. Volume 67. P.1739.
4. Muxin N.A., Balkarov N.M., Maksimov N.A. Clinical manifestations of disorders of purine metabolism in internship practice // Ter.archiv.- 1994.- №1. -FROM. 35-39.
5. Lawrence RC, Helmisk CG, Arnett F.C. et al. Estimates of the prevalence of arthritis and selected musculoskeletal system in the United States.// Arthr. Revm. 2008. № 41. S. 778-99.
6. Muxin N.A., Serov V.V., Maksimov NA. Some features of the hyperuricemic variant of occult glomerulonephritis. // Ter. Archive. 2000. № 6. P.43-46.d complications of the disease is a topical issue of therapy.
7. Kudasheva F.M., Eliseev M.S., Barskova V.G., Nasanova V.A. Comparison of the onset rate of analgesic and anti-inflammatory effects of different nimesulide and diclofenac sodium in gouty arthritis // Ter.archiv. 2007. № 5. S. 35-40
8. Bergar L., Yu T. Renal changes in gout nephropathy // Am. J. Med. 1975. V. 59. P. 605-608
9. Nakagawa T., Mazalli M., Kang D-H. Et al. Hyperuricemia causes glomerular hypertrophy in rats. // Am. J. Nefrol. 2003. Volume 23. R. 2-7.
10. Xosla U.M. et al. Hyperuricemia causes endothelial dysfunction. // Kidney lnternationa. 2005. Volume 67. P.1739.
11. Shcherbak A.V., Balkarov I.M., Kozlovskaya L.V. Other fibrinological activity of urine as an indicator of renal injury with impaired uric acid metabolism. // Ter. Archive. 2001. T. 73 (6). S. 34-37.
12. Faschini F. Et al. Correlation between insulin-mediated glucose uptake and uric acid concentration in urine // SAMA. 1991. № 266. P. 3008-3011.
13. Vazguez-Mellado J., Garcia S.G., Vanguez S.G. et al. Metabolic syndrome and ischemic heart disease in gout nephropathy // J. Clinic. Revmatol. 2004. Volume 10 (3). P. 105-109.
14. Fam A.G. gout nephropathy duo and insulin resistance syndrome // J. Revm. 2002. Volume 29. P. 1350-1355
15. Nakayama., Hyperuricemia causes glomerular hypertrophy in rats. // Am. J. Nefrol. 2010. Volume 23. R. 2-7.

Volume: 02 Issue: 11 Nov– 2023 **ISSN: 2720-6866**
http://journals.academiczone.net/index.php/rjtds

16. Gersh M.S., Renal function in gout nephropathy // Am. J. Med. 2007. V.59. P. 605-608
17. Cohen M., Emmerson B. Rheumatology of gout. 1994. V. 7. P. 1-16
18. Kanbay M., Metabolic pathways of high-density lipoprotein, low-density lipoprotein, and triglycerides: flow rewire. // Am. J. Cardiol. 2000. V. 86. (12 appendix L). P. 5-10
19. Shaymanova, Y., & Qarshiboyeva, Z. (2023). O ‘ZBEK TILIDAGI NEOLOGIZMLARNING TASNIFI CLASSIFICATION OF NEOLOGISMS IN THE UZBEK LANGUAGE. QISHLOQ XO'JALIGI, ATROF-MUHIT VA BARQAROR RIVOJLANISH MILLIY KONFERENSIYASI, 93-96.
20. Toshniyazovna, S. Y., & Beknazarovna, E. M. (2023). Theoretical Substantiation of the Formation of the Language Competence of Students in the Course of Studying Syntactic Norms. Periodica Journal of Modern Philosophy, Social Sciences and Humanities, 18, 63-65.
21. Shaymanova, Y. T., & Qarshiboyeva, Z. A. (2022). RUS OLIMI AN SAMOYLOVICHNING SHARQ TILLARINI O'RGANISHGA QO’SHGAN HISSASI. Oriental renaissance: Innovative, educational, natural and social sciences, 2(Special Issue
22. Abduvaxabovna, K. Z. (2022). SOME LINGPOOPETIC FEATURES OF RHETORICAL INTERROGATIVE SENTENCES. Galaxy International Interdisciplinary Research Journal,
23. Bozorova, G. R. (2022). EFFECTIVE WAYS TO TEACH THE LIFE AND CREATIVITY OF MUHAMMAD YUSUF IN SCHOOL LITERATURE EDUCATION. Международный журнал языка, образования, перевода, 3(2).
24. Bozorova, G. (2023). " Turfa zakovatlar nazariyasi" asosida o'quv topshiriqlarini ishlab chiqish. “VETERINARIYA SOHASINI RIVOJLANTIRISHDA IJTIMOIY-GUMANITAR FANLARNING O ‘RNI” MAVZUSIDAGI Respublika ilmiy-amaliy konferensiya materiallari TO ‘PLAMI.