22. URIC ACID – AS A MODERN CARDIOVASCULAR RISK FACTOR

Author: Syed Razak

Scientific adviser: Alexandra Grejdieru, MD, Associate Professor, Discipline of Cardiology, Department of Internal Medicine, *Nicolae Testemitanu* State University of Medicine and Pharmacy of the Republic of Moldova.

Introduction. Uric acid (UA) is the end product of purine metabolism in higher animals, such as humans and great apes UA synthesis and excretion in the body are balanced under physiological conditions. Hyperuricemia occurs when this balance is disrupted. Male UA levels greater than 7 mg/dL and female UA levels greater than 6 mg/dL are considered hyperuricemia in most cases. Hyperuricemia is frequently associated with diseases caused by an unhealthy lifestyle. Concomitant hyperuricemia affects approximately 25–40% of untreated hypertensive patients and an association between elevated serum uric acid (SUA) and hypertension (HT) has been described in adults in several large epidemiological studies. A number of recent small clinical trials have demonstrated that SUA-lowering agents such as allopurinol and probenecid can lower blood pressure (BP) in adolescents, indicating that UA is an independent risk factor for the development of high blood pressure

Aim of study. Hypertension is strongly associated with elevated serum uric acid (sUA), but the exact reason for this is not known. Hyperinsulinemia caused by insulin resistance increases sodium reabsorption in the kidneys, which may result in high blood pressure. Additionally, endothelial dysfunction caused by oxidative stress is a significant contributor to the development of hypertension. According to research, UA significantly increased the production of reactive oxygen species (ROS) and angiotensin II in human endothelial cells. Many studies have been conducted in recent years that have demonstrated a link between sUA and hypertension. A 10-year follow-up of a prospective randomized study of 5748 healthy adolescents revealed that elevated sUA was closely associated with hypertension and the metabolic syndrome, according to the findings. According to a large-scale meta-analysis of 55,607 subjects from 18 prospective cohort studies, the incidence of hypertension increased by 13 percent for every 1 mg/dl increase in serum uric acid (sUA). The two main classes of ULT (urate lowering treatments) drugs are currently in use in clinical practice are those that inhibit UA synthesis (XO inhibitors, such as allopurinol, febuxostat, and others) and those that increase UA excretion (ULT drugs that act on the kidneys) (e.g., benzbromarone, probenecid, etc.). Current research confirms that ULT has a positive effect on hypertensive patients under the age of 40. A notable example is the use of sodium glucose cotransporter 2 inhibitors (SGLT-2; dapagliflozin, empagliflozin, canagliflozin, and others) to effectively lower sUA levels by increasing the rate of UA excretion thus helps in lowering hypertension.

Methods and materials. In this article, the literature review was analyzed from PubMed, Google Scholar and NCBI sites.

Results. The relationship between hyperuricemia and cardiovascular disease is becoming increasingly clear, which can be attributed to the advancement of research in the field of UA. The mechanism by which uric acid is causing hypertension is becoming clear and by blocking this mechanism we can lower the level of hyperuricemia. Some drugs such as Allopurinol can help in reducing hypertension.

Conclusion. The study clarifies the treatment associated with hypertension caused by hyperuricemia is effective in decreasing the level of UA levels.