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Zhdan V.M.,
DMSc, Professor, Head of the Family Medicine and Therapy Chair at Higher State Educational Establishment of Ukraine "Ukrainian Medical Stomatological Academy",
Kitura O.E.,
PhD, Associate Professor of Internal Medicine and Emergency Medicine Chair at Higher State Educational Establishment of Ukraine "Ukrainian Medical Stomatological Academy",
Kitura E.M.,
PhD, Associate Professor of Family Medicine and Therapy Chair at Higher State Educational Establishment of Ukraine "Ukrainian Medical Stomatological Academy",
Babanina M.Y.,
PhD, Associate Professor of Family Medicine and Therapy Chair at Higher State Educational Establishment of Ukraine "Ukrainian Medical Stomatological Academy",
Tkachenko M.V.,
PhD, Family Medicine and Therapy Chair at Higher State Educational Establishment of Ukraine "Ukrainian Medical Stomatological Academy",
Ivanytskyi I.V.
PhD, Associate Professor of Family Medicine and Therapy Chair at Higher State Educational Establishment of Ukraine "Ukrainian Medical Stomatological Academy"
E-mail: e.kitura@mail.ru

HYPERURICEMIA AND ARTERIAL HYPERTENSION IN GENERAL PRACTICE

Recently, the issue of correlation between elevated level of uric acid (UA), arterial hypertension (AH) and other metabolic disorders is of great interest for researchers. It has been proved by evidence that hyperuricemia (HU) is an independent factor for coronary heart disease cardiovascular morbidity and related mortality in patients with coronary heart disease [8,1548; 3,287], chronic and acute heart failure [2,78], AH and prehypertension and metabolic syndrome [9,6]. However, UA is more significant as a risk factor for cardiovascular events in patients with AH, proved by the data of perspective studies [5,160]. It has been proved by evidence that elevation of UA level in blood plasma may precede the occurrence of AH in apparently healthy individuals [10,1080]. In this way, essential hypertension has been observed in 25% of patients with AH before treatment and 50% of patients, treated with diuretics. Furthermore, arterial hypertension is more often revealed in individuals with hyperuricemia than with normal UA level in blood plasma. Positive dynamics between the level of diastolic AH and UA and enlargement of left ventricular hypertrophy in

patients with AH with hyperuricemia as compared with patients with normoruricemia [5,160].

The role of UA as an independent predictor of cardiovascular events and related mortality in patients with AH has been investigated by SHEP study, involved 4327 patients, aged above 60 years with isolated systolic hypertension who were treated for 5 years with thiazide diuretics, supplemented with atenololum or reserpinum, if necessary [6,1154] .

The findings of the present study validated the linear dependence of the number of cardiovascular events from the initial UA level, with the exception of strokes. It has been proved by evidence that diuretic therapy in therapeutic doses led to elevation of UA level in half of the patients during the year.

The reasons for hyperuricemia are variable. Elevation of UA level may be caused by kidney damage, frequent consumption of fatty meats, seafood, alcohol, as well as obesity that increase the risk for hyperuricemia by 3 times. Numerous medications, including loop and thiazide diuretics can change and impair the renal UA clearance.

Hyperuricemia is also recognized as an important component of the metabolic syndrome (MS). In initial diagnostics of MS hyperuricemia is considered as the main component with hypertension and hyperlipidemia.

PIUMA Study investigated the important role of UA as an independent predictor of cardiovascular morbidity and mortality, involved 1720 patients with AH who have been observed for 12 years. In patients with AH with initial UA level grater than 0,396 mmol/l the risk of cardiovascular events, fatal cardiovascular events and total mortality were significantly higher than in patients with UA level from 0,268 mmol/l to 0,309 mmol/l [10,1076].

Consequently, the following evidences of UA correlation with AH development exist to date:

- elevated UA level predicts the development of hypertension;
- elevated uric acid level is observed in 25-60% of patients with untreated hypertension and almost 90% of adolescents with essential hypertension and the recent debut;
- lowering of UA level with xanthine oxidase inhibitors reduces blood pressure in adolescents with arterial hypertension.

Clarifying the relationship of UA to the risk of cardiovascular disease is of clinical significance. Firstly, the incidence rate and spread of gout and HU has been registered in the society. Secondly, asymptomatic HU is not an indication for urine-lowering therapy.

Accordingly, the need to develop methods for correcting asymptomatic hyperuricemia is crucial. The reasonability of administration of xanthine oxidase inhibitors (Allopurinolum) to patients with cardiovascular disease requires validation in randomized trials. Therefore, it is crucial to perform selection of drugs appropriately for patients with AH and hyperuricemia for positive impact on purine metabolism and other conditions, related to its disorder.

A successful AH control is one of the components of the effective HU treatment in toto that should meet the general requirements for antihypertensive therapy [1,20]. Antihypertensive agents are to be of a positive impact on the purine and lipid metabolism, insulin sensitivity with no negative impact on manifested nephropathy. Interrelation of complicated metabolic process should be taken into consideration in patients with AH and HU not to increase the risk of therapeutically-induced gout.

Similarly, drug metabolic safety is of great importance, as well as drug selection, possessing the ability to reduce the risk of cardiovascular complications. It has been shown that there is no safe type of diuretics, all drugs of this group inhibit UA excretion, leading to HU; β-blockers also are not the drugs of choice in AH and HU conjunction, as they increase insulin- resistance, hyperinsulinemia, increasing HU.

Presently, it is known that calcium antagonists of dihydropyridine series of prolonged effect (Amlodipinum) are metabolically neutral agents, promoting optimal AH control; ACE inhibitors, angiotensin II receptors blockers with positive hyperuricemial effect due to reduced urate reabsorption in the proximal kidney tubules. Moreover, Losartanum is able to eliminate HU, associated with administration of thiazide and thiazide-like diuretics.

None of the sartana, with the exception of Losartanum, are able to reduce UA in combination with diuretics [7,304].

Conclusion:

1. Consequently, the reliable correlation between hyperuricemia and AH has been established to date.

2. Such metabolically neutral agents as dihydropyridine calcium antagonists of dihydropyridine series, ACE inhibitors, angiotensin II receptors blockers should be used for effective AH control.

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