The Significance of Microelements Deficiency in Cardiological Practice

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ABSTRACT

The consequences and causes of magnesium deficiency in cardiovascular diseases are considered. The features of magnesium metabolism in atherosclerosis, arterial hypertension, and heart failure are described. The prospects of therapy with magnesium preparations in cardiology practice are discussed.

Keywords: magnesium deficiency, blood pressure, atherosclerosis, endothelial cells, heart failure, renin- angiotensin system, diuretics.

Introduction. The importance of magnesium in the body. The human body at birth contains about 760 mg of magnesium, approximately 5 g at the age of 4-5 months and almost 25 g in adulthood. At the same time, 30-40% of the magnesium contained in the body is found in the muscles and soft tissues, 1% is found in the extracellular fluid, and the rest is contained in the bone tissue. The importance of magnesium is difficult to overestimate. So, almost all chemical processes in the body require the participation of enzymes. The enzyme system basically consists of 3 components: a specific protein molecule (the enzyme itself), a smaller molecule (most often a vitamin, such as pyridoxine, or vitamin B 6) and a positively charged metal ion, such as zinc, copper, manganese, or magnesium. Magnesium is the second most abundant intracellular cation after potassium and serves as a cofactor for over 300 enzymatic reactions, including energy metabolism, protein synthesis, and nucleic acids. From all of the above, we can conclude that magnesium is a vital element, the lack of which can cause serious disturbances in the body's vital functions. In this article, we will try to describe the nosological forms of cardiovascular diseases (CVD) that are associated with magnesium deficiency.

Magnesium and atherosclerosis. There is strong evidence that the concentration of extracellular magnesium plays an important role in the regulation of endothelial cell activity [1]. Indeed, magnesium levels affect nitric oxide synthesis [2], intracellular calcium release [1], LDL uptake and metabolism [3], membrane permeability to water and albumin [4], and endothelial cell proliferation [5]. In addition, magnesium deficiency enhances the cytotoxic effect of free radicals in endothelial cells [6]. Since endothelial cells play an important role in atherogenesis [7], it can be hypothesized that magnesium affects the mechanisms of atherosclerosis development. I. Maler et al . [8] in their study in 2004 proved that the inhibition of endothelial cell growth that occurs with magnesium deficiency stops after its normal level is restored, thus indicating that no irreversible changes occur with magnesium deficiency. Most likely, stimulation of cell growth occurs due to the action of intracellular magnesium, which catalyzes the reactions necessary for the process of endothelial cell proliferation. Since

magnesium plays the role of a natural calcium antagonist, it can also be assumed that the balance between these two ions also affects cell growth processes.

Data from other studies also indicate the direct involvement of magnesium in the pathogenesis of CVD in general and atherogenesis in particular. For example, the consumption of water enriched with magnesium ions by mice with a reduced level of apolipoprotein (which has a strong anti-atherosclerotic effect), along with a high-cholesterol diet, led to inhibition atherogenesis [9].

At the same time, oral administration of magnesium preparations is associated with a significant improvement in endothelial function in patients with coronary insufficiency [10]. According to the ARIC study (Atherosclerosis risk in Communities Study), a high risk of developing coronary insufficiency is noted in patients with low levels of magnesium in the blood serum, even taking into account traditional risk factors for developing coronary insufficiency [11].

In cultured endothelial cells, a low concentration of magnesium increases the adhesion of monocytes to them, since the adhesion molecule of vascular cells (vascular cell adhesion molecule - VCAM). Under these conditions, the activity of an inhibitor of the plasminogen activator-1 (plasminogen activator inhibitor-1 - PAI-1), the level of pro-inflammatory cytokines (interleukin 3 IL-1a) increases , endothelial proliferation worsens, and cell aging occurs [12, 13]. In a study in mice with hereditary hypomagnesemia (MgL mice , hypomagnesemia in mice), indicates a significant decrease in endothelial function [14]. Confirming the results obtained in cultured cells, the MgL aorta also showed a higher expression of VCAM and PAI-1 than in the control group [14].

Magnesium and arterial hypertension (AH). The inverse correlation between magnesium levels (in serum and/or tissues) and blood pressure (BP) levels has long been known. In addition, the relationship between the renin- angiotensin system (RAS), magnesium and blood pressure has been described. Patients with hypertension and high plasma renin activity (ARP) have lower serum magnesium concentrations than those with normal blood pressure [15]; thus, serum magnesium concentration is inversely related to ARP. Since an increase in ARP indicates activation of the RAS, it is quite possible that angiotension II - dependent AH is at least in part due to the consequences of hypomagnesemia .

In earlier studies, the total level of magnesium ions was determined, which did not always reflect a reliable ratio between intra- and extracellular magnesium. At present, thanks to fluorescent methods for determining the concentration of Mg2+ and Mg2+-specific electrodes, it has become possible to study the content of magnesium ions directly in living cells. At the same time, according to modern analytical data, in the cells of many types of patients with AH, a significantly reduced concentration of magnesium ions is determined, even if the level of total magnesium is within the normal range [16]. The main reasons for the change in magnesium metabolism in hypertension are unclear, most likely, genetic, dietary and hormonal factors, as well as ongoing drug therapy, play a role.

Not all clinical studies support magnesium depletion in hypertension. In some studies, in patients with hypertension, compared with individuals with normal blood pressure, no changes in the level of magnesium were detected [17], while others even indicate an increase in the concentration of Mg2+ in erythrocytes in patients with essential hypertension [18]. Moreover, several epidemiological studies failed to find an association between magnesium intake and BP levels [19]. It follows from this that not all hypertensive patients have a magnesium deficiency, just as not all people with magnesium deficiency are hypertensive patients. Despite conflicting views on the role of magnesium in the development of hypertension, there are subgroups of hypertensive patients who always have impaired magnesium metabolism. These include African Americans, overweight patients, patients with insulin resistance , hypertriglyceridemia , and patients with severe or malignant forms of hypertension [20].

Heart failure (HF). C. Adamopoulos [21] et al. in their work indicate that the content of magnesium in serum <2 mEq / l increases the risk of death in patients with chronic heart failure (CHF). This is one of the first studies to investigate the long-term effects of low

magnesium levels on mortality and hospitalization in patients with CHF. According to this work, hypomagnesaemia directly associated with cardiovascular mortality but not with hospitalization for heart disease. The authors hypothesize that magnesium deficiency is associated with sudden cardiac death and life-threatening arrhythmias [22—24]. In addition, hypomagnesaemia can cause myocardial fibrosis and increase platelet aggregation, which also affects cardiovascular mortality [25, 26]. Finally, low serum magnesium levels may be a marker for the progression of HF. Aldosterone, a neurohormone whose activity is elevated in HF, increases magnesium excretion in the urine, thereby causing hypomagnesemia [27].

In addition, magnesium levels are affected by medications taken by patients with heart failure. Diuretics used in CHF affect the level of magnesium excretion in the urine in different ways. Since the kidney magnesium reabsorption occurs in the loop of Henle , furosemide and other loop diuretics cause significant loss of magnesium in the urine. Thiazide diuretics, by inhibiting magnesium reabsorption in other areas, also cause hypomagnesemia , but to a lesser extent than furosemide . At the same time, spironolactone , amiloride , and triamterene reduce urinary magnesium loss [28]. Regarding the effect of angiotensin-converting enzyme (ACE) inhibitors on the level of magnesium in the blood, there is a version according to which patients with higher doses of ACE inhibitors have reduced activity of the RAS, which has a beneficial effect on the level of magnesium in the blood.

Based on the foregoing, we can conclude that in patients with CHF, it may be appropriate to study the level of magnesium in the blood, and in case of its deficiency, oral preparations containing magnesium and/or aldosterone inhibitors should improve the prognosis .

Conclusion. The modern Western diet contributes to insufficient intake of magnesium from food, as micronutrient-poor high-calorie foods are preferred, and magnesium salts, which cause increased “hardness” of water, are removed from the water. Oral administration of magnesium preparations to compensate for its deficiency can be useful in a number of pathologies, including hypertension, coronary heart disease, coronary insufficiency, heart rhythm disturbances, as well as chronic fatigue syndrome, many types of musculoskeletal disorders, epilepsy, bronchial asthma, panic disorders and many other organic and mental pathologies. However, in practice, supplements containing magnesium are rarely prescribed by doctors, which indicates an underestimation of the importance of this element. Until recently, interest in magnesium in traditional medicine was observed only among obstetricians who used magnesium sulfate for the relief of hypertension, preeclampsia and eclampsia of pregnant women. Recently, interest in the behavior of magnesium has spread to physicians in other specialties. Of course, you can not treat magnesium as a miracle cure that can solve all the problems in modern medicine at once. So, S. Patrick et al. [29] in their study put an end to the assumption that intravenous administration of magnesium into the coronary vessels contributes to a significant reduction in mortality during an acute heart attack. It may be more correct to raise the question of the preventive correction of magnesium deficiency in the development of CVD using oral organic magnesium salts such as magnesium citrate, magnesium pidolate and magnesium lactate , especially in combination with pyridoxine as a magnesium protector. A multicenter observational study including 2000 patients showed that insufficient magnesium levels corresponded to a significant increase in the risk of the following conditions: R56.8. convulsions; I63.0. Ischemic cerebral infarction; I10. Essential primary hypertension; I34.1. Mitral valve prolapse; F43.0. Acute reaction to stress; I20.0. Unstable angina; I47.9. Paroxysmal tachycardia, unspecified, etc. [30]. Benefits of Magnesium Citrate and Pidolate compared not only with inorganic salts, but also with some organic salts (orotate , gluconate), it has a higher content of elemental magnesium, better bioavailability , especially in combination with pyridoxine. Citrate is an ideal fully utilizable carrier of magnesium into the cell. There are no magic substances and drugs, just as there are no cures for all diseases. But there are useful and somewhat underestimated groups of drugs that, apparently, will occupy their niche in the complex treatment of patients.

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