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**THE EFFECT OF MINERALODIPECITE ON THE FUNCTION OF THE
GASTROINTESTINAL TRACT**

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Annotation: The presented article "The effect of mineralodipeptide on the function of the gastrointestinal tract" is devoted to microelementosis. Trace element correction in modern practical medicine is gaining more and more supporters, convinced of the vital need to fill the deficiency of essential trace elements for the successful treatment of patients with various pathologies. A lack of minerals can have a significant impact on the function of the gastrointestinal tract, leading to pathological changes in the entire body, premature cell aging, diabetes, and carcinogenesis. It should be borne in mind that trace elements significantly affect the functioning of pro- and antioxidant systems.

Key words: Gastrointestinal tract, macronutrients, trace elements, magnesium, iron, selenium, zinc, manganese, copper.

RELEVANCE

Recent studies have proven the important role of trace element status in the pathogenesis of many diseases of human internal organs and systems [1,2]. The relationship of the mechanisms of ulceration and carcinogenesis in the mucous membrane of the gastrointestinal tract (GIT) with geographical and biogeochemical factors, as well as with the peculiarities of the nutritional stereotype of the population of various regions of the world [2,3] and is also subject to active study. Epidemiological and clinical studies in recent years indicate that the structure of chronic morbidity is dominated by diseases of the digestive system, among which the leading place is occupied by inflammatory diseases of the upper gastrointestinal tract: chronic gastritis, gastroduodenitis. At the same time, studies of the microelement composition of blood serum with inflammatory changes in the mucous membranes of the gastrointestinal tract in most cases revealed a deficiency of trace elements with antioxidant properties.

The homeostasis of micro- and macronutrients is inextricably linked to the functioning of the digestive system [1,2]. In the pathogenesis of any imbalance of the elemental status, there is a mandatory pathogenetic component associated with disruption of the gastrointestinal tract. On the one hand, absorption and assimilation, as well as partial excretion of micro- and macronutrients, depend on the condition of the gastrointestinal tract mucosa; on the other hand, micro- and macronutrients are actively involved in the formation and functioning of the gastrointestinal tract itself [1-4]. Magnesium, calcium, and zinc ions and bioligand compositions with Mg, Zn, and Ca play a special role in maintaining the normal functioning of the gastrointestinal tract [2-11].

Magnesium is one of the key electrolytes of the body and the main intracellular cations. As a universal regulator of biochemical and physiological processes, magnesium participates in energy, plastic and electrolyte metabolism. [1, 5]. Magnesium is extremely important for the normal functioning of the nervous system, including for the normal innervation of the gastrointestinal tract [5, 10]. Magnesium ions reduce the excitability of neurons and the transmission of nerve impulses. This is a kind of natural insulating material in the path of nerve impulses [2]. Given the close relationship between the brain and internal organs, magnesium ions are necessary for the multilateral processes of regulating the digestive system as a whole.

In the clinical picture of a patient with magnesium deficiency, specific neuromuscular signs can be identified, including digestive disorders: diarrhea, sometimes constipation ("irritated" colon), abdominal pain, and a lump in the throat (spasm in the pharynx) [7]. This once again confirms the importance of magnesium ions for the processes of excitability of nerve and muscle cells of the gastrointestinal tract. The role of magnesium sulfate as a laxative and choleric agent is well known [9,10]. In the first case, magnesium sulfate, being slowly absorbed from the intestine, retains water in the intestinal lumen, increasing osmotic pressure, stimulates receptors, peristalsis, and enhances the release of cholecystikinin. The choleric effect of magnesium, its effect on gallbladder tone and kinetics is associated with the irritating effect of this macronutrient. When a magnesium sulfate solution is injected through a duodenal probe, its irritating effect is directly manifested. Reflexes resulting from irritation of the mucous membrane of the duodenum during oral administration of a magnesium-containing drug explain the ejection of bile [12].

Magnesium is necessary to stabilize the activity of the main liver enzymes [12, 13]. At the same time, when the liver is damaged, there is a loss of magnesium, and, consequently, a violation of the main enzyme systems. In particular, a decrease in the activity of Md-dependent ATPASE in the liver of rats treated with sodium arsenate was found [12]. In the presence of Md ions, the maximum activity of acetylneuraminat cytidyl transferase in frog liver and ribose phosphate cyprophoserokinase in rat liver was noted [8]. Magnesium increases the amidase activity of microsomes against the background of the introduction of magnesium into the incubation medium of the microsomal fraction of the rat liver [5].

The inhibitory effect of Mg ions on hydrochloric acid secretion in both the basal and stimulated phases of gastric secretion has been established. In high concentrations, Md blocks the release of acetylcholine and inhibits the acid-forming function of the stomach. The role of magnesium in preventing the action of histamine and gastrin is not excluded [12]. Various conditions and diseases accompanied by magnesium deficiency can cause the development of extracretory insufficiency of the pancreas (against the background of secretin stimulation, Mg increases the secretion of pancreatic enzymes) [4]. Magnesium deficiency occurs quite quickly in intestinal diseases such as ulcerative colitis, Crohn's disease, malabsorption syndrome (celiac disease, lactase deficiency, cystic fibrosis), intestinal infections, etc. The reason for the development of such a deficiency is a violation of the absorption of magnesium in the small intestine [5, 11].

Intracellularly, magnesium performs a multifunctional role, being a substrate and activator of such enzymes as kinases (hexokinase, creatine kinase), ATP-az, cyclases (adenylate cyclase). It is no coincidence that magnesium is able to enhance the coupling of oxidation and phosphorylation in many tissues, including the gastric mucosa [11]. Magnesium deficiency in the diet not only leads to a decrease in the blood flow rate of most organs and an increase in the resistance of peripheral vessels, but can also cause hypertension and a decrease in microcirculation in

capillaries [10]. Thus, based on the idea of adequate blood supply to the gastric mucosa as one of the factors protecting it from aggressive influences, Md ions are among the factors that increase the resistance of the gastric mucosa [4].

Magnesium deficiency is known to increase the body's sensitivity to viral and bacterial infections [6,7]. The restoration of magnesium levels in children potentiates the antibacterial effect of specific antihelicobacter therapy. Magnesium deficiency (a pathological condition classified according to ICD-10 as E61.3) is accompanied by a decrease in the overall immune resistance of the body, which contributes to the formation of various inflammatory diseases of the digestive system [3, 6, 10].

The macronutrient calcium plays key roles in all types of metabolism (protein, mineral, fat, carbohydrate, energy) [13]. Ca ions and bioligand calcium compositions control the processes of bone formation, blood coagulation, intercellular signaling interaction, and membrane formation. Calcium plays an important role in the functioning of the digestive system [7]. Ca ions (like Md ions) are responsible for the motility of the gastrointestinal tract and are an essential component in the processes of excitation and contraction of smooth muscle cells of the digestive tract [9].

Against the background of calcium imbalance, a pathological background arises for the formation of disorders of the motor function of the muscular layer of the gastrointestinal tract in the child's body, which can lead to various gastroenterological pathologies. GASTROINTESTINAL smooth muscle tone disorders are clinically manifested by either hypoperistaltic symptoms (atony, constipation) or hyperperistaltic symptoms (diarrhea, dyskinesia) [13]. A vicious circle is forming against the background of a shortage of Md and Ca. Magnesium-calcium deficiency, causing motor dysfunction of actin filaments and microvilli, leads to further disruption of the absorption of macro- and microelements of the small intestine mucosa, which in turn further exacerbates the motor dysfunction of the gastrointestinal tract and impaired trophic mucosa. In children with chronic gastritis, a method for studying the content of Ca ions in saliva is possible for noninvasive diagnosis of gastroesophageal and duodenogastric reflux [7]. The Ca-Md-dependent process of smooth muscle contraction underlies the regulation of vascular tone of the mucous membrane of the digestive tract, maintaining adequate blood flow to the submucosal layer of the gastrointestinal tract [1] The level of ionized Ca in the blood determines the secretory function of the stomach [7]. Calcium ions are necessary both for "triggering" cellular metabolic reactions underlying the biosynthesis of hydrochloric acid, and for the release of gastric secretion stimulants - acetylcholine, gastrin, histamine. In addition to stimulating the production of hydrochloric acid, Ca activates the activity of the main pepsinogen-producing cells [8]. Under the influence of calcium, gastric secretion increases due to increased blood flow in the gastric mucosa. Calcium deficiency and the associated violation of normal peristalsis and secretory activity, inadequate blood supply to the gastric mucosa, duodenum are important etiological factors in the development of various pathological conditions of the gastrointestinal tract [10].

In addition, Ca ions are involved in the mechanisms of secretion of pancreatic enzymes. The activating effect is manifested both at the level of the acinar cells themselves and through the mediation of the effects of acetylcholine, cholecystikinin - pancreasimine, and cerulein [7,8]. An increase in the level of calcium and a decrease in the level of magnesium in gastric juice, mucosal tissue and parietal gel was revealed in children with peptic ulcer of the stomach and duodenum during the acute phase of the process. Structural changes in the mucous membrane of the upper

gastrointestinal tract and shifts in functional parameters in the examined children were closely correlated [9].

In recent years, the role of calcium ions in cell proliferation and differentiation, including cells of the mucous membrane of the digestive tract, has been established [10]. It has been proven that the deficiency of this macronutrient disrupts the processes of renewal and regeneration of the cell population in intestinal crypts by blocking the cell cycle in phase b1, which in turn leads to a decrease in the healing rate of ulcerative and erosive defects [4].

Zinc is the only metal present in each class of enzymes, and zinc cannot be replaced by any other metal. Despite its low concentration in the blood, its stable bonds with macromolecules and coordination lability make zinc available to all body tissues in order to further meet the need for proteins and enzymes performing various biological functions [5].

Zinc plays an important role in the synthesis of protein and nucleic acids [1,2]. Zinc is a part of the genetic apparatus of a cell, representing up to 100 zinc-containing nucleoproteins and is indispensable at many key stages of gene expression [10]. Therefore, zinc has a direct effect on the growth and proliferation of cells throughout the child's body, including the organs of the digestive system. The rapid renewal of the intestinal epithelium and, consequently, the intensive formation of new cells determines the primary importance of zinc for the mucous membrane of the small intestine and requires its constant presence in the intestine in sufficient quantities [2]. Zinc deficiency in the child's body can result in stunted growth and sexual development, delayed wound healing, impaired taste and appetite, secondary immunological insufficiency, diarrhea, allergic diseases, hair loss, and dermatitis [1,6]. For the first time, zinc was discussed back in the late 80s, when it was proved that children who received artificial milk formulas not enriched with zinc had lower indicators of physical development compared with children receiving mixtures enriched with zinc [7]. Subsequently, a clear relationship was established between zinc deficiency and immune disorders in children and an increased risk of infectious diseases, including diarrhea [8]. There are clinical studies showing that zinc deficiency is detected in infants and young children with prolonged diarrhea. The inclusion of zinc preparations in the treatment of such children makes it possible to relieve the symptoms of diarrhea [4]. The mechanism of action of zinc on the taste analyzer is explained by its presence in a special protein produced in the parotid salivary glands. It has been shown that this protein specifically binds to the membranes of the taste buds, regulating their growth rate and the intake of nutrients into them [1]. The appointment of zinc preparations helps to restore taste perception in children with its congenital disorder. A decrease in the zinc content in children's bodies leads to a deterioration in the function of the taste analyzer, which is restored when zinc-containing foods are introduced into the diet.

The Zn content becomes especially important in conditions of inflammatory lesions of the mucous membrane of the digestive tract [2]. Zinc is a cofactor of repair and regeneration processes due to the antioxidant properties of this trace element in toxicological models and stabilization of the permeability of cytoplasmic membranes damaged as a result of lipid peroxidation [4,2]. Zinc is also supposed to have a stabilizing effect on cytoplasmic membranes, preventing the release of hydrolytic enzymes such as cathepsin O and collagenase, which control the breakdown of damaged tissues. There is evidence that zinc modifies the course of the inflammatory process and accelerates collagen synthesis in a healing wound. The role of zinc in the synthesis of DNA and RNA, which is especially intense in regenerating tissue, should not be discounted [1]. In adult patients with duodenal ulcer, a decrease in plasma zinc levels by 30% of

normal and an increase in zinc content in the mucous membrane of the gastroduodenal region during the period of exacerbation of the disease were found. Apparently, during the period of exacerbation of the inflammatory process in the duodenal mucosa, the need for zinc increases. Zinc is mobilized from the blood, where the zinc level becomes correspondingly low [5,6]. An increase in zinc levels in the tissues of the gastrointestinal tract under conditions of inflammation may be associated with an increase in the activity of enzymes involved in the repair processes [5].

CONCLUSION

Summarizing the above literature data, it is necessary to emphasize once again the significant role of Mg, Ca, and Zn ions in the formation of the structure and maintenance of the functional activity of the digestive system in children. The imbalance of these macro- and microelements is one of the triggers of a cascade of pathological reactions from the gastrointestinal tract and contributes to the progression and chronization of various forms of gastroduodenal pathology.

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