developmental stages, with the highest incidence observed among infants (24%) and toddlers (20%). This pattern suggests that younger children may be particularly susceptible to chloride homeostasis disturbances, possibly related to immature renal regulatory mechanisms or increased vulnerability to dehydration. Hyperkalemia were identified in only 3.8% of those tested. Elevated creatinine kinase levels were observed in 3.8% of whole cases. Notably, both cases occurred in female infants, suggesting a possible age- and sex-specific pattern. Other biochemical parameters (sodium, calcium, magnesium, LDH, phosphorus, glucose) were within normal ranges for the majority of tested patients.

Conclusions. Children with non-epileptic paroxysmal disorders have a variety of metabolic patterns that differ by sex and age. Hyperchloremia emerges as the most prevalent abnormality, affecting nearly half of tested patients with particular frequency in infants and toddlers. This finding highlights the importance of routine electrolyte monitoring in pediatric patients presenting with paroxysmal symptoms and suggests that chloride homeostasis disturbances may contribute to the pathophysiology of these events, either directly or as markers of underlying metabolic stress.

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MECHANISMS OF DEVELOPMENT AND THERAPY METHODS OF ENDOCRINE OPHTHALMOPATHY

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Relevance. The relevance of studying endocrine ophthalmopathy (EO) is due to the high prevalence of this complication of autoimmune thyroid diseases, particularly diffuse toxic goiter (Graves' disease, GD), and the high risk of developing visual impairment leading to patient disability [1]. Understanding the pathogenesis of EO is crucial for developing methods for its prevention and treatment. Furthermore, the treatment of this difficult-to-treat complication of GD remains relevant.

Target. To study the mechanisms of development of endocrine ophthalmopathy and current treatment methods for this condition.

Research methods. The study utilized exploratory, analytical, and comparative methods. International articles published on PubMed and medical guidelines of the Republic of Belarus and the Russian Federation were used.

Results and their discussion. Currently, the main theory behind the development of EO is the immune theory. Specifically, proinflammatory cytokines (lymphokines, growth factors) released by active T-lymphocytes and macrophages lead to increased secretion of glycosaminoglycans by fibroblasts, which causes swelling of the extraocular muscles, swelling of the retrobulbar tissue, and subsequent exophthalmos [2].

Resveratrol, in turn, has the ability to reduce levels of proinflammatory cytokines – interleukin-6, -8, and tumor necrosis factor-alpha (TNF-α), while celastrol not only inhibits the expression of interleukin-6 and -8, but also intercellular adhesion molecule-1 (ICAM-1) and cyclooxygenase (COX-2), and suppresses COX-2-mediated high levels of PGE2. Teprotumab has shown significant improvement in the treatment of moderate to severe EO, compared with intravenous methylprednisolone. Tocilizumab, an interleukin-6 receptor blocker, is an effective treatment option for patients with EO who are steroid-resistant [3]. Thus, surgical intervention, and preferably, radioiodine therapy, if possible, is the most effective method.

Conclusions. Thus, the main link in the pathogenesis of EO in GD is the immune mechanism, with increased production of proinflammatory cytokines and the presence of TSHRs in orbital fibroblasts. In turn, treatment of this severe, disabling complication involves the use of various methylprednisolone regimens depending on the activity of the autoimmune inflammatory process, smoking cessation, the use of corneal moisturizers, the wearing of sunglasses, and the use of selenium and pathogenetic therapy drugs such as podaten, icariin, resveratrol, celastrol, teprotumab, and tocilizumab. Moreover, surgery and, if possible, radioiodine therapy are the most effective methods for preventing the development or progression of EO.

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