Pathogenetic basis of the formation of myopia in children with poly-morbidity

Statement of the Problem: Myopia in childhood and adolescence is the most common cause of visual impairment. Often myopia is seen separately from the concomitant pathology, despite the fact that these diseases prevalent in myopia. Concomitant chronic pathology in children often associated with undifferentiated connective tissue dysplasia. The purpose of this study is to determine the patterns of the formation of axial myopia in children with poly-morbidity.

Methods: Three groups of children aged 11 to 17 years are formed. The main group consisted of 45 children with axial myopia and poly-morbidity. Poly-morbidity included: chronic tonsillitis and bronchial asthma. 38 children had axial myopia without clinical signs of concomitant chronic diseases. 35 children had emmetropia without concomitant chronic diseases. We examined surface phenotype of peripheral blood lymphocytes, the level of retinol and malondialdehyde in the blood.

Results: Significant decrease in the number of CD3+, CD4+, CD20+, CD54+, mIgM lymphocytes, an increase malondialdehyde level and decrease retinol level in the blood in children with myopia and poly-morbidity. In children with myopia without concomitant pathology, there is a significant increase in the number of CD20+, CD95+, CD54+ lymphocytes, a decrease retinol level and an increase malondialdehyde level in the blood.

Conclusion: The patho-genetically grounded concept of formation of myopia in children with poly-morbidity is formulated. Poly-morbidity enhances the growth of the eye due to the acceleration of scleral remodeling. These processes are mediated by activation of lipid peroxidation and disturbance of the formation of nitric oxide. Immuno-depression increases the frequency of exacerbation of chronic pathology which leads to an increase in the number of pro-inflammatory cytokines, affecting scleral fibroblasts. The decrease in the retinol level, due to its consumption as an antioxidant and oxidation to all trans retinoic acid. All trans- retinoic acid through gene expression increases the proliferation of fibroblasts.

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Biography

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