CLINICAL AND DIAGNOSTIC SIGNIFICANCE OF URINARY GLYCOSAMINOGLYCAN DETERMINATION IN COMPUTER VISION SYNDROME

Mamatkhujaev Minkhojiddin Sadirdinkhoji ugli
Basic doctoral student
Andijan State Medical Institute (Andijan, Uzbekistan)

Mamatkhujaeva Gulnarakhan Najmidinovna
MD, Associate Professor
Andijan State Medical Institute (Andijan, Uzbekistan)

Introduction

Computer Vision Syndrome (CVS) is one of the most common functional-metabolic conditions affecting the visual organ, caused by prolonged digital strain. Despite a significant number of studies dedicated to the clinical and functional manifestations of CVS, systemic metabolic mechanisms, including those related to the state of connective tissue, have not been sufficiently studied.

Glycosaminoglycans are key components of the extracellular matrix that determine the hydrophilic and structural-mechanical properties of eye tissues. Changes in their metabolism reflect the processes of remodeling and degradation of proteoglycans and can be considered an objective biochemical marker of connective tissue dysmetabolism. In computer vision syndrome, especially during its prolonged course and in combination with progressive myopia, conditions are created for the activation of these processes, which is accompanied by an increase in the excretion of glycosaminoglycans.

Determination of urinary glycosaminoglycan levels is a non-invasive, reproducible, and objective method that enables the detection of metabolic changes. This indicator is particularly significant for the early diagnosis of patients with computer vision syndrome, including the identification of subclinical forms of metabolic disorders.

Purpose of the study.

The purpose of the study was to assess the nature of changes in the excretion of glycosaminoglycans in urine in patients with computer vision syndrome, depending on the severity of the clinical course.

The aim of the study was to evaluate the nature of changes in urinary glycosaminoglycan excretion in patients with computer vision syndrome, depending on the severity of its clinical course.

Materials and methods.

The object of the study was 160 computer users with clinical signs of computer vision syndrome. The control group consisted of 40 computer users without signs of computer vision syndrome. Comprehensive ophthalmological examination included: determining visual acuity without correction and with correction, sciscopy, autorefractometry, biomicroscopy, study of binocular and accommodation functions, direct and reverse ophthalmoscopy, conducting the Schirmer I test. The results were expressed in mg/mmol of creatinine with mandatory normalization of its concentration. Statistical processing of the data was performed using variation statistics methods; differences were considered significant at $p < 0,05$.

Results and discussion:

In the control group, the level of glycosaminoglycan excretion was 3.36±0.16 mg/mmol of creatinine, which corresponds to the physiological norm and reflects the balanced exchange of connective tissue.

In patients with subclinical levels of computer vision syndrome, the indicator increased to 4.68±0.13 mg/mmol, exceeding the control values by almost 40%. This fact indicates early metabolic involvement of connective tissue, even with minimal clinical manifestations.

In I degree CVS (Computer Vision Syndrome), the level of glycosaminoglycans reached 5.23±0.13 mg/mmol, corresponding to the upper limit of the norm and reflecting the formation of persistent metabolic changes. In patients with grade II computer vision syndrome, a further increase in glycosaminoglycan excretion to 5.90±0.09 mg/mmol was observed, indicating the progression of connective tissue dysmetabolism and characterized by lower variability of the indicator.

Maximum values were recorded in grade III-IV computer vision syndrome - 6.65±0.10 mg/mmol, which is almost twice as high as the indicators of the control group and indicates pronounced remodeling of the extracellular matrix.

The data obtained demonstrate that changes in glycosaminoglycan excretion in computer vision syndrome occur early and are degree-dependent. Even at the subclinical stage of computer vision syndrome, metabolic shifts are detected, preceding the development of pronounced clinical symptoms. As the syndrome progresses in severity, a consistent and nearly linear increase in the level of glycosaminoglycans is observed, reflecting the intensification of proteoglycan degradation and remodeling processes.

It should be noted that even in severe forms of computer vision syndrome, the level of glycosaminoglycans remains within the range of moderate elevation, which distinguishes this syndrome from systemic autoimmune ophthalmopathies and confirms the predominantly metabolic, rather than destructive, nature of the observed changes. This underscores the appropriateness of using glycosaminoglycan excretion as a sensitive, yet non-specific marker of

connective tissue involvement in computer vision syndrome.

Conclusion

Computer vision syndrome is accompanied by increased urinary excretion of glycosaminoglycans, which can be detected even at the subclinical level.

The level of glycosaminoglycans progressively increases as the clinical course of computer vision syndrome worsens.

The determination of glycosaminoglycans in urine can be considered an objective laboratory marker of the metabolic component of computer vision syndrome and an additional criterion for assessing its severity.