Comparative Evaluation of MMP and Cystatin C in Chronic Nephritic Syndrome in Children

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ABSTRACT

Currently, methods of molecular diagnostics have begun to be actively developed, which not only complement traditional research methods, but also provide insight from the point of view of molecular pathophysiology. It is expected that a key role in the diagnosis of kidney disease is increasingly played by the identification of genes and their changes in the course of the disease, which predict the course of diseases. Changes in the chromosomal polymorphic genes of matrix metalloproteinase and its tissue inhibitors, as well as their effect on the glomerular filtration rate in children with chronic glomerulonephritis, determine the prognosis of the disease.

Keywords: Chronic glomerulonephritis, matrix metalloproteinase, tissue inhibitor, Cystatin S.

Introduction.

Due to the significant prevalence of chronic kidney disease and the high level of disability, one of the most important health care tasks is the prevention and effective diagnosis and treatment of children with chronic nephritic syndrome [1,2,3].

In recent years, the genetic mechanisms of the formation of chronic respiratory diseases have become the object of large-scale research throughout the world.

An integral part of preventive programs today is genetic screening. Conducting a molecular genetic study can reduce the incidence of chronic nephritic syndrome in children by identifying a predisposition to its development. At the same time, the identification of gene polymorphism with the manifestation of the syndrome in children can contribute to solving the problems of forming risk groups and implementing preventive measures among these groups, as well as a better understanding of the pathogenesis of this condition [4,5].

In this regard, the question of what is the quantitative contribution of genetic mechanisms to the development of the leading symptoms of nephritic syndrome in children is very relevant. Today, it is promising to study not only genetic and molecular, but also special markers for detecting a disease, such as Cystatin C, leading to the development of the disease.

Purpose of work:

Comparative evaluation of MMP and Cystatin C in children with chronic nephritic syndrome.

Material and methods:

Genetic research of the MMP-9 gene was carried out in the laboratory of immunoregulation of the Institute of Immunology and Human Genomics, Academy of Sciences of the Republic of Uzbekistan. We examined 40 children with chronic nephritic syndrome aged 5 to 17 years who were treated in the nephrology department of a multidisciplinary children's medical center in the city of Samarkand. All examined sick children were determined by the level of MMP-9 polymorphic genes in the blood by PCR analysis.

In the DNA of blood leukocytes of patients, MMP-9 genes were determined. The isolated DNA was analyzed by the standard nucleosorb method using Diatom™ DNAPrep 200 kits (IsoGen Laboratory, Moscow, Russia). Typing of DNA samples was carried out using a specific oligonucleotide primer with gene regions. PCR analysis using a set of reagents for PCR amplification of DNA GenePak™ PCRCore (IsoGen Laboratory LLC).

To standardize the results, the ratio of MMP-9 to the level of Cystatin C in the blood of patients was calculated. Definitions of Cystatin. C were carried out in the SWISS-LAB clinical diagnostic laboratory in Tashkent. Blood sampling was carried out in accordance with the rules of the stage of biochemical studies. Serum was obtained by centrifuging samples at 3000 g for 10 min. The level of cystatin C concentration was studied using commercial kits "KonelabT-Series CYSTATIN-C" (Finland). The determination of cystatin C in the presented kit is based on the principle of immunoturbidimetry.

The measured concentrations are in the range of 0.44-7.0 mg/l. The reference intervals suggested by the reagent manufacturer are 0.40-1.20 mg/l for the age group 5 to 17 years. All patients with CG showed a decrease in MMP-9, which corresponds to the development of inflammation and the accumulation of extracellular matrix proteins, which is a sign of the risk of developing sclerotic changes in the kidney tissue. Accordingly, these changes affect the glomerular filtration rate (GFR), as Cystatin. C is a gold marker of GFR, in all age categories the amount of Cystatin C exceeded the norm, and it was inversely proportional to the level of MMP-9. The connection of MMP with Cystatin C explains the aggravation of the process.

Conclusions:

Monitoring of parameters of MMP-9 and Cystatin C in the immune-inflammatory process in the renal tissue is important in the complex of studies in patients with chronic nephritic syndrome. The definition of this marker is necessary when assessing the development of chronic nephritic syndrome in children. The data obtained can be used for early diagnosis of the sclerotic process, assessment of the prognosis and outcome of the disease, monitoring of ongoing therapy in children with chronic nephritic syndrome.

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