New approaches to the treatment of acute glomerulonephritis

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Acute glomerulonephritis is an acute immuno-inflammatory disease with a predominant lesion of the glomerular apparatus of both kidneys, manifested by renal and systemic manifestations. By origin, glomerulonephritis are divided into primary and secondary (with systemic and dysmetabolic diseases). Along the course, glomerulonephritis are divided into acute, subacute, chronic.

Acute glomerulonephritis can develop after prophylactic vaccinations, when the vaccine and sera act as antigens or destructive factors of immune responses in a previously sensitized body. Sensitization is important in the development of glomerulonephritis after a sting of bees, snakes, etc. In a certain percentage of cases, there is a direct connection between the development of glomerulonephritis and cooling of the body, under the influence of which, apparently, the properties of the protein change with the acquisition of antigenic characteristics (cryoglobulin) or latent streptococcal infection [1].

On the basis of numerous studies, there are two main immune mechanisms responsible for the development of acute glomerulonephritis: 1) primary autoimmune process with the formation of antibodies to the capillaries of the glomeruli, 2) immunocomplex. In the first case, antibodies to the basement membrane of the glomeruli are produced as a result of the acquisition of its antigenic properties, followed by the formation of immune complexes on the territory of the glomeruli and their damage. With this mechanism of development of the disease, observed in humans in 10-15% of cases, the antigen-antibody reaction occurs with the binding of complement and with the participation of the blood coagulation system, histamine, serotonin and other factors. With the immunocomplex mechanism of development of glomerulonephritis, observed in 85–90% of cases, kidney damage occurs under the action of circulating soluble antigen-antibody complexes, which are deposited in the glomerular capillaries. In this case, the antigen can be autogenous or, more often, of exogenous origin (streptococcal, etc.).

At the heart of the formation of anti-inflammatory immunity kidney lies activation of self-defense factors at various levels - extra- and intracellular, as well as on the surface of cells Below are the protective factors, the introduction of which to experimental animals effectively suppresses inflammation in various immuno-inflammatory diseases and is promising for the development of new directions of therapeutic effects on humans.

Interleukin-1 (IL-1) is a pro-inflammatory cytokine produced by inflammatory cells that causes the release and expression of other inflammatory mediators. Tissue inhibitors of matrix metalloproteinases (TIMP), inactivators of leukocytes and resident cells, interleukin-10, regulatory immune cells (T-lymphocytes, macrophages and dendritic cells), lipoxins, beta-chaperones.

Lipoxins promote the formation of M2 macrophages, leading to an increase in their phagocytic activity, not associated with the release of proinflammatory cytokines [2], have an antiproliferative effect on glomerular mesangial cells and are potential inhibitors of vascular endothelial growth factor (VEGF) [3, 4]. In addition, lipoxins can increase renal blood flow and glomerular filtration rate [5].

Insufficient expression of HSP 27 in podocytes can lead to the loss of the normal structure of the filtration barrier of the renal glomeruli and the development of proteinuria [6].

Thus, an increase in the synthesis of HSPs inside the cell in response to various damaging factors, incl. inflammatory, is an adaptive defense mechanism that increases the resistance of cells to cellular stress and prevents cell death by stabilizing and restoring damaged protein molecules [667]. HSPs can be released into the extracellular environment or expressed on the cell surface and thus control inflammation [8]. Strengthening endogenous defense mechanisms by introducing key protective factors capable of modulating inflammation in the kidney and “switching” this process towards limitation is a promising direction of therapeutic action in progressive kidney diseases.

Taking into account the pathogenetic cascade of damage, it is possible to control the processes of treating patients with the help of OTI immune preparations.

table

Anti-inflammatory factors of kidney tissue

Extracellular / cell surface localized	
Cytokine inhibitors	IL-1ra sTNFR Decorin
Protease inhibitors	TIMP
	DAF CR1 C1 inhibitor Factor H Vitronectin
Anti-inflammatory cytokines	IL-10 IL-4 IL-13 TGF - 1
Anti-inflammatory eicosanoids	Prostaglandin E2, I2 Lipoxin A4, B4
Antithrombotic molecules	Tissue factor inhibitor Tissue-type plasminogen activator Urokinase-type plasminogen activator
Intracellular protection	
Warm shock proteins (27, 60, 70, 90) Hemoxygenase-1 Antioxidants (SOD, catalase, glutathione peroxidase) Cyclinase inhibitors (p21, p27)	

Clinical example 1

Patient G., 23 years old, complained of weakness, heaviness in the kidney area,

dysuria, discoloration of urine. At the first visit to a therapist, she was sent to a hospital, where she underwent therapy with a diagnosis of acute glomerulonephritis. However, after hospitalization, the same complaints persisted, with which she applied for bioresonance therapy. When applied in October 2013, hemoglobin 78 g / l, leukocytes 9.2 x 10^{nine}/ l, platelets 455 x 10^{nine}/ l, protein in urine is 1.67, leukocytes 15–20 in p.z., erythrocytes cover the field of view.

The treatment was carried out according to the method of A.A. Hovsepyan, additionally with preparations from OHOM, OTI, Medpharma. Violations of IL 1.6, SD4 were tested.

After two weeks, the scores improved. The color of urine returned to normal, after a month the weakness disappeared. Hemoglobin increased, after 2 months. in the analysis of urine protein was not found, leukocytes 3–4 VPZ, er 1–2 VPZ, hemoglobin 110 in the blood, all indicators are within normal values. Indicators of IL, CD4 are not burdened.

The patient has been observed for 1.4 years without negative dynamics.

Clinical example 2

Patient N., 51, complained of weakness, dysuria, urine of the color of "meat slops", increased blood pressure, edema of the lower extremities. At the first visit to the Medical Center, she was directed to hospitalization in the nephrology department with a diagnosis of acute glomerulonephritis, and a kidney biopsy was recommended to clarify the diagnosis. The patient refused this intervention, received treatment in the hospital with curantil, heparin, doxycycline without clinical and laboratory effect. She applied for treatment in May 2014. In urine during examination b - 0.258 g / l, erythrocytes cover the entire field of view, beats. weight 1005, col. meat slops, leukocytes 3–5 in the PC, hyaline cylinders units. According to b / x without pathology, at to cardiolipin neg., Blood test: hemoglobin - 117, leukocytes - 6.7 x 10^{nine}, platelets - 220, ESR - 17 mm / h. The treatment was carried out with a bioresonance device, drainage preparations of the company "OHOM", psychocorrection with drugs "Medpharma", immunocorrection with drugs of the company "OTI". After 3 weeks, the indicators of urine and blood tests returned to normal. A month later, the patient was offered MRI to exclude brain pathology by a neuropathologist in connection with complaints of polyneuropathy. After this examination, the symptoms of the disease returned and the tests worsened. Repeated therapy according to the method of A.A. Hovsepyan with psychocorrection according to V. Sinelnikov led to stable positive results. The patient is observed for 8 months. without negative dynamics.

Taking into account the onset of the disease before 3 months, the absence of azotemia, the stable clinical state of the patients, there was the possibility of outpatient management with parallel monitoring of the state of the laboratory and by related specialists, which was regarded by the doctors of the clinics as a unique case of self-healing. These examples prove once again that the body's capabilities increase with the correct approach. This approach is possible with bioresonance treatment.

Literature

1. Chebotareva N.V., Bobkova I.N., Kozlovskaya L.V., Li O.A. Significance of violations self-defense mechanisms of the kidney in chronic glomerulonephritis // Clinical Nephrology, 2011 / №1. - S. 8-14.

2. Godson C., Mitchell S., Harvey K. et al. Cutting edge: Lipoxins rapidly stimulate nonphlogistic phagocytosis of apoptotic neutrophils by monocytederived macrophages // J Immunol 2000; 164: 1663–1667.

3. McMahon B., Mitchell D., Shattock R. et al. Lipoxin, leukotriene and PDGF receptors cross-talk to regulate mesangial cell proliferation. FASEB J 2002; 16: 1817-1819.

4. Fierro IM, Kutok JL, Serhan CN Novel lipid mediator regulators of endothelial cell proliferation and migration: Aspirin-triggered-15R-lipoxin A4 and lipoxin A4. J Pharmacol Exp Ther 2002; 300: 385-392.

5. Katoh T., Takahashi K., DeBoer DK et al. Renal hemodynamic actions of lipoxins in rats: A comparative physiological study. Am J Physiol 1992; 263: 436-442.

6. Smoyer WE, Gupta A., Mundel P. et al. Altered expression of glomerular heat shock protein 27 in experimental nephrotic syndrome. J Clin Invest 1996; 97: 2697-2704.

7. Welch WJ Mammalian stress response: Cell physiology, structure / function of stress proteins, and implication for medicine and disease. Physiol Rev 1992; 72: 1063-1081.

8. Multhoff G., Hightower LE Cell surface expression of heat shock proteins and the immune response. Cell Stress Chaperones 1996; 1: 167-176.

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