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THE SIGNIFICANCE OF IMMUNOCOMPLEX MECHANISMS DURING CHRONIC GLOMERULONEPHRITIS

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Resume: A high level of circulating immune complexes in patients with glomerulonephritis is observed in connection with the activation of the immune response to pathogenic antigens, and with a decrease in the mechanism of their elimination. The latter may also be associated with a weakening of the function of cells of the monocyte-macrophage system - cells that eliminate immune complexes. The presence of a high CIC index in chronic GN is an alarming sign indicating an ongoing immuno-inflammatory process in the body.

Keywords: glomerulonephritis, circulating immune complexes.

It has been established that circulating immune complexes are formed after each antigen-antibody encounter and are destroyed by the efforts of mononuclear phagocytes upon completion of complement activation (1,2,4,7,12,58,59,60,61,62). In the case of an excess of antigens, the antibody loses its ability to neutralize the virus, instead of which it forms circulating immune complexes that settle in the kidneys or blood vessels of other organs, causing inflammatory processes there (3,5,6,9,32,34,36,38,55,56,57). The consequence of this is tissue damage and the occurrence of systemic diseases, such as vasculitis, glomerulonephritis, etc. autoimmune diseases.

Glomerulonephritis (GN) is a group of diseases with an immuno–inflammatory lesion of the glomeruli, which can have both acute and chronic course (10,11,13,14,33,35,37,39,41).

The etiology and pathogenesis are similar to those of acute glomerulonephritis, but in addition, the mechanisms of self-progression of the disease play an important role, which can be divided into 2 groups:

immuno-inflammatory and closely related inflammatory-hemocoagulation; non-immune.

Immune - inflammatory mechanisms develop in 2 ways:

formation of autoantibodies to the basement membrane of glomerular capillaries, their fixation on the basement membrane, complement activation and damage to the basement membrane;

formation of circulating immune glomeruli and damage to the latter.

Persistence and chronization of the immune mechanism are caused by the persistence of the etiological factor, insufficiency of local phagocytosis, an unbalanced, timely non-stopping immune response. Persistence of immune mechanisms leads to activation of the kinin system, disturbances in the microcirculation system (15,17,19,21,23,40,42,44,46).

Nonimmune mechanisms of progression of chronic glomerulonephritis:

damage to the renal tubules due to prolonged proteinuria;

According to Brenner's hypothesis, as kidney damage progresses, the function of the fallen, sclerosed nephrons is taken over by the remaining nephrons, which leads to an increase in glomerular filtration in them, in turn, this hyperfiltration damages the glomeruli, contributing to their sclerosis;

arterial hypertension worsens the function of the preserved glomeruli and also leads to their sclerosis.

The root causes of the disease can be different (infections, autoimmune diseases, etc.). The agent enters the blood, circulating immune complexes (CIC) are formed, after which this agent also enters the kidneys (25,27,29,31,43,45,48,51,52,53,54). Further, the CIC or infectious agent has a direct destructive effect on the cell, cell lysis and phagocytosis of immune complexes occur. As a result of this reaction, enzymes are released and the compliment system is activated. These immune complexes or infectious agents can infect the basement membrane (one of the layers of the three-layer filter). After damage to the basement membrane, activation of T-lymphocytes, activation of platelets occurs, resulting in increased blood clotting – hypercoagulation, proliferation of mesangiocytes, as well as the influx of neutrophils, monocytes, lymphocytes with the formation of infiltrates. The whole cascade of inflammation is triggered (16,18,20,22,24,26,28,30,47,49,50).

The basement membrane is affected, mesangial cells and endothelium begin to proliferate. The body reacts very actively to the invasion of foreign agents. If the immune complexes or inflammation are suppressed completely and the immune complexes are removed, the patient recovers. If it was not possible to cope with this and the immune complexes were not completely removed and remained on the membrane, chronic glomerulonephritis is formed.

Immune complexes can also accumulate under the epithelium, inside the membrane, subendothelially and as a result damage the renal filter. The glomerular structure is disrupted, an inflammatory reaction develops, the basement membrane thickens, and all this leads to the development of fibrosis. It is fibrosis that gives rise to impaired renal function and the development of renal failure. From the point of view of functional disorders, it should be noted that in the case of replacement of the filter with fibrous tissue, it does not cope with its task, and the protein begins to pass into the Bowman space, which leads to proteinuria and hematuria, because not only protein begins to pass through the filters, but also shaped elements of blood.

It is usually possible to establish chronic GN when significant, irreversible damage has already occurred in the kidneys with the development of nephrosclerosis and the possibility of pathogenetic therapy that prevents the progression of the pathological process in the kidneys has been missed. The above dictates the need to search for new non-invasive biomarkers, the use of which would allow timely detection of the progression, chronization of GN and prescribe timely pathogenetic treatment of GN.

An essential place in the maintenance of the inflammatory process in the body is given by circulating immune complexes (CIC). The pathogenic effect of CIC is associated with their ability to activate the complement system, which aggravates the development of an inflammatory reaction in the localization zone of viral antigens or cells associated with viral proteins of the body.

All patients with GN had a high rate of both large (CIC, 3%) and small (CIC, 4%) CIC. The difference between the indicators of large and small CICS in groups with and without SAT had a significant difference (P0,05 and P0,01, respectively). Thus, in GN, the level of large and small CICS in the group of patients was almost 2.1 times higher than the same indicators in the control group, which consisted of healthy volunteers, without clinical, anamnestic and laboratory indicators of renal tissue damage. A high level of CIC in patients with GN is observed in connection

with the activation of the immune response to pathogenic antigens, and with a decrease in the mechanism of their elimination. The latter may also be associated with a weakening of the function of cells of the monocyte-macrophage system - cells that eliminate immune complexes.

Thus, the presence of a high CIC index in chronic GN is an alarming sign indicating an ongoing immuno-inflammatory process in the body.

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