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CHRONIC RENAL FAILURE CHRONIC GLOMERULONEPHRITIS

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Abstract: Kidney disease (CK) is a disease that occurs when the condition of the kidney deteriorates. As a result, people's lives become worse. Kidney disease affects one in ten people worldwide. This is one of the main reasons for high medical costs. In high-income countries, the cost of transplantation and dialysis is between 2% and 3% of the annual medical budget [1]. Chronic kidney failure makes it difficult for the body to remove excess fluid from the blood. Advanced kidney disease can cause dangerous levels of fluid, electrolytes, and waste products to accumulate in the body. This can lead to complications such as high blood pressure, anemia, bone weakness, and nerve damage.

Key words: Kidney disease, chronic glomerulonephritis, macrohematuria, tubulointerstitial.

After getting acquainted with all types of glomerulopathies, we should return to consider the sad consequence of these conditions - chronic glomerulonephritis, which is the main cause of chronic kidney failure. More than 60% of patients diagnosed with "chronic glomerulonephritis" need permanent hemodialysis or a new kidney transplant. It is assumed that chronic glomerulonephritis begins mainly as a result of focal glomerulosclerosis, membranous and membranous-proliferative glomerulonephritis. Most chronic glomerulonephritis is observed in young and middle-aged people. In classical cases, the kidneys do not swell symmetrically. Their surface becomes grainy. When examined with a microscope, glomeruli and the cavity of Shumlyansky-Bowman's capsule are sclerosed in all cases, sometimes there is complete hyalinosis in the balls. Clotting of glomeruli is the last stage of any glomerulonephritis, and it is difficult to determine what pathological process started in the places where the glomeruli are closed. It should be noted that progressive glomerular sclerosis or hyalinosis disrupts blood

circulation between the afferent arteriole and the efferent arteriole, which in turn causes secondary damage to the glomerulus. causes atrophy and formation of fibrotic tissue in them. The walls of medium- and small-caliber arteries are thickened and their path narrows, which causes secondary hypertension and atrophy of renal parenchymatous elements. Chronic glomerulonephritis: glomerular hyalinosis. An infiltration consisting of lymphocytes and plasmocytes is found in the interstitium. As the pathological process worsens, all the components of the kidney are damaged, and accordingly, kidney failure begins as a result.

In most cases, chronic glomerulonephritis begins suddenly, and the pain becomes apparent at a very late stage, after the symptoms of kidney failure appear. Some patients may develop nephrotic or nephritic syndrome. As the glomerular pathway becomes blocked (obliteration), the pathway for protein excretion along with urine becomes blocked, which leads to worsening of nephrotic syndrome. Hypertension, like microhematuria, is a constant clinical symptom. Macrohematuria is observed occasionally. The outcome of this disease is bad, progressive uremia leads to death. Counting from the time the first symptoms of the disease appear, the patient can live up to 10 years on average. Dialysis prolongs the life of patients with chronic glomerulonephritis. As can be seen from the above-mentioned data, secondary pathological processes can begin in the interstitium and tubules when the glomeruli are damaged. However, in some kidney diseases, it is observed that the interstitium and tubules are damaged in the primary order. In this case, the tubules alone are damaged, but the interstitium survives. All tubulointerstitial nephritis are divided into two main groups: I) tubules and interstitium are actually damaged due to inflammation (tubulointerstitial nephritis), 2) tubules are actually damaged due to ischemia or due to the influence of poisons, such an event begins with acute tubular nephrosis, acute kidney failure leads to exacerbation Tubulointerstitial nephritis is divided into infectious and non-infectious types, depending on the reasons for which it appeared. The first of these includes pyelonephritis due to bacteria, and the second includes damage to the tubules due to the effects of drugs and metabolic changes (hypokalemia). Such nephritis may be immune-related in nature. Interstitial nephritis, which belongs to the second group, is divided into acute and chronic types, depending on how it progresses.

Drug-related acute tubulointerstitial nephritis is a hypersensitivity reaction to drugs, mainly synthetic penicillins, diuretics, and nonsteroidal anti-inflammatory drugs. Such a reaction usually begins a few days after taking the drug (latent period). The temperature rises, polymorphous rashes appear on the skin, eosmophilia, hematuria, a little proteinuria begin, and eosinophils appear in the urine. Renal function can be impaired to varying degrees. Histological examination of kidney biopsies reveals the presence of interstitial edema, peritubular mononuclear infiltration, tubular necrosis. Interstitial infiltrates also contain neutrophils and eosinophils. Eosinophilia, mononuclear infiltration in the interstitium, and the presence of immunoglobulin G along the tubular basement membrane indicate that acute interstitial nephritis is immune in nature. In addition, an increase in the amount of E immunoglobulins in the blood is characteristic. However, the type of antigen involved in the initiation of drug-related acute interstitial nephritis and the type of hypersensitivity reaction have not yet been determined. As the drug or its metabolites pass through the kidneys, they are thought to be filtered into the primary urine and then reabsorbed into the tubules, damaging their basement membrane. The drug becomes a complete antigen, which combines with the proteins of the basement membrane and causes the initiation of an immunological reaction and the deposition of immune complexes on the membrane.

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