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Clinical Signs and Treatment of Patients with Chronic Kidney Disease

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Abstract: Chronic kidney disease is a long-term disease, a progressive decline in renal function. Symptoms develop gradually and in advanced stages include anorexia, nausea, vomiting, nocturia, lethargy, chronic fatigue, itching, and edema. The general condition and working capacity of patients progressively worsens.

Keywords: kidney disease, treatment, Clinical.

Chronic renal failure is a slowly progressive impairment of renal excretory function, lasting for months or years, and defined by a decrease in glomerular filtration rate below normal, which is usually indirectly determined by measuring serum creatinine. In contrast to acute renal failure, it develops gradually due to progressive irreversible loss of the kidney parenchyma. The number of functioning nephrons decreases. The risk of developing and progressing chronic kidney disease is increased by a number of widely accepted factors. Arterial hypertension, diabetes mellitus, hyperlipidemia, obesity, smoking, lower urinary tract infections and urinary tract obstruction, autoimmune diseases and others are common causes of chronic kidney disease. Toxic damage to the kidneys, alcohol and its surrogates, exposure to lead, mercury, fungicides, disinfectants, heroin, organic solvents adversely affect the kidney parenchyma. Chronic kidney disease can result from many other causes, from kidney loss due to trauma to hereditary diseases such as polycystic kidney disease.

There are four stages. Polyuric - clinical manifestations associated with the underlying disease, as well as polyuria, nocturia and isosthenuria. Azotemic, oligoanuric - the stage of clinical manifestations, the appearance of intoxication: anorexia, neurological disorders (headache, apathy, decreased vision, insomnia), pain in the bones and joints, itching. Dyspeptic disorders appear - diarrhea, vomiting. From the side of the cardiovascular system - tachycardia, arrhythmia. Stage of decompensation - stomatitis, gingivitis, pleurisy, pericarditis, pulmonary edema join. Terminal (uremic, anuric) stage. The outcome is usually fatal, the only way out in this situation is a radical kidney transplant. This stage can also be delayed by lifelong hemodialysis. The concentrations of creatinine and urea in the blood (which are largely dependent on glomerular filtration) begin to rise hyperbolically. Urea and creatinine levels are not the main symptoms of uremia; they are markers for many other substances (some not yet identified) that lead to symptoms. Plasma sodium concentrations are usually normal, and hypervolemia occurs only infrequently with limited or excessive intake of sodium or water. Heart failure develops due to sodium and water overload, in particular in patients with reduced cardiac reserve. For substances whose secretion is controlled primarily through distal nephron secretion (eg, potassium), renal adaptation is usually normal, unless renal failure is advanced or potassium intake in the diet is excessive. Anorexia, nausea, vomiting, weight loss, stomatitis, and bad taste in the mouth are very common. Skin tone may turn yellow-brown. Sometimes urea crystals are shed on the surface of the skin with sweat, forming a uremic frost. Itching can cause serious discomfort. Nutritional deficiencies leading to generalized tissue loss are the hallmark of chronic uremia. Diagnosis is based on laboratory tests of renal function, supplemented if necessary by renal biopsy. Initially, the

loss of function of the kidney tissue has almost no obvious pathological manifestations, because the remaining tissue is working hard (functional adaptation of the kidneys). Laboratory diagnosis of glomerular filtration. Electrolytes, the concentration of urea nitrogen in the blood, creatinine, phosphates, calcium, complete blood count show the pathology and help with the differentiation of diseases. If necessary, an ultrasound examination is performed, which reveals qualitative pathologies of the kidneys. Ultrasound of the kidneys usually helps to identify obstructive uropathy and differentiate acute renal failure from CKD based on the size of the kidneys. If ultrasound reveals reduced sclerosed kidneys; the high risk of the procedure outweighs the low diagnostic value. Chronic kidney disease is a lesion of an organ that persists for three months or more due to the action of various etiological factors, the anatomical basis of which is the process of replacing normal anatomical structures with fibrosis, leading to its dysfunction. Fibrosis is an irreversible, chronic condition that occurs in response to damage to compartments or individual cell populations of the kidney. The progression of replacement fibrosis and the degree of its severity determines the degree of impaired renal function. Fibrosis can be the result of both acute processes with the development of necrobiosis or apoptosis of cell populations, and slowly progressive pathological processes (subcellular, cellular, tissue levels) associated with the action of various etiological factors. The identification of such chronic processes also forms the basis of the clinical diagnosis of CKD.There are three treatment options for chronic kidney disease: medical (non-surgical) treatment, dialysis, or a kidney transplant. In kidney diseases, treatment consists of specific treatment for a specific disease and nephroprotective treatment, universal for all kidney pathologies. Specific treatment is prescribed depending on the specific disease. With glomerulonephritis, kidney damage in systemic connective tissue diseases, steroids are used. With infectious lesions of the kidneys and urinary tract - antibiotics. In diabetic nephropathy - correction of blood glucose levels. The main in nephroprotective treatment is the blockade of the renin-angiotensin-aldosterone system due to several groups of drugs: angiotensin-converting enzyme blockers, angiotensin receptor blockers, aldosterone antagonists, direct renin inhibitors, etc. The most important is the treatment that reduces the level of proteinuria by normalizing intraglomerular hypertension (blockade of the RAAS) and protection of the proximal epithelium from toxic endocytosis of proteins (antioxidants). A non-specific but important treatment is antihypertensive therapy for concomitant hypertension). All patients with chronic kidney disease initially begin conservative management (medication, dietary advice and monitoring).

Serious damage in chronic kidney disease (end-stage kidney disease) requires dialysis or transplantation.

It is most effective to initiate appropriate therapy in the early stages of CKD. Most patients do very well in the early stages of CKD and have no symptoms with proper therapy. Due to the lack of symptoms, many patients and their families do not recognize the seriousness of the disease and stop taking medication and dietary restrictions. Discontinuation of therapy can lead to a rapid deterioration in kidney damage and in a short time such patients may need such expensive treatment as dialysis or kidney transplant.

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