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PATHOGENESIS AND TREATMENT SCHEME OF RENAL DYSFUNCTION IN SYSTEMIC SCLEROSIS AND SYSTEMIC LUPUS ERYTHEMATOSUS.

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Abstract:

Systemic connective tissue diseases such as systemic sclerosis and systemic lupus erythematosus often result in significant renal complications. These kidney pathologies, including scleroderma nephropathy and lupus nephritis, are associated with immune dysregulation, the presence of autoantibodies, and cytokine-induced fibrosis or immune complex deposition. Early detection of renal dysfunction in these conditions is crucial due to their contribution to increased morbidity and mortality. This article explores the pathogenesis and clinical features of kidney involvement in both diseases and outlines contemporary treatment strategies, including the use of corticosteroids, cytotoxic drugs, and antihypertensive agents. Timely diagnosis and personalized therapy can significantly improve patient outcomes and reduce long-term complications.

Keywords: Systemic sclerosis, systemic lupus erythematosus, renal dysfunction, scleroderma nephropathy, lupus nephritis, autoimmune diseases, kidney biopsy, cytokines, immune complexes, ACE inhibitors, cyclophosphamide, autoantibodies, glomerulonephritis, hypertension.

INTRODUCTION: Subclinical or overt signs of kidney damage have a significant adverse effect on the clinical course of connective tissue systemic diseases. Kidney dysfunction is characteristic of systemic connective tissue diseases such as systemic lupus erythematosus, rheumatoid arthritis, systemic scleroderma, Sjogren's syndrome, dermatomyositis, and polymyositis. In patients with systemic scleroderma, the development of chronic kidney disease is 5%, while in systemic lupus erythematosus it is 50% (1). Kidney organs are the most involved in the pathological process in systemic diseases of the kidney connective tissue. Kidney damage can develop directly as a complication of systemic diseases or as a result of drugs used in their treatment. The development of kidney dysfunction in all systemic diseases of the connective tissue depends on almost the same pathogenetic mechanism, ie pathophysiological processes caused by the influence of autoantibodies. But it differs in some aspects.

SYSTEMIC SCLERODERMA: Systemic scleroderma is an autoimmune disease of connective tissue, the main clinical symptoms of which are widespread microcirculation disorders, fibrosis of the skin and internal organs. Severe and often sudden kidney damage can occur in patients with diffuse scleroderma and antibodies against RNA polymerase III, often within the first 4-5 years of the disease (2). The leading mechanism of scleroderma nephropathy is associated with the development of antibodies to vascular endothelial cells in patients with systemic scleroderma. In scleroderma, the factor leading to the acute development of renal pathology is renal vasospasm, which damages the vascular endothelium, develops proliferative angiopathy, thrombosis, and fibrinoid necrosis of small arteries and arterioles. Activation of endothelium, fibroblasts and immune cells, lymphocytes, monocytes leads to increased synthesis of cytokines and growth factors (IL-1, 4, 6, 8, TGF-β, PDGF) that stimulate collagen production and sclerosis processes. Two proteins have been implicated in the pathogenesis of scleroderma nephropathy (3): GPATCH2L and CTNND2. Overexpression of GPATCH2L by the renal tubular epithelium leads to changes in renal artery tone and intravascular volume, which are closely related to the development of arterial hypertension. Increased activation of the CTNND2 protein is likely associated with endothelial dysfunction and causes the development of vasculitis and thrombotic microangiopathy. Clinical signs of the disease in the acute form disappear with rapidly progressive kidney failure and severe arterial hypertension. Chronic nephropathy occurs with the gradual development of changes in urine, hypertension and nephrotic syndrome. Diagnosis is based on clinical and laboratory indicators of general nasopharyngeal analysis, general blood analysis, biochemical analysis of blood, determination of autoimmune markers, and final diagnosis is based on kidney biopsy. Changes in the

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general urinalysis are represented by microhematuria, proteinuria and cylindruria. A general blood test is characterized by thrombocytopenia and hemolytic anemia. Biochemical analysis reveals an increased amount of calcium, hypercreatininemia, hyperkalemia, and hypoproteinemia. The results of the Rehberg-Tareev test show a decrease in glomerular filtration. Studies of specific antinuclear antibodies are still ongoing. (3)

Treatment of sclerodermic nephropathy.

-Nonsteroidal anti-inflammatory drugs. These are widely used due to their anti-inflammatory and pain-relieving properties. One of the side effects of nonsteroidal anti-inflammatory drugs is a sharp decrease in glomerular filtration.

-Methotrexate

- Antibacterial drugs

- cyclophosphamide can be used together with corticosteroids

Angiotensin-converting enzyme inhibitors or angiotensin II receptor antagonists should be used to eliminate arterial hypertension.

SYSTEMIC LUPUS ERYTHEMATOSUS: Systemic lupus erythematosus is a chronic autoimmune disease with a multivariable appearance, course and prognosis, which develops on the background of a genetically determined imperfection of the immune system and is characterized by the formation of large-scale autoantibodies to the components of nuclear cells. Cytoplasm and membranes, disruption of cellular immunity, resulting in the development of autoimmune inflammation. Kidney pathology in the form of chronic glomerulonephritis develops in 40-60% of patients with systemic lupus erythematosus. Its pathophysiology is based on the mechanism of immune complex formation. The immune complex is composed of: antinuclear antibodies, high IgG antinuclear antibody binding complement, anti-DNA antibody (4).

Clinical signs of renal dysfunction in systemic lupus erythematosus may include nephrotic edema and arterial hypertension along with the underlying disease. Changes in the functional state of the kidneys are evaluated by determining the amount of creatinine in the total urine and blood. Microalbuminuria, proteinuria, and hematuria are indicators that we should pay attention to in the general urine analysis. In patients with such changes, it is necessary to think about hypertension of unknown etiology and other pathologies of the kidneys. The final conclusion about kidney damage is made after a biopsy.

Treatment of systemic lupus erythematosus nephropathy.

- as an antihypertensive treatment, angiotensin-converting enzyme inhibitors are used

- cyclophosphamide can be used together with corticosteroids in active and recurrent nephritis.

When the level of the disease is high, cyclophosphamide can be dripped into the vein, and prednisolone should be taken 50-60 mg orally 1 time per day for 6-12 months. It is necessary to determine the individual and single amount of Prednisolone. Anticoagulants can also be used if thrombotic conditions are observed.

CONCLUSION: Renal dysfunction is one of the most severe and life-threatening complications of systemic connective tissue diseases such as systemic sclerosis and systemic lupus erythematosus. The pathogenesis of these conditions involves immune complex formation, autoantibody production, and cytokine-mediated inflammation, leading to fibrosis and vascular damage. Kidney involvement is often diagnosed late, contributing to increased rates of disability and mortality. Therefore, early detection of renal function abnormalities and timely initiation of individualized, comprehensive treatment are crucial for improving patient quality of life and slowing disease progression. The use of modern diagnostic methods and immunosuppressive therapies, along with control of arterial hypertension and prevention of cardiovascular complications, can significantly prolong patient survival.

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