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OPTIMIZATION OF DIAGNOSIS AND TREATMENT OF HIV-INFECTED PATIENTS WITH RENAL PATHOLOGY

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Abstract:

The review analyzes the prevalence, pathogenesis, diagnosis, and treatment of HIV infection. The main clinical and morphological variants of kidney involvement in HIV infection are presented. The prevalence of kidney damage in HIV infection ranges from 20% to 30% and includes clinical and morphological variants such as HIV-associated nephropathy (HIVAN), immune complex HIV-associated kidney disease, and thrombotic microangiopathy. In individuals with HIV infection who are not receiving antiretroviral therapy (ART), the most common type of kidney disease is HIVAN.

Keywords: HIV, HIV-associated nephropathy, immune system, kidney damage, HIV infection

Among human infectious diseases that are accompanied by progressive changes in the immune system, HIV infection remains one of the most pressing issues in modern medicine. The prevalence of HIV infection, which affects all layers of the world's population, is estimated to be in a very wide range. HIV targets immune system cells that have CD4 receptors on their surface: T-helper cells, monocytes, macrophages, dendritic cells, and microglial cells. There is evidence of the virus's tropism for the epithelium of the rectum, Langerhans islets, thymic cells, lymph nodes, and the spleen. According to current data, HIV circulates in the internal fluids of the infected person's body in the form of a viral particle – a virion, which contains the virus's ribonucleic acids (RNA).

According to research, the kidneys are among the organs that are subjected to various infectious and non-infectious influences. Numerous clinical studies have shown that the kidneys are damaged by HIV infection as a result of direct HIV exposure, and later, as kidney diseases progress, other secondary factors may contribute. These include the use of narcotic substances (injectable drugs), the presence of opportunistic diseases (OD), co-infections (hepatitis C and B viruses), the use of nephrotoxic drugs to treat OD and related conditions, vascular pathology, arterial hypertension (AH), and diabetes mellitus (DM). As for the hepatitis viruses, in individuals with HIV, the prevalence of HCV infection is high, and in drug-dependent patients, it exceeds 70%.

According to researchers, kidney pathology in HIV infection is detected in 20-30% of cases. In a cross-sectional study by A.P. Rebrov and colleagues (2015) involving 65 individuals with HIV infection, signs of kidney damage were observed in 51% (33) of cases. Among patients in the nephrology department, the leading manifestations of kidney diseases were proteinuria of varying severity (up to nephrotic syndrome), macrohematuria, and azotemia. Kidney damage in HIV infection can clinically begin with manifestations of acute or chronic nephropathy. One of the causes of acute kidney injury (AKI) in HIV infection is myopathies, which occur in 30% of cases and are accompanied by elevated levels of creatine kinase, primarily the CK-MM fraction. Before the introduction of highly active ART, the most common findings in HIV-infected individuals were acute tubular necrosis, hemolytic-uremic syndrome, and others. Causes of AKI in individuals with HIV infection include antiretroviral drugs such as indinavir and tenofovir. The pathogenesis of AKI in HIV infection is complex and involves drug overdose, comorbidities such as diabetes mellitus (DM), arterial hypertension (AH), opportunistic diseases (OD), sepsis, the use of medications to treat secondary infections, crystalluria, and others. Currently, kidney damage caused by direct HIV exposure includes: HIV-associated nephropathy (HIVAN), immune complex HIV-associated kidney disease (HIV-ICKD), and thrombotic microangiopathy.

HIV-associated kidney disease. According to current data, the persistence of HIV causes antigenic stimulation, the formation of antibodies to the virus, and the deposition of immune complexes in the kidneys. IgM and IgG can bind circulating HIV antigens, resulting in the formation of circulating immune complexes (CIC). At all stages of HIV infection, CICs are formed, which, in turn, express pro-inflammatory factors in kidney tissues, cause cell death through necrosis, apoptosis, or cellular dysfunction,

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increase matrix synthesis, decrease matrix degradation, release cytokines, chemokines, adhesion molecules, and growth factors. The histological substrate of this disease is membranous, diffuse, or membranous-proliferative glomerulonephritis. The clinical manifestations and course of HIV-associated kidney disease (HIV-AKD) do not differ from those in the general population and are characterized by acute nephritic and/or nephrotic syndromes, accompanied by dysmorphic erythrocyturia, impaired kidney function, and hypertension (HT). According to several researchers, immunoglobulin A nephropathy in HIV infection is latent with minimal changes in urinary sediment. The relationship between immunoglobulin A nephropathy and HIV was confirmed by the presence of immune complexes with HIV antigens in the blood and kidney tissue of patients. There is evidence that timely diagnosis and adequate ART significantly reduce the risks of HIV-AKD progression. In a retrospective study by O. Muñoz-Velandia and colleagues (2020), the immunologically mediated kidney disease related to HIV infection was analyzed. According to the study results, among 1509 HIV-positive individuals, HIV-AKD was identified in 22. The researchers reported a cumulative incidence of 1.45%. At the time of HIV-AKD diagnosis, total kidney function, i.e., estimated glomerular filtration rate (eGFR), was higher than 30 mL/min in 90.8% of patients, and 77.2% had subnephrotic proteinuria. The study demonstrated that the number of CD4 cells and the CD4/CD8 ratio influence the nitrogen-excreting function in HIV-AKD. In a cross-sectional study by A. Aliyannissa and colleagues (2020), correlations between the number of CD4 cells and calculated eGFR, as well as the protein/creatinine ratio in urine, were studied in HIV-positive children. At the time of the study, all participants were receiving ART. The study found that the number of CD4 cells positively correlated with calculated eGFR ($r=0.473$; $p=0.001$) and negatively correlated with the protein/creatinine ratio in urine ($r=-0.284$, $p=0.034$). A noteworthy finding from this study is that individuals with HIV infection had optimal kidney function (eGFR 90 mL/min/1.73 m²). Proteinuria was detected in 12 patients (28.6%) and was not significantly associated with the clinical stages of HIV infection. These data suggest that in HIV infection, the degree of immunodeficiency correlates with the severity of kidney damage. Therefore, it is important to monitor not only the level of immune-regulatory cells but also kidney function in individuals with HIV infection, regardless of disease stage. T. Fiseha and A. Gebreweld evaluated the prevalence and factors affecting eGFR among Ethiopian HIV-positive patients at baseline before starting ART and during subsequent follow-up. The researchers assessed kidney function using eGFR calculated by the MDRD method. Among 353 patients, 70 (19.8%) already showed signs of renal insufficiency (eGFR <60 mL/min/1.73 m²). Factors associated with decreased kidney function included female sex (odds ratio (OR) 3.52, 95% confidence interval (CI) 1.75–7.09), CD4 count <200 cells/mm³ (OR 2.75, 95% CI 1.40–5.42), body mass index (BMI) <25 kg/m² (OR 3.04, 95% CI 1.15–8.92), hemoglobin concentration (OR 2.19, 95% CI 1.16–4.09), and total cholesterol level (OR 3.15; 95% CI 1.68–5.92). Notably, the prevalence of renal insufficiency in this study increased from 19.8% (initially) to 22.1% during the follow-up period. Throughout the follow-up period, factors contributing to the development of renal insufficiency included older age (OR 3.85, 95% CI 2.03–7.31), female sex (OR 4.18, 95% CI 2.08–8.40), low baseline CD4 count (OR 2.41, 95% CI 1.24–4.69), low current CD4 count (OR 2.32, 95% CI 1.15–4.68), high BMI (OR 2.91, 95% CI 1.49–5.71), and low hemoglobin (OR 3.38, 95% CI 2.00–7.46).

HIV infection diagnosis: The diagnosis of HIV infection is made by an infectious disease doctor at the AIDS Center through a comprehensive assessment of epidemiological data, clinical examination results, and laboratory tests. The first step and standard method of laboratory diagnosis for HIV infection is the simultaneous detection of antibodies to HIV 1, 2, and the HIV p24 antigen. The p24 antigen can be detected on the 15th day, the first antibodies on the 30th day, and the later ones in 3-6 months. At the AIDS Center laboratory, you can undergo an HIV test for detecting antibodies and antigens. In addition to the standard HIV test, which involves taking blood from a vein, there are express tests for HIV. These tests can be done with a drop of blood from the finger or from saliva. Such tests are mostly aimed at detecting antibodies. Express tests are used in mobile testing units, at outreach events for awareness, counseling, and voluntary HIV testing. It is the law: every HIV test using simple/rapid tests must be accompanied by a blood test using standard methods to detect antibodies to HIV 1, 2, and p24 antigen or the patient must be referred for testing using standard methods.

HIV infection drug therapy includes basic therapy (which is determined by the stage of the disease and the level of CD4+ lymphocytes) and therapy for secondary and associated diseases. Basic therapy refers to treatment that is determined by the stage and phase of the disease, as well as the laboratory markers of HIV infection progression (CD4+

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lymphocyte levels and HIV RNA levels). Basic therapy includes antiretroviral therapy and chemoprophylaxis for secondary diseases.

Currently, the main component of treatment for HIV-infected patients is antiretroviral therapy (ART), which allows for controlled disease progression. This means a state where, despite the impossibility of complete cure, it is possible to halt the progression of the disease, achieve regression of secondary diseases (if they have developed), and restore the patient's ability to work.

Antiretroviral therapy (ART) is based on prescribing drugs to suppress HIV replication. These drugs are called antiretroviral drugs.

Conclusion. Analysis of the available literature data allows us to conclude that various mechanisms of kidney disease development in individuals with HIV infection are well known today. The increasing number of patients suffering from both HIV infection and chronic kidney disease has significant epidemiological and clinical implications. Despite the use of highly active antiretroviral therapy and adjunctive therapy for HIV infection, kidney damage remains a leading factor in the prognosis of this disease, which underscores the need for early detection of kidney damage during the early stages of HIV infection in order to prevent unfavorable outcomes at later stages. Considering that kidney damage, regardless of the stage of HIV infection, is characterized by a progressive course with the development of severe complications, timely diagnosis of HIV-associated chronic nephropathies will determine the treatment strategy, which will help prevent or slow the progression of the disease.

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