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DIFFICULTIES IN DIAGNOSING THE HUMAN IMMUNODEFICIENCY VIRUS (HIV) OCCURRING UNDER THE GUISE OF SYSTEMIC LUPUS ERYTHEMATOSUS

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Abstract:

In the state of radiation, the differential diagnosis of systemic lupus erythematosus and HIV infection is related to the similarity of the clinical picture and etix diseaseThe resulting viral particles attack new cells, causing cell death and disrupting their interaction with other cells, leading to progressive changes in immunity - immunodeficiency. As a result of the immune shifts, resistance to secondary infections and neoplasms is reduced. In addition, due to direct cytotoxic action or as a result of indirect action (autoimmune mechanisms) the cells of the nervous system, blood, cardiovascular, musculoskeletal, endocrine and other systems may be affected. The pathological processes caused by HIV are superimposed on a wide range of changes caused by secondary diseases that have developed against a background of progressive immunodeficiency. All this leads to multiple organ involvement and a variety of clinical symptoms of HiV infection, bringing it closer to systemic diseases of connective tissue, especially systemic lupus erythematosus (SLE).

Keywords: systemic lupus erythematosus, HIV-infection, differential diagnosis.

INTRODUCTION. Rheumatic masks of HIV infection are a variety of lesions of the peripheral joints and spine, as well as systemic manifestations that accompany the development of this infection and mimic true rheumatic diseases and syndromes (synonymous with rheumatic syndromes in HIV infection). In addition to HIV itself, such syndromes are known for a number of opportunistic infections (fungal, mycoplasmal, herpesvirus, etc.). Finally, of particular interest is the change in the clinical course of pre-existing rheumatic diseases in the case of infection of patients with HIV.

According to modern concepts, HIV infection is a disease caused by the human immunodeficiency virus (HIV), which develops after a long-term persistence of the pathogen in lymphocytes, macrophages and cells of the nervous tissue and is characterized by slowly progressive dysfunction of the immune system. HIV-infected people are people in whose body HIV is detected; this category includes both HIV carriers and patients with clinical manifestations of the infection, including those with AIDS. Acquired immunodeficiency syndrome (AIDS) is a particularly dangerous infectious disease, the terminal stage of HIV infection, which occurs with damage to the immune and nervous systems, manifested by the development of severe viral, bacterial, parasitic lesions and / or malignant neoplasms that lead to the death of the patient. Patients with AIDS are people with a variety of pathological manifestations caused by a deep defeat of the immune system by HIV infection [1,11].

The causative agent of AIDS - a retrovirus, first discovered in 1983 in Paris by R. Ugallo and Coobi, circulates in the body of an infected person in the form of a viral particle - a virion containing the RNA of the virus. Possessing the highest affinity for CD4-lymphocytes, it infects them, penetrating into the cell genome, as a result of which the lymphocyte begins to produce new viral particles containing HIV RNA.

The resulting viral particles attack new cells, causing their death and disrupting their interaction with other cells, which leads to progressive changes in immunity - immunodeficiency. As a result of changes in immunity, resistance to secondary infections and neoplasms decreases. In addition, due to a direct cytotoxic effect or as a result of an indirect effect (autoimmune mechanisms), cells of the nervous system, blood, cardiovascular, musculoskeletal, endocrine and other systems may be damaged.

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against the background of progressive immunodeficiency. All this causes multiple organ lesions and a variety of clinical symptoms of HIV infection, bringing it closer to systemic connective tissue diseases, primarily systemic lupus erythematosus (SLE)

A distinctive feature of SLE - a disease from the group of diffuse connective tissue diseases (DBST) - is the similarity of immune disorders with HIV infection discovered in recent years. SLE develops on the basis of a genetically determined imperfection of immunoregulatory processes [1,3], leading to the formation of many antibodies, immunocomplex inflammation, which results in damage to organs and systems. There are a number of indirect confirmations of the starting role of RNA-containing and so-called slow viruses (retroviruses) in the development of the pathological process.

Combine SLE and HIV infection with such clinical symptoms as dermatitis, which has a similar localization on the cheeks and wings of the nose, lymphadenopathy, multiple organ pathology, the addition of various infections against the background of immunodeficiency, etc. [3]. The classic course of SLE in most cases begins with fever, fatigue, joint pain with swelling, as well as the appearance of various skin rashes, which are more often localized on the face, neck and chest.

Less often in the debut, photosensitization, serositis (pleurisy and pericarditis), arthritis (often symmetrical) of the small joints of the hands are encountered. Possible damage to the wrist and knee joints. Morning stiffness is not typical. For skin lesions, the appearance of vascular erythema is typical, passing without a trace and discoid foci with hyperemic edges, infiltration, cicatricial deformity and depigmentation in the center. Erythema is most often located in the nose and cheeks with the formation of a "butterfly" figure. Less commonly, at the onset of SLE, dry or effusion pleurisy, heart damage (pericarditis, myocarditis), kidney damage (proteinuria), nervous system damage (migraine, mental disorder, neuropathy, thrombosis) are detected [4].

The fundamental similarity of the immunological mechanisms of SLE and HIV infection is especially pronounced in the similarity of laboratory parameters: lympho-, thrombocytopenia, anemia, a decrease in the number of T-helpers and cytotoxicity, impaired function of leukocytes and activation of B-lymphocyte synthesis, an increase in the level of CEC, antibodies to DNA, phospholipids, formation of rheumatoid factor, etc. [5]. Difficulties in diagnosing SLE are associated with a diverse clinical picture, when new signs of the disease appear during the course of the disease, and a number of symptoms disappear. Often there are cases of an unusual onset of the disease with the absence of skin manifestations, a monosymptomatic course of the disease [6].

Undoubtedly, the similarity of the pathogenetic mechanisms and clinical signs of SLE and HIV infection, which has acquired the scale of a pandemic, is equally unfavorable in its extreme manifestations, makes the problem of their differentiation and adequate treatment extremely relevant and complex [7].

In this regard, of interest is the clinical observation of patient G, 41 years old, who was treated in the rheumatology department of the first clinic of the Tashkent Medical

Academy in October 2015. As follows from the anamnesis, the disease began in September 2014 ., when the patient noted a rise in body temperature to 38 degrees Celsius, erythematous spots on the skin of the face, nose (in the form of a "butterfly"), increased hair loss, weakness, pain in the heart area, painful sores in the oral cavity, pain in joints of the hands and feet. After insolation, erythematous spots on the face increased and appeared on the neck. Febrile fever persisted. During the illness, she lost 12 kilograms. Upon admission to the hospital, the condition was moderate, body temperature 38.0 degrees Celsius, erythematous spots on the skin of the face, nose (in the form of a "butterfly"), as well as other parts of the body, injection of the sclera. Movements in the small joints of the hands, feet, wrist and ankle joints are sharply limited due to pain. Palpated axillary lymph nodes of elastic consistency, painful to the touch, not soldered to the surrounding tissues, more than 1 cm in diameter, aphthous changes were found on the oral mucosa. Heart sounds are muffled, pulse is 100 in 1 minute, rhythmic, blood pressure is 130/80 mm Hg. In KLA, hemoglobin is 87 g/l, er. — 2.8*1012/l, lake. - 5.4 * 109 / l, leukocyte formula - p -, s - 79%, l - 18%, e - 1%, m - 2%, platelets - 264 * 109 / l, ESR 18 mm / hour . In OAM, protein was determined up to 0.066 g/l, epithelium-10-11 in the field of view; leukocytes-25-26 in the field of view; No significant changes were found in the biochemical analysis of blood. LE cells were not detected, RF was negative. Skin biopsy: morphology similar to systemic lupus erythematosus. ECG:

Sinus rhythm.Heart rate 96 in 1 min. Violation of blood circulation in the anterior septal region of the left ventricle. X-ray of the chest: signs of bronchitis Ultrasound: chronic pyelonephritis of the right kidney, some changes in the liver parenchyma.

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Thus, on the basis of the study, it was possible to identify the following signs of the disease, which are the diagnostic criteria for SLE of the American Rheumatological Association (1982): photosensitivity, non-erosive arthritis, oral ulcers, anemia, dermatitis on the face like a "butterfly".

Due to their sufficient number, and also taking into account the female sex, young age, persistent fever, significant and rapid weight loss (more than 10 kg in 5 months), alopecia, vasculitis, lymphocytopenia, the diagnosis was made: SLE, subacute course, activity 2 tbsp., dermatitis, polyarthritis, FN 2, alopecia, anemia, aphthous stomatitis, lymphadenopathy, vasculitis (coronaritis).
Taking into account the activity of SLE, treatment was prescribed: prednisolone at a dose of 20 mg/day orally, pulse therapy with methylprednisolone 1000 mg for 3 days and cyclophosphamide 1000 mg for 1 day. In addition, drugs from the group of non-steroidal anti-inflammatory drugs were prescribed - melbek 15 mg (a selective inhibitor of COX-2) was used. During therapy, some improvement in well-being was noted, manifested in a decrease in the severity of arthritis, dermatitis, and febrile fever. Additional blood tests for Plasmodium malaria gave negative results. By this time, the results of ELISA were obtained, which revealed antibodies to HIV infection. In an extended examination, a positive result obtained by ELISA was confirmed by immunoblotting. The patient was transferred to the infectious diseases hospital, and subsequently was under observation in the city AIDS center.

All this cast doubt on the diagnosis of SLE. How, in this case, to regard the symptoms of the disease, corresponding to the diagnostic criteria for SLE? As a confirmation of SLE in a patient who also suffers from AIDS, or as a lupus "mask" of HIV infection? According to the literature, SLE cannot develop in an HIV-infected patient with a low content of CD4 cells, since its pathogenesis is based on the interaction of these cells with class II histocompatibility antigens. In this regard, it becomes clear that the developed "rheumatic" manifestations of the disease should be regarded as a lupus-like syndrome of HIV infection. As rightly noted by E.N. Okhotnikova, "HIV infection is insidious and, taking on masks of various pathologies, takes revenge on us for neglecting it" [8].

CONCLUSIONS:

1. The similarity of pathogenetic mechanisms (including autoimmune) and clinical signs in HIV infection and SLE make differential diagnosis between these diseases an extremely difficult task.
2. In order to avoid diagnostic errors with severe consequences, when making a diagnosis of SLE, the doctor should be especially vigilant about HIV infection (interpret clinical signs taking into account a thorough history taking, the use of modern diagnostic tests).
3. In connection with the possibility of developing secondary immunodeficiency during SLE therapy, one should approach its appointment according to strict indications.

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