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COURSE OF HERPES VIRUS IN HIV INFECTION

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Abstract:

The main feature of herpes viruses is their lifelong persistence in the body, even if a person is infected once. The course of a chronic infectious process depends on the state of the immune system of the virus carrier [6]. Viral reactivation aggravates secondary immunodeficiency. Manifestations of herpetic infection are many-sided - from rashes on the lips and aphthous stomatitis to generalized forms with widespread damage to the skin and mucous membranes involving various organs and systems in the pathological process.

Keywords: Herpes viruses

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The group of herpes viruses includes several morphologically similar pathogens of human diseases, the main of which are: the herpes simplex virus Herpes simplex of two types (type 1 - predominantly nongenital and type 2 - predominantly genital localization of the lesion), Varicella-Zoster virus (the causative agent of chickenpox smallpox and herpes zoster), Epstein-Barr virus (EBV) and cytomegalovirus (CMV). According to the WHO classification, herpes simplex is an AIDS-defining disease [1]. CMV is one of the main causative agents of secondary (opportunistic) infections in AIDS, causing rhinitis, encephalitis, pneumonia and other diseases. In human immunodeficiency virus (HIV) infection, lymphomas of greatest interest are the second most common tumors in patients with this disease, occurring in 3–4% of cases. Approximately 12–16% of patients in the AIDS stage die from lymphomas [2]. Some of these tumors, such as Burkitt's lymphoma (African lymphoma), are etiologically related to EBV. It has been established that herpes viruses can activate the HIV genome and are a cofactor in the progression of HIV infection and AIDS [3]. In recent years, it has been shown that the activation of herpesvirus infections in HIV-infected patients, along with increased HIV replication, contributes to an increased release of its virions in places of herpetic skin rashes, which do not always have the appearance characteristic of herpes, thereby contributing to the possible infection of HIV in healthy people by contact by [2, 3]. The range of clinical manifestations of herpesvirus infections depends on the localization of the pathological process and its prevalence, the state of the patient's immune system and the type of pathogen. Clinical symptoms of infection against the background of an immunodeficiency state can be severe, with generalization and complications, which often determine the course and outcome of the underlying disease. At the same time, in patients with HIV and herpesvirus infections, the function of innate immunity is impaired to a greater extent than with monoherpes infection, which can also serve as an additional laboratory criterion for the severity of the disease [3]. Since the receptors of the 4th cluster of differentiation (Cluster of Differentiation - CD) are, to a certain extent, receptors for binding to HIV, they take an active part in the pathological process with a gradual decrease in their level in the peripheral blood. And the herpes virus probably activates HIV and promotes more rapid destruction of virus target cells, including CD4+ lymphocytes [4, 5].

The purpose of this work was to determine the spectrum of clinical manifestations of herpesvirus infections in patients with HIV infection depending on the content of CD4+ lymphocytes.

Material and methods. Under observation were 353 patients with HIV infection (220 men and 133 women) hospitalized in the 24-hour hospital of the Andijan Clinical Center for the Prevention and Control of AIDS in Andijan. The age of the patients ranged from 19 to 68 years (average 32.5±0.8 years). All patients were examined for herpesvirus infections using enzyme immunoassay in the first days after admission to the hospital. The polymerase chain reaction ("Amply Sensi" SPF "Litekh") examined blood, cerebrospinal fluid, smears of the oropharynx, urethra, cervical canal, rash area for the presence of genetic material of CMV, herpes simplex viruses, EBV, herpes virus

human type 6 and toxoplasma. The content of various groups of lymphocytes was determined by flow

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cytofluorometry (Ortum spectrum) using monoclonal antibodies against CD.

In 48 cases the disease was fatal. Depending on the content of CD4+ lymphocytes in the blood, all patients were conditionally divided into three groups. The first group consisted of 159 people with a blood CD4+ lymphocyte count of less than 200 per 1 μl. The average number of CD4+ cells in patients of group 1 was 78.55±9.21 per 1 μl (in healthy people - 998.76±98.51 per 1 μl). Group 2 included 148 people with a count of CD4+ lymphocytes from 200 to 500 per 1 μl (on average 371.22±11.97 per 1 μl). The third group consisted of 46 patients with a CD4+ lymphocyte count of more than 500 in 1 μl of blood (on average, 597.25 ± 17.63 in 1 μl). Statistical analysis was carried out using parametric statistics using Student's test.

Results of the study and discussion of the data obtained. When analyzing the frequency distribution of nosological forms of herpesvirus infections with clinical laboratory manifestation in patients with HIV and infection, it was found that most often these diseases worsened in the clinically expressed stages of HIV infection when the level of CD4+ lymphocytes was less than 200 per 1 μl (Table). At the same time, the most common were exacerbations of infection caused by the herpes simplex virus type 1 (88.5%) and herpes zoster (86.5%). According to the results of polymerase chain reaction, other viruses of this group were found in 96% of patients with herpes simplex virus in various environments. In half of the cases, a mixed infection was detected - three viruses: herpes simplex virus, EBV and herpes virus type 6. Mixed infection with two pathogens was also quite often recorded: herpes simplex virus and EBV, herpes simplex virus and herpes virus type 6 (22 and 15% cases respectively). EBV was most often detected (in 61.5% of cases, mainly in epithelial cells of the oropharynx, less often in saliva and peripheral blood lymphocytes) and herpes virus type 6 – in 58.2% of cases. CMV was detected only in 27% of cases in epithelial cells of the cervical canal, urethra, cerebrospinal fluid and peripheral blood lymphocytes. It should be noted that in 9.1% of patients, DNA of herpes simplex virus types 1 and 2 was detected in several samples; in particular, with anogenital rashes, it was detected in epithelial cells of the oropharynx and peripheral blood lymphocytes.

In patients of group 1, the most severe and varied manifestations of opportunistic infections were observed. Thus, the herpes simplex virus caused mucocutaneous lesions of the usual localization (wings of the nose, red border of the lips, skin of the chin, labia, pubic and inguinal areas, penis, buttock area), which were often recurrent, widespread and long-lasting (more than 1 months) did not heal. These processes were characterized by ulcerative-necrotic changes, when in place of herpetic vesicles, deep ulcers with jagged edges, 2 cm or more in diameter. Their involution with crust rejection, epithelization and scarring took place very slowly. In the vast majority of patients (70%), the rashes were localized on the face (herpes labialis et nasalis), in 30% of cases the common form was diagnosed. The frequency of relapses of infection in these patients reached 20 per year, i.e., practically it was of a continuously recurrent nature. Exacerbations of the disease were characterized by complaints of general malaise, headache, weakness, and fever. Clinical the deterioration was accompanied by negative immunological dynamics: a sharp decrease in the number of CD3+ and CD4+ T-lymphocytes, natural killer cells and a decrease in their functional activity.

With genital herpes, which was observed in the majority of patients in group 1 (69.7%), in every tenth case, along with the perianal area, the distal part of the rectum was involved in the process, which was accompanied by pain during defecation and constipation. In this case, fever, enlarged inguinal lymph nodes and symptoms of sacral neuropathy were observed, which was accompanied by neurasthenic (44.5%) and depressive (39.7%) disorders.

Disseminated herpes simplex virus infection was diagnosed in 7.4% of patients in group 1 and was probably associated with viremia, accompanied by fever, weakness, disseminated intravascular coagulation and organ damage (hepatitis, meningoencephalitis, pneumonia). It is known that in the general incidence of serous meningitis in people, the share of herpetic meningitis is 0.5–3%, and among HIV-infected people in the active stage of the disease – 10–12% [1, 2]. It is possible that the herpes simplex virus enters the brain through the hematogenous and/or neural route. Primary reproduction of viruses in the ganglia with subsequent spread to the brain cannot be ruled out [6]. It should be noted that a feature of the clinic of herpetic meningoencephalitis in HIV-infected people (5.7%) was the presence of a wide range of psychopathic disorders - from gross organic to functional-reactive. When diagnosing, we took into account not only the clinical manifestations of the disease, its dynamics, and the severity of the condition, but alsothe presence of magnetic tomographic signs of focal encephalitis, as well as the results of a serological study and polymerase chain reaction of bloodand cerebrospinal fluid.

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Mortality in severe generalized herpes simplex was, according to our data, 80% in patients of group 1, while generalized forms of tuberculosis infection (25%) and candidiasis with visceral lesions were recorded. In groups 2 and 3, only candidal infection of the mucous membranes without visceral manifestations was diagnosed.

All those examined, regardless of the level of CD4+ cells, were seropositive for Toxoplasma. At the same time, markers of activation of Toxoplasma infection were found in most cases in patients of the 1st group (72%) and in a third of patients of the 2nd group. In patients of group 3 it occurred least often – 11% of cases. Generalized toxoplasmosis was diagnosed in 5 patients of group 1 only, which was confirmed detection of characteristic foci of brain damage, identification of Toxoplasma gondii DNA in the cerebrospinal fluid and blood. All cases were fatal. In accordance with WHO criteria, stage V HIV infection was diagnosed in 76 patients of group 1 (48%), which was significantly more common than in patients of group 2 - 10 people (6.8%).

In group 1, patients with herpes zoster predominated (86.5%). In 43% of cases, the rashes were localized on the face along the branches of the trigeminal nerve and in 35.7% of cases - in the chest and lumbar region. Only 3% of patients in groups 2 and 3 had this disease without complications. In most cases (87.3%), it was complicated by ganglioneuritis. When herpetic eruptions were localized on the face, eye damage and facial nerve paresis were noted. Noteworthy was the fact that in all patients, HIV infection was first detected in a medical institution where treatment for herpes zoster was carried out.

It is known that co-infection with CMV and HIV leads to additional immunosuppression, dissemination of pathogens and generalization of the infectious process [1, 3]. Among patients of group 1, 81 cases of manifest CMV infection were registered. CMV DNA was detected in the blood and cerebrospinal fluid; when examining biopsies of affected organs, specific giant cells were visualized. The clinic observed fever, granulocytopenia, thrombocytopenic purpura, maculopapular rash, interstitial pneumonia, encephalitis, and ulcerative lesions of the gastrointestinal tract. In the remaining patients of group 1 (49%) and in almost all those examined in groups 2 and 3, markers of latent CMV infection were determined in an enzyme-linked immunosorbent assay.

Of particular interest were patients receiving highly active antiretroviral therapy. In these cases, reactivation of herpesvirus infection occurred at a later period (later than 4 weeks). Of the total number of patients receiving this treatment, this occurred in 8.8% of cases, which corresponds to literature data [2, 3]. Probably, there was a so-called paradoxical reaction to antiretroviral therapy, when at the beginning of treatment an exacerbation of opportunistic infections may occur.

Thus, changes in the immune system increased as HIV infection progressed due to the addition or activation of opportunistic diseases. When the level of CD4+ lymphocytes in the blood of patients decreased to less than 200 per 1 μl, severe forms of herpesvirus infections prevailed, and in 25% of cases the pathological process became generalized. In more than half of patients with HIV infection with a low level of CD4+ lymphocytes, a combination of herpes simplex with other opportunistic infections (tuberculosis, toxoplasmosis, etc.) was observed, which contributed to an increase in the incidence of deaths.

LITERATURE

1. Butylsky A.N., Kuznik B.I., Rosenberg V.Ya. Dynamics of immunity parameters in patients at various stages of HIV infection // Med. immunol. 2005. T. 7, no. 2–3. pp. 153–154.
2. Isakov V.A., Arkhipova E.I., Isakov D.V. Human herpesvirus infections / ed. V.A. Isakov. St. Petersburg: SpetsLit, 2006. 300 p.
3. Kalinina N.M., Ketlinsky S.A. Immunology of HIV infection // Immunodeficiency states / under. ed. V.S. Smirnova, I.S. Freidlin. St. Petersburg: Foliant, 2000. pp. 411–445.
4. Clinical recommendations. HIV infection and AIDS / ed. V.V. Pokrovsky. M.: GEOTAR-Media, 2006. 128 p.
5. Onishchenko G.G. Priorities for countering the HIV/AIDS epidemic in the Russian Federation at the present stage // STIs. 2004. No. 3. P. 3–5.