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CLINICAL AND PATHOGENETIC FEATURES OF DAMAGE TO THE NERVOUS SYSTEM AT THE INITIAL CLINICAL STAGES OF HIV-INFECTION

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The aim of the study – investigating the features of clinical symptoms in HIV-positive people in the early stages of the disease and their pathogenetic justification.

The study used theoretical methods based on the analysis and synthesis of studying the information of the modern world from the PubMed and Scopus databases, clinical observations, as well as deductive-inductive methods.

Conclusions. *As a result of the war in Ukraine, the epidemiological control of infectious diseases on the territory of the country was violated. HIV-infection and the absence or atypicality of its clinical symptoms in infected patients deserve special attention, which is based on a detailed understanding of the pathological processes of neuroAIDS. Damage to astrocyte cells disrupts the connection of cells of the structural and functional complex of the brain with other cells and forms the fundamental basis for understanding clinical symptoms. Most often in the form of: neuropsychic, cognitive somatic and neurological disorders. The pathogenic effect of the virus contributes to spasm of cerebral microvessels, is the cause of local hypoxic lesions, microstrokes, and in late cases – lacunar cerebral infarcts. The active involvement of brain cells in the pathogenesis of HIV-infection has been confirmed by many studies. Along with standard diagnostic methods, it is advisable to use psychoneurological and cognitive tests and scales.*

A number of shortcomings have been identified that can reduce the reliability of diagnostic studies when establishing a diagnosis.

Understanding the pathogenetic picture will allow choosing «indicators» for the general diagnostic scheme and increasing the probability of establishing the correct diagnosis, adjusting ART and predicting the further development of the disease.

Key words: *clinic; pathogenesis; asymptomatic course; neuroAIDS; HIV; tests; scales.*

HIV-infection has always occupied one of the key places among socially dangerous diseases in European countries.

Due to the war in Ukraine, cases of sexual and physical violence have increased, the number of surgical interventions has increased, and the systems of usual epidemiological control of diseases have been violated. Russia's military aggression led to the migration of millions of Ukrainians to different European countries. All this in the near future can multiply the incidence of socially significant infectious diseases [1–3].

An important aspect of diagnosis is that HIV-infection in patients is often diagnosed at the II, III and IV clinical stages of the disease. For example, clinical stage II is characterized by hyperreactivity of the humoral link of immunity, which is clinically accompanied by generalized lymphadenopathy in patients [4]. Specific symptoms or HIV-indicating diseases already in the later stages will force the general practitioner to check and establish the diagnosis. Today, the study of the clinical stage I of HIV-infection attracts great interest. At this stage, the symptoms are nonspecific, subtle and difficult to differential diagnosis without laboratory methods. In the asymptomatic course, clinicopathological disorders can only be suspected using specific cognitive testing, scales of depression and anxiety, determine using instrumental (MRI) and laboratory (the amount of virus RNA in the cerebrospinal fluid) research methods which are occasionally used by general practitioners [4–7]. Understanding the features of pathological processes and the clinical symptoms caused by them will help improve the effectiveness of early diagnosis of HIV-infection.

The aim of the study – investigating the features of clinical symptoms in HIV-positive people in the early stages of the disease and their pathogenetic justification.

The study used theoretical methods based on the analysis and synthesis of studying the information of the modern world from the PubMed and Scopus databases, clinical observations, as well as deductive-inductive methods.

HIV-infection is an immunosuppressive, retroviral disease with clinical symptoms progressing in stages and a wide variety of both primary and secondary complications

of various origins. To identify possible cases of both latent and atypical clinical symptoms, it is necessary to know the important pathogenetic aspects of HIV-infection and the involvement of cell varieties that have CD4 receptors in the pathological process [8]. CD4 receptors are present in a large population of resident macrophages, astrocytic cells, endotheliocytes of small vessels of the meninges, and ependyma of the cerebral ventricles [9–11]. «CD4-marked» cells should indicate a varied and specific clinical symptomatology.

In the first hours of active replication, the retrovirus in the body crosses the blood-brain barrier (BBB), infecting brain cells, closely linking the pathogenesis of neuroAIDS with the state of the BBB and the immune system of the central nervous system (CNS) [5].

First of all, HIV affects neuroglial cells, which are characterized by rapid mitotic division. Thus, the sites of virus localization quickly spread in the brain tissue, increasing the likelihood of inflammation, disruption of the BBB, and brain homeostasis [12]. It is important to understand that neuroglia are divided into several subtypes, the most significant of which are astroglia, microglia, and ependyma [10].

Astroglia is formed by different types of astrocytes. They constitute the main part of the neurovascular unit, which ensures the functioning of the BBB, control of the physiological activity of neurons and other important cells. With their processes, astrocytes form synaptic clefts «contacts» on the surface of other types of astrocytes, pia mater cells, cerebral endotheliocytes, and neurons, connecting all CNS cells with each other and maintaining a balance between them [12].

In the human cerebral cortex, one astrocyte cell is able to regulate about 2 million cell synapses of various subtypes. With the help of gap junctions between neighboring astrocytes, cells are able to quickly exchange information and regulate other neuronal regions in topographically distant parts of the CNS, accurately and quickly eliminating the imbalance of brain tissue homeostasis [5, 9]. Due to the disruption of the cellular structures of the brain and the triggering of the inflammatory pathways TNF and LF-1 by the virus, neurotoxic cathepsin is released from lysosomes. This leads to a disruption in the synthesis of the main neuronal proteins, which are primarily an essential condition for the normal functioning of astrocytic glial cells [12, 13]. Thus, the retrovirus causes a disruption in the communication of cells of the structural-functional complex of the central nervous system, changes the normal functioning of neurotransmitters and the acid-base environment, to which neuronal cells are very sensitive.

Violation of the function or physiological structure of the sites of the synaptic complex leads to excessive or insufficient production of neurotransmitters. As a result, there is a local impairment of the functional activity of neuronal regions, the environment of which is controlled by astrocytes [14]. This explains the violation of cognitive and somatic functions: memory, intellectual abilities, learning, attention, sleep disturbance. Mental disorders, emotional and behavioral disorders, namely: mood deterioration, narrowing of the circle of interests, various phobias, autonomic lability, depression and apathy, irritability, and often mental disorders. The presence and severity of these disorders depends on the totality of all pathological factors and the characteristics of the body as a whole [14–16].

According to the classification of the All-Ukrainian Association of Neurology and Reflexology (UANR), 40 % of cognitive impairments occur at the subclinical stage of disorders. When clinical symptoms are completely absent, the disorders are minor and hardly noticeable, capable of affecting the performance of complex professional activities. All other symptoms are divided between moderate and severe and appear in the later stages of HIV-infection with a large number of AIDS-defining diseases [5, 17].

Microglial cells are found on the entire surface of the CNS. Microgliocytes are functionally similar to macrophages and are called microglial (resident) macrophages. After infiltration by the virus, such cells are the main source of the retrovirus in the nervous system and one of the important factors in the damage to human neuronal structures. The infiltration of viral material into the cell accelerates its apoptotic death with dysfunction of the latter and the release, for example, of such neurotoxic products of the virus activity as the p-17 protein, the Vpr regulatory protein, and cytochrome C [7]. All this provokes a stressful situation for the tissues of the central nervous system, increasing its active immune response, causing autoimmune inflammation. The number of microgliocytes near the site of damage increases due to pre-existing immune cells. Local inflammation in the brain increases the permeability of microvessels, which allows macrophages to migrate into the nervous system from extracerebral sites, synthesizing signal substances – cytokines. This is what can close the pathological circle of a progressive inflammatory process [18, 19].

Thanks to the above data, meningism and aseptic meningitis are explained. Early and frequent manifestations of which are headaches, not expressed meningeal symptoms and moderate intoxication syndrome. Quite often, such cases are confused with migraines, common headaches of unknown origin, chronic fatigue syndrome, or the common cold. In such cases, the epidemiological history is not collected deeply enough, differential diagnosis is not fully carried out, neglecting the expediency of cognitive or psychosomatic testing. The latter could point to the need

for instrumental imaging of CNS tissues.

In the absence of a focal neurological picture, these nonspecific symptoms in HIV-infected individuals indicate the presence of multiple or small-focal pathological inflammatory processes of the brain of viral and autoimmune origin [16, 20, 21]. Most cases of aseptic meningitis occur in an atypical erased form, which leaves the course of a retroviral infection unnoticed. The only manifestations of which are headaches and pleocytosis in the CSF [16, 22].

Today, it is not well understood whether the brain ependyma is directly involved in the pathogenesis of HIV-infection. But given its role in the formation of the BBB (close contact with other glial cells), its localization on the pia mater, the functions of control and regulation of cerebrospinal fluid, we can assume that ependymocytes and thymocytes are secondary involved in the complex processes of neuroAIDS [23]. Probably, a thorough study of the pathological processes of these cells will make it possible to characterize in detail the focal cerebral symptoms and predict the severity of the course of neurological complications in the early stages of the disease.

Data on the active involvement of neuroglia in the pathogenesis of HIV-infection, the formation of latent reservoirs, and the presence of latent symptoms, which are confirmed by many studies [5, 6, 12, 24], are presented. It was noted that in many HIV-positive patients there was no association between cognitive impairment and the clinical stage of the disease. That is, neuropsychological and cognitive disorders can be observed not only in the later stages of the disease, as a reaction to the rapid activity of opportunistic infections. Cognitive and neuropsychological symptoms often appear in patients at clinical stages I and II of HIV-infection. Interestingly, these patients have already achieved sustained serum aviremia and asymptomatic disease on ART. Also, in these individuals, possible external factors of aggression were excluded and the direct effect of the virus was proved by studying the proportional increase in clinical symptoms, the level of viral load in the cerebrospinal fluid, and pathological changes in the CNS tissues on MRI [5].

Confirmation of the analysis of our data was found in the description of the case of the phenomenon of discordance (escape) of HIV-infection [6]. In the studied patients with long-term and complete optimally diagnostic suppression of the retrovirus in the blood (<50 RNA copies), there suddenly appeared increasing sensory, motor and cognitive symptoms with the dominance of the latter. Namely, the examined patients were dominated by manifestations of cognitive impairment of varying severity, increased emotional excitability, depression and anxiety, persistent headaches, cerebellar dysarthria and cerebellar ataxia. Manic symptoms, tactile allodynia, and temporo-spatial disorientation were less common. No abnormalities were found in clinical blood tests. But in the CSF, resistant, mutated viral subpopulations were found. Instrumental diagnostics revealed white matter hyperintensity on T2-weighted FLAIR (Fluid attenuation inversion recovery) sequences [5, 6].

Consideration of the blood microcirculation system in the CNS deserves special attention.

Since it is very closely related to the state of the BBB and the pathogenesis of neuroAIDS in general. The presence of CD4 receptors on the surface of the endotheliocytes of the choroid plexuses of the meninges and ventricles in an infected body leads to damage caused by the virus and its waste products. There is a destruction of the cell wall of microcapillaries, an increase in the permeability of the BBB, and the involvement of brain tissues in inflammatory processes of an autoimmune and viral nature. These changes, together with a number of neurotoxic factors of the virus, lead to virus-induced encephalitis. This contributes to the disruption of the CSF circulation in the ventricles of the brain, the involvement of ependymal cells in the pathogenesis. Consequently, the pressure increases and the stable environment in the ventricles and spinal space is disturbed [6, 7, 25, 26]. Thus, clinically, we observe in patients a sudden headache, mild meningeal symptoms. Significant fluctuations in intracranial pressure may worsen the psychosomatic and cognitive symptoms described above. But already against the background of a violation of the functional structure of the vascular wall, in conditions of aseptic chronic inflammation and constant fluctuations in intracranial pressure, thrombocytopenia, which often develops with HIV-infection, closes the pathogenetic link [15, 16, 22].

The pathogenic effect of the virus can contribute to the obstruction of small vessels with subsequent degeneration of endotheliocytes – their replacement with lipids and collagen (lipogyalinosis) – which is the cause of local hypoxic lesions of brain areas, small microstrokes and hemorrhages, and in late cases – lacunar cerebral infarctions [4]. This picture will be characterized by a typical clinic of cerebral ischemia and necrosis, depending on the location of the pathology. Considering the above changes, the pathogenesis is based on a progressive regional microvascular spasm, which at the initial stages can provoke dyscirculatory encephalopathy. Which, first of all, will be characterized by vestibular and autonomic disorders (dizziness, nausea, fluctuations in blood pressure) and gradual depression of the cognitive sphere with psychoneurological accompaniment. In the later stages, with the addition of opportunistic infections, an increase in the likelihood of extensive strokes and subarachnoid hemorrhages [6, 27].

The Generalized Anxiety Disorder Scale (GAD-7) is often used to identify anxiety disorders and depressive conditions that may be a consequence of the pathological picture described above. To obtain in-depth information about the mental and cognitive state of health, test questionnaires are used: The hospital anxiety and depression scale (HADS), Mini-Mental State Examination (MMSE) and the Hamilton scale [28, 29].

By using this diagnostic combination of tests as an adjunct to the initial examination of patients, the clinician can gain sufficient evidence to conduct a more detailed diagnosis. For example, an examination of the cerebrospinal fluid, an MRI of the brain, or a referral to a consultation with other specialists. However, we see shortcomings in such a diagnostic scheme for HIV-positive patients, especially in the early stages of the disease. It is not always possible to accurately take into account all external influencing factors on test results. For example, chronic diseases not related to HIV, severe emotional stress associated with the war in Ukraine, or bad habits that patients may hide can significantly reduce the reliability of test results and, as a result, the diagnosis of HIV-infection.

Thus, the pathogenetic picture associated with the clinical symptoms of HIV-positive patients, described above, can serve as a foundation for the search for «indicators» with which we can increase the reliability of psychological and cognitive testing schemes. For example, by using data from a patient's epidemiological history along with hematological indices and blood counts, we are likely to not only increase the likelihood of an HIV diagnosis, but also determine the type of inflammation and its intensity. And adding to this scheme VL and quantitative indicators of T-lymphocytes taken from HIV-positive patients, it will be possible to speak about the quality of antiretroviral therapy, the patient's propensity for treatment and predict the further development of retroviral infection.

Conclusions

1. HIV-infection continues to spread in Europe and occupies a key place among socially dangerous diseases. Due to the war in Ukraine, control over infectious diseases has been disrupted. The number of people at risk of HIV-infection has increased by 35 % compared to 2021. The complexity of the differential diagnosis of retroviral infection and the features of the asymptomatic course require a detailed study of the early signs of HIV-infection.

2. Infection and damage to astrocytic cells disrupts the connection of cells of the structural-functional complex of the CNS with other brain cells. Local inflammation in the CNS, caused by already existing cells of the immune system, increases the permeability of microvessels, which allows macrophages to migrate into the nervous system from outside the brain areas, synthesizing cytokines that provoke the phenomena of meningism, aseptic meningitis and virus-induced encephalitis with its characteristic symptoms. The pathogenic effect of the virus contributes to the obstruction of small vessels and is the cause of local hypoxic lesions of brain areas, small microstrokes and hemorrhages, and in late cases, lacunar cerebral infarcts.

3. The active involvement of neuroglia in the pathogenesis of HIV-infection has been confirmed by many studies. Where in the surveyed HIV-positive patients there is no relationship between cognitive impairment and the clinical stage of the disease. The number of detected HIV RNA copies in blood plasma has a significant reciprocal correlation with HIV RNA in the brain, which confirms the destructive effect of the virus on the BBB, the slow development of neurological, mental and somatic symptoms. The use of the scales of generalized anxiety disorder (GAD-7) in combination with The hospital anxiety and depression scale (HADS), Mini-Mental State Examination (MMSE) and the Hamilton scale has a number of disadvantages that can significantly reduce the reliability in diagnosing infectious diseases.

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КЛІНІКО-ПАТОГЕНЕТИЧНІ ОСОБЛИВОСТІ УРАЖЕННЯ НЕРВОВОЇ СИСТЕМИ НА ПОЧАТКОВИХ КЛІНІЧНИХ СТАДІЯХ ВІЛ-ІНФЕКЦІЇ

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РЕЗЮМЕ. Мета – вивчення особливостей клінічної симптоматики у ВІЛ-позитивних осіб на ранніх стадіях захворювання та їх патогенетичне обґрунтування.

У дослідженні використовували теоретичні методи, побудовані на аналізі та синтезі дослідження сучасної всесвітньої інформації баз даних PubMed та Scopus, клінічних спостережень, дедуктивно-індуктивних методів.

***Висновки.** Через розв'язану війну росії проти України був порушений епідеміологічний контроль за інфекційною захворюваністю на території держави. Особливої уваги заслуговує ВІЛ-інфекція та особливості її клінічної симптоматики. У інфікованих пацієнтів вона ґрунтується на детальному розумінні патологічних процесів нейроСНІДу.*

Пошкодження астроцитів порушує зв'язок клітин структурно-функціонального комплексу мозку з іншими клітинами і є фундаментальною основою розуміння клінічної симптоматики найчастіше у вигляді нейропсихічних, когнітивних соматичних та неврологічних розладів.

Патогенетична дія вірусу сприяє обструкції мікросудин мозку та є причиною локальних гіпоксичних уражень, мікроінсультів, а у пізніх випадках – лакунарних інфарктів мозку. Активне залучення клітин мозку у патогенез ВІЛ-інфекції підтверджено багатьма дослідженнями.

Разом із стандартними методами діагностики доцільно використовувати комплекси психоневрологічних і когнітивних тестувань та шкал.

Виявлено ряд недоліків, які здатні знизити достовірність діагностичних тестів під час встановлення діагнозу. Розуміння патогенетичної картини дозволить обрати «індикатори» для загальної схеми діагностики та збільшення вірогідності правильного діагнозу, відкоригувати антиретровірусну терапію та спрогнозувати подальший розвиток хвороби.

***Ключові слова:** клініка, патогенез, безсимптомне носійство, нейроСНІД, ВІЛ, тести, шкали.*

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