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Demyelinating disease can hide HIV-infection Case report

A significant part of patients with HIV / AIDS develops damage to the nervous system. There are also cases where opportunistic infections of the nervous system, especially herpes viral origin, can hide the underlying disease, making it difficult diagnosis.

Here, we report a case of a 41-years-old female presented to The Center of Infectious disorders of the Nervous System (Kyiv, Ukraine) in August, 2018 after developing acute fever following by a left side hemiparesis, violation of coordination. Previously, a demyelinating disease was suspected. Tuberculosis and HIV denied. Her physical examination showed tremor in her hands during a Barre-probe. She performed the coordination tests with intent, staggering in the Romberg pose. A small brain lesion was revealed at MRI. Antibodies to HSV1/2, CMV, Tox. gondii were found in the CSF and blood. Blood PCR was reported to be positive for DNA EBV, and RNA HCV. A rapid HIV test was negative. A repeated blood test performed 10 days after admission showed low level of $CD4^+$ T cells (36 cells/1 μl), and HIV RNA (850.104 cp/ml). HIV antibodies were also revealed. As a result, patient was transferred to a specialized department for further treatment.

Conclusions. Considering high clinical polymorphism of HIV/AIDS, physicians of all specialties should be alert for the possible neurologic manifestations of this disease to timely examine patients.

Key words

HIV-infection, demyelinating disease, herpesviruses.

The problem of HIV infection for more than 25 years remains relevant for the world community; the scale of the HIV spreading has become global in nature and poses a real threat to the socio-economic development of most countries. At present, the epidemic situation with HIV remains tense in Ukraine: HIV continues to disseminate among the population, and the cumulative number of HIV-infected people and AIDS patients is increasing. According to official data, at the beginning of 2018, there were 244.000 HIV-infected persons of all age groups in Ukraine [1]. In 2016, 14.334 new cases of HIV infection were detected, with 57.7 % of people diagnosed with clinical conditions at later stages (CD4 < 350 cells/mm^3), of which 30 % had an immunosuppression below 200 cells/mm^3 [1]. This testifies to the untimely diagnosis of HIV status, and as a consequence, the inadequate quality of medical care. The reason for this is the high clinical polymorphism of HIV/AIDS, the similarity with various diseases (masks), which often leads to erroneous diagnoses in such patients. Diagnostic and therapeutic errors contribute to the lack of orientation of doctors, whose specializing is outside the plane of infectious diseases and HIV-medicine, in the manifestations of this disease. Sometimes patients deliberately do not inform the medical staff about their HIV status or are themselves in the dark about it (especially those who are not in the categories of high risk of infection). In addition, existing regulations on compulsory HIV testing for certain clinical and epidemiological grounds in some cases inhibit or make it difficult to establish the correct diagnosis.

Significant parts of patients with HIV/AIDS develop damage to the nervous system, which often

leads to a lethal outcome. There are also cases where opportunistic infections of the nervous system, most often of herpes viral origin, can hide the underlying disease, making it difficult diagnosis [2, 3]. Below we present a case when HIV/AIDS was concealed under the mask of demyelinating disease.

Case presentation

A 41-years-old female presented to the Center of infectious disorders of the nervous system (Kyiv, Ukraine) in August, 2018 with complaints of numbness in the left half of the body, left-side hemiparesis, impaired pelvic organ function by type of non-sustainability, violation of coordination. The patient was in good health until a month before admission to our clinic. Ill had acute onset when the left palm numbness was occurred, and quickly progressed. A left arm paresis appeared after a week, then the left leg was joined to the process. She turned to the neurologist of a local hospital, and was directed to the brain MRI. During the examination in the projection of the right visual hillock, a small irregular-shaped lesion, with unexpressed boundaries, was revealed. Intravenous contrast was accumulated along the periphery like a ring (Figure). Screening for antibodies to HIV at the same time was negative. Debut of multiple sclerosis or toxoplasmosis of the central nervous system were supposed, and the patient was transferred to our clinic.

On admission, general condition was of moderate severity. Axillary temperature was normal. The patient was normotensive with pulse beats of 70 per minute. Her physical examination showed slow mentation along with generalized slowing of her responses to verbal commands and also generalized weakness. She was emotionally labile, good contact, well oriented, and answered the questions adequately. There was left-side partial loss of sensitivity. The abdominal reflexes were asymmetrical, sharply reduced. There was tremor in her hands during a Barre-probe. Meningeal symptoms were not detected. She performed the coordination tests with intent, staggering in the Romberg pose. Episodes of uncontrollable urination were recorded. According to the patient, she has one sexual partner during last year, and four years ago she was tested for HIV with negative result. In 2017 she underwent massive dental intervention. In the same year she was treated in the dermatology department for pemphigus.

A lumbar puncture was performed just on admission and CSF analysis revealed cytosis of 8 cell/μl (lymphocytes). The protein and glucose levels were 0.41 and 3.2 mmol/L respectively (the synchronous serum values were 6.1 and 4.9 mmol/L). G class antibodies to HSV1/2, CMV, Tox. gondii were revealed.

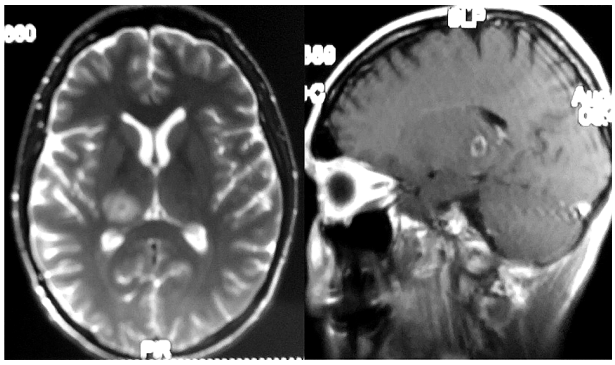

Figure. **Focus of the brain demyelination on MR imaging a week before admission**

General blood test showed leukopenia ($2.4 \cdot 10^9$/l) with relative lymphocytosis (77%) and very low proportion of $CD4^+$ (11%). M class antibodies to VZV, and CMV; total antibodies in high titer to HCV, and high titer IgG Tox. gondii were also detected. Blood PCR was reported to be positive for EBV DNA (2.4 lg), and HCV RNA ($2.6 \cdot 10^4$ IU/ml).

In view of the above, prescribed therapy included antitoxoplasmic, antiviral, neuroprotective and hepatoprotective medicines. At the same time, a rapid HIV test was conducted with the permission of the patient (the result was negative). The treatment has proven to be partially successful at least clinically: the patient's condition improved, volume of movements in the limbs increased, areas of sensitivity appeared on the palms, decreased vestibulo-atactic and cerebrostenic manifestations, uncontrolled urination stopped. A repeated blood test performed 10 days after admission showed very low level of $CD4^+$ T cells (36 cells/1 μl). RT-PCR blood test revealed HIV-1 RNA (850.104 cp/ml). As a result of the conversation with the patient, her informed agreement was reached on the re-examination of HIV antibodies. Test was carried out and was positive.

The patient with final diagnosis encephalitis with liquor-hypertensive, vestibulo-atactic, cerebrostenic syndromes, left-side hemiparesis, and hemianaestesia underlined with mixed infection (VZV, EBV, CMV, Tox. gondii in the activation phase), severe course; HIV infection, clinical stage IV; chronic Hepatitis C, was transferred to a specialized department for further treatment.

Discussion

The presented case reflects not only the diversity of the HIV infection itself, but also, to a certain extent, the insufficient awareness of outpatient physicians and hospitals about the manifestations of the disease, including its late stages. In our observations, although the patient had one of the classic AIDS lab-indicator (deep depletion of $CD4^+$ lym-

phocytes), which is the basis for an appropriate examination [4]. However, since antibodies to HIV were not firstly identified as a result of a weak immune response to HIV antigens in conditions of severe immunodeficiency, the assumption of HIV/AIDS in the outpatient stage did not occur. The clinical protocol of antiretroviral therapy of HIV infection in adults and adolescents, approved by order of the Ministry of Health of Ukraine, N 551 dated 07/12/2010, which regulates the basic principles of diagnosis and treatment of HIV-infected persons, recommends that the diagnosis of HIV infection should be based on the presence of antibodies to the virus. Since the «gold standard» for the diagnosis of HIV infection is currently PCR, we believe that this protocol needs to be modified accordingly.

Conclusions

The given clinical example testifies to the possibility of masking clinical manifestations of HIV/AIDS under the signs of demyelinating diseases of the central nervous system. A one-time negative test for HIV antibodies does not exclude the presence of the virus, especially with underlying leukopenia and T cells depletion.

The author declare no conflict of interest.

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Демієлінізуюче захворювання, яке може приховувати ВІЛ-інфекцію Клінічний випадок

У значної частини пацієнтів з ВІЛ/СНІДом пошкоджується нервова система. Спостерігаються також випадки, коли опортуністичні інфекції нервової системи, особливо герпесвірусного походження, можуть приховувати основне захворювання, що ускладнює діагностику.

Ми повідомляємо про випадок захворювання 41-річної жінки, яка поступила до Центру інфекційних уражень нервової системи (Київ, Україна) в серпні 2018 р. після розвитку гострої лихоманки, що супроводжувалася лівостороннім геміпарезом, порушенням координації, з підозрою на демієлінізуюче захворювання. Туберкульоз і ВІЛ не виявлено. Фізикальне обстеження показало тремтіння в руках під час проби Барре. Координаторні тести виконувала з інтенцією, хиткість у позі Ромберга. При МРТ виявлено невелике вогнище. Антитіла до HSV1/2, CMV, Tox. gondii були виявлені в ліквор і крові. При дослідженні крові були виявлені ДНК EBV і РНК HCV. Експрес-тест на ВІЛ виявився негативним. Повторний аналіз крові, проведений через 10 днів, показав низький рівень $CD4^+$ Т-клітин (36 клітин/1 мкл) і РНК ВІЛ (850 104 коп/мл). Також були виявлені антитіла до ВІЛ. В результаті пацієнтка була переведена в спеціалізоване відділення для подальшого лікування.

Висновки. З огляду на високий клінічний поліморфізм ВІЛ/СНІДу лікарі всіх спеціальностей повинні бути уважні до можливих неврологічних виявів цього захворювання для своєчасної діагностики та лікування пацієнтів.

Ключові слова: ВІЛ-інфекція, демієлінізуюче захворювання, герпесвіруси.

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Демиелинизирующее заболевание, которое может скрывать ВИЧ-инфекцию. Клинический случай

У значительной части пациентов с ВИЧ/СПИДом повреждается нервная система. Наблюдаются также случаи, когда оппортунистические инфекции нервной системы, особенно герпесвирусного происхождения, могут скрыть основное заболевание, что затрудняет диагностику.

Мы сообщаем о случае заболевания 41-летней женщины, поступившей в Центр инфекционных поражений нервной системы (Киев, Украина) в августе 2018 г. после развития острой лихорадки, сопровождавшейся левосторонним гемипарезом, нарушением координации, с подозрением на демиелинизирующее заболевание. Туберкулез и ВИЧ не обнаружены. Физикальное обследование показало дрожь в руках во время пробы Барре. Координаторные тесты выполняла с интенцией, шаткостью в позе Ромберга. При МРТ обнаружен небольшой очаг. Антитела к HSV1/2, CMV, Tox. gondii были обнаружены в ликворе и крови. При исследовании крови были выявлены ДНК EBV и РНК HCV. Экспресс-тест на ВИЧ оказался отрицательным. Повторный анализ крови, проведенный через 10 дней после поступления, показал низкий уровень $CD4^+$ Т-клеток (36 клеток/1 мкл) и РНК ВИЧ (850 104 коп/мл). Также были выявлены антитела к ВИЧ. В результате пациентка была переведена в специализированное отделение для дальнейшего лечения.

Выводы. Учитывая высокий клинический полиморфизм ВИЧ/СПИДа, врачи всех специальностей должны быть внимательны к возможным неврологическим проявлениям этого заболевания для своевременной диагностики и лечения пациентов.

Ключевые слова: ВИЧ-инфекция, демиелинизирующее заболевание, герпесвирусы.

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