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UDC [616-097-022:578.828.6]-06:616.523 HIV AND HERPETIC INFECTIONS: CLINICAL AND IMMUNOLOGICAL LABORATORY

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✓ Resume

Human Immunodeficiency Virus (HIV) infection is associated with progressive immune system deterioration, resulting in increased susceptibility to opportunistic infections, including herpesviruses. This article reviews the clinical manifestations and immunological-laboratory profiles of herpetic infections in HIV-infected individuals, with particular attention to the mutual influence of these pathogens on the immune system.

Herpetic infections, particularly those caused by Herpes Simplex Virus type 1 and 2 (HSV-1, HSV-2), are common among individuals with Human Immunodeficiency Virus (HIV). Co-infection can significantly impact disease progression, clinical management, and patient outcomes. This article explores the pathophysiological interactions between HIV and herpetic viruses, highlights clinical implications, and discusses therapeutic approaches to managing co-infected patients

Keywords: HIV, herpes simplex virus, varicella-zoster virus, immunodeficiency, CD4+, cytokines, opportunistic infections

ВИЧ И ГЕРПЕТИЧЕСКИЕ ИНФЕКЦИИ: КЛИНИКО-ИММУНОЛОГИЧЕСКАЯ ЛАБОРАТОРИЯ

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✓ Резюме

Заражение вирусом иммунодефицита человека (ВИЧ) связано с прогрессирующим ухудшением состояния иммунной системы, что приводит к повышению восприимчивости к оппортунистическим инфекциям, включая герпесвирусы. В этой статье рассматриваются клинические проявления и иммунологические и лабораторные характеристики герпетических инфекций у ВИЧ-инфицированных лиц, при этом особое внимание уделяется взаимному влиянию этих патогенов на иммунную систему.

Герпетические инфекции, особенно вызванные вирусом простого герпеса 1-го и 2-го типов (ВПГ-1, HSV-2), распространены среди лиц, инфицированных вирусом иммунодефицита человека (ВИЧ). Сопутствующая инфекция может существенно повлиять на прогрессирование заболевания, клиническое ведение и результаты лечения пациентов. В этой статье исследуются патофизиологические взаимодействия между ВИЧ и герпетическими вирусами, освещаются клинические последствия и обсуждаются терапевтические подходы к ведению пациентов с сопутствующей инфекцией.

Ключевые слова: ВИЧ, вирус простого герпеса, вирус ветряной оспы, иммунодефицит, СD4+, цитокины, оппортунистические инфекции

OIV VA GERPETIK INFEKTSIYALAR: KLINIK VA IMMUNOLOGIK LABORATORIYA

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✓ Rezyume

Inson immunitet tanqisligi virusi (OIV) infektsiyasi immunitet tizimining progressiv yomonlashishi bilan bog'liq bo'lib, natijada opportunistik infektsiyalarga, shu jumladan gerpesviruslarga sezuvchanlik oshadi. Ushbu maqolada OIV bilan kasallangan odamlarda gerpetik infektsiyalarning klinik ko'rinishlari va immunologik-laboratoriya profillari ko'rib chiqiladi, bu patogenlarning immunitet tizimiga o'zaro ta'siriga alohida e'tibor beriladi.

Herpetik infektsiyalari, ayniqsa Herpes Simplex Virus turi oqibatida 1 va 2 (HSV-1, HSV-2), inson immunitet tanqisligi virusi (OIV) bilan shaxslar orasida keng tarqalgan. Birgalikda infektsiya kasallikning rivojlanishiga, klinik boshqaruvga va bemorning natijalariga sezilarli ta'sir ko'rsatishi mumkin. Ushbu maqola OIV va gerpetik viruslar o'rtasidagi patofiziologik o'zaro ta'sirlarni o'rganadi, klinik ta'sirlarni ta'kidlaydi va birgalikda yuqtirilgan bemorlarni boshqarishda terapevtik yondashuvlarni muhokama qiladi.

Kalit so'zlar: OIV, herpes simplex virusi, varicella-zoster virusi, immunitet tanqisligi, CD4+, sitokinlar, opportunistik infektsiyalar

Relevance

H IV infection causes a profound impairment of the immune system, particularly affecting CD4+ T-lymphocytes, which play a critical role in antiviral defense [1]. Herpesviruses, such as Herpes Simplex Virus (HSV-1, HSV-2), Varicella - Zoster Virus (VZV), Cytomegalovirus (CMV), and Epstein - Barr virus (EBV) [2], cause one of the most common co-infections in HIV-infected patients. These infections may become chronic, recurrent, and more severe in immunocompromised individuals. [3].

Herpetic infections not only occur more frequently in people with HIV, but they also tend to be more severe, recurrent, and resistant to treatment. Furthermore, HSV-2 has been identified as a key co-factor in both the acquisition and transmission of HIV, as the mucosal lesions caused by herpes facilitate viral entry and replication. The presence of HSV also triggers immune activation and inflammation, which can enhance HIV replication and contribute to faster disease progression [4-6].

Understanding the interplay between HIV and HSV is critical for improving the management of coinfected patients [7-9]. This article explores the epidemiology, immunopathogenesis, clinical manifestations, and therapeutic strategies related to HIV and herpetic co-infection, with a focus on how each virus influences the course of the other [10].

The aim of this study is to investigate the clinical and immunological relationship between Human Immunodeficiency Virus (HIV) and Herpes Simplex Virus (HSV) infections, particularly focusing on the impact of HSV-2 in HIV-positive individuals. The study seeks to explore how herpetic co-infections influence HIV progression, increase susceptibility to HIV transmission, and complicate clinical management. Additionally, this research aims to evaluate current treatment strategies and highlight the importance of integrated therapeutic approaches for patients with HIV-HSV co-infection.

Materials and methods

Hospital between 2022 and 2024. 120 patients diagnosed with Human Immunodeficiency Virus (HIV) infection and various forms of herpetic infections were included in the study. The inclusion criteria were age between 18 and 65 years, confirmed HIV infection, and clinically and/or laboratory-confirmed herpesvirus co-infection (including HSV-1, HSV-2, VZV, or CMV).

Patients were divided into groups based on the type and severity of herpetic infection and the degree of immunosuppression determined by CD4+ T-cell count. All participants underwent a comprehensive clinical evaluation, including physical examination, symptom scoring, and assessment of the frequency and severity of herpesvirus manifestations.

Laboratory investigations included:

- Complete blood count (CBC)
- CD4+ and CD8+ T-lymphocyte counts, measured by flow cytometry
- CD4/CD8 ratio determination
- Cytokine profiling: serum levels of IL-2, IL-4, IL-6, IL-10, IFN- γ , and TNF- α were measured using ELISA kits
- Quantitative PCR for herpesvirus DNA (HSV, VZV, CMV) in blood samples
- HIV viral load testing using RT-PCR



All patients received standard antiretroviral therapy (ART), and antiviral treatment for herpetic infections was administered according to national clinical protocols.

Statistical analysis was performed using SPSS software version XX. Results were expressed as mean \pm standard deviation (SD), and comparisons between groups were made using Student's t-test or chi-square test, with a significance level set at p < 0.05.

In total, 120 HIV-positive patients with confirmed herpetic infections were examined at the Bukhara Infectious Diseases Hospital. The average age of the patients was 38.4 ± 10.2 years, with 51.7% being male and 48.3% female. The most common co-infections were caused by Herpes Simplex Virus types 1 and 2 (46.7%), followed by Varicella-Zoster Virus (26.7%), Cytomegalovirus (16.7%), and mixed herpesvirus infections (10%).

Results and discussions

Immunological testing revealed that patients with severe or recurrent herpetic manifestations had significantly reduced CD4+ T-cell counts (mean 186 ± 52 cells/mm³) compared to those with milder forms (mean 342 ± 68 cells/mm³, p < 0.01). The majority of patients (84.2%) showed an inverted CD4/CD8 ratio, and elevated CD8+ T-cell counts were noted across the cohort. Analysis of cytokine profiles indicated increased levels of pro-inflammatory cytokines such as IL-6 and TNF- α , particularly in patients co-infected with CMV or disseminated VZV, whereas IFN- γ levels were markedly reduced in these cases.

Clinically, recurrent and chronic ulcerative lesions were most frequently observed in patients with HSV-2 infection, while disseminated herpes zoster involving multiple dermatomes occurred in patients with CD4+ counts below 150 cells/mm³. CMV-related complications, such as retinitis and colitis, were documented in patients with advanced immunosuppression.

Moreover, statistical analysis showed a significant correlation between higher HIV viral load and the increased frequency and severity of herpetic infections (p < 0.05). Patients receiving regular antiretroviral therapy with undetectable viral load experienced fewer and less severe episodes of herpesvirus reactivation.

The findings of this study confirm that the co-infection of HIV and Herpes Simplex Virus, particularly HSV-2, creates a complex interaction that worsens the clinical outcomes of both infections. The increased prevalence and severity of HSV infections in HIV-positive individuals are largely due to the compromised immune system caused by HIV. This immunosuppression facilitates frequent reactivation of HSV, leading to chronic and atypical lesions that are more difficult to treat. Moreover, HSV-induced mucosal ulcerations provide an entry point for HIV, increasing the risk of transmission and accelerating HIV replication. The bidirectional nature of this relationship means that controlling one infection can positively influence the course of the other. Suppressive antiviral therapy has been shown to reduce HSV outbreaks and may contribute to lowering HIV viral load, but it is not sufficient without effective antiretroviral therapy. The role of ART is crucial as it restores immune function, helping to reduce both HIV progression and HSV reactivation. These findings emphasize the need for integrated treatment strategies that address both infections simultaneously. Future research should focus on improving therapeutic options and developing vaccines to prevent HSV infection, which could potentially reduce HIV transmission rates.

Conclusion

The findings of this study demonstrate that herpetic infections are highly prevalent and clinically significant among HIV-infected patients, particularly those with advanced immunosuppression. A clear correlation was observed between low CD4+ T-cell counts, elevated HIV viral load, and the increased frequency and severity of herpesvirus reactivation. Immunological markers, including cytokine imbalances and reversed CD4/CD8 ratios, further underline the depth of immune dysfunction in coinfected individuals.

Early diagnosis through clinical assessment and immunological-laboratory monitoring, particularly CD4+ counts, cytokine profiles, and viral load measurements, is essential for timely initiation of antiviral therapy and optimization of antiretroviral treatment. The results suggest that integrated management of HIV and herpesvirus co-infections can significantly improve patient outcomes and reduce the burden of opportunistic infections in this population.

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