

Co-Infection of Herpes Family among HIV-I Positive Cases in Jaipur, RajasthanAshish Kumar Panda¹, Nitya Vyas², Pankaj Kumar Sharma³, Jitendra Panda⁴¹Ph.D. Scholar, Department of Microbiology, Mahatma Gandhi Medical College, Jaipur²Professor, Department of Microbiology, Mahatma Gandhi Medical College and Hospital, Jaipur³Ph.D. In Pharmacy, Diabetes, Thyroid and Endocrine Centre, Jaipur⁴Assistant Professor, Department of Microbiology, RUHS Medical College, Jaipur

Received: 13-05-2023 / Revised: 11-06-2023 / Accepted: 30-07-2023

Corresponding author: Ashish Kumar Panda

Conflict of interest: Nil

Abstract:

Acquired Immunodeficiency Syndrome (AIDS) is a medical condition caused by the Human Immunodeficiency Virus (HIV). The first recognized cases of AIDS were reported in the early 1980s, primarily among gay men in the United States. Coinfection of herpes family viruses, especially Herpes simplex virus (HSV) and Cytomegalovirus (CMV), is relatively common among HIV-1 positive individuals. HIV weakens the immune system, making individuals more susceptible to opportunistic infections, including infections caused by herpes viruses. In this study Total 150 HIV Positive patients were enrolled. Co-infection & Synergic effect of Herpesviridae viruses in HIV-1 Positive cases were correlated with CD-4 Count and HIV viral load. Total 8(5.33%) cases of multiple coinfections were found among 150 HIV-1 positive patients. It showed that due to poor immunity these multiple coinfections of herpes family was found among such cases.

Keywords: Human Immunodeficiency Virus (HIV); Herpes simplex virus (HSV).

This is an Open Access article that uses a funding model which does not charge readers or their institutions for access and distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>) and the Budapest Open Access Initiative (<http://www.budapestopenaccessinitiative.org/read>), which permit unrestricted use, distribution, and reproduction in any medium, provided original work is properly credited.

Introduction

Initially, the disease was misunderstood, and it took some time to identify the virus responsible for causing AIDS. In 1983-1984, researchers, primarily Dr. Luc Montagnier and his team at the Pasteur Institute in France, and later Dr. Robert Gallo and his team in the United States, independently discovered and identified the virus known as Human Immunodeficiency Virus (HIV) as the cause of AIDS.[1] As the virus spread, cases of AIDS were reported in various countries and among different populations, including heterosexual individuals, intravenous drug users, and recipients of contaminated blood products.[2]

HSV is a common viral infection that causes oral herpes (HSV-1) and genital herpes (HSV-2). In HIV-1 positive individuals, recurrent or severe herpes infections are more likely to occur due to the compromised immune system.[3] These herpes infections can be more challenging to treat and may require more aggressive antiviral therapy. CMV is another member of the herpes family and is usually asymptomatic in healthy individuals. However, in people with HIV-1, CMV can cause significant health issues; especially in cases of severe immunosuppression.[4-5] CMV retinitis is a well-known complication in advanced HIV disease, leading to vision problems and even blindness. CMV infections can also affect other organs,

including the gastrointestinal tract and central nervous system.[6] Coinfection with herpes viruses can pose several challenges for HIV-1 positive individuals, as these infections can exacerbate the immune deficiency and lead to more severe complications. Additionally, persistent herpes infections can increase viral replication of HIV-1, potentially leading to higher viral loads and increased risk of transmission to others.[7-8]

Materials & Methods

Total 150 HIV Positive patients were enrolled in this study. All the samples were collected from the patients attending ICTC at Mahatma Gandhi Medical College & Hospital, Jaipur from period 2019-22. Blood samples of HIV positive cases.

5 ml of whole blood sample was taken from each patient in 3ml Plain & 2ml EDTA vial. Such samples were centrifuged within 6 hours of collection at 4000 RPM for 10 minutes in order to separate serum/Plasma Each serum sample was distributed into two aliquots of 500µl.

one aliquot was used for RNA extraction; other part was used for herpeviridae serology test. EDTA samples were used for CD4 estimation.

Results: Total 8(5.33%) cases of multiple coinfections were found among 150 HIV-1 positive patients. Mean CD4+ count of cases were 230 & mean HIV-1 RNA viral load was 192110 IU/ml were found (Table 1). Maximum CD4+ count was observed in the Patient suffering from HSV1+EBV

co-infection (Fig. 1). While maximum HIV-1 RNA viral load was observed in the Patient suffering from HSV1+ VZV co-infection (Fig. 2). It showed that due to poor immunity these multiple coinfections of herpes family were found among such cases.

Table-1: Correlation of CD4 count & HIV-1 RNA viral load among multiple coinfection cases

HIV CASE ID	Multiple Coinfection	Total Cases	CD4+	HIV-1 RNA Viral Load (IU/ml)
HIV10	HSV1+ VZV	1	55	1030000
HIV93	HSV1+EBV	1	576	49700
HIV51	HSV2+CMV	1	122	Target not detected
HIV57	HSV2+CMV	1	134	87900
HIV29	HSV2+ HHV8	1	311	168000
HIV113	CMV+ EBV	1	340	7200
HIV132	CMV+VZV	1	55	1830
HIV149	CMV+HHV8	1	247	140

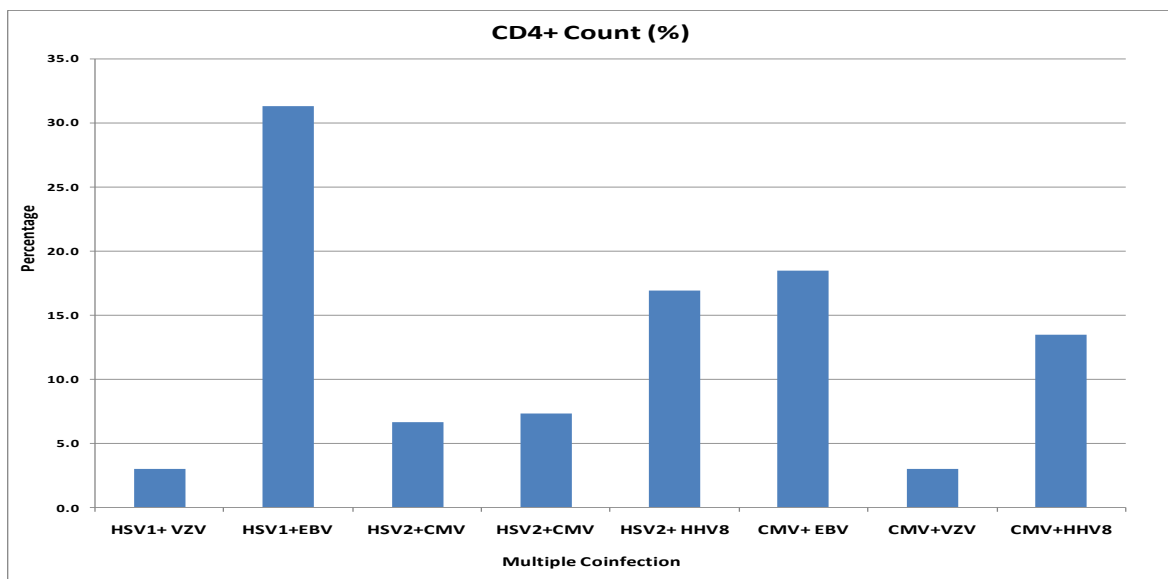


Figure 1: CD4 count among the multiple coinfection cases

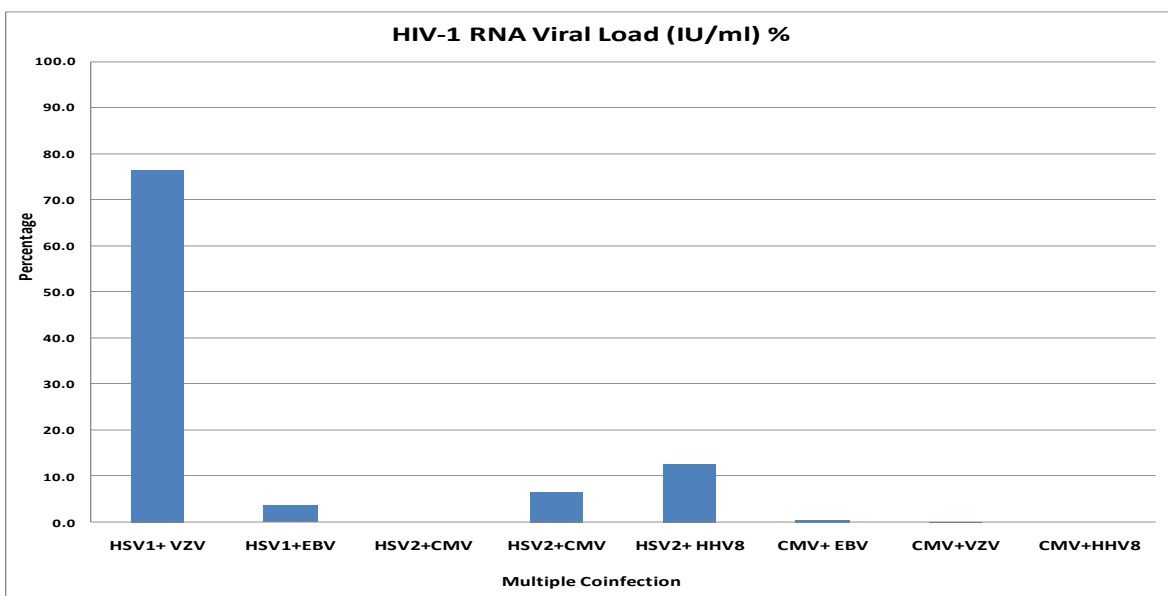


Figure 2: HIV-1 RNA viral load among the multiple coinfection cases

Discussion

The data represent Eight cases of HIV-positive individuals with different coinfections involving herpes family viruses. The CD4+ counts range from 55 to 576, indicating varying degrees of immune suppression across cases. The HIV-1 RNA viral load also varies significantly, with some cases showing high viral replication (e.g., 1,030,000 IU/ml) and others with viral loads not detected. Some coinfections involve the same herpes viruses (e.g., HSV2+CMV in two cases), indicating the diversity of coinfection possibilities. In a study conducted by da Silva Neto et al.[9] found the synergistically deleterious interaction of HIV with pro-oncogenic viruses. Viral infection-related cancers such as lymphomas (EBV-related), Kaposi-sarcoma (KSV-related), anal/cervical cancer (HPV-related), and liver cancer (HCV- and/or HBV/HDV-related) have a much higher incidence among HIV+ persons than in the general population.

Conclusion

Opportunistic viral infections play a major role in the progression of the disease in HIV infected patients. The co-infections with these viruses would require specific treatment when immunosuppression advances. Hence, it is important to detect these opportunistic viruses in HIV infected individuals to reduce morbidity and mortality.

References

1. Farzadegan H, Hoover DR, Astemborski J, Lyles CM, Margolick JB. Sex differences in HIV-1 viral load and progression to AIDS. *Lancet*. 1998; 352: 1510–1514.
2. Fauci AS. The human immunodeficiency virus: infectivity and mechanisms of pathogenesis. *Science*. 1988; 239: 617-16.
3. latzmann D, Barre-Sinoussi F, Nugeyre MT, et al. Selective tropism of lymphadenopathy-associated virus (LAV) for helper-inducer T lymphocytes. *Science* 1984; 1984; 225: 59-66.
4. Cohen MS, Chen YQ, McCauley M, Gamble T, Hosseinipour MC. Prevention of HIV-1 infection with early antiretroviral therapy. *N Engl J Med*. 2011; 365: 493–505.
5. Vaibhav SC, Arpit V, Supriya S, Kale BP. To Study Correlation of Oxidative Parameters and CD4 Count in HIV Patients Before and After 3 Month of HAART. *International Journal of Toxicological and Pharmacological Research* 2022; 12(9); 358-362.
6. Dodig M, Tavill AS. Hepatitis C and human immunodeficiency virus coinfections. *J Clin Gastroenterol*. 2001; 33:367-374.
7. Bhattacharya S, Badrinath S, Hamide A, Sujatha S. Co-infection with hepatitis C virus and human immunodeficiency virus among patients with sexually transmitted diseases in Pondicherry, South India. *Indian J Pathol Microbiol*. 2003; 46:495-497.
8. Rando RF, Pelett PE, Luciw PA, Bohan CA, Srinivasan A. Transactivation of human immunodeficiency virus by herpesviruses. *Oncogene* 1987; 1: 13-18.
9. Neto MM, Carlos B, Alvaro HB, Cancer during HIV infection. *APMIS*. 2020; 128(2): 121-128.