Treatment of primary HIV infection
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A case of Kaposi’s sarcoma during primary HIV-1 infection

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Abstract

The majority of cases of Kaposi’s sarcoma (KS) occur at low CD4 T-cell counts during chronic HIV-1 infection. We present a case of KS, which was diagnosed during primary HIV-1 infection. This report aims to draw attention that KS may occur early in the course of HIV-1 infection and that primary HIV-1 infection may rapidly progress to AIDS.
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Introduction

Kaposi’s sarcoma (KS) is a low-grade vascular tumour caused by human herpesvirus 8 (HHV8). It is one of the most common neoplasms in HIV-1 infected individuals. With the introduction of combination antiretroviral therapy (cART), the incidence of KS has decreased significantly. Traditionally, a low CD4 T-cell count during chronic HIV-1 infection was the most important factor associated with the development of KS, although recent studies have shown that the majority of KS now occurs at higher CD4 T-cell counts. We report a patient who presented with KS during primary HIV-1 infection (PHI).

Case Report

A 48-year-old homosexual man was diagnosed with PHI in June 2009, based on an indeterminate Western blot (p24 and gp120/160 antibodies were detected), and a plasma viral load (pVL) of 267,479 cps/ml. Three weeks prior to the diagnosis, the patient had experienced symptoms which were compatible with an acute retroviral syndrome: fever, sore throat, a dry cough, myalgia, fatigue and weight loss of 5 kg. Physical examination revealed no abnormalities. Laboratory investigations showed a mild anaemia, thrombocytopenia and elevated liver enzymes. CD4+ T-cell count was 210 cells/mm³, CD8+ 2310 cells/mm³ and CD4/8 ratio 0.09. No transmitted drug resistance mutations were present and the HIV-1 strain belonged to subtype B. Using MT-2 assay to define HIV-1 tropism, no CXCR4-tropic virus was detected. Hepatitis B/C and syphilis serology were negative.

11 weeks after HIV diagnosis, the patient discovered a small purple-brown lesion on his left forearm which was clinically suspect for KS (Figure 1). The diagnosis was confirmed with a skin biopsy. The CD4+ T-cell count had increased to 500 cells/mm³ and the pVL had dropped significantly to 64,218 cps/ml. The HHV8 load was 799 cps/ml. Retrospectively, sequential HHV8 loads were performed on stored plasma samples and HHV8 load at the time of PHI diagnosis was 163 cps/ml (Figure 2). Other possible underlying diseases like Castleman’s disease, another HHV8 related disease, were excluded.

Since the patient was recently diagnosed with PHI and was recovering from the PHI-associated transient low CD4+ T-cell count and peak pVL, we decided to defer cART to await spontaneous regression. During the following months, the KS lesion on the arm remained stable and no new lesions occurred. 36 weeks after HIV diagnosis, the CD4+ T-cell count had dropped to 310 cells/mm³; the pVL was 181,388 cps/ml and the HHV8 load had increased to 10,600 cps/ml (Figure 2). cART was initiated. The patient responded well to therapy. Within eight weeks, HIV-1...
viral suppression in plasma was achieved, CD4+ T-cell count increased to 430 cells/mm³, and plasma HHV8 became undetectable. The KS lesion disappeared within the following six months.

Discussion

The natural history of HIV-1 infection varies widely between patients and may be affected by viral and/or host factors. Rapid progression to AIDS shortly after PHI has been described previously6,7, including a case of KS diagnosed 24 months after PHI8. Our patient was symptomatic during PHI, which is a strong predictor of AIDS progression9. To our knowledge, this is the first patient in whom KS was diagnosed during PHI.

Unfortunately we were not able to determine the IgM and IgG antibody titers against HHV8 to distinguish between an acute HHV8 infection or a reactivation of an existing HHV8 infection. Since the patient had a transient immunosuppression and a peak pVL as a result of PHI, reactivation of KS seems likely, caused by the immunodeficiency itself and/or by direct effects of HIV-11.

This case demonstrates that KS may occur early in the course of HIV-1 infection and that symptomatic PHI may rapidly progress to AIDS.

Contributors

MLG drafted the manuscript. MLG and JMP treated the patient. MC retrieved the HHV8 results. All authors read and approved the final manuscript. The authors wish to thank prof. dr. Henry J.C. de Vries for his useful comments on the manuscript.

Consent

Informed consent for publication was obtained from the patient.

Figure 2. CD4+ T-cell count, HIV-1 RNA and HHV8 DNA in sequential plasma samples from the time of HIV-1 diagnosis. cART, combination antiretroviral therapy; KS, Kaposi’s sarcoma; PHI, Primary HIV-1 infection. Note. The filled symbols denote undetectable plasma HIV-1 RNA or HHV8 DNA.
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References


