AIDS-related Kaposis sarcoma pathogenesis

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Kaposis sarcoma (KS), the most common tumor associated with human immunodeficiency virus-I (HIV-I) and human herpesvirus-8 (HHV-8) (also referred to as Kaposis associated herpesvirus (KSHV) infection, develops in approximately 20 percent of patients infected with HIV-I. The lesions of this multicentric vascular neoplasm are purplish patches, plaques, or nodules. Although highly active antiretroviral therapy (HAART) can prolong the time to treatment failure in patients with KS, nearly every patient with AIDS-related (or epidemic) KS eventually develops disseminated disease. Progression usually occurs in an orderly fashion from new localized or widespread mucocutaneous lesions to more numerous lesions and generalized skin disease with involvement of lyrnph nodes, gastrointestinal tract (GIT), lungs and other organs. Studies have shown that KS cells themselves are not infected with HIV-I; therefore, it is widely accepted than HIV-I does not play a direct oncogenic role in AIDS-KS. However, the precise role of HIV-I in AIDS-KS is still not completely understood, and there is considerable debate over whether HIV-I plays a passive role (through the induction of immunosuppression) or a more direct role in the pathogenesis of this disease. We formulate a mathematical model to study the dynamics of HIV-I related KS pathogenesis. In this model, it is assumed that HIV-I infects only the CD4 + T cells and HHV-8 infects the B- cells, which largely remain latently infected and only become reactivated after exposure to inflammatory cytokines and other growth factors secreted from HIV-I infected cells. The Infection free and Infection persistent equilibria have been found and their stability established. It is found that the disease can exist even if both sub-group reproduction numbers, relating to HIV-I and HHV-8, are less than unity.

Keywords: Kaposi's sarcoma; HIV-I; HHV-8.