Kaposi’s sarcoma (KS) is a vascular neoplasm that is often found in a human immunodeficiency virus (HIV)-infected patient. Oral involvement is not uncommon, especially in those patients with acquired immune deficiency syndrome.1 In this paper, the clinicopathological features of a KS at the hard palate of an HIV-infected patient are reported.

A 49-year-old male patient was referred to the dental department of our hospital for evaluation of a swelling at the hard palate for several months. He was diagnosed as having HIV infection and syphilis for a few years. He just started to receive antiretroviral therapy for HIV infection and penicillin medication for syphilis. His CD4+ lymphocyte number was 3% of total lymphocytes with a viral load of 72,900 copies/mL. Intraoral examination revealed a soft tissue mass at the anterior and middle region of the hard palate. The tumor was red to purple with a surface ulceration on the anterior portion of the mass (Figure 1A). From the clinical presentation of the tumor and the past medical history of HIV infection, a KS was highly suspected. An incisional biopsy was performed by an oral surgeon. The histopathological examination of the biopsyed tissue section showed a neoplasm composed of spindle-shaped tumor cells arranged in a sheet and fascicular pattern. Numerous extravasated erythrocytes were present in the slit-like vascular spaces among tumor cells (Figures 1B and 1C). Immunohistochemical staining showed that approximately 60–70% of spindle-shaped tumor cells were positive for human herpesvirus 8 (HHV-8) viral antigens (Figure 1D). Therefore, the final histopathological diagnosis was a KS. The patient was referred to the Division of Infectious Diseases, Department of Medicine for further treatment.

It is well-established that the HHV-8 is responsible for the development of KS. HHV-8 is a human rhadinovirus that can be localized in KS tumor cells by in situ hybridization.1 However, immunohistochemistry is a more convenient technique in the pathological laboratory to help identify the specific cells or tumors,2,3 thus, the detection of HHV-8 can now be easily achieved in formalin-fixed, paraffin-embedded tissues with commercially available monoclonal antibodies to several different lytic and latent viral antigens.1 In addition to KS, HHV-8 is also strongly associated with multicentric Castleman’s disease, and a rare primary effusion B-cell lymphoma. The seroprevalence of HHV-8, based on detection of latent and lytic proteins, is 2–5%
for healthy donors and 40–50% for HIV-1 patients without KS. This virus can be transmitted both sexually and through saliva and blood. HHV-8 is a transforming virus, as evidenced by its presence in human malignancies, by the in vitro transforming properties of several of its viral genes, and by its ability to transform some primary cells in culture. However, HHV-8-induced transformation needs the help of other immunosuppressive cytokines to complete the development of HHV-8-associated malignancies. The presence of KS in the oral cavity indicates severe immune deterioration in our HIV-infected patient. The marked immune destruction (as evidenced by a low CD4+ T cell count of 3%) further helps the development of KS in our HIV-infected patient.

References