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CASE REPORT

Multifocal oral Kaposi sarcoma as inaugural manifestation of HIV infection: a clinical case report

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ABSTRACT

Oral Kaposi sarcoma (KS) may constitute the initial clinical manifestation of undiagnosed HIV infection, representing a significant diagnostic challenge in dental practice. As oral involvement often precedes cutaneous lesions, its presence should immediately prompt clinical investigation for underlying immunosuppression. A 32-year-old female with no relevant medical history or identifiable traditional risk factors for HIV presented with multifocal, asymptomatic oral lesions that had progressed over four months. Clinical examination revealed multiple red-purple nodules on the gingiva and palate. Histopathological analysis confirmed Kaposi sarcoma with positive HHV-8 immunostaining. Subsequent laboratory investigations revealed an advanced HIV infection, with a CD4 count of 98/mm³. The absence of conventional risk factors in this patient highlights an atypical presentation, emphasizing the need for high clinical vigilance. Management involving antiretroviral therapy (ART) combined with local chemotherapy led to complete lesion regression at the 6-month follow-up. The patient remained asymptomatic with sustained virological suppression at the 12-month mark. This case underscores the critical importance of recognizing oral Kaposi sarcoma as a potential inaugural sign of HIV. Oral healthcare providers must maintain a high index of suspicion when encountering characteristic violaceous oral lesions, regardless of the patient's demographic profile. Early recognition and multidisciplinary management are essential for achieving optimal clinical outcomes.

Keywords: Antiretroviral therapy, Diagnostic challenge, HIV infection, Immunosuppression, Oral Kaposi sarcoma.

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1. INTRODUCTION

Kaposi sarcoma (KS) is a multifocal angioproliferative neoplasm caused by human herpesvirus 8 (HHV-8), predominantly affecting immunocompromised individuals, particularly those with HIV infection (1). Since its original description by Moritz Kaposi in 1872, this condition has evolved from a rare entity to a significant health concern following the HIV/AIDS pandemic. Four distinct clinical variants are recognized: classic (chronic), endemic (African lymphadenopathic), iatrogenic (transplant-associated), and epidemic (AIDS-related) forms (2). While cutaneous manifestations are most common, oral cavity involvement occurs in 30-50% of HIV-associated KS cases, often preceding skin lesions and occasionally representing the initial clinical manifestation of undiagnosed HIV infection (3). Importantly, the Mediterranean basin—including Algeria—presents intermediate HHV-8 seroprevalence rates estimated at 15-25%, and regional HIV cohort data indicate that KS constitutes one of the most frequent AIDS-defining conditions in North Africa, affecting approximately 8-12% of newly diagnosed HIV patients; this epidemiological context underscores the particular relevance of KS recognition for practitioners operating in our regional healthcare setting.

The oral manifestations typically present as violaceous macules, plaques, or nodules, with predilection for the hard palate and attached gingiva (4). Recognition of these characteristic lesions is critical for early HIV diagnosis and timely therapeutic intervention. However, the clinical presentation can be challenging, particularly in patients without known risk factors or when lesions exhibit atypical features. This case report presents a diagnostically challenging case of multifocal oral KS in a young woman with no known medical history, which ultimately revealed previously undiagnosed advanced HIV infection. We emphasize the diagnostic approach, treatment outcomes, and the critical role of oral healthcare providers in identifying HIV-associated oral pathology.

2. CASE REPORT

A 32-year-old female patient presented to the Department of Oral Pathology and Surgery with progressive multifocal oral lesions that had developed over 4 months. The patient reported no pain, hemorrhage, or functional impairment but expressed concern regarding the aesthetic appearance and gradual enlargement of the lesions. She denied fever, weight loss, night sweats, or other systemic symptoms. The patient had no known medical conditions, was not taking any medications, and had never undergone HIV testing. No traditional risk factors for HIV infection were identified during initial history-taking.

Extraoral examination revealed no cervical, submandibular, or supraclavicular lymphadenopathy. No cutaneous lesions were observed on visible skin surfaces. The patient appeared alert and cooperative without signs of acute distress. Intraoral examination demonstrated multiple red-purple nodular lesions ranging from 0.5 to 1.5 cm in diameter, located on the attached gingiva of the maxillary anterior region extending to the hard palate (Figure 1). Specifically, three lesions were identified: a dominant gingival nodule measuring 1.5 × 1.2 cm at the level of teeth 11-12, a second gingival lesion measuring 0.8 × 0.7 cm at the level of teeth 21-22, and a palatal nodule measuring 1.0 × 0.9 cm in the anterior hard palate region. These individual measurements are provided to allow objective quantification of therapeutic response at subsequent follow-up visits. The lesions exhibited smooth, non-ulcerated surfaces with characteristic violaceous coloration. Digital pressure did not result in blanching. Surrounding oral tissues appeared normal with no evidence of bone involvement or tooth mobility. Oral hygiene was fair with mild plaque accumulation. Comparative photographic documentation at 6 and 12 months post-treatment was planned in order to provide objective visual evidence of lesion regression; acquisition of these before/after images remains an important complement to the clinical description and should be included in future communications on this case.

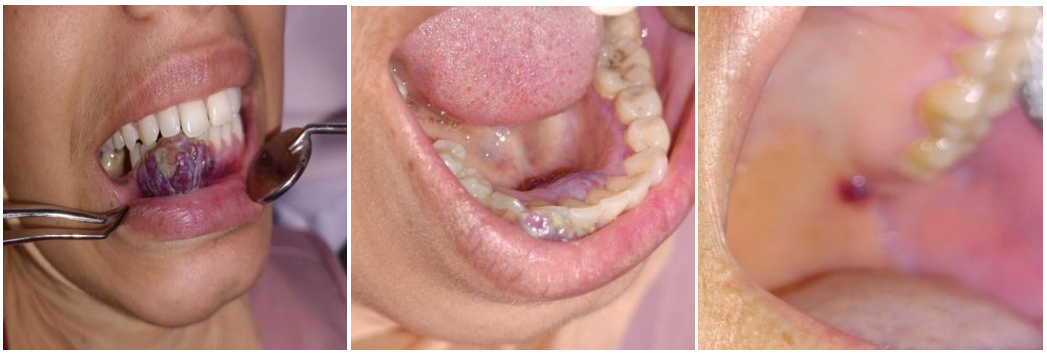

Figure 1. Initial clinical presentation demonstrating multifocal violaceous nodules on the maxillary gingiva and hard palate. Note the characteristic purple coloration and smooth surface of the lesions.

Based on the clinical presentation, Kaposi sarcoma was the primary consideration given the violaceous color, multifocal distribution, and palatal/gingival location. Alternative diagnoses included pyogenic granuloma, peripheral giant cell granuloma, hemangioma or other vascular malformations, bacillary angiomatosis, and angiosarcoma. The characteristic clinical features strongly suggested Kaposi sarcoma, prompting immediate biopsy and comprehensive medical evaluation including HIV screening. Bacillary angiomatosis (BA) merits specific comment, as it can closely simulate KS in immunocompromised patients, both clinically and histopathologically. In our patient, BA was formally excluded on the following grounds: (1) immunohistochemical analysis demonstrated diffuse HHV-8 (LANA-1) nuclear positivity, which is absent in BA; (2) Warthin-Starry silver staining performed on the biopsy specimen did not reveal the bacillary clusters characteristic of Bartonella spp. infection; and (3) serology for Bartonella henselae and Bartonella quintana was negative. This formal exclusion is particularly important in the context of advanced immunosuppression (CD4+ count 98 cells/mm^3), where BA represents a genuine diagnostic pitfall.

An incisional biopsy was performed under local anesthesia (2% lidocaine with 1:100,000 epinephrine) from the largest gingival lesion. The specimen was fixed in 10% neutral buffered formalin and submitted for histopathological and immunohistochemical examination (Figure 2).

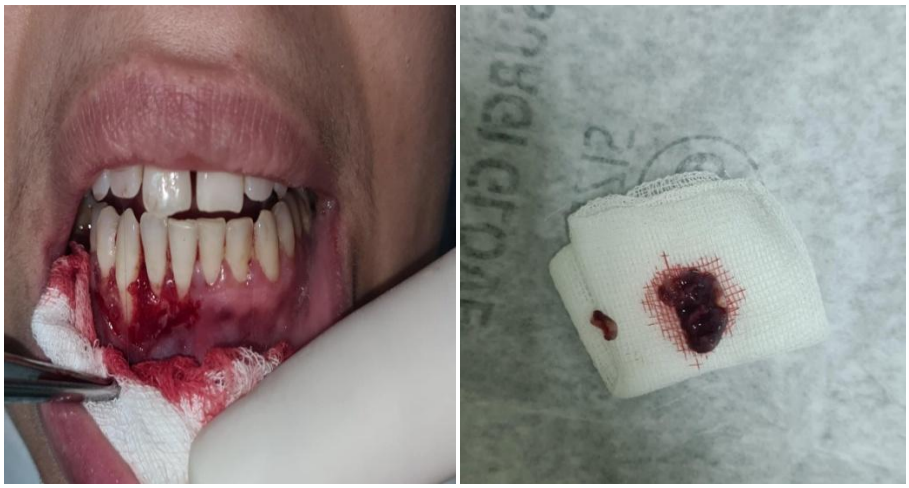

Figure 2. Post-biopsy appearance of the gingival lesion site (left) and excised surgical specimen (right).

Microscopic examination with H&E staining revealed proliferation of spindle-shaped cells arranged in interlacing fascicles, numerous dilated slit-like vascular spaces containing extravasated red blood cells, prominent hemosiderin deposition throughout the lesion, spindle cells with mild nuclear atypia and occasional mitotic figures, and chronic inflammatory infiltrate composed of lymphocytes and plasma cells (Figure 3). Immunohistochemical studies demonstrated HHV-8 (LANA-1) diffuse positive nuclear staining in spindle cells (Figure 4), CD31 positive staining in endothelial cells, CD34 positive in spindle cell population, and variable Factor VIII positivity. The final histopathological diagnosis confirmed Kaposi sarcoma.

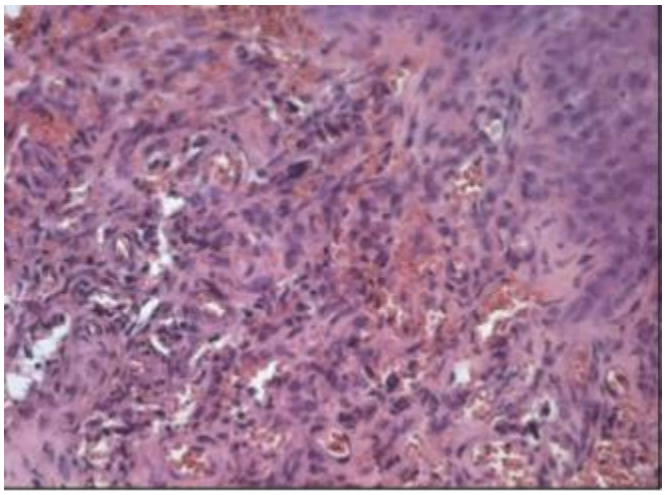

Figure 3. Histopathological examination (H&E staining, ×200) showing characteristic spindle cell proliferation with slit-like vascular spaces and numerous extravasated red blood cells.

Following histopathological confirmation, comprehensive laboratory evaluation was performed. HIV serology and immunological assessment showed HIV-1 ELISA positive, HIV-1 Western Blot positive (confirmatory), CD4+ T-cell count 98 cells/mm³ (reference range: 500-1500 cells/mm³), HIV viral load 150,000 copies/mL, and CD4/CD8 ratio 0.12 (reference range: >1.0). Complete blood count revealed hemoglobin 9.2 g/dL indicating anemia, total leukocyte count 3,200 cells/mm³ indicating leukopenia, total lymphocytes 800 cells/mm³ indicating lymphopenia, and platelets 180,000/mm³ within normal limits. Additional investigations including hepatitis B

surface antigen, hepatitis C antibody, and syphilis serology were all negative. Chest radiograph showed no pulmonary lesions, and abdominal ultrasonography revealed no hepatosplenomegaly or lymphadenopathy.

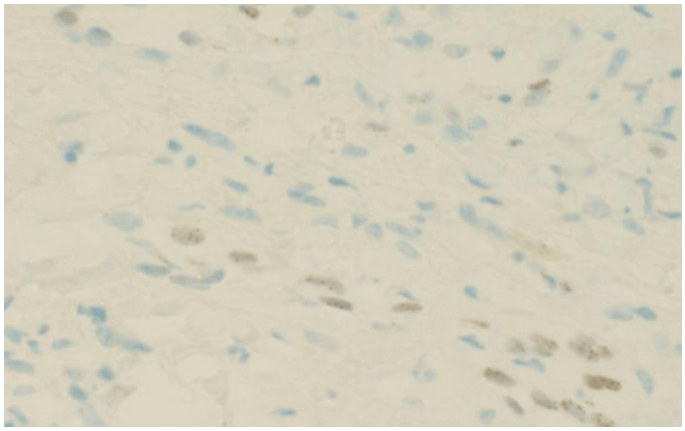

Figure 4. Immunohistochemical staining for HHV-8 (LANA-1, ×400) demonstrating positive nuclear staining in spindle cells, confirming Kaposi sarcoma diagnosis.

Extension workup included complete dermatological examination which identified no cutaneous KS lesions, lymph node examination showing no peripheral lymphadenopathy, ophthalmological examination revealing no ocular involvement, CT scan of head and neck demonstrating soft tissue thickening in palatal region corresponding to clinical lesions with no osseous involvement or pathological lymph nodes, and chest CT showing no evidence of pulmonary KS or opportunistic infections. The investigations confirmed advanced HIV infection (CDC Category C3) with isolated oral KS without systemic dissemination.

A multidisciplinary treatment plan was established in consultation with infectious disease specialists, oncologists, and oral surgeons. The treatment included Highly Active Antiretroviral Therapy (HAART) consisting of Tenofovir disoproxil fumarate/Emtricitabine 300/200 mg once daily and Efavirenz 600 mg once daily at bedtime. Local treatment for oral KS lesions involved intralesional vinblastine chemotherapy (0.1 mg/mL) administered at 2-week intervals to larger lesions for a total of 4 treatment sessions. Prophylactic therapy with Trimethoprim-sulfamethoxazole 960 mg three times weekly for Pneumocystis jirovecii pneumonia prophylaxis was initiated. Supportive care included nutritional counseling and multivitamin supplementation with regular monitoring of viral load, CD4 count, and liver/renal function. Patient education regarding HIV transmission prevention, importance of medication adherence, and recognition of symptoms requiring immediate medical attention was provided.

At 3-month follow-up, significant reduction in oral KS lesion size (>50% reduction) was observed. CD4+ count improved to 180 cells/mm³, HIV viral load decreased to 2,500 copies/mL, no new oral or cutaneous lesions appeared, and good medication tolerance was noted. At 6-month follow-up, complete clinical resolution of oral KS lesions was achieved. CD4+ count reached 385 cells/mm³, HIV viral load became undetectable (<50 copies/mL), hemoglobin normalized to 12.8 g/dL, and excellent oral hygiene maintenance was observed. At 12-month follow-up, continued complete remission of oral KS was maintained with CD4+ count 450 cells/mm³, HIV viral load persistently undetectable, no adverse effects from antiretroviral therapy, and excellent medication adherence maintained. The patient achieved complete clinical and virological response, demonstrating the efficacy of early diagnosis and appropriate treatment initiation.

3. DISCUSSION

This case represents a diagnostically challenging presentation of oral KS as the inaugural manifestation of previously undiagnosed HIV infection in a young female patient without apparent risk factors. Several features distinguish this case from typical presentations described in the literature and warrant detailed analytical comparison. First, the patient's demographic profile—a 32-year-old female with no known medical history or identified HIV risk factors—represents an atypical presentation. Most published series report oral KS predominantly in males with a male-to-female ratio approximately 8:1 and with known HIV infection or identified risk factors (5). Reznik (2005) documented that HIV-associated KS occurs primarily in men who have sex with men and in populations with high HHV-8 seroprevalence (5). This case underscores that HIV-associated KS can occur in individuals without traditional epidemiological risk

profiles, emphasizing the importance of maintaining clinical suspicion based solely on lesion characteristics rather than patient demographics. Second, the multifocal oral presentation without concurrent cutaneous involvement is relatively uncommon. While Patton et al. (2013) reported that oral lesions may precede skin manifestations in 15-20% of AIDS-related KS cases (3), isolated oral disease at initial presentation occurs in only approximately 8-12% of cases according to recent multicenter studies by Mosam et al. (2012) (6). Our patient's presentation with exclusive oral involvement at diagnosis highlights the critical role of oral healthcare providers as potential first-contact clinicians for HIV detection. This finding is particularly significant given that many patients may present to dental clinics before seeking medical care, making oral healthcare providers key participants in early HIV diagnosis.

Third, the relatively advanced immunosuppression (CD4+ count 98 cells/mm³) despite the absence of other AIDS-defining conditions or clinical symptoms suggests prolonged undiagnosed HIV infection. According to Centers for Disease Control classification, a CD4 count below 200 cells/mm³ defines AIDS regardless of clinical symptoms. The fact that our patient remained relatively asymptomatic despite such profound immunosuppression raises important questions about the natural history of HIV infection and emphasizes the need for routine HIV screening in dental practice when encountering suspicious oral lesions, regardless of patient-reported medical history or symptomatology.

The primary diagnostic challenge in this case was the initial absence of known HIV status combined with the patient's atypical demographic profile. Recent literature by Schwartz (2004) emphasizes that delayed recognition of oral KS contributes to late HIV diagnosis in approximately 5-8% of new HIV cases in endemic regions (7). In our case, the characteristic violaceous coloration and multifocal distribution on the palate and gingiva provided crucial diagnostic clues that prompted immediate investigation. Comparison with similar case series reveals important diagnostic patterns. Lager et al. (2003) analyzed 70 oral KS cases from South Africa and noted that correct clinical diagnosis was achieved in only 62% of cases prior to biopsy, with inflammatory hyperplasias being the most common misdiagnosis (4). In contrast, our immediate clinical suspicion was facilitated by recognition of the pathognomonic violaceous color and typical anatomical distribution, highlighting the value of systematic clinical examination and awareness of HIV-associated oral pathology.

The differential diagnosis in this case required careful consideration of several entities. Unlike pyogenic granulomas, which typically present as solitary pedunculated lesions with friable surfaces prone to bleeding, our patient's lesions were multifocal, sessile, and non-hemorrhagic (8). Peripheral giant cell granulomas, while potentially purple-blue in color, generally occur as solitary gingival masses (9). The multifocality and palatal involvement strongly suggested KS over these alternative diagnoses. The absence of blanching on digital pressure, a characteristic feature of true neoplastic vascular proliferation, further distinguished KS from reactive vascular lesions.

The histopathological diagnosis demonstrated classic features of KS including spindle cell proliferation, slit-like vascular spaces, and extravasated erythrocytes. However, the critical diagnostic element was HHV-8 (LANA-1) immunohistochemistry, which showed diffuse nuclear positivity confirming the diagnosis. Schwartz et al. (2008) demonstrated that HHV-8 immunohistochemistry has 95-100% sensitivity and specificity for KS diagnosis, making it superior to clinical assessment alone and essential for definitive diagnosis (10). In diagnostically challenging cases, particularly early patch-stage lesions that may lack characteristic histological features, HHV-8 immunostaining provides definitive confirmation. The immunohistochemical profile in our case (CD31+, CD34+, HHV-8+) aligns with established diagnostic criteria for KS. The variable Factor VIII positivity, as observed in our case, is well-documented in the literature and reflects the mixed endothelial-mesenchymal phenotype of KS spindle cells (11).

The management strategy employed in this case—combining HAART with local intralesional chemotherapy—reflects current evidence-based approaches for localized oral KS. Martinez et al. (2006) demonstrated that HAART alone achieves complete or partial remission in approximately 60-80% of localized KS cases through immune reconstitution (8). The mechanism involves restoration of immune surveillance against HHV-8 infected cells and reduction in inflammatory cytokines that promote KS tumor growth. However, the addition of local therapy may accelerate lesion regression and improve cosmetic outcomes, particularly for anterior oral lesions affecting patient quality of life and causing aesthetic concerns, as in our case. Our patient's excellent response, with complete clinical resolution at 6 months, compares favorably with published outcomes. Mosam et al. (2010) in their systematic review reported median time to complete response of 8-12 months with HAART alone, and 4-6 months when combining HAART with local therapy (12). Our 6-month timeline for complete resolution supports the benefit of combined treatment approaches for symptomatic or cosmetically concerning oral KS.

The virological and immunological outcomes were equally impressive and merit detailed analysis. The rise in CD4+ count from 98 to 450 cells/mm³ over 12 months, accompanied by sustained viral suppression to undetectable levels, demonstrates effective immune reconstitution. This degree of immune recovery correlates with significantly reduced risk of KS recurrence. Bower et al. (2014) documented in their prospective study that recurrence rates are less than 5% when CD4 counts are maintained above 350 cells/mm³ with sustained viral suppression (13). Our patient's achievement of CD4 count above 450 cells/mm³ suggests excellent long-term

prognosis. The rapid virological response, achieving undetectable viral load within 6 months, is consistent with optimal adherence to antiretroviral therapy and absence of drug resistance mutations.

The patient's North African origin warrants consideration of regional HHV-8 epidemiology. HHV-8 seroprevalence varies dramatically geographically, ranging from less than 5% in Northern Europe and North America to 30-50% in Mediterranean and sub-Saharan African populations (14). Algeria, located in the Mediterranean basin, has intermediate HHV-8 seroprevalence estimated at 15-25%, which may contribute to increased KS incidence among HIV-positive individuals in this region. Recent data from North African HIV cohorts indicate that KS remains one of the most common AIDS-defining conditions in this region, occurring in approximately 8-12% of newly diagnosed HIV patients (15). This emphasizes the continued importance of KS recognition in Mediterranean countries despite global advances in HIV care.

This case exemplifies the critical role of oral healthcare providers in early HIV detection. Dental professionals are often among the first healthcare providers to encounter oral manifestations of HIV infection. Recent surveys have found that 12-15% of newly diagnosed HIV patients had visited dental clinics in the preceding year with unrecognized HIV-associated oral lesions (16). This represents a significant missed opportunity for earlier diagnosis and intervention. The importance of systematic HIV testing following oral KS diagnosis cannot be overstated. Current guidelines from the American Academy of Oral Medicine recommend HIV screening for all patients presenting with oral KS, regardless of known HIV status or perceived risk factors (17). Our case reinforces this recommendation, as the patient had no identified risk factors yet harbored advanced HIV infection that would have remained undiagnosed without oral examination prompting investigation.

The multidisciplinary approach employed in this case—involving oral surgeons, infectious disease specialists, pathologists, and oncologists—represents optimal management strategy. This collaborative approach ensures comprehensive assessment, appropriate treatment selection, and adequate follow-up, ultimately improving patient outcomes. The coordination between different specialties allowed for simultaneous management of both the oral lesions and underlying systemic disease, optimization of antiretroviral regimen, monitoring for immune reconstitution inflammatory syndrome, and early detection of potential complications or treatment side effects.

4. CONCLUSION

This case illustrates the pivotal role of oral healthcare providers in detecting HIV infection through its inaugural oral manifestations. Characteristic violaceous multifocal lesions affecting the palate and gingiva must immediately prompt suspicion for Kaposi sarcoma, regardless of patient demographics or the absence of conventional risk factors. Definitive diagnosis relies on the integration of clinical assessment, HHV-8 immunohistochemistry, and comprehensive HIV workup including CD4+ count and viral load. Multidisciplinary management combining early HAART initiation and local intralesional therapy can achieve remarkable outcomes, as evidenced in this case by complete clinical remission, immune reconstitution from 98 to 450 CD4+ cells/mm^3, and sustained virological suppression within 12 months. These results reinforce the continued importance of KS recognition in Mediterranean regions with intermediate HHV-8 endemicity, and support the systematic integration of HIV screening protocols into dental practice guidelines to reduce diagnostic delays in this vulnerable population.

Abbreviations

KS: Kaposi Sarcoma; HIV: Human Immunodeficiency Virus; HHV-8: Human Herpesvirus 8; HAART: Highly Active Antiretroviral Therapy; CT: Computed Tomography; H&E: Hematoxylin and Eosin; CD: Cluster of Differentiation; ELISA: Enzyme-Linked Immunosorbent Assay; LANA: Latency-Associated Nuclear Antigen

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REFERENCES

1. Antman KH, Chang Y. Kaposi's sarcoma. N Engl J Med. 2000;342(14):1027-38. https://doi.org/10.1056/NEJM200004063421407
2. Friedman-Kien AE. Disseminated Kaposi's sarcoma syndrome in young homosexual men. J Am Acad Dermatol. 1981;5(4):468-71. https://doi.org/10.1016/s0190-9622(81)80010-2

3. Patton LL. Oral lesions associated with human immunodeficiency virus disease. Dent Clin North Am. 2013;57(4):673-98. https://doi.org/10.1016/j.cden.2013.07.005
4. Lager I, Altini M, Coleman H, Ali H. Oral Kaposi's sarcoma: a clinicopathologic study from South Africa. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2003;96(6):701-10. https://doi.org/10.1016/s1079-2104(03)00370-6
5. Reznik DA. Oral manifestations of HIV disease. Top HIV Med. 2005;13(5):143-8.
6. Mosam A, Shaik F, Uldrick TS, Esterhuizen T, Friedland GH, Scadden DT, et al. A randomized controlled trial of highly active antiretroviral therapy versus highly active antiretroviral therapy and chemotherapy in therapy-naive patients with HIV-associated Kaposi sarcoma in South Africa. J Acquir Immune Defic Syndr. 2012;60(2):150-7. https://doi.org/10.1097/QAI.0b013e318251aedd
7. Schwartz RA. Kaposi's sarcoma: an update. J Surg Oncol. 2004;87(3):146-51. https://doi.org/10.1002/jso.20090
8. Martinez V, Caumes E, Gambotti L, Ittah H, Morini JP, Deleuze J, et al. Remission from Kaposi's sarcoma on HAART is associated with suppression of HIV replication and is independent of protease inhibitor therapy. Br J Cancer. 2006;94(7):1000-6. https://doi.org/10.1038/sj.bjc.6603056
9. Coelho CM, Zucoloto S, Lopes RA. Denture-induced fibrous inflammatory hyperplasia: a retrospective study in a school of dentistry. Int J Prosthodont. 2000;13(2):148-51.
10. Schwartz RA, Micali G, Nasca MR, Scuderi L. Kaposi sarcoma: a continuing conundrum. J Am Acad Dermatol. 2008;59(2):179-206. https://doi.org/10.1016/j.jaad.2008.05.001
11. Stürzl M, Ascherl G, Blasig C, Opalka B, Ensoli B, Browning PJ. Expression of platelet-derived growth factor and its receptor in AIDS-related Kaposi sarcoma in vivo suggests paracrine and autocrine mechanisms of tumor maintenance. Proc Natl Acad Sci USA. 1995;92(10):4657-61.
12. Mosam A, Aboobaker J, Shaik F. Kaposi's sarcoma in sub-Saharan Africa: a current perspective. Curr Opin Infect Dis. 2010;23(2):119-23. https://doi.org/10.1097/QCO.0b013e328335b01a
13. Bower M, Dalla Pria A, Coyle C, Andrews E, Tittle V, Dhoot S, et al. Prospective stage-stratified approach to AIDS-related Kaposi's sarcoma. J Clin Oncol. 2014;32(5):409-14. https://doi.org/10.1200/JCO.2013.51.6757
14. Dedicoat M, Newton R. Review of the distribution of Kaposi's sarcoma-associated herpesvirus (KSHV) in Africa in relation to the incidence of Kaposi's sarcoma. Br J Cancer. 2003;88(1):1-3. https://doi.org/10.1038/sj.bjc.6600745
15. Janier M, Hegyi V, Dupin N, Unemo M, Tiplica GS, Potočnik M, et al. 2014 European guideline on the management of syphilis. J Eur Acad Dermatol Venereol. 2014;28(12):1581-93. https://doi.org/10.1111/jdv.12734
16. Shiboski CH, Webster-Cyriaque JY, Ghannoum M, Greenspan D, Greenspan JS. An update on oral lesions of HIV disease. Dent Clin North Am. 2019;63(4):583-613.
17. Ramos-Gomez F, Folayan MO, Anil S, Pandya A, Jayaraman J, Oski ME, et al. Pediatric HIV: the role of the dental provider in early identification. Pediatr Dent. 2020;42(4):253-262.