AIDS

On June 5, 1981, when the Centers for Disease Control (CDC) reported five cases of *Pneumocystis carinii* pneumonia in young homosexual men in Los Angeles, few suspected that it heralded a pandemic of acquired immunodeficiency syndrome (AIDS). In 1983, a retrovirus (later named the human immunodeficiency virus, or HIV) was isolated from a patient with AIDS

Incidence and Prevalence

At the end of 2003, an estimated 1,039,000 to 1,185,000 persons in the United States were living with HIV/AIDS; in 24% to 27%, the condition remained undiagnosed, and patients were unaware of their HIV infection. In 2004, more than 4.9 million people were infected by HIV, and about 3.1 million individuals died No vaccine or defi nitive treatment exists for this nationwide epidemic. Currently approved groups of antiretroviral drugs (highly active antiretroviral therapy [HAART]) may help slow the progression of infection, but no cure is yet known. The best treatment approach for AIDS, as for other infectious diseases, continues to be public health measures such as preventive education, early detection, and counseling for infected patients and their contacts (family, lover, friends, coworkers) and the use of drugs to slow the progress of the disease. Recent changes in the epidemiology of AIDS have been a positive testimony to this approach

Pathophysiology and Complications

In addition to its transfer by sexual means and the parenteral transfer of infected blood, AIDS may be transmitted vertically, probably at birth, or transplacentally to infants born of infected mothers. The most common method of sexual transmission in the United States is homosexual anal intercourse; however, heterosexual transmission has been documented from infected males to noninfected females. Transmission from infected females to noninfected males also has been reported. Heterosexual transmission of HIV can occur through sexual contact of carriers who are heterosexual intravenous (IV) drug users, bisexual males, or blood recipients of either gender. HIV has been found in saliva, but transmission via saliva has not

been demonstrated. HIV has also been isolated from tears, breast milk, cerebrospinal fluid, amniotic fluid, and urine. However, blood, semen, breast milk, and vaginal secretions are the only fluids that have been shown to be associated with transmission of the virus. Casual contact has not been demonstrated as a means of transmission.

By the year 2006, more than 1.9 million Americans were estimated to have been exposed to the AIDS virus. Current data suggest that most if not all of these individuals will develop AIDS as defi ned by the CDC. Antibody positivity to HIV means that the person has been infected with the virus and can be viremic. Individuals who appear to be most susceptible to developing AIDS are those with repeated exposure to the virus who also have an immune system that has been challenged by repeated exposure to various antigens (semen, hepatitis B, or blood products). Individuals infected with the virus develop antibodies, usually within 6 to 12 weeks. Most infected individuals develop a viremia within 2 to 6 weeks. A few may take up to 6 months to achieve seroconversion. In rare cases, as long as 35 months may be required for seroconversion to occur. The incubation period for AIDS appears to be lengthy for most individuals (mean, 10 to 12 years). Only about 30% of individuals with AIDS are dead within 3years of diagnosis, whereas approximately 50% live beyond 10 years. Once it has gained access to the bloodstream, HIV selectively seeks out T lymphocytes (specifically T4 or T-helper lymphocytes). The virus binds to the CD4 lymphocyte cell surface specifi cally through the highly glycosylated outer surface envelope (gp120) proteins. Upon infection, reverse transcriptase catalyzes the synthesis of a haploid, double-stranded DNA provirus, which becomes incorporated into the chromosomal DNA of the host cell. Thus integrated, the provirus genetic material may remain latent in an unexpressed form until events occur that activate it, at which time DNA transcription rapidly occurs and new virions are produced. The virus is lymphotropic; hence, the cells it selects for replication are soon destroyed. Once the virus has taken hold, it soon causes a reduction in the total number of T-helper cells, and a marked shift in the ratio of T4 to T8 lymphocytes occurs. The normal ratio of T-helper to T-suppressor lymphocytes is about 2:1 (60% T-helper, 30% T-suppressor). In AIDS, the T4/T8 ratio is reversed. This marked reduction in T-helper lymphocytes, to a great degree, explains the lack of immune

response seen in patients with AIDS and most likely is related to the increase in malignant disease that has been found to be associated with AIDS, including Kaposi's sarcoma, lymphoma, carcinoma of the cervix, and carcinoma of the rectum.

Most (more than 50%) individuals exposed to the virus at first develop an acute, brief viremia (seroconversion sickness) within 2 to 6 weeks of HIV exposure. A concomitant, temporary fall occurs in CD4 cells (lymphopenia, along with high titers of plasma HIV), but patients do not develop evidence of immune suppression. Various flulike symptoms occur in this acute seroconversion sickness that usually last about 2 to 4 weeks. Only an estimated 20% of these individuals seek medical attention. These individuals respond by producing antibodies to HIV (i.e., anti-gp120 and anti-p24), and cytotoxic T lymphocyte levels increase. Immune abnormalities in HIV disease consist of progressive depletion of CD4+ T lymphocytes with ultimate pancytopenia, impaired lymphocyte proliferation, and cytokine responses to mitogens and antigens; impaired cytotoxic lymphocyte function and natural killer cell activity; anergy to skin testing; and diminished antibody responses to new antigens.The virus also may infect neurons or macrophages in the CNS, allowing its presence within the body in latent form. Individuals may demonstrate a viremia on occasion and hence are considered carriers of the virus who have the potential to infect others. Of special concern is the fact that circulating antibodies fail to neutralize the virus because it has the capacity to alter its antigenicity. An alarming characteristic of these patients, in addition to their potential to be infectious and to develop AIDS, is that approximately 50% develop signs of dementia that can be rapidly progressive. In most cases, after a long asymptomatic period, the CD4 count continues to drop and HIV continues to proliferate; in addition, the infected individual may develop signs and symptoms such as lymphadenopathy, fever, weight loss, diarrhea, night sweats, pharyngitis, rashes, myalgia and arthralgia, headache, neuropathy and malaise, and eventually,AIDS

CLINICAL PRESENTATION

Signs and Symptoms

During the first 2 to 6 weeks after initial infection with HIV, more than 50% of patients develop an acute flulike viremia that may last 10 to 14 days.

Others may not manifest this symptom complex. Patients have HIV but are antibody negative. They usually experience seroconversion within 6 weeks to 6 months and then demonstrate antibodies. The severity of the initial acute infection with HIV is predictive of the course the infection will follow. In one study, 78% of individuals with a long-lasting acute illness developed AIDS within 3 years; by contrast, only 10% of those individuals with no acute illness at seroconversion developed AIDS within 3 years. Once exposure to HIV and seroconversion have occurred, three groups of patients may be identified.

Group 1. Immediate post-HIV exposure. After a brief postseroconversion sickness syndrome, these individuals are antibody positive to HIV but are asymptomatic and show no other laboratory abnormalities.

Group 2. Progressive immunosuppression, HIVsymptomatic stage. Individuals who demonstrate various laboratory changes (i.e., lymphopenia: T-helper/TFigure suppressor ratio usually less than 1) in addition to HIV antibody positivity also may show some clinical signs or symptoms, such as enlarged lymph nodes, night sweats, weight loss, oral candidiasis, fever, malaise, and diarrhea.

Group 3. Individuals who have AIDS, including Kaposi's sarcoma, wasting syndrome, lymphoma, cervical or rectal carcinoma, CNS symptoms with dementia, a life-threatening opportunistic infection (i.e., tuberculosis, pneumonia), a CD4 count of less than 200, and an altered T-helper/T-suppressor ratio of 0.5 or less. They are HIV antibody positive and can demonstrate generalized lymphadenopathy with severe weight loss, fatigue, chronic diarrhea, chronic fever, and night sweats . HIV may infect the CNS and often leads to a progressive form of dementia. Patients may become confused and disoriented or may experience short-term memory defi cits. Others develop severe depression or paranoia and show suicidal tendencies.

Laboratory Findings

HIV can be isolated from the blood, semen, breast milk, tears, and saliva of many patients with AIDS. Most patients exposed to the virus, with or without clinical evidence of disease, show antibodies to the virus. Patients with advanced HIV infection or AIDS have an altered ratio of T4/T8 lymphocytes, a decrease in total number of lymphocytes, thrombocytopenia, anemia, a slight alteration in the humoral antibody system, and a decreased

ability to show delayed allergic reactions to skin testing (cutaneous anergy). In 1985, several screening tests became available for identifi cation of antibodies to HIV. The enzyme-linked immunosorbent assay (ELISA) is sensitive but has a high rate of false-positive results. Current practice is to screen first with ELISA. If the first results are positive, a second ELISA is performed. All positive results are then rescreened with a second test, the Western blot analysis. This combination of screening tests is accurate more than 99% of the time. Positive ELISA and Western blot test results indicate only that the individual has been exposed to the AIDS virus. If results of the Western blot are indeterminate, HIV infection is rarely, if ever, positive.
These tests, however, do not indicate the status of the HIV infection or whether AIDS is present. Neither do they show if the patient is viremic because a special test, the DNA polymerase chain reaction (PCR) for direct detection of the virus, would need to be performed. This test is considerably more expensive. However, patients with positive results from the ELISA and Western blot tests are considered potentially infectious.

MEDICAL MANAGEMENT

No effective treatment or cure for AIDS is known. Antiviral agents have been unsuccessful in killing the HIV virus. However, zidovudine (AZT or Retrovir) has been shown to exert signifi cant inhibitory effects on in vitro replication and the cytopathogenicity of HIV. AZT has been found to prolong life in both asymptomatic and symptomatic HIV-infected individuals, although no evidence suggests that AZT is effective in preventing infection once exposure to the virus has occurred Medical management of the HIV-infected patient must include counseling regarding safe sexual practices, how to avoid the spread of HIV, and how to minimize exposure to high-risk pathogens. The physician provides baseline and ongoing assessments of the patient, as well as antiretroviral therapy and preventive treatment in the form of vaccine and prophylaxis against opportunistic infection. One goal is to achieve maximum reduction of the viral load and to maintain this reduction for as long as possible to slow disease progression. Another goal is to restore the CD4 count back to a normal range. The physician should use HAART in a manner that will achieve viral suppression and immune reconstitution while at the same time

preventing emergence of resistance and limiting drug toxicity. Long-term goals are to delay disease progression, prolong life, and improve quality of life.

Antiretroviral Therapy

The antiretroviral agents that are currently available for the management of HIV/AIDS. The first antiretroviral agent used in the AIDS epidemic was zidovudine (AZT); however, viral resistance emerged often during the initial 6 to 12 months of therapy. Therapy now consists of a combination of regimens, including at least one potent agent that will minimize viral replication, thus reducing mutation rates that can lead to resistance. The development of more potent antiretroviral agents in the mid-1990s and the recognition that combination therapy was more effective than monotherapy have led to improved clinical benefits.These regimens have represented a major advance in the treatment of HIV-infected patients who have access to medical care and are capable of taking these drugs and willing to do so.Taking these drugs, given their toxicities, costs, and inconvenience, presents a major challenge for patients.

The antiretroviral agents are used to restore immune dysfunction by inhibiting viral replication. Four different types of agents that are now available—protease inhibitors (PIs), nucleoside reverse transcriptase inhibitors (NRTIs), non-nucleoside reverse transcriptase inhibitors (NNRTIs), and nucleotides and the fusion inhibitors (enfuvirtide is the fi rst member of this class to become available)—have just received approval from the U.S. Food and Drug Administration (FDA) for clinical use. These agents are usually used in combinations known as HAART. Patients who are on these medications must be closely monitored for effectiveness and drug toxicity.The drug regimen that is initiated should be potent enough to suppress the viral load to below the level of assay detection for a prolonged period. This requires at least two drugs, including at least one that is a PI, an NNRTI, or abacavir. Usually, two NRTIs are used. Currently, regimens consisting of efavirenz or nevirapine or lopinavir-ritonavir plus lamivudine plus either zidovudine or tenofovir are popular. The first drug that allows AIDS to be treated with one pill a day has just recently won federal approval—a development that government officials said would both simplify and improve treatment of the disease. The drug, called *Atripla,* is a

combination of three once-a-day drugs that are already on the market—Sustiva (Bristol-Myers Squibb), and Viread and Emtriva (Gilead Sciences). Only a decade ago, when cocktails of AIDS drugs began to be used, patients sometimes had to take two dozen or more pills a day. All patients with acute antiretroviral syndrome and patients within 6 months of seroconversion should be offered antiretroviral treatment.4,20 Also, all patients with symptoms ascribed to HIV infection and symptomatic patients with CD4+ T-cell count less than 350/mm3 are offered treatment.4,20 Another group of patients who should be offered treatment with antiretroviral agents are those with plasma HIV RNA levels greater than 55,000 copies/mL.4,20 Treatment is generally initiated for asymptomatic patients who have a rapid drop in CD4+ T-cell count or high viral loads (730 to 55,000 copies/mL). Asymptomatic patients with stable CD4+ T-cell counts and low viral loads are generally followed without treatment. Antiretroviral therapy is strongly recommended for patients with CD4+ T-cell counts of fewer than 200/ mm3 and for those with AIDS.4,20

Vaccination. Immunization with killed microbial products or recombinant products is considered safe in HIVinfected adults. The HIV-infected adult should receive the following products before his/her CD4+ T-cell count drops to below 200/mm3: 23-valent polysaccharide pneumococcal vaccination, hepatitis A vaccine (all patients negative for HAV antibody), hepatitis B vaccine (all completed phase 1 and 2 trials in humans, and two vaccine candidates (recombinant gp120 proteins) have completed phase 3 effi cacy trials. The following approaches are under consideration for the development of an HIV-1 vaccine: whole virus vaccines, envelope protein vaccines, synthetic peptide vaccines, internal core protein vaccines, live vector vaccines, nucleic acid vaccines, and live-attenuated virus vaccines.

DENTAL MANAGEMENT

Health history, head and neck examination, intraoral soft tissue examination, and complete periodontal and dental examinations should be performed on all new patients. History and clinical fi ndings may indicate that the patient has HIV/AIDS. Patients with HIV/AIDS and those at high risk for it realize their lack of true privacy on questionnaires; in addition, an AIDS phobia or homophobia may exist among members of the dental staff. Thus, answers to certain questions may be less than honest. As a result, a patient's history

should be obtained whenever possible via caring, understanding, verbal communication, with sharing of knowledge and facts through honesty, avoidance of direct personal questions, and openness with the patient. Because of the sensitive social and legal issues associated with AIDS, direct questions are not recommended for inclusion in the health history questionnaire, but certain questions suggestive of a high risk for AIDS or related conditions may be included on the health questionnaire, and verbal follow-up is required. Patients who, on the basis of history or clinical fi ndings, are found to be at high risk for AIDS or related conditions should be referred for HIV testing, medical evaluation, other appropriate diagnostic procedures, and psychosocial intervention. The dentist should not undertake diagnostic laboratory screening but rather should refer the patient to an appropriate medical facility. Thi should follow a discussion concerning the clinical findings and the possibility that AIDS or a related condition is present. At this time, sexual preference, IV drug use, and so forth may be discussed and often are mentioned by the patient. The patient should be strongly encouraged to seek diagnostic and supportive medical services.

Patients at high risk for AIDS and those in whom AIDS or HIV has been diagnosed should be treated identically to any other patient, that is, with standard precautions.

Several guidelines have emerged regarding the rights of dentists and patients with AIDS, including the following:

• Dental treatment may not be withheld because the patient refuses to undergo testing for HIV exposure. The dentist should assume that this type of patient is a potential carrier of HIV and should treat the person using standard precautions, just as the dentist would for any other patient.

• A patient with AIDS who needs emergency dental treatment may not be refused care because the dentist does not want to treat patients with AIDS.

• No medical or scientific reason exists to justify why patients with AIDS who seek routine dental care may be declined treatment by the dentist, regardless of the dentist's reason. However, if the dentist and the patient agree, the dentist may refer this patient to someone who would be more willing to provide treatment.

• A patient who has been under the care of a dentist and then develops AIDS or a related condition must be treated by that dentist or by a referral that is

satisfactory for and agreed to by the patient. The CDC and the American Dental Association recommend that infected dentists should inform the patient of his or her HIV status and should receive consent or refrain from performing invasive procedures.

Treatment Planning Considerations

A major consideration in dental treatment of the patient with HIV/AIDS involves determining the current CD4+ lymphocyte count and level of immunosuppression of the patient. Other points of emphasis in dental treatment planning include the level of viral load, which may be related to susceptibility to opportunistic infections and rate of progression of AIDS. The dentist should be knowledgeable about the presence and status of opportunistic infections and the medications that the patient may be taking for therapy or prophylaxis for these opportunistic infections. Patients who have been exposed to the AIDS virus and are HIV seropositive but asymptomatic may receive all indicated dental treatment. Generally,this is true for patients with a CD4+ cell count of more than 400. Patients who are symptomatic for the early stages of AIDS (i.e., CD4+ cell count lower than 200) have increased susceptibility to opportunistic infection and may be effectively medicated with prophylactic drugs.

The patient with AIDS can receive almost any dental care needed and desired once the possibility of significant immunosuppression, neutropenia, or thrombocytopenia has been ruled out. Complex treatment plans should not be undertaken before an honest and open discussion about the long-term prognosis of the patient's medical condition has occurred.

Dental treatment of the HIV-infected patient without symptoms is no different from that provided for any other patient in the practice. Standard precautions must be used for *all* patients. Any oral lesions found should be diagnosed, then managed by appropriate local and systemic treatment or referred for diagnosis and treatment. Patients with lesions suggestive of HIV infection must be evaluated for possible HIV. Patients may be medicated with drugs that are prophylactic for *P carinii* pneumonia, candidiasis, herpes simplex virus (HSV) or CMV, or other opportunistic disease, and these medications must be carefully considered in dental treatment planning. Care in prescribing other medications must be exercised with these, or any, medications after which the patient may experience adverse drug effects,

including allergic reactions, toxic drug reactions, hepatotoxicity, immunosuppression, anemia, serious drug interactions, and other potential problems. Most often, consultation with the patient's physician is mandatory.Patients with severe thrombocytopenia may require special measures (platelet replacement) before any surgical procedures (including scaling and curettage) are performed. Patients with advanced immunosuppression and neutropenia may require prophylaxis for invasive procedures (CD4 cell count below 200/mm3 and/or neutrophil count lower than 500/mm3). Acetaminophen should be used with caution in patients treated with zidovudine (Retrovir) because studies have suggested that granulocytopenia and anemia, associated with zidovudine, may be intensifi ed; also, aspirin should not be given to patients with thrombocytopenia. Antacids, phenytoin, cimetidine, and rifampin should not be given to patients who are being treated with ketoconazole because of the possibility of altered absorption and metabolism. Medical consultation is necessary for symptomatic HIV-infected patients before surgical procedures are performed. Current platelet count and white blood cell count should be available. Patients with abnormal test results may require special management. All these matters must be discussed in detail with the patient's physician.

Any source of oral or dental infection should be eliminated in HIV-infected patients, who often require more frequent recall appointments for maintenance of periodontal health. Daily use of chlorhexidine mouth rinse may be helpful. In patients with periodontal disease whose general health status is not clear, periodontal scaling for several teeth can be provided to allow assessment of tissue response and bleeding. If no problems are noted, the rest of the mouth can be treated. Root canal therapy may carry a slightly increased risk for postoperative infection in patients with advanced HIV disease. Infection can be treated through local and systemic measures. Individuals with severe symptoms of AIDS may be best managed by treatment of their more urgent dental needs to prevent pain and infection, with deferment of extensive restorative procedures. The main objectives of care are to prevent infection and to keep the patient free of dental or oral pain. When one is planning invasive dental procedures, attention must be paid to the prevention of infection and excessive bleeding in patients with

severe immunosuppression, neutropenia, and thrombocytopenia. This may involve the use of prophylactic antibiotics in patients with neutrophil count lower than 500 cells/mm3. White blood cell and differential counts, as well as a platelet count, should be ordered before any surgical procedure is undertaken. If significant thrombocytopenia occurs, platelet replacement may be needed. If severe neutropenia is present, antibiotic prophylaxis may be necessary. Medical consultation should precede any dental treatment.

Occupational Exposure to HIV Postexposure antiretroviral prophylaxis is recommended in cases of high-risk exposure to HIV-infected blood. High-risk exposure includes the following: high level of viremia in the source patient, exposure to large volume of infected fl uid, deep penetrating injury with sharp device covered with visible blood from the infected patient, and needlestick injury during injection of the infected patient. Postexposure prophylaxis should consist of two NRTIs with or without a potent PI to minimize toxicity and afford high tolerability. If drug resistance in the source patient is identifi ed, an alternative regimen should be considered. Prophylaxis should be initiated within a few hours of exposure, if possible. This should be continued for 4 weeks. The exposed dental health care worker should be followed with antibody testing for HIV infection at baseline, 6 weeks, 12 weeks, and 6 months. If the exposed dental health care worker is pregnant, unknown but possible risks of postexposure prophylaxis to the fetus versus the risk of infection should be discussed.

Oral Complications and Manifestations

Clinical fi ndings that may suggest a high risk for AIDS or related conditions include candidiasis of the oral mucosa, bluish purple or red lesion or lesions that upon biopsy are identifi ed as Kaposi’s sarcoma, hairy leukoplakia of the lateral borders of the tongue, and other oral lesions associated with HIV infection, such as HSV, herpes zoster, recurrent aphthous ulcerations, linear gingival erythema, necrotizing ulcerative periodontitis, and necrotizing stomatitis Other oral conditions noted to occur in association with HIV infection are oral warts, facial palsy, trigeminal neuropathy, salivary gland enlargement, xerostomia, and melanotic pigmentation. Candidiasis, hairy leukoplakia, specific forms of periodontal disease (i.e., linear gingival erythema and necrotizing ulcerative periodontitis), Kaposi’s sarcoma, and non-Hodgkin’s lymphoma are believed to be strongly associated with HIV

infection.Worldwide, candidiasis is the most common oral manifestation of HIV infection. Oral candidiasis diagnosed in HIV-infected patients with persistent generalized lymphadenopathy may be of predictive value for the subsequent development of AIDS. The appearance of pseudomembranous candidiasis in HIV-infected individuals has been shown to be a strong indicator for the progression of infection to AIDS. The erythematous form of candidiasis also indicates progression toward AIDS. This information might be helpful to dental clinicians in evaluating patients for the initial diagnosis of HIV/AIDS or in determining stage of infection and level of immunosuppression. However, the oral manifestations of candidiasis that occurred more recently may be masked by earlier use of prophylactic antifungal agents. The variant of Kaposi's sarcoma that is associated with AIDS has been called *epidemic.* Epidemic Kaposi's sarcoma most often is disseminated throughout the body and runs a fulminant clinical course, with a lower than 20% survival rate at 2 years if associated with opportunistic infection. The literature suggests that Epstein-Barr virus (EBV) is not associated with any other oral white lesions that must be differentiated from hairy leukoplakia. Thus, hairy leukoplakia may be caused by reactivation of EBV in the oral mucosa in association with HIV-induced immune deficiency. The diagnosis of hairy leukoplakia is based on the clinical appearance of the lesion, its lack of response to antifungal therapy, and histologic findings. The finding of hairy leukoplakia also has predictive value for the subsequent development of AIDS.

Lymphadenopathy at cervical and submandibular locations often is an early finding in patients infected with HIV. This condition is persistent and may be found in the absence of any current infection or medications known to cause lymph node enlargement. The nodes tend to be larger than 1 cm in diameter, and multiple sites of enlargement may be found. The dentist should perform head and neck and intraoral soft tissue examinations on all patients. White lesions in the mouth must be found and the patient managed in such a way that a diagnosis is established. This may involve cell study, culture, and biopsy by the dentist or referral to an oral surgeon. If red or purple lesions are found that cannot be explained by history (e.g., trauma, burn, chemical, physical) or proved by clinical observation (healing within 7

to 10 days), biopsy must be performed. Persistent lymphadenopathy must be investigated by referral for medical evaluation, diagnosis, and treatment.

BOX 19-1

Categorization of HIV Exposures

GROUP 1
HIV antibody positive—asymptomatic

GROUP 2
CD4 <400
Constitutional symptoms (i.e., fever, malaise, lymphadenopathy, diarrhea); opportunistic infections

GROUP 3
AIDS; CD4 <200
Kaposi's sarcoma, lymphoma, pneumonia, cervical carcinoma, etc.

HIV, Human immunodeficiency virus; AIDS, acquired immunodeficiency syndrome.

BOX 19-2

Signs and Symptoms of HIV Infection

INITIAL EXPOSURE OR INFECTION
(SEROCONVERSION SYNDROME)
- Flulike symptoms—fever, weakness, 10 to 14 days
- Asymptomatic stage
- Serologic evidence of infection
- No signs or symptoms

SYMPTOMATIC STAGE
- Serologic evidence of infection
- T4/T8 ratio reduced to about 1
- Persistent lymphadenopathy
- Oral candidiasis
- Constitutional symptoms—night sweats, diarrhea, weight loss, fever, malaise, weakness

ADVANCED SYMPTOMATIC STAGE
- Serologic evidence of infection
- T4/T8 ratio suppressed to less than 0.5
- HIV encephalopathy
- HIV wasting syndrome
- Major opportunistic infections
- Neoplasms—Kaposi's sarcoma, lymphoma, carcinoma of rectum

HIV, Human immunodeficiency syndrome.

TABLE 19-4
Common Immunosuppression-Related Diseases Based on T-Helper Cell Level*

T-Helper Cell Count	Disease
>400	Most patients have no signs of immunosuppression-associated disease
301 to 400	Bacterial skin infections = staphylococcal
201 to 300	Herpes zoster Candidiasis Tinea pedis Oral hairy leukoplakia
101 to 200	Tuberculosis *Pneumocystis carinii* pneumonia Histoplasmosis Coccidioidomycosis Cryptococcal meningitis Toxoplasmosis Herpes simplex Cryptosporidiosis Kaposi's sarcoma
0 to 100	Wasting syndrome Cytomegalovirus Lymphoma *Mycobacterium avium* complex

*The T-helper cell range is the common range for onset of the disease. The disease could first appear at a higher or lower T-cell level.[53]

BOX 19-3

Dental Management of the Patient With AIDS or a Related Condition: General Procedures

KEY POINTS

- Consult whenever possible with the patient's physician to establish current status; if severe thrombocytopenia is present (<50,000), platelet replacement may be needed before surgical procedures are performed.
- Determine whether prophylactic antibiotics are needed to protect patients with severe immune neutropenia (<500 cells/mm^3) from postoperative infection.
- Render only more immediately needed treatment for patients with advanced AIDS.
- In most cases, provide dental procedures in accordance with the patient's wants and needs.
- Inform all personnel working with patients with AIDS of the relative risks involved and how they can be minimized.

AIDS, Acquired immunodeficiency syndrome.

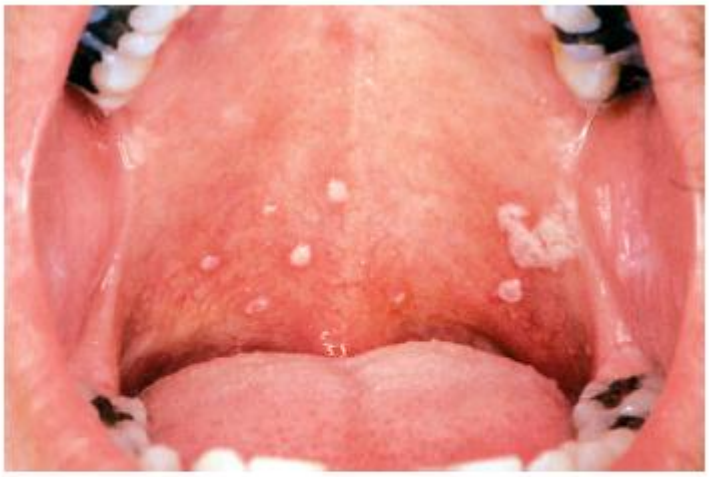

Figure 19-5. White lesion on the palate in a patient with AIDS. The lesion could be removed with a tongue blade. The underlying mucosa was erythematous. Clinical and cytologic findings supported the diagnosis of pseudomembranous candidiasis. (From Silverman S Jr. Color Atlas of Oral Manifestations of AIDS, 2nd ed. St. Louis, Mosby, 1996.)

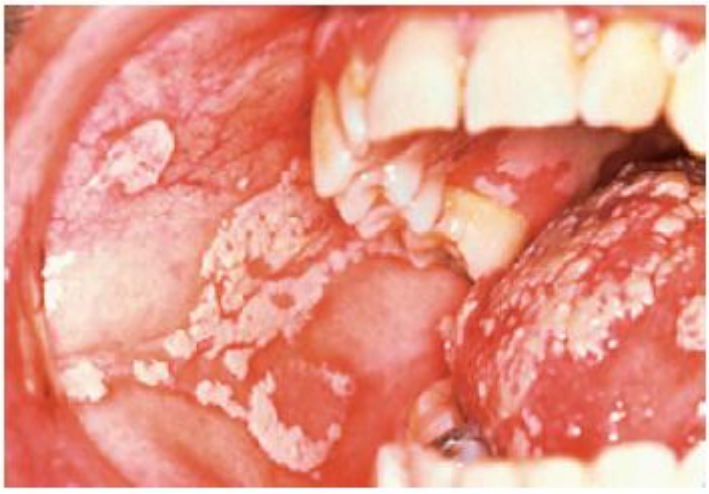

Figure 19-6. Note the white lesions on the oral mucosa. The diagnosis of pseudomembranous candidiasis was established. (Courtesy Eric Haus, Chicago,)

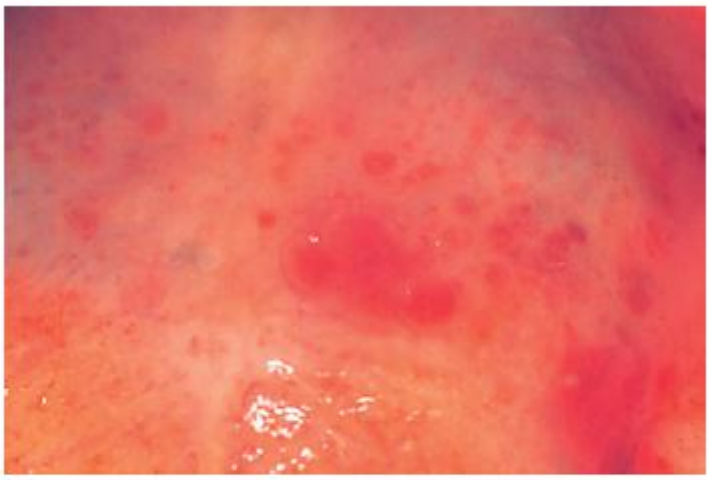

Figure 19-7. Erythematous palatal lesion in an HIV antibody–positive patient. Smears taken from the lesion showed hyphae and spores consistent with *Candida*. The lesion healed after a 2-week course of antifungal medications. A diagnosis of erythematous candidiasis was made on the basis of clinical laboratory findings. (Courtesy Eric Haus, Chicago.)

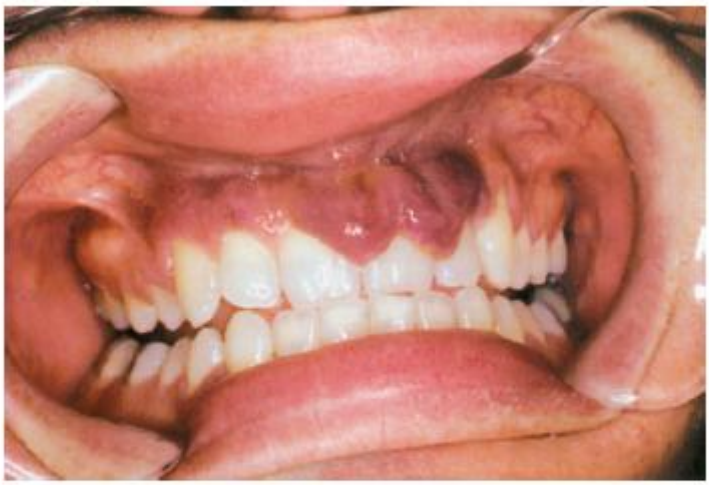

Figure 19-12. Kaposi's sarcoma of the gingiva. (From Silverman S Jr. Color Atlas of Oral Manifestations of AIDS, 2nd ed. St. Louis, Mosby, 1996.)

Suggestive Reading

James W Little, Craig S Miller, Nelson L Rhodus. Dental management of medically compromised patient, 9th edition, Elsevier, 2018