Viral Diseases: Oral Involvement and Complications

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Faculty Disclosure

Contributing faculty, Mark J. Szarejko, DDS, FAGD, has disclosed no relevant financial relationship with any product manufacturer or service provider mentioned.

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Director Disclosure

The director has disclosed no relevant financial relationship with any product manufacturer or service provider mentioned.

Audience

This course is designed for all dental professionals involved in evaluating and maintaining patients' oral health.

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Course Objective

The purpose of this course is to provide dental professionals with a review of several viral organisms and the effect they have directly and indirectly upon dental treatment, oral health, and systemic health.

Learning Objectives

Upon completion of this course, you should be able to:

1. Describe the lifecycle of viruses.
2. Identify hepatitis viral infections and their effects on oral health.
3. Differentiate various opportunistic viral infections in patients with HIV/AIDS, particularly those related to herpes viruses.
4. Outline characteristics of oral human papillomavirus infection.
5. Compare oral signs of measles and mumps.
6. Discuss the impact of the coxsackievirus on oral and systemic health.

Sections marked with this symbol include evidence-based practice recommendations. The level of evidence and/or strength of recommendation, as provided by the evidence-based source, are also included so you may determine the validity or relevance of the information. These sections may be used in conjunction with the course material for better application to your daily practice.

INTRODUCTION

Organisms within the microbial community can achieve a degree of pathogenicity that can have a wide range of ramifications. Local and systemic infections of bacterial, fungal, and viral origin can have effects ranging from mild discomfort to serious morbidity and even death. The immunocompetence of the host and the virulence of the pathogenic microbial species do much to establish the degree of involvement and the success of antimicrobial medications. Microbial resistance to some of these medications is a mounting public health problem. Genetic mutations among these pathogens pose another obstacle to successful pharmacotherapeutic intervention.

Viral disease can be particularly difficult to treat, and many viral diseases are incurable. In many cases, patients with hepatitis C (HCV) never eliminate the virus and develop a chronic carrier state. The herpes simplex virus type 1 (HSV-1) and human herpesvirus 3 (HHV-3) remain dormant in nerve ganglia and manifest as initial or recurrent herpetic lesions and shingles, respectively. Antiviral medications can reduce the severity of outbreaks associated with these viruses but can never fully eradicate them. Opportunistic viral oral infections among patients with human immunodeficiency virus/acquired immune deficiency syndrome (HIV/AIDS), who have had transplants, or who take immunosuppressive medications can be fatal. The many subtypes of the human papillomavirus (HPV) can cause oral papillomas and have come under increased scrutiny as potential risk factors in the development of oral cancer. Despite immunizations, measles and mumps still have periodic outbreaks and can have dire consequences in the young, old, and immunocompromised. Subtypes of the coxsackievirus can cause painful oral and cutaneous lesions. This course will highlight the most common oral manifestations of several viral organisms that have systemic involvement and those that have an affinity for the oral and maxillofacial complex. Their impact and interrelations with oral and systemic health will also be discussed.

VIRAL ORGANISMS IN THE SPECTRUM OF MICROBIAL LIFE

The primary difference between viruses and other infectious agents is in the method of reproduction. Viruses do not divide as a means of propagation; they exist in two separate phases. Virions are virus particles that are complete, before contact with a host cell; at this phase, virions are infective. This particle consists of genetic information surrounded by a protein capsule. Upon contact with receptor sites of a suitable host cell, the virion injects its RNA or DNA into the cell, at which point it becomes a virus. The host cell's usual biosynthetic processes then cease and are directed to the replication of the virus [1]. New virions exit the cell and continue this pattern upon contact with another host cell. Transmission of viral diseases can occur via respiratory droplets, through direct contact of mucous membranes, and by the exchange of bodily fluids, such as blood and semen.

Viruses do not produce soluble toxins but cause damage by subverting the appropriate function of the host cell and by their own replication. The relationship between the CD4+ lymphocytes and HIV and that of the hepatitis virus subtypes and the hepatocytes are classic examples of viral damage at the cellular level that causes grave systemic consequences. Conventional mechanisms of antimicrobial therapy, such as the lysis of the cell walls of gram-positive bacteria and altered cell membrane permeability of fungal organisms by antifungal medications, do not apply to viruses. Antiviral medications can modify viral illnesses, but none are curative. Viral organisms can produce a vast array of problems, from the inconvenience associated with the common cold (caused by members of the adenovirus family) to the spectrum of devastating opportunistic infections seen in patients with HIV/AIDS. The problems of dental caries, periodontal disease, and odontogenic infections are of bacterial origin, while the fungal organism *Candida albicans* can cause oral fungal infections. Viruses can affect the oral and maxillofacial complex directly, such as HSV-1, or indirectly, as seen in immunosuppression

among patients with HIV. While viral organisms are not responsible for common dental problems, these pathogens can create other systemic and oral problems and can be life-altering.

HEPATITIS VIRUSES

Hepatitis is essentially inflammation of the hepatocytes, the cells of the liver. Infectious hepatitis is associated with many different causes, including viruses, secondary syphilis, and extrapulmonary tuberculosis [2]. Noninfectious hepatitis can result from the prolonged use of medications prescribed for chronic illnesses and illicit drug and alcohol abuse. Among the infectious forms of hepatitis, acute viral hepatitis is the most common. Six hepatitis viruses have been identified to date: types A, B, C, D, E, and G, each of which belongs to a different viral family and has unique pathophysiologic features. Of these types, hepatitis G is less well understood and is not believed to be a significant source of disease in humans.

Hepatitis viruses replicate within the hepatocytes with a resultant cellular ballooning degeneration and necrosis. The long-term replacement of the hepatocytes with connective tissue is called cirrhosis. Symptoms of acute hepatitis can vary considerably and will depend upon the viral type and host response. Right upper-quadrant pain, nausea, vomiting, joint pain, and loss of appetite are among the possible presenting symptoms. Jaundice is relatively common among these patients as a result of accumulation of bilirubin, the degradation product of hemoglobin, in the plasma. The actual frequency of this sign varies, occurring in approximately 70% of hepatitis A infections, 30% of hepatitis B infections, and 25% in hepatitis C and E infections [3]. When liver disease is present, the conjugation of bilirubin with glucuronic acid and excretion into the intestine is impaired. The normal plasma concentration of bilirubin is less than 1 mg/100 mL; jaundice is defined as a concentration of 2.3 mg/100 mL or greater [4]. Complications from viral hepatitis can include cirrhosis, liver cancer, and fulminant hepatitis. Acute and rapid destruction of the hepatocytes and a resultant high mortality rate is characteristic of fulminant hepatitis. However distinctive these symptoms and complications may be, many patients infected with the hepatitis viruses have no symptoms. Among those with hepatitis A, 10% are asymptomatic, as are approximately 60% to 70% of patients with hepatitis B and 70% to 90% of patients with HCV [5].

HEPATITIS A VIRUS

Hepatitis A is the most common form of hepatitis in the United States [6]. It is a single-stranded RNA virus, a picornavirus, that is transmitted primarily by the fecal-oral route. It can be spread by personal contact, by eating contaminated food, or by drinking contaminated water. Bloodborne transmission is rare but possible in the early stages of the disease, when blood levels of the virus are the highest. There is no chronic stage associated with hepatitis A, and recovery from the disease confers immunity against future infections. There are no specific oral lesions associated with this disease, although the oral mucosa may appear yellow to yellow-brown in patients in whom jaundice is present. Most patients recover within a few weeks, although prolonged symptoms and relapse can occur with a few patients.

Dental treatment should be deferred until the patient recovers. Treatment includes adequate nutrition and hydration and the avoidance of potentially hepatotoxic substances, such as alcohol and acetaminophen. After a complete recovery is made from hepatitis A, there are no specific restrictions for dental treatment. Infection can be prevented by the use of established vaccines; two single-antigen vaccines and one combination (both hepatitis A and hepatitis B) vaccine are currently available for hepatitis A [7].

HEPATITIS B VIRUS

Hepatitis B is a DNA virus and a member of the Hepadnaviridae family. The hepatitis B virus can survive outside the body and remain infective for seven days or longer on contaminated surfaces, medical devices, and instruments that have not been properly processed and sterilized [8]. A case of dental patient-to-patient transmission of hepatitis B in New Mexico was documented in 2001 [9; 10]. In this case, a female patient, 60 years of age with no risk factors for hepatitis B but without the vaccination for this disease, had seven teeth extracted at an oral surgeon's office. The patient developed hepatitis B infection within four months, from which she subsequently recovered. Investigators from the Centers for Disease Control and Prevention (CDC) observed that none of the 15 employees of the office were infected; 14 had been vaccinated. Further observation of the infection control protocol and instrument processing and sterilizing revealed no deviations from accepted infection control practices. It was discovered that another patient who did not disclose being a hepatitis B carrier had a high viral load at the time of her own oral surgery three hours earlier at the same office operatory. The investigators speculated that the patient-to-patient transmission likely originated from a contaminated environmental surface [9].

This case demonstrates that it is critical for all healthcare professionals to avoid complacency about the potential transmission of this virus. The Occupational Safety and Health Administration (OSHA) requires the employers of healthcare workers to offer the hepatitis B virus vaccination at no cost to employees, and most workers do obtain the vaccination series [11]. However, this only affords protection to the individual worker from an infected patient. Healthcare professionals should maintain exacting standards of infection control to prevent a contaminated operatory surface from becoming a means of transmission of this disease. Beyond this isolated case, the most common methods of transmission of hepatitis B virus are the exchange of blood or other bodily fluids, sharing needles contaminated with the virus, and accidental exposure through contaminated needlesticks or other sharp instruments [12]. However, vaccination is an effective means of preventing infection. There are three single-antigen vaccines for active immunization against hepatitis B virus: Engerix-B, Recombivax HB, and Heplisav-B. However, several combination vaccines also contain a hepatitis B vaccine. For example, Twinrix is a combined vaccine for the prevention of both hepatitis A and B infections, and Pediarix is a combined hepatitis B, diphtheria, tetanus, acellular pertussis (DTaP), and inactivated poliovirus (IPV) vaccine for children between 6 weeks and 7 years of age [12]. Among adults who are vaccinated, 90% acquire immunity from these vaccines [13]. Testing that demonstrates the presence of the antibody to the surface antigen of the hepatitis B virus reflects recovery and immunity from this disease and is the only clinical parameter to do so. A lack of symptoms, such as jaundice or right upper-quadrant pain, are not absolute indicators of recovery or immunity from any form of hepatitis.

Approximately 95% of patients who acquire hepatitis B eliminate the infection and develop antibodies against the virus; the remainder cannot clear the virus and develop chronic hepatitis B infection [12]. Medications such as interferon alfa-2b, adefovir, and lamivudine are the most common medications used to treat patients with chronic hepatitis B infection [12]. Each has the potential to have deleterious side effects and can also have adverse reactions when combined with other medications.

Interferon alfa-2b can result in many multisystemic side effects [14]. Potential oral effects include xerostomia, gingivitis, alteration in the sensation of taste, and paresthesia. A greater concern exists for the potential hematologic side effects. Thrombocytopenia, a condition in which the circulating platelet population is decreased, is an obvious concern for patients who require oral or periodontal surgery. Myelosuppression, specifically neutropenia, can reduce the number of neutrophils available. The potential for opportunistic oral infections, postsurgical infections, periodontal disease, and odontogenic infections increase. This medication can also increase the anticoagulant effect of warfarin if taken concurrently. Dental treatment for those who are receiving interferon alfa-2b should be limited to procedures directed toward the elimination of acute pain or odontogenic infections. The patient's physician should be consulted before any such procedure is initiated. Appropriate laboratory tests, such as a complete blood count (CBC) with differential (to determine if platelets and leukocytes are within a safe range), should be completed prior to dental treatment. Additional tests to determine bleeding time may be required.

The antiretroviral agent adefovir has far fewer systemic effects compared to interferon alfa-2b, but when it is combined with ibuprofen the risk of nephrotoxicity is increased. Ibuprofen can also increase the bioavailability of adefovir and increase the toxic potential of the medication. Dental professionals should consult with the patient's physician to identify an alternative analgesic that is efficacious and safe for patients who take adefovir.

Lamivudine is another antiretroviral agent that has been used to treat chronic hepatitis B infection. It has fewer adverse systemic ramifications compared to interferon alfa-2b, but more than adefovir. As with interferon alpha-2b, the potential for neutropenia is present and should be considered for all patients taking lamivudine [14]. Lamivudine is no longer recommended due to a high rate of resistance with long-term use [24]. Other antivirals, specifically tenofovir AF, tenofovir DF, and entecavir, are currently recommended as first-line therapy for hepatitis B infection [82].

Hepatic disease of any origin can cause problems with hemostasis and the metabolism of drugs. Both issues are a concern for any dental patient with chronic hepatitis B virus infection, especially those for whom surgical procedures are necessary to eliminate pain and odontogenic infections. The liver is the source of the production of all protein coagulation factors, and any form of chronic viral hepatitis infection can compromise the patient's ability to achieve hemostasis. If a medication is administered, such as interferon alfa 2b, that causes thrombocytopenia with a consequent decrease in platelet production, the ability to achieve postsurgical hemostasis can be further compromised. Laboratory tests such as the prothrombin time (PT) and the partial thromboplastin time (PTT) can be used to assess the adequacy of the coagulation factors for the intrinsic and extrinsic pathways. When these tests are combined with a CBC, to determine the platelet population, and the results from instruments such as a platelet function analyzer (PFA -100), to discern between adequate and inadequate platelet function, the clinician can be provided with a presurgical assessment of the patient's ability to achieve hemostasis [15].

Another major function of the liver is the metabolism of medications such as local anesthetics, analgesics, antibiotics, and sedatives used in dental treatment. The metabolism of the amide local anesthetics, such as lidocaine, mepivacaine, etidocaine, and bupivacaine, occurs primarily in the liver [16].

Articaine has both amide and ester linkage, and most of its metabolism occurs in the plasma via plasma carboxyesterase. Hepatic metabolism of articaine is minimal. Analgesics such as aspirin, ibuprofen, and acetaminophen, alone or combined with narcotics such as codeine, hydrocodone, or oxycodone, also rely on hepatic metabolism. Similarly, antibiotics such as amoxicillin, ampicillin, clindamycin, and tetracycline utilize the liver for primary metabolism [3]. Before any of these medications are prescribed or administered to any patient with active hepatitis or chronic hepatitis infection, the patient's physician should be consulted. The current state of hepatic function must be assessed to determine the ability of the liver to metabolize the medications adequately. Dosages and the frequency of administration of any of the medications will require adjustments that correspond to the degree of the compromise of hepatic function.

HEPATITIS D VIRUS

Hepatitis D virus, or hepatitis delta virus, is an incomplete RNA virus that was first identified in 1977. This pathogen requires the presence of the hepatitis B virus for its replication [17]. The outer shell of the intact hepatitis B virus, known as the surface antigen, is a component of an envelope protein that surrounds the viral RNA of hepatitis D virus and protects it until a target cell, such as a hepatocyte, can be found. After the viral RNA is injected into a host cell, the helper function of the hepatitis B virus is not required.

The means of transmission for both hepatitis B and D are similar. Immunity that is acquired from the vaccination series for the hepatitis B virus also protects against hepatitis D virus infection. This disease can occur as a simultaneous coinfection of active hepatitis B virus infection or as a superinfection in a patient with chronic hepatitis B [17]. The precautions discussed regarding the metabolism of medications used in dental treatment and postsurgical hemostasis apply to both viruses. There are no specific oral lesions associated with hepatitis D infection; however, the oral mucosa can appear pale or yellow in patients who experience jaundice.

HEPATITIS C VIRUS

HCV is an RNA virus originating from the Flaviviridae family. At least seven distinct HCV genotypes (genotypes 1–7) have been identified along with more than 67 subtypes. Genotype 1 is the most common in the United States [18].

HCV is less efficiently transmitted than hepatitis B, but more easily transmitted than HIV. Transmission occurs from inoculation of infected blood or blood products and bodily fluids. Sharing needles among infected drug abusers and percutaneous exposure from contaminated needles or dental/medical instruments are other means of transmission. Most patients infected with HCV remain asymptomatic for many years [18]. As opposed to hepatitis B, the majority of patients do not clear HCV and develop immunity. Approximately 75% to 85% of patients develop chronic HCV infection and about 10% to 20% develop cirrhosis [18; 19; 20]. This disease is the most common cause of end-stage liver disease and liver transplantation in the United States [18; 21].

Genotype information is helpful in defining the epidemiology of HCV and in making recommendations regarding treatment [18]. Pharmacologic treatment is indicated for patients with detectable blood titers of HCV, elevated liver enzymes, such as aminotransferase, and histologic liver biopsy findings that reflect a progression of this disease. Until 2013, the mainstay pharmacotherapeutic regimen was a combination of peginterferon alpha-2a and ribavirin. In 2013, the FDA approved a new direct acting antiviral drug, sofosbuvir, to treat chronic HCV infection [18; 23]. Sofosbuvir is a nucleotide analogue inhibitor of the HCV virus and has proven efficacy when used as a component of combination

therapy [18; 23].

In the past decade, several new drugs have been approved for the treatment of HCV infection. In 2014, the FDA approved the combination ledipasvir/sofosbuvir to treat chronic HCV infection [23; 24; 25]. Ledipasvir/sofosbuvir 90/400 mg once daily is indicated for chronic HCV infection genotypes 1, 4, 5, or 6.

In 2016, the FDA approved the direct-acting antiviral combinations elbasvir/grazoprevir and sofosbuvir/velpatasvir [23; 24; 25]. Elbasvir/grazoprevir one tablet (50/100 mg) once daily is indicated for the treatment of chronic HCV infection, genotypes 1 or 4. Sofosbuvir/velpatasvir one tablet (400/100 mg) daily for twelve weeks is indicated for treatment of chronic HCV infection, genotypes 1, 2, 3, 4, 5, or 6.

In 2017, the FDA approved the direct-acting antiviral combinations sofosbuvir/velpatasvir/voxilaprevir and glecaprevir/pibrentasvir. Sofosbuvir/velpatasvir/voxilaprevir (400/100/100 mg) is indicated once daily for 12 weeks for treatment of chronic HCV infection with genotypes 1, 2, 3, 4, 5, or 6 [23; 24]. Glecaprevir/pibrentasvir (100/40 mg) is indicated three times daily for eight weeks for genotypes 1, 2, 3, 4, 5, or 6 [23; 24].

Ribavirin is an antiviral medication that inhibits the replication of RNA and DNA viruses and is associated with various adverse systemic effects, including those involving the hematologic system. As with medications used to treat other hepatitis virus infections, the resultant decrease in the leukocyte population and absolute neutrophil count can facilitate the development of odontogenic infections, opportunistic oral infections, and postsurgical infections, and thrombocytopenia can reduce the capacity for adequate postsurgical hemostasis. Ribavirin is usually compatible with the medications most commonly used in the scope of dental treatment.

Peginterferon alpha-2a can cause even more adverse systemic effects compared to ribavirin. Shared problems include the decrease in the leukocyte population, especially the neutrophils, and thrombocytopenia [26]. No dental treatment should be started when patients are on this dual therapy unless the appropriate hematologic laboratory values and the current status of the patient's viral load allow for the patient's physician to grant approval.

Drugs approved since 2013, and subsequently discontinued, include simeprevir, ombitasvir/paritaprevir/ritonavir, and daclatasvir [23; 24].

There is no current vaccination against HCV. All healthcare professionals should adhere to standard precautions for infection control to minimize the risk of contracting this disease from percutaneous exposure or the accidental inoculation of unprotected body surfaces from infected patients' blood [18]. No specific oral lesions are characteristic of patients with HCV infection.

The U.S. Preventive Services Task Force recommends screening for hepatitis C virus infection in all adults 18 to 79 years of age.

(https://jamanetwork.com/journals/jama/fullarticle/2762186. Last accessed May 29, 2026.)

Level of Evidence/Strength of Recommendation: B (There is high certainty that the net benefit is moderate or there is moderate certainty that the net benefit is moderate to substantial.)

HEPATITIS E VIRUS

The RNA virus hepatitis E is from the Caliciviridae viral family. It is transmitted primarily by the fecal-oral route and by consuming contaminated foods or water. It has a low frequency of occurrence in the United States, appearing most often in travelers who have visited countries in which problems with contaminated water supplies and food sources exist. Symptoms of this disease include abdominal pain, dark urine, jaundice, joint pain, nausea, and vomiting. In pregnant women, hepatitis E infection is associated with a higher morbidity and even death [27]. No uniquely identifying oral lesions are present. The oral mucosa may appear pale or yellow if jaundice occurs. Dental treatment should be deferred until the infection has resolved.

OPPORTUNISTIC VIRAL INFECTIONS AND CONDITIONS IN PATIENTS WITH HIV/AIDS

Few diseases in the course of history have had such impact on the medical, political, philosophic, and sociologic aspects of life as has HIV/AIDS. At the end of 2022, the CDC estimated that 1,109,418 people were living with diagnosed HIV infection in the United States and 6 territories and freely associated states; 19,310 individuals with HIV/AIDS died in 2022 [28]. Death is usually caused by complications from one or more opportunistic infections that have proliferated due to the patient's decimated immune system. The annual rate of diagnoses essentially remained the same in 2018 and 2022, at 11.4 per 100,000 and 11.3 per 100,000, respectively [28].

This pathogen from the lentivirus family is now known to be of two subtypes (HIV-1 and HIV-2), with several strains associated with each. These variations contribute to the complexity of medical treatment for this disease. HIV can infect any cell in the body; however, it has a predilection for T-helper lymphocytes (CD4+ cells) and macrophages [29]. Numerous surface receptors on the CD4+ cells facilitate the attachment of the HIV surface glycoprotein (gp 120) and thus the ability of the HIV virion to inject its viral RNA into the host cell. An enzyme called reverse transcriptase that is produced by the HIV polymerase gene permits the incorporation of HIV RNA into the DNA of the host nucleus [30]. This is the manner by which HIV RNA is propagated and the host cell is destroyed. When the CD4+ count falls below 200 cells/mcL, the HIV infection has progressed to AIDS. At this point, bacterial, fungal, and viral pathogens that can normally be contained by a healthy immune system will manifest as opportunistic infections with high morbidity and mortality. This discussion will focus upon opportunistic viral infections as they relate to the oral and maxillofacial complex and their local, regional, and systemic manifestations.

HERPES VIRUS OPPORTUNISTIC INFECTIONS

In patients with AIDS, opportunistic herpes virus infections can have extensive regional and systemic involvement with a high morbidity and systemic complications. Many patients with HIV/AIDS have overlapping opportunistic bacterial or fungal infections, which further challenges the scarce reserves of an already impaired immune system.

Herpes Simplex Virus-1

HSV-1 is responsible for the most frequently occurring viral oral lesions. An initial form, primary herpetic gingivostomatitis, occurs after infection with the virus, most often in children, and is characterized by painful oral ulcerations, fever, and flu-like symptoms. HSV-1 is not eliminated with the healing of these lesions but migrates to a regional nerve ganglion, where it can remain dormant for an extended period of time. The oral reactivation of HSV-1 is called recurrent herpes labialis, and these lesions are more commonly referred to as "cold sores" or "fever blisters." The classic presentation of these lesions features small vesicles that coalesce to form larger vesicles and rupture to form shallow ulcers surrounded by an erythematous border. These lesions most frequently involve the skin adjacent to the lip with an extension onto the lip commonplace. The lesions of HSV-1 are local and circumscribed and will heal without scarring. Application of topical acyclovir to the infected area or the use of oral acyclovir, valacyclovir, or famciclovir for 5 to 10 days will accelerate the healing process [31].

EVIDENCE-BASED PRACTICE RECOMMENDATION

The American Academy of Oral Medicine recognizes that delaying care until an herpes simplex virus lesion is scabbed over or completely healed is prudent for minimizing recurrences and spread of the infection, and that the presence of an infectious herpes simplex lesion orally or periorally can be a reason for deferral of care.

(https://www.aaom.com/index.php?option=com_content&view=article&id=161:clinical-practice-statement--dental-care-for-the-patient-with-an-oral-herpetic-lesion&catid=24:clinical-practice-statement. Last accessed May 29, 2026.)

Level of Evidence: Expert Opinion/Consensus Statement

HSV-1 can have a radically different clinical course and prognosis in patients with AIDS. The duration of the HSV-1 lesions in patients with HIV persists beyond the usual two weeks seen in immunocompetent patients. Lesions that persist for more than four weeks in patients with HIV fulfill the diagnostic criteria established by the CDC for an AIDS-defining illness [31]. The depth of these lesions is superficial, with more epithelial involvement and less connective tissue involvement than in uninfected patients. Lesions can become extensive and involve large areas of the skin. The ulcerated surface areas can also serve as portals of entry for systemic bacterial and fungal coinfections. The pain from intra-oral herpetic lesions can compromise the ability to eat and swallow at a time when adequate nutrition is essential.

Topical acyclovir ointment used to treat recurrent herpes labialis in immunocompetent patients can be of little utility in treating the more virulent HSV-1 lesions in patients with HIV/AIDS. Systemic antiviral medications such as ganciclovir, valacyclovir, or famciclovir may be required [31; 32]. However, ganciclovir can cause bone marrow suppression and further compromise the immune system [32]. Ganciclovir should only be used when HSV-1 infections are of such magnitude that the therapeutic benefits outweigh the potential side effects. Dental treatment should be deferred until the lesions are resolved, as the pain associated with the larger recurrent HSV-1 lesions would preclude the ability of the patient to withstand manipulations of the oral tissues during dental procedures. Compression of oral tissues that are laden with viral particles can inoculate adjacent sites and extend the infection. Aerosols created when high-speed hand pieces or ultrasonic instruments are used can contain viral particles and may endanger the members of the dental team. A physician should be consulted if there is any doubt that an HSV-1 infection in a patient with HIV/AIDS remains unresolved despite the use of systemic antiviral medications.

Varicella Zoster Virus

There are two pathologic manifestations of the varicella zoster virus (VZV), also known as HHV-3. The initial infection, primarily seen in children, is chickenpox. Years and even decades later, the reactivation of VZV causes shingles [33]. It is this later pathologic expression that can have devastating effects for patients with HIV/AIDS.

VZV can lie dormant in the cranial nerve or dorsal root ganglia for decades until an event such as immunosuppression causes its advancement from the nerve ganglia to the peripheral nerve endings [33]. Involvement of the oral and maxillofacial complex is primarily seen in the facial nerve and among the ophthalmic, maxillary, and mandibular divisions of the trigeminal nerve.

Lesions associated with VZV infections are most often cutaneous but can be intra-oral. Unilateral involvement of a given nerve is a classic sign of shingles. The initial lesions feature coalescing vesicles that unite to form shallow ulcerations, which can take several weeks to heal in immunocompetent patients. Some patients develop postherpetic neuralgia, with severe pain in the areas in which VZV lesions occurred [33]. Patients may be prescribed narcotic and/or antidepressant medications to palliate the pain and symptoms that accompany postherpetic neuralgia. Dentists should refrain from prescribing these patients any further narcotics for pain associated with dental concerns to decrease the risk of overdose toxicity.

Patients with HIV/AIDS who develop shingles will have a prolonged duration and more aggressive manifestation. Intra-oral lesions occurring on the tongue and oral mucosa can be extremely painful and compromise the ability to masticate food properly and to swallow. Immunocompromised patients can also have more frequent recurrences of shingles compared to immunocompetent patients. Bacterial and fungal organisms can infiltrate the VZV lesions to cause complex secondary infections. Extension of the intra-oral lesions can involve destruction of the supporting alveolar bone and ultimately tooth loss. Patients who wear complete dentures or partial dentures may not be able to wear these prostheses when VZV lesions affect their supporting tissue base. Oral hygiene will be a challenge in areas where these viral lesions are present. Mouth rinses with alcohol can irritate and desiccate the oral tissues and should be avoided.

Treatment for shingles among patients with HIV/AIDS consists of the administration of systemic antiviral agents such as acyclovir, valacyclovir, and famciclovir. The dosage and frequency of administration of these medications can require adjustment in patients with compromised renal function. Unfortunately, the prognosis for patients with HIV/AIDS who develop shingles is poor.

Epstein-Barr Virus

The Epstein-Barr virus (or HHV-4) has a global distribution, and 95% of adults have antibodies against this virus [34]. Epstein-Barr virus can be found in the saliva of 40% adults at any time. After the initial infection resolves, an individual remains a carrier for life. The virus can cause disease in both immunocompetent and immunosuppressed individuals. In patients with HIV/AIDS, it can result in infectious mononucleosis, oral hairy leukoplakia, or Burkitt lymphoma [35].

Infectious Mononucleosis

This disease begins with an Epstein-Barr viral infection of epithelial cells in the oropharyngeal region with subsequent infection of the B lymphocytes that reside in the tonsillar crypts. The systemic circulation of these B lymphocytes stimulates a proliferation of a large number of the reactive T-lymphocytes [36]. The clinical presentation of infectious mononucleosis reflects the heightened reactive T-lymphocyte response and the cytokines they produce [3].

Patients are usually febrile and present with sore throat, fatigue, weight loss, inflammation, enlargement of the tonsils, and lymphadenopathy. Palatal petechiae and a pharyngeal exudate are seen in approximately one-third of these patients [37; 38]. Disease is spread mainly by oral secretions [38; 39].

Mononucleosis is usually a self-limiting disease. Treatment is palliative and is designed to decrease the associated symptoms. Acetaminophen and nonsteroidal anti-inflammatory drugs can decrease the pain and discomfort caused by mononucleosis [39]. Rinses with 2% viscous lidocaine can provide temporary relief from pharyngitis. This disease has higher morbidity and complications among those who are immunocompromised, especially if concurrent oral opportunistic infections are present to prolong the recovery. Rare oral complications from infectious mononucleosis include a cranial nerve deficit, a parotid mass with a subsequent facial nerve palsy, and lingual tonsillitis [40].

Oral Hairy Leukoplakia

Oral hairy leukoplakia is not unique to patients with HIV/AIDS but is the result of generalized immunosuppression. This condition can occur in patients who are taking immunosuppressive medications to decrease the risk of rejection after organ or bone marrow transplant. People who are immunocompetent have rare occurrences of this lesion [41]. Among patients with HIV, oral hairy leukoplakia can occur when the CD4+ cell count is 300 cells/mcL or less; its appearance reflects a progressive deterioration of the immune system and advancement toward AIDS [42].

Oral hairy leukoplakia lesions are caused by the reactivation and replication of Epstein-Barr virus within the epithelial cells [43]. The oral presentation features characteristic corrugated, white striations, most frequently on the lateral surfaces of the tongue. Occasional involvement of the dorsum of the tongue or the buccal mucosa is also possible. These adherent lesions are generally asymptomatic and can feature bilateral or unilateral involvement [41]. Large lesions may be traumatized by occlusion, sharp edges of teeth, partial denture attachments, or the acrylic extensions of complete dentures. Pain is usually not associated with oral hairy leukoplakia, and many lesions are discovered during routine clinical exams. Treatment with oral acyclovir will cause remission, but the lesions will reappear upon cessation of therapy. Valacyclovir and famciclovir have higher oral bioavailability than acyclovir and can be dosed less often but also do not eliminate the latent state of infection [41]. The lesions should not interfere with routine dental treatment. Because their appearance is simultaneous with decreasing immunocompetence, it is advisable to obtain a CBC with differential and CD4+ levels before invasive procedures are planned.

Burkitt Lymphoma

Burkitt lymphoma is a B-cell (non-Hodgkin) lymphoma that has aggressive growth characteristics [44]. There are three variations of this disease. Endemic Burkitt lymphoma is common in Central Africa and usually affects young children; the nonendemic form is more common in Western countries and affects older children and young adults. A third form is seen in patients with HIV/AIDS [45]. The Epstein-Barr virus has been found in approximately 25% to 30% of patients with HIV/AIDS who have these tumors [3; 45]. The exact mechanism by which and the extent that Epstein-Barr virus contributes to the development of these lesions is not known [45].

When Burkitt lymphoma tumors involve the jaw, the posterior region is most commonly affected. The expansion of the lesions can move and loosen teeth to the extent that extractions are required. Pain, loss of function, and paresthesia are among the symptoms that accompany the rapid growth of these lesions [45].

Treatment involves high-dose chemotherapy with medications such as combination vincristine, doxorubicin, and methotrexate [46]. These chemotherapeutic agents have a low margin of safety, and their systemic dissemination will reduce the cells that constitute the formed elements of human blood. Cells associated with the immune system, such as lymphocytes and granular leukocytes, are highly susceptible to the tumoricidal doses of these medications. Because patients with HIV/AIDS already have decreased immunocompetence, administration of these medications can subject patients to systemically disseminated opportunistic infections [45]. Chemotherapy can also decrease the platelet count with subsequent concerns about internal bleeding and the ability to achieve hemostasis. In immunocompetent patients, the cells of the formed elements of human blood will usually return to their normal range after the cessation of chemotherapy. This may not occur in patients with HIV, as non-Hodgkin lymphomas usually occur when there is advanced deterioration of the immune system.

Dental treatment should only be undertaken in attempt to palliate pain and eliminate odontogenic infections; all procedures should be approved by the patient's physician. Invasive treatment may not be permitted, and pharmacotherapeutic intervention is usually the only means of treatment.

Cytomegalovirus

Cytomegalovirus (CMV) is the largest virus to infect humans. Among every 100 adults in the United States, 50 to 80 are infected with CMV by the time they are 40 years of age [47]. CMV shares the characteristic of latency seen in other members of the herpes virus family and can be reactivated when conditions such as immunosuppression occur. Most patients who develop CMV-related pathology have a CD4+ lymphocyte count of 100 cells/mcL or less [34]. Oral manifestations can include ulcerations anywhere on the oral mucosa. These ulcerations are nonindurated with a "punched-out" appearance and a varying erythematous border [48]. They can be very painful and have an extended duration due to the heightened stage of immunosuppression at which CMV is reactivated.

The ability to wear and function with partial dentures or complete dentures may be difficult or impossible if lesions are located upon any mucosal surface that supports these prostheses. A soft diet supplemented with liquid nutritional supplements may be required. Analgesic medications and topical anesthetics can palliate the pain associated with these ulcerations and facilitate the ability to masticate and swallow properly.

An association between necrotizing gingivitis and the reactivation of CMV has been reported [49]. This can lead to extensive damage of the soft tissue, and given the degree of immunosuppression, it can develop into a stomatitis with a high degree of morbidity and risk of mortality. Antiviral medications such as valganciclovir, ganciclovir, and foscarnet taken either orally or intravenously are required when CMV infections cannot be contained by the patient's immune system. These medications have numerous side effects, including bone marrow suppression, that can further decrease the competency of an immune system that is already severely compromised. The patient's primary care physician or a specialist in infectious diseases are best suited to prescribe these medications.

Kaposi Sarcoma

Kaposi sarcoma is a malignancy of the endothelial cells that compose small blood vessels. The human herpes virus type 8 (HHV-8) plays a role in the development of these endothelial malignancies, although its exact role is uncertain. The DNA sequence of HHV-8 has been recovered from Kaposi sarcoma lesions in patients who are HIV-positive and HIV-negative [50]. When it occurs in patients with HIV, it is considered an AIDS-defining illness, and it remains the most common malignancy in this population [51].

Cutaneous and intra-oral lesions can occur and assume a variety of anatomical configurations. The palate is the most common site of intra-oral Kaposi sarcoma lesions, with the gingival tissues and tongue also exhibiting frequent occurrences. Lesions can vary in size as they extend from the tissue surface from which they originate [51]. Larger lesions can interfere with speech, mastication, proper oral hygiene, and the ability to wear partial or complete dentures. Extension into and subsequent destruction of the underlying alveolar bone is also possible.

These lesions can present in a variety of colors, most frequently red, blue, violet, and brown [51]. The lesions may be barely perceptible at the onset, but they can become large singular or multiple lesions that encompass a large surface area. Trauma to these larger lesions can cause hemorrhage, secondary infection, and/or systemic infections. Because patients with HIV/AIDS may have a reduced platelet count due to cyclic thrombocytopenia, proper coagulation may be difficult to achieve.

An incisional biopsy is still the means by which Kaposi sarcoma is diagnosed. Treatment options include surgical or laser removal, immunotherapy with alpha interferon and systemic antiviral therapy (e.g. zidovudine), or direct injections of sclerosing agents into smaller and well-localized lesions [52].

Recurrence of the initial lesion or the development of new lesions can occur as immunocompetence rapidly diminishes. The overall prognosis for patients with HIV/AIDS when Kaposi sarcoma lesions appear is poor. The use of the combination of antiviral medications to manage the HIV infection has decreased the incidence of Kaposi sarcoma lesions [51; 53]. These medications decrease the replication of HIV and increase the CD4+ count, so susceptibility to opportunistic infections is deferred, but not eliminated. The cost of these medications can be prohibitive, and the multiple side effects can discourage patient compliance.

Dental treatment should be limited to procedures that are absolutely mandatory to eliminate odontogenic pain and infection. Because the CD4+ levels are low and viral levels are high when a patient develops Kaposi sarcoma, consultation with the patient's physician and/or an infectious disease specialist should be completed before any dental treatment is initiated.

SALIVARY GLAND PROBLEMS

Salivary gland disease can occur in patients with HIV/AIDS and can cause unilateral or bilateral enlargement of the salivary glands, most commonly the parotid glands. Xerostomia can result from this glandular involvement and may be exacerbated by the use of antiviral medications [54].

The parotid gland produces serous secretions, and it is the only salivary gland that contains actual lymphoid tissue inside its capsule. The submandibular salivary gland produces both mucous and serous secretions and has lymph nodes adjacent to but not within its capsule [55]. The development of lymphoepithelial cysts within the parotid gland is a pathologic process unique to patients with HIV infection. Diagnostic imaging, such as computed tomography or magnetic resonance imaging procedures, can reveal several cysts within the parotid gland, the cumulative growth of which is manifested as visible enlargement of the gland. Lymphocytic infiltration and subsequent enlargement of the parotid gland occurs in a significant number of HIV-infected individuals [56].

When cystic lesions or a diffuse lymphocytic infiltration impede the flow of serous secretions from the parotid gland, several oral problems can develop. A decrease in the lubricating function of saliva can decrease a patient's ability to wear partial or complete dentures. When the tissue-bearing surfaces of these prostheses become less lubricated and more fragile, they are subject to the development of traumatic ulcers, which can serve as a means of local, regional, and systemic dissemination for oral pathogens. When salivary flow is decreased, a commensurate decrease in salivary immunoglobulins and compounds occurs. These substances are a first-line defense against intra-oral microbial growth, and a decrease in their oral concentrations can result in the development of opportunistic oral infections, especially in immunocompromised patients.

A decrease in salivary flow can also result in the increased retention of plaque on the teeth and an increased susceptibility to caries and periodontal disease. Patients who develop xerostomia secondary to salivary gland enlargement should be advised to maintain meticulous oral hygiene and should have more frequent recall appointments. Chewing sugar-free gum can stimulate the production of saliva from some of the residual elements of the salivary glands and may help maintain oral hygiene.

A decrease in salivary production can cause difficulty during mastication and swallowing, so patients may require frequent sips of water to facilitate these functions. The use of medications such as pilocarpine and cevimeline, which stimulate the muscarinic-type acetylcholine receptors within the salivary glands, can improve salivary production [40]. Because patients with HIV/AIDS are often taking several medications for their condition and other medical problems, pilocarpine or cevimeline should only be prescribed if there are no potential adverse drug reactions among all prescribed medications. The use of antiviral therapy has resulted in a decreased degree of salivary gland enlargement, but an interruption or discontinuance of this therapy can allow for a recurrence of this problem [57].

HUMAN PAPILLOMAVIRUS

There are more than 150 different genotypes that comprise the HPV family [58]. As opposed to the characteristic ballooning degeneration and cellular lysis that most viruses cause, members of the HPV family cause proliferation of the epithelial tissues that can result in a multitude of benign and malignant lesions [59]. Only a select few of the HPV genotypes have oral or facial patterns of distribution.

HPV genotypes 2 and 4 cause the common skin wart (verruca vulgaris) and may cause lesions within the oral cavity or, more frequently, near the skin of the lower lip. Nearly one-third of these intra-oral lesions occur on the hard and soft palates and the uvula [60]. The solitary lesions are usually asymptomatic, sessile, and not pedunculated. Patients who have skin warts around nail beds or on their fingers can transmit the HPV virus to the oral tissues by parafunctional habits, such as the biting of fingernails. Conservative surgical excision with the submission of the tissue specimen for histologic analysis is required to determine if there are any malignant aspects within the lesion. Recurrences are possible.

Anogenital warts (condyloma acuminatum) are associated with the HPV-6 and HPV-11 genotypes and occur in the genital and/or anal region [61]. Oral-genital contact is the primary means by which these lesions infect the oral tissues. Unlike the lesions of verruca vulgaris or squamous papillomas, anogenital warts in the oral region develop as multiple soft sessile lesions that are moderately infectious [62]. There is a higher degree of viral infection in the peripheral tissue adjacent to these lesions. Therefore, a wider margin of surgical excision is required when the specimens are excised and submitted for histologic analysis. This higher level of virulence also accounts for a higher recurrence rate of oral anogenital warts compared to those of verruca vulgaris [30].

The lesions of verruca vulgaris and of condyloma acuminatum do not present any specific restrictions for dental treatment. However, their discovery upon a soft-tissue examination should prompt the clinician to discuss the patient's awareness and duration of these lesions. Any lesion that has been present for more than two weeks should be excised and biopsied. Immunocompromised patients typically have higher recurrence rates after the removal of these lesions. After histologic analysis has confirmed the diagnosis of condyloma acuminatum, patients should be educated as to the means of transmission to minimize the development of new oral lesions and the transmission of the virus to others.

Genotypes HPV-6 and 11 are also the cause of lesions called squamous papillomas, which are the most frequently occurring epithelial oral lesion. The morphology of these lesions can vary and may appear to have individual finger-like projections or wider sessile bases with a corrugated topography [59]. The lingual frenum, palate, buccal mucosa, and lips are the most common oral and facial areas in which these lesions develop [63]. Most lesions are solitary, pedunculated, and asymptomatic. These lesions can be excised surgically or with laser ablation, with each specimen submitted for histologic analysis. Larger lesions on the buccal mucosa or the lips may be traumatized during mastication and become secondarily infected. These lesions pose no specific restrictions for dental treatment. However, due to the multitude of forms in which oral cancer can appear, clinicians should excise any lesion that involves the oral and maxillofacial complex that has not healed within two weeks of its initial presentation.

HECK DISEASE

Focal epithelial hyperplasia, or Heck disease, is an HPV-13 or HPV-32 infection most commonly seen among Native Americans and Alaskan Natives [64]. The clinical presentation features multiple soft, dome-shaped lesions on the buccal or labial mucosa and the tongue [65]. These lesions are potentially contagious but do not interfere with routine dental treatment. Larger lesions on the buccal or labial mucosa or the lips can be traumatized during occlusion. Surgical excision can become complicated when there are multiple overlapping lesions. Excised lesions should be biopsied to determine their nature and whether they are benign or malignant, which cannot be discerned strictly from the clinical appearance. Specific immunodiagnostic techniques, such as in situ DNA hybridization, must be performed on submitted histologic samples to confirm the presence of HPV-13 and/or HPV-32 [66].

Focal epithelial hyperplasia lesions are most common among children, but adults can also be affected. The viral-induced hyperplasia of the epithelium can result in a mucosal layer that is 8 to 10 mm thicker than average. Some lesions will regress spontaneously as time progresses. Lesions associated with Heck disease do not develop into an oral carcinoma, but their presence can be an oral manifestation of HIV/AIDS [67].

ONCOGENIC POTENTIAL

The more than 40 genotypes of HPV that can infect the genital areas are the most frequently sexually transmitted infection in the United States [68]. Cervical cancer has a strong association with several HPV genotypes, and there has been a heightened interest in the relationship between various HPV genotypes and oral and maxillofacial malignant and premalignant lesions.

In a study of 5,046 specimens of head and neck squamous cell carcinomas, nearly 26% contained HPV varied genotypes, with the HPV-16 genotype being the most prevalent [69; 22]. A separate study showed that the HPV-16 genotype is the causative agent for 90% of cases of HPV-associated oropharyngeal squamous cell carcinomas [22]. The presence of HPV was higher among oropharyngeal carcinomas (35.6%) than among squamous cell carcinomas that developed in the oral cavity (23.5%) [69]. However, the presence of HPV DNA within a biopsied specimen does not establish a definitive causal relationship. The development of any malignancy is the result of a combination of factors that can vary considerably among individuals. Most patients who develop premalignant or malignant lesions within the oral cavity and/or oropharyngeal regions have a history of tobacco use. However, an increasing number of patients with oral cancer have not used these products, and an exact etiology among them is unknown. HPV may be a consideration in the development of oral cancer, although its precise role as an oncogenic agent remains unknown. It has been suggested that HPV may reduce the ability of specific tumor suppressor genes to function properly, thereby facilitating the growth and proliferation of cancer cells.

MEASLES

The measles virus is one of several members of the RNA paramyxovirus group. Although most children in the United States are inoculated against measles as part of the standard childhood immunization schedule (as a component of the measles-mumps-rubella [MMR] vaccine), outbreaks of this disease still occur, particularly in undervaccinated communities.

Approximately 10 million cases and 128,000 deaths occur worldwide annually [70]. Although most outbreaks occur in foreign countries or in unvaccinated adults returning from international travel, there is an increased risk associated with unvaccinated children [71]. Due to parental concerns regarding vaccine safety, an increasing number of children are not receiving measles vaccinations, despite a lack of scientific evidence linking the vaccine to adverse long-term effects, including autism [10; 71].

The measles can be spread easily to susceptible individuals via contaminated airborne respiratory droplets. A 7- to 10-day incubation period is followed by the development of a fever, cough, conjunctivitis, rhinitis, photophobia, and the characteristic red maculopapular rash. The rash typically begins on the forehead and behind the ears, then spreads to other areas of the body [72].

Oral manifestations of measles occur, but alone are not diagnostic for the disease. A common feature is the development of Koplik spots [72]. These small, white necrotic spots surrounded by an erythematous halo usually appear on the buccal mucosa with variable symptomatology. Mucosal ulcerations and gingivitis can also occur but are not an identifying feature of measles.

Treatment for measles is usually palliative and supportive [73; 74]. Oral analgesics can decrease the discomfort and fever that accompanies the disease. Oral lesions can complicate mastication and swallowing, so softer foods and liquid nutritional supplements may be necessary. With supportive care, most patients recover completely. However, some will develop complications, such as encephalitis and secondary infections, that can have significant morbidity and even result in death. The World Health Organization and the CDC recommend that severe cases of measles in children be treated with vitamin A, administered immediately on diagnosis and repeated the next day [74; 75].

MUMPS

The mumps virus is another member of the paramyxovirus family, and it causes the most common disease affecting the salivary glands. Like the measles, the mumps are relatively uncommon due to childhood vaccination efforts. However, outbreaks continue to occur [76]. The mumps primarily affects children, adolescents, and young adults. Again, as with the measles, religious beliefs or parental fears due to misinformation regarding the MMR vaccine and autism have led to decreasing immunization rates. Patients who have not received the MMR vaccine for any reason risk contracting the measles, mumps, or rubella and the potential serious systemic complications that may ensue.

Approximately 70% to 80% of affected patients have bilateral swelling of the parotid glands, while the remainder have unilateral swelling [77]. The submandibular and the sublingual salivary glands can also be infected, but rarely without simultaneous involvement of the parotid gland. Although unilateral or bilateral enlargement of the parotid glands is the most distinguishing feature of this disease, it is a systemic viral infection affecting the entire body. The nervous system, glandular tissue, and organs such as the liver, kidney, and pancreas can also be affected by the mumps virus [77; 78].

Transmission occurs by direct contact or by the inhalation of viral-laden aerosolized droplets. The oral and maxillofacial manifestations of the mumps include facial swelling adjacent to the affected parotid glands and intra-oral swelling and erythema around the parotid duct, also known as Stensen duct [77]. Swelling of the parotid gland can cause trismus, or difficulty opening the mouth. Swelling and edema in the parotid region can place pressure and cause pain on the adjacent musculature that controls the functional movement of the jaws, such as during mastication.

Salivary flow may be compromised as a result of inflammation and edema within the salivary gland ducts, most notably Stensen duct. Pain can result when an action such as eating stimulates salivation in glandular and duct elements that are temporarily obstructed from edema and inflammation.

Treatment is supportive and palliative in nature [77]. Medications such as acetaminophen can provide some analgesic and antipyretic activity. A soft diet may be required if there is difficulty with the opening and closing axis of the jaw. Corticosteroid administration may be used in cases of severe swelling and inflammation. Most patients require several days for the glandular swelling to subside and will make a complete recovery. Some may develop serious complications such as meningitis, encephalitis, and myocarditis. Approximately one-third of postpubertal males with mumps will experience a painful testicular complication known as epididymo-orchitis, a condition that can result in sterility in rare instances [77].

It is possible that the prodromal pain associated with the mumps may be mistaken by the patient for temporomandibular joint (TMJ) pain, and the swelling may be mistakenly attributed to odontogenic infection. When an oral evaluation in conjunction with a thorough review of the patient's current and past medical history and their immunization status precludes pathology of dental origin, the patient should be referred to his or her physician for an immediate evaluation.

COXSACKIEVIRUS

Coxsackievirus is also a member of the picornavirus family and causes two conditions: herpangina and hand, foot, and mouth disease, both of which have prominent oral manifestations. The means of transmission for each of these diseases is usually by infected salivary droplets, but the fecal-oral route is also possible.

HAND, FOOT, AND MOUTH DISEASE

Hand, foot, and mouth disease is usually caused by coxsackievirus A16, although other coxsackievirus genotypes can serve as etiologic agents [79; 80]. The disease primarily affects children during the summer and early fall. It usually begins with fever, loss of appetite, fatigue, and sore throat, followed within one or two days by oral sores.

The oral lesions of hand, foot, and mouth disease feature vesicles that quickly rupture to form shallow ulcerations encircled by an erythematous halo. Any area of the oral mucosa can be affected, but lesions occur most often on the palate, tongue, and buccal mucosa. The lesions can be difficult to distinguish among other oral ulcerative lesions, such as aphthous ulcers. However, hand, foot, and mouth disease usually features similar cutaneous lesions on the hands and feet, although the lesions may be limited to cutaneous or oral manifestations in some cases. Afflicted patients are most contagious during the initial week of their infection. Coxsackievirus A16 remains for weeks after symptoms have dissipated, and the patient remains infectious to others during this time [80].

Treatment for hand, foot, and mouth disease is supportive and palliative. Painful oral lesions can complicate a patient's ability to masticate food and to swallow, so softer, blander foods and liquid nutritional supplements may be required. Analgesics compatible with the patient's medical history may be used to decrease oral discomfort [80]. Patients with hand, foot, and mouth disease should be instructed not to scratch or manipulate fluid-filled vesicles in any manner as the contents are infectious and can inoculate other areas.

There is no vaccine available to prevent hand, foot, and mouth disease [80]. Recovery from the disease only confers immunity against the viral strain that caused the infection, not against other coxsackievirus genotypes. It is a self-limiting disease, but complications, such as viral meningitis or encephalitis, can occur.

HERPANGINA

As noted, herpangina is caused by any one of the several genotypes of the coxsackievirus, not by a member of the herpesvirus family, as the name might suggest. This infection occurs most frequently in young children, but adults can also become infected. Patients develop a fever, sore throat, headache, and malaise. Infected salivary droplets are the primary means of transmission, but the fecal-oral route is also a possibility [81].

Vesicular eruptions associated with herpangina rupture to form shallow ulcerations with erythematous halos in the posterior aspect of the mouth. The soft palate, uvula, and tonsillar pillars and fauces are the common areas of involvement. This predilection for the posterior aspect of the mouth and oropharynx differentiates these lesions from aphthous ulcers and from hand, foot, and mouth disease, the lesions of which appear in all areas of the oral cavity [80].

Treatment is palliative and supportive. Herpangina lesions can be very painful, especially during swallowing. Patients may require a soft diet with liquid nutritional supplements. Analgesics may be required in a liquid formulation, as pills or capsules can be difficult to swallow, especially for children [80]. Acidic foods or beverages should be avoided. Immunity to a herpangina infection is limited to the coxsackievirus genotype that caused the infection. Ulcerations generally regress in approximately one week, and most patients recover quickly without systemic complications.

CONCLUSION

This course has reviewed several viruses and their associated local, regional, and systemic pathology. Unlike bacterial and fungal infections, infections of viral origin can be attenuated and treated with antiviral medications, but generally cannot be cured. Prevention is often the most important step to minimizing the effects of these diseases. Hepatitis A and B and MMR vaccinations are excellent preventive measures, but unfortunately, most viral diseases have no available vaccinations. Therefore, patients should be advised to take steps to prevent transmission of the viruses.

Emerging viruses and mutations of current viruses will continue to challenge the allied health professions. Preventive measures and active treatment protocols can decrease the morbidity and mortality that can result from viral infections.

Dental professionals should consult with a patient's physician prior to dental treatment when diseases of viral origin are present. Issues such as immunocompetence and the ability to coagulate properly can be altered by these diseases and should be addressed before any invasive procedures. A respect for the diverse range of pathogenicity that viral organisms can cause is an essential component of maintaining a patient's oral and systemic health.

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