

Common Childhood Viral Infectious Diseases with Oral Symptoms

Çağla Şimşek¹, Büşra Karaduran¹, Bilge Keskin², Mine Koruyucu¹

¹Department of Pedodontics, İstanbul University, Faculty of Dentistry, İstanbul, Türkiye

Cite this article as: Simsek C, Karaduran B, Keskin B, Koruyucu M. Common childhood viral infectious diseases with oral symptoms. Essent Dent. 2024;3(2):63-71.

Abstract

The entry of a pathogenic microorganism into the body can result in infectious diseases. The prevalence of viral infectious diseases is notably high during childhood, causing various rashes on the body and oral manifestations, and presenting a risk of transmission. This makes it a crucial and significant concern in pediatric dentistry. Attention should be given to the features, locations, and durations of the rashes they induce. Pediatric dentists can play a substantial role in diagnosing and treating various viral infectious diseases, often necessitating collaboration with pediatricians in a multidisciplinary approach. This review specifically focuses on common viral infections in childhood that impact the oral region, pose a risk of transmission, and hold significance in the field of pediatric dentistry. It emphasizes infectious diseases caused by measles, rubella, mumps, viruses of the herpes family, Erythema infectiosum, hand-foot-mouth disease, acquired immunodeficiency syndrome, and hepatitis viruses.

Keywords: Pediatric dentistry, infection, viral

INTRODUCTION

The category of illnesses, referred to as infections, is identified by pathological symptoms arising from the general or local colonization of a specific and pathogenic microorganism that enters the human body through any means. These illnesses have the potential to be transmitted from one individual to another under specific conditions and are defined by the symptoms induced by the microorganisms themselves or their toxins. In childhood, infectious diseases with these characteristics are particularly significant due to their frequent occurrence, with many of them being caused by viruses. Often, direct diagnosis can be made solely through clinical examination. In a child with a high fever accompanied by maculopapular rash, consideration should be given to infectious viral diseases because viral infections commonly affect mucous membranes and can create specific lesions in the oral mucosa that may be crucial in differential diagnosis. Recognized transmission routes include transplacental, perinatal, and postnatal exposures during childbirth. Diagnostic challenges may be encountered in their identification, and these diseases can be categorized based on the clinical appearance of the exanthem and the causative virus.2

MEASLES

Measles disease is caused by a single-stranded RNA virus within the paramyxovirus family. The virus is highly sensitive to thermal changes, leading to rapid inactivation. While it can occur at any age, it is more commonly manifested in children than in adults. It is a viral disease with high infectivity, transmitted through droplets and aerosols. The virus enters and begins to replicate primarily in epithelial cells of the mouth and nose, especially in the trachea and bronchi. Some of the virus also enters the circulatory system and establishes itself in lymphoid tissue cells.³ The course of the disease typically lasts for approximately 7-10 days. Fever, cough, nasal congestion/nasal discharge, and conjunctivitis are early indicators of measles. This prodromal period persists for 2-4 days, during which measles is contagious, and symptoms such as fatigue,

Corresponding author: Büşra Karaduran e-mail: bsrkaraduran@gmail.com



Content of this journal is licensed under a Creative Commons Attribution-NonCommercial 4.0 International License.

Received: January 9, 2024 Revision Requested: February 9, 2024

Last Revision Received: February 9, 2024 Accepted: February 19, 2024

Publication Date: March 18, 2024

² Private Haliç Panorama Oral and Dental Health Polyclinic, İstanbul, Türkiye

myalgia, photophobia, and periorbital edema may also occur in conjunction with nasal congestion.²

In many patients, small bluish-white maculopapular rashes, approximately 1 mm in diameter, surrounded by a pink-red ring, are observed on the buccal mucosa. These rashes, known as Koplik spots, are pathognomonic for measles. They appear around the opening of Stensen's duct, near the second molar teeth, on the buccal mucosa. Typically, they manifest 1–2 days before the initial rash and can be seen up to 1–2 days after its onset. By the third day of the rash, the oral mucosa returns to normal.² Koplik spots indicating the onset of measles are shown in Figure 1.⁴

After the initial symptoms resolve, brownish-red hyperpigmented maculopapular rashes, a characteristic feature of measles, appear on the patient's face and neck, spreading across the body. They reappear, originating from the same areas, and then gradually vanish, starting from the head region. This cycle typically lasts 3-7 days. The most contagious period for patients is the 4 days before the rash emerges and the 4 days after its appearance. In essence, measles is highly contagious within 7-8 days following the onset of the first symptom. Desquamation in rashes is uncommon. In malnourished children, rashes are more likely to merge, displaying a dark red color.⁴

Measles can contribute to the development of pneumonia in both children and adults. Additionally, it may give rise to complications such as otitis media, pneumonia, post-infectious encephalitis, and, in rare cases, death. Complications are more prevalent in cases where fever persists for an extended period after the appearance of the rash. In children, pneumonia is a significant contributor to mortality, while in adults, encephalitis may occur more frequently. The repercussions of these complications are severe in underdeveloped and developing countries, where mortality rates are high.^{5,6} Individuals with vitamin A deficiency are at higher risk of developing complications associated with measles.

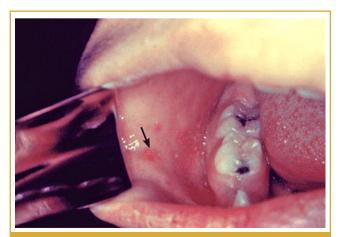


Figure 1. Koplik spots (arrow).4

Furthermore, measles can exacerbate vitamin A deficiency, potentially leading to blindness in cases where there is already a lack of vitamin A.⁶ Adequate vitamin A supplementation plays a crucial role in the prognosis of the disease.⁷

The sole method of protection against measles is through the administration of the combination vaccine known as the measles, mumps, rubella vaccine (MMR vaccine). Individuals who have received the MMR vaccine tend to experience milder symptoms or may even show no signs of the disease. Moreover, vaccinated individuals do not contribute to the transmission of the virus. While measles generally resolves spontaneously, hospitalization may be necessary in some cases, even in the absence of complications. A child who comes into contact with measles can be protected from the disease by receiving the measles vaccine within 1–2 days of exposure. Active vaccination is recommended when the child is still between 9 and 12 months old. Undergoing the illness provides the individual with lifelong immunity.

There is no specific antiviral treatment for measles. Attention should be given to the care of the patient's mouth, eyes, and skin. It is recommended to provide watery and soft foods. If the child has a fever, antipyretic medications can be used. In the case of vitamin deficiency, vitamin supplements should be administered. Timely detection of infected children during a measles outbreak is crucial to prevent the further spread of the disease. Bed rest should be prescribed to the patient, and individuals not affected by the illness should be kept at a distance. In cases accompanied by otitis media, antibiotics from the penicillin group may be prescribed.⁷

RUBELLA

Rubella is caused by rubivirus, a single-stranded RNA virus from the togavirus family. The virus is highly sensitive to heat, pH changes, and various chemical agents, making it easily inactivated. The contagious period of the virus is typically around 20 days, and it commonly manifests in children aged 5-10. The incubation period is 16-18 days. The course of the disease is mild and is often accompanied by pharyngitis and lymphadenopathy. It is characterized by moderate fever and macular rash. Rubella is transmitted through droplets and transplacental routes.8 In children, rubella manifests with a rash starting on the face and neck, extending to the chest and limbs, lasting approximately 3 days. Adolescents and adults experience a prodromal phase lasting 1-5 days before the rash, featuring symptoms like fever, headache, fatique, loss of appetite, conjunctivitis, runny nose, sore throat, cough, and lymphadenopathy. Enlargement of lymph nodes, notably in suboccipital, posterior cervical, and posterior auricular regions (Theodor phenomenon), begins at least a week before the rash, causing painful lymphadenopathies with redness on the face and neck. Simple erythematous stomatitis, starting from the soft palate and progressing to the hard



Figure 2. Forchheimer spots on the soft palate.

palate, or initial discolorations resembling Forchheimer spots on the soft palate, may occur briefly (Figure 2).9

Rubella virus is a teratogenic virus. During pregnancy, especially if the mother becomes infected with the rubella virus in the first trimester, the virus can pass to the fetus through the placenta, causing chronic infection and leading to various congenital anomalies. Complications such as stillbirth, miscarriage, or premature birth can occur due to maternal rubella infection. Complications that may arise if the child is born alive include congenital heart anomalies, microcephaly, intellectual disability, deafness, dental and facial defects, and developmental delays. Dental complications that can be observed include enamel hypoplasias, malocclusions, tooth deficiencies, and cleft palates. These complications can occur together or independently.¹⁰

Rubella generally resolves spontaneously. There is a vaccine available, administered through the MMR vaccine. Rubella is resistant to antivirals. It is among the mildest of infectious diseases in children. Treatment is symptomatic, and bed rest is recommended if there is a fever.¹¹

MUMPS

Mumps, caused by paramyxovirus, an enveloped, single-stranded RNA virus, also termed epidemic parotitis, is a contagious childhood disease transmitted through direct contact or droplets. Typically, outbreaks occur from late winter to spring, but year-round occurrences alongside other diseases are possible. Fever often accompanies the disease, with a 2–3-week incubation period. The virus enters through oral and nasal mucosae, multiplying in the upper respiratory

epithelium during incubation, and spreading to lymph nodes and bloodstream. Subsequently, dissemination to the parotid gland and central nervous system meninges occurs through the blood, followed by secondary spread to other organs, known as secondary viremia.¹²

Mumps primarily affects the parotid glands but can rarely involve the pancreas, testes, or brain. In some cases, inflammation of the central nervous system may also occur. Swelling and tenderness manifest in the area where the parotid glands are located, extending downward from the front of the earlobe to the back of the jaw, causing the earlobes to appear elevated. The swelling typically begins in one of the parotid glands, and within a few days, the other may become affected in the same way. Edema can occur due to the obstruction of lymphatics by inflamed salivary glands. Redness and congestion in the throat are observed, accompanied by pain during swallowing. The infectious period extends from 2 days before the onset of parotitis to 5 days after. 12,13 Swelling lasts for 4-10 days and may be accompanied by trismus and muscle pain. When the patient yawns, tries to eat, or opens the mouth, they experience pain due to the constriction or complete blockage of the ducts of the parotid and other salivary glands. This pain is felt because saliva cannot flow into the mouth. Foods that stimulate salivary secretion or chewing functions can exacerbate the pain. The opening of Stensen's duct, the duct of the parotid gland, is often reddened at the level of the maxillary molars. Children who have had mumps can rarely develop some complications after the disease, including neurological complications such as sudden sensorineural deafness, aseptic meningitis, and encephalitis. Transient hearing loss observed in mumps patients can also rarely result in permanent deafness.¹⁴ Recovery typically occurs spontaneously and completely within approximately 7-10 days. Symptomatic and supportive treatments are applied in cases of parotitis. Analgesic-antipyretic medications can be used. Local application of cold or warm compresses and massage may help reduce pain. Avoidance of foods that stimulate salivary secretion, consumption of soft and liquid foods, and, if necessary, intravenous fluid administration are recommended. Bed rest is advised for the patient, and they should be kept away from individuals who have not had the disease.15

Mumps is less contagious than measles and chickenpox. Recovering from the disease provides lifelong immunity. The safest way to protect against mumps is through vaccination. It can be administered as part of the MMR vaccine or as a standalone vaccine. The first dose of the mumps vaccine is usually given at 12–18 months of age, with the second dose administered between 2 and 6 years old. 16,17

HUMAN HERPES VIRUSES

Oral herpes virus infections are typically diagnosed by dentists. Saliva serves as a source for the herpes virus, and due to its presence, it can lead to various localized and systemic

infections. It is common in children, and except for the varicella zoster virus (VZV), which is primarily transmitted through aerosol, direct contact is usually required for the transmission of herpes viruses in children. None of the human herpes viruses (HHVs), except VZV causing chickenpox, are symptomatic during initial infection and do not show any signs. However, the primary symptoms caused by other herpes viruses include gingivostomatitis for herpes simplex virus type 1 (HSV-1), genital herpes for herpes simplex virus type 2 (HSV-2), mononucleosis for Epstein–Barr virus (EBV), and measles for human herpes virus type 6.18

HERPES SIMPLEX VIRUS

There are 2 types of herpes simplex virus (HSV): HSV-1 and HSV-2. HSV-1 and HSV-2 share similar cell structure and pathogenesis, but they differ in their potential to cause disease, the anatomical region where symptoms occur, and epidemiology. HSV-1 is a member of the Alphaherpesvirinae subfamily. It is an enveloped virus with a diameter of 100-110 nm, a linear, double-stranded DNA. It typically forms vesicles on the face and affects the area around the mouth and nose. Herpes simplex virus type 1 infection also has 2 different types within it. Primary infection usually appears as widespread gingivostomatitis in children under 4-5 years of age. Secondary infection is defined in individuals with a history of primary infection when the virus reactivates at a later stage. On the other hand, HSV-2 affects the genital area, leading to the formation of rashes. 19,20

Herpes simplex virus type 1 infections can be transmitted through direct contact with asymptomatic or symptomatic infected individuals. The most common symptom when the virus is transmitted is a cold sore. 19,20 In children, HSV-1 infection typically presents with symptoms such as fever, fatigue, loss of appetite, irritability, sore throat, bad breath, and difficulty swallowing, manifesting as gingivostomatitis. After an asymptomatic period or 7-10-day incubation period, it manifests as painful vesicles with characteristic boundaries on the mucocutaneous surface. Cervical lymphadenopathy may accompany the condition. Additionally, the hard and soft palate can be affected along with the appearance of large, shallow, and yellowish ulcers on the tongue, lips, gum, tonsils, buccal, and pharyngeal mucosa. 19,21 Moreover, it can manifest itself with skin infections such as eczema herpeticum, genital herpes, and herpetic whitlow.²² Herpetic whitlow can occur in HSV-1 infected children through finger sucking and nail-biting, as well as in dentists who come into contact with infected child patients without using protective equipment.^{20,23,24}

In HSV-1 infection, healing is usually observed within 7-10 days without leaving a scar, but recurrences can occur. Antiviral therapy helps limit the course of HSV infection. ^{20,25} Dentists should have a thorough understanding of the pathology and clinical symptoms of the disease and be

able to accurately diagnose it in the clinical settings. The diagnosis of the disease can be made by the dentist in the clinical setting.²⁶

Acyclovir is highly effective antiviral medication widely used in the treatment of HSV infections, chickenpox, and shingles. Treatment with acyclovir should be initiated within 72 hours of the onset of the first symptom. The recommended oral dose of acyclovir is 40-80 mg/kg per day, divided into 3-4 doses, for 1 week.²¹ Parents should be informed about the potential side effects of acyclovir, such as vomiting, weakness, and headache, and should monitor the patient accordingly. However, it is essential to remember that many infections resolve spontaneously, and even if the treatment is initiated with delay, it may not necessarily alter the duration or severity of the symptoms.^{26,27} Topical antiviral agents, usually in the form of ointments, are commonly used, and lesions typically improve within 6-8 days of their application. In addition to medication, providing adequate fluid supplementation to the patient is crucial. Some patients may need hospitalization to prevent dehydration. It is recommended that the patient wash their hands frequently and thoroughly and avoid close contact with others. Parents should also be educated about the transmission of the virus.²⁸

VARICELLA ZOSTER VIRUS

Varicella-zoster virus (VZV) belongs to the Varicellovirus genus and has a double-stranded linear DNA molecule. It can affect the branches of the trigeminal nerve in the facial region, leading to discomfort in the face, mouth, eyes, or tongue. When it affects the mandibular (third) or maxillary (second) branch of the trigeminal nerve, symptoms may manifest within the oral cavity. However, the most commonly observed trigeminal nerve damage (in 50% of cases) is ocular involvement.²⁹

Varicella-zoster virus causes 2 distinct diseases. Varicella (chickenpox, primary infection) is typically observed in children, while herpes zoster (shingles, recurrent infection) is more common in adults. The disease provides lifelong immunity, but the virus remains latent in the body. Unlike easily recurring viruses such as HSV, the latent VZV does not easily reactivate, but when it does reactivate years later, it can lead to shingles. Individuals in contact with someone who has had shingles may be susceptible to contracting the disease again. The primary concern for oneself or a child's parents is to avoid secondary infections. Since shingles is an endogenous infection, it can occur irregularly and infrequently at any age, but it is rarely reported in individuals under 10 years old. Chickenpox, on the other hand, is a childhood disease with an exanthematous vesicular rash. It is transmitted from the upper respiratory mucosa, oropharynx, or conjunctiva, and is highly contagious. While chickenpox is more prevalent in late winter to early spring, there is no specific timeframe associated with the occurrence of shingles.³⁰ The incubation period is 2-3 weeks. Widespread vesicular rash and fever are the main clinical manifestations. As long as new rashes appear, the fever persists. The rash may be accompanied by significant itching. The vesicles of chickenpox are typically observed on the mucosa of the tongue, gums, lips, cheeks, soft palate, nasopharynx, larynx, and genital organs. These vesicles spread from the periphery to the center (centripetal). The spread of vesicles begins on the face and the roots of the hair (on the scalp). Subsequently, they are seen on the back, abdomen, chest, arms, and legs. The acute phase lasts for 5-6 days, and scabs begin to peel off within 10-14 days.³¹ One of the main challenges for dentists in making a diagnosis is determining the cause of vesiculo-ulcerative disorders in the oral cavity. The possibility that VZV may contribute to the etiopathogenesis and root resorption of periapical pathologies should also be taken into consideration.32

Varicella–zoster virus causes fewer vesicles in the oral cavity compared to HSV. Dentists can diagnose the disease based on the clinical symptoms caused by these 2 viruses, but more precise identification can be achieved through laboratory tests.³³ Conditions such as trigeminal neuralgia, acute pulpitis, jaw osteonecrosis, odontalgia, periapical lesions, periodontal destruction, and osteomyelitis, where frequent episodes of pain are common, can sometimes mislead dentists in making a diagnosis and providing treatment.³²

The most effective way to protect against this virus is vaccination. In the postnatal period or in certain medical conditions, Varicella-zoster immune globulin serum can be used in children who have active chickenpox disease. Generally, treatment is not required. The patient is usually re-evaluated 1 week later to observe whether the symptoms have improved. The use of antipyretics can be beneficial during periods of high fever. If itching is severe, anti-itch creams can be applied topically. It is crucial to control children to prevent them from scratching and rupturing the vesicles, avoiding inoculation. Emphasis should be placed on oral and skin hygiene, and bed rest is highly important. Early diagnosis and detailed medical and dental history contribute to the success of the treatment.³⁴

INFECTIOUS MONONUCLEOSIS (KISSING DISEASE)

Infectious mononucleosis is caused by the EBV, a double-stranded DNA virus belonging to the gamma herpes virus family. Epstein-Barr virus can settle on B lymphocytes and activate them. The prevalence of EBV in the adult population is 90%-95%, while in children, it is around 60%-70%. Transmission commonly occurs through saliva or kissing. In children, transmission can happen by sharing toys or objects that come into contact with the mouth. The incubation period in children is typically 10-14 days. The initial symptoms of the disease often include fever, fatigue, loss of appetite, sore throat, pharyngitis, and myalqia. Lymphadenopathy

follows these prodromal symptoms.³⁵ The tonsils are the initial site of the virus's involvement and also serve as the source. The duration and severity of the disease can vary significantly. Petechiae that disappear in a few days on the soft palate, necrotizing ulcerative gingivitis, and edema in the uvula and infraorbital area can be observed. The disease tends to be mild in children, while it is more severe and prolonged in adults. Fever usually rises up to 39° and gradually decreases within an average of 6 days. In severe cases, it can go up to 40°-41° and may last for 3 weeks or longer. In children under 4 years of age with severe conditions, hepatosplenomegaly, rhinitis, and cough may be observed. In children under the age of 2, the disease is generally asymptomatic. 36,37 However, it generally has a self-limiting nature. During the acute phase, bed rest and antipyretic and analgesic medications can be given to reduce fever and alleviate throat pain. The use of corticosteroids in the treatment of infectious mononucleosis is controversial. Although it has been found to shorten the duration of fever and alleviate symptoms, it is not highly recommended due to its potential to disrupt the immune system balance. However, it may be administered in cases of severe complications such as airway obstruction, significant thrombocytopenia, hemolytic anemia, central nervous system involvement, myocarditis, and pericarditis.38

Currently, there is no vaccine available for EBV. Due to its malignant potential, live attenuated vaccines are not recommended. Patients typically recover on their own within 4–6 weeks. The use of ampicillin or amoxicillin should be avoided as it can cause a rash in individuals with this disease. Even 18 months after clinical improvement in patients infected with this virus, EBV has been detected in throat gargle samples, and it can reactivate in individuals with suppressed immune systems.^{38,39}

CYTOMEGALOVIRUS

Cytomegalovirus (CMV) is a member of the herpesvirus family that can remain latent in salivary glands, endothelium, macrophages, and lymphocytes. It is commonly found in saliva, blood, urine, tears, respiratory secretions, and breast milk. Transmission in infants can occur through the placenta, breast milk, or direct contact. It is typically observed in newborns and immunocompromised individuals, with about 90% of cases being asymptomatic. The clinical manifestation of the virus occurs either due to primary infection or reactivation when the immune system is suppressed. Symptomatic acute CMV infections are rare but can lead to various diseases ranging from infectious mononucleosis to fatal multiple organ failure. In CMV-induced mononucleosis, fever, chills, sore throat, headache, and fatigue are typical symptoms. Exudative pharyngitis, lymphadenopathy, hepatosplenomegaly, muscle pain, abdominal pain, cough, maculopapular rash, and diarrhea may accompany the condition. Some cases may also present with xerostomia, and swelling and pain in the salivary glands can be observed.⁴⁰

It can lead to morbidity and mortality in immunocompromised children. Congenital and neonatal infections may present complications such as hepatosplenomegaly, jaundice, purpura, mental retardation, deafness, and microcephaly. While most CMV infections resolve spontaneously, treatment is crucial, especially in immunocompromised patients. The applied treatment should aim to prevent recurrence in the patient. Intravenous ganciclovir is used in the treatment of both CMV and HSV in patients with oral ulcers. There is currently no vaccine developed specifically for CMV infection. 40

EXANTEM SUBITUM (ROSEOLA INFANTUM, SIXTH DISEASE, HHV-6)

The causative agent of the disease is HHV-6. The disease has 2 types: the rash-type infection caused by the Human B cell lymphotropic virus (HBLV) and the nonrash-type infection. It is commonly observed as sporadic cases in children aged 6 months-3 years. The likelihood of contagion is unknown, and it is more frequently encountered in the spring and fall months. Transmission occurs through droplets and saliva, with an incubation period of 7–17 days. The initial symptoms of the disease include restlessness, mild cold symptoms, and the most prominent symptom, the sudden onset of high fever reaching up to 40°C. The fever lasts for 3-5 days. Other observable symptoms include cough and diarrhea. Febrile convulsions (fever seizures) occur in 6%-15% of cases. Nausea and vomiting are rare. Cervical and occipital lymphadenopathy may also accompany the disease.⁴² During the period of high fever before the rash, periorbital edema can occur. After the fever subsides, maculopapular rashes, measuring 2-3 mm in diameter, begin to appear on the face and body, spreading to the arms and legs. These rashes last for 1-2 days, and itching is not observed. In some patients, they are only present on the face and body, and the color fades when pressure is applied with a finger. Ulcers may appear in the uvulopalatoglossal region in infants, and these ulcers are referred to as Nagayama signs. Irritation, inflammation of the tympanic membrane, a runny nose, and abdominal pain may also accompany the symptoms.⁴³ It usually heals without leaving a scar. 42 Supportive care, consisting of fluid supplementation and antipyretics, is generally sufficient for treatment. In cases of febrile convulsions, acetaminophen is recommended as a fever reducer.43

ERYTHEMA INFECTIOSUM (FIFTH DISEASE)

The causative agent of the disease is Parvovirus B19, a single-stranded DNA virus. It is commonly observed in individuals aged 5-15 years. Transmission occurs through respiratory droplets or direct contact with the blood of an infected person. The disease is often seen in epidemic form, especially during the winter and spring months.⁴⁴ The course of the disease is mild. The incubation period is 4-19 days, and there is no prodromal period. The clinical manifestations in

children infected with the virus consist of 3 stages: In the first stage, called "slapped cheek" or "butterfly appearance," pallor around the lips and erythematous rash on the cheeks are observed. In the second stage, reticular rashes appear on the trunk and the inner parts of the arms/legs begin to fade centrally. These rashes are usually seen in extensor areas and are very itchy but can heal without peeling, and they are not found on the hands and feet. The defined manifestations in the third stage are recurrent rashes resulting from exposure to the sun, taking hot showers, excitement, exercising, or minor traumas. Flare-ups occur intermittently and last an average of 11 days. Symptoms accompanying this clinical picture include fever, headache, sore throat, abdominal pain, runny nose, fatique, arthralgia, and myalgia. The patient's infectivity ends when the rash begins. Although there is no significant anemia in healthy children, some patients with anemia, where the lifespan of red blood cells is shortened, may experience aplastic crises, and in immunocompromised patients, it may lead to chronic bone marrow insufficiency.⁴⁵ Patients typically recover on their own, and symptomatic treatment is rarely necessary. Transfusion is recommended for patients experiencing aplastic crises, while immunoglobulin administration is suggested for patients with immunodeficiency.⁴⁴

HAND-FOOT-MOUTH DISEASE

Hand-foot-mouth disease results from the Coxsackie virus A16, transmitted through respiratory droplets or fecal-oral contact. It is highly contagious, often affecting children under 5. Symptoms include fever, sore throat, dysphagia, and intraoral blisters (2-7 mm) on the lips, tongue, gums, and throat. Similar rashes may appear on the palms and soles.⁴⁶ The incubation period is 3-6 days. The initial virus site in the oral mucosa is the buccal mucosa, spreading to lymph nodes within 24 hours. The disease presents with mild symptoms like low fever, fatigue, lymphadenopathy, and mouth sores, typically not severe. The main complaint is usually pain from oral lesions, accompanied by excessive salivation and reluctance to eat. Oral lesions start as vesicles, later rupturing into ulcers surrounded by an erythematous area with a greenish fibrinous membrane. Multiple lesions can occur anywhere in the mouth, with the soft palate, lateral or dorsal side of the tongue, and buccal mucosa being the most commonly affected areas.⁴⁷ Lesions usually self-heal in 7-10 days, with the illness lasting around 10-14 days. Although rare, there is a potential for the Coxsackie virus to reach the brain, causing complications like viral meningitis or encephalitis. Treatment is generally symptomatic, including mouth gargles for oral relief. Adequate fluid intake is crucial, and antipyretic medications may be given for high fever.46

The diagnosis of Hand-Foot-and-Mouth disease relies on a detailed medical history and clinical examination.⁴⁶ Oral lesions associated with this disease may be mistaken for aphthous ulcers, chickenpox, or herpangina. However, aphthous ulcers typically differ as they do not initially form blisters and

are more painful and recurrent. Chickenpox rashes are usually concentrated on the body and rarely manifest as oral lesions, although some cases may involve the palms of the hands and soles of the feet. Herpangina, caused by Coxsackie virus A, presents with oral ulcers similar to those in hand, foot, and mouth disease, involving the tonsils, pharyngeal mucosa, soft palate, and posterior buccal mucosa. ⁴⁷

ACQUIRED IMMUNODEFICIENCY SYNDROME

Acquired immunodeficiency syndrome (AIDS) is caused by the Human Immunodeficiency Virus (HIV), a retrovirus from the RNA virus family. Transmission occurs through direct blood contact, perinatal transmission, contamination, or sexual abuse of an infected person. Acquired immunodeficiency syndrome in young children is often transmitted from mother to baby. In perinatally infected children, viral suppression is less effective than in adults, leading to a poor prognosis and often resulting in death. The initial manifestation of AIDS involves various oral lesions such as gingivitis, periodontitis, necrotizing stomatitis, candidiasis, hyperpigmentation, salivary gland infections, aphthous ulcers, herpes simplex, hairy leukoplakia, Kaposi's sarcoma, and papilloma. Main symptoms include lymphadenopathy, hepatosplenomegaly, chronic diarrhea, night sweats, and weight loss, accompanied by bacterial infections, sepsis, pneumonia, meningitis, otitis media, sinusitis, urinary tract infection, and infections of bones, joints, skin, and soft tissues. 48,49 The suppression of the immune system leads to the reactivation of the virus from the inactive state to the active state, allowing the disease to recur. Early diagnosis and treatment of HIV are crucial to prevent the development of opportunistic infections. This way, it becomes possible to prevent the progression of AIDS disease.50

Acquired immunodeficiency syndrome is typically diagnosed using an ELISA test. Alternative methods like Western blot, immunofluorescence assay, viral culture, and antigen tests can also aid in diagnosis. In the differential diagnosis,

consideration should be given to herpangina, primary herpetic gingivostomatitis, and erythema multiforme. It is common in children for symptoms not to improve or to recur. Combination therapy with potent antiretroviral drugs is administered for AIDS treatment.⁵⁰

HEPATITIS (JAUNDICE)

Hepatitis virus infections constitute a significant concern among infectious diseases in dentistry. Recent studies have identified 6 hepatitis viruses, namely A, B, C, D, E, and G. The diagnosis of jaundice caused by the Hepatitis A virus, which is particularly common in newborns and children, is determined by an increase in bilirubin levels in the blood. The elevation of bilirubin occurs as a result of hemoglobin breakdown in the blood. Individuals with jaundice exhibit diffuse, uniform, and yellowish discoloration of the skin and mucous membranes. The intensity of the discoloration is directly proportional to the bilirubin levels in the blood. The discoloration, starting from the sclera, is especially observed in the soft palate and the floor of the mouth. Jaundice may be confused with the discoloration seen in hypercarotenemia, but the lack of scleral involvement in this condition allows differentiation. The treatment of jaundice varies depending on the etiological factor. In neonatal jaundice, the condition may resolve spontaneously, but in some cases that do not improve, babies are placed in a crib and treated with UV light.⁴⁰

The etiologies and oral findings of common childhood viral diseases described in this review study are summarized in Table 1.

CONCLUSION

Viral rashes in children are usually acute infectious diseases that often resolve without specific treatment. These illnesses often present specific symptoms in the oral cavity, affecting soft tissues, oropharynx, and salivary glands. Identifying characteristic lesions in the oral mucosa can aid in specific

Table 1. Etiologies and Oral Findings of Common Viral Diseases in Children

Disease	Etiology	Oral Symptoms
Measles	Measles virus	Koplik spots
Rubella	Rubivirus	Forchheimer spots
Mumps	Paramyxovirus	Swelling and tenderness in the area where the parotid glands
HSV-1	Herpes simplex	Gingivostomatitis, yellowish ulcers
VZV	Varicellovirus genus	Vesiculo-ulcerative disorders
Infectious mononucleosis	EBV	Loss of appetite, sore throat, pharyngitis, and myalgia
CMV	CMV	Oral ulcers, xerostomia, and swelling and pain in the salivary glands
Sixth disease	HHV-6	Nonrash-type infection
Fifth disease	Parvovirus B19	Pallor around the lips and erythematous rash on the cheeks
Hand-foot-mouth disease	Coxsackie virus A16	Fever, sore throat, dysphagia, and intraoral blisters
AIDS	HIV	Gingivitis, periodontitis, necrotizing stomatitis, candidiasis, hyperpigmentation, salivary gland infections, aphthous ulcers, herpes simplex, hairy leukoplakia, Kaposi's sarcoma, and papilloma
Hepatitis A	Hepatitis A virus	Yellowish discoloration of the skin and mucous membranes

diagnoses. Infectious diseases with oral clinical findings can be a significant source of morbidity and mortality in children. It is crucial to recognize and treat common infectious diseases during childhood based on oral manifestations.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – M.K., Ç.Ş., B.K.; Design – M.K., B.K., Ç.Ş., B.K.; Supervision – M.K., Ç.Ş., B.K.; Resource – Ç.Ş., B.K., B.K.; Data Collection and/or Processing – B.K., Ç.Ş., B.K.; Analysis and Interpretation – M.K., Ç.Ş., B.K.; Literature Search – Ç.Ş., B.K.; Writing – Ç.Ş., B.K., B.K.; Critical Review – M.K., Ç.Ş., B.K.

Declaration of Interests: The authors have no conflicts of interest to declare.

Funding: The authors declared that this study has received no financial support.

REFERENCES

- Lynch DP. Oral viral infections. Clin Dermatol. 2000;18(5):619-628. [CrossRef]
- Alter SJ, Bennett JS, Koranyi K, Kreppel A, Simon R. Common childhood viral infections. Curr Probl Pediatr Adolesc Health Care. 2015;45(2):21–53. [CrossRef]
- O'Donnell S, Davies F, Vardhan M, Nee P. Could this be measles? *Emerg Med J.* 2019;36(5):310–314. [CrossRef]
- 4. Kumar D, Sabella C. Measles: back again. Cleve Clin J Med. 2016;83(5):340–344. [CrossRef]
- Stock I. Measles. Med Monatsschr Pharm. 2009;32(4): 118-127.
- Cherry J, Demmler-Harrison GJ, Kaplan SL, Steinback WJ, Hotez PJ. Feigin and Cherry's textbook of pediatric infectious diseases e-book. Amsterdam: Elsevier Health Sciences; 2013.
- Moss WJ, Griffin DE. Measles. Lancet. 2012;379(9811):153-164. [CrossRef]
- Röteln SI. Noch immer eine bedeutende Infektionskrankheit [Rubella (German measles) -- still a major infectious disease]. Med Monatsschr Pharm. 2012;35(1):14-22; quiz 23.
- 9. Fukuda M, Harada T, Shimizu T, Hiroshige J. Forchheimer spots in rubella. *Intern Med.* 2020;59(13):1673. [CrossRef]
- Bouthry E, Picone O, Hamdi G, Grangeot-Keros L, Ayoubi JM, Vauloup-Fellous C. Rubella and pregnancy: diagnosis, management and outcomes. *Prenat Diagn*. 2014;34(13):1246-1253.
 [CrossRef]
- Winter AK, Moss WJ. Rubella. Lancet. 2022;399(10332):1336-1346. [CrossRef]
- Lam E, Rosen JB, Zucker JR. Mumps: an update on outbreaks, vaccine efficacy, and genomic diversity. Clin Microbiol Rev. 2020;33(2):e00151-19. [CrossRef]
- Kim JS, Oh JS, Kwon SH, Kim MS, Yoon YJ. Mumps presenting as a parotid abscess. J Craniofac Surg. 2016;27(3):e233-e235.
 [CrossRef]
- Rubin S, Eckhaus M, Rennick LJ, Bamford CG, Duprex WP. Molecular biology, pathogenesis and pathology of mumps virus. J Pathol. 2015;235(2):242–252. [CrossRef]
- Centers for Disease Control and Prevention (CDC). Updated recommendations for isolation of persons with mumps. MMWR Morb Mortal Wkly Rep. 2008;57(40):1103-1105.

- 16. Bankamp B, Hickman C, Icenogle JP, Rota PA. Successes and challenges for preventing measles, mumps and rubella by vaccination. *Curr Opin Virol*. 2019;34:110–116. [CrossRef]
- 17. Kauffmann F, Heffernan C, Meurice F, Ota MOC, Vetter V, Casabona G. Measles, mumps, rubella prevention: how can we do better? *Expert Rev Vaccines*. 2021;20(7):811–826. [CrossRef]
- Crimi S, Fiorillo L, Bianchi A, et al. Herpes virus, oral clinical signs and QoL: systematic review of recent data. *Viruses*. 2019; 11(5):463. [CrossRef]
- Darji K, Frisch S, Adjei Boakye E, Siegfried E. Characterization of children with recurrent eczema herpeticum and response to treatment with interferon-gamma. *Pediatr Dermatol*. 2017;34(6):686-689. [CrossRef]
- 20. Saleh D, Yarrarapu SNS, Sharma S. Herpes simplex type 1. In: *StatPearls* [Online]. StatPearls Publishing. 2021. Available at: https://www.ncbi.nlm.nih.gov/books/NBK482197/ [Ziyaret Tarihi; 27 Mart 2022].
- 21. Goldman RD. Acyclovir for herpetic gingivostomatitis in children. *Can Fam Physician*. 2016;62(5):403–404.
- 22. Drugge JM, Allen PJ. A nurse practitioner's guide to the management of herpes simplex virus-1 in children. *Pediatr Nurs*. 2008;34(4):310-318.
- 23. Pfaff F, Groth M, Sauerbrei A, Zell R. Genotyping of herpes simplex virus type 1 by whole-genome sequencing. *J Gen Virol*. 2016;97(10):2732-2741. [CrossRef]
- 24. van Oeffelen L, Biekram M, Poeran J, et al. Update on neonatal herpes simplex epidemiology in the Netherlands: a health problem of increasing concern? *Pediatr Infect Dis J*. 2018;37(8):806-813. [CrossRef]
- Mostafa HH, Thompson TW, Konen AJ, et al. Herpes simplex virus 1 mutant with point mutations in UL39 is impaired for acute viral replication in mice, establishment of latency, and explant-induced replication in mice, establishment of latency, and explant-induced reactivation. J Virol. 2018;92(7):e01654-17. [CrossRef]
- Wutzler P. Antiviral therapy of herpes simplex and varicellazoster virus infections. *Intervirology*. 1997;40(5–6):343–356.
 [CrossRef]
- Aoki FY, Law BJ, Hammond GW, Acyclovir-Gingivostomatitis Research Group. Acyclovir (ACV) suspension for treatment of acute herpes simplex virus (HSV) gingivostomatitis in children: a placebo (PL) controlled, double blind trial. Presented at 33rd Interscience Conference on Antimicrobial Agents and Chemotherapy, New Orleans, LA; 1993.
- 28. Gupta S, Sreenivasan V, Patil PB. Dental complications of herpes zoster: two case reports and review of literature. *Indian J Dent Res.* 2015;26(2):214–219. [CrossRef]
- 29. Martin JB, Beal MF. Disorders of the cranial nerves. *Harrison's Princ Intern Med.* 1998:2377–2380.
- 30. Hairston BR, Bruce AJ, Rogers RS 3rd. Viral diseases of the oral mucosa. *Dermatol Clin*. 2003;21(1):17–32. [CrossRef]
- 31. Makos C, Noussios G, Peios M, et al. Herpes zoster of the trigeminal nerve– two cases reports. *Int J Neurol*. 2010; 13(2):1–6.
- 32. Stoopler ET, Pinto A, DeRossi SS, Sollecito TP. Herpes simplex and varicella–zoster infections: clinical and laboratory diagnosis. *Gen Dent.* 2003;51(3):281–287.
- 33. Pocketdentistry.com [internet]. Update on oral herpes virus infections. Available at: https://pocketdentistry.com/update-on-oral-herpes-virus-infections/. (Accessed October 29, 2016)

- 34. Mendieta C, Miranda J, Brunet LI, Gargallo J, Berini L. Alveolar bone necrosis and tooth exfoliation following herpes zoster infection: a review of the literature and case report. *J Periodontol.* 2005;76(1):148–153. [CrossRef]
- 35. Klein E, Kis LL, Klein G. Epstein-Barr virus infection in humans: from harmless to life endangering virus-lymphocyte interactions. *Oncogene*. 2007;26(9):1297-1305. [CrossRef]
- 36. Mendoza N, Diamantis M, Arora A, et al. Mucocutaneous manifestations of Epstein-Barr virus infection. *Am J Clin Dermatol*. 2008;9(5):295–305. [CrossRef]
- 37. Zawar V, Chuh A, Sankalecha S. Erythema multiforme-like lesions in the course of infectious mononucleosis. *J Dermatol Case Rep.* 2009;3(3):44-46. [CrossRef]
- 38. Young LS, Yap LF, Murray PG. Epstein-Barr virus: more than 50 years old and still providing surprises. *Nat Rev Cancer*. 2016;16(12):789-802. [CrossRef]
- 39. Hoover K, Higginbotham K. Epstein-Barr virus. In *StatPearls* [Internet]. Treasure Island, FL: StatPearls Publishing. 2023.
- Vora SB, Englund JA. Cytomegalovirus in immunocompromised children. Curr Opin Infect Dis. 2015;28(4):323–329.
 [CrossRef]
- 41. Kotton CN, Kumar D, Caliendo AM, et al. Updated international consensus guidelines on the management of cytomegalovirus in solid-organ transplantation. *Transplantation*. 2013;96(4):333-360. [CrossRef]

- 42. Stone RC, Micali GA, Schwartz RA. Roseola infantum and its causal human herpesviruses. *Int J Dermatol.* 2014;53(4):397–403. [CrossRef]
- 43. Tesini BL, Epstein LG, Caserta MT. Clinical impact of primary infection with roseoloviruses. *Curr Opin Virol*. 2014;9:91–96. [CrossRef]
- 44. Vafaie J, Schwartz RA. Erythema infectiosum. *J Cutan Med Surg*. 2005;9(4):159–161. [CrossRef]
- 45. Weir E. Parvovirus B19 infection: fifth disease and more. *CMAJ*. 2005;172(6):743. [CrossRef]
- 46. Kumar KB, Kiran AG, Kumar BU. Hand, foot and mouth disease in children: a clinico epidemiological study. *Indian J Paediatr Dermatol*. 2016;17(1):7-12. [CrossRef]
- 47. Burns T, Breathnath SM, Cox N, Griffiths C. Rook's textbook of dermatology. 8th ed. Chichester: John Wiley & Sons, Blackwell Publishing Ltd. 2010.
- 48. Lauritano D, Moreo G, Oberti L, et al. Oral manifestations in HIV-positive children: a systematic review. *Pathogens*. 2020;9(2):88. [CrossRef]
- 49. Araújo JF, Oliveira AEF, Carvalho HLCC, Roma FRVO, Lopes FF. Most common oral manifestations in pediatric patients HIV positive and the effect of highly active antiretroviral therapy. *Cien Saude Colet*. 2018;23(1):115–122. [CrossRef]
- Nandi S, Mondal A, Roberts A, Gandhi S. Biosensor platforms for rapid HIV detection. Adv Clin Chem. 2020;98:1–34. [CrossRef]