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CASE REPORT

Striking Oral Manifestation of Primary Herpetic Gingivostomatitis: A Case Report and Review of Human Herpesvirus Infections

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Background: Human Herpesvirus (HHV) is a double-stranded DNA virus with a size of 120 to 260 nm. HHV is divided into three subclassifications including herpesvirus alpha, herpesvirus beta, herpesvirus gamma. The HHV group could infect the human body including the orofacial area with mild to severe symptoms. This case report will discuss the diagnostic approach by recognizing the characteristics of each HHV group infection.

Case Presentation: A 23-year-old male patient with complaints of mouth ulcers since 7 day, accompanied by pain when swallowing which was preceded by a fever. Extraoral examination showed vesicles, measuring 5 mm, multiple on the upper and lower lips. Intraoral examination showed multiple ulcers, measuring 5 mm on the left buccal mucosa, tongue, and gingiva accompanied by enlargement of the anterior gingiva and palatine rugae. The diagnosis was Primary Herpetic Gingivostomatitis, with herpangina considered as a differential diagnosis.

Case Management: Treatment included acyclovir tablets, nystatin suspension, multivitamins, petroleum jelly, and oral hygiene and dietary instructions. There was improvement in the vesicles on the lips and ulcers on the buccal mucosa, tongue, and gingiva within one week, though white plaque remained on the dorsum of the tongue.

Conclusion: Characteristics of oral manifestations caused by HHV infection include symptoms of fever, vesicles, multiple ulcers measuring less than 1 cm, and gingival enlargement as the basis for establishing a diagnosis of Primary Herpetic Gingivostomatitis related to herpesvirus alpha infection, although ulcers also appear in herpesvirus beta and herpesvirus gamma but usually the ulcers will persist for more than two weeks with a relatively larger size, not accompanied by vesicles, and no gingival enlargement.

Keywords: herpesvirus alpha, human herpesvirus, pathognomonic, primary herpetic gingivostomatitis, PHG

Introduction

Human Herpesvirus (HHV) is a double-stranded DNA virus that causes disease in humans. The virus varies in size between 120 and 260 nm and has a relatively complex structure.[1] Based on Taxonomy, human herpesviruses (HHVs) are divided into three subgroups. The first is the alpha herpesviruses, which include Herpes Simplex Virus-1 (HHV-1), Herpes Simplex Virus-2 (HHV-2), and Varicella-Zoster Virus (VZV, HHV-3). The second group is the beta herpesviruses, comprising Cytomegalovirus (CMV, HHV-5), Human Herpesvirus-6 (HHV-6), and Human Herpesvirus-7 (HHV-7). The last group is the gamma herpesviruses, which include Epstein–Barr Virus (EBV, HHV-4) and Kaposi's Sarcoma-Associated Herpesvirus (KSHV, HHV-8).[2,3] All HHV groups are associated with clinical symptoms in human, including in the orofacial region, with varying degrees of severity from mild to severe.[4] The risk factors for HHV infection are various, for example in cancer and autoimmune patients who are susceptible to HHV-1 to HHV-7.[5] Infections while in patients with Human Immunodeficiency Virus are susceptible to HHV-8 infections.[6]

The prevalence of HHV infection varies in each subclassification. The World Oral Health Organization (WHO) states that there are differences in the prevalence of the alpha herpes virus group. The prevalence of HSV-1

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reaches 3,800 million people from the total population in the world, in HSV-2 infection there are 520 million people globally have been infected, and 140 million people are infected with VZV worldwide.[7] Beta Herpesvirus Group infections also have their own prevalence, for example in CMV infection which reaches 59% of the population globally,[8] while for HHV-6 infection the percentage reaches 100% of adults have been exposed to the infection before,[9] and HHV-7 contributes 95% of the total population in the world.[10] The last group of HHV, namely gamma herpesvirus, also has an increasing prevalence every year, for example in EBV infection which globally shows a figure of 90% of people have been infected with the virus,[11] while the prevalence of KHSV mentioned by the Global Cancer Observatory (GLOBOCAN) reaches 35,813 cases worldwide.

Each group of human herpesviruses (HHV-1 to HHV-8) exhibits its own pathognomonic characteristics, including differences in lesion location and morphology. Nevertheless, they share common features, particularly viral latency and similar routes of transmission, which include body fluids such as saliva, sexual activity, shared cosmetics, direct mouth-to-mouth contact, and the use of common eating utensils.[12] Considering the epidemiology of the entire HHV family, the incidence of HHV-1 (HSV-1), including primary HSV-1 infection such as Primary Herpetic Gingivostomatitis (PHG), is relatively high. Although this infection predominantly affects children, it can also occur in adults.[13,14] PHG could generally heal on its own, but the symptoms of PHG would affect the quality of life of sufferers caused by lesions in the extraoral and intraoral areas, in addition this disease caused several complications such as meningitis encephalitis so that drug therapy is needed to suppress the infection. Drug therapy is usually in the form of antivirals, multivitamins are also provided as supportive treatment.[2,15–18]

Recognizing the characteristics or pathognomonic lesions of each HHV group provide to be one of the considerations of dentists to make a diagnosis in PHG patients, although ideally examinations such as PCR and ELISA methods are needed to confirm the diagnosis.[2] Given the paucity of this clinical presentation reported in the literature, documenting this case may provide valuable insights to improve early recognition of the distinctive features of PHG lesions with other HHV types, as well as the management of oral manifestations of PHG.

Case Report

First Visit

A 23-year-old man came to the Padjadjaran University Dental and Oral Hospital (RSGM Unpad) with complaints of difficulty swallowing, canker sores on the tongue, and scabs on his lips since 7 days before coming to the RSGM. There was a history of fever 5 days before the lesions appeared. To relieve symptoms, the patient took paracetamol tablets but there was still no improvement. There is no history of recurrent stomatitis, chickenpox and systemic diseases in this patient. Extraoral examination showed dry lips without exfoliation, accompanied by multiple vesicles on the upper and lower lips. The results of intraoral examination found ulcer lesions measuring approximately 5 mm on the left buccal mucosa, anterior tongue dorsum, left tongue lateral, left tongue ventral. Enlargement of the gingival margin area accompanied by erythema is also seen in the palatine rugae area, while in the dorsum of the tongue and upper anterior gingiva there is a yellowish white plaque that can be scraped off leaving a reddish area (Figure 1).

The diagnosis of the patient's oral cavity condition included Primary Herpetic Gingivostomatitis (PHG) and pseudomembranous type Oral Candidiasis. Due to limited access and rapid clinical resolution, laboratory confirmation was not performed. However, the diagnosis was based on classic pathognomonic features in line with established clinical criteria. Several topical and systemic drugs were given to the patient, such as anti-viral acyclovir taken 3×400 mg/day, anti-fungal with oral nystatin oral suspension 4x2ml/day, multivitamins 1x1/day and applying petroleum jelly to the upper and lower lips 3x1/day. Instructions to maintain oral hygiene, adequate hydration of at least 2L of water/day, adequate rest, and maintaining a healthy, balanced diet as non-pharmacological therapy. The patient was also instructed to self-isolate to prevent the transmission of infection and was advised to consult the periodontist department for calculus control and endodontist for caries evaluation.

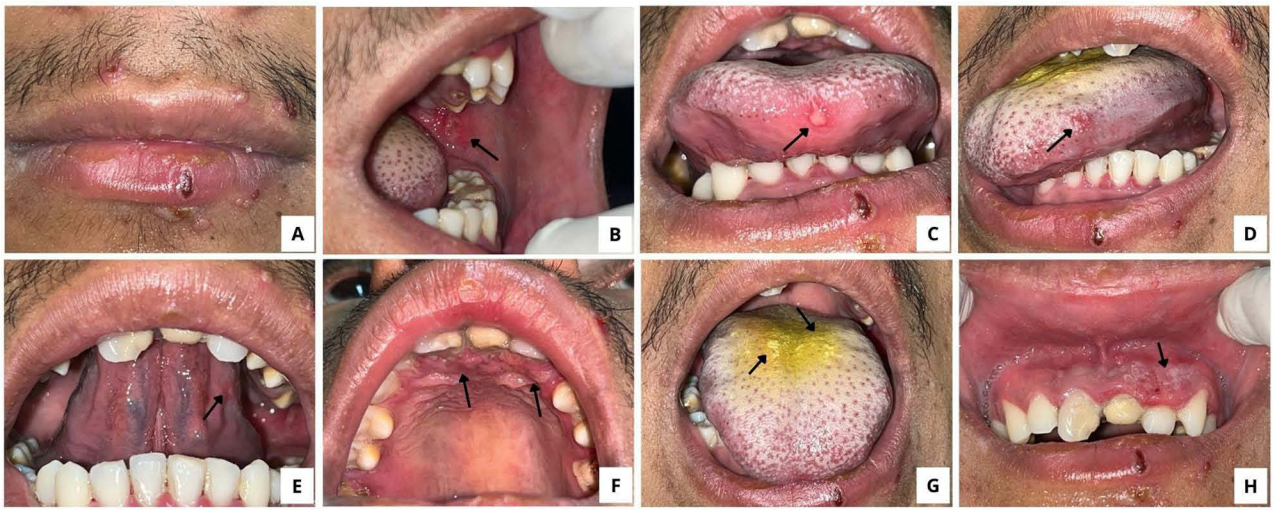

Figure 1 (**A**) Condition of the lips. (**B**–**E**) Ulcers are present on the left buccal mucosa, anterior dorsum of the tongue, left lateral tongue, and left ventral tongue (black arrows). (**F**) Enlargement of the palatine rugae area (black arrow). (**G**–**H**) White plaques on the dorsum of the tongue and upper anterior gingival mucosa (black arrows).

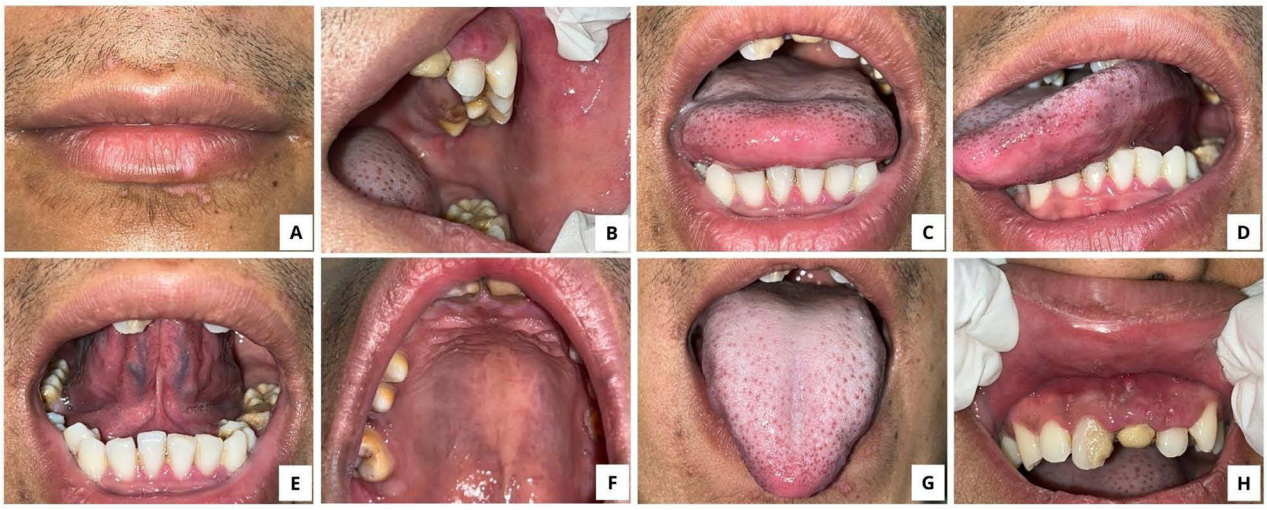

Figure 2 (**A**–**H**) There were no lesions on the lips, upper labial mucosa, left buccal mucosa, dorsum of the tongue, lateral tongue, left ventral tongue and palatine rugae after seven days treatment.

Second Visit

The 2nd visit was conducted 7 days after the first visit, the patient stated that after taking acyclovir, multivitamins and nystatin, the lesions in his oral cavity had improved and complaints of pain when swallowing were no longer present. Extraoral clinical examination did not find any vesicle or crust lesions, and no ulcer lesions or enlargement accompanied by erythema were found on the gingiva, but a layer of white plaque on the dorsum of the tongue was still visible (Figure 2). The dentist who treated the patient said that the patient should continue to maintain oral hygiene even though the previous complaints were gone.

Discussion

The case showed the presence of vesicle lesions in the extraoral, while in the intraoral there were ulcerations, erythema in the gingival area and white pseudomembranous plaques. Vesicle and ulcer lesions in the patient appeared due to primary infection of Herpes Simplex Virus 1 (HSV-1) which comes from the subclassification of herpesvirus alpha. The diagnosis

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of the patient is Primary Herpetic Gingivostomatitis (PHG), while the white pseudomembranous plaque lesions on the dorsum of the tongue and upper anterior gingiva appeared due to a decrease in the patient's immune system, causing oral candidiasis as an opportunistic infection. The emergence of oral candidiasis is also exacerbated by poor oral hygiene in patients.[19] In the research of Plotkin et al, it was stated that there was a supportive attachment to the biofilm between HSV and Candida *Sp* because both have the same receptors and both species can continue to be in the oronasopharyngeal area.[20] A disease typically presents with its own characteristic clinical signs and symptoms. In clinical practice, a feature that is uniquely associated with a disease is referred to as pathognomonic, as stated in the study of Koziol et al, which refers to the neurophysiology dictionary, that pathognomonic is a characteristic sign of a disease and is not found in other diseases.[21]

The characteristics or pathognomonic lesions of PHG in this case are located in the extraoral clinical picture in the form of vesicles, while the intraoral part is an ulcer on the left buccal mucosa, lateral tongue, and ventral tongue accompanied by enlargement of the gingival margin area. The PHG will continue to the latent life cycle stage and can be reactivated, causing the emergence of herpes labialis and recurrent intraoral herpes (Table 1).[22] PHG is usually accompanied by symptoms of fever, decreased appetite, and malaise.[23,24] From the clinical features and symptoms of the patient, this is in accordance with what was explained in the study of Huang et al, that patients with PHG infection will show symptoms of sore throat, vesicles will then burst and become ulcers on the oral mucosa including the gingiva, in addition there could be enlargement and redness to bleeding in the gingival margin area (Table 1).[25] The study by Coppola et al, also mentioned other characteristics of PHG which are preceded by prodromal symptoms including fever accompanied by headache, malaise, and muscle fatigue.[24] Vesicle and ulcer not only appear in HSV-1 infections but also appear in HSV-2 infections with different locations, namely on the lower extremities, especially the genital area such as the uvula, cervix, and vagina.[2,26] Vesicle also appear in patients infected with VZV. The characteristic difference lies in the vesicles that would be continue to become pustules that spread from the face to the upper and lower extremities. The lesions will then develop into crusts during the healing process (Table 1).[27] In general, the clinical features of HHV group infections shows similarities, for example vesicle lesions, ulcers, papular and reddish rashes, such as in the study of Pinana et al, which stated that Cytomegalovirus (CMV) infection has the main clinical manifestation of ulceration in the oral cavity and usually the lesions are more than 2 weeks, the ulceration appears wider, deeper and accompanied by indurated edge. Ulceration is usually painful, the number are able to single or multiple with a reddish.[28] CMV could also infect the retina, digestive tract, nervous system, lungs, and liver (Table 1).[8,29]

The next characteristic is HHV-6, this virus is divided into two subclassification, namely HHV-6 A and HHV-6 B. HHV-6A is usually associated with thyroiditis, autoimmune conditions Multiple Sclerosis, and hepatitis, while HHV-6B is associated with roseola infantum (exanthema subitum) in children, the main symptoms usually begin with fever to seizures accompanied by a reddish rash on the skin.[9,30,31] Reddish rash also occurs with HHV-7, but the rash is usually accompanied by non-itchy papules and the location of the lesion starts from the abdominal area then spreads to the neck and face. Other accompanying symptoms are anorexia, leukopenia, mild diarrhea, palpebral edema, mild inflammation of the pharynx, and occipital lymphadenopathy,[10] Other ulcerative lesions are also found in EBV infection, as described in

Table 1 Clinical Comparison Table of Oral Ulcers in HHV-1, HHV-3, and HHV-5

Virus	Oral Lesion Characteristics	Additional Features
HHV-1 (HSV-1)	Multiple painful vesicles that rupture into ulcers, mainly on gingiva, buccal mucosa, tongue, and lips. Often accompanied by gingival erythema and swelling.[24,25]	Prodromal symptoms: fever, malaise, sore throat. Can recur as herpes labialis or recurrent intraoral herpes.[22]
HHV-3 (VZV / Varicella-Zoster Virus)	Vesicles that evolve into pustules and crusts; intraoral lesions appear as painful ulcers on the palate, buccal mucosa, and tongue.[27]	Lesions spread from face to the trunk and then to the upper and lower extremities; may follow dermatomal distribution.[27]
HHV-5 (CMV / Cytomegalovirus)	Large, deep, persistent (>2 weeks) oral ulcers with indurated edges. Painful, single or multiple.[28]	Can involve other organs: retina, GI tract, lungs, liver, nervous system. Usually in immunocompromised patients.[28,29]

the study by Damania et al, which states that EBV has clinical characteristics in the form of ulceration in the mucocutaneous area including the oral cavity, oropharynx, skin and digestive tract. EBV is associated with several diseases such as Non-Hodgkin Lymphoma, Systemic Lupus Erythematosus, and Gastric Carcinoma.[11,32,33] HHV infection may also cause tumor lesions, such as in KHSV/HHV-8 infection which is vascular and endothelial cell neoplasia in patients with Human Immunodeficiency (HIV).[34,35] The clinical feature is in the form of tumors in the skin and oral cavity. The location of oral cavity manifestations varies including the tongue, lips, gums, oropharynx and buccal mucosa.[36] The appearance of vesicles, gingival inflammation and ulceration in these patients cannot be separated from the pathogenesis of the HSV-1 virus.

In general, the life cycle of this herpes virus is divided into two, namely the lytic cycle (productive) and the latent cycle (inactive). The primary herpesvirus certain to bind to cell receptors on the skin and mucous membranes, then produce glycoprotein gE/gL complex which to provide transport between virus cells and host cells which result in decreased synthesis of innate immunity and adaptive immunity in host cells, this results in an inflammatory response in the area of infection and causes tissue damage in the form of vesicle lesions which would be followed by ulcers on the mucosa.[37] This lytic cycle stage the virus provide express 80 gene products then activated the proteins needed in viral DNA replication.[38] The latency cycle indicates the success of the herpes virus in deceiving its host's immune system.[39] This virus also be latent in the trigeminal nerve ganglion could be reactivated if there is a trigger that stimulates it, for example stress, exposure to ultraviolet light, and fever.[40,41] Primary infection of HSV-1 usually targets non-keratinized mucosa, but it is possible that keratinized mucosa also be damaged. Unlike primary HSV1 infection, this secondary infection will usually affect more keratinized mucosa. HSV 1 virus can be latent in peripheral neurons beneath the surface of the mucosal epithelium that has been damaged in a previous primary infection process.[42]

The therapy given to the patient was acyclovir antiviral tablets, multivitamin tablets, nystatin oral suspension and petroleum jelly. Considerations in selecting the drug refer to several studies, one of which is a study by Hassan et al, which states that acyclovir could be interfere with the replication of HSV virus DNA with the help of the thymidine kinase enzyme will change the genetic code of the virus so that it produces false nucleotides with result in inhibition of HSV virus DNA synthesis.[43] The administration of nystatin oral suspension to the patient was based on considerations of candida infection characterized by pseudomembranous plaque on the dorsum of the tongue and the upper anterior gingiva of the patient, it was influenced by the patient not brushing his teeth because of pain in his oral cavity. Nystatin is a polyene antifungal that works by binding sterols in the fungal cell wall, resulting in increased cell permeability resulting in leakage of intracellular molecules.[44] Supportive therapy is also given, namely multivitamins that aim to increase the patient's immune system, with the aim of the patient's body condition has a defense against HSV virus infection and prevents further complications, the recommendation for using multivitamins is sufficient with 1x1/day,[45] in addition, the patient also given petroleum jelly to maintain the moisture of the lips and protect the skin from sunlight exposure to the lips.[46] Instructions to maintain oral hygiene and maintain a balanced diet are also given to the patient. The entire series of pharmacological and non-pharmacological therapies given to the patient gave satisfactory results.

Establishing a diagnosis of PHG disease caused by HSV-1 infection actually could be seen from the patient's anamnesis and clinical examination, such as in the study by Turton et al which stated that the characteristics of perioral vesicles, ulcers in the oral cavity including the gingiva, inflammation of the gingival margin area and starting with prodromal symptoms are very common symptoms in PHG without conducting supporting examinations.[47] The study by Lukisari et al used pathognomonic considerations and symptoms in PHG patients such as ulcers and gingival enlargement accompanied by prodromal symptoms to approach the diagnosis,[48] According to Nath P et al, primary herpetic gingivostomatitis (PHG) due to HSV-1 can be confirmed by PCR, noted for its high sensitivity, or by ELISA serology, which distinguishes between HSV-1 and HSV-2.[49] Based on theory, HSV-1 infection could be recured, usually after primary infection from the HSV-1 virus, it caused by secondary infections commonly known as reactivation which occurs due to certain triggers.[38] Various factors may act as triggers, such as physical or emotional stress, fever, exposure to ultraviolet light and hormonal changes,[50] this is one of the important knowledge that needs to be conveyed by dentists to patients so that patients provide to several preventive efforts by avoiding triggers.

Conclusion

The characteristic or pathognomonic oral manifestations caused by HSV-1 infection, namely Primary Herpetic Gingivostomatitis (PHG), include fever, the appearance of multiple vesicles and ulcers measuring less than 1 cm, and erythematous gingival margin enlargement. Understanding the distinctive features of each HHV group can assist clinicians in facilitating a potential diagnosis of HSV-1 infection, even in the absence of laboratory testing. The management provided in this case was guided primarily by the recognition of pathognomonic PHG lesions, which, despite the lack of laboratory confirmation, resulted in a favorable clinical outcome.

Consent Statements

The patient has approved and written informed consent for the publication of this case report, including the images. Universitas padjadjaran as the institution has also approved the publication of this article.

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Disclosure

The authors declare no conflicts of interest in this work.

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