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Case Report

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Beyond the Blister: A Case Report on Recurrent Herpes Labialis

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Abstract

The most prevalent viral infection in humans, herpes labialis is caused by the herpes simplex virus 1 (HSV-1). This disease is generally seen in childhood and adolescence. It is a contagious viral infection due to direct contact with active lesions and infected body fluids. It can affect the individuals who are immunocompromised like suffering from Human Immunodeficiency Virus (HIV) infection, undergoing chemotherapy and people who had history of oral herpes. The individuals with exposure to ultraviolet rays, fever, hormonal changes under stress can also become the trigger point for the recurrent infection. The present article reports a case of 60 years old female presented with persistent ulcers on her lower lip for 5 days followed by prodromal symptoms characteristically diagnostic of herpes labialis. She was treated by topical antiviral medication. Treatment is necessary with antiviral drugs therapy for reducing the significant pain and discomfort caused during the infection. Early detection and management is of utmost importance to reduce symptoms and avoid further complications. This case report provides valuable clinical insights into the diagnosis and management of recurrent herpes labialis (RHL), a common but often ignored oral viral condition. Also, it emphasizes the efficacy of topical acyclovir and the supportive role of multivitamin therapy in enhancing immune response and tissue healing.

Keywords: acyclovir, antiviral therapy, herpes simplex virus (HSV), immunity, stress

Introduction

Herpes simplex virus type 1 recurrent oral HSV-1 infection causes the common oral mucosal disorders including acute herpetic gingivostomatitis (AHG) and herpes labialis (HL). HL, commonly known as cold sores. This is a condition characterized by the formation of multiple blisters on the vermilion border of the lips or the surrounding skin.[1,2] HSV is highly contagious and usually occurs in childhood. It is highly prevalent viral disease worldwide. Many of the affected patients are asymptomatic. Despite of its majority, prevalence virus remain dormant in the body (latent) and has potential for reactivation. This reactivation can be triggered by ultraviolet rays (sunlight), fever, hormonal changes under stress. Therefore, it is of major concern to prevent the recurrence. Antiviral drugs like acyclovir, famciclovir can be used to manage acute episodes and its recurrences.[3] The present article discusses an occurrence of HL accompanied by AHG in a 60-year-old woman, who was effectively treated with a topical antiviral medication. It is done by evaluating and managing symptoms and preventing future episodes, contributing to a better understanding of the multifaceted treatment strategies for HL. Public awareness and education about the virus and its transmission can also help to reduce the spread of HSV-1 and decrease the stigma associated with the condition.

Case description

A 60-year-old woman presented to the Department of Oral Medicine and Radiology with a chief complaint of persistent ulcers on her lower lip for 5 days. She reported that her symptoms began with a fever and vesicles on lower lip and in oral cavity, which subsequently developed into ulcers. Within 3 days, most of the intraoral vesicles had ruptured. The patient reported a history of occurrence of similar lesions twice in the past, approximately 1 month and 3 months prior, which resolved within a week without treatment. Notably, there was no history of similar lesions on other parts of the body, including the genitals, eyes, or skin. Her medical history revealed a 4-year history of hypertension, for which she was currently receiving medication and her blood pressure is now normal. The patient's family history was non-contributory. All of the vital signs were found to be within the normal range during the general physical examination. Lymph node assessment showed no evidence of regional lymphadenopathy.

Extraoral examination revealed multiple haemorrhagic crusted ulcers on the lower lip and vermilion border with erythematous base, measuring approximately 1 x 0.5 cm in size, with a roughly oval shape. There were 5-6 ulcers in total, characterized by well-defined borders and sloping edges. Ulcers were mildly tender on palpation and no bleeding or pus discharge seen on manipulation (Figure 1).

Figure 1: Multiple haemorrhagic crusted vesicles and ulcers on lower lip and vermillion border.

Intra oral examination showed adequate mouth opening and no abnormalities were detected on soft tissue examination. The marginal and attached gingiva in lower arch was observed to be swollen, bright red, and highly tender, with bleeding on probing, consistent with a diagnosis of acute gingivitis (Figure 2). Acute gingivitis is managed through the removal of local irritants that includes scaling and polishing to eliminate plaque and calculus, followed by patient education on oral hygiene practices. Also the antimicrobial mouth rinses, chlorhexidine gluconate 0.12-0.2% was advised for 7days to reduce bacterial load and inflammation.[4]

Based on the patient's history and clinical presentation, a provisional diagnosis of recurrent herpes labialis (RHL) was made, and the patient was subsequently treated with antiviral medication. Topical 5% acyclovir cream was prescribed, to be applied five times a day for 7 days to the affected area. The patient was also prescribed vitamin B complex supplements for 7 days, and the lesion resolved completely within a week. At the follow-up visit seven days later, the lesions had completely resolved (Figure 3). The patient received counselling about her condition, as well as preventive measures regarding recurrence to help avoid HSV infection.

Figure 2: Swollen bright red marginal and attached gingiva in lower arch.

Figure 3: Showing healed lesions after 7 days.

Discussion

The HSV-1 virus will penetrate the mucosal surface and be transmitted through oral secretions, by direct contact with contaminated saliva or respiratory droplets. HSV enters host cells through the interaction of several glycoproteins on the surface of the viral envelope with host cell surface receptors. Glycoprotein B (gB) in the virus will bind to the heparin sulfate (HS) receptor on host cells so that the HSV-1 virus will settle in the dorsal ganglia, especially the trigeminal nerve ganglion and can be reactive if there is a trigger.[5] This form of reactivation of the HSV-1 virus can be triggered if there is a decrease in immunity due to changes in weather, fever, sun exposure, emotional stress, trauma, menstruation, systemic diseases, allergies, and immunosuppression. Recurrent lesions in immunocompromised patients are often more aggressive and take an extended period to heal than normal patients.[6] In this case report, it is suspected that the trigger factor for HSV infection might be reduced immunity with ageing.

The trigger factor becomes a stimulus in the sensory nucleus where the virus is dormant in the latent phase, both in the central and peripheral nerves. When virus reactivation occurs, sufferers usually feel tingling, itching and erythema in the affected area, whereas secondary infection often occurs in the labial area or outside the vermilion border. The next phase is the inflammatory phase which usually occurs on the first day, the virus begins to produce and infect nerve endings. The cells will react to the invasion of the virus which is characterized by erythema. On the second to third day there is a pre-afternoon phase which is characterized by the appearance of papules and vesicles that feel itchy and sensitive to touch.[7]

About 16% and 38% of people are affected by RHL.[1] RHL is usually diagnosed based on the patient's medical history and clinical presentation. One of the interesting or incomparable presentation in this case was bilateral involvement. Nevertheless, it is well known that RHL typically presents as unilateral lesions, often on the vermilion border of the upper or lower lip. The possible explanation for this bilateral involvement could be a wider distribution of viral reactivation across bilateral nerve branches, a compromised immune status due to aging, or a higher local viral load.[8] Although the lesion showed characteristic clinical feature such as confinement to the lower lip and vermillion border, differential diagnosis was considered to rule out other possible ulcerative or vesiculobullous conditions with overlapping clinical features. Similar presentations can occur in conditions such as aphthous stomatitis, erythema multiforme, herpangina, and varicella zoster infection. However, aphthous ulcers typically appear on non-keratinized mucosa (e.g., buccal mucosa, labial mucosa), and lack the prodromal symptoms seen in herpes. Erythema multiforme often involves the entire lip with characteristic target lesions on the skin,

which were absent. Herpangina presents with ulcers in the posterior part of the oral cavity. Similarly, Varicella zoster virus (VZV) infection typically shows a dermatomal, unilateral distribution and may involve skin and mucosa simultaneously. In contrast, this case showed clustered, crusted ulcers localized to the lower lip, preceded by prodromal signs, and a rapid therapeutic response to topical acyclovir, all of which are characteristic of RHL.[8,9]

Laboratory tests such as the tzanck test, immunofluorescence, and serological assays for anti-HSV IgM and IgG can be used to confirm the diagnosis during times of uncertainty. However, viral culture remains the gold standard for confirming the diagnosis as it isolates live virus for strain identification and confirms active infection. It is also useful for performing antiviral sensitivity testing when resistance is suspected.[10]

Most cases of herpes simplex infection resolve on their own. However, antiviral therapy provides substantial clinical benefits for most symptomatic patients and is the cornerstone of treatment. The three antiviral medications commonly used to manage symptomatic HSV infections are acyclovir, valacyclovir hydrochloride, and famciclovir.[1,11] Acyclovir has been demonstrated to be safe for prolonged suppression of RHL and genital herpes infections, despite the availability of other antiviral drugs. Acyclovir systemically at a dosage of 5 mg/kg to 10 mg/kg is the suggested regimen; this should be given every 8 hours for 2 to 7 days, or until clinical improvement is observed. After this oral antiviral therapy should be given to ensure a complete treatment duration of at least 10 days.[11] Acyclovir administered systemically reduces pain and accelerates the healing process and resolution of viral shedding. Lesion location and the infection history (primary or recurring) determine the dosage and duration of the therapy. It is critical to understand that instances of HSV encephalitis and serious HSV infections in people who are immunocompromised require high-dose intravenous acyclovir, which is frequently started therapeutically.

In the present case, therapy was in the form of causative and supportive care using topical antivirals, namely 5% acyclovir cream and multivitamins. The patient was treated with topical 5% acyclovir cream, applied four times a day for seven days. Starting treatment early, ideally within 48 hours of symptoms, helps to speed up healing and reduce discomfort. Because the patient's general condition was good, for the treatment of infection in this case it was enough to use topical drugs that are applied to the lesion area.[12] Acyclovir first breaks down to acyclovir monophosphate by viral thymidine kinase which is found in viruses. Cellular guanylate kinase then transforms this to acyclovir diphosphate, then phosphoglycerate kinase, phosphoenolpyruvate carboxykinase, and pyruvate kinase finally convert it to acyclovir triphosphate. Acyclovir triphosphate is the cause of the inhibitory effect of viral DNA polymerase and viral replication of genomes. acyclovir triphosphate blocks DNA synthesis by serving as a chain terminator. Since acyclovir selectively inhibits normal cellular thymidine kinase and has minimal cytotoxicity, it cannot be efficiently used as a substrate by uninfected cells. Patients with hypersensitivity, particularly those with renal failure, immunocompromised hosts, possible risk of thrombotic thrombocytopenic purpura (TTP) should not use acyclovir.[13]

In this case report, it is suspected that the trigger factor for HSV infection might be reduced immunity with ageing. In this case, the most probable trigger factor for the RHL was age-related immunosenescence, as the patient was 60 years old. With advancing age, the immune system undergoes functional decline, leading to decreased surveillance and reactivation control of latent viruses like HSV-1.[14] Immunocompromised individuals, such as those with Human Immnodeficiency Virus (HIV), are at higher risk of severe HSV infections and may require systemic or intravenous antivirals. Since acyclovir is primarily excreted by the kidneys, monitoring renal function is essential to avoid potential nephrotoxicity.[15]

Host immunity is a key factor in viral infection. Improving the immune system, both natural and adaptive, can be achieved by improving nutrition, improving psychological conditions and administering multivitamins. Multivitamin Becom-zet® which consist of vitamin B complex, vitamin C, E and zinc which acts as a catalyst and regulator of biochemical reactions in the body. Vitamin B is a water-soluble vitamin that serves as a cofactor and coenzyme, playing a significant role in the immune response by supporting the function of CD8 T cells and natural killer (NK) cells. It can increase the patient's immune system through adequate intake of the vitamins. Multi vitamins needed and prevent the occurrence of functional metabolic disorders that cause reduced vitamin

intake also accelerates the change of proline and lysine residues in procollagen to hydroxyproline and hydroxylysine in collagen synthesis.[12,16] This process plays a key role in speeding up the healing process. Patients are also advised to manage stress and adequate rest and maintain nutrition and hydration to prevent recurrence of herpes labialis.

Conclusions

The dentist plays a critical role in the comprehensive management of RHL. From early diagnosis and accurate differentiation of lesions to the initiation of appropriate antiviral therapy and patient education, their involvement ensures timely and effective management. By addressing potential triggers, advising on preventive strategies, and providing supportive care, dentists help to reduce the frequency and severity of recurrences.

Clinical significance

Early recognition and prompt antiviral treatment of recurrent herpes labialis can significantly reduce lesion severity, healing time, and patient discomfort, emphasizing the importance of timely clinical intervention.

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