Herpes labialis: A case series of 12 patients

Tejavathi Nagaraj1, Lakshmi Balraj1, Haritma Nigam1, Sida Tagore2

1Department of Oral Medicine and Radiology, Sri Rajiv Gandhi Dental College and Hospital, Cholanagar, Bengaluru - 560 032, Karnataka, India, 2Department of General Pathology, Sri Rajiv Gandhi College of Dental Sciences & Hospital, Bengaluru, Karnataka, India

Abstract
Herpes labialis is a common viral infection affecting man worldwide. The infection is known for causing blisters and sores on the lips, tongue, or gums. Soon after an initial infection, the virus has a tendency to remain in a dormant state within the nerve cells of the face. Although recurrent herpes labialis is not quite serious, the relapses are common. An effective management program is hence needed to curb the spread of this contagious disease among larger masses. We report a series of 12 cases of herpes labialis in adults over a period of 1 year. The patients were prescribed with antiviral agents and were advised against any treatment during the active period of the disease and were followed up on a regular basis. This article has highlighted the approach to patients suffering from herpes labialis and outlines preferred treatment modality to be adopted by the practitioner in each situation.

Keywords: Herpes labialis, labial mucosa, infections

Introduction
Herpes simplex virus (HSV), an acute infectious disease is one of the most common viral diseases affecting man, involving principally tissues; those are derived from ectoderm and consist of eyes, skin, mucous membrane as well as the central nervous system. There are immunologically two different types of HSV; these include Type 1 which usually affect the face, lips, oral cavity and upper body skin and Type 2 which affects the genitals and lower body.1 HSV-1 affecting primarily the lips, termed as herpes simplex labialis (HSL), continues to remain a global public health issue for which various treatment modalities have been tried but with minimal success.

HSL primarily is seen affecting preschool or kindergarten children, adolescents, and young adults.2 Recurrences of HSL manifesting as cold sores can continue throughout adulthood. Typically, the primary infection is more severe than the recurrences; viral shedding is observed to be maximum in the initial episode though the amount of virus shed appears not to be related to the severity of the attack.3 The patient usually experiences constitutional symptoms such as general malaise, weakness, fever, and loss of appetite; along with sudden onset of multiple intraoral vesicles that rupture quickly, leaving painful ulcers. Consequently, the virus ascends the sensory nerve axons after the primary infection and establishes a chronic, latent infection in various ganglia. These may include trigeminal, facial and vagus ganglia.4

Evidence has been established on latent infection frequently developing over the epithelium of the lips.4 There is a need for “trigger” to reactivate the dormant virus. These may include physical sun exposure, menstruation, psychological stress, trauma, illnesses, and immunosuppression. The trigger activates the dormant virus which then soon begins to replicate, finally leaving the ganglion and traversing along peripheral nerves. This results in the formation of vesicles along the course of the nerves at the specific mucosal sites. Repeated viral attacks involving other branches of a single neuron can result in larger lesions by coalescing smaller vesicles.5

This article presents a series of 12 cases of herpes labialis recorded over a period of 1 year.

Patients and Methods
This study consisted of patients who had reported to Oral Medicine and Radiology Department of Sri Rajiv Gandhi College of Dental Sciences and Hospital, Bengaluru, with different chief complaints. Among all the patients who had reported to the department over a period of 1 year (March 2015-March 2016); herpes labialis occurred as an incidental finding in 14 among them.
Detailed case histories of these patients were recorded. All of them shared a similar history of fever following which fluid-filled blisters had occurred in and around the lips. These lesions were preceded by mild intermittent fever, general weakness, and malaise. Some of them also experienced slight tingling or itching sensation over the affected region. Case history was then followed by a thorough clinical examination.

These patients had presented with encrusted lesions over the lips since about 5-6 days. Clinical examination revealed clusters of ruptured vesicles ranging from 0.5 to 2 mm in size, roughly ovoid in shape, present over various site on lips and labial mucosa [Figure 1]. These appeared ulcerated and most of them are covered by a brownish crust. Some were tender on palpation.

These lesions limited the maintenance of oral hygiene and proper intake of food, but intravenous rehydration was not necessary. No regional lymph node involvement was found except in one of the patients in whom solitary submandibular lymph node was palpable which was mobile and soft in consistency. Based on the history and clinical examination, a provisional diagnosis of herpes labialis was given for these patients.

Routine investigations including hemoglobin count and complete blood count were done, which showed values within normal limits. Confirmation was done by taking smears from the lesions which were in the healing phase and subjected to Tzanck test which showed multinucleated giant cells.

The patients were treated with a 7-day course of 4 times application of topical acyclovir (ointment 5%) and were put on periodic follow-up. The lesions resolved completely and uneventfully within 7-8 days.

Discussion

Reactivation of HSV-1 is the major cause of herpes labialis. This produces virions which are active virus particles. Virions spread from infected neurons by anterograde axoplasmic transport to various mucocutaneous sites causing recurrent herpes infection [Figure 2].

Earlier, it was believed that infectivity was closely tied to clinical evidence of infection. However, viral reactivation does not always result in clinical disease like primary infections. However, many studies have been conducted and now it has been documented that the virus is shed asymptomatically by most of the population at least once a month. It is also stated that many individuals shedding the virus roughly more than six times a month contribute to the dissemination of HSV-1 infection. The mean duration of viral shedding was reported to range from 1 to 3 days, though viral shedding for periods longer than 3 days has been observed in about 10% of the patients.

Clinical features

The initial latency is between 2 and 10 days. In primary infection, localized aggregation of small herpetic blisters is observed. It may occur on any site of the body, particularly the lips, genitalia and fingers. In severe cases, small blisters spread on the whole body.

After the initial primary infection by HSV-1 involving the oro-facial region, 3 well-recognized sequelae resulting from reactivation of HSV from latency have been identified which include:

1. Recrudescent herpes labialis
2. Asymptomatic shedding of HSV-1 in saliva
3. Localized intraoral recrudescences.

Christie et al. reported fourth sequelae that of recrudescent intraoral HSV infection simulating primary herpetic gingivostomatitis.

HSL commences with prodromes such as pruritis or itching and discomfort in the lips and their periphery, including the anterior naris cheeks and orbital region. After
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During the first day or two, edematous erythema appears and small blisters with central umbilication occur and aggregate, sometimes coalescing to form irregularly shaped blisters. Consequently, the blisters form pustules, erosions, and crusts. They heal in about 1 week.

**Histopathology**

Repetitive replication of viral DNA leads to ballooning degeneration and reticular degeneration of infected epidermal cells. These are observed as ballooning cells containing intranuclear inclusion bodies by smear staining of the blister contents [Figure 3].

**Laboratory diagnosis**

Tzanck test, detection of the virus using monoclonal antibodies and serological diagnosis are conducted. HSV-infected epidermal cells are easily and quickly observed by Tzanck test. Monoclonal antibody detection is conducted to differentiate between HSV-1 and 2 as well as varicella zoster virus. Serological diagnosis is made by enzyme-linked immunosorbent assay.

**Management**

Preventive therapy commonly instituted consists of a sun block or an antiviral drug; considered as the mainstay for patients who experience frequent recurrences [Table 1]. Such therapy could result in suppression of an individual patient’s response to a specific trigger. However, to prevent the chances of development of a drug-resistant viral strain, this therapy is not recommended for patients who experience just 1 or 2 lesions annually. Acyclovir cream, which is a topical antiviral formulation available over-the-counter (OTC); is used by most of the patients. Delays related to obtaining prescriptions which are required for topical famciclovir and systemic use of acyclovir or valacyclovir has resulted in the use of these OTC formulations in a large scale.

**Conclusion**

The probable diagnosis of HSV infection usually is based on the patient’s medical history, symptomatology, and clinical findings. However, laboratory confirmatory tests may be required when the clinical features are atypical or when patients are immunocompromised. Many patients are hesitant to consult their oral physicians rather tend to resort to OTC medication. Although orolabial herpetic infections are usually self-limiting, successful management of such cases is necessary.

**Clinical significance**

This clinical trials-based study has highlighted the process by which 12 patients were diagnosed of herpes labialis, and their consecutive treatment plan and periodic follow-up. This study can be added for further such prospective voluminous clinical trials related to HSV.

**References**

7. Miller GS, Danaher RJ. Asymptomatic shedding of herpes...