Two Cases of Recurrent Herpetic Infection of the Tongue

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Summary: Recently, a number of reports have been published on recurrent herpetic infection of the oral mucosa. In most of these cases, the infected tissue is the fixed intraoral mucosa, such as the gingiva or hard palate. Infection of movable mucosa such as the tongue, which is reported in the present paper, has not been reported in detail previously. In each of the two cases reported in the present paper, intraoral lesions were diagnosed as recurrent herpetic glossitis after isolation of the herpes simplex virus (HSV).

Key words herpes simplex virus type-1, recurrent herpetic infection, tongue, aphtha, movable mucosa

Introduction

Intraoral infections with herpes simplex virus type-1 (HSV-1), such as the herpetic gingivostomatitis and the herpetic labialis, were well-documented as primary and recurrent lesions, respectively (Pindborg, 1973). Since Griffin (1965) identified recurrent herpetic infection of the oral mucosa, many such cases have been reported (Weathers and Griffin, 1970; Southam, 1980; Kabashima, 1984; Kameyama, 1987). However, in the vast majority of cases, herpetic lesions were principally localized on immovable mucosa, such as the attached gingiva and hard palate. Only one report has described a herpetic lesion on the tongue (Kabashima, 1984).

In the present report, we present two cases of recurrent herpetic infection on the tongue, and discuss these cases within the context of previous literature.

Case Report

Case 1: A 22-year-old woman visited our clinic with soreness localized on the left dorsal side of the tongue. Examination revealed a 6×5 mm, irregularly-shaped lesion on the left dorsal side of the tip of the tongue. The lesion was nearly circular in shape, had slightly irregular margins, and was covered with a gray pseudomembrane. In addition, two small like erosions, one measuring 2 mm and the other measuring 1×2 mm in diameter, were identified at the lateral margin of the tongue and in the center of the dorsal side of the tongue, respectively. Soreness was also noted at these erosions. No erosion or ulcer was identified on other oral mucosa, and the teeth had no sharp edges that could have been responsible for the erosions (Fig. 1). No notable systemic abnormalities were observed, except for a mild fever of 37.4 °C. Related lymph nodes were not swollen. The patient stated that
she had a fever of 39°C 4 days previously, and subsequently developed a headache, nasal stuffiness, and a tingling pain at the center of the dorsal side of the tongue. One day previous to the visit, the erosions appeared in the region extending from the tip to the left dorsal side of the tongue. The patient stated that she had no history of the herpes labialis, but that her mother had the condition. After a diagnosis of the herpes-glossitis, acyclovir was administered. In addition, a steroid drug was topically applied to the erosions, and a gargle was prescribed. Four days later, the erosions had reduced in size, and the soreness had nearly disappeared. Both the erosions and the soreness had completely disappeared when she visited us again 11 days later.

Case 2: A 45-year-old woman came to our clinic for examination of contact pain on the left dorsal side of the tongue. A cluster of vesicles with erythema was identified on the left dorsal side of the tongue, and soreness was noted (Fig. 2). Other oral mucosa showed no erosions or ulcers. No systemic abnormalities were observed. The patient stated that she had no previous history of the herpes labialis. Under a tentative diagnosis of the herpes-glossitis, acyclovir drug for topical application was prescribed. Although the patient was to be observed, she did not appear at our clinic again, and the outcome of her condition is unknown.

Materials and Methods

Specimens of both cases was collected on the day of the visit to the clinic by wiping the lingual ulcer with a sterile periostial elevator. The saliva was suspended in 2 ml phosphate buffered saline (PBS) supplemented with 4% bovine serum. Serum was also obtained on the first day, in order to test for the presence of HSV-1 antibody.

Cells: GMK (green monkey kidney) cells cultured in Eagle's minimum essential medium (MEM) supplemented with 10% bovine serum were used for the isolation and assay of the virus, and for antibody titration.
Isolation of the virus: Specimens were centrifuged at 3,000 rpm for 10 min. One milliliter of supernatant was inoculated onto the GMK cells, and incubated at room temperature in a stationary rack for 2 hs. After washing twice with Hank's balanced salt solution, fresh MEM supplemented with 2% bovine serum was added after the inoculum was removed. The cultures were incubated at 37 °C, and checked for cytopathic effect (CPE) daily for 1 week. CPE characteristics of HSV were observed, and the culture was frozen.

HSV type specificity: Samples showing CPE were identified as HSV-1 or 2 using a neutralization assay with guinea-pig antisera specific for HSV-1 and 2. Ten-fold serially-diluted virus samples were mixed with equal volumes (0.5 ml) of HSV-1 (at a dilution of 1:25) or HSV-2 (1:70) specific antisera. After 1 hour incubation at 37 °C, the mixture was coated onto GMK cells for identification of type specificity.

Titration of serum antibody: Serum from each patient was examined for HSV-1-neutralizing antibody titer using a plaque assay. Heat-inactivated (56 °C, 30 min) serum was serially diluted from 1:10 to 1:640; 0.5 ml diluted serum was mixed with an equal volume of virus suspension (HSV-1 strain YH) containing 1,000 PFU/ml. After 1 hour of incubation at 37 °C 0.2 ml of the mixture was coated onto GMK monolayers for the plaque assay. After 4 days of incubation at 37 °C the cells were stained with crystal violet, and the number of plaques was counted. The highest dilution of serum that reduced plaque formation by 50% was taken to be the neutralizing antibody titer (Elion, 1977).

Results

Viruses isolated from both cases were completely neutralized using HSV-1 specific antiserum, but not using HSV-2 specific antiserum. Thus, the isolates were identified as HSV-1. The neutralizing antibody titer in case 1 was 640, as confirmed in 2 trials. The neutralizing antibody titer in case 2 was 160 (Table 1).

Based on these results, both cases were diagnosed as recurrent herpetic infection on the tongue.

Discussion

Recurrent HSV lesions on the oral mucosa were first reported by Griffin (1965), who identified four such cases using cytodiagnosis and a fluorescent antibody method. Since then, Weathers and Griffin (1970), Southam (1980), and Kameyama et al. (1987) have also reported cases of intraoral HSV infection. In each of these reports, the site of infection was either the gingiva or the hard palate.
Thus, HSV infection of the movable oral mucosa has not yet been reported, with the exception of a study by Kabashima (1984), in which one case of HSV infection on the movable oral mucosa was briefly recorded without detail.

Due to the rarity of HSV infection of the movable oral mucosa, local development of the lesion on the fixed oral mucosa has been considered to be a definitive characteristic of recurrent intraoral HSV infection. However, since the stimulation of nerve terminals has experimentally been proven to influence the reactivation of HSV (Openshow et al. 1979), limiting the site of recurrent HSV infection only to the intraoral fixed mucosa lacks rationale.

Although a typical characteristic of recurrent HSV lesions on the intraoral mucosa is a cluster of small (1 mm in diameter), shallow, circular, red erosions, the recurrent lesions identified on the tongue in the present study presented somewhat differently. This may be due to the high mobility of the tongue, which crushes the blisters into an erosion at a very early stage, naturally resulting in a secondary infection that follows the anatomical shape of the lingual papilla. This process has been described by Kameyama et al. (1987) as follows: lesions on the gingiva or palatal mucosa change over time. At an early stage, the lesion features a clustery, small, circular erosion, gradually fuses into a gourd-shaped erosion, and finally develops into an irregularly-shaped erosion. In the case of the tongue, the same process is thought to occur more rapidly due to high mobility. In the present study, observation revealed irregular margins of the erosion, indicating the fusion of small circular erosions. This feature may provide an important clue for diagnosis.

Although neither of our patients had a previous history of the herpes labialis, the herpes-glossitis was diagnosed in case 1 because three circular erosions were separately and distinctly located on the tongue, the titer of neutralizing antibody collected as the pair serum showed no change, and HSV was isolated from the erosion surface. In case 2, although only one relatively large conglomerate vesicle was observed, the herpes-labialis was diagnosed because the margins of the erosion were irregular, indicating the fusion of many small circular erosions. Herpes infection was highly suspected due to previous medical history, and was confirmed after HSV isolation.

For accurate diagnosis of intraoral local mucosal infection with herpes simplex virus, the possibility of other mucosal diseases must be eliminated.

In order to diagnose herpetic infection, we believe that isolation of HSV from intraoral lesions is conclusive. However, since the isolation of HSV involves a complicated procedure that takes at least 24 hs, a faster and more reliable diagnostic method is desirable.

References
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