Case report

Primary Herpetic Gingivostomatitis in an Adult Patient: A Case Report

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Abstract: Acute Herpetic Gingivostomatitis represents the main pattern of primary infection with herpes simplex viruses. Although the virus exists in eight various forms, herpes simplex virus type 1 causes most of the oral infections. Acute Herpetic Gingivostomatitis typically affects children, but this infection also occurs rarely in adults. Proper diagnosis and treatment are essential. This article presents an acute episode of primary herpetic gingivostomatits in an adult patient with emphasis on clinical features and management.

Keywords: Acute herpetic gingivostomatitis; Herpes simplex virus; Stomatitis; Viral infection.

INTRODUCTION

As rightly said, oral cavity is the mirror of the body. Various lesions of oral cavity might be the expression of some systemic disease or infection, whereas others might just have local etiology. In either of the cases, diagnosing the proper etiology is the most important part, so that the proper treatment could be given to the patient. Various lesions covering the exposed part of the gingiva, along with the rest of the mucosa, are seen. One such infection is acute herpetic gingivostomatitis (AHGS).

This infection is very commonly misdiagnosed as allergic stomatitis, apthous ulcers, erythema multiforme, but retrieving the correct detailed history from the patient and correlating it with the clinical features, can help us to reach to the correct diagnosis. The causative agent for acute herpetic gingivostomatitis has been identified as Herpes Simplex Virus (HSV). The tissue preferentially involved by herpes simplex virus, now often referred to as herpes hominis are derived from ectoderm and consist principally of skin, mucous membrane, eyes and central nervous system.[1]

There are 80 known herpes viruses, and eight of them are known to cause infection in humans. These include, herpes simplex virus (HSV 1 and 2), varicella-zoster virus (HHV-3), Epstein-Barr virus (HHV-4), cytomegalovirus (HHV-5), and human herpes virus 6 (HHV-6), HHV-7 and HHV-8.[2,3] All herpes viruses contain a deoxyribonucleic acid (DNA) nucleus and can remain latent in host neural cells, thereby evading the host immune response. HSV is a double-stranded DNA virus and is a member of the human herpes virus (HHV) family officially known as Herpetoviridae.[4, 5,6] These are two immunologically different types of HSV: type 1 – usually affecting the face, lips, oral cavity and upper body skin and type 2 – usually affecting the genitals and skin of the lower body.[1]

Herpetic gingivostomatitis is a contagious disease.[7] It is very common in patients under 10 years of age, even though it also affects both teenagers and adults, in a lower frequency.[8] Its transmission is through direct contact with lesions or through saliva.[8] The clinical characteristics are preceded by a prodromal state which includes: fever, general discomfort, asthenia, lymphadenopathies in some cases, dysphagia and irritability.[9]

Clinically, the most frequently found lesions are: very specific buccal ulcers, small, round, preceded by vesicles, which, in most cases, are not perceived, because they break down a few hours after their apparition. They are principally localized in the keratinized buccal mucosa (gum, hard palate and lingual dorsum). [10] In some cases, the immunologic state of the patient plays an important role. Because, the lesions can spread and complicate the clinical picture and management, it is necessary to investigate about the immune-suppression in any patient with an infection by herpes simplex.[11]

CASE REPORT

A 32 year female patient reported to the department of Periodontology with the chief complaint of sore mouth, burning sensation and inability to eat. Medical history revealed that the patient had fever 3 days back. The

patient had not taken any medical or dental treatment for the problem. Family history was non-contributory. A thorough history revealed that the lesions began as vesicles that burst rapidly. On extra oral examination, sub-mental and sub-mandibular lymph nodes were soft, mobile, palpable and tender, bilaterally. On intraoral clinical examination, fiery red, inflamed, erythmatous, extremely tender gingiva was seen. Lesions were more prominent in the maxillary and mandibular anterior teeth region. Marginal and palatal gingiva was covered with yellowish-white, pseudo-membranous slough. Gingival bleeding was noted even on slight provocation. According to the patient, no such episodes had occurred before. Correlating the history of illness and clinical evaluation, the diagnosis of Acute herpetic gingivostomatitis was made.

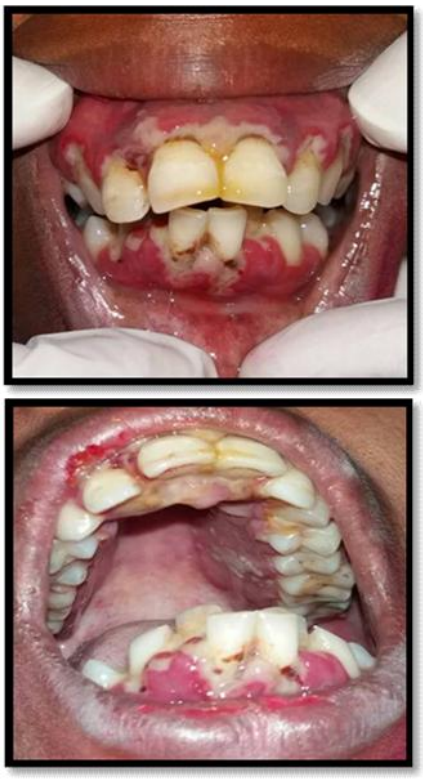

Figure1 a & b: *Acute herpetic gingivostomatitis showing minute vesicles on palate and yellowish-white pseudo membranous slough in labial and palatal aspect of anterior teeth.*

The patient was prescribed orally, acyclovir 200 mg, every 4 hours for 5 days. Also topical application of antiviral gel, 3 to 4 times in a day, was advised. Patient was also instructed to apply topical antiseptic, anaesthetic ointment before taking some eatables. Benzydamine hydrochloride mouthwash (0.15 g/100 ml) was prescribed for symptomatic relief from burning sensation along with analgesics and antipyretics (acelofenac 100 mg and paracetamol 500 mg) to address fever and malaise along with multivitamins for two times a day for 5 days. Patient was instructed to eat nutritious, soft and blend diet and to follow-up after a week. On follow-up visit, patient showed complete remission of lesions. Thorough full mouth disinfection, followed by supragingival and subgingival scaling was done following the remission period.

DISCUSSION

All herpes simplex viruses contain DNA nucleus which can remain latent in host neural cells, thereby, evading host immune response.[12]Two of the known herpesviridae, HSV-1 and HSV-2 are responsible for primary and recurrent mucocutaneous herpetic infections. The HSV is a double stranded DNA virus of which the HSV-1 type is responsible for oral, facial and ocular infections including primary herpetic gingivostomatitis.

The two forms of HSV have a similar structure, but differ in antigenicity and their clinical presentation, although HSV-2 is reputed to be of greater virulence.[3] Structurally, the herpes virus is made up of three components: capsid shell—which consists of proteins and double-stranded DNA: envelop—which consists of a lipid bilayer with 11 embedded glycoproteins, four of which are essential for viral entry into host cells: and tegument—which is a proteinaceous region between the capsid and the envelope.[13]

Following exposure, the virions attach to the host cells, which are mediated by envelop-related viral proteins. Once the virus has gained entry into the cytoplasm, it loses its capsid proteins by the process known as uncoating and the viral nucleic acid is transported into the host-cell nucleus. In the host-cell nucleus, the viral genome is replicated. In the next step, the new viral genome is transcribed into mRNA, which subsequently is translocated to host-cell ribosomes. The viral proteins synthesized by

host-cell ribosomes are assembled with the duplicate viral genome. Assembly is followed by maturation, a process essential for the newly formed virions to become infectious. The newly synthesized viruses, in turn, may infect other epithelial cells or enter sensory nerve endings.[14,15]

Primary acute herpetic gingivostomatitis is the most common pattern of symptomatic primary herpetic infection, and in the majority of cases, it is related to HSV-1 infection. It is more commonly observed in children in the age bracket from 1 to 5 years of age, and rarely in adolescents and young adults.[16,17] The peak of incidence is seen, from 6 months to 5 years. It rarely affects children under the age of six months, who apparently present circulating antibodies transmitted by the mother. The greater occurrence in children may be justified by the wide dissemination of the virus and due to early exposure to it.[18]

The present article describes the clinical case of an adult patient, who suffered an attack of acute herpetic gingivostomatitis. The differential diagnosis included streptococcal pharyngitis; erythema multiforme; necrotizing ulcerative gingivitis; and aphthous stomatitis.[14] Patient's history along with clinical features was typically suggestive of primary herpetic gingivostomatitis. The diagnosis of primary herpetic gingivostomatitis is generally defined by the clinical data, and no confirmative tests are necessary.[19]

Here, one should keep in mind that primary infection by HSV-1 can be subclinical or symptomatic.[20] Degree of viral replication, host's response to the pathogen, and the speed with which latency is established will determine the severity of herpetic infection.[18] The severity and quantity of intraoral lesions may significantly reduce dietary intake and predispose the patient to dehydration. Thus, it is important to balance any decrease in intake with fluids.

The recognition of the classic presentation of signs and symptoms is important, particularly in middle-aged and elderly people, in whom the superimposition of dehydration due to AHGS can complicate pre-existing medical conditions such as diabetes mellitus and kidney disease. Palliative and supportive management of orolabial herpetic infections variably consists of controlling fever and pain, preventing dehydration, and shortening the duration of lesions. Antiviral chemotherapy is available for the treatment of patients at increased risk of complications.[21] Primary line of treatment includes dietary supplements, and patients should be advised to rest, avoid smoking tobacco and drinking alcoholic beverages, eat a soft balance diet, and ensure an adequate intake of fluids, vitamins, and minerals.[2,21] Application topical anesthetic, analgesics before each meal, effectively reduces pain during eating. Benzocaine, 20%, may be a better alternative in the management of young and the debilitated when aspiration and the possibility of excessive systemic absorption are a concern.

CONCLUSION: The acute form of HSV virus infection is highly transmissible. This potential is of particular interest to dental professionals who are exposed to contact with herpetic lesions on a day to day basis. Most herpetic infections are transmitted from infected persons to others through direct contact with a lesion or infected body fluids. For this reason, gloves and safety glasses must be used during the examination, especially given that the risk of asymptomatic shedding is omnipresent. Patients should also be advised to minimize intimate contact when active lesions are present, as they are at risk of spreading the virus.

The dentist (periodontist) plays a remarkable role in diagnosis of primary acute herpetic gingivostomatis considering that due to rich symptomatology of oral lesions. This being so, it is important for him/her to be capable of recognizing the disease and creating the best condition for the well-being of the patient.

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