CASE REPORT

Management Of Acute (Primary) Herpetic Gingi vost omatitis In An Adolescent Patient: A Case Report

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ABSTRACT
Primary herpetic gingivostomati tis(PHG) is an infection of the oral cavity caused by herpes simplex virus type 1 (HSV-1). It occurs most often in infants and children younger than 6 years of age but can also be seen in adolescents and adults. Patients present with diffuse, erythematous, shiny involvement of the gingiva and the adjacent oral mucosa, with varying degrees of edema and gingival bleeding. In its initial stages, its presence is characterized by discrete, spherical gray vesicles, which may occur on the gingiva, labial and buccal mucosae, soft palate, pharynx, sublingual mucosa, and tongue[7]. This article presents an acute episode of primary herpetic gingivostomatitis in a 13-year-old female patient.
Keywords: Acute(primary) herpetic gingivostomatitis, adolescent

INTRODUCTION

PHG is a common pediatric infection usually seen before 6 years of age [1]. The causative organism in 90% of cases is HSV type 1, with complications that range from indolent cold sores [1] to dehydration [2] and even life-threatening encephalitis [3]. For an accurate diagnosis, PHG has to be differentiated clinically from herpeti form aphthous stomatitis, recurrent HSV infection, acute necrotizing ulcerative gingivitis, allergic stomatitis, ery thema multi forme, and ulcers due to chemotherapy. PHG is characterized by eryt hema, edema, capillary proliferation, and widespread vesicular eruptions affecting the vermilion border of the lip and labial mucosa, tongue, buccal and vestibular mucosa, hard and [3-5]. The condition is highly contagious [3] and a diagnosis of PHG is made based on the clinical presentation of ery the matous gingiva, mucosal hemorrhages, and clusters of small erupted vesicles throughout the mouth in children with symptoms though most children will be asymptomatic [4,5]. Symptomatic relief primarily involves pain management and oral fluids to prevent dehydration until the viral infection subsides [6]. This case study highlights a symptomatic PHG case requiring an astute diagnosis and a comprehensive treatment plan. It highlights the presentation of PHG and the possible differential diagnosis that could be considered based on current literature. This case study also emphasizes the importance for oral health clinicians to have a holistic approach to the management of oral disease as treating the patient holistically can bring about positive changes in the person's oral health status, awareness, and behaviors.

CASE PRESENTATION

A 13-year-old, female patient presented with the main complaint of oral ulcers which she noticed 24 hours ago. The ulcers were accompanied by severe pain, bleeding, and weight loss, and the patient reported not being able to eat any food for the last 3 days. The patient also reported a fever and malaise which started 7 days ago. The ulceration started at the right corner of the lips intraorally and then the involvement of the whole upper lip and tip of the tongue was reported.

Upon intraoral examination, a large ery the matous ulcer (convergence of smaller ulcers) measuring 1-2 cm in size on the right buccal mucosa with well-defined margins, inflamed and edematous edges, pale floor covered with smooth granulation tissue was observed (figure 1). On palpation the area was tender and bleeding was observed. The surrounding area was red, edematous, and glossy in appearance. The labial mucosa had the presence of multiple smaller ulcers with similar appearance (Figure 2). The tongue also showed multiple ulcers at the tip with a similar appearance (Figure 3). The gingiva showed redness, rolled edges, and a soft consistency along with an absence of stippling. The subma dibular lymph nodes on the right side were inflamed and palpable.

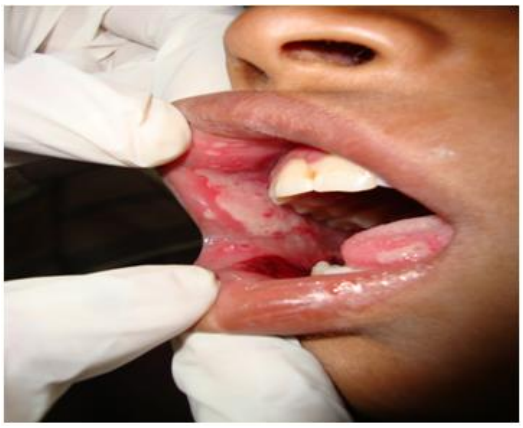

Figure: 1 Ulceration on the buccal mucosa with well-defined margin and edematous edges.

Figure: 2 Multiple ulcers on the labial mucosa

Figure: 3 Multiple ulcers involving the tip of the tongu Provisional diagnosis

Based on history and clinical examination showing multiple ulcerations, gingival condition, and fever a provisional diagnosis of Primary Herpetic Gingi osto matitis was put forth. Investigations The diagnosis of primary herpetic gingivostomatitis is generally defined by the clinical data, and no confirmative tests are necessary. The available investigations are:

- Viral culture is considered the gold standard and the most sensitive of the diagnostic techniques but is generally limited to the hospital setting.
- Direct immun ofl uore scence technique is also not readily available and is restricted to hospital settings.
- Tzanck test demonstrates multinucleated epithelial giant cells which are consistent with a

herpes virus infection. It generally detects only about 60% of herpes simplex virus (HSV) infections.

- Smears also yield no information as to whether the viral agent is HSV-1, HSV-2, or Varicella zoster virus.

To confirm the diagnosis, cytological smear preparation was done from a scraped sample of the buccal mucosa. H&E stained cytological smear showed infected cells arranged in clusters and neutrophil infiltration along with ballooning degeneration which is a characteristic histological feature of PHG.

Differential diagnosis

- Ery thema multi for me
- Stevens-Johnson syndrome
- Bullous lichen planus
- Desquamative gingivitis
- Recurrent aphthous stomatitis

MANAGEMENT

The patient was treated with Acyclovir (Zovirax oral suspension) in a syrup form which the patient was instructed to consume 3 times daily for 5 days and a Paracetamol oral suspension (Crocin oral suspension) 2 times daily for 5 days after meals. Treatment of the patient also included making her rinse with an anti-inflammatory and analgesic oral rinse containing benzy damine hydrochloride (Tan tum Oral rinse) to ease the topical oral pain. The patient was also prescribed a B-Complex forte multivitamin supplement (Becosule capsules) 1 time daily for 5 days to overcome any nutritional deficiencies arising due to difficulty in eating. A recall appointment for 1 week' time was given for assessment and further treatment. On presentation for her recall appointment, she had regained her health, and the ulcers had healed without scarring (Figure - 4,5). On her second appointment after 2 weeks of treatment, complete healing was observed (Figure - 6,7)

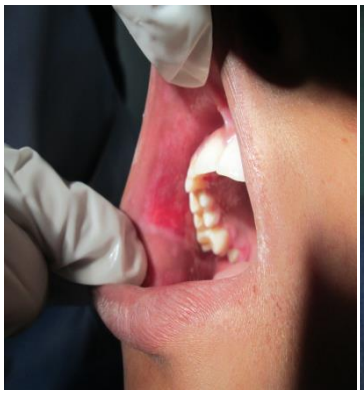

Figure -4

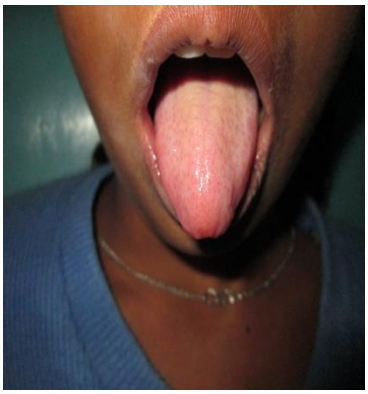

Figure - 5

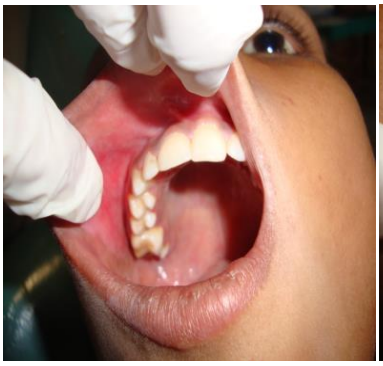

Figure - 6

Figure - 7

DISCUSSION

Two of the known Herpesviridae, herpes simplex viruses type 1 (HSV-1) and HSV-2, are responsible for primary and recurrent mucocutaneous herpetic infections. HSV-1 is predominately associated with orolabial infections, while HSV-2 is predominately associated with genital disease. Orogenital contact may allow either serotype to cause oral or genital lesions. The two forms of HSV have a similar structure, but differ in antigenicity, although HSV-2 is reputed to be of greater virulence [8]. 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.
- tegument—which is a proteinaceous region between the capsid and the envelope [9].Following exposure, the virions attach to the host cells which is 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 [10][11].Most herpetic infections are transmitted from infected persons to others through direct contact with a lesion or infected body fluids, for example, vesicular exudates, saliva, and genital fluids. Clinically, HSV-1 infections begin with prodromal symptoms of fever, loss of appetite, malaise, and myalgia. Within a few days of prodromal symptoms, erythema and clusters of vesicles and/or ulcers appear on the hard palate, attached gingival and dorsum of the tongue, and non-keratinized mucosa of buccal and labial mucosa, ventral tongue and soft palate [9][12]. Vesicles break down to form ulcers that are usually 1–5 mm and coalesce to form larger ulcers with scalloped borders and marked surrounding erythema. The gingiva is often fiery red, and the mouth is extremely painful, causing difficulty in eating. (Our patient reported all these features.) It is important to distinguish primary from recurrent herpetic infection. History may help distinguish primary from secondary infection, as patients with a secondary infection will recall previous episodes of vesicular eruptions on their lips [10][11][13].

CONCLUSION

PHG in some instances is asymptomatic and even goes unnoticed, however in symptomatic cases it can be a very debilitating condition. Oral health clinicians need to be aware of the indicative signs and symptoms of this condition and not confuse it with other similar conditions on presentation. A holistic approach to the management of oral disease must be always adhered to, with the vision that it can bring about positive changes in the person's oral health status, awareness, and behavior.

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