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Herpetic Gingivostomatitis with Secondary Impetigo Contagiosa: A Case Report

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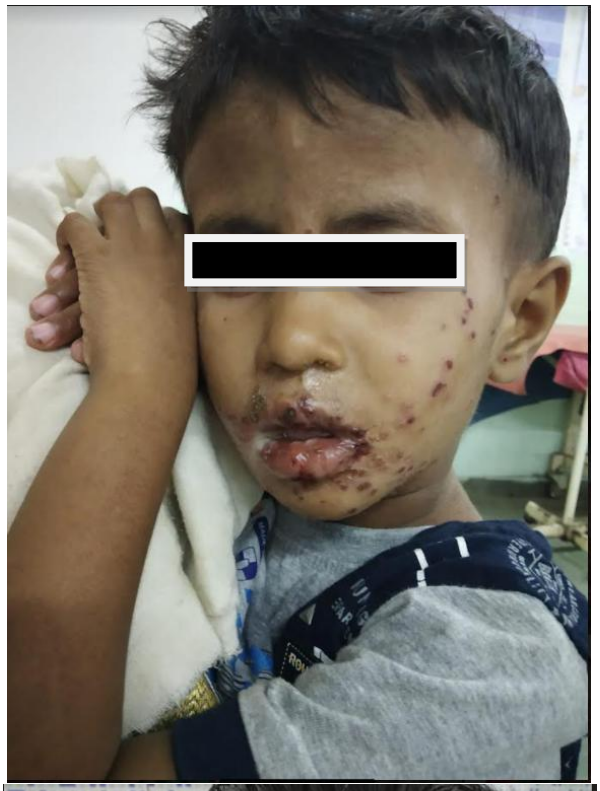
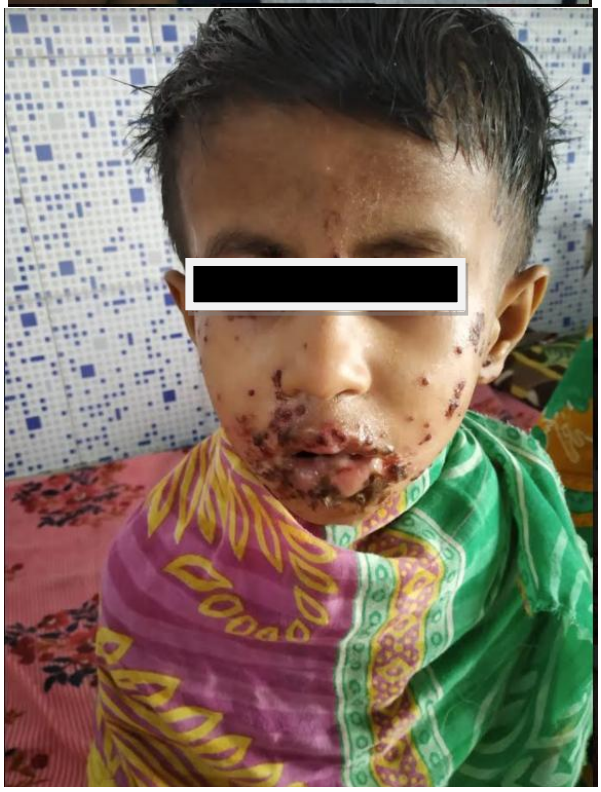
Abstract
Acute herpetic gingivostomatitis represents the pattern of infection with herpes simplex virus. HSV is a double stranded DNA virus and belongs to HHV (herpes human virus). Around 90% of these herpetic gingivostomatitis cases are caused by HSV type-I and sometimes by HSV type-II. This infection is usually seen in the perioral region, source of infection being saliva or previous lesions, although not always symptomatic can present with symptoms in 10% of cases. Rarely, we may see a superadded infection on the raw lesion, complication by staphylococcus aureus, resulting into and secondary impetigo. Source of infections in such cases will the commensal staphylococcus which is usually present as a nasal commensal, may travel through the nasal discharge and colonizing the already raw lesions and hence leading to a secondary impetigo contagiosa. Here, we shall report a case of two siblings 8 years and 6 years old with acute herpetic gingivostomatitis complicated by a secondary impetigo contagiosa.

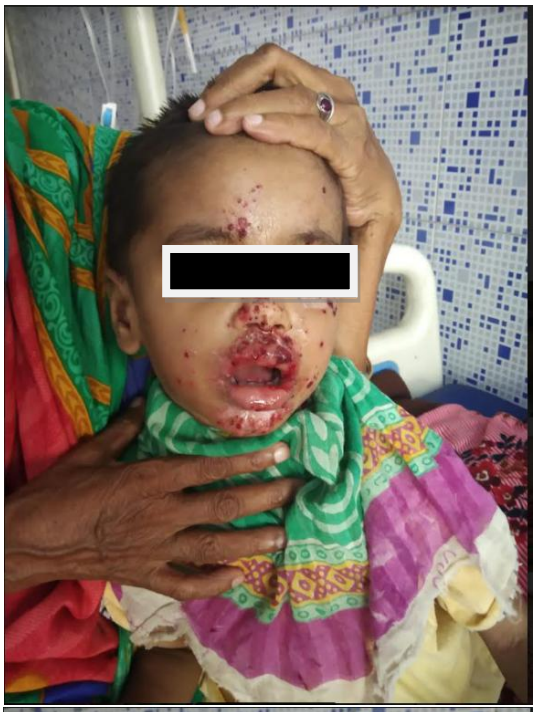
Case

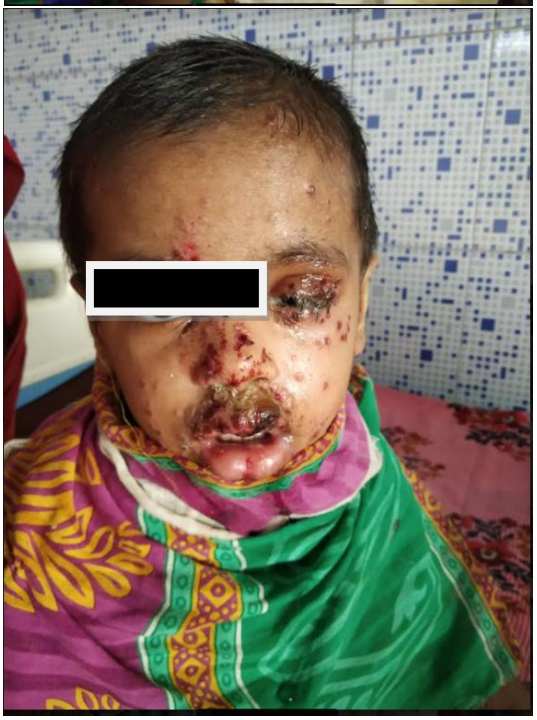
The two siblings aged 9 years and 8 years, female child presented to us with the complains of multiple eruptions covering the perioral and nasolabial fold and some lesions on the cheeks, there was also a complain of decreased feeding and difficulty in opening mouth. On examination, there were multiple eruptions, some in the form of vesicular eruptions and some having and erythematous base with a honey-colored crust, there was however involvement of the eyelids as well but with sparing of the conjunctiva. On oral examination there were multiple shallow oral ulcerscovering the base of the tongue and the lateral border as well. The patients had no oozing from the ulcers but however there was episode of increased salivation. On the basis of the clinical features a diagnosis of primary herpetic gingivostomatitis complicated with secondary impetigo contagiosa was made and treatment was started, patient has responded well showing healed lesion at around 7 days of treatment.



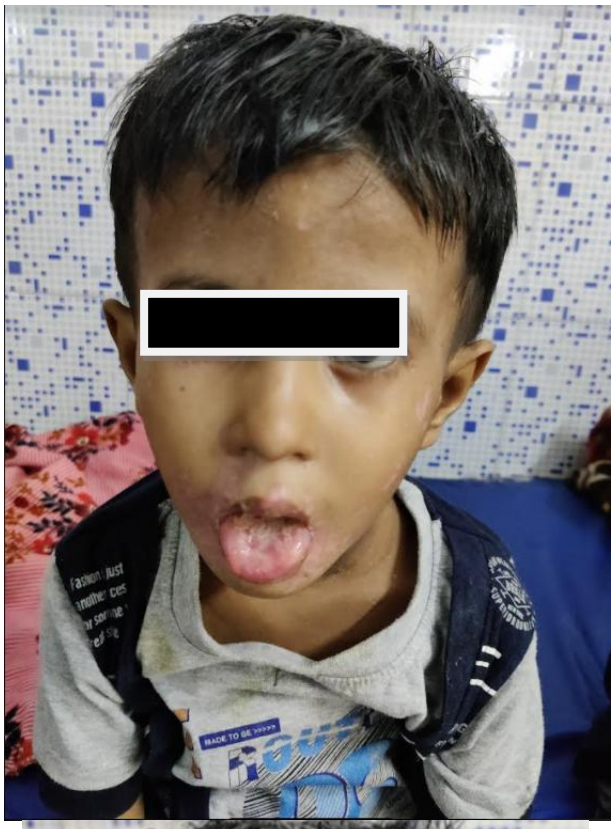


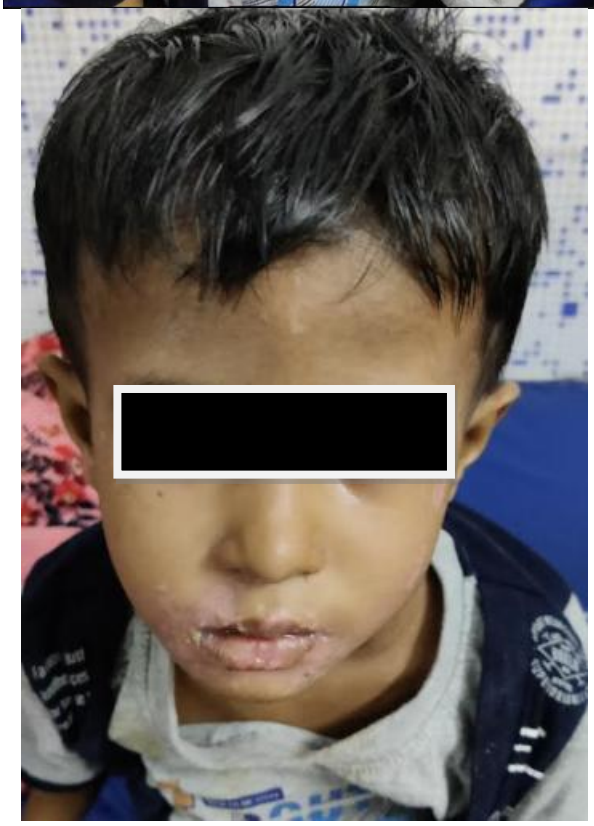
PIC 1(A) & 1(B): Sibling 1: multiple vesicular and honey crusted lesions, oral ulcers



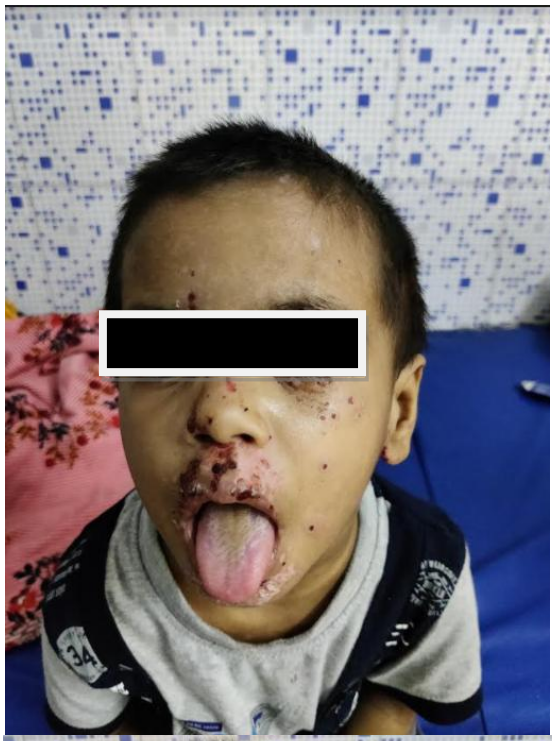


PIC 2(A) & (B): Sibling 2: multiple vesicular and honey crusted lesion, oral ulcer, sparing of conjunctivae





PIC 3(A) & 3(B): Sibling 1: after 1 week of treatment



PIC 4(A) & 4(B): Sibling 2: after 5 days of treatment

Treatment

The patient was instructed to start with oral liquid diet first then gradually moving to soft, nutritious diet

Medications prescribed:

1. Acyclovir at 20mg/kg for 5 days
2. Benzydamine hydrochloride mouth wash
3. Azithromycinat 10mg/kg for 5 days

4. Ointment mupirocin
5. Local lignocaine gel
6. Analgesics and antipyretics
7. B complex with zinc syrup

Discussion

Two of the known Herpesviridae, herpes simplex viruses type 1 (HSV-1) and HSV-2, are most commonly responsible for primary and recurrent mucocutaneous herpetic infections in children and adolescents age group. 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. Structurally, the herpes virus is made up of three components:

- Capsid Shell— which consists 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.

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 synthesised 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 synthesised viruses, in turn, may infect other epithelial cells or enter sensory nerve endings.

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 few days of prodromal symptoms, erythema and clusters of vesicles and/or ulcers appear on the hard palate, attached gingival and dorsum of tongue and non-keratinised mucosa of buccal and labial mucosa, ventral tongue and soft palate. 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 with all these features.)

As we have different flora of bacteria as commensal n different parts of the body, we are having Staphylococcus aureus as a commensal in the external nares, thus turning it into a major contagion for nosocomial infection or secondary infection of previous skin lesions. Here in this case patient first developed herpetic gingivostomatitis, vesicular eruptions, which when burst out were secondarily infected by staphylococcus present already in the nares, as evident by the erythematous base with golden crusts.

Conclusion

Herpetic gingivostomatitis until superadded with secondary infection, antibiotics do not play any role. Unlike coxsackie infection which predominately affects posterior part of the mouth cavity, this herpetic lesion predominately affects the anterior part involving tongue, gingivae, lips which responds amicably to acyclovir. Local soothing agent with benzocaine allays the pain. B-complex with zinc hastens early recovery

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