Case Report

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A Case Report of Erythema Multiforme

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ABSTRACT

Erythema multiforme is a reactive mucocutaneous disorder which is an acute, self-limiting, inflammatory disorder characterized by degrees of blistering and ulceration. This disorder is created hypersensitivity reaction which is triggered by certain infection, medication , food additives or chemicals. We report a case of erythema multiforme managed with prophylactic valacyclovir and systemic corticosteroid. An 47 years old female patients had lesions in the oral cavity, lips, hand and ear which had been diagnosed as erythema multiforme minor. This was not related to drug intake; it was related to food intake. We can treat this disease with valacyclovir for 2weeks to control this disorder.

KEY WORDS:

Erythema Multiforme, Mucocuteneous Disorder, Valacyclovir

INTRODUCTION:

Erythema multiforme is a reactive, inflammatory, self-healing, hypersensitive mucocutaneous disorder which is characterized by a skin eruption with or without oral or other mucous membrane lesions. This disorder could be related to immunologic hypersensitivity reaction to certain infections, medications and other triggering factor like as food additives.

Erythema multiforme are four types, they are major, minor, Steven Johnson syndrome and toxic epidermal necrolysis. Erythema multiforme minor shows typical target lesions, raised atypical target lesions, minimal mucous membrane involvement and when present only 1 site most commonly in the mouth. Erythema multiforme major shows cutaneous lesion, typically both oral mucosa are involved. Oral lesions usually widespread and severe. Symmetrically distributed typical target lesions or atypical raised target lesions or both. Stevens-Johnson syndrome are mainly different from erythema multiforme major is based on the location of lesions and the presence of systemic symptoms. Primarily atypical flat target lesions and macules rather than classic target lesions. Multiple mucosal sites involved with scarring of the mucosal lesions. Toxic epidermal necrolysis shows widespread purpuric macules or flat atypical targets.

When we treat this patient We found intra-oral lesions in angle of the mouth, right and left buccal mucosa extending up to the posterior part of the palate. Also, found skin lesion (target lesions) in fingers and ulcer in both ear.

In this report, we discuss the case of an 47 year old female patient who was clinically diagnosed with erythema multiforme minor. The disease was controlled by the use of valacyclovir to prevent further recurrence. We can give 9 days course of systemic prednisolone.in first 3 days we can give 60 mg thrice daily, next 3 days 40mg twice daily and last 3 days 20mg once daily. In attached to systemic prednisolone we also give valacyclovir 1gm for 5 days twice daily 12 hour interval. We also used esomeprazole magnesium trihydrate 20 mg and chlorhexidine gluconate .2% gargle.

CASE REPORT:

An 47 years female patient came to the update Dental College & Hospital with the complaints of painful ulcers on right and left buccal mucosa extending up to the posterior part of the palate and there is hemorrhagic clusters on lips. Also found skin lesion in hand finger and ulcer in both ear. The patient had started treatment with Allopurinol, Folinic acid, Prednisolone, Gliclazide, Lactulose. She also given history of intake Pineapple after this she developed ulcers on mouth and ulcer on ear, hemorrhagic crust on lip and also developed skin lesion.

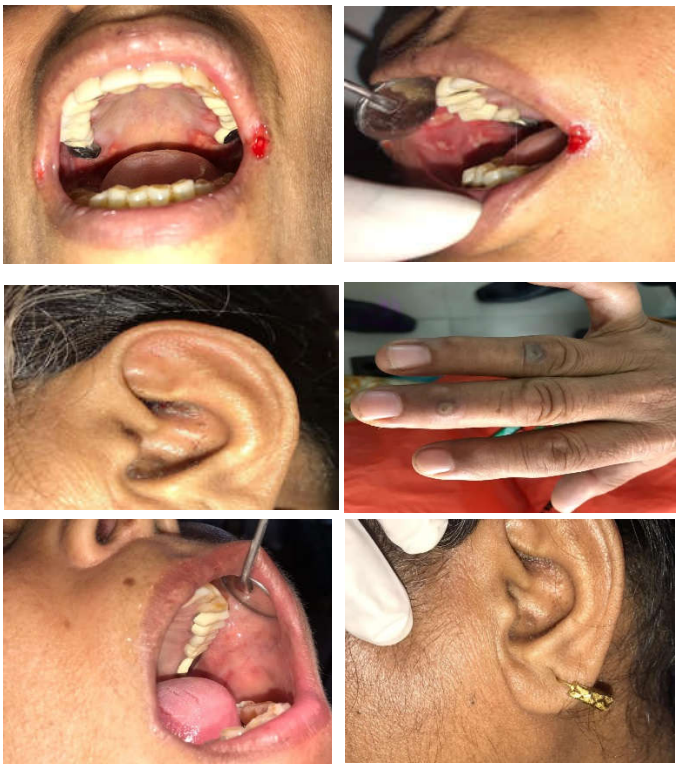
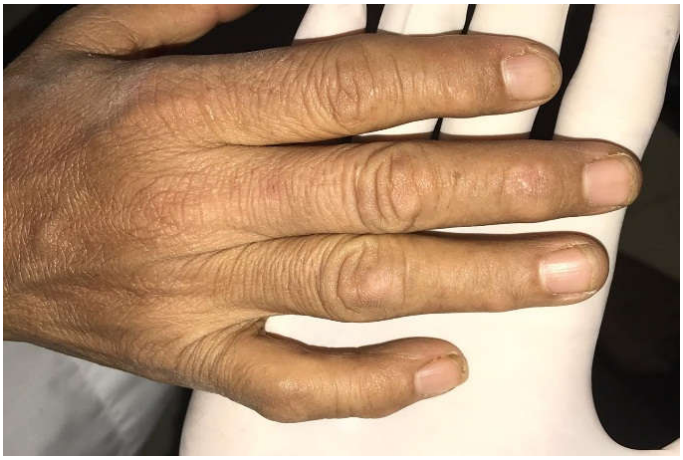


Figure: 1 Diffuse ulcerations in the oral mucosa involving bilateral buccal mucosa & posterior palate & angle of the mouth. Figure-2: Diffuse ulcerations in the oral mucosa involving bilateral buccal mucosa & angle of the mouth . Figure-3: Lesion also involving the external ear, Figure-4, Diffuse ulcerations in the oral mucosa involving bilateral buccal mucosa & angle of the mouth. Figure:5 Lesion also involving the external ear. Figure: 5-Round skin lesion & necrotic center (target lesion) Seen on the hands. Figure: 6-There is no ulceration of buccal mucosa, angle of the mouth & posterior part of the palate, Complete healing of lesion on ear.

This disease was diagnosed as erythema multiforme minor and this was triggered by Pineapple. We treat this patient 5 days course of valacyclovir and 9 day course of prednisolone. In first 3days we can give prednisolone 60mg thrice daily, next 3 days 40mg twice daily and last 3 days 20mg once daily. We can maintain this tapering dose. The course of valacyclovir 1mg given 12 hour interval twice daily for 5 days. And also given esomeprazole magnesium trihydrate and chlorhexidine gluconate .2% gargle. Renal & Liver functions were monitored during the coarse of treatment.No oral and skin lesions during the 7days of treatment and the disease is currently under control.



Complete healing of skin lesion on hands.

Discussion:

Erythema multiforme is an acute, sometimes recurrent, mucocutaneous condition of uncertain etiopathogenesis that can follow the administration of drugs orinfections. Infection with HSV is the most common feature in the development of erythema multiforme minor. Herpes-associated erythema multiforme (HAEM) can be found several days or weeks following an episode of HSV. Both HSV types 1 and 2 have been shown to precipitate HAEM,3 and health history, clinical observations and prospective studies indicate that most cases of erythema multiforme are preceded by infection with HSV,9 although it is important to emphasize that HSV infection may be clinically silent.10 HSV DNA has been detected in 60% of patients clinically diagnosed with recurrent HAEM and in 50% of patients with recurrent idiopathic erythema multiforme using polymerase chain reaction (PCR) of skin biopsy specimens.6 Another study11 revealed that the cutaneous lesions of patients with HAEM were infected with HSV-1 in 66.7% of cases, HSV-2 in 27.8% of cases and with both HSV types in 5.6% of cases. Typically, an erythema multiforme (minor or major) lesion begins 10–14 days following the clinical manifestations of an HSV infection. The lip is the most common site of preceding HSV infection in case of HAEM.12 In the present case, the serology for HSV was positive, confirming that the erythema multiforme was associated with an HSV infection. However, it is important to emphasize that HSV was identified only during the second episode of the disease and that HAEM was confirmed at the third episode. Several studies1,13 have demonstrated that the pathogenesis of HAEM is consistent with a delayed hypersensitivity reaction. The disease begins with the transport of HSV DNA fragments by circulating peripheral blood mononuclear CD34+ cells (Langerhans cell precursors) to keratinocytes, which leads to the recruitment of HSV-specific CD4+ TH1 cells. The

inflammatory cascade is initiated by interferon-γ (IFN-γ), which is released from the CD4+ cells in response to viral antigens, and immunomodulated epidermal damage subsequently begins. 1,13,14 PCR has been employed to detect the presence of HSV DNA in HAEM lesions and tissues, and HSV genes can also be identified with reverse transcriptase PCR or immunohistochemistry using antibodies to specific viral genes. Detection of IFN-γ in HAEM lesions can also be used as evidence of virus involvement.1 Serology to identify HSV-1 and HSV-2 and to detect specific IgM and IgG antibodies may confirm a suspected history of HSV infection, although it is not necessary for diagnosis.2 The diagnosis of HAEM is clinical and is easier when the patient develops target lesions with a preceding or coexisting HSV infection. The finding of typical skin or oral lesions (or both) in a patient with suspected HAEM supports the clinical diagnosis. In our case, diffuse ulcerations in the oral mucosa involving the buccal mucosa, the labial mucosa and hemorrhagic crusts on the lips as well as the classic skin lesions were seen Pronounced systemic signs and symptoms (cutaneous and mucosal lesions) suggested the diagnosis of erythema multiforme major.

Histopathologic examination revealed a pattern that is characteristic of erythema multiforme, but is not pathognomonic.2 Subepithelial or intraepithelial vesiculation is usually seen in association with necrotic basal keratinocytes, and subepithelial edema and intense inflammatory infiltration (lymphocytes, neutrophils and often eosinophils) are present; again, these features are characteristic of erythema multiforme, but not pathognomonic. Often, the inflammatory infiltrate is arranged in a perivascular orientation that is typically seen in erythema multiforme.4 Changes affecting both the epithelium and supporting connective tissue were seen in the present case. All the symptoms together, including the clinical and histologic features as well as the patient's HSV-positive status and symptom recurrences, confirmed the diagnosis of HAEM.HAEM is often effectively managed with acyclovir (200mg, 5 times a day for 5 days), but only if the therapeutic scheme is started in the first few days. If erythema multiforme keeps recurring, a continuous low dose of oral acyclovir is necessary.3 Oral acyclovir has been shown to be effective at preventing recurrent HAEM,10 and the protocols may include 200–800 mg/day for 26 weeks.4,10,17,18 If acyclovir treatment fails, valacyclovir can also be prescribed (500 mg twice a day). The latter has greater oral bioavailability and is more effective at suppressing recurrent HAEM.19 During the second and third episodes in this case, the patient was treated with acyclovir (1,000 mg/day), and prophylactic use of acyclovir was prescribed to prevent recurrences. The dosage of an antiviral medication may be reduced once the patient is free of recurrences for 4 months, and the drug may eventually be discontinued.2

In our case, the patient was treated for 5days with valacyclovir, starting with 2 gm/day.

CONCLUSION:

An important step in the management of erythema multiforme is recognition and withdrawl or prevention of contact with the causative agent. The diagnosis of erythema multiforme should be based on the clinical signs, symptoms and a thorough patient medical history and it is highly linked with Herpes virus or can be drug induced erythema multiforme . Patients should be informed about the condition and the importance of preventing recurrences.

REFERENCES:

1. Kokuba H, Aurelian L, Burnett JW. Herpes simplex virus associated erythema multiforme (HAEM) is mechanistically distinct from druinduced erythema multiforme: interferon-γ is expressed in HAEM lesions and tumor necrosis factor-α in drug-induced erythema multiforme lesions. J Invest Dermatol. 1999;113(5):808-15. https://doi.org/10.1046/j.1523-1747.1999.00754.x PMid:10571738

2. Lamoreux MR, Sternbach MR, Hsu WT. Erythema multiforme. Am Fam Physician. 2006;74(11):1883-8.

3. Al-Johani KA, Fedele S, Porter SR. Erythema multiforme and related disorders. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2007;103(5):642-54. https://doi.org/10.1016/j.tripleo.2006.12.008 PMid:17344075

4. Farthing P, Bagan JV, Scully C. Mucosal diseases series. Number IV. Erythema multiforme. Oral Dis. 2005;11(5):261-7. https://doi.org/10.1111/j.1601-0825.2005.01141.x PMid:16120111

5. Weston WL. Herpes-associated erythema multiforme. J Invest Dermatol. 2005;124(6):xv-xvi. https://doi.org/10.1111/j.0022-202X.2005.23764.x PMid:15999399

6. Ng PP, Sun YJ, Tan HH, Tan SH. Detection of herpes simplex virus genomic DNA in various subsets of Erythema polymerase chain reaction. Dermatology. 2003;207(4):349-53. https://doi.org/10.1159/000074112 PMid:14657624

7. Nanda S, Pandhi D, Reddy BS. Erythema multiforme in a 9-day-old neonate. Pediatr Dermatol. 2003;20(5):454-5. https://doi.org/10.1046/j.1525-1470.2003.20332.x PMid:14521571

8. Aburto C, Torres R, Caro A, Salinas E. Síndrome de Stevens-Johnson asociado a infección por Mycoplasma pneumoniae y vírus herpes [StevensJohnson syndrome associated with Mycoplasma pneumoniae and herpes virus infection]. Folia Dermatol Peru. 2005;16(2):81-4. [In Spanish; English abstract].

9. Kokuba H, Imafuku S, Huang S, Aurelian L, Burnett JW. Erythema multiforme lesions are associated with expression of a herpes simplex virus (HSV) gene and qualitative alterations in the HSV-specific T-cell response. Br J Dermatol. 1998;138(6):952-64. https://doi.org/10.1046/j.1365-2133.1998.02260.x PMid:9747355

10. Fernández García JR, Alcaraz Vera M, Ruiz Jiménez MA, Rodríguez Murillo JM, Hens Pérez A. Eritema multiforme. Rev Esp Pediatr. 2000;56:2025.

11. Sun Y, Chan RK, Tan SH, Ng PP. Detection and genotyping of human herpes simplex viruses in cutaneous lesions of erythema multiforme by nested PCR. J Med Virol. 2003;71(3):423-8. https://doi.org/10.1002/jmv.10502 PMid:12966549

12. Farthing PM, Maragou P, Coates M, Tatnall F, Leigh IM, Williams DM. Characteristics of the oral lesions in patients with cutaneous recurrent erythema multiforme. J Oral Pathol Med. 1995;24(1):9-13. https://doi.org/10.1111/j.1600-0714.1995.tb01122.x PMid:7722922

13. Aurelian L, Ono F, Burnett J. Herpes simplex virus (HSV)-associated erythema multiforme (HAEM): a viral disease with an autoimmune component. Dermatol Online J. 2003;9(1):1.
14. Spandau U, Brocker EB, Kampgen E, Gillitzer R. CC and CXC chemokines are differentially expressed in erythema multiforme in vivo. Arch Dermatol. 2002;138(8):1027-33. https://doi.org/10.1001/archderm.138.8.1027
PMid:12164740
15. Gavaldá-Esteve C, Murillo-Cortés J, Poveda-Roda R. Eritema multiforme. Revisión y puesta al dia [Erythema multiforme: revision and update]. RCOE. 2004;9(4):415-23. https://doi.org/10.4321/S1138-123X2004000400004
16. Shin HT, Chang MW. Drug eruptions in children. Curr Probl Pediatr. 2001;31(7):207-34.
https://doi.org/10.1016/S1538-5442(01)70015-6
https://doi.org/10.1067/mps.2001.117632
17. Bowers KE. Oral blistering diseases. Clin Dermatol. 2000;18(5):513-23. https://doi.org/10.1016/S0738-081X(00)00141-3
Hernanz JM, González-Beato M, Pico M, Pérez S, Marengo S. Eritema exudativo multiforme "minor." Acta