

## Erythema Multiforme Minor- A Case Report

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### ABSTRACT

Erythema multiforme (EM) early diagnosis is most important as it is a lethal mucocutaneous disorder. There are a number of etiologic agents associated with EM which include microbiological, drugs, chemicals, and immune disorders, and essentially all present as oral mucosal ulceration and erosions with bloody encrustations and tissue tags and most often associated with characteristic dermal target/iris lesion. Here, we report a case of EM minor with characteristic oral and dermal lesions in a 25-year-old male patient.

### INTRODUCTION

Erythema multiforme is an acute, self-limiting, recrudescing, ulcerative mucocutaneous disorder that presents as a reactive lesion to antigenic challenge<sup>[1]</sup>. EM principally is caused by antigens, which are essentially drugs or microbes<sup>[2]</sup>.

### CASE REPORT

A 25-year-old male patient presented to the department with multiple bleeding painful mouth ulcers for the past 2 days. History reveals that the patient used lip balm, following which he developed vesicles in the labial and buccal mucosa. The vesicles eventually ruptured leaving raw, bloody painful ulcers owing to which he was unable

to consume either solid or liquid foods. Concurrently patient developed dermal lesions which were preceded by severe itching.

Cutaneous target lesions on general examination presented as a central red papule or flat erythema surrounded by pale concentric rings of erythema. Oral lesions manifested as multiple ulcers involving both keratinized and non-keratinized mucosa with large areas of denuded surface epithelium with tissue tags and few intact vesicles filled with clear fluid, were also present. And actively bleeding hemorrhagic encrustations on labial mucosa were also noticed. On bilateral mucosa, there were large diffuse ulcers and erosions extending from the corner of the mouth to the retro molar region

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covered by pseudomembranous slough surrounded by an erythematous halo and non-indurated base. Identical ulcerative lesion was also present in the mid palatal region.

Based on history, bloody encrustations in lower labial mucosa and presence of pathognomic cutaneous target/iris lesions and an intact vesicle, the present case was diagnosed as EM minor. Herpetic gingivostomatitis was considered as a differential diagnosis as the lesion involved masticatory mucosa and commonly affects children and teenagers and mucous membrane pemphigoid and pemphigus vulgaris were also included in the differential diagnosis as it is also an acute vesiculobulbous mucocutaneous disease.

The patient was initially started with a single morning dose of systemic corticosteroid prednisolone 20 mg b.i.d along with topical steroid clobetasol propionate 0.05% and benzydamine hydrochloride 0.15% mouth wash for ten days. Systemic steroids were tapered to 10 mg every week as clinically lesion was regressing up to a maintenance dose of 5 mg. The patient was instructed to follow meticulous oral hygiene instructions. After 3 weeks absolute resolution of oral and dermal lesions were achieved. The patient is under review for 6 months with no episodes of recurrence.

## DISCUSSION

Erythema multiforme was first mentioned in literature by von hebra in 1866 where its characteristic peripheral manifestation of lesions was mentioned<sup>[4]</sup>. Classification of EM could either based on the severity of dermal and mucosal involvement or by its aetiology. EM major would involve at least two mucosal surfaces<sup>[5]</sup> and symmetrical distribution of target or iris lesions and EM minor affects only one mucosal surface and minimal atypical target lesions. Based on aetiology it could be

classified into infections, drugs, chemical or immune-mediated. Among this, over 90% of cases occurs because of herpes simplex virus infection<sup>[2,6]</sup>.

EM is thought to be the result of viral antigen cells homing HSV DNA polymerase challenge against cell-mediated immunity. The virus in the acral circulation gets phagocytosed by mononuclear cells such as macrophages and CD34+ Langerhans cells which carry cutaneous lymphocyte antigen receptors. The phagocytosed viral nuclear material is transferred to the keratinocyte in the epidermis. Tethering of endothelial cells to Langerhans cells containing HSV antigens is further catalyzed by upregulation of E-cadherins. And finally, an inflammatory response is ensured by upregulation of adhesion molecules<sup>[3]</sup>. On the other hand CD8+ T-cell attack and expression of TNF- $\alpha$  in the lesion are involved in drug-associated EM<sup>[7]</sup>.

EM usually involves individuals of II- IV decade. Oral lesion manifest as multiple vesicle or bullae which eventually rupture leading to large areas of sloughing painful bleeding ulcers. Recrudescence episodes affect the quality of life in certain patients<sup>[6,8,9]</sup>.

Clinical history and clinical presentation still prevail as the confirmatory test for EM<sup>[2]</sup>, as there are no ideal investigations to substantiate the diagnosis. Biopsies are beneficial only during vesicular phase as ulcerative phase biopsies turn non-specific once they rupture.

Management starts with the identification of causative agents. If the precipitating factor is a drug, refrain from using that drug. If viral, acyclovir 400 mg q.i.d or valacyclovir 500 mg b.i.d for six months would be beneficial. Symptomatic treatment includes the use of topical analgesics, anesthetic mouthwashes together with soft bland diet<sup>[9]</sup>. The mainstream treatment still prevails to be topical and tapering dose protocol of systemic steroids<sup>[4]</sup>, augmented with immuno-modulators such as

cyclosporine, dapsone, cyclophosphamide, levamisole which are preferred in case of recalcitrant cases<sup>[2]</sup>.



Bloody crustations on lower lip



Intact vesicle in left labial mucosa



Atypical target lesions



Generalized large spreading denuded epithelial surface



Second day lesions



Completely healed mucosal lesions after 3 weeks



Resolved Cutaneous lesions after 3 weeks

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