

Erythema Multiforme: A Case Report

Taseer Bashir¹,
Saluja Arti²,
Gupta Jyoti³,
Yadav Monu¹,
Krishnan Vijay¹,
Ahmad Naeem⁴

¹Post-graduate Student, Department of Oral Medicine, Career Post Graduate Institute of Dental Sciences, Lucknow, Uttar Pradesh, India, ²Senior Lecturer, Department of Oral Medicine, Career Post Graduate Institute of Dental Sciences, Lucknow, Uttar Pradesh, India, ³Professor, Department of Oral Medicine, Career Post Graduate Institute of Dental Sciences, Lucknow, Uttar Pradesh, India, ⁴Senior Lecturer, Department of Prosthodontics, Career Post Graduate Institute of Dental Sciences, Lucknow, Uttar Pradesh, India

Corresponding Author: Dr. Taseer Bashir, Post-graduate Student, Department of Oral Medicine, Career Post Graduate Institute of Dental Sciences, Lucknow, Uttar Pradesh, India.
Phone: +91-9415132145. E-mail: taseer_bds@yahoo.co.in

Abstract

Erythema multiforme (EM) is a mucocutaneous disorder, which ranges from a mild, self-limited, cutaneous, exanthematous variant with minimal oral involvement to a progressive, fulminating, severe variant with extensive mucocutaneous epithelial necrosis (Stevens-Johnson syndrome [SJS]; and toxic epidermal necrolysis). There are no specific diagnostic tests for EM, and the diagnosis is mainly clinically supported if necessary by biopsy. EM results from a cell-mediated immune reaction against a precipitating factor. There are no specific diagnostic tests for EM, and the diagnosis is mainly clinical supported if necessary by biopsy. Here, a case report of a female patient who was successfully diagnosed with EM using cytosmear is being discussed.

Keywords: Cytosmear, Erythema multiforme, Target lesion, Vesico-bullous

INTRODUCTION

Ferdinand Von Hebra described erythema multiforme (EM) in the year 1866 as a self-limited and acute skin disease that is symmetrically scattered on the extremities with a typical recurring concentric pattern in the form of “target lesion.”¹ It is a mucocutaneous reactive disorder comprising of variants in a range from a mild, exanthematous, self-limited and cutaneous variant with least oral involvement known as EM minor; to a more severe, fulminating and progressive variant with an extensive mucocutaneous epithelial necrosis known as Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN).²

Etiology

About 50% of cases are idiopathic. Most notable causes are infectious agents and drugs. Infectious causes are more common in children and are implicated more commonly in EM. Herpes simplex infection is most common cause in young adults.³

Clinical Findings

- Symmetrically distributed erythematous expanding macule or papule evolve into classic iris or target lesion

with bright red borders and central petechiae vesicles or purpura

- Lesions show centripetal spread
- Burning sensation is noticed in affected areas
- Rash favors palm and soles, dorsum of the hands, and extensor surface of extremities and face
- Nonspecific prodromal symptom such as fever malaise myalgia, arthralgia, headache, sore throat, cough, nausea, vomiting may appear 1-14 days before the skin lesions develop.

Skin Lesions

These are classified as typical targets, raised atypical target, flat atypical targets and erythematous macule with or without blisters. These lesions are present as symmetrical distributions on the extensor surfaces of the extremities.

Oral Findings

Oral lesion appears along with skin lesion in 70% of the cases. Oral lesions start as bullae on an erythematous base, but intact bullae are rarely seen by the clinician because they break rapidly.⁴

Histo-pathological Picture

EM has high density of cell infiltrate rich in T-lymphocyte.⁵

CASE REPORT

A female patient named Afreen Bano aged 37 years reported to the Department of Oral Medicine and Radiology with the chief complaint of pain and ulcers in the mouth since 5 months.

History of Present Illness

Revealed that the patient was suffering from ulcers and pain in the mouth since 5 months. She had a difficult swallow. Ulcers were painful and bled on rupturing. Pain was sudden in onset, severe in intensity, continuous in nature and was of a lancinating type. She had a burning sensation. Initially, lesions started as the vesicles that ruptured in 2-3 days. Firstly lesion appeared on the palate.

During extra-oral examination, vesicles in axilla and dorsal surface of the hand were present. Lips were crusted and bled. In intra-oral examination, mixed red and white, diffused large, irregular lesions were present on the buccal mucosa of the right and left side, palate and tongue along with the necrosed tissue. Initially, lesions start in the forms of the vesicles that ruptured in 2-3 days to form the ulcer, which bled on palpation. Lesion was tender on palpation and was non scrapable.

Differential Diagnosis

As history and clinical features were suggestive of vesicobullous lesion or viral lesion differential diagnosis of pemphigus vulgaris, bullous lichen planus, herpes zoster, herpes simplex were formed.

Investigations

Complete blood count and cytosmear was suggested.

Complete blood count

Hb - 10 g %, bleeding time - 3 min 30 s, clotting time - 5 min 30 s, neutrophil count was found to be 74%, lymphocyte count was 24% eosinophil count was 02% monocyte count was 00% and basophils 00%.

Cytosmear

Cytosmear revealed numerous acantholytic cells which appear to be cytomorphologically normal with interspersed neutrophils. And histopathological impression is of acute intraepithelial vesiculobullous lesion.

Treatment

Local application of kenakort 2 times daily, local application of gentian violet, oral dose of corticosteroid, i.e., prednisolone 30 mg twice a day for 1 week was prescribed to the patient. Antiseptic, analgesic and anesthetic mouthwash containing benzydamine hydrochloride, diphenhydramine hydrochloride and diclonine was given to the patient.

DISCUSSION

There are no specific diagnostic tests for EM and the diagnosis is mainly clinical supported if necessary by biopsy. Biopsy of perilesional tissue, with histological and immunostaining examination are essential if a specific diagnosis is required.

Most cases of EM are self-limited, with lesions evolving over 1-2 weeks and subsequently resolving within 2-3 weeks. Patients who form keloids may be at higher risk. Hypopigmentation or hyperpigmentation may follow resolution of lesions.

Recurrence is common in EM (up to one-third of cases) but is not common in SJS/TEN. Failure to diagnose SJS early in the course may result in a premature discharge of the patient, with subsequent deterioration in patient's condition. Patients and parents, when appropriate, should be warned about potential long-term complications. In this case report, the lesions changed from an early papular erythema to the late target lesion consisting of a peripheral elevated erythematous area and a central depressed area. This "time-dependent" characteristic of lesions was in accordance with an earlier study on 35 subjects (Imamura, Horio 1992).⁶

A diagnosis of EM can be difficult to establish, and there can be a need to differentiate from viral stomatitides, pemphigus, TEN and the sub-epithelial immune blistering disorders (pemphigoid and others) (Marinho *et al.* 1999).⁷

The oral mucosa was the most affected mucosal region in EM with a predilection for the lip mucosa in this case report, which is in accordance with a previous study done on 22 subjects (Sanchis, Bagan 2010).⁸

Special Concerns

Pregnancy may contribute to the development of EM. EM is rare in children younger than 3 years. EM is rare in persons older than 50 years. EM is more common in younger males, whereas SJS/TEN occurs equally in the sexes with predominance in older patients.

CONCLUSION

EM minor/major, SJS and TEN represent a spectrum of immunologically mediated disorders that are often precipitated by infection or drug therapy. The exact pathogenic mechanisms of each disorder remain unclear. Patients can sometimes have resolution of the disease with various immunosuppressive, antimicrobial, and supportive strategies. Severe disease, however, can still lead

to significant long-term morbidity and mortality. As there remains no specific diagnostic test, early clinical recognition of disease remains essential to promptly initiate appropriate treatment.

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