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Case Report

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A CASE REPORT ON DICLOFENAC INDUCED ERYTHEMA MULTIFORME

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ABSTRACT

Introduction: Erythema multiforme (EM) is distinctive hypersensitivity syndrome characterized by skin and mucous membrane lesions. EM shows typical clinical patterns. Based on the severity and the number of mucosal sites involved, the disease has been sub-classified into EM minor and major. Case Report: A 36 years female developed Erythematous rash initially in lower limbs gradually progressed to trunk and upper limbs over duration of 6 days. Patient has a complaint of myalgia & sore throat prior to appearance of rash following 5 days of treatment with Diclofenac. There was history of ankle sprain for which she was prescribed tab Diclofenac by medical practitioner. Characteristic target lesions were present over upper and lower limb and trunk. The Nikolsky sign was negative.

Investigations revealed moderate leukocytosis. Diclofenac was stopped and patient was treated with Dexamethasone (4mg/day) over a period of 8 days and then gradually tapered off, chlorphenaramine malate (2mg/day) for 5days, soframycin for local application twice daily. Skin lesions starting healing with in a period of 15days. The rash resolved over 1month. Discussion: Case was diagnosed as Drug induced Erythematous multiforme minor. Conclusion: Prompt identification and withdrawal of the culprit drug and rapid initiating supportive care in an appropriate setting is the mainstay for the management of EM.

KEYWORDS: Erythematous multiforme, Diclofenac, Dexamethasone.

INTRODUCTION

Erythema multiforme (EM) is an acute, self-limited, and sometimes recurring skin condition that is considered to be a type IV hypersensitivity reaction associated with certain infections, medications, and other various triggers. [1] Erythema multiforme may be present within a wide spectrum of severity. Erythema multiforme minor represents a localized eruption of the skin with minimal or no mucosal involvement. The papules evolve into pathognomonic target or iris lesions that appear within a 72-hour period and begin on the extremities. Lesions remain in a fixed location for at least 7 days and then begin to heal. The hallmark of erythema multiforme (EM) is a target lesion with variable mucous membrane involvement. The initial lesion is a dull-red, purpuric macule or urticarial plaque that expands slightly to a maximum of 2 cm over 24-48 hours. [2,3] In the center, a small papule, vesicle, or bulla develops, flattens, and then may clear. An intermediate ring develops and becomes raised, pale, and edematous. The periphery gradually changes to become cyanotic or violaceous and forms a typical concentric, "target" lesion. Some lesions consist of only 2 concentric rings. Polycyclic or arcuate lesions may occur. Some lesions appear at areas of previous trauma (Koebner phenomenon). Postinflammatory hyperpigmentation or hypopigmentation may occur. The Nikolsky sign is negative. [4]

Erythema multiforme may be induced by medications, but infectious agents are also considered to be a major cause of Erythema multiforme.^[5] However, approximately 50% of cases are related to medication use.^[6]

CASE STUDY

Subjective Data: A 36years female came with complaints of Erythematous rash initially in lower limbs gradually progressed to trunk and upper limbs over duration of 6 days. Generalized Rash since 3days.

History

- Development of multiple fluid filled lesions associated with Erythema predominately over legs and few lesions on trunk
- Myalgia & sore throat prior to appearance of Rash.
 Past Medication history:
- Use of Diclofenac 100mg (Diclomol SR) over a period of 5 days for ankle sprain.

Objective Data

- Multiple bullae with clear fluid over Erythematous base distributed over both limbs and trunk extremities.
- Erythematous confluent rash over both potent medical aspect of both legs, trunk, limbs.
- Target lesions+ over both limbs & trunk.
- Bilateral pedal edema +
- Nikolsky sign is negative



The images shows the presence of target lesions.

Laboratory Test

The laboratory data reveals there is moderate Leukocytosis i.e 18,200 cells/mm³ the normal range is 5,000 to 10,000 cells/mm³. There is also slight decrease in hemoglobin value 11gm% (12-15.5gm%), Platelets was found to be adequate.

Treatment

Based on the subjective and objective data it was diagnosed as ERYTHEMA MULTIFORME.

Initially patient was given with the Deflazacort (glucocorticoid) was given over a duration of 1 day and then continued with the following treatment.

- Injection. Dexamethasone (corticosteroid) 4mg/day was given. It is an anti-inflammatory drug.
- o Injection. Chlorphenaramine Malate (Anti-histamine) for systemic relief.
- o Injection Pantoprazole 40mg/day was prescribed.
- o Soframycin ointment for local application was given to apply on lesion twice daily.

The same treatment was continued for 5 days and patient started recovering. Lesion started healing. Skin lesions starting healing with in a period of 15days. The rash resolved over

1month. Dexamethasone dose was then tapered off. After a period of 1month patient has discharged with no complaints. Further follow up of the patient to be done to prevent reoccurrence.

DISCUSSION

- Globally, the frequency of erythema multiforme is estimated at approximately 1.2-6 cases per million individuals per year [7]
- Erythema multiforme is currently more common in younger females (male-to-female ratio, range of 3:2)^[7,8]
- In this case, the patient was admitted to the hospital with Erythematous rashes over limbs and trunk.
- Drugs account for 65-80% of the cases. An immune mechanism is implicated in the pathogenesis but its nature is still unclear.
- Patient was prescribed with the anti-histamine and corticosteroid to reduce the hypersensitivity reactions.
- The patient started recovering with in a period of 15 days and the rashes resolved over 1month.
- o The patient was recovered completely in a month and got discharges from hospital.
- o Follow up for further to prevent reoccurrence.

CONCLUSION

Drug induced EM is a rare and less described variant. EM is often triggered rarely by adverse drug reactions. Even though primary attack of drug induced EM is confined to the lesion the subsequent attack can produce more severe forms of EM (EM minor, EM major) involving their skin.^[3] Prompt identification and withdrawal of the culprit drug and rapid initiating supportive care in an appropriate setting is the mainstay for the management of EM.^[8] Clinicians and clinical pharmacist should pay kind attention in such cases to also educate patients and their caretakers in recognizing and reporting the occurrences of any side effects.^[9]

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