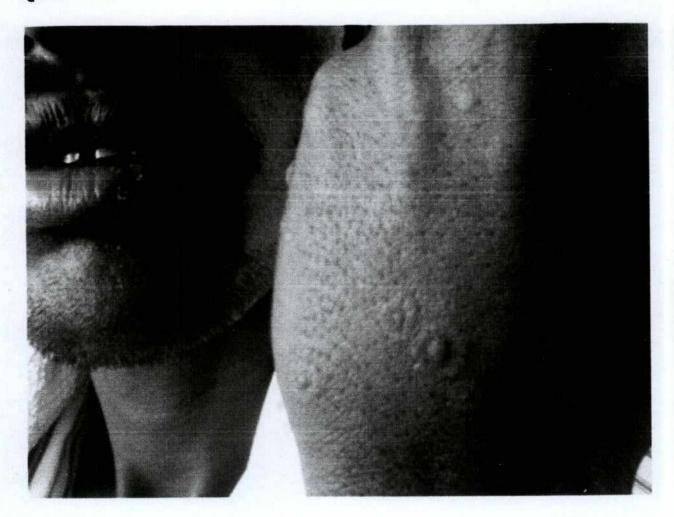
Quiz

PUB: 62/20/2



A 35yr old male patient came with history of multiple lesions over the extremities, trunk, and abdomen. Patient also gave history of recurrent vesicular lesions over the lips.

Examination revealed grouped vesicles with crusting over the angle of the left lower lip. Skin showed multiple erythematous well defined annular plaques 2-3cm diameter with three distinct zones (target lesions) over the extremities.

Q: What is your diagnosis?

Answer on page No. 100

Corresponding author:

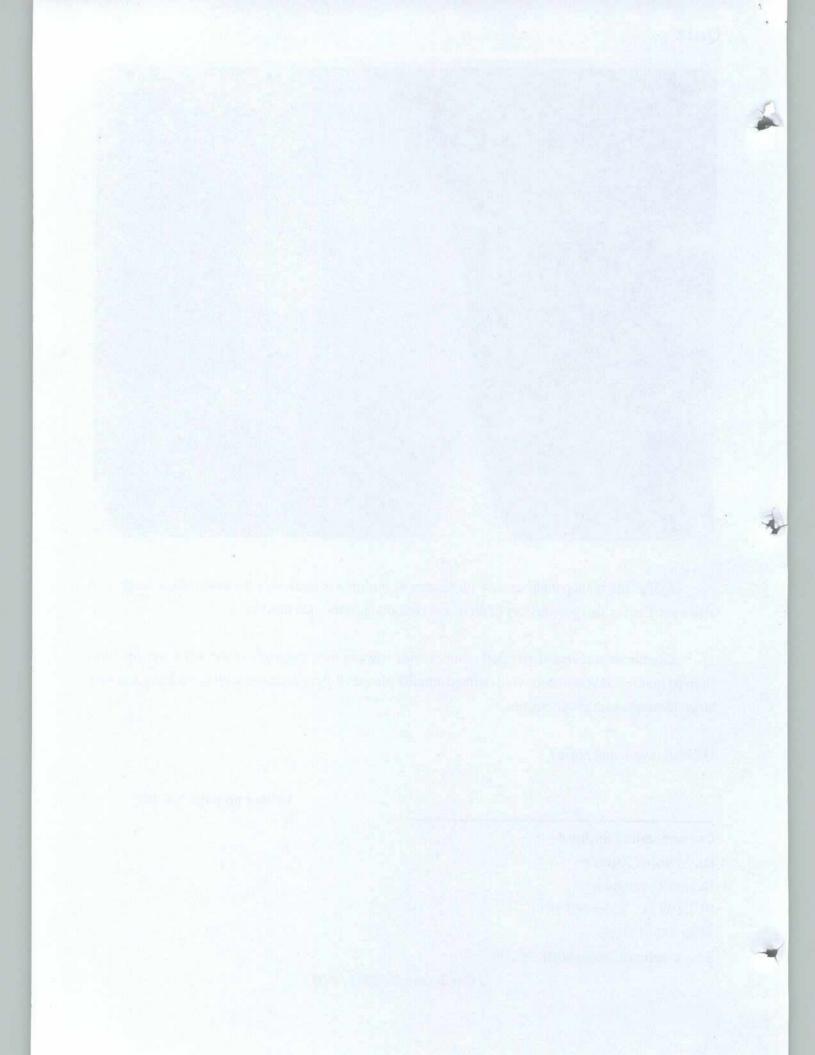
Dr. V. Shiva Kumar

Dept of Dermatology,

RLJH & RC, Kolar 563 101

Mob: 9886151969

Email: dermashiva@rediffmail.com



Answer to Quiz (Page No.97)

Erythema Multiforme

HISTORY

EM was first described by the Austrian dermatologist Ferdinand von Hebra in 1860. [1] The disease he described was mild with a sudden onset of hundreds of red papules. By daily observation, von Hebra recognized that some of the original papules evolved into lesions with concentric zones of colour change, which he termed 'target' lesions. [2]

ETIOLOGY

Causes of EM are far-reaching and most commonly include HSV infection, M pneumoniae, and systemic drugs. Other documented associations include countless infections (other than HSV or M pneumoniae), malignancy, connective tissue disease, immunization, radiation, inflammatory bowel disease, sarcoidosis, and menstruation. [1,3,5] In contrast, recurrent EM usually occurs secondary to HSV.

Other, rarely reported causes of recurrent EM include recurrent M pneumoniae infections, hepatitis C virus, polymorphic light eruption, and foodstuff (patch testing has indicated benzoic acid sensitivity)

It is estimated that 15-63% cases of EM are secondary to infection with herpes simplex virus (HSV). Like HSV, herpes associated EM is self limiting, often recurrent disorder that is clinically distinct from drug induced EM.

HSV

The herpes labialis may precede the onset of the cutaneous lesions, occur simultaneously,

or be evident after the target lesions of EM have appeared. Most commonly, herpes labialis precedes target lesions of EM by 3-14 days. It is presumed that most cases in children and young adults are due to HSV type 1, but documented cases of HSV type 2 in adolescents and young adults have been reported.

CLINICALFEATURES

Erythema multiforme is a polymorphic eruption composed of symmetrically distributed macules, papules, bullae, and target lesions that have a propensity for distant extrimities and oral mucosa.^[1]

The characteristic elementary skin lesion of EM is the *typical target lesion*. The latter measures <3 cm in diameter, has a regular round shape and a well-defined border, and it consists of at least three distinct zones, e.g. two concentric rings of color change surrounding a central circular zone that has evidence of damage to the epidermis in the form of bulla formation or crust. Such a typical target lesion is sometimes referred to as an 'iris lesion' because of its rainbow-like appearance.

In EM, a history of an abrupt onset of skin lesions is obtained, with almost all of the lesions appearing within 24 hours and full development by 72 hours^[2,4] The individual lesions remain fixed at the same site for 7 days or more.

DIFFERENTIALS.[5]

Many conditions may include the production of 'target-like' lesions and mimic EM, including the giant urticaria, fixed drug eruptions, sub-acute cutaneous LE, erythema annulare centrifugum, and several forms of

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vasculitis.

TREATMENT

- Usually self limiting.
- Symptomatic management with topical antibiotics for erosions and soothening agents.
- Oral Anti histamines for 3-4 days.
- Treatment of recurrent HSV-associated erythema multiforme, if started by the patientin the prodrome stage (with a 5-day course of aciclovir), will often prevent development of erythema multiforme. If that is not effective and attacks are frequent, a 6-month course of prophylactic aciclovir should be tried even in patients in whom HSV is not obviously a precipitating factor.

REFERENCE

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- 3. Schofield JK, Tatnall FM, Leigh IM. Recurrent erythema multiforme: clinical features and treatment in a large series of patients. *Br J Dermatol* 1993; 128:542-545.
- 4. DarraghTM, Egbert BM, Berger TG, et al. Identification of herpes simplex virus DNA in lesions of erythema multiforme by the polymerase chain reaction. *J Am Acad Dermatol* 1991; 24:23-26.
- 5. Huff JC, Weston WL. Recurrent erythema multiforme. *Medicine (Baltimore)* 1989; 68:133-140.

Source of Support: Nil Conflict of Interest: Nil

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