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).[1] 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.

CLINICAL FEATURES

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.*[1] 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.[1] Such a typical target lesion is sometimes referred to as an 'iris lesion' because of its rainbow-like appearance.[3]

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

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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