LETTERS TO THE EDITOR

ATYPICAL SUBCORNEAL PUSTULAR DERMATOSIS

To the Editor,

A 43-year-old male presented with a minimally itchy diffuse erythematous maculopapular eruption on the trunk (lateral aspects of chest and abdomen), neck and proximal upper and lower limbs of two days duration. He had taken eight tablets of furazolidone and four tablets of levamisole for diarrhoea two days prior to the onset of lesions. Dermatological examination revealed diffuse erythematous maculopapular eruption with ill-defined margins on aforementioned areas with complete sparing of axillary and groin flexures. Face and mucous membranes were not involved. Patient was comfortable except for low grade fever. At this juncture there were no pustules and, with the history, possibility of drug eruption was high. A day later discrete flaccid vesicles were seen progressing to vesico-pustules with characteristic hypopyon formation. Gram stain of pus from pustules showed large number of neutrophils and no bacteria. Histopathology was consistent with clinical diagnosis of subcorneal pustular dermatosis (SCPD) and there was dramatic improvement with dapsone.

Six of the seven patients initially described by Sneddon and Wilkinson were women and mean age of onset was 54.8 years.[1] However younger cases have been described in India in males.[2,3]

The eruptions tend to coalesce and produce annular, circinate or bizarre patterns over mainly axillae, groins and sub-mammary regions, abdomen and flexor aspects of limbs.

The atypical features of the case described are: (a) Male sex, (b) Younger age of onset, (c) Sudden onset, (d) Low grade fever, (e) Sparing of flexures, and (f) Lack of annular or circinate lesions.

The features of SCPD as depicted by this case can easily mimick a drug eruption, erythema multiforme and acute generalised pustular psoriasis of Von Zumbusch. Therefore SCPD should from a differential diagnosis of every case of generalised pustular dermatosis irrespective of distribution of lesions till proved otherwise.

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ONYCHOMADESIS IN STEVENS JOHNSON SYNDROME

To the Editor,

Loss or partial loss of the nail may result from a bullous eruption affecting the tips of the digits. Any drug that can induce bullae may cause nail changes or nail loss due to destruction of the nail matrix.[1] We report a case of onychomadesis and temporary shedding of the nails following Stevens-Johnson syndrome.

A 28-year-old male on treatment with antituberculous drugs (INH, rifampicin, thiacetazone) developed generalised pruritus, erythema and bullous eruptions. Bullae were seen over the trunk and extremities including the fingers and toes. Ocular involvement

manifested as congestion and discharge. Oral and genital erosions were also present. Stevens Johnson syndrome was suspected and the antituberculous drugs were withdrawn. Oral steroids (prednisolone-30mg/day) was started. The lesions subsided with desquamation and post-inflammatory hypopigmentation. Six weeks later, proximal separation and shedding of all the nails over the fingers and toes was observed. Clinically some nails showed onychomadesis and the rest of his fingers and toes showed anonychia. No treatment was advised and two months later new nails had started appearing.

Nail plate deformity and frequently complete shedding with scarring are seen in severe erythema multiforme type of drug reaction to sulphonamides, phenytoin, and barbiturates.[2]

Permanent anonychia after Stevens Johnson syndrome has been reported.[3] Temporary loss has been described due to large doses of cloxacillin and cephaloridine.[4] Onychomadesis has been observed in pemphigus vulgaris.[5]

In the case presented above, the temporary nail changes may be due to involvement of the proximal nail folds by vesicles, the inflammation being severe enough to overwhelm the nail matrix.

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HIGH INCIDENCE OF POLYMORPHIC LIGHT ERUPTION IN KOTA

To the Editor,

Polymorphic light eruption (PLE) is a common, intermittent, UVR-induced eruption characterized by nonscarrring, erythematous itchy papules, plaques or vesicles over exposed skin.[1] The severity of the disease is maximum during spring and summer and young females are more commonly affected than males.[1] Although all ethnic groups are affected, PLE has been found to be most common in temperate regions, affecting upto 10-20% of the population.[2,3] The disease is not uncommon in tropical countries. However, its exact incidence in India is unknown.

Over the last one year we have been seeing quite a number of cases of PLE in Kota. Of 3583 registered cases seen in the Skin out patient department of our hospital during October 1994 to November 1995, 384 (10.71%) cases were of PLE. This is quite a high incidence. The first author (SD) has the experience of working in Calcutta and Chandigarh but has not seen such a large number of case of PLE in those two cities. Neither such high incidence of this disease has been reported from other parts of India.

Kota is situated in the south-eastern part of Rajasthan with the maximum temperature varying between 47^0-50^0C during summer and minimum temperature 6^0-9^0C during winter. Sunlight is quite plentiful almost throughout the year except during the months of July and