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 Stevens Johnson syndrome was suspected and the antituberculous drugs were withdrawn.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

August.

The increased availability of UVR could be responsible for such high incidence of PLE. However, we do not come across other types of idiopathic photodermatoses like actinic prurigo, juvenile spring eruption, solar urticaria more frequently in our population. Therefore, some factor other UVR must be playing a significant role in precipitating the disease in this region.

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BART SYNDROME

To the Editor,

Bart syndrome with autosomal dominant inheritance represents a distinct disease where in addition to cutaneous bullae, congenital localized absence of skin (CLAS), mucosal ulcers, deformities or absence of nails and deformed teeth are present.[1,2] Bart syndrome has been reported from India.[3]

Three out of 6 siblings from a Bihari muslim family suffered from Bart syndrome. Siblings number 2,4 and 6 had disease and it confirmed the autosomal dominant inheritance pattern.

Case no.1 was a 6-year-old male who had a large, wide linear ulcer on right shin, foot, thigh and second ulcer near left knee. Ulcers healed after 3 to 4 months with residual hyperpigmentation and superficial scarring. Bullae on skin and mucosae of mouth developed since 2 months of age. Skin over sides of scalp, clavicular fossae, axillae, groins and iliac fossae, showed bilateral symmetrical, pigmented, wrinkled skin interspersed with irregular hypopigmented macules and bullae appeared intermittently in these areas. In addition bullae of nail folds, pulps of fingers and toes were seen. Initially bullae had clear fluid which later turned haemorrhagic. Bullae increased in size for 2-3 days, ruptured leading to superficial ulcers which healed rapidly in 4-5 days without any scars. Bulla spread and Nikolsky's signs were negative. All nails of hands and feet were deformed and partially dystrophied. Bullae on tongue, lips and buccal mucosae were observed which ruptured quickly leaving behind thick whitish surface. Teeth were normal upto 3 years of age and later premature partial or total shedding of teeth was seen. Although milestones were delayed yet child had average intelligence and built. Child was product of normal vaginal delivery in a nonconsanguineous marriage. General physical and systemic examinations were normal. Routine investigations were normal except anaemia. Histopathologically big split in relation to dermoepidermal junction with band like dense collection of mononuclears admixed with eosinophils, plasma cells and occasional PMNL beneath it were seen (Fig.1). PAS stain revealed PAS positive basement membrane at the floor of bullous cavity. Case no.2 was a female with similar scar and pigmentation on right knee, shin and foot from healing of ulcers of CLAS. She had similar mucosal lesions and bullae to case 1. Pigmentation interspered with irregular hypopigmented macules was less prominent. Nail deformity was mild and teeth were normal.