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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References

1. Hawk JLM. Cutaneous photobiology. In : Champion RH, Burton JL, Ebling FJG, eds. Textbook of dermatology. Oxford: Blackwell, 1992: 856-9.
2. Morison WL, Stern RS. Polymorphous light eruption: a common reaction uncommonly recognised. Acta Derm Venereol 1982; 62; 237-40.
3. Ros A, Wennersten G. Current aspects of polymorphous light eruptions in Sweden. Photodermatol 1986; 3: 298-302.

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.

Fig. 1. Big bulla at dermoepidermal junction with basal cell forming the roof of bulla and prominent mononuclear infiltrate at the base of the bulla (H&E, x100).

Third patient who was 10 years old was not seen by us. Distribution of bullae in our cases was distinct and can help in diagnosis of Bart syndrome. Degeneration of basal cells was not seen and therefore bullae of Bart syndrome were different from epidermolysis bullosa (EB) simplex. CLAS and EB are rare congenital skin diseases with unknown aetiology and their simultaneous presence suggests that they may be related.

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References

1. Bart BJ, Gorlin RJ, Anderson EV, et al. Congenital localized absence of skin and associated abnormalities resembling epidermolysis bullosa. A new syndrome. Arch Dermatol 1966:93:296-304.
2. Bart BJ. Epidermolysis bullosa and congenital localized absence of skin. Arch Dermatol 1970; 101:78-81.
3. Gaikwad AK, Shende R, Khedkar MY. Bart syndrome. Ind J Dermatol Venereol Leprol 1993; 59:151-3.

BLISTER BEETLE DERMATITIS

To the Editor,

A clinicopathological appraisal of 54 cases of an unusual, seasonal erythemato-vesicular dermatitis seen in post monsoon two and a half months period in the local population of Jodhpur was done. The age ranged between 15 months to 70 years. The common sites of involvement were the exposed parts of the body mainly face and extremities. The main presenting symptoms in majority of the cases were burning sensation, pain and itching of variable intensity. The spectrum of the lesions seen in this study were linear, herpetiformis, zosteriform, patchy and bizzare. The causative beetle could not be found out, however, patients gave positive history of some bite. As extensive study of beetle dermatitis has been done by Handa et al.[1]

The skin biopsy obtained showed characteristic spongiosis and necrosis of superficial epidermal cells with a variable density of focal round cell infiltration in upper dermis.

All the patients responded well in 4-5 days to topical application of emollient creams with no sequlae.

It is being stressed that the condition be kept in differential diagnosis while examining erythemato-vesicular lesions specially on exposed parts during rainy season.

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