skin faculty. This specific reaction may get stalled in vitiligo patients as their ability to sweat off excess toxin concentration is lost. In acquired hostile environment the colour determinant may undergo structural modification through conjugation with the available toxins with complementary surfaces at the vacant spaces as predicted by Sawhney,[2] losing its property to impart natural shade to epidermis. The resultant structural crisis sets in the trigger mechanism of the pigment dilution in the stratum corneum with slow progression with the percutaneous diffusion of these toxins in skin matrix.

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CLINICAL PROFILE OF PSORIASIS IN WESTERN RAJASTHAN : STUDY OF 300 CASES

To the Editor,

Psoriasis is a common genetically determined, chronic recurrent papulo-squamous dermatosis, characterized by circumscribed, erythematous patches of various sizes covered with silvery white scales. The lesions tend to become confluent and may persist indefinitely. The disease is unpredictable and capricious in its course but is usually chronic.[1]

A total of 300 cases of psoriasis from western Rajasthan were evaluated clinically in a period of one year ie, January to December 1994. Total outdoor registration was 62261 and male to female ration being 1.6:1. Incidence of psoriasis was 0.48% while male to female ration was 3:1. Most of the patients (205;68.34%) had onset of their disease in second to fourth decade of life. Youngest patient was 6 months while oldest was 79 years old. Maximum number of cases were of psoriasis vulgaris (225;75%), second commonest being palmoplantar psoriasis (17;5.67%), followed by sebopsoriasis (14;4.67%) etc.

In our study itching was experienced by 259 (86.33%) of patients while only 41 (13.67%) were asymptomatic, similar observation has been made by others.[2] The incidence of diabetes mellitus in psoriasis reported in literature is 2.4% to 5.7% .[3,4] Similar observation was made in 8 (2.67%) patients in our study. We also noted coincidental diseases in family members of the patients and observed that there was diabetes mellitus in 11 (3.67%) and vitiligo in 6 (2%). Therefore we hypothesise that these three conditions may be interrelated and probably having similar genetic predisposition. The paucity of such a study in literature from this region prompted us to undertake this work

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HYPOHIDROTIC ECTODERMAL DYSPLASIA IN TWO SIBLINGS

To the Editor,

Ectodermal dysplasia is a rare condition occurring in an estimated 1/100,000 live births. It embrases several abnormalities whose common denominator is a morphological alteration of ectoderm derived organ and tissues.

Case 1, an 11-year-old Hindu boy born of first degree consanguinity, presented with inability to sweat, heat intolerance and absence of teeth. Positive findings on examination were sparse fine hair over scalp, absent eyebrows and lashes, frontal bossing, saddle nose, prominent chin, protruding lips, hyperpigmentation over periorbital and perioral area with photophobia, cone shaped teeth which were two in number. Skin was thin and dry, nails were normal, systemic examination was normal. Case 2 was preterm female baby delivered at 8 months gestation, younger sibling of Case 1 and was seen on first day for sparse and scanty hair, wrinkled eyelids and absence of eyebrows and eyelashes, nails were normal.

The facial features of individuals suffering from ectodermal dysplasia are consistently similar.The typical facies is characterised by frontal bossing, malar hypoplasia, flattened nasal bridge, reccessed colemella, thick everted lips, wrinkled hyperpigmented periorbital skin and prominent low set ears as was seen in Case 1. Skin over entire body is dry and hypopigmented, hair is sparse, unruly and lightly pigmented and eyebrows and eyelashes are sparse or absent, as was noted in both the cases. Anodontia or hypodontia with widely spaced peg shaped teeth are consistent features as was seen in Case 1.

In most of the cases, hypohidrotic ectodermal dysplasia is inherited as x-linked recessive trait with full expression only in male, however an autosomal recessive mode of inheritence may be operative in some families,[1] as probably occurred in the reported family.

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ECTHYMA GANGRENOSUM WITHOUT BACTEREMIA

To the Editor,

A 30-year-old otherwise healthy housewife presented with two coin-sized punched out ulcers covered with grey-black eschar and surrounded by an erythematous halo over her right scapular region of 2 weeks duration without any preceding history of trauma or drug intake. It started as a painless red macule which enlarged, evolved into a haemorrhagic bulla which ruptured forming a gangrenous ulcer. Ulcers were non-tender and mobile over the underlying structures. Her vital parameters and systemic examination were normal and there was no evidence of bacteremia in the patient.

Haemogram, urinalysis, X-ray chest, blood sugar, VDRL test, ELISA for HIV, peripheral blood smear and blood culture were normal. Smear and culture from the skin lesions revealed isolation of P aeruginosa; and biopsy from the ulcer revealed bacterial vasculitis with a dense bacillary infiltration of the media and adventitia of blood vessels consistent with the diagnosis of ecthyma gangrenosum.

The four major dermatologic manifestations of severe systemic Pseudomonas aeruginosa infection are ecthyma gangrenosum, vesicular lesions,