pemphigoid. For want of facilities immunofluorescence studies were not done.

Initial therapy for 2 weeks with dapsone 100 mg, once daily didn't show any response. Later therapy with oral tetracycline (500 mg tds) was started. Within two weeks appearance of new lesion stopped and the older lesion began to heal. Total clearance of the lesions was noted by the end of fourth week. Same dosage was continued for another 2 months and then reduced by 500 mg every fortnight. Five months followup after discontinuation of tetracycline did not show any relapse. No toxicity and morbidity was noted.

Systemic steroids remain the mainstay of therapy for generalised bullous pemphigoid. To reduce the dose of steroid and their side effects, immuno-suppressive agents such as azathioprine, cyclophosphamide, methotrexate, or chlorambucil are frequently used.[3]

Therapy with systemic steroids in an elderly debilitated patient has considerable potential toxicity. Topical steroids have generally been reserved for localized disease. Combination of tetracycline and niacinamide may also control moderate or severe bullous pemphigoid.[4] Recently treatment of generalized bullous pemphigoid with oral tetracycline and midpotency topical steroid cream has also been reported.

Tetracyclines suppress inflammation by inhibiting neutrophil chemotaxis and random migration in vitro and in vivo.[5] In bullous pemphigoid it may inhibit the complement-mediated inflammatory response to the basement membrane zone and mediators of the inflammatory response.[3] In addition, tetracycline has been shown to affect the cohesion of the dermoepidermal junction.[6]

It is worth trying a more benign treatment first rather than using systemic steroids or immunosuppressive agents, especially in bullous pemphigoid patients with associated medical problems.

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SUBCORNEAL PUSTULAR DERMATOSIS

To the Editor,

Subcorneal pustular dermatosis (SCPD) is documented to be a disease of middle-aged women. Six of the seven patients initially described by Sneddon and Wilkinson were women and the mean age of onset was 54.8 years.[1] However. most of the cases reported in India are in a relatively younger age group and are males[2,3] as in the following report.

A 45-yerar-old male patient presented with recurrent vesiculopustular lesions localised predominantly on axillae and groins, not associated with itching, fever or any systemic complaints of 10 years duration. He had been treated with various topical and systemic antibiotics in past and response was marginal

and temporary. Routine urine and haematological investigations were within normal limits. ASO titre was negative and pus swab from lesions did not grow any pathogen. Histopathology confirmed the diagnosis of subcorneal pustular dermatosis and significant regression of lesions was evident after dapsone therapy.

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FOCAL DERMAL HYPOPLASIA (GOLTZ SYNDROME)

To the Editor,

Focal dermal hypoplasia, also known as Goltz syndrome is characterized by widespread dysplasia affecting the tissues derived from embryonic ectoderm and mesoderm, with a striking underdevelopment of the dermis.[1] A 12-year-old girl was seen with hypopigmented and hyperpigmented macules and scars arranged in linear and retiform patterns bilaterally on the trunk (Fig.1). At birth she had a few linear raw areas on the skin of chest that healed in 6 months leaving atrophic, depigmented scars. Other anomalies noted were: a linear hyperpigmented, depressed atrophic area on the forehead, notching of the alae nasi on the left side, and macrochelia of lower lip (Fig.2). There were multiple, soft, compressible swellings on the chest that resulted from herniation of fat through the underdeveloped skin. Other congenital anomalies seen in her included complete

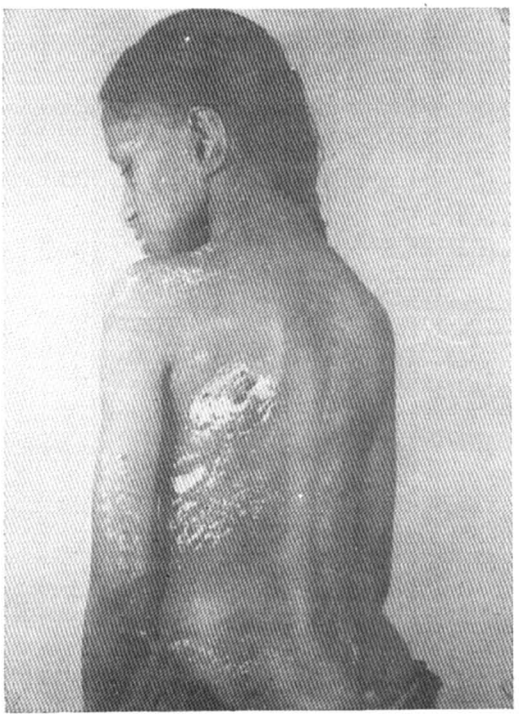

Fig 1. Goltz syndrome. Note linear,depigmented and hyperpigmented macules on trunk.

syndactyly of the left middle and ring fingers, microphthalmia with coloboma of the iris on the left eye, lumbo-sacral lordosis and a high-arched palate.

Routine laboratory tests on blood, urine and stools were normal. X-ray of the spine revealed spina bifida occulta of thoracic vertebrae (T_1 and T_2). Histology of the atrophic leasion on the chest revealed marked reduction in thickness of the dermis, the adipose tissue extending almost to the level of the papillary dermis. All the clinical features suggested a diagnosis of Goltz syndrome in this patient. Macrochelia and high arched palate were the additional features seen in our patient. The skin lesions in Goltz syndrome follow the course of Blaschko's lines, consistant with mosaicism secondary to lyonization of the affected X-chromosomes.[2]