appears to be inhibition of polymorphonuclear (PMN) cell activity, notably chemotaxis. But in PLCA, inflammatory cells are scarce or absent. So colchicine probably does not act via its usual mode of action. i.e. through suppression of PMN function in PLCA.

Now we can have a glimpse of the origin of macular and lichen amyloidosis. In these cases amyloid originates from degenerated epidermal cells in susceptible subjects. Epidermal origin of amyloid has been confirmed by electron microscopic study and positive staining with antikeratin monoclonal antibody. Degenerated epidermal cells contain tonofilaments and lysosomes. It may be assumed that degenerated tonofilaments are recognised as foreign bodies by cells own lysosomal enzymes. Such digestion produces amyloid filaments by conversion of alpha-pleated sheet configuration of tonofilaments to beta-configuration of amyloid. This amyloid gets deposited extracellularly in close apposition to basal layer of the epidermis and contains a few melanophages.

Now, we can conclude that colchicine probably blocks the release of lysosomal enzymes within the degenerated epidermal cells thereby preventing conversion of tonofilaments into amyloid. Preexisting amyloid is cleared by body's own digestive mechanism.

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DRY, SCALY DERMATITIS OF SCROTUM

To the Editor

We often see patients having pruritic scaly dermatoses of scrotum. Scrotal skin becomes dry with dirty brown scales. Lesion often has mild serosanguinous discharge which soon dries up to form brown crusts. Whole area becomes erythematous and sometimes telangiectasia may be seen. Patients are adult males concerned by intense itching and often burning sensation of scrotum, particularly at bed time. They suffer for months or years together with relapse and remission. Area involved is diffuse often spreading to the undersurface of penis upto prepucial margin. Scales are loosely attached, some seem to enjoy picking of scales. Patients are commonly seen to be under stress and may be depressed.

In a minority of patients seborrhoeic dermatitis involving classical areas are seen, who readily respond to topical hydrocortisone, while in a few patients oro-oculo-genital syndrome is a feature due to riboflavin and/or zinc deficiency.[1] Riboflavin and other vitamin B complex deficiency occasionally produce scrotal dermatitis, perleche, sore lips, tongue, and mouth.[2]

In differential diagnosis, lichen simplex chronicus has a well-defined margin. Diffuseness of the lesion and lack of involvement of other areas of the body excludes psoriasis. Sparing of the groins and negative fungal scraping excludes dermatophytosis.

Regarding treatment, topical corticosteroid in ointment base gives temporary relief of symptoms but often invites secondary fungal infection. In cases of riboflavin

deficiency 5-15mg. riboflavin two times daily for two weeks is curative. Simple emollients make skin moist, reduce intensity of itching. Tricyclic antidepressant doxepin 25-50mg. per day helps some patients. Anxiolytic alprazolam or antipsychotic thioridazine is helpful in some. Systemic or topical therapy has to be given intermittently for a long time.

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ANNULAR ERYTHEMATOUS LESION SECONDARY TO IMMUNOTHERAPY

To the Editor

Immunotherapy is a relatively safe treatment. However systemic and cutaneous side effects can occur.[1] We report a case of annular erythematous lesion at the injection site in a patient receiving immunotherapy.

A 36- year- old male photographer was referred to the Dermatology department for evaluation of a skin rash following desensitization injections. He was suffering from allergic rhinitis since 8 years and had been prick tested with 22 allergens consisting of mite, pollen, fungi, insects, dusts, danders and foods obtained from Allergen Division Curewell (India) Ltd. He was tested positive for the following : Pollens - *Chenopadium ablum, Ricinus communis , Cassia siamea;* insects - Male and female cockroaches; dog epithelia; culvularia fungus and house dust mite. Immunotherapy was commenced with a mixture of allergens.

The patient initially received two injections per week from a vial containing 1:5000 dilution of the solution without developing any side effects . When injections with 1: 500 solution were commenced he developed pruritic erythematous papular lesions in an annular fashion around the injections site. The lesion used to appear within 24 hours and resolve completly within 10 days without any residual pigmentation or scarring. He developed this lesion following each injection taken weekly. No lesions appeared in other parts of the body and there was no history of angioedema. A skin biopsy revealed epidermis with foci of mild spongiosis and exocytosis of inflammatory cells. Dermis showed moderate perivascular and peri-follicular lymphocytic infiltrate.

Various dermatologic manifestations following desensitization treatment reported include local urticarial reactions which are by far the most common,[2] others being digital vasculitis,[3] persistent, itchy subcutaneous nodules[4] and cold urticaria.[5] In our case the skin lesion could have been a delayed hypersensitivity reaction as the patient developed it later in the course of therapy, sensitization having been induced with the initial injections.

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