subcutaneous tissue helped us to clinch the diagnosis.

The cause of deposition of calcium in idiopathic calcinosis is not known.[1] Mild degenerative changes in the connective tissue; lowered CO^2 tension in the tissues which reduces the solubility of calcium, may be the predisposing factors.

Disodium EDTA is reported to produce favourable results.[4] The efficacy of corticosteroids is doubtful. Surgical removal of painful nodular deposits may offer temporary relief. We gave our patient a low calcium diet which is often of great help specially if combined with cellulose phosphate. As the patient was lost to follow up, the ultimate outcome is not known. The prognosis however is generally poor.

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EXTENSIVE TINEA CORPORIS CAUSED BY *T VERRUCOSUM* IN A PATIENT OF HIV INFECTION

To the Editor,

A 30-year-old man residing in suburban Madras was referred to the Department of Dermatology, Madras Medical College, Madras, for the treatment of scaly, patchy skin lesions throughout the body suggestive of tinea infection. He was subsequently found to be seropositive for HIV. Skin scrapings were collected for direct microscopy and culture. KOH examination showed the presence of fungal hyphae and chlamydospores. Culture on Sabouraud's dextrose agar (SDA) was very slow growing with growth appearing after 60 days. The colony was slightly folded, heaped glabrous and grayish brown in colour. No pigmentation was observed on the reverse side of the colony. The colony morphology on SDA, enhanced growth with thiamine and inositol, rapid hydrolysis of casein were suggestive of *Trichophyton verrucosum.*

Lactophenol cotton blue preparation of the fungus grown on SDA showed distorted hyphae with sparse antler like branching and chlamydospores in chains. The fungus grown on enriched media showed tear shaped microconidia and rat-tail shaped macroconidia. The macroconidia had 3-5 cells and was shaped like a string bean. Microscopy confirmed the identity of the fungus as *Trichophyton verrucosum.*

Trichophyton verrucosum is more frequently associated with cattle ringworm disease. However, isolation of *Trichophyton verrucosum* from human ringworm disease has been reported from India.[1,2] Klokke et al[3] have reported the isolation of this species from human ringworm disease in South India. However, in previous reports, the infection was described to be erythematous, inflammatory, pustular, localized lesions which responded to treatment. In the present study we report the isolation of *Trichophyton verrucosum* from a case of extensive tinea corporis in a HIV+patient. The lesion was non-

pustular and non-inflammatory with scaling and absence of a well-defined margin.

Immunosuppression due to HIV infection might have led to this chronic non-inflammatory, non-pustular, extensive infection caused by a zoophilic species. Perhaps, this is the first report of an extensive, non-inflammatory tinea corporis caused by *Trichophyton verrucosum.*

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BENIGN FAMILIAL CHRONIC PEMPHIGUS IN A DIABETIC

To the Editor,

A 48-year-old male patient presented with recurrent vesicular eruptions on an erythematous base with a surrounding zone of hyperpigmentation appearing over axillae, groin and later on over neck and cubital fossa with no involvement of mucosal surfaces. The complaint dated back to 5 years. The course was one of remissions and recurrences. On routine hematological examination and urinalysis patient was detected to be a diabetic.

Patient did not respond to antibiotics like tetracycline and erythromycin, topical steroids and antifungals, although his diabetes was controlled by oral antidiabetics. Later on dapsone was started and the patient improved remarkably as has been noted by other authors.[1] According to the patient his late father had similar history of recurrent lesions over his neck and flexural sites, and his only sibling was unaffected. Histopathological examination of the biopsy specimen showed features consistent with clinical diagnosis of Hailey-Hailey disease.

Benign familial chronic pemphigus (Hailey-Hailey disease) is transmitted through an autosomal dominant gene with incomplete penetrance with a family history in 70% of the cases. In our case an autosomal dominant mode of inheritance is suggested. Although non insulin dependent diabetes mellitus is known to run in families, its mode of inheritance is not known and in our case no definite family history of diabetes could be obtained. So the occurrence of diabetes mellitus in our case might be an association or is fortuitous.

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ORAL LICHEN PLANUS CAUSED BY DENTAL AMALGAM

To the Editor,

Lichen planus and lichenoid lesions are known to be provoked by many chemicals and drugs. Dental metals like mercury and silver have been implicated in the aetiopathogenesis, probably due to contact allergy[1,2] although an electrogalvanic effect has also been