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.*

S Arun Mozhi Balajee, Thangam Menon, S Ranganathan, Thirunavukkarasu
Madras

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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.

J N Dave, S V Shah, N S Vora, K Roy, A Ghosh, B J Cardoso
Ahmedabad

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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

postulated.[3,4] We report a patient who developed oral lichen planus following dental fillings with amalgam containing mercury. A 19-year-old male presented with reticulate bluish white lesions on both buccal mucosa of 2 years duration, in relation to teeth filled with amalgam 4 years ago. Patch testing with dental series (Chemotech AB, Sweden) using Van der Bend chambers showed a positive reaction to elemental mercury (1%) in petrolatum.

Histopathology showed features of LP with basal cell degeneration and band of inflammatory infiltrate in upper dermis. A standard direct immunofluorescence showed a ragged fibrin basement membrane zone band. Fibrin band on immunofluorescence has been reported in LP.[5]

The amalgam fillings were replaced with an inert posterior composite. Two months later the lesions had subsided and patient is asymptomatic.

A diagnosis of oral LP was made based on clinical features, histopathology and immunofluorescence. As the patient was sensitive to mercury and as the lesions appeared following the amalgam filling and subsided following its removal we further feel that in this case mercury in the dental amalgam could have been the precipitating or provoking factor. In a case of oral LP with dental fillings we therefore recommend patch testing with relevant metals, removal of amalgam filling if found positive and replacement with an alternative material.

Sandra A, CR Srinivas, Sathish Pai, Keerthilatha Pai, Nirmala R
Manipal

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TOPICAL TRIAMCINOLONE ACETONIDE IN AN INDIGENOUS ORABASE IN ORAL LICHEN PLANUS

To the Editor,

Oral lichen planus (LP) affects upto 1% of the population.[1] It is about eight times more common than cutaneous LP.[1] The treatment of oral LP is a therapeutic problem. Topical steroids in a conventional cream base do not adhere to the oral mucosa for a sufficiently long time to cause therapeutic action. Use of intralesional corticosteroids, though effective, has the drawback of pain at injection sites and risk of secondary infection. Other therapeutic modalities include oral vitamin A,[2] topical cyclosporine (100 mg/ ml) in the form of an oral rinse[3] and temarotene,[4] a new oral retinoid. Orabase[5] (a gel of carboxymethylcellulose, pectin and gelatin), available commercially in the West, is an ideal vehicle for topical corticosteroids for oral mucosa. We have developed an indigenous orabase and used it as a vehicle for triamcinolone acetonide (40 mg/ml). This was prepared by adding Vi syneral syrup (30 ml) and Moisol (hydroxypropyl cellulose) eye drops (10 ml). Triamcinolone acetonide (40 mg/ml)