

CORRESPONDENCE

This department is for the publication of informal communications that are of interest because they are informative and stimulating, and for the discussion of controversial matters. The mandate of this JOURNAL is to disseminate information relating to leprosy in particular and also other mycobacterial diseases. Dissident comment or interpretation on published research is of course valid, but personality attacks on individuals would seem unnecessary. Political comments, valid or not, also are unwelcome. They might result in interference with the distribution of the JOURNAL and thus interfere with its prime purpose.

Milia in Leprosy

TO THE EDITOR:

Milia are superficially located, whitish, globoid firm lesions 1–2 mm in size⁽³⁾. Primary milia arise spontaneously on the face in predisposed individuals and are derived from the lowest portion of the infundibulum of the vellus hairs. Secondary milia may develop from any epithelial structure, such as a hair follicle and an eccrine sweat duct⁽³⁾. We report two cases of milia following a subsiding type 1 leprosy reaction.

Case 1

A 50-year-old male with mid-borderline Hansen's disease on treatment with multidrug therapy (rifampin, clofazimine and dapsone) developed erythematous nodules and plaques over his face, trunk and extremities. Type 1 reaction was diagnosed and prednisolone (30 mg/day) was started.

The patient was reviewed at regular intervals. After 4 months of therapy all lesions had flattened and there was post-inflammatory pigmentation and atrophy. Multiple, asymptomatic, pin-head-size, whitish papules were seen overlying the subsiding reaction lesions, sparing the normal skin (Fig. 1). Milia was diagnosed clinically and the lesions were extracted. However, a biopsy was not performed.

Case 2

A 24-year-old male diagnosed to have borderline tuberculoid Hansen's disease on treatment with multidrug therapy (dapsone and rifampin) developed reddish plaques over the face, ears, trunk and extremities with a right-side foot drop. Type 1 reaction

was diagnosed and prednisolone (30 mg/day) was started. On reviewing the patient 4 months later, all of the lesions had flattened. Multiple milia were seen over the ears and the subsiding lesions (Fig. 2). Extraction was advised.

Secondary milia represent retention cysts caused by proliferative tendencies of the epithelium after injury⁽³⁾. In our case, the

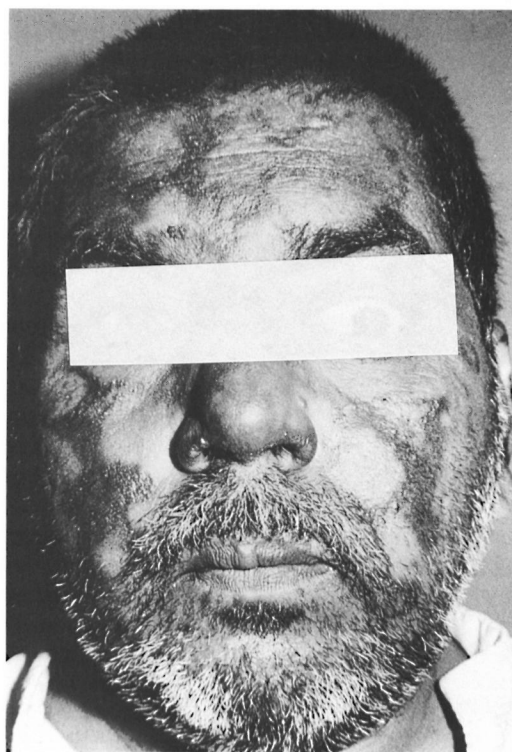


FIG. 1. Milia over subsiding lesions on the face.

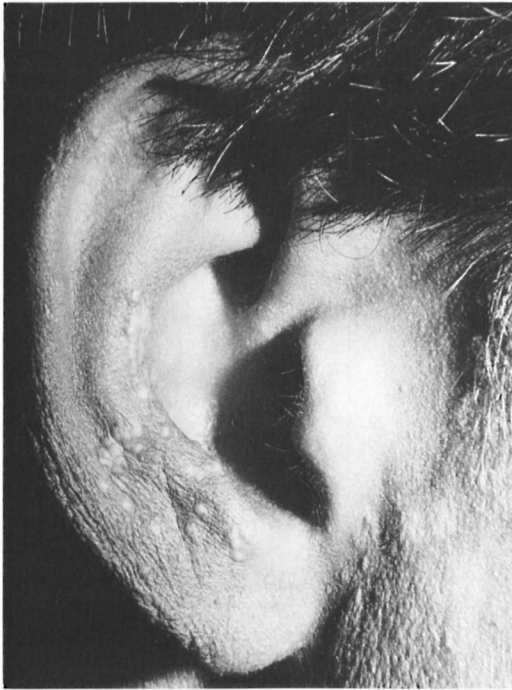


FIG. 2. Milia over the ears.

reaction could lead to epidermal damage, and the subsequent repair could be responsible for the development of milia. Secondary milia often is seen in diseases associated with subepidermal bullae, such as bullous

pemphigoid, dystrophic epidermolysis bullosa, porphyria cutanea tarda and lichen sclerosus et atrophicus, after dermabrasion, and following trauma (2). It also has been noted after topical steroid therapy (4) and topical 5 fluorouracil therapy (1).

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HIV-1 Infection and Leprosy

TO THE EDITOR:

This letter is to call attention to mounting evidence that HIV infection is a risk factor for leprosy. We published in 1989 that SIV (simian immunodeficiency virus), the sooty mangabey monkey equivalent of the human AIDS virus (which is virtually identical to the human AIDS virus, HIV-2), increases the susceptibility of rhesus monkeys (*Macaca mulatta*) to experimental leprosy, and that there is an increased incidence of multibacillary cases in SIV-coinfected monkeys. Subsequently, we reported on the pathology of such dually infected rhesus monkeys. Our publications are as follows:

B. Gormus, *et al.*, Interactions between simian immunodeficiency virus and *Mycobacterium leprae* in experimentally-inoculated rhesus monkeys. *J. Infect. Dis.* **160**:405–413 (1989) and G. Baskin, *et al.*, Pathology of dual *Mycobacterium leprae* and simian immunodeficiency virus infection in rhesus monkeys. *Int. J. Lepr.* **58**:358–364 (1990).

In recent years, two reports of which we are aware published observations dealing with the same subject matter in humans. The first, by J. Ponnighaus, *et al.* (Is HIV infection a risk factor for leprosy? *Int. J. Lepr.* **59**:221–228, 1992) observed no increased risk for leprosy among HIV-posi-