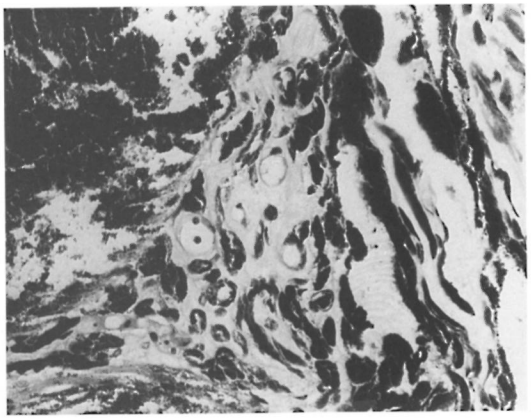

FIG. 2. Photomicrograph showing blood vessels with thick hyalinized walls characteristic of those normally found in the iris. A few scattered pigment-filled macrophages are also seen (H&E ×200).

gination of neutrophils. No AFB were seen in any of the sections.

The histopathological demonstration of profuse thin-walled capillaries (Fig. 1) in the iris of this patient was inconsistent with the usual vasculature seen in the normal iris. The vessels of the normal iris appear thick walled, with the endothelial lining having a thick collar of collagen fibrils (Fig. 2).

Cavernous hemangioma occurring in the iris of a leprosy patient has been reported ([2]). The histopathology in this patient suggests the occurrence of a capillary hemangioma which, although by itself not significant, is an uncommon feature ([1]). Since the probability of a prominently vascularized tumor of the iris being a true hemangioma is low ([3]), and the patient had a history of having had iridocyclitis in the left eye and histology demonstrated features of chronic inflammation with lymphocytes and plasma cells infiltrating the constrictor pupillae muscle, it is possible that the abnormal vasculature could be that of a fibrovascular proliferative response to inflammation. Whatever be the etiology of the capillary proliferation, we are reporting it because of the rarity of its manifestation.

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Chromoblastomycosis Simulating Lepromatous Leprosy

TO THE EDITOR:

Chromoblastomycosis is a chronic infection of the skin caused by any of the several species of dematiaceous fungi. In most cases it begins on the extremities, particularly the feet. We report a woman who developed multiple plaques of chromoblastomycosis on the skin of the cheek and earlobe and mucosae of the nostrils and nasal septum. The buccal branch of right facial nerve also was involved, resulting in paralysis of the orbicularis oris. All of these features closely simulated leprosy clinically, and the patient was wrongly diagnosed and treated as having lepromatous leprosy by a general practitioner.

A 42-year-old widow developed multiple papules on the right earlobe, right cheek and mucosa of the nostrils 3 months follow-

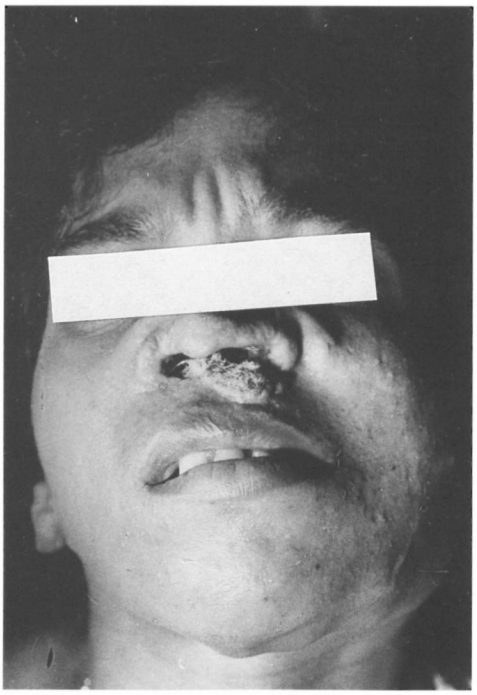

FIG. 1. Verrucous plaque of chromoblastomycosis on the face extending into the nasal cavity causing destruction of the nasal septum. Note facial asymmetry due to paralysis of the buccal branch of right facial nerve.

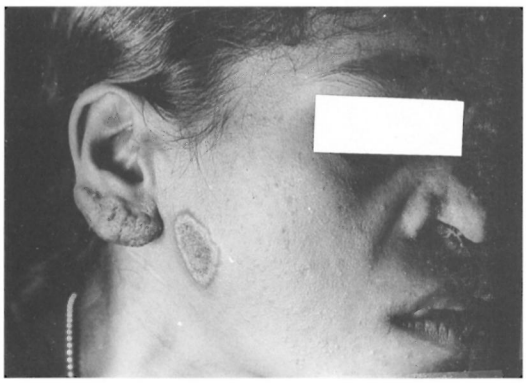

FIG. 2. Infiltration of the earlobe, an annular plaque on the cheek and destruction of ala nasi.

ing an injury from thorns from a cactus. The papules increased in size and spread peripherally to form an oval annular plaque of 4 × 3 cm on the cheek, a diffuse, infiltrated plaque on the right earlobe and an erythematous, verrucous plaque and ulceration of lower part of the cartilagenous portion of the nasal septum and right ala nasi (Figs. 1 and 2). There was no sensory deficit in the plaques and the oral cavity was normal. There was no nerve thickening but the buccal branch of the right facial nerve was affected, causing paralysis of the orbicularis oris of that side (Fig. 1). All other cranial nerves were normal. There were no other skin or mucosal lesions. All systems were clinically normal.

Routine laboratory tests on blood, such as total and differential leukocyte count, hemoglobin, ESR, blood sugar and blood urea, were within normal limits. The blood VDRL (Venereal Disease Research Laboratory) test was negative. Urinalysis was normal. The slit-skin smears from the earlobe and from the plaque, and scrapings from the nasal mucosa did not show acid-fast bacilli. The Mantoux test was negative. Epidermal scrapings from the earlobe and from the plaque on the cheek when examined in 10% KOH showed a few thick-walled, rounded, brownish-black bodies (sclerotic cells). A skin biopsy from the plaque on the cheek revealed a hyperkeratotic and acanthotic epidermis with small neutrophilic abscesses and tuberculoid formations in the dermis composed of epithelioid cells and multinucleated giant cells. Culture of the biopsy specimen from the nasal lesion in Sabouraud's agar yielded growth of *Cladosporium carrionii*. X-rays of the skull, face and chest did not show any abnormality except deformity of nasal septum corresponding to clinical destruction of the part. The CSF was normal and there were no fungal bodies in it.

The patient was treated with ketoconazole 200 mg twice daily orally for 2 months, but did not show any response. On discharge she was prescribed oral itraconazole but did not turn up for follow up.

Infiltration of the earlobe, presence of a raised plaque on the cheek, destruction of the cartilage of the nasal septum and nostril, and isolated paralysis of the buccal branch of the right facial nerve were the features that simulated lepromatous leprosy in our patient. These made the general practitioner diagnose and treat the case wrongly as leprosy. Chromoblastomycosis commonly affects the skin only. Involvement of the nasal mucosa as occurred in our patient is extremely rare, although one such case was reported by Jakanitzu, *et al.* ([1]). Although the verrucous nature of the surface of the plaques, demonstration of "sclerotic cells"

in the epidermal scrapings and characteristic histopathologic features easily differentiate chromoblastomycosis from leprosy lesions, some difficulty may occur if one is not aware of the possibility of chromoblastomycosis. A cutaneous plaque of chromoblastomycosis closely simulating tuberculoid leprosy clinically and histopathologically has been reported from India ([2]). Chromoblastomycosis developing in a residual patch of leprosy also has been reported ([3]). These reports and the present report underline the importance of considering chromoblastomycosis also in the differential diagnoses of all types of leprosy.

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Comparison of Pentoxifylline, Thalidomide and Prednisone in the Treatment of ENL

To the Editor:

Erythema nodosum leprosum (ENL) is an inflammatory reaction that occurs in approximately 40%–50% of lepromatous leprosy patients, most commonly during treatment with antileprosy drugs. ENL is characterized by the appearance of painful, erythematous, subcutaneous nodules which are tender to the touch. These lesions are not necessarily associated with pre-existing leprosy lesions. Systemic manifestations including fever, malaise, lymphadenopathy, neuritis, and arthralgia are often observed. It appears that the pro-inflammatory cytokine tumor necrosis factor-alpha (TNF-α) may play an important role in the development of this syndrome since high plasma levels of TNF-α are found in patients during episodes of active ENL ([10]). Moreover, treatment of ENL patients with thalidomide alleviates the clinical symptoms concomitant with a reduction in plasma TNF-α levels ([8]). *In vitro,* thalidomide selectively inhibits the production of TNF-α by lipopolysaccharide-stimulated monocytes ([6, 9]).

Although thalidomide is the drug of choice for the treatment of ENL ([3, 7, 12, 13]) it is a potent teratogenic drug ([4, 5]) and is not safe in women of child-bearing potential. Glucocorticoids, a family of drugs known to inhibit cytokine production by leukocytes, are also used for the treatment of ENL. Unfortunately, the prolonged use of these drugs is associated with toxicities, including immunosuppression. In an attempt to identify other treatments for ENL, alternative TNF-α inhibitors are under consideration. One such drug is pentoxifylline, a methylxanthine derivative which has been used clinically for intermittent claudication. The drug has been shown to inhibit TNF-α production *in vitro* ([2, 14]) and *in vivo* ([1, 8]).

We have now tested whether pentoxifylline if effective in alleviating the signs and symptoms of ENL and have compared the efficacy of the drug in controlling the symptoms and dermatologic manifestations of ENL to the efficacy of thalidomide and of steroids. Sixteen multibacillary leprosy patients with ENL were graded for severity of disease symptoms at the start of the study and weekly thereafter (The Table). Plasma was collected at baseline and weekly for cytokine evaluation, and biopsies of ENL lesions were taken at baseline and day 2 for histologic evaluation of the response in the skin.

RESULTS AND DISCUSSION

Clinical response. The manifestations of ENL were graded for severity of disease