

CORRESPONDENCE

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Trigeminal Trophic Syndrome in Hansen's Disease

TO THE EDITOR:

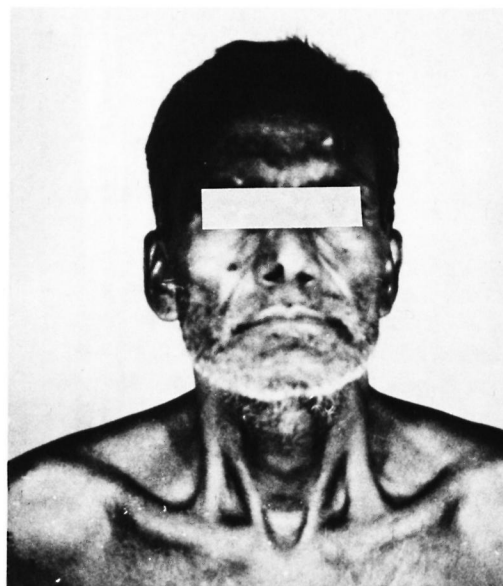
A 60-year-old man, a known case of mid-borderline Hansen's disease, was seen for facial nerve palsy. There was no history of transient ischemic attack, convulsion, dimness of vision, or herpes zoster. On examination, he had features of Hansen's disease—left-sided facial palsy of lower motor neuron type and anesthesia over the trigeminal area, loss of corneal reflex and destruction of the ala nasi on the right side (The Figure). A clinical diagnosis of Hansen's disease with trigeminal trophic syndrome was made. A neurologist's opinion was sought to exclude other causes of facial and trigeminal nerve involvement which ruled out the possibility of any organic cause in the brain attributable to the nerve damage (both trigeminal and facial). A slit-skin smear for acid-fast bacilli was negative. The patient is being treated with the multidrug regimen for multibacillary Hansen's disease and is being followed up.

The trigeminal trophic syndrome is an uncommon clinical entity presenting with trophic ulcer(s) over the already anesthetic or paraesthetic area of the trigeminal nerve distribution beginning in the ala nasi. It results from destruction of the fibers that carry pain and temperature senses, and is due to various causes such as disease, injury, or surgery (1).

The lesions producing such destruction may be of central or peripheral origin (3). The central connection of the trigeminal nerve may be affected by tumors, syringobulbia, multiple sclerosis, or occlusion of the posterior cerebellar artery. The periph-

eral causes encountered are tabes dorsalis, granulomatous meningitis, tumors of the nerve, pressure from without on the nerve, fracture of the base of the skull, and alcohol injection into the gasserian ganglion. The dermatological diseases giving rise to such changes are herpes zoster and Hansen's disease.

As a result of trauma to the anesthetic or paraesthetic area, a crusted ulcer begins usually in the ala nasi of the affected side. If further trauma is not prevented, the ulcer extends to even destroy the nasal cartilage.



THE FIGURE. Destruction is seen of the right ala nasi with trophic ulcerations on the same side; note hypopigmented patch on left shoulder.

The ulceration may spread to the adjacent anesthetic areas as well, and these ulcers easily may be mistaken for basal-cell epitheliomas because of the site and crusting. Due to their bizarre clinical appearance, they may even simulate dermatitis artifacts. Bleeding, scarring, and deformity are the troublesome complications of such ulcers. In addition to cutaneous ulcers, corneal anesthesia may also be noted (particularly in patients whose trigeminal trophic syndrome is secondary to Hansen's disease).

Although trophic changes in the trigeminal area have been reported to result from various causes such as the injection of alcohol into the gasserian ganglion⁽²⁾, their occurrence secondary to Hansen's disease, as in our case, is very rare. To the best of our knowledge, no textbook on Hansen's disease includes trigeminal trophic syndrome as one of the trophic complications of leprosy.

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Response to Phytohemagglutinin of LL Patients' Lymphocytes Preincubated in Culture Media

TO THE EDITOR:

Patients with leprosy present a well-known, wide spectrum of clinical manifestations⁽¹⁾. Recently, we have published that lymphocytes from lepromatous (LL) patients stimulated with T mitogens are deficient in the synthesis of interleukin-2 (IL-2). However, the cells possess receptors for IL-2^(3,4). Mohaghehpour, *et al.*⁽⁶⁾ have published that the failure of T lymphocytes from LL patients to respond to *Mycobacterium leprae* was associated with the defective expression of IL-2 receptors, and that this deficiency was not corrected by exogenous IL-2. Later, they reported⁽⁵⁾ that when T lymphocytes from LL patients were preincubated in culture media for 48 hr and then stimulated with specific antigen, the cells recovered their ability to proliferate.

According to the above, we wondered if T cells from LL patients were also able to recover their capacity of proliferation when they were preincubated in culture media and then stimulated with a nonspecific T mitogen, such as phytohemagglutinin (PHA).

Study subjects. Based on Mohaghehpour, *et al.*'s patients' classifications⁽⁵⁾ as "responders" and "nonresponders," we tested 23 patients (8 "responders" and 15 "nonresponders") from the Instituto Dermatológico at Guadalajara, Jalisco, Mexico, diagnosed as having LL according to international criteria⁽⁷⁾.

Mononuclear cells. Heparinized blood (20 IU/ml) was obtained from each subject by venipuncture. After centrifugation on a Ficoll-hypaque gradient⁽¹⁾, the mononuclear cells were recovered and washed. The