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Great auricular nerve involvement in leprosy: Scope for misdiagnosis
Ramesh V, Jain RK, Avninder S*

ABSTRACT
Three patients with neuritis of the great auricular nerve (GAN) have been reported. Two patients seen by physicians and an otolaryngologist had prominent and tender cord along the neck with facial edema and history of fainting attack in one, and erythema and hyperaesthesia of the ear in the other simulating vascular occlusion, which were confirmed to be leprosy in Type 1 reaction by the dermatologist. In the third, cold abscess in the nerve that had persisted after anti-leprosy treatment was mistaken as tuberculous cervical lymphadenitis by a surgeon since aspiration had revealed acid-fast bacilli. The probable reasons for misdiagnosis include rarity of involvement of the GAN and its proximity to main blood vessels, and the need for careful interpretation of laboratory results.

KEY WORDS: Great auricular nerve, leprosy

Case Report
Some presentations of leprosy continue to be misdiagnosed since the cardinal clinical features like anesthetic skin patch and thickened and/or tender nerves are not readily made out. The present report illustrates situations in which a faulty diagnosis can be made by doctors in specialties other than dermatology.

Case History
Case 1
A 37-year-old man with a history of fainting attack, swollen cheek and a thickened cord on the right side of neck was suspected to have vascular occlusion. Blood investigations, Doppler, CT scan and magnetic resonance imaging (MRI) showed no abnormality. With anti-inflammatory drugs the swelling had decreased revealing an inflamed patch and right eyelid weakness for which our opinion was sought. The cord had been present for over five years and since recently an intensely red patch on his right cheek had appeared followed by pain and increase in thickening of the cord. The shooting pains had made him swoon. Examination revealed a large, circular, raised red plaque on the right side of the face extending from the lower forehead to the mandibular area including pinna, with incomplete closure of right lower eyelid. The right great auricular nerve (GAN, also called greater auricular nerve) was markedly thickened and moderately tender. Skin biopsy showed features of borderline tuberculoid (BT) leprosy in Type 1 lepra reaction: no acid-fast bacilli were seen. Oral prednisolone 60 mg/day given in tapering dose for six weeks gave good relief. Currently he is on paucibacillary multi-drug therapy (PBMDT) comprising rifampicin 600 mg every month and dapsone 100 mg daily.

Case 2
A 26-year-old man was seen by a physician for a thickened cord on the left side of the neck with redness and burning of the left ear of four months duration. Suspecting external jugular vein thrombosis he was sent to the otolaryngologist who recommended a Doppler and sought our opinion. On examination the left GAN was found to be tender and markedly thickened. An irregular, erythematous and hyperesthetic plaque was seen over the retroauricular area, the ear lobule and part of the left external ear [Figure 1]. As before, histopathology revealed BT in reaction with the infiltrate extending into the subcutaneous fat.

Case 3
A 22-year-old man with a neck swelling had consulted a surgeon who had diagnosed tuberculous lymphadenitis and started antitubercular treatment since fine needle aspiration had revealed occasional acid-fast bacilli (AFB). Observing no change after two months, the patient was referred to us. A slightly tender, subcutaneous, firm nodule was felt on the right lateral side of the middle of the neck [Figure 2] along the course of a moderately thickened GAN. Enquiry disclosed that he had taken anti-leprosy therapy three years ago for an anesthetic patch on the back of the neck. The patch had repigmented and nerve thickening had regressed over time forming a small central swelling. He was reassured that no treatment was required.

Discussion
Leprosy is a neurocutaneous mycobacterial infection with predominantly cutaneous manifestations. When localized only to the peripheral nerve(s) it is primary neuritic leprosy. A wide
from destructive mechanisms within the confines of the nerve.\cite{5} Nerve abscesses are a well-recognized manifestation occurring during the natural course of paucibacillary leprosy (cold abscess) or in acute exacerbations called reactions (hot abscess).\cite{3} Cold abscesses appear as beaded or localized swelling(s) that can persist for years.\cite{4} Moreover, bacilli lurk in nerve than in skin and are likely to persist as they lie protected from destructive mechanisms within the confines of the nerve.\cite{3} This is no cause for concern if the patient has been treated.

Apart from thickening, nerve abscesses are a well-recognized manifestation occurring during the natural course of paucibacillary leprosy (cold abscess) or in acute exacerbations called reactions (hot abscess).\cite{3} Cold abscesses appear as beaded or localized swelling(s) that can persist for years.\cite{4} Moreover, bacilli lurk in nerve than in skin and are likely to persist as they lie protected from destructive mechanisms within the confines of the nerve.\cite{3} This is no cause for concern if the patient has been treated.

The first two patients had a similar presentation. Attention to the lesions was drawn after an acute episode of reaction. In a third of those with neuritic leprosy, skin lesions can appear after years, sometimes as reactions.\cite{6} The hyperaesthetic and erythematous skin lesions, markedly thickened GAN, and the fainting spell led the physician to suspect vascular occlusion. Such unusual presentations have misled even leprologists to consider a cerebrovascular accident\cite{3} and similar to their experience we feel that the fainting spell in our patient was a neuritic episode due to facial nerve involvement. Early presentations of leprous peripheral neuritis can cause acral paraesthesia, so much so that in neuralgia due to affection of the trigeminal nerve, one should consider leprosy, especially in those with a family history of the disease or when the patient hails from an endemic area.\cite{7} A variety of misleading symptoms associated with thickened GAN ranging from chest pain to paraesthesias have been reported.\cite{8}

In the last patient who had completed anti-leprosy treatment the loose material formed a localized swelling simulating lymph node. Mycobacterium leprae which is less acid-fast when compared to M. tuberculosis had shown up on Ziehl-Neelsen stain that employed a higher strength of 20% H₂SO₄. This could have been due to technical errors or use of reagents and acid stored for long, which can lower its strength.\cite{9}

From a recent report in which a cardiovascular surgeon mistook GAN thickening for external jugular vein thrombosis\cite{10} and ours, it is obvious that various specialists have misdiagnosed leprosy. The reasons may be because GAN is situated in an area traversed by major vessels where neuritis can simulate vascular occlusion or lymph node swelling. Probably in no other disease is the GAN so markedly thickened, with the rare exception of Charcot-Marie-Tooth disease, a hereditary motor sensory neuropathy. Additionally, in training programs\cite{10} stress is generally laid on examination of mixed nerves with motor functions to prevent deformities.

**References**


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