Leprosy nerve abscess in Indian male, misdiagnosed as tuberculous lymphadenitis and neuroma

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Accepted for publication 21 May 2013

Leprosy is a great mimic with a wide spectrum of signs and symptoms. Nerve abscesses, although common¹ have rarely been reported in the literature, especially in pure neuritic leprosy.²,³ The incidence of these abscesses is higher in males and the commonest nerve involved is the ulnar nerve, with the greater auricular nerve being the most commonly affected sensory nerve.⁴ Abscesses are often misdiagnosed and case reports exist in the literature of a variety of unusual presentations.⁵

A 24-year male, resident of U.P, India, presented to surgery Out Patient Department (OPD) with a painful swelling over the right upper arm (Figure 1).

It was abrupt in onset, non-progressive, painful, persistent and associated with occasional paraesthesia and fever off and on, but no sensory loss or motor weakness. Based on the presence of a mobile, firm, tender swelling, a provisional diagnosis of tuberculous lymphadenitis was made. The routine blood investigations and chest X-ray were normal. The Mantoux reading was 12 mm, ESR 40 mm and lesional fine needle aspiration (FNAC) revealed caseous necrosis. High resolution ultrasonogram (USG) showed a well-defined elliptical lesion of size 1.5(×2.0 cm on the medial aspect of the distal right arm with an anechoic centre and thickened, irregular, echogenic walls. The patient was therefore started on Category I anti-tubercular therapy (Isoniazid, Rifampicin, Ethambutol, Pyrazinamide) for tuberculous lymphadenitis, and after 6 months was changed to Category II (Category 1 plus Streptomycin) on the grounds of possible drug resistance, since there was no improvement in

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the symptoms or size of the swelling despite regular treatment. In fact, his clinical condition deteriorated with the appearance of sensory loss and motor weakness in the ulnar nerve distribution, leading to progressive bending of medial two fingers of right hand. Magnetic resonance imaging (MRI) was done which revealed an enhancing oval lesion along the right ulnar nerve in the distal third, hyperintense on T2W and intermediate on T1W suggesting neuroma formation. For this the patient was offered surgical excision, which he refused.

The patient then presented to the Dermatology OPD with swelling over the lower arm and sensorimotor complaints. There was no family history of Hansen’s disease and mucocutaneous examination revealed no madarosis, infiltration or light coloured lesions. On nerve examination, the right ulnar nerve and bilateral greater auricular nerves were enlarged with sensory loss along ulnar nerve distribution and partial claw hand (right). There was a single, well defined, firm, nodular, tender, non mobile swelling of size 2 × 2 cm felt along the lower one third of the right ulnar nerve just above the medial epicondyle (Figure 1). The motor power in the hypothenar muscles was 3/5 (MRC scale). A skin biopsy over the area of sensory loss was inconclusive. Repeat FNAC showed only necrosis without any granuloma. A slit skin smear, skin biopsy and FNAC did not reveal acid fast bacilli (AFB). Nerve biopsy revealed the focal loss of large myelinated nerve fibres without inflammation, granuloma, vasculitis or AFB. Nerve conduction velocity (NCV) revealed absent F wave in right ulnar nerve. Lepromin testing could not be done. A repeat USG to look for the origin of swelling revealed a well-defined elliptical hypoechoic lesion without any evidence of liquefaction on the medial aspect of the right arm attached to the ulnar nerve. On the basis of clinical findings and investigations, a diagnosis of Pure Neuritic Hansen’s disease with nerve abscess with Grade 2 deformity was made and the patient was started on anti-inflammatory agents, corticosteroids (started on 60 mg, tapered over 6 months) and WHO PB-MDT along with rest and later physiotherapy of the affected limb. The swelling showed some improvement in terms of size and symptoms. However, the patient had episodes of shooting pain, even while on therapy. Muscle charting did not show any improvement or worsening in the motor power.

Although there are case reports of the therapeutic role of INH and Rifampicin in the management of leprotic nerve abscess, in our patient there was no improvement despite
taking these drugs for 12 months due to misdiagnosis of TB. There are conflicting opinions regarding the management of persistent abscesses in leprosy, the guidelines being surgical excision for an infected hot abscess and oral steroids with or without surgical management in other cases. The indications for surgical treatment in these cases include pain not controlled by steroids, requirement for high doses of steroids, and increasing sensory and/or motor deficit. Surgery is advised because it results in less morbidity than long-term steroids. In our case the patient did not respond well to oral corticosteroids in terms of sensory complaints or motor deficit. There were, however, no signs of activity on the overlying skin (erythema) or in the investigations (USG and MRI suggestive of fibrosis) and no progression of sensorimotor complaints. Thus in our case, the decision to resort to surgery just on the basis of the subjective symptoms without any signs of activity could not be justified, as the surgeon’s knife can cause more damage than improvement. This case is being reported because of the rarity of nerve abscess in pure neuritic Hansen, and its misdiagnosis among physicians and surgeons, and to draw attention to the management of resistant abscesses.

References