

LEPROMATOUS LEPROSY PRESENTING AS A SWELLING IN THE NECK

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A 25-year-old electrician presented with gradually progressive, asymptomatic swelling on left side of the neck since 2 years. The swelling which was initially diagnosed as cervical lymphadenitis by the internist represented the enlarged left great auricular nerve. Cutaneous examination revealed an ill-defined, hypoaesthetic macule with minimal atrophy on the pinna of the left ear. The histopathology of the nerve showed a lepromatous neuritis with bacteriological index (BI) of 5+.

Key Words: Lepromatous leprosy, Neck-swelling

Introduction

Inflammation and destruction of peripheral nerves is a unique feature of leprosy and every leprosy patient has peripheral nerve involvement.¹ Clinically the patients present with sensory and/or motor loss along the distribution of the affected nerve. Examination usually reveals thickened, tender and/or nodular nerve trunks. However, if the nerve is superficially located, it may be visible as swelling/cord-like structure in that area. The histopathological diagnosis depends largely on the demonstration of *Mycobacterium leprae* (*M. leprae*) and the granulomatous infiltrate caused by it in the cutaneous nerves.¹ We describe a patient of lepromatous leprosy who presented with a swelling on the left side of the neck.

Case Report

A 25-year-old man had a 2-year history of

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gradually progressive asymptomatic swelling on the left side of the neck. There was no history of any hypoaesthetic skin lesion associated with the swelling or elsewhere on the body. The patient was initially seen in the out patient department of Medicine where a diagnosis of tubercular cervical lymphadenitis was considered. A fine needle aspiration cytology, repeated twice, revealed ill-defined granulomatous infiltrate with numerous acid-fast bacilli.

Examination revealed an ill-defined, firm, mildly tender swelling of 3 x 2 cms with uneven surface on the left side of the neck. There was an erythematous 5 x 2 cms macule with minimal atrophy on the pinna of the left ear extending on the left cheek. The lesion had 50% sensory loss to temperature while the sensation to pain and touch were unaltered. The supra-orbital nerve on the left side was also thickened. The eye examination revealed partial lid lag of the left eye. There were no other skin lesions or thickened nerves and no peripheral sensory loss. Systemic examination was unremarkable.

Histopathological examination of the nerve biopsy specimen revealed ill-defined granulomas consisting of

epithelioid cells with numerous lymphocytes and histiocytes. Ziehl Neelsen's stain for acid fast bacilli was strongly positive with BI of 5+. Skin biopsy from the pinna of the ear showed atrophy of the epidermis and a Grenz zone with focal perivascular and periappendageal lymphohistiocytic infiltrate in the dermis. Routine haematological parameters, liver and renal function tests, chest skiagram, stool and urine examination were all within normal limits. A diagnosis of lepromatous leprosy was made and the patient was treated with multi-drug therapy as recommended by WHO for multi-bacillary leprosy.

Discussion

Neurotropism is one of the characteristic features of *M. leprae*. The peripheral nerves serve as immunologically protected site and thus the bacilli preferentially thrive there.¹ Leprosy can manifest in many unusual ways²⁻⁴ The diagnosis in such situations can be missed. This patient had a single nerve involvement in the neck which manifested as a swelling and was misdiagnosed as cervical lymphadenitis. Since the face and the neck area has rich overlapping nerve supply, the sensory alterations in a lesion of leprosy may not be appreciated. A close examination of the skin must be done which may reveal a lesion in the area supplied by the nerve. However, the diagnosis should be confirmed by histopathological examination of the skin lesion and the nerve tissue.

In the recent past several investigators have reported a significant discrepancy in the histopathological and bacteriological picture between the nerve and skin lesions in a significant number of patients.⁵⁻⁶ This difference is more marked towards the tuberculoid end of the spectrum.^{6,9} In general a higher bacillary load and a more lepromatous picture is exhibited in nerve histopathology than in the skin lesion.⁵⁻⁹ Clinically our patient had tuberculoid leprosy but nerve histopathology revealed numerous acid fast bacilli with a BI of 5+ which was consistent with lepromatous leprosy.

In upto one-third of clinically diagnosed patients of leprosy where skin biopsy has shown non-specific changes, the histopathological examination of the nerve has revealed the features of leprosy.^{5-7,9} The present criteria for classifying patients into multibacillary and paucibacillary leprosy are mainly based on the clinical features, slit skin smear and the histopathology of the skin lesions.¹⁰ This does not take into consideration the histopathological changes in the nerve which has raised certain questions regarding the rationale of WHO's recommendations for treatment of leprosy in such patients.⁸ Therefore it seems reasonable to speculate that a proportion of patients who are considered paucibacillary by present criteria are potentially harbouring multibacillary leprosy in the nerves. So these patients should be treated with multibacillary regimens.

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