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Case Report

Delayed diagnosis of neural signs of leprosy

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Abstract Leprosy is a chronic granulomatous disease caused by *Mycobacterium leprae*. It mainly affects the peripheral nerves and skin. Neural symptoms often precede the appearance of skin lesions but in many cases leprosy is not considered as a cause, leading to a delay in diagnosis. Ensuing nerve damage leads to deformity and disability. We herein, describe two adult males who first presented with neural symptoms, but in both the cases the diagnosis of leprosy was delayed by several years. The outcome in both was a visible deformity.

Key words
Leprosy, nerve damage, disability.

Introduction

Mycobacterium leprae, the causative agent of leprosy has a predilection for neural tissue. It probably enters the nerves through endoneural blood vessels[1] and the target cell is the Schwann cell. Nerve damage occurs in skin lesions and in peripheral nerve trunks. Damage to dermal nerve fibers causes loss of sensation and sweating, inside the skin lesion. Peripheral nerve trunks are vulnerable where they are superficial or are in fibro-osseous tunnels. A slight increase in the nerve diameter raises the intra-neural pressure, causing nerve compression and ischemia. Resulting nerve damage leads to anesthesia and muscle weakness.[2]

The cases presented here are of 2 adult males, representing the military and paramilitary forces of Pakistan. Both the cases first presented with neural symptoms, but leprosy was not considered as a cause and the diagnosis was delayed by several years. Ensuing nerve damage led to a visible deformity in both.

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Case 1

A 34-year-old army soldier was referred to Marie Adelaide Leprosy Centre (MALC) from the dermatology department of PNS Shifa hospital, Karachi. He complained of nasal stuffiness and bleeding, off and on for the past 4-5 months, together with pain in both feet and hyperesthesia in the skin. He also complained of loss of sensation on the medial side of his right hand, for the past 12-13 years and weakness in the same hand, for 5-6 years.

A review of his medical record showed that he had first reported at a military hospital in 2004, with complaints of pain and numbness on the ulnar side of his right forearm. On examination he was found to have impaired sensation on the same side of his right forearm and hand, with wasting of right hand muscles. Nerve conduction and EMG studies had suggested ulnar nerve compression at the right elbow. A month later, he was operated upon to relieve nerve compression **(Figure 1)**. Re-exploration was done under general anesthesia in 2006. No improvement was noted in periodic follow-up examinations done by plastic surgeons, during the next 10 years.

In February 2016, he consulted a dermatologist at another military hospital for his skin lesions, which he had noticed during

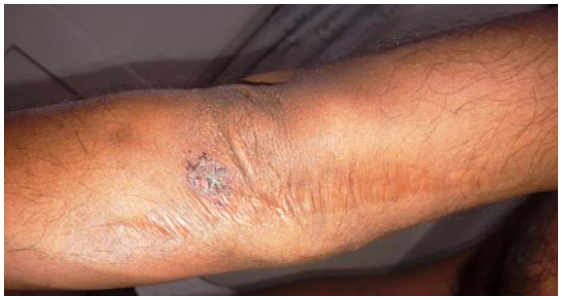

Figure 1 Scar of previous ulnar nerve surgery.

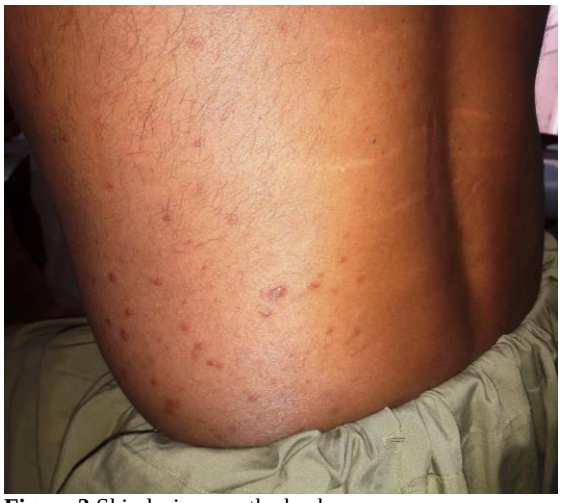

Figure 2 Skin lesions on the back

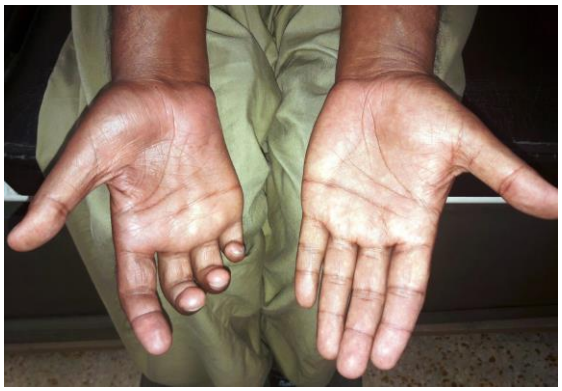

Figure 3 Muscle wasting and claw-hand

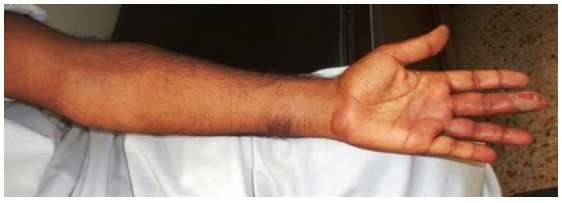

Figure 4 Hypopigmented patch on the left forearm and an ulcer in an anesthetic left middle finger.

the preceding 4 months. His skin biopsy was taken and the histopathology report confirmed leprosy. On examination he was found to have papular infiltration on his ears and forehead, papules and nodules on both upper limbs and lower trunk **(Figure 2)**.He had bilateral enlargement of ulnar, radial cutaneous, common peroneal and posterior tibial nerves. There was loss of sensation in his right lower forearm and hand, together with wasting of the small muscles and an ulnar-median clawing **(Figure 3)**.

His skin smears taken at MALC showed a bacterial index of 1.6+, for *M. leprae*. His skin biopsy reported foamy macrophages in the dermis, with acid-fast bacilli (AFB) on Wade-Fite stain. His leprosy was diagnosed 12 to 13 years after the first appearance of neural symptoms and only when he presented with skin lesions to a dermatologist.

Case 2

A 32-year-old male, serving as a soldier in the Rangers paramilitary force was referred to MALC from the neurology department of JPMC, a public sector tertiary-care hospital in Karachi. He presented with a single hypopigmented patch on his left arm, with impaired sensation. He had first noticed it 3 years back. He had an ulcer on the palmar surface of his left middle finger and complained of loss of sensation in that finger, for the past 5 years **(Figure 4)**. He had also started feeling weakness in his left hand, during the last 5-6 months.

Since 2011 he had been to different doctors at major public sector hospitals in Larkana and Karachi, as well as, an armed forces hospital in Karachi. An electrophysiological study done in 2013 was found to be abnormal and suggested very mild effects of an early left C7 radiculopathy. No evidence of peripheral nerve entrapment or myopathy was seen. An MRI cervical spine was recommended. It reported diffuse disc bulges at the levels of C2-3 and C5-6, causing mild thecal indentation without significant foraminal narrowing and radicular compression. An MRI cervical spine repeated in 2015 was found to be normal, together with a colour Doppler of both arms.

On examination he was found to have enlargement of both ulnar, left median and right posterior tibial nerves. There was loss of sensation on the medial side of his left hand and in the lateral 3 fingers. There was weakness of the lumbrical muscles of all 4 fingers, as well as, of the abductor digiti minimi in his left hand.

His skin smear taken at MALC was negative for AFB. His nerve conduction study suggested a left ulnar axonal neuropathy, at the elbow joint. He was diagnosed to have leprosy on the basis of 2 out of the 3 cardinal features, namely loss of sensation inside a hypopigmented skin lesion and nerve enlargement, at sites of predilection.

Discussion

In the first case reported by us, the diagnosis was delayed by 13 years and in the second case by 5 years. Delay in diagnosis increases the risk of nerve damage. A significant amount of nerve damage can be prevented if the patients are diagnosed early, more than by any other intervention later.[3]

Out of the total 446 new leprosy cases detected in Pakistan in 2015, 85 (19%) had a visible deformity (grade 2 disability) due to nerve damage.[4] This was higher than the figure of 6.7% globally.[4] Grade 2 disabilities (G2D) indicate the level of awareness of early signs and symptoms and of health seeking response in the community, as well as, the capacity of the health system to recognize and treat leprosy at an early stage, i.e. before the development of disabilities.[4]

In a study done in the United Kingdom, delay in diagnosis of leprosy was found to have occurred in 82% of cases and 68% of patients had nerve damage resulting in disability. The mean lag time (from onset of symptoms to diagnosis) was 3.1 years, median 1.8 years and range 0.2-15.2 years.[5] In the same study, both neurologists and orthopaedic surgeons did not consider leprosy as a possible cause of ulnar neuropathy in 4 cases.

In a study done in Thailand, 72% of multibacillary (MB) and 67% of paucibacillary (PB) patients had a delay in detection of 2 years. The median delay was 2.3 years in the PB and 1.9 years in the MB group.[6]

In a case-control study in Ethiopia, out of a total of 273 patients included in the study, 1 in every 5 reported a delay in diagnosis by the health services. Disabled patients had a median delay of 26 months, while non-disabled patients had a delay of 1 year.[7]

Leprosy is the leading infectious cause of disability.[8] Anaesthetic skin lesions are characteristic feature of leprosy and it is the commonest cause of nerve thickening. Delay in diagnosis can be avoided by testing undiagnosed skin lesions for anaesthesia and by considering leprosy as a possible cause of unexplained neuropathy and unexplained foot ulcers.[5]

References

1. Pearson JM, Ross WF. Nerve involvement in leprosy: pathology, differential diagnosis and principles of management. *Lepr Rev*. 1975;**46**:199-212.
2. Lockwood DNJ. Leprosy. In: Griffiths C, Barker J, Bleiker T, Chalmers R, Creamer D, editors. *Rook's Textbook of Dermatology, 9th edn, Vol 2*. Oxford: John Wiley & Sons; 2016. P. 28.1-28.18.
3. Richardus JH, Finlay KM, Croft RP, Smith WC. Nerve function impairment in leprosy at diagnosis and at completion of MDT: a retrospective cohort study of 786 patients in Bangladesh. *Lepr Rev*. 1996;**67**:297-305.
4. World Health Organization. Global leprosy update, 2015. *Wkly Epidemiol Rec*. 2016;**91**:405-20.
5. Lockwood DN, Reid AJ. The diagnosis of leprosy is delayed in the United Kingdom. *QJM*. 2001;**94**:207-12.
6. Schreuder PA. The occurrence of reactions and impairments in leprosy: experience in the leprosy control program of three provinces in Northeastern

Thailand, 1978-1995. 1. Overview of the study. *Int J Lepr*. 1998;**66**:149-58.

7. Bekri W, Gebre S, Mengiste A, Saunderson PR, Zewge S. Delay in presentation and start of treatment in leprosy patients: a case-control study of disabled and non-disabled patients in three different settings in Ethiopia. *Int J Lepr Other Mycobact Dis.* 1998;**66**:1-9.
8. Rodrigues LC, Lockwood DN. Leprosy now: epidemiology, progress, challenges and research gaps. *Lancet Infect Dis*. 2011;**11**:464-70.