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Delayed Diagnosis

Leprosy presenting as unilateral foot drop in an immigrant boy

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Summary: This is a case report of a boy who presented with unilateral foot drop approximately 1 year after traumatic injury from skateboarding. Exploratory surgical operation was eventually performed, and histological examination of the surgical specimen revealed leprous peripheral neuropathy of the peroneal nerve. The patient had immigrated from Vietnam to the United States 3 years previously. The occurrence, albeit rare, of this entity in the immigrants from endemic leprosy regions is discussed.

Introduction

During the latter part of this century, the incidence of leprosy in the United States has risen steadily from 50–60 cases per year some 30 years ago to 341 in 1985.[1,2] The influx of Southeast Asian immigrants has contributed heavily to this rise during the past 20 years.[1,2] Nonetheless, the diagnosis of leprous neuropathy is often long delayed because of the failure to recognize this entity in the absence of obvious skin manifestations. This report describes the clinical and pathological findings in a 16 year old Vietnamese immigrant who presented with unilateral foot drop due to leprous involvement of the peroneal nerve. I am not aware of any previously recorded example in the Western literature in which the initial clinical presentation of leprous neuropathy was that of foot drop without prominent skin lesion.

Case report

A 16 year old boy, who had immigrated from Vietnam in 1987, was admitted for right peroneal nerve resection with sural nerve graft placement in 1990. He had fallen off a skateboard 1 year prior to admission, in 1989. One month after the skateboarding injury, he was seen to be dragging his right foot. A local practitioner observed right foot drop, absence of tactile sensation over the anterior thigh involving the L_3 and L_4 dermatomes, and hypoaesthesia over the entire lower leg. There was no sensory loss over the ears, dorsal surface of the hands, or nose.

Three months prior to admission, evaluation confirmed the presence of complete right foot drop and diminished pinprick perception in the distribution of the right peroneal nerve. The peroneal nerve was not enlarged or tender, and deep tendon reflexes were normal in both legs. Nerve conduction studies showed absence of the right sural sensory potentials. There was no recordable peroneal motor and a markedly diminished peroneal nerve motor response over the extensor digitorum brevis with stimulation at the head of the fibula. The left motor peroneal conduction was normal. Electromyography showed evidence of chronic denervation of the peroneus longus and brevis in the form of fibrillations, positive sharp waves and polyphasic motor units, with a reduced recruitment pattern. Magnetic resonance imaging of the lumbosacral spine showed no evidence of spinal cord or nerve root compression.

Operative exploration and decompression of the nerve was undertaken at the head of the fibula on the day after admission. With the aid of an operating microscope the epineurial compartment was opened and seven fascicles identified. The two most laterally situated fascicles showed fusiform enlargement, thought possibly to be indicative of a nerve sheath tumour. Intraoperative nerve stimulation showed no evidence of electrical conduction across the abnormal nerve segments. These segments were resected and a sural nerve graft placed. The remaining five fascicles responded normally to nerve stimulation.

Pathological examination of the operative specimen showed the presence of a widespread inflammatory infiltrate composed predominantly

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of plasma cells and lymphocytes. The nerve trunk showed evidence of marked loss of axons and proliferation of connective tissue. Many acid-fast organisms were seen, both within globi and within Schwann cells. These findings were judged to be indicative of lepromatous leprosy. Postoperatively, in view of the pathological findings, a careful search was made for the presence of skin lesions to suggest leprosy. Only a 2.5 × 2.0 cm hypertrophic scar was observed over the right knee, but there was no hypo- or hyperpigmentation or hypoaesthesia. Slit skin smears were not performed. His other nerves were examined, but they were not enlarged or tender. He has subsequently been maintained on a course of long-term, multi-drug therapy with dapsone, clofazimine and rifampicin.

Discussion

There are only a few reports in the Western literature of leprous neuropathy in the absence of prominent cutaneous involvement.[3-8] The difficulties encountered in making the diagnosis are attested to by the long interval between the onset of neural disease and the demonstration of the causative organism in most instances. In the case that forms the subject of this report, the difficulty was further compounded by the history of prior traumatic injury and by the rarity with which peroneal involvement is seen in relative isolation. The most frequent sites of involvement are the radial and ulnar nerves at the wrist, and the great auricular nerves. Other commonly involved nerves include the lateral popliteal nerve around the neck of the fibula, the posterior tibial nerve along the posteromedial aspect of the ankle, the facial nerve and the trigeminal nerve. Single or multiple areas may be involved.[3] The diagnosis can be made only when there is a high index of suspicion, particularly in a patient who has immigrated from an endemic site within the 3 years preceding the onset of neurological dysfunction. In the absence of prominent cutaneous manifestations, demonstration of the aetiological agent must rest on operative sampling of the affected nerve.

In retrospect, had they been more alert to the possibility of leprosy, it is likely that the diagnosis in the patient would have been made earlier and with a less extensive procedure. I wish, therefore, to emphasize to all primary care physicians that leprosy must be considered in the differential diagnosis of any individual with peripheral neuropathy who has immigrated from a region in which the disorder is endemic.

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