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Tibialis posterior tendon transfer for post - organophosphorous induced lateral popliteal nerve palsy with foot drop - case report

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Abstract

Introduction :- We report a case of organophosphorous induced lateral popliteal nerve palsy, a rare toxicity from ingestion of organophosphorous esters. A 32 years old male presented with high stepping gait and left foot drop, loss of sensations on dorsum of 1st web space left foot and normal deep tendon reflexes in bilateral lower limbs. He had consultation with neurologist and was treated conservatively for the lateral popliteal nerve palsy. Ipsilateral tibialis posterior tendon was transferred by extra-membranous route to tibialis anterior, extensor hallucis longus, extensor digitorum longus tendons under optimum tension. At 6 months of follow up patient had full active grade 4 ankle & toes dorsiflexion with grade 5 active plantar flexion.

Key words: organophosphorous induced nerve palsy, foot drop, tibialis posterior transfer.

Introduction

The commonest cause of the lateral popliteal nerve (LPN) palsy is trauma. Organophosphorous (OP) poisoning rarely leads to delayed lumbar plexopathy leading to LPN palsy or isolated LPN palsy with resultant foot drop. [1, 2] The literature is sparse on the OP induced LPN palsy. The tibialis posterior tendon transfer is well established technique for the treatment of post traumatic LPN palsy for correction of foot drop. [3, 4, 5] However there is no reported surgical treatment for OP induced LPN palsy. We did extra-membranous tibialis posterior tendon transfer for the correction of foot drop in this rare patient of OP induced LPN palsy.

A 32 years old male patient presented 4 years after ingestion of organophosphorous with left foot drop, loss of sensations on dorsum of 1st web space of the left foot. There was no sign of recovery of the lateral popliteal nerve palsy.

Case Report

A 32 years old male presented with left foot drop, loss of sensations on dorsum of 1st web space of the left foot since last 4 years. He had normal deep tendon reflexes in bilateral lower limbs. He had ingested OP esters 4 years before the presentation and was treated for the poisoning at local hospital.

Clinical, Electrophysiological (NCV & EMG) study of left leg showed non excitability of LPN with electrophysiological signs of denervation of tibialis anterior (TA), extensor hallucis longus (EHL) and extensor digitorum longus (EDL) muscles and Magnetic Resonance Imaging (MRI) of lumbo-sacral spine was normal.

The tibialis posterior tendon transfer was planned for the correction of the foot drop. The patient was given pre-operative exercises to strengthen the TP muscle for 3 weeks.

Ipsilateral Tibialis Posterior Tendon [Fig. 1, 2, 3, 4, 5] was transferred by extra-membranous route [Fig.6 , 7 , 8] to TA, EHL and EDL tendons under optimum tension. Limb was immobilized in a long above knee cast with knee in 30 degree flexion, ankle in 90 degrees of dorsiflexion and toes in neutral position. [Fig. 11] Non weight bearing ambulation using walker was resumed after one week. After 3 weeks above knee cast was converted to below knee cast for another 3 weeks. After 6 weeks post operatively cast was removed and dynamic ankle foot orthosis was given for another 3 months. Active & active assisted ankle & toe range of motion exercises were started and patient was asked to walk full weight bearing without support. Patient was followed up every month.

Results

At 6 months of follow up patient had full active grade 4 ankle & toes dorsiflexion with gr 5 active plantar flexion.[Fig.12]

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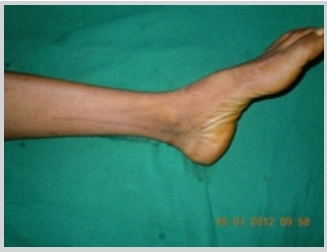
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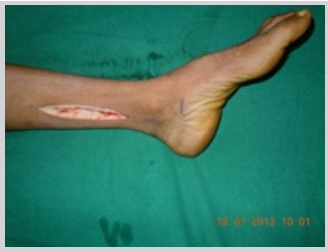
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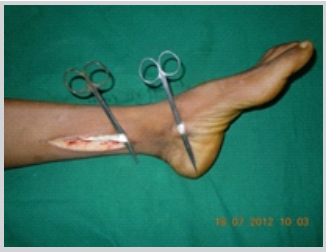
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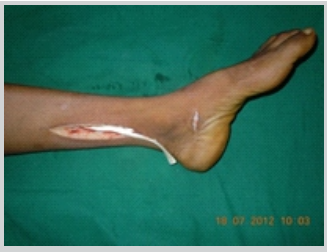
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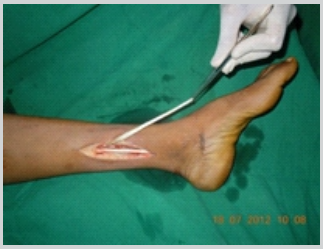
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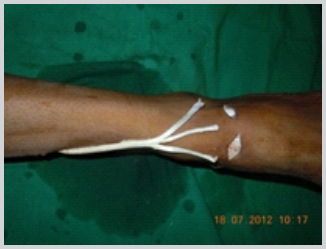
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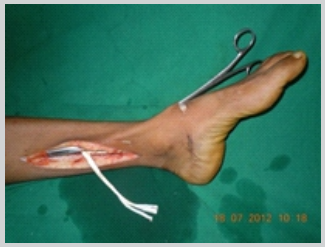
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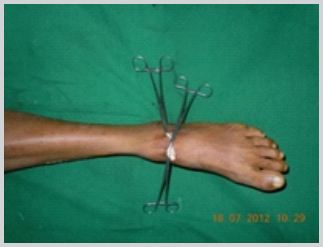
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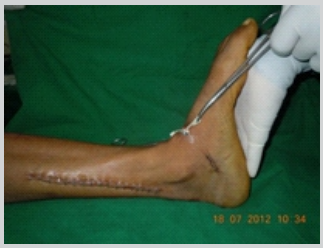
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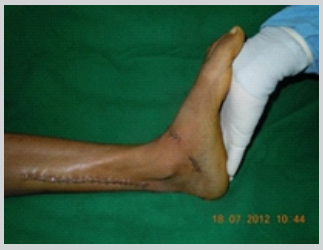
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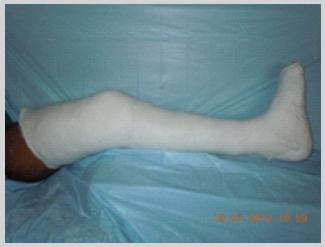
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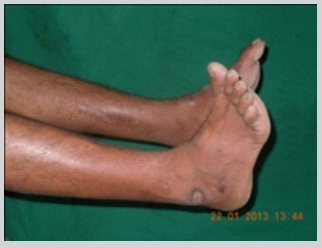
Figure 12

Discussion

Organophosphate-induced delayed polyneuropathy (OPIDP) is a rare toxicity resulting from exposure to certain organophosphorous (OP) esters. [1,2] It is characterized by distal degeneration of some axons of both the peripheral and central nervous systems occurring 1-4 weeks after single or short-term exposures. Cramping muscle pain in the lower limbs, distal numbness and paraesthesiae occur, followed by progressive weakness, depression of deep tendon reflexes in the lower limbs and, in severe cases, in the upper limbs. [1, 2] Signs include high-stepping gait associated with bilateral foot drop and, in severe cases, quadriplegia with foot and wrist drop as well as pyramidal signs. In time, there might be significant recovery of the peripheral nerve function but, depending on the degree of pyramidal involvement, spastic ataxia may be a permanent outcome of severe OPIDP.

At onset, the electrophysiological changes include reduced amplitude of the compound muscle potential, increased distal latencies and normal or slightly reduced nerve conduction velocities. The progression of the disease, usually over a few days, may lead to non-excitability of the nerve with electromyographical signs of denervation. Nerve biopsies have been performed in a few cases and showed axonal degeneration with secondary demyelination. [1]

Clinical, electrophysiological and histological findings in patients accidentally poisoned with the organophosphorus insecticide Dipterex are reported [2] Three to five weeks after insecticide ingestion signs of a distal sensorimotor (preponderantly motor) neuropathy occurred. The patients complain of paraesthesia in the lower limbs. There was distal weakness mainly of the legs, foot drop, difficult gait and muscle hypotonia. Ankle jerk was abolished while other tendon reflexes persisted. Two months or even later after poisoning, knee jerks in all the patients were very brisk and more and less accompanied by other pyramidal signs (Patellar clonus, abolishment of abdominal cutaneous reflexes, Babinski's sign). Clinical, electrophysiological and nerve biopsy data revealed a "dying-back" neuropathy in such patients.

Distal muscle fatigue was confirmed by failure of neuromuscular transmission on repetitive nerve stimulation. [2]

There is no response to the conservative treatment after the established nerve palsy. The tibialis posterior tendon transfer is time tested and excellent technique for the correction of the foot drop deformity. It has the advantage of restoring the active dorsiflexion of the foot with no secondary deficit in the foot.

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