

# ORGANOPHOSPHATE INDUCED DELAYED POLYNEUROPATHY: AN UNUSUAL PRESENTATION

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DOI: 10.5455/ijmsph.2013.220420132

Received Date: 13.03.2013

Accepted Date: 22.04.2013

## ABSTRACT

Organophosphate poisoning is an important cause of suicides in hospital admission. The present case is of a female presenting with loss of power in all four limbs two weeks following a suicidal attempt. Nerve conduction velocity test revealed axonal type of motor neuropathy in bilateral median and ulnar nerve and peroneal and tibial nerves were unexcitable bilaterally. Therefore, in all cases of neuropathy, ingestion of organophosphate, either accidental or suicidal should be looked for even if the initial phases of intoxication are not clinically well defined.

**KEY-WORDS:** Organophosphate Poisoning; Nerve Conduction Velocity Test; Motor Neuropathy

## Introduction

Organophosphates (OPs), potent inhibitors of acetylcholinesterase, are commonly used as pesticides throughout the world. Exposures to OPs cause a significant number of poisonings and deaths every year. The most common source of exposure is pesticide use in the agricultural industry, although cases of intentional poisoning may also occur.<sup>[1]</sup>

Organophosphorus poisoning could manifest in three phases (acute cholinergic effects, intermediate syndrome and organophosphate-induced delayed neuropathy).<sup>[2]</sup> Organophosphate induced delayed polyneuropathy (OPIDP), a sensory-motor distal axonopathy, usually occurs 1-4 weeks after single or short-term exposures and it may be progressive and severe. 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.<sup>[3]</sup>

Diagnosis of organophosphate-induced neuropathy rests on a history of appropriate exposure in a patient with progressive motor deficit greater than sensory neuropathy. Electrodiagnostic studies demonstrate an axonal neuropathy. There are no specific features and nerve biopsy reveals axonal degeneration with

secondary demyelination.<sup>[1,3]</sup> The clinical picture of the toxic neuropathy may be characterized by a distal paresis in the lower limbs associated with sensitive symptoms.<sup>[4]</sup>

We present a 25-year-old female patient who ingested an unknown organophosphorus in a suicide attempt which resulted in delayed peripheral neuropathy.

## Case Report

A 25-year-old female patient presented at the emergency department of a tertiary care hospital in eastern India complaining of excessive salivation, nausea, vomiting and abdominal cramp. The patient, in an attempt to commit suicide, ingested large amounts of an organophosphate insecticide few hours before.

Physical examination revealed miotic pupils and fall of arterial blood pressure. She received atropine and pralidoxime as treatment and improved symptomatically. During her psychiatric examination, the patient reflected depressive mood. After regression of her symptoms, she was discharged from the hospital after five days.

However, two weeks after the discharge, she presented at the Medicine OPD complaining of weakness and loss of power in all four limbs. She

was admitted in the hospital for further investigations. The neurological examination revealed quadriplegia with motor deficit in all the limbs. The ankle jerk was abolished while the knee jerk preserved. Cranial nerves were not involved. There was loss of temperature discrimination and nociception in distal lower limbs. Touch pressure, vibratory and joint position senses were not involved.

Nerve conduction velocity test revealed axonal type of motor neuropathy in bilateral median and ulnar nerve and peroneal and tibial nerves were unexcitable bilaterally. Sensory parameters were normal in median and ulnar nerves while bilateral sural SNAPs were absent. Electromyography revealed marked distal denervation of the lower limb muscles and partial distal denervation of the upper ones.

Therefore, keeping in view of history of organophosphate poisoning followed by signs of quadriplegia with axonal motor neuropathy pattern on electrophysiology, a diagnosis of organophosphate induced delayed neuropathy was established.

The patient was managed conservatively and was advised psychiatric counselling and physiotherapy after discharge. She was also advised regular follow up and after 30 months the clinical condition of her arms greatly improved. Partial recovery was also evident in the legs.

## Discussion

Organophosphorus (OP) compounds constitute a heterogeneous category of chemical agents with wide-spread use throughout the world, mainly in agriculture. On exposure, they have the potential to irreversibly inhibit the cholinesterase, acetylcholinesterase, and neuropathy target esterase (NTE), in humans and animals.<sup>[5]</sup>

These products are readily available "over the counter", despite them being a major cause of morbidity and mortality. Exposure to organophosphates in an attempt to commit suicide is a key problem, particularly in the developing countries, and is a more common cause of poisoning than the chronic exposure

experienced by farmers or sprayers in contact with pesticides.<sup>[6]</sup> Exposure may occur transdermally, via the respiratory tree or from the gastrointestinal tract.

Acute organophosphate insecticide poisoning can manifest 3 different phases of toxic effects based largely on the time of occurrence<sup>[3]</sup>:

*Type I Paralysis or Cholinergic Crisis:* Excessive stimulation of muscarinic receptors is responsible for intense cholinergic effects, which are always apparent within a day of exposure, often within hours. Symptoms include tachycardia or bradycardia, diarrhea, vomiting, fasciculation, sweating, salivation and micturition.<sup>[7]</sup>

*Type II Paralysis or Intermediate Syndrome:* It follows the intense cholinergic crisis of organophosphorus poisoning and occurs in up to 20%-50% of cases depending on the severity of poisoning, its duration, and on the type of organophosphorus compound. The pathogenesis is presumed to be dysfunction of neuromuscular junction caused by down regulation of presynaptic and postsynaptic Nicotinic receptors due to release of excessive Ach and Ca<sup>2+</sup> respectively.<sup>3</sup> The cardinal features comprise muscular weakness, affecting predominantly the proximal limbs muscles and neck flexors. Cranial-nerve palsies are common.<sup>[8,9]</sup>

*Type III Paralysis or Organophosphate Induced Delayed Neuropathy (OPIDP):* It is characterized by cramping muscle pain in the legs, paresthesia, and motor shortcoming beginning 10 days to 3 weeks after the initial exposure. It is a pure motor or predominantly motor axonal neuropathy characterized by wrist drop and foot drop with minimal or no sensory loss which occurs 7-20 days after exposure to an OP agent.<sup>[10]</sup>

OPIDP is a rare toxicity resulting from exposure to certain organophosphorus (OP) esters. The pathogenesis of OPIDP is presumed to be due to phosphorylation and ageing of an enzyme in axons called neurotoxic esterase or neuropathic target esterase (NTE). Inhibition of NTE causes degeneration of predominantly long axons, with loss of myelin and macrophage accumulation in nerves leading to motor axonal neuropathy.<sup>[11]</sup>

The symptoms in OPIDP are predominantly motor. Weakness appears early and initially involves legs muscles before those of the hands. Sensitive symptoms as well as paresthesia and hyperesthesia may be present. Proximal weakness may be present in severe cases. 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.<sup>[6]</sup>

In the present case, the patient presented with quadriparesis after two weeks of the intoxication. The motor function improved gradually over 2 years.

## Conclusion

Therefore, ingestion of organophosphate, either accidental or suicidal should be considered in cases of neuropathy, even if the initial phases of intoxication are not clinically well defined.

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**Cite this article as:** Datta S, Bhattacharjee S. Organophosphate induced delayed polyneuropathy: An unusual presentation. Int J Med Sci Public Health 2013; 2:760-762.

**Source of Support:** Nil

**Conflict of interest:** None declared