

## Organophosphate Induced Neuropathy: A Case Report

Ashwin Arvind Rajbhoj<sup>1\*</sup>, Deepak Giri<sup>1</sup>, Sandip Patel<sup>1</sup>, Kulbhushan Marathe<sup>1</sup>, Tanmay Yermal<sup>1</sup>, M. A. Ghanekar<sup>2</sup>

<sup>1</sup>Post Graduate Resident, Department of Medicine, PDVVPF's Medical College and Dr. Vikhe Patil Hospital, Vilad Ghat, Ahmednagar, India

<sup>2</sup>Professor and Head, Department of Medicine, PDVVPF's Medical College and Dr. Vikhe Patil Hospital, Vilad Ghat, Ahmednagar, India

### \*Corresponding Author:

Name: Dr. Ashwin Arvind Rajbhoj

Email: [ash127win@gmail.com](mailto:ash127win@gmail.com)

**Abstract:** Organophosphorus (OP) compounds constitute a heterogeneous category of chemical agents with wide-spread use throughout the world, mainly in agriculture. The ability of some of these chemicals to cause an irreversible, progressive delayed neuropathy was recognized and diagnosis rests on a history of appropriate exposure in a patient with progressive motor deficit greater than sensory neuropathy. We describe a 19 year old female who developed distal weakness in the lower limbs to the extent that she could not walk without support 3 weeks later after she consumed chlorpyrifos based insecticide. Electrophysiological study confirmed fairly symmetrical lower limb motor axonal neuropathy. It is therefore recommended that, every patient of Organophosphate poisoning should be followed up for at least one month.

**Keywords:** Organophosphate, Weakness, Neuropathy, Electrophysiology.

### INTRODUCTION

Organophosphorus compounds are chemical agents mainly in agriculture with wide-spread use throughout the world. Particularly in the developing countries organophosphates are used in an attempt to commit suicide. Some of these chemicals have the ability to cause an irreversible, progressive delayed neuropathy, recognized as early as the 1890s, when a 15% solution of tri-ortho-cresyl phosphate (TOCP) or tri-ortho-tolyl phosphate (TOTP) was used to treat tuberculosis [1].

An example of organophosphate induced neuropathy occurred during the 1930. Thousands of men in the American South and Midwest developed arm and leg weakness and pain after drinking "Ginger Jake," (a "medicinal" alcohol substitute) that contained an adulterated Jamaican ginger extract containing TOCP [2].

Diagnosis of organophosphate induced neuropathy rests on a history of appropriate exposure in a patient with progressive motor deficit greater than sensory neuropathy. Clinically, toxic neuropathy may be characterized by a distal paresis in the lower limbs that is associated with sensitive symptoms [3]. Electro diagnostic studies demonstrate axonal neuropathy [4, 5].

### CASE REPORT

A 19 year old young female consumed a suicidal organophosphate insecticide (100 ml of chlorpyrifos). She had vomiting with severe abdominal pain, pinpoint pupils and was immediately treated with atropine and pralidoxime. She was unconscious with oxygen support initially which gradually improved over a period of next 48 hours. She recovered completely and was discharged from the hospital after 7 days.

After 15 days she developed tingling/numbness and slight weakness of lower limb over a period of 7 days. After which, she developed distal weakness in the lower limbs which progressed in next 3 days to the extent that she could not walk without support. There was no weakness in upper limbs with no involvement of bowel and bladder. General physical examination was normal. On neurological examination cranial nerve examination was normal. There was atrophy of distal group of muscle with increased tone in lower limbs. Lower limb power was grade 0 with exaggerated knee and ankle reflexes. Planters were bilaterally absent. There was no sensory deficit. Other systemic examinations were normal.

On investigation her routine blood tests and biochemical parameters were normal. X ray spine was normal. Electrophysiological study revealed markedly reduced amplitude of the compound muscle action

potential (CMAP) with reduced motor nerve conduction velocity (MNCV) in left tibial nerve. CMAP and MNCV were not recordable in peroneal nerve of both lower limbs and mildly reduced in right median and ulnar nerve. Sensory nerve action potential (SNAP) was normal in both upper limbs but mildly reduced in right lower limb. These findings were suggestive of generalized, fairly symmetrical lower limb motor axonal neuropathy. Therefore, in view of history of organophosphate poisoning, she was diagnosed of having organophosphate induced delayed neuropathy.

**Table 1: Electrophysiological study (CMAP = Compound muscle action potential, NCV = Nerve conduction velocity; NR = No response; Rt = Right; Lt = Left)**

	Normal values	Patients values
<b>Motor nerve conduction</b>		
NCV(m/s)		
Rt. Tibial	>48	44.3
Lt. Tibial	>48	37.2
Rt. Peroneal	>44	NR
Lt. Peroneal	>44	NR
<b>CMAP Amplitude(mV)</b>		
Rt. Tibial	>5	0.5
Lt. Tibial	>5	0.3
Rt. Peroneal	>2	NR
Lt. peroneal	>2	NR

## DISCUSSION

Organophosphate compounds are commonly used as insecticides, petroleum additives, lubricants, plastics modifiers, antioxidants and flame-retardants [6].

Accidental pesticide exposure from agricultural spraying may cause intoxication. It may occur in individuals who mix or apply the pesticide or through dermal exposure [16]. In India, pesticides containing organophosphate are also intentionally ingested in suicide attempts as they are readily available and accessible. They may be absorbed via skin or respiratory and gastrointestinal tracts [6]. The organophosphate esters are lipophilic that results in penetration into the central and peripheral nervous systems [7]. The different organophosphorus compounds associated with neuropathy include tri-*o*-cresyl phosphate (TOCP), mipafox, leptophos, trichlorfon, chlorpyrifos, trichlorfon, malathion, metrifonate parathion and metamidophos [8].

The clinical sequences of organophosphorous poisoning can be divided in 3 steps: Type I, II and III.

In Type I syndrome excessive stimulation of muscarinic receptors occurs and is responsible for intense cholinergic effects. Cholinergic symptoms include tachycardia or bradycardia, diarrhea, vomiting, fasciculation, salivation, sweating and micturition. They are always apparent within a day of exposure, often within hours. Atropine is used for treatment and doses vary depending on the clinical state [6].

Type II syndrome or intermediate syndrome is characterized by intense cholinergic crisis. It occurs in up to 20%-50% of cases that depends on the severity of poisoning, duration, and type of compound [9]. The symptoms include muscular weakness, affecting predominantly the proximal limb muscles and neck flexors. Cranial-nerve palsies are common. Type II syndrome is associated with death risk due to associated respiratory depression. The clinical courses may last from 5-18 days [10].

Type III syndrome or organophosphate induced delayed neuropathy (OPIDN) is associated with cramping muscle pain in the legs, paraesthesia and motor shortcoming that begins in 10 days to 3 weeks after initial exposure. Signs of Type III syndrome include foot drop, absent ankle jerks, weakness of intrinsic hand muscles, hip and knee flexors [11].

Senanayake's [12] reported, nearly 50% of patients had some evidence of pyramidal tract dysfunction. Khurana and Prabhakar [13] reported that pyramidal involvement is usually present. Initially, peripheral nerve involvement predominates, while corticospinal tract signs evolve more slowly.

In this case after ingesting chlorpyrifos, patient had pyramidal tract involvement in the form of increased knee and ankle reflexes. It shows that the quantity of chlorpyrifos ingested by the patient was substantial.

Fisher reported, a patient who was exposed to organophosphates, had no acute manifestations of organophosphate toxicity. But he developed polyradiculitis, elevated cerebral spinal fluid protein level, cranial nerve involvement and electromyography pattern that are characteristics of the Guillain-Barré syndrome [14]. Bekarovski *et al.* also reported rare clinical presentation after intense organophosphate poisoning [15].

## CONCLUSION

Ingestion of organophosphate, either accidental or suicidal should be considered in cases of neuropathy; even there is no clinically well defined initial phase.

Patients with severe deficits may not recover completely; residual claw hand deformity, persistent atrophy, and foot drop, spasticity and ataxia may be there [6]. The most important differential diagnosis of OPIDN include acute disseminated encephalomyelitis and Guillain-Barré syndrome [8]. Intoxications should also be part of the differential diagnostic of paraparesis. Every patient of organophosphate poisoning should be followed up for at least one month.

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