

# Delayed onset neuropathy along with recurrent laryngeal nerve palsy due to organophosphate poisoning and the role of physiotherapy rehabilitation

Jaimala Vijay Shetye, Swati M. Surkar, Niteen D. Karnik<sup>1</sup>, Amita A. Mehta

## Abstract

Organophosphorus poisoning is a major global cause of health problems and the leading cause of mortality and morbidity in the developing countries. In this, the inhibition of acetyl-choline esterase and neurotoxic esterase along with nicotinic receptor involvement produces three well-identified and documented clinical phases: The initial cholinergic phase, which is a medical emergency often requiring management in an intensive care unit; the intermediate syndrome, during which prolonged ventilator care is necessary; and finally delayed neurotoxicity. Vocal cord paralysis is rare and leads to aphonia. Role of physiotherapy rehabilitation is substantial in all three stages and aims at early weaning off from mechanical ventilator until the functional independence and community integration of the patient.

**Keywords:** Organophosphate induced delayed neurotoxicity, organophosphate poisoning, physiotherapy, recurrent laryngeal palsy

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## Introduction

The increasing global use of pesticides has led to organophosphate (OP) poisoning becoming a leading cause of mortality and morbidity in developing countries.<sup>[1]</sup> Acute OP poisoning is known to have neurotoxic effects. Cholinergic crisis, intermediate syndrome (IMS), and OP-induced delayed neurotoxicity (OPIDN) are the three stages observed following OP intoxication.<sup>[2-7]</sup> OPIDN results in sensory-motor distal axonopathy characterized by distal degeneration of some axons of both the peripheral and central nervous systems; even resulting in pyramidal tract and posterior column involvement.<sup>[8,9]</sup> Recurrent laryngeal palsy is very rare and leads to aphonia in these patients.<sup>[10,11]</sup>

We document the case of a patient who showed all three stages of OP poisoning along with corticospinal tract signs, recurrent laryngeal nerve palsy, aphonia, and tracheal stenosis and the role of physiotherapy in her rehabilitation.

## Case Report

A 20-year-old female patient attempted suicide by ingesting a large amount of OP insecticide. She was admitted in a private hospital in an unconscious, gasping state with frothing from the mouth, smelling of insecticide. Pupils were miotic and fasciculations were evident. She was immediately intubated and was kept on synchronized intermittent mandatory ventilation (MV).

Seven days later she was brought to Medical and Neurological Intensive care unit of our hospital on MV. On admission, she had poor respiratory effort. She was conscious, oriented, had grade four muscle power in upper and lower limbs, without sensory symptoms. On day 12, lobar pneumonia was diagnosed. She had copious purulent secretions which necessitated tracheostomy.

## From:

Departments of Physiotherapy, <sup>1</sup>Medicine (Medical ICU), Seth G. S. Medical College and K. E. M. Hospital, Mumbai, Maharashtra, India

## Correspondence:

Dr. Jaimala Vijay Shetye, P. T. School and Center, 1<sup>st</sup> Floor, Dhrumal Bajaj Ortho Center, Seth G. S. Medical College and K. E. M. Hospital, Parel, Mumbai - 400 012, Maharashtra, India. E-mail: [jaimalavs@gmail.com](mailto:jaimalavs@gmail.com)

She was treated with Piperacillin-Tazobactam. Chest physiotherapy along with respiratory muscle training by inspiratory muscle trainer with threshold resistance for strengthening of weak respiratory muscles proved an adjunct for the weaning off of the patient from mechanical ventilation. In stepdown care, her muscle power slowly reduced to grade two in upper limb and grade three in lower limb. Three days post-extubation, she developed rapid onset deterioration in muscle power to grade zero and absent deep tendon reflexes, but without respiratory deterioration. She developed bilateral foot drop and claw hand deformity. Nerve conduction studies indicated pure motor axonal neuropathy. Patient developed stridor when an attempt to close the tracheotomy was carried out. Bronchoscopy revealed tracheal stenosis 2.3 cm distal to the vocal chords along with bilateral adductor vocal cord paralysis indicating involvement of recurrent laryngeal nerve, which explains aphonia evident during partial closure trial of tracheostomy.

Forty five days from the ingestion of OP insecticide, she developed grade II spasticity according to Modified Ashworth Scale in bilateral upper and lower limbs with flexor spasms in lower limbs and exaggerated deep tendon reflexes, which indicate pyramidal tract involvement. Sensitive symptoms of muscle pain, tenderness and cramps developed in bilateral quadriceps and calf muscles. This suggested the presence of OPIDN.

Physiotherapy was aimed at preventing neuro-musculoskeletal co-morbidity. Electrical stimulation with intermittent galvanic current was given to paralyzed distal muscles of upper and lower extremities to restore their properties and hence prevent further muscle wasting. Strengthening and proprioceptive neuromuscular facilitation for weak muscles, stretching of hand flexors and gastrosoleus and orthotic devices prevented further deformities. Tilt table supported standing helped to overcome the effects of prolonged bed rest and postural hypotension. After discharge the patient was referred to a community rehabilitation center for further management.

Marital discord with violently abusive husband was the reason for this patient to attempt suicide. The patient and her relatives were counseled by psychiatrist. Her parents gave her emotional support and agreed to support her socially and financially. She was happy to separate from her husband.

## Discussion

The liposolubility of OP esters in insecticides permits penetration in central and peripheral nervous system.

The clinical phases included an acute cholinergic crisis characterized by tachycardia or bradycardia, diarrhea, vomiting, fasciculation, sweating, salivation and micturition, convulsion and coma. Inhibition of acetyl-choline esterase leads to accumulation of acetylcholine at nerve synapses and neuromuscular junctions resulting in overstimulation of acetylcholine receptors. The initial over-stimulation followed by paralysis of cholinergic synaptic transmission in the central nervous system, autonomic ganglia, and parasympathetic and sympathetic nerve endings is the known mechanism for cholinergic crisis phase.

Subsequently, the effect on the nicotinic receptor appears and the patient gets IMS. This was characterized in this patient by limb, neck muscle weakness, respiratory fatigue or failure and cranial nerve palsy. Mechanical ventilation was needed for a short duration during this phase. She later developed OPIDN characterized by cramping muscle pain, paresthesia and motor weakness and absent deep tendon reflexes with normal joint position and vibration sense.

Six weeks after poisoning, corticospinal tract signs evolved slowly and our patient showed spasticity, abnormal posturing and exaggerated deep tendon reflexes. This is termed as OPIDN. The depression of neurotoxic esterase by some OP insecticides in addition to choline esterase can contribute to delayed neurotoxicity.

Our case showed all three stages along with the recurrent laryngeal nerve palsy, which is rare. This delayed effect could be the result of phosphorylation of nervous tissue protein with resulting Wallerian axonal degeneration. The recurrent laryngeal nerve palsy could also be the effect of mechanical factors due to prolonged intubation and effect of tracheostomy and ventilation. OPIDN is generally sensory motor neuropathy, but our patient showed exclusive motor symptoms. Nerve conduction studies also supported the clinical findings by exhibiting motor axonal neuropathy.

The treatment of OPIDN is long-term and physiotherapy plays a substantial role in making patient functionally independent and enabling integration into the community. This suggests that ongoing evaluation of changes in signs is essential for appropriate selection of physiotherapy modality in various phases. Rehabilitation strategies need to be planned before discharge and communication with the patient and community rehabilitation physiotherapist is essential during further follow-up to achieve functional integrity of the patient.

At present that is 18 months from onset, our patient is gainfully self-employed doing mehendi art work.

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