



A Case Report

A Rare Case of Organophosphate Induced Delayed Neuropathy

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Abstract :

In this study we report a case of Organophosphate Induced Delayed Neuropathy (OPIDN) that is a rare clinical condition that occurs in association with ingestion of large amounts of organophosphorus compound. The clinical picture ranges from transient paraesthesia, tingling, and numbness to symmetrical motor weakness starting distally in lower limbs which then ascends to involve the upper limbs as well. Here is a case of 40-years-old female who presented with vomiting, excessive salivation and altered mentation. She had an alleged history of ingestion of organophosphate compound, dichlorvos 70 EC. She experienced an episode of generalised tonic-clonic seizures, required mechanical ventilation, and was managed in the ICU. On the 10th day she developed bilateral lower limb symmetrical weakness, however neck and eye movements were preserved. Higher mental function and cranial nerves functions were intact. MRI brain and cervical spine revealed no abnormality. Nerve conduction study was suggestive of motor axonal

neuropathy in all four limbs. She was diagnosed as organophosphate induced delayed neuropathy and was managed with steroids.

Keywords: organophosphates; pesticide; dichlorvos 70 EC; neuropathy; neuropathy target esterase; steroids

Introduction:

Organophosphorus (OP) is widely used as a pesticide worldwide in agriculture, public health, and households.[1] These are the organic derivatives of phosphorous-containing acids, which act on the neuromuscular junction (NMJ).[2] According to World Health Organization (WHO) reports, three million OP poisoning cases are reported annually around the globe.[3,4] In India, deaths due to OPC poisoning are more common in southern and central India.[5] They are widely used for self-harm due to their ready availability and easy accessibility.[6] OPC are irreversible inhibitors of choline esterase and pseudo-cholinesterase (butyrylcholinesterase) enzymes, which cause the breakdown of acetylcholine (neurotransmitter) into choline and acetic acid.[5,7] Organophosphorus poisoning can be divided into three phases: Acute cholinergic effects, intermediate syndrome, and organophosphate-induced delayed neuropathy.[8] The initial phase is characterized by excessive stimulation of muscarinic and nicotinic receptors. Symptoms typically appear within hours to a day of exposure and include cholinergic effects such as increased salivation, lacrimation, pinpoint pupils, and shortness of breath.[8] Following the cholinergic

crisis, this intermediate phase can affect 20%–50% of cases. It is marked by muscle weakness, especially in proximal limb muscles and neck flexors, along with cranial-nerve palsies. Symptoms typically occur 24–96 h after exposure and may last from 5 to 18 days.[9] Organophosphorus-induced delayed neurotoxicity (OPIDN) also known as Type III syndrome can present as a central-peripheral distal axonopathy and is characterized by cramp-like muscle pain with a tingling sensation followed by paraplegia or tetraplegia.[8,10] It typically begins 10 days to 3 weeks after initial exposure, and the course is subacute, typically occurring within 2 weeks after initial symptoms. This condition primarily affects the motor nerves.[11]

Case report:

This case describes a 40-year-old female who presented to the emergency department of Hamidia Hospital, Bhopal, with altered sensorium, excessive salivation, and vomiting following the ingestion of approximately 200 ml of pesticide containing dichlorvos 70 EC (an organophosphate compound). According to the accompanying attendant, the patient experienced 3 to 4 episodes of vomiting after ingestion, followed by which she fell on the floor and was not listening or following attendant's instructions. She was promptly brought to the hospital where she presented with disorientation, moist skin, drooling, and bilateral pinpoint pupils. The vitals were relatively stable: heart rate 64 bpm, blood pressure 128/80 mmHg, respiratory rate 14 breaths per minute, and SpO₂ 96% on room air. Upon neurological examination, no significant findings were noted initially. The patient had no history of any known comorbidities, psychiatric illness, or relevant family history. Given the symptoms and history of pesticide ingestion, the diagnosis of organophosphate poisoning was made, and treatment was initiated, that included gastric lavage and administration of atropine. During this period, she had an episode of a generalized tonic-clonic seizure, which led to aspiration, necessitating mechanical ventilation. Despite the intensive treatment in the emergency room, including 24 cc of atropine (administered in escalating doses), the patient could not be weaned off mechanical ventilation. She continued to

require repeated endotracheal tube suction, and bilateral fine crepitations were heard on chest auscultation. She also developed irritability, and later, ventilator-associated pneumonia (VAP), which further prolonged her stay on the ventilator. Her white blood cell count rose to 24,000/ μ L with elevated C-reactive protein (CRP) levels, prompting the use of broad-spectrum antibiotics. On the 10th day, the patient exhibited decreased movement in the lower limbs, while her neck and eye movements remained preserved. A comprehensive neurological examination revealed normal higher mental functions and intact cranial nerve functions. There was muscle weakness in the upper limbs (power 3/5 at the wrist and 2/5 at the elbow and shoulder) and marked weakness in the lower limbs, with foot drop and reduced power (1/5 at the ankle joints and metatarsophalangeal joints, 2/5 at the knee joints). Reflexes were diminished in both lower limbs, and tone was reduced. Sensory examination was normal, and the patient's lab results showed mild anaemia, normal leukocyte and platelet counts, and stable renal and liver functions with hypokalaemia (serum potassium 2.4 mmol/L). [Table 1: Laboratory Parameters of the Patient]. Hypokalaemia was corrected, but the motor weakness persisted. On day 12, the patient was successfully extubated after her pneumonia improved, though her lower limb weakness persisted. A neurology consultation was sought, and both MRI of the brain and cervical spine revealed no abnormalities. Nerve conduction studies confirmed the presence of motor axonal neuropathy affecting all four limbs. Image 1: Nerve Conduction Study of Bilateral upper and lower limbs. The patient was then treated with a 3-day course of 1g/day of intravenous methylprednisolone, vitamin B complex supplements, and regular extensive physiotherapy of all four limbs. Gradually, the patient regained strength in her upper limbs by day 20 and in her lower limbs by day 26. She was discharged after a significant recovery, with near-total resolution of the neurological deficits.

Discussion:

Organophosphates are one of the most common insecticides used worldwide. They can cause toxic effects if ingested, inhaled or absorbed

via transdermal route. Their lipid solubility allows them to penetrate the central and peripheral nervous system. OPIDN as the name suggests is a late neurological complication of ingestion of certain organophosphate esters. It is characterised by distal degeneration of some axons of both the peripheral and central nervous systems occurring 1–4 weeks after single or short-term exposures. The exact pathogenesis of the OPIDN is not clear yet but it is presumed that it is caused by the covalent inhibition of neuropathy target esterase (NTE).[13] NTE is located in endoplasmic reticulum (ER) of neural tissues such as the brain, spinal cord, and peripheral nerves, which catalyzes the deacylation of phosphatidylcholine (PC) and lysophosphatidylcholine (LPC) to glycerophosphocholine (GPC). This results an increase of PC, LPC, and sphingomyelin levels and a decrease of GPC, phosphatidylethanolamine, this disturbed metabolism of important membrane phospholipids, leads to axonopathy.[13] Organophosphate-induced delayed neuropathy (OPIDN) primarily affects motor function, with weakness typically beginning in the leg muscles before gradually spreading to the hand muscles. Symptoms can vary in severity but the hallmark is the gradual onset of weakness, which tends to worsen over time before partial recovery occurs. While sensory complaints are rare, patients often report heightened sensitivity, experiencing symptoms such as paraesthesia (tingling or prickling sensations) and hyperesthesia (increased sensitivity to stimuli) which contribute to the discomfort and functional challenges patients experience. Guillain Barré syndrome, acute disseminated encephalomyelitis, transverse myelitis, infectious/inflammatory myelopathy, Vitamin B12 and copper deficiency myelopathy are a few of the differential diagnoses of OPIDN. [14] Although there is no specific treatment for OPIDN, use of injectable steroids, vitamin B12 agents and physiotherapy has proven to be beneficial.

Conclusion:

Organophosphate-induced delayed neuropathy (OPIDN) is a rare but significant neurological complication following acute organophosphate poisoning, characterized

predominantly by motor axonal involvement. This case highlights the importance of early recognition of delayed neurological deficits even after initial stabilization of cholinergic symptoms. A high index of suspicion, timely neurophysiological evaluation, and exclusion of other differentials are essential for accurate diagnosis. Although no definitive treatment exists, supportive management with steroids, vitamins, and physiotherapy can lead to substantial recovery. This case underscores the need for close follow-up of patients with organophosphate poisoning to identify and manage late-onset complications effectively.

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Table 1 : Laboratory Parameters of the Patient

Lab parameters	Value	Unit	Normal Range
Haemoglobin	8.4	g/dl	11-13
WBCs	9630	per cu.mm	5000-11000
Platelets	2.13	per cu.mm	1.25-4.50
MCV	87.70	fL	
RDW	18.90	%	11.5-14.5
Urea	39	mg/dl	17-43
Creatinine	0.07	mg/dl	0.51-0.95
Uric acid	5.37	mg/dl	2.6-6
S. Sodium	136.8	mmol/L	136-145
S. Chloride	104.4	mmol/L	98-107
S. Potassium	2.76	mmol/L	3.5-5.0
S. Magnesium	2.3	mg/dl	1.8-2.4
S. Calcium	7.1	mg/dl	8.5-10.1
Total Bilirubin	0.59	mg/dl	2.6-6
Direct Bilirubin	0.11	mg/dl	0.3-1.2
SGOT	14.10	U/L	0-35
SGPT	13.71	U/L	0-35
ALP	79	U/L	30-120
S. Protein	5.88	gm/dl	6.6-8.3
S. Albumin	3.5	gm/dl	3.5-5.2

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