VOLUME - 14, ISSUE - 03, MARCH - 2025 • PRINT ISSN No. 2277 - 8160 • DOI : 10.36106/gjra

Original Research Paper | Internal Medicine

ORGANOPHOSPHORUS COMPOUND-INDUCED DELAYED NEUROPATHY: A RARE COMPLICATION OF OP POISONING

Dr. Mary Lalremruati — 3rd Year Medicine Resident

Dr. Chintan Chauhan — 2nd Year Medicine Resident

ABSTRACT Introduction: Organophosphorus compound-induced delayed neuropathy (OPIDN) is a rare neurological complication following organophosphate poisoning. It results from irreversible inhibition of neuropathy target esterase (NTE), leading to axonal degeneration in the peripheral and central nervous system. Symptoms typically appear 1–3 weeks post-exposure.

KEYWORDS :

INTRODUCTION

Organophosphorus (OP) compounds are widely used as pesticides and industrial chemicals. OP poisoning primarily causes cholinergic toxicity due to acetylcholinesterase inhibition. However, some OP compounds can also cause organophosphorus-induced delayed neuropathy (OPIDN), a distinct entity characterized by delayed onset of neurological symptoms (1–3 weeks post-exposure) due to neuropathy target esterase (NTE) inhibition. This leads to axonal degeneration, particularly affecting long myelinated nerves. Unlike acute OP poisoning, OPIDN is not related to cholinergic toxicity and lacks a specific antidote.

Case Presentation

Patient Information

Age/Sex: 18-year-old female
Presenting Complaint: Intentional ingestion of chlorpyrifos pesticide
Initial Symptoms: Nausea, vomiting, abdominal discomfort
Clinical Findings on Admission
Consciousness: Alert, oriented

Vital Signs:
Pulse: 88 bpm
BP: 110/76 mmHg
SpO_2: 90% on room air
RR: 18/min
Pupils: Miotic, equal (0.5 mm)

Hospital Course
Day 1-7: Patient developed respiratory distress, requiring intubation; extubated on Day 7.
Day 12: Developed progressive lower limb weakness, without upper limb involvement.

Neurological Examination (Day 12)
Tone:
Normal in upper limbs
Decreased in lower limbs

Power:
Upper limbs: 5/5
Lower limbs: 3/5 (hip, knee, ankle)

Reflexes:
Upper limbs: Present bilaterally
Lower limbs: Absent (knee and ankle)
Plantar Reflex: Bilaterally absent
Cranial Nerves, Sensory & Higher Mental Function: Intact

Laboratory & Diagnostic Findings

Haemoglobin	11gmdl
Total leucocyte count	10000
Platelet	2.6 lacs
RBS	101 gmdl
Urea	20mg%
creatinine	0.5
Sr cholinesterase	Day 1- 201 IU/L Day 3 - 345 IU/L Day 7 - 567 IU/L DAY 12 - 2877 IU/L
CXR	WNL

MRI BRAIN : WNL
CSF : acellular, sugar 59mg/dl ,protein 78 mg/dl
NCV : suggestive of demylination

Treatment & Course
Methylprednisolone 1 g IV OD for 5 days Tapered oral prednisolone (60 mg OD, gradually reduced over 4 weeks)

Response to Therapy:
By Day 4 of steroid therapy, tone normalized
Power improved from 3/5 to 4/5 in lower limbs

DISCUSSION

OPIDN is a rare but significant complication of OP poisoning. The pathophysiology involves irreversible inhibition of NTE, leading to distal axonopathy. Symptoms include progressive lower limb weakness, absent reflexes, and motor dysfunction, typically appearing 1-3 weeks post-exposure. Unlike acute OP poisoning, OPIDN is not due to cholinergic toxicity and lacks a specific antidote.

Factors Affecting Prognosis:

1. Severity of Initial Toxicity: Higher exposure levels correlate with prolonged neuropathy.
2. Early Management: Prompt recognition and supportive care improve recovery chances.
3. Extent of Axonal Damage: Severe axonopathy results in permanent deficits.
4. Neuroregeneration Capacity: Partial nerve recovery is possible over months to years.

CONCLUSION

OPIDN is a rare but potentially debilitating complication of OP poisoning. This case underscores the delayed neurological manifestations, particularly progressive lower limb weakness. Early diagnosis, supportive care, and corticosteroid therapy may improve outcomes, but recovery varies based on axonal damage severity.

Patient Perspective

The patient was educated about OPIDN and advised long-term neurological follow-up. She showed gradual improvement but required ongoing physiotherapy.

Informed Consent

Written informed consent was obtained from the patient for publication of this case report.

REFERENCES

1. Ellenhorn's Medical Toxicity: Diagnosis & Treatment of Human Poisoning.
2. Sivagnanam S et al. Potential Therapeutic Agents in OP Poisoning. Critical Care, 2002.
3. Patial R, Kapoor D et al. Pralidoxime for Reducing Nicotinic Cholinergic Morbidity in OP Poisoning.