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RESEARCH ARTICLE

CHLORPYRIPHOS INDUCED DELAYED MYELOPATHY & MOTOR NEUROPATHY

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ABSTRACT

Introduction: Organophosphate intoxication can cause serious life threatening acute neurological complications such as seizures, paralysis, neuromuscular and cardiac conduction disorders. Less often, a predominantly motor and delayed axonal neuropathy can occur. This syndrome is due to inhibition of neuropathic target esterase. ***Case report:*** Herein we describe a 20-year-old-male patient who after ingestion of large amount of (200 ml) Chlorpyriphos based insecticide had an acute cholinergic crisis followed by development of lower limb weakness 8 weeks after initial exposure to organophosphorous. Pyramidal tract involvement was also observed as the patient developed spastic paraparesis in lower limbs. Electrophysiological study was characterized by motor axonal polyneuropathy. ***Conclusion:*** This was a case of organophosphate induced delayed neuropathy with myelopathy leading to spastic paraparesis. Hence all patients with organophosphorous poisoning should be under regular follow up and examined for neurological involvement.

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INTRODUCTION

Organophosphate (OP) poisoning is the most common poisoning in India accounting for approximately 50% of the poisoning related hospital admissions. It can present as acute cholinergic crisis, intermediate syndrome and organophosphate-induced delayed neuropathy (OPIDN)[1]. Acute cholinergic crisis, as a result of inhibition of acetylcholinesterase, can manifest either with the involvement of muscarinic (lacrimation, salivation, miosis, bradycardia, emesis, diarrhoea, etc.) or nicotinic receptors (muscle weakness, fasciculation, cramps, twitching). After about 24–96 h, intermediate syndrome, presenting as weakness of the proximal limb muscles, flexors of neck and respiratory muscles can occur[2]. OPIDN is a central peripheral distal axonopathy: peripheral distal axonopathy can predominantly present as a motor polyneuropathy, and central axonopathy can present with myelopathic features[3,4]. Delayed neuropathy is a rare complication of organophosphate poisoning and a result of inhibition of neuropathy target esterase instead of acetylcholinesterase. Among many thousands, only a small number of organophosphates can produce polyneuropathy with delayed onset. Organophosphate compounds that are known to produce distal axonal polyneuropathy in humans are triorthocresyl phosphate, leptophos, mipafox, trichlorofon, merphos, diptelay, systox,mecarbam, methamidophos, and Chlorpyrifos[5].

Hereby presenting a 20 year old male presented with features of organophosphate-induced delayed myelopathy & neuropathy following the ingestion of chlorpyrifos. However, there are only a few case reports of delayed neuropathy following OP insecticide exposure and all cases from India were secondary to Dichlorovas.

CASE REPORT

A 20-year-old previously healthy male farmer consumed a large amount of organophosphorus insecticide Chlorpyriphos(50%) 200 ml with suicidal intent around 8 weeks before being admitted to our hospital. Emergency management was done with gastric lavage, atropine infusion, and pralidoxime. The patient gradually improved and was discharged after 6 days of hospital stay. After 8 weeks of organophosphate ingestion, he again presented to our emergency department with gradual onset of bilateral weakness of lower extremities for 1 month. The patient initially noticed a dragging of his feet while walking. His symptoms progressed over a period of 1 month to the extent that he needed support both in standing from sitting position and walking on levelled ground. The patient found difficulty in removing and wearing slippers. His bladder and bowel habits were normal. At presentation, the patient was afebrile, oriented to time, place, and person. Higher mental functions, cranial nerve and sensory examinations were normal. There was no muscle atrophy, but spasticity was present in both the lower limbs with a power of

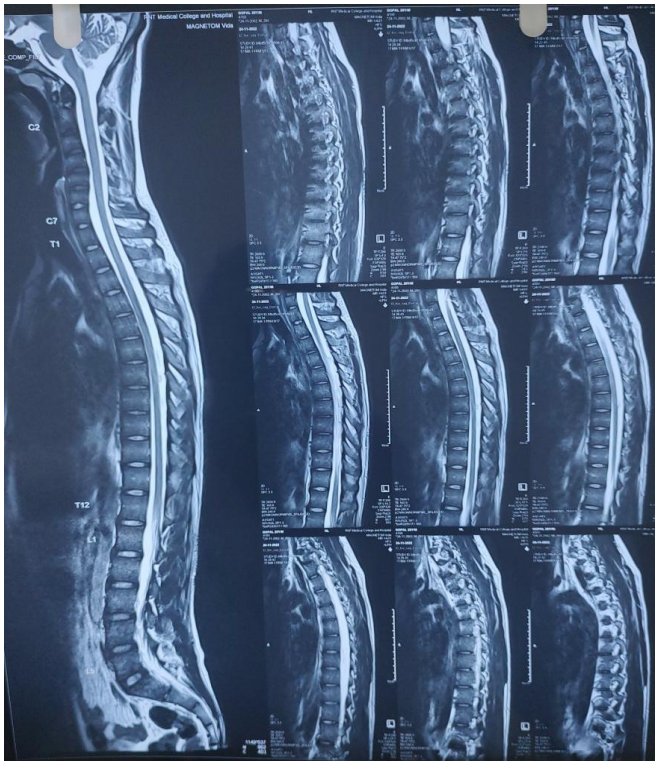
Medical Research Council(MRC)[6.] 5/5 in bilateral hips and 4/5 in bilateral knees and 1/5 ankles. Lower limb reflexes were brisk with bilateral extensor plantar response. Tone, power and reflexes were normal in both the upper limbs. Respiratory, cardiovascular and abdominal systems were essentially normal. Investigations of this patient revealed normal blood investigations(Table1). CSF analysis was normal. MRI spine was normal. Nerve conduction velocity studies showed features suggestive of Pure Motor Axonal Polyneuropathy. Sensory Nerve studies were within normal range. Compound muscle Action Potential(CMAP) amplitude reduced in Peroneal and Tibial nerves. The patient was treated with a 5-day course of 1 g/day of intravenous methylprednisolone, calcium and vitamin B1 supplements, and regular extensive physiotherapy of the lower limbs. The progressive symptoms of the patient improved during the course of treatment .

Table 1

LAB PARAMETER	REFERENCE	VALUE
TLC	3.5-10,000/µL	9100
Hb	11-17.5 g/dL	16.1
PLATELET COUNT	150-450 * 1000/µL	197
UREA	15-45 mg/dL	37
CREATININE	0.6-1.3 mg/dL	0.66
AST	15-37 U/L	44
ALT	35-65 U/L	65
Na	135-145 mmol/L	140
K	3.5-4.5 mmol/L	3.5
Calcium	8.5-10.1 mg/dL	10.0
VITAMIN B12	183-887 pg/mL	708
HIV		NR
HBsAg		NR
VDRL		NR

NCV STUDY FINDINGS

NERVE	LATENCY(msec)	AMPLITUDE(mV)
R PERONEAL	4.4	0.8
L PERONEAL	NR	NR
R TIBIAL	7.08	1.8
L TIBIAL	6.27	0.8



MRI SPINAL CORD – NORMAL

DISCUSSION

Organic insecticides are compounds that have been used globally for pest control for over 100 years. Due to their ready availability and easy accessibility, they have been frequently used as suicidal agents in India. Three different type of neurological presentations have been recognized following OP poisoning. Type I paralysis or cholinergic crisis occurs due to excessive stimulation of muscarinic receptors by Ach due to blockade of acetyl cholinesterase by an OP agent. Type II paralysis or intermediate syndrome is a distinct clinical entity having incidence of 8-49% and it usually appears 24 to 96 hours after poisoning. The pathogenesis is presumed to be dysfunction of neuromuscular junction caused by downregulation of presynaptic and postsynaptic Nicotinic receptors due to release of excessive Ach and Ca2+ respectively. The cardinal clinical features comprise muscular weakness affecting predominantly the proximal muscles and neck flexors. Recovery is rule in 5-18 days unless infections or cardiac arrhythmias complicate the course.[8,9] Type III paralysis or organophosphate induced delayed neuropathy (OPIDN) is a pure motor or predominantly motor axonal neuropathy characterized by wrist drop and foot drop with minimal or no sensory loss which occurs 7-28 days after exposure to an OP agent[6,7].OPIDN is an uncommon and rare cause of peripheral neuropathy. The cardinal feature is weakness which appears initially in distal leg muscles followed by small muscles of the hands and later it may extend proximally.

Clinical involvement of the corticospinal tracts and the dorsal columns become apparent when the peripheral neuropathy improves. Our patient also showed spinal cord involvement in the form of increased knee and ankle reflexes and extensor plantar . The prognosis in mild neuropathy is good but with severe neuropathy, partial recovery occurs in 6-12 months and usually left with deficits i.e., claw hand, foot drop, ataxia.[10] The pathogenesis of OPIDN 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.[6,7]The use of thiamine and high dose methylprednisolone has been shown to be beneficial in such patients. In India, there are very few case reports which documents dual neurotoxicity i.e. cholinergic crisis followed by delayed neuropathy and all these cases were following use of Dichlorovas. But our patient had delayed myelopathy and neuropathy after ingesting chlorpyrifos. Myelopathic cases reported in the literature have delayed onset spastic quadriparesis with bladder involvement, spastic quadriparesis with sensory motor neuropathy and pure motor spastic paraparesis. These presentations were after 18 months of exposure to the organophosphate. Our case also had a pure motor spastic paraparesis[3]. These features presented quite early (8 weeks) in our case. Corticosteroids have also been used as a neuroprotective drug in OPIDN[11]. A similar early presentation with dorsal cord atrophy at 2 months postexposure had been reported. It is therefore recommended that, every patient of OP poisoning should be followed up for at least one month.

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