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RESEARCH ARTICLE

ORGANOPHOSPHATE INDUCED DELAYED PERIPHERAL POLYNEUROPATHY: A RARE CASE REPORT

[1]Dr Mahesh Dave, [2]Dr. Sushma, N., [3]Dr. Anshul Doshi and [4]Dr. Shubhangi Gupta

[1] Senior Professor and Unit Head, Department of General Medicine, RNT Medical College, Udaipur, Rajasthan
[2,3,4] Junior resident (third year), Department of General Medicine, RNT Medical College, Udaipur, Rajasthan

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Corresponding author:
Dr. Sushma, N.,

ABSTRACT

Organophosphorus induced delayed peripheral polyneuropathy is rare clinical condition. The possible underlying pathophysiology is inhibition of neuropathy target esterase(NTE) resulting in both peripheral and central axonal neuropathy. Clinical manifestation includes sensory involvement, distal weakness with foot drop and hand drop. Even pyramidal tracts can be affected .we presenting the case of 15 year old female patient presented with bilateral symmetrical distal lower limb weakness (both foot drop and weakness of plantar flexion with atrophy of bilateral legs) without sensory symptoms developed after 2 weeks of consumption of Chlorpyriphos and cypermethrin (150 ml) .Nerve conduction study revealed distal pure motor axonal neuropathy of both tibial and peroneal nerve bilaterally.

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INTRODUCTION

Organophosphorus (OP) compounds cause most self poisoning in India because of easy availability and lack of stringent laws. Incidence of suicidal poisoning from organophosphorus compound ranges from 10.3% to 43.8%. (1) The pathophysiological basis of for the clinical manifestation of OP poisoning is inactivation of the enzyme, acetylcholinesterase at the peripheral muscarinic and nicotinic nerve terminals and junctions. Additionally these agents also inhibit the enzyme neuropathy target esterase (NTE) which is responsible for the delayed polyneuropathy in some of the patients. Clinical manifestation OP poisoning based on involvement of muscarinic (lacrimation, salivation, miosis, bradycardia, emesis, diarrhea, etc.) or nicotinic receptors (muscle weakness, fasciculation, cramps, twitching) that constitute acute cholinergic crisis. After about 24–96 h, intermediate syndrome, presenting as weakness of the proximal limb muscles, flexors of neck and respiratory muscles can occur.organophosphorous induced delayed polyneuropathy (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. These usually develop 7–20 days after exposure to an OP agent (2). We presenting the case of 15 year old female presented with OPIDN following ingestion of Chlorpyriphos and cypermethrin.

CASE REPORT

A 15 year old previously healthy female patient presented to MBGH and RNT medical college with history of consumption of a large amount of organophosphorus insecticide (Chlorpyriphos and cypermethrin 150 ml) with a suicidal purpose around 6 month before being admitted to this hospital. At the time she was hospitalized in district hospital for having frothing, nausea, vomiting, and altered sensorium and was managed vigorously by gastric lavage, inj Atropine, and pralidoxime .After recovery from cholinergic crisis patient was discharged 8 days after hospitalization. After remaining asymptomatic for 10 days she started noticing difficulty in lifting the both feet off the ground and difficulty in wearing and holding the chappal, insidious onset gradually progressive in nature over a period of 8 days she noticed thinning of both legs below knee not associated with twitching of muscle. Since than weakness and thinning was not progressed and it was bilaterally symmetrical. No weakness in getting up from squatting position .no weakness in bilateral upper limbs ,no history suggestive of sensory loss both in upper limb and lower limb There was no history of fever, back ache, joint pain, and swelling of joints prior to this event. There was no history of root pain, girdle like sensation or bowel and bladder involvement. On neurological examination the patient had normal higher mental function and cranial nerves functions were intact. Motor system examination revealed atrophy bilateral leg below knee (figure 1), and weakness of both lower

limbs, no fasciculations were present. Power of proximal lower limbs was 5/5(at hip and knee) distal was 2/5(at ankle both dorsiflexion and plantar flexion) however the power in upper limb was normal. Tone of the lower limb muscles was normal. Ankle jerk was present and knee jerk were exaggerated but biceps, triceps and supinator jerks were normal. Plantar reflex could be assessed because of weak dorsiflexion of foot and big toe. Abdominal and anal reflexes were present. Touch, temperature, pain and proprioception, vibration was preserved there was no sensory impairment. Gait of the patient was high stepping gait. Investigations of this patient revealed normal blood investigations

Renal function test: Urea:43mmol/L;Creatinine:0.7 mg//dL;Sodium:138mEq/L; Potassium:3.9mEq/L

Liver function test Total bilirubin: 16µmol/L; Direct bilirubin:4 µmol/L; Aspartate aminotransferase:10U/L;

Alanine aminotransferase: 18U/L; Alkaline phosphatase: 128U/L;Prothrombin time:12seconds
Vitamin B12:282 pg/mL; Folic acid:5.2ng/mL

Total and differential counts: WBC:6400 Cells/mm3; RBC:4.8Cells/mm3;Hemoglobin:16 gram% PCV:46.3%; MCV:95f/l; **MCH**:33pg; MCHC:34%; Platelets:262,000 Cells/mm3

Serology: Hiv: negative; HbsAg: negative; VDRL:non reactive. Nerve conduction study shows pure motor axonal neuropathy of both tibial and peroneal nerve bilaterally. Sensory conductions were normal.

MRI spine was normal.

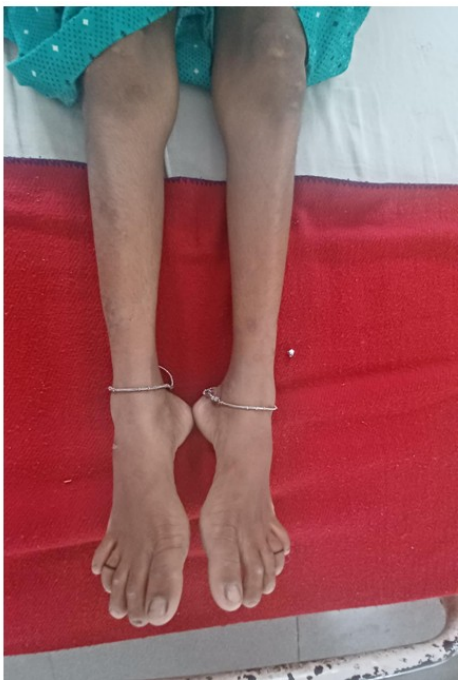

Figure 1. Shows atrophy of both legs with foot drop

DISCUSSION

Organophosphate compounds are the most common insecticides used worldwide. They are absorbed through transdermal, trans conjunctival, lungs on inhalation of dust or droplets, and gastrointestinal mucosa (3). The organophosphates associated with delayed neuropathy are Triorthocresyl phosphate, Chlorpyriphos, mipafox, fipronil, malathion, parathion, and matriphonate.(4) The most dangerous organophosphate ester is tri-o-cresyl phosphate.

Type 3 paralysis that is also called as OPIDN occurs between 10 to 3 weeks after poisoning .The exact pathogenesis of the OPIDN is yet to know, but it has been proposed that OPIDN is caused by the covalent inhibition of neuropathy target esterase (NTE).(5) NTE is located in endoplasmic reticulum (ER) of neural tissues such as the brain, spinal cord, and peripheral nerves, which deacylate phosphatidylcholine (PC) and lysophosphatidylcholine (LPC) to glycerophosphocholine (GPC), this disturbed metabolism of important membrane phospholipids, leads to axonopathy.(5) Abnormalities including axon transport and deranged axon membrane integrity resulting in neuropathy. OPIDN can be classified into four stages: Latent, Progressive, Stationary and Improvement stages (6). Latent period is characterized by a delay of 10 days to 3 weeks in developing neurological symptoms. In the progressive phase, signs and symptoms advance rapidly to present with motor sensory polyneuropathy. Sensory symptoms can include both positive and negative symptoms like cramping, tingling, burning pain in the calves, and glove and stocking type of sensory loss. Motor signs comprise foot drop and may progress to involve all four limbs with flaccid paralysis. During the stationary phase, neurological symptoms persist. As the patient enters the improvement phase, the sensory symptoms resolve prior to motor symptoms. The peripheral nervous system regenerates during this phase and hence spasticity with exaggerated reflexes occurs as a sign of unmasking of the lesion in the spinal cord. Our patient presented to us probably in stationary and improvement phase with atrophy of both legs below knee and weakness of both dorsiflexion(foot drop) and plantar flexion with preserved ankle reflex and exaggerated knee reflex without sensory involvement. This could represent an overlap between the progressive and the improvement phase. This also suggests a rapid peripheral nerve regeneration with unmasking of lesion in spinal cord. Luiz felipe et al(6) Showed the appearance of clinical features of corticospinal tract and dorsal column involvement with disappearance of peripheral neuropathy.

Our patient was treated with oral prednisolone and physiotherapy and asked to follow up after 15 days.

CONCLUSION

Distal pure motor axonal polyneuropathy is rare neurological manifestation of Organophosphorus poisoning .hence while evaluating for peripheral neuropathy asking for history of OP poisoning is essential and even follow up of patients OP poisoning for OPIDN should be kept in mind.

REFERENCES

1. Ahmed SM, Das B, Nadeem A, Samal RK. Survival pattern in patients with acute organophosphate poisoning on mechanical ventilation: A retrospective intensive care unit-based study in a tertiary care teaching hospital. Indian journal of anaesthesia. 2014 Jan 1;58(1):11-7.
2. Nayak P, Mallick AK, Mishra S, et al. Organophosphorusinduced toxic myeloneuropathy: series of three adolescent patients with short review. J Pediatr Neurosci 2019; 14(1): 42–45.

3. Berger AR. Toxic peripheral neuropathies. In: Samuels MA, Feske S, editors. Office Practice of Neurology. New York: Churchill Livinsgtone; 1996. P 538-9.
4. Araújo AQ. Organophosphorus polyneuropathy. Rev Bras Neurol Psiq 1997;1:75-7
5. Zhu L, Wang P, Sun YJ, Xu MY, Wu YJ. Disturbed phospholipid homeostasis in endoplasmic reticulum initiates tri-o-cresyl phosphate-induced delayed neurotoxicity. Sci Rep 2016;6:37574.

1. Abou-Donia MB. Organophosphorus ester-induced chronic neurotoxicity. Arch Environ Health 2003; 58(8): 484–497.
2. Luiz Felipe R Vasconcellos, Ana Claudia Leite, Osvaldo J M Nascimento. Organophosphate-induced delayed neuropthay. Arq. Neuro-Psiquiatr. Vol.60 no.4 Sao Paulo Dec.2002.

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