

Case Report

Unilateral vocal cord palsy in Organophosphorous poisoning - A case report

Sathyam Gopagani^{1*}, E.A. Ashok Kumar²

¹Post Graduate student, ²Professor

Department of General Medicine, M N R Medical College and Hospital, Medak, Telangana, India

*Corresponding author email: sathyamgopagani@gmail.com

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Abstract

We have described here a new variant of life-threatening organophosphate toxicity syndrome, which produces a brief vocal cord paralysis. A 18 years old male patient with recent ingestion of unknown quantity of Organophosphorous poison, presented to our hospital with acute symptoms of profuse sweating, increased salivation and lacrimation for the first 12 hours. Later on he developed quadriplegia-lower motor neuron (LMN) type - Acute polyneuropathy-myelinopathy. He developed hoarseness of voice and regurgitation of fluids with both 9 and 10 cranial nerve palsies. The patient had developed acute Organophosphorous poison toxicity initially, followed by Intermediate Syndrome after 24 hours of ingestion with vocal cord palsy.

Key words

Intermediate syndrome, Vocal cord palsy, Organophosphorous poisoning.

Introduction

There are 3 recognized types of toxicity syndromes in organophosphorous compound poisoning based on time of onset: 1) acute (instantaneous), 2) intermediate (slightly delayed, i.e., hours to days), and 3) delayed (weeks to months). Ingestion of large doses of insecticides leads to a cholinergic crisis and

possible death (acute-type syndrome). It is a medical emergency which may require intensive treatment. The crisis occurs as a result of accumulation of acetyl choline (Ach) at muscarinic and nicotinic sites. Exposures to lower doses may cause the intermediate-type or delayed-type toxic syndrome [1]. The intermediate-type syndrome is characterized by

slightly delayed polyneuropathy. It develops 12 to 96 hours post exposure and reflects a prolonged action of ACh on nicotinic receptors. Transient vocal cord paralysis has also been reported in association with the intermediate-type syndrome. Organophosphate-induced delayed polyneuropathy (OPIDP) is a rare toxicity resulting from exposure to certain Organophosphorous (OP) esters. It is characterized 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 delayed-type syndrome can produce muscle weakness lasting for a few months in some cases [2-5].

Case report

A 18 years old male patient was brought to the casualty of MNR Medical College, Sangareddy with history of consumption of unknown quantity of Organophosphorous poison of 12 hours duration. He was initially taken to a peripheral health centre where he was treated with 10 ampoules of Atropine in 500 ml normal saline and 2 g of Pralidoxime.

On arrival to the casualty, patient was conscious, well oriented with history of profuse sweating, salivation and vomiting before coming to the hospital. On examination, pupils were bilaterally equal, pin point and reacting to light. Pulse rate was 110/min, regular, normal volume. Blood pressure was 120/80 mm of Hg. Temperature was 98.4 F. Respiratory Rate was 30/min. SpO₂-94% at room air. Examination of cardiovascular and respiratory system was normal. Nervous System exam showed 9, 10 cranial nerve palsies +, pupils were bilateral constricted reacting to light. Tone was decreased in all the limbs. Power was 4/5 in all the limbs. He had proximal more than distal weakness. Deep tendon reflexes (DTR) were all absent. Plantars was not elicitable. No sensory loss was noted. Bladder and bowel were unaffected.

Treatment

Treatment was given as below.

- Gastric lavage was done with nasogastric tube 18 FG with 2 liters of normal saline until clear fluid aspirate was obtained.
- Oral secretions were aspirated.
- Inj. Atropine - 10 amp/ IV/ hourly.
- Inj. Pralidoxime - 500 mg/IV/tid.
- IV fluids 1 NS, 1 DNS.
- Inj. Ceftriaxone 1g/IV/bid.
- Inj. Metronidazole 500 mg/IV/tid.
- Inj. Ondansetron 4 mg/IV/sos.
- Inj. Optineuron 1 ampoule/ IV / daily

2nd day

Patient was conscious, coherent, well oriented to time and place. Patient was complaining of cough with regurgitation of fluids. On examination, temperature - 98.4°F, PR - 110/min, BP - 110/80 mm of Hg. CVS and RS were normal. Nervous System showed bilateral dilated pupils, reacting to light. Dysphagia was present for both solids and liquids. Tone was normal. Power was 4 in all limbs. Deep tendon reflexes were diminished. Plantars showed bilaterally flexor response. Atropine dose was decreased to 4 amp, IV, 4th hourly. ENT consultation was taken. Left vocal cord palsy was noted.

3rd day

Patient was complaining of cough. On examination, Pupils were bilaterally midsized and reacting to light. Dysphagia was present. Truncal muscle weakness was present. Power was 4/5 in all limbs. Deep tendon reflexes showed knee reflexes were absent bilaterally, ankle reflexes - 2+, biceps - 1+ on right side and absent on left side. Plantar reflex showed flexor response on both sides. Atropine was given 4 amp, IV, 6th hourly.

4th day

Pupils were bilateral normal size and reacting to light. Dysphagia was present. Truncal weakness was present. Power was 4-5 in right upper limb and 4+/5 in remaining 3 limbs. Deep tendon reflexes showed knee and biceps reflexes were absent, ankle reflexes were 2+. Plantars showed

bilateral flexor response. Atropine was given 4 amp, IV, bid. Ryle's tube was removed.

Investigations

- **Complete blood picture:** Hb - 15gm/dl, total WBC count - 14000 cells/mm³, DLC: N- 68%, E - 1%, B - 0%, L - 30%, M - 1%, Platelet count - 2.3lakhs/mm³.
- Blood urea - 41 mg/dl, serum creatinine - 0.9 mg/dl
- RBS - 69 mg/dl, Na -151meq/ltr, K - 4.8 meq/ltr, Cl - 115 meq/l.
- **ABG analysis:** P^H -7.38 , PCO₂-32.2 mm of Hg, PO₂- 69 mm of Hg , HCO₃⁻- 18.5 mmol/L, Na⁺ -154meq/liter, K⁺ - 4.0meq/liter , Cl⁻ - 120 meq/liter.

Discussion

Organophosphate (OP) poisoning is a frequent reason for admission to hospitals and Intensive Care Units in developing countries [6]. This is a case of Organophosphorous poisoning with usual cholinergic symptoms in the initial 24 hours, with gradual involvement of 9th and 10th cranial nerve from the 2nd day. This suggests the possibility of vocal cord palsy as a part of intermediate syndrome and the need for treating the patients with relatively high doses of atropine should be emphasized. The term intermediate syndrome was first coined by Senanayake from Sri Lanka in 1987 [4], but intermediate syndrome was first described by Wadia as type II paralysis in 1974 [5]. It was called as Intermediate because it appears after the acute cholinergic phase but before the expected onset of delayed neuropathy. The cardinal features of this syndrome are cranial nerve palsies, weakness of neck flexors, proximal muscle weakness and respiratory muscle paralysis which usually develops between 12 to 96 hours of ingestion of the poison.

Conclusion

Physicians should account for the neurotoxic effects of organophosphate poisoning during the first line management of exposed patients. Isolated bilateral/ unilateral vocal cord paralysis should be excluded as a cause, if dysphonia or respiratory distress occurs in patients with intermediate syndrome.

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