Case Report

Organophosphorous Induced Hemiparesis and Chorieform Movements: Rare Neurological Sequelae

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Abstract

Organophosphorous poisoning is one of the common medical emergency and is due to inhibition of the neurotransmitter acetyl cholinesterase. It manifests as either acute cholinergic crisis, intermediate syndrome of delayed polyneuropathy. But there are rare neurological sequelae of organophosphate poisoning. Here we present one such rare sequela in form of hemiparesis and chorieform movements.

Keywords: Organophosphate; Insecticide; Poisoning; Neurological manifestations

Introduction

Organophosphorous poisoning is caused by excessive acetylcholine activity at the post synaptic membrane in central nervous system (CNS), nicotinic receptors of the pre-ganglionic sympathetic and parasympathetic ganglia, and neuromuscular junction and muscaranic receptors of post-ganglionic parasympathetic fibers. Organophosphorous poisoning has three definitive neurological syndromes- the initial acute cholinergic crisis, intermediate syndrome and delayed organophosphate induced polyneuropathy. [1] Other rare presentations like extrapyramidal presentation, Parkinsonism, cognitive defects, amyotrophic lateral sclerosis and visual loss have been documented in the literature as well. But there has been no mention of hemiparesis in the literature so far. We present here a case of organophosphate poisoning induced hemiparesis and chorieform movements.

Case History

A 27 yr old male was admitted to the intensive care unit with alleged history of consumption of Quinolphos (diethyl organophosphate- acetyl cholinesterase inhibitor) of about 500 ml along with alcohol about 3-4 hrs prior to admission with suicidal intent. The relatives gave a history of frothing from mouth and unconsciousness after which he was rushed to the hospital. There was no history of vomiting, seizures, abdominal pain etc. On examination patient was unconscious (E1V1M3), afebrile, had miosis, was diaphoretic with a pulse(PR) of 62 beats/min, blood pressure(BP) of 110/ 80 mm of Hg and was having gasping respirations. The patient was immediately intubated with 8.5 no endotracheal tube and put on pressure support mechanical ventilation with FiO_2 of 100%. On further examination it was noted that he had bilateral crepitations on respiratory examination. There were fasciculation's present on his upper trunk and bilateral plantars were not illicitable. Rest of the examination was normal.

The patient was given a thorough gastric lavage followed by full body wash. Simultaneously specific treatment for Organophosphorous poisoning was started in the form Inj atropine 1.8 IV bolus and repeated (in dose of 3 mg) till PR was more than 80/min and the secretions had reduced. This was followed by an initial infusion of 3 mg (5 ml) per hour to be titrated according to pupil size, heart rate and secretions. He was also given injection Pralidoxime (PAM) 45 mg/kg IV bolus over 30 minutes followed by an infusion of 2 mg/kg/hr. His blood was sent for routine biochemistry along with blood for poison analysis and forensic sampling. His gastric lavage was also sending for same. All investigations were normal with random blood sugar (RBS) of 128 mg/dl, serum sodium 134 mEq/l, serum potassium 4.3 mEq/l, serum creatinine of 0.8 mg/dl, blood urea of 24 mg/dl. His arterial blood gas analysis (ABG) 2 hrs after intubation had a pH of 7.4, PaO_2 of 85, PCO_2 38 and HCO_3 24 mEq/l. His chest x-ray showed mild pulmonary congestion. Plasma cholinesterase levels could not be done due to unavailability of the tests.

After 4-5 hrs of admission patient started showing signs of improvement. He was conscious (E4VTM5), with oxygen saturation of 98%, afebrile, PR=128 /min and BP=120/80 mm of Hg. His pupils were dilated equally and chest was clear. His atropine was tapered. Over the next 16-20 hrs patient was weaned of ventilator as the patient had good respiratory efforts and was generating a tidal volume of > 5 ml/kg and pressure support of 5 cm of H_2O. Subsequently patient was maintaining oxygen saturation on ambient air. His blood and gastric lavage for poison analysis for positive for Quinolphos poison.

After 48 hrs of admission patient started spiking fever (38.4 degree Celsius) without chills and rigors and had one episode of generalized tonic clonic seizure lasting for about 2-3 minutes

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followed by loss of consciousness. This seizure was treated with injection diazepam 10 mg slow IV. His electrolytes and RBS were repeated and were normal. His blood leucocyte counts were marginally increased but the blood culture did not reveal any bacterial infection. The atropine dose was gradually tapered off and stopped. Subsequently after 2 days the patient started having chorieform movements of the right upper and lower limb along with weakness of right half of body. There was no history of subsequent fever, seizures or any change in consciousness. On examination patient was conscious, oriented, afebrile, PR=84/min, BP=120/80 mm of Hg, respiratory rate (RR)=14/min, bilateral pupils normal size and reacting to light. There was no neck rigidity. Neurological examination revealed power of 3/5 in right upper and lower limbs along with hypotonia; deep tendon reflexes were present and normal. Right Babinski reflex was extensor and left side was normal. Choreoathetoid movements of right upper and lower limbs and rest tremors were also noted. He did not have any memory deficit (Mini Mental Status Examination score=30). There were no cranial nerve palsies, no weakness of the respiratory or neck muscles and rest of the examination was normal. A computerized tomography (CT) scan of head was done which revealed non enhancing hypodense area in bilateral caudate nucleus and gangliocapsular region. He was treated conservatively and was started on tab Phenobarbitone to which he responded and there was decrease in his abnormal movements and the patient was discharged. Patient was followed up after 2 weeks in outpatient setting and he showed remarkable improvement in his power of right upper and lower limbs as well as decrease in the movements.

Discussion

Organophosphates inhibit the neurotransmitter acetyl cholinesterase, leading to symptoms related to the autonomous nervous system (abdominal cramps, nausea, diarrheoa, salivation, and miosis) and the central nervous system (dizziness, tremor, anxiety, and confusion). Symptoms usually occur within hours of exposure and typically disappear within days or weeks as new cholinesterase is synthesized. Certain organophosphates also inhibit another enzyme called neuropathy target esterase which is responsible for peripheral neuropathy (axonopathy) 10-14 days after exposure (intermediate syndrome). [2] There has also been documented evidence of other neurological syndromes with neurobehavioral changes and these have been termed as chronic organophosphate induced neuropsychiatric changes (COPIND). [1] The muscaranic and nicotinic side effects of organophosphate poisoning are well known and so are intermediate syndrome and COPIND. [3] There have been a few case reports of extrapyramidal manifestations due to organophosphate poisoning. The extrapyramidal features were dystonia, rest tremor, cog-wheel rigidity, and choreo-athetosis. The delay in onset of these signs, following poisoning, varied from 4 to 40 days, and they disappeared spontaneously in about 1 to 4 weeks in those who survived. [4] A couple of studies in literature have described organophosphate poisoning induced Parkinsonism, which in some cases has responded to treatment with amantadine. [5] But there are a few neurological symptoms such as chorea and psychiatric disturbances such as psychoses and depression which are relatively less known. [6] Few studies have also documented Alzheimer's disease, amyotrophic lateral sclerosis and cortical visual loss due to organophosphate poisoning. [7,8] But after a thorough literature search we could not find any reports of hemiparesis as a complication of organophosphate poisoning. So here we present probably a first ever case of hemiparesis and chorieform movements due to organophosphate poisoning.

Various mechanisms for these rare presentations have been postulated. The human extrapyramidal system is rich in cholinergic neurons and acetylcholinesterase (AChE). Inhibition of AChE by organophosphates, which has ready access to central neurons on account of its lipid solubility, was postulated as the mechanism underlying the extrapyramidal manifestations. [4]

Organophosphate poisoning cause's irreversible AChE inhibition resulting in raised acetylcholine (ACh) concentrations. The striatum contains large aspiny cholinergic interneurons which are likely to stimulate efferent enkephalin-containing GABA projections to the globus pallidus externus leading via increased glutaminergic excitation in the subthalamic nucleus to reduced cortical glutamate stimulation (indirect pathway of the corticostriatopallidothalamocortical circuit). Therefore, it can be speculated that reduced striatal AChE activity resulted in a decrease of cortical glutamate stimulation which clinically mimicked a dopamine deficiency syndrome presenting as Parkinsonism. [5] Organophosphate (OP)-induced brain damage is progressive damage to the brain, resulting from the cholinergic neuronal excitotoxicity and dysfunction induced by OP-induced irreversible AChE inhibition. This delayed secondary neuronal damage that occurs mainly in the cholinergic regions of the brain that contain dense accumulations of cholinergic neurons and the majority of cholinergic projection, might be largely responsible for persistent profound neuropsychiatric and neurological impairments. [9] In our case the chorieform movements after 4 days of admission due to organophosphate poisoning could also be due to inhibition of AChE at basal ganglia.

In an attempt to understand the various neurological manifestations of organophosphate toxicity there have been quite a few studies which have focused on imaging of the brain in these patients. In a study on the brain of paraoxon intoxicated rats, T2-weighted (Magnetic resonance imaging) MRI and Hydrogen-1 magnetic resonance spectroscopy (1H-MR-spectroscopy) were done before intoxication, 3 h, 24 h, and 8 days post intoxication. T2 prolongation mainly in the thalami and cortex was evident as early as 3 h after intoxication (4-6% increase in T2 values, $p<0.05$). This suggests that Organophosphates-induced brain damage is obvious as early as 3 h post intoxication. [10] Another study demonstrated abnormal regional cerebral blood flow in patients of organophosphate poisoning by using (99m)Tc-hexamethylpropylene amine oxime (HMPAO) brain single photon emission computed tomography (SPECT). Changes were found in the frontal, temporal, parietal lobes within the first week after poisoning. [11] Our patient showed revealed non

enhancing hypodense area in bilateral caudate nucleus and gangliocapsular region on CT scan which could be responsible for his hemiparesis.

Conclusion

This case highlights the importance of delayed and rare neurological complications of organophosphate poisoning. Neuroprotective strategies to limit or prevent this secondary neuronal damage are under development and may prevent long term disability due to organophosphates.

Conflict of Interest

The authors disclose that they have no conflicts of interest.

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