

Toxic Cardiac Manifestation in a Young Male with Organophosphate Poisoning with Breakthrough Seizures

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ABSTRACT

Organ phosphorus compounds are used as pesticides and represent a common cause of poisoning in developing countries including Pakistan due to their widespread availability. Toxicity due to these agents can affect many organs including the heart. Here, we report a case of acute organophosphorus poisoning in a patient, followed by acute myocardial infarction; documented by clinical features, electrocardiographic changes, and elevated cardiac enzymes. This case report will help a physician to look for acute myocardial injury in a patient with OP poisoning to reduce fatal complications and improve mortality, as such presentation with OP poisoning is rarely seen especially in a pediatric population.

INTRODUCTION

The readily available organophosphorus compounds are known to be acetylcholinesterase inhibitors and are becoming the common cause of acute pesticide poisonings along with other major health burdens in Pakistan. The World Health Organization (WHO) quantifies the OPCs related poisonings in the developing countries to have been doubled up in the last 10 years[1, 2]. Moreover, the death toll has soared up to 250,000 expires per year due to its poisoning in Southeast Asian countries like Pakistan, India, and Bangladesh[3, 4]. Insecticides have always been the leading accidental poisoning agents in under five age children due to their exploratory nature in South Asia[5].

These poisoning are majorly accidental in the pediatric population and suicidal in adults and have been reported to be 3 to 25% in low-middle-income countries due to lack of recognition and poor emergency medical services and about 1% in high economic strata.⁶ In literature review done in Pakistan declare around 40% out of 1900 cases dealt in the emergency department were of acute OP poisoning leading to increase mortality rate[6, 7]. It is a well-known fact that the organophosphate compounds act on the acetyl-cholinesterase enzyme by inhibiting its actions on the nervous system be it central or peripheral. The accumulated Acetylcholine then acts on both nicotinic and muscarinic receptors producing the respective effects. Non-specific OPC poisonings remain indistinguishable from various other commonly presenting illnesses in pediatric population allergic manifestations, viral infections, acute gastroenteritis or encephalopathy related to drug overdoses, liver failure, or kidney damages. Challenges exist in the presentations of these poisonings between children and adult that has been studied in multiple case series scrutinizing individual cases involving both age groups. In one case series review, it was mentioned that children presented more with CNS-related changes like seizures, decrease tone, lethargy, altered behavior, and coma than the adult ones[8]. What is known is that these compounds cause myocardial damage by hyperstimulation

of both the muscarinic and nicotinic receptors. Cardiac arrhythmia, pulmonary edema, hypertension, hypotension, and noncardiogenic pulmonary edema are life-threatening complications [9, 10]. There is limited data on acute myocardial infarction following OPCs intoxication (MI) and later leading to 1 degree or even complete heart block[11, 12]

The dilemma remains in our scenario due to scarcity of data revealing cardiac manifestations, so the frequency, mechanism, complications are unclear particularly in a pediatric population. Here we report a case of organophosphorus ingestion with atypical presentation of altered sensorium and seizures which later was complicated by myocardial damage with diffuse S-T segment depression and function.

CASE PRESENTATION

16 years old male, resident of rural area, known case of epilepsy was admitted to the emergency department of our hospital with a history of altered level of consciousness and tonic-clonic seizures along with frothing of mouth, and urinary incontinence for 5 hours. He was taking carbamazepine and risperidone for epilepsy and was compliant with medications. On arrival, a patient has blood pressures of 100/70 mmHg, a Heart rate of 122 beats/min, a Respiratory rate of 36 breaths/min and was not febrile, and was having saturations of around 92% on room air. On examination His Glasgow coma scale could not be assessed due to active seizures, tone was increased generally, his pupils were pinpoint, planters were downgoing bilaterally, s1+s2 with no added sound, upon chest auscultation normal vesicular breathing. For seizures he was given intravenous levetiracetam 1000mg stat dose with intravenous phenytoin, Seizures did not abort and the patient was intubated due to status epilepticus. His baseline workup sent which showed hemoglobin 11.7(11-15 g/dL) ,Total leukocyte count 17.1 (4.0-10.8 X 10⁹/L), Platelets 256 (154-433 X 10⁹) , Coagulation profile Prothrombin time with INR ration was 12.7/1.2(12-14 sec), His metabolic profile showed BUN 7(6-20mg/dL), Creatinine 0.6(0.5-1mg/dL), Sodium 135 (136-154mmol/L), Potassium 2.5(3.5-5.1mmol/L), Chloride 98(98-107 mmol/L), Bicarbonate 29.9 (20-31 mmol/L), Lactate 1.9.

ECG was done which showed diffuse ST depressions in anterior leads (Figure-1), troponin was checked which came out to be significantly raised 4.26 (<0.006) followed by 2nd troponin of 11.19. A bedside echocardiogram was done which showed reduced LV function of about 20-25% with severe global hypokinesia. Based on history and examination, a differential of breakthrough seizure and Organophosphorus poisoning was made. Cholinesterase levels were checked which were 584 (5400-13200U/L). He was started on Atropine infusion and Pralidoxime was given. Seizures were aborted with intravenous benzodiazepines. Pediatrics ICU, neurology, and cardiology teams were consulted and a Toxicology review was done, Child was subsequently managed on the lines of Organophosphate poisoning, Breakthrough seizures, and cardiogenic shock. 4 days post ICU, cholinesterase levels improved to 6046U/L, troponin levels started to decline. Repeat Echo has done which showed improved LV functions with an estimated EF of 48%. The child was extubated and monitored. Discharged after 7 days from admission.

DISCUSSION

A literature review highlighted atypical presentation in 80% of the patients with OP poisoning, and they were managed with wrong provisional impression¹⁹, In other case studies, 88% of patients did not have any history of OP ingestion.²⁰ Reviews of case series indicate that pediatric

organophosphate poisonings often manifest with hypotonia or mental status changes such as lethargy and coma, as well as seizures, the latter being relatively rare in adult OP poisoning. The overstimulation of nicotinic receptors manifests as anxiety, headache, seizures, difficulty in walking, decremental effects on respiratory and circulatory systems, tremor, and potentially coma. Along with this, the excess muscarinic drive produces miosis, wheezing, pulmonary edema, increased salivation, lacrimation, sweating, peristaltic activity, and increased frequency of micturition[13, 14].

Cardio toxic effects of organophosphate poisoning have been documented but rare in presentation, mechanism of cardiotoxicity by these compounds is still completely unknown although there are a few mechanisms postulated according to which there are three phases of cardiotoxicity sympathetic or parasympathetic overactivity, hypoxemia, acidosis, dyselectrolytes, and direct cardiotoxicity described by Ludomirsky et al[15]. The first phase is sympathetic overstimulation presenting as raising heart rate and blood pressure. The second phase later comprises of parasympathetic override characterizing lowering heart rate and blood pressure with life-threatening ST-T changes and arrhythmic tendencies. The prolonged last phase is associated with sudden death which can be a result of the prolonged QT interval and polymorphic ventricular tachycardia. Kathi et al[10]. also highlighted cardiac abnormalities in a set of total 37 patients with OPC poisoning that later manifested with severe cardiac complications in which around 62.5% of these 37 pts. The remaining 29.7% reflected ST-T changes secondary to myocardium ischemia. In the literature review, few case reports are available with variable EKG changes, to the best of our knowledge, no association has been seen of acute myocardial injury with OP poisoning.

Moreover, ECG changes found in two studies post OPC poisoning done by Paul UK, Bhattacharyya AK, and Yurumez et al., defined as prolonged Q-Tc interval changes in their patients with derangements of ST-segment, prolongation of P-R interval, inversion of T-wave along with, atrial and ventricular fibrillation[16, 17]. The current case can be an unusual presentation of acute myocardial infarction due to organophosphorus poisoning. Management mainly remains symptomatic and cardiac support. Careful cardiac monitoring with pulse oximetry and an electrocardiogram should be monitored suspiciously. Specific antidotes include atropine and pralidoxime have been utilized in its prompt management. Atropine blocks muscarinic receptors and decreases acetylcholine-related cholinergic crisis. Pralidoxime in comparison to atropine acts in regenerating acetylcholinesterase (AChE), which is rendered non-functional by the organophosphorus. Additionally, its anti-inflammatory effects via IL-10, it has been utilized in the treatment of complicated OP poisoning involving the kidneys, myocardium, liver, and lung parenchyma[18]

CONCLUSION

OP poisoning presents with typical signs and symptoms of cholinergic excess and rarely can cause acute myocardial toxicity. Its timely diagnosis along with management of this rare complication may confer a favorable prognosis in saving many precious lives from this treatable poisoning but if ignored, can result in a progressively worsening clinical outcome.

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