Acute Organophosphorus Poisoning Complicated by Acute Coronary Syndrome

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Abstract
We report a case of 30 year old alcoholic male admitted with vomiting, drowsiness, limb weakness and fasciculations after alleged history of consumption of 30 ml of chlorpyriphos insecticide. He had low serum cholinesterase levels. With standard treatment for organophosphorus poisoning (OPP), he improved gradually until day 5, when he developed neck and limb weakness and respiratory distress. This intermediate syndrome was treated with oximes, atropine and artificial ventilation. During treatment, his ECG showed fresh changes of ST elevation. High CPK & CPK-MB levels, septal hypokinesia on 2D echo suggested acute coronary syndrome. Coronary angiography was postponed due to his bedridden and obtunded status. The patient finally recovered fully by day 15 and was discharged. Acute coronary syndrome is a rare occurrence in OP poisoning. The present case thus emphasises the need for careful electrocardiographic and enzymatic monitoring of all patients of organophosphorus poisoning to prevent potential cardiac complication which can prove fatal.

Introduction
Organophosphorus insecticides are used extensively in horticulture and agriculture. Due to easy availability, poisoning with these agents has become very common in India.
Cardiac complications often accompany poisoning with these compounds, which may be serious and often fatal. These complications are potentially preventable, if recognised early and treated adequately. The extent, frequency and pathogenesis of the cardiac toxicity from these compounds has not been clearly defined. Current knowledge of cardiac manifestation consists of only limited studies and case reports. We report a case of acute organophosphorus poisoning complicated by acute coronary syndrome.

Case Report
A 30 yr old male, chronic alcoholic was admitted in medical ICU with alleged history of consumption of 30 ml of chlorpyriphos insecticide under influence of alcohol followed by vomiting and drowsiness since 16 hrs. On examination, P-128/min, BP-130/80 mm Hg, RR- 22/min, chest had bilateral crepitations, heart sounds were normal. On neurological examination patient was drowsy with grade 4 power in limbs with spontaneous fasciculations more in calves and thighs. ECG showed sinus tachycardia (Figure 1). Patient was kept under observation, watched for respiratory muscle weakness, and deterioration of neurological weakness. Patient was started on atropine injections, inj. glycopyrrolate 0.2 mg, antibiotics, antacids, lorazepam etc. He being a chronic alcoholic, was also put on dextrose drip and vitamin B₁ and B₁₂. Till day 4, patient showed a good recovery neurologically. Then patient again started developing weakness of limbs, difficulty in holding up neck and fasciculations on examination. There was respiratory distress also. At this stage, intermediate syndrome
was diagnosed and treatment started with ventilatory support; atropine and oximes were continued.

On day 5, patient became restless and his ECG tracing showed ST elevations lead I, V2, V3, and T wave inversions in inferior and chest leads (Figure 2). In addition, he also had chest discomfort. CPK and CPK-MB levels were high. Antianginals and LMWH was started. Patient had no prior cardiac illness nor any risks for cardiac disease. 2D Echo showed hypokinesia of anterior wall of left ventricle (LV), mildly dilated LV, EF 50%, suggestive of acute coronary syndrome. Laboratory parameters showed that Sr. cholinesterase on days 1, 3, 5, 6 were 1533, 1825, 2220, 5475 IU respectively (normal range-4500-12200 IU/L). Hb 18 gm%, TLC 17000, Sr. creatinine- 1.0 mg%, Electrolytes were normal. CPK was 218, CPK-MB was 72. Coronary angiography was postponed due to his bedridden and obtunded status. The patient finally recovered fully by day 15 and was discharged.

Discussion

The term Intermediate syndrome was coined because it appears between the Acute cholinergic phase and expected onset of delayed neuropathy.1,2 In intermediate syndrome (IMS), most commonly affected muscles are facial, extraocular, palatal, respiratory, proximal limb muscles. Clinical examination is most reliable means of identifying IMS.1

This delayed muscle weakness often without fasciculations or cholinergic features typically occurs 24-96 hrs after acute OP poisoning. Majority of reported cases after initial cholinergic phase improved with atropine and oxime therapy showed relapse in 48 hrs after presentation. Treatment of IMS is largely supportive with airway protection and ventilatory assistance. Pralidoxime or atropine therapy has no substantial data but nevertheless given/continued due to theories of inadequate dosing and persistent effects of OP at nicotinic receptors on motor nerves proximal to neuromuscular junction. Serial repetitive nerve stimulation studies have been most commonly used and are the most accessible for clinicians. Although electrophysiological features closely parallel clinical severity during progression of IMS, the same is not true during recovery. Electrophysiological changes sometimes improve long before the patient recovers normal strength and respiratory function in intermediate syndrome. The weakness and paralysis commonly resolve in 5 - 18 days.1

Concurrent alcohol and OP (organophosphorus) consumption which is common in alcoholics as in our patient can lead to higher amounts of consumption of OP thus leading to higher levels of OP in blood and high risk of deaths.3

In the first few hours of poisoning, cardiac events such as arrhythmias (atrial fibrillation, ventricular tachycardia), noncardiogenic pulmonary oedema, ECG changes like transient elevation of ST segment, prolonged Q-Tc interval, conduction defects, sinus bradycardia and sinus tachycardia, hypertension have been reported.4-6 The mechanism of cardiotoxicity in organophosphorus poisoning is uncertain. Both sympathetic and parasympathetic overactivity have been shown to cause myocardial damage. It has been postulated that parasympathetic overactivity plays a major role in coronary artery spasm and coronary artery spasm has been induced with acetylcholine. Few cases of transient myocardial infarction with raised cardiac enzymes have been reported in literature. Possible mechanisms include sympathetic and parasympathetic overactivity, hypoxaemia, acidosis, electrolyte derangements and a direct toxic effect of the OP compound on myocardium. Cardiac toxicity is caused by more than one mechanism as suggested by electrolyte changes as well as cardiac enzyme elevations4. However coronary angiography in such patients is not studied or reported in literature.
We have reported this case because ACS is a rare event in acute OP poisoning. Our patient had low cholinesterase levels on day 5, and he was still on atropine indicating that he was still in cholinergic crisis at the time of nicotinic manifestation in form of intermediate syndrome. So it was unlikely that the ACS was coincidental but was a complication of acute OP poisoning.

**References**

1. Richard Clark, Insecticides- Organophosphates and Carbamates, Goldfrank’s Toxicologic Emergencies, chapter 109, Pg 1503.