

Atypical manifestations of organophosphorus poisoning following subcutaneous injection of Dichlorvos with suicidal intention

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Abstract

Current case report describes a 37-year-old female patient who was admitted to the hospital following subcutaneous injection of Dichlorvos with an insulin syringe. The only peripheral cholinergic sign observed on admission was excessive salivation with bilateral pyramidal tract signs. Locally she had necrosis of skin and subcutaneous tissue with surrounding blisters. In the subsequent course of her illness, she developed respiratory arrest requiring ventilator support. She also had delayed extrapyramidal manifestations. Relevant literature is reviewed. Possibility of route-specific, delayed predominant central nervous system effect of Dichlorvos postulated.

Keywords: Central nervous system manifestations, respiratory arrest, subcutaneous dichlorvos

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Introduction

Dichlorvos (dimethyl-2, 2-dichloro vinyl phosphate) is an organophosphorus (OP) insecticide with moderate human toxicity, used for insect control in food storage areas, greenhouses, barns and control of insects on livestock. Parenteral Dichlorvos administration with suicidal intention is rarely reported in the literature. In one case report, extensive muscle necrosis was reported following intramuscular injection of Dichlorvos.^[1] In another case, following injection of Dichlorvos subcutaneously in both wrists, a young patient developed compartment syndrome requiring fasciotomy.^[2] In both cases authors did not observe any evidence of systemic toxicity. In the following report, I describe the case of a young female patient, who developed atypical signs of OP poisoning following subcutaneous injection of Dichlorvos.

Case Report

The present case report is about a 37-year-old, previously healthy female, who was admitted to our intensive care unit 18-h after alleged self-injection of 76% Dichlorvos (NUVOS) 3 ml in the upper left arm (3 times with a 1 ml Insulin syringe). Within an hour of the injections, she developed local pain and swelling. 8-10 h later she noticed excessive salivation. On examination, she had normal body temperature with heart rate of 90/min, blood pressure 110/70 mmHg, respiratory rate 14/min and pulse oximeter showing 100% saturation on room air. She had bruxism and bubbling of saliva from the mouth. Her pupils were bilateral 2 mm, reacting to light. No crackles were heard on chest auscultation. On the nervous system examination, she was jittery with slurred speech and normal cranial nerves. She had hypertonia, brisk tendon reflexes, bilateral sustained ankle clonus and bilateral extensor plantar reflex. Local examination revealed a blister and a black necrotizing lesion over the anterior aspect of the left arm [Figure 1]. Blood gas analysis showed normal values. She was rapidly atropinized with incremental boluses of atropine (total dose 12 mg) in next 30 min (targeting a dry mouth) followed by infusion of 1.8 mg/h.

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Figure 1: Local skin necrosis with surrounding blisters

Pralidoxime infusion was started at 500 mg/h following a loading dose of 1000 mg over 20 min. Intravenous Clindamycin (600 mg q 8 h) was administered empirically for presumed local wound infection. Debridement of the wound showed predominant involvement of the subcutaneous tissue plane with minimal necrosis of the superficial fibers of biceps brachii. Initial routine laboratory investigations were unremarkable except elevated total count ($16200/\text{mm}^3$) and mild respiratory alkalosis.

Her clinical course was complicated by respiratory arrest 9-h after hospital admission (27-h after the injection of Dichlorovos) leading to hypoxia and bradycardia. Arterial blood gas analysis post resuscitation showed-pH 7.28, PO_2 -36 mmHg, PCO_2 -54 mmHg, HCO_3 -22 mmHg. She was intubated and started on mechanical ventilation. Serum choline esterase (pseudo cholinesterase) level of the same day showed a very low value of <1 kU/L (normal range 3.93-10.8 kU/L). Subsequent 3 days course were unremarkable. She could be successfully weaned off from the ventilator on day 4.

On day 5, while on atropine infusion she was found to be delirious. She was also noticed to have extrapyramidal manifestations including rigidity and left sided gaze preference. Atropine infusion was stopped. In subsequent days, she showed gradual improvement in her neurological status. Extrapyramidal manifestations lasted for 3 days. Pralidoxime infusion was continued empirically for total 7 days. She and was finally discharged from the hospital on day 10.

Discussion

In this case, other than the unusual route of administration, a number of atypical manifestations

were found. This includes relatively delayed respiratory failure and central nervous system (CNS) manifestations with few muscarinic signs.

Two independent mechanisms are thought to be responsible for respiratory failure following OP insecticide poisoning – cholinergic CNS depression in the early stage (associated with other cholinergic features) and late progressive neuromuscular transmission defect.^[3] Second mechanism is part of the syndrome described as “Type II paralysis” by Wadia *et al.* or intermediate syndrome by Senanayake and Karalliedde.^[4,5] Our patient developed respiratory arrest 27-h after exposure to Dichlorovos. At that time she was conscious and adequately atropinized without any peripheral muscarinic sign. In her clinical course she never had neck or proximal muscle weakness, though the clinical findings were not correlated electrophysiologically.

Hyperactivation of cholinergic neurons is responsible for the CNS manifestations of OP insecticides.^[6] Usually CNS signs are manifested early in the course in the presence of peripheral muscarinic signs. In a large series published on neurological manifestations of OP compounds, 8 of 200 patients had pyramidal tract signs in the form of only hyper-reflexia manifesting within 24 h of exposure.^[4] All these patients were unconscious with florid cholinergic manifestations, including constricted pupils.^[4] In their original series of intermediate syndrome Senanayake and Karalliedde, observed hyperreflexia in one of their patients (with flexor plantar reflex) and even she had typical muscarinic signs.^[5] Extrapyramidal signs of OP poisoning are often transient and may be missed in a sick patient. In a Taiwanese series extrapyramidal signs were noticed within 4 days of intoxication.^[7] In our patient, CNS manifestations (both pyramidal tract and extrapyramidal) were delayed and were associated with relatively scanty peripheral muscarinic signs.

Possible explanation for these atypical manifestations in our patient could be route specific (in this case subcutaneous) effect of OP compound (Dichlorovos) causing isolated delayed CNS depression, with few peripheral cholinergic signs. Animal experiments have shown route specific (subcutaneous or intravenous) cardiovascular and neuromuscular effect of OP compounds in rats.^[8] This hypothesis may be tested by further experiments on large animals.

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