Multi-organ Dysfunction Syndrome with Dual Organophosphate Pesticides Poisoning

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ABSTRACT

Organophosphate (OP) pesticide self-poisoning is common in developing countries. Poisoning with dual OP compounds is rare. Multi-organ dysfunction after OP poisoning has a high mortality rate. We report the case of a 27-year-old man who developed multi-organ dysfunction syndrome with fatal outcome after intentional ingestion of 50:50 mixture of two OP compounds, dichlorvos and profenofos.

Key words: Dual organophosphate pesticides, multi-organ dysfunction syndrome, poisoning

INTRODUCTION

Organophosphate (OP) pesticide self-poisoning is common in developing countries. OP pesticides are inhibitors of both muscarinic and nicotinic acetylcholinesterase and affect the central nervous system (CNS). Multi-organ dysfunction is a rare event after OP poisoning, but associated with high mortality rate.[1] We report a case of 27-year-old man who consumed a 50:50 mixture of two OP compounds, dichlorvos and profenofos in an attempt of deliberate self-harm. He developed multi-organ dysfunction requiring intensive medical care and mechanical ventilation with fatal outcome.

CASE REPORT

A 27-year-old male was brought to the emergency department in an unconscious state. History from family members revealed that patient had ingested about 100 ml of preformed mixture (50:50) of dichlorvos and profenofos used as pesticide for agriculture purpose. He developed nausea, vomiting, abdominal cramps, difficulty in breathing and altered level of consciousness within 1 h of ingestion of poison. He had no significant past medical history or addiction. On arrival, approximately 6 h after ingestion, he was comatose with Glasgow coma scale of 3/15, pulse 94/min, respiratory rate 7/min, SpO_2 85% on room air and systolic blood pressure 80 mm of Hg. His pupils were small in size with sluggish reaction to light and there were no focal neurological signs. There were coarse crepitations on chest auscultation. Examination of the abdomen and cardiovascular system was unremarkable. Clinical severity assessment showed severe poisoning: Acute physiology and chronic health evaluation (APACHE II) score 26, Glasgow coma scale of three and poison severity scale of three. He was immediately intubated, gastric lavage was performed and atropine, pralidoxime, bicarbonate infusion was started. Patient was shifted to intensive care unit on inotropic support. His initial investigations showed white blood cells 31400/mm^3, hemoglobin 13.5 mg/dL, platelets 328000/mm^3, random blood sugar 120 mg/dL, serum creatinine 1.73 mg/dL, serum lipase 425 U/L, serum glutamic-pyruvic transaminase (SGPT) 350 IU/L, serum glutamic oxaloacetic transaminase (SGOT) 310 IU/L, serum cholinesterase level 329/L and deranged coagulation profile (international normalized ratio 2.2 and activated partial thromboplastin time 97). Urine

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analysis revealed pH 5.5, specific gravity 1.030 and proteinuria. Arterial blood gas analysis on FiO_2 28% was pH 7.03, PCO_2 55 mm Hg, PO_2 77 mm Hg and HCO_3 of 7 mEq/L. Chest X-ray showed bilateral infiltrates. Other investigations, including creatinine phosphokinase, serum electrolytes were within normal limits. Electrocardiogram and computed tomography scan head were unremarkable. Hepatitis B surface antigen, hepatitis C virus and human immunodeficiency virus serology was negative.

During 48 h after admission, his condition remained critical requiring inotropes, hemodialysis and ventilatory support. Arterial blood gas analysis showed persistent metabolic acidosis and hypoxia pH 7.14, PCO_2 44, PO_2 79, HCO_3 13, PO_2/FIO_2 0.80. Creatinine phosphokinase increased to 10504 U/L. SGPT and SGOT peaked at 11320 IU/L and 15800 IU/L.

Despite intensive resuscitation, the patient expired due to cardiac arrest 48 h after hospitalization.

DISCUSSION

World-wide, an estimated 3,000,000 people are exposed to OP or carbamate agents each year, with up to 300,000 fatalities. It leads to mortality in 10-50% of cases according to different studies.[2] Toxicity generally results from accidental or intentional ingestion of or exposure to agricultural pesticides.[3] Fatalities from acute OP agent poisoning generally result from respiratory failure due to a combination of depression of the CNS respiratory center, neuromuscular weakness, excessive respiratory secretions and bronchoconstriction. Fatalities also occur due to cardiovascular collapse; although the mechanism of this dysfunction is not completely understood, inappropriate vasodilation may play a role.[4] Age, amount ingested, APACHE II score, initial cholinesterase level and respiratory failure requiring mechanical ventilation are significantly associated with a poor outcome. Pre-treatment metabolic and respiratory acidosis directly correlates between the severity of poisoning and mortality.[5]

Studies of significant human toxicity due to dichlorvos and profenofos have been reported in the medical literature. In a retrospective study of 68 patients with acute OP poisoning, Kang *et al.* analyzed patient survival according to initial parameters, including the initial APACHE II score, serum cholinesterase level and hemoperfusion and evaluated the mortality according to OP types. The APACHE II score was a significant predictor of mortality. The mortality was 0% for 21 patients with dichlorvos poisoning. The mean patient APACHE II score was 5.52 ± 5.01 in this group. However, other OPs showed different mortality. They suggested that different OP have different toxicities.[6]

In a prospective cohort of patients with acute profenofos or prothiofos self-poisoning, Eddleston *et al.* analyzed clinical course and responsiveness to therapy of people poisoned with two S-alkyl OP insecticides – profenofos and prothiofos. In this study, of the 95 patients poisoned with profenofos and 12 with prothiofos, 11 patients poisoned with profenofos and one prothiofos patient died. They suggested that compared with other commonly used OP insecticides, profenofos and prothiofos are of moderately severe toxicity, causing relatively delayed respiratory failure and death. There was no apparent response to oxime therapy.[7] Another prospective study reported by Eddleston *et al*. analyzed 802 patients with chlorpyrifos, dimethoate or fenthion self-poisoning. They suggested that OP insecticide poisoning is not a single entity, with substantial variability in the clinical course, response to oximes and outcome.[8] Specific treatment advice for particular OP insecticides is not supplied, despite wide variation in animal toxicity, fat solubility, metabolism, selectivity for acetylcholinesterase over other serine esterases, side groups attached to the phosphate and speed of ageing (loss of an alkyl side chain that prevents reactivation by oximes), that might affect poisoning severity and response to treatment.[9,10]

Poisoning with a mixture of two OP pesticides is rare. To the best of our knowledge, there are no cases in the published literature on poisoning with a mixture of two OP pesticides. In our patient, amount ingested, high APACHE II score, metabolic acidosis and respiratory failure predicted the outcome.

CONCLUSION

Poisoning with a mixture of two OP pesticide may result in severe poisoning with multi-organ dysfunction syndrome and fatal outcome due to variable clinical course and response to oximes with the current treatment protocol.

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