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Intravenous organophosphate (dichlorvos) injection and the prolonged effect of toxin

Büyükcam, Fatih[a]; Cebeci, Zübeyir[b]; Zengin, Yilmaz[a]; Kaya, Ural[a]

Author Information

[a]Departments of Emergency Medicine

[b]Anesthesiology and Reanimation, Diskapi Yildirim Beyazit Training and Education Hospital, Ankara, Turkey

Correspondence to Fatih Büyükcam, MD, Department of Emergency Medicine, Diskapi Yildirim Beyazit Training and Education Hospital, Irfan Bastug Street, 06110 Ankara, Turkey Tel: +903123432421; fax: +903123186690; e-mail: fatihbuyukcam@gmail.com

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Introduction

Organophosphorous compounds (OPCs) are among the most important causes of poisoning in Turkey, as in many developing countries [1]. The mode of exposure to insecticides varies, including dermal, gastrointestinal, inhalational, and intravenous routes. Earlier few cases of intravenous exposure were reported [1]. Herein, we report a case of a patient who injected OPC intravenously, notable for early and late manifestations of OPC exposure.

Case report

A 19-year-old male complaining of nausea and abdominal pain for 1 h was admitted to the emergency department Diskapi Yildirim Beyazit Training and Education Hospital, Turkey. He gave a history of injection of nearly 1 ml (550 mg-9.48 mg/kg) of an insecticide {2,2-dichlorovinyl dimethyl phosphate [dichlorvos, Festline-DDVP (Dogal, Istanbul, Turkey)], concentrated emulsion=550 g/l} injected intravenously through cubital vein 2 h earlier. He had no history of chronic medical disease or long-standing medication. On admission, physical examination included body weight of 58 kg, blood pressure of 120/70 mmHg, and heart rate of 56 beats/min. The Glasgow Coma Scale (GCS) was 15 with isocoric-miotic pupils, normal light reflex, and normal skeletal reflexes. On auscultation of his chest, normal vesicular breath sounds were heard without added sounds. Diffuse abdominal tenderness was present; there was no evidence of secretions. Plasma pseudocholinesterase level was 3073 IU (normal range: 3714-11 513 IU) on admission and 2346 IU after 12 h (Table 1). Other laboratory tests were normal. Initially, 1 mg of atropine was applied as a loading dose with further 1 mg boluses given for bradycardiac episodes at 2, 3, and 5 h. A bolus dose of 1 mg 2-hydroxyiminomethyl-1-methyl-pyridinium chloride (pralidoxime) was given and continued with 500 mg/h intravenously for 48 h. After 2 h of admission he became unconscious and the GCS was 7 (eye: 1,

motor: 4, verbal: 2). His blood pressure was 85/60 mmHg and heart rate was 47 beats/min. Three tonic clonic seizures, each less than 5 min duration were seen in the first 5 h of admission. During seizures, intravenous doses of diazepam (5 mg) were applied. At the seventh hour of admission the GCS was 15; his blood pressure and heart rate were normalized.

Days	Plasma pseudocholinesterase level (normal range: 3714–11 513 IU)
On admission	3073
At 12 h	2346
Third day (after 48 h of PAM therapy)	3778
Fourth day (miosis and blurred vision)	2534
Sixth day	3412
Eighth day	2513
Ninth day	3110
Tenth day	3232
Eleventh day	2970
Twelfth day	5279
Fourteenth day	3910

PAM, 2-hydroxyiminomethyl-1-methyl-pyridinium chloride.

Table 1 Patient's characteristics

Pralidoxime infusion continued for 48 h. During treatment period, the pseudocholinesterase level was 2346 IU

at the 12 h and 3778 IU at the 48 h. On the fourth day, he experienced blurred vision for 5 h with miotic pupils. There was a further reduction in his pseudocholinesterase level to 2534 IU. Pralidoxime infusion was started again at 500 mg/h and was continued for 7 days and the level of pseudocholinesterase was checked daily. On the eleventh day of admission his plasma pseudocholinesterase level was 5279 IU, and the pralidoxime infusion was stopped. The pseudocholinesterase levels were found to be in normal ranges for 2 days without further clinical event, and therefore he was discharged. He was reviewed on a week post discharge and no abnormalities were seen.

Discussion

OPCs inhibit both cholinesterase and pseudocholinesterase activities irreversibly resulting in the accumulation of acetylcholine at synapses, causing overstimulation of both central and peripheral nervous systems [1]. Nicotinic manifestations of OPCs include increased or decreased muscle power and skeletal muscle fasciculations; muscarinic manifestations include excessive salivation, miosis, and diarrhea. The most frequent reported signs are miosis, vomiting, hypersalivation, respiratory distress, abdominal pain, and depressed level of consciousness and muscle fasciculation [2]. As respiratory failure is the major reason for mortality, careful monitoring, appropriate management, and early recognition of this complication may decrease the mortality rate among these patients [1]. Treatment is primarily supportive and includes decontamination, protection of airways and oxygen support, usage of a muscarinic antagonist, fluids and an acetylcholinesterase reactivator [3].

In the literature, few cases have been reported as intravenous or intramuscular injection of OPCs. Reported effects of intravenous or intramuscular injection are respiratory difficulty, thrombophlebitis, cellulitis, necrosis, and vasospasms [4]. In this study, we report the possible cardiac and neurologic effects of intravenous OPC administration. On the fourth day of follow-up we saw the long-term effect of the poison; ocular symptoms coupled with a repeat reduction in pseudocholinesterase levels. The severity of symptoms depends on the amount of the insecticide injected but even 1 ml of OPC could cause gastrointestinal, cardiac, and late neurological symptoms as reported in this study.

OPC-induced intermediate syndrome usually has an acute onset within 24–96 h after the antidote administration and cholinergic effect has worn off. At least 5–18 days are usually required for resolution of symptoms. Prolonged suppression of the enzyme, acetylcholinesterase, is observed in this syndrome. It usually occurs without fasciculations or other cholinergic manifestations, and the muscles innervated by motor cranial nerves, neck flexors, proximal limb muscles, or respiratory muscles are predominantly affected [5]. The delayed effects in our case may be a result of intermediate syndrome.

In conclusion, in case of intravenous exposure of OPCs, frequent neurologic examinations should be made to evaluate the neuromuscular effect. Clinicians must consider all the possible early and late effects, and patients must be hospitalized until the pseudocholinesterase levels normalize. Delayed toxicity potential after cessation of pralidoxime should be kept in mind.

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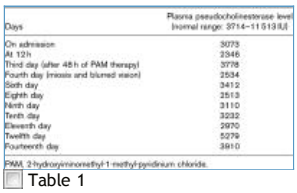

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Table 1

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