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Case Report: Severe Accidental Organophosphorus Poisoning in Two Children

Alireza Aminisefat[1], Khadijeh Saravani[2*], Mahdieh Saravani[3]

1. Student Research Committee, Zabol University of Medical Sciences, Zabol, Iran.
2. Department of Medical, Assistant Professor of Forensic Medicine and Toxicology, Zabol University of Medical Sciences, Zabol, Iran.
3. Department of Pediatric, Children and Adolescent Health Research center, Zahedan University of Medical Sciences, Khash, Iran.

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ABSTRACT

Background: Organophosphorus (OP) compounds have intense anticholinesterase activity and are commonly used as insecticides and pesticides. OP poisoning is a global health problem and poses a threat to human health.

Case Report: Here we report two sisters, aged 3 and 4 years, who had inadvertently eaten agricultural pesticides. These patients had a decreased level of consciousness, nausea, and vomiting. We do not have pralidoxime in this center and the adjacent center, so we used atropine as the primary drug in treating these patients. However, after 4 days, the 3-year-old patient felt a weakness in upper and lower limb movements.

Conclusion: In the treatment of OP poisoning, it is necessary to pay special attention to delayed complications such as intermediate syndrome.

1. Introduction

Organophosphorus (OP) compounds have vigorous anticholinesterase activity and are commonly used as insecticides and pesticides [1]. OP poisoning is a global health problem due to occupational, accidental, or intentional exposure (to commit suicide), especially in developing countries, and poses a threat to human health [2]. According to the previous studies, OP poisoning can kill up to 200000 people per year with 10.20% mortality [3]. These compounds are absorbed from the oral, respiratory, and transdermal pathways and expose people to poisoning [4]. In acute OP poisoning, the most common causes of death are usually respiratory failure and cardiac arrest, which is caused by its cholinergic effects [5]. In this case study, we reported a 3-year-old patient who developed weakness in upper and lower limb movements without respiratory failure and loss of consciousness after being poisoned with OP compounds and her 4-year-old sister, who just lost consciousness.

2. Case presentation

The patients were two sisters, aged 3 and 4, brought to the emergency department with nausea and vomiting. They had inadvertently eaten agricultural pesticides. The poisoning occurred 3 hours before the patients were visited in the hospital. The poison used was chlor-

*** Corresponding Author:**
Khadijeh Saravani, MD.
Address: *Department of Medical, Assistant Professor of Forensic Medicine and Toxicology, Zabol University of Medical Sciences, Zabol, Iran.*
Tel: *+98 (543) 2225402*
E-mail: *dr.kh.saravani93@gmail.com*

pyrifos, which was brought to the hospital the next day by their family.

At the time of presentation, they were well oriented and cooperative, with a Glasgow Coma Score (GCS) of 15. Both had tachycardia, and their other vital signs were stable (BP: 100/60 and 100/65 mm Hg, respiratory rate: 22 and 20 breath per minute, the temperature was 37.5°C and 37.4°C, SPO2: 98% and 99% on room air for 3-year-old and 4-year-old sisters, respectively). They had a runny nose but no runny eyes or mouth. There was no diarrhea or fasciculation, and examination of the pupils and lungs was normal. The results of the other tests were generally not significant.

After 5 hours from the onset of symptoms, the 3-year-old patient experienced a decrease in the level of consciousness. The lab results showed leukocytosis of 24.3 x 109 /l (normal range 4 to 10 x 103 /mm3), normal liver function, renal function, and serum levels of sodium and potassium. Other laboratory results were within normal limits. No conduction abnormalities were observed on an electrocardiogram. Arterial blood gas analysis showed a pH of 7.37, PO2 of 30.9 mm Hg and PCO2 of 36.4 mm Hg, and SPO2 of 58.4%. Her brain CT and chest x-ray were normal (Figure 1).

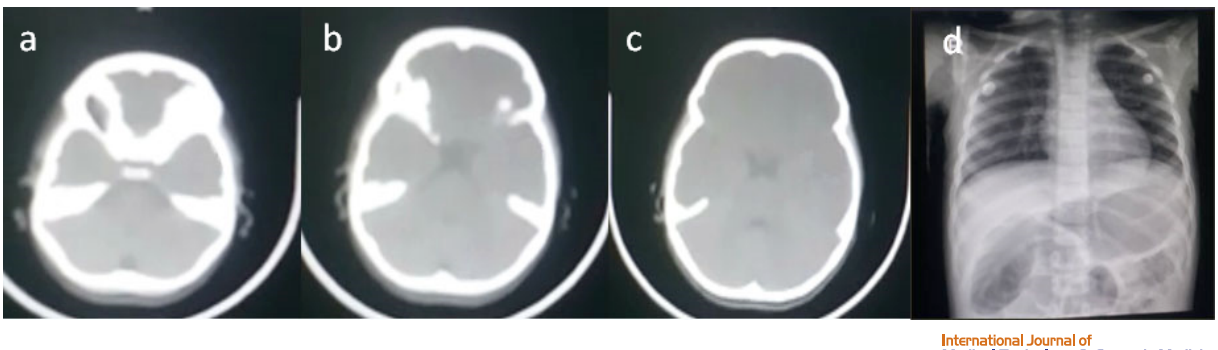
Shortly afterward, the 4-year-old patient lost her consciousness. The lab results showed leukocytosis of 14.2 x 109 /l (normal range 4 to 10 x 103 /mm3), and other laboratory results were within normal limits. Arterial blood gas analysis showed a pH of 7.42, PO2 of 42.6 mm Hg, PCO2 of 29.7 mm Hg, and SPO2 of 80.2%. Their serum and blood cell acetylcholinesterase (AChE) levels were not assayed due to the test's unavailability at our facility.

The patients regained consciousness after receiving 0.5 mg atropine. After 5 minutes, they lost consciousness and received 1 mg atropine again. By reducing their consciousness level, they received 2 mg, 4 mg, and then 4 mg atropine in 5 minutes intervals until they got fully conscious. To continue treatment, they received continuous drip of 1 mg, 0.5 mg, and then 0.25 mg in 12 hours intervals, and then atropine was discontinued. Twenty-four hours after stopping atropine, high-fat foods, preferably milk, were started for the patients. After 24 hours of eating high-fat foods, the 4-year-old girl general condition improved, and her test results were normal. So she was discharged from the hospital in good general condition. But the 3-year-old girl had a weakness in the movement of the upper and lower limbs. And this paralysis and weakness in limbs movement continued for 4 days after discontinuation of atropine.

3. Discussion

OP poisoning is a concern, especially in rural areas due to the availability of insecticides and pesticides. OPs compounds cause an irreversible inhibition of the esterase enzymes, especially AChE. This enzyme hydrolyzes the neurotransmitter acetylcholine (ACh) in synapses and red blood cell membranes [6]. This defect causes the accumulation of ACh and overstimulation of its receptors in synapses of the autonomic nervous system, CNS, and neuromuscular junctions [7]. Thus the symptoms of this poisoning are muscarinic and nicotinic side effects. The muscarinic symptoms include myosis, lacrimation, excessive salivation, bradycardia, diarrhea, and wheezing. The nicotinic symptoms include hypertension, fasciculation, and tachycardia [8]. However, patients can experience central effects such as fatigue, dizziness, and in severe cases, loss of consciousness, paroxysmal convulsion, respiratory failure, and even death [9] (Table 1).

In this study, on the fourth day after admission to the hospital, the 3-year-old patient experienced weakness in the movements of the upper and lower limbs known as intermediate syndrome (IMS) complications. IMS is



International Journal of
Medical Toxicology & Forensic Medicine

Figure 1. Radiographic imaging of the 3-year-old patient. (a-c) Brain CT and (d) Chest x-ray findings were normal.

Table 1. Symptoms and signs of organophosphorus poisoning (acute cholinergic phase of OP poisoning)

Central Receptors	Nicotinic Receptors	Muscarinic Receptors
Dizziness	Hypertension	Hypotension
Loss of consciousness	Tachycardia	Bradycardia
Paroxysmal convulsion	Weakness	Myosis
Coma	Fasciculation	Excessive salivation
Respiratory failure		Bronchospasm
Tremors		Cough
Absent reflexes		Nausea/vomiting
Anxiety		Wheezing
Ataxia		Lacrimation
		Bronchorrhoea
		Rhinorrhea

International Journal of
Medical Toxicology & Forensic Medicine

a complication of poisoning with OP compounds. It is usually seen 2-4 days after the disappearance of the acute symptoms of the cholinergic phase of OP poisoning but before organophosphate-induced delayed polyneuropathy. IMS symptoms can manifest as weakness of the respiratory muscles such as the diaphragm, intercostal and cervical muscles and rarely the weakness of the proximal muscles of the limbs [10]. In this study, two sisters were poisoned with chlorpyrifos, which is a relatively low-toxic organophosphate, and the occurrence of the secondary IMS of chlorpyrifos is infrequent [11]. In IMS, recovery usually occurs within 4 to 18 days, and sensory capacities are generally not impaired [12].

There is still no consensus on the pathophysiology of IMS. Yet, some studies have suggested that the dynamic of AChE and desensitization of ACh receptors is the basis for the development of IMS [10]. Many studies have indicated that the nature of OP compounds, the severity of poisoning, and inadequate oxime therapy can play a role in creating IMS [13]. In this article, a 3-year-old patient developed weakness in limb movements, but her sister was discharged without any specific symptoms. Since the primary treatment for both was atropine, the severity of the poisoning seems to play a critical role in the development of IMS. However, considering IMS only cannot be convincing due to insufficient treatment with oxime.

The muscarinic receptor antagonist (atropine) and oxime AChE reactivators (pralidoxime) are used as the main drugs for the treatment of OP poisoning [14]. Studies have shown that in the treatment of OP poisoning, hemoperfusion and continuous injection of atropine increase the survival rate of patients and reduces the reactivation time of cholinesterase, thus reducing the incidence of complications [15]. We do not have pralidoxime in this center and the adjacent center, so we used atropine as the main drug in treating these patients.

4. Conclusion

OP poisoning manifests with an acute cholinergic syndrome, IMS, and delayed polyneuropathy, which IMS and delayed neuropathy do not occur acutely and are observed with a delay. Therefore, it is necessary to pay special attention to these complications in the poisoning with OP compounds. We do not have pralidoxime in this center, and we cannot use it in OP poisoning. However, it is not clear how much pralidoxime can prevent IMS.

Ethical Considerations

Compliance with ethical guidelines

This study was approved by the Ethics Committee of Zabol University of Medical Sciences.

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Author's contributions

Data collection and Data analysis Khadijeh Saravani and Mahdieh Saravani; Writing the manuscript: Alireza Aminisefat; Review, editing, approve the final version of the manuscript: All authors.

Conflict of interest

The authors declared no conflict of interest.

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