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A Rare Case Of Inhalational Organophosphorus Toxicity

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Abstract:
Organophosphorus poisoning is commonly encountered in agricultural settings where the usage of pesticides is more. These compounds are also known for their potential as chemical warfare agents due to their toxic effects on the nervous system. This case report highlights a rare incident of acute inhalation organophosphorus poisoning in a 38-year-old male, who presented to the emergency department with symptoms of giddiness, nausea, vomiting and generalized fatigue. Early diagnosis and treatment are important in managing this potential life-threatening condition. The patient's clinical presentation, diagnostic findings and treatment are discussed here.

Keywords: *organophosphate poisoning, acetylcholinesterase, atropine sulfate, bradycardia. Introduction:*

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The history of organophosphorus poisoning is closely related to advancement in chemical and agricultural fields. Following World War II, the use of organophosphorus (OP) compounds increased rapidly in agriculture. Parathion, malathion, and diazinon are commonly used insecticides globally. OP poisoning can occur through ingestion, dermal absorption and inhalation. These compounds inhibit the enzyme acetylcholinesterase, leading to accumulation of acetylcholine in the body. The symptoms of OP poisoning range from mild headache, dizziness to severe respiratory failure, convulsions and coma. This case report describes an acute incidence of OP poisoning due to inhalation exposure in a 38-year-old male worker at a cardamom estate.

I. Case Presentation:

A 38-year-old male presented to the emergency department with the complaints of giddiness, nausea, vomiting, and generalized fatigue. The onset of symptoms occurred 15 minutes after the patient had mixed pesticide powder in a closed room, leading to suspected inhalational organophosphate poisoning.

On examination, the patient has bradycardia with pulse rate of 48 bpm, bp-110/70 MMHG, Spo2-95%, diaphoretic, and exhibited normal pupil size. Despite the absence of other overt cholinergic symptoms, the clinical suspicion of organophosphate poisoning was strong due to the patient's occupational exposure and acute onset of symptoms. Initially treated with inj.atropine, inj.ondansetron and inj.pantoprazole. The patient was observed for 12 hours. His condition stabilized following atropine administration, and a bolus dose of 500ml of 0.9% of normal saline. Routine blood investigations were normal.The diagnosis of organophosphate poisoning was supported by a significant reduction in serum cholinesterase, consistent with the inhalation exposure.

Renal Function Test	
Serum Creatinine	0.9
Serum Electrolytes	
Sodium 137.18 Mmol/L	137.18 Mmol/L
Potassium 3.20 Mmol/L	3.20 Mmol/L
Blood Ph	7.42
Complete Blood Count	
Haemoglobin 15.8 G/Dl	15.8g/Dl

Total Count	9,500 Cells/Cu.Mm
Lymphocytes	42%
Neutrophil	50%
Eosinophils	05%
Monocytes	03%
Rbs	5.3 M/Cu.Mm
Pcv	46.4%
Mcv	86.1 Fl
Mch	29.3 Pg
Mchc	34.1 G/Dl
Platelet Count	2.1 Lakhs/Cumm

II. Discussion:

Organophosphorus (OP) compounds are widely utilized as pesticides, herbicides, and chemical warfare agents, particularly in the form of nerve gasses. Some of the most recognized organophosphorus compounds include malathion, parathion, fenthion, diazinon, dimethoate, chlorpyrifos, paraoxon, and soman. The incidence of poisoning from these substances tends to be significantly higher in males compared to females, with reported cases showing an 80% incidence in males and 20% in females, resulting in male to female ratio of 4:1.[1]

Exposure to organophosphorus (OP) compounds can result in a range of clinical manifestations, which are categorized into muscarinic and nicotinic symptoms due to their effects on different receptors in the body. Muscarinic symptoms include nausea, vomiting, abdominal cramps, diarrhea, oronasal froth, cyanosis, pulmonary edema, bradycardia, hypotension, miosis (pupil constriction), increased sweating, and urinary bladder incontinence. These symptoms reflect the overstimulation of the parasympathetic nervous system. In contrast, nicotinic symptoms, which arise from the stimulation of the sympathetic nervous system, include tachycardia, muscle fasciculations, paralysis, hypertension, altered consciousness, coma, and more severe conditions like intermediate syndrome and organophosphate-induced delayed polyneuropathy (OPIDPN). Acute cholinergic syndrome, often following the ingestion of organophosphate pesticides, can lead to these symptoms [1].

Delayed manifestations, including intermediate syndrome, delayed-onset coma, and extrapyramidal symptoms often arise within a few days of exposure, reflecting a prolonged cholinergic crisis, whereas OPIDP typically emerges much later, around 2-3 weeks post-exposure, and can extend up to 4 weeks. This distinction underscores the variable nature of OP poisoning, where early symptoms are more acute and directly linked to cholinergic excess, while later symptoms are the result of delayed and progressive neurological damage, necessitating different clinical management approaches [2]

The route of exposure to organophosphorus (OP) compounds is a critical factor influencing the rapidity and severity of symptom onset. Common routes of exposure include inhalation, dermal (skin) contact, and ingestion. Among these, inhalational exposure is particularly concerning due to its capacity to trigger symptoms almost immediately, often within minutes, as the toxins are quickly absorbed through the respiratory system and enter the bloodstream. In contrast, dermal and ingestional exposures typically result in a slower onset of symptoms, as the absorption process through the skin or gastrointestinal tract is less rapid. This variability in symptom onset based on the exposure route is crucial for clinical assessment and underscores the need for prompt and appropriate treatment strategies tailored to the specific exposure circumstances.[2]

Organophosphate poisoning presents a significant health risk, especially in individuals with occupational exposure, as highlighted in this case report. The patient exhibited symptoms such as giddiness, nausea, vomiting, and bradycardia following inhalation exposure to pesticide powder. These symptoms are characteristic of organophosphate toxicity, where organophosphates inhibit acetylcholinesterase, leading to excessive accumulation of acetylcholine and overstimulation of cholinergic receptors [3,4]. This case aligns with the expected presentation of organophosphate poisoning, as described in the literature [5,6]

Treatment with atropine was essential in managing this patient's condition. Atropine, a muscarinic antagonist, is essential for counteracting the effects of excessive acetylcholine at muscarinic receptors [7,8]. This treatment is supported by evidence showing that early and adequate dosing of atropine significantly

reduces mortality and morbidity in organophosphate poisoning cases [9,10]. The use of normal saline to address potential dehydration and support cardiovascular function was also appropriate, aligning with standard management practices [11,12].

Despite the successful stabilization with atropine, the patient's presentation lacked some common symptoms of organophosphate poisoning, such as miosis. This variability in clinical presentation emphasizes the heterogeneous nature of organophosphate poisoning, where symptom expression can vary depending on factors like dose and exposure route [13,14]. This case highlights the importance of a high index of suspicion and a comprehensive evaluation in suspected cases of organophosphate poisoning, as symptom severity and manifestations can differ widely [15,16].

The diagnosis was confirmed by a significant reduction in serum cholinesterase levels, a key diagnostic marker for organophosphate poisoning. The correlation between serum cholinesterase levels and poisoning severity is well-documented, underscoring the importance of this biomarker in diagnosis and monitoring (15). The variability in clinical presentation, as seen in this case, further underscores the need for individualized treatment plans and ongoing research to refine management strategies [17].

In summary, this case emphasizes the critical importance of prompt recognition and management of organophosphate poisoning. The effective use of atropine and supportive care were crucial in stabilizing the patient. Continued research into treatment protocols and diagnostic tools is essential for improving outcomes and managing the diverse presentations of organophosphate poisoning.

III. Conclusion:

The variability in clinical presentation observed in cases of organophosphate poisoning highlights the necessity for individualized treatment plans tailored to each patient's unique symptoms and circumstances. This case reinforces the critical importance of prompt recognition and intervention in managing organophosphate toxicity, where the timely administration of atropine and comprehensive supportive care played a pivotal role in the patient's stabilization and recovery. However, the complexities and diverse manifestations of organophosphate poisoning underscore the ongoing need for research aimed at refining treatment protocols and enhancing diagnostic accuracy. Advances in these areas are vital to improving patient outcomes, ensuring that healthcare providers can effectively manage the broad spectrum of clinical scenarios associated with organophosphate exposure. Continued exploration into novel therapeutic strategies and diagnostic tools will be essential in addressing the challenges posed by this serious and often life-threatening condition.

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