Chlorpyrifos Ingestion in a Child Leading to Mechanical Ventilation: A Case Report from Oman and Review of the Literature

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Abstract

Organophosphate (OP) poisoning in children remains a serious health concern particularly in settings where exposure risk remains significant. We report a case of a 2-year-7-month-old Omani boy who developed acute cholinergic syndrome following accidental chlorpyrifos ingestion. He presented with hypersalivation, miosis, bradycardia, and altered mental status. Initial emergency management included airway maintenance, intravenous atropine, and pralidoxime. On the second day, the patient developed respiratory depression requiring intubation and mechanical ventilation, along with recurrent muscarinic symptoms necessitating frequent atropine administration. He also developed seizures, which were managed with clonazepam. He was admitted in the paediatric intensive care unit (PICU), during which he received frequent atropine dosing and supportive neurologic monitoring. After four days of PICU stay, the patient recovered fully without neurological sequelae, so extubated and moved to the paediatric ward for monitoring and rehabilitation, then discharged in stable condition on the ninth day. This case highlights the importance of early recognition, dynamic atropine dosing based on symptom recurrence, and seizure monitoring. It also contributes to the limited regional literature on pediatric OP toxicity. To our knowledge, this is the first reported pediatric case of acute chlorpyrifos poisoning in Oman. Emphasizing public awareness and safe pesticide storage remains essential in preventing similar incidents.

Keywords: Organophosphate Poisoning; Chlorpyrifos; Cholinergic Crisis; Atropine; Pralidoxime; Seizures.

Introduction

Paediatric poisoning remains a major public health issue in the Sultanate of Oman, especially among young children who are more vulnerable to toxic exposures. The majority of cases involve animal bites and stings, which account for 59.5% of reported incidents. Ingestions of harmful substances represent 38.5% of cases, including medications, food items, and household chemicals. Traditional remedies contribute to 7% of poisoning cases, highlighting the continued use of traditional medicine in the community.[1,2] Chlorpyrifos is a widely used organophosphate (OP) pesticide, often applied in agriculture due to its low cost and accessibility.[3] Despite regulatory restrictions in some countries, it remains available in many regions, especially where household and agricultural chemicals are easy to obtain.[3] Pesticide poisoning is a major global health concern, with the World Health Organization estimating around 150,000 deaths annually, most occurring in South Asia.[4] OP compounds are a major contributor to these deaths because of their high toxicity and widespread use.[3] In developing countries, OP poisoning is commonly linked to occupational exposure or intentional self-harm,[5] while in developed settings it still occurs through accidental exposure and can lead to severe outcomes, particularly in children.[6] OP exposure affects both genders and all age groups.[7] In children, skin absorption is a frequent route, whereas ingestion is more common in adults.[8,9] Although

the exact prevalence of OP poisoning in Oman is not well documented, chlorpyrifos remains a relevant cause of poisoning globally due to its widespread availability and high toxicity.

Chlorpyrifos and other OP compounds irreversibly inhibit acetylcholinesterase (AChE), causing acetylcholine accumulation at synapses and overstimulation of muscarinic and nicotinic receptors.[10] OP poisoning progresses through three clinical phases: an acute cholinergic phase within 30 minutes to 3 hours, an intermediate syndrome between 24 and 96 hours involving muscle paralysis, and a delayed polyneuropathy phase after 2 to 4 weeks, marked by distal weakness and motor deficits.[11]

Diagnosis of OP poisoning is mainly clinical, based on history of exposure and physical findings.[3-5] A trial dose of atropine (0.01–0.02 mg/kg) may support the diagnosis if symptoms improve. Red blood cell AChE levels are more reliable for assessing toxicity than plasma levels.[12] Initial management includes airway support, oxygen, intravenous fluids, and correction of electrolytes.[3,4,8] Decontamination involves removing contaminated clothing and washing the skin with soap and water.[4,6] Definitive treatment includes atropine and pralidoxime (2-PAM).[11] Atropine, a muscarinic receptor antagonist that counteracts excess acetylcholine, is started with an IV bolus of 0.05 mg/kg and adjusted based on clinical response. In contrast, pralidoxime, which reactivates acetylcholinesterase by cleaving its bond with the organophosphate, is dosed according to poisoning severity.[12] If treatment is delayed beyond 24 hours, AChE inhibition may become irreversible.[10] Early recognition and prompt management are essential for improving outcomes.[3,7,8] Here, we report a case of a young boy who ingested chlorpyrifos and developed acute symptoms consistent with OP poisoning.

Case Report

A 2-year and 7-month-old previously healthy boy weighing 12 kg was referred from a local health centre to our secondary hospital emergency department (ED) with a four-hour history of suspected ingestion of approximately 250 mL of chlorpyrifos (Dursban) insecticide. The ingestion was unwitnessed. Approximately one hour after ingestion, he developed abdominal pain followed by vomiting of thick, reddish fluid.

On examination, he was conscious, alert, and clinically well hydrated, with a temperature of 39°C. He had hypersalivation requiring suctioning. Respiratory examination revealed tachypnea and bilateral wheeze, although oxygen saturation was maintained on room air. He was tachycardic; the remainder of the cardiovascular examination was unremarkable. His pupils were 2 mm and reactive to light. Neurological and abdominal examinations were within normal limits.

Venous blood gas analysis showed a pH of 7.34, pCO_2 of 41 mmHg, pO_2 of 44 mmHg, bicarbonate of 21.7 mmol/L, base excess –3.5 mmol/L, and lactate of 2.1 mmol/L, consistent with mild metabolic acidosis. Complete blood count revealed neutrophilic leukocytosis, with a white blood cell count of $15.7 \times 10^3/\mu L$ and neutrophils of $7.77 \times 10^3/\mu L$. Serum potassium was 3.1 mmol/L, indicating hypokalemia. This was likely due to a combination of potassium loss from vomiting and a transcellular shift caused by the adrenergic response associated with chlorpyrifos toxicity. Renal and liver function tests, coagulation profile, and random blood glucose were within normal limits. The patient received initial emergency care with intravenous atropine (500 mcg) and pralidoxime (0.24 g). Red blood cell acetylcholinesterase (AChE) levels were not measured or monitored in this case due to limited availability of the test at our facility. Diagnosis and treatment decisions were guided by clinical findings. He was admitted to the pediatric intensive care unit (PICU), kept nil per os, and managed with intravenous fluids (0.9% saline at 44 mL/h), intravenous Augmentin (30 mg/kg/dose TID), and intravenous paracetamol as needed. A nasogastric tube and urinary catheter were placed for monitoring. Chest X-ray was unremarkable, and blood cultures remained sterile.

Within the first 24 hours of admission, the patient developed signs of recurrent cholinergic crisis, including tremors, miosis, bradycardia, and increased nasogastric secretions. Shortly after, he experienced a drop in consciousness, with eyes not opening to deep stimulation. As he was unable to maintain his airway, endotracheal intubation was performed. During the procedure, a strong pesticide odor was noted. He was started on synchronized intermittent mandatory ventilation (SIMV) with FiO_2 30%, peak inspiratory pressure (PIP) of 15 cmH_2O, and respiratory rate (RR) of 25 breaths per minute. Sedation was initiated with intravenous midazolam, fentanyl, and

cisatracurium, followed by continuous infusion. Post-intubation venous blood gas confirmed compensated respiratory acidosis. He received three additional doses of intravenous atropine (500 mcg each) and one dose of pralidoxime (0.24 g). Atropine was administered repeatedly based on symptom recurrence, in line with current practice, as there are no universally established guidelines for exact dosing intervals or total duration in pediatric organophosphate poisoning. He temporarily stabilized, with clear lungs and reactive pupils. Later the same day, he developed sudden respiratory deterioration characterized by wheezing, prolonged expiration, likely due to recurrence of cholinergic effects. These effects led to increased bronchial secretions and bronchospasm, causing lower airway narrowing and an obstructive respiratory pattern. He also had wide pulse pressure, fever (40°C), and respiratory acidosis on repeat blood gas. Ventilator settings were adjusted (FiO_2 40%, RR 30, PIP 16 cmH_2O). He received a STAT dose of atropine (500 mcg), was started on salbutamol nebulization every 4 hours and intravenous hydrocortisone every 6 hours, and antibiotics were escalated to intravenous ceftriaxone. A repeat septic workup was sent.

He subsequently developed upper limb myoclonic jerks, brownish nasogastric aspirate, and dark urine. Hemoglobin dropped from 11.5 to 9.8 g/dL. He remained hemodynamically stable and was given one unit of packed red blood cells and intravenous omeprazole. Brain CT was normal. An electroencephalogram (EEG) was not performed, as the myoclonic activity was non-progressive, resolved with treatment, and the patient showed no further neurological deterioration.

On day four of admission (day three of intubation), his respiratory status improved, with resolution of wheezing. Atropine was continued every 30 minutes, along with ongoing bronchodilator and steroid therapy. Neurological symptoms, including jerky upper limb movements and facial twitching, persisted but were non-progressive. A total of 300 mcg of atropine and 0.72 g of pralidoxime were administered during this period, and both atropine and pralidoxime were discontinued.

Shortly after, the patient developed a recurrence of bronchorrhea and wheezing, requiring five additional doses of atropine (600 mcg total). Symptoms improved, and enteral feeding was reintroduced. He was subsequently transferred to the pediatric high-dependency unit, remained clinically stable, and was gradually reinitiated on oral feeding. His mobility improved, and assisted ambulation was encouraged. Due to persistent upper limb twitching, oral clonazepam was started for symptomatic control. He was later able to walk independently and was discharged in stable condition with no neurological deficits. Clonazepam was continued as a discharge medication for a total duration of 15 days. The overall duration of illness from presentation to discharge was nine days.

At follow-up two weeks later, the child had returned to his baseline state. He was active, walking independently, and free of respiratory or neurological symptoms. Vital signs were normal, and systemic examination was unremarkable. Clonazepam was discontinued. The family was counselled on household chemical safety and instructed to seek immediate care if concerning symptoms recurred.

Discussion

Chlorpyrifos is a widely used OP pesticide and remains a common cause of accidental poisoning in children due to its accessibility in both household and agricultural settings.[3,13] Its toxic effects are mediated through irreversible inhibition of acetylcholinesterase, resulting in excessive accumulation of acetylcholine and overstimulation of muscarinic and nicotinic receptors.[10] In paediatric cases, ingestion is the predominant route of exposure, and early recognition is critical to prevent life-threatening cholinergic crises and neurological sequelae.[4,8,9]

Initial management in this case was guided by supportive measures, with emphasis on airway protection, hemodynamic stabilization, and seizure prevention. Given the risk of aspiration, gastric lavage was avoided. Atropine and pralidoxime remain the cornerstones of treatment.[11] Our patient achieved full atropinisation with 2000 mcg of atropine initially, followed by tapering doses. However, a recurrence of cholinergic symptoms necessitated an additional 3000 mcg, highlighting the dynamic nature of OP poisoning. Pralidoxime was administered concurrently after clinical diagnosis was confirmed. Although early diazepam use is supported in literature to reduce neurotoxicity,[12] our patient received midazolam-based sedation during ventilation and was discharged on clonazepam for provoked seizure activity. However, prophylactic antiseizure medication is not routinely

recommended in chlorpyrifos poisoning unless seizures occur. Treatment should be guided by clinical signs, with benzodiazepines reserved for patients who develop seizure activity. Caregiver education focused on home monitoring and early recognition of neurological deterioration.

The variability of OP toxicity outcomes in children has been well documented. Some cases resolve with supportive care, while others result in long-term deficits or death. A U.S. case series involving coumaphos ingestion reported two children requiring intensive care and dialysis; one developed hypoxic brain injury and needed prolonged rehabilitation.[14] Similarly, an Indian case described a 12-year-old with intermediate syndrome following fenthion ingestion, who recovered fully with PICU support.[4] In Oman, Al Amrani et al. reported a delayed-onset myelopathy in a 7-year-old boy two weeks after OP exposure, emphasizing the risk of late neurological complications despite initial clinical improvement.[15] These cases, summarized in Table 1, reflect the diverse spectrum of OP poisoning presentations and outcomes.

Table 1: Summary of published pediatric OP poisoning cases (2011–2024).

Country	Year	Age	Gender	OP Compound	Accidental or Intentional	Duration of Illness	ICU / Ventilation	Treatment	Outcome
India[4]	2024	19 months	Male	Unidentified OP	Accidental	19 days	Yes – 6 days ventilation	Atropine, pralidoxime	Full recovery
South Africa[16]	2024	15 months	Female	Methamidophos	Accidental	~10 days	Yes – CPAP support	Atropine	Full recovery
Oman[15]	2023	7 years	Male	Unidentified OP	Accidental	Delayed symptoms after 2 weeks	No (not reported)	Atropine, rehabilitation	Paraplegia due to delayed myelopathy
USA (CA)[17]	2022	7 years	Male	Coumaphos	Accidental	>90 days	Yes – 11 days ventilation	Atropine, pralidoxime, dialysis	Severe neurologic sequelae
USA (CA)[17]	2022	10 years	Female	Coumaphos	Accidental	16 days	Yes – 6 days ventilation	Atropine, pralidoxime, dialysis	Full recovery
USA (TX)[18]	2021	Infant	Male	Unspecified OP	Accidental	Few days	Yes – ventilation	Atropine, pralidoxime	Full recovery
East Africa[13]	2021	<5 years	Male	Unidentified OP	Accidental	24–48 hrs	Yes – CPR + oxygen	Atropine	Full recovery
India[19]	2014	12 years	Female	Fenthion	Intentional (suicidal)	18 days	Yes – ICU monitoring	Atropine, fluids	Full recovery
Malawi[20]	2011	12 days	Female	Unspecified OP	Accidental (secondary exposure)	Few days	Yes – NICU	Atropine	Full recovery

In contrast, our patient presented early following confirmed chlorpyrifos ingestion, received prompt antidotal therapy, and had no residual neurological deficits at discharge or follow-up. To our knowledge, this is the first documented paediatric case of acute chlorpyrifos poisoning in Oman. The case underscores the importance of identifying the specific compound, initiating atropinization without delay, and arranging appropriate follow-up to detect delayed neurotoxicity such as organophosphate-induced delayed polyneuropathy (OPIDP).[9] Ongoing vigilance is necessary even after apparent recovery, as symptom recurrence or late complications may develop days to weeks after exposure.[6,9,15]

Poisoning in children can be a sign of neglect or accidental harm from poor caregiver supervision. When this is suspected, it's important to assess the family's situation, including caregiver awareness of safety measures, mental health, and social stressors. If neglect is likely, especially in repeated or severe cases, child protection services should be involved. Supporting and educating caregivers can help prevent future incidents and ensure the child's safety.

Conclusion

This case highlights the need for secure storage of pesticides to prevent accidental pediatric exposure. Early recognition of organophosphate toxidrome and prompt initiation of atropine and pralidoxime are critical for favorable outcomes. Careful documentation of antidotal dosing facilitates individualized treatment adjustment. Structured post-discharge follow-up remains essential to monitor for delayed neurotoxicity and to support long-term recovery.

Disclosure

The authors declared no conflicts of interest. Written consent was obtained from the patient/kin of the patient.

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