Scholars Journal of Medical Case Reports
Abbreviated Key Title: Sch J Med Case Rep
ISSN 2347-9507 (Print) | ISSN 2347-6559 (Online)
Journal homepage: https://saspublishers.com

OPEN ACCESS

Anesthesia

Severe Acute Organophosphate Poisoning in a Child in a Rural Intensive Care Unit: Clinical, Diagnostic and Therapeutic Aspects

Ndiaye Saliou[1*], Samb Marième[2], Mbengue Mansour[3], Bah Mamadou Diawo[4]

[1]Anesthesia and Intensive Care Unit, Thierno Birahim Regional Hospital Ndao from Kaffrine
[2]Biology Laboratory, Thierno Birahim Regional Hospital Ndao from Kaffrine
[3]Nephrology and Dialysis Service Sheikh Anta Diop University, Senegal
[4]Anesthesia-Resuscitation Department, Cheikh Anta Diop University, Senegal

DOI: https://doi.org/10.36347/sjmcr.2025.v13i12.003 | **Received:** 09.10.2025 | **Accepted:** 03.12.2025 | **Published:** 05.12.2025

***Corresponding author:** Ndiaye Saliou
Anesthesia and Intensive Care Unit, Thierno Birahim Regional Hospital Ndao from Kaffrine

Abstract **Case Report**

Background: Organophosphates are commonly used in our environment, especially in agriculture as insecticides, and are responsible for high mortality rates in cases of severe acute poisoning, particularly in our context where resuscitation resources are insufficient. Organophosphate poisoning is most often accidental in children. It is rare but serious. The clinical presentation is dominated by a muscarinic, nicotinic, and central nervous system syndrome, requiring primarily symptomatic management in an intensive care setting. We report here a case of severe acute organophosphate poisoning in a child. ***Case Description***: A six-year-old child, not attending school and with no prior medical history, was admitted to the intensive care unit for the management of a sudden onset of altered consciousness associated with respiratory distress. Questioning of the father revealed a history of accidental ingestion of an unspecified quantity of an organophosphate insecticide in agricultural fields. Treatment was primarily symptomatic, combining resuscitation measures and atropine. ***Conclusion***: Organophosphate poisoning in children poses a problem in developing countries. Recognizing the variable clinical manifestations and providing early therapeutic intervention in the intensive care unit can lead to favorable outcomes, as demonstrated by our case.

Keywords: Poisoning, Child, Clinic, Treatment, Rural Resuscitation.

INTRODUCTION

Organophosphate (OP) compounds are widely used agricultural insecticides that pose a significant public health concern, particularly in developing countries. Accidental exposure, especially among children, remains frequent due to inadequate storage practices and their high toxicity. The pathophysiology of OP poisoning involves inhibition of acetylcholinesterase, leading to accumulation of acetylcholine at cholinergic synapses and resulting in characteristic muscarinic, nicotinic, and central nervous system manifestations. In pediatric populations, neurological symptoms such as altered consciousness, seizures, and coma are often predominant, making early diagnosis challenging. Rapid recognition and management are essential to prevent severe complications and mortality. This report describes a case of severe acute organophosphate poisoning in a six-year-old child, emphasizing the clinical presentation, therapeutic challenges, and favorable outcome following prompt symptomatic treatment despite the unavailability of specific antidotal therapy.

CASE DESCRIPTION

A six-year-old, homeschooled child with no significant past medical history was admitted to the intensive care unit for management of altered consciousness and respiratory distress. The symptoms had begun 36 hours prior to hospitalization with the sudden onset of gastrointestinal disturbances such as vomiting and abdominal pain. Upon admission, he presented with a coma (Glasgow Coma Scale score of 8) associated with episodes of generalized seizures and bilateral pinpoint pupils. A pleuropulmonary examination revealed tachypnea (30 breaths/min) with an oxygen saturation of 96% (15 liters/minute) on a high-concentration face mask, hypersalivation, and significant tracheobronchial obstruction. He was hemodynamically stable with a blood pressure of 95/55 mmHg and a heart rate of 135 bpm. The temperature was 38.1°C.

Paraclinical examinations revealed an inflammatory syndrome (CRP 157 mg/L, leukocytosis 12,700/mm³, anemia 9 g/dL), hyperamylasemia 169 IU/L, and a positive procalcitonin level of 50 ng/mL. Gastrointestinal and blood cultures were negative. Metabolic testing and brain CT scan were normal. Further questioning with the father revealed that the child had accidentally ingested an unspecified quantity of an organophosphate insecticide in agricultural fields. The first clinical signs appeared four hours after ingestion. Cholinesterase activity and organophosphate metabolites were not measured in blood or urine. A diagnosis of severe acute organophosphate poisoning was made given the context of ingestion of the product by the parents and the cholinergic syndrome with life-threatening distress in a child with no prior medical history. Resuscitation measures with respiratory support and sedation/ anaesthetics were initiated. Treatment with atropine at a dose of 0.5 mg/h by continuous infusion was rapidly started until the appearance of signs of atropinization (total duration of 28 hours). The antidote (pralidoxime) was not available. The outcome was favorable in less than 48 hours, with the child beginning to regain consciousness on day 4 and extubation on day 6 of hospitalization. The child had presented with confusion and a Glasgow Coma Scale (GCS) score of 13 without motor deficits. He was transferred to the pediatric ward and fully regained consciousness on day 15. The child returned home on day 18 with a prescription for motor physiotherapy sessions and after a month the clinical examination was without abnormalities.

DISCUSSIONS

Organophosphate poisoning, often accidental, preferentially affects children under five years of age, who are most often contaminated orally after ingesting the improperly stored toxin [2]. The number of children involved in these incidents is poorly known because few studies have focused on the pediatric characteristics of organophosphate poisoning [3, 4]. Poisoning in children, often accidental, is less severe than poisoning in adults, which is generally intentional due to higher concentrations of industrial products. Our patient was older but not attending school and lived in a rural area where children, often accompanied by their parents, frequently use these products in agriculture. The clinical presentation of organophosphate poisoning related to a cholinergic crisis is characterized by three main syndromes, which are not always present: a muscarinic syndrome, a nicotinic syndrome, and a central syndrome. Diagnosis is not easy in children. Indeed, it is only suspected on admission in 57% of cases in the series by Zweiner and Ginsburg [5]. Neurological disorders are predominant on admission in children, whereas muscarinic signs are inconsistent in the series by Lifshitz [6]. Our patient met some of these clinical diagnostic criteria, presenting with a muscarinic syndrome and a prominent central nervous system syndrome characterized by altered consciousness and episodes of seizures. All of these symptoms occurred 24 hours after ingestion of the organophosphate. In Lifshitz 's study, all children had neurological symptoms (hypotonia, stupor, or even coma) upon admission, while muscarinic signs were inconsistent. This is likely due to increased permeability of the blood-brain barrier, facilitating the penetration of organophosphates into the brain, or to preferential inhibition of acetylcholinesterases in the central nervous system [6]. In all cases, neurological signs are typical in children, and the absence of muscarinic signs should not rule out the diagnosis. A cholinergic crisis with muscarinic and nicotinic signs requires intensive care management. It occurs after a highly variable delay, from a few minutes after inhalation to 24 hours after cutaneous or gastrointestinal poisoning [7]. Diagnostic confirmation is based on the collapse of erythrocyte cholinesterase activity [8]. In our case, the diagnosis of severe acute poisoning was made given the context of ingestion of the product brought by the parents and the cholinergic syndrome with life-threatening distress in a child with no prior medical history. We did not have access to enzyme activity testing. Treatment is primarily symptomatic, aiming to rapidly address any life-threatening distress, but also includes gastrointestinal decontamination and antidote treatment. Symptomatic treatment is based on maintaining vital functions: oxygen therapy, possibly intubation and mechanical ventilation, fluid resuscitation, and vasoactive drugs in cases of hemodynamic failure [9]. Decontamination by gastric lavage is recommended in acute organophosphate poisoning. It must be performed early, within one hour, due to the rapid absorption of organophosphates [10]. Our patient did not receive gastric lavage due to a delay in treatment, making it difficult to manage in a remote area. We implemented resuscitation measures combining respiratory support, sedation, and tracheobronchial suctioning for 48 hours. Specific treatment involves pralidoxime and atropine. Atropine constitutes the core of the pathophysiological treatment [9]. Atropine acts in competition with acetylcholine at muscarinic receptors, treating bronchospasm and bronchial hypersecretion without addressing neuromuscular phenomena [10]. Doses of approximately 0.015 to 0.05 mg/kg are necessary and should be repeated until signs of atropinization appear (mydriasis, tachycardia) [2]. A study conducted in Sri Lanka in 1991 during a pralidoxime shortage showed that treating moderate to severe poisoning with atropine alone had the same efficacy in terms of mortality, duration of ventilation, or hospitalization as the standard treatment with the atropine- pralidoxime combination [11]. Our patient had received atropine treatment via syringe pump electrical. The outcome was favorable, and the child was transferred to the pediatric ward for follow-up. In the reported case, well-managed and rapidly initiated symptomatic treatment led to a favorable outcome without the need for an antidote. Organophosphate insecticide poisoning results in death in 4 to 30% of cases. The presence of hemodynamic failure and the

need for mechanical ventilation increase the mortality risk to approximately 50% [12].

CONCLUSION

Organophore poisoning is a public health problem in developing countries. It often manifests as neurological symptoms in children. Treatment, which is primarily symptomatic and antidotal, can be difficult due to the scarcity of resuscitation equipment in some regions.

Abbreviations: GCS: Glasgow Coma Scale, OP: organophosphate.

REFERENCES

1. Achour S, Rhalem N, Khattab A, Soulaymani R (2009) Clinical and therapeutic characteristics of organophosphate poisoning: data from the Moroccan Poison Control and Pharmacovigilance Center (CAPM). Elsevier Masson SAS 57(1): S4.
2. Paget C, Menard S, Wroblewski I, Gout J, Danel V, Bost M (2002) Acute organophosphate poisoning following the use of an insecticide as an anti-lice shampoo. Archive de Pediatrie 9 (9): 913-916.
3. Emerson GM, Gray NM, Jelinek GA, Mountain D, Mead HJ (1999) Organophosphate poisoning in Perth, Western Australia, 1987-1996. J Emerg Med 17(2): 273–7.
4. Mortensen ML (1986) Management of acute childhood poisonings caused by selected insecticides and herbicides. Pediatr Clin North Am 33(2) :421-45.
5. Zwiener RJ, Ginsburg CM (1988) Organophosphate and carbamate poisoning in infants and children. Pediatrics 81(1): 121-6.
6. Lifshitz M, Shahak E, Sofer S (1988) Carbamate and organophosphate poisoning in young Children. Pediatrics Emerg Care 15(2):102-3.
7. Rousseau JM, Rüttimann M, Brinquin L (2000) Acute poisoning by organophosphate neurotoxins: insecticides and chemical weapons. Ann Fr Anesth Réanimation 19(8): 588-98.
8. Pontal PG (2007) Organophosphate insecticides, carbamates, synthetic pyrethroids and miscellaneous. *EMC - Occupational and Environmental Pathology* 6(2):1-24.
9. Bismuth, Baud, Conso (1998) Clinical Toxicology. 5th edition. Flammarion médecine sciences Publications, Paris, France. p 1092.
10. Eddleston M, Buckley NA, Eyer P, Dawson AH (2008) Management of acute organophosphorus pesticide poisoning. Lancet 371(9612):597–607.
11. De Silva HJ, Wijewickrema R, Senanayake N (1992) Does pralidoxime affects outcome of management in acute organophosphorus Poisoning ? Lancet London Engl 339(8802): 1136-8.
12. Thabet H, Brahmi N, Kouraïchi N, Elghord H, Amamou M (2009). Organophosphate pesticide poisoning: new concepts. Réanimation 18(7):633-9.