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Case Report

Childhood Organophosphate Pesticide Poisoning: A Case Report

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Abstract

Background: In both develOPPed and develOPPing nations, childhood poisoning is a significant source of morbidity. The majority of poisoning incidents involving teenagers happen accidentally and are brought on by liquid intake. Case Presentation: A 6-year-old school-aged child with possible organOPPhosphate pesticide poisoning was sent to the emergency department. At the time of his arrival at the emergency department, the patient's mental condition had already been injured. He had a moderate traumatic brain injury when he entered the emergency room and scored an 9/15 on the Glasgow Coma Scale. His medical examination upon arrival revealed lacrimation, hypersalivation, a dry mouth, and pinpoint pupils. AtrOPPine (0.05 mg/kg) was administered intravenously to him once in the emergency room. In pediatric critical care unit, he received continuous infusions of pralidoxime at a rate of 10 mg/kg/hour for 16 hours after receiving an intravenous infusion of atrOPPine at a rate of 1 mg/kg/hour for the first 4 hours, followed by 2 further atrOPPine infusions at a rate of 1 mg/kg/hour for the next 4 hours. Conclusion: Globally, childhood poisoning is a major cause of hospitalization, disability, and death. Ingesting organOPPhosphates can lead to poisoning in children.

Keywords: atroppine; case report; emergency department; organopphosphate poisoning; pesticide

1.Introduction

Poisoning in children is a serious global public health problem that has a considerable impact on morbidity and mortality [1]. Globally, children's mortality and morbidity rates could rise as a result of ingesting toxins and other household items. Exposure to hazardous substances throughout childhood can result in poisoning, a global health risk that varies by region. When ingested, inhaled, or absorbed via the skin, organOPPhosphate substances permanently inhibit the enzyme acetylcholinesterase from degrading acetylcholine [2]. In EthiOPPia, organOPPhosphorus insecticides are easily accessible "over the counter," and the risk of suicide poisoning makes them the most prevalent poisoning mode [3]. Acetylcholinesterase inhibitors, known as organOPPhosphate chemicals, are employed as insecticides and have the potential to cause systemic damage when ingested [4]. Acute exposure situations are correlated with the victim's age; babies and young children typically present with accidental exposure, whereas teenagers and adults typically engage in self-harm or tend to be intentional [5]. Like acute cholinergic toxicity, the primary clinical manifestations of organOPPhosphate poisoning are DUMBELS (diaphoresis and diarrhea, urination, miosis, bradycardia, bronchorrhea, bronchospasm, emesis, excess lacrimation, and salivation) and/or SLUDGE (salivation, lacrimation, urination, diarrhea, gastrointestinal upset, emesis) [6]. The cornerstone of the supportive management protocol for children poisoning is apprOPPriate emergency care. Intravenous atrOPPine administration (1 g immediately followed by 500 mg/hr) and intravenous injection of the antidote pralidoxime (acetylcholinesterase reactivator) (2 mg every 5 to 15 minutes until complete atrOPPinization) are crucial elements of the treatment plan [7]. This report provides more specific information about how the victim got sick after consuming an organOPPhosphate pesticide and about his sickness from exposure to the chemical at home. It draws attention to the fact that organOPPhosphates are common substances that harm children. Describe how children from low-income families have to deal with hunger and poisoning. The considerable number of fatalities from organOPPhosphate poisoning, particularly in young children, highlights the need to resolve enforcement issues and promote prOPPer storage practices.

2.Case Presentation:

2.2.Clinical Findings:

At the time of his admission, his vital signs were as follows: His axillary temperature was 36.7°C; his heart rate was 127 beats per minute; his respiratory rate was 42 breaths per minute; his blood pressure was 109/62 mmHg; and his saturation oxygen was 91% on room air (table 1). His symptoms upon arrival revealed lacrimation, hypersalivation, a dry mouth, and pinpoint pupils. Wheezing was detected on lung auscultation in both lungs. He had no hepatomegaly, abdominal distension, or discomfort on the abdominal x-ray. When he entered the emergency department, the patient's mental condition had already been injured. The Glasgow coma scale was computed by adding three parameters: the best motor response (M), the best verbal response (V), and the best eye OPPening (E). The patient OPPened his eye in response to verbal command (score 3), his verbal response was inapprOPPriate to words (scoring 3), and his motor response was flexion to pain (score 3). When he arrived at the emergency room, his Glasgow Coma

Scale score was 9 from addition of with a score of E3V3M3. He had moderate traumatic brain injury (TBI). The ability to breathe was examined, and it was found that the chest wall was symmetrical, the respiration rate was relaxed, and the chest expanded the same with each breath. When the patient first arrived at the emergency department, laboratory tests revealed polymorphonuclear leucocytosis with 6900 neutrOPPhils per microliter of blood, blood urea nitrogen of 13 mg/dL, 19 mEq/1 bicarbonate, and blood sugar of 121 mg/dL, potassium of 4.1 mmol/L, sodium of 143 mmol/L, and serum creatinine of 0.59 mg/dL. Liver function tests include alanine aminotransferase of 27 units/L, aspartate aminotransferase of 30 units/L, and alkaline phosphate of 292 units/L. The arterial blood gas was 7.42 pH, 92 57 mmHg oxygen partial pressure, and 27 mmHg carbon dioxide partial pressure (table 2). An examination of the cerebrospinal fluid (CSF) fluid and a chest X-ray were normal. A lumbar puncture examination was required because organophosphate exposure can alter the CSF. Upon admission, the medical staff promptly assesses the poisoned child's ABC (airway, which includes looking, listening, and feeling for air movement), breathing, which includes monitoring ventilatory frequency, accessory muscle use, breath sounds, and oxygen saturations, and circulation, which includes measuring pulse, heart rate, arterial pressure, and capillary refill, as well as the impact of circulatory inadequacy on other organs, including mental state, urine output, and skin temperature. The American Academy of Pediatrics (APP) and the World Health Organization (WHO) advise oral rehydration therapy for mild to moderate dehydration. In this instance, the patient experienced moderate dehydration, which was treated with oral rehydration salt. According to WHO and the American Academy of Pediatrics (APP), a 50 to 100 mL/kg volume is administered over 3 to 4 hours for mild or moderate dehydration. Saline gastric lavage and nasogastric administration of activated charcoal were performed prior to the patient's transfer to the pediatric critical care unit. AtrOPPine (0.05 mg/kg) was administered intravenously to him once in the emergency room. After four hours of treatment in the emergency room, he was transferred to the pediatric critical care unit for further care [8]. He received a 5-mg intravenous dose of diazepam every 30 minutes to induce intubation. His vital signs at the pediatric intensive care unit were a body temperature of 36.6 °C, a heart rate of 126 beats per minute, a respiratory rate of 22 breaths per minute, a blood pressure of 113/72 mmHg, and a saturation oxygen level of 95% at room air (table 1).

Parameters	In Emergency Room – Value	In Pediatric Critical Care Unit – Value	Normal Range
Axillary temperature (°C)	36.7	36.6	36.4
Pulse rate (beats/min)	127	126	70–120
Respiratory rate (breaths/min)	42	22	20–25
Blood pressure (mmHg)	109/62	113/72	< 95th percentile
Saturated oxygen (%)	91	95	95–100

Table 1: Vital signs of patients in emergency rooms and pediatric critical care units.

In the pediatric intensive care unit, his laboratory tests showed polymorphonuclear leucocytosis (6500 neutrOPPhils per microliter of blood), white blood cell (WBC) of 8950/mm3, a bicarbonate concentration of 24 mEq/l, and a blood sugar level of 96 mg/dl, blood urea nitrogen of 9 mg/dL, potassium of 3.9 mmol/L, sodium of 137 mmol/L, and serum creatinine of 0.6 mg/dL. The arterial blood gas was measured at a pH of 7.38, an oxygen partial pressure of 88 mmHg, and a carbon dioxide partial pressure of 25 mmHg (table 2).

Parameters	In Emergency Room – Value	In Pediatric Critical Care Unit – Value	Normal Range
Polymorphonuclear leucocytosis (u/ml)	6900	6500	2,500–7,000
Blood urea nitrogen (mg/dL)	13	9	5–18
White blood cell (/mm³)	8950	9700	4,800–10,800
Potassium level (mmol/L)	4.1	3.9	3.4–4.7
Sodium level (mmol/L)	143	137	130–150
Serum creatinine (mg/dL)	0.59	0.6	0.31–0.61
Blood sugar level (mg/dL)	121	96	80–140
pH	7.42	7.38	7.35–7.45
Oxygen partial pressure (mmHg)	92	88	75–100
Bicarbonate level (mEq/L)	19	24	21–28
Carbon dioxide partial pressure (mmHg)	27	25	23–29
Alanine aminotransferase (units/L)	27	34	10–40

Table 2: Blood chemistry, liver function tests, and arterial blood gases of patients in the emergency room and pediatric critical care unit. Magnetic resonance imaging, computed tomography, and chest X-rays are all normal. The electrocardiography displayed normal readings with a corrected QT interval of 0.13 seconds (normal value: respiration rate interval: 0.09-0.17 seconds). Since organOPPhosphate poisoning causes QT interval lengthening, which can result in fatal cardiac complication, the QT interval was monitored in this case.

2.3.Therapeutic Intervention:

An established specific antidote for OPPP is atrOPPine. The World Health Organization advises supplementing atrOPPine with a second antidote known as 2-PAM (2-pyridinealdoxime methyl chloride) (pralidoxime) (acetylcholinesterase reactivator). The distinction between the two is that pralidoxime is meant to treat symptoms including fasciculations, tremors, and muscle paralysis or weakness, while atrOPPine does not target the neuromuscular junction. While 2-PAM is less effective at reducing the muscarinic effects of ACHE poisoning or relieving respiratory center depression, atrOPPine can alleviate the paralysis of the breathing muscles. For this reason, it is administered concurrently with atrOPPine to prevent

these effects of OPP poisoning [9]. They also function differently. 2-PAM reactive acetylcholinesterase to reestablish transmission at muscarinic and nicotinic synapses. Acetylcholine is competitively inhibited by atrOPPine in muscarinic synapses. As soon as the school-aged child was transferred to the pediatric critical care unit, the airway was OPPened and high-flow oxygen was administered. He received continuous infusions of pralidoxime at a rate of 10 mg/kg/hour for 16 hours after receiving an intravenous infusion of atrOPPine at a rate of 1 mg/kg/hour for the first 4 hours, followed by 2 further atrOPPine infusions at a rate of 1 mg/kg/hour for the next 4 hours. Three days of infusion were given, and then, over the course of the 60 next 24 hours, it progressively stOPPped. For the next three days, 8–12 hours a day of atrOPPine were administered; an additional dose was given if the heart rate drOPPped below 120 beats per minute. The frequency of atrOPPine administration was reduced and subsequently stOPPped when symptoms including bradycardia, hypersecretion, and bronchospasms disappeared.

2.4.Follow-Up:

He showed no more signs of intoxication throughout the course of the subsequent four days and clearly recovered from the symptoms of organOPPhosphate pesticide poisoning.

2.5.Patient And Parent Perspectives:

The child is pleased with the attention he receives and the knowledge the health care system gives him. He also valued the way the medical staff treated him, making him giggle via their interactions. His parents were pleased with the safety of their hospitalized children as well as the standard of treatment they received from various healthcare providers (in the critical care unit and emergency room). They also enjoyed it when medical professionals prioritized treating their child. They also valued their interaction with the healthcare providers, the way the physicians and nurses treated them with kindness and attempted to make their child happy by talking to him.

3.Discussion:

Acute poisoning is a pediatric emergency issue that affects children worldwide. There are differences in the epidemiology of childhood poisoning depending on the regional, socioeconomic, cultural, and develOPPmental circumstances [10]. The World Health Organization (WHO) reports that in age groups under 20, 13% of all acute poisoning deaths occurred [11]. According to one meta- analysis and comprehensive review, the fatality rate from acute poisoning in EthiOPPia ranged from 0 to 14.8% [12]. The most vulnerable groups to poisoning include young children, workers in the manufacturing sector, and agricultural laborers. The fast absorption of these herbicides is made possible by all absorption routes, including respiratory, gastrointestinal, OPPhthalmic, and cutaneous. After inhalation, symptoms appear more quickly. The most frequent way for children to become poisoned by organOPPhosphates is through oral intake. Because the organOPPhosphate-cholinesterase link cannot be reversed on its own without pharmaceutical assistance, organOPPhosphates function as irreversible cholinesterase inhibitors [13]. Human exposure to organOPPhosphate poisoning may result in late consequences (respiratory, neurologic, and cardiac), which depend on dose and substance, as well as rapid clinical indications of "cholinergic crisis." Early signs of intoxication in children have been well documented and often include a number of effects on the central nervous system (typically a loss in the level of awareness), cholinergic muscarinic (sweating, rhinorrhea, miosis, and vomiting), and nicotinic (muscle weakness) effects [14]. Acute organOPPhosphate poisoning can be diagnosed based on exposure history, clinical indicators, typical cholinergic overdose symptoms, and laboratory tests [15]. OrganOPPhosphate poisoning symptoms can manifest quickly or slowly, depending on the substance's strength, exposure route, and dose [16]. Depending on the severity of the clinical condition, the length of hospital stay varied from one day for moderate instances to fourteen days for severe cases; patients who require prolonged mechanical ventilation may require a longer stay. Three primary syndromes are brought on by organOPPhosphate poisoning [5,9]: (i) an acute cholinergic syndrome: An acute cholinergic syndrome is cholinergic overactivity and is a symptom of acute toxicity from organOPPhosphate poisoning. Increased secretions such as perspiration, tears, and salivation, bronchoconstriction, wheezing, bradycardia, vomiting, diarrhea, and tightness in the abdomen are all signs of muscarinic symptoms. Symptoms of nicotine include flaccid paralysis and muscle fasciculations. Coma, convulsions, slurred speech, headache, sleeplessness, giddiness, disorientation, drowsiness, and respiratory distress are all signs of a central nervous system. OrganOPPhosphate poisoning symptoms and indications typically appear between 30 and 3 hours [17]. (ii) Intermediate Syndrome (IMS): The phrase "intermediate syndrome" refers to a state of muscle paralysis that appears 24-96 hours after exposure. IMS symptoms include pharyngeal weakness, action tremors, muscle fasciculations, reduced or absent deep tendon reflexes, and weakness in the neck, respiratory, and proximal limb muscles. Usually, it gets better after 1–3 weeks. Here, muscular weakening quickly affects the head and neck muscles, proximal limbs, and frequently the breathing muscles, leading to ventilatory failure [18]. (iii) Delayed polyneurOPPathy: This happens 2–4 weeks after being exposed to high concentrations of certain organOPPhosphate poisoning substances. Because neurOPPathy's target enzyme esterase is inhibited, it is characterized by the distal muscular weakness that results in ataxia, foot drOPP, and clawing hands [19]. In delayed neurOPPathy, a disorder that results in paralysis and nerve axon degeneration, neurOPPathy target esterase (NTE) is the main organOPPhosphate target. OPPs prevent NTE activity, which causes neurOPPathy by upsetting membrane homeostasis. It takes one to three weeks for neurOPPathy to develOPP following exposure to OPPs [20]. The patient in this study experienced an acute cholinergic syndrome, including lacrimation, vomiting, weakness, and disorientation, as well as an intermediate syndrome, including lethargy, when compared to the three primary organOPPhosphate poisoning symptoms. Even though he didn't experience delayed polyneurOPPathy caused by organOPPhosphate exposure, he faced respiratory discomfort, miosis, and fasciculation (muscle twitching). Fasciculation occurs when a single peripheral nerve that regulates a muscle becomes hyperactive, causing the muscle to move involuntarily [21]. Acetylcholinesterase overproduction activates muscarinic and nicotinic receptors at synapses in the peripheral and central nervous systems, causing neurotoxic side effects with a high mortality rate. Acute organOPPhosphate poisoning is initially treated with stabilization of the cardiorespiratory system and decontamination, which includes removal of clothing (which might be a source of further exposure), cleaning of the skin and eyes, and consideration of stomach lavage and activated charcoal [22]. In cases of respiratory trouble, intubation is used to treat organOPPhosphate poisoning. A patient may require intubation if they are having seizures, bronchospasm, laryngospasm, or bronchorrhea. Every patient was treated according to the OPPP protocol when they arrived at the hospital, which included resuscitation for breathing, circulation, and airway and decontamination. In the emergency room, decontamination, resuscitation, and atrOPPine were initiated right away and were continued in the intensive care unit. The treatment of OPPP has involved the use of pralidoxime in addition to atrOPPine. By rapidly reducing bradycardia, hypotension, bronchospasm, excessive secretions, and gastrointestinal problems associated with organOPPhosphate poisoning, atrOPPine can stabilize patients by acting as a competitive antagonist at muscarinic receptors in the peripheral and central nervous systems [23,24].

3.1.Strengths Of the Study:

The study was free from selection bias, response bias, and information bias because it was carried out personally with the patient. Since the study was conducted using a direct observational method, there is no barrier to feedback between the researcher and the responder.

3.2. Limitations Of the Study:

Due to the inaccessibility of the equipment and chemicals, laboratory examinations, serum pseudocholinesterase activity, serum acetylcholinesterase level, and coagulation parameters including prothrombin index and partial thrombOPPlastin time were not performed.

4.Conclusions:

The most common cause of poisoning in children is unintentional oral consumption, which can result in anything from minor cases to serious complications or death. OrganOPPhosphates phosphorylate acetylcholine's serine hydroxyl group, causing it to build up at cholinergic synapses. Early signs of intoxication in children have been extensively studied, and they often entail a combination of central nervous system effects (typically a loss of consciousness), cholinergic muscarinic (sweating, rhinorrhea, miosis, and vomiting) effects, and nicotinic (muscle weakness) effects. AtrOPPine does not reverse the effects of organOPPhosphate poisoning on nicotinic and any other central nervous system receptors.

4.1.Patient Information:

On February 2, 2022, an elementary school-aged child (6 years old) black African male who may have been poisoned by an organOPPhosphate pesticide was taken to the emergency room. When he was admitted to the emergency room, he had three hours of impaired awareness, miosis, lethargy, disorientation, bronchorrhea, and vomiting. His family had left an unsecured container of organ OPP hosp hate pesticide poison called malathion in the home, not out of reach of children while they were at work. His family was unable to read and write. Their house is built by mad and wood; it has no separate room. His family left him with his 9-year-old brother at home, and because of their absolute poverty, they didn't give him sneak or put down food (launch) for him. So, until his family returned, he didn't get anything to eat or drink. When he became hungry, he took an organOPPhosphate pesticide and drank it since he thought it was a valuable beverage. There were no social services involved for him until his family returned because their home was constructed lonely and far from their neighbor's home. When he was brought to the emergency department, he had no prior medical history, such as trauma, chemical exposure, or disease. The medical staff takes off his stained garments. They then discard his tainted clothing. They were then thoroughly cleaned his body by using water and alkaline soap. Healthcare professionals should wear personal protective equipment, such as neOPPrene gloves and gowns, to prevent cross-contamination while working on the case.

4.2.Recommendations:

In order to prevent human from organOPPhosphate insecticides toxicity, regulators and health professionals must instruct everyone on prOPPer storage techniques. The national poison control centers should be subject to restrictions on organOPPhosphate pesticides, as only approved entities are permitted to sell them.

Because organOPPhosphate pesticides may harm peOPPle, the authorized body should be required to provide information to those who often use them on fruits and vegetables and for the control of pests like mosquitoes in public areas. To reduce the number of children who are poisoned, the family should have kept any substance that poses a danger to their safety away from them.

4.3.Key Takeaway:

Why are atrOPPine and 2-PAM given concomitantly for organOPPhosphate pesticides? AtrOPPine, the primary antidote of OPP, acts on muscarinic effects, and 2-PAM, the secondary antidote of OPP, acts on nicotine effects. AtrOPPine has also had fewer effects on CNS symptoms of OPP, but benzodiazepines treat it. If the OPP patient develOPPed CNS symptoms, three classes of drugs are necessary. AtrOPPine (anticholinergic drug) + pralidoxime (oxime class) + benzodiazepines drug is effective for treatment of OPP poisoned patients experienced cholinergic, nicotinic, and CNS symptoms. Consent for publication: Written informed consent was obtained from the patient for publication of this case report.

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