CASE REPORT

Accidental poisoning with aluminum phosphide presenting with excessive cholinergic symptoms with response to atropine: A case report

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ABSTRACT

Accidental poisoning in children, though underreported in our environment, is common and could prove fatal. It is important to identify the primary chemical agent that is responsible for the poisoning. We present a case of accidental ingestion of fish poisoned with aluminum phosphide (AlP) used as rat poisoning by a 14-month-old girl. At presentation, the actual chemical content of the poison was not available and clinical features were suggestive of organophosphate poisoning. She was commenced on atropine together with other treatment, on which she made remarkable improvement. The atropine was continued with complete resolution of symptoms on the third day of admission. We, therefore, report a serendipitous use of atropine in the management of AlP poisoning with successful outcome.

Key words: Aluminum phosphide, atropine, organophosphate, poisoning, rat poisoning

INTRODUTION

AlP is a commonly used fumigant in storage of grains, especially in developing countries. It is described as a "near-ideal" fumigant for grains—cheap, highly potent with no effect on seed viability, and thus, safe for humans when used for this purpose.[1,2] It is also used as a rodenticide. It is available in the form of a tablet, powder/granules, and dusts. Ingestion of pesticides for suicidal intent has been reported worldwide[2,3] with more cases reported in the past two decades, especially in Northern India, where AlP used for suicide has reached an epidemic proportion.[4,5] Here in Nigeria, like in many African countries, there is a paucity of reported cases of AlP poisoning.[2] However, there are reported cases of rodenticide/insecticide poisoning with no much mentioning of the actual chemical content.[6] Many of these cases are homicide/suicide but some are accidental. The latter is more common in toddlers.[7-10] Many of these forms of poisoning from rodenticides/insecticides are often managed as organophosphate poisoning, an active ingredient commonly used in insecticides/pesticides.

The active pesticidal ingredient in AlP is phosphine, a trihydrate of phosphorus (PH_3).[11] It is a very lethal chemical, with mortality reported in the range of 50–100%.[12] It has no known antidote, and management is mainly supportive. Phosphine is formed when AlP is exposed to water and/or hydrochloric acid in the stomach, where it is rapidly absorbed into the circulation. The mechanism of action by which it exerts it toxicity, though not fully understand, is believed to be one or all of the following three means, which are closely related.[11,13]

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1. Neural/behavioral: This is believed to be mediated by inhibition of acetyl cholinesterase, which is an enzyme that is responsible for degradation of acetyl choline, the major neurotransmitter in the parasympathetic nervous system. Thus, the patient presents with features of excessive parasympathetic activities ranging from excitation/agitation to convulsions. These features were successfully treated, especially in studies using insects by atropine and oximes which are the known antidotes of organophosphate poisoning.
2. Mitochondrial toxicity leading to a metabolic crisis
3. Generation of reactive oxygen species and/or some reactive phosphorus species that cause cellular/tissue damage.

Although, studies have shown that AlP poisoning has no known specific pattern of presentation, the most common in the lethal forms is refractory cardiogenic shock.[13]

Accidental ingestion of poison is most common in children younger than two years of age because of their exploratory nature and the tendency to put everything in their mouth.[7,8]

We report a case of acute poisoning from accidental ingestion of AlP in a 14-month-old girl. At presentation, the chemical agent contained in the poison was not known and her predominant features were those of excessive parasympathetic activities. A presumptive diagnosis of organophosphate poisoning was made; she was commenced on atropine among other types of treatment on which she improved. This case highlights the possible benefit of the use of atropine in the management of AlP poisoning, especially when a patient presents with features of excessive parasympathetic activities, more so in resource-limited settings.

CASE REPORT

A 14-month-old girl was brought to the emergency pediatrics unit (EPU) with complaints of cough, difficulty with breathing, excessive secretion from the mouth, and restlessness of sudden onset noticed an hour before presentation. She was well until about an hour before presentation (about 30 min after she was last seen) when her elder sister, a 9-year-old, heard her coughing vigorously from the house garage. On checking on her, she also noticed that her breathing was fast. She was having excessive secretion from the mouth with a garlic-like smell. She was restless. She had no convulsion or fever. She also noticed she had a remnant of fish poisoned with rat poison in her hand and mouth. The poisoned fish was used as bait for rats the previous night. She quickly dislodged the one in her mouth and informed the parents. She was given a mixture of milk and palm oil and she vomited once. However, she was rushed to the EPU on account of worsening of the symptoms. Other aspects of her history were not adversely remarkable. Her developmental milestone was normal. At presentation, she was dyspneic and tachypneic with a respiratory rate of 80cpm, with widespread coarse crepitations. Her oxygen saturation in room air was 82%, pulse rate was 180bpm, BP was 100/60 mmHg, and capillary refill time was prompt. Her temperature was normal (36.3°C). She was restless and irritable. Her pupils were 4 mm bilaterally and equally reactive to light. She had no redness of the oral mucosa or mouth ulcer. Other examination findings were essentially normal. Her random blood sugar was 11.2 mmol/L. The full blood count showed leukocytosis with neutrophilia and thrombocytosis. The urea, electrolyte, and creatinine were normal. At presentation, the actual chemical constituent of the poison was not known and the relatives were asked to bring the poison with its label, which they did at the fourth hour into the admission. A presumptive diagnosis of organophosphate poisoning was made based on the clinical presentation and respiratory symptoms believed to be due to bronchochorrhea. She was placed on intranasal oxygen, given intravenous atropine, 0.15 mg and lactated ringers fluid at maintenance. About 3 h later, the secretion from the mouth had resolved. She was less restless. The pulse rate, respiratory rate, and SPO_2 have all improved (PR-170 bpm, RR-70 cpm, and SpO_2 96% on intranasal O_2 at 2 L/min, respectively). However, she was found to be febrile (39.3°C). She was exposed and tepid sponged, and was continued on the earlier management with atropine continued six hourly for 24 h. She had gastric lavage with coconut oil. Her blood sugar normalized at the fourth hour of admission, though no insulin was given. Fever resolved at the sixth hour and continued to show progressive improvement, with complete resolution of the respiratory symptoms on the third day of admission and was weaned off oxygen on the same day. She passed loose stool once each on the second and third days of admission; they were nonbloody, non-mucoid. From the label, the poison contain AIP and each tablet generates 33% W/W phosphine.

DISCUSSION

The clinical features of AlP poisoning are highly variable but usually multisystemic.[14] The diagnosis is usually made by a clinical history of exposure to the poison, clinical features, and the presence of a garlic-like smell. It can be confirmed by the silver nitrate test on gastric aspirate of the patient.[4,15] Our index case had a positive history of exposure to AlP and a garlic-like smell, with clinical features in keeping with those

reported from several studies. Many studies have reported many features, clinical and laboratory, associated with poor prognosis and high mortalities.[4,13,15] Within the limit of the resources available in our facilities, we found hyperglycemia and leukocytosis among the poor prognostic factors in our index case.[14] Because AlP has no known specific antidote, its management is mainly supportive; it includes measures such as gastric lavage with coconut oil, potassium permanganate, and activated charcoal; supplemental oxygen with or without mechanical ventilation; ionotropic drugs such as adrenaline, noradrenaline, digoxin, dopamine, and dobutamine.[14,15] Other medications such as intravenous infusions of sodium biocarbonate and magnesium sulfate have been used to treat metabolic acidosis.[13,15] There are recent studies evaluating the role of various antioxidants also, with some promising findings.[13,16] Our index case had gastric lavage with coconut oil, supplemental oxygen, intravenous fluid, and intravenous boluses of atropine, 0.02 mg/kg (0.15 mg) at presentation, 3 h after and then six hourly for 24 h. Although the atropine was started on the premises of the presumptive diagnosis of organophosphate poisoning based on the presenting features, it was continued despite the confirmation of AlP poisoning 4 h into the admission because of the remarkable improvement that the patient had on the usage of atropine along with other medications. Studies in insects have shown beneficial effects of atropine in the management of AlP poisoning but not in vertebrates[11] and, to the best of our knowledge, the report of its use for this purpose in humans is not available. However, earlier experiences in our center on the management of "rat poisoning" (chemical contents mostly unspecified/unknown), mostly unreported, have also employed the use of atropine in patients with excessive salivation and suspected bronchochorrhea with significant clinical outcome.[6] Atropine antagonizes the muscarinic effects of acetylcholine and attenuates the excessive parasympathetic effects such as excessive secretions/bronchochorrhea. It may also reduce the cardiac manifestations such as arrhythmias and cardiogenic shock, which are associated with poor outcome in AlP poisoning.[17] Our index case who was commenced on atropine within two hours of exposure to AlP had remarkable improvement of her respiratory symptoms. Although, for logistic reasons, we were not able to do an ECG at presentation, other than the tachycardia, clinically she had no features to suggest arrhythmias and she never developed cardiogenic shock; her pulses were regular, and blood pressure measurements were normal with prompt capillary refill time. Although emphasis should be on the prevention of AlP poisoning, there is a need to consider early use of atropine in patients with AlP poisoning, especially those with excessive cholinergic stimulation such as our index case.

CONCLUSION

Doctors should be aware of AlP poisoning from rodenticide/fumigant exposure. Therefore, an attempt should be made to identify the primary chemical agent(s) associated with each poisoning case, because overlaps in clinical features may be experienced.

Recommendation

Further studies may help elucidate the effectiveness or otherwise of atropine in the management of AlP poisoning. Regulation of production and usage of these chemicals would reduce poisoning and its attendant effects. Furthermore, there is the need for educating the people on the dangers of these harmful chemicals and the need for parents to properly supervise their wards to avert such dangers.

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Conflicts of interest

There are no conflicts of interest.

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