CASE REPORT

Amitraz: An unusual poisoning

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ABSTRACT

Suicidal poisoning is rarely associated with intake of rare poisons, as the victim would grab any harmful chemical substance available in the vicinity for ingestion in rage. We report a case of suicidal intake of amitraz, a non-systemic acaricide, an ectoparasite repellant and insecticide used in veterinary medicine. Amitraz intake is rarely lethal and management is symptomatic. Lack of a specific antidote and management protocols for amitraz intoxication, leave only the previous case reports valuable for physicians dealing with it. We report a case of a patient presenting after ten hours of poison ingestion with unconsciousness, miosis and persistent bradycardia, the most frequently reported symptoms associated with this type of poisoning. The patient was managed with symptomatic treatment, her condition started improving after 12 hours and she was discharged in good health over next 36 hours.

Keywords: Amitraz poisoning; Acaricide: Suicidal attempt; Symptomatic treatment

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INTRODUCTION

Amitraz is an insecticide/acaricide of the formamidine pesticides group of the amidine chemical family.[1] It is generally used to control animal ectoparasites and has α_2-adrenergic agonist activity. A limited number of human intoxication cases have been reported in the literature with distribution of cases scattered worldwide.[1,2] Extensive search of literature revealed that only a few cases have been reported on poisoning with this insecticide in Southeast Asia. Commercial preparation of amitraz contains 12.5-20% of the drug in organic solvents, especially xylene, which is a component of paints, cleaners, and glues. Poisoning via amitraz occurs through oral, inhalational (most potential), and dermal routes.[3]The toxic effects of amitraz are due to its α_2-adrenergic agonist actions in the central nervous system and both α_1 and α_2 adrenergic receptor stimulation in the periphery. It also inhibits monoamine oxidase (MAO) enzyme activity and prostaglandin E2 synthesis, though some of these effects may be dose dependent.[4]

Toxic effects include numerous signs and symptoms varying from nausea, vomiting, bradycardia, hypotension or hypertension, hypothermia, hyperglycemia, polyuria, decreased gastrointestinal motility and intestinal distension, miosis, CNS depression with drowsiness, respiratory depression, convulsions and coma.[5] Most of the cases of human intoxication reported worldwide were suicidal attempts. Limited reports have been encountered from Southeast Asia, thereby limiting the general awareness regarding this toxin amongst clinicians. We report a similar case of suicidal poisoning with amitraz who was managed using standard detoxification guidelines.

CASE REPORT

A 22 years old male, ingested 2-3 tablespoons of amitraz poison in a suicidal attempt at night and was admitted to the emergency department almost after 10 hours. Relatives informed that the patient had 4-5 episodes of vomiting and loss of sensorium since last night. An empty bottle of pesticide amitraz 12.5% in 10 ml formulation was recovered from patient bedside. On examination the patient was unconscious with a Glasgow coma scale of 6/15. Pupils were bilaterally constricted and sluggishly reacting to light. Abdomen was soft on palpation and bowel sounds were normal on auscultation. Vitals signs revealed a heart rate of 50 beats/min, BP 100/76 mmHg, respiratory rate 16-20/ min and oxygen saturation (SpO2) 98% on 6L/min of oxygen supplied via facemask.

Patient was intubated and kept on ventilatory support using controlled mode ventilation (CMV). Activated charcoal tablets were administered via nasogastric tube and gastric lavage was performed till the disappearance of chemical odor in the aspirate. The evacuated gastric contents were sent for toxicological analysis. The investigation profile comprising of complete blood count, liver and renal

function tests, coagulation, serum electrolytes and blood sugar were all within normal limits. Arterial blood gas (ABG) analysis revealed metabolic acidosis with a pH of 7.23 which improved progressively in serial analysis.

As no specific antidote for amitraz poisoning exists, symptomatic treatment, comprising of intravenous atropine (1-2 mg every hour), antacids, multivitamins, diuretics and maintenance fluids, was instituted. A consistent finding was the presence of sinus bradycardia and the heart rate remained between 48-58 beats/min on continuous ECG monitoring. Hyperglycemia, commonly associated with amitraz poisoning, was not observed in our case, probably due to lower levels of poison absorption. No sedation was supplemented whilst being on ventilatory support. The patient started regaining consciousness with spontaneous eye opening and purposeful response to verbal stimuli after 12 hours of intensive care. The patient was weaned from ventilator, extubated the same day, and shifted to the ward on the next day. He was discharged from hospital on 4th day in good health.

DISCUSSION

Amitraz is a pharmaceutical, veterinary, and an agricultural product used for the treatment of generalized demodicosis in dogs and ticks and mites in cattle.[4] Intoxication with amitraz is commonly suicidal and infrequently accidental. It has shown to have reversible toxic effects on both animals and human beings which are rarely lethal or long lasting beyond 48 hours.[5,6] Since there are few reported human intoxications by this pesticide, the existing information about it has been built on animal studies or isolated case reports.[2-4]

In animals, amitraz show signs of CNS depression or stimulation according to the dose levels. At lower doses CNS stimulation may occur, manifested by hyperreactivity to external stimuli such as handling and increased food consumption. Higher doses have a CNS depressent effect with reduced spontaneous activity, bradycardia, hypothermia and respiratory depression leading to death. Animals that survive after toxic ingestion of a potentially lethal dose of amitraz show complete recovery from all signs and symptoms in about 7-10 days.[5]

The clinical signs and symptoms of amitraz toxicity in previous human cases include CNS depression, drowsiness, vomiting, miosis, bradycardia, hypotension, and hyperglycemia.[6] The toxic effects of amitraz, e.g. sedation, bradycardia and hypotension, occur due to α_2-receptor stimulation and mimic clonidine like syndrome and are the most frequently reported symptoms in this poisoning.[4] Amitraz also inhibits prostaglandin E2 synthesis in vivo contributing to its antipyretic and anti inflammatory activity, which explains hypothermia observed frequently in affected cases. Amitraz and its metabolites cause a characteristic 'mothball-like' or 'dry-cleaning' odour in the poisoned patient, which is often particularly noticeable on endotracheal suctioning.[7]

The basic approach to the patient with amitraz poisoning includes initial stabilization, measures to reduce absorption and to improve elimination of the toxin. Although activated charcoal and cathartic effects have not been evaluated, these are still included in the treatment protocol of these patients. Atropine is useful for treating hemodynamically unstable bradycardia. In some animal studies α_2-adrenergic antagonist such as yohimbine has shown to reverse most of the signs in amitraz poisoning.[8] However, till date, no studies or isolated reports warrant the use of these agents in humans, so they may be considered only in severe or non-responsive cases. Respiratory depression and need to protect airway may require intubation and elective ventilation till the patient regains consciousness.[9,10]

The presence of miosis, respiratory depression and bradycardia can confuse the clinical picture with that of organophosphate or opioid poisoning both of which need to be excluded. Levels of blood urea, creatinine, and sodium and potassium usually do not change in this poisoning, which is consistent with our observation.[5] We did not observed hyperglycemia, hypothermia and severe/unresponsive hypotension requiring inotropic support at any point of management, indicating that these effects may be dose-dependent.[4,12] Our patient had a relatively quick recovery and his condition improved after 24 hours of poison ingestion. Future studies on animal models can focus on evaluation of the efficacy and safety of α_2-adrenergic antagonists in antagonizing amitraz toxicity.

In conclusion, basic approach to a patient with amitraz poisoning consists of initial stabilization, reducing absorption, and increasing elimination of the toxin. Despite a life threatening clinical picture, amitraz poisoning in humans carries a low mortality when appropriate supportive therapy is given. Recovery usually occurs within 12-48 hours and the patients are discharged without any organ dysfunction.

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Difficult airway: An anesthesiologist's nightmare

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A 5 years old young boy with some obvious ENT malformations was referred to our newly commissioned hospital for a surgical intervention of his cleft palate. He had only one nostril and one ear on the right side. There was a small growth where the left ear would have been.

The procedure was to be carried out by a group of flying doctors from the African Medical Research Foundation (AMREF) based in Nairobi (Kenya), in one of their regular surgical camps in this part of the world. I was the only anesthesiologist posted to that hospital at the time, working with nurses that I had to train and supervise in the three operating rooms.

I was busy monitoring an infant under general anesthesia, when I was called by one of the two nurse anesthetists, trained by me and capable of doing long general and regional anesthesia lists with minimal help, to help with intubating the boy because according to them the 'airway was very strange'.

I joined the team and looked inside the throat and their worries were confirmed; neither the epiglottis nor the vocal cords were visible; half of the soft and hard palate was missing. I tried with all the blades in the set but failed to intubate. All long this period, the child's saturation was well maintained as we interrupted the attempts with episodic ventilation.

While pondering on what to do next, a surgeon came up and told me, "Sorry, I should have told you earlier, this patient was planned for this kind of surgery at the National Hospital a few weeks ago, but was postponed due to failed intubation".

I thought of a bougie, but none was in sight for such a small human being. I then improvised a neonatal urethral catheter stylet for an introducer and succeeded in pushing it through a very narrow anatomical orifice in the larynx leading to what I believed was the opening at vocal cords to the trachea, threaded over it my armored 3.5mm endotracheal tube and by God's grace into the trachea I went. Capnography vindicated my successful intubation!

This would have been the happy ending to a challenging situation, unfortunately it was not! Just before the cleft was repaired, the surgeon packed the throat with a wet gauze and then proceeded with the surgery. At the end of the good work, she inspected for any obvious bleeders, and got the conviction that all was dry and pink. The anesthetic gases had been turned low except for oxygen, the boy was breathing spontaneously without any struggle. She then decided to pull out the packed gauze. To my horror the tube too got pulled out!!

The boy, who was breathing spontaneously, started to cough after the tube's accidental removal. Remember! with the repair, no access to the airway was now possible!! We turned the patient on his side, sucked secretions a few times, and by the grace of God, the child stabilized. He was shifted to the ward after he awakened up fully. Six months later, he came for a checkup by another team in robust health. To our good luck, no further surgery was deemed necessary.