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AMITRAZ POISONING : A CASE REPORT OF UNUSUAL PESTICIDE POISONING

General Medicine

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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 six hours of poison ingestion with unconsciousness and persistent bradycardia, the most frequently reported symptoms associated with this type of poisoning. The patient then developed respiratory depression for which endotracheal intubation was done and was managed with symptomatic treatment, her condition started improving after 14 hours, she was then gradually weaned off and extubated, She was discharged in good health with no residual disease after 3 days.

KEYWORDS

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 16 years old female, ingested unknown amount of amitraz poison in a suicidal attempt at afternoon and was admitted to the emergency department almost after 6 hours after being treated primarily at peripheral health center . According to the relatives, patient ingested some unknown amount of Amitraz solution in the farm after which she came home and felt drowsy and informed relatives about the incident. An empty bottle of pesticide amitraz 12.5% in 10 ml formulation was found by the relatives. Patient was immediately taken to private hospital where she was given IV fluids and supplemental oxygen and was referred to our hospital for further management. On examination, her heart rate was 52 beats/min, BP 100/76 mmHg, respiratory rate 12-14/ min and oxygen saturation (SpO2) 98% on 6L/min of oxygen supplied via facemask, the patient was unconscious with a Glasgow coma scale of 7/15. Pupils were bilaterally semidilated and sluggishly reacting to light. Abdomen was soft on palpation and bowel sounds were normal on auscultation. Blood glucose levels was 193 mg/dl and ABG(Arterial blood gas) analysis revealed respiratory acidosis with a pH of 7.32 with PaCO2 of 59 mmHg. Patient was intubated and kept on ventilatory support using volume controlled ventilation. Activated charcoal powder were administered via nasogastric tube and gastric lavage was performed. 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. which improved progressively in serial analysis after intubation.

As no specific antidote for amitraz poisoning exists, symptomatic treatment with antacids, multivitamins 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.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 14 hours of intensive care. Respiratory acidosis improved on serial ABG analysis and the patient was weaned from ventilator, extubated the next day. The patient improved and was shifted to the ward on the next day. She was discharged from hospital after 3 days in good health without residual disease.

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]

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 her 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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