Amitraz Poisoning; A case study

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ABSTRACT

Amitraz, an insecticide/acaricide of the formamidine pesticides group, is a α2 adrenergic agonist and of the amine chemical family generally used to control animal ectoparasites. Poisoning due to amitraz is rare and characterized by central nervous system and respiratory depression, bradycardia, hypotension, hypothermia, hyperglycemia, nausea and vomiting. Few cases of intoxications in human beings due to this pesticide have been published in the literature. However, a clear and specific treatment protocol does not exist and this makes the successful managements of this poisoning (presented in the case reports) a probable useful guide for clinical practitioners in other poison centers. Management of amitraz poisoning is still considered to be supportive and symptomatic. We present a case of amitraz poisoning who successfully managed by supportive treatments in a 20 years old female.

Keywords: Amitraz; Bradycardia; Miosis; Central nervous system

Amitraz, a triazapentadiene compound and a member of the amine chemical family is a formamidine pesticides used worldwide. It is used as an insecticide/acaricide to control animal ectoparasites [1-23]. Commercial formulations of amitraz generally contain 12.5-20% of the drug in organic solvents, especially xylene, which is itself used in paints, cleaners, and glues [4]. Amitraz is a α2-adrenergic agonist stimulating α2 adrenergic receptors in the central nervous system (CNS) and both α1 and α2 adrenergic receptors in the periphery. It also inhibits monoamine oxidase (MAO) enzyme activity and prostaglandin E2 synthesis [5].

Poisoning occurs through oral, inhalational (the most potential), and dermal routes and is accompanied by numerous signs and symptoms varying from CNS depression (drowsiness, coma, and convulsion), to miosis, or rarely, mydriasis, respiratory depression, bradycardia, hypotension, hypertension, hypothermia or fever, hyperglycemia, polyuria, vomiting, decreased gastrointestinal motility, and intestinal distension [4]. Adverse reactions and side effects have been reported in animals exposed to the product; however, only few human intoxication cases have been reported in the literature. We present a young female patient with amitraz poisoning who was conservatively managed in intensive care unit (ICU) for 36 hours and experienced complete recovery.

CASE STUDY

A 20-year-old female referred to L.G. Hospital in Ahmedabad, Gujarat, India after the ingestion of 2 to 3 full table spoons of amitraz chemical (10% solution) in a suicidal attempt. Her first symptoms had begun about one hour post ingestion and included nausea and dizziness, after which vomiting had ensued. Her family had immediately brought her to our center where gastric lavage with normal saline and administration of activated charcoal (1 g/kg) were performed. She was then admitted to ICU for further management.

At presentation, she was drowsy but followed the verbal commands. Her blood pressure, pulse rate, respiratory rate, and temperature were 126/80 mmHg, 90 bpm, 24/min, and 36.8°C, respectively. Analysis of blood gases showed PaO2 of 106.4, O2 saturation of 96%, pH of 7.40, PCO2 of 34.0, and HCO3⁺ of 21.6. Other lab tests were as follow: blood urea nitrogen: 13 mg/dL; creatinine: 0.80 mg/dL; sodium: 138.9 mEq/L;
Amitraz Poisoning; A case study

potassium: 4.48 mEq/L; alanine transaminase: 15.7; bradycardia by stimulating the dorsal motor nucleus of the vagal nerve. It has been claimed that atropine increases heart rate and prevents amitraz-induced hyperglycemia: 2.2 mg/dL. In complete blood count, bradycardia in animals [2]. We administered atropine to our patient only once with adult dose. We believe were reported to be 6.72 g/dL, 8260/mm³, and atropine is effective in amitraz poisoning only when 4.58×10⁹/mm³, respectively. Chest X-Ray was normal. bradycardia exists.

One unit of packed cell was injected due to the low hemoglobin level. No special treatment was performed. The active metabolite inhibit insulin and stimulate glucagon except for gastric decontamination and cardiac and respiratory monitoring. Atropine (once; 4mg stat) was administered for the treatment of the patient’s and colleagues that reported hyperglycemia in nearly all the patients who were treated by administration of one dose of lidoine (1.5 mg/kg) and resolved in 24 hours. By the following day, she was completely conscious and aspartate transaminase was also detected in almost 20% of the patients which was not detected in our case. She completely recovered and was discharged from the hospital in the afternoon of the second day of admission.

**DISCUSSION**

Formamidines have been shown to have reversible toxic effects on both animals and human beings [4]. Since there are few reported human intoxications by this pesticide, the existing information about it is frequently built on animal studies. The median lethal dose in acute oral toxicity (LD₅₀) for the rats is 800 mg/kg [3,4].

The clinical signs and symptoms of this poisoning reported in previous reports include CNS depression, drowsiness, vomiting, miosis, bradycardia, hypotension, and hyperglycemia. The duration of CNS depression has ranged from a few hours to 24 h [4].

Bradycardia began within 30-150 minutes and resolved within 6-20 h in our case. Sedative effects of α₂-agonists are dose-dependent [1]. Coma, absence of light reflex, and respiratory failure are due to the ingestion of greater amounts of amitraz supporting its dose-dependent effects. Our patient was fully conscious after 24 h. This amitraz can result in respiratory depression, time has been reported to be 2-48 h in previous reports. Hypotension, bradycardia, hematuria, and edema were detected [10].

The effect of amitraz on α₁- and α₂-receptors causes hyperemia at the injection site which again are benign bradycardia [5]. In addition, literature reported and resolve without complications [12].

Hyperglycemia, hypotension, and bradycardia in amitraz in conclusion, basic approach to a patient with poisoning and attributed them to the alpha-2 adrenergic agonist action of amitraz [6]. In our case, absorption, and increasing elimination of the toxin. Bradycardia was also present accompanying with miosis. Medical management is essentially symptomatic and which developed during the course of hospitalization. supportive. No specific antidote exists [2].

Co-existence of bradycardia, miosis, and the respiratory depression leads to confusion with organophosphate or have not been evaluated, they are still considered in the controversy. Most studies, however, have reported central nervous systems. Increased intake may lead to atropine to resolve both miosis and bradycardia. Severe effects including coma and respiratory failure. Atropine is the first line therapy for the bradycardia. With supportive management, prognosis is good and resulted from vagal stimulation and atrioventricular the patients are discharged without any organ blocks. Alpha-2 adrenergic drugs can also cause dysfunction. This is similar to the results of Demirel et

REFERENCES


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