CASE REPORT

Amitraz Poisoning; A case study

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ABSTRACT

Amitraz, an insecticide/acaricide of the formamidine pesticides group, is a ß2 adrenergic agonist and of the amide 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 management 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 amide chemical family is a formamidine pesticides used worldwide. It is used as an insecticide/acaricide to control animal ectoparasites [1-3]. 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 ß2-adrenergic agonist stimulating ß2 adrenergic receptors in the central nervous system (CNS) and both ß1 and ß2 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 an intensive care unit (ICU) for 36 hours and experienced a 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;

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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 miosis. 

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One unit of packed cell was injected due to the low hemoglobin level. No special treatment was performed except for gastric decontamination and cardiac monitoring. Atropine (once; 4mg stat) was administered for the treatment of the patient’s transient bradycardia. During the ICU stay, the patient 64% of the cases [7]. Avsarogullari et al reported developed premature ventricular contractions (PVCs) and hyperglycemia and fast deterioration of the patients which were treated by administration of one dose of 0.48% (within 5 minutes after the ingestion of the toxin) that had been administered during the ICU stay, the patient’s creatinine, and the serum sodium and potassium do not change in this poisoning [2]. However, Kalyoncu and colleagues have reported hypotremia in their three cases [9]. This is while our patient did not show any evidence of electrolyte abnormalities. On the other hand, while analysis of blood gases was normal in our case, Kalyoncu and associates have reported respiratory alkalosis in two, respiratory acidosis in three, and metabolic acidosis in five cases [9].

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]. CNS symptoms 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 effect [10]. It is interesting to know that intravenous administration of amitraz can result in respiratory depression, time has been reported to be 2-48 h in previous reports. Hypotension, bradycardia, hematura, and edema are the effects. Our patient was fully conscious after 24 h. This amitraz can result in respiratory depression, although activated charcoal and cathartic effects are often used during organophosphate 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 effect of amitraz on α1- and α2-receptors causes hyperemia at the injection site which again are benign to α2-receptors in amitraz poisoning consists initial stabilization, reducing 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 poisoning. Although activated charcoal and cathartic effects have not been evaluated, they are still considered in the treatment protocol of these patients. Attention must be paid to the evaluation of the respiratory, cardiac, and 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 atroventricular 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 al. who reported in pr...

REFERENCES


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