CASE REPORT

Amitraz Poisoning; A case study

TEJAS PRAJAPATI1, NIMESH PATEL2, NASIM ZAMANI3, OMID MEHRPOUR4,5*

For author affiliations, see end of text.
Received April 11, 2012; Accepted May 28, 2012
This paper is available online at http://ijpt.iums.ac.ir

ABSTRACT

Amitraz, an insecticide/acaricide of the formamidine pesticides group, is a α2 adrenergic agonist and of the amidine 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 clinicians 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 amidine 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 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 a 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 verbal commands. Her blood pressure, pulse rate, respiratory rate, and temperature were 126/80 mmHg, 60/90 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


dipotassium: 4.48 mEq/L; alanine transaminase: 15.7 IU/L; blood glucose: 95 mg/dL (normal range, 70 to 110 mg/dL); PT: 14.7; INR: 1.03; calcium: 9.33 mg/dL; and increases heart rate and prevents amitraz-induced edema and dysfunction similar to a 54 α and cardiac function was normal. During the ICU stay, the patient was treated by respiratory monitoring. Atropine (once; 4mg/kg) was administered for the treatment of the patient’s bradycardia in animals [2]. We administered atropine to the patient one day with anticholinergic effects except for gastric decontamination and cardiac arrest, we did not detect hyperglycemia in our case. respiratory monitoring. Atropine (once; 4mg stat) was administered. This is in contrast with the previous study by Demirel et al [3] and colleagues that reported hyperglycemia in nearly 24% of their patients which was not detected in our case.

The clinical signs and symptoms of this poisoning reported in previous reports include CNS depression, drowsiness, vomiting, miosis, bradycardia, hypertension, 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 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 and 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 amitraz poisoning consists initial stabilization, reducing adrenergic agonist action of amitraz [6]. In our case, amitraz 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].

Coexistence of bradycardia, miosis, and the respiratory depression leads to confusion with organophosphate poisoning 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 controversial. 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 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.

REFERENCES


CURRENT AUTHOR ADDRESSES

Tejas Prajapati, Department of Forensic Medicine & Toxicology, AMC MET Medical College, L. G. Hospital, Ahmedabad, Gujarat, India.

Nimesh Patel, Consultant Physician, Sanjeevi Heart & Medical Hospital, Ahmedbad, Gujarat, India.

Nasim Zamani, Department of Clinical Toxicology, Loghman Hakim Hospital, Shahid Behesht University of Medical Sciences, Tehran, Iran.

Omid Mehrpour, Addiction Research Center (ADRC), Mashhad University of Medical Sciences; Medical Toxicology and Drug Abuse Research Center (MTDRC), Pasdaran Avenue, Birjand University of Medical Sciences (BUMS), Birjand 9713643138, Iran. E-mail: omid.mehrpour@yahoo.com.au (Corresponding author)