CASE REPORT

Amitraz Poisoning; A case study

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

For author affiliations, see end of text.

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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, hyperthermia, 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, hyperthermia, 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.

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;
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 and fast deterioration of the patients. On the other hand, while a 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].

We observed PVCs in our patient’s electrocardiogram (ECG) which recovered after 24 hours. In contrast, in a study by Aydin and coworkers, non-specific ST changes were detected in the ECGs of seven children with no history of cardiac disease who completely resolved in 24 h and PVCs were not detected [10]. Our case is interestingly very similar to a 54-year-old patient who had referred to Elinav and associates (with a clonidine-like syndrome) and managed in the same way [11]. Although not related to our patient, it is interesting to know that intravenous administration of 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].

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].

Coexistence of bradycardia, miosis, and the respiratory depression leads to confusion with organophosphate or 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 ativoventricular 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


CURRENT AUTHOR ADDRESSES

Prajapati et al.


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

Nimesh Patel, Consultant Physician, Sanjeevani Heart & Medical Hospital, Ahmedabad, Gujarat, India.

Nasim Zamani, Department of Clinical Toxicology, Loghman Hakim Hospital, Shahid Beheshti 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)