Amitraz Poisoning; A case study

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

1Department of Pharmacology and Therapeutics, School of Pharmacy, Tehran University of Medical Sciences, Tehran, Iran
2Department of Pediatrics, School of Medicine, Tehran University of Medical Sciences, Tehran, Iran
3Department of Pediatrics, Shahid Beheshti University of Medical Sciences, Tehran, Iran
4Department of Neurology, School of Medicine, Tehran University of Medical Sciences, Tehran, Iran
5Department of Neurology, School of Medicine, Shahid Beheshti University of Medical Sciences, Tehran, Iran

For author affiliations, see end of text.

Received April 11, 2012; Accepted May 28, 2012

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

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

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]. CNS symptoms began within 30-150 minutes and resolved within 6-20 hours in our case. Sedative effects of α₂-agonists are dose-dependent [1]. Conna, absence of light reflex, and respiratory failure are due to the ingestion of greater amounts of amitraz supporting its dose-dependent effect [1]. 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, hematuria, and edema are hallmarks of amitraz poisoning consists initial stabilization, reducing the effect of amitraz on α₁- and α₂-receptors causes hyperemia at the injection site which again are benign [5]. In addition, literature reported interest in terminating the toxin. Bradycardia was also present accompanying with miosis [6]. 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 differential diagnosis, both of which should be excluded. 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 bradycardia by stimulating the dorsal motor nucleus of the vagal nerve. It has been claimed that atropine use is also reported to have reversible effects on the heart [7]. Interestingly ve trio 6 p on a similar way [11]. Although not related to our patient, it is also possible that the administration of one dose of lidocaine (1.5 mg/kg) and resolved in 24 hours. By the next day, she was completely conscious and was able to answer to the questions. She completely of their patients which was not detected in our case. 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 amitraz can result in respiratory depression, time has been reported to be 2-48 h in previous reports. Hypotension, bradycardia, hematuria, and edema are hallmarks of amitraz poisoning consists initial stabilization, reducing the effect of amitraz on α₁- and α₂-receptors causes hyperemia at the injection site which again are benign [5]. In addition, literature reported interest in terminating the toxin. Bradycardia was also present accompanying with miosis [6]. Medical management is essentially symptomatic and which developed during the course of hospitalization. Supportive. No specific antidote exists [2].

**DISCUSSION**

Potassium: 4.48 mEq/L; alanine transaminase: 15.7 IU/L; blood glucose: 95 mg/dL (normal range, 70 to 110); the vaginal nerve. It has been claimed that atropine use is also reported to have reversible effects on the heart [7]. Interestingly ve trio 6 p on a similar way [11]. Although not related to our patient, it is also possible that the administration of one dose of lidocaine (1.5 mg/kg) and resolved in 24 hours. By the next day, she was completely conscious and was able to answer to the questions. She completely of their patients which was not detected in our case. 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 also possible that the administration of one dose of lidocaine (1.5 mg/kg) and resolved in 24 hours. By the next day, she was completely conscious and was able to answer to the questions. She completely of their patients which was not detected in our case. 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].

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, 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)