CASE REPORT

Amitraz Poisoning; A case study

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Received April 11, 2012; Accepted May 28, 2012

ABSTRACT

Amitraz, an insecticide/acaricide of the formamidine pesticides group, is a \( \alpha_2 \) adrenergic agonist and one 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 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 was conservatively managed in our center through 36 hours and experienced a complete recovery.

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 \( \alpha_2 \)-adrenergic agonist stimulating \( \alpha_2 \) adrenergic receptors in the central nervous system (CNS) and both \( \alpha_1 \) and \( \alpha_2 \) 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 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;
poisoning: 4.48 mEq/L; alanine transaminase: 15.7128; bradycardia by stimulating the dorsal motor nucleus of the vagal nerve. It has been claimed that atropine increases heart rate and prevents amitraz-induced hypotension, except for gastric decontamination and cardiac and respiratory monitoring. Atropine (once; 4mg stat) was 123. This is in contrast with the previous study by Demirel and colleagues that reported hyperglycemia in nearly all of the patients’ cases. 

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]. Coena, absence of light reflex, and respiratory failure are due to the ingestion of greater amounts of amitraz. 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 of amitraz on α1- and α2-receptors causes hyperemia at the injection site which again are benign. 

Co-existence of bradycardia, miosis, and the respiratory depression leads to confusion with organophosphate or nerve agent poisoning. Amitraz poisoning caused initial stabilization, reducing atropine agonist action of amitraz [6]. In our case, amitraz poisoning consists initial stabilization, reducing and resolve without complications [12]. 

In conclusion, basic approach to a patient with poisoning and attributed them to the α2-agonist agonist action of amitraz [6]. In our case, amitraz poisoning caused initial stabilization, reducing and resolve without complications [12]. 

Atropine is the first line therapy for the bradycardia. With supportive management, prognosis is good and resulted from vagal stimulation and late ventricular 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


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