CASE REPORT

Amitraz Poisoning; A case study

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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, 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

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 [1-3]. 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), tachymiosis, 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.

CASE STUDY

A 20-year-old female referred to L.G. Hospital in Ahmedabad, Gujarat, India after the ingestion of 2 to 3 fulltable 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, 60 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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65potassium: 4.48 mEq/L; alanine transaminase: 15.7; bradycardia by stimulating the dorsal motor nucleus of 66IU/L; blood glucose: 95 mg/dL (normal range, 70 to 110); the vagal nerve. It has been claimed that atropine 67mg/dL; PT: 14.7; INR: 1.03; calcium: 9.33 mg/dL; and increases heart rate and prevents amitraz-induced 68magnesium: 2.2 mg/dL. In complete blood count, bradycardia in animals [2]. We administered atropine to 69hemoglobin, white blood cell, and red blood cell count. Our patient only once with adult dose. We believe 70were reported to be 6.72 g/dL, 8260/mm³, and atropine is effective in amitraz poisoning only when 714.58×10⁹/mm³, respectively. Chest X-Ray was normal. Bradycardia exists.
72One unit of packed cell was injected due to the low. Although it has been declared that atropine and its 73hemoglobin level. No special treatment was performed. Active metabolite inhibit insulin and stimulate glucagon 74except for gastric decontamination and cardiac and secretions, we did not detect hyperglycemia in our case. 75respiratory monitoring. Atropine (once; 4mg stat) was. This is in contrast with the previous study by Demirel 76also administered for the treatment of the patient’s and colleagues that reported hyperglycemia in nearly 77transient bradycardia. During the ICU stay, the patient 64% of the cases [7]. Avasaragullari et al reported 78developed premature ventricular contractions (PVCs) hyperglycemia and fast deterioration of the patients 79which were treated by administration of one dose of (within 5 minutes after the ingestion of the toxin) that 80lidocaine (1.5 mg/kg) and resolved in 24 hours. By the 135 were both absent in our case [8]. Elevations of the 81following day, she was completely conscious and was aspartate transaminase was also detected in almost 20% 82able to answer to the questions. She completely of their patients which was not detected in our case. 83recovered and was discharged from the hospital in the afternoon of the second day of admission.
84
85Discussion
86Formamidines have been shown to have reversible 87toxic effects on both animals and human beings [4]. 88Since there are few reported human intoxications by this 89pesticide, the existing information about it is frequently 90built on animal studies. The median lethal dose in its 91acute oral toxicity (LD₅₀) for the rat is 800 mg/kg [3,4]. 92The clinical signs and symptoms of this poisoning 93reported in previous reports include CNS depression, 94drowsiness, vomiting, miosis, bradycardia, hypotension, 95and hyperglycemia. The duration of CNS depression has 96ranged from a few hours to 24 h [4]. CNS symptoms 97began within 30-150 minutes and resolved within 6-20 h. 98Our case is interestingly very similar to a 54-year- 99old patient who had referred to Elinav and associates 100(with a clonidine-like syndrome) and managed in the 101same way [11]. Although not related to our patient, It is 102interesting to know that intravenous administration of 103amitraz can result in respiratory depression, time has been reported to be 2-48 h in previous reports. Hypotension, bradycardia, hematuria, and edema are 104The effect of amitraz on α₁- and α₂-receptors causes hyperemia at the injection site which again are benign 105bradycardia [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 in amitraz poisoning consists initial stabilization, reducing 106adrenoceptor agonist action of amitraz [6]. In our case, amitraz was also present accompanying with miosis. Medical management is essentially symptomatic and 107which developed during the course of hospitalization. Supportive. No specific antidote exists [2]. 108
109Co-existence of bradycardia, miosis, and the respiratory depression leads to confusion with organophosphate or have not been evaluated, they are still considered in the 110opioid poisonings, both of which should be excluded. Treatment protocol of these patients. Attention must be 111Using atropine for treatment of bradycardia is paid to the evaluation of the respiratory, cardiac, and controversial. Most studies, however, have reported central nervous systems. Increased intake may lead to 112atropine to resolve both miosis and bradycardia. Severe effects including coma and respiratory failure. 113Atropine is the first line therapy for the bradycardia. With supportive management, prognosis is good and 114resulted from vagal stimulation and atroventricular the patients are discharged without any organ 115blocks. Alpha-2 adrenergic drugs can also cause dysfunction. This is similar to the results of Demirel et

REFERENCES


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