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 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 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 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 IU/L; blood glucose: 95 mg/dL (normal range, 70 to 110); the vagal nerve. It has been claimed that atropine 6 mg/dL; PT: 14.7; INR: 1.03; calcium: 9.33 mg/dL; and increases heart rate and prevents amitraz-induced 8 magnesium: 2.2 mg/dL. In complete blood count, bradycardia in animals [2]. We administered atropine to 10 hemoglobin, white blood cells, and red blood cell count of our patient only once with adult dose. We believe 12 were reported to be 6.72 g/dL, 8260/mm3, and atropine is effective in amitraz poisoning only when 14 7.458×109/mm3, respectively. Chest X-Ray was normal. Bradycardia exists. 16

One unit of packed cell was injected due to the low 18 Although it has been declared that amitraz and its 20 hemoglobin level. No special treatment was performed. Active metabolite inhibit insulin and stimulate glucagon 22 except for gastric decontamination and cardiac and respiratory monitoring. Atropine (once; 4mg stat) was 24 This is in contrast with the previous study by Demirel 26 also administered for the treatment of the patient’s and colleagues that reported hyperglycemia in our case. 28 transient bradycardia. During the ICU stay, the patient 30 64% of the cases [7]. Avsarogullari et al. reported 32 developed premature ventricular contractions (PVCs) 34 hyperglycemia and fast deterioration of the patients 36 which were treated by administration of one dose of 38 (within 5 minutes after the ingestion of the toxin) that 39 lidocaine (1.5 mg/kg) and resolved in 24 hours. By the next 41 day, she was completely conscious and 43 aspartate transaminase was also detected in almost 20% 45 of the patients which was not detected in our case.

recovered and was discharged from the hospital in 47 the afternoon of the second day of admission.

**Discussion**

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 92 built on animal studies. The median lethal dose in 94 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, 98 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 108 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 115 amounts of amitraz supporting its dose-dependent 117 interesting to know that intravenous administration of 119 effects. Our patient was fully conscious after 24 h. This 120 amitraz can result in respiratory depression, time has been reported to be 2-48 h in previous reports. 121 Hypotension, bradycardia, hematuria, and edema and The effect of amitraz on α1- and α2-receptors causes 124 hyperemia at the injection site which again are benign 125 bradycardia [5]. In addition, literature reported 127 resolve without complications [12].

Hyperglycemia, hypotension, and bradycardia in amitraz 131 In conclusion, basic approach to a patient with poisoning and attributed them to the alpha-2 133 amitraz poisoning consists initial stabilization, reducing 135 adrenoceptor agonist action of amitraz [6]. In our case, 137 absorption, and increasing elimination of the toxin. Bradycardia was also present accompanying with miosis 140 Medical management is essentially symptomatic and 142 which developed during the course of hospitalization. 144 supportive. No specific antidote exists [2].

Coexistence of bradycardia, miosis, and the respiratory 148 Although activated charcoal and cathartic effects 150 depression leads to confusion with organophosphate not have been evaluated, they are still considered in the 152 controversy. Most studies, however, have reported 154 central nervous systems. Increased intake may lead to 156 Atropine is the first line therapy for the bradycardia. 158 resulted from vagal stimulation and atrioventricular 159 blocks. Alpha-2 adrenergic drugs can also cause 161 dysfunction. This is similar to the results of Demirel et

REFERENCES


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