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 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;
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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 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 h 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 amounts of amitraz supporting its dose-dependent [5]. 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. Amitraz poisoning consists initial stabilization, reducing 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 receptor. Amitraz poisoning consists initial stabilization, reducing hypotension, bradycardia, hematuria, and edema and some cases with miosis [3]. 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 poisoning. There 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 and also cause hyperglycemia. The effect of amitraz on the α₁- and α₂-receptors causes hyperemia at the injection site which again are benign.

REFERENCES


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