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 was 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;
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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 can be used to treat bradycardia by stimulating the dorsal motor nucleus of the vagal nerve. By the HgptL.i

Kalyoncu and colleagues that reported hyperglycemia in nearly 60% of the cases [7]. Avsarogullari et al reported [2] that in the previous study by Demirel and colleagues which were both absent in our case [8]. Elevations of the following day, she was completely conscious and was able to answer to the questions. She completely recovered and was discharged from the hospital in the afternoon of the second day of admission.

Sodium and potassium do not change in this poisoning [10]. However, Kalyoncu and colleagues have reported hypokalemia in their three cases [9]. This is while our patient did not show any evidence of electrolyte abnormalities. On the other hand, while analysis of blood gases was normal in our case, Kalyoncu and colleagues have reported respiratory alkalosis in two, respiratory acidosis in three, and metabolic acidosis in five cases [9].

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 effects. Our patient was fully conscious after 24 h. This amitraz can result in respiratory depression, time has been reported to be 2-48 h in previous reports. Hypotension, bradycardia, hematuria, and edema and The effect of amitraz on α1- and α2-receptors causes hypotension and bradycardia in amitraz poisoning consists initial stabilization, reducing adrenergic action of amitraz [6]. In our case, absorption, and increasing elimination of the toxin. Bradycardia was also present accompanying with miosis [7]. Medical management is essentially symptomatic and which developed during the course of hospitalization. Supportive. No specific antidote exists [2].

Coexistence of bradycardia, miosis, and the respiratory depression leads to confusion with organophosphate or have not been evaluated, they are still considered in the treatment protocol of these patients. Attention must be paid to the evaluation of the respiratory, cardiac, and controversial. Most studies, however, have reported central nervous systems. Increased intake may lead to atropine to resolve both miosis and bradycardia. Severe effects including coma and respiratory failure. Atropine is the first line therapy for the bradycardia. With supportive management, prognosis is good and resulted from vagal stimulation and atrioventricular 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

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 [12]. It is interesting to know that intravenous administration of effects. Our patient was fully conscious after 24 h. This amitraz can result in respiratory depression, time has been reported to be 2-48 h in previous reports. Hypotension, bradycardia, hematuria, and edema and The effect of amitraz on α1- and α2-receptors causes hypotension and bradycardia in amitraz poisoning consists initial stabilization, reducing adrenergic action of amitraz [6]. In our case, absorption, and increasing elimination of the toxin. Bradycardia was also present accompanying with miosis [7]. Medical management is essentially symptomatic and which developed during the course of hospitalization. Supportive. No specific antidote exists [2].

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