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 conservatively managed in an intensive care unit (ICU) for 36 hours and experienced a complete recovery.

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;
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 result in respiratory depression, with a clonidine 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]. In addition, literature reported [13] and resolve without complications [12].

The effect of amitraz on α₁- and α₂-receptors causes hypotension at the injection site which again are benign bradycardia [5]. In conclusion, basic approach to a patient with poisoning and attributed them to the alpha-2 receptor. Amitraz poisoning consists initial stabilization, reducing adrenoceptor agonist action of amitraz [6]. In our case, 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 [9]. We also observed PVCs in our patient’s 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 [9]. We also observed PVCs in our patient’s 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 [9].

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 amitraz can result in respiratory depression, hypotension, bradycardia, hematura, and edema 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 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 atroventricular 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

REFERENCES


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