**CASE REPORT**

**Amitraz Poisoning; A case study**

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**ABSTRACT**

Amitraz, an insecticide/acaricide of the formamidine pesticides group, is a β₂ 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;
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65 potassium: 4.48 mEq/L; alanine transaminase: 15.7; bradycardia by stimulating the dorsal motor nucleus of 66 IVU/L; blood glucose: 95 mg/dL (normal range, 70 to 110); the vagal nerve. It has been claimed that atropine 67 (mg/dL); PT: 14.7; INR: 1.03; calcium: 9.33 mg/dL; and increases heart rate and prevents amitraz-induced 68 magnesium: 2.2 mg/dL. In complete blood count, bradycardia in animals [2]. We administered atropine to 69 hemoglobin, white blood cells, and red blood cell count of our patient only once with adult dose. We believe 70 were reported to be 6.72 g/dL, 8260/mm³, and atropine is effective in amitraz poisoning only when 71 4.58×10⁹/mm³, respectively. Chest X-Ray was normal. Bradycardia exists.

72 One unit of packed cell was injected due to the low. Although it has been declared that atropin and its 73 hemoglobin level. No special treatment was performed. Active metabolite inhibits insulin and stimulate glucagon 74 except for gastric decontamination and cardiac and secretion, we did not detect hyperglycemia in our case. 75 Respiratory monitoring. Atropine (once; 4mg stat) was [3]. This is in contrast with the previous study by Demirel 76 also administered for the treatment of the patient's transient bradycardia. During the ICU stay, the patient 64% of the cases [7]. Avsarogullari et al reported developed premature ventricular contractions (PVCs) [8]. Hyperglycemia and fast deterioration of the patients 77 which were treated by administration of one dose of 34-(within 5 minutes after the ingestion of the toxin) that 78 lidocaine (1.5 mg/kg) and resolved in 24 hours. By the 80 were both absent in our case [8]. Elevations of the following day, she was completely conscious and was aspartate transaminase was also detected in almost 20% able to answer to the questions. She completely of their patients which was not detected in our case. 79 recovered and was discharged from the hospital in 14. Normally, levels of BUN, creatinine, and the serum 80 the afternoon of the second day of admission.

81 DISCUSSION

82 Formamidines have been shown to have reversible toxic effects on both animals and human beings [4]. 83 Since there are few reported human intoxications by this pesticide, the existing information about it is frequently 84 built on animal studies. The median lethal dose in its acute oral toxicity (LD₅₀) for the rats is 800 mg/kg [3,4]. 85 The clinical signs and symptoms of this poisoning 86 reported in previous reports include CNS depression, 87 drowsiness, vomiting, miosis, bradycardia, hypotension, and hyperglycemia. The duration of CNS depression has 88 ranged from a few hours to 24 h [4]. CNS symptoms began within 30-150 minutes and resolved within 6-20 h. 89 Our case is interestingly very similar to a 54-year-old patient who had referred to Elimav 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 90 effects. Our patient was fully conscious after 24 h. This 91 amitraz can result in respiratory depression, 92 time has been reported to be 2-48 h in previous reports. Hypotension, bradycardia, hematura, and edema and 93 The effect of amitraz on α₁- and α₂-receptors causes hyperemia at the injection site which again are benign 94 bradycardia [5]. In addition, literature reported and resolve without complications [12].

95 Hypotension, hypotension, and bradycardia in amitraz. In conclusion, basic approach to a patient with poisoning and attributed them to the alpha-2 and amitraz poisoning consists initial stabilization, reducing 96 adrenoceptor agonist action of amitraz [6]. In our case, absorption, and increasing elimination of the toxin. 97 Bradycardia was also present accompanying with miosis. Medical management is essentially symptomatic and 98 which developed during the course of hospitalization. Supportive. No specific antidote exists [2].

99 Co-existence of bradycardia, miosis, and the respiratory 169 Although activated charcoal and cathartic effects 100 depression leads to confusion with organophosphate or have not been evaluated, they are still considered in the 101 opioid poisonings, both of which should be excluded. Treatment protocol of these patients. Attention must be 102 Using 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 103 atropine to resolve both miosis and bradycardia. Severe effects including coma and respiratory failure. 104 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

REFERENCES


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