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

Amitraz, a triazapentadiene compound and a
member of the amidine chemical family is a
formamidine pesticides used worldwide. It is used as an
insecticide/acaricide to control animal ectoparasites [1-
23]. Commercial formulations of amitraz generally
contain 12.5-20% of the drug in organic solvents,
especially xylene, which is itself used in paints,
cleaners, and glues [4]. Amitraz is a α2-adrenergic
agonist stimulating α2 adrenergic receptors in the central
nervous system (CNS) and both α1 and α2 adrenergic
receptors in the periphery. It also inhibits monoamine
oxidase (MAO) enzyme activity and prostaglandin E2
synthesis [5].

Poisoning occurs through oral, inhalational (the most
potential), and dermal routes and is accompanied by
numerous signs and symptoms varying from CNS
depression (drowsiness, coma, and convulsion), to
miosis, or rarely, mydriasis, respiratory depression,
bradycardia, hypotension, hypertension, hypothermia or
fever, hyperglycemia, polyuria, vomiting, decreased
gastrointestinal motility, and intestinal distension [4].
Adverse reactions and side effects have been reported in
animals exposed to the product; however, only few
human intoxication cases have been reported in the
literature. We present a young female patient with

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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potassium: 4.48 mEq/L; alanine transaminase: 15.7; bradycardia by stimulating the dorsal motor nucleus of the vagal nerve. It has been claimed that atropine increases heart rate and prevents amitraz-induced hypotension, bradycardia in animals [2]. We administered atropine to 107 one patient only once with adult dose. We believe it was reported to be 6.72 g/dL, 8260/mm³, and atropine is effective in amitraz poisoning only when urine creatinine clearance is > 80 mEq/L; potassium: 5.1 mEq/L; PT: 14.7; INR: 1.03; calcium: 9.33 mg/dL; and elevations of the BUN and PVCs were not detected in our case [4, 5, 6]. As shown in Table 1, in a 6-month study by Demirel et al. we did not detect hyperglycemia in our case. Atrasarguli et al. have reported 107 developed premature ventricular contractions (PVCs), hyperglycemia and fast deterioration of the patients which were treated by administration of one dose of PVCs (within 5 minutes after the ingestion of the toxin) that lidoicaine (1.5 mg/kg) and resolved in 24 hours. By the end of the fifth day of admission, she was completely conscious and aspartate transaminase was also detected in almost 20% of her patients which was not detected in our case.

One unit of packed cell was injected due to the low hemoglobin, white blood cells, and red blood cell count in our case. Although it has been declared that amitraz and its metabolites are very persistent in human blood [7], and we found a normal range, 70 to 110, respectively. Chest X-Ray was normal. Bradycardia exists.

One of the most severe effects including coma and respiratory failure. Usually, levels of BUN, creatinine, and the serum sodium and potassium do not change in this poisoning [2]. However, Kalyoncu and colleagues have reported hyponatremia 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 associates 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 hyperemia at the injection site which again are benign effects [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-2A amitraz poisoning consists initial stabilization, reducing the adrenergic agonist action of amitraz [6]. In our case, absorption, and increasing elimination of the toxin. Bradycardia was also present accompanying with miosis. 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 bradycardia and also cause dysfunction. This is similar to the results of Demirel et al.

REFERENCES


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