Amitraz Poisoning: The (Un) Common Poisoning

William Wilson, Shakuntala Murty
Department of Emergency Medicine, St. John’s Medical College, Bengaluru, Karnataka, India

Abstract

Pesticide poisoning is always a clinical conundrum for the emergency physician (EP), the complexity of which increases when the pesticide has no antidote! Over the past decade, there has been a sharp increase in cases of Amitraz poisoning, a pesticide routinely used in veterinary medicine, available without a prescription. The usual presentation includes bradycardia, hypotension, poor sensorium, and miosis. In the absence of accurate history, these clinical features can be confused with the cholinergic toxidrome of organophosphorus poisoning. There is a dearth of literature regarding the presentation and protocols for the management of Amitraz poisoning with data mostly based on animal studies and pediatric case reports. Currently, the available medical literature in the form of case reports and case series form an invaluable source of information to the EP to formulate a working diagnosis and methodical approach to this pesticide. Here, we present two case reports highlighting the characteristic clinical features and bringing to light how an organized approach to the toxin can give satisfactory results.

Keywords: Amitraz, pesticide poisoning, toxicology

INTRODUCTION

Amitraz, 1,5-di-(2,4-dimethylphenyl)-3-methyl-1,3,5-triazapenta-1,4-diene, is an acaricide/insecticide of formamidine pesticides, being used worldwide to control pests in animals.[1] It is commonly indicated for the treatment of generalized demodicosis in dogs, cattle, and sheep as an ectoparasite and to control psylla infestations of pears and mites on cotton crops.[2]

A number of case reports of poisoning with Amitraz have been reported from across the world with most cases being reported from the West,[3] few from India as well;[4,5] however, majority remain in the pediatric age group.[6] With no preventable vaccine, cure, or reliable diagnostics, the threat is real.

Amitraz is a pharmacologically active compound which has a2-agonist actions. The stimulation effect of a2-receptors is in part responsible for neurotoxic and proconvulsant effects.[2] It also inhibits monoamine oxidase enzyme activity and prostaglandin E2 synthesis.[7]

Amitraz is available without a physicians’ prescription across the world in all leading chemist shops with a variety of trade names, and in India, the common brand name is RIDD.[5]

Clinical presentation of the diseases can be confusing and can easily be misdiagnosed as organophosphorus poisoning.[8]

With rising number of cases, and to create awareness, we report two cases of suicidal attempts using Amitraz with the focus on clinical features and a structured management protocol.

CASE REPORTS

Case 1

A 23-year-old male was rushed into ED after consumption of unknown pesticide along with alcohol. The patient had vomited multiple times and had diffuse abdominal pain.

On arrival, the patient was in altered mental status with a Glasgow Coma Scale (GCS) of E2V4M4 pulse106, blood pressure (BP) 154/10, RR 38. The abdomen was soft with minimal tenderness, and bilateral air entry was equal. Pupils were sluggish and 2 mm reactive.

Investigations included blood sugars of 154 and the arterial blood gas (ABG) which showed metabolic acidosis with a pH of 7.33.

Address for correspondence: Dr. William Wilson, C/o Dr. Shakuntala Murty, Department of Emergency Medicine, John Nagar, Koramangala, Sarjapur Main Road, Bengaluru - 560 003, Karnataka, India. E-mail: drwillwilson@gmail.com

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The patient was progressively becoming drowsy, and in view of impending respiratory failure, the patient was intubated and put on mechanical ventilation which was followed by a gastric lavage. As identity and details of the actual pesticide consumed were unknown and presence of the cholinergic toxidrome (bradycardia, miosis, respiratory depression), organophosphorus poisoning was considered and the patient was started on atropine and pralidoxime infusion.

The patient attenders soon returned with the empty bottle of the pesticide which was Amitraz 12.5% in 10 ml formulation. Pralidoxime and atropine infusions were stopped and the patient was shifted to the Intensive Care Unit (ICU) for further management.

In the ICU, the patient was hemodynamically stable but showed persistent bradycardia. The liver function tests, renal function tests (blood urea, creatinine, serum electrolytes) and other blood counts and coagulation parameters were within normal limits. Supportive treatment was given to the patient including an antibiotic for the nonhomogeneous opacity on the X-ray, and within 24 h, the patient was weaned off the ventilator and extubated. On progressive recuperation, the patient was shifted to the general ward, and on the 4th day, he was discharged in good health.

**Case 2**
A 33-year-old male with alleged history of consumption of Amitraz was brought to the ED within 5 h of consumption of 15–20 ml of Amitraz (12.5% formulation).

With initial complaints of multiple episodes of vomiting, the patient received a gastric lavage in a primary health center and was referred to our ED for further management. On arrival, the patient was comatose with a GCS E1V1M2 pulse 102/min, BP 110/70, RR 8–10. On systemic examination, per abdomen and respiratory system were found to be normal. Pupils were 2 mm sluggishly reactive to light.

Investigations included blood sugars of 192 and the ABG which showed metabolic acidosis with a pH of 7.23.

The patient was intubated, in view of impending respiratory arrest, and was shifted to the ICU. The patient was mechanically ventilated and remained hemodynamically stable. The investigations including renal function tests (blood urea, creatinine, electrolytes), liver function tests, and blood counts were normal.

With an uneventful stay in the ICU, the patient was extubated within 36 h and was discharged from the ward on day 5.

The details regarding the two cases have been compared and tabulated in Table 1.

**Conclusion**
The toxic effects of Amitraz on animals are well reported; however, not much data are available with respect to humans. Studies on animals with Amitraz have shown a variety of characteristics (effects) such as bradycardia, hypotension,
decreased spontaneous activity, and death caused by respiratory depression. The clinical features and fatal results were all dose dependent, with the acute oral lethal dose 50 for rats was found to be 800 mg/kg body weight.\[^{[9]}\]

Previous reported studies on human have shown clinical features of bradycardia, hypotension miosis/mydriasis, and drowsiness along with investigations showing acidosis on the blood gas and hyperglycemia.

Our case studies reported near similar clinical and investigatory picture. However, the patients, at no point, were hypotensive and did not require inotropic support for the same. The difference in clinical features can be attributed to the dose of consumption and the dose-dependent features.

Organophosphorus poisoning and Amitraz toxicity overlap in their presentation with the presence of bradycardia miosis and respiratory depression. Various studies have documented the use of atropine as the initial treatment modality.\[^{[8,10]}\] The subtle differences in clinical features include the absence of a hypersecretory state (salivation, lacrimation, emesis, defecation, and bronchorrhea) and the presence of hypothermia, along with laboratory tests such as hyperglycemia and a normal serum cholinesterase level suggested an alternate possibility.

Both our patients had consumed Amitraz as a suicide attempt, and current literature in adults points to deliberate self-harm as the major reason of consumption with insufficient data regarding predisposing conditions such as depression or dependence.

In the ICU, the patients were hemodynamically stable but had persistent bradycardia which did not warrant the use of atropine. Amitraz does not have a known antidote, and the management revolves around symptomatic and supportive care.

With increasing number of cases, the emergency physician needs to be aware of the lethal effects of this readily available pesticide, identify the constellation of signs and symptoms with focused investigation and treatment to reduce the morbidity caused by this pesticide.

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**Conflicts of interest**

There are no conflicts of interest.

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