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Case Report

Amitraz poisoning: a rare case of suicidal poisoning from Malwa region of Punjab and role of early gastric lavage in preventing complications

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ABSTRACT

Amitraz, a widely-used pesticide for both crops and animals globally, effectively controls pests such as ticks and mites in livestock, as well as red spider mites in fruit crops. Its formulation includes triazapenta 1,4 diene from the formamidine family. Despite its widespread use, only a few cases of poisoning have been reported worldwide, mostly stemming from accidental exposure. We present a case involving a 28-year-old woman from the Malwa region of Punjab, who intentionally ingested 30 ml of amitraz solution (12.5% w/v) following a marital dispute, resulting in a suicide attempt. The patient fully recovered within 72 hours and was discharged with resolved symptoms on the third day. Amitraz poisoning typically occurs due to accidental ingestion. In our case, the patient experienced central nervous system depression accompanied by bradycardia (Figure 1), necessitating treatment with atropine. These effects persisted for 72 hours. The clinical manifestations observed in our patient align with the mechanism of action on alpha 1 and alpha 2 receptors, with dizziness and bradycardia being prominent features. Respiratory involvement is also possible, while liver and renal effects are rare. Early gastric lavage is recommended for managing amitraz poisoning.

Keywords: Amitraz poisoning, Early gastric lavage, Bradycardia, Suicidal

INTRODUCTION

Amitraz, a widely-used pesticide utilized on crops and animals globally, effectively combats pests such as ticks and mites in livestock, as well as red spider mites in fruit crops. It contains triazapenta-1,4 diene from the formamidine family.[1,2] Clinical manifestations in humans encompass CNS effects like depression, changes in pupil size, respiratory and cardiovascular depression (including bradycardia and hypotension), variations in body temperature, elevated blood sugar levels, increased urination, and decreased gastrointestinal motility. Its mechanism of action involves activity on alpha receptors, both centrally on alpha 1 receptors and peripherally on alpha 1 and 2 receptors[3]. Poisoning can occur through oral, dermal, or inhalational routes, with oral ingestion being the most common and leading to more severe symptoms. The minimum reported toxic dose is 3.57 mg/kg, and cases of poisoning ranging from 2 to 50 ml have been documented. Due to the limited number of reported cases, there is a lack of general awareness among physicians, particularly in Asian countries.[4]

This review focuses on case reports of amitraz poisoning, discussing its effects, management strategies, and potential mechanisms of action. Management primarily involves supportive care, with early gastric lavage playing a crucial role in preventing complications. Various alpha 2 agonists have been attempted in animals to reverse the effects of amitraz, yet no specific antidote

has been tested in humans.[2,5] Accidental ingestion represents the most common mode of presentation, underscoring the importance of precautionary measures.[6]

CASE REPORT

We present a case involving a 28-year-old female residing in the Malwa region of Punjab, who ingested 30 ml of amitraz solution (12.5% w/v) following a marital dispute, leading to a suicidal act. The patient, weighing 66 kg and measuring 165 cm, experienced amitraz poisoning at a rate of 56.8 mg/kg. Within 15 minutes of ingestion, the patient felt dizzy, followed by nausea and discomfort upon swallowing.

Upon admission to the hospital two hours after ingestion, the patient scored 13 out of 15 on the Glasgow coma scale (E3V4M6), with equal and bilaterally reactive pupils and normal deep tendon reflexes. The patient presented with bradycardia, with a heart rate of 37/min, blood pressure of 114/60 mmHg, and a respiratory rate of 14/min.

Upon presentation, gastric lavage was performed, followed by intravenous fluid bolus and maintenance doses. The patient's bradycardia persisted for 48 hours, and she remained drowsy for 54 hours. Although experiencing nausea, the patient did not vomit and had not passed stools for two days. Respiratory depression, convulsions, and hypothermia did not occur, and urine output remained normal.

Table 1: Routine investigations of patient during hospital stay.

Type	Value	Unit
2024-02-19		
Calcium	8.7	mg/dl
Chloride	119	mEq/l
Creatinine	0.38	mg/dl
Hb (haemoglobin)	10	g/dl
WBC (Total white blood cells)	6500	cells /mm^3
Platelet count	104000	cells/mm^3
Potassium	3.8	mEq/l
Serum urea	16	mg/dl
Sodium	138	mEq/l

Electrocardiogram (ECG) revealed sinus bradycardia with a normal QT interval, and blood sugar levels were consistently within normal limits. Throughout the patient's stay, complete blood counts, liver function tests, renal function tests, and serum electrolytes were monitored (Table 1), all showing normal results. Urine routine and serum creatinine remained normal, and arterial blood gas (ABG) analysis indicated a pH of 7.41, HCO3 of 22, and PCO2 of 38 mmHg, with no signs of hypoxia.

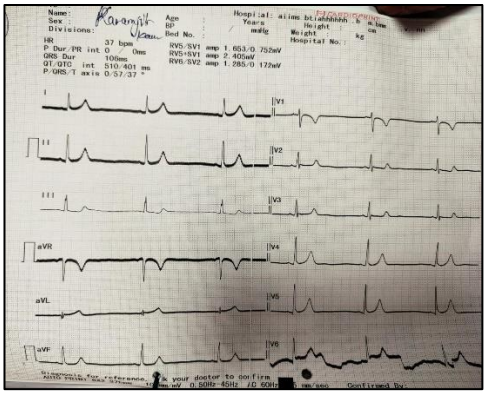

Figure 1: ECG at presentation showing bradycardia with heart rate 37 bpm.

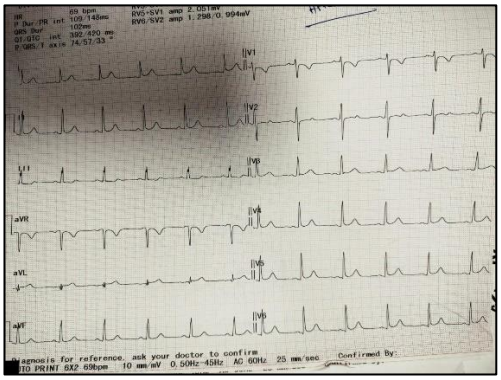

Figure 2: ECG at discharge showing normalisation of bradycardia with Heart rate of 69 bpm.

Bradycardia was managed with two doses of 0.4 mg atropine upon presentation, followed by a dosage of 0.4 mg three times a day for two days. The patient fully recovered after 72 hours and was discharged on the third day.

DISCUSSION

Amitraz acts as an agonist on alpha 2 adrenergic receptors, stimulating these receptors in the central nervous system (CNS) and both alpha 1 and 2 receptors in the periphery, while also inhibiting the activity of monoamine oxidase and prostaglandin E2 synthesis.[3] Its effects in animals resemble those of clonidine and can often be mistaken for organophosphate poisoning or carbamate toxicity. The acute oral median lethal dose (LD50) in rats is 523-800 mg/kg and 1600 mg/kg in mice, with two human deaths reported, one involving the ingestion of 6 grams of amitraz. The minimum toxic dose is reported to be 3.57 mg/kg, with our patient ingesting 3750 mg (56.8 mg/kg).[4] Clinical manifestations typically include CNS depression, respiratory depression, and cardiovascular effects.[6] The onset of action ranges from 30 to 180 minutes following ingestion, with CNS depression observed within 30 to 90 minutes and resolving in 8 to 14 hours.[7] Our patient experienced dizziness after 15 minutes of ingestion, which is relatively

rapid. Most patients recover within 48 hours, with our patient recovering in 72 hours and being discharged. Dizziness, a common complaint in our case, is likely due to the agonist action on alpha 2 receptors.[5] Miosis with loss of light reflex has also been observed, although our patient had normal pupil size and bilateral reactivity to light. Action on alpha 1 and 2 receptors can cause bradycardia and hypotension, with some patients requiring atropine and intravenous fluids for resuscitation. Respiratory depression is common in amitraz poisoning, but our patient's respiratory system remained intact. Serum electrolyte levels and blood urea nitrogen are typically within normal limits in most cases. Amitraz, along with its active metabolite BTS 27271, acts on alpha 2 adrenergic receptors in pancreatic islets, inhibiting insulin and stimulating glucagon secretion. It has also been reported to increase urination, likely due to alpha 2 receptor stimulation causing decreased antidiuretic hormone (ADH) and renin secretion.[8]

Amitraz is known to inhibit prostaglandin E2 synthesis, explaining its antipyretic and anti-inflammatory activity in vivo.[2,3] There is no specific antidote for amitraz poisoning, and management is supportive, involving monitoring and evaluation of respiratory, cardiac, and CNS systems. Gastric lavage has been attempted in many cases, including ours, with atropine sulfate used to increase heart rate and prevent amitraz-induced bradycardia.[6] Yohimbine, an alpha 2 antagonist, has been shown to prevent various effects of amitraz poisoning.[3]

The American association of poison centers (AAPC) and the European association of poison centers and clinical toxicologists (EAPCCT) recommend against routine gastric lavage, suggesting it only be performed if the patient presents within one hour of ingestion and if a potentially lethal ingestion has occurred.[9] In our case, due to early presentation and an unclear dose, gastric lavage was performed after excluding contraindications, and the patient was managed symptomatically.[2] Although activated charcoal could have been considered, it was not administered. We suggest the use of gastric lavage in cases of amitraz poisoning where patients present early and a large amount of amitraz has been ingested. Atropine is effective in controlling bradycardia in patients with amitraz poisoning, and we used it as needed to maintain a pulse rate above 50/minute (Figure 2). The case fatality rate of amitraz poisoning is 1.9%, and our patient was discharged asymptomatic on the third day.[10]

CONCLUSION

Amitraz poisoning is usually accidental ingestion. we present a young female from Malwa region of Punjab who developed CNS depression with bradycardia requiring atropine. Effect lasted for 72 hours. Clinical manifestations in our patient can be explained by mechanism of action on alpha 1 and alpha 2 receptors. Dizziness and bradycardia are main clinical features of amitraz poisoning, were predominant feature in our patient. Respiratory system can also be involved. Liver and renal system are rarely affected which was true for our patient.[6] We recommend early gastric lavage in amitraz poisoning for management with risks excluded. Mortality after amitraz is very low with case fatality rate being 1.9%. Our patient was discharged asymptomatic at day 3 of presentation.

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