Neurological effects of an unusual insecticide poison: Amitraz

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ABSTRACT

Amitraz is a triazapentadiene compound belonging to amidine family. As an insecticide and acaricide, it has been used to control red spider mites, scale insects, aphids, leaf worms, whitefly, bollworms, and pear psylla on Oregon pear crops. Poisoning is secondary to accidental or suicidal inhalation and ingestion of the compound. The toxicity profile is mostly in the form of alteration of the nervous system resulting in various clinical manifestations. We describe a case report of amitraz poisoning presenting with coma.

Keywords: Amitraz, insecticide poison, neurological manifestations, outcome

Background

Amitraz, 1,5 di-(2,4-dimethylphenyl)-3- methyl-1,3,5-tri-aza-penta-1,4 diene, is a member of amidine chemical family. This compound acts through stimulation of central \( \alpha_2 \) adrenergic receptors and peripheral \( \alpha_1 \) and \( \alpha_2 \) receptors. Case reports of the same are rare. Understanding of the spectrum of clinical manifestations has been based on case reports. Most of the case reports have been based on accidental consumption of the same by children.

Case Report

A 23-year-old male was brought to the emergency department with a history of consumption of 15 ml of amitraz poison in suicidal intent after 3 h of consumption. He had multiple episodes of vomiting immediately after consumption. Following which, he became drowsy and later became unconscious. There were no history of seizures, history of illicit drug usage, and no medical or psychiatric comorbidities.

On examination, the patient was unconscious with a Glasgow Coma Scale of 2T/15. His vitals were normal. On examination of the central nervous system (CNS), pupils were dilated (5 mm) and nonreactive to light bilaterally. However, his oculocephalic reflex was present. Other systemic examination was normal. The patient was intubated because of low sensorium and Type 2 respiratory failure, and he was transferred to the Intensive Care Unit.

Baseline blood investigations were normal as shown in Table 1. Electrocardiogram (ECG) showed sinus rhythm, and his chest X-ray was normal. The patient was managed conservatively. Meticulous monitoring of fluid, electrolytes, and CNS status was continued, with which his sensorium slowly improved after 24 h, and he was extubated within 48 h. Subsequently, he was transferred to ward and discharged after appropriate psychiatric consultation.

Discussion

The incidence of amitraz poisoning is increasing worldwide because of its easy accessibility and availability. Route of exposure has been mostly ingestional, transdermal, and...
Clinical features include 10,400. α-agonists are dose dependent. 

Results 3.08 360.

Use of activated charcoal and cathartics can be considered. It also inhibits monoamine oxidase enzyme activity and prostaglandin E2 synthesis. Clinical features include CNS depression, respiratory depression, bradycardia, miosis, hypotension, polyuria (inhibition of antidiuretic hormone and renin), hyperthermia, hyperglycemia, intestinal distension, vomiting, and rarely mydriasis. The most common manifestation of this poisoning that was observed in many studies was CNS depression within 30–180 min. Our patient had deterioration of sensorium progressing to coma within approximately 50 min after consumption of the poison. The sedative effects of α2-agonists are dose dependent. Presence of coma, absence of light reflex, and respiratory failure in our patient are probably due to the ingestion of a greater amount of amitraz, which supports its dose-dependent effects on the body systems. The time required for resolution of CNS depression was 2–48 h in the previous case reports which is similar to our patient, whose sensorium improved after 24 h of ingestion.

Table 1: Baseline laboratory investigations

| Investigations | Results |
|---------------|---------|
| Hemoglobin (g%) | 17.1 |
| White blood cell total count | 10,400 |
| Platelet count (lakhs) | 3.08 |
| Creatinine (mg%) | 1.24 |
| Urea | 17 |
| Sodium (mmol/L) | 146 |
| Potassium (mmol/L) | 4.3 |
| LDH | 360 |
| Total bilirubin (mg/dl) | 1.5 |
| Direct bilirubin (mg%) | 0.4 |
| Total protein (g/dl) | 6.9 |
|Albumin (g/dl) | 4.4 |
| AST (U/L) | 48 |
| ALT (U/L) | 16 |
| Alkaline phosphatase (U/L) | 89 |

LDH: Lactate dehydrogenase; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase

Amitraz stimulates central adrenergic α2 receptors and peripheral α1 and α2 receptors. It also inhibits monoamine oxidase enzyme activity and prostaglandin E2 synthesis. Clinical features include CNS depression, respiratory depression, bradycardia, miosis, hypotension, polyuria (inhibition of antidiuretic hormone and renin), hyperthermia, hyperglycemia, intestinal distension, vomiting, and rarely mydriasis. The most common manifestation of this poisoning that was observed in many studies was CNS depression within 30–180 min. Our patient had deterioration of sensorium progressing to coma within approximately 50 min after consumption of the poison. The sedative effects of α2-agonists are dose dependent. Presence of coma, absence of light reflex, and respiratory failure in our patient are probably due to the ingestion of a greater amount of amitraz, which supports its dose-dependent effects on the body systems. The time required for resolution of CNS depression was 2–48 h in the previous case reports which is similar to our patient, whose sensorium improved after 24 h of ingestion.

Brady卡rdia and miosis were due to stimulation of peripheral α1 and α2 adrenergic receptors. The presence of the above features can also be seen in opioid and organophosphorus poisoning. Moreover, it has to be considered in patients where the details of the poison are unknown. Our patient presented with mydriasis which is a rare presentation. Moreover, this delayed mydriasis has been reported earlier. Atropine is effective in treating symptomatic bradycardia. Amitraz and its metabolite inhibit insulin release and stimulate glucagon secretion, thereby leading to hyperglycemia. Prostaglandin E2 inhibition by amitraz leads to hyperthermia.

Serum sodium, potassium, blood urea nitrogen, and creatinine levels have been reported to be normal, but Kalyoncu et al. reported hyponatremia, respiratory alkalosis, respiratory acidosis, and metabolic acidosis in patients. Nonspecific ST changes are also reported in the ECGs of children with complete recovery in 24 h.

In the absence of an antidote, management is purely supportive and symptomatic. Use of activated charcoal and cathartics can be considered.

Without early initiation of supportive care, mortality is higher. With supportive treatment, chance of recovery is optimal. To prevent this trend of rising amitraz poisoning, steps need to be taken. Timely intervention of regulatory authorities and national poison control centers by promoting awareness among general public and health-care workers might go a long way in solving the problem.

Conclusion

Cases of amitraz poisoning are on the rise. Toxidrome secondary to amitraz is mostly in the form of various CNS manifestations along with respiratory depression. Management is mostly supportive. Prognosis is good with early recognition and intervention.

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

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