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Clinico-socio-epidemiological profile of amitraz poisoning: a case series

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ABSTRACT

Background: Amitraz, a formamidine compound widely used as an acaricide and pesticide, poses a significant threat to human health due to its toxic properties. This paper investigates the clinical-socio-epidemiological profile, PSS score, and complications of amitraz poisoning.

Methods: We report a case series of 8 patients with acute amitraz poisoning patients who presented to the emergency department of SVIMS, Tirupati, Andhra Pradesh, with consumption of only amitraz compound, not one combination of any other compound during the period from March 2020 to June 2021. These cases were studied and guided by literature.

Results: The study revealed diverse symptoms, including vomiting, diarrhea, bradycardia, and hyperglycemia among affected individuals. Laboratory investigations showed metabolic acidosis in 75% of cases and miosis in 62.5% of cases. Supportive care led to successful recovery in the majority of cases, with only one fatality reported.

Conclusions: Amitraz poisoning presents varied clinical manifestations and requires prompt recognition and management. Despite lacking a specific antidote, supportive care leads to favorable outcomes in most cases. Increased awareness, preventive measures, and regulatory interventions are essential to mitigate the growing incidence of amitraz poisoning.

KEYWORDS

Amitraz; Bradycardia; Hyperglycemia; Insecticide; Poisoning severity score

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Introduction

Around 1230 A.D., the term "poison" first appeared in English literature to designate a concoction made with lethal substances [1]. Pesticides, sedatives, chemicals, alcohol, plant toxins, and domestic poisons seem to be the most frequent causes of poisoning in India [2]. Our interest is to research amitraz toxicity among these substances. Amitraz belongs to the formamidine family. The Chemical formula is 1,5 di-(2,4-dimethylphenyl)-3-methyl-1,3,5-triazapenta-1,4-diene and the chemical name is Methanimidamide, N-(2,4-dimethylphenyl)-N-(((2,4-dimethylphenyl)methyl)-N-methyl-) [3]. The Molecular Formula is C19H23N3. The Physical characteristics appeared as white monoclinic crystals with a melting point of 187-189 °F (86-87 °C), which is insoluble in water. It is unstable under acidic conditions. When heated to decomposition, it emits toxic fumes of nitrogen oxides.

Amitraz is used as an acaricide for canines and animals as well as a pesticide for fruit crops in agriculture [4]. The mechanism of action of amitraz against ticks, mites, and demodectic and sarcoptic mange with that of metaflumizone, a sodium channel blocker, broadening the spectrum of activity by adding efficacy against fleas and lice. The most common form in which it is offered for sale is as a 12.5 to 50% aqueous solution, which is diluted in water at a ratio of 1:100–1000 before use [5,6]. The most common substance that is used as a solvent in the majority of the preparations is xylene, which is also a component of paints, cleaners, and glue [7]. Amitraz is listed as a group C "possible" human carcinogen and as being in the "Class III-slightly toxic" category [8]. In 1969, it was first synthesized in England [9]. In 1983, the first occurrence of poisoning in a human was documented [10]. Without a prescription, amitraz is offered by all reputable pharmacies under various brand names. The most popular brand name in India is "Ridd".

Amitraz has pharmacological activity and acts as an alpha-2 agonist. Both xylene and amitraz cause symptoms and signs in humans who have been exposed to the chemicals [11]. Neurotoxic and proconvulsant effects are primarily brought on by the stimulatory effect on alpha-2 receptors. Additionally, it affects the production of PGE2 and MAO activity. Amitraz poisoning frequently happens by ingestion, contact with the skin, or inhalation. Amitraz's toxic effects result from its adrenergic agonist action on the central nervous system and stimulation of both the adrenergic receptor types 1 and 2 in the peripheral area [12]. Clinical signs and symptoms include depression of the central nervous system, such as (mydriasis, drowsiness and convulsions), respiratory depression, bradycardia, hyperthermia or hypothermia, hypotension, vomiting, decreased gastrointestinal motility, hyperglycemia, polyuria and intestinal distension [13]. Acute toxic effects of xylene include periods of neuroirritability, CNS depression, poor motor coordination, ataxia, stupor, and nystagmus [6].

Physician knowledge of amitraz poisoning and its management is still lacking, which is probably why amitraz

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intoxication is underreported in isolated rural areas. It is frequently mixed up with poisoning from organophosphate or carbamate (OPC) and opioid poisoning. In recent years, India has reported several cases of amitraz poisoning [14]. In this case series on amitraz intoxication, we focus on demographic data information, clinical features, and the poisoning severity score of amitraz poisoning.

Material and Methods:

Study design

Clinico-socio-epidemiological profile, PSS score, and complications of amitraz poisoning: a descriptive observational study was conducted in the department of emergency medicine at SVIMS tertiary care hospital in Rayalaseema from March 2020 to June 2021.

Inclusion criteria

In the mentioned study period, eight cases of amitraz poisoning with a definitive history were included. The included cases were analyzed as per age, gender, occupation, duration since consumption, quantity consumed, the intention of poisoning, route of administration, socioeconomic status using the modified Kuppuswamy scale 2019, grading of the poisoning severity score at admission, symptoms, signs, systematic examination, investigations, treatment given, the requirement of ventilator support, duration of hospital stay, grading of the poisoning severity score at discharge, and final outcome.

Exclusion criteria

Cases with ages less than 17 and pregnant women were excluded.

Analytical methods

Laboratory investigations that were done in these cases are complete blood count using "Haematology blood cell counter (BC 5300 Mindary)", urine examination using Auto Analyzer AU 680 Beckman 1071019", renal function test using "Semi-auto analyzer Stat fax 3368", liver function test using "Coagulation Analyzer (Diamed)", blood sugar levels (i.e., random blood sugar more than 200 mg/dl considered hyperglycemia), urine output, arterial blood gas analysis using "Radiometer 754R25BN006", serum electrolytes using "SM Diagnostics 170291547", chest x-ray and electrocardiogram.

Statistical analysis

Data was recorded by the participants on a structured proforma. Data entry and analysis were done using Windows Excel 2019 (Windows Corporation, Redmond, WA). All the entries were double-checked for any possible errors. Frequencies, proportions, and percentages were calculated for qualitative data. The mean and standard deviation were calculated for quantitative data. IEC was obtained from the Institutional Ethics Committee, i.e., IEC No. 1012; Letter Roc. No. AS/11/IEC/SVIMS/17.

Ethical statements

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for clinical information to be reported in the journal. The patient understands that his name and initials will not be published and due efforts will be made to conceal his identity, but anonymity cannot be guaranteed.

Results and Discussion

Table 1 presents demographic information, clinical manifestations, and laboratory findings of poisoning cases. The data illustrates varying ages, genders, modes of exposure, symptoms, vital signs, and outcomes among the subjects, providing a comprehensive overview of the study cohort's characteristics.

Table 1. Demographic data, clinical, and laboratory findings of cases.

Sl. no	1	2	3	4	5	6	7	8
Age (years)	18	56	29	18	18	30	38	21
Gender	Female	Male	Male	Female	Female	Female	Male	Male
Place of poisoning	Residence	Residence	Residence	Residence	Residence	Residence	Residence	Residence
Type of exposure	Suicidal	Suicidal	Suicidal	Suicidal	Suicidal	Suicidal	Suicidal	Suicidal
Mode of Intoxication	Oral	Oral	Oral	Oral	Oral	Oral	Oral	Oral
Amount Ingested (ml)	15ml	200ml	50ml	15ml	20ml	20ml	30ml	20ml
Time lag	5 hr	4 hr	3 hr	5hr	4 hr	8 hr	6 hr	5 hr
Vomiting	-	+	+	+	+	-	+	+
Diarrhoea	-	+	+	+	-	-	-	-
Pain abdomen	-	+	+	+	+	-	-	-
Disorientation	+	+	-	-	-	-	-	-
Drowsiness	+	+	-	-	-	-	+	+
Body Temperature ◦F	98.4	100	99	98.4	98.4	102	98.8	98.6
Heart rate/min	68	88	91	68	66	52	96	90
Respiratory rate/min	18	20	24	21	24	18	20	20

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Blood Pressure (mmHg)	110/60	150/90	110/70	110/60	135/80	130/70	150/90	130/80
Bradycardia during hospital stay	+	-	-	+	+	+	-	-
Hypotension during hospital stay	+	+	+	-	-	-	-	-
Blood glucose (mg/dl)	132	205	110	140	324	152	167	154
ABG	Metabolic Acidosis	Metabolic Acidosis	Metabolic Acidosis	Metabolic Alkalosis	Metabolic Acidosis	Metabolic Acidosis	Respiratory Acidosis	Metabolic Acidosis
Urine Output	Normal	Normal	Normal	Normal	Normal	Normal	Normal	Normal
Mechanical Ventilation time	-	12 hours	4 days	-	-	-	-	-
Inotropes duration of stay	-	12 hr	4 days	-	-	-	-	-
PSS Score at admission	2	3	3	1	1	1	3	1
PSS Score at discharge	0	4	0	0	0	0	0	0
Outcome	Recovered	Demised	Recovered	Recovered	Recovered	Recovered	Recovered	Recovered

Tables 2-4 show that four cases (50%) were males and four cases (50%) were females, and the male-to-female ratio was 1:1. Among them, the maximum (37.5%) cases were present in the age group of eighteen age: < 30 years were (75%), and >30 years were (25%). The maximum number of cases were daily wagers and students, i.e., 25%, followed by farmers, home guards, housewives, and skilled salespeople (12.5%).

Table 2. Showing gender wise distribution of cases.

Gender	Males	Females	Total
Number of patients	4	4	8
Percent of patients %	50	50	100

Table 3. Age wise distribution of cases.

Age (in years)	No. of patients	Percentage (%)
18	3	37.5
21	1	12.5
29	1	12.5
30	1	12.5
38	1	12.5
56	1	12.5

Table 5 indicates that vomiting (75%), pain abdomen and drowsiness (50% each) were the most common symptoms. Other less common symptoms were diarrhea and sweating (37.5%) each, and disorientation (25%). Tables 6 and 7 show bradycardia (50%), hypotension (25%), and hypertension (25%). Table 8 shows hyperthermia in 2 patients (25%). Table 9 indicates a pupillary abnormality (miosis) present in 5 (62.5%) cases.

As per Table 10, hyperglycemia was seen in 2 (25%) of cases, and Table 11 depicts that metabolic acidosis was seen in 6 (75%) cases. Table 12 shows that the maximum number of cases, 6 (75%) were treated with oxygen support. One case with respiratory acidosis and one with metabolic acidosis were treated with mechanical ventilation.

Table 13 shows the maximum number of patients belonging to upper middle class 4 (50%) according to the modified Kuppuswamy scale 2019, followed by lower middle class 2 (25%).

Table 14 indicates that 4 (50%) had a minor (1) poisoning severity score at admission, followed by 3 (37.5%) with a severe (3) poisoning severity score. At discharge, 7 (87.5%) had a (0) poisoning severity score, followed by 1 (12.5%) with a fatal (4) poisoning severity score. Table 15 shows 3 (37.5%) cases in which patients stayed more than 3 days in the hospital.

Table 4. Occupation wise distribution of cases.

Occupation	Farmer	Daily wager	Home guard	Housewife	Skilled worker	Sales	Student
No. of patients	1	2	1	1	1		2
Percent of patients %	12.5	25	12.5	12.5	12.5		25

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Table 5. Incidence of symptoms in cases.

Symptoms	Vomiting	Diarrhoea	Sweating	Disorientation	Pain abdomen	Drowsiness
No. of patients	6	3	3	2	4	4
Percent of patients %	75	37.5	37.5	25	50	50

Table 6. Effect of amitraz poison on heart rate.

Heart rate/min	Bradycardia	Normal	Tachycardia	Total
No. of patients	4	4	0	8
Percent of patients %	50	50	0	100

Table 7. Effect of amitraz poison on blood pressure.

Blood Pressure (mm of hg)	Normal	Hypotension	Hypertension	Total
No. of patients	4	2	2	8
Percent of patients %	50	25	25	100

Table 8. Effect of amitraz poison on body temperature.

Temperature	Normal	Hyperthermia	Total
No. of patients	6	2	8
Percent of patients %	75	25	100

Table 9. Effect of amitraz poison on the size of the pupils.

Size of pupils	Normal	Miosis	Total
No. of patients	3	5	8
Percent of patients %	37.5	62.5	100

Table 10. Effects of amitraz poison on blood sugar level.

Blood Sugar	Normal	Hyperglycaemia	Total
No. of patients	6	2	8
Percent of patients %	75	25	100

Table 11. Effects of amitraz poison on arterial blood gas analysis.

ABGA	Metabolic Acidosis	Respiratory Acidosis	Metabolic Alkalosis	Total
No. of patients	6	1	1	8
Percent of patients %	75	12.5	12.5	100

Table 12. Distribution of patients according to management.

Management	Oxygen Support	Ventilatory Support	Total
No. of patients	6	2	8
Percent of patients %	75	25	100

Table 13. Distribution of socioeconomic status according to the modified Kuppuswamy scale.

Management	Lower Class	Lower Middle	Upper Lower	Upper Middle
No. of patients	1	2	1	4

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Table 14. Grading of Poisoning Severity Score at admission and discharge.

Poisoning Severity Score	At Admission	At Discharge
None	0	7
Minor	4	0
Moderate	1	0
Severe	3	0
Fatal	0	0

Table 15. Duration of hospital stay.

Duration of hospital stay	No. of patients	Percent of patients
≤ 3 days	5	62.5
>3days	3	37.5

Except for a single fatality, all other 7 cases were recovered completely. Lab investigations such as renal function test, liver function test, complete urine examination, serum electrolytes, chest x-ray and electrocardiogram were within limits. Gastric lavage was given to all eight patients. All cases were treated with intravenous fluids. Antibiotic therapies were given to all the intubated patients and oxygen support patients. Hypotension, hypertension, hyperthermia, Bradycardia, and pupillary abnormalities responded slowly to the given treatment.

A study done by Deepak et al. [15] showed that 64.7% of the cases had a loss of consciousness upon admission. In the present study, it was observed that 37.5% of the subjects had a loss of consciousness upon admission. The prevailing neurological abnormality observed in cases of amitraz poisoning was central nervous system depression. The manifestation of the toxin's effects can vary, presenting as somnolence, lethargy, or total unconsciousness, contingent upon the quantity of the poison ingested [6]. This phenomenon can be attributed to the modelling of central α2 adrenergic receptors as well as the involvement of the xylene solvent [16].

The results of a study conducted by Avsarogullari et al. [17], it was found that 8.7% of the cases exhibited bradycardia, while the other 91.3% of the cases had a normal heart rate. In the present investigation, it was observed that 50% of the cases had bradycardia, a proportion that surpasses that reported in the prior study. The average heart rate was found to be 77.37 ± 15.82. Bradycardia is induced by the activation of the dorsal motor nucleus of the vagal nerve via the presynaptic α2 adrenergic agonist mechanism [18].

In the study published by Agin et al. [19], it was shown that 14.33% of the cases exhibited hypotension, while the other 85.66% of the cases demonstrated normal blood pressure levels. Following a study conducted by Demirel et al. [20], a total of 2.22% of cases reported having hypertension. In the research done, it was observed that 25% of the cases exhibited hypotension, 25% of the cases displayed hypertension, and the remaining 50% of the cases showed normal blood pressure levels.

The study conducted by Deepak et al. [15] reported a mean systolic blood pressure of 119.4 mmHg and a mean diastolic blood pressure of 78.2 mmHg. In the research that was carried out, the average systolic blood pressure was found to be 128.12 ± 16.88 mmHg, while the diastolic blood pressure was measured at 75 ± 11.95 mmHg. The activation of central α2 adrenergic receptor agonists leads to the stimulation of presynaptic receptors, resulting in hypotension and a reduction in peripheral sympathetic activity. This ultimately leads to a decrease in blood pressure, which is further enhanced by the suppressive effects of xylene [18].

In the study conducted by Ertekin et al. [21], it was shown that 9.33% of the cases exhibited hypothermia, while the remaining 90.66% maintained a normal body temperature. In a study published by Ulukaya et al. [16], 33% of patients experienced a rise in body temperature of 98.4 °C. Whereas in the present study, it was observed that 25% of the patient's exhibited hyperthermia, while the remaining 75% maintained a normal body temperature.

According to a study conducted by Avsarogullari et al. [17], Miosis was seen in 26.08% of the cases, while the remaining 60.86% had pupils of normal size. In the current research, it was observed that 62.5% of the cases had miosis, a finding that is notably higher compared to the results reported in a prior study. Additionally, 37.5% of the cases displayed a pupil size within the normal range. The administration of α2 adrenergic agonists at low dosages results in the constriction of the pupil (miosis) through its pre-synaptic mechanism. Conversely, at greater doses, these agonists are recognized to induce mydriasis [18] via their post-synaptic impact.

According to a study carried out by Avsarogullari et al. [17] the prevalence of hyperglycemia was found to be 60.6% among the cases examined, while 39.13% of the cases exhibited normal blood sugar levels. But in the study, it was observed that 25% of the patient's exhibited hyperglycemia, a proportion that is notably lower compared to the findings of the previous study. Conversely, the remaining 75% of cases showed normal blood glucose levels.

The mean blood sugar level of 132.18 mg% was reported in a study conducted by Deepak et al. [15]. In this study, the average blood sugar level was shown to be 173 ± 66.97 mg%. The etiology of hyperglycemia in these individuals can be attributed to the stimulation of glucagon secretion and the inhibition of insulin secretion through α2 adrenergic receptor activation, resulting in a subsequent reduction in insulin release [22]. The presence of hyperglycaemia resulting from acute poisoning has demonstrated potential as a reliable indicator for assessing the severity of poisoning and predicting clinical outcomes. It is well established that amitraz poisoning is associated with the development of hyperglycemia [23].

In a study published by Kalyoncu et al. [24], two patients were diagnosed with respiratory alkalosis, three cases with respiratory acidosis, and five cases with metabolic acidosis. A study conducted by Surendra et al. [13], revealed that out of the total patients examined, six had respiratory acidosis, and two showed respiratory alkalosis. In this study investigation, a total of six cases (75%) exhibited metabolic acidosis, whereas one case each of metabolic alkalosis and respiratory acidosis was seen.

According to a study by Surendra et al. [13], 37.5% of the cases necessitated ventilatory support. In the present investigation, a notable finding was that 25% of cases

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necessitated ventilatory support, a proportion that is much lower in comparison to the findings of a previous study. Respiratory depression occurs as a result of the suppression of the reaction to carbon dioxide (CO2) through a direct impact on the respiratory center [25].

According to a study conducted by Rastogi et al. [26], found a significant majority (83.33%) of the cases examined to be associated with those from a lower socioeconomic class. Whereas in this case series, it was observed that 50% of the patients belonged to the upper middle class, while 25% of the cases belonged to the lower middle class.

In a study conducted by Ertekin et al. [21], the maximum length of hospitalization was seen to be five days, while the smallest duration of hospital stay was recorded as one day. In this analysis, most number of days a patient may stay in the hospital was 20, and the shortest time was 12 hours.

Treatment and outcome

The primary strategy employed in the management of amitraz poisoning cases involves the achievement of hemodynamic stability by appropriate hydration, maintenance of airway patency, administration of oxygen, mitigation of toxic substance absorption, and implementation of measures to enhance toxin removal from the body [13]. The use of gastric lavage during the critical period following ingestion of amitraz has been demonstrated to be a life-saving intervention for individuals experiencing acute poisoning [16]. Given the absence of a targeted antidote for amitraz poisoning, the medical approach mostly revolves around providing symptomatic and supportive care. Several animal investigations have provided evidence that α2 adrenergic antagonists, such as atipamezole and yohimbine, can reverse a significant portion of the clinical and biochemical manifestations associated with amitraz poisoning [27,28]. Nevertheless, given the absence of research or isolated reports documenting the utilization of these antagonists in human subjects, their potential application may be contemplated in instances of severe or unresponsive conditions. Despite the presence of severe life-threatening clinical manifestations, the majority of the cases exhibited successful recovery, except for a single individual.

The low fatality rate of amitraz poisoning gives a favourable prognosis. The likely explanation for the relatively low fatality rate can be attributed to the widespread availability of the chemical in a solution with a concentration of 12.5%. To achieve a deadly effect, a substantial quantity must be ingested at this level of dilution [29]. The dose and mode of intoxication of the poison appear to be the primary determinants influencing the clinical trajectory and prognosis. In the absence of randomized trials, it is not possible to derive any definitive findings regarding the optimal approach to managing this particular poisoning [15].

The purpose of the present study is to highlight the increasing cases of amitraz intoxication, which can be attributed to its widespread use in veterinary medicine on a global scale. The use of the poisoning severity score as a metric for the classification of acute amitraz poisoning aids in the assessment of the severity of the poisoning incident. Manufacturers, regulatory authorities, and national poison control centers have a crucial role in prevention due to the prevalence of accidental ingestion as the most frequently reported method of amitraz poisoning. To mitigate the occurrence of amitraz poisoning, it is imperative to provide public health information as a major preventive measure against poisoning through various media outlets. Additionally, the implementation of new legislation mandating safety covers on containers containing poisonous substances is warranted.

Conclusions

Since amitraz is widely used and easily accessible, it is anticipated that human poisoning with this substance will increase. Amitraz is a lethal poison that primarily affects the central nervous system and respiratory system and has no known antidote. Patients who have experienced pesticide exposure, hyperglycemia, and lack of the characteristic symptoms of organophosphate poisoning should always be evaluated for amitraz poisoning. Early and vigorous patient management contributes to a full recovery, which is the best possible patient result. The most prevalent symptoms were diarrhea and vomiting. The most typical symptoms were bradycardia and hyperglycemia. The supportive therapy received a positive reaction, and the evaluation of the severity of the poisoning showed excellent grading and severity outcomes.

Disclosure statement

No potential conflict of interest was reported by the authors.

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