Original Article

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A Case Report of Amitraz Poisoning

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Abstract

Amitraz is a pesticide commonly used in agriculture and veterinary practice to repel ectoparasites and insects. Poisoning with amitraz is rare especially for the purpose of self-harm. Although rarely fatal, it can result in significant symptoms. Management typically involves symptomatic treatment. We report a 34-year-old man who attempted suicide by ingesting amitraz presenting with unconsciousness. He had miosis, persistent bradycardia and extreme hypotension. Symptomatic treatment recovered him well with discharge from care with no complications and good health within 36 hours.

Keywords - bradycardia; hypotension; poisoning; vomiting; drowsiness.

Introduction

Amitraz (BTS27419**),** a pesticide known as a formamidine and a derivative of dimethylformamidine, is commonly utilized in agriculture and veterinary medicine to control insects[1, 2].Amitraz'salpha-2 adrenoceptor agonist action may play a role in manifesting the symptoms its of ingestion[3]. Incidents of human poisoning caused by amitraz are exceptionally rare and have limited documentation in the literature. The reported symptoms associated with ingestion include central nervous system depression, hypothermia, bradycardia, hypotension, hyperglycaemia, glycosuria, vomiting and respiratory failure[4].

Case presentation

A previously healthy 22-year-old male ingested 10 ml of 12.5% amitraz for deliberate self-harm. The patient was brought to the hospital 2 hours later exhibiting drowsiness. His blood pressure was 90/60 mmHg, and his pulse rate was 60/min. The respiratory rate was 12/min with an oxygen saturation of 92% on room air. The patient's Glasgow coma scale (GCS) was 9/15 (E-2, V-2, M-5) and his pupils were bilaterally reactive to light, measuring 2 mm. The rest of the systems examination were normal.

Arterial blood gas analysis indicated a type II respiratory failure, with a pCO2 of 60.1 mmHg(35mmHg-45mmHg) and a pO2 of 70 mmHg(75mmHg-100mmHg) pH-7.29(7.35-7.45), HCO3-28mEq/L(22mEq/L-24mEq/L) and lactate – 0.8. The patient was administered two doses of intravenous naloxone 0.4 mg within a 5-minute interval for suspicion of opioid poisoning. However, there was no improvement following initial management. Thirty minutes later, the patient's GCS decreased to 4/15 (E-1, V-1, M-2), and he became bradypnoeic, with a rate of 8/min. A repeat arterial blood gas revealed worsening type 2 respiratory failure, with a pCO2 of 70 mmHg (35mmHg-

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45mmHg) and a PaO2 of 65 mmHg(75mmHg-100mmHg),pH-7.19(7.35-7.45), HCO3- 29.5mEq/L (22mEq/L-24mEq/L)and lactate-1.0.The patient underwent elective intubation.

The patient received intensive care for 3 days, with continuous hemodynamic monitoring. On the fourth day he was safely extubated and stepped down to ward-based care. He was diagnosed of moderate level of depression and was initiated on oral fluoxetine 20mg mane. The patient did not have any residual medical complications and was euthymic on review 1 month after termination of care.

Table 1: Investigation results of the patient

White blood cell count	10.3 x 10^9	7.5x 10^9
neutrophils-	67.6%,	63.6%,
lymphocytes	22.4%	20.4%
Hemobglobin	13 g/dl	12.3 g/dl
platelets	342 x 10^9	339 x 10^9
Serum creatinine	88µmol/l (74-110)	77µmol/l (74-110)
Blood urea	3.1mmol/l (2.8-7.2)	3.0mmol/l (2.8-7.2)
Serum Na	135.2mmol/l (135-145)	138.3mmol/l (135-145)
Serum K	3.5 mmol/l (3.5-5.1)	4.1mmol/l (3.5-5.1)
Serum Calcium	2.2mmol/l (2.02-2.6)	
Aspartate aminotranseferase	23.2U/l (<50)	24.3U/l (<50)
Alanine aminotranseferase	22.3U/l (<50)	20U/l (<50)
Total Bilirubin	20 µmol/l(5-21)	19 µmol/l (5-21)
Direct Bilirubin	4.2 µmol/l(0-3.4)	4 µmol/l (0-3.4)
INR	1.01	
Creatinine phosphokinase	131(<171)	
Non-Contrast CT Brain	Normal	
Urine toxicology screen	Negative	

Discussion

Intoxication with amitraz in adults is usually suicidal and infrequently accidental. Only few reported human intoxications by this pesticide have been cited in literature, the existing information about it has been from animal studies or isolated case reports. Amitraz poisoning occurs via oral, dermal or inhalational routes. The toxic effects of amitraz are due to its α2-adrenergic agonist actions in the central nervous system and both α1 and α2 adrenergic receptorstimulation in the periphery. It also inhibits monoamine oxidase (MAO) enzyme activity and prostaglandin E2 synthesis. Some of these effects may be dose dependent. It has shown to have rapid toxic effects on both animals and human beings but rarely last beyond 48 hours[5, 6, 7, 8].

The primary clinical manifestation of amitraz poisoning involves a depressive effect on the central nervous system, resulting in decreased spontaneous activity. Other common symptoms include miosis, bradycardia, hypotension, hypothermia, hyperglycaemia and respiratory depression that can potentially lead to death. With prompt management, complete recovery from all signs and symptoms typically occurs within a span of 3-4 days. In cases where lower doses of amitraz are involved, individuals may exhibit heightened sensitivity and hyperactivity to external stimuli[5].

It is important to consider that symptoms and signs similar to its toxicity can also be observed in cases of organophosphate (OP) poisoning, as well as with the ingestion of substances such as clonidine, opioids, barbiturates, benzodiazepines, phenothiazines, and tricyclic antidepressants during an overdose. The onset of action in most reported cases of amitraz poisoning ranged from 30 to 180 minutes following ingestion. Our patient was discovered unconscious by a relative approximately one hour after ingesting the pesticide.

The primary symptoms observed in our patient were unconsciousness, respiratory depression, bradycardia, and hypotension. The central nervous system depression associated with amitraz poisoning is primarily attributed to its effect on α2-adrenergic receptors. The presence of respiratory depression alongside central nervous system depression may

indicate a direct inhibitory effect of amitraz on the respiratory centre. Additionally, the α1 and α2 agonistic actions of amitraz contribute to the development of bradycardia and hypotension, as reported in several case studies[5].

Since there is no specific antidote available for the treatment of amitraz poisoning, the medical management primarily focuses on symptomatic and supportive care[9]. This approach involves stabilizing the patient's haemodynamics, ensuring a clear airway, and implementing measures to minimize the absorption of the toxic substance. In the case of our patient, gastric lavage was performed upon presentation to the hospital. Dopamine, a medication with inotropic and chronotropic effects and in doses of 5-10 µg/kg/min, dopamine stimulates β1 adrenergic receptors and increases cardiac output by increasing cardiac contractility with variable effects on heart rate. Since only very few case reports on inotrope use in amitraz poisoning are available, convincing data to support any inotrope as the preferred first-line is lacking. To counteract the bradycardia and hypotension caused by amitraz, dopamine in doses of 5–10 µg/kg/min as used in our patient, can be the choice of inotrope.

Conclusion

Despite the severe clinical presentation involving central nervous system and cardiovascular depression, most reported cases of amitraz poisoning in humans have shown a favorable outcome, with recovery typically occurring within 12 to 48 hours. Patients were discharged without experiencing any organ dysfunction. The management of amitraz poisoning relies heavily on previous case reports and review articles, as there is currently no specific antidote or standardized treatment protocol available. Supportive and symptomatic care is the mainstay of management, with close monitoring of the nervous system, cardiovascular system, and respiratory system.

Conflict of interest: Nil

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Ethical clearance: Since this is a case report no ethical clearance needed. Informed consent was taken from the patient

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