

Amitraz Poisoning: Unusual Lethal Poisoning

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Abstract:

Background: Amitraz is a ectoparasiticide and pesticide used widely on animals and in agriculture. It contains triazapentadiene, a centrally acting alpha-2 adrenergic agonist. Though Amitraz is widely used worldwide, its poisoning is uncommon in humans. Children are frequently involved due to accidental exposure whereas in adults suicidal attempts are more common. Poisoning occurs via oral, dermal and inhalational routes.

Case presentation:

Case 1: We describe a case of Amitraz poisoning in a 21 years old young female. She developed bradycardia and hypotension and required oxygen support and intensive care unit stay. She had a quick recovery after she was treated symptomatically and was discharged well after 4 days.

Case 2: Case of Amitraz poisoning in a 23 years old young male. He was unconscious but vitals were maintained. He required ventilatory support and intensive care unit stay. He had a late recovery after he was treated symptomatically and was discharged well after 6 days.

Conclusion: There is currently no known antidote for amitraz poisoning and the management is mainly supportive and symptomatic. Public education on potential hazards of amitraz poisoning is very important for community health.

Keywords: Amitraz, Poisoning, Adult

I. Introduction

Amitraz, 1,5 di-(2,4-dimethylphenyl)-3-methyl-1,3,5-tri-aza-penta-1,4 diene, is a formamidine pesticide which is increasingly being used as an insecticide and an acaricide¹. Its varied uses include treatment and control of generalized demodicosis in canines, ticks and mites on cattle and sheep, psylla infection of pears and also for control of red spider mites on fruit crop. Amitraz is an uncommon poisoning in humans and may occur via oral, dermal or inhalational routes. Being a centrally acting alpha-2 adrenergic agonist, it can present with life-threatening effects such as varying degrees of central nervous system (CNS) depression, hypotension, bradycardia, hypothermia and respiratory depression. In a retrospective study of 44 adults, Demirel et al³ showed a correlation between the amount of amitraz ingested and the length of CNS depression observed. Atabek and colleagues suggest that higher doses of amitraz results in mydriasis while lower doses leads to miosis⁴. Amitraz is also known to inhibit the enzyme monoamine oxidase and to inhibit prostaglandin synthesis but the clinical significance of these actions remains unknown⁴. A limited number of case reports of human intoxication have been published.

II. Case Report S

Case 1: A 21 year old female patient, was brought to emergency department (ED) with a history of consumption of 30ml of Amitraz poison two hours before being brought to ED. The patient had few episodes of vomiting and was semi-conscious at the time of admission. She was given gastric lavage and intravenous crystalloids were started. Over the next couple of hours, her sensorium deteriorated. There was no history suggestive of seizures. The patient did not have any known premorbid medical condition and had no history of prescription drug usage.

Her heart rate was 106 beats per minute and blood pressure was 90/50 mm Hg. She had a shallow respiration with a respiratory rate of 22/min. Oxygen saturation at presentation was 99% with oxygen delivered via venturi face mask. On examination of the CNS, her pupils were bilaterally constricted. Other systemic examinations were normal. There were no excessive oral secretions or any fasciculations. Baseline line blood investigations (complete blood count, renal function test, serum electrolytes, liver function test), plasma cholinesterase level, electrocardiogram, chest X-ray, routine urine testing were normal. Blood alcohol level was normal. The patient was given supportive treatment in our intensive care unit in the form of intravenous fluids, proton pump inhibitors and oxygen inhalation. Later patient became conscious with a

Glasgow Coma Scale (GCS) of 13/15. She was transferred to the general ward and was subsequently discharged after consulting the psychiatrist.

Case 2: A 23 year old male patient, was brought to emergency department (ED) with a history of consumption of 50ml of Amitraz poison four hours before being brought to ED. The patient had few episodes of vomiting and was unconscious with shallow respiration at the time of admission. He was given a gastric lavage and intravenous crystalloids were started. There was no history suggestive of seizures. The patient did not have any known premorbid medical condition and had no history of prescription drug usage. His heart rate and blood pressure were maintained. Oxygen saturation was 95% with SIMV ventilation. On examination of the CNS, his pupils were bilaterally constricted. Other systemic examinations were normal. There were no excessive oral secretions or any fasciculations. Baseline line blood investigations (complete blood count, renal function test, serum electrolytes, liver function test), plasma cholinesterase level, electrocardiogram, chest X-ray, routine urine testing were normal. Blood alcohol level was normal. The patient was given supportive treatment in our intensive care unit in the form of intravenous fluids, proton pump inhibitors and oxygen by mechanical ventilatory support. He was transferred to the general ward after weaning from ventilator support and was subsequently discharged after consulting the psychiatrist.

III. Discussion

Amitraz is an alpha2 adrenergic receptor agonist. It stimulates α_2 receptors in the CNS, α_2 and α_1 receptors in the periphery and also inhibits monoamine oxidase (MAO) enzyme activity and prostaglandin E2 synthesis⁵. The clinical manifestations of amitraz (impaired consciousness, drowsiness, vomiting, disorientation, miosis, mydriasis, hypotension, bradycardia, respiratory depression, hypothermia, generalized seizures, hyperglycemia and glycosuria) can be explained by the agonist action of amitraz on α_1 and α_2 receptors. Drowsiness is the predominant manifestation whereas seizures and deep coma is also reported. Bradycardia and hypotension are the cardiovascular manifestations and respiratory depression too is common. Liver biochemistry, renal biochemistry, serum electrolyte and blood gases are rarely effected. The effects of amitraz in animals resemble that of pure alpha 2 adrenergic agonist drugs like clonidine⁵. It can also be misdiagnosed as organophosphate or carbamate toxicity, since all three share several similar clinical features⁶. Opioids, barbiturates, benzodiazepines, phenothiazines and tricyclic antidepressants can also display similar symptoms and signs in overdose⁵.

In the laboratory findings, hyperglycemia and glycosuria are considered the hallmark of amitraz poisoning and are thought to be caused by inhibition of insulin release and stimulation of glucagon secretion⁷. Other laboratory findings are considered nonspecific and may include minimal increases in liver enzymes. Blood urea nitrogen, creatinine and electrolytes are usually not affected but polyuria has been reported in both children and adults and may be due to decreased antidiuretic hormone (ADH) and renin secretion, inhibition of ADH effect, and enhanced diuresis by increased glomerular filtration rate². We would like to emphasize that although Amitraz poisoning has a life-threatening presentation, most cases result in complete recovery if appropriate supportive treatment is instituted in a timely fashion.

IV. Conclusion

Extensive use and easy availability of amitraz has lead to increased human poisoning cases. Amitraz poisoning should always be considered in patients with a combination of pesticide exposure, hyperglycemia, and without the classical features of organophosphate poisoning. Increased awareness along with high index of suspicion among clinicians and health workers is required for timely diagnosis and appropriate inpatient management of this otherwise potentially life-threatening intoxication.

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