

A Clinical Conundrum Called Amitraz Poisoning – A Case Report

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Abstract

Amitraz is a nonsystemic insecticide and acaricide which is sometimes also used as scabicide. Due to its widespread use, amitraz poisoning has emerged during the past decade and a half although the literatures on human intoxication cases are scarce. Amitraz poisoning can present with numerous symptoms involving central nervous, cardiovascular, and respiratory systems. The mainstay of treatment is supportive and symptomatic. We present the case of a 60-year-old gentleman who was shifted from another hospital in unconscious state with an alleged history of consumption of an unknown substance which later was known to be amitraz.

Keywords: Amitraz, bradycardia, insecticide, mydriasis, poisoning

INTRODUCTION

Amitraz is a triazapentadiene compound, a member of the amidine chemical family, and is used worldwide for controlling the ectoparasites in animals.^[1,2] Commercial formulations of amitraz generally contain 12.5%–20% of the drug in organic solvents, especially xylene, which is also used as a solvent in paints, cleaners, and glues. It is diluted with water before applying to plants and animals.^[3] Amitraz is an alpha-2 adrenergic agonist as it stimulates the alpha-2 adrenergic receptor sites in the central nervous system (CNS) and alpha-1 and alpha-2 adrenergic receptor sites in the periphery.^[4] It also inhibits the monoamine oxidase enzyme activity and prostaglandin E2 synthesis. Amitraz poisoning may occur through the oral or dermal routes and potentially, by inhalation.^[5] Amitraz poisoning has increased in the recent years probably because of the easy availability of the product without prescription.

CASE REPORT

A 60-year-old gentleman was shifted to the Intensive Care Unit (ICU) of our tertiary care center from a nursing home with an alleged history of consumption of unknown substance. The patient had received a gastric lavage with doses of atropine (due to bradycardia) and pralidoxime. On examination, the patient was in unconscious state with mydriasis with negative light reflex, Glasgow Coma Scale of 3/15, and a

Full Outline of Unresponsiveness score of 3/16. Vital signs were as follows: blood pressure 140/90 mmHg, pulse rate 80/min, respiratory rate 10/min, and temperature 37°C. There were signs of respiratory depression with blood gas analysis showing PaO₂: 96.3, O₂ saturation: 94, pH: 7.15, pCO₂: 68.4, and HCO₃⁻: 23.5 on 15 L of O₂. The patient was electively intubated and mechanically ventilated with intermittent positive-pressure ventilation mode. Routine blood investigations such as complete blood count, liver and renal function tests, coagulation profile, and random blood sugar turned out to be within normal limits. Chest X-ray was normal. Electrocardiography (ECG) was normal.

The patient was initiated on atropine and pralidoxime infusions suspecting organophosphorus (OP) poisoning based on some symptoms and being one of the most common intoxications in our country. However, there was still a sliver of doubt as the patient did not exhibit diaphoresis, salivation, lacrimation, and bronchospasm. After stabilization of the patient, history taking revealed the patient to be found unconscious on the kitchen floor with evidence of vomitus in the sink. Few relatives who

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Access this article online

Quick Response Code:



Website:
www.ijccm.org

DOI:
10.4103/ijccm.IJCCM_241_17

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How to cite this article: Saseedharan S, Pathrose EJ, Madhav BV. A clinical conundrum called amitraz poisoning. Indian J Crit Care Med 2018;22:195-6.

stayed back at the residence found an empty bottle lodged in a corner of the kitchen which was handed over to us. It was a 15-ml bottle of amitraz 12.5% solution. The atropine and pralidoxime infusions were discontinued based on the circumstantial evidence of the amitraz bottle.

After 8 h, the patient began to regain consciousness and was weaned off the ventilator and extubated the following day. He was observed in the ICU for another day followed by another day in the ward and was subsequently discharged from the hospital.

DISCUSSION

Our case indicates that amitraz poisoning can be easily misdiagnosed as OP poisoning due to similarity of features, thereby making it necessary for clinicians to be aware of the substance and the distinguishing factors from OP poisoning.

Our patient had unconsciousness, bradycardia, miosis followed by mydriasis, and respiratory depression with lack of fasciculations and hypersecretory state which goes in favor of amitraz poisoning. Dhooria and Agarwal^[6] in a systematic review involving 310 cases explain the systematic effects of amitraz with its underlying mechanism. Adjunctive investigations such as complete blood count, liver/renal function tests, coagulation profile, random blood glucose, and ECG are usually within normal limits.^[7,8]

The crucial point to be noted from our case is that emphasis should be given to retrieve the container so that the underlying formulation can be noted which is essential toward constructing a plan of management.^[9]

Management is supportive due to lack of a specific antidote. The crux of it depend on respiratory, cardiovascular, and CNS stability.^[10] Unconscious patients should be intubated for airway securement to prevent aspiration. Nearly 33% of patients develop respiratory failure and 20% need mechanical ventilation; however, they get extubated within 48 h (within 24 h in our case).^[6] Intravenous fluids and vasopressors/inotropes should be administered as per the need of the moment. One debatable point in the treatment is the use of atropine. While majority of the literature states that atropine is effective precisely in symptomatic bradycardia and atrioventricular blocks,^[10] some authors believe that it can be used in bradycardia induced by other mechanisms such as amitraz toxicity.^[11]

Recovery in cases of amitraz poisoning is rapid with an excellent prognosis.^[5,9-11]

CONCLUSION

It can be concluded with certainty that amitraz poisoning can be confused with OP poisoning due the mimicry of some symptoms. Differentiation needs clinician's knowledge of the mechanism of action of the poison with either retrieval of the container or thorough questioning of the attendants regarding the substance consumed. The management is conservative due to nonavailability of a specific antidote. It carries a good outcome with no long-term morbidity.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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