IJMAHS

CASE STUDY REPORT

Amitraz Poisoning – A Rare Pesticide Poisoning

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ABSTRACT:

Amitraz, an insecticide/acaricide of the formamidine pesticide group , is a alpha 2 adrenergic agonist used to a great extent in veterinary and agricultural products for the treatment of ectoparasitic manifestations. In the current article we report the findings of a case of 22 year old female who consumed about 50 ml Amitraz poison by oral route as a suicidal attempt. On arrival to Emergency Department the patient presented in deep comatose state, respiratory depression, bradycardia, hypotension, miosis, hypothermia and hyperglycemia.she recovered completely within 48 hours with adequate supportive care. The case report throws considerable light on the management of Amitraz poisoning ,good prognosis with early recognition, initial stabilisation, reducing absorption, supportive management with Iv fluids, airway management,monitoring urine output and other supportive care, very few cases of intoxications in human beings due to the pesticide have been published in literature It has become imparative to intruct the pesticide manufacturers to initiate suitable measures to decrease the incidence of Amitraz poisoning by prominent and clear warning labels on the containers and potential hazards of the compound.

Key-words: Amitraz; poisoning; alpha 2 adrenergic agonist; miosis

INTRODUCTION

Amitraz, a triazapentadiene compound and a member of the amidine chemical family is a formamidine pesticide which is increasingly being used as an insecticide and an acaricide to control animal ectoparasites [1-3]. The formulations available for chemical use contain 12.5-50% in an organic solvent called xylene, which itself is used in plant cleaners and glues[4] which adds to the toxic effect[4]. Amitraz is a Alpha 2 adrenergic agonist stimulating alpha 2 adrenergic receptors in the Central Nervous System (CNS) and both alpha 1 and alpha 2 adrenergic receptors in the periphery. Poisoning occurs throgh oral, inhalational (the mostpotential), and dermal routes and is accompanied by numerous signs and symptoms varying from CNS depression (drowsiness, coma, and convulsion), to miosis, or rarely, mydriasis, respiratory depression,bradycardia, hypotension,hypothermia or fever, hyperglycemia, polyuria, vomiting, decreased gastrointestinal motility, and intestinal distension [4].Adverse effects and side effects have been reported in animals exposed to the product : however only few cases of human toxication have been published in Indian literature. We present a young female patient with Amitraz poisoning who was conservatively managed with complete recovery hence significantly contributing to the limited human toxicological data.

CASE HISTORY

An 22 year old female was brought to our Emergency Department (ED) with a history of suicidal consumption of about 50 ml Amitraz poison eight hours before being brought to our ED, her first symptom had begun about 30 minutes post ingesion and included nausea and vomiting, thus she was taken to a hospital in their locality where intravenous crystalloids were started and referred to our centre .On arrival to our department the patient was deeply comatose with a GCS scale of 4/15. Her pulse rate, respiratory rate, blood pressure and temperature were 50/min, blood pressure was 92/64 mm of Hg,16/minand 36.8 degree celsius respectively. On examination of CNS her pupil were bilaterally constricted,all four limbs had hypotonia and there was bilateral flexor plantar response. Other systemic examination were normal,there was no exessive oral secretions or any fasciculations.Gastric lavage with activated charcoal was given and

patients airway was secured with endotracheal intubation due to low GCS. She was then admitted to ICU for further management her lab tests (Complete blood count,serum electrolytes,renal function tests, liver function tests) , serum pseudocholinestrase levels, electrocardiography, routine urine tests and chest xray were normal except glucose level of 245 mg/dl.A urine test for drugs of abuse was negative and blood alcohol levels were normal. Ct brain plain was done which was normal.She was treated with supportive care in the ICU with IV Flluids, respiratory and cardiac monitoring ,Atropine (once 2mg stat) was adminitered for transient bradycardia.over the next 48 hours she gradually improved and was extubated. Her vitals signs were Heart rate of 70/min and blood pressure was 110/70 mm of Hg . By the following day she was completely concious and was able to answer the question and she was shifted to general ward and was discharged after consultation with a psychiatrist.

DISCUSSION:

Amitraz is increasingly being used worldwide in veterinary and agricultural products for the treatment of ectoparasitic manifestations. Formamidines show reversible toxic effects on both animals and humans[1] .The present knowledge about Amitraz and Foramine pesticides is usually built on animal studies as the available human intoxication is limited .It can cause poisoning in animals and humans via oral, inhalational or dermal routes .The toxicity from this poisoning can be attributed both Amitraz and the solvent, xylene. Although the ingested dose of Amitraz can not be determined because it is diluted 1 part in 500 before usage. The acute oral medical lethal dose (LD50) for the rats is 800/kg body weight.[3,4] .The clinical features of this poisoning reported in previous reports include CNS depression, drowsiness, vomiting, miosis, bradycardia, hypotension, and hyperglycemia. The duration of CNS depression has ranged from a few hours to 24 h [4]. CNS symptoms began within 120-180 minutes and resolved within 12-24 hrs in our case. Sedative effects of α2-agonists are dose dependent [1]. Coma, absence of light reflex, and respiratory failure are due to the ingestion of greater amounts of amitraz supporting its dose-dependent effects. Our patient was fully conscious after 48hrs. This time has been reported to be 2-48 h in previous reports.

The effect of amitraz on α1– and α2-receptors causes bradycardia [5]. In addition, literature reported hyperglycemia, hypotension, and bradycardia in amitraz poisoning and attributed them to the alpha-2 adrenoceptor agonist action of amitraz [6]. In our case, bradycardia was also present accompanying with miosis .

Co-existence of bradycardia, miosis, and the respiratory depression leads to confusion with organophosphate or opioid poisonings, both of which should be excluded. Using atropine for treatment of bradycardia is controversial. Most studies, however, have reported atropine to resolve both miosis and bradycardia. Atropine is the first line therapy for the bradycardia resulted from vagal stimulation and atrioventricular blocks. Alpha-2 adrenergic drugs can also cause bradycardia by stimulating the dorsal motor nucleus of the vagus nerve. Studies have shown that atropine increases the heart rate and prevents Amitraz induced bradycardia in Animals (2). In our patient atropine was given once with the adult dose.

Amitraz and its active metabolites inhibits insulin and stimulate glucagon secretion , hyperglycemia was detected in our case as reported in previous studies by Demirel and colleagues[7]

Kalyoncu and colleagues have reported hyponatremia in their three cases [9], Usually BUN, creatinine, serum sodium and potassium do not change with this poisoning, in our case creatinine, serum potassium and sodium were normal. Kalyoncu and associates have reported respiratory alkalosis in two,respiratory acidosis in three and metabolic acidosis in five cases[9] ,in our patient the analysis of blood gases were normal. Avsarogullari et al reported hyperglycemia and fast deterioration of the patients with amitraz poisoning (within 5 minutes of ingestion of toxin)[8]

Whenever a patient presents with bradycardia and miosis, organophosphorus compound poisoning should be considered as a differential diagnosis a along with Amitraz. Other signs and symptoms of organophosphorus compound should be looked for and a cholinesterase level should be done. Amitraz levels in blood was not done because it was unavailable at our institute and other referral laboratories.

It is made clear that the basic approach to a patient with amitraz poisoning involves initial stabilisation, reducing absorption and increasing elimination of the toxin .there is no specific antidote [2] medical management involves supportive measures like gastric lavage , activated

charcoal administration and securing the airway. Depending on the patient's condition additional measures like oxygen supplementation or mechanical ventilation for respiratory depression, atropine for severe bradycardia, intravenous fluids and vasopressors for hypotension, diazepam or lorazepam for seizures.

This case report throws considerable light on the management of Amitraz poisoning, good prognosis with early recognition and timely supportive management as the available human toxicological data are limited. When appropriate timely supportive treatment is given, Amitraz intoxication in humans caries a low morbidity and mortality inspite of rapidly progressing and life threatening clinical picture. It has become imperative to instruct the pesticide manufacturers to initiate suitable measures to decrease the incidence of Amitraz poisoning

by placing prominent and clear warning labels on containers.

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Site this article as: Vikas G krishnamurthy, Narendra S S, Sunil manohar RV, Amitraz Poisoning – A rare pesticide Poisoning, Int J Medicine and Allied Health Sciences, 2014; 4: 284-286