ECRONICON OPEN ACCESS

EC EMERGENCY MEDICINE AND CRITICAL CARE
Case Report

Amitraz - An Opiod and Organophosphorus Poison Mimic

Aditi S Patankar[1] and ND Karnik[2]*

[1]*Junior Resident, Department of Internal Medicine, Lokmanya Tilak Municipal Medical College and General Hospital, Sion, Mumbai, India*

[2]*Professor and Head, Department of Internal Medicine, Lokmanya Tilak Municipal Medical College and General Hospital, Sion, Mumbai, India*

***Corresponding Author**: ND Karnik, Professor and Head, Department of Internal Medicine, Lokmanya Tilak Municipal Medical College and General Hospital, Sion, Mumbai, India.

Received: August 10, 2019; **Published:** September 30, 2019

Abstract

A 35 year old male working at a dog parlour attempted suicide by consuming amitraz, a veterinary pesticide, in an impulsive suicide attempt. He developed bradycardia, respiratory depression, hypothermia and needed mechanical ventilation. Early treatment in MICU resulted in prompt and complete recovery in 48 hours.

Keywords: *Amitraz; Poison; Bradycardia*

Introduction

Amitraz is a compound which belongs to the formamidine chemical family. It is used by veterinarians to eradicate pests from animal fur. Commercial formulations of amitraz generally contain 12.5 - 20% of the drug in organic solvents like xylene. It causes bradycardia, hypotension, hypothermia and meiosis, thus mimicking organophosphorous poisoning. Treatment is supportive and there is complete recovery.

Case Report

35 yr old male, working at a dog salon consumed 60 ml of RIDD liquid containing amitraz 12.5% wt/vol (Figure 1) as a suicidal attempt following an argument with his wife. He was brought to the emergency services 3 hours later in a drowsy state with laboured breathing. There was no history of vomiting, excessive sweating, urinary incontinence, convulsions or previous suicidal attempts.

Figure 1: *RIDD liquid.*

On examination, the patient was afebrile, drowsy but arousable, normotensive with a regular pulse of 60/min and respiratory rate of 21/min. Pupils were bilaterally pin point and non-reactive to light. There was no neck lag or fasciculations. On the Glasgow coma scale, he had a score of 14. Arterial blood gas analysis (ABG)on admission revealed a pH of 7.4 and bicarbonate of 21.5 meq/L with carbon dioxide washout ($paCO_2$ 30mmHg). Na- 143 meq/L/ K- 3.0meq/L. A nasogastric tube was inserted and the milky frothy sample obtained on aspiration lacked the characteristic smell of organophosphorous compounds. Gastric lavage followed by charcoal instillation was then given. Over the next 30 minutes his pulse rate dropped to 45/min and respiratory rate dropped to 10/min while maintaining saturation of 99% on pulse oximetry. His score on Glasgow Coma Scale dropped to 12 points. ABG now revealed pH- 7.39/pCO_2 of 41.6 mmHg/HCO_3 of 24.9 meq/L/pO_2 of 75.8 mmHg. In view of respiratory depression, drowsy state and unusual nature of poison, patient was transferred to the medical intensive care unit, was intubated and needed mechanical ventilation. Atropine was used as required for management of bradycardia. A screening for urinary toxins was negative for barbiturate, benzodiazepine and opioids. Normal serum cholinesterase levels (7568 IU/L) ruled out organophosphorous poisoning (normal level - 6000 – 12000 IU/L). Thyroid function tests were normal. Chest X-ray was within normal limits. The laboratory investigations are outlined in table 1. Electrocardiogram was suggestive of Osborn waves in lead V2-V5 (Figure 2). Patient needed 2.4 mg of atropine (0.6 mg/ml) totally over 48 hours. His pulse rate stabilised, he regained consciousness and was extubated at the end of 48 hrs. Repeat electrocardiogram was done which showed resolution of Osborn waves (Figure 3). Psychiatry evaluation was done and he was started on quetiapine in view of deliberate self harm (DSH). Patient was discharged on day 5 of admission with instructions to follow up in DSH counselling group.

Date	27.1.19	28.1.19
Hemoglobin (mg/dl)	16	15.9
WBC count/mm^3	14200	17700
Platelets/mm^3	158000	149000
Random blood sugar (mg/dl)	244	150
BUN/Serum Creatinine (mg/dl)	11/1.1	14/1.3
SGOT/SGPT (IU/L)	54/38	
Serum bilirubin (mg/dl)	1.2	

Table 1: *Laboratory investigations.*

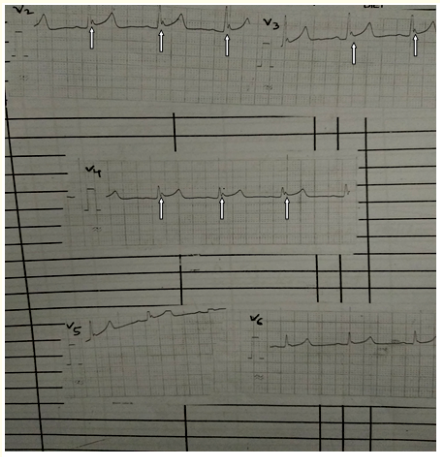

Figure 2: *ECG on admission shows Osborn waves (arrows).*

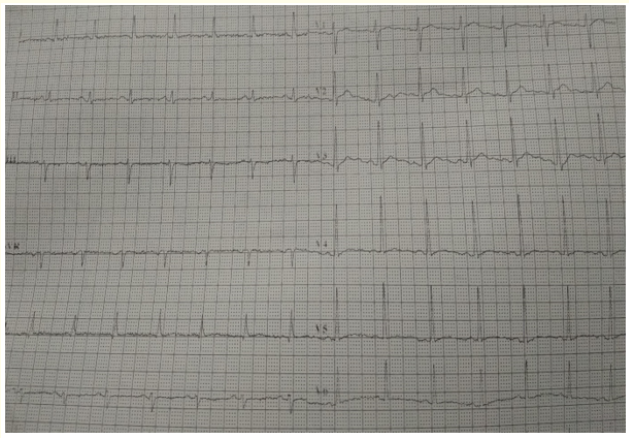

Figure 3: *Resolution of Osborn Waves on ECG after 48 hours.*

Discussion and Conclusion

Amitraz is an insecticide used in veterinary industry worldwide [1]. It is known to cause poisoning in animals and humans when ingested, inhaled, or after skin exposure. The toxic dose reported is 3.75 mg/kg[2] [2]. Mechanism of action: Amitraz stimulates α_2 adrenergic receptor sites in the central nervous system (CNS) and α_1 adrenergic and α_2 adrenergic receptor sites in the periphery similar to the drug clonidine [3]. The clinical features include central nervous system depression, bradycardia, hypotension, hypothermia, meiosis, absent pupillary reflexes [1]. Central nervous system depression was the predominant feature in this case, consistent with the effect of amitraz on α_2-adrenergic receptors [1]. The α_1- and α_2-agonistic action of amitraz leads to bradycardia by stimulating the dorsal motor nucleus of the vagal nerve [4]. The co-existence of bradycardia, meiosis and respiratory depression necessitates exclusion of close differentials like organophosphate or opioid poisoning. However, in contrast to organophosphorous poison, amitraz causes reduced salivation with decreased gastrointestinal motility. In animal experiments with amitraz, atropine has been found to increase the heart rate [5-7]. Bradycardia responded to atropine in this case. However, there is no role of atropine in absence of bradycardia. Amitraz and its active metabolite inhibit insulin and stimulate glucagon secretion resulting in hyperglycaemia [8]. A random glucose level of 249 mg/dl was noted in our patient (Table 1). Non -specific electrocardiogram changes are found in amitraz poisoning [7]. In this case there was presence of Osborn waves in chest leads probably secondary to amitraz induced hypothermia. In conclusion, there is no specific antidote for amitraz poisoning and the management is supportive and symptomatic [9]. With the rising number of dog salons and increased use of animal hygiene products more awareness is required regarding the safety profile of these products and their possible deleterious effects on humans. Clear guidelines are required for categorisation of these products as per their toxic potential.

Disclosures

Dr. N.D. Karnik and Dr. Aditi. S. Patankar have neither commercial or financial conflicts of interest nor any relevant funding sources.

Bibliography

1. Yilmaz HL and Yildizdas DR. "Amitraz poisoning, an emerging problem: epidemiology, clinical features, management, and preventive strategies". *Archives of Disease in Childhood* 88.2 (2003): 130-134.

2. Bonsall JL and Turnbull GJ. "Extrapolation from Safety Data to Management of Poisoning with Reference to Amitraz (a Formamidine Pesticide) and Xylene". *Human Toxicology* 2.4 (1983): 587-592.

3. Jorens PG., *et al*. "An unusual poisoning with the unusual pesticide amitraz". *Human and Experimental Toxicology* 16.10 (1997): 600-601.

4. Sanders KH and Jurna I. "Effects of urapidil, clonidine, prazosin and propranolol on autonomic nerve activity, blood pressure and heart rate in anaesthetized rats and cats". *European Journal of Pharmacology* 110.2 (1985): 181-190.

5. Jones RD. "Xylene/amitraz: a pharmacologic review and profile". *Veterinary and Human Toxicology* 32.5 (1990): 446-448.

6. Cullen LK and Reynoldson JA. "Cardiovascular responses to amitraz in the presence of autonomic antagonists and agonists". *Archives Internationales de Pharmacodynamie et de Thérapie* 296 (1988): 45-56.

7. Hsu WH., *et al*. "Effect of amitraz on heart rate and aortic blood pressure in conscious dogs: Influence of atropine, prazosin, tolazoline, and yohimbine". *Toxicology and Applied Pharmacology* 84.2 (1986): 418-422.

8. Smith BE., *et al*. "Amitraz-induced glucose intolerance in rats: antagonism by yohimbine but not by prazosin". *Archives of Toxicology* 64.8 (1990): 680-683.

9. Eizadi-Mood N., *et al*. "Amitraz poisoning treatment: still supportive?" *Iranian Journal of Pharmaceutical Research* 10.1 (2011): 155-158.

Volume 3 Issue 10 October 2019