



## Amitraz Poisoning: An Under Recognised and Unusual Poisoning: A Case Report

Authors

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### Abstract

*Amitraz poisoning is an unusual, but definitely with a potential to become lethal poisoning. Amitraz is a Triazapentadiene – a  $\alpha_2$  adrenergic agonist used widely in veterinary and agricultural field for the treatment of Ectoparasitic infestation. We report a case presented to our ER after two hours of ingestion of AMITRAZ. On examination patient was comatose with response to only deep painful stimuli. She was also in bradycardia, hypotension and metabolic acidosis. The patient was managed with symptomatic treatment. Patient's condition started to improve after 18 hrs of admission and she was discharged after 4 days.*

*Though Amitraz poisoning can be fatal, if identified and treated well within time, even with minimal symptomatic management we can save the patient to complete recovery*

**Keywords:** Amitraz,  $\alpha_2$  Agonist, CNS depression.

### Introduction

Amitraz (Fig.1) is a pesticide used worldwide. It is a triazapentadiene<sup>[1]</sup> compound which is a centrally acting  $\alpha_2$  adrenergic agonist



**Fig. 1**

It is a member of formamidane pesticide. Commercial formulations of Amitraz generally

contain 12.5 – 50% of the drug in organic solvents especially xylene is used as a solvent. A limited number of human intoxication cases have been published in literature. Poisoning from Amitraz is under recognized even in areas where it is widely available.

Potentially serious side effects have been reported in cases exposed to the product when left untreated or unnoticed however if treated early this poison carries a very good prognosis.

### Case Report

16 years old female was brought to us following ingestion of 10ml of Amitraz 12.5% mixed with water after 3hrs of consumption. The relatives

noticed that the patient was unresponsive even after 45 min post consumption hence they brought her to us. She had 3 episodes of vomiting prior to ER admission. Patient was not having any significant medical ailments before and she was not on any regular medication. At the time of presentation she was unconscious with Glasgow coma scale of 6/15. She had a pulse rate of 52/min and blood pressure of 88/60mm Hg. Her respiratory rate was 16/min with temperature of 98.4<sup>o</sup>F. Her oxygen saturation was 98% with 6L/min of oxygen supplied via facemask. Motor system examination revealed hypotonia of all four limbs with diminished reflexes. There was no fasciculation. Plantar reflex was normal bilaterally. Pupils were bilaterally constricted with size 1.5mm reacting to light. Optic fundus examination was normal. Further detailed neurological examination could not be elicited. Examination of other systems was normal.

Gastric decontamination was done using gastric lavage and activated charcoal. Blood sample was taken for analysis.

The investigation profile comprising of complete blood count, liver and renal function tests, serum electrolytes were normal. Random blood sugar level was 195 mg/dl. Arterial blood gas analysis showed metabolic acidosis (Fig. 2)<sup>[2]</sup> which improved progressively in serial arterial blood gas analysis. CT brain was normal. Electrocardiogram revealed sinus bradycardia (Fig. 3).

Serum cholinesterase level was done to rule out organophosphorous poisoning and was normal. Hence she was treated symptomatically. She had persistent bradycardia with heart rate of 46-58/min. However her heart rate was maintained with intravenous Atropine boluses. Only symptomatic treatment and supportive measures were given to the patient. She regained consciousness and responded to oral commands after 16 hrs of intensive care.

The patient was then shifted out of ICU safely on 3<sup>rd</sup> day of admission due to fast recovery. On the 5<sup>th</sup> day of admission was discharged from the hospital with good health after proper counseling.

Measurement report % M.M.C & H		
OMNI C	15252	
Date,Time	22.04.2018 08:29	
Operator ID		
Sample no.	9560	
Pat ID	NANDINI	
First name		
Last name		
Gender	Unknown	
Sample type	Blood	
Blood type	Arterial	
Baro	767.0 mmHg	
Temp.	37.0 °C	
A/F	adult	
P50	26.7 mmHg	
R	0.840	
FIO2	0.210	
PO2	139.0 mmHg	
PCO2	33.2 mmHg	
pH	7.335	
Na	Slope nOk 1074	
Cl	153.3 mmol/L	
iCa	0.219 mmol/L	
K	3.09 mmol/L	
Hct	24.6 %	
		cHCO3 17.3 mmol/L
		ctCO2(P) 18.3 mmol/L
		SO2(c) 98.8 %
		BE -7.4 mmol/L
		BEecf -8.6 mmol/L
		SB 40.6 mmol/L
		ctO2 21.0 Vol-%
		ctCO2(B) 15.2 mmol/L
		pHst 7.288
		cHCO3st 18.4 mmol/L
		H+ 46.3 nmol/L
		PAO2 139.0 mmHg
		AaDO2 0.0 mmHg
		a/AO2 100.0 %
		RI 0 %
		iCa 0.212 mmol/L
		AG Missing data 1007
		pHt 7.335
		H+t 46.3 nmol/L
		PCO2t 33.2 mmHg
		PO2t 139.0 mmHg
		PAO2t 139.0 mmHg
		a/AO2t 0.0 mmHg
		a/AO2t 100.0 %
		Kit 0 %
		Hct(c) Missing data 1008
		M.M.C Missing data 1007
		React -7.3 mmol/L
		asm Missing data 1007
		a/F Index 662.1 mmHg

Fig. 2

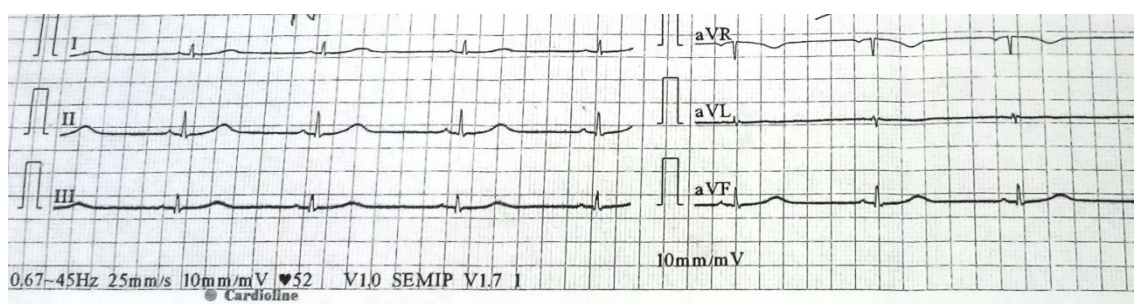


Fig. 3

### Mechanism of Action of the compound

Amitraz exerts its action on central nervous system by stimulation of central  $\alpha_2$  adrenergic receptors and it produce effect in the form of sleepiness, drowsiness or complete loss of

consciousness depending on the dose of toxin consumed. There was a positive correlation observed between the amount of Amitraz consumed and duration of CNS depression. Amitraz exposure causes constriction of pupil<sup>[3]</sup> at

lower doses but may cause dilation at higher doses by stimulation of  $\alpha_2$  adrenergic receptors and its vagomimetic activity produces bradycardia, hypotension and arrhythmias.

Amitraz and its active metabolites inhibit insulin secretion and stimulate glucocorticoid release producing a hyperglycemic<sup>[4]</sup> state.

### Discussion

Amitraz is a pharmaceutical product used worldwide for the treatment of ectoparasitic infestation; it produces toxic effect in animals and humans when ingested, inhaled or after skin exposure. Studies in animals showed that oral lethal dose (LD50) as 523-800mg/Kg in rats and 1600mg/Kg in mice<sup>[5,6]</sup>.

The toxic effect of Amitraz including sedation, bradycardia and hypotension occur due to  $\alpha_2$  adrenergic receptor stimulation and thus its mimics clonidine<sup>[7]</sup> like syndrome. Hyperglycemia was detected in our case as reported in previous studies. However CNS manifestations were more profound in our case. The recovery time from CNS depression took about 16hrs<sup>[8]</sup> with no left over neurological deficit.

The co-existence of Miosis, respiratory depression, bradycardia often mislead physician into diagnosing the patient with OPC poisoning. Certain features point towards Amitraz poisoning as opposed to OPC toxicity. These included presence of hyperglycemia and reduced gastrointestinal motility with absence of fasciculation or hypersecretory state. Serum cholinesterase levels also will be normal which will be usually low in OPC poisoning.

Though there is no specific antidote for this toxin. It has an excellent prognosis even with supportive management if recognized early and given a prompt treatment.

### Conclusion

This case report throws considerable light on the management of Amitraz poisoning with emphasis on good prognosis with early recognition and

timely supportive management. There was no randomized controlled trial available till now hence no conclusions can be drawn on the ideal management strategy for Amitraz poisoning. In our case we would like to emphasize that the incidence of Amitraz intoxication is increasing day by day due to its worldwide use and easy availability. Though there is no specific antidote for this toxin, it carries an excellent prognosis with supportive management.

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