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Case Report: Amitraz Poisoning ;Mimicking Brain Death

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Abstract: *Brain death guidelines should be used with caution in patient with drug intoxication. Poisoning from amitraz is under recognized even in areas where it is widely available. It is known to cause profound CNS depression. We are presenting a case of amitraz poisoning .*

Keyword: *amitraz poisoning ,brain death mimics.*

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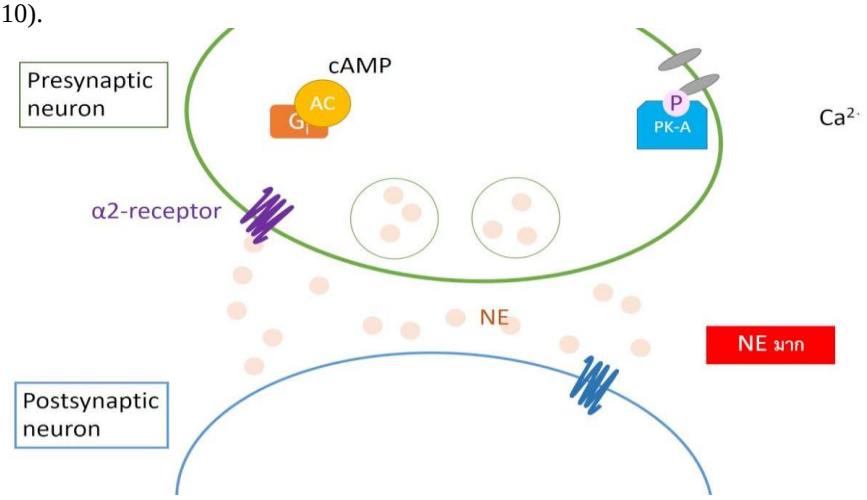
I. Introduction

Amitraz, chemically 1,5 di-(2,4-dimethylphenyl)-3-methyl-1,3,5-triazapenta-1,4-diene is a member of the **formamidine** family of pesticide's(1,4) .It has acaricide and insecticide properties used to control ticks in cattle, sheep, goats and dogs [2]. Commercial formulations generally contain 12.5–20% of the drug in organic solvents, especially **xylene** [3].It acts as an agonist on both pre- and post-synaptic α_2-adrenergic receptors. Presynaptic receptor stimulation inhibits norepinephrine discharge, while stimulation of postsynaptic receptors leads to effects similar to α_1-stimulation.It also acts as a monoamine synthesis and prostaglandin E2 inhibitor(10).



II. Case Report

A 22 year old young male , brought with history of consumption of @10-15ml liquid amitraz . He was immediately taken to local hospital , had one episode of vomiting , intubated within 1 hr. of consumption and referred to our institute with continuous atropine drip .

We received patient in casualty with GCS- 3 /15pupils fully dilated not reacting to light , absent deep tendon reflexes ,dolls eye reflex negative ,no fasciculations ,heart rate of 90/min with continuous atropine drip , BP130/80mmhg .Immediate gastric lavage performed. Patient shifted to ICU. On evaluation(fig2 and 3) SOFA SCORE 6/APACHE2 score was 16.We initially managed him with inj. atropine 20 ml/hr. infusion maintaining heart rate above 70/min .No spontaneous breathing was noted.

Vitals↓ Days →	1	2	3	4	5
1.Pulse	80/min	60/min	90/min	105/min	100/min
2.MAP	103	115	84	78	96
3.RR	16	16	16	20	26
4. SOFA Score	6		4	4	2
5APACHE 2 Score	16		11	10	5
6.GCS	3	3	4	4	10
7.Pupils	DNR	DNR	DNR	DNR	DNR
8.Atropine Infusion	20ml/hr	20ml/hr	20ml/hr	10ml/hr	off
9.Spontaneous Breathing	Absent	Absent	Absent	Present	Present
10.Vasopressor Support	NIL	NIL	Present	Present	tapered off

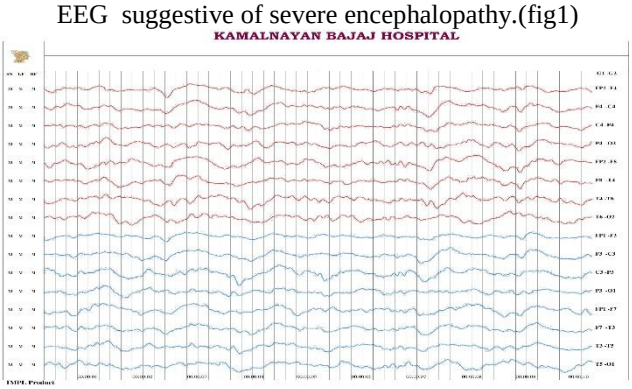
FIG2:Showing clinical parameters of patient , organ dysfunction scores and Intervention required .

Abbreviations:- MAP:-Mean arterial pressure, RR:- respiratory rate, SOFA Score:- Sequential organ failure score, APACHE 2 Score:-Acute physiology and chronic health evaluation 2 score ,GCS:- Glasgow coma scale, DNR:-Dilated not reacting to light

	DAY1	DAY2	DAY3	DAY5	
HB	13.1				
HCT	37.8				
TLC	10500				
PLATELET	1.78				
UREA	19	11	16	33	
CRAETININE		0.8	0.5	0.7	0.7
NA+	142	144	140	145	
K+	2.9	3.5	3.7	3.7	
CL-	110	111	111	110	
LFT	NORMAL			NORMAL	
INR	1.33				
PTT	30/29.99				

FIG3:-Laboratory parameters of patient.

EEG suggestive of severe encephalopathy.(fig1)



His general condition remained same for next 72hr, patient was managed with ventilation and supportive treatment .On third day there was drop in MAP noted , vasopressor support with inj Noradrenaline 8mg 5ml/hr started. On same day response to painful stimulus was noticed .

In MRI brain(fig4) there was evidence of T2,Flair hyperintense in the **rt Globus pallidus** suggestive of toxic encephalopathy . After 84hrs of consumption patient was awake , irritable with GCS 10 /SOFA 4 /APACHE 2 -5.Inj atropine and Noradrenaline tapered off , extubated on 5th day .

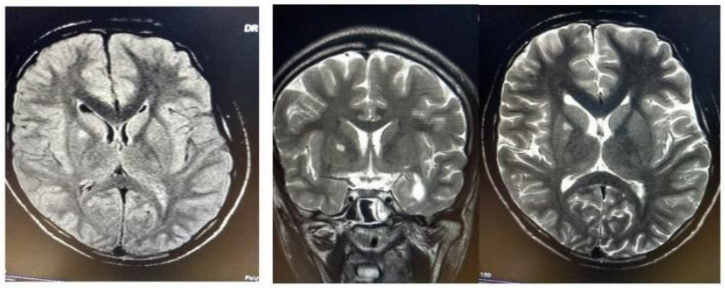


Fig 4 :- MRI Brain:Evidence of T2,Flair hyperintense lesion in the **rt Globus pallidus** suggestive of toxic encephalopathy

On 11th day of hospitalization , As his condition improved , patient had given discharge.

III. Discussion

Sullvian et al 2012reported a 40-year-old woman, was brought following with drug overdose ?baclofen. Several days after admission ,she was declared brain dead and scheduled for organ donation. She was taken for organ harvesting, but she opened her eyes in OT.(9).

J. Chakraboy et al in 2011 reported one case with amitraz poisoning. they received patient with deterioration of sensorium progressing to deep coma within few hours after consumption of the poison.(7)

It can cause poisoning in animals and humans when ingested, inhaled, or after skin exposure(4) The minimal toxic dose previously reported was 3.57 mg/kg.(1) With this clinical presentation ,in the EPA classification Amitraz is included as Class III – slightly toxic.(5)

Shitole et. al .CNS depression which is probably attributable to alpha 2 – adrenoreceptor stimulatory action was the prominent signs in our cases, symptoms began within 2 hours and resolved within 18 hours.(5)
Brain death should not even be thought of, until the following reversible causes of coma have been excluded.(8)

1.**Intoxication (alcohol), drugs** including muscle relaxants which
depress the central nervous system (CNS)
2. primary hypothermia,
3. metabolic and endocrinal disorders

Central nervous system (CNS) depression was the most commonneurological abnormality in amitraz poisoning(fig5). Almost all patients regain consciousness by 48 hr. This is possibly due to the short elimination half-life..(10)
There is no antidote, animal studies have demonstrated α2-adrenergic antagonists such as yohimbine and atipamezole can reverse most of the clinical and laboratory signs (11).
It got good prognosis with supportive management .(6) Though activated charcoal is relatively safer but the clinical benefit is again uncertain. Atropine used to treat symptomatic bradycardia in many of the patients, sometimes dopamine for bradycardia(1,3).
Role of Naloxone has been successfully explained in clonidine poisoning (α2-adrenergic agonist) but has proved to be ineffective in animal studies of amitraz poisoning(12)

IV. Conclusion

Amitraz is an uncommon source of poisoning, but it could be fatal in very small amount (10-15ml), close to brain dead, continuing supportive management , can improve survival in most patients.

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