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"ATROPINE INDUCED PSYCHOSIS: A CASE REPORT OF MANAGEMENT IN ORGANOPHOSPHATE POISONING"

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Abstract: Atropine, a naturally occurring alkaloid derived from plants of the nightshade family, is commonly used in the treatment of various conditions such as organophosphate poisoning, mydriasis induction, and pre-medication for anaesthesia. While atropine is generally safe, its administration can lead to rare allergic or toxic reactions. We present a case of atropine-induced psychosis in a 29-year-old male patient who accidentally consumed an organophosphate compound, allegedly due to dispute with his spouse.

The patient presented with excessive salivation and altered sensorium, and subsequently experienced vomiting and altered consciousness. Immediate medical attention was sought, and the patient was intubated for respiratory support. In the intensive care unit, the patient received decontamination procedures and mechanical ventilation to manage respiratory distress. Laboratory tests revealed features consistent with organophosphate poisoning, including relative neurocytosis and increased serum cholinesterase levels.

Supportive care was initiated with intravenous fluids and oxygen therapy, while continuous monitoring of vital signs, glucose levels, and symptoms of cholinergic excess (SLUDGE) was conducted. Atropine was administered at a rate of 5 ml/hour to maintain the pulse rate, and intravenous pralidoxime (PAM) was given as an antidote. On the following day, the patient developed symptoms of atropine-induced psychosis, including disorientation, agitation, dilated pupils, tachycardia, and decreased urination with blood contents, muscle twitches, jerks, and delirium.

The patient's condition was managed by gradually tapering the atropine dose and discontinuing it after signs of complete atropinisation. Medications such as midazolam and vecuronium were administered to relieve agitation and anxiety, while haloperidol was given for managing psychiatric effects. Prophylactic treatment with antiulcerants and antibiotics was also administered. Atropine was completely stopped on the tenth day, and the patient was discharged in a stable condition.

Atropine remains the mainstay for pharmacological management of organophosphate poisoning, with dosage adjustments made to maintain vital signs and prevent respiratory complications. Toxic reactions to atropine can occur due to its anticholinergic effects, leading to various peripheral and central manifestations. Adverse effects are dose-dependent and usually reversible upon discontinuation of therapy. Understanding the potential toxic effects and idiosyncratic reactions to atropine is crucial for appropriate management in cases of poisoning.

Index Terms Atropine, organophosphate poisoning, atropine-induced psychosis, case report, management

I. INTRODUCTION

Atropine, derived from plants like Atropa belladonna, is a natural alkaloid that acts as an antagonist to acetylcholine's muscarinic-like actions in the central and peripheral nervous systems. Its major action involves competitive or surmountable antagonism (1).

Atropine is an anticholinergic agent administered via various routes, including IV, IM, ophthalmic, and oral. FDA-approved uses include amblyopia treatment, cardiac arrest management, cycloplegic refraction, mydriasis induction, organophosphate poisoning treatment, pre-medication for anesthesia, and managing toxic effects from consuming mushrooms (2). Although rare, the administration of atropine to a large population for intoxication treatment carries the risk of allergic or toxic reactions, as reported in the literature (3). In cases of poisoning caused by organic phosphate cholinesterase inhibitors found in certain insecticides and chemical warfare nerve gases, high doses of atropine alleviate muscarinic-like symptoms and some central nervous system manifestations. Atropine's CNS effects include headache, flushing, nervousness, drowsiness, weakness, dizziness, and insomnia (2).

This case report discusses a patient who experienced atropine-induced psychosis following a suspected poison ingestion and the subsequent management of the condition.

CASE PRESENTATION:

A male patient of 29 years old weighing 55 kg admitted to emergency department with chief complaints of excessive salivation, altered sensorium.

On diagnosis, based on medical history it was found that patient has accidental consumption of organo phosphorous compound (?malathion), at fields due to an dispute with the spouse. After consumption patient was conscious for some time later family members identified frothing and took him to the hospital.

During hospitalization he has 3-4 episodes of vomiting, and altered sensorium, so, he got intubated for oxygen.

As an initial phase, primary care was given by decontaminating the patient and simultaneously the trachea was intubated. Patient was shifted to ICU and his respiration was supported with mechanical ventilation manage the respiratory distress. Continuous cardiac monitoring and pulse oximetry was established; an ECG was performed.

Vital signs at time of admission are as follows:

BP-140/90 mm of hg PR-120 bpm RR-20 CPM GCS-E2/3V7M

Patient's vital signs revealed hypertension with BP 140/100mmHg.Rest of the physical examination was normal except for sluggish breath sounds. Features were consistent with organophosphate poisoning and the patient was managed conservatively.

Haematologic analysis revealed relative neutrocytosis, and serum cholinesterase shows increased levels, while the other parameters were normal.

The laboratory results at the time of admission.

S.NO	LABORATORY TEST	RESULT	
1	HB	14g/dl	
2	PCV	42%	
3.	TLC	17000cells/mm	
4	RBC	4.50m/cumm	
5	S.CPK	818mcg/l	
6	S.CHOLINESTERASE	13495u/l	
7	CREATININE	0.68mg/dl	$\mathbf{a} > \mathbf{a}$
200 L		10	

Initially the patient was started with supportive care using IV Fluids and oxygen therapy.Pulse oxymetry, Blood Glucose levels, Cardiac parameters and symptoms of SLUDGE (salivation, lacrimation, urination, defecation, GI cramping, Emesis) were constantly monitored. Repeated bolus doses of atropine was administered at a rate of 5 ml/hour to maintain pulse rate.IV Pralidoxime (PAM) 500mg in 100ml of normal saline was administered every 8 hourly as an antidote.

On next day patient started experiencing symptoms of disorientation, agitation, nonresponsive dilated pupils, tachycardia (>130bpm),decreased urination with blood contents and sudden muscle muscle twitches and jerks, delirium, muscle weakness. Based on these symptoms developing following the atropine administration, a diagnosis of atropine induced psychosis was made. Casualty assessment of atropine induced psychosis was done using Noranjo Casualty assessment scale and WHO-UMC scale.

The patient was appropriately managed by tapering the dose of atropine to about 1ml/hr and discontinued after the appearance of signs of complete atropinisation. Midazolam 25mg IV +inj.Vecuronium 25mg was administered to relieve agitation and anxiety. Inj.haloperidol 1mg SOS was given for managing the psychiatric effects. Antiulcerants (pantoprazole-40mg IV OD) and Injection Piptaz(piperacillin/ tazobactum) 4.5gm IV TID was administered as prophylactic. Atropine was stopped completely

on the tenth day and the patient was discharged in a much stable condition.

DISCUSSION:

Atropine is the primary pharmacological intervention for the management of organophosphate (OP) poisoning. The dosage of atropine is tailored to each individual, aiming to maintain a heart rate above 80 beats per minute, systolic blood pressure above 80mmHg, and prevent bronchorrhea or bronchospasm (4).

Adverse reactions to atropine stem from its anti-cholinergic properties and can present with diverse peripheral and central symptoms. Even at standard therapeutic doses, toxic effects may occur due to substantial inter-individual variations in susceptibility to atropine (idiosyncrasy). Consequently, signs of overdose may manifest despite doses falling within the usual therapeutic range. The variability in atropine toxicity is exemplified by reported cases where adults have experienced fatalities

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with doses as low as 100 mg (and 10 mg in children), while others have successfully recovered from intoxication following a 1 g dose of atropine (3).

Most adverse effects are associated with the antimuscarinic action of atropine and are typically reversible upon discontinuation of treatment. The frequency and severity of these adverse effects correlate with the dosage administered. Serious adverse effects primarily arise from excessive doses resulting from single or repeated injections of atropine (1).

CONCLUSION:

When using atropine for the treatment of organophosphate intoxication, it is important to exercise caution in prescribing doses, particularly with IV administration. Timely withdrawal of the causative drug is crucial to prevent further complications. In cases of atropine-induced psychosis, benzodiazepines and antipsychotics are commonly employed for treatment. In the present case, symptomatic management is appropriate, considering the patient's history of substance abuse. Long-term therapy is not necessary, as anticholinergic toxicity is expected to resolve within a few days after discontinuation of the offending agent.

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