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A Systemic Review of Organophosphorus Poisoning by Analyzing the Case Report

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ABSTRACT

Introduction: Poison is any substance that obstructs with ordinary body functions and is capable of affecting adverse effects in living organisms. Self-poisoning from organophosphorus [OP] pesticides is a significant clinical issue in rural areas of developing countries and is responsible for an estimated 2,00,000 fatalities annually. Suspicion recognizable clinical symptoms the odour of pesticides or solvents and decreased butyrylcholinesterase activity in the blood are used for diagnosis.

Case Study: A 30 years old female patient was admitted in a Tertiary Care Hospital, with an alleged history of dimethoate compound poisoning. On arrival the patient was drowsy responds to painful stimuli and afebrile. The treatment was begin with Gastric lavage with normal saline Inj.Pralidoxime in an intravenous route and Atropine was also administered intravenously and repeated every 5 minutes until the pupil dilated and Ranitidine was given to prevent ulceration.

Discussion: The initial method to treat organophosphorus poisoning is to decontaminate the patient by removing and destroying all clothing and using drying agent such as flour sand or bentonite. The patient's irrational usage of medication therapy causes a worsening of the condition and a 10- to 15-day hospitalization.

Conclusion: As per the standard treatment guidelines, atropine should be given as first-line therapy because muscarinic effects are reversed by atropine. By providing this to the patient, you will minimise the severity of their illness minimise down on their stay in the hospital and ultimately save their lives.

KEYWORDS: Organophosphorous, Pralidoxime, Atropine, Muscarinic effect, Nicotinic effect.

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INTRODUCTION

Poison is any substance, which obstructs with ordinary body functions and is capable of affecting adverse effects in living organisms. Poisoning can take place by means of ingestion, inhalation or contact purposefully or accidentally that causes injury or damage to the body. Self-poisoning from organophosphorus[OP] pesticides is a significant clinical issue in rural areas of developing countries and is responsible for an estimated 2,00,000 fatalities annually. Organophosphates are utilized as medicines, pesticides and weapons –grade nerve agents . Often within minutes, and it can take upto a week for symptoms to go away. The amount consumed, the methods of absorption, and the pace of metabolic breakdown of the pesticides all affect how severe the symptoms are[1][7].

Anticholinesterases, which are composed of organophosphorous chemicals, significantly increase morbidity and mortality in India. These anticholinesterases cause three distinct neurological syndromes: the first, a life-threatening acute cholinergic crisis that frequently requires management in an intensive care unit; the second, an intermediate syndrome, in which patient frequently requires respiratory support due to the prevalence of cranial nerve palsies, proximal muscle weakness, and respiratory muscle weakness; and the third, a delayed organophosphate –induced polyneuropathy[2].

Suspicion, recognizable clinical symptoms, the odour of pesticides or solvents, and decreased butyrylcholinesterase or acetylcholinesterase activity in the blood are used to make the diagnosis. Several organophosphates have a characteristic petroleum or garlic order that can also aid in diagnosis. A trial

of atropine may be used if poisoning by organophosphates is suspected but not verified. Moreover, blood tests for CBC, glucose and troponin levels, renal function, and arterial blood gas should be requested. A portable that can measure is available now. Sinus bradycardia due to PS activation will be shown on the ECG[1].

MECHANISM OF TOXICITY

Acetylcholinesterase (AChE), which is present in synaptic connections and red blood cells, and butyrylcholinesterase (also known as pseudocholinesterases (PChE) or plasmacholinesterases), which is present in the blood, are two enzymes that organophosphorus pesticides inhibit. Acetylcholine is broken down by each of these enzymes. Acetylcholine is hydrolysed by acetylcholinesterase into two fragments acetic acid and choline[3]. Blood artery dilatation, a slowed heartbeat, bronchiole constriction, and in severe cases, respiratory failure that results in death are some of the repercussions[4].

1. The most clinical significant impact of organophosphorus poisoning is ACh blockade because it causes excessive acetylcholine buildup at muscarinic receptors, nicotinic receptors, and in the central nervous system (CNS).
2. Whenever the OP binds covalently to the enzyme, ACh may be permanently inhibited. Depending on the exposure pathway and the particular OP, the pace of ageing might vary from a few minutes to days. In general, dimethyl OP compounds (such as DIMETHOATE) age longer than diethyl agents (Eg: CHLORPYRIFOS)[3].

CLINICAL FEATURES:[5]

MUSCARINIC RECEPTOR	NICOTINIC RECEPTOR	CENTRAL RECEPTOR
Cardiovascular	Cardiovascular	General effects
Bradycardia	Tachycardia	Anxiety
Hypotension	Hypertension	Restlessness
		Ataxia
Respiratory	Musculoskeletal	Convulsions
Rhinorrhoea	Weakness	Insomnia
Bronchorrhoea	Fasciculations	Dysarthria
Bronchospasm	Cramps	Tremors
Cough	Paralysis	Coma
		Absent reflexes
Gastrointestinal		Respiratory depression
Nausea		Circulatory collapse
Vomiting		
Increased salivation		
Abdominal cramps		
Diarrhoea		
Facial incontinence		
Genitourinary		
Urinary continence		
Eyes		
Blurred vision		
Increased lacrimation		
Miosis		
Glands		
Excessive salivation		

CASE STUDY

A 30 years old female patient was admitted in a Secondary Care Hospital, Perambalur with a alleged history of dimethoate compound poisoning and the chief complaints precipitated were drowsiness, garlic like odour from her mouth and increased lacrimal and mouth secretions results in slurred speech. On arrival, the patient was drowsy, responds to painful stimuli and afebrile. She had a Heart Rate of 136 bpm, Blood pressure of 103/83 mmhg and non-reactive pupils. Lab investigations such as Platelets, Differential count, Red Blood Cell and Liver Function Test (LFT) reflects normal level. Patient Haemoglobin level

founds to be declined than the normal level and founds to be 9.8 g/dl. The treatment was begin with Gastric lavage with normal saline of 5 pint and Inj. Pralidoxime 2 gm in IV. After the stomach wash, Atropine 5 amp was administered intravenously and repeated every 5 minutes until the pupil dilated, Heart rate is decreased to 121 bpm and Inj.Ranitidine 50 mg was given in IV. The time duration for each cycle is 10 min and examination was done for 3 cycles and the number of cycles depends on the patients clinical condition.While checking at first cycle, suddenly patient went to cardiac arrest. Patient was gasping and not oriented and not respond to painful stimulus. So, CPR was started, bag and mask ventilation given with Inj. Adrenaline and Inj. Atropine given. Patient was not responded. During second cycle, patient was not responded. So, CPR started with bag and mask ventilation and Inj. Adrenaline and Inj. Atropine was given. Patient was not responded. During third cycle, CPR started with Bag and Mask Ventilation given with Inj. Adrenaline and Inj. Atropine given. Patient was not responded.

DISCUSSION

The initial method to treat the patient with organophosphorus poisoning is the personal protective equipments should be used by the health care professionals treating the poisoned patient. The next step is to decontaminate the patient which means removing and destroying all clothing because it may be contaminated even after washing[6][8]. The patients skin needs to be flushed with water. Dry agents such as flour, sand or bentonite also can be used to decontaminate the skin. Activated Charcoal can be given if the patient presents within 1 hour of ingestion. The antidotes Pralidoxime and Atropine are approved by the WHO to be used after OP pesticide poisoning[6].

Gastric lavage with 5% sodium bicarbonate may be given if swallowed[9]. Atropine is given with 2 to 5 mg IV initially and double the dose administered every 5 minutes. Atropine will reverse muscarinic but not nicotinic effects[3]. The most important indication for redosing atropine is persistent wheezing or bronchorrhea. Pralidoxime is an oxime that reactivates the cholinesterase enzymes when before enzyme aging. Within 48 hours of ingestion, Pralidoxime is administered[8]. It should be given as a loading dose (30 to 50 mg/kg total of 1 to 2 in adults) over 30 minutes. Followed by continuous infusion of 8 to 20 mg/kg/hr (upto 650mg/hr).It is most effective if started early, before irreversible phosphorylation of cholinesterase occur (aging), may still effective if given late[3].

Pralidoxime has three main outcomes:

- ✓ A direct reaction converting the organophosphates to harmless compound
- ✓ A transient reaction protecting the enzyme from further inhibition
- ✓ Reactivation of the inhibited alkyl phosphorylated enzyme to free the active unit

The patient received the following pints of normal saline, 2 gm of injectable pralidoxime, and a stomach wash at the same time. After that, the patient was treated Inj. Atropine 5 amp. We draw the conclusion from this case study that atropine should be taken prior to pralidoxime because OP drugs primarily affect muscarinic receptors. Hence, we can minimize the muscarinic effects by giving atropine initially.

CONCLUSION

According to ROBB EL, atropine must be administered before pralidoxime in order to prevent the exacerbation of muscarinic symptoms. The muscarinic action will be reversed with atropine. As per the recommended course of treatment, atropine injections are given every 10 minutes until atropinism symptoms manifest. The drying of all secretions, restlessness, tachycardia, dryness of tongue, dilated pupils are the symptoms of atropinism. Give Inj. pralidoxime right away in situations of moderate to severe disease[10]. The patient's irrational usage of medication therapy causes a worsening of the condition and a 10 to 15 day hospitalization. As per the standard treatment guidelines, atropine should be given as first-line therapy because muscarinic effects are reversed by atropine By providing this to the patient, you will minimise the severity of their illness, minimise down on their stay in the hospital, and ultimately save their lives. In the event of a suspicion of OP poison, the treating physicians have to proceed with wariness and must deliver the proper therapy in accordance with the standard recommendation.This instance emphasis the significance of understanding complicated drug therapy administration in an era of growing irrational drug therapy practices and the discouragement that can occur when these administration disregarded.

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