



Case report on impending respiratory failure due to alleged organophosphorus compound poisoning (CHLORPYRIFOS)

Pilla Sai Sushmitha^{*1}, Chadaram Pragnatha¹, Divya Pathivada¹, Sandhya Vanapalli¹, Reethika.V²

^{*1}Department of Pharmacy Practice, Avanthi Institute of Pharmaceutical Sciences, Vizianagaram

²Clinical Research Coordinator, AIG Hospitals

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Abstract

In many parts of the world, particularly in impoverished nations, organophosphate compounds in few incidences operate as suicide agents, are frequently employed as pesticides which are strong inhibitors of the acetylcholinesterase enzyme. A 43-year-old male, was admitted to an emergency department at 9:00am, with a two-hour history of nausea and elevated secretions of salivation via the mouth. He had no previous history of psychiatric or neurological disorders but with extreme causes of Respiratory Failure due to COVID-19. On three days before his admission, he quarrelled with his wife, became extremely depressed, and decided to commit suicide. He had a two-hour history of nausea and intermittent vomiting, diarrhoea and a one-hour history of persistent increased vomiting, increased salivation secretions through the mouth, chills, progressive sweating, difficulty breathing, and dizziness. Upon admission, his neurological examination in the emergency department revealed a Glasgow Coma Scale score of 9/15. On admission, he was placed on two litres per minute of intranasal oxygen via the nasal cannula. On the same day, he was treated by gastric lavage and administered atropine 0.15 mg intravenously, and the dose was doubled every 10 minutes until atropinisation was achieved, and a bolus dose of 500ml of 0.9% of normal saline was initiated immediately.

Keyword: Chlorpyrifos, Atropinisation, gastric lavage, Acetylcholinesterase.

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*Corresponding Author

Pilla Sai Sushmitha

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Introduction

Throughout the world, an estimated 3 million people are exposed to organophosphates (OP) or carbamates yearly, with up to 10% (300,000) fatalities [1]. OP compound poisoning is one of the serious health problems faced in worldwide, it ranks 45th in the total death in the world. The highest incidence has been reported from exposure of Agriculture Pesticides- [44%], House holding agents [38%], and Followed by drugs [11.8%]. OP poisoning is one of the most common causes of poisoning in developing countries like India and has been used for intentional self-poisoning. In India, the exact numbers of cases or fatalities are difficult to calculate due to an unavailability of databases or a poison surveillance system [3]. Poisoning occurs mostly by voluntary ingestion, inhalation or by absorption

through the skin. (5)OP poisoning may manifest acutely with the cholinergic crisis, respiratory distress, and intermediate syndrome or with delayed toxicity.

OP acts by blocking the activity of acetylcholinesterase thus stimulating cholinergic as well as nicotinic receptors. Cholinergic effects can be reversed by atropine but for most neurological manifestations, which are mediated through nicotinic effects [12].

OP can have an effect on other organ systems which although rare, can worsen the presentation and prognosis of the patient including respiratory Failure.

Respiratory failure is a serious condition that makes it difficult to breathe on your own. Respiratory failure develops when the lungs can't get enough oxygen into the blood. We breathe oxygen from the air into our lungs, and we breathe out carbon dioxide, which is a waste gas made in the body's cells.

Case Report

A 43 years old male was transported by an ambulance escorted to the emergency department [ED] with the chief complaint of intake of CHLORPYRIFOS with

unknown quantity patient presented with shortness of breath, drowsiness, blurred vision, vomiting, 2 – 4 episodes of diarrhoea, increased salivation secretions through the mouth, chills progressive sweating. At 5:30 am, He drank organophosphate insecticide [chlorpyrifos]. He had no previous history of psychiatric or neurological disorders but, with an extreme causes of respiratory failure due covid-19 on 3 days before his admission, he quarrelled with his wife, became extremely depressed and decided to commit suicide. He arrived at the hospital with nausea and sporadic vomiting as well as an hour of persisting vomiting. On examination -Increases salivation through the mouth, chills, progressive sweating, breathing difficulties and dizziness. Gastric lavage was done right away. On admission the vital signs and laboratory testing & physical examination he had a mean arteriole blood pressure of 90/60 mmHg, a pulse rate of 68 beats per minute, a body temperature of 101°C ,an oxygen saturation level of 94% with RA, and a respiratory rate of 19 breaths per minute.

Days	Blood pressure mmHg	SPO2%	Respiratory rate[BPM]
Day 1	90/60	94 %	11
Day 2	100/70	96%	14
Day 3	120/80	98%	19

laboratory investigation of the patient revealed blood urea nitrogen of 26.5 mg/dl [normal value: 6-21 mg/dl], serum creatinine of 0.6mg/dl [normal value: 0.59 – 1.04mg/dl], serum phosphate level of 2.9mg/ml [normal value 2.8-4.5mg/dl], serum sodium level of 141mEq/L [normal value 135-145mEq/L] and serum potassium level of mEq/L [normal value 3.5-5.5mEq/L], white blood cells count 13200 cells/mm³ [normal value 4500-1100 cells/mm³]. His Glasgow coma scale reading was 9/15 upon admission, according to the emergency department neurological evaluation during a physical examination it was discovered that he had heavy sweating, saliva dripping from his mouth his stomach was smooth with normal bowel sounds on auscultation, but his pupil were constricted in both direction, he was

sleepy with dyspnoea and there was noticeable discomfort near the epigastrium

He was intubated with mechanical ventilation 8hours later for hypotension and profound dyspnoea. He was started on 2lits of intranasal oxygen per minute upon admission via a nasal cannula. He received atropine 0.15mg intra venous on the same day with the dose being doubled every 10mins to achieve atropinization and then a bolus dose of 500ml of 0.9% with normal saline was started right away. He required ventilator support for one week and after 9 days he has recovered completely.

Medication chart was shown in the below table:

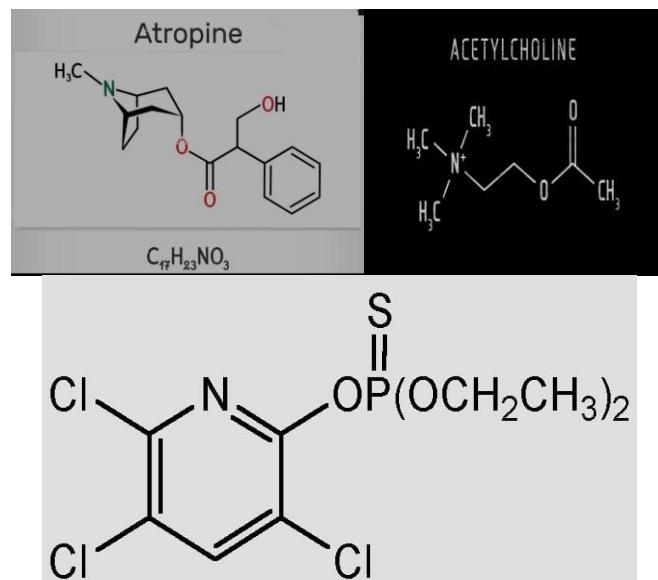
Trade Name	Generic Name	Direction	Dose/ Frequency	Purpose
INJ. Atropine	Atropine	IV	48 mg in 10 NS @100ml/h r(OD)	Anticholinergic drug
INJ. PAM	Pralidoxime	IV	1 g in 100ml NS (BD)	Anticholinergic drug
INJ. PANTOP	Pantoprazole	IV	40 mg (OD)	Antacid/ proton pump inhibitor
INJ. Ceftriaxone	Ceftriaxone	IV	1 g (BD)	Antibiotic
INJ. ZOFER	Ondansetron	IV	4 mg (TID)	Antiemetic
INJ. Haloperidol LA	Haloperidol	IM	1cc (SOS)	Antipsychotic
Inj. PCM		IV	1gm(SOS)	

Discussion

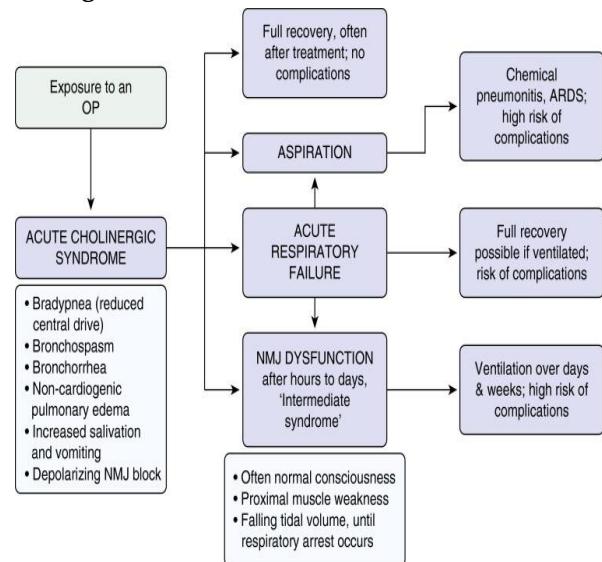
Organophosphorus poisoning is most common in developing countries especially in India. Mainly poison occurs by voluntary ingestion, inhalation or absorption through the skin. By self-ingestion through intramuscularly or intravenous route it can cause toxicity. On other hand OP poisoning occur through parenteral route has been described by very few authors. Diagnosis becomes difficult when the victim's

history of parenteral administration of OP compound is not available. OP pesticides are most used for suicidal purpose it can cause poison through occupational exposure while spraying in field is an important mobility of poisoning [11]. Chloryrifos, also known as chloryrifos pesticides that has been used on crops, animals and buildings, and in other stings, to kill a number of pests, including insects and worms. It acts on the nervous systems of insects by inhibiting the acetylcholinesterase enzyme [4]. Chloryrifos is considered moderately hazardous to humans (class II) by the world health organisation based on acute toxicity information dating to 1999 [7]. Exposure surpassing recommended levels has been linked to neurological effects, persistent development disorders, and autoimmune disorders. Exposure during pregnancy may harm the mental developmental of children [5]. In the United Kingdom, the use of chloryrifos was banned as of 1 April 2016 [6]. As of 2020, chloryrifos and chloryrifos methyl were banned thoroughly the European union, the EU also applied to have chloryrifos listed as a persistent organic pollutants (8). As of August 18, 2021 the US environmental protection agency (EPA) announced a ban on the use of chloryrifos on food crops in the United States (10). Most home uses of chloryrifos has already been banned in the US and Canada 2001 [9]. It is banned in number of other countries and jurisdictions as well. The chloryrifos ban on food crops is the result of a 1999 lawsuit filed by NRDL to force the EPA to take action on the riskiest pesticides, as well as five additional successful court orders obtained by earth justice to force the EPA to take action on a 2007 petition to ban chloryrifos filed by NRDC and the pesticides action network of North America (PANNA) [9, 11]. Primarily, chloryrifos and organophosphate pesticides interfere with signalling from the neurotransmitter acetylcholine. One chloryrifos metabolite, chloryrifoxone, binds permanently to the enzyme acetylcholinesterase, preventing this enzyme from deactivating acetylcholine in the synapse [2]. By irreversibly inhibiting acetylcholinesterase, chloryrifos leads to a build-up of acetylcholine between neurons and a stronger, longer-lasting signal to the next neuron. Only when new molecules of acetylcholinesterase have been synthesized can normal function return [13]. Acute symptoms of chloryrifos poisoning only occur when more than 70% acetylcholinesterase molecules are inhibited. Once AChE has been inactivated, ACh accumulates

throughout the nervous system, resulting over stimulation of muscarinic and nicotinic receptors [14]. The clinical presentations and the outcome of OP poisoning depend not only on the pesticide but also one the dose of the victim's taken.



Pathogenesis



Conclusion

In phase of typical symptoms such as those described for our patient they should be a high suspicion of organophosphorus exposure even when the patient family is not aware of it. People should be educated about poisoning hazardous and should be aware about the poisoning. Clinical and healthcare professional should provide safety measures in rural place, how to prevent and give some information about the emergency condition and to establish, conduct poison

information centres with people who lack in knowledge regarding toxicity. Patient should interact with family members to overcome depression and to educate about the Op poisoning. To maintain a stable diet and medication until recovery. If not may have the chances of respiratory attack or failure.

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