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Case Report Open Access

Organophosphate (OP) Poisoning Case Report by the Ingestion of a Potential Lethal Dose; its Management and Appropriate Protocol

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Abstract

Organophosphate (OP) poisoning continues to be a recurrent cause for admission to hospitals and intensive care units (ICU) in under emergent countries. OP poisoning is the most commonly prevailing up to the ratio of (27.64%) and has the highest death ratio (13.88%) of poisoning in Asia. This poisonings causes up to 25% mortality rate worldwide. In this case presentation a young girl age was sent to ICU after ingestion of OP insecticide. Where medical practitioner prescribed some irrational medications which are of veto use and also don't follow customary protocols for treating this poisoning case, the main reason behind this miss hap would probably be the lack of pharmacist intervention in health care team. This is the major drawback of our public sector hospitals in Karachi, Pakistan. Responsiveness and right treatment protocols can trim down both mortality and morbidity rates in the city and prompt appropriate therapeutic dealings can execute better prognosis in these types of urgent situations and may decreased further impediments.

Keywords: Organophosphate poisoning; Case study; Lack of pharmacist; Irrational medications

Introduction

Organophosphorus pesticide self-poisoning is an important concern in rural regions of the developing country, and kills an estimated 200,000 people every year. Accidental poisoning ratio far less but is also a dilemma in places where highly toxic organophosphorus pesticides are available. Organophosphate (OP) compounds have a vast variety of chemicals used in both domestic and industrial settings. Some examples of OP include insecticides, nerve gases, ophthalmic agents and antihelmintics. [1-5].

Signs and symptoms of OP poisoning

These are divided into three different categories.

- Muscarinic effects
- Nicotinic effects
- Central nervous system (CNS) effects

Muscarinic effects of OP are (excess salivation, lacrimation, urination, emesis, GI upset, diaphoresis, diarrhea; urination; miosis; bradycardia, bronchospasm, and salivation).

Nicotinic signs and symptoms include muscle fasciculations, cramping and weakness. Autonomic nicotinic effects include hypertension, tachycardia, mydriasis, and pallor. CNS effects include the following: Anxiety, Emotional liability, Restlessness, Confusion, Ataxia, Tremors, Seizures, and Coma.

The key mechanism of action of OP pesticides is inhibition of carboxyl ester hydrolases, mainly acetylcholinesterase (AChE). AChE is an enzyme that is used to degrade the neurotransmitter acetylcholine (ACh) into choline and acetic acid. ACh is found in the central and peripheral nervous system and accumulates throughout the nervous system, resulting in overstimulation of nicotinic and muscarinic receptors [6-8].

Case presentation

A young 20 years old girl was escorted to intensive care units after intake of OP insecticide in a suicidal attempt. She had shortness of breath, decreased altered level of consciousness, diarrhea, miosis, hypersalivation and restlessness and seizures during admission. Vital signs show pulse rate of 62 per min and blood pressure of 120/80 mmHg while respiratory rate of 14 per min [9,10].

Brand	Generic	Dose/frequency	Direction	Purpose
Contrathione	Pralidoxime methylsulphate	1 g/tid	IV	Cholinesterase reactivators
Ruling	Omeprazole	40 mg	IV	Acidity/indigestion
Atropine	Atropine	10 mL/h	IV	Antidote of OPP
Dayline	Ceftriaxone	750 mg	IV	Treatment of infection

Levofloxacin	Levofloxacin	750 mg/OD	IV	Respiratory tract infection
D/W 5%	Fluid	IV 1000 mL	Infusion	Fluid replacement

Table 1: Current Medication Chart.

Chest X-ray showed acute respiratory distress. After immediate resuscitation, she was treated with atropine and pralidoxime methylsulphate along with broad spectrum antibiotics. Atropine was given 10 mL/h through IV and the dose was titrated as per her clinical response. The patient also received pralidoxime. Pralidoxime was given at a dose of 1 g infusion, three times per day for initial two days. While on maintenance, features of toxicity re-appeared and she again required atropine in bolus dose. She required ventilator support for one week and after 9 days she recovered completely. Her current medication chart was shown in Table 1.

Patient's Laboratory and Diagnostic Data

Diagnosis

OP poisoning.

Major Strategies: The standard protocol for OP poisoning was not follow. She was not treated with phenytoin or diazepam sodium for seizures. No medication was given for her restlessness. Her restlessness controlled by diazepam.

Initial antibiotics were ceftriaxone and levofloxacin but during the course of illness, there were deterioration of chest shadows and antibiotics should be changed to meropenem and linezolid.

There was also a foremost drug interaction exited between amikacin and levofloxacin.

Discussion and Conclusion

Medical management of organophosphorus pesticide poisoning is difficult, especially with having fewer resources of underprivileged localities. Clinical practice is frequently less than ideal, with pitiable initial resuscitation and stabilization, and deprived use of antidotes. However, most of the research regarding acute organophosphorus poisoning in humans has been published in last decade, which is a positive development. We expect that in the next decade continuing research by a number of groups will finally provide clear guidance on how to treat organophosphorus pesticides poisoning.

Initial stabilization

Severe acute seizures are a medical emergency. Medical practitioners must ensure that the patient has adequate breathing. Ideally, oxygen should be provided on the primary thing. On the other hand, little verification ropes that the common suggested drug atropine by the doctors should not be given until oxygen is available. While in those hospitals that have veto access to oxygen, atropine should be given early to patients. The patient should be placed in the left lateral position, with the neck extended. This position helps keep the airway patent, and could decrease pyloric emptying and absorption of poison.

Decontamination

Take out all the clothing's from the patient and gently cleanse the patients with soap and water because OP are hydrolyzed readily in aqueous solutions. Health care providers should keep themselves away from contamination or utilize proper preventive protocols while handling the patients. Use proper protective substances like gloves and gowns, when decontaminating those patients. Use masks for respiratory protection. Wash eyes of the patients using isotonic sodium chloride solution. Intraosseous administration has been found useful in rapid delivery of atropine into the bloodstream, as shown in the studies of pigs [11,12]. Intravenous glycopyrrolate or diphenhydramine may also provide another centrally acting anticholinergic agent used to treat muscarinic toxicity if atropine is unavailable.

Principles of therapy

Treatment protocol includes first recovery of patients, giving oxygen, a muscarinic antagonist (usually atropine), fluids, and an AChE reactivator (pralidoxime that act as ChE re activator by removal of the phosphate group). Gastric decontamination should be considered only after the patient has been fully resuscitated and stabilized. Patients must be carefully observed after stabilization because of intermediate syndrome, and intermittent cholinergic features occurring with fat-soluble organophosphorus.

Atropine acts as physiological antidote as it antagonizes muscarinic mediated events. Atropine preliminary loading dose is 2-5 mg and will repeat every 5-10 min until signs of atropinisation appear. Following this, given infusion at the rate of 0.02-0.08 mg/kg/min and the dose is titrated as per the clinical response. Pralidoxime is generally used in the dose of 1 g every 6-8 hours; current studies have shown better outcome with high-dose infusion, 18-24 g/day. Organophosphorus pesticide poisoning induced seizure if treated with diazepam.

Sufficient oxygenation and airway control are imperative in OP poisonings. Intubation may be necessary in cases of respiratory distress because of laryngospasm, bronchospasm or bronchorrhea. Immediate use of atropine may eliminate the need for intubation. Succinylcholine should be keep away because it is degraded by plasma cholinesterase and may result in prolonged paralysis.

Different researches show drug interaction between levofloxacin and amikacin. Drug interactions are exceptional with levofloxacin; nevertheless, co administration with antacids or with other agents containing divalent or trivalent cations reduces levofloxacin absorption. The agent should prove to be more effectual than older fluoroquinolones, particularly for infections caused by pneumococci as it's highly resistant to penicillin [13].

Incessant cardiac monitoring, pulse oximetry and an electrocardiogram should be monitored suspiciously. The use of intravenous magnesium sulfate has been reported as beneficial for OP toxicity, basis may rivet ACh antagonism or ventricular membrane stabilization [10-12].

Conflict of Interest

The authors assert no imminent conflicts of interest with respect to the authorship, research, and/or publication of this article.

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