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Case Report *Volume 7 Issue 1*

An Autopsy Case Report of Organophosphorus Poisoning and its Associated Respiratory Complications

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Abstract

In developing nations like India, organophosphate (OP) chemicals are among the most often utilized substances for intentional self-harm. Acetylcholinesterase, an enzyme that hydrolyses the neurotransmitter acetylcholine in the central and peripheral nervous systems (PNS), is inhibited by OP chemicals. In the case of insecticides, these anticholinesterase substances are thought to produce a delayed intermediate syndrome that need extended breathing, as well as an acute cholinergic syndrome characterized by diminished consciousness and respiratory failure.(2)Most of the time, acute respiratory failure caused by both central and peripheral mechanisms is the main cause of death. Preclinical and clinical research conducted over the past 20 years, however, has revealed a more nuanced picture of the respiratory complications following OP insecticide poisoning. These complications include the involvement of solvents in OP toxicity, the onset of delayed neuromuscular junction dysfunction during the cholinergic syndrome, and aspiration leading to pneumonia and acute respiratory distress syndrome.

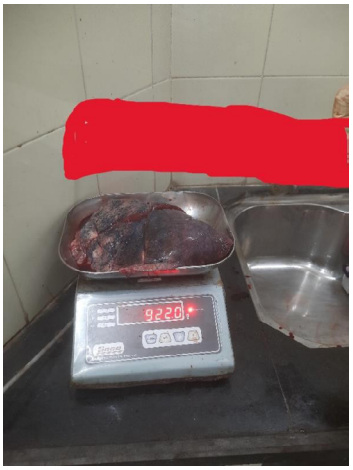
Keywords: Organophosphorus; Insecticide

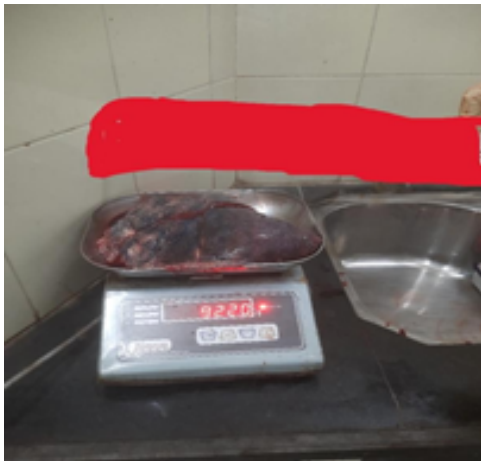
Case Report

A 65-year-old elderly male patient presented to the emergency department with a history of consuming an unknown quantity of OP with the alleged intent of suicide. After two hours of consumption, initial manifestations included vomiting and sweating, followed by difficulty in breathing and a low level of consciousness. The patient was initially taken to a local hospital where two litres of Ryle's Tube (RT) wash were given, followed by stat doses of atropine and pralidoxime. Due to a low Glasgow Coma Scale (GCS) score (E2V4M5), the patient was intubated and then referred to a tertiary centre for intensive care. Cholinesterase levels were measured at 1266.7 U/L (normal range: 4000 U/L-11000 U/L). He remained admitted at the hospital for one month and 2 days. During course of treatment, patient developed sepsis with ventilator associated pneumonia, following which he developed sudden bradycardia and hypotension followed by cardiac arrest [1,2].

Gross findings of lungs during autopsy

- Right lung weighs: 1172 gm.
- Left lung weighs: 922 gm.





RT Lung LT Lung

Both lungs were heavy, firm, boggy and reddish in appearance.
Cut section: multiple pus points mixed with blood noted.

Histopathology report of lungs shows

Replacement of alveolar spaces by proliferating fibroblasts admixed with chronic inflammatory infiltrate. Areas of alveolar hemorrhage along with hemosiderin laden macrophages also seen. Pulmonary edema fluid also noted.
Opinion: bilateral lungs- **Organising Pneumonia.**

Biochemistry and Pharmacology

Particularly acetylcholinesterase (AChE) and butyrylcholinesterase (BuChE), OP substances block these esterase enzymes [3]. At cholinergic synapses, AChE hydrolyses acetylcholine, reducing activity [4]. The effects of inhibition include excessive acetylcholine and cholinergic overstimulation in the tissues and neurological systems of the peripheral, central, and autonomic (parasympathetic and sympathetic) branches. AChE that has been inhibited may revive on its own, more quickly with the use of an oxime medication (such as pralidoxime), or irreversibly bond to the OP, a process referred to as "aging" [5].

While OP drugs inhibit several additional enzymes, such as BuChE, the therapeutic importance of this inhibition remains uncertain [6]. When compared to wild-type mice, an AChE knockout mouse exhibits the same clinical characteristics and greater sensitivity during VX poisoning [7] indicating the involvement of additional mechanisms of OP toxicity. Not all elements of OP poisoning have clinical data accessible. Thus, animal data are included in this review. Studies have revealed that the pharmacokinetics, dynamics, and responsiveness to therapy of OP vary significantly amongst species. Pig [8] and primate [8] models are currently thought to be the most clinically relevant animal models, data from these species are preferred.

Discussion

Acute cholinergic crisis, delayed neuromuscular dysfunction, and recurring cholinergic toxicity all contribute to respiratory problems in OP poisoning. Two common patterns of respiratory failure were identified in a large Sri Lankan case series of proven OP insecticide exposure:

- Respiratory failure requiring early intubation within 2 hours of exposure in unconscious patients during the acute cholinergic crisis (58% of intubated patients).
- Respiratory failure occurring later (often more than 24 h after exposure) in conscious patients without cholinergic signs (32% of intubated patients) [9].
- Patients who needed to be intubated after 24 hours needed to be ventilated for much longer.

In the largest postmortem case series yet published, there were 85 patients with OP pesticide poisoning (treated with appropriate dosages of atropine: 12–24 mg every hour, up to 1g/24h). Of the patients who died within 24 hours (n =

36), 25% had parenchymal bleeding and 75% had pulmonary interstitial edema [10]. Neuronal and nonneuronal cholinergic activation of the cilia, mucus glands, and cells that produce periciliary fluid causes bronchorrhea [11,12]. While atropine inhibits the generation of extra fluid, it has no effect on the lymphatics and interstitial space's ability to remove fluid from the alveolus [12]. Aspiration or inhalation are examples of pulmonary consequences of poisoning that can cause ARDS directly. Hematogenous exposure to OP chemicals can cause ARDS indirectly. Given that more than 50% of non-poisoning cases may go undetected with ARDS [13].

Approximately two thirds of the 49 patients who passed away within 24 hours in an autopsy case series of 85 patients suffering from OP pesticide poisoning had segmental or lobar consolidation, most likely as a result of aspiration [10]. This result is not limited to critical care units (ICUs) with limited resources; in a German ICU, 82% (27/33) of patients who had been exposed to OP pesticide had aspiration pneumonia [14].

OPs can result in vomiting, increased secretions, unconsciousness, loss of protection for the airways, and seizures. Aspiration is typical when these signs appear prior to hospital presentation. Following forced emesis or stomach lavage, patients may also aspirate [15]. When self-poisoning occurs, aspiration lengthens hospital stays, increases morbidity, case fatalities, and raises health care costs [16]. Patients receiving ventilation for NMJ dysfunction or acute cholinergic poisoning are susceptible to consequences such ventilator-associated pneumonia (VAP) [17] and ventilator-induced lung damage [18]. While the frequency of these issues in patients exposed to OP is unknown, following current VAP prevention [19-21] and best ICU practices may help these patients have lower rates of morbidity.

Conclusions

Poisoning with OP insecticides is a common suicide technique used worldwide, and OP nerve agents have the capacity to kill a lot of people. As respiratory failure and associated consequences are the main cause of death for both OP types, early resuscitation is crucial to lowering morbidity and mortality. Numerous studies have shown that early atropinisation, along with appropriate critical care and close monitoring for the development of NMJ dysfunction and recurrent cholinergic toxicity, can save thousands of lives annually.

References

1. Puneeth RM, Karthik CH (2023) Organophosphorus Poisoning Induced Extrapyramidal Intermediate Syndrome. Journal of Clinical and Diagnostic Research 17(9): 12-13.

2. Elspeth HJ, James DOJ, Simpson AJ, Sciuto AM, Eddleston M (2014) Respiratory Complications of Organophosphorus Nerve Agent and Insecticide Poisoning. Implications for Respiratory and Critical Care. American Journal of Respiratory and Critical Care Medicine 190(12): 1342-1354.

3. Lotti M (2001) Clinical toxicology of anticholinesterase agents in humans. Handb Pesticide Toxicol 2: 1043–1085.

4. Nachmansohn D, Feld EA (1947) Studies on cholinesterase; on the mechanism of diisopropyl fluorophosphateactioninvivo. JBiolChem 171: 715–724.

5. Buckley NA, Eddleston M, Li Y, Bevan M, Robertson J (2001) Oximes for acute organophosphate pesticide poisoning. Cochrane Database Syst Rev, pp: CD005085.

6. Casida JE, Quistad GB (2004) Organophosphate toxicology: safety aspects of nonacetylcholinesterase secondary targets. Chem Res Toxicol 17: 983-998.

7. Duysen EG, Li B, Xie W, Schopfer LM, Anderson RS, et al. (2001) Evidence for nonacetylcholinesterase targets of organophosphorus nerve agent: supersensitivity of acetylcholinesterase knockout mouse to VX lethality. J Pharmacol Exp Ther 299: 528-535.

8. Dorandeu F, Mikler JR, Thiermann H, Tenn C, Davidson C, et al. (2007) Swine models in the design of more effective medical countermeasures against organophosphorus poisoning. Toxicology 2007 33: 128-144.

9. Eddleston M, Mohamed F, Davies JOJ, Eyer P, Worek F, et al. (2006) Respiratory failure in acute organophosphorus pesticide self-poisoning. QJM 99: 513-522.

10. Kamat SR, Heera S, Potdar PV, Shah SV, Bhambure NM, et al. (1989) Bombay experience in intensive respiratory care over 6 years. J Postgrad Med 35: 123-134.

11. Kummer W, Lips KS, Pfeil U (2008) The epithelial cholinergic system of the airways. Histochem Cell Biol 130: 219-234.

12. Takemura Y, Helms MN, Eaton AF, Self J, Ramosevac S, et al. (2013) Cholinergic regulation of epithelial sodium channels in rat alveolar type 2 epithelial cells. Am J Physiol Lung Cell Mol Physiol 304: L428-L437.

13. Ferguson ND, Frutos-Vivar F, Esteban A, Fern´andez-Segoviano P, José Antonio A, et al. (2005) Acute respiratory distress syndrome: underrecognition by clinicians and diagnostic accuracy of three clinical definitions. Crit Care Med 33: 2228-2234.

14. Hrabetz H, Thiermann H, Felgenhauer N, Zilker T, Haller B, et al. (2013) Organophosphate poisoning in the developed world- a single centre experience from here to the millennium. Chem Biol Interact 206: 561-568.

15. Eddleston M, Haggalla S, Reginald K, Sudarshan K, Senthilkumaran M, et al. (2007) The hazards of gastric lavage for intentional self poisoning in a resource poor location. Clin Toxicol (Phila) 45: 136-143.