



## Suicidal ingestion of organophosphate compound (Sniper): A case report

Otu Etta<sup>1</sup>, Eyo E. Ekpe<sup>2</sup>, MaryMagdalene Antai<sup>1</sup>

<sup>1</sup>Department of Anaesthesia, University of Uyo Teaching Hospital, Uyo, Akwa Ibom State

<sup>2</sup>Cardiothoracic Unit, Department of surgery, University of Uyo Teaching Hospital, Uyo, Akwa Ibom State

### Abstract

**Background:** Organophosphate insecticide is one of the leading causes of poisoning in many developing Countries.

**Aim:** To describe a typical case of organophosphate poisoning and discuss its management.

**Case Presentation:** An 18 years old female Supermarket sales attendant presented with history of intentional ingestion of organophosphate(OP) compound(Sniper) to commit suicide. She was unconscious, and in severe respiratory distress. Her baseline vital signs were as follows: Pulse rate 52/m, Blood pressure-140/80mmHg, respiratory rate 32/m, and temperature 37.6°C.

She had bilateral coarse crepitations and the SpO<sub>2</sub> ranged between 40-65%. She was managed with intravenous normal saline, atropine at 1mg stat, then 2mg repeated every 5 minutes up to four times, followed by 8mg added to 500ml of normal saline in continuous infusion for 5 hours.

Also, she was intubated and manually ventilated in addition to other supportive care in ICU. She regained consciousness within 24hours and was extubated. However, she developed a sudden acute cardiorespiratory distress and died in the second day of ICU admission.

**Conclusion:** Severe organophosphate poisoning may be fatal. Early presentation to the hospital, prompt diagnosis and treatment with appropriate dose regimen of atropine, and other supportive care may improve survival.

### Introduction

Organophosphate(OP) compounds are commonly used as insecticides internationally due to their widespread availability, low cost, and reactive rapid degradation following application.<sup>1</sup> OP insecticides inhibit both cholinesterase and pseudocholinesterase activities, as they are irreversible cholinesterase inhibitors.<sup>2,3</sup>

The inhibition of cholinesterase activity leads to accumulation of acetylcholine at synapses, causing overstimulation and disruption of neurotransmission in both central and peripheral

nervous systems.<sup>3</sup>

Organophosphate insecticides is one of the leading causes of poisoning in many developing countries.<sup>4</sup> OP poisoning is commonly due to accidental or suicidal exposures through dermal, gastrointestinal, inhalational and intravenous routes<sup>2,3</sup>

One of the lethal complications following OP poisoning is the development of respiratory failure, commonly due to gastric aspiration, excessive secretions, neuromuscular involvement, acute respiratory distress syndrome, sepsis etc.<sup>5</sup> Fatal issues are often related to a delay in diagnosis, or an improper management.<sup>2</sup>

The mortality from OP poisoning in some developing countries is decreasing due to early diagnosis and improved critical care services.

In Nigeria reports on OP poisoning is scanty,<sup>6</sup> though reports of fatalities from OP poisoning in

**Corresponding Author: Dr. Otu Etta**

Department of Anaesthesia,  
University of Uyo Teaching Hospital, Uyo,  
Akwa Ibom State, Nigeria.  
E-mail: otuetta@yahoo.com

news media are common, hence, knowledge of management protocol among medical personnels in such situation may not be solid. This study describes a typical case of OP poisoning and discusses its management.

### Case Presentation

An 18years old female Supermarket sales attendant was brought to our emergency room with history of loss of consciousness, difficulty in breathing and vomiting of about one hour duration.

She was said to have ingested an organophosphate compound (sniper), in an attempt to commit suicide after she was caught stealing on Closed Circuit Television (CCTV) camera.

An initial physical examination revealed a characteristic pungent smell of sniper in her immediate vicinity. She was unconscious with a Glasgow Coma Scale(GCS) of 3/15, there was excessive oral secretion. The pupils were pinpoint, and her baseline vital signs were as follows: Pulse rate (52/m), blood pressure(140/80mmHg), respiratory rate(32/m), and temperature(37.6°C). In addition, chest auscultation revealed shallow, vesicular breath sound with bilateral coarse crepitations, and her peripheral oxygen saturation(SpO<sub>2</sub>) ranged between 40 – 65%. Other examination features were within normal limits. The baseline random blood sugar was 13.3mmol/L.

A diagnosis of respiratory failure from possible gastric aspiration following organophosphate poisoning was made. She was immediately commenced on intravenous normal saline, oxygen therapy via nasal prongs at 3L/min, atropine 1mg and urethral catheterization to monitor the urine output. However, there was no significant improvement following the initial measures as the SpO<sub>2</sub> remained consistently below 80%, hence, an endotracheal intubation and manual ventilation was performed. This was facilitated by intravenous atropine 0.6mg, sodium thiopentone 50mg, and suxamethonium 50mg. An additional 8mg of atropine at 2mg every 5minutes was given; followed by 8mg of atropine added to 500ml of normal saline to run at 100mls/hr.

The patient was admitted into the Intensive Care Unit(ICU), where a nasogastric tube was passed which drained about 500mls of offensive smelling dark coloured fluid. Manual ventilation continued

with AMBU bag as there was no functional ventilator at that moment, in addition to continuous monitoring of vital signs and other supportive care. She developed hypotension with average blood pressure of 80/40mmHg which was unresponsive to fluid resuscitation, hence, intravenous dopamine at 10mcg/kg/min was commenced.

Within 24hours of admission in the ICU, the patient's clinical status improved remarkably. The GCS was 11/15, pulse rate(138b/m), BP – 105/55mmHg(on dopamine support), respiratory rate (18/m), and SpO<sub>2</sub> (92–96%). She was subsequently extubated, and given oxygen through face mask at 4L/min.



On the second day in ICU, she suddenly started deteriorating with worsening respiratory distress. The respiratory rate increased to 52/m, blood pressure decreased to 80/40mm Hg which no longer responded to dopamine, bilateral coarse crepitation persisted with SpO<sub>2</sub> of 88%. She subsequently developed a cardiac arrest, and cardiopulmonary resuscitation was commenced. Resuscitation lasted for about 45 minutes without return of spontaneous circulation and she was certified clinically dead.

### Discussion

The use of organophosphate compound in suicide attempts is increasing.<sup>1,2</sup> In a recent review of Intensive Care management of organophosphate

insecticide poisoning in 62 patients, Coskun et al<sup>1</sup> reported that suicide attempt accounted for 73% of the patients.

Our patient was an 18 years old sales attendant in a supermarket, when she discovered that her criminal activities were detected on CCTV Camera, she simply went to the shelf and picked a bottle of sniper and drank in the toilet, where she was later found in coma.

This reveals the free access of people to a potentially poisonous substance. Probably a regulation that limits access to organophosphate compound will stem the tide of suicide following OP compound exposure.

Our patient had features of severe organophosphate poisoning. She presented in coma with GCS of 3/15, bilateral coarse crepitations, SpO<sub>2</sub> between 40-65%, pinpoint pupils, hypersecretions, sweating and unstable vital signs. The severity of OP poisoning is dependent on the type of OP compound, and the volume consumed.<sup>1,7,8</sup> She consumed a full bottle(100ml) of sniper containing 1000g/litre of 2,3-dichlorovinyl phosphate(DDVP) as active ingredient. The mortality from severe OP poisoning is high.<sup>1,2,9</sup> In a recent review of three patients with OP poisoning, Eze and colleagues<sup>9</sup> reported that the only patient who died out of the three had severe poisoning.

Our patient was managed with atropine, oxygen, endotracheal intubation and manual ventilation, in addition to other supportive care. Atropine and pralidoxime are the mainstay of treatment in OP poisoning.<sup>1,2,8</sup>

Atropine has been the core antidotal treatment in OP poisoning since 1950s.<sup>7</sup> It is a competitive cholinergic (muscarinic) receptor antagonist with good central nervous system penetration, allowing it to be effective wherever muscarinic receptors are overstimulated by acetylcholine. Atropine is administered intravenously to restore cardiorespiratory function rapidly- a process often termed "atropinization". It is used to reverse bradycardia and improve systolic blood pressure to greater than 80mmHg. At the same time to reduce bronchorrhoea, reverse bronchospasm and improve oxygenation.<sup>7</sup>

Our patient received a total of 17mg of atropine at 1mg stat, then 2mg bolus, repeated every five minutes up to four doses, followed by 8mg in 500ml

of normal saline given over 5 hours.

Till date, no dose-response studies have identified the ideal dose regimen for atropine.<sup>1,2,7</sup> In a recent study on ICU management of OP poisoning, atropine was given as a continuous infusion after a loading dose of 1mg every 5 minutes up to 3 to 4 doses, followed by continuous infusion at 0.5-2mg/hr until control of the hypersecretion symptoms occurred. Atropine was discontinued 24hrs after all signs of atropinisation (facial flushing, dilatation of pupils, dryness of mouth, tachycardia) occurred.<sup>1</sup>

A previous randomized clinical trial in Bangladesh documented that sequentially increasing(doubling) bolus doses till atropinization is achieved, followed by a continuous infusion at 20-30% of the total atropinization dose titrated to effect compared with repeat bolus regimen reduced mean time for atropinization from 152 minutes to 24 minutes, and mortality was reduced from 22.5% to 8%.<sup>10</sup>

Our patient received four boluses of atropine and continuous infusion for only 5 hours. Probably, atropinization was not sustained, hence the relapse. Pralidoxime was not used in our patient because it was not available. Pralidoxime, like other oximes reactivates acetylcholinesterase(AChE), thereby making AChE available to control acetylcholine activity.<sup>7</sup> However, there remains uncertainty about whether this efficacy translates into clinical benefit on the ward.<sup>7</sup>

Previous studies have shown lack of improvement in mortality in patients treated with pralidoxime compared with placebo.<sup>11,12</sup> This was attributed to delay in patients arriving the hospital, resulting in pralidoxime administration after irreversible ageing(formation of OP-pralidoxime bond) has occurred. Such toxicity will not respond to oximes.<sup>13</sup>

Also, administration of suboptimal doses of pralidoxime in those studies have been implicated in its inefficiency.<sup>14</sup> Currently, it is unclear which group of patients will benefit clinically from pralidoxime administration.<sup>7</sup> In the meantime, sick patients should receive oximes in a critical care environment, with full support of airway and ventilation, plus titrated administration of atropine.<sup>7</sup>

In a previous study, Acikalin and colleagues<sup>8</sup> investigated prognostic factors determining mortality in 80 patients managed for OP poisoning,

They documented that mechanical ventilation, low GCS, comorbidities, long hospital stay, elevated creatinine and low AChE levels without regeneration in the first 48 hours of admission were poor prognostic factors in OP poisoning.

Our patient, no doubt had features of severe OP poisoning. She presented in coma with a GCS of 3/15, and was promptly intubated and manually ventilated using a self inflating bag. She regained consciousness and was communicating until the second day in ICU when she suddenly developed acute cardiorespiratory distress and died. Although the immediate cause of death was not clear, it could probably be attributed to one of the acute complications of severe OP poisoning which include arrhythmias, pulmonary embolism, acute respiratory distress syndrome (ARDS) etc.<sup>8</sup>

The quality of supportive care provided during OP poisoning can significantly affect the outcome.<sup>1</sup> In a recent study, Coskun et al<sup>1</sup> observed a reduction in mortality from OP poisoning to below 15%. They attributed this reduction partly to better access to supportive therapy such as ICU care, ventilator support, respiratory support, and hemoperfusion.

In that study, all patients diagnosed of OP poisoning received gastric lavage, activated charcoal via nasogastric tube, and cleansing of the patients with soap and water in the emergency department before transfer to the ICU.<sup>1</sup>

Our patient did not benefit from top level supportive care because they were lacking at that moment. Probably, improvement in emergency department and ICU facilities, a more informed and ready health workers, in addition to increase in dosage and duration of atropine administration will improve the outcome of organophosphate poisoning in future.

### Conclusion

Severe organophosphate poisoning may be fatal. Early presentation to the hospital, prompt diagnosis and treatment with appropriate dose regimen of atropine, and other supportive care may improve survival.

### References:

1. Coskun R, Gundogan K, Sezgin GC, Topaloglu US, Hebbar G, Guven M, Sungur M. A retrospective review of Intensive Care management of organophosphate insecticide poisoning: single centre experience. *Niger J Clin Pract* 2015;18:644-50
2. Sungur M, Guven M. Intensive Care management of organophosphate insecticide poisoning. *Crit Care* 2001;5:211-215
3. Namba T, Nolte CT, Jackrel J, Grob D. Poisoning due to organophosphate insecticides. Acute and chronic manifestations *Am J Med* 1971;50:475-492.
4. Ozturk MA, Kelestimur F, Kurtoglu S, Guven K, Arslan D. Anticholinesterase poisoning in Turkey-clinical, laboratory and radiologic evaluation of 269 cases. *Hum Exp Toxicol* 1990;9:273-279.
5. Eddleston M, Buckley NA, Eyer P, Dawson AH. Management of acute organophorous pesticide poisoning. *Lancet* 2008;371:597-607.
6. Ijezie E, Megbalayin FF, Edem K. Ijezie AE. Accidental organophosphate poisoning in a child in Uyo, Nigeria: a public health alert. *Int J Sci Rep* 2016;2:106-109
7. Eddleston M, Chowdhury FR. Pharmacological treatment of organophorous insecticide poisoning: the old and the (possible) new. *Br J Clin Pharmacol* 2016;81:462-470.
8. Acikalin A, Disel NR, Matyar S, Sebe A, Kekec Z, Gokel Y, Karakoc E. Prognostic factors determining morbidity and mortality in organophosphate poisoning. *Pak J Med Sci* 2017;33:534-539
9. Eze JN, Ndu IK, Edelu BO. Teenage organophosphate insecticide poisoning: an ugly trend in Enugu, Nigeria. *Journal of Community Medicine and Primary Health Care* 2018;30:99-108
10. Abedin MJ, Sayeed AA, Basher A, Maude RJ, Hoque G, Faiz MA. Open-label of atropine bolus injection versus incremental boluses plus infusion for organophosphate poisoning in Bangladesh. *J Med Toxicol* 2012;8:108-7
11. Samuel J, Thomas K, Jeyaseelan L, Peter JV, Cherian AM. Incidence of intermediate syndrome in organophosphate poisoning. *J Assoc Physicians India* 1995;43:321-3
12. de Silva HJ, Wijewickrema R, Senanayake N. Does pralidoxime affect outcome of management in acute organophosphate poisoning? *Lancet* 1992;339:1136-8.

13. Eddleston M, Street JM, Self I, Thompson A, King T et al. A role of solvents in the toxicity of agricultural organophosphate pesticides. *Toxicity* 2012;294:94-103.
14. Johnson MK, Jacobsen D, Meredith TJ, Eyer P, Heath AJ, Ligtenstein DA, Marrs TC, Szinics L, Vale JA, Haines JA. Evaluation of antidotes for poisoning by organophosphorous pesticides. *Emerg Med* 2000;12:22-37