

Airway Obstruction due to Mucus Plug in Patients with Organophosphorus Poisoning– Atropine the Culprit? A Case Series

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Organophosphorus poisoning is a common cause of death in developing countries. These compounds inhibit acetylcholinesterase leading to accumulation of acetylcholine, causing overstimulation of nicotinic and muscarinic receptors. Atropine and oximes are the mainstay of treatment. Atropine helps in countering muscarinic effects and decreasing secretions. It also makes the secretions thick, tenacious and may obstruct the airway. We observed five cases of organophosphorus poisoning being treated with atropine developing sudden respiratory distress attributed to mucus plug obstructing the airway which was confused with intermediate syndrome. After removal of the mucus plug these patients recovered uneventfully.

Keywords: atropine, organophosphorus poisoning, respiratory distress, mucus plug, airway obstruction.

Introduction

Primary mechanism of organophosphorus (OP) toxicity is inhibition of acetylcholinesterase in central and peripheral nervous systems, causing accumulation of acetylcholine at nicotinic and muscarinic receptors.¹ Action on muscarinic receptors causes increased salivation, bradycardia, lacrimation, emesis, and miosis.² Atropine counters muscarinic effects and decreases secretions.³ However, it makes secretions thick and viscous, which are difficult to swallow in patients with muscle weakness and also difficult to remove by suction.

We report five cases of OP poisoning, where we strongly suspect that use of atropine could be the culprit, leading to mucus plug formation and airway obstruction.

Case Report

Out of 150 cases of suicidal OP poisoning admitted to a tertiary care hospital and managed for various levels of toxicity and complications,

including intermediate syndrome over a five years period, we report five cases which developed respiratory distress. (Table 1)

OP poisoning was diagnosed based on positive history of ingestion and characteristic smell. The patients presented between 30-60 minutes of ingestion (mean 45 ± 15 min) with nausea, vomiting and sweating. Supportive measures like stomach wash, body wash, humidified oxygen and intravenous fluids were initiated.

Blood sample was drawn for routine investigations and pseudocholinesterase (PChE) estimation. Haemodynamics (mean \pm SD) on admission were heart rate (HR) 68.2 ± 3.03 beats per minute (bpm), SpO₂ $94.6 \pm 2.4\%$, respiratory rate (RR) 17.4 ± 1.94 /min and mean arterial pressure 86 ± 9.22 mmHg.

Initial treatment consisted of loading dose of Inj. Pralidoxime 1g and Inj. Atropine 1-3mg, intravenously. Monitoring of patients was continued. Dose of atropine was doubled as per institutional protocol (every 3 to 5 min) till target heart rate between 110-120 bpm was reached. After this atropine infusion was started at 10-20% of the bolus given.

Patients were assessed every 15 min for features of atropinization (heart rate > 110 bpm, clear chest). Intravenous fluids were continued based on haemodynamics and urine output was monitored. Regular suctioning of oral cavity was done. Patients were also monitored for

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development of intermediate syndrome and atropine toxicity.

During 3rd to 5th days (mean 4.2 days) of ICU stay, these patients developed sudden difficulty in breathing, tachypnoea (RR 32.4±2.07/min),

tachycardia (HR 121.4±3.84/bpm), use of accessory muscles of breathing and oxygen desaturation (79.8±1.83%). Suspecting intermediate syndrome, intubation was planned. During laryngoscopy, mucus plug blocking the airway was observed over the vocal cords.

Table 1
Details of Patients, Poisoning and Management

Parameters	Case 1	Case 2	Case 3	Case 4	Case 5
Age (Years)	20	27	37	50	63
Sex	Male	Male	Female	Male	Male
History of Smoking	No	Yes	No	Yes	No
Compound consumed	Methyl parathion	Ethyl parathion	Ethyl parathion	Methyl parathion	Methyl parathion
Ingested Quantity (ml) (approximately)	50	75	50	100	75
PChE levels on Admission At Intubation (Normal range- 5,000-15,000IU/L)	1410 1375	1670 1540	2080 1950	1370 1290	1250 1200
Total dose of Atropine (mg)	430	450	256	580	514
Presentation of Respiratory Distress (Hours after admission)	96	114	74	82	112
Duration of ICU stay (days)	9	11	8	13	15

Figure 1(a)
Mucus Plug covering the vocal cords at the time of intubation



Figure 1(b)
Visible vocal cords after removal of mucus plug

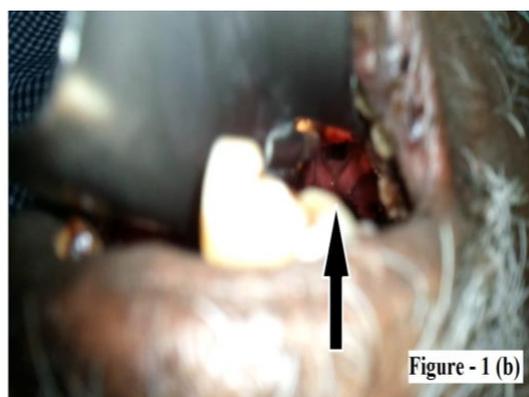


Figure 2
Mucus plug removed during Intubation



The thick mucus plug could not be sucked out and was removed by Magill's forceps. Patients were intubated and mechanically ventilated. Blood samples were collected for PChE estimation. Atropine infusion was continued, but rate was decreased by 20% of the ongoing rate. All five patients improved over 4 to 6 hours after the incident with no further adverse respiratory episodes. The patients were extubated 24 hours later, after confirmation of consciousness, adequacy of muscle power and haemodynamic stability. Chest x-rays were taken before extubation. Atropine was subsequently tapered and stopped.

Discussion

Atropine blocks production of respiratory secretions in response to cholinergic stimulation but has no effect on the baseline secretions. It has also been clearly demonstrated to depress ciliary beat frequency, to slow airway mucociliary clearance which can reduce large airway mucus transport and make the secretions thick and tenacious.^{4,5}

The mean dose of atropinization in these patients was 11.6 ± 3.28 mg and mean dose infused hourly was 3.31 ± 0.78 mg. The mean total dose of atropine received was 446 ± 121.4 mg, with mean duration of 139.2 ± 49.18 hours. Studies show that the dose of atropine cannot be fixed and is variable with a wide range between 150mg to 1100mg during management of OP poisoning.⁶ Mean atropine dose in our patients was within this range. Based on clinical suspicion, the dose of atropine was decreased by 20% after intubation as the thick mucus plug causing airway obstruction and necessitating intubation was attributed to atropine effect with no other

classical signs of overdose such as confusion, pyrexia, absent bowel sounds.²

The sudden respiratory distress observed was initially suspected to be due to intermediate syndrome which is common between 1 to 4 days after OP compound consumption. But recovery of patients after removal of the mucus plug within 24 hours indicates the absence of intermediate syndrome in which muscle weakness generally persists for an average of 4 – 18 days.⁷ Mask ventilation with high flow oxygen, non-invasive ventilation or insertion of a supraglottic airway device to maintain the airway after removal of mucus plug may have been considered to avoid intubation and ventilation. However, due to acuteness of the condition with rapid desaturation, and as intermediate syndrome was suspected, endotracheal intubation was performed and mechanical ventilation commenced. The mean PChE level on admission was 1556 ± 330.57 IU/L and at the time of respiratory distress, 1471 ± 295.64 IU/L, which confirms that the deterioration was not related to OP poisoning.⁸

Endotracheal tube blockage in mechanically ventilated patients is a well-known risk, especially so in those patients on atropine because of drying up of bronchial secretions. Lung collapse due to mucus plug formation in three cases of OP poisoning being treated with Inj. glycopyrrolate has also been described.⁹

In smokers muco-ciliary activity is inhibited and viscoelastic properties of mucus change.¹⁰ The two patients with history of smoking did not have any history of bronchitis or COPD and were not on any anticholinergic medication. We did not find any difference in the time of respiratory distress in these patients and hence could not hypothetically relate the incident to smoking.

The chest X-ray taken prior to extubation was normal indicating there was no underlying lung pathology for the sudden occurrence of respiratory distress. After extubation, patients continued to receive atropine at lower doses. We did not observe any recurrence of mucus plug formation causing respiratory distress post extubation.

Conclusion

Atropine used in the management of OP poisoning may be a causative factor in airway obstruction, with potential for producing

respiratory distress. Observation and anticipation of this rare effect in non-intubated atropinised patients is important as it can be easily confused with intermediate syndrome.

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