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## CASE REPORTS

### KETAMINE AS A BRONCHODILATOR AND ANTIEPILEPTIC IN ORGANOPHOSPHORUS POISONING

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#### **Abstract**

Organophosphorus poisoning by inhalational route in a young infant is rarely reported. A three month old female child with organophosphorus poisoning presented with respiratory failure and convulsions. In addition to atropine and pralidoxime, ketamine was added to management owing to its bronchodilator and antiepileptic properties. The child improved following initiation of ketamine infusion.

**Keywords:** organophosphorus, ketamine, bronchodilator, antiepileptic.

#### **Introduction**

Organophosphate compounds are commonly used as insecticides in household and agriculture. These compounds are well absorbed from all the routes. Inhalation of vapor may cause nasal irritation, bronchoconstriction, wheezing and increased bronchial or salivary secretions. Organophosphate compounds act by phosphorylating the active or esteratic site of acetylcholine esterase leading to an irreversible inhibition of the cholinesterase resulting in excessive accumulation of acetylcholine at receptor sites. Signs and symptoms are due to excess acetylcholine. (1) Organophosphorus poisoning by inhalational route in a young infant has been rarely reported. We report a case of 3 month old girl with history of exposure to organophosphate insecticide with respiratory failure

and convulsions. Plasma cholinesterase was very low consistent with organophosphate poisoning. Ketamine was added as adjunctive therapy to conventional bronchodilators and mechanical ventilation. To the best of our knowledge use of ketamine as a bronchodilator and antiepileptic in organophosphorus poisoning has not been reported previously.

#### **Case Report**

A 3 month old female child was brought to our emergency department with respiratory failure. On thorough history we came to know that her elder sister had placed a cap of organophosphorus bottle near the nose of patient. Patient was immediately intubated and put on mechanical ventilation. She had rectal temperature of 36.80C, heart rate 117/minute, blood pressure 96/58 mm of Hg, oxygen saturation was 78%. Chest examination revealed crepitations and rhonchi. Arterial blood gas showed a pH of 7.4, PaO<sub>2</sub> of 78 mm Hg, PaCO<sub>2</sub> 50 mm Hg and a normal bicarbonate. Chest X-ray showed diffuse bilateral infiltrates. She was treated with fluid infusions, atropine (0.05mg/kg every 15 minute) and pralidoxime (25mg/kg every 6 hr). Nebulization with levosalbutamol, corticosteroids and terbutaline infusion were started to relieve bronchospasm. Patient developed convulsions for which intravenous midazolam was given. In view of persistent bronchospasm despite of mechanical ventilation and bronchodilators and ongoing seizures, ketamine was

initiated. Ketamine was given in a bolus dose of 0.5 mg/kg followed by continuous infusion of 1mg/kg/hour. Within 24 hours of starting ketamine infusion, ventilatory requirements decreased. The frequency of atropine administration was reduced and finally stopped after 24 hours when bradycardia, hypersecretion and bronchospasm disappeared. Extubation was done after 24 hours of ketamine infusion. There were no further convulsions. All laboratory values were normal, except for a decreased phosphocholine esterase (PCE) [0.2Ku/l (reference range:4.6-10.4Ku/l)] Repeat PCE was 4.6 Ku/l on 6th day. The patient was discharged without sequelae.

### Discussion

Organophosphorus insecticides are commonly used in agriculture. They are rapidly absorbed by all routes but in our patient the absorption was by inhalation which is very rare. (2) Treatment is aimed at reversal of muscarinic signs with atropine and enzymes activation by pralidoximes. Frequent atropine dose or continuous titrated infusion are used to achieve drying of secretion and resolution of bradycardia. The role of ketamine as a bronchodilator and antiepileptic is controversial and very few studies advocating these properties exist. (3) The unidentified role of ketamine as a bronchodilator was first brought to light by Betts and Parkin in 1971.(4) In many experimental studies, ketamine has been found to alter respiratory mechanics and produce airway relaxation by acting on various receptors and inflammatory cascades which mediate bronchospasm. (5) Ketamine increases synaptic catecholamine levels by blocking the re-uptake of norepinephrine into presynaptic sympathetic neurons. These endogenous catecholamines act on  $\beta_2$  receptors and lead to bronchodilation. (6) Ketamine exerts an anti-cholinergic effect on bronchial smooth muscles by inhibiting vagal outflow. (7) It decreases calcium influx in smooth muscles by inhibiting L-type calcium channels, and resultant decrease in intracellular calcium relaxes airway smooth muscle. (8) Ketamine has been empirically used as a bronchodilator in severe status asthmaticus refractory to routine medications and has been found to obviate the need for mechanical ventilation in various studies. (9) Ketamine also has antiepileptic effect. The accumulation of acetylcholine and amino acid neurotransmission is responsible for the nerve agent-induced status epilepticus and brain damage in organophosphorus poisoning. Ketamine induced NMDA receptor channel non-competitive blocking explains its neuroprotective and anticonvulsant properties. (10,11)

### Conclusion

Although most patients of organophosphorus poisoning respond to conventional treatment few patients may pose a challenge. The use of ketamine may relieve bronchospasm and alleviate the need for mechanical ventilation. Thus in our patient ketamine served dual purpose of bronchodilation and seizure control.

### Authors Contribution

Both the authors were responsible for conceptual designing, acquisition of data and critical framing of the manuscript. The final version of submitted manuscript was approved by all the authors. SG will act as guarantor.

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**Conflict of Interest :** None

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