

## Case Reports

### Severe Organophosphate Poisoning in a Neonate

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Organophosphates (OP) are widely used as insecticides. Accidental OP poisoning is reported world over. These compounds inhibit the enzyme acetyl-cholinesterase within the peripheral and central nervous system by phosphorylation of its esteratic site. The resultant accumulation of acetylcholine at the cholinergic receptor sites, leads to signs of cholinergic toxicity. Accidental OP sites, leads to signs of cholinergic toxicity. Accidental OP poisoning has rarely been reported in neonates. The only case report that we could find was that of a one month old infant who presented with apnea and profuse secretions(1). We present a 25-day-old infant with OP toxicity.

#### Case Report

A 25-day-old girl was referred with a diagnosis of bronchopneumonia. The baby had poor feeding, lethargy, jerky respiration and excessive secretions for preceding 24 hr. She was breastfed and had no history of trauma, exposure to chemicals, or any previous illness. On examination she weighed 2.5 kg, had profuse clear secretions from the trachea and oropharynx and

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severe respiratory distress characterized by chest wall retractions. Respiratory rate was 30/min and irregular and the heart rate was 60/min. She had pin-point non-reactive pupils, generalized hypotonia, absent deep tendon reflexes, and occasional twitching movements of the limbs. Abdomen was soft, and bowel sounds were normal.

Emergency airway management comprised immediate oropharyngeal suction, endotracheal intubation and assisted ventilation. In view of signs of cholinergic over-activity, atropine was administered in doses of 0.05 mg/kg intravenously and repeated within 15 min. It was followed by an increase in heart rate to 120/min and pupillary size to 3 mm. A presumptive diagnosis of organophosphate poisoning was made. However, repeated direct questioning of the parents did not reveal history of accidental ingestion or exposure to any such compound. Gastric lavage and skin decontamination was undertaken, a dose of pralidoxime (PAM) (25 mg/kg) given and the baby shifted to the Intensive Care Unit.

Gastric lavage revealed a clear fluid. Laboratory investigations showed polymorphonuclear leucocytosis (absolute PMN count 7600/cu mm) and normal blood levels of electrolytes, urea and creatinine. Arterial blood gases showed pH of 7.36, pO<sub>2</sub> 75 mm Hg, pCO<sub>2</sub> 22 mm Hg and HCO<sub>3</sub> 16.5 mEq/l. The coagulation parameters including prothrombin index and partial thromboplastin time were normal. The level of blood glucose was 92 mg/dl. Chest X-ray and cranial ultrasound did not show any abnormality and CSF examination was normal. The serum acetyl-cholinesterase level was reduced to 2.14 nmoles product formed/min/mg protein (lab control-13.41 nmoles product formed/ min/mg protein).

Atropine infusion was started at a rate of 0.01 mg/kg/h and increased to 0.02 mg/

kg/h for complete atropinization as assessed from bronchial secretions, heart rate and pupil size. The infusion was continued for 3 days after which it was tapered off over a 24-hr period. Over next 3 days atropine was administered 8-12 hourly; an additional dose was given whenever the heart rate dropped below 80 beats/minute or when the tracheo-bronchial secretions increased. After 7 days the heart rate stabilized between 120-130/min.

In view of irregular shallow respiration at admission, the patients were put on mechanical ventilation. As the respiratory efforts improved and secretions decreased the FiO<sub>2</sub> and ventilator rates were gradually decreased, put on synchronised intermittent mandatory ventilation on day 6 and weaned off on day 8 of hospitalization. She again required assisted ventilation for another four days, from day 10 to 14, because of nosocomial pneumonia. She was discharged after 20 days of hospital stay with no obvious neurologic or pulmonary sequelae. On follow up visit at 4 months of age the baby was thriving well and had attained developmental milestones appropriate for the age.

### Discussion

The diagnosis of OP poisoning is based chiefly on a history of exposure and the characteristic signs of cholinergic overdose. However, a history of exposure may not be evident sometimes(1), as happened in our case. A lack of history of exposure in the presence of cholinergic toxicity warrants a therapeutic trial with atropine. In contrast to adults, CNS depression manifesting as coma, stupor, hypotonia and muscle weakness is seen in 90% children(2). Respiratory distress, miosis and excessive salivation also occur frequently(1,2,3). The characteristic SLUD complex (salivation, lacrimation, urination and diarrhea) is an unreliable indicator of OP poisoning in infants

and children. Bradycardia, which was a prominent sign in our patient is found only in 15-20% children(2). Fasciculation's noted in our patient are uncommon in children; seizures occur in one-fourth of all patients(2).

Difficulty in diagnosis of OP poisoning in children is frequently seen. Zweiner and Ginsburg(1) observed that of 20 referred patients, only in 4 was the diagnosis correct prior to transfer. The differential diagnosis includes head trauma(1), bronchopneumonia(1), opiate overdose(4), diabetic keto acidosis(5) and carbamate poisoning(6).

The rapidity of onset of symptoms of OP poisoning depends on the dose, route of exposure and the potency of the compound. It may vary from minutes in case of massive ingestion or inhalation to several days if the agent is highly fat soluble(7). On an average most patients become symptomatic within 24 h of exposure.

The diagnostic hallmark of OP poisoning is reduction in serum and RBC cholinesterase activity. Although RBC acetyl-cholinesterase activity is considered more sensitive and specific for OP poisoning, a recent study noted no disparity between the RBC and serum cholinesterase activity measured on admission in 20 out of 24 patients(1). In clinical practice, therefore both serum and RBC cholinesterase levels may be used for confirming the diagnosis of OP poisoning. A 50% or greater decrease in activity below normal laboratory values is consistent with the diagnosis of organophosphate poisoning. Our patient had serum cholinesterase activity less than 20% of the control value.

Therapy is aimed at supporting ventilation as respiratory failure is the usual cause of death. Decontamination is essential to prevent further absorption from the skin as well as to prevent

contamination of medical personnel. Atropine antagonises the central and the muscarinic effects, but has little effect on nicotinic receptors. The recommended dose is 0.05 mg/kg IV followed by 0.02 to 0.05 mg/kg every 5-10 minutes till complete atropinisation is achieved. A continuous infusion of atropine at 0.02 to 0.08 mg/kg/h has also been used to maintain a steady atropinised state(8). The response to atropine is judged by the improvement in the signs of cholinergic toxicity. Signs of improvement after 12-24 hr are indications to begin a gradual tapering of the drug. However, depending on the fat solubility of the organophosphate involved, atropine may be required for days to weeks(8). Our patient needed atropine for one week because of recurrent bradycardia.

PAM, a cholinesterase reactivator which hastens the restoration of the enzyme activity at neuromuscular junction helps in reversing respiratory muscle paralysis and muscle fasciculation. It should be administered as an intravenous infusion over 20 min in a dose of 25-50 mg/kg within 24-48 h of exposure(3). The dose may be repeated after 1-2 h and then at 10-12 h intervals if cholinergic signs recur(9). We did not use PAM after the first dose. Recently, a quaternary ammonium compound glycopyrrolate has been used as an antidote to OP poisoning. It is as effective as atropine, and causes less tachycardia, and fewer CNS effects(10).

After recovery the patient should be observed for 24-48 h to ensure that symptoms do not recur as the effects of the antidote wear off. If relapse occurs incomplete skin and stomach decontamination should be considered. Pulmonary complications occur frequently(1). Pleomorphic ventricular tachycardia(10), pulmonary edema and a demyelinating polyneuropathy(11) are some of the reported late effects of OP poisoning.

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