Organophosphate Poisoning in a Neonate

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ABSTRACT

Organophosphates (OP) are used worldwide as insecticides and are a common cause of poisoning, particularly in developing countries. OP poisoning is potentially fatal if not recognised and treated early. It manifests as cholinergic toxicity. Reportedly, accidental poisoning is rare in neonates, which can be attributed to the difficulty in diagnosing it in children owing to their variable presentations and unavailable exposure history. We present a case of a 27-day baby who presented with accidental OP poisoning. After management in the hospital, the baby was discharged home and is under close follow-up. The outcome depends upon early detection of poisoning and timely management with antidote.

Key Words: *Neonate, Poisoning, Organophosphate*.

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INTRODUCTION

Organophosphates (OPs) are commonly used insecticides and cause acetylcholinesterase inhibition in the blood and nervous system. OP poisoning is a common emergency in hospitals in developing countries, especially in the Southeast Asia. In OP poisoning, acetylcholine is not broken down in the body without phosphodiesterase, resulting in accumulation, leading to cholinergic toxicity.[1] The symptoms include diaphoresis, diarrhoea, urination, miosis, bradycardia, bronchospasm, excessive secretion, emesis, lethargy, lacrimation, and salivation. The mainstay of treatment is initial resuscitation followed by atropine and pralidoxime.[2] Even after the management of acute poisoning, long-term sequelae including neuropathy can occur weeks after ingestion. Diazepam has been suggested to prevent neurocognitive dysfunction.[3] Close follow-ups are recommended in these patients.

CASE REPORT

A 27-day healthy baby boy, born at full-term *via* caesarean section, had an accidental unintentional ingestion of OP poison (2 drops) given by the mother due to the similarity of an insecticide bottle with an infantile colic drops bottle (Figure 1).

On August 1, 2024, the infant presented to the Neonatal Intensive Care Unit (NICU) with complaints of lethargy, hyperthermia, excessive oropharyngeal secretions, and jerky breathing for one hour.

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On examination, he weighed 3.7 kg and had severe respiratory distress characterised by bronchospasm and subcostal retraction, with a respiratory rate of 29 breaths/min (irregular), heart rate of 68 beats/min, and temperature of 102˚F. He also had pinpoint non-reactive pupils (Figure 2), hypotonia, and jerking movements of the limbs. However, the abdomen was soft and non-tender, and bowel sounds were normal.

Emergency airway management comprised immediate oropharyngeal suctioning, endotracheal intubation, and mechanical ventilation. Gastric lavage was done which revealed clear fluid. Due to signs of cholinergic over-activity, atropine was administered intravenously at a dose of 0.05 mg/kg, repeated every 10 minutes till complete atropinisation occurred. Diagnosis was made based on clinical signs and symptoms, as acetylcholinesterase levels were unavailable. Pralidoxime was given at a dose of 25 mg/kg, infused over one hour, and repeated every 12 hours. Atropinisation was completed after three days and pupils became dilated (Figure 3).

The baby was kept in the NICU for seven days, and laboratory investigations showed white cell count of 5400/µL which increased to 7400/µL in subsequent days. C-reactive protein was 0.8 mg/dL. Arterial blood gases showed pH of 7.3, pCO_2 of 49.2 mmHg, and HCO_3 of 24.7 mEq/L. Renal function tests, serum electrolytes, and serum calcium levels were within the normal range. After two days, the feed was built up and the baby was weaned-off from the mechanical ventilation support. Intravenous magnesium sulphate was administered at a dose of 30 mg/kg for bronchospasm. The baby was kept on midazolam infusion (0.1 µg/kg/min) for sedation for three days and then gradually tapered off. Intravenous antibiotics, nebulisation with salbutamol, and intravenous hydrocortisone were also prescribed for pneumonitis and bronchospasm. On the 4th day of admission, the baby was shifted to warmer care. Oxygen

was off on the 5th day of admission, and the patient was discharged on the 7th day of admission with advice to follow-up after five days.

Figure 1: The bottle on the left was mistaken for colic drops on the right.

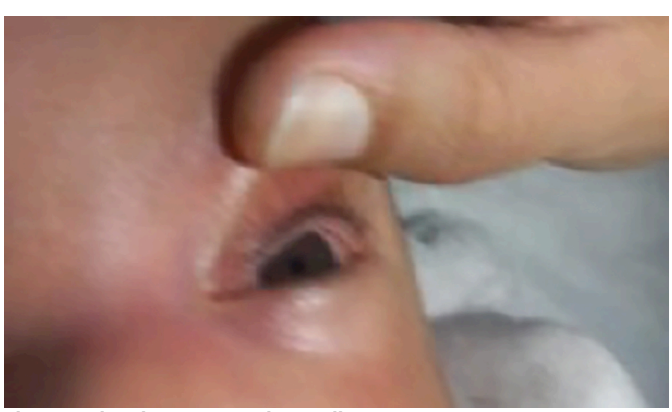

Figure 2: Pinpoint, non-reactive pupils.

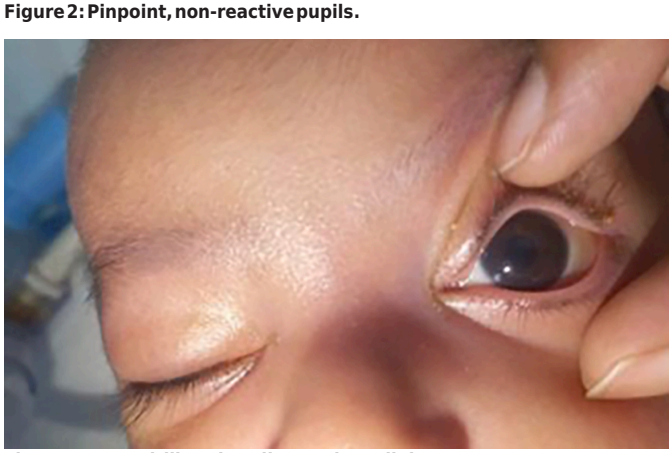

Figure 3: Normal dilated pupils reactive to light.

DISCUSSION

The OP poisoning is rare in neonates. Most cases reported in neonates are transplacentally acquired and accidental or homicidal ingestion.[4] Although OP poisoning is a global health hazard, documented case reports of such patients are mainly from the Southeast Asian region.[5] Differential diagnoses included sepsis, bronchiolitis, bronchopneumonia, and head trauma.[6] OP poisoning presents with respiratory difficulties and can be misdiagnosed as pneumonia.[7] OPs manifest their toxicity by affecting muscarinic and nicotinic receptors.[2] The mechanism of toxicity is acetylcholinesterase inhibition causing accumulation of acetylcholine neurotransmitter. Acetylcholine accumulation causes continuous stimulation of acetylcholine receptors in the nervous system.[8] Muscarinic signs are manifested as diaphoresis, diarrhoea, urination, miosis, bronchospasm, bradycardia, emesis, lacrimation, and salivation. The nicotinic signs include muscle fasciculation and respiratory paralysis. Central nervous system involvement results in drowsiness, convulsions, and coma.[9] Neuropathy is a complication of OP poisoning caused by sensory and motor axonal degeneration of the peripheral nerves and spinal cord.[2] Treatment starts with resuscitation followed by atropine and pralidoxime administration. The determinants of outcome are the time duration from ingestion to initial presentation, rapid stabilisation, and definitive treatment in the form of antidote administration. The foremost aim of therapy is to support ventilation to prevent death by respiratory failure.[10] Atropine is the main treatment for OP poisoning. The recommended atropine dosage is 0.02–0.05 mg/kg repeated every 5–10 minutes until atropinisation is achieved. Atropinisation is characterised by pupillary dilation, drying of bronchial and mucus membrane secretions, and tachycardia.[8] Pralidoxime is a cholinesterase reactivator that accelerates the restoration of enzyme activity at the neuromuscular junction, causing the reversal of respiratory muscle paralysis. Pralidoxime is administered at a dose of 25-50 mg/kg in the form of infusion.[10] Patients who recover from acute toxicity may suffer from neurological sequelae. Diazepam is used for the prevention of neurocognitive dysfunction and treatment of seizures by reactivation of cholines-terase.[8] Recent studies suggest glycopyrrolate as an alternative antidote to OP, as it is equally effective as atropine with fewer side effects.[11]

As OP poisoning can present with variable signs and symptoms, healthcare professionals need high levels of vigilance to diagnose this poisoning. In highly suspected cases, direct leading questions regarding exposure may be required. Another important issue highlighted in this case is the importance of safety education for families, as this case presented the administration of OP by confusing it with medicine. Parents should always be educated to double-check before giving medicine to children and to keep containers with similar appearances separately.

PATIENT'S CONSENT:
Informed consent was obtained from the parents of the patient.

COMPETING INTEREST:
The authors declared no conflict of interest.

AUTHORS' CONTRIBUTION:
AK: Conception, design of the work, drafting, and revising it critically for important intellectual content.
AT: Substantial contributions to the design of the work.
YZ: Acquisition, interpretation, and drafting of the work.
AE: Design of the work, acquisition, and drafting of the work.
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All authors approved the final version of the manuscript to be published.

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