CASE REPORT

ORGANOPHOSPHORUS POISONING IN A NEONATE

Chheda Akash H, Hiremath Ashwini, Khadse Sandhya, Kulkarni Rajesh, Valvi Chhaya, Anandi Shobi

Abstract

Organophosphate (OP) poisoning is a potentially fatal but completely treatable condition that is still very prevalent in our country. Early recognition and treatment is of paramount importance in preventing fatality. Neonatal organophosphate poisoning is very rare. Only three cases have been reported so far in neonates; two accidental and one transplacental poisoning. We report the 4th case of neonatal OP poisoning and the 1st of its type, a case of homicidal OP poisoning in a 15 days old girl baby, presenting with respiratory distress, nystagmus and excessive secretions.

Keywords: Organophosphate, neonatal, homicidal, pupillary.

Introduction

Neonatal sepsis is a very common condition in neonatal intensive care unit (NICU). (1) Late onset sepsis manifests as pneumonia or meningitis. Pneumonia typically presents with tachypnea, chest retraction, grunting, cyanosis, apneic spells, decreased activity and poor feeding. (2) Organophosphate (OP) poisoning being very rare in neonates can also present with respiratory difficulties and can be misdiagnosed as pneumonia. (3) The following case report shows that OP poisoning though very rare should be suspected in neonates when cholinergic signs (excessive secretions and miosis) are present along with respiratory distress.

Case Report

A 15 days old healthy breast fed girl baby, born of full term normal delivery came with complaints of excessive secretions from mouth, roving eye movements and poor feeding for the past three hours. Baby was referred with a differential diagnosis of bronchopneumonia or late onset sepsis. There was no history of fever, convulsions or trauma. On initial examination, heart rate was 126/min, respiratory rate was 34/min and was irregular, capillary refill time was < 3secs and peripheral pulses were well felt. There were excessive secretions from mouth, irregular jerky respiration and nystagmus. However cry, activity and tone were good. Oxygen was given by continuous positive airway pressure, patient was kept nil by mouth on maintenance intravenous fluids and intravenous antibiotics (ampicillin and gentamicin) were started. Arterial blood gas on admission revealed pH of 7.43, paO2 of 108 mm Hg, oxygen saturation of 97%. Complete blood count revealed polymorphonuclear leucocytosis (total leucocyte count was 17200/mm^3 with absolute neutrophil count of 10,460/cumm), hemoglobin was 13.2 gm% and platelet count was 332000/cumm. Serum creatinine was 0.4 mg/dl, blood urea was 12 mg/dl, bilirubin was 0.4 mg/dl and liver enzyme levels, ionic calcium were within normal limits. CRP was negative, blood culture collected on admission showed no growth and the first chest X-ray was normal. Within three hours of admission, patient deteriorated and developed shallow irregular respiration requiring more oxygen. Heart rate decreased to 100/min, activity was depressed and hypotonia appeared. At this point pupillary examination revealed pin point pupils not reactive to light. Patient was intubated and ventilated after 4 hours of admission in view of shallow respiration and poor sensorium. In view of cholinergic signs, OP poisoning was suspected and on retrospective questioning, history of homicidal ingestion of thymit (10% phorate- restricted use pesticide) granules was elicited. In our case the toxic substance was fed three hours before reaching the hospital. Atropine was given as 0.05 mg/kg bolus over 10 minutes and repeated every 10 minutes till complete atropinisation occurred and then an infusion of 0.02 mg/kg/hour was given for first 36 hours. Levels of serum acetyl cholinesterase levels were 466 units (normal: 2710- 11510 units), confirming the diagnosis of OP poisoning. Pralidoxime was given at 25mg/kg over 1 hour, repeated every 12 hours for 5 times. Repeat cholinesterase levels on second day showed minimal improvement (566 units). Patient showed initial clinical improvement with the disappearance of cholinergic signs and heart rate of more than 130 beats/min after 48 hours, however patient started having hypotension requiring inotrope support and increased oxygen requirement and developed pneumonia resulting in multi-organ failure. Patient succumbed on the fourth day of hospitalization. Gastric lavage analysis was inconclusive. Medico legal postmortem confirmed the intoxication by organophosphorus compound.

Discussion

Organophosphorus poisoning in neonates is rare and very few cases have been reported so far. Most of the cases reported are of babies born to mothers who had OP poisoning by insecticidal ingestion, either suicidal or homicidal, just before delivery i.e. transplacentally acquired. Other modes of poisoning can be either by inhalation, or ingestion, either accidental or homicidal. (4-6)

Organophosphorus compounds inhibit acetyl cholinesterase in the central and peripheral nervous system causing an increase in acetylcholine with the resultant muscarinic, nicotinic and central signs. (7) Muscarinic signs go by the acronym DUMBELS (diaphoresis, diarrhea, urination, miosis, bronchorrhea, bronchospasm, bradycardia, excessive lacrimation and salivation). Nicotinic signs include muscle fasciculation, weakness and respiratory paralysis. Central signs include central nervous system (CNS) depression / convulsion and coma. (8) In our case CNS depression was seen with poor sensorium and hypotonia.

OP compounds cause 3 types of paralysis (9): a) Type I- acute paralysis due to continuous depolarization of neuromuscular junction, b) Type II- Intermediate syndrome which develops 24-96 hours after initial

improvement, presents as respiratory distress and weakness of proximal muscles, neck and trunk. Neuromuscular transmission defect or toxin induced muscle instability may be responsible for type II paralysis; however incomplete treatment may be the cause. c) Type III-Delayed polyneuropathy after 2-3 weeks of op exposure involves distal limbs and spares proximal muscles, trunk, neck and cranial nerves. Our case initially showed type I paralysis but later also showed type II paralysis.

Therapy is aimed at supporting ventilation as respiratory failure is the usual cause of death. Decontamination is important to prevent further absorption from the skin and also to prevent contamination of medical personnel. Atropine antagonizes the central and the muscarinic effects of acetylcholine, but has little effect against its nicotinic action. (10) Pralidoxime is a cholinesterase reactivator which hastens the restoration of the enzyme activity at the neuromuscular junction and helps to reverse respiratory muscle paralysis and muscle fasciculation. It should be administered as an intravenous infusion over 20 min in a dose of 25-50mg/kg within 24-48 h of exposure. (11) The dose may be repeated after 1-2 h and then at 10-12 h intervals if cholinergic signs recur. (12) 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. (13)

Conclusion

High degree of alertness is required to diagnose organophosphate poisoning in neonates. In neonates all cases of respiratory distress should not be labeled as bronchopneumonia or sepsis especially when symptoms such as excessive secretions, nystagmus, and CNS depression are present. Pupillary examination can clinch the diagnosis of OP poisoning especially when associated cholinergic signs are present and therefore is important and should be routinely performed in neonatal examination.

Conflict of Interest : None

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From: Department of Pediatrics, B.J.Medical College and Sassoon General Hospital, Pune, India.

Address for Correspondence: Dr Akash Chheda, A-203, Chitrakut Building, S.G.Barvenagar, Bhattwadi, Ghatkopar (west), Mumbai- 400084.

Email : akashchheda12@gmail.com

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