DOI: 10.7860/IJNMR/2022/58467.2358

Case Report

Paediatrics Section

Organophosphorus Poisoning with Homicidal Intention in a Neonate

LAXMAN BASANY[1], ROJA AEPALA[2]

ABSTRACT

Organophosphorus Compounds (OPC) are widely used as pesticides, and poisoning due to OPC is very rare in neonates. A 12-day-old female neonate was admitted with gasping respiration, excessive oral secretions and cold extremities. She had frothing from mouth and nose with an offensive odour, was hypothermic, hypotonic, cyanosed and was in mild stupor. Pupils were pin pointed, capillary refill time was 4-5 sec and had bilateral crepitations of lungs on auscultation. The baby was intubated, given 0.1 mg of atropine followed by 0.9% saline bolus and gastric lavage. She was treated with empirical antibiotics, atropine, Pralidoxime (PAM) and respiratory support. The baby responded well to treatment and was discharged on 10th day of admission. This could be the second case of Organophosphorus (OP) poisoning with homicidal intention reported in neonates.

Keywords: Atropine, Newborn, Organophosphate, Pralidoxime

CASE REPORT

A 12-day-old female neonate was referred to the Neonatal Intensive Care Unit (NICU), with a history of OP poisoning. The baby was born to a 23-year-old primigravida mother, who had regular antenatal check-ups and normal antenatal period. The baby was born by normal vaginal delivery with birth weight of 2450 grams. She cried immediately after birth and was breastfed.

However, 3 hours prior to admission, she had poor feeding, lethargy, jerky respiration and excessive secretions. She was exclusively breastfed and weighed 2520 grams. The baby had profuse secretions from the mouth, with irregular respiration and bradycardia with heart rate of 72/min. She had non reactive pupils, hypotonia and was in mild stupor [Table/Fig-1]. Questioning the mother and relatives revealed that the baby was deliberately fed monocrotophos (an organophosphate insecticide) mixed with milk by the maternal aunt, 4 hours prior to admission.

[Table/Fig-1]: Pin-point pupil.

Emergency airway management comprised of immediate oropharyngeal suction, endotracheal intubation and assisted ventilation. In view of signs of cholinergic overactivity, 0.1 mg of atropine was administered. Following administration of atropine, heart rate increased from 72/min to 120/min after 10 min. A presumptive diagnosis of OP poisoning was made based on history, clinical features and prompt response to atropine [1,2].

Gastric lavage and skin decontamination were done. After administration of 2 doses each of 0.1 mg atropine 15 minutes apart, atropine infusion was started at 0.02 mg/kg/hour. The dose was increased to a maximum of 0.08 mg/kg/hour for complete atropinisation as assessed from bronchial secretions, heart rate and pupil size. Atropine infusion was continued for 2 days, after which it was tapered over a 24 hour period and stopped. Loading dose of Pralidoxime (PAM) at 25 mg/kg was given over 30 min followed by infusion at 8 mg/kg/hour for 48 hours [3].

Laboratory investigations showed leucocytosis (White Blood Cell count 18600/cu mm) and normal blood levels of glucose, urea, creatinine and electrolytes. Arterial blood gas showed pH of 7.32, PaO_2 62 mm Hg, PCO_2 52 mm Hg and HCO_3 of 17.1 mEq/1. Prothrombin time (PT) and Activated thromboplastin time (APTT) were normal. Septic screen was negative. Serum cholinesterase level was low at 1.66 kU/L (reference value 4.62-11.5 kU/L) which confirmed the diagnosis of exposure to OPC.

In view of gasping respiration at admission, the baby was put on mechanical ventilation. As respiratory efforts improved and secretions decreased, ventilator settings were weaned and baby was extubated after 72 hours of conventional ventilation. Feeds were started on day 4 of admission and baby tolerated feeds well. Baby was discharged after 10 days of hospital stay with no obvious neurologic or pulmonary sequelae. Parents and relatives were counselled by social worker prior to discharge. Baby had normal growth and development on follow-up visit at 4 months of age.

DISCUSSION

Organophosphates (OP) are widely used as pesticides. Accidental OP poisoning is reported all over the world but is extremely rare in neonates. These compounds phosphorylate the esteric site of the enzyme acetylcholinesterase in the peripheral and central nervous system causing accumulation of acetylcholine at the cholinergic receptor sites leading to cholinergic toxicity [1].

Literature search of PubMed, EMBASE, MEDLINE and Google scholar done from 1970 till June 2022 using the words: neonate, poisoning, organophosphorus, atropine and PAM showed only 8

cases of OP poisoning in neonates including one case of homicidal poisoning in a 15-day-old neonate [Table/Fig-2] [2]. The index 12-day-old female neonate was brought with gasping respiration, excessive oral secretions and cold extremities. The baby was hypothermic, hypotonic, cyanosed, and was in mild stupor. Pupils were pin pointed and capillary refill time was 4-5 sec with bilateral crepitations of lungs.

Author and Year	Age in days	Sex	Presenting symptoms and signs#	Source of poison	Diagnosis	Treatment	Outcome
Kaur I et al., 1996 [11]	25	F	Bradycardia, fasciculations	Not identified	Clinical, low pseudo cholinesterase	Ventilation, Atropine, PAM	Survived
Van Wyk L and Els I 2008 [8]	10	M	Bradycardia	Herbal medicine	Low pseudo cholinesterase	Ventilation, Atropine infusion	Survived
O'Reilly DA and Heikens GT 2011 [12]	13	F	Lethargy, respiratory distress, secretions, poor feeding and miosis	Details not known	Details not known	Atropine	Survived
Meena SS and Kumar TVR, 2014 [13]	6	F	Lethargy, respiratory distress, secretions, poor feeding and miosis	Not identified	Clinical, low pseudo cholinesterase	Atropine, PAM	Survived
Chedda A et al., 2014 [2]	15	F	Nystagmus	Thymit (10% phorate)**	Clinical, low pseudo cholinesterase	Ventilation, Atropine, PAM, inotropes	Died
Kumar P et al., 2015 [14]	8	M	Seizures	Herbal medicine	Clinical, low pseudo cholinesterase	Supportive care	Survived
Verma A et al., 2019 [15]	5	F	Oral mucosal burns	Mosquito repellent	Clinical	Supportive care	Survived
Das JC et al., 2019 [16]	23	-	Bradycardia, cyanosis	Cloth contaminated with pesticide	Clinical	Ventilation, Atropine, PAM	Survived
Present study, 2022	12	F	Bradycardia, cyanosis	Monocrotophos**	Clinical, low pseudo cholinesterase	Ventilation, Atropine, PAM	Survived

[Table/Fig-2]: Clinical profile of neonates with OP poisoning showing source of exposure, presenting features, management and outcome.
**Homicidal poisoning

The diagnosis of OP poisoning was based on the history of exposure and characteristic signs of cholinergic toxicity [1]. History of exposure may not be evident, unless revealed by relatives as in the present case. Presence of features suggestive of cholinergic toxicity warrants a therapeutic trial with atropine even when there is no history of exposure to OPC. Unlike adults, infants and children usually manifest with neurological signs such as stupor, coma, and weakness of muscles. Miosis, excessive salivation and respiratory distress are seen in almost all cases of OP poisoning in infants [3]. The SLUD complex (Salivation, Lacrimation, Urination and Diarrhoea) is characteristic of OP poisoning in adults, but is unreliable in infants and children. Only 15-20% of children present with bradycardia, which was present in the current case at admission. The baby didn't have any fasciculations or seizures, which are seen in 25% of patients [3]. OP poisoning is rare, mimics sepsis and needs high index of suspicion to diagnose. Zweiner RJ and Ginsburg MC observed that diagnosis was made in only 20% of cases prior to transfer to higher centre [4]. These cases are usually diagnosed based on history of exposure or contact and the characteristic clinical picture especially presence of pin point pupils, excessive secretions and the offensive odour of the patient's breath and secretions that characterise these compounds.

The differential diagnosis includes sepsis, bronchopneumonia, opiate overdosage, diabetic ketoacidosis and carbamate poisoning [5]. The onset of symptoms of OP poisoning varies from minutes to several days depending on the dose ingested, route of exposure, lipid solubility and potency of the compound [6]. Most patients become symptomatic within 24 hours of exposure. Most deaths occur due to cardiorespiratory failure, obstruction due to bronchial secretions and delay in diagnosis and initiation of treatment [6]. The hallmark of diagnosis of OP poisoning is reduction in serum and RBC cholinesterase activity. RBC cholinesterase activity is considered to be more sensitive and specific for OP poisoning, and it correlates well with serum cholinesterase activity in 80% of cases [7]. In clinical practice, both serum and RBC cholinesterase levels may be used to confirm the diagnosis of OP poisoning. Decrease in cholinesterase activity by ≥50% confirms the diagnosis of OP poisoning [7].

Therapy is aimed at antagonising cholinergic effects and providing respiratory support as respiratory failure is the usual cause of death [3]. Decontamination prevents further absorption of OPC from the skin and contamination of medical personnel.

Atropine antagonises muscarinic and central effects, but has little effect on nicotinic receptors. The recommended dose is 0.02-0.05 mg/kg i.v. given every 5-10 minutes or as continuous infusion at 0.02-0.08 mg/kg/hour to maintain a steady state [8]. The response to atropine is judged by the resolution of cholinergic signs. However, atropine may be required for days to weeks depending on the fat solubility of the OP preparation [3,9].

PAM is a cholinesterase reactivator which restores the enzyme activity at neuromuscular junction causing reversal of respiratory muscle paralysis. PAM should be administered within 24 hours of OP poisoning at a dose of 25-50 mg/kg as an infusion over 20 minutes. The dose may be repeated after 1-2 hour and then at intervals of 10-12 hours, if cholinergic signs recur or as a continuous infusion to maintain a steady state [10]. Recently, a quaternary ammonium compound glycopyrrolate was found to be as effective as atropine with fewer adverse effects and is being used as an antidote to OP poisoning [9]. After recovery, the patient should be monitored for next 24-48 hours to identify recurrence of effects as the action of antidote wears off. Late effects of poisoning include ventricular tachycardia, pulmonary oedema and demyelinating polyneuropathy [11].

CONCLUSION(S)

OP poisoning is uncommon in neonates and in the absence of history of exposure, diagnosis requires a high index of suspicion as delay in diagnosis has a fatal outcome. A thorough physical

examination to identify miosis and excessive secretions and acute onset of illness in a previously well-baby is an indication to suspect OPC poisoning. To our knowledge, this could be the second case of OPC poisoning with homicidal intention reported in a neonate. Community awareness, social welfare programs and education of girl child would play a major role in preventing such tragedies.

REFERENCES

[1] Leibson T, Lifshitz M. Organophosphate and carbamate poisoning: Review of the current literature and summary of clinical and laboratory experience in southern Israel. Isr Med Assoc J. 2008;10(11):767-70. PMID: 19070283.

[2] Chheda A H, Hiremath A, Khadse S, Kulkarni R, Valvi C, Shobi A. Organophosphorus Poisoning in a Neonate. Pediatr Oncall J Res.2014;11(2):48-49.

[3] Eddleston M, Buckley NA, Eyer P, Dawson AH. Management of acute organophosphorus pesticide poisoning. Lancet Res.2008;371(9612):597-607.

[4] Zweiner RJ, Ginsburg MC. Organophosphate and carbamate poisoning in infants and children. Paediatrics Res.1988;81(1):121-26.

[5] O'Malley M. Clinical evaluation of pesticide exposure and poisonings. Lancet Res. 1997;349(9059):1161-66.

[6] Peter JV, Sudarsan TI, Moran JL. Clinical features of organophosphate poisoning: A review of different classification systems and approaches. Indian J Crit Care Med Res. 2014;18(11): 735-745.

[7] Aygun D. Serum acetylcholinesterase and prognosis of acute organophosphate poisoning. J Toxicol Clin Toxicol Res. 2002;40(7):903-10.

[8] Van Wyk L and Els I. Neonatal cholinergic syndrome-Organophosphate poisoning or herbal medicine intoxication? South African Journal of Child Health Res. 2008;2(1):26-27.

[9] Merril DG, Mihim FG. Prolonged toxicity of organophosphate poisoning. Crit Care Med Res.1982;10(8): 550-52.

[10] Bardin PG, Eeden S. Organophosphate poisoning: Grading the severity and comparing treatment between atropine and glycopyrrolate. Crit Care Med Res. 1990:18(9):956-60.

[11] Kaur I, Jayashree K, Hiranandani M, Singhi SC. Severe organophosphate poisoning in a neonate. Indian Pediatr Res. 1996;33(6):517-19.

[12] O'Reilly DA, Heikens GT. Organophosphate poisoning in a 12-day-old infant: Case report. Annals of Tropical Paediatrics Res. 2011; 31(3):263-67.

[13] Meena SS, Kumar TVR. Organophosphorus Poisoning and Neonatal cholinergic syndrome. Indian Journal of Applied Research Res. 2014; 4(2):36-37.

[14] Kumar P, Sanketh R, Krishnan L. Organophosphorus compound poisoning in newborn. Indian Journal of Child Health Res. 2015;2(2):91-93.

[15] Verma A, Maria A, Singh A. Case 1: Oral Burns as a Presentation of Accidental Organophosphorus Poisoning in a Neonate. Neoreviews Res. 2019;20(1):e37-e40.

[16] Das JC, Hasan SH, Sharmin T, Chowdhury M, Khyrunnessa F, Paul N, et al. Organophosphorus Compounds Poisoning in a Neonate: A Case Report. Mymensingh Med J Res.2019;28(2):470-73.

PARTICULARS OF CONTRIBUTORS:

1. Neonatologist, Department of Neonatology, Dolphin Kids Hospital, Hanamkonda, Telangana, India.
2. Consultant, Department of Pediatrician, Dolphin Kids Hospital, Hanamkonda, Telangana, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Laxman Basany,
Flat 301 B, Legend Galaxy, Kothapet, Hyderabad, Telangana, India.
E-mail: laxmanbasani@yahoo.co.in

PLAGIARISM CHECKING METHODS: [Jain H et al.]

- Plagiarism X-checker: Jun 25, 2022
- Manual Googling: Sep 09, 2022
- iThenticate Software: Sep 20, 2022 (20%)

ETYMOLOGY: Author Origin

AUTHOR DECLARATION:

- Financial or Other Competing Interests: None
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. Yes

Date of Submission: **Jun 15, 2022**
Date of Peer Review: **Jul 28, 2022**
Date of Acceptance: **Sep 16, 2022**
Date of Publishing: **Dec 31, 2022**