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Organophoshorous Poisoning and Neonatal cholinergic syndrome

KEYWORDS: Organophosphorous poisoning, Neonate, cholinergic syndrome

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ABSTRACT *Organophosphorous poisoning in neonates is a rare occurrence in tropics and masquerades clinically as sepsis. We report a case of newborn with such affection who recovered well with treatment.*

Introduction
Organophosphorous poisoning in neonates is uncommon and requires high index of suspicion to diagnose it. The clinical presentation is often similar as sign and symptoms of sepsis like respiratory difficulty, copious oral secretions, diarrhea, decreased oral acceptance, lethargy, jitteriness and seizures. This may require prolonged hospitalization and there may be recurrence of manifestations due to chronic or recurrent exposure. There are few cases reported in literature. [1-3] We report a neonate with such affection, who recovered well with treatment.

Case Report
A 6 days old female baby, delivered by full term normal vaginal delivery with no antenatal and perinatal complications was admitted to our hospital with the history of decrease oral acceptance, vomiting and lethargy for 2 days. There was no history of fever, loose stool or seizures. On examination, child was found to be hypothermic, lethargic, and pale, with mottled skin. She had excessive oral secretions, respiratory distress in the form of chest indrawing, retractions, and nasal flaring associated with tachycardia. CNS examination revealed pin-point pupils but other cranial nerves were normal. A preliminary diagnosis of neonatal sepsis was made and advised for first line antibiotics. Child was hypotonic but neonatal reflexes were fairly elicitable. Her sepsis screen was negative, blood gas analysis showed mild metabolic acidosis, blood dextrose, renal function and electrolytes reports were normal. An ultrasonography of cranium performed and was normal. Due to persistent jitteriness, RD, increased oral secretions, watery nasal discharge and persistent pin- point pupil, history was reviewed. Then they said that an older sibling (5 years) gave something (liquid) after which child had lethargy and decrease oral acceptance. With this history and physical examination findings, possibility of organophosphorus toxicity was considered. There Serum cholinesterase level was very low 0.25 kU/L (Normal 3.93-10.80 kU/L; Spectrophotometry method). Mother`s breast milk and serum samples were normal. The signs and symptoms improved after atropine and pralidoxime. Cholinesterase levels came back to near normal value after 6th day of exposure and the patient was discharged. The patient remained asymptomatic and was accepting breast milk feeding well. The baby is being followed up regularly in pediatric clinic and she is showing normal developments without squeal.

Discussion
Organophosphorus poisoning in neonates is rare and a very few cases have been reported so far. Most of the cases reported are of babies born to mother who had organophosphorus 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) Few cases have been reported when neonates were given herbal medicines contaminated with organophosphorus compounds. (6) In our case, history of older sibling gave something (liquid) after which child had lethargy and decrease oral acceptance but details about this liquid not provided by parents so possibility of homicidal not be excluded and sex of child (female) also supportive for this. CNS depression manifests as coma, stupor, lethargy, hypotonic, and muscle weakness is seen in 90% children. (7) Respiratory difficulty, miosis, and excessive salivation also occur common .(1,7,8) The rapidity of onset of symptoms of OP poisoning depends on the dose, route of exposure and the compound potency. It may vary from minutes in case of massive ingestion or inhalation to several days if the agent is highly fat soluble. (9) On an average most patients become symptomatic within 24 h of exposure. Diagnosis of organophosphorus poisoning in neonates needs high index of suspicion as the clinical presentation simulates sepsis. It can be proved by demonstrating low levels and low activity of RBC Cholinesterase and pseudocholine esterase in the baby. Mother's serum or breast milk cholinesterase level could add in confirming the diagnosis. (10,11) A decrease of 40% from baseline produces symptoms and a decrease of 80% from baseline leads to severe symptoms. 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 25% of the control value. Treatment includes general measures like airway control and adequate oxygenation and ventilation, and cardiovascular support, and ECG monitoring. Decontamination is essential step of treatment for prevent further absorption from the skin as well as to prevent contamination of health care providers. The mainstays of medical therapy in organophosphate (OP) poisoning include atropine, pralidoxime (2-PAM), and benzodiazepines (eg, diazepam) (10,11) Atropine antagonises the central and the muscarinic effects. 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 at 0.08 mg/kg/h has also been used to maintain a steady atropinised state (12). 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(12). Pralidoxime (2-PAM, Protopam) nucleophilic agent that reactivates the phosphorylated choline esterase there by reverses muscle paralysis (nicotinic effects). Current recommendation is administration within 48 h of OP poisoning. 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 (8). The dose may be repeated after 1-2 h and then at 10-12 h intervals if cholinergic signs recur(13). Because it does not significantly relieve depression of respiratory center or decrease muscarinic effects of AChE

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poisoning, administer atropine concomitantly to block these effects of OP poisoning. Signs of atropinization might occur earlier with addition of 2-PAM to treatment regimen. 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. The patient should be followed up regularly as the organophosphorus compounds may affect the development of an infant, especially the social milestones (14,15).

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