various subsets of JIA to define the rightful place of leflunomide.

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Recurrent Neonatal Organophoshorus Poisoning

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Organophosphorus poisoning in neonates is extremely rare and needs high index of suspicion to diagnose it. The clinical presentation is often confused with the features of sepsis like apnea, copious oral secretions, diarrhea, letharginess, seizures. There may be recurrence of manifestations due to chronic exposure. We report a classic case admitted in the intensive care unit of our hospital.

Key words: *Apnea, Seizures, Sepsis.*

Organophosphorus poisoning is rare in neonates.Transplacental route is the most common mode of transmission,others being inhalation and ingestion; either accidental or homicidal. The clinical manifestation often simulates that of sepsis and leads to diagnostic dilemma.Careful clinical examination and early intervention is needed to treat the patient.

CASE REPORT

A 17-days-old Egyptian girl, a product of non-consanguineous parents delivered by full term normal vaginal delivery with no antenatal and perinatal complications was admitted to our hospital with the history of poor feeding and poor activity of one day duration. There was no history of fever or seizures. On examination, child was found to be hypothermic, lethargic, and pale, with mottled skin. She had recurrent apnea associated with bradycardia. She had profuse salivation and frothy secretions from mouth. CNS examination revealed pin-point pupils but other cranial nerves were normal. Child was hypotonic but neonatal reflexes were fairly elicitable. Her hemogram and electrolytes were normal and blood gas analysis showed mild metabolic acidosis. The baby was put on nasal CPAP along with other supportive therapy. Careful interrogation of parents revealed the history of pesticide (Diazinon) spray at home, two days prior to the development of symptoms in the baby. With this history and physical examination findings, strong possibility of organophosphorus toxicity was considered and child was treated with multiple doses of Atropine and two doses of pralidoxime. The patient's activity improved after atropine and pralidoxime doses. There were no further episodes of apnea, bradycardia or miosis. The patient was weaned off from CPAP. Her septic and metabolic screen came negative. Serum cholinesterase level was very low 137U/L (Normal 5000-12000U/L).The patient was discharged without sequale after two days. The patient remained asymptomatic at home and was feeding (breast milk) well. She was admitted again with similar clinical presentation within 48 hours and was treated with atropine and pralidoxime till recovery. She had a very low serum cholinesterase level at this admission (150 U/L). In view of repeated poisoning in the child, breast milk was stopped temporarily as it was suspected as one of the source of repeated exposure and the mother's serum cholinesterase level was also sent, which turned out to be low too (1600 U/L). Child had complete recovery after treatment.

Cholinesterase levels came back to normal value after few days. Parents were sent to social worker for counseling but child abuse was ruled out. The baby is being followed up regularly in pediatric neurology department and she is showing normal developmental milestones according to her age.

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 [1-3]. Few cases have been reported when neonates were given herbal medicines contaminated with organophosphorus compounds [3]. In our case, the house was sprayed with very strong organophosphorus compound, diazinon and the baby might have been exposed to poison either by inhalation or through breast milk.

Diagnosis of organophosphorus poisoning in children 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 [4,5]. Treatment includes general measures like airway support and ventilation, cardiovascular support, pulse oximetry and ECG monitoring. The mainstay of medical therapy in organophosphate poisoning include atropine, pralidoxime (2-PAM), and benzodiazepines (eg, diazepam) [4,5]. Atropine reverses muscarnic effects and PAM reactivates the phosphorylated choline esterase there by reverses muscle paralysis (nicotinic effects). The patient should be followed up regularly as the organophosphorus compounds may affect the development of an infant, especially the social milestones [6,7].

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