LETTER TO EDITOR (VIEWERS CHOICE)

UNUSUAL CAUSE OF ENCEPHALOPATHY IN AN INFANT - ORGANOPHOSPHATE POISONING

Poovazhagi V, Ehilarasu R. Sivaraman T

Key words: Organophosphate poisoning, infant

A 45 day old infant was brought to our emergency room with history of irritablity, poor feeding and loose stools for two days duration. There was no history of fever. The infant was delivered as full term with birth weight of 2.6 kg with an uneventful neonatal period and was on exclusive breast feeds. On examination baby had tachycardia with a heart rate of 190/min, respiratory rate of 67/min, with increased work of breathing, afebrile and glasgow coma scale (GCS) was 6. Infant had bilateral constricted pupils, and was hypotonic. Baby was noted to have non convulsive status epilepticus and was controlled with 2 doses of inj lorazepam and loading dose of inj phenobarbitone and was intubated in view of poor GCS. Initial blood sugars were 250 mg/dl , urine was negative for ketone, and blood gases were suggestive of respiratory alkalosis . Electrolytes, urea , creatinine, complete blood counts , SGOT ,SGPT, lactate and ammonia were within normal limits. CT brain was normal. Lumbar puncture revealed a normal CSF. At 6 hours of admission infant was found to have dehydration, with excessive tracheal and oral secretions. Pupils were pin point at this examination, infant had persistent tachycardia. Urine output was 2ml/kg/hour. In view of persistent encephalopathy without fever, profuse secretions and sweaty moist skin with dehydration possiblity of OPC poisoning was thought of and samples were sent for serum pseudo cholinesterase levels. A trial of atropine (0.05 mg/kg) was given and the heart rate further increased to 260/ minute after two doses. ECG revealed sinus tachycardia. Infant received Pralidoxime infusion (25 g/kg/dose for two doses). On day 2 of admission infant had no excessive secretions, heart rate was within normal limits and GCS improved to 12. Infant was weaned off the ventilatory support and on day 4 pupils increased in size and was normal at discharge on day 6 of hospitalization. Initial plasma pseudocholinesterase levels were very low 825u/L (normal 6000-8000u/L). Repeat pseudocholinesterase levels on day 6 of illness had increased to 3400U/L. Retrospective history did not reveal the exact source of poisoning ,however the mother gave a history that the infant enroute to the clinician visit on the first day of illness was through a paddy field where recent insectiside spraying activity was undertaken.

Organophosphate poisoning in neonates and young infants is rarely encountered. Unlike in adults where most of the poisoning is due to suicidal attempts, in young infants it is unintentional(1) as accidental exposure or homicidal. The absorption of organophosphorus compounds could be through the GIT, skin, mucosal membranes (inhalational and through the conjunctiva) or rarely through application of home-made shampoo.(2) The clinical presentation in young children and infants are different from those in adults.(2,3) Organophosphate compounds are anti-esterase insecticides and poisoning results in the bonding of these compounds with the enzyme acetylcholinesterase and this bonding can be reversible

with reactivation of the enzyme over time or can become irreversible by the process called ageing. This inactivation of enzyme leads to accumilation of the neurotransmitter acetylcholine at the muscarinic and nicotinic sites and this leads to the classical toxidrome described for organophosphate poisoning. Muscarinic symptoms include diaphoresis, wheezing, pulmonary edema, increased pulmonary and nasopharyngeal secretions, increased intestinal motility bradycardia and miosis. Nicotinic symptoms include fasiculations, tachycardia, respiratory muscle weakness, decreased respiratory efforts, tachycardia and hypertension. CNS symptoms include, irritablity, seizures, restlessness, confusion, headache, ataxia, coma, respiratory paralysis. The classic symptoms of organophosphate poisoning does not occur in children. In the absence of the baseline serum cholinesterase levels the acute serum sample and a convalescent sample may be used for comparison. Red cell cholinesterase levels can be diagnostic but not commonly available. But clinical diagnosis and response to atropine and pralidoxime is much more rewarding than awaiting serum levels for diagnosis. Atropine may not be very useful in reversing the neuromuscular toxicity (respiratory muscle weakness) as this does not bind to the nicotinic receptors(3). Presence of tachycardia is not a contra indication to atropine. Reported incidence of mortality in organophosphate poisoning is children is about 6.4%.(4) Cases have been reported even in the new born period due to accidental exposure by spillage over the skin(5). In this infant the presence of encephalopathy, excessive salivation, sweating with moist skin and unexplained dehydration with pin point pupils were the clue for diagnosis.

REFERENCES

1. Lifshitz M, Shahak E, Sofer S. Carbamate and organophosphate poisoning in young children. Pediatr Emerg Care 1999; 15: 102-103.
2. Sadaka Y, Broides A, Tzion RL, Lifshitz M. Organophosphate acetylcholine esterase inhibitor poisoning from a home-made shampoo. J Emerg Trauma Shock. 2011; 4: 433-434.
3. Van Heel W, Hachimi-Idrissi S. Accidental organophosphate insecticide intoxication in children: a reminder. Int J Emerg Med 2011; 4: 32.
4. Levy KF, Tenenbaum AN, Wexler ID, Amitai Y. Unintentional organophosphate intoxication in children. Pediatr Emerg Care. 2007; 23: 716-718.
5. Poovazhagi V, Srinivasan K, Gowrishankar NC, Hussain MM. Organophosphate poisoning in a neonate. Indian J Pract Pediatr. 2007; 9: 135-136

From: Department of Pediatric Intensive care, Institute of Child Health and hospital for Children, Chennai.

Address for Correspondence: Dr Poovazhagi V, 8/11 Manjolai street, Kalaimagal nagar, Ekkaduithangal, Chennai 600 032, India.
Email: poomuthu@yahoo.com.

E-published: 1st July 2013 Art # 37

DOI: 10.7199/ped.oncall.2013.37

Quick Response Code