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CASE REPORTS

THE MYSTERY LIES IN THE EYES AND THE HISTORY - ORGANOPHOSPHATE COMPOUND POISONING IN AN INFANT

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ABSTRACT

Pesticides are common among the poisoning substances in children. Exposure to organophosphate compounds (OPC) leads to acute poisoning from the irreversible inhibition of the enzyme acetyl-cholinesterase. A 6 months old infant presented with incessant cry, refusal of feeds, hurried breathing and lethargy. On examination, the infant was febrile, had tachycardia, tachypnea with pooling of oral secretions, eyelid twitchings, pinpoint pupils and upbeat nystagmus. Since the infant had signs and symptoms of cholinergic toxindrome, OPC poisoning was considered. Serum cholinesterase level was very low (91 U/L). She was treated with intravenous (IV) atropine infusion and pralidoxime to which she responded. We conclude that in infants when pin point pupils and excessive oral secretions are noted, one should consider rare causes like toxin exposure.

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KEYWORDS

Cholinergic toxindrome, infant, pupillary, pesticide drift.

Introduction

Poisoning is the fourth most common cause for injury related mortality in children as per World Health Organization (WHO) report on childhood injury prevention.[1] Pesticides are common among the poisonous substances in children less than 5 years of age in South Asia.[2] Organophosphate compounds (OPC) were developed as human nerve gas agents during the 1930s and some of these were later adapted as pesticides at lower doses.[3] High exposure to OPC leads to acute poisoning from the irreversible inhibition of the enzyme acetyl cholinesterase (AChE), resulting in cholinergic toxindrome (including constricted pupils, excessive salivation, bronchoconstriction, mental confusion, convulsions or tremors, and in some cases, death).[3,4] Additionally, delayed polyneuropathy has been described in association with high exposure.[3,4] We present a 6 months old infant with OPC poisoning due to environmental exposure and diagnosis based on clinical history and examination and confirmed by very low levels of serum cholinesterase.

Case Report

A six months old female infant was brought to the emergency room (ER) with hurried breathing and lethargy for the past 3 hours. This infant developed incessant cry, excessive perspiration, refusal of feeds, vomiting and two episodes of loose stools 16 hours prior to the ER visit. At arrival to the ER, she was sick looking, febrile (102°F), and her breathing was noisy with oral secretions. On examination, she was lethargic with poor interaction with parents and hypotonic with brisk reflexes. She had eyelid twitchings, pinpoint pupils and upbeat nystagmus. She was tachypneic (rate: 65/min) with excessive work of breathing in the form of nasal flaring, subcostal, intercostal and substernal retractions. Her heart rate was 220/min, blood pressure was 82/47 mm Hg and capillary refill time was <2s. Abdomen was soft and doughy with good bowel sounds. The differential diagnosis considered at this stage were acute central nervous system (CNS) infection, pontine hemorrhage and acute poisoning due to toxins. She was initially stabilized with airway suctioning, oxygen through prongs, intravenous (IV) fluids and IV ceftriaxone. She had seizures after admission which were aborted with IV lorazepam. Her blood sugar was 92 mg/dl. ECG showed sinus tachycardia with normal P waves and QRS complexes. Since she continued to have shallow respirations with desaturation she was electively intubated and ventilated. Since the infant had signs and symptoms of cholinergic crisis notably the pinpoint pupils, suspecting inadvertant OPC exposure, blood sample for cholinesterase enzyme level was sent and IV atropine (0.05 mg/kg) was given. Then an elaborate history was taken. She was the fourth offspring to parents of migrant workers from Rajasthan. She was a well thriving infant (6 kg) born at term with birth weight of 2 kg and immunised for age. Two days prior to the onset of symptoms her family shifted to a new rented house. This house was sprayed with pesticides two days prior to their arrival to eradicate termites. Her mother said that there was obvious smell of pesticides inside the house. The infant being non-ambulant would have been constantly exposed to the air contaminated with OPC in the house. On requesting, the parents brought the pesticides used. The compounds used were Ekalux (Quinalphos 25%) - OPC, Luphos (Monochrotophos 36%) - OPC, ACT 150 (Thiamethaxon 25%) - neonicotinic compound. Blood counts showed neutrophilic leucocytosis (white blood cells: 20,600/cumm, polymorphs - 74%, lymphocytes - 23%) with thrombocytosis (platelets: 7,38,000

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cells/cumm). CRP was negative. Serum electrolytes, renal function tests and SGPT were normal. Chest X-ray and ultrasound cranium were normal. Serum cholinesterase level was 91 U/L (normal range 5320 - 12920 U/L). She was treated with IV atropine infusion (0.02 mg/kg/hour), titrated to drying of secretions and pralidoxime (30 mg/kg) was given to reactivate cholinesterase enzyme. Subsequently eight hours later she improved clinically and was gradually weaned off from ventilator and extubated 24 hours later. Repeat serum cholinesterase levels were in rising trend (day 2 - 146 U/L and day 3 - 605 U/L) following which atropine infusion was gradually weaned and pralidoxime discontinued after 48 hours. Oxygen was weaned off by 48 hours and she was started on oral feeds. Her vitals were stable with no weakness and chest was clear. Her serum cholinesterase level was 1621 U/L on day 7 and she was discharged. On follow up two weeks later, she was alert and active with no respiratory or neurological weaknesses.

Discussion

Pesticides are still among the common causes for acute poisoning in children of low-middle income countries like India.[1] The route of OPC exposure determines the rapidity of symptom onset. Of the common routes of exposure - inhalational, cutaneous and ingestional; inhalational has the fastest onset, within few minutes of exposure.[5] The clinical manifestations are based on the overstimulation of nicotinic and muscarinic receptors. Following exposure to OPC, muscarinic symptoms SLUDGE (Salivation, Lacrimation, Urination, Defecation, Gastric cramps) manifest within minutes to hours.[5] Nicotinic signs include muscle fasiculations, weakness and respiratory paralysis. Central nervous system (CNS) signs include depression, irritability, seizures and coma.[5] The symptoms are categorized as acute (within 24 hours), delayed (intermediate syndrome) 24-hours to 2-weeks and late (delayed polyneuropathy) beyond 2-3 weeks.[5] Unlike adults, infants mainly present with acute CNS depression and do not demonstrate typical muscarinic effects. Symptoms such as acute respiratory failure, bradycardia and fasciculations are more common in children.[6,7] Tachycardia, rather than bradycardia, has also been observed upon presentation in majority of children as noticed in our case.[6]

The initial management should be directed towards securing airway, as respiratory failure is the usual cause of death and maintain circulation. The diagnosis of OPC poisoning in our patient was based on clinical findings associated with cholinergic crisis (excessive oral secretions, pinpoint pupils and respiratory failure) and history of inhalational exposure. Specific antidote for OPC is atropine and pralidoxime based on the type of pesticide to which the individual is exposed. Atropine reverses the central and muscarinic effects of acetylcholine but has little effect on its nicotinic action.[8] Pralidoxime is a cholineterase reactivator which hastens the restoration of the enzyme activity at the neuromuscular junction thereby reversing respiratory muscle paralysis and muscle fasciculation. It is administered as an intravenous infusion over 20 min in a dose of 25-50 mg/kg within 24-48 hrs of exposure. The dose may be repeated after 1-2 hrs of exposure and then at 10-12 hrs intervals if cholinergic signs recur.[9]

Pesticide drift refers to unintentional diffusion of pesticides in environment and is a recognized mode of accidental pesticide poisoning as in our case.[2] Necessary steps should be taken in advance for the residents to reduce human exposure during application of OPC pesticides in methods such as aerial spraying for domestic use.[4] Infants are more prone to adverse effects of pesticides due to higher body surface area exposure relative to body weight.[10]

Conclusion

Although OPC poisoning is rare in infants, having a high index of suspicion will help in early diagnosis and prevent morbidity as well as mortality. Diligent history taking, including the events that led to the current presentation and detailed physical examination including the examination of pupils helped in the early diagnosis and initiation of specific treatment. Diagnosis can be confirmed by measuring serum cholinesterase levels and also be used for monitoring recovery. Pesticide drift as a mode of accidental poisoning should also be kept in mind and parental awareness will help in avoiding this easily preventable childhood poisoning.

Compliance with Ethical Standards

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