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Rare Homicidal Organophosphorus Poisoning in a Neonatal Twins

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Abstract

Organophosphate (OP) compounds widespread use throughout the world, mainly in agriculture, may cause serious life-threatening conditions. We report the case of neonatal twins presented with poor feeding, lethargy, jerky respiration and excessive frothy secretions, pinpoint pupils and bradycardia. Father disgruntled over his wife giving birth to twin girls, attempted to poison the new born twin females and landed himself behind the bars. The symptoms appeared and poisoning was confirmed by hospital authorities by CCTV at the delivery center and was referred to our Hospital.

Diagnosis of OP poisoning based on history of exposure, characteristic clinical manifestations of toxicity. Management consists of airway support ventilation, fluid resuscitation as well as atropine incremental doses and antidote Pralidoxime. Clinical suspicion and prompt specific treatment save lives without any sequelae.

Plasma pseudo cholinesterase analysis is a cost effective and a reliable indicator for organophosphate intoxications and could be used for confirmation and therapeutic monitoring.

Keywords: Plasma Pseudo Cholinesterase (PCE); Insecticides; Intoxication; Organophosphorus Compound (OP); Antidote; Organophosphorus Intoxication (OI)

Introduction

Organophosphates (OP) are frequently used in rural areas in agriculture. Intentional ingestion of organophosphates (Chlortyriphos) is associated with a high mortality rate [1]. Intoxication of OP induces irreversible inhibition of acetylcholinesterase. Organophosphates phosphorylate the serine hydroxyl group of acetylcholine, leading to Acetylcholinesterase (AChE) deficiency leading to accumulation of acetylcholine at the cholinergic synapses [2].

AChE is critical for nerve function, so the irreversible blockage of this enzyme, which causes acetylcholine accumulation, results in muscle overstimulation and fasciculation. If this blockade is not reversed within 24 hours, large amounts of acetylcholinesterase are permanently destroyed [3].

This enzyme is found in erythrocytes and plasma. Severity and/or the elimination time of OI can be determined, by measuring cholinesterase in blood, either by measuring plasma Pseudocholinesterase (PCE) or by measuring the cholinesterase in erythrocytes (which is thought to reflect the cholinesterase in neurons and neuromuscular junctions).

Measurement of cholinesterase in blood widely available cost effective and therefore commonly used [4].

Herein, we report one day old female twins were brought, upon being intentionally poisoned with an organophosphate solution, developed severe OI associated with Respiratory failure and central nervous system (CNS) depression.

Case Study

One day girl twins were brought into the emergency department with poor feeding, lethargy, jerky respiration and excessive secretions for preceding 4 hours. Neonates were breastfed and had no history of trauma, exposure to chemicals, or any previous illness. On examination twin, one weighed 2.5 kg, had profuse clear secretions from the trachea and oropharynx.

At emergency, ER doctor had smelled a peculiar and unpleasant odour. The clinical examination of the twins showed pinpoint pupils (2 mm diameter), frothing and secretions from mouth and nose, severe respiratory distress characterized by chest wall retractions.

Tachypnea rate was 70/min and irregular and bradycardia heart rate was 60/min. Elder twin baby had pin-point non-reactive pupils, generalized hypotonia, absent deep tendon reflexes, and occasional twitching movements of the limbs (Figure 1).

Figure 1: Twin 1 requiring invasive ventilation-(tachycardia) excessive cholinergic activity.

Airway management comprised immediate clearing of secretions, intubation and ventilation for 2 days followed assisted Non-invasive ventilation with CPAP for 4 more days. Clinical signs suggestive of cholinergic over-activity, atropine was administered at incremental doses of 0.05 mg/kg intravenously and repeated within fifteen minutes and Pralidoxime was given in 30mg/kg dose state. It was followed by an increase in heart rate to 120/min and pupillary dilatation to 3 mm. A presumptive diagnosis of organophosphate was made. As the respiratory efforts improved and secretions decreased the FiO2 and ventilator rates were gradually decreased, put on synchronised intermittent mandatory ventilation on for three days and extubated to CPAP. No obvious neurologic or pulmonary sequelae. On follow up visit at 2 months of age the baby was thriving well and had attained developmental milestones appropriate for the age. Shortly thereafter, another twin baby with the exact same unpleasant odour, respiratory distress, was brought to the emergency department as baby was pale in colour and poor perfusion with poor breathing efforts required fluid resuscitation and non-invasive ventilation. She was afebrile and had a heart rate of 108 beats/min, the respiration was shallow at a rate of 51 breaths/min with bilateral wheezing and bronchial secretions. Her pupils were 1 mm in diameter, and the CRT was prolonged up to 4 s. She was stabilized with non-invasive ventilation for two days and a 20 ml/kg bolus of saline fluid through a secured intravenous vascular catheter. Because OI had been suspected earlier for her sibling, atropine (0.05 mg/kg) was given to prevent further decline. All laboratory values were normal, except for a decreased PCE. The twin one PCE was 0.3 kU/l and the twin 2 was 0.2 kU/l (laboratory reference range: 4.6-10.4 kU/l). The children were shifted from the NICU on day 6 and discharged from the hospital on day 10 without obvious neurologic or pulmonary sequelae. On a follow-up visit at 2 months of age, the baby was thriving well and had attained developmental milestones appropriate for the age.

Results and Discussion

Organophosphorus poisoning in neonates is not so common and only five cases have been reported so far as per our knowledge. Most of the cases reported are from India, as it is common to use in rural India for agriculture and few cases of transplacental organophosphorus poisoning by insecticidal ingestion, either suicidal or homicidal, just before delivery. Poisoning can be either by inhalation, or ingestion, [4-6]. Few cases have been reported when neonates were given herbal medicines contaminated with organophosphorus compounds [6]. Easy availability of these compounds, mainly in countries like India, has resulted in a gradual increase in accidental and suicidal poisoning. Mainstay of treatment is supportive therapy and atropinisation for the reversal of symptoms. Reduction in serum and Red-Blood-Cell (RBC) cholinesterase activity are confirmatory laboratory parameters in diagnosing OP poisoning. After recovery, the patient should be monitored closely for up to 72 hours to ensure that symptoms do not recur as the effects of atropinisation wear off.

Conclusion

This case report emphasizes that misuse of organophosporus (OP) compounds, may be life threatening. High index of suspicion particularly if clinical signs are correlating with OP poisoning. Management consists of supportive therapy which includes temperature, airway support and early specific antidote will be useful.

PCE analysis is an easy indicator of OI and can be used for treatment monitoring and progression of the disease.

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