

ed that while planning a Nutritional and Health Education Programme, beliefs regarding culturally accepted and restricted foods in the particular area should be given due consideration.

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Transplacentally Acquired Carbamate Insecticide (Baygon) Poisoning in a Neonate

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Human beings can be exposed to cholinesterase inhibitor pesticides (organophosphates and carbamates) by different ways(1) but probably one of the most indirect ways of exposure to pesticides concerns the

mother and the developing fetus. Only reports of fetal death with malformation of the limbs in which the mothers had organophosphorus insecticide poisoning(2) are available, but no newborn was reported to have overt symptoms of poisoning. Here we report the potential of carbamates to induce acute clinical toxicity in a newborn

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baby following transplacental passage of the poison from the mother.

Case Report

A 25-year-old primigravida mother with uncomplicated pregnancy was hospitalized at 37 weeks of gestation with acute toxic manifestations of cholinesterase inhibitor poisoning 2 hours after consumption of unspecified quantity of Baygon (Propoxur) granules. On hospitalization, she was atropinized by continuous intravenous infusion of atropine at a rate of 1 mg/minute. While being atropinized, she went into spontaneous labor and delivered a 3 kg male baby after 9 hours of start of atropine therapy. Fetal bradycardia (40-60 per minute) and meconium stained liquor were noticed before delivery and the baby had severe asphyxia with apgar score of I at 1 minute and 5 at 5 minutes, requiring resuscitation with IPPV for 30 minutes.

After successful resuscitation, he was transferred to neonatal intensive care unit (NICU) where his sensorium was found obtunded with bilateral fixed dilated pupils, absence of all neonatal reflexes including oculovestibular, tonic neck and stretch reflexes without any decerebration. He had flaccid paralysis of all four limbs, twitching of facial and limb muscles. He also had marked tachycardia (heart rate >200/min) but no cardiomegaly or murmurs and in addition had shallow respiration with poor effort (RR 52/min; no recession). The baby was pink in room air and arterial blood gases done at 4 hours of age showed pH 7.34, paO_2 120, pCO_2 25 and HCO_3^- 13.5. The baby also had hyperglycemia with blood sugar ranging from 190-250 mg/dl. Lungs were clear with normal breath sounds. Soon after transfer to NICU; Inj. Pralidoxime (PAM) 75 mg IV was administered slowly

over 30 minutes. At 4 hours of age, he developed multifocal clonic seizures which required administration of luminal and subsequently dilantin. By then tachycardia started settling with a heart rate varying between 150-160/min and pupils became of normal size and reaction. Subsequently, he developed copious oral secretion and miosis for which atropine was administered in the dose of 0.02 mg/kg IV at 7 hours of age and Inj. PAM 75mg IV was repeated at 10 hours of age. At 24 hours, he again developed signs of parasympathetic over activity hence 2nd dose of atropine and 3rd dose of PAM were repeated. Throughout, the baby was comatose and maintaining normal temperature but at 36 hours of age he developed hyperthermia, tachycardia (HR 170-180/min), mydriasis and retention of urine. At 48 h of age, he developed recurrent apneic spell. The arterial blood gas showed hypoxemia and hypercarbia requiring mechanical ventilation. While on ventilator with hardly any spontaneous respiratory efforts, the child developed recurrent attacks of generalised seizures inspite of adequate anticonvulsant therapy. He developed intractable hypotension and died after 18 hours of start of ventilation.

Discussion

The toxicologic effects of carbamates are primarily due to a build up of Acetylcholine resulting in muscarinic, nicotinic and minimal central effects of cholinergic toxicity(1,3). Unlike adults and children, in whom muscarinic effects of cholinergic toxicity predominate at time of initial presentation(1), in this neonate, nicotinic and central muscarinic effects predominated. The nicotinic effects of acute toxicity encountered soon after birth were diminished respiratory effect, twitching of muscles, flaccid paralysis of all four limbs and hy-

perglycemia. Tachycardia and bilateral fixed dilated pupils noticed after successful resuscitation of the baby were thought to be due to the transplacental passage of atropine injected to mother. Atropine undergoes rapid placental Transfer with significant fetal uptake within 5 minutes of administration. Fetal levels of 24-87% of maternal levels have been reported depending on the time after injection and these effects could last for few hours(4,5). However, these manifestations could also be attributed to the nicotinic effects of severe anticholinergic toxicity(6-10) and hence making management very difficult. However subsequently, the constricted pupil which dilated following administration of IV atropine could be considered presumptive evidence of atropinization(11,12).

Though the CNS effects of carbamate usually donot occur due to its inability to cross the blood barrier well(13), in this neonate the central muscarinic effects were quite marked which might have caused severe perinatal asphyxia. In addition, transplacental passage of atropine following energetic maternal atropinization also might have contributed to birth asphyxia.

CNS manifestations of altered sensorium, recurrent seizure, cessation of spontaneous respiration and hypotension which the baby had, though indicative of hypoxic-ischemic encephalopathy (HIE) were also contributed by mixture of nicotinic, muscarinic and central effect of carbamate toxicity and the effect of atropine. Atropine alleviates muscarinic and CNS effects of acetylcholine following carbamate toxicity, but by itself can cause coma(14). As atropine treats the effects of the excess acetylcholine being generated but does not reverse the mechanism of poisoning itself(7,8,15,16), larger doses of the drug at

frequent intervals (energetic atropinization) is needed in acute carbamate toxicity. In the present case it was very difficult to titrate the dose of atropine to control cholinergic toxicity because of confusing picture produced by severe, birth asphyxia.

With the lack of studies in human subjects, the use of oximes in carbamate poisoning is controversial. PAM does not cross the blood brain barrier; however, it does seem to help reverse CNS effects(7,8). Toxicologists are divided in their beliefs, but most concur that PAM should be used in the acute management of poisoning if the poisoning is potentially fatal and if a patient known to have carbamate poisoning, does not respond to atropinization(10-13,17).

To monitor therapy, estimation of RBC cholinesterase level has been recommended(6,17), but its level may not correlate accurately with the clinical status and the means to obtain them are not readily available in many hospitals(18).

Death in this baby though seems primarily due to HIE following severe perinatal asphyxia, carbamate poisoning might have contributed significantly to it. There is no information of similar poisoning in newborn baby and the clinical description and treatment recommendation have been derived from adult data.

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