Lesson of the Week

Carbon monoxide poisoning in an infant

H VENNING, D ROBERTON, A D MILNER

Carbon monoxide poisoning presents two risks to infants. The non-specific presenting symptoms make it difficult to diagnose, and the special characteristics of fetal haemoglobin may lead to greater tissue hypoxia than in older children or adults.

Case report

A 13-week-old baby girl travelled for six hours in a carry-cot that had been placed behind the rear seat of a one-year-old estate car. At the end of the journey she was hypotonic and had vomited. She was taken in the same car to a local hospital and was transferred by ambulance to our care.

Shortly after arrival she began convulsing. There was no clinical evidence of hyperpyrexia, aspiration, or infection. Plasma electrolyte, glucose, and ammonia concentrations and a chest x-ray examination were normal. Her arterial blood pH was 7.33, with a base deficit of 8 mmol (mEq)/l. The cerebrospinal fluid was normal. Her blood carboxyhaemoglobin (COHb) saturation, measured by a spectrophotometric method, was 60% two hours after she was removed from the car. COHb saturations at 5 hours and 17 hours after exposure were 25% and 10% respectively. Her parents recalled having minor symptoms themselves when questioned directly. The mother's COHb at 5 hours was 10%, the father's—who smoked 10 cigarettes a day—15%. The infant's 6-year-old brother was asymptomatic, and his COHb saturation was not measured.

The baby was given 100% oxygen, dexamethasone, and fluids were restricted. She was unconscious for 48 hours. The convulsions were difficult to control and occurred intermittently for 18 days. Persisting minor neurological abnormalities resolved six weeks later, and her progress has been excellent. A hole in the floor of the rear of the car had been caused by a minor accident a few weeks before and exhaust gases had probably leaked into the car.

Comment

Carbon monoxide poisoning is a recognised sequel of fires and accidents with home heating and cooking units. Faulty motor vehicle exhaust systems may allow fumes containing carbon monoxide to leak into the passenger compartment, especially if the bodywork is damaged.¹ Carbon monoxide is often not suspected because of the non-specific nature of many of the effects, and young infants are at particular risk. An altered state of consciousness with or without convulsions is a common presentation of systemic, infective, or metabolic illness at this

Carbon monoxide poisoning is harder to diagnose in infants than in older children or adults

age. When there are no signs or symptoms in other family members an environmental cause may not be considered.

The affinity of haemoglobin for carbon monoxide is approximately 240 times that for oxygen.² In addition to displacing oxygen, carbon monoxide binding to haemoglobin causes a definite shift of the oxygen dissociation curve to the left, owing to the increased affinity of the remaining haemoglobin for oxygen.² Thus lower partial pressures of oxygen in tissues are required before oxygen is released. Fetal haemoglobin (HbF) probably has a similar affinity for carbon monoxide, as adult haemoglobin (HbA),^{3 4} but it also has an oxygen dissociation curve that is to the left of HbA. HbF accounts for 65% to 85% of the haemoglobin present in an infant at birth, falling to 20% at 3 months of age, and 5% to 10% at 5 months.⁵ When the HbF dissociation curve shifts even further to the left because of carbon monoxide the tissue hypoxia in a young infant may be greater for the same degree of exposure to carbon monoxide than in older children or adults. Furthermore, a critically diminished concentration of tissue oxygen may increase the affinity of intracellular oxygen-transport systems for carbon monoxide. For example, the affinity of myoglobin for carbon monoxide in skeletal muscle and myocardium rises to 2.5 times that of haemoglobin if the P_{aO_2} is 5.32 kPa (40 mmHg) or less.⁴

When unexplained neurological symptoms and signs occur in an infant who has been a passenger in a vehicle the COHb saturation must be measured. Laboratory diagnosis is simple and quick, and treatment with 100% oxygen or hyperbaric oxygen will reduce the half life of COHb and improve tissue and cellular oxygenation.

Correspondence should be addressed to D Robertson, Department of Child Health, E Floor, East Block, University Hospital, Queen's Medical Centre, Nottingham NG7 2UH.

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University Hospital, Nottingham NG7 2UH

H VENNING, BMEDSCI, MRCP, paediatric senior house officer

D ROBERTON, MD, FRACP, paediatric registrar

A D MILNER, MD, FRCP, professor of paediatric respiratory medicine