

forehead, and over the next six months these grew and coalesced into a small reddish coloured plaque. This lesion has subsequently remained the same size. At 2 years two small hypomelanotic macules were visible under Wood's light, but no other skin signs have appeared.

Discussion

Seizures in the first year of life, especially infantile spasms, are an important presenting feature of tuberous sclerosis. Early diagnosis is important for genetic counselling and predicting prognosis. In infants with infantile spasms a careful search for the signs of tuberous sclerosis should be performed, in particular, skin examination (including Wood's light), retinal examination, and cranial computed tomography. Diagnostic skin signs (facial angiofibromas, ungual fibromas, and shagreen patches) are rarely present at this age, and a family history of tuberous sclerosis is only occasionally obtained. Much emphasis has thus been placed on finding depigmented patches of skin or hair. These lesions occur in 90% of cases eventually but are present in only 60% at presentation with tuberous sclerosis. Furthermore, the presence of a single white patch is difficult to interpret, as an examination of normal newborns' skin has shown that 0.8% have hypopigmented naevi 1–2 cm in diameter, which may subsequently disappear.⁴

Attention has only recently been drawn to the diagnostic importance of the forehead plaque. Histologically, these lesions are similar to facial angiofibromas, except that they have a less prominent vascular component.⁵ The two cases presented

here illustrate the importance of the forehead plaque as an early sign of tuberous sclerosis. Its recognition in case 1 would have led to the diagnosis being made at the time of presentation. In his case other signs appeared much later and even his computed tomogram in isolation was not diagnostic, having only a single non-calcified paraventricular lesion. The plaque was initially misdiagnosed as a strawberry naevus, which should appear shortly after birth. In case 2 the plaque remains the clearest sign, though the diagnosis was established early by computed tomography.

We thank Dr D C L Savage for permission to report one of his patients.

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Mercury as a health hazard

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SUMMARY Pink disease has virtually disappeared since teething powders were withdrawn.¹ We describe a case in a boy who was exposed to metallic mercury vapour. We discuss the potential health hazard of spilled elemental mercury in the house and the difficulties of removing it from the environment.

Case report

The patient was the only child of healthy unrelated parents. There were no perinatal problems and his early developmental milestones were normal. His general health was good until the family moved house when he was 18 months old. One month later he became irritable and anorexic. He developed a cough and began dribbling saliva. His hands and feet

became swollen. He could no longer walk and had difficulty sitting upright. Over the next few months he gradually lost his language and social skills and became apathetic and withdrawn.

On examination his hands and feet were swollen, with bright pink peeling skin. He was sweating excessively yet was afebrile. The optic fundi were normal, as were his cranial nerves. There was pronounced proximal muscle weakness and hypotonia of both upper and lower limbs. He was unable to sit unsupported. The peripheral muscle strength was normal. All tendon reflexes were present but reduced. His blood pressure was normal at 80/50 and the remainder of the systemic examination was unremarkable.

The clinical diagnosis of pink disease was supported by a urinary mercury concentration of 350 nmol/l (normal <100 nmol/l). Neither his father nor his mother were symptomatic, their urinary concentrations being 26 and 72 nmol/l, respectively.

A detailed history of treatment with drugs excluded the possible ingestion of any mercury containing compound, but the parents learned that the previous occupants of the house had used mercury either to silver telescopic mirrors or to guild replica coinage. This family had left the area and their state of health was not known.

A search of the child's home for environmental mercury was conducted with a Data Acquisition DA 1500 portable mains operated mercury vapour detector. This instrument has a threshold of detection of roughly 1 µg/m³ of air. Airborne concentrations downstairs were low (<3 µg/m³) and no source could be found. Upstairs appreciable concentrations were found in the baby's bedroom (10–12 µg/m³), with much higher concentrations near the vacuum cleaner and near floor level (up to 300 µg/m³). Lifting the loosely laid carpet revealed droplets of mercury on the linoleum underneath, and the carpet back was impregnated.

Recommendations were given concerning decontamination procedure. The local authority engaged a firm of house preservation specialists who dusted the floor with flowers of sulphur and unsuccessfully attempted to remove the sulphur and mercury with the household vacuum cleaner. This procedure was repeated using an industrial vacuum cleaner that was neither fitted with high efficiency exhaust filtration nor vented outside the house. These actions disseminated mercury throughout the house (up to 1000 µg/m³).

Instruction was then given to the firm to use washes of slaked lime and flowers of sulphur rather than dry sulphur and to use detergent to recover the now widely distributed sulphur dust, as it was shown by lifting the floorboards that the mercury had

penetrated to the joists and supporting brick work. After the adoption of satisfactory and thorough procedures mercury concentrations did not exceed 1 µg/m³ throughout the house and the family re-occupied it.

After admission to hospital the baby showed signs of slight improvement and in view of this penicillamine, a potentially toxic drug, was not prescribed. Over the next three months the child made a gradual but complete recovery. The last sign to disappear was the hypotonia.

Discussion

Metallic mercury is volatile at room temperature. Concentrations of vapour up to 3000 µg/m³ can arise from spilled elemental mercury at room temperature, depending on the conditions of ventilation. The current standard for occupational (inhalation) exposure to mercury vapour is given by the Health and Safety Executive as a recommended occupational exposure limit of 50 µg/m³ (eight hours time weighted average value) applicable to long term exposure.² This is a standard for adults at work and must not be used as a 'safe concentration' for domestic exposure nor for children.

The developing infant was exposed to a higher concentration of mercury while crawling on the floor (300 µg/m³) than occurred in the room environment at a height of 1.5 metres (10–12 µg/m³). An infant is also liable to retain higher percentages of absorbed mercury in the brain than an adult. A number of cases of excessive absorption of mercury have been reported ensuing from the introduction of metallic mercury in an uncontrolled manner in the home.^{3–6} We suggest mercury may be found where:

(1) The premises were formerly used by a business, such as dental surgery, laboratory, instrument or jewellery manufacture or repair, farming, or police.

(2) Domestic equipment—for example, a barometer—has been broken or children or hobbyists have used mercury.

This case shows the importance of spilled elemental mercury as a potential health hazard in the home and underlines the need for specialist professional guidance for organisations undertaking the removal. The authors (RLK, AHS) will be pleased to give advice and the British Occupational Hygiene Society (1 St Andrews Place, Regents Park, London NW1 4LB) will refer an enquirer to an appropriate regional authority.

We thank Professor O P Gray for his help and allowing us to report his patient.

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Transient congenital hypothyroidism after topical iodine in pregnancy and lactation

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SUMMARY A 6 week old girl with transient congenital hypothyroidism is described. The hypothyroidism was associated with multiple applications of povidone iodine during pregnancy and lactation. This case illustrates the potential hazard of using topical solutions containing iodine during pregnancy and lactation.

Congenital goiter and, rarely, transient hypothyroidism have been reported to occur sporadically in infants whose mothers received iodides during pregnancy for a variety of non-thyroid diseases or hyperthyroidism.

In these cases iodides had generally been taken orally by the pregnant mother.^{1,2} A few cases of congenital transient hypothyroidism secondary to maternal exposure to procedures using contrast media that contain iodine have been described.³

We report a case of a prolonged but transient hypothyroidism without goiter in a girl whose mother used povidone iodine preparations during pregnancy and lactation.

Case report

A 6 week old Jewish girl of Ashkenazi origin, the youngest of five healthy siblings, was referred to our day care unit because of a low serum total thyroxine concentration of 3 µg/dl (normal value >7 µg/dl) revealed by routine screening for congenital hypothyroidism. She was born at term after an uneventful pregnancy and delivery. Her birth weight was 4000 g. During the pregnancy the mother

washed herself almost daily with a solution of povidone iodine, which is a complex of iodine and polyvinylpyrrolidone, which releases free iodine in solution, with 1% available iodine. She had also rubbed large areas of her skin with povidone iodine ointment (1%) because of spreading furunculosis. These procedures were continued during lactation until referral.

The mother's aunt and uncle suffered from a thyroid disorder, the nature of which could not be elucidated.

Physical examination revealed an infant in good condition with mild jaundice. Her weight was 5.1 kg, length 54.5 cm, and head circumference 40 cm. Body temperature was normal. No goiter was palpable. The face was round with a protruding tongue. Respiration was noisy but there were no signs of respiratory distress. The abdomen was large and the liver was palpated 3 cm below the costal margin. A large umbilical hernia was present. Neurological examination yielded normal results, except for moderately delayed deep tendon reflexes. Laboratory tests revealed a serum concentration of free thyroxine 0.4 ng/dl (normal values 0.7–2 ng/dl). Thyroid stimulating hormone (TSH) was 99 µU/ml (normal value 0.8–5 µU/ml). Indirect bilirubin was 5.6 mg% (95 µmol/l). Thyroid antibodies were negative in the child and her mother. A thyroid scan yielded normal results.

A diagnosis of primary hypothyroidism was made on the basis of raised TSH and decreased thyroxine concentrations. Treatment with L-thyroxine sodium was started. On follow up, there was an increase in thyroxine and a decrease in TSH concentrations (Table).