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An 11-month-old Pakistani girl was admitted to our hospital in February, 1996, because of drowsiness, malaise, and anorexia. She had been well until 6 weeks before admission, when she lost her appetite, stopped crawling or standing up, dribbled from her mouth, sweated profusely, and scratched her skin continuously. On examination, we saw an irritable, sick, clammy girl in opisthotonus with a generalised pruritic rash and swollen, red, cold hands and feet with desquamation (figure). She had persistent tachycardia (180 bpm), and hypertension (130/90 mm Hg), but normal body temperature (37°C). Cytological and biochemical analysis of lumbar cerebrospinal fluid was normal. There was no indication of an infectious disease. Her symptoms fitted with the diagnosis of phaeochromocytoma, which was supported by increased concentrations of plasma and urinary catecholamines: plasma noradrenaline was 7·52 nmol/L, and urine noradrenaline 358 nmol/mmol creatinine. Plasma adrenaline was 2·81 nmol/L and urine adrenaline 108 nmol/mmol creatinine. However, abdominal ultrasound and chest radiography showed no tumour. Iodine-131-labelled metaiodobenzylguanidine scan showed normal adrenal glands and no evidence of extra-adrenal phaeochromocytoma.

At that time, her 6-year-old sister came to hospital with similar, though less pronounced, symptoms. An environmental cause of the symptoms was suspected, particularly acrodynia (“pink disease”). Acrodynia is seen in children after exposure to mercury.1 When asked, her mother reported that 2 weeks before the younger child’s symptoms started, mercury from a broken thermometer dropped on the carpet in the children’s room and had not been retrievable. No other source of mercury poisoning (from cosmetics or medicines) could be identified. Mercury concentration in a urine sample taken on admission, measured with atomic absorption spectrometry, was 12·6 g/L, slightly above the accepted normal value of less than 10 g/L. Exposure was confirmed by the mercury concentration of 1·2 μg/g in her hair (<0·25 μg/g in unexposed controls). After 12 days in hospital, urine mercury dropped spontaneously to 4·1 μg/L. Despite these seemingly low concentrations, we decided to treat the patient with 2,3-dimercaptosuccinic acid, 10 mg/kg three times daily. After 1 day of treatment, urinary mercury increased six-fold (24·6 μg/L). After 3 months of treatment the symptoms had disappeared totally and urinary mercury had fallen below the detection limit of 1 μg/L. The children were last seen in September, 1996, both in good health.

Despite the extent of information on mercury poisoning, the clinical similarities between acrodynia and phaeochromocytoma and an increase in adrenaline and noradrenaline concentrations have been reported only sporadically.2,3 Acrodynia has become a rare symptom complex, which can be recognised easily in the fully developed form. Characteristic symptoms are pink hands and feet (hence “pink disease”), desquamation, scarlet cheeks and nosetip, alopecia, salivation, loss of several or all of the teeth, occasional loss of nails or phalanges, excessive perspiration, transient rashes, pronounced hypotonia, itching, burning and severe pain of the extremities, increased pulse rate and blood pressure, photophobia, insomnia, and apathy alternating with extreme irritability.1 This case shows that apparently low exposure to mercury may lead to overt mercury poisoning through inhalation of mercury vapour.1,3,4 Urinary excretion of mercury can be “normal”, even in overt acrodynia.3 The normal value still generally accepted (<10 μg/L) is based on the detection limit of obsolete methods. Our findings indicate that this value may have to be altered to as low as 1 μg/L in children.

References