MERCURY POISONING IN CHILDREN
A Report of Two Patients

by

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THE SUGGESTION IN 1948 that mercury poisoning was the principal cause of pink disease (acrodynia) in children (Warkany and Hubbard, 1948; Fanconi and Botezn, 1948) resulted in the withdrawal from sale of all mercury containing teething powders in the United Kingdom. There followed a dramatic decline in the reported frequency of the disease (Dathan and Harvey, 1965) and during the past twenty years very few case reports have been published. This present report of two children with mercury poisoning, one of whom presented with a classical picture of pink disease, emphasises the risk of mercury absorption through the skin and highlights the dangers of folk lore medicine as a source of mercury poisoning.

Case History

Case 1. N.Y., a male child aged 20 months, was admitted to the Royal Belfast Hospital for Sick Children in November 1973 with a five week history of extreme irritability, anorexia and refusal to walk, although he had started to walk at one year. His parents also noticed that his hands and feet had become very red and that he sweated profusely. He had not appeared feverish and had not vomited. In his past medical history the child had had infantile eczema since he was three months old which had been treated intermittently with betamethasone valerate.

On clinical examination the child appeared ill and miserable. There was marked photophobia. The palms of his hands and the soles of his feet were red, mildly swollen, cold and moist. He was generally hypotonic and tendon reflexes were reduced. His cranial nerves and sensory system appeared to be intact. Examination of his cardiovascular, respiratory and alimentary systems revealed no abnormality. His skin was free from eczema.

Because the history and clinical findings suggested pink disease, a detailed environmental history was obtained to elicit the possible source of mercury. The family lived in Antrim and the father worked in a nearby factory with analine dyes but had no contact with mercury. Although they lived near Greenmount Agricultural College, there had been no spraying with mercury-containing compounds.

Two months before the child's admission his mother had sought advice from a person in Aghagallon, Co. Armagh, who was reputed to have a "cure" for eczema. This consisted of a belt of damp lint (cost 50p) which was wrapped round the child's waist and left on for one month. The belt had been removed about six weeks before his hospital admission and one week before the onset of symptoms.

The mother obtained a second belt at our request; when opened up it contained metallic mercury in a dispersing agent which could not be identified.

On investigation, urinalysis showed no proteinuria or haematuria. His haemoglobin was 13.9 g/100 ml, white cell count 8,800/c.cmm, 50% polymorphs, 1% eosinophils, 43% lymphocytes and 6% monocytes. Blood urea was 21 mg/100 ml.

	Mercury Estimations μg/100 ml			
	'Normal'	*At time of diagnosis*	*2 weeks*	*4 weeks*
Serum mercury	0.1	7.5	4.1	—
Urinary mercury excretion	10	50	50	8

No specific therapy was given initially but when the raised serum level of mercury was reported it was decided to treat the child with chelating agents. Therapy with oral D-penicillamine 250 mg daily was commenced. This was discontinued after one week because of frequent vomiting. No further chelating agents were given. The child's symptoms subsided gradually and he was discharged home after three weeks in hospital. He has remained well throughout a twelve month follow-up, except for his eczema which has continued to recur from time to time.

Case 2. G.F., a boy of 10 months, was transferred to the Royal Belfast Hospital for Sick Children in November 1972 from a peripheral hospital where he had been admitted two days previously with vomiting, pyrexia and marked irritability of four days duration.

The patient had previously been well except for severe infantile eczema which had been present from one month of age. This had been difficult to control and the child's mother volunteered the information that she had taken him to a person in Hilltown, Co. Down, who was reputed to have a "cure" for eczema. She was given a thick yellow ointment which had been liberally applied to the child's skin for five days prior to his original hospital admission. On subsequent analysis by the forensic laboratory this ointment was found to contain 2 per cent metallic mercury. He had not been immunized and smallpox vaccination had not been given. Both father and grandfather were atopic subjects but there was no other relevant family history. The family lived in Newry and father was unemployed.

On clinical examination he appeared pale and miserable with mild photophobia. His temperature was 39.5°C and he had a tachycardia of 160/min. He had generalised lymphadenopathy and splenomegaly. His eczematous skin was grossly infected and encrusted with many vesicles at the periphery of the lesions. His haemoglobin was 7.7 g/100 ml. White cell count 14,900/cu.mm, 43% polymorphs, 51% lymphocytes, 4% eosinophils, 8% monocytes. Platelets 276,000/cu.mm ESR 18 mm fall in first hour. Blood urea 35mg/100 ml Na=142 mEq/1, K=5.9 bEq/1, Cl=107 mEq/1. Total plasma proteins were 5.5 g/100 ml, albumin 2.3 g/100 ml, globulin 3.2 g/100 ml, α_1 globulin 0.6 g/100 ml, α_2 globulin 1.4 g/100 ml, β globulin 1.0 g/100 ml, θ globulin 0.2 g/100 ml.

	Immunoglobulins (results in mg per 100 ml)			
	On admission	*1 week later*	*2 weeks later*	*4 weeks later*
IgG	320	350	570	700
IgA	50	0	70	90
IgM	25	48	50	56

Multiple bacterial swabs from his skin and repeated blood cultures were sterile. Scrapings from the skin vesicles were examined under the electron microscope and found to contain particles of herpes simplex virus. The serum mercury level was 21 μg/100 ml. The urine contained 100 μg mercury/100 ml.

The child was treated with parenteral ampicillin and cloxacillin and a blood transfusion. He required sedation with diazepam and methdilazme (Dilosyn). His skin was treated locally with framycetin ointment and 0.5 per cent idoxuridine in a simple base. Chelating agents were not given.

His illness pursued a turbulent course with a spiking fever and marked irritability for seven days. Thereafter the fever subsided and the child improved. After one month's intensive local treatment his skin returned to normal. He was discharged after six weeks and has remained well at follow-up over eighteen months.

Discussion

Although the symptoms and signs of the condition had been recognised since the early years of this century, it was Swift (1914) writing from Australia who first gave a comprehensive description of pink disease in children. In 1948 Warkany and Hubbard in Cincinnati and Fanconi and Botezn in Switzerland drew attention to the close relationship of this disorder with elevated levels of mercury in the serum and urine of affected patients. Though now generally accepted that pink disease is a manifestation of mercury intoxication, the pathophysiology is still not fully understood. Many features are attributable to overaction of the sympathetic and parasympathetic nervous system, for example sweating, erythroedema, vomiting, tachycardia, photophobia and excessive salivation. Other symptoms, such as hypotonia, irritability and paresthesia, and in the long term, mental retardation, may be the result of heavy metal poisoning of enzyme systems concerned with ammonia, glutamine and pyruvate metabolism (Farber and Vawter, 1966). It is also known, however, that people can tolerate excessive quantities of mercury without evidence of intoxication, for example psoriatic patients (Inman, 1956) and it has been postulated that those who show clinical evidence of mercury intoxication may be demonstrating an idiosyncracy to the metal.

The first child illustrates the classical picture of pink disease with abject misery, marked hypotonia, photophobia, excessive sweating and erythroedema of the hands and feet. The symptomatology of the second child was more likely to be due to his systemic herpes infection (Kaposi's varicelliform eruption) than to mercury poisoning. The severity of this infection is shown by the depression of his immune system

with secondary hypogammaglobulinaemia which gradually returned to normal as he improved. However some of the findings of vomiting, fever, irritability and tachycardia are common to both conditions and the levels of serum and urinary mercury were certainly elevated.

Traditionally, the treatment of mercury poisoning has been with the chelating agents British anti-lewsite (BAL), edathamil calcium disodium (EDTA) or D-penicillamine. However, the efficacy of these drugs in pink disease has never been conclusively proven. In a controlled trial in Australia in which infants with pink disease were treated with edathamil calcium disodium no significant clinical improvement was recorded and there was no concomitant increase in mercury excretion (McCoy, Carré and Freeman, 1960). In the one patient here described who was treated with D-penicillamine recovery was almost certainly unrelated to therapy. Ganglion blocking drugs have also been used to control the autonomic disturbances. Bower (1954) found in a group of nine children that although symptoms caused by over-action of the autonomic system were ameliorated, the effect was of short duration as the children rapidly became tolerant of the drug.

During the past twenty years the few reported cases of pink disease have resulted from exposure to mercury-containing dusting powders or ointments (e.g. McGregor, 1964). Both children here described had metallic mercury applied to eczematous skin. While the eczema may have contributed to excessive absorption, it is known that absorption through the skin is related more to the type of mercury and the base in which it is carried than to the integrity of the skin surface (Wied, 1964). Very few skin preparations now used in paediatrics contain mercury and those which do, contain very low concentrations.

These two children illustrate again the potential danger of mercury absorption through the skin. While most doctors recognise this danger and no longer prescribe mercury-containing ointments for children, unfortunately the same cannot be said of unqualified people reputed to have the "cure" for eczema.

Summary

Two children aged 20 months and 10 months with mercury poisoning are described. One child wore a belt containing metallic mercury and the other child had two per cent mercury ointment applied liberally all over his body. Toxic effects can result from absorption of the heavy metal through the skin. Attention is drawn to the fact that mercury poisoning in children can still occur since preparations with a high mercury content are available from non-medical sources. In each of these patients the parents had sought a lay cure for eczema.

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BOOK REVIEWS

VENEREAL DISEASES. By Ambrose King and Claude Nicol. Third Edition. (Pp. 384, figures 180. £7.50). London: Baillière Tindall. 1975.

THIS attractively presented book which was written for both undergraduate and postgraduate students, is also suitable as a reference book for the general physician. It can be strongly recommended for anyone interested in the study of sexually transmitted infections.

There is not sufficient difference between the second and third editions to justify a further outlay for anyone who already possesses the second edition.

T.H.

CLINICS IN HAEMATOLOGY. Volume 4. No. 1. February 1975. Haemolytic Anaemias. Edited by T. A. J. Prankerd and A. J. Bellingham. (Pp. 260. Figs 36. 3 times yearly—£12.00). London and Philadelphia: W. B. Saunders. 1975.

THIS is the latest of a very well known and respected series which presents up-to-date accounts of selected subjects in haematology and the topic of this issue is the haemolytic anaemias. A varied and cosmopolitan flavour is given to the book as the contributors of its fourteen chapters represent seven different countries. Nevertheless, the presentation of the subject is orderly. The initial chapters deal with advances in knowledge of the physiochemical structure of the red cell, its metabolism and a discussion of red cell antigens and their interaction with antibodies. Following this review of basic matters the clinical states resulting from red cell enzyme deficiencies, hereditary spherocytosis, immune and drug-induced haemolytic anaemias are discussed. There are also chapters on paroxysmal nocturnal haemoglobinuria and an especially interesting one on the role of the spleen in haemolytic disorders.

This book is for the specialist in haematology who will find it a valuable review of recently gained knowledge on haemolytic anaemias.

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