

Lead Poisoning in Childhood in Ceylon

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Lead poisoning in childhood has been reported mainly from Japan, France, the United States, Great Britain, and Australia. Attention is drawn in this paper to the incidence of lead intoxication in childhood in Ceylon, seen and diagnosed within the past three years.

Whereas in past reports the principal mode of lead intoxication in childhood has been from ingestion, in the present series inhalation of fumes, and absorption by way of skin, have been important.

Material and Methods

Our series is comprised of 12 cases investigated in one unit of Lady Ridgeway Children's Hospital, Colombo, and detailed in the Table. Six of the children affected were from families of workers employed in a small-scale jewellery industry, located within the same premises occupied by the families, and inhalation of fumes was probably the route of intoxication. The remaining 6 cases arose from sources similar to those described by previous authors, or were not identified.

Lead in blood and urine. Samples collected in lead-free containers were wet-ashed with concentrated nitric acid and estimated by colorimetric dithizone extraction. A modification of the Woessner and Cholak (1953) method using extraction of the dithizone complex at high pH 11 was used (Snyder, 1947). Parallel blank estimations were run with each sample to correct for reagent contamination, or contamination from manipulative operations.

Coproporphyrin in urine. A semiquantitative method of coproporphyrin estimation was used (Waldman and Seidman, 1950). 10 ml. urine was treated with 1 ml. glacial acetic acid and three drops of hydrogen peroxide, shaken with 2 ml. ether, stood in the dark for 30 minutes, and viewed under ultraviolet light. The ether layer fluoresces from violet to dark red for a positive reaction, and blue to blue-green for a negative reaction. The positive results are visually graded as \pm (violet) to $++++$ (dark red), with $++$ on this scale corresponding to 0-50 $\mu\text{g./litre}$, as normal baseline for coproporphyrin excretion, and $++++$ corresponding to 800-1600 $\mu\text{g./litre}$.

Stippled cell count. Thin blood films, fixed with methyl alcohol, air-dried at room temperature, and stained with alkaline methylene blue, were washed to an apple-green colour, and examined under dark-ground illumination. Stippled cell count on 100 fields of the film was expressed as a count per million erythrocytes.

Results

Findings in the 12 cases are summarized in the Table. Brief case records typifying the source of lead in 5 cases follow.

Case Reports

Case 2. A 6-year-old boy presented with a one-day history of repeated convulsions. He had been well until two days before, when he had passed a few loose stools containing roundworms.

The child's clothes and exposed parts of the body were covered with a film of greyish dust. His rectum was loaded with roundworms. Urine examination revealed moderate glycosuria; blood urea was 45 mg./100 ml. and uric acid 2.4 mg./100 ml. CSF was under raised pressure: protein 80 mg. and sugar 95 mg./100 ml., with no excess of cells. X-ray films of long bones showed dense metaphyseal lead lines.

Hb 8 g./100 ml.; and there was obvious stippling of red cells. Lead content in the first 24-hour collection of urine was 4.3 mg./litre. Blood lead on the 4th day after admission was 500 $\mu\text{g./100 g. blood}$. Coproporphyrin III in urine was also much increased.

He was treated with anticonvulsants and intravenous drips of calcium EDTA, 1 g. daily for five days. Fits were brought under complete control only 3 days after admission, and he became fully conscious only four days later. A second course of EDTA was given later.

Case 1. The sister of Case 2, this 3-year-old girl also presented with generalized convulsions and died within 24 hours of admission. CSF was under pressure, with 11 lymphocytes, 1 neutrophil/c.mm.; protein 60 mg., and sugar 195 mg./100 ml. Blood urea 55 mg./100 ml. The red cells were not noticed to show basophilic stippling. X-ray films of long bones taken after death showed dense metaphyseal lead lines. Partial necropsy showed congestion and oedema of the brain. In retrospect, death was attributed to lead encephalopathy.

TABLE
Details of 12 Cases

Case No.	Sex	Age (yr.)	Suspected Source of Lead	Presenting Symptoms and History
1	F	3	Refining jeweller's wastes	Generalized convulsions
2	M	6	Refining jeweller's wastes	Generalized convulsions, vomiting a week before admission; unconscious at time of admission
3	F	9	Refining jeweller's wastes	Anaemia
4	M	7/12	Refining jeweller's wastes	Admitted after vomiting for 3 wk.; anaemic
5	M	3	Refining jeweller's wastes	Admitted on 17.12.64 with history of vomiting—subsequent admission on 26.12.64 for generalized convulsions; fits in ward
6	M	5	Refining jeweller's wastes	Generalized convulsions for a week before admission
7	F	11/12	Reconditioning of batteries—recovery of scrap lead	Generalized convulsions preceded by gastrointestinal disturbances over past 3-4 mth.
8	F	1½	Reconditioning of batteries—recovery of scrap lead	Generalized convulsions; 'fits' from age of 9 mth.
9	M	5	Type foundry	Recurrent abdominal pain, pica, and anaemia
10	M	5	Not traced	Vomiting and refusal of foods for 2 wk. before admission; generalized convulsions on admission; limbs spastic; papilloedema; raised intracranial pressure
11	M	2½	Not traced	Febrile convulsions, past history of anaemia
12	M	1	Not traced	Vomiting and generalized convulsions 3 days before admission

* Code index to lead in urine figures: B—Before treatment with chelating agent; D—during treatment; E—end of course.

Case 7. An 11-month-old girl, the sole surviving sib of three children whose parents lived in a slum area in the city of Colombo, presented with a one-day history of generalized convulsions. Two other children of these parents had died at the age of 15 months and 18 months, respectively, also of generalized convulsions. The skiagrams of long bones showed dense metaphyseal lead lines, and the diagnosis of lead poisoning was confirmed by the other laboratory investigations (see Table). The baby was treated with a course of EDTA and discharged a month after admission. The parents took her back to live in the same surroundings.

She was readmitted two months later, with vomiting and generalized convulsions. Anaemia was now more severe. She was kept in hospital for nearly 3 months, mainly in order to separate her from the home environment, suspected to be the source of poisoning. She was treated for anaemia, but not with chelating agents on this occasion, and eventually returned to the same home surroundings.

She was readmitted for a third time with fever and convulsions six weeks later. Once again she had stippled cells, glycosuria, and CSF which was under raised tension, with protein 80 mg./100 ml. and 2 neutrophils and 30 lymphocytes/c.mm. She was given a second course of EDTA, and after 2 months was discharged to different lodgings. She has not been readmitted subsequently.

This family lived in a house opening on to a yard, used as a workplace for the reconditioning of car batteries, the father being employed on this work. The house was

subject to heavy contamination by fumes blowing in from the yard.

Case 6. A 3-year-old mentally retarded boy was admitted with a history of generalized convulsions which had occurred intermittently for a week. He had been constipated and had refused food for the month preceding. He had had previous admissions for generalized convulsions with mental subnormality.

He was febrile, had blue lines on upper and lower gum margins, and his limbs were spastic. X-ray films of long bones showed dense lead lines. Hb was 6 g./100 ml., the red cells were stippled. CSF was clear and under raised pressure, with 3 polymorphs and 1 lymphocyte per c.mm., protein 180 mg., and sugar 45 mg./100 ml.

He continued to have repeated convulsions and was treated with anticonvulsants and penicillamine, 200 mg. t.d.s. Clinical improvement was poor, and combined therapy with EDTA and dimercaprol (4 mg./kg. 4-hourly) was instituted.

At the time of discharge from hospital his appetite had improved considerably, Hb had risen to 11 g./100 ml., and he was more alert. He returned to live in the same surroundings, in one room of a slum with his grandmother. The adjoining room was used as a smithy by night. The fumes from this workshop blew freely into the room occupied by the boy.

Case 9. A 5-year-old boy had been seen on many occasions as an out-patient for recurrent attacks of colicky abdominal pain unaccompanied by vomiting.

of Lead Poisoning

Lead in Urine* (mg./l.)	Lead in Blood (mg./100 ml.)	Coproporphyrin III in Urine	Stippled Cell Count /10 ⁶ Erythrocytes	Other Findings and Notes†
—	—	—	—	Died before treatment; sister of Case 2, admitted 2 weeks before Case 2 Hb 5·6 g./100 ml.
4·32 —B	—	+++	20,006	
—B	—	+	—	
0·94 —D	0·500	+++	18,400	
0·040 —E	—	—	8000	
0·125 —B	0·105	+++	1539	Sister of Cases 1 and 2 Hb 7·4 g./100 ml.
0·395 —D	—	—	—	
—	—	—	—	Hb 6·9 g./100 ml.
—	—	+++	2250	Died before treatment instituted; brother of Case 4
—	—	—	—	
—	—	—	1162	Hb 6 g./100 ml.; 4 children in neighbourhood said to have died of convulsions within last year
0·180 —B	—	—	—	Sibs aged 1½ yr. and 1½ yr. had died with similar symptoms 4 and 2 yr. before
0·868 —D	—	+++	—	
0·187 —E	—	—	—	
1·6 —B	0·149	++	35,517	
9·75 —D	0·250	+++	—	
0·56 —E	—	—	—	
—	—	—	Stippled cells, positive	
0·290	0·121	—	—	Died 5 dy. after admission; sib of 3½ yr., died of convulsions 2 mth. earlier
—	—	—	—	Died
—	—	—	—	

† All 12 cases showed lead-lines in skiagrams of long bones.

As a 6-month-old baby he had three febrile episodes during which some excess of pus cells in the urine, but no organisms, were found; blood urea was normal; Mantoux test negative. X-ray films of long bones showed moderately dense metaphyseal lines suggestive of lead intoxication. Hb 7·5 g./100 ml.; blood showed a few stippled cells. No blood or urinary lead estimations have been possible in order to confirm the presumptive diagnosis of lead poisoning.

The parents live in premises where a lead type foundry is located in close proximity to the home, and the child is allowed to stray into the foundry.

Environmental Factors

In 6 of the cases, typified by Case 1 above, the source of lead was traced to a gold recovery process used in jewellery manufacture. In Ceylon, the jewellery industry is a small-scale industry, practised as a skilled craft among certain communities. The subsidiary operation of recovery of gold and silver from jeweller's wastes which accumulate from polishing, lapping, and drilling, is also a small-scale industry where methods are primitive, and which is also confined to certain ethnic groups. These communities are scattered throughout the crowded slum areas of the city where they live in tenement or shanty dwellings, so that any occupational health hazard associated with this industry is enhanced by the crowded and insanitary conditions.

In the cases concerned, the process of gold recovery was carried out by a community of about 100-200 people in a small shanty town situated on a river bank in the city outskirts. The settlement consisted of wattle and daub houses, surrounded by yards which were used for gold recovery operations. Children played about freely in the yards and sheds housing the primitive furnaces in which the fusing operations were performed. The wastes are collected and handpicked to remove large scraps of paper, rags, etc., and subjected to a wet-sieving similar to the primitive 'panning' process. The heavy residues from the 'panning' are collected and subjected to a process similar to the classical 'cupellation' process in which they are ground and mixed with smelted lead, and fused in a shallow bricked-up hearth. The crude hand-operated blowers used to blow a current of air over the molten mass create and disperse clouds of smoke laden with lead fumes and lead oxide dust in a finely dispersed state. This also occurs in the subsequent operation where the cooled fused mass containing the oxides is reduced with charcoal. The children not only played about in the yards and sheds, but also assisted in these operations. There must also be considerable accumulation of lead-oxide on the mud floors of the sheds.

The second source of lead poisoning in these cases

was a battery repair and reconditioning establishment. Operations involving the release of lead into the atmosphere included the breaking of battery plates (lead-oxide), and the smelting of the scrap thus recovered in an open melting pot.

The third source of lead poisoning was traced to a workshop reported to be a smithy, where lead was used as one of the work materials, and where a small bricked-up hearth was used indoors for fusing operations.

The fourth source was a lead-type foundry located among dwelling houses.

Discussion

Clinical diagnosis of lead poisoning. As has been often stressed, it is awareness of the possibility of lead intoxication that leads to diagnosis.

Febrile convulsions may be a presenting sign, since encephalopathy can be precipitated in a quiescent case by release of lead from the skeleton during acute infections and metabolic disturbances.

Anaemia was found in 6 of our cases; anaemia is a very common symptom of children seeking admission to hospital in the tropics, and if hypochromic and microcytic, it is put down to iron deficiency or to helminthiasis, if stool examinations also reveal the presence of hookworm or whipworm ova.

The difficulties of making a clinical diagnosis of lead poisoning in children in the tropics are therefore great. The clinician needs to be aware of the

multifarious uses of lead and of the numerous sources from which lead intoxication can occur.

Summary

A series of 12 cases of lead poisoning in children in Colombo is reported. In addition to the sources of lead which are generally recognized, in 6 cases the source was traced to the fact that the children were living in premises where small-scale gold recovery from jeweller's wastes was carried out. Inhalation of lead-containing fumes was a feature in many of the cases.

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