CASE REPORT

Lead Poisoning in Childhood

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Summary

We report here a case of lead poisoning in a 20 month old girl who presented with acute encephalopathy and status epilepticus. The major clues leading to the diagnosis were the occupational family history and dense lead lines on X-ray of the long bones.

She showed evidence of neurological dysfunction in the initial phase, but she improved steadily, regaining her motor power partially and her vision, although some cognitive and language deficits were already evident. She will need long-term neurological assessment and evaluation to ascertain the extent of permanent brain damage.

Key words: Lead poisoning, childhood, prevention.

Introduction

Lead poisoning is a well-known occupational health hazard in adults. In children, lead poisoning is usually associated with ingestion of chips of lead-based paint. It is also well-appreciated that the other major routes of exposure include lead-contained dust and soil.

Lead is found anywhere in our environment, thus our children are exposed to varying degrees of severity. The Centre for Disease Control (CDC) of USA, in 1991, declared that a blood lead level of 10 ug/dl is indicative of lead poisoning[1]. Recent studies have shown that lead levels lower than 25 ug/dl can be detrimental to cognitive development[2], producing lasting adverse effects upon brain function[3].

Therefore, every effort should be undertaken to prevent this preventable disease of childhood. In Malaysia, lead poisoning in childhood has rarely been reported, especially in relation to occupational health hazards. The actual reasons behind this could be under-diagnosis or under-reporting. It is with this in mind that this case report is made.

Case History

AH was a 20 month old girl who was admitted in September 1992 with a 3 day history of fever, followed by one episode of generalised tonic-clonic convulsions lasting about 30 mins. Her developmental milestones had previously been normal. She was the only child and had an uneventful birth history. Her father was a general worker in the manufacturing area and also worked part-time as a security guard. The mother was the factory canteen operator. The family had been living in the staff quarters within the factory compound for the last 4 months. There was no history of pica. There was no family history of mental retardation, epilepsy or porphyria.

On examination, she was deeply comatose with feeble flexion of the upper limbs to deep pain. There was no neck stiffness. The pupils were equal and reactive and there was gross papilloedema in both fundi. She was hypotonic, hyporeflexic in the upper limbs and areflexic in the lower limbs. Plantar responses were not elicited. Other systems were essentially normal.

An initial diagnosis of meningitis/encephalitis was made. A lumbar puncture was not done on admission, because she was very ill. There was cerebral oedema on the urgent CT scan done. The blood film showed normocytic normochromic anaemia with a haemoglobin of 8 gm/dl. The renal biochemistry and liver function tests were normal. Serum phosphate was slightly reduced (2.1 umol/L). X-rays of the knees, wrists and humerus showed dense lines in the metaphyses. The abdominal X-ray did not show any radio-opaque flecks.

With the initial provisional diagnosis of meningitis, she was started on intravenous antibiotics. She developed status epilepticus and required several intravenous anticonvulsants. Cerebral resuscitation was instituted on the day of admission. Mannitol and dexamethasone were also used to reduce the cerebral oedema.

A diagnosis of lead encephalopathy was made after review of the X-rays of the long bones. Intramuscular chelation therapy with dimercaprol was thus started with this provisional diagnosis and was given for 10 days. At the same time, an urgent blood specimen sent to the government laboratory showed a blood lead level of 73 ug/dl. Her urine test for corporphyrin was positive. Subsequently, she was maintained on oral penicillamine.

The following regimen was used:

Acute therapy:		
Days 1 and 2	:	2.5 mg/kg/dose every 4 hours
Day 3	:	2.5 mg/kg/dose every 6 hours
Days 4 to 10	:	2.5 mg/kg/dose daily
Maintenance therapy:		
Oral penicillamine	:	2.5 mg/kg/day

The maintenance therapy was to be maintained for 3 to 6 months, depending on her lead levels.

The following results were her lead levels during treatment:

Day of treatment	Blood level (ug/dl)
Day 1	73
Day 3	49
Day 4	46
Day 5	45
Day 7	40
Day 8	26

She made reasonably fair progress within a week. Her conscious level improved by Day 5. There was gradual return of the deep tendon reflexes by the second week. By the third week, she could sit up and had satisfactory hand-eye coordination skills. However, she was still unable to walk and demonstrated a loss in her speech and language skills.

Her father's and mother's blood lead levels were 61 ug/dl and 38 ug/dl respectively.

The factory was visited by the Occupational Health Department of the Ministry of Health and various measures to prevent recurrence of poisoning were instituted.

Discussion

A diagnosis of lead poisoning/encephalopathy was made here based on the occupational family history and X-ray of the long bones. The high blood lead level then confirmed the clinical impression. If we had easily available laboratory facilities, the diagnosis would have been made earlier.

Childhood lead poisoning is recognised as a significant public health problem in the USA and Australia. Various epidemiological data suggest that about 4% of the children aged 1 to 5 years have increased lead absorption. Young children who live close to lead smelters or battery factories are at high risk of lead poisoning. Workers exposed to lead who wear their dirty workclothes home contaminate their homes and cars, and their children may also show lead toxicity.

The key management in lead poisoning is prevention. Before any effective preventive measures can be undertaken, the source of exposure should be identified. Several measures have been taken in Malaysia, such as the introduction of unleaded petrol, lead-free paint and lead-free plumbing for domestic water supply. However, abatement of lead exposure from contaminated dust and soil has generally been unsuccessful in most countries, including developed ones.

References

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