Does lead poisoning occur in Canadian children?

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Lead poisoning in preschool children due to the ingestion of paint chips peeling from the walls in inner city (slum) housing is well known in the United States.[1,2] It is not perceived to be a problem in Canada, although sound scientific data supporting this position are lacking. Two such cases with profound sequelae are described.

Case reports

Case 1

A 32-month-old girl was brought in for medical assessment because of a pigeon-toed gait. There was a 17-month history of a voracious appetite for the paint peeling from the walls and window sills of her inner city home. She had been born at 26 weeks' gestation, weighing 1270 g. Her neonatal course had been uncomplicated, and her development had been normal at 12 and 14 months.

The toddler was alert and in no distress. Except for her gait the physical and neurologic findings were normal. Anemia was diagnosed (hemoglobin concentration 100 g/L), and iron was prescribed. A blood sample was drawn to determine the lead and free erythrocyte protoporphyrin (FEP) concentrations. An orthopedist felt that the problem was skeletal, not neuromuscular, and recommended the use of corrective footwear.

Two weeks later lead poisoning was diagnosed (blood lead concentration 4.05 μmol/L, FEP concentration 11.8 μmol/L [upper limits of acceptability at that time were 1.45 and 0.9 μmol/L respectively]). The values fulfilled the criteria of the US Centers for Disease Control, Atlanta, for class IV lead poisoning.[3] The girl was admitted to hospital for 5 days of parenteral chelation therapy (dimercaprol, 500 mg/m^2 daily, and calcium disodium edetate, 1500 mg/m^2 daily). At that time a marked deterioration in her affect was noted; this irritability persisted for several days. Iron deficiency was biochemically confirmed. X-ray films demonstrated lead lines in the long bones and numerous radiopaque densities in the abdomen. A neurologist found no specific abnormalities.

After the parenteral therapy oral chelation therapy with penicillamine, 125 mg twice daily, was begun and the girl discharged; in the interim her mother had rented another apartment. After inspecting the original residence city health officials found that samples of chipped plaster and peeling paint contained up to 122 000 ppm of lead (upper limit of acceptability 5000 ppm in Canada and 600 ppm in the United States); a local improvement order was issued.

Oral chelation therapy was continued for 3 months. The pica disappeared after correction of the iron deficiency. A child development consultant found uneven and delayed development at 2 and 14 months after hospital discharge. Because of a deteriorating gait the same neurologist diagnosed upper motor neuron dysfunction at follow-up 1 year later. At age 8 years the girl had to repeat grade 2, and psychologic assessment identified low normal intelligence with specific weaknesses (delays of 2 to 3 years) in areas of abstract reasoning, visual sequencing, visual motor integration and receptive vocabulary.

Case 2

On hearing of case 1 a colleague recalled a similar case; I reviewed the hospital chart. A 26-month-old boy had been brought to the hospital comatose after a generalized seizure. He had been seen five or six times over the previous month because of persistent vomiting for which no cause was found. Anemia (hemoglobin concentration 80 g/L) was treated with iron. Pregnancy, labour, delivery and early development had been normal.

The boy was afebrile, dehydrated and comatose and had papilledema. Lumbar puncture ruled out intracranial infection and revealed increased pressure. X-ray films showed lead lines in the long bones

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and numerous radiopaque densities in the abdomen. Basophilic stippling was found in his blood smear.

Lead encephalopathy was diagnosed, and treatment was started with dimercaprol and calcium disodium edetate at the same doses as in case 1. Lead poisoning was confirmed (urinary concentration of lead 1320 [normally 10 to 60] μg/L and of coproporphyrin 1500 [normally less than 250] μg/L), as was iron deficiency. The boy's level of consciousness began to improve after several days of therapy, and he gradually became alert. The sequelae of the encephalopathy have included moderate mental retardation and a chronic seizure disorder requiring therapy with multiple anticonvulsants.

During the boy's hospital stay a history of pica for peeling paint was discovered. Analysis of paint chips from his home revealed a lead concentration of 25 000 ppm. The city health department issued a local improvement order.

Comments

Lead poisoning was diagnosed in case 1 because of the examining physician's high level of suspicion. Although encephalopathy was not present a distinct deterioration in the child's affect was observed over the 2 weeks before therapy. Her mother had reported an improvement in this during intravenous and early oral chelation therapy; thus, it seemed that encephalopathy was imminent. Because of her prematurity the contribution of lead poisoning to the subsequent motor, perceptual and cognitive defects is uncertain. However, the results of developmental assessments before the onset of lead poisoning were normal. Case 2 is a classic example of lead encephalopathy with disastrous sequelae. No other risk factors were identified for his seizure disorder or mental retardation.

Important risk factors for lead poisoning (peeling paint containing enormous amounts of lead and iron deficiency) were present in both cases. Iron deficiency is believed to be the single most important factor predisposing to increased lead absorption:[2] it leads to pica[4] and increases the gastrointestinal absorption of lead.[2]

Lead poisoning due to chronic paint ingestion by preschool children has been well described in the United States[1,2] and Australia.[5] The cases presented here are worthy of attention because lead poisoning is not perceived to be a problem in Canada. However, there have been no blood lead concentration surveys among children in inner city areas to validate this perception. City health officials, after screening children living close to lead polluting industries, found that the mean blood lead concentrations were within normal limits, and there were no examples of gross elevation (Dr. Colin D'Cunha, Toronto, and Dr. Douglas Luckhurst, Winnipeg: personal communication, 1989). However, the children lived in working class neighbourhoods and not dilapidated inner city slums, which are usually the high-risk areas for pediatric lead poisoning.

A study published in 1976 revealed that 5% to 8% of children in Montreal had blood lead levels over 1.95 μmol/L.[6] However, the focus of the study was to verify a laboratory lead determination technique, and thus there were no accompanying epidemiologic data. A case of a 2½-year-old girl from London, Ont., with lead encephalopathy was reported.[7,8] She had pica for peeling paint that contained lead. Six other children with lead poisoning were subsequently identified from the region. Unfortunately, clinical and epidemiologic data were not provided. However, iron deficiency and pica for peeling paint were common to several of the children (Dr. Roland Carson: personal commmication, 1989).

Lead as the cause of frank encephalopathy could be missed. A controlled study in Michigan identified three such cases through retrospective analysis of stored autopsy material from preschool children who died of unknown neurologic conditions.[9]

The two cases described here, as well as those from London, Ont.,[7,8] show that frank lead poisoning due to chronic paint ingestion by young children has occurred in Canada. There are probably milder, undetected cases. Recent research supports the position that there is no known safe body burden of lead, and amounts previously considered to be innocuous have negatively affected the neurologic development of children.[1] Such children have no symptoms or signs of lead poisoning. The prevalence of neurotoxic lead levels in asymptomatic children is unknown in Canada. Sound epidemiologic studies of blood lead concentrations involving children living in Canadian inner city areas are needed to define the extent of lead poisoning from lead-based paint.

References

1. Needleman HL: The persistent threat of lead: medical and sociological issues. *Curr Probl Pediatr* 1988; 18: 699-744
2. Landrigan PJ, DiLiberti JH, Gehlbach SH et al: Statement on lead poisoning. *Pediatrics* 1987; 79: 457-465
3. US Centers for Disease Control: Preventing lead poisoning in young children. *J Pediatr* 1978; 93: 709-720
4. Crosby WH: Pica: a compulsion caused by iron deficiency. *Br J Haematol* 1976; 34: 341-342
5. Inglis JA, Henderson DA, Emmerson BT: The pathology and pathogenesis of chronic lead nephropathy occurring in Queensland. *J Pathol* 1978; 124: 65-76
6. Mehkeri KA, Romanowski M, Smallbone B: Use of a filter disc micro-sampling atomic absorption method for blood lead level screening. *Am Ind Hyg Assoc J* 1976; 37: 541-545
7. Haust HL, Ali H, Haines DSM et al: Short-term administration of dimercaptopropanol (BAL) and calcium disodium edetate (EDTA) for diagnostic and therapeutic lead mobilization. *Int J Biochem* 1980; 12: 897-904
8. Poon HC, Carson R, Peter F et al: The lead program at CPRI. *Clin Biochem* 1989; 22: 213-219
9. Meester WD, Eisenga B: Death from lead poisoning in children [abstr]. *Vet Hum Toxicol* 1984; 26: 404