

Lead poisoning from “lead-free” paint

A 4-year-old boy had been living with his mother and siblings for 8 months since their move into a home built in the 1950s. Although the boy was well, his mother noticed that he was eating paint stripped from the walls of their new home and reported her concern to their family physician. On examination, the boy had paint chips under his nails, and his blood lead level was very elevated (4.70 $\mu\text{mol/L}$; normally < 0.48 $\mu\text{mol/L}$). He also had microcytic anemia (hemoglobin level 65 g/L, mean corpuscular volume 52 fL) and a low serum iron level (1.8 $\mu\text{mol/L}$).

The boy was referred to a local emergency department for further assessment and treatment. An abdominal radiograph revealed abundant radio-opaque material throughout the large bowel (Fig. 1). The boy was admitted to hospital and underwent whole-bowel irrigation with a polyethylene glycol and electrolyte solution (GoLYTELY, 500 mL/h). Parenteral chelation (with dimercaprol given intramuscularly and calcium disodium edetate given intravenously) was also initiated. Paint chips were seen in the rectal effluent until 36 hours after initiation of the bowel irrigation; a repeat radiograph 41 hours after bowel irrigation was started revealed some residual radio-opaque materials in the right colon (Fig. 2). After a second cycle of parenteral chelation the boy was discharged home with a blood lead level of 1.73 $\mu\text{mol/L}$.

None of the boy's family was found to have elevated blood lead levels. A home visit by local public health officials revealed numerous scrapes on the house walls, mainly in the bathroom where the child apparently hid often to eat paint chips. Although paint scrapings from the house were tested and found to contain lead, the levels were within the legal limits to be considered “lead free” (< 0.5% dry weight). No



other source of lead was found in the home or local environment.

Lead is toxic to the hematologic, renal and nervous systems in a highly dose-dependent and variable fashion. Chronic lead exposure may lead to developmental delay and, when blood levels are very high (e.g., > 4.80 $\mu\text{mol/L}$), acute toxic encephalopathy.^{1,2} However, most patients with elevated lead levels are either asymptomatic or have a variety of nonspecific symptoms such as anorexia, vomiting or abdominal pain. Patients at high risk of toxic effects include children 9 months to 3 years old and patients living near point sources of lead pollution.³

Although lead poisoning is now rare in Canada, exposure from a variety of possible environmental sources must be considered if a patient has an elevated lead level.³ It should be considered in children with pica or those with newly recognized microcytic anemia if at risk for lead poisoning. Pica, or compulsive eating of nonnutritive substances for at least 1 month, is not uncommon in children and must be distinguished from “mouthing” observed in young infants.⁴

The addition of lead in paint was once widespread because of its use as pigment, dispersing agent and drying agent but mostly because it provided durability to the paint.⁵ In 1975

Canada's Hazardous Products Act prohibited the use of paint containing more than 0.5% of lead (dry weight) for indoor use and on furniture and toys but not on playground equipment (municipalities decide whether paint can be used on public playground equipment). Screening for lead toxicity is usually recommended for children living in houses built before 1950.³ However, our case demonstrates that lead toxicity should also be suspected in anyone with pica who eats paint, even if it is “lead free.”

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