Prevalence of Radiographic Evidence of Paint Chip Ingestion Among Children with Moderate to Severe Lead Poisoning, St Louis, Missouri, 1989 Through 1990

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ABSTRACT. Although experts once believed that ingesting chips of lead-based paint was the major cause of lead poisoning among children, conventional wisdom now holds that lead-contaminated dust and soil are the major routes of exposure. Data from a childhood lead-poisoning treatment clinic were examined to assess the frequency with which children ingest paint chips. For this study, the reports on abdominal radiographs of 90 children with moderate to severe lead poisoning who had received their first chelation treatment during 1989 or 1990 were reviewed. According to a radiologist's evaluation, 13 of 90 abdominal radiographs (14%; 95% confidence interval [CI] 7% to 22%) showed evidence of paint chip ingestion. Of 46 children with blood lead levels ≥ 55 μg/dL, 12 (26%) had radiographs that showed paint chips, whereas only 1 (2%) of 44 children with blood lead levels < 55 μg/dL had such radiographs (prevalence ratio = 11.5; 95% CI 1.6 to 84.6). The actual proportion of children with moderate to severe lead poisoning who have consumed leaded-paint chips is likely to be higher than this estimate based on radiographic evidence. While lead-contaminated dust is a major source of lead exposure, ingestion of leaded-paint chips clearly remains an important source of exposure among children with moderate to severe lead poisoning. *Pediatrics* 1992;89:740–742;
lead poisoning, lead paint, radiograph.

ABBREVIATIONS. LPPPC, Lead Poisoning Prevention Program Clinic; BLL, blood lead level; CI, confidence interval.

The earliest cases of childhood lead poisoning from lead-based paint were reported in Australia at the turn of the century.[1] Subsequently, ever-increasing numbers of cases were reported in the United States, particularly in old, central-city slums. Although the Consumer Product Safety Commission banned the addition of lead to paint in 1977,[2] previously applied leaded paint remains the principal source of high-dose lead exposure in the United States today.[3] The US Department of Housing and Urban Development estimates that 74% of privately owned, occupied housing built before 1980 contains some lead-based paint.[4] In the past, ingestion of leaded-paint chips, often due to pica, was considered to be the main cause of lead poisoning. In more recent years, it has become apparent that many children are poisoned through normal hand-to-mouth activity resulting in inadvertent ingestion of lead-contaminated dust and soil.[5,6] In fact, a recent report to Congress suggested that paint chip ingestion is "relatively infrequent."[4]

The issue of how often the direct ingestion of leaded-paint chips is involved in lead exposure has practical implications. For example, better knowledge of exposure pathways could help officials improve strategies for investigating the environments of lead-poisoned children; it could help them set priorities for which homes should be lead-abated first; and it could help them determine what temporary measures could be used to reduce a child's exposure to lead in situations where immediate, complete deleading of the home may not be feasible. We, therefore, conducted a study to estimate the frequency of leaded-paint chip ingestion among children with moderate to severe lead poisoning.

METHODS

The St Louis Division of Health's Lead Poisoning Prevention Program Clinic (LPPPC) provides confirmatory testing, evaluation, treatment, and follow-up care for children younger than 7 years of age with lead poisoning. Of the more than 12 000 children screened for lead poisoning each year by the St Louis Division of Health, 912 children in 1989 and 548 children in 1990 had lead poisoning and received medical evaluation and care at the LPPPC.

Children who are to receive chelation therapy at the LPPPC undergo an abdominal radiograph to detect evidence of leaded-paint chips in the intestinal tract. All radiographs are reviewed by a radiologist from a local teaching hospital.

We abstracted available medical and demographic data from the records of all children who received abdominal radiographs at LPPPC during 1989 and 1990 and who would routinely receive chelation therapy because they met one of two criteria: age < 7 years and blood lead level (BLL) ≥ 50 μg/dL (2.41 μmol/L); or age < 3 years and BLL ≥ 40 μg/dL (1.93 μmol/L) and a positive provocative chelation test. Only children who received their first chelation treatment or abdominal radiograph during 1989 and 1990 were included in the analysis. For children who had received more than one radiograph during this time, we selected the radiograph taken before the first chelation treatment.

Confidence intervals (CIs) for proportions were calculated using a normal approximation to the binomial. The statistical significance of differences between children with positive radiographs and children with negative radiographs was assessed using Student's t test (continuous variables) or the χ^2 test (discrete variables). Unconditional logistic regression was used to assess the association between BLL and radiograph results adjusted for other factors.

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RESULTS

During 1989 and 1990, 177 children had 232 abdominal radiographs done at the clinic. Ninety children met our selection criteria and were included in this study. The mean age of these children was 2.5 years (range 9 months to 6.5 years). Almost all of the children were identified as black (97%); 52% of the children were female. The mean BLL for these children was 56 μg/dL (median 55 μg/dL), and the mean erythrocyte protoporphyrin level was 139 μg/dL (median 120 μg/dL).

According to the radiologist's evaluation, 13 of 90 children (14%; 95% CI 7% to 22%) had evidence of paint chip ingestion on their abdominal radiographs. The Figure is an example of such a positive film. Children with positive radiographs had significantly higher BLLs than did those with negative radiographs (Table). Sex, age, and erythrocyte protoporphyrin levels were not found to be significantly associated with radiographic results. While most positive radiographs were taken during the warmer months (10 from May through September), the proportion positive during those months (17%) did not differ significantly from that found during other months ($P = .3$).

For the subgroup of children with a BLL equal to or greater than the median value (≥55 μg/dL, ≥2.65 μmol/L), 26% (12 of 46) had radiographic evidence of paint chip ingestion, compared with 2% (1 of 44) of the children with a BLL < 55 μg/dL (prevalence ratio 11.5; 95% CI 1.6 to 84.6; $P = .001$). In a logistic regression model, BLL, treated as a continuous variable, remained a statistically significant ($P = .003$) predictor of a positive radiograph after we adjusted for sex and age (<2 years vs ≥2 years).

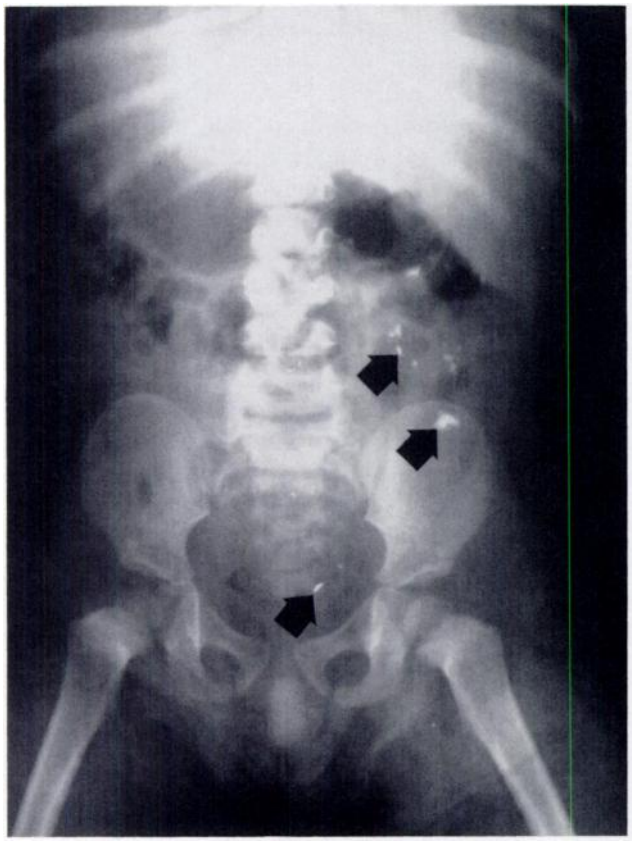

Figure. Abdominal radiograph of a child, showing radiographic densities in the colon (arrows). These densities are evidence that the child had recently ingested leaded-paint chips.

TABLE. Comparison of Lead-Poisoned Children With Radiographic Evidence of Paint Chip Ingestion With Lead-Poisoned Children Without Such Evidence

	Positive Radiograph (n = 13)	Negative Radiograph (n = 77)	*P* Value
Blood lead level, μg/dL			
Mean	63	55	.0003
Median	58	54	
Range	50–85	44–72	
Erythrocyte protoporphyrin level, μg/dL			
Mean	146	137	.706
Median	146	119	
Range	24–416	30–370	
Mean age, y	2.6	2.5	.656
Sex, % male	54	47	.638

DISCUSSION

In the clinic population we studied, 14% of children who were evaluated prior to an initial course of chelation therapy had radiographic evidence of leaded-paint chip ingestion. This is probably an underestimate of the true frequency of this behavior among children with moderate to severe lead poisoning. First, because the transit time of ingested material through a child's gastrointestinal tract may range from several hours to a few days, radiographs will only reveal recent paint chip ingestion. Second, the amount and size of the paint chips must be sufficient to be recognized on the radiograph.

In this clinic population, evidence of paint chip ingestion was associated with higher BLLs; almost all positive radiographs (12 of 13) were for children with BLLs ≥ 55 μg/dL. Although these data do not give us any information on the source or pathway of lead exposure for children with lower BLLs than those included in this study, they do provide strong evidence that ingestion of leaded-paint chips is an important source of lead poisoning for children with moderate to severe lead poisoning.

Because of limitations in the data, we were unable to evaluate certain questions about sources of the paint chips and the relative importance of paint chip ingestion. In particular, we could not determine whether children with positive radiographs had eaten fallen paint chips or had chewed on painted surfaces or objects. We also could not determine what proportion of lead exposure among children with positive radiographs was due to ingestion of the paint chips discernible in the radiographs and what proportion occurred through ingestion of lead-contaminated dust. Still, the results of this study point out the need for continuing concern about the ingestion of leaded paint itself, not just the ingestion of lead-contaminated dust, as an important cause of childhood lead poisoning in the United States.

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ABSTRACT

Rougemont A, Breslow N, Brenner E, Moret AL, Dumbo O, Dolo A, Soula G, Perrin L. Epidemiological basis for clinical diagnosis of childhood malaria in endemic zone in West Africa. *The Lancet.* 1991;338:1292–1295.

The authors studied simple clinical criteria to assist in distinguishing childhood malaria from other common febrile disorders. In areas endemic for malaria, the presence of malaria parasites on blood smear does not necessarily mean that malaria is the cause of a child's symptoms. The authors randomly selected 1 of every 3 febrile children, ages 2–9 years, who were matched with children of the same age, sex, and ethnic group who presented for reasons other than fever. They enrolled 557 children with fever and 557 controls. Children were examined, temperature was taken, and capillary blood sampled for thick and thin smear examination and hematocrits. Data analysis focussed on four variables: wet or dry season, probable origin of fever, duration of fever classified as less than 3 days or 3 days or longer before presentation, and degree of fever. The authors found that 39 non-febrile controls had more than 10,000 parasites per microliter. During the dry season few children, cases or controls, had parasite counts above 10,000 per microliter. There was no evidence that parasitemia was related to occurrence of fever in the dry season but it was strongly related during the rainy season. They noted that this decision framework requires health workers fully trained to detect other causes of fever, such as pneumonia, meningitis, or measles. They also note that some areas may lack clear seasonal patterns of malaria transmission and might, therefore, require different criteria in the clinical algorithm.

Comment: Immunity to malaria develops in endemic areas but varies significantly among individuals living in the same area. Those with substantial immunity may tolerate substantial parasitemia whereas those without immunity may develop severe malaria with few parasites. Casual diagnosis of malaria, on the basis of smear only, may result in missed diagnoses of other infectious illnesses. These authors propose a reasonable treatment criterion for children living in an endemic area with wet and dry seasons.

Submitted by Karen Olness, MD