

SOUTHWEST CENTER FOR PEDIATRIC ENVIRONMENTAL HEALTH,
AND UNIVERSITY OF TEXAS HEALTH CENTER AT TYLER

An Unexplained Case of Elevated Blood Lead in a Hispanic Child

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A 6-month-old child presented to a local pediatrician with an elevated blood lead level (BLL) of 41 µg/dL. The child was treated as an outpatient for chelation therapy by a toxicologist. Subsequent BLLs obtained at 8 and 13 months of age were 40 µg/dL and 42 µg/dL, respectively. Siblings and family members had BLLs < 5 µg/dL except for the mother, who had a BLL of 14 µg/dL when the child was 6 months of age. Home inspections and phone calls to the family revealed no sources of lead from paint, dust, toys, mini-blinds, keys, food, water, or any take-home exposure. The family denied use of folk remedies such as Greta and Azarcon. The child was breast-fed, but the mother's BLL was not sufficiently high to explain the elevated BLL in the child. Housekeeping was excellent. The mother did admit to cooking beans in Mexican pottery (pieces found outside were positive for lead), but she discontinued use after the initial lead check at 6 months. The bean pot was not a likely source, as none of the family had elevated BLLs including a 5-year-old sister. Follow-up testing of blood lead when the child was 15 months of age revealed values of 28 µg/dL for the child and 9 µg/dL for the mother. Subsequent testing of the child shows a slow decline. The slow release of lead suggests depletion of bone stores acquired during pregnancy, possibly due to pica behavior of the mother during pregnancy. **Key words:** adult pica, blood lead, children, Hispanic family, lead-glazed pottery, lead kinetics. *Environ Health Perspect* 112:222–225 (2004). doi:10.1289/ehp.6486 available via <http://dx.doi.org/> [Online 12 November 2003]

Case Presentation

The Southwest Center for Pediatric Environmental Health (SWCPEH) received a referral from the Texas Department of Health (TDH) in late August 2002 regarding a case of unexplained elevated blood lead levels (BLLs) in an 11-month-old female Hispanic child. The child's pediatrician reported a BLL of 41 µg/dL to the TDH initially when the child was 6 months of age (March 2002), and a confirming test 1 week later of 36 µg/dL. The mother and a 5-year-old sibling were tested in March 2002 with BLLs of 14 µg/dL and negative (< 5 µg/dL), respectively. According to Centers for Disease Control and Prevention (CDC) guidelines, the level of concern for BLL in children under 6 years of age is 10 µg/dL (CDC 1991).

The residents of the home included the child's mother, father, 5-year-old sister, and grandfather. At 9 months of age, the child had anemia (hemoglobin, 6.4 mg/dL; hematocrit, 21.2%), which was treated with supplemental iron, but otherwise, the child had normal growth and development. In early April 2002, the child's pediatrician referred the child to a toxicologist, who prescribed a course of succimer to reduce BLLs (Horowitz

and Mirkin 2001; Rogan 2000; Rogan et al. 2001). Repeat blood lead tests in late April (40 µg/dL) and in mid-June (37 µg/dL) revealed that the child still had elevated BLLs. A second round of chelation therapy was not prescribed because elimination of the source of lead exposure was considered to be more important; chelation therapy would be futile in the case of continuing exposure.

The persistently elevated BLL raises two questions: What was the source of the lead, and why did succimer treatment not reduce the child's BLL?

In early May 2002, the TDH sent an English-speaking investigator to the home. All members of the family spoke English except the child's grandfather. An inspection was conducted using a standardized lead questionnaire addressing the usual sources of lead in the home and testing a variety of surfaces with a Niton XL 309 portable X-ray fluorescence meter (XRF; Niton LLC, Billerica, MA) (CDC 1991). The investigator's report indicated immaculate housekeeping in the family's well-maintained mobile home. No sources of lead were identified from the questionnaire. Tests of floors, windows, walls, mini-blinds, and soil outside the house were negative by

XRF. Forty readings were taken; nearly all were between the detection limit and 0.5 mg lead/cm². The evaluation criteria for a "positive" lead reading are any values > 1 mg/cm². The infant did not play outside. The child spent most of her time inside the home with her mother, but she and her mother also frequently visited the mother's sister who lived nearby. The mother's sister had young children also, but her children never had a lead problem, according to the mother.

Repeat testing of the child for blood lead in mid-June 2002 revealed a continued elevation in BLL (37 µg/dL). In late July 2002, the TDH called for assistance from the SWCPEH.

Discussions with the child's pediatrician established that *a*) the child was breast-fed exclusively for 6 months; *b*) the child was screened by finger stick as part of the Texas Health Steps program (a program for high-risk children), and a venous blood confirmatory specimen was collected 1 week later; *c*) the child began eating solid food at 6 months of age (baby food and bottled water purchased from the local market); and *d*) the pediatrician reported no apparent source of lead based on discussions with the mother.

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The demographics of the family revealed the child's mother to be an articulate second-generation person of Mexican heritage who was the primary caregiver for her child. At the initial visit in March 2002, the mother and 5-year-old sister were tested. The child's father was employed as a roofer and had no apparent occupational exposure. The child's grandfather was a part-time roofer and part-time caregiver for the child. No BLL data were available initially for the father or the grandfather.

In early August 2002, we made numerous phone calls to the child's pediatrician, the child's mother, and the TDH. During these discussions, no apparent new source of lead was determined. The family was advised to practice good hygiene with frequent hand washing and washing of the child's toys, as well as ensuring adequate iron and calcium in the diet. In September 2002, the BLL for the child remained unchanged at 42 µg/dL. Table 1 shows a comprehensive list of BLL results for the child and the family members at different times. Table 2 shows the child's hematology data.

In early October 2002, two of the authors (D.C.C. and C.F.B.) and the TDH inspector made a return visit to the home. Brady, who speaks Spanish and understands Mexican cultural traditions, accompanied the site-visit team, primarily to speak with the child's grandfather and to seek out environmental sources of lead. During this visit, additional family history was obtained along with an extensive evaluation of nontraditional sources of lead. (CDC 2002a). The family was questioned and the home examined for evidence of lead-containing toys, paint on the crib, lead in the bathing tub, pots, pans, cosmetics, ceramics, imported foods and candies, candles, key chains, herbal remedies, lead fishing sinkers, and munitions. In addition, the family denied use of "greta" and "azarcon," traditional folk remedies that often contain 60–70% lead by weight; these remedies are traditionally given to children with "empaño," usually defined as stomach cramps or stomachache (CDC 2002a). A broken piece of Mexican pottery (Figure 1) found beside the garbage can contained 25 mg lead/cm²; we considered values > 1 mg lead/cm² "positive" test for lead. Two other sources of lead were identified: a discarded metal cupola with lead-based paint located on a hill some distance

from the home, and a small amount of lead on a battery-powered toy car used by the 5-year-old sister. Neither of these two sources could be an exposure pathway for the young child.

Additional family history revealed that beans were cooked in the Mexican lead-glazed pot, a piece of which was found outside. The child's mother stated that she had used the pot for cooking beans during her pregnancy and until the initial elevated BLL was found when the child was 6 months of age. At that time, she destroyed the pot and discontinued using any lead-glazed Mexican pottery. Between 6 and 12 months of age, the child began eating traditional Mexican foods along with baby food and bottled water. The family home was supplied with city water, and the home used nonmetallic plumbing. Additional information obtained during this visit revealed that the family did not have pets; the child did not use a pacifier; take-home exposure by those working outside the home was unlikely; and there was no lead-containing jewelry in the home. Additional testing of carpet dust for lead was negative (< 0.02% lead by weight). A timeline for the preceding events is shown in Table 3.

Discussion

How can the consistently elevated BLL in this child be explained in the absence of any identified source of lead? How can we explain the inability of oral chelation therapy to lower BLLs in the child? Why does no other family member have an elevated BLL? How can the slightly elevated BLL in the mother be explained in the absence of any identified source of lead? In order to answer these questions, we examined the facts and developed a hypothesis.

At first testing, the infant was ingesting only breast milk. The source of lead at that time must have been related to the mother because no environmental sources were identified (unless someone was giving the child a folk remedy and not telling us). The mother's elevated BLL (14 µg/dL) at the time of the first testing of the child would not explain the significantly higher BLLs in her nursing child. Breast-feeding alone could not explain the elevation of BLL in the child (Gulson et al. 1998).

The mother cooked beans in a lead-glazed pot until the child's first BLL testing, but this

cannot explain the source of lead in the child, because the mother's milk was the only source of food for the child. As the pot was destroyed at that time, it is unlikely that pica behavior by the child could account for the elevation in BLL, even though pica behavior in older children is a rather common source of lead. In addition, if the bean pot were the source of lead, then other members of the family who consumed the beans would be expected to show some elevations in BLL. However, BLLs in other family members were not elevated.

The lack of any identifiable source of lead in the household and the lack of elevated BLLs in other family members, except for the minor elevation in the mother, rules out a common source of lead in the home. The focus is thus on the slightly elevated BLL in the mother as the source of elevated BLL in the child.

One possible explanation is that exposure occurred *in utero* from pica activity of the mother and not from eating food cooked in lead-glazed pottery. Several studies have shown that pica behavior in adults is not uncommon. Indeed, there are reports of chronic pica in adults dating back to 1968 (Mitchell 1968), and more recent studies have also confirmed pica behavior in adults (Krengel and Geyser 1978). Several studies have reported pica behavior in women during pregnancy (Bronstein and Dollar 1974; Goldstein 1998), and other studies have reported maternal pica behavior in Hispanics (Rothenberg et al. 1999; Shannon 2003; Simpson et al. 2000).

Does maternal lead pica behavior during pregnancy result in elevations of BLL in the newborn or child? Hamilton et al. (2001) noted that a mother who apparently chewed pieces of lead-glazed pottery during pregnancy presented with an elevated BLL at delivery (119 µg/dL). Her child, who had an umbilical cord BLL of 114 µg/dL, was treated with a double exchange transfusion and intravenous

Table 2. Child's hematology data.

Date	Hemoglobin (mg/dL)	Hematocrit (%)
Jun 2002	6.4	21.2
Sep 2002	11.1	33.6
Nov 2002	11.8	38.3
Mar 2003	12.3	36.3

Table 1. Blood lead levels (µg/dL) for the family.

Person	Mar 2002		Apr 2002	Jun 2002	Sep 2002	Oct 2002	Nov 2002	Jan 2003	Mar 2003
	Early	Late							
Female child	41	36	40	37	42	36	28	21	16
5-Year-old sister	<5	—	—	—	—	—	<5	—	—
Mother	14	—	—	—	—	—	9	—	—
Father	—	—	—	—	—	—	3	—	—
Grandfather	—	—	—	—	—	—	2	—	—

—, No data available.



Figure 1. Broken piece of lead-glazed pottery found in yard.

EDTA for 5 days. The BLL in the child dropped to 13 $\mu\text{g}/\text{dL}$, and the child was reported to be doing fine, although no long term follow-up was noted (Hamilton et al. 2001). Others have shown that maternal pica behavior during pregnancy can result in elevated bone lead not only in the mother but also in the newborn (Markowitz and Shen 2001). Therefore, maternal lead pica during pregnancy could lead to an elevated BLL in the child.

If we assume that maternal lead pica behavior during pregnancy can lead to elevated blood and bone lead levels in newborns, how can we explain the relative stability of BLLs in the child as well as in the mother? Also, how can we explain the decline in lead levels on the basis of biokinetic models for lead? The kinetics of lead in adults is based on the International Commission for Radiation Protection biokinetic model (Pounds and Leggett 1998), which describes the elimination kinetics of lead from the blood as having two half-lives: a rapid half-life of 27 days representing losses from circulating blood, and a long half-life of 10,000 days (27 years) representing losses from bone. Note that the first BLL data on the mother was 14 $\mu\text{g}/\text{dL}$ when the child was 6 months of age. The follow-up BLL in the mother some 10 months later was 9 $\mu\text{g}/\text{dL}$. Figure 2 is a plot of the child's BLLs showing a gradual decline over time. Although there is no data on the BLLs of the mother at delivery or in the neonate, it seems reasonable that what is observed is a gradual release of lead from the bone compartment in both the mother and the child. The elimination rates for mother and child can be calculated as

$$k_{cl} \text{ for child} = 0.07 \mu\text{g}/\text{dL}/\text{day} \\ (n = 8 \text{ samples})$$

$$k_{cl} \text{ for mother} = 0.05 \mu\text{g}/\text{dL}/\text{day} \\ (n = 2 \text{ samples}),$$

where k_{cl} is the elimination constant. Although these elimination rates are oversimplified,

Table 3. Timeline and blood lead levels, index case.

Date	Event	BLL
Oct 2001	Birth of child	
Mid-Mar 2002	6-Month checkup	41 $\mu\text{g}/\text{dL}$
Late Mar 2002	Recheck	36 $\mu\text{g}/\text{dL}$
Early Apr 2002	Oral chelation with succimer	
Late Apr 2002	Blood lead check	40 $\mu\text{g}/\text{dL}$
Early May 2002	Initial home inspection, TDH	
Mid-June 2002	Blood lead check	37 $\mu\text{g}/\text{dL}$
Late Jul 2002	SWCPEH contacted	
Early Sep 2002	Blood lead check	42 $\mu\text{g}/\text{dL}$
Early Oct 2002	SWCPEH, TDH visit	36 $\mu\text{g}/\text{dL}$
Nov 2002	Blood lead check	28 $\mu\text{g}/\text{dL}$
Jan 2003	Blood lead check	21 $\mu\text{g}/\text{dL}$
Late Mar 2003	Blood lead check	16 $\mu\text{g}/\text{dL}$

based on a small number of points, they are similar for mother and child, which indicates a possible common source of lead and a slow release from bone.

The decrease in the child's BLL is more rapid than the 27-year half-life (presumably the bone lead component) would predict, but the biokinetic data is for adults. It is reasonable to assume that *in utero* lead exposure during the time of rapid bone development and growth would result in increased lead mobilization in the child. Also, as the child grows, blood volume increases, resulting in a dilution of the lead content.

Why did oral chelation not cause more significant reduction in BLLs in the child? (The mother confirmed that the child was given the entire dose of chelating agent, but because it was given as outpatient treatment, there is no independent confirmation.) First of all, chelation of asymptomatic children with BLLs between 20 and 44 $\mu\text{g}/\text{dL}$ has been recently questioned (Rogan 2000). It seems reasonable that chelation therapy is effective in removing lead from blood and tissues in rapid equilibrium with the blood. However, if the child had substantial bone stores, the removal of lead from the blood would only be temporary because releases from the bone would increase during and after chelation until steady state was reestablished. This may explain the apparent lack of effectiveness for chelation therapy in this child.

On the basis of the assumptions described above, the BLL in the child could be expected to decrease about 2–3 $\mu\text{g}/\text{dL}/\text{month}$. According to the CDC (1991), children should be rechecked at quarterly intervals if BLLs exceed 20 $\mu\text{g}/\text{dL}$, and they should be evaluated neurobehaviorally if BLLs remain > 20 $\mu\text{g}/\text{dL}$. Also, the family was advised to maintain a diet rich in calcium, iron, and vitamin C and to use dairy products regularly as a source of calcium as recommended by the CDC (2002b).

After the investigation ended, the mother did admit to chewing on particles of glazed Mexican pottery during her pregnancy.

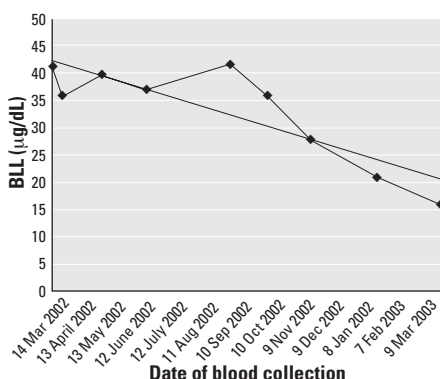


Figure 2. Child's BLLs over time.

Conclusions

This case report demonstrates that an investigation into unexplained lead exposure may need to include site visits by health professionals who are not only bilingual but also culturally sensitive. The identification of pica behavior in this Hispanic mother was revealed to Brady during a quiet moment; it must have been embarrassing to the mother that her behavior was responsible for her child's elevated BLL.

This case also demonstrates that all possible sources of lead should be investigated to determine the source of exposure. If this case study was part of a clinical investigation, many additional tests could have been performed. If BLLs were known for the mother and child at delivery, the hypothesis would have been easier to prove; for example, a number of studies could have been performed to evaluate bone stores in the mother. However, this case took place in a rural county of east Texas, far from a research center, and it was only brought to our attention by an observant local pediatrician.

Many cases in the literature have reported take-home exposure by parents who work in an industry where lead is present. These must be investigated, as well as those traditional sources of lead identified in standard questionnaires. As this case demonstrates, it may be necessary to consider cultural practices in specific ethnic groups to determine possible lead exposure from, for example, the use of folk medicines or lead-glazed pottery. Pica behavior in pregnant women is not unique to Hispanics; however, this woman's behavior probably would not have been discovered without the trust that developed because culturally sensitive health professionals expressed genuine concern. This case also demonstrated the real value of the Pediatric Environmental Health Specialty Units as a resource and how they can work together with local health professionals to resolve public health issues important to children.

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