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Epidemiologic Characteristics of Children with Blood Lead Levels ≥45 μg/dL

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Objectives To identify risk factors and describe outcomes for children newly identified with blood lead levels (BLLs) ≥45 μg/dL in New York City (NYC) during 2004-2010 to promote timely identification as well as inform clinical practice and public health policy.

Study design Inclusion criteria were residence in NYC and an elevated confirmatory venous test within 2 weeks of the initial BLL ≥45 μg/dL. Data collected during case coordination of these children were linked with blood testing data and home inspection reports. Children with BLLs ≥45 μg/dL also were compared with the general population of children younger than 18 years of age in NYC.

Results A total of 145 children <18 years of age were newly identified with BLLs ≥45 μg/dL. The mean age was 3.83 years, and the median time for BLL to decline below 10 μg/dL was 3.26 years. Major reported risk factors were eating paint (36%), spending time outside the US (34%), having a developmental delay (27%), using imported products (26%), being foreign born (14%), being of Pakistani descent (12%), eating soil (5%), and having sickle cell disease (4%). Compared with the age-standardized NYC population, cases were more likely to be Asian or black and live in housing built before 1940.

Conclusions Although the incidence of lead poisoning has declined in the US, severe cases still occur. Physicians should be especially vigilant in certain at-risk populations including children who eat paint chips or soil, spend time outside the US (particularly in Pakistan), use imported products, or have developmental delays or sickle cell disease. *(J Pediatr 2016;■■:■■-■■).*

National and local policies have led to large reductions in the prevalence of lead poisoning. Two national surveys of children aged 1-5 years showed a decline in the prevalence of blood lead levels (BLLs) ≥10 μg/dL from 88.2% in 1976-1980 to 0.8% in 2007-2010.[1,2] Children in New York City (NYC) newly identified with BLLs ≥10 μg/dL declined 92% from 21 575 to 1634 between 1995 and 2009.[3]

As the incidence of BLLs ≥10 μg/dL has declined and evidence of health effects at lower BLLs has accumulated,[4] attention has been directed increasingly to less severe lead exposures. Although low-level exposures can result in long-term, irreversible cognitive deficits, greater exposures can lead to organ damage and death. Deaths from lead poisoning still occur, with at least 2 child fatalities in the US since 2000, a Sudanese refugee child exposed to lead paint in New Hampshire and a child who swallowed a charm in Minnesota.[5,6]

The objective of this review is to describe NYC children younger than 18 years of age who were newly identified with severe lead poisoning, defined here as BLLs ≥45 μg/dL. Data collected during case coordination and environmental investigation on the potential exposures are presented, as well as demographics, associated risk factors, results of abdominal radiographs, and the time to decline to a BLL <10 μg/dL. Understanding the incidence, sources, and treatment outcomes of lead poisoning at BLLs ≥45 μg/dL can promote timely identification of cases as well as help inform clinical practice and public health policy.

Methods

All cases with initial venous BLLs ≥45 μg/dL among children younger than 18 years of age from 2004 through 2010 were reviewed. Inclusion criteria were residence in NYC and an elevated confirmatory venous test within 2 weeks of the initial BLL ≥45 μg/dL. The institutional review board of the NYC Department of Health and Mental Hygiene (DOHMH) deemed this project exempt research.

BLL	Blood lead level
DOHMH	Department of Health and Mental Hygiene
NYC	New York City
PDD	Pervasive development disorder
RA	Risk assessment
SCD	Sickle cell disease
XRF	X-ray fluorescence

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The Lead Poisoning Prevention Program within the DOHMH maintains a registry containing all blood lead test results on children in NYC. New York State mandates blood lead testing at ages 1 and 2 years. The registry links BLL tests for a child to basic demographic information and notes entered by DOHMH caseworkers. The system is also linked to various city and state data sources including vital records, immunization records, and housing databases.

Home visits by DOHMH inspectors offered to families of children with BLLs ≥15 μg/dL involve an environmental inspection and a standardized interview with an adult family member, typically the child's guardian. A risk assessment (RA) form is completed that includes responses to questions about the child's behavior as well as information about recent home renovations/repairs, occupations/hobbies of household members, and use of imported products. If certain imported items are available, they are tested for lead content.

In 2006, the RA form was revised with 2 notable changes. Before 2006, the RA asked about home renovations in the last 3 months, whereas since 2006, the RA asks about home renovations in the last 12 months. In addition, the RA used since 2006 specifically asks about eating paint. In 2007, the RA was made electronic.

During home visits, inspectors use handheld x-ray fluorescence (XRF) devices to evaluate the lead content of nonintact painted surfaces as well as windowsills regardless of paint condition. An XRF reading ≥1 mg/cm^2 lead is considered a hazard, and the NYC health code mandates landlords abate lead hazards in homes of children with lead poisoning.[7] The presence or absence of lead paint hazards for each case was documented. In homes with more than 1 inspection, XRF results from the earliest inspection were used because an inspection typically results in abatement of lead hazards.

Case coordination staff communicates with health care providers. Providers are advised to obtain an abdominal radiograph to check for recent ingestion of radiopaque material before initiating chelation and use a cathartic if radiopaque particles are found, as some chelating agents may increase lead absorption. Staff asked providers for abdominal radiograph results, and beginning in 2006, for the results of nutritional and developmental assessments. Children originally reported to have language delays or attention deficit hyperactivity disorder but later reported to be autistic were only reported in the autism category.

A single dataset was created by reviewing all demographic fields and case event notes for all the cases that met review criteria in the registry. In addition, RA forms were matched to cases. Electronic RA form data were exported and reviewed in spreadsheets, whereas older forms were reviewed manually. In cases with more than 1 RA form, answers to questions about risk factors were recorded as "yes" if an affirmative response was given on any of the RA forms or in any of the case comments.

Comparison Data

Demographic data including age, sex, race/ethnicity, borough, and poverty by census tract for all children <18 years of age in NYC were obtained from the 2010 US Census and 2005-2009 American Community Survey accessed through NYC EpiQuery.[8] The cases were grouped by year(s) of age (<1; 1-2; 3-4; 5-9; 10-17 years). Each case was classified into 1 of 5 racial/ethnic categories: non-Hispanic Asian, non-Hispanic black, non-Hispanic white, Hispanic of any race, and other (including non-Hispanic mixed race, Native Hawaiian/Pacific Islander, and non-Hispanic of unknown race). In addition, census tracts in which >30% of the population lived below the poverty level were examined.[9] The address at the time of first home inspection was used to look up the year the housing was built in the NYC PLUTO database, and the same database provided a distribution of year built for all buildings in NYC with residential units.[10] We also reviewed NYC immunization records to fill in missing Medicaid data.

Statistical Analyses

Proportions of cases in each demographic group were compared with the proportion in the NYC population standardized by year with EpiQuery. Statistical comparisons were performed with the χ^2 test for categorical variables and *t* test for continuous variables. An exact binomial test was used when expected counts were less than 5. Survival analysis with Bonferroni correction and censoring were used to analyze time to decline to BLL <10 μg/dL. Analyses were performed with SAS 9.1 for Windows (SAS Institute, Cary, North Carolina).[11]

Results

A total of 145 children <18 years of age newly identified with BLLs ≥45 μg/dL from 2004 to 2010 in NYC were found in the database, a mean of 20.7 children annually. Hereafter these are referred to as "cases."

The mean age for cases with BLL ≥45 μg/dL was 3.83 years (95% CI 3.34-4.32). Cases were more likely to be in the 1- to 2-year and 3- to 4-year age groups than in age groups older than 5 years ($P < .001$ for each comparison; **Table I**). Four cases (3%) were newborns (first BLL test at 0-8 days of age) born to mothers with known elevated BLLs.

Distribution of cases differed from the standardized NYC population by race/ethnicity ($P < .001$), borough of residence ($P < .001$), and year housing was built ($P < .001$). Cases were more likely to be Asian ($P < .001$) and black ($P = .005$) and less likely to be white ($P < .001$) than the age-standardized NYC population. Cases were more likely to live in Brooklyn ($P < .001$) and less likely to live in Manhattan ($P = .004$). The residences of cases also were more likely to have been built before 1940 ($P < .001$; **Table I**).

Of the 145 cases, 119 (82%) reported a Medicaid number, and in 2009 only 57.9% of live births in NYC were covered by Medicaid. No difference was found in census tract poverty rates between cases and the NYC population (**Table I**).

Foreign Origin, Travel History, and Use of Imported Products

One hundred twenty-five of the 145 (86%) cases were born in the US. Of the 20 (14%) foreign born, 10 (50%) were born

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Table I. Comparison of demographic characteristics and risk factors between BLL ≥45 μg/dL cases and NYC population

Characteristics	BLL ≥45 μg/dL cases (n = 145), n (%)	NYC population* (n = 1 913 736), (%)	P value†
Sex			.19
Female	63 (44)	(48.9)	–
Male	82 (57)	(51.1)	–
Age, y			<.001
<1‡	6 (4)	(6.2)	.30
1-2	63 (44)	(11.9)	<.001
3-4	46 (32)	(12.1)	<.001
5-9	21 (15)	(27.0)	<.001
10-17	9 (6)	(42.8)	<.001
Race/ethnicity			<.001
Asian Non-Hispanic	38 (26)	(11.1)	<.001
Black Non-Hispanic	52 (36)	(25.7)	.005
Any Hispanic	43 (30)	(33.7)	.30
Other	6 (4)	(2.4)	.17
White non-Hispanic	6 (4)	(27.1)	<.001
Poverty status by census tract			.70
<30% below poverty level	100 (69)	(67.5)	–
≥30% below poverty level	45 (31)	(32.5)	–
Borough			<.001
Bronx	28 (19)	(19.9)	.86
Brooklyn	71 (49)	(33.5)	<.001
Manhattan	10 (7)	(15.7)	.004
Queens	30 (21)	(25.6)	.18
Staten Island	6 (4)	(5.4)	.51
Housing year built§			<.001
Pre-1940	114 (79)	(60.6)	–
1940 or later	31 (21)	(39.4)	–
Child's birth country	*n = 145*	–	
US	125 (86)	–	–
Other country	20 (14)	–	–
Pakistan	10 (7)	–	–
Mother's birth country¶	*n = 95*		<.001
US	25 (26)	(42.9)	–
Other country	70 (74)	(57.1)	–
Pakistan	18 (19)	unknown	–
Mexico	13 (14)	unknown	–
Traveled in last 12 mo	*n = 137*		–
Yes	46 (34)	–	–

*NYC EpiQuery 2004-2009, age-standardized for comparisons by sex, race/ethnicity, and borough (not age-standardized for comparison by age).
†*P* values are for χ^2 test unless otherwise specified.
‡Age groupings from American Community Survey 2005-2009 data.
§1940 used based on Housing and Urban Development data showing that housing before 1940 more likely to have lead paint as well as more likely to have lead paint hazards. American Healthy Homes Survey: Lead and Arsenic Findings. April 2011.
¶Comparison population for mother's birth country from NYC Birth and Infant Mortality Trend report.[12]

in Pakistan (Table I). Country of birth for the child's mother was known for 95 cases. Seventy (74%) mothers were born in foreign countries, including Pakistan (18, 19%), Mexico (13, 14%), Bangladesh (4), Dominican Republic (4), Nigeria (4), Jamaica (3), Ecuador (2), Guyana (2), Haiti (2), and Mali (2). By comparison for the period 2004-2009, 51.7% of births in NYC were to foreign-born women and less than 1.3% of women 18 years and older in NYC were born in Pakistan.[8,12] In total, of the children whose mother's country of birth was known, 10 children were born in Pakistan (including one set of siblings) and an additional 8 were born to mothers of Pakistani birth (including another set of siblings) for a total of 18 (12%; Table I).

Table II. Other risk factors of BLL ≥45 μg/dL cases

Risk factors for lead poisoning	BLL ≥45 μg/dL cases, n (%)
Use of imported products	n = 103
None reported	76 (74)
Any reported	27 (26)
Food	24 (23)
Pottery	7 (7)
Cosmetic	7 (7)
Medicine	5 (5)
Behavioral risk factors	n = 145
Mouths, chews, or eats nonfood items	83 (57)
Mouths, chews, or eats toys	51 (35)
Mouths surfaces	22 (15)
Mouths windowsills	9 (6)
Eats paint chips or soil	n = 103
Eats paint chips	37 (36)
Soil or sand	5 (5)
Lead hazard at residence	n = 145
No	26 (18)
Yes	119 (82)
Family member occupation hazard	n = 145
Does not have lead hazard	118 (81)
Does have lead hazard	27 (19)
Home repairs in last 12 mo	n = 145
No	91 (63)
Yes	54 (37)
Developmental delay	n = 115
None reported	84 (73)
Any reported	31 (27)
Language delay	15 (13)
PDD, including autism	7 (6)
Attention deficit hyperactivity disorder	5 (4)
Other delay not specified	8 (7)

Forty-six cases (34%) had traveled or resided outside the US in the past 12 months (Table I). The countries where more than 1 child had spent time were Pakistan (18, 12%), followed by the Dominican Republic (4), Bangladesh (3), Mexico (3), China (2), and Ecuador (2).

The use of imported products was assessed for 103 of the children, of whom 27 (26%) reported using at least 1 imported product (Table II). Of these 103 children, 24 (23%) reported using imported food, 7 (7%) pottery, 7 (7%) cosmetics, and 5 (5%) medicines. In 6 children of Pakistani descent, surma (a cosmetic) use was reported. A total of 96 products were available for testing, and 12 (13%) contained more than 10 ppm lead. Three samples of surma all had high lead levels (1.4%, 22%, and 25%). The 9 other imported items containing more than 10 ppm of lead included a cough remedy powder purchased in Russia (750 000 ppm or 75%), an abdominal discomfort remedy made in South Korea (8075 ppm), chapulinas (dried, spiced grasshoppers) imported from Mexico (2900 ppm), 2 samples of turmeric imported from Bangladesh from 2 unrelated cases (1424 ppm and 2000 ppm), cassava grain from Nigeria (38 ppm), ingested incense ash from Gambia (31 ppm), a yellow metal toy car from China that a child mouthed (19 ppm), and chili powder from Bangladesh (12 ppm).

BLL Testing and History

Twenty-three (16%) cases were identified as having a BLL ≥45 μg/dL on their first recorded blood lead test (Table III).

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Table III. Characteristics of BLL ≥45 µg/dL cases

Characteristics	BLL ≥45 µg/dL cases, n (%)
Previous BLL tests	n = 145
No previous test	23 (16)
1	34 (23)
2	29 (20)
3 or more	59 (41)
Highest previous BLL	n = 145
No previous test	23 (16)
0-4	26 (18)
5-14	26 (18)
15-24	17 (12)
25-34	14 (10)
35-44	39 (27)
First BLL ≥45 µg/dL	n = 145
45-54	100 (69)
55-69	38 (26)
70+	7 (5)
Peak BLL ≥45 µg/dL	n = 145
45-54	82 (57)
55-69	45 (31)
70+	18 (12)
Abdominal radiograph for radiopaque material	n = 145
None reported	48 (33)
Negative	72 (50)
Positive	25 (17)
Chelation history	n = 145
No chelation	19 (13)
1 course	87 (60)
2 courses	22 (15)
3+ courses	17 (12)

Of those, 3 were newborns, 9 were recent immigrants to the US (including 7 born in Pakistan), and an additional 7 had traveled abroad (3 to Pakistan, 1 each to Bangladesh, China, Ecuador, and Canada).

The distribution of the initial BLLs ≥45 µg/dL included 100 (69%) with BLLs 45-54 µg/dL, 38 (26%) with BLLs 55-69 µg/dL, and 7 (5%) with BLLs ≥70 µg/dL. In 47 cases (32%), the initial BLL ≥45 µg/dL was not the greatest (peak) BLL. Peak BLL readings for cases included 82 (57%) with BLLs 45-54 µg/dL, 45 (31%) with BLLs 55-69 µg/dL, and 18 (12%) with BLLs ≥70 µg/dL. The greatest reported BLL was 227 µg/dL.

Treatment, Radiograph History, Behavioral Risk Factors

Chelating agents typically are given to children with BLLs ≥45 µg/dL on repeat venous blood lead analysis. Chelating agents used included oral dimercaptosuccinic acid, intravenous calcium edetate, and intramuscular British anti-Lewisite. On the basis of the BLL, a course of chelation typically lasts from 5 to 19 days and may include a combination of these agents. Cases had between 0 and 9 documented courses of chelation (Table III). The majority of cases (87, 60%) received 1 course. One newborn received an exchange transfusion.

The results of abdominal radiographs were documented for 97 cases (67%). Of these, 25 (26%) cases had an abdominal radiograph positive for radiopaque particles suggestive of lead paint chips. Of the 103 families asked whether their child mouthed or ate paint chips or soil, 37 (36%) reported their child eating paint chips and 5 (5%) reported their child eating soil or sand. Of the 37 cases who reported eating paint, 8 (22%) had positive abdominal radiographs. Of the 66 cases who denied eating paint, 9 cases (14%) had positive abdominal radiographs.

Other Risk Factors and Associations

Lead paint hazards were found in 82% of the homes via XRF testing. Recent home repairs were reported by 37% of cases. Having a household member with an occupation/hobby at risk of lead exposure was reported by 19% (Table II). In this study, associations were found between lead poisoning and anemia, sickle cell disease (SCD), and developmental delays including autism. Twenty-three (16%) cases were reported to be anemic. Six (4%) had SCD (5 with sickle cell SS anemia, 1 with hemoglobin SC disease) and all were non-Hispanic black. Records included data on developmental delays gathered through contacts with families and developmental assessments by providers for 115 cases. Of these, 31 (27%) reported developmental delays, of which 15 (13%) were language delays and 7 (6%) were autism. The "other delay not specified" category included gross motor delay, impaired sensory integration, hypertonia, learning disability, and nonspecified development delays.

The cases with reported autism and SCD were older than cases without these disorders. Although the mean age for the cases with autism was greater than the mean age of the 108 cases without autism (M = 5.99 years vs M = 3.69 years, respectively), this difference was not statistically different. Cases with SCD were significantly older (6.97 years, 95% CI 3.61-10.33) than those without this disorder ($P = .008$).

Time to Decline

Product-limit survival estimates were calculated by using the time from first BLL ≥45 µg/dL until BLL declined to <10 µg/dL. Median time to decline was 3.26 years (95% CI 2.99-4.05). Cases were grouped by chelation history. Median time of decline to <10 µg/dL differed by chelation history. Median time to decline was 2.33 years (95% CI 1.05-3.28 with 30.8% censored) for cases with no chelation, 3.01 years (95% CI 2.45-3.28 with 40.9% censored) for cases with 1 chelation, and 6.53 years (95% CI 3.51-6.53 with 75.0% censored) for cases with 2 or more chelations.

The median time to decline to <10 µg/dL for the 4 newborns was 1.05 years, which was statistically different than the median time to decline to <10 µg/dL of 3.26 years for non-newborn cases (log-rank $X^2 = 13.52$, $P < .001$).

Discussion

Overall, paint is the most common high-dose source of lead for children in the US.[13] A previous study of children in St Louis found a 26% prevalence rate of positive radiographs with BLLs ≥55 µg/dL as opposed to only 2% with BLLs 45-54 µg/dL.[14] This case review found a much greater prevalence rate of 48% positive radiographs with BLLs ≥55 µg/dL and 17% with BLLs 45-54 µg/dL. Therefore, this review confirms that paint con-

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sumption is common among children with BLLs ≥45 μg/dL, with overall 26% having a positive abdominal radiograph suggestive of lead paint consumption and 36% having been observed eating paint.

A previous matched case-control study found an increased risk of lead poisoning for children born in other countries compared with US-born children.[15] Our study, in which foreign-born children were overrepresented compared with the general NYC population, lends further support to this conclusion. Lead poisoning remains a problem in Pakistan,[16-18] which may partially explain why a greater-than-expected proportion of children with BLLs ≥45 μg/dL were of Pakistani descent.

Lead poisoning can result from many sources of exposure,[19,20] and a variety of potential sources were found. One report estimated that ≥30% of children with elevated BLLs were not exposed to lead paint hazards.[4] Imported spices, cosmetics, and medicines have been linked to pediatric and adult lead exposure, leading to the recommendation that physicians routinely screen for exposure to such products.[21,22]

Lead poisoning has been associated with other health problems, including SCD and autism. Children with SCD may have dysfunctional eating patterns as a coping mechanism, which may result in more ingestion of lead.[23] Similar to Issaivanan et al,[23] we found that children with lead poisoning with SCD were older than cases without SCD. Children with pervasive development disorder (PDD) behave like younger children, and, therefore, their hand-to-mouth activity persists longer. In 1 study of lead poisoning in children with PDD, including autism, the children with PDD were older than the children with lead poisoning without PDD.[24] The same study also found that "despite close monitoring, state-mandated environmental inspection and prompt lead hazard reduction or alternative housing," children with PDD were more likely to be re-exposed to lead.[24]

We report time to decline to <10 μg/dL among a large cohort of children with BLLs ≥45 μg/dL. One study found that in children with peak BLLs <30 μg/dL, time to decline to <10 μg/dL was 24 months for BLLs of 25-29 μg/dL and 20.9 months for BLLs of 20-24 μg/dL.[25] The longer median time to decline for children receiving more chelations probably indicates more extensive exposures over a longer period of time or re-exposures. In 32% of cases, the initial BLL ≥45 μg/dL was not the peak BLL. This highlights the need for long-term surveillance of these children to identify re-exposures quickly. In contrast, the neonates limited time of exposure (<9 months) and removal from their sources at birth (their mothers) probably accounts for their shorter time to decline.

The demographic comparison data we used to compare our sample with the NYC population (age, race, borough, poverty status, and housing age) were not available on an individual-level basis, preventing us from performing multivariate analyses. Furthermore, socioeconomic status data for cases was not available; thus, Medicaid enrollment and census tract poverty level were used as proxies.

Risk behavior data, such as consumption of lead paint and imported products, were not available to compare with the general NYC population younger than 18 years. There may have been significant underreporting of risk factors, such as eating paint or the use of imported products. Some questions were not asked before 2006, limiting our sample size for those questions. In addition, the self-reported risk factors and developmental delay data may have limited validity because the RA form was a screening tool for identifying potential lead exposures for the purposes of intervention and education, rather than a highly validated instrument developed specifically for research. ■

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