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Raj P. Warrier

Jan Waisanen

Sam R. Kottamasu

Sayeed Sultana

K. Ratnakar Kini

See next page for additional authors

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Childhood Lead Poisoning

Authors

Raj P. Warrier, Jan Waisanen, Sam R. Kottamasu, Sayeed Sultana, K. Ratnakar Kini, William L. Kestenberg, John Strauthers, and Craig Foreback

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Childhood Lead Poisoning

Raj P. Warrier, MBBS, MD, DCH,* Jan Waisanen, RN,[†] Sam R. Kottamasu, MD,[‡] Sayeeda Sultana, MD,[§] K. Ratnakar Kini, MD,^{||} William L. Kestenberg, MD,[¶] John Strauthers, BS,[#] and Craig Foreback, PhD**

Although lead encephalopathy is rare, lead intoxication continues to be a significant problem in Detroit. Neuropsychological effects may occur even at low levels of lead. Since treatment of lead poisoning consists of a painful series of injections, it is imperative that more attention be given to

Since its discovery about 5,000 years ago, lead has become an increasingly essential part of daily life. Some current uses of this metal include gasoline, food cans, lead acid batteries, and electronic gadgets. Although lead has no physiological role in the body, subclinical effects of lead poisoning, particularly central nervous system effects, have been detected in children having minimally elevated blood levels (1,2). Each year about 7% of the children tested are found to have undue lead absorption, with about 0.4% of them being in the highest risk category (3-5). Our experience with lead poisoning in children during a three-year period at Henry Ford Hospital is reviewed herein.

Materials and Methods

At Henry Ford Hospital, lead levels were measured by atomic absorption spectrophotometry, and erythrocyte protoporphyrin (EP) concentration (normal: 20 µg/dL) was measured fluorometrically (6). Lead levels were measured by the Detroit Health Department Laboratories by anodic stripping voltammetry. Patients with beta-thalassemia trait or sickle-cell hemoglobin were identified by hemoglobin electrophoresis. Serum ferritin level was measured by radioimmunoassay (7). The criteria for diagnosing anemia included 1) hemoglobin value below 11.0 g/dL for children between 0.5 to 6 years and 2) hemoglobin value below 11.5 g/dL for children between 6 and 12 years (normal: 12.0 g/dL; range: 10.5 to 14.0). Iron deficiency was defined as ferritin level below 10 μ g/L (normal: 10 to 300 μ g/L) and/or transferrin saturation below 16% (normal: 20% to 55%) (8,9). Radiographs of the abdomen and long bones were also performed on these children.

the prevention of this disease. To confirm the magnitude of the problems, our experience with lead poisoning during a three-year period at Henry Ford Hospital's Pediatric Department is reviewed.

Results

The Pediatrics Department at Henry Ford Hospital serves as a tertiary referral center for the Detroit Lead Control Program, which screened 53,896 children from Jan 1, 1980, to Dec 31, 1982. Elevated lead (Pb) or EP levels were found in 2,045 (3.78%) of the children. Ninety-eight cases (0.18% of the total, or 4.7% of the positive cases) were Class IV by the risk categorization (Table). During the three-year period at the hospital, 55 children, including patients in the Detroit Lead Control Program and those from other referral sources, underwent chelation therapy.

Of the 55 children treated for lead poisoning during the three-year period, 46 (84%) patients were categorized as Class IV and nine (16%) as Class III patients, based on the Centers for Disease Control (CDC) criteria for risk classification of children with lead poisoning. Multiple chelations were required in four cases, including five chelations in one instance.

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^{*}Louisiana State University Medical Center, New Orleans, Louisiana.

Formerly Department of Pediatrics, Henry Ford Hospital

[†]Outpatient Division, Henry Ford Hospital

[‡]Department of Radiology, Division of Pediatric Radiology, Henry Ford Hospital

[§]Detroit Health Department. Formerly Plymouth Center, Henry Ford Hospital ^{ID}Department of Pediatrics, Henry Ford Hospital

¹Mt Carmel Hospital, Detroit, Michigan. Formerly University of Michigan Medical School

[#]Detroit Health Department, Lead Poison Control Unit

^{**}Department of Pathology, Division of Chemistry, Henry Ford Hospital

Address reprint requests to Dr Warrier, Department of Pediatrics, Louisiana State University Medical Center, 1542 Tulane Ave, New Orleans, LA 70112.

| | Risk Classification of Asymptomatic Children, Adapted from Centers for Disease Control (3) | | | | |
|--------------------|---|--|--------|---------|------|
| Blood Lead (µg/dL) | Test Results | Erythrocyte Protoporphyrin (μ g/dL Whole Blood) | | | |
| | | <49 | 50-109 | 110-249 | >250 |
| | <29 | 1 | 1a | 1a | EPP* |
| | 30-49 | 1b | П | Ш | 111 |
| | 50-69 | xx^{\dagger} | 111 | 111 | IV |
| | >70 | xx | xx | IV | IV |
| | | | | | |

Table Risk Classification of Asymptomatic Children, Adapted from Centers for Disease Control (3)

*Erythropoietic protoporphyria. Iron deficiency may cause EP elevations to 300 $\mu g/dL,$ although rarely.

[†]xx = Combination of results not generally observed in practice

The children's ages ranged from 16 months to 108 months (m: 33.6 months). Existing neurological problems such as mental retardation and seizure disorder were noted in four (7.2%) children, and clinical symptoms attributable to lead poisoning were noted in seven (13%). Pica was present in 46 (84%) of the patients. Radiopaque paint chips in the intestinal tract were noted on abdominal radiographs in 21 (39%) children. Radiographs of the long bones showed thick, transverse, radiodense, metaphyseal bands in 22 (40%) of these cases.

Lead levels ranged from 45 μ g/dL to 111 μ g/dL (m: 68 μ g/dL), EP levels from 110 μ g/dL to 801 μ g/dL (m: 290 μ g/dL), and hemoglobin from 6.8 g/dL to 13.5 g/dL (m: 10.6 g/dL). Mean corpuscular volume varied from 54 fL to 85 fL (m: 73 fL). The serum ferritin range was from 8 μ g/dL to 40 μ g/dL (m: 20 μ g/dL). Forty-four (80%) of these children were tested for iron deficiency; 39% were found to be iron deficient by at least one of the criteria for iron deficiency, and 50% were anemic. Sickle-cell trait was detected in two instances, sickle beta-thalassemia in one case, beta-thalassemia minor in two cases, and hemoglobin AC trait in one instance.

Discussion

Current research in lead poisoning is centered on the prevention and early detection of subtle neuropsychological defects, the effects of lead on various enzyme systems, and the interaction of nutrition with lead toxicity. Lead encephalopathy may be rare, but the risk of lead poisoning is not solely restricted to residents of old, dilapidated housing (10) or to urban migrant families.

All the children in our study were from the Detroit and Highland Park inner city areas. Sachs et al (11) reviewed their experience with 1155 children in an urban setting and noted pica in 74% of their patients. Radiodense, meta-physeal lines in long bones were detected in 82%. In our patients pica was present in 84% and metaphyseal lines in 40% (Fig 1).

Evidence of paint chips in the intestinal tract (Fig 2) was noted radiologically in 39% of the children in our study, as compared to 17% of those with lower levels of lead and 100% of those with blood lead levels approaching 100 μ g/dL in the Sachs et al series (11).

Lead affects metabolism through its action on enzyme systems. The hematopoietic effect of lead is a sensitive indicator of the metal's metabolic effects at cellular and functional levels. Microcytic, hypochromic anemia with basophilic stippling of the red blood cells is often the clinical end-point of hematological changes. Basophilic stippling is not useful for determining exposure to lead because it is nonspecific; dose-response data are not yet available.

Elevation of EP levels is an early manifestation of hematopoietic effects that ultimately may induce anemia. Anemia accompanying lead poisoning involves a multitude of factors (12); Yip et al (13) noted a 30% incidence of anemia in lead-intoxicated children. Of the children in our study, 50% were anemic, and 44% were iron deficient. Cohen et al (14) believed that if iron deficiency and thalassemia were excluded, microcytic, hypochromic anemia would be uncommon in lead poisoning.

Multiple chelations were required in four of our patients, including five chelations for one patient. All the children tolerated the chelations with calcium EDTA and British antilewisite administered in accordance with the CDC protocol (3). These drugs were administered with procaine hydrochloride (Elkinson-Sinn, Cherry Hill, NJ) and hyaluronidase to try to decrease local pain and inflammation. The lack of abscess formation or severe restriction of physical activity may indicate potential benefit with use of these drugs; however, the long and painful series of injections is a major drawback of the treatment program. Chelations had to be repeated in 7% of the cases because lead levels did not decrease to low-risk categories. A recurrence rate of 3% was noted in the Sachs et al experience.

Despite preventive measures and screening programs, lead poisoning continues to be a significant problem. Neuropsy-

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Fig 2 Scattered radiopaque particulate material in intestinal tract consistent with pica.

chological changes may result from chronic low-level lead poisoning (15,16), and significant behavioral and intellectual defects may occur, especially in young, growing children. Deficits in fine motor function, attention disorders, learning disabilities, or emotional disturbances that impair young children's progress at school and in society may all be manifestations of subclinical chronic lead poisoning (17), as a consequence of prenatal and neonatal lead exposure (18). It remains imperative for the pediatric team to continue to work closely with the community, government, and social workers in the prevention, early detection, and appropriate treatment of lead poisoning in children.

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chological, hematological, and other complications continue to arise because of low-level chronic lead poisoning. Significant biochemical alterations with subtle neuropsy-

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