Lead Poisoning in a Child After a Gunshot Injury

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Lead poisoning is a common disease that, if not detected, can lead to developmental delay and other serious sequelae. We report the case of a child with retained intracranial lead pellets from a gunshot injury, in whom elevated blood lead levels were detected approximately 1 year after the injury. No environmental source of lead was found, and a twin sister living in the same dwelling had considerably lower lead levels. The patient's lead levels diminished after each of four courses of chelation, but rebounded each time to potentially toxic levels after termination of therapy. Physicians should be particularly alert in screening for elevated lead levels in children with retained bullet fragments. In patients in whom removal of the bullet fragments is impractical, the potential risks of long-term chelation therapy must be weighed against the risks of lead toxicity.
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Lead poisoning is a reportable disease in the United States, affecting nearly 700,000 American children between the ages of 6 months and 5 years.[1] The usual source of environmental exposure to lead is paint in old houses that are dilapidated or undergoing renovation.[2] Few cases of elevated lead levels from retained bullet fragments have been reported in the literature. To our knowledge, only one other case has been described in a child.[3] We describe the case of a boy who has had a consistently elevated blood lead level after a gunshot wound to the head.

Case Report

On October 17, 1988, a 4-year-old boy was hospitalized after accidently shooting himself while playing with his grandfather's gun. The entry site of the bullet was the right maxillary sinus, with fragments projecting into the right frontoparietal area. He underwent two craniotomies for debridement and removal of bullet fragments. A postoperative computed tomography (CT) scan showed multiple retained bullet fragments in the paranasal sinuses and the right orbit. The patient had left-sided hemiparesis and was blind in the right eye. After a prolonged hospitalization, he was transferred to a rehabilitation hospital, where he continued to receive intensive physical and speech therapy.

At a follow-up office visit in November 1989, the patient had recovered sufficient left-sided strength to be ambulatory. He was asymptomatic and a physical examination revealed no abnormal findings other than some residual left-sided weakness and blindess in the right eye. Developmental examination showed modest delays in language and attainment of other developmental milestones. During routine screening, the patient's blood lead and free erythrocyte protoporphyrin (FEP) levels were found to be elevated (2.00 μmol/L [41 μg/dL] and 2.04 μmol/L [115 μg/dL], respectively). The chronology of the patient's lead levels is shown in Table 1. Three years before the injury, a screening test had shown an FEP level of 0.06 μmol/L (34 μg/dL). A moderately elevated FEP level of 0.90 μmol/L (51 μg/dL) and a lead level of 2.05 mmol/L (42 μg/dL) were noted a year later. The patient's lead level 9 months before the gunshot injury was 1.15 μmol/L (24 μg/dL).

When routine screening tests performed after the gunshot wound revealed an elevated lead level, the patient was admitted to the hospital for chelation therapy with edetate calcium disodium (CaEDTA). Laboratory tests at admission showed a lead level of 2.15 μmol/L (45 μg/dL), an FEP level of 2.24 μmol/L (126 μg/dL), a hemoglobin level of 123 g/L (12.3 g/dL), a mean corpuscular volume of 76 fL, an iron level of 8.3 μmol/L (46 μg/dL), and a total iron-binding capacity of 54 μmol/L (300 μg/dL) with 15% saturation. An abdominal radiograph did not show any lead chips. The patient was living in a recently painted apartment building with his mother,

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Table 1. Chronology of Events and Corresponding Blood Lead and Free Erythrocyte Protoporphyrin (FEP) Levels in a Child

Date	Event	Lead μmol/L (μg/dL)	FEP μmol/L (μg/dL)
9/20/85	Screening test	—	0.60 (34)
09/30/86	Screening test	2.05 (42)	0.90 (51)
01/12/87	Screening test	1.15 (24)	—
10/17/88	Gunshot injury	—	—
11/06/89	Screening tests	2.00 (41)	2.04 (115)
11/28/89	Hospital admission, CaEDTA chelation (5 d)	2.15 (45)	2.24 (126)
12/15/89	Follow-up tests	1.80 (37)	1.80 (102)
3/19/90	Follow-up tests	1.40 (29)	0.52 (29)
9/11/90	Follow-up tests	2.40 (50)	4.08 (230)
9/24/90	Hospital admission, CaEDTA and dimercaprol chelation (5 d)	2.15 (45)	4.54 (256)
9/30/90	End of chelation therapy	0.75 (16)	—
10/12/90	Follow-up tests	2.25 (47)	3.72 (210)
11/02/90	D-penicillamine chelation (8 wk)	—	—
11/13/90	Ongoing chelation therapy	1.30 (27)	—
1/18/91	Follow-up tests	2.30 (48)	0.86 (49)
7/08/90	Follow-up tests	1.85 (38)	0.66 (37)
9/09/91	Follow-up tests	1.95 (40)	0.78 (44)
9/20/91	Succimer chelation (19 d)	—	—
11/04/91	Follow-up tests	1.05 (22)	0.88 (50)

twin sister, maternal aunt, and her 1-year-old son. Inspection of the apartment by the public health department lead team revealed no environmental source of lead exposure. The patient was discharged after 5 days of chelation therapy.

At a follow-up examination in December 1989, the child's serum lead level was 1.80 μmol/L (37 μg/dL) and his FEP level was 1.80 μmol/L (102 μg/dL). In March 1990, lead and FEP levels were 1.40 μmol/L (29 μg/dL) and 0.52 μmol/L (29 μg/dL), respectively. The patient was lost to follow-up until August 1990. Results of repeat testing at that time showed a lead level of 2.40 μmol/L (50 μg/dL) and an FEP level of 4.08 μmol/L (230 μg/dL). He was living in the same apartment, which the public health department again determined to be free of any source of environmental lead. He was admitted to the hospital in September 1990. The patient was asymptomatic, and physical examination findings were normal except for the residual neurologic deficits from the gunshot wound. An abdominal radiograph did not show any lead chips. Laboratory tests at the time of admission showed a lead level of 2.15 μmol/L (45 μg/dL), an FEP level of 4.54 μmol/L (256 μg/dL), a hemoglobin level of 119 g/L (11.9 g/dL), and a mean corpuscular volume of 75 fL. Chelation therapy with CaEDTA and dimercaprol (BAL) was performed. The patient's serum level decreased to 0.75 μmol/L (16 μg/d) by the end of chelation therapy. Two weeks after the patient was discharged from the hospital, his lead and FEP levels were 2.25 μmol/L (47 μg/dL) and 3.72 μmol/L (210 μg/dL), respectively. The patient remained asymptomatic, and subsequent abdominal radiographs did not show any lead chips. He was started on an 8-week course of outpatient chelation with D-penicillamine (PCA) in November 1990. After 2 weeks of chelation therapy, the patient's serum lead level had decreased to 1.30 μmol/L (27 μg/dL). Follow-up laboratory test 3 weeks after terminating outpatient therapy showed lead and FEP levels of 2.30 μmol/L (48 μg/dL) and 0.86 μmol/L (49 μg/dL), respectively. The patient was lost to follow-up until July 1991, when repeat testing showed a lead level of 1.85 μmol/L (38 μg/dL) and an FEP level of 0.66 μmol/L (37 μg/dL). Persistently elevated serum lead and FEP levels (1.95 μmol/L [40 μg/dL] and 0.78 μmol/L [44 μg/dL], respectively) were found in September 1991. At that time, he was treated with a 19-day course of succimer (300 mg orally, twice a day). One month after outpatient oral chelation therapy, his blood lead level was 1.05 μmol/L (22 μg/dL) and his FEP was 0.88 μmol/L (50 μg/dL).

Of interest, the patient's twin sister living in the same household had significantly lower screening levels of lead. Her FEP was 0.28 μmol/L (16 μg/dL) in April 1988, and in January 1989, it was 0.46 μmol/L (26 μg/dL). Repeat tests in September 1990 showed a lead level of 0.95 μmol/L (20 μg/dL) and an FEP level of 0.24 μmol/L (14 μg/dL).

Discussion

Lead intoxication is a clinically important finding in children. Lead poisoning causes nonspecific symptoms of anorexia, vomiting, constipation, abdominal pain, and weight loss. It can also result in anemia and renal toxicity. In severe cases, it can cause an acute encephalopathy with lethargy, stupor, coma, convulsions, and persistent vomiting.[4] Chronic exposure to low levels of lead may lead to learning deficits, changes in behavior, short stature, and poor weight gain.[5]

Lead-based paint is currently the most common cause of lead intoxication in children. Other sources of enviromental lead exposure include contaminated drinking water, food, or soil, as well as inhalation of lead from household dust, air contaminated by industrial wastes, and leaded gasoline fumes. Retained bullet fragments and pellets are a rare, but previously reported source of

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lead toxicity in adults. A recent study of 23 children with retained bullet fragments from gunshot wounds found no clinical or laboratory evidence of resultant lead toxicity.[6] The one previously reported case of lead intoxication in a child resulting from a bullet wound involved multiple retained metal fragments in the abdomen, pelvis, and spinal canal.[3]

Reports of lead poisoning from retained bullet fragments in adults have identified several risk factors for toxicity.[3,7–11] First, the location of the fragments is an important factor in subsequent poisoning. In most reported cases, the metal fragments were in contact with the synovial fluid of a joint that the bullet penetrated. It has been theorized that the acidic synovial fluid acts as a solvent, dissolving the lead, which is then absorbed by the blood.[12] This process is facilitated by frictional forces in the joints. In contrast, bullet fragments lodged in soft tissue may become covered by an avascular fibrotic membrane, which prevents lead dissolution and subsequent increases in blood lead levels. Second, multiple, small pellets have a larger combined surface area than a single bullet of comparable lead content. Toxicity from multiple pellets is more common and usually has an earlier onset than that caused by a bullet. Our patient had an increased serum lead level about 1 year after the injury. Third, the duration of exposure is positively associated with toxicity. Finally, the type of bullet and its metal content is also a risk factor. Bullets with a high lead content and those without a coating are more likely to cause lead toxicity. There are no clear guidelines on screening adults with retained bullet fragments. Lead poisoning should be suspected in such patients, however, particularly if they have any of the risk factors described above.

If elevated lead levels are found in patients with retained bullet fragments, removal of the lead source is the first line of treatment. If surgical removal of retained bullets is not practical, however, chelation therapy is recommended for those with elevated blood lead levels. Chelation before surgery is indicated for all patients, since surgery may cause mobilization of lead stores and result in acute poisoning postoperatively.[8] In this particular case, the intracranial location of the retained bullet fragments made the potential morbidity from their removal unacceptable. The patient appeared to be asymptomatic in spite of elevated lead levels; however, it is difficult to separate developmental delay caused by the gunshot wound and debridement from delay caused by lead toxicity.

The likelihood that the bullet fragments were the source of the elevated lead levels is strengthened by the considerably lower blood lead levels in the patient's twin sister, who lived in the same home during the time in which the patient had elevated levels. The one elevated lead level recorded 2 years before the gunshot injury raises the possibility that the patient had previously been exposed to another source of lead, although the subsequently lower lead level 9 months before the injury and the inability of an experienced public health team to find an environmental lead source during multiple visits to the patient's home make it unlikely that another source was responsible for the subsequent elevated lead levels. In addition, after the patient's repeated elevated blood lead levels, his mother remained alert to alternative sources of lead exposure, particularly those to which the patient's twin was not exposed. The patient's mother did not observe any other potential source.

The long-term implications of chronic chelation therapy are not well described,[13] but may be preferable to the risks of continued high blood lead levels. Prolonged use of CaEDTA has been associated with toxicity (generally mild) and has caused deaths due to renal failure.[14] A small series of patients with Wilson's disease, who had been given penicillamine for copper toxicity, tolerated chelation courses ranging in length from 9 to 13 years, with improvement in their primary disease. In spite of initial sensitivity reactions, no side effects from long-term chelation therapy were recognized.[15] The first three chelation courses were successful in temporarily reducing the blood lead levels in our patient; however, each time, the amount of lead in the patient's blood returned to a potentially toxic level after terminating therapy. Furthermore, 1 month after completing the last chelation therapy with succimer, the patient's blood lead level remained relatively elevated.

Succimer (also known as 2,3-dimercaptosuccinic acid [DMSA]) is a new oral chelating agent recently approved by the Food and Drug Administration. It is indicated for treatment of children with blood lead levels greater than 2.15 μmol/L (45 μg/dL). Succimer has been shown to be effective in reducing blood lead levels,[16] and apparently has less toxicity and less effect on urinary excretion of calcium, copper, iron, and zinc than CaEDTA.[16] There is limited clinical experience using succimer, however, and no studies of the safety of its long-term use have been reported.

Increasing recent consensus about the toxic effects of even low doses of lead[17] strengthens the argument for treating our patient, in spite of a lack of evidence that chelation therapy for low blood lead levels is beneficial.[18] Needleman et al[5] found that school performance and cognitive function remained below average at 11-year follow-up of young adults with childhood histories of elevated lead levels.[5] The continued use of short-term chelation therapy or prolonged courses of outpatient chelation therapy remain a therapeutic option for our patient, but careful monitoring for signs of toxicity will be necessary.

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Recently, the Centers for Disease Control issued new guidelines for prevention and management of lead poisoning in children.[19] Virtually all preschool children in the United States are considered at risk for lead poisoning and should be screened. The new recommendations emphasize the importance of using venous lead levels for diagnosis (as opposed to capillary blood samples or protoporphyrin screening tests). Based on new scientific evidence, a blood lead level greater than 0.50 μmol/L (10 μg/dL) is now considered unsafe. Blood lead levels of 0.50 μmol/L to 0.70 μmol/L (10 μg/dL to 14 μg/dL) are considered borderline and should be repeated every 3 to 4 months. For lead levels equal to or greater than 0.70 μmol/L (15 μg/dL), individual management and efforts should be undertaken to identify and eliminate potential sources of lead. Children with lead levels greater than 2.15 μmol/L (45 μg/dL) should receive chelation therapy in addition to detailed environmental assessment.

In conclusion, physicians should be alert to the possibility of lead poisoning in patients with retained metal fragments. It would be reasonable to perform screening tests at regular intervals for several years on children who have been injured by a bullet. Diagnosis and early treatment of elevated lead levels in children are important to prevent irreversible cognitive and behavioral delays.

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