ARTICLES continued

Lead Poisoning in a Child With a Gunshot Wound

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ABSTRACT. Lead poisoning has infrequently resulted from gunshot wounds with retained lead particles in adults. This has not been previously found in children. The case of an 8-year-old boy in whom lead poisoning developed soon after a gunshot wound is reported. The child had no symptoms directly related to lead poisoning, but he received chelation therapy. The case demonstrates the need to consider lead poisoning in children with retained particles following gunshot wounds. *Pediatrics* 1986;77:413–416; *lead poisoning, gunshot wound, injury, bullet, chelation.*

Lead poisoning from exposure to lead-based paint is still a common occurrence in the United States. Lead exposure may also result from automotive emissions into the air.[1] In addition, lead poisoning may result from the use of pottery that is not fired at high enough temperatures, from burning battery casings,[2] and from ingestion of lead-containing items such as curtain weights.[3] In adults, there have been infrequent reports of lead poisoning that occurred after gunshot wounds with retained lead particles.[4–10] This has not been previously reported in children.

We report the case of a child in whom evidence of lead toxicity developed shortly after sustaining a serious wound from a shotgun blast. The case emphasizes the need to consider lead poisoning in children who have retained bullets or lead pellets after injury with firearms.

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CASE REPORT

An 8-year-old boy was brought to the emergency department of the Children's Hospital of Philadelphia and was found to be paraplegic and in shock from hypoperfusion due to a gunshot wound to the right paraspinal area. He was injured with a shotgun that fired uncoated lead birdshot according to ballistics experts at the Philadelphia police department. The child was stabilized in the emergency department and was taken to the operating room for exploratory laparotomy. In the operating room, he required a right nephrectomy, repair of a splenic laceration, and debridement of the liver with resection of part of the inferior portion. A large right paraspinal entrance wound was explored and debrided, and several thoracic vertebral bodies were noted to be fractured. Radiographs revealed multiple small fragments of heavy metal throughout the abdomen and pelvis (Fig 1), which were not removed intraoperatively.

Following a brief stay in the intensive care unit of the Children's Hospital of Philadelphia, the child was transferred to the Children's Seashore House Annex in Philadelphia for rehabilitation. The child was paraplegic at a level of L-2, and this did not improve postoperatively. A computed tomographic scan revealed fragments of metal in the spinal cord (Fig 2). Also, postoperatively the child had prolonged feeding intolerance with frequent vomiting. Evaluation of the gastrointestinal tract revealed no pathologic condition, and the emesis was believed to be nonorganic in origin. However, the possibility of lead toxicity was considered, and thus, 2 months after his injury, a lead level was obtained. The results disclosed the following values: lead, 40 μg/dL; free erythrocyte protoporphyrin (FEP), 88 μg/dL. A review of the child's medical records revealed much lower lead levels when he was a toddler (Table). Wrist and ankle radiographs at

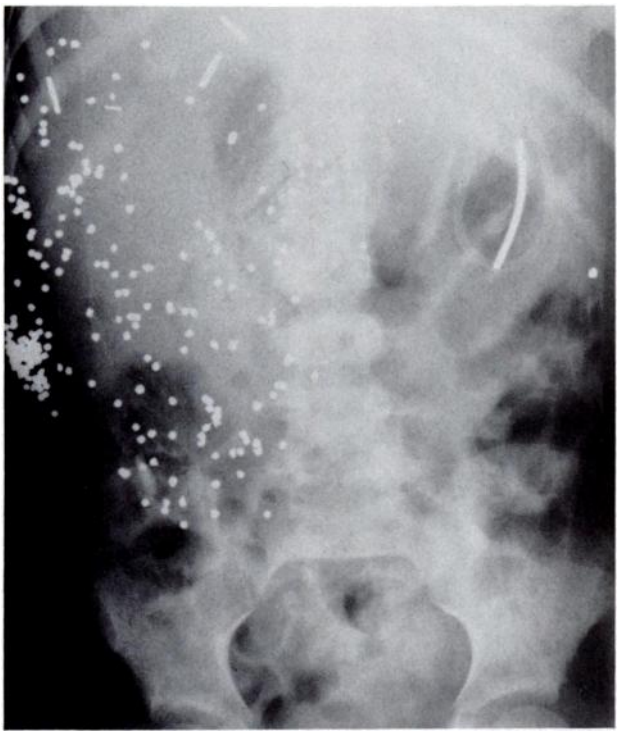

Fig 1. Abdominal radiograph with multiple small fragments of heavy metal (birdshot). (Few surgical clips are seen in right upper quadrant).

that time were without long bone evidence of lead poisoning.

Eleven weeks after the injury, laboratory studies were repeated and the lead level was found to be 60 μg/dL and FEP 149 μg/dL. Chelation therapy was started with edetate disodium calcium ($CaNa_2$ EDTA), 250 mg, given intravenously every six hours for five days. During the first 24 hours of chelation, the child excreted 389 μg of lead, and during the last day of chelation this was reduced to 137 μg of lead. Following chelation, the blood lead level was dramatically reduced, but 2 months afterward, it increased to 31 μg/dL (Table).

The child received intensive physical and occupational therapy, as well as psychiatric intervention for 6 months. His feeding problems slowly resolved, and he exhibited no additional symptoms related to lead poisoning. He was not treated with oral chelating agents and was discharged from the hospital with plans for outpatient follow-up.

DISCUSSION

Injuries involving firearms are increasing dramatically in this country, and young children are now often victimized. However, lead toxicity from a gunshot wound has not been previously reported in young children. Lead poisoning from retained lead bullets has been suspected since the late 19th

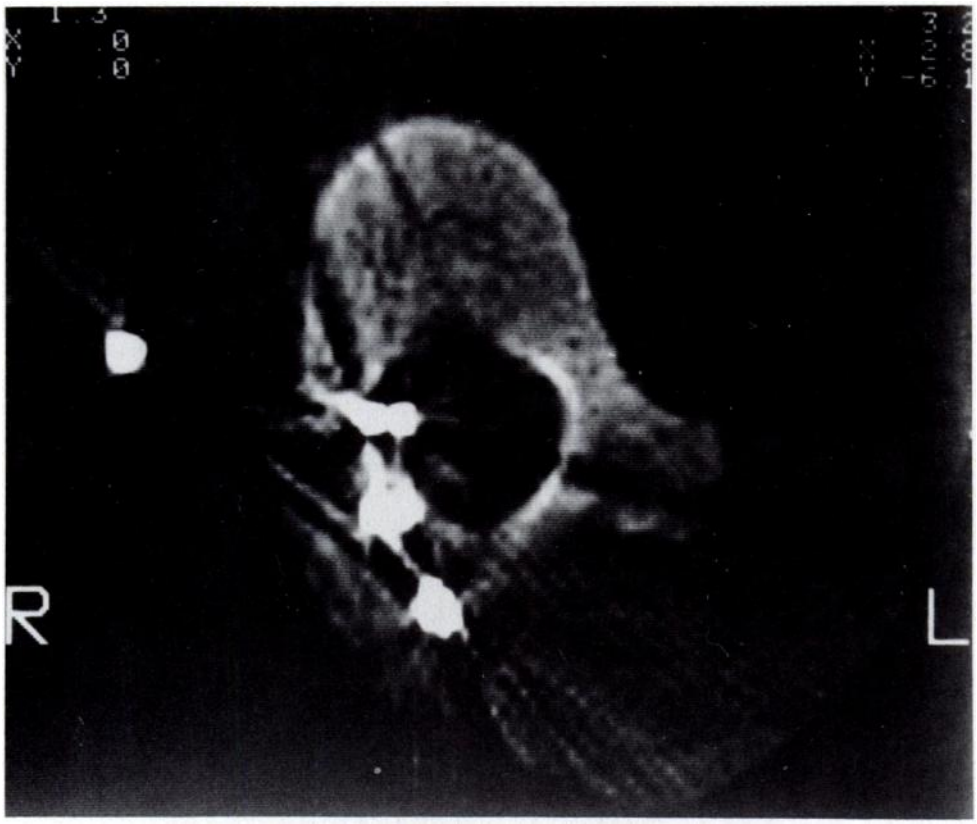

Fig 2. Computed tomographic scan reveals metallic fragments (birdshot) in spinal canal.

TABLE. Lead Levels in a Child With a Gunshot Wound

Date	Lead Levels (μg/dL)	Free Erythrocyte Protoporphyrin (μg/dL)
3/20/78 (age 23 mo)	23	50
4/2/81 (age 5 yr)	29	12
7/2/84 (gunshot wound) (age 8 yr)		
9/4/84	40	88
9/27/84	60	149
10/5/84 (chelation begun)		
10/9/84	25	117
10/17/84	18	96
11/14/84	29	139
12/14/84	31	136

century,[4] but this could not be proven until laboratory tests became available to measure blood and urine lead levels. In 1976, Switz et al[5] verified the first case in the English literature of lead poisoning due to a retained lead bullet. Since then, there have been scattered reports of adults with lead poisoning due to injury with bullets, buckshot, and shrapnel.[5-10]

In the adult patients reported previously, a variety of signs and symptoms of lead poisoning has been noticed. Most presented with anorexia, abdominal pain, vomiting, and anemia, and in several encephalopathy[6,9,10] and seizures[10] developed as lead levels increased to 350 to 500 μg/dL.[9,10] Peripheral neuropathy (a rare symptom in children with lead poisoning) and fatalities have also been reported in adults with previous gunshot wounds.[6,10] Our patient never exhibited neurologic signs of lead toxicity because of early chelation therapy. It is also unlikely that his vomiting was due to lead poisoning because it persisted long after chelation therapy. In addition, many children tolerate higher lead levels without gastrointestinal symptoms.

Several authors[5,8,9] have stressed that the surface area of retained lead particles plays a critical role in the development of lead poisoning following a gunshot wound. Thus, although a retained single bullet may take 20 to 40 years to produce symptoms (average 17 years), shrapnel injuries may produce lead poisoning in an average of 10 years in most cases.[9] Remarkably, buckshot injuries result in lead poisoning after much shorter intervals of two days to 2 years (average 8 months.)[9] Our patient had laboratory evidence of lead toxicity within 2 months of his injury. This rapid development may well have been due to the tremendous load of birdshot (large surface area) in his body as noted on his initial x-ray films (Fig 1). It was impossible to remove all such particles even with wound debridement and liver resection.

The location of the retained lead particles also plays an important role in the development of lead poisoning.[5,6,8-10] Because many bullets lodge in soft tissue where they become encapsulated in dense fibrous tissue, they are not likely to cause lead poisoning.[5-8] However, in almost all previous reports of lead poisoning due to bullet wounds, the bullets were lodged in joint spaces. It is believed that chronic bathing in synovial fluid dissolved the lead which was then mobilized into the vascular system.[4,10] Also, bullets in joints are susceptible to joint motion, and frictional forces may increase the rate of dissolution.[5,8,9] In a few cases, fragments were retained in the intervertebral disk space where hypovascularity would predictably inhibit the mobilization of lead. However, in these cases, communicating cysts with retained lead particles developed and again provided a solvent for the lead.[8,10] Our patient was found on computed tomographic scan to have at least one small fragment of birdshot in his spinal canal (Fig 2). It is conceivable that CSF continuously bathes this particle(s) and increases the lead level in his vascular system. However, because neurologic symptoms of lead poisoning have not been found in this child, it is unlikely that there is a significant contribution of lead from the CSF.

Obviously, the length of time that one is exposed to lead increases the risk of poisoning.[8,9] Because lead poisoning after gunshot wounds often takes years to develop, it is unlikely to be seen in the pediatric population. Still, our patient may be at risk for lead poisoning for the rest of his life.

Finally, the type of ammunition involved is important in the development of lead poisoning. Some bullets contain lead or lead alloys with a metal jacket or coating, whereas others are uncoated. Those that are uncoated have a greater surface area of lead for dissolution. The particles recovered from our patient were uncoated lead birdshot fragments which are smaller than that of buckshot. This type of ammunition is commonly used for hunting small fowl and can be purchased cheaply in any gun store.

The treatment of lead poisoning due to retained lead particles from gunshot wounds is similar to that due to any type of lead exposure. Ideally, the source of lead should be removed. However, if surgical removal is attempted, it should be done cautiously. Linden et al[10] warn that lead in suspension may surround the tissues containing the bullet and this toxic material could spill. They recommend chelation prior to surgery. Several authors have recommended chelation with dimercaprol (BAL) and $CaNa_2$ EDTA, as well as oral penicillamine.[5-10]

Our patient received only $CaNa_2$ EDTA because he was asymptomatic and had class III lead poisoning. Oral penicillamine has not been tried in this child as yet because of its potential renal toxicity and his previous nephrectomy.

In summary, lead poisoning should be considered in any child with retained particles from a gunshot wound, especially if there are numerous fragments or bullets located near a joint or bursa. With domestic violence on the increase, children are likely to be victims more often, and lead poisoning should be diagnosed before symptoms develop. Diagnostic testing and treatment for such lead poisoning is the same as for any other type of inorganic lead toxicity.

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ON KILLING THEORIES

Human knowledge is a special case of animal knowledge. From amoeba to Einstein learning takes the same course: it starts from a problem caused by the clash between expectations and experience, proceeds to a solution, and then to the testing of that solution. With the emergence of science, failure need no longer be fatal: we can kill off our theories, instead 'of being killed off ourselves.'

Submitted by Student

From Magee B: Philosophy and the Real World. An Introduction to Karl Popper. La Salle: Open Court, 1985.