

Case Report

Elevated Blood Lead Levels Resulting from the Ingestion of Air Rifle Pellets

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Abstract

There have been numerous reports of lead poisoning resulting from the ingestion of foreign bodies. A case involving the ingestion of spent air rifle pellets is described. No clinical symptoms were observed, despite the fact that the young child exhibited elevated blood lead levels as high as 2.7 $\mu\text{mol/L}$ (56 $\mu\text{g/dL}$). X-rays of the child's abdomen confirmed the ingestion of the pellets. The patient was treated with laxatives, and the pellets were successfully passed over the course of the next few days. Prior to release from the hospital, the child's blood lead level had dropped to 1.7 $\mu\text{mol/L}$ (35 $\mu\text{g/dL}$).

Introduction

Lead ore is mined for a variety of uses, including the production of storage batteries, miscellaneous chemicals, paints, ammunition, and metal products (e.g., solder, pipes, sheet lead). As a result of smelting, refining, recycling, and manufacturing processes, lead is widely distributed throughout the environment. In fact, compounds containing lead can be found in virtually all parts of the environment, including plants, animal tissues, air, water, and soil. Perhaps the most significant historical source of atmospheric lead has been the combustion of leaded gasoline. Both the United States and Canada, however, have passed legislation prohibiting the use of leaded fuels.

The general population is exposed to lead and compounds containing lead through the inhalation of ambient air, consumption of drinking water, and oral ingestion of foods, dust, or soil. Workers in smelters and metal refining operations, battery manufacturing plants, radiator repair workshops, and indoor weapon firing ranges may be exposed to dust and vapors containing lead.

Lead has long been recognized as a potential hazard to human health. The effects of lead upon human health appear to be similar regardless of the route by which it enters the body (1). Lead is believed to affect almost every organ and system in

the body, in particular the kidneys and the central nervous system. Exposure to lead is especially hazardous to fetuses and young children. Exposure of pregnant women may lead to premature birth, low birth weight, and possibly even abortion. The toxic effects of lead upon young children include reduced intelligence, learning difficulties, behavioral disturbances, and reduced growth (2).

Children absorb and retain much higher amounts of lead than adults, and therefore, chronic low-level exposure is also significant. Children tend to ingest more dust than adults because of increased hand-to-mouth activity and generally lower concern about overall hygiene. Also, some children chew on articles covered by lead-containing paint. More recently, there was considerable controversy regarding the potential exposure of children to lead in plastic mini-blinds (3).

As a result of the toxicity of lead, numerous steps have been taken to reduce the potential exposure sources. For example, the use of leaded gasoline has been restricted in Canada since 1990 (2). This has drastically reduced the level of airborne lead, especially in urban centers. The use of lead pipes and solder in houses and buildings had previously been a significant source of lead in drinking water; however, the use of these materials for plumbing purposes has been curtailed. The use of lead-soldered cans for food packaging has also been virtually eliminated.

Despite these steps, there is still one route of exposure which governments cannot regulate: ingestion of non-food items containing lead. There have been numerous reports of cases of lead poisoning that resulted from the ingestion of lead-containing foreign bodies (4–10). There have also been a number of unusual cases reported including the ingestion of lead-based ceramic glazes (11), dried red lead (12), solder (13), a clothing accessory (14), and large quantities of lead shot (15). Lethal poisoning from the ingestion of lead is rare, but has been reported (16–19).

The ingestion of lead-containing foreign bodies has been found to result in complications associated with retention of the ingested object in the appendix. Lead shot has been retained in the appendix, leading to increased blood lead levels (20–22). Lead shot accidentally ingested during the consumption of hunted game meat has been reported to result in perforated appendicitis (23). In a study of 62 patients having

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retained lead shot in the appendix, no clinical evidence of lead poisoning was observed despite the fact that the number of retained shot pellets ranged from 1 to over 200 (24). In some cases involving ingestion of a foreign body containing lead, it was necessary to perform an appendectomy (25–27).

In this case report, we have presented both blood lead and x-ray evidence associated with an accidental poisoning that resulted from a young child ingesting lead-containing rifle pellets.

Case History

A father of three young children had been shooting an air rifle (pellet gun) in the basement of the family's home for a period of several months. During this time period, the spent pellets were collected and placed in an old 4-L milk container. The two older children, aged 2.5 and 4 years, had been playing in the basement one morning. At approximately 10 a.m., the 4-year old informed her parents that her younger sister had eaten some of the pellets. The parents immediately took the child to the local rural hospital where the physician on duty examined the girl. The physician noted that the girl was in no observable distress and in fact appeared to be quite content. A preliminary examination of the child revealed no signs of jaundice, pallor, or cyanosis. There was no discoloration of the mucous membranes and no observable respiratory difficulties. The child was alert and capable of moving all limbs in a normal, controlled manner.

The parents informed the physician that, to the best of their knowledge, this was the first known episode of ingestion of the pellets. X-rays of the abdomen taken at 11:07 a.m. on that day (day 1) revealed the presence of about 40 to 50 pellets mainly in the large bowel. A venous blood sample was drawn at 11:30 a.m. and submitted to the laboratory for lead analysis. The child was given laxatives in order to promote movement of the bowels. The child was held overnight in the hospital for observation.

The child's regular physician examined her early the next morning and found no related symptoms other than possibly mild fatigue. The child's abdomen was soft and not tender. No pellets had been passed, and the girl was transferred to the children's ward of a hospital in a nearby major urban center. A second blood sample was taken at 2:45 p.m. on day 2. The child's condition continued to be asymptomatic. Some of the ingested pellets were passed at approximately 7 p.m. on day 2.

The girl passed a number of pellets over the next two days. A third blood sample was collected almost 94 h after ingestion (day 5). An x-ray taken that same morning confirmed that virtually all of the pellets had been passed by the child. The parents requested that the child be discharged from the hospital and placed in their care. Because she did not exhibit any symptoms of discomfort and the pellets had been voided, the child was allowed to go home.

Materials and Methods

Specimens received were whole blood with EDTA as the anticoagulant. All samples submitted to the laboratory consisted of venous blood.

All blood lead analyses were performed using a Varian SpectrAA-300 atomic absorption spectrophotometer (AAS) equipped with a graphite furnace and Zeeman background correction system which was used in conjunction with a programmable Varian autosampler. The hollow cathode Pb lamp (part no. 56-101029-00) and the pyrolytically coated partitions (part no. 63100012-00) were purchased from Varian (Mulgrave, Australia). A lamp current of 5 mA was used for all analyses. The analytical resonance wavelength used was 283.3 nm with the slit width set to 0.5 nm. The graphite furnace program is shown in Table I. A programmable Varian autosampler was used to inject 5 μ L of sample into the graphite tube.

The AAS was calibrated using matrix-based standards that were prepared in whole blood. A three-point calibration curve was generated using whole blood standards that contained 0.17, 0.34, and 0.68 μ mol/L of lead. The response over this range of concentrations was found to be linear. Any samples above 0.68 μ mol/L were diluted to fall within the analytical working range (i.e., quantitation was performed by interpolation of the calibration curve). Separate dilutions were made at both 1:5 and 1:10 to verify the accuracy of the dilution process.

As part of the intralaboratory quality assurance protocol, a certified reference material was processed with each analytical run. The certified reference material consisted of a stable, lyophilized human whole blood control (Seronom trace elements control, batch no. 203056) purchased from Nycomed Pharma (Oslo, Norway). The lyophilized material was reconstituted with deionized, distilled water in accordance with the supplier's instructions. This yielded a whole blood control that contained 1.85 μ mol/L (38.5 μ g/dL) of lead.

Results

X-rays of the child's abdomen taken approximately 1 h after the suspected time of ingestion revealed the presence of an estimated 40 to 50 pellets, mainly in the large bowel (Figure 1). The blood sample collected approximately 1.5 h after the pre-

Table I. Graphite Furnace AAS Temperature Program*

Step no.	Temp (°C)	Ramp time (s)	Hold time (s)	Gas flow (L/min)
1	120	20	20	3.0
2	400	15	7	3.0
3	2600	1	7	0
4	40	13	2	3.0

* Extra steps were employed at the end of the furnace program to allow the graphite tube to cool slowly to 40°C and thus prevent spattering of the subsequent injection. Along with this modification, an extended dispensing rate (with hot inject, 40°C) was used to ensure that all the dispensed liquid would be contained within the platform cavity for subsequent temperature programming.

sumed ingestion time was found to contain 2.7 $\mu\text{mol/L}$ (56 $\mu\text{g/dL}$) of lead.

The second blood sample, which was taken approximately 29 h after ingestion (day 2), was found to contain 1.7 $\mu\text{mol/L}$ (35 $\mu\text{g/dL}$) of lead. According to the physician's report, a blood smear performed on a sample taken on day 2 did not indicate signs of lead toxicity. Analysis of the third blood sample, taken 94 h after ingestion (day 5), revealed that the blood lead level had remained at 1.7 $\mu\text{mol/L}$ (35 $\mu\text{g/dL}$). An x-ray taken the morning of day 5 indicated that virtually all of the pellets had been passed.

At the suggestion of the family's physician, the oldest child was also asked to return to the local hospital for an x-ray of the abdomen and to have a blood sample collected for lead analysis. The x-ray was negative for foreign bodies, and the lead level in the older sibling's blood sample was near the practical quantitation limit of the test (0.1 $\mu\text{mol/L}$ or 2 $\mu\text{g/dL}$). The youngest of the three children (5 months) did not spend any time in the basement and was not tested.

Discussion

X-ray evidence confirmed that the 2.5-year old child had ingested a number of air rifle pellets as suspected based upon information provided by her older sister. Lead encephalopathy has been reported in young children having blood lead levels in

excess of 3.4 to 4.8 $\mu\text{mol/L}$ (70 to 100 $\mu\text{g/dL}$). (19). In this particular case, the patient's blood lead level was found to be as high as 2.7 $\mu\text{mol/L}$ (56 $\mu\text{g/dL}$) and may in fact have maximized at a higher concentration. The blood lead levels in a series of three samples (2.7, 1.7, and 1.7 $\mu\text{mol/L}$) collected over a five-day period were all considerably higher than the intervention guideline of 0.5 $\mu\text{mol/L}$ (10 $\mu\text{g/dL}$) recommended by Health Canada (2). Based on previous tests performed on blood samples collected as a result of the lead in the miniblinds controversy, it was found that most children with general environmental exposure had blood lead levels at or below the practical quantitation limit of the analytical method (0.1 $\mu\text{mol/L}$ or 2 $\mu\text{g/dL}$). Information gathered by the attending physician during interview of the family at the time of admission to the hospital did not reveal any other apparent source of exposure to lead in the household. This was supported by the blood lead result for the eldest sister.

Despite the fact that elevated blood lead levels were found in all of the blood samples tested, no symptoms of acute poisoning were observed. In other cases involving the ingestion of lead-containing foreign bodies, symptoms such as abdominal pain, vomiting, constipation, and lethargy have accompanied elevated blood lead levels. Although these non-specific symptoms have been observed in young children whose blood lead levels exceed 2.4 to 3.4 $\mu\text{mol/L}$ (50 to 70 $\mu\text{g/dL}$), the absence of these conditions cannot be used in the diagnosis of suspected lead poisoning (19).

In a similar case reported in the literature, a 5.5-year old girl consumed a large quantity of tiny, round lead shot used in an ankle weight (15). The girl's blood lead level was found to be 2.7 $\mu\text{mol/L}$ (57 $\mu\text{g/dL}$) 13 h after ingestion. The highest blood lead level was found to be 3.8 $\mu\text{mol/L}$ (79 $\mu\text{g/dL}$) approximately 36 h after ingestion. Chelation therapy was employed for 19 days, at the end of which the child's blood lead level had dropped to 0.7 $\mu\text{mol/L}$ (14.3 $\mu\text{g/dL}$). Other than an initial incidence of vomiting and abdominal pain prior to being admitted to the hospital emergency department, no apparent symptoms of lead poisoning were observed.

Unfortunately, in the case described herein, the parents chose not to have further tests performed to monitor the child's blood lead level. It is therefore not known how rapidly the child's blood lead level decreased with time or how long this level remained above Health Canada's intervention guideline of 0.5 $\mu\text{mol/L}$ (10 $\mu\text{g/dL}$). It is also uncertain what impact, if any, a presumably one-time exposure to a significant quantity of lead would have in this case.

References

1. Agency for Toxic Substances and Disease Registry (ATSDR). ToxFAQs - Lead. www.atsdr.cdc.gov/tfacts13.html, November 2001.
2. Federal-Provincial Committee on Environmental and Occupational Health. Update of Evidence for Low-Level Effects of Lead and Blood Lead Invention Levels and Strategies—Final Report of the Working Group. Health Canada, Environmental Health Directorate, Ottawa, ON, Canada, 1994, pp 2–13
3. Health Canada. It's Your Health—Lead and Human Health.



Figure 1. X-ray of patient's torso revealing presence of ingested metallic foreign bodies.

- Health Canada, Minister of Supply and Services, Ottawa, ON, Canada, 1996, pp 1–4.
4. E. Blank and J. Howieson. Lead poisoning from a curtain weight letter. *J. Am. Med. Assoc.* **249**: 2176–2177 (1983).
 5. L.F. Durback, G.P. Wedin, and D.E. Seidler. Management of lead foreign body ingestion. *J. Toxicol. Clin. Toxicol.* **27**: 173–182 (1989).
 6. J.F. Wiley, II, F.M. Henretig, and S.M. Selbst. Blood lead levels in children with foreign bodies. *Pediatrics* **89**: 593–596 (1992).
 7. J.D. Sprinkle, Jr. and E.A. Hingsbergen. Retained foreign body: associations with elevated lead levels, pica, and duodenal anomaly. *Pediatr. Radiol.* **25**: 528–529 (1995).
 8. J.A. Fergusson, G. Malecky, and E. Simpson. Lead foreign body ingestion in children. *J. Paediatr. Child Health* **33**: 542–544 (1997).
 9. W.A. Gleason, Jr. Advisability of colonoscopy in the management of ingested lead poisoning. *Arch. Pediatr. Adolesc. Med.* **152**: 1247 (1998).
 10. E. Mowad, I. Haddad, and D.J. Gemmel. Management of lead poisoning from ingested fishing sinkers. *Arch. Pediatr. Adolesc. Med.* **152**: 485–488 (1998).
 11. M.V. Vance, S.C. Curry, J.M. Bradley, D.B. Kunkel, R.D. Gerkin, and G.R. Bond. Acute lead poisoning in nursing home and psychiatric patients from the ingestion of lead-based ceramic glazes. *Arch. Intern. Med.* **150**: 2085–2092 (1990).
 12. J.W. Nortier, B. Sangster, and R.G. van Kestern. Acute lead poisoning with hemolysis and liver toxicity after ingestion of red lead. *Vet. Hum. Toxicol.* **22**: 145–147 (1980).
 13. W.I. Manton and C.R. Malloy. Distribution of lead in body fluids after ingestion of soft solder. *Br. J. Ind. Med.* **40**: 51–57 (1983).
 14. D. Esernio-Jenssen, A. Donatelli-Guagenti, and H.C. Mofenson. Severe lead poisoning from an imported clothing accessory: “watch” out for lead. *J. Toxicol. Clin. Toxicol.* **34**: 329–333 (1996).
 15. P.E. McKinney. Acute elevation of blood lead levels within hours of ingestion of large quantities of lead shot. *J. Toxicol. Clin. Toxicol.* **38**: 435–440 (2000).
 16. N. Forsby, B. Fristedt, and B. Kjellman. Acute, lethal poisoning after ingestion of metallic lead. *Acta Paediatr. Scand. Suppl* **177**: 107 (1967).
 17. C.D. Hugelmeyer, J.C. Moorhead, L. Horenblas, and M.T. Bayer. Fatal lead encephalopathy following foreign body ingestion: case report. *J. Emerg. Med.* **6**: 397–400 (1988).
 18. CDC. Epidemiologic notes and reports: fatal pediatric poisoning from leaded paint—Wisconsin, 1990. *MMWR Morb. Mortal Wkly. Rep.* **40**: 193–195 (1991).
 19. CDC. Fatal pediatric lead poisoning—New Hampshire, 2000. *MMWR Morb. Mortal Wkly. Rep.* **50**: 457–459 (2001).
 20. F.E. Hillman. A rare case of chronic lead poisoning; polyneuropathy traced to lead shot in the appendix. *Ind. Med. Surg.* **36**: 488–492 (1967).
 21. W.B. Rydell, Jr. Bullet appendicitis. A new form of lead poisoning. *Rocky Mt. Med. J.* **67**: 48–50 (1970).
 22. H.H. Madsen, T. Skjodt, P.J. Jorgensen, and P. Grandjean. Blood lead levels in patients with lead shot retained in the appendix. *Acta Radiol.* **29**: 745–746 (1988).
 23. L.S. Carey. Lead shot appendicitis in northern native people. *J. Can. Assoc. Radiol.* **28**: 171–174 (1977).
 24. E.R. Reddy. Retained lead shot in the appendix. *J. Can. Assoc. Radiol.* **36**: 47–48 (1985).
 25. C.M. Balch and D. Silver. Foreign bodies in the appendix. Report of eight cases and review of the literature. *Arch. Surg.* **102**: 14–20 (1971).
 26. V. Durlach, F. Lisovoski, A. Gross, G. Ostermann, and M. Leutenegger. Appendectomy in an unusual case of lead poisoning. *Lancet* **1**: 687–688 (1986).
 27. J.D. Lyons and H.C. Filston. Lead intoxication from a pellet entrapped in the appendix of a child: treatment considerations. *J. Paediatr. Surg.* **29**: 1618–1620 (1994).

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