GUNSHOT-INDUCED PLUMBISM IN AN ADULT MALE

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Our objective is to present a case of symptomatic lead toxicity (plumbism) with abdominal colic and hemolytic anemia following a gunshot wound. It is a retrospective case report and the setting is in a teaching hospital in south central Los Angeles. The case report is that of a patient who presented with abdominal pain, generalized weakness, and hypertension following multiple gunshot wounds, 15 years previously. Other causes of abdominal pain and weakness—such as diabetes mellitus, alcohol abuse, pancreatitis, and substance abuse—were ruled out. Interventions included treatment with the newer oral chelating agent, Succimer (2, 3-dimercaptosuccinic acid), and subsequent surgery. The main outcome was the initial reduction in blood lead levels with improvement of symptoms. Because of a recurrent rise in the blood lead levels, the patient was again treated with Succimer and underwent surgery to remove two bullet fragments from the face. We conclude that lead toxicity should be ruled out in patients presenting with abdominal cramps and a history of a gunshot wound. Prompt therapy—including environmental intervention and chelation therapy—is mandatory, and surgical intervention may be necessary. (*J Natl Med Assoc.* 2003;95:986–990.)

Key words: lead poisoning ♦ plumbism ♦ anemia ♦ gunshot

INTRODUCTION

Although the data on plumbism in adult populations is scant, recent studies suggest that older adults are not only susceptible to lead poisoning but have an increased risk of cumulative lead toxicity by virtue of their age.[1] Retained bullet fragments, initially thought to be harmless, may sometimes lead to symptomatic lead intoxication.[2,3,4] The clinical picture of poisoning from systemic absorption of lead bullets resembles that of lead poisoning from chronic ingestion. The duration between lead exposure and the development of symptoms ranges from days to years, and patients may be asymptomatic for long periods of time.[5]

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Case Report

A 39-year-old African-American male presented with recurrent, sharp abdominal pain for two weeks, and anorexia, irritability, generalized weakness, headache, and constipation for two months. He denied any nausea, vomiting, or noticeable weight loss. There was no history of smoking, consumption of illicit drugs, alcohol abuse, or exposure to any known source of lead. Past history was significant for an exploratory laparotomy, following multiple gunshot wounds 15 years prior, and mild hypertension, which was untreated. Physical examination findings of significance included pale conjunctiva, grayish discoloration of the gingival tooth border (consistent with 'lead line'), an elevated blood pressure of 164/100 mmHg, mild generalized abdominal tenderness on deep palpation with a surgical scar noted. A complete neurologic

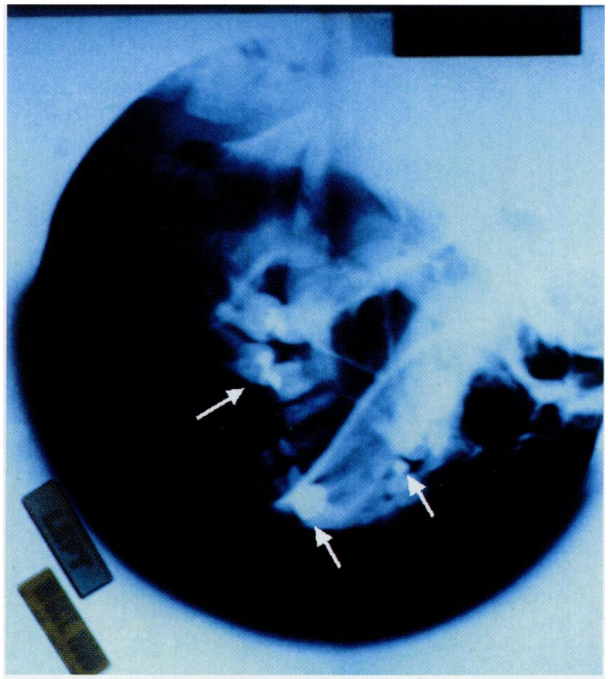

Figure 1
Facial Radiograph

Table 1. Sources of Lead Exposure

Occupational: Printers; painters; plumbers; pipe fitters; welders; radiator and auto repairers and gas station attendants who worked with leaded gasoline; lead miners; solderers and smelters; shipbuilders; battery manufacturers; cable makers; gun makers; workers in plastic, zinc, steel, copper, brass, bronze, scrap metal, rubber, glass, ceramics industries; and those in construction, demolition; and deleading jobs.[1,14,16]

Environmental: Inhalation of lead dust from demolition sites, contaminated clothes, inhalation of fumes or dust contaminated with leaded gasoline vapors.[14,16]

Recreational: Target shooting in indoor firing ranges, hobby ceramicists, gasoline sniffing, moonshine whiskey, lead-contaminated heroin or amphetamines.[14,16,21,22]

Miscellaneous: Alternative medicines, such as herbal medicines; traditional Mexican remedies (Azarcon, Greta); cooking in clay pots; lead-glazed ceramic ware; pica; retained bullets after gunshot(s) to soft tissue, bones, or joints.[13,16,19,23]

examination was performed, and apart from emotional instability, was negative. There were no signs of peripheral neuropathy.

Initial laboratory tests revealed hemoglobin of 7.6 g/dl (N:>14.0 g/dl), total white blood cell count of 10,800/ml (normal differential) and platelet count of 340,000/ml. The reticulocyte count was 10.2% (N:<1.5%). The stool guaiac was negative, and G6PD and hemoglobin electrophoresis were normal. Total serum bilirubin was 1.6 mg/dl (normal 0.3–1.0 mg/dl) with indirect bilirubin 1.3 mg/dl (normal 0.2–0.7 mg/dl) and lactate dehydrogenase 221 U/L (normal 100–190 U/L). Electrolytes, including blood urea nitrogen (BUN) and creatinine, were normal. The urinalysis was normal and did not show proteinuria. A review of the peripheral blood smear showed basophilic stippling of the red blood cells, hypochromia, polychromasia, and spherocytosis. The initial blood lead level was 201.9 µg/dl (normal, 0–9 µg/dl). His facial, chest, and abdominal radiographs revealed metallic bullet fragments (Figures 1, 2, 3). A computerized tomography (CT) scan of the head was normal and magnetic resonance imaging (MRI) of the head was not done.

The patient was treated with Succimer (2, 3 dimercaptosuccinic acid), 10 mg/kg every eight hours for five days, followed by 10 mg/kg every 12 hours for an additional two weeks. This resulted in significant symptomatic improvement and a decrease in the blood lead levels to 64 µg/dl. Three months later, the patient returned with similar complaints, and the blood lead level was found to be 159 µg/dl. He was again treated with the same regimen of Succimer therapy for 19 days, following which most symptoms resolved. There was still some generalized weakness and irritability. Repeat serum lead levels obtained after Succimer therapy revealed a blood lead level of 29 µg/dl. Two bullet fragments were removed from the face, but the patient declined further surgery.

DISCUSSION

Lead has been used in both households and industry; consequently, environmental lead exposure is widespread. Lead paints, air polluted with industrial fumes and automobile exhausts, contaminated water and soil, canned foods, and vegetables grown in lead-contaminated soil are among more common culprits, especially in places lacking strict regulatory laws.[1] Other possible sources of lead

exposure are noted in Table 1. Although lead poisoning is a condition known since ancient times, it is still considered to be one of the most common environmental diseases, and in the United States, it is a reportable one.[1,6]

With the increasing popularity of alternative therapies—including herbal medicines, Ayurveda, and traditional Chinese medicines (TCMs)—the risk of lead toxicity may increase. In India, a form of traditional medicine called Ayurveda is usually made of plant products and occasionally made from animal products; however, metals and minerals are sometimes used. The metals usually consist of lead, mercury, arsenic, or rarely gold.[7,8,9,10] Chinese herbal medicines (CHM) and Chinese proprietary medicines (CPM) are often adulterated with substituted herbs, heavy metals, and western medicines. Adulterants may include heavy metals, such as lead and cadmium.[11,12] Traditional Mexican remedies, such as Azarcon and Greta—used for digestive problems—contain up to 86% and 90% lead respectively.[13]

The Occupational Safety and Health Administration (OSHA) mandates that blood lead levels in

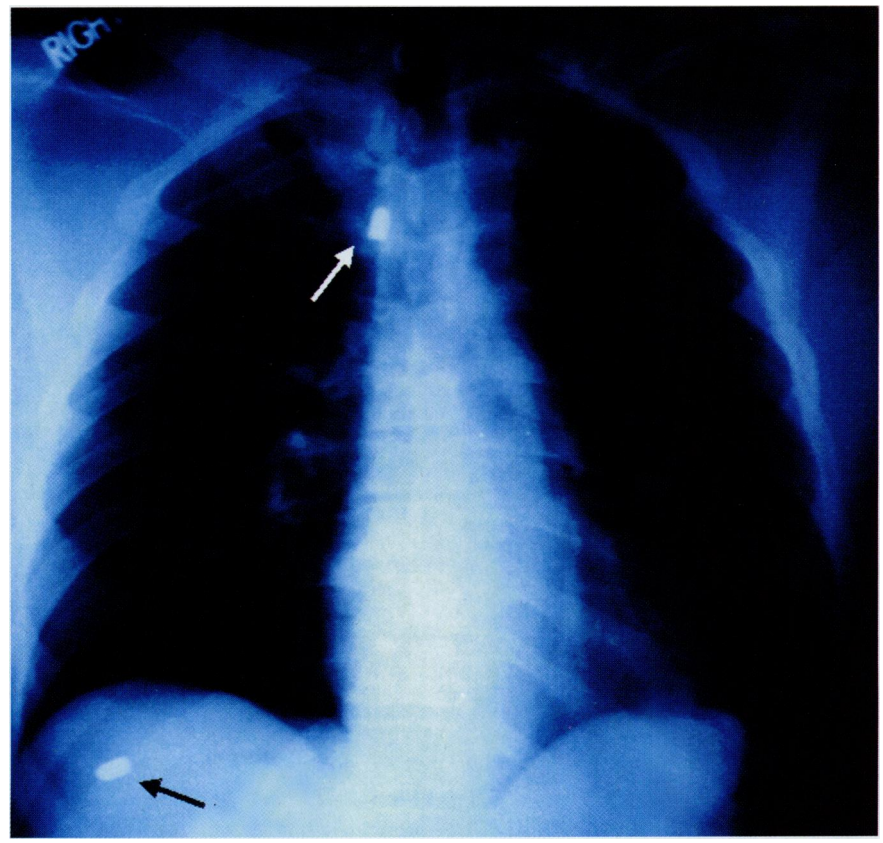

Figure 2
Chest Radiograph

Table 2. Clinical Effects of Lead Toxicity

Neurological[1,16]
Neurobehavioral retardation
Peripheral neuropathies, paresthesia, paralysis
Headache, irritability, lethargy, fatigue, tremor
Encephalopathy, seizures, insomnia, memory loss

Gastrointestinal[2]
Abdominal pain, anorexia, nausea, vomiting, constipation, elevated liver enzymes, hepatitis, bluish-gray discoloration of gingiva

Hematopoietic[2,3]
Anemia (normochromic, hypochromic, hemolytic)
Basophilic stippling of red blood cells
Erythrocyte protoporphyrin elevation

Renal[1,16,17]
Nephropathy
Renal tubular dysfunction, proteinuria
Excretion of ß2-microglobulin and N-acetylglucosidase in urine
Hypertension

Reproductive[16]
Male: Decreased libido, oligospermia, dysspermia
Female: Increased risk of infertility, preterm delivery, fetal malformations, chromosomal aberrations

Musculo-skeletal[1]
Muscular fatigue, arthralgia, arthritis

workers with occupational lead exposure be less than 50 μg/dL.[1,6] According to the Centers for Disease Control (CDC), childhood blood lead levels of 10 μg/dL or greater are a cause for concern. Therefore, they have recommended reducing permissible lead levels to 10 μg/dL or less in children.[1,14] There is evidence to suggest that blood lead levels as low as 15 μg/dL at an early age may result in subsequent psychomotor and intellectual dysfunction.[15]

Our patient did not have a history of any obvious lead exposure other than retained bullet fragments; therefore, the clinical diagnosis of gunshot-induced plumbism was made. The manifestations of lead poisoning may be diversified and involve multiple organ systems, as noted in Table 2. Hemolytic anemia is a well-known complication of lead toxicity.[3] Our patient's peripheral smear, reticulocytosis, elevated indirect bilirubin, and LDH levels were consistent with hemolysis. Additionally, and of particular importance, is the effect of chronic lead exposure on the kidneys. Patients may develop lead nephropathy, renal failure, and hypertension—even with low levels of lead. Lead nephropathy is a chronic tubulointerstitial disease with proteinuria, tubular atrophy, and fibrosis. Patients may present with hyperuricemia, gout, and hypertension. Our patient did have hypertension but normal uric acid levels, BUN, creatinine, and urinalysis. Lead toxicity often remains a subtle condition, so the diagnosis may be easily missed. This may result in an adverse prognosis or even a fatal outcome.[1,14,16,17]

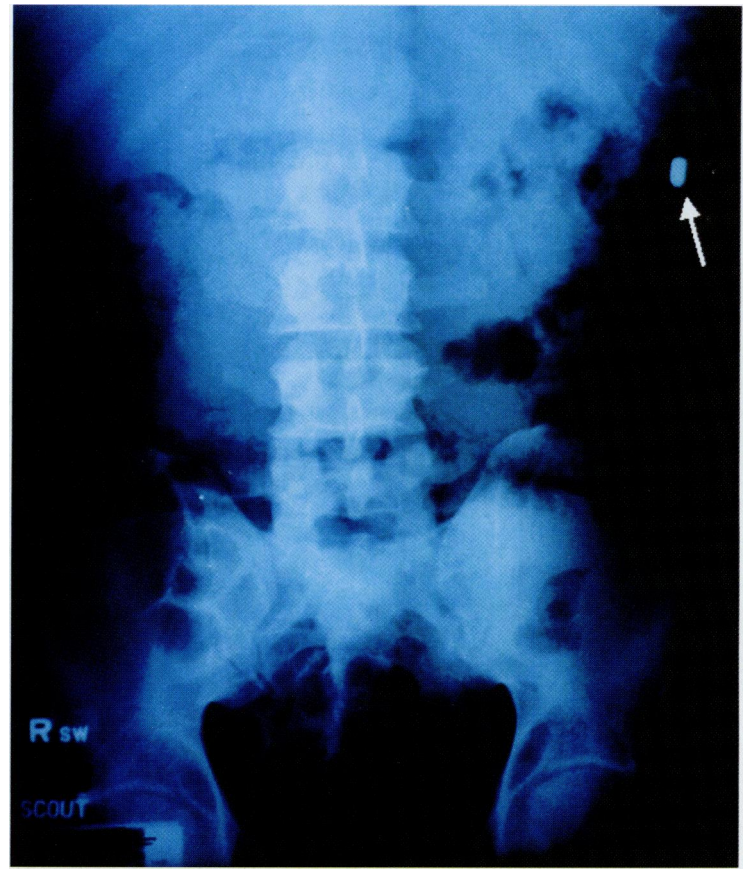

Figure 3
Plain Abdominal Radiograph

Factors which may modify the risk of lead toxicity from retained bullets include: location, number, and lead content of the bullets.[18] A bullet lodged in a joint being exposed to acidic synovial fluid and joint movements carries a higher risk of lead absorption than bullets located in soft tissues. Likewise, multiple, small bullet fragments have an increased surface area and a greater risk of lead toxicity as compared to a single bullet.[5]

Chelation therapy should be urgently started after a diagnosis of lead poisoning has been made. In cases of lead toxicity due to retained bullets, surgical removal of the bullets should be considered when feasible; however, surgery should be preceded by chelation therapy to avoid possible risks of acute exacerbation of lead toxicity. Depending upon total body stores of lead and response to therapy, some patients may require multiple courses of chelation therapy.[16,19]

Our patient had chelation therapy with Succimer (also known as 2, 3 dimercaptosuccinic acid or DMSA), a new oral chelating agent, but declined surgical removal of retained pellets initially. He consented to surgery after becoming symptomatic the second time. Succimer is effective in decreasing blood lead levels and has substantial advantages over other chelating agents, such as dimercaprol (BAL) and calcium edetate (EDTA). Advantages include oral administration, feasibility of outpatient therapy, avoidance of hospitalization expenses, and absence of significant toxicity, as it is a more specific lead chelator.[16,19,20]

CONCLUSION

Our patient presented with diversified manifestations of lead poisoning—including abdominal colic, generalized weakness, and hemolytic anemia—which responded to chelation therapy. Lead toxicity or poisoning should be ruled out in patients presenting with abdominal cramps and a history of a gunshot wound with or without other lead related exposure. A high index of suspicion and a low threshold for obtaining diagnostic tests is prudent in making the diagnosis. Prompt therapy, including environmental intervention and chelation therapy, is mandatory. Surgical removal of retained bullets may be necessary to prevent an adverse outcome in cases of plumbism.

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