

A fatal case of lead poisoning due to a retained bullet

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ABSTRACT Lead poisoning from a retained bullet or missile is rare and is usually dependent on the location of the missile in a bone or immediately adjacent to a joint. A review of the literature revealed only 14 cases in which there was adequate laboratory documentation of plumbism caused by a retained bullet or missile. Only one of these previously reported cases resulted in death. We report a second death due to lead poisoning from a retained bullet with elevated blood lead levels documented by toxicologic analysis.

The most common causes of lead poisoning in the United States are ingestion of lead-based paint, industrial or environmental exposure, and consumption of "moonshine" liquor contaminated with lead. Lead poisoning from a retained bullet or missile is rare. Even more unusual is death due to this entity, with only one case well-documented in the literature.⁽¹⁾ We report a second fatal case of lead poisoning due to a retained bullet in which toxic blood lead levels were documented.

CASE HISTORY

A 54-year-old, black female had been followed in the arthritis clinic at Parkland Memorial Hospital. In October, she was seen in the emergency room with complaints of constipation and abdominal pain. The pain was poorly characterized but was not associated with nausea, vomiting, hematemesis, hematochezia, or melena. She was treated symptomatically for constipation and discharged from the emergency room. She returned 3 weeks later with the complaint of black stools. The guaiac test was 1+ positive. Sigmoidoscopy, barium enema, upper GI series, and oral cholecystogram was performed on an outpatient basis and were negative. A routine blood count revealed a hemoglobin of 7.6 g% and hematocrit of 23.9 vol%. She was admitted for evaluation of her anemia. Her past medical history

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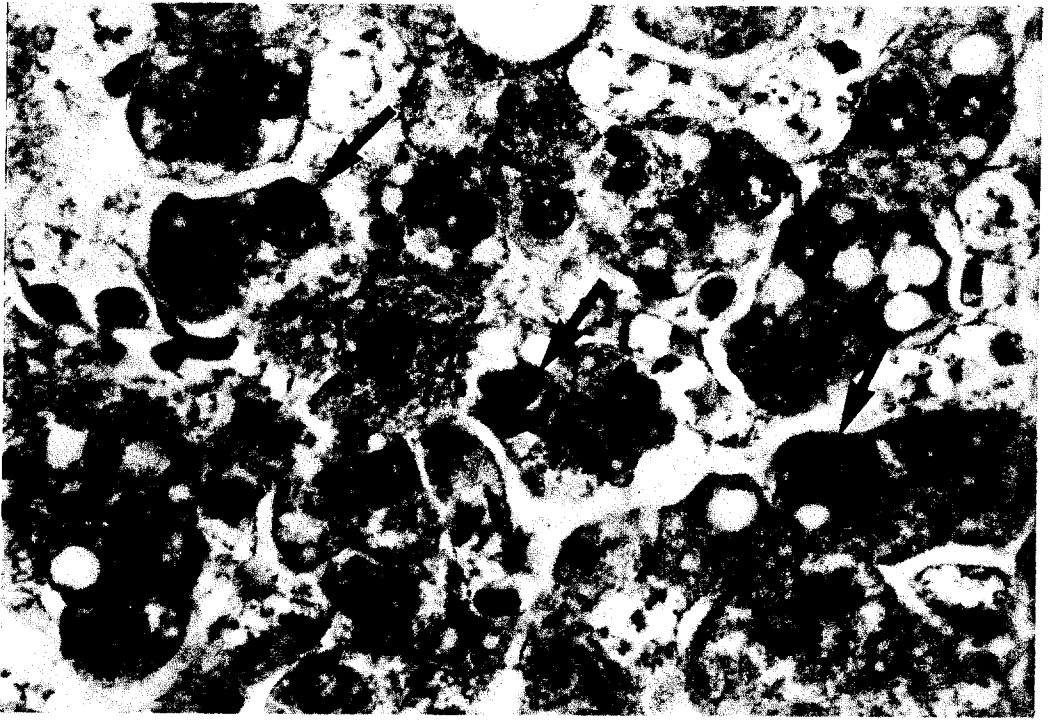


FIGURE 1
Intranuclear inclusions in hepatocytes (arrows).

was significant for a gunshot wound to the right knee 5 months prior to this admission.

Physical examination revealed a short, obese, black female in no distress. Her blood pressure was 140/90 mm Hg, her pulse was 96/minute, respiratory rate 16/minute, and she was afebrile. Examination of the head and neck was remarkable for pale conjunctivae. The chest was clear. Cardiovascular examination revealed a grade II/VI systolic ejection murmur at the lower left sternal border. The remainder of the physical examination including the neurological examination was within normal limits. Stool guaiac test was negative. Laboratory data revealed a hemoglobin of 6.9 g%, hematocrit of 21.3 vol%, MCV of 84, and MCHC of 32.3. The reticulocyte count was 5% (1% corrected) and the peripheral smear was remarkable for basophilic stippling, mild anisocytosis and poikilocytosis, polychromassia, hypochromasia, and target cells. There were no schistocytes or burr cells noted. Her white count was 5,500 with a normal differential. The platelet count was 388,000. Serum electrolytes, glucose, BUN, creatine, and routine urinalysis were within normal limits, as were clotting parameters. Sickle cell preparation and test for antinuclear

antibody were also negative. Total protein, albumin, and serum protein electrophoresis were normal. Liver function tests revealed an SGOT of 106 IU/L, with a normal total bilirubin, alkaline phosphatase, CPK, and LDH. Serum iron and iron-binding capacity were within normal limits. A chest x-ray revealed several calcified granulomas.

For the first several days in the hospital she continued to complain of pain in her knees and abdominal pain. On the evening of the eighth hospital day she was found unresponsive on the floor with her eyes tonically deviated to the right. Her vital signs were normal and neurological exam documented postical confusion. No focal neurological signs were detected. The following morning the patient had a grand mal seizure witnessed by the house staff. Skull films were taken and there was no abnormality noted. Her serum electrolytes, serum calcium, and serum magnesium levels within normal limits. A general toxicology screen was negative. Lumbar puncture revealed clear fluid with a protein of 130 $\mu\text{g/L}$, glucose of 73 mg/dl (plasma glucose 114 mg/dl), 81 RBC's/ mm^3 and 15 WBC's/ mm^3 (2% neutrophils, 87% lymphocytes, 11% monocytes). Cytology revealed no malignant cells. Bacterial

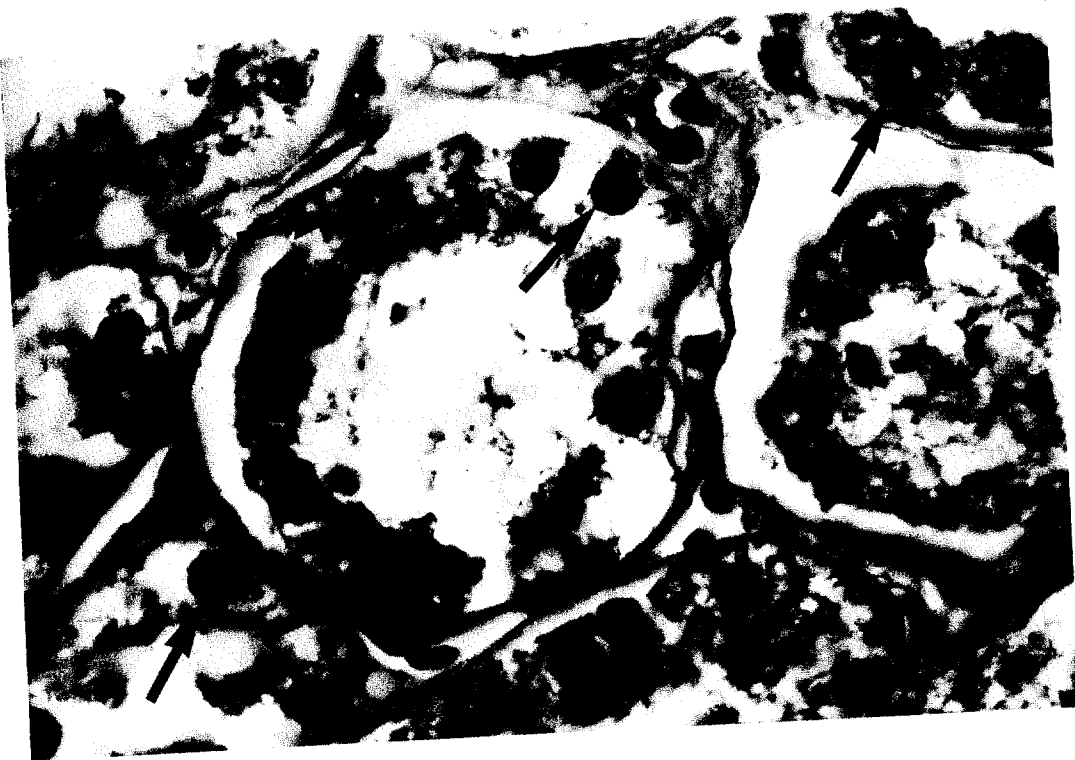


FIGURE 2
Intranuclear inclusions in cells of proximal convoluted tubules (arrows).

cultures and VDRL were negative. India ink preparation and a cryptococcal antigen were negative as were acid fast smear and culture. She was begun on phenobarbital and diphenylhydantoin. Over the next several days she appeared disoriented. Her vital signs remained normal and no focal neurological signs developed. Her anemia remained unchanged on oral iron supplements. On the 13th hospital day the patient had four grand mal seizures, following which she was comatose with Cheyne-Stokes respirations. She responded to pain by moving her right side, had positive doll's eyes, exhibited decreased tone with hyperreflexia on the left side, and had upgoing Babinski's bilaterally. A repeat lumbar puncture was normal. A CT scan of the brain was compatible with diffuse cerebritis with possible compression of the left lateral ventricle. The following morning, she was found unresponsive without vital signs. Cardiopulmonary resuscitation was unsuccessful.

The diagnosis of lead poisoning was never considered clinically in this case and appropriate toxicologic analysis was not requested. In part, this may have been due to the misconception that "routine" toxicologic screen covers all potentially lethal drugs

or substances. Death was believed to have been due to a central nervous system lesion. As the cause of death was believed to be natural, this case was not reported to the medical examiner's office. Autopsy permission was obtained by the hospital autopsy service.

At autopsy, pertinent findings were confined to the head. The brain was swollen with uncal herniation and necrosis. Secondary brain stem hemorrhages were present. No cause of the cerebral edema could be found. During the autopsy, it was suggested that this death might represent a case of lead poisoning on the basis of the clinically observed basophilic stippling. In view of this finding, the prosector submitted blood obtained at autopsy to the Clinical Toxicology Laboratory of the Southwestern Institute of Forensic Sciences for lead analysis. The Institute, composed of the Medical Examiner's Office and the County Criminal Investigation Laboratory, performs all clinical toxicology analyses for Parkland Memorial Hospital. A check of the Institute's records revealed that a specimen of the patient's blood had been submitted to the Forensic Laboratory 9 days after admission. At that time, a screen for acid, basic, and neutral drugs was requested. The



FIGURE 3
Deformed .32 caliber bullet and fragments in soft tissue proximal and lateral to femoral condyles.

screen was negative. There was no request for a heavy metal screen. The blood obtained at that time had been retained by the laboratory; therefore, both postmortem and antemortem blood was available for toxicological analysis for heavy metals.

Analysis of the blood obtained postmortem by flameless atomic absorption⁽²⁾ revealed a lead level of 5.3 mg/liter. The antemortem blood obtained 5 days prior to death had a lead level of 5.1 mg/liter. Blood lead above 0.6 mg/liter is considered toxic in adults in our laboratory.

Microscopic examination of the tissue removed at autopsy revealed characteristic eosinophilic, intranuclear inclusions in hepatocytes and cells of the proximal tubules of the kidneys (Figs. 1 and 2). Many of the perivascular spaces in the brain contained aggregates of PAS-positive pink-staining homogenous material.

Because the diagnosis of lead poisoning due to a retained bullet was not seriously entertained at autopsy, no attempt was made to recover the bullet or

to determine its proximity to the knee joint. Following the report of this case to the medical examiner's office and the discovery of toxic levels of lead in the blood, x-rays taken at the time the deceased had been shot in the leg were obtained and reviewed. These showed a flattened, deformed lead .32 caliber revolver bullet lodged in the soft tissue anterolateral to the distal femur and immediately proximal to the condyles (Fig. 3). Smaller fragments of lead were present adjacent to the main missile mass. The location of the bullet was consistent with it being in or in communication with, the suprapatellar bursa.

In order to exclude other possible causes of plumbism, the medical examiner's office contacted and interviewed the deceased's next of kin. No history of environmental exposure to lead could be elicited. The deceased had been a housewife for a number of years. She did not consume 'moonshine.' There were no earthenware dishes or bowls in the house. She lived with her sons, all of whom consumed the same food that she did.

DISCUSSION

Lead poisoning caused by a retained missile is extremely rare with only 14 well-documented cases in the world literature to our knowledge. In an excellent review of the subject, Machle in 1940 discussed the 40 cases of plumbism caused by retained bullets or missiles reported at that time.⁽³⁾ In 11 of these cases the patient's urine lead level was described as either present (nine patients) or present in significant concentration (two patients). In only three of the 40 patients were blood lead levels reported. In the remaining 29 patients, neither the urine or blood lead levels were known or detected. Machle added two of his own cases to the three that he considered had adequate quantitative analytical analysis of body fluid for lead, both with documented elevated urine and blood lead levels. Since then, four other cases of lead intoxication caused by retained bullets with documentation by toxicologic analysis have been reported in the English literature^(1,4-6) with five others ascertained by Dillman et al. from the foreign literature.⁽⁷⁻¹¹⁾

The dissolution of the bullet or missile has been shown to be related to its cellular and chemical milieu.^(3,12) The solubility of lead is greater in fats and body fluids than in water, and this solubility is increased under elevated carbon dioxide tension or with increased pH.⁽¹³⁾ Machle noted the high incidence of bone or joint bullet lodgement with clinical evidence of lead intoxication,⁽¹⁾ and this association

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CONCLUSION

The diagnosis of lead poisoning in the authors' case was confirmed by the markedly elevated levels of lead in both antemortem and postmortem blood. Characteristic intranuclear inclusions in the cells of the liver and kidneys and PAS-positive perivascular edema fluid in the brain confirmed histologically the diagnosis of saturnism. This represents the second case of death due to lead poisoning from a retained bullet documented by toxicologic analysis. □