CASE REPORT

Plumbism or Lead Intoxication Mimicking an Abdominal Tumor

Peter Dedeken,¹ Vernon Louw,^{2,3} Ann-Karolien Vandooren,² Geert Verstegen,⁴
Willy Goossens,⁴ Bénédicte Dubois¹

¹Department of Neurology, University Hospital Gasthuisberg, Leuven, Belgium; ²Department of Haematology, University Hospital Gasthuisberg, Leuven, Belgium; ³Clinical Haematology Unit, Department of Internal Medicine, Universitas Hospital, Bloemfontein, South Africa; ⁴Department of Laboratory Medicine, University Hospital Gasthuisberg, Leuven, Belgium.

The clinical presentation of lead intoxication may vary widely and in the absence of a high clinical index of suspicion, the diagnosis may be missed. The effects of lead on mitochondrial oxidative phosphorylation and its interaction with calcium-mediated processes explain the heterogeneous presentation. In this case report, the diagnosis was finally made when bilateral wrist drop developed on top of abdominal cramps and anemia. Before, ascites raised the suspicion of a tumor. Therefore, each element of the triad of unexplained anemia, abdominal cramps, and bilateral wrist (or foot) drop should lead any physician to consider the diagnosis of lead intoxication. This case also illustrates the importance of a careful and meticulous social history in patient management.

KEY WORDS: lead intoxication; plumbism; ascites.

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INTRODUCTION

A 64-year-old woman was referred to her local hospital with a 6-month history of anorexia, weight loss, and colicky abdominal pain. She was an active and healthy white woman with no previous medical or family history of note. Clinical examination revealed bilateral ankle edema and a slightly distended abdomen. Plain abdominal x-rays in an upright position did not show any air under the diaphragms nor any features suggestive of small or large bowel obstruction. Abdominal ultrasound and computer tomography showed ascites, but no evidence of organ enlargement, hydronephrosis, abdominal masses, or lymphadenopathy. Tumor markers were normal and the only abnormality found was the presence of a normochromic, normocytic anemia with a hemoglobin of 9.4 g/dL (12.0–16.0 g/dL). Gastroscopy and colonoscopy revealed no abnormalities. Paracentesis yielded cells with a malignant appearance, suggestive of adenocarcinoma. Therefore, although a laparoscopy did not reveal macroscopic evidence of malignancy, a total hysterectomy with omentectomy was performed 2 months after initial presentation. On pathologic examination, no adenocarcinoma was found.

The patient was admitted to the hospital again 4 months after initial presentation with persistent colicky abdominal pain, and she was referred to our institution. Her full blood count showed a microcytic anemia (hemoglobin 6.9 g/dL) with

normal serum iron and ferritin levels as well as a raised indirect and total bilirubin, without any other biochemical features suggestive of hemolysis. A peripheral blood and bone marrow smear and biopsy revealed a leukopenia, erythroid lineage hypercellularity, and marked basophilic stippling (Fig. 1). A blood transfusion was performed. Later, a Coombs reaction turned out to be weakly IgG positive, accompanied by positive cold hemagglutinins but no complement use, initially thought to suggest a possible chronic low-grade hemolytic anemia.

Six months after initial presentation, however, the patient developed a subacute right-sided wrist drop, followed by left-sided wrist drop a few days later (Fig. 2). Neurological examination was normal except for a marked attention deficit, severe impairment of short-term memory, and weakness in both upper limbs. The latter was more pronounced distally than proximally, with the extensor muscles affected more severely than the flexors. Electromyography showed a bilateral motor axonal polyneuropathy that was most pronounced in the extensor muscles.

The clinical suspicion of lead intoxication based on hematological and neurological signs was confirmed by the highly elevated blood lead level of 146 µg/dL (<25). Further testing showed raised free erythrocyte protoporphyrin of 13122 µg/L RBC (<550) combined with raised zinc protoporphyrin of 12353 µg/L RBC (<400), urinary δ-amino-laevulinic acid (ALA) of 65.4 mg/L (<5.7), and high urine coproporphyrin of 635 µg/L (<130). There was also an increased amount of lead in a 24-hour urine collection of 966 µg/24 hours (<50), with an increased lead/creatinine ratio.

Chelation therapy was started immediately with 2,3-dimercapto-succinate (DMSA). One month follow-up showed improved attention and mild improvement of the extensor function. Biochemically, the blood lead level had decreased to 43 µg/dL. After 6 months, further improvement was documented and the patient was able to write again.

On subsequent questioning, it became clear that this patient had been exposed to lead in various ways and over a prolonged period of time. As a child, she worked in her father's shop, applying lead to make wooden shoes. Later, she helped her brother in his paint shop. Five years before the onset of clinical symptoms, she had the lead water piping in her house replaced, which up to then had been used for drinking water.

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Address correspondence and requests for reprints to Dr. Dubois: Department of Neurology, University Hospital Gasthuisberg, Herestraat 49, B-3000 Leuven, Belgium (e-mail: Benedicte.Dubois@uz.kuleuven.ac.be).

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FIGURE 1. Basophilic stippling on red blood cells. Basophilic stippling of red blood cells is due to ribosomal aggregation. Although this is not specific for lead poisoning, the diagnostic possibilities are confined to only a few other conditions: pyrimidine 5' nucleotidase deficiency, thalassemia, and vitamin B12 deficiency.

The most important factor, however, was her hobby of flower arranging, for which she used large amounts of lead to make decorative figures in which she arranged the flowers. Her family went back to the house and removed 50 kg of lead from the enclosed basement area where she made the leaden figures.

DISCUSSION

While we are unable to explain the mechanism of ascites in our patient, this is the first case report of ascites associated with lead intoxication. The suspicion of a malignancy delayed diagnosis and treatment, and led to unnecessary interventions.

The most common presenting symptoms of chronic lead exposure are shown in Table 2.¹ Lead intoxication is generally

FIGURE 2. Bilateral wrist drop. The predominant involvement of distal extensor muscles is typical of lead neuropathy. This figure illustrates the inability of the patient to extend the wrist in order to lift the coin from the table.

Table 1. Laboratory Values

	Actual Value	Normal Values
Tumor markers		
Carcinoembriogenic antigen	<0.5 µg/L	≤ 2.5
CA 125	6 kU/L	≤ 35
CA 19.9	4 kU/L	≤ 37
Hemoglobin	6.9 g/dL	12 to 16
Red blood cell count	$2.52 \times 10^{12}/L$	3.9 to 5.6
White blood cell count	$4.7 \times 10^9/L$	4 to 10
Platelets	$173 \times 10^9/L$	150 to 450
Mean corpuscular volume	88.9 fL	76 to 96
Red cell distribution width (RDW)	18.8%	11.7 to 14.5
Serum iron	159 µg/L	50 to 170
Ferritin	124 µg/L	15 to 300
Transferring saturation	36%	16 to 45
Coombs test	Weakly IgG positive	Negative
Cold hemagglutinins	Positive	Negative
Complement		
Total	106%	70 to 140
C3	0.71 g/L	0.79 to 1.52
C3d	< 1.1%	≤ 2.4
C4	0.16 g/L	0.16 to 0.38
Paracentesis	$2.3 \times 10^9/L$ leukocytes (84% lymphocytes, 16% monocytes) malignant cells, suggestive of adenocarcinoma	

because of professional exposure (battery manufacturers, lead mining and processing, painting, shipbuilding, plumbing).²⁻⁵ Other ways include environmental exposure (traffic, industry), hobbies (glass-in-lead, ceramics, or like in this case for flower arranging),²⁻⁴ or accidental (folk remedies, moonshine whiskey).¹

Lead interferes with many biochemical pathways. The inhibition of 5-amino-laevulinic-acid-dehydratase (ALA-D) and ferrochelatase^{2,6} causes decreased production and incorporation of hemoglobin. Second, lead mimics calcium ions and therefore interferes with calcium-related processes, which influences gastrointestinal motility, neuronal function, and bone formation. For instance, inactivation of the blood-brain barrier and resulting brain edema result in a variety of neurological symptoms. Furthermore, calcium competition in mitochondrial oxidative phosphorylation,^{2,6} and interaction with nucleic acid physiology⁷ explain the possible effect on every organ system.

The diagnostic gold standard is the blood lead level. Other functional tests mainly show the biochemical effects: free erythrocyte protoporphyrin and zinc protoporphyrin are markers for toxic effects on the bone marrow. Urinary lead excretion as well as ALA excretion and ALA-D enzymatic activity are good markers for recent exposure, but may fluctuate and are less reliable.^{3,6,7}

Table 2. Continuum of Symptoms Associated with Ongoing Lead Exposure

Impaired abilities (patient may be asymptomatic < 40 µg/dL)
Decreased learning, verbal ability, and memory
Lowered IQ, hyperactivity, or ADHD in children
Hypertension
Mild toxicity (40 to 60 µg/dL)
Myalgia or paresthesia
Irritability
Occasional abdominal discomfort
Mild fatigue or lethargy
Moderate toxicity (> 60 µg/dL)
Arthralgia
Headache
Vomiting
Constipation
General fatigue
Tremor
Diffuse abdominal pain
Weight loss
Severe toxicity (> 100 µg/dL)
Paresis or paralysis
Lead nephropathy
Lead line (blue-black) on gingival tissue
Encephalopathy: seizures, changes in consciousness, coma, death
Colic (intermittent, severe abdominal cramps)

IQ, intellectual quotient; ADHD, attention deficit and hyperactivity disorder.

The main therapeutic measure is avoiding further exposure to lead. Blood lead levels drop 2 to 4 weeks after elimination of the exposure but in chronically exposed patients, these may remain high because of release of lead from bones, where lead levels may have accumulated over years. Medical treatment mainly consists of chelation by CaNa₂-EDTA and DMSA.^{2,6} Chelation may be considered when blood lead levels

exceed 80 µg/dL in asymptomatic patients and 50 µg/dL in symptomatic patients.

CONCLUSION

The initial presentation of lead intoxication may vary, and a high index of suspicion is required to make this diagnosis. As in this case, the diagnosis may be missed, which may lead to unnecessary procedures and may prevent patients from being treated expeditiously. Therefore, each element of the triad of unexplained anemia, abdominal cramps, and bilateral wrist (or foot) drop should lead any physician to consider and exclude the diagnosis of lead intoxication. This case also illustrates the importance of a careful and meticulous social history in patient management.

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