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CASE REPORT | COLON

Acute Colonic Pseudo-Obstruction as a Manifestation of Lead Intoxication in a Conservator

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ABSTRACT

We report a case of a 39-year-old art conservator who presented complaining of abdominal pain and constipation. His laboratory results showed normocytic normochromic anemia and abnormal liver tests. Computed tomography revealed distention of the whole colon without obstruction. Evaluation of anemia was compatible with nonimmune hemolysis. A liver biopsy showed accumulation of ferric pigment in Kupffer cells. Given the typical findings in the blood smear and the epidemiological context, a serum lead assay was performed (92 μg/dL). This clinical case illustrates the need for gastroenterologists to recognize digestive manifestations of systemic diseases, including intoxications.

INTRODUCTION

Lead poisoning causes nonspecific symptoms such as abdominal pain, constipation, irritability, concentration difficulties, and anemia. It can have a broad clinical spectrum, running as a multisystemic disease. The constellation of symptoms may not accurately correlate with traditional indices of exposure.[1] According to the U.S. National Notifiable Diseases Surveillance System, the diagnosis of lead poisoning is confirmed by measuring the blood lead levels; a level of 5 μg/dL or higher is considered elevated.[2] It is a difficult diagnosis to make, where the clinical history (especially the epidemiological inquiry) assumes particular relevance. We describe a case of lead intoxication characterized by colonic pseudo-obstruction, hemolytic anemia, and abnormal liver chemistries.

CASE REPORT

A 39-year-old male art conservator with no major medical history presented with a 3-week history of abdominal pain. In the previous 5 months, he had been working in the rehabilitation of an early 20th-century church. He also had 7-day constipation and new-onset jaundice. Two months before the admission, he started having headache, mood alterations (easy irritability, anxiety, and short-term memory impairment), dysgeusia (metallic taste), decreased libido, and sexual impotence. Neurological symptoms such as altered muscle strength and altered sensory function were not reported.

Physical examination was notable for darkening of the gums, jaundice, abdominal distension, and diffuse abdominal pain. Neurological examination was normal. His laboratory results revealed a normocytic normochromic anemia. The liver chemistries showed indirect hyperbilirubinemia (1.83 mg/dL), with mild aminotransferase elevation (alanine aminotransferase 76 IU/L; AST 45 IU/L, about 2 times the upper level of normal) and normal alkaline phosphatase (108 IU/L). The abdominal radiographs revealed gaseous distention of the colon, with radiopaque aspect in the right quadrants suggestive of fecal content. The abdominal computed tomography showed diffuse colon dilatation, particularly at the cecum (longitudinal diameter of 6.7 cm) and transverse levels (Figure 1).

A colonic pseudo-obstruction was assumed, with no defined etiological diagnosis. The patient was hospitalized for conservative treatment and further investigation. Upper endoscopy and colonoscopy showed no pathological findings. After 2 days of supportive

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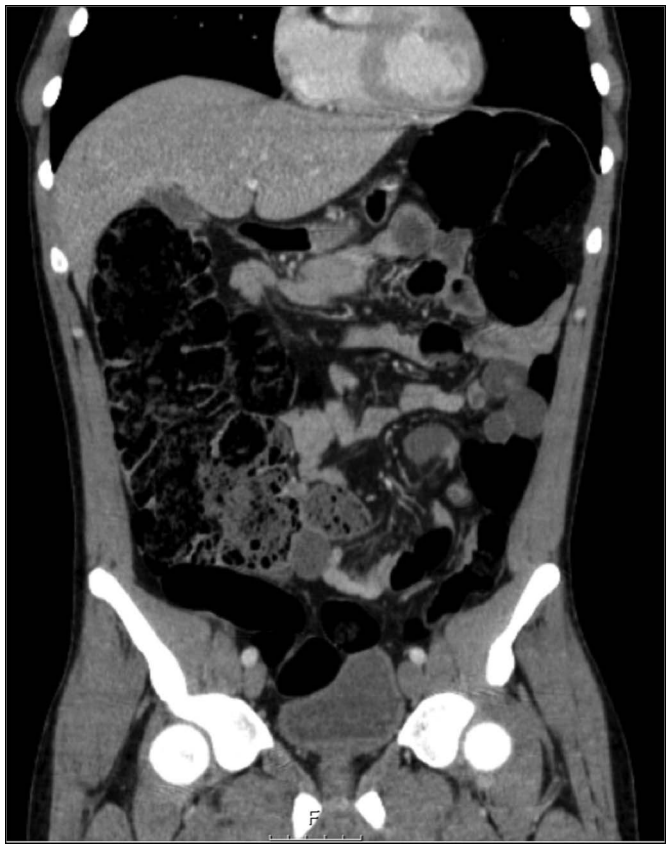

Figure 1. Abdominal computed tomography showing colonic distension without evidence of obstruction.

care with *nil per os* and nasogastric tube decompression, bowel movements restarted. Abdominal radiograph reevaluation showed intestinal distension improvement. Evaluation of anemia was compatible with nonimmune hemolysis. The peripheral blood smear had abundant stomatocytes and polychromatic cells with basophilic dotting (Figure 2).

An increase in transaminases levels was noted, reaching a maximum on the fifth day of hospital stay (aspartate aminotransferase 265 IU/L; alanine aminotransferase 336 IU/L, about 10 times the upper limit of normal). The etiological study for liver disease (including viral serologies, autoimmunity, iron kinetics, ceruloplasmin, and urinary copper) was negative. We performed a liver biopsy that showed the presence of mixed hepatocellular steatosis, mild hepatocellular siderosis, and accumulation of ferric pigment in Kupffer cells (Figure 3).

Given the clinical presentation, the typical findings in the blood smear, and the epidemiological context, a serum lead assay was performed and was compatible with toxic levels (92 μg/dL). Chelation with calcium disodium edetate was started at a dose of 20 mg/kg/d for 5 days, without intercurrences. The patient was discharged after the chelation therapy, with resolution of colonic disease. Clinical response was monitored at the ambulatory department. After 2 cycles of chelation, a significant decrease in serum lead levels was observed (8 μg/dL).

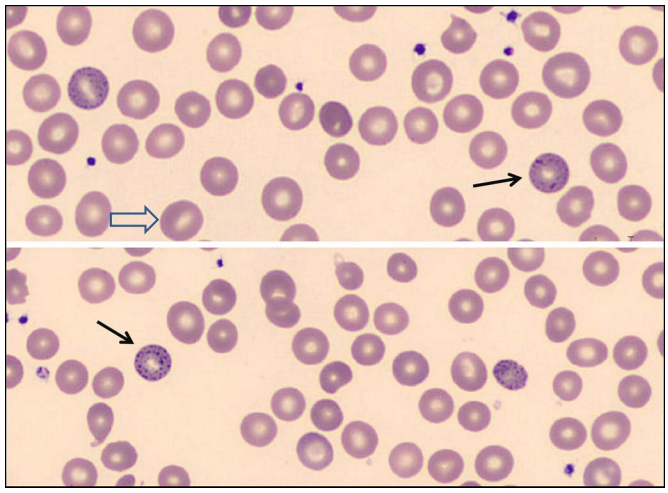

Figure 2. Peripheral blood smear with stomatocytes (open arrow) and polychromatic cells with basophilic dotting (arrows).

DISCUSSION

Most cases with elevated blood lead levels come from workplace exposures. One source of contamination can be the paint used in construction sites and at home.[3] The lead content of paint was unregulated until 1977. In 2000, it was estimated that approximately 1 million construction workers in the United States were occupationally exposed to lead.[4] In this case, the patient had been working for several months in a church, which was built before regulation of paint lead levels. In addition to chronic occupational exposure, we supposed that the conservation project he was working on contributed to an acute increase in serum lead levels.

This patient presented with abdominal pain. The most common causes of abdominal pain and life-threatening emergencies were ruled out after radiological and endoscopic study. A diagnosis of pseudo-obstruction was assumed. This

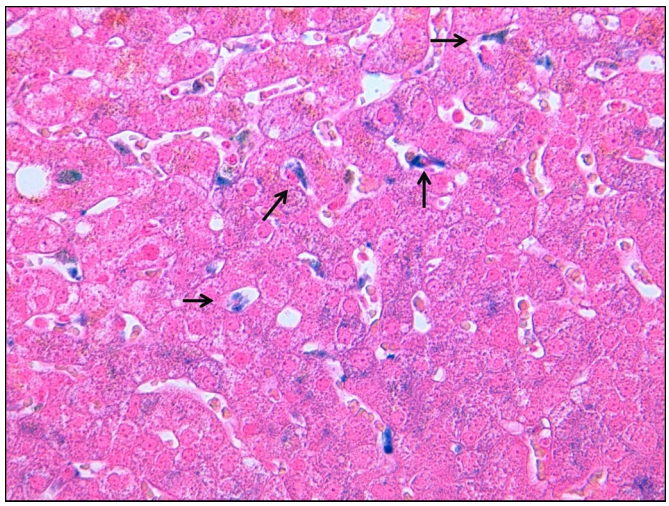

Figure 3. Liver biopsy with accumulation of ferric pigment in Kupffer cells (arrows).

disease is characterized by signs and symptoms of a mechanical obstruction of the small or large bowel in the absence of a mechanical cause. The incidence of acute colonic pseudo-obstruction is approximately 100 cases out of 100,000 inpatient admissions per year.[5] It can be associated with a metabolic imbalance or impairment of the autonomic nervous system, which can be seen in lead poisoning.[6] There are a number of similarities between lead neuropathy and the neuropathy associated with porphyria.[7] The inhibition of 5-aminolevulinic acid dehydratase and consequent overproduction of aminolevulinic acid mediate abdominal pain and autonomic dysfunction in lead intoxication and acute porphyria.[8]

Given the hemolytic anemia and unconjugated hyperbilirubinemia, we interpreted the change in liver chemistries due to transient ferric accumulation in hepatic cells, as shown in liver biopsy. This aspect needs to be validated in clinical studies. Lead intoxication is not directly related to liver toxicity. Another case report shows a similar pattern of abnormal liver chemistry tests, without serological explanation.[11]

Our patient had a constellation of vague, nonspecific symptoms centered in neuropsychiatric conditions. Prolonged low levels of lead intoxication have been described in asymptomatic adults, possibly as low as 5–10 μg/dL. It may still have long-term effects on renal, cardiovascular, cognitive, and endocrine/sexual functions (a decrease of libido, altered sperm quality, and endocrine function).[12] These levels can be caused by a continuous release of lead by bone (the primary reservoir after absorption) without exposure to lead at the time of serological testing.[13] Structural changes in the brain can explain the neurocognitive effects of lead intoxication.[14]

Chelation treatment thresholds vary according to blood lead levels, symptomatic condition, and special circumstances (such as pregnancy). When blood lead levels exceed 80 μg/dL, chelation treatment is generally recommended, regardless of symptoms. Acute symptoms, such as abdominal pain, constipation, headache, and anemia, generally respond to chelation therapy.[15] This clinical case illustrates the need for clinicians (emergency physicians, internists, gastroenterologists, and surgeons) to recognize digestive manifestations of systemic diseases. This is of the utmost importance in cases with nonspecific clinical findings, which the acute presentation may affect the digestive system.

DISCLOSURES

Author contributions: P. Costa-Moreira, R. Coelho, and I. Pita wrote the manuscript. G. Macedo revised the manuscript. P. Costa-Moreira is the article guarantor.

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Informed consent was obtained for this case report.

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REFERENCES

1. Cullen MR, Robins JM, Eskenazi B. Adult inorganic lead intoxication: Presentation of 31 new cases and a review of recent advances in the literature. *Medicine.* 1983;62(4):221–47.
2. Prevention N-CfDCa. Lead, elevated blood levels—2016 Case definition. 2017 (https://wwwn.cdc.gov/nndss/conditions/lead-elevated-blood-levels/case-definition/2016/).
3. Adult blood lead epidemiology and surveillance—United States, 2005–2007. *MMWR Morb Mortal Wkly Rep.* 2009;58(14):365–9.
4. Levin SM, Goldberg M. Clinical evaluation and management of lead-exposed construction workers. *Am J Ind Med.* 2000;37(1):23–43.
5. Ross SW, Oommen B, Wormer BA, et al. Acute colonic pseudo-obstruction: Defining the epidemiology, treatment, and adverse outcomes of Ogilvie's syndrome. *Am Surg.* 2016;82(2):102–11.
6. Vanek VW, Al-Salti M. Acute pseudo-obstruction of the colon (Ogilvie's syndrome). An analysis of 400 cases. *Dis Colon Rectum.* 1986;29(3):203–10.
7. Thomson RM, Parry GJ. Neuropathies associated with excessive exposure to lead. *Muscle Nerve.* 2006;33(6):732–41.
8. Anderson KE, Bloomer JR, Bonkovsky HL, et al. Recommendations for the diagnosis and treatment of the acute porphyrias. *Ann Intern Med.* 2005;142(6):439–50.
9. Valentine WN, Paglia DE, Fink K, Madokoro G. Lead poisoning: Association with hemolytic anemia, basophilic stippling, erythrocyte pyrimidine 5'-nucleotidase deficiency, and intraerythrocytic accumulation of pyrimidines. *J Clin Invest.* 1976;58(4):926–32.
10. Cheson BD, Rom WN, Webber RC. Basophilic stippling of red blood cells: A nonspecific finding of multiple etiology. *Am J Ind Med.* 1984;5(4):327–34.
11. Friedman LS, Simmons LH, Goldman RH, Sohani AR. Case records of the Massachusetts General Hospital. Case 12-2014. A 59-year-old man with fatigue, abdominal pain, anemia, and abnormal liver function. *N Engl J Med.* 2014;370(16):1542–50.
12. HH RHG. Adult occupational lead poisoning. 2016 (www.uptodate.com). Accessed August 26, 2018.
13. Hu H. Poorly controlled hypertension in a painter with chronic lead toxicity. *Environ Health Perspect.* 2001;109(1):95–9.
14. Stewart WF, Schwartz BS, Davatzikos C, et al. Past adult lead exposure is linked to neurodegeneration measured by brain MRI. *Neurology.* 2006;66(10):1476–84.
15. Kosnett MJ, Wedeen RP, Rothenberg SJ, et al. Recommendations for medical management of adult lead exposure. *Environ Health Perspect.* 2007;115(3):463–71.