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CASE REPORT | PATHOLOGY

Lead Poisoning From a Ceramic Jug Presenting as Recurrent Abdominal Pain and Jaundice

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Abstract

Lead poisoning may present with non-specific symptoms that may result in unnecessary investigations. We report a case of acute lead poisoning in a previously healthy 28-year-old man who presented with recurrent abdominal pain, jaundice, constipation, and weight loss. An extensive diagnostic work-up was completed with inconclusive results. A detailed history revealed an unusual source of lead exposure. Chelation therapy resulted in substantial clinical and biochemical improvement.

Introduction

Acute abdominal pain has a broad differential diagnosis that includes life-threatening etiologies such as ruptured aortic aneurysm, intestinal infarction, intestinal perforation, as well as more common causes such as appendicitis, pancreatitis, cholecystitis, and inflammatory bowel disease.[1] Electrolyte abnormalities causing pseudo-obstruction, metabolic causes such as porphyria, and acute lead poisoning are also on the differential.[2] Unrecognized lead poisoning can be misdiagnosed and may lead to unnecessary gastrointestinal evaluations and even abdominal surgery.

Case Report

A 28-year-old previously healthy man presented with a 1-week history of worsening diffuse abdominal pain associated with anorexia and difficulty sleeping. He had no other gastrointestinal or systemic symptoms. Initial investigations showed an elevated ALT of 242 U/L, total bilirubin of 5.3 mg/dL, normal alkaline phosphatase and normal gammaglutamyl transpeptidase (GGT). An abdominal/pelvic ultrasound was normal and the patient was discharged.

One week later, he presented to another hospital with severe abdominal pain and jaundice. His physical examination was remarkable for both scleral icterus and diffuse abdominal tenderness. The laboratory testing showed persistent elevation of liver values, with indirect hyperbilirubinemia and hemoglobin of 12.2 g/dL. A abdominal computerized tomography (CT) scan and repeat abdominal ultrasound ruled out surgical causes for the pain. The patient was admitted to the hospital with a working diagnosis of acute hepatitis.

Celiac serology showed elevated anti-tissue transglutaminase antibody (anti-IgA tTG and IgG tTG) titre at 192 IU/mL. Esophagogastroduodenoscopy showed scalloping of the duodenal bulb and biopsies confirmed moderate to focally severe crypt hyperplastic villous atrophy with focal increase in the intraepithelial lymphocytes, which confirmed a diagnosis of celiac disease. Work-up for other causes of abnormal liver tests was unrevealing, including viral hepa-

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Figure 1. The inside of the jug showing erosions of the inner surface.

titis serology, magnetic resonance cholangiopancreatography, and liver biopsy. He was started on a gluten-free diet and his abdominal pain subsided over the following 11 days. His ALT and total bilirubin had decreased at hospital discharge. The working diagnoses were abdominal pain and elevated transaminases secondary to celiac disease, and unconjugated hyperbilirubinemia secondary to Gilbert's syndrome.

Two weeks later, he presented to a third hospital with recurrence of the acute abdominal pain, jaundice, constipation, and weight loss of 12 kg in 4 weeks. The initial investigations showed hemoglobin of 11.7 g/dL, ALT of 73 U/L, total bilirubin of 7.5 mg/dL, direct bilirubin of 0.6 mg/dL, and microscopic hematuria. His anti-tTG titre had decreased to 54.3 IU/mL while on the gluten-free diet. A repeat abdominal ultrasound was normal, and colonoscopy and magnetic resonance enterography showed no concerning findings. Further work-up to rule out hemolysis (lactate dehydrogenase, serum haptoglobin, and Coombs test), porphyria, paroxysmal nocturnal hemoglobinuria, and C1 esterase deficiency was negative.

On further review of the social, travel, and personal history, it was found that he purchased a ceramic jug from Greece 2 months earlier and used it to drink water daily (Figure 1). The jug tested positive for lead using a home test kit. A blood lead level was obtained and was markedly elevated at 89 µg/dL (reference range 0-5 µg/dL). Chelation therapy was initiated with dimercaptosuccinic acid (DMSA) 30 mg/kg/day orally divided into 3 doses for 5 days, followed by 20 mg/kg/day orally in 2 doses for 14 days. On subsequent follow-up his blood lead level had decreased to 23 µg/dL.

Discussion

The use of lead in ceramic food ware has an extensive history that dates back to ancient Egyptian times. The addition of lead compounds to silicate glass and glaze compositions improves their physical properties. However, if lead glasses or glazes are improperly formulated, toxic amounts of lead can migrate to food substances in contact with the defective surface of the food ware.[3] Exposure to lead can also occur in a variety of workplaces, such as manufacturing or spaces that require use of batteries, pigments, ammunitions, and other lead-containing products.[4]

After absorption, lead is distributed to the blood, soft tissues, and bones. In the blood, 99% of lead is bound to erythrocytes and 1% is free in plasma in exchange with soft tissues (kidney, brain, liver, bone marrow). Lead in the blood is excreted via the kidneys and has a half-life of 30 days in the presence of a normal kidney function. Lead in the skeleton has a half-life of decades and can be released during times of bone turnover such as hyperthyroidism, pregnancy, breast-feeding and menopause.[5]

Acute lead poisoning can present with the following symptoms and signs: colicky abdominal pain (lead colic), joint pain, constipation, anorexia, muscle aches, headaches, decreased libido, sleep disturbance, irritability, fatigue, anemia, nephropathy, confusion, encephalopathy, and seizures. Lead lines (bluish discoloration of the gingiva) are a non-specific finding. Chronic lead poisoning is the result of repeated long-term exposure and can be asymptomatic or may present with a spectrum of cardiovascular and neuropsychiatric diseases, which is associated with increased risk of death.[6]

Classic laboratory findings in patients with chronic lead poisoning include a microcytic anemia and basophilic stippling. Neither of these were found in our patient because his exposure occurred over a relatively short time period of 2-3 months. Basophilic stippling of the red blood cells is found in up to 60% of children with chronic lead poisoning, but the incidence in adults is unknown.[7] Elevated aminotranserases have been reported with acute lead exposures.[8] However, in our patient, we cannot exclude the possibility that his elevated liver enzymes and hyperbilirubinemia were related to celiac disease and Gilbert's syndrome, respectively, and were not related to his lead exposure.

Our case illustrates that lead poisoning can present with nonspecific clinical features and highlights the importance of focusing on a patient's home, hobbies, occupation, and travel history to help reveal possible lead exposures. The decision to initiate oral chelation therapy was based on a blood lead level well above 70 µg/dL and clinical features consistent with lead poisoning.

Disclosures

Author contributions: M. Mohamed wrote the manuscript, searched the literature, and is the article guarantor. A. Ugarte-Torres wrote the manuscript and searched the literature. H. Groshaus, K. Rioux, M. Yarema critically revised the manuscript.

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