

## Clinical Communications: Adults

### LEAD TOXICITY AS AN ETIOLOGY FOR ABDOMINAL PAIN IN THE EMERGENCY DEPARTMENT

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**Abstract—Background:** Abdominal pain is an uncommon presentation of lead toxicity in the emergency department (ED). However, making the diagnosis is important in avoiding unnecessary testing and the long-term sequelae of lead toxicity. **Objectives:** To illustrate possible presentations of abdominal pain secondary to lead toxicity and highlight the importance of taking a thorough patient history. **Case Report:** We report 2 patients who presented to the ED with abdominal pain and underwent extensive evaluations that did not reveal an etiology. At follow-up visits, their occupational histories revealed possible lead exposures from working for a bullet-recycling company. Tests revealed that each patient had extremely high lead levels and they were both treated for lead toxicity. Their abdominal pain resolved as their lead levels decreased. **Conclusion:** These cases demonstrate a rare but significant cause of abdominal pain in the ED. Although history-taking in the ED is necessarily brief, these cases underscore the importance of obtaining an occupational history. © 2014 Elsevier Inc.

**Keywords—**lead toxicity; abdominal pain; lead; occupational history

#### INTRODUCTION

Abdominal pain is one of the most common complaints in the emergency department (ED). Lead toxicity is a rare cause of abdominal pain, but has significant implications if the diagnosis is not made (1–3). Lead exposure still

occurs in certain occupational settings, and toxic levels can have significant morbidity and mortality if not diagnosed and treated (2–4). We present 2 patients who were seen in our ED within a 2-year period with a chief complaint of abdominal pain. Despite extensive evaluations, no etiology was found for either patient in the ED or the hospital. However, an occupational history led to the correct diagnosis in both cases. Although lead toxicity as a cause of abdominal pain has been reported in some of the occupational medicine and gastroenterology literature, it has never been reported in the emergency medicine literature.

#### CASE REPORTS

##### *Patient 1*

A 29-year-old man presented to the ED with a 2-week history of intermittent, crampy, lower abdominal pain that was associated with nausea, vomiting, and constipation. He stated that he had vomited approximately three times per day and had not had a bowel movement for the past week. He also reported a decreased appetite and had not experienced fever. He denied a history of similar symptoms or prior abdominal surgeries. He reported visiting another ED in the previous week and having a computed tomography (CT) scan of the abdomen and pelvis that was negative. On presentation to the ED, his vital signs were within normal limits and he appeared

in moderate distress. On physical examination, his abdomen was soft and mildly distended with decreased bowel sounds. He was moderately tender in the lower abdomen. The remainder of the physical examination was normal. His laboratory results demonstrated a white blood cell count of 9100/mm<sup>3</sup>, hemoglobin of 12.6 g/dL, and a hematocrit of 38.0%. The mean corpuscular volume was normal at 83.5 fL. The automated differential did not detect any ovalocytes or basophilic stippling. The electrolytes, blood urea nitrogen (BUN), creatinine, and glucose were all within normal limits. The aspartate aminotransferase (AST), alanine aminotransferase (ALT), serum protein, and alkaline phosphatase were all normal. The total bilirubin was 2.6 mg/dL and the unconjugated bilirubin was 2.2 mg/dL. The lipase was normal. An abdominal radiograph demonstrated mildly dilated loops of small and large intestine. A CT scan of the abdomen and pelvis demonstrated mild gaseous dilatation of the colon and no other pathology. Surgical consultation was obtained in the ED and was noncontributory. The gastroenterology service was able to perform endoscopy the following morning, so the internal medicine service admitted the patient for pain control overnight.

The patient's admission history and physical examination revealed that he worked for a company that recycled lead bullets. He was started on a bowel prep overnight in anticipation of endoscopy. He reported moderate improvement in his pain with bowel movements. The next day, he underwent upper endoscopy that showed mild to moderate gastritis in the antrum and a small hiatal hernia. A colonoscopy was also performed, but was suboptimal due to inadequate bowel prep. No pathology was seen on colonoscopy. There was a report that one of the physicians on the gastroenterology service performed a manual blood smear, saw basophilic stippling, and then ordered a lead level. The patient was discharged on the second hospital day with improvement of his abdominal symptoms. He was discharged on pantoprazole.

The patient's whole blood lead level returned after he was discharged and was 88 µg/dL. A medical toxicology consultation was requested and the patient was contacted to return to the toxicology clinic for further evaluation. An occupational history obtained at that time revealed that, for the past 3 months, he had worked for a company in North Carolina that recycled lead bullets from firing ranges. The company hired employees from other states as independent contractors and sent teams to firing ranges throughout the country to collect bullets and melt the lead into bricks for recycling. Large rubber barriers are commonly used at firing ranges to stop bullets. The teams of independent contractors shoveled dirt, bullets, and rubber fragments onto a conveyor belt. They then used leaf blowers to separate the dirt and rubber from the lead

bullets. The patient reported wearing a respirator "most of the time."

The patient was treated with succimer, 10 mg/kg twice daily for 10 days and was followed up through the medical toxicology clinic. Two weeks later his whole blood lead level was 54 µg/dL and his abdominal pain had completely resolved. His bilirubin level was normal at the time and his hemoglobin and hematocrit had increased to 13.6 g/dL and 40.5%, respectively. The patient did not return to work. Three months later, his whole blood lead level was 35 µg/dL. His hemoglobin and hematocrit were within normal range and he remained asymptomatic.

### *Patient 2*

A 42-year-old man presented to the ED three times during a 3-week period complaining of moderate abdominal pain, nausea, vomiting, and constipation. The abdominal pain was described as colicky, periumbilical in location, and radiating to the back. He also reported an unquantified weight loss over this 3-week period. He denied fever, shortness of breath, or chest pain. His abdominal examination on the first presentation to the ED was documented as soft and mildly distended with decreased bowel sounds. He was moderately tender in both lower abdominal quadrants. On the first hospital visit, laboratory tests demonstrated normal ALT, AST, protein, lipase, and alkaline phosphatase. His total bilirubin was mildly elevated at 1.4 mg/dL, and the unconjugated bilirubin was also 1.4 mg/dL. He was diagnosed with "generalized abdominal pain" and "suspicion of alcoholic gastritis" and discharged on promethazine and famotidine.

Thirteen days later he presented again with continued similar symptoms and pain in the left upper and left lower quadrants. The electrolytes, BUN, and creatinine were all normal. The white blood cell count was elevated at 14,600/mm<sup>3</sup>. The hemoglobin and hematocrit were 11.1 g/dL and 33.3%, respectively. The mean corpuscular volume was 85.6 fL. The manual differential showed occasional ovalocytes, basophilic stippling, and polychromasia. An occupational history during the second ED visit revealed that he worked recycling lead bullets. A whole blood lead level was ordered. He was diagnosed with "acute gastritis" and discharged.

The patient returned 5 days later with continued abdominal pain. At this time, he also described some insomnia, irritability, occasional headaches, dizziness on standing, and numbness and tingling in his right hand. At this visit, his white blood cell count had decreased to 10,400/mm<sup>3</sup>. His electrolytes, BUN, creatinine, glucose, and liver function tests were normal. The lead level that had been ordered 5 days earlier was looked up and was 105 µg/dL. It had not been followed up after the patient's previous discharge. The level was repeated on this

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