

Lead Poisoning and Recurrent Abdominal Pain

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Abstract: Severe abdominal colic because of lead poisoning is an uncommon condition in adults. The diagnosis of lead toxicity is often delayed and abdominal pain is mistaken for acute abdomen. We describe three blood brothers who were involved in pottery glazing and suffered from repeated episodes of severe abdominal pain, nausea, vomiting, constipation and anemia due to lead toxicity. The patients had a history of several hospitalizations and one or two unnecessary laparotomies. One patient had wrists drop and weakness of the fingers extensors. All three patients had microcytic microchromic anemia with basophilic stippling of the erythrocytes, lead lines in X-ray of the knee joint and high blood lead levels. A diagnosis of lead poisoning was made and a course of chelating treatment started. Motor neuropathy, anemia and all gastrointestinal symptoms disappeared. Our report highlights the importance of taking a detailed occupational history and considering lead poisoning in the differential diagnosis of acute abdominal colic of unclear cause.

Key words: Abdominal pain, Laparotomy, Lead toxicity, Nausea, Vomiting

Introduction

In adults, lead toxicity occurs most commonly during occupational exposure^{1,2}, but non-occupational exposure such as using contaminated herbal medicines or drugs has also been reported³⁻⁷. Inhalation and ingestion are generally two potential routes of lead absorption⁸. The body accumulates lead in blood, soft tissues and bone. Lead intoxication affects the central and peripheral nervous systems, renal function, vascular system and the gastrointestinal tract^{1,2}. It causes anemia, kidney impairment, neuropathy and gastrointestinal symptoms.

Lead inhibits heme synthesis and results in anemia by reducing the circulating levels of hemoglobin^{1,2}. The red blood cells become small (microcytic) and pale (microchromic). Basophilic stippling of erythrocytes due to clustering of ribosomes and microcytosis is most likely to be seen. Peripheral neuropathy is a common sign of lead toxicity in adults with occupational exposure^{1,2}. Lead-induced neuropathy commonly involves the extensor muscles

and on the radial and peroneal nerve causes “wrist drop” and “foot drop”. Sensory loss is minor in this neuropathy.

Diagnosis of lead poisoning is usually based on elevated blood lead level. Radiological examination may show “lead lines”, the dense transverse bands, across the metaphyses of long bones and along the margins of flat bones^{1,2}. Lead lines of the gingiva, Burton’s lines, may be seen. They are purple-blue lines within gingival tissue. The primary management of lead poisoning is source identification and exposure cessation^{9,10}.

In this report we describe three blood brothers who worked in the same workplace and suffered from nausea, vomiting, severe recurrent abdominal colic, constipation and anemia due to lead toxicity. Unsuspected lead poisoning resulted in delayed diagnosis and unnecessary laparotomies.

Case Reports

In May 2002, a 43-yr-old man was admitted to the local hospital in Hamadan, Iran with severe abdominal colic, nausea, vomiting and constipation. Physical examination demonstrated diffuse abdominal tenderness without

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peritoneal signs. He had a history of repeated episodes of similar complaints in the past. The symptoms had started some years earlier, causing several hospitalizations and two emergent laparotomies with no abnormal findings. Upper gastrointestinal endoscopy, colonoscopy, small bowel series and abdominal ultrasound showed no abnormalities. Regular physical examinations and blood tests including total and differential leukocyte count, serum amylase, uric acid, glucose, creatinine and liver function tests showed normal results. He had anemia (hemoglobin 11.0 g/dl), thus a search for the etiology of anemia was initiated. The peripheral blood films showed microcytic microchromic erythrocytes with basophilic stippling. An occupational history revealed that he has been a pottery glazer for 30 yr. Blood lead level was 99 $\mu\text{g}/\text{dl}$ and the X-ray of the knee joint showed lead lines (Fig. 1).

Few weeks later, his two younger brothers were also hospitalized due to acute abdominal pain, nausea, vomiting and constipation. The second patient, a 25-yr-old, had a history of a pottery glazing for 15 yr. All his symptoms had been recurrent for few years. He had also a history of several hospitalizations and was operated on twice because of acute abdominal pain. Regular blood tests, upper gastrointestinal endoscopy, colonoscopy, small bowel series and abdominal ultrasound showed normal findings. During the preceding six months he had a progressive weakness in his hands and was unable to work. The patient could not extend his wrists and hanged them flaccidly (Fig. 2). In the physical examination, weakness of the wrists and fingers extensors (wrist drop) was seen. Sensation of the wrists and fingers was intact. Needle electromyography of the arms and hands showed signs of motor nerve neuropathy including reduction of the compound muscle action potential amplitudes, segmental demyelination and axonal degeneration. Microcytic microchromic anemia (hemoglobin 10.6 g/dl) with basophilic stippling of the erythrocytes, lead lines in X-ray of the knee joint, Bruton's line of the gingiva and high blood lead level (77 $\mu\text{g}/\text{dl}$) were found.

The third patient, a 23-yr-old man with a job as a pottery glazer for five years, had also been hospitalized several times and operated on once as a result of similar complaints. Physical examinations, blood tests, upper gastrointestinal endoscopy, colonoscopy, small bowel series and abdominal ultrasound rendered no abnormalities. Electromyography of the arms and hands showed reduction of the compound muscle action potential amplitudes and axonal degeneration. Microcytic microchromic anemia (hemoglobin 9.3 g/dl) with basophilic stippling of the erythrocytes, lead lines in the X-ray of the knee joint and high blood lead level (104 $\mu\text{g}/\text{dl}$)

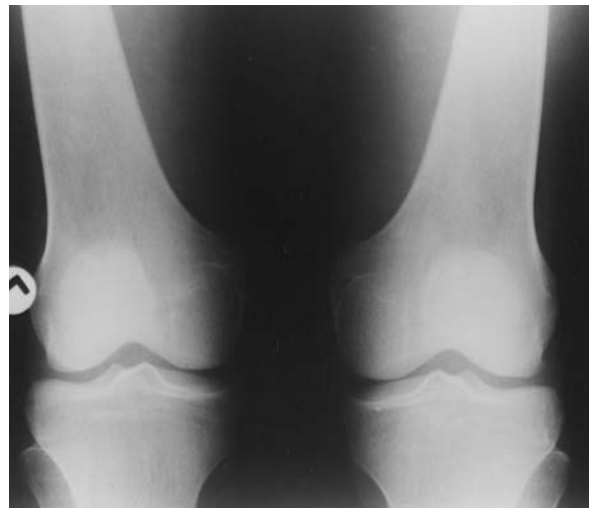


Fig. 1. Lead lines (dense transverse metaphyseal bands) in X-ray image of the knee joint (patient 1).



Fig. 2. Wrists drop, weakness of the wrists and fingers extensors (patient 2).

were found.

A diagnosis of lead poisoning was made and a course of chelating treatment started with intravenous calcium disodium ethylenediaminetetraacetate (CaNa₂EDTA) and continued with oral dimercaprol (BAL)^{1,2}. In addition, exposure to lead was terminated and the patients have changed their jobs. Motor neuropathy, anemia and all gastrointestinal symptoms disappeared. The patients have been free of symptoms for over three years by now.

Discussion

Lead is still used in ceramic industries in some Asian countries². Lead glaze is commonly used for hand-crafted pottery in Iran to produce certain colors and to help prevent

cracking. Lead is a significant occupational hazard in ceramic industries. Inhalation of airborne lead and ingestion of lead through contaminated hands are generally the common sources of lead absorption in lead-glazed ceramics workers^{2, 8}.

The city of Lalejin in Hamadan province is a main center of hand-crafted pottery in Iran. The city with 15,000 inhabitants has several hundred traditional pottery workshops and about a hundred glazing workshops. Lead, copper, zinc and magnesium are used in glazing workshops. These small workshops have only few workers and no preventive measures against heavy metal toxicity.

Lead toxicity is an uncommon cause of acute abdominal pain in adults. Several cases of acute abdominal colic due to lead toxicity have been reported^{3, 5, 6, 11–14}. This report shows that even today lead intoxication occasionally occurs and the suspicion of lead poisoning does not come readily to mind. The diagnosis of lead toxicity is often delayed and abdominal pain is mistaken for acute appendicitis, acute cholecystitis or other causes of acute abdomen.

The exact pathogenic mechanism of lead-induced abdominal colic is unknown. Three possible mechanisms have been proposed: changes in the visceral smooth muscle tone through lead action on the visceral autonomic nervous system, alterations in sodium transport in the small intestinal mucosa, and lead-induced interstitial pancreatitis¹⁵. Only a minority of patients with lead toxicity manifest severe abdominal colic. Genetic factors may make certain individuals more vulnerable to lead^{16, 17}. The fact that all three patients were blood brothers may suggest a genetic susceptibility for this adverse effect.

The classic form of lead neuropathy consists of weakness of the wrist and finger extensors¹⁸. The motor lead neuropathy usually develops after a high level of exposure and prognosis for recovery is good after prompt termination of exposure.

In summary, the present report highlights the importance of taking a detailed occupational history and considering lead poisoning in the differential diagnosis of acute abdominal colic of unclear cause. The monitoring of blood lead level in patients suffering from recurrent abdominal colic is a useful method to identify those with high blood lead level¹⁸.

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