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Case Report

ABDOMINAL PAIN DUE TO LEAD-CONTAMINATED OPIUM: A NEW SOURCE OF INORGANIC LEAD POISONING IN IRAN

Mohsen Masoodi MD•, Mohammad-Reza Zali MD, Mohammad-Javad Ehsani-Ardakani MD, Amir-Hoshang Mohammad-Alizadeh MD, Kazem Aiassofi MD, Rahim Aghazadeh MD, Ahmad Shavakhi MD, Mohammad-Hossein Somi MD, Mohammad -Hossein Antikchi MD, Saeed Yazdani MD

Although the incidence of occupational and adult lead poisoning has declined, the problem still exists. We encountered three patients with lead poisoning in Iran, all of whom associated with presented with diffuse abdominal pain, which was at times colicky in nature, anemia, constipation, nausea, vomiting, and slightly abnormal liver biochemistries. A history of opium ingestion was present in each of these patients. None of the patients reported known occupational exposure to toxins. Diagnoses of lead poisoning were confirmed through the detection of elevated blood lead levels. The cause of lead poisoning was attributed to the ingestion of contaminated opium. Opium adulterated with lead had not been previously recognized as a source of lead poisoning in Iran. It is, therefore, pointed out that lead poisoning should be considered as a differential diagnosis for acute abdominal colic of unclear cause in patients with opium addiction.

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Introduction

Lead toxicity has been recognized for thousands of years and still exists today. The majority of elevated blood lead levels in the adults come from workplace exposure.[1, 2] Inorganic lead is absorbed from the lungs or gastrointestinal tract.[3]

Lead is a toxic metal that affects many organ systems and functions in humans,[1, 3] and can present with nonspecific signs and symptoms such as abdominal pain, constipation, irritability, and anemia.[2, 4, 5] Owing to the increasing levels of safety at work, the incidence of occupational lead poisoning has decreased and new forms of non-occupational poisoning have emerged. Lead poisoning due to drug or heroin addiction has been reported in few studies.[6 – 11] In this report we present three patients from Iran with abdominal pain, elevated liver enzymes, and anemia due to ingestion of lead-contaminated opium.

Authors' affiliation: The Research Center for Gastroenterology and Liver Disease, Shaheed Beheshti University of Medical Sciences, Tehran, Iran.

•**Corresponding author and reprints:** Mohsen Masoodi MD, The Research Center for Gastroenterology and Liver Disease, Taleghani Hospital, Tabnak St., Evin, Tehran, Iran. P.O. Box: 19835-187, E-mail: masoodi47@yahoo.com.

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Case Reports

Case 1

A 34-year-old man who worked as a driver was hospitalized due to a ten-day history of recurrent bouts of abdominal pain, as well as nausea and vomiting. There was no known occupational exposure to toxins. The patient had a history of opium ingestion and continued to ingest opium at the time of hospitalization. His family history was unremarkable. On physical examination the patient had pain without guarding over the abdomen. His vital signs, neurologic, and rectal examinations were all normal.

Electrocardiogram, chest radiograph, abdominal ultrasonography, esophagogastro-duodenoscopy, and CT scan of the abdomen were all within normal limits. Laboratory tests revealed normochromic, normocytic anemia (hemoglobin: 11 g/dL), basophilic stippling of erythrocytes,

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elevated liver enzymes (ALT: 83 U/L, AST: 176 U/L) and total bilirubin (2.1 mg/dL), negative results for the HBs antigen and HCV antibody, and normal leukocyte count, ferritin, and renal function tests. The diagnosis of lead poisoning was then considered and confirmed by an elevated blood lead level (95 µg/dL) apresent symptoms and response to therapy. Whereby, symptoms improved after a five-day course of treatment with calcium EDTA and discontinuation of self-ingested opium. After one month of follow-up, laboratory data were normal and the patient was asymptomatic.

Case 2

A 57-year-old business man (peddler), was admitted to the hospital for progressive and markedly diffuse abdominal pain, at times colicky, as well as nausea and severe constipation for more than 30 days. The patient had a history of opium ingestion and cigarette smoking, and continued opium ingestion during the time of hospitalization. There was no known occupational exposure to toxins. His family history was unremarkable. On physical examination he had pain without guarding over the abdomen.

Laboratory investigations were as follows: hemoglobin, 10.5 g/dL; MCV, 82 fl: ALT, 87 U/L; and AST, 90 U/L. Renal function tests, urinalysis, bilirubin, alkaline phosphate, HBs antigen, HCV antibody, electrocardiogram, chest radiograph, esophagogastroduodenoscopy, colonoscopy, ultra-sonografy, and CT scan of the abdomen were all normal. The diagnosis of lead poisoning was then considered and confirmed by an elevated blood lead level (81 µg/dL) and the detection of lead in the opium sample. The patient's symptoms improved after four days of treatment with calcium EDTA and discontinuing ingestion of the opium. After 3 weeks of follow-up, laboratory data were normal and the patient was asymptomatic.

Case 3

A 45-year-old laborer, was hospitalized due to repeated attacks of severe epigastric and periumbilical pain which started two weeks prior to admission. There was no known occupa-tional exposure to toxins. He had a history of opium ingestion and had increased his opium intake in order to believe his abdominal pain decreased albeit without any benefit. His family history was unremarkable. On physical examination he had pain with mild tenderness over the abdomen. His vital signs, as well as neurologic and rectal examinations were all normal. Electrocardiogram, chest radiograph, abdominal ultrasonography, esophago-gastroduodenoscopy, and CT scan of the abdomen were all within normal limits. Laboratory tests showed normochromic, normocytic anemia (hemoglobin 9 g/dL), elevated liver enzymes (ALT: 80U/L, AST: 67 U/L) total bilirubin (1.4 mg/dL), negative results for HBs antigen, HCV antibody, normal leukocyte count, ferritin, and renal function tests. A diagnosis of lead poisoning was then considered and confirmed by an elevated blood lead level (37.5 µg/dL) and symptom response after four days discontinuation of opium intake. There was no need to use chelating agents in this patient. After one month of follow-up, laboratory data were normal and the patient was asymptomatic.

Discussion

Although the incidence of occupational lead poisoning has decreased, the problem still exists, and new forms of nonoccupational poisoning have emerged. Adult lead poisoning often goes unrecognized for long periods of time because of a low index of suspicion.[6, 12] Inorganic lead is absorbed from the lungs or the gastrointestinal tract and is a toxic metal that affects many organ systems and functions in humans.[1, 3]

Manifestations of lead poisoning vary from individual to individual. It may present with nonspecific signs and symptoms such as abdominal pain (lead colic), constipation, irritability, neuropathy, myalgia, joint pain, muscle aches, headache, anorexia, decreased libido, concen-tration difficulties, deficits in short-term memory, nausea, gingival lead lines, anemia, basophilic stippling of the RBCs, and nephropathy.[1, 7, 13, 14, 15] Combinations of abdominal pain and anemia,[2, 6, 12, 16, 17] as well as constipation[18, 19] are frequent symptoms of lead poisoning that were also present in our patients.

Patients with unrecognized lead poisoning presenting with symptoms of abdominal pain can be misdiagnosed as acute cholecystitis, chronic pancreatitis, and acute abdomen.[20, 21] In these instances, unnecessary gastrointestinal evaluations and abdominal surgeries are often performed.[5] Unrecognized cases of lead poisoning can also present with a prolonged history of unexplained abdominal pain and constipation, leading to hospital admission and numerous investigations.[19]

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Lead poisoning can cause liver damage ranging from increased liver enzymes[21 – 23] to hepatic failure.[10] In our patients, were increased, and after treatment and discontinuation of contaminated opium, liver enzymes converted to normal levels and anemia with basophilic stippling of the RBCs improved.

The majority of adult elevated blood lead levels come from occupational exposure. Lead exposure can occur in numerous work settings, such as manufacturing or use of batteries, solder, ammunitions, paint, car radiators, cable and wires, some cosmetics, ceramic ware with lead glazes, and tin cans.[1, 24] Other sources of lead poisoning include inhalation of fumes from burned car batteries, and ingestion of flaking paint, ingestion of some herbal medicines, consuming food cooked in nonstandard aluminum or brass containers, eating contaminated soil, drinking water from lead pipes, ingestion of solid metallic lead, automotive fuels, and ambient air quality.[4, 16, 22]

Inorganic lead poisoning due to intravenous injection or inhalation of heroin adulterated with lead has been recognized since 1989.[6, 7] Until then there were few reports of contaminated heroin as a source of lead intoxication.[8, 9, 25] Additionally, acute lead poisoning as a result of self-injection of lead and opium pills, crushed and suspended in water, has been reported.[10, 11, 23] In our patients, the source of lead poisoning was ingestion of lead-contaminated opium. The cause of this contamination is unknown, but it might have been due to the addition of lead for increasing the weight of the opium by salesman or smugglers.

In order to accurately determine the lead content, samples must be collected with lead-free equipment. To get reliable results, tests should be done by laboratories experienced in lead analysis, with intralaboratory quality controls and atomic absorption spectrometry as the method of choice. Diagnosis of lead poisoning is based on elevated blood lead levels (defined as equal or greater than to 25 µg/dL) and eliminating the source of lead exposure is milestone in treatment.[26] Lead levels in our patients also were above 25 µg/dL, and all of them were symptomatic and good response to treatment.

Many of the toxic effects of lead are reversible if lead poisoning is identified early and exposure to the source of contamination removed. Chelation therapy is needed in more severe cases to decrease blood lead levels faster, thereby facilitating clinical improvement.[3] Lead poisoning may be fatal, therefore, it should always be considered in the differential diagnosis of unexplained anemia or abdominal symptoms and whenever a disparity is observed between the symptoms and the abdominal findings in a patient with abdominal pain. In these cases, blood lead levels must be obtained as part of the survey for the correct diagnosis of abdominal pain of uncertain etiology. In addition, sources other than occupational exposures, such as contaminated opium should also be considered.

References

1 Agency for Toxic Substances and Disease Registry. *Toxicological Profile for Lead*. Atlanta, GA: US Department of Health and Human Services, Public Health Service; 1999: 587.

2 Tandon SK, Chatterjee M, Bhargava A, Shukla V, Bihari V. Lead poisoning in Indian silver refiners. *Sci Total Environ.* 2001; **281:** 177 – 182.

3 Fischbein A. Occupational and environmental exposure to lead. In: Rom WN, ed. *Environmental and Occupational Medicine*. Philadelphia: Lippincott-Raven; 1998: 973.

4 Hasegawa S, Nakayama K, Iwakiri K, et al. Herbal medicine-associated lead intoxication. *Intern Med*. 1997; **36:** 56 – 58.

5 Wolf C, Binder R, Barth A, Konnaris C, Rudiger HW. Chronic anemia and abdominal pain as a sequela of lead poisoning [in German]. *Dtsch Med Wochenschr*. 2001; **126:** 556 – 558.

6 Algora M, Martin-Castillo A, Zabala P, Fernandez MN. Lead poisoning due to drug addiction: a new source of poisoning with clinical interest and important epidemiological consequences [in Spanish]. *An Med Interna*. 1989; **6:** 483 – 485.

7 Antonini G, Palmieri G, Millefiorini E, Spagnoli LG, Millefiorini M. Lead poisoning during heroin addiction. *Ital J Neurol Sci*. 1989; **10:** 105 – 108.

8 Parras F, Patier JL, Ezpeleta C. Lead-contaminated heroin as a source of inorganic-lead intoxication. *N Engl J Med.* 1987; **316:** 755.

9 Fitzsimons EJ, Dagg JH. Lead poisoning in a drug addict; the intravenous injection of suppository extracts. *Br J Clin Pract*. 1982; **36:** 284 – 285.

10 Beattie AD, Briggs JD, Canavan JS, Doyle D, Mullin PJ, Watson AA. Acute lead poisoning: five cases resulting from self-injection of lead and opium. *Q J Med.* 1975; **44:** 275 – 284.

11 Chia BL, Leng CK, Hsii FP, Yap MH, Lee YK. Lead poisoning from contaminated opium. *Br Med J*. 1973; **1:** 354.

12 Zatlin GS, Senaldi EM, Bruckheim AH. Adult lead poisoning. *Am Fam Physician*. 1985; **32:** 137 – 143.

13 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 (Baltimore).* 1983; **62:** 221 – 247.

14 Hwang YF, Strickland GT, Chang NK, Beckner WM. Chronic industrial exposure to lead in 63 subjects. I. Clinical and erythrokinetic findings. *Southeast Asian J*

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Trop Med Public Health. 1976; **7:** 559 – 568.
15 Gignoux L, Cortinovis-Tourniaire P, Grimaud J, Moreau T, Confavreux C. A brachial form of motor neuropathy caused by lead poisoning [in French]. *Rev Neurol (Paris)*. 1998; **154:** 771 – 773.
16 Geier B, Abgrall JF, Robaskiewicz M, et al. Saturnine colic and hemolytic anemia due to ingestion of solid lead. Apropos of a case [in French]. *Ann Gastroenterol Hepatol (Paris)*. 1988; **24:** 193 – 195.
17 de Haro L, Prost N, Gambini D, et al. Lead poisoning in adults. Experience of the Poison Control Center of Marseille from 1993 to 2000 [in French]. *Presse Med*. 2001; **30:** 1817 – 1820.
18 Monte-Secades R, Garcia-Pais MJ, Rabunal-Rey R, Guerrero-Lombardia J. A 71-year-old woman with abdominal pain and constipation [in Spanish]. *Rev Clin Esp*. 2001; **201:** 609 – 610.
19 Horing E, Radtke KU, von Gaisberg U. Acute lead poisoning [in German]. *Dtsch Med Wochenschr*. 1991; **116:** 175 – 178.
20 Dequanter D, Lefebvre JC, Takieddine M, Belva P, Vaneukem P. An acute pseudo-cholecystitis [in French]. *Rev Med Brux*. 2001; **22:** 439 – 441.
21 Anderson NR, Gama R, Kapadia S. Herbal remedy poisoning presenting with acute abdomen and raised urine porphyrins. *Ann Clin Biochem*. 2001; **38:** 408 – 410.
22 Ibrahim AS, Latif AH. Adult lead poisoning from a herbal medicine. *Saudi Med J*. 2002; **23:** 591 – 593.
23 Beattie AD, Mullin PJ, Baxter RH, Moore MR. Acute lead poisoning: an unusual cause of hepatitis. *Scott Med J*. 1979; **24:** 318 – 321.
24 Centers for Disease Control (CDC) and Prevention. Potential risk for lead exposure in dental offices. *MMWR Morb Mortal Wkly Rep*. 2001; **50:** 873 – 874.
25 D'Alessandro-Gandolfo L, Macri A, Biolcati G, et al. An unusual mechanism of lead poisoning. Presentation of a case. *Recenti Prog Med*. 1989; **80:** 140 – 141.
26 U.S. Department of Labor, Occupational Safety, and Health Administration. 29 CFR Part 1926: Lead exposure in construction; interim final rule. *Fed Reg*. 1993; 58:84.

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