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Case Report

Acute lead poisoning in an opium user: a case report

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ABSTRACT

Background: Excess amounts of lead in serum may affect different organ systems and cause lead poisoning. This toxicity mostly happens in chronic and occupational settings. This report comprises the clinical presentation of an acute case of lead toxicity with a much uncommon source of poisoning: opium.

Case Presentation: A 25 year old man was presented to us with abdominal pain, nausea and vomiting, severe weight loss, generalized bone pain, and jaundice. He had six years history of addiction to oral and inhalation forms of opium. Pallor and jaundice were observed in his mucosa and bluish pigmentation was evident at the gum-teeth line. Hepatosplenomegaly and lymphadenopathy were not detected. Upper GI endoscopy was normal. Liver enzymes and indirect billirubin were increased; however, alkaline phosphatase was in normal range. Laboratory tests were indicative of hemolytic anemia without autoimmune origin. Bone marrow aspiration and biopsy were indicative of erythroid hyperplasia. According to the symptoms and the clinical symptoms of lead poisoning, the lead level was measured both in the serum and in the opium sample the patient used to use. 350mcg/dl of lead in the serum and the very high lead content of the opium sample confirmed the diagnosis; therefore, patient was treated with Calcium-EDTA and BAL that caused decrease of lead level and elimination of symptoms.

Keywords: *opium, lead poisoning,*.

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INTRODUCTION

Iran is currently one of the main pathways of opium trafficking from Afghanistan to the rest of the world, and consequently opium use is a matter of health significance in Iran.

One study has previously raised the concerns about the high lead content of opium in Iranian market (1), and one study has documented the high opium lead levels in one of Iran's major areas involved in opium use (2). However, this study presents an acute case of lead poisoning that was proved to be related to contaminated opium.

Case Presentation

A 25 year old man who was an inhaling and ingesting opium user was referred to our clinic with nausea, vomiting, and abdominal pain lasting for 2 months and becoming more severe recently. He had lost about 8 kg of weight in the month preceding referral, and was complaining of malaise, weakness, excess sweating, darkened urine color and generalized bone pain which was dominant at his back. There was no concurrence of symptoms and food consumption.

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Physical examination showed pallor and jaundice, which were apparent in his sclera and skin. Moreover, bluish pigmentation of the gum-tooth line was found in advance (Figure 1), but further investigation yielded no other remarkable findings.

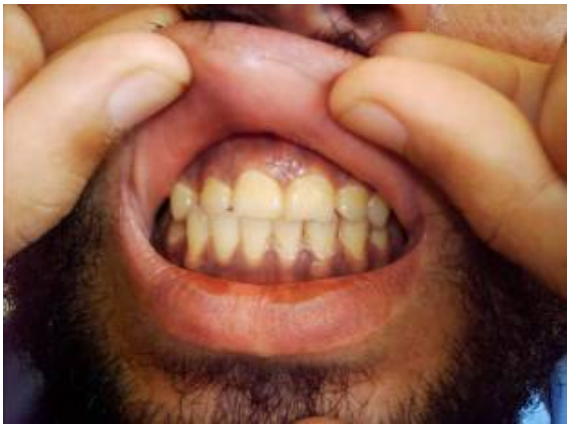

Figure 1. Bluish pigmentation of gum-teeth line.

The complete blood count and peripheral blood smear indicated normal white cell profile, normal platelet count, normocytic-hypochromic anemia, increased Reticulocyte count (8%) and 3% of Normoblasts. CRP was 1+ with an erythrocyte sedimentation rate of 11 mm/h. Total Iron Binding Capacity (TIBC) was normal (372 ng/ml). Wright's test and Direct and indirect Coomb's tests were negative. BUN, Creatinin and blood electrolytes were in normal range.

Liver enzymes, alanine amino transferase (ALT) and aspartate aminotransferase (AST), were elevated and therefore, complementary tests were requested (Table 1). In addition the patient was negative for certain viral markers: HBs Ag, HIV Ab and HCV Ab. In order to rule out autoimmune processes inside the liver and biliary system, serum immunologic markers were measured in advance; nonetheless, anti-mitochondrial antibody (AMA), anti-nuclear antibody (ANA), and anti-liver kidney microsomal antibody type 1 (ALKM-1) were reported to be negative.

Imaging studies including chest radiography, upper gastrointestinal endoscopy, pelvic and abdominal sonography, and colonoscopy yielded no remarkable finding. Bone marrow aspiration and biopsy was performed in advance and the results were indicative of erythroid hyperplasia.

Table 1. Complementary tests

Test	Value	Normal range
ALT (u/l)	118	10-40
AST (u/l)	93	12-38
ALP (u/l)	275	25-100
IgA (mg/dl)	325	4-350
IgG (mg/dl)	1550	600-1600
IgM (mg/dl)	210	55-300
Total Bilirubin (mg/dl)	4	0.3-1.2
Direct Bilirubin (mg/dl)	1	0.0-0.2
LDH (u/l)	395	208-387
Serum Ceruloplasmin (mg/dl)	20	18-45
AFP (ng/dl)	0.5	0-10

ALT: Alanin aminotransferase; AST: Aspartate aminotransferase; ALP: Alkaline phosphatase; LDH: Lactate dehydrogenase; AFP: Alfa-fetoprotein; u/l: Microgram/liter; mg/dl: Miligram/deciliter; ng/dl: Nanogram/deciliter

As the viral, metabolic and autoimmune causes of liver disorder were ruled out, the evidences of lead poisoning were assessed eventually both in serum and in the opium specimen that the patient used to consume. Serum lead level was reported to be 350 mcg/dl (normal range: 2-25 mcg/dl), and the lead content of the opium sample was very high, so the lead toxicity was considered the cause of patient's current state.

The patient was treated with 5 mg/kg of Calcium-EDTA and BAL. By decreasing lead level the symptoms disappeared. In treatment followup the abdominal discomfort and vomiting and extremities pain subsided after initiation of treatment over 2 weeks and the laboratory abnormalities such as anemia and LFT abnormality returned to normal level after 45 days

DISCUSSION

Lead poisoning arises from the excess amounts of lead in serum. Clinical manifestations are

highly dependent on individuals; acute, sub-acute and chronic exposure may make distinct manifestations in each case (3,4). If serum lead level exceeds 80 mcg/dl, the manifestation will be that of severe toxicity: abdominal pain (lead colic), constipation, muscular and skeletal pain, headache, anorexia, decreased libido, dizziness, short-term memory loss, and wrist/ankle drop due to axonal damage that primarily affects motor nerves. Anemia, nephropathy and basophilic stripping on blood smear might be present also (5).

Lower levels of lead (30-70 mcg/dl), may cause vague and nonspecific symptoms in adults such as myalgia, fatigue, irritability, insomnia, anorexia, impaired short-term memory, and difficulty of concentration (5-7). Chronic lead exposure has also been linked to hypertension (without evidence of renal damage) by some studies (8); probably, the risk of hypertension increases in postmenopausal women and people with low dietary calcium (8).

Chronic exposure is associated with a number of other potential findings such as decline of neurocognitive function and electrocardiographic evidence of conduction delay. In the setting of chronic exposure, lead toxicity can be symptomatic at lower blood levels; for example, lead levels as low as 20 to 29 mcg/dl, or even below 10 mcg/dl have been linked to an increase in circulatory and cardiovascular mortalities (9-13).

Chronic lead toxicity may cause pregnancy complications including increased number of miscarriages, stillbirth, low birth weight, and increased rate of eclampsia and preeclampsia. Men with chronic lead exposure (and blood lead levels between 40 and 70 mcg/dl) have been found in some studies to have an increased percent of dysmorphic sperm and decreased sperm concentration (9,10).

Nowadays, lead toxicity is mainly an occupational disease. Lead exposure can occur in numerous work settings, such as manufacturing or use of batteries, pigments, solder, ammunitions, paint, car radiators, cable and wires, some cosmetics, ceramic ware with lead glazes, and tin cans. Primary and secondary lead smelting and refinement are associated with considerable exposure (10).

The evaluation of adults with potential lead toxicity comprises taking medical and environmental history to identify potential sources of exposure and symptoms consistent with lead poisoning, looking for signs on physical examination, documenting and measuring environmental/occupational exposures, and seeking laboratory evidence to confirm excessive lead exposure or organ system damage. Laboratory tests that measure the items related to lead toxicity are the free erythrocyte protoporphyrin (FEP), complete blood count with smear morphology, blood urea nitrogen (BUN), serum creatinine, and urinalysis. The effect of lead on hemoglobin synthesis can be measured by free erythrocyte protoporphyrin (FEP) or zinc protoporphyrin (ZPP) , therefore both can be used as indicators of lead exposure (and effect) over the preceding three-month period (determined by the average 120 day lifespan of the erythrocyte). It is important to note that the BUN and creatinine are not sensitive indicators of renal damage, since these tests do not rise until the kidney function is over 50 percent lost (7-9). Depending upon the setting and patients' symptoms, the following tests might be performed: X-ray fluorescence/ Neurobehavioral test.

Reduction or removal lead exposure is the key first step in treating all cases of excessive lead absorption. In general, we tend to recommend chelation like 5 mic/kg/day of calcium-EDTA for individuals with blood lead levels greater than 80 mcg/dl. The treatment is continued by slow infusion for 5 days. In addition, chelation is also recommended for symptomatic individuals with blood lead levels between 60 and 80 mcg/dl (7).

DMSA (2,3-dimercaptosuccinic acid, Succimer) is an oral chelating agent that is FDA-approved for treatment of lead poisoned children, and also is effective in adults. The recommended dose from the manufacturer is 10 mg/kg three times per day for five days, followed by 10 mg/kg twice per day for two weeks; nonetheless, the dose could be increased, especially in heavier adults (7).

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Correction and apology:

In an article by Pourhoseingholi et al. in our April issue (GHFBB, 2008;1(2):71-77), the affiliation of Soghrat Faghihzadeh has been wrongly stated. With regret for any kind of inconvenience this mistake would have caused, the correct affiliation of the mentioned author will be included in the online version. It merits mentioning that the correct affiliation of the mentioned author is “Departments of Oncology, Taleghani Hospital, Shahid Beheshti University, M.C., Tehran, Iran”.