


# Bilateral Wrist Drop Due to Lead Poisoning in a Young Woman With Opium Addiction

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**ABSTRACT:** Opium addiction can cause symptoms in the central or peripheral neurological systems, as well as gastrointestinal disorders and anemia; in such situations, lead poisoning should be considered and chelation therapy should be started as soon as possible. In adults, lead poisoning is an unusual cause of abdominal pain. A common form of lead neuropathy includes weakness of the wrist and finger extensors. We describe a 24-year-old female who developed severe lead poisoning after 3 years of opium consumption, leading to gastrointestinal complaints and bilateral wrist drops.

**KEYWORDS:** Opium addiction, lead poisoning, neuropathy, wrist drop

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## Introduction

Lead poisoning due to the ingestion of lead-contaminated opium has become a serious health problem in Iran in recent years.<sup>1</sup> Because of its valuable characteristics, lead (Pb) is widely utilized and found in the human environment. Lead is a bluish-gray metal that is extremely poisonous. Its corrosion resistance, density, and low melting point make it a well-known metal with several applications in industry.<sup>2,3</sup> In humans, it can cause acute and chronic poisoning.<sup>2</sup> Lead is a hazardous substance that affects a variety of organ systems. Nonspecific clinical manifestations of lead poisoning include abdominal colic pain, loss of appetite, constipation, myalgia, decreased libido, irritability, seizures, and anemia.<sup>4</sup> Lead poisoning can cause abdominal pain that is misdiagnosed as an acute abdomen.<sup>5</sup> Car exhausts, contaminated food, industrial emissions, contaminated soil, and opium and its derivatives are all examples of occupational or environmental poisoning caused by lead.<sup>6</sup>

The majority of cases of lead poisoning, particularly lead neuropathy, are caused by industrial exposure. The typical type of lead neuropathy is characterized by weakness in the wrist and finger extensors, which results in wrist drop, also known as radial nerve palsy.<sup>7</sup> Lead poisoning can also affect any component of the central or peripheral nervous systems (CNS, PNS), depending on the quantity and duration of exposure.

Motor neuron degeneration commonly arises as a result of a high-level acute exposure.<sup>8</sup>

In adults, lead poisoning is an unusual cause of abdominal pain. Several examples of colic abdominal pain induced by lead poisoning have been described in scientific papers.<sup>9,10</sup> In this report, we describe a patient who suffered from colic abdominal pain and had motor neuron impairment in the upper extremities as a result of contaminated opium. She had no concept of the dangers of lead and had only received symptomatic therapy for a year.

## Case Presentation

In January 2020, a 24-year-old female was referred to an emergency room in northern Iran with no history of underlying disease and a history of ingesting opium orally for 2 to 3 years due to abdominal pain that had worsened for about a month. She also suffering from bilateral wrist drop. The patient also experienced wrist drop.

The abdomen pain was colic-like, exacerbating in the evenings and radiating to the back. The patient complained of weakness and fatigue, as well as nausea, vomiting, constipation, decreased appetite, weight loss, and paresthesia in the upper limbs. A year ago, she had an abortion. She was hospitalized at a local hospital about 3 months ago for seizures, rectorrhagia, and menstrual disorders. She had a neurology



assessment, a gastrointestinal endoscopy, a colonoscopy, and an abdominal ultrasound in our hospital, all of which showed her normal.

On examination, the blood pressure is 150/100 mmHg, the heart rate is 98 beats/minute, the respiratory rate is 22 breaths/minute, and the body temperature is 37.3°C. The conjunctiva appeared pale; the abdomen was soft with generalized tenderness. Deep tendon reflexes in the upper limbs, biceps, and triceps were absent. No sensory impairment was detected. Bilateral muscles force of the both upper limbs reduced and the muscular power in the upper extremities was 2/5 and 1/5 in the proximal and distal portions, respectively. The median and ulnar nerves were moderately affected, while the radial nerve was severely affected. Electromyography (EMG) and nerve conduction velocity (NCV) results, as well as a neurologic examination, revealed no sensory loss, but there was motor neuropathy in the upper extremities. The results of her laboratory investigation were shown on the Table 1.

The patient's anemia was of the microcytic-hypochromic type, and basophilic stippling of the erythrocytes was positive in the analysis of blood cells due to the blood lead level (BLL), which was 95.7 µg/dL which confirmed the diagnosis of acute lead poisoning. Then treatment with chelator began. Oral opium was discontinued immediately, methadone was started at a dose of 25 mg twice a day (BID) to control withdrawal symptoms, and lactulose syrup 10 mL 3 times a day (TDS) was administered. Chelation therapy, such as deep intramuscular injection (IM) of British Anti-Lewisite (BAL) at a dosage of 4 mg/kg (200 mg TDS for 5 days), has also been begun for lead poisoning. After the elapse of 4 hours since the initial dose of BAL, a continuous slow intravenous infusion (IV) calcium disodium edentate (CaNa<sub>2</sub>EDTA) with a dosage of 30 mg/kg; (200 mg 4 times a day for 7 days) was added. Following that, oral succimer was begun at a rate of 10 mg/kg every 8 hours for 5 days, then every 12 hours for 14 days.

Following treatment, BLL was reduced to less than 37 µg/dL, hemoglobin (HB) was increased to 11 g/dL, sensory loss and motor neuropathy in the upper extremities remained. There was no fecal or urinary incontinence, and the patient's gastrointestinal symptoms had improved significantly, with all para-clinical results being normal. Finally, the patient was referred to a physiotherapy and rehabilitation clinic for wrist drop. Unfortunately, the neurological complication of the lead remained, and the response to treatment was slow. However, the patient reported that the upper limb weakness had persisted at a one-year follow-up examination. Written informed consent was obtained from the patient for the publication of this case report. This study was conducted according to the Declaration of Helsinki Principles. Also, CARE guidelines and methodology were followed in this study.

**Table 1.** Baseline laboratory results.

PARAMETER	VALUE	REFERENCE VALUE	UNIT
Na	138	135-145	mEq/L
K	3.3	3.5-5	mEq/L
BUN	28	13-43	mg/dL
Cr	1	0.6-1.2	mg/dL
Ca	9.6	8-12	mg/dL
Mg	1.8	1.7-2.2	mg/dL
P	3.3	2.5-4.5	mg/dL
TG	123	Less than 150	mg/dL
Chol total	262	Less than 200	mg/dL
FBS	92	70-110	mg/dL
Amylase	109	80-86	U/L
Lipase	53	Up to 60	U/L
AST	41	10-40	U/L
ALT	42	45	U/L
ALP	289	80-306	U/L
Bil total	1.5	0.3-1.2	mg/dL
Bil direct	0.7	<0.3	mg/dL
Hb	8	12-16	gr/dL
Hct	24	36-48	%
Retic	1.1	0.5-2	%
iron	73	65-176	µg/dL
Vitamin B12	1,008	160-950	pg/mL
Lead	95.7	<10	µg/dL

Abbreviations: Bil, Bilirubin; BUN, Blood urea nitrogen; Chol, Cholesterol; Cr, Creatinine; Hct, Hematocrit; Retic, Reticulocyte; TG, Triglycerides.

## Discussion

Acute lead poisoning causes nausea, abdominal pain, headaches, cognitive disorders, and emotional impairment, whereas chronic lead poisoning causes fatigue, weakness, a decrease in brain and cognitive function, motor impairment, psychiatric disorders, and, in some cases, a decrease in nerve conduction velocity.<sup>11</sup> Manifestations of lead-induced encephalopathy include irritability, headache, fatigue, memory loss, weakness, limb paralysis, seizures, delirium, hallucinations, coma, and, eventually, death.<sup>12</sup> It should be mentioned that nervous impairment caused by lead-contaminated oral opium ingestion is uncommon, whereas most patients present with anemia

and gastrointestinal symptoms.<sup>13</sup> A combined neuropathy consisting of motor and sensory nervous system disorders has been reported in some studies. Lead-induced neuropathy typically affects the radial and peroneal nerves, as well as the nerves of the extensor muscles, resulting in wrist and foot drop, whereas sensory processing abnormalities are less prevalent.<sup>14</sup>

BLLs of more than 100 µg/dL are usually associated with encephalopathy. In the case presented, the patient's wrist drop was caused by a delayed diagnosis and continued opium consumption. The anemia and gastrointestinal problems improved after receiving immediate treatment. Chelating agent therapy can enhance lead excretion and reduce blood lead levels, but there is no conclusive evidence that these agents are beneficial in the treatment of neuropathic symptoms. Also, in our case, she developed sensory and motor neuropathy as a result of long-term exposure to high levels of lead.

The toxic effects of lead can damage both the peripheral and central nervous systems. Peripheral neuropathy, also known as lead palsy, is caused by degenerative changes in motor neurons and their axons, with myelin sheaths as a side effect.<sup>15</sup> It's a type of motor neuropathy that involves the upper extremities more than the lower, and it usually manifests as a symmetrical or asymmetrical wrist drop.<sup>16</sup> We are currently dealing with new types of non-occupational lead poisoning (opium addiction), and diagnosing lead poisoning in this situation necessitates a high index of suspicion as well as a thorough and exact history.

Beigmohammadi et al reported a male patient with an oral opium addiction and BLL of 200 µg/dL who suffered gastrointestinal and neurological symptoms such as weakness of the lower limbs, despite treatment with chelations. The patient then became quadriplegic. In our case, gastrointestinal and hematologic symptoms improved, but the wrist drop remained.<sup>17</sup>

As Meybodi et al. demonstrated in a study on a number of patients with lead poisoning, 25 patients have been addicted to oral opium for different periods of time. The BLL was  $61 \pm 145$  µg/dL. They have not found a significant statistical correlation between the duration of addiction and BLL in addicted patients. Most patients complained of nausea, anorexia, abdominal pain, weight loss, and constipation. The most common musculoskeletal symptom was muscle weakness. A number of patients have complained of symptoms such as pain in the extremities, paresthesia, wrist drop, and reduced vision and hearing. However, in our patient, despite the presence of gastrointestinal symptoms, muscle weakness, paresthesia, and menstrual disorders, she did not complain of vision and hearing loss.<sup>18</sup>

Moharriet al. reported 2 cases from a family with severe lead poisoning due to oral opium ingestion. They were

hospitalized with neurological symptoms (unconsciousness, delirium, and hyperirritability), gastrointestinal (icter and abdominal pain), and anemia. The older patient showed neuropathy symptoms, including paralysis and absent deep tendon reflexes, and a low level of consciousness. The patients' BLL indicated a high level of lead in both of them ( $\geq 150$  µg/dL). Unfortunately, one of the patients died due to a cardiovascular collapse.<sup>19</sup>

## Conclusion

Any patient with gastrointestinal complaints and hematological abnormalities, especially neuropathy or neurologic findings, should be investigated for lead poisoning, particularly if they have an opium addiction. Early detection of lead poisoning in opium users would benefit from BLL assessment, as a delay in treatment could result in irreversible motor neuron impairment. BLL is the method of early detection, that may be suitable for the patient.

## Author Contributions

FGh and ZZ were involved in the interpretation and collecting of data and editing of the manuscript. HA, ESB involved in writing and preparing the final version of the manuscript. MS was responsible for collecting data and submitting the manuscript. All authors reviewed the paper and approved the final version of the manuscript.

## Data Availability Statement

The data is available to the correspondent author and can be obtained upon request.

## Informed Consent

Written informed consent was obtained from the patient for the publication of this case report.

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