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Case Report

Subacute axonal motor polyneuropathy due to lead poisoning in an oral opium addict; a case report

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Abstract

Introduction: Lead poisoning usually occurs in occupational situations, however, there are sometimes non-occupational toxicities and opium-related lead poisoning has been reported recently. Neuropathy due to lead poisoning can also occur.

Case Report: In this study, a 43 year old man with a history of oral opium use with the complaint of progressive muscle weakness was reported. Muscle forces in the upper and lower limbs were 0/5 and 1/5, respectively. Serum lead level was 88.8 µg/dl. Electrodiagnostic studies were indicative of subacute moderate to severe axonal motor polyneuropathy. Following the treatment, the muscle force was improved and lead level was decreased.

Conclusion: Motor neuropathy due to lead poisoning is a rare but possible complication. Due to the increased incidence of opium related lead toxicity in Ardabil, Iran, we recommend to consider lead poisoning as one of the possible differential diagnosis of peripheral neuropathy to initiate early and proper treatment.

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Introduction

Lead poisoning is well recognized worldwide among adults and children. It can present with a variety of presentations including abdominal pain, constipation, irritability, and anemia. Fatigue, myalgias, arthralgias, renal failure, and neurologic deficits may also be seen.[1-3] Both the central nervous system and the peripheral nervous system become affected in lead exposure. Peripheral neuropathy may cause reduced motor activity causing muscular weakness, especially of the exterior muscles, fatigue and lack of muscular coordination.[1]

It is reported that lead is available in opioids used in Iran.[4] There are various case reports and case series regarding lead poisoning due to opium use.[5-7]

In the present study, an oral opium addict man patient with the complaint of abdominal pain and progressive quadreparesis has been reported.

Case Report

A 43-year old man addicted to oral opium referred to our emergency department, Imam Khomeini Hospital, Ardabil, Iran, with the complaint of a progressive muscle weakness of upper and lower limbs since the last 7 days. The patient had a history of a recurrent abdominal pain during the last month before visiting our hospital. He had no past medical history. He was heavy smoker (15 boxes a year) and used oral opium during the last 8 years.

The presenting symptoms were muscle weakness, muscle pain, fatigue, undocumented weight loss, palpitation, constipation, nausea and insomnia. At presentation, the patient was agitated and had delirium. Neurologic evaluation showed

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decreased muscle forces (0/5 and 1/5 in upper and lower limbs, respectively) with absent deep tendon reflexes (DTR). Other evaluations were normal. Laboratory findings showed mild normochromic anemia, with normal sodium, potassium, urea and creatinine, calcium and phosphorus and normal liver function tests. The patient had an erythrocyte sedimentation rate (ESR) of 60 and 1+ C-reactive protein (CRP). Peripheral blood sampling did not show any basophilic stippling. During the period that the patient presented to our hospital, there was an increased incidence of lead poisoning among oral opium addicts in the region under study. Serum lead level was 88.8 µg/dl which was indicative of lead poisoning.

Electromyography (EMG) and nerve conduction study (NCS) were performed which showed decreased compound muscle action potential (CMAP) amplitude, normal sensory nerve action potential (SNAP), normal F wave and H reflex. There was fibrillation, positive sharp waves (PSW) and polyphasic motor unit action potential (MUAP) in some muscles. Results were indicative of subacute moderate to severe axonal motor polyneuropathy. Given the involvement of upper limb more than lower limb, EMG-NCS findings and increased serum lead level, neuropathy due to lead poisoning was considered as the diagnosis.

The patient was treated with D-Penicillamine 250 mg QID, gluconate calcium IV QID, Vitamin B1 300, B6 and clonidine daily. In order to prevent opiate withdrawal signs and symptoms, 5 mg methadone three times a day was administered.

Six days after treatment, the proximal muscle force in upper and lower limbs improved to 2/5 and 4/5, respectively. DTRs were changed from absent to hypoactive. After 25 days, the muscle force was 5/5 in both upper and lower limbs with normal DTRs. The patient was discharged with full recovery of muscle force and DTRs. D-Penicillamine was ordered to be taken at home three times a day for two weeks.

The patient was followed for 6 months with no report of complications. Serum lead level was 9 µg/dl at the last follow-up.

A written informed consent was received from the patient for publishing his information and images.

Discussion

Peripheral neuropathy refers to disorders of the peripheral nervous system. These disorders have numerous causes and diverse presentations; various systemic diseases, toxic exposures, medications, infections, and hereditary disorders can cause peripheral neuropathy. Metabolic disorders like diabetes, hypothyroidism, and nutritional deficiencies are the most common and treatable forms. Complete blood count, evaluation of electrolytes and metabolic profile, ESR and CRP, thyroid-stimulating hormone (TSH), blood sugar and vitamin B12 levels are necessary to evaluate a patient with peripheral neuropathy.[8]

It has been shown that adulteration of lead opioids could cause severe lead toxicities.[9] In the last few months, there was increased rate of acute and persistent abdominal pain among addicts in the region under study which increased the possibility of lead poisoning. Therefore, lead levels were evaluated among all patients with a history of oral opium addiction with possible symptoms of lead poisoning.

The peripheral nervous system is a major target organ in lead intoxication. It is important to diagnose lead neuropathy, as it is potentially reversible, hence other systemic complications can be prevented with early detection and treatment.[1,10] Most cases of lead neuropathies are reported among patients with industrial exposure,[10] however, there is only two previous reports regarding quadriparesis and neuropathy due to lead poisoning among the oral opium users.[7,11]

In both cases above, the blood lead level was > 200 µg/dl,[7,11] however, this rate was 88.8 µg/dl for the patient in the present study. Although there is a generally weak relationship between the development of lead neuropathy and blood lead levels,

however, neurologic symptoms usually occur in higher lead levels.[10] EMG-NCS are performed for the evaluation of peripheral neuropathy, especially if the diagnosis remains unclear.[8] EMG-NCS in the present study and two other studies[7,11] were indicative of motor neuron disease.

With the termination of the source of exposure and proper treatment, these patients usually fully recover,[10] as seen in the patient of the present study and two other case reports.[7,11]

Conclusion

Motor neuropathy due to lead poisoning is a rare but possible complication. Due to the increased incidence of opium related lead toxicity in the region under study, we recommend to consider lead poisoning as a possible differential diagnosis of peripheral neuropathy to initiate early and proper treatment.

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None.

Authors' Contribution

All authors have read and approved the paper. Both the researchers participated in patient diagnosis, treatment, data collection, writing, critical revision and drafting of the paper.

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Conflict of Interest

Authors have no conflict of interest.

Ethical Approval

The study was approved by ethics committee of Ardabil University of Medical Sciences.

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