

QUADRIPLEGIA DUE TO LEAD-CONTAMINATED OPIUM

- Case Report -

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Abstract

Purpose: Utilization of lead-contaminated opium may lead to severe motor neuron impairment and quadriplegia.

Case report: Forty years oriented old male, opium addict, was admitted to the ICU, with headache, nausea and abdominal pain, and weakness in his lower and upper extremities without definitive diagnosis. The past medical and occupational history was negative. Laboratory investigation showed; anemia (Hb 7.7 g/dl), slightly elevated liver function tests, elevated total bilirubin, and ESR. Abdominal sonography and brain CT scan were normal. EMG and NCV results and neurologic examination were suggestive for Guillain-Barre. He underwent five sessions of plasmapheresis. Blood lead level was $>200 \mu\text{g/dl}$. He received dimercaprol (BAL) and calcium disodium edetate (CaEDTA) for two five days session. Upon discharge from ICU all laboratory tests were normal and blood lead level was reduced, but he was quadriplegic.

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Conclusion: The delayed treatment of lead poisoning may lead to irreversible motor neuron defect.

Keywords: Lead, Lead poisoning, Opium, Quadriplegia.

Introduction

Lead exposure is arguably the oldest known occupational health hazard with the potential for causing irreversible health effects, before it is clinically recognized. Occupational lead poisoning has been a recognized health hazard for more than 2000 years¹. Classic form of lead neuropathy consists of weakness of the wrist and finger extensors². But any part of the CNS or peripheral nervous systems can be affected by lead intoxication, depending on the level and duration of exposure³. The occurrence of motor neuron disease usually develops after an acute high level of exposure.

We present a case of pure motor neuron impairment in both upper and lower extremities from contaminated opium.

Case Report

A clerk forty years male was admitted to hospital with the complaint of paresthesia in both upper and lower extremities. A month earlier he had suffered several times of headache, nausea and abdominal pain and with no definitive diagnosis. Two weeks prior to his complaints, he developed weakness over his lower extremity which progressed to upper arms, upon admission to intensive care unit. He was alert, oriented but complained of nausea and abdominal pain. His vital signs were normal.

Physical examination revealed diffuse abdominal tenderness with no guarding and liver edge was palpated 2 cm below the costal margin. Neurologic examination showed decreased deep tendon reflexes in lower extremity (+1) and muscle strength in proximal and distal part of extremity were 2/5 and 3/5 respectively.

No sensory symptoms were detected. Later in the day he developed quadriplegia with no sensory involvement. In spite of slightly involved

respiratory muscles he had no obvious respiratory distress and could move his neck and shoulder. Laboratory investigation showed; anemia (Hb 7.7 g/dl), slightly elevated liver function tests, total bilirubin (4.2 mg/dl), and ESR (80 mm). Abdominal sonography and brain CT scan were normal.

The patient was evaluated by a neurologist. Electromyography (EMG) and nerve conduction velocity (NCV) results and neurologic examination were suggestive for AMAN Guillain-Barre (Acute Motor Axonal Neuropathy) and he underwent five sessions of plasmapheresis.

Two days later he developed severe respiratory failure, his trachea was intubated and mechanical ventilation started. He showed no signs of improvement. He was reexamined and laboratory data were reviewed. It was discovered that the patient was heavy oral opioid abuser and through out his course of hospital stay, he received opium by visitors. Blood lead level was $>200 \mu\text{g/dl}$ which confirmed the diagnosis of acute lead poisoning.

Gastrointestinal decontamination was performed and at the same time dimercaprol (BAL) 3 mg/kg IM, every four hours started. After the elapse of four hours since the initial dose of BAL, continuous slow IV infusion of calcium disodium edetate (CaEDTA) 30 mg/kg/day, was added. This protocol was continued for two five days session with an interval of three days washout period. Blood lead level was reduced to $62 \mu\text{g/dl}$.

Following treatment, oral succimer was started for three days. Patient's anemia was corrected by transfusion of 3 units of packed RBCs and he improved steadily and was successfully weaned from ventilator. Upon discharge from the ICU all laboratory tests were normal, blood lead level was less than $20 \mu\text{g/dl}$, and neurologic examination showed no sensory loss, but motor neuropathy in upper and lower extremities was still present. There was no fecal or urinary incontinence. Patient was referred to a rehabilitation center.

Discussion

Occupational lead poisoning is decreasing because of primary prevention through the use of engineering controls, personal protective

equipment and good work practices. At present we are facing new forms of nonoccupational lead poisoning⁴ and diagnosis of lead poisoning in this setting requires a high index of suspicion and a careful precise history. The infrequency of classic diagnostic signs and the nonspecific nature of symptoms frequently contribute to misdiagnosis⁵.

The toxic effects of lead can affect both peripheral and central nervous systems. Peripheral neuropathy or lead palsy is due to the degenerative changes in the motor neurons and their axons, with secondary effects involving the myelin sheaths^{6,7}. It is a pure motor neuropathy affecting the upper more than the lower extremities, mostly presenting as a symmetric or asymmetric wrist drop³. Lower Extremity involvements also may occur.

In our case, while in hospital the patient had the opportunity to be visited by one of his relatives through whom he received opium. Poor history taking, acute onset of neurologic signs and similarities of NCV and EMG findings with that of Guillain Barre contributed to misdiagnosis and mistreatment of patient. Later we found out the patient had a long history of opium abuse and for the previous two months ago he had started taking opium orally.

There are reports of lead poisoning due to heroin and opium addiction^{8,9,10,11,12}. Upon reviewing of literature, only one report was found of flaccid quadriplegia and respiratory paralysis following acute lead poisoning¹³.

Relationships between blood lead concentrations and clinical findings have generally been based on sub acute and chronic exposure, and interindividual variability in response to high levels may result following acute extensive exposure. Overt neuropathy is usually associated with blood lead concentrations greater than 100 µg/dl.

In the case presented the delayed diagnosis and the continuous receipt by patient of opium, resulted in quadriplegia and respiratory paralysis. Following immediate treatment respiratory paralysis resolved and patient was successfully weaned from ventilator with little improvement of the quadriplegia. Therapy with chelating agents can enhance lead excretion and these agents are able to lower blood levels, but there is no

conclusive evidence that those agents help in the resolution of neuropathic symptoms^{3,14}.

Taking lead contaminated opium was the source of lead poisoning in this patient. It seems that drug dealers might have added lead to opium in order to increase the weight of the opium.

We recommend that any patient complaining of gastrointestinal complaints and hematological changes and especially with neuropathy or neurologic findings, should be screened for lead poisoning. High blood lead level and delay in treatment may lead to irreversible motor neuron defect.

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