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Motor Neuron Disease Due to Exposure to Lead

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Abstract Motor neuron disease as a manifestation of lead poisoning is rare, we report a 32-year-old man with a history of heavy oral opioid abuser that present with progressive flaccid quadriparesis and dysarthria during 2 weeks. In Electromyogram (EMG) and Nerve Conduction Study (NCS}, there was evidence of motor neuron disease. He was found to have high blood lead levels and was diagnosed to have lead poisoning. He was treated with d-Penicillamine for 8 weeks that his symptoms improved markedly. This paper focuses on the presentation of lead poisoning with motor neuron disease and concludes that any patient with motor neuron disease and suspicious to Amyotrophic lateral sclerosis (*ALS*), due to the reversibility of symptoms, lead poisoning should be considered.

Keywords Motor neuron disease, Flaccid quadriparesis, Lead poisoning

1. Introduction

Lead is a toxic heavy metal and is a potent occupational toxin. Lead poisoning is a serious fatal condition. It commonly results of inhaling the dust of inorganic lead salt, painting, and lead smelting and in children is by ingestion from window sills and painting plaster walls [1, 2]. Another uncommon exposure to lead is from contaminated opium [3]. Classic manifestations of lead poisoning are abdominal pain, anemia, motormononeuropathy in the distribution of the radial nerve, bilateral foot drop and less commonly is Guillain-Barre-like polyneuropathy [5]. Motor neuron disease due to lead toxication is very rare. We report a case of acute motor neuron disease due to chronic exposure to lead contaminated opium.

2. Case Report

A 32-year-old Iranian man presented to the hospital with 2 weeks history of dysarthria and upper limb weakness more proximal which progressed to lower limbs. Two weeks prior to his weakness, he also experiences abdominal pain, constipation, nausea and headache that was refractory to treatment. His weakness was very progressive that was very disabled on the day of admission. Past Medical and family history were noncontributory. On habit history, the patient was a heavy oral opioid abuser. In physical examination, His vital signs were normal, he was slow to respond to questions and there is blue lining at the dental margin of the gums. His abdomen revealed generalized tenderness. Neurologic examination showed decreased all deep tendon reflexes, muscle strength in proximal and distal upper limbs were 1/5 and 2/5 respectively and in lower limbs were 2/5 and 3/5 respectively. Sensory examination was completely normal. laboratory tests showed anemia (7.8mg/dl), elevated liver function test (ALT:55, AST:54, ALP:251), Bilirubin:2.3, ESR and CRP was normal, nerve conduction velocity (NCV) was completely normal, but electromyography (EMG) show neurogenic pattern with spontaneous activity (SA) in all distal and proximal, paraspinal and tongue muscles. The EEG shows generalized theta activity of 5-7 Hz. The blood lead levels were 256 µg/dl and blood level of arsenic was 0.15 g/dL that confirmed the diagnosis of motor neuron disease due to lead poisoning. The patient was started on d-penicillamine1000 mg/day for 8 weeks, at the follow-up visit, after 8 weeks he had marked improvement, in the examination, muscle strength was 4/5 in upper and lower limbs and lead levels reached 45 µg/dl.

3. Discussion

Inorganic lead is a toxic metal that affects many organ systems, including brain, liver, kidney, peripheral nerve, bone marrow, muscles and joints. In addition to the common route of toxication that absorbed from the lungs or the gastrointestinal tract, poisoning due to intravenous injection with lead has been reported [6]. The poisoning in our patient was from ingestion of contaminated opium that may be due to increasing the weight of the opium that is very uncommon. The clinical manifestations of lead poisoning are different in children and adults, in former usually present with anorexia, apathy, nausea, vomiting, abdominal pain, drowsiness that ultimately progress to stupor, seizure and coma [7]. In adults, encephalopathy is rare and usual manifestations are abdominal pain, anemia, fatigue and peripheral neuropathy

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[8]. Actually the peripheral and central nervous system appear to be sensitive to lead-induced toxicity, in the brain appear that lead cause disruption of blood-brain barrier and subsequent passage of plasma into the brain and cause brain edema and in peripheral nerve cause degenerative change in axon with secondary involving the myelin sheaths that predominantly affect peripheral motor nerve [9, 10]. In our patient abdominal pain, weakness, fatigue and anemia precede the involvement of nervous system, then developed insidiously motor neuron disease and mild encephalopathy. The presentation of lead poisoning with motor neuron disease is very rare, but some researchers believe that this does not exclude the possibility that lead can affect the motor neuron [11]. There are many genetic and environmental factors that associated with motor neuron disease including developmental disease, heavy metals, smoking, agricultural chemicals, electromagnetic fields and stressful activity. It seems that the cumulative effect of this factor increases the risk of this disease [12, 13]. According to Campbell and colleague from 74 cases of motor neuron disease, 15% had a history of extensive exposure to lead, compared with 5.4% of a control group [14]. In considering the possible reversibility of lead-induced motor neuron disease and poor prognosis in amyotrophic lateral sclerosis (ALS) in any patient with diagnosis of ALS, lead poisoning should be considered.

4. Conclusions

Motor neuron disease as a manifestation of lead poisoning is rare, but in any case with motorneuron, especially atypical presentation and associated with anemia, abdominal pain and confessional state, lead toxicity should be considered because any delay in diagnosis and treatment cause severe disability like ALS.

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