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CASE REPORT

Hyper Acute Quadriplegia with Chronic Lead Toxicity; a Case Report

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Abstract: Industrial lead toxicity is more common among miners. This type of toxicity occurs in two forms: acute and chronic. Chronic toxicity is associated with different levels of brain dysfunction, motor impairment, cognitive dysfunction, and neuropsychiatric problems, including depression, anxiety, irritability, and emotional disorders. However, quadriplegia induced by chronic toxicity is very rare. Here we report a 37-year-old male patient with a history of desert hunting, where he used to roll lead bullets in his mouth, who was admitted with sensory impairment, muscle weakness, and quadriplegia and final diagnosis of lead toxicity.

Keywords: Lead poisoning, nervous system, adult; quadriplegia; emergency service, hospital; case report

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1. Introduction

Industrial lead poisoning is one of the oldest types of occupational poisoning, which dates back to at least 5000 years ago. Nonorganic lead can be found in workers of ceramic glaze, food canning, color, and cosmetic companies. There is also nonorganic lead in drinking water, industrial dust, and motor vehicle exhaust. Lead levels are higher among adult workers who are in contact with industrial lead. This type of lead is mainly absorbed through the lungs and gastrointestinal tract (1, 2).

Neurotoxicity occurs due to physiological and neurological changes, which are caused by exposure to toxic agents. It may cause changes in cognition, memory, and mood or result in the development of psychiatric disorders (3). Neurotoxicity induced by heavy metals, including lead, mercury, and arsenic, has been explored more. This type of toxicity has two common forms: acute and chronic. Symptoms of acute poisoning include nausea, headache, cognitive problems, and emotional disorders.

Chronic exposure often occurs in industrial workplaces. In chronic exposure, symptoms are mostly neurological and psychiatric, including depression, anxiety, and irritability. Chronic exposure to lead may cause symptoms, such as fatigue, brain dysfunction, motor impairment, and cognitive dysfunction in general (4). Studies show that reduction of sensory responses and cognitive impairment occur in male workers with blood lead levels ≥ 40 μg/100ml (5). Peripheral motor neuropathy and reduced velocity of peripheral nerve conduction have been also reported in some cases of chronic lead toxicity (6, 7).

At high doses, lead toxicity causes irritability, headache, mental fatigue, reduced concentration, memory loss, tremor, and hallucinations. It may even result in quadriplegia, seizure, delirium, coma, or death. In the event of lead toxicity, early diagnosis, careful evaluation, and immediate treatment are very important (8). Therefore, it is necessary to be familiar with the rare symptoms of chronic lead toxicity.

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2. Case Presentation

The patient was a 37-year-old married man, who was a hunter of wild desert animals and used to roll lead bullets in his mouth. In the spring of 2015, he was referred to the hospital with abdominal pain and tenderness which had initiated almost 30 days before his admission. The patient was admitted to the surgical gastroenterology department due to abdominal pain and tenderness, weight loss, loss of appetite, and diffuse itching. On the tenth day of hospitalization, the patient suddenly developed sensory impairment, muscle weakness, and quadriplegia. With suspicion to super acute toxicity, he was transferred to the poisoning department.

The results of abdominal and pelvic computed tomography (CT) scans were normal, just like the results of endoscopy and colonoscopy. Moreover, the results of Doppler ultrasound and brain magnetic resonance imaging (MRI) were normal. Electromyography (EMG) and nerve conduction velocity (NCV) suggested axonal neuropathy in all the limbs.

The patient's neurological symptoms, such as motor neuropathy, quadriplegia, walking disability, and paresthesia, were exacerbated since the time of admission. Cognitive disorders, including thought disorders, accompanied by distress, anxiety, and delirium, were evident in the patient. Laboratory findings showed increased levels of liver enzymes (AST: 220 unit/L, ALT;120 unit/L), bilirubin(Total: 3 mg/dl with Direct: 1/2 mg/dl), lactate dehydrogenase (LDH: 750 unit/L), amylases (220 unit/L), leukocytosis (22000/m^3), and anemia (Mean Hemoglobin: 8.5 g/dl, MCV: 75). No basophilic stippling was observed in the peripheral blood smear during hospitalization.

The patient's medical history indicated chronic abdominal pain about four years before his referral for which he was hospitalized for several days and was discharged with a good general condition. With suspicion of lead toxicity the serum lead level was measured, which indicated a lead level of 150 μcg/dL. Considering the likelihood of acute lead poisoning with repeated attacks, chelation therapy was applied according to the standard protocols of the medical toxicology book. Therapeutic instructions included Livergol tablets, adenosine monophosphate (AMP), bronchoalveolar lavage (BAL), ethylenediaminetetraacetic acid (EDTA), calcium, and sodium. During treatment with BAL, the patient experienced stress disorders, exacerbated delirium, and tachycardia.

Methadone was initiated to overcome the suspected drug withdrawal syndrome. However, the symptoms did not resolve, and sedative medications such as midazolam were used to manage them. After two days of treatment with BAL, which led to restlessness, distress, and tachycardia, BAL treatment was temporarily terminated. After stabilization, BAL was continued in the intensive care unit (ICU).

BAL was continued with the same dosage as the patient was hospitalized in the ICU. His general condition gradually improved, and BAL-induced complications (tachycardia, hypertension, and distress), which persisted for three to five days, were resolved. Once the lead level reached 120 μcg/dL, treatment was continued with ETDA+BAL with the same dosage. After three days of treatment, the lead level reached 85 μcg/dL. Therefore, BAL was terminated and substituted with oral succimer.

After three days of treatment with succimer, the lead level reached 65 μcg/dL. At this lead level, BAL and EDTA administration was terminated, and succimer was continued with the same dosage. During treatment, the ferritin level became normal. After 15 days of treatment, the patient's anemia was improved and the level of liver enzymes gradually decreased and eventually reached the normal level after three weeks of treatment. After four months of treatment with succimer, the lead level reached 60 μcg/dL; treatment continued with succimer (divided doses every 12 hours). However, neurological symptoms, specifically muscle weakness and neuropathy, remained unchanged. With proper physiotherapy, most disorders, including muscle weakness of the upper and lower extremities, improved. After one year of follow-up, he was completely alert, without any neurological deficits; his cognition and perception also improved.

3. Discussion

Several studies have shown that industrial lead exposure can cause memory impairment and reduce the processing speed, reading comprehension, motor skills, and executive functions. Moreover, anxiety, depression, and phobia are probable in these patients. In our study, the patient suffered from tremor, paresthesia, peripheral neuropathy, quadriplegia, and walking disability. In a case report by Beig Mohammadi et al., entitled "Quadriplegia induced by lead-contaminated opium", a patient was examined with quadriplegia. Despite the resolution of all symptoms and reduction of blood lead level, the patient was discharged with quadriplegia (9). In the present report, which is only different from the mentioned study in terms of the contamination source, the patient was completely recovered after the treatment period and one-year follow-up.

A study by D. A. Gidlow showed that in industrial workers with lead levels above 40 μg/100ml, neurotoxicity was characterized by motor deficiency and cognitive problems and even in some cases by peripheral neuropathy. Memory and concentration problems, besides visual impairment, occur at lead levels higher than 50 μg/100ml (1). A study by Sansar W, which was conducted on mice, found that lead levels of 100 μg/100ml can cause changes in the glial and neuronal systems and result in functional and behavioral impairments

(10).

In a study of 2930 cases, Robert A. Goyer concluded that infants, whose umbilical cord blood lead levels were higher than 10 μg/100ml, had sensory, motor, and visual impairments, as well as cognitive problems during development (11). Moreover, in a review study, Lisa H. Mason concluded that high levels of lead toxicity in workers cause seizure, lethargy, and coma. In acute cases of lead toxicity, reversible neuropathy occurs (8).

In another study, Wilhelm M found that cognitive and behavioral changes occur at a lead level of 100 μg/100ml; these disorders were more serious in younger patients (12). In our study, long-term lead exposure and high lead level resulted in neuropathy, numbness, and quadriplegia, which gradually resolved with proper chelation therapy. Moreover, in a study on industrial workers Baker EL found that workers with lead levels above 40 μg/100ml experienced problems, such as mood and emotional disorders, exacerbated depression, confusion, anger, and increased tension and fatigue. In the long run, these workers experienced complications, such as memory, cognitive, visual, and verbal impairments (13). In this study, motor problems were not reported. Slight motor problems have been reported in several studies, although quadriplegia and walking disability were not found in the literature; quadriplegia was only reported in some cases. In this study, we reported a patient with a lead level of 150 μg/100ml, associated with quadriplegia and walking disability.

4. Conclusion:

Workers who are in contact with industrial lead are exposed to chronic lead toxicity. Delayed detection of serious symptoms, such as cognitive, sensory, and functional disorders, can be life-threatening for these workers. Therefore, workers exposed to industrial lead are recommended to undergo periodic and regular examinations to prevent any possible complications.

5. Appendix

5.1. Acknowledgements

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5.2. Author's contribution

All authors meet the four criteria of authorship contribution based on the recommendations of the international committee of medical journal editors.

5.3. Conflict of interest

The authors declared no potential conflict of interest.

5.4. Funding

None.

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