

Lead Neuropathy Presenting As Bilateral Wrist Drop



Neurology

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ABSTRACT

Lead neuropathy is one of the most morbid forms of lead exposure in humans. It mostly occurs through industrial exposure and is mostly seen in people working in battery making factories. Lead neuropathies are mostly pure motor in type but sensory neuropathies are also described in literature with chronic exposure to lead. We describe a case of pure motor lead neuropathy in a young male working in a lead factory from central India who presented with bilateral wrist drop.

Introduction

Toxic neuropathies are rare forms of neuropathies which generally result in length dependent axonal neuropathy with the exception of diphtheria and a few toxic neuropathies. Because of the geographical, occupational and health related conditions, toxic neuropathies are likely to be more common than reported and impose a serious threat to health.¹ Lead neuropathy is a rare toxic neuropathy and has been reported mostly in battery workers and silver refining workers. In the west, strict monitoring in industry has reduced the risk of overt lead neuropathy but it remains to pose a threat to health in the developing countries like India. The common form of lead neuropathy consists of weakness that primarily involves the wrist and finger extensors but which later spreads to other muscles and minimal sensory involvement. Less commonly, there is distally accentuated sensory and motor involvement. The motor neuropathy variant is more likely to develop following relatively short-term exposure to high lead concentrations and evolves in a subacute fashion. Prognosis for recovery in this form is good as long as exposure is terminated promptly. The distal sensory and motor neuropathy variant develops after many years of exposure, evolves more slowly, and recovery is less certain. Lead intoxication in humans causes axonal neuropathy, but in some other species it causes a primarily demyelinating neuropathy. It is possible to prevent lead neuropathy by good industrial hygiene. Close monitoring must identify excessive lead exposure before it causes overt neuropathy.

If any evidence of excessive exposure is found or if overt neuropathy develops, exposure must be terminated immediately. The role of chelation therapy in the treatment of lead neuropathy remains controversial.²

Case report

A 30 year old male was admitted to the department of Neurology, with complaints of weakness of both the hands since 2 months. The weakness was distal in distribution involving extensors of wrist and fingers such that he was not able to extend the fingers. This weakness was relatively symmetrical, distal and progressive reaching its maximum deficit within a period of 15 days such that he was not able to do his routine activities of daily living. There was no proximal weakness, sensory loss or any weakness in the lower limbs. There was no bladder or bowel involvement. Craniobulbar involvement was not seen. There was no history of diabetes mellitus or alcoholism.

Patient works in a lead factory locally where he had been working for last 13 years and his job there was to make lead batteries using his ungloved hands to fill the raw material containing lead. There was no history of using masks while working. He had developed baldism 6 years back. Two of his male coworkers also had developed such complaints. He was also admitted in a local hospital one year back for complaints of severe abdominal pain and constipation which subsided without a definitive treatment. His coworkers although did not report any such weakness like complaints.

On examination, patient was found to have wasting in the extensor compartment of both the upper limbs (Image 1,2). It was also associated with weakness in the extensor compartment of forearms as well as hands (bilateral wrist drop). His lumbricals as well as all the interossei were also found to be weak bilaterally. His deep tendon reflexes were all normally present in the upper as well as lower limbs. His sensory examination was absolutely normal. A clinical suspicion of lead neuropathy was made and he was subjected to nerve conduction studies which revealed distal pure motor axonal neuropathy with secondary demyelination. His serum lead level estimation was done which was 222.6 mcg/dl (normal value < 5 mcg/dl). He was started on lead chelation therapy with penicillamine.

Patient was however lost to follow up.

Discussion

Lead neuropathy is a traditionally described pure motor disorder, which particularly picks out overused muscle groups such as the wrist extensors and mostly manifests as wrist drop and foot drop. Patients of lead intoxication with subacute motor weakness usually improve after removal from a toxic source. In contrast, there is no evidence that sensory neuropathic patients with chronic lead exposure improve during their first years after stopping exposure. Their blood and urinary lead concentrations remained increased four to five years after removal from exposure, despite chelation therapy; elimination of lead from bone takes decades. Many of the chronically exposed patients also showed toxic effects on other body organs, such as the brain, cardiovascular system, gastrointestinal tract, and red blood cells.

Few studies of exposed workers with raised lead concentrations have not noted clinical neuropathic abnormalities,

despite mild electrophysiological abnormalities.^{3,4}

There is no method for timed quantification of the concentration of lead in inhaled fumes during occupations such as metal cutting or lead smelting, or of oral ingestion because of poor hand cleaning. Serum lead concentration in our patient was 222.6 mcg/dl which is much higher than the permitted value of less than 5 mcg/dl.⁵

Previous electrophysiological studies have concentrated on groups of workers exposed to lead but generally free from neuropathic symptoms, or on those occasional patients with subacute motor weakness associated with lead toxicity.^{6,7} Our patient showed abnormalities of motor conduction in the form of decreased amplitudes, increased distal latency and decreased conduction velocities in upper limbs with clinical motor abnormalities also seen in upper limbs. Sensory nerve conduction has been studied less often in patients with lead exposure. Abnormalities of sensory nerve action potentials have not been reported, but slight slowing of sensory conduction has been seen.^{5,8}

Two different neuromuscular syndromes can result from lead intoxication: a subacute predominantly motor syndrome and a chronic sensory and autonomic syndrome.

Lead intoxication has been known to cause multiple abnormalities of porphyrin metabolism. Plumboporphyrin muscle weakness occurs in the context of normal lead concentrations in rare patients with Aminolevulinic Acid (ALA) dehydrase deficiency.

Conclusion

Lead neuropathy is an important industrial hazard. Lead intoxication may affect the neuromuscular system in two different ways. First, the subacute predominantly motor syndrome traditionally associated with high dose lead intoxication is probably secondary to lead induced porphyria. Secondly, patients with extremely long term lead intoxication manifest a mild sensory and autonomic polyneuropathy which probably reflects a direct neurotoxic effect of lead. Extensive health and safety controls on workplace exposure are needed to prevent these dangerous effects.

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Images



Image 1: Showing bilateral wrist drop



Image 2: Showing wrist drop with wasting of muscles of hand