LEAD POISONING PRESENTING AS A MIMIC OF ACUTE STROKE

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INTRODUCTION

Stroke mimics are usually non-vascular disease processes. Stroke mimics are difficult to diagnose in an emergency room situation and may be initially treated as stroke. This case report highlights the importance of history taking, especially occupational history, in the differential diagnosis of stroke. We report the case of a 40-year-old man who developed symptoms suggestive of posterior circulation stroke after he was exposed to lead chloride at his workplace. We also stress the need to recognize mimics at presentation in order to arrive at an early and appropriate management of patients.

ABSTRACT

Stroke mimics are usually non-vascular disease processes. Stroke mimics are difficult to diagnose in an emergency room situation and may be initially treated as stroke. This case report highlights the importance of history taking, especially occupational history, in the differential diagnosis of stroke. We report the case of a 40-year-old man who developed symptoms suggestive of posterior circulation stroke after he was exposed to lead chloride at his workplace. We also stress the need to recognize mimics at presentation in order to arrive at an early and appropriate management of patients.

KEYWORDS: lead poisoning, stroke mimics, occupational history.

INTRODUCTION

Stroke mimics are defined as disorders suggestive of acute focal brain dysfunction but turning out to be non-vascular in origin. Stroke mimics constitute 15–20% of all cases presenting to the Emergency department. Disorders that mimic stroke are important for emergency physicians and neurologists. The common stroke mimics are toxic-metabolic pathologies, seizure disorders, multiple sclerosis (MS), degenerative neurological conditions, hemiplegic migraine, intracranial tumors and peripheral neuropathies.[1] Here we present the case of a 40-year-old man with lead poisoning who presented with signs of posterior circulation stroke mimicking as stroke. In addition we briefly discuss the neurological complication of lead poisoning.

CASE PRESENTATION

A 40-year-old man presented with sudden onset of slurred speech, vertigo, vomiting, weakness of left side of his body and unsteadiness of gait since 2 days. There was no significant past history of co morbid illness. He did not report any fever, recent vaccination or trauma.

On admission, his vital signs showed blood pressure of 150/90 mmHg. Neurological examination showed a Glasgow coma scale of 15, spastic dysarthria, bilateral grade 2 horizontal nystagmus, left crural monoparesis of Medical research council grade 4/5, broad based ataxic gait, and extensor plantar on left side. However he demonstrated no objective weakness in upper limbs, and sensory deficit in any limbs. Examinations of his respiratory, cardiovascular and gastrointestinal system were unremarkable. In view of the above history and clinical findings, a diagnosis of acute stroke possibly involving the vertebrobasilar territory, was made and was further investigated.

Computed tomography brain showed bilateral symmetric and extensive grey-matter cerebral calcifications involving putamen, caudate nucleus, thalamus, anterior temporal cortex, midbrain, pons and cerebellar dentate nucleus (Figure 1). Magnetic resonance imaging (MRI) brain showed normal diffusion weight sequences, T2 and flair showed hyper intense lesions involving similar regions as mentioned above in brain CT scan, which were hyper intense in GRE sequence without contrast enhancement (figure 2,3), suggestive of mineralization.
Figure 1: Computed tomography showing grey-white matter junction hyperdensity suggestive of mineralization (Marked by red arrow).

Figure 2: Magnetic resonance imaging T2 flair sequence showing symmetrical hyperintensity involving putamen, caudate nucleus, thalamus, and anterior temporal cortex (marked by black arrow).

Figure 3: Magnetic resonance imaging T2 Flair axial sequence showing involvement of pons marked by red arrow.

A routine blood investigation including complete blood count, renal function, liver function tests, and thyroid functions showed normal results. Blood levels of calcium, magnesium, phosphorus, glucose, ferritin, iron, lactate, Vitamin D, calcitonin, and parathyroid hormone were within normal range. Urine for calciuria and phosphaturia showed normal results.

Due to these negative test results and the presence of staining of teeth with bony pains, our patient's environmental exposure and occupational history was considered. He had been working since 14 years in a factory that manufactures powder for PVC pipes, the content of which being lead chloride. Serum lead level was estimated, which was raised. The blood lead level was 60.74 microgram/dl (Normal value in adults <25 microgram/dl). He was observed on the ward and discharged on the fifth day with advice to stay off work until the neurological symptoms were cleared. He was treated with lead chelating agent d-pencillamine for 5 days, followed by repeat lead estimation after 4 weeks. He required a total of three courses of d-pencillamine to bring the blood lead level to normal range. Follow up visits showed complete recovery and asymptomatic at 12 weeks.

We considered the above mentioned symptoms were due to chronic lead poisoning, presenting as a mimic of acute posterior circulation stroke which was completely reversible following lead chelating agent.

**DISCUSSION**

Various conditions may mimic stroke. It is important to reach the correct diagnosis early so as to offer an early medical treatment and better prognosis, especially in young. The two most common stroke mimics are hypoglycaemia and seizure. Stroke mimic due to toxin are very rare.[2,3] Lead poisoning presenting as stroke is rare in clinical practice and has not been reported in literature.

Lead poisoning has been recognized as a major public health risk, particularly in developing countries. Lead primarily affects the central nervous, haematopoietic, hepatic and renal system producing serious disorders. Compared to other organ systems, the nervous system appears to be the most sensitive and chief target for lead induced toxicity. The effects on the peripheral nervous system are more pronounced in adults while the central nervous system is more prominently affected in children.[4] Within the brain, lead-induce damage in the prefrontal cerebral cortex, hippocampus, and cerebellum can lead to a variety of neurological disorders, such as brain damage, mental retardation, behavioral problems, nerve damage, and possibly Alzheimer’s disease, Parkinson’s disease, and schizophrenia. Chronic lead poisoning can cause various neurological and cutaneous manifestations. (Table 1).
Table 1: Neurological and Cutaneous manifestations of lead poisoning.

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<thead>
<tr>
<th>Neurological manifestations</th>
<th>Distinguishing features</th>
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<tr>
<td><strong>Cerebral forms</strong></td>
<td><strong>Neuromuscular forms</strong></td>
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| 1) Encephalopathy<sup>[4]</sup>  
a) Moderate exposure to lead  
b) High exposure to lead  
2) Parkinson’s disease<sup>[6]</sup>  
3) Alzhemers Dementia<sup>[6]</sup>  
Cognitive decline following chronic lead exposure.  
Personality changes with delusion, hallucinations. |
| **Neuromuscular forms** | **Cutaneous manifestations**<sup>[4,5,6]</sup> |
| 1) Neuropathy<sup>[5]</sup>  
2) Muscular forms<sup>[5,6]</sup>  
a) Muscle weakness especially of extensor muscles  
b) Muscular tremor due to lack of muscle coordinations  
c) Muscle fatigue | Skin  
Nail  
Hair  
Dental  
| Plumbic skin tone with pallor  
Bluish lead line in nail markedly seen in finger nails than toe nails  
Loss of hair, premature graying of hair  
a) Blue line at distal margin of gums called as gingival lead line  
b) Accumulation of lead in teeth giving staining of teeth, later loss of teeth. |

Note: Information from references<sup>[4,5,6]</sup>

High concentration of lead interferes with various cellular, intracellular and molecular pathway, by oxidative stress and ionic mechanism causing toxicological manifestations. Lead has the ability to substitute other bivalent cations like Ca<sup>2+</sup>, Mg<sup>2+</sup>, Fe<sup>2+</sup> and monovalent cations like Na<sup>+</sup>, the ionic mechanism contributes principally to neurological deficits, as lead, after replacing calcium ions, becomes competent to cross the blood-brain barrier (BBB) at an appreciable rate. After crossing the BBB, lead accumulates in astroglial cells (containing lead binding proteins).<sup>[4]</sup>

In adults the most common classical presentation of severe lead toxicity is bilateral wrist drop, which was not present in our case. Moreover nail and gingival lead lines were lacking. It has been reported that neuropathic features develop only when lead levels are more than 70 microgram/dl.<sup>[5,6]</sup> We emphasize that central nervous system involvement can occur at a level lower than that required for peripheral nervous system involvement. Hence in such cases classical skin, nail and gingival changes can be absent in lead poisoning affecting the central nervous involvement.

**CONCLUSION**

Stroke mimics can cause diagnostic dilemma and clinicians should be aware of the toxins mimking as stroke for correct diagnosis and early management of these reversible conditions. Further the change in occupational environment should be addressed to prevent recurrence in future.

**REFERENCES**


