Title: Occupational Lead Exposure and Its Neurological Impact: "A Silent Workplace Hazard''

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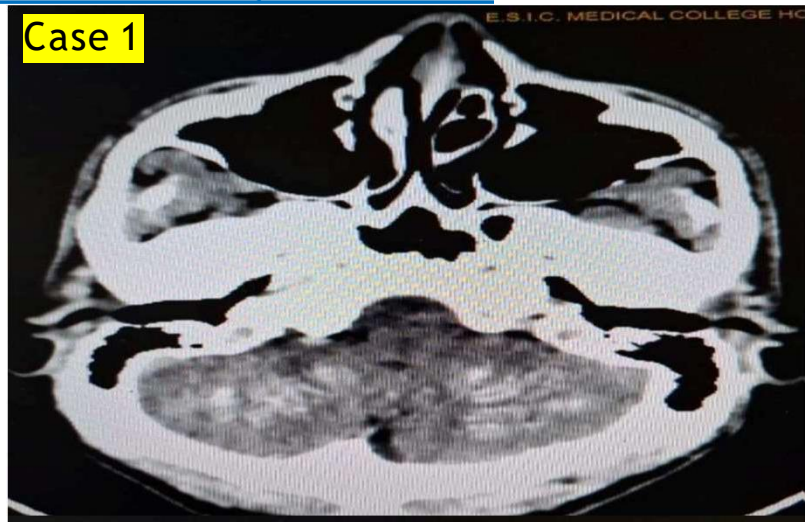
Background and Aims :

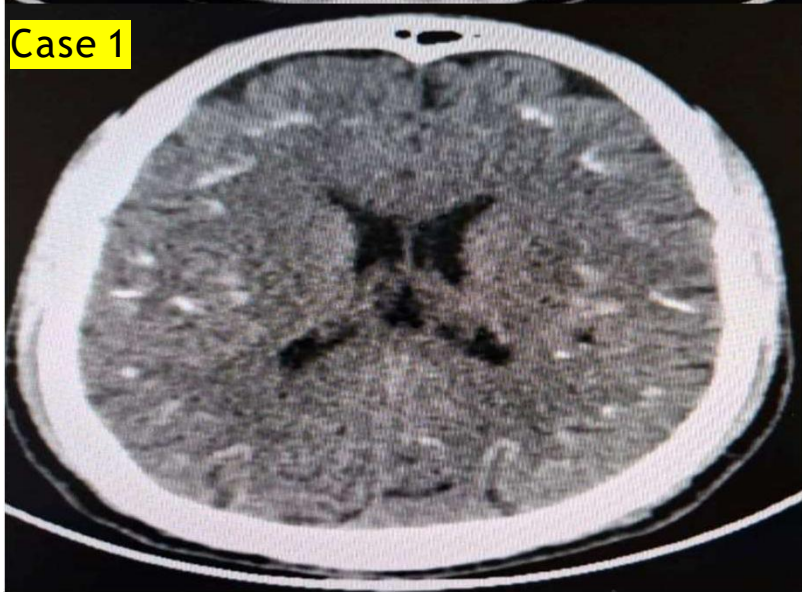
- Lead is a heavy metal that can enter the body through inhalation, ingestion or dermal contact
- While lead poisoning is often associated with children, adults remain at significant risk particularly in occupation settings such as battery manufacturing, construction and metal smelting
- The neurological effects of lead range from cognitive impairment and mood disorders to irreversible neurodegeneration
- The exact pathophysiology of lead induced cerebral calcification is not fully understood but it is thought to involve lead induced cerebrovascular injury, which leads to dystrophic calcification in damaged areas of brain
- Lead may also disrupt calcium phosphorus metabolism, alter blood brain barrier (BBB) function and contribute to calcium deposition in the subcortical areas, basal ganglia, vermis and cerebellum. These calcifications can appear as punctiform, curvilinear, speck like or diffuse patterns on imaging
- These effects may take years to surface by when the damage is often extensive and irreversible, hence the term silent hazard
- We present three patients with chronic lead exposure with diverse clinical presentations

Materials and methods:

Case 1:

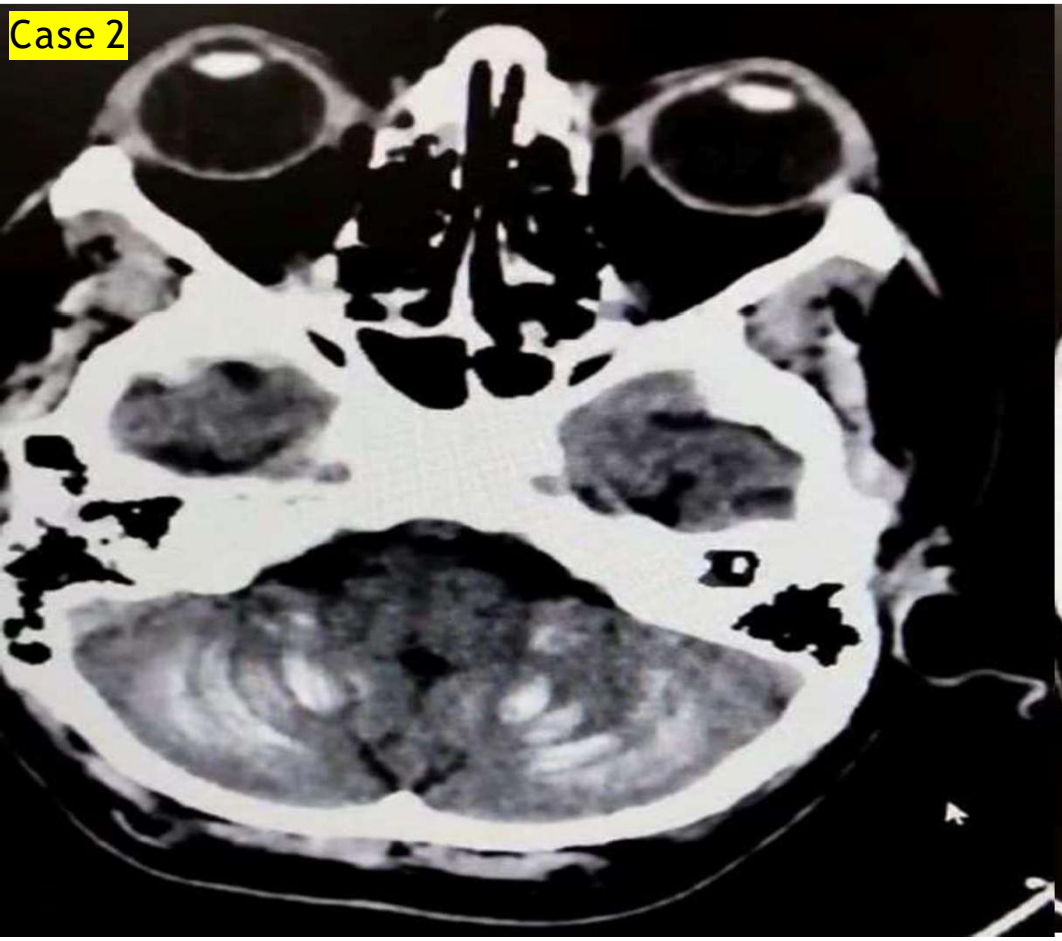
- 46yr old male battery worker by occupation presented with sudden onset dizziness
- Neurological examination : normal
- CT brain s/o bilateral symmetric confluent hyperdensities of 50-60 HU involving subcortical areas of both cerebral and cerebellar hemispheres
- Blood parameters : microcytic hypochromic anemia
- Serum calcium, Magnesium, Phosphate, PTH levels : Normal
- Serum lead levels : 79.50 mcg/dL

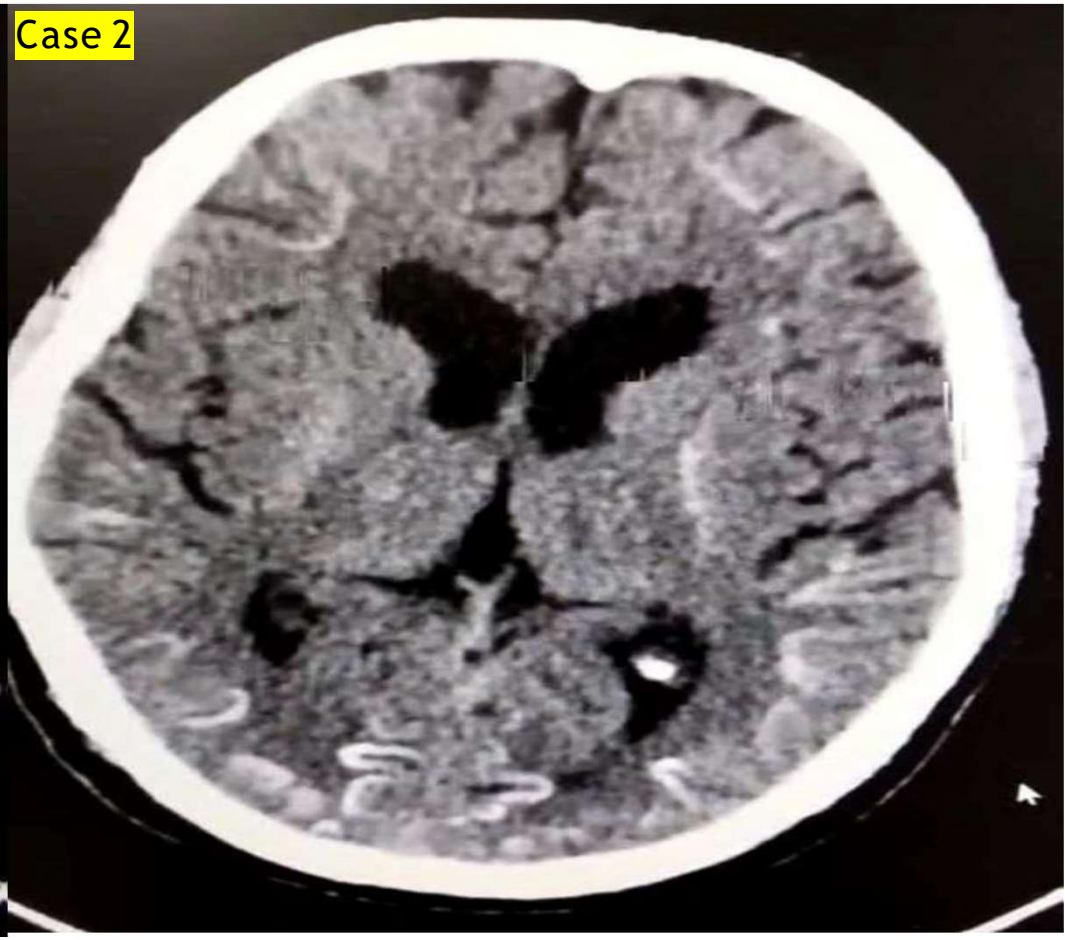




Case 2:

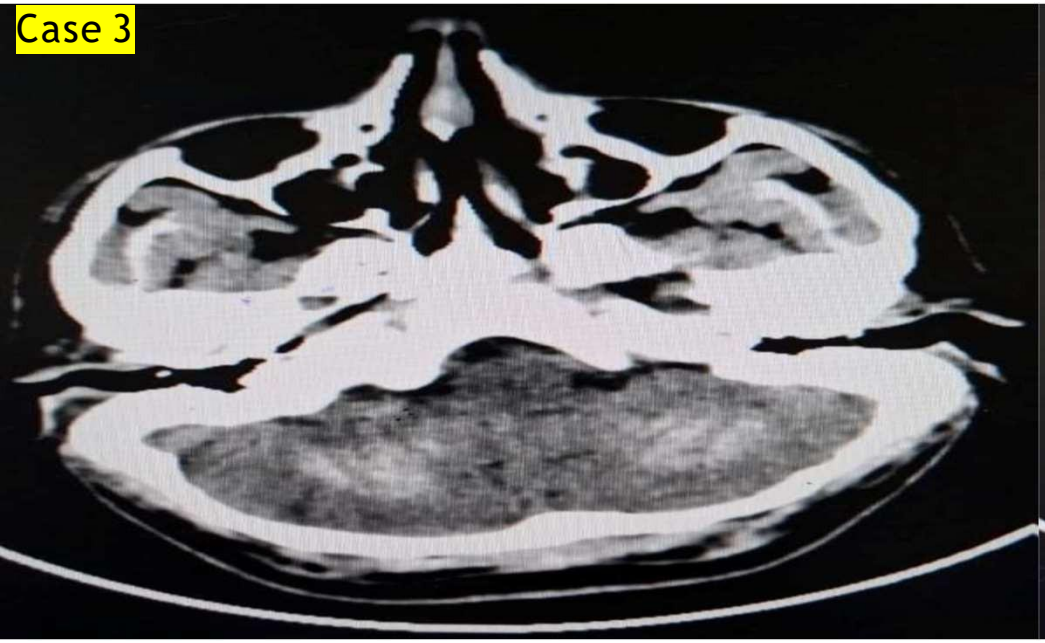
- 55yr old male battery worker by occupation presented with sudden onset vertigo and fall
- Neurological examination: normal
- CT brain : calcification which is extensive and has symmetrical involvement of basal ganglia, thalami and dentate nucleus and subcortical white matter
- Serum Calcium, Magnesium, Phosphate and PTH levels: Normal
- Serum lead levels : 85mcg / dL

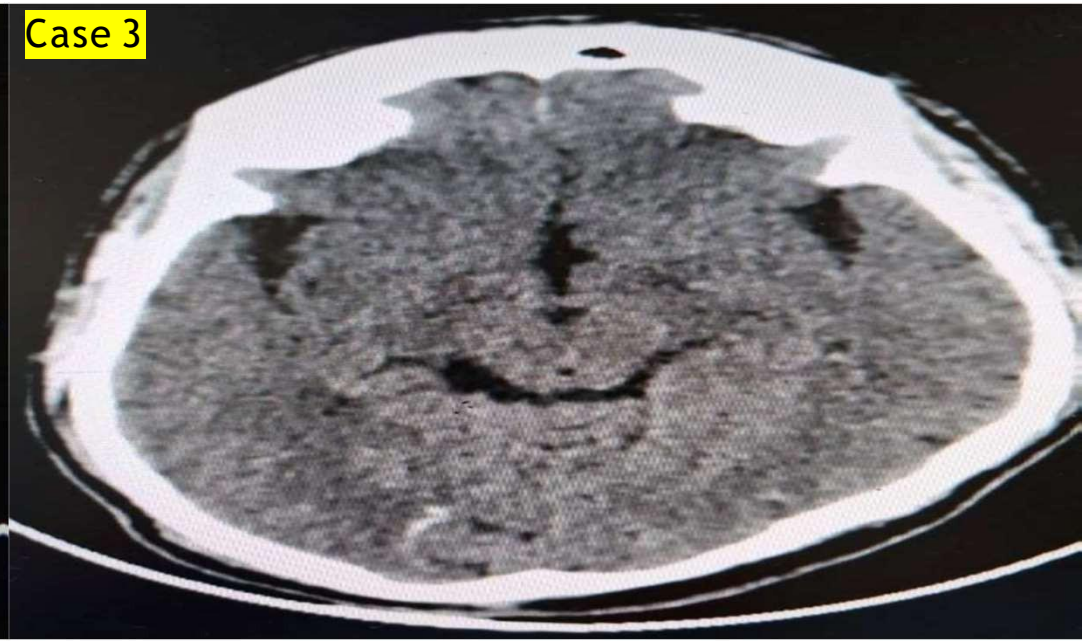




Case 3

- 60 yr old male, battery worker by occupation 10yrs back presented with sudden onset dizziness lasting few seconds since 2 -3 yrs.
- Also has history of sudden onset of distortion of visual images followed by uprolling of eyeballs with flickering movements of eyelids lasting few seconds with intact awareness. Occurrence of such episodes once every 3-4 months since past 10yrs. Neurological examination: Normal.
- CT brain s/o multiple hyperdense foci of calcifications involving bilateral basal ganglia, subcortical white matter of bilateral cerebellum and occipital lobes. EEG : Normal.
- Blood parameters : microcytic hypochromic anemia. Peripheral smear s/o basophilic stippling. Serum Calcium, Magnesium, Phosphate, PTH levels: Normal. Serum lead levels : 2mcg/dL





Results and Conclusions:

- Lead exposure can potentially lead to cerebral calcifications, especially in the context of chronic or high level exposure, through mechanisms related to calcium mimicry and metabolic disruption.
- This is more common in children and rare in adults but possible , usually in the setting of chronic high level exposure
- More research is needed to establish direct causation, but clinicians should be aware of this potential association, especially in symptomatic individuals with a relevant exposure history.