ISSN: 2319-6505

Available Online at http://journalijcar.org

International Journal of Current Advanced Research
Vol 5, Issue 10, pp 1372-1374, October 2016

RESEARCH ARTICLE

International Journal of Current Advanced Research

ISSN: 2319 - 6475

CLINICO- RADIOLOGICAL PRESENTATION OF AN OCCUPATIONAL CHRONIC LEAD HAZARD

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A R T I C L E I N F O

Article History:

Received 9th July, 2016
Received in revised form 15thAugust, 2016
Accepted 28th September, 2016
Published online 28th October, 2016

Key words:

Lead cerebral, cerebeller calcification

A B S T R A C T

Lead is one of the ubiquitous and versatile metallic elements which humans are exposed inevitably. We are presenting a male patient of occupationally acquired lead exposure causing him to have severe anaemia and status epilepticus due to severe calcification of cortical gyri and subcortical regions as evidenced through higher levels of serum lead levels. Imaging of the brain has highlighted the importance of screening of the people for lead in the peripheral blood with classical radiological manifestations.

INTRODUCTION

Lead ranks as one of the most serious environmental poisons amongst the toxic heavy metals all over the world. The common sources of lead exposure to humans are through the use of certain products containing lead such as lead solders, lead-based paints, cosmetics and ceramics, folk and herbal medicines, environmental emissions containing lead, lead containing drink and food and most importantly through occupations such as lead acid battery manufacturing, smelting, alloying and casting (D'souza *et al*. 2011)(1)

Literatures on imaging in chronic lead toxicity are sparse. Bilateral symmetric calcification of cerebellar hemispheres, vermis, subcortical areas of cerebral hemispheres and deep gray nuclei on plain CT of brain are highly suggestive of chronic lead toxicity in the appropriate clinical scenario.

Case report

29 yr old man presented to our hospital with history of recurrent seizures. He was a chronic smoker and alcoholic with last binge drinking 2 days prior to the presentation. Following which he had recurrent episodes (7-8) of seizures without any interictal regaining of consciousness (status epilepticus). In that status he was brought to emergency department of our hospital. Semiology of seizures was right focal seizures with secondary generalization. He was treated urgently with antiepileptic drugs, Leviteracetam, loading dose of 1.5gm followed by maintenance dose of 500 mg three times a day. He regained his sensorium without any focal deficits after 4 hours of treatment with antiepileptic drugs. His hematological profile (pretransfusion) had shown severe anemia of 5.4gm/dl, with PCV and MCV were 22.5% and 58.9 fl respectively and type of anemia was microcytic hypochromia without punctuate basophilia. His reticulocyte count was 1.0% and ESR was 42 mm for 1st hour.

He was transfused with 2 pints of packed cell RBC and post transfusion hemoglobin was found to be 8.4 gm/dl. His metabolic profile had shown normal renal function tests, liver function tests (except ALT -63 U/L), serum electrolytes and serum uric acid levels were within normal limits. His Gamma glutamyl transferase was found to be high – 90 U/L (10-66 U/L) and ammonia was 54 micromol/l (11-35 micromol/l). His blood total calcium and phosphorous levels (total serum protein) were normal and paratharmone was within normal limits-47 pg/ml (15-65 pg/ml). ECG showed sinus tachycardia and chest x ray was unremarkabale. EEG was done interictally and showed no abnormalities. Nerve conduction studies revealed normal motor and sensory conductions (done on both sides) suggesting no evidence of neuropathy.He had significant occupational history. He worked in the lead battery workshop for a period of about 10 years from 1995 to 2005, without any proper protection from the exposure. Subsequently he worked as a painter till his present illness. No Burtonian lines over gums.

In view of history of chronic exposure to lead, he was referred to the National Referral Centre for Lead Poisoning in India (NRCLPI). Screening involved the estimation of blood lead levels an indicator of chronic lead exposure. The blood lead (PbB) levels were expectantly in high range - 45.6 microgram/dl (normal being <9micrograms/dl).To see the consistency repeat blood lead levels done after a gap of 7 days in the same lab, was found to be still high -48 micrograms/dl. He was treated with chelating agent.

Skeletal survey with x ray of both forearms, hands, femur and both knee joints did not reveal any metaphyseal bands. Ultrasonogram of abdomen was unremarkable. CT scanof the brain showed extensive bilateral symmetric calcification of cerebellar hemispheres, vermis, sub cortical areas, basal ganglia and thalami. MRI brain revealed symmetric hyperintense signal changes in bilateral insular, external capsules, anterior temporal lobes and hippocampi on T2 FLAIR & T2W SE sequences. T1w images were unremarkable.Venobold sequence showed, mineralization of deep grey matter including bilateral globus pallid, putamen, thalami, subthalamic nuclei, red nuclei, substantianigra and bilateral cerebellar dentate nuclei. However Venobold failed to demonstrate subcortical, cortical and cerebellar mineral deposition seen onplain CT.

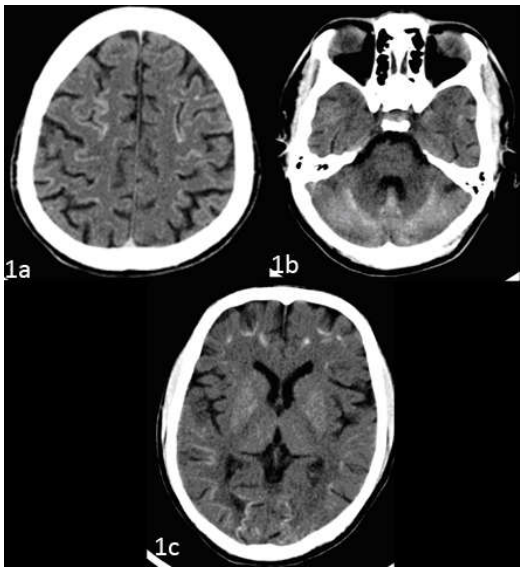

FIG.1a Hyperdensity of supratentorial cortical gyri
FIG.1b hyperdensity of cerebellar grey matter including vermis
FIG.1c Patchy bilateral basal ganglia and subcoritcal whitematter calcification

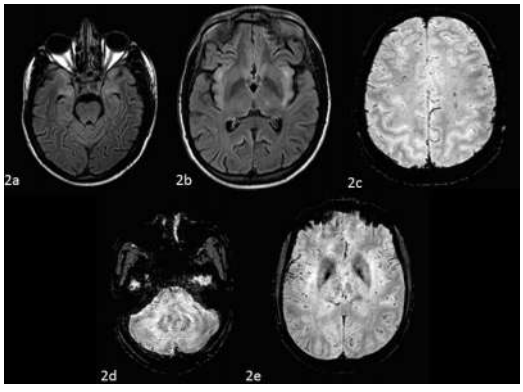

Fig 2a -2b- Axial FLAIR MRI Brain showing hyperintensities of symmetrical insular and medial temporal regions,Fig 2c-2e- Axial SWI MRI Brain showing mineralisation of symmetrical deep grey nuclei and dentate nuclei of cerebellum

DISCUSSION

This young man has presented to us with Status epilepticus, the etiology of which could be due to acute alcoholic abuse or secondary to the brain calcification due to chronic lead poisoning. His unique radiological findings on plain CT head have evoked interest in search of the etiology. Similar radiological findings were published by Benson *et al* 1985 (2) and Reyes *et al* 1985 (3) where in it was found in individuals exposed to high levels of lead. Cerebellar calcification due to chronic lead exposure was previously demonstrated by Tonge *et al* (4) & Reyes *et al* (3).

Experimental evidence in rats indicates a particular predilection of the cerebellum to deposition of calcium following exposure to excessive lead with particular affinity of the cerebellar capillaries (Goldsteine *et al* 1974) (5). It is due to the susceptibility of cerebellar capillaries to the lead (Silbergelo *et al* 1980) (6).

Lead poisoning involves any part of the central nervous system or peripheral nervous system depending on the level and duration of exposure. The gateway of Lead is the astroglia and the neurons via voltage-sensitive calcium channels.

Tonge *et al* (4) postulated that the proteinaceous exudates is incompletely cleared and undergoes dystrophic calcification. He further demonstrated a proteinaceous matrix within the cerebellar vascular calcifications in his autopsy series.

These findings were subsequently confirmed by cerebral CT in similar patients by Graham J *et al* (7). Similarly, Swartz *et al* (8) described cortical calcification in the cerebrum in a patient who had occupational history of lead exposure.

Symmetrical intracranial calci cations are seen in abnormal calcium and phosphate metabolism, such as hypoparathyroidism and pseudohypoparathyroidism.They are also seen in patients undergoing chemotherapy or irradiation and in those with Aicardi-Goutières syndrome, Cockayne syndrome, or Fahr's disease. Fahr's disease shows calcification in Globipallidi, putamen, thalamus, cerebral white matter, dentate nuclei and cerebellum. These patients usually present with behavioral disturbances and extra-pyramidal symptoms (Ming-Tsung Sun *et al* 2009) (9). In elderly brains, physiological calcification of vessels, basal ganglia, pineal gland, choroid plexus, dura, and habenula is normal (Patricio F. Reyes *et al* 1986) (10). Symmetric T2FLAIR hyperintensities noted in the insular region and hippocampi seems to be a post ictal phenomenon in our case. MRI failed to give us any clues suggestive of chronic lead toxicity and it was only the plain CT head which triggered cascade of investigations towards lead toxicity. This case report emphasis the importance of plain CT head in the era of MRI and strengthen its role in emergency unit.

The decreased Hb and elevated ALT levels have further indicated lead's effects on hematopoietic and hepatic systems, respectively. Absence of basophilic stippling despite the lead intoxication in the present study has been reported previously (Timpo *et al*. 1979) (11) and is not a reliable marker for lead poisoning.

CONCLUSION

In-detail clinical evaluation of the patient is very important for identifying ill effecs of lead over the brain and the body systems. Chelation therapy must be accompanied by environmental intervention. The potential health hazards of lead poisoning are still rising due to lack of awareness and education about self and environmental care. Intracranial calcifications can be physiological or pathological. Sometimes CT imaging is the first to give a clue in an unsuspected cases as in our patient who presented to us with status epilepticus. In addition to detailed medical/environmental history with laboratory examination

radiological investigation plays an important role in diagnosis of lead toxicity.

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