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Lead Encephalopathy: Uncommon Presentation Of Lead Toxicity

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Abstract

Lead toxicity is a known occupational hazard in our country. Lead is rapidly absorbed from ingestion, inhalation and direct skin contact routes and it's systemic effects are present at all possible levels of exposure.(1) Despite various occupational and public health measures that are in place to control lead exposure, cases of lead toxicity are still being reported. It is a cumulative toxin that causes detrimental effects on the hematopoietic, renal, reproductive and nervous system.(2) Lead toxicity and its complications are of grave public concern as it is a form of preventable cumulative toxicity.

Keywords: Lead Toxicity, Lead poisoning, Lead Encephalopathy

Introduction

Here we report a case of 30-year-old male patient admitted in our hospital with complaints of new onset abnormal body movements since 2 days, vomiting since 1 day and altered sensorium since 1 day. He was also a follow-up case of seizure disorder on Tab Sodium Valproate 500 mg twice a day for last 3 years. There was no history of fever, neck rigidity, blurring of vision or non compliance to medication. On presentation patient was conscious but not oriented to place or time. On examination pallor was present and rest of the systemic examination was unremarkable. Initial blood investigations revealed haemoglobin of 7.7 g/dl, Total leukocyte count of 10.4 thou/uL, Erythrocyte Sedimentation Rate of 50 mm/hr, Blood Urea & Nitrogen of 53.6 mg/dL, S.Creatinine of 2.09 mg/dL, S.Iron 48 ug/dL, S.Ferritin 514.7 ng/mL and peripheral smear showed microcytic hypochromic anaemia. Fundus evaluation was normal with no signs of raised intracranial pressure. Patient was then undertaken for lumbar puncture for cerebrospinal fluid analysis that was within normal limits. Non-contrast computerized tomography of head was done which showed evidence of multiple subcortical calcifications. History and examination were reviewed, patient was found to be working in a battery factory for last 14 years with history of exposure to molten lead and lead fumes. On examination of oral cavity bluish discolouration of gums was also noted, consistent with Burtonian Lines. There were no other neurological deficits. For confirmation whole blood lead levels and 24 hour Lead Urine levels were sent and were found to be >100 ug/dL (normal range < 25 ug/dL) and 215 ug/L (normal range < 80 ug/L) respectively.

As patient's symptoms were in line with features of lead toxicity and encephalopathy with whole blood lead levels of more than 100 ug/dL, patient was started on chelation therapy with intramuscular Dimercaprol 4 mg/kg every 4 hourly for 7 days and intravenous EDTA (ethylenediamine tetra acetic

acid) 50 mg/kg/day in 2 divided doses for 7 days along with Iron, Zinc and Vitamin C supplementation with adequate hydration. Whole blood lead levels were repeated after initial therapy and were found to be 36 ug/dL. Ultrasound kidney, urinary bladder (USG-KUB) showed moderate hydronephrosis with ureteric calculus on left side for which urology consultation was taken and double J-Stent was placed. Patient improved symptomatically and was then discharged on Tablet Sodium Valproate 500 mg twice a day, Tablet Calcium Carbonate 500 mg once a day, Tablet D-Penicillamine 250 mg 4 times a day for one month and was also advised for change in occupation. Patient was again reviewed after 6 months. On evaluation Burtonian lines and pallor had disappeared. Whole blood lead levels were 21.9 ug/dL, haemoglobin was 15 g/dL, rest of the blood parameters had improved and patient was symptomatically and clinically better.

Discussion:

Patients with lead toxicity commonly present with anaemia, pain abdomen and neurological deficits in form of wrist or foot drop. Lead encephalopathy is uncommon but an important manifestation of toxicity. [3] The incidence of lead encephalopathy happens to be less common in adults in comparison to children as mature adult brains tends to sequester lead away from its mitochondrial site of action within the cerebral and cerebellar neurons. Lead encephalopathy may present in form of altered behavior, seizures and altered sensorium. [4][5] A lower threshold for seizures is seen in lead toxicity which can be attributed due to alteration in the blood brain barrier and inhibition of gamma-aminobutyric acid (GABA) mediated neurotransmission especially when whole blood lead levels are more than 70 ug/dL. [6] Blood lead levels of > 70 µg/dL also result in cerebral oedema, encephalopathy and confusion, drowsiness, coma and even death. [7] Other manifestations include bluish discolouration of gums – Burtonian lines, hypertension, microcytic hypochromic anaemia associated with punctate basophilic stippling of erythrocytes in some cases, abdominal colic, chronic tubulointerstitial renal disease, lead bands at epiphyses of the long bones, and predisposition to gout. Combination of circulating lead with sulphur produced by commensal flora of oral cavity causes deposition of the formed lead sulphide as the Burtonian lines. Though a rare presentation, but if detected it can hasten the diagnosis of lead toxicity. [8] Anaemia seen in lead toxicity and poisoning occurs due to disruption of haem synthesis through inhibition of key enzymes by lead involved in the process : δ-aminolaevulinic acid dehydratase, coproporphyrinogen oxidase and ferrochelatase. Lead also causes inhibition of pyrimidine 5′-nucleotidase and can attach to the erythrocyte membrane to interfere with Na^+-K^+-ATP enzyme thereby increasing the propensity of erythrocyte to haemolyse. [8] The Centre for Disease Control and Prevention, Atlanta, United States of America, has defined elevated whole blood lead levels as more than 25 µg/dl of whole blood in adults and more than 5 ug/dL in young children. Currently chelation therapy is recommend by the Centre for Disease Control and Prevention only when the blood lead level goes beyond 45 µg/dL. [9] Common chelating agents that are used in lead toxicity include:

1. Calcium disodium ethylene diamine tetra-acetic acid (CaNa2EDTA)
2. D-penicillamine
3. Meso 2,3-dimercaptosuccinic acid (DMSA)
4. Sodium 2,3-dimercaptopropane-1-sulphonate (DMPS)/Dimercaprol

Duration and choice of therapy depends on the initial presentation and whole blood lead levels. For lead levels of more than 70 ug/dL initiation of therapy with Dimercaprol is recommended and in cases with levels of more than 100 ug/dL Dimercaprol is given along Calcium disodium ethylene diamine tetra-acetic acid (CaNa2EDTA). [10]

Our patient was treated initially with a combination of intramuscular Dimercaprol and intravenous EDTA (ethylenediamine tetra acetic acid) along with Iron, Zinc and Vitamin C supplementation with adequate hydration. Upon discharge chelation was continued with D-penicillamine despite reduced levels (<45 ug/dL) after initial therapy in view of lead encephalopathy and possibility of increase in blood lead levels in future due to lead re-mobilization from bones. Patient was also advised for change in occupation. During follow up patient did not report any fresh complaints, remained seizure free and improved clinically and on laboratory parameters.

Learning Points

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1. Lead poisoning is a preventable form of cumulative toxicity and appropriate measures can potentially help a large number of factory workers who are at high risk to suffer from fatal complications secondary to lead toxicity.
2. Lead encephalopathy is an uncommon manifestation of lead toxicity which can be fatal if not timely detected.
3. Apart from initial chelation therapy it is imperative for patients to be on regular follow up for evaluation of persisting complications and to measure blood lead levels for repeat chelation therapy if required.
4. Patients should be advised for use of personal protective equipment or change in occupation if possible to decrease exposure to lead.

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Figure 1: Black Arrow Head showing multiple subcortical calcifications on Non-Contrast CT Head

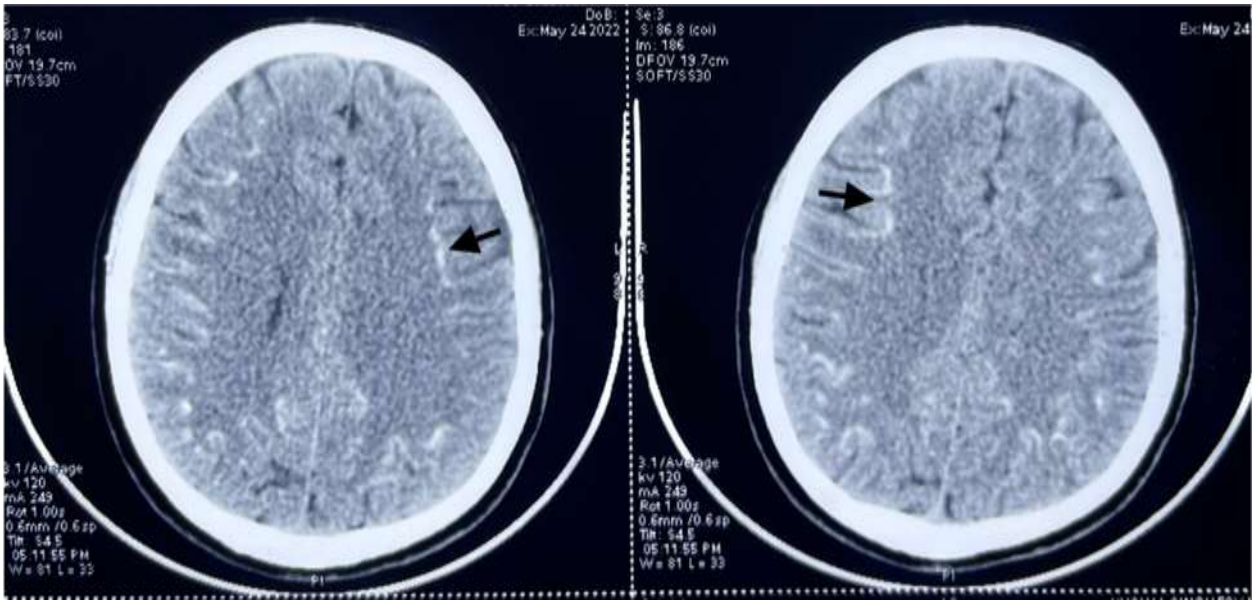

Figure 2: White Arrow Head showing Burtonian Lines along the lower gums

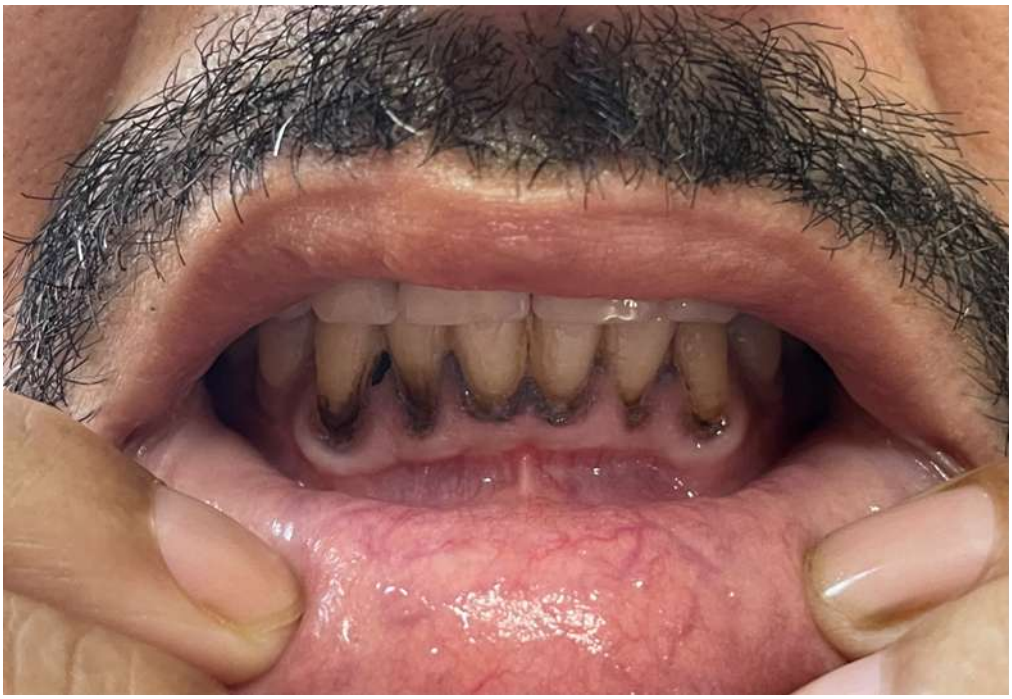

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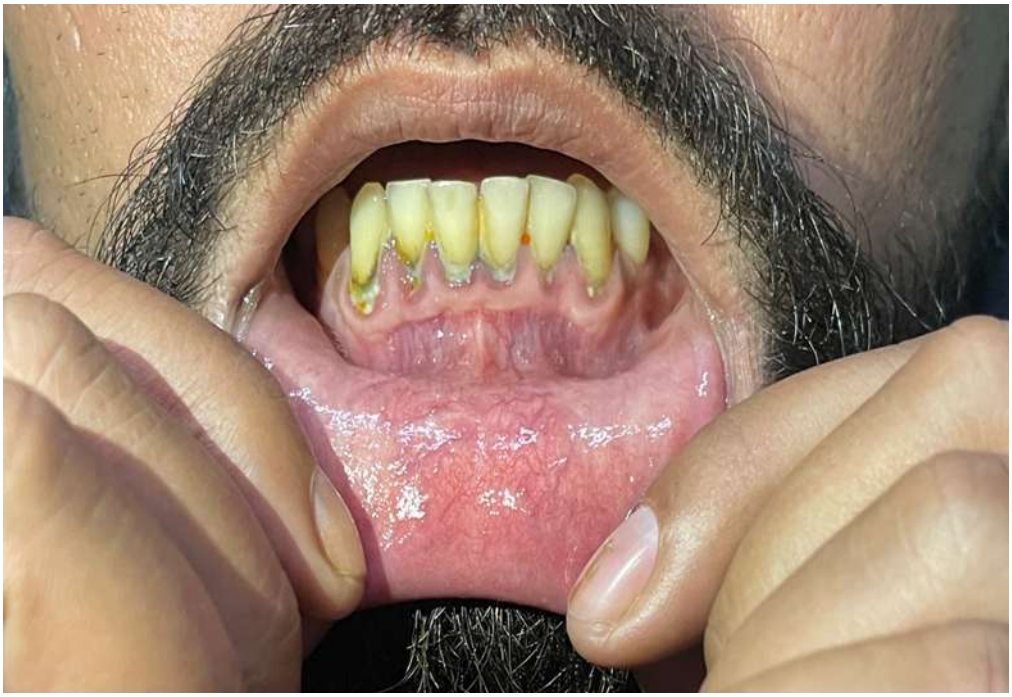
Figure 3: Disappearence of Burtonian Lines on follow up after 6 months of chelation therapy



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