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Imaging findings of a metabolic encephalopathy-A missed case

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ABSTRACT:
Lead poisoning, also known as plumbism and saturnism is a common occupational health hazard in developing countries. Occupational exposure and traditional medicine usage are the most common causes of lead poisoning in adults.This is a case report of a 30-year-old male, who worked in battery manufacturing unit and presented with acute confusion, seizures and behavioural abnormalities. He was previously hospitalised 2 years backand was an undiagnosed case of lead poisoning. The findings were established by computed tomography and magnetic resonance imaging, and the final diagnosis was confirmed by laboratory tests.In this paper we review the neuroimaging features of chronic lead poisoning in a patient who was a missed case for two years.

KEYWORDS: Lead poisoning, occupational exposure, cortical laminar calcification on CT, T2 and FLAIR hyperintensities on MRI, Burton's lines.

I. CASE REPORT:

A 30-year-old male, had a past history of seizures, chronic recurrent abdominal colic and vomiting for 3 years, for which he was admitted twice in 2019. At the time of admission, he had anaemia with haemoglobin (Hb) level of 8mg/dl. His blood pressure was high (160/90 mmHg). His ultrasound (USG) abdomen and magnetic resonance imaging (MRI) brain were normal at that time (Figure 1). The patient was managed conservatively and discharged on anti-epileptics, antihypertensives and folic acid.

Now he presented with acute confusion, generalised tonic-clonic seizures, pain abdomen and vomiting for past 1 week. Patient was disoriented, confused and restless at the time of examination. General physical examination revealed pallor, ataxia and hypertension.

Complete hemogram revealed haemoglobin of 6.3g/dl and microcytic normochromic anaemia without evidence of basophilic stippling.His haematocrit level was low [19.4% (normal range- 37-47%)].His renal function tests were deranged with serum creatinine level of 1.82 mg/dl (normal range0.5-1.5 mg/dl). Total leukocyte count and platelet counts were within normal limits.His parathyroid hormone levels were within normal limits.

For further evaluation, a non-contrast computed tomography (NCCT) head and MRI brain was done.

Imaging findings:

NCCTbrain was done. It depicted cortical laminar calcifications in a curvilinear pattern along the grey-white matter junction in bilateral (b/l) cerebral hemispheres(Figure 2a, 2b). Bilateral symmetrical calcifications were also seen involving cerebellar hemispheres(Figure 2c).

The differentials considered were lead toxicity, parathyroid disorders, Fahr disease and mineralising microangiopathy.

MRI brain revealed T2& FLAIR hyperintensitiesinvolving (b/l) periventricular white matter (Figure 3a), b/l basal ganglia and medial thalamus (Figure 3b) and b/l medial temporal lobes (Figure 3c).No diffusion restriction or post contrast enhancement was seen.

Keeping in view of CT and MRI findings, the diagnosis of metabolic encephalopathy likely due to lead toxicity was kept.

The patient was retrospectively examined for signs of lead toxicity. It revealed dark blue lines in gums (Figure 4), also known as Burton's lines. Occupational history revealed that he was a worker in battery manufacturing unit for more than 10 years.

Following that, the serum and urine lead levels were sent by the physician which came out to be raised (>100ug/dl).

Management:

The patient was treated with antiepileptics, anti-hypertensives and dimercaprol (chelating

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agent). He was advised further avoidance of lead exposure.

II. DISCUSSION:

Lead encephalopathy was first documented in 1925.[1]It is much rarer in adults than in children.[2] In adults, lead encephalopathy is more serious and can be sometime lethal.Mechanism of action of lead is that it acts as a cellular toxin and inhibits mitochondrial respiration.It inhibits Na+/K+ -ATPase pump and interferes with cellular energy metabolism.[3]

Lead can cause acute, chronic or subclinical toxicity. In acute form, patients present with headache, vomiting, seizures, stupor, paralysis, coma. In chronic lead toxicity, as in our case, patients present with neurodegeneration, disorientation, drowsiness, tremors, stupor, ataxia, seizures, cognitive decline and behavioural abnormalities.[4]

In severe conditions, cerebral edema develops leading to vomiting, stupor, coma and death. Other manifestations of lead poisoning include blue lines in gums (Burtonian lines), abdominal colic, hypertension, microcytic hypochromic anaemia, tingling in hands and feet, constipation and lead bandsat epiphysis of long bones.[2]

The US Centers for Disease Control and Prevention and the World Health Organization state that a blood lead level of 10 µg/dL or above is a cause for concern; however, lead may impair development and have harmful health effects even at lower levels, and there is no known safe exposure level.[5][6]

In our case, the patient had blood lead levels of >100ug/dl. He had anaemia which is due to inhibition of pyrimidine 5'-nucleotidase.He also had seizures which is due to alteration in blood-brain barrier. Abdominal colic is due to contraction of smooth muscles of intestinal wall.Basophilic stippling is more pronounced in bone marrow than in the peripheral blood.[2]

CT imaging reveals symmetrical white matter hypodensities involving b/l cerebral hemispheres thalami and cerebellar hemispheres.Lead intoxication can cause intracranial calcification.in previous reports, speckled calcifications are seen in subcortical white matter.[7]Perelman et al reported an incidence of 84% cerebellar calcification at autopsy in a study of 44 adults with a history of chronic lead poisoning.[8]Schroter et al reported that CT brain in chronic lead poisoning showed bilateral symmetric calcification in the subcortical area of the cerebral hemispheres and basal ganglia.Schroter et al reported high signal intensities in the periventricular white matter, basal ganglia, insula, posterior thalamus, and pons.[9]

Fahr disease or primary familial brain calcification is characterised by abnormal calcium deposition in b/l basal ganglia, dentate nuclei and white matter with subsequent atrophy. [10]Mineralising microangiopathy is characterised by parenchymal cerebral calcifications and is usually seen in children as a complication of cranial radiotherapy and chemotherapy.[11] Primary hyperparathyroidism is characterised by symmetrical calcifications in the basal ganglia, dentate nucleus of the cerebellum and in the union of the grey and white matter.[12] idiopathic hypoparathyroidism results in b/l symmetrical calcification in thalamus, basal ganglia and grey white matter junction.[13]

TEACHING POINT:

Lead encephalopathy is an uncommon manifestation of lead toxicity in adults and is predominantly seen in workers in battery manufacturing units.Besides CT and MRI findings, an adult patient presenting with chronic abdominal pain, anemia and seizures must be asked for occupational history or lead exposure for early diagnosis and management of this generally missed toxic entity.

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FIGURES:

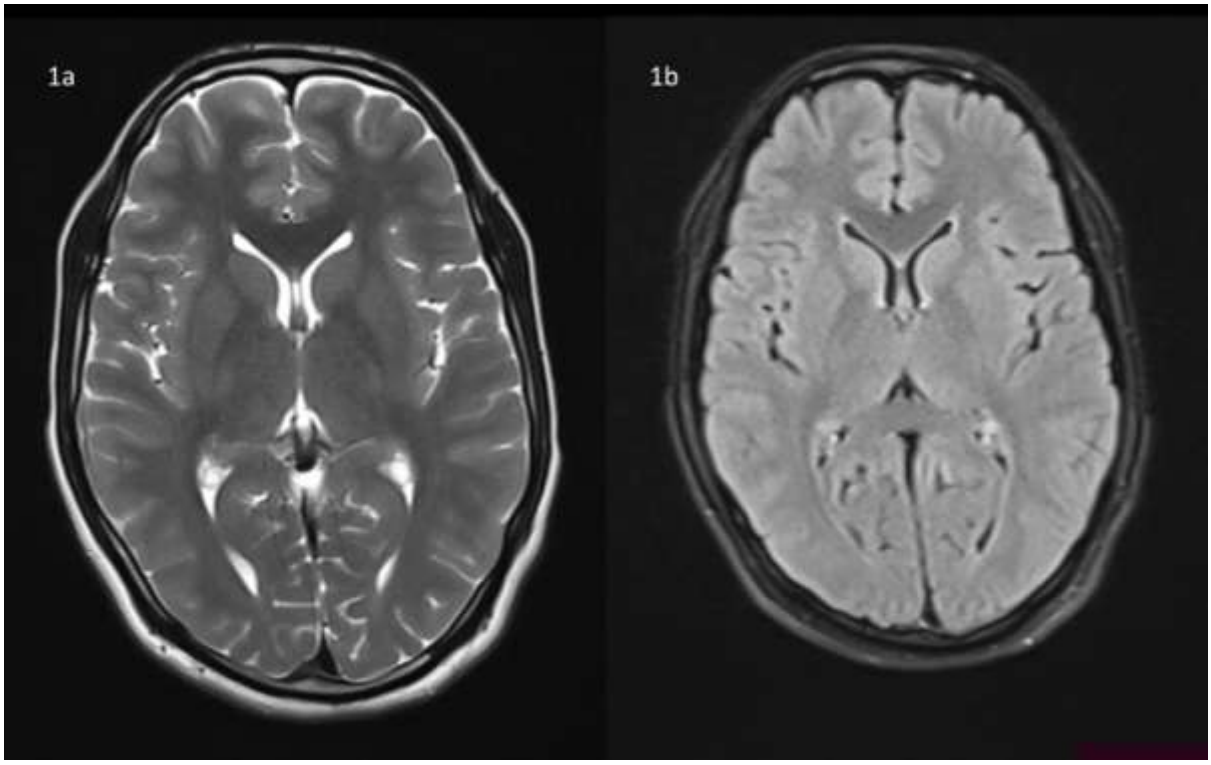

Figure 1: T2 weighted (1a) and FLAIR (1b) axial images revealed normal study for brain parenchyma

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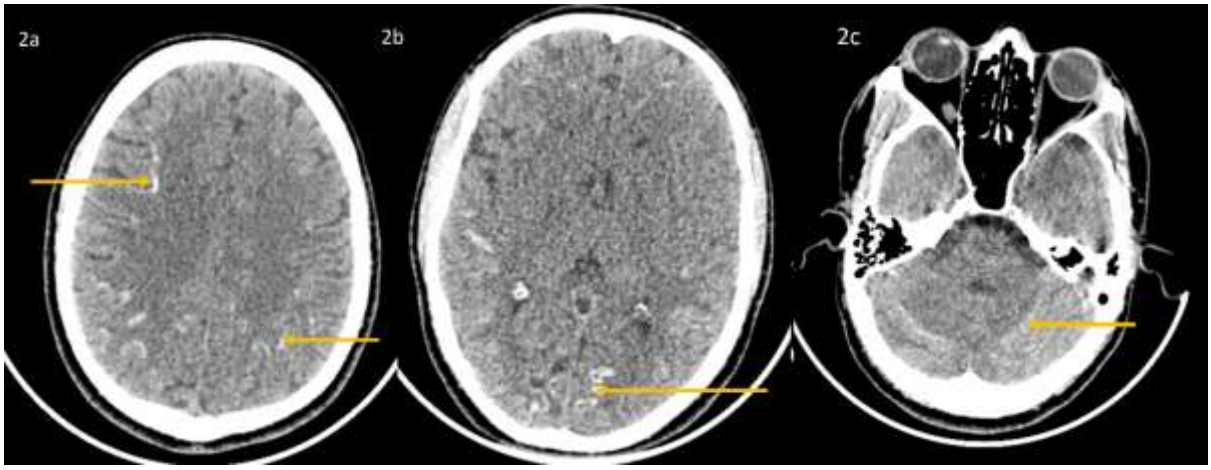

Figure 2: A 30-year-old male with lead encephalopathy

Technique: Non-contrast commuted tomography (NCCT) was performed
Findings:
2a, 2b) Symmetrical cortical and subcortical laminar calcification in curvilinear pattern in bilateral cerebral hemispheres (arrows)
2c) Symmetrical laminar calcifications in bilateral cerebellar hemispheres(arrow).

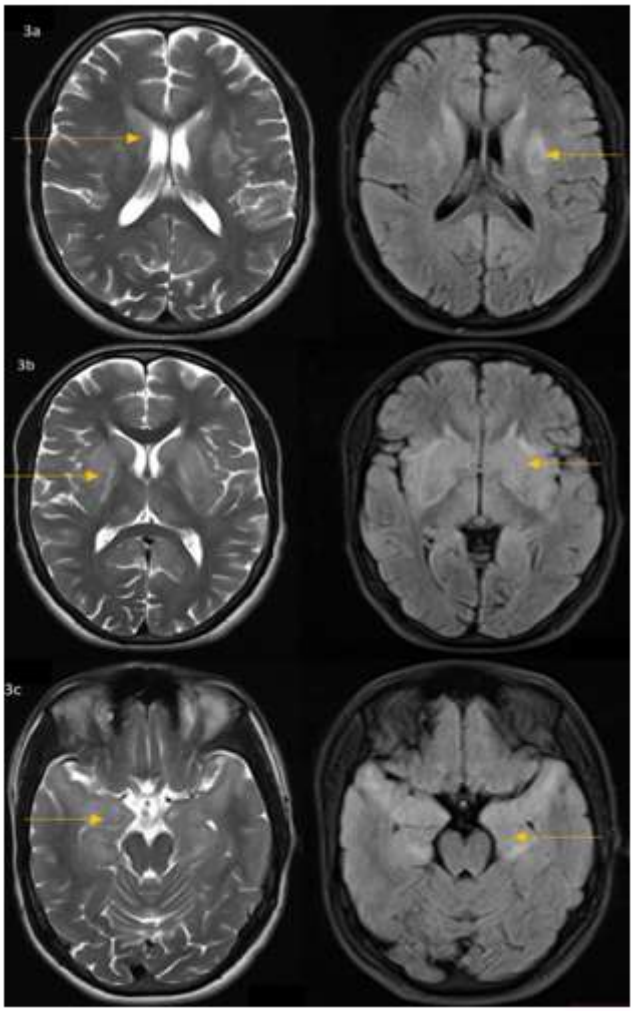

Figure 3: A 30-year-old male with lead encephalopathy

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Technique: Contrast enhanced MRI brain was performed
Findings: T2 and FLAIR hyperintensities involving bilateral periventricular white matter (Figure 3a), bilateral basal ganglia and thalami (Figure 3b) and bilateral medial temporal lobes (Figure 3c).

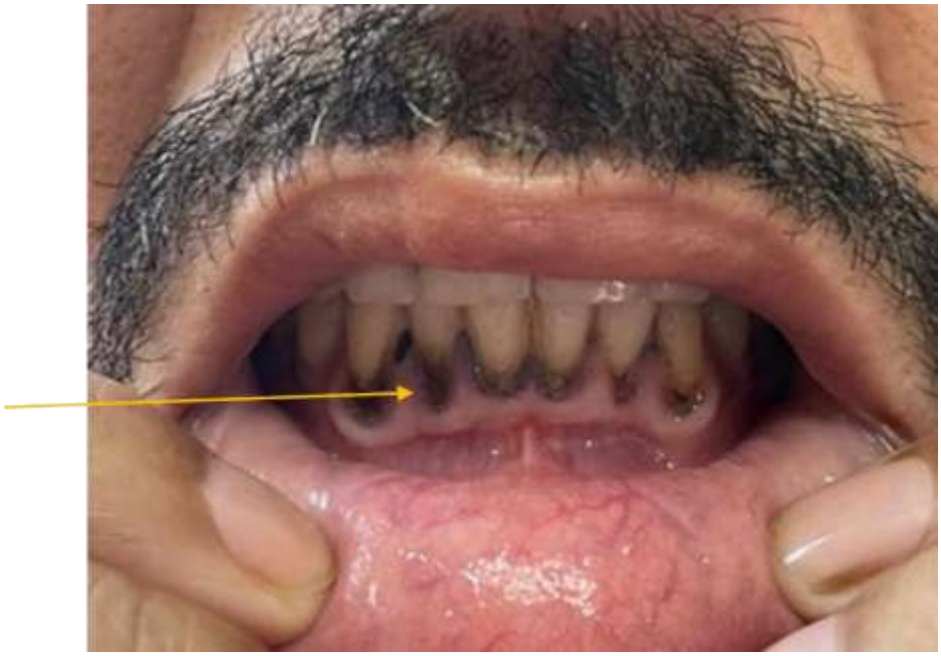

Figure 4: Blue lines in gums, also known as Burton's lines.

TABLE:

Etiology	Occupational exposure, traditional medicine intake
Age predilection	More common in adults with occupational history
Risk factors	Chronic lead exposure Traditional medicine (ayurvedic medicine) intake Inhalation of toxic fumes Intake of contaminated water Lead paint
Treatment	Avoidance of lead exposure Symptomatic management Chelating agents
Imaging findings	Bilateral basal ganglia, cerebellar and cortical laminar calcifications on NCCT Symmetrical T2/FLAIR hyperintensities in bilateral basal ganglia, thalamus and cerebellar hemispheres T1 hyperintensities corresponding to areas of calcifications on CT

Table 1: Summary of lead encephalopathy in adults

Differential Diagnosis	**Clinical**	**CT Brain**	**MRI Brain**
Lead encephalopathy	• Chronic abdominal pain • Nausea, vomiting • Anemia • Seizures • Altered sensorium • Lead lines in	• cortical laminar calcifications • basal ganglia calcifications • white matter hypodensities in b/l thalami, cerebral and cerebellar hemispheres	• symmetrical T2 and FLAIR hyperintensities in b/l thalamus, basal ganglia, occipital lobes and cerebellum • T1 hyperintensities corresponding to areas of

	gums		calcifications on CT
Mineralising microangiopathy	• Children • Complication of cranial radiotherapy and chemotherapy	• Calcification in b/l basal ganglia and subcortical white matter	• T1 hyperintensities in b/l basal ganglia and affected regions
Hyperparathyroidism	• abdominal pain • renal calculi • bone pain • depression, confusion • constipation	• Calcification in b/l basal ganglia, dentate nucleus and grey white matter junction	• Symmetrical T1 hyperintensities in b/l basal ganglia and cerebellum
Fahr disease	• Asymptomatic • Psychosis • Gait disturbance	• Calcification in b/l basal ganglia and subcortical white matter	• T1 hyperintensities in b/l basal ganglia and affected regions
Hypoparathyroidism	• Tetany • Emotional liability • Short stature	• Calcification in b/l basal ganglia and subcortical white matter	• T1 hyperintensities in b/l basal ganglia and affected regions

Table2: Differential diagnosis table for bilateral symmetrical intracranial calcifications.

ABBREVIATIONS:
Hb: haemoglobin
USG: ultrasound
MRI: magnetic resonance imaging
NCCT: non-contrast computed tomography
b/l: bilateral