has been stable except for a few bouts of anger outbursts for which he is on follow up with child psychiatrist. He is on close follow-up and may require further chelation.

Lead is not known to serve any significant biological function and deposition does not spare any organ in the body [1]. It has high affinity for the skeleton and chronic exposure often sequesters large proportion in the bones followed by the kidneys [4]. After a period of initial exposure lead is redistributed to the soft tissues. If cessation of exposure occurs at this juncture, there is a decrease in the blood lead levels post the initial rise [5]. Bone, being a dynamic tissue, undergoes remodelling throughout life which is regulated by a wide range of hormones and local availability factors. Prolonged exposure also results in slow release of lead from the bone stores over a protracted period of time [4]. Children are at high risk of lead poisoning as they are in a state of constant growth and development. Moreover, the growing bones in children undergo perpetual remodelling which allows lead to be regularly reintroduced into the blood stream [6]. Chelation therapy brings down the blood lead levels acutely only to rebound within weeks to months after treatment. Often, repeated courses of chelation are required [5].

This case report emphasizes the need for long term follow-up with periodic monitoring of lead levels in children with chronic lead poisoning to assess the need for repeated chelation therapy. Blood lead concentration may rise to toxic levels even after removal of exposure due to constant re-distribution in a growing child.

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Fig. 1 X-ray left knee of the index patient showing lead lines (arrows) over lower end of femur and upper end of tibia.

Autoimmune Hypophysitis in Systemic Lupus Erythematosus

Autoimmune hypophysitis (AH) is a rare autoimmune disease that occurs when the pituitary gland is infiltrated with lymphocytes and plasma cells, leading to impaired hormonal secretion. Rare cases of association of systemic lupus erythematosus (SLE) with AH have been reported in literature but mainly in adult population. AH commonly involves anterior pituitary; labelled as lymphocytic adenohypophysitis (LAH) but it can also involve posterior pituitary which is called lymphocytic infundibulo-neurohypophysitis (LINH) [1-4].

Herein, we report a rare case of lupus in a male child who presented with features of central hypothyroidism and diabetes insipidus that was diagnosed as SLE-associated AH. He was treated with pulse methylprednisolone and cyclophosphamide with hormone replacement.

A 14-year-old male child, fourth issue of a non-consanguineous marriage was admitted with history of fever, weight loss, pallor and generalized weakness since one month. There was no history of rash, bleeding manifestations, abdominal distension, night sweats, oral ulcers, icterus, headaches, visual disturbances or joint swelling. He had received multiple oral antibiotics with no improvement. In the past, he had suffered a stroke at ten years of age with MRI brain showing acute lacunar infarct in right corona radiata. Birth history was uneventful and he was immunized as per schedule.
Anthropometric parameters and vitals, including blood pressure, were normal. Clinical examination revealed malar rash, oral ulcers, severe pallor with moderate hepatosplenomegaly. Laboratory investigations revealed anemia (Hb, 6.4 g/dL), direct Coombs test (DCT) positive, normal white blood cell (WBC) counts (WBC, 7.9×10^3/L), Neutrophils 49%, Lymphocytes 51%), thrombocytopenia (Platelet count, 30,000/cmm), raised ESR (81 mm at end of one hour), prolonged activated partial thromboplastin time (APTT) [Test, 54 sec (26.9-36.3)], high spot urine protein creatinine ratio (0.9, normal <0.2) with normal liver enzymes, serum electrolytes and X-Ray chest. Immunological investigations showed strongly positive anti-nuclear antibody (ANA titres 1:2560, speckled pattern), low serum complement C3 (C3-60 mg/dL; normal range 82-173 mg/dL) and C4 (C4 11.2 mg/dL; normal range: 13-46 mg/dL), positive anti-cardiolipin IgM antibody, beta-2 glycoprotein IgM antibody and lupus anti-coagulant. Anti-double stranded DNA antibody and anti-Smith (anti-Sm) antibody were negative. Ophthalmology examination showed retinal hemorrhages. Thyroid function test revealed central hypothyroidism [low free T3 (<1 pg/mL), low free T4 (0.46 mg/dL), and low TSH (<0.01uIU/mL)], with positive anti-thyroid peroxidase (anti-TPO) antibodies. During the hospital stay, child started developing delirium and agitation along with polyuria. Serum osmolality was high (320 mOsm/kg). Magnetic resonance imaging (MRI) showed absence of posterior pituitary bright spot on T1-weighted imaging consistent with diabetes insipidus in a lupus patient, endocrine hormonal evaluation and an MRI of pituitary gland is warranted to rule out AH.

In our patient, SLE was diagnosed based on constitutional symptoms, malar rash, oral ulcers, thrombocytopenia with auto-immune hemolytic anemia (Evans syndrome), low WBC counts, high ESR, ANA positivity, low complement levels, positive antiphospholipid antibody, and high urine protein creatinine ratio. AH is a rare disease, mainly affecting females though in our case it was a male. It has incidence reported to be 1 in 9 million [5] but the actual number may be more, particularly as IgG4-RD and involvement of the hypophysis by systemic pathologies has increasingly been recognised [6]. AH can be primary (idiopathic) or secondary to sella and parasella lesions, systemic diseases, or drugs (mainly immune checkpoint inhibitors). The predominant feature of LINH is central diabetes insipidus which was present in our patient. The diagnosis of AH was made when our patient showed features of central hypothryoidism and diabetes insipidus with loss of normal posterior pituitary bright spot on T1-weighted MRI. The gold standard of diagnosis is pituitary biopsy which reveals massive infiltration of lymphocytes and plasma cells in the pituitary gland but is usually denied by patients due to its invasive nature. Our patient is similar to the case reported by Jing, et al. [4] in which a 15 year lupus child revealed low levels of sex hormones, thyroid hormones and serum cortisol with MRI of pituitary region demonstrating an enlargement of the pituitary stalk. She was diagnosed as LINH associated with cortisol and MRI of pituitary demonstrated IgG4-related disease (IgG4-RD). We treated our patient with steroids, cyclophosphamide, hydroxy-chloroquine, hormone replacement and warfarin with significant clinical improvement in constitutional symptoms, normalization of acute phase reactants, complement levels, thyroid function and urine proteinuria at 3 months of follow up. Warfarin was added to treatment protocol considering positive antiphospholipid antibody and history of prior lacunar infarct. To the best of our knowledge this is the first case reported from India with features of AH in a case of juvenile SLE.

We would like to thank the patient's family for their cooperation and allowing us to publish this case report.

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