setting of proven tuberculosis suggested CAPS. Clinical improvement only after steroids, along with complete disappearance of APLA by 6 months confirmed CAPS with tuberculosis as the etiology.

Persistent tubercular infection with a rapid clinical deterioration (multiorgan dysfunction) should raise the possibility of inflammatory complications like hemophagocytosis or CAPS. CAPS being a potentially life-threatening condition, a high index of suspicion, early diagnosis and aggressive treatment with steroids, anticoagulation and occasionally plasmapheresis is needed for a favorable clinical outcome.

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Repetited Chelation in Lead Encephalopathy

Lead is an abundantly distributed heavy metal in our environment which in higher concentrations is hazardous to the body [1]. Nervous system remains the most severely affected, effect being more pronounced on growing children [2]. Common sources are lead based paint, lead contaminated air, soil, dust, drinking water through lead soldered pipes, lead coated vessels used for cooking, traditional medications and certain cosmetics [1]. Absorbtion of lead varies depending on the chemical form and the mode of exposure (ingestion > inhalation >transdermal). The half life of lead in blood and soft tissues is 35 days as (dL). Skeletal survey showed lead lines over lower end of femur showed high lead levels of 80.31 mcg/dL (acceptable upto 5 mcg/dL). Neuro-imaging was normal. Heavy metal screening of blood showed mild leucocytosis, with minimally elevated proteins. Cerebrospinal fluid Gene analysis showed leucocytosis, with minimally elevated proteins. Cerebrospinal fluid Gene analysis showed leucocytosis, with minimally elevated proteins. Cerebrospinal fluid Gene X pert for tuberculosis was negative. Neuro-imaging was normal. Heavy metal screening of blood showed high lead levels of 80.31 mcg/dL (acceptable upto 5 mcg/dL). Skeletal survey showed lead lines over lower end of femur (Fig. 1). Parents were screened and their blood lead levels (BLL) were within normal limits.

He underwent lead chelation therapy with Dimercapto-succinic acid 30 mg/kg/day for 5 days followed by 20 mg/kg/day for 14 days. Other effective agents including Dimercaprol and Edetate disodium calcium (CaNa2EDTA) could not be procured at that time. Supplementation with Iron, vitamin D, zinc, vitamin C was done. He was stabilized, anticonvulsants were gradually weaned off. BLL dropped to 38.08 mcg/dL. On review after 2 months, BLL showed a rise to 56.38 mcg/dL. Child, however remained asymptomatic. Repeat chelation therapy was given and BLL dropped further to 32.9 mcg/dL only to rise to 62.9 mcg/dL in 2 months. He has undergone four doses of periodic chelation at the time of writing this report. He

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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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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.