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Tuberculosis in Catastrophic Antiphospholipid Antibody Syndrome

Antiphospholipid syndrome (APS) describes patients with antibodies targeting phospholipid molecules causing recurrent arteriovenous thromboses, fetal losses, thrombocytopenia along with antiphospholipid antibodies viz. lupus anticoagulant and the anticardiolipin antibodies [1]. It can be either primary, or secondary that is triggered by infections or malignancies [2]. Catastrophic APS (CAPS), also known as Asherson syndrome, is a rare accelerated variant characterized by the rapid appearance/progression of more than three organ thromboses, with microangiopathy leading to multiorgan failure. We describe a child with pulmonary tuberculosis triggering CAPS.

A 14-year-old girl presented with increasing pyrexia and cough for 3 weeks. Mild left upper/middle zone crepitations were present with no respiratory distress. Investigations on admission revealed anemia (hemoglobin 8.5 g/dL), leucocytosis (leucocyte count 13×10^9/L, polymorphs 85%), thrombocytopenia (platelet count 45×10^9/L), with mildly elevated C reactive protein (10 mg/dL) and erythrocyte sedimentation rate (ESR 40 mm/h). Blood cultures were sterile. With an initial chest roentgenogram revealing pneumonitis, she was commenced on antibiotics (amoxicillin and clavulanic acid) with no response. Worsening in the productive cough with persistent fever, despite 7 days of antibiotics, prompted a computed tomography (CT) scan, which revealed a left upper consolidation with cavitatory changes and necrotic mediastinal lymphadenopathy. Sputum tested positive for *Mycobacterium tuberculosis* and anti-tubercular therapy (ATT) was commenced.

While on ATT, she developed severe bifrontal headache, vomiting and worsening respiratory distress. Magnetic resonance imaging of brain was performed, which was suggestive of thrombosis of superior sagittal venous sinus, right transverse sinus and sigmoid sinus with thrombus extending to proximal internal jugular vein. Repeat investigations revealed anemia, falling leucocyte counts (5.8×10^9/L), thrombocytopenia (platelet count 60×10^9/L), increasing CRP (107mg/dL), high ESR (120 mm/h) and transaminitis. Activated partial thromboplastin time (aPTT) was prolonged. Thrombophilia work-up was suggestive of antiphospholipid antibody (APLA) positivity [Lupus anticoagulant positive; Prolonged Russel viper venom time (RVVT), 78.5 (control- 48.3) seconds; anti-cardiolipin (ACL) IgG, 15 U/mL (normal range 0-12.5 GPL U/mL); Beta 2 glycoprotein 1 IgG, 30 U/mL (control 12 U/mL)]. Protein C, Protein S, anti-thrombin levels were unremarkable. Hyperfibrinogenemia (450 mg/dL), hypocomplementemia (C3, 80.8mg/dL and C4 9.2 mg/dL), positive direct coombs test, elevated serum lactate dehydrogenase (600 U/L), high serum ferritin (800 ng/mL) and significant proteinuria (spot urine protein creatinine ratio 1.2) were other positive findings. Peripheral smear ruled out schistocytes. Antinuclear antibody (ANA), Rheumatoid factor and anti-neutrophil cytoplasmic antibodies (ANCA) were negative. A CT angiogram revealed bilateral segmental pulmonary thromboembolism, ground glass changes and diffuse alveolar hemorrhage (DAH) with multiple splenic infarcts. With imaging and laboratory evidence of progressive multiorgan dysfunction, a diagnosis of CAPS triggered by pulmonary tuberculosis was made.

Child was started on parenteral methyl prednisolone followed by oral steroids and anticoagulation with low molecular weight heparin, while continuing ATT. Fever and cough resolved over the next 2 and 4 weeks, respectively. Steroids were tapered over 3 months. Repeat APLA work-up after 12 weeks confirmed APLA positivity. Anticoagulation was continued for 6 months. APLA tested negative at 6 months ruling out primary APS and establishing tuberculosis as the etiology. Repeat neuroimaging at 6 months showed significant resolution of the thrombus.

CAPS is a rare life-threatening autoimmune disease defined by definite criteria [3]. The diagnostic urgency of CAPS lies in a canonical onset of multiorgan thromboses/dysfunction, thrombotic microangiopathies (TMAs), systemic inflammatory response syndrome (SIRS) mimicking septicemia and a high mortality rate. Skin infections (18%) and human immunodeficiency virus infection (17%) are the most common associated infections [4]. ACL antibodies have been reported in a proportion of tuberculosis patients compared to normal controls [5]. Pneumonitis in the lower respiratory tract and systemic inflammation causes endothelial activation along with a reduction in the anti-coagulant mechanisms, impaired fibrinolysis with resultant hypercoagulopathy and pulmonary and systemic thrombin generation [6]. Besides, molecular mimicry between *M. tuberculosis* and β-2GPI molecule has also been proposed for the development of CAPS. In the index child, presence of such rapidly progressive deterioration with microangiopathy and thromboses (with APLA positivity) in the

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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Repeated 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 compared to bones being 5-20 years. Bone stores release lead to the blood which may add up to a toxicologically significant amount [3]. We report a boy with lead encephalopathy, who required repeated chelation therapy.

A 7-year-old boy, presented with refractory status epilepticus. He was the first born child of a non-consanguineous marriage from Nilgiris, Tamilnadu, with unremarkable neonatal period. He was developmentally and neurologically normal with no behavioural problems and immunized upto date as per national immunization schedule. There was no family history of seizures. The child was on traditional medicines for about a year for vitiligo over lips and face. He developed unilateral headache 10 days prior to the onset of seizures along with intermittent abdominal pain and vomiting for which symptomatic treatment was given. One such episode of vomiting was followed by right sided focal seizures. The child was given parental anticonvulsants. However, due to worsening of sensorium and uncontrolled seizures, he was put on mechanical ventilation and started on multiple anticonvulsants. Child was gradually stabilized and extubated.

Laboratory analysis showed leucocytosis with polymorphic preponderance and microcytic hypochromic anemia. Serum Iron was low - 21 mcg/dL [Normal 50-120 mcg/dL] though serum ferritin and total iron binding capacity were within normal range. Liver functions, renal functions and coagulation profile were normal. Cerebrospinal fluid analysis showed mild 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 Dimercaptosuccinic 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