(chr17:g.7915502G>A; Depth 60x) that results in the amino acid substitution of glutamic acid for glycine at codon 597(p.Gly597Glu; ENST00000254854.4) in the tyrosine kinase domain of the GUCY2D protein, confirming diagnosis of LCA type 1. Child is currently under visual rehabilitation therapy and follow-up. Genetic counselling during next pregnancy identified the same defect in the fetus, leading to medical termination.

LCA, an autosomal recessive disorder, is a rare cause of congenital blindness with a prevalence of 2-3 per million, occurring due to degeneration of retinal photoreceptor cells [1]. FDA approved gene therapy (voretigene neparvovecrzyl) is available for those with *RPE65* mutation [4]. Infants present with progressive poor vision since birth, nystagmus, photophobia, hyperopia, keratoconus and a classic behavioral pattern, Franceschetti oculo-digital sign, which involves repetitive poking, pressing, and rubbing of eyes with hand. These oculo-digital mannerisms mechanically stimulate dysfunctional retinal photoreceptors by production of phosphene, but can lead to atrophy of orbital fat and enopthalmos [4]. Though characteristic of LCA, oculo-digital sign can also be a nonspecific marker of poor vision in infants.

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Acalypha indica-Induced Hemolysis and Methemoglobinemia in a Child With G6PD Deficiency

Acalypha indica (*kuppaimeni*) leaf extract is an herbal remedy, widely used locally for treatment of minor ailments. A 5-years-old boy presented to the emergency room with acute onset of passage of red colored urine and yellowish discoloration of eyes for a day. He was given ~50 mL of *kuppaimeni* leaves concoction for an upper respiratory infection 6-8 hours before the onset of symptoms. There was no significant past history or family history. Child was pale and icteric at admission, SpO2 in room air was 80%, which did not improve with supplementary oxygen. Sensorium was normal, and examination of other systems were normal.

Laboratory workup showed anemia (hemoglobin, 4.5g/dL), reticulocytosis (10%), hyperbilirubinemia (indirect bilirubin, 7.1 mg/dL), high aspartate transaminase (277 U/L), normal alanine transaminase (33 U/L), high lactate dehydrogenase (7253 U/L) and hemoglobinuria suggesting acute intravascular hemolysis. Serum creatinine was normal. Peripheral smear showed blister and bite cells with polychromasia. Direct Coomb test was negative. Glucose-6-Phosphate-Dehydrogenase assay showed low level of 133 IU per million RBC (normal value, 202–522 IU). Co-oximetry was done, which showed elevated methemoglobin (10.5%).

He was managed with high flow oxygen, packed red cell transfusion, hyper-hydration and diuresis. Methylene blue for treatment of methemoglobinemia was considered but deferred in view of low G6PD levels. Child started improving on next day, and was discharged home on the fourth day. On follow-up, repeat G6PD level was low (100 IU per million RBC), confirming G6PD deficiency.

Hemolysis after use of *A. indica* has been reported previously [1]. In a systematic review, it has been categorized as a possible cause of hemolysis [2]. There are reports of symptomatic methemoglobinemia accompanying hemolytic crisis in G6PD-deficient individuals [3]. G6PD deficiency results in diminished production of NADPH through pentose phosphate pathway. NADPH deficiency leads to deficient glutathione production which is useful to protect hemoglobin from oxidative damage. This finally culminates in methemoglobinemia during oxidative stress induced G6PD deficiency hemolytic crisis [3].

We report this case to increase awareness regarding *A. indica*-induced hemolysis, in association with methemoglobinemia in G6PD deficiency.

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Acute Flaccid Myelitis: Are We Vigilant Enough?

With the eradication of poliomyelitis from most countries, acute flaccid myelitis (AFM) due to non-polio enteroviruses and other viruses is an emerging disease. Besides the vaccine-associated paralytic polio, AFM outbreaks due to other viruses are also a hazard. AFM comprises of patients with acute flaccid paralysis (AFP), characteristically asymmetric limb weakness, with MRI suggestive of a spinal cord lesion in grey matter and spanning one or more vertebral segments [1]. Over the last decade, multiple outbreaks have been reported from countries such as USA, European countries, and Japan [1]. Two outbreaks have already been reported from India [2,3]. However, the pathogen testing was limited and inconclusive in both the cohorts. Even with ongoing AFP surveillance, AFM has not been frequently reported from India. Similar to Australia, we believe that there is misdiagnosis and under-recognition of AFM. During the initial disease course, AFM is frequently misdiagnosed as transverse myelitis due to an often extensive involvement of the spinal cord, not classically limited to the grey matter of the spinal cord [5]. Hence, there is a need for creating awareness regarding this evolving entity.

With many viruses involved such as EVD68, EVA71, etc. and poor yield of pathogen testing, it is often difficult to establish causality for AFM [1-3]. Therefore, it is time that patients with AFP should also be tested for other viruses beyond the poliovirus. This can later help in strengthening the AFP surveillance system. Survey studies for non-polio AFM throughout the country may be an initial step in this aspect, in the absence of active ongoing surveillance. However, the surveys need to be more robust to capture the epidemiological aspects of both AFM and associated respiratory/gastrointestinal illnesses. The key epidemiological parameters should include the whereabouts of patients (for source identification), age group, details of neuroimaging, and virological studies, contact tracing, etc. for patients in both the groups. Besides, AFM clusters and outbreaks need to be investigated meticulously to avoid an epidemic staring at us.

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Acute Flaccid Paralysis in a Child: It Is Not Guillain-Barré Syndrome Always!

A 6-year-old-girl presented with complaints of difficulty in walking for 5 days. Initially, the child started limping on the left side, followed by unable to bear weight; within two days, the right lower limb also got involved and she became non-ambulatory. She also complained of dull aching pain in lower limbs, especially in the upper thigh, more on the left side. There was no history of proceeding febrile illness, trauma or intra-muscular injection. She was completely immunized as per the national immunization schedule. She had tenderness in the left flank, lower back and bilateral thigh, keeping the hips in a semi-flexed position, not allowing any passive movement or formal tone examination. Even knee jerks could not be elicited bilaterally. In the left hip joint power was 2/5 and 3/5 power in the right knee, left hip and knee joint. A clinical possibility of acute flaccid paralysis (AFP) was kept, with a differential diagnosis of Guillain-Barré syndrome (GBS), viral myositis, polymyositis, transverse myelitis, paralytic polio myelitis, Perthes disease, septic arthritis of the hip joint and pseudoparalysis due to unnoticed trauma, or with pelvis/femur fracture. On investigations, *X*-ray of the hips, nerve conduction study and serum creatine phosphokinase were normal. Ultra-sonogram revealed a heterogeneous collection in left iliopsoas muscle, extending to the pelvis and inguinal region. Pus was drained by percutaneous pigtail catheter and she responded favorably to intravenous vancomycin and she was able to walk after three days.