Cardiac channelopathies are caused by mutations in proteins related to the intra-cellular transport of sodium, potassium and calcium ions. These ion channel abnormalities predispose the patient to episodes of lethal ventricular arrhythmias such as TdP and ventricular tachycardia (VT) or fibrillation (VF). While some may die during such arrhythmias, these lethal arrhythmias can be non-sustained with spontaneous termination. Cardiac output is significantly decreased during these non-sustained episodes and the resultant cerebral hypoperfusion may result in seizures and/or syncope [3]. The term 'torsadogenic seizures' has been used to describe this kind of paroxysmal activity in the past [4]. Misdiagnosis as epilepsy has been shown to be the most important reason for delay in diagnosis and could potentially result in sudden death [5].

A careful history can provide clues to the diagnosis in most patients. A family history of SUD, seizures or syncope in multiple members should raise the suspicion of a cardiac channelopathy [6], as was seen in three of our cases. Recurrent seizures despite appropriate therapy and especially in the absence of pathogenic EEG changes should raise the suspicion of a cardiac channelopathy [6].

Genetic testing by a targeted panel of genes implicated in cardiac channelopathies or an exome wide screening is performed through next generation sequencing. A positive genetic test allows genotype specific therapy [7]. Genetic testing also permits cascade testing in other (even asymptomatic) family members [8] and helps identify individuals at risk and provide appropriate treatment even before clinical manifestations occur. This is especially important in channelopathies as a normal ECG does not rule out the presence of the phenotype. The QTc interval may be normal on a baseline ECG in affected individuals and an exercise stress test or provocative testing with adrenaline may be necessary to identify QTc prolongation. In our series, cascade screening permitted diagnosis in an asymptomatic mother as well as an aunt in whom the symptoms had wrongly been attributed to seizures.

In conclusion, seizures may be the clinical presentation in children with cardiac channelopathy. Red flags such as a family history of sudden death, seizures associated with auditory triggers and recurrent seizures with a normal EEG should raise suspicion and prompt referral to a pediatric cardiologist.The institution of appropriate lifestyle modifications and pharmacological therapy could result in control of symptoms.

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Ivermectin Poisoning – Report of Successful Management

Ivermectin is an endectoparasiticide and is the drug of choice in filariasis, scabies (crusted or if topical treatment has failed), and several other infestations [1]. It has a low rate of adverse reactions with toxicity occurring only with overdosing, resulting in adverse prognosis, as no specific antidotes are available [1,2]. Although rare in children, over 150 cases of serious neurological toxicities have been reported in adults [1]. We report a case of ivermectin toxicity with encephalopathy, shock and aspiration pneumonia in a young child and its successful management.

A 6-year-old previously healthy girl (weighing 20.5 kg) presented with a history of accidental consumption of 60 mL of 1% w/v (600 mg) ivermectin lotion (30 mg/kg). She was undergoing treatment for scabies. After four hours of consumption, she had two episodes of vomiting followed by generalized tonic-clonic movements and loss of consciousness. On arrival, she was unresponsive, with Glasgow Coma Scale (GCS) 6/15, with tachycardia, tachypnea, hypotension and oxygen saturation of 79% at room air. Pupils were equal bilaterally (3 mm) with sluggish reaction. There were no signs of meningeal irritation. There was generalized hypotonia with absent deep tendon reflexes and fundoscopic examination was normal. Excessive salivation and bilateral crepitations were present. The child was admitted to the pediatric intensive care unit. Given the poor GCS and respiratory failure, the child was intubated, ventilated, and started on parenteral antibiotics ceftriaxone and clindamycin, along with supportive measures. In view of continuing seizures, intravenous midazolam and

phenytoin were given. In addition to saline bolus, noradrenaline infusion for hypotension was started and carefully titrated. Blood gas analysis showed respiratory and metabolic acidosis. The hemogram on admission showed hemoglobin 9.5 g/dL, platelet count 173×10^9/L; and total leucocyte counts 7.2×10^9/L (neutrophils, 76.7%). Liver function tests and renal function tests, coagulation profile and blood glucose were normal. C-reactive protein (CRP) was elevated 36.4 mg/L. Chest *X*-ray showed left upper lobe collapse and bilateral opacities. The National poison information centre was consulted and supportive management was advised as there was no specific antidote. Her urine output remained normal.

Patient started having high grade fever after 24 hours. Repeat investigations showed leucocytosis of 13.5×10^9/L (neutrophils 83%) and CRP of 67.8 mg/L. Blood culture and endotracheal secretions for culture showed no growth. In view of shock and hypotension, echocardiography was done, which showed normal left ventricular ejection fraction of 65%. Gradually, sensorium improved in the form of intermittent awakening after 48 hours, and GCS became 13/15 by day five. Patient became afebrile after four days. Hemodynamic improvement started only after day three, and vasopressors were slowly tapered off. Ventilatory requirements which were high initially, also decreased from day three and in view of neurological and hemodynamic stability, child was weaned off from ventilator on day five and extubated. Repeat hemogram, CRP and Chest *X*-ray became normal by eighth day. Neuroimaging could not be done initially as the child was critical, and was refused later by parents in view of clinical improvement. She was discharged after nine days of hospitalization in a stable condition on oral clindamycin (total duration of 14 days).

Ivermectin, in standard therapeutic doses, has both excellent parasiticidal efficacy and high tolerability [1]. Ivermectin does not readily cross the blood-brain barrier (BBB) in humans as it is effluxed by ATP-binding cassette subfamily B member 1 (ABCB1) transporter also called P-glycoprotein drug pump or mdr-1 located in the blood/brain barrier [1,3]. Hence, neurological adverse reactions are rare unless there is overdosage [1]. Our patient ingested 30 mg/kg of ivermectin, which was almost 100 times the recommended dose. Usually, a single oral dose of 150 to 300 mcg/kg is recommended, and 200 mcg/kg in scabies [4,5]. We suspected ivermectin poisoning due to the history, since encephalopathy and coma are well-documented side effects of ivermectin treatment in animals, and after ruling out other usual causes of coma. Severe neurological toxicities have been reported in public health programs in Africa, possibly due to concomitant infestations with high densities of loa loa, genetic predisposition, and co-infestations [1,6]. Additional intake of drugs that inhibit CYP3A4 and polymorphisms in the *mdr*-1 gene could also result in toxicity [1]. A recent case report of ivermectin taken in recommended dose, by a 13-year-old child, attributed the resulting neuro-toxicity, to human *ABCB1* nonsense mutations, which had led to loss of the neurological protective ABCB1 activity [3].

In our patient, despite there being no specific antidote, vigorous monitoring, and supportive critical care treatment proved to be lifesaving.

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Multisystem Inflammatory Syndrome in Children Related to COVID-19 With Urticarial Vasculitis – A Double Whammy!

There is still a dearth of data of the involvement of skin in the coronavirus disease 2019 (COVID-19), especially in pediatric patients. Herein, we describe the report of a child with COVID-19 related multisystem inflammatory syndrome in children (MIS-C), who developed hypocomplementemic urticarial vasculitis syndrome (HUVS) after recovery.

A previously healthy 18-month-old boy with a five day history of fever and abdominal tenderness was admitted to the pediatric in-patient department. His mother was suffering from COVID-19. On mucocutaneous examination, the child had multiple annular polycyclic erythematous plaques on trunk with conjuctival erythema (**Fig. 1**). The lesions had been rapidly progressive and persistent for the last three days. The child was febrile (39.4°C) and hypoxemic. The child was also experiencing diarrhea for three days along with hypotension (blood pressure 90/60 mmHg). Laboratory investigations revealed a positive RT-PCR for SARS-CoV-2 on two tests done three days apart, along with metabolic acidosis, leukocytosis, neutrophilia, lymphopenia, anemia and hypoalbuminemia with albuminuria.