

including fever, ionic disturbances (hyponatremia, hypophosphatemia), as well as damage to the myocardium itself in the course of PIMS-TS syndrome (i.e., changes in the coronary arteries) with negative family history [5-7].

In conclusion, Brugada syndrome is a disease, which, when detected too late, can result in SCD. However, as our two cases show, its diagnosis as well as the implementation of appropriate preventive therapy is not always easy.

Note: Both authors contributed equally to this work.

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Infantile Anti-N-Methyl-D-Aspartate Receptor Encephalitis Post-SARS-CoV-2 Infection

The spectrum of neurological conditions associated with severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) is evolving. Here, we describe a case of N-methyl-D-aspartate receptor encephalitis (NMDAR-E) with possible temporal association with SARS-CoV-2.

A 10-month-old typically developing boy presented with poor feeding and irritability for 5 days. On day 3 of illness, he developed fever and loose stools with 2 episodes of convulsions on day 5 of illness, when he was brought to our hospital. It was associated with loss of pre-morbidly normal eye contact. He had an upper respiratory tract infection (URTI) 40 days prior to illness onset. At presentation, his axillary temperature was 98.2^o F, pulse rate was 92/minute, respiratory rate was 24/minute and blood pressure was 84/54 mmHg. General physical and systemic examinations were unremarkable. On neurological examination, baby was not interested in surroundings and had poor interaction with caregivers. Cranial nerve examination was unremarkable. Motor system examination revealed normal power and tone, with brisk deep tendon reflexes. Peri-oral dyskinesias and bilateral striatal toe were present. Cerebellar and meningeal signs were absent. In view of fever, diarrhea, seizures, acute onset encephalopathy with extrapyramidal movements, possibilities considered at admission were post-infectious immune-mediated conditions (central nervous system demyelination, autoimmune encephalitis, post-COVID multisystem inflammatory syndrome (MIS-C)) and inherited metabolic disorder. Prior to referral, baby had a normal cerebrospinal fluid (CSF) study and C-reactive protein (CRP) with elevated white cell count (WBC, 26×10⁹/L).

Initial investigations at our center revealed elevated WBC (24 ×10⁹/L, N61L30), normal CRP (1 mg/L) and procalcitonin (0.25 ng/mL). SARS-CoV-2 IgG antibodies were strongly positive (index-20.7, >1.0 positive). Erythrocyte sedimentation rate (22 mm/first hour), lactate dehydrogenase (515 U/L), ferritin (19.5 ng/mL) and echocardiography (normal) were not consistent with MIS-C.

Over the next 24 hours, extrapyramidal movements worsened with appearance of generalized and oro-linguo-buccal dystonia with athetosis. Hence, possibility of anti-NMDA encephalitis was considered. MRI brain was normal. CSF showed 20 cells (95%L), sugar 65 mg/dL (blood sugar: 102 mg/dL), protein 27 mg/dL. CSF-polymerase chain reaction (PCR) was negative (*Escherchia coli K1, Hemophilus influenzae, Listeria monocytogenes, Streptococcus agalactiae, Streptococcus pneumoniae, Cytomegalovirus, enterovirus, HSV1, HSV2, HHV6, Human parechovirus, Varicella zoster virus and Crypto-coccus neoformans/gatii*). CSF sample for NMDA antibodies was sent to the laboratory.

Child was started on intravenous immunoglobulin (2 g/kg) and pulse methylprednisolone (30 mg/kg/day for 5 days) on day 8 of illness. CSF sample was reported strongly positive for anti-NMDA antibodies (indirect immunofluorescence assay). Computed tomography (CT) of abdomen and pelvis for tumor screening was negative. By day 5 of pulse steroids, there was no improvement in extrapyramidal movements or encephalopathy. Considering severe infantile form of anti-NMDAR encephalitis poorly responsive to first line therapy, weekly rituximab infusion (375 mg/m²/dose/week for 4 doses) was initiated in the second week of illness, along with addition of azathioprine for long-term immunosuppression (2 mg/kg/day). Two weeks after last rituximab dose, baby remained encephalopathic. Extrapyramidal movements were partially controlled with clonidine, baclofen and clonazepam. In view of refractory disease, monthly cyclophos-

phamide (750 mg/m²/dose) was administered for 3 doses. Following the first dose, baby achieved sustained eye contact and neck-control within a week. By one month, he could recognize parents, sit with support, creep and vocalize; mild oro-motor dyskinesia and bilateral hand athetosis persisted. Symptoms completely resolved after the second cyclophosphamide dose. Steroids were tapered off over 3 months after initial pulse dose. At the time of last follow-up, extrapyramidal movements were well controlled and baby was regaining age-appropriate milestones.

To the best of our knowledge, this is the first case of anti-NMDAR-E associated with SARS-CoV-2 in an infant aged <12 months. Anti-NMDAR-E, characterized by severe movement with encephalopathy, can be triggered by viral infections or tumors. Herpes simplex virus (HSV) encephalitis is the most commonly associated viral trigger, and can result in anti-NMDAR-E 4-6 weeks, or longer after an acute encephalitis episode [1].

SARS-CoV-2 is known to result in strong immune activation, which is broadly termed as MIS-C [2]. Post-SARS-CoV-2 immune-mediated manifestations can present within two weeks to a median of 25-45 days after an acute infection [3]. Anti-NMDAR-E associated with SARS-CoV-2 has been reported in only three children aged 23 months [4], 7 years [5] and 14 years [6]. All three children had a positive SARS-CoV-2 RT-PCR with evolution to encephalitis from acute infection in two children, and no clinical infection in one child. In our case, only IgG antibodies were positive, indicating a prior infection, which on history may be correlated with the preceding URTI. Considering the high population seropositivity, a true cause-effect relation cannot be ascertained. A positive RT-PCR test during the acute URTI episode and a positive family history would have strengthened

the causal association. Molecular mimicry probably best explains the pathogenesis for SARS-CoV-2 associated anti-NMDAR-E. Whether it can result in late-onset CNS encephalitis, similar to HSV encephalitis, remains to be elucidated. The present report, in conjunction with previous reports, supports the association of SARS-CoV-2 with NMDARE. Future research focusing on association between SARS-CoV-2 and early autoimmunity can help understand the underlying pathogenesis.

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ERRATA

Please note following corrections in the article titled “Early goal-directed therapy with and without intermittent superior vena cava oxygen saturation monitoring in pediatric septic shock: A randomized controlled trial.” published in *Indian Pediatr.* 2021;58:1124-30.

In Table I, which shows baseline characteristics of the study participants, mean (SD) lactate value in the control group should be ‘4.6 (2.9) mmol/L’ in place of ‘66.3 (10.4) mmol/L.’ in the same table, mean (SD) ScvO₂ in the intervention group should be ‘66.3(10.4)%’ in place of ‘4.6 (2.9)%.’

Appropriate corrections have been done in the web version at <https://www.indianpediatrics.net/dec2021/1124.pdf>

Please note following corrections in the article titled “Low-dose (0.05 unit/kg/hour) vs standard-dose (0.1 unit/kg/hour) insulin in the management of pediatric diabetic ketoacidosis: A randomized double-blind controlled trial” published in *Indian Pediatr.* 2021;58:617-23.

On page 620, column II, first para, second sentence should be “The hypokalemia was more in malnourished children in the standard-dose group ($P=0.31$), and more children in the standard-dose group required a higher concentration of dextrose and tapering of insulin infusion at least once to counter the falling blood glucose (Table II).” in place of “The hypokalemia was more in malnourished children in the standard-dose group ($P=0.31$), and more children in the standard-dose group required a higher concentration of dextrose and tapering of insulin infusion at least once to counter the falling blood glucose $P>0.005$.”

Appropriate corrections have been done in the web version at <https://www.indianpediatrics.net/july2021/617.pdf>