CORRESPONDENCE



Acute Fulminant Cerebral Edema Presenting as Refractory Status Epilepticus in a SARS-CoV-2 PCR–Positive Child without Pulmonary Involvement

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To the Editor: CNS manifestations of SARS-CoV-2 infections are due to direct invasion of the virus and/or an immune response (cytokine storm).

We report a 10-y-old boy who presented with coryzal febrile illness and repetitive GTCS with a history of two brief seizures at 9 y (normal MRI and EEG without treatment). Over the course of 12 h, due to repetitive seizures and the worsening of GCS, he was ventilated. Multiple antiseizure medications and symptomatic treatment were instituted as per protocol. Continuous EEG showed periodic lateralized epileptiform discharges (PLEDS) with a few brief electrographic seizures, hence midazolam infusion was started and maximized (6 h) followed by thiopental, to achieve a burst suppression pattern within 12 h. Hemogram, biochemistry, CXR, MRI brain, and CSF (with viral PCR) were normal. Nasal swab SARS-CoV-2 RT-PCR was positive (with a negative RAT). At 48 h, EEG showed disappearance of the burst suppression pattern with nonreacting bilateral equal pupils, while MRI revealed global hypoxic insult with bilateral uncal, tonsillar, and transtentorial herniation. On the 14th day, the child succumbed to illness with diagnosis of RSE in SARS-CoV-2 PCR-positive with AFCE.

AFCE due to SARS-CoV-2 has not been reported from India. Systemic effect of viral pathogen and/or trigger of electrophysiological cascade in previously epileptic patient may be an association or mere coincidence.

AFCE was defined as encephalitis that progressed to diffuse cerebral edema on neuroimaging and/or autopsy, excluding other recognized etiology like toxin, etc. with significant mortality [1]. A 9-y-old girl [2] and a 7-y-old boy [3] died from AFCE (COVID-19 PCR +) in the absence of a viral pathogen in the CNS. Six other children from Taiwan also presented with encephalopathy and seizures, while onset of neurological symptoms to signs of brain herniation ranged from 0 to 39 h [4].

AFCE in febrile encephalopathy should be timely diagnosed with adequate clinical and radiological evidence to aid appropriate management and prognostication.

Declarations

Conflict of Interest None.

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