



## Acute Encephalitis Beyond the Usual in the COVID Era

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*To the Editor:* An 8-y-old female child presented with a history of fever for 10 d, severe headache, and multiple episodes of vomiting. On admission she was delirious; and other than bilateral conjunctival congestion and oral mucositis, remaining clinical examination was noncontributory. In view of acute encephalitis syndrome, she was started on ceftriaxone, acyclovir, and doxycycline.

Investigations revealed Hb 6.6 g/dL, WBC count 13,600 cells/cumm with 75% neutrophils, platelet count 60,000 cells/cumm, CRP 135.3 mg/L. Serology for dengue, scrub typhus, smear for malarial parasite were negative. MRI brain was normal. Over the next 36 h, drowsiness increased with episodic violent behavior. In view of nonpurulent conjunctivitis, mucositis, thrombocytopenia, and very high CRP, an inflammatory etiology was considered and further tests showed ferritin 358 ng/mL, NT Pro BNP 20,774 pg/mL, D-dimer 5.81, and negative COVID RT-PCR, while COVID IgG was positive. Echocardiography showed left anterior descending (LAD) coronary aneurysm(+4.84 Z).

She was diagnosed as multisystem inflammatory syndrome in children (MIS-C) and started on IV immunoglobulin (IVIg) 1 g/kg with methylprednisolone 10 mg/kg/d for 3 d followed by oral prednisolone (2 mg/kg), which was gradually tapered and stopped. Subcutaneous enoxaparin was also initiated along with low-dose aspirin. Mentation improved and echocardiography after 1 wk showed diminution of LAD aneurysm. CSF was planned but was eventually not done as she improved on immunomodulatory therapy.

Neurological manifestations in SARS-CoV-2 are reported mainly in adults and include encephalitis, meningitis, encephalopathy, stroke, seizures, anosmia [1], possible

pathophysiology being direct CNS invasion by the virus [2]. However, very few studies describe similar affection with MIS-C [3], and here the encephalopathy is attributed as a feature of systemic inflammatory response.

With the ongoing pandemic in the absence of a known cause, MIS-C should be a differential in patients with febrile encephalopathy and elevated inflammatory markers. Timely institution of IVIg and corticosteroids [4] may be lifesaving.

### Declarations

**Conflict of Interest** None.

### References

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