

Case Report

Acute disseminated encephalomyelitis in SARS-CoV2 seropositive child

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ABSTRACT

In this case report, a 2-year-old boy presented with UMN type of diplegia with involvement of cerebral cortex. He was afebrile with normal inflammatory markers, negative RT-PCR throat swab, negative COVID IgM and positive IgG antibodies with CSF showing elevated proteins with lymphocytosis on a background of MRI brain depicting a picture of white matter hyperintense signals on T2 FLAIR and resolution on follow-up MRI which confirms the possibility of acute disseminated encephalomyelitis (ADEM) following recent SARS-CoV2 infection.

Keywords: Diplegia, ADEM, SARS-CoV2

INTRODUCTION

Majority of children infected with SARS-CoV2 were asymptomatic or mildly symptomatic. In pediatric age group, SARS-CoV2 had varied neurological presentations ranging from headache, dizziness, anosmia, ageusia, myopathy, fatigue to serious presentations as encephalitis, meningitis, stroke, acute transverse myelitis and Guillain-Barre syndrome.¹ In paediatric age group very few reports of neurological manifestations have been described.² Here we report a case of ADEM in SARS-COV-2 sero-positive child.

CASE REPORT

A previously healthy 2-year-old boy presented with fever for 1 week. Four days after the onset of fever, child had acute, progressive, bilateral lower limb weakness (inability to run, walk and stand). He had no history of breathing difficulty, swallowing difficulty, drooling of saliva, nasal regurgitation. No H/o cranial nerve palsy, LOC, Seizures, sensory disturbance and visual disturbance. No H/o neck pain, photophobia, tingling or numbness. No H/o abdominal pain, rash or recent vaccination.

On admission, child was irritable, afebrile with stable vitals and maintaining saturation in room air. Neurological examination revealed normal cranial nerves and fundus. Gag reflex was present. Motor examination revealed a power of 4/5 in both upper limbs and 2/5 in both lower limbs with exaggerated DTR. Plantar reflex was bilateral extensor. Ankle clonus was present. He had no neck flexor weakness or diaphragmatic weakness.

Laboratory data showed leukocytosis (17×10^3 WBC/ μ l) with lymphocytic predominance. Hemoglobin, renal and liver function tests were normal. CRP 4mg/dl, ESR 16mm/hr., ferritin 220 ng/ml, LDH 275 U/l, D-dimer 98 ng/ml, Triglycerides 154 mg/dl. PT, APTT-normal, ECHO-normal. Lumbar puncture was performed which revealed normal opening pressure, clear CSF, elevated protein (0.6 g/dl) with lymphocytic pleocytosis (52 cells/ mm^2) and Normal glucose (0.1 mg/dl). RT-PCR for SARS-CoV2, Zika virus and Dengue virus serotypes 1 to 4 (DENV 1-4) were negative in the CSF. Chest x-ray was normal. Magnetic Resonance Imaging (MRI) of the brain was done on fourth day of onset of neurological symptoms which showed symmetrical T2/FLAIR hyperintensity in periventricular white matter of both parietal lobes suggestive of ADEM. There was no optic

neuritis, hemorrhagic deposits or calcification in the brain parenchyma.

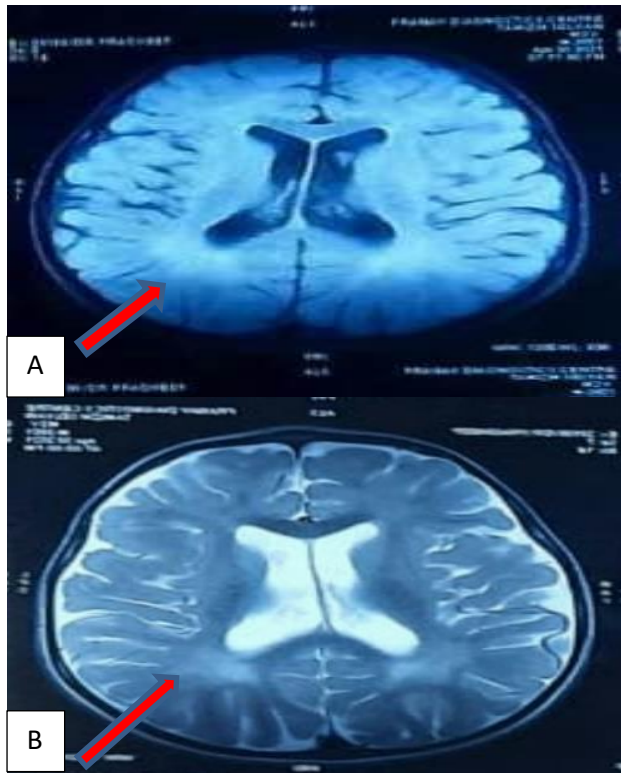


Figure 1 (A and B): Axial FLAIR magnetic resonance imaging brain showing hyperintense lesion in bilateral periventricular region (arrow).

In view of SARS-CoV2 pandemic, a possible neurological manifestation of COVID-19 was suspected. A serological test and RT-PCR test for SARS-CoV2 was performed which showed elevated SARS CoV2 IgG levels and normal IgM levels. RT PCR turned out to be negative at the time of admission and discharge. Child was treated with Methyl prednisolone (30 mg/kg/day) for 5 days. Child clinically improved. He was discharged after 7 days with oral steroids tapering over 6 weeks. On follow up MRI at 3 months of onset of the illness, there was resolution with no neurological deficits.

DISCUSSION

Acute disseminated encephalomyelitis is an inflammatory, demyelinating event in early childhood presenting with acute onset polyfocal neurological deficit accompanied by encephalopathy and changes compatible with demyelination in brain MRI.³ Pathogenesis of

ADEM involves a possible molecular mimicry induced by an infection or immunization triggering CNS auto-antibody production.⁴ International paediatric multiple sclerosis society group put forth the following diagnostic criteria for ADEM. The criteria involve 4 parameters namely, 1. A first polyfocal clinical CNS event with presumed inflammatory demyelinating cause 2. Encephalopathy (unexplained by fever, systemic illness or post-ictal symptoms), 3. Brain MRI consistent with demyelination during the acute phase (<3 months), 4. No new clinical or MRI findings 3 months or more after the clinical onset.⁵ In this case report, child had elevated IgG COVID antibody levels in the blood with CSF showing elevated proteins with lymphocytosis on a background of MRI brain showing white matter hyperintense signals and resolution on follow-up which confirms the possibility of ADEM following recent SARS-CoV2 infection.

CONCLUSION

Through this case report, we present an unusual post-infectious neurological complication of SARS-CoV2 infection in children with good outcome.

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