

## Acute Ataxia and Myoclonus in COVID-19: A Case Series

Sir,

Acute ataxia and myoclonus in adults have a variety of causes including postinfectious cerebellar immune-mediated inflammation.<sup>[1]</sup> Over the past nine months, numerous neurological complications of the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) have been reported. Amongst the various neurological manifestations, acute cerebrovascular accident and encephalopathy have been frequently published in literature.<sup>[2]</sup> Although seen in association with other viral infections, myoclonus in coronavirus disease 2019 (COVID-19) has been rarely reported.<sup>[3,4]</sup>

We present a case series of three patients presenting with movement abnormalities in relation to infection with COVID-19.

The first patient, a 60-year-old male, presented with acute onset involuntary jerking movements of upper and lower limbs, and difficulty in walking, of 10 days' duration, following fever and cough 2 weeks prior. Clinical examination revealed normal vital parameters and oxygen saturation, normal mentation, ocular movements and speech. He had bilateral limb ataxia, dysmetria and gait ataxia [Video 1a]. There was generalized myoclonus on posture and action, which was not sensitive to tactile or auditory stimuli [Video 1b]. Negative myoclonus was not present.

The patient's routine haemogram, biochemistry and thyroid function tests were normal. Chest radiograph showed bi-basal fluffy shadows suggestive of COVID-19. Computed tomography (CT) of brain with contrast was normal. His throat swab was positive for SARS-CoV-2 by cartridge-based nucleic acid amplification test (CBNAAT). COVID-19 IgM and IgG antibodies were present in serum. Cerebrospinal fluid (CSF) was positive for COVID-19 IgG antibody but was otherwise normal.

Following treatment with intravenous and oral steroids, oral levetiracetam and clonazepam, patient made a complete recovery over 2 weeks [Video 1c].

The second patient was a 53-year-old male, with acute onset of generalized jerky movements, imbalance and difficulty in walking for 5 days, following fever a week prior. His vitals (including oxygen saturation at admission), general and neurological examination including speech and eye movements were normal. He had bilateral limb ataxia and dysmetria, along with marked gait ataxia. Multifocal action myoclonus was present, with no stimulus sensitivity to touch or sound [Video 2a]. He had a peculiar bouncing gait. His haemogram, renal and liver function tests, electrolytes and thyroid functions were normal. CT brain with contrast was unremarkable. His throat swab was negative for SARS-CoV-2 (CBNAAT). COVID IgM antibody was present in serum, and IgG antibody was detected in serum and CSF. CSF was acellular with normal protein and sugar levels. High-resolution computed tomography (HRCT) chest showed no evidence of acute COVID-19.

The patient was treated with intravenous methylprednisolone, oral steroids and clonazepam, with complete recovery over 10 days [Video 2b].

The third patient was a 47-year-old male who presented with involuntary jerking movements of limbs at rest and on activity, and one episode of generalized convulsions, 10 days after an episode of fever.

He had truncal and limb ataxia with postural kinetic tremors in the upper limbs in addition to action myoclonus [Video 3a]. There was no sensitivity to touch or sound. He also had intermittent chaotic, multidirectional saccadic oscillations suggesting opsoclonus [Video 3b]. Speech was dysarthric with a jerky, explosive component. Patient was hypoxic on room air.

His routine haemogram, renal and liver functions, blood sugars and thyroid functions were normal. CT brain with contrast was unremarkable. His throat swab was positive for SARS-CoV-2 (CBNAAT). COVID IgM antibody was present in serum, and Ig G antibody was detected in serum and CSF. CSF was acellular with normal protein and sugar levels. HRCT chest showed COVID severity score of 12/25.

The patient was treated with intravenous methylprednisolone for 5 days, followed by oral steroids for a week, along with oral clonazepam, with good response to treatment [Video 3c].

A wide range of neurological findings have been described in COVID-19. These range from hypogeusia and hyposmia in

mild cases to diffuse corticospinal tract signs, encephalopathy and movement disorders in severe cases.<sup>[2]</sup>

The pathophysiology of neurological involvement in COVID-19 is postulated to be a para or postinfectious immune-mediated mechanism or direct neuroinvasion by SARS-CoV.<sup>[5,6]</sup>

In our short case series, the three patients developed myoclonus and ataxia approximately 7–14 days after the onset of fever. The third patient had the classic ocular features of opsoclonus, in addition. Although none of them had encephalopathy, the third patient had a single episode of convulsions. This patient also required oxygen support, unlike the two other patients.

CT brain with contrast was normal in all three patients, a finding similar to that reported with other cases of ataxia-myoclonus in COVID-19.<sup>[3,6]</sup> Magnetic resonance imaging of the brain, which could have picked up subtle changes, was not possible due to logistical problems during the pandemic.

SARS-CoV-2 has been detected in a single case report, in the CSF fluid of a patient with meningoencephalitis.<sup>[7]</sup> Other studies, failing to detect SARS-CoV-2 in CSF, have proposed post-viral autoimmunity or molecular mimicry between the viral particles and neuronal autoantigens, as the cause for the neurological complications.<sup>[8]</sup>

Our patients had positive serum and CSF antibody test (with normal routine CSF examination). In the published literature, CSF studies for COVID antibody IgG have been positive even in the absence of a positive CSF or nasopharyngeal SARS-CoV-2 RT-PCR.<sup>[9,10]</sup> The fact that our patients already had serum and CSF IgG positivity points to a possible reinfection with COVID-19 at this episode, having already developed IgG response to a previous infection 6–8 weeks ago. Whether a reinfection with COVID-19 in the setting of IgG positivity triggers an enhanced autoimmune response causing CNS involvement is a matter of interest and debate.

Besides treatment for the neurological syndrome, treatment for COVID-19 in our patients included antibiotics, low-molecular weight heparin, and oral ivermectin and doxycycline. In the reported cases, the neurological treatment ranged from only symptomatic therapy to immunotherapy and symptomatic treatment with levetiracetam, clonazepam, valproic acid and lorazepam, with good response.<sup>[3,4,6]</sup>

### Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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**Conflicts of interest**

There are no conflicts of interest.

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