# Case Report

# Generalised Myoclonus and Cerebellar Ataxia Associated with COVID-19 : A Case Report

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COVID-19 pandemic is a Global burden to Public Health. An array of Neurological Manifestations have been reported to be associated with COVID-19 like Anosmia, Cerebrovascular accident, Meningitis, Encephalitis, Seizures, Guillain-Barré Syndrome (GBS), Acute Disseminated Encephalomyelitis (ADEM) etc. Generalised Myoclonus and Cerebellar Ataxia, is a less common Neurological Manifestation when compared to others. Here, we report a case of Generalized Myoclonus and Cerebellar Ataxia following COVID-19 infection. The possible mechanisms of Myoclonus and Ataxia following COVID-19 are also discussed.

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### CASE REPORT

The COVID-19 pandemic may present with various Neurological Manifestations, of which movement disorders are rare. We encountered this middle aged male with COVID-19 infection who developed generalized Myoclonus and Cerebellar ataxia which responded well to immunotherapy.

A 52-year-old gentleman, diabetic, hypertensive, chronic smoker, non alcoholic presented with complaints of cough and breathlessness associated with generalised tremulousness and unsteadiness of 7 days duration. He had jerky movements of both upper limbs and lower limbs at rest, which was predominantly proximal and exacerbated on doing activities. The unsteadiness was present on standing and walking with sway to either sides while walking. He did not give history of exacerbation of unsteadiness in dark. He gave no history of sensation of walking on cotton wool. There was no history of vertiginous sensation or Tinnitus. He also complained of fatigue, decreased appetite and weight loss. Fifteen days before Admission, he had low grade fever which resolved after taking Anti-pyretics for 3 days.

**Examinations** — On examination, the patient was conscious and oriented. There was no clouding of consciousness. Cranial nerve examination was normal. Bulk, Tone, Power and deep tendon reflexes were normal in all four limbs. The sensory system examination was normal. He had action induced myoclonus involving trunk,

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#### Editor's Comment :

Neurological manifestations are seen in all grades of severity of COVID-19. So further Neuroepidemiological studies and International registries are needed to help define the full Neurological spectrum of SARS-CoV-2 disease. Further research is needed to prove the plausible post or para infectious origin of Myoclonus and Ataxia.

upper limbs and lower limbs. He had bilateral horizontal gaze evoked nystagmus, which was ill- sustained. Mild scanning dysarthria was present. Finger, finger- Nose Test and Heel-shin test were suggestive of Bilateral Limb Ataxia. He had severe degree of stance and Gait Ataxia. Tandem walking was not possible. There was no bradykinesia.

Routine blood tests including Complete Blood Count, Random Blood Sugar, Liver Function Tests, Serum Electrolytes and Renal Function Tests were normal. Thyroid profile was normal. HIV serology was negative. Inflammatory markers including CRP, D-dimer, IL-6 and serum ferritin were within normal limits. EEG was normal. CT scan chest (Figs 1&2) showed consolidation in Right Lung with Right Pleural Effusion along with few patchy opacities in left lung suggestive of COVID bronchopneumonia with secondary infection.

MRI Brain with contrast study was normal. CSF analysis was normal. Reverse Transcription Polymerase Chain Reaction (RT-PCR) for SARS-CoV 2 was positive. Serum Anti-nuclear antigen. Rheumatoid factor, C-reactive Protein (CRP) and Extractable Nuclear Antigen Profile were negative.

The patient was treated with seven days course of IV antibiotics, IV Dexamethasone 8mg tds and prophylactic dose of anticoagulants. Patient was given 4 litres/min Oxygen for 2 days, after which he was maintained on room air. On the 5th day after admission, he was started on Tab Sodium Valproate 600mg/day and Tab

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Fig 1 & 2 — CT chest showing consolidation in right lung with right pleural effusion along with few patchy opacities in left lung suggestive of Covid bronchopneumonia with secondary infection

Clonazepam 1mg at bed time for symptomatic management of Myoclonus. Patient improved significantly over the next one week. Repeat CT chest with contrast showed complete resolution of pleural effusion and partial resolution of pneumonia. Sputum was negative for acid fast bacilli.

## **D**ISCUSSION

Our patient had subacute onset generalised myoclonus and cerebellar ataxia associated with COVID-19 infection. Respiratory manifestations of COVID-19 are well documented, but rare Neurological manifestations are also being recognised. They are being reported not only in severe cases but also in mild and moderate cases.

Our patient had generalized myoclonus, predominantly involving the proximal limbs. As there was no jerk locked cortical potential in EEG associated with the Myoclonus, the Myoclonus was probably of subcortical origin. The patient had Pancerebellar involvement, as evidenced by Nystagmus, Dysarthria, Bilateral limb ataxia, Truncal ataxia and Stance / Gait ataxia.

The following are the possible mechanisms by which COVID-19 causes neurological complications: 1-3

- Viral Neurotropism, accompanied by vascular, transcribrial and/or Neuronal retrograde dissemination.
- Autoimmune response, by means of Molecular Mimicry and Cytokine storm.
- Multiorgan dysfunction, due to Cardiorespiratory and metabolic causes.

Our patient had no anosmia or clouding of consciousness. CSF analysis and MRI Brain were normal. Hence viral neurotropism seems less likely. Patient developed myoclonus and ataxia during the 2<sup>nd</sup> week of illness, progressed over 10 days and later started improving. There were no biochemical abnormalities [Random Blood Sugar (RBS), RFT, LFT and Serum Electrolytes were normal]. There was no history suggestive of hypoxic brain injury. There was no history of intake of drugs causing Myoclonus (like

Fluoroquinolones, Opioids, Antipsychotic and Antidepressants). Hence, para infectious etiology of Myoclonus and Cerebellar Ataxia due to autoimmune response seems more likely.

On reviewing literature, we found many case reports<sup>4-6</sup> similar to our case. In the earlier published reports, patients had presented in the 2<sup>nd</sup> or 3<sup>rd</sup> week of illness, mean age of presentation was 53.2 years and all patients were males. In the above reports, few patients were on steroids, few patients got steroids and IVIg and few others underwent Plasmapheresis also, depending upon the severity of illness.

But all patients responded well to therapy, indicating an autoimmune Etiopathophysiology. Time of onset of illness after COVID (2<sup>nd</sup> or 3<sup>rd</sup> week), subacute course, good response to Immunomodulatory Therapy, monophasic illness and exclusion of structural, metabolic and toxic causes, favour a parainfectious or postinfectious autoimmune response.

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