Para-infectious cerebellar ataxia post Covid 19 infection

A 65 year old non-smoker hypertensive and diabetic male on permanent pacemaker (for complete heart block 5 years back) was admitted to our hospital for severe covid pneumonia and RT-PCR was positive. He was discharged from the hospital after he recovered from his illness and was Covid-19 RT-PCR negative. One week later he was again admitted to our Neurology department with complaints of difficulty in maintaining balance while walking. Two days back patient had started with difficulty in holding severe tremors in either of his hands while holding a cup of tea. On trying to get up and walk, patient could not maintain himself in normal posture and would develop severe tremors in his legs and would sway to either side. He had no history of weakness of any body part, abnormal body movements or alteration in sensorium. There was no history of drug intake known to cause imbalance of gait. On examination he was afebrile, hemodynamically stable with normal respiratory, abdomen and cardiovascular examinations. His neurological examination revealed normal higher mental functions, normal sensory and motor examinations. However he had cerebellar signs in the form of bidirectional nystagmus, severe dysarthria, severe intentional tremors, bilateral disdiadokinesia, gait ataxia and dysmetria. Based on these acute onset cerebellar symptoms and signs, a diagnosis of cerebellitis as made. Baseline investigations and thyroid profile were normal. CT scan of the brain was normal; MRI could not be done in view of permanent pacemaker. CSF analysis was normal. CSF for autoimmune, paraneoplastic and neurotrophic viral panel was negative. Patient was diagnosed as a case of post-infectious cerebellitis due to Covid-19 infection and was treated with methylprednisolone (1 gram/daily for 5 days) pulse therapy. He improved significantly with treatment and was able to walk without support and without developing tremors of limbs. His speech also improved significantly.

Neurological manifestations of Covid-19 infection are being reported worldwide, affecting both central and peripheral nervous system. Although exact mechanisms are not known, direct neuronal injury, immune mediated injury and injury secondary to hypoxemia have been proposed as etiopathogenic mechanisms responsible for the neurological involvement of human coronavirus disease 2019 (COVID-19). The virus may gain access to central nervous system either through haematogenous route or through olfactory bulb. Most of the studies report neurological involvement in 20-40% of patients hospitalised due to Covid 19 virus infection; mostly in the form of encephalopathy, meningitis, acute disseminated encephalomyelitis and strokes. Cerebellar ataxia occurring post Covid 19 infection has been rarely reported in literature further adding to gamut of neurological involvement of Covid 19 infection. Since, in our patient, ataxia occurred after Covid 19 infection had resolved and CSF was normal, the cerebellar involvement was possibly immune mediated in nature. The rapid response with steroid pulse therapy further strengthens this hypothesis. However CSF Covid-19 RT-PCR could not be done as it is not available in our country and is a limitation of this case report. As this pandemic has surrounded humanity on all sides and varied neurologic manifestations of this infection are being recognised, neurologists should remain updated and should not ignore any of these clinical presentations.

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