Movement Disorders
CLINICAL PRACTICE

CORE-Myoclonus Syndrome: A Proposed Neurological Initial Manifestation of COVID-19

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We read with great interest the publication by Schellekens et al.[1] providing insights into a case of postinfectious myoclonus *combined* with cerebellar ataxia after SARS-CoV-2 infection.

Three other COVID-19 patients with myoclonus were summarized in the same issue of *Movement Disorder Clinical Practice*. All of whom had severe respiratory symptoms and required intensive care.[2] Three further myoclonus-cases in COVID-19 in mostly severe manifestation were reported elsewhere.[3] However, Schellekens et al. point out that myoclonus can also occur in minor affected patients.

Common in most cases was the delayed onset of myoclonus with regard to respiratory symptoms, leading to the hypothesis of a postinfectious, immune-mediated pathomechanism.

We would like to add two case reports to the observations of Schellekens et al. showing that myoclonus occurs also in otherwise mild COVID-cases and, moreover, can be part of the initial neurological symptoms and thus occurs para- rather than postinfectious.

As first case, we report a 29-year old female health-care worker with a history of autoimmune-associated diseases (atopic eczema, alopecia areata) who was admitted 9 days after positive for SARS-CoV-2-RNA-swab in a screening examination as contact person. Three days before she had unremarkable symptoms (slight nausea, disgust, loss of appetite). On admission, she experienced myoclonic jerks of her left arm with phasic dystonia of her shoulder and trunk. Neurological examination further showed upper-limb-ataxia and a unilateral ptosis. She did not have any major respiratory symptoms or fever. An extensive workup revealed i.a. autonomic dysfunction (hypertensive dysregulation in the Schellong-Test), periods of generalized slowing in the electroencephalography and slowed processing speed, impaired verbal fluency and verbal memory in neuropsychological testing. Proximal electromyography of arms and legs showed signs of myopathia and myositis. Cerebral MRI and cerebrospinal fluid (CSF) was normal including negative SARS-CoV-2-PCR. Blood tests showed lymphopenia, elevated LDH and isolated Serum-Borrellia-IgM (OspC) positivity with unclear relevance. The patient fully recovered after 10 days on the ward without specific treatment.

The second case, a 59-year old man, presented with myoclonic jerks and phasic dystonia of the neck and headache described as stabbing, high-intensity pain. No respiratory symptoms occurred. Neurological examination revealed slight dysmetria / ataxia and intention tremor of the extremities and left sided hypoesthesia. SARS-CoV-2-PCR swabs were positive. MRI showed FLAIR-hyperintensities (right cortical and subcortical parietal) as residuum of a stroke in 2008. Laboratory testing showed CSF-pleocytosis (15 cells/μL) and a mild blood–brain-barrier impairment. A monoclonal banding / paraproteinemia was identified. The patient was treated with i.v. aciclovir until CSF-PCR for SARS-CoV-2, HSV, VZV, CMV, EBV and Entero-Virus were proved negative. The patient left hospital fully recovered 10 days after admission.

Together with the reports mentioned above and a previous published case from our clinic[4] we summarize main similarities of the patients: parainfectious onset, myoclonus/myclonus-ataxia/myoclonus-dystonia, mild symptomatic younger patients, in some cases autonomic dysfunction and sometimes prior autoimmune history or antiviral treatment history. We consider that syndrome a parainfections (rhomb)encephalitis.

We would like to propose a name that depicts the common property of all discussed cases as the central point of a NEURO-COVID manifestation: *Coronavirus-(rhomb)encephalopathy (CORE)*. Our cases initially manifested parainfectious as *CORE-myoclonus syndrome*. The cited literature[1,2,3] shows that this syndrome can also occur postinfectious as *CORE-myoclonus* or *-ataxia*. Finally, we found in two of our three cases autonomic dysfunction as part of the CORE-syndrome. We consider that finding also of interest.

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Although not proven, it can be discussed (besides i.a. cardiologic reasons) as one of multifactorial causes why syncopes or near-syncopes might have lead to a high number of hospital admissions due to COVID-19.[5]

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