LETTER TO THE EDITOR

Neuro-COVID Requires Comprehensive Work-up

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With interest, we read the article by Mehra et al. about a 13-year old female with the multisystem inflammatory syndrome (MIS) during an infection with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), clinically manifesting with multiorgan failure [acute demyelinating encephalomyelitis (ADEM), Guillain-Barre syndrome (GBS), renal insufficiency, myocardial damage, and hepatopathy].[1] The patient benefited from ventilatory support, steroids, immunoglobulins, and plasma exchange (PLEX).[1] It was concluded that MIS can manifest with severe compromise of the central and peripheral nervous system (CNS and PNS).[1] The study is appealing but raises the following comments and concerns.

The patient was diagnosed with GBS, but the subtype was not specified. According to the electrophysiological data, acute motor axonal neuropathy (AMAN) is the most likely subtype. However, diagnosing GBS according to the Brighton criteria requires cerebrospinal fluid (CSF) investigations and demonstration of a dissociation cyto-albuminique (DCA).[2] Unfortunately, CSF investigations were not carried out, so the diagnosis of GBS remains questionable.

Since various drugs frequently applied for the treatment of coronavirus 2019 (COVID-19) are neurotoxic, such as daptomycin, linezolide, lopinavir, ritonavir, hydroxychloroquine, cisatracurium, clindamycin, or tocilizumab [Finsterer, submitted], we should know if the patient received any of these compounds in addition to steroids, immunoglobulins, and PLEX. It is also crucial that critical ill neuropathy was excluded as a differential of muscle weakness.

The discrepancy between normal sensory nerve conduction studies (NCSs) and "absent motor responses to pain stimuli" should be explained. According to this statement, the patient rather had acute, motor and sensory, axonal neuropathy (AMSAN) than AMAN.

The same as with GBS is also true for ADEM. ADEM was diagnosed without CSF investigations (usually pleocytosis >50/3). No magnetic resonance imaging of the spinal cord was provided. Grey matter lesions in the basal ganglia and the thalamus were not described.[3]

We do not agree with the notion that simultaneous occurrence of CNS and PNS abnormalities in SARS-CoV-2-infected patients is rare.[1] If anosmia is regarded as PNS involvement in COVID-19, all COVID-19 patients with anosmia/hyposmia or with dysgeusia/hypogeusia and CNS disease should be regarded as COVID-19 patients with simultaneous CNS/PNS involvement. Furthermore, if myalgia is regarded as a symptom of muscle involvement in the SARS-CoV-2 infection, all patients with CNS disease and myalgia have simultaneous CNS/PNS involvement. Since ageusia/anosmia and myalgia are frequent manifestations of COVID-19, simultaneous CNS/PNS involvement in COVID-19 is highly prevalent. Furthermore, Sancho-Saldaña et al. reported a patient with GBS and leptomeningeal enhancement.[4]

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Recently, it has been recommended to apply infliximab to patients with MIS. Thus, we should know if infliximab was considered as a therapeutic option in the presented patient, and, if applied, if it was beneficial or not.

Missing is the specification of the myocardial damage occurring during the SARS-CoV-2 infection. We should know if the patient was diagnosed with myocarditis, endocarditis, or Takotsubo syndrome (TTS) increasingly recognized as cardiac complications of a SARS-CoV-2 infection.[5] It is also conceivable that the patient had developed an intraventricular thrombus since SARS-CoV-2 is frequently associated with thrombotic events. The discrepancy between high proBNP and only mild systolic dysfunction should be explained.

Missing are neurological exam and quantification of quadriparesis. Missing are the electroencephalography results. Missing are the NCSs of the phrenic nerve. A limitation is that Table 1 does not provide reference limits.

Overall, this interesting case report has a number of limitations, which should be addressed before conclusions as those presented are drawn. The main limitation is that no CSF investigations were carried out, making the diagnoses of both GBS and ADEM questionable. There is also a need to specify cardiac, gastrointestinal, and mucocutaneous involvement in MIS. Missing information should be provided.

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