SARS-CoV-2 and Its Neurological Implications

Abstract:

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CORRESPONDENCE

With interest we read the review article by Ellul, M. et al., (2020) about neurological abnormalities in SARS-CoV-infected patients (Ellul, M. et al., 2020). We have the following comments and concerns.

A neurological disease not addressed in the review is myasthenia. There are several reports indicating that SARS-CoV-2 can deteriorate myasthenia (Singh, S., & Govindarajan, R. 2020) or even trigger myasthenic crises (Delly, F. et al., 2020). Since patients with myasthenia are at increased risk due to their mostly immunological disease and immune-modulatory treatment, actions need to be taken to avoid a SARS-CoV-2 infection in these patients.

Other neurological manifestations not addressed in the review are muscle cramping, tics, and tremor reported in <1% of 917 Chinese SARS-CoV-2-infected (Xiong, W. et al., 2020). In some patients the virus may also trigger rhabdomyolysis.

We do not agree that Guillain-Barre syndrome has been reported in only 19 patients. In a recent meta-analysis, 24 patients with polyradiculitis were presented (Finsterer, J. et al., 2020). Fourteen patients were diagnosed with AIDP, four with AMAN, three with MFS, and two with AMSAN (Finsterer, J. et al., 2020). In none of them was the virus detected in the CSF, why a cross-reaction of the immune system against the virus and components of the radices or the extensive immunological response to the virus were made responsible (Finsterer, J. et al., 2020). Since this analysis (end of May) at least three more patients have been reported.

Encephalopathy is a hazy term indicating cerebral disease in general, that should be avoided. In a recent review of SARS-CoV-2-associated meningitis/encephalitis (submitted end of June) it turned out that the majority of the patients with SARS-CoV-2-associated encephalopathy has para-infectious, non-vascular, non-hypoxic, presumably immune-mediated CNS-disease, classified as auto-immune encephalitis (n=11), acute, hemorrhagic, necrotising encephalopathy (n=2), or acute disseminated encephalomyelitis (n=3). No virus was found in the CSF in any of them. Only in 14/48 patients CNS-disease was classified as infectious (meningitis (n=1), encephalitis (n=5), meningo-encephalitis (n=5), myelitis (n=3)). The virus was present in the CSF in only 4/14 patients. Fifteen/48 patients received virostatics, 21 antibiotics, 11 steroids, 5 immunoglobulins, 7 plasma exchange, 8 antiepileptics, 8 chloroquine, and 11 required mechanical ventilation. Twenty-two patients recovered, 2 did not, and 6 died.

In conclusion, infection with SARS-CoV-2 definitively concerns the neurologist and has therapeutic implications. Peripheral neuronal networks (pulmonary, enteric, pharyngeal, nasal) are affected before the CNS. There are indications that CNS/PNS are more widely and frequently directly or indirectly involved than anticipated. In most cases, SARS-CoV-2-associated CNS/PNS disease is immune-mediated. Thus, in most of these patients immuno-suppressive/immune-modulatory treatment, immunoglobulins, or plasma exchange should be considered.
REFERENCES