

Outcome of SARS-CoV-2 associated Guillain-Barre syndrome depends on early treatment and vaccination

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With interest we read the review article by Zuberbühler et al about 47 patients with SARS-CoV-2 associated Guillain-Barre syndrome (SAG) collected through a PubMed search as per the 4th July 2020 [1]. It was concluded that SAG resembles Guillain-Barre syndrome (GBS) triggered by agents other than SARS-CoV-2 in clinical presentation and response to treatment and that the question if the prevalence of GBS increased since the outbreak of the pandemic is unsolved [1]. The review is appealing but raises the following comments and concerns.

We do not agree with the statement that 'about 57% of COVID-19 patients' develop mild neurological symptoms [1]. Considering anosmia/hyposmia and hypogeusia/ageusia as neurological manifestations of COVID-19, neurological involvement is much more common than anticipated. In a descriptive, observational, single-center study of 256 health-care workers from Spain, anosmia/hyposmia, was reported in 68% and taste dysfunction in 70% of the cases [2]. In a cross-sectional, observational study involving 234 health care professionals with COVID-19 from Spain, smell/taste dysfunction was reported by 67.8% [3]. Re-

garding headache as a complication of SARS-CoV-2 infections, it has been reported by up to 60.7% among 234 Spanish health care professionals [3].

Concerning the two patients with polyneuritis cranialis, the VII, IX, and X cranial nerve (facial palsy, dysphagia) were regarded as affected in one patient and in the other patient the III, IX, and X cranial nerve (ptosis, dysphonia, dysphagia) [1], we should be told how dysphonia and dysphagia were unequivocally attributed to affection of cranial nerves and not to other causes.

Cerebro-spinal fluid (CSF) investigations are usually negative for SARS-CoV-2 RNA [4], except for an adult patient [5] and a pediatric patient [6]. We should be told if the presence of IgG against SARS-CoV-2 in patient 38 of table I (Helbok et al) was due to disruption of the blood brain barrier or if virus RNA could be detected in this patient as well.

In most studies about SAG a clear male preponderance has been found [4]. However, there is at best only mild or no male preponderance among COVID-19 patients [7]. Can this discrepancy be explained by the consideration that hormones play a role in the pathogenesis of SAG?

Extra-pulmonary manifestations of COVID-19 at onset are frequent and may initially obscure the viral infection [8]. Overlooking an asymptomatic SARS-CoV-2 infection or extra-pulmonary COVID-19 prior to onset of GBS may be responsible for erroneously classifying SAG as GBS due to other triggers and for the findings in some studies that the prevalence of GBS has not increased since the outbreak of the pandemic.

A point naturally not addressed in the review due to the publication date, is the question if SARS-CoV-2 vaccination influence the prevalence of SAG. Only few data about this issue have been published thus far. In a review about the frequency of publications about SAG before and after introduction of SARS-CoV-2 vaccination it turned out that the number of

publications amounted to 192 in the second half of 2020 and declined after introduction of SARS-CoV-2 vaccination in December 2020 to 75 publications in the first half of 2021 [Finsterer, in press]. Whether this decline in publications about SAG by 61% is due to a vaccination effect or due to other factors remains speculative. Anyhow, there are several arguments that favour an effect of the vaccinations.

Overall, the interesting review has some limitations which should be addressed before drawing final conclusions. Neurological compromise in COVID-19 and particularly SAG is more frequent than anticipated. Early recognition of SAG may result in earlier access to treatment and thus better outcome.

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