The spectrum of SARS-CoV-2 associated polyradiculitis is broad

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Letter to the Editor

With interest we read the article by Li et al. about 44 patients with SARS-CoV-2 associated Guillain-Barre syndrome (GBS) collected until 26th June 2016.[1] The review is appealing but has a number of limitations.

The number of patients with SARS-CoV-2 associated GBS is much higher than provided in the present review. Until August 8th 62 patients had been reported and as per the end of December 2020 220 patients with SARS-CoV-2 associated GBS had been published.[2]. Since then further patients accumulated in the literature.

The latency between onset of COVID-19 and the onset of GBS is much longer than given in the present review. In the review about 220 patients with SARS-CoV-2 associated GBS the latency ranged between -10 and +90 days,[2] suggesting that the immune response against SARS-CoV-2 is maintained for a much longer period than anticipated in the present work.

The classification of GBS subtypes in table-1 is unclear. The difference between the subtypes "demyelinating" and "acute, inflammatory, demyelinating polyneuropathy (AIDP)" should be explained. Patients with the "demyelinating" subtype should be combined with those of the "AIDP" subtype. Patients with the "sensory-motor axonal" subtype should be combined with those of the "acute, motor and sensory, axonal neuropathy (AMSAN)" subtype. For the subtype "acute motor axonal (AMA)" GBS the term "acute, motor axonal neuropathy (AMAN)" should be used.

Not only subtypes AIDP, AMAN, AMSAN, and Miller-Fisher syndrome (MFS) may develop after a SARS-CoV-2 infection but also the pharyngeal, cervical, brachial (PCB) variant and the polyneuritis cranialis (PNC) variant.[2].

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2 Finsterer J, Scorza FA. Guillain-Barre syndrome in 220 patients with COVID-19. Egypt J Neurol Psychiatr Neurosurg. 2021;57(1):55. doi: 10.1186/s41983-021-00310-7.