Letter to the Editor

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Neuro-COVID in the young

Josef Finsterer [1*], Fulvio A Scorza [2], Antonio-Carlos G Almeida [3]
[1] PhD, MD. Neurology & Neurophysiology Center, Vienna, Austria
[2] MD. Disciplina de Neurociência. Universidade Federal de São Paulo/Escola Paulista de Medicina (UNIFESP/EPM). São Paulo, Brasil
[3] MD. Centro de Neurociências e Saúde da Mulher Professor Geraldo Rodrigues de Lima. Escola Paulista de Medicina/Universidade Federal de São Paulo (EPM/UNIFESP). São Paulo, Brasil

* Corresponding Author: **Josef Finsterer**

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Introduction

We read with interest the article by Saini *et al*. on a study of 18 pediatric patients with neurological involvement in COVID-19 collected at a tertiary hospital in India [1]. During an observational period of five months, six COVID-19 patients with epilepsy, 5 with Guillain Barre syndrome (GBS), three with demyelinating central nervous system (CNS) disorders, two with cerebrovascular disease, and two with autoimmune encephalitis were diagnosed [1]. It was concluded that steroids could be beneficial while treating patients with neurological involvement in COVID-19, particularly if a patient presents with high inflammatory markers [1]. The study is promising but raises concerns that should be discussed.

We disagree with the statement that there is molecular mimicry between SARS-CoV-2 components and ganglioside or myelin oligodendrocyte glycoprotein (MOG) [1]. In fact, there is no connection between epitopes of SARS-CoV-2, particularly the spike protein, und surface markers of the myelon sheaths or axolemma [2].

We also disagree with the notion that post-COVID demyelination can be attributed to the prothrombotic state [1]. Demyelination in COVID-19 usually does not originate from thrombosis.

Furthermore, we disagree with the statement that active T-lymphocytes react with host antigens [1]. In fact, cytokines and chemokines produced by CD4+ T-cells react with host antigens [3].

There are some open questions. Unusually, one patient with GBS had seizures [1]. Readers should know if this patient had seizures prior to COVID-19. One patient with GBS was positive for antibodies to Borrelia burgdorferi [1]. How was Bannwarth syndrome ruled out in this patient? Only one patient with autoimmune encephalitis had an abnormal MRI, and one was positive for antibodies to NMDAR [1]. How was the patient diagnosed with autoimmune encephalitis with normal cerebral imaging? How can the authors be sure that reversible cerebral vasoconstriction syndrome (RCVS) and posterior, reversible encephalopathy

syndrome (PRES) are due to autonomic dysfunction [1] Meanwhile, several hundred patients with SARS-CoV-2 related GBS were reported, several of whom had autonomic dysfunction. However, RCVS and PRES were extremely rare, suggesting that autonomic dysfunction is not responsible for PRES and RCVS. Patient-12 and one GBS patient had seizures [1]. Why were these two patients not included in the epilepsy patient group?

There are several discrepancies. According to table 1, one GBS patient had seizures [1] but in table 2 none of the GBS patients had seizures. According to table 2, CSF investigations were not carried out in patient-10 [1]. However, one line below it is mentioned that the patient had positive oligoclonal bands (OCB) in the CSF [1]. These discrepancies require clarification.

Seizures in COVID-19 patients are usually due to a structural lesion of the brain [4]. Readers should know the results of cerebral MRI in the six patients with seizures.

Another limitation of the study is that no definition for the term "post-COVID" was provided [1]. Cut-offs for the latency between onset of COVID-19 and onset of GBS should be provided.

Overall, the interesting study has limitations that call the results and their interpretation into question. Clarifying these weaknesses would strengthen the conclusions and could improve the study. Neuro-COVID is an increasingly recognised manifestation of SARS-CoV-2 in pediatric and adult patients that requires extensive work-up to optimise diagnostic and therapeutic strategies.

Declarations
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Disclosures

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