Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

side. He was empirically treated with a course of 5 days intravenous high-dose methylprednisolone, without any consistent effect. Because of the worsening of tremor in his right extremities, in a follow-up visit on June 29, biperiden was added at a dose of 2 mg daily, and increased to 4 mg daily after 1 week, which resulted in improvement of the tremor.

The mechanism that led to the presumed degeneration of nigrostriatal dopaminergic nerve terminals is unclear. Perhaps a susceptible genetic makeup made our patient vulnerable to immunologically mediated mitochondrial injury and neuronal oxidative stress. Another hypothesis could be that the virus causes inflammation via microglial activation, contributing to protein aggregation and neurodegeneration.[3] However, the short time interval between the acute infection and the parkinsonian symptoms makes this hypothesis unlikely. Other researchers have proposed the so-called multiple hit hypothesis, by which the combination of toxic stress and an inhibition of neuroprotective responses can lead to neuronal death.[4]

Parkinson's disease is often preceded by anosmia, which is a common feature of SARS-CoV-2 infection.[5] Immune activation in the olfactory system might eventually lead to the misfolding of α-synuclein and the development of Parkinson's disease.[6] This mechanism is supported by post-mortem studies, showing increased levels of TNF,[7] IL1, and IL6.[8] Moreover, patients with Parkinson's disease had an elevated CSF antibody response to seasonal coronaviruses, compared with age-matched healthy controls.[9]

In Ashkenazi-Jewish people with Parkinson's disease, about a third are carriers of either a *GBA* or a *LRRK2* mutation.[10] A genetic analysis for these mutations and 62 other mutations associated with the disease was negative and our patient had no previous family history of Parkinson's disease. However, we cannot exclude an interaction between other, less frequent mutations and SARS-CoV-2. The temporal association between the episode of SARS-CoV-2 infection and parkinsonian symptoms, which appeared during the acute infection, is intriguing. Before his admission to the Department of Neurology, the patient had tested negative for SARS-CoV-2 on real-time RT-PCR on two occasions; however, he was then found positive for anti-SARS-CoV-2 IgG antibodies in serum, but negative for these antibodies in CSF. Nonetheless, we cannot exclude the possibility that SARS-CoV-2 entered the CNS, particularly in view of the olfactory involvement and borderline pleocytosis.

We declare no competing interests.

Mikhal E Cohen, Roni Eichel, Bettina Steiner-Birmanns, Amir Janah, Maxim Ioshpa, Rachel Bar-Shalom, Jefri J Paul, Hanaa Gaber, Volha Skrahina, Natan M Bornstein, Gilad Yahalom

mikhalc@szmc.org.il

Department of Neurology (MEC, RE, BS-B, AJ, MI, NMB, GY) and Department of Nuclear Medicine (RB-S), Shaare Zedek Medical Center, Jerusalem, Israel; Faculty of Medicine, The Hebrew University of Jerusalem, Jerusalem, Israel (MEC, RE, BS-B, AJ, MI, NMB, GY, RB-S); and CENTOGENE, Rostock, Germany (JJP, HG, VS)

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NeuroCOVID: it's time to join forces globally

Since the recognition of the severe acute respiratory syndrome coronavirus 2 outbreak in December, 2019, there are now over 22·1 million COVID-19 cases worldwide, with more than 780 220 deaths. Reports of neurological manifestations associated with COVID-19 range from mild (headache, hyposmia, ageusia, myalgia, and fatigue or sleepiness) to severe (encephalopathy, ischemic and haemorrhagic strokes, seizures, hypoxic-ischaemic brain injury, and Guillain-Barré and other autoimmune syndromes),[1–3] with prevalence rates ranging from 6% to 84%.[1–3] The true prevalence, underlying mechanisms (infectious, autoimmune, secondary to systemic complications), and outcomes of COVID-19 neurological manifestations remain a key knowledge gap.

Many global initiatives have emerged to address these critical questions.[4] The rapid and parallel implementation of these initiatives in a pandemic has resulted in discrepant data elements and definitions of neurological symptoms and signs. Furthermore, fragmented scientific approaches and overlapping consortia, in which centres can contribute data to multiple registries, raise the possibility of double-counting in future meta-analysis. All of these factors threaten the scientific rigour and yield of these combined global efforts. To address this issue, the European Academy of Neurology (EAN) and the Neurocritical Care Society (NCS)-endorsed Global Consortium Studies of Neurological Dysfunction in

COVID-19 (GCS-NeuroCOVID) established a formal collaboration, thus forming the largest global network to date. An important research priority is to develop consensus and harmonisation of data elements with uniform definitions, which was emphasised in a recent Editorial in *The Lancet Neurology*.[4]

The design and principals of the GCS-NeuroCOVID consortium studies were previously reported.[5] The GCS-NeuroCOVID group, in close partnership with the Pediatrics Neurocritical Care Research Group, formed and rapidly developed a paediatrics arm of the consortium to investigate the effects of COVID-19 in children and adolescents. Currently, the GCS-NeuroCOVID consortium includes 123 sites registered for adults and 96 sites registered for paediatrics across all continents (appendix).

On the **Global Consortium Study of Neurological Dysfunction in COVID-19** see https://www.neurocriticalcare.org/research/covid-19-research-opportunities

See **Online** for appendix

In parallel, the EAN created a prospective registry (The EAN NeuroCOVID Registry Consortium [ENERGY]) to evaluate the prevalence of neurological manifestations in confirmed COVID-19 cases and their outcomes at 6 months and 12 months. So far, over 254 sites have registered to ENERGY from 69 countries and three continents. This initiative was preceded by a survey of 2343 clinicians on neurological manifestations, completed on April 27, 2020, by the EAN-core COVID-19 task force.[1]

See **Online** for appendix

On the **EAN Neuro-COVID Registry Consortium** see https://forms.gle/hS6zFCV3e6vXkCjJ8

Together, this new global collaborative effort has extensive global outreach, with 473 sites representing all continents (appendix). In addition to global data elements and the harmonisation of definitions, this collaborative brings together complementary neurological expertise from acute resuscitation and critical care to outpatient clinic and rehabilitation settings, encompassing all ages of the population. This strong global collaborative infrastructure will serve as a crucial framework for current and future pandemics that threaten global neurological health.

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**Raimund Helbok, Sherry Hsiang-Yi Chou, Ettore Beghi, Shraddha Mainali, Jennifer Frontera, Courtney Robertson, Ericka Fink, Michelle Schober, Elena Moro, Molly McNett, *Claudio L Bassetti, on behalf of the GCS-NeuroCOVID consortium and the EAN COVID task force*

raimund.helbok@i-med.ac.at

Department of Neurology, Medical University of Innsbruck, Innsbruck 6020, Austria (RH); University of Pittsburgh School of Medicine, Pittsburgh, PA, USA (SH-YC, EF); Department of Neuroscience, Istituto di Ricerche Farmacologiche Mario Negri IRCCS, Milan, Italy (EB); Department of Neurology, College of Medicine (SM), and College of Nursing (MM), The Ohio State University, Columbus, OH, USA; New York University Grossman School of Medicine, New York, NY, USA (JF); The Johns Hopkins University School of Medicine, Baltimore, MD, USA (CR); University of Utah School of Medicine, Salt Lake City, UT, USA (MS); Division of Neurology, Centre Hospitalier Universitaire de Grenoble, Grenoble Alpes University, Grenoble, France (EM); and Department of Neurology, University of Bern, Inselspital, Bern, Switzerland (CLB)

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Lessons from a neurology consult service for patients with COVID-19

In March, 2020, the USA watched anxiously as the number of COVID-19 cases rose throughout the country. Yale New Haven Hospital, a tertiary care centre in Connecticut that is less than 80 miles east of New York city, swiftly planned for a large surge in patient numbers. Preliminary reports described frequent and varied neurological complications of COVID-19 (appendix p 1).[1,2] In anticipation, the Yale New Haven Hospital neurology department created a subspecialty consult service led by a neuroinfectious disease specialist, to diagnose and treat an expected wave of patients with novel neurological issues. Although neurologists worldwide have participated in the care of patients with COVID-19 on medical wards and in intensive care units, to our knowledge very few health systems have created services solely to manage the neurological complications of COVID-19.

From April 6 to May 29, 2020, the Yale NeuroCOVID team reviewed 100 cases, unburdening the primary neurology consult service, identified trends in disease presentations and demographics, and provided diagnostic and treatment recommendations based on emerging scientific literature (appendix p 2). In reviewing our NeuroCOVID patients, it was striking to observe that 25 (25%) were Hispanic and 25 (25%) were Black, while Connecticut is demographically 17% Hispanic and 12% Black.[3] This disproportionate representation of non-white patients requiring NeuroCOVID consultation parallels racial and ethnic disparities in COVID-19 disease presentations noted in the USA and Europe.[4] Hispanic NeuroCOVID patients were younger (median age 55 years, IQR 45–68) and had lower in-hospital mortality (16%) compared with white or Black patients (median ages 72 years, IQR 64–81 and