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LETTER TO THE EDITOR
EDİTÖRE MEKTUP

# Response to *SARS-CoV-2 associated Guillain-Barre syndrome after awaking on the ICU: Consider differentials*

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To the Editor,

Likewise, we would like to re-emphasize that, the patients work-up involved a detailed history, laboratory and other essential studies which have been summarized in the paper. During admission the patient did not have any abnormal neurological findings. His intubation was due to development of severe acute respiratory distress syndrome. His medical history included only chronic obstructive pulmonary disease and ischemic heart disease, for which he had been hospitalized several times. He did not have diabetes, renal insufficiency nor any malignancies diagnosed during these hospitalizations. His serum vitamin B12 and serum 25OH vitamin D levels were within normal levels. He was not on any drugs that may have caused neuropathy. To clarify the course of disease, he was admitted to the emergency department with symptoms that had been present for the past

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few days, and was admitted to the intensive care unit (ICU) and intubated on the forth day of his hospital admission. Neurological compromise could only be detected several days after his sedatives were weaned, into the third week of his ICU stay. So the definite time for start of neurological compromise cannot be defined clearly, but was noted about a month after onset of infection.

Regarding the authors' claim, we want to emphasize that we explained exclusion of critically ill neuropathy or critically ill myopathy as the main differential diagnoses of tetraplegia sufficiently in the paper. (i) We outlined the process of exclusion in the Discussion as follows: "Generalized weakness in the ICU should prompt primarily the diagnosis of critical illness neuropathy and myopathy (CINM). CINM can be a challenging diagnosis in the ICU, even if it causes more proximal than distal weakness, and its clinical differentiation from Guillain Barré syndrome (GBS) can be difficult. However, electrophysiologic findings clearly presented a demyelinating polyradiculoneuropathy in this patient"(1). (ii) We strongly recommend the authors to look in detail at the referenced letter about lopinavir. Oddly, the referenced letter mentioned a case with HIV-AIDS who developed a distal symmetric neuropathy at the 6th month of didanosine, stavudine, and nevirapine treatment which has abated after cessation of former agents and replacement with lopinavir/ritonavir and saquinavir (2). However, it is told without convincing evidence that after three years of exposure to the new regimen including lopinavir/ritonavir the distal symmetric neuropathy re-occurred. Another shady aspect of this case is the nonexistence of detailed electromyography (EMG) findings. Nonetheless, our case has only a few weeks of exposure of lopinavir/ritonavir, not the months or years of exposure as in the referenced case. The referenced case report on neuropathy associated with hydroxychloroquine again has similar chronicity of exposure, one of the patients had four years of exposure and the other has nine months of exposure (3). Therefore, both lopinavir/ritonavir and hydroxychloroquine-associated neuropathy do not represent acute-subacute symptoms of our case in terms of duration of exposure. (iii) In addition, the constellation of albumin-cytological dissociation in cerebrospinal fluid (CSF), increased distal latencies, and temporal dispersion in the motor nerve conduction study unequivocally distinguish AIDP from AMSAN (4). Especially, temporal dispersion in compound muscle action potential is not only an unexpected but also a contradictory finding in any axonal subtype of GBS (4). (iv) The authors claimed that Bickerstaff brainstem encephalitis and immune-mediated encephalitis should be ruled out with MRI. However, Bickerstaff brainstem encephalitis is seen together with the Miller-Fisher variant of GBS and it has a classical triad of ophthalmoplegia, ataxia, and areflexia (5). Our patient has no ophthalmoplegia, therefore we think it is unreasonable to perform an magnetic resonance imaging (MRI) for that differential diagnosis. Also, there is no mild pleocytosis in CSF to hint at encephalitis.

Actually, the effect of IVIG was partially beneficial, and by this the patient had been extubated and his motor functions were getting better. He was hemodynamically stabile. His daily troponin levels were not on the rise. We were planning to discharge him from the ICU to the wards when a sudden deterioration (within minutes) occurred. Therefore, myocarditis or Takotsubo syndrome were not considered. However, since he was known to have an advanced stage ischemic heart disease, most probable cause was considered to be a cardiac event. Other differentials cannot be ruled out for certain. An autopsy would be revealing, but given the conditions of a new onset pandemic, it was not plausible.

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