Increased Cases of Acute Polyneuropathy in COVID-19 Pandemic; What Awaits Neurologists

Mehmet Sinan Özeren*

Department of Mathematics and Statistics, Istanbul Technical University, İstanbul, Turkey

***Corresponding author:**

Blood tests also revealed lymphopenia and thrombocytopenia while 2 patients had normal lymphocyte and thrombocyte counts (Table 1)

Age/ sex	Days between COVID-19 symptoms and GBS onset	Neurological examainat ion	Respirato ycomlints	GBS symptoms	Electroph ysiol- ogy: Neuropath y type and GBS elec-trophysiol ogic subtype
1.75/M	15 days after	proximal and distal lower limb weakness, hypoactive deep tendon refexes in upper limb and absent in lower limb	Yes (concurrent pneumonia)	Progressio n of limb weakness and inability to walk Loss of ambulation ,	Mixed demyelinati ng and axonal Predomina ntly AMAN
2.74/FM	17 days after	Hypoaesth esia ,parap aresis paraesthes ia in the lower limb		Hypoaesth esia, weakness paraesthes ia in the lower limb	Motor sensory axonal AMSAN
3.80/FM	19 days after	Paraparesi s, pain in the lower limb and hyporefexi a at the lower limb	no	Lower limb weakness, and difficulty walking	Axonal AMAN
4.80/FM	12 days after	Paraparesi s,paresthe sia pain and arerefexia at the lower limb/ hyporeflexi a inthe upper limb	yes	progressiv e ascending paraesthes ia of distal lower limb,loss of ambulation	Motor sensory axonal AMSAN
5.70/FM	16 days after	Paraparesi s, and arerefexia at the lower limb	no	Flaccid paraparesi s, arefexia a tlower limb and loss of ambulation	Axonal AMAN axonal,
6.83/FM	16 days after	Lower limb weakness, hypoactive deep tendon refexes in Uupper limb and absentppein and			

SARS-CoV-2 is capable of causing an excessive immune reaction with an increased level of cytokines as Interleukin-6 (IL-6), which are produced by activated leukocytes and stimulate the inflammatory cascade leading to extensive tissue damage. IL-6 plays an important role in multiple organ dysfunctions, which is often fatal for patients with COVID-19.

In the literature, although polyneuropathy was found to be more common in men (50 vs. 23 cases: 68.5% vs. 31.5%), we had only one male patient in our series of ten cases. On the basis of this observational series involving ten patients, it is not possible to determine whether severe deficits and axonal involvement are typical features of COVID-19–associated acute polyneuropathy. However, since March 2020, when the first COVID-19 cases were recorded in our country, we have found that the total number of acute polyneuropathy cases we have followed up in our clinic are increased, approximately three times more than the previous year. Thus, it is possible that acute polyneuropathy is linked to the COVID-19 infection. Common patient characteristics in this series beside COVID-19 were increased ferritin and anemia. Ferritin is a key mediator of immune dysregulation, especially under extreme hyperserotonemia, via direct immune-suppressive and pro-inflammatory effects, contributing to the cytokine storm and it is known that they face a higher probability to experience serious complications from COVID-19. Laboratory findings in patients with severe COVID-19 showed data consistent with cytokine storm involving elevated inflammatory markers, including ferritin, which has been associated with critical and life-threatening illness. The height of ferritin detected during the COVID period also requires more caution in terms of polyneuropathy that may develop, however, concomitant iron deficiency might play a role in the etiology of polyneuropathy. When the literature is examined, we know that acute polyneuropathy is not observed in every case of COVID-19 with iron deficiency anemia. Thus, the association between COVID-19, iron deficiency anemia and acute polyneuropathy is obscure.

Conclusion

We add to the literature 10 cases of GBS related to COVID-19 infection supporting SARS-Cov-2 virus could be a triggering factor of GBS. Studies assisted by histopathological evidence could show us the fate of patients with axonal neuropathy, which occurs acutely but can be reflected in the chronic period [5]. However, more cases with epidemiological data should be studied and future investigations should be carried out in this regard. Awareness of possible causal association between acute polyneuropathy and COVID19, recommend long term follow up of COVID 19 patients for neurologic complications. Finally, it is recognized that research on the relationship between COVID-19 and the nervous system surely would not be limited to the current period but would also serve basis for providing knowledge and treatment for future pandemics.

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