Journal of Experimental and Clinical Medicine
https://dergipark.org.tr/omujecm

Case Report

J Exp Clin Med
2022; 39(1): 277-279
doi: 10.52142/omujecm.39.1.52

Acute motor axonal neuropathy associated with COVID-19

Mehlika PANPALLI ATEŞ *, **Beyza Nur ÇETİN**, **Beria Nur ERTUĞRUL**, **Ahmet Enes YÜKSEKTEPE**, **Selim Selçuk ÇOMOĞLU**

Department of Neurology, University of Health Sciences, Diskapi Yildirim Beyazit Training and Research Hospital, Ankara, Turkey

Received: 10.05.2021 • **Accepted/Published Online:** 12.06.2021 • **Final Version:** 01.01.2022

Abstract

COVID-19, which is considered a global pandemic, is constantly being renewed. Acute motor axonal neuropathy (AMAN) is a rare, axonal variant of Guillain-Barré syndrome (GBS). This article presents a 69-year-old female patient diagnosed with AMAN due to a COVID-19 infection. Sixty-nine-year-old female patient who started myalgia, headache, cough 14 days ago, and was added diarrhea, fever, loss of smell and taste. Nasopharyngeal SARS-CoV-2 polymerase chain reaction (PCR) was found to be positive. Ten days later, numbness and weakness developed in the distal extremities. As a result of the patient's neurological examination, acute motor axonal neuropathy (AMAN) was diagnosed.

Keywords: acute motor axonal neuropathy, COVID-19, guillain-Barré syndrome, pandemic, SARS-CoV2

1. Introduction

This virus is called SARS-CoV-2 and is named by World Health Organization as Coronavirus Disease 2019; COVID-19 (1, 2).

Both SARS and COVID-19 bind to the angiotensin-converting enzyme-2 receptors (3). This receptor is situated in the cell membrane of several organs; including the lung, kidney, liver, skeletal muscle and nervous system (4).

The SARS-CoV-2 infection has been reported to have neurological complications such as febrile seizures, headache, dizziness, confusion, myalgia, loss of taste and smell, encephalitis, stroke, and acute peripheral nerve disorders (4-6).

Coronavirus starts as a nasal infection, and it enters the central nervous system through the olfactory bulb. It causes inflammation and demyelination, which may lead to temporary loss of smell and taste in patients without typical symptoms (6).

GBS is an inflammatory polyradiculoneuropathy characterized by acute, ascending motor weakness, or cranial nerve weakness, loss of deep tendon reflexes, mild sensory abnormalities, and dysautonomic symptoms, accompanied by muscle or radicular pain (7). AMAN is a very rare form of symmetric neuropathy. GBS, which affects the motor fibers, and is characterized by axonal degeneration. Two-thirds of GBS associated with a viral infection 1-3 weeks before. Diarrhea due to Campylobacter jejuni enteritis is usually observed in the prodromal period (8).

Here, we report the AMAN case related to COVID-19, which is a rare subtype of a GBS.

2. Case Report

A sixty-nine-year-old female patient, who started to have weakness and fatigue 14 days ago. With the addition of myalgia, headache, cough, diarrhea, high fever, inability to taste and smell, she underwent nasopharyngeal SARS-CoV-2 polymerase chain reaction (PCR), and it was found to be positive. Ten days after the PCR test, she came to the emergency department due to the numbness in the distal extremities and the subsequent ascending weakness.

The patient had a history of hypothyroidism, diabetes mellitus, hypertension.

The neurological examination of the patient's bilateral upper and lower extremities were 2/5 in the proximals, 3/5 in the distals. The deep tendon reflexes were globally abolic. The patient's clinic was consistent with progressive flaccid

The laboratory tests results were follows: serum glucose 147 mg/dL; blood urea nitrogen: 35 mg/dL; creatinine 0.5 mg/dL; alanine aminotransferase 18.8 U/L; aspartate aminotransferase 16.9 U/L; sodium 138 mEq/L; potassium 3.56 mEq/L; white blood cell count 15600/µL (neutrophils=81.8%; lymphocytes=11.3%); Erythrocyte sedimentation rate 42 mm/hour, C-reactive protein 24.9 mg/L, hemoglobin 12.7 g/dL. The patient's Campylobacter jejuni test was negative.

Lumbar puncture was performed on the patient. Cerebrospinal fluid (CSF) proteins=150 mg/dL (normal=0–40 mg/dL), white blood cells=0×106/L (normal=0–8×106/L). Microbiologic testing on CSF was negative (including HSV,

* Correspondence: muefhulkika@gmail.com

VZV, EBV, CMV, HIV-1, Borrelia Burgdorferi IgM/IgG). CSF analysis demonstrated albuminocytological dissociation.

In the first electroneuromyography evaluation of the patient's (complaints on the 2nd day) is; median, peroneal, posterior tibial nerve compound muscle action potential (CMAP) could not be obtained, ulnar nerve CMAP amplitudes were reduced, ulnar nerve F response was prolonged, other F responses could not be obtained. All sensory nerve conduction studies were normal. No resting activity was observed in the needle examination.

Neurophysiologic findings at 2 days, and 7 days after neurological symptom onset were consistent with subtype of GBS; AMAN (Table 1).

Lung computed tomography showed diffused consolidations, ground-glass opacities in both lungs, and bilateral pleural effusion (Fig. 1).

The patient was hospitalized, intravenous immunoglobulin (0,4 g/kg per day during five days) was started. In addition; Favipiravir (in first day 2*1600mg, and next 4 days 2*600mg), low-molecular-weight heparin (LMWH) (enoxaparin sodium), intermittent O_2 (2L/min) were given. The patient's weakness improve on the 3rd day of treatmen

Table 1. Patient characteristics and results

Motor Nerve Conduction Study		First ENMG (2 days later)			Second ENMG (9 days later)		
Nerve (left)		Wrist	Elbow		Wrist	Elbow	
Median	Latency (ms)	PY			PY		
	NCV (m/s)						
	Amplitude (μV)						
	F Response Latency (ms)		PY			PY	
		Wrist	Bellow elbow	Above the elbow	Wrist	Below elbow	Above the elbow
Ulnar	Latency (ms)	3.5	7.04	8.42	3.42	7,02	8.36
	NVC (m/s)		53.7	50.7		50	41
	Amplitude (μV)	1790	1090	1150	2360	2250	2120
	F Response Latency (ms)		36.9			35.25	
		Ankle	Head of Fibula	Popliteal	Ankle	Head of Fibula	Popliteal
Peroneal	Latency (ms)	NP			NP		
	NCV (m/s)						
	Amplitude (μV)						
	F Response Latency (ms)		NP			NP	
		Ankle	Popliteal		Ankle	Poplitea l	
Posterior Tibial	Latency (ms) NP	NP			NP		
	NCV (m/s)						
	Amplitude (μV)						
	F Response Latency (ms)		NP			NP	

NP: No Potential *: All of sensory nerve studies and needle ENMG studies were normal

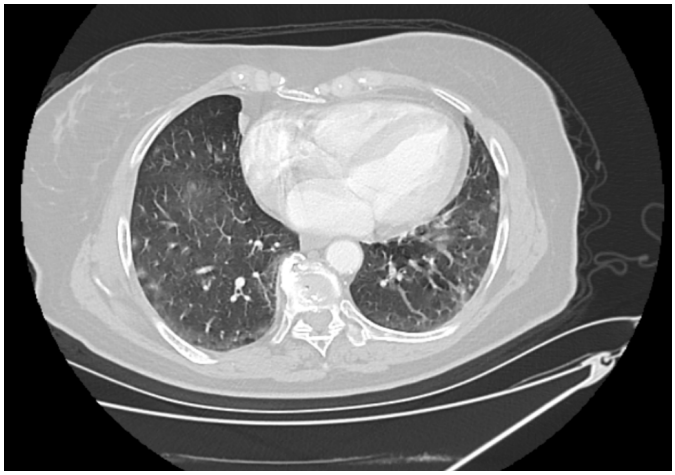

Fig. 1. Lung computed tomography showed diffused consolidations and ground-glass opacities in both lungs, and bilateral pleural effusion

3. Discussion

There are studies suggesting that the neuroinvasive potential of COVID-19 (4, 5). The neurological symptoms of COVID-19 infection are due to their effects on the central nervous system (headache, dizziness, changes in consciousness, acute brain disorder, seizure, and ataxia), and the peripheral nervous system (anosmia, ageusia, and visual impairment) (2, 4). There are also reports of a relationship between GBS and Coronavirus infections (2, 9).

COVID-19 has an incubation period of approximately 5.2 days. The interval between the onset of COVID-19 symptoms and the first symptoms of GBS ranged from 5 to 10 days. The average 10-day interval between the onset of viral disease and the first symptoms of GBS is similar to the interval seen in GBS occurring other infections. Furthermore, demyelinating polyneuropathy has been widely observed in most of these reports (2, 9). The period between the first day of onset of COVID-19 symptoms, and the onset of GBS was 12 days and an axonal polyneuropathy developed in our patient.

According to a report compiling previous case reports, most of the reported patients were over 50 years of age and male, reflecting the underlying demographic characteristics

COVID-19. The mean age of the patients was 57.2±15.82, the youngest patient was 5, and the oldest patient was 84 years old (1, 2).

The need for mechanical ventilation was reported to be more pronounced compared to patients with GBS without COVID-19 (11). Our patient was closely followed up with O_2 during her hospitalization, there was no need for mechanical ventilation. IVIg was preferred of our patient, and LMWH was administered simultaneously. We did not preferred plasma exchanges, because of the hemodynamic status of COVID-19 patients was unstable, and more healthcare professionals were exposed to the patient for longer-periods of time. Negative PCR analysis in CSF also means that there is no direct root infection or intrathecal viral replication, and supports a dysimmune response mechanism after infection (2, 10).

Although GBS has developed due to the follow-up of patients mostly in intensive care units, this may hinder them from being noticed. It should not be surprising that GBS does not come to mind immediately especially considering that these intensive care units are not neurological intensive care units.

It is difficult to diagnose, given that most of the GBS patients associated with COVID-19 do not have any symptoms of COVID-19 at the time of admission. In patients presenting with neurological diseases such as GBS, encephalomyelitis, myositis without systemic COVID-19 symptoms; the presence of anosmia/agusia/cranial neuropathy and lymphocytopenia/thrombocytopenia are red flags that increase the suspicion of early diagnosis for COVID-19 (6,10).

In patients with increased respiratory distress or in need of intensive care, this condition may not only be due to lung involvement but may also develop due to neurological diseases such as GBS. Early diagnosis and treatment of patients may provide treatment and follow-up without the need for intensive care units.

References

1. Caress JB, Castoro RJ, Simmons Z, Scelsa SN, Lewis RA, Ahlawat A, et al. COVID-19–associated Guillain-Barré syndrome: The early pandemic experience. Muscle & Nerve. 2020; 62: 485–491. doi: 10.1002/mus.27024.
2. Rahimi K. Guillain-Barre syndrome during COVID-19 pandemic: an overview of the reports. Neurol Sci. 2020; 41: 3149–3156 https://doi.org/10.1007/s10072-020-04693-y
3. Parsamanesh N, Pezeshgi A, Hemmati M, Jameshorani M, Saboory E. Neurological manifestations of coronavirus infections: role of angiotensin-converting enzyme 2 in COVID-19. Int J Neurosci. 2021:1-8. doi: 10.1080/00207454.2020.1849193.
4. Mao L, Jin H, Wang M, Hu Y, Chen S, He Q, et al. Neurologic Manifestations of Hospitalized Patients With Coronavirus Disease 2019 in Wuhan, China. JAMA Neurol. 2020; 77(6): 683-690. doi: 10.1001/jamaneurol.2020.1127.
5. Helms J, Kremer S, Merdji H, Clere-Jehl R, Schenck M, Kummerlen C, et al. Neurologic Features in Severe SARS-CoV-2 Infection. N Engl J Med. 2020; 382(23): 2268-2270. doi: 10.1056/NEJMc2008597.
6. Scheidl E, Canseco DD, Hadji-Naumov A, Bereznai B. Guillain-Barré syndrome during SARS-CoV-2 pandemic: A case report and review of recent literatüre. J Peripher Nerv Syst. 2020; 1-4. doi: 10.1111/jns.12382.
7. Ates MP, Dibek DM, Guven H, Comoglu SS. A Rare Complication of Bariatric Surgery; Guillain Barre Syndrome. J Clin Images. 2020; 3(1): 1051. doi: 10.33582/2690-8662/1051.
8. Panpalli Ates M, Guven H, Comoglu SS. Temporary conduction block in acute motor axonal neuropathy. Acta Neurol Belg. 2016; 116(3): 425-427. doi: 10.1007/s13760-015-0543-8.
9. Paybast S, Gorji R, Mavandadi S. Guillain-Barré Syndrome as a Neurological Complication of Novel COVID-19 Infection: A Case Report and Review of the Literature. Neurologist. 2020; 25(4): 101-103. doi: 10.1097/NRL.0000000000000291.
10. Dalakas MC. Guillain-Barré syndrome: The first documented COVID-19-triggered autoimmune neurologic disease: More to come with myositis in the offing. Neurol Neuroimmunol Neuroinflamm. 2020; 7(5): e781. doi:10.1212/NXI.0000000000000781