Case Report / Olgu Sunumu

COVID-19: A Threat of Chronic Inflammatory Demyelinating Polyneuropathy Attack
COVİD-19: Kronik İnflamatuar Demiyelinizan Polinöropati Atağı İçin Bir Tehdit'

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Abstract: Although COVID-19 has emerged as a disease affecting the respiratory system, increasing data show it is not limited to the respiratory system but also affects both the central and peripheral nervous systems. Since it affects many people and has been declared a pandemic, information about the course of the disease and the treatment plan during and/or after COVID-19 in patients with chronic neurological disease is becoming increasingly important. A 42-year-old male patient who was hospitalized in the infectious diseases service because of being COVID-19 positive was evaluated with difficulty breathing and swallowing and weakness in four extremities. On neurological examination, muscle strength was 3/5 in all four extremities according to the Medical Research Council (MRC) scale, and there was widespread hypoesthesia in all four extremities. Deep tendon reflexes were completely absent. The first complaints of our patient started six years ago, and the diagnosis of CIDP was made two years after the first complaint, with typical clinical features and electrophysiological findings. In this case report, we share a patient with COVID-19 and chronic inflammatory demyelinating polyneuropathy (CIDP), and we aim to report the clinical worsening of CIDP with COVID-19.
Keywords: Chronic Inflammatory Demyelinating Polyradiculoneuropathy, COVID-19

Özet: COVID-19 solunum sistemini etkileyen bir hastalık olarak ortaya çıkmış olsa da, artan veriler solunum sistemi ile sınırlı olmadığını, hem merkezi hem de periferik sinir sistemini de etkilediğini göstermektedir. Pandemi olarak ilan edilmesi ve birçok insanı etkilemesi nedeniyle, kronik nörolojik hastalığı olan hastalarda COVID-19 sırasında ve/veya sonrasında hastalığın seyri ve tedavi planı ile ilgili bilgiler giderek önem kazanmaktadır. COVID-19 pozitif olması nedeniyle enfeksiyon hastalıkları servisine yatırılan 42 yaşındaki erkek hasta, solunum ve yutma güçlüğü, dört ekstremitede güçsüzlük şikayeti ile değerlendirildi. Nörolojik muayenesinde dört taraflı kas gücü 3/5 ve dört ekstremitede yaygın hipoestezi saptandı. Derin tendon refleksleri genellikle yoktu. Hastamızın ilk şikayetleri 6 yıl önce başlamış, tipik klinik özellikleri, elektrofizyolojik bulguları sonucunda 2 yıl sonra CIDP tanısı konmuştu. Bu vaka raporunda, COVID-19 ve kronik inflamatuar demiyelinizan polinöropatili (KİDP) bir hastayı paylaşıyoruz ve COVID-19 ile KİDP'nin klinik kötüleşmesini bildirmeyi amaçladık.
Anahtar Kelimeler: CIDP, COVID-19, polinöropati atağı

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1. Introduction

Chronic inflammatory demyelinating polyneuropathy is the most common demyelinating polyneuropathy type. It causes symmetrical muscle involvement that lasts longer than 8 weeks, may progress with attacks, and is characterized primarily by diffuse sensory involvement. It is thought to be an autoimmune disease, but it has demonstrated no genetic predisposition (1). Steroids, IVIG, and plasma exchange are used in the treatment, and its most important feature is that it is a treatment-responsive neuropathy.

Although it is known that COVID-19 is a disease that affects the respiratory system primarily, it has been shown that central nervous system involvement is not uncommon in recent studies and reported cases. Demonstration of the presence of SARS-CoV-2 in the cerebrospinal fluid of patients diagnosed with COVID-19 also supports nervous system involvement (2). Also, our knowledge about the course of the disease and the treatment strategy increases day by day in patients with existing chronic neurological diseases after COVID, but it is still not sufficient.

After COVID-19, neurological complications such as acute cerebrovascular events, encephalitis, and Guillain-Barre syndrome (GBS) have been reported (3). Cases of COVID-19 with a diagnosis of myasthenia gravis have been reported. However, in the literature, we found only one case with a diagnosis of CIDP that worsened in terms of CIDP because of COVID-19 (4).

2. Case Report

The first complaint of our 42-year-old male patient started six years ago. Because of typical clinical features, electrophysiological findings, and albuminocytologic dissociation in the cerebrospinal fluid, he was diagnosed with CIDP 2 years after the first symptoms.

Steroid treatment was tried, but we could not achieve a significant response. Then, the patient was followed with intravenous immunoglobulin (IVIG) therapy every 21 days, and the active course of the disease regressed. During the COVID-19 pandemic, the patient's mother had respiratory symptoms, and the polymerase chain reaction (PCR) result for COVID-19 was positive. The PCR test was performed because the patient had contact with his mother. There was a ground-glass appearance in the lower zones of both lungs on thorax computed tomography (CT). In laboratory tests, hemogram, d-dimer, and fibrinogen were within normal limits at admission. Ferritin was slightly higher at 273 ng/ml (normal: 15–260). During the follow-up period, d-dimer increased to 2.11 mg/LL (normal: 0-0.55), and ferritin increased to 1213 ng/ml. CRP 37.5 mg/LL (normal: 0-5), procalcitonin 0.16 (<0.5: bacterial infection other than systemic infection or viral infection), and CKMB 2.4 ng/ml (normal: 0-5.2) were detected. The patient was consulted by the infectious diseases and clinical microbiology departments, and favipiravir, dexamethasone, and symptomatic therapy was started. Then the patient was evaluated for CIDP exacerbation because of difficulty breathing and swallowing and weakness in four extremities. His neurological examination revealed a four-sided muscle strength of 3/5 and diffuse hypoesthesia in all four extremities. It had been 18 days since the last IVIG dose.

Electromyography (EMG) performed approximately 1 month before the CIDP attack revealed sensorimotor polyneuropathy accompanied by common symmetrical demyelinating-type conduction blocks (Table 1). Post-attack CIDP EMG revealed temporal dispersion, conduction blocks in the peroneal and tibial nerves, and extended sensorimotor polyneuropathy (Table 1).

Hence, a total of 2 g/kg IVIG treatment was given to the patient for 5 days. Significant improvement was observed in the patient's clinic, and we discharged him on the 12th day after we completed the treatment process.

Table 1. Pre-attack and Post-attack CIDP nerve conduction study findings of the patient

Pre-attack CIDP nerve conduction study findings of the patient

Peripheral Nerve	Distal latency (m/sn)	Amplitude(M-mV/S-uV) Distal/Proximal	Nerve conduction velocity (m/sn)	F-M latency (ms)
Median motor right	3.67	8.2 / 6.9	52.5	28.5
Median motor left	3.54	7.8/ 7.1	51.2	26.7
Ulnar motor right	2.82	9.3/ 7.1	51.8	30.6
Peroneal motor right	13.1	2.2/ 2.2	33.9	NR
Peroneal motor left	10.5	2.7/ 2.4	34.2	NR
Tibial motor right	3.56	8.0/ 1.2	28.3	54.8
Median sensory right	NR	NR	NR	
Median sensory left	NR	NR	NR	
Ulnar sensory right	NR	NR	NR	
Radial sensory right	2.79	9.3	41.4	
Sural sensory right	3.16	2.3	42.9	
Sural sensory left	2.9	3.4	44.3	
Peroneus superfic sensory right	NR	NR	NR	
Post-attack CIDP nerve conduction study findings of the patient				
Median motor right	4.02	7.1/ 3.1	39.4	NR
Median motor right	3.67	7.0/ 3.8	39.8	NR
Ulnar motor right	3.8	7.8/ 6.2	47.5	NR
Peroneal motor right	17.5	0.83/ 0.46	25.6	NR
Peroneal motor left	13.6	1.6 / 1.1	29.7	NR
Tibial motor right	6.72	4.5/ 1.5	28.3	NR
Median sensory right	NR	NR	NR	
Median sensory left	NR	NR	NR	
Ulnar sensory right	NR	NR	NR	
Radial sensory right	NR	NR	NR	
Sural sensory right	NR	NR	NR	
Sural sensory left	NR	NR	NR	
Peroneus superfic sensory right	NR	NR	NR	

(No Response ; NR)

3. Discussion and Conclusion

Although the mechanism of peripheral nervous system involvement in COVID-19 is not fully understood, it is thought that it is primarily related to the immune system and that the virus has a direct cytotoxic effect on peripheral nerves in Guillain-Barré syndrome (GBS) (5). Another theory emphasizes molecular similarity mechanics.

Cases of GBS seen after COVID-19 are older, with a more severe clinical course and more common demyelinating neuropathy, unlike typical GBS (5). Although a case of myasthenia gravis developing after COVID-19 has not been reported, worsening after COVID-19 has been reported in patients diagnosed with existing myasthenia gravis (5).

A 69-year-old female patient with no known neurological disease in the literature was diagnosed with CIDP induced by COVID-19 (6). Apart from this, two patients with COVID-19 diagnosed with CIDP have been reported. A 69-year-old male patient presented with a fever and neurological clinical deterioration (7). On the other hand, a 53-year-old female patient was diagnosed with COVID-19 after she was admitted with only neurological deterioration without fever, respiratory, or gastroenterological complaints (8). In both patients, COVID-19 was negative in nasopharyngeal samples and positive in samples taken by bronchoscopy. It has been interpreted that inadequate coughing because of muscle weakness in GBS and CIDP patients may explain the lower viral load in the upper respiratory tract than in the lower respiratory tract (8). It showed clinical improvement in both patients with IVIG therapy. The diagnosis of COVID-19 after clinical worsening occurred in both patients and our patient suggests that COVID-19 is associated with neurological deterioration.

Clinical improvement with IVIG treatment given to patients for CIDP attacks also supports this hypothesis.

Although the data are insufficient to prove the relationship between COVID-19 and CIDP worsening at this stage, it suggests that care should be taken in terms of possible clinical worsening in cases of suspected and/or diagnosed COVID-19 in patients followed up with a diagnosis of immune-mediated chronic neuropathy. More case data and research are needed on this subject.

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Ethics