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Case Report

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Guillain Barre Syndrome After COVID-19 Infection

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ABSTRACT

Unexplained pneumonia cases caused by a new coronavirus known as SARS-COV-2, occurred in Wuhan, China since December 2019. Culprit of the worldwide pandemic of the coronavirus disease 2019 (COVID-19), SARS-CoV-2 attach to the angiotensin-converting enzyme 2 receptor, affecting multiple systems such as respiratory, vascular, renal and central nervous system. Most common neurological symptoms are headache, dizziness, nausea, vomiting, myalgia, anosmia, ageusia and altered consciousness; however, there are reports that suggests an association between Guillain-Barre syndrome (GBS) and COVID-19 infection. Guillain-Barre syndrome (GBS) is an autoimmune polyradiculoneuropathy that characterized with progressive weakness of the limbs and reduction or loss of tendon reflexes. Protein concentrations of cerebrospinal fluid (CSF) are increased and the white cell count is normal. Plasma exchange and intravenous immunoglobulin (IVIG) used in treatment with supportive care and most of patients have full recovery. In this paper we aimed to present a patient with positive real time polymerase chain reaction (RT- PCR) test presenting to the emergency department (ED) with the symptoms of GBS.

Keywords: Guillain-barre, COVID-19, COVID

ÖZ

COVID-19 Enfeksiyonu sonrası Gullian Barre Sendromu

Aralık 2019 tarihinden itibaren, Çin'in Wuhan kentinden ortaya çıkan ve tüm dünyayı kısa sürede etkisi altına alan SARS-COV-2 olarak adlandırılan bir yeni koronavirüsün sebep olduğu nedeni açıklanamayan pnömoni vakaları ortaya çıktı. Koronavirüs hastalığı 2019 (COVID-19) pandemisinin sorumlusu SARS-CoV-2 anjiotensin dönüştürücü enzim 2 reseptörüne bağlanıp solunum, vasküler, renal ve merkezi sinir sistemi gibi çoklu sistemleri etkiler. En yaygın nörolojik bulgular baş ağrısı, baş dönmesi, bulantı, kusma, kas ağrısı, koku alamama, tat alamama ve bilincin değişken durumları olmakla birlikte COVID-19 enfeksiyonu ve Guillain-Barre Sebdromu (GBS) arasında bir ilişki kuran çalışmalar da mevcuttur. Guillain-Barre sendromu (GBS) uzuvların progresif zayıflığı ve tendon reflekslerinde azalma veya kayıp ile karakterize otoimmün poliradikülonöropatidir. Beyin-omurilik sıvısı (BOS) protein konsantrasyonları artmış ve beyaz küre sayısı normaldir. Tedavide destekleyici bakıma ek olarak plazma değişimi ve intravenöz immünglobulin (IVIG) kullanılır ve hastaların çoğu tamamen iyileşir. Bu çalışmanın amacı gerçek zamanlı polimeraz zincir reaksiyonu (RT-PCR) testi pozitif olan ve acil servise GBS bulguları ile başvuran bir hastayı sunmaktır.

Anahtar Kelimeler: Guillain-barre, COVID-19, COVID

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INTRODUCTION

Unexplained pneumonia cases caused by a new coronavirus occurred in Wuhan, China in December 2019 (1). This virus, which was similar to SARS-CoV, was called SARS-CoV-2. World Health Organisation (WHO) named it as the coronavirus disease 2019 (COVID-19) (2,3). SARS-CoV and SARS-CoV-2 both attach to the angiotensin-con-

verting enzyme 2 receptor (4). It affects multiple systems as respiratory, vascular, renal and central nervous system (5,6). Guillain-Barre syndrome (GBS) is an autoimmune polyradiculoneuropathy characterized with progressive weakness of the limbs and reduction or loss of tendon reflexes (7). Protein concentrations of cerebrospinal fluid (CSF) are increased, and the white cell count is normal (8,9). Plasma exchange and intravenous immunoglobulin (IVIG) are used in treatment with supportive care, and most of the patients have full recovery (7,10,11). There are reports that suggest an association between GBS and COVID-19 infection (12).

CASE

We aimed to report an 81-year-old female patient presented to the ED with the complaint of progressive symmetric weakness on the lower limb for two days. She had a real time polymerase chain reaction (PCR) test which was reported positive for COVID-19 infection two weeks ago, and her treatment was arranged with favipiravir and paracetamol. At the time of admission, the patient had no complaints or symptoms with the respiratory system. Her bladder and bowel functions were normal. On physical examination, her vital signs were all normal, the weakness was symmetrical and muscle power was diminished (1/5) in lower limbs, deep tendon reflexes were absent, and the Babinski sign was reckless on both sides. Fine touch sensation was normal. Meningeal irritation signs were negative. Patient had coronary artery disease with 60% ejection fraction in her past medical history and used metoprolol and clopidogrel daily. She had no prior clinically relevant background. It was planned to investigate spinal cord diseases, paraneoplasia and infectious diseases in the patient. Laboratory results are summarized in Table 1. According to the tests performed, the patient had no linked infection that might be associated with GBS except COVID-19. Cervical, thoracic and lumbar vertebra computed tomography showed a normal finding except for mild herniation of two intervertebral discs and degeneration. Medulla spinalis and brain magnetic resonance imaging (MRI) was done and showed age-matched brain atrophy and no elucidatory spinal finding. Abdominal ultrasound was normal. Lung CT was normal except fibroatelectatic bands in the subpleural space (Figure 1, marked with arrow). Electro diagnostic parameters demonstrated decreased amplitude and velocity in both motor and sensory nerves although F waves were normal. Electromyography showed decreased recruitment, and findings were consistent with acute motor-sensory polyneuropathy (Tables 2, 3). Cerebrospinal fluid (CSF) analysis revealed 61.2 mg/dl glucose, 132 mg/dl chlorine, 733 mg/dl protein, 25 mg/dl lactate dehydrogenase, and cell count analysis was clear. There was no reproduction in the culture of CSF, and Gram staining was clear. The patient received 0.40 g/kg/day intravenous immunoglobulin (IVIG) for a duration of five days according to clinical manifestations related to GBS. The patient was treated with IVIG for five days. On the follow up, there was no need for respiratory or ventilator support. The patient was discharged on the 14th day of hospitalization with 3/5 motor power. On first month observation, patient regained ambulation with 4/5 motor power.

Table 1. The laboratory results of the patient

Test	Patient	Reference	Test	Patient	Reference
Serum glucose	89 mg/dl	74-106	ALT	10 IU/L	5-35
BUN	73 mg/dl	17-43	AST	30 IU/L	5-35
Creatinine	1 mg/dl	0.51-0.95	Sodium	141 mmol/L	136-146
Potassium	4.1 mmol/L	3.5-5.5	WBC	7700 cells/microliter (neutrophils= 71.6%; lymphocytes= 17%)	3.8-11.8
ESR	51 mm/h	0-20	CRP	19.5 mg/L	0-8
Hb	16.4 g/dl	10.9-14.3	Urinalysis	Normal	
EBV VCA IgM	Negative		Anti rubella IgM	Negative	
EBV VCA IgG	Positive		Anti rubella IgG	Positive	
Anti CMV IgM	Negative		Anti toxoplasma IgM	Negative	
Anti CMV IgG	Positive		Anti toxoplasma IgG	Positive	
Anti HCV	Negative		Anti HAV IgM	Negative	
Anti HIV	Negative		HbsAg	Negative	

BUN: Blood urea nitrogen, ALT: Alanine aminotransferase, AST: Aspartate aminotransferase, WBC: White blood cell count, ESR: Erythrocyte sedimentation rate, CRP: C-reactive protein, Hb: hemoglobin.

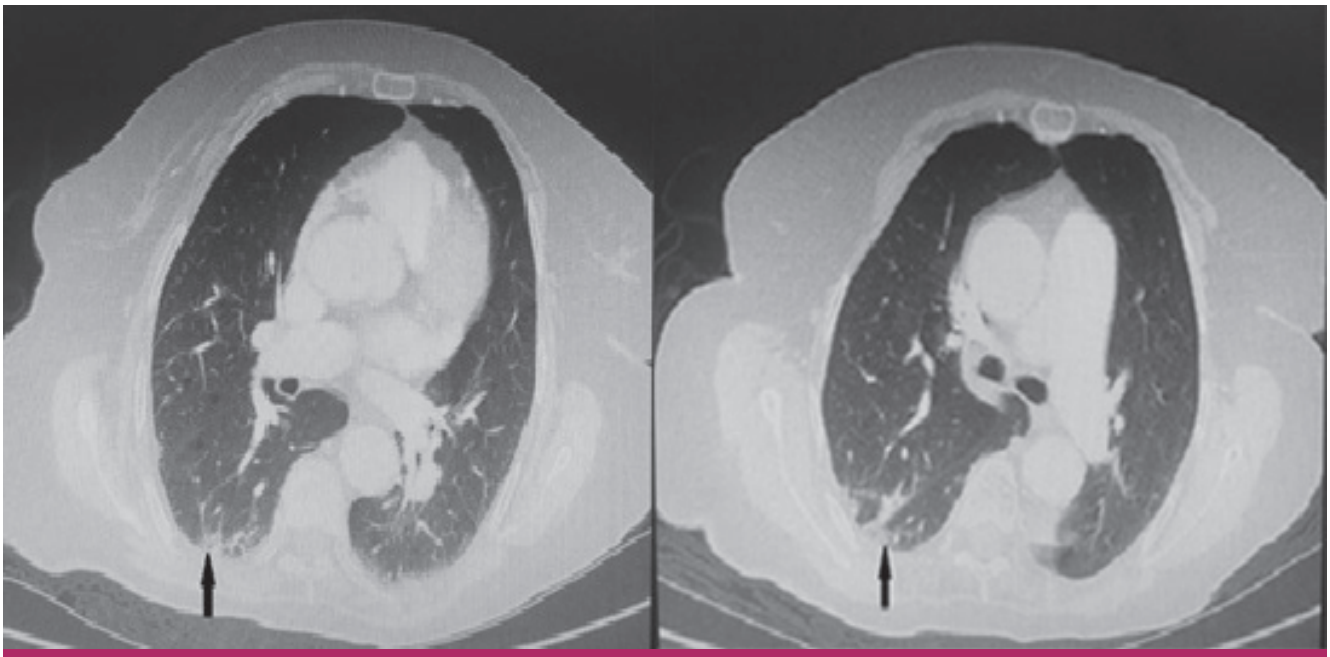

Figure 1. Computed thorax tomography of the patient.

Table 2. Nerve conduction studies; anti sensory summary table

Nerve stimulated	Stimulated site	Peak (ms)	P-T Amp (μV)	Delta-P (ms)	Vel (m/s)	Normal (m/s)	F wave (ms)
Left median anti sensory	Wrist/2nd Digit					>39	
Right median anti sensory	Wrist/2nd Digit	3.8	16.6	3.8	34	>39	20.78
Right sural anti sensory	Calf/Lat Mall	3.5/3.5	15.3/8.4	3.5	37	33.8	
Left ulnar anti sensory	Wrist/5th Digit					>37	
Right ulnar anti sensory	Wrist/5th Digit		NR			>37	21.17

NR: No response, P-T Amp: Peak to peak Amplitude, Vel: Velocity.

Table 3. Nerve conduction studies; motor summary table

Nerve stimulated	Stimulated site	Onset (ms)	Normal onset (ms)	P-T Amp (mV)	Normal P-T Amp (mV)	Vel (m/s)	Normal Vel (m/s)
Right fibular motor	Ankle B Fib	5.2 12.7	<5	3.5 3.6	>3.6	45	>40
Left median motor	Wrist elbow	4.1 9.7	<3.8	9.1 5.9	>4.3	45	>49.7
Right median motor	Wrist elbow	3.8 9.0	<3.8	7.3 6.4	>4.3	48	>49.7
Right tibial motor	Ankle knee	4.9 16.5	<6	1.6 1.5	>3.6	34	>39.6
Left ulnar motor	Wrist B elbow	2.5 6.8	<3.3	14.5 14.8	>7	56	>49.9
Right ulnar motor	Wrist B elbow	2.4 7.1	<3.3	12.0 10.2	>7	51	>49.9

P-T Amp: Peak to peak Amplitude, Vel: Velocity, B Elbow: Below Elbow, B Fib: Below Fibula.

DISCUSSION

COVID-19 has similar effects on the nervous system like SARS-CoV and MERS-CoV. There are previous studies reporting acute polyneuropathy associated with SARS-CoV and MERS-CoV (13-17). There is similarity in the sequencing of the SARS-CoV and SARS-CoV-2 spike proteins, and SARS-CoV-2 also uses ACE2 as a functional receptor (18). Most patients presented with paresthesia and progressive, flaccid quadriparesis and showed albumin-cytologic dissociation in CSF study. Acute Inflammatory Demyelinating Polyneuropathy

subtype was most commonly observed (19,20). Developing axonal polyneuropathies in the context of a viral infection suggests that the virus can cause a neural inflammatory reaction through immune mimicry, or presents as part of an inflammatory response syndrome (16). However, those mechanisms of SARS-CoV-2 related neuropathy need to be clarified. An abnormal increase in diagnoses of GBS and the prevalence of older patients were increased in the pandemic period of COVID-19. Before the pandemic, mean age of GBS was 40 years. According to cases after the pandemic, mean age of GBS has been reported as 60 years. Our patient is over the mean age of GBS, similar to the other cases reported in the pandemic period. Based on case reports, we suggest a possible association between COVID-19 infection and GBS, and SARS-CoV-2 is potentially triggering GBS (12). Several case reports have been published and shown that on average, 11.92 ± 6.20 days after COVID-19 infection, neurological symptoms of GBS begin (21). Our patient was on the 14th day of COVID-19 infection, which is similar to the average duration reported in studies. The patient was treated successfully and regained ambulation. Although the relationship between COVID-19 and GBS is still studied, it is important to report cases for collecting data pool. GBS should not be misdiagnosed when individuals who have or have had COVID-19 infection, present with neurologic symptoms.

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