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Guillain Barre syndrome associated with COVID-19 infection: A case report

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ABSTRACT

Novel outbreak with coronavirus 2019 began since 31 December 2019. Coronaviruses can cause multiple systemic infections that respiratory complications are the most obvious symptoms. In this report, we describe the symptoms of Guillain Barre syndrome (GBS) in one infected patient with COVID-19, for the first time. We reported a 65-years- old male patient with complaints of acute progressive symmetric ascending quadriparesis. Two weeks prior to hospitalization, the patient suffered from cough, fever, and RT-PCR was reported positive for COVID-19 infection. The electrodiagnostic test showed that the patient is an AMSAN variant of GBS. COVID-19 stimulates inflammatory cells and produces various inflammatory cytokines and as a result, it creates immune-mediated processes. GBS is an immune-mediated disorder and molecular mimicry as a mechanism of autoimmune disorder plays an important role in creating it. It is unclear whether COVID-19 induces the production of antibodies against specific gangliosides. Further investigations should be conducted about the mechanism of GBS in patients with COVID-19, in the future.

1. Introduction

On 31 December 2019, a novel coronavirus (COVID-19) was detected in Wuhan City, Hubei Province of China [1]. COVID-19 is a new beta coronavirus, which enters the cell via fusion with angiotensin-converting enzyme 2 (ACE2) receptor [2]. The symptoms of COVID-19 are dependent on the age and the patient's underlying medical illness and also the condition of the immune system [3]. Chen and et al. reported that most of the infected patients suffer from an underlying disease including hypertension, cardiovascular disease, and diabetes mellitus [4]. Coronaviruses can cause multiple systemic infections that respiratory complications are the most recognizable symptoms similar to severe acute respiratory syndrome coronavirus (SARS-CoV). The most prevailing symptoms at the onset of disease, after an incubation period of approximately 5.2 days, are Fever, cough, dyspnea, myalgia, headache, and diarrhea [5]. Some studies reported gastrointestinal complications, acute cardiac damage, and acute renal failure due to COVID-19 infection [6,7]. Mao and et al evaluated neurological symptoms in 214 patients infected with COVID-19 (8). Of 214 hospitalized patients, 36.4% had nervous system manifestations including dizziness, headache, hypogeusia, hyposmia, muscle damage, ischemic and hemorrhage stroke [8]. In our knowledge, up to now, no reported neuropathy and/or Guillain–Barré syndrome (GBS) due to COVID-19 infection. GBS is an acute immune-mediated disease of the peripheral nerves and nerve roots (polyradiculoneuropathy) that is usually elicited by various infections [9]. The classic clinical manifestations of GBS is progressive, ascending, symmetrical flaccid limbs paralysis, along with areflexia or hyporeflexia and with or without cranial nerve involvement, which can progress over the course of days to several weeks [9]. Two-thirds of patients usually report respiratory tract or gastrointestinal infection 2–4 weeks prior to the onset of neurological symptoms of GBS (10). In this report, we describe GBS symptoms in one infected patient with COVID-19, for the first time.

2. Case presentation

A 65-years- old male patient was admitted to the emergency department, with symptoms of acute progressive symmetric ascending quadriparesis. Neurological manifestations of the patient began with acute progressive weakness of distal lower extremities, five days before admission. At that time, the symptoms progressed from distal limbs to proximal limbs and he had been quadriplegia one day before admission. There was facial paresis bilaterally. He had no urinary and fecal incontinence. Two weeks prior to hospitalization, the patient suffered from cough, fever and sometimes dyspnea. At that time, he referred to an infectious disease specialist and was diagnosed with COVID-19 after examining oropharyngeal sampling, and chest computer tomography (CT). Reverse transcription-polymerase chain reaction (RT-PCR) for COVID-19 was positive and the patient was treated with hydroxychloroquine, Lopinavir/Ritonavir (LPV/RTV) and Azithromycin. In the past medical history, the patient was a well-known case of type 2 diabetes mellitus and was treated with metformin medication.

On physical examination, the patient was afebrile with blood pressure 120/80 mm/hg, heart rate 73 beats/minute, respiratory rate 18/minute, and oxygen saturation of 95% on room air. The patient was conscious and had no dyspnea, at the time of hospitalization. The muscle strength examination showed weakness in four limbs with a Medical Research Council (MRC) scale of 2/5 in proximal, 3/5 in distal of the upper extremities and 1/5 in proximal, 2/5 in distal of the lower extremities. Deep tendon reflexes were absent generally. There was a reduction in the vibration and fine touch sensation distal to the ankle joints and also bifacial nerve

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palsy (House–Brackmann grade 3). He had no spine sensory level. Meningeal irritation signs and upper motor neuron disorder signs were negative. The laboratory examination results were follows: serum glucose 159 mg/dL; blood urea nitrogen: 19 mg/dL; creatinine 0.8 mg/dL; alanine aminotransferase 35 IU/L; aspartate aminotransferase 47 IU/L; sodium 135 mmol/L; potassium 3.9 mmol/L; white blood cell count 14,700 cells per microliter (neutrophils = 82.7%; lymphocytes = 10.4%); Erythrocyte sedimentation rate 72 mm/hour, C-reactive protein 2+, hemoglobin 11.6 g/dL and negative glucose and ketone in complete urinalysis. Cervical and brain magnetic resonance imaging (MRI) was done and showed a normal finding except for mild herniation of two intervertebral discs. Lung CT showed diffused consolidations and ground-glass opacities in both lungs, and bilateral pleural effusion (Fig. 1). On day 9, the neurophysiological study was performed. Electro diagnostic parameters demonstrated decreased amplitude at compound muscle action potential and no response at sensory nerve action potential. Electromyography showed decreased recruitment. These findings are consistent with acute motor-sensory axonal neuropathy (Table 1). Cerebrospinal fluid (CSF) analysis was not performed due to a lack of consent. Our patient received 0.40 g/kg/day intravenous Immunoglobulin for a duration of five days according to clinical manifestations related to GBS.

3. Discussion

In this study, we reported Guillain Barre syndrome in a patient infected with COVID-19. COVID-19 is a beta coronavirus akin to SARS and middle east respiratory syndrome (MERS) such that, fever, cough, and difficulty breathing are the first symptoms that commonly were reported in infected patients with these viruses ([11]. Neurological symptoms associated with COVID-19 infection have been reported by Mao and et al. study [8]. The neurological manifestations had also reported in other beta coronavirus (SARS and MERS) that had been including polyneuropathy, myopathy, stroke and GBS [12]. To our knowledge, neuropathy and GBS have not been reported by infection with COVID-19, yet. Our patient had classic symptoms of GBS that had been begun approximately two weak after respiratory tract infection with COVID-19. For the reason that this patient had a preceding infection history, relative symmetric limbs weakness with a monophasic course, we suggested GBS variant as a possible diagnosis [9]. Electrodiagnostic findings confirmed this disorder. Studies on coronaviruses have been shown that these viruses have neurotrophic and neuroinvasive characteristics [11]. Although, these studied no performed about COVID-19 but the arrangement of SARS and MERS, especially SARS, are very similar to that of COVID-19 (11). Both SARS and COVID-19 attach to the angiotensin-converting enzyme 2 receptor [5,11]. This receptor detected in the cell membrane of numerous human organs, including lung, kidney, liver, nervous system and skeletal muscle [8]. The mechanism of GBS formation in patients infected with COVID-19 has not yet been investigated. COVID-19 stimulates inflammatory cells and produces various inflammatory cytokines and as a result, it creates immune-mediated processes [5]. GBS is an immune-mediated disorder and molecular mimicry as a mechanism of autoimmune disorder plays an important role in creating it [10]. It is unclear whether COVID-19 induces the pro-

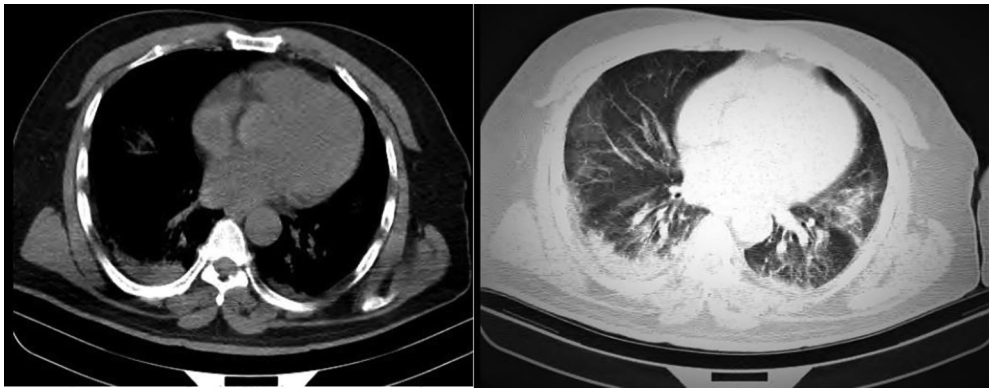

Fig. 1. Chest computer tomography revealed ill-defined opacities in both of lungs.

Table 1
Nerve conduction study parameters in the patient with GBS.

Nerve Stimulated	Stimulation Site	*Amplitude		Latency (ms)		Conduction velocity		F wave	
		RT	LT	RT	LT	RT	LT	RT	LT
Median (s)	Wrist	NR	NR	NR	NR	NR	NR		
Ulnar(s)	Wrist	NR	NR	NR	NR	NR	NR		
Sural (s)	Calf	NR	4.5	NR	2.00	NR	35		
Median (m)	Wrist	2.3	1.8	6.61	7.59			49	52
	AF	1.9	1.3	13.90	14.27	31	41		
Ulnar (m)	Wrist	4.7	4.4	4.17	4.69			41.9	48
	BE	4.6	3.5	8.22	8.52				
	AE	4.5	3.4	9.90	10.20	46	39		
Tibial (m)	Ankle	1.00	0.4	9.95	12.19			NR	NR
	Popliteal F.	0.90	0.4	20.63	27.03	29	21		
Peroneal(m)	Ankle	NR	NR	NR	NR	NR	NR		

* Amplitude motor = mV, Sensory= μV; m = motor study; s = sensory study; RT = right; LT = left; AF = antecubital fossa; BE = below elbow; AE = above elbow; BF = below fibula; LP = lateral popliteal fossa; NR = no response; GBS = Guillain Barre syndrome.

duction of antibodies against specific gangliosides that usually appear with certain forms of GBS. Further investigations should be conducted about the mechanism of GBS in patients with COVID-19, in the future.

4. Conclusion

In summary, to our knowledge, this is the first reported case of GBS in a patient infected with COVID-19. Given that the most common symptoms of infection with COVID-19 were reported respiratory infections and two-thirds of Guillain-Barre patients usually mention respiratory infections before the onset of symptoms, hence GBS should be considered as neurological complications of infection with COVID-19. Therapy with IVIG or plasmapheresis should be initiated along with antiviral treatment.

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Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jocn.2020.04.062.

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