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COVID-19-Associated Guillain-Barré Syndrome: A Case Report

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Abstract **Case Report**

Guillain-Barré syndrome (GBS) is an inflammatory polyradiculoneuropathy associated with numerous viral infections. Recently, there have been many case reports describing the association between coronavirus disease-2019 (COVID-19) and GBS [1]. 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 67-years- old male patient with no pathological history, complaints of acute progressive symmetric ascending quadriparesis. Two weeks prior to hospitalization, the patient suffered from fever and cough. After hospitalization a thoracic CT scan showed 10-25% of consolidation and ground glass opacities. RT-PCR was reported positive for COVID-19 infection. Electrodiagnostic tests five days after neurological symptom onset showed a demyelinating pattern in accordance with Guillain–Barré syndrome (GBS) criteria.

Keywords: COVID-19, Guillain-Barré syndrome (GBS), fever and cough, neurological symptom.

INTRODUCTION

The novel coronavirus (COVID-19) is a global pandemic. Although the main clinical manifestation of COVID-19 is respiratory involvement, there is evidence suggesting the neuroinvasive potential of COVID-19 (2). We report a case of a patient affected by COVID-19, presenting with symmetric ascending quadriparesis with the final diagnosis of Guillain-Barré syndrome.

CASE REPORT

A 67-year-old man with no pathological history presented with symptoms of acute progressive symmetric ascending quadriparesis. Neurological manifestations of the patient began with acute progressive weakness of distal lower extremities, seven days before admission. There was facial paresis bilaterally. He had no urinary and fecal incontinence. Two weeks prior to hospitalization, the patient suffered from vomiting, cough, and fever with no further explorations.

On physical examination, the patient was febrile, hypertendive with blood pressure 230/15 mm/hg, heart rate 100 beats/minute, respiratory rate 30/minute, and oxygen saturation of 87% on room air. The patient was conscious, at the time of hospitalization. The muscle strength examination showed weakness in four limbs with a Medical Research Council (MRC) scale of 1/5 in proximal, 2/5 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. 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 2.95 g/L; blood urea nitrogen: 0.58 g/L; creatinine 5.9 mg/L; sodium 122 mmol/L; white blood cell count 12,940 cells per microliter (neutrophils = 70%; lymphocytes = 18.5%); Erythrocyte sedimentation rate 68 mm/hour, C-reactive protein 1.2, hemoglobin 16.7 g/dL and negative glucose and ketone in complete urinalysis. Lung CT showed diffused consolidations and ground-glass opacities in both lungs (Fig-1). RT-PCR was reported positive for COVID-19 infection. The neurophysiological study was performed. Electro diagnostic parameters demonstrated decreased amplitude at compound muscle action potential. These findings are consistent with acute motor-sensory axonal neuropathy (Table-1). Cerebrospinal fluid (CSF) analysis was performed and albuminocytologic dissociation was noted (prot=4, cell=3). Our patient received 0.40 g/kg/day intravenous Immunoglobulin for a duration of five days according to clinical manifestations related to GBS. The patient complained swallowing disturbance with a risk of suffocation as liquids took the wrong path. The patient was admitted in ICU and mechanically ventilated because of respiratory insufficiency. He he deceased 3 days later.

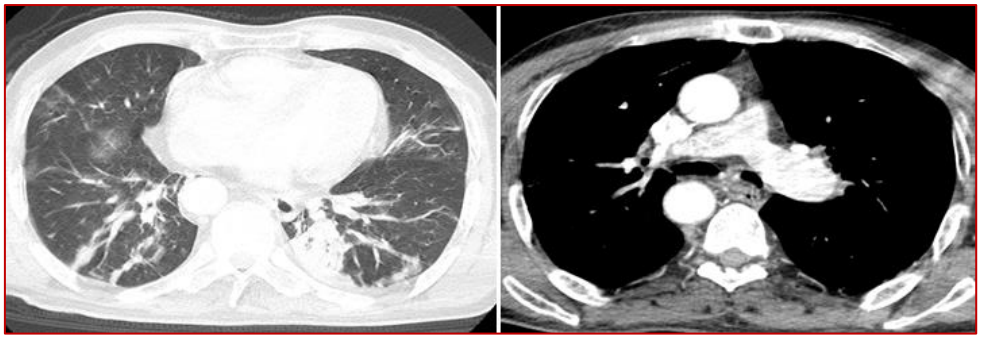

Fig-1: CT scan with enhanced contrast, lung window and mediastinal window, demonstrating
10-25% consolidation and ground glass opacities. No signs of pulmonary embolism on the mediastinal window with a right aortic arch

Table-1: Motor and sensitive nerve conduction study

SNC

Nerf / Sites	Rec. Site	Onset Lat ms	Peak Lat ms	Amp.2-3 µV	Segments	Distance mm	Velocity m/s	Vel CT m/s
D Cubital - Digit V (Antidromic)								
Poignet	Dig V	Abs	Abs	Abs	Poignet - Dig V		Abs	Abs
G Cubital - Digit V (Antidromic)								
Poignet	Dig V	Abs	Abs	Abs	Poignet - Dig V		Abs	Abs
D Radial - Anatomical snuff box (Avant-bras)								
Avant bras	Poignet	2.23	2.75	15.1	Avant-bras - Poignet	100	45	44,9
G Radial - Anatomical snuff box (Avant-bras)								
Avant bras	Poignet	2.33	2.85	17,9	Avant-bras - Poignet	135	58	57,9
G Sural - Cheville (Mollet)								
Mollet	Cheville	Abs	Abs	Abs	Mollet - Cheville		Abs	Abs
D Sural - Cheville (Mollet)								
Mollet	Cheville	Abs	Abs	Abs	Mollet - Cheville		Abs	Abs
D Médian - Digit III (Antidromic)								
Poignet	Dig III	Abs	Abs	Abs	Poignet - Dig III		Abs	Abs
G Median - Digit III (Antidromic)								
Poignet	Dig III	1,98	2,33	17,6	Poignet - Dig III	80	40	40,4

MNC

Nerf / Sites	Muscle	Latency ms	Amplitude mV	Duration ms	Rel Amp %	Segments	Distance mm	Lat Diff ms	Velocity m/s	Vel CT m/s
D SPI - FGO										
Cheville	FGO	15,04	2,6	10,92	100	Cheville - FGO				
Pop fossa	FGO	30,71	1,3	10,48	48,5	Pop fossa - Cheville	380	15,67	24	24,3
G SPI - FGO										
Cheville	FGO	16,08	2,9	8,83	100	Cheville - FGO				
Pop fossa	EDB	31,08	2,0	8,86	70 6	Pop fossa - Cheville	380	15,00	25	25,3
D Médian - CAP										
Poignet	APB	8,65	2,3	7,88	100	Poignet - APB				
Coude	APB	16,85	1,8	7,67	78,5	Coude - Poignet	300	8,21	37	36,5
G Médian - CAP										
Poignet	APB	5,75	2,8	6,33	100	Poignet - APB				

DISCUSSION

Despite numerous case reports of GBS associated with COVID-19, the prevalence remains unclear, since ascertainment and reporting are uneven and the total number of concurrent COVID-19 patients is not known. Most patients reported were over age 50 years and male which likely reflects the underlying demographics of diagnosed COVID-19 early in the pandemic. That is, older age and male gender are risk factors for more severe COVID-19, and the incidence of GBS rises with age [3,4]. The male predominance is slightly higher than that reported in a large series of non-COVID-19 GBS cases40 [1].

Neurological symptoms associated with COVID-19 infection have been reported by Mao and et al., study [5]. The neurological manifestations had also reported in other beta coronavirus (SARS and MERS) that had been including polyneuropathy, myopathy, stroke and GBS [6]

GBS is an acute immune-mediated disease of the peripheral nerves and nerve roots (polyradiculoneuropathy) that is usually elicited by various infections [7]. 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 [7]. Two-thirds of patients usually report respiratory tract or gastrointestinal infection 2–4 weeks prior to the onset of neurological symptoms of GBS [8]. In this report, we describe GBS symptoms in one infected patient with COVID-19

COVID-19 stimulates inflammatory cells and produces various inflammatory cytokines and as a result, it creates immune-mediated processes [9]. GBS is an immune-mediated disorder and molecular mimicry as a mechanism of autoimmune disorder plays an important role in creating it [8].

The role of the thoracic CT scann is no longer to establish for the earyl diagnosis of the covid-19 pneumonia, it certainly helps for the early management of patients and complications including neurological ones. However even if it is unclear whether a causal relationship exists between GBS and Covid-19 physicians should be aware of this possible complication when dealing with COVID-19 patients.

CONCLUSION

GBS is a neurological emergency. Quick recognition of symptoms and diagnosis is important in the management of these patients. A causal relationship between GBS and Covid-19 is yet to establish but physicians should be aware of it using todays tools such as thoracic CT and RT-PCR in patient with GBS and a history of fever or cough to guide the etiological diagnosis

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