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Case Report

Guillain Barre syndrome as a complication of SARS-CoV-2 infection: A case report

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ABSTRACT

Introduction: Covid-19 infection usually manifests with respiratory symptoms, but neurological signs might be the mean symptom revealing this infection such as Guillain Barre syndrome (GBS).
COVID-19 associated GBS seems to be more severe than non-COVID-19 GBS.
Case management: We reported a 49 old-man admitted in the intensive care unit for bilateral ascending symmetrical paresthesia associated with lower limb numbness and sphincter disorders two weeks after an upper respiratory infection. The diagnosis of post-Covid-19 GBS was maintained, and the evolution was favorable after Intravenous Immunoglobulin (IVIg) and plasma exchange (PLEX) as a second therapy.
Conclusion: This case report suggest the probable causal link between COVID 19 and GBS. This severe association prompts us to do further research that may help professionals in an early diagnosis and early treatment thus improving morbidity and mortality.

1. Introduction

Guillain-Barré Syndrome (GBS) is a rare peripheral immune-mediated neuropathy manifesting with bilateral ascending symmetrical weakness occurring one to two weeks after immune stimulation or infection [1]; It has been mentioned in the literature as a sequela of COVID-19 infection [2].

The management of this condition requires supportive care, intravenous immunoglobulin (IVIg), and Plasma exchange (PLEX) [3,4].

The rate of recovery is approximatively 80% and the force in the lower limb regains after 6 months [3,4].

In this paper, we will report the clinical case of 49 year-old-man with no medical history, admitted to the intensive care unit for GBS occurring 2 weeks after COVID-19 infection.

2. Case presentation

A 49-year-old man with no medical history was admitted to our intensive care unit for bilateral ascending symmetrical paresthesia with lower limb numbness, nocturnal low back pain, and sphincter disorders: urinary and anal retention, 14 days after an upper respiratory infection with Covid-19 confirmed by a nasopharyngeal swab testing for SARS-CoV-2 with real-time polymerase chain reaction assay (RT-PCR).

The initial clinical assessment was as follows, Glasgow coma-scale (GCS) 15/15 with gait ataxia and a peripheral radicular sensitive-motor neurogenic syndrome of both lower limbs. The muscle force was 0/5 in the lower limb and 2/5 in the upper limb, with abolition of osteo-tendinous reflexes, thermalgic and tactile sensitivity. Coordination was preserved and the cranial nerves were spared.

The cerebrospinal fluid (CSF) analysis showed albumin-cytologic dissociation: protein level of 65 mg/dl and cells 2/mm3.

An Encephalic and medullar MRI (magnetic resonance imaging) was

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Table 1
Neurophysiological findings.

Motor nerve conduction study			Latency proximal/distal (ms)	Amplitude proximal/distal (mV)	Velocity (m/s)	Sensitive nerve conduction study	Latency proximal/distal (ms)	Amplitude proximal/distal (mV	Velocity (m/s)
Medial	L	wrist	3.3	7.5	–	MEDIAL (L)	1.5	38.9	9.6
		elbow	8.1	8.2	62.5				
		crease	13.7	9.3	67.3				
		ERB point							
Medial	R	wrist	3.6	7.3	64.5	Medial (R)	1.434.8	104	
		elbow	8.3	8.2	66.1				
		crease	14.0	8.8					
		ERB point							
Ulnar	L	wrist	2.5	5.2	–	Ulnar (L)	1.4	23.3	–
		elbow	7.9	6.6	55	(R)	NE	NE	NE
		crease	14.2	5.1	60.8				
		ERB point							
Ulnar	R	wrist	2.5	4.5	–	Radial (L)	1.3	17.0	–
		elbow	8.1	5.2	53				
		crease	13.4	3.8	72.4				
		ERB point							
Tibial	L	Malleolus	5.0	4.3	–	Radial (R)	NE	NE	NE
		Popliteal fossa	15.1	4.2	41.6				
Tibial	R	Malleolus	6.1	6.4	–	Sural (L)	1.0	11.1	
		Popliteal fossa	15.9	5.3	40.6				
Fibular	R	Head of	4.9	3.4	–	Sural (R)	NE	NE	NE
	L	Fibula	4.9	3.4	–				

L = left; R = right; NE = not evocable.
This case report follows scare guidelines [5].

performed as normal. The electromyography (EMG) showed prolonged F wave latency of external and internal popliteal sciatic nerves bilaterally (EPS and IPS), otherwise normal motor and sensory nerve conduction (Table 1).

The complete blood count was as follow: Hemoglobin 12,6 g/l, white blood cells 6170/mm3, platetes 336 000/mm3, Ionogram: Natremia 136 mmol/L, Kaliemia 4,2 mmol/L, calcemia 85 mmol/L, normal kidney function: urea 0.24 g/L, Creatinine 9,98 mg/dL, CRP 1,77 mg/L, high level of ferritin with 266,51μg/L and interleukin-6 (IL 6) 4,8 pg/mL.

The chest radiography and computed tomography (CT) scan were negative for pneumonia.

Initial treatment of intravenous immunoglobulin (IVIG) was started with a dose of 0.4 g/kg/day for 5 days with no improvement leading to initiating plasma exchange (PLEX) therapy: for 7 sessions with an exchange of 3 L of plasma each session.

The patient performed daily physical rehabilitation in bed with a physiotherapist. On day 28 of GBS installation, he showed a significant improvement of force muscles in both upper and lower limbs. Throughout his stay in the intensive care unit, oral nutrition was maintained, as was social interaction face-to-face with loved ones. Indeed, he was informed daily of his health state. Two months later, he was referred to the rehabilitation center.

3. Discussion

The lungs are the most affected organs of Covid-19 infection. However extra respiratory manifestations are also observed, such as cardiac, renal, neurologic and gastro intestinal symptoms.

GBS has been described as a complication of COVID-19 infection [6,7]. The recent studies showed a possible link between the two affections [8,9].

The mechanism of neurological manifestation of Covid-19 can be explained by the presence of ACE2 receptors on neuronal tissues, and the skeletal muscles, directly or indirectly through inflammatory response of hypoxic injury [9,10]. Further studies are necessary to comprehend the link between COVID-19 infection and the nervous system.

The GBS is the first neurological manifestation occurring between 5 and 21 days after COVID-19 [11]. In this case, our patient represented the progression of numbness and weakness 14 days after symptoms.

De Sanctis et al. reported that cough and fever were the most frequent symptoms in patients Showing GBS after COVID-19 infection [12].

It is important to highlight that respiratory failure was present in GBS related to SARS-CoV2, this suggests the coexistence of COVID-19 pneumonia and GBS respiratory muscle weakness [13]. Our reported observation indicates that the patient had isolated GBS without respiratory signs due to Covid-19 infection and his CT scan was negative for pneumonia.

Covid-19 has been found to be implicated in severe cases of GBS [14,15]. Other pathogens can also be identified such as cytomegalovirus, Epstein–Barr virus, Mycoplasma pneumonia, Haemophilus influenzae, and influenza A virus [16].

The treatment was initiated in our patient with a course of IVIg, followed by plasma exchange (PLEX) as a second therapy, which has shown its efficiency, as in most studies Intravenous immunoglobulins and PLEX are the two main immunotherapy treatments and the most common regime adopted for GBS [17,18].

Our patient was satisfied with our medical care. Despite his transfer to the rehabilitation center, our medical team continues to follow up on his state of health.

We summarize that GBS also occurs in patients with COVID-19 infection, who have never had respiratory symptoms before. further studies should be conducted on the link between early neurological symptoms and the neurological consequences of COVID-19.

4. Conclusion

According to the case reports from around the world, it is possible that SARS-CoV-2 potentially triggering GBS. Clinical presentation, CFS, electrophysiological outcomes, and response to treatment are similar in COVID-19 infection associated with GBS and GBS related to others viruses.

The post-COVID-19 neurological sequelae might require early recognition of GBS symptoms to improve the quality of life and reduce

the high risk of mortality.

Consent

Obtained.

Ethical approval

The ethical committee approval was not required give the article type case report. However, the written consent to publish the clinical data of the patients were given and is available to check by the handling editor if needed.

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This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Author contribution

EL AIDOUNI Ghizlane: Corresponding author, study concept, data collection, data analysis, writing review & editing. TAOUIHAR Salma: Study concept, data collection, data analysis. AABDI Mohammed: Writing review. MERBOUH Manal: Contributor. EL KAOUINI Abderrahim: Contributor. BOUABDALLAOUI Amine: Contributor. ES-SAAD Ounci: Contributor. BKIYAR Houssam: Supervision and data validation. HOUSNI Brahim: Supervision and data validation.

Registration of research studies

This is not an original research project involving human participants in an interventional or an observational study but a case report. This registration was not required.

Guarantor

EL AIDOUNI Ghizlane.

Declaration of competing interest

The authors state that they have no conflicts of interest for this report.

References

[1] Aabdi Mohamed, Mellagui Yassine, Bensaid Amine, Bkiyar Houssam, Housni Brahim, Guillain Barré syndrom during the post-partum period, Cureus 12 (12) (2020 Dec 10), e12021, https://doi.org/10.7759/cureus.12021.

[2] Antonio Zito, Enrico Alfonsi, Diego Franciotta, Massimiliano Todisco, Matteo Gastaldi, Matteo Cotta Ramusino, Mauro Ceroni, Alfredo Costa, COVID-19 and Guillain–Barré syndrome: a case report and review of literature, Front. Neurol. 11 (2020) 909, https://doi.org/10.3389/fneur.2020.00909.

[3] S.E. Leonhard, M.R. Mandarakas, F.A. Gondim, K. Bateman, M.L. Ferreira, D. R. Cornblath, P.A. van Doorn, M.E. Dourado, R.A.C. Hughes, B. Islam, et al., Diagnosis and management of Guillain-Barré syndrome in ten steps, Nat. Rev. Neurol. 15 (2019) 671–683, https://doi.org/10.1038/s41582-019-0250-9.

[4] Pasquale Sansone, Luca Gregorio Giaccari, Caterina Aurilio, Francesco Coppolino, Valentina Esposito, Marco Fiore, Antonella Paladini, Maria Beatrice Passavanti, Vincenzo Pota, Maria Caterina Pace, Post-infectious Guillain–Barré syndrome related to SARS-CoV-2 infection: a systematic review, Life (Basel) 11 (2) (2021 Feb) 167, https://doi.org/10.3390/life11020167.

[5] R.A. Agha, T. Franchi, C. Sohrabi, G. Mathew, for the SCARE Group, The SCARE 2020 guideline: updating consensus surgical CAse REport (SCARE) guidelines, Int. J. Surg. 84 (2020) 226–230, https://doi.org/10.1016/j.ijsu.2020.10.034.

[6] Meysam Abolmaali, Matineh Heidari, Marjan Zeinali, Parichehr Moghaddam, Mona Ramezani Ghamsari, Mahin Jamshidi Makiani, Zahra Mirzaasgari, Guillain–Barré syndrome as a parainfectious manifestation of SARS-CoV-2 infection: a case series, J. Clin. Neurosci. 83 (2021 Jan) 119–122, https://doi.org/10.1016/j.jocn.2020.11.013.

[7] L. Mao, M. Wang, S. Chen, Q. He, J. Chang, C. Hong, et al., Neurological manifestations of hospitalized patients with COVID-19 in Wuhan, China: a retrospective case series study, JAMA Neurol 77 (6) (2020 Jun 1) 683–690, https://doi.org/10.1001/jamaneurol.2020.1127.

[8] Kaveh Rahimi, Guillain-Barre syndrome during COVID-19 pandemic: an overview of the reports, Neurol. Sci. 41 (11) (2020 Nov) 3149–3156, https://doi.org/10.1007/s10072-020-04693-y.

[9] V. Montalvan, J. Lee, T. Bueso, J. De Toledo, K. Rivas, Neurological manifestations of COVID-19 and other coronavirus infections: a systematic review, Clin. Neurol. Neurosurg. 194 (2020), 105921, https://doi.org/10.1016/j.clineuro.2020.105921.

[10] Y. Wu, X. Xu, Z. Chen, J. Duan, K. Hashimoto, L. Yang, et al., Nervous system involvement after infection with COVID-19 and other coronaviruses, Brain Behav. Immun. (2020), https://doi.org/10.1016/j.bbi.2020.03.031.

[11] L.M. Trujillo Gittermann, S.N. Valenzuela Feris, A. Von Oetinger Giacoman, Relation between COVID-19 and Guillain Barré syndrome in adults: a systematic review, Neurologia 35 (9) (2020 November-December) 646–654, https://doi.org/10.1016/j.nrl.2020.07.004.

[12] P. De Sanctis, P.E. Doneddu, L. Viganò, C. Selmi, E. Nobile-Orazio, Guillain–Barré syndrome associated with SARS-CoV-2 infection. A systematicr eview, Eur. J. Neurol. 27 (11) (2020) 2361–2370, https://doi.org/10.1111/ene.14462. Epub 2020 Sep. 11.

[13] C. Tassorelli, F. Mojoli, F. Baldanti, R. Bruno, M. Benazzo, COVID-19: what if the brain had a role in causing the deaths? Eur. J. Neurol. 27 (9) (2020 Sep) e41–e42, https://doi.org/10.1111/ene.14275.

[14] H. Zhao, D. Shen, H. Zhou, J. Liu, S. Chen, Guillain-Barre syndrome associated with SARS-CoV-2 infection: causality or coincidence? Lancet Neurol. 19 (2020) 383–384, https://doi.org/10.1016/S1474-4422(20)30109-5.

[15] P. Alberti, S. Beretta, M. Piatti, A. Karantzoulis, M.L. Piatti, P. Santoro, M. Viganò, G. Giovannelli, F. Pirro, D.A. Montisano, I. Appollonio, C. Ferrarese, Guillain-Barré syndrome related to COVID-19 infection, Neurol. Neuroimmunol. Neuroinflamm (2020), https://doi.org/10.1212/NXI.0000000000000741.

[16] B.C. Jacobs, et al., The spectrum of antecedent infections in Guillain-Barre syndrome: a case–control study, Neurology 51 (1998) 1110–1115.

[17] R.A.C. Hughes, A.V. Swan, P.A. van Doorn, Intravenous immunoglobulin for Guillain-Barré syndrome, Cochrane Database Syst. Rev. 2014 (9) (2014 Sep 19), CD002063, https://doi.org/10.1002/14651858.CD002063.

[18] J.C. Raphaël, S. Chevret, R.A. Hughes, D. Annane, Plasma exchange for Guillain-Barré syndrome, Cochrane Database Syst. Rev. 2 (2002), CD001798, https://doi.org/10.1002/14651858.CD001798.