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 (IVlg), 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...
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. This case report follows scare guidelines [5].

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 associated with 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.

### Table 1

| 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) |
|-----------------------------|------------------------------|--------------------------------|----------------|-------------------------------|-----------------------------|--------------------------------|----------------|
| Tibial L Malleolus Popliteal fossa | 6.1 | 6.4 | 40.6 | Tibial R Malleolus Popliteal fossa | 15.5 | 15.6 | 6.4 |
| Tibial L Malleolus Popliteal fossa | 5.0 | 4.3 | 41.6 | Tibial R Malleolus Popliteal fossa | 15.8 | 15.5 | 6.2 |
| Tibial L Malleolus Popliteal fossa | 4.9 | 3.4 | 72.4 | Tibial R Malleolus Popliteal fossa | 15.9 | 15.3 | 6.0 |
| Tibial L Malleolus Popliteal fossa | 4.9 | 3.4 | 40.6 | Tibial R Malleolus Popliteal fossa | 15.9 | 15.3 | 6.0 |
| Tibial L Malleolus Popliteal fossa | 4.9 | 3.4 | 72.4 | Tibial R Malleolus Popliteal fossa | 15.9 | 15.3 | 6.0 |
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L = left; R = right; NE = not evocable.

This case report follows scare guidelines [5].
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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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 KAOUNI 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.

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