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Guillain-Barré Syndrome as Symptom of COVID-19 Infection: A Case Report

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Abstract

Several neurological manifestations have been cited in literature as indicative symptoms or associated with acute coronavirus infection syndrome. Guillain-Barré syndrome (GBS) is one of the most frequently encountered among these neurological manifestations. Our case is a post-covid 19 and to the best of aur knowledge, this case is the first reported from Tunisia.

Abbreviations

GBS: Guillain- Barré syndrome, MRI: magnetic resonance imaging

Keywords

Guillain –Barre syndrome, Covid-19, MRI, paraplegia

1. INTRODUCTION

During Corona Virus pandemic, GBS is the most neurological complication of covid-19 infection that is reported in literature. Indeed, at least 220 cases have been reported to date [1]. Various studies in progress are trying to find an explication of the relationship between the virus and this disease as well as many other neurological symptoms. In most of these cases, GBS manifestations appear after a flu-syndrome related to Covid-19 infection. In the midst of this crisis, and through our case where GBS was a circumstance of covid-19 infections' discovery, this article aims to enrich experiences about the diagnosis and the management of GBS post-COVID-19 cases reported in literature.

2. CASE PRESENTATION

We report the case of a 53-year-old woman, without a past medical history, presented to the emergency department with a 5 days history of increasing difficulty walking and exacerbated events of urine leakage. The aggravation of her clinical symptoms, marked by a paraplegia and an anesthesia of both lower limbs, urged her to consult a doctor.

In the emergency room, her hemodynamic status was correct with blood pressure of 120/70 mmHg and pulse rate of 80 beats per minute. She had no cough, fever or anosmia at that time. Her respiratory rate and oxygen saturation were normal.

Neurological examination showed a neurological symmetrical weakness on distal and proximal extremities, with lowing of muscle strength; lower extremities measured of level 1/5 and upper extremities measured of level 3/5. She had also sensory disorders with dysesthesia and decrease in sensation to pain and temperature at her lower extremities. There was a weakness of deep tendon reflexes, but no abnormalities of the cranial nerves.

The patient was admitted in neurologic department on suspicion of spinal cord compression. So, a Magnetic Resonance Imaging (MRI) was undergone, showed a thickening and enhancement of intrathecal spinal nerve roots as well as cauda equina (**figure 1**). Otherwise, no cerebrospinal signal abnormality was found.

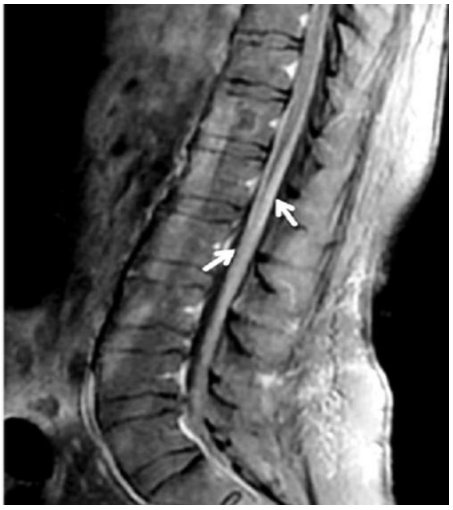

Figure1. *Sagittal Fat-Sat T1- weighted image gadolinium contrast of lumbar spine demonstrates a thickening and enhancement of nerve roots of cauda equina.*

Thus, the axial T2 weighted MRI image through the dorsal spine revealed a hypersignal at the level of posterior basal segments of lungs. A coronal STIR weighted MRI image was performed confirming the presence of confluent bilateral hyper signals, predominantly basal and posterior of lung segments (**figure 2**).

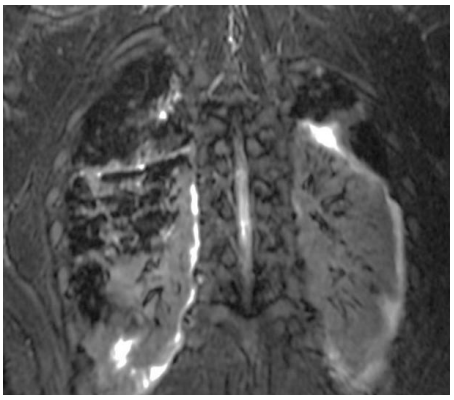

Figure2. *Coronal T2 weighted STIR image of the thorax demonstrate a confluent bilateral hyper signals in basal and posterior of lung segments.*

Based on MRI data and the neurological deficit of the patient, a Covid 19 pneumonia complicated by a GBS has been suspected.

At first, a Covid PCR test was done therefore came back positive. Afterwards, to confirm the GBS, a lumbar puncture was done and the cerebrospinal fluid analysis showed an albumin-cytological dissociation with rising protein levels and a hypoglycorrhachia, while culture was negative.

An electromyogram was performed, confirming the diagnosis of an acute demyelinating polyneuropathy with decreased nerve conduction speed. So, the diagnosis of GBS post-covid infection was retained. Thus prompting initiation of intravenous immunoglobulin (IVIg, 0, 5 g/kg for 5 days).

Meanwhile, the patient had rapid deterioration of her state, with progression of the muscle weakness. Thus, she developed an acute respiratory distress syndrome needing his admission to the intensive care unit for monitoring and further evaluation.

Under antibiotic and immunotherapy, the patient's condition has stabilized and from the 10th day of hospitalization, she started to have a progressive recovery of muscle tone, she had improvement of motor skills and sensory detection, as well as walking; she starts walking with help. Three days later and, despite à prophylactic heparin therapy, the patient presented an altered state of consciousness, with dyspnea and desaturation. A computed tomography angiography was undergone revealed a massive pulmonary embolism.

Despite all resuscitation measures, the patient died of thromboembolic complications two days later.

3. Discussion

Since the start of the epidemic, various symptoms related to covid-19 infection has been encountred. Until now, the main target has been the respiratory system, but also many other systems were affected [2]. The neurological system seems to be particularly affected and neurological disorders were frequently described. This neurological damage can occur in the acute phase by branches of the olfactory and trigeminal nerve or, in post-infectious phase, by immune phenomenon. The GBS is the most reported neurological complications among all these complications reported throughout the world [3].

As is well known, GBS is related to immune system disorders which attack the cells of the peripheral nervous system. The common clinical presentation is a weakness or even a progressive paralysis, usually starts in the lower limbs and ascends up to the head and neck. Several infectious agents preceding GBS have been reported such as herpes virus, HIV and influenza virus. The corona virus is another agent added to the list of causative agents [3].

According to a study conducted by Mao et al in Wuhan, towards 70% of all cases reported in literature patients have a flu-syndrome 1 to 3 weeks before GBS discovered and as long as these symptoms are severe, most of the patients have neurologic symptoms [4]. Among these symptoms, sudden loss of smell and taste is reported in up to 60% of COVID-19 infection [5]. In contrast, our patient was almost without respiratory symptoms at the initial post-covid period.

Many studies have noted that there are epidemiological and clinical differences between GBS before and after the epidemic. Indeed, post-covid GBS infection has been reported in older patients with a mean age of 60 years compared to 40 years previously. In addition, damage to the cranial nerves was frequently observed in post-covid 19 GBS in 47% of cases versus 5% of cases [4].

The diagnosis is based mainly on history and physical examination. The classic features, found in para-clinical examinations such as the albumio-cytologic dissociation in the CSF examination and sensorimotor polyradiculoneuropathy in the electrophysical studies, are used to rule out other diagnosis particularly in a typical presentation [6].

Medullary MRI can be helpful in diagnosis, whether is normal for excluding differential diagnosis or is abnormal with leptomeningeal enhancement of nerve roots and spinal cord to confirm the diagnosis [7].

Several theories have been reported to find an explication of the relationship between GBS and COVID-19, but until our days mechanism is not clearly determined. Based on the main pathophysiology for the neurological presentation of GBS; demyelination and/or axonal damage to peripheral nerves or roots, there are two hypotheses mostly adopted: The first is the immune theory which considered that the Covid 19 virus produces inflammatory cyokines responsible for anibodes production against of GBS antigens [8]. While the second theory postulate the active retrograde transport mechanism of virus from the peripheral neuron, via the cribriform plate close to the olfacory bulb, to the brain for a direct attack on the nervous system [9].

The majority of cases of GBS post-covid reported in literature have been treated with immunoglobulin. All cases showed a favorable evolution with good prognosis, except a few cases have died by respiratory failure. Our patient died by a thromboembolic complication, despite the good neurologic recovery [5].

Ultimately, the exact prevalence of GBS post-COVID-19 is still unknown and further investigations into how the SARS-COV-2 is related to GBS are needed. Between times, GBS should be kept in mind by clinicians, one time patient has covid 19 symptoms presented moving difficulty and paresthesia to avoid complications and also to make an early treatment.

Competing Interests

All authors have no conflict of interest.

This work is original and not submitted with other publishers.

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