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CASE REPORT

Mononeuritis Multiplex Associated with Sars-Cov2-Covid-19 Infection: Case Report

Enrique Estévez-Rivera, MD[1], Javier Benavides-Hinestroza, MD[1], Hernando Rubiano, MD[2], Herney Andrés García-Perdomo, MD[2,3*]

[1]*Department of Physical Medicine and Rehabilitation, Medicine School, Universidad del Valle, Colombia*

[2]*Neurology Department, Sebastián de Belalcazar Clinic, Colombia*

[3]*Division of Urology, Department of Surgery, Universidad del Valle, Colombia*

***Corresponding author:** *Herney Andrés García-Perdomo, MD, Division of Urology, Department of Surgery, Medicine School, Universidad del Valle, Calle 4B # 36-00, Cali, Colombia, Tel: 573212195102*

Keywords

Covid-19, SARS-CoV2, Mononeuritis Multiplex

Introduction

Since the appearance coronavirus disease (COVID-19) in December 2019, the infection has reached pandemic proportions, causing globally so far (July 04, 2020), 11,185,627 confirmed cases and more than 528,232 deaths [1].

Neurotropism is one of the common characteristics described for some types of pathogenic coronaviruses such as SARS-CoV (2002) and MERS-CoV (2012) [2]. Endothelium, glial cells, and neurons have reported receptor expression for angiotensin-converting enzyme 2 (ACE2), making them a potential target for SARS-CoV-2.

Currently, there are reports of 36.4% of the neurological manifestations of COVID-19; compromising the CNS 24.8%, 8.9% the PNS, and 10.7% of the musculoskeletal system [3].

The most commonly reported condition within peripheral involvement is cases of olfactory changes (hyposmia or anosmia) or taste (ageusia), followed by Guillain-Barre Syndrome.

This report aims to describe a rare complication in a patient with a confirmed diagnosis of SARS-CoV2-COVID-19; whose clinical and electrophysiological characteristics suggest Mononeuritis Multiplex.

Case Description

We present the case of a 63-year-old female patient from Ipiales (Nariño, Colombia) with COVID-19 Pneumonia (positive PCR test on April 9, 2020, repeated on April 29, 2020, confirming the diagnosis). He was hospitalized in the ICU for 50 days and required prolonged mechanical ventilation. Significant pathological history: Hypertension and hypothyroidism. During her ICU hospitalization, she had kidney failure that required several dialysis sessions. Simultaneously, she showed persistent febrile syndrome and a neurological deficit. It mainly consisted on: distal muscle weakness (quadriparesis - without cranial nerve involvement), and global areflexia with sphincter control.

We found a conscious patient on admission, afebrile (36.5 °C), Blood pressure: 130/75 mmHg, HR: 88/min, FR: 18/min, oxygen saturation: 92%.

The neurological examination of admission reported a conscious and alert patient, cranial nerves without alterations, and no respiratory distress signs. Motor examination revealed flaccid quadriparesis with greater involvement of the lower extremities (0/5 muscle strength in the lower extremities and 2/5 in the upper extremities using the Medical Research Council - MRC scale). Predominantly distal and generalized areflexia,

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Table 1: Sensitive conductions.

Nerve	Latency* (ms)	Amplitude (uV)	Drivingspeed (m/s)
R Median Anti	**3.9** (< 3.6)	29.5 (> 10)	**36** (> 39)
R Ulnar Anti	3.1 (< 3.7)	16.4 (> 15)	45 (> 38)
R SupPeron Anti	3.7 (< 4.4)	**4.1** (> 5)	38 (> 32)
L SupPeron Anti	4.2 (< 4.4)	**4.0** (> 5)	33 (> 32)
L Sural Anti	3.8 (< 4.0)	**4.2** (> 5)	35 (> 32)

R = Right, L = Left, Anti = Antidromic, Latency* = Latency taken at the potential peak.

Normal values indicated in brackets according to the Cadwell equipment software.

Table 2: Motor conductions.

Nerve	Latency (ms)	Amplitude (uV)	Driving speed (m/s)
R Median	**4.4** (< 4.2)	**3.9** (> 5.0)	50 (> 50)
R Ulnar (Abd DigMinimi)	2.9 (< 4.2)	4.5 (> 3.0)	54 (> 53)
L Peroneal (Ext D Brev)	**No answer ***	-	-
R Peroneal (Ext D Brev)	3.9 (< 6.1)	**0.7** (> 2.5)	56 (> 38)
Tibial L (Abd Hall Brev)	5.1 (< 6.1)	**1.7** (> 3.0)	52 (> 35)
Tibial R (Abd Hall Brev)	5.3 (< 6.1)	6.6 (> 3.0)	51 (> 35)

R = Right, L = Left, Abd Dig Minimi = Abductor DigitiMinimi, Ext D Brev = Extender Digitorum Brevis, Abd Hall Brev = Abductor Hallucis Brevis, No response * = Absence of PAMC

Normal values indicated in brackets according to the Cadwell equipment software

with a bladder catheter and complete rectal sphincter control. Sensory examination showed a patchy distribution of hypoesthesia in the lower extremities and perineal region. She presented pressure ulcers in the malleolar region of the right lower extremity, the sacral region and the right scapular area, and necrotic lesions in the right hallux's pulp and dorsum. There were traces of scarring without signs of infection.

Admission laboratory tests reported: Hemoglobin 11.2 g/dl, hematocrit 35%, total leukocyte count 16,400/mm^3 (neutrophils: 60.4%, lymphocytes: 32.6%), creatinine: 0.37 mg/dl, BUN: 16.4 mg/dl, alkaline phosphatase: 93 U/l, Potassium: 3.88 mE/l, Sodium: 142 mE/l, Total protein: 7.04 g/dl, alanine aminotransferase: 29.4 U/l, aspartate aminotransferase: 30.5 U/l, procalcitonin: 0.08 ng/dl, total creatinkinase (CK): 41 umol / L, partial time of thromboplastin: 33.8 sgs (control 28.2 sgs), thrombin time: 13 sgs (control 11.9 sgs), urine culture > 100,000 Escherichia Coli colonies, ANTI-SARS-CoV-2 IgG Antibodies: Positive, ANTI-SARS-CoV-2 IgM Antibodies: Negative, detection of COVID-19 by PCR : Negative. Chest X-ray reports hyperventilated lungs without radiological signs of an acute active inflammatory process. Serial blood cultures were reported as negative.

Simple cervical and brain CT showed subcortical granulomatous calcifications at the frontal and parietal levels without inflammatory activity signs. Simple cerebral MRI showed an isolated and small hyperintense focus in the white matter. This finding was nonspecific since they are predominantly subcortical and may correspond to microangiopathic changes. Besides, we found calcifications in both cerebral hemispheres that are better visible on previous CT scan together with a small focus of malacia in the left thalamus. During the hospitalization, the patient presented pain and functional limitation in the left elbow, for which X-ray was taken, which showed an old fracture in consolidation with an enhancement of the distal humeral cortex. We perform an MRI that showed a hyperintensity in the medial and lateral epicondyle. The orthopaedics group immobilized and gave analgesia.

The electrophysiological study of three extremities (not performed on the left upper extremity due to being immobilized), performed on the 8th day of hospitalization, reported the following results (Table 1 and Table 2).

The EMG study evidenced an increased insertion activity with signs of membrane instability (fibrillations 1+ and acute positive waves 2+) in bilateral Tibialis Anterior, left gastrocnemius, extensor Hallucis Longus, with motor units. The recruitment pattern decreased in these muscles.

Discussion

To date, the complications of the peripheral nervous system most commonly associated with SARS-CoV-2 infection are olfactory and gustatory [4,5]. Also, there is a description of an acute demyelinating inflammatory polyneuropathy or a Guillain-Barre syndrome [6-8].

Mononeuritis Multiplex is a disorder that denotes injury to individual nerves that do not have a root pattern or a length-dependent pattern. Clinically, it can present as an asymmetric, predominantly sensitive asynchronous lesion or as a motor neuropathy that involves two or more separate nerves with a random distribution pat-

tern; bilateral, distal, proximal, or combinations of these [9]. Electrophysiologically, the neuro conducting studies generally show axonal neuropathy and where the side-by-side comparison shows significant asymmetry in amplitudes [10]. Mononeuritis multiplex can manifest many pathologies, including infectious, inflammatory, neoplastic, toxic, metabolic, and hereditary. However, the most common presentation pattern in neuropathies is from a vasculitic origin [11], which is why these should always be a differential diagnosis.

Vasculitis is a condition of undetermined origin. The main characteristic is the progressive occlusion of the arterial lumen until reaching its complete obstruction. It can affect a wide variety of organs, including the lungs, kidneys, skin, brain, and peripheral nerve. There is an alteration of the immune system in which it produces autoantibodies; many of them directed against multiple antigens located in the basement membrane of blood vessels. The primary autoantibodies produced are ANCA (anti-neutrophil cytoplasmic antibodies); their role is essential for the diagnostic orientation of this pathology [12].

Vasculitic neuropathy is commonly associated with systemic vasculitis, polyarteritis nodosa, or rheumatic vasculitis. It can appear in a single organ or system and, in some cases, the skin, brain, and peripheral nervous system [12]. It is characterized by the acute or subacute onset of pain and motor deficit produced by damage to the vasa nervorum and underlying ischemic injury. This type of neurological complication is prevalent in patients with primary and secondary systemic vasculitis such as rheumatoid arthritis, viral infections, and diabetes mellitus [13]. Peripheral nerve involvement is present in about 60-70% [13].

Vasculitis of large nerve arterioles results in sensory-motor disturbance, neuropathic pain, and focal weakness. These findings can affect various nerves after a few weeks.

The PNS injury, in this context, consists of a multifocal axonal neuropathy of acute or subacute onset due to ischemic infarcts in the nerve trunks. The electrophysiological patterns represent different syndromes underlying different pathophysiological mechanisms. These patterns can present as fully established lesions or, in initial cases, as partial lesions producing inaccurate interpretations [14]. Vascular involvement of a peripheral nerve produces focal axonal damage that manifests itself electrophysiologically with a reduction in the amplitude of motor or sensory action potentials, with average or poorly reduced conduction velocities. If the ischemic nerve injury covers all the fibers, there is no potential. Needle electromyography study may show signs of membrane instability with the presence of fibrillation and acute positive waves [15].

In the vascular compromise of peripheral nerves and the typical axonal neuropathy pattern, there is a conduction block. Depending on the duration of the ischemia and the time elapsed after it, axonal oedema, demyelination (which may be initially reversible) observe when the ischemia is mild and transient, up to Wallerian axonal degeneration [16].

In the case of the patient mention above, were due to complications during the hospitalization in the UCI, such as kidney failure that required dialysis associated with necrotic lesions in the pulp of the knuckles, could suggest the possibility of an underlying vasculitis process. Some reports show the presence of this type of lesions in the picture of Mononeuritis Multiplex [17]. This case differentiates from critically ill neuropathy, which tends to appear later in the course of the disease [7].

Conclusion

The report of this case highlights the possible correlation between COVID-19 infection and an alternative presentation of peripheral nervous system compromise: Mononeuritis Multiplex. Our case suggests that COVID-19 can injure the peripheral nerve even during the resolution of the pneumonic process. The disturbance of the immune system due to infection by SARS-CoV2 COVID-19 is not an inadvertent fact. We need to clarify the pathophysiology of this complication performing clinical studies.

Conflict of Interest

Nothing to declare.

Financial Source

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