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CEREBROVASCULAR ACCIDENT ASSOCIATED WITH COVID-19: EXCEPTIONAL CASE

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ABSTRACT

The coronavirus disease 2019 (COVID-19), caused by the SARS-CoV-2 virus, is causing enormous global public health problems due to its vertiginous expansion, for which it was declared by the World Health Organization (WHO) as a public health emergency of global importance and classified as a pandemic. At the beginning, the main associated symptoms were fever, cough, dyspnea, and respiratory distress. To this "common or typical clinical picture" are added the clinical manifestations that reflect the involvement of other organs or systems, including the central nervous system (CNS). SARS-CoV-2 can affect the CNS in two ways: directly and indirectly because of the immune response and / or the systemic processes that accompany this disease. The route by which SARS-CoV-2 reaches the CNS is not exactly known, the following alternatives have been proposed: hematogenous / lymphatic route, through the cribriform plate of the ethmoid bone (by contiguity) and trans- neuronal; current evidence suggests that the latter through the olfactory nerve would be the main access route. As for the neurological clinical manifestations, they are also nonspecific and possibly systemic in nature, among them are headache, myalgia, dizziness, fatigue in addition to acute cerebrovascular disease, altered consciousness, and skeletal muscle injury1,2,3,4.

Due to the importance of the subject, this special article is published, in which the most important aspects related to the neurological manifestations that appear during the SARS-CoV-2 infection are analyzed, through a clinical case.5,6

Objective

Describe the neurological involvement associated with Covid-19, presenting a clinical case.

Design

Prospective, observational. The data was collected from May 28 to September 8, 2020 in a special care center designated for COVID-19 (Hospital Enrique Garcés-Quito).

Methodology

This is a systematic review of stroke associated with COVID-19 in an adult patient, emphasizing its clinical characteristics and short-term complications.

KEYWORDS : COVID-19, Stroke, Cerebral infarction.

INTRODUCTION

Coronaviruses are encapsulated viruses and have one of the largest genomes among single-stranded, positive-sense RNA viruses, ranging in length from 26 to 32 kilobases. The term 'coronavirus' is due to the peculiar crown-shaped appearance of its envelope, visible by electron microscopy, which is surrounded by spike-shaped membrane glycoproteins. Coronaviruses belong to the subfamily Orthocoronavirinae, family Coronaviridae, order Nidovirales. Coronaviruses have remarkable genetic diversity and a high recombination capacity; This explains the interspecies jump of emerging coronaviruses that have affected humans in recent decades. SARS-CoV-2 is transmitted through the respiratory route by small droplets that are dispersed one or two meters when talking or coughing.[7,8,9.]

The mean incubation period is five days (mean range: 3-7, with a maximum of 14 days). During the viral replication phase, which lasts several days, subjects may present mild symptoms as a result of the effect of the virus and of the innate immune response. Lower respiratory tract involvement occurs when the immune system fails to stop the spread and replication of the virus and respiratory symptoms arise as a result of the cytopathic effect on lung cells.[10,11]

During this outbreak, numerous studies have described primarily respiratory and gastrointestinal symptoms, distinctive laboratory findings, and characteristic pulmonary radiological abnormalities. But recently, Mao et, made the first description (preprint) in February 2020 (later published JAMA Neurology in April), focusing on the neurological manifestations of SARS-CoV-2 infection.[12]

Nowadays, it is unknown if the cerebrovascular accidents (CVA) related to SARS-CoV-2 are due to a direct effect of the virus or indirect, related to the prothrombotic effect of the inflammatory response. Given that the CNS is not exempt from commitment by the new coronavirus, we describe a case of ischemic stroke associated with COVID-19.

CASE PRESENTATION

This is a 56-year-old male patient with a history of atrial fibrillation, hypothyroidism, and depression. Usual treatment: bisoprolol, digoxin, levothyroxine and fluoxetine. Symptoms started 5 days prior to hospital admission, characterized by intense holocranial headache, dry cough, and left upper limb

weakness. Upon admission for emergency: vital signs: BP: 140 / 80mmHg, HR: 110 bpm, RR: 26 rpm; Basal saturation 88%. On neurological examination, he was found drowsy, disoriented in time, space and person, with non-proportional left hemiparesis 4/5, left hemihypoesthesia, left Babinski.

Arrhythmic heart sounds, bilateral basal crackles lung auscultation. Extension tests: the electrocardiogram (ECG) atrial fibrillation, cerebral tomography targeted cerebral infarction in the right MCA territory, and at the temporo-occipital level (Photo 1), the RT-PCR for SARS-CoV-2 in a nasopharyngeal swab sample was positive. The study was complemented with blood tests highlighting lymphopenia (755 / ul), elevated DHL (420 U / L), elevated D-dimer (12.6 ug / ml), high CRP (26.9 mg / dl).

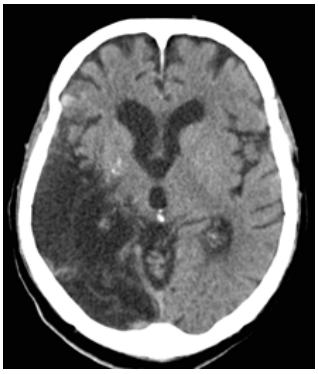

Photo 1. Skull CT: Cerebral Infarction In The Territory Of The Right Mca And At The Temporo-occipital Level

The simple chest tomography showed a ground-glass pattern (Photo 2).

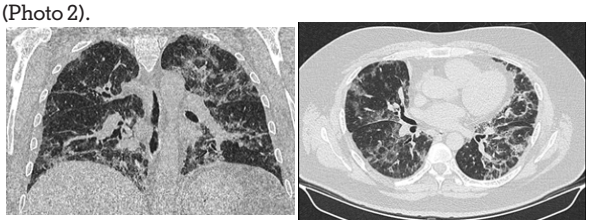

Photo 2. Chest CT: Coronal, Axial: Bilateral Diffuse Ground Glass Opacity, Likewise Thickening Of The Axial Peribroncovascular Interstitium.

Treatment with oxygen therapy support is prescribed, associated with hydroxychloroquine and azithromycin, in addition to ceftriaxone due to suspected superinfection with bacterial pneumonia in the community. (due to leukocytosis with neutrophilia in the hemogram and elevated CRP). During its evolution, it presents signs of respiratory distress, tachypneic, use of accessory muscles and saturations of 60% ambient air, gasometry shows mixed acidosis (ph 7.17 pco2 43.4 po2 63.3 hco3 15.5 pafi 79.1 gsa ph 7.17, po2 63.3, pco2 43.4, eb -12.4, hco3 15, glucose 327, lact 1.4, sat 85%, pa / fi 79), which required management in intensive care, mechanical ventilation with parameters: Pressure PC 16, PEEP 12, FIO2 100%, FR 32, I present hypotensions marked 60 / 40mmHg, diaphoresis, tachypnea, use of accessory muscles. Consecutively it presents refractory desaturation to increase of FIO2; The patient progresses to bradycardia and the central and peripheral pulse is not palpable. Upon monitoring without reading the electrocardiographic leads, the patient dies, 15 days after admission.

DISCUSSION

Ischemic strokes have been related to infections by different viruses, highlighting those described in severe disease caused by SARS-CoV, since it presents a similarity in its genomic sequence of 82% with SARS-CoV-2.[13,14]

The series of cases of ischemic stroke and COVID-19 with the highest number of patients is that published by Yanan Li et al; the authors conducted a retrospective study in a Wuhan hospital; of 221 patients with COVID-19, 13 patients (5.9%) had stroke, of which 11 were ischemic. These patients were mostly older adults and with a higher prevalence of vascular risk factors (high blood pressure, diabetes, or smoking) compared to patients without stroke. In addition, these patients developed severe COVID-19. Since March 25, our hospital has received patients with covid-19, of these, 35% had ischemic stroke. In patients with severe and critical COVID-19, more cases of stroke are reported. A pooled analysis of 4 studies showed an increased risk of developing severe SARS-CoV-2 disease in patients with cerebrovascular attacks. In Li et al's case series, 10 of the 11 patients who developed ischemic stroke had severe COVID-19. In an intensive care unit (ICU) of a French hospital, out of 58 patients admitted, 3 patients (5.1%) developed acute cerebral infarcts. In different studies of patients of Chinese origin with COVID-19, the presence of lymphopenia, increased DHL, elevated D-dimer and CRP have been described as markers of poor prognosis. Inflammation is also increasingly recognized as a key contributor to the pathophysiology of cerebrovascular disease. In our patient, these markers of poor prognosis were present, the same that led to his death.[15,16,17]

CONCLUSIONS

The difficult task of the international scientific community to stop the spread and mortality of covid-19 is full of questions. It is vitally important to delve into this subject in order to define the value of the damage that SARS-CoV-2 sepsis can cause in the nervous system and its repercussion on the complications and evolution of the disease; but at the same time, it is essential to alert the medical community to the possibility that alterations of the central and peripheral nervous system may occur in patients affected by covid-19.

CONFLICT OF INTERESTS

The authors declare that they have no conflict of interest.

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