MEDDOCS Open Access Publisher

ISSN: 2639-9237

Journal of Case Reports and Medical Images

Open Access | Case Report

Rapid Progression of Cerebral Atrophy Associated with SARS-Cov-2 Infection: Case Report

Azin Aein[1]; Shekhar Khanpara[1]; Luis C Nunez-Rubiano[1]; Lilit A Sargsyan[2]; John A Lincoln[3]; Roy F Riascos[1]*

[1]Department of Diagnostic and Interventional Imaging.

[2]Department of Internal Medicine.

[3]Department of Neurology, McGovern School of Medicine, University of Texas Health Science Center, Houston, Texas, USA.

***Corresponding Author(s): Roy Riascos**

Radiology & Neurosurgery, Department of Diagnostic and Interventional Imaging, The University of Texas Health Science Center at Houston, McGovern medical school 6431 Fannin Street MSB 2130B, Houston, TX, USA.

Tel: +1-713-704-1704; Email: Roy.F.Riascos@uth.tmc.edu

Received: Feb 10, 2023

Accepted: Mar 02, 2023

Published Online: Mar 06, 2023

Journal: Journal of Case Reports and Medical Images

Publisher: MedDocs Publishers LLC

Online edition: http://meddocsonline.org/

Keywords: COVID-19; Cerebral Atrophy; Encephalopathy; Brain.

Abbreviations: COVID-19: Coronavirus disease 2019; SARS-CoV-2: Severe acute respiratory syndrome coronavirus 2; CNS: Central nervous system; MRI: Magnetic resonance imaging; CT: Computed tomography; DWI: Diffusion-weighted imaging; SWI: Susceptibility weighted imaging; WHO: World Health Organization.

Abstract

COVID-19 is nowadays understood as a systemic disease rather than an acute respiratory infection. Neurotrophic properties have been attributed to this virus, generating great concern due to its long-lasting consequences. Hereby we present the first case of rapid progression of cerebral atrophy associated with SARS-CoV-2 infection. A middle-aged adult with recent history of SARS-CoV-2 pneumonia presented with altered mental status. Metabolic and infectious causes were ruled out, imaging studies revealed cerebral atrophy. Follow up MRI demonstrated a significant decrease in cerebral volume. Diffuse leukoencephalopathy secondary to SARS-CoV-2 infection might be related to brain parenchymal atrophy.

Introduction

Since the WHO's March 11th 2020 declaration of COVID-19 outbreak as a pandemic, a growing number of neurologic manifestations of this disease are continuously being reported. Besides the wide variety of clinical features, radiological manifestations such as cerebrovascular diseases, inflammatory processes and cranial nerve affection have been described [1]. Because neurological complications can be particularly devastating in young and middle-aged population given their extended duration of disability, it is important to recognize the association between SARS-CoV-2 and the nervous system. Herein we report a case of generalized brain parenchymal volume loss following recent COVID-19 pneumonia, presenting with altered mental status and rapid deterioration.

Cite this article: Aein A, Shekhar K, Nunez-Rubiano LC, Sargsyan LA, Lincoln JA et, al. Rapid Progression of Cerebral Atrophy Associated with SARS-Cov-2 Infection: Case Report. J Case Rep Clin Images. 2023: 6(1): 1133.

Case report

A 41-year-old African-American man with history of recent COVID-19 pneumonia presented to the emergency department with altered mental status. The patient was found to be obtunded and hypoglycemic, and intubation was indicated. Medical history was significant for rheumatoid arthritis, gout, hypertension, poorly controlled type 2 diabetes mellitus, chronic congestive heart failure, and end stage renal disease on peritoneal dialysis that began 4 months before presentation. He was hospitalized two months earlier due to debilitating SARS-CoV-2 pneumonia that required non-invasive mechanical ventilation (bilevel positive airway pressure).

Upon arrival, the non-contrast head CT showed no acute intracranial hemorrhage or infarction, and no white matter abnormalities were documented. Electroencephalogram was consistent with moderate to severe diffuse encephalopathy without epileptiform activity or seizures. Cerebrospinal fluid analysis and extensive blood work-up were unremarkable. The patient received an empiric course of acyclovir, and hemodialysis was indicated; however, there was no clinical improvement.

The follow up brain MRI one week later showed confluent areas of restricted diffusion within periventricular and deep white matter of bilateral frontoparietal lobes **(Figure 1 A, B)**. Two-week follow-up MRI showed a new T2 hyperintensity involving bilateral hippocampi with T2 shine through. New areas of microhemorrhages were seen within the splenium of the corpus callosum and the left periatrial white matter **(Figure 1 C, D)**. Persistent restricted diffusion was again observed.

Follow-up MRI 2.5 months after initial presentation demonstrated persistence of the previously observed microhemorrhages, as well as the confluent and symmetrical DWI/T2 signal hyperintensities within periventricular and deep white matter. Figure 2 demonstrates evolution of cerebral atrophy over the 2.5-month interval. Brain parenchymal volume was calculated on 2D T2-weighted images using the TeraRecon Aquarius software 4.4.12.194, (TeraRecon, Foster City, CA). A decrease of 8.2% of brain tissue occurred from the first to the second scan, and a decrease of 15.2% was observed when comparing the initial scan with the third one **(Figure 2)**.

Discussion

Various theories attempt to explain the spectrum of neuroimaging findings among COVID-19 patients. Endotheliitis with thrombotic microangiopathy, in association with COVID-19 cytokine release syndrome has been postulated as the cause of white matter lesions. Given the imaging similarities to carbon monoxide poisoning and cardiopulmonary arrest, hypoxic injury may play an important role [2]. Moreover, the presence of microhemorrhages observed in high-altitude cerebral edema supports the theory of a hypoxic insult [3].

White matter lesions are usually diffuse, symmetrical, and localized among supratentorial subcortical, periventricular and deep white matter [2]. Microhemorrhages in these patients tend to occur in the corpus callosum and juxtacortical white matter [4]. Brain atrophy might represent a consequence of delayed hypoxic-ischemic leukoencephalopathy (DHIL). DHIL has been described in patients with prolonged hypoxemia and cerebral hypoperfusion, with imaging findings that resemble the ones of this patient [5].

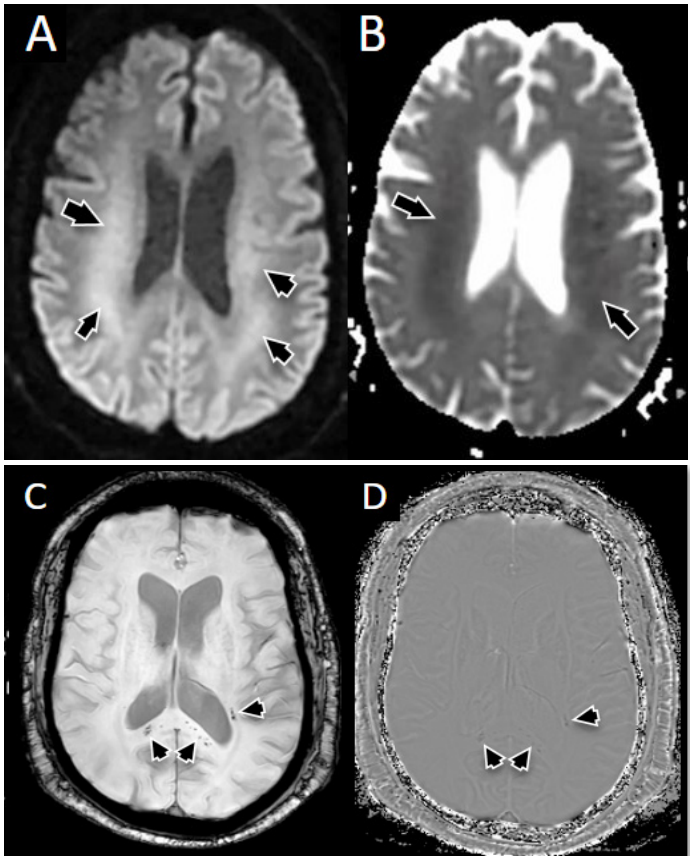

Figure 1: One week follow up MRI (A, B): Axial DWI **(A)** and ADC map showed areas of restricted diffusion within periventricular and deep white matter of bilateral frontal and parietal lobes (black arrows). 2-week follow up MRI (C,D): Axial SWI (C) and phase (D) showed the new areas of microhemorrhages within the splenium of corpus callosum and left periatrial white matter (black arrowheads).

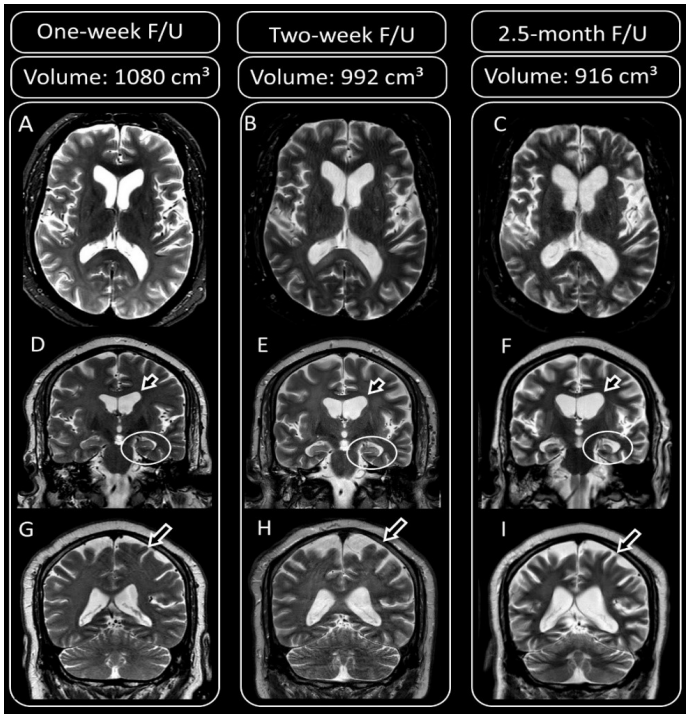

Figure 2: Comparison among three different MRI scans; axial T2-weighted images (**A,B, and C**), coronal T2-weighted images at the level of hippocampi **(D,E, and F)**, and coronal T2-weighted images more posterior at the level of cerebellum **(G,H, and I)** show increasing prominence of lateral ventricle and the cerebral sulci during the time which is consistent with generalized cerebral atrophy.

To our knowledge, this is the first case to document rapidly progressive cerebral atrophy associated with COVID-19 infection. Even though this patient had known risk factors for small vessel disease, such a progression of brain atrophy is not expected in a short period of time. Due to the lack of dementia history and the fact that extensive work-up ruled-out a different etiology, we hypothesize that delayed diffuse leukoencephalopathy might lead to cerebral atrophy.

Conclusion

Neurological manifestations of COVID-19 represent an emerging concern of worldwide proportions. Recognizing consequences of SARS-CoV-2 infection will result in a better understanding and approach of the disease. We hypothesize that cerebral atrophy may represent a delayed consequence, that must be considered for further evaluation.

Funding: None.

Previous Presentation: None.

Ethical considerations: The patient's health care power of attorney provided written informed consent for publication of the data.

Disclosure of relationships/potential conflicts of interest

The authors have no personal, financial, or institutional interest in the materials or devices described in this manuscript.

Acknowledgment

The authors thank Winston Zingg-De Jongh for editorial assistance.

References

1. Ladopoulos T, Zand R, Shahjouei S, Chang JJ, Motte J, et al. COVID-19: Neuroimaging Features of a Pandemic. Journal of Neuroimaging. 2021; 31: 228-243.
2. Radmanesh A, Derman A, Lui YW, Raz E, Loh JP, Hagiwara M, et al. COVID-19 -associated Diffuse Leukoencephalopathy and Microhemorrhages. Radiology. 2020; 202040.
3. Schommer K, Kallenberg K, Lutz K, Bärtsch P, Knauth M. Hemosiderin deposition in the brain as footprint of high-altitude cerebral edema. Neurology. 2013; 81: 1776-1779.
4. Fitsiori A, Pugin D, Thieffry C, Lalive P, Vargas MI. COVID-19 is Associated with an Unusual Pattern of Brain Microbleeds in Critically Ill Patients. J Neuroimaging. 2020; 30: 593-597.
5. Gulko E, Oleksk ML, Gomes W, Ali S, Mehta H, Overby P, et al. MRI Brain Findings in 126 Patients with COVID-19: Initial Observations from a Descriptive Literature Review. American Journal of Neuroradiology 2020; 41: 2199-2203.