Case Report

URF PUBLISHERS
connect with research world

American Journal of Psychology and Brain Studies

https://urfpublishers.com/journal/american-psychology

Vol: 2 & Iss: 3

Multiple Arterial and Cerebral Venous Thrombosis in the Setting of Covid-19 Infection: A Case Report

Running title: Cerebrovascular disturbances and Covid-19

Usaamah Khan[1], Hasti Izadpanah[1], Taylor Haight[1], Maha Alattar[1], Marco Aurelio M. Freire[2] and Daniel Falcao[1]*

[1]VCU Health Systems, Virginia Commonwealth University, Richmond 23284 VA, USA

[2]Graduate Program of Natural Sciences, Federal University of Sergipe, Itabaiana 49510-000 SE, Brazil

Citation: Khan U, Izadpanah H, Haight T, et al. Multiple Arterial and Cerebral Venous Thrombosis in the Setting of Covid-19 Infection: A Case Report. *Am J Psychol & Brain Stud*, 2025;2(3):150-154.

Received: 21 August, 2025; **Accepted:** 27 August, 2025; **Published:** 29 August, 2025

***Corresponding author:** Daniel Falcao, DO, FAAN, Department of Neurology, VCU Health Systems, Virginia Commonwealth University, Richmond 23284 VA, USA

ABSTRACT

The world faced a pandemic caused by Coronavirus disease 2019 (Covid-19), which emerged at the end of 2019 as an outbreak in China. Besides its typical clinical symptoms (fever, dry cough, fatigue and shortness of breath), Covid-19 has been associated with neurological manifestations such as headache, migraine, encephalitis and cerebrovascular accidents, with an increase in ischemic stroke patients and concurrent Covid-19 being reported. Such a scenario requires further investigation for a better understanding of its impact on the nervous system. We present a case of a 64-year-old male patient with a past medical history of diabetes, hypertension, coronary artery disease and prior deep venous thrombosis and pulmonary embolism provoked in a setting of knee surgery admitted in the hospital with shortness of breath, nausea and fever, who was diagnosed with multiple arterial occlusions and a cerebral venous thrombosis associated with Covid-19. The present study reinforces the notion that vascular damage seems to be directly associated with SARS-CoV-2 infection. The underlying mechanisms of virus action on the brain vasculature are yet to be properly characterized.

Keywords: COVID-19; SARS-CoV-2; Pandemic; Cerebrovascular accident; Cerebral thrombosis

Introduction

In December 2019, the first cases of coronavirus disease 2019 (Covid-19), caused by the novel severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), were reported in Wuhan, Hubei province, China. Afterward, there was the rapid spread of the outbreak, resulting in the most terrifying global pandemic faced since the Spanish flu in 1918[1]. On January 30th, 2020, Covid-19 was declared a Public Health Emergency of International Concern by the World Health Organization (WHO)[2].

To date, Covid-19 affected almost 780 million people globally, with more than seven million deaths had been recorded, with the highest number of notifications observed in the United States, China, India, France, Germany and Brazil[3], with a significantly higher mortality rate in the aged and in people with previous comorbidities[4]. In addition to commonly described SARS-Cov-2 clinical symptoms (fever, dry cough, fatigue and shortness of breath), a growing number of neurological disturbances such as acute cerebrovascular disease, meningitis, migraine and encephalitis associated with Covid-19 have been reported[5-9]. An increase in both morbidity and mortality of patients with ischemic stroke and concurrent Covid-19 has also been observed[10].

Currently, long Covid presents as a significant concern, remaining a global issue and imposing a substantial burden on healthcare systems. According to the Office for National Statistics (ONS), approximately 1.9 million individuals in the United Kingdom, equivalent to around 2.9% of the population, reported experiencing long Covid symptoms as of March 2023. Fatigue emerges as the predominant and debilitating symptom, emphasizing the critical need for effective treatment options while patients await relief[11]. A notable proportion of SARS-CoV-2 infections in the general population lead to persistent infections lasting a month or more[12], with evidence indicating their persistence in the brain for prolonged periods[13].

Two main hypotheses explaining how SARS-CoV-2 affects the nervous system have arisen: 1. The virus directly infects the nervous system; or 2. The virus disturbs the nervous system through its systemic effects on the entire body (see[14]). In the light of this, further investigation into the neurological complications of SARS-Cov-2 infection has become even more important. In the present report, we present a case of an elderly patient who suffered multiple arterial occlusions and cerebral venous thrombosis associated with Covid-19.

Case Description

A 64-year-old Caucasian male with a past medical history of diabetes, hypertension, coronary artery disease and prior deep vein thrombosis and pulmonary embolism provoked in a setting of knee surgery initially presented with shortness of breath, nausea and fever (38.4ºC), being admitted for work up for sepsis **(Table 1)**. Chest X-ray scan showed peripheral ground-glass opacities, worse in the right lung field, in a pattern consistent with acute respiratory distress syndrome **(Figure 1)**.

Table 1: Patient demographics and presentation.

Characteristics	Patient
Age	64
Sex	Male
Ethnic group	Caucasian
Past medical history	Diabetes, Hypertension, CAD, Provoked DVT
Initial symptoms	Shortness of breath, fever
Neurological deficits	Day 4: Right arm and leg weakness, right facial droop, dysarthria Day 7: Worsening right arm and leg weakness, right facial droop, dysarthria, left arm weakness
Disposition	Rehabilitation

CAD: Coronary artery disease; DVT: Deep vein thrombosis.

His oxygen requirement escalated to 10 liters via nasal cannula and the patient was diagnosed with Covid-19 after real-time reverse-transcription polymerase chain reaction (qRT-PCR) examination **(Table 2)**.

On Day 4 of admission, the patient developed acute onset of slurred speech, right facial droop and right arm and leg weakness with an initial National Institute of Health Stroke Scale (NIHSS) of 9. Urgent non-contrast computed tomography (CT) head imaging demonstrated a left hyper dense middle cerebral artery sign **(Figure 2)**.

The patient was not a candidate for pharmacological thrombolytic therapy given concurrent melena and thrombocytopenia with a platelet count of 54,000 **(Table 2)**. Computed tomography angiogram (CTA) of the head and neck demonstrated a left posterior cerebral artery (PCA) P2 occlusion as well as multi-focal stenosis of the left PCA **(Figure 2)**.

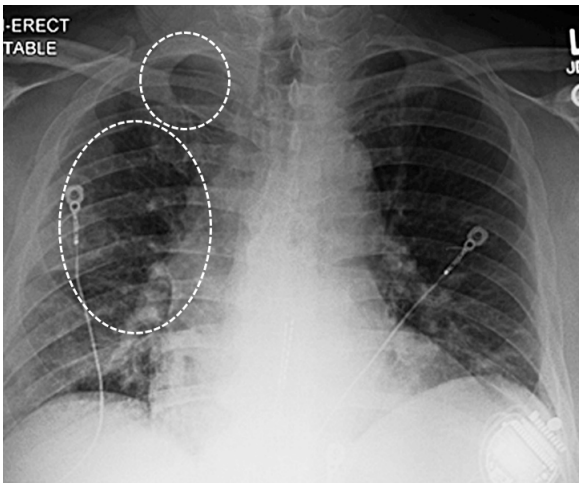

Figure 1: Chest X-Ray showing lung parenchyma diminished with ongoing basilar hypoaeration. Predominantly peripheral groundglass hazy opacities, worse in the right lung field (outlined by the dashed lines), are typical for induced-Covid-19 pneumonia.

Table 2: Pertinent lab findings.

Test	Result
Covid-19 qRT-PCR	Detected
D-dimer	2.43 mcg/mL
PT/PTT/INR	14.7 sec / 31 sec / 1.1
Haptoglobin	349 mg/Dl
C-Reactive protein	10.0 mg/Dl
Creatine kinase	2863 units/L
Ferritin	974 ng/Ml
Lactate dehydrogenase	387 units/L
Hemoglobin A1C	9.6%
Total cholesterol	126 mg/dL
Low density lipoprotein (LDL)	85 mg/dL
Triglyceride	104 mg/dL
Platelet count	54,000

qRT-PCR: Quantitative reverse transcription PCR; PT: Prothrombin time; PPT: Partial prothrombin time; INR: International normalized ratio.

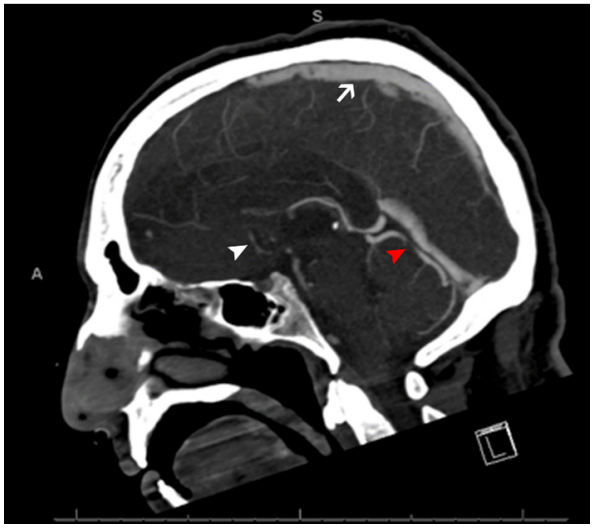

Figure 2: Computed tomography showing neurologic alterations in the elderly patient with Covid-19 pneumonia. Computed tomography venography (CTV) shows extensive occlusion in superior sagittal Sinus

(white arrow). Computed tomography angiography (CTA), showing both the Anterior cerebral artery (ACA) A2 occlusion (white arrowhead) and Posterior cerebral artery (PCA) P2 occlusion (red arrowhead).

He was a candidate for mechanical thrombectomy and was started on aspirin for secondary prevention. For the treatment of Covid-19 pneumonia, he received convalescent plasma, remdesivir and dexamethasone and his respiratory status remained stable with supplemental oxygen via a nasal cannula. However, he was unable to undergo a Magnetic Resonance Imaging (MRI) brain scan to further assess ischemia due to the presence of a spinal cord stimulator.

On Day 7 of admission, the patient experienced a significant change in his neurological examination, with worsening right-sided weakness and the development of new left-sided weakness. Repeat imaging, including non-contrast CT, CTA of the head and neck and CT venography (CTV), revealed multifocal ischemic areas, including occlusions in the left ACA A3 segment, left PCA P2 segment and the anterior portion of the superior sagittal sinus **(Figure 2)**. A transthoracic echocardiogram showed no intra-cardiac thrombus and the left ventricular ejection fraction was normal at 55%. Anticoagulation therapy was initially withheld due to thrombocytopenia, but by hospital day 14, as the platelet count had normalized, the patient was started on apixaban to treat Covid-19-related hypercoagulable disease. The patient showed continued improvement in respiratory status and was eventually discharged on 3L nasal cannula to a rehabilitation facility, with plans for outpatient follow-up in the stroke clinic.

Discussion

In this report, we present the case of an elderly patient with multiple medical comorbidities who developed multiple arterial occlusions and cerebral venous sinus thrombosis associated with a Covid-19 infection.

The hypercoagulable state linked to SARS-CoV-2 was identified early on, as the prevalence of thrombosis and cerebrovascular ischemic events was notably higher in this population compared to critically ill patients without Covid-19. Initially, the cause of this hypercoagulable state was not well understood, with early theories suggesting either resistance to fibrinolysis or an increased thrombotic potential[15]. As the mechanisms of SARS-CoV-2 infection became clearer, so did the understanding of its role in thrombus formation.

The virus itself is a positive-sense RNA virus with an envelope, glycoprotein and spike protein[16]. The spike protein of the virus is crucial for its ability to cause significant respiratory infections. This protein binds to the receptor-binding domain of the angiotensin-converting enzyme 2 (ACE2) receptor, expressed on type II pneumocytes in the respiratory tract and also present in other body sites, including endothelial cells of blood vessels[17]. When the virus binds to the receptor and begins to replicate, this leads to a prothrombotic state[18]. Damage to these endothelial cells and viral replication cause platelet adhesion to vascular proteins as a repair mechanism, triggering inflammation and the clotting cascade[17].

Many patients with SARS-CoV-2 infection have been observed to have elevated levels of biomarkers such as D-dimer and fibrinogen, indicating a hypercoagulable state and increased risk of clot formation. Notwithstanding, non-survivors have more frequently been found to have elevated D-dimer levels combined with low fibrinogen, which is indicative of conditions more consistent with consumptive coagulopathies, such as sepsis-induced coagulopathy (SIC) and disseminated intravascular coagulation (DIC)[17]. This suggests that multiple mechanisms may contribute to the increased clot formation observed in these patients. Additionally, many individuals already have other risk factors for thrombosis, such as obesity and advanced age[19]; some may also possess a genetic predisposition to clotting. Furthermore, in the context of severe illness, many patients are often immobilized[20]. Moreover, elevated levels of these biomarkers suggest that blood clots may contribute to post-Covid cognitive issues. Fibrinogen may directly affect the brain and its blood vessels, while elevated D-dimer levels often indicate lung blood clots, potentially causing brain oxygen deprivation[21]. Consistent with this, individuals presenting elevated D-dimer levels are at a higher risk for both brain fog and respiratory complications. Therefore, SARS-CoV-2 infection may potentially contribute to lasting cognitive disturbances[21].

When patients develop SIC or DIC, this deposition of small fibrin thrombi in the small vessels, while platelets and clotting factors are rapidly consumed. In the setting of SARS-CoV-2 infection, this thrombotic microangiopathy is complicated by the additionally present endotheliopathy[18].

Our patient initially presented with an arterial occlusion, which was in line with his hypercoagulable state due to SARS-CoV-2 infection. After 7 days, his condition worsened, leading to multiple arterial occlusions and cerebral venous thrombosis. This progression aligns with the timelines reported in other studies, which have noted an increase in D-dimer levels and a decrease in fibrinogen levels up to around day 10. This pattern is consistent with a consumptive coagulopathic state that heightens the risk of clot formation, in addition to the endotheliopathy observed at admission[22]. Cerebrovascular complications observed even in otherwise healthy and young patients can be explained by this state which can lead to clotting through two potential mechanisms.

The effects of SARS-CoV-2 infection are reported to be significantly more severe in aged individuals compared to younger ones. This increased severity is related to a higher expression of the mediators required for viral entry into the airways and alveolar epithelium in the elderly, particularly the elevated levels of ACE2 and Transmembrane Serine Protease 2 (TMPRSS2)[19]. Also, smoking, hypertension, diabetes and pre-existing coagulation disorders contribute to a higher incidence of vascular damage in elderly individuals affected by SARS-CoV-2 infection. This results in a higher mortality rate within this group[23], raising concerns among healthcare professionals and causing a significant impact on society worldwide[24].

Following the critical phase of the pandemic, characterized by a peak in fatalities, a central question emerges: what are the lasting effects of SARS-CoV-2 infection on the body? Concerning brain function, the term “brain fog”, though not clinically defined, is utilized to encompass a spectrum of symptoms including reduced concentration, slower thinking, confusion, forgetfulness and mental fatigue after Covid-19 infection. Healthcare specialists express a shared concern about the potential long-term impacts of Covid-19 on cognitive function[25,26]. Nevertheless, accurately gauging these effects

in a sizable population has proven challenging until now, underscoring the need for future studies to address this issue.

Conclusions and Future Prospects

The present report is in congruence with studies that emerged during the acute phase of Covid-19 pandemic evaluating the direct action of SARS-CoV-2 infection on the nervous system[27-29] (see[30] for a review), including neuroinflammatory processes following viral infection[31,32]. As a future step, further investigation monitoring the recovery condition of Covid-19 patients will be informatively important for an understanding of the dynamics of the long-term impact caused by the infection on the brain, in order to assess the relationship of SARS-CoV-2 infection with the prognosis of increase of late neurological disorders in these patients, with special attention on the brain vasculature disturbances.

Declarations

Ethics approval and consent to participate

The study was conducted according to the guidelines of the Declaration of Helsinki. Ethical review and approval were waived for this work, due to the nature of study (Case report). Written informed consent was obtained from the patient to report his clinical information in the manuscript.

Consent for publication

Not applicable.

Availability of data and materials

The authors confirm that the data supporting the findings of this study are available within the manuscript, which are available on request from the corresponding author.

Conflicts of interest

The authors declare the inexistence of any financial interests or other dual commitments that represent potential conflicts of interest related to the present manuscript.

Funding

MAMF was a recipient of a CAPES Brazil fellowship (Finance code 001).

Authors' contributions

Conceived and planned the study: DF; Wrote the manuscript: UK, HI, TH, MA, MAMF, DF; Approved the final version: UK, HI, TH, MA, MAMF, DF.

Acknowledgements

We would like to thank the patient involved in the study. The authors dedicate this study to all persons who lost the fight against Covid-19 all over the world and their relatives.

References

1. Ashton J. COVID-19 and the 'Spanish' flu. J R Soc Med 2020;113:197-198.
2. Sohrabi C, Alsafi Z, O'Neill N, et al. World Health Organization declares global emergency: A review of the 2019 novel coronavirus (COVID- 19). Int J Surg 2020;76:71-76.
3. COVID-19 Coronavirus Pandemic [online]. Available at: https://data.who.int/dashboards/covid19/cases?n=o
4. Zheng Z, Peng F, Xu B, et al. Risk factors of critical & mortal COVID-19 cases: A systematic literature review and meta-analysis. J Infect 2020;81:16-25.
5. Ahmed MU, Hanif M, Ali MJ, et al. Neurological manifestations of COVID-19 (SARS-CoV-2): a review. Front Neurol 2020;11:518.
6. Helms J, Kremer S, Hamid M, et al. Neurologic features in severe SARS-COV-2 infection. N Engl J Med 2020;382:2268-2270.
7. Mao L, Jin H, Wang M, et al. Neurologic manifestations of hospitalized patients with coronavirus disease 2019 in Wuhan, China. JAMA Neurol 2020;77:683-690.
8. Naz S, Hanif M, Haider MA, Ali MJ, Ahmed MU, Saleem S. Meningitis as an initial presentation of COVID-19: A case report. Front Public Health 2020;8:474.
9. Singh J, Ali A. Headache as the presenting symptom in 2 patients with COVID-19 and a history of migraine: 2 Case Reports. Headache 2020;60:1773-1776.
10. de Havenon A, Ney JP, Callaghan B, et al. Impact of COVID-19 on outcomes in ischemic stroke patients in the United States. J Stroke Cerebrovas Dis 2021;30:105535.
11. Office for National Statistics (ONS), released 30 March 2023, ONS website, statistical bulletin, Prevalence of ongoing symptoms following coronavirus (COVID-19) infection in the UK: 30 March 2023.
12. Ghafari M, Hall M, Golubchik T, et al. Prevalence of persistent SARS-CoV-2 in a large community surveillance study. Nature 2024;626:1094-1101.
13. Stein SR, Ramelli SC, Grazioli A, et al. SARS-CoV-2 infection and persistence in the human body and brain at autopsy. Nature 2022;612:758-763.
14. Xie Z, Hui H, Zhao Z, et al. Nervous system manifestations related to COVID-19 and their possible mechanisms. Brain Res Bull 2022;187:63-74.
15. Weiss E, Roux O, Moyer JD, et al. Fibrinolysis resistance: A potential mechanism underlying COVID-19 coagulopathy. Thromb Haemost 2020;120:1343-1345.
16. Alanagreh L, Alzoughool F, Atoum M. The Human Coronavirus Disease COVID-19: Its origin, characteristics and insights into potential drugs and its mechanisms. Pathogens 2020;9:331.
17. Biswas S, Thakur V, Kaur P, Khan A, Kulshrestha S, Kumar P. Blood clots in COVID-19 patients: Simplifying the curious mystery. Med Hypotheses 2021;146:110371.
18. Connors JM, Levy JH. COVID-19 and its implications for thrombosis and anticoagulation. Blood 2020;135:2033-2040.
19. Previtali E, Bucciarelli P, Passamonti SM, Martinell I. Risk factors for venous and arterial thrombosis. Blood Transfus 2011;9:120-138.
20. Willyard C. Coronavirus blood-clot mystery intensifies. Nature 2020;581:250.
21. Taquet M, Skorniewska Z, Hampshire A, et al. Acute blood biomarker profiles predict cognitive deficits 6 and 12 months after COVID-19 hospitalization. Nat Med 2023;29:2498-2508.
22. Eljilany I, Elzouki AN. D-Dimer, Fibrinogen and IL-6 in COVID-19 Patients with Suspected Venous Thromboembolism: A Narrative Review. Vasc Health Risk Manag 2020;16:455-462.
23. Mendes A, Herrmann FR, Genton L, et al. Incidence, characteristics and clinical relevance of acute stroke in old patients hospitalized with COVID-19. BMC Geriatr 2021;21:52.
24. Freire MAM, Khan U, Falcao D. COVID-19 and stroke: perspectives and concerns. Health Sci J 2020;10:4-5.
25. Hampshire A, Azor A, Atchison C, et al. Cognition and Memory after Covid-19 in a Large Community Sample. N Engl J Med 2024;390:806-818.

26. Al-Aly Z, Rosen CJ. Long Covid and Impaired Cognition - More Evidence and More Work to Do. N Engl J Med 2024;390:858-860.

27. Saitta L, Molin A, Villani F, et al. Brain microvascular occlusive disorder in COVID-19: a case report. Neurol Sci 2020;41:3401-3404.

28. Sharifi-Razavi A, Karimi N, Zarvani A, Cheraghmakani H, Baghbanian SM. Ischemic stroke associated with novel coronavirus 2019: a report of three cases. Int J Neurosci 2020;17:1-5.

29. Hingorani KS, Bhadola S, Cervantes-Arslanian AM. Covid-19 and the brain. Trends Cardiovasc Med 2022;32:323-330.

30. Tsivgoulis G, Palaiodimou L, Zand R, et al. COVID-19 and cerebrovascular diseases: a comprehensive overview. Ther Adv Neurol Disord 2020;13:1756286420978004.

31. Welberg L. COVID-19 and brain aging. Nat Neurosci 2023;26:1.

32. Raza ML, Imam MH, Zehra W, Jamil S. Neuro-inflammatory pathways in COVID-19-induced central nervous system injury: Implications for prevention and treatment strategies. Exp Neurol 2024;382:114984.