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Case Report

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Fatal Ischemic Stroke in a Patient with Mild Covid-19 Infection

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Abstract

Coronavirus disease 2019 (COVID-19) is a worldwide pandemic. It is currently established that patients with COVID-19 have a high risk of thromboembolic complications such as acute ischemic stroke which emerge as a serious complication of infection by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). In this sense, we present the case of a 68 year old male with a history of mitral valve replacement that developed a devastating acute ischemic stroke in multiple vascular territories in the setting of mild COVID-19 infection despite adherence to therapeutic anticoagulation. It is likely that multiple mechanisms are involved in stroke related to COVID-19. Although, vascular inflammation and coagulopathy are emerging as features defining pathology underlying COVID-19. Given the failure of anticoagulants in the prevention of ischemic strokes in some cases, despite an optimized anticoagulation strategy, more research is required to explore new methods of anti-thrombotic and anti-inflammatory therapy for stroke prevention in patients diagnosed with COVID-19.

Keywords: Coronavirus disease 2019 • Acute ischemic stroke • SARS-CoV-2 • Vasculitis • Anticoagulant

Introduction

With more than 500 million cases worldwide, COVID-19 was declared as a pandemic by the World Health Organization in March 11th 2020 and had an unprecedented global outreach. Although the disease was associated first with respiratory dysfunction, increasing reports of neurological complications were reported, including acute cerebrovascular events, mainly ischemic; encephalopathies; dysimmune syndromes of the central nervous system; Guillain-Barre syndrome; and various attacks of cranial or peripheral nerves. These manifestations may reflect either direct viral infection or dysregulation of the immune response which converge in hyper inflammation processes and dysfunction of the coagulation system. Stroke is still the most common neurological complication of COVID-19 which may have distinct characteristics in terms of disease mechanism, but also in terms of clinical and radiological specificities with implication for diagnosis and treatment. In this case report, we present a COVID-19 patient who developed a devastating ischemic stroke in multiple vascular territories during hospitalization.

Case Report

We report a 68 years old male with past medical history of mitral valve replacement complicated by atrial fibrillation for which he was on warfarin. He was admitted to the hospital with fever, coughs and generalized weakness over the last three days. On presentation, his vital signs were unremarkable. Chest computed tomography (CT) showed atypical pattern of COVID-19. The diagnosis of COVID-19 was confirmed by polymerase chain reaction on a nasopharyngeal swab. He was treated with hydroxychloroquine, azithromycin, and prednisone and low molecular weight heparin at therapeutic dose of 60 mg/12 h. Through-out his hospitalization, the patient remained stable; he did not require oxygen and did not present any neurological or cardiac symptoms. On day 7 of admission, repeat viral PCR was negative for SARS-CoV2. On day 8, the patient was drowsy and dysarthric without evidence of motor deficit or meningeal irritation. A head CT scan was performed and did not show any acute intracranial abnormalities. Shortly thereafter, his neurological status declined rapidly, progressing to coma with anisocoria. He was emergently intubated and transferred to the intensive care unit. Magnetic resonant imaging (MRI) of the brain revealed multiple bilateral infarcts on diffusion weighted imaging sequence involving both anterior and posterior circulations. He had elevated inflammatory markers including C-reactive protein (CRP), ferritin and procalcitonin, in addition to elevated D-Dimer. Transthoracic echocardiogram (TTE) was normal with normal ejection fraction and no evidence of prosthetic valve thrombosis. No treatment was performed and the patient was pronounced dead in the next hours (Figure 1).

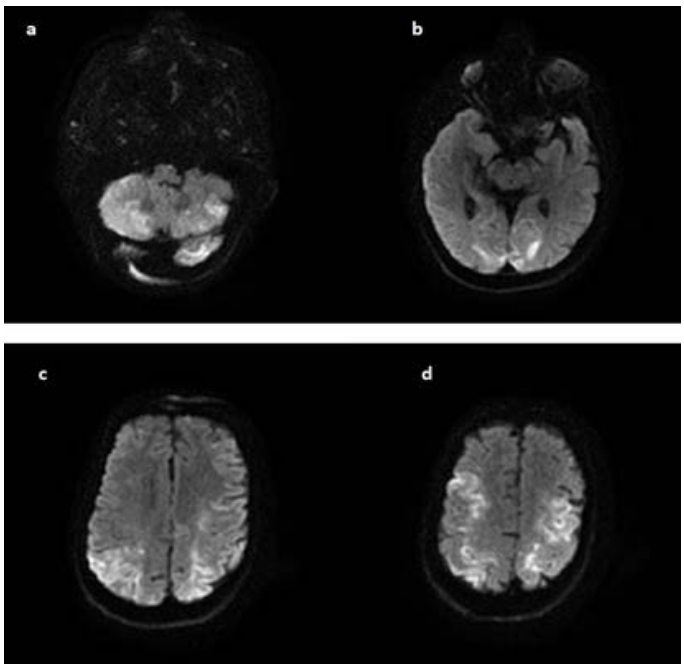

Figure 1. MRI brain, axial images of the diffusion weighted imaging (DWI) sequence at different levels (a, b, c and d) demonstrates multifocal diffusion restriction involving both of anterior and posterior territories.

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Discussion

Stroke is one of the major neurological complications associated with SARS-CoV2 infection. In the majority of cases, it is an ischemic stroke. Li et al [1,2] published one of the first studies describing the risk of stroke among SARS-CoV2 hospitalized patients. They observed a 5% risk of ischemic stroke, 0.5% cerebral venous sinus thrombosis and 0.5% cerebral hemorrhage [3]. In a multinational observational study, including a total of 17.799 hospitalized SARS-CoV2 patients, 156 (0.9%) had a stroke among them 123 (0.7%) had an ischemic stroke [2]. This variance of incidence can be explained by differences in the population studied. A higher incidence was observed in studies comprising severe COVID-19 infected patients compared to those that included all hospitalized patients with laboratory proven COVID-19 infection. Moreover, the different studies were conducted in different countries with varying ethnic demographic [4]. Although ischemic stroke related to COVID-19 has been observed in young patients without known risk factors, it mainly occurs in older patients, with cardiovascular and cerebrovascular risk factors and severe infection [5]. Typically, the median time from first COVID-19 symptoms to identify of stroke is 10 days. Rarely, stroke is the first manifestation [6].

Atypical clinical presentations were described. Usually, a patient with ischemic stroke manifests focal neurologic signs which may be particularly challenging to identify in some cases of encephalopathy such as in the current case. In a French study which reported neurologic features in severe SARS-CoV2 infection in 58 patients, thirteen patients had MRI brain performed for unexplained encephalopathy symptoms. Three patients were found to have an ischemic stroke with no focal neurological symptoms [7].

Acute ischemic stroke represents the most frequent Neuro-radiologic abnormality seen among patients with Neuro-COVID symptoms, usually involving the large vessel followed by multiple vascular territory infarcts. The most common location of the large vessel occlusion is the middle cerebral artery and the internal carotid artery. Less frequently, cases of small vessel infarcts, occlusion of the basilary artery, vertebral artery, posterior cerebral artery and anterior cerebral artery have been also reported [8].

There are several potential mechanisms for increased risk of ischemic stroke during COVID-19. Angiotensin converting enzyme which is the target site of SARS-CoV2 is expressed by nerve cells. This renders the brain at risk of direct endothelial cell infection and diffuse inflammation. The acute inflammation caused by COVID-19 is prone to be followed by an endothelial cell dysfunction and a hypercoagulable state due to the increased concentration of pro-inflammatory factors and cytokine storm [9]. In an American retrospective cohort study of patients who have a stroke with COVID-19, it was found that most strokes were cryptogenic with higher D-dimer levels at the time of stroke which suggest the role of hypercoagulability induced by COVID-19 [10]. Cardiac involvement is also a prominent feature of COVID-19, leading to stress cardiomyopathy, direct myocardial injury and arrhythmias with potential increased risk of ischemic stroke [11]. There is also evidence for a putative role of anti-phospholipid antibodies in COVID-19 related thrombotic events [12].

Defining the etiology of stroke allows the selection of the right treatment to prevent recurrence. In fact, determining the mechanism becomes difficult in the presence of a potential major cardiac source of embolism especially when the stroke occurs in multiple and bilateral territories [13]. Given that our patient was on therapeutic doses of anticoagulation and his transthoracic echocardiogram did not show intracardiac thrombus, we suggest that the virally mediated vasculitis induced by COVID-19 contributed very likely to ischemic stroke. This scenario was previously reported in multiple case reports and case series of COVID-19 patients who have a high risk of cardioembolic stroke due to a pre-existing cardiovascular disease or cardiovascular complication of COVID-19. They still developed major acute cerebrovascular events despite the consistent use of therapeutic dose anticoagulation [14].

Current recommendations suggest that every hospitalized COVID-19 patient should be placed on anticoagulation prophylaxis. However, optimal dose and duration of anticoagulation are not defined. As for patients who have a high cardio embolic risk it is obviously recommended to use curative dose anticoagulation. The choice of drug will depend on the possibility of oral treatment, renal function and other clinical conditions. As heparins are not expected to interact with drugs used for COVID-19 treatments, they may be considered as a safe alternative to oral anticoagulation for stroke prevention. Interestingly, in addition to the antithrombotic effect, the anti-inflammatory actions of heparin might be also relevant in this setting [15].

Conclusion

In summary, this case provides further evidence that vascular inflammation induced by the SARS-CoV2 contributed to an increased risk of arterial thrombosis, even among patients with non-severe COVID-19 disease. Although anticoagulant and anti-inflammatory therapy has been shown to be effective, a number of patients with COVID-19 develop drastic thrombotic complications. Therefore, it is necessary to carry out further research examining new therapeutic interventions that focus on the pathophysiology of ischemic stroke.

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Competing Interests

There are no conflicts of interests.

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