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Letter to Editor

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COVID-19 re-infection presenting as acute ischemic stroke

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Dear Editor

Acute ischemic stroke (AIS) is a life threatening complication of Coronavirus disease 2019 (COVID-19) infection [1]. The true incidence of AIS in COVID-19 patients remains still unclear, varying from 0.9% in a study conducted in New York [2] to 2.34% in a Chinese study conducted in Wuhan [3]. Increasing reports suggest an association between COVID-19 and AIS, although the underlying mechanisms remain still uncertain; however, AIS has been documented in young COVID-19 infected individuals and also in complete absence of cardiovascular risk factors or other comorbidities [4-6]. It has been proposed that COVID-19 may induce a prothrombotic condition, as supported by the elevated levels of d-dimer, fibrinogen and factor VIII frequently observed in such population [7,8].

Recent reports described cases of re-infection due to COVID-19, mainly in elderly people or in immune-compromised subjects [9-11]. However, in most of these reports, symptoms of re-infection were of lower severity compared to the primary infection and mainly affecting the respiratory system. This report describes a case of COVID-19 re-infection presenting as acute ischemic stroke.

A 61-years old woman was firstly diagnosed with COVID-19 infection at March 9th; she was completely asymptomatic except for mild rhinitis and underwent to nasopharyngeal swab due to a close contact with a positive subject in the previous days. She did not have any chronic diseases and did not take chronic therapies at that time; further, the patient never smoked and did not have significant alcohol intake during her life. COVID-19 related symptoms rapidly recovered and two nasopharyngeal swabs were negative after 2 weeks. She did not receive any specific treatment at that time. At November 15th the patient was admitted in the Emergency room due to the sudden onset of aphasia, apraxia of speech, mental confusion and agitation. These symptoms completely recovered in 2 hours. She underwent to a brain CT with contrast medium that showed occlusion of the terminal parietal branch M4 on the left, leading to an ischemic area in the left parietal area. Neurologist did not consider necessary to proceed at mechanical revascularization or revascularization by using recombinant tissue plasminogen activator (rTPA) due to the distal localization of the lesion and to the progressive recovery of symptoms. Electrocardiogram did not show cardiac arrhythmias. Chest X-ray was normal. A treatment with both clopidogrel and cardioaspirin was then started. She was tested for COVID-19 infection before being admitted to the ward; nasopharyngeal swab was still positive and serum antibodies were negative as for recent infection. A MRI was finally performed showing multiple, small ischemic lesions in left parietal and frontal lobes. She underwent to Holter electrocardiography that did not reveal any cardiac arrhythmias. Trans-thoracic ecochardiography was completely normal. Screening for auto-immune diseases was negative; homocysteine, protein C and protein S plasma levels were also in the normal range. During a 12-days long hospitalization, no respiratory symptoms occurred, but nasopharyngeal swab was still positive at discharge, and remained positive for another 13 days, always in absence of other clinical symptoms.

At our knowledge, this is the first case of COVID-19 re-infection presenting as an acute ischemic stroke. There are several potential explanations for the relation between COVID-19 infection and ischemic stroke occurrence; it has been proposed that viral infection may induce a direct vasculopathic effect or potentiate the prothrombotic pattern through several mechanisms including immune-mediated platelet activation, dehydration and infection-induced cardiac arrhythmias. It has been also reported the possibility that ischemic stroke occurs with more severe symptoms and complications in COVID-19 infected individuals [12]. The increased stroke severity at admission in COVID-19 associated stroke patients compared with the non-COVID-19 population may explain the worse outcome; in particular, the multi-system involvement of COVID-19 complications, including acute respiratory distress symptoms, cardiac arrhythmias, acute cardiac injuries, pulmonary embolism, cytokine release syndrome and secondary infections, may also contribute to the increased mortality observed in these subjects. Our patient had very mild respiratory symptoms during primary COVID-19 infection and was completely asymptomatic from a respiratory point of view during re-infection. She did not have any significant cardiovascular risk factors at the time of re-infection; therefore we can supposed that a significant role in the occurrence of the acute ischemic stroke may been due to the COVID-19 re-infection itself. This finding reinforces the awareness that COVID-19 re-infection is a concrete possibility when, after a variable period of time, the immune protection fails; further, it should reinforce the possibility that also COVID-19 re-infections may be associated with significant systemic cardiovascular or neurologic complications, as acute ischemic stroke, even in absence of respiratory symptoms.

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