

Occam's Razor, Stroke, and COVID-19



We read with great interest the article by Goldberg et al¹ reporting a case of coronavirus 2019 (COVID-19)-related cerebral infarcts in a 64-year-old man with hypertension and evidence of mild, diffuse atherosclerotic disease on brain and neck imaging. They concluded that manifestations of COVID-19 are not limited to the respiratory system and that neuroradiologists should be aware of the associated cerebrovascular disease and its potential underlying etiologies.

We should like to make some comments on this, in the hope that they may be of use.

Piacenza was the second most heavily hit Italian city by COVID-19. This allowed us to evaluate what was going on from a neurologic point of view. The period that followed the outbreak declaration witnessed the publication of only a few small case series of patients with stroke and COVID-19, leaving a scarcity of information on stroke in this unknown pathologic scenario. Moreover, the scientific literature we do have is limited to anecdotal reports, and anecdotal evidence is generally considered of limited value because it is collected in a casual or informal manner and is dependent on personal testimony. Therefore, we are of the opinion that the hypothesis that there is a direct "cause-effect" relationship between COVID-19 infection and stroke occurrence should be further investigated by prospective and large-volume studies. Despite this drawback, the role the thrombophilic state induced by COVID-19 played and the hypothesis of an increased stroke risk in infected patients was debated.^{2,3} Noteworthy is the fact that pandemic hotspots have an extremely high COVID-19 prevalence, allowing the possibility of an incidental association between infection and neurologic manifestations.

Goldberg, et al¹ reported a patient with COVID-19 whose CT angiography evidenced a segmental high-grade stenosis of the right proximal internal carotid artery, associated with wall extensive calcifications. In line with Occam's razor principle,⁴ "the simplest solution is most likely the right one," we ask ourselves if it would not be better to consider that the atheroembolic etiology from a large-vessel disease would suffice to explain the ischemic stroke. Moreover, it reports generic evidence of "coagulopathy,"

while neglecting the complexity of the diagnosis of a thrombophilic state induced by the presence of antiphospholipid antibody and anticardiolipin immunoglobulin (aPL). For example, these aPLs should be persistent (at medium/high titer on ≥ 2 consecutive occasions at least 12 weeks apart) to have a pathogenic role. Indeed, the laboratory data they reported fits well into the sphere of an unspecific laboratory sign of a sepsis, as often occurs without any pathologic relevance. The hypothesis made that associated activation of immune and inflammatory pathways led to plaque disruption and was a source of thrombosis (indeed, probably a free-floating thrombus) is not supported by further instrumental investigations. Different from carotid duplex sonography, CT angiography provides only a snapshot image of the thrombus and is unable to evidence any floating nature (ie, moving in cyclical motion with the cardiac cycles, attached to the arterial wall).

Our hospital has managed about 900 patients with COVID-19 during the past 2 months, without observing any rare stroke etiology or unforeseen high incidence in stroke subtypes.

The only observation was that the stroke severity correlated with interstitial pneumonia extension, as documented by the chest CT scan.

We think that applying the rules of the art of healing must be part and parcel of our profession. However, we must strive for evidence in the form of international multicenter studies on the occurrence of different types of cerebrovascular diseases during the COVID-19 pandemic. Hopefully time will be on our side.

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