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# Risk of cerebral stroke in coronavirus patients and its prevention

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There are 178 million COVID-19 cases overall, and the number of deaths is 3.86 million roughly. A wide range of variety was reported as the cause of death in COVID-19 patients. Most diagnosed cases present with pulmonary symptoms, but many cases are seen with initial or associated cerebral manifestations.

In SARS-CoV-2, systemic inflammation ensues and causes multiorgan failure, namely lungs, kidneys, and the brain. The possible mechanism is thought to be immune-mediated. This is responsible for the development of the devastating systemic inflammatory response syndrome (1). As there is an expression of angiotensin-converting enzyme (ACE 2) receptors on neuron and glial cells of the central nervous system; also, a target to COVID-19. In COVID-19 patients, serum D-dimer level is increased, a potential source of embolic vascular event. Moreover, excessive inflammation, decreased tissue perfusion, immobilization, and abnormal coagulopathy predispose to thrombo-vascular events in COVID-19 patients. It is associated with vascular endothelial dysfunction, increased thrombin production, and platelet activation may predispose patients to venous and arterial events, including stroke. (2). The current postulations explain that severe COVID-19 infection sufferers may develop an early hyperinflammatory state from cytokine storm followed by a prothrombotic condition that is frequently complicated by both venous and arterial thromboembolism. COVID-19 infections may infect macrophages, astrocytes, and microglia. Then it activates glial cells and promotes a pro-inflammatory state; the cytokines can cross the BBB and are associated with acute necrotizing encephalopathy (1). Clinicians should be worried about thrombotic sequelae such as stroke in both early and late phases of infection. Elevated biomarkers, namely fibrinogen, D-dimer, factor VIII, and vWF, indicate further evidence of the prothrombotic condition. It is reported that antiphospholipid Ab is commonly found in COVID-19 patients. This antibody raises the risk of thrombosis by several mechanisms –induction of cellular activation, inhibition of natural anticoagulant and fibrinolytic systems, and activating the complement system (3).

The neurological manifestation was reported at Wuhan, China, where patients presented with central nervous system (CNS) symptoms 36.4%, peripheral nervous symptoms 8.9%, and skeletal muscle symptoms 10.7% (1,4). Several studies have highlighted reports of acute cerebrovascular disease (CVD) happening after COVID-19 recovery; both intracerebral hemorrhage (ICH) and acute ischemic stroke (AIS) are included, later is more common (5).

More meticulously, this involvement was recorded as Acute ischemic stroke (incidence 0.9-2.7%), large vessel occlusion prevalence is 40.9% (4). A data interpretation said that The median age of stroke was 60, where males are slightly predominant (87/136, 63.97%). 19 patients suffered from hemorrhagic stroke (HS), four of them experienced subarachnoid hemorrhage (SAH), six patients went through a transient ischemic attack (TIA), and a total of 170 had an acute ischemic stroke. Another Report says that stroke is common among young adult and middle-aged patients suffering from COVID-19. Many of them presenting with stroke have underly-

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ing cerebrovascular risk factors, like hypertension, diabetes mellitus, hyperlipidemia, smoking, and previous history of stroke (6).

Coronaviruses (CoVs) generally exhibit neurotrophic properties, and so, there are several contributory mechanisms why and how it damages the CNS. The important one is the genetic material and proteins of many viruses have been identified in CSF or brain tissues. The neuronal pathway is one of the other possible routes. The virus may disrupt the nasal epithelium and is released mostly on the apical and basolateral sides of the epithelial cells; from there, it reaches the bloodstream or lymphatic system to reach other tissues. The viruses can also migrate via neuronal transport to infect motor or sensory nerves, resulting in CNS hypoxia, which increases anaerobic metabolism in the brain. Such patients with neurological symptoms need specialist supervision. As COVID-19-related strokes seem to have a poor prognosis and mortality rate is merely high, If the possibility of any cerebrovascular event arises, complete diagnostic workup by cranial CT and/or MRI along with angiography should be performed as early as possible. If AIS patients are eligible, one should attempt thrombolysis and mechanical thrombectomy. Unfortunately, there is no reliable data found regarding the rate of hemorrhagic transformation followed by any intervention. If no contraindications, a prophylactic dose of LMWH is considered in all patients hospitalized for SARS-CoV-2 infection, and the post-discharge anticoagulation should also be based on after assessing the person's VTE and bleeding risk. Many trials are now evaluating full-dose anticoagulation in COVID-19 patients without any diagnosed indication (e.g., arterial thrombosis or VTE, heart valve replacements, stroke prevention in atrial fibrillation). Clinical data says that there may be biologic plausibility of the thrombotic phenomena in COVID-19 patients and potential use of antithrombotic drugs (2,7).

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