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# Clinicopathological Dying of COVID-19

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## Introduction

Forensic Science, Medicine and Pathology explores all aspects of modern day forensics, applying equally to children or adults, either living or the deceased. The range of topics covered includes forensic science, medicine, nursing, and pathology, as well as toxicology, human identification, mass disasters/mass war graves, profiling, imaging, policing, wound assessment, sexual assault, anthropology, archeology, forensic search, entomology, botany, biology, veterinary pathology, and DNA. Forensic Science, Medicine, and Pathology presents a balance of forensic research and reviews from around the world to reflect modern advances through peer-reviewed papers, short communications, meeting proceedings and case reports.

We assess the utility of a Centers for Disease Control and Prevention (CDC) guidelines-based coronavirus disease 2019 (COVID-19) screening checklist for postmortem severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) surveillance, detailing the relationship between the histologic findings at autopsy and attribution of death to COVID-19. SARS-CoV-2 nasopharyngeal swabs were collected at the time of autopsy in all "checklist-positive" decedents. Additional "checklist-negative" decedents were randomly tested daily. Lung slides were blindly reviewed by 3 pathologists, assessing for the presence of diffuse alveolar damage (DAD) and other findings. Sixteen decedents had positive postmortem SARS-CoV-2 nasopharyngeal swabs and underwent complete autopsies. Seven decedents had positive screening checklists. Of these, 4 had DAD and 1 had COVID-19–associated thromboembolic disease. Of the 9 decedents with negative screening checklists, 2 had DAD, but only 1 was attributed to COVID-19; the other was likely drug related. Acute bronchopneumonia was the second most common finding, and aspiration was the likely etiology in cases without concomitant DAD.

COVID-19–related DAD was identified more commonly in decedents who screened positive by CDC checklist, but false-negatives did occur. Medical examiner offices should maintain a low threshold for random testing of decedents even when COVID-19 is not suspected. The severe acute respiratory syndrome coronavirus 2 (SARSCoV-2) pandemic has revealed diverse potential neurological complications of coronavirus disease 2019 (COVID-19). Coronaviruses are established neurovirulent organisms, and neurological symptoms occur in 88% of patients with severe disease.

Hematogenous spread to the brain may occur through damaged capillary endothelium or by binding angiotensinconverting enzyme 2 (ACE2) receptors to cross the blood-brain barrier, where virus enters neurons and glial cells.1–4 Dissemination via retrograde transport in the olfactory bulb is another possibility. Once in the brain, transsynaptic transfer has been postulated.5 One major complication of "neuro-COVID" is stroke, which may be linked to SARS-CoV-2 hypercoagulability. COVID-19 patients have a stroke incidence 8 times higher than those hospitalized with influenza, occurring in 3%of hospitalized patients, with an incidence of 6% in those with severe disease. Stroke may be the first presentation of COVID19 but is usually delayed with respect to symptom onset.6,7 In a series from China, predominantly older COVID-19 stroke patients with other cardiovascular risk factors were described, with an incidence of 5%among hospitalized patients.8 On the other hand, young patients with COVID-19 and large-vessel stroke are rarely described. A recent report documented 5 cases of large-vessel stroke in patients younger than 50 years in whom COVID-19 infection was diagnosed.9 the independent relationship of viral respiratory tract infection and ischemic stroke is not a new concept.2 General viral infections increase stroke risk due to vascular inflammation, platelet activation, and hypercoagulability.

Additionally, inflammatory destabilization of atheromatous plaques is a suggested mechanism.2,4 Influenza viruses aggravate ischemic brain injury as well as increase the risk of hemorrhage after thrombolytic therapy.6 Large-vessel stroke has also been previously reported in association with SARS-CoV-1.10 It should be observed that secondary infarcts may be due to the general hypotension and hypoxia in patients with severe systemic illness.6 There are many possible mechanisms of stroke; however, as yet, there is no proven direct causal link.2 Comorbidities common to both COVID-19 and stroke, such as diabetes, obesity, and hypertension, may partly explain the coincidence of the 2 pathologies. Such patients are often older, with more evidence of systemic inflammation.4,5 These factors strongly influence microglial phenotype, which modulates the local effect of the virus in the brain.4 Of interest, ACE2 receptor expression is increased in ischemic brains and in the brains of diabetics, making viral entry to the CNS more likely.3 In turn, viral binding to ACE2 receptors can raise blood pressures, increasing the risk of cerebral hemorrhage.

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