

# Early postmortem brain MRI findings in COVID-19 non-survivors

Tim Coolen, MD, Valentina Lolli, MD, Niloufar Sadeghi, MD, PhD, et al.

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## Correspondence

Dr. De Tiège  
xdetiege@ulb.ac.be

## Study question

What structural brain abnormalities are present in persons who die from coronavirus disease 2019 (COVID-19)?

## What is known and what this paper adds

The protein that severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) uses as a cellular entry point is expressed on glia and neurons, and case studies have reported various neurologic manifestations in patients with COVID-19. This investigation's results show that people who die from COVID-19 may have parenchymal brain abnormalities.

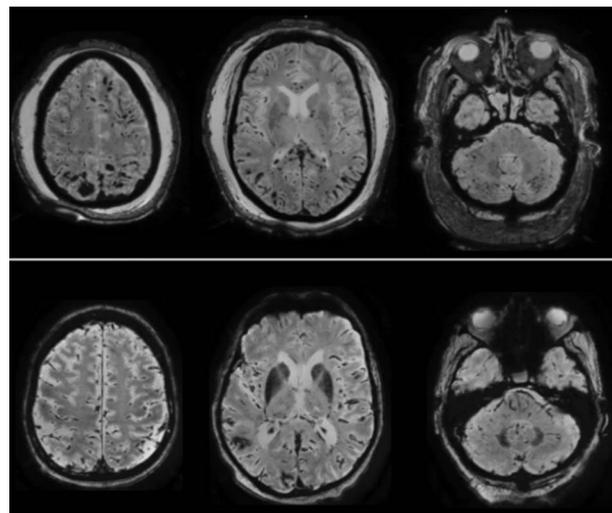
## Methods

This prospective case-series study includes data from 19 consecutive adults decedents (74% male; mean age at death, 77 years; range, 49–94 years) without known focal brain lesions who had positive nasopharyngeal swab specimens for SARS-CoV-2 and CT changes suggestive of COVID-19. The MRI scans were done within 24 hours of death and included whole-brain axial 3-dimensional (3D) T1-weighted imaging, sagittal 3D T2-weighted fluid-attenuated inversion-recovery imaging, axial 3D susceptibility-weighted imaging, and axial diffusion-weighted imaging. MRI data were first screened for signal abnormalities that could be confidently attributed to early postmortem changes based on prior descriptions (T1WI signal of basal ganglia and thalami, suppression of fat signal intensity on T2WI, increased signal intensity of the cortical ribbon and the ventricular wall on DWI, and globally reduced apparent diffusion coefficient (ADC) values in the brain parenchyma) and for potential additional postmortem changes that have not been reported. MRI data were subsequently screened for signal abnormalities reflecting antemortem changes, and these were classified as recent (i.e., potentially related to COVID-19: hemorrhages, oedema, and olfactory clefts and bulbs abnormalities) and long-standing (i.e., unlikely related to COVID-19: white matter changes, enlargement of perivascular spaces, cerebral atrophy, and late-stage lacunar ischemic or hemorrhagic changes.)

## Results and study limitations

Four decedents had parenchymal brain abnormalities, including subcortical micro- and macrobleeds (n = 2),

**Figure** Susceptibility-weighted images from a decedent with diffuse micro- and macrobleeds (top) and a decedent with subcortical macrobleeds (bottom)



corticocortical edematous changes evocative of posterior reversible encephalopathy syndrome (n = 1), and nonspecific deep white matter changes (n = 1). Four others had asymmetric olfactory bulbs without downstream olfactory tract abnormalities. No decedents had MRI abnormalities in the brainstem that could support a neurologic contribution to the respiratory distress commonly observed in patients with COVID-19. The present study's limitations include its small sample size and the fact that the included decedents all had severe COVID-19 pathology, which may limit generalizability to patients with less severe cases.

## Study funding and competing interests

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