Inability to Read After Prolonged COVID-19 Hospitalization: MRI With Clinical Correlation

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Coronavirus Disease 2019 (COVID-19) is a viral condition caused by severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2). Studies suggest that SARS-CoV-2, such as most coronaviruses, is capable of invading the central nervous system to exacerbate or cause neurological disease (1). In addition, the hypercoagulable and inflammatory states seen in COVID-19 are reported to cause multi-organ dysfunction including strokes (2). Given the system-wide complications reported in cases of SARS-CoV-2, there is great interest in the neuro-ophthalmological sequelae of COVID-19. We describe a patient who detected difficulty with reading the right side of words after intensive care unit hospitalization with SARS-CoV-2 and was found to have visual field defects due to ischemic stroke.

A 68-year-old woman was referred to the neuro-ophthalmology clinic because of reported difficulty reading. The patient’s medical history was significant for a 55-day hospitalization with COVID-19 confirmed by polymerase chain reaction and complicated by 30 days on mechanical ventilation. The patient’s chief complaint during outpatient rehabilitation was simply “I am missing parts of words when I am reading.” On examination, the patient demonstrated the ability to write but could not see the right side of words. The best-corrected visual acuity was 20/25 in the right eye and 20/20 in the left eye. Visual fields by confrontation suggested a right inferior homonymous hemianopsia. The funduscopic examination was unremarkable. The patient was cognitively intact and the remainder of the neurologic examination was normal except for mild proximal muscle weakness.

The medical history was unrevealing for any risk factors of stroke such as hypertension, diabetes, or smoking but was notable for ulcerative colitis. Ocular history was noncontributory.

Automated visual fields (HVF) revealed a right homonymous hemianopsia and a subtle left superior homonymous quadrantanopsia. On follow-up visit 17 days later, her examination revealed improved strength of the upper and lower extremities. Repeat HVF 30-2 showed an improving incongruous partial right homonymous hemianopsia and significant improvement in the left superior homonymous quadrantanopsia.  

FIG. 1. 30-2 automated visual fields from the patient’s first and second visit spaced 17 days apart. A. Patient’s first visit notable for a right homonymous hemianopsia and a subtle left superior homonymous quadrantanopsia. B. Patient’s second visit with a resolution of the left superior visual field defects and an improvement in the right hemianopsia.
The clinical image was suggestive of stroke, prompting neuroimaging after the patient’s first visit. MRI brain with and without contrast revealed a large subacute to chronic infarct in the left occipitotemporal region (Figs. 2A, B) and a smaller right occipital infarct (Figs. 2C, D). T2 imaging showed widespread signal loss consistent with petechial hemorrhage. T2 GRE (sensitive to blood products) showed numerous areas of curvilinear and punctate signal loss consistent with hemosiderin deposition and chronic microhemorrhages (Fig. 2E). Note that, in the case of these findings, chronic refers to greater than or equal to 6 weeks, which is well within the patient’s hospital course. Neuro-radiology’s impression is that these are infarcts because of thromboembolism and/or hypoxia. Computed tomographic angiography of the head and neck was supportive of a left occipital hemorrhagic infarct; no flow-limiting stenosis or aneurysm was observed. Bilateral ground glass opacities were noted in the lung apices, consistent with the patient’s history of COVID-19. Echocardiogram was normal.

We describe a case of a patient with visual field deficits consistent with stroke who presented shortly after hospitalization with COVID-19. Reports show that COVID-19 can affect multiple organ systems in part because of its hypercoagulable state, as was observed by this patient’s markedly elevated D-dimer, mildly elevated fibrinogen, and necrosis of the first and second digits of the left hand. Although brain imaging of patients with COVID-19-related thrombotic complications is still being characterized, our patient’s MRI findings are in line with those previously described in patients at other centers. In a recent article by Radmanesh et al, of 242 confirmed COVID-19 positive patients, 5.4% were found to have acute or subacute infarcts on MRI, similar to our patient (3). This is not surprising as a hypercoagulable state is associated with COVID-19 (1).

In addition, the microhemorrhages seen on MRI are reminiscent of other patients on long-term ventilation and are suggested as a late complication of critically ill COVID-19 patients (4). It is important to mention that although our findings seem to correlate well clinically, these imaging findings can also be seen in encephalitis, amyloid angiopathy, small vessel vasculitis, and endocarditis. Although these conditions could potentially explain the imaging findings, they are not clinically supported in this patient.

This case is of interest because ophthalmologists and neuro-ophthalmologists need to be aware of the possible visual impact of COVID-19. This patient had no neurological complaints during her hospitalization. The patient’s infarcts were found only after presenting to neuro-ophthalmology for reading difficulties. To the best of our knowledge, this is the first reported account of a patient with COVID-19-associated visual field defects found after hospitalization. We hope to increase awareness of the potential neuro-ophthalmic impact of COVID-19.

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