Abnormalities of Cortical Thickness, Subcortical Shapes, and White Matter Integrity in Subcortical Vascular Cognitive Impairment

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Abstract: Subcortical vascular cognitive impairment (sVCI) is caused by lacunar infarcts or extensive and/or diffuse lesions in the white matter that may disrupt the white matter circuitry connecting cortical and subcortical regions and result in the degeneration of neurons in these regions. This study used structural magnetic resonance imaging (MRI) and high angular resolution diffusion imaging (HARDI) techniques to examine cortical thickness, subcortical shapes, and white matter integrity in mild vascular cognitive impairment no dementia (VCIND Mild) and moderate-to-severe VCI (MSVCI). Our study found that compared to controls ($n = 25$), VCIND Mild ($n = 25$), and MSVCI ($n = 30$) showed thinner cortex predominantly in the frontal cortex. The cortex in MSVCI was thinner in the parietal and lateral temporal cortices than that in VCIND Mild. Moreover, compared to controls, VCIND Mild and MSVCI showed smaller shapes (i.e., volume reduction) in the thalamus, putamen, and globus pallidus and ventricular enlargement. Finally, compared to controls, VCIND Mild and MSVCI showed an increased mean diffusivity in the white matter, while decreased generalized fractional anisotropy was only found in the MSVCI subjects. The major axonal bundles involved in the white matter abnormalities were mainly toward the frontal regions, including the internal capsule/corona radiata, uncinate fasciculus, and anterior section of the inferior fronto-occipital fasciculus, and were anatomically connected to the affected cortical and subcortical structures. Our findings suggest that abnormalities in cortical, subcortical, and white matter morphology in sVCI occur in anatomically connected structures, and that abnormalities progress along a similar trajectory from the mild to moderate and severe conditions.