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Opinion

Late Life Depression in Adults with Small Vessel Disease

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H Cerebral small vessel disease (SVD) refers to a group of pathological conditions affecting the small perforating arterioles, capillaries, and venules in the brain. Most people over the age of 60 have some neuroradiologic evidence of SVD, which is a substantial contributor to cognitive impairments, gait abnormalities, stroke, and dementia progression. Reduced processing speed and executive dysfunction are two common cognitive abnormalities linked with SVD. Even among people with neuroradiologically equivalent degrees of vascular disease burden, the pattern and intensity of cognitive impairments varies greatly. Other factors may affect the ability to sustain cognitive functions in the presence of white matter abnormalities, based on individual variances in the cognitive presentation and course of SVD. Pathophysiologic mechanisms driving specific cognitive phenotypes in SVD might benefit from a better understanding of the elements that lead to these symptom differences.

Late-life depression (LLD) may affect the presence and severity of cognitive abnormalities in older persons with SVD, generating a vulnerability that contributes to the clinical manifestation of cerebrovascular illness. LLD is associated with cognitive abnormalities, including slow processing speed, episodic memory loss, and executive dysfunction. These deficiencies are most noticeable during a depressive episode, although they frequently linger after antidepressant treatment. Individuals with LLD who have persistent cognitive abnormalities have a faster rate of cognitive and functional decline, disability, and dementia progression. The processes that link depression severity to cognitive impairment are unknown. In the context of cerebrovascular burden, which is common in LLD, problems in hormone control, immunological signalling, neurotrophic support, and lipid metabolism may contribute to cognitive deficiencies.

The link between depression and the cognitive manifestations of SVD remains a mystery, especially in samples without overt neurocognitive disorders. One theory is that in the presence of SVD, the neurobiological mechanisms that predispose people to LLD may increase cognitive weaknesses. As a result, LLD may play a role in the cognitive heterogeneity associated with white matter abnormalities in older adults, aggravating processing speed deficiencies and highlighting impairments in higher-order domains. With conflicting results, studies examining this idea have primarily relied on macrostructural indicators of SVD, such as white matter hyperintensities (WMH) and lacunes of putative vascular origin. SVD, on the other hand, is a widespread brain illness that causes microstructural damage in addition to obvious lesions.

Diffusion-weighted imaging, which detects small tissue abnormalities in normal-appearing white matter, may be able to capture the clinical extent and pathogenesis of SVD better than standard markers. Peak width of skeletonized mean diffusivity (PSMD), a new diffusion-weighted imaging-derived metric, has been validated as a diagnostic for SVD and reveals strong relationships with cognitive deficiencies associated to vascular pathology. PSMD captures the distribution of mean diffusivity (MD) values across the white matter compartment via skeletonization and histogram analysis, resulting in a global estimate of diffuse white matter dysfunction. PSMD outperformed traditional MRI measures in diagnosing cognitive deficits in samples with sporadic and inherited SVD and linked more strongly with cognitive performance in healthy older community groups with SVD imaging abnormalities, according to multiple recent investigations (WMH). PSMD has yet to be tested in depressed older persons, but it may help to better define the scope of cognitive abnormalities associated with vascular dysfunction in LLD.

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