

Hypoperfusion of brain parenchyma is associated with the severity of chronic cerebrospinal venous insufficiency in patients with multiple sclerosis: a cross-sectional preliminary report

Several studies have reported hypoperfusion of the brain parenchyma in multiple sclerosis (MS) patients. We hypothesized a possible relationship between abnormal perfusion in MS and hampered venous outflow at the extracranial level, a condition possibly associated with MS and known as chronic cerebrospinal venous insufficiency (CCSVI).

Methods: We investigated the relationship between CCSVI and cerebral perfusion in 16 CCSVI MS patients and 8 age- and sex-matched healthy controls.

Subjects were scanned in a 3-T scanner using dynamic susceptibility, contrast-enhanced, perfusion-weighted imaging. Cerebral blood flow (CBF), cerebral blood volume (CBV) and mean transit time (MTT) were measured in the gray matter (GM), white matter (WM) and the subcortical GM (SGM).

The severity of CCSVI was assessed according to the venous hemodynamic insufficiency severity score (VHISS) on the basis of the number of venous segments exhibiting flow abnormalities.

Results: There was a significant association between increased VHISS and decreased CBF in the majority of examined regions of the brain parenchyma in MS patients. The most robust correlations were observed for GM and WM ($r = -0.70$ to -0.71 , $P < 0.002$ and P corrected = 0.022), and for the putamen, thalamus, pulvinar nucleus of thalamus, globus pallidus and hippocampus ($r = -0.59$ to -0.71 , $P < 0.01$ and P corrected < 0.05).

No results for correlation between VHISS and CBV or MTT survived multiple comparison correction.

Conclusions: This pilot study is the first to report a significant relationship between the severity of CCSVI and hypoperfusion in the brain parenchyma. These preliminary findings should be confirmed in a larger cohort of MS patients to ensure that they generalize to the MS population as a whole.

Reduced perfusion could contribute to the known mechanisms of virtual hypoxia in degenerated axons.

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