Vascular cognitive impairment

Vascular cognitive impairment (VCI) represents the second most common cause of dementia after Alzheimer's disease, and pathological changes in cerebral vascular structure and function are pivotal causes of VCI. Cognitive impairment caused by arterial ischemia has been extensively studied the whole time; the influence of cerebral venous congestion on cognitive impairment draws doctors' attention in recent clinical practice, but the underlying neuropathophysiological alterations are not entirely understood. A study by Wei et al. elucidated the specific pathogenetic role of cerebral venous congestion in cognitive-behavioral deterioration and possible electrophysiological mechanisms. Using cerebral venous congestion rat models, they found these rats exhibited decreased long-term potentiation (LTP) in the hippocampal dentate gyrus and impaired spatial learning and memory. Based on untargeted metabolomics, N-acetyl-L-cysteine (NAC) deficiency was detected in cerebral venous congestion rats; supplementation with NAC appeared to ameliorate synaptic deficits, rescue impaired LTP, and mitigate cognitive impairment. In a cohort of cerebral venous congestion patients, NAC levels were decreased; NAC concentration was negatively correlated with subjective cognitive decline (SCD) score but positively correlated with mini-mental state examination (MMSE) score. These findings provide a new perspective on cognitive impairment and support further exploration of NAC as a therapeutic target for the prevention and treatment of VCI [1)]

Vascular cognitive impairment (VCI) is a critical issue in moyamoya disease (MMD). However, the glucose metabolic pattern in these patients is still unknown. This study aimed to identify the metabolic signature of cognitive impairment in patients with MMD using 18F-2-fluoro-2-deoxy-D-glucose positron emission tomography (18F-FDG PET) and establish a classifier to identify VCI in patients with MMD. One hundred fifty-two patients with MMD who underwent brain 18F-FDG PET scans before surgery were enrolled and classified into nonvascular cognitive impairment (non-VCI, n = 52) and vascular cognitive impairment (VCI, n = 100) groups according to neuropsychological test results. Additionally, thirty-three health controls (HCs) were also enrolled. Compared to HCs, patients in the VCI group exhibited extensive hypometabolism in the bilateral frontal and cingulate regions and hypermetabolism in the bilateral cerebellum, while patients in the non-VCI group showed hypermetabolism only in the cerebellum and slight hypometabolism in the frontal and temporal regions. In addition, we found that the patients in the VCI group showed hypometabolism mainly in the left basal ganglia compared to those in the non-VCI group. The sparse representation-based classifier algorithm taking the SUVr of 116 Anatomical Automatic Labeling (AAL) areas as features distinguished patients in the VCI and non-VCI groups with an accuracy of 82.4%. This study demonstrated a characteristic metabolic pattern that can distinguish patients with MMD without VCI from those with VCI, namely, hypometabolic lesions in the left hemisphere played a more important role in cognitive decline in patients with MMD [2)].

1)
Wei H, Jiang H, Zhou Y, Xiao X, Zhou C, Ji X. Cerebral venous congestion alters brain metabolite profiles, impairing cognitive function. J Cereb Blood Flow Metab. 2023 Jun 13:271678×231182244. doi: 10.1177/0271678×231182244. Epub ahead of print. PMID: 37309740.
2)
Weng R, Ren S, Su J, Ni W, Yang C, Gao X, Xiao W, Zhang X, Jiang H, Guan Y, Huang Q, Gu Y. 18F-FDG PET and a classifier algorithm reveal a characteristic glucose metabolic pattern in adult patients with moyamoya disease and vascular cognitive impairment. Brain Imaging Behav. 2023 Jan 13. doi: 10.1007/s11682-022-00752-4. Epub ahead of print. PMID: 36637715.