Cerebral venous congestion

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)]

Postoperative intracranial hypotension-associated venous congestion (PIHV) is a rare event.

Kurzbuch et al. report the case of a patient presenting with PIHV after spinal surgery following the sudden loss of cerebrospinal fluid (CSF) induced by suction drainage.

A 69-year-old patient underwent uneventful revision surgery for wound dehiscence after lumbar surgery with the placement of a subfascial suction drain.

Results: Postoperatively, the patient presented with fluctuating consciousness and a generalized tonic-clonic seizure. Computed tomography (CT) and serial magnetic resonance imaging (MRI) was performed showing convexity subarachnoid hemorrhages (SAHs), diffuse swelling of the brain and thalami and striatum bilaterally without diffusion restriction, and signs of intracranial hypertension resulting in pseudohypoxic brain swelling in PIHV. A dural leak at L3-L4 was treated with several CT-guided patches combining autologous blood and fibrin glue injections. The patient recovered without the neurologic deficit and the follow-up MRI revealed a progressive complete reversal of brain swelling and re-expansion of CSF spaces.

PIHV is a rare but potentially fatal entity. Awareness of PIHV after cranial or spinal surgery leads to early treatment of CSF hypovolemia and possibly better clinical outcomes. Following acute CSF volume loss, an acute elevation of cerebral blood volume overcoming autoregulatory mechanisms seems a likely explanation for diffuse cerebral vasogenic edema and SAH in PIHV [2)].

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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)
Kurzbuch AR, Bourlond B, García Martínez JJ, Bonjour T, Novaes NP, Tuleasca C, Millán DS. Postoperative Intracranial Hypotension-Associated Venous Congestion after Spinal Surgery Managed

with Multiple Blood Patches: Case Report. J Neurol Surg A Cent Eur Neurosurg. 2022 Oct 27. doi: 10.1055/s-0042-1757173. Epub ahead of print. PMID: 36302518.