Venous outflow obstruction

Traumatic brain injury (TBI) is a global public health problem. As an important cause of secondary injury, cerebrovascular reaction can cause secondary bleeding, venous sinus thrombosis, and malignant brain swelling. Clinical studies have confirmed that intracranial venous return disorder is closely related to the Traumatic brain injury prognosis, yet the specific molecular mechanism involved in this process is still unclear. A study used an acute subdural hematoma (ASDH) model with cranial venous outflow obstruction (CVO) to explore how CVO aggravates the pathological process after TBI, especially for inflammation and tissue damage. The results suggest that intracranial venous return disorder exacerbates neurological deficits and brain edema in rats with ASDH by aggravating the destruction of endothelial cell tight junctions (TJs) proteins and promoting the expression of inflammatory factors, the activation of microglia and expression of recombinant A disintegrin and metalloprotease 17 (ADAM17) as well as the secretion of solTNF-α, a soluble form of tumor necrosis factor-alpha (TNFα), which in turn increase IκB-α [1].

[1]
inhibitor of the transcription factor nuclear factor-κB) and NF-κB p65. Our study revealed a molecular basis of how CVO aggravates inflammation and tissue damage ((Wang C, Xian L, Zheng S, Li J, Chen X, Wang S. Cranial venous-outflow obstruction promotes neuroinflammation via ADAM17/solTNF-α/NF-κB pathway following experimental TBI. Brain Res Bull. 2023 Nov;204:110804. doi: 10.1016/j.brainresbull.2023.110804. Epub 2023 Oct 31. PMID: 37918697.