Hydrocephalus after intraventricular hemorrhage

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Hydrocephalus remains a common sequela of intraventricular hemorrhage (IVH) despite adequate drainage of the hematoma, including endoscopic surgery, intraventricular fibrinolysis, and external ventricular drainage (EVD). Moreover, the appropriate timing for conversion from EVD to ventriculoperitoneal shunt (VPS) is uncertain. This study aimed to evaluate the predictors of shunt dependency in patients with IVH based on the early EVD weaning protocol in our institution.

Noiphithak et al. retrospectively reviewed medical records of patients who were diagnosed with primary IVH and secondary IVH from spontaneous intracerebral hemorrhage during the period 2018-2021. Predictors associated with shunt dependency were identified using a logistic regression model. The cutoff point of each variable was selected by receiver operating characteristic curve analysis. Furthermore, shunt complications were reported as a safety measure of our early EVD weaning protocol.

The analysis included 106 patients. After IVH treatment, 15 (14%) patients required ventriculoperitoneal shunt, whereas 91 (86%) patients were shunt-free. The diameter of posttreatment temporal horn and the degree of IVH reduction were the significant predictors of shunt dependency. Moreover, patients with IVH reduction of >45% and temporal horn diameter of <9 mm had a lower probability of shunt dependency. Shunt failure was found in 2 (13.3%) patients.

This study showed that a large temporal horn diameter and a lower degree of IVH removal were predictors of shunt dependency in patients with IVH. In addition, early conversion from EVD to ventriculoperitoneal shunt is safe and feasible [1)].

Intraventricular hemorrhage (IVH) is a subtype of intracerebral hemorrhage (ICH) with high morbidity and mortality. Posthemorrhagic hydrocephalus (PHH) is a common and major complication that affects prognosis, but the mechanism is still unclear. Inflammation and fibrosis have been well established as the major causes of PHH after IVH. Cao et al. aimed to investigate the effects of

metformin on IVH in adult male mice and further explored the underlying molecular mechanisms of these effects. In the acute phase, metformin treatment exerted dose-dependent neuroprotective effects by reducing periependymal apoptosis and neuronal degeneration and decreasing brain edema. Moreover, high-dose metformin reduced inflammatory cell infiltration and the release of pro-inflammatory factors, thus protecting ependymal structure integrity and subependymal neurons. In the chronic phase, metformin administration improved neurocognitive function and reduced delayed hydrocephalus. Additionally, metformin significantly inhibited basal subarachnoid fibrosis and ependymal glial scarring. The ependymal structures were partially restored. Mechanically, IVH reduced phospho-AMPK (p-AMPK) and SIRT1 expression and activated the phospho-NF-κB (p-NF-κB) inflammatory signaling pathway. However, metformin treatment increased AMPK/SIRT1 expression and lowered the protein expression of p-NF-κB and its downstream inflammation. Compound C and EX527 administration reversed the anti-inflammatory effect of metformin. In conclusion, metformin attenuated neuroinflammation and subsequent fibrosis after IVH by regulating AMPK /SIRT1/ NF-κB pathways, thereby reducing delayed hydrocephalus. Metformin may be a promising therapeutic agent to prevent delayed hydrocephalus following IVH [2)]

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Noiphithak R, Phumichard T, Ratanavinitkul W, Rukskul P. Prediction of Permanent Shunt Dependency in Patients with Intraventricular Hemorrhage: Outcomes of Early External Ventricular Drainage Weaning Protocol. World Neurosurg. 2023 Nov;179:e575-e581. doi: 10.1016/j.wneu.2023.09.008. Epub 2023 Sep 7. PMID: 37689359.

2)
Cao Y, Liu C, Li G, Gao W, Tang H, Fan S, Tang X, Zhao L, Wang H, Peng A, You C, Tong A, Zhou L. Metformin Alleviates Delayed Hydrocephalus after Intraventricular Hemorrhage by Inhibiting Inflammation and Fibrosis. Transl Stroke Res. 2022 Jul 19. doi: 10.1007/s12975-022-01026-3. Epub ahead of print. PMID: 35852765.