

Brain injury following aneurysmal subarachnoid hemorrhage

- [Cerebral Amyloid Angiopathy With Progressive Cortical Superficial Siderosis and Convexity Subarachnoid Hemorrhage in an Apolipoprotein E \(APOE\) epsilon2/epsilon2 Homozygous Patient: A Case Report and Literature Review](#)
- [New-Onset Psychosis Associated With Acute Cerebral Hemorrhage: A Case Report](#)
- [Retraction: Apolipoprotein E-mimetic peptide COG1410 promotes autophagy by phosphorylating GSK-3beta in early brain injury following experimental subarachnoid hemorrhage](#)
- [Reliability of Antemortem CT Scan Findings as an Adjunct to Autopsy in Visualizing Intracranial Hemorrhage](#)
- [Naochuxue formula attenuates early brain injury following subarachnoid hemorrhage by inhibiting neuronal apoptosis via network pharmacology and in vivo experiments](#)
- [Role of Magnetic Resonance Venography in the Evaluation of Cerebral Veins and Sinuses Occlusion](#)
- [Correction to "Metrnl/C-KIT Axis Attenuates Early Brain Injury Following Subarachnoid Hemorrhage by Inhibiting Neuronal Ferroptosis"](#)
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[Aneurysmal subarachnoid hemorrhage](#) can result in [brain injury](#) and [impairments](#), including cognitive and neurological deficits, [memory](#) problems, [mood](#) changes, and difficulty with daily activities. The severity of brain injury following aSAH varies depending on factors such as the location and size of the bleed, the amount of [brain tissue](#) affected, and the speed of treatment.

[Oxidative stress](#) and [neuroapoptosis](#) are key pathological processes after subarachnoid hemorrhage (SAH)

Sphingosine-1-phosphate (S1P) is generated intracellularly and, when transported to the [extracellular](#) compartment, predominantly signals through S1P receptors. The S1P signaling pathway has been implicated in the pathophysiology of neurological injury following [aneurysmal subarachnoid hemorrhage](#) (aSAH).

In a review, Gaastra et al. bring together all the available data regarding the role of S1P in neurological injury following aSAH. There is an agreement in the literature that S1P increases in the [cerebrospinal fluid](#) following aSAH and leads to cerebral artery [vasospasm](#). On the other hand, the role of S1P in the parenchyma is less clear-cut, with different studies arguing for beneficial and deleterious effects. A parsimonious interpretation of this apparently conflicting data is presented. They discuss the potential of S1P receptor modulators, in clinical use for [multiple sclerosis](#), to be repurposed for aSAH. Finally, they highlight the gaps in the knowledge of S1P signaling in humans, the clinical challenges of targeting the S1P pathway after aSAH, and other research priorities ¹⁾.

Early [brain injury](#) and [hydrocephalus](#) (HCP) are important mediators of poor outcomes in subarachnoid hemorrhage (SAH) patients. Injection of SAH patients' CSF into the rat ventricle leads to HCP as well as subependymal injury compared with injection of control CSF ²⁾.

Brain edema after aneurysmal subarachnoid hemorrhage

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1)

Gaastra B, Zhang J, Tapper W, Bulters D, Galea I. Sphingosine-1-phosphate Signalling in Aneurysmal Subarachnoid Haemorrhage: Basic Science to Clinical Translation. *Transl Stroke Res*. 2023 Feb 7. doi: 10.1007/s12975-023-01133-9. Epub ahead of print. PMID: 36749550.

2)

Li P, Chaudhary N, Gemmete JJ, Thompson BG, Hua Y, Xi G, Pandey AS. Intraventricular Injection of Noncellular Cerebrospinal Fluid from Subarachnoid Hemorrhage Patient into Rat Ventricles Leads to Ventricular Enlargement and Periventricular Injury. *Acta Neurochir Suppl*. 2016;121:331-4. doi: 10.1007/978-3-319-18497-5_57. PubMed PMID: 26463970.

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