

Seizure after aneurysmal subarachnoid hemorrhage

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Epidemiology

Literature has reported [seizure](#) rates to be as high as 27% in this population ¹⁾.

More recently published studies have found seizure rates to be significantly lower than previously described (1-10%) ^{2) 3)}.

Risk factors

Paavola et al. examined data for 760 [consecutive](#) 12-month [survivors](#) of [aneurysmal subarachnoid hemorrhage](#), born in 1950 or after, with a first aSAH from January 1, 1995, to December 31, 2018. Of the 760 patients (median age, 47 years; 53% females; median follow-up, 11 years), 111 (15%) developed [epilepsy](#) at a median of 7 months (interquartile range, 2-14 months) after admission for aSAH. Of the 2240 [population](#) controls and 4653 first-degree relatives of aSAH patients, 23 (0.9%) and 80 (1.7%) respectively developed epilepsy during the follow-up period. Among the 79 patients with epilepsy in first-degree relatives, 22 (28%) developed epilepsy after aSAH; in contrast, among the 683 patients with no epilepsy in first-degree relatives, 89 (13%) developed epilepsy after aSAH. Having at least one relative with epilepsy was an independent risk factor for epilepsy after aSAH (hazard ratio, 2.44; 95% confidence interval, 1.51-3.95). Cumulative 1-year rates by first-degree relationship were 40% with one or more children with epilepsy, 38% with one or more affected parents, 5% with one or more affected siblings, and 10% with no relatives with epilepsy.

Patients who developed [epilepsy](#) after aSAH were significantly more likely to have first-degree relatives with epilepsy than those who did not develop epilepsy after the aSAH ⁴⁾.

Epilepsy is a common and serious complication of **subarachnoid hemorrhage** (SAH), giving rise to increased **morbidity** and **mortality**. It's difficult to identify patients at high risk of epilepsy and the application of **antiepileptic drugs** (AEDs) following SAH is a controversial topic. Therefore, it's pressingy needed to gain a better understanding of the risk factors, underlying mechanisms, and the optimization of therapeutic strategies for epilepsy after SAH. **Neuroinflammation**, characterized by **microglial** activation and the release of inflammatory **cytokines** has drawn growing attention due to its influence on patients with epilepsy after SAH. In a review, Wang et al. discussed the **risk factors** for epilepsy after SAH and emphasize the critical role of microglia. Then they discussed how various **molecules** arising from pathophysiological changes after SAH activates specific receptors such as **TLR4**, **NLRP3**, **RAGE**, **P2X7R** and initiate the downstream inflammatory pathways. Additionally, they focused on the significant responses implicated in epilepsy including neuronal **excitotoxicity**, the disruption of the **blood-brain barrier** (BBB), and the change of immune responses. As the application of AEDs for seizure prophylaxis after SAH remains controversial, the regulation of neuroinflammation targeting the key pathological molecules could be a promising therapeutic method. While neuroinflammation appears to contribute to epilepsy after SAH, more comprehensive experiments on their relationships are needed ⁵⁾.

Complications

Seizure activity has been associated with secondary neurologic injury including reduced **cerebral blood flow** and **intracranial hypertension** ⁶⁾.

Prohylaxis

see [Anticonvulsant in aneurysmal subarachnoid hemorrhage](#).

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⁶⁾

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