Cognitive disorder after subarachnoid hemorrhage

Subarachnoid hemorrhage (SAH) leads to significant long-term cognitive deficits, so-called the post-SAH syndrome. Existing neurological scales used to assess outcomes of SAH are focused on sensory-motor functions. To better evaluate short-term and chronic consequences of SAH, Regnier-Golanov et al. explored and validated a battery of neurobehavioral tests to gauge the functional outcomes in mice after the circle of Willis perforation-induced SAH. The 18-point Garcia scale, applied up to 4 days, detected impairment only at the 24-h time point and showed no significant difference between the Sham and SAH group. A decrease in locomotion was detected at 4-days post-surgery in the open field test but recovered at 30 days in Sham and SAH groups. However, an anxiety-like behavior undetected at 4 days developed at 30 days in SAH mice. At 4-days post-surgery, Y-maze revealed an impairment in working spatial memory in SAH mice, and dyadic social interactions showed a decrease in the sociability in SAH mice, which spent less time interacting with the stimulus mouse. At 30 days after ictus, SAH mice displayed mild spatial learning and memory deficits in the Barnes maze as they committed significantly more errors and used more time to find the escape box but still were able to learn the task. They also observed cognitive dysfunction in the SAH mice in the novel object recognition test. Taken together, these data suggest dysfunction of the limbic system and hippocampus in particular. They suggest a battery of 5 basic behavioral tests allowing to detect neurocognitive deficits in a sub-acute and chronic phase following the SAH

Aneurysmal subarachnoid hemorrhage (aSAH) has a very high mortality (>25%) and significant morbidity (>50%) among the survivors, and most survivors experience significant cognitive decline across multiple domains, including executive function.

A study of Ma et al., from the Department of Neurosurgery, Southwest Hospital, Third Military Medical University (Army Medical University), Chongqing, China, explores a potential treatment for cognitive dysfunction following SAH with the demonstration that multi-target drug Cattle encephalon glycoside and ignotin injection (CEGI) can relieve cognitive disorder by decreasing hippocampal neuron apoptosis following SAH in rats. Experimentally, 110 male SD rats were separated at random into Sham (20), SAH+Vehicle (30), SAH+4ml/kg CEGI (30), and SAH+1ml/kg CEGI groups (30) and an endovascular perforation model was created to induce SAH. We discovered that the number of TUNEL positive neurons in the hippocampus was markedly decreased in SAH+4ml/kg and SAH+1ml/kg CEGI groups compared to the SAH+Vehicle group. This finding was associated with an observed decrease in Bax/Bcl-2 ratio, cytochrome-c and PUMA expression, and the suppression of caspase-3 activation following SAH. In Morris water maze tests, the SAH+4ml/kg CEGI group demonstrated a decreased escape latency time and increase in time spent in the target quadrant as well as crossing times of platform region. These results indicate that high doses of CEGI can decrease hippocampal neuron apoptosis and relieve cognitive dysfunction in rats, suggesting that multitarget-drug CEGI exhibits a neuroprotective effect in SAH via the mitochondrial apoptosis pathway.
