Background: We recently demonstrated that a cortical stroke induces persistent cognitive impairment associated with secondary neurodegenerative processes in the hippocampus, a region remote from the primary infarct. Interestingly, we found deposition of amyloid-Î² around cerebral vessels at 84 days post-stroke. There is emerging evidence suggesting that deposition of amyloid-Î² around cerebral vessels may lead to cerebrovascular structural changes, neurovascular dysfunction, and disruption of blood-brain barrier (BBB) integrity.
Aim: The aim of the current study was to investigate the spatiotemporal cerebrovascular changes after cortical stroke.
Method: This was done using the photothrombotic stroke model targeting the motor and somatosensory cortices of mice. Cerebrovascular morphology, as well as the colocalization of amyloid-Î² with vasculature and blood-brain-barrier integrity were assessed in the hippocampal regions at 7, 28 and 84 days post-stroke.
Results: Our findings showed the cerebrovascular changes were extended beyond the peri-infarct region to the ipsilateral hippocampus and were sustained out to 84 days post-stroke. Interestingly, we found a decrease in average vessel diameter at 84 days post-stroke and that that vessels with amyloid-Î² deposited around their walls were narrower than those without amyloid-Î² accumulation. Lastly, we demonstrated sustained vascular leakage in ipsilateral hippocampus, indicative of a compromised blood-brain-barrier.
Conclusions: Our findings indicated that cortical stroke induces remote hippocampal cerebrovascular dysregulation, and potentially contributes to the progression of post-stroke cognitive impairment.