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Abstract 7

Cardiomyopathy After Subarachnoid Hemorrhage Is Mediated by Neutrophils

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Background: Patients with subarachnoid hemorrhage (SAH) from the rupture of a cerebral aneurysm often experience severe acute cardiomyopathy. The prevailing theory is that this cardiomyopathy is caused by catecholamine release during the stress of the SAH. Although stress catecholamine release is clearly associated with cardiomyopathy, the rapidity and severity of the onset make it unlikely that this is the only mechanism of injury. There is a great deal of research on the "neuroinflammatory reflex" that suggests that there is direct cerebral control of some aspects of inflammation.

Hypothesis: We hypothesize that inflammation due to catecholamine surges and an unchecked vagal response leads to inflammation of the myocardium after SAH.

Methods: C57B/6 mice were separated into three groups: sham surgery, SAH, and SAH with prior neutrophil depletion. Sham surgery was accomplished by visualization of the dura mater over the occiput of the mouse and injection of 50 μL of saline. SAH was done similarly except that a subarachnoid vein was sectioned instead of saline injection. Neutrophil depletion was accomplished by IP injection of the neutrophil-depleting Ly6G/C antibody RB6-8C5 24 hours prior to SAH. Animals then underwent echocardiography 24 and 48 hours after SAH. Ejection fraction (EF), heart rate, and fractional shortening (FS) were recorded. After the final echocardiogram, the mice were sacrificed and the heart was sectioned and stained with antibodies for neutrophils (7/4) and the myocardial cell death marker (annexin V).

Results: SAH animals had decreased echocardiographic EF and FS compared to controls. Neutrophil depletion ameliorated the cardiomyopathy. Histology showed less myocardial cell death after neutrophil depletion and fewer neutrophils in the myocardium.

Conclusions: Neutrophils appear to play a role in the cardiomyopathy of SAH.

* BHBI = Bakken Heart-Brain Institute

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