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Investigation of Interleukin 6 following Subarachnoid Hemorrhage as a Marker for Vasospasm: Targeting Inflammatory Activation as Potential Treatment

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Background:

Cerebral vasospasm (CV) can contribute significant morbidity for subarachnoid hemorrhage (SAH) patients. A key unknown is how CV induction is triggered following SAH.

Methods:

c57/bl6 wild type and c57/bl6 IL-6 female knockout (KO) mice were utilized with groups: saline injected, SAH, SAH + IL-6 blockade, SAH IL-6 KO, SAH IL-6 KO + IL-6 administration. For SAH, 50um blood was collected from tail puncture and administered into basal cisterns. IL-6 blockade was given at various time points. Various markers of neuroinflammation were measured with western blot and immunohistochemistry. Cerebral blood flow was also measured. Vasospasm was measured via cardiac injection of india-ink/gelatin. Turning test and Garcia's modified SAH score were utilized. $P<0.05$ was considered significant.

Results:

IL-6 expression peaked 3 days following SAH ($p<0.05$). Human IL-6 was increased in aneurysmal blood ($p<0.05$). Receptor upregulation was periventricular and perivascular. A significant increase in BBB markers endothelin 1 and occludin were noted following SAH but reduced with IL-6 blockade ($p<0.01$). CV occurred 5 days post SAH but was absent in IL-6 KO mice and mitigated with IL-6 blockade ($p<0.05$). IL-6 blockade, and IL-6 KO mitigated effects of SAH on cerebral blood flow ($p<0.05$). SAH mice had impaired performance on turn test and poor Modified Garcia Scores compared to saline and IL-6 blockade. A distinct microglia phenotype was noted day 5 in the SAH group (overlap coefficients $r=0.96$ and $r=0.94$) for Arg1 and iNOS, which was altered by IL-6 blockade. Day 7, a significant increase in toll-like receptor 4 and Stat3 were noted. This was mitigated by IL-6 blockade and IL-6 KO, which also reduced Caspase 3 ($p<0.05$). Ventricular dilation and increased tunel positivity were noted day 9 but resolved by IL-6 blockade ($p<0.05$).

Conclusion:

correlation between IL-6 and CV has been well documented. We show that a mechanistic connection exists via the inflammatory response, and IL-6 blockade provides benefit in reducing CV and its consequences.

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Biography:

Brandon Lucke-Wold_was born and raised in Colorado Springs, CO. He graduated magna cum laude with a BS in Neuroscience and distinction in honors from Baylor University. He completed his MD/PhD, Master's in Clinical and Translational Research, and the Global Health Track at West Virginia University School of Medicine. His research focus was on traumatic brain injury, neurosurgical simulation, and stroke. At West Virginia University, he also served as a health coach for the Diabetes Prevention and Management program in Morgantown and Charleston, WV, which significantly improved health outcomes for participants. In addition to his research and public health projects, he is a co-founder of the biotechnology company Wright-Wold Scientific, the pharmaceutical company CTE cure, and was a science advocate on Capitol Hill through the Washington Fellow's program. He has also served as president of the WVU chapters for the American Association of Pharmaceutical Scientists, Neurosurgery Interest group, and Erlenmeyer Initiative Entrepreneur group. In addition, he has served as vice president for the graduate student neuroscience interest group, Nu Rho Psi Honor Society, and medical students for global health. He was an active member of the Gold Humanism Honor Society and Alpha Omega Alpha Honor Society. He is currently a member of the UF House Staff Council and Positive Culture Committee

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