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Evaluation of Soluble Amyloid Beta as a Cause of Post-Stroke Cognitive Impairment and Dementia

Abstract

Chronic cognitive impairment and dementia are prevalent among stroke survivors. Understanding the cellular mechanisms mediating ischemia-induced neuronal dysfunction is critical for developing diagnostic tools and treatment strategies that can be implemented in the intensive care unit and hospital setting to improve the long-term cognition of stroke survivors. Given the coexistence of Alzheimer's disease (AD) pathology with vascular dysfunction, studies are warranted that assess whether neurodegenerative pathways directly cause synaptic dysfunction after an acute ischemic event. This proposal will study the role of amyloid beta, a peptide that accumulates in the brains of AD patients, in mediating the synaptic and cognitive deficits that are present after a large vessel stroke. We anticipate that the findings from this proposal will stimulate future studies investigating the role of amyloid beta in other areas of acute brain ischemia and hypoxia that are relevant to both the ICU and operative environment.