Study examines amyloid deposition in patients with traumatic brain injury

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Patients with traumatic brain injury (TBI) had increased deposits of β-Amyloid (Aβ) plaques, a hallmark of Alzheimer Disease (AD), in some areas of their brains in a study by Young T. Hong, Ph.D., of the University of Cambridge, England, and colleagues.

There may be epidemiological or pathophysiological (changes because of injury) links between TBI and AD, and Aβ plaques are found in as many as 30 percent of patients who die in the acute phase after a TBI. The plaques appear within hours of the injury and can occur in patients of all ages, according to the study background.

Researchers used imaging and brain tissue acquired during autopsies to examine Aβ deposition in patients with TBI. Researchers performed positron emission tomography (PET) imaging using carbon 11-labeled Pittsburgh Compound B ([11 C]PIB), a marker of brain amyloid deposition, in 15 participants with a TBI and 11 healthy patients. Autopsy-acquired brain tissue was obtained from 16 people who had a TBI, as well as seven patients with a nonneurological cause of death.

The study's findings indicate that patients with TBI showed increases in [11 C]PIB binding, which may be a marker of Aβ plaque in some areas of the brain.

"The use of ([11 C]PIB PET for amyloid imaging following TBI provides us with the potential for understanding the pathophysiology of TBI, for characterizing the mechanistic drivers of disease progression or

suboptimal recovery in the subacute phase of TBI, for identifying patients at high risk of accelerated AD, and for evaluating the potential of antiamyloid therapies," the authors conclude.

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