Temporal and spatial map of microglia in primary blast-induced neurotrauma

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Due to recent military conflicts and increased civilian attack, there has been an elevation in the number of blast-induced traumatic brain injury (bTBI) cases. These often result in secondary pathophysiological changes that result in behavioral (e.g., PTSD) and memory (e.g., Alzheimer’s Disease) deficits and in some cases unprovoked seizures (e.g., Epilepsy). A primary blast wave can cause damage and displacement of neurons and their networks and neuroinflammation. The cascade of cellular events that follows exposure to a primary blast is similar to that of other forms of contusion mediated trauma, yet the subsequent pathology may be unique due to the over-pressure of the blast wave over the brain and body. Microglial activation can mediate neuroinflammation following traumatic brain injury. Previous studies support microglial proliferation and activation within hours to months following bTBI, yet a spatiotemporal profile of these changes has not yet been described. The objective of this study was to determine changes in microglia activation and proliferation in response to bTBI at 3 and 14 days following the injury relative to controls (n=3/group). To determine microglial changes we used immunohistochemistry with antibodies against IBA1, a marker for microglia. Densitometry analysis was used to measure differences in the IBA1 immunoreactivity in different brain regions. Our preliminary analysis supports that strong immunoreactivity for microglia is evident in all control and experimental brains. Densitometry analyses are under way to determine potential changes between control and experimental groups.