Secondary brain injury

- Targeted Neuroimmune Modulation via FGF21-Loaded Dual-Layer Electrospun Nanofibrous Scaffold to Suppress Secondary Injury After Severe Traumatic Brain Injury
- Cellular and Molecular Interactions in CNS Injury: The Role of Immune Cells and Inflammatory Responses in Damage and Repair
- Upper Limb Movement Performance in Individuals Sustaining Mild Traumatic Brain Injuries
- Management Challenges of Psychosis and Aggression Secondary to Traumatic Brain Injury: A Report of Two Cases
- Meta-Analysis of Goal Setting and Physical Treatment Categorisation for Focal Spasticity Following Stroke or Other Acquired Brain Injury
- Are isolated linear fractures over major dural venous sinuses a risk factor for sinus thrombosis in mild TBI?
- The potential impact of clindamycin on neurosurgery patients: a randomized controlled trial
- A Customized Neural Transcranial Magnetic Stimulation Target for Functional Disability Among Veterans With Co-Occurring Alcohol Use Disorder and Mild Traumatic Brain Injury: Protocol for a Pilot Randomized Controlled Trial

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Secondary injury is a term applied to the destructive and self-propagating biological changes in cells and tissues that lead to their dysfunction or death over hours to weeks after the initial insult (the "primary injury"). In most contexts, the initial injury is usually mechanical.

Traumatic brain injury (TBI) is associated with secondary injury to the central nervous system (CNS) via inflammatory mechanisms. The combination of polytrauma and TBI further exacerbates the inflammatory response to injury; however, combined injury phenomena have not been thoroughly studied. In a study, Rowland et al., examined the inflammatory differences between patients with TBI versus patients with polytrauma, but no TBI (polytrauma). They hypothesized that patients with TBI have a heightened early inflammatory response compared with polytrauma.

They conducted a single-center retrospective study of a cohort of patients with polytrauma, who were enrolled in the PROPPR study. These patients had blood samples prospectively collected at eight time points in the first 3 days of admission. Using radiological data to determine TBI, the polytrauma cohort was dichotomized into TBI (n = 30) or polytrauma (n = 54). Inflammatory biomarkers were measured using ELISA. Data across time were compared for TBI versus polytrauma groups using Wilcoxon rank-sum test. Network analysis techniques were used to systematically characterize the inflammatory responses at admission.

Patients with TBI (51.6%) had a higher 30-day mortality compared with polytrauma (16.9%) ($P < 0.001$). Expression levels of IL6, IL8, and CCL2 were elevated from the 2-h through 24-h time points, becoming significant at the 6-h time point (IL6, IL8, and CCL2; $P < 0.05$) (Fig. 2). CSF3 showed a similar pattern, but did not attain significance. TBI and polytrauma networks underwent diverging trends from admission to the 6-h time point.

Patients with TBI demonstrated upregulations in proinflammatory cytokines IL6, IL8, and CCL2. Utilizing informatics methods, they were able to identify temporal differences in network trends, as well as uncharacterized cytokines and chemokines in TBI. These data suggest TBI induces a distinct inflammatory response and pathologically heightened inflammatory response in the presence of

polytrauma and may propagate worsened patient outcomes including mortality [1].

Raised intracranial pressure levels increases the risk for secondary brain ischemia and is highly correlated with poor outcome, which highlights the importance of aggressive ICP-directed treatment [2] [3] [4] [5] [6] [7] [8].

The definition of a secondary insult can be intracranial pressure > 20 mm Hg.

Cerebral perfusion pressure < 60 mm Hg.

Systolic blood pressure < 100 mm Hg for 5 minutes or more.

Secondary injury is an indirect result of the insult. It results from processes initiated by the trauma.

Microglial inflammation plays a vital role in intracerebral hemorrhage (ICH)-induced secondary brain injury. IL-17A has been identified to promote microglia activation, but the role in the pathology following ICH remains unclear.

It occurs in the hours and days following the primary brain injury and plays a large role in the brain damage and death that results from traumatic brain injury (TBI).

Unlike in most forms of trauma a large percentage of the people killed by brain trauma do not die right away but rather days to weeks after the event.

In addition, rather than improving after being hospitalized as most patients with other types of injuries do, about 40% of people with TBI deteriorate.

This is often a result of secondary injury, which can damage even neurons that were unharmed in the primary injury. It occurs after a variety of brain insults including subarachnoid hemorrhage, stroke, and traumatic brain injury and involves metabolic cascades.

Secondary injury can result from complications of the injury:

Changes in the blood flow to the brain

Cerebral hypoxia

Cerebral ischemia

Hypotension

Cerebral edema

Intracranial hypertension

Other secondary insults include hypercapnia (excessive carbon dioxide levels in the blood), acidosis (excessively acidic blood), meningitis, and brain abscess.

In addition, alterations in the release of neurotransmitters (the chemicals used by brain cells to communicate) can cause secondary injury. Imbalances in some neurotransmitters can lead to excitotoxicity, damage to brain cells that results from overactivation of biochemical receptors for excitatory neurotransmitters (those that increase the likelihood that a neuron will fire). Excitotoxicity

can cause a variety of negative effects, including damage to cells by free radicals, potentially leading to neurodegeneration. Another factor in secondary injury is loss of cerebral autoregulation, the ability of the brain's blood vessels to regulate cerebral blood flow.

Other factors in secondary damage are breakdown of the blood–brain barrier, edema, ischemia and hypoxia.

Ischemia is one of the leading causes of secondary brain damage after head trauma.

Similar mechanisms are involved in secondary injury after ischemia, trauma, and injuries resulting when a person does not get enough oxygen. After stroke, an ischemic cascade, a set of biochemical cascades takes place.

Secondary brain injury starts after the initial traumatic impact and marked by an increase in the intracellular calcium concentrations.This cascadeeventually results in membrane lipid peroxidation and neuronal cell death.

Brain injury after subarachnoid hemorrhage

Brain injury after subarachnoid hemorrhage.

Oral Care in neurosurgical intensive care units

A study included 18 patients, seven women and 11 men, aged 36-76 years with different neurosurgical diagnoses. The total number of nursing interventions analyzed was 1,717. The most common kind of secondary insults after a nursing measure was high intracranial pressure (n = 93) followed by low cerebral perfusion pressure (n = 43) and low systolic blood pressure (n = 14). Repositioning (n = 39) and simultaneous interventions (n = 32) were the nursing interventions causing most secondary insults. There were substantial variations between the patients; only one patient had no secondary insult. There were, overall, a limited number of secondary insults related to nursing interventions when a standardized management protocol system was applied to reduce the occurrence of secondary insults. Patients with an increased risk of secondary insults should be recognized, and their care and treatment should be carefully planned and performed to avoid secondary insults [9].

Treatment

Secondary brain injury treatment.

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