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Commentary

Causes, Signs and Treatment for Traumatic Brain Injury

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DESCRIPTION

Traumatic Brain Injury (TBI) is a mechanical head injury that results in brain damage and loss of sensation. Symptoms for adults having traumatic brain injury are experiencing weakness, numbness, poor coordination, convulsions, or seizures, vomiting. The severity of TBI is measured using the Glasgow Coma Scale. TBI consists of two distinct phases, Primary and secondary. Primary injury causes focal and diffuse damage and usually causes necrotic cell loss, whereas secondary injury is associated with apoptosis. Mild injuries doesn't require any treatment. Anti-seizure drugs, Coma-inducing drugs, Diuretics will be given to limit secondary damage to the brain immediately. Cognitive impairments that occur after TBI are caused by the loss of neurons and synapses through shearing forces, activation of apoptotic cascades, glutamate excitotoxicity, alterations in neurotrophic signaling, and impaired astrocytic support. TBI may also be a risk factor for development of Alzheimer disease, because these two neurodegenerative states share much common pathology. Excitotoxicity is the cascade of intracellular events initiated by excessive stimulation by neurotransmitters leading to intracellular calcium overload which plays an important role in mediating secondary neuronal injury after TBI. While a variety of neurotransmitters could potentially trigger excitotoxic cell injury, glutamate is thought to be the primary contributor because of its potent effect on increasing intracellular calcium through ionotropic receptors. Microdialysis studies in rodents after both fluid percussion injury and controlled cortical impact have demonstrated a substantial increase in extracellular glutamate proportional to the severity of injury. A number of clinical observations also support the hypothesis that TBI increases extracellular glutamate. A prospective microdialysis study in patients suffering with severe TBI showed an increase in extracellular glutamate in over 75% of patients; interestingly, values normalized over the course of 120 hours in 60% of these patients, but persistent elevations of extracellular glutamate were associated with increased mortality.

Traumatic Brain Injury caused due to transfer of forces to head by hitting an object that cause movement of brain within the skull, or various combinations of mechanical forces. It is now generally accepted that TBI is not a single pathophysiological event but a complex process. It causes structural and functional changes that result in neurological damage. Primary injury is caused by external forces (direct contact with the brain or inertial forces) acting at the moment of injury, resulting in focal, multifocal, or diffuse damage to cerebral vessels, axons, neurons, and glial cells. The type and severity of the resulting injury depends on the nature of the original force. In contrast, secondary injury is an ongoing process that will sustain from minutes to years after primary injury. Secondary injury is the result of a cascade of metabolic, neurochemical, cellular and molecular events resulting from the primary injury. Such mechanisms ultimately lead to brain cell death, plasticity, tissue damage, and atrophy.

Biochemical changes are responsible for secondary damage include disruption of cellular calcium homeostasis, glutamate excitotoxicity, mitochondrial dysfunction, increased free radical formation, inflammation, and increased lipids, diffuse axonal injury and disruption of the blood-brain barrier. In particular, all of the above damaged neuronal tissue releases chemokines, which in turn activate immune cells at the site of injury. A recent study described that differential downstream production of cytokines in enriched human cortical neuronal cultures exposed to levels of cytokines similar to those observed in the brain after traumatic brain injury in humans. The innate immune system is a complex network of cells and signaling mediators that serves as first line of defense against invading pathogens and damage. In case of TBI, in which initial mechanical trauma causes direct damage and astrocytes that initiate a cascade of immune events induced by the release of damage-associated molecular patterns. Stimulation of non-specific immune responses leads to the production of mononuclear phagocytes (peripheral monocyte-derived macrophages and neutrophils) from the blood to the brain and activates resident glia. These cells produce various pro-inflammatory mediators, Reactive Oxygen Species (ROS), and pro-apoptotic proteins that perpetuate neuronal injury, a chronic component of TBI-induced neuropathology.

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