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Poster Presentation

Traumatic Brain Injury

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Abstract

It has been estimated that more than 1.7 million individuals suffer a traumatic brain injury (TBI) each year. TBI causes secondary biochemical and metabolic changes which contribute to subsequent tissue damage and associated neuronal cell death. Two early and delayed events occur after TBI, which results in neurological deficits. Primary injury events include the mechanical damage that occur at the time of trauma to neurons, axons, glia and blood vessels as a result of shearing, tearing or stretching but secondary injury starts over minutes to days and even months after the initial traumatic insult and results from delayed biochemical, metabolic and cellular changes that are triggered by the primary event. Studying the secondary injury cascade could develop therapeutic window for pharmacological or other treatment to prevent progressive tissue damage and improve outcome. Processes such as depolarization, disturbances of ionic homeostasis, and release of neurotransmitters (such as excitatory amino acids), lipid degradation, mitochondrial dysfunction, and initiation of inflammatory and immune processes are some mechanisms that occur in secondary injury event. Through these events large amounts of toxic and pro-inflammatory molecules such as nitric oxide, prostaglandins, reactive oxygen and nitrogen species, and pro-inflammatory cytokines generate, which lead to lipid peroxidation, blood-brain barrier (BBB) disruption and the development of edema. Edema, phenomena that intracranial pressure increases, can contribute local hypoxia and ischemia, secondary hemorrhage and additional neuronal cell death via necrosis or apoptosis. For improving neuroprotective strategies to control and decrease subsequent injurious events after TBI, studying on acute mechanisms in secondary injury event is necessary. With studying on this process, therapeutic window could be opened against a major cause of mortality, traumatic Brain Injury.

Keywords: Traumatic Brain Injury, Edema, Neurological Deficits.

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