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Oral Presentation

Direct and Indirect Insults of Traumatic Brain Injury

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Abstract

In Traumatic Brain Injury (TBI) primary injuries result immediately from the initial trauma. This injury occurs at the moment of trauma and includes contusion, damage to blood vessels, and axonal shearing. The blood brain barrier and meninges may be damaged in the primary injury, and neurons may die. In treatment terms, this type of injury is exclusively sensitive to preventive but not therapeutic measures. The secondary insult (delayed non-mechanical damage) represents consecutive pathological processes initiated at the moment of injury with delayed clinical presentation. After TBI, CBF autoregulation (i.e. cerebrovascular constriction or dilation in response to increases or decreases in CPP) is impaired or abolished in most patients. It is important to note that diagnosing hypoperfusion or hyperperfusion is only valid after assessing measurements of CBF in relation to those of cerebral oxygen consumption. Both cerebral ischaemia and hyperaemia refer to a mismatch between CBF and cerebral metabolism. For example, low flow with normal or high metabolic rate represents an ischaemic situation whereas high CBF with normal or reduced metabolic rate represents cerebral hyperaemia. This 'ischaemia-like' pattern leads to accumulation of lactic acid due to anaerobic glycolysis, increased membrane permeability, and consecutive oedema formation. Since the anaerobic metabolism is inadequate to maintain cellular energy states, the ATP-stores deplete and failure of energy-dependent membrane ion pumps occurs. The second stage of the pathophysiological cascade is characterized by terminal membrane depolarization along with excessive release of excitatory neurotransmitters. This events leads to increase the intracellular concentration of free fatty acids and free radicals, DNA fragmentation and inhibition of DNA repair. These events lead to membrane degradation of vascular and cellular structures and ultimately necrotic or programmed cell death (apoptosis).

Keywords: Traumatic Brain Injury, CBF, Ischaemia.

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