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Ky Parrott
Otterbein University, ky.parrott@otterbein.edu

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**Pathophysiology of Traumatic Brain Injury**

Traumatic Brain Injury (TBI) is an injury to the brain caused by a sudden, significant impact to the cranial vault or a blow to the head from an external force (Jennet & Vilasopo, 2012, p. 282). When force is applied to the head, the brain moves back and forth causing brain fibers to stretch, break blood vessels to bleed, and inflammation to occur (King, Brughall, Hume, & Gossamer, 2014, p. 46). TBIs have primary and secondary brain injury effects. The primary brain injury is the physical damage to the brain and extracranial contents that is immediately sustained at the time of injury due to the direct contact, acceleration-deceleration, and rotational forces (Kauf & Both, 2009, p. 32). Secondary brain injury refers to the cellular processes of molecular pathways caused by the initial damage to the central nervous system (Jennet & Vilasopo, 2012, p. 10). Secondary injuries include neuroinflammation, cell death, ionic disturbances, blood-brain barrier disruption, and mitochondrial toxicity (Jennet & Vilasopo, 2012, p. 10).

This alteration in normal brain function, or neurocognitive cascade, requires energy to re-establish homeostasis. However, immediately following a TBI, there is a decrease in cerebral blood flow and oxygen, and ongoing mitochondrial dysfunction, which impairs energy supply and demand (King et al., 2014, p. 52).

When an individual sustains a head injury, glutamate is released from the presynaptic nerve terminal at an uncontrolled rate, and excitotoxicity occurs, in which neurons become damaged due to persistent glutamate exposure (Tran, 2014, p. 31). After sustaining a head injury, hypoxia occurs, which causes an upregulation of APOT and astrocytes are unable to remove excess glutamate from the extracellular space. The elevated exposure to glutamate leads to the activation of the calcium channel N- methyl-D-aspartate (NMDA), triggering mitochondrial dysfunction and intracellular calcium extrusion (Tran, 2014, p. 31). When intracerebral extracellular calcium exceeds the extracellular rate, calcium is segregated within the mitochondria to maintain intracellular homeostasis. Mitochondria are sensitive to calcium fluctuations and excess calcium stimulates the opening of the mitochondrial permeability transition pore (mPTP) (Tran, 2014, p. 31). This disruption triggers more calcium to invade the mitochondria, which in return causes the mitochondria to swell and burst. When the mPTP is open, cytochrome c is released and interacts with apoptosis protein activator factor-1 and forms apoptotic. Apoptosis provides avenues to apoptosis and degrades the cells to apoptosis, or regulated cell death (Tran, 2014, p. 32).

Concussion

It is estimated there are 1.6-3.8 million annual sports related concussions, or mild traumatic brain injuries (mTBI) (King et al., 2014, p. 46). In individuals 12 to 24 years, sports related mTBI is the most common form of TBI (King et al., 2014, p. 449). Concussion injuries occur in both men and women and can occur to individuals of all ages. Sports related mild traumatic brain injuries could be attributed to all athletic activities including football, soccer, basketball, hockey, non-contact hockey, lacrosse, wrestling, rowing, softball, and cheerleading.

While considered a “mild” form of a TBI, concussions, especially subsequent concussions, can be detrimental. The severity of the signs and symptoms then determine whether the concussion is mild, moderate, or severe. Common symptoms for a mTBI include headache, amnesia, confusion, dizziness, numbness or tingling to extremities, light and noise sensitivity, dizziness, nausea, loss of balance, blurred vision or changes in sleeping patterns (Mason, 2013, p. 205). Late concussion symptoms include difficulties with memory or concentration problems, and increased aggression or a short temper (Mason, 2012, p. 209). These symptoms may last for weeks or months. Individuals who experience multiple concussions are at a significantly higher risk for developing a severe TBI, and fail to show substantial symptoms for an extended period of time (Mason, 2013, p. 209).

While these symptoms could last from days to months, individuals should be closely observed for the first few days following a mTBI. Individuals should be monitored for post-concussion changes, return, vomiting, sleep changes, neck pain, worsening headache, loss of consciousness in significant change in mental status, or these considered red flags and should be considered emergency situations (Mason, 2013, p. 209). Once an individual has received a concussion they should practice prevention of future mTBIs to minimize long-term complications.

**Conclusions**

Sports related traumatic brain injuries affect an estimated 1.6 to 3.8 million people each year (Mason et al., 2014, p. 459). While these injuries are frequently overlooked as minor, the long-term consequences can be detrimental to the individual and their families. Those who suffer multiple mTBIs in their athletic career are at significant risk for functional, psychological, and cognitive impairments. The impact of the education regarding the pathophysiology behind mTBI is imperative to the individual and understanding the changes occurring in the brain to cause cognitive impairment. Athletes, families, friends, coaches, and teachers should also be educated regarding signs and symptoms of mTBIs or the risks associated with multiple concussions, and prevention strategies for future mTBIs. As Advanced Practice Nurses, we can empower our community of educational and resources to adequately diagnose and manage traumatic brain injuries in our athletes.