Mini Review

Should a History of Mild Traumatic Brain Injury Alter Anesthetic Management?

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Introduction

Mild Traumatic Brain Injury (mTBI) is a prevalent injury with long-lasting neuropathologic and neurophysiologic effects. The Centers for Disease Control and Prevention (CDC) estimates that mTBI accounts for >75% of the 1.5 million cases/year of TBI at a cost of $17 billion/year [1]. The average incidence of mTBI has been estimated as 0.05% in the US population [2]. Military cases of mTBI may account for an additional 330,000 cases/year [3]. mTBI is not uniformly defined. It is usually defined based on clinical and brain imaging findings. mTBI may be defined as “traumatically induced structural injury or physiological disruption of brain function as a result of an external force, with normal CT [Computed Tomography] structural imaging, loss of consciousness <30 min, alteration of mental state<24 h, post-traumatic amnesia <1 day, and Glasgow Coma Score of 13-15” [4]. Other brain imaging modalities may be more sensitive than CT scan in detecting changes after mTBI. For example, Magnetic Resonance Imaging (MRI) with diffusion tensor imaging and Blood Oxygen-Level Dependence (BOLD) imaging may demonstrate significant changes after concussion; even with negative CT scanning [5]. This indicates that mTBI is an under-recognized problem with both acute and chronic physiologic changes with clinical implications.

Acute mild traumatic brain injury

mTBI significantly alters Cerebral Blood Flow (CBF) and metabolism. There is acute loss or attenuation of CBF autoregulation and responsiveness to CO2 levels [6]. These changes increase the risk of cerebral hypoperfusion and cerebral ischemia with moderate levels of hypotension and hyperventilation [7,8]. This loss of CBF autoregulation may last for 2 weeks after mTBI [9]. This susceptibility to hypoperfusion and cerebral ischemia may exist without gross head trauma or alteration in Glasgow Coma Score (GCS) and supports considering the lower limit of CBF autoregulation to be 70 mm Hg rather than 50 mm Hg [10]. Even a single incidence of mTBI may alter Cerebral Metabolic Rate (CMR) and function resulting in clinical and radiographic findings. Some of these findings may be evident only with sensitive imaging and clinical testing modalities such as BOLD signals of MRI and correlative neurocognitive testing for the affected area, such as the dorsolateral prefrontal cortex which is critical for cognitive function. Imaging changes may correlate with symptom progression or improvement [11].

Chronic or repeated mild traumatic brain injury

Chronic Traumatic Encephalopathy (CTE) refers to clinical and pathologic signs of neurodegeneration that occur in people with repeated mTBI, including athletes and military personnel. A study by the Center for the Study of Traumatic Encephalopathy (CSTE) at Boston University showed that 80% of patients with chronic mTBI have post-mortem neuropathologic changes of CTE including pathologic accumulation of amyloid-β peptide (A-β) and tau protein [12]. Animal models of CTE have replicated human pathologic and metabolic findings including accumulation of A-β and tau and a decrease in CMR for glucose (CMRg) in the parietal and hippocampal areas [13]. Furthermore, animal studies have demonstrated that inhalation anesthetic drugs may accelerate or induce neurodegenerative changes such as caspase activation and accumulation of A-β and tau [14,15]. This evidence of inhalation anesthetic-induced acceleration of neurodegenerative changes may present an additional concern for administering general anesthesia for patients with chronic mTBI or CTE who might have neurodegenerative changes.

Conclusion

Increased recognition of the prevalence and impact of mTBI and CTE is demonstrating the need for more research in this area including guidelines for anesthetic care for patients with these
disorders [16]. Current understanding of mTBI-induced changes in CBF autoregulation, vascular CO2 reactivity and CMR demonstrates increased susceptibility of mTBI patients to hypoperfusion and cerebral ischemia. Furthermore, the possible role of inhalation anesthetic-induced acceleration of neurodegeneration may present an additional risk of CTE patients with evidence of neurodegeneration. It is crucial, in the setting of any trauma, to inquire about any mTBI event. And in cases of suspected or evident mTBI, there is evidence to support maintaining MAP>70 mm Hg. If an ICP monitor is available, the current Brain Trauma Foundation (BTF) Guidelines recommend maintaining CPP between 50-70 mm Hg and ICP below 20 mm Hg [17]. It is less clear at this time if any recommendations can be made regarding avoiding inhalation anesthetics in patients with repeated mTBI or CTE.

References