Traumatic Brain Injury

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Introduction

Traumatic brain injury (TBI) remains a significant problem in both the pediatric and adult populations. Approximately 500,000–700,000 pediatric TBI incidents and 2.5 million adults are suffered from TBI every year [1-4]. TBI is ranged from mild to severe and its management depends of the severity of the injury. The injuries from TBI can be classified into primary and secondary injury. Primary injury, which is considered irreversible but preventable, is the injury took place at the time of trauma such as the direct damage to the parenchyma of the brain, producing axonal shear and rupture. On the other hand, the secondary injury defines as the injury occurring following the initial TBI, ranging from inflammation that can increase intracranial pressure (ICP), which in turn causing further damages. Therefore, the goal of most medical management after TBI is to decrease the insult caused by secondary injury with the aim of maximizing the patient’s recovery from the primary insult [5]. Similar treatments are also employed in pediatric patients with TBI. Preventing of secondary injury is even more important pediatric population given their brains still have significant potential for plasticity [6].

Concussion is one of the most common pediatric injuries. Its symptoms vary depending on the given situation and can include cognitive challenges, sleep disturbances, headaches, and ataxia. Headache is one of the most disabling symptoms that patients often complain about after a concussion [7]. Children often need a longer rehabilitation time post-concussion compared to adults [8,9].

In the case of trauma patients with a suspicion of TBI, usually computed tomography (CT) is the imaging of choice given its high sensitivity for blood and the short duration of scanning. However, in certain mild TBI cases imaging may not be needed. The classically used “Canadian CT Head Rule” is a useful guide to follow to help deciding the need for a CT of the head in the setting of mild TBI [10].

The coagulation panel that includes Normalized Ratio or PT/INR, and activated Partial Thromboplastin Time or aPTT should be sent on admission. A prompt correction of a patient’s INR to 1.3 or lower has been shown to decrease hematoma expansion and improves outcomes [11]. While platelet transfusion is recommended for thrombocytopenia (platelet count<100,000) for patients with intracranial hemorrhage, others have found that its use to counter the effect of antiplatelet agents, such as aspirin or clopidogrel, may not improve outcome [12]. Novel new agents for reversal of newer anticoagulants, such as direct thrombin inhibitors or Factor Xa inhibitors are promising, such as idarucizumab for dabigatran reversal [13] or andexanet alfa for reversal of anti-Factor Xa agents [14].

Monitoring of ICP is recommended for all TBI patients without clinically followable neurological exams [15] or whose GCS<8 with an abnormal CT scan of the head [16]. Most institutions use the external ventricular drain (EVD) or the fiber optic intraparenchymal probe to record ICP. Due to the loss of cerebral blood flow autoregulation at high ICP, which can lead to hypoperfusion of the brain, ICP must be kept below 20 mmHg, commonly through the employment of sedation and hypertonic therapy [17].

Decompressive craniectomy is often employed as a last resort to treat medically intractable ICP [18-21]. It has been showed in the DECRA trial that improvement ICP control and shorter stay in ICU in patients treated with the bilateral frontotemporoparietal craniectomy, but this also led to more unfavorable outcomes based on the extended glasgow outcome scale (GOS), which was believed to be secondary to the improvement in the survival of vegetative patients [22]. In consistent with this, a recent randomized study called RESCUEicp demonstrated that decompressive craniectomy in patients with TBI resulted in a lower mortality rate at 6 months compared to medical treatment alone[23,24].

References


