Dr Adam Humble April 2020

What is the role of decompressive craniectomy in the setting of TBI?

Traumatic brain injury (TBI) is a major problem worldwide and particularly in low and middle income counties. TBI and the long term disabilities associated with it pose significant challenges to survivors, their caregivers and the health care system. A potentially devastating consequence of TBI and significant predictor of morbidity and mortality is elevated intracranial pressure¹. Elevated intracranial pressure occurs when there is expansion of intracranial volume within the enclosed inelastic container of the skull. First line treatment for elevated ICP involves reducing the intracranial volume, that is, the blood, brain cells, intra/extracellular fluid and the cerebral spinal fluid. When medical intracranial volume reduction therapy is exhausted the skull itself can be targeted in what is known as a decompressive craniectomy (DC).

In a decompressive craniectomy, a large portion of the skull is removed to allow for the underlying brain tissue to swell and thus reduce the intracranial pressure and improve cerebral perfusion. The location and amount of skull removed depends predominately on the site and size of brain injury. There are two distinct clinical scenarios in which decompressive craniectomy is performed. Primary, or prophylactic DC is done when a patient undergoing an operation for evacuation of a dural lesion is expected to have post surgical elevated ICP. Secondary DC is done when patients with continuous ICP monitoring have elevated ICP refractory to medical therapy. DC has a long history dating back to 1894 and the pendulum has swung back and forth regarding its role in the management of elevated ICP in TBI². The most relevant and highest quality of the studies investigating the role of DC in TBI will be discussed here.

Decompressive Craniectomy in Diffuse Traumatic Brain Injury by Cooper et al. (DECRA Trial) was a large randomized trial in 2011 aimed at determining the efficacy of DC in refractory elevated ICP. 155 adult patients with severe TBI and ICP greater than 20mmHg for 15 minutes despite first line therapy were assigned either DC or continuing medical therapy. If the patient required surgical evacuation of an intracranial lesion they were excluded. Although DC was associated with shorter ICU stays and lower ICP, DC group had worse outcomes on the Extended Glasglow Outcome Score at 6 months and mortality was the same between groups ³. DECRA has been criticized for its low threshold for inclusion of 15 minutes of ICP greater than 20mmHg . The extensive bilateral craniectomy used in the DECRA trial is also criticized as not being reflective of typical practice.

RESCUEicp by Hutchinson et al. was conducted to further evaluate the role of DC in TBI and address the shortcomings of DECRA. Patients aged 10 to 65 years old with TBI who had raised ICP greater than 25mmHg for 1 to 12 hours despite medical therapy were treated with either medical therapy or DC. In contrast to the DECRA trial, patients requiring surgical evacuation of a hematoma were included. DC was associated with lower mortality however the surgical group had a greater proportion of survivors with unfavorable neurologic outcomes. Both groups showed similar rates of good outcomes. The number need to treat to avoid death at one year was five. RESCUEicp took place over 10 years, in which practice changed making the population somewhat heterogenous. 37% of the medical treatment arm received DC potentially impacting results⁴.

Data in the pediatric population is limited. A literature review looking at 78 cases of pediatric TBI who received DC within 24 hours showed favorable results in terms of immediate ICP control and long term follow up. Rankin scores on follow up at 48 months showed a median of 2, meaning slight disability⁵. A small single center randomized control trial from Taylor et al in 2001 showed early DC resulted in lower

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ICP and potentially better neurologic outcome at 6 months although statistical significance was not reached 6 .

Overall, there is limited high quality evidence for who should receive DC and what results can be expected. In adults with ICP greater than 25mmHg who are refractory to medical treatment, based on RESCUEicp trial, DC will lower ICP and reduce mortality at 6 months at the cost of potential poor neurologic outcome. It is important for the care providers to recognize that the definition of a "bad outcome" is different for every patient and their families. It is crucial to express to families and decision makers that although DC may improve survival, there is potential for severe neurologic disability, so that families can make a decision in line with the patient's values. The studies discussed here use ICP as a trigger for intervention; however, as mentioned earlier, the highest burden of TBI is in middle and low income countries where access to ICP monitoring is limited if at all present, so the global applicability of these studies is limited.

- 1. Sahuquillo J, Dennis JA. Decompressive craniectomy for the treatment of high intracranial pressure in closed traumatic brain injury. Cochrane Database Syst Rev 2019 December 31;12:CD003983.
- 2. Schirmer CM, Ackil AA, Malek AM. Decompressive craniectomy. Neurocrit Care 2008;8(3):456-470.
- 3. Cooper DJ, Rosenfeld JV, Murray L, Arabi YM, Davies AR, D'Urso P, et al. Decompressive craniectomy in diffuse traumatic brain injury. N Engl J Med 2011 April 21;364(16):1493-1502.
- 4. Hutchinson PJ, Kolias AG, Timofeev IS, Corteen EA, Czosnyka M, Timothy J, et al. Trial of decompressive craniectomy for traumatic intracranial hypertension. N Engl J Med 2016 September 22;375(12):1119-1130.
- 5. Elsawaf Y, Anetsberger S, Luzzi S, Elbabaa SK. Early decompressive craniectomy as management for severe TBI in the pediatric population: A comprehensive literature review. World Neurosurg 2020 February 18.
- 6. Taylor A, Butt W, Rosenfeld J, Shann F, Ditchfield M, Lewis E, et al. A randomized trial of very early decompressive craniectomy in children with traumatic brain injury and sustained intracranial hypertension. Childs Nerv Syst 2001 February 01;17(3):154-162.