Decompressive Craniectomy in Traumatic Brain Injury: The Edge Effect

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Decompressive Craniectomy in Traumatic Brain Injury: The Edge Effect

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A 25-year-old male presented with history of blunt trauma to the head with decreased level of consciousness and computed tomography (CT) evidence of a left acute subdural hematoma with 2.2 cm of midline shift, and no intra-parenchymal lesions. The patient underwent a left decompressive craniectomy with evacuation of the subdural hematoma. No venous injury was noticed intra-operatively. Post-operatively the patient remained difficult to rouse, off all sedatives, five days later.

Magnetic resonance imaging (MRI) including diffusion weighted imaging (DWI) was conducted to assess the degree of ischemic insult to the brain. Diffusion weighted imaging demonstrated hyperintense signal throughout the left hemisphere (Figure 1). The cortex was herniated 3.25cm through the craniectomy site. The highest intensity noted on DWI was focused around the edges of the craniectomy at the superior and inferior margins of the bony decompression (Figure 1). This was confirmed to indicate true restricted diffusion on apparent diffusion coefficient (ADC), negating the likelihood of T2 weighted shine through (Figure 2). Fluid-attenuated inversion recovery (FLAIR) and gradient echo (GRE) sequencing failed to demonstrate hemorrhage in these areas.

Craniectomy for diffuse cerebral edema in the setting of trauma is a controversial topic in the treatment of medically refractory elevated intracranial pressures. Randomized prospective trials to date have demonstrated inconclusive mixed results in terms of clinical outcome[1-3]. The findings above suggest an ischemic "edge effect" that has been described sparingly in the literature[4]. Some of the MRI findings (left

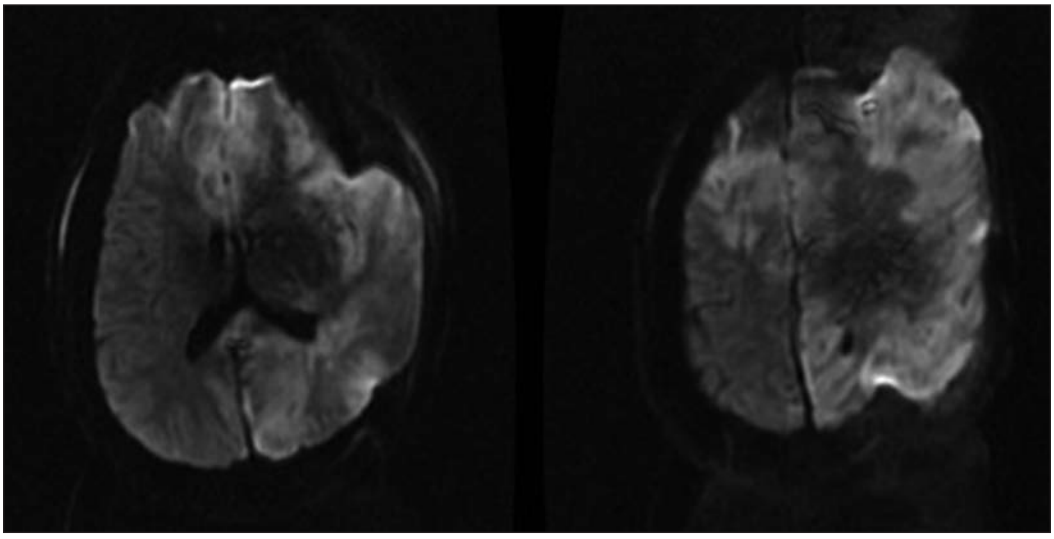

Figure 1: *DWI at inferior (left) and superior (right) of craniectomy site demonstrating hyperintensity at boney edges, suggestive of restricted diffusion, and ischemia.*

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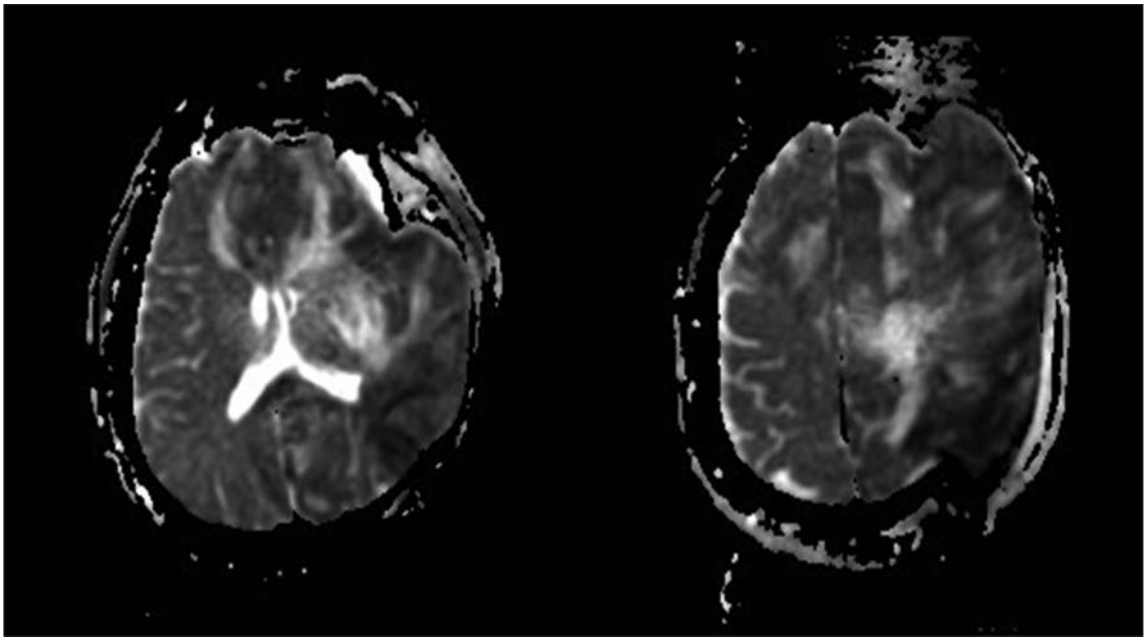

Figure 2: *ADC maps demonstrating cerebral hypo-intensity at the craniectomy edges, confirming restricted diffusion at boney margins described in Figure 1.*

occipital and lateral cortical restricted diffusion) cannot be explained in their entirety due to cortical compression at the craniectomy edge, and may relate to cortical compression from the subdural hematoma pre-operatively.

Complications associated with craniectomy in this setting include the theoretical risk of axonal stretch and ischemia (thought to be venous in nature) associated with the herniating brain at the boney margins of the craniectomy[4]. Currently we know that there is improvement in partial brain tissue oxygenation ($PbtO_2$)[5,6], cerebral microdialysis[6], and pressure reactivity indices[6] through invasive monitoring post-craniectomy for trauma. This has yet been correlated to improved clinical outcome.

We have a poor understanding of the physiological effects on herniated brain at the edges of a craniectomy site. We know that small movements in axonal position, as in diffuse axonal injury, can lead to devastating neurological consequences. The toll that extensive axonal stretch post-craniectomy has on the neurologic outcome is unknown. In addition, the degree of ischemia at the craniectomy edge has yet to be well described. Despite DWI evidence here of focal restricted diffusion at the craniectomy site edges, it is not known what physiological changes are taking place. Furthermore, this entity has yet to be correlated to clinical outcome.

With increasing availability of the MRI capabilities of tractography and functional blood oxygen level dependent (BOLD) sequencing, hopefully in the near future further light will be shed on the significance of the “edge effect”.

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