Fatima N, J Nucl Med Radiol Radiat Ther 2019, 4: 012
DOI: 10.24966/NMRR-7419/100012

HSOA Journal of
Nuclear Medicine, Radiology & Radiation Therapy

Editorial

Neuroimaging in Decompressive Craniectomy in Traumatic Brain Injury

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Editorial

Intracranial pressure elevation and ultimately reduction in the cerebral perfusion pressure is the pathophysiological mechanism that occurs following head trauma. This would successively cause detrimental effects on cerebral oxygen metabolism and can lead to catastrophic events [1-3]. In addition, brain edema is an independent prognostic factor in traumatic brain injury with a mortality of over 10 times in patients with documented brain edema [4].

Therefore, Decompressive Craniectomy (DC) is considered to provide instantaneous and definitive relief of raised Intracranial Pressure ICP [5,6]. In most of the cases, DC is performed following the protocol for the treatment of refractory intracranial edema and hypertension as a secondary procedure (Secondary Decompressive Craniectomy) [7,8]. Timing of the DC (early vs. late option) plays an important role as it may change the pathophysiological responses. It is reported that the right time for DC is by clinical follow up, repeated CT scans, and continuous ICP and CPP monitoring [9,10].

CT scan is relatively cost-effective imaging modality, compatible with life support and monitors instrumentation surgical clips and implants and suitable in trauma settings that can be performed in the early postoperative period to detect potential complications. Although it has been perceived that the extra-axial collection is difficult to detect because of the artifact caused by the overlying calvarium, this issue has been resolved using multidector state of the art CT technology. Magnetic Resonance Imaging MRI which is more sensitive than CT in the postoperative period, however its use is contraindicated due to MRI incompatibility with life support or surgical applied instrumentation [7].

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Citation: Fatima N (2019) Neuroimaging in Decompressive Craniectomy in Traumatic Brain Injury. J Nucl Med Radiol Radiat Ther 4: 012.

Received: May 06, 2019; **Accepted:** May 08, 2019; **Published:** May 15, 2019

The objective of this editorial review was to examine the role of pre- and post-operative CT scan findings in patients with severe closed head injury who underwent DC. Thus, determining the correlation of radiographic features in predicting clinical outcome of patients at 6-months of clinical follow up.

Traumatic brain injury is a leading cause of morbidity and mortality. It is considered that cerebral contusion after trauma induces the life-threatening brain swelling within 2-3 hours. Second peak of brain swelling occurs within 2-5 days due to blood cell breakdown products and activated inflammatory cascades. So, surgery should be performed as soon as possible, not after 5 days of occurrence [1,8]. The neurological assessment of patients in postoperative period can be altered due to sedation, intubation and ventilation. Therefore, CT scan is considered to be an important tool in determining the clinical status of patients.

Diagnostic Efficacy of Post-Operative CT Scans

In acute conditions of traumatic brain injury, early diagnosis and aggressive management may help in prevention of secondary brain damage. Thus, improving the prognosis of the patients and reducing the hospital stay and health care costs [11]. CT scan has diagnostic role by identifying the cerebral and cranial pathology. In addition, it provides anatomic localization in neuronavigation, which helps in planning the skin incision and guide in placement of burr holes. CT scan also has prognostic implication that helps in deciding the aggressiveness of the management plan [11].

Whereas in the chronic management of head injury, neuroimaging through CT scan helps in identifying the post-operative changes in the neurophysiology by alteration in the cerebral blood flow and cerebrospinal fluid, therapies to prevent the secondary brain damage, long-term prognosis of the patients [12,13]. This provides information for a multi-disciplinary approach toward management of patients with severe head trauma.

Complications following Decompressive Craniectomy

There is alteration in the cerebral compliance, cerebral auto regulation, cerebral blood flow, and CSF circulation as a result of decompressive craniectomy [10]. The rate of complications increases after removal of bone flap ultimately leading to increase mortality and morbidity. Expansion of the hematomas, new sub dural or epidural hematomas contralateral to the DC, external cerebral herniation, and sub duralhygroma, paradoxical herniation can develop [11]. Later patient can develop syndrome of trephined which include neurological, cognitive and psychiatric deficit. The most serious and fatal outcome is the persistent vegetative state [14].

XJ Yang et al [15] found that after decompressive craniectomy in patients with traumatic brain injury, the incidence of shunt dependent hydrocephalus, sub dural fluid collection, and CSF leakage from the scalp incision has increased tremendously. Scalp swelling in the early post-operative period is the most common finding as it is composed of edematous fluid, hemorrhage, cerebrospinal fluid (CSF) and air, in different amounts. It resolves over several weeks [16].

Expansion of hemorrhagic contusions

Following the removal of bone flap, there is loss of tamponade effect leading to ipsilateral or on rare circumstances contralateral expansion of hematoma [14,18]. This expansion of hematoma in turn is associated with poor clinical and functional outcome of patients. Flint et al [17] found that there is a higher incidence of new or expanded hematoma following decompressive craniectomy in traumatic brain injury. 81.5% of this new hemorrhage was ipsilateral to the hemicraniectomy.

Post-operative sub dural effusion

Subdural effusion was defined as a newly appearing subdural fluid collection on serial cranial CT scans [19]. The incidence of sub duralhygroma is relatively common after decompressive craniectomy. This is mainly due to alterations in the cerebral perfusion pressure [21]. Most of these sub duralhygromas resolves spontaneously but the fluid collection can increase for up to 1 month. This can lead to midline shift and thus can impair the cognition.

Post-traumatic hydrocephalus

Post-traumatic hydrocephalus occurs within 6 months after surgery, with ventricular dilation not due to brain atrophy associated with neurological deterioration [19]. Posttraumatic Hydrocephalus can develop after decompression especially if there is hemorrhage and infection [18]. Choi et al. [22] reported an incidence of posttraumatic hydrocephalus of 4 % among patients receiving conservative management versus 20.7% among patients who underwent decompressive craniectomy. Honeybul et al.[23] in a prospective observational cohort study concluded that after severe traumatic brain injury, the mechanical complications including brain herniation, subdural effusion, and hydrocephalus requiring ventriculo-peritoneal shunt are more common.

External cerebral herniation

External cerebral herniation was defined as extension by more than 1.5 cm of brain tissue through the center of the craniectomy skull defect [19]. Following decompression, there is herniation of the brain tissue through the skull defect as a result of increased Trans capillary leakage of edema fluid or hyper perfusion of the brain tissue [14]. This ultimately leads to pinching of the cortical veins or laceration of the brain tissue through the defect edge, which causes ischemia and necrosis [18,24].

Basal cisterns

Toutant et al.[25]1984 assessed the appearance of basal cisterns in 218 patients with severe traumatic head injury. The mortality rates were 77%, 39%, and 22% among those with absent, compressed, and normal basal cisterns, respectively. Yanaki et al. [26], in a retrospective study showed a positive predictive value of 77% to unfavorable outcome in the presence of compressed basal cisterns.

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