Editorial

Neuroimaging in Decompressive Craniectomy in Traumatic Brain Injury

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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 decompression. 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].

XI 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 subdural hygroma is relatively common after decompressive craniectomy. This is mainly due to alterations in the cerebral perfusion pressure [21]. Most of these sub dural hygromas 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 dilatation 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.

References
