Decompressive laparotomy for treatment of refractory intracranial hypertension, thinking out of the box

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Intracranial pressure management is the corner stone to the success of treatment of traumatic brain injury (TBI).[1] The management strategy of severe TBI is variable by institution and region of practice but entails successive utilization of head elevation, sedation, paralysis, CSF drainage, hyperosmolar therapy, hyperventilation, and barbiturate coma.[2] The ultimate escalation of therapy is the utilization of decompressive craniectomy in cases of refractory intracranial hypertension.[3] The relationship between intracranial, intra-thoracic, and intra-abdominal pressure and pressure translocation has been known for some time.[4] This is a physiological factor seldom involved in the approach to refractory intracranial hypertension, which could be secondary to abdominal compartment syndrome (ACS). We present such a concept through the following case of a polytrauma victim.

A 22-year-old male who was involved in a high-speed motor vehicle crash with an initial Glasgow coma scale (GCS) of 7 and a left dilated pupil. He was intubated in the field and suffered a brief episode of ventricular fibrillation with a systolic blood pressure of 80 mm Hg en-route to our hospital. A CT of the brain showed multiple contusions in the frontal lobes bilaterally as well as the right parietal lobe. There was a small right frontotemporal subdural hematoma with effacement of the sulci. His CT abdomen showed a splenic blush for which he was submitted to endovascular angio-embolization. While in the angio-suite, he sustained a generalized tonic-clonic convulsion lasting a couple of minutes for which he was started on anti-epileptic medication (phenytoin 1g loading followed by 100 mg IV TID, Phenytoin sodium, Mylan Laboratories, Morgantown, WV, USA). Additional injuries included multiple facial fractures as well as pelvic and femur fractures. He was transferred to the intensive care unit where a fiber optic intracranial pressure monitor (Codman®, The DePuy Company, Johnson & Johnson Family of Companies, Warsaw, IN, USA) was inserted. Initial intracranial pressure (ICP) was controlled by hyperosmolar therapy. Repeat imaging in 24 hours showed no interval changes, and the ICP trends were stable. On post-trama day 4, the ICP was controlled, but he was noted to have a tense abdomen. His fluid balance was positive since admission by 2.3 Lt. On post-trauma day 5, he had an ICP surge up to 40 mm Hg at 3:00 am that responded to an additional dose of mannitol. A repeat CT scan carried out urgently showed no change in the contusions, the peri-contusion edema, or the small subdural collection. The ICP was stable for the following few hours but he had a tense abdomen and was exhibiting a positive hepatojugular reflex, with an ICP rising from a baseline of 12-15 to 38-40 mm Hg, and a bladder pressure of 29 mm Hg. He had a positive fluid balance of several liters by this time, and the general surgery team was consulted to rule out an ACS. At 12:30 pm on the same day, the patient had an ICP surge to 70 mm Hg with bilateral dilated pupils. This plateau wave failed to respond to additional doses of mannitol, hypertonic saline, and hyperventilation. Given the recent CT findings of stable intracranial contusions and the objective finding of fluid overload and signs of increased intra-abdominal pressure, an emergency bedside laparotomy was performed by the general surgery team. The ICP decreased immediately to 24 mm Hg with normalization of the pupil size. A repeat CT showed no interval changes. He was then transferred to the operating room for a completion laparotomy and proper seal of the wound. By this time, the ICP was between 10 and 15 mm Hg. He had a persistent hypotensive period despite aggressive fluid resuscitation with mean arterial pressure in the 60's with an ICP trending upwards of 20-30 mm Hg. Because of his poor hemodynamic condition, and fear of compromised cerebral perfusion, he was submitted to a left sided decompressive craniectomy with a significant drop in the ICP to 5-7 mm Hg. This expedited weaning of hyperosmolar therapy, which optimized the resuscitation protocol for his hemodynamic instability. He developed an abdominal wall hernia, which was treated by surgical closure with mesh. This developed an infection and was revised and treated with antibiotics for 8-weeks post-operatively, with eventual good wound healing.

He continued to improve neurologically with no ICP derangement. He was discharged to a rehabilitation center and later returned home where he lives with his family yielding a Glasgow outcome scale of 4, and can attend to simple responsibilities in the household. He underwent an uneventful cranioplasty 11 months after his TBI.

Disclosure. The authors declare no conflicting interests, support or funding from any drug company.

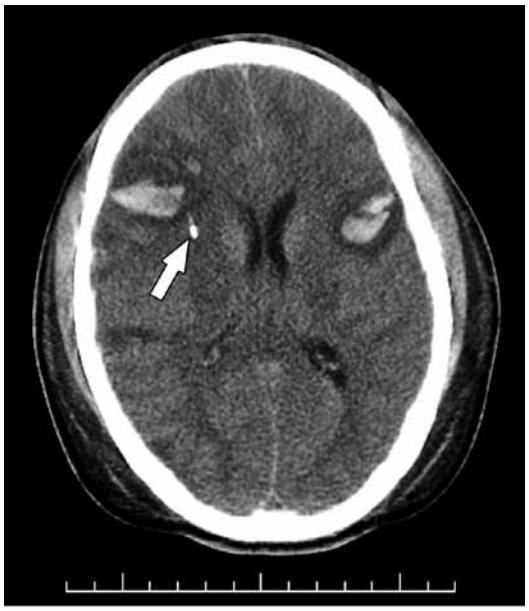

Figure 1 - A non-contrast CT scan of the brain showing bilateral frontal contusions and limited pericontusional edema with preserved gray-white matter differentiation across both cerebral hemispheres. A fiber optic intracranial pressure sensor is noted in the right frontal lobe (arrow).

Polytrauma patients represent a challenging cohort from the standpoint of management of their TBI.[5] Conventional ICU strategies for managing polytrauma patients employ aggressive fluid therapy allowing for liberal administration of resuscitation fluids resulting in a significant overload that has the potential for increasing the intra-abdominal pressure. The other strategy employed in polytrauma victims is that of ventilatory support, which utilizes higher airway pressure for maximal alveolar recruitment to optimize gas exchange and improve overall oxygenation. Abdominal compartment syndrome is a clinical entity with physiological derangement secondary to raised intra-abdominal pressure.[6] It generally manifests as worsening respiratory failure, decreasing cardiac output, inadequate renal perfusion, and oliguria. The abdomen is usually distended, and the bladder pressure is elevated above 30-35 mm Hg. It is known that elevation of intra-abdominal pressure displaces the diaphragm cephalad and leads to elevation of the intra-thoracic pressure, which impairs cerebral venous outflow. This impairment of the jugular venous system out flow results in an increased ICP and reduces cerebral perfusion pressure with worsening of the intracranial homeostasis.[7]

The interconnection of body cavities; namely, abdominal, thoracic, and intracranial compartments, and their pressure translocation has been documented in several animal and human studies.[5] In our patient; the brunt of the trauma involved the abdominal compartment with a significant splenic injury resulting in hemodynamic instability from the outset requiring aggressive fluid management. At the same time, his intracranial injury (scattered small contusions) would generally be associated with ICP elevations in a range correctable by moderate hyperosmolar therapy (**Figure 1**). Despite a stable intracranial injury as documented by serial imaging, he continued to suffer significant ICP plateau waves. This occurred in the context of evidence for a progressive increase in the intra-abdominal pressure. The choice of decompressive hemicraniectomy was deferred, in light of the bilateral pathology, requiring theoretically a bilateral hemispheric craniectomy or a bifrontal craniectomy to address both hemispheres, an approach known for its increased morbidity.[8]

Unexplained worsening of the ICP in a polytrauma victim should be considered an early sign of ACS, warranting the measurement of the bladder pressure and a general surgery consultation for earlier evaluation and follow up. In patients with ACS and TBI, the optimum cut-off for bladder pressure is not known, but lowering the intervention trigger for management to 20 mm Hg is found to be the lowest for sustainable control of the ICP and improvement of outcome. When a full-blown ACS occurs, the benefit of abdominal release in terms of ICP control is diminished.[7] Decompressive laparotomy may be achieved by a formal opening of the fascia and interval closure of the ventral hernia.[7] Depending on the timing of application and the extent of intracranial injury, 4 ICP response patterns could be identified; immediate and sustained reduction of ICP, delayed, sustained reduction of ICP, no reduction of ICP, and an initial reduction with rebound elevation of ICP.[4,7] Patients with no response or rebound intracranial hypertension tend to be older with lack of reduction of central venous pressure post laparotomy denoting a persistent driving force of the intracranial hypertension. However, it could be attributed to the advanced stage of the cerebral trauma and edema, due to the prolonged pressure translocation into the intracranial compartment allowing for secondary brain tissue injury to enter a self-sustaining vicious circle that fails to yield even if the pressure is released. Thus, even with temporary alteration of the natural progression of the pathological process, the intracranial pressure rebounds requiring directed therapy for cranial decompression. The combined decompression of the abdominal and cranial compartment has been described.[7]

In conclusion, decompressive laparotomy may be effective in the treatment of refractory intracranial hypertension especially in the younger patient population with polytrauma where intracranial hypertension is not attributed to mass effect and is associated with signs of ACS. Serial bladder pressure measurement and a lower threshold for ACS diagnosis should be employed in every patient with difficult ICP control to detect development of ACS, and help optimize the timing of escalation of therapy.

Received 12th May 2013. Accepted 31st July 2013.

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