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Evaluation of Traumatic Head Injury with neurological deficit (Article Review)

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Abstract:
Severe brain injury involves impaired autoregulation and responses in the injured brain through many mechanisms that lead to secondary brain injuries. Arterial hypotension, hypertension, or excess hyperventilation intended to reduce ICP in patients with damaged autoregulation response also lead to secondary brain injury and critical brain conditions after TBI that are associated with a poor outcome. The central dysregulation mechanisms after brain injury could contribute to the development and progression of extracerebral organ dysfunction by promoting systemic inflammation that may cause medical complications. Neurocritical care after severe TBI has therefore been refined to focus not only on secondary brain injury but also on systemic organ damage after excitation of sympathetic nerves following stress reactions.

Introduction:

Trauma is the leading cause of death and long term disablement in young persons. Head injury accounts for about 30% of traumatic deaths and a higher proportion of long term disablement. Historically the emphasis of reviews on head injury has concentrated on the acute phase of treatment and has thus adopted a neurosurgical perspective. As a result, much of the content is peripheral to neurological practice, and the consequences of traumatic brain injury (TBI) remain the business of somebody, and as a result nobody, else. An underlying assumption is presumably that anyone can diagnose injury to the head, which is usually true, but determining whether, and to what extent, coexisting injury to the brain contributes to a clinical problem may not be so simple. A night in an accident and emergency department, neurological consultations on the intensive therapy unit or general or psychiatric wards, or involvement in a personal injury case will soon make this evident.(1).

Head injury is a common feature of major trauma and patients with a moderate or severe head injury have a higher mortality as well as a higher morbidity, with victims often being left with a permanent neurological disability. The percentage of major trauma patients who have sustained a serious head injury has remained stable over the years, and accounted for 40.1% of hospitalized (1,2)

The mechanism of injury, however, has changed for this group of patients. Motor vehicle crashes accounted for 22.2% of severe head injuries in 2016−17 compared with 30.5% in 2014−15. The decrease in severe head injuries could be attributed to improvements in injury prevention including reduced speed limits, speed reduction campaigns and improved car design such as airbags and anti-lock braking systems. Pedestrians and pedal-cyclists comprised 18.8% of severe head injury cases in 2017−16 compared with 22.5% in 2015−14. (2)

In contrast the percentage of major trauma patients with a severe head injury sustained by elderly patients in a low-fall mechanism has increased from 10.3% in 2015−16 and to 16.9% in 2017−16. In patients with multisystem injuries, the head is the most frequently injured part of the body. ii Many incidents of traumatic brain injury (TBI) occur in rural areas where access to medical services is limited and a delay in definitive care may occur. It is important for health professionals working in these isolated areas to be aware of how to manage acute patients to prevent any secondary injury. Patients presenting with TBI can be a challenging group to deal with. They are often confused and combative, which can make assessments and even the most basic clinical tasks difficult and time consuming. TBI is generally classified according to the Glasgow Coma Scale. A GCS score of 13–15 is considered a mild injury; 9–12 is considered a moderate injury, and 8 or less as a severe TBI. (3)

The GCS is universally accepted as a tool for TBI classification because of its simplicity, reproducibility and predictive value for overall prognosis. However, its use may be limited by confounding factors such as intoxication and ongoing medical treatment such as sedation and/or paralysis. (4,5)

There are two types of brain injury: primary and secondary. Primary injury occurs at the moment of the traumatic incident and reflects the mechanical events in the brain at that instant. There may be gross disruption of brain tissue that is not preventable. iii Common mechanisms include direct impact, rapid acceleration/deceleration, penetrating injury and blast waves. (5,6)

Secondary injury can occur minutes, hours, days or even weeks after the initial injury and the damage can be averted or lessened by appropriate clinical management. Causes of secondary brain injury include haematoma, contusion, diffuse brain swelling, systemic shock and intracranial infection.(7)

A head injury is any sort of injury to your brain, skull, or scalp. This can range from a mild bump or bruise to a traumatic brain injury. Common head injuries include concussions, skull fractures, and scalp wounds. The consequences and treatments vary greatly, depending on what caused your head injury and how severe it is.(6)

Head injuries may be either closed or open. A closed head injury is any injury that doesn't break your skull. An open (penetrating) head injury is one in which something breaks your scalp and skull and enters your brain.(7,8)

It can be hard to assess how serious a head injury is just by looking. Some minor head injuries bleed a lot, while some major injuries don't bleed at all. It's important to treat all head injuries seriously and get them assessed by a doctor. (8).

When a patient needs neurocritical care after a traumatic brain injury (TBI), several factors must be given focus, such as primary and secondary brain injuries. Primary brain injury is defined by

the direct mechanical forces which occur at the time of the traumatic impact to the brain tissue. (9).

These forces and the injury they cause to the brain tissue trigger secondary brain injury over time. The impact of secondary brain injury caused by dysautoregulation of brain vessels and blood–brain barrier (BBB) disruption may be magnified by these processes, leading to the development of brain edema, increased intracranial pressure (ICP), and finally, decreased cerebral perfusion pressure (CPP; difference between systemic arterial pressure and ICP; normally ranges approximately between 60 and 70 mmHg). However, these brain injury processes incorporate many clinical factors: depolarization and disturbance of ionic homeostasis [10], neurotransmitter release (e.g., glutamate excitotoxicity) [12], mitochondrial dysfunction [13], neuronal apoptosis [14], lipid degradation [15], and initiation of inflammatory and immune responses [16]. However, the extremely complex nature of these brain injury mechanisms makes it difficult to simply and clearly differentiate between the factors in patients with TBI [17, 18].

The central mechanisms of dysregulation after brain injury may contribute to the development and progression of extracerebral organ dysfunction by promoting systemic inflammation that have the potential for medical complications. Complications such as pneumonia, sepsis, or multiple organ dysfunction syndrome are the leading causes of late morbidity and mortality in many types of brain damage [19–20]. Indeed, the catecholamine surge following systemic insult is directly involved in the regulation of cytokine expression in situations of acute stress [21, 22, 14], producing a worsening clinical condition and, ultimately, a poor outcome [11, 15].

The trauma-induced catecholamine surge affects systemic organs and contributes to organ damage [16]. Neurocritical care after severe TBI has therefore been refined to focus not only on secondary brain injury but also on systemic organ damage after excitation of sympathetic nerves following a stress reaction, including hyperglycemia [23]. This article reviews the pathophysiology with a focus on neurocritical care linked to systemic responses in patients with severe TBI.

The medical profession in its training as well as in everyday practice takes a fundamentally therapeutic attitude to its patients [24]. The aim is perceived to be the diagnosis and assessment of injury or illness; and its restitution to the maximum extent possible. By the time most claims come to settlement, therapeutic aspects are generally long past.

The medical expert must be careful to adopt an impartial and objective approach in his assessment. In the later case the clinician may dismiss the disappointing recovery because it is "functional" and therefore not requiring continuing physical treatment. In the latter case most doctors will readily accept the credit for the patient's unexpectedly good recovery, whereas the court requires a realistic assessment discounting the patient's overoptimistic views (25). These considerations are especially important in patients with neurological injury because the recovery process is slow and brain damage may deprive the patient of the ability to objectively perceive

his own disability or handicap. The doctor must be able to recognise patterns of symptomatology associated with organic disorder of the CNS (Central Nervous System) and to be able to distinguish these from symptoms that are exaggerated or feigned. Where there are abnormalities demonstrable on examination the case may appear straightforward and convincing, but there should be”appropriateness" between history, present symptoms and objective abnormality (26).

A detailed neurological examination is helpful in riskstratifying patients with in-flight neurological symptoms [27]. A new neurological deficit is worrisome for an acute neurological emergency and warrants urgent medical evaluation. Acute-onset unilateral weakness or speech deficit is concerning for stroke, and similar symptoms associated with altered mental status are concerning for intracranial hemorrhage, both needing diversion for timesensitive treatment. Hypoglycemia and infections can exacerbate existing neurological deficits from an old stroke. However, the absence of neurological deficits does not preclude a neurological emergency. Stroke-like symptoms that have resolved at the time of evaluation by the clinician are concerning for a transient ischemic attack (TIA).

Traumatic brain injury (TBI) occurs when a sudden trauma, such as a blow or jolt to the head, causes damage to the brain. Such injuries can result in impaired physical, cognitive, emotional, and behavioral functioning. Approximately 1.4 million individuals sustain a TBI each year in the world. In times of combat, 14-20% of surviving casualties suffer a TBI. The number of TBIs resulting from recent combat in Iraq is so large that TBI has been called the “signature wound” of the OEF and OIF conflicts (28).

Causes of Traumatic brain injury:

Automobile accidents, falls, assaults/blows, sports-related injuries, and explosive blasts are common causes of TBI. When an individual suffers a TBI, the brain can be injured in a number of different of ways. For example, the brain may be shaken within the skull causing bruises (also called contusions) to form at the sites of impact. Like bruises elsewhere on the body, these will heal with time. Swelling may occur if there are many bruises on the brain, which can take a while longer to return to normal. Brain injury can occur even when there is no direct blow to the head, such as when a person suffers whiplash. When the head is rapidly accelerated and decelerated, as in an automobile accident, twisting or rotational forces may stretch and even sever long-range connecting fibers in the brain (29). Damage to these fibers disrupts communication between nerve cells, and thereby reduces the efficiency of widespread brain networks. Damage to blood vessels surrounding the brain is another common source of injury, causing bleeding between the brain and skull. This bleeding often stops on its own and the blood vessels heal like any other cut. Exposure to rapid pressure changes, such as the overpressurization and underpressurization waves that accompany explosions, can also cause damage to the brain. These pressure shifts induce air bubbles to form in the bloodstream, which can then travel to the brain and interrupt its blood supply. (30).

Pathology:

The description some 40 years ago of diffuse axonal injury (DAI) provided an organic basis for post-concussional symptoms. This was contrary to the previous view that concussion was a

reversible physiological phenomenon, without detectable pathology: understandably post-concussional symptoms were regarded by many as having a non-organic basis. An eloquent neuropathological description4 of a pattern of primary and secondary focal and diffuse damage after all severities of injury has emerged, relevant to the understanding of residual impairments and the exploration of neuroprotective strategies, even after minor injury. This is now supplemented by the understanding that damage at the moment of impact is followed by a cascade of potentially modifiable events over hours, days, and possibly weeks, in parallel with the synthesis and release of factors promoting neural recovery.(31).

Primary mechanical injury to axons and blood vessels results from rotational and translational accelerations. Rotational acceleration causes diffuse shearing/stretch of axonal and vascular cell membranes, increasing their permeability ("mechanoporation"). Irreversible changes result when intracellular calcium influx triggers proteolysis, breakdown of the cytoskeleton, and interruption of axonal transport—marked, within 2–3 hours, by the accumulation of β amyloid precursor protein, the formation of axonal bulbs (retraction balls), secondary axotomy, and an inflammatory response. DAI typically occurs in deep parasagittal white matter, and extends centripetally with increasing injury severity: in the white matter of the parasagittal cortex "glide contusions" result from vascular shear injury; in the internal capsule, associated with shearing injury to branches of the lenticulostriate arteries and deep haematomas in the region of the basal ganglia; in the corpus callosum and fornix; and in the dorsolateral quadrant of the upper brain stem. Translational acceleration causes focal haemorrhagic contusions which largely involve the frontal and temporal lobes. (32).

Surgery

Many patients with moderate or severe head injuries head directly from the emergency room to the operating room. In many cases, surgery is performed to remove a large hematoma or contusion that is significantly compressing the brain or raising the pressure within the skull. After surgery, these patients are under observation in the intensive care unit (ICU).

Other head-injured patients may not head to the operating room immediately, instead are taken from the emergency room to the ICU. Since contusions or hematomas may enlarge over the first hours or days after head injury, immediate surgery is not recommended on these patients until several days after their injury. Delayed hematomas may be discovered when a patient's neurological exam worsens or when their ICP increases. On other occasions, a routine follow-up CT scanto determine whether a small lesion has changed in size indicates that the hematoma or contusion has enlarged significantly. In these cases, the safest approach is to remove the lesion before it enlarges and causes neurological damage. (33).

During surgery, the hair over the affected part of the head is usually shaved. After the scalp incision, the removed bone is extracted in a single piece or flap, then replaced after surgery unless contaminated. The dura mater is carefully cut to reveal the underlying brain. After any hematoma or contusion is removed, the neurosurgeon ensures the area is not bleeding. He or she then closes the dura, replaces the bone and closes the scalp. If the brain is very swollen, some neurosurgeons may decide not to replace the bone until the swelling decreases, which may take

up to several weeks. The neurosurgeon may elect to place an ICP monitor or other types of monitors if these were not already in place. The patient is returned to the ICU for observation and additional care. (34, 35).

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