Review

Head Injuries, a general approach

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ABSTRACT

When a person with a head injury arrives at the hospital, doctors and nurses first check life signs: cardiac work, blood pressure and breathing. For those who do not breathe satisfactorily, a extractor fan may be required. Doctors immediately assess the patient's state of mind and memory. They also test the brain's basic functions by checking the size of the pupils and their response to light, assessing reactions to sensations such as heat and stab of the needle, and testing the ability to move their arms or legs. Computed tomography or magnetic resonance imaging is required to evaluate possible brain injury. Traumatic brain injury occurs when an external mechanical force causes brain dysfunction. Brain trauma is usually the result of a violent hit to the head. An object that pierces the skull, such as a bullet, can also cause brain trauma. Mild trauma can be caused by temporary brain cell dysfunction. More serious brain trauma can lead to bruising, torn tissue, bleeding and other physical injuries to the brain, which can result in long-term complications or even death. Head injuries are the cause of disability and death of people under the age of 50, more than any other type of neurological impairment. Nearly half of people with severe head injuries die. The brain can be damaged even when the skull is not pierced.

1. INTRODUCTION

The medical profession in its training as well as in everyday practice takes a fundamentally therapeutic attitude to its patients [1]. 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 over-optimistic views. 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.

2. NEUROLOGICAL EXAMINATION

A detailed neurological examination is helpful in risk-stratifying patients with in-flight neurological symptoms [2]. 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 time-sensitive 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).

3. DAMAGE

A head injury may be referred to according to the direct impact of the trauma as a 'closed' or 'penetrating' injury [3]. A penetrating injury occurs as a result of an object penetrating the scalp, fracturing the skull and piercing the brain.This is less common than a closed head injury, in which an external force creates a violent movement within the brain that damages the structures within it.

An RTA (Road Traffic Accidents), for example, results in rapid acceleration, deceleration and sometimes also rotation of the brain within the skull. As a result of these sudden movements, there is a widespread disruption of the neuronal pathways, and sometimes blood vessels acting like cheese wires cut through the softer neuronal structures. In an older person the blood vessels may be more friable and frequently bleed. Damage is also incurred to the frontal and temporal lobes of the brain as the soft brain tissue is moved over the surface of the bony prominences inside the skull. The frontal lobes are responsible for higher intellectual functions such as planning and organisation, as well as the control of behaviour and emotions. It is also the seat of personality, with a small area called the 'anterior

cingulated cortex' believed to contain the 'I' we all feel we have inside us. The temporal lobes are concerned with memory (both longterm memories and procedural memories are stored there) and language. Depending upon the force of the impact, reciprocal damage to the occipital lobe (concerned with vision) may occur as the brain rebounds from the front of the skull to the back.

4. CONCUSSION

One of the key elements to establish is the concussion history of the patient [4]. The use of helmets, while protective to a certain extent, does little to prevent rotational injuries.

Concussion is typically managed in the outpatient setting. While the most important aspects of the ED (Emergency Department) encounter include ruling out life- and limb-threatening injuries, establishing a diagnosis of concussion can have profound impact on the long-term outcomes of the individual.

No treatment has been proven to speed recovery from a concussion. Some advocate for strict rest (no physical activities, no reading, no television/computers/phones) for 5-7 days, but compliance will be an issue. Others have suggested an early active rehabilitation approach including strict rest in the first day or two, followed by the gradual introduction of mental activities and subsequently progressively intense physical activities.

5. TRAUMATIC BRAIN INJURY

Traumatic brain injury is a major cause of death and disability [5]. The incidence is rising due to increasing motor vehicle accidents in low- and middle-income countries and falls of members of the aging population in high-income countries. Violence is reported to cause closed-head injury in about 7–10% of cases. Penetrating injuries are more common with the more frequent use of firearms, and a greater amount of blast injuries became the result of improved explosive devices used in terrorist and other attacks. More than 1.4 million patients with head injuries are treated annually in US emergency departments, and 21% of these patients are hospitalized. Almost 10% of all deaths in the United States are caused by injury, and about half of traumatic deaths involve the brain. In the United States, a head injury occurs every 7 seconds and a death due to traumatic brain injury every 5 minutes. The annual financial burden accounts to US $60 billion.

Brain injuries occur at all ages, but the peak is in young adults between the ages of 15 and 25 years. Head injury is the leading cause of death among people younger than 25 years. Men are affected three to four times as often as women.

Traumatic brain injury can be classified according to the mechanism of injury, clinical severity, structural damage on imaging, and prognosis.

In CHI (closed head injury), the brain may be injured in a number of ways but, strictly speaking, the skull usually has not been fractured by any external object [6]. A common cause of CHI is a high-speed motor vehicle accident in which the brain, moving at a high speed, comes to a sudden stop. The fluid-filled meninges provide some protection, but depending on the speed and what the head comes into contact with, the brain may be forced against the inner skull and damaged. The basal portion of the skull has many bony protuberances, and brain regions coming into contact with these are particularly vulnerable. The point of impact is known as the coup injury, and as the brain reacts to the impact, it may also recoil and hit the opposite side of the inner skull, resulting in a contrecoup injury. These injuries may be in the form of bruising of the cortex or may result in more significant focal brain damage at deeper levels, where an intracerebral hemorrhage may occur, leading to a space-occupying hematoma. In addition, again because of the physics of the moving brain, a diffuse axonal injury (DAI) may result as the cerebrum twists upon the brainstem, stretching, and tearing a large number of neuronal axons diffusely throughout the brain.

The physician must methodically assess a patient's stability, and determine if interventions are required prior to a full and complete assessment [7]. One must pay close attention to the ABCs (Airway, Breathing, and Circulation) in any trauma patient. Attention should be paid to maintaining an appropriate and stable airway, to assure adequate breathing, and to assess circulation and obtain IV access. Upon completion of the ABCs, attention should be turned to the neurological status of the patient, while periodically reassessing the airway, breathing, and circulation. This is vital because, as mentioned earlier, even brief episodes of hypotension or hypoxia can lead to worse outcomes by causing preventable secondary brain injury.

Patients with significant head injury will often need their airway actively managed to prevent aspiration from vomiting, and to oxygenate and ventilate the comatose patient. Have a low threshold to intubate the head-injured patient. After completion of the primary survey (ABCs), the priority shifts to looking for other injuries including neurological ones. One typically uses the Glasgow Coma Score. The Glasgow Coma Scale (GCS) is a commonly used 15-point scale to assess a patient's neurological status after head injury. A score of 14 or higher is considered a minor head injury; 9–13 is a moderate head injury, and 8 or below is a severe head injury.

6. TUMOR

The signs of either a primary or metastatic brain tumor are the result of the tumor's location, mass effect, and rate of growth and of metabolic disturbances [8]. The Monro-Kellie doctrine describes the relationship between intracranial volume (composed of brain, CSF (cerebrospinal fluid), and blood) and ICP (intraparenchymal cerebral pressure). The brain can accommodate enlarging mass lesions until a critical volume is reached. The actual volume tolerated is increased if growth is gradual. At this point, the ICP increases dramatically. Normally, the endothelial tight junctions of cerebral vessels (blood-brain barrier) prevent the leakage of large solutes and water into the brain. Vessels in cerebral tumors tend to have less constant tight junctions and may lack certain enzymes that degrade vasoactive substances in the brain such as leukotrienes. Reactive edema fluid thus accumulates in the extracellular space adjacent to the tumor. Edema can cause neurologic deterioration by increasing ICP and causing a midline shift of brain structures. Hyponatremia can occur in patients with CNS (central nervous system) neoplasm and may cause cytotoxic edema and seizures. This can occur secondary to cerebral salt wasting. Conversely, hypernatremia can result from hypothalamic dysfunction and lack of antidiuretic hormone response and is referred to as diabetes insipidus. These patients lose excessive amounts of free water. Any serum sodium abnormality can result in altered mental status and eventually coma and neuronal cell death.

7. IMAGING

Major injuries to rule out include cervical spine fractures or ligamentous injuries, intracranial bleeding and blunt cerebrovascular injuries [3]. Imaging studies are not necessary in all cases, but should be considered if the clinical suspicion is high. The NICE guidelines on head injury state that a CT (computer tomography) scan is indicated if any of the following are present:

- GCS < 13 on initial assessment
- GCS < 15 at 2 hours after injury on assessment in the Emergency Department
- Suspected open or depressed skull fracture
- Any sign of basal skull fracture
- Post-traumatic seizure
- Focal neurological deficit
- More than one episode of vomiting since the head injury
- Dangerous mechanism of injury
- Anti-coagulant use (e.g. warfarin, NOACs)
- > 30 minutes of retrograde amnesia

8. MINOR INJURIES

A concussion is defined as nonpenetrating head trauma resulting in a brief loss of consciousness [9]. There is no definition for the length of a "brief" period, but some experts allow up to 6 hours before consciousness returns. Most important, patients with a concussion will regain normal consciousness and have a normal neurological examination findings and a normal CT scan of the brain. In as many as 30% of patients who experience a concussion, postconcussive syndrome (PCS) will develop .Patients with PCS may have headache, nausea, emesis, memory loss, dizziness, diplopia, blurred vision, emotional lability, or sleep disturbances after a minor head injury. Fixed neurological deficits are not part of PCS, and any patient with a fixed deficit requires careful evaluation. PCS usually lasts 2–4 months. Typically, the symptoms peak 4–6 weeks after the injury.

It is important to always look for a contrecoup injury to the brain with any head trauma. Contrecoup injuries occur when a force strikes one side of the head hard enough to shift the brain in the opposite direction of the injury, and inertia then forces the brain to strike the opposite side of the intracranial cavity, thus causing an injury to the opposite side of the brain from the original trauma.

Both direct trauma and contrecoup injuries can cause intracerebral and extracerebral bleeding, or both. Intracerebral bleeding, such as cerebral contusion, is caused by the disruption of microvasculature typically within the parenchyma. That disruption can cause the arteries beneath the arachnoid and above the pia to bleed, called a subarachnoid hemorrhage. Nearly half of all patients with minor head injury will have a cerebral contusion visible on CT scan.

9. DIAGNOSIS

In contrast to the generalized symptoms of mass effect, edema, and sodium imbalance, which include headache, nausea and vomiting, and mental status changes, the precise location of a tumor can cause specific neurologic deficits [8]. These deficits include aphasia, memory or personality disturbances, hemiparesis, and visual or sensory impairment. In many patients, no neurologic deficit is present on initial presentation, and a seizure is the first indication of a CNS neoplasm. In the awake patient, the history should be taken carefully to determine the exact initial symptoms and the rate at which the problems have advanced. This information can indicate the approximate location in the nervous system and serves as a clue to the rate of tumor growth. Neurologic examination in the ICU should include ophthalmoscopy for papilledema, detailed mental status and language assessment, cranial nerve tests, motor, sensory, and reflex tests, and testing of cerebellar function.

Several disease processes should be considered when evaluating a patient who presents with confusion, headache, dysphasia, motor or sensory deficits, seizures, hyponatremia, or any combination of these findings. Stroke usually presents with a sudden onset of fixed neurologic signs and differs from the progressive course of a CNS neoplasm. The gradual progression of a neurodegenerative disease can result in symptoms similar to those caused by a mass lesion, but preliminary CT scanning or MRI can rule out this lesion. Infections such as meningitis, encephalitis, and especially cerebral abscess can result in global or focal neurologic dysfunction and seizures but frequently are accompanied by fever, leukocytosis, and (in the case of cerebral abscess) characteristic findings on CT scan or MRI (magnetic resonance imaging). An important distinction to be made is whether the mass represents a primary or metastatic lesion. In the latter, a general physical examination with radiologic studies and metastatic evaluation is required.

Spinal shock is often confused with the neurogenic shock of sympathetic interruption [10]. They are different entities. Complete separation of the spinal cord from the brain abolishes voluntary movement and sensory perception and causes changes in cord physiology and reflex activity. Acute cord confusion is a simple explanation of the resulting pathophysiology. Spinal shock is manifested by the transient cessation of cord activity in the normal cord below the injury. The cord distal to the injury is unable to function as one would expect from a newly created upper motor neuron lesion. Spinal shock may last for a few hours to several weeks, depending on the segmental level and extent of the cord injury. During this period, both somatic and autonomic reflexes below the injured segments disappear. Spinal shock has been attributed to the sudden loss of descending facilitatory impulses from higher centres. Recovery from spinal shock is heralded by the return of the Babinski response, followed by the perineal reflexes. In quadriplegia and high paraplegia, as the cord recovers from spinal shock, either recovery of function (depending on the degree of injury resolution at the injury site) occurs or, more commonly, spasticity develops. If the cord injury is at the conus medullaris or the cauda equina, unless recovery occurs, a lower motor neuron pattern with areflexia remains.

Patients without shock but with persisting abnormalities of mental status unresponsive to the administration of dextrose, naloxone, and thiamine must be assumed to have significant head injuries [11]. Importantly, however, in patients with a serious head injury and established or evolving shock, the customary means of reducing intracranial pressure (restriction of fluids, the administration of furosemide, mannitol) must be abandoned and the more immediately life-threatening deficit in intravascular volume corrected aggressively. In

any patient presenting with trauma, but particularly in the patient with an abnormal mental status, the possibility of occult chest or abdominal injuries (or both) must be carefully investigated and excluded. When injuries to these areas are noted and pose an immediate threat to life, the neck must remain immobilized while appropriate intervention is pursued.

10. CONCLUSION

The final consequences of a head injury range from complete recovery to death. The type and severity of the disability depends on where and how badly the brain was damaged. Many brain functions can perform multiple areas of the brain, and unharmed areas sometimes take on functions that were lost when the other area was damaged, allowing partial recovery. However, as people get older, the brain becomes less able to shift functions from one area to another. For example, speaking skills in young children control several areas of the brain, but in adults they are collected on one side of the brain. If the areas of speech in the left hemisphere are severely damaged before age 8, the right hemisphere can assume almost normal speech function. However, injuries to the speech area in adulthood are likely to remain permanently flawed. Some functions such as vision and movement of the arm or leg monitoring unique areas on one side of the brain. Damage to any of these areas usually causes permanent defects. However, rehabilitation can help people minimize the impact of these defects on function. People with severe head injuries sometimes get amnesia and cannot remember the event just before and after they have lost consciousness. Those who regain consciousness in the first week are most likely to recover their memory.

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