Review Article

An Overview of Childhood Head Injuries

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Summary

Childhood Head Injury (CHI) was reviewed to highlight those aspects of epidemiology, pathogenesis and management which often generate controversies in surgical circles. Some aspects of C.H.I. which are also recent in scope are also emphasized to ensure a successful outcome.

Keyword: Childhood, head injury, pathogenesis, management, outcome.

Introduction

Approximately 500,000 children in the UK present to hospitals each year with head injuries[1]. At the Jos University Teaching Hospital, about 102 new cases of childhood head injuries (CHI) are recorded each year out of which a total of 7% will be severe leading to death or other life-threatening complications[2].

Childhood head injuries are therefore a source of significant mortality, major disability and psychological burden for society the world over. This review, therefore, aims to highlight those aspects of C.H.I. (epidemiology, pathogenesis, clinical presentation and management) that are important to ensure a successful outcome especially in tropical Africa.

Epidemiology, Pathophysiology And Clinical Presentation

In CHI, an external force results in injury to the scalp, cranium, brain, parenchyma and intracranial vessels usually in combination. Road traffic accidents involving occupants and pedestrians still remain the most common cause of CHI the world over[2,3,4]. Open scalp wounds can lead to ensanguating haemorrhage and are classified as open head injuries once the pericraium is breached. Extravasation of tissue fluids and blood into various layers of the scalp and underneath the pericranium is common in various types of birth injuries. The Shaken Impact Syndrome (SIS) is usually due to parentally inflicted CHI in children less than 2 years resulting in rupture of the emissary veins of the scalp with subgaleal haematoma. Raccoon eyes and sub-conjuctival haemorrhage[5]. SIS is beginning to attract attention in urban areas of the Tropics where it was virtually non-existent.

Falls which were the second most common cause of CHI in our series are the commonest cause of depressed skull fractures[2]. In another report from U.K., 52% of those with CHI suffered from depressed skull fractures secondary to concentrated forces indenting the relatively malleable skull of children[6]. Depressed skull fractures are frequently associated with underlying primary focal coup lesions resulting in seizures and secondary infection[7]. Basal skull fractures in children frequently pass unnoticed resulting in meningitis, which is one cause of prolonged coma in children. Most skull fractures in growing children will heal rapidly without significant contour irregularity. Skull fractures in infants associated with dural tears, however, may progressively enlarge as a result of bone erosion by lepto-meningeal or even brain herniation, especially at the parietal area of the skull. These post-injury pulsatile masses are known as "growing skull fractures" and have a prevalence

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of 1.2% in Europe[8]. They do also occur in tropical Africa[9].

Diffuse axonal injury in the white matter of the brain (DAI) is now thought to be the most important mechanism of primary brain damage resulting in different degrees of alteration in the level of consciousness at presentation in CHI[7]. It was S. J. Strich who first noticed cerebral atrophy and hydrocephalus in head injured patients who died after a prolonged vegetative state[10]. Lately, cerebella atrophy has been reported after severe traumatic head injury in children[11]. The relationship between late neuro-pathological MRI findings and cognitive sequelae is now subject of further research. DAI are often associated with interventricular bleeding of birth injuries in which the infant present with a high pitched cry and the clenched fist sign. They heal by microglial formation which is a cause of late seizure in CHI. Focal cerebral lacerations are the prototype of early seizures in CHI. Both DAI and primary focal brain damage usually co-exist in CHI due to the relatively large head size of children.

What is then the flow chart indicating the mechanisms of primary brain damage and secondary brain damage better known as the complications of CHI? Impaired auto-regulation of the brain occurs early in secondary brain damage. Auto-regulation of cerebral blood vessels normally maintains a constant cerebral blood flow (CBF) over a wide range of systemic blood pressure (MAP). Failure of this system implies that CBF becomes passively dependent on MAP[12] resulting in accumulation of toxic metabolites like carbon-dioxide and products of anoxia. Delayed brain swelling ensues leading to increased intracranial pressure (ICP), brain shift and global cerebral ischaemia[13].

Summarizing therefore, CHI normally runs a prolonged and bizarre course in which the magnitude of the injuring forces do not parallel the clinical manifestations[14]. Some of the most common and interesting complications not mentioned include sub-dural haematoma and abscesses, excessive growth of micoglial tissue in open head injury in which the dural has not been closed and has been infected[2]. This can also occur iatrogenically following craniectomies.

Assessment, Investigations and Acute Care

The initial management of CHI aims at resuscitating the patient when necessary, assessing the severity of the primary brain injury, prevent or reversing any secondary brain injury and stabilizing the oxygenation and haemodynamics of the patient[15]. There must be an integrated multi-disciplinary approach that is based on the Advanced Trauma Life Support (ATLS)[16]. Clearing and maintaining the airway must be accompanied by cervical spine control since 1.7-6% of patients with head injury will have a cervical spine fracture[17,18]. Hyperflexion/extension, axial compression injuries are often associated with transient neural deficits. No abnormalities are detected on plain spinal x-rays due to the cartilaginous nature of the growing childhood spine. Spinal cord injury without obvious radiographic abnormality (SCIWORA) calls for a high index of diagnostic suspicion to prevent its attendant morbidity and mortality[19,20]. Head injury results in greatly raised circulating levels of catecholamine leading to cardiac arrhythmias and neurogenic pulmonary oedema heralded by the rapid development of hypoxaemia[21].

Normal (0.9%) saline or 5% dextrose in 0.9% saline appear to be the ideal fluids for pre-hospital resuscitation of patients with CHI since administering hypo-osmolar I.V. fluids like 0.45% saline and 5% dextrose in water has been conclusively shown to worsen cerebral oedema in closed head trauma in rats 2 hours after the injury[22]. Bilateral fixed dilated pupils in severe CHI may be due to inadequate cerebral perfusion, secondary to systemic hypotension, primary and secondary brain stem lesions with transtentorial herniation.

Adequate blood transfusion must be considered to make good the mean systemic blood pressure before further assessment. Neurological disability

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is best assessed by the Glasgow Coma Score (GCS) employing the modification of ATLS as it concerns the best verbal score for children less than 4 years of age[16]. The child is cored 1, if the response is none, 2 if the child is restless and agitated, 3 if persistently irritable, 4 if he cries but is consolable and 5 if he utters appropriate words or social smile, fixes and follows. Among the many advantages of the GCS is that it enables the medical officer to dispose his patients at the district hospital level[23]. Children with mild head injury with a G.C.S. of 14-15 could be observed for 48 hours in hospital or sent home with oral and written instructions on when to return as an emergency. Those with moderate head injury, GCS of 9-13 or severe head injury, GCS of 9-13 need to be referred to centres with specialist neuro-surgical services for investigation and intervention. Any patient still in coma following adequate resuscitation requires immediate non-contrast enhanced CT-Scan with a safe airway, preferably intubated[12].

Knowledge of the state of the intracranial contents then allows specific treatment of any intracranial injury to be commenced. Findings based on Traumatic Coma Data Bank (T.C.D.B.), Computed Tomographic Scan classification[24] include: I, normal scan; II, DAI; III, DAI plus swelling; IV, DAI plus shift; V, mass lesion surgically evacuated; VI, mass lesion not operated. Magnetic Resonance Imaging (MRI) studies of brain show the true extent of brain involvement, CT-Scan do not show brain stem lesions[25].

Based on MRI findings, Grade I lesions involved the hemispheres only; Grade II lesions were unilateral lesions of the brain stem at any level with or without supratentorial lesions; Grade III lesions were bilateral lesions of the mesencephalon with or without supratentorial lesions; Grade IV were bilateral lesions of the pons with or without any of the foregoing lesions of lesser grades. Brain swelling is treated with brain shrinking solutions like mannitol or frusemide, intermittent positive pressure ventilation and surgical decompression. Bifrontal decompressive craniectomy significantly reduces post-traumatic intracranial hypertension and is associated with a favourable outcome in 70% of patients[26]. The therapeutic effectiveness of hypothermia is limited in patients who present with papillary abnormality on admission and brain swelling during surgery[22].

Chronic Care, Prognosis and Outcome

Although there are a variety of neurologic sequelae of CHI, the most common of these include seizures, headache and syncope. These could be secondary to penetrating injury, intracranial haematoma[12,28] or cerebral laceration. Febrile seizures could be due to post-traumatic meningitis. Afebrile seizures frequently complicate CHI and a follow-up EEG is often indicated and imaging studies can appropriate on a non-urgent basis[29]. Headaches are best approached by assessing the temporal course and associated clinical features. Most patients are diagnosed with either tension, migraine or chronic progressive headache (CPH)[30].

In CPH, an intracranial process must be addressed and pursued with appropriate imaging. Syncope can generally be categorized as autonomic, cardiac or non-cardiac, investigated and appropriately managed.

Post-traumatic stress disorder (PTSD) at 1 year in post CHI developed in 13% of children with severe CHI accompanied by traumatic amnesia and was associated with female gender and early post-CHI anxiety symptoms[31].

Prompt recognition of growing skull fractures and repair by regional craniectomy, pericranial dural inlay graft and immediate contour re-construction with rigidly fixed cranial bone graft ensure reliable aesthetic and functional restoration of the growing neurocranion[9,32]. There is a linear relationship between age and mortality in severe head injuries; children as a group have a much better outcome, lower mortality and a decreased incidence of traumatic intracranial haematoma compared with an adult population[33]. The infant

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brain however is more vulnerable to lasting deficits and is more prone to post-traumatic seizure development. Post-traumatic epilepsy itself causes deterioration in outcome because of the developmental disturbance at epilepsy onset and often unfavourable course. Physically abused children (SIS) with their often multiple and repeated head injuries are predestined for a poor outcome[34]. The development of pediatric trauma centers (PTC) in U.S.A. and Europe has no doubt contributed to the improved outcome in severe CHI[35].

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