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Review Article

A prospective Study of Cranial Injuries: A Review of Management Traumatic brain Injury

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Abstract

Head injuries (HI) are one of the most common causes of death, morbidity and disabilities in young adults. Epidemiological studies allow a quantitative estimation in terms of incidence and a qualitative estimate for the identification of risk factors in specific populations. These estimates may enable appropriate prevention programs. Estimates of annual incidence rates depend on territories, periods and methodological tools. Annual rates for hospitalized patients are found between 150 and 300/100,000 inhabitants. Severity of HI can be assessed by the Glasgow Coma Scale (GCS), the Abbreviated Injury Scale (AIS) or the Post-Traumatic Amnesia duration. Annual incidences of severe HI will depend on the selected score: around 25/100,000 inhabitants for cerebral trauma with intracranial injuries, around 9/100,000 for the most severe HI, with an AIS maximum of 5 with coma. The male:female ratio increases with degree of severity. Traffic accidents were the most frequent cause of HI. Many patients have associated injuries, worsening the outcome. Some risk factors are considered. Preventive measures are mainly conducted for traffic accidents, and include speed limit and regulations on helmet or seat belt use. Results of these measures are analysed

Postoperative dysfunction affected one cranial nerve in 25 patients, two nerves in four and three nerves in one patient. Most cranial nerve injuries were asymptomatic or mild in severity, resolved in one to 12 months and probably were caused by intraoperative retraction.

Keywords

Traumatic Brain Injury, Head Injury, Concussion, Bibliometric .

Introduction

Inflammation is an important part of the pathophysiology of traumatic brain injury. Although the central nervous system differs from the other organs because of the almost complete isolation from the blood stream mediated by the blood–brain barrier, the main steps characterizing the immune activation within the brain follow a scenario similar to that in other organs. The key players in these processes are the numerous immune mediators released within minutes of the primary injury. They guide a sequence of events including expression of adhesion molecules, cellular infiltration, and additional secretion of inflammatory molecules and growth factors, resulting in either regeneration or cell death. The question is this: to what extent is inflammation beneficial for the injured brain tissue, and how does it contribute to secondary brain damage and progressive neuronal loss? This review briefly reports recent evidence supporting the dual, the beneficial, or the deleterious role of neuroinflammation after traumatic brain injury.

Pathological and pathophysiological features

Primary and secondary injury

TBI is divided classically into two distinct phases: primary injury followed by delayed secondary injury. Primary injury arises from external physical forces applied to the head producing skull fractures, haematomas, and deformation and destruction of brain tissue, including contusions and axonal injury. Secondary injury develops over time with activation of multiple molecular and cellular pathways.Axonal stretching during injury can cause dysregulation of transmembrane ion fluxes and impaired axonal transport, and damaged axons could be vulnerable to secondary axotomy and demyelination.

Changes in ionic permeability and release of excitatory neurotransmitters, particularly glutamate, propagate damage through energy failure and overload of free radicals. Altered cellular permeability also increases calcium influx, which causes mitochondrial dysfunction, triggering further energy defects and necrotic and apoptotic processes.

Heterogeneity of TBI

TBI is typically classified according to clinical severity, with severe injury usually categorised on the basis of a total Glasgow Coma Scale (GCS) score of 8 or less.TBI produces various lesions that range from mild injury to devastating damage. Expanding haematomas—extradural or subdural—might need emergency surgical removal in the first hours after injury; intraparenchymal contusions can increase over hours or days and need surgery as well. More subtle lesions such as traumatic axonal injury (the term commonly used, diffuse axonal injury, strictly only applies when involving three or more locations.

Specific features of TBI in elderly people

TBI in older patients typically results from low-energy impacts such as ground-level falls,with a higher proportion of subdural haematomas and fewer contusions or epidural haematomas in this group than in younger patient.Cerebral atrophy and increased CSF space could buffer new pathological intracranial masses, which could be linked to a lower incidence of raised ICP.The GCS might underestimate the severity of brain injury in elderly patients, making a case for higher score thresholds to trigger triage of older patients to specialist centres.

Furthermore, age-related comorbidities (eg, diabetes, chronic cardiorespiratory disease, and renal dysfunction) reduce physiological reserve and increase the incidence and severity of brain damage due to second insults such as hypoxia and hypotension.

Method

The Medline was searched for TBI related articles from about 1980 to 2003 including terms such as –epidemiology‖, –head injury‖, –brain injury‖ and others. From the research reports identified, we checked references for additional relevant reports and from those reports we abstracted data on TBI incidence, severity, external cause, gender, mortality, prevalence, cost and related factors.

Annual rates for hospitalized patients are found between 150 and 300/100000 inhabitants. Severity of HI can be assessed by the Glasgow Coma Scale (GCS), the Abbreviated Injury Scale (AIS) or the Post-Traumatic Amnesia duration. Annual incidences of severe HI will depend on the selected score: around 25/100000 inhabitants for cerebral trauma with intracranial injuries, around 9/100000 for the most severe HI, with an AIS maximum of 5 with coma. The male: female ratio increases with degree of severity. Traffic accidents were the most frequent cause of HI. Many patients have associated injuries, worsening the outcome. Some risk factors are considered. Preventive measures are mainly conducted for traffic accidents, and include speed limit and regulations on helmet or seat belt use. Results of these mesures are analysed.

Targeted ICU management with aggressive therapies

No treatments in the ICU are risk free, and the more aggressive interventions for restoring cerebral homoeostasis have substantial potential to cause harm.

Aggressive therapies in elderly patients

Aggressive therapies are linked to severe side-effects and might not be tolerated by frail older patients with impaired physiological reserve (panel). The high incidence of cardiorespiratory comorbidities in such individuals might further reduce the ability of patients to tolerate some of the aggressive interventions (eg, augmentation of CPP, barbiturates, and hypothermia) used in the critical care of TBI. Therefore, careful monitoring of systemic physiology is mandatory, and caution is needed with haemodynamic augmentation and second-tier therapies for high ICP in these patients.

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