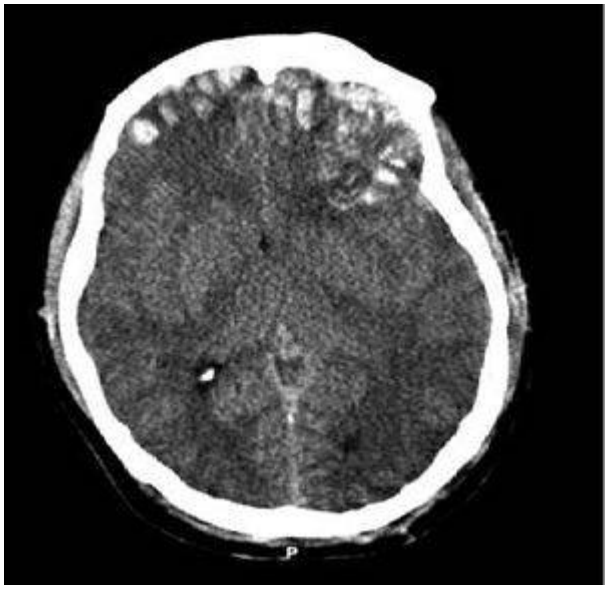
Severe traumatic brain injury outcome



Prediction models integrating general information, clinical manifestations, and auxiliary examination results may provide a reliable and rapid method to evaluate and predict the early prognosis of TBI patients

Age, Glasgow coma scale score, Apolipoprotein E genotype, damaged area, serum C-reactive protein, and interleukin 8 (IL-8) levels, and Marshall computed tomography classification were found associated with early Traumatic brain injury outcome. The accuracy of the early prognosis prediction model (EPPM) was 80%, and the sensitivity and specificity of the EPPM were 78.8% and 80.8% in the training set. The accuracy of the EPPM was 79%, and the sensitivity and specificity of the EPPM were 66.7% and 86.2% in the validation set. The accuracy of the early EPPM was 69.1%, and the sensitivity and specificity of the EPPM were 67.9% and 77.8% in the testing set. [1].

A deep learning model of head computed tomography and clinical information can be used to predict 6-month severe traumatic brain injury outcome [2].

Younger age, modified Fisher scale (mFS) score, and Intracerebral hemorrhage volume are associated with Intracranial pressure elevation in patients with a severe traumatic brain injury. Imaging features may stratify patients by their risk of subsequent ICP elevation [3].

There has been a secular trend towards reduced incidence of severe traumatic brain injury in the first world, driven by public health interventions such as seatbelt legislation, helmet use, and workplace health and safety regulations. This has paralleled improved outcomes following TBI delivered in a large part by the widespread establishment of specialised neurointensive care [4].

Impact

The impact of a moderate to severe brain injury depends on the following:

The severity of initial injury

Rate/completeness of physiological recovery

Functions affected

Meaning of dysfunction to the individual

Resources available to aid recovery

Areas of function not affected by TBI

Sex differences

Females exhibited more favorable cerebral physiology post-Traumatic Brain Injury, particularly better mitochondrial function, and reduced excitotoxicity, but this did not translate into better clinical outcomes compared to males. Future studies need to further explore potential sex differences in secondary injury mechanisms in TBI [5].

Smoking

A study found that pre-injury vascular risk factors, especially smoking, are associated with worse outcomes after TBI. Aggressive post-injury treatment of vascular risk factors may be a promising strategy to improve Traumatic Brain Injury outcomes [6].

Effect of trauma center designation in severe traumatic brain injury outcome

see Effect of trauma center designation in severe traumatic brain injury outcome

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Mortality or severe disability affects the majority of patients after severe traumatic brain injury (TBI). Adherence to the brain trauma foundation severe traumatic brain injury guidelines has overall improved outcomes; however, traditional as well as novel interventions towards intracranial hypertension and secondary brain injury have come under scrutiny after series of negative randomized controlled trials. In fact, it would not be unfair to say there has been no single major breakthrough in the management of severe TBI in the last two decades. One plausible hypothesis for the aforementioned failures is that by the time treatment is initiated for neuroprotection, or

physiologic optimization, irreversible brain injury has already set in. Lazaridis et al., and others, have developed predictive models based on machine learning from continuous time series of intracranial pressure and partial pressure of brain tissue oxygen. These models provide accurate predictions of physiologic crises events in a timely fashion, offering the opportunity for an earlier application of targeted interventions. In a article, Lazaridis et al., review the rationale for prediction, discuss available predictive models with examples, and offer suggestions for their future prospective testing in conjunction with preventive clinical algorithms [7].

Determining the prognostic significance of clinical factors for patients with severe head injury can lead to an improved understanding of the pathophysiology of head injury and to improvement in therapy. A technique known as the sequential Bayes method has been used previously for the purpose of prognosis. The application of this method assumes that prognostic factors are statistically independent. It is now known that they are not. Violation of the assumption of independence may produce errors in determining prognosis. As an alternative technique for predicting the outcome of patients with severe head injury, a logistic regression model is proposed. A preliminary evaluation of the method using data from 115 patients with head injury shows the feasibility of using early data to predict outcome accurately and of being able to rank input variables in order of their prognostc significance. [8].

A prospective and consecutive series of 225 patients with severe head injuries who were managed in a uniform way was analyzed to relate outcome to several clinical variables. Good recovery or moderate disability were achieved by 56% of the patients, 10% remained severely disabled or vegetative, and 34% died. Factors important in predicting a poor outcome included the presence of intracranial hematoma, increasing age, motor impairment, impaired or absent eye movements or pupillary light reflexes, early hypotension, hypoxemia or hypercarbia, and raised intracranial pressure over 20 mm Hg despite artificial ventilation. Most of these predictive factors were assessed on admission, but a subset of 158 patients was identified in whom coma was present on admission and was known to have persisted at least until the following day. Although the mortality in this subset (40%) was higher than in the total series, it was lower than in several comparable reported series of patients with severe head injury. Predictive correlations were equally strong in the entire series and in the subset of 158 patients with coma. A plea is made for inclusion in the definition of “severe head injury” of all patients who do not obey commands or utter recognizable words on admission to the hospital after early resuscitation [9].

Survival rate of isolated severe TBI patients who required an emergent neurosurgical intervention could be time dependent. These patients might benefit from expedited process (computed tomographic scan, neurosurgical consultation, etc.) to shorten the time to surgical intervention [10].

The impact of a moderate to severe brain injury can include:

Cognitive deficits including difficulties with:

Attention Concentration Distractibility Memory Speed of Processing Confusion Perseveration Impulsiveness Language Processing “Executive functions” Speech and Language

not understanding the spoken word (receptive aphasia) difficulty speaking and being understood

(expressive aphasia) slurred speech speaking very fast or very slow problems reading problems writing Sensory

difficulties with interpretation of touch, temperature, movement, limb position and fine discrimination Perceptual

the integration or patterning of sensory impressions into psychologically meaningful data Vision

partial or total loss of vision weakness of eye muscles and double vision (diplopia) blurred vision problems judging distance involuntary eye movements (nystagmus) intolerance of light (photophobia) Hearing

decrease or loss of hearing ringing in the ears (tinnitus) increased sensitivity to sounds Smell

loss or diminished sense of smell (anosmia) Taste

loss or diminished sense of taste Seizures

the convulsions associated with epilepsy that can be several types and can involve disruption in consciousness, sensory perception, or motor movements Physical Changes

Physical paralysis/spasticity Chronic pain Control of bowel and bladder Sleep disorders Loss of stamina Appetite changes Regulation of body temperature Menstrual difficulties Social-Emotional

Dependent behaviors Emotional ability Lack of motivation Irritability Aggression Depression Disinhibition Denial/lack of awareness

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Both single predictors from early clinical examination and multiple hospitalization variables/parameters can be used to determine the long-term prognosis of TBI. Predictive models like the IMPACT or CRASH prognosis calculator (based on large sample sizes) can predict mortality and unfavorable outcomes. Moreover, imaging techniques like MRI (Magnetic Resonance Imaging) can also predict consciousness recovery and mental recovery in severe TBI, while biomarkers associated with stress correlate with, and hence can be used to predict, severity and mortality. All predictors have limitations in clinical application. Further studies comparing different predictors and models are required to resolve limitations of current predictors [11)].

Prediction

Clinical outcome prediction following traumatic brain injury (TBI) is a widely investigated field of research. Several outcome prediction models have been developed for prognosis after TBI. There are two main prognostic models: International Mission for Prognosis and Clinical Trials in Traumatic Brain Injury (IMPACT) prognosis calculator and the Corticosteroid Randomization after Significant Head Injury (CRASH) prognosis calculator. The prognosis model has three or four levels:

(1) model A included age, motor GCS, and pupil reactivity

(2) model B included predictors from model A with CT characteristics

(3) model C included predictors from model B with laboratory parameters.

In consideration of the fact that interventions after admission, such as ICP management also have prognostic value for outcome predictions and may improve the models' performance, Yuan F et al developed another prediction model (model D) which includes ICP. With the development of molecular biology, a handful of brain injury biomarkers were reported that may improve the predictive power of prognostic models, including neuron-specific enolase (NSE), glial fibrillary acid protein (GFAP), S-100β protein, tumour necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), myelin basic protein (MBP), cleaved tau protein (C-tau), spectrin breakdown products (SBDPs), and ubiquitin C-terminal hydrolase-L1 (UCH-L1), and sex hormones. A total of 40 manuscripts reporting 11 biomarkers were identified in the literature. Many substances have been implicated as potential biomarkers for TBI; however, no single biomarker has shown the necessary sensitivity and specificity for predicting outcome. The limited number of publications in this field underscores the need for further investigation. Through fluid biomarker analysis, the advent of multi-analyte profiling technology has enabled substantial advances in the diagnosis and treatment of a variety of conditions. Application of this technology to create a bio-signature for TBI using multiple biomarkers in combination will hopefully facilitate much-needed advances. We believe that further investigations about brain injury biomarkers may improve the predictive power of the contemporary outcome calculators and prognostic models, and eventually improve the care of patients with TBI [12].

Injury site, injury type, and injury degree are the main risk factors for post-traumatic epilepsy. Traumatic brain injury outcome can be affected by early post-traumatic epilepsy. [13].

Insurance and racial disparities continue to exist for TBI patients. Insurance status appears to have an impact on short- and long-term outcomes to a greater degree than patient race [14].

CRASH

CRASH

IMPACT

IMPACT

Mortality

Traumatic brain injury mortality.

Quality of Life after Brain Injury

see Quality of Life after Brain Injury.

Traumatic brain injury complications

Traumatic brain injury complications.

Statins

Statins have been shown to improve traumatic brain injury outcome in animal models. The aim of a study was to determine the effect of preinjury statins on outcomes in TBI patients.

Lokhandwala et al. performed a 4-y (2014-2017) review of a TBI database and included all patients aged ≥18 y with severe isolated TBI. Patients were stratified into those who were on statins and those who were not and were matched (1:2 ratio) using propensity score matching. The primary outcome was in-Hospital mortality. The secondary outcomes were skilled nursing facility disposition, Glasgow Outcome Scale-extended score, and hospital and intensive care unit length of stay (LOS).

They identified 1359 patients, of which 270 were matched (statin: 90, no-statin: 180). Mean age was 55 ± 8y, median Glasgow Coma Scale was 10 (8-12), and median head-abbreviated injury scale was 3 (3-5). Matched groups were similar in age, mechanism of injury, Glasgow Coma Scale, Injury Severity Score, neurosurgical intervention, type and size of intracranial hemorrhage, and preinjury anticoagulant or antiplatelet use. The overall in-Hospital mortality rate was 18%. Patients who received statins had lower rates of in-Hospital mortality (11% versus 21%, P = 0.01), skilled nursing facility disposition (19% versus 28%; P = 0.04), and a higher median Glasgow Outcome Scale-extended (11 [9-13] versus 9 [8-10]; P = 0.04). No differences were found between the two groups in terms of hospital LOS (6 [4-9] versus 5 [3-8]; P = 0.34) and intensive care unit LOS (3 [3-6] versus 4 [3-5]; P = 0.09).

Preinjury statin use in isolated traumatic brain injury patients is associated with improved outcomes. This finding warrants further investigations to evaluate the potential beneficial role of statins as a therapeutic drug in a TBI [15].

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