Traumatic brain injury complications

- A Comparison of Quantitative Pupillometry and VOMS in Division 1 Female Soccer Players
- Distinct clinical phenotypes and their neuroanatomic correlates in chronic traumatic brain injury
- Identification of Serum Biomarkers for Blast-induced Traumatic Brain Injuries: Low vs. High-intensity Exposure in a Rat Model
- Clinical experience implanting a miniature externally powered vagus nerve stimulator
- Comparison of Complications in Early and Late Cranioplasty Following Decompressive Craniectomy Due to Traumatic Brain Injury: Systematic Review and Meta-Analysis
- Electro-Acupuncture to Treat Disorder of Consciousness (AcuDoc): Study Protocol for a Randomized Sham-Controlled Trial
- Association between traumatic brain injury and risk of developing infections in the central nervous system and periphery
- Sleep-Wake Disorders After Traumatic Brain Injury: Pathophysiology, Clinical Management, and Future

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1. ≈ 75% will exhibit an traumatic intracranial hematoma

a) may be present on initial evaluation and can then worsen

b) may develop in a delayed fashion

delayed epidural hematoma (EDH)

delayed subdural hematoma (SDH)

delayed traumatic contusions. see Traumatic intracerebral hemorrhage

Posttraumatic diffuse cerebral edema

Tension pneumocephalus

Hyponatremia

Hypoxia: etiologies include pneumothorax, MI, CHF…

Hepatic encephalopathy

Hypoglycemia: including insulin reaction

Adrenal insufficiency

Drug or alcohol withdrawal

Dural sinus thrombosis

Carotid artery dissection (or rarely, vertebral)

c) SAH: due to rupture of aneurysm (spontaneous or posttraumatic) or carotid cavernous fistula (CCF)

Cerebral embolism: including fat embolism syndrome

Hypotension (shock).

Alzheimer’s disease

Traumatic brain injury (TBI) is associated with increased dementia risk.

Alzheimer’s disease.

Brain abscess

Traumatic brain abscess.

Intracranial hypertension

see Intracranial hypertension after traumatic brain injury

Brain edema

Brain edema can result from a combination of several pathological mechanisms associated with primary and secondary injury patterns in traumatic brain injury (TBI).

As pressure within the skull increases, brain tissue displacement can lead to brain herniation, resulting in disability or death.

see Anticoagulation in traumatic brain injury.

Harris et al, suggest a link between head injury and Parkinson's disease and indicates further scrutiny of workplace incurred head injuries is warranted [1].

Cerebral contusion

Cortical cerebral contusions are one of the most common computed tomography (CT) findings in head injury [2] [3].

Cerebral Venous Sinus Thrombosis

Cerebral Venous Sinus Thrombosis.

Cerebrospinal fluid fistula

see Traumatic cerebrospinal fluid fistula.

Chronic traumatic encephalopathy

Chronic traumatic encephalopathy.

Deep-Vein Thrombosis

Deep-Vein Thrombosis.

Delayed deterioration

Delayed deterioration.

Coagulopathy

The occurrence of coagulopathy in patients with traumatic brain injury (TBI) is related to severe complications. The authors performed the first systematic review to investigate whether biomarkers can predict the occurrence of hypocoagulopathy or progressive hemorrhagic injury in patients with TBI. Methods: The authors included studies that performed a receiver operating characteristics analysis for the biomarker and provided a clear value along with the respective sensitivity and specificity. Additionally, they attempted to classify each biomarker, taking into account its physiological role. Results: Twelve studies were included. All biomarkers were protein molecules, except in one study that examined the prognostic role of glucose. Copeptin had the highest sensitivity, and S100A12 had the highest specificity in predicting coagulopathy, while IL-33 had the highest sensitivity and GALECTIN-3 had the highest specificity in predicting progressive hemorrhagic injury. Conclusion: The study of the role of biomarkers in predicting the occurrence of coagulopathy in patients with TBI remains in its infancy [4)].

Disseminated intravascular coagulation

Disseminated intravascular coagulation.

Empty sella syndrome

Empty sella syndrome.

Growing skull fracture

Growing skull fracture.

Nerve palsy

Abducens nerve palsy.

Oculomotor nerve palsy.

Olfactory loss

Olfactory loss due to head trauma is a frequent finding. It is attributed to the tearing or severing of the olfactory fibers at the cribriform plate. In contrast, posttraumatic gustatory loss is observed and reported rarely and the underlying mechanism is less understood. Rahban et al. present a case of a concomitant post-traumatic anosmia and ageusia. Imaging showed a considerable frontobasal brain damage and it is speculated that the gustatory impairment is due to a central injury of the secondary taste cortex. Based on this observation, Rahban et al.we believe that this clinical presentation might be much more frequent than previously reported [5)].

Autonomic impairment after acute traumatic brain injury has been associated independently with both increased morbidity and mortality. Links between autonomic impairment and increased intracranial pressure or impaired cerebral autoregulation have been described as well. However, relationships between autonomic impairment, intracranial pressure, impaired cerebral autoregulation, and outcome remain poorly explored.

Osteomyelitis of the skull

Skull Osteomyelitis.

Pituitary dysfunction

Diabetes insipidus.

see Posttraumatic hypopituitarism

hypogonadotropic hypogonadism

Pneumonia

Pneumonia in traumatic brain injury

Postconcussive syndrome

Postconcussive syndrome

Posttraumatic epilepsy

see Posttraumatic epilepsy.

Posttraumatic hydrocephalus

Posttraumatic hydrocephalus.

Posttraumatic meningitis

Posttraumatic meningitis.

Posttraumatic stress disorder

Posttraumatic stress disorder.

Pulmonary embolism

Pulmonary embolism.

Secondary Parkinsonism

Secondary parkinsonism

SIADH

SIADH.

Subdural empyema

Subdural empyema.

Traumatic intracranial hemorrhage

Traumatic intracranial hemorrhage.

Post-traumatic hearing loss

Post-traumatic hearing loss

Spasticity

Findings advocate for a person-centered approach in spasticity management, emphasizing the integration of sensory impairment strategies into rehabilitation to enhance functional outcomes and quality of life. Such an approach aims to improve functional outcomes and enhance the quality of life for individuals experiencing spasticity post-stroke or TBI. Future directions include targeted interventions to alleviate these sensations, support better rehabilitation results and improve patient experiences [6].

References

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