Traumatic brain injury complications

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
   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

Alzheimer’s disease.

Brain abscess

Traumatic brain abscess.

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.

Cerebral contusion

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

Cerebrospinal fluid otorrhea

Cerebrospinal fluid rhinorrhea

Chronic traumatic encephalopathy

Deep vein thrombosis

Delayed deterioration

Disseminated intravascular coagulation

Empty sella syndrome

Growing skull fracture

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. believe that this clinical presentation might be much more frequent than previously reported 4).

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

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.

References


