# Mild traumatic brain injury

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## Definition

Mild TBI, often called “concussion,” is defined by a GCS of 14 to 15 and accounts for over 80% of TBI [1].

see Mild traumatic brain injury definition.

## Epidemiology

Mild traumatic brain injury epidemiology.

## Classification

see Mild Traumatic Brain Injury Classification.

## Biomarkers

see Mild Traumatic Brain Injury Biomarkers.

S100B in mild traumatic brain injury

## Neurometabolic cascade

Recommendation: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3211100/

The initial ionic flux and glutamate release result in significant energy demands and a period of metabolic crisis for the injured brain. These physiological perturbations can now be linked to clinical characteristics of concussion, including migrainous symptoms, vulnerability to repeat injury, and cognitive impairment. Furthermore, advanced neuroimaging now allows a research window to monitor postconcussion pathophysiology in humans noninvasively. There is also increasing concern about the risk for chronic or even progressive neurobehavioral impairment after concussion/mild traumatic brain injury. Critical studies are underway to better link the acute pathobiology of concussion with potential mechanisms of chronic cell death, dysfunction, and neurodegeneration [2].

### Glutamate release and ionic disequilibrium

As a result of mechanical trauma, neuronal cell membranes and axons undergo disruptive stretching, leading to temporary ionic disequilibrium [3].

As a result, levels of extracellular potassium increase drastically, and indiscriminate glutamate release occurs [4].

Glutamate release activates N-methyl-D-aspartate receptors, which leads to accumulation of intracellular calcium [5] [6] [7] , causing mitochondrial respiration dysfunction, protease activation, and often initiating apoptosis [8] [9]. Elevated glutamate levels were also found to be significantly correlated with derangements in lactate, potassium, brain tissue pH, and brain tissue CO2 levels in human studies [10]. Additionally, sodium channel upregulation, fueled by ATPase proteins depending on glucose for energy, is observed following axonal stretch injuries [11].

### Energy crisis and mitochondrial dysfunction

In combination, the cellular response to the above-mentioned ionic shifts and the downstream effects of the neurotransmitter release lead to an acute energy crisis. This occurs when, to restore ionic equilibrium, adenosine-triphosphate (ATP) -dependent sodium-potassium ion transporter pump activity increases, which augments local cerebral glucose demand [12].

Further metabolic demand is incurred by ATP-dependent sodium channel upregulation. This occurs in the face of mitochondrial dysfunction, leading cells to primarily utilize glycolytic pathways instead of aerobic metabolism for energy, and causing extracellular lactate accumulation as a byproduct [13]. This acidosis, caused by hyperglycolysis, has been shown to worsen membrane permeability, ionic disequilibrium, and cerebral edema [14].

Some evidence shows that the lactate produced by this process may eventually be utilized as a source of energy by the neurons once mitochondrial oxidative respiration normalizes; in fact, one study showed that in moderate to severe TBI the incidence of abnormally high levels of lactate uptake were seen in 28% of subjects [15]. The same study showed that patients exhibiting a higher rate of

brain lactate uptake relative to arterial lactate levels tended to have more favorable outcomes compared to others with lower relative lactate uptake.

### Alterations in cerebral blood flow

Some studies have shown that cerebral blood flow decreases immediately following the insult, and the amount of time it remains lowered seems to depend on the severity of the injury [16) 17)].

Other studies, however, show no significant differences in CBF following mild TBI in subjects over 30 years of age [18)]. In pediatric studies, CBF has been seen to increase during the first day following mild TBI, followed by decreased CBF for many days after [19) 20)]. Data comparing cerebral blood flow in pediatric TBI patients has shown impaired autoregulation in 42% of moderate and severe and 17% of mild injuries [21)].

### Histopathologic changes

The underlying histopathologic changes that occur are relatively unknown. In order to improve understanding of acute injury mechanisms, appropriately designed pre-clinical models must be utilized.

The clinical relevance of compression wave injury models revolves around the ability to produce consistent histopathologic deficits. Mild traumatic brain injuries activate similar neuroinflammatory cascades, cell death markers and increases in amyloid precursor protein in both humans and rodents. Humans, however, infrequently succumb to mild traumatic brain injuries and, therefore, the intensity and magnitude of impacts must be inferred. Understanding compression wave properties and mechanical loading could help link the histopathologic deficits seen in rodents to what might be happening in human brains following concussions [22)].

## Clinical Features

see Mild traumatic brain injury clinical features.

## Diagnosis

see Mild traumatic brain injury diagnosis.

## Management

Mild traumatic brain injury management.

# Guidelines

Mild traumatic brain injury guideline

# Treatment

Mild traumatic brain injury treatment.

# Complications

see Mild traumatic brain injury complications.

# Case series

Mild traumatic brain injury case series.

# Case reports

Mild traumatic brain injury case reports.

# Mild traumatic brain injury case reports from the General University Hospital Alicante

## I14973

The patient was involved in a high-impact bus collision and initially presented with a Glasgow Coma Scale (GCS) score of 15. Emergency medical services (EMS) noted amnesia of the incident but no focal neurological deficits. Despite this, her condition necessitated further evaluation due to the severity of the accident. She was hemodynamically stable upon presentation, leading to her transfer to the emergency department (ED) of Hospital de (Hospital Name).

Clinical Examination:

GCS: 15 Blood Pressure: 134/82 mmHg Heart Rate: 82 bpm Oxygen Saturation: 99% (room air) Temperature: 36.2°C The patient was normocolored, well-hydrated, and oriented. Neurological examination revealed intact cranial nerves, normal motor and sensory function, and a stable mental status. An occipital laceration was sutured.

Diagnostic Imaging:

CT Brain:

Diffuse SAH predominantly in the frontoparietal regions and Sylvian fissures. Bilateral frontobasal contusions, with greater involvement on the right side. Bilateral frontal subdural hematomas with extension into the anterior interhemispheric fissure. Occipital fracture extending into the clivus, with a minor fracture on the right clivus. CT Cervical Spine: Normal alignment with no fractures.

CT Thorax:

Focal ground-glass opacity in segment 6 of the left lung, suggesting possible infection, inflammation, or minor pulmonary contusion. No pleural or pericardial effusions. CT Abdominal-Pelvic:

Ovarian cyst (3.6 cm) on the right. No significant injuries to solid organs or free fluid. Clinical Management:

The patient maintained a stable GCS throughout her hospitalization. Severe headaches and nausea were managed with continuous infusion of dexketoprofen, tramadol, and metoclopramide, resulting in significant symptomatic relief. She was hemodynamically stable, afebrile, and demonstrated adequate respiratory function.

Consultations:

Ophthalmology: Evaluated for diplopia and papilledema. Follow-up with neuro-ophthalmology was recommended for further evaluation of vision changes. Outcome:

The patient exhibited favorable clinical and radiological outcomes. She was stable upon discharge with a GCS of 15, no new neurological deficits, and no signs of infection or additional complications. Outpatient follow-up included neurosurgery and neuro-ophthalmology consultations.

## I14966

A 63-year-old male was found unconscious in the street with an occipital scalp laceration following alcohol consumption. He had no recollection of the events leading up to his condition. His medical history included a past hematoma, craniectomy, and a prior TBI in the context of alcohol intoxication.

Clinical Findings: On initial examination, the patient had a Glasgow Coma Scale (GCS) score of 14. He exhibited difficulty with gait and slow speech. A fluctuating cephalohematoma was noted on physical examination. The patient's vital signs were stable, with a blood pressure of 130/87 mmHg, heart rate of 80 beats per minute, and oxygen saturation of 98% on room air.

Imaging and Diagnostic Workup:

CT Brain: Showed a right frontobasal intraparenchymal hematoma up to 29 mm, with associated extra-axial hemorrhage in the frontotemporal and parietal regions, as well as a subgaleal hematoma. Notable findings included a fracture of the posterior inferior left parietal bone and changes consistent with a previous craniectomy. CT Cervicothoracic-Abdominopelvic: Excluded significant thoracic or abdominal pathology but identified a hemangioma in the liver and an aneurysm of the right common iliac artery with thrombus. Management:

Initial Treatment: The patient received fluid therapy (500 ml of 5% dextrose) and intramuscular thiamine. Pain management was achieved with conventional analgesics. Neurological Monitoring: Regular monitoring was performed, with follow-up CT scans showing stability of intracranial lesions.

Outcome: The patient was stable, conscious, and oriented throughout his ICU stay. No new neurological deficits were observed. Given the stability of the patient's condition and the absence of significant progression of lesions, he was transferred.

## I14543

A 92-year-old man with a fall-related injury. The patient has a history of polycythemia vera under treatment with acenocoumarol.

Clinical Findings: The patient was admitted to the emergency department due to traumatic brain injury with an incised contused wound in the left frontal region. An urgent non-contrast-enhanced Computed Tomography (CT) scan of the brain was performed.

Results of the Brain CT Scan:

Linear hyperdensities were identified in the right parasagittal frontal sulci and in the anterior interhemispheric fissure, without significant associated mass effect, suggesting small foci of subarachnoid hemorrhage. Focal hypodensities were observed in the right thalamus and right basal ganglia, consistent with chronic lacunar infarctions. The midline is centered, and the basal cisterns are permeable. . Evolution: On the second day of admission, the patient is afebrile with a blood pressure of 134/77. He presents a somewhat bradypsychic general status, but according to his son, this is consistent with his baseline state. No other relevant clinical focalities are evident. The left frontal contused wound appears to be in good condition.

The follow-up CT scan was the same

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Paroxysmal Atrial Fibrillation: The patient is anticoagulated with apixaban (Eliquis). History of Stroke (Cerebrovascular Accident - ACV): Previous experience of a vertebrobasilar ischemic stroke, treated with mechanical thrombectomy. Chronic Kidney Disease (CKD): With a glomerular filtration rate (GFR) of 33 ml/min in the last measurement. Ophthalmological Issues: Bilateral blindness with a history of central retinal vein thrombosis in the left eye. Surgical Interventions: Include bilateral herniorrhaphy, tonsillectomy, osteosynthesis of right malleolus fracture, and excision of basal cell carcinoma on the left shoulder. Baseline:

Partially dependent for Activities of Daily Living (ADL) due to bilateral blindness. Functional Classification (FC) NYHA II-III/IV (according to the New York Heart Association classification). Adequate support from family members and a caregiver. Current Treatment: Includes medications for blood pressure control, anticoagulation, vitamin supplements, statins, and medications for anxiety and gastroesophageal reflux.

Findings in Brain CT:

Traumatic subarachnoid hemorrhage (SAH): Hemorrhagic foci are observed on the frontal-superior left lobar surface and on the anterior lobar surface of the right temporal lobe. These are suggestive of post-traumatic hemorrhagic contusions. Subgaleal Hematoma: A subgaleal hematoma of up to 18 mm is present in the right frontal region in relation to the post-traumatic contusion. Chronic Microangiopathy: Images suggestive of chronic microangiopathy (leukoaraiosis) are seen in the deep white matter, indicating age-related changes in the brain. Global Ventriculomegaly: Attributable to cerebral volume loss and atrophy, without associated mass effect. Evans Index: Within normal range

Conclusion:

Confirmation of post-traumatic subarachnoid hemorrhage without complications in subsequent clinical and radiological follow-up.

Consultation with hematology is requested to restart anticoagulant therapy. In summary, the patient presents findings related to mild traumatic brain injury, and the continuation of treatment, including the anticoagulation Resumption after hematological evaluation, is planned. The importance of clinical and radiological follow-up is emphasized to ensure the absence of complications.

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