Chapter 14: Traumatic brain injury (TBI)

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1 Introduction

A traumatic brain injury (TBI) refers to injury to the brain, whereas head injury is a broader concept involving injury to any structures of the head. A head injury can be closed or penetrating. A TBI can further be due to blunt force trauma, acceleration-deceleration or rotational forces, a blast exposure to the head, or can be due to a penetrating injury e.g., a bullet or sharp object. The severity of a TBI is classified according to a combination of the Glasgow Coma Scale (GCS, Table 1), the duration of loss of consciousness (LOC) and duration of post-traumatic amnesia (PTA, Table 2). PTA denotes the period of delirium (disorientation, confusion, and disrupted anterograde and retrograde memory function) that follows a TBI. A mild TBI is also often referred to as a concussion. If a mild TBI has radiological evidence of traumatic intracranial injuries (e.g., haematomas, haemorrhages, or contusions) then it is referred to as a complicated mild TBI (Lepage et al., 2020).

Table 1: Glasgow Coma Scale (GCS)

Response scale	**Score**
Eye opening response	
Spontaneously	4
To speech/command	3
To pain (not applied to the face)	2
None	1
Verbal response	
Orientated	5
Confused conversation	4
Inappropriate words	3
Incomprehensible speech	2
None	1
Motor response	
Obeys commands	6
Purposeful movement to pain	5

	Withdraws from pain	4
	Decorticate (spastic flexion) posture to pain	3
	Decerebrate (rigid extension) posture to pain	2
	None	1
	Total:	__ / 15
Mild: 13 – 15; Moderate: 9 – 12; Severe ≤ 8		

Table 2: Severity classification of traumatic brain injury (TBI)

Injury characteristics	**Mild TBI**	**Moderate TBI**	**Severe TBI**
LOC	≤ 30 min	30 min – 24 hrs	≥ 24 hrs
PTA[a]	≤ 24 hrs	24 hrs – 7 days	≥ 7 days
Initial GCS	13 – 15[b]	9 - 12	3 - 8
[a] Includes alteration of consciousness (e.g., confusion or disorientation) [b] Not < 13 at 30 min GCS, Glasgow Coma Scale; LOC, loss of consciousness; PTA, posttraumatic amnesia			

2 Epidemiology

2.1. Prevalence

TBIs are a major contributor to disability and mortality globally. The burden of TBIs is higher in Sub-Saharan Africa than the global average (Adegboyega et al., 2021). Although the majority of TBIs are of mild severity, they have an immense impact on society due to the economic costs related to permanent disability, frequently in young people.

2.2. Aetiology

Closed head injury is a much more common cause of TBI than penetrating head injuries. In the United States (US) the most common causes of TBIs are falls, followed by the head being struck by/to an object, road traffic accidents (RTAs) and assaults (Byars and Jorge, 2017). Whereas in Sub-Saharan Africa the most frequent causes of TBIs are RTAs, followed by assaults and falls (Adegboyega et al., 2021). Falls occur at higher rates in young children and elderly, whereas RTAs and assaults occur most frequently in young adults. The most frequent causes of mortality are due to firearms, RTAs and falls. Mild TBIs in the context of contact sports are observed most frequently in adolescents and young adults (the age groups commonly engaged in contact sports).

2.3. Risk factors

TBIs are more frequently observed in young children, the elderly, and adolescents. In Sub-Saharan Africa, TBIs are more common among young males (aged 20-40 years) (Adegboyega et al., 2021). Males have higher rates of TBIs which are linked to greater risk-

taking behaviours. Rates of TBIs are also higher in lower socio-economic strata and in both the US and South Africa rates are higher in black compared to white ethnic groups. Substance use, particularly alcohol, is associated with greater risk for TBI. Prior TBIs also increase the risk of subsequent TBIs, with the risk increasing with number of prior TBIs.

3 Pathophysiology

3.1. Classification of mechanisms of injury

Mechanisms of head injury can be classified according to whether the injury is closed or penetrating, into primary and secondary mechanisms of injury and into focal or diffuse injury (Figure 1). A TBI can involve different mechanisms e.g., focal and diffuse injury co-occurring. In penetrating or open TBIs the extent of damage is dependent on the velocity of the object, with higher velocity objects (e.g., bullets) producing more widespread injuries and lower velocity objects (e.g., knife) producing more localised damage. The damage that results directly from the forces during the injury are the *primary mechanisms*, whereas the processes that cause injury at any time point after the initial insult are the *secondary mechanisms* of injury. The consequences of inertial forces tend to be more severe if there are rotational forces as well as acceleration/deceleration forces (Fleminger, 2009). Subdural hematoma can result from inertial forces due to tearing of the bridging veins. Secondary injury is more likely in more severe TBI. Focal brain lesions (e.g., contusions, lacerations, and haemorrhages) tend to occur on the surface of the brain and particularly in the frontal and temporal lobes, and intracerebral haemorrhages also frequently involve the basal ganglia. Traumatic (or diffuse) axonal injury (TAI) tends to affect the central white matter and the grey-white interface, the corpus callosum, thalamus and the dorsolateral upper brainstem.

3.2. Pathophysiology

There are various pathways through which TBI cause injury.

TBI can result in

- Cell death (necrosis and apoptosis).
- Microglial activation and inflammation.
- Repair mechanisms such as reactive synaptogenesis and axonal sprouting.

TAI mechanisms of injury

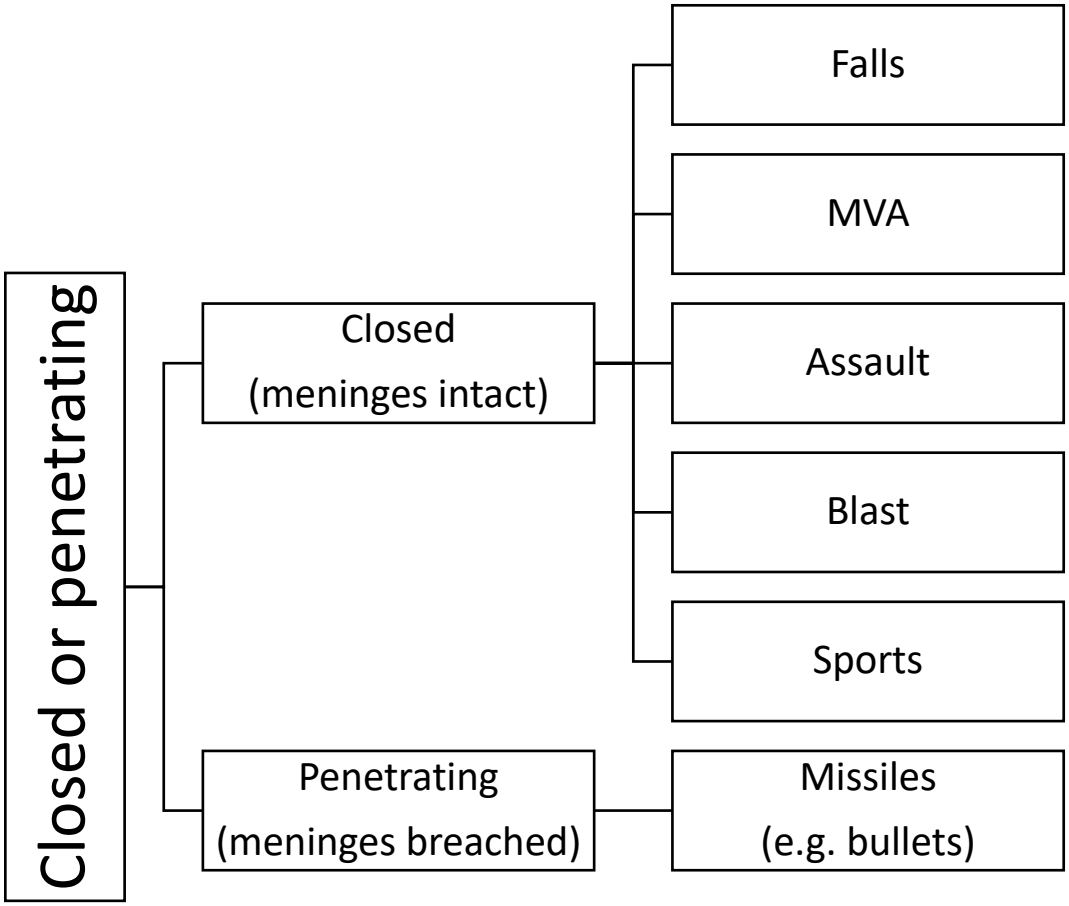
- Stretching of axons and myelin sheaths.
- Changes in cell membrane permeability leading to an influx of calcium.
- Calcium leads to impaired oxidative metabolism in mitochondria.

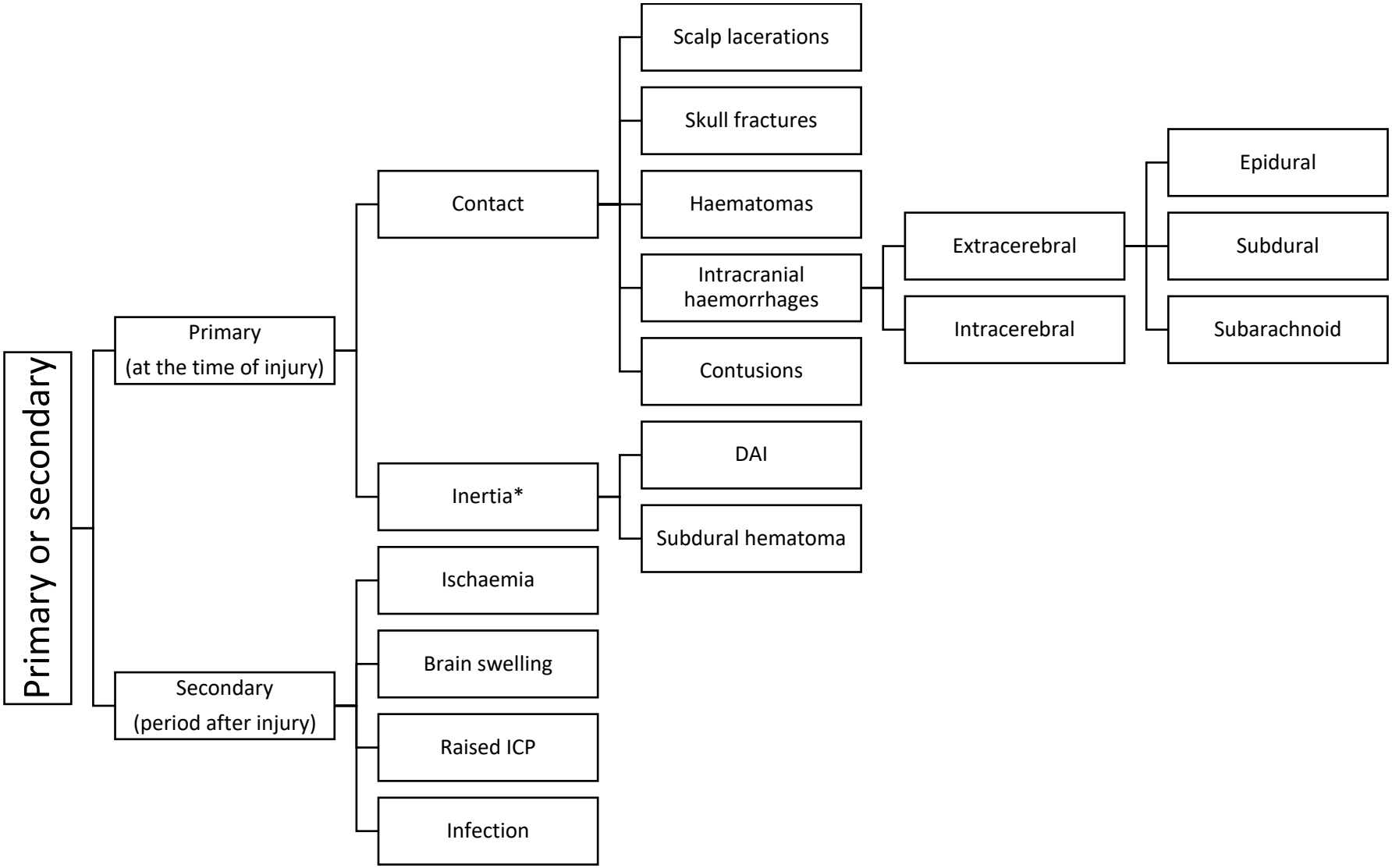
- Microtubules break down.
- Axonal dysfunction and resultant astrogliosis and microglial activation.

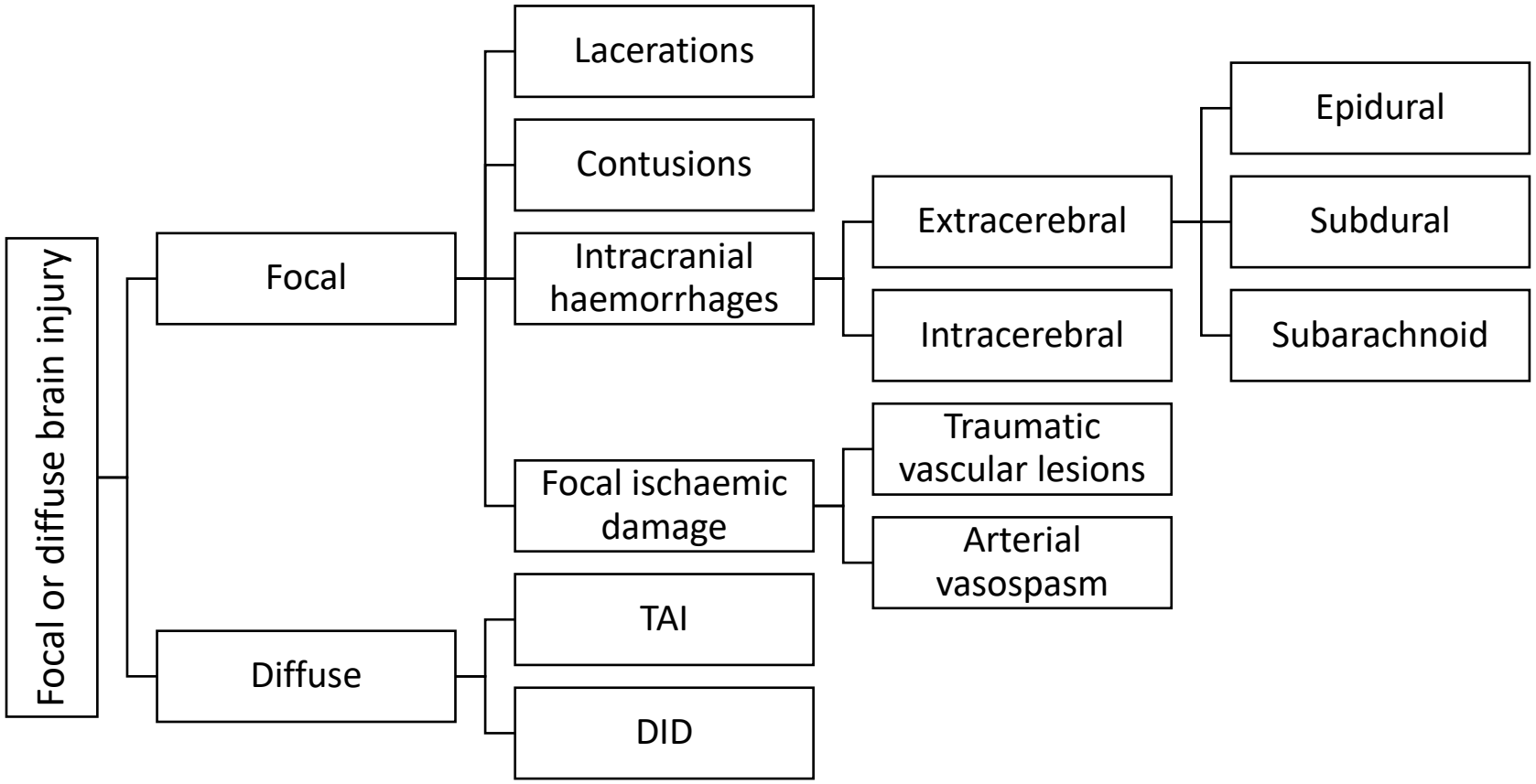
Changes in neurotransmitter systems

- Increased glutamate and N-methyl-D-aspartate (NMDA) receptor stimulation with consequent excitotoxicity.
- Decreased inhibitory gamma-aminobutyric acid (GABA) neurotransmission.
- Early increased cholinergic activity and later reduction, especially in the hippocampus and neocortex.
- Early increased monoamine levels (serotonin, noradrenaline) and possible later down-regulation.
- Dysregulation of dopamine pathways.

Figure 1: Classification of traumatic brain injury (TBI)




Primary or secondary
Primary
(at the time of injury)
Secondary
(period after injury)
Contact
Inertia*
Ischaemia
Brain swelling
Raised ICP
Infection
Scalp lacerations
Skull fractures
Haematomas
Intracranial haemorrhages
Contusions
DAI
Subdural hematoma
Extracerebral
Intracerebral
Epidural
Subdural
Subarachnoid



* Acceleration/deceleration and rotational

DAI, diffuse axonal injury; DID, diffuse ischeamic damage; ICP, intracranial pressure; MVA, motor vehicle accident; TAI, traumatic axonal injury

4 Investigations

Neuroimaging (computed tomography [CT] and magnetic resonance imaging [MRI]) is routinely used to evaluate patients with TBI. CT is indicated when there are rapid changes in a patient's neurological condition as it can detect hematomas, but MRI is better at visualising other lesions such as contusions, white matter lesions and small subdural haemorrhages. Certain MRI sequences, such as diffusion weighted imaging (DWI), gradient echo (GRE) and diffusion tensor imaging (DTI) are better for identifying axonal injury. Atrophy on imaging is associated with persisting cognitive impairments. Functional imaging, such as positron emission tomography (PET) and single photon emission computed tomography (SPECT) can be useful in identifying mild TBI.
Electroencephalogram (EEG) is used to identify a post traumatic seizure focus and to diagnose status epilepticus and brain death. There is a search to identify diagnostic and prognostic serum biomarkers for TBI. Although none have been established for clinical use, research has demonstrated that markers of neuronal injury, such as serum S-100B, correlate with severity and outcomes of TBI.

5 Consequences of TBI

Acute symptoms of TBI include altered consciousness or coma, disorientation, headache, nausea, and dizziness. Seizures can also occur and can contribute to secondary injury. The risk for seizures is highest in the first 6 months post injury. In the chronic phase TBI can result in neurological impairments, such as paresis, speech impairments and neuropathies. In studies from Sub-Saharan Africa, cognitive effects are most frequently reported post TBI, followed by physical and emotional/behavioural consequences (Adegboyega et al., 2021). TBI can also result in injury to the pituitary which can result in acute and longer term endocrinopathies. In the acute phase diabetes insipidus due to antidiuretic hormone (ADH) deficiency is most commonly observed and later growth hormone deficiency is most common.

Mild TBI can present with symptoms of concussion which include headache, nausea, dizziness, fatigue, irritability, light and sound sensitivity, attention and memory impairments and slowed processing speed (these can be grouped into cognitive, emotional and somatic symptoms). These typically improve and resolve over days to weeks. If symptoms persist beyond 3 months a post-concussive syndrome (PCS) should be considered. PCS shares features with somatic symptom disorders, with anxiety and pre-occupation with symptoms disrupting normal recovery. Risk factors include psychological and personality factors, involvement in litigation, and excessive rest.

A number of neuropsychiatric disorders and problems can develop secondary to TBI (Table 3). To diagnose a disorder as being due to TBI, the onset of the disorder should be temporally related to TBI (onset shortly after TBI) and must be biologically plausible i.e., the area/extent of injury could explain the symptoms. However, TBI can also be a risk factor for the later development of neuropsychiatric disorders. TBI related variables, such as the severity, location, and post-traumatic epilepsy play a role in the neuropsychiatric disorders and problems that develop. Psychiatric disorders prior to the TBI also increase the risk of psychiatric problems post TBI.

Table 3: Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) neuropsychiatric disorders and problems that can develop in relation to traumatic brain injury (TBI)

Neurocognitive disorders (NCDs) Delirium due to TBI Major NCD due to TBI Mild NCD due to TBI
Personality disorders Personality change due to TBI
Neurodevelopmental disorders Intellectual developmental disorder (IDD) When TBI occurs in the developmental period. If loss of previously acquired cognitive skills – can diagnose both IDD and NCD.
Schizophrenia and other psychotic disorders Psychotic disorder due to TBI
Bipolar and related disorders Bipolar and related disorder due to TBI
Depressive disorders Depressive disorder due to TBI
Anxiety disorders Anxiety disorder due to TBI
Trauma- and stressor-related disorders Posttraumatic stress disorder (PTSD) Acute stress disorder Adjustment disorders
Substance-related and addictive disorders Substance use disorders
Somatic symptom and related disorders Somatic symptom disorder Excessive thoughts, feelings or behaviours related to somatic symptoms of the TBI or consequences thereof. Psychological factors affecting other medical conditions Psychological factors that interfere with the course or treatment of TBI. Factitious disorder Intentional production of symptoms to present as ill/injured in the absence of obvious external rewards.
Sleep-wake disorder Insomnia disorder

Hypersomnolence disorder
Other Other specified mental disorder due to TBI Unspecified mental disorder due to TBI Other conditions that may be a focus of clinical attention Malingering involves intentional production of symptoms or exaggerating symptoms motivated by external rewards, such as financial compensation, avoiding work, or obtaining drugs.

5.1. Acute neuropsychiatric presentations

The acute neuropsychiatric presentations are primarily those related to delirium, such as agitation, restlessness, and PTA. The delirium can also include perceptual disturbances, delusions, and affective lability. Irritability is also observed post TBI, including mild TBI. Agitation can hamper rehabilitation and worsen prognosis. Multiple factors can contribute to delirium post TBI, including cerebral injury, brain oedema, hypoxia, seizures, infections, metabolic disturbances, medications and drug or alcohol withdrawal.

5.2. Neurocognitive impairments (NCI)

According to the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) a major or mild neurocognitive disorder (NCD) due to TBI can be diagnosed it the criteria for a major or mild NCD are met and there is evidence of a TBI, with one or more of: LOC, PTA, disorientation and confusion, and neurological signs (on clinical examination or imaging) (American Psychiatric Association, 2013). Importantly, the NCD has to have an onset immediately following the TBI, or as soon as consciousness is regained, and should last beyond the acute post-injury period. Other cognitive problems that can develop in relation to TBI that are not included within the DSM-5 NCD due to TBI are chronic traumatic encephalopathy (CTE) and TBI associated increased risk for the later development of other NCDs e.g., Alzheimer's disease.

Cognitive impairments following TBI are variable, but commonly involve complex attention, executive function, learning, memory, processing speed, and social cognition. In TBIs with focal injuries there can be other specific deficits, such as aphasia and apraxia. In addition, individuals may often have subtle neurological signs, such as primitive reflexes or impaired saccades or smooth-pursuit eye movements. Individuals with NCD secondary to TBI often have anosognosia and may thus be relatively unaware of their deficits.

In addition to cognitive impairment, individuals with NCD due to TBI often have personality and behavioural changes, such as aggression, lability, disinhibition, and apathy. In addition,

they may have other somatic complaints including headache, sensitivity to noise and light, fatigue, and dizziness.

Although more severe TBI is associated with a greater risk of NCD due to TBI, the severity of the TBI does not always correlate with the severity of the NCD as other factors also play a role. Risk factors for poorer cognitive outcomes include repeated TBI, older age, low premorbid cognitive function, comorbidity (medical and psychiatric), substance use, genetic factors such as apolipoprotein E4 (APOE4) allele carries, and lack of access to psychosocial support and rehabilitation interventions. Generally, impairment is worst in the early aftermath of the TBI with improvement noted over weeks to months. Improvement can continue over years, although the progress becomes more gradual. With a mild TBI the NCI generally resolve within days to weeks, with complete resolution within a few months.

Neurological signs and imaging findings may align with cognitive impairment, e.g., frontal encephalomalacia and primitive reflects with executive function deficits. There are no biomarkers of NCD due to TBI and the diagnosis is made clinically with the aid of neuropsychological testing (where available and applicable). NCD due to TBI is associated with significant functional impairments related to the cognitive deficits, but also associated physical and neurological deficits, personality and behaviour changes and comorbid psychiatric disorders.

Beyond directly causing a NCD, TBI may be a risk factor for later development of a NCD, particularly more severe TBI. The risk seems to be more pronounced for men. TBI may contribute to risk of dementia by lowering cognitive reserve, by increasing risk for cerebrovascular disease, or by initiating a cascade e.g., amyloid deposition, that leads to neurodegeneration.

5.2.1 Chronic traumatic encephalopathy (CTE)

CTE denotes a neurodegenerative disease that develops secondary to repetitive (usually mild) TBIs diagnosed based on characteristic deposits of hyperphosphorylated tau (p-tau) on neuropathology. Traumatic encephalopathy syndrome (TES) refers to the clinical entity observed in relation to CTE.

The consensus criteria for TES (Katz et al., 2021) require:

- A history of repetitive head impacts (such as through contact sports, military service, or domestic violence).
- Progressive cognitive impairment and/or neurobehavioural dysregulation.

    - Cognitive impairment entails subjective report of a decline in cognitive function, alongside deficits on testing, primarily in episodic memory and executive function.
    - Neurobehavioural dysregulation entails a change in the baseline functioning involving difficulty regulating emotions and behaviour, such as anger outbursts and emotional lability.
- The clinical presentation is not better explained by another neurocognitive, medical or psychiatric disorder or condition.

The diagnosis of TES is supported by a delayed onset of impairments (i.e., not directly following the head impacts - suggesting a neurodegenerative process), motor signs including parkinsonism and motor neuron disease, and psychiatric features, including anxiety, apathy, depression, and paranoia. The criteria for TES are for use in research settings and should not be applied in standard clinical practice at this stage.

5.3. Personality changes due to TBI

Personality changes may be a prominent feature post TBI, with disinhibition, aggression and apathy commonly observed. The DSM-5 criteria for personality change due to another medical condition denote a personality change from the person's previous characteristics, which causes distress and impairment. In the case of TBI the personality changes develop directly after the TBI and are not limited to the period of delirium or better explained by another mental disorder. The subtypes of personality change include labile, disinhibited, aggressive, apathetic, paranoid, other, combined, and unspecified types. Personality changes can occur in the context of a NCD but can also be diagnosed as a distinct disorder if the personality change is due to the TBI and is prominent in the clinical picture. The location of the TBI can be associated with specific personality changes e.g., frontal lobe injury with apathy, disinhibition, affect dysregulation and hypersexuality and temporal lobe injury with aggression and poor impulse control.

5.4. Mood disorders

Depressive disorders commonly develop following TBI and if developing directly following the TBI and aetiologically linked can be diagnosed as a depressive disorder due to TBI. TBI also appears to increase the risk for later depressive episodes. Depressive disorders are the most frequently observed psychiatric disorders following TBI, with rates of 25-50% reported during the first year. Risk factors include prior depressive or anxiety disorders, substance use, and poor social functioning and socio-economic circumstances. TBI appears to play an aetiological role in the depressive disorders as rates are higher than comparably injured patients without TBI. Depressive disorders are associated with greater aggression and

suicide risk, although patients with TBI also appear to have an increased risk of suicide independent of depression.

In the context of manic or hypomanic symptoms developing post TBI a bipolar and related disorder due to TBI can be diagnosed. Mood disorders have a detrimental effect on functioning and recovery of patients with TBI.

5.5. Trauma- and stressor-related disorders

Patients with TBI are more likely to develop posttraumatic stress disorder (PTSD) than patients with other injuries. Damage caused by TBI can impair the ability to regulate fear responses to the trauma. There is symptom overlap between TBI and PTSD, such as impaired concentration, anger outbursts and sleep difficulties which can complicate diagnosis. PTSD and TBI appear to act synergistically to worsen both functioning and recovery from TBI and PTSD symptoms. Brief mood, anxiety and behavioural symptoms on the milder spectrum that develop post TBI could also fit in with a diagnosis of an adjustment disorder.

5.6. Anxiety disorders

There is limited research regarding anxiety disorders, although these can also develop secondary to TBI. Generalised anxiety disorder (GAD) may be the most frequent anxiety disorder in the context of TBI. Patients with TBI also have increased rates of panic disorder, agoraphobia, and social anxiety disorder. Patients also frequently present with depression with anxious distress which is associated with poorer outcomes than depression alone.

5.7. Substance-related and addictive disorders

Substance use disorders (SUD) are a risk factor for TBI and have a significant influence on outcomes. SUD can worsen cognitive, rehabilitation, psychiatric, and psychosocial outcomes post TBI. TBI can also contribute to greater difficulty controlling addictive or impulsive behaviours related to SUD.

5.8. Psychotic disorders

A psychotic disorder secondary to TBI can be diagnosed if there are prominent hallucinations and delusions occurring post-TBI that arise outside of delirium. Psychosis secondary to TBI is associated with cognitive impairment, seizures, and imaging and EEG changes in the frontal and temporal lobes. TBI may also be a risk factor for the development of schizophrenia.

5.9. Other problems

Pseudobulbar affect (PBA) or pathological laughter and crying involves sudden spontaneous uncontrollable affective outbursts (e.g., crying or laughing spells). Patients report that the emotional outburst are out of proportion to their underlying mood. PBA is seen especially following frontal lobe lesions.

Apathy is commonly observed post TBI and may present as personality change due to TBI. Apathy denotes an impairment in motivation, drive, initiation, and emotional responsiveness. Although apathy does not involve low mood or sadness, it can co-occur with depression. Apathy can have a marked impact on engagement in treatment and functional outcomes and is often of greater concern to caregivers than patients. Caregivers should be psycho-educated about the nature of apathy and encouraged to assist the person with apathy to engage in activities.

6 Treatment of neuropsychiatric disorders in the context of TBI

Early treatment of neuropsychiatric problems may improve outcomes for patients with TBI. There is limited evidence to guide the treatment of psychiatric disorders due to TBI. Standard treatment approaches used in these conditions (e.g., depression or psychosis) are generally recommended with some specific considerations related to TBI. A holistic multidisciplinary biopsychosocial treatment approach is advocated.

6.2. Psychosocial interventions

A multidisciplinary comprehensive rehabilitation programme may produce the best outcomes for patients with TBI. Psychoeducation of patients and caregivers should form a component of the management of all neuropsychiatric disorders. Patients and caregivers can also be connected with available support groups for individuals with TBI and their families. Behavioural interventions can improve self-care, behaviour, and interpersonal skills in patients with TBI. Cognitive rehabilitation can be used to improve cognitive outcomes in NCD secondary to TBI. These can entail training to improve attention and memory, compensatory strategies (e.g., carrying a notebook) and metacognitive training. Psychotherapy can improve long-term outcomes and should be offered to patients and their caregivers when indicated. Evidence based psychotherapies, such as cognitive behavioural therapy (CBT), can be utilised to treat psychiatric disorders due to TBI, depending on the individual's ability to engage in them. Strategies such as motivational interviewing and substance interventions can assist in addressing SUD. Occupational therapy can assist in improving functioning and in helping patients return to work. TBI can place a significant burden on the family and caregivers and support structures should be harnessed to aid them. Supporting caregivers also improves outcomes for the person with the TBI.

6.3. Biological interventions

Individuals with TBI are more sensitive to drug side effects and therefore dosages should be started low and increased slowly up to standard therapeutic doses. Drugs that are not too sedating, that don't adversely affect cognition and that don't have a significant impact on seizure threshold are preferred in patients with TBI. Thus, benzodiazepines and drugs with potent anticholinergic effects should preferably be avoided.

Antipsychotics may be used in the management of delirium and agitation in the acute phase and in psychotic and bipolar disorders due to TBI, although impact of individual drugs on seizure risk should be taken into consideration. Other mood stabilisers (e.g., lithium and valproate) can also be used to manage bipolar disorders due to TBI

Antiseizure drugs (ASD) can also be useful in the management of epilepsy, delirium and agitation and to prevent seizures in the first week post severe TBI. Prophylactic ASD use in milder TBI and beyond 7 days is usually not indicated.

Stimulants (e.g., methylphenidate) can be used to improve attention and executive function in patients with NCDs secondary to TBI. Other agents that can be used to address NCI include amantadine, bromocriptine, cholinesterase inhibitors and memantine.

Selective serotonin reuptake inhibitors (SSRIs) are generally the first-line pharmacotherapy for depressive and anxiety disorders and PTSD in the context of TBI. Prazosin may be useful for PTSD-related nightmares. SSRIs can also be offered to assist in the management of PBA and apathy, although they have been reported to both improve and worsen apathy. Other options are combination dextromethorphan and quinidine for PBA and stimulants and dopamine agonists may be helpful for apathy. Neurostimulation approaches, such as electroconvulsive therapy (ECT) and transcranial magnetic stimulation (TMS) can also be used to treat depressive disorders post TBI, although seizure risk needs to be taken into consideration.

7 Course and prognosis

Structured measures like the Glasgow Outcome Scale (GOS) and the Glasgow Outcome Scale-Extended (GOSE) can be used to measure outcomes post TBI (Wilson et al., 2021). The GOS rates impairment ranging from death, a vegetative state, severe- and moderate-disability to good recovery. The GOSE includes questions regarding functioning in relation to following commands, independence in and out of the home, work/study, social and leisure activities and family relationships and friendships to assist in determining the level of disability and recovery.

TBI can result in death and significant morbidity and disability. About 1.1% of the US population have disability related to TBI. NCI can impair ability to return to work or studies

and behavioural changes can impact interpersonal relationships. Patient motivation and insight can influence rehabilitation outcomes. Outcomes are related to the location and severity of the TBI, age and treatment received. Outcomes for those with complicated mild TBI are more equivalent to those with moderate TBI. Studies from Sub-Saharan Africa have reported better outcomes in children compared to adults (Adegboyega et al., 2021). Other factors that influence outcomes include level of education, socioeconomic status, psychiatric morbidity, substance use, social functioning, and availability of rehabilitation and psychosocial support. Limited resources have a significant impact on TBI care in Sub-Saharan Africa and a lack of counselling, educational materials, and support are evident (Adegboyega et al., 2021).

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