Beacon Clinical Topic
Traumatic Brain Injury

The link between traumatic brain injury (TBI) and psychiatric illness is dramatically illustrated in the 1848 case of Phineas Gage, a railroad worker who sustained a penetrating frontal lobe injury that resulted in dramatic personality changes. "Shell shock" and "combat fatigue", terms coined in World War I for what are now considered PTSD symptoms, were initially considered secondary to the stress of combat. Several physicians—Frederick Mott, Bennet Omalu (portrayed in the 2015 movie "Concussion") and most recently Daniel Perl—have challenged this view, based on findings of physical changes in the brains of soldiers with PTSD. The current view is that some behavioral symptoms after a TBI result directly from brain changes while others are more related to emotional stress of the event.

1. DEFINITIONS

A TBI is defined as an external force to the brain (e.g., head strikes, rapid acceleration/deceleration, foreign body penetration, blasts or explosions) that results in one of five symptoms within 24 hours:

1. Loss of consciousness (LOC)
2. Post-traumatic amnesia (PTA)
3. Alteration of consciousness (AOC), including confusion, disorientation, slowed thinking
4. Transient or non-transient neurological deficits (weakness, imbalance, visual changes, praxis, paresis/plegia, aphasia, or sensory loss)
5. An intracranial lesion (Note: Sustaining a skull fracture without intracranial change is not a TBI.)

Severity:

- TBIs are classified as mild (approximately 80%) or moderate/severe (approximately 20%). LOC > 30 minutes and PTA > 24 hours must be one of the five symptoms in moderate/severe TBIs. Mild TBIs require only an altered mental status.
- A Glasgow Coma Scale between below 13 and a positive CT scan are also present in moderate-to-severe TBIs.

Epidemiology: TBI
Incidence: 1.5 - 2 million new TBIs annually in the US (0.5% of the general population).

Prevalence: Approximately 20% of the population has had a TBI during their lifetime.

TBI-associated psychiatric symptoms

- 30-40% prevalence at one-year post TBI, depending on severity and nature of injury.
- Mood, anxiety (including PTSD), substance use disorders, and subtle personality changes (due to frontal lobe effects on executive function) account for most post-TBI neuropsychiatric syndromes.
- The incidence of TBI psychosis, while rare, is up to 4x greater in patients who have had a TBI.

2. DIAGNOSIS

In the *DSM-5*, TBI is classified as one of the Neurocognitive Disorders (NCDs). This is a new section that replaces *DSM-IV*'s Dementia, Delirium, Amnestic, and Other Cognitive Disorders (d/o's), where TBI was classified as dementia due to head trauma. The four general criteria for a mild or major NCD are: a) moderately (in mild NCD) or significantly (in major NCD) reduced cognitive function from a previous level of performance; b) impairment of independent function (for major NCD only); c) the cognitive impairment is not exclusive to post-TBI period of delirium; and d) the cognitive deficit is not primarily due to another *DSM* diagnosis.

In this *DSM-5* section, if a specific physiological etiology for the NCD is known, it is listed. Thus for TBIs, the *DSM-5* diagnosis would be either mild or major NCD due to a TBI. For ICD-10 coding, a specifier is added as to whether the NCD presents with behavioral symptoms (though not in *DSM-5*).

3. ASSESSMENT

Clinical: Clinicians should interview both patient and family; gather the patient's pre-injury history; and conduct a brief neurological assessment. With unresponsive patients, they should try to get a minimal response, i.e., "open your eyes." With mute patients, they should try to elicit non-verbal responses.

Measurement: Symptom-specific tests complement the clinical interview (e.g., Apathy Evaluation Scale for motivation; Present State Examination for depression and anxiety; and Overt Aggression Scale for agitation).

Imaging: A CT scan is usually sufficient for mild TBIs. An MRI is usually needed to assess moderate/severe TBIs. DTI (tractography) is used to assess diffuse axonal injury and white brain matter changes in the brain.

Identifying Psychiatric Symptoms: The primary assessment question for behavioral health clinicians is to distinguish psychiatric conditions, such as depression, from behavioral symptoms secondary to the TBI. To do this, the clinician should make an assessment of whether a post-TBI behavioral condition meets *DSM* or ICD criterion for a diagnosis other than NCD secondary to TBI. The table below presents information to help with this distinction.

4. TREATMENT

Treatment involves the following modalities:

A. Rehab: Combined OT, PT, and speech and therapy, that includes a "neuro-cognitive" component
B. Environmental modifications: Managing number and relationship of visitors, optimizing sensory stimuli, optimizing time in bed, regulating sleep cycle, use of communicative augmentation devices in mute patients, providing daily schedules and other organization supports
C. Behavioral plans that address disruptive behavior and other psychiatric symptoms
D. Medical management of contributing factors to emotional distress (seizures, pain, spasticity, visual changes, nausea, infections, feeding impairments)
E. General principles of medication management in TBI:
    a. Sedating medications (benzodiazepines, antipsychotics) should be avoided, when possible.
    b. If antipsychotics are needed, atypical agents should be used.
    c. For early-phase agitation, propranolol, benzodiazepines, and alpha agonists are often needed.
    d. For reversed sleep cycle, trazodone is the most common medication.
    e. For insomnia due to nightmares and/or PTSD, minipress (Prozosin) is a first-line agent.
    f. Amantadine is a first-line agent to help with early-phase awakening.
    g. For alertness and cognitive deficits, stimulants are first line (after waking phase).
    h. SSRIs and TCAs are useful when anxiety or depressive symptoms are not responding to behavioral strategies.

5. PROGNOSIS

Mild TBIs: Outcomes are favorable. Post-concussive symptoms in mild TBIs (headaches, irritability, concentration, memory deficits, insomnia, dizziness, fatigue) generally resolve within 1-3 months. These symptoms persist in 10-20% of mild TBIs.

Moderate-to-severe TBIs: Most patients do not have full symptom resolution, though the degree of residual impairment varies. The most common post-TBI symptoms after a major TBI are similar to mild TBI post-concussive symptoms, but depression, anxiety, and mood lability are more common.

6. SUMMARY AND TAKEAWAYS

Post-TBI behavioral conditions, particularly mood, anxiety, substance use disorders and personality changes, are 2-3x greater than baseline rates. The behavioral health clinician is charged with the difficult task of making the best assessment possible of whether a symptom is normal adjustment, brain-based, or a psychiatric syndrome.

7. THREE QUESTIONS FOR CLINICAL DISCUSSION

A. How does knowing a patient has a pre-existing TBI impact hospital admission decisions for patients presenting aggression?
B. When would medical or physical rehabilitation admission be a more appropriate placement for a patient with both a TBI and psychiatric needs?
C. If a patient has a TBI secondary to a suicide attempt, should the patient go to a psychiatric unit prior to a medical rehabilitation facility? What factors will help you with this decision?

8. KEY REFERENCES AND RESOURCES FOR FURTHER INFORMATION

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N. Karamouz, J.B. Levine, G.L. Fricchione, in press, Chronic Medical Illness and Rehabilitation. In: T.A. Stern, O. Freudenreich, F.A. G.L. Fricchione, J.F. Rosenbaum (Eds), *Massachusetts General Hospital Handbook of General Hospital Psychiatry, 7th Edition*, Elsevier: Philadelphia, PA

Table 1: TBI Behavioral Syndromes and Associated Psychiatric Syndromes

TBI Behavioral Syndromes and Associated Psychiatric Syndromes			
TBI Syndrome	**Associated Psychiatric Syndrome/Symptoms**	**Location in TBI**	**Differentiating TBI versus Psychiatric Syndrome**
Low motivation	Depressive syndromes	Orbital PFC, bilateral anterior cingulate	Presence of cognitive depressive symptoms in depression versus fatigue, other vegetative symptoms in TBI
Neuro-irritability/ agitation	Mood disorders	Ventral medial PFC	Duration, less sustained in TBI, more easily distracted from emotional distress in TBI
Pseudobulbar syndromes	Anxiety	Cortical control of cranial nerves	Absent or negligible stimulus in TBI
Neglect syndromes (anosognosia)	Denial in conduct and personality disorders, substance use, psychosis, ASD	R parietal	Not resistant to feedback in TBI
Poor planning (apraxia)	ADHD, somatoform d/o's, developmental delay	L medial frontal gyrus to L ventral parietal	Limited planning, intention versus ability to complete task
Aprosadia (flat affect)	ASD, communication d/o, depression	R frontal/temporal lesions	Congruence of verbal and non-verbal communication
Non-fluent aphasia (absent or slow speech, word finding deficits)	Selective mutism, language disorders, dyslexia	Broca's area	Effort to use intact receptive language greater in TBI
Fluent aphasia	Psychosis, mania	R frontal and L parietal circuits involving Wernicke's	Non-language cognitive function can be performed in TBI
Reasoning, concept formation	ADHD, conduct d/o	Dorsal lateral PFC	Sustained attention preserved in TBI
General alertness	ADHD	Superior medial frontal gyrus	Ability to organize and plan in tact
Self and social awareness	ASD	Orbital PFC	Not distractible, can initiate in TBI
Slow processing speed	Learning deficit	Diffuse axonal injury	Degree of change from baseline in TBI
Delusions (anagnosia)	Delusional d/o, psychosis	Posterior, anterior temporal and PFC	Delusional content, hallucination type, minimal thought form change in TBI

Image: Overlap and Distinction Between TBI and Related Psychiatric Syndromes