Traumatic Brain Injury & Concussion

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Overview of Talk

• Defining the mild end of the spectrum (mTBI)
• Examining the controversies & functional consequences associated with mTBI
• Discussing the borderlands between mild and moderate TBI
• Perspectives for Underwriting
Moses’ first and last day as a lifeguard.
Comparison of Annual Incidence

TBI = 1,500,000  Spinal Cord Inj = 11,000  Breast Cancer = 176,300
MS = 10,400  HIV/AIDS = 51,334

Based on data from the Centers for Disease Control and Prevention, the Brain Injury Association of America, American Cancer Society, and the National Multiple Sclerosis Society
General Statistics

• Every 21 seconds a person in the U.S. sustains a TBI

• 5.3 million Americans ( > 2% of population) live with disabilities associated with TBI

• The costs associated with TBI are estimated to be $48.3 billion annually

Incidence & Severity of TBI

- Severe: 10%
- Moderate: 10%
- Mild: 80%
Mechanism of Injury

- M.V.A.
- Falls
- Assault
- Sports
Forms of Acquired Brain Injury

- Focal/Penetrating
- Blunt
- Whiplash
ACRM Definition of Mild Traumatic Brain Injury

A traumatically induced disruption of brain function, as manifested by at least one of the following:

1. Any period of loss of consciousness
2. Any loss of memory for events immediately before or after the accident
3. Any alteration in mental state at the time of the accident (e.g. dazed, disoriented, or confused)
4. Focal neurological deficit(s) that may or may not be transient

However, the severity of injury does not exceed the following:

- PTA no greater than 24 hours
- LOC of approximately 30 minutes or less
- After 30 minutes, an initial GCS of 13-15
ACRM Definition of Mild Traumatic Brain Injury: Important Points

- No Loss of Consciousness Required
- No Blow to the Head Required
- Inclusion of the “Complicated” Mild Traumatic Brain Injury
- May encourage reliance on retrospective description of complaints: “I remember that I felt dazed.”
- Does not focus on heterogeneity within “mild” TBI (GCS = 13-15).
- The meaning of 24 hrs of PTA or 30 hrs of LOC
Concussion: Trauma-induced alteration in mental status that may or may not involve loss of consciousness, with confusion and amnesia identified as the hallmarks of concussion.
**ANA Standards for Gradation of Sports Concussion**

- **Grade I:** Transient confusion, no LOC, concussion symptoms or mental status abnormalities on examination that resolve in less than 15 minutes

- **Grade II:** Transient confusion, no LOC, concussion sx or mental status abnormalities on examination that last more than 15 minutes

- **Grade III:** Characterized by any LOC either brief (seconds) or prolonged (minutes)

Pathophysiology: Primary Injury (immediately upon impact)

- **MACROSCOPIC**
  - contusions at site of impact
  - contrecoup injury - often orbital frontal lobe & tips of temporal lobes
  - laceration of brain from depressed skull fracture

- **MICROSCOPIC**
  - widespread or stretching of nerve fibers (axons)
“Acceleration-Deceleration Injury”

- Head motion is stopped suddenly usually as a result of striking a fixed object
- Brain continues forward due to its own inertia, causing stretching of neuronal axons
- Injury worse with rotational force
3-D MR Surface Image With Superimposed:

**Rotational**

**Linear**

**Tensile/Stretching Effects**

Ziejewski, M. Biomechanics of head injury, head trauma cases: Law and Medicine (2nd ed.). New York (in press)
“Diffuse Axonal Injury”

- Stretching or destruction of nerve cell caused by unequal forces acting on different parts of the brain
- Stress points include interface between gray and white matter, corpus callosum, and brainstem (the brain and cake)
- Microvasculature also prone to injury, so bleeds frequently occur
Secondary Injury & Neurochemical Cascades

- Increases in acetylcholine and excitatory amino acids, such as glutamate causing widespread neuronal depolarization.
- Within two minutes of mTBI there can be a 2-fold increase in extracellular potassium (10-fold in moderate brain injury).
- These changes lead to lactic acid build up within the cells and a state of metabolic depression (i.e. decreased metabolism and cerebral blood flow).
- A metabolic window of vulnerability (30 days)
Microscopic Injury: Axonal Retraction Balls
Diffusion Tensor Imaging
Stevens & Lovejoy, in press
Clinical Caveats with Standard Neuroimaging

- Caution is required with diagnostic neuroimaging. Structural imaging (CT & MRI) has poor sensitivity to axonal injury. Functional imaging is overly sensitive to everything.

- When a Lesion or Damage exists it is usually larger than can be visualized in terms of disruption of cerebral functioning.

- Temporal and frontal areas are most vulnerable to focal damage (learning & memory, executive functions, emotional control).

- Rarely trust the day of injury imaging.
Chronic Post-Injury Effects of TBI Demonstrated in Relation to Structural and Functional Neuroimaging Studies

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Vulnerable Areas

Dorsal View of CT Imaging in TBI

Left temporal & right frontal hemorrhages
Effacement of left ventricle

9 days and continued expansion of ventricles with loss of brain tissue atrophy

Continued expansion of ventricles several months later
Impairments Following MTBI

- Initial Posttraumatic Amnesia
- Neurocognitive Deficits (attention, new learning, & information processing speed)
- Postconcussive Complaints
Memory Impairments from Closed Head Injury Related to Time

- Preinjury Memory Functioning
- Injury
- Unconscious
- Retrograde Amnesia
- Anterograde Memory Impairments
- Post-Traumatic Amnesia
- Recovery
Amnesia: Hollywood Never Forgets
Postconcussive Syndrome (PCS) “Subjective Complaints”

- **Physical** - headache, dizziness, photophobia, phonophobia, tinnitus, insomnia, nausea, fatigue
- **Emotional/Behavioral** - irritability, mood changes (e.g. anxiety, depression)
- **Cognitive** - attention, speed of mental processing, new learning/memory
- Care required due to base rate problems
Research on Athletes vs. MVAs

**CONCUSSION**

Look on the Bright side. For one brief, glorious moment, you forgot you were on the CUBS.
Postconcussive Complaints Over Time in Uncomplicated MTBI Patients

Neurocognitive Deficits in MTBI: SAC Total Scores Across Time

HH Sampele: Mechanism of Injury (N = 220)
Mechanism of Injury (N = 220)

- MVA
- Fall
- MCA
- Struck
- Assault
- Other
Mechanism of Injury (N = 220)
Further Characterizing the Sample

- 71% Male
- 75.9% Caucasian
- 11.8% African American
- 10.9% Hispanic
- 1.4% Asians/others
- Age 34 yrs (18yrs- 83yrs)
- Educ 12.5 yrs (2- 21 yrs)
- GCS 14 (13 - 15)
- PTA 56 minutes
- LOC 5.7 minutes

- 20% positive cranial CT
- 36% substance abuse Hx
- 9% learning disability
- 27% psychiatric Hx
  - depression, anxiety, psychotic spectrum, etc
- 45% orthopedic injury
- 50% past medical Hx
  - diabetes, HTN, concussion, asthma, seizures, cardiac etc.
Postconcussive Complaints Over Time

- Nausea
- Blurred Vision
- Headache
- Dizziness
- Fatigue
- Phonophobia
- Irritability
- Memory
- Concentration
- Anxiety
- Depression
- Judgment
- Sleep

Percentage

- Inpatient
- One month
Long-Term Cognitive Consequences of TBI

- Concussion & TBI as a risk factor for later neurocognitive declines.

- Chronic Traumatic Encephalopathy and/or Alzheimer’s Type Process

Autopsy results: 45-year-old NFL player, multiple concussions
Long-Term Cognitive Consequences of TBI

• Reported odds ratio of 9.9 for head injury with LOC in association with later risk of AD. Odds ratio of 3.3 & 3.1 for head injury w/o LOC.

Reduced Brain Volume Over Time


Expression of Cognitive Decline
Cognitive Reserve
or Cognitive Frailty
From an Underwriting Perspective

- Do you inquire about past history of concussion/TBI at the time of application?
- Where on the spectrum from Mild to Severe?
  - gathering data (E.R., rehab, primary care, neuropsych)
- How remote was the injury?
- Problems with occupational or social adjustment?
- Neuropsychological Evaluation?
- Cognitive Screening?
THEY’RE HAPPY
Because they eat
LARD

issued by the Lard Information Council
Cognitive Underwriting Cases
Case #1

- 66 yo male retired college professor applying for life and LTC insurance

- APS notes:
  - a history of mild depression stable on Paxil 20mg (8 yr hx)
  - Other meds = lisinopril & Norvasc
  - Mild HTN controlled with meds (since age 60)
  - single comment of needing to make lists to remember things to do and feeling more distracted. He attributes this to getting older.
Case # 1 (cont’d)

• He underwent a telephone cognitive screening 3 yrs ago and failed, in the context of reported increased depression following the recent death of his mother.

• He chose to undergo neuropsych testing to improve his insurability.

• The results were as follows:

  • Recent neuropsych eval = Comprehensive evaluation reported largely WNL. However closer inspection of data indicated 30 point diff between verbal and nonverbal IQ, shallow learning curve for visual memory tests with good cued recall, perseverative responding on verbal list learning with adequate recall and improved recall with cueing.
Case #2

- 52 yo male director of operations for a distribution company. Salary $145,000/yr. Applying for life, DI and LTC insurance
- BMI 30
- Fasting glucose in the APS 119, 114. A1c 5.7
- T chol 236, HDL 47, LDL 159, chol/HDL 5.06, TG 150
Case #2 (cont’d)

• APS last note 4/08 - under lots of stress, fatigue. “also thinks he has a little short term memory loss. Cannot be more specific about that and has a lot on his mind” Blood work and CPE ordered. No further information.

• Two years ago had brain MRI for c/o headaches. In office note, nl with only finding of mild small vessel changes. No actual report. Headaches resolved.
Case #2 (cont’d)

• How do you approach this for each line of business?
• What is the risk for DI, LTC, life?
Case #3

• 60 yo male co-owner of an accounting firm. s/p CABG 1999. Hx of self reported memory difficulties. But returned to work post surgery and remains employed in a high level position.

• Had neuropsych testing in 2004 with noted declines in aspects of executive function, verbal fluency, and confrontation naming. Delayed memory scores fall 1SD below expectation and are consistent with rehearsal trial performance. Perseverative responding observed with improved performance on cueing trials. Applicant was diagnosed hypothyroid 1 week prior to testing.

• Repeat testing 2007. Revealed identical neurocognitive pattern.
Case #3 (cont’d)

• Last office visit in 10/08 revealed no change in symptoms and negative ROS.
• Last stress echo 6 months prior to application was normal with nl EF and good exercise tolerance.
• Compliant with medical management of CAD risk.
Case #3 (cont’d)

• What do we know about post pump syndrome?
• What do subtle abnormalities mean when they are stable in a high functioning individual?
Case #4

• 36 yo M high paid executive

• On application
  – numbness in fingers 2007
  – EMG, MRI done. Symptoms resolved

• APS
  – hand sxs c/o finger numbness with pain travelling up the forearm. Dx -cubital tunnel syndrome. Sxs resolved
  – History of 2 concussions
Case #4 (cont’d)

- High school football injury with concussion, neck injury requiring a neck brace for 6 months
- Combat injury with bleed into the brain
  - loss of consciousness for a few hours, post traumatic amnesia
  - Recovered with no clinical symptoms
Case #4 - 2007 Brain MRI findings

• “Asymmetric enlargement of the ventricles likely due to encephalomalacia of the frontal lobes and the periventricular region”
Case #4 - Traumatic brain injury

- Normal loss of brain matter over time
- Loss from injury accelerates normal loss
- What is the DI, life and LTC insurance risk?