Title: Traumatic Brain Injury and Alcohol Use Disorders
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I. Introduction (Slide 2)
   A. Alcohol misuse common in the gen. pop.
      1. 80% lifetime use
      2. 15% lifetime abuse
      3. 10% lifetime dependence
      4. Intoxication, abuse or dependence=alcohol use disorders (AUD’s)
   B. Traumatic brain injury (TBI) each year in US common (Slide 3)
      1. 1.4 million total
         a. 50,000 die
         b. 235,000 hospitalized
         c. 1.1 million to emergency room
      2. Mechanisms of TBI more common with alcohol
         a. Falls-28% (#1 cause death in old)
         b. Motor vehicle accidents (MVA’s)-20% (#1 cause hospitalization)
         c. Struck by/against object-19%
         d. Assaults/violence-11% (#1 cause death in young)
   3. Location/type of TBI (Slide 4)
      a. Anywhere in brain
      b. Visible-brain bruise, tissue deformity
         i. Invisible-axon damage
      c. Frontal lobe damage = “fingerprint” of TBI due to:
i. Acceleration/deceleration of brain

ii. Bony structure of skull

(Slide 5)

4. TBI can cause:  

a. Frontal syndrome (Mnemonic=VALIUM)
   i. ↓ Vision/hearing
   ii. ↓ Attention/concentration
   iii. ↓ Language skills
   iv. ↓ Insight
   v. Unacceptable behaviors
   vi. ↓ Memory
      i. Impulsivity
      ii. Sexual disinhibition
      iii. Violence

b. Physical/neurologic problems
   i. Seizures
   ii. Headaches
   iii. Unsteady gait
   iv. Spasticity
   v. Tremor

c. 40% develop depression

C. Relationship complex (Slide 7)
   1. Alcohol misuse ↑ risk of TBI
   2. TBI ↑ risk of alcohol misuse for some (↓ judgment)
   3. AUD + TBI can ↓ recovery from either

D. This lecture covers: (Slide 8)
   1. Definitions
   2. Epidemiology/outcomes for AUDs before TBI
   3. Epidemiology/outcomes for AUDs post-TBI
   4. Prevention AUD + TBI
   5. Treatment
II. Definitions

A. TBI

1. Defined by level of consciousness at time of TBI, using Glasgow Coma Scale (GCS) (Slide 9)
   a. Standard neurologic assessment
   b. Best response in eye movement + verbal response + motor = total GCS
   c. Lowest score 3 = deep coma or death
   d. Highest score 15 = fully awake, responsive

2. Mild TBI (concussion) (Slide 10)
   a. If available, GCS: 13-15, or
   b. Loss of consciousness (LOC): ≤ 30 minutes, or
   c. Post-traumatic amnesia (PTA): ≤ 24 hours, or
   d. Dazed, confused, “seeing stars”
   e. Commonly called concussion
   f. 85-90% symptom free w/in days-weeks

3. Moderate TBI
   a. GCS: 9-12, or
   b. LOC: 30 minutes-24 hours, or
   c. PTA: > 24 hours < 7 days
   d. Recovery variable; not linearly related to severity

4. Severe TBI
   a. GCS: 3-8 or
   b. LOC: >24 hours or
   c. PTA: > 7 days
   d. Recovery variable; not linearly related to severity

B. DSM IV definitions of AUD’s, etc. (Slide 11)

1. Legal intoxication usually~0.8% blood alcohol level (BAL)
2. Std drink =10-12 gm alcohol= ~ 0.2 gm/dl
   a. 12 oz. beer
   b. 5 oz. wine
c. 1 oz spirits (gin, vodka, whiskey, et al.)

3. Acute intoxication: 1 +; (Slide 12)
   a. Slurred speech
   b. Incoordination
   c. Unsteady gait
   d. Nystagmus (rapid involuntary movement of eyes)
   e. ↓ Attention or memory
   f. Stupor or coma

4. Abuse: 1 + recurrent in same 12 mos.;
   a. ↓ Ability to fulfill role obligations
   b. Use in physically hazardous situations
   c. Legal problems
   d. Social or interpersonal probs.
   e. Never dependent

5. Alcohol dependence: 3 + recurrent in same 12 mos.;
   a. Tolerance (need more for effect)
   b. Withdrawal (rebound signs of intoxication)
   c. Use heavier or longer than intended
   d. Desire and inability to cut down
   e. Activities aborted
   f. Long time spent in alcohol-related activities
   g. Ongoing use despite consequences

(Slide 13)

III. Epidemiology/outcomes AUD before TBI (Slide 14)

A. Epidemiology of intoxication + TBI \(^{11,12}\)

1. 45% of TBI hospitalized legally intoxicated

2. Intoxication < 19yo ↑ risky TBI-related behaviors; \(^{13,14,15}\)
   a. 1.6 X ↑ Drinking + driving
   b. 2 X ↑ Driving after 5 or more drinks
   c. 1.8 X Riding w/ drunk driver
   d. 2.6 X ↑ injuries requiring medical attention
e. 2.5 X ↑ violent behavior
f. ↑ TBI 2° violence

B. Intoxication affects TBI outcome (Slide 15)
   1. Can mask TBI (e.g. intracranial bleed) 2° sim. signs16
      a. Unsteady gait, confusion, belligerence, unresponsiveness etc.
      b. Missed diagnosis of TBI → inappropriate discharge, worse outcome, poss. death
   2. ↑ Severity of TBI 17
      a. ↓ GCS
      b. ↑ Damage on CT scan
   3. ↑ Intensity of tx. 18
      a. 3X ↑ ICU days
      b. 3X ↑ ventilator days
      c. 2.5 X ↑ benzodiazepines
      d. 2 X ↑ opioids
   4. ↓ Scores on cognitive tests 1mo. + after injury 19,20
      a. Verbal IQ ↓ 10%
      b. Processing speed ↓ 10%
   5. 2.5 X risk of repeat trauma next 2 years21

C. Epidemiology abuse/dependence before TBI (Slide 16)
   1. Prior abuse/dependence: 37% TBI (w/o intoxication at TBI)22
   2. Abuse/dependence → risk of TBI in any 12 mos. ↑ 60%23

D. Abuse/dependence before TBI affects TBI outcome
   1. Post-injury unemployment 3X > TBI alone24
   2. Life satisfaction <TBI alone25
   3. ↑ Risk multiple TBI’s26

(Slide 17)

IV. Epidemiology/outcomes of AUD’s post-TBI (Slide 18)

A. Epidemiology
   1. 50% w/ AUD before TBI ↓ alcohol use after TBI27
2. Abstinence rates after TBI ↑ 2X (15% to 30%)
3. ~ 30% of all in AUD tx. have unreported hx. of TBI
4. Sub-group develop/maintain AUD post-TBI (Slide 19)
   a. ~ 25% (10% gen.pop.)
   b. Risk factors:
      i. Pre-TBI AUD
      ii. Major depressive disorder post-TBI
      iii. Less physical disability-independent, can obtain/use alcohol
      iv. Male
      v. Younger age
      vi. Uninsured or on Medicaid
      vii. Unmarried

B. AUD post-TBI affects TBI outcome (Slide 20)
   1. ↓ Neuron reorganization from alcohol ↓ natural healing process
   2. TBI + AUD may ↑ brain atrophy
   3. TBI + AUD death by suicide 4X > TBI alone
   4. TBI +AUD death by suicide 7X > gen. pub.
   5. ↑ Involvement in criminal justice system
   6. Alcohol use ↑ impact of TBI symptoms
      a. Gait/balance problems
      b. Poor judgment
      c. Poor insight
      d. Inappropriateness
      e. Depression

(Slide 21) (Slide 22)

V. Prevention TBI/AUD combo
   A. ID/tx. of AUD ↓ risky behaviors → TBI’s
   B. Successful US public health interventions re MVA (Slide 23)
      1. MVA’s previously #1 cause of TBI, now #2 (falls #1)
2. MADD (Mothers Against Drunk Driving)
   a. Started 1980
   b. Advocates tougher drunk driving laws

   a. ↓ All fatalities 45%
   b. ↓ All injuries 50%
   c. ↓ TBI’s 38%

4. Laws ↓ BAC to 0.08% ↓ alcohol related fatalities and related TBI’s 36%

C. Public Health Challenges (Slide 24)
   1. Mandatory helmet law ↓ TBI 67% for motorcycle riders
   2. Highest rate of drunk driving=motorcycle riders
   3. 2X TBI deaths in states w/o helmet law
   4. <50% of states w/ mandatory helmet law
      (Slide 25)

VI. Treatment Approaches
   A. As TBI have ↑ risk AUD, need search for acute withdrawal
      1. Assessment of acute withdrawal (may be masked by TBI)
         (Slide 26)
         a. Symptoms
            i. Anxiety/Agitation
            ii. Insomnia
            iii. Irritability
         b. Signs
            i. Tremor
            ii. ↑ Reflexes
            iii. Sweating
            iv. Unstable blood pressure
            v. ↑ Pulse
            vi. Disorientation/confusion
         c. Interview for symptoms/amt. of use if possible
i. Life problems re alcohol
ii. Usual and max. drinks past wk.
iii. Time since last drink
iv. Previous withdrawal episodes
d. Physical exam for signs of withdrawal/chronic use
   i. Enlarged liver and spleen
   ii. Cardiac arrhythmia (“holiday heart”)
e. Labs
   i. BAL
   ii. Gamma glutamyl transpeptidase (GGT) >35 units
   iii. Carbohydrate deficient transferring (CDT) 20 u or 2.6%
   iv. LFT’s, CBC, Utox, etc.

2. Treatment of acute withdrawal (Slide 27)
   a. Benzodiazepines (benzos) = gold standard
   i. “Start low, go slow”  
      1’. After TBI, ↑ sensitivity to med effects
         a’. Sedation
         b’. Respiratory depression
      2’. Low initial doses – ½ reg. dose
      3’. ↑ cautiously, based on symptoms + signs
   ii. Longer-acting benzos preferred
      1’. Chlordiazepoxide (Librium)
         a’. Start 25 mg. PO QID
         b’. ↑ or ↓ Based on clin. assess
      2’. Diazepam (Valium)
         a’. 5 mg PO QID
         b’. ↑ or ↓ Based on clin. assess.
   b. Overmedication → respiratory depression or coma
c. Undermedication → agitation, delirium, seizure

B. Early-phase treatment/ prevention of AUD’s (Slide 28)

2. Motivation to change alcohol behaviors↑ after TBI
   a. If dependent pre-TBI,↑ motivation
   b. ↑ # of drinks/week pre-TBI, ↑ motivation
   c. ↑ BAL at injury ≠ ↑ motivation
   d. Motivation highest 1st yr. post-TBI

3. Motivational interviewing (MI) effective at this stage
   a. Non-directive interview
   b. Patient-centered, empathetic
   c. Elicits behavior change
   d. Explores/resolves ambivalence

4. MI + coping skills training > MI alone
   a. Avoid situations/people likely to ↑ drinking
   b. Relaxation-exercise, breathing exercises
   c. Distraction-hobbies, “keeping busy”
   d. Identification/participation in meaningful activity

C. Treatment of chronic AUD + TBI (Slide 29)

2. No evidence-based algorithm for treatment

   a. Cognitive barriers
      i. ↓ Attention, judgment, insight, language skills
      ii. ↓ Short-term memory, etc. ↓ behavior control
   b. Interpersonal barriers 2° ↓ cognition/↑ behavior problems
      i. Frustrate caregivers
      ii. Impair fn. in self-help groups
   c. System barriers
      i. High cost of AUD + TBI care
      ii. Most inpt. AUD programs exclude TBI
      iii. Outpatient tx. may not be enough
4. Modify std. assessment of AUD for TBI (Slide 30)
   a. Routine screen for all TBI patients \(^50\)
      i. AUDIT
         1'. Alcohol Use Disorders Identification Test
         2'. 10 questions, takes little time
      ii. GGT; CDT
   b. Multiple assessments needed \(^51\)
      i. Interview pt. alone re alcohol use
      ii. Review records for evidence AUD, past TBI
      iii. Interview pt.’s family \(^52\)
   c. Accommodate deficits/behaviors in work up
      i. ↑ Time
      ii. Frequent breaks
      iii. Maintain positive interaction

5. Modify std. tx. of AUD for TBI \(^53\) (Slide 31)
   a. External motivators ↑ effectiveness of tx.
      i. Financial incentives, concrete tx. plans \(^54\)
         1'. 50% ↑ in tx. retention
         2'. May ↑ perceived value of tx.
      ii. Intensive case mgmt. \(^55\)
         1'. ↑ access to tx. and rehab.
         2'. ↑ continuity of tx.
         3'. Ongoing support patient/family
      iii. Peer support provides; \(^56\)
         1'. Emotional support
         2'. Knowledge about TBI/resources
         3'. Advocacy skills
         4'. Shared experiences
   b. Modify treatment conditions
      i. ↑ Time
      ii. ↓ noise/visual distractions
iii. Frequent breaks in individual + group settings  
c. ↑ Cognitive retention  
i. Pt. takes notes  
ii. Pt. tapes session and listens again later  
iii. Patient/caregiver repeat main points  
iv. Role-play (coping strategies, relapse triggers)  
d. Content simplification (Slide 32)  
i. Concrete, not abstract, e.g.  
   1’. Pt. uses decision making form (pros v. cons, alternative choices)  
   2’. Break complex tasks into steps  
ii. Behavioral focus (not insight-oriented) e.g.  
   1’. List specific activities to replace drinking  
   2’. Pictorial daily schedule  
   3’. Alarm/watch to initiate these activities  

6. Pharmacology (Slide 33)  
a. Monitor for alcohol and TBI medication interactions  
i. If pt on benzos for spasticity/anxiety, alcohol → sedation, respiratory depression  
ii. If pt on anti-seizure medications: alcohol → ↓ cognitive processing speed, ↓ effectiveness  
   1’. Valproic acid (Depakote)  
   2’. Gabapentin (Neurontin)  
   3’. Carbamezapine (Tegretol)  
iii. If pt on propanolol for tremor: alcohol → sedation, arrhythmia, heart failure  
iv. If pt on Selective Serotonin Reuptake Inhibitors (SSRI’s) for post-TBI depression (fluoxetine/ Prozac, sertraline/Zoloft, etc): alcohol → sedation, ↓ cog processing speed  
v. Many other med/alcohol interactions possible
b. Medications to stop drinking\textsuperscript{59} (Slide 34)
   i. Disulfiram (Antabuse) contraindicated
   ii. Naltrexone (Revia) 50-100 mg/d
      1’. Opioid receptor antagonist
      2’. Reduces cravings, ↑ abstinence once achieved
      3’. No studies for TBI
   iii. Acamprosate (Campral) ~ 2g/d
      1’. ↑ GABA (inhibits), ↓ glutamate (excites)
      2’. Reduces cravings, ↑ abstinence once achieved

c. “Start low, go slow” 2° ↑ medication sensitivity

d. Monitor medication adherence closely

D. This talk has covered (Slide 35)
   1. Definitions
   2. Epidemiology/outcomes for AUDs before TBI
   3. Epidemiology/outcomes for AUDs post-TBI
   4. Prevention AUD+TBI
   5. Treatment
VII. REFERENCES

34. CDC, 2008


