

MEAD TRAUMA

EMERGENEY MEDJEJNE

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Objectives

- □ Current definition and scoring of TBI & CHI
- □ To scan or not to scan MHI patients?
- □ Identify key strategies to optimize care of the TBI/CHI patient in the ED
- □ Describe the concept of neuro-protective agents

Epidemiology

- □ ED visits: 1.1 million/yr
- □ ~50,000 deaths/yr
- □ Penetrating increasing
- □ Blunt decreasing
- □ Highest incidence rate:
 - □ 5 years old
 - □ >85 years old

Classification of Head Injury

- \square Severe: GCS ≤ 8 (~10%)
- □ Moderate: GCS 9-13 (~10%)
 - "Talk and deteriorate"
- □ Minor: GCS 14-15 (75-80%)
 - Period of confusion, disorientation
 - Amnesia
 - Signs of neurologic / neuropsychological dysfunction
 - □ LOC < 30 min

Glasgow Coma Scale

- □ Published in 1974
- Designed to provide a classification for serial neuro exams in ICU patients, comatose for >6 hours
- ☐ Standardize the assessment
- □ Developed prior to CT
- □ Not good for a single assessment

Treatment of Severe Head Injuries

- □ ABCs
- □ Airway: Neuroprotective RSS
 - Lidocaine (1.5mg/kg) IV
 - Fentanyl (3ug/kg) IV
 - Etomidate (0.3mg/kg) TV
 - Sux (1-2mg/kg) SV



Pathophysiology of Brain Injury

- □ Primary Insult:
 - Direct tissue damage at the time of impact
 - Treatment goal: injury prevention
- □ Secondary Insult:
 - Tissue injury occurring after the initial injury due to multiple causes
 - Treatment goal: prevent these types of insults with appropriate treatment

Causes of Secondary Injury

Systemic:

- □ Hypotension
- □ Hypoxia
- □ Anemia
- □ Altered glucose
- □ Hyperthermia
- □ Hyper/hypo-capnia

Intracranial:

- □ IC hypertension
- □ Direct compression
- □ Cerebral edema
- □ Vasospasm
- ☐ Hydrocephalus
- □ Infection
- □ Seizures

What is the Evidence?

- □ Decades of basic & clinical studies on neurotrauma & stroke research
- □ Scarce data showing measurable or consistent benefit for specific therapies
- Many promising therapies have been tested in clinical setting with disappointing results
- □ <u>Overall</u>: no consensus for recommendation of one single specific treatment as a standard
 - Intubate, oxygenate, ventilate, sedate
 - Myperventilation ± Mannitol if herniation

Hypotension & Hypoxia with Head Injury

- Perfusion (oxygenation) to watershed areas decreased with low BP & hypoxia
- □ Hypotension
 - Perfusion of ischemic tissue passively dependent on arterial pressure
 - **□** Goal:
 - Volume resuscitation and expansion (NS better than LR) for SBP >90mmHg
 - Vasopressors
- □ Hypoxia
 - Kills neurons
 - Hyperoxia also shown to ↑ oxidative brain injury in animal models
 - □ Goal:
 - Treat hypoxia aggressively
 - lacksquare 100% oxygen appropriate for resuscitation then FiO2 titrated down after ROSC
 - Avoid anemia (HCT 30%) as it reduces oxygen-carrying capacity
 - Associated with increased mortality

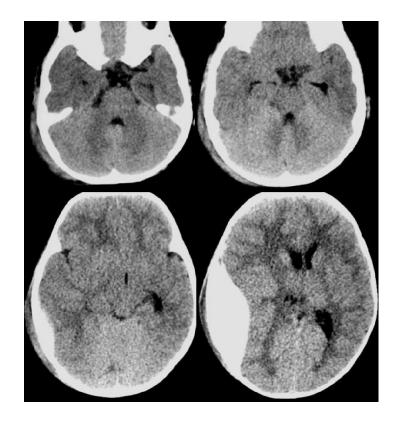
Glycemic Control

- □ Hyperglycemia has detrimental effects on:

 - Metabolism
 - Edema formation
 - Neurologic outcome
- □ Goal:
 - Normalglycemia with insulin
 - Avoid glucose administration (unless documented hypoglycemia)

Cushing's Reflex

- □ ICP puts pressure on brainstem
- □ Triad of:
 - Hypertension
 - 🗖 Bradycardia
 - Respiratory irregularity



Uncal Merniation

Hyperventilation in TBI

- Theory: \(\psi \) ICP via constriction of cerebral vasculature and reduced brain volume
- □ Change: 2-4% ↓ in cerebral blood flow with every 1mmHg change in pCO2
 - Goal pCO2 30-35 mmHg
 - Onset in 30 sec, peaks at 8 min
- Most likely harmful if prolonged, aggressive, or used prophylactically
 - Severe vasoconstriction with pCO2 <25mmHg is harmful (Marion, Crit Care Med, 2002)

Initial Hyperventilation: Recommendations

- □ Cochrane Database: data insufficient to suggest benefit or harm
- □ Brain Trauma Foundation:
 - 1. Prophylactic hyperventilation is to be avoided
 - 2. Hyperventilate for brief periods when there is acute neuro deterioration
 - 3. <u>Hyperventilate for \uparrow ICP</u> that is refractory to sedation, paralysis, CSF drainage, & osmotic divinesis
 - 4. No hyperventilation during initial 24 hours

Osmotic Diuresis: Mannitol

- $\square \downarrow \mathscr{HP} \& \uparrow MAP$
- Mechanism of action:
 - Immediate effect as a plasma expander: ↑ cerebral blood flow & cerebral oxygen delivery
 - 2. Delayed effect (30 min 6 hr): osmotic agent
- No placebo controlled trials
- Adverse effects:
 - Hypotension if volume depleted
 - May stimulate bleeding
 - Renal failure
 - Concentrated in brain tissue with prolonged infusion

Hypertonic Saline (HS)

- □ As prophylaxis or treatment of increased ICF inconclusive
- □ Hypertonic saline (up to 7.5% NaCl):
 - Mypothesis: cerebral swelling may be prevented by altering the osmolar load
 - Keep serum Na < 155mmol/L</p>
 - Also good for fluid resuscitation: Sub-analysis of RCTs suggest that patients who are hypotensive with TBI and given HS have improved outcome
 - Wade, J Trauma 1997 (2x as likely to survive in HS group)
 - Vassar, J Trauma 1993

Sedation, Positioning & Paralysis

- □ Comatose brain responds to external stimuli by increasing cerebral metabolism
- □ Protection from stimuli may prevent imbalance between oxygen supply and demand
- □ Goal:
 - Restrict activities that cause ↑ ICP
 - Titrate sedative or anaesthetics
 - □ Elevate head of bed 30°
 - Not proven to be beneficial
 - Paralysis as needed to control ICP

Corticosteroids

- Steroids shown to be beneficial in patients with cerebral edema and brain tumors
- □ Trials in patients with TBI have not shown benefit
- ☐ Recommendations:
 - Cochrane Database: no benefit shown in 19 trials
 - CRASH Trial (Roberts, Lancet 2004):
 - Increased risk of death with steroids not recommended
 - Brain Trauma Foundation:
 - Not recommended in TBI

Hypothermia & TBI

- □ No evidenced-based support for improved mortality or morbidity with prophylactic hypothermia in moderate or severe TBI
- □ Meta-analysis by Brain Trauma Foundation
 - Hypothermia >48 hrs was associated with ↓ mortality
 - Limited results due to small sample size



Coagulopathy & TBI

Coagulopathy may be prior to or a result of the TBS

- 1) Patient on Warfarin
- 2) TBS activates the clotting system
- □ Close monitoring of coagulation parameters
 - Coagulopathy at time of ED arrival is bad
 - 50% vs 17% mortality (Wafaisade, Neurocrit Care 2010)
- \square More severe the TBI, the higher the risk of DIC (may occur rapidly)
- □ Some authors recommend FFP in those with a GSC 6 (May, Am Surg 1997) or <8 (Talving, J Trauma 2009)

Warfarin and Head Trauma

- $\square \uparrow \mathbf{r}$ ish of SCH in patients with minor head trauma
- \square \uparrow **r**ish of worse outcomes once TBI occurs
- □ Aggressive ED treatment warranted
- □ Early CT scanning
- □ Treatment of coagulopathy if bleed on CT
- □ Careful discharge instructions if negative CT scan

Holmes, "Beyond GCS," ACEP 2011

Post-traumatic Seizures

- □ Early post-traumatic seizures (7 days of injury):
 - Occurs in 4-25% of those with TBI
- □ Late post-traumatic seizures (>7 days of injury):
 - Occurs in 9-42% of those with TBI
- □ Risk factors for post-traumatic seizures:
 - Seizure at time of injury or ED presentation
 - □ GCS (10 (severe HS))
 - Cortical contusion
 - Subdural/epidural/intracerebral hematoma
 - Depressed skull fracture
 - Penetrating injury
 - Prior seizure history

Anticonvulsants: Recommendations

- □ Evidence:
 - Carbamazepine vs placebo: Glotzner, Neurochiurgia Stuttg, 1983
 - Dilantin vs. placebo: Temkin, NEJM, 1990
 - Both studies showed reduction in early seizures but no difference in late seizures
- □ Recommendations:
 - Cochrane: 10 RCTs
 - NNT to prevent 1 seizure = 10
 - No reduction in mortality
 - No evidence that late seizures are reduced
 - Brain Trauma Foundation
 - 1. Dilantin and CMFP prevent early post-traumatic seizures and are recommended
 - 2. Anticonvulsants not recommended for preventing late seizures

Antibiotic Prophylaxis?



- □ Abx prophylaxis for patients w/ CSF leah?
- □ Most CSF leaks resolve spontaneously in 1 week
 - Meta-analysis: Abx generally not offered prophylactically in 1st week of CSF rhinorrhea from basilar shull # (Clin Infect Dis 1998)
 - World J Surg 2001: pts. w/ post-traumatic CSF leakage had 2x the incidence of meningitis w/o prophylaxis
- □ Abx prophylaxis indicated in penetrating head injury, open shull #s, and complicated scalp lacerations

Moderate MS

□ Management principles same as for severe HI



Minor M.J.: To Scan or Not to Scan?

Cost of a CT Scan? (CAD)



Indications for CI's in MMI Patients

New Orleans Criteria

- □ Haydel et al. (NSJM 2000)
 - 7 criteria: H/A, vomiting,
 age >60, drug/etoh intoxication,
 short-term memory deficit, trauma
 above the clavicles, post-traumatic
 seizure



Canadian CT Head Rule

Stiell et al. (JAMA 2005)

- Migh rish criteria (for neurological intervention)
- Medium risk criteria (for brain injury on CT)



Canadian ET Mead Rule





100% Sensitive 70% Specific 32% would need CT

98% Sensitive 50% Specific 54% would need CT

External Validation of EEMR & NOC in MHI

Smits et al. JAMA 2005

- □ Both NOC & CEHR are highly SN (100% for both) for neurosurgical intervention
- □ However, CCHR has lower SN than NOC (83% vs 99%) for neurocranial traumatic or clinically important CT findings
- □ The CHR was more specific (40% vs 3%) and efficient
- □ Widespread implementation of CHR would dramatically ↓ use of CT & result in cost effectiveness

Concussion (complicated minor TBF)

- □ Most common type of head injury
- Caused by acceleration-deceleration or rotational injury to a freely mobile head
- □ Concussive signs & symptoms:
 - SOC, traumatic amnesia, headache, nausea, blurry vision, vertigo, sleep disturbance, emotional lability, and difficulty concentrating
 - May last weeks to months
- <u>Ireatment</u>: depends on severity but centers on avoiding further contact during at risk periods

Questions?

