OPTIMIZING CARE AFTER SEVERE TBI: REHABILITATION 101

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CASE PRESENTATION: ICU CARE

J.K. is a 19 yo male s/p GSW to head with initial GCS 8. Evaluation on day 2 reveals elevated ICP's (>30 mmHg) and localizing on left.

You are asked about:

- Seizure Prophylaxis
- DVT Prophylaxis
- GI Ulcer Prophylaxis
- Therapy Interventions

ACUTE MANAGEMENT OF SEVERE TBI: POST-TRAUMATIC SEIZURES

POST-TRAUMATIC SEIZURES: BACKGROUND

- TBI-related seizures account for 20% of symptomatic epilepsy.

  Hauser: Epilepsia 1991:32;429-45

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- PTS accounts for 5% of all cases of epilepsy.

  Hauser: Epilepsia 1991:32;429-45

- Late PTS is present in 4-7% all TBI, ~20% rehab TBI, and 35-50% penetrating TBI patients.

  Yablon: Arch PM&R 1993:74;983-1001

POST-TRAUMATIC SEIZURES: BACKGROUND

- PTS results in higher acute and long-term costs after TBI.

- PTS results in poorer short-term (physical and cognitive function) and long-term (vocational) outcomes after TBI.

Schwab: Neurology 1993:43;95-103

Dikmen: Epilepsia 1978:19;177-83

Armstrong: Arch PM&R 1990:71;156-60

PTS: RISK FACTORS

- Chronic EtOH
- Family History
- Age
- Penetrating Injury
- Length of Unconsciousness
- Severity of Injury
- Length of PTA

Annegers: N Engl J Med 1998:338;20-4

Evans: Neurology 1961:12;665-74

- Intracranial Hemorrhage
- Cerebral Contusions
- Focal Neuro Deficits
- Depressed Skull Fxs
- Retain Bone or Metal Fragments
- Lesion Location

Yablon: Arch PM&R 1993:74;983-1001

Caveness: Epilepsia 1961:2;123-9

PTS RISK FACTORS: EEG

- EEG findings do not show a direct association with increased risk of early or late PTS.

Heikinnen: Stereotact Funct Neurosurg 1990:54-5;25-33

Courjon: Epilepsia 1970:11;29-36

Paillas: Epilepsia 1970:11;5-16

Jennett: Epilepsia 1975:16;251-6

PTS: EARLY VS. LATE

- "Immediate" PTS = < 24 hours post-TBI
- "Early" PTS = ≤ 7 days post-TBI
- "Late" PTS = > 7 days post-TBI

PTS: EARLY VS. LATE

- Early PTS is associated with increased risk late PTS.

  Annegers: N Engl J Med 1998:338;20-4

  Jennett: Scot Med J 1973:18;8-13

- 50-66% of individuals with late PTS will experience first seizure w/in 12 months.

- 75% of individuals with late PTS will experience first seizure w/in 24 months.

  Walker: J Neurosurg 1959:16;600-10

  Salazar: Neurology 1985:35;1406-14

PTS PROPHYLAXIS

- 73% reduction in early PTS and 50% reduction in 1 year PTS in individuals given phenytoin for 1 week post-TBI.

- No proven benefits to giving prophylaxis >7 days post-TBI.
  Temkin:N Engl J Med 1990:323;497-502

- No benefit to use of up to 1 month VPA.
  Temkin: J NeuroSurg 1999:91;593-600

- AANS and AAPM&R recommend 7 days of either PTH or CBZ post-TBI.

MODEL SYSTEMS: PTS RESEARCH

- NIDILIR TBI Model Systems project includes12,000+ individuals with TBI from 16+ sites across the U.S. followed longitudinally from acute care since 1988.

- For those individuals with late (1+ week) PTS
  - 40% late PTS occurred at 7 days - 1 month
  - 63% late PTS occurred 7 days - 6 months
  - 80% late PTS occurred 7 days - 12 months
  - 93% late PTS occurred 7 days to 18 months
  - 7% occurred after 18 months

ACUTE MANAGEMENT OF SEVERE TBI: DEEP VENOUS THROMBOSIS

DVT PROPHYLAXIS

- All adult trauma patients admitted for >24 hours have at least a "Moderate" risk of DVT and require chemical prophylaxis.

- Patients with ≤4 risk factors for DVT require SQ Heparin 5000 bid and SCD's.

- Patients with >4 risk factors for DVT require LMWH (e.g., Lovenox 30 units SQ bid) or Coumadin (INR 2-3) prophylaxis.

Clagett: Chest 1998;114(5):531-560

DVT PROPHYLAXIS

- DVT prophylaxis should continue until:
  - patient is ambulating to/from BR prn
  - an appropriate time has passed (SCI 8-12 weeks, hip/femur fx 5 weeks)
  - patient is returning home and continuing is not feasible.

- IVC or SVC filters are not appropriate DVT prophylaxis for any patient.

- One-third of patients with DVT will develop post-Phlebitic Syndrome.

Clagett: Chest 1998;114(5):531-560

DVT PROPHYLAXIS

- SQ Heparin can be begun immediately on any patient, except w/ a history of Heparin-Induced Thrombocytopenia
- LMWH can be begun as soon as 36 hours after intracranial or spinal trauma.
- ASA, low-dose Coumadin, and non-thigh high SCD's reduce risk, but not to an optimal level and therefore should not be used as primary prophylaxis.

Clagett: Chest 1998;114(5):531-560

DVT MANAGEMENT

- All DVT's (UE, Calf, w/IVC filter, etc.) should be treated for 3 months.
- All PE's should be treated for 6 months.
- Heparinization (LMWH or IV) should be given at least 5 days, check platelets on Day 3, then q3-7 days while on it.
- Coumadin should be delayed until 24 hours of heparinization is completed.

Clagett: Chest 1998;114(5):531-560

ACUTE MANAGEMENT OF SEVERE TBI: GI ULCER PROPHYLAXIS

GI ULCER PROPHYLAXIS

- The use of H2-Blockers has been demonstrated to decrease ICU-related stress ulceration of the GI tract in specific patient populations (e.g., burns).
- No specific information in patients with TBI, with or w/o PEG/J tubes.
- Newer H2-Blockers, while expensive, have limited CNS effects.
- High risk patients (h/o PUD, h/o GERD, comatose, > 65 years old) are appropriate for prophylaxis while in ICU.
- No clear indication for all TBI patients in ICU.

ACUTE MANAGEMENT OF SEVERE TBI: REHABILITATION THERAPY

THERAPY INTERVENTION

- In the face of elevated ICP's (>10 mmHg), therapy must be limited to:
  - Appropriate positioning of limbs and head
  - Use of non-customized orthotics, as needed
  - Repositioning (turning) every 4 hours
  - Limiting inappropriate environmental stimulation.

- Elevated BP (>180/100) would require similar restrictions.

CASE PRESENTATION: ACUTE CARE

- The patient undergoes extensive interventions, including craniectomy, and his ICP's decreases to <10 mmHg. He remains minimally responsive and intermittently localizing on the left.

- You are asked to provide input into:
  - Therapy interventions
  - Prediction of outcome
  - Low serum sodium
  - HO prophylaxis

ACUTE MANAGEMENT OF SEVERE TBI: REHABILITATION THERAPY

THERAPY INTERVENTIONS

- Protective helmet required until appropriate for cranioplasty (4-12 weeks).

- Full mobilization recommended, monitoring BP, ICP (if possible) and pulse oxygenation, including;
  - Long and short sitting in bed
  - OOB to Chair
  - Sit to Stand and transfers
  - Limb AAROM
  - Sensory stimulation

- Use of oral stimuli may be appropriate if the patient is controlling his secretions well.

THERAPY INTERVENTIONS

- If persistent cognitive depression is anticipated for >2-3 weeks, a PEG/J tube should be recommended.
- Partner with ICU nursing for carryover.
- Family involvement.

ACUTE MANAGEMENT OF SEVERE TBI: PROGNOSTICATE OUTCOME

WHY PREDICT OUTCOME?

- Inpatient Rehabilitation is expensive (U.S. $500-1500/day); patients receiving it should have a realistic chance of returning home.
- More therapy is not better, it's only more. The right mix of medical, nursing, and therapy services is important.
- Choosing the most appropriate setting for each patient requires a "best guess" of outcome.
- The patient, family, and rehabilitation team needs an estimate of LOS and D/C goals.

COMMONLY USED OUTCOME MEASURES

- Mortality
- Glasgow Outcome Scale, modified
- Disability Rating Scale
- Functional Independence Measure
- Return to Work

DOMAINS OF PREDICTION

- Demographics
- Injury Characteristics
- Clinical Features
- Radiologic and Electrophysiologic Data
- Rehabilitation Interventions
- Surgical Interventions
- Pharmacologic Interventions

OUTCOME PREDICTORS: DEMOGRAPHICS

- Age is a key predictor of recovery. Age > 40, 50, and 60 has been associated with poorer outcome.
- Alcohol use pre- and peri-injury has been associated with poor cognitive outcome.
- Race, Gender and Socioeconomic Status have not been well-studied.

OUTCOME PREDICTORS: INJURY CHARACTERISTICS

- Etiology of Injury has not been demonstrated as a predictor of outcome.

- Injury severity predicts 3, 6, and 12 month outcome (function, work)
  - Glasgow Coma Scale (3-15)
    - Very Severe = 3-5
    - Severe = 6-8
    - Moderate = 9-12
    - Mild = 13-15

OUTCOME PREDICTORS: INJURY CHARACTERISTICS

- Injury severity predicts 3, 6, and 12 month outcome (function, work)
  - Brainstem Reflexes
  - Glasgow-Liege Scale (GCS + BSR)

- Intracranial Pressure
  - >20 (sustained) mmHg
  - >40 mmHg

OUTCOME PREDICTORS: INJURY CHARACTERISTICS

- Length of Coma (GCS < 8)
  - <1 week better than >4 weeks
  - Proportionate relationship
- Length of Post-Traumatic Amnesia
  - <70 on the GOAT
  - Proportionate relationship with key periods being <1 week and <6 weeks

OUTCOME PREDICTORS: CLINICAL FEATURES

- Individuals who have a Disorder of Consciousness (DOC) at 1 month
  - 3 months = 33% awake
  - 6 months = 46% awake
  - 1 year = 52% awake

- Individuals with DOC who emerge in the first year post-TBI
  - 28% severely disabled
  - 17% moderately disabled
  - 65%good recovery

OUTCOME PREDICTORS: CLINICAL FEATURES

- Muscle Tone: Flaccidity correlates with poorer outcome.
- Respiratory failure correlates with poorer outcome.
- Motor strength, DTR's, sensation, balance, coordination deficits have not been well studied.
- DRS at admission correlates with D/C status and 1 year return to work.

OUTCOME PREDICTORS: RADIOLOGY

- Intracranial masses correlate with poorer outcome.
- Intracerebral contusions correlate with poorer outcome.
- Epidural hematomas have a better prognosis than subdural hematoma (probable age bias).
- Midline shift > 10 mm correlates with poorer outcome.
- Brainstem lesions correlate with poorer outcome.

OUTCOME PREDICTORS: ELECTROPHYSIOLOGY

- Abnormal EEG worse prognosis than normal EEG.
- Combining EEG, SSEP, BAER, and VEP correlates with outcome

OUTCOME PREDICTORS: REHABILITATION

- Directed Multisensory Stimulation (DMS) demonstrated superior (increased responsiveness, improved RLAS, improved GCS) versus Non-Directed Stimulation (NDS) in RLAS II patients

  Hall: *Brain Injury* 1992:6:435-45

- Comatose and acute TBI patients receiving greater therapy intensity (by 60%) demonstrated a 31% decrease in length of stay. Blackerby:*Brain Injury* 1989;4:167-73

OUTCOME PREDICTORS: REHABILITATION

- Formal TBI Rehabilitation results in an increased rate of return to the community, decreased utilization of medical services, and decreased disability.

  Cope:*Brain Injury* 1995;9:649-70

  Bell:*Arch Phys Med Rehabil* 1998;79S:21-5

- Acute rehabilitation utilizing a dedicated TBI program resulted in decreased LOS, improved cognitive skills, and improved return to home rates.

  Mackay:*Arch Phys Med Rehab* 1992;73:635-41

OUTCOME PREDICTORS: REHABILITATION

- Interdisciplinary Team versus Multi-disciplinary Team demonstrated improved functional outcome, maintenance of gains, and reduced caregiver stresses.

  Semlyen:*Arch Phys Med Rehabil* 1998;79:678-83

- TBI patients >3 months post-injury demonstrated improvement in behavior, physical ability, functional skills, and independent living. Maintained improve-ments 18 months post-completion.

  Malec:*Brain Injury* 1993;7:15-29

  Mills:*Brain Injury* 1992;6:219-28

OUTCOME PREDICTORS: REHABILITATION

- Acute TBI patients stratified into high versus low intensity therapy groups demonstrated improved RLAS levels and cognitive skills at discharge.

  Spivack:*Brain Injury* 1992;6:419-34

- Multiple regression analysis revealed that intensity of PT, OT, and SLP services did not affect outcome, but greater Psychology services intensity resulted in improved cognitive skills at discharge.

  Heinemann:*Am J Phys Med Rehabil* 1995;74:315-26

OUTCOME PREDICTORS: REHABILITATION

- Multiple regression analysis revealed that intensity of PT and OT services did not affect outcome, but greater Psychology services intensity resulted in improved cognition and greater SLP services intensity resulted in improved cognitive and physical skills at discharge.

Cifu:*Arch Phys Med Rehabil* 1997;78:1029

ACUTE MANAGEMENT OF SEVERE TBI: NEUROENDOCRINE DYSFUNCTION

NEUROENDOCRINE MANAGEMENT

- Diabetes Insipidus (DI)
  - Not common in TBI.
  - Hypernatremia is usually due to dehydration.
  - Intranasal DDAVP is easy treatment (1-2 squirts daily).

- Syndrome of Inappropriate ADH secretion (SIADH)
  - Common in TBI.
  - Hyponatremia in the elderly can also be due to dehydration.
  - Bactrim and Tegretol also cause SIADH.

NEUROENDOCRINE MANAGEMENT

- SIADH
  - Fluid restriction (1.0 - 1.5 Liters) for 3-5 days "cures" most SIADH.
  - NaCl dietary supplements may help.
  - The use of "hot salts" (3% Saline IV) outside of ICU is not appropriate – consult Endocrinologist.

- Acute issues with growth and sex hormone abnormalities are unusual, but may be issues in the outpatient setting.
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- Other neuroendocrine abnormalities after trauma are rare -> consult Endocrinologist

ACUTE MANAGEMENT OF SEVERE TBI: HETEROTOPIC OSSIFICATION

HETEROTOPIC OSSIFICATION

- Diagnosis
  - High index of suspicion
  - Typically 4-12 weeks after trauma
  - Clinical
    - Decreased and/or painful ROM
    - Swelling
    - Erythema
    - Warmth
    - Low-grade fever
- Imaging
  - Triple-phase bone scan detects findings acutely
  - Plain X-rays detect findings 2-4 weeks after onset

HETEROTOPIC OSSIFICATION

- Treatment
  - Prevention and/or early detection are critical
  - Radiation – 400-600 Gray
  - Pharmocologic
    - NSAID's
    - Etidronate
  - Rehabilitation
    - Controlled, frequent PROM – PAINFUL!

CASE PRESENTATION: INITIAL REHABILITATION CARE

- J.K. improves to a "high functioning" RLAS III and is transferred to inpatient rehabilitation.

- You are asked to provide input on the following issues:
  - Increased limb and truncal tone
  - Increased pain with questionable fracture
  - Poor participation in therapy due to hypoarousal and inattention

REHABILITATION MANAGEMENT OF SEVERE TBI: SPASTICITY

SPASTICITY MANAGEMENT

- Spasticity = Velocity dependent increase in resistance to stretch.
- Tone = Resistance to stretch or movement.
- Both are commonly seen signs of an upper motor neuron abnormality, that typically resolve as the lesion evolves.
- Neither is inherently “bad” for the patient. Can be injury evolution or sign of secondary issue.

SPASTICITY MANAGEMENT

- Etiology of spasticity is not well understood:
  - imbalance of excitatory (Ach, NE) and inhibitory (GABA) neurotransmitters
  - excessive firing of muscle spindle fibers due to inappropriate neuronal feedback
  - peripheral sensations may be misinterpreted centrally
  - central motor impulses may be inappropriate

- While typically due to the intrinsic injury, be aware of;
  - Hydrocephalus
  - Occult fracture
  - Heterotopic Ossification
  - Infection
  - Bowel or Bladder obstruction

SPASTICITY MANAGEMENT

- Treatment should be initiated if the spasticity is limiting function, ROM, or is causing pain.
- Potential side effects of treatment must be weighed against potential benefits.
- Treatment should be used at dose/intensity sufficient to either normalize tone or allow for good positioning.

SPASTICITY MANAGEMENT: FIRST LINE

- Decreasing noxious stimuli (infection, skin irritation, edema, DVT, distended hollow organ).
- Appropriate positioning (normalizes spindle function) and stretching (spindle).
- Controlled ROM (normalizes spindle function).
- Repetitive tactile stimulation – Vibration, Massage, Tapping, Stroking, Electrical (TENS/FES)
- Thermal stimulation (central and peripheral targets)
  - Heat (superficial stimulation-> central)
  - Cold (superficial stimulation-> central and local decrease in neuromuscular junction function)

SPASTICITY MANAGEMENT: SECOND LINE

- Inhibitive splinting and casting - tactile stimulation (heat, pressure) and constant stretch (spindle)

- Dynamic splinting and serial splinting add a more aggressive stretching component
  - cost of dynamic splinting is prohibitive
  - skin breakdown is common

- Deep heating modalities (ultrasound) should be used with dynamic splinting
  - Allows for increased extensibility of connective tissue
  - Accelerates improvement of contractures and shortened tissue

SPASTICITY MANAGEMENT: THIRD LINE

- Systemic medications are effective, but often have systemic side effects:
  - Hepatotoxicity (Baclofen, Dantrium)
  - Generalized weakness (Dantrium)
  - Lethargy (Zanaflex, Baclofen, Valium)
  - Hypotension (Zanaflex)
  - Addiction (Valium)

- Preferred choice if multiple limbs are involved.

- May be used in concert with focal blockade.

SPASTICITY MANAGEMENT: FOURTH LINE

- Focal blockade is preferred when spasticity is limited to single limb or few muscles:
  - Direct blockade of nervous innervation at either peripheral nerve or neuromuscular junction.
  - No systemic side effects.
  - Long-lasting blockade.
- May be used in concert with systemic agents.
- Multiple choices of agents with varying benefits

SPASTICITY MANAGEMENT: FOURTH LINE

- Local Anesthetics (Lidocaine, Marcaine)
  - Temporarily (2-12 hours) blocks conduction of nerve signals. Works immediately.
  - Eliminates spasticity in specific nerve distribution or muscle.
  - Nerve/muscle motor point (where nerve innervates) must be isolated electrically.
  - Diagnostic rather than therapeutic.
  - Inexpensive.

SPASTICITY MANAGEMENT: FOURTH LINE

- Phenol (1-10% Aqueous Solution)
  - Direct neurocidal agent, effect lasts for 3-6 months (until nerve regenerates). Works immediately.
  - Eliminates spasticity in specific nerve distribution or muscle.
  - Nerve/muscle motor point (where nerve innervates) must be isolated electrically.
  - Inexpensive.

SPASTICITY MANAGEMENT: FOURTH LINE

- Botulinum Toxin
  - Neurotoxin that prevents the release of acetylcholine (Ach) from presynaptic vacuoles at the neuromuscular junction.
  - Produces paralysis of the muscle for 2-4 months.
  - Maximal effects take 2 weeks.
  - Expensive.
  - Small risk for antibody production with repeated usage

SPASTICITY MANAGEMENT: FIFTH LINE

- Neurotomy/Cordotomy - permanently "disconnect" peripheral and central nervous system.
- Muscle or tendon splitting/lengthening procedure - weakens spastic muscles.
- Muscular reattachment - realigns muscle contraction vectors to "normalize" function.
- Expensive and irreversible.

REHABILITATION MANAGEMENT OF SEVERE TBI: PAIN

PAIN MANAGEMENT

- In cognitively impaired patient with likely source of pain (fracture, burn, contracture) consider scheduled medication for therapy and/or mobilization
  - Scheduled Tylenol (± brief use of NSAID's) is highly effective.
  - Rib Fractures are always painful and in the elderly are often severely debilitating.
  - Pain is a common cause of TBI agitation.

- If no obvious source, consider plain film X-ray, triple phase bone scan and/or duplex doppler ultrasonography.

REHABILITATION MANAGEMENT OF SEVERE TBI: DOC AND COGNITIVE DYSFUNCTION

DOC INTERVENTION

- While research supports the role of rehabilitation care and medications (Amantadine) to reduce the duration of DOC, the results are small.

- Optimizing medical and nursing care while establishing a family/nursing-based program of cognitive and physical interventions is goal with RLAS I-III.

- Individuals who are demonstrating RLAS III attributes (and better) may be appropriate for a trial of increased rehabilitation intensity.

COGNITIVE INTERVENTIONS: HYPOAROUSAL

- No reliable data to support the efficacy of pharmacologic intervention in the vegetative or comatose patient.
- All you get is a very "alert"-looking comatose patient.
- Small trials do support use of neurostimulants in "emerging" patients (RLAS III) -> Amantadine 300 mg/day.

COGNITIVE INTERVENTIONS: HYPOATTENTION

- Neurostimulants have been demonstrated to improve attention and functional improvements after TBI, particularly in individuals who are RLAS V-VII.

- Methylphenidate has the most clinically demonstrated efficacy for individuals who have progressed to RLAS IV and above. Other stimulants have similar effect.

- Methylphenidate 5-20 mg q 7am and 12 pm.

COGNITIVE INTERVENTIONS: HYPOATTENTION AND HYPOAROUSAL

- Medications
  - Dopaminergic Agents
    - Amantadine
    - Bromocriptine
    - Levodopa/Carbidopa (Sinemet)
    - Pergolide
    - Selegiline
  - Stimulants
    - Methylphenidate
    - Dextro-amphetamine
    - Pemoline

CASE PRESENTATION: ONGOING REHABILITATION

- J.K. demonstrates increased alertness and cognitive abilities, but begins having difficulty with restless behavior.

- You are asked to provide input on the following issues:
  - Agitation
  - Depression
  - Hydrocephalus

REHABILITATION MANAGEMENT OF SEVERE TBI: AGITATION

BEHAVIORAL INTERVENTIONS: AGITATION

- Agitation occurs in >50% of all TBI patients as part of the normal recovery, however other causes should be considered.
  - Delirium
  - Seizures,
  - Pain
  - Hypoxia
  - Hydrocephalus

- Agitation should be measured with the Agitated Behavior Scale and treated with environmental and behavioral interventions.

- Pharmacologic treatment should only be implemented in specific behaviors are identified and goals established.

BEHAVIORAL INTERVENTIONS: AGITATION

Assessment

- Agitated Behavior Scale
  - Assess pattern of agitation
  - Documentation
  - Evaluate effectiveness of intervention
- Assess for correctable etiology
  - Sleep/Wake Charting
  - Medical Management
  - Environment triggers
  - Engagement issues

Treatment

- Behavioral for ABS>21
  - Establish desired behavior
  - Positive reinforcement
  - Shaping
  - Structured therapy
- Physical Controls
- Pharmacologic

BEHAVIORAL INTERVENTIONS: AGITATION

- Pharmacologic interventions – for ABS >35
  - Beta-Blockers are most appropriate for "Aggression"
    - Scheduled q8-12 hours
    - Begin at 10 mg q12 and increase by 30mg daily
    - Hypotension usually a non-issues >180mg/day
  - AED's (VPA, CBZ) are most appropriate for "Restlessness"
    - Schedule q8-12
    - Use standard AED levels to monitor
  - TCA's are most appropriate for "Emotional"
    - Use standard antidepressant dosing
  - Prn = Ativan or Risperidone

REHABILITATION MANAGEMENT OF SEVERE TBI: DEPRESSION

COGNITIVE INTERVENTIONS: DEPRESSION

- Depression occurs in up to 50% acute trauma patients.
- Vegetative symptoms are often hard to distinguish.
- Psychiatrists rarely have greater insight than treating MD's.
- No validated diagnostic tool.
- Trial of Methylphenidate or SSRI may be best diagnostic tool – see effect in 72 hours. If clinical depression suspected, 6 months treatment indicated.

REHABILITATION MANAGEMENT OF SEVERE TBI: HYDROCEPHALUS

HYDROCEPHALUS

- Most commonly seen with significant blood load, penetrating injury, CSF infection, older age, and prior history.
- Maintain high index of suspicion since a plateau of progress may be the only sign.
- Seizures, hypertension and agitation may occur.
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- Head CT scan 1 month post-injury in all patients with severe TBI and then serially as warranted clinically.
- If clinically suspected and CT non-diagnostic, consider
  - CSF tap or flow study
  - ventriculostomy or V-P shunt

QUESTIONS?

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