Traumatic Brain Injury: A Review

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Abstract

This article reviews issues related to traumatic brain injuries.

EPIDEMIOLOGY

- Approximately 1.6 million head injuries occur every year in the U.S.
- Nearly 60,000 deaths from TBI yearly
- Accounts for ~50% of deaths from trauma
- Financial burden: $100 billion annually
- Mortality: 25-35%
- Primary contributing factors: MVA, Falls, Alcohol

PATHOPHYSIOLOGY:

PRIMARY BRAIN INJURY:

Focal: cerebral contusions, hematomas, fractures
Diffuse axonal injury:shearing/inertial forces

SECONDARY BRAIN INJURY:

Due to neuronal damage from systemic physiologic tesponses to
the initial injury
Hypotension, hypoxia

CEREBROVASCULAR REGULATION:

MONROE-KELLIE DOCTRINE:

The cranial vault is a fixed space consisting of 3 compartments:
Parenchyma (80%)
CSF (10%)
Blood (10%)

Therefore, expansion of one compartment results in a compensatory decrease in another in order to maintain ICP

Cerebral Blood Flow is determined by:

CPP
PaO2
PaCO2
Cerebral metabolic demand

Normal CBF: 50 ml/min/100g of brain tissue or 60-160 mm Hg)

Autoregulation: CBF is maintained over a wide range of CPP; between 50-150 mm Hg, by autoregulation of cerebral vasculature, but becomes deranged with brain injury (CBF becomes linearly dependent on MAP)

Effects of BP, PaO2, and PaCO2 on CBF:

Decreased paO2/CaO2: ↓ CBF
Cerebral vasodilation
↑ cerebral blood volume
↑ICP

Increased paCO2: ↑ CBF
Cerebral vasodilation

CPP = MAP - ICP
Normal: 70-100 mm Hg
Used to assess the adequacy of cerebral perfusion because CBF is
Difficult to measure clinically

Evolution of CBF after TBI:
Initial hypoperfusion phase
Hyperemia (increased CBF)
Second hypoperfusion phase (cerebral vasospasm)
Recovery phase (CBF ~ CMRO2)
Begins 2-3 weeks after onset
Continues for weeks, months

ICP: Reflects the volume of: Brain parenchyma
CSF

Intravascular blood

Normal: < 15 mm Hg

CBV + CSF + Parenchyma

Ischemia Hydrocephalus Edema
Acidosis comm.vs.noncom. Ischemia
 paCO2` (cytotoxic)
Hyperthermia Vasogenic
 venous P (impaired BBB)

CLINICAL SIGNS OF ELEVATED ICP:

- Depressed level of consciousness
- Hypertension
- +/- Bradycardia
- Irregular respiratory pattern
- Headache
- Nausea/Vomiting
- Papilledema
- Third or Sixth CN palsies

INITIAL MANAGEMENT AND RESUSCITATION:

Primary survey (ABCDE's)

Monitor hemodynamic and oxygenation status
Avoid or immediately correct: Hypotension (SBP < 90 mm Hg)
Hypoxia (paO2 < 60, O2 Saturation < 90%)
Maintain MAP > 90 mm Hg to allow CPP > 70 mm Hg

Secondary survey (AMPLE hx)

Neurological reevaluation: GCS (pre- & postresuscitation scores important)

EYE OPENING

- Spontaneous 4
- Voice 3
- Pain 2
- None 1

BEST VERBAL RESPONSE

- Oriented 5
- Confused 4
- Inappr. Words 3
- Incompr. Sounds 2
- None 1

BEST MOTOR RESPONSE

- Obeys commands 6
- Localizes pain 5
- Withdraws to pain 4
- Flexion 3
- Extension 2
- None 1

- GCS Severity of TBI
- 13 -15 Mild
- 9 - 12 Moderate
- 3 -8 Severe

- Pupillary light reaction
- Oculoocephalics (Doll's eyes)
- Oculovestibulars (Caloric)

MANAGEMENT OF TBI:

General Principles: CT of head & Neurosurgical evaluation

1. Patient positioning: HOB > 30 degrees
Cervical collar
Head midline
2. Fluid management: NS, LR
3. Fever management
4. Stress ulcer prophylaxis
5. DVT prophylaxis
6. Monitor electrolytes (Na, Mg)

MANAGEMENT OF INTRACRANIAL HYPERTENSION:

Airway Management:

Intubation-Indications: Respiratory distress Motor posturing/Absence of response to pain

Hypoxia/Hypercapnia
GCS < 8
Seizures
Increased ICP
Need for analgesics/sedatives
Significant associated injuries

Rapid sequence intubation recommended:
Preparation
Preoxygenation
Pretreatment (LOAD)
Paralysis AFTER induction
Protection + Positioning
Placement with proof
Post Intubation management

TREATMENT OF ELEVATED ICP:

REDUCTION OF CEREBRAL BLOOD VOLUME:

Avoid hypoxia or hypercarbia
Hyperventilation - no longer used in the modern era

In the absence of increased ICP, chronic prolonged Hyperventilation therapy (paCO2 < 25) should be avoided after severe TBI.

GUIDELINE:

The use of prophylactic hyperventilation (PaCO2 <35) therapy during the first 24 hours after severe TBI should be avoided due to concerns of compromising cerebral perfusion at a time when CBF is reduced.

OPTIONS:

Hyperventilation therapy may be necessary for brief periods when there is an acute neurological deterioration or for longer periods if there is ICH refractory to sedation, paralysis, CSF drainage, & osmotic diuretics. Jugular venous oxygen saturation, arterial-jugular venous O2 content differences & CBF monitoring may help identify cerebral ischemia if hyperventilation resulting in a PaCO2 < 30 is necessary.

Promote venous drainage (elevate HOB)
Treat severe hypertension
Medications

BARBITURATE COMA:

Guideline: High dose barbiturate therapy may be considered in HD stable salvageable SHI patients with ICH refractory to maximal medical and surgical interventions

Pentobarbital: Eisenberg RCT:
Loading dose: 10 mg/kg over 30 minutes
5 mg/kg q 1 hour x 3
Maintenance: 1 mg/kg/hr

Moniter EEG for 30-60s. burst suppression pattern

REDUCTION OF CSF VOLUME:

Decrease CSF production (lasix, acetazolamide)

Ventricular drainage/ICP monitoring

Indications: SHI (GCS <8 after resuscitation) + abnormal CT
SHI with a normal CT if >2 present at admission:
Age > 40
Unilateral/bilateral motor posturing
SBP <90 mm Hg

Not routinely indicated in patients with mild or moderate head injury
Physicians may choose to monitor ICP in conscious patients with traumatic mass lesions

Initiate ICP therapy at an upper threshold of 20-25 mm Hg
Published clinical experience indicates that ICP monitoring:

1. Aids in the earlier detection of intracranial mass lesions
2. Can limit the indiscriminate use of therapies to control ICP which themselves can be potentially harmful
3. Can reduce ICP by CSF drainage
4. Helps in determining prognosis
5. May improve outcome

REDUCTION OF BRAIN TISSUE VOLUME:

Administer Lasix (0.5 - 1 mg/kg IV)

Osmotic therapy:

Guidelines:
Mannitol is effective for control of elevated ICP after SHI
Effective doses range from 0.25 - 1 g/kg
(Effect begins within 10-20'; peaks @ 20-60'; duration 4-6 h)
(Monitor Sna, Sosm; maintain Sosm <320)

(Maintain euvolemia by adequate fluid replacement)

Options:
Indications prior to ICP monitoring:
Signs of transtentorial herniation
Progressive neurological deterioration not attributable to extracranial pathology
Intermittent boluses may be more effective than continuous infusion

Surgical Decompression: Decompressive hemicraniectomy

PROPHYLACTIC ANTICONVULSANTS:

Standard: The prophylactic use of Phenytoin, Carbamazepime, or
Phenobarbital is NOT recommended for prevention of late post-traumatic seizures (> 7 days)
Options: Anticonvulsants may be used to prevent early (<7 days)
PTS in patients at high risk for seizures following head injury
Risk factors include: GCS score <10
Cortical contusion
Depressed skull fracture
SDH, Epidural hematoma
Intracerebral hematoma
Penetrating head wound
Seizure within 24 hours of injury
Phenytoin and Carbamezipime have been shown to be effective.
However, available evidence does not indicate that prevention of early PTS improves outcome following head injury.

MEDICAL MANAGEMENT:

Nutritional support

Patients with SHI: Hypermetabolic, hypercatabolic, hyperglycemic
Altered immune function (cellular immunity)
Altered gastric motility (ICP, Dilantin, NMB)

Early (within first 24h) administration of enteral nutrition blunts the hypermetabolic response after injury in critically ill patients

Initial goal: 25-30 kcal/kg/day
1.5-2.0 g protein/kg/day

Replace 140% of RME in nonparalyzed patients, & 100% of RME in paralyzed patients
Provide at least 15% of calories as protein by the 7th post-traumatic day.

Algorithm:

Figure 1

Change to intact protein/high nitrogen formula when TF tolerated for 3 days

MEDICAL COMPLICATIONS:

CV: MI
Pulmonary: PNA, Aspiration (peaks 5-10d post-trauma)
ARDS
Endocrine/Metabolic: SIADH, Cerebral salt wasting
DI
Skin: Pressure ulcers
Electrolyte disturbances
Hypotension
PNA* Independent predictors of increased
Sepsis* morbidity/mortality
Coagulopathy: Peaks during the first 3 days
Due to: Blood transfusion/resuscitation
Brain tissue injury/destruction
Medications
(Sepsis)
Associated with the systemic release of parenchymal tissue thromboplastin, etc.
DIC ~ degree of brain injury
Principles of therapy:
Routinely monitor platelets, coags

Maintain platelets > 100,000
Treat the underlying pathology

PROGNOSIS AND OUTCOME:

Extent of recovery is dependent on:
Patient's age
Severity of injury
Type of intracranial disorder
Recovery is often slow
Significant disability is common
Rehabilitation

References

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