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Traumatic Brain Injury

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1. Introduction

Almost 1.7 million Americans sustain traumatic brain injury (TBI) annually according to the center for disease control,275,000 get hospitalized while 52,000 die of traumatic brain injury. 1.365 million are treated and released from the Emergency Department . About 80 to 90,000 sustain long term disability. The rate of death from TBI remains low, 6/100,000, but the cost to society remains high. Survivors suffer from long term sequelae such as seizures, loss of memory and cognitive impairment. According to one estimate, half of the hospitalized patients end up requiring neuro-rehabilitation. Falls are the leading cause of TBI in children and adults over the age of 65. TBI in patients under the age of 19 have increased in the United States over the last decade (Morbidity and Mortality Weekly Report (MMWR) Nonfatal Traumatic Brain Injuries Related to Sports and Recreation Activities Among Persons Aged ≤19 Years --- United States, 2001--2009 October 7, 2011 / 60(39);1337-1342). MVA s are the major cause of TBI and TBI related death in young adults.

Types of Injury:

- Scalp laceration
- Skull fractures
- Epidural hematoma
- Subdural hematoma
- Intra-cranial hemorrhage

2. Scalp Injuries and skull fractures

Scalp lacerations tend to bleed profusely and while bleeding can easily be controlled with surgical staples, they may be associated with penetrating injury to the brain parenchyma. Motor vehicle crashes (MVCs) and blast injuries are also associated with fractures of the skull. The parietal bone is mostly commonly fractured and is associated with epidural hematomas due to tearing of the middle meningeal artery. Linear skull fractures are associated with delayed epidural hematomas and may be cause of sudden neurological worsening. Depressed skull fractures are associated with high kinetic energy resulting in CSF leaks presenting as otorrhoea or rhinorrhea (shattered cribriform plate.). Immunofixation with beta-2 transferrin can help differentiate CSF from other body fluids. Depressed skull fractures and fractures involving the sinuses have a high incidence of

infection. It is therefore prudent to start the patient on antibiotic prophylaxis. Both, Clindamycin 600 mg IV Q6hr and Vancomycin 1G Q12 is also acceptable. Radiographs have no role in the diagnosis of skull fractures due to their low sensitivity and specifity in defining underlying brain damage. Head CT with bone windows should be done in the emergency department. The Brain Trauma Foundation recommends all fractures depressed greater than the thickness of the cranium be surgically elevated.

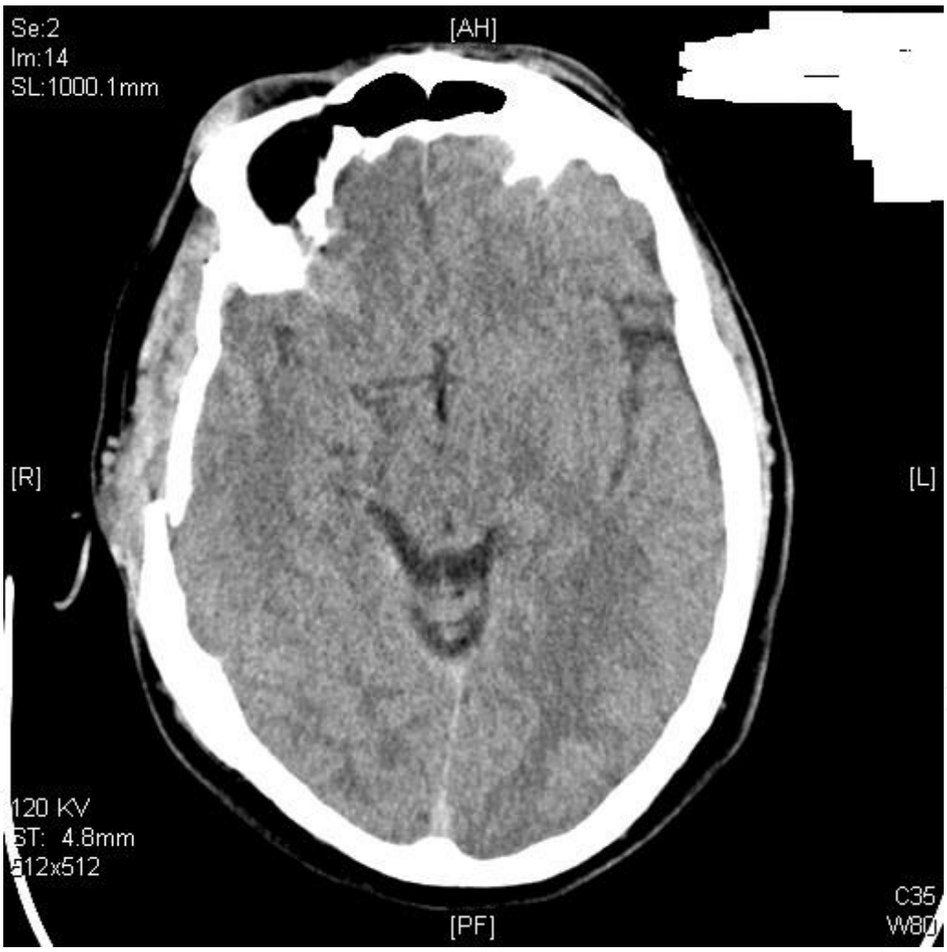

Fig. 1. Depressed Right temporal Bone Fracture requiring surgical elevation. Image coutesy: Dr. Azedine Medhkour, MD, Neurosurgery, University of Toledo Medical Center.

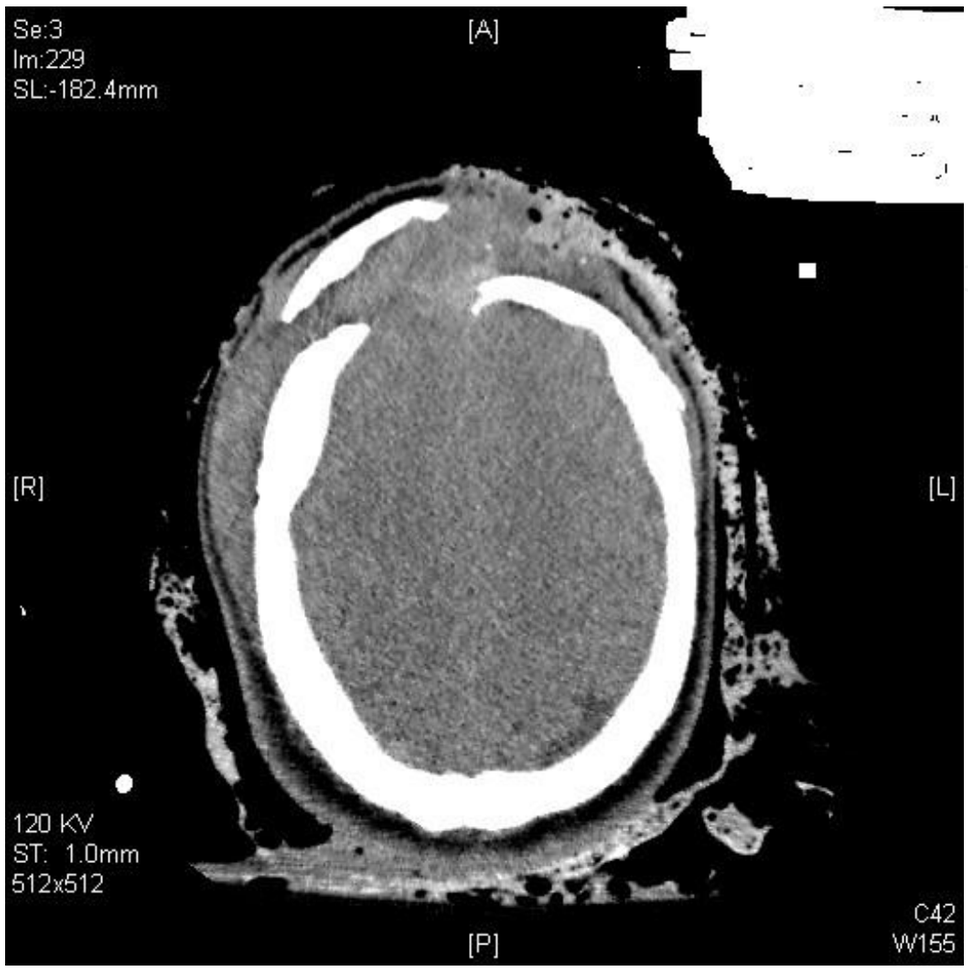

Fig. 2. Anterior displacement of comminuted frontal bone fracture. Image coutesy: Dr. Azedine Medhkour, MD, Neurosurgery, University of Toledo Medical Center.

3. Intra-cranial hemorrage

3.1 Epidural hematoma

Epidural hematoma forms secondary to trauma to the skull and underlying vessels e.g. torn meningeal artery. 9 to 10% present as a delayed hematoma. Anisocoria (papillary difference or >2 mm) is present in 67% of the patients.

3.2 Subdural hematoma

Subdural hematoma is seen in 0.5 to 5% of head injuries and is usually associated with traffic trauma and falls in the elderly especially those on anticoagulants. All patients reporting LoC should be evaluated for a syncopal episode prior to fall or MVA. The evaluation should include 2D trans-thoracic echo for undiagnosed aortic and mitral valve stenosis, EKG and cardiac enzymes to rule out myocardial infarction as well as a carotid ultrasound to detect plaques and/or critical narrowing. 24 hr. Holter monitoring and tilt table may also be necessary as arrhythmias and postural hypotention could be possible

causes of loss of consciousness. Patients over the age of 65 are frequently on anti-hypertensive medications and diuretics.

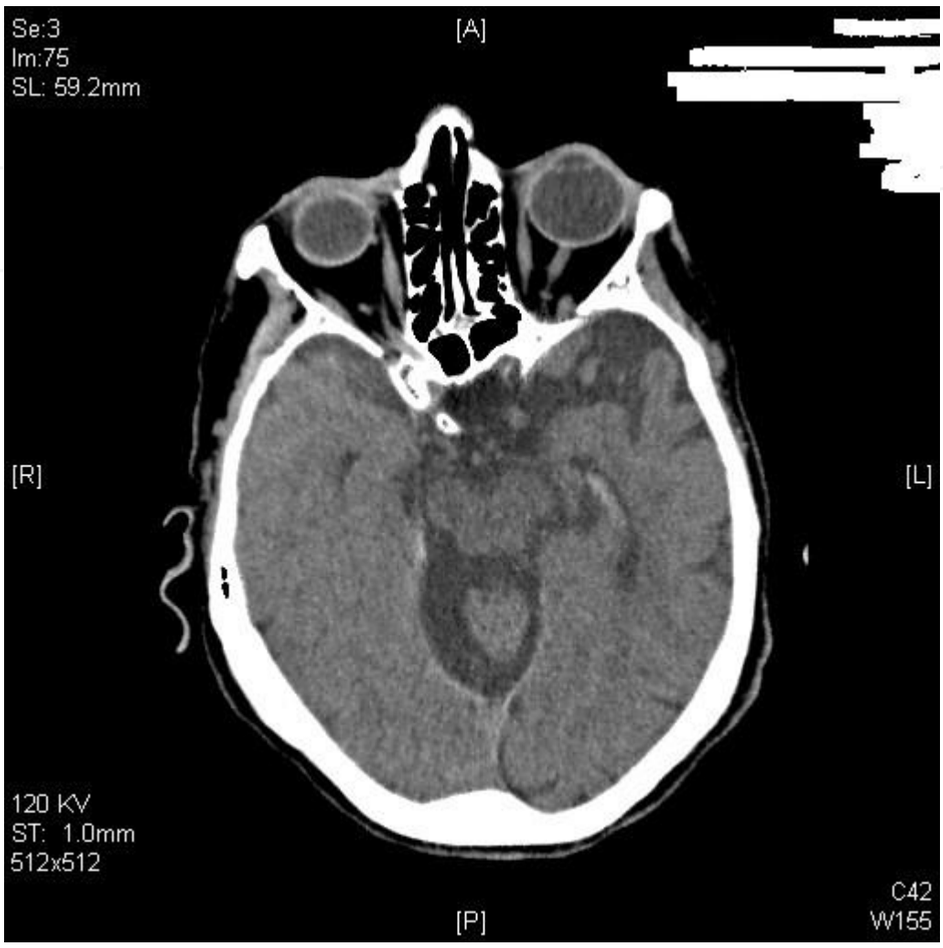

Fig. 3. Subarachanoid hemorrhage in anterior brainstem. Image coutesy: Dr. Azedine Medhkour, MD, Neurosurgery, University of Toledo Medical Center.

4. Intra-parenchymal hemorrhage

In 4 to 23% of TBI the brain is injured as it accelerates along the irregularities of the inner table of the skull.

- *50% pts are unconscious at presentation
- *2/3 rd require Surgery in the 1st 48 hrs.
- *70% of delayed ICH occurs in the 1st 48 hrs.

4.1 Contusion

Contusions are the most commonly identified injuries on CT and are due to sudden deceleration injury with brain impacting on the inner table of the frontal or middle fossa . Contusions may also appear distant from the area of impact (counter-coup). The resulting multi-polar areas of injury, ischemia and edema lead to rapid rise of ICP and chances of

herniation. Patients usually have focal neurologic deficits with contusions but sudden deterioration may be a sign of hemorrhage into the contusion. A repeat CT may be indicated under the circumstances.

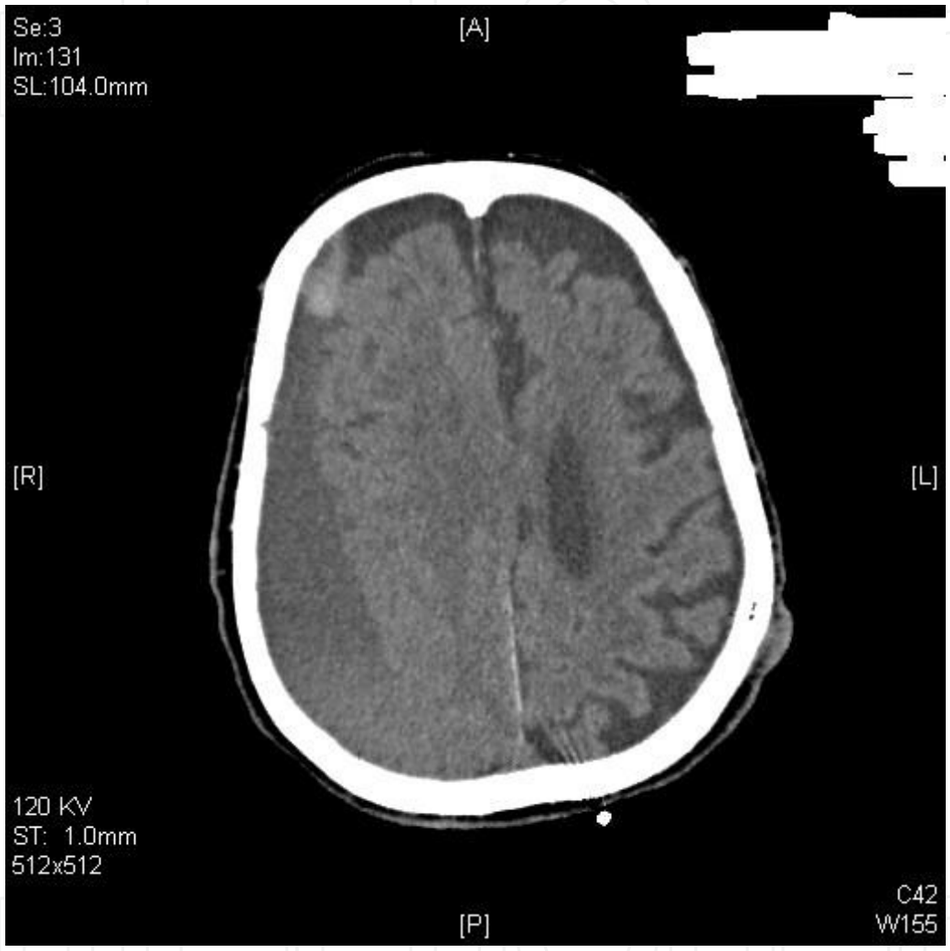

Fig. 4. Acute on chronic, mutipolar subdural hemorrhage with midline shift. Image coutesy: Dr. Azedine Medhkour, MD, Neurosurgery, University of Toledo Medical Center.

4.2 Concussion / diffuse axonal injury

Concussion is the temporary loss of consciousness and is on the same continuum as diffuse axonal injury (DAI) . DAI refers to disruption of intra-cerebral axons at the grey/ white matter junction due to angular forces. DAI carries the worst prognosis of all TBIs. The extent of damage may not be visible on the first CT scan. Unconsciousness lasting over 6 hours without sedation should raise suspicion for DAI and may warrant a gradient echo MRI for definitive diagnosis.

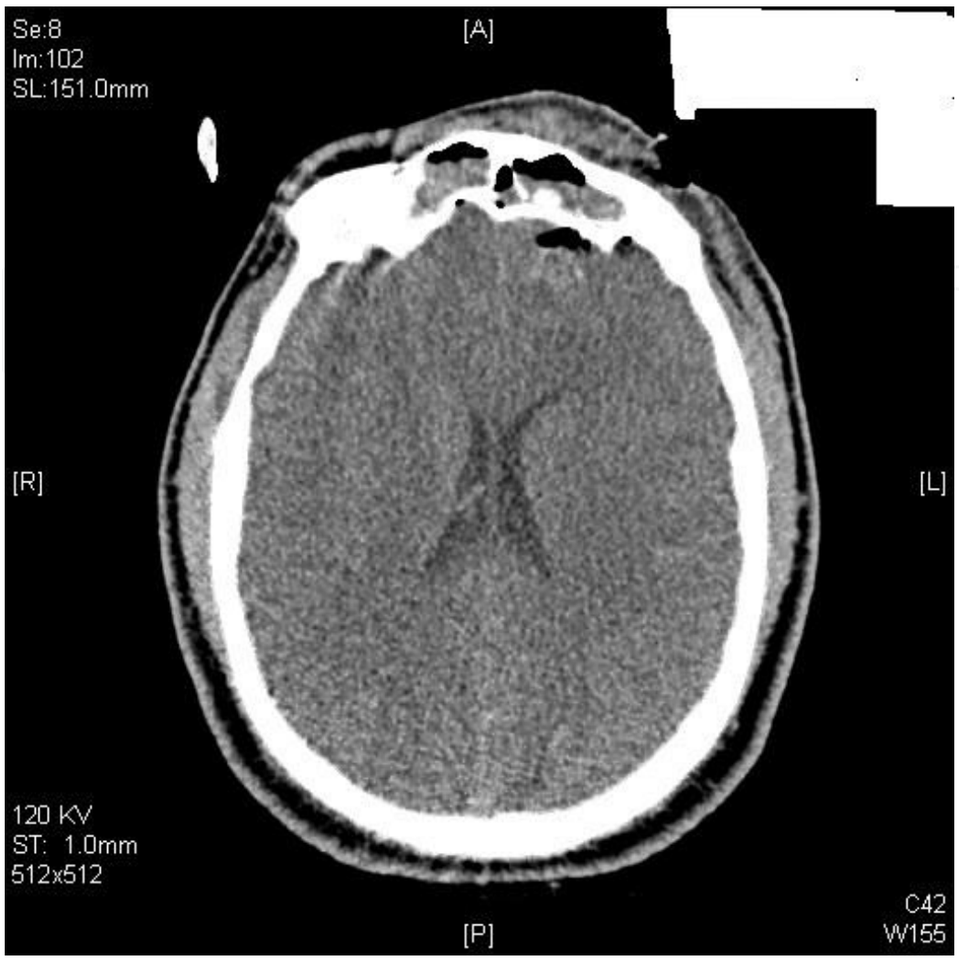

Fig. 5. Severe DAI with loss of grey and white matter differentiation. Image coutesy: Dr. Azedine Medhkour, MD, Neurosurgery, University of Toledo Medical Center.

5. Early evaluation and treatment

The Glasgow Coma Scale (GCS) remains the cornerstone of early assessment by clinicians in the ED . But in the pre-hospital setting simple observations such as combativeness, alcohol intoxication, high speed MVA, loss of short term memory ,vomiting, age over 60 and trauma above the clavicles are signs that underlying brain injury is likely and early interventions such as airway protection may be necessary. All patients with suspected head injury should be immobilized due to high incidence of co-existing spine injuries. Evaluation of ABCs i.e. Airway , breathing and circulation should be the first priority Tracheal intubation for GCS less than 6 should be performed, when possible prior to transport. All patients should be resuscitated with isotonic saline. (0.9% NaCl) Hypotonic and dextrose containing fluids should be avoided as they can worsen brain edema. Small volume resuscitation with hypertonic saline in patients with poly-trauma involving the head is also acceptable. 3% saline (8ml/kg) was associated with similar hemodynamic benefits when compared with high volume isotonic resuscitation i.e. Lactated ringer at 33 ml/kg. in experimental models of head injury. Contrary to popular belief LR infusion is not associated with Calcium influx

or progression of neuronal injury. However recipients of HS had higher serum Na and lower ICPs.

6. Principles of management

6.1 Prevent secondary injury

The initial insult to the brain at the time of primary impact is only a portion of the damage sustained in TBI. Secondary insults compound the initial injury. Secondary injury is caused by hypotension and hypoxia. Early hypotension, especially in the field, strongly correlates with poor outcome and mortality. The prognosis of TBI is primarily dependant on the ability to escape secondary injury. Threshold values for intervention are:

- S_aO_2 <90,
- ICP >20,
- CPP <60,
- SBP <90.
- Hypo and Hypercarbia i.e. <28 or>45.

Hypoxia occurs in 22.4 % of severe TBI patients and 55% of patients are hypoxic on tracheal intubation. Hypoxia for 10 to 20 min i.e. $S_aO_2$less than 90% is an independent predictor of poor outcome.

Goals of Management	
O2 sat	>97%
SBP	>90mmHg
CPP	>60mmHg
Blood Glucose	100-150mg/dl
ICP	<20
Temp.	<37.5

Table 1.

6.2 Sedation

Light sedation may be indicated for anxious patients. Sedation decreases $CMRO_2$ while agitation and combativeness can raise ICP considerably. Midazolam, lorazepam and propofol can be used for this purpose.

7. Elevated ICP

7.1 Head position

Keeping the head of bed elevated to 30 degrees and in a midline position may prevent kinking of internal jugular veins and obstruction of venous outflow. Patients with spine injuries and log roll precautions should be kept in reverse Trendelenberg to aid venous drainage.

7.2 Mannitol therapy

Mannitol is an osmotic diuretic used to reduce intracranial pressure and brain edema. It expands plasma volume by creating an osmotic gradient and improves cerebral blood flow. However, it should only be used with caution patients with congestive heart disease (CHF) or renal disease. Mannitol is typically given as a bolus 0.25grams to 1.5 grams per kilogram IV in hemo-dynamically stable with no active source of intracranial bleeding. It has consistently been shown in studies to reduce ICP and can help temporize the patient's condition while definitive therapy is determined. At present there is no evidence to support its use as a continuous infusion. An ICP monitor may be placed before a second bolus is administered to determine the effect on ICP. Serum osmolality should be maintained between 300 to 320 Osm/L. Higher values are associated with acute kidney injury.

7.3 Hypertonic saline

3 % saline can be used to decrease cerebral edema and decrease ICP. HS can be started at 20 to 50 cc/hr. with serial monitoring of serum sodium every 4 hours. Rate of rise of serum sodium should not be greater 0.5meq/l /hr or 2 meq/l/ 2 hrs. The maximum allowable rise is 10 meq/24 hrs. Central pontine myelinolyis (locked-in-syndrome) can occur in patients with pre-existing hyponatremia. HS can also be given as a 250 cc bolus. Effects of HS are mediated not only by its ability to expand plasma blood volume but also by its ability to attenuate the immunolgic response by preventing neutrophil activation and improving cerebral blood flow.

7.4 ICP Monitoring

ICP monitoring is indicated in all salvageable patients with: severe TBI i.e. GCS 3-8 after resuscitation; patients with CT scans showing hematoma, contusion, swelling or herniation; and in all patients with normal CT but 2 or more of the following features: age>40,unitaleral or bilateral posturing or SBP< 90. All these are at significant risk for intra-cranial hypertension. CT scans cannot reliably predict ICH. The incidence of intracranial hypertension can be greater than 60 % in comatose patients with GCS<9. ICP monitors are usually intra-parenchymal with a fiber-optic transducer at the tip. Epidural and sub-arachanoid bolts are also inserted and offer a high degree of accuracy.Intra-ventricular devices offer the additional advantage of draining CSF with rapid reduction of ICP in patients who cannot tolerate mannitol due to hemodynamic instability. ICP monitoring also allows the clinician to identify patients with intra-cranial hypertension that is refractory to medical management who require decompressive craniotomy.

8. Seizure prophylaxis

Reduced CBF (<20cc/ml/100g/min) or loss of consciousness stimulates in a threshold pattern the release of excitatory neuro-transmitters. The CSF concentrations of glutamate, glycine and aspartate increase up to 8 fold. These levels remain elevated up to a week after the initial inciting factor. These increased levels have been described as the mechanism for post-traumatic seizures. Seizures dramatically increase $CMRO_2$ and are associated with a worse prognosis. Current practice is to use levectiectetam 500 mg q12 hours for a week. Phenytoin, phosphenytoin and valproic acid can also be used. Unlike levectiectetam,

phenytoin requires frequent lab checks to monitor therapeutic levels and is associated with adverse effects such as enzyme induction, myocardial depression, and fever. In one study almost half of the patients with post-injury seizures had therapeutic blood levels of phenytoin. Valproic acid has been associated with a higher incidence of death than the other antiepileptic medications.

8.1 Reversal of anti-coagulation

Elderly patients are frequently on anti-thrombotics for a variety of conditions such as DVT, chronic atrial fibrillation, ischemic stroke, coronary artery disease, or stent placements. Urgent reversal of anti-coagulation is necessary in this patient population. Rapid enlargement of an intracranial hematoma can lead to midline shift, mass effect, herniation and deterioration of neurological status. The incidence of re-bleeding is also very high . 10-15ml/kg of FFP should be given for urgent reversal of warfarin. Vitamin K should be added unless the reversal is meant to be transient. Administration of prothrombin complex concentrate (Profilnine) contains factors II,VII, IX and X and leads to normalization of INR within 10 min and has a half life of 6 to 8 hours. PCC has the advantage of a predictable response, rapid correction of INR and low volume which makes it ideal in patients with CHF. A dose of 25 U/Kg should be given for INR <4.0 and 50U/Kg for INR >4.0. Platelet transfusions may be required for patients on aspirin or clopidogrel. Recombinant activated factor VII was originally developed for hemophiliacs but its use had recently been extended to coagulopathic trauma patients. Factor VII at a dose of 90 mcg/kg leads to a more rapid reversal of coagulopathy and reduces the time to neurosurgical intervention. But, its use is limited by cost and thrombo-embolic complications especially in patients with coronary stents.

Apixaban and Dabigatran, are two of the new drugs being prescribed both for DVT and stroke prophylaxis to patients with history of pulmonary embolism, mechanical valve and chronic Atrial fibrillation. The former is a Factor Xa inhibitor and the reversal would be the same as heparin and lovenox i.e. FFP and RBC transfusions. However, the latter is a direct thrombin inhibitor, no specific antidotes exist at this time. This drug was approved by FDA in Oct. 2011. We don't have enough experience with it yet. We do know that prothrombin complex does not reverse it well. And that Factor II inhibition is one of its mechanisms of action which makes Factor VII a potential reversal agent. But no definitive recommendations can be made at this time.

8.2 Normothermia

Mild hypothermia is beneficial. Fever increases $CMRO_2$ so the patient's temperature should always be maintained at less than 37.5°C. While blood in the subarachanoid space and infection are the two leading causes of fever, diencephalic seizures can manifest themselves as a paroxysmal increase in temperature, heart rate , respiratory rate, and rigors . They can occur in the absence of any blood in the brain. Paroxysmal autonomic instability with dystonia or PAID is another term coined to describe the episodic nature of this syndrome.

8.3 Vasospasm prophylaxis

Vasospasm occurs in 20 to 35% of severe TBI and is especially common in blast injuries and are a cause of neurologic decline. Transcranial Doppler studies can be performed at the

bedside where there is a high index of suspicion. Peak velocities greater than 200 cm/sec or less than 120 cm/sec are reasonably accurate in including or excluding vasospasm. However computed tomography angiography (CTA) should be used to confirm the diagnosis for values lying in the intermediate range. Nicardipine infusions can be used in patients with an established diagnosis of vasospasm. Magnesium infusions can be used to keep serum Mg above 3.5 for patients at high risk for vasospasm to reduce the incidence.

8.4 Steroids

Steroids have no role in the management of traumatic brain injury. They are not associated with decreased ICP or improved outcome. Methylpredisolone was found to be associated with worsened outcome in the CRASH(Corticosteroid randomization After Significant Head Injury) Trial in 2004.

8.5 Beta-blockade

Survivors of traumatic brain injury are subjected to catecholamine surge manifested by hyperthermia, tachycardia, tachypnea, arrhythmias and eventually cardiac necrosis. All of which may contribute to secondary insults. There is some evidence to suggest immunosuppression as a cause of catecholamine excess. Beta blockers offer survival benefit by suppressing the systemic effects of catecholamines.

9. Neurologic recovery

Amantidine is increasingly being used in TBI patients who develop diffuse axonal injury. It has shown to improve neurologic status by increasing dopamine levels in the brain. It can also be used in patients with frontal lobe lesions who are difficult to arouse. Atypical anti-psychotics like Aripiprazole (Abilify) is increasingly being used for improve cognitive function in TBI patients. Incidentally, Patients whoever were on Statins pre-injury were found to have better outcomes in a retrospective study.

9.1 Intravenous estrogens

Recent interest in IV estrogen use for TBI stems from the clinical observation that women in the reproductive age group generally do better than male cohorts after TBI. Also, levels of CSF estradiol were considerably higher in the male survivors compared to non-survivors. Estrogens are believed to induce the synthesis of heat shock proteins, reduce levels of cytokines and are anti-apoptotic. Animal studies have shown up to a 60 % reduction in the area of ischemic injury when IV estrogens were administered within 30 min. post-injury. Similar benefits were observed in spinal cord injury. The dose currently recommended for resuscitative purposes is a one time dose of 0.5mg/kg. The National Institute of Health Resuscitation Consortium is now considering implementation of a multi-centered trial after positive results from their pilot study.

10. Conclusion

Traumatic brain injury is one of the leading causes of death and disability in adults. Care of the TBI patients in the emergency department should be protocolized based on ATLS

guidelines i.e. Airway, Breathing and Circulation. Definitive airway in the form of endotracheal intubation should be performed in moderate to severe TBI. Hypotension and hypoxia must be avoided. Early CT imaging and neurosurgical consultation/intervention should be sought.

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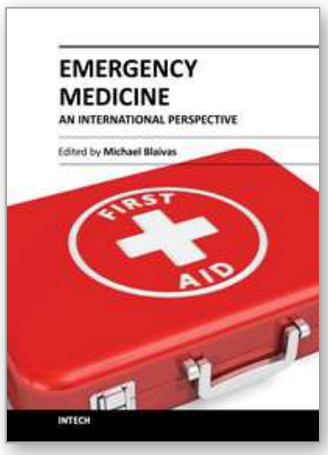

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