

Brain Injury

1. Discuss the epidemiology of brain trauma

2 million cases/yr, 500K admissions, 1/7 DOA, 80% mild, 10% moderate, 10% severe, 90K disabled, responsible for 50% traumatic deaths

2. Explain the Glasgow Coma Scale

Reproducible, widely used method of quantifying level of consciousness. (a) eye opening - 4 pts for spontaneous, 3 for opening to voice, 2 for opening to pain, 1 for no opening. (b) best motor response - 6 for obeys commands, 5 for localizes to pain, 4 for withdrawal from pain, 3 for flexor posturing, 2 for extensor posturing, 1 for no motor response. (c) best verbal response - 5 for conversant and oriented, 4 for conversant and disoriented, 3 for use of inappropriate words, 2, for incomprehensible sounds, 1 for no verbal response. Total score below 8 defined as coma.

- 3. Distinguish among mild, moderate, and severe brain injury

Mild = brief LOC, complete recovery, 10% permanent disability, may have persistent HA, memory deficits, difficulty w/ ADL for months.

Moderate = usual LOC, 10-15% w/ focal Ct abnormalities, may be lethargic/stuporous/combatative, 7% mortality, 66% permanent disability, likely to have long-term sequelae

Severe = Glasgow Coma Scale < 8 on initial exam, deterioration to GCS < 8, 40% focal mass lesion, 33% brainstem compromise, 36% mortality, survivors have long term issues.

4. Contrast cerebral concussion from cerebral contusion

Concussion = temporary LOC that occurs at time of impact, associated w/ short period of amnesia, majority have normal CT/MRI, injury result of physiologic not structural injury to brain, 5% will have intracranial hemorrhage, no evidence of macro/micro injury.

Contusion = result of scraping and bruising of brain as it moves along inner surface of skull, inferior frontal and temporal lobes common sites, lateral force may cause contusion deep to site of impact (coup) or on opposite pole of brain (counter-coup), may evolve to larger lesions over time, hemorrhage/edema/necrosis, more severe can rip into pia and cause SAH, deficits correspond to contused area.

5. Describe the presentations of traumatic epidural hematoma vs. traumatic subdural hematoma

Epidural hematoma = usually lateral skull fracture that lacerates middle meningeal artery/vein may not have LOC initially, lucid interval 1-2 hrs, then develop HA, obtundation, hemiparesis, and ipsilateral pupillary dilation from uncal herniation, bulging convex pattern on CT. young men more commonly, 5-43% mortality, triphasic presentation = unconscious, lucid, unconscious.

Subdural hematoma = HA and altered consciousness, time between trauma and onset is

longer than epidural, hemorrhage in cerebral convexities, usually from venous source, CT shows crescentic collection of blood across cerebral convexity. 50-90% mortality, rupture bridging veins, bleed into potential dural space, seen especially in elderly and EtOH

6. Relate the prognosis of gunshot wounds of the brain

60-70% dead at scene, 30-40% arriving: <20 mortality if awake/conscious, 90-100% mortality if unconscious, usually fatal if crosses midline, transverses both hemispheres, lodges in ventricle.

7. Distinguish between primary and secondary insults in the pathophysiology of traumatic brain injury

Primary = blow to head, direct trauma to brain, resulting SAR, epidural, direct head trauma, and acceleration/deceleration injuries.

Secondary = (a) systemic-hypotension, hypoxia, anemia, HTN, drastic change in Co₂, electrolytes, glucose, acid/base disturbances. (b) intracranial - ischemia, mass, edema, hydrocephalus, infection, seizure, hemorrhage.

8. Discuss the importance of the Monro-Kellie Doctrine

Three compartments (brain, CSF, blood) in confined space, intracranial pressure is determined by the sum of volumes in each compartment. Monroe-Kelly = to keep pressure in brain stable, volume change in one compartment must be offset by an equal and opposite volume change in one of the other compartments. Normal brain has tremendous capacity to control perfusion, keeps ICP low and blood flow stable. Metabolic autoregulation occurs, get vasodilation w/ hypoxia, hypercapnia, and hypoperfusion. Pressure autoregulation also occurs, increase pressure causes constriction, can result in change in arterial bed diameter from 200-300% and alter blood volume by 500-900%. In brain injury, lose autoregulation.

9. Examine the importance of intracranial pressure monitoring and when it is indicated

There is a strong correlation between successful lowering of ICP and better survival. ICP monitoring provides early warning of secondary insults. Monitoring is also important for cerebral perfusion pressure management. It is used to minimize the risks associated w/ treatment modalities. ICP can be manipulated to attenuate secondary insults by employing the monro-kellie doctrine.

ICP should be monitored" when GCS \leq 8, abnormal CT scan, or GCS \leq 8 w/ normal CT scan and 2 or more of the following (a) evidence of hypotension (b) 40 years old or older (c) motor posturing.

10. Describe the calculation of the cerebral perfusion pressure

