

Traumatic head injury

3rd year medicine

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INTRODUCTION

- Trauma is the leading cause of death in young children and young adults
- Head injury is a major contributor to mortality in more than 50%
- The cost of head injury is measured by:
 - a) Emotional cost of the loss or significant physical impairment of a family member
 - b) The cost to the community of loss of potential worth measured in achievements and dollars not earned
 - c) The financial cost of caring for these people not only during the acute phase of the illness but also long term - recently estimates US\$ 50 billion per year
- The impetus therefore is to understand the mechanisms and pathological processes involved and attempt to improve outcome from this condition
- These notes refer to craniocerebral trauma

INCIDENCE

- Causes of trauma to the nervous system (all age groups)

Road accidents	35%	
Accidental falls	28%	
Accidents (work related)	11%	
Assault*	10%	*locality dependant !!!!
Other causes	16%	

Bimodal cause distribution – 15-19 years = MVA, > 70 years = falls
- Incidence by age for neurotrauma:
 - 15 to 19 years = peak incidence
 - male > female
- Neurotrauma incidence by anatomical site:
 - Head 85%
 - Spine 13%
 - Peripheral nerves etc 2%

CLASSIFICATION

- 3 main methods

1. Injury mechanism

MISSILE INJURIES

A. High velocity

- Predominantly gunshot wounds
- Most lethal type of injury
- Injury determined by:
 - projectile characteristics (hollow or solid)
 - projectile orientation
 - temporary cavitation and shock wave
- Produce both a focal and diffuse type of injury with widespread destruction

B. Low velocity

- Classically the baseball/golf ball injury to the temple
- Produces a focal injury
- May be associated with underlying fracture and tearing of middle meningeal artery
- Can result in extradural haematoma

- Localised injury with good recovery possibility

NON MISSILE INJURIES

A. High velocity

- Typically the high velocity car accident although significant velocity of impact may occur with a simple fall from standing height (eg bar stool head injury)
- Involves angular acceleration, rotation and deceleration forces combined with an impact
- Imparts shear and strain injury to the axons and blood vessels
- Often results in diffuse injuries with significant morbidity and mortality
- Often associated with acute subdural haematomas, contusions and intracerebral haemorrhages

B. Low velocity

- Associated with striking the head against a hard object - during a fall, standing up or from an assault
- Associated with a focal injury that is localised to the area of impact.
- May not be associated with loss of consciousness
- Usually associated with good recovery although neurological deficit will depend upon location of focal injury (ie over the motor strip = motor weakness etc)

2. Pathological classification

PRIMARY BRAIN INJURY

- Arise from the initial traumatic event
- Result of mechanical forces producing tissue deformation at the time of injury
- Injury thresholds for different structures differ
- The range of lesions may include:
 - Scalp laceration
 - Skull fracture
 - Axonal injury
 - Vascular injury:
 - a) Cerebral contusions
 - b) Haemorrhage (subdural, epidural, intracerebral, subarachnoid)

SECONDARY BRAIN INJURY

- Secondary manifestations of craniocerebral trauma that is delayed in onset (minutes to weeks after injury)
- Often due to secondary insults such as poor oxygenation and perfusion of traumatised brain tissue.
- This additional "secondary insult" may exacerbate the original injury and is frequently more devastating than the initial injury
- This type of injury may be preventable by improved management techniques
- Secondary brain injury may include:
 - Traumatic ischemia and infarction
 - Hypoxic injury
 - Cerebral edema
 - Cerebral herniation
 - Infection

AXONAL INJURY

- Damage to axons may be considered according to the pathological classification of primary and secondary brain injury

A. Primary axotomy

- Axonal damage at the moment of trauma
- More commonly seen at the severe end of the spectrum although some degree now recognised in all head injury

- Simplistically a large axolemmal tear allows an influx of calcium and thereby activation of calcium-activated proteases. These proteases are associated with severe cytoskeletal disruption at the site of the axonal tear resulting in rapid disconnection of the distal axon. This is a permanent change and is irreversible (ie brain damage)
- Like primary brain injury this is a difficult problem to treat apart from preventing the injury in the first place. Research efforts, particularly in the pharmaceutical field are endeavoring to produce drugs that either limit the effect of axonal injury or offer prevention.

B. Secondary axotomy

- This follows the pattern of primary axotomy however the degree of cytoskeletal disruption is sublethal to the axon and it survives however it displays increased sensitivity to hypoxic/ischaemic insults. So along comes a secondary insult and finishes off the job - resulting in axonal disconnection and irreversible axonal injury
- This process may be delayed for minutes to some weeks after the initial insult.

3. Clinical classification

- Neurotrauma may be classified according to clinical criteria which is perhaps most useful in assessment and treatment of these patients.
- This involves classification according to the 15 point Glasgow Coma Scale which consists of 3 components with the following score values:
 - a) Eye opening**
 - 4 points: Spontaneous
 - 3 points: To sound
 - 2 points: To pain
 - 1 point: No opening
 - b) Verbal response**
 - 5 points: Orientated
 - 4 points: Confused
 - 3 points: Inappropriate words
 - 2 points: Incomprehensible sounds
 - 1 point: No response
 - c) Best motor response**
 - 6 points: Follows commands
 - 5 points: Localises to stimulus
 - 4 points: Withdraws to pain
 - 3 points: Abnormal flexor posturing
 - 2 points: Abnormal extensor posturing
 - 1 point: No response
- Correlation with injury severity:
 - a) GCS 14 or better = Mild head injury
 - b) GCS 9 to 13 = Moderate head injury
 - c) GCS 8 or less = Severe head injury

TRAUMATIC LESIONS

1. Diffuse axonal injury

- Defined as the presence of diffuse damage to axons in the cerebral hemispheres, corpus callosum, brainstem and cerebellum
- Is a pathological definition and diagnosis – not clinical

INCIDENCE

- 50% of primary injuries
- Some degree of axonal damage present in nearly **all** head injuries

CLINICAL PRESENTATION

- Severe end of the spectrum lose consciousness at impact
- 14 to 30% may have a lucid interval

ETIOLOGY

- Includes axonal shearing and damage resulting from rotational and acceleration and deceleration forces.
- Mechanisms include those outlined for primary and secondary axotomy

LOCATION

- Specifically affects:
 - a) Lobar white matter - at the gray-white matter interface
 - b) Corpus callosum
 - c) Dorsolateral aspects of the upper brainstem

IMAGING

- Initial CT scan may be negative
- CT and MR appearance rely on associated vascular injury in above locations

2. Skull fracture

- Present on 2/3 of CT scans in head trauma
- Up to 35% of severe head injury may have no identifiable fracture
- May be classified as linear, depressed or diastatic
- Linear fractures associated with EDH, SDH
- Depressed fractures associated with local parenchymal injury

3. Epidural hematoma

INCIDENCE

- 1 to 4 % overall

CLINICAL PRESENTATION

- Classically associated with a "lucid interval" (about 50% of cases)
- Delayed development in 30%
- Complain of headache, focal signs, deteriorating GCS
- Motor signs on contralateral side, pupil dilation ipsilateral (usually)

ETIOLOGY

- Lacerated middle meningeal artery (80%)
- Lacerated dural venous sinus

LOCATION

- Temporoparietal between the dural and skull
- 5% are bilateral

IMAGING

- Classically a biconvex mass that displaces the brain away from the skull

OUTCOME

- Mortality 5%

4. Acute subdural hematoma

INCIDENCE

- 10 to 20% of severe head injury
- 30% of fatal injury

CLINICAL PRESENTATION

- Most often associated with poor conscious state on admission to hospital

ETIOLOGY

- May result from:
 - a) tearing of bridging veins
 - b) rupture of temporal intracerebral hematoma ("burst temporal lobe")

LOCATION

- Most often frontoparietal convexities
- Middle cranial fossa

IMAGING

- CT - crescent shaped lesion spreading diffusely over the hemisphere and adhering to it's contour but displacing the brain away from the skull.

OUTCOME

- Mortality ranges as high as 85%
- Depends on many other factors including age

5. Chronic subdural hematoma

- These lesions by definition are more than 3 weeks old

INCIDENCE

- 1 to 2 per 100,00
- Occur in the older age group - mean age 63 years

CLINICAL PRESENTATION

- A history of trauma in < 50% (often forgotten)
- Present with minor symptoms: headache, confusion, TIA like Sx

ETIOLOGY

- Tearing of bridging veins
- Brain is atrophic and accommodates initial acute hemorrhage asymptotically

OUTCOME

- Mortality 10%
- 80% resume normal functioning

6. Subarachnoid hemorrhage

- 60 to 80% of severe injuries
- Often an indicator of the severity of the injury
- May be associated with vasospasm

7. Cortical contusions

- Superficial foci of hemorrhage along the surface of the brain
- Induced by the brain striking the bony ridges within the cranium
- Occur most often beneath the frontal and temporal lobes

8. Intracerebral hemorrhage

- Occurs in 15% of severe head injury
- 1/3 have LOC at admission
- Due to rupture of intrinsic vessels
- Often occurs at time of injury although 10% may be delayed

9. Cerebral edema

- Occurs to some degree in most head injury although may be severe in 10 to 20%
- Variety of mechanisms responsible including: cytotoxic, vasogenic, osmotic and interstitial
- May evolve in a number of patterns including:
 - a) swelling around contusion, intracerebral hemorrhages
 - b) diffuse swelling of 1 hemisphere
 - c) diffuse swelling of entire brain
- May be associated with uncontrollable intracranial pressure in 15% of severe injury and result in cerebral herniation and death

RAISED INTRACRANIAL PRESSURE AND CEREBRAL HERNIATION

Raised intracranial pressure

- Occurs in 70 to 80% following severe TBI
- Important determinant of outcome
- 10 to 15% will ultimately develop medically and surgically intractable raised intracranial pressure with an associated mortality of 84 to 100%
- Causes may include:
 - a) Diffuse head injury
 - b) Diffuse associated cerebral edema
 - c) Intracranial hemorrhage
 - d) Hydrocephalus
- Raised intracranial pressure if left untreated may ultimately result in cerebral herniation
- Clinical symptoms may include:
 - a) headache
 - b) meningism
 - c) altered conscious state
 - d) cranial nerve palsy – 3rd and 6th cranial nerves
 - e) Long tract signs - hemiparesis

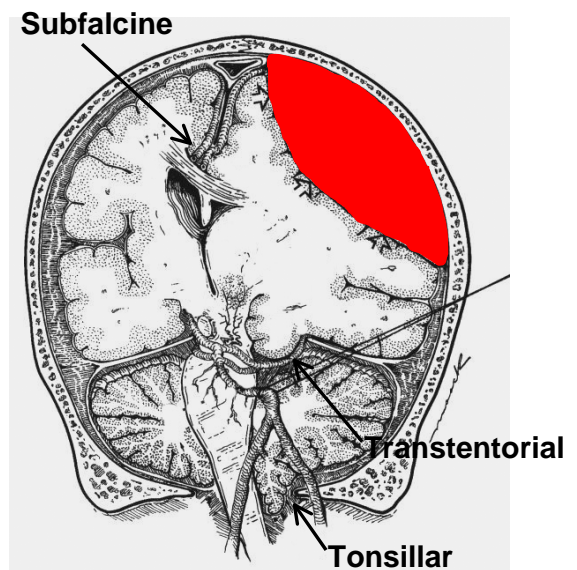
Cerebral herniation

SUBFALCINE HERNIATION

- Herniation of lateral ventricles beneath falx may result in:
 - a) Compression of the anterior cerebral arteries - infarction
 - b) Compression of the ventricular system resulting in obstruction and hydrocephalus in the opposite ventricle

TRANSTENTORIAL HERNIATION

- Medial temporal lobe herniates through the tentorial incisura and may result in
 - a) Compression of the posterior cerebral artery - infarction
 - b) Compression of the 3rd nerve - ipsilateral dilated pupil
 - c) Tearing of perforating arteries from the basilar artery - resulting in Duret hemorrhages
 - d) Compression of the ipsilateral cerebral peduncle - contralateral hemiparesis
 - e) Occasionally may produce a contusion in the contralateral cerebral peduncle as it strikes the opposite edge of the tentorium producing ipsilateral hemiparesis (Kernohans notch)



TONSILLAR HERNIATION

- Cerebellar tonsils descending through the foramen magnum
- Medullary compression results in:
 - a) Hypertension (+ associated bradycardia from peripheral vagal receptors) = Cushing response
 - b) Respiratory irregularities - Cheyne Stokes respiration
 - c) Death if not corrected

EARLY MEDICAL MANAGEMENT OF RAISED ICP

- Make the clinical diagnosis
- ABC (correct hypoxia, if GCS \leq 8 = intubate, fluid resuscitate)
- Reassess the patient
- If clinically improved – go to diagnostic procedure (CT scan)
- If not clinically improved – consider hyperventilation and iv mannitol (100 ml of 20%)
- Go to diagnostic procedure (CT scan)
- Neurosurgery consultation

LONG TERM SEQUELAE OF INJURY

- Pneumocephalus, CSF leaks
- Cranial nerve injury
- Diabetes insipidus
- Encephalomalacia and atrophy
- Hydrocephalus
- Post traumatic epilepsy
- Persistent vegetative state
- Focal neurological deficits
- Functional memory deficits
- Association with the development of Alzheimer's disease (Apo E allele)