Neuroimaging of vascular/secondary effects and sequelae of head trauma.

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Guidelines for identifying trauma patients at high risk for blunt cerebrovascular injury

- Cervical spine fracture with
  - Foramen transversarium involvement, or
  - 30% or more subluxation, or
  - Significant rotation or distraction mechanism
- Basilar skull fracture crossing carotid canal, foramen lacerum, or cavernous sinus
- Severe facial fractures (LeFort II or III; naso-orbital ethmoid complex, facial smash).
- Carotid or vertebral artery perivascular hematoma
- Horner’s syndrome
- GCS<6 at 24 h after initial assessment
- Neurologic examination incongruent with brain imaging
- Stroke or TIA
- Hanging attempt with cervical hematomas or cervical spine fractures

Primary vascular injuries

- Traumatic arterial dissection
  - Extracranial carotid dissections
  - Intracranial dissections or dissecting aneurysms
  - Vertebral artery dissection
- Carotid-cavernous fistulas
- Traumatic intracranial aneurysms
- Traumatic venous thrombosis

Traumatic carotid dissections

- 70% of carotid dissections involve both the cervical and petrous portions of the artery
- Dissections should be suspected if:
  - Neurologic deficits
  - Infarcts
  - Evidence of significant skull trauma
- Aneurysms occur in 58% of traumatic dissections
- Intimal flap present in 20-35% of cases
- Carotid occlusion occur in 20% of dissections
Intracranial dissections or dissecting aneurysms

- Affect the supraclinoid carotid or middle cerebral arteries
- The location of the intramural hematoma is usually subintimal
- Dissecting aneurysms often present with a "string-of-beads" appearance

Vertebral artery dissection

- 20% of all cervical vascular injuries
- A 24% to 46% incidence of vertebral artery injury associated with major blunt cervical spine trauma including fractures, subluxations or severe hyperextension or hyperflexion injury.
- The angiographic findings: stenosis, aneurysms and intimal flaps.
Traumatic intracranial aneurysms classification

- Proximal to the circle of Willis
  - Infraclinoid carotid artery
  - Supraclinoid carotid artery
  - Vertebrobasilar
- Distal to the circle of Willis
  - Subcortical
  - Cortical
TIA mechanisms of injury

- Infraclinoid carotid and basilar artery aneurysms are associated with:
  - Basilar skull fractures
- Supraclinoid carotid artery aneurysms can be the result of:
  - Either movement of the supraclinoid segment against the anterior clinoid process
  - Or stretching of the carotid artery
- Distal subcortical aneurysms occurs along:
  - The anterior cerebral artery and its branches
  - Traumatic movement of the brain and vessels against the fixed falx cerebri
- Posterior cerebral artery aneurysms
  - Result of trauma of the vessel against the tentorium

TIA diagnosis

- Patients with history of trauma and recurrent epistaxis, visual loss, progressive cranial nerve palsy or an enlarging skull fracture
- MRI/MRA/CTA → Arteriography
- Delayed formation of aneurysms
ICA just below the skull base
Petrosal internal carotid artery
Anterior aspect of the cavernous ICA
Pericallosal and callosomarginal arteries
Peripheral MCA
Secondary lesions

- The secondary effects of craniocerebral trauma may be more devastating than the primary injuries.
- Secondary lesions are those that develop subsequent to initial impact.
- They arise from
  - Either sequelae of primary lesions
  - Or the neurologic effects of systemic injuries
- Secondary lesions are potentially preventable, provided that causative factors are quickly recognized and appropriate treatment promptly instituted.
BRAIN INJURY

- Tissue damage
- Cerebral swelling/edema
  - Cytotoxic edema
  - Vasogenic edema
  - Brain swelling
- Cell lesion
- Ruptured BBB
- Cerebral vasoplegia

ICP
CPP
CBF

- Ischemia
- Hypoxia
- Hypercapnia
- Ischemia
- Hypotension
- Loss of autoregulation
- Vasoparalysis

Evaluation of relevant CT scan findings

- Status of the basal cisterns
- Midline shift
- Subarachnoid hemorrhage in the basal cisterns
Status of the basal cisterns

- Compressed or absent basal cisterns indicate a threefold risk of raised intracranial pressure and the status of the basal cisterns is related to outcome.
- The degree of mass effect is evaluated at the level of midbrain:
  - Open (all limbs open)
  - Partially closed (one or two limbs obliterated)
  - Completely closed (all limbs obliterated)

Midline shift

- The presence of midline shift is inversely related to prognosis.
- Midline shift at the level of foramen of Monro should be determined by first measuring the width of the intracranial space (A); next the distance from the bone to the septum pellucidum is measured (B).
- \[ \text{Midline shift} = \frac{A}{2} - B \]
Different types of cerebral herniations

- Subfalcine h.
- Transtentorial h.
  - Lateral
    - Anterior=uncal
    - Posterior=parahypocampal
  - Central
    - Ascending
    - Descending
- Transphenoidal h.
  - Descending
  - Ascending
- Tonsillar h.
- External h.


Subfalcine herniation

- It is caused by unilateral frontal, parietal or temporal lobe mass effect or edema
- The ipsilateral cingulated gyrus is pushed down and under the rigid midline falx
- Owing to arterial compression:
  - Focal necrosis of the cingulated gyrus
  - Infarction involving the ACA territory
Transtentorial herniation

- Symptoms may result from displacement, compression and stretching of the brainstem and cranial nerves
- Hemorrhage and infarction caused by compression and tearing of arteries and veins
- Increasing edema and intracranial pressure caused by venous obstruction
- Hydrocephalus caused by obstruction of the aquedcut
Post-traumatic cerebral infarction

- Mechanisms
  - Direct vascular compression by mass effect (81-88%)
  - Cerebral vasospasm
  - Vascular injury
  - Systemic hypoperfusion

PTCI

- PTCI is most common in the PCA distribution

- PTCI is an indication of a poor clinical outcome, especially among patients with:
  - Associated subdural hematoma
  - Brain swelling/edema
  - tSAH
DISTRIBUTION OF POSTTRAUMATIC CEREBRAL INFARCTION IN 16 PATIENTS


Day 0

Day 1
Outcome in patients with PTCI

- **Good recovery**: 43.8%
- **Moderate disability**: 12.5%
- **Severe disability**: 12.5%
- **Vegetative state**: 6.2%
- **Death**: 25%

Traumatic cerebral edema/swelling

- **Vasogenic** due to blood-brain barrier disruption - extracellular water accumulation
- **Cytotoxic/cellular** due to sustained intracellular water accumulation
- **Osmotic** caused by osmotic imbalances between brain and tissue
- **Hydrocephalic edema/interstitial** from edema related to an obstruction of CSF outflow

Traumatic diffuse cerebral edema/swelling

- **Early**
  - Gyral swelling, sulcal effacement, loss of gray-white matter interface
- **Late**
  - Diffuse low attenuation of the brain, generalized effacement of subarachnoid cisterns
  - "Pseudosubarachnoid hemorrhage"
  - "White cerebellum sign"
  - "The reversal sign"
Sequelae of head trauma

- **Early**
  - Delayed intracerebral hematoma
  - Delayed subdural hematoma
  - Delayed epidural hematoma
  - CSF leak or fistula

- **Late**
  - Cerebral atrophy
  - Cerebellar atrophy
  - Atrophy of the corpus callosum
  - Acquired cephalocele
  - Leptomeningeal cyst
  - hypopituitarism
Leptomeningeal cyst

- When a skull fracture is accompanied by a tear of the dura, meninges and brain tissue may herniate into the fracture, preventing healing of the fractures and permitting cerebrospinal fluid pulsation to enlarge the fracture and extent into the subgaleal space.
- 90% < 3 years.
- The average time of diagnosis is 14 months after the initial fracture.
Post-traumatic hydrocephalus

- PTH as low as 0.7% or as high as 20%
- PTH can present acutely, subacutely and syndrome of NPH
- PTH is to be distinguished from posttraumatic ventriculomegaly due to cerebral atrophy
- The appropriate management of these patients include:
  - Study of CSF dynamics
  - And/or ICP monitoring
Traumatic cerebrospinal fluid leak

- Acute CSF fistulas occur in 2 to 11% of all patients with closed head trauma
- 70% occurs within 1 week
- CT-cisternography
- High-definition CT alone
- MRI-CISS
conclusion

- CTA has been shown to be a good non-invasive alternative technique to catheter angiography for the initial assessment of **cerebrovascular trauma** and **vascular injuries** of the neck.
- Neuroimaging is playing a crucial role in defining the mechanisms of **secondary injury** in traumatic brain injury and, in turn, potentially identifying targets of new therapy.
- CT is adequate in the evaluation for early onset of **sequelae of head trauma**, MRI is the modality of choice for evaluation of subacute and later onset of delayed sequelae of head trauma.

Radiology in traumatic brain injury has a bright future.