Surgery – Dr. Ari – Lecture 4 – Intracranial Hematomas

INTRACEREBRAL HEMATOMA

They are due to areas of contusions coalescing into contusional hematoma
These appear as hyperdense lesions on CT scan with associated mass effect and midline shift

EPIDURAL HEMATOMA (EDH)

Is a traumatic accumulation of blood between the inner table of the skull and the stripped-off dural membrane
Extradural hematoma Occurs as a result of:
1. Temporal bone fracture with laceration of MMA
2. Fractured bone edges
3. Dural venous sinuses
A familiar convex configuration due to the adherence of the dura to the calvarium
There is typically a lucid period
Requires urgent evacuation via a burr hole prior to craniotomy

Clinical Features

History:
Fewer than 20% of patients demonstrate the classic presentation of a lucid interval.
Following injury, the patient may be continually comatose, briefly comatose and recovered, or continually conscious.
Severe headache
Vomiting
Seizure
Patients with posterior fossa EDH may have a dramatic delayed deterioration. The patient can be conscious and talking and a minute later apneic, comatose, and minutes from death.

Physical:
Cushing response consisting of the following can indicate increased ICP:
- Hypertension
- Bradycardia
- Bradypnea
Level of consciousness may be decreased, with decreased or fluctuating GCS.
Contusion, laceration, or bony step-off may be observed in the area of injury.
Dilated, sluggish, or fixed pupil(s), bilateral or ipsilateral to injury, suggest increased ICP or herniation.
Classic triad indicating transtentorial herniation consists of the following:
- Coma
- Fixed and dilated pupil(s)
- Decerebration

Hemiplegia contralateral to injury with herniation may be observed.

Because the underlying brain has usually been minimally injured, prognosis is excellent if treated aggressively.

EDH is uncommon in elderly patients because the dura is strongly adhered to the inner table of the skull. In case series of EDH, fewer than 10% of patients are older than 50 years.

**SUBDURAL HEMATOMA**

An acute subdural hematoma (SDH) is a rapidly clotting blood collection below the inner layer of the dura but external to the brain and arachnoid membrane. Two further stages, subacute and chronic, may develop with untreated acute SDH. Each type has distinctly different clinical, pathological, and imaging characteristics.

Resulting from torn bridging veins draining blood from the cortex to the dura or from cortical lacerations or from dural venous sinuses

Appear as a concave configuration

Classified to:
1. Acute: hyperdense, urgent evacuation
2. Subacute: from 7 days to 3 weeks
3. Chronic: 3 months

**Chronic Subdural hematoma**

Most common in infants and in adults over 60 years of ages.

Progressive neurological deficits more than 2 weeks after the trauma

Hypodense in CT scan

Can be drained through burr holes

Suspect acute SDH whenever the patient has experienced a mechanism of moderately severe to severe blunt head trauma.

Patients generally lose consciousness, but this is not an absolute.

Chronic SDH is more difficult to anticipate, and about half of such cases offer no history of head trauma. Patients often present with progressive symptoms such as unexplained headache, personality changes, signs of increased ICP, or hemiparesis/plegia.

Any degree or type of coagulopathy should heighten suspicion of SDH.

**SUBARACHNOID HEMORRHAGE**

Bleeding from intracranial vessels lie in the SA space which give off small perforating branches to the brain tissue or from an associated aneurysm.
Incidence
It represents about 5-10% of all non-traumatic ICH with an incidence of approximately 10-15/100,000 population per year.

Causes of SAH
Rupture of a berry aneurysm.
Rupture of AVM (6%) (Children)
Rare Causes:
- Bleeding from a tumor
- Bleeding disorders
- Blood dyscrasias
- Rupture of spinal AVM
- Undiagnosed cause in 15%

Presenting features:
1. Headache:
   - Sudden onset of a severe headache of a type not experienced by the patient
   - Described as a blow to the head
2. Deteriorated level of consciousness
3. Meningism
   - Headache, fever, neck stiffness, photophobia, and vomiting
4. Focal neurological signs: Due to:
   - Concomitant ICH: MCA (Temporal lobe and Parietal lobe), ACA (Frontal lobe)
   - Local pressure effect of aneurysm (PCA and 3rd nerve palsy)
   - Cerebral vasospasm (2-3 days after the bleeding)
5. Epilepsy
6. Optic fundi: papilloedema
7. Retinal hemorrhage (subhyaloid)
8. Reactive hypertension
9. Pyrexia

Diagnosis
History and examination:
- Sudden severe headache
- Decrease conscious state
- Meningism

CT scan
It will show the hemorrhage in 95% of patients with SAH (if within 48 hours of the bled)
Hydrocephalus
ICH
Aneurysm or AVM
Tumor
MRI
Not routinely used
Used in spinal AVM which cause SAH
CT/MR angiography
Non-invasive techniques
Will detect up to 95% of intracranial aneurysms
LP:
Xanthochromia:
Allow CSF to drip into 3 consecutive tubes
Cerebral angiography
Aneurysm
AVM
Vessel spasm

ANEURYSM
The most common cause of SAH with a maximum incidence in the 4th-5th decades of life
The majority of aneurysms occur in constant positions of the circle of Willis and about 85%
occurs in the anterior half of the circle
Clinical presentation
Rupture
Compression from aneurysm sac
  Visual
  Cranial nerves
  Pituitary
  Brainstem
incidental
Management
Severity of initial hemorrhage
Rebleed: 50% within 6 weeks
Antifibrinolytic agents are not used because of thrombotic phenomena (DVT, cerebral
thrombosis)
CEREBRA VASOSPASM

Hypertensive and hypervolemic therapy
Calcium channel blocking agents (Nimodipine)

Surgery:
Occlusion of the neck
Reinforcement of the sac
Proximal ligation of the feeding vessel
Endovascular procedures (detachable coils)

AVM
Most common cause of SAH in children and young patients
Clinical presentation:
  - Hemorrhage
  - Epilepsy
  - Headache
  - Progressive neurological deficits

Management
Excision
Stereotactic radiosurgery
Embolisation

[See the slideshow for the images.]