Intracranial Bleeds

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A Brief Review of Neuroanatomy:

Meningeal Layers:
- Dura mater
- Arachnoid
- Pia mater

Meningeal Spaces:
- Epidural – contains meningeal arteries & veins
- Subdural – traversed by “bridging” veins
- Subarachnoid - communicates with ventricles, includes cisterns, contains CSF, circle of Willis
Circle of Willis
(note proximity of CN III to the PCA)
Neuroanatomy – MR T1 axial

Optic nerve
Infundibulum
Oculomotor nerve
Posterior cerebral artery
Quadrigeminal cistern

Sphenoid sinus
Uncus
Crus cerebri
Cerebral aqueduct
Cerebellar vermis
Straight sinus
Superior sagittal sinus

I = Interpeduncular Cistern

Fix, *High Yield Neuroanatomy*
Interhemispheric fissure
Gyrus rectus
Optic tract
Mamillary nucleus
Red nucleus
Trigone
Superior colliculus
Middle cerebral artery
Uncus/amygdala
Crus cerebri
Substantia nigra
Posterior cerebral artery
Quadrigeminal cistern
Cerebellar vermis
Straight sinus
Superior sagittal sinus
Menu of Tests if Suspicious of an Intracranial **Bleed:**

Head CT  

or  

CT of Head
Advantages of CT over MRI

• CT detects early blood; MRI does not
  - **CT attenuation**: blood > brain due to globin protein in hemoglobin (Hct in hyperacute bleed is very high).
    - Therefore obtain **NON-CONTRAST CT**
    - attenuation increases 1st 1-3 days with clot retraction, then decreases with degradation
- **MR intensity** based on paramagnetic effects of hemoglobin breakdown products.

  - Oxyhgb = diamagnetic \(\rightarrow\) Isointense
  - Deoxyhgb (12-48hrs) = paramagnetic \(\rightarrow\) hyperintense
  - intensity increases with further breakdown to methgb
Advantages of CT over MRI cont‘d

• Modality of choice to assess for skull and facial fx’s
• Greater access to patient – status may decline
• Fast
• Widely available
MR does have its moments:

- Diffuse axonal injury – no blood
- Contusion w/out significant hemorrhage
- Deep cerebral or brain stem injury
- Small subdural hematoma
- Subacute subdural hematoma
Intracranial Hemorrhage: 4 main types

From outside to inside:

- Epidural (EDH)
- Subdural (SDH)
- Subarachnoid (SAH)
- Intraparenchymal (IPH)
And Variations Thereof:

- Multiple distinct injuries (as in trauma)
  - any combo of bleeds
- Extension
  - frequently SAH or IPH into ventricular system
- Intraventricular hemorrhage (IVH) can occur in isolation
  - secondary to shearing of subependymal veins (breach of blood/CSF barrier at choroid plexus)
Epidural Hemorrhage: Etiology

- Tear of middle meningeal artery or vein with subsequent bleeding into potential space
- Secondary to:
  1) Trauma with associated temporal bone fx
  2) Surgery
EDH: Clinical Presentation

- +/- initial loss of consciousness
- 50% with **lucid interval** for several hrs before obtundation, marked by sx:
  - H/A, N/V
  - Seizure
  - **Focal Neuro sx**: classically ipsilateral **blown pupil** & contralateral **hemiparesis** 2° uncal herniation
    - **Kernohan’s notch** may induce ipsilateral hemiparesis (compression of contralateral cerebral peduncle by tentorial edge)
    - **Blown pupil always ipsilateral to bleed**
EDH: Appearance on CT

- **Biconvex**, high attenuation extra-axial mass
  - does not cross suture margins
- **Fx of temporal bone (90% of cases)**
  - associated pneumocephalus
- **Areas of low attenuation = swirling blood**
  - indicative of active bleeding/rapid expansion
- **Mass effect:**
  - midline shift (calcified pineal gland helpful)
  - compression of ventricle, effacement of sulci
  - herniation: uncal, subfalcine
EDH

Swirling blood

Air

Calcified Pineal Gland

Midline Shift

Zee & Go, “CT of Head Trauma,” Neuroimaging Clinics of North America
**EDH: Treatment**

- Prompt craniotomy & evacuation essential to prevent fatal outcome
- If **CN III** involvement – early intervention necessary to regain function
Subdural Hemorrhage: Etiology

- Laceration of bridging cortical veins 2° to:
  - Trauma
  - Sudden acceleration & deceleration
  - Spontaneous rupture (~25% no h/o trauma)
  - Rapid decompression of obstructive hydrocephalus after shunt placement
- More common in elderly 2° to brain atrophy
## SDH: Clinical Presentation

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Signs</th>
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<tbody>
<tr>
<td>Vomiting</td>
<td>Depression of consciousness</td>
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<tr>
<td>Weakness</td>
<td>Pupillary asymmetry</td>
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<tr>
<td>Confusion</td>
<td>Motor asymmetry</td>
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<tr>
<td>Headache</td>
<td>Confusion &amp; Memory loss</td>
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<tr>
<td>Speech disturbance</td>
<td>Aphasia</td>
</tr>
<tr>
<td>Seizure</td>
<td>Papilledema</td>
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</table>
SDH: Clinical Presentation cont’d

• Depends on chronicity
  – In acute stage after trauma, often find focal neurologic deficits, but:
  – Sx & signs may be absent, nonspecific, or nonlocalizing especially with chronic SDH
  – HA especially chronic & altered consciousness (esp acute/subacute) are most common findings
• Look for signs of herniation as in EDH
# SDH: Appearance on CT

- **Attenuation depends on chronicity**

<table>
<thead>
<tr>
<th></th>
<th>Hyperacuté (hrs)</th>
<th>Acute (&lt;3d)</th>
<th>Subacute (4-20d)</th>
<th>Chronic (&gt;3wks)</th>
<th>Acute on Chronic</th>
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</thead>
<tbody>
<tr>
<td><strong>Hyperacuté</strong></td>
<td>Hyperattenuated</td>
<td>Hyperattenuated</td>
<td>Isoattenuated</td>
<td>Hypoattenuated</td>
<td>Distinct areas of hyper- &amp; hypoatt’n</td>
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<tr>
<td><strong>Acute</strong></td>
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</table>
SDH: Appearance on CT cont’d

• **Crescentic** extra-axial collection – not bound by suture margins, often tracks along entire hemispheric surface
  - SDH may mimic biconvex EDH in 1st 6 hrs

• Rebleeding into chronic SDH → layering of attenuations & calcified **membranes**

• **Isoattenuated subacute SDH** can be difficult to detect
  - Look for displacement of gray matter (att’n > “fatty” white matter), buckling of white matter, effacement of sulci
SDH: Appearance on CT cont’d

• Traumatic SDH often associated with other brain injury: contusion, IPH, 2\textsuperscript{nd} SDH
• Despite thin appearance, contains large vol.
• \textit{Edema + large vol = more mass effect than expected from size of SDH alone}
  – Midline shift, herniation, ventricular shift & compression
Hyperacute SDH: Unresponsive 80yo woman

Crescentic hyperattenuation with hypoattenuated swirling

Uncal herniation with compression of cisterns & midbrain
Acute on Subacute SDH: Unresponsive 95 yo 2 wks after SDH

Effaced sulci & flattened gyri adjacent to subacute SDH

Acute new blood
**Acute on Chronic SDH:** unresponsive 76 yo man

Calcified membrane of chronic SDH (note extensive midline shift)
SDH: Treatment

- **Hyperacute/Acute** → Neurosurgical emergency → large craniotomy & evacuation
- **Subacute** → next day elective surgery
- **Chronic** → elective surgery → small craniotomy so as not to disturb vascularity of membranes
Subarachnoid Hemorrhage: Etiology

- **Traumatic** - w/ bleeding from 3 sources:
  1) Direct injury to pia vessels
  2) Hemorrhagic cortical contusion (IPH)
  3) Extension from intraventricular hemorrhage

- **Non-traumatic** (less common)
  - Ruptured aneurysms (75%) -- Occur at branching points of circle of Willis
  - Ruptured AVMs (10%)
SAH: Clinical Presentation

- **Traumatic** – variable external & neuro signs
- **Nontraumatic**: signs & symptoms 2º to subarachnoid blood:
  - N/V, confusion, obtundation, LOC
  - “Worst headache of my life” – HA of sudden onset but most significant for its **newness**
  - Increased blood pressure
  - Fever (meningeal irritation)
  - Nuchal rigidity/meningeal signs (hrs after HA)
  - Peripapillary retinal hemorrhages = most suggestive of dx (due to increased ICP)
SAH: Clinical Presentation

• Nontraumatic
  – **Ruptured aneurysm**: usually no focal neuro signs
    • Exception = CNIII palsy 2° PCA aneurysm
  – **Ruptured AVM**: may produce focal neuro signs
    • AVMs often occur in MCA distribution: aphasia, hemiparesis, visual field defect
SAH: Appearance on CT

- **Trauma** → high attenuation in sulci
- **Aneurysm** → high attenuation in basilar cisterns (region of circle of Willis)
  - Creates “star” pattern
- Assess for intraparenchymal extension
  - Common w/ AVMs & occasional w/ high pressure aneurysms of ICA/MCA
- Assess for complicating hydrocephalus
Let’s discuss aneurysmal rupture:
**SAH:** 58 yo woman with ACA aneurysm

Note high attenuation blood in:

- **Interpeduncular cistern**
- **Suprasellar cistern**
- **Sylvian fissure**
SAH 2º to ruptured aneurysm: Complications

- Recurrence of hemorrhage – 20% w/in 10-14 days after aneurysmal rupture
- Intraparenchymal extension
- Arterial vasospasm → ischemia (days 4-14)
- Hydrocephalus 2º impaired CSF absorption
  – Progressive somnolence, impaired upgaze
- Seizure – only w/ cortical injury
SAH: Further Radiological W/U

- MRA &/or 4 vessel angiography to assess for aneurysm(s)/AVMs
- Transcranial Doppler (TCD) – to assess vasospasm
SAH: Treatment

- **Medical**
  - Bed rest, head elevated, analgesics, sedation
  - BP: reduce to 160/100, avoid hypotension
  - Ca channel blockers – vasospasm prophylaxis
- **Surgical** - Candidacy determined by sx & level of consciousness (no surgery if stupor or coma)
  - AVM: if accessible, resxn/ligation/embolization
  - Aneurysm: clip neck or coil placement
Intraparenchymal Hemorrhage: Etiology

• **Trauma**
  – Contusion = laceration of cortical parenchyma (coup or contrecoup), +/- LOC, +/- hemorrhage

• **Nontraumatic**
  – **HTN** – acute or chronic
  – Coagulopathies/anticoagulation
  – Ruptured AVMs
  – Hemorrhage into tumors
  – Hemorrhage into infarcts
  – Drug use – cocaine, amphetamines
  – Amyloid angiopathy
IPH: Clinical Presentation

- Variable: depends on anatomical location
- Traumatic $\rightarrow$ cortical $\rightarrow$ focal signs, seizure
- Nontraumatic
  - HTN: single penetrating arteries
    - Putamen/Caudate
    - Thalamus
    - Pons
    - Cerebellum
    - White matter
IPH: Clinical Presentation

- **Selected hemorrhagic syndromes:**
  - **Putamen or Thalamus:** contralateral sensorimotor deficit (proximity to internal capsule)
  - **Pons:** early coma (reticular activating system), pinpoint pupils, absent horiz eye movements
  - **Cerebellum:** N/V, vertigo, gait ataxia
    - **Neurosurgical emergency**
IPH: Appearance on CT

Rt. Caudate IPH

Rt. Parietal white matter IPH
**IPH: Treatment**

- **Medical** – not much you can do:
  - BP: antihypertensive therapy = controversial
    - Reduced BP can → hypoperfusion, b/c chronic HTN causes loss of autoregulation
  - Mannitol or steroids for edema →?effective

- **Surgical**
  - Cerebellar decompression = critical
  - Cerebral decompression if large & accessible
Our Patient

- 46 yo woman with sudden onset HA, Vomiting, L hemiparesis, & Lethargy
- CT @ Outside hospital showed R basal ganglia IPH w/ ventricular and cistern (SAH) blood
- Pt transferred to BIDMC and underwent both MRA and 4 vessel angiography
Our patient: MRA showed a 2.5cm aneurysm at R ICA/MCA junction

Aneurysm

Internal Carotids

Middle Cerebrals

Vertebrals

Basilar

Anterior Cerebrals
Our Patient: Angiography

Film findings: confirmed R ICA/MCA aneurysm
Our Patient

• Pt underwent R ventricular drain placement and admitted to SICU
• Next day underwent craniotomy w/ clipping of R ICA bifurcation aneurysm
• 2 days post-op pt blew R pupil
• A head CT was obtained:
Our Patient: Post-operative Head CT

Large R basal ganglia IPH

IVH & midline shift evident
Our Patient

- A left side ventricular drain was placed with resolution of blown pupil
- Pt improved neurologically
- Discharged to acute rehabilitation with L hemiplegia
References


• Fix, James D., PhD, High-Yield Neuroanatomy, 2000.


• Zee, Chi Shing, MD & Go, John, MD, “CT of Head Trauma,” Neuroimaging Clinics of North America, 8(3), 525-539.
Acknowledgements

- Peter Warinner, MD
- Matt Spencer, MD
- Daniel Saurborn, MD
- Ram Chavali, MD
- Beverlee Turner
- Matt Halpern -- for letting me wear his tie
- Larry Barbaras and Ben Crandall our web masters