Subdural Hematoma

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Acute SDH

Chronic SDH

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Newborns

Older infants

Subdural Tap

Left updated: August 8, 2020

Epidemiology

• men : women = 3:1
• most patients > 70 yrs.
• more common than EDH.

1. Acute SDH

• age: < 20 yrs
• more common in very young and elderly
• typically results from vigorous head motion (acc., deceleration injury)
• iatrogenic: subdural tap, subdural catheter, or ventriculostomy

2. Spontaneous

• (30% of chronic SDH, i.e. intrinsic susceptibility – prone to recur)

• coup injury
• contrecoup injury
• contusions
• lacerations, intracerebral hematomas
• parenchymal injury
• bridging veins
• small veins (e.g. PComA → convexital SDH; distal ACA → parafalcine SDH)
• intracerebral hemorrhage
• ruptured intracranial aneurysm
• other vascular etiologies

Pathology

• SDH is not usually associated with skull fractures
• SDH is more common in infants (both have larger subarachnoid space)
• SDH occurs with associated parenchymal injury
• SDH may reach > 100 mL before becoming symptomatic!

Pathogenesis

• movement of brain relative to skull – ruptures venous sinuses, capillaries
• subdural space – run from cortical surface to dural sinus; commonly found along sagittal sinus and around anterior tip of temporal lobe.
• rarely, bleeding source may be cortical arteries or oozing brain laceration.

Classification

Acute SDH manifests during first 72 hours.

• common type of traumatic intracranial hematoma (5-30% of severe head injuries; ~ 1% of mild head injuries)

• commonly (> 50%) associated with extensive primary brain injury!!!
• diffuse parenchymal injury, contusions, lacerations, intracerebral hematomas - play major role in outcome!!!
• more common in elderly and in infants (both have larger subarachnoid space - allows for more movement between brain and dura).

Chronic SDH manifests when > 20 days old.

• most common after age 50 with apparently insignificant head trauma.

SDH is more common in very young and elderly (vs. EDH)

• average age of trauma patient without acute SDH - 26 years
• average age of patient with acute SDH - 41 years.

• mortality
• simple SDH (if no other brain injury) = 20%; complicated SDH (e.g. with contusions) = 60%.
• GCS 12-15 = 9%.
• GCS 3-5 = 76%.

Subacute SDH manifests when 3-20 days old.
• surgical literature favors > 3 days; radiological literature favors > 7 days.

Etiology

1. Vigorous head motion (acceleration-deceleration injury*) – may be trivial!

2. Spontaneous

• (30% of chronic SDH, i.e. intrinsic susceptibility – prone to recur)

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Location

1. along cerebral convexities - most common!
2. along interhemispheric fissure and tentorium* (often associated with shaken baby syndrome)
• < 1%: between occipital lobe and tentorium
3. posterior fossa (< 1%): cerebellum undergoes little movement; most SDHs here are result of parenchymal cerebellar injury – posterior fossa SDHs have highest mortality!
4. subdural space (unlike epidural space) is not confined by cranial sutures and has no adhesions – SDH rapidly spreads along entire hemisphere and into hemispheric fissure, limited only by dural reflections at midline / tentorium.
5. bilateral SDHs (< 10%) are more common in infants - adhesions in subdural space are absent at birth and develop with aging.

Subdural Hematoma

• SDH is rapidly clotting
• cannot tamponade beginning hematoma
• brain atrophy (→ regional ischemia

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Chronic SDH manifests when > 20 days old.

• most common after age 50 with apparently insignificant head trauma.
most are derived from subdural hygroma; minority develop from untreated acute SDH.

• commonly associated with cerebral atrophy.

• risk factors for chronic SDH: elderly with cerebral atrophy, chronic alcoholism, epilepsy, bleeding disorders, arachnoid cysts, cardiovascular disease (hypertension, arteriosclerosis).

• 8.7-32% are bilateral.

• mortality ≈ 5-10%.

• small SDHs often spontaneously resorb; larger SDHs liquify (in ≈ 1 week) and form encapsulating vascular membranes (fibroblasts grow from dural surface and form thicker outer membrane by about 7 days, and thinner inner membrane after 2-3 weeks), rarely calcifies.

  - blood in chronic SDH has liquid consistency, typically resembling crank case oil (can be drained through burr holes!).
  - membrane consists of many fragile capillaries, intact and lysed RBCs, hemosiderin-laden macrophages, and granulation tissue.
  - organized hematoma is firmly attached by fibrous tissue to dura and is not at all adherent to arachnoid (arachnoid does not contribute to membrane formation).

• at some point, critical mass is reached (hematoma assumes biconvex shape and becomes symptomatic):
  a) bleeding into chronic SDH (small recurrent hemorrhages from thin-walled vessels within membrane due to repeated minor trauma); up to 45% chronic SDHs rebleed; risk of rebleeding is greatest in first few months.
  b) osmotic swelling – due to blood breakdown and increased protein content (draws water osmotically across subdural membrane → clot enlargement).

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Dura has been reflected above to reveal bridging veins:

Dura has been reflected back (with small portion visible at lower right) to reveal subdural hematoma:

Here is bilateral chronic subdural hematoma; blood clots are brown to tan because of organization:
**CLINICAL FEATURES**

A. Clinically silent.

B. Brain compression (slow venous bleeding enables large hematomas to form before clinical signs appear) - can progress rapidly or slowly.

Most acute SDHs manifest within 48 hours.  
- 50% of patients are unconscious from time of injury; (SEMIELUDIC INTERVAL is observed in 30-70% cases.  
- there may be focal signs* (due to prolonged brain tissue compression under hematoma), but often clinical manifestations are nonlocalizing (due to ICP); later, brain herniation may develop.  
*deficits are soft (not as profound as in other hematomas); hemianesthesia, hemianopsia are seldom observed (anatomic structures are deep and not easily compressed)

In pre-CT era, chronic SDH earned label “great imitator” because of variable course and presentation (sometimes mistaken for dementia, stroke, or brain tumor!).  
- in 25-50% cases, there is no clear history of head trauma.  
- signs or symptoms fluctuate in ≤24% cases (mimic TIA’s).  
- headache (90%), mild hemiparesis (45-58%), confusion (50%), drowsiness (40-50%), personality changes, papilledema, gast dysfunction, seizures are most common presenting features.

N.B. after hematomas have exerted pressure on brain for long time (perhaps year or more), removing them does little to improve cognitive function.

**Nonseizure, Stereotypical, and Intermittent Symptoms (NESSIS)**  
- NESSIS clinically more commonly manifests as symptoms lasting > 5 minutes, dysphasia, preserved awareness, lack of positive symptomatology (such as clonic movements), lack of response to AEDs.  
- possible pathophysiology – cortical spreading depression or depolarization (CSD) (phenomenon similar to seen in SAH-associated vasospasm, stroke. TBI) – first described in animals in 1940 by Leão.  
- EEG is negative for ictal or epileptiform discharges*, EEG most commonly shows focal or generalized slowing.  
*N.B. scalp EEG has only 70% sensitivity to detect ictal or epileptiform discharges; differentiation from seizures is difficult without EEG; some clinical features are helpful:

<table>
<thead>
<tr>
<th>Feature</th>
<th>Odds ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Negative symptoms</td>
<td>1.00 (1.00-1.00)</td>
</tr>
<tr>
<td>Duration ≥ 5 min</td>
<td>1.02 (1.00-1.04)</td>
</tr>
<tr>
<td>Dysphasia</td>
<td>1.02 (1.00-1.04)</td>
</tr>
<tr>
<td>Migraine</td>
<td>1.02 (1.00-1.04)</td>
</tr>
<tr>
<td>≥ 5 epiphanies</td>
<td>1.00 (1.00-1.00)</td>
</tr>
<tr>
<td>Stereotypy</td>
<td>1.00 (1.00-1.00)</td>
</tr>
<tr>
<td>Impaired awareness</td>
<td>1.00 (1.00-1.00)</td>
</tr>
<tr>
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*Proposed Scoring System for the Diagnosis of NNESSIS (before EEG) (sensitivity of 96.6%, specificity of 100%)

**DIAGNOSIS**

**Hemostasis** (PT, aPTT, platelet count)

**LP** (absolutely contraindicated) – xanthochromia, variable number of RBCs.

**Noncontrast CT** - crescentic collection over hemispheric convexity without extension into depths of sulci; can cross suture lines and continue along falx and tentorium (do not cross midline!):
Acute SDH - hypodense* (40-90 HU); rarely, can appear isodense:
  a. low hematocrit (anemia)
  b. hyperacute clot (< 1 h old)
  c. active bleeding
  • small hematomas may not be depicted because attenuation similar to adjacent inner table of skull (H: wider CT window and level, e.g. 240 and 80 HU).

Subacute SDH - isodense*, important signs - effacement of cortical sulci, displacement of gray matter-white matter junction (‘bulking’); membranes are not vascularized.
  • better visualization - MRI (high signal on T1) or contrast-enhanced CT (opacification of cortical vessels - definition of brain margins).

Chronic SDH – hypodense* (15-30 HU, i.e. isodense to CSF); vascularized membranes enhance with contrast.
  *compared to brain.

Rule of thumb: blood remains denser than brain for 1 week, and less dense after 3 weeks.

- underlying brain is flattened (mass effect), and subarachnoid space is often clear.
  - absent midline shift - suspect contralateral mass (e.g. ≥ 20% chronic SDHs are bilateral);
    useful sign – ventricular frontal horns lie closer together (‘rabbit’s ear’ configuration).
  - excessive midline shift - suspect underlying cerebral edema.

- in interhemispheric SDH, falx cerebri appears thickened and irregular.
  - interhemispheric: SDH may mimic SAH (subarachnoid blood clears after several days; SDH remains wedge-shaped, smooth-bordered, hyperattenuating lesion).
  - interhemispheric: SDH may spread onto tentorium → characteristic ‘comma shape’ on axial CT.

- posterior fossa SDH does not cross midline or extend above tentorium (vs. EDH).

- temporal and tentorial SDHs are better detected on coronal MRI (than on axial CT).

- rebleeding into subacute / chronic SDH makes hematoma biconvex and heterogeneous density (mixed old and fresh blood, sedimentation levels) – in general, looks like EDH with heterogeneous density.

- size of extra-axial hematoma is more important factor than whether blood is epidual or subdural in location!

Suspect child abuse! (esp. if posterior interhemispheric & tentorial SDHs)

- osmotic swelling of chronic SDH makes hematoma biconvex and water density – in general, looks like EDH with water density.

MR is most sensitive imaging test! (esp. in subacute and chronic phase).

Absence of clear history of trauma → angiography (search for ruptured aneurysm or dural AV fistula).

- angiographic signs of SDH – avascular zone between skull and brain with away dislocation of major vessels.

MRI - subacute SDH with extension into anterior interhemispheric fissure (note that sutures do not contain spread):

SDH with adjacent SAH due to ruptured MCA aneurysm:
Subdural Hematoma

SDH due to ruptured right PCoM aneurysm:

Right frontal subacute SDH; note displaced gray matter–white matter junction, and midline shift:

Subacute-on-chronic SDH with blood–fluid level (acute hemorrhage into chronic collection):

Acute-on-chronic SDH:

Subacute SDH - less dense than brain but denser than CSF; it is denser posteriorly; midline displacement is greater than would be expected from size of lesion - suggests extensive underlying swelling; contralateral (left) ventricle is dilated.
Bilateral SDHs, right greater in size than left.

Source of picture: “WebPath—the Internet Pathology Laboratory for Medical Education” (by Edward C. Klatt, MD) >>

**Acute SDH**

MRI - bilateral SDHs (arrows) with suboccipital extension.

Interhemispheric acute SDH.
**Subdural Hematoma**

Bilateral chronic SDHs, midline shift is absent.

Chronic SDH with no mass effect.

Bilateral chronic SDHs with acute rebleeding.

Acute SDH extending over entire left hemisphere; sedimentation level is seen (patient has clotting dysfunction).

**Subacute SDHs:**

A) Noncontrast CT inward displacement of gray-white matter junction of left cerebral hemisphere; small sedimentation level is apparent in posterior portion.
Acute and subacute SDHs:
A. T1-MRI - large fluid collections (white arrows) consistent with large subacute SDHs.
B. T2-MRI - hypointense signal (white arrows) representing subacute blood and hyperintense signal (black arrows) representing more recent hematoma.

Calcified SDHs:
A. In frontal view, bilateral shell-like calcifications form cast of cerebral hemispheres.
B. In lateral view, outer calcified membranes are near bony skull, and inner membranes are separated from outer by relatively clear zones.

TREATMENT
In all cases, hematoma complete resolution should be documented (conservatively treated acute SDH can evolve into chronic SDH).

CONSERVATIVE MANAGEMENT
- Surgical drainage is not required in many cases (acute SDH may disappear spontaneously, but may evolve into subacute or chronic lesion!) - managed with serial CT and close neurological observation in a neurosurgical ICU.
- Seizure prophylaxis and other measures → see p. TrH1.
- Monitor coagulation parameters!

CHRONIC SDH
- Chronic SDH can be treated with TRANEXAMIC ACID (TXA) 650-750 mg PO daily for 30 days without concomitant surgery; tranexamic acid might simultaneously inhibit fibrinolytic and inflammatory (kinin-kallikrein) systems, which might consequently resolve CSDH; some experts administer it postop (after bur hole washout).
• Dr. Okonkwo (UPMC) gives MEDROL, DEXAFAC, Dr. Day gives DEXAMETHASONE 2 mg qibas for 2 weeks, then tapers based on repeat CT findings.

• EG-1964 (Edge Therapeutics, Inc.) - polymer-based filament that contains APOTENON - pancreatic trypsin inhibitor.  
  — indicated to prevent rebleeding and reduce the need for blood transfusions following cardiac bypass surgery  
  — IV administration has serious thrombotic side effects  
  — EG-1964 delivers sustained dose of aprotonin over 21-28 days directly to site of SDH

• chronic SDH growth is due to the highly friable nature of the vascularized membrane that forms after initial injury; proposed treatment for recurrent (or new in high risk patients) symptomatic SDH - middle meningeal artery (MMA) embolization with the goal of eliminating the arterial supply to this vascularized membrane.  
  — use PVA particles - able to travel distally within the MMA vascularity and cover a broad anatomic area, limiting the chance of new collaterals forming distally (vs. liquid embolics such as Onyx or nBCA glue provide more of a “stump” embolization proximally).

**HEMATOMA EVACUATION**

**ACUTE SDH**

Guidelines: "Surgical management of acute subdural hematomas" in Neurosurgery. 2006 May;58(3 Suppl):S16–24

- indications for ASAP surgery: see also p. TIH 1
  a) acute SDH with a thickness > 10 mm, regardless of the GCS score
  b) midline shift > 5 mm, regardless of the GCS score.  
  c) GCS < 9 and GCS decreased between the time of injury and hospital admission by ≥ 2 points
  d) GCS < 9 and asymmetric or fixed and dilated pupils
  e) GCS < 9 and ICP > 20 mm Hg

- acute SDH in coma (GCS < 9) → ICP monitoring.

- surgical evacuation of an acute SDH should be performed using a craniotomy ± bone flap removal and duraplasty. see p. Op320 >>

*delay for 4 hours is a benchmark of neurosurgical care:

[Graph showing Timing of Surgery From Injury vs. % Deficit]

Selig et al. (1981)

Wilberger et al. (1991)

Wilberger et al. (1991) study also revealed that the appropriate strategies to prevent secondary brain injury may be more important than the timing of surgery in determining outcome (i.e. an increased focus on how to manage ICP during the interval between injury and surgery).

**CHRONIC SDH**

- any coexisting pathology must be reversed ASAP.

- must be surgically evacuated if symptomatic* (+ significant mass effect on imaging):
  *except mild headaches

A. Burr hole craniotomy ("burr hole washout") - procedure of choice.  
  see p. Op320 >>

B. Twist drill craniotomy – at bedside for very sick patient. see p. Op320 >>

C. Craniotomy – indicated for:
  a) multicellular / calcified SDHs.  
  b) burr hole / twist drill drainage failures.  

N.B. some experts advocate craniotomy as first choice for SDH but studies show that all results (complications, dispo, recurrences requiring reoperation*, mortality) are worse than with B/HWO

*24% (vs. 6-7% for B/HWO)

Postoperatively see p. Op320 >>
PROGNOSIS

Most important prognostic factors:
1. Concomitant primary brain injury
2. GCS score
3. Age (esp. > 40 yrs).
4. Time from trauma to surgical evacuation of hematoma (esp. > 4 hours)

After surgical evacuation, subdural hematoma occurs in 5–30% patients. Chronic SDH increases mortality 17-fold at 1 year.

SDH IN INFANTS

NEWBORNS
• SDH was once considered most common intracranial birth injury (but now ↓ with improved obstetric care).
• In majority of cases, bleeding is bilateral and located over dorsolateral surfaces of frontal and parietal lobes.
• Clinically: seizures, pallor, tense anterior fontanel, rapidly enlarging head, hypotonia, poor Moro reflex.
  - may be abnormal at birth (no spontaneous respiration, severe hypotension, seizures, retinal hemorrhage).
  - Initial manifestation may be generalized seizures within first 6 months of life.
  - Acute cases may progress to herniation.
• Diagnosis – ultrasound, CT, funduscopic (50% show retinal or subhyaloid hemorrhages).
  - If SDH interfered with brain growth, skull vault may be thick, with paucity of convolutional impressions, and hypoplasia of air cells and paranasal sinuses.
  - Calcification streaks / plaques, often parallel to vault, may be visible in capsule of chronic SDH.
• Treatment – evacuation through craniotomy.

OLDER INFANTS
• SDH is most common intracranial lesion in children < 2 years:
  a) shaken baby syndrome!! (chronic SDH in infants who do not yet walk)
  - See p. TH20 >>
  b) Complication of shunt procedures
  - Breeding disorders
  - Clinically: macrocrania (symmetrical or asymmetrical), tense anterior fontanel, lethargy / irritability, seizures, poor feeding, vomiting, failure to thrive.
• Diagnosis – CT, diagnostic subdural tap should be done bilaterally, even if positive on one side.
• Treatment – repeated daily subdural taps monitored by CT, head circumference measurements.
  - Resorption of hematoma must occur over weeks → months.
  - If > 10 taps are done (symptoms persist after 2 wk of daily drainage) → surgical treatment (e.g. subdural-peritoneal shunting or subdural-subgaleal shunting for 6 months; historically – subtemporal craniectomy – to let fluid drain into pterygoid fossa).
  - Removal of membranes was once thought to be important to avoid brain growth restriction, but this no longer seems necessary.
• 25% have some psychomotor retardation.

SUBDURAL TAP
• Anterior fontanelle becomes effectively closed between 9 and 18 months.
• Equipment – sterile prep and drape, goves, razor, blunt short-beveled spinal needle with stylet (20 G, 1½ or 2½ inch), manometer, CSF collection tubes, and local anesthesia (if desired).
• Scalp is shaved anterior to ears (over lateral margins of anterior fontanelle).
• Restrain infant by bundling him in sheet; scalp is shaved over lateral margins of anterior fontanelle where it meets coronal (avoid excessive neck flexion).
• Raise skin wheal of local anesthetic at puncture site.
• Insert needle through skin at extreme lateral limit of anterior fontanelle where it meets coronal suture (i.e. at least 2-3 cm from midline → to prevent sagittal sinus injury).
  - Use zigzag puncture to prevent later fluid leakage (puncture dislocated skin at right angle, then aim needle laterally).
  - Dura is entered with sudden “popping” sensation.
  - Cerebral cortex is ≈ 1.5 cm from skin surface (attachment of hemostat 5 mm).
• Remove stylet – subdural fluid is allowed to drain spontaneously (fluid is never aspirated - risk of drawing pial vessels into point of needle).

SUBDURAL HEMATOMA

TH13 (10)
- only 10-20 mL of subdural fluid should be removed from each side at one time (removing larger amounts may precipitate rebleeding or shock).
- if pial vessel is punctured, bleeding will usually cease spontaneously.
- pressure is measured with manometer.
- specimens are collected for Gram staining, culture, cells, glucose, and protein.

- remove needle → apply pressure → firm sterile dressing → place in sitting position (to prevent leakage).
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