Hemorrhagic

Intracerebral Hemorrhage

Intraparenchymal Hemorrhage (IPH)

Definition

Abnormal blood in the brain parenchyma deriving from arteries / small arterioles. Localized hematoma that dissects through tissue, spreading along WM


Location: 50% deep, 35% lobar, 10% cerebellar, 6% brainstem (REF: Stroke. 2005; 36: 934–937 http://stroke.ahajournals.org/content/36/5/934.long)

Epidemiology


Age: vs 45-54 yo, <45 = IR 0.1, >85 = IR 10 (REF: Lancet Neurology, Volume 9, Issue 2, Pages 167 - 176, February 2010)


Differential of Location

Outer -> Inner

Subgaleal

Epidural (EDH)

Subdural (SDH)

Subarachnoid (SAH)

Intraparenchymal (IPH)

Intraventricular (IVH)

Pathophysiology

Occur in penetrating vessels, sharp angles, in thalamus, basal ganglia, pons, & cerebellum

Cerebral Microbleeds


Mechanism of Brain injury

Direct mechanical injury and edema due to expansion of clot

Decreased blood flow around clot à ischemia: cytotoxic edema, release of inflammatory mediators

Increased ICP

Brain herniation
Etiologies

Hypertension
Cerebral amyloid angiopathy
AVM or dural AVM
SAH: aneurysmal, RCVS, traumatic
Anticoagulant or Thrombolytic use
Cavernous angioma
CNS vasculitis
Cocaine induced
Hemorrhagic Transformation of Ischemic infarction
Intracranial Neoplasm or Infection
Trauma: contusion
Cerebral Venous/Sinus Thrombosis

Risk factors

Older Age
Hypercholesterolemia
HTN
Smoking
EtOH, over-the-counter, or recreational drugs
DM
Anticoagulation or antithrombotic
Trauma
Prior stroke
Hematologic or coagulopathic conditions including liver disease


**Underlying conditions**

Bleeding disorder, trauma, AVM, aneurysm, HTN, venous thrombosis, hemorrhagic mass lesion or ischemia, amyloid (usually lobar)

**Clinical features**


Vomiting: ICH > SAH or ischemic stroke

Impaired LOC, increased BP common

Stupor / coma: Poor prognostic sign

EKG: Can have prolonged QT / depressed ST segment, flat / inverted / peak T waves


<table>
<thead>
<tr>
<th>Symptoms</th>
<th>n</th>
<th>Hemorrhage %</th>
<th>Sens</th>
<th>Spec</th>
<th>LR+</th>
<th>LR-</th>
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<tbody>
<tr>
<td>Age60</td>
<td>1510</td>
<td>16%</td>
<td>50</td>
<td>70</td>
<td>1.7</td>
<td>0.71</td>
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<tr>
<td>Sz w/deficit</td>
<td>2497</td>
<td>15%</td>
<td>9</td>
<td>98</td>
<td>4.7</td>
<td>0.93</td>
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<tr>
<td>Vomit</td>
<td>2947</td>
<td>20%</td>
<td>34</td>
<td>93</td>
<td>3.0</td>
<td>0.73</td>
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<tr>
<td>HA</td>
<td>3974</td>
<td>18%</td>
<td>46</td>
<td>82</td>
<td>2.9</td>
<td>0.66</td>
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<tr>
<td>Loss of C</td>
<td>174</td>
<td>43%</td>
<td>47</td>
<td>82</td>
<td>2.6</td>
<td>0.65</td>
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<tr>
<td>Acute Onset</td>
<td>887</td>
<td>12%</td>
<td>44</td>
<td>32</td>
<td>0.65</td>
<td>1.7</td>
</tr>
</tbody>
</table>

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<table>
<thead>
<tr>
<th>Signs</th>
<th>n</th>
<th>Heme</th>
<th>Sens</th>
<th>Spec</th>
<th>LR+</th>
<th>LR-</th>
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<tbody>
<tr>
<td>DBP&gt;110</td>
<td>50</td>
<td>46%</td>
<td>48</td>
<td>89</td>
<td>4.3</td>
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<tr>
<td>LoC: Coma</td>
<td>1161</td>
<td>19%</td>
<td>35</td>
<td>94</td>
<td>6.2</td>
<td>0.83</td>
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<tr>
<td>LoC: Drowsy</td>
<td>1161</td>
<td>19%</td>
<td>32</td>
<td>92</td>
<td>2.0</td>
<td></td>
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<tr>
<td>LoC: Alert</td>
<td>274</td>
<td>42%</td>
<td>23</td>
<td>31</td>
<td>0.36</td>
<td></td>
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<tr>
<td>Hemiparesis</td>
<td>3420</td>
<td>15%</td>
<td>63</td>
<td>33</td>
<td>0.96</td>
<td>1.1</td>
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<tr>
<td>Neck stiff</td>
<td>223</td>
<td>43%</td>
<td>20</td>
<td>97</td>
<td>5.0</td>
<td>0.56</td>
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<tr>
<td>Kernig/Brudzinski</td>
<td>50</td>
<td>46%</td>
<td>15</td>
<td>98</td>
<td>8.2</td>
<td>0.87</td>
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<tr>
<td>Cervical bruit</td>
<td>1510</td>
<td>16%</td>
<td>1</td>
<td>93</td>
<td>0.12</td>
<td>1.1</td>
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<tr>
<td>Plantar: Ext x2</td>
<td>370</td>
<td>29%</td>
<td>16</td>
<td>92</td>
<td>1.8</td>
<td></td>
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<tr>
<td>Plantar: Ext x1</td>
<td>370</td>
<td>29%</td>
<td>62</td>
<td>39</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Planter: Flex x2</td>
<td>370</td>
<td>29%</td>
<td>11</td>
<td>74</td>
<td>0.45</td>
<td></td>
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<tr>
<td>AFib on EKG</td>
<td>1087</td>
<td>13%</td>
<td>2</td>
<td>82</td>
<td>0.19</td>
<td>1.2</td>
</tr>
</tbody>
</table>

**Features by location**

Putamen: hemiplegia, hemisensory loss, homonymous hemianopsia, faze palsy, stupor, coma

Cerebellar hemorrhage (dentate) imbalance, headache, neck stiffness, gaze palsy, facial weakness

Thalamic: hemiparesis, hemisensory loss, transient homonymous hemianopsia

Lobar: Often frontal / parietal / occipital
Pontine: Pinpoint paralysis, total paralysis

Comparison to Ischemic Stroke

SCAN rule for mild strokes (NIHSS 3)

Severe HTN: initial BP180/110 (OR 14.5, 1.8-114)

Confusion at onset (OR 8.2, 2.9-23)

Anticoagulation (OR 7.8, 2.2-28)

Nausea & Vomiting (OR 15.7, 5.4-46)

<table>
<thead>
<tr>
<th>SCAN Score</th>
<th>ICH pts (n=32)</th>
<th>All pts (n=614)</th>
<th>p(ICH)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>3%</td>
<td>70%</td>
<td>0.2%</td>
</tr>
<tr>
<td>1</td>
<td>56%</td>
<td>24%</td>
<td>12%</td>
</tr>
<tr>
<td>2</td>
<td>41%</td>
<td>6%</td>
<td>38%</td>
</tr>
</tbody>
</table>

(REF: J Neurol Neurosurg Psychiatry 2010;81:271-275 http://jnnp.bmj.com/content/81/3/271.full)

Children

<table>
<thead>
<tr>
<th></th>
<th>Ischemic</th>
<th>Hemorrhagic</th>
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</thead>
<tbody>
<tr>
<td>Focal limb weak</td>
<td>64</td>
<td>35</td>
</tr>
<tr>
<td>Facial weak</td>
<td>60</td>
<td>35</td>
</tr>
<tr>
<td>Speech disturb</td>
<td>46</td>
<td>13</td>
</tr>
<tr>
<td>Limb ataxia</td>
<td>26</td>
<td>6</td>
</tr>
<tr>
<td>HA</td>
<td>46</td>
<td>73</td>
</tr>
<tr>
<td>Vomit</td>
<td>18</td>
<td>58</td>
</tr>
<tr>
<td>AMS</td>
<td>18</td>
<td>48</td>
</tr>
</tbody>
</table>


Workup

ABC & Vitals

History: Last seen normal, initial symptoms & progression. MS, HA, n/v

PMH: Vasc risk factors (HTN, DM, dyslipid, smoking), coagulopathy, meds (anticoag, antiplt, antiHTN, stimulants), PSH (carotids), dementia, sz., liver

Physical: BP, temp. GCS: If dec 2 en route to ED (>20%), then mortality >75%

Labs: Chem 7 (glu & Cr ~ worse outcome), CBC, PTT/INR, D-dimer, fibrinogen, LFTs, type & screen

EKG, CXR, U/A, Tox to r/o cocaine in young-mid age, urine HCG

STAT Non-contrast head CT

Initial monitoring & management in ICU


Neuroimaging

Non-contrast head CT (w/in min)
Hyperacute blood: bright (unless pt is anemic)

Subacute: isodense, chronically, hypodense ± ring enhancement


Vascular Imaging

Contrast extravasation: higher risk expansion

Secondary: AVM, tumor, moyamoya, CVST

CTA/V vs MRA/V; consider conventional angio if very high suspicion

Radiographic suspicion of secondary ICH: SAH, noncircular hematoma, early edema out of proportion, unusual location, other abnormal structures/mass, sinus abnormal signals

MRI

T1 / T2 / GRE (100% sensitive) (REF: Stroke 2004 Feb;35(2):502-6 http://stroke.ahajournals.org/content/35/2/502.long)

<table>
<thead>
<tr>
<th>Phase</th>
<th>Time</th>
<th>Hgb</th>
<th>T1</th>
<th>T2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hyperacute</td>
<td>&lt;24 h</td>
<td>Oxy-Hgb (intracell)</td>
<td>Iso or hypo</td>
<td>Hyper</td>
</tr>
<tr>
<td>Acute</td>
<td>1-3 d</td>
<td>Deoxy-Hgb (intracell)</td>
<td>Iso or hypo</td>
<td>Hypo</td>
</tr>
<tr>
<td>Early subacute</td>
<td>&gt;3 d</td>
<td>Met-Hgb (intracell)</td>
<td>Hyper</td>
<td>Hypo</td>
</tr>
<tr>
<td>Late subacute</td>
<td>&gt;7 d</td>
<td>Met-Hgb (extracell)</td>
<td>Hyper</td>
<td>Hyper</td>
</tr>
<tr>
<td>Chronic</td>
<td>&gt;14 d</td>
<td>Hemosiderin/Ferritin (extracell)</td>
<td>Iso or hypo</td>
<td>Hypo</td>
</tr>
</tbody>
</table>

Acute -> Sub (e->l) -> chronic (T1, T2): liDee, BiDee, BaBy, DaDa

(REF: http://emedicine.medscape.com/article/1163977-diagnosis#Table1)

(REF: Stroke 1996 Dec;27(12):2321-4 http://stroke.ahajournals.org/content/27/12/2321.long)

Management

Coagulopathies

Reversal of anticoagulation

Repeat CT q12h until stable x2 scans

(REF: http://www2.massgeneral.org/stopstroke/protocolAdultHemorrhage.aspx)

Warfarin or elevated INR

Vit K 10mg IV <1mg/min

FFP 10-20ml/kg (1u ~200ml -> 4-5u) w/designated runner

Risk of ICH doubles for each increase of 0.5 in INR > 4.5, but most ICH occurs w/INR 2-3

INR ~ hematoma growth, prognosis

Repeat INR q4h x24h, then q6h x36h

If INR >1.3 at 4h, repeat VitK & FFP, eval for DIC, consider prothrombin complex concentrate w/blood bank

PCC (prothrombin complex concentrate; factors 9, 2, 10, >7): faster reversal of INR, less volume than FFP, no clear clinical benefit yet

Other factors: Cryoprecipitate (1, 8, 13, vWF), factor 8 & 9

Heparin

d/c heparin, order protamine sulfate based on time since last heparin

<table>
<thead>
<tr>
<th>Time since last heparin</th>
<th>Protamine dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;30 min</td>
<td>1 mg / 100 U</td>
</tr>
<tr>
<td>Time Range</td>
<td>Dose Range</td>
</tr>
<tr>
<td>------------------</td>
<td>--------------------------</td>
</tr>
<tr>
<td>30-60 min</td>
<td>0.5-0.75 mg / 100 U</td>
</tr>
<tr>
<td>60-120 min</td>
<td>0.375-0.5 mg / 100 U</td>
</tr>
<tr>
<td>&gt;120 min</td>
<td>0.25 - 0.375 mg / 100 U</td>
</tr>
</tbody>
</table>

Run slowly in IV (<5 mg/min), total dose <50mg

Monitor for signs of anaphylaxis; the risk is higher in diabetics who have received insulin.

Repeat PTT q1h x4h, then q4h x12h

Low molecular wt Heparin

Protamine reverses only about 60% of the anti-factor Xa activity of LMWH

Enoxaparin: 1 mg protamine / mg enoxaparin

Dalteparin or tinzaparin: 1 mg protamine / 100 anti-Xa IU of dalteparin or tinzaparin

Repeat PTT 2-4 hr later. If elevated, consider repeat half dose

Direct Thrombin Inhibitors

Argatroban, Lepirudin, Bivalirudin, Ximelagatran

No specific antidote

Consider antifibrinolytic agents such as E-aminocaproic acid (10g IV in 250ml NS IV / 1hr), but risk of pathologic thrombosis

Consult blood bank or heme

Thrombolytic Agents

Consult Neurosurgery

If fibrinogen <100 mg/dL, cryoprecipitate 0.15 units/kg to nearest integer

If still bleeding at 1 hr & fibrinogen level <100 mg/dL, repeat cryoprecipitate dose

Give anti-fibrinolytic: eg, aminocaproic acid (Amicar) 5gm bolus i.v. over 15-30 min but has risk of thrombotic complications

Platlets

ASA/antiplt agents: increase mortality, can consider plt transfusion but no clear benefit in obs studies (REF: J Stroke Cerebrovasc Dis. 2009 May-Jun;18(3):221-8). "The usefulness of platelet transfusions in ICH pts w/a history of antplatelet use is unclear & is considered investigational." (REF: http://stroke.ahajournals.org/cgi/content/full/41/9/2108)

Plt <100k/uL: transfuse to >100k

Plt dysfunction (Von Willebrand syndrome, uremic, congenital, recent antiplt): 0.3 µg/kg DDAVP IV / 30 min. Consult heme to dose VWF factor concentrate


Recombinant activated factor VII (rFVIIa)


BP & CPP

Balance bleeding pressure vs cerebral perfusion pressure (CPP)

Autoregulation may be impaired

If using infusion or neuro status deteriorating: place A-line, Central Venous Catheter

Watch for clinical deterioration when lowering BP

AHA recs

If SBP 150-220, lowering to 140 is “probably safe”
SBP >200 mmHg or MAP >150 mmHg: IV drip, BP q5min

SBP >180 mmHg or MAP >130 mmHg w/elevated ICP: IV bolus or drip for CPP >60-80m, monitor ICP

SBP >180 mmHg or MAP >130 mmHg w/o elevated ICP: IV bolus or drip for BP 160/90 or MAP 110, reexamine q15min

**Trials**

**INTERACT:** BP < 140 vs 1999 guidelines improves relative hematoma growth, but only a trend for adjusted or absolute growth, & no clear effect on clinical outcomes (REF: Lancet Neurol. 2008 May;7(5):391-399 [http://linkinghub.elsevier.com/retrieve/pii/S1474-4422(08)70069-3](http://linkinghub.elsevier.com/retrieve/pii/S1474-4422(08)70069-3))

**ATACH (Ongoing until 8/2010):** nicardipine to control SBP 110-140 vs.140-170 vs 170-200 (REF: clinicaltrials.gov)


Mild-moderate benefit on secondary outcomes, but not primary outcome, for SBP<140 vs. liberal guideline care

![Graph](image)

**BP Meds**

<table>
<thead>
<tr>
<th>Drug</th>
<th>IV bolus</th>
<th>IV drip</th>
</tr>
</thead>
<tbody>
<tr>
<td>labetalol</td>
<td>5-20 mg q15min</td>
<td>2 mg/min (max 300 mg/d)</td>
</tr>
<tr>
<td>nicardipine</td>
<td></td>
<td>2.5-15 mg/hr</td>
</tr>
<tr>
<td>esmolol</td>
<td>250 µg/kg load</td>
<td>25-300 µg/kg/min</td>
</tr>
<tr>
<td>enalapril</td>
<td>0.625-5 mg q6h</td>
<td></td>
</tr>
<tr>
<td>hydralazine</td>
<td>5-20 mg q30min</td>
<td>1.5-5 µg/kg/min</td>
</tr>
<tr>
<td>nitrpide</td>
<td></td>
<td>0.1-10 µg/kg/min</td>
</tr>
<tr>
<td>nitroglycerin</td>
<td></td>
<td>20-400 µg/kg/min</td>
</tr>
</tbody>
</table>

**Hypotension**

Diagnose cause

**Fluids**
Monitor CVP

Goal SBP >90 mmHg, MAP >65 mmHg

<table>
<thead>
<tr>
<th>Drug</th>
<th>Mech</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phenylephrine</td>
<td></td>
<td>2-10 µg/kg/min</td>
</tr>
<tr>
<td>Dopamine</td>
<td></td>
<td>2-20 µg/kg/min</td>
</tr>
<tr>
<td>Norepinephrine</td>
<td></td>
<td>0.05-0.2 µg/kg/min</td>
</tr>
</tbody>
</table>

ICP & CPP

ICP can increase by hematoma growth, edema, ischemia, hydrocephalus, or IVH

Goal is to decrease hydrostatic & increase oncotic pressures to keep fluid intravascular

Monitors have not been tested in RCT: CBF, brain tissue oxygenation, intracerebral microdialysis, transcranial Doppler U/S.

ICP (consider monitor/treatment): GCS8, clinical e/o transtentorial herniation, significant IVH, or hydrocephalus

CPP 50-70 may be reasonable depending on autoregulation

EVD reasonable for pt w/decreased LoC

Most studied measures can also cause harm

Head position: Elevated head of bed to 30º, head midline to improve JV outflow (may decrease CPP if hypoBP)

Sedation: propofol, etomidate, or midazolam. Analgesia/antitussive: morphine or alfentanil (balance pain & ICP w/clinical evaluation)

Intraventricular cathteter: monitor/lower ICP, esp in setting of hydrocephalus (may cause colonization 0-19%/meningitis 6-22%)

Neuromuscular blockade: decrease intrathoracic pressure (but associated w/PNA, sepsis; obscures sz)

Mannitol: for serum osm 300-320 mOsm/kg to decrease tissue fluid (but may cause hypovolemia /hyperosmotic state)

Hypertonic saline: no clear guidelines

Hyperventilation: rapid reduction of ICP by decrease CO₂ to 30-35 mmHg (but transient, rebound >6hr, & lowers CBF)

Barbiturate coma: lower refractory intracerebral hypertension by decreasing metabolic activity, reducing CBF & ICP, monitor EEG (may cause hypoBP)

IVH

45% w/spontaneous ICH (secondary/esp BG/thal> primary)

Intraventricular rtPA appears to have a fairly low complication rate, but efficacy & safety of this treatment is uncertain & is considered investigational (REF: Clot Lysis: Evaluating Accelerated Resolution of IVH (CLEAR-IVH) Trial, prospective open-label [http://www.ncbi.nlm.nih.gov/pubmed/19066112])

CLEAR-IVH: 1.0 mg rtPA through an external ventricular drain every 8 h up to 12 doses, or until clot reduction or clinical endpoint is met. CT scans are taken daily to monitor clot resolution & check for unexpected bleeding events. (REF: Acta Neurochir Suppl. 2008;105:217-20. PMID: 19066112) (REF: [http://braininjuryoutcomes.com/bios/clear-about](http://braininjuryoutcomes.com/bios/clear-about))

Neurosurgery

Bolt, EVD, or craniotomy ± hematoma evacuation

Surgery benefit uncertain for most pts

No clear benefit for ultra-early removal, which may increase risk of recurrent bleeding


Unclear role for minimally invasive techniques, which is investigational (could consider endoscopic aspiration if >10ml, <60 yo) (REF: J Neurosurg. 1989; 70: 530–535). RCT is ongoing as of 2010 (REF: MIS+rtPA for ICH Evacuation, MISTIE, clinicaltrials.gov) (REF: http://braininjuryoutcomes.com/studies/mistie)


STICH II did not show a clear statistically significant benefit of surgery (REF: http://www.thelancet.com/journals/lancet/article/PIIS0140-6736(13)60986-1/fulltext)

DVT prophylaxis

Pneumatic compression + elastic stockings (DVT by U/S by day 10: 4.7% combined, 15.9% stockings alone)

If stable x48hr, can use heparin SC (performance measure) (REF: J Neurol Neurosurg Psychiatry. 1991; 54: 466–467 http://www.ncbi.nlm.nih.gov/pmc/articles/PMC488553/)

If DVT/PE: consider IVC filter acutely, need to weigh risks/benefits for anticoagulation vs recurrence after several wk (e.g. amyloid higher risk than HTN, AFib, overall status)

Glucose

DM: regular wt based basal (NPH bid), bolus, & sliding scale regular insulin

Glucose >140 mg/dl: continuous IV insulin or regular sliding scale insulin

Seizures

Clinical sz 2.7-17% (higher w/lobar), most near onset

Cont EEG sz activity 28% <3d, may be nonconvulsive, even w/prophylactic AED

Treat w/AED: clinical sz or MS who have electrographic sz

No prophylactic AEDs (previously considered briefly for lobar ICH)

Continuous EEG probably indicated for MS out of proportion to brain injury

(REF: http://stroke.ahajournals.org/cgi/content/full/41/9/2108#SEC4)

Temp

Temp >37.5º for >24h associated w/IVH (REF: Neurology. 2000; 54: 354–361 http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2117587/)

Control sources of fever (risk of PNA 2/2 aspiration, UTI 2/2 catheter)

Maintain temperature 38º using PO/PR acetaminophen 650 mg q6h

In the setting of poor airway control or if temperature remains elevated despite acetaminophen, consider external cooling (reduces ICP but >1-2d may be associated w/pulmonary, infectious, coagulation, & electrolyte problems)

Early mobilization & rehab

Prognosis


Best predictors of Death <30d = Volume ICH (ABC/2), GCS

Independent Functional Outcome = 12-40%

Associated w/better outcomes: cortical location, mild neurological dysfunction, & low fibrinogen levels

Goals of Care by Guidelines

(REF: http://stroke.ahajournals.org/cgi/content/full/41/9/2108#SEC9)

Aggressive full care early after ICH onset & postponement of new DNR orders until at least the second full day of hospitalization is probably recommended.

Pts who are DNR should receive all other appropriate medical & surgical interventions unless otherwise explicitly indicated

ICH Score

(REF: Stroke 2001 Apr;32(4):891-7; http://stroke.ahajournals.org/cgi/content/short/32/4/891)


GCS (3-4 +2, 5-12 +1, 13-15 +0)

ICH (30 ml +1, <30 ml +0)

Intraventricular ext (present +1, absent +0)

Infratentorial origin (yes +1, no +0)

Age (80 +1, <80 +0)

<table>
<thead>
<tr>
<th>Score</th>
<th>Death 30d</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>0%</td>
</tr>
<tr>
<td>1</td>
<td>13%</td>
</tr>
<tr>
<td>2</td>
<td>26%</td>
</tr>
<tr>
<td>3</td>
<td>72%</td>
</tr>
<tr>
<td>4</td>
<td>97%</td>
</tr>
<tr>
<td>5</td>
<td>100%</td>
</tr>
</tbody>
</table>

FUNC Score

(REF: Stroke. 2008;39:2304; http://stroke.ahajournals.org/cgi/content/short/39/8/2304)

(REF: http://www2.massgeneral.org/stopstroke/funcCalculator.aspx)

ICH volume (<30ml +4, 30-60 +2, >60 +0)

Age (<70 +2, 70-79 +1, 80 +0)

ICH Location (Lobar +2, Deep +1, Infratentorial +0)

GCS (9 +2, 8 +0)

Pre-ICH cog impair (No +1, Yes 0)

<table>
<thead>
<tr>
<th>Func independent (GOS4)</th>
<th>Entire cohort</th>
<th>Survivors only</th>
</tr>
</thead>
<tbody>
<tr>
<td>%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>FUNC score</td>
<td></td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>82%</td>
<td>95%</td>
</tr>
<tr>
<td>9-10</td>
<td>66%</td>
<td>75%</td>
</tr>
<tr>
<td>8</td>
<td>42%</td>
<td>48%</td>
</tr>
<tr>
<td>5-7</td>
<td>13%</td>
<td>29%</td>
</tr>
<tr>
<td>0-4</td>
<td>0%</td>
<td>0%</td>
</tr>
</tbody>
</table>

Followup

Recurrence 2.1-3.7%/pt-yr, usually >ischemic risk

Risks: age, anticoag, previous bleed, apoE2 or 4, GRE microbleeds; BP, alcohol

BP: ICH 2% -> 1% w/11 mmHg decrease SBP (REF: Stroke 2004 Jan;35(1):116-21, PROGRESS trial). Goal <140/90 (or <130/80 w/DM or CKD)

Stop smoking, heavy alcohol, & stimulants


Statin: Some suggested increased risk in IPH, but followup data supports continued use w/OR for 90d mortality 0.5 & for good outcome 2 (REF: http://www.neurology.org/content/76/18/1581.abstract)

**Restarting Anticoagulation**

Consider individual risk/benefits: (re)bleeding (w/anticoag) vs. thromboembolic disease/stroke (w/o anticoag)

Favor stopping anticoag: lobar ICH, presence of multiple microbleeds on MRI, low CHADS2 score, & difficulties controlling INR (REF: Controversies in Stroke: The Dilemma of Resuming Anticoagulation After Intracranial Hemorrhage Little Evidence Facing Big Fears. http://stroke.ahajournals.org/content/early/2011/11/03/STROKEAHA.111.631689.cita)

Favor restarting anticoag: deep ICH, secondary prevention, high CHADS2 score, mechanical valve, or hypercoagulable state (REF: Controversies in Stroke: The Dilemma of Resuming Anticoagulation After Intracranial Hemorrhage Little Evidence Facing Big Fears. http://stroke.ahajournals.org/content/early/2011/11/03/STROKEAHA.111.631689.cita)


Prosthetic valves: usually needs to be restarted


**Examples**


Favors late:
Modeled warfarin RR 5 for ICH, RR 0.1 for ischemic event (REF: Stroke. 2010 Dec;41(12):2860-6. Optimal timing of resumption of warfarin after intracranial hemorrhage. PMID: 21030703. http://stroke.ahajournals.org/content/41/12/2860.full)

Favors early:

Freq of ICH/TE events:

Timing of warfarin restart by ICH/TE events:
Predict rebleed: younger, trauma, SDH, cardiac indication, higher intensity anticoag, warfarin + ASA (OR 2.8)

Predict thromboembolism (50% strokes): younger, spinal multiple hemorrhages, nontraum, lower intensity anticoag. Mitral replacement 2x Aortic


Other series


Retrospective compare ±warfarin (median 10d): 5/23 on warfarin had ICH, 5/29 not on warfarin has 5 TE events


Cerebral Amyloid Angiopathy (CAA)

BostonCriteria

Definite CAA

Full postmortem examination showing severe CAA
Probable CAA w/Supporting Pathology

Evacuated specimen showing CAA

Probable CAA (age >55)

Full clinical evaluation w/MRI

Multiple lobar/corticosubcortical bleeds, no other cause

Possible CAA (age >55)

Full clinical evaluation w/MRI

Single lobar/corticosubcortical bleed, no other cause

MRI

(REF: http://www.neurology.org/content/77/15/1446.full)

Epidemiology


Relationships were not as strong or distinct with further f/u (REF: Poels. Prevalence and risk factors of cerebral microbleeds: an update of the Rotterdamscan study. Stroke. 2010 Oct;41(10 Suppl):S103-6. PMID: 20876479. http://stroke.ahajournals.org/content/41/10_suppl_1/S103.long)

Age dependent: 2.3% between 65 & 74, 12.1% above 85

Pathophysiology

Usually cerebral/lobar


Deposition of congophilic material in small / medium arteries, same amyloid as Alzheimer’s

Increases chance of hemorrhage in cortex / subcortical WM, cluster in same lobe (temporal >> occipital >> frontal)

Microhemmorhage detected by gradient echo

Can manifest as transient neurologic symptoms


Management

Diagnosed by microhemmorhage on MRI

Reduce ASA dose (recurrence of bleed)

Subarachnoid Hemorrhage (SAH)

Definition
Bleeding into subarachnoid space (between the arachnoid & the dura, where arteries run)

Epidemiology
10% of all strokes. 5-10% of people have small, saccular aneurysms, & subarachnoid hemorrhage rate is 3-25 / 100K (REF: Connolly. Guidelines for the Management of Aneurysmal Subarachnoid Hemorrhage. Stroke. 2012 May 3. PMID: 22556195.http://stroke.ahajournals.org/content/43/6/1711.full)

Pathophysiology
Rupture of artery due to aneurysm / vessel / bleeding disorder
Blood spreads quickly in the CSF, lasts a few sec, common rebleeding

Etiologies
Rupture of saccular aneurysms at branches of major vessels (85%)
Occult aneurysms may be missed initially

Nonaneurysmal (15%) may originate in perimesencephalic artery, arteriovenous malformations, intracranial dissection (4%), venous thrombosis, sickle cell disease, bleeding disorder, pituitary apoplexy, trauma, cocaine, tumor, Moyamoya, vasculitis, reversible vasoconstriction syndrome, reversible posterior leukoencephalopathy syndrome

Mycotic aneurysm: arterial dilatation due to destruction of vessel wall by infection

Natural History

Figure 1. Intervals between Successive Bleeding Episodes in Patients with More than One Hemorrhage.
Figure 2. Intervals between Subarachnoid Hemorrhage (SAH) and Onset of a Preoperative Focal Neurologic Deficit.

Figure 3. Probability of Survival after First Subarachnoid Hemorrhage from Intracranial Aneurysm for Residents of Rochester, Minnesota, for the Period 1955 through 1966.

Risk factors

(REF: Stroke. 2005 Dec;36(12):2773-80 http://stroke.ahajournals.org/content/36/12/2773.long)

(REF: Stroke. 2012; 43: 1711-1737 http://stroke.ahajournals.org/content/43/6/1711.full)

Smoking: RR 2-5

Hypertension: RR 2.5 (REF: )

Genetic risk: Polycystic kidney disease, type IV Ehlers-Danlos, glucocorticoid-remediable aldosteronism, familial intracranial aneurysm syndrome (RR 4.2), family hx

Stimulants: cocaine & phenylpropanoamine

Sex: Female (50-60%)

Age: highest prevalence 40-60 but long tails

Alcohol: OR 1.5, RR 2.1

African-American > Caucasian

Anticoagulation: increases severity of bleeds

Antiplt may increase risk: new ASA (OR 2.52; 1.37-4.62); long term diprydamole (2.09; 1.04-4.23); inconclusive for clopidogrel (REF: J Thromb Haemost 2010 Jul; 8:1468.)

**Clinical features**

High level of suspicion for SAH w/acute onset of severe headache, possibly 25% may have SAH (REF: Lancet 1994 Aug 27;344(8922):590-3 http://linkinghub.elsevier.com/retrieve/pii/S0140-6736(94)91970-4)

Classic: acute onset, meningismus, photophobia, nasea (REF: utdol.com)

EMS should assume SAH if 1 of HA, abnormal LoC, & vomiting


Sentinel headaches can be reported by 10-43% pts w/SAH retrospectively vs ~5% matched controls (REF: Cephalalgia 2003 Dec;23(10):935-41. PMID 14984225 http://cep.sagepub.com/content/23/10/935.long)

High risk clinical characteristics for subarachnoid haemorrhage in pts w/acute headache: prospective cohort study

<table>
<thead>
<tr>
<th>Subarachnoid haemorrhage</th>
<th>P value</th>
<th>(n=103)</th>
</tr>
</thead>
<tbody>
<tr>
<td>No (n=1869)</td>
<td>Yes (n=130)</td>
<td></td>
</tr>
<tr>
<td>Mean age (years)</td>
<td>42.6</td>
<td>54.4</td>
</tr>
<tr>
<td>Female</td>
<td>60.6 (1133)</td>
<td>56.9 (74)</td>
</tr>
<tr>
<td>Mean time from onset to peak (minutes)</td>
<td>9.2</td>
<td>3.4</td>
</tr>
<tr>
<td>Mean pain severity at peak (0-10)</td>
<td>8.6</td>
<td>9.3</td>
</tr>
<tr>
<td>Onset during exertion</td>
<td>10.7 (200)</td>
<td>23.1 (30)</td>
</tr>
<tr>
<td>Onset during sexual activity</td>
<td>6.0 (112)</td>
<td>5.5 (7)</td>
</tr>
<tr>
<td>Headache awoke patient from sleep</td>
<td>19.3 (361)</td>
<td>10.8 (14)</td>
</tr>
<tr>
<td>Worst headache of life</td>
<td>77.5 (1448)</td>
<td>93.1 (121)</td>
</tr>
<tr>
<td>Loss of consciousness</td>
<td>4.5 (84)</td>
<td>16.9 (22)</td>
</tr>
<tr>
<td>Witnessed loss of consciousness</td>
<td>2.5 (47)</td>
<td>11.5 (15)</td>
</tr>
<tr>
<td>Needed to rest</td>
<td>24.0 (449)</td>
<td>43.9 (57)</td>
</tr>
<tr>
<td>Complaint of neck stiffness or pain</td>
<td>30.9 (578)</td>
<td>71.1 (92)</td>
</tr>
<tr>
<td>Vomiting</td>
<td>26.3 (492)</td>
<td>58.6 (76)</td>
</tr>
<tr>
<td>Ambulance</td>
<td>16.7 (312)</td>
<td>56.9 (74)</td>
</tr>
<tr>
<td>Emergency department transfer</td>
<td>7.9 (148)</td>
<td>18.5 (24)</td>
</tr>
</tbody>
</table>


**Workup**
Non-contrast CT

CT should be performed for suspected SAH

MRI (T2/FLAIR) can be as sensitive as CT but is less practical

CT most sensitive early: 98-100% <12hr, 93% @24hr, 57-85% @6d (REF: Acad Emerg Med 1996 Sep;3 (9):827-31)

Newer CT’s may be even more sensitive (100% @1-5d, 96% after), recommended 1-3d, but results have not been replicated in community EDs (REF: Neurosurgery 2010 May; 66:900.)

<table>
<thead>
<tr>
<th></th>
<th>Sens</th>
<th>Spec</th>
<th>LR+</th>
<th>LR-</th>
<th>PPV</th>
<th>NPV</th>
</tr>
</thead>
<tbody>
<tr>
<td>All pt</td>
<td>92.9 (89.0-95.5)</td>
<td>100</td>
<td>Inf</td>
<td>0.07 (0.05-0.11)</td>
<td>100</td>
<td>99.4 (99.1-99.6)</td>
</tr>
<tr>
<td>CT 6 h</td>
<td>953</td>
<td>100</td>
<td>97.0-100.0</td>
<td>100</td>
<td>99.5-100</td>
<td></td>
</tr>
<tr>
<td>CT &gt;6 h</td>
<td>2179</td>
<td>85.7 (78.3-90.9)</td>
<td>100</td>
<td>0.14 (0.14-0.17)</td>
<td>100</td>
<td>99.2 (98.7-99.5)</td>
</tr>
</tbody>
</table>

CT 100% sens & NPV <6h (REF: Perry JJ et al. Sensitivity of computed tomography performed within six hours of onset of headache for diagnosis of subarachnoid haemorrhage: Prospective cohort study. BMJ 2011 Jul 18; 343:d4277. http://dx.doi.org/10.1136/bmj.d4277)

LP

If high suspicion, LP strongly recommended if CT negative (REF: J Neurol Neurosurg Psychiatry 1990; 53:365 http://jnnp.bmj.com/content/53/5/365.full.pdf+html).


High pressure, high RBC

Sens 93%, spec 95%


Distinguishing from traumatic tap (or other conditions)

RBC decrease from tube 1 -> 4: Not reliable unless final count wnl


CTA/MRA

Identify etiology

Useful to detect aneurysms >3mm

<table>
<thead>
<tr>
<th></th>
<th>CTA</th>
<th>MRA</th>
</tr>
</thead>
<tbody>
<tr>
<td>5mm</td>
<td>95-100%</td>
<td>85-100%</td>
</tr>
<tr>
<td>&lt;5mm</td>
<td>65-83%</td>
<td>56%</td>
</tr>
</tbody>
</table>

Consider premed for pt w/renal insufficiency (NS, bicarb, or acetylcysteine)

If angio negative – Consider repeating 4-14d if no aneurysm found initially (REF: utdol.com)

Immediatley reimage after surgery to ensure complete obliteration, as well as delayed imaging

Angiography is still gold standard

Grading

Hunt & Hess
1. Asymptomatic or mild headache & slight nuchal rigidity
2. Moderate to severe headache, stiff neck, no neurologic deficit except cranial nerve palsy
3. Drowsy or confused, mild focal neurologic deficit
4. Stupor, moderate or severe hemiparesis
5. Deep coma, decerebrate posturing

The grade is advanced one level for the presence of serious systemic disease (hypertension, diabetes, severe arteriosclerosis, chronic pulmonary disease) or vasospasm on angiography.

**Fisher Scale**

<table>
<thead>
<tr>
<th>Group 1</th>
<th>No blood detected</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 2</td>
<td>Diffuse deposition or thin layer w/all vertical layers of blood (in interhemispheric fissure, insular cistern, or ambient cistern) &lt;1 mm thick</td>
</tr>
<tr>
<td>Group 3</td>
<td>Localized clots and/or vertical layers of blood 1 mm thick</td>
</tr>
<tr>
<td>Group 4</td>
<td>Intracerebral or intraventricular clots w/diffuse or no subarachnoid blood</td>
</tr>
</tbody>
</table>

**Management**

(REF: Decision Making in Neurocritical Care By Jennifer Frontera)

**Airway, Breathing, Circulation**

If intubating, use RSI & place gastric tube to reduce aspiration.

Tox screen if younger pt or h/o abuse.

General care: admit ICU, cardiac & hemodynamic monitoring, bed rest, stool softeners, analgesia, pneumatic compression stockings.

BP: Unclear optimal BP (SBP <160 mmHg reasonable). Balance stroke, HTN rebleed, & CPP. Can use labetalol, esmolol, or nicardipine as vasodilators, but not nitroprusside (can raise ICP, cyanide toxicity). Can use norepi or phenylephrine as vasopressors. Use titrable drip until aneurysm obliteration. Consider hypertension for delayed cerebral ischemia.


**Metabolic:** Correct hypoxia, hyperthermia, hyperglycemia, electrolytes (esp hypoMg, hypoNa)

Nimodipine (60mg q4h po x21d) decreases infarct or poor outcomes (~20% vs ~33%). NNT 13. (REF: BMJ 1989 Mar 11;298(6674):636-42). Consider 30mg for SBP 120-140, hold for SBP 120 mmHg.


Consider aminocaproic acid if<3d, aneurysm treatment will be delayed >12h, & no h/o stroke, MI, PVD, or abnormal EKG (4g IV x1h then 1g IV q1h, hold 1-3h prior to angio). No benefit from clazosentan (endothelin receptor antag) (REF: Macdonald RL et al. Clazosentan, an endothelin receptor antagonist, in pts with aneurysmal subarachnoid haemorrhage undergoing surgical clipping: A randomised, double-blind, placebo-controlled phase 3 trial (CONSCIOUS-2). Lancet Neurol 2011 Jul; 10:618 http://www.thelancet.com/journals/laneur/article/PiIS1474-4422(11)70108-9/fulltext).

Stop antithrombotics, reverse anticoagulation.

**Surgery**

Monitor ICP: Consider ventriculostomy.

Sz: consider ppx in immediate posthemorrhagic period (fosphenytoin 20mg/kg IV load, then phenytoin 100mg IV q8h).

Endovascular coiling or surgical clipping should be performed early on ruptured aneurysms.

Consider transfer to high volume center w/endovascular teams

Endovascular coiling

(REF: Stroke. 1999; 30: 470–476 http://stroke.ahajournals.org/content/30/2/470.full)

Consider coiling if aneurysm is amenable to both coiling & clipping.


Rate depends on aneurysm size, shape, h/o SAH, & partial occlusion (Rebleed 1.1% complete vs 21% partial) (REF: J Neurosurg. 2003; 98: 959–966 http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3490167/)


Requires followup imaging, eg angio in 6 mo (recurrence 33.6%, mean 12.3 mo) (REF: Stroke. 2003; 34: 1398–1403 http://stroke.ahajournals.org/content/34/6/1398.full)

Surgical clipping


Ligation of parent artery may lower risk of rebleed, but likelier worse than surgical clipping

Complications

Rebleed

5% of pts <24hr (esp 2-12hr), 1%/d x1mo, 3%/yr after 3mo

Predicted by severity of initial bleed, interval to admission, high BP, worse neuro status, large aneurysm, high Hunt-Hess grade

Vasospasm


Can monitor w/transcranial Doppler (TCD) for severe spasm (REF: Neurology. 2004; 62: 1468–1481 http://www.neurology.org/content/62/9/1468.long)

Can also consider MRI, or angio

Consider balloon angioplasty for proximal vessels (REF: Stroke. 2000; 31: 111–117 http://stroke.ahajournals.org/content/31/1/111.full)

Hydrocephalus

20-30% of pts <72 hr

Often accompanied by IVH

Acute symptomatic hydrocephalus: EVD or lumbar drain

No clear benefit to weaning EVD>24h.

Chronic symptomatic hydrocephalus: permanent CSF diversion

Ventriculostomy can be beneficial in pts w/ventriculomegaly & diminished level of consciousness after acute SAH, but no clear benefit of routine fenestration of lamina terminalis

Seizure

4-21% of pts (REF: Neurology. 2000; 55: 258–265 [http://www.neurology.org/content/55/2/258.long](http://www.neurology.org/content/55/2/258.long))


May be nonconvulsive, be particularly wary in comatose pts

Risks: MCA aneurysm, intraparenchymal hematoma, infarcts, HTN


Overal benefit not clear, guideline is to consider in immediate posthemorrhagic period but not to use routine for long-term w/o clearer risk factors

Hyponatremia

10-30% of pts

Risks: poor clinical grade, AComm aneurysms, hydrocephalus

Avoid hypovolemia & hypotonic fluids

Consider fluid checks, wts, CVP to monitor volume status

Treat first w/isotonic saline

Reasonable to correct hypoNa w/fludrocortisones or hypertonic saline

Other Complications

Ischemia: Consider hypertension for delayed cerebral ischemia

Increased intracranial pressure

Cardiac abnormalities

Hypothalamic dysfunction / pituitary dysfunction

Prognosis

A variety of scales grade clinical status; conflicting usefulness

GCS: Level of alertness

World Federation of Neurological Surgeons: GCS + motor scales

Rate of new aneurysms = 1-2%/yr (REF:)

Perimesencephalic nonaneurysmal subarachnoid hemorrhage has better outcome (REF: utdol.com)
Subdural Hematoma (SDH)

(REF: utdol.com)

Definition
Blood between dura & arachnoid layers

Epidemiology
Acute hematomas: middle age, men
Chronically: older adults
Complicated: 11% of mild head injuring, 20% of severe head injuries

Pathophys
Tearing of bridging veins between dura & arachnoid, resulting in low pressure buildup of blood, usually stopped by rising ICP or compression of clot
Can also be caused by arterial tear (20-30% of SDH)
Low CSF pressure can contribute

Risks/Etiologies
Head trauma: falls, MVA, assaults
Cerebral atrophy: elderly, alcohol abuse, h/o TBI
Antithrombotic agents: ASA, warfarin (esp INR > 4)
SAH/aneurysms: 0.5-7.9% of SAH

Other
AVM
Dural mets
Coagulopathy
Meningioma

Chronic Changes
Collagen & fibroblasts spread to form outer & inner membranes, encapsulating clot in ~2 wks
SDH may liquefy to form hygroma w/calcified membranes
>1/2 liquefy & tend to enlarge by recurrent/acute-on-chronic bleeding or osmotic draw of water into high protein hygroma
Subdural hygromas may also be a consequence of traumatic brain injury followed by brain atrophy, dehydration, or decreased ICP (brain pulls arachnoid, dura remains attached to skull, pseudmembranes & vessels form)

Presentation
Acute: transient lucid interval followed by ICP symptoms -> coma
Hypoperfusion can cause infarction, esp in posterior fossa
Chronic (can be wks): HA, light-headedness, cognitive impairment, apathy, somnolence, seizures.
Global > focal deficits

Focal deficits may be ipsi or contra

Transient Neurologic Deficits


Workup

Non-contrast CT

Crescent shaped lesion

Can cross sutures, but is limited by dural attachments

Acute=hyperdense, subacute to chronic=iso/hypodense

B SDH easier to miss

r/o other bleeds, TBI, fractures

Left – acute, Right – chronic

(REF: http://radiopaedia.org/articles/subdural-haemorrhage)

MRI

T1 more sensitive than CT (97 vs 79%) (REF: AJR Am J Roentgenol 1988 Mar;150(3):673-82 http://www.ajronline.org/doi/abs/10.2214/ajr.150.3.673)

Acute: hypointense on T2 (deoxyHgb)

Wks: bright on T1 & T2 (metHgb)

Months: dark on T1 (hemosiderin)

Other

Angiography may be helpful in cases where no trauma can be identified

LP contraindicated due to risk of herniation w/space-occupying lesion

Management

Deterioration may develop within hours to wk

Reverse Anticoagulation

Weigh risks & benefits of d/c’ing anticoag & antiplt

Can use prothrombin complex concentrates, recombinant human factor VIIa, or FFP

Vit K 10mg slow IV

Goal INR <1.2 for surgery


<table>
<thead>
<tr>
<th>Severity of bleed</th>
<th>Risk of thrombosis</th>
<th>Target INR</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Condition</td>
<td>Grade</td>
<td>Value</td>
</tr>
<tr>
<td>---------------------------------</td>
<td>---------------</td>
<td>-------</td>
</tr>
<tr>
<td>moderate</td>
<td>high</td>
<td>2-2.1</td>
</tr>
<tr>
<td>serious</td>
<td>moderate</td>
<td>1.5</td>
</tr>
<tr>
<td>serious or life-threatening</td>
<td>low</td>
<td>1.0</td>
</tr>
</tbody>
</table>

Dose (ml plasma or IU PCC) = (target INR as% - present INR as%) x wt kg

<table>
<thead>
<tr>
<th>INR</th>
<th>~% plasma</th>
</tr>
</thead>
<tbody>
<tr>
<td>5.0</td>
<td>5</td>
</tr>
<tr>
<td>4.0-4.9</td>
<td>10</td>
</tr>
<tr>
<td>2.6-3.2</td>
<td>15</td>
</tr>
<tr>
<td>2.2-2.5</td>
<td>20</td>
</tr>
<tr>
<td>1.9-2.1</td>
<td>25</td>
</tr>
<tr>
<td>1.7-1.8</td>
<td>30</td>
</tr>
<tr>
<td>1.4-1.6</td>
<td>40</td>
</tr>
<tr>
<td>1.0</td>
<td>100</td>
</tr>
</tbody>
</table>

Neurosurgery


Craniotomy, decompressive craniectomy, or burr holes ± ligation of bleeding vessel

Emergency: mortality 30-47% w/in 2-4hr of deterioration vs 80-90% if delayed

Indications

Clot thickness >10 mm (REF: Acta Neurochir (Wien). 1993;121(3-4):100-8)
GCS worse by 2 from injury to admission
Asymmetric or fixed & dilated pupils
Nonoperative management

For neurologically stable pts w/o surgical indications
Admit to ICU w/ICP monitoring if GCS 8
Manage ICP (but no role for steroids)
If ICP consistently >20 mmHg -> surgery
FollowupCT w/in 6-8 hr


Chronic SDH

Consider surgery w/mod-severe or progresive impairments & potential for recovery
Can try 1 burr holes w/flexible catheter (Jackson-Pratt drain) x several days until drainage subsides (REF: Mayer, S, Rowland, L. Head injury. In: Merritt's Neurology, Rowland, L (Ed), Lippincott Williams & Wilkins, Philadelphia 2000. p.401)
Drain can prevent recurrence (9% vs 24%) & reduce 6 mo mortality (9% vs 18%) (REF: Lancet. 2009 Sep 26;374(9695):1067-73)
If rebleeds, can do craniotomy w/resection of membranes around clot

AED

Prognosis

Mortality 40-60%

GCS=3 (93% mortality), GCS=7 (0% mortality) (REF: Neurol Med Chir (Tokyo) 1993 Jan;33(1):13-8 https://www.jstage.jst.go.jp/article/nmc1959/33/1/33_1_13/_pdf)


Other likely poor factors: associated intra & extracranial injuries, thickness/vol, midline shift, reduced patency of basal cisterns

Epidural Hemorrhage (EDH)

Definition

Bleeding of major arteries outside the dural space

Epidemiology

1-4% of severe traumatic head injury cases, peak 20-30 y, rare after 50-60, skull fracture present 75-90%

Pathophys

Tearing of major arteries (most often middle meningeal) in the setting of linear acceleration of the skull / or fracture

Etiologies

Trauma (most common)
Infection, abscess
Coagulopathy
Epidural AVM
Neurosurgery, spinal procedure
Pregnancy, sickle cell disease, lupus, heart surgery, paget’s

Differential Diagnosis

Subdural, subarachnoid

Clinical features

At onset: Headache, nausea, vomiting, drowsiness, confusion, decreases consciousness, aphasia, seizures, hemiparesis

Can have lucid interval, & then progress to coma. Also can have transient LOC initially

With progression, elevated ICP, dilated pupil, cushing reflex (bradycardia, hypotension, respiratory depression)

Diagnostic studies

LP Contraindicated
Non-contrast head CT:

Lens shaped area of bright blood, respects suture lines

Estimate volume by ABC/2 (Diameter Width number of slices slice thickness)

Can be negative in anemia, hypotension, early scanning, or venous bleeding
MRI, more sensitive
Initially, hypotense on T2, then bright on T1/T2, then hypointense on T1.
Only study for spine EDH

Angiography
Best for AVM

Management

Urgent neurosurgical consultation
OR if declining neurological status, pupillary signs, CT findings
Consider non-operative management w/volume < 30, midline shift < 5, & no deficits

Close observation
Reverse anticoagulation
Consider hemostatic therapy
No steroids

Prognosis

GCS
Old age (>31)
Time between decline & surgery
ICP
Large hematoma, midline shift, mixed clot density,
Presence of other lesions