ICH

Introduction

- Intracerebral hemorrhage (ICH) is a hemorrhage within the brain parenchyma
- Commonly referred to as “Hypertensive hemorrhage” in the past
- Second most common form of stroke, but most deadly

Epidemiology

- Incidence : second most common form of stroke (15 – 30%)
  - 12 – 15 cases per 100000 /yr
- Recent studies show approximately twice the incidence of SAH
- Incidence increase in age ≥ 55
- Over 20% of patient deteriorate by ≥ 2 between first EMS and then E/R
- 15 – 23% deteriorates further within the first few hours in the hospital

Risk factors

- Age : the incidence increase significantly after age 55
  - Doubles with each decades of age until age > 80, where incidence is 25 times that during previous decades
- Gender : more common in men
- Race : Blacks more than whites, may also be higher in Asians
- Previous stroke : increase risk to 23:1

Risk factor II

- Alcohol consumption : recent use and chronic use
- Cigarette smoking : increase the risks of SAH and ischemic infarction but probably does not increase the risks of ICH
- Street drugs : cocaine, amphetamines, phencyclidine
- Liver dysfunction : hemostasis may be impaired on the basis of thrombocytopenia, reduced coagulation factors and hyperfibrinolysis

Alcohol consumption

e 84.1 Relative risk of ICH with EtOH consumption

od prior to ICH	Amount[a] (g EtOH)	Relative risk
hours	41–120	4.6
	>120	11.3
eek	1–150	2.0
	151–300	4.3
	>300	6.5

tandard drink= 12 g EtOH

Location of ICH

- Common arterial feeders of ICH

- Lenticulostriates : the source of putaminal hemorrhage
- Thalamoperforators
- Paramedian branches of BA
- Intraventricular hemorrhages : occurs in 45% of sICH and is an independent risk factors for worse outcome

Common sites for ICH

ble 84.2 Common sites for ICH (modified[15])

	Location
0	striate body (basal ganglia); putamen most common; also includes: lenticular nucleus, internal capsule, globus pallidus
5	thalamus
0–15	pons (≈ 90% of these are genuinely hypertensive)
0	cerebellum
0–20	cerebral white matter
-6	brainstem

Lobar hemorrhage

- Account for 10 -32% of all nontraumatic ICHs
- Are more likely to be associated with structural abnormalities then deep hemorrhages
- Have a more benign outcome than ganglionic-thalamic ICHs

Etiology of lobar ICHs

- Extension of a deep hemorrhage
- Cerebral amyloid angiopathy
  - The most common causes of lobar ICH in elderly normotensive pts
- Trauma
- Hemorrhagic transformation of an ischemic infarct
- Hemorrhagic tumor
- Cerebrovascular malformation
- Rupture of an aneurysm
- Idiopathic

Internal capsule hemorrhages - Etiologies

- Hypertension
  - Acute hypertension : as may occur in eclampsia
  - Chronic hypertension : degenerative changes within blood vessels
- Associated with acutely increased CBF, esp. areas previously rendered ischemic
  - Following carotid endarterectomy
  - Following repair of congenital heart defects in children
  - Previous stroke
  - Migraine
  - Following surgery to remove an AVM

Etiologies II

- Vascular anomalies
  - AVM
  - Aneurysm rupture
  - Venous angioma rupture
- Arteriopathies
  - Amyloid angiopathy
  - Fibrinoid necrosis
  - Lipohyalinosis
  - Cerebral arteritis
- Brain tumor
- Coagulation or clotting disorders

Etiologies III

- CNS infection
- Venous or dural sinus thrombosis
- Drug related
- Posttraumatic
- Pregnancy related
- Postoperative
- Idiopathic

Cerebellar hemorrhage etiologies

- Etiologies are similar to ICH of any location, however, some nuances
- Hypertension is a factor in up to 2/3 of cerebellar hemorrhages
- AVM is a consideration, aneurysm is very rare
- May be related to recent previous spinal or supratentorial surgery

Cerebral amyloid angiopathy

- Pathological deposition of beta amyloid protein within the media of sma meningeal and cortical vessels without evidence of systemic amyloidosi
- Should be suspected in patient with recurrent hemorrhages that are lob in location
- Incidence increases with age : CAA present in about 50% of those > 70 y

Table 84.3 Criteria for the diagnosis of cerebral amyloid angiopathy (CAA)[73]

Diagnosis	Criteria
definite CAA	full postmortem exam showing all 3 of the following: a) lobar, cortical, or corticosubcortical hemorrhage b) severe CAA c) absence of another diagnostic lesion
probable CAA with supporting pathological evidence	clinical data & pathological tissue showing all 3 of the following: a) lobar, cortical, or corticosubcortical hemorrhage b) some degree of vascular amyloid deposition in specimen c) absence of another diagnostic lesion
probable CAA	clinical data and MRI findings showing all 3 of the following: a) age ≥ 60 yrs b) multiple hemorrhages restricted to the lobar, cortical, or corticosubcortical region c) absence of another cause of hemorrhage[a]
possible CAA	clinical data and MRI findings: a) age ≥ 60 yrs b) single lobar, cortical, or corticosubcortical hemorrhage without another cause[a], or multiple hemorrhages with a possible but not a definite cause[a], or with some hemorrhages in an atypical location (e.g. brainstem)

[a] e.g. excessive anticoagulation (INR > 3.0), head trauma, ischemic stroke, CNS tumor, cerebrovascular malformation, vasculitis, or blood dyscrasia

Hemorrhagic brain tumors

- Malignant tumor most commonly associated with ICH
  - Glioblastom
  - Lymphoma
  - Metastatic tumors
    - Melanoma ≈ 40% hemorrhage
    - Choriocarcinoma ≈ 60% hemorrhage
    - Renal cell carcinoma
    - Bronchogenic carcinoma ≈ 9% hemorrahge
- Malignant tumors that hemorrhage less commonly
  - Medulloblastom
  - Gliomas

- Some benign brain tumors associated with ICH
  - Meningiomas : intratumoral, subdural, and nearby parenchymal hemorrahge
  - Pituitary adenoma
  - Oligodendroglioma
  - Hemangioblastom
  - Vestibular schwannoma
  - Cerebellar astrocytoma

Anticoagulation preceding ICH

- 10% of patient on warfarin develop a significant bleeding complication per year, including ICH (65% mortality)

- The risks of ICH in patients treated with warfarin for a-fib 0-0.3%/year
  - 1.8%/year in elderly group

- The risk of hemorrhagic complication was increased with the length and also the variability of the PT, and during the first 3 month of anticoagulation.

Clinical

- The neurologic deficit with ICH is characterized by a smooth progressive onset over minutes to hours
  - Unlike embolic/ischemic stroke where deficit is maximal at onset

- Severe headache, vomiting and alternations in level of consciousness may be more common

Prodrome

- TIA-like symptoms may precede lobar hemorrhages in pts with CAA
  - Up to ≈ 50% for whom a complete history is obtainable

- Unlike typical TIAs, these usually consist of numbness, tingling, or weakness that gradually spreads in a manner reminiscent of a Jacksonian-march and my spill-over vascular territories

Putaminal ICH

- The most common site for ICH

- Smooth gradual deterioration in 62%
  - Maximal deficit at onset in 30%
- Never fluctuating
- Contralateral hemiparesis, may progress to hemiplegia or even coma

- H/A in 14% of onset, no H/A at any time in72%

Thalamic hemorrhage

- Classically contralateral hemisensory loss
- Also hemiparesis when the internal capsule in involved
- Extension into upper brainstem
  - Vertical gaze palsy, retraction nystagmus, skew deviation, loss of convergence, ptosis, miosis, anisocoria +- unreactive pupils.
- H/A in 20-40%

Cerebellar hemorrhage

- Symptoms of increased ICP due to hydrocephalus
  - Compression of the 4th ventricle
  - Extension of the hemorrhage into the ventricle system

- Direct compression of brainstem
  - Facial palsy : due to pressure on the facial colliculus
  - Classically become comatose without first having hemiparesis, unlike many supratentorial etiologies

Lobar hemorrhage

- Syndromes associated with hemorrhage in the 4 cerebral lobes

- Frontal lobe : frontal headache with contralateral hemiparesis usually in the arm with mild leg and facial weakness
- Parietal lobe : contralateral hemisensory deficit and mild hemiparesis
- Occipital lobe : ipsilateral eye pain and contralateral homonymous hemianopsia
- Temporal lobe : fluent dysphasia with poor auditory comprehension but relatively good repetition

Delay deterioration

- Usually due to any combination of the following :

- Rebleeding
- Edema
- Hydrocephalus
- Seizure
- Increased ICP

Early rebleeding

- Rebleeding : more common in basal ganglion hemorrhages
- The incidence of hematoma enlargement
  - 33-38% in 1-3 hrs
  - 16% in 3-6 hrs
  - 14% between 24hrs of onset

- Risks of early bleeding
  - Spot sign on CTA

Evaluation

Practice guideline: Initial diagnosis & assessment in spontaneous ICH

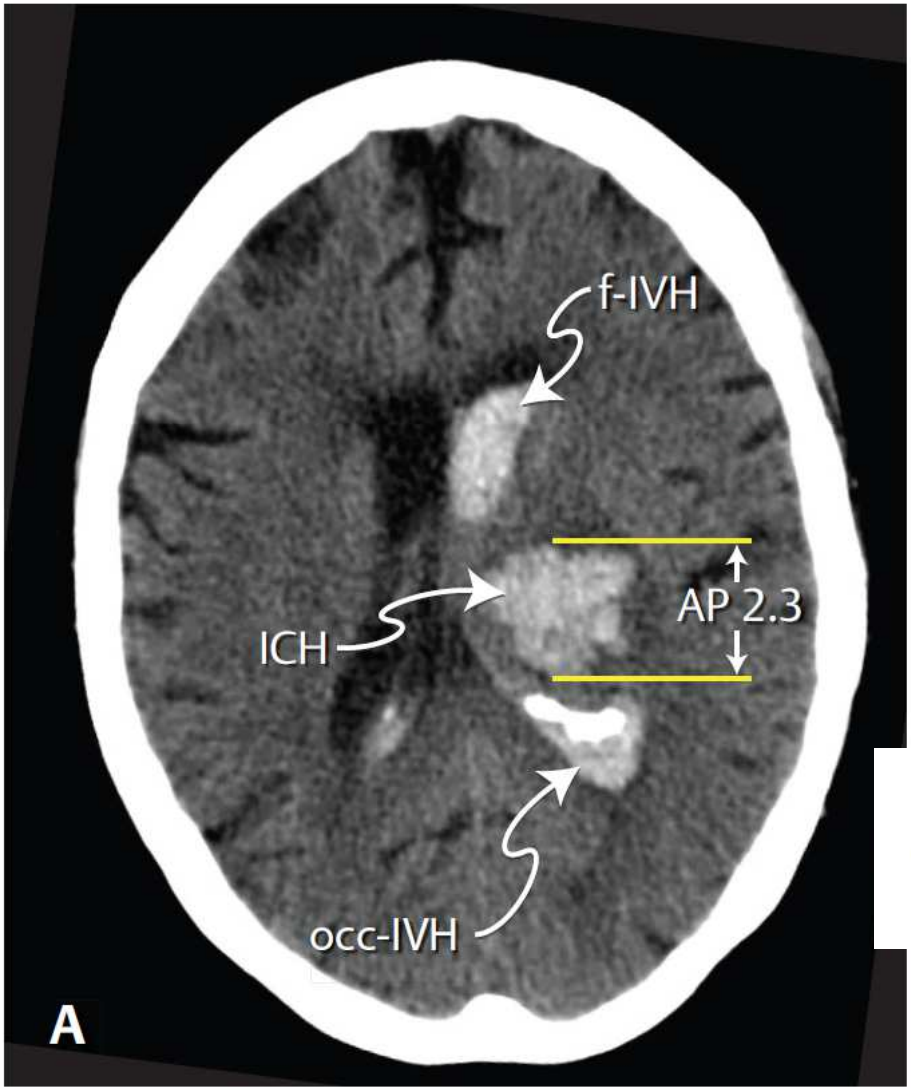
Level I[5]:

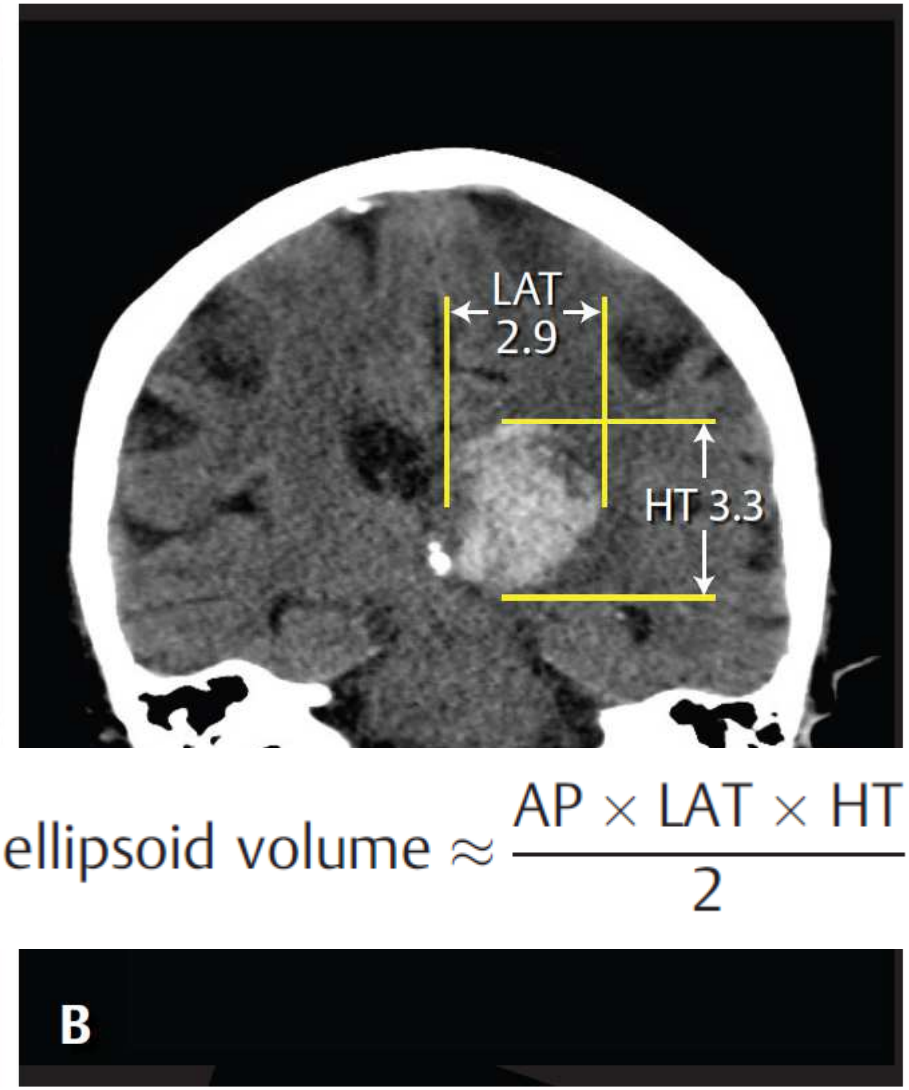
- obtain a baseline severity score
- rapid imaging with noncontrast CT (or MRI) to differentiate from ischemic stroke

Level II[[5]:

- consider CTA & contrast CT to identify patients at risk for hematoma expansion
- consider CT venogram, contrast CT, contrast MRI, MRA and/or catheter angiogram as appropriate when clinical or imaging suspicion of underlying abnormality (vascular or neoplastic)

Volume measurements on CT scan





$$\text{ellipsoid volume} \approx \frac{AP \times LAT \times HT}{2}$$

MRI

Table 84.4 Variation of brain MRI signal characteristics of intraparenchymal blood over time (modified[109])

Phase	Approximate time after onset	Hemoglobin (Hgb) state	T1 MRI[a]	T2 MRI[a]	Mnemonic[b]
hyperacute	0–6 hrs[c]	oxy-Hgb (intracellular)	I	B (slight ↑)	I be
acute	6–72 hrs	deoxy-Hgb (intracellular)	I (or slight ↑)	D	iddy
early subacute	3–7 d	met-Hgb (intracellular)	B	D	biddy
late subacute	7–14 d	met-Hgb (extracellular[d])	B	B	baby
chronic	>2 weeks[e]	hemosiderin (intracellular)	D (slight ↑)	D	doodoo

[a]B = bright (hyperintense compared to brain), D = dark (hypointense), I = isointense
[b]silly/easy to remember words made from the I, B or D in the preceding columns
[c]some authors consider up to about 24 hrs as hyperacute
[d]when RBCs lyse, the Hgb becomes extracellular
[e]the center of the clot may be isointense on T1 and slightly hyperintense on T2

ICH score

Table 84.5 ICH Score[113]

Feature	Finding	Points
GCS (Glasgow coma scale score; ▶ Table 18.1)	3–4	2
	5–12	1
	13–15	0
Age[a]	≥ 80 years	1
	< 80	0
Location	infratentorial	1
	supratentorial	0
ICH volume see Eq (84.1)	≥ 30 cc	1
	< 30 cc	0
Intraventricular blood	yes	1
	no	0
"ICH Score" = Total Points		0–6

[a]possible bias since treatment decisions in elderly patients may have differed from younger patients

Mortality based on ICH score

e 84.6 Mortality based on ICH Score

Score[a]	30-day mortality	
	0%	(26 pts)
	13%	(32 pts)
	26%	(27 pts)
	72%	(32 pts)
	97%	(29 pts)
	100%	(6 pts)
	? 100%[b]	(0 pts)

n ▶ Table 84.5

pt. in the study had a score of 6, but "it is expected this would be associated with high rate of mortality

Nonsurgical management

- Hypertension may contribute to further bleeding
- Maintain normothermia
- Seizures are treated with appropriate AEDs
- Hemostatic issues
- Treat intracranial hypertension
- EVD for hydrocephalus
- Swallowing

Blood pressure management

Practice guideline: Blood pressure management in patients with ICH

1. patients with SBP 150–200 mm Hg and no contraindication to acute BP management: lowering SBP to 140[a] is safe (Level I[5]) and improves functional outcome (Level II[5])
2. patients with SBP > 200 mm Hg: it is reasonable to consider aggressive reduction of BP with continuous IV infusion and monitoring of BP (Level II[5])

[a] since the publication of these guidelines,[5] the INTERACT-II[116] & ATACH-2[117] trials have shown that rebleeding occurs despite blood pressure control, and that reducing SBP < 140 mm Hg is associated with increased incidence of adverse renal events[117] probably from hypoperfusion

Hemostasis

1. for severe coagulation factor deficiency or severe thrombocytopenia: replace deficient factors or administer platelets (Level I[5])
2. patients on vitamin K antagonists (VKA) (e.g. warfarin) with elevated INR:
   a) withhold VKA (Level I[5])
   b) replace vitamin K-dependent clotting factors (Level I[5])
   c) correct the INR (Level I[5])
      - consider prothrombin complex concentrate (PCC) (p. 181) over FFP because PCC may have fewer complications, and corrects INR faster & closer to normal (Level II[5])
      - ✖ not recommended: rFVIIa (doesn't replace all clotting factors & may not restore clotting in vivo despite normalization of INR, & thromboembolic complications also occur) (Level III[5])
   d) administer IV vitamin K (Level I[5])
3. patients on dabigatran (Pradaxa®)[a], rivaroxaban (Xarelto®)[a] or apixaban (Eliquis®)[a]: consider treatment with activated PCC factor eight bypassing activity (FEIBA), other PCCs or vFVIIa (Level II[5]); consider dialysis for dabigatran (Level II[5])
4. patients on heparin: consider reversal with protamine sulfate (Level II[5])
5. patients on antiplatelet drugs: platelet transfusion is of uncertain benefit (Level II[5])
6. ✖ not recommended: rFVIIa in unselected ICH patients (no clear clinical benefit) (Level III[5])

Surgical treatment

- Cerebellar hemorrhage with neurologic deterioration, or brainstem compression and/or obstructive hydrocephalus
- Surgical removal of the clot should be done ASAP

- Supratentorial ICH
  - Early ICH evacuation is not clearly superior to evacuation when the patient deteriorates
  - Deteriorating pts : ICH evaluation may be considered as life-saving
  - Pts in coma, or large ICH with sig. midline shift, or refractory IICP, DC with or without ICH evacuation may reduce mortality

Indications for surgery

- One randomized prospective study found lower mortality for patients with GCS 7-10 treated surgically, however all survivor were severely disabled

- The decision to operate therefore must be individualized based on pt's neurologic condition, size and location of hematoma, age

Management of cerebellar hemorrhage

- Pts with GCS $\geqq$ 14 and hematoma < 4cm, treat conservatively
- Pts with GCS $\leqq$ 13 or hematoma $\geqq$ 4cm or with hydrocephalus or brainstem compression : surgical evacuation ASAP
- Pts with absent brainstem reflexes and flaccid quadriplegia : intensive therapy is not indicated

Outcome

- Thalamic hemorrhage tend to destroy the internal capsule are more likely to produce hemiplegia than hemorrhages lateral to the IC that compress but do not disrupt the IC

- Mortality : overall 30-day mortality rate is ≈ 44% for ICH
  - Similar to SAH (≈ 46%)

- Patients with lobar ICH tend to fare better than deep ICH with only ≈ % mortality in one study