Stroke III: Cerebral Hemorrhage

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Background

- Intracranial hemorrhage or hemorrhagic stroke:
  - 15-20% of stroke
- 4 subtypes

4 Subtypes

- Intracerebral hemorrhage (ICH) or intraparenchymal cerebral hemorrhage: primary hemorrhage into the brain parenchyma
  - 10-15% of stroke is primary ICH
    - 30% in blacks and Asians
    - eclampsia causes 40% of ICH in pregnancy
  - note: ischemic stroke may undergo hemorrhagic transformation

- Subarachnoid hemorrhage (SAH): hemorrhage into the subarachnoid space
  - 5% of stroke

- Subdural hemotoma (SDH): hemorrhage into the subdural space

- Epidural hemotoma (EDH): hemorrhage into the epidural space

Causes of Cerebral Hemorrhage

- Hypertension
- Intracranial aneurysm
- Arteriovenous malformation (AVM)
- Trauma
- Bleeding diathesis
- Complication of anticoagulant therapy
- Illicit drug use
- Mycotic aneurysm

Causes of Cerebral Hemorrhage

- Hemorrhage into a primary or metastatic brain tumor
- Hemorrhage into a brain abscess
- Arteritis (primary, connective tissue disorder, syphilitic, etc.)
- Amyloid angiopathy
- Hemorrhagic leukoencephalopathy
- Idiopathic, possibly cryptic arteriovenous malformation
ICH: Hypertension
- The most common risk factor for spontaneous ICH
- 4 common locations of ICH
  - 50% basal ganglia (especially putamen 40%, caudate 7%), plus thalamus 10% = 60%
  - 20% lobar
  - 10% pons
  - 10% cerebellum
- Pathogenesis; HTN causes:
  - fibrinoid neurosis of penetrating and subcortical arteries
  - formation of Charcot-Bouchard microaneurysms
  - predisposing to hemorrhage

ICH: Cerebral Lobar Hemorrhage
- 50% hypertensive
- 50% not related to hypertension
  - amyloid angiopathy
  - AVM
  - aneurysm

ICH: Amyloid Angiopathy
- Patients typically 60 years and older
- Involved blood vessels friable
- Surgical evacuation should be avoided

ICH: Bleeding Diathesis
- Thrombocytopenia, platelets < 30,000/uL
- Factor VIII and Factor IX deficiencies, genetic
- Hypofibrinogenemia
- Disseminated intravascular coagulation (DIC)

ICH: Drugs
- Drugs of abuse: cocaine, amphetamines
  - sympathomimetic effect may cause acute rise in BP
  - promote vasculitis
- Anticoagulants
  - Warfarin
  - Heparin
  - Enoxaparin

ICH: Other
- Hemorrhage into CNS neoplasm
  - primary neoplasm
  - metastasis: lung, breast, melanoma, thyroid, renal
- Embolism
  - septic embolism in bacterial endocarditis
  - non-septic embolism in marantic endocarditis
- Hemorrhage into brain abscess
ICH: Clinical Presentation

- Acute severe HA
- Obtundation or coma
- Markedly elevated BP
- Neurologic deficit maximal at onset

ICH: Neuroanatomic localization
- Putamen: contralateral hemiparesis and hemisensory loss, aphasia (left cerebral), hemineglect (right cerebral)
- Thalamus: contralateral hemisensory loss
- Cerebellar: ipsilateral dysmetria, gait ataxia
- Pons: paraparesis or quadriplegic, horizontal gaze palsy

ICH: Evaluation

- Neuroimaging
  - Head CT, CT angiography
  - Brain MRI, Head MRA
  - Cerebral angiography
- Risk factor assessment, including blood pressure
- Laboratory
  - CBC, diff, plt
  - PT, PTT, INR
  - Fibrinogen
  - Drug screen
  - Syphilis reagin
  - Westergren ESR, ANA
  - ABG
  - Factor VIII and IX in selected patients

Head CT

Left Basal Ganglia Hemorrhage

ICH: Treatment

- Airway and respiratory support
- Blood pressure control: labetalol, sodium nitroprusside
- Surgical evacuation for lobar hemorrhage, cerebellar hemorrhage
- Heparin induced: protamine sulfate
- Warfarin induced: fresh frozen plasma
  - Note that vitamin K may take 8 to 24 hours for full effect
- Acute obstructive hydrocephalus from intraventricular hemorrhagic extension: ventricular shunt

SAH: Intracranial Aneurysm Epidemiology

- Aneurysmal SAH annual US incidence is 1 in 10,000
  - 0.5-1.0% of adults have cerebral aneurysm
  - Multiple aneurysms common, approximately 25%
- Risk factors
  - Female, sex, age, cigarette smoking, HTN, heavy ETOH, sympathomimetic agents
  - Genetic Marfan’s syndrome, Ehlers-Danlos syndrome IV, NF 1, AD polycystic kidney disease
SAH: Intracranial Aneurysm

Epidemiology
- 40% mortality rate from aneurysmal SAH
- Neuroanatomic localization
  - 85% aneurysms in anterior circulation
  - Junction of ICA + PCoA
  - ACoA
  - MCA trifurcation
  - 15% aneurysms in posterior circulation
  - Basilar tip
  - Junction of vertebral artery and PICA

Aneurysmal SAH: Clinical Presentation
- Severe HA, worst in life
- “Warning leak” in 25% of patients
- Meningeal irritation
- Syncope
- Retinal subhyloid hemorrhage
- Focal signs
  - PCoA: 3rd CN palsy with pupillary dilatation
  - MCA: contralateral paresis
  - ACoA: bilateral LE paresis
  - Basilar: coma, vertical gaze paresis

Aneurysmal SAH: Evaluation
- Head CT: 90-95% sensitive in 1st 24 hours
- Lumbar puncture nearly 100% sensitive
- CTA, MRA
- Cerebral angiography

Head CT

Subarachnoid Hemorrhage and Intraventricular Hemorrhage

Cerebral Angiography

Posterior Communicating Artery Aneurysm

Aneurysmal SAH: Treatment
- Blood pressure control
- Early surgical clipping
  - 10-20% rebleeding rate in 1st 24-72 hours if early surgery cannot be performed
- Endovascular occlusion: coil and other developing techniques
Aneurysmal SAH: Complications
- 50% develop cerebral vasospasm in the 1st 3-15 days
  - may cause ischemic infarct
  - nimodipine, a calcium channel blocker, used to reduce incidence
- 20% have acute obstructive hydrocephalus
  - ventriculostomy
- 10-30% develop hyponatremia
- 25% have seizures

Aneurysmal SAH: Prognosis
- 40% mortality
- 3-4% rebleeding risk in 1st 24 hours
- 1-2% per day rebleeding risk in 1st month
- 3% rebleeding risk per year after 1st 3 months

Anterior venous Malformation (AVM): Epidemiology
- Embryonal anomaly—conglomeration of arteries and veins without intervening capillaries
- Clinically evident most often ages 10-40 years
- 0.15% prevalence; most remain asymptomatic
- Men affected 2x rate of women
- 4.6% hemorrhagic mortality rate

AVM: Clinical Presentation
- 50% present with SAH or ICH
  - AVM the 2nd most common cause of spontaneous SAH
- 30% present with seizures
  - 1% of such patients hemorrhage in 1st year
- 20% present with HA, focal neurologic deficits, or cognitive impairment
- Evaluation is similar to that for cerebral aneurysm

Arteriovenous Malformation
CT, Cerebral Angiography, MRI

AVM: Treatment
- Surgical excision
- Endovascular occlusion: balloon, coil, glue, sclerosing drugs
- Radiotherapy to promote occlusive vascular injury and thrombosis
Subdural Hematoma

- Pathogenesis: shearing of bridging veins between the brain surface and adjacent dural venous sinuses causes bleeding in the subdural space
  - SDH may absorb spontaneously or become encapsulated
- Acute SDH carries 50% mortality due to trauma
- Chronic SDH develops more slowly
  - risk factors: minor trauma
- SDH, SAH and ICH often co-exist

SDH: Clinical Features

- 3 features most common
  - HA
  - decreased alertness
  - cortical abnormalities
- Seizures may occur

SDH

- Diagnosis, neuroimaging
  - crescent-shaped density over brain outer surface against dura and inner skull surface
  - Appearance changes over time
    - acute SDH: hyperdense on CT
    - subacute SDH: isodense on CT, hyperdense on T1 MRI
    - chronic SDH: hypodense on CT
- Management
  - surgical excision
  - observation

Head CT

Epidural Hematoma

- Pathogenesis: tear of middle meningeal artery causes blood collection between skull and dura mater
  - most common location is along lateral wall of middle cranial fossa
- Rare compared to SDH, usually due to severe head trauma

EDH: Clinical Features

- Head trauma
  - loss of consciousness
  - lucid interval
  - progressive decreased alertness
- Headache
- Cortical abnormalities
EDH

- Diagnosis, neuroimaging
  - Biconvex hyperdensity on CT
- Management
  - Surgical evacuation

Head CT

Right Epidural Hematoma

Intracranial Hemorrhage: Summary

- 4 types: ICH, SAH, SDH, EDH
- Spontaneous or traumatic
- Risk factors: HTN, aneurysm, AVM, anticoagulants, trauma
- Mortality and morbidity are high
- Management: risk factors, surgery, endovascular