CNS PATH: Intracranial Mass Lesions, Vascular Lesions, & Hemmorahages & Ischemia

Cerebral Edema: accumulation of excess fluid within brain parenchyma

types

- Vasogenic edema: disruption in BBB→ fluid shifts from vascular compartment to extracellular spaces (vessels damaged)
  - Causes: high altitude (hypoxia causes endothelial injury), severely high BP, tumors that release vasoactive molecules (gliomas)
- Cytotoxic edema: intracellular edema of cells due to cell injury by ischemia (neurons damaged)
- Osmotic edema: due to ↓ serum osmotic pressure

Clinical features: headache, vomiting, blurry vision, papilledema, Cushing's triad (HTN, ↓ HR, irregular breathing), herniation & death

Hydrocephalus

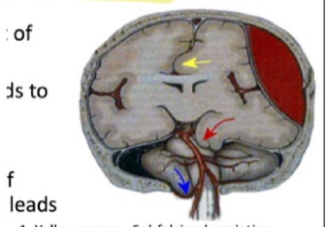
Noncommunicating
- CSF circulation = blocked
- MCC= masses (tumors, hemorrhage, infection) block foramen Monroe
- Arnold chairi

Communicating
- CSF not blocked, flows freely b/t ventricles
- MCC = overproduction of CSF ex) choroid plexus papilloma
- Deficient reabsorption of CSF (meningitis, SA hemorrhage)

Normal Pressure
- Compensatory increase of CASF due to loss of neural tissue

Herniations

Subfalcine
- Displacement of cingulate gyrus under falxcerebri
- Compression of anterior cerebral artery



1. Yellow arrow - Subfalcine herniation
2. Blue arrow - Foramen magnum herniation
3. Red arrow - Transtentorial (uncal) herniation

Tonsillar
- Displacement of cerebellar tonsils into foramen magnum
- Compression of brainstem→ resulting in cardiopulmonary arrest

Posterior block

Transtentorial
-
- Downward displacement → rupture basilar artery branches→ fatal

Uncal
- Subtype of transtentorial herniation
- Displacement of temporal lobe uncus
- Compression of CNIII (eye = down & out & dilated) & posterior cerebral artery (occipital lobe infarction)
-

Duret Hemorrhages
- Associated w/ transtentorial herniation
- Rupture of paramedian artery

Result: tearing of pontine penetrating veins & arteries suppling upper brain

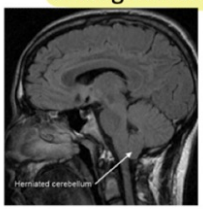
Developmental Malformations

Neural tube defect
- Failure of neural tube closure abnormalities involving neural tissue, meninges, and overlying bone or soft tissue
- Associated w/ low folate, methotrexate & valproic acid
- ↑ alpha-fetoprotein

Can result in
1. Anencephaly: no brain/skull

Dandy walker malformation
- cystic malformations on the cerebellum that blocks CSF→ dilated 4th ventricle
- motor affected (gait, eye movements, paralysis/spasticity)

Arnold Chiari malformation
- cerebellum displaced through foramen magnum



Syringomyelia
- abnormal fluid filled cavity that develops within central canal of spinal cord
- results from disrupted CSF drainage

causes
- Arnold chiari malformation
- post trauma

Tuberous sclerosis
- mutation in TSC1 (hamartin) & TSC2 (tuberin)
- pts develop hamartomas & benign tumors of brain, skin, kidneys, heart, eyes, lungs
- adenoma sebaceum (angiofibroma): facial skin lesions

Effects of Mass Lesions

↑ICP

An initial increase in volume results in a small increase in pressure because of intracranial compensation. Once intracranial compensation is exhausted, additional increases in intracranial volume result in a dramatic rise in intracranial pressure. **Pathologic intracranial hypertension: ≥ 20 mmHg.**

SYMPTOMS: ***headache*** *(mediated by pain fibers of trigeminal nerve in the dura & blood vessels),* ***depressed global consciousness*** *(due to local mass effect or pressure on midbrain reticular formation),* ***vomiting***

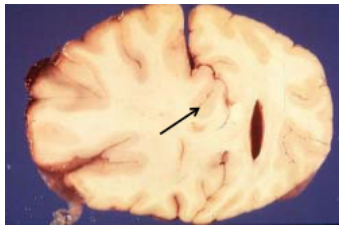
CLINICAL: CN6 palsy, **papilledema** (blurring of optic disc margins, loss of cupping), **CUSHING'S TRIAD*** (bradycardia, respiratory depression, & HTN)

TREATMENT: underlying disease, **STEROIDS + MANNITOL** to ↓edema & ↑osmotic pressure so fluid moves out of brain cells, **HYPERVENTILATION** to ↓ICP

SUBFALCINE HERNIATION

Occurs when mass lesion occurs ***supratentorially*** *in one hemisphere; often herniation of* ***Cingulate Gyrus***

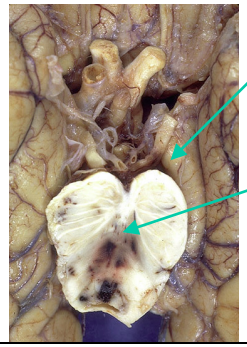
GROSS: MIDLINE SHIFT TO OPPOSITE SIDE beneath the falx

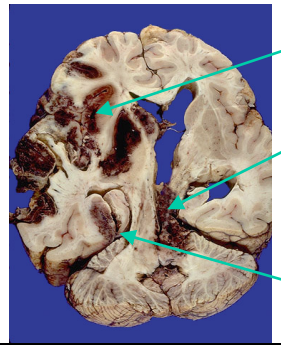


COMPLICATIONS: compression of **ipsi anterior horn of lateral ventricles & compression of branches of ACA → *weakness & sensory loss of LE***

UNCAL HERNIATION

1. **KERNOHAN'S NOTCH PHENOMENON:** *contralateral pupillary dilation, ipsilateral weakness due to compression of* ***cerebral peduncles***
2. **DURET HEMORRHAGES:** *due to herniation downward with compression of* ***brainstem***
3. **POSTERIOR CEREBRAL ARTERY (PCA) COMPRESSION → OCCIPITAL LOBE INFARCTION:** *Duret hemorrhages*
4. **CN 3 PALSY:** *ptosis, mydriasis, down & out*

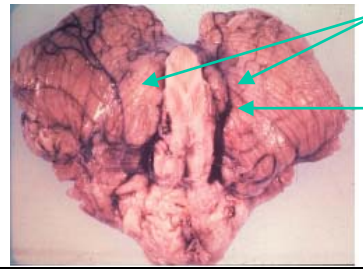




TONSILLAR HERNIATION

CAUSES: mass lesion of the **cerebellum** or **brainstem;** downward herniation of hemispheres

COMPLICATIONS: major route of escape of cerebellum is into the ***foramen magnum*** causing compression of cerebellum + compression of **medullary respiratory centers & consciousness centers → DEATH**



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2 major types of CVA: ischemic & hemorrhagic

Ischemic (insufficient blood flow)

Global cerebral ischemia→ results in liquefactive necrosis

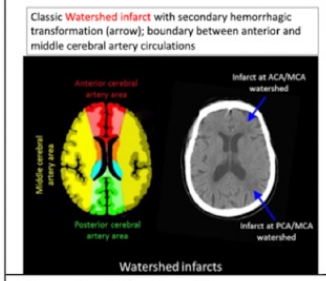
- Generalized cerebral ischemia due to reduced perfusion or oxygenation
- Result from: severe systemic hypotension, hypovolemic shock, profound hypoglycemia, carbon monoxide poisoning

Mild: complete recovery

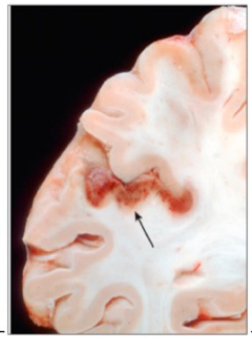
Severe: widespread neuronal death, brain dead

Watershed infarct

- Occur @ distal portions
- Seen after hypotensive episodes
- Border zone b/t ACA & MCA = greatest risk
- Sickle shaped band of necrosis over the cerebral convexity produced
- Paresis/paralysis of limbs, sparing hands & feet, face
- PCA-MCA watershed → bilateral vision loss



Classic Watershed infarct with secondary hemorrhagic transformation (arrow); boundary between anterior and middle cerebral artery circulations


Watershed infarct

Focal cerebral ischemia

- Localized ischemia to brain due to arterial occlusion or low perfusion
- Can cause a pale infarct→infarct can evolve into hemorrhagic (petechial) infarct due to reperfusion
- MC results from: embolus, thrombus & hyaline arteriolosclerosis

1. Emboli (originate from)
   - Cardiac thrombi: MI, Afib, valvular disease
   - arterial thrombi: carotid arteries→ atheromatous thrombus
   - paradoxical emboli: mostly children w/ cardiac problems
   - emboli MC lodge in MCA
2. Thrombotic occlusion
   - Atherosclerotic plaque rupture
   - Infectious vasculitis: seen w/ syphilis, TB, immunosuppression
3. Hyaline arteriolosclerosis: complication of HTN→ cause lacunar infarcts (deep portions of brain)

Cerebral infarct…

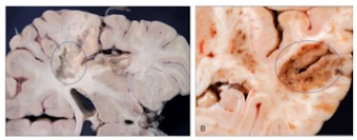
Morphology

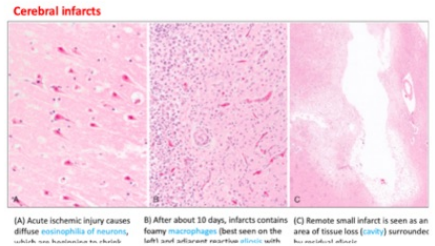
Gross

- pale, soft, swollen by 48hrs
- Liquefies @ day 10-20→ fluid filled cavity (liquefactive necrosis)

Microscopic

- Swollen, red neurons
- 1st several days: neutrophils infiltrate→ 2-3 weeks: replaced w/ macrophages→ several months: astrocytes form glial fibers


Non-hemorrhagic infarct | Hemorrhagic infarct



(A) Acute ischemic injury causes diffuse eosinophilia of neurons, B) After about 10 days, infarcts contains foamy macrophages (best seen on the (C) Remote small infarct is seen as an area of tissue loss (cavity) surrounde

Treatment

- Recombinant tissue plasminogen activator within 3-5hrs

Clinical

1. Middle cerebral artery: MC embolic occlusion site

Clinical Features of Stroke, affected by the VESSEL involved.

1. Middle cerebral artery is the most frequent site of embolic occlusion.
- Sensory loss of the contralateral face, arm and leg
- Hemiparesis or hemiplegia of the contralateral face (lower half)
- Hemiparesis or hemiplegia of the contralateral upper and lower extremities
- Aphasia
- Gaze preference towards the side of the lesion

2) Anterior Cerebral Artery:

- Contralateral hemiparesis and sensory loss in the lower limbs more marked than in the upper limbs.
- Transcortical motor aphasia (lack of fluency with intact comprehension and repetition)
- Urinary incontinence
- Dysarthria
- Frontal release signs (re-emergence of primitive reflexes in adults as a result of frontal lobe damage)

3) Posterior Cerebral Artery

- Vertigo, nausea
- Contralateral homonymous hemianopia
- Contralateral sensory loss due to lateral thalamic involvement memory deficits
- If dominant hemisphere (usually left)- alexia (inability to read or understand the reading).

2. Anterior cerebral artery
3. Posterior cerebral artery

Hemorrhagic (due to hemorrhage)

Intracerebral hemorrhage (bleeding within brain parenchyma)

- Spontaneous (nontraumatic), rupture of small penetrating branches of MCA (lenticulostriate branches)→ Charcot-Bouchard microaneurysms
- MCC = hypertension
- Occur in basal ganglia, thalamus, pons, etc.

Subarachnoid hemorrhage (bleeding into subarachnoid space)

- Rupture of berry aneurysm (located in anterior circle of Willis)
- Other causes: malformations, tumors, anticoagulants
- Sudden headache (worst headache of my life), vomiting, rapid loss of consciousness, neck rigidity
- Develop over time due to defects in vessel structure

↑ risk of aneurysm development
- connective tissue genetic disorder (Ehlers-Danlos syndrome, Marfan syndrome)
- cerebral arteriovenous malformation
- HTN, drugs, smoking

Hypertensive cerebrovascular disease

Lacunar infarcts
- small cystic infarcts

- Occurs 2ndary to hyaline arteriosclerosis
- Involves deep grey matter (BG & thalamus), internal capsule, pons
- Caused by occlusion of single penetrating branch of large cerebral artery (lenticulostriae vessels)
- Internal capsule→ pure motor stroke
- Thalamus→ pure sensory stroke

Slit hemorrhages

- Small cerebral infarcts associated w/ rupture of small-caliber penetrating vessels due to chronic hypertension
- Leaves behind slit like cavity w/ gliosis

Hypertensive encephalopathy

- Brain dysfunction due to malignant hypertension
- ↑ ICP & global cerebral dysfunction: Confusion headaches, vomiting, convulsions, coma
- Petechiae
- Fibrinoid necrosis of arterioles→ infarcts w/ neuro defects (dementia, paresis/paraplegia)

	Ischemic stroke	Intracerebral hemorrhage	Subarachnoid hemorrhage
Incidence	- About 85%	- About 10%	- About 5%
Causes	- Embolism - Thrombosis - Small vessel occlusion (lipohyalinosis) - Systemic hypoperfusison	- Ruptured cerebral artery or micro-aneurysm - Trauma - Reperfusion injury after ischemic stroke	- Ruptured berry aneurysm - AV malformation
Risk Factors	- Age above 65 years - Hypertension - Diabetes mellitus - Atrial fibrillation - Carotid artery stenosis	- Age above 65 years - Hypertension - Vasculitis - Malignancy - Ischemic stroke	- Hypertension - Smoking - Family history
Pathology	- Pale infarct liquefactive necrosis glial scarring	- Hematoma surrounded by pale infarct and edema - Hemosiderin lined cavity with glial scarring	- Hematoma surrounded by pale infarct and edema - Hemosiderin lined cavity with glial scarring

BERRY ANEURYSMS:

- Thin-walled saccular **outpouchings** that lack a media layer
- Frequently located **in the anterior circle of Willis** at branch points of the anterior communicating artery.
- Aneurysms develop over time because of underlying defects in the vessel media.
- Increased risk for aneurysm development in patients with:
  - connective tissue genetic disorders (Ehler-Danlos syndrome, Marfan syndrome),
  - autosomal dominant polycystic kidney disease
  - cerebral arteriovenous malformation
  - Hypertension, Drugs (e.g., amphetamine), Smoking