Basal Ganglia anatomy

• GP + Putamen = Lentiform nucleus
• Caudate head + Lentiform nucleus = Striatum
Normal enhancing brain

• Choroid plexus
• Pineal
• Pituitary (post pit already bright on T1) and stalk
• Cavernous sinus
• Nasal mucosa
• Extraocular muscles
• CTZ (medulla)
Bright on DWI

• Acute infarct (<10days)
• Hemorrhage ("all bets off")—hyperacute to late subacute
• Abscess
• MS (active enhancing lesions)
• PRES
• Herpes encephalitis
• Sustained seizure activity
• CO poisoning
• Osmotic myelinolysis
• CJ
• Epidermoid cyst (follow CSF except FLAIR and DWI)
• Hypercellular mass
  • Meningioma (20%)
  • Lymphoma (enhancing components; essentially never bleeds)
  • High-grade gliomas/PNET (based on cellularity; non-necrotic portions)
Restricted diffusion DWI+

- Acute and sub acute ischemic stroke - Usually takes 7-14 days for hyperintensity to subside
- Hemorrhagic stroke
- Trauma, Diffuse Axonal Injury
- Acute demyelination
- Marchiafava Bignami disease
- Hypoglycemic Encephalopathy
- Encephalitis – Herpes, CJD, Influenza like encephalitis
- Focal cerebritis, Acute cerebellitis, Brain Abscess and Empyema
- Choroid plexus cyst
- Epidermoid cyst
- Cholesteatoma
- Medulloblastoma
- Atypical Meningioma
- Thrombus dural venous sinuses
- Near mastoid and roof of orbit (Air-bone interface artifact)
VASCULAR
Capillary telangiectasia

- Faint (feathery) blush of enhancement in pons
- 50% can be faintly T2 bright (may have faint susceptibility on GRE)
- No mass effect
- Usually pons
- Can be seen in cerebellum
Venous angioma or DVA

- Subtle vein traversing white matter on post-Gad imaging
- Caput medusa sign (collection of smaller veins draining into collecting vein which in turn may drain into dural sinus or ependymal vein)
- Can be located superficial or deep
- Best seen on postGad imaging
- May be assoc with cavernoma
Cavernoma

- Popcorn bright on T2 with low peripheral hemosiderin rim
- Partly T1 bright due to met heme
- Blooming on GRE
- Minimal to no edema or mass effect
- 50% have calcifications
- May have assoc DVA
AVM

- Clustered serpiginous flow voids (curvilinear tangle of vessels)
- Arterial feeders (can be more than one)
- Nidus (focal nidus vs no well-formed nidus; size <3cm, 3-6cm, >6cm)
- Enlarged early draining venous channels (drain into superficial only vs deep venous sinus)
- Spetzler-Martin grading system
- Eloquent brain (sensorimotor, language, visual cortex, hypothalamus, thalamus, brain stem, cerebellar nuclei)
Dural AVF

• 50-60yo
• Abnormal connection between dural arteries, which are branches of the external carotid (ECA) with the venous sinuses
• Usually acquired (s/p trauma, craniotomy, dural venous thrombosis, XRT)—not congenital unlike AVM
• Lacks nidus!!
• Location: transverse/sigmoid colon (most common), carotid-cavernous fistula
• Complication: hemorrhage, intracranial HTN
• Subtle on CT (look for dilated/tortuous cortical veins along cortical surface and thrombosed sinus)
• Better visualized on MRA
Cavernous-carotid fistula

- Proptosis (painful)
- Pulsatile exophthalmos
- Enlarged superior ophthalmic veins
- Enlarged cavernous sinus with prominent flow voids
- May have enlarged EOM
- DDx: cavernous sinus thrombosis
Virchow-robin spaces

- Perivascular spaces
  - Inferior 3rd of BG
  - Centrum semiovale
  - Midbrain at jct of substantia nigra and cerebral peduncle

- Iso to CSF on all sequences
- No rim of gliosis on FLAIR unlike lacune
VRS mimics

• Lacunar infarct
  • Usually ≥5mm
  • Unilateral
  • Acute=T2/FLAIR bright and +DWI
  • Chronic=T2 bright, ring of gliosis on FLAIR, and -DWI
  • Enhancement is variable

• MS
  • Periventricular and juxtacortical WM
  • Avoid
  • Perpendicular to lat vent (dawson’s fingers)
  • Acute=hypoT1, hyperT2/FLAIR, solid/ring enhance
  • Chronic=ring of T1 bright, hyperT2/FLAIR, don’t enhance

• Cryptococci
  • If immunocompromised w/ dilated VRS, think of this
  • Involves BG, thalami, and midbrain

• Cysticercosis
  • Acute vesicular=iso to CSF on all sequences (may see discrete eccentric scolex hyperintense to CSF) and don’t enhance
  • Colloid vesicular=cyst midly hyperintense to CSF with mild to marked edema; thick cyst wall enhancement (also scolex)
  • Granular nodular=thick retracted cyst wall with nodular or ring enhancement; decreasing edema
  • Nodular calcified=lesion shrunken; completely calcified; hypointense on all sequences; GRE susceptibility
Lacunar infarct

- Small vessel occlusion usually involving deep gray nuclei

- In order of incidence
  - Putamen
  - Thalamus
  - Pons
  - Caudate
  - Posterior limb of internal capsule
Cerebral fat embolization syndrome

• Hx of fracture
• Preceded by abnormal chest findings (infarcts)
• Later develop small microinfarcts in watershed territories (frontal lobes and centrum semiovale) with microhemorrhages
• Mimic DAI but not a gray-white jct
SVIC grading

• Fazekas I: Mild, few small punctate lesions in the deep white matter (normal aging in >40yo)
• Fazekas II: Moderate; larger WMLs that are beginning to become confluent (abnormal <75yo)
• Fazekas III: Severe; confluent T2 hyper intensity (abnl in any age gp and may indicate poorly controlled DM and HTN)
Lateral Medullary infarct

- Wallenberg’s syndrome
- Occlusion of PICA or vertebral artery
Medial Medullary infarct

- Anterior spinal artery
Sup Cerebellar Artery infarct

- Arises from basilar apex
- Supplies sup vermis and cerebellum (also deep cerebellar white matter and dentate nucleus)
Anterior choroidal artery infarct

- AChA originates from supraclinoid ICA just distal to origin of PCOM
- Extending from medial temporal lobe and extending superiorly along posterior limb on internal capsule and posterior periventricular region
- AChA sullies choroid plexus (lateral and 3rd ventricles), optic chiasm, posterior limb of internal capsule, lateral geniculate body, GP, tail of caudate, hippocampus, amygdala, substantia nigra and crus cerebri
Recurrent artery of Heubner infarct

• Originates form A2 segment (occasionally from A1 segment)
• Take u-turn and supplies caudate head, anterior limb of internal capsule and anterior third of putamen
ICA

• Carotid Siphon=S-shaped portion on ICA (comprised of cavernous and supraclinoid portions)
• Petrous → Cavernous → Supraclinoid
• Ophthalamic artery originates from supraclinoid portion (helps distinguish cavernous from supraclinoid segment)
Persistent trigeminal artery

- Unlike fetal PCA, it originates from cavernous ICA (no supraclinoid ICA) and connects to Basilar.
- Arises from the cavernous ICA extends posteriorly to join basilar artery usually between the origins of the superior and anterior inferior cerebellar arteries.
- Has a parasellar course.
- May have associated aneurysm.
Fetal PCA

- Absent or hypoplastic P1 segment of PCA
- PCOM originating from ICA continuing as PCA

- DWI in a Young patient with an internal carotid artery occlusion resulting in acute MCA and PCA territory infarcts due to a fetal origin of posterior cerebral artery.
Persistent fetal arteries

- **Primitive anastomosis** btwn ICA and vertebrobasilar system
- **Persistent trigeminal (PTA)**
  - Most common 0.1-0.5%
  - Cavernous ICA to basilar
  - ↑ AVMs, aneurysms, stroke
- **Persistent otic (POA)**
  - Petrous ICA to basilar thru IAC
- **Persistent hypoglossal (PHA)**
  - Second most common after PTA
  - Cervical ICA to basilar thru hypoglossal canal
  - Basilar may be hypoplastic or absent below
- **Pro-atlantal intersegmental**: btwn arch C1 & occiput, ECA, or ICA to vertebral artery
PCOM infundibulum

• Funnel shaped dilatation at origin of PCOM
• Usually conical in shape with dia <2mm
• Base along ICA and PCOM arises at its apex
PCOM aneurysm

- CN III (oculomotor) palsy leading to ptosis and myosis
Dolico-ectasia of VB artery

• Abnormal elongation, dilatation and tortuosity of the vertebral and basilar arteries
• Criteria for dolichoectasia is basilar artery diameter of more than 4.5 mm
• May present with CN compressions, most commonly affects CN VII and V (may compress and distort ventrolateral aspect of medulla)
Trolard (orange) and Labbe (blue)
Sinus thrombosis

- Loss of T2 flow void (may see partial re-canalization in chronic cases)
- T1 bright signal thrombus (subacute)
- Loss of flow-related enhancement on non-con 2D TOF MRV
- SSS→parsaagittal infarcts
- ICV→bilat thalamic infarcts
- Transverse sinus or vein of Labbe→posterolat temporal lobe infarcts

MRI
- Dense sinus on NCCT and empty delta sign CECT
- T1 bright thrombus
- Loss of sinus flow void on T2
- Loss of flow-related enhancement on TOF
- FLAIR pitfall=acute thrombus may be dark
- TOF MRV pitfall=thrombus may be bright

FEATURES
- May be hemorrhagic
- Involve subcortical WM
- Mass-like (mimic CA)
- +/-DWI
Sinus thrombosis
Arachnoid granulations

• Round or ovoid filling defect in venous sinus
• T2 bright and low signal on FLAIR
• Follows CSF signal on all sequences unlike thrombus
Vasospasm s/p aneurysm clipping

Pt develops aphasia 10 days post clipping
Dissection

• Location
  • Spontaneous ICA=just above bifurcation
  • Traumatic ICA=distal near or at skull base
  • Vertebral=at C1-C2

• T1 FS shows bright crescentric hematoma widening vessel diameter

• Vertebral artery does not show T1 bright hematoma but will be narrowed vessel within normal size transverse foramen

• Findings:
  • String sign
  • Aneurysmal dilation (pseudoaneurysm)
  • Occlusion
  • Intimal flap
NON-TUMOR BRAIN
NORMAL VARIANTS

- **NORMAL VARIANT:**
  - lack of myelination post limb of IC
  - aplastic rostrum of CC
  - lipoma of CC (curvilinear)
  - empty sella
  - deviated pit stalk
  - asymmetric pit gland
  - tapered infundibulum
  - ectopic post pit gland vs lipoma
  - virchow robin space (BG, centrum semiovale, midbrain; follow CSF on all seq; no gliosis on FLAIR)
  - falcine lipoma
  - CSP (ant); cavum vellum interpositum (post); CSP et vergae (both)
  - shifted septum pellucidum
  - cystic foci in hippocampal sulcal remnants
  - choroid fissure cyst
  - absent/hypoplastic vascular seg
  - fenestration (focal duplication) vs duplication
  - fetal origin of PCA from ICA
  - primitive trigeminal artery (carotid-basilar anastomosis)
  - bulbous basilar tip vs basilar dolicoectasia (can result in CN 5 compression)
  - dominant artery or venous sinus
  - hi riding jug bulb (above IAC)
  - developmental venous anomaly (venous angioma; caput medusa)
  - venous lake vs granulation tissue w/in calvarium
CSP, CSP et vergae, Cavum veli interpositum

- Cavum septum pellucidum (CSP)=anterior superior triangular with apex pointing post
- Cavum vergae (CV)=posterior superior rectangular
- CSP+CV (cavum septum pellucidum et vergae) can occur together
- Cavum velum interpositum (CVI)=posterior inferior triangular with apex pointing ant
Cavum septum pellucidum

• CS pellucidum (ant)
• CSP et vergae (continuous)
• Cavum villum interpositum (post)
Ependimitis granularis

• Symmetric periventricular high T2 and FLAIR signal along frontal horns of lateral vents
Choroidal fissure cyst

- Along choroidal fissure
- Can be upto 2cm in dia
- Thin wall, non-enhancing
Choroid plexus xanthogranulomas

• Choroid plexus xanthogranulomas are common, incidental and almost invariably asymptomatic lesions
• Basically bilateral adult choroid plexus cysts
Calcifications

- **PINEAL**
  - Seen in ~40-60% of normal pts by age of 20 (rare in <6yo)
  - Should be <1cm in diameter (can be up to 14mm)

- **CHOROID PLEXUS**
  - Very common after age 40
  - Usually atrium of lateral ventricle (uncommon in 4th ventricle)

- Pathologic (TORCH, phakamatoses, cysticercosis, healed abscess, hydatid cyst, HPT, CO/Pb poisoning, Fahr’s, tumors)
T2 bright BG

- “LINT”
  - Lymphoma
  - Ischemia (hypoxia, venous infarcts)
  - Neurodegenerative
    - Wilson
    - Huntington disease
    - Mitochondrial disease (eg Leigh)
- Toxins
  - CO
  - CN
  - Hypoglycemia
  - Methanol
T1 bright BG

- CO
- Fe (Hallervordin spatz)
- Hemorrhage
- Hepatocerebral degeneration
- HPT
BG calcifications

- Physiologic
- Hypoxia
- CO or lead poisoning
- Fahr’s
- Leigh’s
- Hypo/hyper PTH
- Toxo/CMV or congenital HIV
BG hypodensity

• Think O2, CO, Cu
• Hypoxemic insult
• Poisoning (CO, CN)
• Metabolic (Leigh’s=putamen; Wilson’s=putamen+GP, Mitochondrial)
Bilateral thalamic lesions

- Wernicke’s encephalopathy (adults=atrophic mammillary bodies with increased signal within medial thalami and periaqueductal gray→give thamine)
- Leigh’s (in neonates)
- Reversal sign (diffuse cerebral edema)
- HTN bleed if unilateral
- Venous infarct (usually bilat due to internal cerebral vein thrombosis)
Hydrocephalus (Evans index A/B over 0.3 is abnl)
Hypertensive hemorrhage

- Most common location
  - BG (usually putamen; may decompress into ventricle)
  - Thalamic
  - Pontine
  - Cerebellar
- Lobar (parietal or occipital lobes)
Amyloid angiopathy

• Typically >65yo (normotensive)
• Usually cortical/subcortical lobar hemorrhage (esp parietal or occipital) at G-W jct
• Spares BG and periventricular regions
• May have multiple hemorrhages of different stages (microbleeds on GRE)
• May have dementia
Coagulopathy

- Can occur anywhere
- Tend to be large
- Blood fluid level
Hemorrhagic contusion

- Early contusion may be hypodense (mottled/speckled density “salt pepper”)
- Bland contusion are lower density than brain (may have assoc edema)
- Bland contusion may become hemorrhagic within hours to several days after injury
- Associated edema begins 24hrs and increases over next 7days and then slowly resolves
- Contusion usually cortical and hemorrhagic (if no initial hemorrhage, may develop it w/in 72hrs) due to rich vascularity of gray matter
- Mechanism: direct contact w/ skull and shear-strain deformation
- Coup=same side as impact (look for fx)
- Countrecoup=opposite to impact (broader surface of impact)
- Commonly along anterior, inferior, lateral frontal and temporal tips (over bony prominences like petrous pyramid, sphenoid wing, and orbital roof) ← may present with anosmia (olfactory nerve)
- May also see w/in dorsolateral midbrain
- Multiple in 30%
- Overlying cortex always involved (unlike DAI)—at surface or crown of gyri (linear or flame shaped)
- “Salt and pepper” (intermixed w/ edema) ← blood intermixed with normal brain tissue
- F/U CT in 24-48hrs may show enlarging hemorrhage, edema, and multifocal
DDx of parenchymal hemorrhage

• Underlying lesion
  • Tumor – primary or mets (GBM, oligo, mets from lung/RCC/melanoma/chorioCA)
  • Vascular malformation (cavernoma; AVM)
  • Aneurysm
  • Hemorrhagic infarct
• Hypertensive (BG/thalamic/pontine/cerebellar)
• Amyloid angiopathy (>65yo)
• Dural sinus thrombosis (parasagittal; bilateral thalamic, posterolateral temporal lobe)
• Hemorrhagic contusion s/p trauma
• DAI (G-W jct; internal capsule; thalamus; CC esp body and splenium; dorsolateral upper brainstem)
• Coagulopathy
DDX of microbleeds

- Hypertensive bleed (deeper structures)
- Amyloid (cortical, older pts)
- Cavernomas (AV malformations; calcium)
- DAI (GRE preferred) s/p trauma
- Petechial hemorrhage of ischemic stroke
- CADASIL (auto dom arteriopathy)
Gunshot

• Missile effect
  • initial Laceration
  • Crushing of brain tissue by projectile
  • subsequent Cavitation
• Hemorrhagic tract along bullet trajectory
• Low density represents edema and infarction
• Penetrating (entry wound) vs perforating (entry and exit wounds; poor prognosis) injury
• Entry and exit wound sites
• Retained projectile or shrapnel (metallic fragments)
Parenchymal hemorrhage

<table>
<thead>
<tr>
<th>Hemorrhage</th>
<th>T1</th>
<th>T2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hyperacute (&lt;12 hr)</td>
<td>Isointense</td>
<td>Bright</td>
</tr>
<tr>
<td>Acute (12 hr - 2 days)</td>
<td>Isointense</td>
<td>Dark</td>
</tr>
<tr>
<td>Early Subacute (2 - 7 days)</td>
<td>Bright</td>
<td>Dark</td>
</tr>
<tr>
<td>Late Subacute (1 wk -2 mos)</td>
<td>Bright</td>
<td>Bright</td>
</tr>
<tr>
<td>Chronic (&gt;2 mos)</td>
<td>Dark</td>
<td>Dark</td>
</tr>
</tbody>
</table>

I = Isointense    B = Bright    D = Dark

“It Be IdDy BiDdy BaBy DooDoo”
**Hyperacute Hematoma (< 12 hours)**

- Oxyhemoglobin
  - Hyperacute hemorrhage (<12 hr)
  - 0 unpaired electrons - Diamagnetic
  - $T_1$ (iso), $T_2$/FLAIR (bright)
  - GRE (variable), DWI (bright)

**Acute Hematoma (12 hours - 2 days)**

- Deoxyhemoglobin
  - Acute hemorrhage (12 hr - 2 d)
  - 4 unpaired electrons - Paramagnetic
  - $T_1$ (iso), $T_2$/FLAIR (dark)
  - GRE (dark), DWI (dark)

**Early Subacute Hematoma (2 days - 1 week)**

- Methemoglobin (Intracellular)
  - Early subacute hematoma (2 d - 1 wk)
  - 5 unpaired electrons - Paramagnetic
  - $T_1$ (bright), $T_2$/FLAIR (dark)
  - GRE (dark), DWI (dark)
Late Subacute Hematoma
(1 week - 2 months)

Methemoglobin (Extracellular)
Late subacute hematoma (1 wk - 2 mo)
5 unpaired electrons - Paramagnetic
T1 (bright), T2/FLAIR (bright)
GRE (bright), DWI (bright)

Ferritin/Hemosiderin
Chronic hematoma periphery (>1-2 mo)
$10^2$-$10^6$ unpaired electrons - Superparamagnetic
T1 (bright), T2/FLAIR (dark)
GRE (dark), DWI (dark)

Chronic Hematoma
(> 2 months)

Hemichromes
Chronic hematoma center (> 1-2 mo)
0 unpaired electrons - Diamagnetic
T1 (dark), T2/FLAIR (bright)
GRE (bright), DWI (variable)
CT appearance of hemorrhage. Serial CT scans of right thalamic hematoma. (A) Acute ICH in the right thalamus with mean attenuation 65 HU. (B) CT performed 8 days later than (A); the periphery of the hematoma is now isodense to the brain while the center of the hematoma has mean attenuation 45 HU. (C) CT performed 13 days later than (A) shows continued evolution of the hematoma with decreasing attenuation. (D) CT performed 5 months later than (A) shows a small area of encephalomalacia in
HIE (global cerebral hypoxia)

• CT
  • White cerebellum sign (diffuse hemispheric edema sparing cerebellum and brainstem)
  • Reversal sign (WM higher intensity than gray matter; deep gray matter is dark)

• MRI
  • During the first 24 hours, there may be restricted diffusion in the cerebellar hemispheres, BG, or cerebral cortex (in particular, the perirolandic and occipital cortices); thalami, brainstem or hippocampi may also be involved
DDX: HIE, seizure, JC
CO poisoning

• Bilateral GP infarcts
• Ddx: Wilson’s dz etc
Mega cisterna magna

• Prominent retro-cerebellar CSF space
• Subarachnoid septa is seen within cistern
• No vermian or cerebellar anomaly
• No communication with 4\textsuperscript{th} ventricle (Dandy Walker malformation)
• No ventriculomegaly
• Tentorium cerebelli normally located
• Isointense to CSF on all sequences (including DWI)
Dandy Walker malformation

- Hypoplastic vermis (may also result in small cerebellar hemispheres)
- Cystic dilation of 4\textsuperscript{th} ventricle extending posteriorly
- Enlarged posterior fossa with torcular-lambdoid inversion (torcula or sinus confluence located above level of lambdoid suture due to high tentorium)
- May result in ventriculomegaly
PRES

• Patchy T2 and FLAIR hyperintensities within bilateral (asymmetric) occipito-parietal lobes
• Involves cortical and subcortical regions
• Negative DWI
• R/o dural venous thrombosis
Marchiafava Bignami disease

• High T2 and FLAIR signal intensity typically involving body of the corpus callosum, followed by the genu, and finally the splenium (entire corpus callosum may be also involved)

• Red wine (not just wine)
Intracranial hypotension

- Upright postural headache
- Reduction in CSF
- Findings
  - Diffuse thickening of the pachymeninges with enhancement or subdural effusion/hematoma in advanced cases
  - Engorgement of dural venous sinuses
  - Enlargement of the pituitary
  - Slit like lateral ventricles
  - AP elongation of mid brain
  - Sagging brain stem
  - Protrusion of cerebellar tonsils
Pseudotumor cerebri

• Now known as idiopathic intracranial hypertension (IIH)
• Increased opening pressure
• Prominent subarchnoid space around optic nerves and tortuosity
• Clinical papilledema (may see flattened optic disc at insertion of optic nerve on MRI bilaterally)
• Empty sella
• Slit like ventricles (not commonly seen)
• Focal compression-related pseudostenosis within bilateral mid transverse sinuses
Etiology of increased ICP

• Normal ICP=80-150mmHg

• Etiologies
  • IIH (pseudotumor cerebri)
  • Intracranial mass
  • Diffuse cerebral edema
  • Dural sinus thrombosis
Pyogenic abscess

- early involvement=cerebritis (edema and patchy enhancement)
- thin regular rim of enhancement (inner margin may be smoother than slightly irregular outer margin during early stages)
- may have adjacent daughter cyst (multiloculated on post-Gad)
- enhancing rim is thinner towards ventricle side and thicker towards gray-matter side
- light bulb bright on DWI centrally (dark ADC)—due to high viscosity pus
- intermediate low intensity T2 rim with bright centrally and surrounding vasogenic edema
- T1 dark centrally
Abscess

• T1 post-gad: Ring enhancing (smooth/regular non-nodular); hypointense surrounding vasogenic edema
• T1: central low intensity (hyperintense to CSF) and peripheral low intensity vasogenic edema
• T2/FLAIR: central high intensity (hypointense to CSF) and peripheral high intensity vasogenic edema and intermediate intensity capsule
• DWI: high DWI centrally (low ADC); may or may not have subtle surrounding hyperintensity (T2 shin thru from edema)
• GRE: low intensity rim (complete in 75%; smooth in 90%); may have dual rim sign (hyperintense line located inside low intensity rim)
• MRS: elevated lactate and succinate (more specific but not always present) peaks

• NOTE: typical appearance is seen with pyogenic abscess; tuberculoma and toxo have little restricted diffusion; fungal abscess have variable DWI
• DWI can be used to monitor response to therapy
Ring enhancing lesion

- Mets (irregular/nodular rim enhancement)
- Abscess (regular rim enhancement; T2 dark rim; increased lactate peak on MRS, DWI bright)—looks similar to Toxo and both can have adjacent satellite lesions
- GBM
- Infarct
- Contusion
- Demyelination (MS=incomplete rim enhancement of active plaque)
- Resolving hematoma
- AIDS (Lymphoma=subependymal; Toxo likes BG and periaqueductal gray)

- MS
- Abscess
- Toxo vs Lymphoma (AIDS)
- Mets
Enhancing ventricular margins ddx

- Ventriculitis/ependymitis (shunting; abscess; TORCH)
- Subependymal spread of CA
- CNS lymphoma
ADEM

• Acute disseminated encephalomyelitis
• 1-2 week viral infx or post-vaccination acute demyelination of WM
• Esp children and adolescent
• Multifocal subcortical WM lesions (may also involve thalami, brainstem, spinal cord) with punctate/rim/arc enhancement (open ring sign)
• Peripheral restricted diffusion (not central)
Cortical laminar necrosis

• T1 hyperintense gyriform staining on cortical grey matter associated with infarction
Subacute infarct

• Gyriform enhancement helps confirm subacute infarct when there is pseudonormalization of DWI
Subarachnoid hemorrhage (SAH)
Central pontine myelinolysis

- Centrally (Trident sign) within pons
- +restricted diffusion
- Rapid correction of hyponatremia (low N+)
- ODMS (osmotic demyelination syndrome) = pons + extra-pontine involvement (BG)
- No enhancement
- DDX: brainstem glioma, infarct
Wernicke’s encephalopathy

- Signal abnormality in peri aqueductal grey matter of mid brain, mammillary bodies, hypothalamus > paramedian portion of thalami
- Triad of Ataxia, Ophthalmoplegia (especially VI CN palsy) and Altered sensorium
- Thiamine deficiency in alcoholics
Multiple System Atrophy

• Hot cross bun (cruciform T2 hyperintensity within pons)
Cerebellar atrophy

- Alcohol (vermis)
- Chronic dilantin therapy for seizure (phenytoin)
- Multisystem atrophy (includes spinocerebellar degeneration)
- Friedreich ataxia
- Olivopontocerebellar degeneration
- Superficial siderosis
- Cerebellitis
- Paraneoplastic (oat cell)
- XRT
Spinocerebellar degeneration or atrophy

• Atrophy of brainstem and cerebellum
Cerebellar degeneration

• Cerebellum trophy (brainstem spared)
Wallerian degeneration

- Gliosis along the ipsilateral descending white matter tracts traversing through internal capsule, midbrain, pons, medulla
Huntington’s chorea

- Caudate head atrophy
- Boxcar like lateral ventricles

(a) Control
- Caudate nucleus
- Putamen

(b) Patient with Huntington’s disease
- Lateral ventricles
Joubert syndrome

• Underdeveloped or absent vermis
• Molar tooth mid-brain
• Batwing appearance of 4th ventricle
Progressive Supranuclear Palsy

• Hummingbird midbrain on sagittal view
• Parkinsonian features and vertical gaze abnormality
Fahr’s dz

- Bilateral symmetric calcification in cerebral sub cortical white matter, caudate nuclei, lentiform nuclei, thalami and dentate nuclei of cerebellum
Sturge Weber

- Left hemiatrophy
- Dense cortical gyriform calcification
- Leptomeningeal enhancement
- Poor development of ipsilateral hemicranium
- Thickened ipsilateral bony calvarium
- Enlargement of ipsilateral choroid plexus
- Hyperpneumatisation of frontal sinus
CC agenesis

- Parallel non converging widely separated lateral ventricles on axial
- Splenium of CC (posterior) not visualized
- Occipital horn dilated – Colpocephaly
- Pointed or trident shaped frontal horns.
- Non visualisation of normal stripe of CC on mid sagittal sections, gyri and sulci directly radiating from roof of third ventricle
- High riding third ventricle
- Absent cingulate gyrus which is normally seen parallel and cranial to CC
- Vertically originated hippocampi with key hole appearance of temporal horns on coronal sections
- Probst bundles, the abnormal longitudinally orientated band of white matter track medial to lateral ventricles seen on coronal sections
- May seen an associated lipoma or inter hemispheric cyst if any
CC agenesis with lipoma
Superficial siderosis

• Abnormal hemosiderin staining of subarachnoid space (may be diffuse or focal)

• Commonly overlying cerebral and cerebellar convexity, basal cisterns, ventral surface of brain stem on GRE

• Results from excessive and repetitive subarachnoid bleed
Acute cerebellitis

- Diffuse faint DWI and FLAIR swelling of cerebellum
DDx for sulcus hyperintensity on FLAIR

- SAH (may have hydrocephalus; if have blood in ventricles)
- Meningitis (may have hydrocephalus; ventricles are clean)
- Granulomatous infx (TB/fungal) and sarcoidosis
- Carcinomatosis
- Recent myelogram (iodinated contrast)
Mesial temporal sclerosis

• Hippocampal atrophy with abnormal T2/FLAIR hyperintensity

Other findings:
• Enlarged ipsilateral temporal horn of lateral ventricle
• Atrophy of fornix and mamillary body
• Loss of hippocampal head digitations
• Atrophy of white matter in parahippocampal gyrus
• Increased T2 signal in anterior temporal white matter
Subdural hygroma

- Acute trauma or post-op (can be idiopathic in kids)
- Subdural fluid (isodense to CSF)
- Due to subarchanoid membrane tear
- Usually bilateral and more or less symmetrical
- Main ddx
  - chronic subdural hematoma (usually unilateral)
  - Also look for spontaneous intracranial hypotension
Empyema

- Subdural or epidural (epidural crosses midline but not sutures)
- Paranasal sinusitis and mastoiditis
- Extra-axial low-intensity fluid with rim-enhancing dural/meninges
- Fluid has restricted diffusion (+DWI)
- Look for complications like cerebritis/abscess, OM (+/- extension into subgaleal space), venous thrombosis
- Ddx: chronic hematoma
Empyema

Axial T1WI shows hypointense subdural fluid collection along the right fronto-parietal convexity (small arrows) with minimal air-fluid level (arrowhead).

The right subdural fluid collection is hyperintense in axial T2WI (arrows).

Axial post-contrast T1WI shows ring enhancement of the right subdural fluid collection (arrow) with associated meningeal enhancement.

Axial diffusion weighted images shows increased signal of the right subdural fluid collection (arrow) with low apparent diffusion coefficient.
Septic emboli

- Multiple ring-enhancing lesion at G-W jct (mimic mets)
- +DWI
- May have associated infarcts and mycotic aneurysms
Watershed infarcts
Cystic encephalomalacia

- CSF density area with septations and cavitations
- Sequela of infarct, infx, trauma
Other cysts

- Porencephalic cyst* (communicates with CSF space; has rim of gliosis)
- Neuroglial cyst* (frontal>temporal lobe)
- Ependymal cyst (intraventricular)
Herpes encephalitis

- Bilateral asymmetric medial temporal lobe and insular cortical edema on FLAIR (also DWI+)
- Also involves inferior frontal lobes
- BG and thalami are classically spared
- +enhancement
- Can be hemorrhagic
HIV encephalitis vs PML

**HIV encephalitis**
- Diffuse periventricular WM hyperintensity
- Symmetric bilateral
- No enhancement and DWI
- Cortical atrophy and ventricular enlargement can be seen

**PML**
- Focal asymmetric multifocal WM abnormality along subcortical u-fibers (usually frontoparietal; can extend into BG; can be bilateral)
- Lesions may coalesce
- May involve both cerebrum and cerebellum (minimal or no mass effect)
- No enhancement and DWI
- Focal neuro symptoms
- Rapid progression and deadly within 6mos
- Paradoxical worsening with HAART tx may be seen
HIV infx

• Toxo
• CMV
• TB
• Cryptococcus
Toxoplasmosis

- BG and periventricular multiple ring-enhancing lesions
- Vasogenic edema (may be hemorrhagic)
- Main ddx in HIV pts is lymphoma esp if solitary and ring-enhancing

Toxo vs Lymphoma
- Lymphoma is usually single lesion with solid enhancement
- Thallium negative (positive if lymphoma)
- Decreased cho peak on MRS (increased cho peak if lymphoma)
- Decreased rCBV on perfusion (increased rCBV if lymphoma)
Cryptococcus

- Gelatinous pseudocysts (thick-walled and septated) in VR space along BG bilaterally (no enhancement)
- Leptomeningeal enhancement
Cysticercosis

• 3phases:
  • Vesicular (cyst with dot sign; dot or scolex may be T1 bright but no enhancement)
  • Colloidal (rim-enhancing with turbid fluid hyperintense than CSF; with surrounding edema; scolex may be seen as eccentric dark focus on T2)
  • Granular (edema decreases; cyst retracts; decreasing enhancement)
  • Nodular (calcified; no edema or enhancement; signal drop out on T2 and T2*)

• Can be intra-ventricular
<table>
<thead>
<tr>
<th>Stage</th>
<th>Type</th>
<th>MRI Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>First</td>
<td>Vesicular</td>
<td>Cyst + Scolex Non enhancement</td>
</tr>
<tr>
<td>Second</td>
<td>Coloidal</td>
<td>Ring enhancement Edema</td>
</tr>
<tr>
<td>Third</td>
<td>Granular nodular</td>
<td>Decreased enhancement and edema Begins calcification</td>
</tr>
<tr>
<td>Fourth</td>
<td>Involution</td>
<td>Obvious calcification on CT and MRI (T2^*WI)</td>
</tr>
</tbody>
</table>

Table 1. Stages of cysticerci on MRI.
TB

• Basilar leptomeningeal enhancement (basilar cisterns along skull base)
• Enhancing (solid or ring) parenchymal nodules (Tuberculomas)
• Vasculitis (may result in infarcts)
Cerebral Palsy (CP)

• Aka spastic quadrapalegic (paraplegic=LE only; hemiplegic=one side more than other; quadriplegic=all limbs)
  • Spastic 75%
  • Ataxia 15%
  • Dyskinesia 5%

• MRI findings
  • PVL (hypoxic-ischemia insult)
  • Porencephalic cyst
  • Infarction
  • Lissencephaly or Polymicrogyria (neuronal migrational anomalies)
  • Disordered WM development
  • Subcortical WM lesions
  • NF
  • TORCH
TUMORS
DDX

• **SIGNS OF EXTRA-AXIAL TUMORS**: CSF cleft (on T2); displaced subarachnoid vessels; cortical gray between mass and white matter; broad dural base; bony reaction

• **Tumors positive on DWI**: restricted diffusion suggests hypercellularity; epidermoid, abscess, cellular tumors (lymphoma/meningioma/PNET etc), mucinous mets, oligodendroglioma

• **High T1 tumors**: blood products (pit apoplexy, hemorrh mets, thrombosed aneur); fat (dermoid cyst, lipoma); cholesterol (colloid cyst); melanin (melanoma mets); slow flow; paramag (cu, mn)

• **Low T2 tumors (hypercellularity, calcium, blood, protein, melanin, flow-voids)**: lymphoma, medulloblastoma, pineoblastoma, PNET, mucinous mets, melanoma mets, colloid cyst, old hemorrhage in tumor or vascular malformation

• **Tumors containing calcium**: meningioma, oligodendroglioma, craniopharyngioma, choroid plexus tumor, ependymoma

• **Dural based tumors**: mets (breast/lung/prostate/neuroblastoma/RCC), chondrosarcoma, meningioma/hemangiopericytoma, lymphoma, sarcoidosis

• **Dural tail**: meningioma, mets, lymphoma

• **Cortical-based or tumor involving gray matter (peripherally located; may present with seizure)**: DNET (esp if bubbly), Oligodendroglioma (calcification; usually not <30yo); Ganglioglioma/gangliocytoma (cystic with enhancing nodule); PXA; ddx= infarct, cerebritis, post-ictal; herpes encephalitis

• **Tumors attached to septum pellucidum**: central neurocytoma (enhances), ependymoma (does not enhance)

• **Intraventricular tumors**: ependymoma, subependymoma, CPP, central neurocytoma, colloid cyst, meningioma, GCA

• **Tumors w/ CSF dissemination (tumoral nodules on surface of brain and spinal cord)**: PNET, medulloblastoma, pineoblastoma, ependymoma, GBM, lymphoma, choroid plexus papillomas
DDX

- **Peds tumors:** JPA, medulloblastoma, PXA, craniopharyngioma, choroid plexus papilloma, PNET
- **Brain mets in kids:** Wilms, neuroblastoma, rhabdomyosarcoma
- **Supratentorial tumors in kids:** astrocytoma, PXA, PNET (infants), DNET, ganglioglioma, craniopharyngioma, pineal tumors
- **Infratentorial tumors in kids:** JPA (1/3), medulloblastoma (1/3), brainstem glioma, ependymoma (least common), CPP
- **Cystic tumor w/ mural nodule:** JPA, PXA (temporal lobe), ganglioglioma (temporal lobe), hemangioblastoma (post fossa), DNET
- **Fat-containing lesions:** lipomas, dermoid cyst, teratoma
- **Calc-containing tumors:** astrocytoma (20%), oligo (90%), mets; ependymoma (50%), CPP (25%), ganglioglioma (40%), menigioma, craniopharyngioma (90%), chordoma, chondrosarcoma
- **Hemorrhagic tumor:** mets (lung, RCC, melanoma, chorioCA, GBM, oligo)
- **Brain mets (can be solitary within hemisphere>cerebellum>BG or brain-stem):** lung, breast, renal, GI, melanoma
- **Skull base tumors:** chordoma, chondrosarcoma, mets/myeloma, lymphoma, esthesioneuroblastoma, paragangioma, sinonasal CA
- **Sellar/Parasellar tumor:** pituitary adenoma, rathke’s cleft cyst, craniopharyngioma, menigioma, dermoid, epidermoid, germinoma, schwannoma, chiasmatic glioma, menigioma, mets
- **CPA tumor:** schwannoma (8th>>5th), arachnoid cyst, menigioma, epidermoid, mets, paragangioma
- **Pineal tumor:** pineal cyst, pineocytoma, germ cell tumor (teratoma/germinoma), PNET, tectal glioma, menigioma, dermoid, arachnoid cyst
- **Enhancing pineal mass:** pineocytoma, pineoblastoma (kids), germinoma (engulfs calcs; females), teratoma, menigioma, VOG aneurysm
Ill-defined cortical based lesions

- **Infarct** (early=cytotoxic edema; late=volume loss; do DWI; may see T1 bright tram-track “laminar necrosis” or “petechial hemorrhage”)
- **Infx** (cerebritis is more diffuse like meningitis, encephalitis is more focal like Herpes; correlate w/ history and lumbar puncture)
- **Tumor** (Infiltrating glioma, lymphoma, mets; give Gad; increased Ch:Cr on MRS)
- Contusion/hemorrhage (CT hyperdense and susceptibility artifact on GRE)
- **TS** (cortical/subcortical tubers)
- Extra-axial meningioma=mimicker
Dense (CT) cerebral mass

• Vascular (aneurysm hematoma, AVM)
• Tumor (medulloblastoma, meningioma, lymphoma, mets)
Calcified Cerebral mass

- Meningioma (extra-axial)
- Oligodendroglioma
- Vasc malformation
- Aneurysm
- Laminar necrosis (prior infarct or XRT)
- Interhemisphere lipoma w/ calcs
- Craniopharyngioma, Teratoma
- TS, and SW in kids
- Choroid plexus papilloma
- Ependymoma
Ring enhancing lesion

- Mets
- Abscess (DWI+)
- GBM
- Infarct (subacute)
- Contusion
- Demyelinating plaque (active; tumofactive MS)
- Resolving hematoma
- Toxoplasmosis vs Lymphoma (immuno-compromised)
Lesions at GW jct

- Mets
- Septic emboli
- DAI
- Cysticercosis
- Vasculitis
CC lesion

- GBM
- Lymphoma
- MS/PMLE/Adrenoleukodystrophy/Marchiafava Bignami
- DAI
- Mets
CNS tumor at birth

- PNET
- Medulloblastoma
- Ependymoma
- Teratoma
- CPP/CP CA
- Craniopharyngioma
- Hypothalamic glioma

<2yo=suratentorial; 2-15yo=post fossa; adults=supratentorial
Extra-axial tumor

• Mets (dural based)
• Meningioma (dural tail, hyperostosis, calcs)
• Arachnoid cyst (follows CSF) vs Epidermoid (high signal on DWI and FLAIR)
• TB, sarcoid, lymphoma/leukemia
• Carcinomatosis
• Cysticercosis
Parasellar Mass

- **Adults**
  - Macroadenoma (encases carotid w/o narrowing it)
  - Meningioma (encases and narrows carotid)
  - Aneurysm
  - Mets

- **Kids**
  - Craniopharyngioma (cystic, calcs; young boys/older women; rarely can be completely intrasellar)
  - Teratoma
  - Glioma (optic/hypothalamic)
  - Hypothalamic Hamartoma (rare; no enhancement)
  - Rathke’s cleft cyst (usually intra-sellar)
  - Ectopic pituitary

- Lymphocytic hypophysitis (enlarged homogenously enhancing pituitary in post-partum women)
- Infundibulum (pituitary stalk) mass
  - **Adults**=TB, sarcoid, carcinomatosis
  - **Kids**=meningitis, EG, lymphoma/leukemia
T1 bright sellar/suprasellar lesion

• Hemorrhagic macroadenoma (pit apoplexy)*
• Craniopharyngioma* (calc, cystic, solid enhancement, bimodal)
• Teratoma*
• Rathke’s cleft cyst (T1 bright, no enhancement)
• Ectopic posterior pituitary (T1 bright; located along stalk)

*=may have fluid level
Cavernous Sinus mass

- Macroadenoma
- Meningioma
- Schwannoma
- Aneurysm
- CCF
Intraventricular mass

• Basilar tip aneurysm
• Mets
• Meningioma (atrium)
• Colloid cyst (dense CT, T1 bright, no enhancement)
• Giant cell astrocytoma (TS)
• Astrocytoma/Ependymoma/subependymoma (4\textsuperscript{th} vent, older pts, no sig enhancement)
• CPP (trigone=kids; 4\textsuperscript{th} vent=adults)
• Central neurocytoma (attached to septum pellucidum; multicystic “grapes”)
• Cysticercosis (T2 target sign with central high signal=sclex)
• Epidermoid cyst (look at FLAIR and DWI)
<table>
<thead>
<tr>
<th>Intraventricular neoplasms</th>
<th>Site</th>
<th>Epidemiology</th>
<th>CT</th>
<th>MRI</th>
<th>Contrast enhancement</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subependymoma</td>
<td>Entirely intraventricular mass</td>
<td>Occur at any age and in both sexes, but most commonly seen in middle age male</td>
<td>Isodense or hypodense</td>
<td>Homogenous</td>
<td>Lateral ventricular lesions show no or minimal enhancement, while the 4th ventricular lesions can enhance</td>
</tr>
<tr>
<td></td>
<td>Commonest site is the 4th and the lateral ventricles</td>
<td></td>
<td>Calcification is rare</td>
<td>T2:hyperintense</td>
<td></td>
</tr>
<tr>
<td>Central neurocytoma</td>
<td>Septum pellucid</td>
<td>Young adults</td>
<td>Calcification is very common</td>
<td>Heterogeneous with cysts and calcification</td>
<td>The solid components show enhancement</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>T2:isoaintense</td>
<td>T1:isoaintense</td>
<td></td>
</tr>
<tr>
<td>Subependymal giant cell astrocytoma</td>
<td>Foramen of Monro</td>
<td>Usually in young patients with tuberous sclerosis</td>
<td>Subependymal calcifications as part of tuberous sclerosis</td>
<td>T2:Hyperintense, T1:Hypointense</td>
<td>Intense enhancement</td>
</tr>
<tr>
<td></td>
<td>Posterior fossa: intraventricular. Supratentorial ependymoma is commonly paraventricular</td>
<td></td>
<td>Isodense heterogenous calcifications and cysts are common</td>
<td>T2:Hyperintense, T1:isoaintense</td>
<td></td>
</tr>
<tr>
<td>Choroid plexus papilloma</td>
<td>Commonest site is the region of choroid plexus in the lateral ventricle</td>
<td>Children younger than 4 years</td>
<td>Isodense or hyperdense and commonly calcify</td>
<td>Heterogeneous and lobulated with cysts</td>
<td>Intense enhancement</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>T2:Hyperintense, T1:isoaintense to hypointense</td>
<td>T2:Hyperintense, T1:isoaintense</td>
<td></td>
</tr>
<tr>
<td>Intraventricular meningioma</td>
<td>Commonest site is the trigone</td>
<td>Middle age, more in female</td>
<td>Homogenous</td>
<td>Homogenous</td>
<td>Intense homogenous enhancement</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Iso or hyperdense, or hyperdense calcification is frequent</td>
<td>T2:iso or hyperintense</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>T1:Hypo to isoaintense</td>
<td>T1:Hypointense</td>
<td></td>
</tr>
<tr>
<td>Gioma</td>
<td>Usually involves the corpus callosum</td>
<td>Middle age, and elderly more in male</td>
<td>Heterogeneous sometimes with hemorrhage</td>
<td>Heterogeneous</td>
<td>High grade tumors are enhancing.</td>
</tr>
</tbody>
</table>

Table 2: Differential diagnosis of subependymoma from intraventricular neoplasms
3rd ventricle mass

- Colloid cyst
- GC astrocytoma (TS)
- Aneurysm (basilar tip)
Pineal tumor

- Pineal calcifications rare before 6yo (60% >20yo)
- Parinaud’s syndrome (fixed upward gaze); obstructive hydrocephalus due to compression of aqueduct
- 60% germ cell tumor (most common germinoma; teratoma etc)
  - “Engulfed” central calcifications with diffuse enhancement
  - Fat/calc/hemorrhage seen with Teratoma
- 30% pineoblastoma/cytoma
  - “Exploded” peripheral calcifications
  - Pineoblastoma (kids; ill-defined; hyperdense CT; >3cm)
  - Pineocytoma (adults; well-defined; iso to hypodense CT; <3cm)
- 10% others
  - Pineal cyst
  - Tectal plate glioma (may not enhance)
  - Meningioma
Germinoma

• Most common pineal tumor
Petrous apex lesion

- Asymmetric pneumatization (non expansile; fat saturation)
- Petrous apicitis (look for mastoiditis)
- Cholestrol granuloma* (T1 and T2 bright, -DWI, no enhancement, can be expansile)
- Cholesteatoma (low T1, hiT2, +DWI, no enhancement, can be expansile)
- Epidermoid (follows CSF except for FLAIR)
- Mets
- Meningioma (enhancement)
- Trigeminal schwannoma
Clivus/Skull base mass

- Chordoma
- Chondrosarcoma
- Plasmacytoma
- Mets
- Lymphoma
- Meningioma
- Epidermoid (scallops brainstem)
Epidermoid vs Arachnoid cyst

- Epidermoid cyst is DWI+ (also not completely isointense to CSF on FLAIR)
- Arachnoid cyst is isointense to CSF on all sequences
Epidermoid
Arachanoid cyst
Tumerfactive MS

- Incomplete rim enhancement (open ring) on gray matter side
- Low rCBV on perfusion scan (unlike tumor)
- T2 shine thru on DWI (unlike abscess)
Colloid cyst

- 2/3\textsuperscript{rd} are hyperdense on NCCT
- T1 bright due to cholesterol content
- Bright on FLAIR
- No enhancement
- No hydrocephalus
Meningioma

- Hyperdense on CT and may be calcified
- Dural-based
- Low T2 (isointense to grey)
- +DWI
- Intense enhancement
- T2 CSF cleft
- Dural tail
- Hyperostosis
- Locations: para-sagittal, sphenoid wing, tentorial, cerebral convexity, suprasellar/parasellar, cavernous sinus, clival, optic nerve sheath, paranasal/olfactory/planus sphenoidale, posterior fossa, cerebellar/infratentorial, CPA, foramen magnum, intraventricular
- DDX: hemangiopericytoma, dural mets (breast, prostate, adenoCA lung, RCC), lymphoma (may have involvement of skull and overlying scalp soft tissues), plasmacytoma (assess for possible MM), neurosarcoidosis (pachymeningeal vs leptomeningeal)
Hemangiopericytoma (mimick meningioma)

- Mimics and hard to differentiate from meningioma
- Highly vascular so look for flow voids
- No hyperostosis, instead could see osseous erosion in some cases
- No calcifications
- High recurrence rate
Hypothalamic glioma

- Located just above suprasellar cistern
- Isointense to gray on T1
- Slightly hyperintense on T2 and FLAIR
- Subtle enhancement (distinguishes it from hypothalamic hamartoma)
Toxo vs Lymphoma

• Findings favoring Toxo
  • Multiple lesions
  • Involving BG
  • Intra-lesional hemosiderin staining on GRE
  • Rim enhancement
  • Eccentric nodule

• Findings favoring Lymphoma
  • Single lesion
  • Hyperdense on CT
  • Periventricular WM
  • Subependymal spread
  • Homogenous enhancement in immunocompetent but rim-enhancement in immunocompromised
Craniopharyngioma

- Multi lobulated, well circumscribed, sellar/suprasellar mass with solid and cystic component
- May have calcifications
Oligodendroglioma

- Chunky/heavy calcifications
- Mean age 40yo
- Cortical based within frontal lobe
- Variable enhancement
Medulloblastoma

- Midline (arises within vermis)
- 70% <20yo
- Grow in circumferential pattern and maintain rounded borders (unlike ependymoma which are pliable)
- Hyperdense CT
- Iso to hypo on T2
- +DWI
- Intense homogeneous enhancement
- CSF dissemination more common than ependymoma
- Very radiosensitive
Ependymoma

• 2/3 infratentorial (kids, adolescent, and young adults)
• 1/3 supratentorial (adults)
• More heterogenous than medulloblastoma
• 50% calc
• May have hemorrhage or cysts
• Heterogenous signal and enhancement
• Plastic tumor (insinuate via Magendie or Luschka foramens)
• CSF dissemination less common than medulloblastoma
• Recur after surgery
Subependymoma

- Rare
- Middle-aged to elderly
- Most commonly within 4\textsuperscript{th} ventricle or Frontal horn of lateral ventricle
- Well-defined lobulated mass
- Typically none to minimal enhancement unlike ependymoma
Central Neurocytoma

- Attached to septum pellucidum
- Bubbly appearance on T2
- May have calcs
- +DWI (hypercellular)
- Variable (usually moderate) enhancement
Ganglioglioma

• <30yo (80%)
• Temporal lobe with seizures
• Cystic with mural nodule (40%)
• May have calcs (50%)
• 50% enhance
PXA

- Adolescent or young adults
- Cystic with mural nodule
- Mural nodule adjacent to leptomeninges
- +enhancement
- May have dural tail
- Temporal lobe with seizure
DNET

- <20yo
- Well-marginated
- Wedge shaped cortical or subcortical
- Non-enhancing
- May appear septated
- Temporal lobe
Hemangioblastoma

- Infratentorial = cystic with mural nodule
- Supratentorial = solid
- Hypervascular
- Associated with VHL
CPP (choroid plexus papilloma)

- Rare
- Lateral ventricle in kids (trigone) <10yo
- 4\textsuperscript{th} ventricle in adults
- 25\% calcs
- Lobulated irregular margins
- Heterogenous intense enhancement
- Intralvesional flow-voids
- Cannot tell from choroid plexus CA (more aggressive and may have parenchymal invasion; almost always in lateral ventricle)
CP xanthogranulomas (cysts)

- Benign
- Usually within choroid in atrium
- 75% +DWI
Rathke cleft cyst

- Congenital
- Well-circumscribed
- 50% T1 hyperintense
- T2 bright
- No enhancement
- Pathognomonic 75% with small non-enhancing intracystic nodule (T2 hypointense)
Pineal cyst

- Usually <1cm
**Mylohyoid** is a sling above which is sublingual space and below is submental space (ant) and submandibular space (post)

**Digastric muscles (ant belly)** are below Mylohyoid and between 2 belly is submental space and laterally is submandibular gland
Contrast-enhanced cervical CT shows heterogeneous attenuation area, poorly defined, in relation to changes flemonosos in the floor of mouth with extension into the left submandibular space, causing ipsilateral pharyngeal space obliteration and displacement of the lateral wall of the pharynx to the right. Associated collections on the floor of the mouth.
Ludwig’s angina

• Usually seen in 20-50yo
• Ludwig angina is a rapidly-spreading life-threatening cellulitis (phlegmonous changes) of the floor of mouth, involving the submandibular, sublingual and submental spaces
• Most cases (85%) are thought to originate from an untreated odontogenic infection (usually 2nd or 3rd molar)
• Due to the anatomy of the submandibular, sublingual and submental spaces and the mylohyoid muscle, spread of infection can occur between these spaces. Subsequent swelling can displace the tongue superiorly and posteriorly leading to potential airway obstruction and asphyxiation. When severe, the floor of mouth swelling can also cause trismus, odynophagia and dysphagia
• Mylohyoid muscle: above it is sublingual space and below it is submandibular space
• When left untreated, this infection can spread inferiorly into the neck and mediastinum
• Complications
  • Abscess, septicemia, airways obstruction
  • OM of mandible
  • IJ thrombophlebitis (Lemierre syndrome)
Ludwig’s angina

• aka Submandibular cellulitis
• Severe form of cervicofascial infection that arises from mandibular 2\textsuperscript{nd} or 3\textsuperscript{rd} molars (wisdom teeth)
• May lead to septicemia and airway obstruction
• Clinically present with tender neck swelling, fever, and septicemia
• Usually involves sublingual (oral cavity), submandibular, or parapharyngeal space (ascends up from retromolar trigone up along pterygomandibular raphe into retromaxillary fat)
Orbital pseudotumor

- Idiopathic inflammatory lesion of the orbit most commonly involving the extra-ocular muscles (most commonly the lateral rectus muscle), but can involve any of the orbital contents including the retro-orbital fat and the lacrimal gland.
Thornwaldt cyst vs Lateral epipharyngeal cyst
Retropharyngeal suppurative adenopathy

- Mimics retropharyngeal abscess
Cholestatatoma vs cholesterol granuloma

<table>
<thead>
<tr>
<th></th>
<th>Cholestatatoma</th>
<th>Cholesterol Granuloma</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>T1</strong></td>
<td>Hypo intense</td>
<td>Hyper intense</td>
</tr>
<tr>
<td><strong>T2</strong></td>
<td>Hyper intense</td>
<td>Hyper intense</td>
</tr>
<tr>
<td><strong>DWI</strong></td>
<td>restricted diffusion</td>
<td>No restricted diffusion</td>
</tr>
<tr>
<td><strong>Post contrast T1</strong></td>
<td>Non enhancing</td>
<td>Enhancing</td>
</tr>
<tr>
<td><strong>CT</strong></td>
<td>Erosion and destruction of ear ossicles, blunting of scutum.</td>
<td>Absent</td>
</tr>
</tbody>
</table>
SPINE
Syrinx

- Syringohydromyelia
- Syrinx (secondary to spinal cord tumor or arachnoiditis)
- Hydromyelia (dilatation of central canal associated with chiari or foramen magnum tumor)
- Syringobulbia (extends into brainstem)
- Cord cyst

<table>
<thead>
<tr>
<th>Hydromyelia</th>
<th>Syringomyelia, syrinx</th>
<th>Syringobulbia</th>
</tr>
</thead>
</table>
| Expansion of the ependymal-lined central canal of the cord | Formation of fluid-filled cleft-like cavity in the inner portion of the cord  
- Seen in Chiari malformations  
- Associated w/ intraspinal tumors  
- Distinctive SSx:  
  - Isolated loss of pain and temperature sensation in the upper extremities | Extension of syringomyelia to the brainstem |

**HISTOLOGIC APPEARANCE:**
- W/ destruction of adjacent gray and white matter
- Surrounded by dense feltwork of reactive gliosis

**EPIDEMIOLOGY:**
- Manifests in 2nd or 3rd decade
CSF flow artifact dorsal to thoracic cord in roomy spinal canal
Gibbs phenomenon (truncation) artifact simulating syrinx within thoracic cord
Spinal dural AVF
Spinal dural AVF

- 50-60yo (M>F)
- Present with progressive pain and LE weakness and sensory changes
- MRI
  - Enlarged edematous cord esp in thoracic spine
  - Serpiginous dorsal intradural, extramedullary T2 flow void usually but not always present (if large may scallop surface of cord)
  - May see T2 hypointensity along periphery of cord—distended pial capillaries due to venous hypertension
  - T2 hyperintensity involves conus in 90% due to orthostasis
  - May have patchy intramedullary enhancement due to chronic venous hypertension
Ventriculus Terminalis (of conus medullaris)

- Terminal ventricle (5th ventricle)
- Widest part of central canal of spinal cord located near conus
TM = transverse myelitis
NMO = neuromyelitis optica

Short segment
- MS
- TM (uncommon)

Partial
- MS

Enhancement
- TM
- Tumor

Swelling +/
- MS
- ADEM

Swelling ++
- TM
- Tumor

Long segment
- TM
- NMO
- Ischemia
- Both halves

Short segment myelopathy
Long segment myelopathy
Cord tumor (intramedullary)

• Short segment (usually 2 vert seg or less)
• Well-delineated T2
• Central cord
• Cord expansion!!
• May have enhancement (enhancement is always concerning)
• May have associated syrinx
• DDx: astrocytoma vs ependymoma
Transverse myelitis

- Idiopathic inflammatory d/o
- Long segment contiguous intramedullary lesion with cord swelling
- Ill-defined T2 signal
- More common in T-spine (10% in C-spine)
- Characteristically involves central cord (MS plaques are dorsal and lateral and span only 2 or less vertebral segments) -- >2/3rd of cross-section of cord
- May have none to subtle patchy or peripheral enhancement
Guillain Barre

- Enhancing nerves roots of cauda equina (thick nodular vs smooth) is non-specific
  - Acute or Chronic inflammatory demyelinating polyneuropathy
  - Arachanoiditis
  - Granulomatous infx (TB, sarcoid)
  - Infiltrative neoplastic d/o like mets or lymphoma

- GBS
  - Ascending paralysis
Arachanoiditis

- Abnormal clumping of cauda equina