Search Pattern

Approach to MRI brain
MRI BRAIN ANATOMY

Preassessment checks
- Sagi T1 for anatomy, incidental mass lesions, T1 signal abnormalities
- DWI and SWI for ischemia and hemorrhage
- T2s for CSF space lesions, vessel flow voids, abnormal signal in the face/neck, paranasal sinuses, mastoid air cells and ear
- FLAIR for brain / cord parenchymal lesions

Hx, indication, prior
Scout/localizer
- Look outside the brain

Assess adequacy technique, limitations
- Field strength, scan parameters to evaluate for pathology
- Key sequences for artifact
- Repeat suboptimal / nondiagnostic sequences

T1

Assess midline structures in the brain/ cord
- Cord/thecal sac -> brainstem -> tegmentum -> Thalamus -> Mammillary body -> infundibulum -> neurohypophysis -> pituitary gland -> optic chiasm
- Cerebellar tonsil position -> obex -> cerebellum (posterior lobe-> anterior lobe) -> aqueduct -> tectum & sup & Inf colliculus -> pineal gland -> posterior commissure
- Corpus callosum (rostrum -> splenium) -> cingulate gyrus -> midline gyri

Assess paramedian and further lateral brain parenchyma
- Ventricles
- Sulci / cortex
- Insula and sylvian fissures
- Assess for abnormal T1 signal in vessels

Look for masses and abnormal / displaced fat signals.
- Check nasal cavity, nasopharynx, oral cavity, oropharynx, base of tongue
- Check globes, paranasal sinuses, mastoid air cells
- Check parotid / submandibular glands
- Check superficial and deep spaces in the face / neck

Assess the bones, especially for preservation of marrow signal

- Examine vertebral bodies, SC, post elements, mandible, calvarium, clivus / skull base

DWI and ADC

Scroll through each lobe of the brain and look at post fossa / brain stem -> extracranial ST.

- Match each DWI hyperintensity with ADC hypointensity
- B0 show blooming similar to SWI sequence.
- High B value images may allow you to detect marrow lesions and other high risk lesions outside the brain.

SWI

LINKS

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- General: sensitive to hemorrhage, calcium, metals, air, and bone; subtle mets
    - Very good for detection of SAH central in brain but not good at peripheral SAH 2/2 low signal in adjacent calvarium.
- Scroll through each lobe of the brain and post fossa / brain stem and extracranial ST separately

T2/FLAIR

Assess CSF spaces

- Look for SAH in 5 regions: apex, dependent sylvian fissures, occipital horns, interpeduncular cistern, foramen magnum.
- Check sulci / convexities, ventricles, basal cisterns, IACs, and Meckel's caves

Check the vessels for preserved flow void, course / caliber, aneurysm / malformations

- Arteries: ICA / ECA, MCA / ACA / PCA
- Veins: dural sinuses / veins, cavernous sinus (blind spot)

FLAIR

Can identify extraaxial hemorrhage, pus and proteinaceous debris.
Best for brain / brain stem parenchyma lesions.
A type of T2WI but CSF is black.
FLAIR shows periventricular and juxtacortical areas very well. Inferior quality to other sequences in posterior fossa and SC.

- Assess the cerebrum, GM / WM, BG, post fossa, brain stem
- Coronal FLAIR images are best for the hippocampus. Also useful for assessing the calvarium, parotid glands, and other parts of the neck.

T1 Post Contrast +Gad +DTPA

- General:
    - Shows BBB disruption
    - Look for enhancing lesions and compare them to T1 precontrast to make sure the signal is not inherently T1
    - Use coronal / sag images for superior cerebellum and post fossa structures. Clivus and spine are best on sagittals.

Check all anatomy: scalp, face / neck, CSF, brain, bones.
Look carefully for subtle leptomeningneal enhancement
Check all surfaces of the brain.

- Blind spots include undersurface of brain and cerebellar folia.
- Look for subtle enhancing lesions at GWJ.
- Check neck / nasopharynx, and other other extra axial structures

If post contrast images are thin-cut / isotropic, these may provide the highest resolution view of the vessels. Assess the arteries and veins / sinuses for aneurysm, malformation, stenosis etc.

Characterize all findings across provided sequences as appropriate

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Two ways to image brain:

- Routine non-contrast brain: Diffusion (DWI, ADC), sagittal T1, Axial flair, T2, Gradient/SWI, axial T1.
- Routine contrast brain: everything above + post contrast T1 (spin echo thick slices or gradient echo thins)

MRI Brain

DWI:

- Raw diffusion comes across as a series of raw values. NOT ALL OF THEM ARE DIFFUSION WEIGHTED IMAGES. Will have very bright water. These are not DWI so to speak, they have a P value of 0 so not weighted towards water. You are seeing underlying T2 properties.
- Good tool for hemorrhagea nd blood products.
- B100: almost everything will be dark with very little signal from skull base and bones. Window until you see no air. ANy area where there is an interface between air and tissue there will be very bright. These are not abnormal, just artifact of sequence. Metal and calcium will show.
- ADC: Anyareas of reduced diffusion will be dark. CSF bright and vitreous of eyes. Typical measurement of fluid will be about 3000.

Precontrast T1 (sagittal and axials)

- WM is brighter than GM (unlike T2 and FLAIR) CSF structures and globes are dark.
- Midline is most important. Optic chiasm, optic tract pituitary and infundibulum, posterior pit normally has T1 hyperintensitiy (neurohypophysis).
- Always assess the marrow signal of the calvarium, skull base, visualized face, and upper spine
    - Marrow replacement / mets are extremely easyt to miss
- Always check midline structures. You should also return to these on post-contrast images to assess enhancement characteristics
    - Look at the pituitary for lesions and the normal “bright spot”

FLAIR and Fat sat T2

- FLAIR: all fluid suppressed. Anything close to water is suppressed. Look at GWD. If WM is darker than overlighting gray matter you are looking at T2. Very good for looking at parenchymal disease.
- T2: less noise in posterior fossa.

GRE and T2

- GRE: bone is black. Blooming around bone tissue. Air , calciu, blood products will be dark. No correction for local heterogenitiy making it senstiivte for blood products.

T1 and FLAIR

- T1: CSF is dark. WM is brighter than GM.
    - Good for ca, blood products, melanin.

        - Can call something contrast enhancing if it is inherently T1 bright.
    - Flair: WM is darker than GM. most reliable way of telling the difference.

T1 precontrast and postcontrast

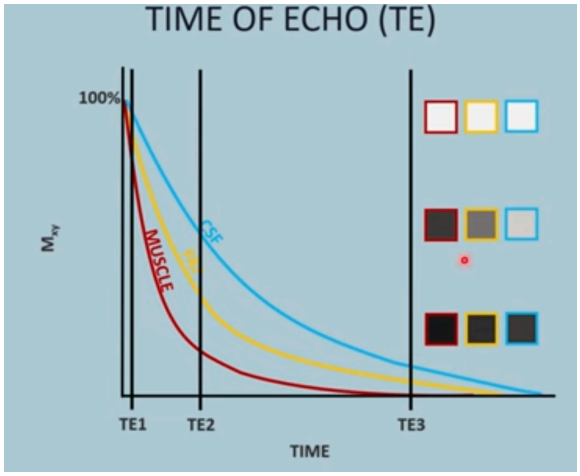
- Look at nasal mucosa. Dark on precontrast and bright on postcontrast. Can also look at pharyngeal mucosa.
- Postcontrast:
    - Used to look for tumors, inf, active inflammation.
    - Normal brightness: enhancement of pituitary infundibulum, pathology of WM and disruption of BBB, will have leakage of contrast into parenchyma.

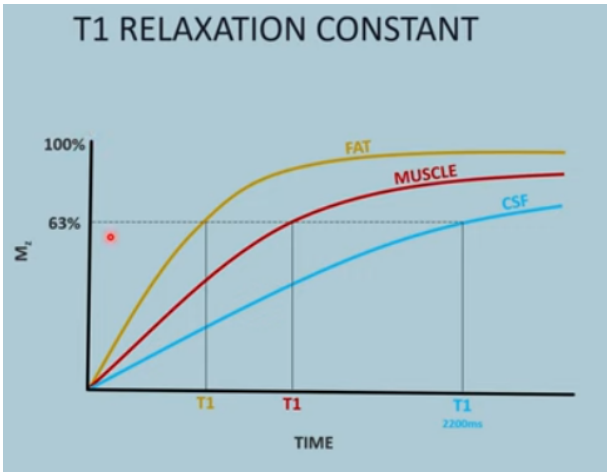
3D T1 Sagittal and axial

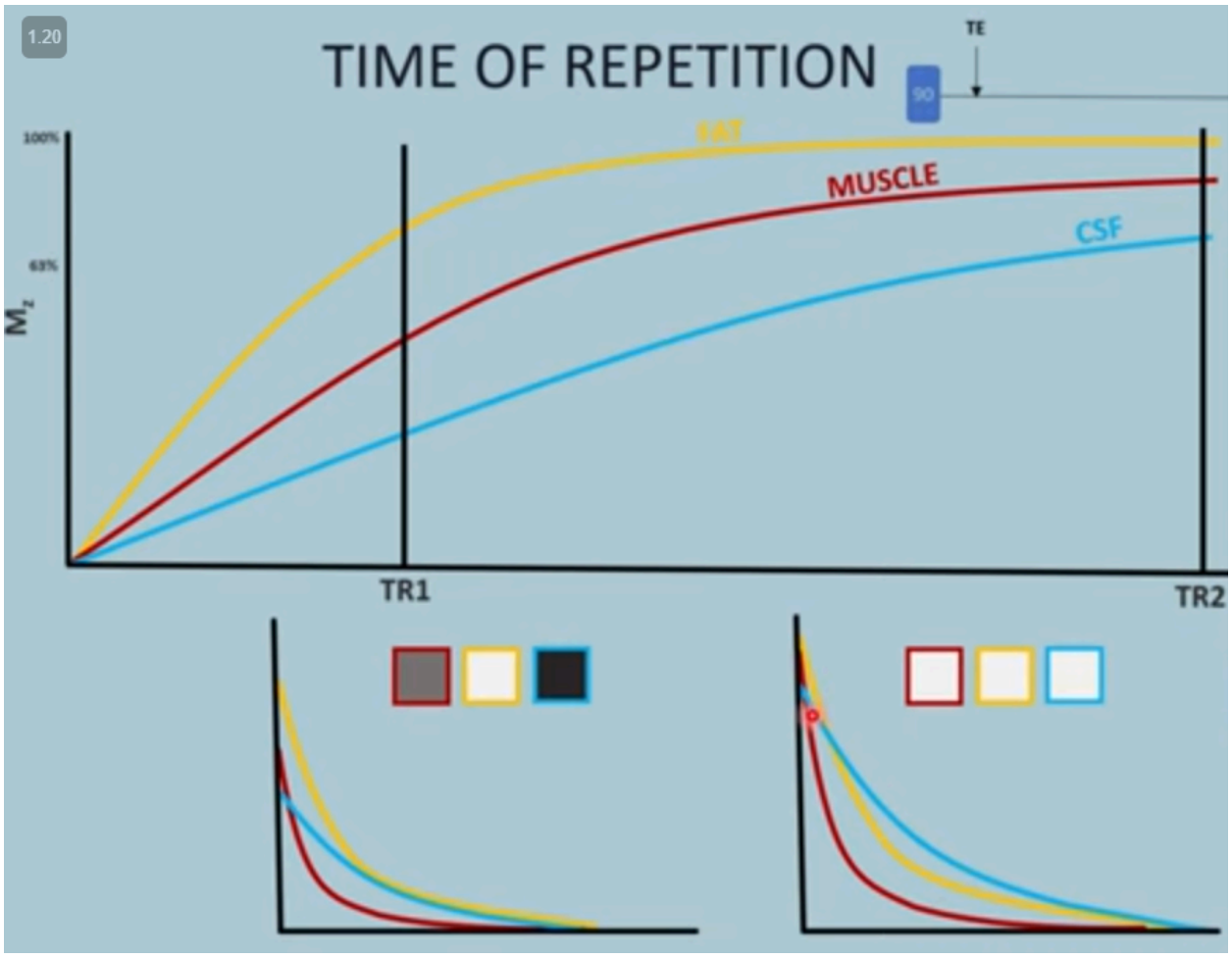
- Slices are thin usually 1 mm. Good sensitivity for small lesions. Also more noise then normally seen. Get very detailed view of brain structures. Less contrast sensitivity but made up with good spatial resolution.
- Good for small contrast enhancing lesions, mets, and reformatting into other planes.

MRI General Overview

BRAIN TUMORS
Notes from Duke Review of MRI principles
MRI physics overview | MRI Physics Course | Radiology Physics Course #1
MRI Machine - Main, Gradient and RF Coils/ Magnets | MRI Physics Course | Radiology P…







<u>T1WI</u>

- T1 represents the efficiency of energy transfer from a resonating proton to its lattice following energy deposition by an RF excitation pulse.

- Molecules that move at a motional frequency similar to Larmor frequency quickly transfer energy to the lattice -> short T1 relaxation time -> increased signal on a T1WI.
    - Fat, a medium sized molecule is an example.
- Small molecules (bulk water) and large molecules (proteins) exhibit motional frequencies much different than the Larmor frequency -> inefficient energy transfer to the lattice -> long T1 relaxation times.
- MR images have a different mix of contrasts (T1, T2, PD) that operators can manipulate to promote a particular weighting to emphasize differences of one particular tissue contrast while minimizing the others.
- In SE sequences, a moderate TR (~600 msec) and a shortest possible TE (<10 msec) produces a T1W image. In GRE sequences, an excitation pulse w a large flip angle, short TR and shortest possible TE produces a T1WI.
- In a T1W sequence, tissues w short T1 relaxation times produce high signal (fat, gad enhanced tissues, proteinaceous fluid), while tissues w long T1 times produce low signal (free water, CSF).
- T1W sequences provide good anatomic info and are used to evaluate enhancing tissues following IV gad.
- Most pathologic tissues are low signal on noncontrast T1W images 2/2 T1 relaxation prolongation from increased extracellular water concentration.
- T1 shortening refers to the reduction of normal T1 relaxation of tissues due to the effects of external agents, causing them to produce high signal on T1WI
- Paramagnetic agents exhibit unpaired electrons that produce a strong local magnetic field and induce both T1 and T2 relaxation of nearby protons. Examples of paramagnetic agents include gad, methemoglobin, and melanin.
- The hydration layer effect refers to the binding of water protons to the surface of macromolecules (proteins) which slows down the motion frequency of water protons, making energy transfer to the lattice more efficient and leading to a reduction in its T1 relation.
- Other less common agents of T1 shortening include ionic Ca, manganese and free radicals.

T2WI

- T2 relaxation is the process where transverse magnetization loses phase coherence which is an exponential decay at the rate of T2
- Loss of transverse mag is primarily due to phase dispersion (loss of phase coherence) among the precessing protons.
- Loss of phase coherence is 2/2 inhomogeneities in the main magnetic field and the spin-spin interactions in the protons' local microenvironment; together these create T2 effects.
- Free induction decay is the oscillating waveform that occurs hen the RF pulse is turned off and reflects the decaying transverse mag. T2 effects determone the rate at which the FID occurs.
- T2 is the loss of mag solely 2/2 proton spin spin interactions w/n protons microscopic environment.
- T2 time for a tissue is the time at which 63% of the original signal is lost during the exponential T2 decay.

- Inherent tissue T1 and T2 characteristics depend on changes to protons within tissue:
    - T1WI: longitudinal recovery / relaxation
        - T1 shortening is hyperintense (bright) / **T1SB** (1 SOB)
        - T1 prolongation is hypointense (dark) / T1PD
    - T2WI: transverse relaxation
        - T2 shortening is hypointense (dark) / T2SD (takes 2 to suck dick)
        - T2 prolongation is hyperintense (bright) / **T2PB**
- The MR image exhibits signal abnormalities rather than the proton relaxation times.
    - A lesion is hyperintense on T2WI or lesion demonstrates T2 prolongation
- Most brain lesions are dark on T1WI and bright on T2WI = T1PD and T2PB

Spin echo T1

- Most brain lesions are hypointense on T1WI 2/2 pathologic prolongation of longitudinal recovery. **The presence of hyperintensity on T1WI is an important clue to specific diagnosis**.
- Causes of T1SB
    - MC: gadolinium, fat, proteinaceous substance.
    - Some paramagnetic stages of blood (intra and extracellular methemoglobin)
    - Melanin
    - Mineralization (copper, iron, manganese)
    - Slowly flowing blood
    - Calcium (rarely, when dispersed, not in bone)
        - Much more common for Ca to be hypointense

Spin echo T2

- Most brain lesions are hyperintense on T2WI. Water has a very long T2 relaxation constant (very bright on T2). Edema is a hallmark of many pathologic processes and causes T2 prolongation. Since most lesions are T2PB, T2SD
- Causes of T2SD:
    - Most paramagnetic stages of blood (except hyperacute blood and extracellular methemoglobin)
    - Calcification
    - Fibrous lesion
    - Highly cellular tumors (high nucleus: cytoplasm ratio producing low lesional water content) such as lymphoma and medulloblastoma.
    - Vascular flow void
    - Mucin: desiccated mucin (seen in desiccated sinus secretions) is T2SD. Conversely, mucinous lesions in the pelvis are hydrated and T2PB.

Fluid attenuation inversion recovery (FLAIR)

- Workhorse of neuroradiology. FLAIR is a T2WI w suppression of water signal based on water's T1 characteristics.
- Normal FLAIR image appears similar to T1WI (CSF is dark on both) though GWM is diff
    - T1WI: normal WM brighter than GM 2/2 fatty myelinated WM has shorter T1 time.
    - FLAIR: WM darker than GM

Spin echo proton density (PD)

- PD images are not used in many neurorads MRI protocols but they do have the highest signal to noise ratio of any MRI sequence.
- PD sequences are useful for MS, especially for seeing demyelinating plaques in post fossa.

Diffuse weighted images and apparent diffusion coefficient (DWI and ADC)

- Diffusion MRI overview
    - DWI and ADC are interpreted together to evaluate the diffusion of tissue
    - DI has revolutionized evaluation of cerebral infarctions; 95% sens and spec w/n min of sx onset. In this setting, diffusion restricted tissue = ischemia.
- DWI based on the principle that brownian motion of water protons can be imaged.
    - Signal is lost with increasing brownian motion. Less signal loss with decreasing brownian motion.
    - Free water (CSF) experiences the most signal attenuation, while many pathologic processes (primarily ischemia) cause reduced diffusivity and less signal loss.
    - DWI Inherently T2WI (obtained via echoplanar technique)
        - Reduced diffusivity is hyperintense (less brownian motion -> less signal loss) and lesions are more conspicuous
- ADC shows pure diffusion information w/o T2WI
    - In contrast to DWI, reduced diffusivity is hypointense on ADC map.
    - Studies show that readers are less sensitive to detecting reduced diffusivity using ADC map alone. For this reason DWI is the primary sequence used to detect diffusion abnormalities.
- Important pitfall
    - T2 shine through: Bc DWI is T2WI, lesions that are inherently hyperintense on T2WI may also be hyperintense on DWI even w/o restricted diffusion.
    - Correlation w ADC map for a corresponding dark spot is essential before concluding that diffusion is restricted.
- Brain diffusion images: obtained in 3 orthogonal gradient planes to account for inherent anisotropy of large WM tracts (tendency of water to diffuse directionally along WM tracts)
- B-value: affects the sensitivity for detecting diffusion abnormalities.
    - Higher the B value = more contrast provided to detect reduced diffusivity
        - Downside to increased Bvalue is a decrease in signal to noise ratio, unless scan time proportionally increased for additional acquisitions.
    - ADC map is calculated from a set of at least 2 different B value images.
- DMRI ddx for reduced diffusion
    - Acute stroke

        - Bacterial abscess
        - Cellular tumors (lymphoma and medulloblastoma)
        - Epidermoid cyst
        - Herpes encephalitis
        - CJD

Gradient recall echo (GRE)

- GRE captures the T2* signal. Bc the 180 degree rephasing pulse is omitted, GRE images are susceptible to signal loss from magnetic field inhomogeneities.
- Hemosiderin and Ca produce inhomogeneities in the magnetic field, which creates blooming artifacts on GRE and makes even small lesions conspicuous.
- Ddx of multiple dark spots on GRE:
    - Hypertensive microbleeds: dark spots primarily in the basal ganglia, thalami, cerebellum and pons
    - Cerebral amyloid angiopathy: dark spots are in subcortical WM, MC the parietal and occipital lobes.
    - Familial cerebral cavernous malformations
    - Axonal shear injury
    - Multiple hemorrhagic mets

Perfusion

- Advanced technique where brain is imaged repeatedly as a bolus of gadolinium contrast is injected. The principle of QMR is based on theory that gadolinium causes a magnetic field disturbance, which counterintuitively transiently decreases the image intensity.
- Perfusion images are echoplanar T2 images, which can be acquired very quickly.
- QMR may be used for evaluation of stroke and tumors.

Magnetic resonance spectroscopy

- MRS describes the chemical composition of a brain region.
- In some circumstances, spectroscopy may help distinguish recurrent tumor vs radiation necrosis and glioblastoma vs mets.
    - GBM is infiltrative and features a gradual transition from abnormal to normal spectroscopy. In contrast, the Mets would be expected to have a more abrupt transition.

Patterns of Brain Enhancement

- BBB is formed by astrocyte foot processes of brain capillary endothelial cells and prevents direct communication b/n systemic capillaries and the protected ECF of brain.
- Micro/macro disruption of BBB produces parenchymal enhancement after contrast admin. Can be 2/2: Infection, inflammation, neoplasm, trauma, vascular etiologies
- Several CNS regions do NOT have a BBB and normally enhance

    - Choroid plexus
    - Pituitary and pineal glands
    - Tuber cinereum (controls circadian rhythm located in inf hypothalamus)
    - Area postrema (controls vomiting, located inf aspect of 4th ventricle)
- Dura lacks BBB but does not enhance.

- Vascular enhancement is 2/2 localized increase in blood flow which may be 2/2 vasodilation, hyperemia, neovascularity or AV shunting.
    - CT arterial phase shows intravascular enhancement
    - CT and MRI parenchymal phase (min after inj)

- Intracranial enhancement may be intra or extraaxial
    - Extraaxial may enhance in pathologic conditions including dura (pachymeninges) and arachnoid (leptomeninges)

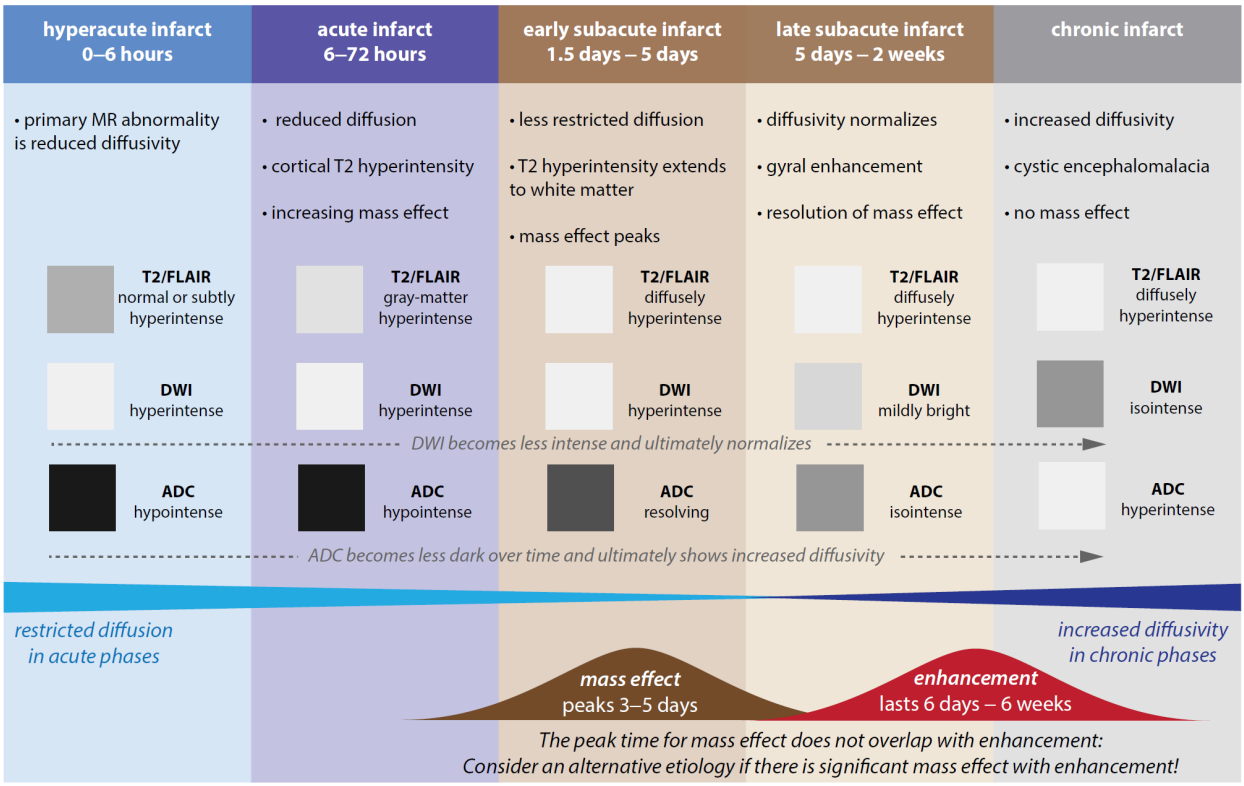
- Periventricular enhancement (intraaxial)
    - Enhancement of the subependymal surface can be either neoplastic, infectious, or demyelinating in etiology. Ddx for periventricular enhancement:
        - Primary CNS lymphoma
            - Can present w periventricular enhancement, solitary brain mass, multiple brain masses
            - Hyperattenuating on CT and demonstrates low ADC and low signal intensity on T2WI 2/2 hypercellularity
            - Rarely involves the meninges except for the setting of systemic lymphoma with spread to the brain.
            - CNS lymphoma is centrally necrotic in immunocompromised patients but usually homogenous in immunocompetent.
        - Infectious ependymitis: MC CMV. features thin linear enhancement along margins of ventricles
        - Primary glial tumor
        - Multiple sclerosis: may affect subependymal surfaces. Though the majority of demyelinating lesions do NOT enhance, an active plaque may.

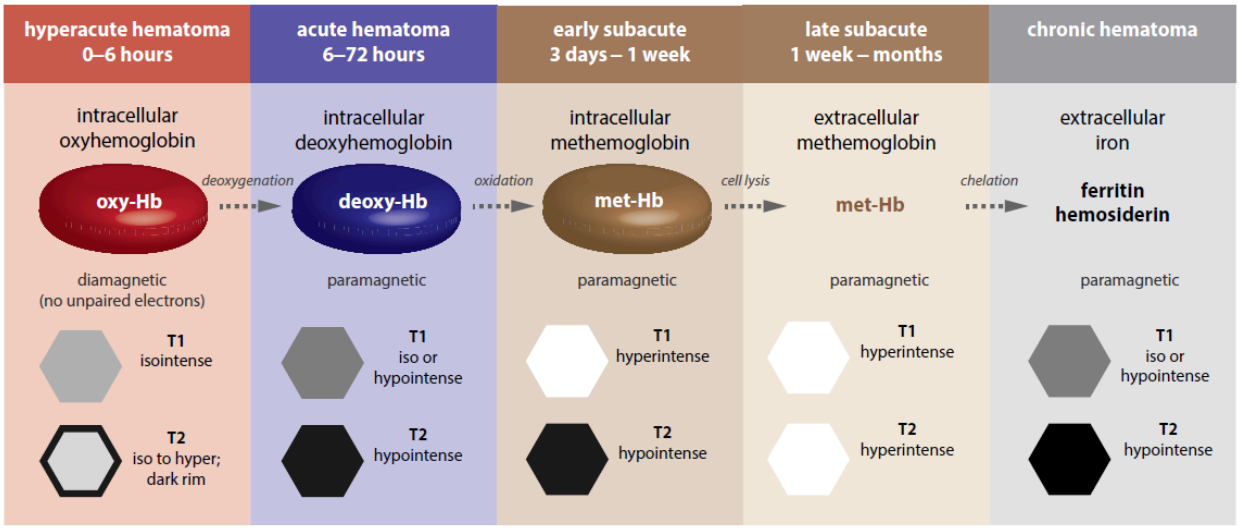
- Gyriform enhancement (intra-axial)
    - Superficial enhancement of cortical surface of brain can be 2/2 cerebral infection, inflammation or ischemia.
    - Ddx for gyriform enhancement:
        - Herpes encephalitis: reactivation of latents HSV1 inf w/n trigeminal ganglion. The medial temporal lobes and cingulate gyrus are usually affected first and enhance due to inflammation, petechial hemorrhage and BBB breakdown. Involved areas demonstrate reduced diffusivity.
        - Meningitis: May cause gyral enhancement in addition to more typical leptomeningeal enhancement.

        - Subacute infarct: gyriform enhancement lasting approx 6 days - 6 weeks after initial ischemic event
            - In contrast, acute infarct may demonstrate vascular enhancement 2/2 reactive collateral vasodilation and resultant hyperemia.
        - PRES: vasogenic WM edema triggered by altered autoregulation that may demonstrate gyral enhancement. PRES may rarely exhibit restricted diffusion.

- Nodular subcortical enhancement (intra-axial)
    - MC 2/2 mets dz.
    - Hematogenously disseminated metastatic dz commonly found at subcortical GWJ. tumor emboli become stuck at the junction b/n the simple vasculature of WM and the highly branching vasculature of the GM.
    - Edema is almost always present w mets of the GWJ. Although slightly more distal cortical mets may not show any edema and may be detectable only on post contrast images.
    - In contrast with arterial mets, venous dissemination of mets (pelvic malignancy spread via Batson prevertebral venous plexus) leads to post fossa dz by transit through the retroclival venous plexus.

MR signal	**High signal (bright)**	**Low signal (dark)**
T1	Fat, melanin, blood (methemoglobin) Proteinaceous fluid Paramagnetic substances (manganese, copper) Chelated gadolinium contrast	Iron Water Air Bone, collagen Most tumors
T2	Water Edema Fat Blood Most tumors	Air Bone Chronic blood/hemosiderin Acute blood (intracellular deoxyhemoglobin) Early subacute blood (methemoglobin)


hyperacute infarct
0–6 hours
acute infarct
6–72 hours
early subacute infarct
1.5 days – 5 days
late subacute infarct
5 days – 2 weeks
chronic infarct
• primary MR abnormality is reduced diffusivity
• reduced diffusion
• cortical T2 hyperintensity
• increasing mass effect
• less restricted diffusion
• T2 hyperintensity extends to white matter
• mass effect peaks
• diffusivity normalizes
• gyral enhancement
• resolution of mass effect
• increased diffusivity
• cystic encephalomalacia
• no mass effect
T2/FLAIR normal or subtly hyperintense
T2/FLAIR gray-matter hyperintense
T2/FLAIR diffusely hyperintense
T2/FLAIR diffusely hyperintense
T2/FLAIR diffusely hyperintense
DWI hyperintense
DWI hyperintense
DWI hyperintense
DWI mildly bright
DWI isointense
DWI becomes less intense and ultimately normalizes
ADC hypointense
ADC hypointense
ADC resolving
ADC isointense
ADC hyperintense
ADC becomes less dark over time and ultimately shows increased diffusivity
restricted diffusion in acute phases
increased diffusivity in chronic phases
mass effect peaks 3–5 days
enhancement lasts 6 days – 6 weeks
The peak time for mass effect does not overlap with enhancement:
Consider an alternative etiology if there is significant mass effect with enhancement!


hyperacute hematoma
0–6 hours
acute hematoma
6–72 hours
early subacute
3 days – 1 week
late subacute
1 week – months
chronic hematoma
intracellular oxyhemoglobin
intracellular deoxyhemoglobin
intracellular methemoglobin
extracellular methemoglobin
extracellular iron
oxy-Hb
deoxygenation
deoxy-Hb
oxidation
met-Hb
cell lysis
met-Hb
chelation
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diamagnetic (no unpaired electrons)
paramagnetic
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paramagnetic
paramagnetic
T1 isointense
T1 iso or hypointense
T1 hyperintense
T1 hyperintense
T1 iso or hypointense
T2 iso to hyper; dark rim
T2 hypointense
T2 hypointense
T2 hyperintense
T2 hypointense

- MR imaging of hemorrhage is complex. The characteristics of blood products change on T1- and T2-weighted sequences as the iron in hemoglobin evolves through physiologic stages:

  Intracellular oxyhemoglobin → *deoxygenation* →
  Intracellular deoxyhemoglobin → *oxidation* →
  Intracellular methemoglobin → *cell lysis* →
  Extracellular methemoglobin → *chelation* →
  Hemosiderin and ferritin

- Each stage of this evolution adheres to a reasonably constant time course in the intra-axial space and allows the radiologist to "date" the hemorrhage based on the unique signal characteristics on T1- and T2-weighted images for each stage.
- In general, all stages of hemorrhage are isointense or slightly dark on T1-weighted images, except for the methemoglobin stages, which are bright.
- Methemoglobin is bright on both T1- and T2-weighted images, except for intracellular methemoglobin, which is dark on T2-weighted images.
- In general, non hyperacute hemorrhage is dark on T2-weighted images, with the exception of extracellular methemoglobin, which is hyperintense on T2-weighted images. A hyperacute hematoma, containing primarily oxyhemoglobin, is slightly hyperintense on T2-weighted images but features a characteristic dark rim representing deoxygenation at the periphery of the clot.
- The inherent slight hyperintensity of oxygenated blood on T2-weighted images becomes apparent in slow flow states, as seen in venous hypertension and moyamoya disease. Slowly flowing blood is not susceptible to the flow-void artifact and the resultant apparently "enhancing" vasculature really represents unmasking of the normal blood signal.
- The expected evolution of blood products is highly dependent on macrophage elimination of blood breakdown products. These rules of thumb are not applicable to extra-axial blood and timing is generally not given for extra-axial blood, such as a subdural hematoma.

RESOURCES

Resources

- Duke Review of MRI Principles is good for practical MRI physics, sequences, pitfalls
- Core Radiology is a solid book
- Eventually get through Neuro Requisites
- CaseStacks is a nice way to scroll through and learn from real high-yield cases
- RadPrimer for questions
- Radiopaedia for rapid lookup
- Radiology Assistant has some excellent articles including for temporal bone (https://radiologyassistant.nl/)
- See both Learning Neuroradiology (https://sites.google.com/a/wisc.edu/neuroradiology/home), which includes practical anatomy and approaches, and Lean Neuroradiology (https://learnneuroradiology.com/), which includes lots of video lectures.

Critical Findings

GUIDELINES ON IMAGING FINDINGS THAT MAY SUGGEST A NEED FOR IMMEDIATE OR URGENT INTERVENTION (CRITICAL FINDINGS)

The following list of imaging findings may constitute Critical Findings that warrant prompt communication with a member of the patient's care team—particularly if new, acute, or worsening. Alternatively, significant imaging findings that are already well known to the care team and not significantly changed may not constitute Critical Findings. Likewise, imaging findings that are expected and/or considered "within normal limits" in the appropriate clinical context (such as recent surgical/procedural intervention) would not be considered Critical Findings.

I. **Any imaging finding considered a Critical Finding in the judgment of the interpreting radiologist.**

II. **Neurological:**
   a. CNS hemorrhage/hematoma with significant mass effect
   b. CNS hemorrhage suspicious for leaking aneurysm
   c. ~~Large acute stroke~~ Any not previously known acute stroke
   d. New/worsening herniation syndrome or hydrocephalus
   e. New/unexpected intracranial infection/empyema
   f. Significantly depressed or displaced skull fracture

III. **Neck and Spine**
   a. Acute airway compromise (such as epiglottitis)
   b. Acute carotid or vertebral artery dissection
   c. Spine fracture with concern for instability
   d. Acute spinal cord compression

IV. **Vascular**
   a. Acute ruptured or dissecting aneurysm
   b. Acute arterial injury, dissection, or occlusion
   c. Significant active hemorrhage and/or large acute hematoma
   d. Acute deep vein thrombosis

V. **Thorax**
   a. New/worsening large, bilateral, or tension pneumothorax
   b. Acute pulmonary embolus
   c. Acute esophageal perforation

VI. **Abdomen/Pelvis**
   a. Unexpected free intraperitoneal air
   b. Unexpected portal venous gas
   c. Significant acute solid organ or bowel injury
   d. High grade bowel obstruction or volvulus with suspicion for ischemia

VII. **Obstetric/Gynecologic/Genitourinary**
   a. Ectopic pregnancy
   b. Testicular or ovarian torsion

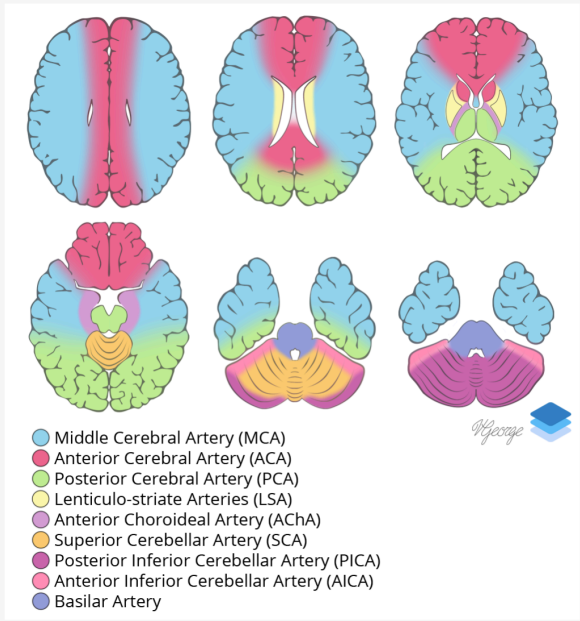
VIII. **Other**
   a. Significant line or tube misplacement
   b. Unexpected retained surgical or procedural foreign body
   c. New/worsening necrotizing fasciitis

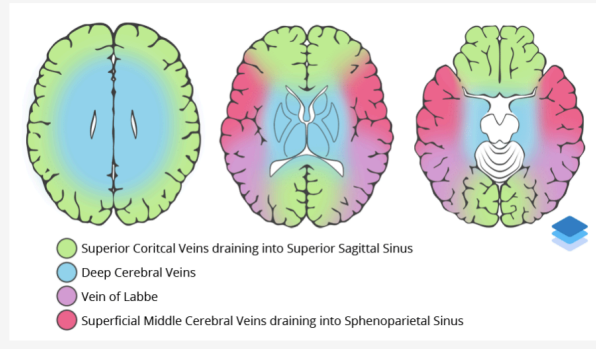
REPORTING

FOLLOW UP POST BRAIN TUMOR RESECTION
The surgeon wants to know if there is any residual tumor left/hyperenhancement.

ISCHEMIC INFARCT

- When you see cerebral infarcts, try to offer a preferred mechanism to help the clinical team determine the best steps.
    - Thrombotic: w/n single arterial territory
    - Embolic: w/n multiple arterial territories
    - Watershed: concentrated on the borders b/n arterial territories
    - Venous w/n venous territory
- Approximate representations of cerebral arterial and venous vascular territories.





DIAGNOSIS	FINDINGS	IMPRESSION
Thrombotic stroke	Large area of restricted diffusion in R MCA territory Associated T2/FLAIR hyperintensity and swelling w R cerebral sulcal effacement and 3 mm R to L midline shift Abnormal flow related signal in the communicating segment of the R ICA and in M1 segment of R MCA Few small foci of susceptibility artifact in the R frontal lobe Confluent subcortical and periventricular white matter T2/FLAIR hyperintensity Generalized cerebral volume loss.	1) Large acute/subacute R MCA territory infarct. Associated cytotoxic edema w resultant R cerebral sulcal effacement and 3 mm R to L midline shift. No evidence of herniation or hydrocephalus. 2) Few small foci of susceptibility artifact in R frontal lobe may represent petechial hemorrhage though there is no evidence of organized hemorrhagic conversion. 3) Abnormal flow-related signal in the communicating segment of R ICA and M1 segment of R mCA concerning for diminished/slow flow or occlusion. Recommend correlation w CTA or catheter angiography 4) confluent subcortical and periventricular WM T2/FLAIR hyperintensity, which though nonspecific

	Remote lacunar infarcts in pons.	likely relates to chronic small vessel disease. Remote lacunar infarcst in the pons. Generalized cerebral volume loss.

Sensitivity for early CVA detection: MRI >> CT
Abnormal signal first on DWI w/n minutes following occlusion.

- ADC signal normalizes more quickly (~10 days) than diffusion bc T2 effects which may transiently or permanently keep diffusion signal mildly elevated.
- T2/FLAIR signal abnormality lags somewhat, typically starting to become apparent 6-8 hrs after occlusion.

3 types of CVA enhancement:

- Arterial (hrs to days after stroke): decreased arterial BF w/n area of acute ischemia results in decreased flow-related signal loss. This increased intrinsic signal combined w the signal for administered contrast material makes these vessels appear more hyperintense than unaffected arteries.
- Meningeal (1-7 days after stroke): resulting from reactive hyperemia in the meninges overlying the infarct. This is the least commonly observed type of enhancement.
- Parenchymal (1 wk - 3 month): resulting from breakdown of BBB. Parenchymal enhancement may occur earlier and more intensely in incomplete infarcts.
    - If you are trying to determine if an enhancing lesion is an infarct vs neoplasm, repeating imaging in a few months can help differentiate the two.

DIAGNOSIS	FINDINGS	IMPRESSION
Watershed strokes	Multifocal areas of restricted diffusion along the R>L ACA/MCA and MCA/PCA watershed zones Associated T2/FLAIR hyperintensity w mild local mass effect but no midline shift or herniation No acute hemorrhage. No hydrocephalus. Patchy subcortical and periventricular T2/FLAIR WM hyperintensities B/L mastoid effusions	Acute multifocal infarcts along the R > L ACA/MCA and MCA/PCA watershed zones. Mild local mass effect w/o midiline shift or herniation. No acute hemorrhage or hydrocephalus. Background of chronic small vessel disease B/l mastoid effusions.

-

PRES

Posterior reversible encephalopathy syndrome is thought to result from hyperperfusion in the setting of HTN due to breakdown of cerebral BP autoregulation, typically affecting the posterior circulation exclusively or at least more severely than the anterior circulation.

High BP is thought to damage the BBB though in a minority of cases, PRES can occur in normotensive patients w systemic illnesses such as sepsis that are thought to lead to endothelial dysfunction.

Many medications have been associated w increased risk of PRES: multiple immunosuppressants used for solid organ transplants and for treatment of autoimmune diseases.

Most commonly involved areas are the parieto occipital region and watershed zone WM, with less common involvement of the frontal lobes, BG, thalamus, brainstem, and cerebrellum.

Hemorrhage is reported in 15% of cases of PRES and is commonly cortical and/or subcortical in location.

Restricted diffusion can also be seen in more severe cases, where edema can progress to infarct. However, the presence of restricted diffusion does not necessarily indicate permanent damage as signal and sx normalization has been reported w timely management.

Whenever you see b/l relatively symmetric edema make sure to exclude dural venous sinus thrombosis.

Sample report:

- Extensive T2/FLAIR signal hyperintensity involving the b/l parietal and occipital lobes involving the GM and WM w associated gyral swelling nad cortical restricted diffusion. Additiona lareas of cortical and subcotical WM T2/FLAIR hyperintensity involving the b/l superior parietal lobules and posterior frontal lobes. Multiple areas of susceptibility artifact centered in the b/l parietaooccipital cortices. Tiny enhancing extraaxial structure along the high L frontal convexity. Remote lacunar infarct in the L caudate head.
- Findings are most suggestive of posterior reversible encephalopathy syndrome involving the b/l parietococciptial regions and to a lesser extent the b/l posterior frontal lobes. Associated areas of developing cortical infarction w multiple small parietooccoipital cortical hemorrhages. No evidence of herniation or hydrocephalus. Tiny enhancing extraaxial structure along the high L frontal convexity which may represent a tiny meningioma. Remote lacunar infarct in L caudate head.

HYPOXIC ISCHEMIC ENCEPHALOPATHY (ADULT)

Underlying cellular pathophysiology:

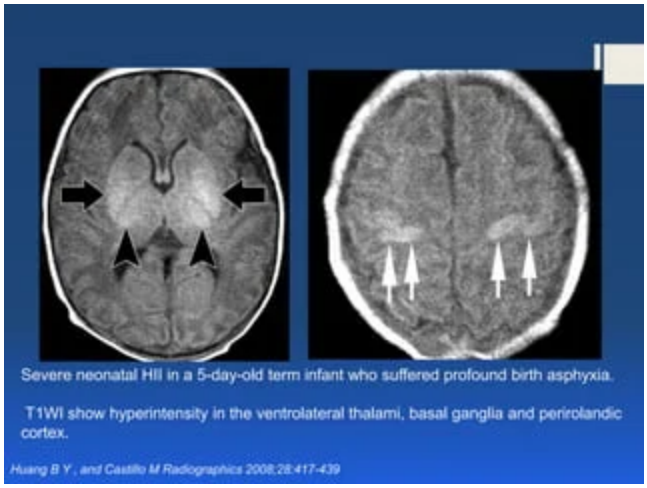
- Injury may relate from impaired blood flow (ischemia) and/or decreased oxygen saturation of blood (hypoxemia) which is more common in children in the setting of respiratory arrest.
- In hypoxic environment, cells switch to anaerobic metabolism causing a more rapid depletion of ATP, lactate accumulation and cell membrane depolarization resulting in release of excitatory neurotransmitters (glutamate) from presynaptic neurons)
- Glutamate binds to NMDA receptors on postsynaptic causing an influx of Ca, which triggers cell membrane damage and triggers apoptotic pathways.
- Cells may die quickly from energy depletion and/or membrane destruction by free radicals OR may have delayed death via apoptosis.

Take aways:

- Cells most prone to HIE are those that are highly metabolically active and have high concentrations of presynaptic excitatory neurotransmitters like glutamate.
- Due to delayed cell death by apoptosis, initial clinical and imaging evaluation may underestimate extent of injury.

Helpful hints

- Mild-moderate insults must be prolonged to result in permanent damage or any imaging findings whereas relatively short severe insults can result in permanent injury. This is particularly true in infants and children.
- ADC maps are more sensitive for injury than diffusion sequences in infants so make sure to look at ADC maps in all cases even when diffusion appears normal.
- Diffusion signal abnormalities begin to be evident w/n hrs of injury and peak at 3-5 days then pseudonormalize at 1 week.
    - If f/u imaging is needed to confirm suspected subtle findings, recommend imaging 3-5 days after initial injury.
- T1 hyperintensity and T2 hypointensity develop more slowly starting to become apparent a few days after the injury.
    - T1 and T2 are particularly helpful when imaging a week after the initial injury when diffusion signal may have deceptively normalized.
- In term and postterm infants, the posterior limb of the internal capsule should be myelinated (myelination begins in the PLIC at about 36 weeks), so compared to the adjacent thalamus and posterior lentiform nucleus (putamen + GP), it should be bright on T1 and dark on T2. these relationships are reversed in HIE involving the BG and thalami.

- 

Hypoxic Ischemic Encephalopathy Imaging Findings by Patient Age and Severity

Age	Severity	Distribution
Preterm Neonate	Mild-moderate	Germinal matrix (< 34 wks) Periventricular white matter (especially < 32 wks)
	Severe	Brainstem and deep gray matter Corpus striatum and perirolandic cortex typically spared in preterm infants < 35 weeks of age
Term Neonate	Mild-moderate	Watershed zones Spares deep gray matter
	Severe	Deep gray matter ± perirolandic cortex
Young Child	Mild-moderate	Watershed zones Spares deep gray matter and periventricular white matter
	Severe	Corpus striatum (especially posterolateral lentiform nuclei) ± cerebral cortex Relative sparing of thalami and perirolandic cortex
Old Child or Adult	Mild-moderate	Watershed zones
	Severe	Deep gray nuclei and cerebral cortex

Sample report:

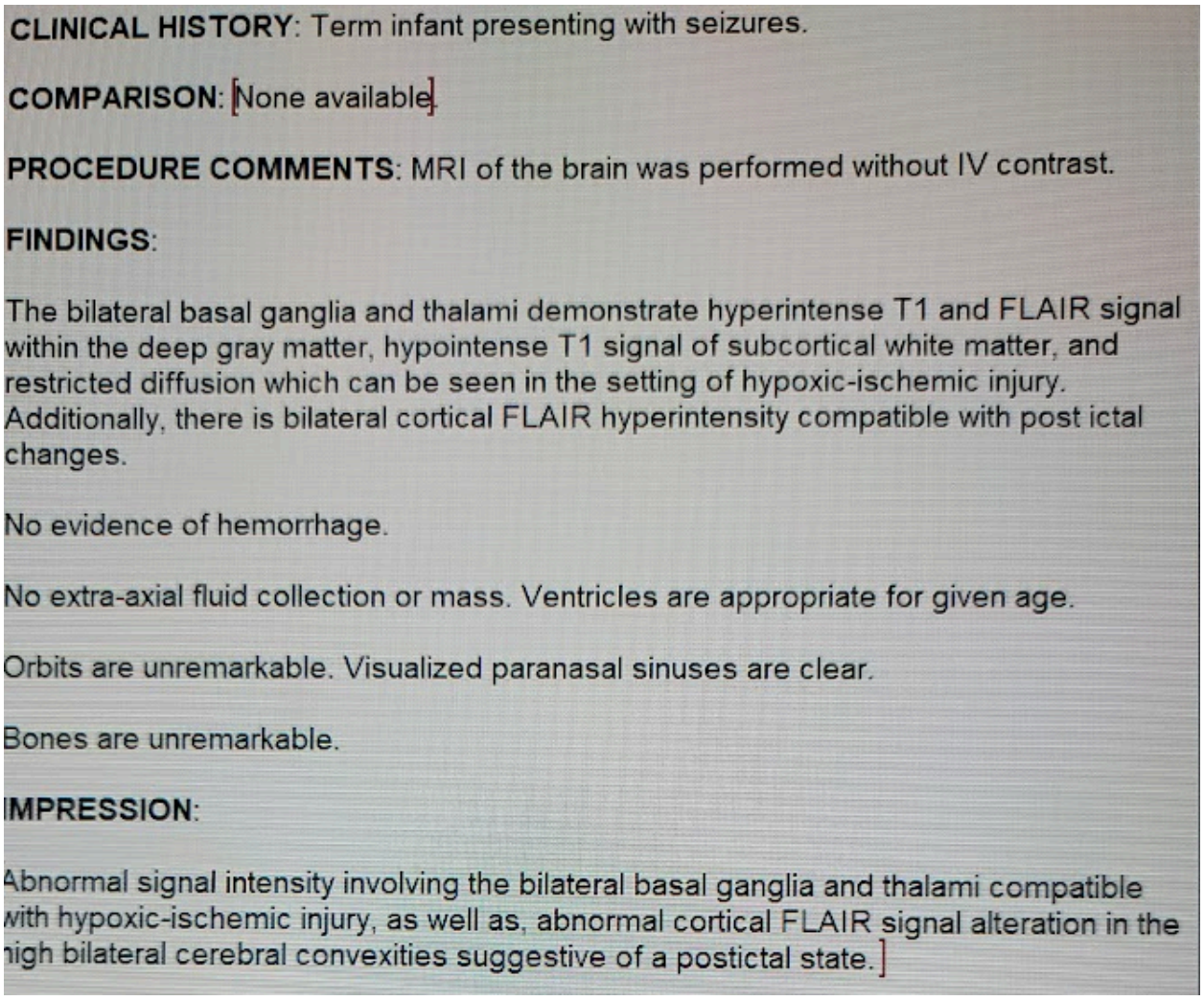
- Case 1: Widespread restricted diffusion involving the b/l cerebral cortices and b/l corpus striatum w corresponding T2/FLAIR hyperintensity. Mild generalized cerebral sulcal effacement and gyral swelling. Mild effacement of both lateral ventricles without midline

shift or effacement of the basal cisterns. T2/FLAIR hyperintense lesion in the L frontal periventricular WM. Small b/l mastoid effusions.

- Case 2: Abnormal T1 signal hyperintensity and corresponding T2 signal hypointensity in the b/l ventrolateral thalami and posterior lentiform nuclei. Areas of restricted diffusion in the posterior limb of the internal capsule and posterior lentiform nucleus bilaterally. Increased T1 signal in the bilateral perirolandic cortex. No acute hemorrhage, mass effect, or hydrocephalus.

Impression:

- Case 1: Findings consistent w severe hypoxic ischemic encephalopathy involving the b/l cerebral cortices and BG. mild generalized cerebral edema w partial effacement of the lateral ventricles but no midline shift, herniation or hydrocephalus. T2/FLAIR hyperintense lesion in the L frontal periventricular WM, which may represent a remote infarct or demyelinating lesion.
- Case 2: findings consistent w severe hypoxic ischemic encephalopathy involving the bilateral BG, ventrolateral thalami, and perirolandic cotices. No acute hemorrhage, mass effect, or hydrocephalus.



CLINICAL HISTORY: Term infant presenting with seizures.

COMPARISON: None available.

PROCEDURE COMMENTS: MRI of the brain was performed without IV contrast.

FINDINGS:

The bilateral basal ganglia and thalami demonstrate hyperintense T1 and FLAIR signal within the deep gray matter, hypointense T1 signal of subcortical white matter, and restricted diffusion which can be seen in the setting of hypoxic-ischemic injury. Additionally, there is bilateral cortical FLAIR hyperintensity compatible with post ictal changes.

No evidence of hemorrhage.

No extra-axial fluid collection or mass. Ventricles are appropriate for given age.

Orbits are unremarkable. Visualized paranasal sinuses are clear.

Bones are unremarkable.

IMPRESSION:

Abnormal signal intensity involving the bilateral basal ganglia and thalami compatible with hypoxic-ischemic injury, as well as, abnormal cortical FLAIR signal alteration in the high bilateral cerebral convexities suggestive of a postictal state.

OSMOTIC DEMYELINATION

T2/FLAIR hyperintensity associated w osmotic demyelination syndrome often lages 1-2 weeks after sx onset though there can be restricted diffusion w/n the first 24 hrs.

Typically there is sparing of the ventrolateral pons and corticospinal tracts.

About half of cases of osmotic demylination involve the pons while the other half involve extrapontine sites (middle cerebellar peduncles, BG, external capsule, thalamus and/or cerebral WM) or both.

Pontine glioma is also a differential consideration for this imaging appearance though ti typically occurs in much younger patients and will not resolve on F/U

Sample report:

- T2/FLAIR hyperintensity and restricted diffusion in the central pons w peripheral sparing and no associated mass effect or abnormal enhancement. These findings are primarily concerning for osmotic demyelination. Recommend correlation w serum sodium measurements. No evidence of acute hemorrhage or hydrocephalus.

LOW GRADE GLIOMA

Gliomas (tumors arising from glial precursors including astrocyte and oligodendrocyte cell lineages) are classified by the WHO as grade I-IV, with grade 1 being a focal tumor w discrete margins (pilocytic astrocytoma) and grades II-IV being infiltrative tumors w/o clear margins.

WHO grade II gliomas rarely enhance, grade III gliomas sometimes enhance and grade IV gliomas almost always enhance as a general rule.

W increased understanding of the behavior of these tumors are a molecular level, these tumors are now classified as IDH wild type)worse) or IDH mutant, and IDH mutant tumors being further subdivided by 1P19q codeletion present (oligodendrogliomas, best prog) or absent.

Knowing and having a basic understanding of the molecular markers when reading F/U can help you anticipate the expected behavior of the tumor over time.

HSV encephalitis can result in abnormal signal involving limbic structures. It is better to over treat for HSV to avoid missing it since delaying diagnosis of a low grade glioma for a few days is unlikely to have any affect on patient outcome whereas delaying tx for HSV can be deadly.

It is very important to look for gyral expansion in areas of T2/FLAIR hyperintensity as this helps differentiate tumors from encephalomalacia.

Sample report

- Infiltrative, mildly expansile area of T2/FLAIR hyperintensity involving the L insula, anterior L temporal pole and inferior L orbitofrontal cortex extending medially w involvement of the posterior aspect of the L gyrus rectus. No associated restricted diffusion, susceptibility artifact, or enhancement. Incidental dilated perivascular spaces in the midbrain.
- Infiltrative mildly expansile area of T2/FLAIR hyperintensity involving the L insula, anterior L temporal pole and inferior L orbitofrontal cortex w/o associated enhancement. This appearance likely represents a low grade glioma, though encephalitis could have a similar appearance in the correct clinical setting.

HIGH GRADE GLIOMA

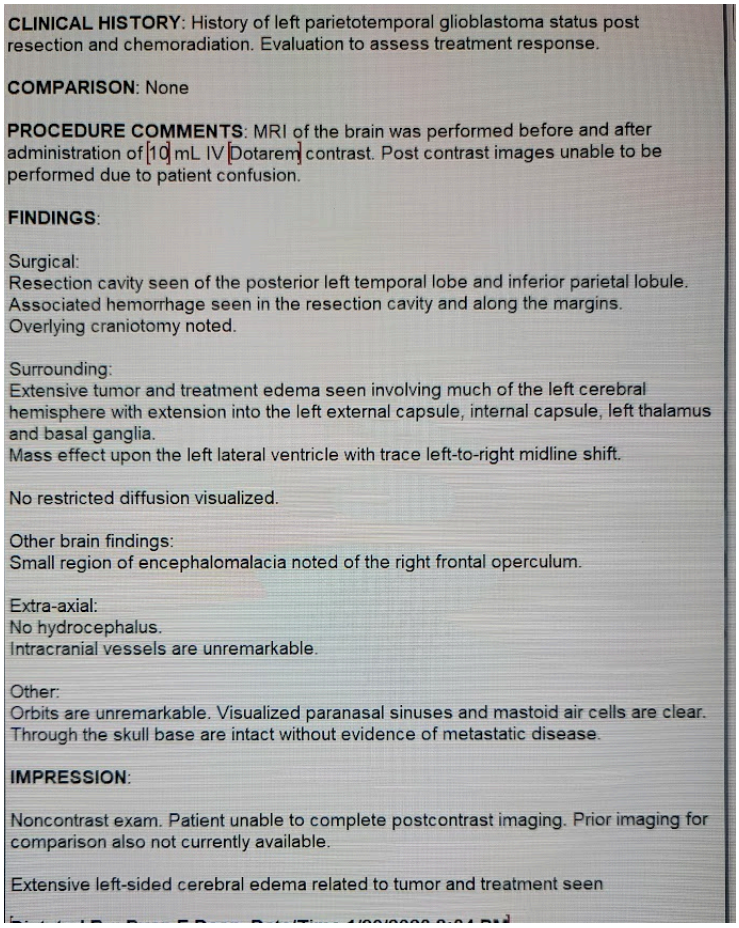
Info same as above.
T2/FLAIR WM hyperintensity around these tumors often represents a combo of infiltrative tumor and edema, so calling it edema is often inaccurate.

Large enhancing, centrally necrotic mass involving the posterior left frontal lobe, superior left temporal lobe, and anterior left parietal lobe with surrounding white matter T2/FLAIR hyperintensity and multiple nearby satellite enhancing lesions, which is concerning for a high grade glioma. The medial margin of the mass abut the ependymal surface of the atrium of the left lateral ventricle, raising the risk for ependymal spread. Recommend surgical evaluation.

Associated mass effect with local sulcal effacement, partial effacement of the left lateral ventricle with entrapment of the left temporal horn, and left to right midline shift measuring 7 mm.

No evidence of acute infarct, hemorrhage, or herniation.

Post resection



CLINICAL HISTORY: History of left parietotemporal glioblastoma status post resection and chemoradiation. Evaluation to assess treatment response.

COMPARISON: None

PROCEDURE COMMENTS: MRI of the brain was performed before and after administration of [10] mL IV [Dotarem] contrast. Post contrast images unable to be performed due to patient confusion.

FINDINGS:

Surgical:
Resection cavity seen of the posterior left temporal lobe and inferior parietal lobule.
Associated hemorrhage seen in the resection cavity and along the margins.
Overlying craniotomy noted.

Surrounding:
Extensive tumor and treatment edema seen involving much of the left cerebral hemisphere with extension into the left external capsule, internal capsule, left thalamus and basal ganglia.
Mass effect upon the left lateral ventricle with trace left-to-right midline shift.

No restricted diffusion visualized.

Other brain findings:
Small region of encephalomalacia noted of the right frontal operculum.

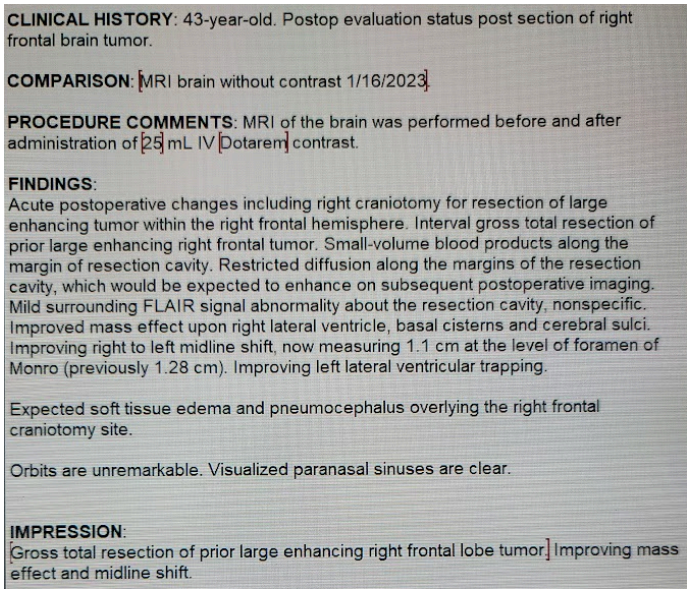
Extra-axial:
No hydrocephalus.
Intracranial vessels are unremarkable.

Other:
Orbits are unremarkable. Visualized paranasal sinuses and mastoid air cells are clear.
Through the skull base are intact without evidence of metastatic disease.

IMPRESSION:

Noncontrast exam. Patient unable to complete postcontrast imaging. Prior imaging for comparison also not currently available.

Extensive left-sided cerebral edema related to tumor and treatment seen



CLINICAL HISTORY: 43-year-old. Postop evaluation status post section of right frontal brain tumor.

COMPARISON: [MRI brain without contrast 1/16/2023]

PROCEDURE COMMENTS: MRI of the brain was performed before and after administration of [25] mL IV [Dotarem] contrast.

FINDINGS:
Acute postoperative changes including right craniotomy for resection of large enhancing tumor within the right frontal hemisphere. Interval gross total resection of prior large enhancing right frontal tumor. Small-volume blood products along the margin of resection cavity. Restricted diffusion along the margins of the resection cavity, which would be expected to enhance on subsequent postoperative imaging. Mild surrounding FLAIR signal abnormality about the resection cavity, nonspecific. Improved mass effect upon right lateral ventricle, basal cisterns and cerebral sulci. Improving right to left midline shift, now measuring 1.1 cm at the level of foramen of Monro (previously 1.28 cm). Improving left lateral ventricular trapping.

Expected soft tissue edema and pneumocephalus overlying the right frontal craniotomy site.

Orbits are unremarkable. Visualized paranasal sinuses are clear.

IMPRESSION:
[Gross total resection of prior large enhancing right frontal lobe tumor.] Improving mass effect and midline shift.

BRAIN METS

The presence of multiple lesions throughout multiple territories in the brain should make you think of hematogenous dissemination of thrombus, infection, or tumor (TIT).

Typically pyogenic abscesses often peripherally enhance like mets BUT should have central restricted diffusion unlike most mets.

- Some hypercellular tumors like small cell lung CA can weakly restrict diffusion

Metastatic dz to the brain is most often parenchymal, classically at the GWJ but can also be leptomeningeal or pachymeningeal.

The lack of multifocal disease does not exclude mets, as 50% of intracranial mets are solitary at time of initial imaging.

Mets that are more likely to hemorrhage include: melanoma, RCC, thyroid CA, choriocarcinoma.

Sample report:

- Numerous peripherally enhancing lesions involving the supratentorial and infratentorial brain with the following index lesions: 1.2 cm lesion in the posterior aspect of the left superior frontal gyrus, 1.3 cm cortically-based lesion in the left inferior frontal gyrus, 4 cm lesion in the right cerebellar hemisphere abutting the sigmoid sinus (which remains patent), and 2 cm lesion in the medial left cerebellar hemisphere/vermis
- The cerebellar lesions have surrounding vasogenic edema and mass effect with resultant partial effacement of the fourth ventricle and inferior displacement of the cerebellar tonsils into the foramen magnum with mild crowding of the upper cervical spinal cord in the foramen magnum. Mild enlargement of the lateral and third ventricles
- Mild vasogenic edema and local mass effect associated with several of the supratentorial lesions
- Several lesions have peripheral restricted diffusion, but none of the lesions have central restricted diffusion
- Several lesions demonstrate internal areas of susceptibility artifact
- No evidence of acute infarct

Numerous peripherally enhancing lesions involving the supratentorial and infratentorial brain which are most concerning for metastatic disease. Several lesions demonstrate internal areas of susceptibility artifact consistent with intralesional hemorrhage. No associated central restricted diffusion to suggest abscess.

Associated mass effect in the posterior fossa with partial effacement of the fourth ventricle and inferior displacement of the cerebellar tonsils into the foramen magnum with mild crowding of the upper cervical spinal cord in the foramen magnum. Mild enlargement of the lateral and third ventricles is concerning for mild/early hydrocephalus.

No evidence of acute infarct.

FINDINGS:

Evaluation is limited by motion artifact.

There is a moderately-sized cortical/subcortical heterogenous mass measuring 3 x 3 x 2 cm with adjacent acute hemorrhage located within the left posterior parietal lobe compatible with hemorrhagic metastasis (series 19 image 93). Mild enhancement along the posterior margin is visualized. There is moderate local vasogenic edema with minimal mass effect.

There is also a well-circumscribed metastatic lesion located within the cortical/subcortical right medial parietal lobe with small degree of vasogenic edema. Additional small hyper enhancing foci within left parietal cortex suspicious for metastasis (series 18 image 135).

No acute ischemic infarction or additional areas of abnormal enhancement. No extra-axial fluid collection. No midline shift, hydrocephalus or herniation.
Cyst
Orbits are unremarkable. Visualized paranasal sinuses are clear.

No evidence of osseous metastasis to calvarium/skull base or visualized cervical spine.

IMPRESSION:

Moderately sized well-circumscribed hemorrhagic metastatic lesion located within left posterior parietal lobe with minimal mass effect. Additional areas of metastasis located within the right posterior parietal and left lateral parietal lobe.

CLINICAL HISTORY: History of newly diagnosed lung mass. Rule out brain metastasis.

COMPARISON: PET/CT scan skull to thigh 1/13/2023.

PROCEDURE COMMENTS: MRI of the brain was performed before and after administration of 15 mL IV Dotarem contrast.

FINDINGS:

The examination is motion degraded, however pertinent anatomic information remains discernible.

The diffusion-weighted acquisition shows no definite evidence of recent ischemic injury.

An avidly enhancing 1.9 x 1.8 x 1.6 cm mass of right superior frontal gyrus. Extensive vasogenic edema present within the right cerebral hemisphere spanning from the anterior frontal lobe to right motor cortex and inferiorly to the corpus callosum. Associated mass effect with effacement of the lateral ventricles, right greater than left, with approximately 0.4 cm of right to left midline shift. No complicating hydrocephalus.

No acute intraparenchymal hemorrhage or infarction. No extra-axial fluid collection.

Orbits are unremarkable. Visualized paranasal sinuses and mastoid air cells are well aerated.

No definite aggressive or destructive lesion within the skull base or calvaria.

IMPRESSION:

A solitary 1.9 cm metastasis of the right frontal lobe with surrounding extensive edema and right to left midline shift without complicating hydrocephalus.

CARBON MONOXIDE POISONING

CM toxicity classically involves the globi pallidi with more severe cases involving other basal ganglia, thalami, brain stem and cerebellum

HYPERAMMONEMIC ENCEPHALOPATHY

HE can be seen in both pediatric and adult patients, most described w inborn errors of metabolism in pediatric population and w acute hepatic dysfunction in adult population.

Common finding is T2/FLAIR hyperintensity and restricted diffusion involving insular cortex and cingulate gyrus bilaterally with more variable involvement of BG, thalami, brain stem, cerebral cortex/subcortical WM. There may be associated microhemorrhages.

Sample report:

- Multifocal restricted diffusion and T2/FLAIR hyperintensity involving the b/l temporal lobes, hippocampi, insular cortices, cingulate gyrus, orbital frontal lobes and to a lesser extent the b/l thalami, which most likely relates to hyperammonemia given clinical hx, though encephalitis could have similar appearance.

WERNICKE ENCEPHALOPATHY

WE is a disorder resulting from thiamine deficiency classically seen in patients w alcoholism.

THiamine deficiency leads to an initially reversible cytotoxic edema which most often involves the periaqueductal GM in the midbrain, medial thalami and hypothalamus along the margins of the 3rd ventricle and mammillary bodies.

Less commonly, the cerebellum, caudate nuclei, splenium of CC and cerebral cortex can also be involved.

DDx: b/l thalamic signal abnormality

- Deep cerebral venous thrombosis
- Artery of Percheron infarct
- CJD
- Meningoencephalitis.

Sample report

- T2/FLAIR signal hyperintensity in the b/l periaqueductal gray matter, medial thalami, hypothalamus and possibly mammillary bodies. Though nonspecific, this distribution of signal abnormality raises concern for Wernicke encephalopathy. No evidence of acute ischemia.
- Scattered T2/FLAIR hyperintensities in the pervientricular and subcortical WM, which though nonspecific are ocmmonly attributable to chronic small vessel disease.
- Extra Axial lesion along the lateral aspect of the R temporal lobe w low signal on all sequences, likely represents bulky dystrophic calcification. Consider post contrast imaging to assess for associated enhancing mass.

DIFFUSE AXONAL INJURY

Diffuse axonal injury is the result of shearing forces w a rotational type injury. In other words, the cortex accelerates differently than the underlying WM, which results in axonal stretch (rarely complete tear) and edema and/or hemorrhage.

CT is relatively insensitive for detecting non hemorrhagic lesions. Visible lesions on CT are often the tip of the iceberg w many more lesions seen on susceptibility weighted MRI sequences.

Typical distribution: GWJ (especially frontoparietal regions), CC, deep gray matter, and brainstem.

Severity:

- Mild / grade 1: lesions at the GWJ
- Moderate / grade 2: lesions in the lobar WM and CC
- Severe / grade 3: lesions in the deep GM or brainstem.

MR findings:

- Hyperintense on FLAIR if nonhemorrhagic
- SWI is more sensitive than GRE and small microbleeds are seen as punctate areas of hypointensity.
- Lesions may restrict diffusion.

Cytotoxic lesions of the corpus callosum are secondary lesions resulting from cytokine toxicity that vary in severity and reversibility. There are many potential causes including:

- Drug toxicity: several chemotherapy agents, metronidazole
- CNS malignancy
- Meningitis / encephalitis
- Metabolic disorders such as Na imbalances, hyperammonemia, myelinolysis, Wernick encephalopathy, Wilson dz
- Trauma
- High altitude sickness, PRES

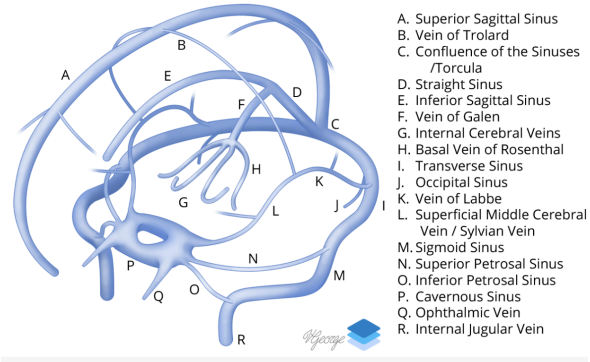
Sample report:

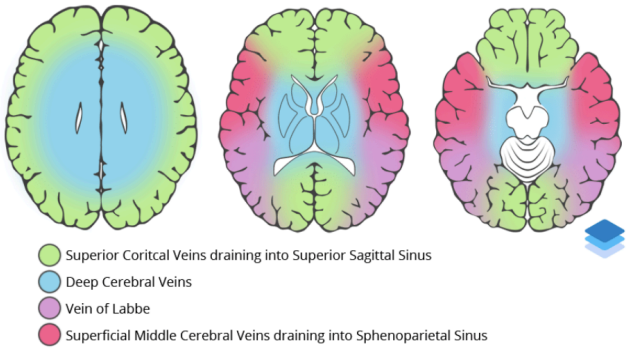
- Severe diffuse axonal injury evidenced by microhemorrhages in the b/l frontotemporal juxtacortical WM, deep gray nuclei, thalami, brainstem, and R cerebellar hemisphere.
- Trace SAH layering in the interpeduncular cistern as well as blood products layering in the occipital horns of both lateral ventricles. No hydrocephalus.
- Faint restricted diffusion in the splenium of the CC, more conspicuous on the ADC map, which could also relate to diffuse axonal injury; however, recommend correlation w evidence of seizure injury.

VENOUS SINUS THROMBOSIS

Approach:

- Think of sinus thrombosis when you see patterns of ischemia atypical for thrombotic infarcts including:
    - Superior sagittal sinus thrombosis -> symmetric parasagittal regions
    - Vein of Labbe -> temporal lobe
    - Deep venous thrombosis (straight sinus, internal cerebral veins) -> b/l thalami
- Venous thrombosis progresses to ischemia much slower than arterial thrombosis (several days to weeks). If thrombus is treat/removed, the area of related edema will often resolve w/o progressing to infarct.
- Severity: Deep venous infarcts > dural venous infarcts due to potential thalamic ischemia
- Just like arterial infarcts, mention any associated mass effect of hemorrhagic transformation.





Sample Findings

- Occlusive/near occlusive dural venous sinus thrombosis extending from the distal superior sagittal sinus into the L transverse sinus, L sigmoid sinus, L jugular bulb and prox L IJV. Thrombus also slightly extends into the proximal R transverse sinus and involves multiple L temporal cortical veins including the vein of Labbe.
- Small area of cortical and subcortical T2/FLAIR hyperintensity in the L middle temporal gyrus with faint cortical restricted diffusion.
    - Associated small area of congestive edema in the L middle temporal gyrus w faint cortical restricted diffusion possibly representing early ischemic changes.
- No acute hemorrhage. No significant mass effect or hydrocephalus. No evidence of proximal intracranial arterial occlusion.

AVM

Important cause of spontaneous intracranial hemorrhage in young to middle aged patients.

Spetzler - Martin grading scale 1-5 point scale for AVM correlates with operative risk (5 highest risk). Inoperable lesions can be assigned a grade of 6. It is calculated by summing the following criteria.

- Size of nidus
  - < 3 cm – 1 point
  - 3-6 cm – 2 points
  - > 6 cm – 3 points
- Eloquence of adjacent brain
  - Noneloquent – 0 points
  - Eloquent – 1 point
- Venous drainage
  - Superficial veins only – 0 points
  - Deep veins – 1 point

- For the purpose of their grading scale, the following are considered eloquent brain:
  - Sensorimotor, language, and visual cortices
  - Hypothalamus and thalamus
  - Internal capsule
  - Brainstem
  - Cerebellar peduncles
  - Cerebellar nuclei

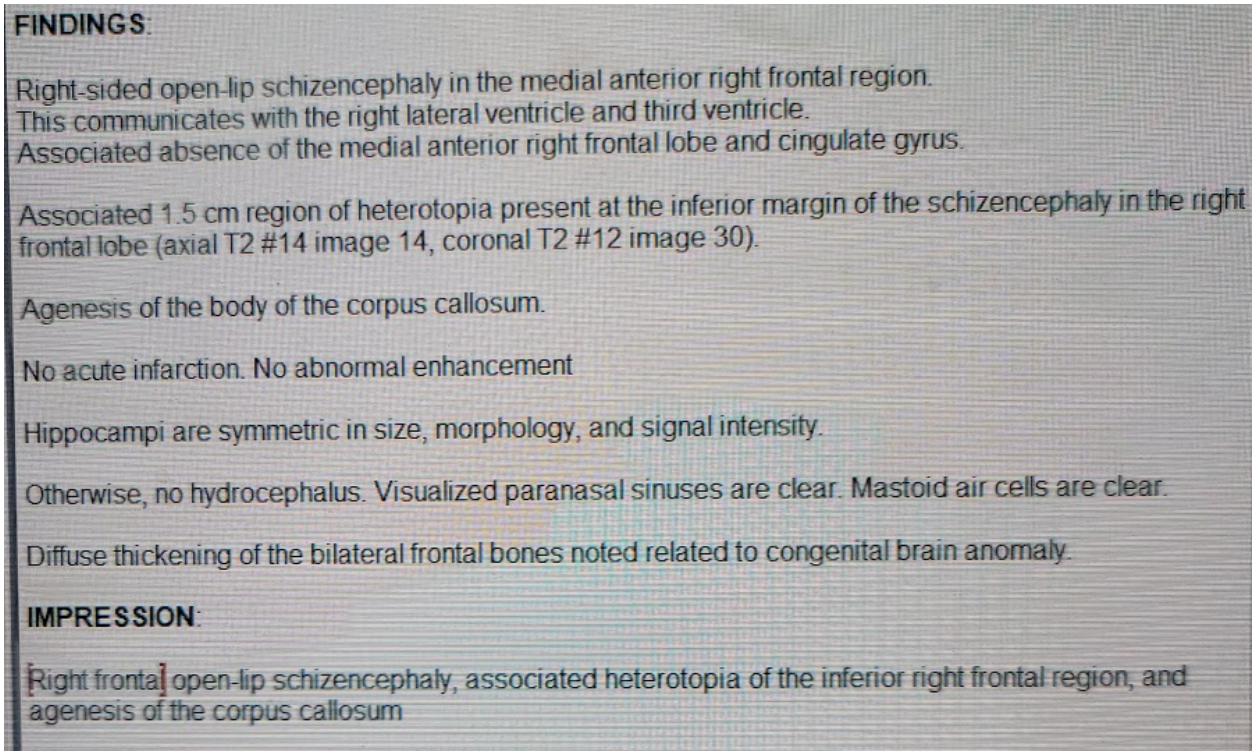
Findings:

- Tangle of vessels centered in the L precentral gyrus at the location of motor hand knob measuring 2 x 1.5 cm w feeding arterial branches from the superior and inferior division of the L MCA and superficial venous drainage via an engorged L cortical vein to the superior sag sinus. No evidence of intranidal aneurysm or deep venous drainage.
- No acute ischemia, hemorrhage, mass effect, or hydrocephalus.

Impression:

- AVM centered in the L precentral gyrus at the location of the motor hand knob measuring 2

Schizencephaly



FINDINGS:

Right-sided open-lip schizencephaly in the medial anterior right frontal region.
This communicates with the right lateral ventricle and third ventricle.
Associated absence of the medial anterior right frontal lobe and cingulate gyrus.

Associated 1.5 cm region of heterotopia present at the inferior margin of the schizencephaly in the right frontal lobe (axial T2 #14 image 14, coronal T2 #12 image 30).

Agenesis of the body of the corpus callosum.

No acute infarction. No abnormal enhancement

Hippocampi are symmetric in size, morphology, and signal intensity.

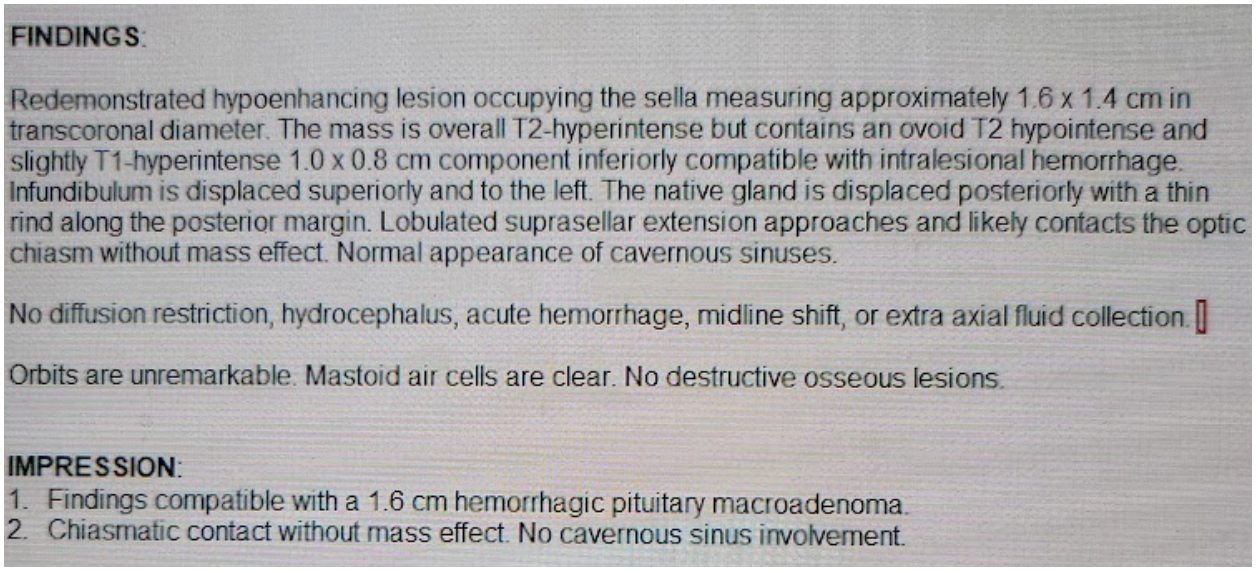
Otherwise, no hydrocephalus. Visualized paranasal sinuses are clear. Mastoid air cells are clear.

Diffuse thickening of the bilateral frontal bones noted related to congenital brain anomaly.

IMPRESSION:

Right frontal open-lip schizencephaly, associated heterotopia of the inferior right frontal region, and agenesis of the corpus callosum

MACROADENOMA



FINDINGS:

Redemonstrated hypoenhancing lesion occupying the sella measuring approximately 1.6 x 1.4 cm in transcoronal diameter. The mass is overall T2-hyperintense but contains an ovoid T2 hypointense and slightly T1-hyperintense 1.0 x 0.8 cm component inferiorly compatible with intralesional hemorrhage. Infundibulum is displaced superiorly and to the left. The native gland is displaced posteriorly with a thin rind along the posterior margin. Lobulated suprasellar extension approaches and likely contacts the optic chiasm without mass effect. Normal appearance of cavernous sinuses.

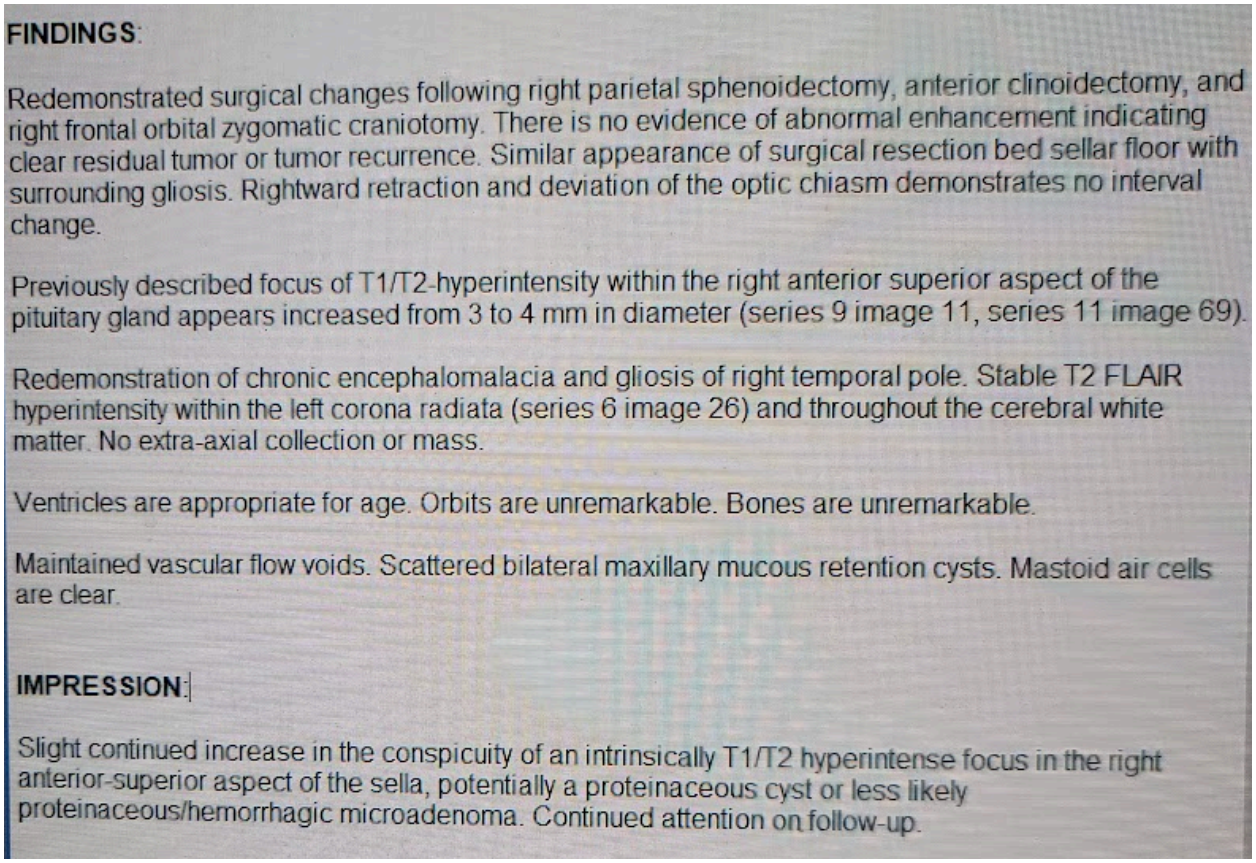
No diffusion restriction, hydrocephalus, acute hemorrhage, midline shift, or extra axial fluid collection.

Orbits are unremarkable. Mastoid air cells are clear. No destructive osseous lesions.

IMPRESSION:

1. Findings compatible with a 1.6 cm hemorrhagic pituitary macroadenoma.
2. Chiasmatic contact without mass effect. No cavernous sinus involvement.

MICROADENOMA



FINDINGS:

Redemonstrated surgical changes following right parietal sphenoidectomy, anterior clinoidectomy, and right frontal orbital zygomatic craniotomy. There is no evidence of abnormal enhancement indicating clear residual tumor or tumor recurrence. Similar appearance of surgical resection bed sellar floor with surrounding gliosis. Rightward retraction and deviation of the optic chiasm demonstrates no interval change.

Previously described focus of T1/T2-hyperintensity within the right anterior superior aspect of the pituitary gland appears increased from 3 to 4 mm in diameter (series 9 image 11, series 11 image 69).

Redemonstration of chronic encephalomalacia and gliosis of right temporal pole. Stable T2 FLAIR hyperintensity within the left corona radiata (series 6 image 26) and throughout the cerebral white matter. No extra-axial collection or mass.

Ventricles are appropriate for age. Orbits are unremarkable. Bones are unremarkable.

Maintained vascular flow voids. Scattered bilateral maxillary mucous retention cysts. Mastoid air cells are clear.

IMPRESSION:

Slight continued increase in the conspicuity of an intrinsically T1/T2 hyperintense focus in the right anterior-superior aspect of the sella, potentially a proteinaceous cyst or less likely proteinaceous/hemorrhagic microadenoma. Continued attention on follow-up.

SEIZURE

CLINICAL HISTORY: History of epilepsy status post right parietal cortical dysplasia resection 2017 and nonepileptic seizures. Currently admitted following complex partial status epilepticus.

COMPARISON: CT head 10/5/2019.

PROCEDURE COMMENTS: MRI of the brain was performed without IV contrast.

FINDINGS:

No areas of restricted diffusion or susceptibility artifact to suggest acute hemorrhage or infarction.

Hippocampal formations maintain normal, symmetric signal and overall volumes. Slightly irregular rounded configuration of left hippocampus, with subtle vertical orientation of collateral sulcus.

Remote right frontoparietal craniotomy, with underlying postoperative encephalomalacia and minimal marginal gliosis of right superior parietal lobule.

No extra-axial fluid collection or mass. Ventricles are appropriate for age.

Orbits are unremarkable. Visualized paranasal sinuses are clear.

Mastoid air cells are clear. Bones are unremarkable.

IMPRESSION:

1. No acute intracranial pathology or acute post ictal stigmata.
2. Partial left hippocampal incomplete inversion.
3. Prior craniotomy and cortical resection of right superior parietal lobule.

DDX

BASAL GANGLIA

Class	Diagnosis	Typical MR Findings
Toxic	Carbon monoxide poisoning	Preferential involvement of the **globi pallidi** (restricted diffusion, ↑T2 signal, ±↑T1 signal) **Delayed leukoencephalopathy** with periventricular white matter ↑T2/FLAIR signal and cerebral atrophy developing over months
	Cyanide	Basal ganglia and cerebral cortex (especially primary sensorimotor cortices) ↑T2/FLAIR signal and restricted diffusion Can have associated **hemorrhagic necrosis** (↑T1 signal) of the basal ganglia and cortical laminar necrosis
	Methanol	Basal ganglia and cerebral white matter ↑T2/FLAIR signal and restricted diffusion **Hemorrhagic putaminal necrosis**, can also involve subcortical white matter Can present with **optic neuritis**

Metabolic	Liver disease	Cirrhosis: ↑T1 signal in the globi pallidi and substantia nigra Acute hyperammonemia: ↑T2/FLAIR signal and restricted diffusion in the bilateral basal ganglia, insula, and cingulate gyri
	Nonketotic hyperglycemia	↑T1 signal, ±↑T2/FLAIR signal in the **lentiform nuclei**, typically **unilateral**
	Hypoglycemia	↑T2/FLAIR signal and restricted diffusion in the bilateral cerebral cortices, hippocampi, and basal ganglia ± white matter involvement
	Total parental nutrition	↑T1 signal in the bilateral globi pallidi and subthalamic nuclei, **T2 signal should be normal**
	Osmotic demyelination	↑T2/FLAIR signal in the **central pons**, can also affect cerebellum, thalami, globi pallidi, and putamina
Ischemic	Lacunar infarcts	Restricted diffusion and ↑T2/FLAIR signal, typically **asymmetric** Artery of Percheron occlusion can cause symmetric bilateral thalamic infarcts
	Deep cerebral venous thrombosis	↑T2/FLAIR signal ± restricted diffusion involving the bilateral basal ganglia, thalami, and internal capsules
	Hypoxic-ischemic encephalopathy	↑T2/FLAIR signal and restricted diffusion involving the bilateral basal ganglia and thalami when severe Affects watershed zones when mild to moderate

Inborn errors of metabolism	Wilson disease	↑T2/FLAIR signal in the basal ganglia (especially putamina) and ventrolateral thalami ± subcortical white matter, brainstem, dentate nuclei Restricted diffusion can be seen early in the disease
	Leigh disease	Symmetric ↑T2/FLAIR signal in the basal ganglia (especially **putamina**), periaqueductal gray matter, and cerebral peduncles ↑Lactate peak on MR spectroscopy
Neuro-degenerative	Wernicke encephalopathy	↑T2/FLAIR signal in the medial thalami, periaqueductal gray matter, tectum, and mamillary bodies
	Neurodegeneration with Brain Iron Accumulation (NBIA)	↓T2 signal in the bilateral globi pallidi Central ↑T2 signal in the subset of patients with pantothenate kinase mutations
	Creutzfeldt-Jakob disease	**Restricted diffusion** and ↑T2/FLAIR signal in the basal ganglia (especially caudate and putamen) and **pulvinar nucleus** of thalamus
	Fahr disease	↓**SWI signal** in the basal ganglia, thalami, dentate nuclei, and subcortical white matter with variable T1/T2 signal

Infectious	Flavivirus encephalitis	↑T2/FLAIR signal in the bilateral basal ganglia Japanese encephalitis classically involves the dorsomedial thalami West Nile virus typically involves the bilateral basal ganglia and thalami diffusely
	Neurotoxoplasmosis	Discrete lesions with peripheral enhancement and variable T1/T2 signal, which can involve the basal ganglia
Neoplastic	CNS lymphoma	↓**T2 signal** and diffuse enhancement in lesions which may be bilateral but are rarely perfectly symmetric Propensity for subependymal spread
	Bilateral thalamic glioma	↑T2/FLAIR signal and enlargement of the bilateral thalami, no enhancement
Other	Neuro-Behçet	Multiple ↑T2/FLAIR hyperintense lesions which can involve the basal ganglia
	Neurofibromatosis type 1	Multifocal ↑T2/FLAIR hyperintense lesions without mass effect, can involve basal ganglia (particularly globi pallidi)