

ORBITAL IMAGING:

Imaging Tips for Ophthalmologists



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OVERVIEW

- Basics: Basic imaging strategies
 - Utility of CT & MRI
 - When to add contrast agents
- When to order “special” sequences:
 - Angiographic imaging (CTA vs MRA vs DSA)
 - Venography
- Review of modality & sequence basics
 - Interpretation tips for DWI & other “special sequences”
 - Pitfalls in interpretation
- Illustrative cases

MODALITY CONSIDERATIONS

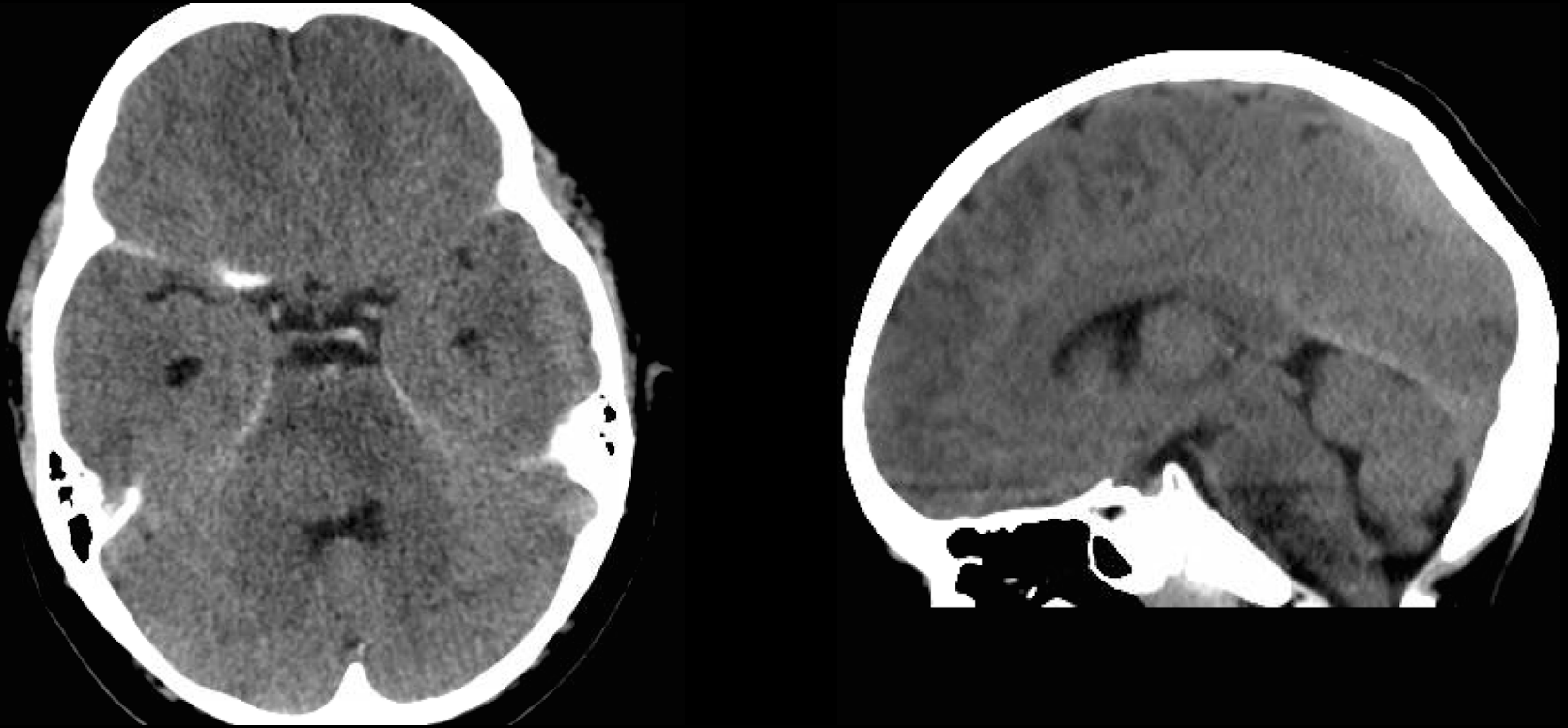
- **Non-contrast CT head (NECT):**
 - Trauma
 - Rule out brain tumor, hemorrhage or stroke
- NECT is a *basic screening tool*
- Limitations of head CT:
 - Relatively low resolution &/or field of view
 - Poor evaluation of orbital pathology
 - Poor soft tissue contrast:
 - Limits evaluation of early acute ischemia
 - Less sensitive for demyelinating disease

What can be missed with head CT?

- Young boy seen in ED
- Acute R 6th CN palsy
- Routine NECT head ordered to r/o mass

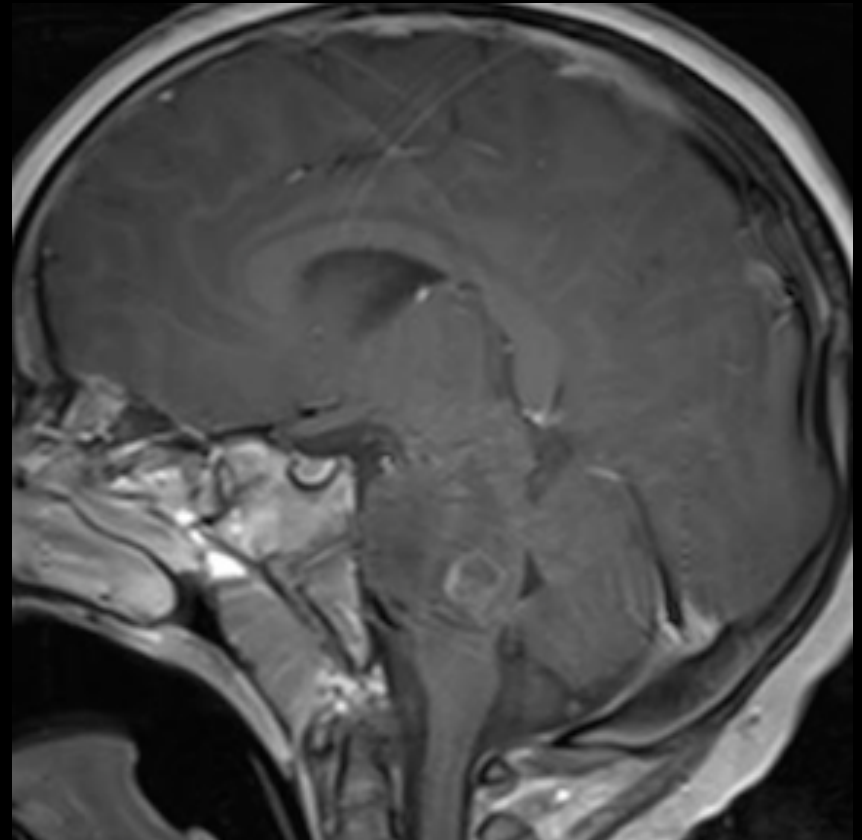
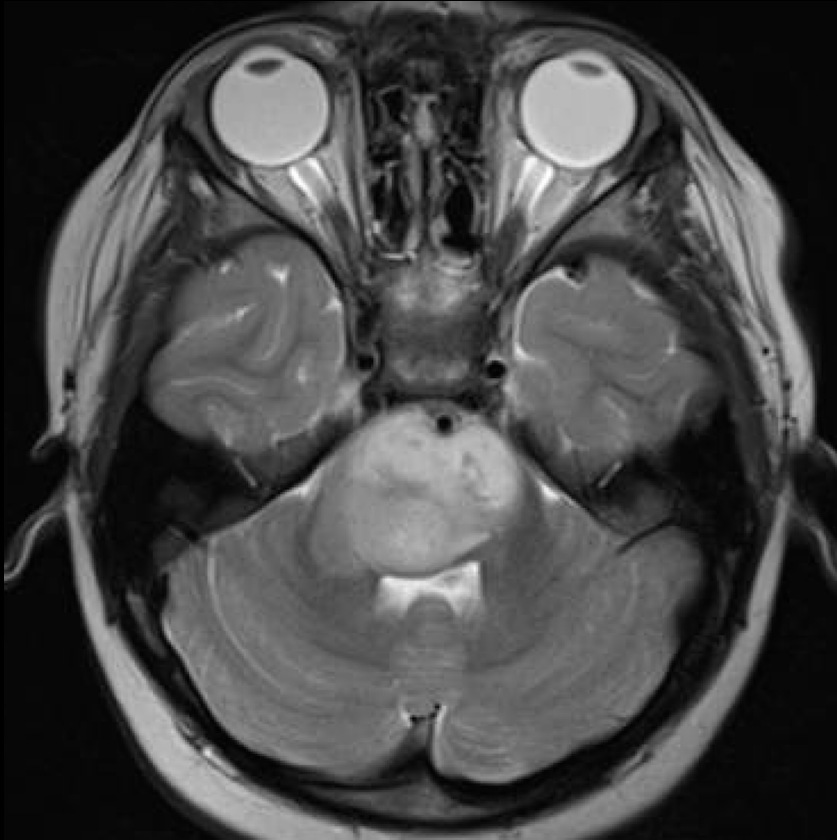


5 Y F: NEW R CN6 PALSY



Subtle mass effect & density abnormalities can be missed
Dedicated exam may be indicated even if the head CT screen appears negative

FOLLOW-UP MRI



Diffuse infiltrating high-grade glioma

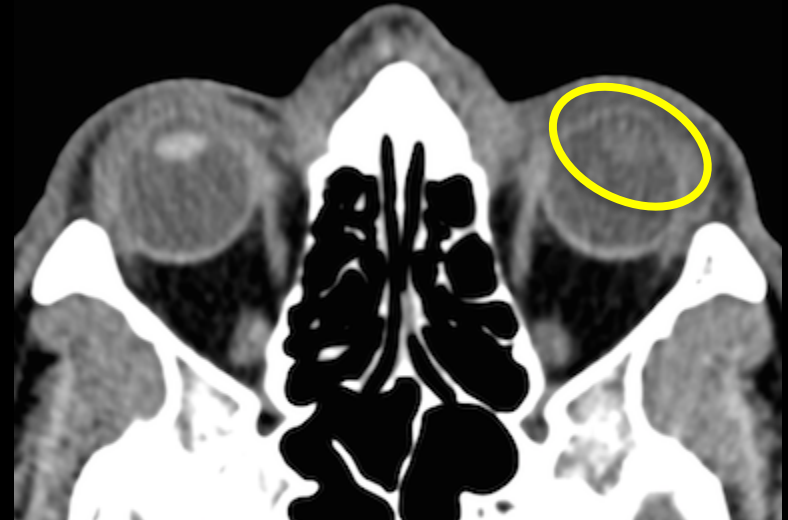
INDICATIONS FOR ORBITAL CT

- Must order **orbital CT** to evaluate the orbits!
 - Trauma, and post-traumatic complications (non-con)
 - Contrast important for suspected infections
- NECT is best for osseous involvement and to evaluate for calcifications or as a general screen
 - Orbital fractures, mucocele, thyroid eye disease
 - Ca++ in retinoblastoma and meningioma
 - General: r/o major mass lesion or hemorrhage
 - Primary involvement of bone/skull base (e.g. fibrous dysplasia, osseous metastasis)

ORBITAL TRAUMA

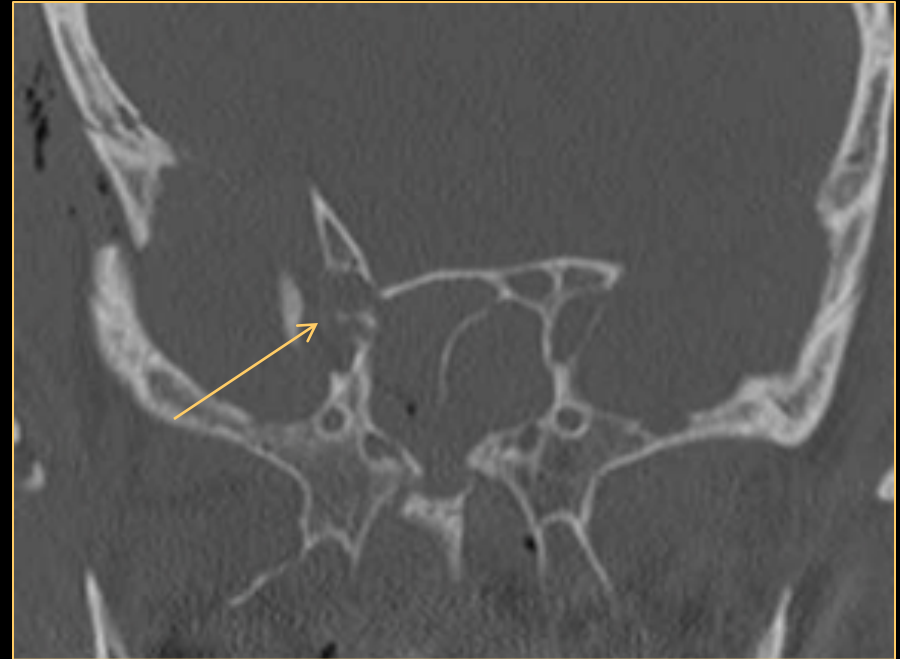
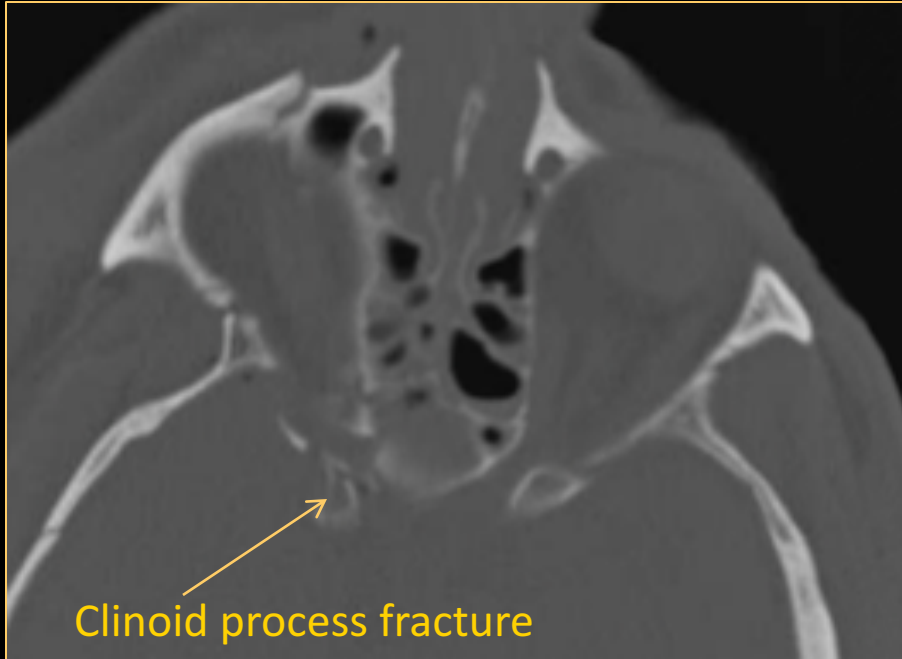
- Imaging in globe rupture
 1. “Flat tire” sign
 2. Globe shape deformity
 3. Ocular volume loss
 4. Asymmetric anterior chamber depth
- Things to watch for:
 - Foreign bodies
 - Lens (presence/density)

Globe rupture, hemorrhage, glass FB



Vanishing lens sign: Posttraumatic cataract

TRAUMATIC OPTIC NEUROPATHY



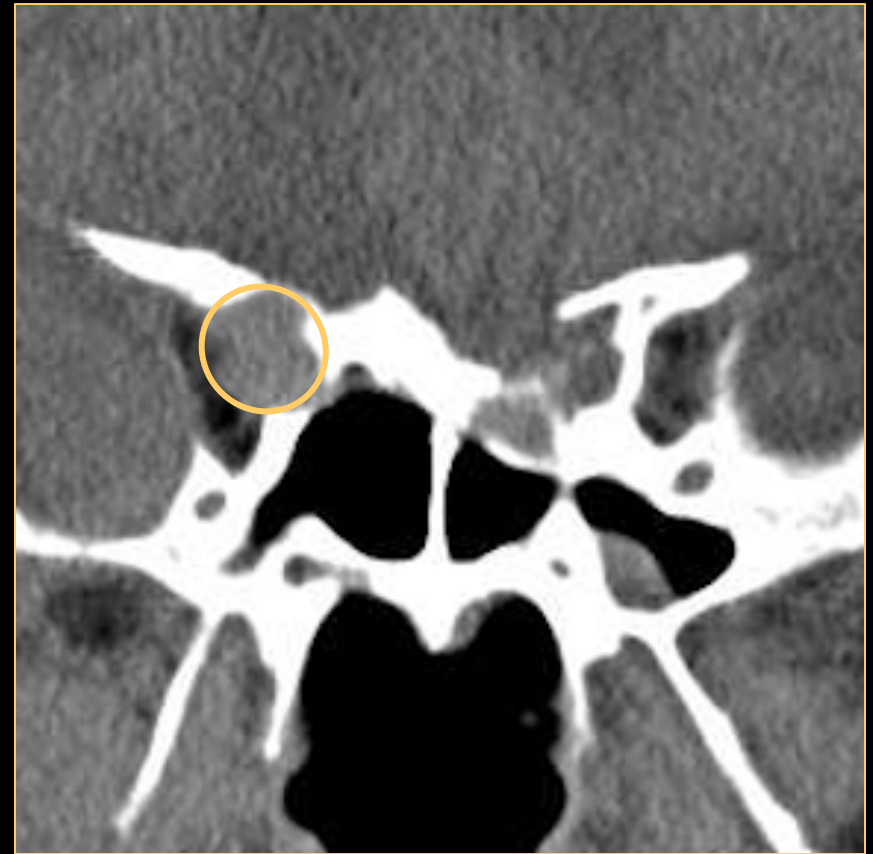
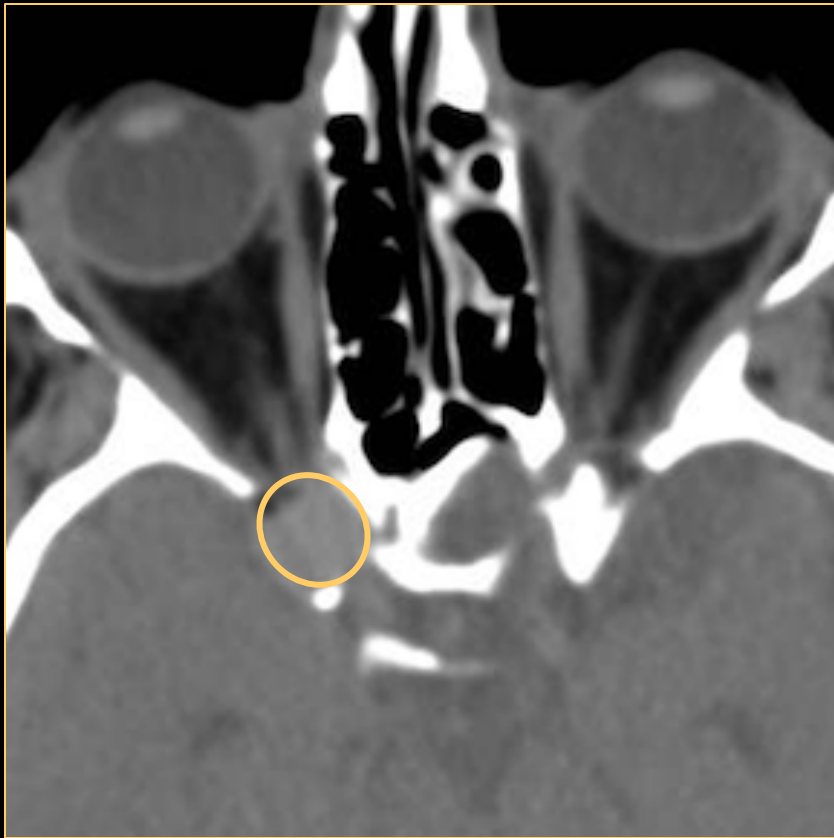
Acute complications
Fracture with impingement

ORBITAL TRAUMA: DELAYED COMPLICATIONS

- CT (orbits) best evaluates osseous findings
 - Acutely: Fractures & soft tissue impingement
 - Delayed: Complications of prior trauma
- Delayed complications: Mucocele
 - Involvement of clinoid process can result in progressive (late) vision loss
- Enhanced orbital MRI is often complementary in orbit & sinus pathology for bony lesions

46 Y M REMOTE TRAUMA, BLIND OS

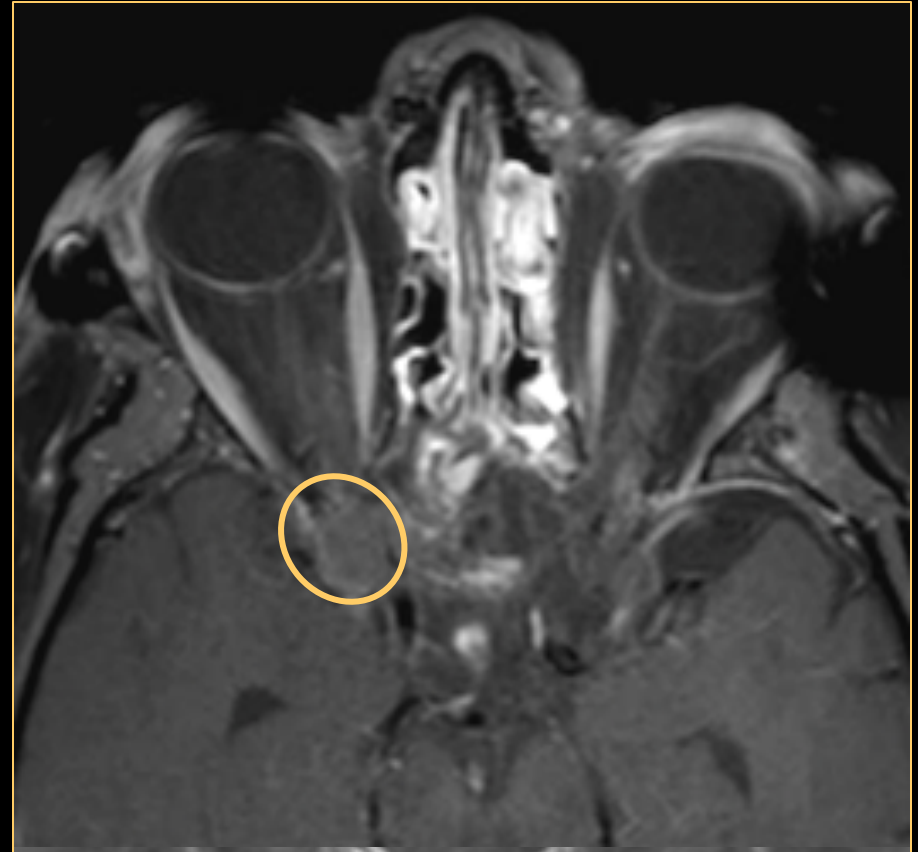
Progressive superior visual field deficit OD



Soft tissue windows from orbital CT

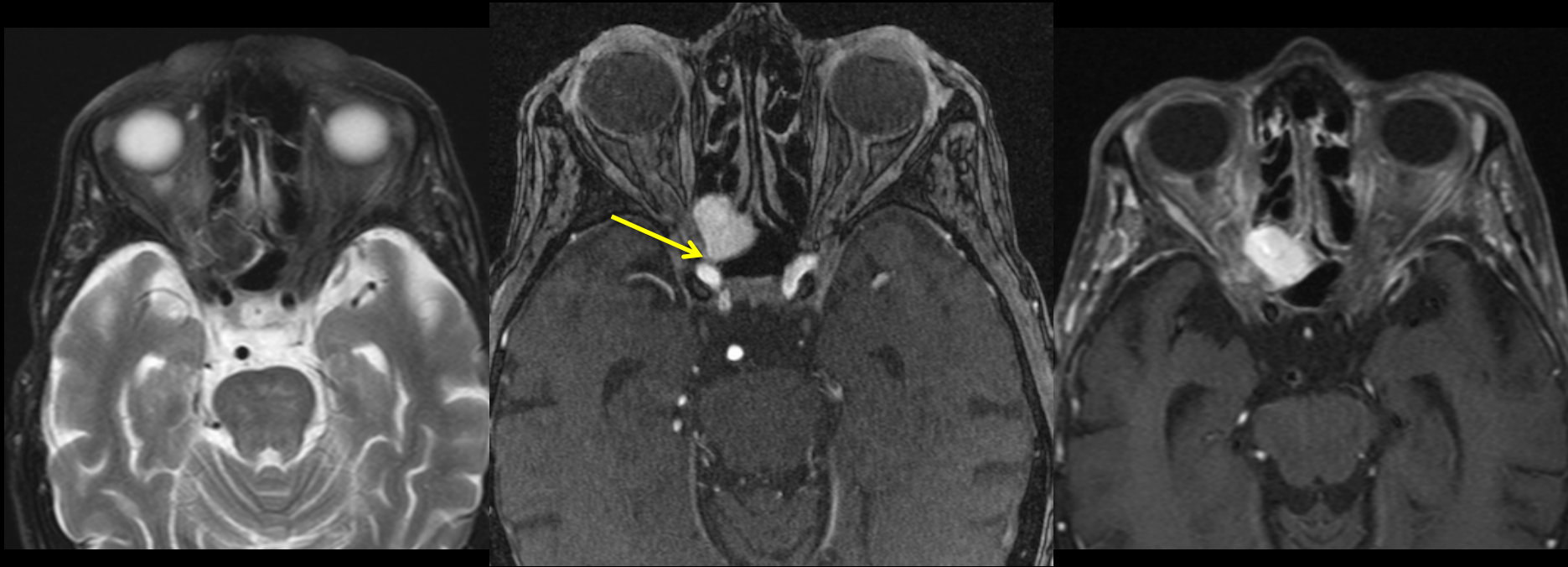
CHRONIC TRAUMATIC OPTIC NEUROPATHY

Clinoid process mucocele



Late complications

GIANT THROMBOSED ICA ANEURYSM



Signal on MRI follows subacute blood if thrombosed

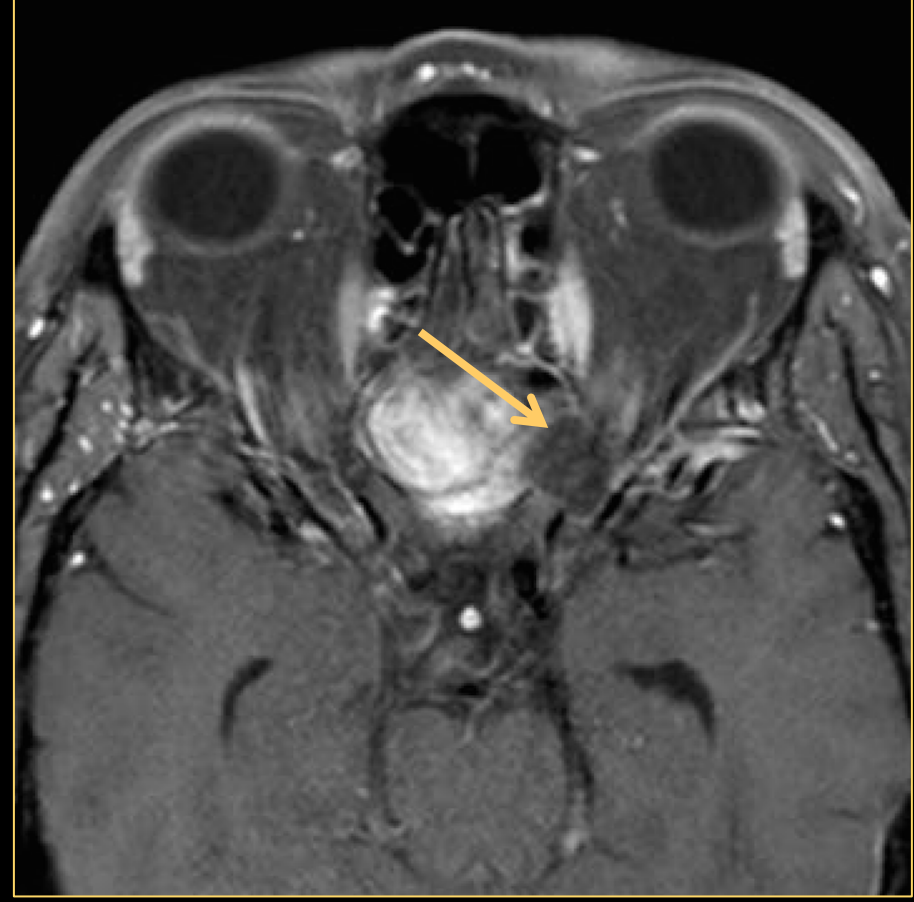
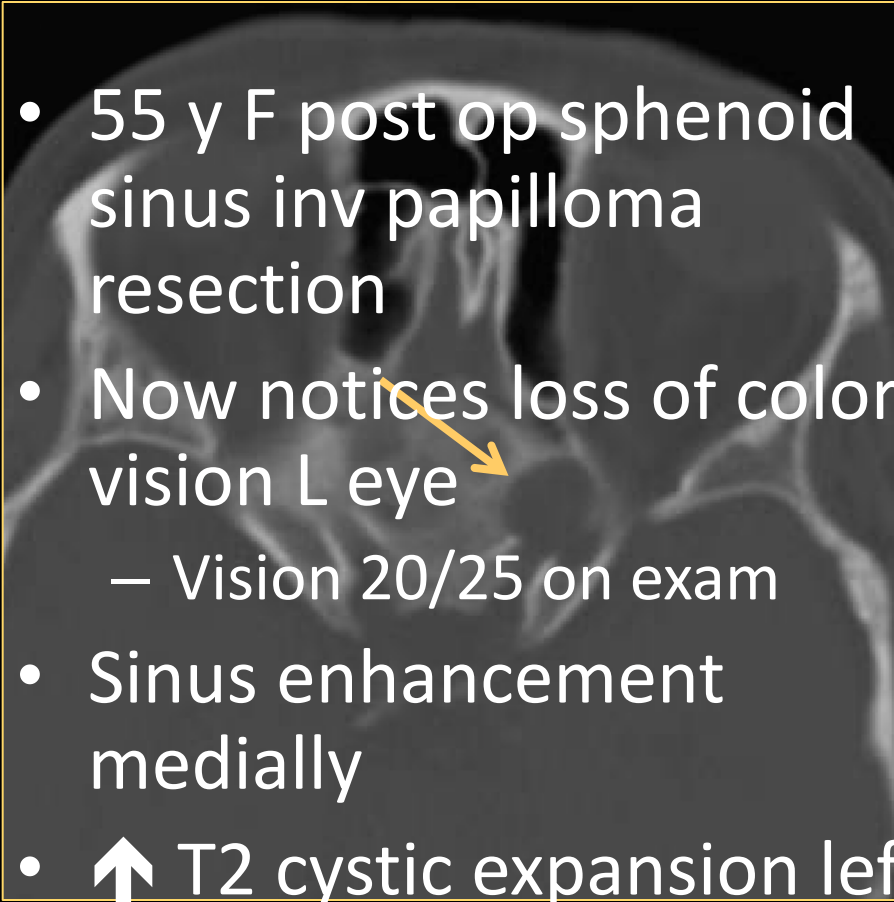
Do not call enhancement without pre-contrast T1

CT benign bone erosion + MRI signal mimics mucocele

Contiguity with vessel key – MRA/CTA to help sort

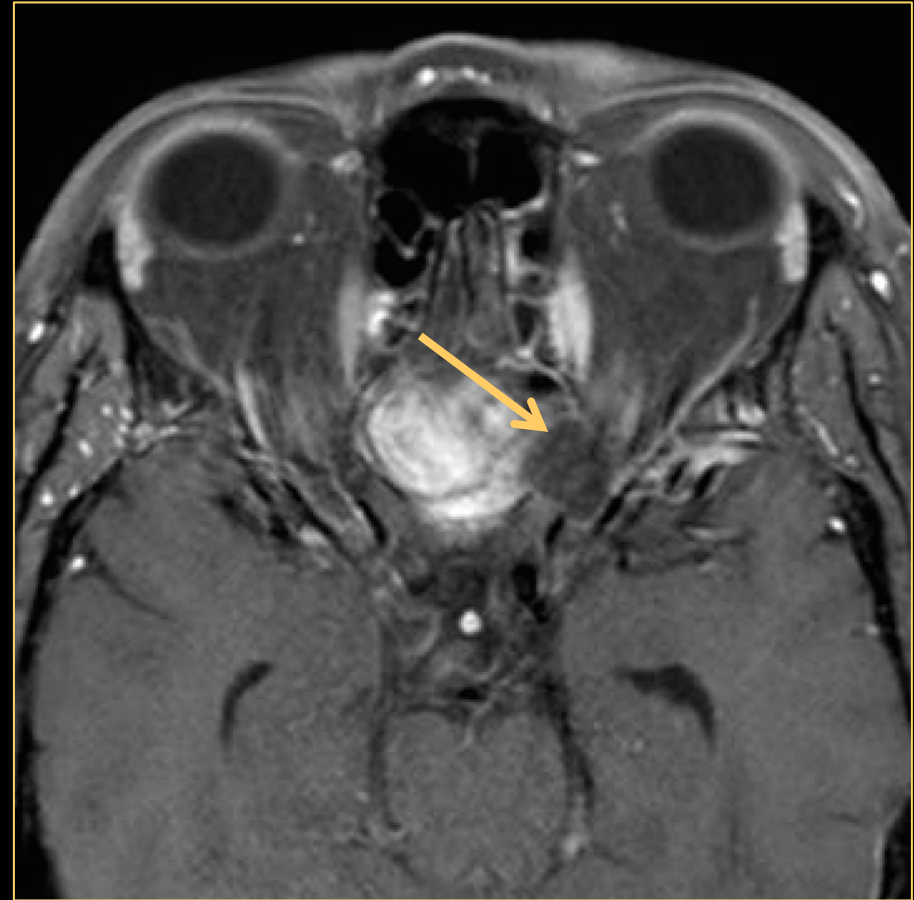
RECURRENT INVERTING PAPILOMA

- 55 y F post op sphenoid sinus inv papilloma resection
- Now notices loss of color vision L eye
– Vision 20/25 on exam
- Sinus enhancement medially
- ↑ T2 cystic expansion left clinoid compresses CN 2



RECURRENT INVERTING PAPILOMA

- Clinical history is key
- Imaging clues
 - MRI shows enhancement in adjacent ethmoids, more than peripheral mucosa
 - CT shows lytic area on with aggressive loss of cortical bone – punched out without bone expansion



WHEN TO DO ORBITAL CT

- **Imaging tips:**
 - Dedicated orbital lesion, proptosis, suspect mass, unable MR
 - Contrast is not required for anatomic assessment or preoperative planning such as thyroid eye disease
- **Contrast is indicated when:**
 - Diagnosis is in doubt (possible idiopathic orbital inflammation or other inflammatory disorder)
 - Possibility of neoplasm
 - Inflammation or infection is suspected
- **Do NOT do: CT with & without contrast:**
 - Just do orbits with contrast
 - Doubles radiation dose (to lens!) & no added value

ORBIT CT WITHOUT CONTRAST

Presented with vision loss, h/o hyperthyroidism post thyroidectomy



Typical case of thyroid eye disease

THYROID ASSOCIATED ORBITOPATHY

- NECT orbits sufficient to confirm diagnosis
- Typical findings:
 - Proptosis
 - Increased orbital fat
 - Extraocular muscle enlargement
 - > 5 mm thickness (“I’M SLOW”)
 - Spares tendon insertions
 - Fatty muscle infiltration
 - Relatively symmetric bilaterally

INDICATIONS FOR MRI

- Suspected orbital or intracranial neoplasm
- **Orbital C+ FS MRI** best to evaluate optic nerve & limited orbital involvement (many need brain too):
 - Neoplasms (glioma, meningioma, “hemangioma”)
 - Radiation-injury
 - Demyelinating diseases & inflammatory disorders
 - Confirm optic atrophy (ass’d anomalies)
- Papilledema: **Brain MRI** to evaluate ↑ ICP
 - MRV to rule out dural sinus thrombosis
- Brain & orbits best in most cases, if there is potential for intracranial involvement

SPECIFIC CLINICAL SCENARIOS

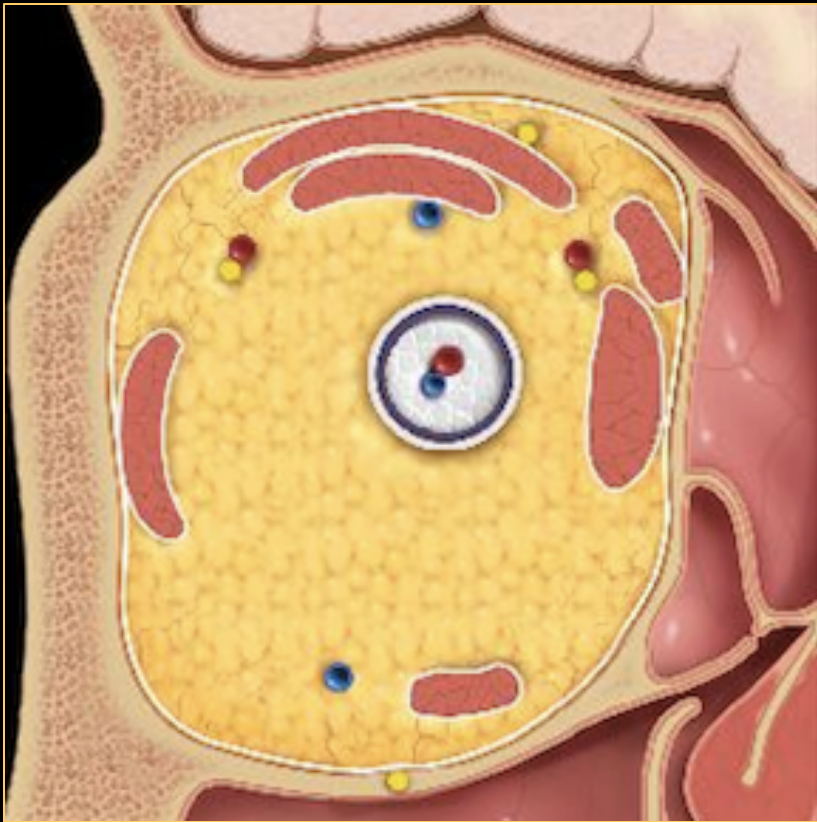
- Bitemporal hemianopsia:
 - Dedicated **pituitary (+/- brain) MRI w & w/o**
 - Best evaluates sellar masses
- Pupil-involving 3rd CN palsy
 - Orbits/brain w & wo: r/o compressive lesion
 - **MRA (or CTA)** to evaluate for p comm aneurysm
- Pupil-sparing 3rd CN palsy
 - Typical in patients with vascular risk factors
 - Likely small vessel ischemia, so brain MRI sufficient
- Pseudopapilledema if suspect optic drusen*:
 - NECT orbits or NECT head
 - B mode ultrasound

MRI ORBITS

Review basics: T1 fat bright, fluid dark



CORONAL ANATOMY



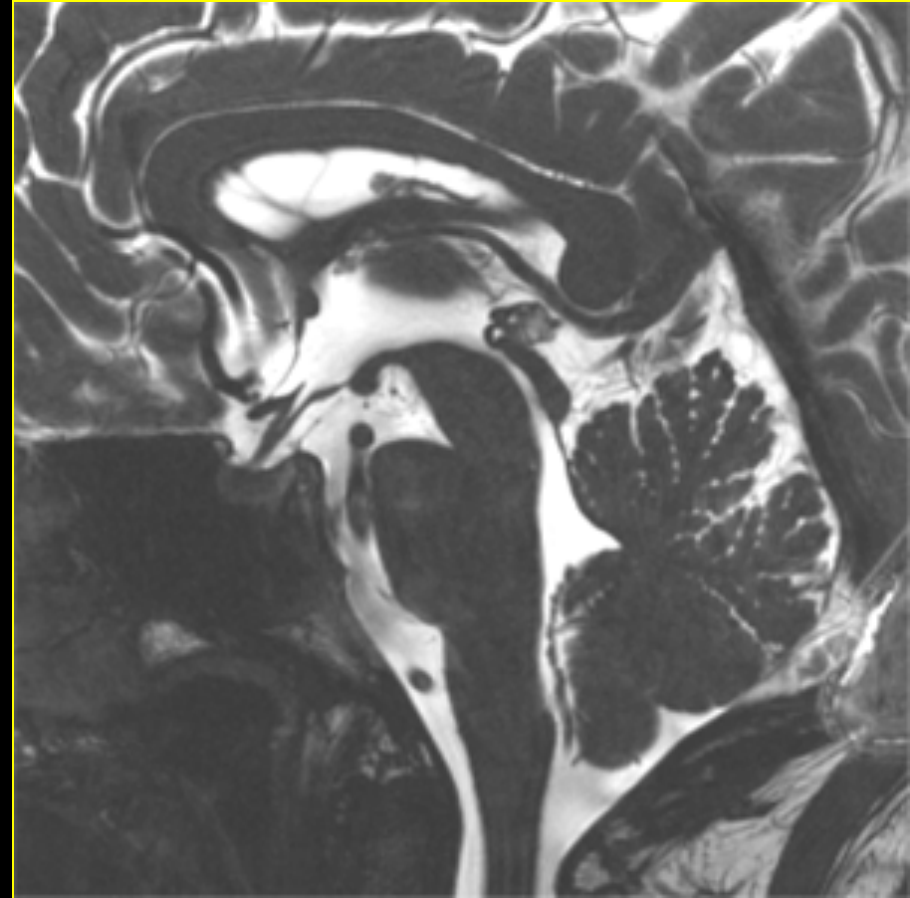
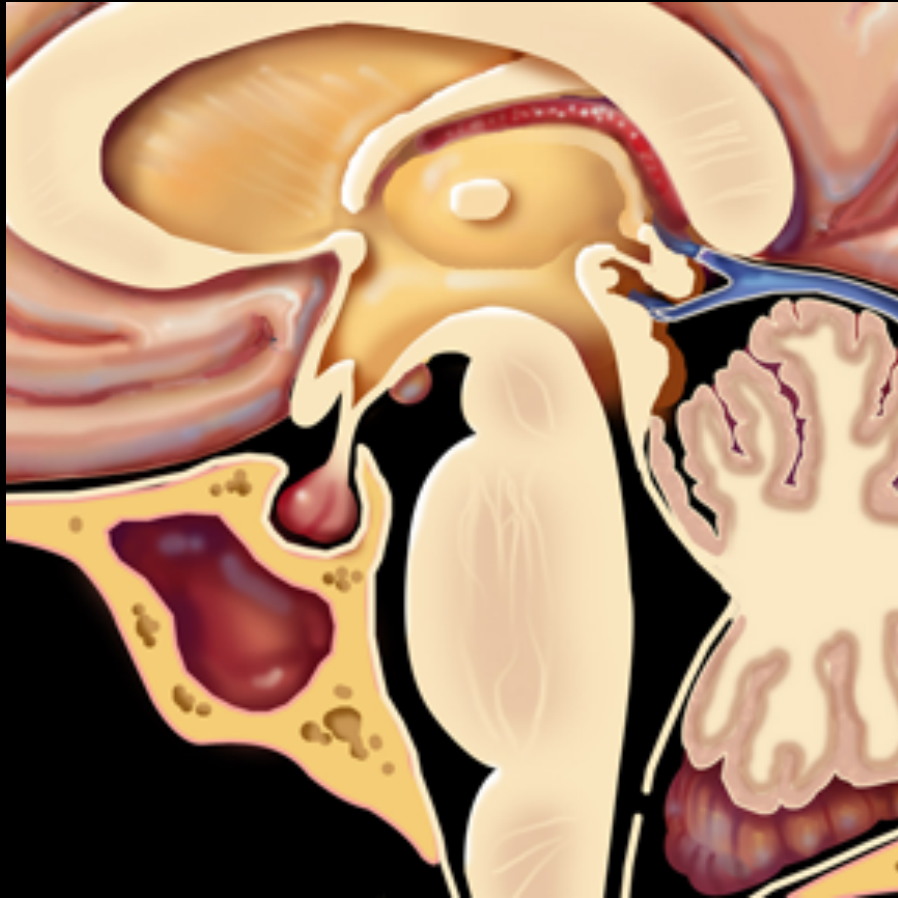
Optic Nerve-Sheath Complex

BITEMPORAL HEMIANOPSIA

- Dedicated pituitary imaging is important for evaluation of suspected sellar mass
- Employs a larger FOV (field of view) & thinner slices to better demonstrate lesions in this region
- Most often pituitary macroadenomas
- Many other pathologies possible

NORMAL SAGITTAL ANATOMY:

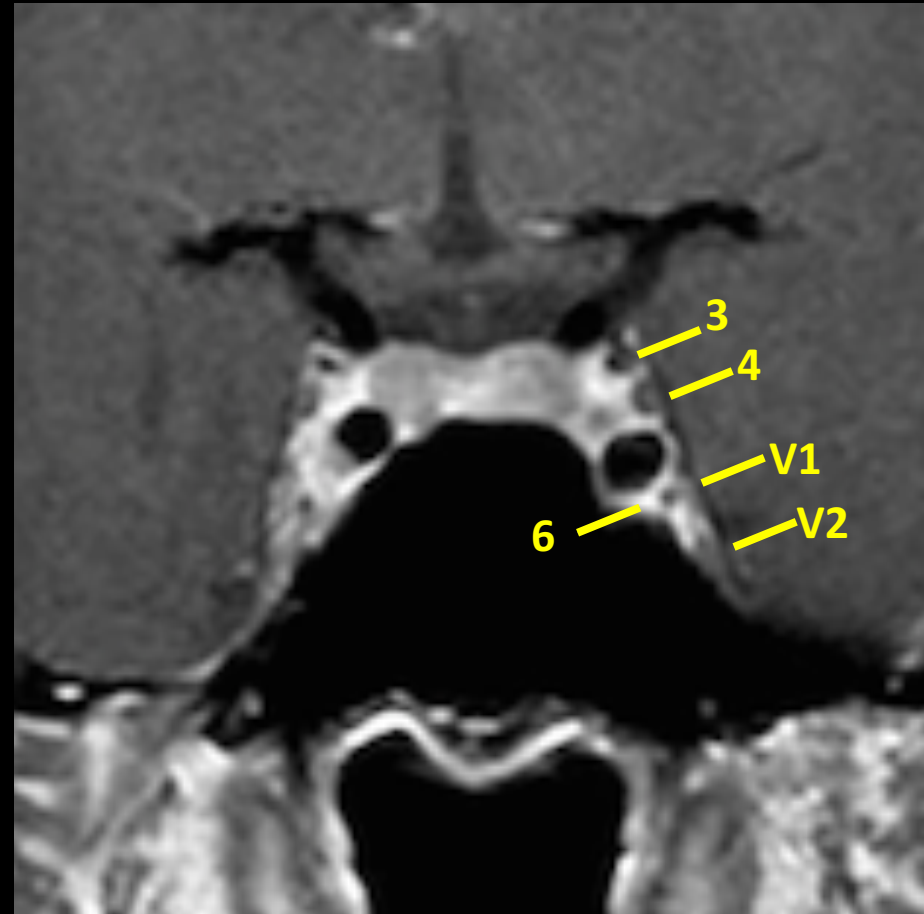
Sellar & Parasellar Region



Basics: T2 weighted MRI - Fluid is white

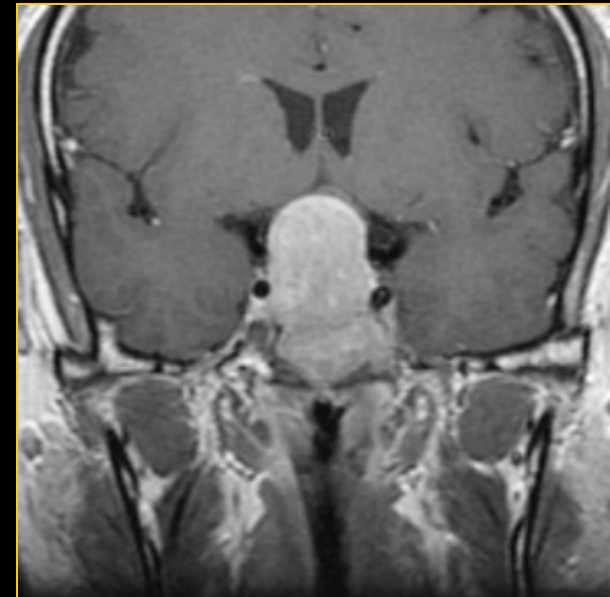
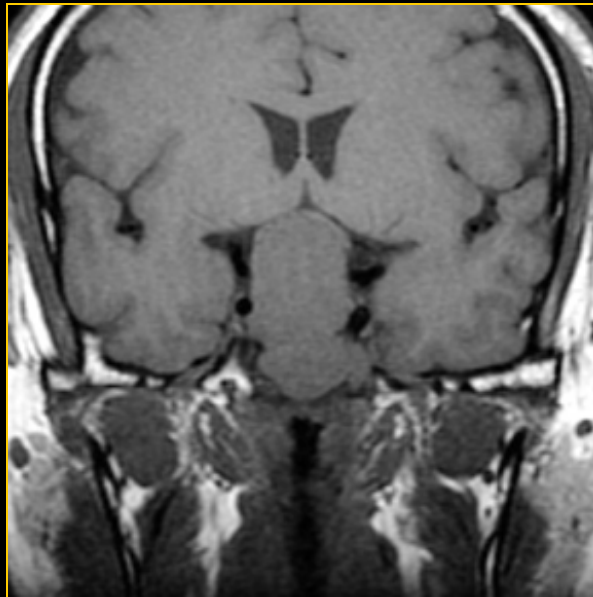
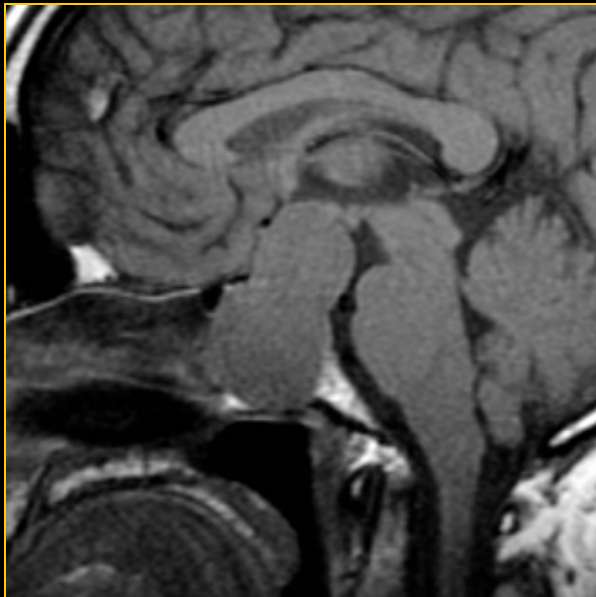
NORMAL CORONAL ANATOMY:

Sellar & Parasellar Region



CENTRAL SKULL BASE PATHOLOGY:

Diagnosis?



- Center of mass: sella
- Spread pattern:
 - Superior → suprasellar cistern
 - Inferior → Sphenoid sinus and/or clivus
- **Invasive pituitary macroadenoma**

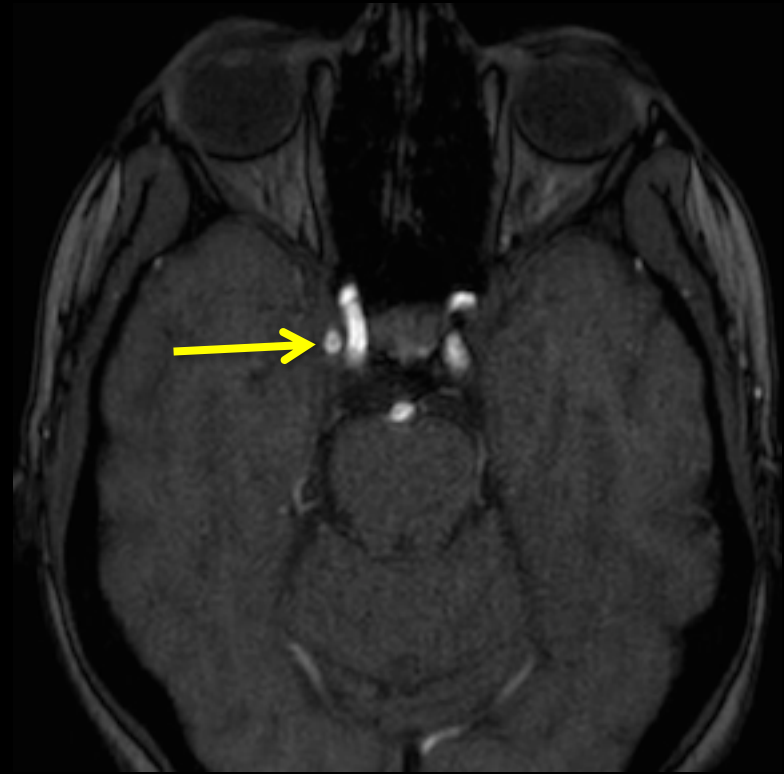
VASCULAR IMAGING

- **Non-invasive alternatives** to conventional DSA
 - CTA head & neck
 - MRA (with or without contrast) head & neck
 - Doppler US neck vessels
- **Conventional angiography** (gold standard)
 - dAVF, CCF, vasculitis
- **Venography** alternatives:
 - CTV (quick & fewest technical artifacts)
 - MRV (longer, really need brain MRI, more artifacts)
 - US (neck only)
 - CTA/CTV or Brain & MRV for CCF or cavernous sinus thrombosis → gold standard DSA
- **Vessel wall imaging (MRI):** Vasculitis vs RCVS

NEW RIGHT CN III PALSY



MIP (maximum intensity projection) image RICA
Right posterior communicating artery aneurysm



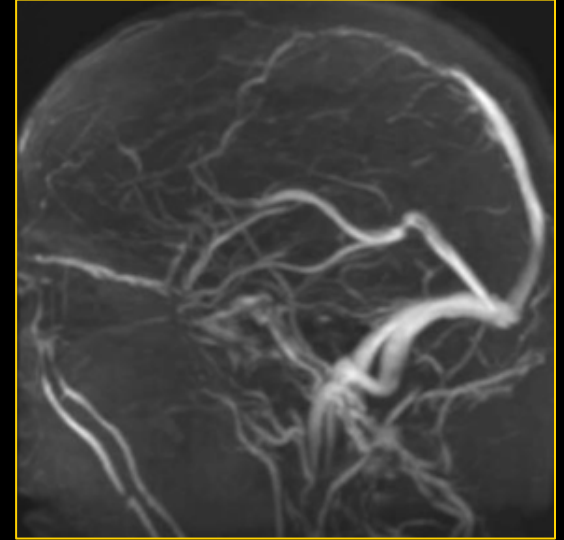
Stacked axial source images from MRA
Helpful to confirm; r/o artifacts

VENOUS DISEASE

Enhanced
CTV



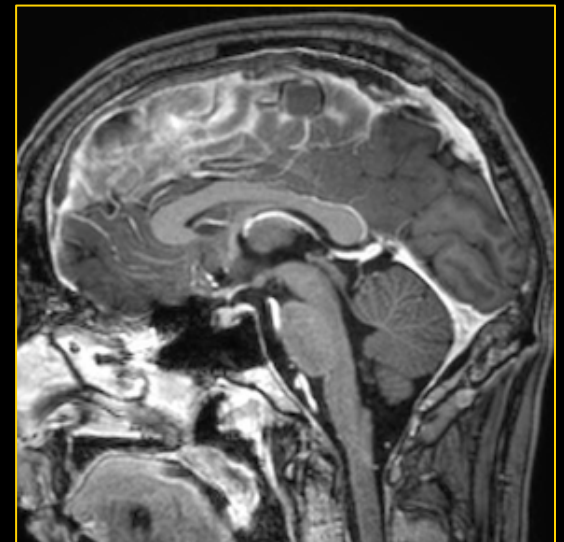
MRV



NECT

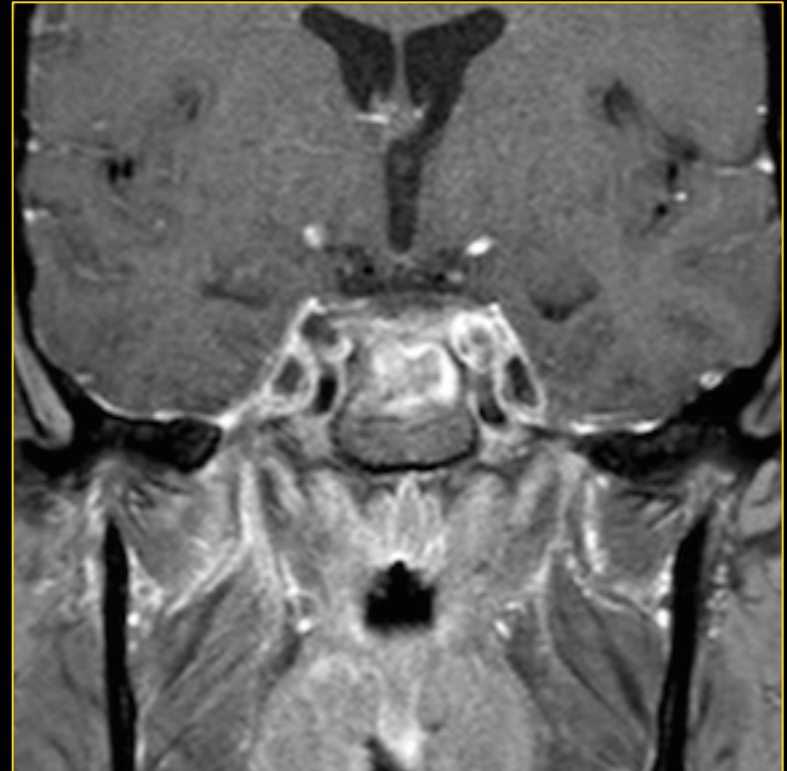
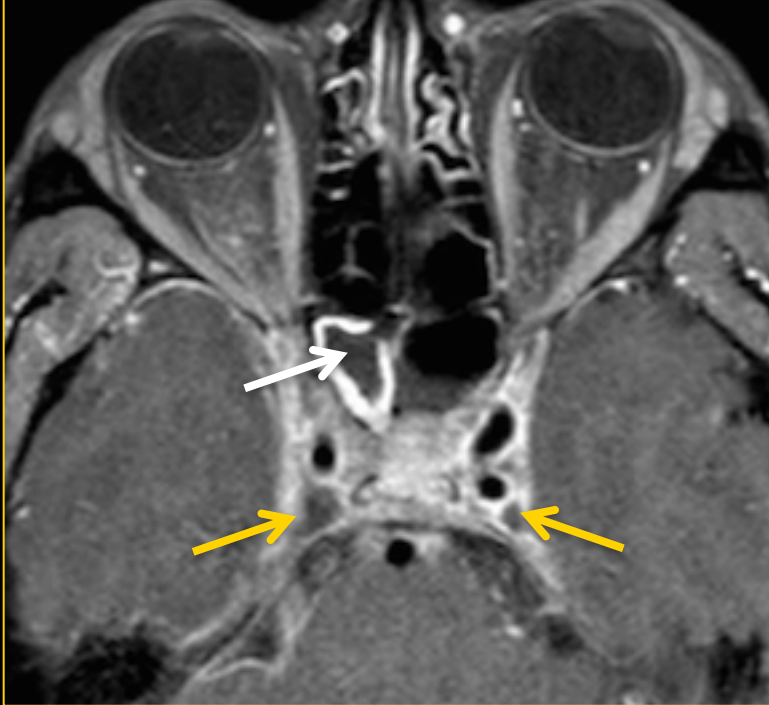


T1 C+



CAVERNOUS SINUS THROMBOSIS

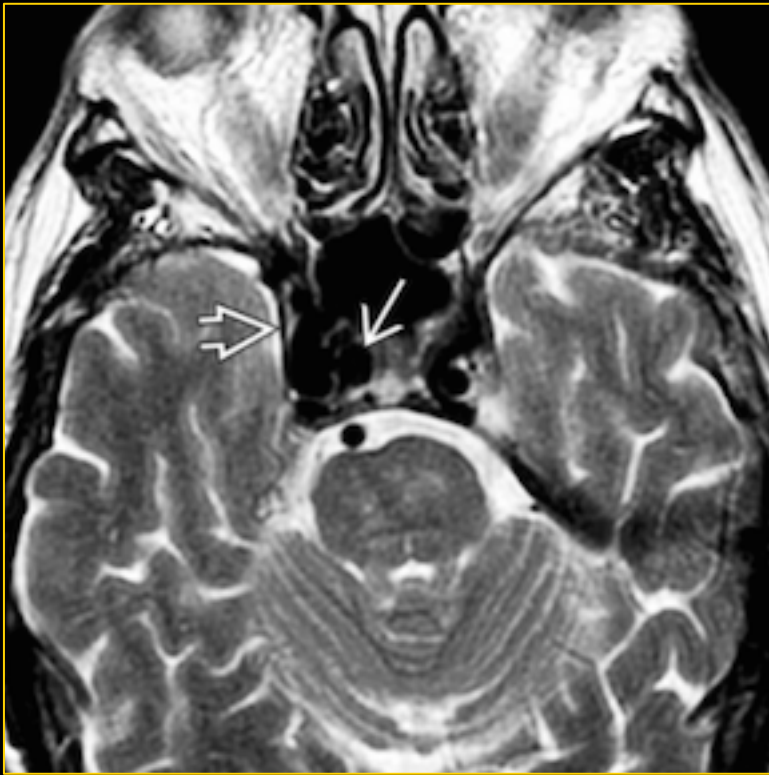
Definitive by C+ orbit MRI



Lack of normal enhancement cavernous sinuses
Look for adjacent sphenoid sinus infection

CAVERNOUS CAROTID FISTULA

Suspicious by MRI



Definitive on DSA; allows tx



MRI: Flow voids in cavernous sinus; enlarged SOV

DSA: Abnormal cavernous sinus filling during arterial injection

COMMON MRI SEQUENCE VARIATIONS

- Orbital enhanced imaging requires fat sat
- STIR is “short tau inversion recovery”:
 - Essentially used to null out fat signal on T2 MR
- FLAIR is “fluid attenuated inversion recovery”
 - Primarily used to assess white matter lesions
 - Particularly useful for MS & Susac syndrome
- DWI is diffusion weighted imaging
 - Not only for stroke: discussion to follow

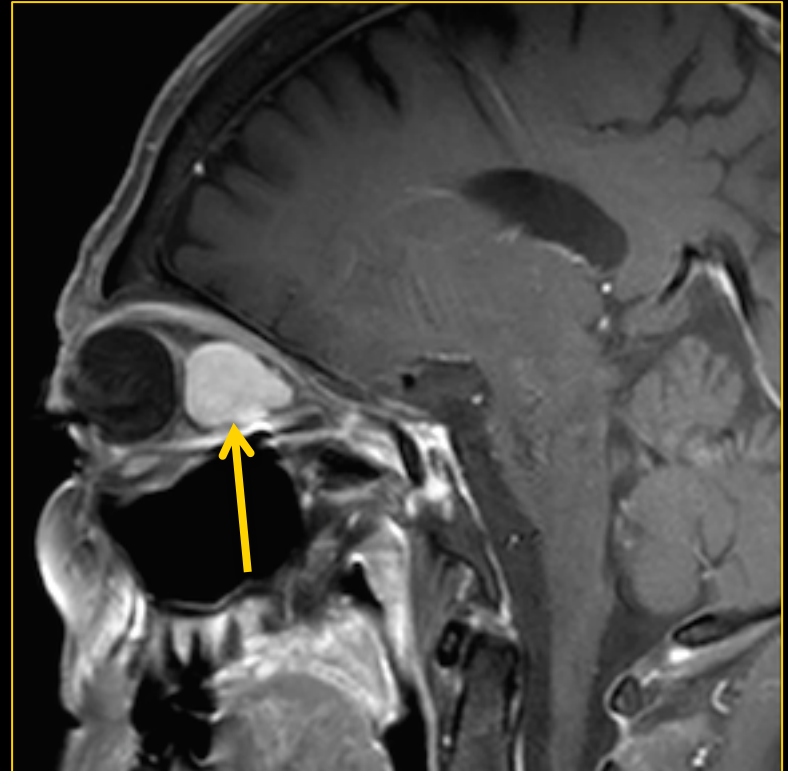
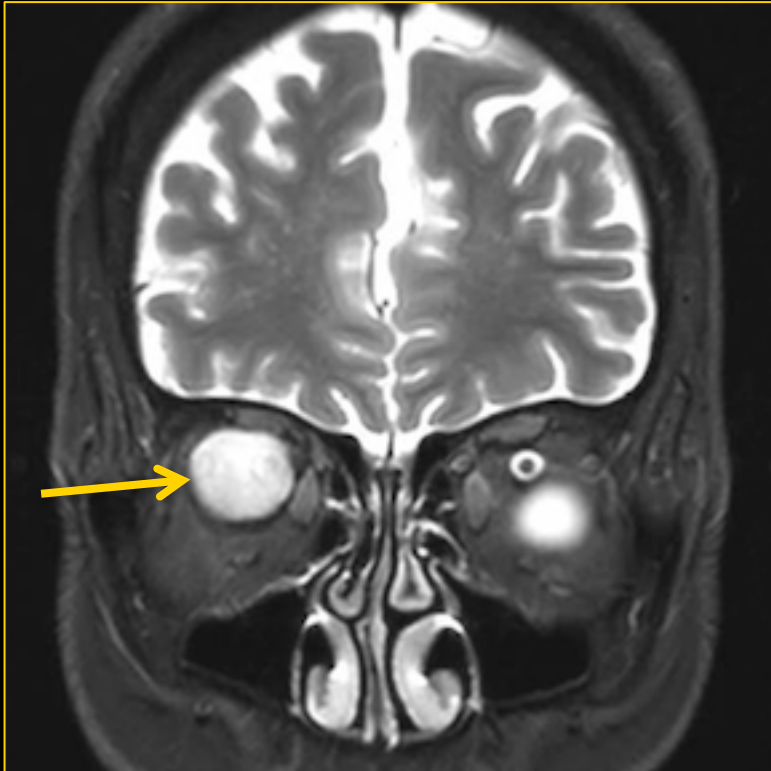
ENHANCED ORBITAL MRI

- Post-contrast T1s require fat saturation
 - Can be source of worse technical (susceptibility) artifacts
- Pre-contrast T1 next best sequence in absence of fat sat post gad
 - Tumor have low T1 signal, demonstrate inherent tissue contrast relative to orbital fat

???



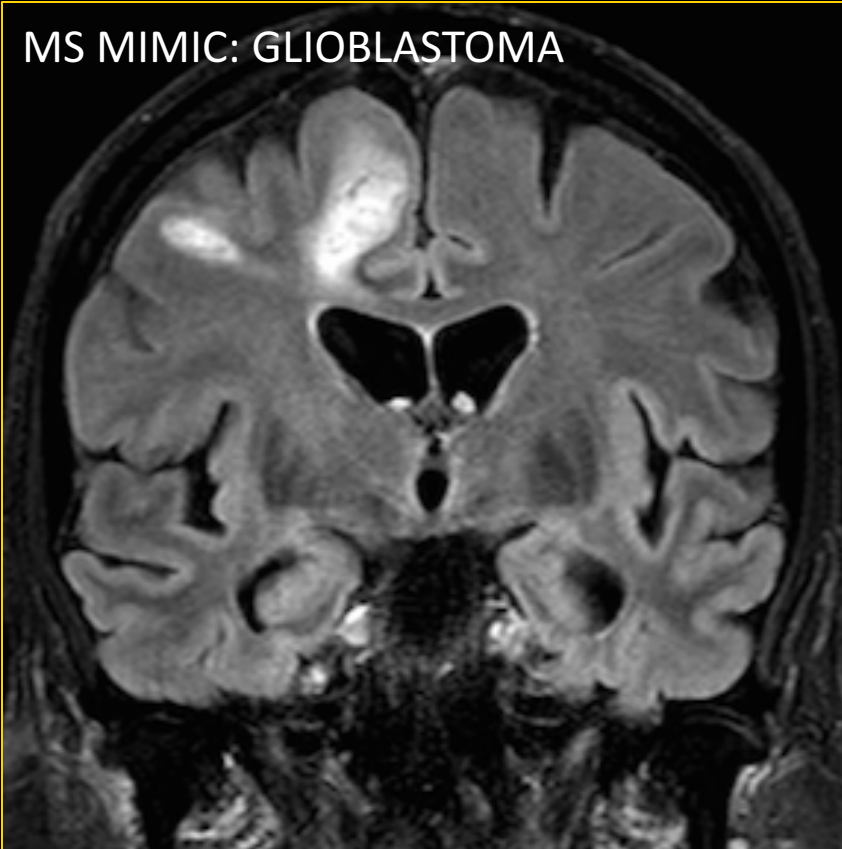
STIR & FAT SAT T1 C+ MR



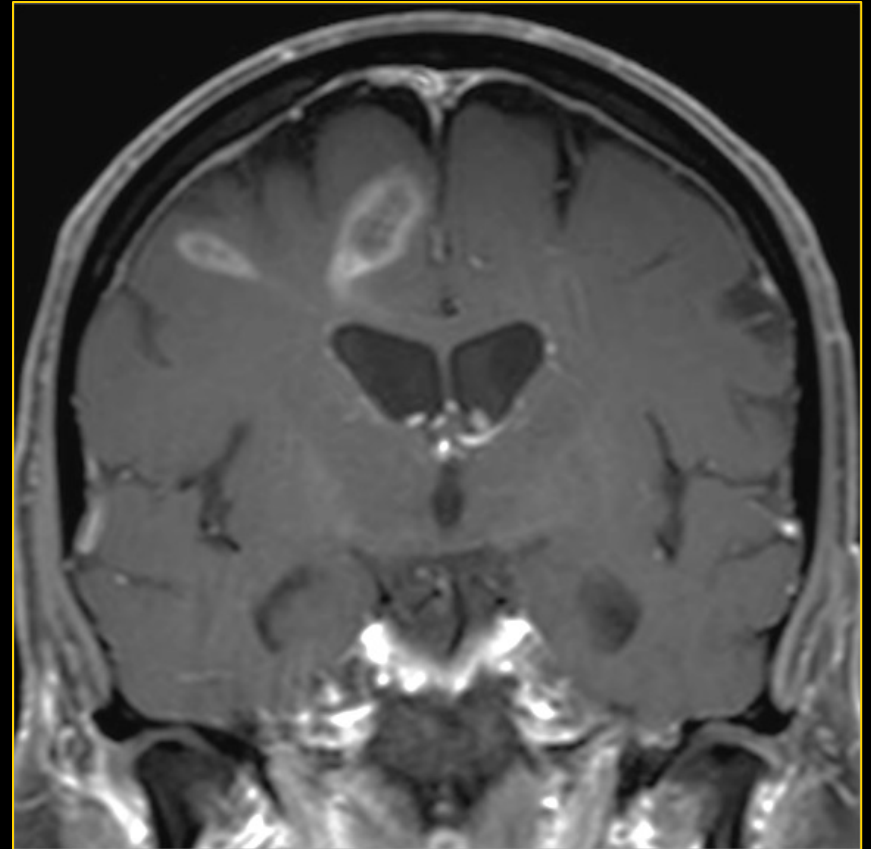
Venous Malformation

FLAIR & T1 C+

MS MIMIC: GLIOBLASTOMA



Complete CSF suppression
GM brighter than WM



CSF dark gray (not black)
GM darker than WM

MONOCULAR VISION LOSS OS

Review basics: T2 fat bright, fluid bright

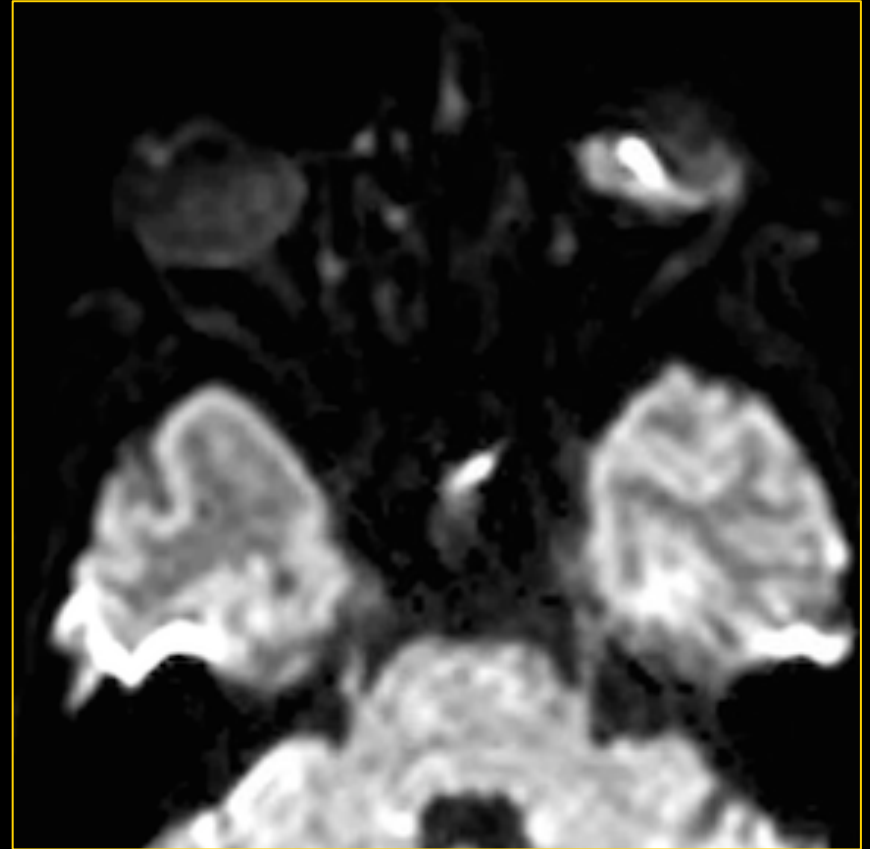
- Review of basics:
 - T2 weighted MRI
 - Fluid bright, fat bright
- Small hypointense signal tissue in posterior left globe



MONOCULAR VISION LOSS:

62 y M immunosuppressed w endophthalmitis

- Imaging sequences are helpful here
- Diffusion weighted imaging performed
- Shows high signal in posterior left globe



DWI is useful in infections
Often added routinely for brain
Not always for orbit/H&N

DWI+ (BRIGHT) LESIONS

aka restricted diffusion

- **Abscess** (Pus - central portion of fluid signal)
- **Epidermoid cysts**
- **Cytotoxic edema in brain**
 - Usually associated with permanent neuronal loss
 - Seen primarily in **acute ischemia**
 - Also uncommonly seen in demyelinating disease
- **Certain neoplasms (with dense cell packing and/or high nuclear:cytoplasm ratio)**
 - **Lymphoma** most classic example
 - Key: No fluid signal on T2 MR – looks like solid tissue (iso- to hypointense relative to brain)

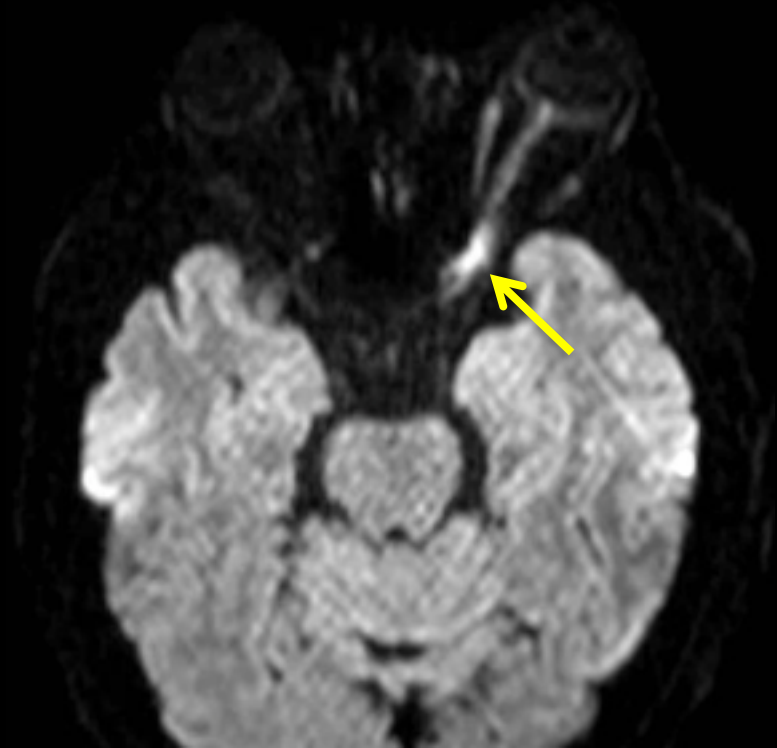
IMAGING TIPS: DWI

- Identifying the DWI sequence
 - Gray brain, black CSF
 - May be labeled “trace” imaging (composite of 3 orthogonal diffusion directions)
 - May be labeled b 1000
- DWI = Composite of diffusion & T2 weighting
 - B0 = T2 weighted component; effectively low res T2
 - Many scanners send interleaved DWI/B0 images
 - The B0 has dark (featureless) brain but white CSF

IMAGING TIPS: DWI

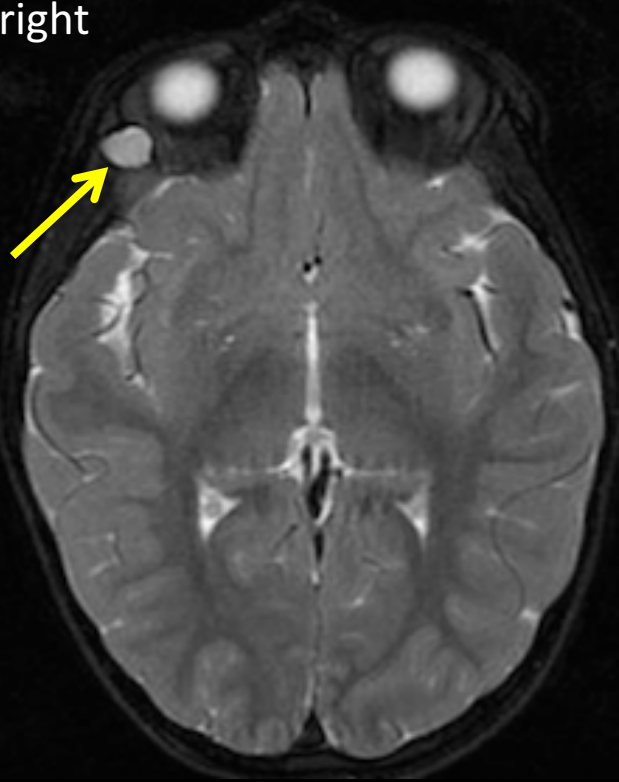
- DWI image: Dark CSF
- Identifying the ADC map
 - Geometric low resolution image
 - Mathematically generated image that subtracts T2 weighting out of the DWI image
 - Should be labeled as such
 - Brain is dark, white CSF

Patient with left optic neuritis

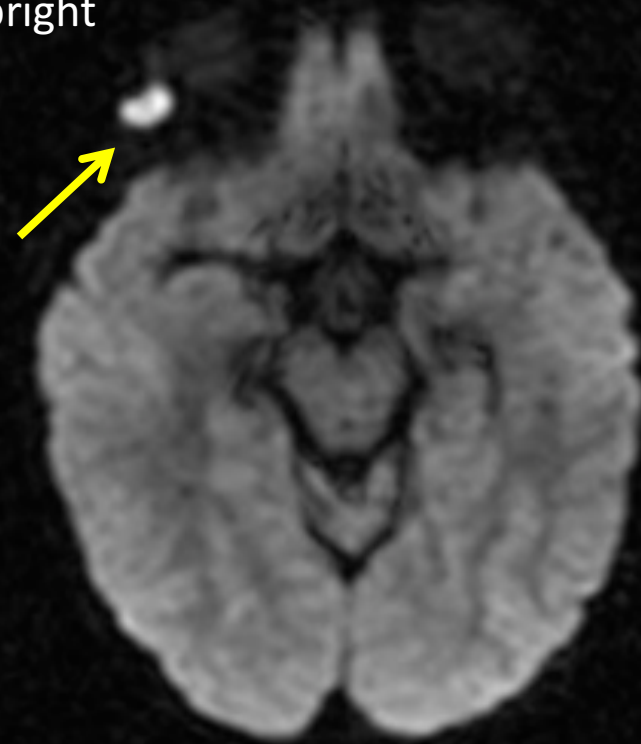


RIGHT ORBITAL MASS IN A CHILD

T2 bright

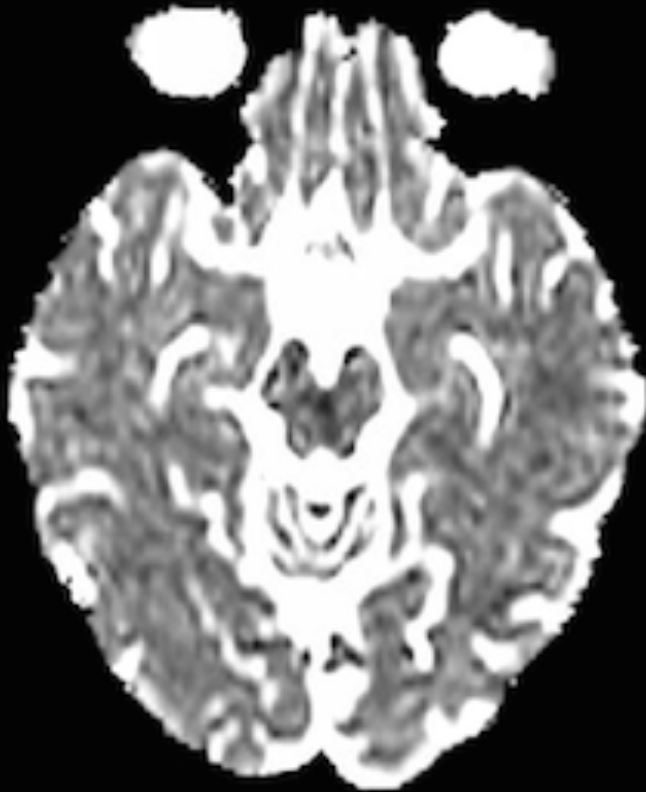


DWI bright



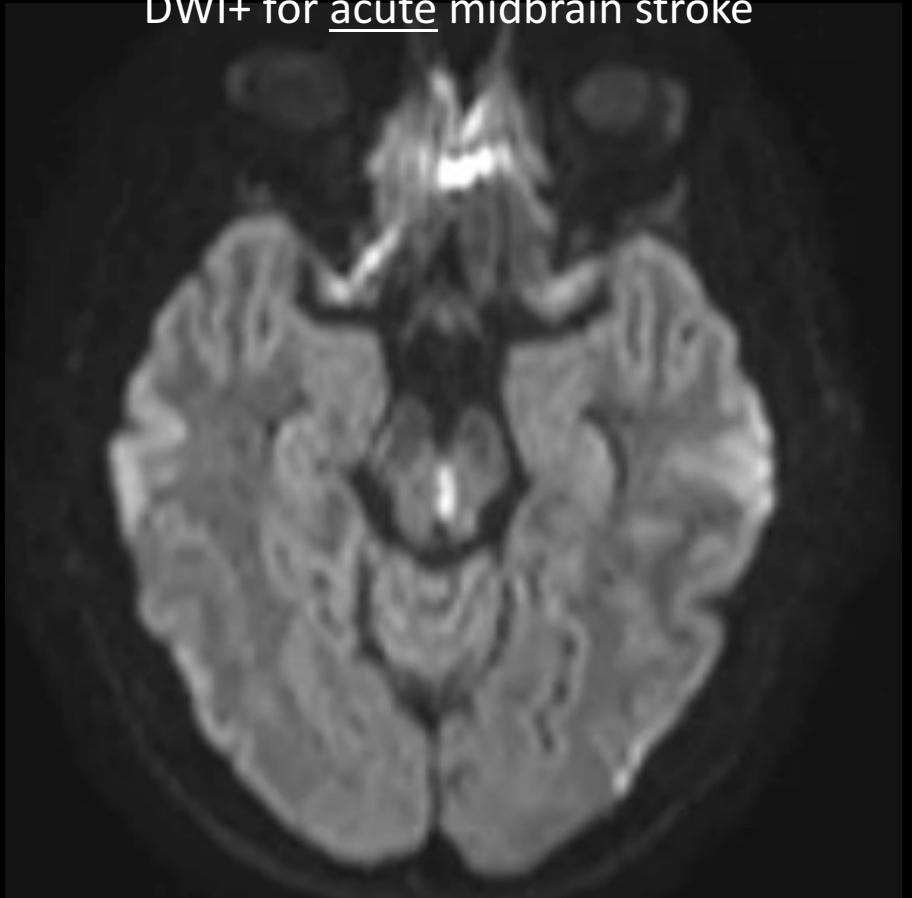
Diagnostic of epidermoid

28 Y F W INTERMITTENT BLURRY VISION; NOW 2 DAYS LINO



ADC: White CSF

DWI+ for acute midbrain stroke

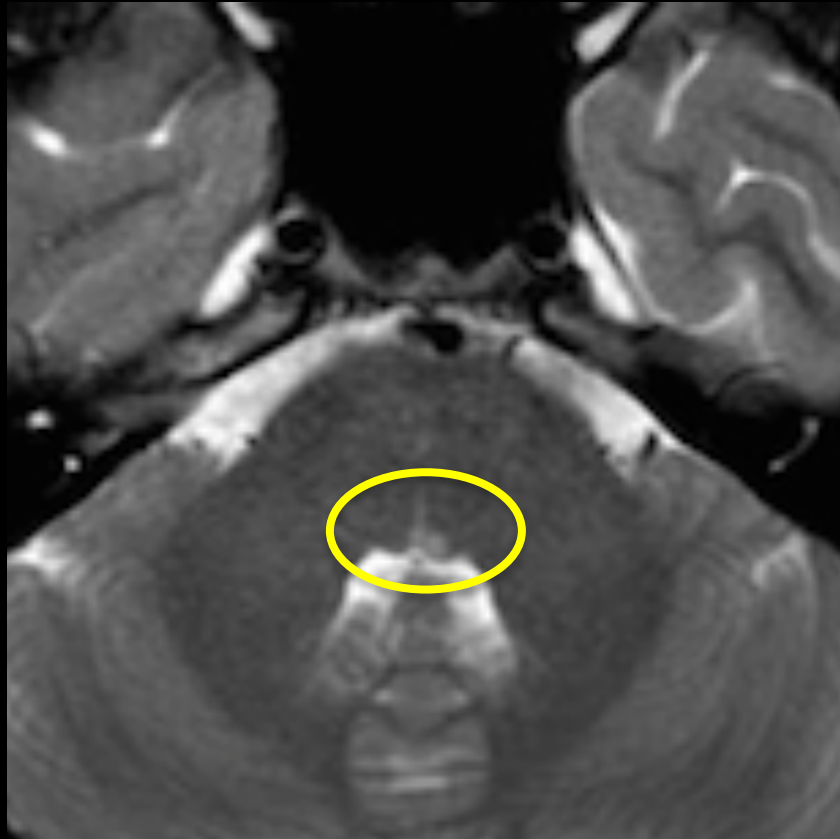


DWI: Dark CSF

IMAGING TIPS ON STROKE

- Easy to miss brainstem stroke if inattentive
- Review **trace image** w DWI – different institutions & vendors vary which sequences are sent to PACS
- Compare to **ADC map** for acuity & technical issues
 - High DWI signal may be due to:
 1. Genuine cytotoxic edema in stroke
 2. Other lesions with restriction (abscess, epidermoid)
 3. Compare to conventional sequences
 - Concurrent ADC hyperintensity suggests
 1. Subacute stroke
 2. “T2 shine through”
- The smaller the stroke, the quicker the change to ADC hyperintensity (days not weeks-months)

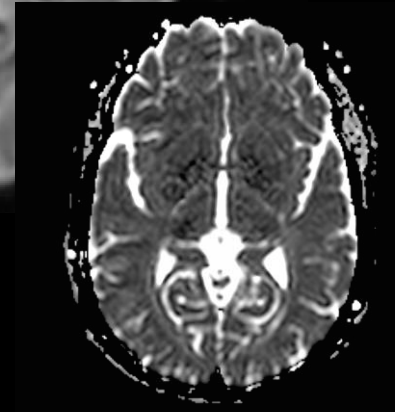
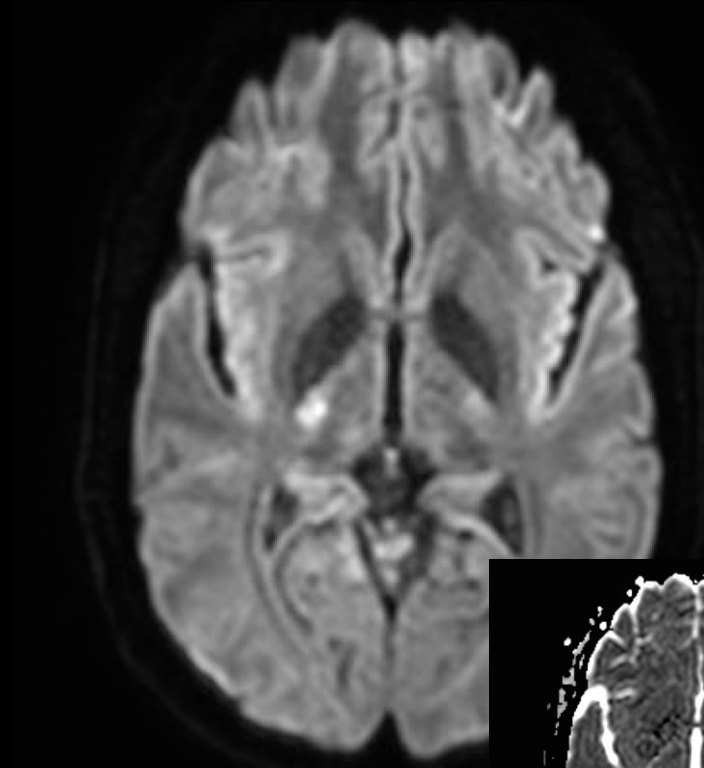
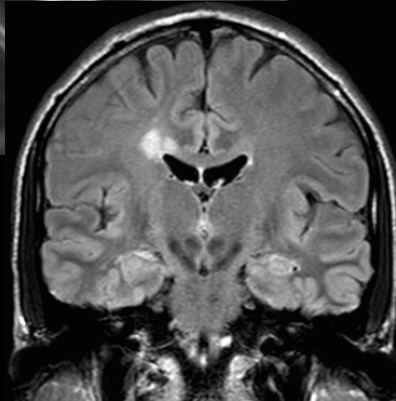
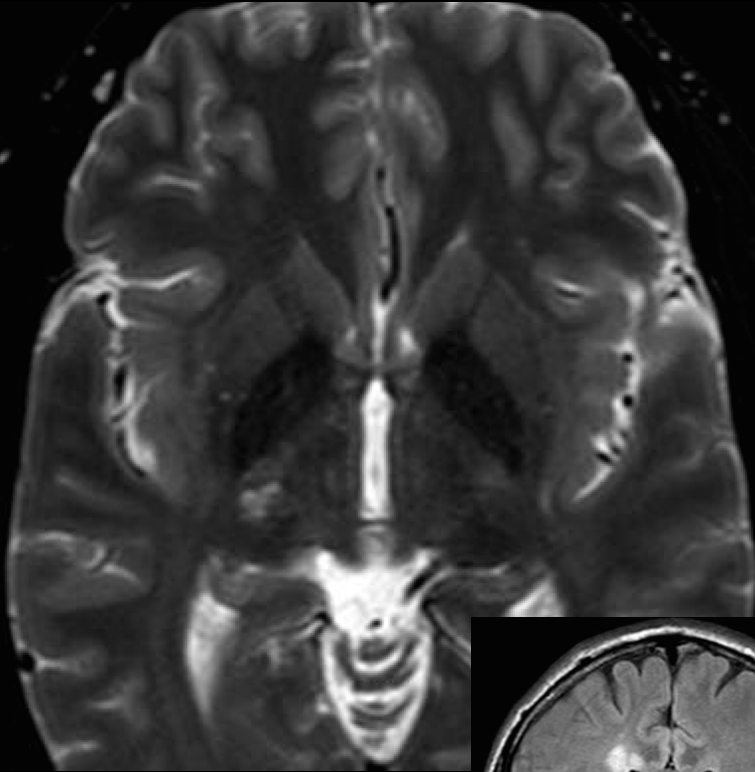
15 Y F WITH DIPLOPIA



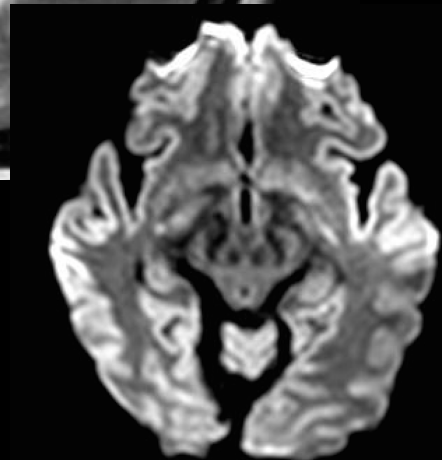
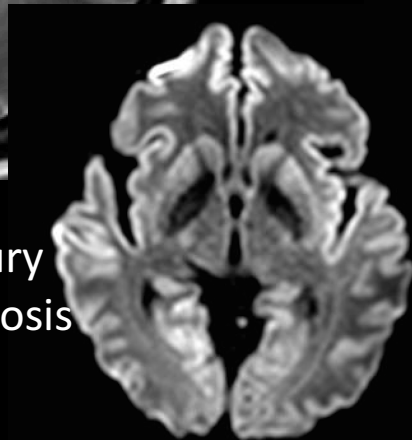
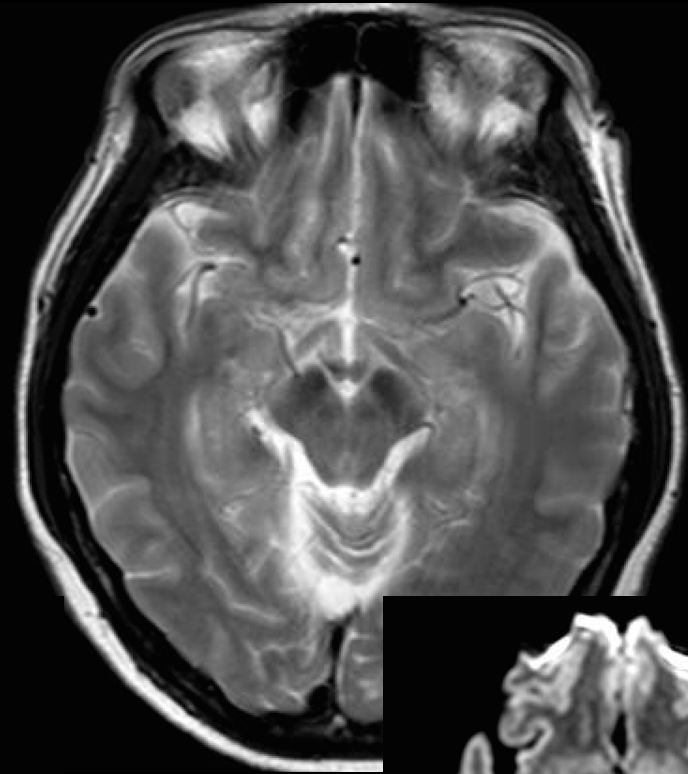
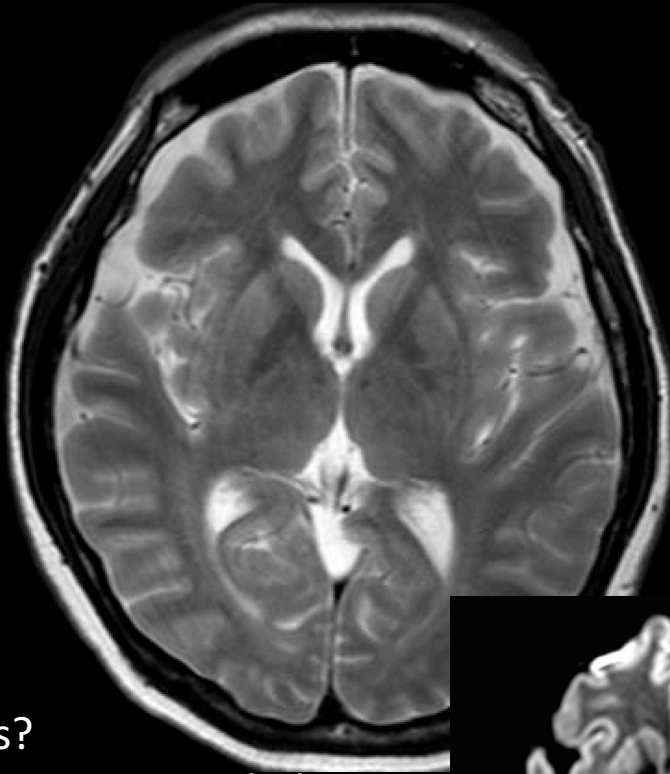
Sudden onset diplopia on awakening 2 weeks ago
Diplopia in primary gaze, nearly eliminated in right gaze
Left CN6 palsy on exam

ACUTE L VISUAL FIELD CUT IN 27 Y M

DWI BRIGHT DEMYELINATION



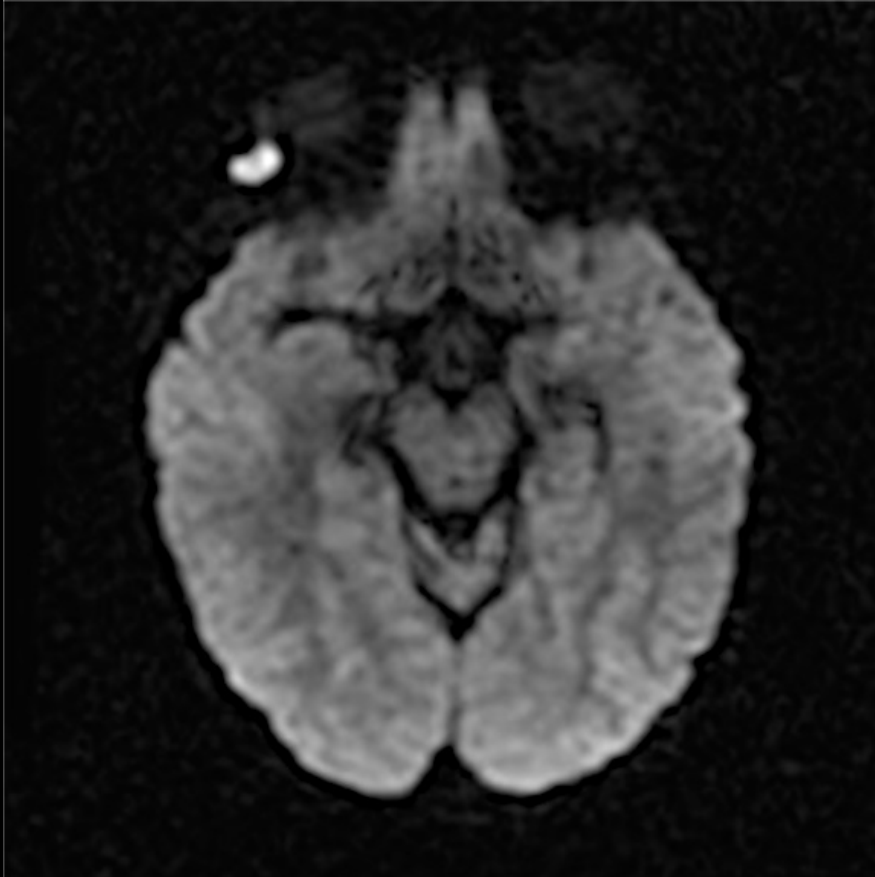
L FIELD CUT & ATAXIA IN 53 Y F



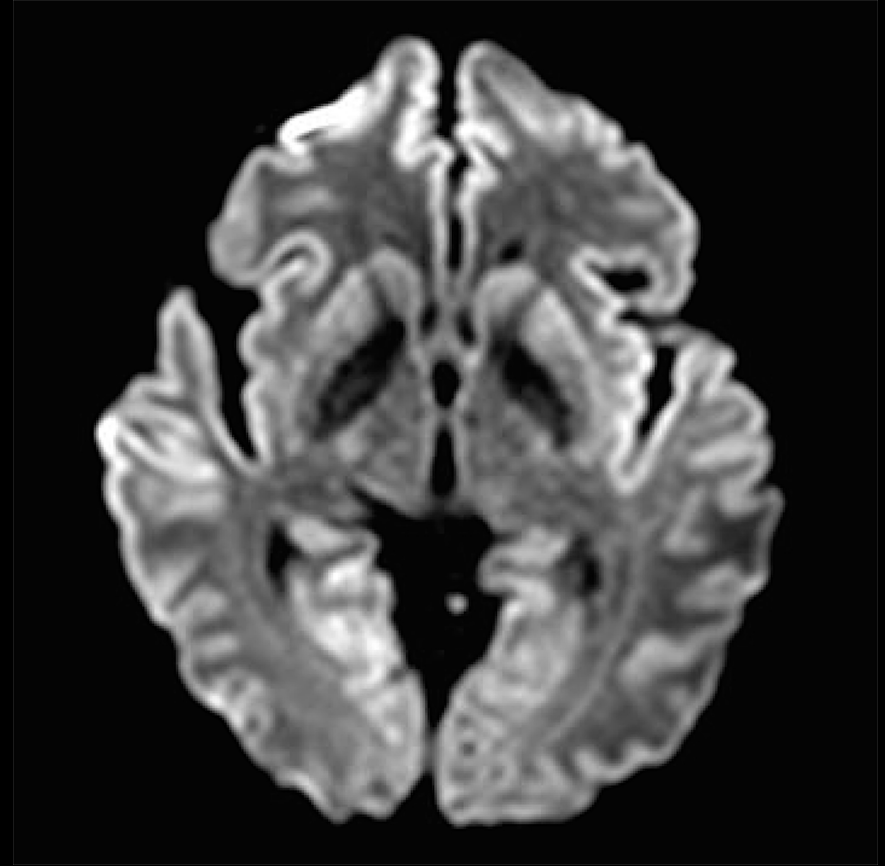
Diagnosis?

1. Rasmussen encephalitis
2. Diffuse hypoxic ischemic injury
3. Leptomeningeal carcinomatosis
4. Creutzfeld-Jacob disease
5. Imaging is normal

AUTOPSY CONFIRMED CJD



Refresh your memory of normal DWI
With our orbit epidermoid case



Bright caudate heads & ant putamen bil
Accentuated cortex, esp lining sylvian fiss

WINDOW ON THE WORLD:

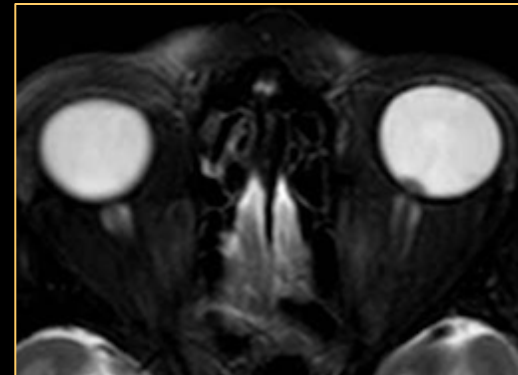
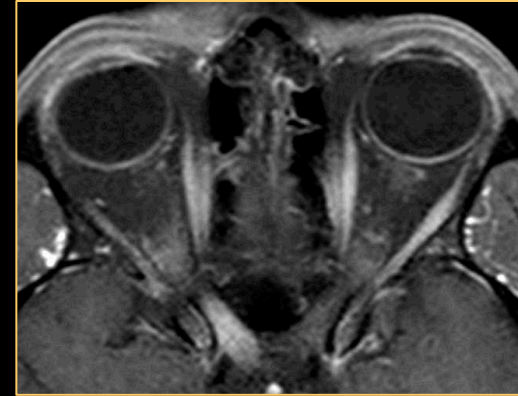
The view from inside



Windy Point, Central Oregon Cascades

ACUTE CN II PATHOLOGY

- **Optic neuritis:**
 - Acute optic nerve inflammation
 - Subacute vision loss (color > acuity)
 - Progression ~ 7-10 days
- **Optic papillitis (Ant optic neuritis)**
 - Optic nerve head (disc) is swollen
 - Implies unilateral eye disease
- **Papilledema:**
 - Bilateral disc edema, implies ↑ ICP
- **Pseudoapilledema***



SUBACUTE-CHRONIC CN II PATHOLOGY

- **Optic neuropathy**

- Inflammatory
- Ischemic
- Mitochondrial
- Nutritional
- Toxic
- Hereditary
- Infiltrative & tumor
- Compressive *

***Role
of
imaging?***


Intrinsic

- *May confirm diagnosis*
- *Define extent*
- *Exclude extrinsic compression*

Extrinsic

* Trauma, neoplastic, inflammatory, etc.

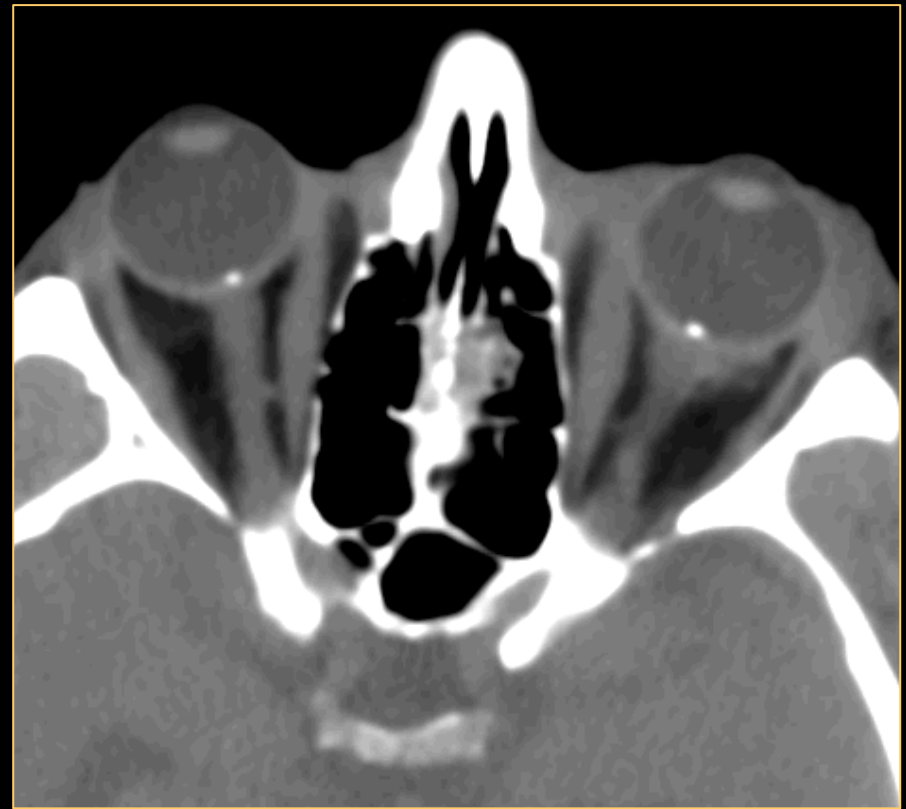
IMAGING THE OPTIC NERVE

- Choice of modality depends on indication
- “Optic neuropathy” – in part depends on clinical suspicion
 - NECT orbits for Graves’ orbitopathy
 - Enhanced (CT/MR) scan for suspected neoplasm
 - Systemic inflammatory disease
 -  ✓ Extraorbital soft tissues! (may mimic NHL)
- Brain imaging (MRI) may be more relevant (e.g. MS eval in optic neuritis or SOD)

PSEUDOPAPILLEDEMA

Optic Disc Drusen

- Pseudopapilledema
 - Optic disc elevation, not true disc edema
 - Usually due to congenital abnormalities
- Affects ~ 1% population
- 75% develop peripheral visual field defects late in life
- NECT demonstrates well d/t calcifications



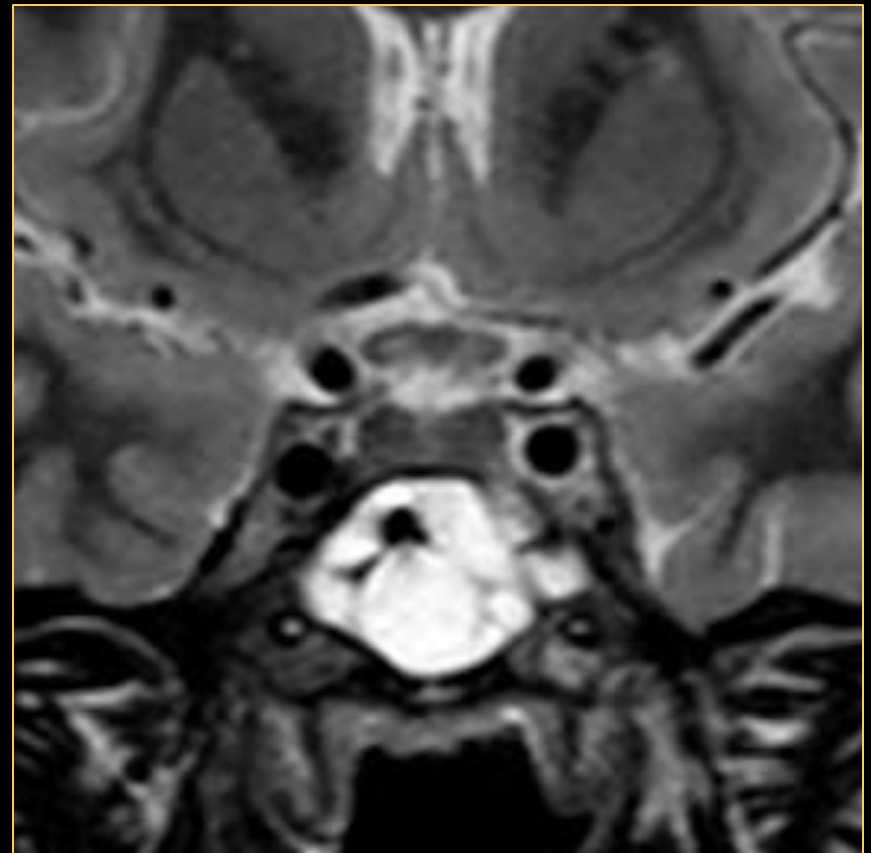
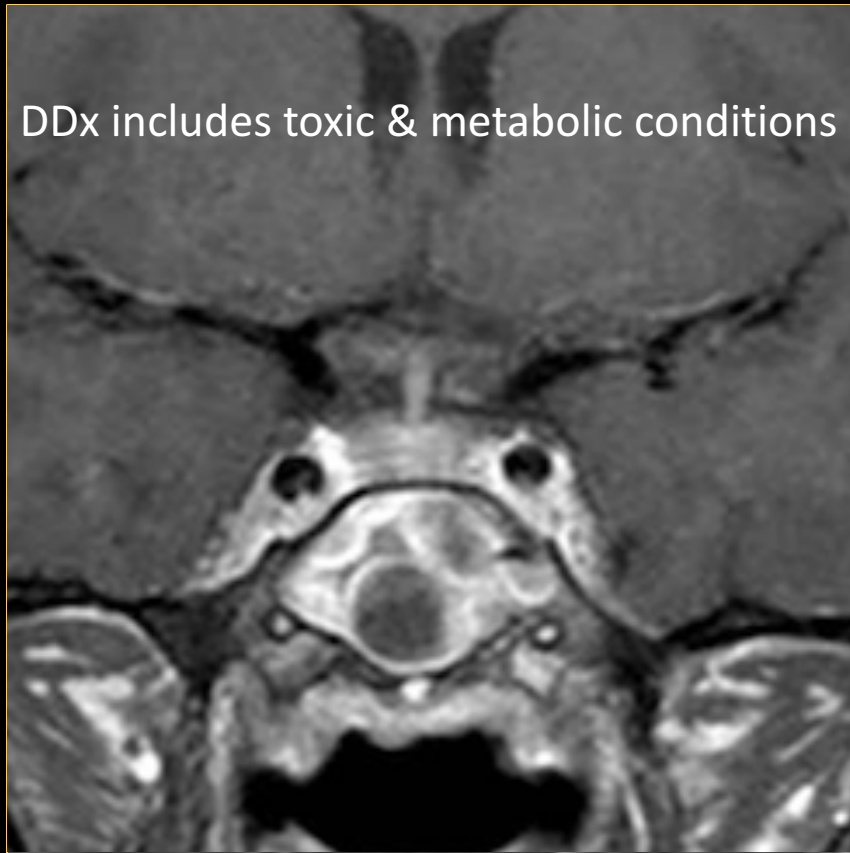
Tuğcu B, Özdemir H. Imaging Methods in the Diagnosis of Optic Disc Drusen. Turk J Ophthalmol. 2016 Oct;46(5):232-236.

SOME TIMES TO CONSIDER MRI

- High soft tissue contrast required
- Optic nerve abnormality
 - Intrinsic vs extrinsic
- Demyelinating disease
- Ischemia
- Spinal cord evaluation/myelopathy

PSEUDOPAPILLEDEMA: 18 Y M

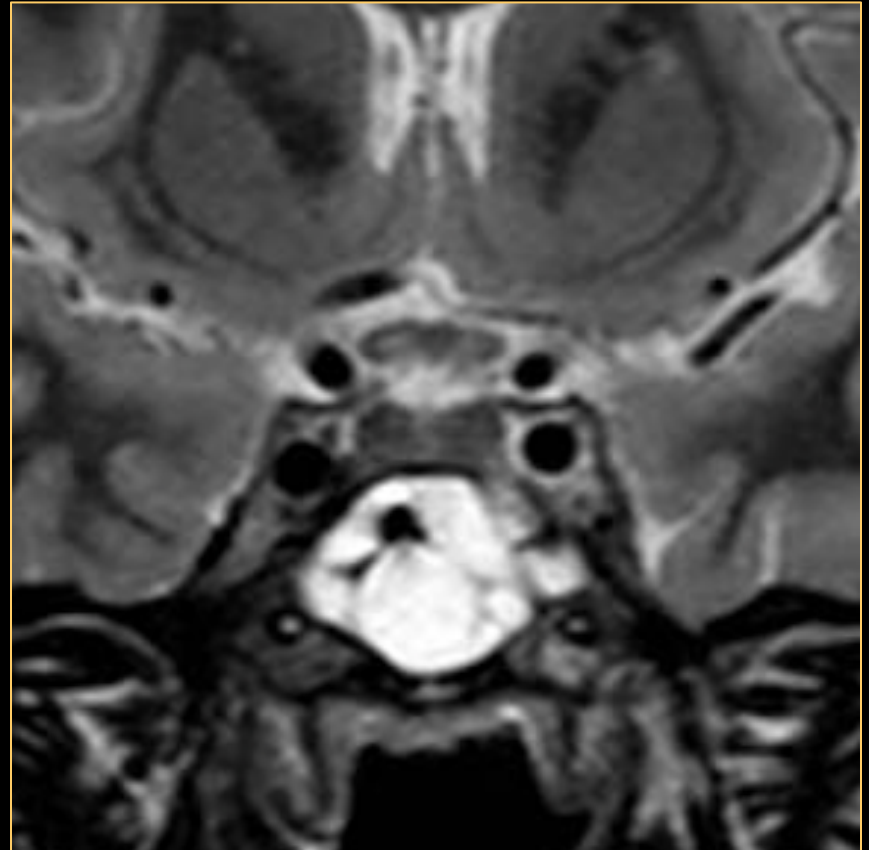
Probable Leber's hereditary optic neuropathy



Progressive central dense vision loss in both eyes over one month, L then R

MRI FINDINGS IN LHON

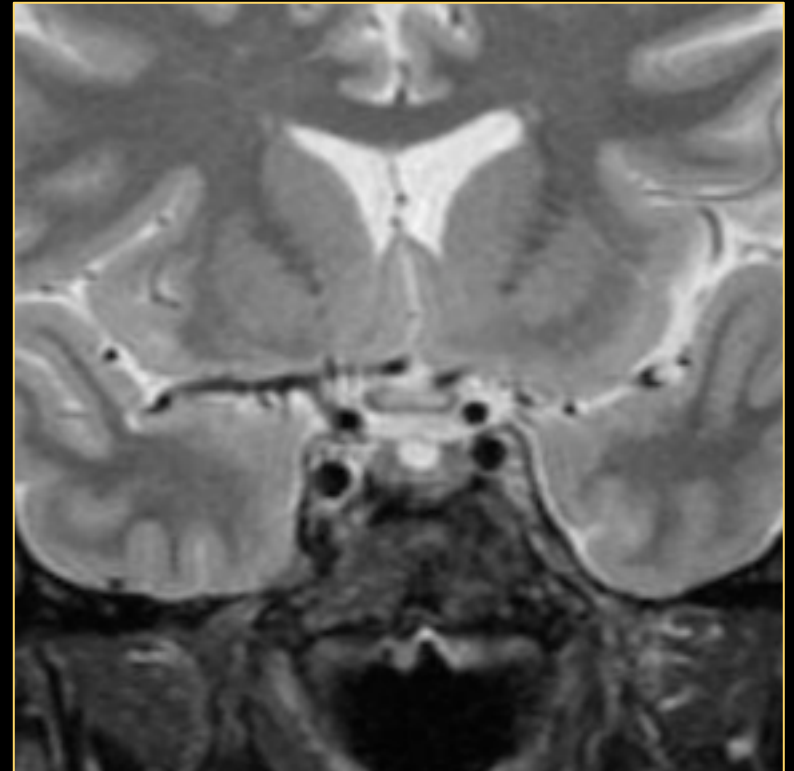
- T2 hyperintensity affecting posterior CN II &/or chiasm
- Central pattern mentioned elsewhere
 - “Oreo sign”
- May enhance
- May have (mild) mass effect
- Assoc’d WM lesions
 - **Not** typical of MS



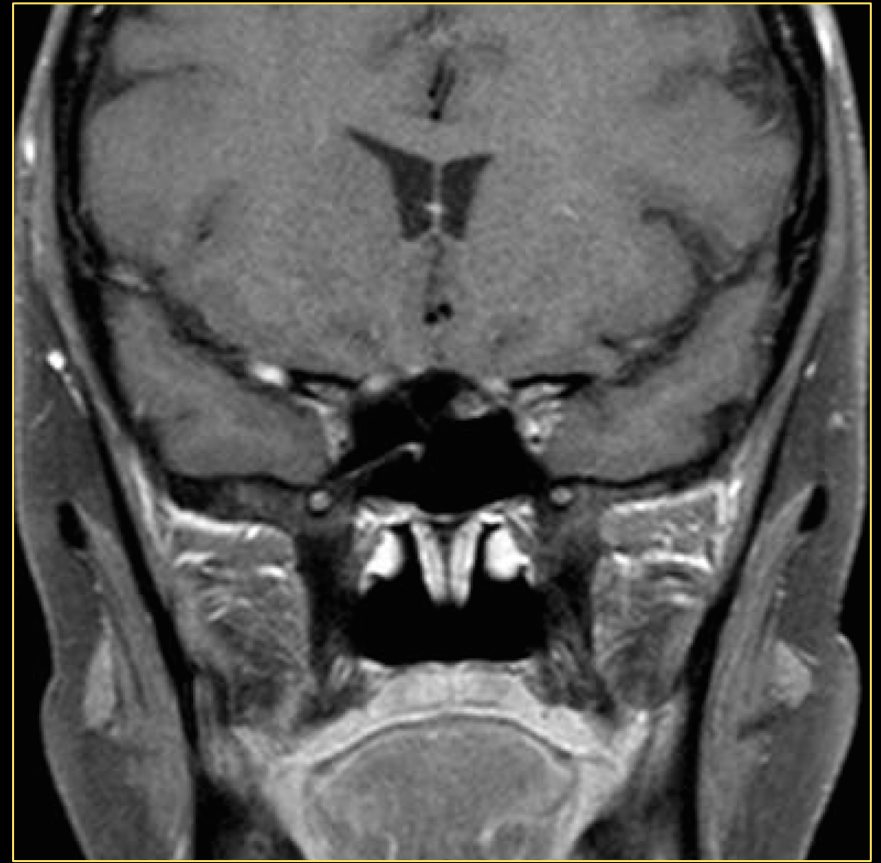
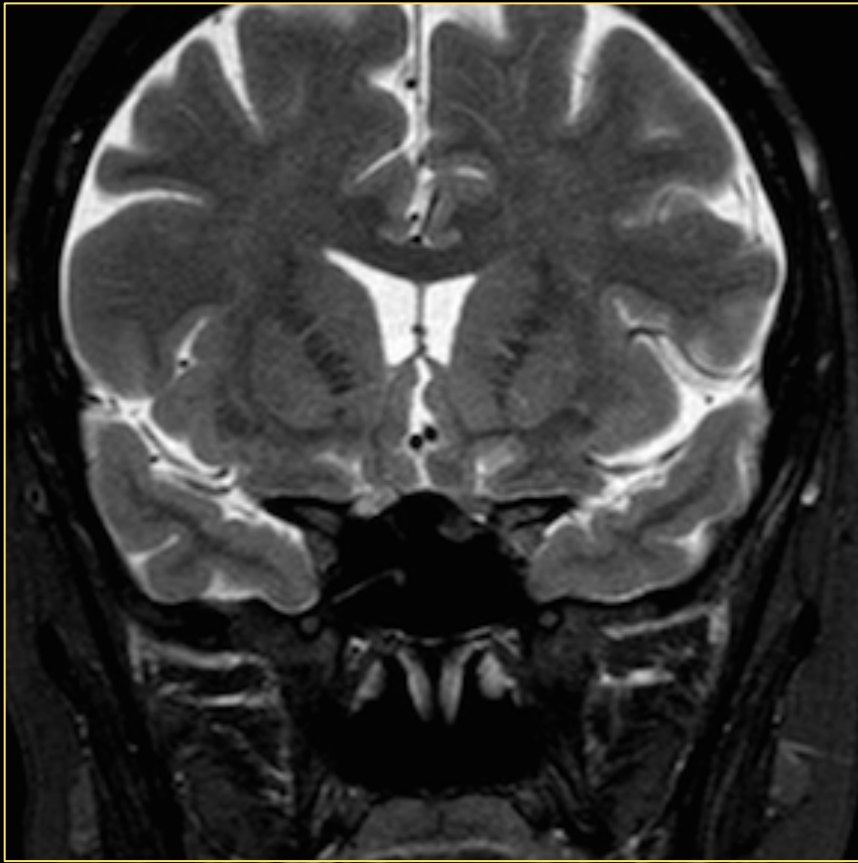
Blanc C et al. MRI of the Optic Nerves and Chiasm in Patients With Leber Hereditary Optic Neuropathy. J Neuroophthalmol. 2018 Jan 3.

39 Y F: PROGRESSIVE CENTRAL SCOTOMAS

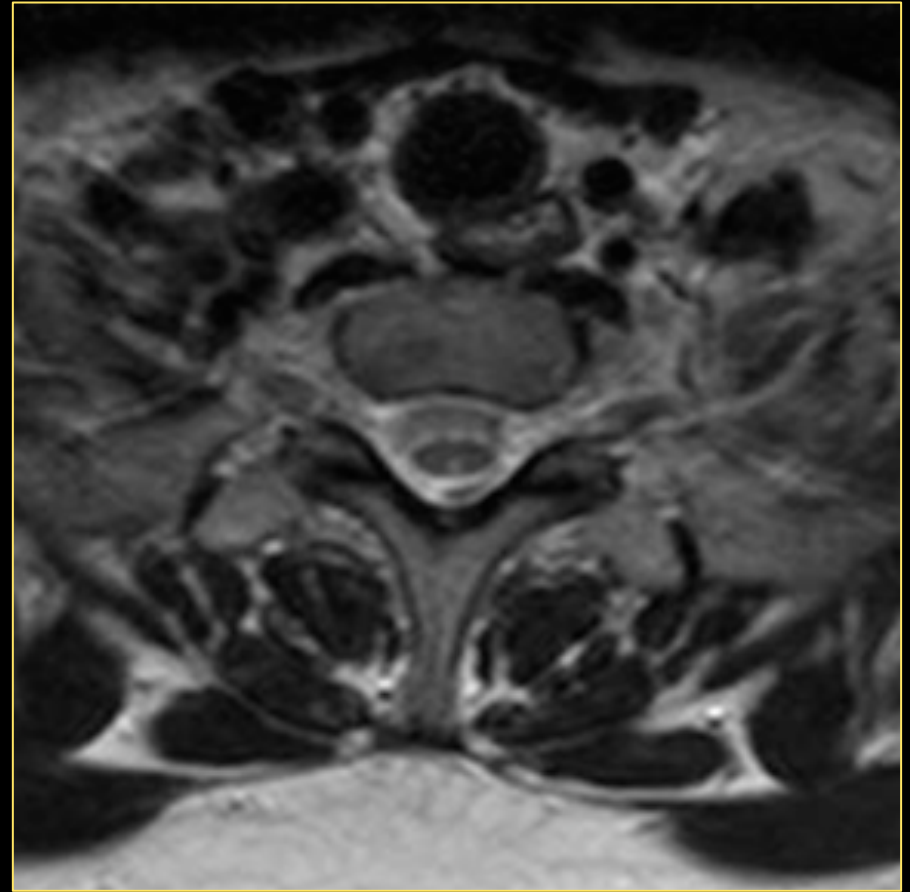
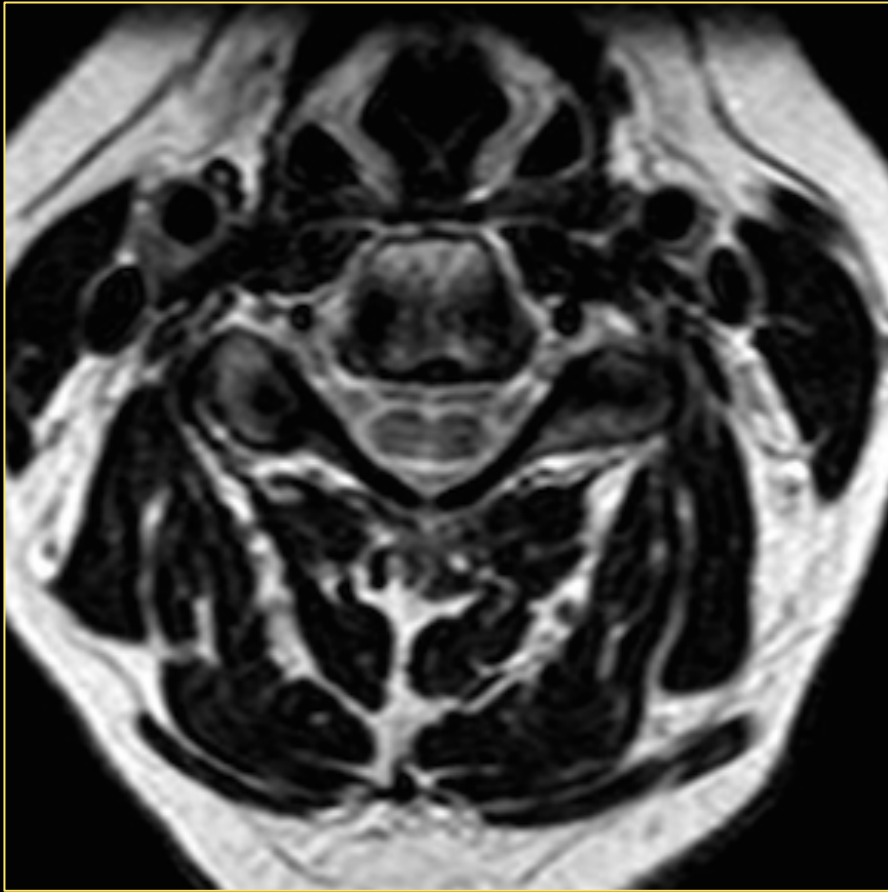
- Tobacco & etOH use
- Recent restrictive diet over 6 months
 - ↓ serum Vit B12 & folate
- More slowly progressive course of vision loss
- Coronal T2 MR:
 - Central hyperintensity involving the posterior CN II &/or optic chiasm



BIL VISION LOSS, HYPERREFLEXIA & NEUROPATHY



SAME PATIENT: AXIAL T2 CSP-TSP

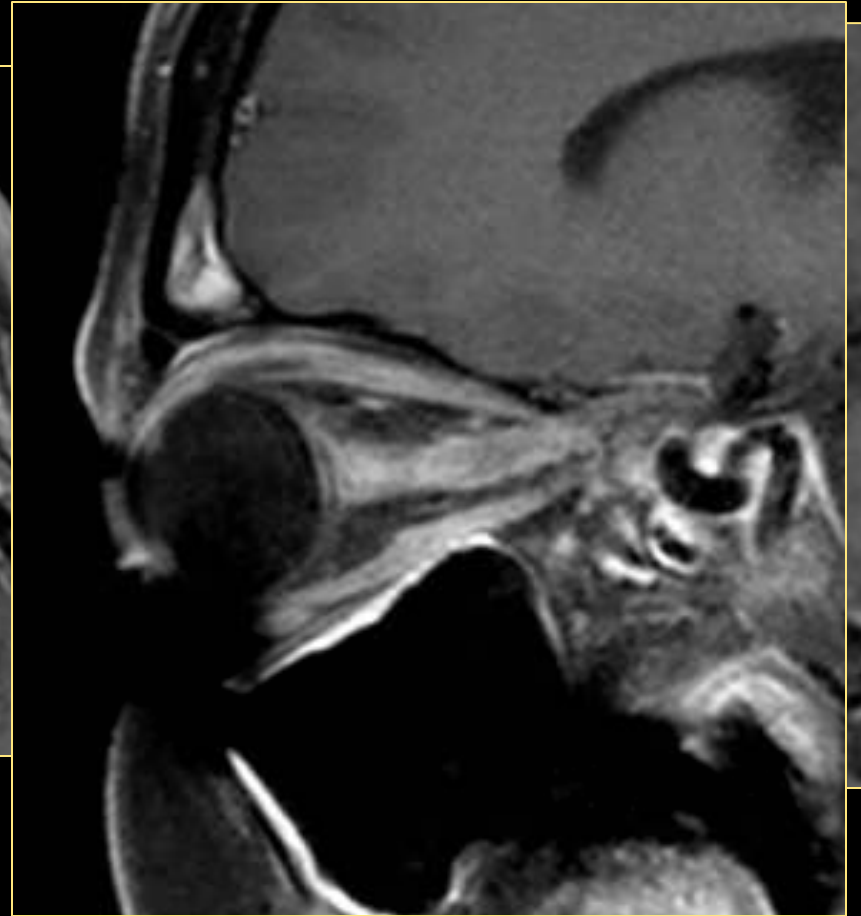
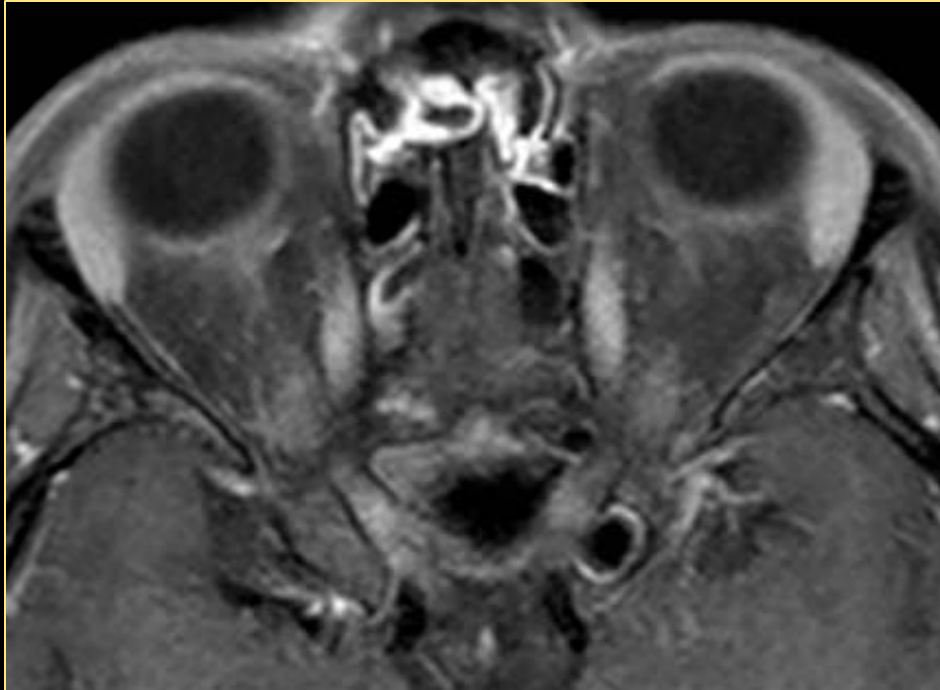


SUBACUTE COMBINED DEGENERATION:

Vitamin B12 Deficiency

- Initially affects spinal cord:
 - Posterior columns T2 hyperintense
- Later affects brain (aka “combined”), CN II
- Patients at risk:
 - Pernicious anemia
 - Crohn’s
 - Disorders with GI malabsorption
 - Vegan/vegetarian diets
- Imaging findings of copper deficiency similar

ACUTE RIGHT SIDED VISION LOSS



Optic Neuritis

NEUROMYELITIS OPTICA

- Inflammatory demyelination
 - Positive NMO antibody (AqP-4)
 - Treatment different from MS
- Typical involvement:
 - Optic neuritis
 - * Spinal demyelination, **longer segment** than MS
 - * **Less brain** involvement
 - May be more cavitory later on

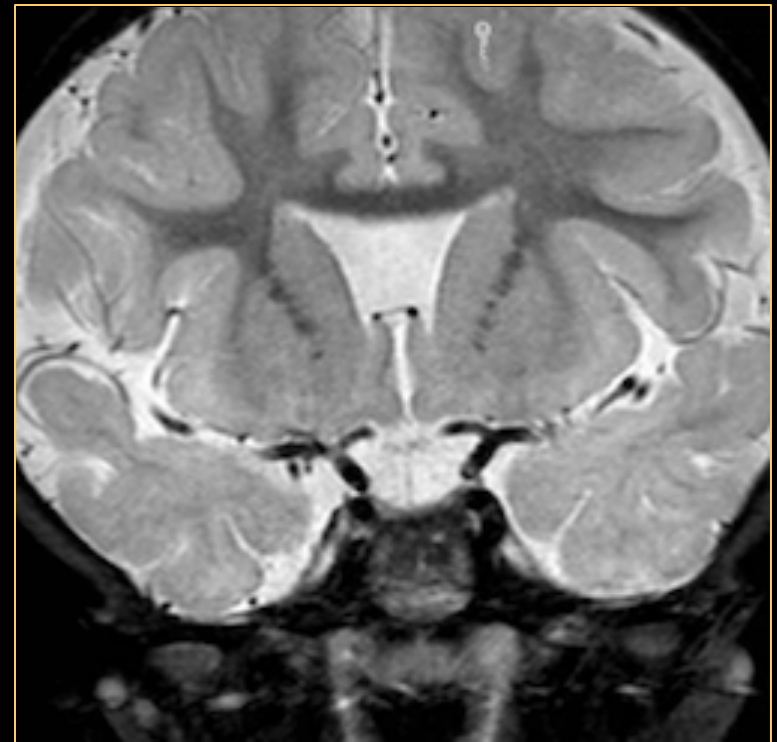
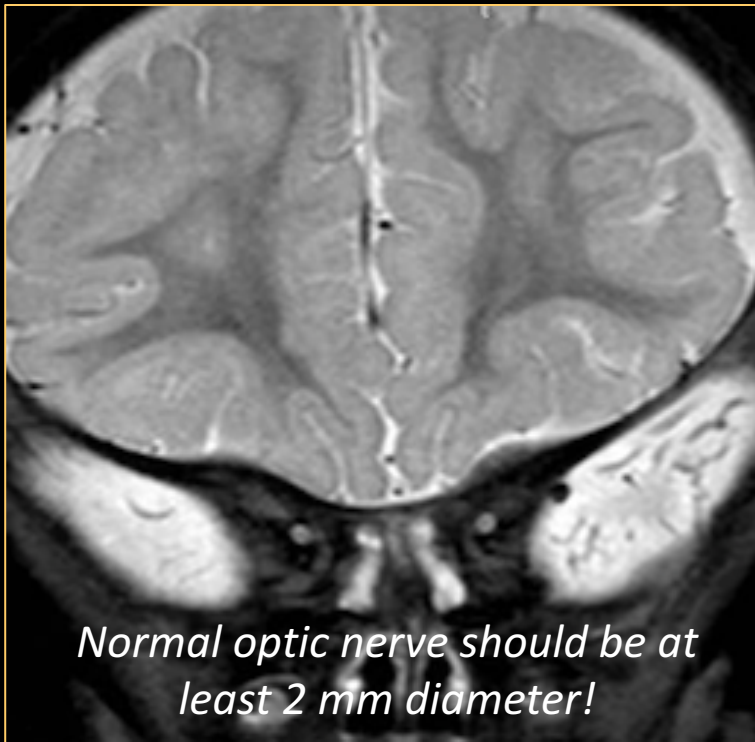


Spinal cord involvement

VISUAL PATHWAY: CONGENITAL

Septo-Optic Dysplasia

High resolution coronal T2-weighted MR



Bilateral optic nerve hypoplasia

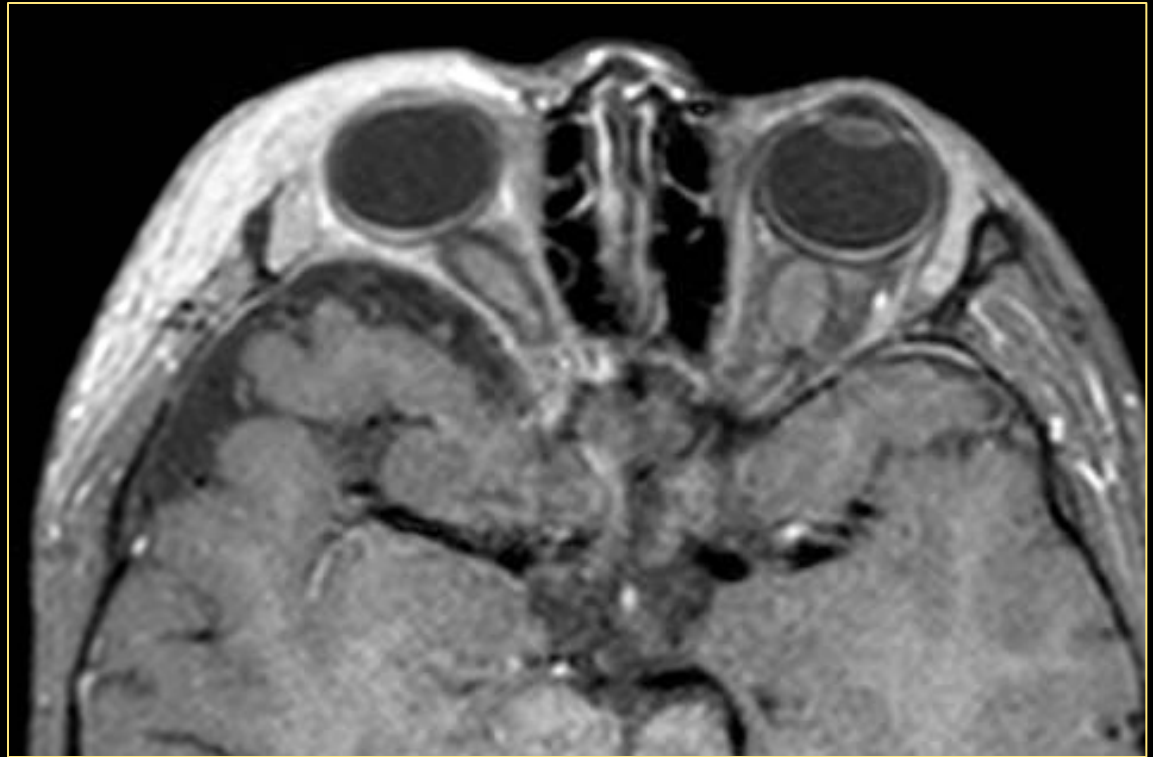
Absent septum pellucidum, pituitary hypoplasia

✓ for perisylvian cortical dysplasias

INTRINSIC/INFILTRATIVE PATHOLOGY:

Visual pathway neoplasms

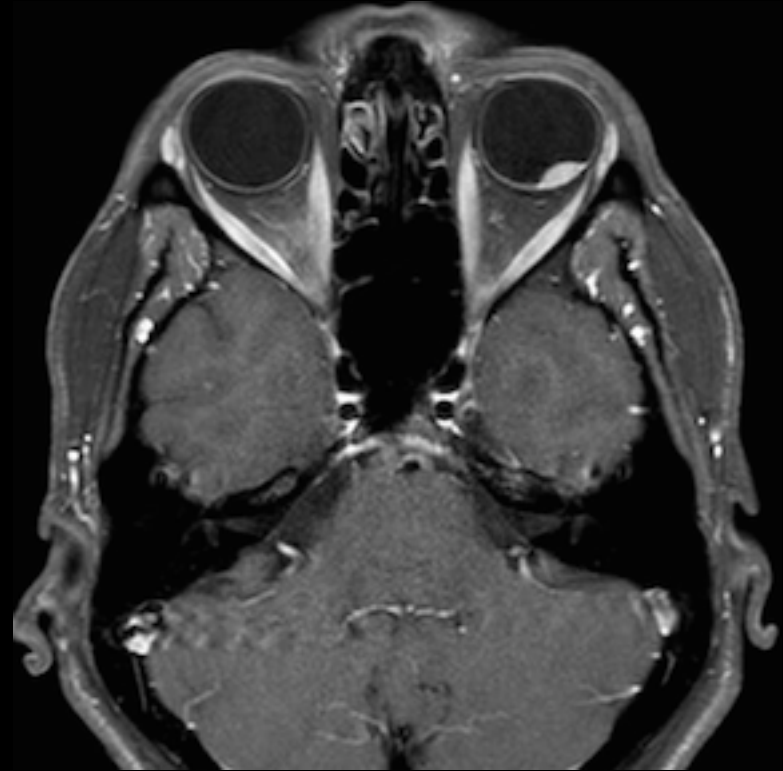
- Bilateral optic nerve gliomas
- Extraorbital findings:
 - Sphenoid wing dysplasia
 - Plexiform NF eyelid



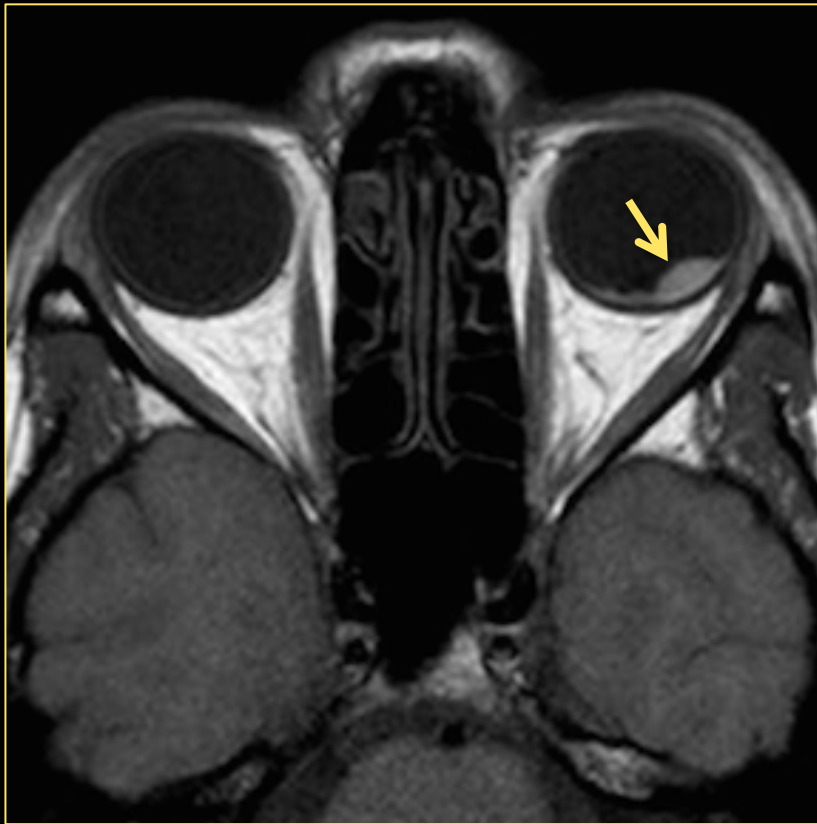
Neurofibromatosis type I

OCULAR MASSES IN ADULTS

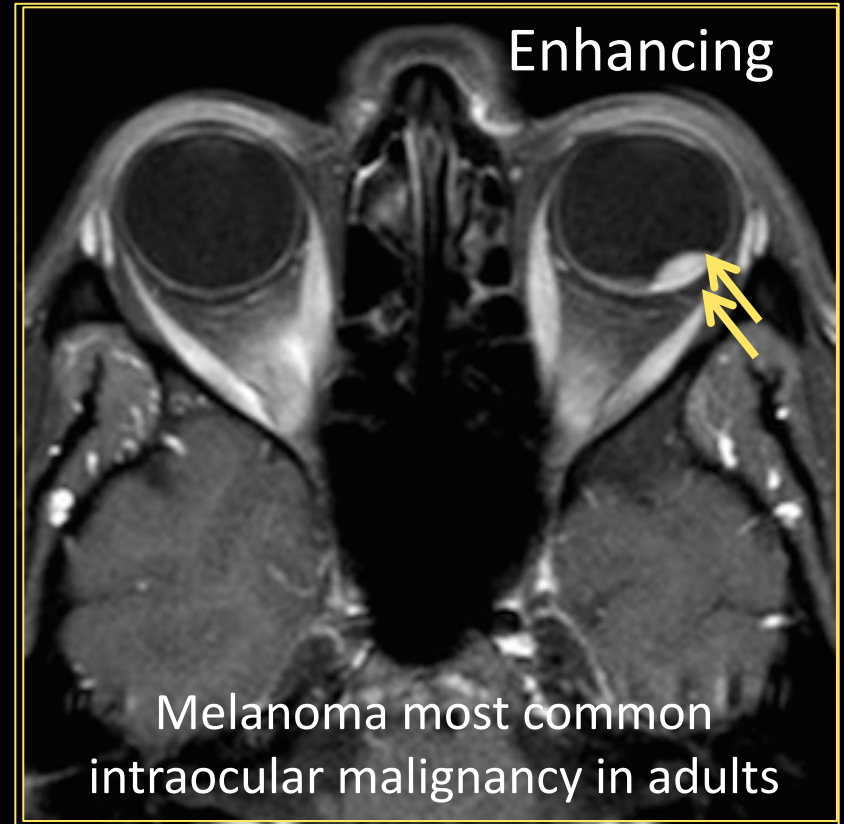
- Uncommon
- Usually small, present early due to vision changes
- Evaluated by B mode US & OCT
- MRI helpful to assess extent of disease & look for other lesions
- Most common adult mass is primary choroidal melanoma
- Lymphomas (may be primary)
- Metastatic disease can occur



44 Y F MIGRAINEUR WITH R UPPER QUADRANTANOPSIA OS + RAPD



Intrinsic ↑ T1 signal

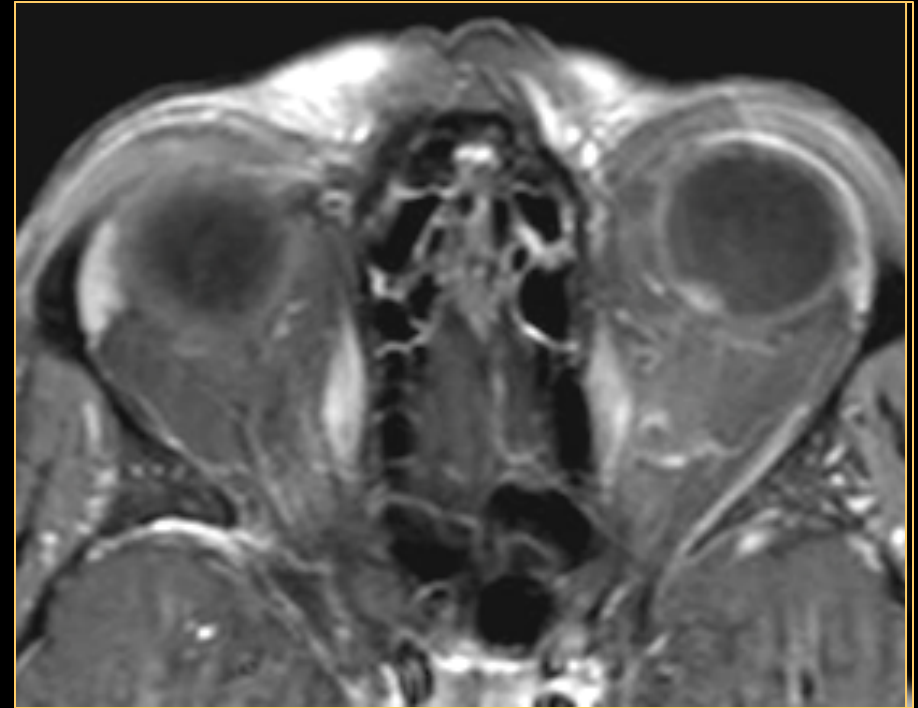
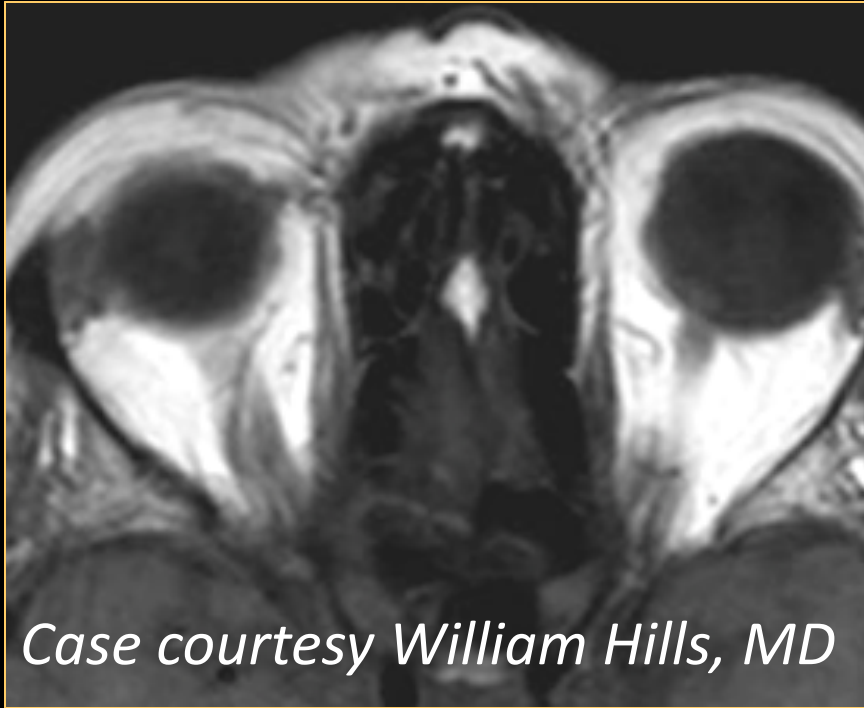


T2 hypointense

Working Dx: Intraocular Melanoma

OPTIC "PAPILLITIS"

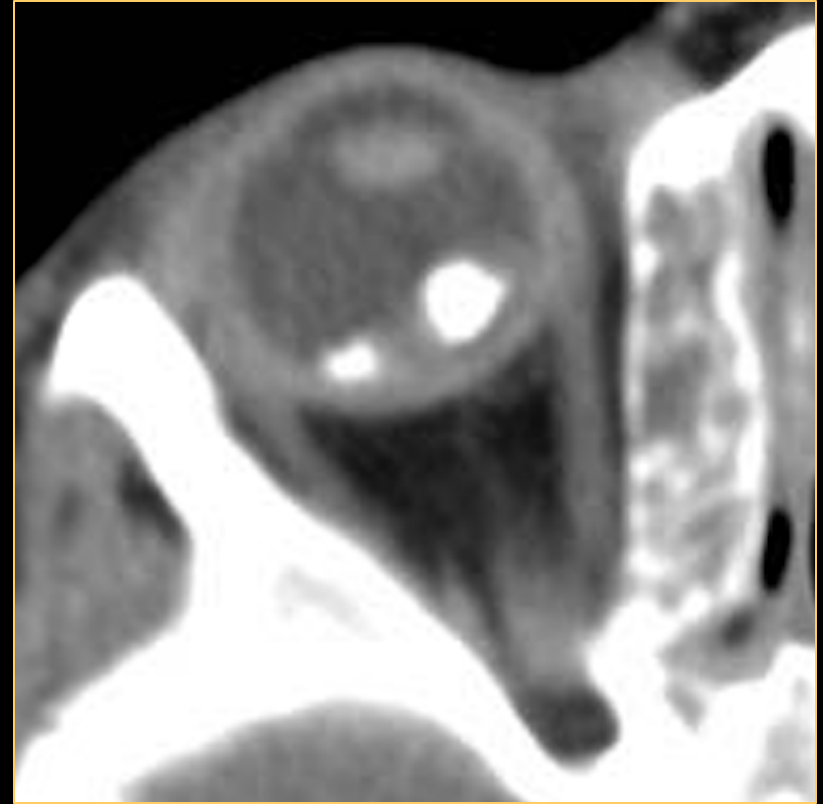
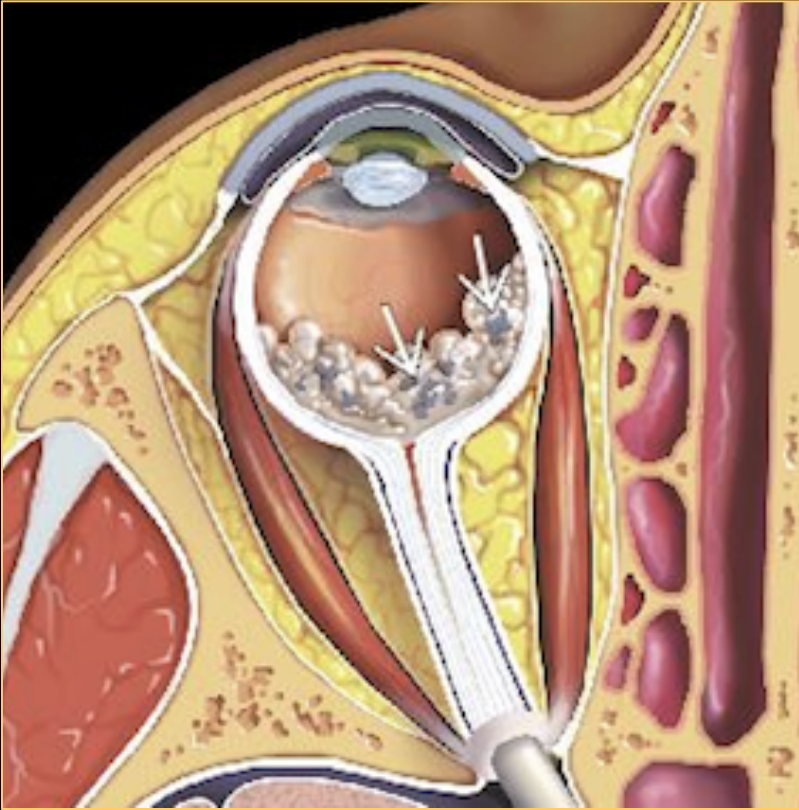
Primary ocular lymphoma



75 y F with unilateral disc edema OS

Visual acuity 20/25

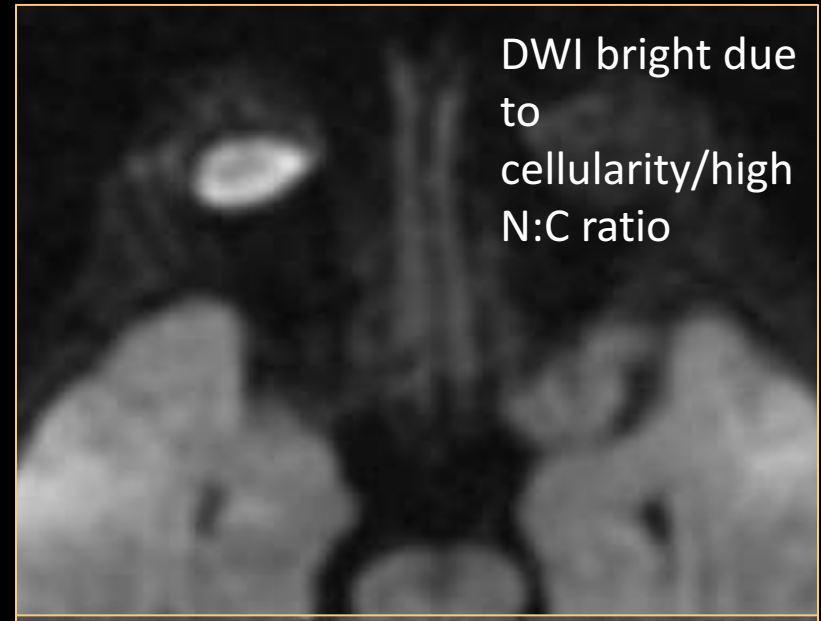
RETINOBLASTOMA



Most common cause of ocular mass in children
Calcifications very common (>90%): CT

PEDIATRIC VISION LOSS

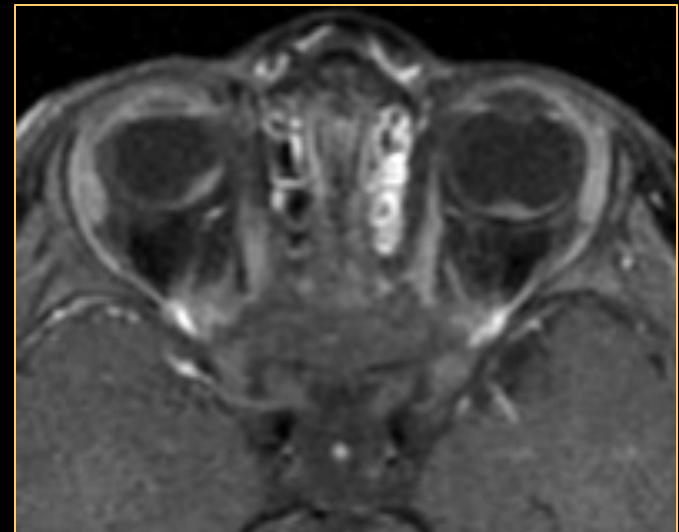
Retinoblastoma



12 mo F, R eye looks “yellow & turns out” x months
Ultrasound showed mass with vitreous involvement
> ½ globe filled with tumor

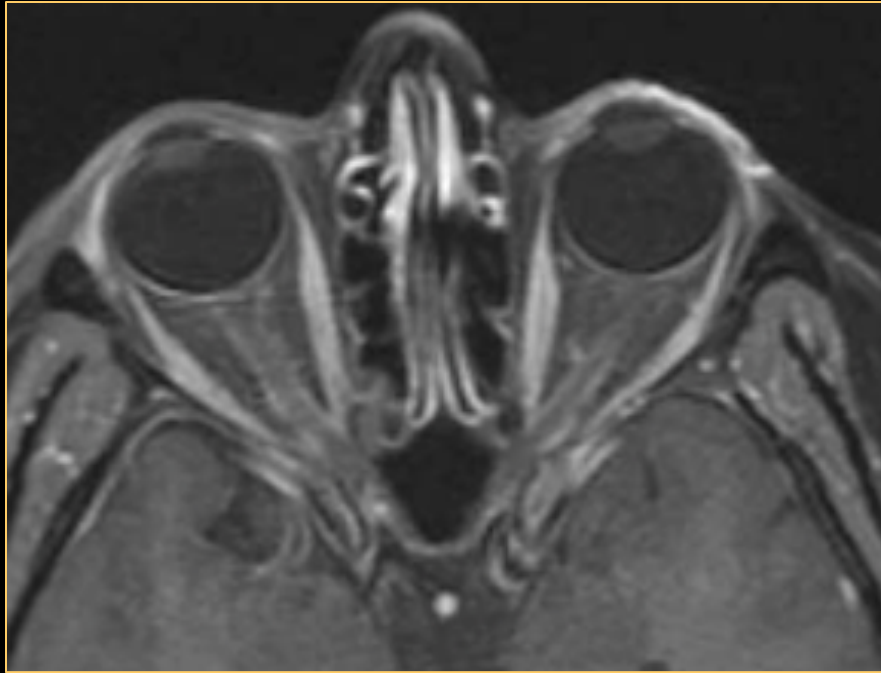
IMAGING CHECKLIST: RETINOBLASTOMA

- Extraocular disease upstages
 - ✓ Optic nerve, orbit, chiasm
 - ✓ Bilateral disease
- Multifocal disease common due to germline mutations
 - Uni-lateral (60%)
 - Bi-lateral (40%)
 - Tri- or quadri- lateral (rare)
 - ✓ Pineal, pituitary



OPTIC NERVE SHEATH DISORDERS:

Pachy- and Lepto-meningeal Diseases



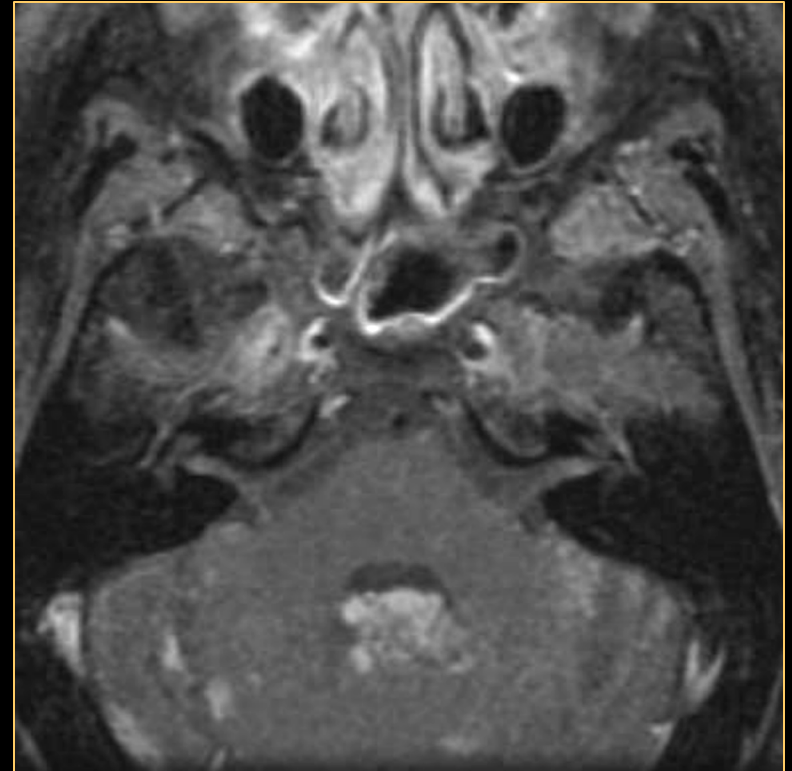
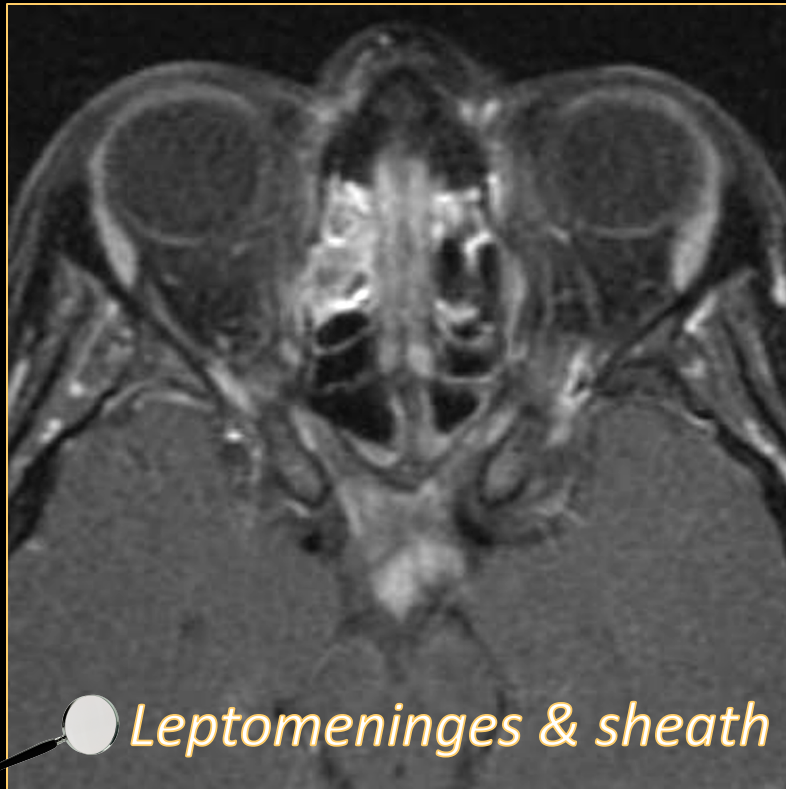
- 41 y F, painful eye movements
- Steroid-responsive
- **Pseudotumor perineuritis (?)**



- Slowly progressive vision loss
- Treated esthesioNB yrs prior
- **Meningioma (RT-induced)**

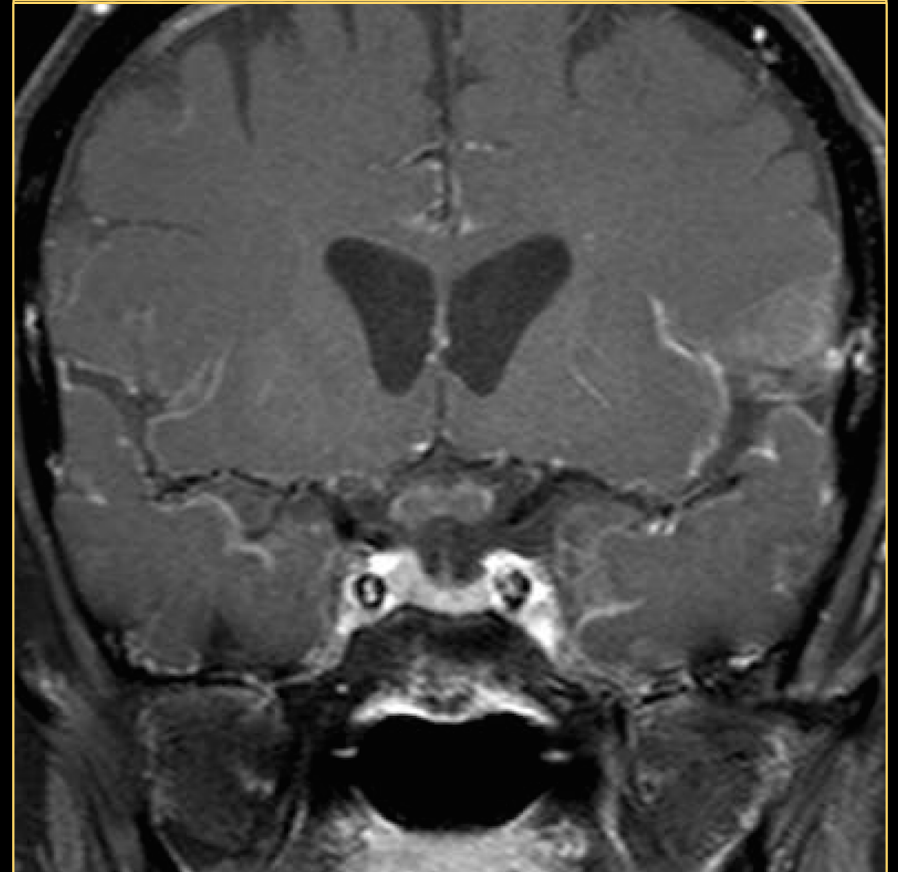
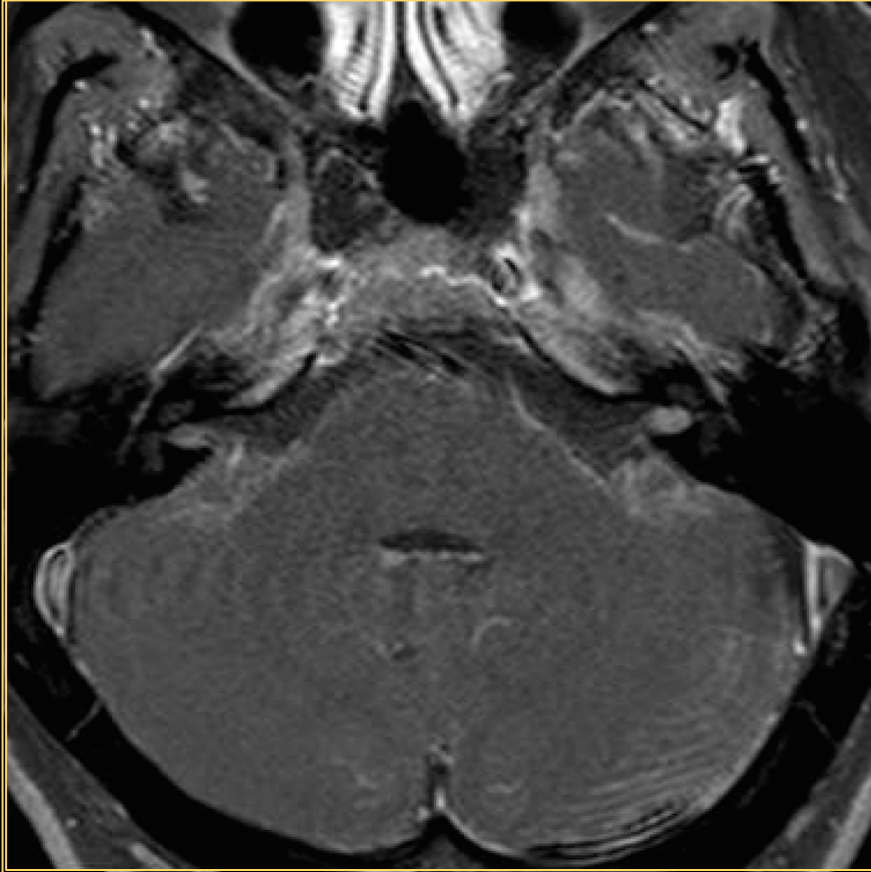
LEPTOMENINGEAL DISEASE

Neurosarcoidosis



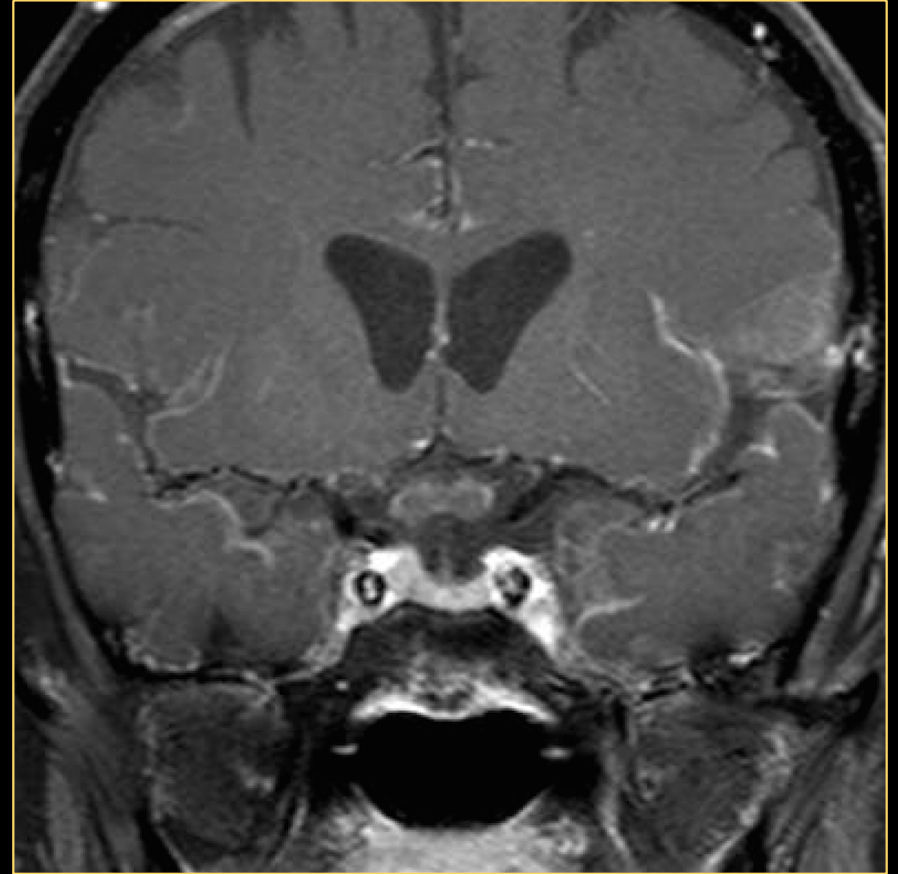
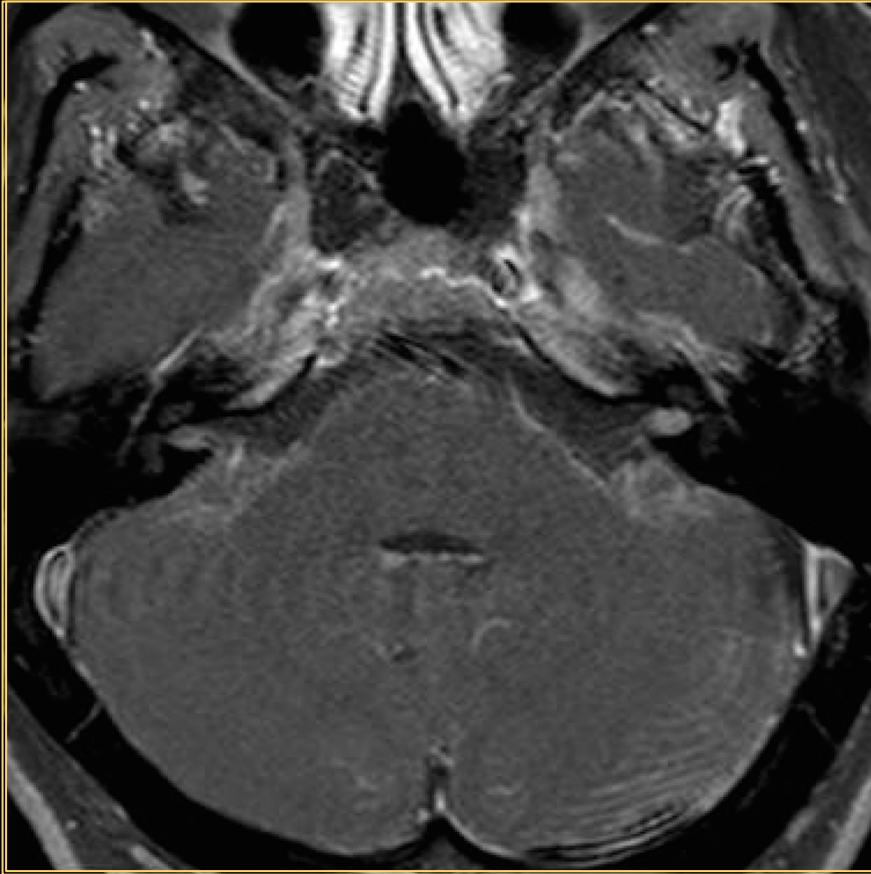
45 y F with R > L decreased vision & imbalance
Bilateral SNHL & pan-hypo-pituitarism

LEPTOMENINGEAL DISEASE



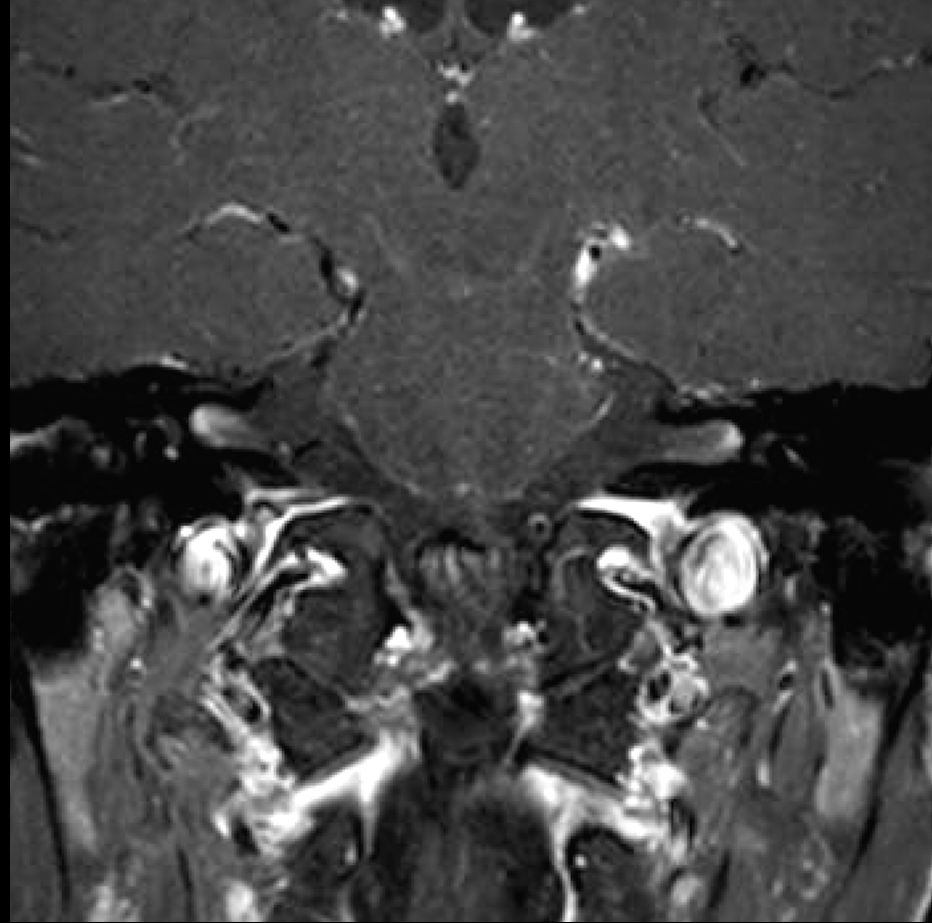
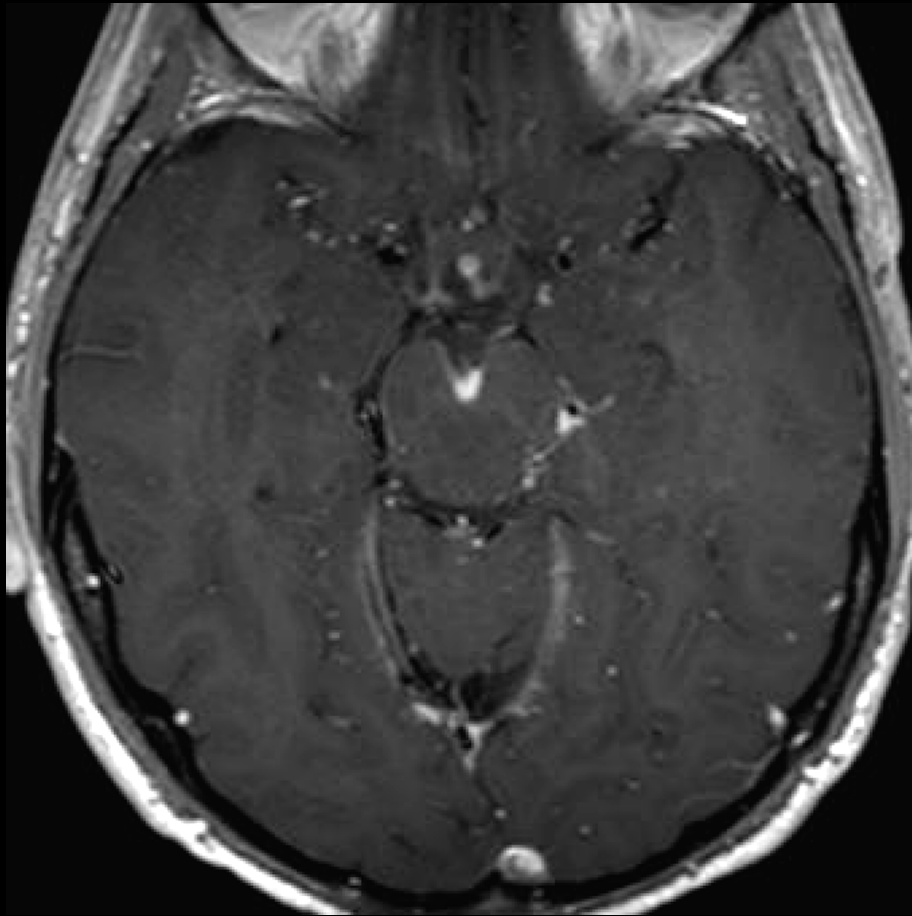
Multiple cranial neuropathies

NEUROTROPIC LYMPHOMA



DDX: Sarcoid, infection

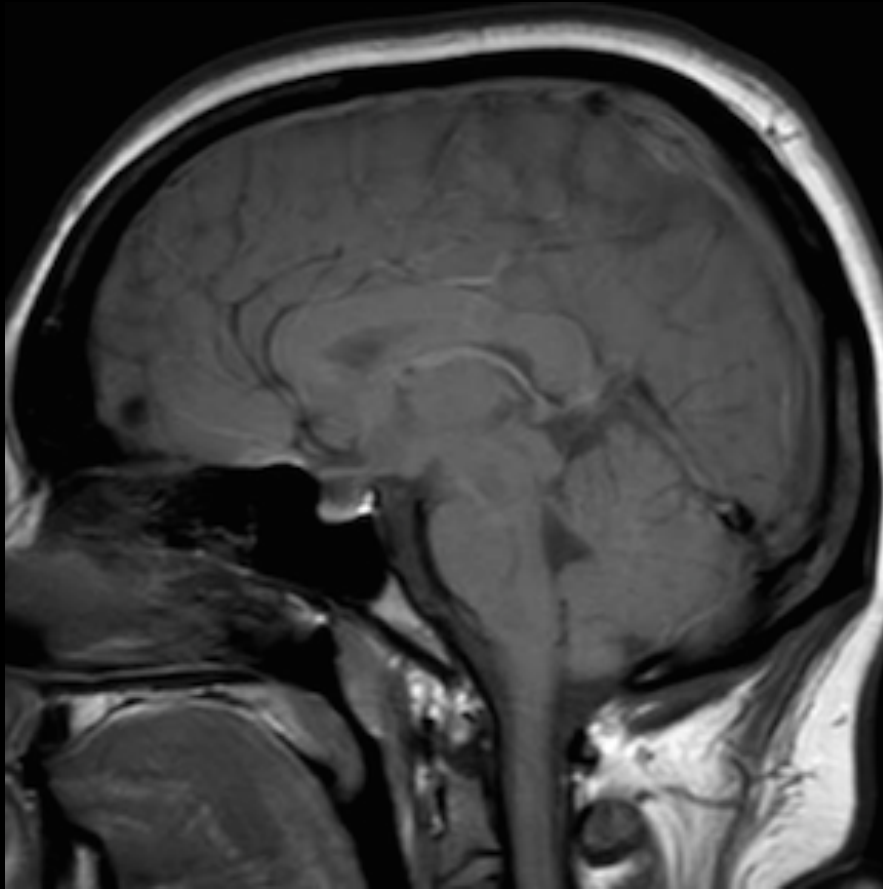
CRYPTOCOCCAL MENINGITIS



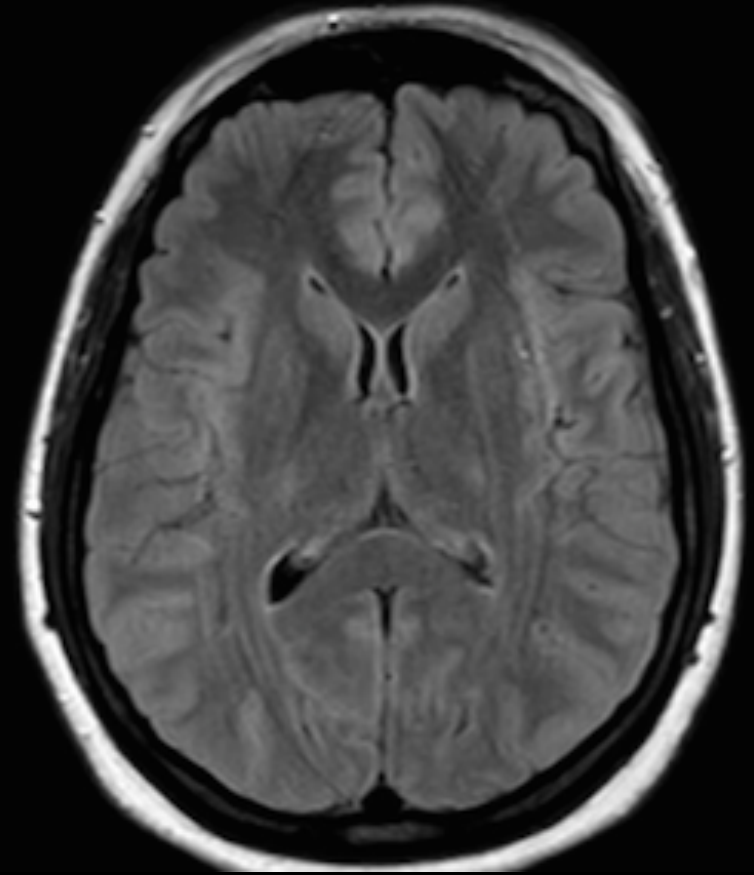
C gattei a pacific NW special!
Affects the immunocompetent

PAPILLEDEMA

17 y F with headaches



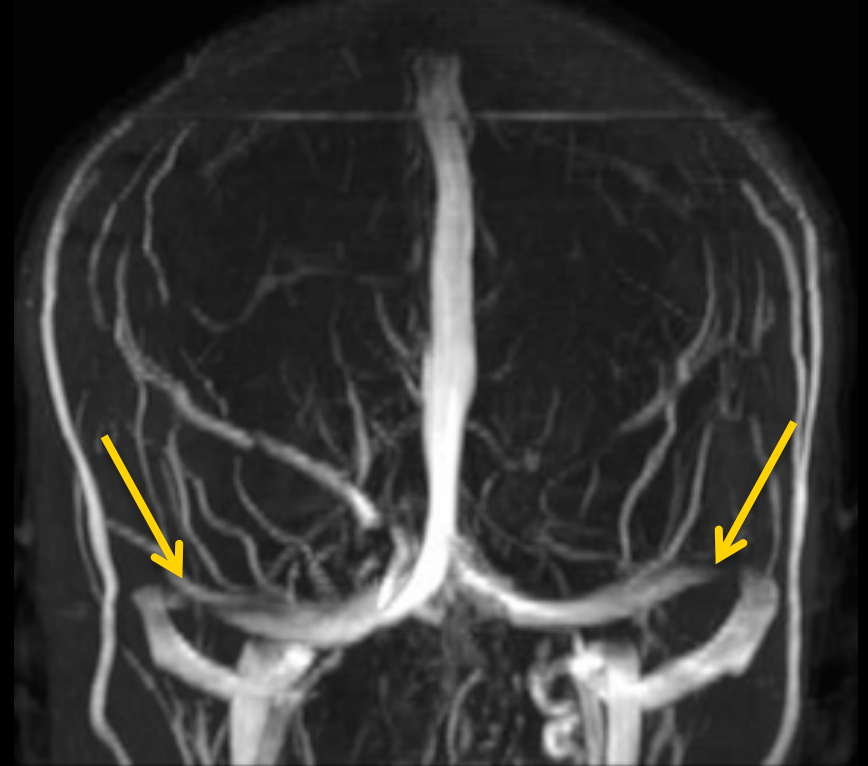
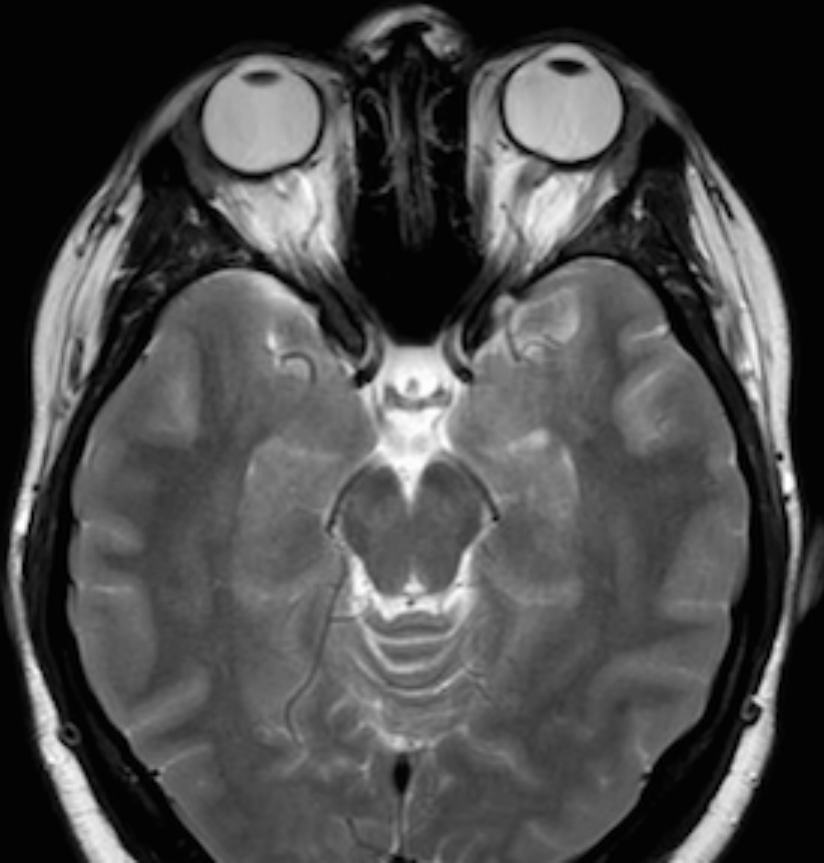
Cisterns effaced?



Ventricles slit-like?

INTRACRANIAL HYPERTENSION

MIP stenosis MUST be confirmed on source



High opening pressure at LP

Bilateral transverse sinus high-grade stenoses

VENOGRAPHY & STENOSIS TREATMENT

- Utility in context of ICH to identify stenosis
- Catheter venography with measurement of pressure gradient across stenosis
- Treatment: Stenting controversial
 - Stenosis: Cause or consequence?
 - Long-term anti-platelet/anti-coag not always B9

A. Rohr et al. Reversibility of Venous Sinus Obstruction in Idiopathic Intracranial Hypertension. American Journal of Neuroradiology Apr 2007, 28 (4) 656-659.

MEDICALLY REFRACTORY IIH: NEXT STEP?

- Optic nerve sheath fenestration (n= 712)
 - Vision better in 59%, HA 44%, papilledema 80%
 - Major/minor complications 1.5%, 16.4%
 - 14.8% required repeat procedure
- CSF diversion (n= 435)
 - Vision better in 54%, HA 80%, papilledema 70%
 - Major/minor complications 7.5%, 32.9%
 - 43% required 1+ surgery
- Dural sinus stenting (n=136)
 - Vision better in 78%, HA 83%, papilledema 97%
 - Major/minor complications 2.9%, 4.4%
 - Redo required in 10.3%

Satti SL et al. Meta-Analysis of CSF Diversion Procedures and Dural Venous Stenting in the Setting of Medically Refractory Idiopathic Intracranial Hypertension. AJNR 2015 Oct;36(10):1899-904.

OUTSIDERS VIEWPOINT



COMPRESSIVE OPTIC NEUROPATHY

- Cause: Anything compressing the nerve
 - e.g. trauma, thyroid eye, inflammatory
 - No inherent optic nerve disease
- Important category since treatable with decompression (unless long-standing)
- Other orbital mass lesions

ORBITAL MASS DDX

- Secondary malignancy (metastatic disease or lymphoma)
- Primary malignancy (from lacrimal apparatus or soft tissue/osseous sarcomas)
- Benign neoplasms – meningioma (schwannoma)
- Inflammatory disorders (GPA, sarcoid, pseudotumor)
- Infectious complications, usually from sinus
- Vascular lesions (venous malformation)

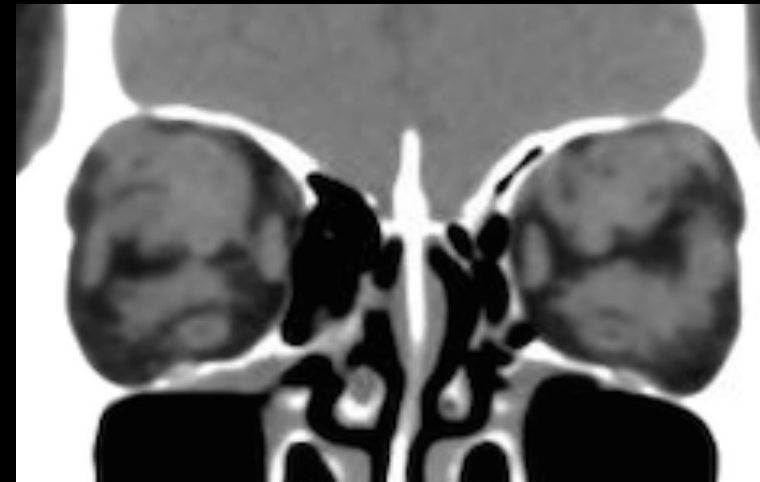
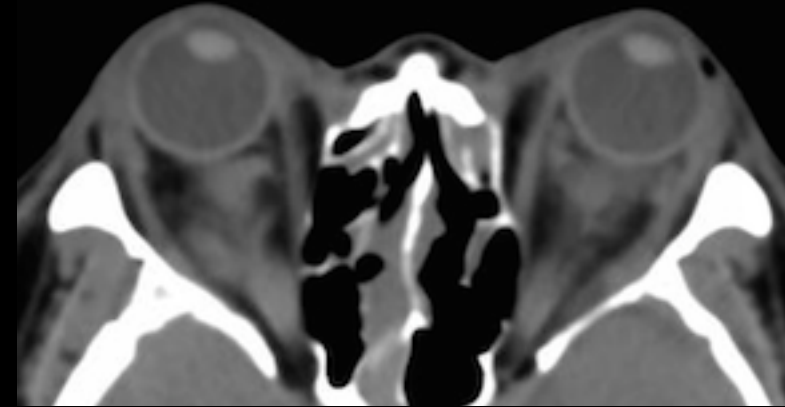
ORBITAL INFLAMMATORY DISEASES

- “Inflammatory” > 50% of all orbital diseases
 - Thyroid ~ 50% of orbital inflammatory disease
 - Infections ~ 45% other inflammatory diseases
- Others (~5%):
 - Idiopathic orbital inflammation (“pseudotumor”)
 - Systemic disease & vasculitis
 - Systemic granulomatous disease
- Inflammatory disease may be intrinsic (involving the nerve itself) or extrinsic

INFLAMMATORY APPEARING TAO

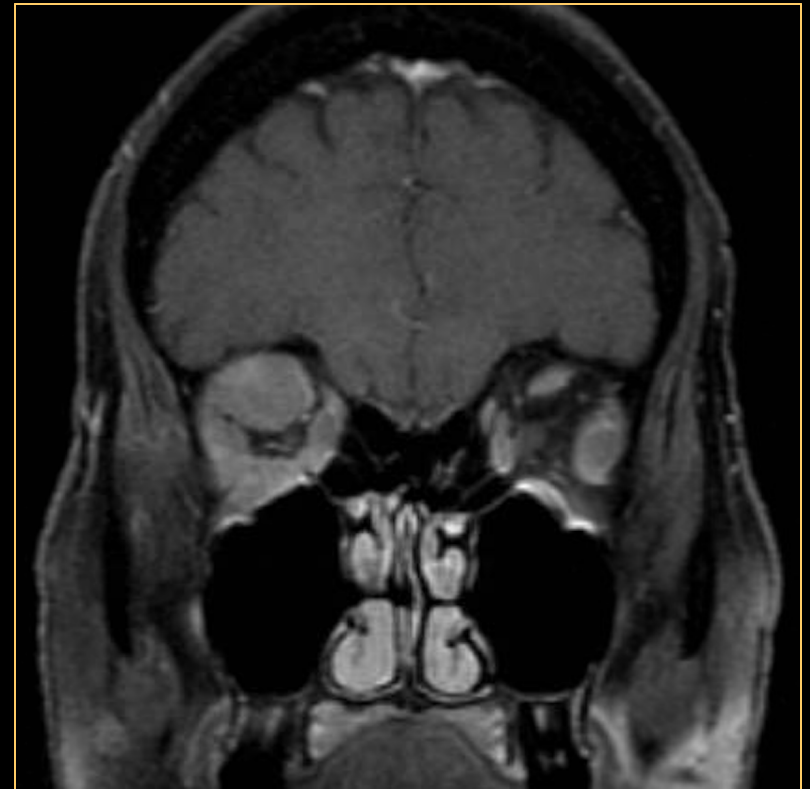
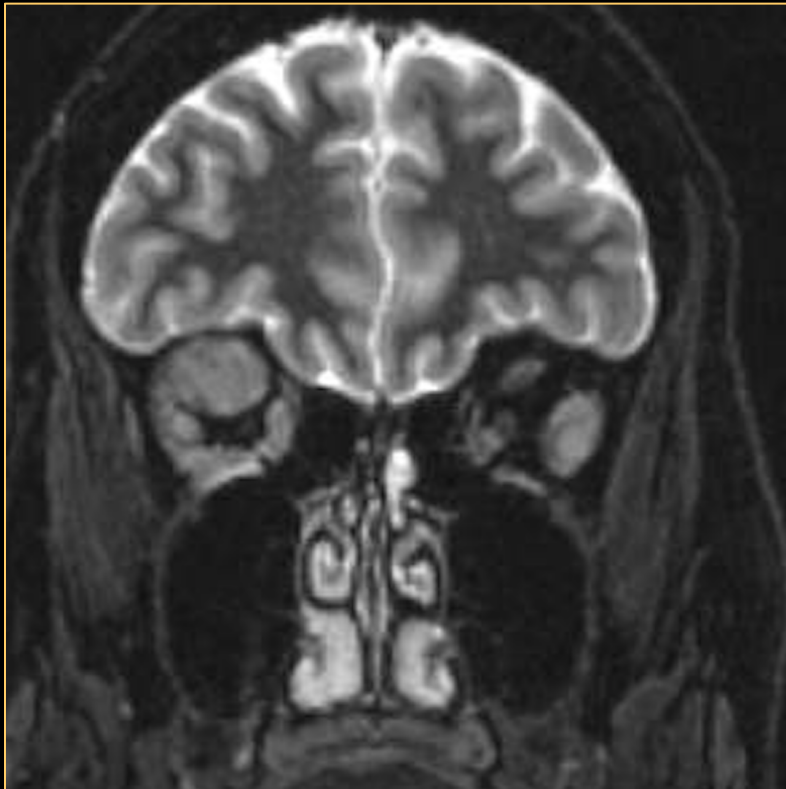
Atypical ragged borders

- Symmetric & bilateral more suggestive
 - May be asymmetric
- Spares tendinous insertions
- Subtle cases? Size ≥ 5 mm thickness (coronal)
- Clinically may be silent
 - Not associated with eye pain
 - 5% lack confirmatory antibodies at time of presentation
 - May cause diplopia rather than proptosis



THYROID ORBITOPATHY?

Orbital Chloromas



47 yo F w blurry vision & R proptosis

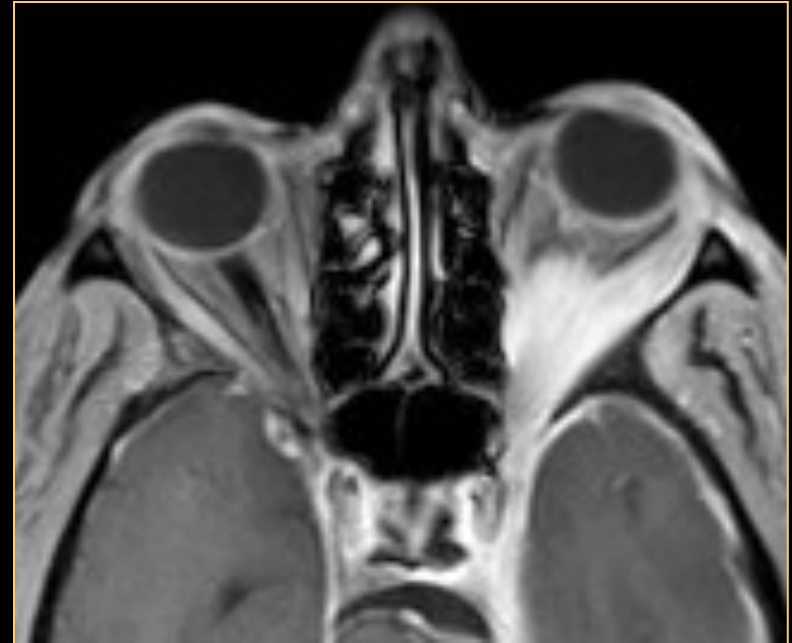
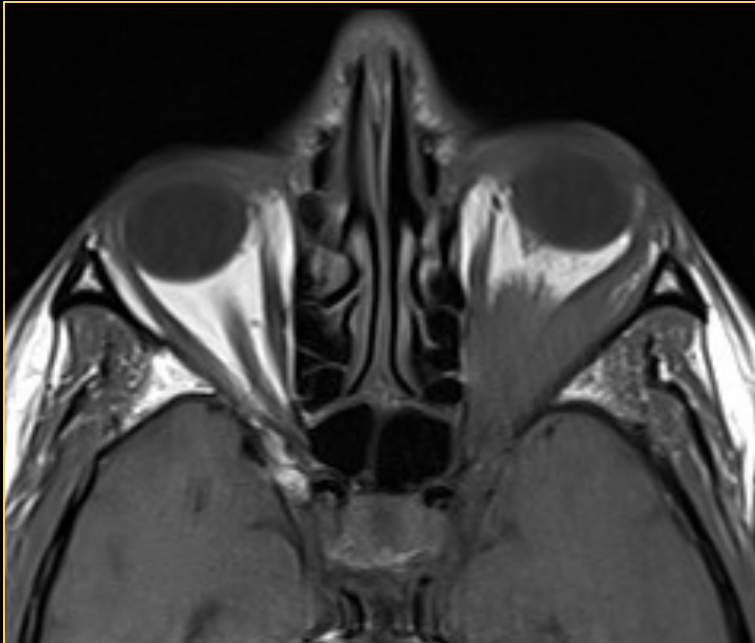
Extraocular muscle enlargement R>L

* Active tx

for AML

INFLAMMATORY DISEASE

Idiopathic Orbital Inflammation (Pseudotumor)



Spectrum of involvement: Mild retrobulbar fat stranding to myositis, lacrimal gland involvement, perineuritis

Orbital apex involvement → Tolosa Hunt Syndrome

IDIOPATHIC ORBITAL INFLAMMATION

Clinicoradiographic features

- Clinically:
 - Eye pain with movement
 - Variable association with autoantibodies
- Imaging spectrum:
 - Myositic form (involves EOM)
 - Lacrimal
 - Inflammatory neuritis or perineuritis
 - Non-specific fat stranding
- Check other soft tissues for adenopathy or parotid masses/lymphoepithelial lesions

84 Y M WITH HX DM

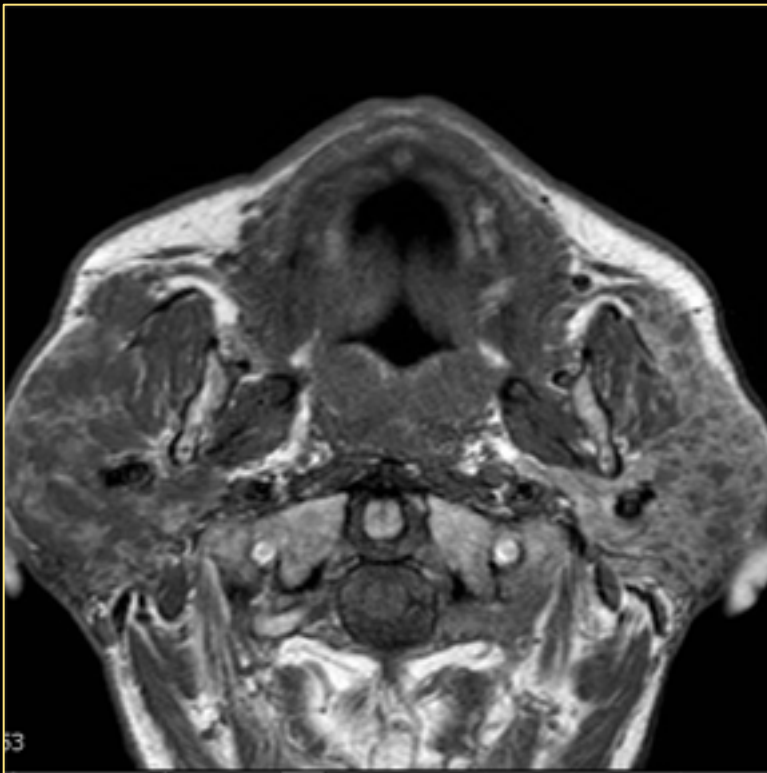
Infiltrative bilateral masses involving lac glands, +/- EOMs & extraconal fat



- Presentation: L eye pain & itching
- Bilateral proptosis & upper lid swelling on exam
- Lac gland bx: Chronic & granulomatous inflammation

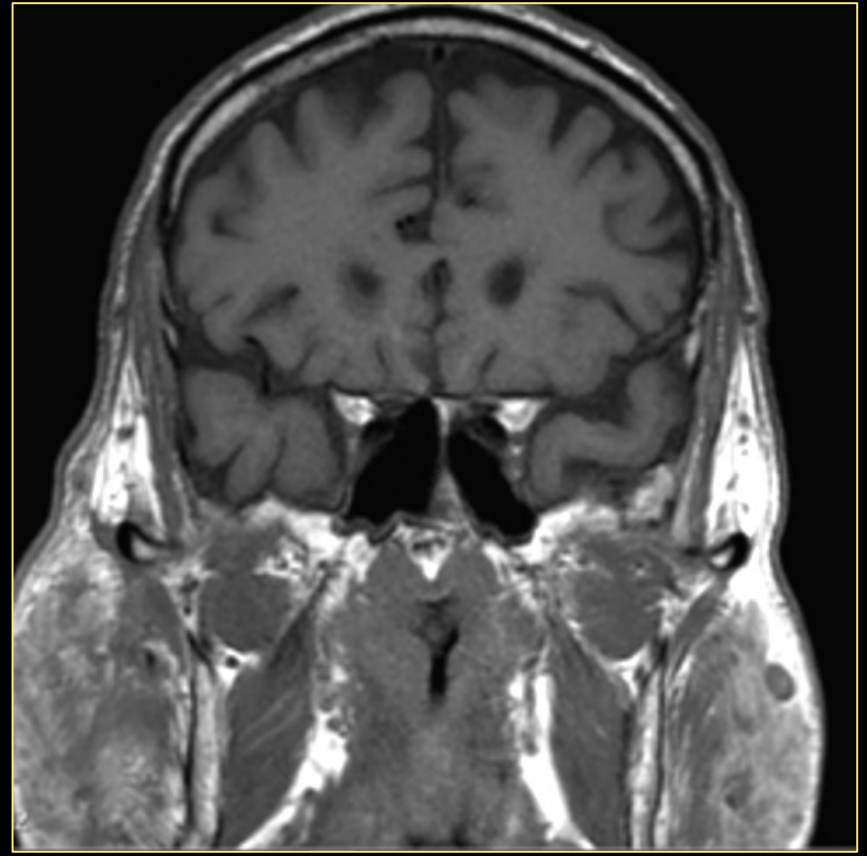
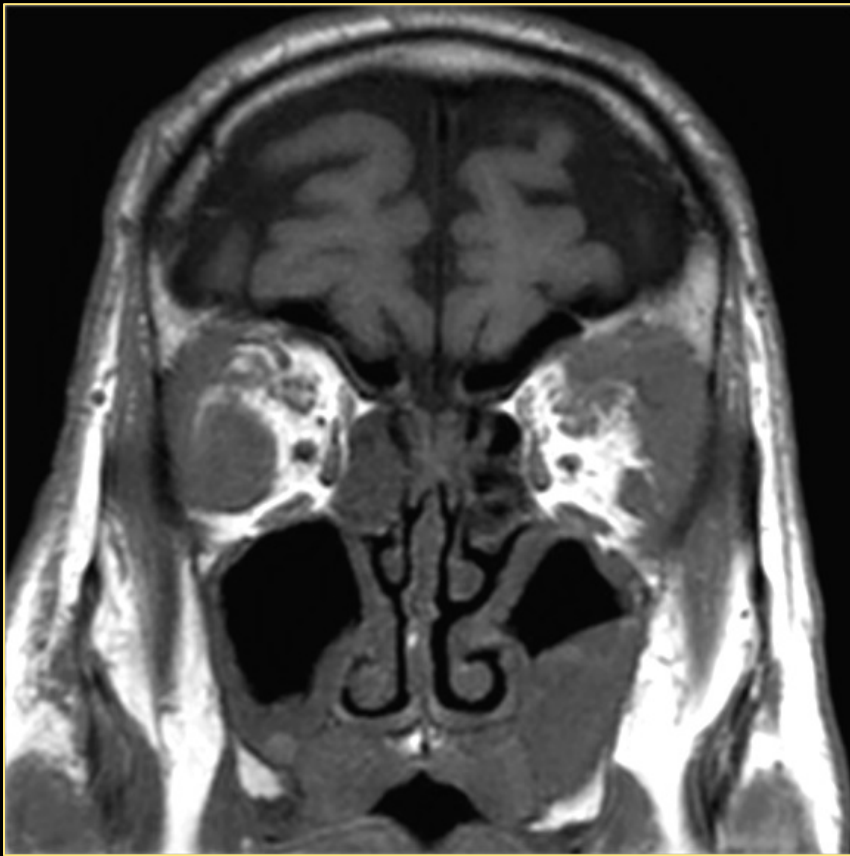
BILATERAL PAROTID ENLARGEMENT

Mikulicz Syndrome (old terminology)



Hx Sjogren's syndrome, + serum anti-Ro and anti-La antibodies

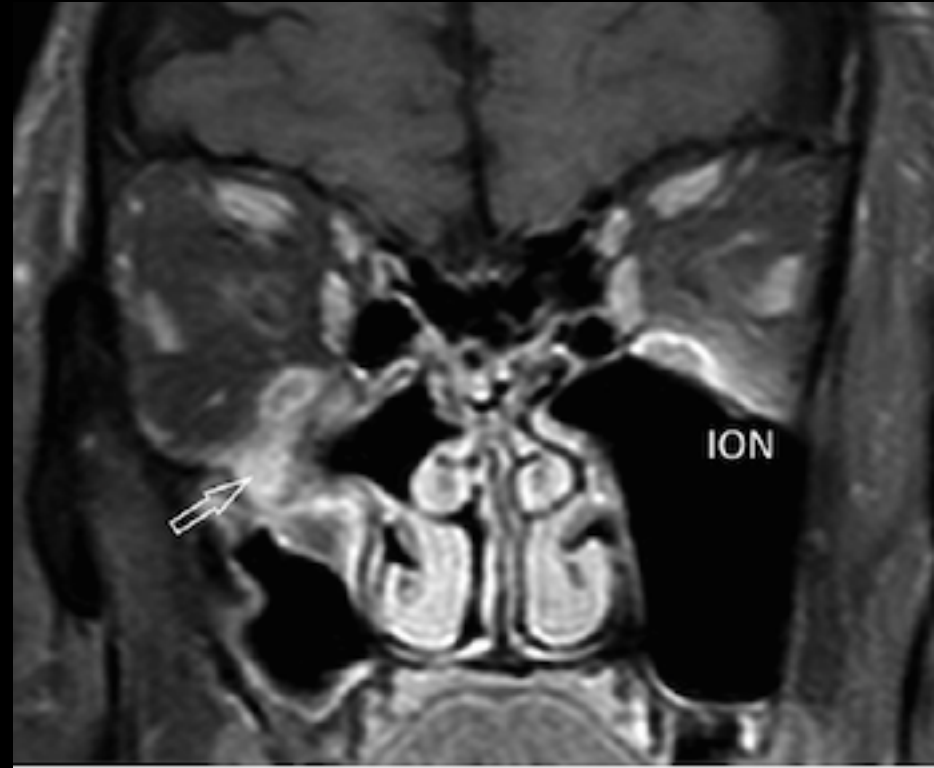
IGG-4+ ORBITAL INFLAMMATION



Patient responded to steroids

IGG-4 RELATED DISEASE

- Imaging tips:
 - Look for other areas involved
 - Orbits & salivary glands common
 - **Trigeminal nerve** involvement typical
 - Infraorbital nerve enlargement more common in IgG-4
 - Serum/path findings important



Thompson A, Whyte A. Imaging of IgG4 Related Disease in the head & Neck. *Clinical Imaging*. 73 (2018) 106-120.

IDIOPATHIC ORBITAL INFLAMMATION

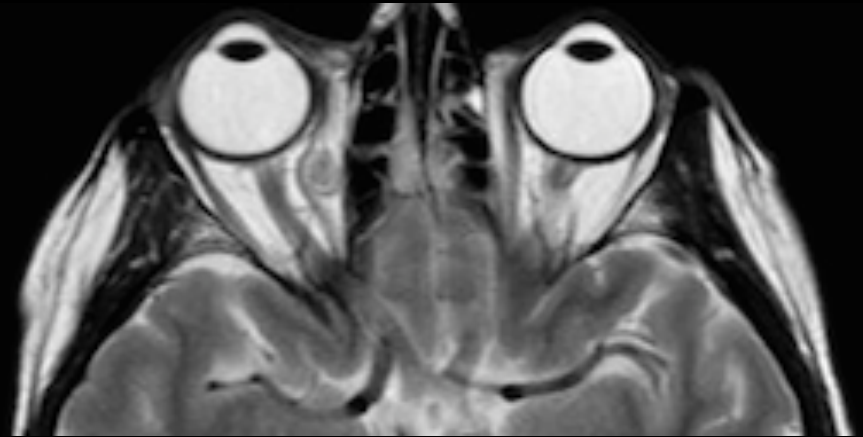
- Old terminology: Orbital pseudotumor
- New classification subdivides orbital inflammatory disease based on serum autoAb:
 - IgG-4 positive
 - IgG-4 negative
 - IgG-4 “disease” generally associated w systemic inv
- IgG-4 + cases respond to steroids & carry increased risk for malignancy (lymphoma)

OTHER RECTUS MUSCLE LESIONS

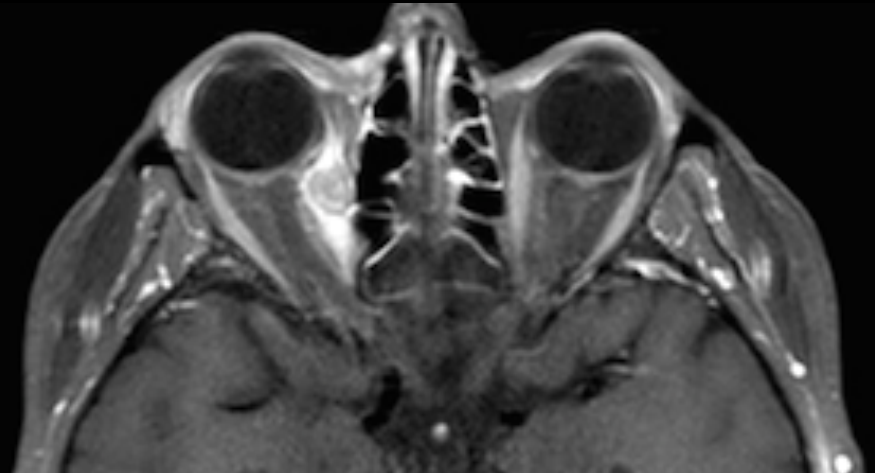
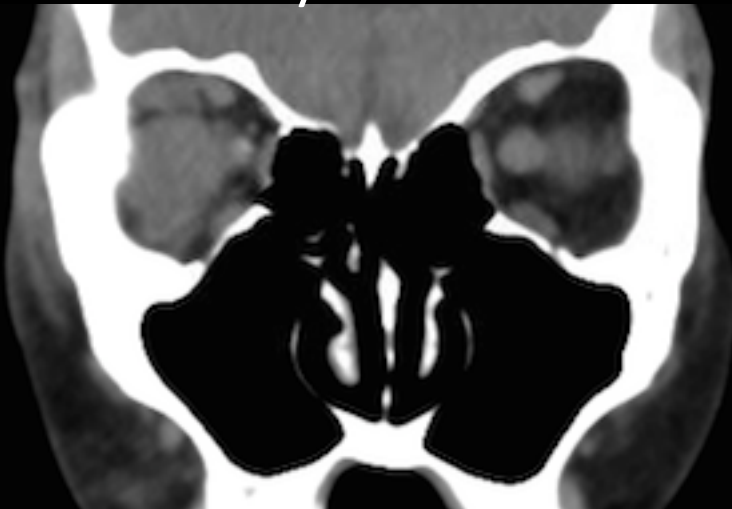
Sinonasal malignancy



61yF with RCC met

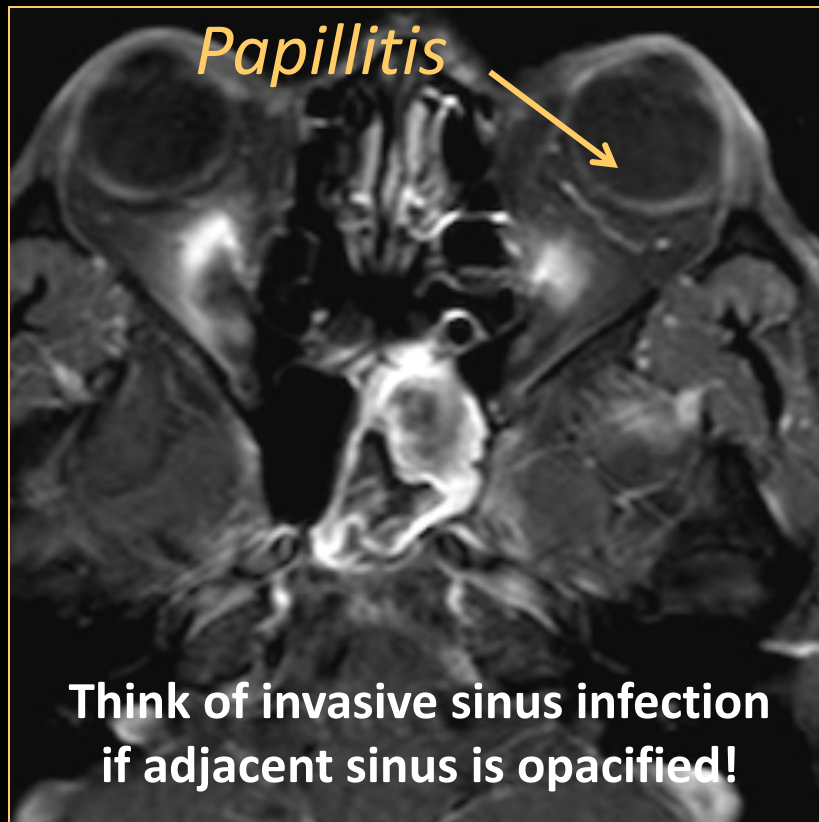


Myeloma



ORBITAL INFECTION

Invasive aspergillosis

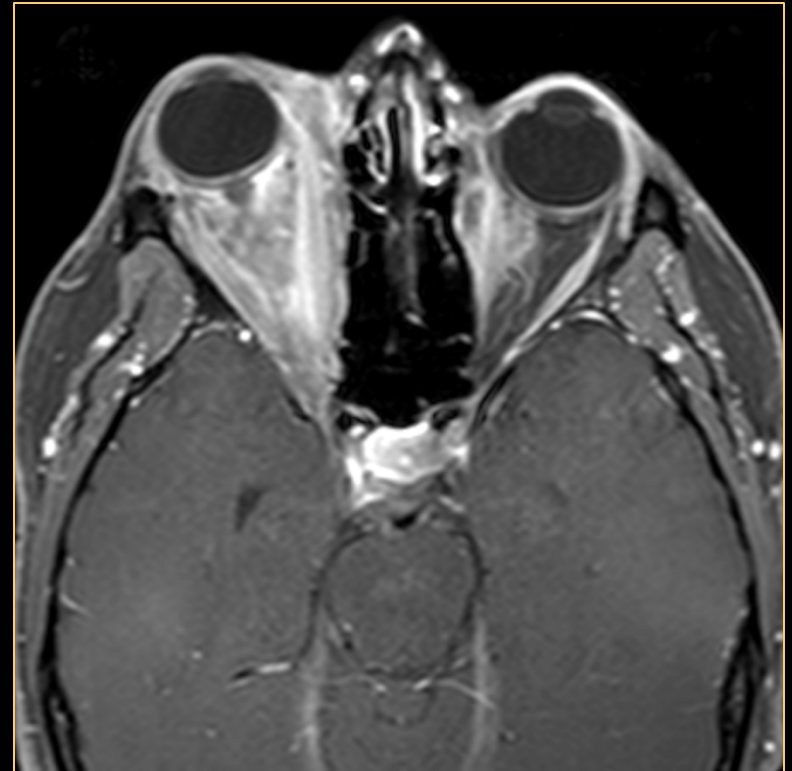
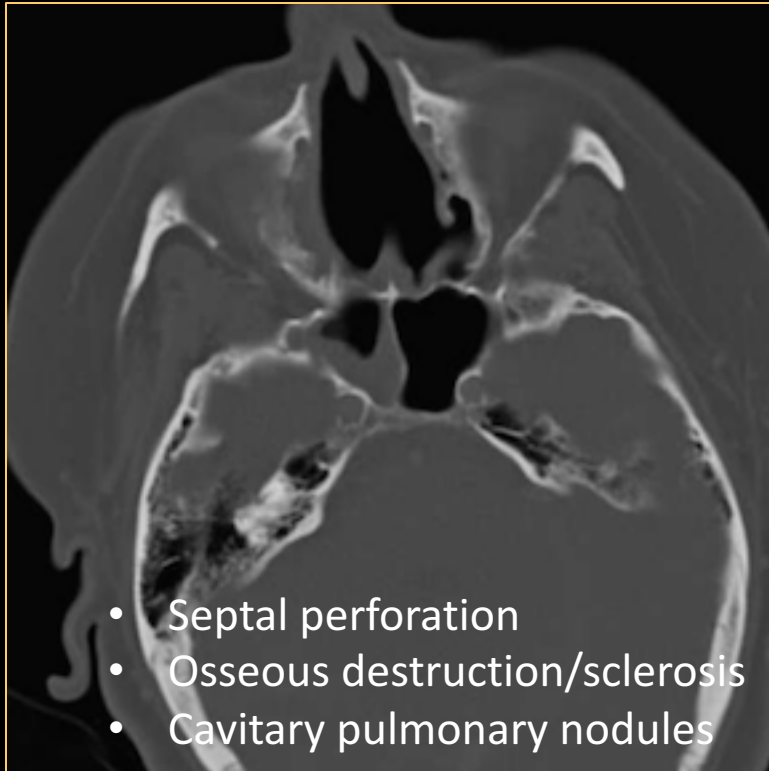


49 y M with acute left-sided vision loss

Chronic steroids, hydroxychloroquine, MTX for dermatomyositis

OPTIC NERVE INFLAMMATORY

Granulomatosis with Polyangiitis (Wegener's)

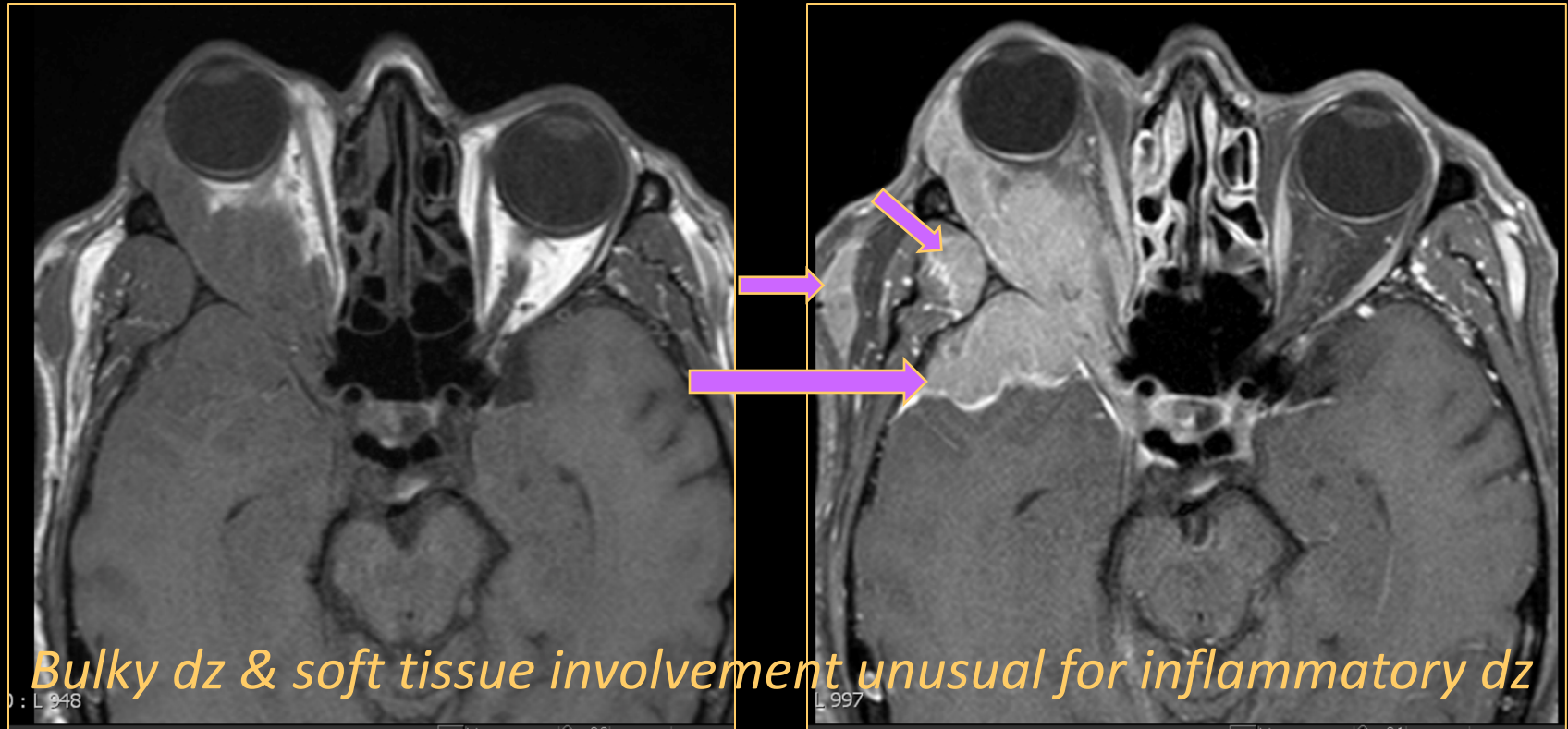


Imaging Checklist (suspect) inflammatory disease:



Paranasal sinuses & chest

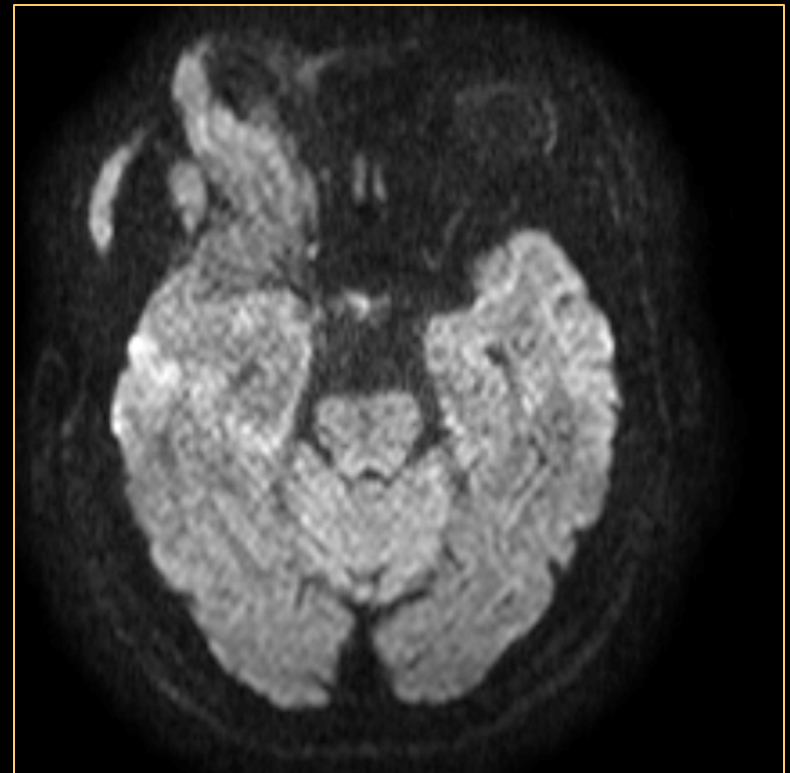
COMPRESSIVE OPTIC NEUROPATHY



- 58 yo M with R sided vision loss & proptosis
- Bulky orbital, dural, & high MS masses

ADDITIONAL IMAGES

MALT Lymphoma

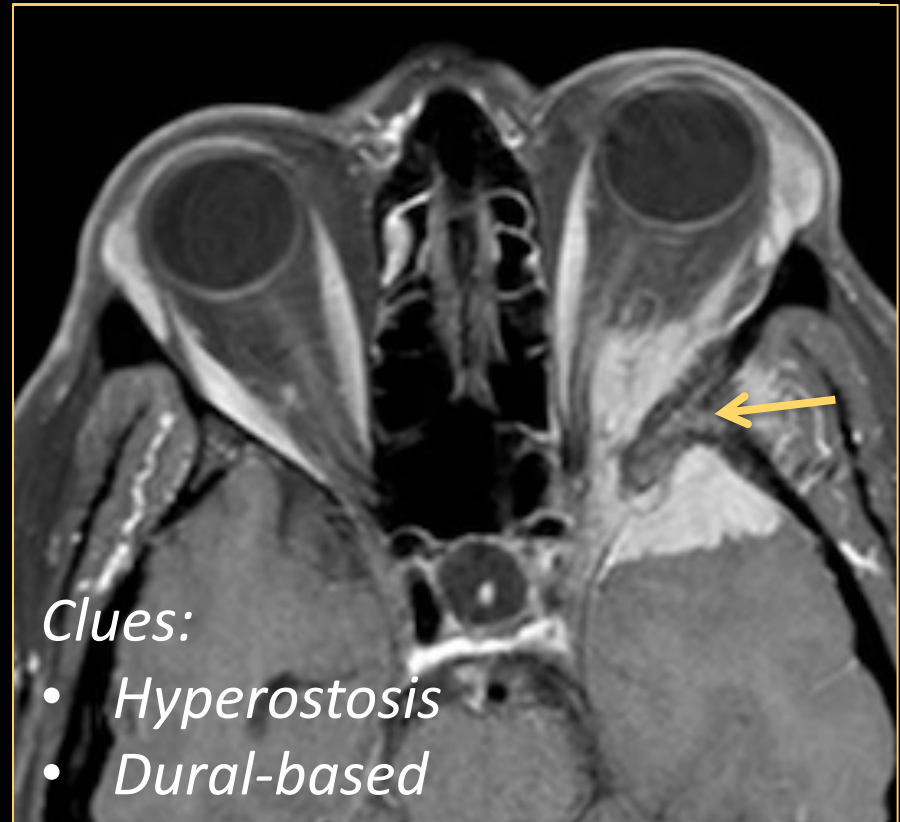
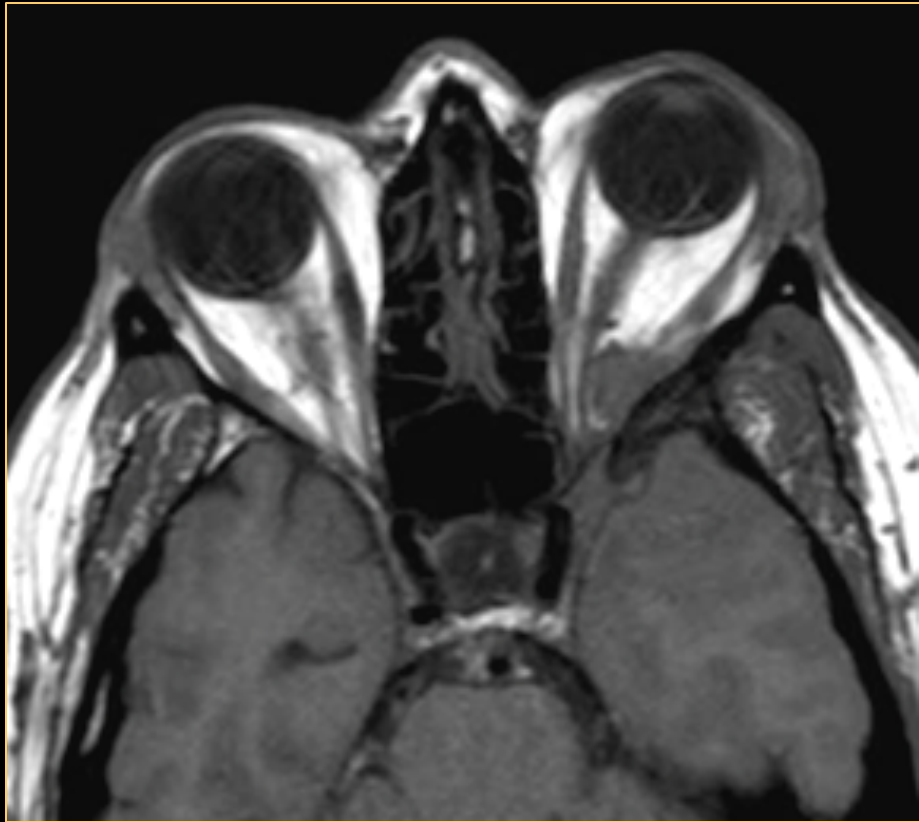


Parotid (and upper cervical) lymphadenopathy *

DWI isointense (not always bright)

51 Y F: 2 YR SLOW VISION LOSS

Sphenoid wing meningioma



Now blind: no light perception OS

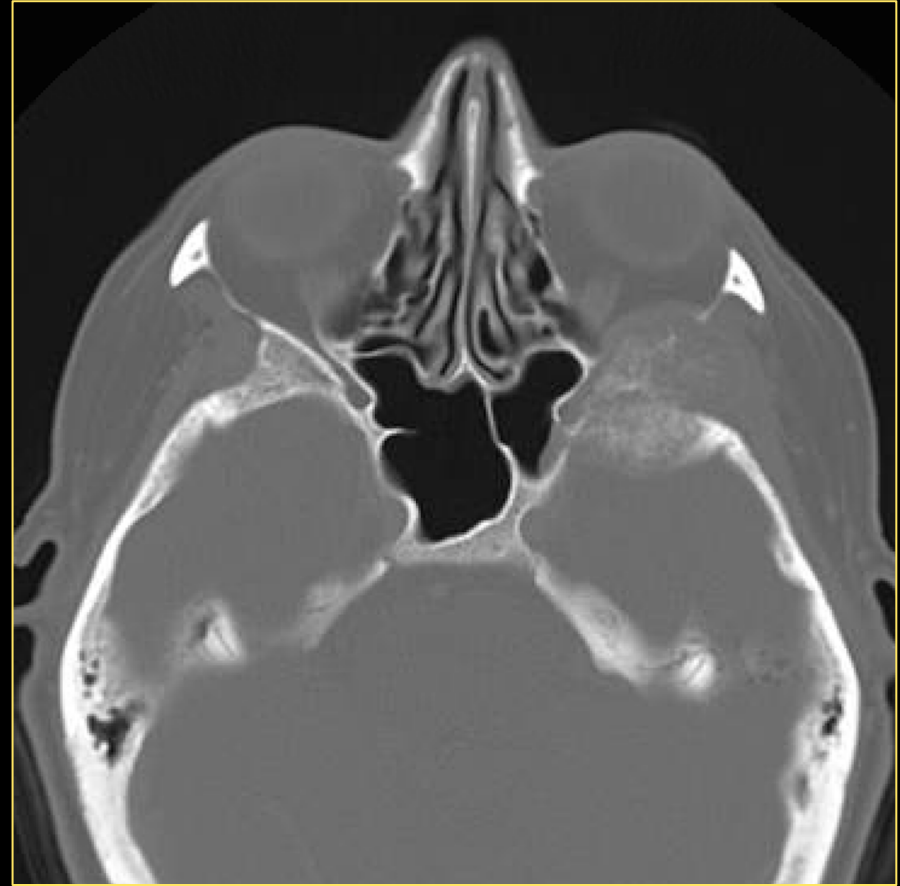
ORBITAL MENINGIOMA

- Imaging approach:
 - Orbital ultrasound: ca++
 - CT: look for ca++
 - MRI: best for extent & intracranial involvement
- Meningioma of optic nerve sheath:
 - Tram track ca++
 - Sheath, not nerve enhancement
 - Frequent extension to orbital apex, intracranial
- Sphenoid wing meningioma:
 - Hyperostosis & tumor ca++ (✓ CT)
 - Dural-based enhancing mass (MRI >> CT)
 - Blastic bone metastasis a possible mimic

WHAT ELSE IS IN THE DDX (HYPEROSTOSIS)?

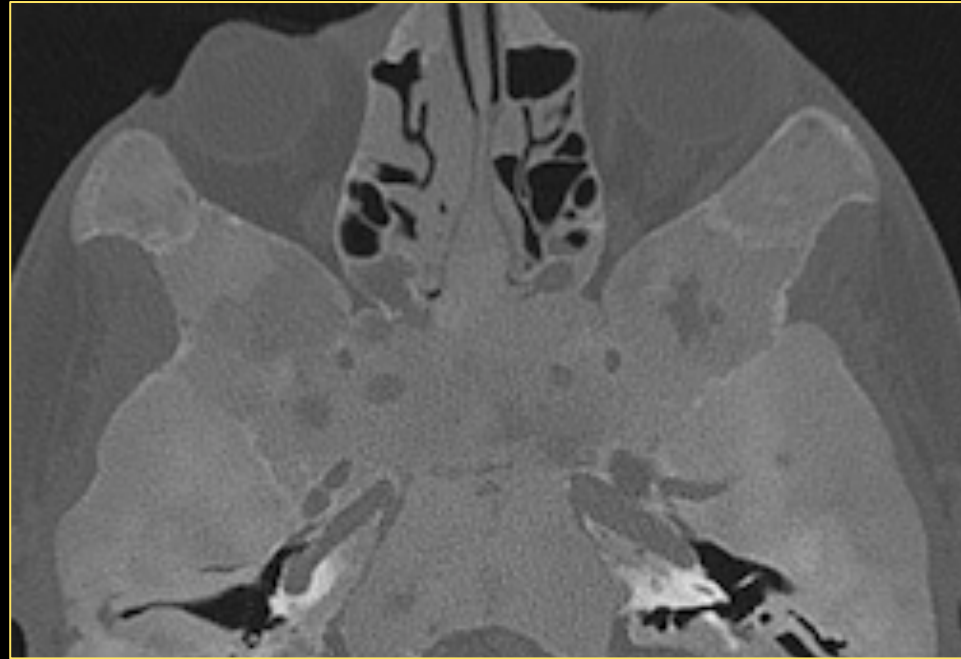
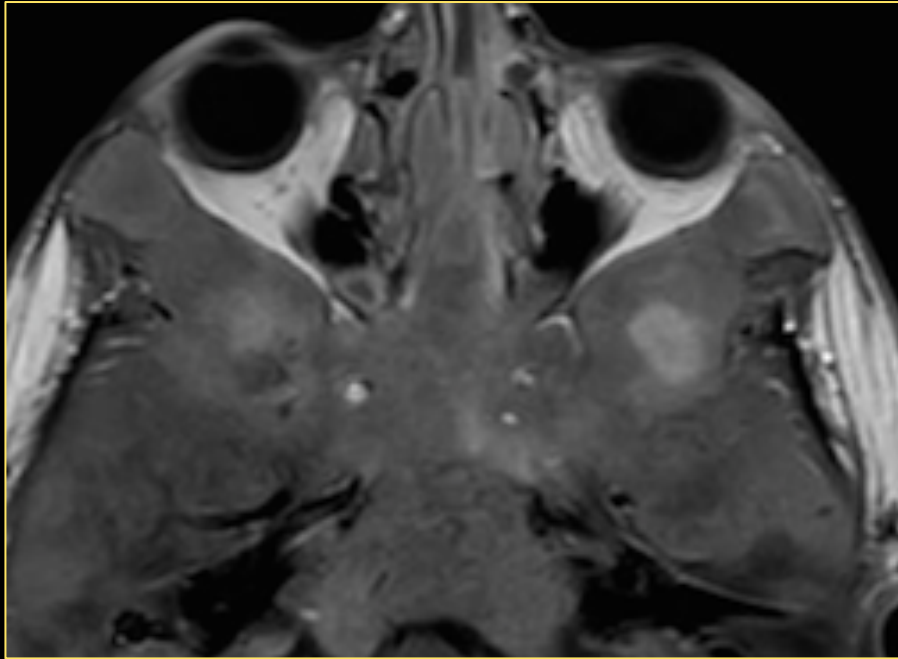
- **Osteoblastic metastasis**
 - Especially *prostate & breast* cause hyperostosis
 - Look for dural tail on enhanced MRI
- **Fibrous dysplasia** mimics several aggressive processes on MRI:
 - CT is diagnostic w “**ground glass density**”
 - NO dural tail of enhancement
 - Bone enhancement in active phase (younger patients)

OSTEOBLASTIC METASTASIS: Colon Cancer Metastasis



FIBROUS DYSPLASIA

McCune Albright Syndrome



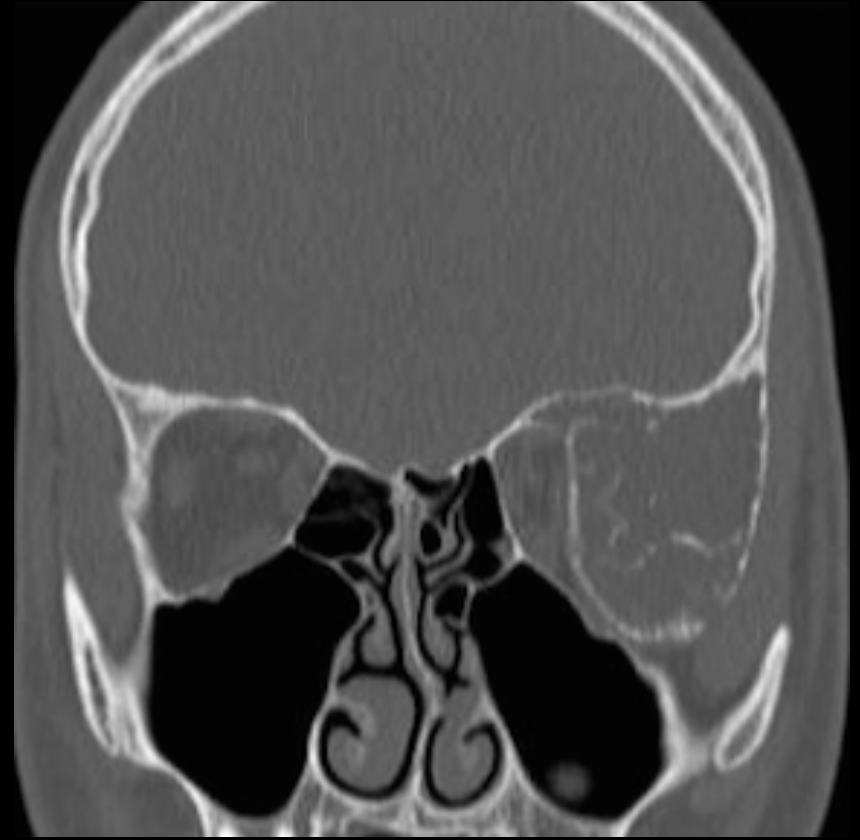
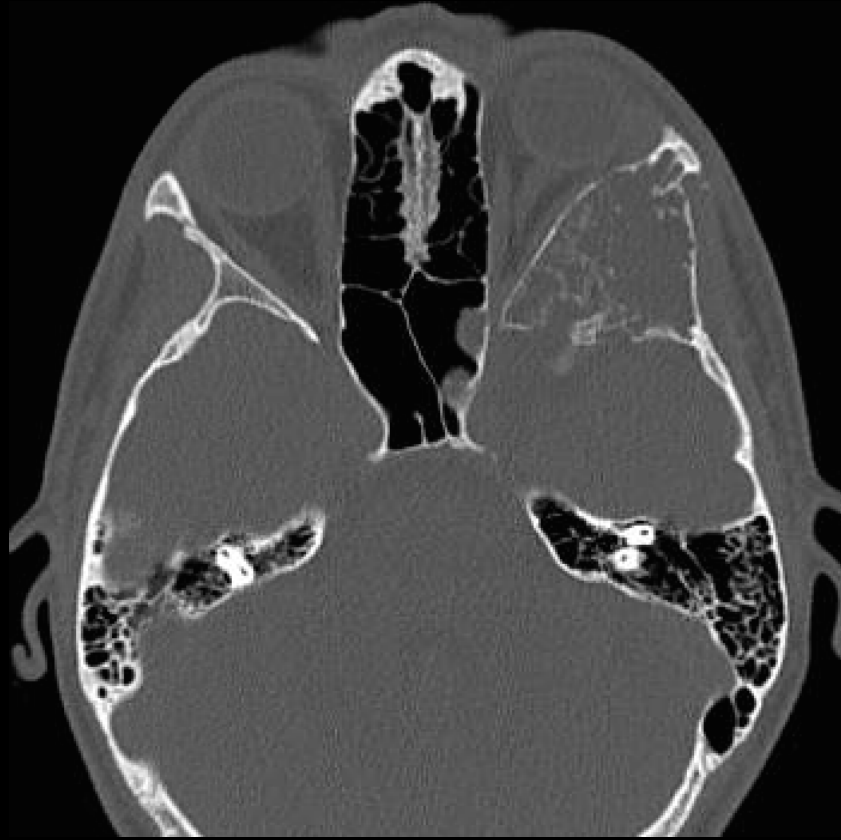


You're nearly there!

CSF LEAKS & CEPHALOCELES

- High resolution CT & MRI complementary
 - CT for skull base defects
 - MRI for CSF contiguity
- Some cases require cisternography
- Imaging characteristics:
 - Fluid density & signal (soft tissue contiguous with brain with cephalocele)
 - DWI follows CSF (no restriction)

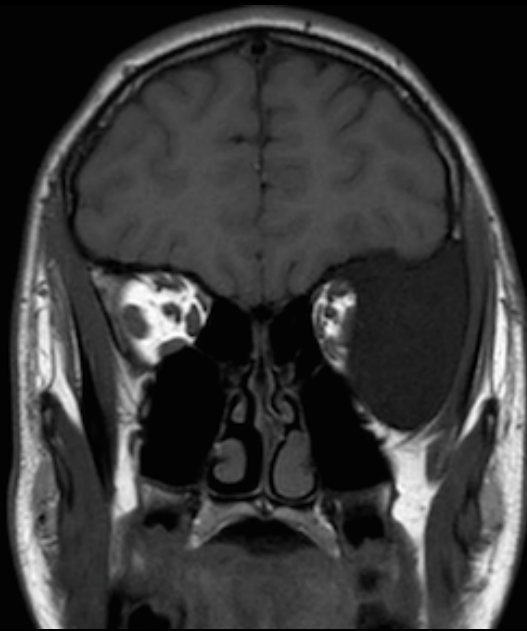
17 Y M: 3 MO PROGRESSIVE L PROPTOSIS



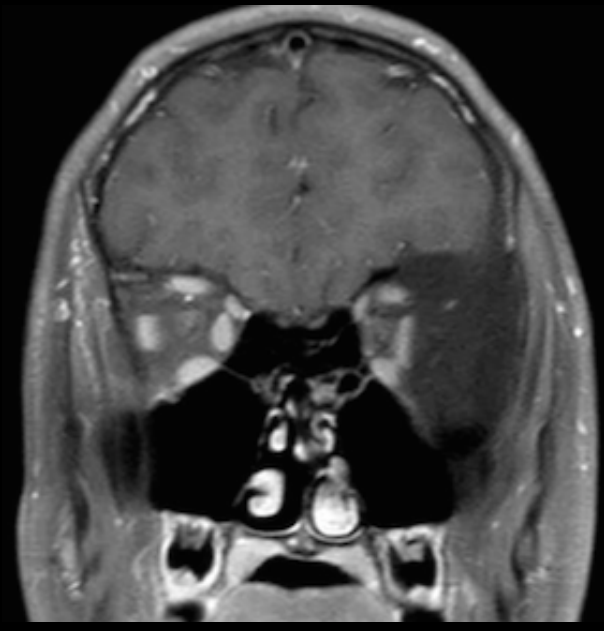
MRI IMAGING



T2: CSF Signal

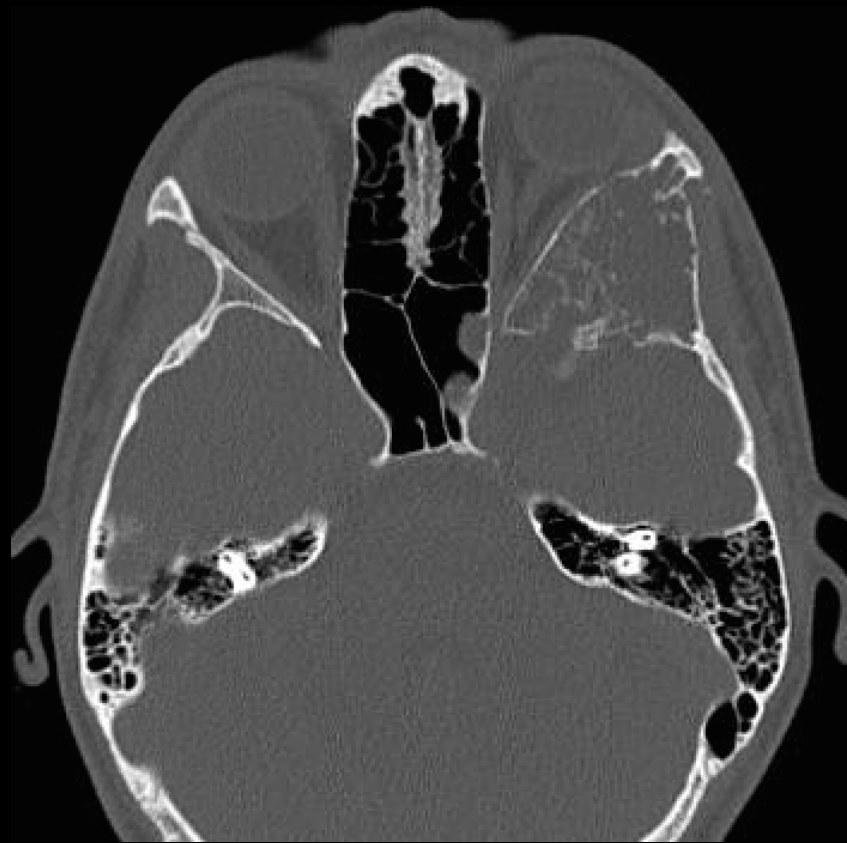


T1: CSF Signal




T1 C+: No enhancement

GIANT PSEUDOMENINGOCELE



IMAGING FOR OPHTHALMOLOGISTS

- NECT best for bony lesions (e.g. trauma), ca++, anatomy, TAO & quick screens
- Order NECT & Enhanced MRI for skull base pathology
- MRI orbits is best for soft tissue detail
 - Brain for suspected intracranial lesions
 - MRA/MRV or CTA/CTV for vascular lesions
 - DWI for abscess, epidermoid, stroke (gray CSF!)
 - Spine for myelopathy
 - ✓ for extraorbital involvement



Thank-you!
Comments/questions/suggestions:
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