ORBIT IMAGING

- **CT ORBIT :**
  - Axial & Coronal
  - -Bone & soft T window.
  - 3:5 mm
  - Contrast 1:2mL/Kg

- **MRI**
  - T1 fat suppression before & After contrastis a must

- **ORBIT PATHOLOGY:**
  - **Globe**
  - **Optic N**
  - **Lacrimal gland**
  - **Intra orbital FAT**
  - **Extra ocular Muscles**
  - **Bones of orbit**

I- I.O.LESIONS

1 . Retinoblastoma

2 . Melanoma

3 . Deposits

4. Others

1. RETINOBLASTOMA

Don't Forget Ca – Bilatrality – Extra ocular extension

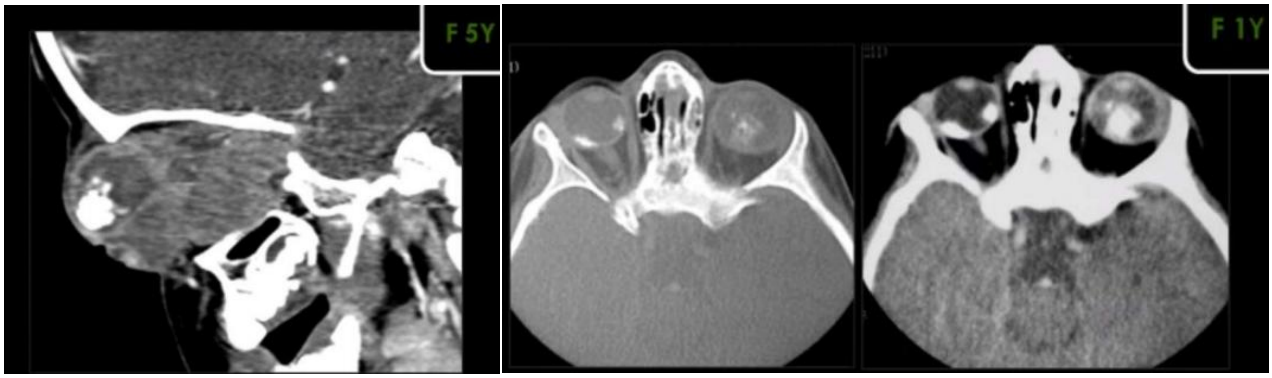
- ***Most common 1ry intra ocular tumor in child (first3 yrs).***
- < 2y - 30% Bilateral
- **CALCIFICATION** is Clue of diagnosis

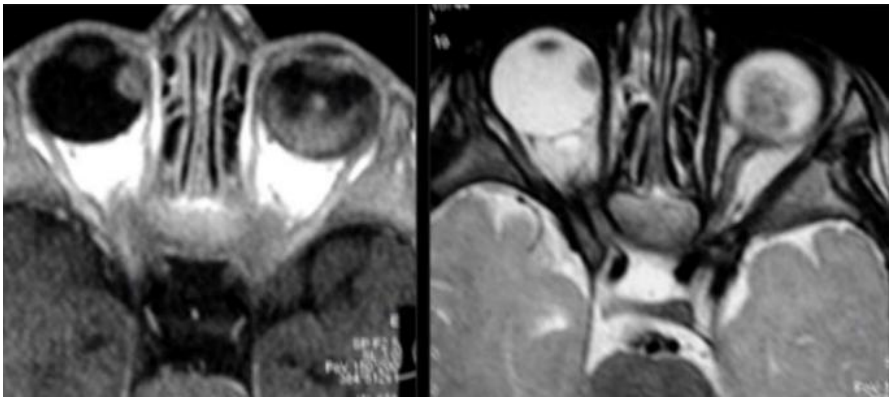
➔ **CT:**

- I.O. Hyper Dense + Cain child < 2Y
- Moderate enhanced -/+ Extra Orbital. Extension
- >- Thick enhancing
- Optic N

➔ **MR:** *MR more sensitive to detect I.C extension*

* T1 iso : Hi * T2 Low (I.e. Against vitreous signal) * Moderate enhancing





Trilateral retinoblastoma = **B**ilateral & **p**ineal body lesion

Tetralateral retinoblastoma = **B**ilateral, **p**ineal body & **S**uprasellar.Lesion

RETINOBLASTOMA MIMICS

- **D.D.:** → *ALL Showing "No Ca"* - "Dense Globe, HiT1& T2"
- **PHPV "Persist HyperPlastic Vitreous**: :
  - Persist Hyaloid Vitreous
  - Small Globe
  - Distorted Lens
  - Retro Lental abnormal shadow.
- **COAT's Disease:** "*Unilateral Retinal Telangectasia*"
  - ~Most in Boys ~ Acquired ~UniLateral
  - ~ 80 % 6:8 y -Normal Globe size - Normal Lens
- **ROP "Retinopathy Of Premature"**

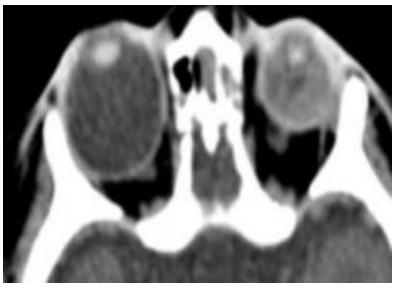
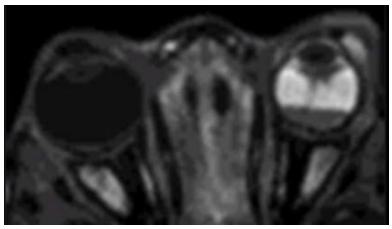
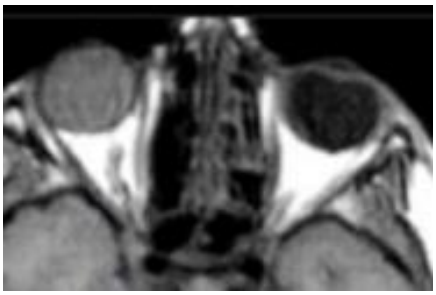
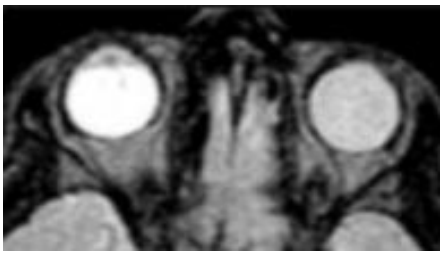
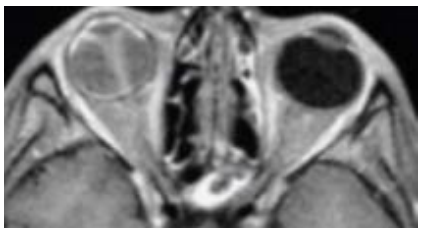
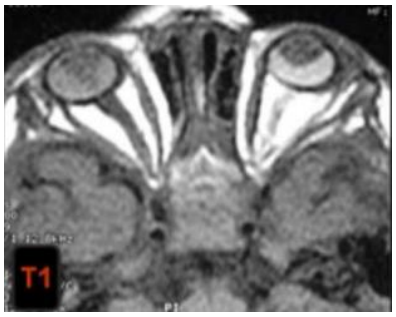
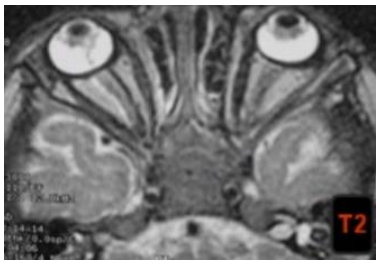
BiLateraL

~ Hi O2 Theraby

= Low density - Low signal T1 & T2WI

~ FibrobLast overgrowth

~ RetroLentaL Fibro scar

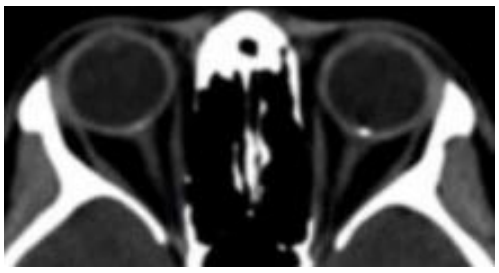
PHPV	COATS	ROP
 	  	 

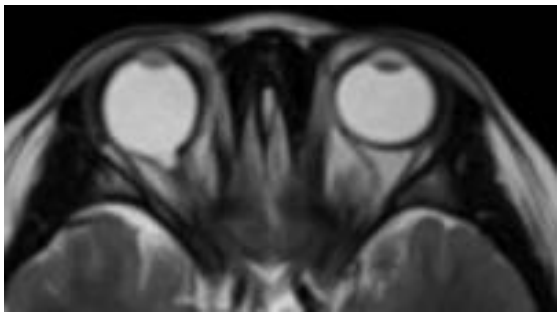
(DRUSEN Disease)

-Rare

-Bilateral Symmetrical

‹Optic disc Calcification





(COLOBOMA)

-Uni or Bi

Defect in wall->Retro

ocular cyst CONTINUOUS e

GLobe cavity .

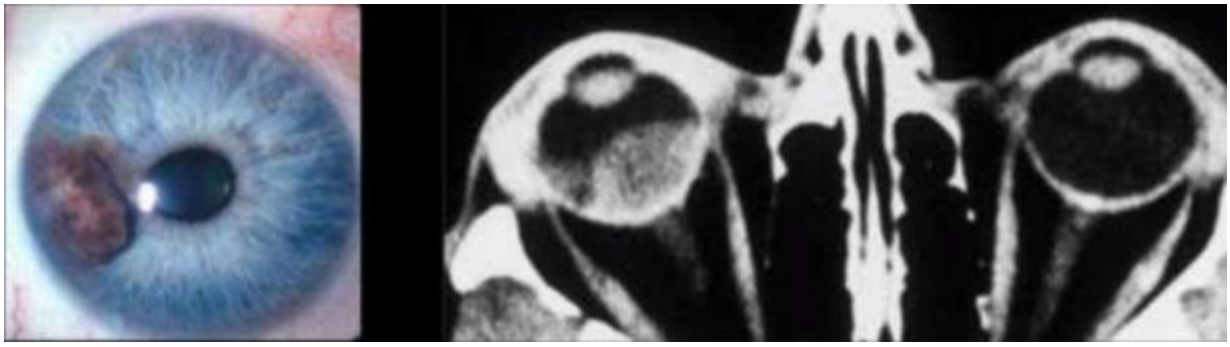
2. MELANOMA

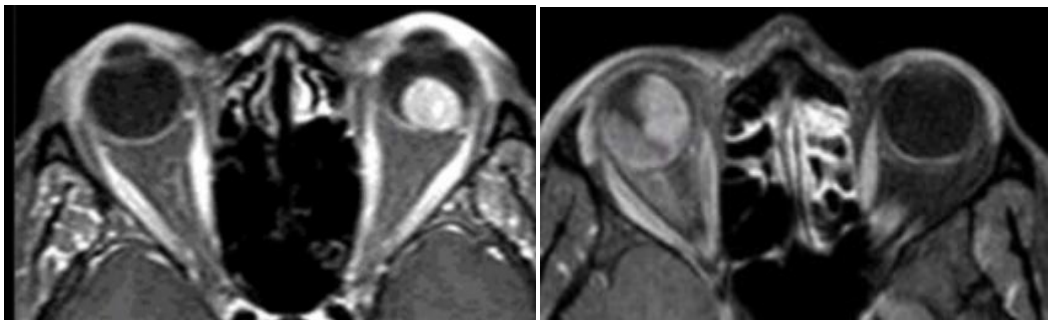
- COMMONEST Adult I.O.primary tumor
- No Ca
- 15 % extra Ocular extension
- May from Ciliary body i.e. anterior located.

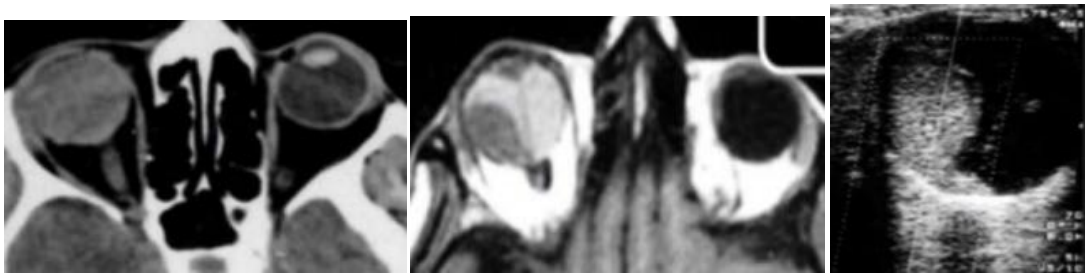
→ CT Well Defined - Hyperdense - Moderate enhancing

→ MR

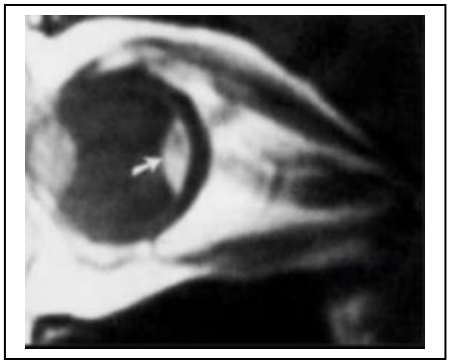
* Melanotic type → T1Hi / T2 Low * Amelanotic → intermediate signal







3. METS

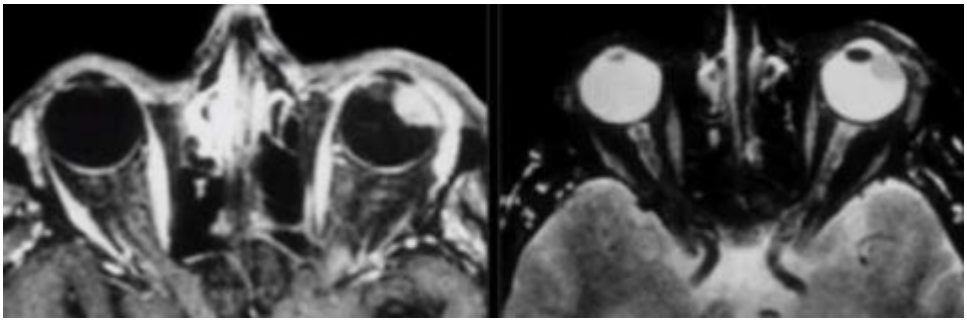


- Metas >Much more common than> Melanoma
- But consedered when +1ry
- Common sources : BREAST , LUNG & RENAL
- **Site**: mainly Choroid due to Hi Vascularity

-Common Extra Ocular extension

➔ **CT** * Isodense Lesion *Enhancing + Known Primary

➔ **MR** * T1 iso/T2 Hi



4 ChoroidaL Hemangioma

- Middle Age -Cong Vascular Hamartoma

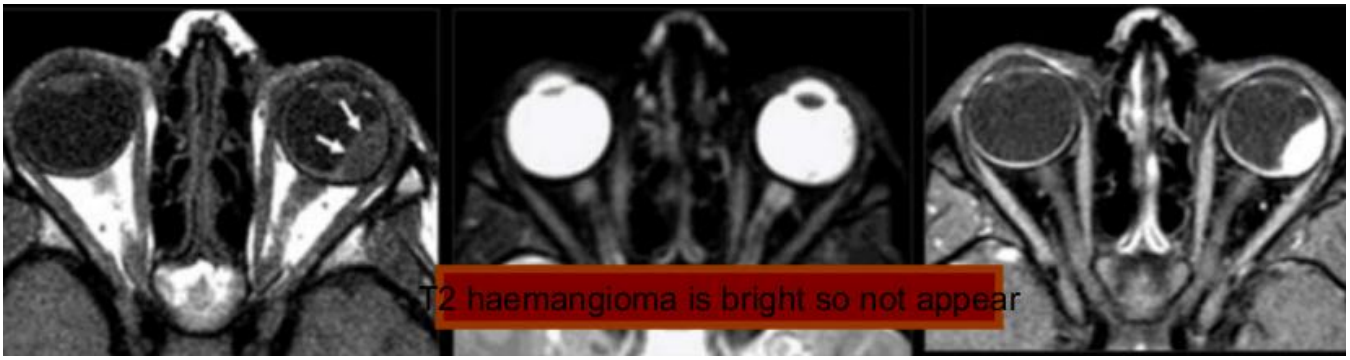
** ADJASCENT TO MACULA

+/-Von Hipple Lindu syndrome.

➔ **CT** ** Strong Enhancing

* HyperDense * JuxtaMacular Lesion

➔ **MR** **Strong Enhancing * Hi T1&T2



< 5 > TRAUMA

NB. Wood has Low density as Air

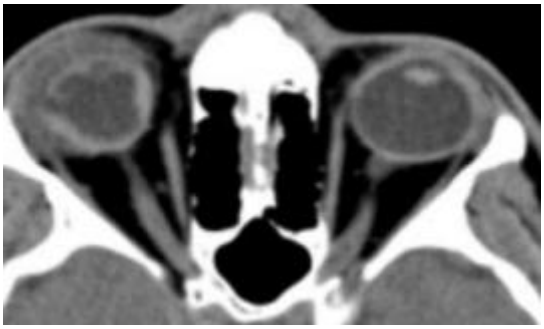
➔***Signs of Rupture Globe:***

a. Flat Tyer apperance

b. Thickened post. Sclera

c. Hazy inner sclera margin

d.I.O. Air (Sure sign)



II- OPTIC NERVE LESIONS

1 . Meningioma	2.Glioma
3 . Deposits	4.Others

IS this mass of OPTIC N?

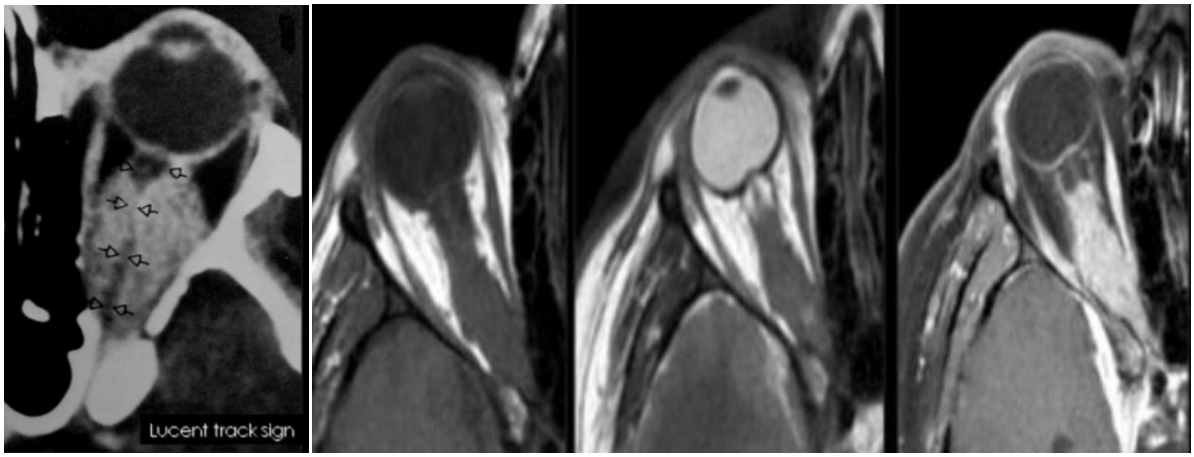
- Lesion must encase it on both sides either You see the nerve through lesion or not.
- Best seen by Coronal Scan

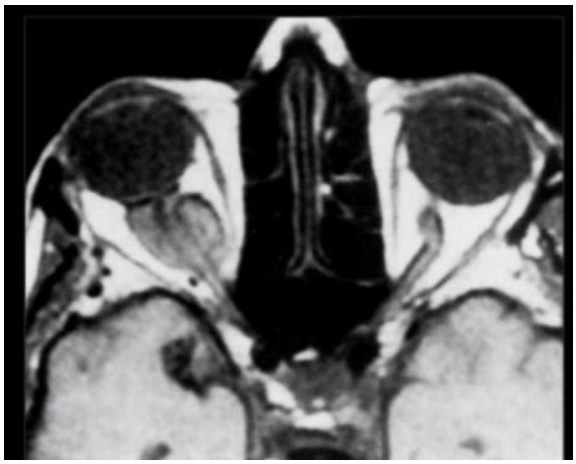
< 1 > MENINGIOMA

- Source : Optic N Sheath - Orbital periosteam
- F>M * 3 rd : 5th Decad * May oocur in Child e NeuroFibromatosis
- Incidence: 5 % of 1ry Orbital Tumors
- Extension : may be to parasellar region
- +/- Ca or Bone sclerotic
  ➔ CT
- Homogenous enhancing , Tubular thickening
- ** LUCENT TRACK SIGN
- -/+ Ca *+/-Extension * -/+ HyperOsteosis

➔ MRI :

- Nerve seen in Lesion
- Variable signal
- Homogenous enhancing



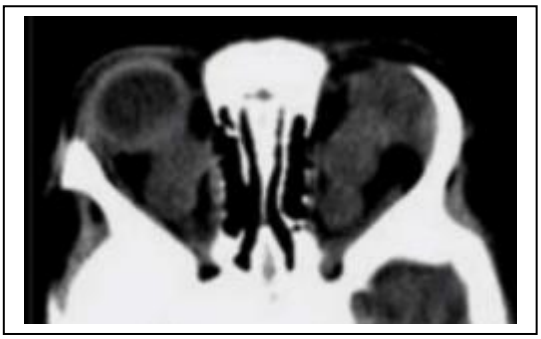


< 2 > OPTIC N GLIOMA

- Low grade Pilocytic Astrocytoma
- 90 < % 20 y.
- -Unilateral or Bilateral esp. with neurofibromatosis
- +/- cranial extension.
- Not extend in Globe.

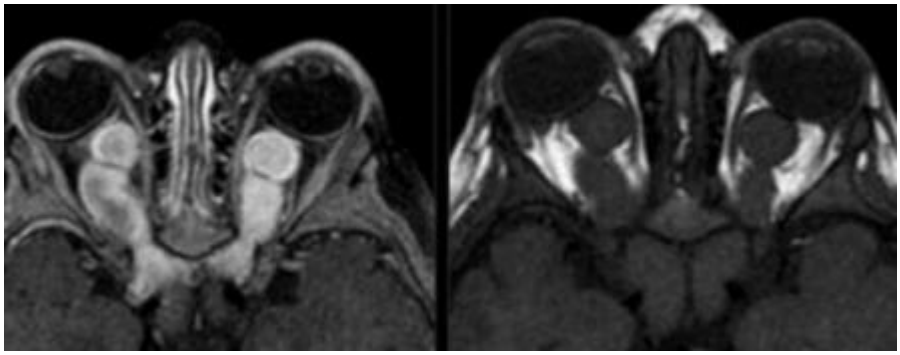
➔ **CT** optic nerve is

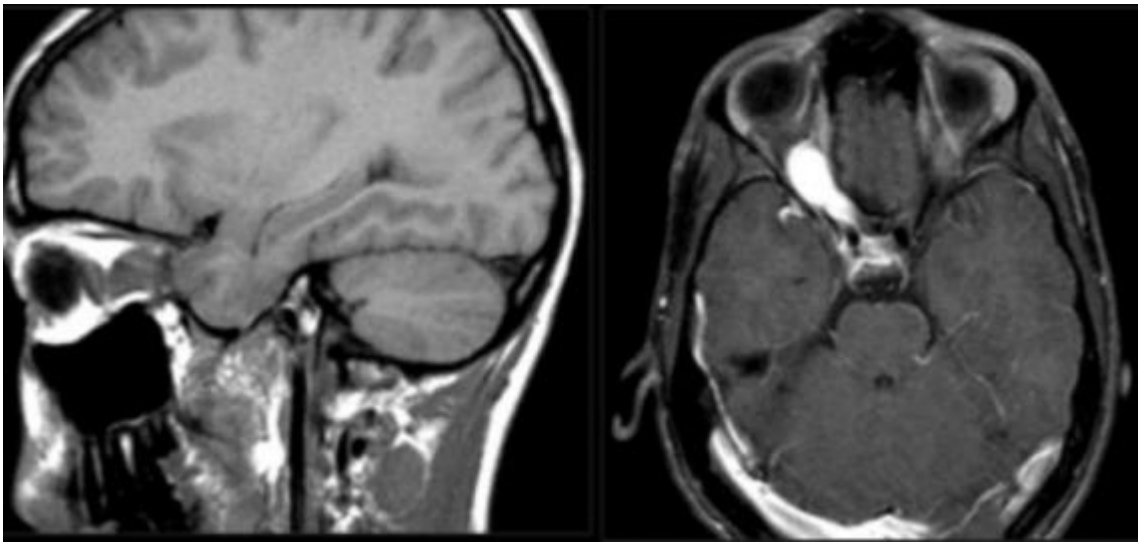
- Diffuse Thickened
- ***Tortuous
- Nerve is not differentiate from lesion

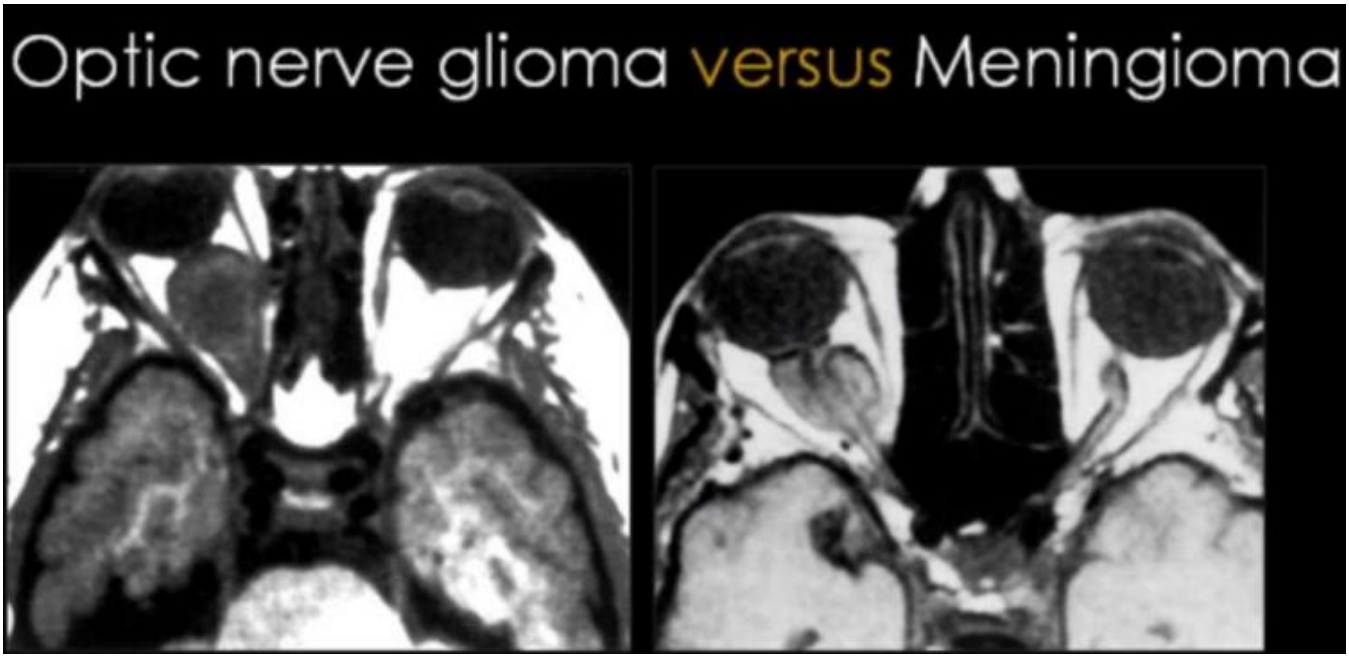


➔ MRI :

- *Lo T1 / Hi T2
- *Homo enhancing *Hetrogenous if Large







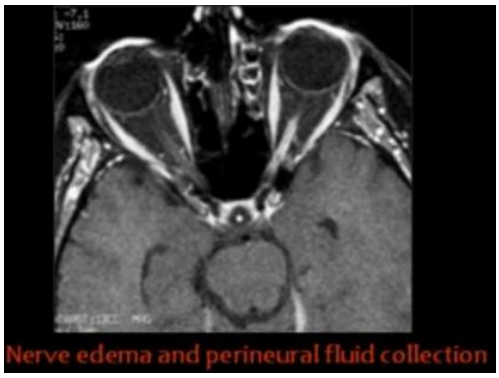
< 3 > OPTIC N NURITIS

-Up to 50% pt e Multiple Sclerosis.

-Dignosed Clinical

-Imaging Role to see:

- causeas MS.
- Nerve edema
- & perineuraL collection



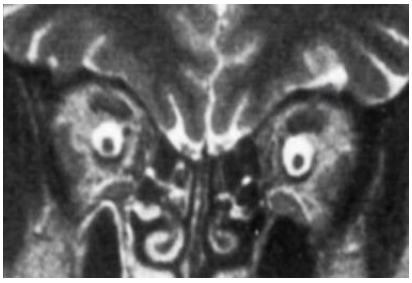
*Optic N Enhance but CALEBER NOT CHANGE.

Enhancement without mass = neuritis

(DILATED CSF SPACE ARROUND Optic N)

-Normal Variant

-MR best show it.



((3.LACRIMAL GLANDS))

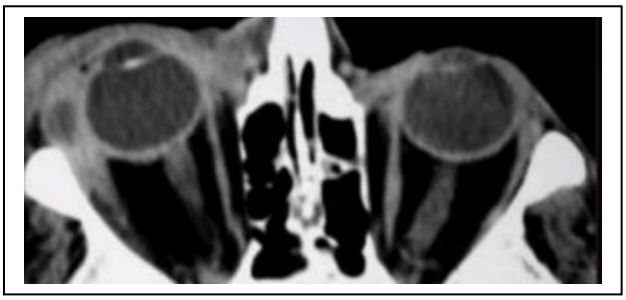
I-UNi Lateral II-Bi LateraL

I - UNILATERAL Lesions

- InfLamatory 50%
- NeopLastic 50%
- Both may not differentiate by imaging, THIS is YOUR LIMIT.

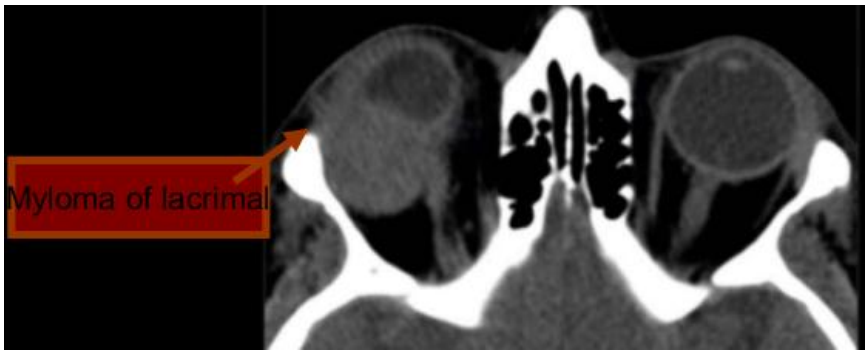
A-INFLAMATORY Lesion:

- Acute or Chronic **Dacryo-adenitis**
- Enhancing : Hetrogenous or Ring



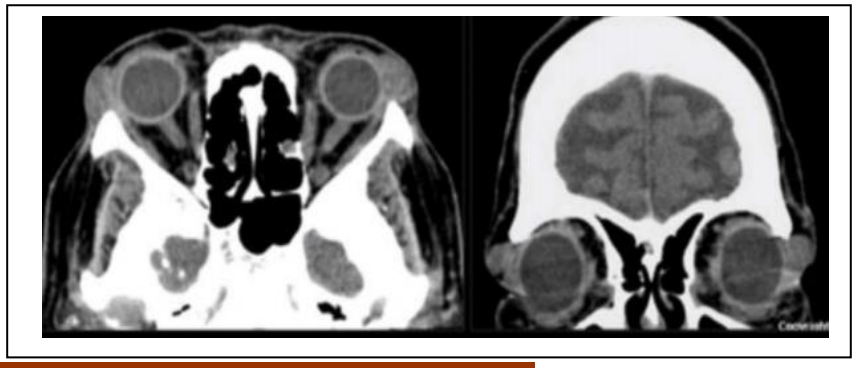
B-NEOPLASTIC:

- **Benign :** Adenoma, Dermoid
- **Malignant :** Carcinoma ,Lymphoma ,Deposit.
  - ⇨ Criteria of Malignancy:
    - Bone erosion is clue
    - +/- I.C. invasion
- No specific Enhancement
- With known primary → Lesion may be **deposit**

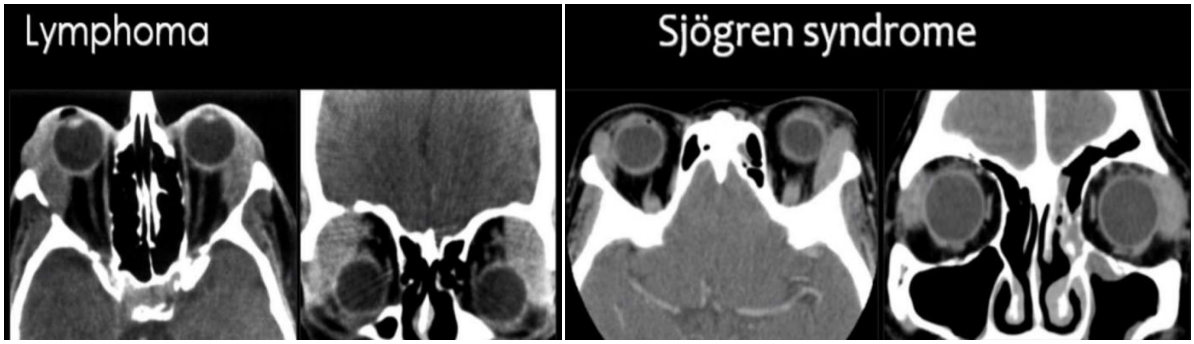


<II>BILATERAL LACRIMAL LESIONS:

Manifestation of systemic disease



DD لن نشخص ولكن نصف فقط ونكتب كل ما سبق.



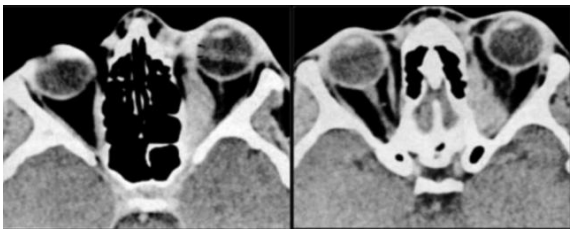
(4.EXTRA OCCULAR MUSCLE LESIONS)

A.SINGLE MUSCLE

1-Inflamatory 50% 2-Neoplastic 50%

➔ **Lateral** is comments to be affected singly.

(I).INFLAMATORY



-**Ethmoids** main source of orbit infection.

so when opaque+Ms les➔ Suggest Inflammatory.

- MYOSITIS PSEUDOTUMOR

-Single Ms esp LateraL -Clincal

+ Respond to Steroid

(II).NEOPLASTIC:

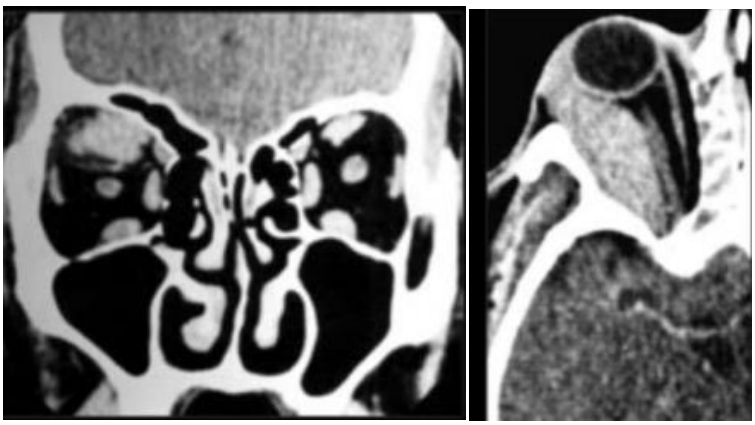
-1ry or 2ndry

- RhabdoMyoSarcoma -Usually ChiLd.
- Lymphoma -May affect multiple muscles esp superior

-D.D. Grave's

*__Signs suggest Malignancy__ : -Thick Ms + bone Erosion

*2rys may be: Local or blood extension.



B.MULTIPLE MUSCLE

-GRAVE's

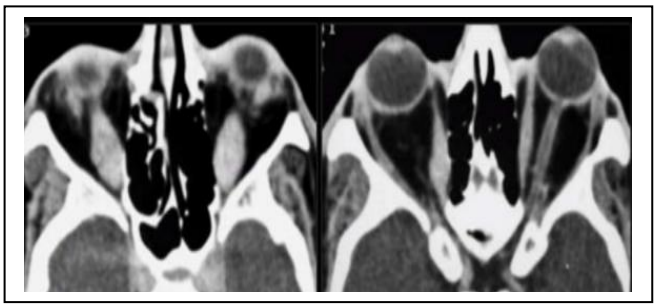
-Others

<<I>>GRAVE's DISEASE

Or Dytrophic Orbitopathy

Commonest cause of Unilateral Proptosis in Adult

*It can affect All orbital contents:



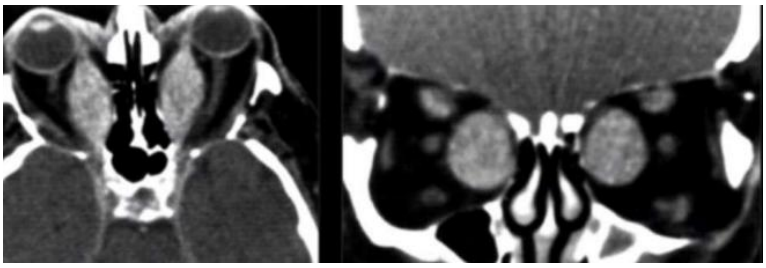
- Fat → increse volume +/- dirty fat
- Lacrimal Gland→ Swelling
- Optic Neuropathy
- MUSCLEs affection: -Unilateral or Bi

-When involve Multiple Muscle

→ commonest are ***Inferior** & ***Medial** **Rectus** > Superior >Least is Lateral

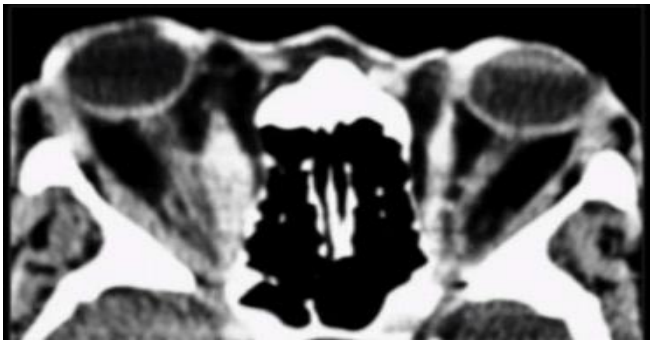
-Involve **muscles bulk only** not tendon→Fusiform shape

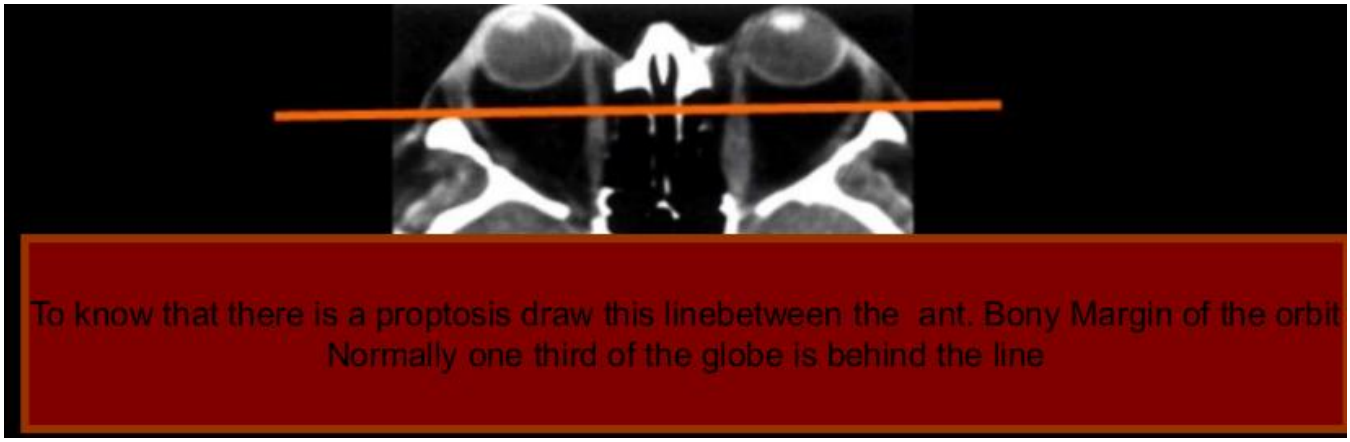
-Strong enhancement

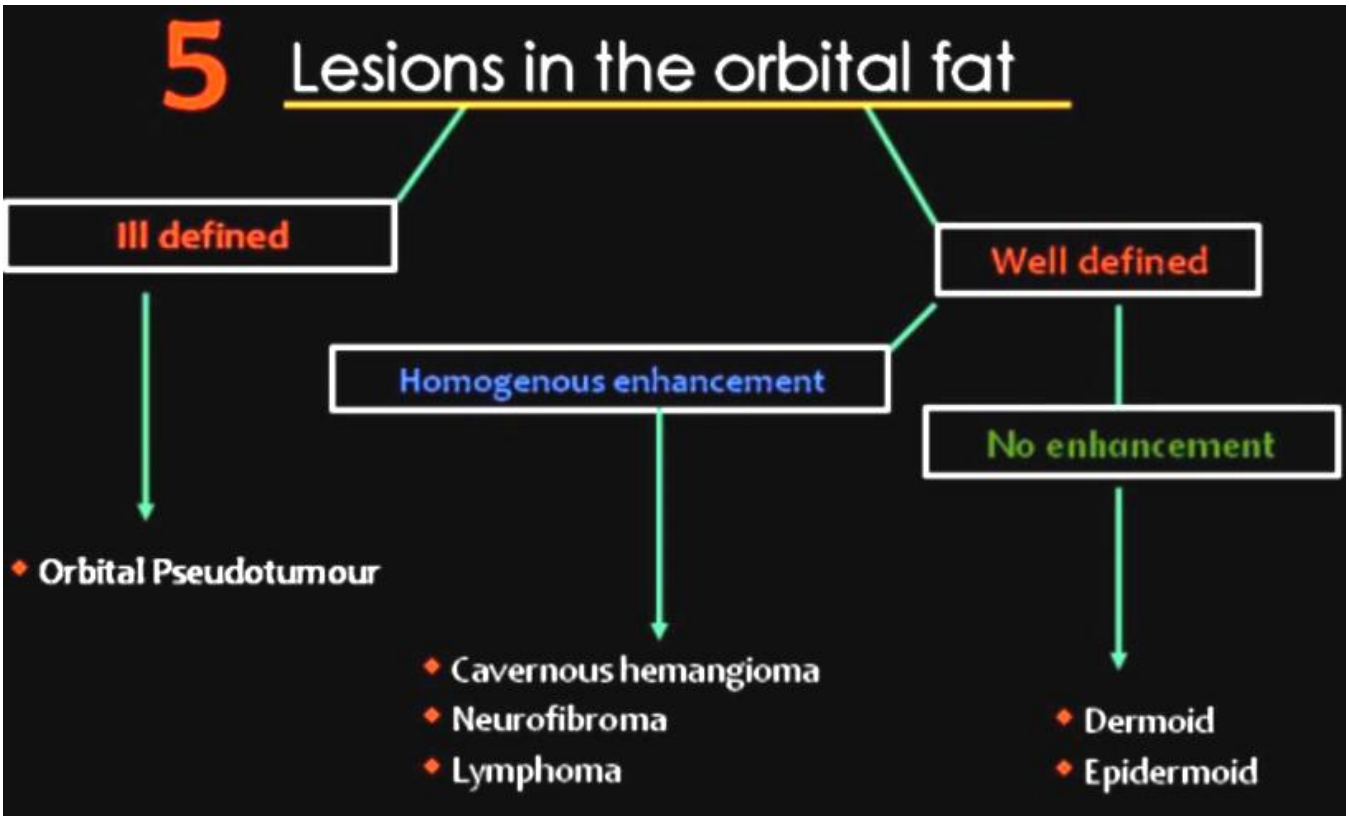


<II>ACROMEGALY

-Moderate Ms enlarge -Less proptosis +Other clinical FEATUREs




To know that there is a proptosis draw this linebetween the ant. Bony Margin of the orbit
Normally one third of the globe is behind the line


5 Lesions in the orbital fat
Ill defined
Well defined
Homogenous enhancement
No enhancement
Orbital Pseudotumour
Cavernous hemangioma
Neurofibroma
Lymphoma
Dermoid
Epidermoid

(((5.FAT LESIONs)))

- When lesion Not arias from any of above→ It is from FAT.
- it may Be:

I.ILL DEFINED

=Orbital PsudoTumor

II.WELL DEFINED:

@No Enhancement :

=Dermoid or epidermoid

@**Homogenous Enhancement** :

=Cav.Hemangioma =NeuroFibroma =Lymphoma

<1>ORBITAL PSEUDOTUMOR

-**Etiology:** Unknown

-Reactive inflammatory

-**Unilateral** usually

-DIAGNOSED .Clinical + -ttt steroids or RADIO Thereby .

-Affect Fat +/-Other structures

=Imaging:

-Detect Affected structures

-& For Follow up

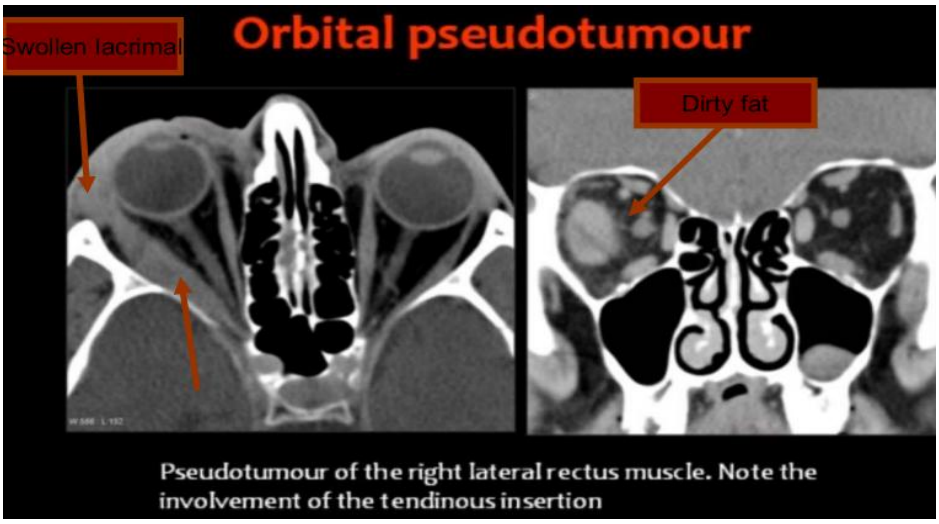
➔ CT:

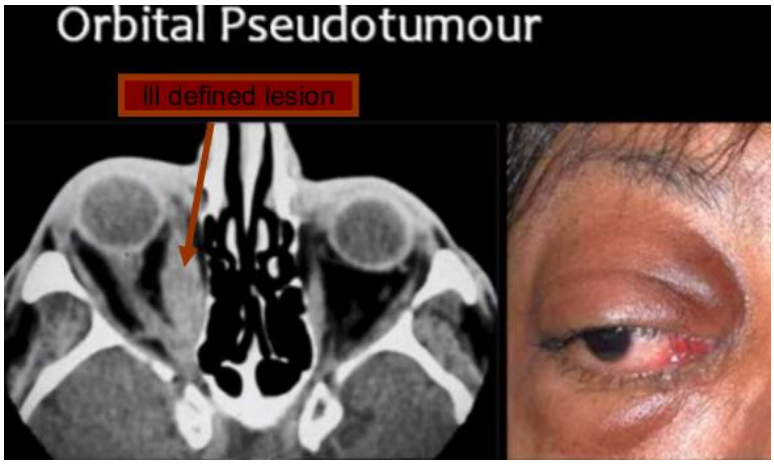
*Ill defined. *IrreguLar

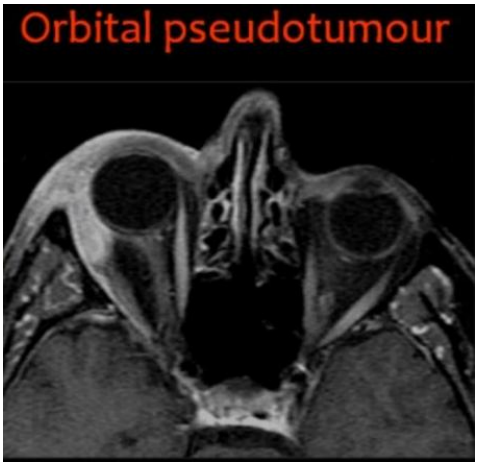
*Dirty Fat

+/-*Thick enhancing post. Sclera

*Ms & Lacr. Glands affection







<>TOLOSA HUNT Synd.
-Variant of PseudoT.
-Idiopathic
-InfL.Process
-from infL.CAV.SINUS
-ttt Steroids
=CT:
*PainfuLL Proptosis
*EnLarg Cav.Sinus
*Enh prepontine cister
*OrbitaL Apex abnormaL soft tissue.

WELL DEFINED
HOMOGENOUS ENHANCING LESIONS

1.CAVERNOUS HEMANGIOMA

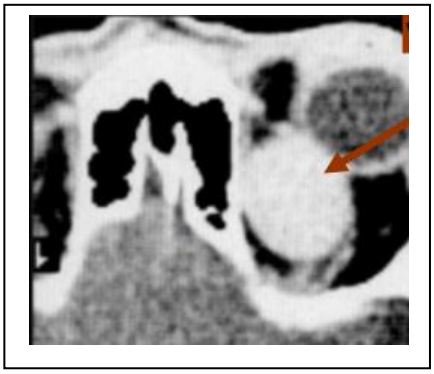
-Common in Adult common

-Single

-Round or ovaL

-Strong Homo enh

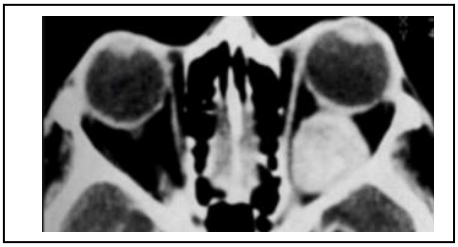
-/+ Ca phleboli



<2>NEUROFIBROMA

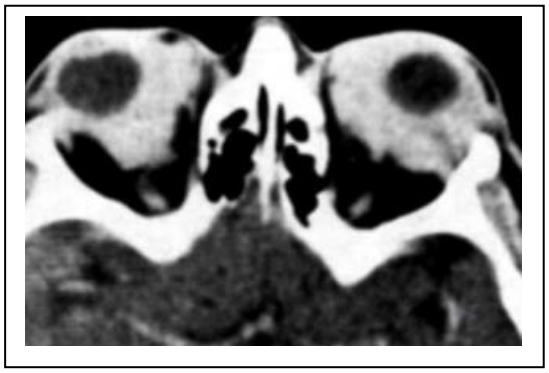
-Adult -Benign -Single

-Enhancing less than < Hemangioma



NeuroFIBROMA not arise from optic nerve
as it has not aneurolemall sheath but
It arise from other prephral nerve
as infraorbital nerve
&cannot diffrentiate from hemangioma
Even by MRI SO diagnose it as hemangioma
As it is the comonest

<3>LYMPHOMA



-Midle age Usually -/+Other Lymphomas

-Multiple > single

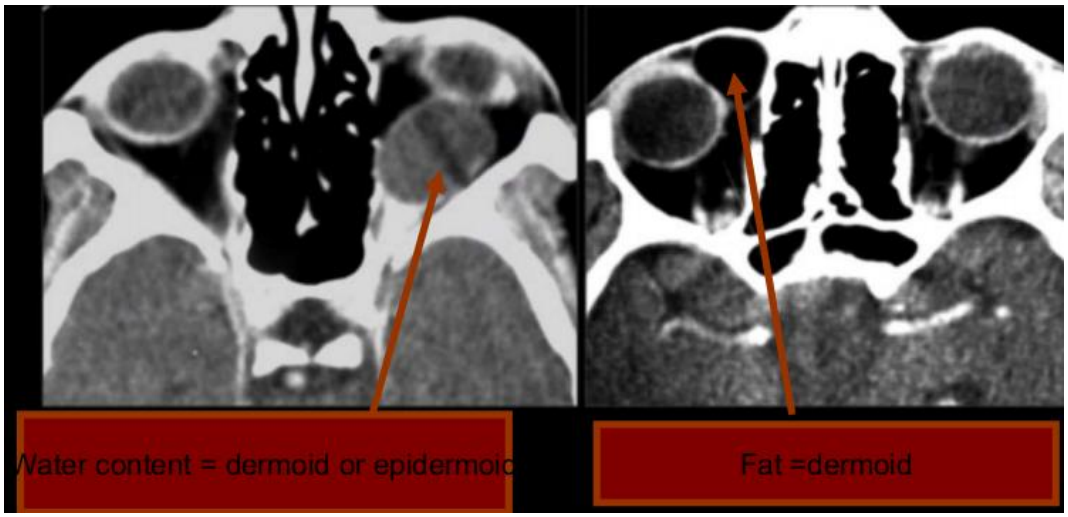
-Tend TO COAT GLOBE

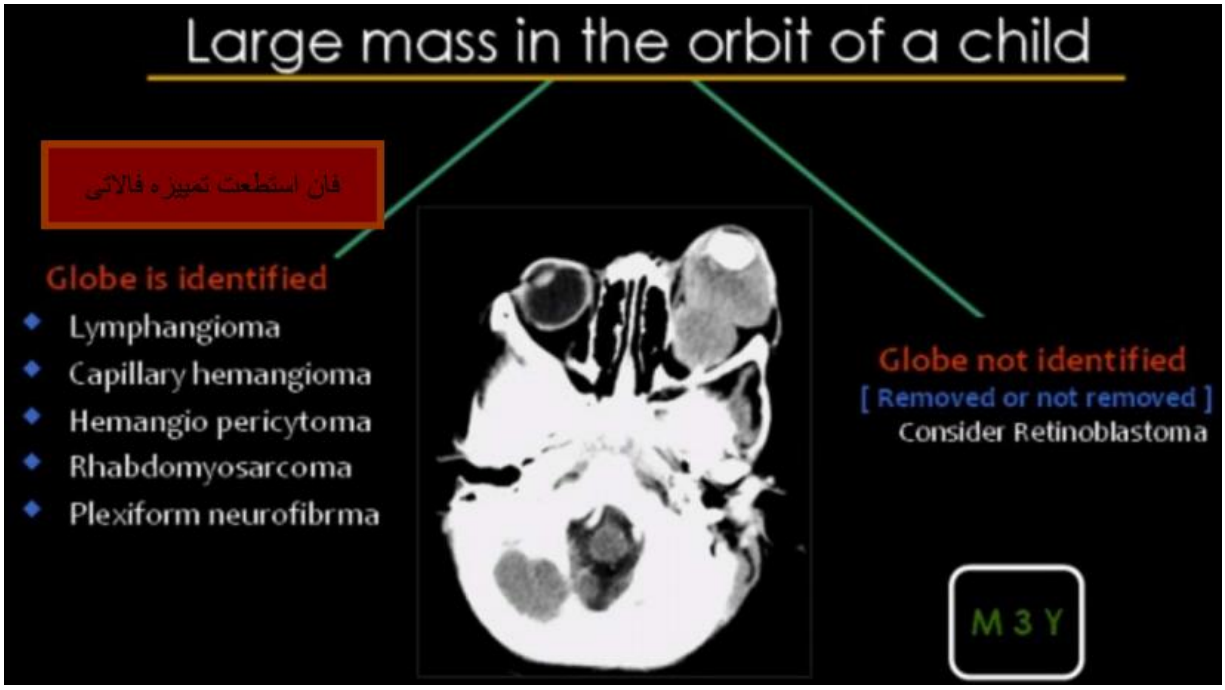
-Bone distruction → Hi Malignant

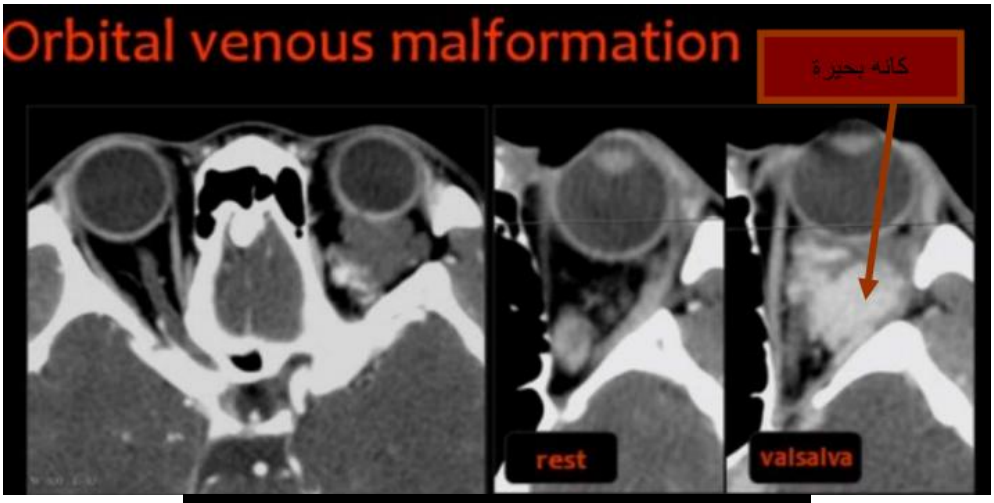
Criteria Suggesting Lymphoma

-Diagnosed Patient -Middle age - multiple Lesions

-Homo well defined encasing globe

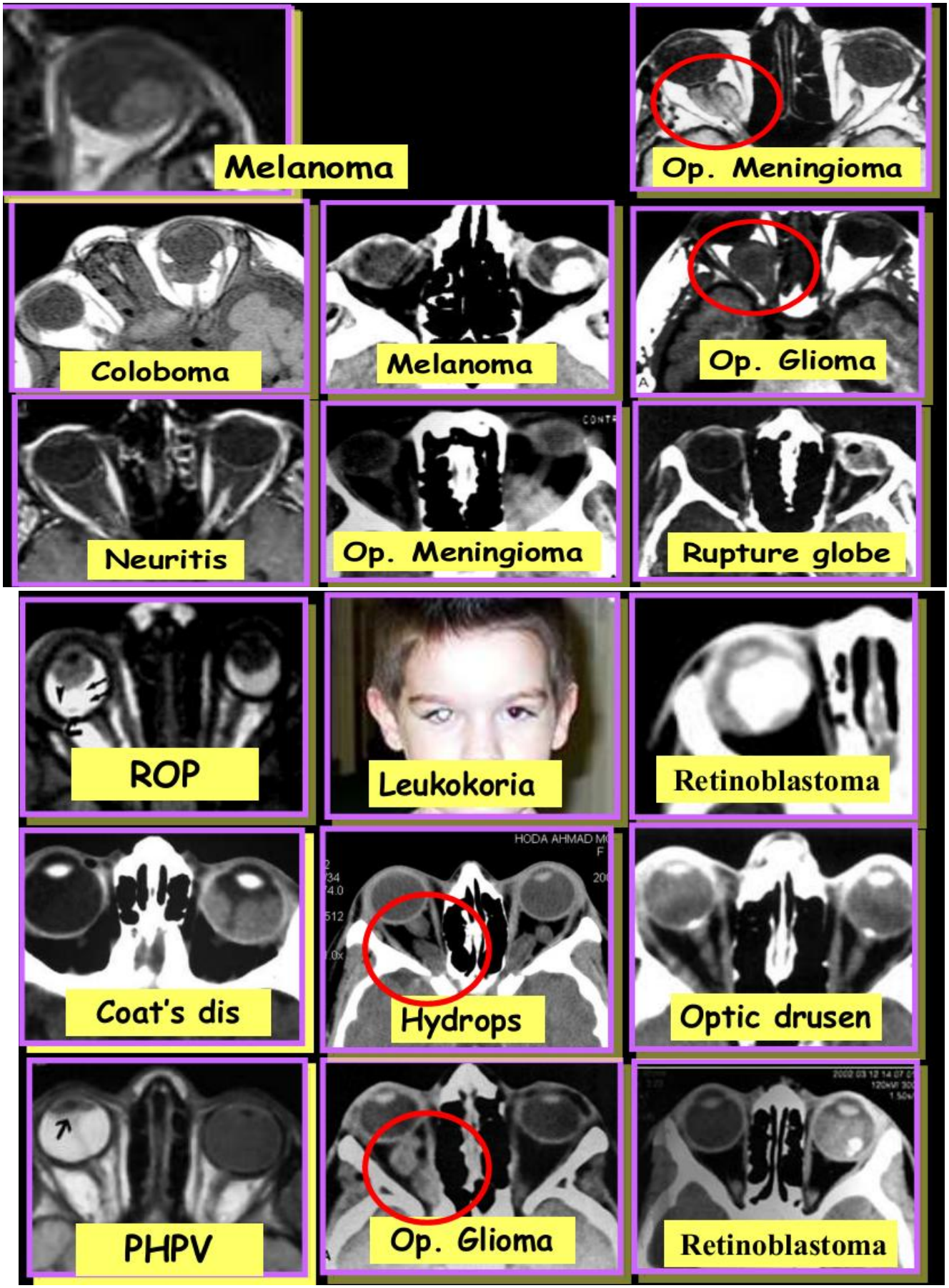






Vascular orbital lesion

- Cavernous hemangioma
- Capillary hemangioma
- Carotid cavernous fistula
- Orbital arteriovenous malformation
- Orbital varix
- Orbital venous malformation
- Melanotic melanoma
- Hemangiopericytoma
- Hyper vascular deposits


Melanoma
Op. Meningioma
Coloboma
Melanoma
Op. Glioma
Neuritis
Op. Meningioma
Rupture globe
ROP
Leukokoria
Retinoblastoma
Coat's dis
Hydrops
Optic drusen
PHPV
Op. Glioma
Retinoblastoma

Sources:

Lecture of Prof.Mamdouh Mahfouz

Dr. Mohammed Fargally PDF edit of lecture