Clinical Ocular Grand Rounds

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Disclosures

Paid consultant for:

Dompe: Honoraria- Advisory Board

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Sun Pharaceuticals: Speakers Bureau

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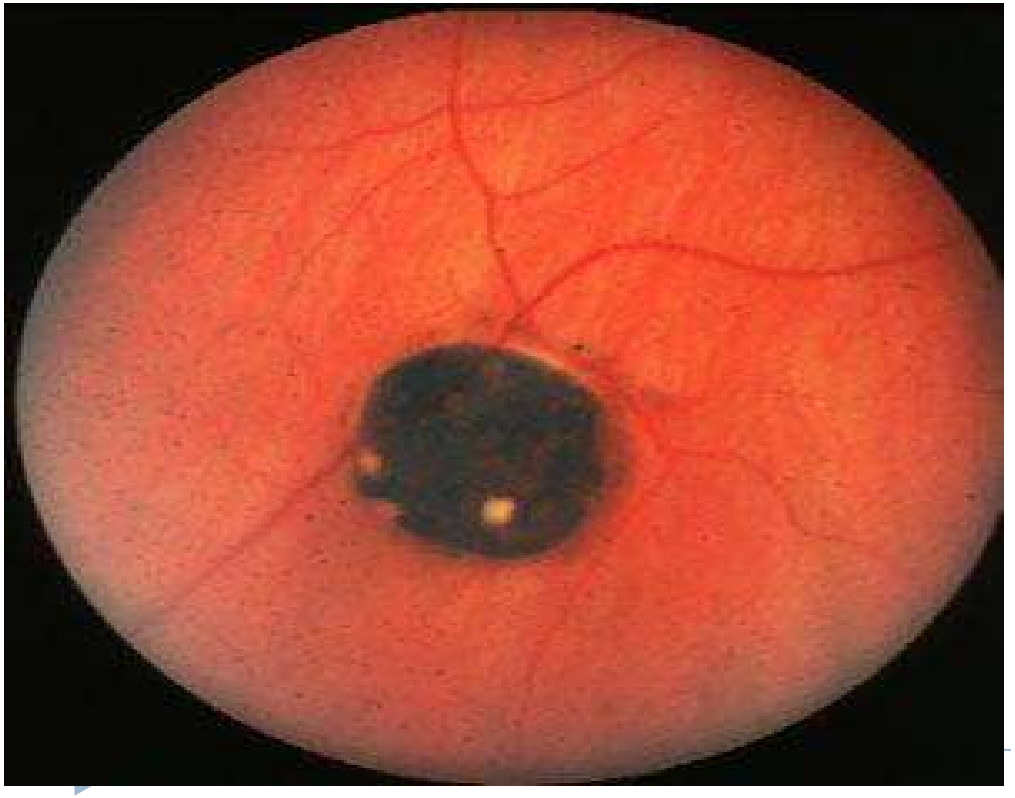
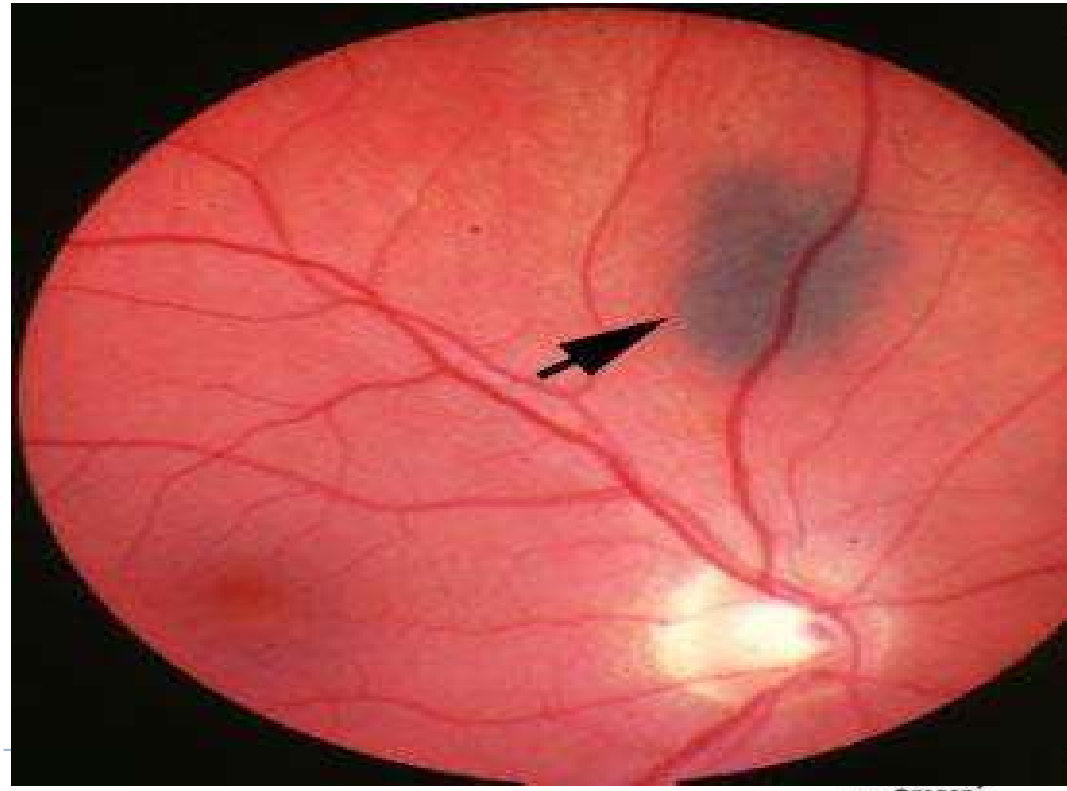
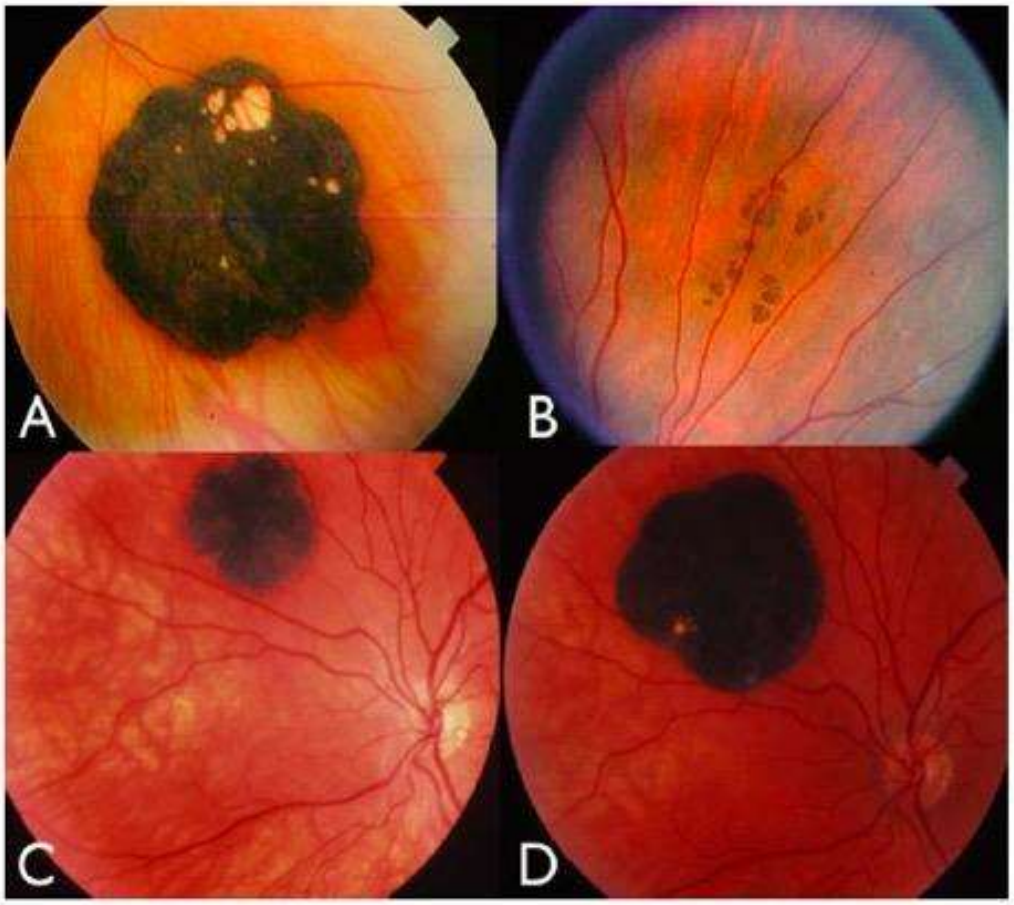
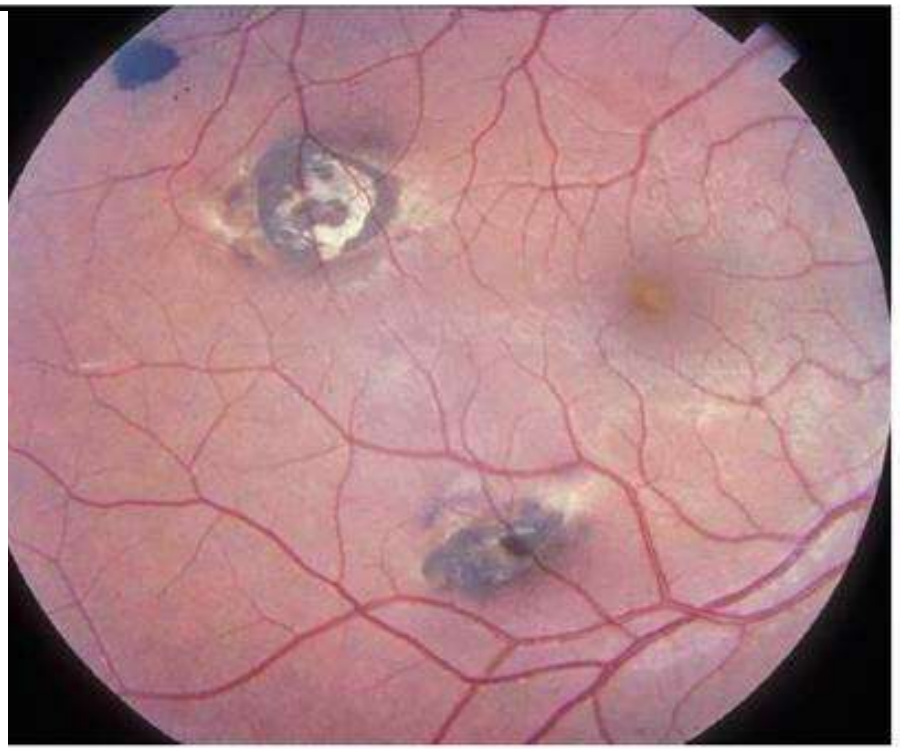
CHRPE vs Nevus





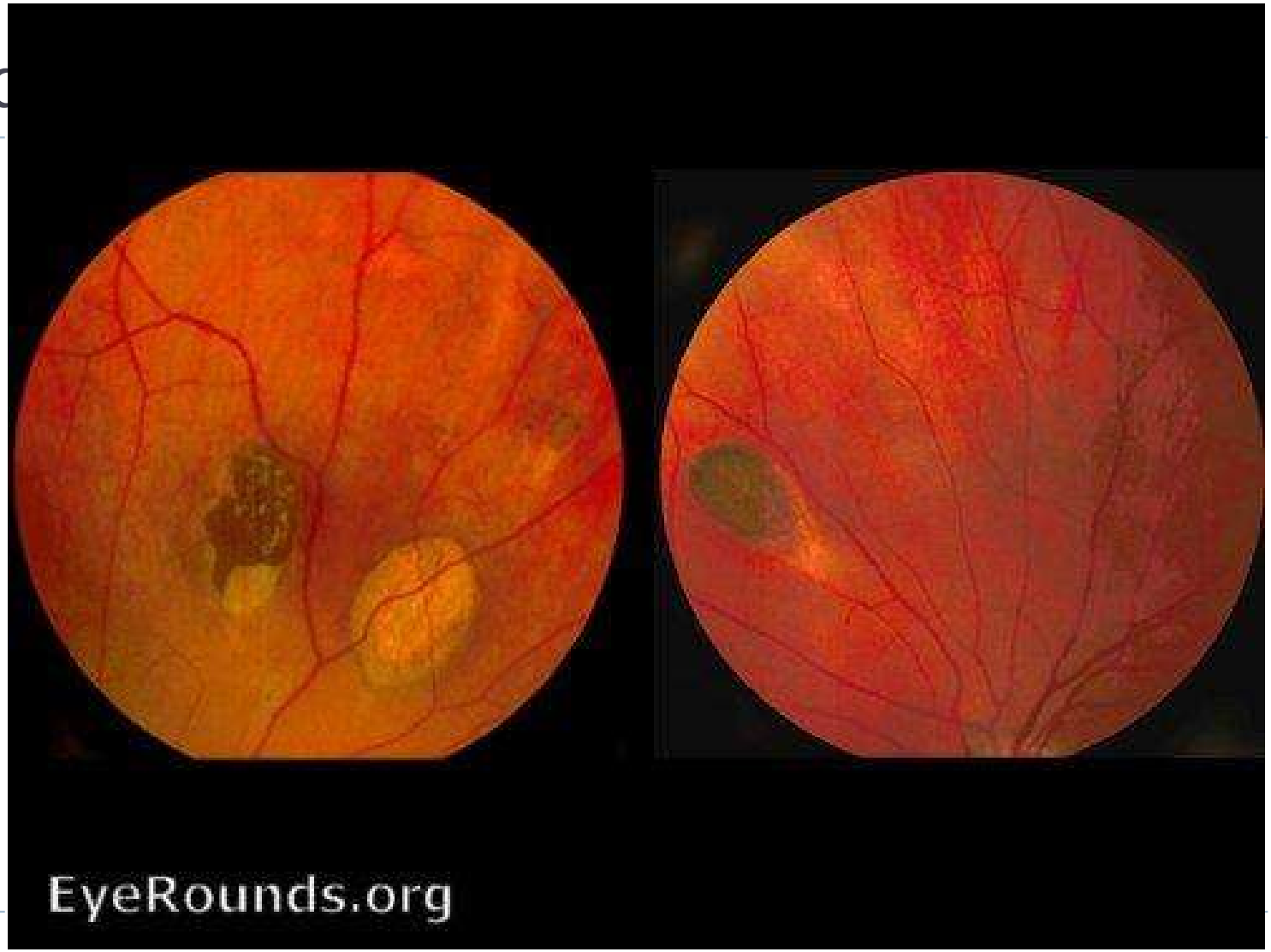
CHRPE vs Hamartomas





URE 4. Retinal pigment epithelial hamartomas (pigmented ocular
ndus lesions) associated with familial adenomatous polyposis
ardner syndrome).

Garc



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Nevi Trivia

- 31% of choroidal nevi show slight enlargement over time without the transformation to a melanoma (Ophthalmology 2011)
- The prevalence of choroidal nevi in the white U.S. population ranges from 4.6% to 7.9%
  - If it is assumed that all choroidal melanomas arise from preexisting nevi, then the published data suggest a low rate (1/8845) of malignant transformation of a choroidal nevus in the U.S. white population. (Ophthalmology 2005)
- Choroidal melanoma risk for metastasis, ranging from 16% to 53% (at 5 years of follow-up) depending on the size of the tumor at the time of diagnosis. (Arch Ophthalmol 1992)

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TFSOM—"To Find Small Ocular Melanoma"

Thickness: lesions >2mm
Fluid: any subretinal fluid (suggestive of serous retinal detachment)
Symptoms: photopsia, vision loss
Orange pigment overlying the lesion
Margin touching optic nerve head (<3mm)

- *None* of these factors = 3% risk of a nevus converting to melanoma in five years. *One* of these factors = 8% risk of conversion in five years. *Two* or more factors = 50% risk of conversion in five years. For any changes noted during the course of follow-up, refer the patient to a retinal practice or an ocular oncology service.

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TFSOM-UHHD:
"To Find Small Ocular Melanoma Using Helpful Hints Daily"

Thickness: lesions >2mm
Fluid: subretinal fluid
Symptoms: photopsia, vision loss
Orange pigment overlying the lesion
Margin touching optic nerve head (<3mm)
Ultrasound **H**ollowness
Halo absence
Drusen absence

- Choroidal nevi showing no features should be initially monitored twice yearly and followed up annually
- 1 or 2 features should be monitored every 4 to 6 months.
- Nevi with 3 or more features should be evaluated at an experienced center for management alternatives and possible treatment owing to the high risk of ultimate growth

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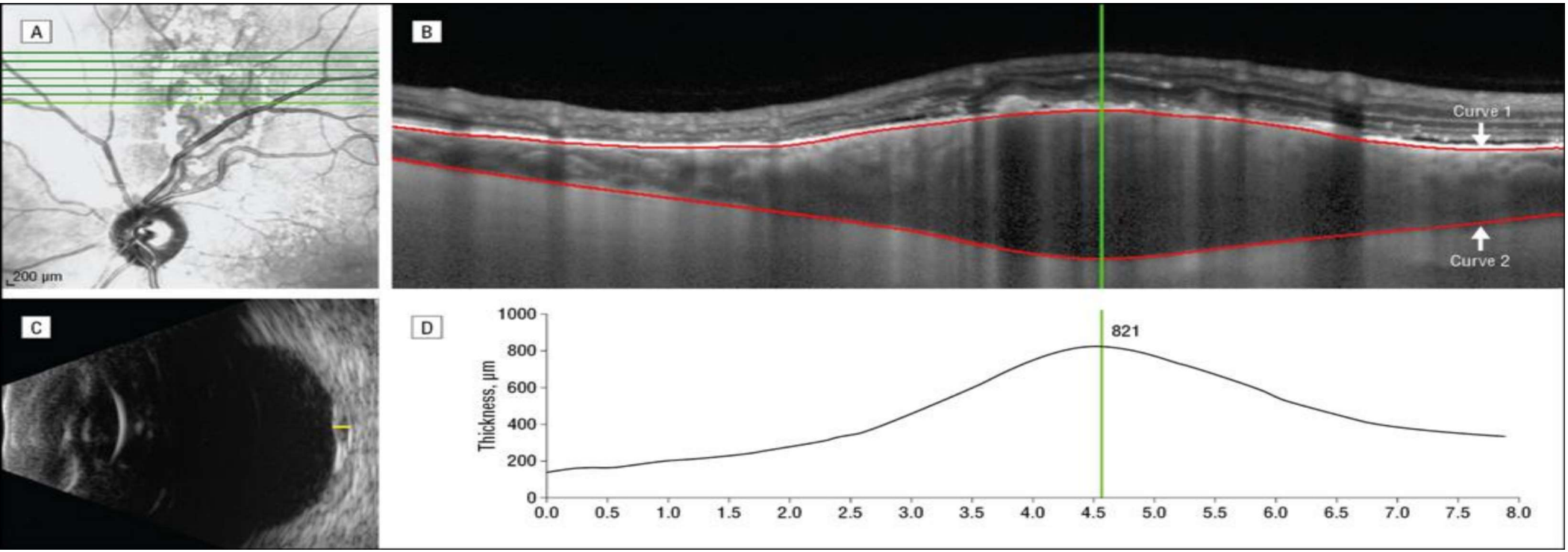
From: **Enhanced Depth Imaging Optical Coherence Tomography of Small Choroidal Melanoma: Comparison With Choroidal Nevus**

Arch Ophthalmol. 2012;130(7):850-856. doi:10.1001/archophthalmol.2012.1135



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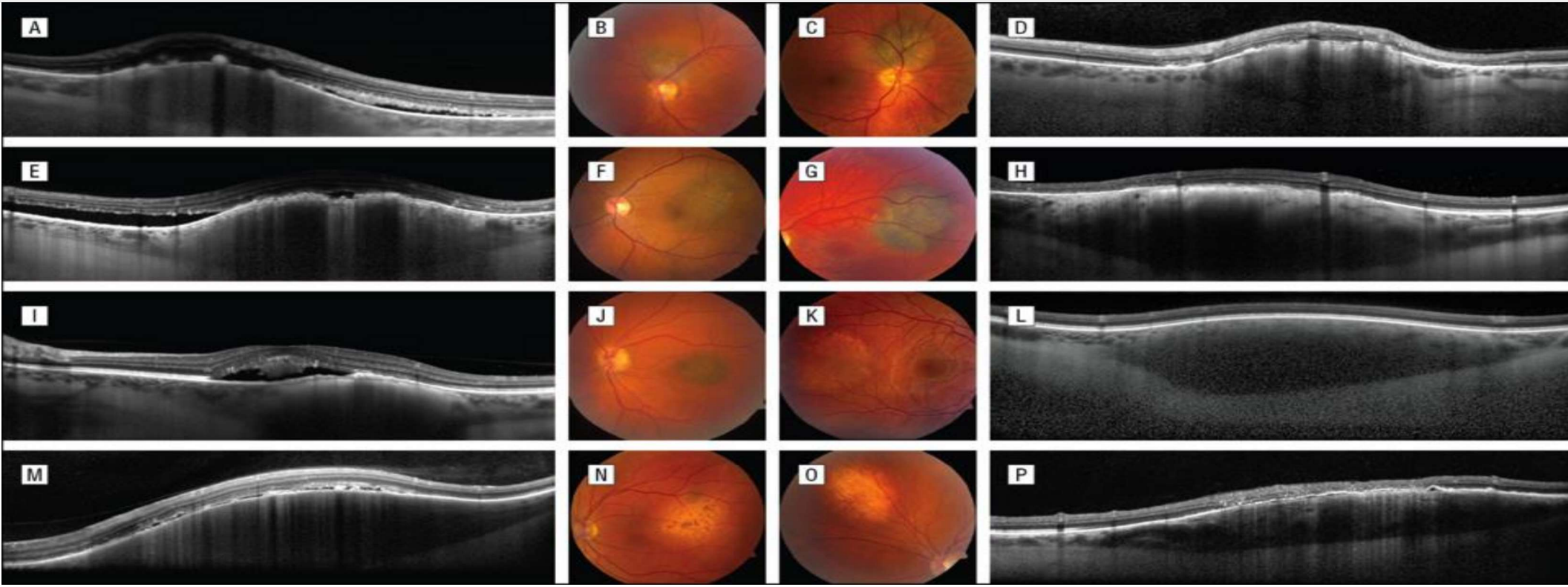
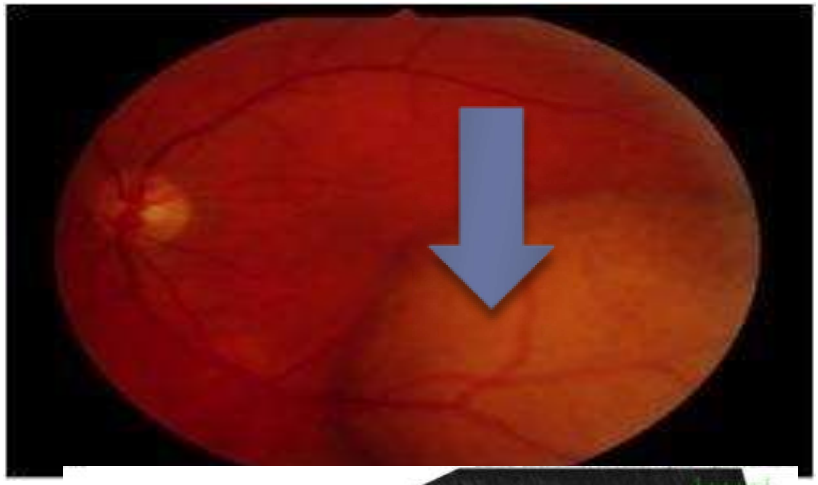
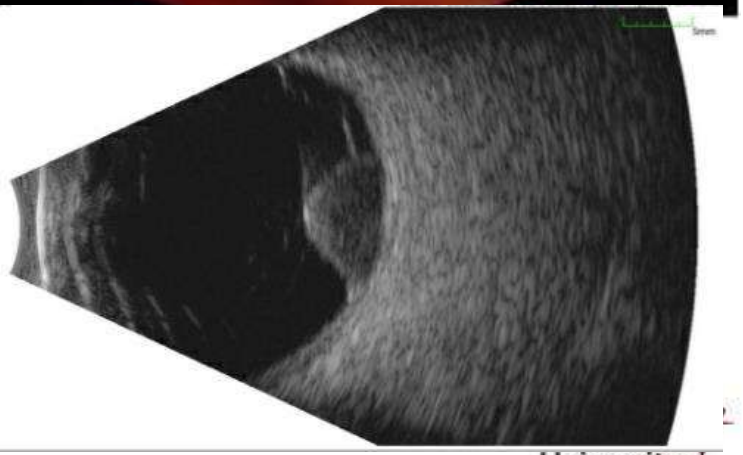


Case

- 65 yr old white male
  - Notices spot in vision in his left eye
  - Diabetes for 15 years
- Vision:20/20 (6/6) and 20/40 (6/12)
- Dilated exam:
  - Large lesion noted in left eye (not noted in exam 6 months previously
  - See photo and B-scan

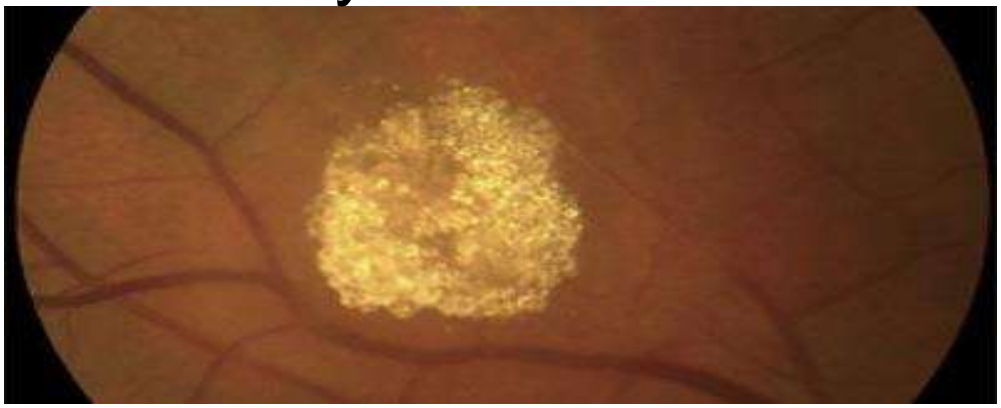




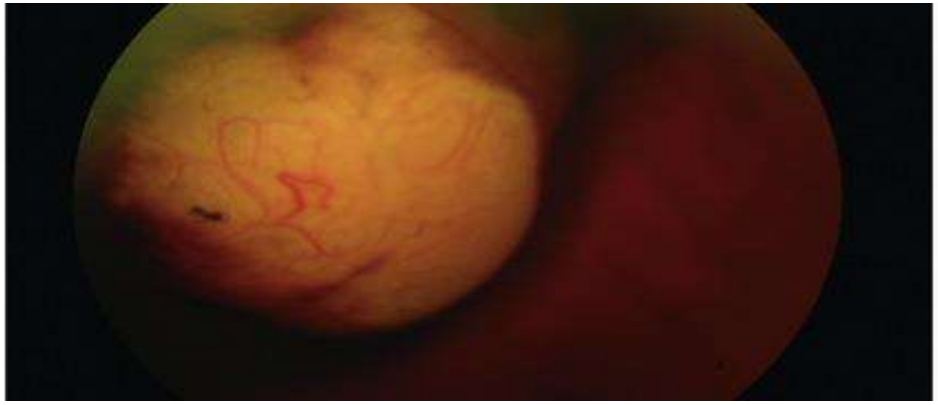
https://eyewiki.aao.org/File:Choroidal_melanoma_bscan.png

Ocular Tumors

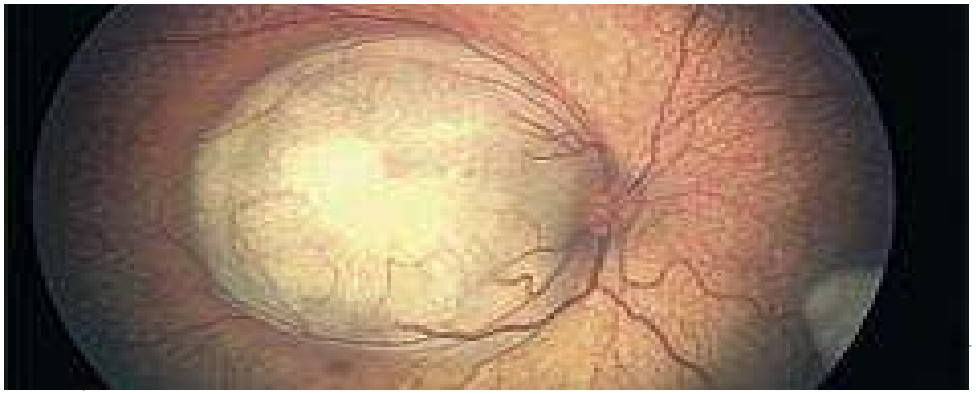
Astrocytic Hamartoma



Amelanotic Melanoma



Retinoblastoma



Metastatic Choroidal Tumor

Choroidal Melanoma Metastases

- 80 to 90% of metastases from uveal melanoma occurred in the liver, less common sites being the skin and lung.
  - Gragoudas ES, Seddon JM, Egan KM, et al. Long-term results of proton beam irradiated uveal melanomas. Ophthalmology. 1987;94:349–53.

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Melanoma and Mortality

- Tumor Size:
  - 5-year mortality after enucleation:
    - 16% for small melanoma,
    - 32% for medium melanoma, and
    - 53% for large melanoma.
  - the prognostic importance of tumor size:
    - each 1-mm increase in melanoma thickness adds approximately 5% increased risk for metastatic disease at 10 years
- Tumor genetics:
  - Chromosome monosomy 3 (apprx 50% of patients)
    - 50% of them develop metastasis within 5 years of diagnosis
    - 70% mortality within 4 years of ocular treatment
    - one of the most important independent risk factors of poor survival

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New Treatment for Choroidal Melanoma

- light-activated AU-011 agent represents the first potential new therapy for choroidal melanoma
- AU-011 is a viral nanoparticle conjugate delivered by intravitreal injection, which targets tumor cells in the choroid and then is activated by ophthalmic laser to disrupt the tumor cell membrane, leading to necrosis.
- Two year prospective study complete

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New Treatment for Choroidal Melanoma

- Total cohort of 36
  - 12 patients in the single-dose cohort demonstrated a modest tumor control rate of 67% with a follow-up period of 9 to 24 months, and
  - 22 patients in the multiple-dose cohort (2 patients lost to follow-up) demonstrated a modest tumor control rate of 77% with a follow-up period of 0.5 to 18 months.
  - Subjects treated with the maximum safe and tolerated dose (80 µg with 2 lasers) with 0.5 months to 6 months follow-up have a tumor control rate of 92% (13 of 14 subjects).
  - Vision was preserved in all patients at 3 months or longer up to 24 months.
- Phase 2 study was begun August 2020 and expected end date September 2024

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Eyelid Lumps and Bumps

- 15-20% of periocular skin lesions are malignant
- Benign vs malignant:
  - Benign lesions are:
    - Well circumscribed and possibly multiple
    - Slow growing
    - Less inflamed
    - Look "stuck on" instead of invasive and deep

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Is it Benign?

- H: loss of hair bearing structures?
- A: asymmetrical?
- A: abnormal blood vessels (telangectasia's)?
- B: borders irregular?
- B: bleeding reported?
- C: multicolored?
- C: change in the size or color of the lesion?
- D: overall diameter > 5 mm?

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Benign Eyelid Lesions

- Most common types of benign eyelid lesions include:
  - Squamous papillomas (skin tags)-most common
  - Hordeola/chalazia
  - Epidermal inclusion cysts
  - Seborrheic keratosis
  - Apocrine hidrocystoma
  - Capillary hemangioma (common vascular lesion of childhood)

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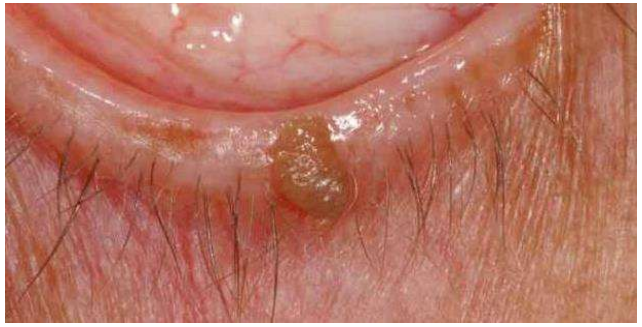
Benign Eyelid Lesions: Squamous Papilloma

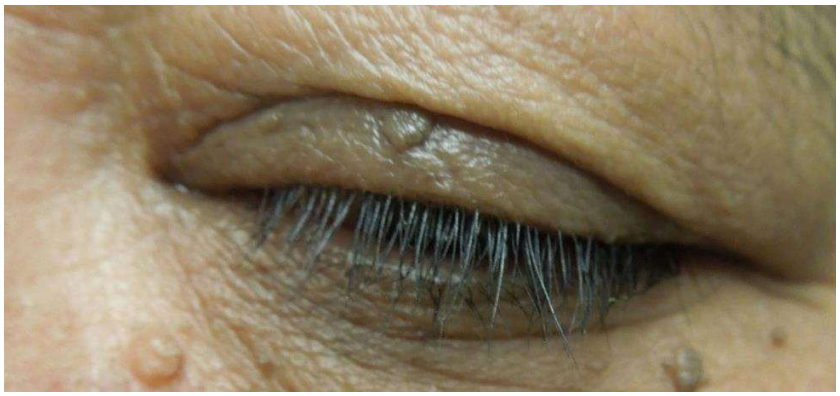
- Most common benign lesion of the eyelid
  - Also known as fibroepithelial polyp or skin tag
- Single or multiple and commonly involve eyelid margin

Benign Eyelid Lesions: Squamous Papilloma

- Flesh colored and maybe:
  - sessile (no stalk) or pedunculated (with a stalk)
- Differentials:
  - seborrheic keratosis,
  - verruca vulgaris and
  - intradermal nevus
- Treatment is excision at the base of the lesion.
  - Radiosurgery: Ellman
  - Cryotherapy
  - Chemical removal e.g TCA



https://www.willseye.org/disease_condition/eyelid-papilloma/



http://morancore.utah.edu/basic-ophthalmology-review/eyelid-masses/

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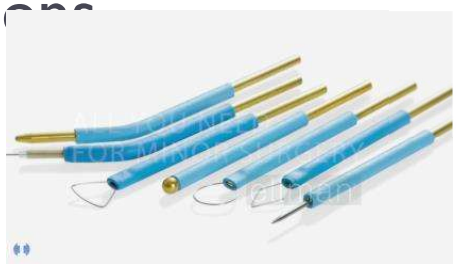
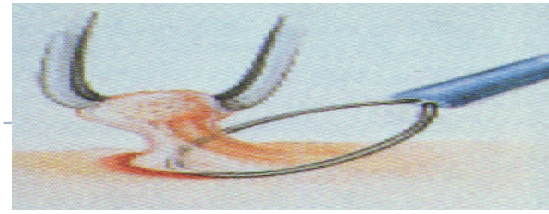
Radiofrequency (RF) Surgery

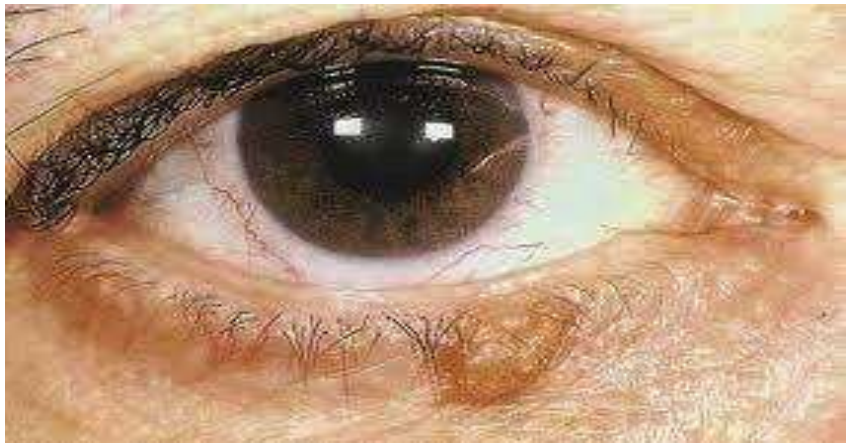
- Radiosurgery is the passage of high frequency radiowaves through soft tissue to cut, coagulate, and/or remove the target tissue
- Cuts and coagulates at the same time
- Nearly bloodless field
- Minimal biopsy artifact damage
- Quick and easy (to do and to learn)
  - Pressureless & bacteria-free incisions
- Minimal lateral heat
- Minimal Post-op pain
- Rapid healing
- Fine control with variety of tips

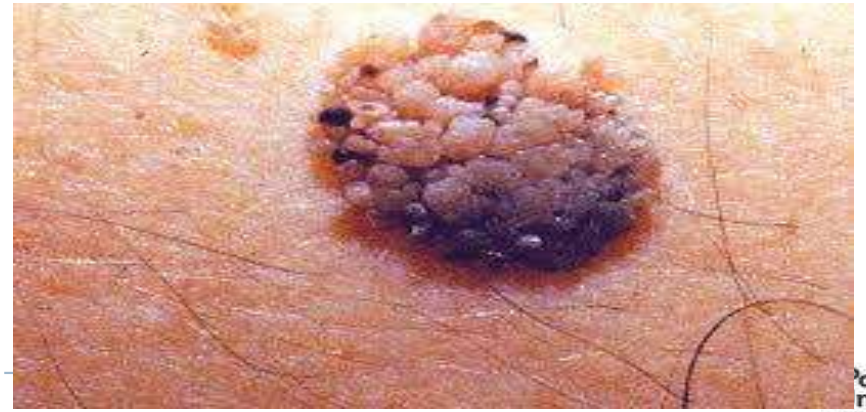




Benign Eyelid Lesions: Seborrheic Keratosis

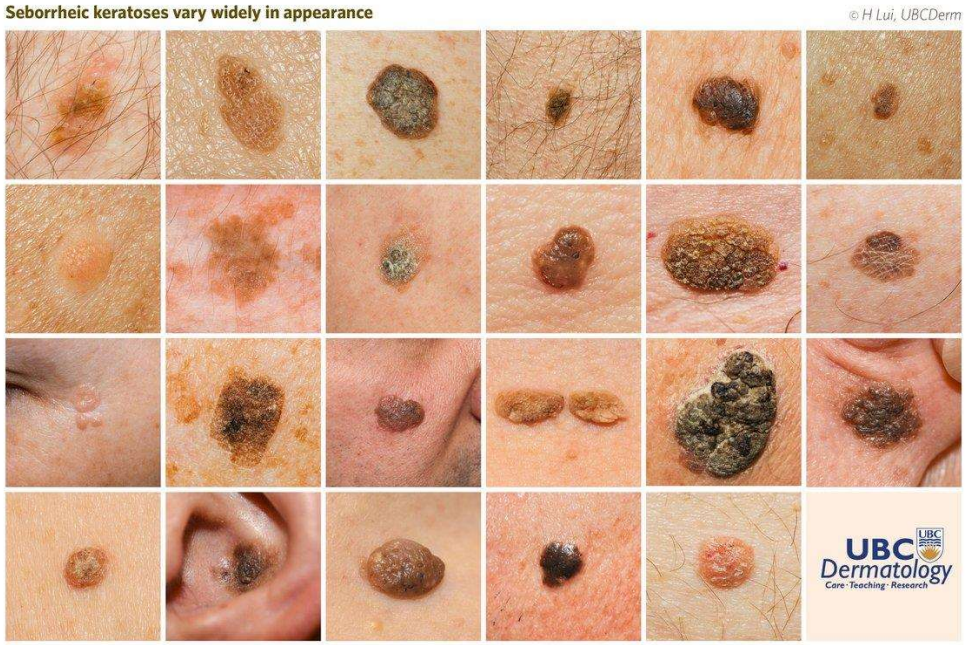
- Also known as senile verruca
- Common and may occur on the face, trunk and extremities
- Usually affect middle-aged and older adults, occurring singly or multiple, greasy, stuck on plaques





Benign Eyelid Lesions: Seborrheic Keratosis

- Color varies from tan to brown and are not considered pre-malignant lesions
- Differentials include skin tags, nevus, verruca vulgaris, actinic keratosis and pigmented BCC
- Simple excision for biopsy or cosmesis or to prevent irritation.



https://www.grepmed.com/images/3892/seborrheic-table-keratoses-dermatology-photo

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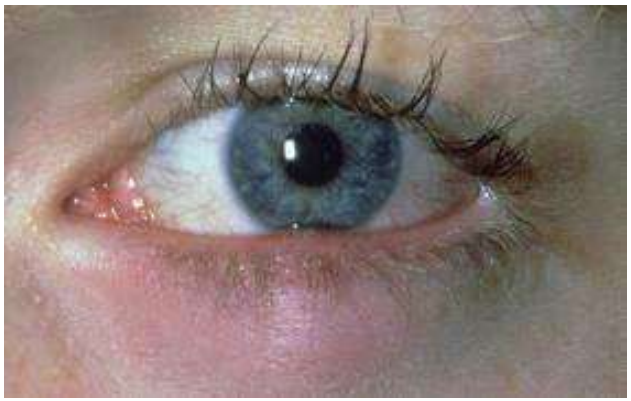
Benign Eyelid Lesions: Hordeola

- Acute purulent inflammation
  - Internal occurs due to obstruction of MG
  - External (stye) from infection of the follicle of a cilium and the adjacent glands of Zeiss or Moll
- Painful edema and erythema,



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Benign Eyelid Lesions: Hordeola

- Typically caused by Staph and often associated with blepharitis
- Treatment includes:
  - hot compresses (e.g. Bruder)
  - topical antibiotics (?)
  - possibly systemic antibiotics
    - Augmentin 875 mg BID x 7days
    - Keflex 500 mg TID-QID x 7 days
- Treat concurrent blepharitis

Demodex

- Demodex is a natural part of human microbiome
- *Demodex folliculorum* live in hair follicles, primarily on the face, as well as in the meibomian glands of the eyelids;
- *Demodex brevis* live in the sebaceous glands of the skin.

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Acne Rosacea and Demodex

- *Demodex folliculorum* frequently occur in greater numbers in those with rosacea and this overabundance is thought to trigger an immune response or possibly certain bacteria associated with the Demodex

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Benign Eyelid Lesions: Chalazia

- Focal inflammatory lesion resulting from obstruction of a meibomian or Zeis gland
- Results in a chronic lipogranulomatous inflammation

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Benign Eyelid Lesions: Chalazia

- May drain spontaneously or persist as a chronic nodule
- Recurrent lesions need to exclude a sebaceous gland carcinoma
- Treatment varies from:
  - hot compresses/massage,
  - intralesional steroid injection (triamcinolone (Kenalog^{R}) or
  - surgical drainage
  - **Latest**: IPL (Intense Pulsed Light)

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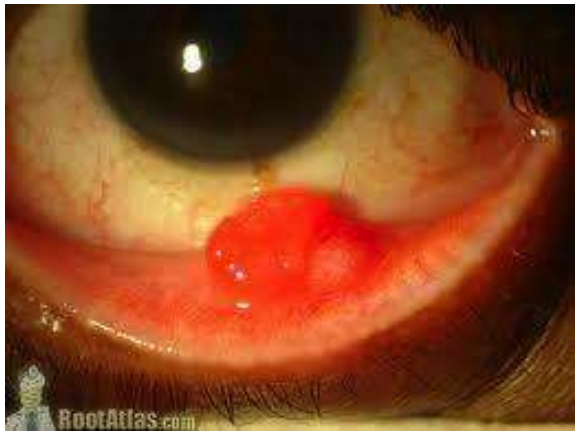
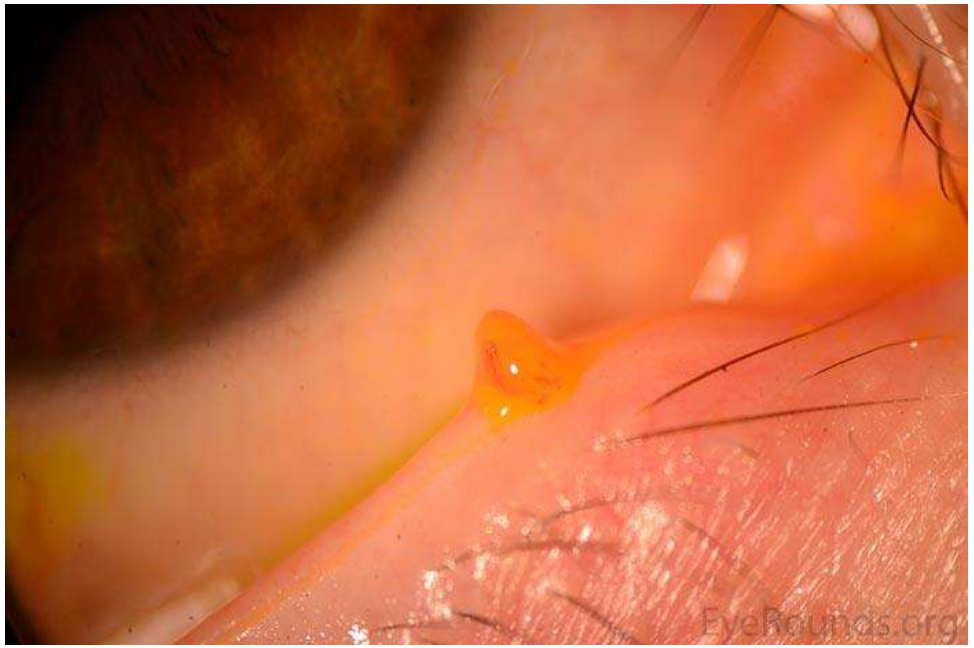
Benign Eyelid Lesions: Pyogenic Granuloma

- Most common acquired vascular lesion to involve the eyelids
- Usually occurs after trauma or surgery as a fast growing, fleshy, red-to-pink mass which readily bleeds with minor contact



Benign Eyelid Lesions: Pyogenic Granuloma

- Differential include Kaposi's sarcoma
- Treatment can include use of steroid to reduce the inflammation or surgical excision at the base of the lesion.



https://webeye.ophth.uiowa.edu/eyeforum/atlas/pages/pyogenic-granuloma/index.htm

Benign Eyelid Lesions: Epidermal Inclusion Cyst

- Appear as slow-growing, round, firm lesions of dermis or subcutaneous tissue
- Eyelid lesions are usually solitary, mobile and less than 1 cm
- Maybe congenital or may arise from trauma

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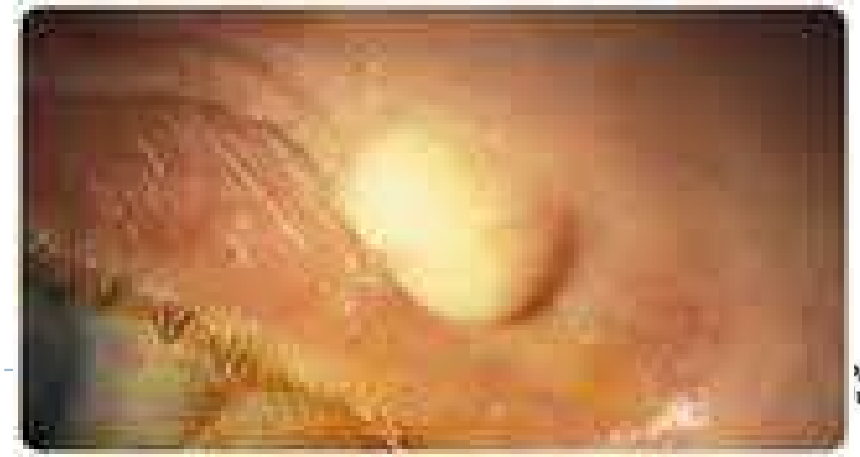
Benign Eyelid Lesions: Epidermal Inclusion Cyst

- May become infected or may rupture
- Differentials include:
  - dermoid cyst,
  - pillar cyst or
  - neurofibroma
- Treatment is complete excision to prevent recurrence.

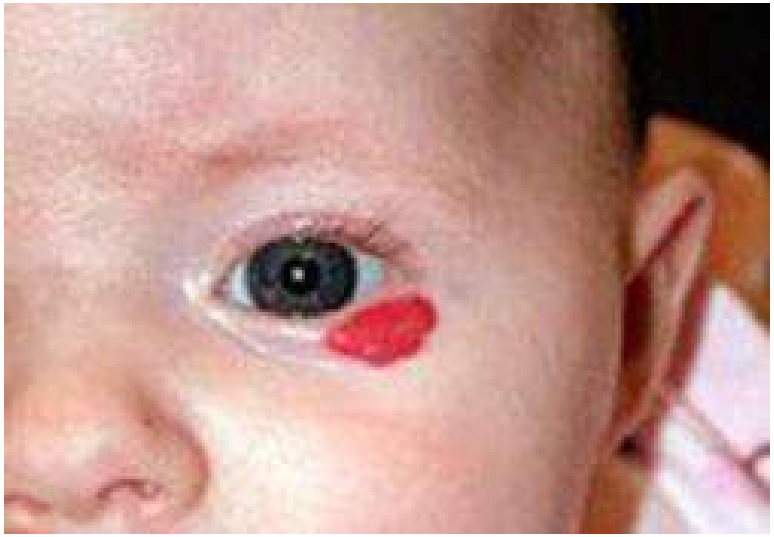


Benign Eyelid Lesions: Capillary Hemangioma

- Most common vascular lesion in childhood (5-10% of infants)
- Females 3:2
- Periorbital may appear as a superficial cutaneous lesion, subcutaneous, deep orbital or combination
- 1/3 visible at birth, remainder manifest by 6 months
- 75% regress to some extent by 7 years

Benign Eyelid Lesions: Capillary Hemangioma

- Classic superficial lesion
  - strawberry lesion, appears as a red, raised, nodular mass which blanches with pressure
- Most common ocular complication is amblyopia
- regression is common, treatment is reserved for patients who have specific ocular, dermatologic or systemic indications for intervention.



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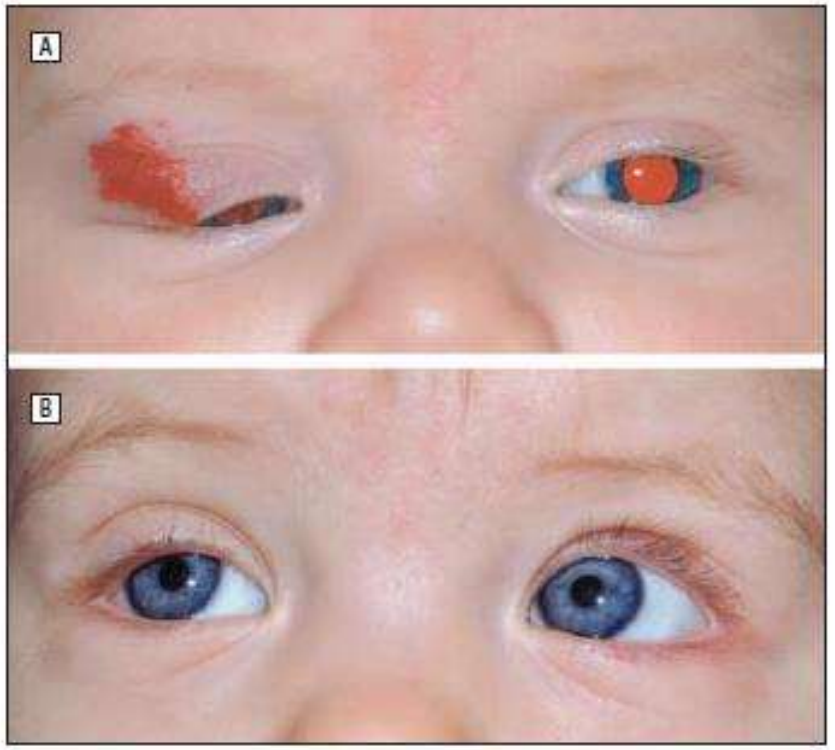
Benign Eyelid Lesions: Capillary Hemangioma

- Mainstay treatment includes the use of oral propanolol
- Recent protocols include use of topical timolol 0.25 or 0.50% Gel Forming Solution (GFS) BID for 3-4 months for superficial hemangiomas
- The exact mechanism of action of β-blockers for the treatment is not yet completely understood, however, it is postulated to inhibit growth by at least four distinct mechanisms: vasoconstriction, inhibition of angiogenesis or vasculogenesis, induction of apoptosis, and recruitment of endothelial progenitor cells (EPCs) to the site of the hemangioma



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Lid Nevi

- Lid nevi:
  - congenital or acquired
  - occur in the anterior lamella of the eyelid and can be visualized at the eyelid margin.
- The **congenital eyelid nevus** is a special category with implications for malignant transformation.
- With time, slow increased pigmentation and slight enlargement can occur.
- An **acquired nevus** generally becomes apparent between the ages of 5 and 10 years as a small, flat, lightly pigmented lesion

Congenital Nevus

- The nevus is generally well circumscribed and not associated with ulceration.
- The congenital nevus of the eyelids may present as a "kissing nevus" in which the melanocytes are present symmetrically on the upper and lower eyelids.
  - Presumably this nevus was present prior to eyelid separation

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Congenital Nevus

- Most nevi of the skin are not considered to be at increased risk of malignancy.
  - However, the large congenital melanocytic nevus appears to have an increased risk of malignant transformation of 4.6% during a 30 year period

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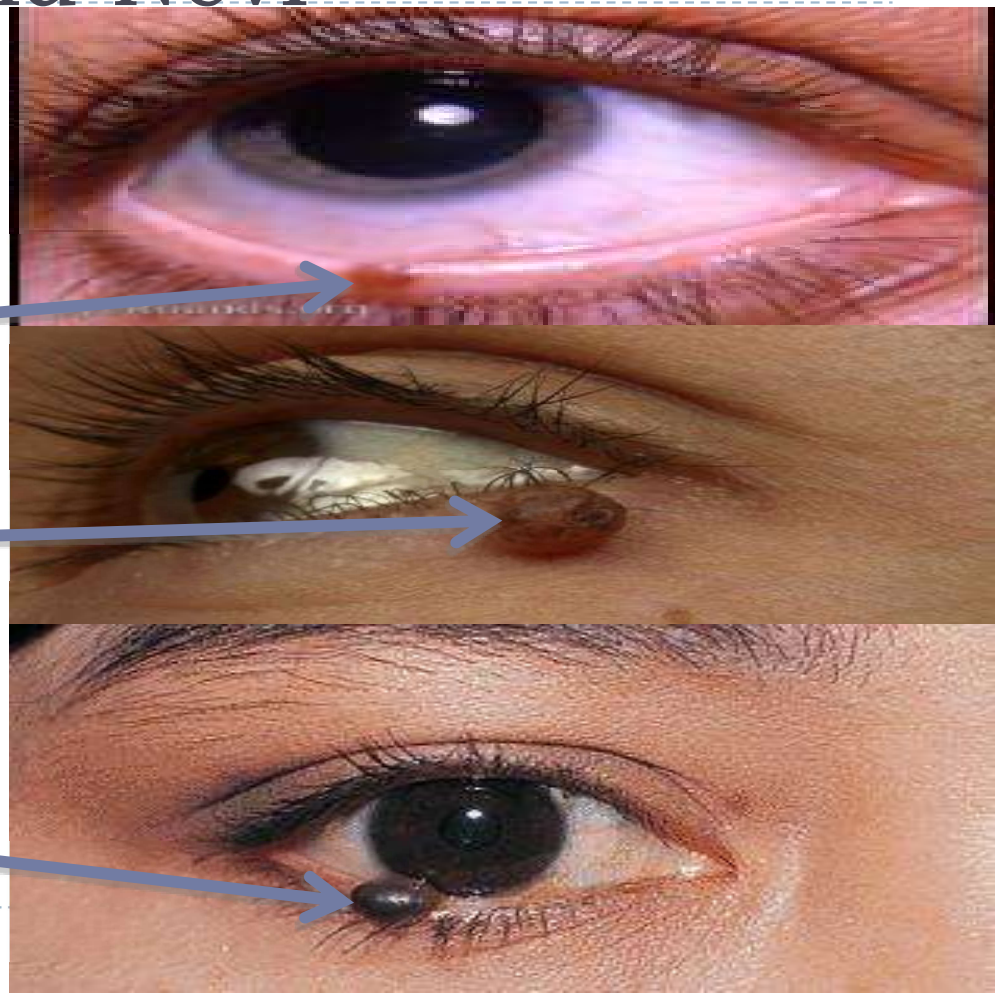
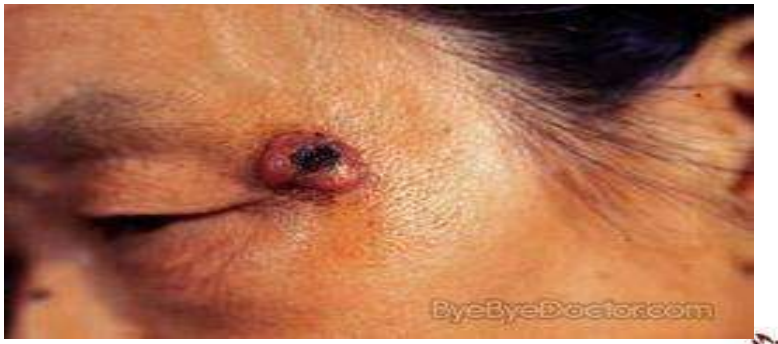
Acquired Lid Nevi

- Acquired nevi are classified as:
  - **junctional** (involving the basal epidermis/dermis junction), typically flat in appearance
  - **intradermal** (involving only the dermis), tend to be dome shaped or pedunculated
  - **compound** (involving both dermis and epidermis) tend to be dome shaped



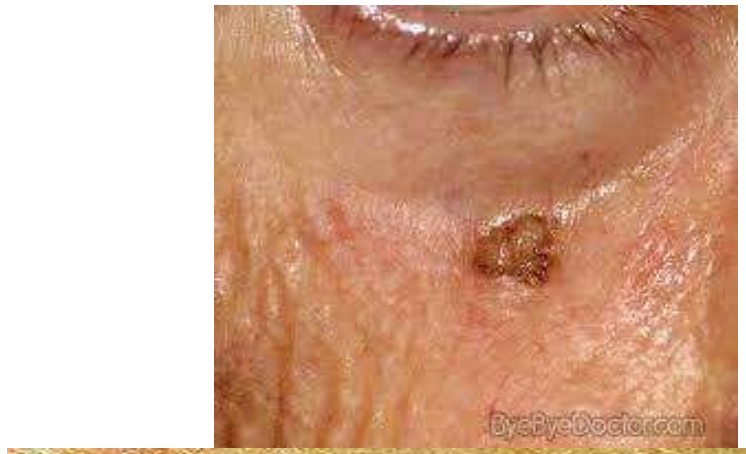
Pre-Malignant Eyelid Lesions: Keratoacanthoma

- Appears as a solitary, rapidly growing nodule on sun exposed areas of middle-aged and older individuals
- Nodule is usually umbilicated with a distinctive crater filled with keratin
- Lesion develops over weeks and undergoes spontaneous involution within 6 mo to leave an atrophic scar
- Complete excision is recommended as there are invasive variants



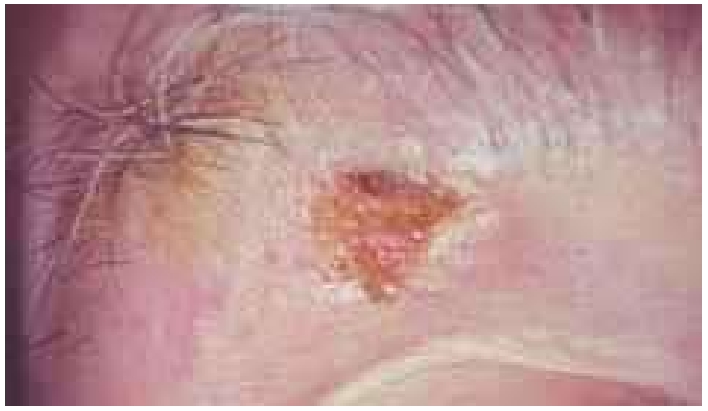
Pre-Malignant Eyelid Lesions: Actinic Keratosis

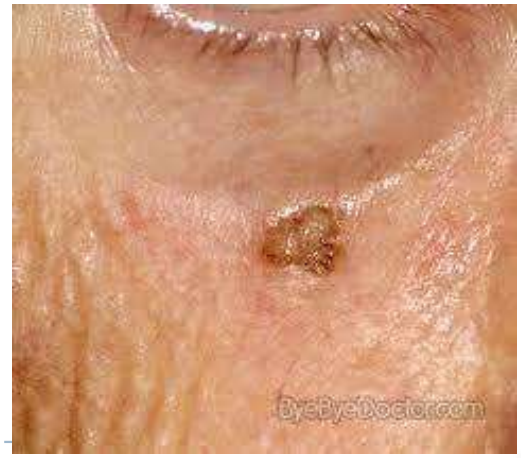
- Also known as solar or senile keratosis
- Most common pre-malignant skin lesion
- Develops on sun-exposed areas and commonly affect the face, hands and scalp (less commonly the eyelids)
  - Predominately white males



Pre-Malignant Eyelid Lesions: Actinic Keratosis

- Appear as multiple, flat-topped papules with an adherent white scale.
- Development of SCC in untreated lesions as high as 20%
- Management is surgical excision or cryotherapy (following biopsy)





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Malignant Eyelid Lesions: Basal Cell Carcinoma (BCC)

- Most common malignant lesion of the lids (85-90% of all malignant epi eyelid tumors)
- 50-60% of BCC affect the lower lid followed by medial canthus 25-30% and upper lid 15%
- Metastases is rare but local invasion is common and can be very destructive

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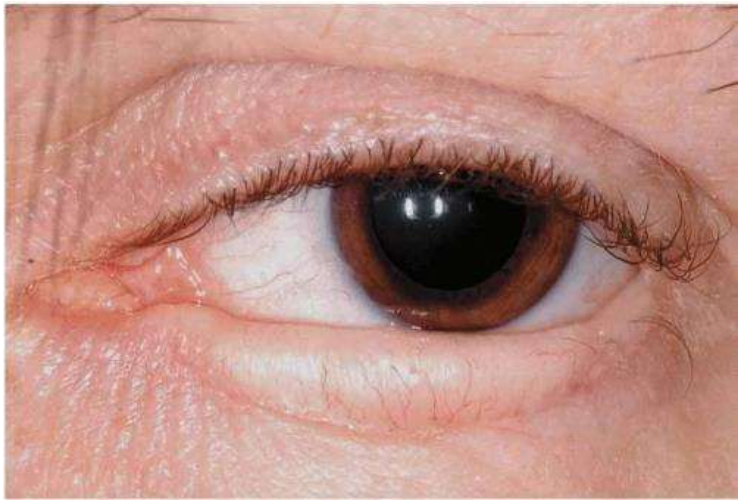
Malignant Eyelid Lesions: Basal Cell Carcinoma

- Diagnosis is initially made from its clinical appearance, especially with the noduloulcerative type with its raised pearly borders and central ulcerated crater
  - categorized into two basic types: noduloulcerative and morpheaform
  - The morpheaform variant is typically diffuse, relatively flat with indistinct borders. This variant is more aggressive and can be invasive despite showing less obvious features.



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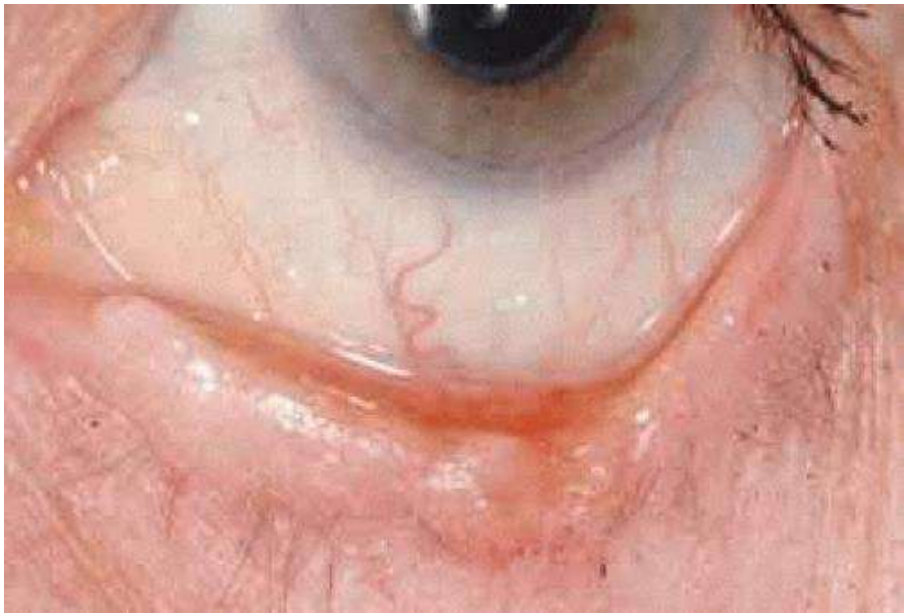
Malignant Eyelid Lesions: Basal Cell Carcinoma

- Definitive diagnosis made on histopathological examination of biopsy specimens
  - loss of adjacent cilia is strongly suggestive of malignancy and occurs commonly with basal cell carcinoma of the eyelid
- Surgery is generally accepted as treatment of choice
  - Mohs' surgery technique



https://entokey.com/tumors-of-the-eyelids/

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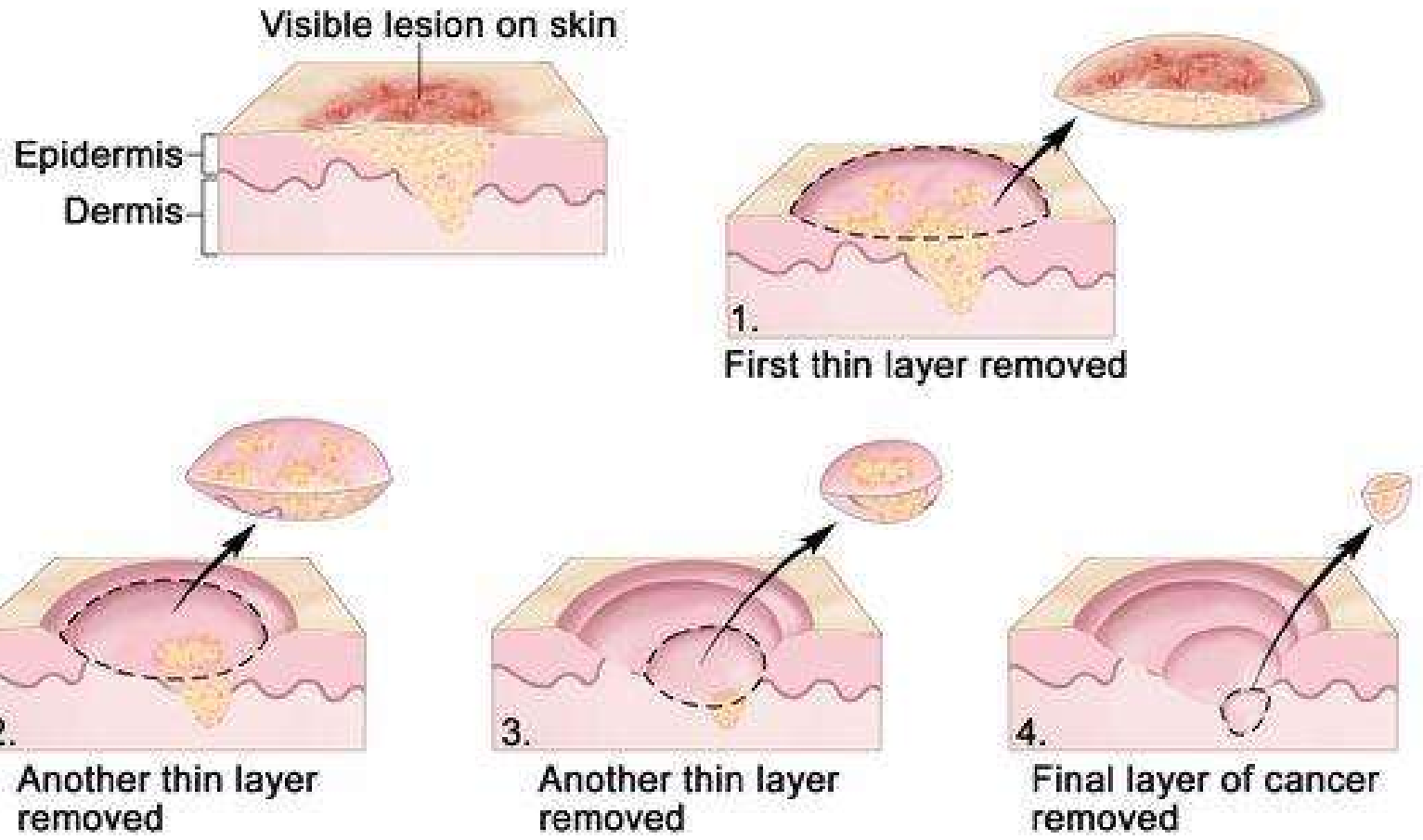
Mohs Surgery

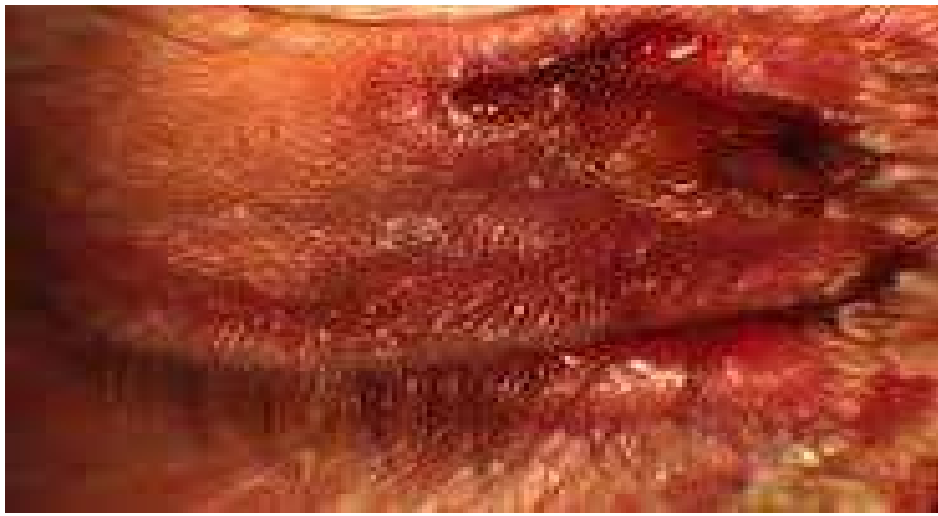


Malignant Eyelid Lesions: Squamous Cell Carcinoma (SCC)

- Much less common than BCC on the eyelid but has much higher potential for metastatic spread
- Typically affects elderly, fair-skinned and usually found on the lower lid

Malignant Eyelid Lesions: Squamous Cell Carcinoma (SCC)

- Presents as an erythematous, indurated, hyperkeratotic plaque or nodule with irregular margins
- Lesions have a high tendency towards ulceration and tend to affect lid margin and medial canthus



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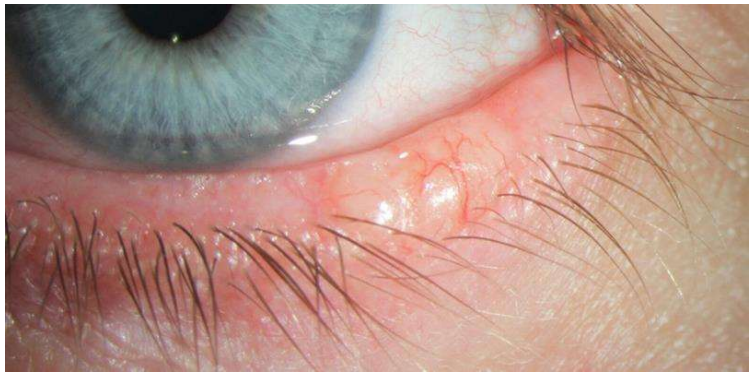
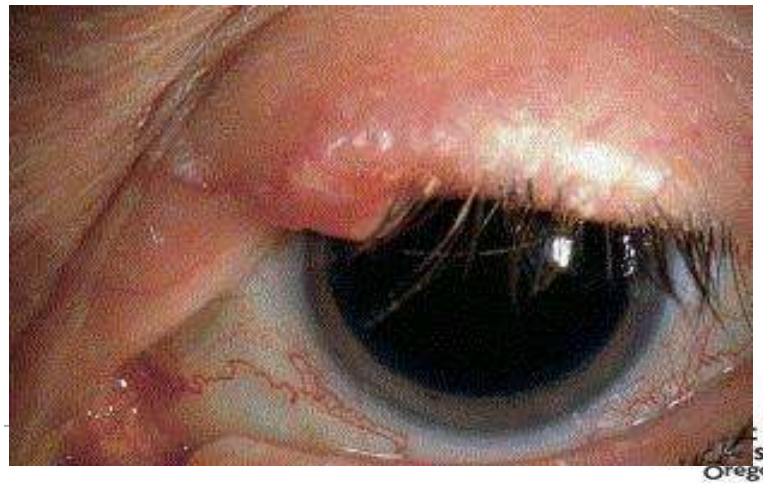
Malignant Eyelid Lesions: Sebaceous Gland Carcinoma

- Highly malignant neoplasm that arises from the meibomian glands, Zeis and the sebaceous glands of the caruncle and eyebrow
- Aggressive tumor with a high recurrence rate, significant metastatic potential and notable mortality rate
  - **rates of misdiagnosis have been reported as high as 50%**

Malignant Eyelid Lesions: Sebaceous Gland Carcinoma

- Relatively rare, 3rd most common eyelid malignancy
- Uncommon in the Caucasian population and represents only 3% of eyelid malignancies,
  - most common eyelid malignancy in Asian Indian population, where it represents approximately 40% or more of eyelid malignancies

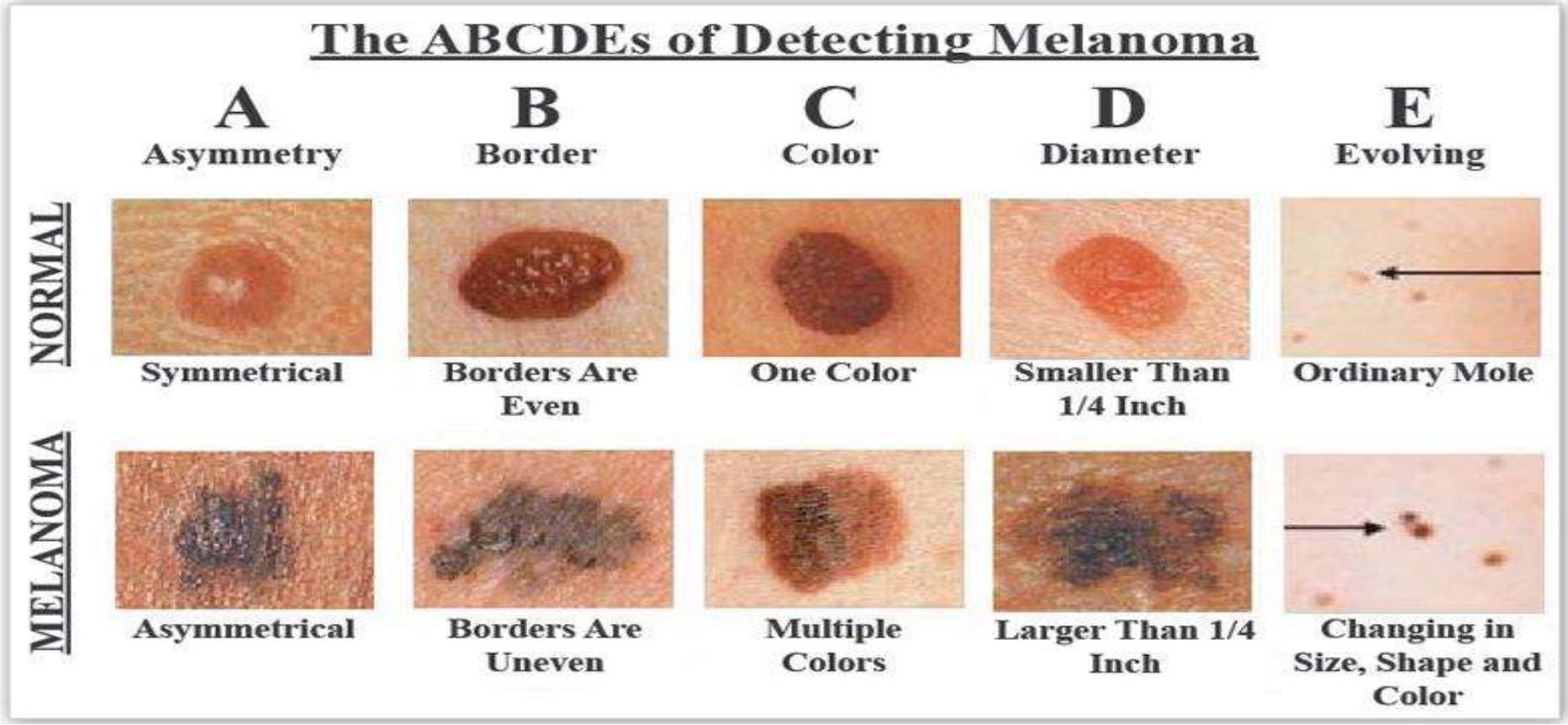




Malignant Eyelid Lesions: Malignant Melanoma

- MM of the eyelid accounts for about 1% of all eyelid malignancies
- Risk factors include congenital and dysplastic nevi, changing cutaneous moles, excessive sun exposure and sun sensitivity, family history, age greater than 20 and white.
- History of severe sunburns rather than cumulative actinic exposure thought to be a major risk factor

Malignant Eyelid Lesions: Malignant Melanoma



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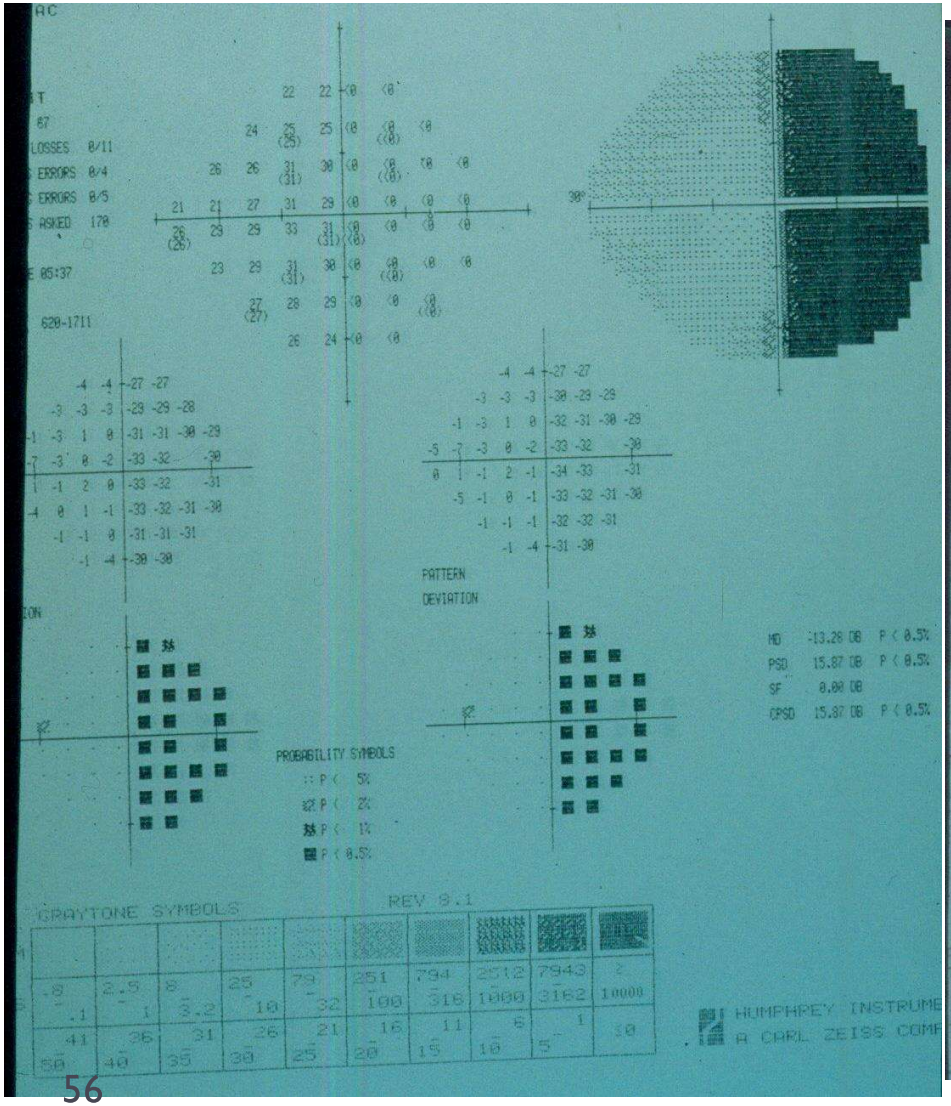
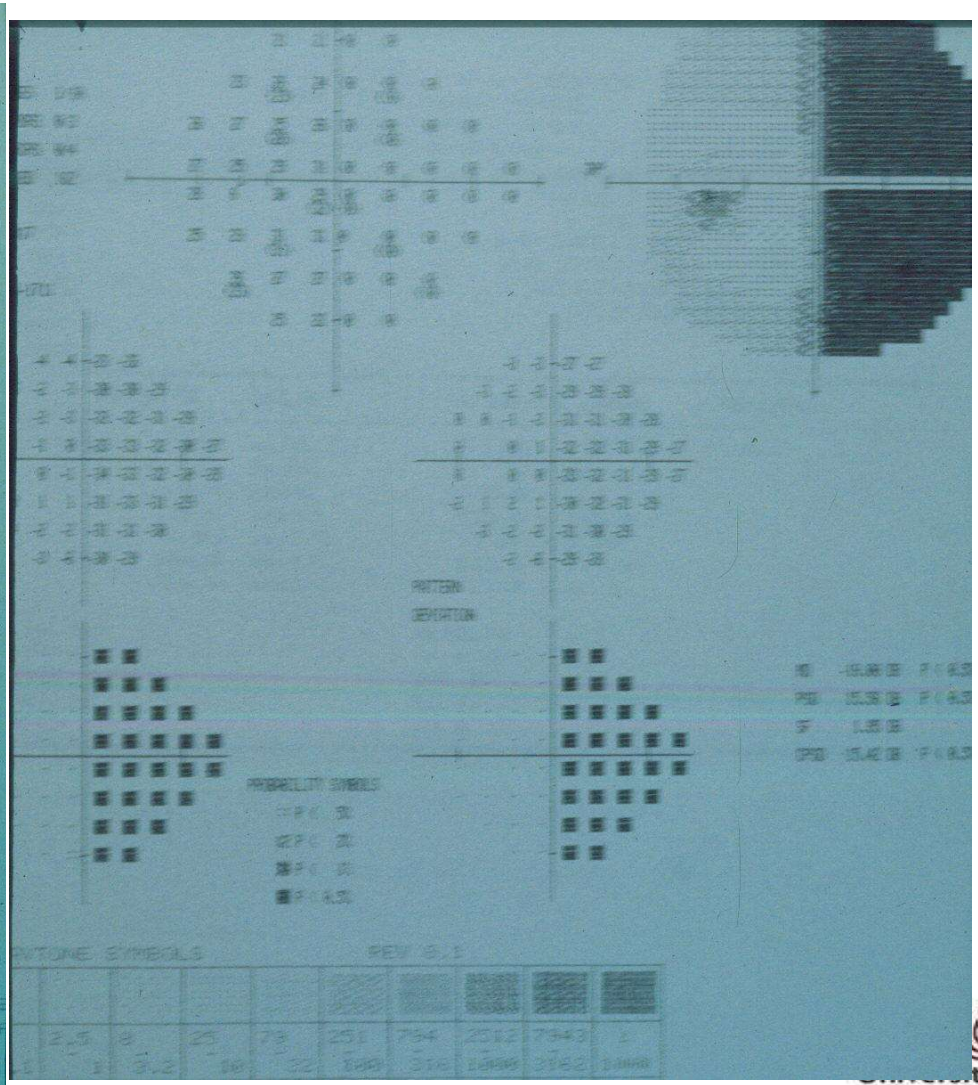
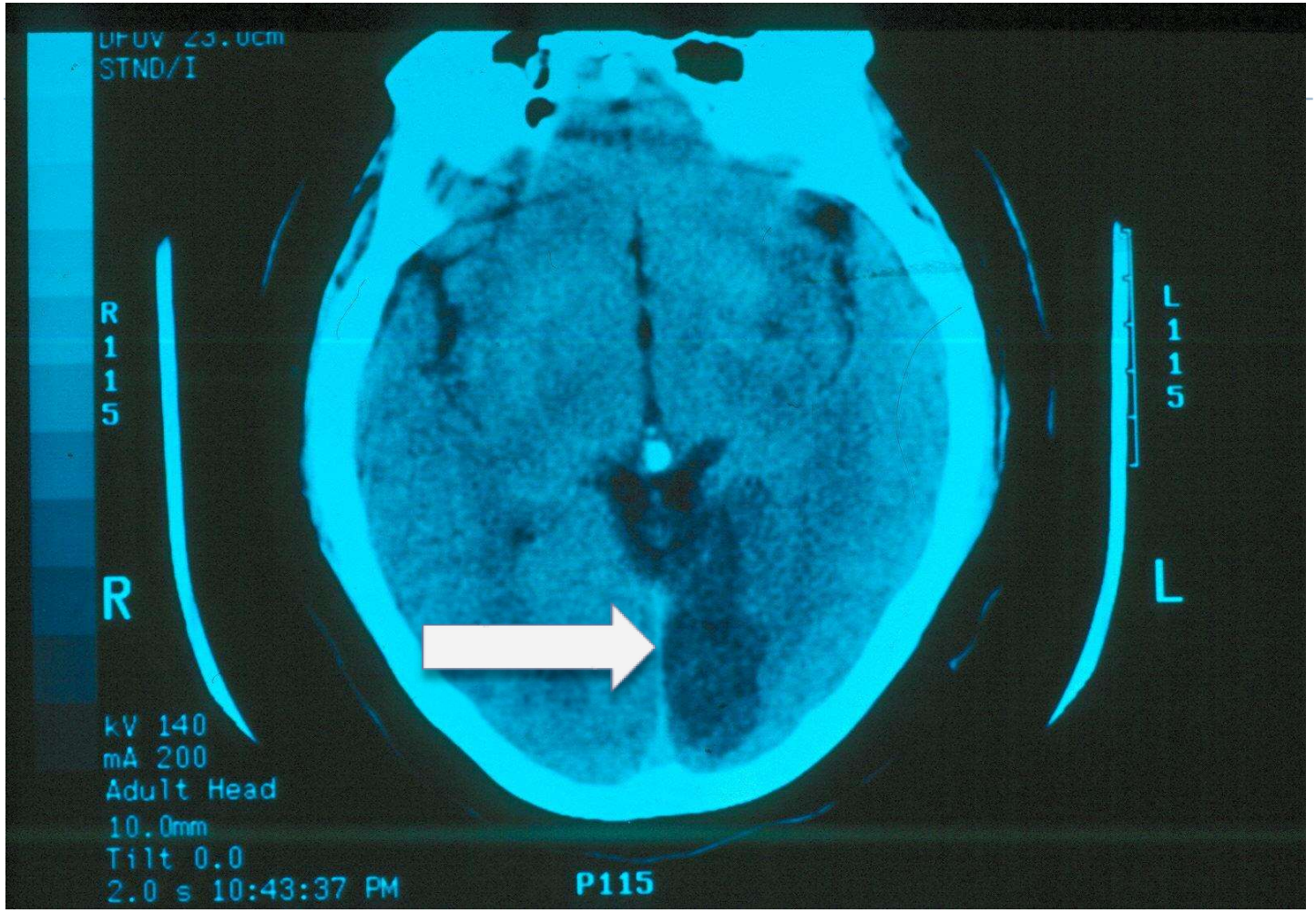
Case Example

- 67 YOF
- HA and vision loss x 2 days
- OHx: unremarkable
- LEE: 3 days ago!
- MHx: unremarkable

Case courtesy of Dr. Tammy Than


LOSSES 0/11
ERRORS 0/4
ERRORS 0/5
ASKED 170
620-1711
PATTERN
DEVIATION
MD -13.28 DB P < 0.5%
PSD 15.87 DB P < 0.5%
SF 0.00 DB
CPSD 15.87 DB P < 0.5%
PROBABILITY SYMBOLS
P < 5%
P < 2%
P < 1%
P < 0.5%
GRAYTONE SYMBOLS
REV 8.1
HUMPHREY INSTRUME
A CARL ZEISS COM





Case courtesy of Dr. Tammy Than

Minocycline?

- Proposed mechanisms
  - ↓ MMPs (MMP-9)
    - Increase in MMP-9 disrupt blood brain barrier and are linked to poor functional recovery
  - Anti-inflammatory
  - Reduction in microglial activation
    - **microglial activation** is believed to play a central role **in** neuroinflammation and pathological progression of ischemic tissue
  - Nitric oxide (NO) production
    - NO plays a neuroprotective role in **acute ischemic stroke**.
  - Inhibition of apoptotic cell death
    - **Apoptosis** may contribute to a significant proportion of neuron death following acute brain **ischemia**

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Acute Stroke Management

- N=152
- Open-label, evaluator masked study
- Minocycline 200 mg QD x 5 d or placebo
- Evaluated on NIH Stroke Scale
  - 0-1 complete/nearly complete improvement
  - 2-7 – mild
  - 8-14 – moderate
  - >15 – severe
  - Day 30: 1.8 versus 7.1

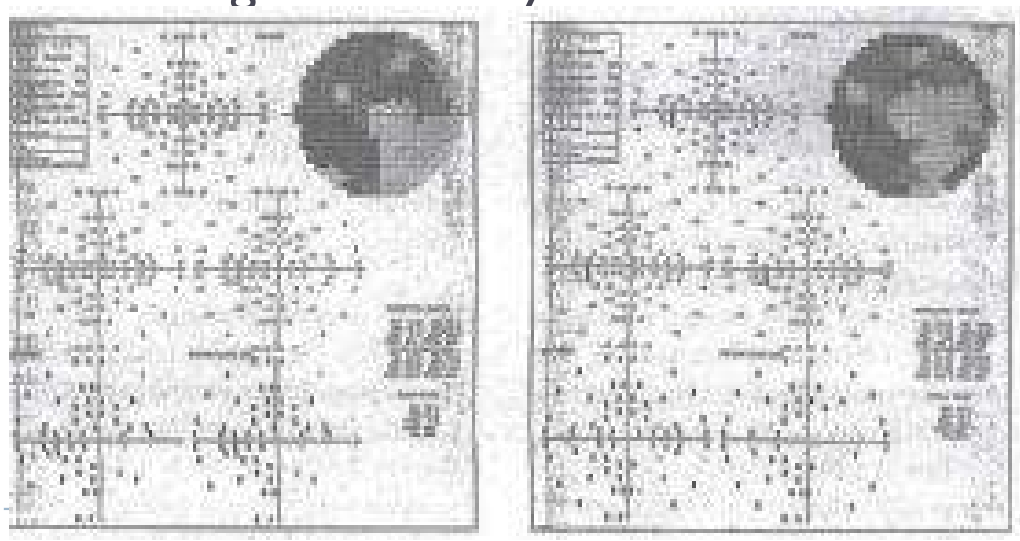
Lampl Y, Boaz M, Gilad R, Lorberboym M, Dabby R, Rapoport A, et al. Minocycline treatment in acute stroke. Neurology. 2007;69(14):1404–10

TEST	Admission	Day 7	Day 30	Day 90
NIHSS - Min	7.5	6.5	1.8	1.6
NIHSS – Cont	7.6	8.1	7.3	6.5
mRS – Min	2.8	1.5	1.1	0.9
mRS – Cont	2.0	3.1	2.7	2.1
BI – Min	70.0	85.9	90.6	94.9
BI – Cont	63.9	61.9	68.5	77.6

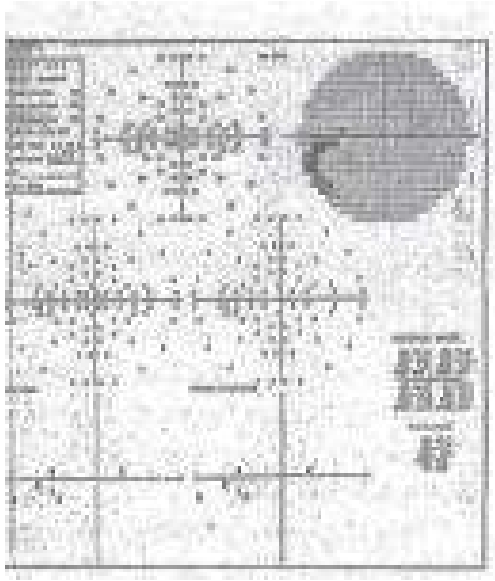
Minocycline for acute stroke treatment: a systematic review and meta-analysis of randomized clinical trials. J Neurol. 2018 Aug;265(8):1871-1879

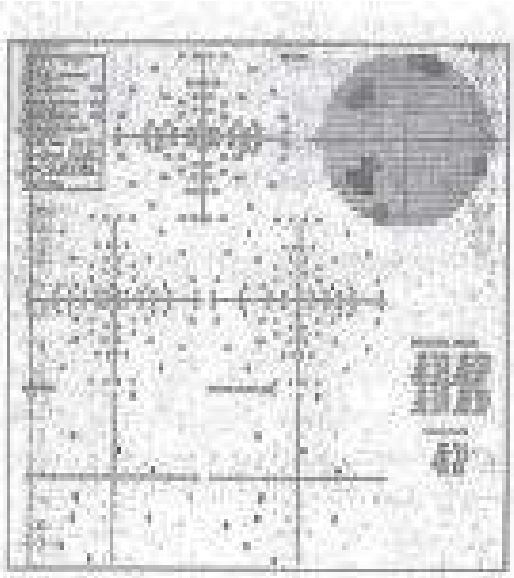
Case Report

- 77 YOM
- Right occipital infarct
- 3 weeks post stroke
  - Minocycline 100 mg BID x 5 days

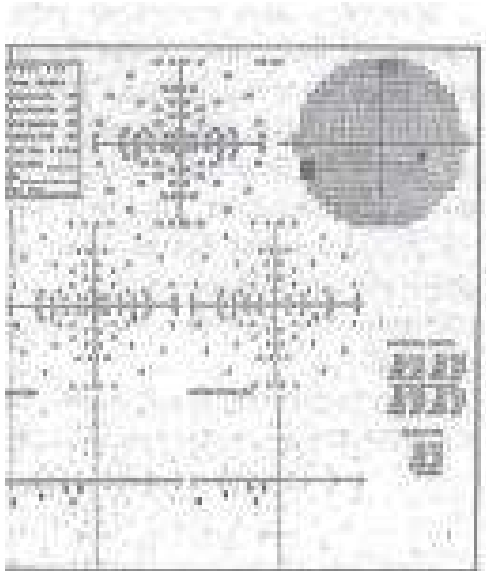


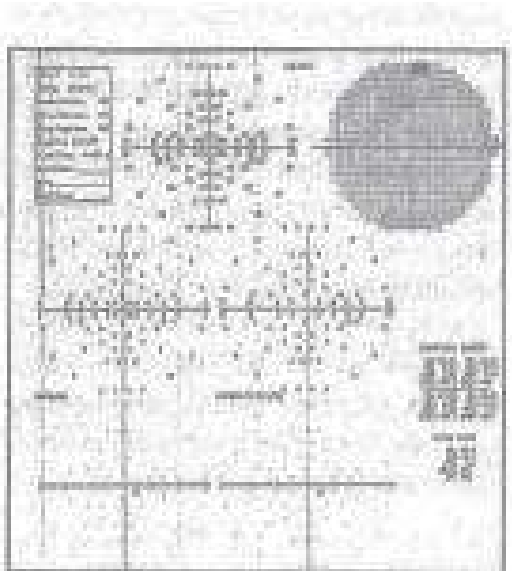
Shortly after TX





1 Year Later





Tetracyclines

- This group includes:
  - Tetracycline (250mg - 500 mg cap BID-QID) needs to be taken 1 hour before or 2 hours after a meal.
  - Minocycline (100 mg cap BID)
  - Doxycycline (20mg - 100 mg cap or tab BID)
    - **In Canada: Apprilon (30 mg doxy + 10 mg slow release doxy)**
- **Rules of Thumb with Doxy:**
  - Do not take before lying down (>2 hours before)
  - Do not take with calcium and avoid antacids
  - Do not take with dairy
  - Do take with food
  - Do educate on sun protection

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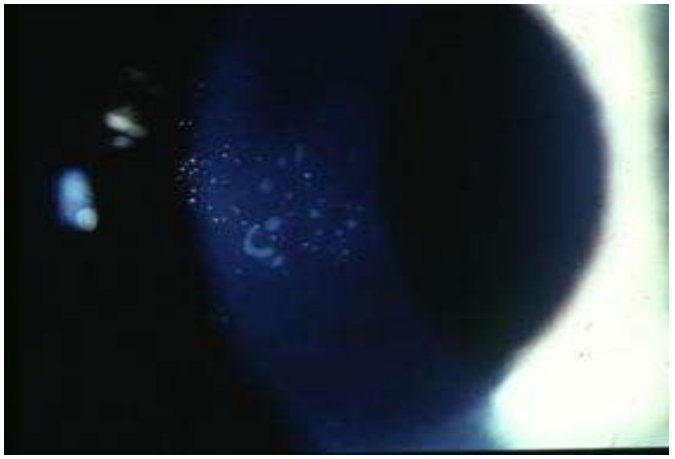
- A 30 yr old female patient presents on an emergent basis complaining that she wakes up in the morning with a sharp stabbing pain in her right eye. It gets better as the day goes on but has been happening frequently over the past several months. What would you recommend for this patient?



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Epithelial (Anterior) Basement Membrane Dystrophy (EBMD or ABMD)

- Primary features of this "dystrophy" are:
  - abnormal corneal epithelial regeneration and maturation,
  - abnormal basement membrane
- Often considered the most common dystrophy, but may actually be an age-related degeneration.
  - large number of patients with this condition,
  - increasing prevalence with increasing age, and
  - its late onset support a degeneration vs. dystrophy.

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Epithelial (Anterior) Basement Membrane Dystrophy (EBMD or ABMD)

- Not all patients are symptomatic
- Most common symptom is mild FB sensation which is worse in dry weather, wind and air conditioning
- Blurred vision from irregular astigmatism or rapid TBUT
- Pain is usually secondary to a RCE (recurrent corneal erosion) in apprx 10%

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Epithelial (Anterior) Basement Membrane Dystrophy (EBMD or ABMD)

- Easy to overlook:
  - typically bilateral though often asymmetric,
  - females>males,
  - often first diagnosed b/w ages of 40-70

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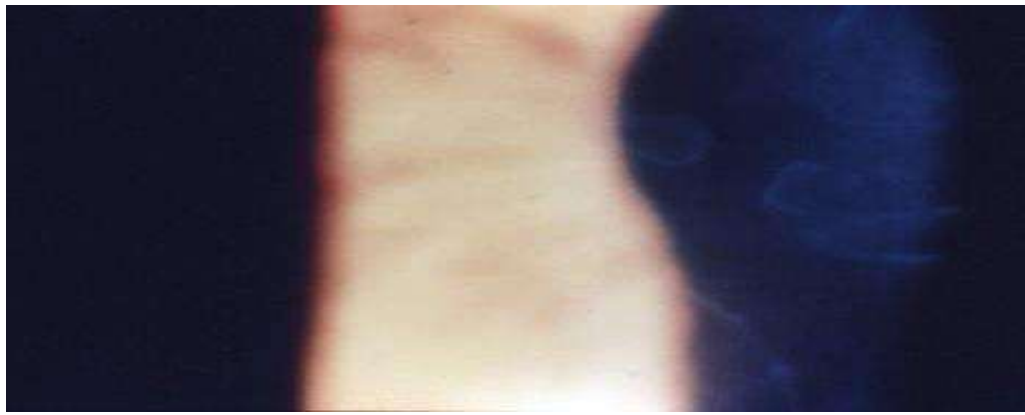
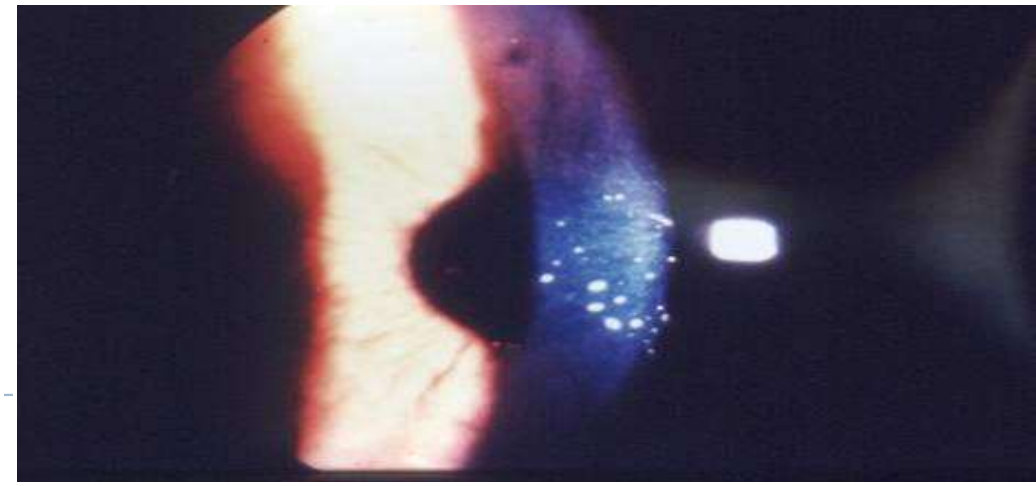
Epithelial (Anterior) Basement Membrane Dystrophy (EBMD or ABMD)

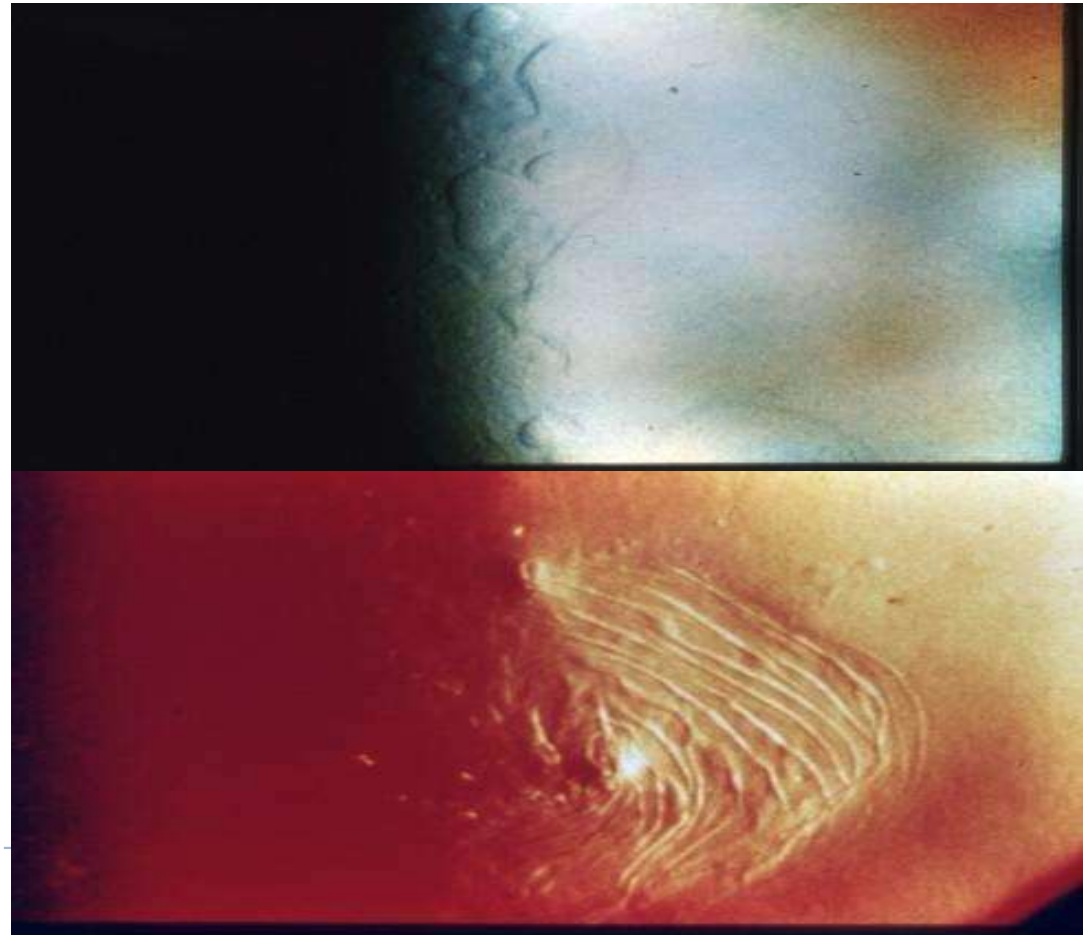
- Most common findings are:
  - chalky patches,
  - intraepithelial microcysts, and
  - fine lines (or any combination) in the central 2/3rd of cornea



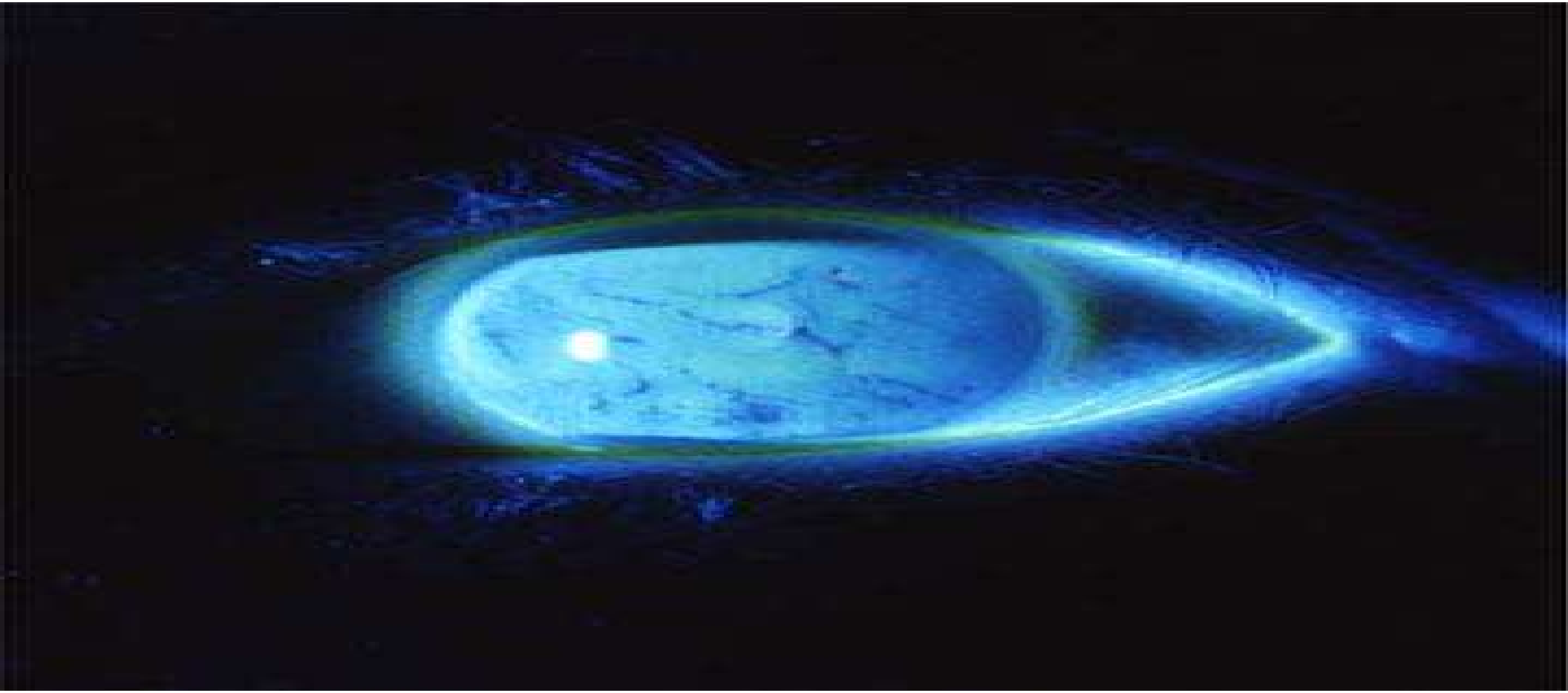


Epithelial (Anterior) Basement Membrane Dystrophy (EBMD or ABMD)

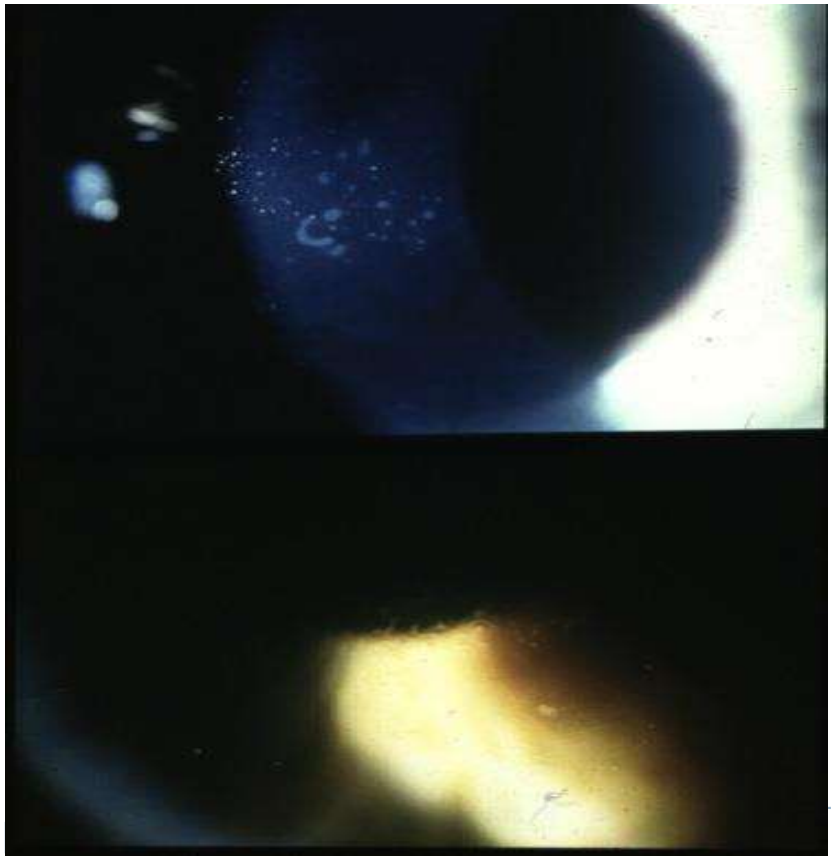
- Often referred to as:
  - maps,
  - dots or
  - fingerprints



EBMD-Negative Staining



Epithelial (Anterior) Basement Membrane Dystrophy (EBMD or ABMD): Treatment



- Typically directed towards preventing RCE
- If RCE's develop:
  - awake with painful eye that improves as day wears on
  - chalky patches/dots in lower 2/3rd of cornea

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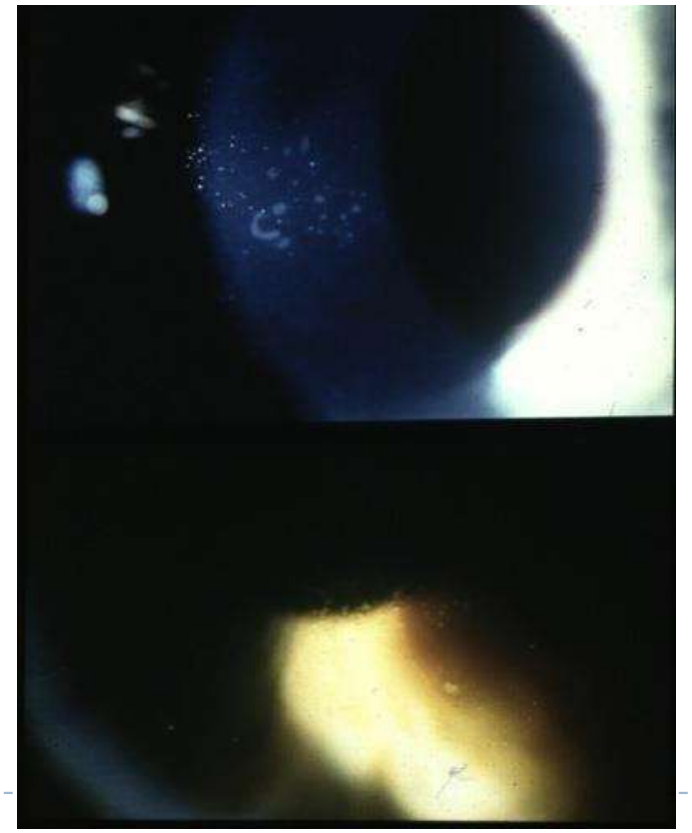
Acute Treatment of RCE



- use of hyperosmotic ointment at bedtime
- bandage contact lens
- Frequent lubrication
- Plugs
- Topical meds
- No ceiling fans
- Night time ointment
- PTK

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Recurrent Corneal Erosion: Treatment

- If severe enough to cause vision loss or repeated episodes:
  - oral doxycycline with/without topical corticosteroid
    - Doxy 50 mg bid and FML tid for 4-8 weeks
    - both meds inhibit key metalloproteinases important in disease pathogenesis
  - debridement,
  - Debridement + diamond burr polishing
  - stromal puncture (not commonly done anymore)
  - PTK
  - Latest development: amniotic membrane transplant e.g. Prokera typically after debridement

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CORNEAL DEBRIDEMENT

- Soften epithelium
- 1-2 gtt topical anesthetic
- q 15-30 seconds for 2-3 minutes
- Use cotton swab, spatula, spud
- or jewelers forceps
- Remove flaps by pulling edges toward center
- Don't pull directly up or out
- Remove flaps down to tight,
- firm edges.
- Tx abrasion (>50-100%)
  - Recurrence Rate 18%

Pictured: Kimura Platinum Spatula

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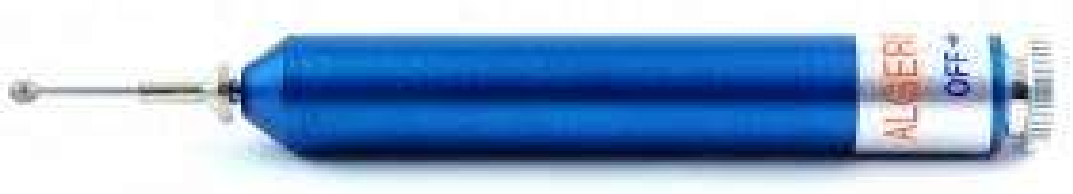
Diamond Burr Polishing

- Removes abnormal basement membrane
- Provides smooth surface for cells to grow



https://www.katena.com/pterygium-burr-3-5mm-w-chuck-k2-4913

Vo, et al (2014): epithelial debridement with diamond burr polishing was 95% effective after single treatment in preventing recurrence for an average of 32 months follow up time

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13 YR Female

CC: noticed that her left eye became blurry and objects were "wavy" a couple of days ago. Sudden onset and she had experienced a headache over the left eye just prior to the vision going blurry.

Ocular Hx: she currently wear glasses for distance

Medical Hx: she is currently not diagnosed with any health problems and is not taking any medications

Entrance Skills

VA with current Rx: 20/30 OD and 20/30 OS

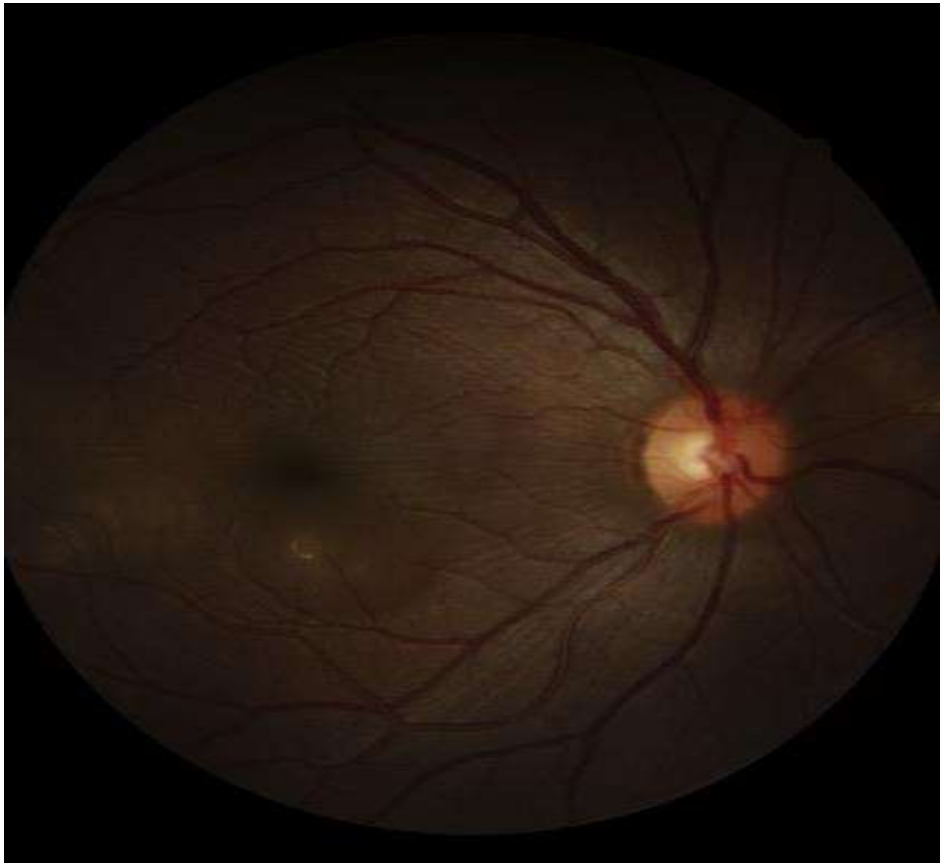
Entrance skills unremarkable

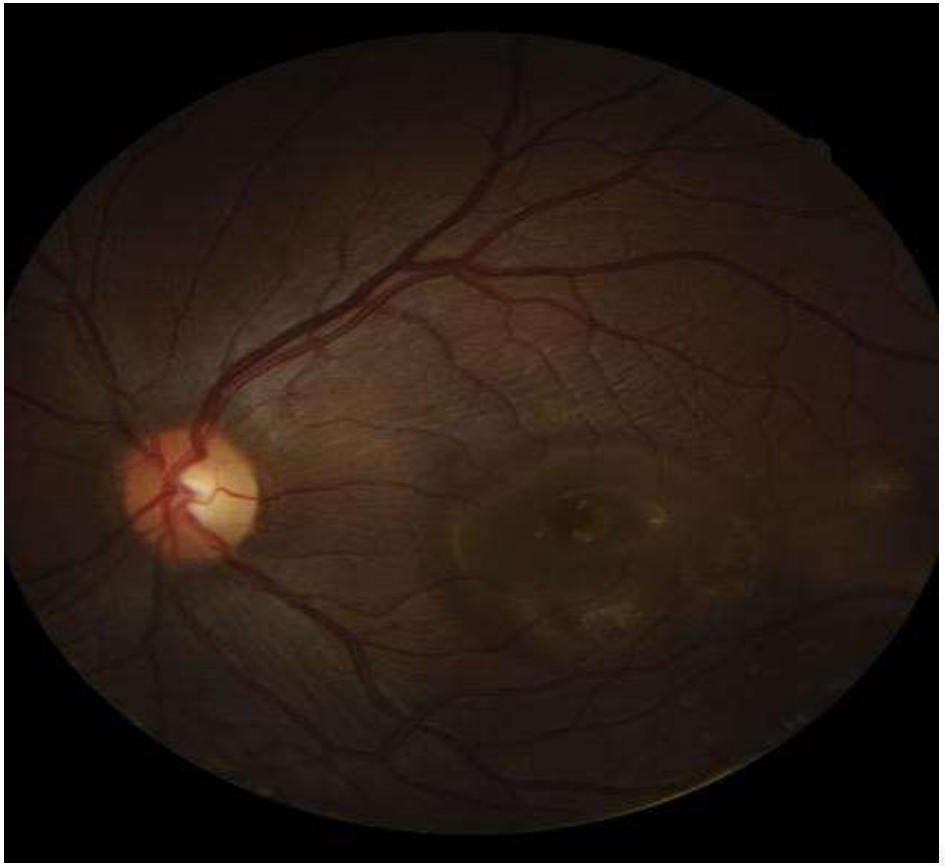
Amsler: metamorphopsia OS

BCVA: 20/20 OD with increased minus, no improvement possible in the left eye

IOP's: 13 mm Hg OD and OS

Fundus Photos





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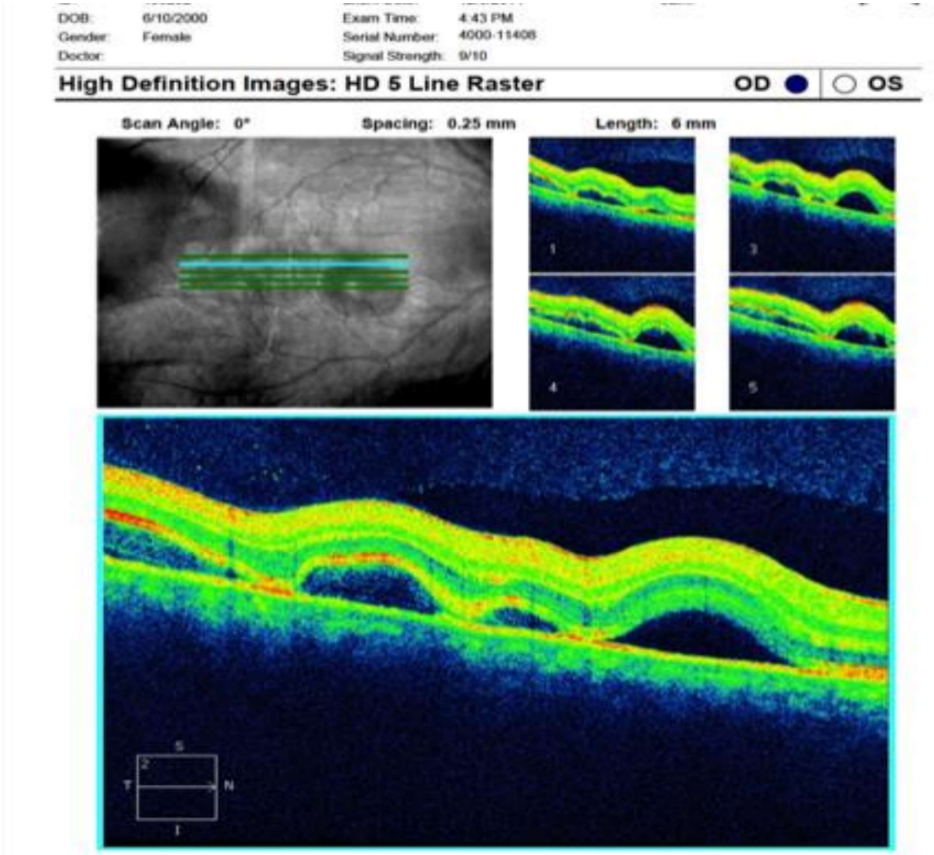
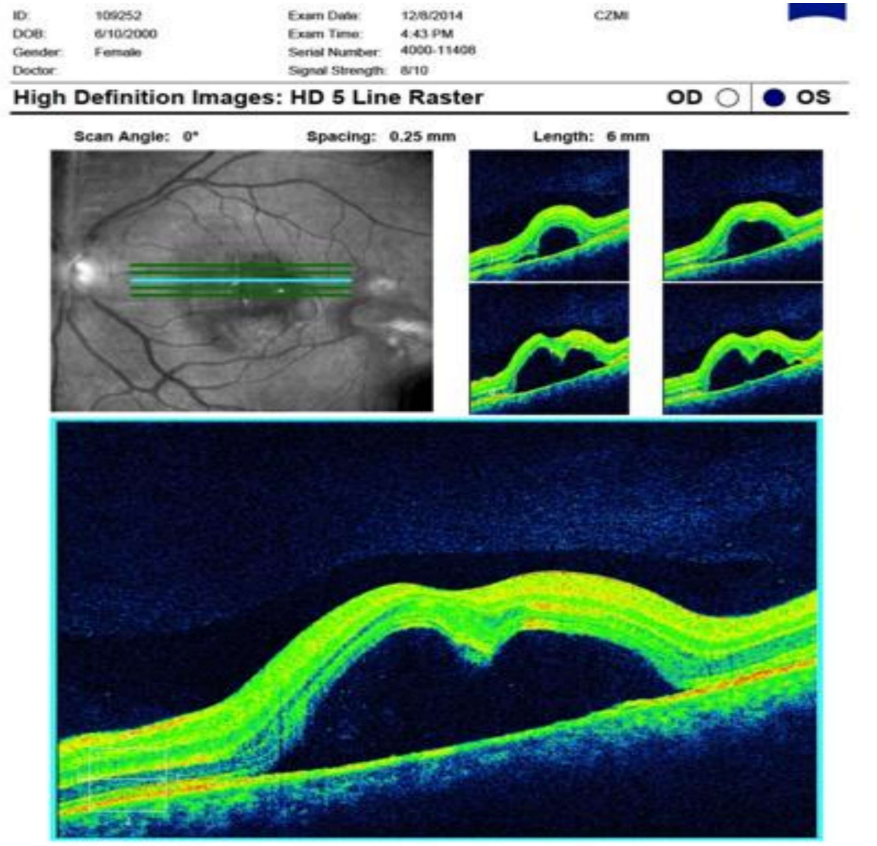
OCT





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Retina Consult

- Referred patient to retina and they confirmed the diagnosis of VKH.
- She was begun on oral prednisone 60 mg per day and she was re-evaluated in 1 week.
- At the follow up, there was reduction in her serous retinopathy and vision was improved.

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From the Experts

- Vogt-Koyanagi-Harada (VKH) disease is a multisystemic disorder characterized by granulomatous panuveitis with exudative retinal detachments that is often associated with neurologic and cutaneous manifestations.
- VKH disease occurs more commonly in patients with a genetic predisposition to the disease, including those from Asian, Middle Eastern, Hispanic, and Native American populations.

From the Experts

- VKH:
  - Patients have no prior history of ocular trauma or surgery
  - Patients have no evidence of another ocular disease based on clinical or laboratory evidence
  - Patients have bilateral ocular involvement.

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From the Experts

- VKH:
  - The neurologic and auditory signs include the following:
    - Malaise, fever, headache, nausea, abdominal pain, stiffness of the neck and back, or a combination of these factors; headache alone is not sufficient to meet the definition of meningitis
    - Tinnitus
    - Cerebrospinal fluid pleocytosis
  - Integumentary signs include the following:
    - Alopecia: loss of body hair
    - Poliosis: loss of pigment in hair
    - Vitiligo: loss of skin pigmentation in blotchy pattern

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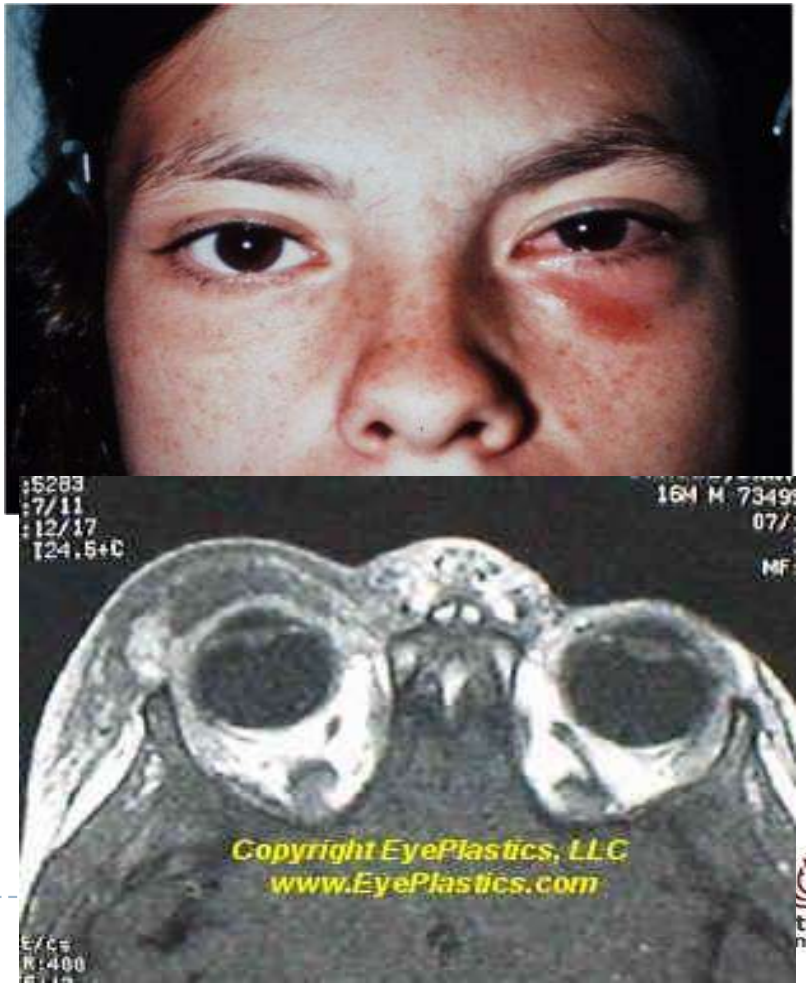
VKH Treatment

- For most patients with bilateral serous detachments and severe visual loss, begin therapy with systemic prednisone (1-2 mg/kg/day).
- The length of treatment and subsequent taper must be individualized for each patient.
  - Most patients require therapy for 6 months and occasionally up to 1 year before successful tapering of systemic corticosteroids.
  - Systemic therapy should not be discontinued during the 3 months following the onset of the disease because of the risk for recurrence.

Preseptal Cellulitis

- Infection and inflammation located anterior to the orbital septum and limited to the superficial periorbital tissues and eyelids.
- Usually follows sinus infection or internal hordeolum (possibly trauma)
- Eyelid swelling, redness, ptosis, pain and low grade fever.



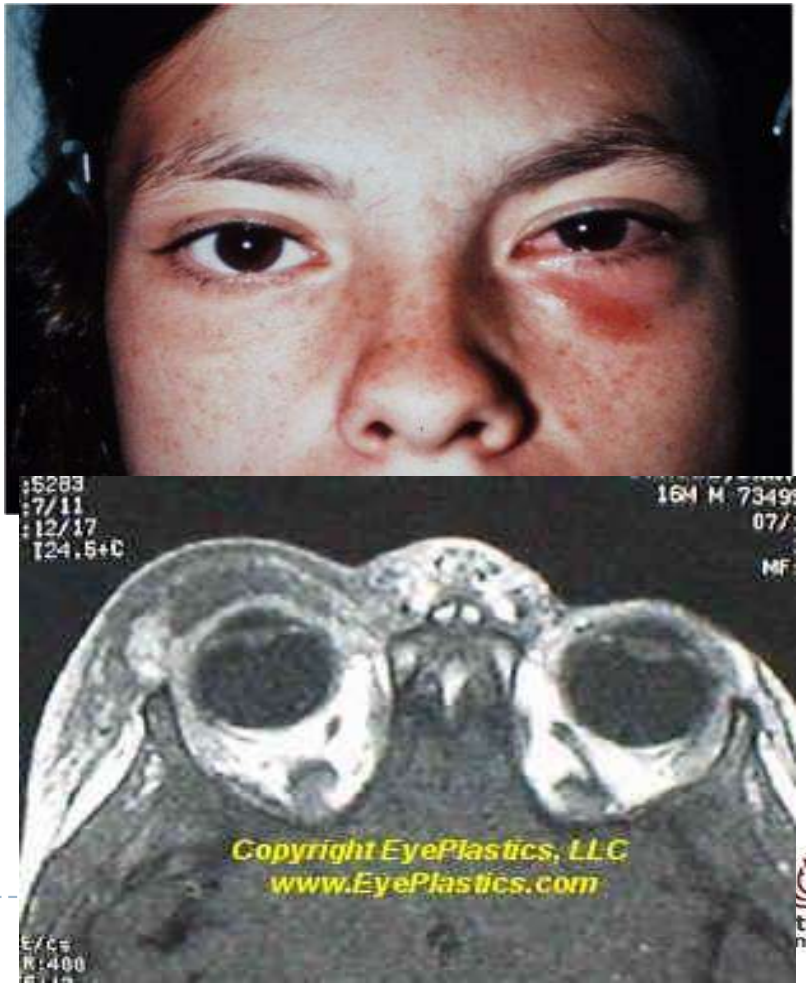
Differentiating Orbital vs. Preseptal

FINDING	ORBITAL	PRESEPTAL
Visual Acuity	Decreased	Normal
Proptosis	Marked	Absent
Chemosis and Hyperemia	Marked	Rare/Mild
Pupils	RAPD	Normal
Pain and Motility	Restricted and Painful	Normal
IOP		Normal
Temperature	102 - 104	Normal/mild elevation
HA and Assoc. Symptoms	Common	Absent

Treatment: Orals for Preseptal, Often IV for Orbital

Preseptal Cellulitis

- Tx:
  - **Clavulin** (***Augmentin) 500 mg TID or 875 mg BID for 5-7 days***
  - ***Keflex 500 mg QID 5-7 days***
  - or if moderate to severe IV Fortaz (ceftazidime) 1-2 g q8h.
  - If MRSA possible, consider Bactrim/Septra



Penicillins: Augmentin

- **Augmentin is amoxicillin with potassium clavulanate (clavulanic acid 125 mg).**
- Clavulanate is a B-Lactamase inhibitor which reduces a bacteria's ability to negate the effect of the amoxicillin by inactivating penicillinase (enzyme that inactivates the antibiotic affect).
  - Dicloxacillin can also be used in infections due to penicillinase-producing staph.

Penicillins: Augmentin

- **Augmentin is very effective for skin and skin structure infections** such as:
  - dacryocystitis,
  - internal hordeola,
  - pre-septal cellulitis.
- Treatment of:
  - otitis media,
  - sinusitis,
  - lower respiratory and urinary infections.
- Given prophylactically to dental surgery patients.

Penicillins: Augmentin

- It has ***low***:
  - GI upset,
  - allergic reaction and anaphylaxis.
- Serious complications include:
  - anemia,
  - pseudomembranous colitis and
  - Stevens-Johnson syndrome.

Penicillins: Augmentin.

Adults:

- **250-500 mg tab q 8hr (tid)** (also available in chewable tablets and suspension)
- or **875 mg q 12hr (bid)**
- 1000 mg XR: q12 hr and not for use in children <16

Peds: <3 mos 30mg/kg/day divided q12hrs using suspension

- >3 mos 45-90mg/kg/day divided q12hrs (otitis media 90mg for 10 days)

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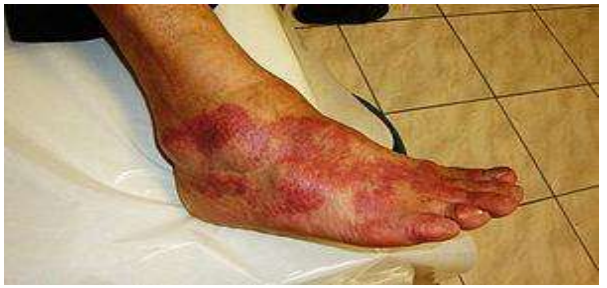
Cephalosporins

- Closely related structurally and functionally to the penicillins,
  - **have the same mode of action,**
  - affected by the same resistance mechanisms.
  - tend to be more resistant to B-lactamases.
- classified as 1st, 2nd, 3rd, 4th and now 5th generation based largely on their bacterial susceptibility patterns and resistance to B-lactamases.
- Typically administered IV or IM, **poor oral absorption**.

Side Effects and Contraindications

- Hypersensitivity Reactions are common.
  - Risk of cross sensitivity with PCN's is higher for 1st generation, but often overestimated for later medications.
  - Used to state the cross sensitivity was ~10%, but now believed to be closer to 3%.



Cephalosporins

- 1st generation: **cefadroxil (Duricef)**, **cefazolin (Ancef)**, **cephalexin (Keflex)**, and cephalothin
- 2nd generations: **cefaclor (Ceclor)**, cefprozil, **cefuroxime (Zinacef)**, cefotetan, cefoxitin
- 3rd generation: cefdinir **(Omnicef)**, cefixime, **cefotaxime (Claforan)**, **ceftazidime (Fortaz)**, ceftibuten, ceftizoxime, **ceftriaxone (Rocephin IM/IV)**.
- 4th generation: cefepime
- *Omnicef, Keflex, Ceclor* (all orally administered) are effective against most gram positive pathogens and especially good for skin and soft tissue infections.

Cephalosporins

- **Keflex (cephalexin):**
  - treatment of respiratory, GI, skin and skin structure, and bone infections as well as otitis media
  - Adults: 250-1000 mg every 6 hours
    - **- typical dosing 500 every 6 hours**
  - Children: 25-100 mg/kg/day divided 6-8 hours

Co-Trimoxazole (Bactrim/Septra)

- Combination of trimethoprim and sulfamethoxazole
  - shows greater antimicrobial activity than equivalent quantities of either drug alone.
- Has broader spectrum of action than the sulfa's and is effective in treating:
  - UTIs and respiratory tract infections
  - often considered for treatment of MRSA skin infections

Co-Trimoxazole (Bactrim/Septra)

- Available:
  - Bactrim/Septra tablets:
    - contains 80 mg trimethoprim and 400 mg sulfamethoxazole
    - dosing **2 tablets every 12 hours**
  - Bactrim DS/Septra DS (Double Strength)
    - contains 160 mg trimethoprim and 800 mg sulfamethoxazole
    - Dosing **1 tablet every 12 hours**