

Anterior segment FAQ's and BAA's

1. Photo hyphema in a child. Etiologies and management?
 1. Ddx: trauma (r/o child abuse), spontaneous: JXG, RB, leukemia, NVI, post surgical, clotting abnormalities, herpetic disease
 2. Most common young males with damage to MAC
 3. Prognosis not dependant on the amount of blood
 4. Major concern is rebleeding varies from 3-30%, usually day 2 to 5
 5. Rebleeding gives poor prognosis as 50% will develop elevated IOPs
6. TREATMENT
 1. Usual: shield, no ASA, limited activity, home if compliant, hospital if not compliant
 2. IOP: Beta blockers and CAI (IF NOT BLACK: CHECK SICKLE CELL)
 - (1) Medical management maximized and IOP remains high think surgical management
 - (2) IOP > 50 3 days, >35 5-7days, total hyphema for 9 days, blood staining then, >24 for 24hrs in sickle, >50% hyphema for more than 8-9 days do AC wash with release of pupil
 3. Amicar? Controversial. Some studies show benefit and some show little benefit. The risk is the toxicity
 4. **Amicar (Aminocaproic acid)**
 - inhibits fibrinolysis prevents plasminogen conversion
 - secondary effects:
 - 1) CNS: nausea, vomiting, vertigo, confusion
 - 2) CVS: postural hypotension, bradycardia, arrhythmia, thrombosis
 - 3) skin: pruritis, cutaneous eruptions, erythema
 - 4) myopathy, cramps
 - 5) nasal stuffiness
 - Dosage:*
 - 1) for hyphema: **50mg/kg Q4H** max 30g/day
 - 2) for life-threatening: 5g oral or IV then 1g Q1h
 - Contraindications: when DIC is going on (do Pt/PTT before giving) - only one in CPS
 5. Indications may include the following:
 1. Monocular
 2. Blacks (shown benefit, caucasions limited benefit)
 3. Sickle
 4. Bleeding diathesis
 5. EtOH associated hyphema (high rate rebleed 50%)

2. Photo of dislocated lens. What is the ddx and management?

1. Indications for **lensectomy with ectopia lentis**

1. Edge of lens bisects pupil.
2. Displacement of lens anteriorly causing secondary glaucoma.
3. Dislocated lens anteriorly causing endothelial damage

2. Ddx ectopia lentis

A) *With systemic conditions*

- 1) Marfan's - chr.15 (fibrillin def.): up
- 2) homocysteinuria (glycoprotein of zonules): down
- 3) Weil-Marchesani: temporal
- 4) Ehlers Danlos (collagen)
- 5) Stickler's (collagen)
- 6) hyperlysinemia (collagen)
- 7) sulfite oxidase deficiency
- 8) tertiary syphilis

B) *with ocular conditions*

- 1) aniridia
- 2) microspherophakia
- 3) buphthalmos
- 4) megalocornea
- 5) high myopia
- 6) uveal coloboma
- 7) Peter's anomaly

C) *other*

- 1) trauma
- 2) simple ectopia lentis (AD) (fibrillin)
- 3) ectopia lentis et pupillae (AR)
- 4) familial (AD)

3. Workup for subluxated lens

A) *History*

- 1) family history: Marfan's (heart, SKM anomalies), homocysteinuria (MR), visual problems
- 2) patient history: trauma, MR, health

B) *Eye exam*

- 1) acuity
- 2) strabismus
- 3) ant. segment (aniridia, PHPV, trauma evidence)
- 4) retinoscopy (myopia)
- 5) U/S: axial length
- 6) family exam

C) *Labs*

- 1) cardiology consult
- 2) cardiac U/S
- 3) urine a.a. (homocystinuria)

4) hand x-ray (Marfan's)

4. Treatment

1. If refraction possible then give rx at pupil centre and give bifocal also since accomodation may be lost or reduced. The refraction may be aphakic or the high myopic refraction of the lens edge. Decision can be difficult. Always give trial of patching if patient is young as amblyopia may be present.
2. Laser with argon to iris to enlarge entrance pupil. YAG to disrupt zonules (usually useless).
3. If question ends with homocysteineuria you should mention B6, low methionine and high cysteine diet. Also may need anticoagulants prior to surgery!
4. Surgery if optical correction is not possible. Either limbal or PP approach can be used as long as vitrectomy (esp in Marfan's) is done. The results are the same.

3. Young female with early bilateral PSCC cataracts. What is Ddx. Discuss JRA.

1. Ddx of cataract in young patients.
 1. Infammatory conditions: JRA, Pars planitis, other uveitides
 2. Allergic: Atopy
 3. Physical: trauma, radiation, chalcosis, siderosis
 4. Toxic: Steroids, cholinesterase.
2. JRA there are several forms
 1. Poly with RF-, __, ANA 25%, RF-, Ant uveitis:rare
 2. Poly with RF+, __, ANA 75%, RF+, Ant uveitis:never
 3. Pauci early (2-5), __, ANA 60%, RF-, Ant uveitis:30%
 4. Pauci late, __, ANA and RF-, but B27 pos, Anterior uveitis 15%
 5. Systemic (still's), __, ANA, RF, B27-, ant uveitis rare
 - (1) Treatment is with steroids
 - (2) NSAIDS
 - (3) Systemic immunosuppressives
 - (4) F/U depends on risk. Less than 7 years with ANA pos and Pauci every3-4 months

4. Koeppen nodules?????

1. Located at pupil margin and can occur in **both** granulomatous and non-granulomatous inflammation. Whereas Busacca are in the stroma and **only** occur with granulomatous inflammation.

5. Eye with PS. Ddx of PS. Discuss sarcoidosis.

1. Ddx of PS is same asuveitis except for FHI, Posner Sclossman and traumatic iritis.
2. Sarcoidosis is amulti system disease. Lungs, skin, and eye disease. 30% of eyes affected.

3. Ocular: uveitis (usually pan) bilateral and granulomatous
 - Posterior segment: granulomas, vitritis, vasculitis, NV
 - Conj and eyelid nodules
 - LGT
 4. Tests: CXR, gallium, ACE, Ca, anergy
6. Iris pigmented tumor. It will be a nevus. Ddx, w/u, management?
 1. Ddx: Nevus, MM, CB with iris invasion, mets, granuloma, ICE, Lisch, JxG, FB.
 2. Histopath: Spindle A and B cells. A predominate in nevi. Epitheloid cellsif malignant.
 3. No work up photographic documentation is all that needs be done.
 4. If it grows you must suspect malignant transformation; Malignant characteristics:
 1. Non Small size (3mm basal diameter, <1mm thick)
 2. Pigment dispersion
 3. Pupil peaked
 4. Ectropion uvae
 5. Cataract
 6. Documented enlargement
 5. If deemed malignant then sector iridectomy vs iridocyclectomy are surgical options but because of the extremly low metastatic rate (2%) some elect to observe unless rapidly enalrgeing. Some even watch if angle is involved. As most of these are not rapidly growing enucleation is low on option list.
7. True exfoliation slide. Ddx
 1. True exfoliation vs. PXF
 2. Infrared radiation or intense heat can cause the anterior layer of the lens capsule to peal off.
 3. No treatment
8. Types of cataracts
 1. Congenital: AD, AR, XR
 2. Age-related
 3. Inflammation: uveitis all types of anterior, intermediate and pan-uveitis
 4. Toxic: steroids, pseudocholinesterase inhibitors, phenothiazines, amiodorone, radiation, copper, iron, electricity
 5. Trauma
 6. Metabolic: DM, low Ca
 7. Syndromes: galactosemia, Alport's, Treacher-Collins, Lowes, Fabrys, Myotonic dystrophy, Wilson's
 8. CNS:NF II, Zellweger, Norrie's
 9. Secondary: post glaucoma, post RD, post vitrectomy
9. Retrobulbar hemorrhage after retrobulbar
 1. First apply pressure to limit extent

2. Then measure IOP and check RAPD and CRAO
 3. Lateral cantholomy and inf cantholysis and if needed sup cantholysis
 4. B-blocker's, CAI, Mannitol
 5. AC tap
 6. Tenon's decompression
 7. Orbital decompression if all else fails
10. Histology of angle with macrophages, Ddx
1. Ddx: Macrophages either have protein from lens:phacolytic or blood: hemolytic
 2. Phacolytic glaucoma occurs with hypermature cataracts. HMW proteins egress out of the lens into the AC where they block and incite macrophages which further incite TM blockage. Children don't have these HMW proteins.
11. Pre-op med for cataract surgery and why?
1. Lid care ant treat occult dacryocystitis is the most important thing
 2. Pre-op Ab't, nobody has proven benefit in preventing endoph but it definitely reduces bacterial counts and until such time as it is disproven it remains the "standard of care". Take your pick, poly trim (misses: anaerobes, pseudomonas, MRSA), ocflox (misses: anaerobes and some strep), gentamycin (no anaerobic and weak for gm positives).
 3. 5% poviodine to cul-de-sac and lashes is key (proven!)
 4. Dilation plus NSAID
12. Unilateral cataract ddx.
1. Trauma
 2. Secondary to glaucoma surgery, vitrectomy, laser, FB
 3. Inflammatory: FHI, uveitis any cause
 4. Congenital: PHPV, Peter's, posterior lenticonus
13. Evaluation with no view to back with cataract.
1. Light projection, RAPD, Red Maddox rod test, Purkinje image all done in office
 2. PAM or laser interferometry? (Weak answer but say it anyways)
 3. B scan
 4. Blue field entoscopy
 5. ERG
14. Hypopion ddx
1. Infectious: post-op, endogenous, trauma, bleb associated
 2. Inflammatory: Behcet's, HLA-B27 (esp Reite's, IBD, psoriatic and pure HLA-B27)
 3. Pseudo: RB, lymphoma, ghost cell "candy stripe"
 4. Contiguous inflammation: corneal ulcer, scleritis
 5. FB esp copper >85%
 6. Drugs: vitrase

15. Aniridia it's inheritance patterns and talk about.

1. **Aniridia**

- AD: 2/3; sporadic: 1/3, there is an AR form
- 1/60000
- chromosome 11
- VA < 20/200
- sporadic aniridia assoc: Wilm's (33%), MR, GU abn.
- U/S or IVP every 3 months if sporadic

Signs

- 1) cornea: pannus (epithelial metaplasia), keratoconus (Duanes')
- 2) angle: angle closure glaucoma
- 3) iris: hypoplasia
- 4) lens: subluxation, cataract
- 5) retina: foveal hypoplasia
- 6) optic nerve: hypoplasia
- 7) neuro: nystagmus
- 8) EOM: strabismus

16. Ddx of leukocoria

1. **Ddx of Leukocoria (white pupil)**

- 1) PHPV (small eye)
- 2) RB (6-18 months)
- 3) toxocara (young child)
- 4) Coat's (child)
- 5) ROP
- 6) coloboma
- 7) cataract
- 8) RD
- 9) retinal dysplasia
- 10) uveitis
- 11) myelinated nerve fibers
- 12) other tumors
- 13) vitreous hemorrhage
- 14) morning glory disk
- 15) Norrie's disease
- 16) FEVR
- 17) combined hamartoma

17. Heterochromia ddx and discuss siderosis

1. Ddx Light iris affected: FHI, Horner's, uveitis, JXG, RB, Waardenburg, tumors
2. Ddx Dark iris affected: FHI, melanoma, tumors, siderosis, chalcosis
3. Siderosis occurs from ferric ions>>initiate Haber- Weiss rxn>>oxidants>>lipd peroxidation, sulfhydryl oxidation and polymerization
4. Epithelium affected
 1. Anterior lens deposits
 2. Heterochromia
 3. Non reactive mydriasis
 4. Secondary OAG
 5. RPE changes

6. PR nyctalopia
 7. ERG: early Abig>>B diminishes>>extinguishes
18. Histopath of DM
1. NVI * showing thinned walled vessels on anterior iris surface.
 2. Lacy vacuolization of iris * involves PE of I swollen with glycogen.
 3. Thickened BM of CB epithelium.
 4. Loss of pericytes on retinal vessels.
 5. NVE
 6. CWS, D/B hem, NFL hem, HE
19. Epithelial downgrowth showing cyst.
1. Epithelial down growth is not as common as fibrous downgrowth
 2. Fibrous downgrowth is more benign.
 3. Epithelial downgrowth has three presentations: epithelial cyst, iris pearl and sheet of downgrowth
 4. Manage only glaucoma with fibrous downgrowth
 5. Confirm: 1) laser to iris: ED will blanche, 2) confocal microscopy, 3) bx
 6. Remove epithelial downgrowth
 1. Pearl tumors are rarely treated because they can remain unchanged
 2. If pearly tumor enlarge then excise with wide local excision of iris
 3. Cyst: suck contents out first with limbal needle the double freeze thw cryo to destroy remaining epi in cyst. Then excise everything affected.
 4. Epi downgrowth in sheets: cryo, EtoH to endothelium, vitrectomy to iris and vitreous affected
 5. NB: Outline affected iris pre-op with argon laser, you must destroy the fistula
 6. Glaucoma managment
 7. If vision is 20/20 with either cyst or sheet: remove. Timing is everything. You have a chance when it is small and not diffusely involving the anterior chamber.
20. Cataract complications
1. Flat chamber post-op
 1. Wound leak +/- iris prolapse>>do Seidel with globe pressure
 2. Pupil block angle closure glaucoma>>gonioscopy>>Zeiss compression
 3. Malignant glaucoma>>is central AC shallow>>UBM
 4. Capsular block syndrome>>UBM>>tx with YAG to ant capsule
 5. Cyclodialysis cleft>>gonio>>Tx: cylco, laser, sew
 6. Inverted PCIOL>>direction of haptics>>PI
 7. SCH delayed>>look at pos pole & U/S
 8. Choroidal effusion>> look & U/S
 2. Wound leak you can try: Aq supp,  steroids, irritating Ab's, patch, CL. If after 3-5 days or at any time if endo touch then go back to OR and repair. You must go to OR if iris prolapse. Excise iris if out for more then 24hours.
 3. Vireous wick: You can observe if VA good and no CME. If large amount to wound

despite sequelae it's better to remove sooner than later.

21. Endophthalmitis

Endophthalmitis

Post op	49-76%	CATARACT (US) 0.072%, (Pakistan) 0.5%
Trauma	16-23%	2.4-30%
Bleb	4-18%	EARLY 0.3% LATE 0.2-18% WITH ANTIMET: 5.7%
Endogenous	0-15%	

Bugs

Post cataract: Staph epidermidis, Staph aureus, Strep species (not pneumococcal)

Bleb: Haemophilus and Strep

Trauma: Bacillus and Staph epi

EVS (<6 weeks)

94% loss of vision

75% pain

85% hypopyon

5.2% wound abnormality

32% red reflex

68% culture positive

Work Up of Endophthalmitis

1. B-scan ultrasound (poor outcome correlates: combined vit&subhyloid opacities, chor det, RD)
2. AC tap: 25G needle on tuberculin syringe, remove 0.1ml
3. VIT TAP: 23G needle 1/2inch needle, 3.5mm to limbus, remove 0.2ml
4. Plate: agar, chocolate, Sabouraud, thioglycollate, anaerobic
5. Gram and Giemsa stain
6. Rest of sample mixed with 95% alcohol and sent to path

Ddx of post op inflammation

1. Severe post inflammation
2. Lens induced uveitis (granulomatous)
3. UGH
4. Abrupt corticosteroid cessation
5. Systemic uveitis
6. SO

Treatment Acute Post-Cataract Endophthalmitis

1. INTRAVITREAL
Vanco 1.0mg in 0.1ml
Ceftazidime 2.25mg (EVS used Amikacin 200-400i g, rare but present risk of macular infarct)
Dexamethasone 400i g (controversial)
2. SUBCONJ
Vanco 25mg in 0.5ml

Ceftazadime 100mg
Dexamethasone 6mg

3. **TOPICAL**

Vanco 50mg/ml
Gentamycin 14mg/ml
Atropine 1%
PF 1%

Treatment Chronic Post Cataract Endophthalmitis (1. P acnes, 2. S epidermidis, 3. Fungi)

1. Tap and intarvitreal Ab's is first step
2. If post capsular pearls are seen then strong consideration to PPV capsulotomy + Ab's
3. If above two fail then en-bloc IOL bag removal with ACIOL.

Treatment of Bleb Endophthalmitis

1. Is vitritis present?
2. If no
culture AC and VIT??? controversial, may start tx and watch closely?
fortified vanco (50mg/ml) + oculflox (0.3%) or gentamycin
topical steroids and cycloplegia
3. If yes then choice is
 - a. PPV with VIT Ab's and Subconj Tx
 - b. Tap and inject

Post Trama Endophthalmitis

1. First culture AC and VIT
2. PPV and Vit Ab's, sub conj and systemic
3. Controversy if Vanco/ceftazadime more effective then adding clinda 1mg/0.1ml (I would give all three.)

Endogenous Endophthalmitis

1. Generally systemic antibiotics.
2. Tap AC and Vit.
3. Vit Ab's plus amphotericin 51 g/0.1ml?
4. If severe vitreous opacities then Vit
5. If fungal:
 - Candida with mild retinitis may try fluconazole 200-400mg po qdaily
 - Candida progressive then systemic amphi
 - If severe vitreous opacities then Vit
 - If aspergillus immediate VIT

Concentrations and Dosages of Principal Antibiotic Agents Used for Treatment of Endophthalmitis

Intraocular	Subconjunctival	Topical	Intravenous*
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Amikacin	0.4 mg	20 mg	14 mg/ml	15 mg/kg/day (doses q8h)
Ampicillin	0.5 mg	100 mg	50 mg/ml	4-12 g/day in 4 doses
Cefazolin	2.25 mg	100 mg	50 mg/ml	2-4 g/day in 3-4 doses
Ceftazidime	2.25 mg	100 mg	50 mg/ml	2 g/day in 2 equal doses
Chloramphen	1 mg	50-100 mg	5 mg/ml	50 mg/kg/day (in doses q6h)
Clindamycin	1 mg	15-50 mg	50 mg/ml	900-1800 mg/day in 2-3 doses
Gentamicin	0.1 mg	20 mg	14 mg/ml	3-5 mg/kg/day in 2-3 doses
Methicillin	2 mg	100 mg	50 mg/ml	6-10 g/day in 4 doses
Tobramycin	0.1 mg	20 mg	14 mg/ml	3-5 mg/kg/day in 2-3 doses
Vancomycin	1 mg	25 mg	50 mg/ml	2 g/day in 2 equal doses

Risks for Post Cataract Endophthalmitis

Pre-Op: Blepharitis, conjunctivitis, NLD obstruction, eyedrops

Op: vit loss, long surgery, contaminated irrigation solutions

21. Dislocated IOL. Sunset.

6. Sunset: inferior haptic has gone through zonules with sulcus IOL
7. Acute event non progressive
8. If VA OK and seems stable....leave it.
9. If bisecting pupil, haptic touching retina options are
 1. Reposition and sew to iris (poor choice)
 2. Scleral fixation
 3. ACIOL

22. AACG

1. Check VA, RAPD, IOP, gonioscopy
2. Indent first
3. Pilo and aq suppressants (beta blocker and apraclonidine) and PF
4. Diamox
5. Wait 45 minutes: attack broken yes then wait 1-3 days for cornea and inflammation to decrease then do PI
6. No then consider osmotic agents
7. **Osmotic agents**
 - 1) *IV agents*
 - a) mannitol (20%)
 - dose: 350-500 cc (1-2 g per kg; 70 kg = 70 g = 350 cc of 20%)
 - b) urea
 - 2) *oral agents*
 - a) glycerin: raises blood glucose

- b) isosorbide: no effect on blood glucose; causes diarrhea
 - side effects*
 - 1) cardiac overload (less for glycerin)
 - 2) subarachnoid or subdural hemorrhage
 - 3) urinary retention
- Precaution: heart failure, renal failure, large prostate (put in catheter for men)

8. **iridotomy:** PI - YAG and Argon

Technique:

Iridectomy Lenses:

Abraham lens (1973): modified Goldmann lens; has +66D plano convex button

Wise lens: has +103 D button

- location: midperiphery of constricted pupil (2/3 from pupil margin) between 10:30 and 1:30

Preop

- 1) apraclonidine drops
- 2)- miotic drops

a) Argon PI

- 50 micron spot size, 0.2 secs, 800 mW
- consider for patients on anti-coagulants (bleeds less)
- usually accomplished in 10 burns
- The easiest irides to penetrate with the argon laser are hazel and light brown.
- The hardest are light blue irides with minimal pigment, and very thick dark brown irides.

b) YAG PI

- 5 mJ shots; 1 pulse per shot

Post op

- measure IOP for 2 hours after treatment (>90% of spikes within 2 hours)
- PF QID x 1 week post laser
- pilo can be continued if on Pilo prelaser
- first postop exam:
 - i) gonio to evaluate angle and assess for PAS
 - ii) dilate to ensure plateau iris not present and to examine fundus
- followup for 6 weeks to evaluate patency

Indications for PI

- 1) acute ACG
- 2) occludable angle: positive provocative tests, narrow angle
- 3) phacomorphic glaucoma
- 4) aphakic

- 5) pseudophakic (ACIOL)
- 6) malignant glaucoma
- 7) silicone oil (6 o'clock)
- 8) plateau iris (does not work on all)
- 9) nonperforate surgical iridectomy
- 10) Fellow eye of a patient with acute angle-closure glaucoma
- 11) Chronic angle-closure glaucoma
- 12) Iris bombe from posterior synechiae
- 13) Prior to ALT to open the angle approach and facilitate treatment

PI complications

- 1) failure to perforate
- 2) acute rise in IOP
- 3) late closure
- 4) cornea, retinal burns
- 5) cataract
- 6) corectopia (especially argon)
- 7) post. synechia
- 8) hyphema
- 9) diplopia

pupilloplasty

- 1) for patients with miotic pupils on pilo
- 2) in acute ACG, if PI and iridoplasty are not possible due to cloudy cornea, this can break the attack by pulling the pupil away from the lens

gonioplasty/iridoplasty

Indications

- 1) plateau iris
- 2) to facilitate PI
- 3) to facilitate ALT
- 4) nanophthalmos

Technique

- 1) topical anesthesia
- 2) 500 micron spot 0.5 sec burn, 300 mW
- 3) six applications per quadrant leaving space in between the applications
- 4) Patients should be told that the treatment might cause slight permanent dilatation of the pupil and that it will cause delayed discoloration of the peripheral iris.

peripheral laser synechialysis

- in early synechial closure, to open a synechially closed angle
- should be attempted before surgical goniosynechialysis

1. Surgical PI if all above fails
23. Oil droplet cataract. Discuss the differential diagnosis of infantile cataracts.

Ddx of Infantile cataracts (I'M HOG)

Bilateral

1. Idiopathic (60%)
2. Hereditary (30%)
 - AD (most common), AR, XL
 - 3. Genetic, metabolic & systemic disease (5%)
 - Hallerman-Streiff syndrome (90%, can resorb)
 - Lowe's syndrome (XL)
 - Smith-Lemli-Opitz
 - Galactosemia (AR)
 - Hypoglycemia
 - Trisomy Down's (21%), Edward (28%), Patua (13%)
 - Alport syndrome (AD)
 - Myotonic dystrophy (AD) iridescent
 - Fabry's disease (XL)
 - Hypoparathyroidism
 - Marfan's syndrome
 - Conradi syndrome (chondrodyplasia)
 - DM
 - Peroxisomal (Zellweger & Conradi)
 - Wilson's
 - 4. Maternal infections (TORCHS) (3%)
 - Rubella
 - CMV
 - Varicella
 - Syphillis
 - Toxo
 - HSV
 - 5. Ocular abnormalities (2%)
 - Aniridia (AD 2/3, sporadic 1/3)
 - Anterior segment dysgenesis
 - Microphthalmia

Unilateral (MOM IT)

1. Idiopathic (80%)
2. Ocular abnormalities (10%)
 - PHPV
 - Posterior lenticonus
 - Anterior segment dysgenesis
 - Posterior pole tumors
3. Trauma (10%)
4. Masked bilateral cts
5. Maternal infection>Rubella (unusual)

Galactosemia can be caused by three different enzyme abnormalities.

Galactose-1-phosphate uridyl transferase
galactokinase
UDP-galactose-4-epimerase

Classic galactosemia is caused by galactose-1-phosphate uridyl transferase. The systemic findings include malnutrition, hepatomegaly, jaundice and mental deficiency. If untreated the disease is fatal. 75% get cataracts. Oil droplet is the classic cataract seen. Eventually a total cataract develops. The cataract can be reversed if treated early with diet devoid of all milk products.

Test by checking urine for non-glucose reducing substance in the urine

Work up for congenital cataracts

A) Unilateral

- 1) history - age of onset, family history
- 2) ocular exam: PHPV, lenticonus, RD, mass
- 3) Labs: TORCH Titer, VDRL (TORCHS)

B) Bilateral

- 1) History - family history, age of onset
- 2) development of child history
- 3) complete ocular exam
- 4) pediatrician and genetics consult
- 5) Labs: TORCH Titre, VDRL, urine reducing substances (galactosemia)
- 6) Optional: urine for a.a. (Lowe's), RBC galactokinase, calcium, phosphorus

24. Scleritis phot. Discuss ddx, w/u and management

4. Types

1. Diffuse
2. Nodular
3. Necrotizing
 1. With inflammation
 2. Without inflammation
4. Posterior

5. Etiology

1. Idiopathic
2. CTD: Wegener's, PAN, SLE, RA, RPC, AS
3. Infectious: syphilis, HSV, HZV, TB
4. FB
5. Hypertension
6. Gout

6. Know how to differentiate from episcleritis

7. W/U

1. CBC, ESR, ANA, RF, FTA-ABS, VDRL, C-ANCA, P-ANCA, uric acid,
2. PPD, CXR, SI joint x-ray

8. Treat:

1. Oral NSAIDS. Indomethacin SR 75mg BID best for mild to moderate scleritis, use gut protector as well
2. If NSAIDS fail or moderate to severe then use oral prednisone 1mg/kg

3. Immunosuppressives if these fail and when patients have systemic vasculitis.
Wegener's: cyclophosphamide
26. NVG
- Etiology: DM, CRVO, OIS, CRAO, others: BRVO, uveitis, RD, tumors, radiation
 - Work up FA & carotid doppler if you can't see to the posterior pole and you are unsure of what the cause of the NVG is. B scan U/S