

OPTICS

Q:Hyperopia:

Manifest: Absolute which is amount of plus needed to just see 20/20, facultative amount of plus you can add beyond absolute and still see 20/20

Latent: Is amount of hyperopia remaining thaty must be pharmmolcologically relaxed.

Q: Pt c hyperopia and X. Give absolute hyperopia.

Q: SRK and individualized A. $SRK=A-2.5AxI-0.9K$ A constant is derived empirically from many cases and gives a est. of where the iol sits in the PC. Each surgeon can further refine there A const. fOr a given IOL type by back solving SRK for A once the post op rx is known.

Glaucoma

Q: High IOP deep AC flat bleb.

Blockage of pre, @, post sclerostomy. Vit, iris, blood, fibrin, fibrosis, healon.

Gonioscopy

1. massage if fibrin, blood or fibrosis
2. pilo if iris in sclerostomy, viscoat if that fails
3. fibrin: ?TPA, ?YAG
4. suturlysis

Q: pigmentary glaucoma

Management

1. Observe for progression
2. Scaffold iris or not?

Consider PI vs. pilocarpine

Pigmentary dispersion was first described by Von Hippel in 1899 . Later Levinsohn suggested that glaucoma in this condition is related to pigment released from iris pigment epithelium. Many years later several investigators agreed or disagreed about the association between pigment and glaucoma . In the recent literature more evidence appeared supporting the association between pigment and glaucoma . Scheie & Cameron in 1981 studied a large number of patients confirmed the association .

Pigmentary dispersion syndrome (PDS) is a bilateral condition , although it can be asymmetric. It affects males more often with a peak at the fourth decade of life (35-45) . Over 80 % of patient are myopic and usually of mild to moderate myopia . It is a relatively common condition and often is overlooked. In the recent literature an autosomal dominant inheritance pattern was described in 4 families from Europe .The gene was mapped to 7q35-36 locus . The ocular findings in PDS is bilateral as described earlier. Krukenberg's spindle, a vertical deposition of pigment is due to aqueous convection currents in the anterior chamber. Endothelial deposits of pigment cells are not believed to interfere with endothelial function. The presence of pigment in the anterior chamber is often mistaken as anterior uveitis, our patient gave a history of treatment with topical steroids for presumed uveitis . Iris transillumination is characteristically in mid - periphery in contrast to pseudo-exfoliation which is at the pupil margin . Counting iris defect spokes can be helpful in following the progression of these patients . The most accepted

theory for pigment release is posterior irido-zonular contact resulting in mechanical friction and release of pigment from posterior iris surface . This has been clearly shown pathologically. What causes the iris to be posteriorly displaced and causes the contact ? The most accepted explanation is that during blinking, there is transient increase in anterior chamber pressure compared with posterior chamber (due secondary to reversed pupil block) . The sagged iris becomes bows backwards resulting in irido-zonular contact. Pigment will then be deposited in the angle. Although the pigmentation is seen 360 degrees, it is best seen at the 12 O'clock angle. Occasionally a second line of pigment anterior to TM is seen (Sampaolesi line). Another site of pigment deposition is at the posterior lens capsule (Scheie`s stripe) . The degree of pigment in the angle poorly correlates with intraocular pressure . The condition was classified into 2 stages clinically & pathologically. Stage 1 (reversible stage) were the liberated pigment enters the intratrabecular spaces & being phagocytosed . At this stage intraocular pressure (IOP) is normal but may show degree of fluctuation higher than normal physiological diurnal fluctuation . In addition there are some provocative factors that result in increase IOP (due to transient trabecular decompensation) , these factors include 10 % phenylephrine & exercise both result in increase pigment release .

Stage 2 is the irreversible stage pathologic ally seen as trabecular sclerosis. At this stage aqueous suppressants play a small role in management . The prevalence of glaucoma & ocular hypertension in eyes with PDS is variable.The prevalence varied from 6 % (202 patients) by Evans to 25.6 % (407 patients) by Scheie & Cameron .

Management of this condition can be challenging . Many people advocate close follow up of these patients with frequent assessments including visual fields especially because some of these patients may have tilted discs which is often difficult to follow. Once there is any suspicion about glaucoma treatment is recommended . Recently a prophylactic peripheral iridotomy has been shown by more than one group of investigators to be effective . This has clearly been demonstrated by UBM were after PI iris position has changed as clearly demonstrated initially by Potash et al (Ophthalmology :101 # 2 , Feb 1994) . Other treatment options are medical with aqueous suppressants. Pilocarpine is less commonly used because of its side effects in younger individuals. However, some people tolerate the slow release (Ocusert) better than pilocarpine drops . It is important to note that the risk of retinal detachment in these patient is higher than the general population. Filtering surgery is an option in uncontrolled increased IOP . Greater percentage of eyes with PDS (23.5%) require filtering surgery when compared with eyes with primary open angle glaucoma (14.5%).

Q: Plot of mean deviations with time. Nothing to say.

Q: Uveitic glaucoma

Inflammatory cells in TM

PAS

PS with bombe

Trabeculitis

NVG

Steroids (1. PG alteration, 2. Supp endo phagocytoc, 3. Alt extracellular matrix GAG, 4.

Increase tight junctions, 4. Stabilize lysozymes, 5. Increase cross linked actin)

Q: Accomodative ET with high AC/A. Nonrefractive accomodative ET

Indications:

If pt does not fuse in distance do not give bifocals. If fuses in distance it is controversial weather bifocals do anything. Parks argues it helps by giving fusion at near.

1: FCR. Wait 1 week and insure compliance. If not add ATR for a few weeks. Repeat FCR in 1-2 months. If amblyopia tx'd and residual ET>10 at distance then surgery. If borderline then I would initiate a PAT. And if deviation increases to surgical level then operate.

Q: DDX leukocoria and Coat's to make dx and tx.
RB, PHPV, Coats', Toxo. Cataracts, RD, Rdysplasia, angiomatosis retinae, astrocytoma, coloboma, vitritis, myelinated NF.

Coats' dx: Observing telangiectatic vessels. Light bulbs. ERD. Male, unilateral
FA. Laser to abnormal vessels, cryo, SB, vit.

Q: Post IO surgery pt develops internal ophthalmoplegia. Management?
I don't know. ?Wait for recovery. Bifocal and miotics??

Cornea & External

Chemical burns

Immediate: irrigation, DEL and sweep

Intermediate: PF q3 hrs, lubrication, TTC and acetylcysteine, diamox if IOP high, vit C

Consider: intense lub, CL, limbal autograft, tarsorrhaphy, flap, PKP

Q: Unilateral blepharitis: neoplastic sebCa, infectious: staph, lice, HSV contact dermatitis, toxic, floppy eyelid syndrome

Q: Trachoma: TTC for 3 weeks and treat all contacts. If younger than 7 give erythromycin for 3 weeks. Lid hygiene and topical ilotycoin.

S: surgery for trichiasis

A: Ab's

F: facial cleanliness

E: Environmental hygiene

Q: international causes of blindness

1. cataract (15 mil)
2. trachoma (8 mil)
3. onchocerciasis (400 000)
4. xeroophthalmia (300 000)
5. glaucoma #1 blacks (NA)
6. DM #1 (NA)
7. ARMD >50years #1

Neuro (Nicolle)

Q: tuberculum sellae: front anterior part of sphenoid body where anterior chiasm sits. Important in meningioma.

Q: Hypotropia:

Paralytic: IV, III, SR, IR, IO, SO

Restrictive: trauma (BOF c restriction), TAO, OIS, Fibrosis syndrome, MED, previous surgery with scarring, radiation, myositis, mets, mass

Q: ptosis

Q Ddx of painful Eophthalmoplegia

Orbital:

infect: OC, mucor, hydatid cysts

inflamm: pseudotumor, ruptured dermoid, Wegener's (vasculitis)

neoplastic: rhabdo, bleed lymphangioma, mets, adenoid cystic, SCC, NPC,

Apex:

Same causes except OIS.

Cavernous sinus:

Vascular: CC fistula, aneurysm, dissecting aneurysm, thrombosis, pituitary apoplexy

Infectious: syphilis, Lyme, TB

Inflamm: sarcoid

Neoplastic

W/u: imaging CT/MRI, FTA-Abs, TB, angiogram, search mets