

RCP exams

Surgery

Q: Phaco and deep AC

DDX

Broke bag, zonular dehiscence, prior vitrectomy, aspiration plugged

1. Stop case
2. Investigate
3. Tamponade with viscoat
4. Convert if not cracked or all four pieces left
5. Try phaco if small piece left

Q: Lid trauma post hockey injury with canalicular laceration

1. ABC
2. Look for lid laceration
3. And canilicular laceration
4. Full eye exam
5. DT, last meal, consent
6. Intubate the system bla bla

Q: Comps of retrobulbar, peri,

1. Perf
2. Hem
3. Brainstem anestehsia
4. Optic nerve contusion
5. Pupil
6. EOM pareisis
7. CRAO
8. Ptosis

Peri you don't have optic nerve needle damage but it takes longer. Toxicity is higher because you inject.

Sub-tenon's: less pareisis, cosmetic

Q: III palsy

Aneurysm, TA, MV, compression, etc.

Temporal artery: br of ECA. Superficial and deep branch. The superficial is above the temporalis muscle. It br into frontal and parietal br.

Don't forget the Cohen-Allen test of the eye.

Pathology:

RB and it's features.

Corneal opacification in center. Lipshultz bodies.

Orbital tumor with bilateral muscle enlargement;
TAO, myositis, CC, mets, trichanosis,, leukemia, lymphoma, amyloid

Retina

Q: BRVO tx and follow up

Wait 3 month

20/40

FA

Tx: ME, NVD

Q: CRVO

>10 DA of non perfusion: 32%

>75 DA of non perfusin 52%

NI convert to I 16-18%

Indeterminate: 83%

Tx if NVI & ANV

Ischemic: close follow up with undilated pupil and gonio exam q1month

NonI: follow q2months

Q: CSME

1. RT 500micron

2. HE 500micron with associated with RT

3. RT within 1DD and 1 DA in size

Retinal tears

When tx

Symptomatic HT

Asymptomatic HT if risk factors or traction

Symptomatic OT

Risk factors for RD

1. aphakia, pseudophakia

2. myopia
3. prior RD
4. fam hx rd
5. vitreous status

Lattice alone

Q: choroidal folds diff from retinal folds
U/S and FA and hyperopia

Q: CNVM
Treat all well defined except big subfoveal with poor vision.

Glaucoma

Q: iopidine and its mode of action
Alpha 2 agonist it decrease Aq.
Side effects: mydriasis, lid elevation, follicular conjunctivitis, conj blanching, tachy
Systemic: dry mouth, lethargy, vasavagal effects

Q: ALT how do you do it.
You know how to do it.
wait 4-6 weeks for response
80% respond 1 year, attrition 10% per year
3% persistent elevation of at least >5mm that need surgery

Q: total hyphema
Indications for AC wash
Blacks & sickle pts: 24mm hg for 24 hours
50mm for 5
35mm for 7
total for 9
blood staining of any amount

Q: pseudophakic and PKP and vit in AC at 12, deep AC, high IOP on max meds

Iop and gonio
Anterior vitrectomy with trab and mitomycin

Q: cyclodestructive
Cyclophoto is preferred
Indications:
VA<20/400

All other procedures failed
Medical reasons can't have surgery

Complication
Hypotony
Ant seg ischemia
Pain
Iritis
Visual loss
Malignant glaucoma
SO

Settings: slow and long like good sex.

Q:

Neuro

ONTT
Swollen disc: management of PC

Supracellar mass ddx

INO

III

Rollins Cornea
Diffuse KP's: FHI, HSV, HZV, CMV, Toxo
FHI

Amyloidosis conjunctival
Primary localized vs systemic

Staging of Fuchs

Three stages of Fuchs' endothelial dystrophy are recognized. 26,27 The three stages usually evolve gradually over a period of 25 years, and like other corneal dystrophies are usually bilateral but asymmetric.

The first stage is the onset of corneal guttata, usually in the fourth decade of life. Subjective symptoms rarely occur until the fifth or sixth decade. During asymptomatic phase, endothelial guttata and pigment dusting can be seen by slit lamp examination on the central corneal endothelium (Fig. 6) and by specular reflection (Fig. 7). The excrescences can become more numerous and confluent so that individual guttata are

completely lost in the beaten-metal appearance of the endothelial surface. The central cornea is involved first, and as the disease progresses it spreads to the periphery.

In the second phase of the disease, blurred vision, glare, and halos around lights develop because of incipient corneal edema in the stroma and epithelium. Epithelial edema can be seen as small droplets (bedewing) on retroillumination with the slit lamp. Epithelial microcysts coalesce to form bullae, which produce varying amounts of pain when they burst; hence this stage gets its name, bullous keratopathy (Fig. 8). Striae form in Descemet's membrane as the cornea thickens posteriorly due to stromal swelling. The arc of Descemet's membrane from limbus to limbus is shortened, causing the wrinkles in Descemet's called striae. The microcystic epithelial vesicles may break, causing foreign body sensations and severe pain with more extensive corneal epithelial disruption. Recurrent corneal erosions, microbial ulceration, and persistent pain may occur in this stage. Corneal sensitivity is usually reduced.

Fig. 8. Giant bulla (arrow) in edematous cornea secondary to Fuchs' dystrophy.

The third and final stage of Fuchs' dystrophy is marked by the development of a pannus of subepithelial connective tissue along the epithelial basement membrane. Peripheral corneal vascularization can also occur at this stage. The degenerative pannus markedly reduces vision, but at the same time it reduces painful epithelial bullous formation and the patient is more comfortable. The epithelial edema in stage 3 is reduced by the pannus, but the stromal edema remains unchanged. Even with the cornea markedly thickened, patients are comfortable because of stabilization of the epithelial layer of cells by the pannus of scar tissue.

Optics
Blue: 488
Yellow: 577
Green: 514
Red: 630-645
YAG: 1064

Astigmatism
Jackson's cross
Retinoscopy
Keratometry
Topography
Stenopeic slit

CL Bifocal
RGP with bifocal segment
Diffraction
Concentric rings

Goals of monovision

Keratometer

Image fixed and object fixed

Uses both reflecting and refracting power of cornea.