Epithelial (Anterior) Basement Membrane Dystrophy (EBMD or ABMD) or Map Dot Fingerprint

- 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
  - its late onset support a degeneration vs. dystrophy

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 approx 10%

ABMD

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

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

- Often referred to as:
  - maps
  - dots
  - fingerprints
EBMD-Negative Staining

Symptoms of RCE
- Pain
- Redness
- Decreased Vision
- Tears

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

Treatment Options

Irregular Astigmatism
- GPORx (Gas Permeable Over Rx)
- PTK

Recurrent Erosions

Bandage Contact lens Focus
Night/Day BC 8.4/13.8
Plano 20/20+
CW 6 pm
Other available treatments

- Debridement
- Stromal Puncture
- PTK
- Amniotic Membrane Transplant e.g. Prokera

Broad Beam Laser

- Makes the bed “sticky”
- Very light treatment

EPITHELIAL DEBRIDEMENT

- Soften epithelium
- 1-2 gtt topical anesthetic
- Use spatula like buttering bread
- Start peripherally using long strokes
- Remove flaps by pulling edges toward center
- Tx abrasion (>50-100%)
  - Recurrence Rate 18%

Stromal Puncture

Diamond Burr Polishing

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

Amniotic Membrane Transplant

- Biologic tissue that promotes healing of the ocular surface
  - antiangiogenic
  - antiscarring
  - antimicrobial
  - anti-inflammatory
- Also used in
  - Burns
  - Neurotrophic ulcers
  - Stem cell damage
  - Persistent epithelial defects
Amniotic Membrane Grafts (AMG)

- Biotissue Prokera, Prokera Slim, Prokera Clear, Amniograft, & Amnioguard

- IOP Ophthalmics-Ambiodisk

Role of Tetracyclines

- Inhibit key metalloproteinases important in disease pathogenesis
  - Can be used with topical steroids
    - Doxy 50 mg bid and FML tid for 4-8 weeks

ProKera

- ProKera
- ProKera Slim
  - Thinner outer ring
  - More comfort and more contact with the cornea tissue
- ProKera Plus
  - Multiple layers of amniotic membrane
  - Longer contact time
  - Chemical burns, Stevens Johnson Syndrome, and severe corneal ulcers

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 recommend taking sun protection

ProKera Clear

- Trephinated 6mm aperture allowing some visual potential
- Chronic inflammatory cases with the limbus being the targeted area of biologic boost and healing

Side Effects of Tetracyclines

- Side effects include gastric discomfort, phototoxicity, effects on calcified tissues, vestibular problems, pseudotumor.
- Pregnancy Category D.
  - Tetracyclines are attracted to embryonic and growing bone tissue.
  - Depress growth of long bones in pregnant women/children.
  - Cause changes in both deciduous and permanent teeth during the time of tooth development (includes discoloration and increased caries)
- Contraindicated in:
  - Women in the last half of pregnancy
  - Lactating women
  - Children under 8 years of age
My Approach to RCE or Visually Significant EMBD

- Epithelial Debridement
- Diamond Burr Polishing
- PTK with broadbeam laser
  - Treat centrally and peripherally to minimize refractive shift
- Bandage CL
- Steroid and Antibiotic for 2 weeks
- Maximize tear film

EBMD and LASIK

Case

- A 68-year-old woman with a history of poorly controlled diabetes presents with poor vision of the left eye for about 2 months.
- She notes an episode of left eye pain 2 months ago that lasted for a week

EBMD and Cataract

- EBMD will impact final outcome
- Irregular topography will lead to inconsistent vision and outcome
- Treat with PTK, then repeat measurements, then cataract surgery

EBMD and Cataract

- Patients who have a history of EBMD may not be ideal candidates for LASIK and should be carefully screened for prior to surgery
  - HOWEVER, if good BCVA with MRx, then can proceed with PRK
Case Presentation

<table>
<thead>
<tr>
<th></th>
<th>OD</th>
<th>OS</th>
</tr>
</thead>
<tbody>
<tr>
<td>VA</td>
<td>20/40</td>
<td>20/800</td>
</tr>
<tr>
<td>Pupils</td>
<td>No APD</td>
<td>mild APD</td>
</tr>
<tr>
<td>SLE</td>
<td>clear</td>
<td>Extensive NVI with angle synechiae</td>
</tr>
<tr>
<td>IOP</td>
<td>18 mm Hg</td>
<td>44 mm Hg</td>
</tr>
<tr>
<td>Fundus</td>
<td>PDR with NV and NVE focal area of subhyaloid hemorrhage</td>
<td>PDR with NV and NVE focal area of subhyaloid hemorrhage</td>
</tr>
<tr>
<td>C/D</td>
<td>0.4</td>
<td>0.7</td>
</tr>
</tbody>
</table>

NVG: Medical Management

- Treatment of the underlying disease and control of IOP.
- The key to NVG management lies in elimination of the angiogenic stimulus.
- Effective treatments for retinal ischemia include:
  - Panretinal photocoagulation (PRP),
  - cryotherapy, and
  - endolaser treatment combined with vitrectomy.
- Despite the reduction of retinal ischemia and additional antiglaucoma medication.
  - NVG frequently exhibits irreversible intraocular pressure (IOP) elevation.

NVG: Anti-VEGF

- New treatment includes the use of anti-VEGF
  - several studies have shown that specific inhibition of VEGF-A inhibits pathologic neovascularization in the iris, choroid, cornea, and retina.
- Several studies propose that anti-VEGF be combined with PRP.

NVG: Anti-VEGF

- Intra-vitreal anti-VEGF has been shown effective in regression of new vessels and reduction of IOP in NVG
  - regression occurs quickly, often within days.
  - however, bevacizumab’s duration of action is short-lived, lasting about 4 weeks.
- Olmos, et. al (March 2016):
  - role of bevacizumab in NVG is that of a temporizing rather than a definitive treatment, and eyes with NVG should uniformly receive PRP to treat ischemia, regardless of prior intravitreal bevacizumab injection(s).
**NVG: Glaucoma Surgery**

- Glaucoma filtering surgery is now considered standard for the treatment of the elevated IOP in NVG patients
- Glaucoma surgery is indicated to optimally control IOP if medical therapy has proven to be inadequate. Includes procedures such as:
  - aqueous tube shunt surgery,
  - cyclodestruction, or
  - antimetabolite-enhanced filtration surgery
- **Update:** anti-VEGF therapy is indicated with each of the surgical interventions to improve outcomes

**Glaucoma Drainage Implant**

- Implantation of a tube shunt
- Most common treatment for glaucoma when medications have proven to be insufficient
- **Update:** recommended now to combine treatment with anti-VEGF with implant surgery for improved efficacy
- Zhou 2016 literature analysis

**Trabeculectomy**

- **Trabeculectomy:**
  - Channel made from TM to bleb
- **Trabeculectomy for NVG** has been considered to be a difficult treatment with low success rates
  - Intraoperative or early postoperative bleeding and inflammation caused by neovascularization adversely affects the scarring process of the filtering bleb.

**Cyclodestructive Procedures**

- Ablating a portion of the ciliary body
  - IOP is lowered by decreasing aqueous humour production
- Destruction of the ciliary body by:
  - Transscleral application of cryotherapy or
  - Transscleral or endoscopic delivery of diode, krypton or Nd:YAG laser.

**Trabeculectomy**

- The use of anti-metabolites improves IOP control and the success of trabeculectomy
- **Update:** recommendation to include intracameral/vitreal injection of anti-VEGF in combination with antimetabolite trabeculectomy for improved outcomes
  - Resolution of neovascularization
  - Improved IOP control
  - Kobayashi 2016

**NVG: End Stage**

- For blind painful eyes with uncontrollable IOP, options include:
  - Continued medical therapy,
  - Cyclodestruction,
  - Retrobulbar alcohol injection, or
  - Enucleation.
Case

- 30 BF presents with eye pain in both eyes for the past several days
  - Severe pain (8/10)
  - Never had eye exam before
- PMHx:
  - Has chronic bronchitis
  - Rash on legs
  - Has recently lost weight and has a fever
  - Taking aspirin for pain

Ocular Health Assessment

- VA: 20/30 OD, OS
- PERRL
- FTFC
- EOM’s: FROM with eye pain in all quadrants
- SLE:
  - 3+ injection,
  - 3+ cells and trace flare,
  - deposits on endo (see photo)
- IOP: 18, 18 mmHg
- DFE:
  - see attached fundus image and fluorescein angiography.

Classification of Uveitis

- 4 main questions we need answered
  - Where is the inflammation located?
  - Is disease acute or chronic?
  - Granulomatous or non-granulomatous?
  - Unilateral or bilateral?

Uveitis

- Uveitis frequently is nonspecific but can be associated with:
  - systemic disease,
  - occur following trauma, or
  - be the result of a primary ocular disorder such as:
    - Fuchs’s heterochromic iridocyclitis or
    - glaucomatocyclitic crisis (ie, Possner-Schlossman syndrome)

Helpful Mnemonic

- Mnemonic for acute forms of non-granulomatous uveitis: BLAIR G
  - B: Behcet’s disease
  - L: Lyme disease
  - A: Ankylosing spondilitis
  - I: Irritable bowel syndrome (Crohns)
  - R: Reactive arthritis
  - G: Glaucomatocyclitic crisis

Uveitis

- The clinical features of anterior uveitis are readily recognizable
  - complaints of:
    - photophobia,
    - pain,
    - blurred or variable vision
  - A change in the blood-aqueous barrier results in the liberation of protein and cellular matter into the anterior chamber and the vitreous.
Uveitis

• Clinical findings of:
  – circumlimbal hyperemia,
  – cells and flare in the aqueous and anterior vitreous, and
  – keratic and trabecular precipitates

Uveitis: Treatment

• "Classical treatment":
  – Pred forte: prednisolone acetate 1% formulation which allows penetration through cornea to anterior chamber
  – dependent upon the severity of the uveitis
  – In severe uveitis an aggressive treatment may require a drop every 15-30 minutes (for 6-8 hours) then every hour (while awake) until the follow up exam
  – Mild to moderate: every 1-3 hours while awake until follow up exam

• "Newer" treatment option:
  – Durezol

Cycloplegics

• Common cycloplegic agents include:
  – cyclopentolate 1-2% tid for mild-to-moderate,
  – homatropine 5% BID
  – scopolamine 0.25%
  – atropine 1% bid-tid for moderate-to-severe inflammation

• most common is the use of Homatropine 5% bid (though challenging to find due to manufacturing)
  – be careful using atropine as there is potential for severe systemic side effects
  – also makes the iris essentially immobile

Cycloplegics

• Cycloplegia:
  – used for reduction of pain,
  – break/prevent the formation of posterior synechiae
  – also functions in the reduction of inflammation

• Cycloplegics may not be enough to break existing synechiae
  – Consider adding a sympathomimetic drug such as phenylephrine which activates the iris dilator muscles and may break the synechiae
  – 2.5% is commonly used as part of "routine" dilation but 10% is also available and is primarily used for breaking synechiae
  – Word of caution: 10% is contraindicated in patients with hypertension or thyrotoxicosis and children under the age of 1.
  – Cardiovascular effects which have been seen primarily in hypertensive include marked increase in blood pressure, syncope, myocardial infarction, tachycardia, arrhythmia and subarachnoid hemorrhage

Treatment Options

• Durezol:
  – Difluprednate
    • only difluorinated steroid
  – Steroid emulsion
  – BAK free
  – Increased "potency" so dosing needs to be less than "classical treatment" with Pred Forte
  • rough recommendation is 1/2 dosing of Pred Forte

Treatment

• Topical administration is most common though periorbital injections and systemic meds are useful for posterior uveitis and difficult cases
• Dosing is dependent upon severity of the inflammation
  – typically you want to hit the uveitis hard and fast!
  – E.g. In severe uveitis an aggressive treatment may require a drop every 15-30 minutes (for 6-8 hours) then every hour (while awake) until the follow up exam
  – Mild to moderate: every 1-2 hours while awake until follow up exam
  – Dosing should continue until the inflammation is gone (i.e. no cells or flare noted in the anterior chamber) before steroid tapering
  – If you have a minimal anterior chamber reaction then steroid may not be necessary at all (e.g. traumatic iritis)
Treatment

• NOTE: it is crucial to taper your steroid treatment!
  – You will have a rebound inflammation if you simply remove your patient from their steroids...especially if the anterior chamber is not completely resolved.
  • Consider beginning taper a day or two after you have seen resolution of the anterior chamber reaction to ensure no residual inflammation

Follow-up

• Every 1-7 days in acute phase depending upon severity and every 1-6 months when stable.
  • On each f/u visit the AC reaction and IOP should be evaluated
  – DFE should be performed for flare-ups, when VA affected, or every 3-6 months.

Treatment

– The taper will be dependent upon how long you have had them on the steroid to get rid of the inflammation!
– Typically, a slow taper is better in order to prevent rebound inflammation
– If the patient has been on the steroid for less than a week a faster taper can be considered.
– Important to inform patient that they may be receiving steroid treatment for a significant time period (weeks to months) and important to not stop treatment even if feeling better.

Follow Up

• If AC reaction improving, then steroid drops can be slowly tapered.
  – cycloplegia can also be tapered as the AC reaction improves.
  – slow taper recommended for chronic granulomatous uveitis.

Treatment

• NSAIDs:
  – do not play an important role in the treatment of an acute uveitis
    • Topical NSAIDs may have a possible role as adjunctive therapy in reducing inflammation and potentially treat CME associated with the uveitis
    • Oral NSAIDs may reduce the chance of recurrence and reduce the total cumulative dose of steroids
      – Note: this has to be balanced with the side effects of chronic oral NSAID use

Systemic Corticosteroids

• Prednisone
  – Available as Oral: 1, 2.5, 5, 10, 20, 50 mg tablets and 1 and 5 mg/mL solution and syrup

• Ocular Treatment Guidelines:
  – Mild to Moderate: Initial dose of 20-40 mg
  – Moderate to Severe: 40 – 60 mg
  – Severe: Begin with 60 mg and increase if necessary
• Specific Conditions: Giant Cell Arteritis
  • 80-100 mg Prednisone
  • Consider IV Methylprednisolone 250 mg IV q6hours for 12 doses
Stromal Dystrophies

- Granular Dystrophy, Type 1
- Granular Dystrophy, Type 2 (granular-lattice)
- Lattice Corneal Dystrophy
- Macular Corneal Dystrophy
- Schnyder Corneal Dystrophy
- Congenital Stromal Corneal Dystrophy
- Fleck Corneal Dystrophy
- Central Cloudy Dystrophy of Francois

Granular Dystrophy, Type 1

- Discrete white granular opacities in central anterior corneal stroma.
  - With age:
    - increasing number, density, size and depth of opacities
    - intervening stroma and peripheral cornea remain clear

Macular Corneal Dystrophy

- Grayish opacities in the superficial stroma
  - With age:
    - extension into deeper stromal layers
    - intervening stroma becomes hazy
    - progressive loss of vision,
    - photophobia and ocular discomfort.

Macular Corneal Dystrophy (Groenouw Type II)

- Surgical treatment usually required by 2nd or 3rd decade of life.
  - PK
  - DALK (Deep Anterior Lamellar Keratoplasty) not indicated as may have damage to Descemets
- Likely need multiple PK

Granular Dystrophy

- RCE are common
- Decreased vision results from subepithelial scarring or dense stromal deposits
- Surgical treatment includes PK or DALK (Deep Anterior Lamellar Keratoplasty)
- Can delay PK with proper PTK treatment
PTK Treatment for GRANULAR

Lattice (Type I)
- Characteristic clinical appearance includes
  - linear
  - refractile
  - branching deposits within the anterior stroma

Lattice Dystrophy
- The central cornea is progressively opacified resulting in scarring and deterioration of vision while the periphery remains clear.
- RCE's often present.
- May require surgical intervention with diminished vision.
  - PTK
  - PK
  - DALK
Granular Dystrophy, Type 2 (Granular-Lattice, Avellino)
• has features of both lattice dystrophy, and granular dystrophy type I
• autosomal dominant
• mutations in the gene, TGFBI on chromosome 5
• Avellino Lab USA has developed the Avellino-Gene Detection System Test that makes LASIK safer

Central Crystalline Dystrophy of Schnyder
• Vision is typically mildly affected though there maybe associated systemic complications
—systemic cholesterol should be evaluated

Granular Dystrophy, Type 2
• PK can improve vision at least temporarily but deposits tend to recur
• LASIK has been reported to exacerbate the number and density of the opacities
• Patients treated with PRK may do better and can retain corneal clarity for a decade or more

Stromal Dystrophy Treatment
• Treat erosions per above
• Can initially treat poor visual acuity with PTK
• May eventually need PKP
• Can re-occur in new graft

Central Crystalline Dystrophy of Schnyder
• Opacities consist of
  — small, needle-shaped refractile crystals that are either white or polychromatic
  — may extend into deeper stroma but epithelium remains normal

Normal Changes to the Endothelium
• Descemet’s layer thickens from 3-17u
• There is a decrease in the # of endothelial cells
  — from 3500 cells/mm2 to 2400
  — this single layer spreads out; lacks mitosis
• High density mitochondria: 90% pump
• Lenses produce reversible polymegathism
Abnormal Changes to the Endothelium

- Endothelial cells become more irregular
- Cells secrete collagen towards Descemet’s causing multilamination = guttata
- This breaks down the barrier function and results in stromal and epithelial edema

Fuch’s Dystrophy

- Endothelium:
  - acts as both a barrier and pump function
  - responsible for maintaining corneal transparency by reducing corneal hydration
- Fuch’s:
  - occurs bilaterally,
  - AD inheritance,
  - females 3x more likely to develop condition

Fuch’s Dystrophy: Guttata

- Corneal guttata
  - excessive accumulation of abnormal endothelial secretions is associated with the disease process
  - usually first noticed in the central cornea in patients 30’s and 40’s
  - corneal physiology is affected adversely by interference with pump action
  - guttata appear as small refractile “drops” on the corneal endo

Fuch’s Dystrophy: Guttata

- closer inspection with specular reflection reveals an “orange peel-like” dimpling of the endo
- with the decreased pump function, the overlying stroma becomes edematous
- long standing corneal edema may result in corneal scarring and RCE

Fuch’s Dystrophy

- Guttae develop along Descemet membrane
- Loss of endothelial cells
- Edema
- Glare and blurry vision
- Worse in the morning
- Autosomal dominant, with onset in later life.
Fuch’s Dystrophy: Guttata

Normal Endothelial Mosaic

Fuch’s Dystrophy

- Patient symptoms vary with degree of guttata and compromised pump function
- Moderate guttata
  - may affect visual function
  - may result in light scatter (haloes)
  - typically noticed upon waking
- With increased disruption to the pump:
  - vision decreases
  - potential development of bullous keratopathy

Fuch’s Dystrophy Endothelial Cell Count

Stages of Fuch’s Dystrophy

Healthy endo: Cornea thin
Endo dropout: Cornea swells, mild vision loss
Severe swelling, blisters on surface
Chronic swelling, surface scarring
Fuch’s: Bullous Keratopathy

Treatment for Endothelial Dystrophies

• No topical medications to treat actual disease

• Eventually need surgery

• Future therapies in development
Descemets Membrane Endothelial Keratoplasty (DMEK)

- What is it
- Who is it for
- Why do we do it

DMEK
First performed in 2006 by Melles
Pure anatomic replacement surgery

Advantages Over DSEK
Better visual acuity
DSEK 20/40 vs DMEK 20/25

Faster vision recovery
DSEK 3 months vs DMEK 1 month

Less rejection
DSEK ~10% vs DMEK < 1%

Case
- 20 WM
  - Worsening vision last 2 years
  - Increasing Astigmatism
- POHx:
  - Allergies
- Meds:
  - none
Assessment

- VA: 20/40 OU
- SLE:
  - OU:
    - Thinning of cornea
    - IOP 14, 14 mm HG
- DFE: Unremarkable

Etiology

- Increased enzyme activities
- Decreased levels of enzyme inhibitors
  - Excess of toxic by-products
  - Destroys the normal corneal matrix
  - Results in thinning and scarring

Keratoconus

- NOT A DYSTROPHY
- Bilateral
- Noninflammatory
- Ectasia of the cornea

Diagnosis

- SLE findings include:
  - central corneal thinning,
  - Fleischer’s ring,
  - scarring at the level of Bowman’s layer or anterior stroma, and
  - vertical striae (Vogt’s lines).

Causes of Keratoconus

- Decreased vision and contact lens intolerance
- EYE RUBBING
  - Atopy,
  - Vernal keratoconjunctivitis
  - Down syndrome
  - Marfan/syndrome
  - Floppy eyelid syndrome,
  - Obstructive sleep apnea

Diagnosis

- Keratoconus tends to progress over 7-8 years and then stabilizes
- Severity is variable b/w patients and is often asymmetric
- Thinning can be extensive:
  - resulting rupture in Descemet’s membrane
    - triggers a sudden influx of aqueous into the cornea (Hydrops)
Diagnosis

• Common refractive or topographic effects include:
  – irregular astigmatism and
  – poor best-corrected visual acuity with specs

EARLY DIAGNOSIS IS KEY

• Waiting too long to treat
  – Increases the chance of LOSS of BCVA
  – Increases the probability of progression to transplant
Keratoconus-Corneal Thinning

INTACS
- Arclike PMMA segments inserted into deep corneal stroma to flatten the central cornea
- Indicated for mild to moderate keratoconus with a clear optical zone and contact lens intolerant
- Does have FDA approval for the treatment of keratoconus in the US
- DOES NOT STOP PROGRESSION

Keratoconus-Vertical Striae

INTACS
- The goal is to improve topography:
  - lift the ectasia to reduce irregular astigmatism
  - flatten the soft tissue to reduce the SE
- These changes should improve the UCVA and increase contact lens or spectacle success.

Treatment
- Correct refractive error
  - Glasses
  - Contact lens
- Correct Topography
  - INTACS
- Correct Cornea
  - PKP
  - DALK
- Treat Disease
  - Cornea Crosslinking
  - Can pair with INTACS

INTACS FOR KCN
Cornea Crosslinking

Riboflavin
(riboflavin 5'-phosphate ophthalmic solution)

plus

Ultraviolet light

Where do cross-links occur?

• Collagen fibrils within lamellae are regulated by an interconnecting network of proteoglycans.¹

• Cross-linking with UVA/riboflavin has no effect on any collagen structural parameter measured by x-ray scattering except uniformity of nearest neighbor interfibrillar spacing.²

• Therefore, it is believed that cross-links are formed predominantly at fibril surfaces and within the protein network surrounding the collagen.²


Cross-linking Procedure

Radical riboflavin and reactive oxygen species are thought to interact with corneal proteins (such as tyrosine and tryptophan) to create chemical bonds within or between collagen fibrils: "cross-links."

Cross-linking improves the biomechanical properties of the cornea by strengthening the corneal tissue in the anterior stroma

Cross-linking Origins

Corneal Cross-linking was introduced by Theo Seiler and colleagues in 1997 in Dresden, Germany.¹

Pre-clinical studies were conducted on porcine corneas using a number of methods that were already used in other areas of medicine to achieve "cross-linking" in collagenous tissues:

• Non-enzymatic glycation (like that which occurs with age)
• Aldehyde reactions
• Irradiation with ultraviolet light and a photosensitizer


Completed Phase III Study

Keratoconus

Completed Phase III Study

Keratoconus
**Completed Phase III Study**

**Post Refractive Surgery Corneal Ectasia**

![Graph showing Mean Change from Baseline](image)

**Keratoconus-Hydrops**

- **Symptoms include:**
  - sudden decrease in best corrected vision,
  - foreign body sensation or pain
- **Signs include:**
  - conjunctival hyperemia/redness,
  - prominent central or inferior corneal edema and clouding along with conjunctival hyperemia
- **Tends to be self-limiting**
  - in 8-10 weeks the endothelial cells regenerate across the ruptured Descemet’s membrane

**Corneal Crosslinking**

- **Goal is to prevent progression**
- **May not improve VA**
- **Can go back to soft contact lenses and scleral lenses in 1-2 weeks**
- **Follow similar to PRK**
  - Longer healing
  - May see haze

**Keratoconus-Hydrops Treatment**

- **May use hyperosmotics and antibiotics to prevent secondary infections**
- **PK’s are indicated if resulting scarring limits correction of vision**

**Keratoconus**

- **Need to identify and treat patients early**
- **Prevent vision loss**
- **Keep patients in current correction**
Keratoconus-Scarring

Penetrating Keratoplasty

Take Home Message

- Identify dystrophies by anatomical layer
- DMEK is a pure anatomical replacement transplant
- Keratoconus needs to be identified early and treated early