Cornea 2017
Keeping the Old, or Breaking the Mold?

Program Directors
Bennie H Jeng MD, Carol L Karp MD, Jennifer Y Li MD

In conjunction with the Cornea Society

Ernest N Morial Convention Center
New Orleans, Louisiana
Saturday, Nov. 11, 2017

Presented by:
The American Academy of Ophthalmology

Supported in part by an unrestricted educational grant from Shire

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Program Director
Jennifer Y Li MD
Program Director

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2017 Cornea Subspecialty Day Planning Group

On behalf of the American Academy of Ophthalmology and the Cornea Society, it is our pleasure to welcome you to New Orleans and Cornea 2017: Keeping the Old, or Breaking the Mold?

Bennie H Jeng MD
Program Director
CoDa Therapeutics: C
EyeGate Pharmaceuticals Inc.: O
Jade Therapeutics: C
Kedrion: C
Santen Inc.: C

Carol L Karp MD
Program Director
None

Jennifer Y Li MD
Program Director
None
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Daniel S Durrie MD, Chair (Refractive Surgery)
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Ocular Therapeutix: C,S
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Slack Publishing: C
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Syndexis: C | TearLab: C

R Michael Siatkowski MD (Pediatric Ophthalmology)
National Eye Institute: S

Kuldev Singh MD (Glaucoma)
Abbott Medical Optics Inc.: C
Aerie: C
Alcon Laboratories, Inc.: C
Allergan: C
Belkin Laser Ltd: C
Glaukos Corporation: C
InjectSense: C
Ivantis: C
Mynosys: C
National Eye Institute: S
Novartis Institute for Biomedical Research: C
Santen, Inc.: C
Shire: C
Thieme Medical Publishers: C
U.S. Food and Drug Administration: S,C

Nicholas J Volpe MD (Neuro-Ophthalmology)
Optotech: C
Opticent Inc.: O

AAO Staff
Ann L’Estrange
None
Melanie Rafaty
None
Lisa Romero
None
Debra Rosencrance
None
Beth Wilson
None
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CME Credit

Academy’s CME Mission Statement
The purpose of the American Academy of Ophthalmology’s Continuing Medical Education (CME) program is to present ophthalmologists with the highest quality lifelong learning opportunities that promote improvement and change in physician practices, performance, or competence, thus enabling such physicians to maintain or improve the competence and professional performance needed to provide the best possible eye care for their patients.

2017 Cornea Subspecialty Day Meeting Learning Objectives
Upon completion of this activity, participants should be able to:

■ List common causes of corneal infections and best practices for management
■ Discuss the role of keratoplasty in the management of patients with corneal disease
■ Review the role of imaging and in-office diagnostics in the treatment of corneal disorders
■ Provide a rationale for treatment of ocular surface disease and inflammatory disorders

2017 Cornea Subspecialty Day Meeting Target Audience
The intended audience for this program is cornea surgeons, comprehensive ophthalmologists with an interest in anterior segment, and allied health personnel who are performing or assisting with cornea surgery.

2017 Cornea Subspecialty Day CME Credit
The American Academy of Ophthalmology is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide CME for physicians.

The Academy designates this live activity for a maximum of 7 AMA PRA Category 1 Credits™. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

Scientific Integrity and Disclosure of Financial Interest
The American Academy of Ophthalmology is committed to ensuring that all CME information is based on the application of research findings and the implementation of evidence-based medicine. It seeks to promote balance, objectivity, and absence of commercial bias in its content. All persons in a position to control the content of this activity must disclose any and all financial interests. The Academy has mechanisms in place to resolve all conflicts of interest prior to an educational activity being delivered to the learners.

The Academy requires all presenters to disclose on their first slide whether they have any financial interests from the past 12 months. Presenters are required to verbally disclose any financial interests that specifically pertain to their presentation.

Control of Content
The American Academy of Ophthalmology considers presenting authors, not coauthors, to be in control of the educational content. It is Academy policy and traditional scientific publishing and professional courtesy to acknowledge all people contributing to the research, regardless of CME control of the live presentation of that content. This acknowledgment is made in a similar way in other Academy CME activities. Though they are acknowledged, coauthors do not have control of the CME content, and their disclosures are not published or resolved.

Attendance Verification for CME Reporting
Before processing your requests for CME credit, the American Academy of Ophthalmology must verify your attendance at Subspecialty Day and/or AAO 2017. In order to be verified for CME or auditing purposes, you must either:

■ Register in advance, receive materials in the mail, and turn in the Subspecialty Day Syllabi exchange voucher(s) onsite;
■ Register in advance and pick up your badge onsite if materials did not arrive before you traveled to the meeting;
■ Register onsite; or
■ Scan the barcode on your badge as you enter an AAO 2017 course or session room.

CME Credit Reporting
Lobby B and Lobby G and Academy Resource Center, Hall G – Booth 3140
Attendees whose attendance has been verified (see above) at AAO 2017 can claim their CME credit online during the meeting. Registrants will receive an email during the meeting with the link and instructions on how to claim credit.

Onsite, you may report credits earned during Subspecialty Day and/or AAO 2017 at the CME Credit Reporting booth.
Academy Members: The CME credit reporting receipt is not a CME transcript. CME transcripts that include AAO 2017 credits entered onsite will be available to Academy members on the Academy’s website beginning Dec. 7, 2017.

After AAO 2017, credits can be claimed at www.aao.org. The Academy transcript cannot list individual course attendance. It will list only the overall credits spent in educational activities at Subspecialty Day and/or AAO 2017.

Nonmembers: The Academy will provide nonmembers with verification of credits earned and reported for a single Academy-sponsored CME activity, but it does not provide CME credit transcripts. To obtain a printed record of your credits, you must report your CME credits onsite at the CME Credit Reporting booths.

Proof of Attendance

The following types of attendance verification will be available during AAO 2017 and Subspecialty Day for those who need it for reimbursement or hospital privileges, or for nonmembers who need it to report CME credit:

- CME credit reporting/proof-of-attendance letters
- Onsite registration receipt
- Instruction course and session verification

Visit www.aao.org/cme for detailed CME reporting information.
Faculty

Mohamed F Abou Shousha MD
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Irvine, CA

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Langhorne, PA

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Philadelphia, PA

Donald Stone MD
Rockville, MD

Mark A Terry MD
Portland, OR

Arun D Singh MD
Cleveland, OH

Donald Tan MD FRCS FRCOphth
Singapore, Singapore

Elmer Y Tu MD
Glenview, IL
Ask a Question Live During the Meeting Using the Mobile Meeting Guide

To ask a question during the meeting, follow the directions below.

- Access at www.aao.org/mobile
- Select “Program Handouts & Evaluations”
- Filter by Meeting—Cornea Meeting
- Select Current Session
- Select “Ask the presenter a question (live)” Link
- Click Submit Question
# Cornea 2017: Keeping the Old, or Breaking the Mold?

In conjunction with the Cornea Society

**(SUNDAY, NOV. 12)**

7:00 AM  | CONTINENTAL BREAKFAST
---|---
8:00 AM  | Welcome and Introductions

<table>
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<th>Time</th>
<th>Session</th>
<th>Title</th>
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<tr>
<td>8:02 AM</td>
<td><strong>Section I:</strong> Corneal Infections—Old Bugs, New Drugs</td>
<td>Introduction</td>
<td>Jennifer Y Li MD</td>
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<tr>
<td>8:04 AM</td>
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<td>Bacterial Keratitis: Are Fortified Antibiotics Still Necessary?</td>
<td>Shahzad I Mian MD</td>
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<td>'Roid Rage: The Controversy over Steroids for Bacterial Keratitis</td>
<td>Thomas M Lietman MD</td>
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<td>8:22 AM</td>
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<td>Fungal Keratitis: Detecting and Disabling the Fungus among Us</td>
<td>Eduardo C Alfonso MD*</td>
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<td>Acanthamoeba Keratitis: Embracing the Challenge</td>
<td>Denise de Freitas MD</td>
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<td>Viral Keratitis: What's New that We Can Do</td>
<td>Todd P Margolis MD PhD</td>
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<td>Cut It Out! Nonmedical Approaches to Infectious Keratitis</td>
<td>Jennifer R Rose-Nussbaumer MD</td>
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<td>Case: The Most Unusual Keratitis</td>
<td>Mark J Mannis MD</td>
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<td><strong>Section II:</strong> Keratoplasty—Are We Doing the Right Thing?</td>
<td>Introduction</td>
<td>Bennie H Jeng MD*</td>
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<td>Should We Still Be Doing DSAEK?</td>
<td>Winston D Chamberlain MD PhD</td>
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<td>Regrafting the Failed Penetrating Keratoplasty: Should We Still Re-PK?</td>
<td>Donald Tan MD FRCS FRCOphth*</td>
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<td>Done with Regrafting? When Is KPro a Better Choice?</td>
<td>Anthony J Aldave MD*</td>
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<td>9:51 AM</td>
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<td>Breaking the Mold: Surgery Starts in the Eye Bank!</td>
<td>Marian Sue Macsai-Kaplan MD*</td>
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<td>Case: What Do I Do with <em>This</em> Cornea?!</td>
<td>Mark A Terry MD*</td>
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* Indicates that the presenter has financial interest. No asterisk indicates that the presenter has no financial interest.
### Section III: Conjunctival Tumors—What’s Old, What’s New?
Moderator: Carol L Karp MD

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<td>Carol L Karp MD</td>
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<td>Advocating for Patients</td>
<td>Darby D Miller MD 18</td>
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<td>What to Do When the Tumor Goes Viral? Conjunctival Papilloma Management</td>
<td>Carol L Shields MD 22</td>
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<td>Does Imaging Help in the Diagnosis of Conjunctival Tumors? Biopsy or Image?</td>
<td>Afshan A Nanji MD 23</td>
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<td>Pigmented Lesions: Watch, or Worry?</td>
<td>Victoria M Cohen FRCOphth 24</td>
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<td>Pterygium: An Evidence-Based Look</td>
<td>Darren G Gregory MD 25</td>
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<td>Case: A Lump that Stumped Me</td>
<td>Arun D Singh MD* 26</td>
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### Section IV: Anterior Segment Imaging—What’s Recent, What’s Decent?
Moderator: Carol L Karp MD

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<td>How Many Technologies Does It Take to Implant a Toric IOL?</td>
<td>David F Chang MD* 27</td>
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<td>1:33 PM</td>
<td>Is Aberrometry Necessary?</td>
<td>Parag A Majmudar MD* 28</td>
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<td>Corneal Imaging to Improve Surgical Outcomes</td>
<td>Mohamed F Abou Shousha MD* 29</td>
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<td>Tomography: How This Helps Me</td>
<td>Renato Ambrósio Jr MD* 30</td>
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<td>Do We Need an Intraoperative OCT?</td>
<td>Francis W Price Jr MD* 34</td>
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<td>Case: Imaging Saved the Day</td>
<td>Sadeer B Hannush MD 35</td>
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### Section V: Keratoconus, A Steep Learning Curve—The Newest in Diagnosis and Management
Moderator: Jennifer Y Li MD

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<td>How to Diagnosis Keratoconus: Tried and True vs. New View</td>
<td>Michael W Belin MD* 36</td>
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<td>Contact Lenses in Keratoconus: What Options Do We Have Now?</td>
<td>Deborah S Jacobs MD* 37</td>
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<td>Considerations Concerning Corneal Collagen Crosslinking</td>
<td>William J Dupps MD PhD* 40</td>
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<td>Tearing Up the Old Paradigm for Management of Acute Hydrops</td>
<td>Mazen Y Choulakian MD 42</td>
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<td>Case: A Corneal Conundrum</td>
<td>Elmer Y Tu MD* 43</td>
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**Section VI: Inflammatory Conditions of the Anterior Segment—It’s Burning Me Up!**

Moderator: Bennie H Jeng MD*

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<td>Point-of-Care Testing for Dry Eyes: Tears of Joy, or Tears from the Expense?</td>
<td>Anat Galor MD*</td>
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<td>Scratching the Old: What’s New in Allergic Conjunctivitis?</td>
<td>Deepinder K Dhaliwal MD*</td>
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<td>Stevens-Johnson Syndrome: A Chronic Problem</td>
<td>Wuqaas M Munir MD</td>
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<td>Revisiting a Dry Topic: Is Anything New with Sjögren Syndrome?</td>
<td>Stephen C Pflugfelder MD*</td>
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<td>Sclera on Fire: What Labs Do I Really Need to Check?</td>
<td>Donald Stone MD</td>
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<td>Case: Not Just Another Red Eye</td>
<td>Christopher J Rapuano MD*</td>
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<td>5:03 PM</td>
<td>Closing Remarks</td>
<td>Bennie H Jeng MD*</td>
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<td>Carol L Karp MD</td>
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<td>Jennifer Y Li MD</td>
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Bacterial Keratitis: Are Fortified Antibiotics Still Necessary?

Shahzad I Mian MD
’Roid Rage: The Controversy over Steroids for Bacterial Keratitis

Thomas M Lietman MD

I. History of the Controversy
   A. Antibiotics typically eliminate bacterial infection before corneal perforation, although a large portion of cases still end up with scars and permanent visual impairment. Adjunctive steroids have been considered since they were introduced, although given the risks, including suppressing the immune response, their widespread use has been limited.
   B. Animal and retrospective human studies have mixed results.
   C. Corneal specialists have become more comfortable with adding steroids through experience.

II. Steroids for Corneal Ulcer Trial
   A. 500-patient, multicenter, randomized controlled trial comparing adjunctive prednisolone phosphate to placebo in laboratory-proven bacterial ulcers
   B. Primary outcome, spectacle-corrected visual acuity at 3 months, similar in both arms
   C. Secondary outcomes
      1. Severe ulcers tended to do better with steroids.
      2. Pseudomonas aeruginosa tended to do better with steroids.
      3. Nocardia species did worse with steroids.
      4. Steroids earlier in the course proved beneficial.
      5. Longer-term results (1-4 years) suggested improvement with steroids.

III. Other Trials

IV. Other Options
   A. Other steroid preparations
   B. Other immunosuppressive agents
   C. Warnings about using steroids in the absence of effective antimicrobial treatment. Necessary to be certain etiology is bacterial.
Fungal Keratitis: Detecting and Disabling the Fungus among Us

Eduardo C Alfonso MD

Detection

There are more than 50,000 species of fungi, but only about 100-150 cause human disease, and fewer cause ocular infections. These vary by geographic region and climate.

Why are ocular fungal infections increasing? Use of antibiotics, immunosuppression, steroids, corneal surgery, contact lenses, evolution of pathogens, and environmental changes may all play a role. Identification is important in the outcome. Clinical diagnosis for fungal keratitis is correct only in 45% of cases, and pretreatment decreases the predictive value and recovery of organisms.

Cultures and smears are the gold standard for the identification of the cause of the infection. Ninety-one percent of smears correlate with culture results, 92% of isolates are recovered in agar within 72 hours, and fungi grow in an average of 2.7 days. The most accurate diagnosis is obtained using molecular techniques for identification.

Target Gene

ITS, the nuclear ribosomal internal transcribed spacer region, lies within the ribosomal DNA and contains the ITS1, ITS2, and 5.8S rDNA sites. It can identify the organism, the source of the organism, and its sensitivity profile, pathogenicity, and mycotoxin production. Other methods to identify the cause and extent of the infection are using the confocal microscope and anterior segment OCT.

Treatment

The only commercially available topical antifungal is natamycin 5% (Alcon); others can be prepared extemporaneously: amphotericin B (methyl ester), voriconazole (Vfend, Pfizer), caspofungin (Cancidas, Merck), and posaconazole (Noxafil, Merck), among others. Latest information on effectiveness comes from the Mycotic Ulcer Treatment Trial (MUTT), which showed that topical natamycin is superior to voriconazole against filamentous fungi, is better for Fusarium cases, and showed no difference between treatments for non-Fusarium cases. Further observations from molecular analysis show not only that molecular classification is important in assisting clinical decision making but that natamycin is better against F. solani, and voriconazole, not as effective, but equal against others. F. solani infections are less likely to respond to medical treatment and more likely to require a keratoplasty than are non-solani Fusarium. Use of steroids is not recommended, dangerous if used too early, but may be used as a diagnostic challenge. Topical drops can be augmented with intrastromal injections of voriconazole (50 micrograms) around the infiltrate, with repeated injections as necessary. The MUTT II showed that oral adjunctive voriconazole is indicated in the treatment of Fusarium keratitis.

Surgical Interventions

Surgical interventions include corneal scraping, which removes epithelium and necrotic material allowing better penetration of topical antifungals, and penetrating keratoplasty, which may be necessary, usually after 2-4 weeks of medical treatment. Other surgical measures include tissue adhesives, amniotic membrane, and conjunctival flaps. Recent studies have supported early keratoplasty (2 weeks), showing eradication of infection in 92.7% of cases. Intraoperative management includes culture and pathology of all removed tissues to determine extension, lavage with BSS, new instruments for donor tissue, intraocular amphotericin B (5-10 mg), or voriconazole (50-100 μg/cc), and use of interrupted sutures. Postoperative management consists of topical and systemic antifungals, cyclosporin A or tacrolimus, and no topical or systemic steroids in the immediate postop period.

Recent success in the use of crosslinking is limited to infections on the anterior 250 microns, but there are no definitive recommendations for every case. We have recently shown that photodynamic therapy may be an additive option for treatment.

Selected Readings

Acanthamoeba Keratitis: Embracing the Challenge

Denise de Freitas MD

Acanthamoeba is an organism widely dispersed naturally in water and soil. Acanthamoeba keratitis (AK) is considered one of the most difficult infections to diagnose and treat. The prognosis is related to the precision of the clinical laboratory diagnosis and precocity of the treatment.

Clinical Features

Patients with AK present with a chronic history of mild keratitis with nonspecific symptoms of intolerance to contact lens (CL) use. Left undiagnosed, the condition can progress to red eye, photophobia, tearing, and usually pain that is disproportionate to the clinical findings, although some patients do not report pain. The biomicroscopic signs include limbitis; dotted and dendritic keratitis; perineural infiltrates; epithelial defects; and corneal thinning, which can evolve to nonspecific perforation and nonspecific or ring-shaped stromal infiltrates and uveitis with varying degrees of severity, with or without hypopyon. Nonspecific mydriasis and cataract and glaucoma syndrome can be observed in severe and advanced cases. Acanthamoeba coinfection, mainly infectious crystalline keratopathy, has been described in the literature.

Diagnosis

The clinical diagnosis of AK is confirmed through complementary laboratory tests such as culture (the gold standard); direct examination; and molecular biology analysis, such as polymerase chain reaction (PCR), confocal microscopy, and histopathology. Culturing is performed using non-nutrient agar and/or soy agar supplemented with Escherichia coli avirulent as a nutrition source for protozoan proliferation. In the direct examination, the Acanthamoeba species cysts on the slide containing the smear of the clinical sample can be visualized using different staining methods, with fluorescent dye calcofluor white being the most suitable for microscopic analysis of specific structures, such as cyst size, pleomorphism, and morphology. Other possible dyes are Giemsa and Acidine orange. PCR generally has high sensitivity (> 80%) and specificity (100%). Histology identifies with reasonable ease the protozoa in the cystic form that is characterized by rounded, pleomorphic structures. From the specific characteristics of the cellular refraction of the protozoa, confocal microscopy allows observation of the cystic form and also the keratoneuritis pattern associated with infection. Depending on the clinical picture, apparatus, and observer, confocal microscopy might facilitate significant sensitivity and specificity.

Treatment

Early treatment, preferably within 2 to 3 weeks of the onset of symptoms and signs, is mandatory for a better disease prognosis. The trophozoites are sensitive to most chemotherapeutic agents; however, the cysts have greater resistance. The treatment of AK has not been standardized and can vary between different specialized outpatient services. The use of antimicrobial drops for AK treatment depends on previous authorization and certification by the health regulatory agency regarding the marketing of medicines in each country. Basically, 2 classes of drugs are used: biguanides and diamidines. Among the biguanides, polyhexamethylene biguanide (PHMB) is used in concentrations ranging from 0.02% to 0.06%; and chlorhexidine, in concentrations ranging from 0.02% to 0.2%, depending on the response. Among the diamidines, propamidine isethionate (Brolene) and hexamidine disethionate (Désomédine) are used at a concentration of 0.1%. The primary therapeutic profile for AK includes topical antimicrobial eye drops hourly around the clock, generally for 2 days according to toxicity, with gradual reduction as the symptoms and clinical signs improve. The treatment time can vary from case to case (average duration: 4-6 months). The tissue chemical toxicity caused by continuous use of antimicrobial drops should be evaluated frequently throughout treatment. If necessary, the medication can be reduced or stopped for a certain period until the tissue toxicity abates. The US FDA recently approved topical miltefosine for treating AK in the United States.

Therapeutic tectonic corneal transplantation is indicated only in cases of refractory AK, which is characterized by persistent positive cultures; in difficult-to-control abscesses; in perforations that cannot be managed with adhesive tissue; and in cases of mydriasis, cataracts, and glaucoma, in which combined corneal transplantation, lens extraction, IOL implantation, and iridoplasty might prevent development of secondary glaucoma refractory to treatment. Anterior lamellar keratoplasty should be performed with caution, since the protozoa tend to reach the deep corneal layers. Different studies have shown that the corneal crosslinking technique using riboflavin associated with application of ultraviolet-A light is ineffective for treating AK.

Prevention

Prevention is essential because the disease has great potential to cause marked visual acuity loss and blindness. Patients should be instructed to always wash and dry their hands thoroughly before handling the CL. A multipurpose cleaning and disinfecting solution should not be “topped-off” or reused in the CL case. The CL case should be washed daily with the multipurpose solution, not tap water. The CL should not be exposed to nonsterile solutions or water from the faucet, pool, pond, bathtub, shower, or sauna, among others. The CL should be rubbed for at least 15 seconds before and after use, because this increases the effectiveness of cleaning and disinfection. The CL and CL solutions should not be used beyond the expiration date; expired solutions should be discarded. If symptoms or signs of eye irritation occur, the CLs should be removed immediately from both eyes and emergency medical attention sought.
Selected Readings


**Viral Keratitis: What’s New that We Can Do?**

**Todd P Margolis MD PhD**

**Herpes Simplex Virus (HSV)**

Since the days of the ancient Egyptians, corneal epithelial debridement has been used as a treatment option for HSV epithelial keratitis. Whereas the average healing time of an untreated HSV epithelial dendrite is 9-10 days, treatment with debridement and patching reduces healing time to 2.5 days.

Several thousand years later, topical antivirals (idoxuridine), followed by vidarabine, trifluorothymidine, acyclovir, and most recently ganciclovir, were introduced for the treatment of HSV epithelial keratitis. The healing time of HSV epithelial keratitis treated with any of these topical antivirals is about 7 days. Over the years these agents have also been widely used as part of therapeutic regimens for the treatment of HSV stromal disease and iritis, but the role of these topical antiviral agents in the management of these immunologically mediated diseases is not clear, other than to prophylax against recurrent infectious keratitis when treating HSV stromal keratitis with a topical corticosteroid (they do not get into the corneal stroma or aqueous at therapeutic levels in the absence of an epithelial defect).

Despite the introduction of topical antivirals in the 1970s, the rates of corneal transplantation for HSV keratitis remained high until the 1990s, corresponding with the increased use of oral acyclovir in the management of HSV keratitis. The average healing time of HSV epithelial keratitis treated with oral acyclovir is also about 7 days. However, prophylaxis with oral antivirals reduces overall recurrences of HSV ocular disease by up to 95%, depending on the agent and dosage used. Generic forms of oral acyclovir, valacyclovir, and famciclovir are all widely available. All 3 of these drugs are easy to take, have a long shelf life, and are extremely well tolerated, even when taken for years. After oral administration, all 3 drugs reach therapeutic levels in the tear film and aqueous.

Many clinicians seem to be excited about the availability of ganciclovir for ocular HSV. Clinical trials support the fact that topical ganciclovir is noninferior to topical acyclovir in the treatment of HSV epithelial keratitis. There is no published evidence at this time that topical ganciclovir is effective for the treatment of HSV stromal keratitis or iritis, or for the prophylaxis of recurrent ocular disease. Topical ganciclovir is more expensive than oral antiviral medications. It is preserved with benzalkonium chloride, and 60% of patients using topical ganciclovir report blurred vision, 20% report irritation, and 5% develop punctate keratitis. Thus, topical ganciclovir is a reasonable alternative to the use of oral antivirals for the treatment of HSV epithelial keratitis (not stromal disease or iritis). However, it is significantly more expensive and has significantly more side effects than oral antivirals.

**Varicella Zoster Virus (VZV)**

Herpes zoster ocular disease is treated in a manner very similar to treatment of HSV ocular disease (oral antivirals to control the virus and topical corticosteroids to control inflammation), except that there is almost never a role for topical antivirals in treating ocular zoster. Treatment doses of the oral antivirals are higher than that generally used for HSV (acyclovir 800 5x/day, famciclovir 500 mg t.i.d., valacyclovir 1000 mg t.i.d.). Note that these doses of antivirals barely reach the ID₉₀ for controlling VZV, so these full doses should always be used (except in cases of renal failure).

The key management issues for VZV that repeatedly come up in my consultation practice are (1) the need for chronic corticosteroids to control chronic VZV ocular inflammation (the patient may be on topical corticosteroids for years), (2) recurrent and chronic infectious ocular zoster (requiring chronic oral antivirals), (3) neurotrophic keratopathy (this is **not** managed with topical lubricants), and (4) postherpetic neuralgia.

**Epidemic Keratoconjunctivitis (EKC)**

Several recent studies on adenoviral DNA recombination have made it pretty clear that our current methods of serotyping virus to identify strains of adenovirus responsible for EKC are deeply flawed. So, it is probably not a bad idea to empty your brain of the adenoviral serotypes responsible for EKC, thus making room for some new concepts in the diagnosis and management of EKC.

The AdenoPlus system has been heralded as a sensitive and specific point-of-care assay for the diagnoses of adenoviral conjunctivitis. The company reports an 85% sensitivity and 98% specificity for this assay, as compared to polymerase chain reaction (PCR). However, an independent study out of Moorfields Eye Hospital (Kam et al, Br J Ophthalmol., 2015) could not replicate this level of sensitivity, finding that the AdenoPlus system reached a sensitivity of only 39.5% as compared to PCR. Ongoing studies are investigating the treatment of EKC with topical povidone iodine and a topical povidone iodine dexamethasone combination.

For years topical corticosteroids have been used to manage the corneal infiltrates associated with ocular adenoviral infection. Tacrolimus and cyclosporine have also been reported to be useful in the management of the superficial keratitis of EKC.
Cut It Out! Nonmedical Approaches to Infectious Keratitis

Jennifer Rose-Nussbaumer MD

I. Corneal Crosslinking (CXL) for Infectious Keratitis

A. In vitro studies suggest that photochemically activated riboflavin is effective against common ocular pathogens.1

B. CXL may also have anti-inflammatory effects and promote resistance of corneal tissue to enzymatic degradation.2,3

C. To date, randomized clinical trials (RCTs) have yielded mixed results (see Table 1).

II. Intrastromal Injection: Voriconazole for Fungal Keratitis

A. May provide steady-state drug concentrations at the site of infection and avoid intervals of subtherapeutic drug dosing

B. Case series have suggested a role, but 1 RCT comparing intrastromal injection to topical voriconazole found significantly improved 3-month visual acuity in the topical voriconazole group.4

III. Therapeutic Penetrating Keratoplasty (TPK)

A. Risk factors

1. Perforation is more common in fungal than bacterial keratitis.

2. Baseline clinical features such as presence of hypopyon and deep and large infiltrate are predictors of perforation and/or need for TPK.

3. Culture positivity after starting treatment is a predictor of eventuating to TPK and may be used as an early indicator of response to therapy.5

B. Outcomes

1. TPK is usually reserved for severe, progressive infections for tectonic support, or to avoid scleral extension.

2. High risk of graft rejection and failure6

3. Optimal timing to prevent these complications is unknown.

C. Deep anterior lamellar keratoplasty may be better due to preservation of host endothelium and reduced risk of intraocular spread of infection.7

References


Table 1. Relevant Randomized Clinical Trials Assessing Corneal Crosslinking

<table>
<thead>
<tr>
<th>Trial</th>
<th>Question</th>
<th>N</th>
<th>Finding</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bamdad et al., 2015⁸</td>
<td>Adjuvant CXL vs. standard therapy for moderate bacterial keratitis</td>
<td>32</td>
<td>Adjuvant CXL shortened the treatment course and resulted in improved outcomes.</td>
<td>Small sample size, investigator was partially unmasked, enrolled exclusively in Iran</td>
</tr>
<tr>
<td>Said et al., 2014⁹</td>
<td>Adjuvant CXL vs. standard therapy for bacterial, fungal, Acanthamoeba, or mixed keratitis</td>
<td>40</td>
<td>No benefit of adjuvant CXL</td>
<td>Inappropriate randomization, inclusion of multiple types of keratitis and mixed keratitis, small sample size, enrolled exclusively in Egypt</td>
</tr>
<tr>
<td>Uddaraju et al., 2015¹⁰</td>
<td>Adjuvant CXL vs. standard therapy for deep fungal keratitis</td>
<td>13</td>
<td>Adjuvant CXL resulted in an increased rate of perforation.</td>
<td>Small sample size, inclusion of only severe fungal ulcers, enrolled exclusively in South India</td>
</tr>
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Case: The Most Unusual Keratitis
Ophthalmia Nodosa: Tarantula-Induced Keratouveitis

Mark J Mannis MD and Enrique O Graue Hernandez MD

Background
Keratitis or ophthalmia nodosa is an entity attributed to exposure to the urticating hairs that cover the dorsal abdomen of New World tarantula species, which are catapulted into the air by the arachnid as a protective mechanism when it is threatened. The tarantula vibrates its hind legs across the dorsal abdomen, projecting the hairs into the surrounding air. The urticating hairs are sturdy, sharp-pointed shafts, 0.3-1.2 mm in length, and with reverse barbs. They are capable of penetrating the skin, conjunctiva, and cornea. Hairs may be embedded in the cornea either directly through the air or by transfer from the surface of the fingers after contact with the spider or its enclosure. Over time, the hairs with reverse barbs can migrate into the deep cornea or anterior chamber. The corneal findings are characteristic, and management consists of removing protruding hairs and controlling inflammation.

Case Presentation
- 17-year-old male, owner of a pet tarantula
- Negative past medical and ocular history
- History of present illness:
  - Two-week history of foreign body sensation
  - Pruritis, photophobia, tearing
  - Conjunctival injection
  - Periocular rash, resolved at time of presentation
- Examination
  - Diffuse conjunctival injection; no discharge
  - Nummular stromal opacities

Discussion
Ophthalmia nodosa was first described in 1906 as a reaction to the sensory setae of caterpillars. Most contemporary cases have been described in relation to tarantulas (Theraphosidae family), which are popular household pets.

The clinical course commonly begins with involvement of the skin and conjunctiva, followed by sometimes deep corneal stromal infiltrates and anterior chamber reaction.

Figure 2.
Tarantula hairs have been classified, and the Type III hair is sharp and barbed, facilitating its penetration and hindering its extrusion or extraction. If possible, hairs should be removed. However, the mainstay of therapy is topical corticosteroid.

Key Clinical Points
- Recognition of typical nummular infiltrates with concomitant conjunctival inflammation
- High index of suspicion for contact with a tarantula
- Long-term follow-up for recurrent inflammation

Selected Readings
Should We Still Be Doing DSAEK?
DMEK vs. Ultrathin DSAEK: The 6-Month DETECT Trial Results

Winston Chamberlain MD PhD, Jennifer Rose-Nussbaumer MD, Charles Lin MD, and Thomas Lietman MD

Endothelial keratoplasty (EK) techniques have evolved rapidly in recent years, and Descemet membrane endothelial keratoplasty (DMEK) has gained in popularity.1 Recent studies suggest that near anatomic replacement of endothelial tissue produces more rapid recovery and improved visual acuity results with DMEK than with Descemet-stripping automated endothelial keratoplasty (DSAEK).2,3 Yet according to the Eye Bank Association of America, DMEK still accounted for only ~23% of endothelial keratoplasties in the United States in 2016 (up from 15% in 2015), while DSAEK accounted for about 46% of all corneal transplants involving endothelial replacement, including penetrating keratoplasties.4

Thus a majority of EK surgeries in the United States are still DSAEK. This is true for various reasons. Many surgeons have not yet adopted DMEK or are early on the DMEK learning curve. Experienced EK surgeons without fellowship training in DMEK may be reluctant to adopt the newer technique since they have excellent and reliable results with DSAEK. Donor preparation, increased intraoperative times, and problems with donor detachment in DMEK can create reluctance among experienced DSAEK surgeons. In addition, many surgeons still feel that DMEK is not applicable to all eyes that require EK, including those that have undergone pars plana vitrectomy, trabeculectomy, or aqueous shunt devices or that have significant anterior chamber abnormalities, such as peripheral anterior synechiae, or iris loss. Even in routine cases of Fuchs dystrophy and bullous keratopathy, the complication rate, including iatrogenic graft failure and graft detachments, is expected to be higher, at least on the surgeon’s learning curve, which could be anywhere from 20-75 cases.2,5

In my DMEK learning curve experience, there were significantly higher numbers of primary graft failures and successful grafts with low endothelial counts as compared to the contemporaneous DSAEK surgeries I was performing.5 Since many U.S. surgeons are still on that learning curve, we may experience a higher range of iatrogenic graft failures in the United States over the next several years.

However, DMEK may produce better outcomes. There are 3 potential mechanisms by which DMEK may provide better visual acuity outcomes than DSAEK: graft thickness,6,7 interface haze,8 and corneal higher-order aberrations.9 Graft thickness has been correlated with BSCVA outcomes among thinner grafts. One retrospective case series found that 71% of thin endothelial grafts (defined as < 131 μm) had BSCVA of 20/25 or better, while only 50% of thick grafts (defined as ≥ 131 μm) achieved this.7 In addition, higher-order aberrations, in particular of the posterior cornea, are increased after DSAEK.9 Theoretically, given the decreased tissue thickness transplanted after DMEK, this would be lessened; however, 1 retrospective series looking at higher-order aberrations in DMEK compared with DSAEK found no difference in posterior aberrations between the 2 groups.10 Finally, interface haze may be increased in DSAEK and has been correlated with BSCVA.11

Ultrathin DSAEK (UT-DSAEK) involves donor preparation with a deep microkeratome pass to produce donor grafts near to or less than 100 μm thick. A recent randomized controlled trial demonstrated excellent results with UT-DSAEK that outperformed thicker DSAEK in visual acuity outcomes up to 1 year after surgery, with no difference in endothelial cell densities or graft dislocation.6 This procedure may have similar results to DMEK but without the technical difficulties. A recent survey suggested that there is an interest among EK experts to understand UT-DSAEK’s role in current EK surgery.12 We previously proposed a randomized controlled trial to compare UT-DSAEK with DMEK.5 Randomized controlled trials are the gold standard in determining preferred surgical and medical interventions and therefore are a necessary next step in the DMEK / DSEK literature. We have since completed enrollment in the 2-year DETECT (Descemet Endothelial Thickness Comparison Trial; Clinical-Trials.gov identifier NCT02373137). DETECT is an intervention, randomized, patient- / assessor-masked clinical trial. We report here on the 6-month data of this trial in 50 eyes with either Fuchs dystrophy or bullous keratopathy that were treated either with UT-DSAEK (graft thickness: 70-90 microns) or DMEK. The study has an 80% power to detect 1 line of difference in BSCVA between the study groups with an alpha of 0.05. The patient and the refracting technician are masked to the surgery type for a 2-year follow-up. Our primary outcome measure is BCVA (ETDRS) at 6, 12, and 24 months. Secondary outcome measures include endothelial cell loss, immunological rejection, graft thickness (pre- and postop), corneal higher-order aberrations, interface haze / light scatter, NEI visual functioning questionnaire (NEI VFQ), and complications, including graft detachments and rebubble rates. Other outcome measures will also be assessed.

References


Spicing Up Penetrating Keratoplasty: Does Femto Really Help?

Marjan Farid MD

I. Early Challenges with Penetrating Keratoplasty
   A. Surgical technique
   B. Suturing
   C. Tissue availability and preservation
   D. Graft infection and rejection

II. Current Challenges with Penetrating Keratoplasty
   A. Rate of visual recovery
   B. Astigmatism
   C. Recovery time and postop healing
   D. Tissue clarity
   E. Refractive goals (contact lens freedom)

III. Advances in Corneal Surgery
   A. Disease-targeted surgery
      1. Descemet-stripping automated endothelial keratoplasty (DSAEK) / Descemet membrane EK (DMEK)
      2. Deep anterior lamellar keratoplasty (DALK)
      3. Stem cell transplant
   B. Femtosecond laser technology
      1. Photodisruption: Creates precise cuts at specified depths, various trephination cut patterns
      2. Femtosecond laser “zig-zag” incision
         a. Wound integrity: More surface area for wound healing, greater mechanical stability of wound incisions, better incision alignment—less torsional or vertical misalignments
         b. Self-sealing laser incisions: Less suture tension with resulting less astigmatism, earlier suture removal, easier suturing, radial incision marks to guide suturing
      3. Femtosecond laser zig-zag PKPs
         a. Consistent levels of astigmatism up to 5 years follow-up
            i. Includes suturing by 10 different fellows in majority of PKs
            ii. Data reported include patients with sutures out.
            iii. Majority of cases with zig-zag C (9 outer mm) → larger graft → lower levels of cyl
         b. Most eyes reaching full potential CDVA by postop month 3, maintained through study period
         c. UCVA / CDVA / cylinder are stable even after suture removal.
      4. Zig-zag DALK
         a. Combined “big-bubble” DALK technique with femtosecond laser zig-zag incision
         b. Risk of rejection becomes almost zero
         c. Extraocular surgery, minimizing risks of open eye surgery

The femtosecond has given surgeons new tools for achieving better and faster visual recovery.
Regrafting the Failed Penetrating Keratoplasty: Should We Still Re-PK?

Donald T H Tan MD FRCS FRCOphth

I. Challenges for Repeat PK for Failed PK
A. Higher allograft rejection rate
B. Higher graft failure rate, shorter graft survival period: Studies show long-term survival rates to range between 21% and 70% (8 studies).

II. Current Alternatives to Repeat PK for Failed PKs
A. Endothelial keratoplasty (EK): Descemet-stripping automated EK (DSAEK) or Descemet membrane EK (DMEK)
B. Boston type 1 keratoprosthesis

III. Advantages of Performing DSAEK (or DMEK) in Failed PKs
A. Potentially lower risk of rejection
B. EK advantages: tectonically stronger eye, closed eye surgery, sutureless, faster visual rehabilitation

IV. Disadvantages of Performing DSAEK in Failed PKs
A. More complex surgery
B. Higher risk of donor dislocation compared to standard DSAEK
C. Residual PK stromal haze or distortion may limit visual acuity.
D. Adoption of original ametropic status of the PK

V. Advantages of Performing Boston Type 1 KPro in Failed PKs
A. No risk of donor rejection affecting central vision
B. Minimal astigmatism, enhanced visual acuity
C. Relative ease of performing surgery (compared to complex EK surgery)

VI. Disadvantages of Performing Boston Type 1 KPro in Failed PKs
A. Complications of Boston type 1 KPro (extrusion, infection, etc.)
B. Shorter follow-up rates in literature; longer follow-up, more complications

VII. Comparative Studies Comparing Repeat PK (PK-PK) with DSAEK for Failed PK (PK-EK)
A. PK-EK 1-year failure rates ranging from 55% to 100% (3 studies)
B. Anshu et al: EK-PK graft survival of 74% at 4 years
C. Kitzmann et al: Trend toward better 3-year survival in PK-EK group
D. Generally better BCVA in PK-EK groups (4 studies)
E. Ang et al: Retrospective analysis within the Singapore Cornea Transplant Study (SCTS)
   1. 113 eyes, all pseudophakic bullous keratopathy (PBK) with failed PKs: 81 PK-PKs, 32 PK-EKs
   2. Five surgeons, EndoGlide DSAEKs, same steroid regimes
   3. Cumulative graft survival probability: PK-PK, 51.3% vs. PK-EK, 86.5% (P = .013)
      a. 1-year survival: PK-PK, 91.9%; PK-EK, 96.2%
      b. 2-year survival: PK-PK, 82.6%; PK-EK, 91.6%
      c. 3-year survival: PK-PK, 66.8%; PK-EK, 86.4%
      d. 5-year survival: PK-PK, 51.3%; PK-EK, 86.4%
   4. More rejection episodes in PK-PK group: 13.6% vs. 3.1%
   5. Multivariate Cox regression analysis: Repeat PK was the only significant risk factor for graft failure; hazards ratio, 10.17 (95% CI, 1.10-93.63; P = .041)
   6. Study conclusion: Performing DSAEK for failed PK is far superior to repeat PK for PBK eyes.

VIII. Current Suggested Approach
Attempt to perform DSAEK for failed PKs, unless there is significant pre-existing, irreversible stromal damage or distortion in the PK, as studies show less rejection, and longer-term graft survival.

IX. Should We Still Be Performing Repeat PK in Eyes with a Previously Failed PK?
Yes, but generally only in eyes with end-stage chronic stromal scarring or distortion in the PK.

X. What about the Role of Boston KPro Type 1 for Repeat PKs?
Current short-term studies comparing graft survival rates between repeat PK and Boston KPro surgery for failed PKs suggest that graft survival is better for the Boston KPro, but graft survival for EKs after PK appears better than Boston KPro results; more long-term KPro results are needed to compare Boston KPro with EK for failed PK.
References


Done with Regrafting? When Is KPro a Better Choice?

Anthony J Aldave MD, Sumayya Ahmad MD, Priya M Mathews MD MPH, Kristina Lindsley MS, Majed Alkharashi MD, Frank S Hwang MD, Sueko M Ng MHS, and Esen Karamursel Akpek MD

Background
In nearly every published series of the Boston type 1 keratoprosthesis, the most frequently implanted keratoprosthesis worldwide, the most common indication for surgery is repeat corneal transplant failure. However, no controlled clinical trials have been performed to compare the outcomes of repeat keratoplasty to keratoprosthesis implantation for graft failure. Thus, in order to attempt to develop evidence-based guidelines regarding optimal management of repeat keratoplasty failure, the published outcomes of repeat penetrating keratoplasty (PK) were compared to those of a multicenter, retrospective review of Boston type 1 keratoprosthesis performed for corneal transplant failure.

Methods
A literature search was performed to identify publications that reported the outcomes of PK for corneal transplant failure. In addition, data were collected from surgeons at 5 centers regarding the outcomes following implantation of the Boston type 1 keratoprosthesis for corneal transplant failure. The primary outcome measure was the percentage of eyes with corrected distance visual acuity (CDVA) ≥ 20/200 at 2 years after surgery, while secondary outcome measures included the percentage of eyes with CDVA ≥ 20/40 at 2 years after surgery; the incidence of graft failure or keratoprosthesis retention failure at 1, 2, and 5 years after surgery; and the incidence of postoperative complications, such as infectious keratitis and development or progression of glaucoma.

Results
The literature search revealed 26 studies that described the results of repeat PK for corneal transplant failure in approximately 5600 patients. During the specified study period, 104 eyes (98 patients) underwent implantation of the Boston type 1 keratoprosthesis for corneal transplant failure at the 5 centers.

Visual Acuity
Two years after surgery, the percentage of eyes with CDVA ≥ 20/200 following repeat PK vs. keratoprosthesis implantation were 42% vs. 57%, while the percentage of eyes with CDVA > 20/40 were 16% vs. 20%, respectively.

Graft Failure / Keratoprosthesis Retention
One, 2, and 5 years after surgery, the cumulative percentage of PKs that failed was 21%, 33%, and 53%, while the percentage of keratoprosthesis retention failures was 1%, 6%, and 25%, respectively.

Complications
Glaucoma either developed or progressed in 25% of eyes following PK and in approximately 30% of eyes following keratoprosthesis implantation. Infectious keratitis developed in 18% of eyes following PK and in 3% of eyes following keratoprosthesis implantation.

Summary
The Boston type 1 keratoprosthesis is the evidence-based procedure of choice for the management of corneal graft failure. However, superior visual outcomes must be weighed against greater risk of sight-threatening complications.

Selected Readings
Breaking the Mold: Surgery Starts in the Eye Bank!

Marian Sue Macsai-Kaplan MD
Case: What Do I Do with This Cornea?!

The Surgical Management of Total Corneal Melts with Perforation: Novel Techniques to Save the Eye, Minimize Rejection, and Preserve the Angle

Mark A Terry MD

Management of extensive corneal and limbal melts from autoimmune disease or infection can be more challenging than managing more central ulcerations and/or perforations. Due to the proximity of a graft to the scleral vasculature, any large penetrating keratoplasty procedure in this area puts the graft at higher risk of rejection and vascularization. When replacing full-thickness tissue in close proximity to the anterior chamber angle, there is a high risk of extensive iridocorneal adhesions, angle closure, and resultant intractable glaucoma. In this presentation, I will present a case for the panel members to discuss regarding their approach to saving the eye, preserving vision, and preventing glaucoma. I will then show a unique surgical strategy to minimize common complications following repair of total corneal melts with limbal involvement.

What Do I Do with This Cornea?

A 46-year-old indigent woman with history of “scratch” 6 days ago presented with total corneal infiltrate and necrosis involving the limbus inferiorly. There was near perforation of the inferior third of the cornea and a 95% hypopyon. There was no view of the anterior chamber. Patient had been on moxifloxacin (Vigamox) from the E.R. for 3 days. Seidel-negative with pressure B-scan showed no involvement of the posterior segment. Preop vision was light perception only. IOP was by palpation, and the eye was felt to be “soft.”

Medical therapy
Cultures and Gram stain were taken and patient was placed on fortified drops of vancomycin and tobramycin every half hour, as well as oral doxycycline. Cultures grew out *Streptococcus pneumoniae*. After 2 days of antibiotics, perforation seemed imminent, and so the patient was taken to the operating theater.

Surgical therapy
What are the options here?

1. Penetrating keratoplasty with a 12-mm limbus-to-limbus graft?
2. Lamellar keratoplasty?
3. Keratoprosthesis?
4. Descemet membrane endothelial keratoplasty?
5. Corneal crosslinking?
6. Enucleation?
7. Referral to your competition?
2017 Advocating for Patients

Darby Miller MD

Ophthalmology’s goal to protect sight and empower lives requires active participation in and commitment to advocacy from every ophthalmologist. Contributions to the following three critical funds are a part of that commitment:

- OPHTHPAC® Fund
- Surgical Scope Fund (SSF)
- State Eye PAC

Please join the dedicated community of ophthalmologists who are contributing to protect quality patient eye care for everybody. The OPHTHPAC Committee is identifying Congressional Advocates in each state to maintain close relationships with federal legislators in order to advance ophthalmology and patient causes. At Mid-Year Forum 2017, we honored nine of those legislators with the Academy’s Visionary Award. This served to recognize them for addressing issues important to us and to our patients. The Secretariat for State Affairs is collaborating closely with state ophthalmology society leaders to protect surgery by surgeons at the state level. This year has seen an unprecedented effort by optometry to advance its scope of practice via legislation rather than education. Our mission of protecting sight and empowering lives requires robust funding of both the Surgical Scope Fund and the OPHTHPAC Fund. Each of us has a responsibility to ensure that these funds are strong.

OPHTHPAC® Fund

OPHTHPAC is a crucial part of the Academy’s strategy to protect and advance ophthalmology’s interests in key areas, including physician payments from Medicare and protecting ophthalmology from federal scope of practice threats. Established in 1985, OPHTHPAC is one of the oldest, largest, and most successful political action committees in the physician community. We are very successful in representing your profession to the U.S. Congress.

As one election cycle ends, a new one starts, yet the pressure to remain vocal on our issues remains. Advocating for our congressional issues is a continuous battle, and OPHTHPAC is always under financial pressure to support our incumbent friends as well as to make new friends with candidates. These relationships allow us to have a seat at the table with legislators willing to work on issues important to us and our patients.

The relationships OPHTHPAC builds with members of Congress is contingent on the financial support we receive from Academy members. Academy member support of OPHTHPAC allows us to advance ophthalmology’s federal issues. We need to increase the number of our colleagues who contribute to OPHTHPAC and the other funds. Right now, major transformations are taking place in health care. To ensure that our federal efforts and our PAC remain strong, we need the support of every ophthalmologist to better our profession and ensure quality eye care for our patients.

The significant impacts that OPHTHPAC has made include the following:

- Derailed the onerous global surgery data collection proposal
- Preserved global surgical payments
- Halted the Part B Drug Demonstration
- Continued efforts in collaboration with subspecialty societies to preserve access to compounded and repackaged drugs such as Avastin

Contributions to OPHTHPAC can be made here at AAO 2017 or online at www.aao.org/ophthpac by clicking “Join.” Leaders of the Cornea Society are part of the Academy’s Ophthalmic Advocacy Leadership Group (OALG), which meets every January in the Washington, D.C., area to provide critical input and to discuss and collaborate on the Academy’s advocacy agenda. The topics discussed at the 2017 OALG agenda included panel discussions on the Merit Based Incentive Payment System (MIPS) and APM implementation, as well as Academy analysis initiatives related to the IRIS® registry. In addition, meeting participants discussed the changing paradigm for optometric scope battles, held a roundtable to discuss challenges for surgical subspecialties, and considered opportunities to ensure physician and patient choice regarding access to pharmaceuticals.

At Mid-Year Forum 2017, the Academy and the Cornea Society ensured a strong presence of cornea specialists to support ophthalmology’s priorities, and a record number of ophthalmologists visited members of Congress and their key health staff to discuss ophthalmology priorities as part of Congressional Advocacy Day. The Cornea Society remains a crucial partner with the Academy in its ongoing federal and state advocacy initiatives.

Surgical Scope Fund

The Surgical Scope Fund (SSF) provides grants to state ophthalmology societies to support their efforts to derail opticometric surgery proposals that pose a threat to patient safety. Since its inception, the Surgery by Surgeons campaign and the SSF, in partnership with state ophthalmology societies, has helped 32 state/territorial ophthalmology societies reject opticometric scope of practice expansion into surgery.

In 2017, your colleagues serving on the Academy’s Secretariat for State Affairs, along with State Governmental Affairs staff and the leaders of state ophthalmology societies, have been put to the task while dealing with an unprecedented number of simultaneous legislative battles. Eleven states have been affected so far this year:

- Alaska
- California
- Florida
- Georgia
- Illinois
- Iowa
- Maryland
- Massachusetts
- Nebraska
- North Carolina
- Pennsylvania
Advocating for Patients

Patient safety setbacks as well as victories will be reviewed during the presentation, but do know that in each of these legislative battles, the benefits from SSF distributions are abundantly clear. The best lobbyists and public relations consultants are contracted as necessary, and media campaigns (including TV, radio, and social media) to educate the voting public are launched when needed to secure success and stop optometry from expanding its scope of practice to include surgery. Each of these endeavors is very expensive, and no one state has the resources to wage one of these battles on its own. Ophthalmologists must join together and donate to the SSF to fight for patient safety when a state faces a scope battle over optometric surgery.

The Academy relies not only on the financial contributions to the SSF from individual ophthalmologists and their practices, but also on the contributions made by ophthalmic state, subspecialty, and specialized interest societies. The Cornea Society contributed to the SSF in 2016, and we thank them and look forward to their contribution in 2017. Contributions to the SSF can be made here at AAO 2017 or online at www.aao.org/ssf.

State Eye PAC

It is also extremely important for all ophthalmologists to support their respective State Eye PACs because campaign contributions to legislators at the state level must come from individual ophthalmologists and cannot come from the Academy, OPTH PAC, or the SSF. The presence of a strong State Eye PAC providing financial support for campaign contributions and legislative education to elect ophthalmology-friendly candidates to the state legislature is critical, as scope of practice battles and many regulatory issues are all fought on the state level.

Action Requested: ADVOCATE FOR YOUR PATIENTS

Academy SSF contributions are used to support the infrastructure necessary in state legislative / regulatory battles and for public education. PAC contributions are necessary at the state and federal levels to help elect officials who will support the interests of our patients. Contributions to each of these three funds are necessary and help us protect sight and empower lives. SSF contributions are completely confidential and may be made with corporate checks or credit cards, unlike PAC contributions, which must be made by individuals and are subject to reporting requirements.

Please respond to your Academy colleagues and be part of the community that contributes to OPTH PAC, the SSF, and your State Eye PAC. Please be part of the community advocating for your patients now.

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Ocular Surface Squamous Neoplasia: Should I Excise, or Do Something Else?

Hans E Grossniklaus MD

I. Ocular Surface Neoplasms

A. Dysplasia (corneal intraepithelial neoplasia [CIN])
   1. Mild: up to 1/3 thickness
   2. Moderate: 1/3 to 2/3 thickness
   3. Severe: 2/3 to full thickness
   4. Carcinoma-in-situ: full thickness

B. Focal (actinic keratosis variety) vs. diffuse CIN

C. Squamous cell carcinoma (SCC) = invasion of substantia propria

D. Variants of SCC
   1. Spindle cell carcinoma
   2. Mucoepidermoid carcinoma

II. Treatment

A. Excision vs. biopsy
   1. Focal (actinic keratosis variety): Excise
   2. Diffuse: Map biopsies

B. Excision technique
   1. Topical / subconjunctival anesthesia
   2. Mark borders of lesion
   3. Excise with 0.12 forceps and Wescott scissors
   4. Excise corneal portion with crescent or 69 blade
   5. Double freeze thaw margins (and base) to −80°C
   6. Close with 6-0 plain suture
   7. Place on cardboard / foam pad and orient
   8. Margins determined based on size of lesion
   9. Corneal (limbal) margin often positive

III. Adjuvant Therapy

A. Mitomycin C (MMC)
   1. 0.02%-0.04% q.i.d.; 2 weeks on, 2 weeks off; may be repeated
   2. Occlude puncta

B. 5-fluorouracil
   1. 1% q.i.d. for 1-2 weeks; may be repeated
   2. May be used if refractory to MMC

C. Interferon (IFN) α2B
   1. 10 million IU/10 cc (compounded) q.i.d. topically for 3-4 months
   2. 10 million IU/1 cc (injected) perilesional every 1-2 weeks (x 3-4 injections)
      a. “Flu in a bottle”
      b. Give acetaminophen / aspirin at time of injection
      c. May be given in OR at time of excision / biopsy in some cases

IV. Diffuse CIN: Consider Other Diagnoses

A. Ocular cicatricial pemphigoid (OCP) biopsies submitted in formalin and Michel’s media for immunofluorescence

B. Pagetoid spread of sebaceous carcinoma
   1. Great mimicker
   2. Biopsy for histology
   3. May have false immunofluorescence (IF) staining

V. Should I Excise, or Do Something Else?

A. Get tissue diagnosis including IF, if needed (OCP, pagetoid spread of sebaceous carcinoma)

B. Actinic keratosis, localized variant, excision usually works; may need adjuvant chemotherapy (IFN) if + margins and/or high grade (severe dysplasia, CIS)

C. Diffuse variant, map biopsies, then treat with adjuvant chemotherapy (IFN)

D. Mode of chemotherapy depends on severity, extent of dysplasia, patient compliance, etc.; may use topical, injections or both for IFN

E. If SCC with deep margin positive, re-excise or consider plaque brachytherapy if needed.

F. If spindle cell carcinoma or mucoepidermoid carcinoma, may need further surgery; monitor for intraocular invasion.

Selected Readings


What to Do When the Tumor Goes Viral . . . Literally
Conjunctival Papilloma Management
Carol L Shields MD

I. Basic Facts
A. Conjunctival papilloma is related to human papillomavirus (HPV) infection.
B. Facts from VaccinateYourFamily.org, founded in 1991 by former first lady Rosalynn Carter to protect families from vaccine-preventable diseases:
   1. HPV is a common virus that can cause cervical, vaginal, and vulvar cancers in females and penile cancer in males. HPV can also cause anal cancer, throat cancer, genital warts, and conjunctival papillomas.
   2. About 79 million Americans are currently infected with HPV.
   3. About 14 million new infections occur per year in the United States.
   4. HPV leads to approximately 11,000 cases of cervical cancer per year in the United States.
   5. HPV is common; nearly all sexually active men and women will be infected at some point in their lives.
   6. Some infected people have no symptoms and the HPV can resolve.
C. HPV types
   1. There are > 100 types of HPV.
   2. Types 6 and 11 cause the common wart of skin, genitals, and conjunctiva. At delivery through the vaginal canal, a child can pick up the mother’s HPV and eventually develop conjunctival papilloma or respiratory papilloma.
   3. Types 6a, 33, and 45 are less commonly found in conjunctival papilloma.
   4. Types 16 and 18 can cause carcinoma of the cervix and conjunctiva.

II. Conjunctival Papilloma Facts
A. Conjunctival papilloma features
   1. Can occur in children (10%) or adults (90%)
   2. Related skin warts (18%), genital warts (3%), and HIV (1%) are noted.
   3. Occurs in the fornix (19%), tarsus (14%), or plica / caruncle (40%)
   4. Those in children are larger and more multifocal, with greater recurrence.
B. Conjunctival papilloma treatments
   1. Surgery: Excision and cryotherapy, “no-touch” technique
   2. Immunotherapy: Good choice
      a. Interferon, topical: 1 million IU/cc q.i.d. for 3 months
      b. Interferon injection ≤ 10 million IU per cc under the mass every 1 month x 4
      c. Cimetidine: 300 mg PO t.i.d.
   3. Chemotherapy: We avoid mitomycin C and 5-fluorouracil for papillomas due to toxicity.
   4. Antiviral therapy: Cidofovir
   5. Photodynamic therapy: Can be effective
   6. Laser therapy: Be careful, as this aerosolizes the HPV and can cause throat papillomas in healthcare workers.
   7. What to do with failures: Check immune status and use immunotherapy.

III. HPV Vaccine: Types of Vaccines
A. Cervarix: Bivalent against types 16 and 18
B. Gardasil: Quadrivalent against types 6, 11, 16, 18
C. Gardasil 9: Nonavalent against types 6, 11, 16, 18, 31, 33, 45, 52, 58
   1. Released 2014 for girls and boys ages 9-15 years old
   2. Three injections
   3. Anticipate 90% of genital warts and cervical cancer prevented
D. Anticipate reduction in conjunctival papillomas and perhaps squamous cell carcinoma

Selected Readings
1. Vaccinate Your Family website, vaccinateyourfamily.org.
Does Imaging Help in the Diagnosis of Conjunctival Tumors? Biopsy or Image?

Afshan Nanji MD

I. Case Presentations

II. Ocular Surface Squamous Neoplasia (OSSN)
   A. Vital dyes: Rose bengal, methylene blue, and toluidine blue are sensitive tests, but are not specific for OSSN.
   B. Cytology
      1. Impression and aspiration cytology are minimally invasive techniques that use cell sampling to evaluate for atypia.
      2. Atypical features include enlarged nuclei, irregular nuclear outline, coarse chromatin, and presence of prominent nucleoli.
      3. Limitations: superficial sampling, decreased sensitivity with keratotic lesions, need for experienced cytologist
   C. In vivo confocal microscopy
      1. Noninvasive technique allowing en face images of the ocular surface down to the cellular level
      2. Atypical features include hyper-reflective pleomorphic cells of varying shapes and sizes, large and bright nuclei.
      3. Mixed results regarding its diagnostic capabilities across various studies
      4. Limitations: requires operator expertise, decreased visualization in highly keratinized lesions
   D. Anterior segment OCT
      1. Noninvasive technique allowing in vivo, cross-sectional, high-resolution images of the ocular surface
      2. Classic characteristics: abrupt transition between normal and abnormal epithelium, epithelial thickening, and hyper-reflectivity in the area of the tumor
      3. Easy to learn and perform
      4. Limitations: shadowing with thick or pigmented tumors, limited ability to detect invasion
   E. Biopsy
      1. Allows for definitive diagnosis
      2. Limitations: invasive, possibility of incomplete sampling, side effect of scarring

III. Conjunctival Melanoma
   A. Cytology
      1. Impression cytology takes advantage of the ascent of atypical melanocytes to the epithelial surface that it is indicative of malignancy.
      2. Limitations: false negative errors, low utility in early precursors to melanoma
   B. In vivo confocal microscopy
      1. Allows detection of cellular changes and invasion
      2. Cellular features suggesting atypia: large cells with prominent nuclei / nucleoli
      3. Limitations: false negatives and only 1 study showing its utility
   C. Anterior segment OCT
      1. High-resolution images can detect cysts that may not be seen on clinical examination.
      2. Clear differentiation between epithelial and subepithelial tumors
      3. Limitations: no ability to detect atypia with primary acquired melanosis, optical shadowing with thick or highly pigmented tumors
   D. Ultrasound biomicroscopy
      1. Noninvasive technique with ability to penetrate opaque tumors, allows for posterior border of tumors to be imaged and to evaluate for invasion
      2. Limitations: limited resolution and restricted availability of the technology
   E. Biopsy
      1. Allows for definitive diagnosis of sampled site
      2. Increased risk of local recurrence, metastatic disease, and tumor-related death with incisional biopsy
Pigmented Lesions: Watch, or Worry?

Victoria M L Cohen FRCOphth

I. History: Key Questions
   A. What was the age of onset?
   B. Has it grown?
   C. Can the pigment be found in both eyes, or one?
   D. What is the genetic background of the patient?
   E. Is the patient immunosuppressed?
   F. Is there a history of sun exposure?
   G. Is there a history of a treated intraocular melanoma?

II. Examination: Key Points
   A. Bilateral / unilateral
   B. Is the pigment multifocal?
   C. Is the pigment flat?
   D. Are there cysts?
   E. Is the pigment mobile?
   F. Are there feeder / intrinsic vessels?
   G. Is there leukoplakia?
   H. Is corneal epithelium / stroma involved?
   I. Double evert the lids at every examination.
   J. Palpate the neck for enlarged lymph nodes.

III. Investigations
   A. Good documentation with slitlamp photography
   B. Anterior segment OCT
   C. Anterior segment, high-resolution B-scan ultrasound or ultrasound biomicroscopy

IV. Surgical Approach
   A. Avoid incision biopsy
   B. Consider controlled map biopsy if multiple sites of primary acquired melanosis.
   C. Dry field
   D. No-touch technique
   E. Avoid subconjunctival injection.
   F. Absolute alcohol for superficial corneal epitheliec-tomy
   G. Lamella dissection of sclera / cornea may be needed (previous surgical notes?).
   H. 2-3 mm margins
   I. Prepare specimen for pathological reporting.

Selected Readings

Pterygium Surgery: Evidence-based Approaches

Darren G Gregory MD

Introduction
Pterygium surgery is an underappreciated art. It is frequently treated as a trivial procedure, often being the first surgery one performs during residency. Recurrent pterygia, however, can be highly problematic for patients and difficult to repair. The potential complications of a recurrent pterygium include unsightly scarring, restricted ocular motility, ocular surface dryness, and decreased vision. It is therefore important for surgeons to choose a surgical technique that safely and effectively minimizes the risk for recurrence.

Surgical Options
Simple excision leaving bare sclera has largely been abandoned (or should be abandoned) as a surgical technique due to an unacceptably high recurrence rate. Conjunctival autografts and amniotic membrane grafts have been used to cover the conjunctival defect following pterygium excision and subsequently decrease the recurrence rate. The grafts can be fixated in place using sutures or fibrin tissue adhesive. Mitomycin C has also been used as an adjunctive therapy to decrease fibroblast activity and thus decrease the rate of pterygium recurrence.

Evidence
The evidence regarding the superiority of the various surgical options is somewhat flawed. Published studies vary with regard to patient populations, pterygium severities, surgical techniques, postoperative care, and recurrence criteria. That being said, all published reports show that the recurrence rate with amniotic membrane grafts is approximately twice the rate of recurrence with conjunctival autografts. Fibrin glue for conjunctival autografts seems to yield shorter surgical times and lower recurrence rates than sutured conjunctival autografts. Adjunctive mitomycin C lowers recurrence rates but risks causing severe, vision-threatening complications such as scleral melts. Its use should be reserved for aggressively recurrent pterygia.

References
Case: A Lump that Stumped Me

Recurrent Conjunctival Myofibrosarcoma: Managed with Triple Application of Episcleral Brachytherapy

Arun D Singh MD

CASE

A 54-year-old man with history of a renal transplant presented with a recurrent conjunctival tumor. Histopathologic diagnosis was established with the use of immunohistochemistry. In total, three $^{125}$Iodine radiation episcleral plaques were used over a period of 49 weeks. Following cicatricial ectropion repair and cataract surgery, visual acuity was 20/20 at 5-year follow-up without evidence of recurrence or radiation retinopathy.

Discussion

Myofibrosarcoma is a rare mesenchymal tumor that can present as ocular surface tumor. Final histopathologic diagnosis can be challenging, and immunohistochemistry is important for evaluation. Myofibrosarcoma should be considered in the clinical differential diagnosis of atypical ocular surface lesions and the histopathologic differential diagnosis of ocular spindle neoplasms.
How Many Technologies Does It Take to Implant a Toric IOL?
Comparison of Toric IOL Rotational Stability

David F Chang MD and Bryan Lee MD JD

I. The Problem
   A. Astigmatic correction requires multiple steps.
   B. Problem of stackable error

   A. 72 Tecnis toric eyes at the end of surgery, then postoperative (PO) hour 1, PO Day 1, PO Month 1, and PO Year 1
   B. Most rotation happened in the first hour after surgery; rotation of 4.1 degrees.
   C. Between PO Hour 1 and PO Year 1, rotation 0.7 degrees

III. Our Study
   A. All toric IOLs, April 2015 to September 2016
   B. Only included eyes with Callisto
   C. Retrospective study
      1. Surgeon chose Alcon vs. Tecnis toric.
      2. Capsular tension ring used at surgeon’s discretion.
   D. Primary outcome: % > 5 or > 10 degrees misalignment at first postop visit

IV. Study Population

V. Repositioning Rate
   Return to the OR because of rotated IOL:
   A. AcrySof: 1.6%
   B. Tecnis: 3.1% ($P = .10$)

VI. Conclusion
   A. AcrySof toric is more rotationally stable than the Tecnis toric.
   B. Tecnis toric clearly has a predisposition to rate counterclockwise.
   C. Trend toward a decreased need to return to the OR to rotate the AcrySof
Is Aberrometry Necessary?
In-Office Aberrometry: Why Do I Care?

Parag A Majmudar MD

I. Basics of Aberrometry
   A. Ray tracing aberrometry uses a series of 256 laser beams through the line of sight through the pupil. The location where the beams contact the retina is captured by a sensor. In an ideal eye, the location for all 256 rays is the fovea. However, lower- and higher-order aberrations in various parts of the eye will result in a wavefront error.
   B. Is unlike Hartmann-Shack, which measures light coming back out of the eye (This is not as physiologic a measure as ray tracing.)

II. What Information Can Aberrometry Provide?
   A. Wavefront map of higher-order aberrations: coma, spherical aberration, trefoil
   B. Root mean square (RMS): Quantification of the aberrations
   C. Refractive maps
   D. Point spread function
   E. Snellen letter aberrations: Simulation of how the letter “E” would look to a patient based on an estimated mathematical derivation of higher-order aberration values
   F. Internal optics aberration analysis: By subtracting corneal aberrations from the total aberration data, it is possible to identify corneal vs. “other” aberrations (typically from crystalline or artificial IOLs).

III. Troubleshooting the Unhappy Multifocal or Refractive Surgery Patient
   A. Identifying the source of vision complaints:
      1. Blur / double vision: coma
      2. Glare / halo / night myopia: spherical aberration
      3. Starburst: trefoil
   B. Wavefront maps can identify whether corneal or “internal” aberrations are responsible.

IV. Identifying Good Candidates for LASIK vs. Refractive Lens Exchange
   If patients have subtle lens changes, it may steer the discussion toward a more appropriate lens-based procedure.

V. Identifying Candidates for Premium Lens
   A. Determining if significant corneal aberrations are present
   B. Measuring optical alignment (angle kappa and angle alpha)

VI. Toric IOL Check
   A. Quickly identifying toric IOL alignment
   B. How much to rotate the lens?

VII. As a Patient Education Tool
   Help patients visually understand their refractive error and benefits of correcting astigmatism at the time of cataract surgery

Selected Readings
Corneal Imaging to Improve Surgical Outcomes

Mohamed Abou Shousha MD

Anterior segment OCT (AS-OCT) has become an essential diagnostic tool for the anterior segment specialist. It provides an in vivo optical biopsy of the cornea to guide the diagnosis and management of anterior segment pathologies.

I. AS-OCT for Corneal Surgery Planning

A. Anterior lamellar keratoplasty: Femtosecond laser–assisted lamellar keratoplasty (FALK)

1. Measure thickness of the corneal pathology using AS-OCT. If residual bed is more than 250 µm, then patient is a candidate for sutureless FALK. This is based on the literature on LASIK that suggested that around 250 µm of residual corneal bed is enough to prevent ectasia.

2. Program femtosecond laser machine to cut at the depth measured using the AS-OCT.

3. Cut both the donor and recipient using the same settings.

4. Remove the recipient’s pathological corneal tissue.

5. Replace it with the corneal donor lenticule.

6. Fit a bandage contact lens over the cornea.

B. Astigmatic keratotomy (AK) and limbal relaxing incisions (LRI): Detect the depth of the cornea at the location of the AK or LRI to decrease the risk of perforation.

C. Intrastromal corneal rings placement

D. Excision of ocular surface tumors

II. Intraoperative OCT to Assist Corneal Surgery

A. DALK

1. Real-time cross-sectional visualization of the corneal layers during surgery without compromising the view of the surgeon or the sterility of the surgical field.

2. AS-OCT helps to guide the depth of the needle tip and air injection in big bubble technique, and to confirm the absence of fluid and air in the interface at the conclusion of surgery.

3. AS-OCT helps detect microperforation of the Descemet membrane and double anterior chamber intraoperatively.

4. AS-OCT helps avoid residual pathology intraoperatively during DALK procedures.

B. Intraoperative OCT in Descemet membrane endothelial keratoplasty and Descemet-stripping automated endothelial keratoplasty surgery (DSEK): Confirm attachment of the graft intraoperatively.

III. AS-OCT for the Postoperative Evaluation of Corneal Surgeries

A. To evaluate detachment of DSAEK grafts; We will show a case with residual Descemet membrane rolled over in the interface, preventing the graft from adhering to the back of the host cornea.

B. Evaluate visually significant interface opacities

C. Diagnosis of epithelial ingrowth

IV. The Future of AS-OCT

A. Corneal microlayer tomography

B. OCT angiography of the anterior segment

References


Tomography: How This Helps Me

Renato Ambrósio Jr MD

I. Key Points

A. Refractive surgery has stimulated considerable progress in corneal and anterior segment imaging and in optical characterization of the eye.

B. From front surface corneal topography, we evolved to 3-dimensional (3-D) corneal tomography with limbus-to-limbus characterization of the front and back corneal surfaces and pachymetric mapping.

C. Corneal anatomical evaluation has further evolved to layered or segmental tomography, with the ability to characterize the corneal epithelial thickness profile and the elevation of the stromal front surface. Further characterization of even more specific structures, such as the Bowman layer and Descemet membrane, have been also demonstrated.

D. There are many clinical applications of 3-D corneal tomography and segmental or layered corneal tomography for different corneal conditions beyond but including refractive surgery.

II. Introduction and History

The continuous need to increase the safety and accuracy of refractive procedures has prompted evolution in corneal imaging, which in turn has impacted the management of many different corneal diseases.

A. The quest to characterize corneal shape began in the middle 1800s with Helmholtz and the keratometer, or ophthalmometer, which was able to measure the corneal curvature within 3 to 4 mm central area.

B. The advent of Placido disc photokeratoscopy with subsequent development of color-coded maps to evaluate the anterior corneal surface represented a major advance in the mid-1980s.1,2

1. Placido disc–based corneal topography is sensitive enough to detect abnormal front curvature patterns of ectatic disease in patients with relatively normal distance corrected visual acuity and unremarkable biomicroscopy.3-5

2. Corneal topography and central corneal thickness have a recognized but limited role in the screening of refractive candidates.5

C. In the late 1990s, the ability to analyze both anterior and posterior corneal surfaces, along with the creation of a pachymetric map in a 3-dimensional reconstruction of the cornea, became possible with corneal tomography devices.6 Horizontal slit scanning tomography was first introduced with the Orbscan (Bausch + Lomb),7 and later, rotational scanning became accessible with the Pentacam (Oculus)8 and other technologies, such as the Galilei (Ziemer), the Sirius (CSO), and the Preciso (iVIS Technologies). Major advances in the development of both topographic and tomographic algorithms have occurred over the 2 decades since, especially for the detection of keratoconus and other ectatic diseases.9-13

D. The concept of segmental or layered evaluation of the corneal thickness was first introduced by Reinstein using digital very high frequency ultrasound.14,15 More recently, the applications of OCT for allowing 3-dimensional visualization and analysis of different corneal layers became commercially available.16-18

III. Applications of Corneal Tomography

A. Screening for ectasia risk prior to refractive surgery

1. The need to enhance sensitivity in detecting mild or subclinical ectatic disease is also supported by the reported cases of ectasia after LASIK without identifiable risk factors (clinical example: see Figures 1 and 2).19,20

These cases, when a thick flap or excessive tissue ablation are excluded, represent the closest to the ideal population for the studies involving screening for ectasia risk. In fact, the analysis of the preoperative data from these cases has provided the most important advances in the field.4,20-22

2. Corneal elevation data in maps depend on the reference surface chosen.23

a. The method of depicting the elevation is the subtraction of the measured surface (either front or back) and a reference shape, which is calculated to have the highest coincident points to a determined area of the cornea that was analyzed. The best-fit sphere (BFS) to the 8-mm zone has been recommended, as it provides adequate data points without the need to use extrapolated data for the majority of cases.13,24

b. The map pattern, the elevation values at the thinnest point and those at maximum elevation within central 4-5 mm zone are the most important characteristics for clinical interpretation.13,24 Different reference shapes, such as the best-fit toric and aspheric ellipsoid (BFTA or BFTE), may be used.25

i. Using the Pentacam, the cut-off criteria for the posterior elevation value at the thinnest point using the BFS was 12 µm; and using the BFTE, 8 µm—with respective sensitivity of 96.28% and 95.04% and specificity of 98.79% and 99.09% for detecting keratoconus.13
Figure 1.

Figure 2.
3. The concept of an enhanced elevation has been introduced by Belin and implemented on the Pentacam. After calculating the standard BFS for the 8-mm corneal zone, a second, “enhanced” BFS for the same zone, excluding the 3.5-mm-diameter zone centered at the thinnest point, is calculated. The difference map from the standard and enhanced BFS will exaggerate any differences (protrusions) within the excluded zone. More than 5 µm of difference for the front elevation and 12 µm difference for the back elevation are considered suspicious. Changes in posterior corneal elevation have been studied to document long-term stability after LASIK, so that using the same BFS for the preoperative corneal information, less than 7 µm operative corneal information, less than 7 µm.

4. Corneal thickness maps enable the characterization of the thinnest point (TP) value and its location, along with thickness distribution. The TP is a more accurate parameter than central thickness for screening ectatic corneal diseases, as well as for calculating the PTA and residual stromal bed (RSB). The pachymetric progression index (PPI) is calculated for every 1 degree of meridians of the cornea, starting from the TP outward.

a. This calculation considers the increase in thickness, comparing to the TP at each point of the cornea, referencing to a normal population.

b. Ambrósio’s relational thickness (ART) values are calculated as the ratios of the TP and the average of the PPI at all meridians (ART-Ave) and the meridian with maximal PPI (ART-Max).

c. The cut-off criteria for ART-Ave for clinical and mild (FFKC) keratoconus were, respectively, 474 µm and 521 µm, with sensitivity and specificity of 99.59% and 98.19% for keratoconus and 91.49% and 93.05% for FFKC. For ART-Max, 386 µm and 416 µm were the cut-offs, with sensitivity and specificity of 99.17% and 97.28% for keratoconus and 85.11% and 93.05% for subclinical disease, respectively.

B. The Pentacam Belin / Ambrósio Enhanced Ectasia Display (BAD)

1. The BAD is a comprehensive display that combines the standard and enhanced BFS elevation maps of the front and back surfaces, and the thickness distribution data. Different topographic parameters are presented as the standard deviation from normality toward disease (d values): anterior and posterior elevation at the TP (8-mm BFS), change in anterior and posterior elevation of the standard and enhanced BFS, thinnest value and location, PPI, ART and maximal curvature (Kmax). The BAD-D final parameter is calculated based on a regression analysis to maximize accuracy for detecting ectatic disease.

2. BAD-D higher than 2.11 was a criterion with sensitivity and specificity of 99.59% and 100% for diagnosing keratoconus, while for detecting mild or subclinical disease, the criteria of higher than 1.22 provided 93.62% sensitivity and 94.56% specificity.

a. BAD-D (v3) values turn yellow in the display when higher than 1.6.

b. Novel series studies demonstrate lower sensitivity for detecting abnormality among the normal topography eyes from VAE cases.

i. This determines the need to further improve the artificial intelligence method for identifying ectasia susceptibility, and even the need to further integrate novel data such as from segmental tomography (ie, epithelial thickness data) and corneal biomechanics.

ii. In a retrospective, nonrandomized study involving preoperative LASIK data from an international pool comprised of 23 post-LASIK ectasia cases and from 266 stable-LASIK cases with over 1 year of follow-up, the criteria of BAD-D higher than 1.29 provided 87% sensitivity and 92.1% specificity. Even though the BAD-D was the most accurate parameter in predicting ectasia risk, the data suggest room for further improvement.

C. Other applications

1. Planning surface ablation with laser phototherapeutic keratectomy epithelial scrape

2. Postoperative understanding of outcomes

3. Planning therapeutic corneal procedures
IV. Conclusions

A. Knowledge of corneal tomography and segmental tomography with epithelial profile represents a major advance in the anatomical and optical characterization of the eye.

B. Such understanding is fundamental in enabling better results in therapeutic procedures for irregular corneas, including cases that had complications related to corneal refractive surgery. While this approach does augment confidence in refractive procedures, there is also a major impact on corneal diseases, such as corneal ectasia.

References


Do We Need an Intraoperative OCT?

Francis W Price Jr MD

Does one need an intraoperative OCT (iOCT)? It depends, and it depends on the type of cases one does. In my experience, iOCT definitely makes a significant difference for lamellar corneal surgery and intracorneal lens sizing. It may also have potential for minimally invasive glaucoma surgery (MIGS) in evaluating placement of implants like the XEN.

iOCT helps improve the safety and efficiency of Descemet membrane endothelial keratoplasty (DMEK) surgery by allowing evaluation of graft orientation while the graft is only partially uncurled. (Typically with the variety of different marking techniques, the graft has to be unfolded most of the way to determine if the endothelium is facing the iris.) iOCT even allows one to determine the orientation in younger, tightly curled donor grafts when there is no separation between the two tightly curled scrolls. Even with thickened, edematous, and hazy corneas, one can not only see the orientation of the graft but also detect subtle problems that might prevent the graft from either completely unfolding or being positioned / centered in the anterior chamber. Just as the operating microscope allows us to see surgery more clearly because of the magnification, iOCT allows us to see another dimension of the tissues: contours of both the donor and recipient that we cannot discern with the coaxial view of operating microscopes.

In Descemet-stripping endothelial keratoplasty (DSEK) surgery, iOCT allows us to see if the graft is actually in apposition to the recipient cornea. In our practice we primarily reserve DSEK for eyes with significant corneal and anterior segment abnormalities. These eyes often have problems that make graft adherence challenging. Determining whether residual fluid is in the graft interface can direct the surgeon to use either milking maneuvers or drainage incisions. On the other hand, if no interface fluid is present, additional treatment is not needed. With both DSEK and DMEK, iOCT can help identify areas of scar tissue or synechia that might interfere with graft positioning and adherence.

In deep anterior lamellar keratoplasty (DALK), a number of reports have shown the advantage of iOCT in positioning the needle close to the Descemet membrane to improve big bubble formation. I find the most significant advantage to be in determining the depth and uniformity of a manual dissection. With iOCT one can evaluate the quality of the dissection plane to make sure the residual stroma is of uniform thickness; this cannot be accomplished with the coaxial view of the operating microscope.

With intracorneal lens surgery, it can be difficult to determine the exact sizing and vault of the implant. One can improve the final vault in the second eye by evaluating the vault of the first eye. iOCT allows us to evaluate the vault in the first eye so the second eye can be more accurately treated the same day, rather than splitting the surgery into two sessions and using in-office OCT to evaluate the vault in the first eye.
Case: Imaging Saved the Day

Sadeer B Hannush MD

Perioperative imaging has changed the landscape of anterior segment surgery in recent years. Optical biometry, Placido-based and elevation topography, aberrometry, specular and confocal microscopy, and optical coherence tomography have empowered the ophthalmic surgeon to make more accurate diagnoses and achieve better surgical results than the same surgeon was able to make just a short decade ago. On occasion, imaging can change the therapeutic approach so significantly that it can save the day! We illustrate 2 such cases:

1. A 72-year-old woman with corneal edema was referred for endothelial keratoplasty. She had a well-positioned posterior chamber IOL and a limited view of the posterior pole. OCT of the macula revealed marked cystoid macular edema (CME). Medical treatment of the CME improved the vision so significantly that the patient elected to delay corneal surgery.

2. An 84-year-old gentleman with Fuchs dystrophy underwent successful Descemet membrane (DM) endothelial keratoplasty with marked clearing of his cornea and improvement in his vision. Three months postoperatively he presented with decreased vision and corneal edema in the same eye. He was advised that his graft had failed and that he needed regrafting. Anterior segment OCT revealed a very shallow DM detachment that was not detectable on slitlamp examination. Rebubbling (air descemetopexy) reattached the DM with complete clearing of the cornea despite a low endothelial cell count.
How to Diagnose Keratoconus: Tried and True vs. New View

Michael W Belin MD

As medicine has moved from treating advanced disease to preventing disease and its sequelae, the treatment of keratoconus and the prevention of ectatic disease have undergone a major change. In the past, treatments were aimed at restoring vision after visual loss, which typically occurs when there is significant anterior corneal surface change to degrade vision. Treatments to slow or prevent progression were not available, and so diagnosing early disease in asymptomatic individuals was not important.

Two major changes have occurred that not only mandate our ability to diagnose clinically evident disease but dictate that we identify individuals in the early stage of disease (subclinical keratoconus), and even patients who appear normal by all imaging but who may be at risk for ectasia if subjected to corneal stress (ie, refractive surgery). Concomitant with this increased need has been a dramatic improvement in our corneal imaging ability.

In the past, corneal diagnostic imaging was limited to anterior surface curvature analysis. As visual loss almost always parallels anterior change, this sufficed when our treatments were limited to patients with significant visual loss. However, as noted above, the diagnosis of early (subclinical) disease is possible only with full tomographic imaging. Tomographic imaging (available from different technologies, Scheimpflug and OCT being the most common) allows a 3-dimensional reconstruction of the anterior segment and measurement of both the anterior and posterior corneal surfaces. Changes on the posterior cornea almost always predate anterior surface changes. These patients have true disease, but they remain asymptomatic. They exhibit posterior ectasia, typically an abnormally displaced thinnest point and abnormal pachymetric progression.

These patients also present for refractive surgery evaluation and, while appearing normal on older technologies (anterior curvature and central pachymetry), need to be excluded from refractive surgery due to the high risk of ectatic progression. One tomographic screening program (Belin / Ambrósio Enhanced Ectasia Display) demonstrates such a patient. There are numerous abnormalities in this highly abnormal eye, in spite of a normal anterior surface (see Figure 2).

Collagen corneal crosslinking (CXL) is another area where full tomographic imaging has great potential advantages over anterior surface analysis. While CXL is currently used to slow or halt progression of symptomatic disease (ie, after visual loss), the greatest potential benefit would be the ability to identify patients who show documented progression prior to visual loss. The example in Figure 3 is from a patient thought to have a non-progressive disease followed over a 3.5-year period. After 3.5 years, anterior surface changes resulted in mild visual loss, but tomographic analysis would have identified significant change 1.5 years earlier, prior to a loss of vision.

Corneal tomography (the “New View”) offers significant advantages over older anterior curvature analysis. The phrase “Tried and True” may be more appropriately applied to this technology.
Contact Lenses in Keratoconus: What Options Do We Have Now?

Deborah S Jacobs MD

I. Contact Lens Use for Keratoconus / Ectasia Involves “Specialty Contact Lenses.”
   A. A “new” field within U.S. optometry
   B. Offered by MDs or ODs globally, depending on national scope of practice / credentialing
   C. Specialty contact lens is a growth area in the global contact lens industry.

II. Specialty Lenses
   A. RGP corneal lenses, keratoconus designs
   B. Piggyback systems
   C. Silicone-hydrogel (Si-Hy) lenses, keratoconus designs
   D. Hybrid lenses
   E. Mini scleral lenses
   F. Scleral lenses
   G. PROSE treatment (prosthetic replacement of the ocular surface ecosystem)

III. Pearls
   A. New Si-Hy lenses with keratoconus designs have extended the use of soft lenses in keratoconus.
   B. New hybrid materials and designs address past failures from lens fragility and hypoxia.
   C. Scleral lenses are in the repertoire of an increasing number of specialty lens fitters.
   D. Scleral lenses are a useful option in cases of RGP corneal lens failure due to instability or tight lens syndrome.
   E. The definition of scleral lenses is evolving. “Mini-scleral,” corneoscleral, and intralimbal lenses may not perform as well as scleral lenses.
   F. PROSE treatment is a good option for contact lens and even scleral lens failures and can accommodate any cone.
   G. PROSE treatment has favorable 1-year outcome in comparison to keratoplasty for moderate to severe keratoconus.

IV. New Paradigm for Contact Lens in Keratoconus
   A. Not a “contact lens failure” without trial of specialty lenses
   B. New designs / materials more comfortable, physiologic
      1. Si-Hy RGP hybrid
      2. Si-Hy keratoconus designs
      3. RGP sclerals / PROSE treatment: Increased expertise among specialty fitters
   C. Penetrating or lamellar keratoplasty only for axial opacity limiting vision, assessed in specialty lens
   D. No regraft for cylinder or recurrence of ectasia without trial of specialty lens

Selected Readings
Deep Anterior Lamellar Keratoplasty vs. Penetrating Keratoplasty: Is One Clearly Better?

W Barry Lee MD

I. Keratoplasty Trends in the United States

II. Deep Anterior Lamellar Keratoplasty (DALK)
   Indications
   A. Central corneal scar
      1. Previous infection
      2. Previous trauma
   B. Anterior corneal dystrophy
   C. Stromal corneal dystrophy
   D. Keratoconus and ectasia

III. DALK Techniques
   A. Manual dissection
   B. Microkeratome-assisted DALK
   C. Anwar’s big bubble DALK
   D. Femtosecond laser-assisted DALK

IV. DALK vs. PK
   A. Complications of penetrating keratoplasty
   B. Risk of intraocular complications
   C. High astigmatism
   D. Poor wound healing
      1. Delayed vision return
      2. Risk of rupture
   E. Endothelial cell loss
   F. Endothelial rejection

V. Penetration Keratoplasty Rejection
   A. Endothelial rejection (ER)
   B. Incidence
      1. ~25% of all PKs undergo ER.
      2. ~5%-10% of all grafts fail from ER.
   C. Significance
      1. Endothelial cell damage
      2. Graft failure
      3. High risk for regraft

VI. Cornea Donor Study
   A. Purpose
      To determine whether donor age is associated with corneal transplant success

B. Methods
   1. Corneas randomly assigned from:
      a. Donors ≥ 66 years old
      b. Donors < 66 years old
   2. 1101 subjects enrolled 1/00 – 0

VII. Endothelial Cell Density Over 5 Years by Donor Age

VIII. Deep Anterior Lamellar Keratoplasty
   A. Advantages over PK
      1. Retains host endothelium
      a. Eliminates endothelial rejection
      b. Reduces endothelial cell loss over time
      c. Prolongs graft survival
      2. Eye bank issues
         a. Do not need excellent endothelium
         b. Saves good endothelial donors for EK
      3. Avoids open sky
      4. Decreased incidence of intraocular complications
         a. Glaucoma
         b. Cataract
         c. Retinal detachment
         d. Cystoid macular edema
         e. Endophthalmitis
         f. Expulsive hemorrhage
      5. Stronger corneal wound
      6. Sutures can be removed sooner.
      7. Shorter duration of topical steroid
   B. Disadvantages
      1. Longer surgical time
      2. Technically more difficult
      3. Visual outcome
         a. Interface haze can limit vision
         b. More residual recipient stroma = increased risk of haze; bare DM = better vision
IX. DALK Postoperative Complications
A. Epithelial healing
B. Suture-related complications
   1. Suture erosion
   2. Suture dehiscence
   3. Suture infiltrate
C. Graft-host disparity
D. Astigmatism
E. Interface haze
F. Rejection
   1. Subepithelial
   2. No endothelial rejection
G. Mild endothelial cell loss
H. Double anterior chamber

X. DALK vs. PK
A. Comparative case series of 30 keratoconus eyes that underwent DALK vs. PK → similar visual outcomes
   1. PK group: More postop astigmatism, endothelial cell loss, rejection episodes
   2. DALK group: Shorter time to suture removal
   3. DALK vs. PK graft survival: Kaplan-Meier rejection-free survival curves showing DALK with fewer rejections
B. Randomized controlled trial of 76 keratoconus eyes that underwent DALK vs. PK
   1. DALK eyes recovered faster vision; topography, spherical equivalent, and final visual outcomes were not different.
   2. DALK eyes showed less rejection and fewer IOP spikes.

XI. DALK vs. PK Rejection
A. DALK vs. PK (corneal scarring or dystrophy) → similar visual and refractive outcomes of 56 eyes
B. PK group: More endothelial cell loss, more rejection episodes
C. DALK group: No endothelial rejections

XII. DALK Survival
A. Retrospective study of 660 consecutive DALK procedures conducted on 502 patients
B. Mean follow-up: 4.5 years
C. Average graft survival rate: 99.3% (rang: 98.5%-100%)
D. Indications
   1. Keratoconus (74%)
   2. Post-herpetic keratitis scarring (15%)
   3. Corneal stromal opacities (11%)
E. Endothelial loss averaged 11%.
   ECD was unchanged between 6 months postoperatively and the last follow-up visits.

XIII. Conclusions
A. DALK is technically challenging to learn but can be mastered with time.
B. DALK and PK have similar visual outcomes.
C. DALK and PK have similar astigmatism outcomes.
D. DALK avoids endothelial rejection and expulsive choroidal hemorrhage, and shows less endothelial cell loss.
E. DALK may have stronger tectonic support, less risk of IOP spikes and quicker vision recovery.
Considerations Concerning Corneal Collagen Crosslinking

Corneal CXL Bibliography

William J Dupps MD PhD, David Glasser MD

Effectiveness


CXL for Infectious Keratitis


Topo-guided PRK plus CXL, LASIK plus CXL


Complications

Epi-on vs. Epi-off

Accelerated Protocols

Cost-Effectiveness
Tearing Up the Old Paradigm for Management of Acute Hydrops

Mazen Y Choulakian MD

Introduction

Acute hydrops is a relatively rare condition, occurring in 2.6%-2.8% of patients diagnosed with keratoconus. There is a male preponderance, and mean age of onset is 25.¹ The pathogenesis of acute hydrops is the following: a break in Descemet membrane and endothelium is followed by aqueous humour entering the cornea, causing marked stromal and epithelial edema. Typically, the edema resolves progressively within 2-4 months, often leaving a visually significant scar in the central or paracentral cornea.² Stromal neovascularization can also be present when edema persists; this may further complicate patient management.

Conservative Management

Observation is a possible option since corneal edema is typically self-limiting. To help with patient comfort, use of lubrication is recommended. This approach should mostly be considered if the break in Descemet membrane is small and corneal edema is mild. Some physicians recommend using a therapeutic contact lens; however, fitting can be difficult in cases of steeper corneas.

Medical Management

Most ophthalmologists tend to use a combination of hypertonic saline drops, topical corticosteroids, cycloplegics, and hypotensives. The hypertonic saline drops cause an osmotic effect on the ocular surface to decrease epithelial and stromal edema. Topical corticosteroids, such as prednisolone acetate 1%, are often prescribed to reduce the inflammatory component of acute hydrops and to help decrease progression of stromal neovascularization. Moreover, topical cycloplegics are used to decrease pain. Topical hypotensives that decrease aqueous fluid production, such as timolol maleate 0.5%, reduce aqueous egress in the stroma.

Surgical Management

In recent years, surgical treatment of acute hydrops has been advocated to hasten resolution of corneal edema and to decrease risk and/or progression of stromal neovascularization. Injection of intracameral gas, pre-Descemetic compression sutures, and keratoplasty have all been reported. Intracameral injection of 0.2 mL of isoexpansile perfluorocarbon, such as 14% C₃F₈ or 20% SF₆, is used to improve reapposition of the torn Descemet to the posterior stroma. This helps close the cleft and promote resolution of the corneal edema. Clinical resolution of edema in these patients is significantly faster than in patients treated solely with a medical approach.³ However, complications from surgical management have been described: pupillary block, cataract formation, and persistent mydriasis. With anterior segment imaging, either anterior segment OCT or ultrasound biomicroscopy, one can measure the size of the Descemet tear. If the tear is of appropriate size, injection of intracameral gas may be indicated. In cases where the tear is too large or too deep, the gas may in fact impede recovery.⁴ Pre-Descemet compression sutures placed perpendicular to the tear may also speed up resolution of stromal edema.⁵ Finally, there are reported cases of progressive Descemet tears that have been managed with endothelial keratoplasty to close the gap from the hydrops.⁶ These novel therapies, from intracameral gas injection to compression sutures to endothelial keratoplasty, may become the mainstays in treatment for patients with acute hydrops in the future.

References

Case: A Corneal Conundrum

Elmer Y Tu MD
I. What Is Dry Eye (DE)?
   A. Symptoms
   B. Signs

II. What Are Mechanisms Underlying DE?
   A. Inflammation
   B. Nerve dysfunction

III. What Point-of-Care Testing Is Available for DE (Including Cost)?
   A. Osmolarity testing (TearLab)
   B. MMP9 (Quidel)
   C. Immunoglobulin E and lactoferrin (Advanced Tear Diagnostics)

IV. What Can These Tests Tell Us about DE?
   A. Underlying pathophysiology
   B. Predicting outcome

V. Information / Cost Analysis

VI. What Can We Expect in the Future?
Scratching the Old: What’s New in Allergic Conjunctivitis?

Deepinder K Dhaliwal MD and Aimee Verner MD

Background

Allergic conjunctivitis is an extremely common condition, with global prevalence of 40% and U.S. prevalence of approximately 20% (approximately 60 million Americans). Allergic conjunctivitis causes a significant reduction in quality of life. Treatment needs to have a rapid onset of action and an extended duration of action and should cause minimal side effects, since ocular allergy can be recurrent and chronic.

The main symptom of allergic conjunctivitis is itching. If the patient does not complain of itching, rethink the diagnosis of ocular allergy. Additional symptoms may include tearing, burning, and foreign body sensation. Signs of allergic conjunctivitis include tearing, conjunctival and eyelid hyperemia, chemosis, and eyelid swelling.

The basic pathophysiology of the main forms of allergic conjunctivitis is a type I hypersensitivity reaction that involves sensitization of the immune system upon first exposure of the antigen. After repeated exposure, the antigen-specific IgE binds to mast cells in the conjunctiva, triggering their degranulation. Released from the mast cell are histamine, tryptase, chymase, heparin, chondroitin sulfate, prostaglandins, thromboxanes, and leukotrienes. This is the acute phase of the allergic response.

Histamine

- binds to H1 receptors on nerve endings and causes itching
- binds to H1 and H2 receptors on conjunctival vessels and causes vasodilation

Non-Vision Threatening Ocular Allergy (>90% of cases)

- Seasonal allergic conjunctivitis (SAC): Caused by airborne pollens from trees, grass, and weeds. Symptoms are episodic and can occur in spring, summer, or fall (depending on exact etiology).
- Perennial allergic conjunctivitis (PAC): Caused by mold, dust mites, cockroaches, animal dander (chronic exposure)

Vision-Threatening Ocular Allergy (small percentage of cases)

- Vernal keratoconjunctivitis (VKC): Found in young boys in equatorial regions of the world
- Atopic keratoconjunctivitis (AKC): Ages 20-50, history of atopic dermatitis (eczema) as child

The majority of allergy patients have nasal symptoms (allergic rhinitis) in addition to conjunctivitis. Allergy can also affect the mucous membranes in the airway and cause more throat itching, as well as asthma. Treatment should be directed to the site of allergy. Oral nonsedating antihistamines such as cetirizine, fexofenadine, loratadine etc. cause significant ocular dryness. This can be problematic, especially if perioperative (prior to laser vision correction or cataract surgery). Our protocol for treating ocular allergy patients is to stop oral antihistamines and instead treat the allergy locally with drops, nasal spray, or inhaler. Also, the antileukotriene montelukast Singulair can be a helpful adjunct to treating allergy systemically without the ocular drying effects.

In patients preparing for surgery, it is not advisable to simply stop the oral antihistamine to avoid the ocular drying effects since untreated ocular allergy can cause severe corneal haze with PRK and increased diffuse lamellar keratitis with LASIK.

Treatment Options

- Old: first-generation antihistamine with vasoconstrictor
  - Problem: Short duration of action (q.i.d. dosing necessary), tachyphylaxis, rebound effect
  - Examples: pheniramine maleate + naphazoline (Naphcon-A, Visine-A, Opcon-A)
- Mast cell stabilizer
  - Problem: Ineffective in relieving existing signs and symptoms of ocular allergy, need to start prior to allergy symptoms
  - Examples: cromolyn sodium, lodoxomide tromethamine, pemirolast potassium, nedocromil sodium
- Antihistamine plus mast cell stabilizer
  - Excellent first-line therapy due to rapid onset of action and prolonged effect
  - Examples: olopatadine HCl (Patanol), ketotifen fumarate (Zaditor, Alaway), azelastine HCl (Optivar), epinastine HCl (Elestat), betopatine (Bepreve)
- Topical steroids: loteprednol etabonate (Alrex and Lotemax)
- Nonsedating oral antihistamines
- Immunotherapy: Allergy shots (subcutaneous immunotherapy [SCIT])
- Nonpharmacologic
  - Education is very important.
  - Allergy testing to figure out the cause of allergy
  - Allergen avoidance, avoid being outdoors during high pollen days. Sunglasses can provide a barrier.
  - No eye rubbing because eye rubbing degranulates mast cells → makes itching worse, creates vicious cycle.
  - Ice cold compresses (stops itch), art tears (dilutes allergen). Avoid punctal plugs.
  - Wash hair, change clothes as soon as enter house. No animals sleeping in bed (if allergic).
  - If dustmite allergy: Hypoallergenic bedding, wash sheets in hot water. Put pillows in dryer on high heat.
New Therapies

- Sublingual immunotherapy (SLIT), in which a tablet is placed under the tongue daily
- 4 FDA approved products: Oralair (5 kinds of northern grass pollen), Grastek (timothy grass pollen), Ragwitek (short ragweed), and Odactra (house dust mites)
- Topical cetirizine
- Newer targets to block inflammatory cascade: spleen tyrosine kinase (Syk), aldehyde-trap, toll-like receptor 2
- New application of existing drug: tacrolimus
- Oral administration of staple foods engineered to express allergens for oral immunotherapy (transgenic rice seeds that express cedar pollen)

References

Stevens-Johnson Syndrome: A Chronic Problem

Wuqaas M Munir MD

I. Background
   A. Classification and overlap of Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN)
   B. Pathogenesis and differential diagnosis of severe immunologic dermatobullous conditions
   C. Incidence and associations of SJS/TEN with ocular involvement

II. Acute SJS/TEN
   A. Systemic manifestations
   B. Ocular manifestations and key examination findings
      1. Inflammation and epithelial sloughing
      2. Membrane formation
      3. Symblepharon
      4. Corneal complications
   C. Acute ocular therapy
      1. Role of systemic therapies (limited)
      2. Topical supportive therapy
      3. Topical immunotherapy
      4. Surgical therapy
         a. Amniotic membrane transplantation: Techniques
         b. Membrane lysis

III. Chronic SJS/TEN
   A. Systemic manifestations
   B. Ocular manifestations and key examination findings
      1. Conjunctival cicatricial changes
      2. Poor tear film
      3. Inhibited eyelid function and eyelid malposition
      4. Motility restriction
      5. Limbal stem cell deficiency
      6. Corneal complications
   C. Functional deficits and effect on activities of daily living
   D. Chronic ocular therapy
      1. Role of systemic therapies
      2. Topical supportive therapy
         a. Lubrication
         b. Autologous serum
         c. Topical immunotherapy
         d. Scleral contact lenses
      3. Surgical therapy
         a. Punctal occlusion
         b. Tarsorrhaphy
         c. Amniotic membrane transplantation
         d. Epithelial or stem cell transplantation
         e. Fornix reconstruction
         f. Keratoplasty
         g. Keratoprosthesis

Selected Readings

Revisiting a Dry Topic: Is Anything New with Sjögren Syndrome?

Stephen C Pflugfelder MD

I. Risk Factors
A. High interferon (IFN) activity correlates with severity of ocular surface disease.
B. Intestinal dysbiosis is associated with more severe ocular and systemic disease severity.

II. Ocular Surface Disease Profile
A. Greatest perturbation of tear function and ocular surface health among dry eye conditions
B. Heightened response to desiccating environmental challenge
C. Severe conjunctival goblet cell loss that may create a vicious immune cycle with:
   1. Loss of immune tolerance
   2. Greater IFN-γ production

III. Diagnosis
Antibodies to salivary gland protein 1 (SP1) and parotid secretory protein (PSP) may identify patients who are Sjögren-specific antibody A and B negative.

IV. Therapy
A. Increase conjunctival goblet cell number / secretion
   1. Blood products (serum / plasma rich in growth factors)
   2. Nasal stimulation of goblet cells
B. Protect the cornea from desiccation with scleral contact lenses

Selected Readings
Sclera on Fire: What Labs Do I Really Need to Check?

Donald U Stone MD

I. Infectious Scleritis
   A. Take a history: Postsurgical, traumatic
      1. Fungal (cultures and stains)
      2. Bacterial (cultures and stains)
   B. Systemic infections, via local or hematogenous spread
      1. Tuberculosis (systemic tests, ± local culture or biopsy)
      2. Viral: herpes family
      3. Lyme borreliosis (serum titers)
      4. Brucellosis (serum titers, culture)

II. Noninfectious Scleritis
   A. Rheumatoid arthritis
   B. Systemic vasculitis
      1. Antineutrophil cytoplasmic antibody associated
      2. Systemic lupus erythematosus
      3. Behçets
   C. Sarcoidosis
   D. HLA B-27 associated
      1. Ankylosing spondylitis
      2. Psoriatic arthritis
      3. Inflammatory bowel disease
   E. Relapsing polychondritis
   F. Bisphosphonate associated

III. Surgically Induced Necrotizing Scleritis

IV. Masquerade – Neoplasia
   A. Choroidal tumor (melanoma)
   B. Conjunctival tumor (squamous or melanocytic)
   C. Lymphoma

V. Summary of Diagnostic Approach
   A. Thorough history
   B. Test for systemic infections
      1. Treponemal (syphilis) test: treponemal antibodies, rapid plasma reagin
      2. Tuberculosis
         a. PPD or serum QuantiFERON test
         b. Chest imaging
   C. Test for systemic autoimmune conditions
      1. Rheumatoid factor, cyclic citrullinated proteins (CCP)
      2. Anti-neutrophil cytoplasmic antibodies (P-ANCA and C-ANCA)
      3. Antinuclear antibodies
      4. HLA B27
      5. Renal function (creatinine) and urinalysis
      6. Complete blood count, erythrocyte sedimentation rate, C-reactive protein are nonspecific.
      7. Other tests based on suspicion
   D. Pregnancy test before imaging or systemic therapy!
      Urine or serum beta-HCG

Selected Readings
Case: Not Just Another Red Eye
Superior Limbic Keratoconjunctivitis

Christopher J Rapuano MD

I. Introduction
Superior limbic keratoconjunctivitis (SLK) is an uncommon, usually bilateral, chronic, relapsing, inflammatory reaction that may be associated with thyroid dysfunction (~30%). It is often found in conjunction with dry eye syndrome and usually affects middle-aged females (3:5:1 female-to-male ratio).

II. Etiology
Unknown, but most likely related to mechanical trauma involving the superior palpebral and lax bulbar conjunctiva

III. Symptoms
A. Foreign-body sensation, burning, photophobia, pain, red eye, excessive blinking
B. May have exacerbations and remissions

IV. Signs
A. Hyperemia, thickening, redundancy, and laxity of superior bulbar conjunctiva
B. Lack of luster and positive staining of superior bulbar conjunctiva with fluorescein, lissamine green, and rose bengal dyes
C. Fine, velvety, superior tarsal papillae
D. Superior corneal filaments, punctate erosions, and occasionally pannus

V. Workup
A. History; ask about thyroid disease.
B. Slitlamp examination with special attention to the superior bulbar and palpebral conjunctiva
C. Fluorescein, lissamine green, or rose bengal staining of superior bulbar conjunctiva
D. Dry eye testing
E. Thyroid function testing

VI. Treatment
A. Preservative-free artificial tear drops every 2 hours. Consider temporary or permanent punctal occlusion.
B. Cyclosporine 0.05% to 2% b.i.d. to q.i.d. may be helpful. Lifitegrast 5% b.i.d. may also be beneficial. Occasionally serum tears can be helpful.
C. Acetylcysteine (eg, Mucomyst) 10% drops q.i.d. for treatment of corneal filaments
D. Topical antihistamine / mast cell stabilizers (eg, azelastine, epinastine, ketotifen, nedocromil, olopatadine, bepotastine, alcaftadine) q.d. to b.i.d. may be helpful.
E. Application of silver nitrate 0.5% solution to superior bulbar and palpebral conjunctiva for 15 to 30 seconds
F. Localized cautery to superior conjunctiva
G. Double freeze-thaw cryotherapy
H. Surgical resection with or without amniotic membrane graft of superior bulbar conjunctiva

VII. Prognosis
Good for improvement in symptoms; fair for complete resolution of symptoms. May be recalcitrant to treatment.

Selected Readings
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