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Solving the problem of the red eye

Jay M. Haynie
Pacific University

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Solving the problem of the red eye

Abstract

This software is designed to aid the clinician and the student in the differential diagnosis and treatment of a red eye. The program, consisting of three disks, was designed with HyperCard v. 1.2.5 for the Macintosh computer. Included in the program are objective and subjective findings, diagnostic tests, suggested treatment regimens and any contraindications, follow-up care and prognosis for forty-five etiologies of a red eye. One of the major features of this program allows the clinician to enter subjective and/or objective findings. Given these findings, the computer will provide a list of possible ocular conditions. From this list, the clinician can select one of the possibilities listed in which he/she feels is most appropriate for that particular patient. At this point the program will proceed to a stack of information specific for the selected condition. Once a choice has been made, the clinician always has the freedom of returning back to the list of possible ocular conditions.

Degree Type

Thesis

Degree Name

Master of Science in Vision Science

Committee Chair

Craig E. Bowen

Subject Categories

Optometry

SOLVING THE PROBLEM OF THE RED EYE

by

Jay M. Haynie, B.S.
Kenneth D. Ridder, B.A.

A thesis submitted to the faculty of the
College of Optometry
Pacific University
Forest Grove, Oregon
for the degree of
Doctor of Optometry
April, 1992

Faculty Advisors:

Craig E.Bowen, O.D.
Salisa K. Williams, O.D.

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Salisa K. Williams, OD

Salisa K. Williams, O.D.

Biographies

Jay M. Haynie received his B.S. in Visual Science from Pacific University, Forest Grove, OR in 1989. He is a candidate for an O.D. degree at Pacific University College of Optometry in May of 1992. He has been a member of Beta Sigma Kappa, Phi Theta Epsilon, and Phi Theta Kappa during his college career. He has currently been selected to the residency program at American Lake Veterans Hospital in Tacoma, WA . Upon completing the residency program, his future plans are to enter private practice in Olympia, WA.

Kenneth D. Ridder received his B.A. in EPO Biology from the University of Colorado, Boulder in 1988. He is a candidate for an O.D. degree at Pacific University College of Optometry in May of 1992. He has been a member of Beta Sigma Kappa, Phi Theta Epsilon, and Phi Theta Kappa during his college career. His future plans include practicing in a group practice or partnership in Colorado or Oregon.

Craig E. Bowen, O.D. received his Bachelor of Science degree from Alma College, Alma MI. He received his doctorate in Optometry from Pacific University College of Optometry in 1986. Clinical Professor of Optometry from 1989 to present. He has a private practice in Tualitin Oregon.

Salisa K. Williams, O.D., a graduate of Northeastern State University College of Optometry, is an Assistant Professor at Pacific University College of Optometry with primary emphasis in the areas of ocular disease and pharmacology. Prior to assuming her current position at Pacific University College of Optometry, Dr. Williams worked in hospital and primary care clinic settings in Alaska and Nevada while employed by Indian Health Service. She maintains affiliations with numerous professional associations. She has presented continuing education throughout the Western United States and is a past recipient of the American Public Health Association's prestigious award for an outstanding paper and project.

Abstract

This software is designed to aid the clinician and the student in the differential diagnosis and treatment of a red eye. The program, consisting of three disks, was designed with HyperCard v. 1.2.5 for the Macintosh computer. Included in the program are objective and subjective findings, diagnostic tests, suggested treatment regimens and any contraindications, follow-up care and prognosis for forty-five etiologies of a red eye. One of the major features of this program allows the clinician to enter subjective and/or objective findings. Given these findings, the computer will provide a list of possible ocular conditions. From this list, the clinician can select one of the possibilities listed in which he/she feels is most appropriate for that particular patient. At this point the program will proceed to a stack of information specific for the selected condition. Once a choice has been made, the clinician always has the freedom of returning back to the list of possible ocular conditions.

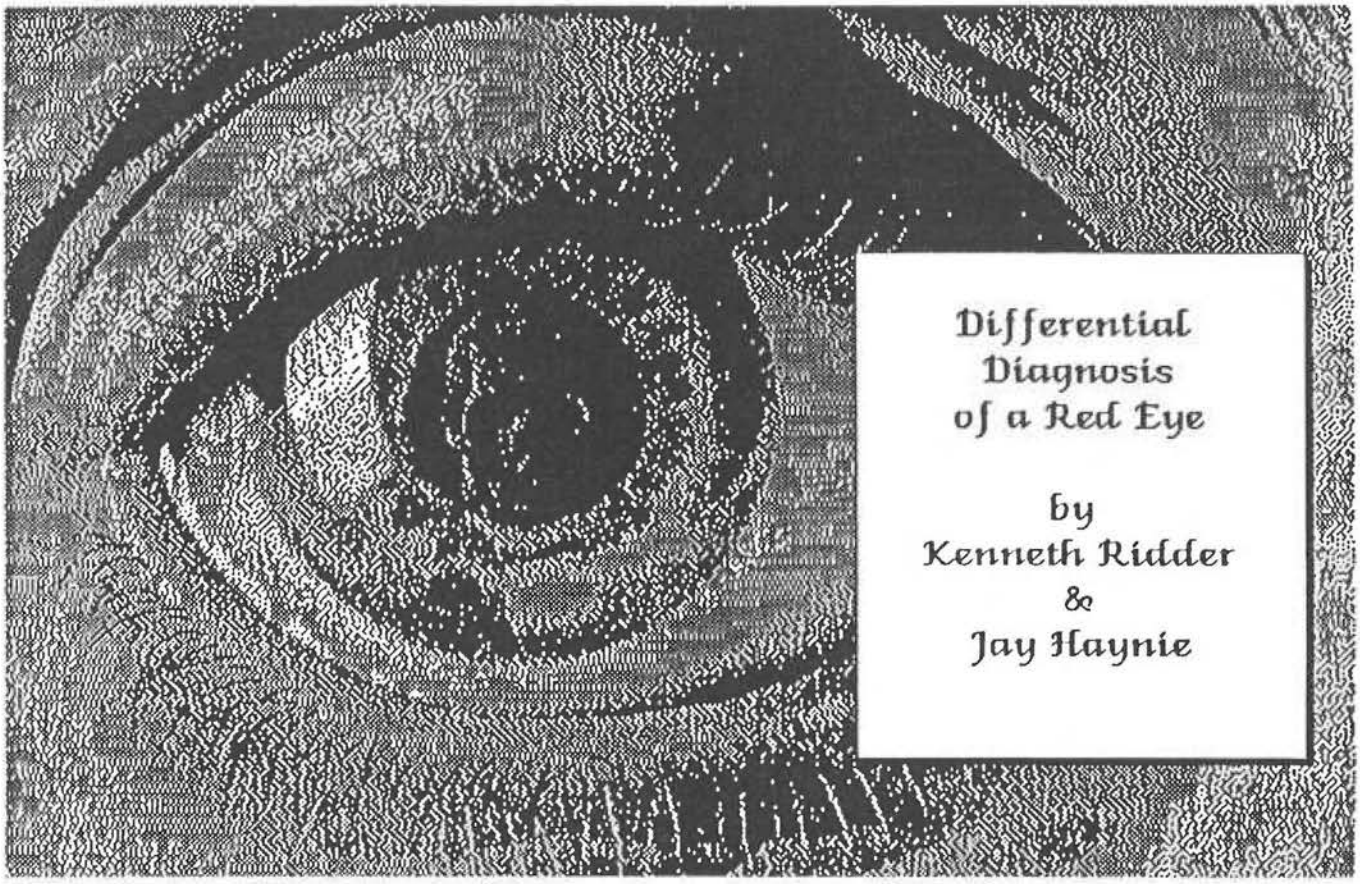
Acknowledgements

The authors would like to thank Dr. Craig Bowen for giving us the idea for such a project.

We would also like to thank Dr. Bowen, Dr. Salisa Williams, Dr. Steven Rogers, Dr. Edward Zayac and Dr. William Shreck for taking the time to proofread and offer their suggestions to our project.

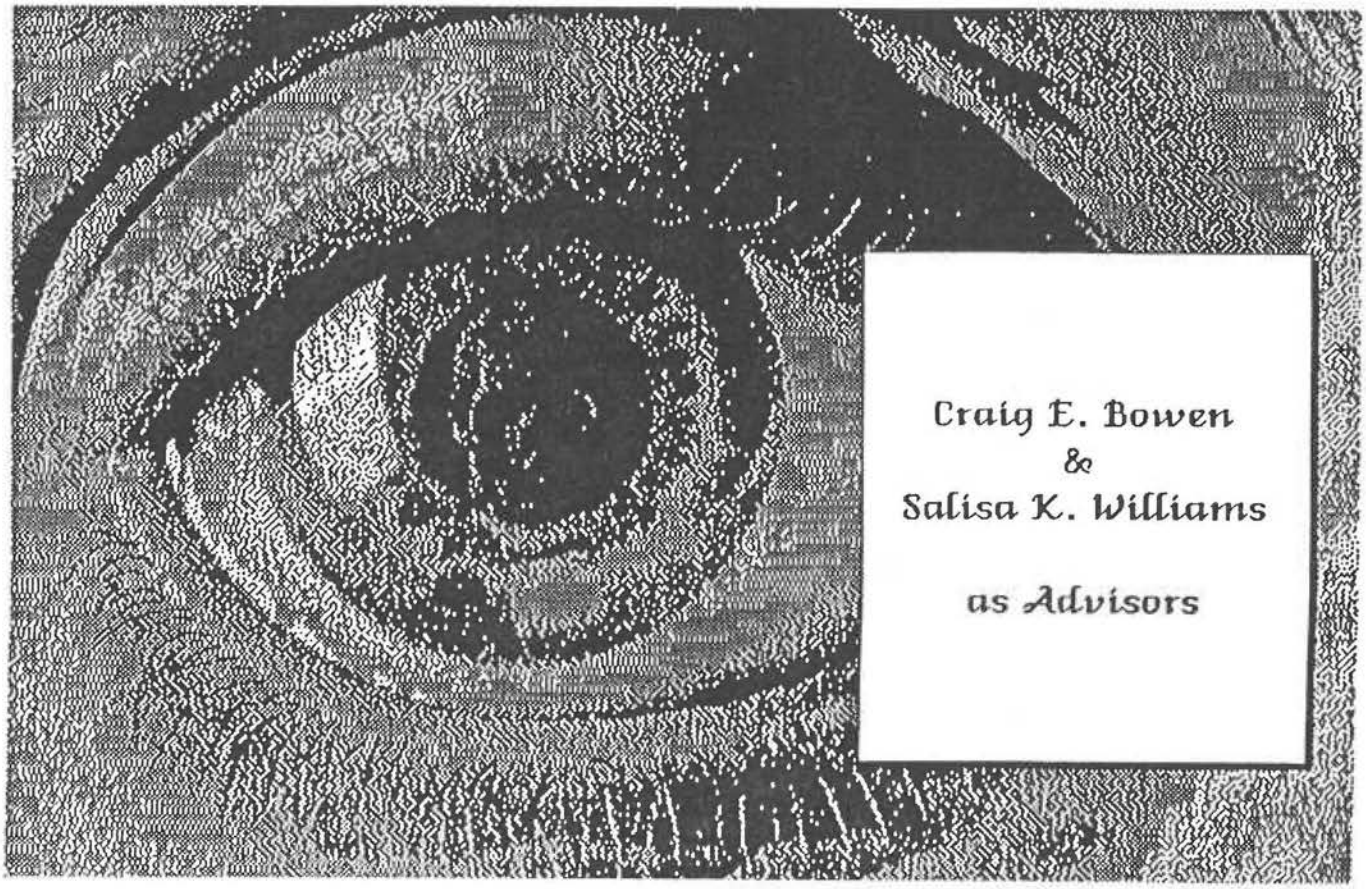
List of conditions covered in this program

Acute Angle Closure Glaucoma
Acute Interstitial Keratitis
Allergic Conjunctivitis
Anterior Uveitis
Bacterial Conjunctivitis
Blepharitis
Canaliculitis
Chemical Burns
Chlamydial Conjunctivitis
Conjunctival Foreign Body
Contact Dermatitis
Contact Lens Related Etiologies
Corneal Abrasion
Corneal Foreign Body
Corneal Ulcer
Entropian
Epidemic Keratoconjunctivitis
Episcleritis
Exposure Keratopathy
Filamentary Keratopathy
Floppy Eyelid Syndrome
Giant Papillary Conjunctivitis
Herpes Simplex Keratitis
Herpes Zoster Ophthalmicus
Hyperacute Bacterial Conjunctivitis
Keratoconjunctivitis Sicca
Ocular Pemphigoid
Ocular Rosacea
Orbital Cellulitis
Parinaud's Conjunctivitis
Phlyctenulosis
Pterygium
Recrurrent Corneal Erosion
Scleritis
Stevens-Johnson Syndrome
Subconjunctival Hemorrhage
Superficial Punctate Keratopathy
Superior Limbic Keratoconjunctivitis
Thermal / UV Keratopathy
Trachoma
Traumatic Iritis
Trichiasis
Vernal Conjunctivitis
Viral Conjunctivitis



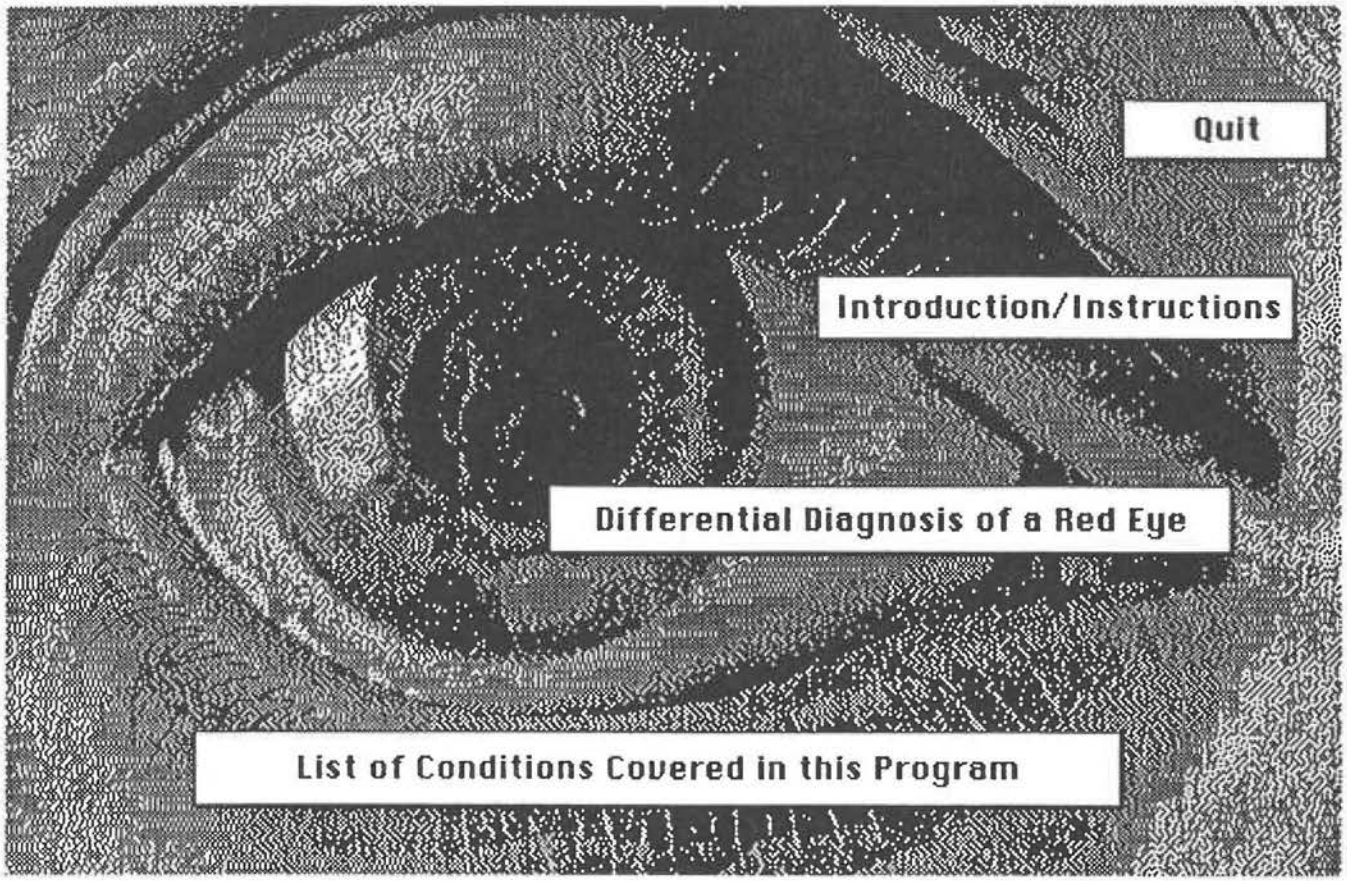
Differential Diagnosis of a Red Eye

by
Kenneth Ridder
&
Jay Haynie



Craig E. Bowen
&
Salisa K. Williams

as Advisors


Quit
Introduction/Instructions
Differential Diagnosis of a Red Eye
List of Conditions Covered in this Program

This software is designed to aid both the student and the clinician in the differential diagnosis of a red eye. There are four main sections to this program:

- Main Menu
- A list of all conditions covered in the program which are separated by tissue involved. This can serve as a menu to rapidly go from disease to disease.
- Differential Diagnosis: By entering signs and symptoms, the computer will come up with a "List of Possibilities".
- 45 "Disease Stacks": These stacks contain information about each condition including treatment and follow-up.

Since ocular conditions do not always present with the same signs and symptoms, we have programmed each condition with the TYPICAL signs and symptoms. As students, we have limited clinical experience and have relied heavily on textbooks and our advisers for the information contained in this project.

We have tried to be as specific as possible in describing the management of those conditions that are commonly treated by optometrists in states with therapeutic laws. Our treatment regimens are general for those conditions that are best treated by other health care professionals. Keep in mind that most therapeutic modalities described here are not the only way to treat that particular condition but are ones that were commonly given in the references we used. Therefore, they are guidelines and not absolutes. It is beyond the scope of this program to list all contraindications and side effects of drugs listed here. Please consult the Physicians Desk Reference if questions exist and to keep abreast of revised recommendations.

Click on arrow for more. ➡

We realize that with a project of this magnitude and our limited clinical experience, that errors and omissions may exist. Again we have strived to be as complete and concise as possible, but we recommend that you use this only as a guide and not as the sole source in treating conditions, especially those that you are not familiar with treating. Therefore, we do not imply or accept professional liability for treatment of those conditions included in this software.

Although every possible cause for a red eye has not been included, we hope that this program is helpful to all that use it, and we welcome any suggestions or corrections so that we can include them in the next version.

Sincerely,

Kenneth Ritter Jay M. Haynie

Credits/References ➡

How to use this program ➡

Credits/ References

- We would like to thank Dr. Craig E. Bowen for giving us the idea for such a project.
- We thank Dr. Bowen, Dr. Salisa Williams, Dr. Steven Rogers, Dr. Edward Zayac, and Dr. William Shreck for proofreading and offering their suggestions.

References Used **Authoring**

- Friedberg M, Rapuano C: Office and Emergency Room Diagnosis and Treatment of Eye Disease Wills Eye Hospital J.B. Lippincott
- Catania, Lou : Primary Care of the Anterior Segment Appleton & Lance
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- Spalton, Hitchings, Hunter: Atlas of Clinical Ophthalmology Lippencott
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- Kanski Clinical Ophthalmology
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- Scott, Clifford Clinical Glaucoma Contact Lens Spectrum11/90
- Yolton, Diane Class Notes Ocular Disease I, II, Pacific University Fall 88, Spring 89
- Williams, Mark and Pepe, Alex Class Handouts Opt. 603 Pacifc University Fall 91
  - Red Eye Work up Alex Pepe
  - Ocular Trauma Alex Pepe
  - Viral Ocular Disease Mark Williams
- Williams, Salisa Class Notes Therapeutic Pharmocolgy, Pacific University Spring 91
- Selected articles from Review of Optometry
- Selected articles from Journal of Ophthalmology

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References Used

Jay Haynie

- Exposure Keratopathy
- Phlyctenulosis
- Blepharitis
- Hyperacute Bacterial Conjunctivitis
- Viral Conjunctivitis
- Chlamydial Conjunctivitis
- Allergic Conjunctivitis
- Parinaud's Conjunctivitis
- Ocular Pemphagoid
- Contact Dermatitis
- Floppy Eyelid Syndrome
- Canaliculitis
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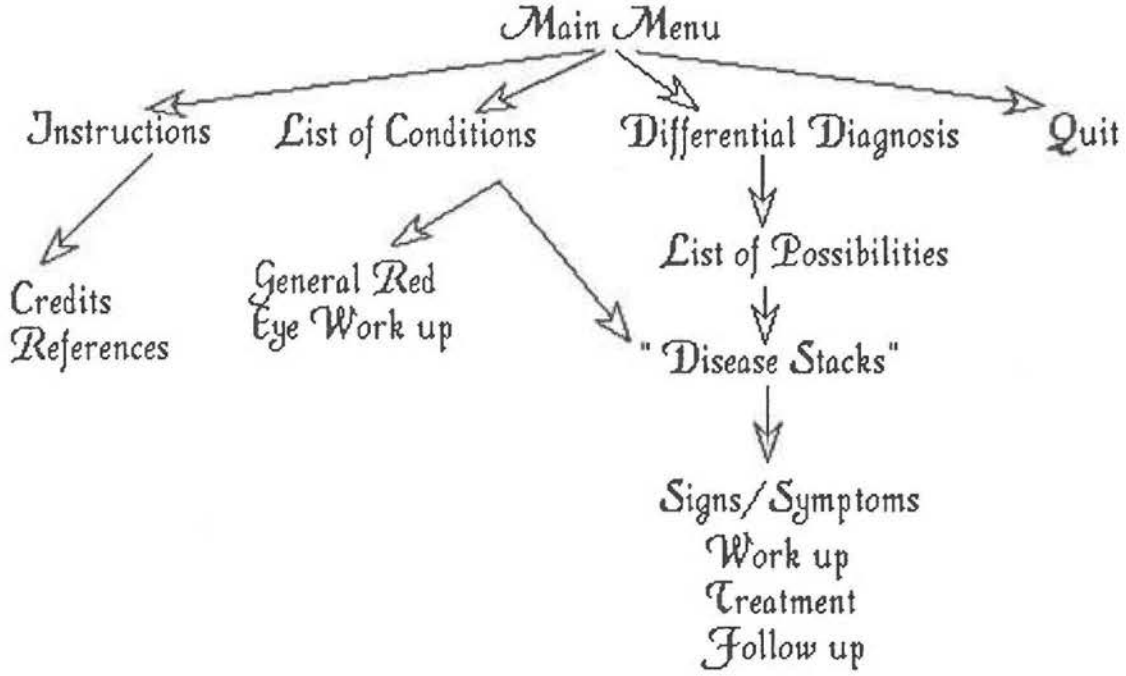
- Ocular Rosacea
- Steven Johnson's Syndrome
- Keratoconjunctivitis Sicca
- Giant Papillary Conjunctivitis
- Bacterial Conjunctivitits
- Trichiasis
- Entropian
- Sub Conjunctival Hemorrhage
- Pterygium
- Epidemic Keratoconjunctivitis
- Trachoma

Authoring

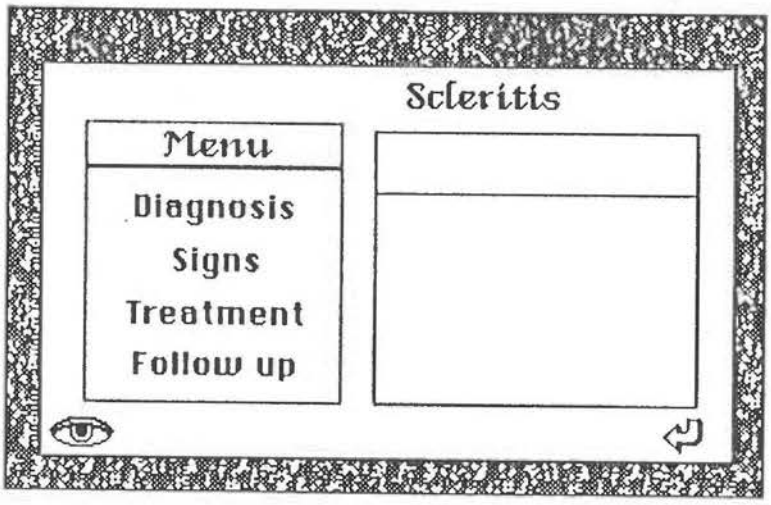
Kenneth Ridder

- DDx of Red Eye (Introduction)
- Differential Diagnosis 3.01
- Corneal Abrasion
- Corneal Ulcer
- Anterior Uveitis
- Acute Angle Closure Glaucoma
- Scleritis
- Chemical Burns
- Corneal Foreign Body
- Conjunctival Foreign Body
- Superficial Punctate Keratopathy
- Filamentary Keratopathy
- Recurrent Corneal Erosion
- Thermal/UV Keratopathy
- Herpes Zoster Ophthalmicus
- Herpes Simplex Keratitis
- Acute Interstitial Keratitis
- Superior Limbic Keratoconjunctivitis
- Episcleritis
- Orbital Cellulitis
- Contact Lens Related
- Traumatic Iritis

This program is set up in the following manner. Navigation between screens is accomplished by "clicking buttons". Click the arrow in the bottom right hand corner of the screen.



Below are some examples of buttons used on the "Menu Cards" for each disease. Click on "Diagnosis" under the Menu below.



Main Menu

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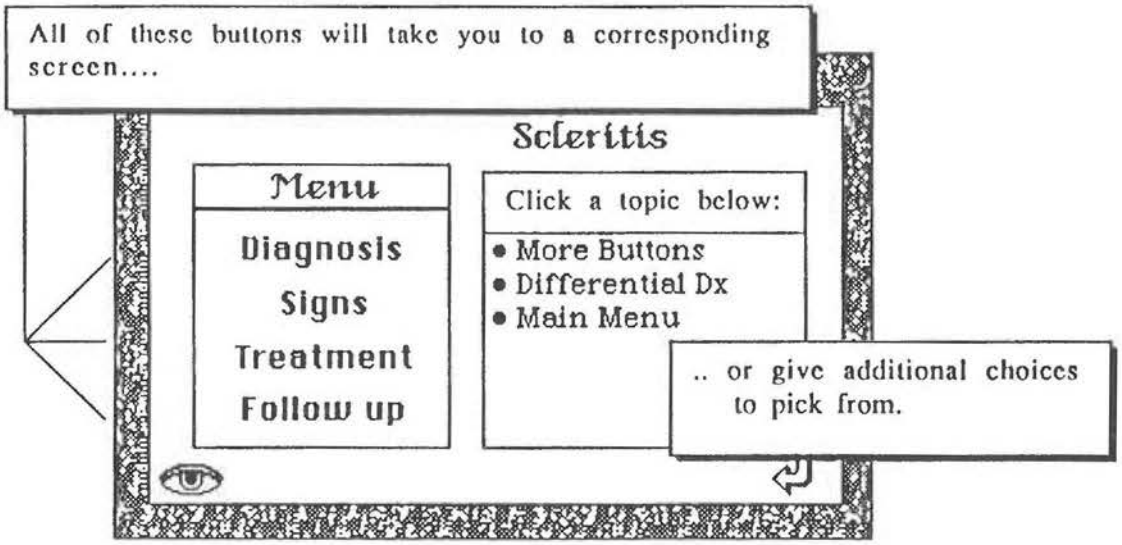
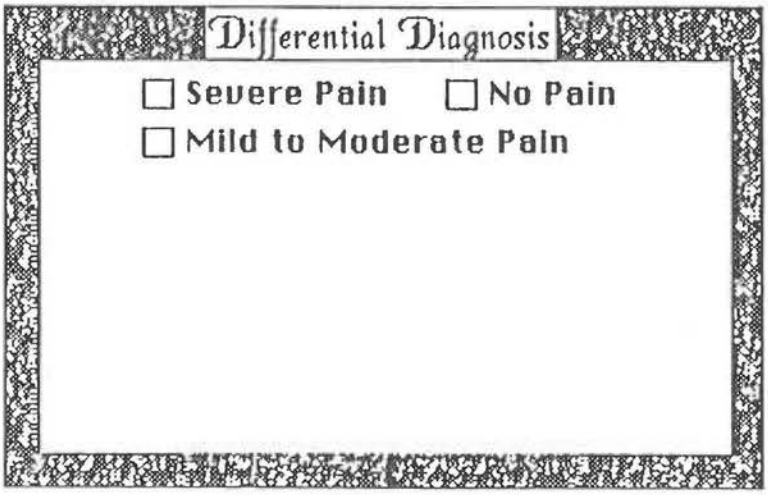


Main Menu

Below is an example of what the "Differential Diagnosis" card looks like. To begin, you must decide if pain is to be included in the differential, and if so to what degree. "Click" one of the buttons below.



Below are more examples of other buttons used in this program. Some will "auto hilite" and some will not. Click the button "No Pain"

No Pain ⟵ Buttons like this are used to enter in signs/symptoms.

Start again
Hide
To Menu
⟶ These buttons perform specific functions.

⟵ Back to "List of Possibilities".

⟵ To Quit program

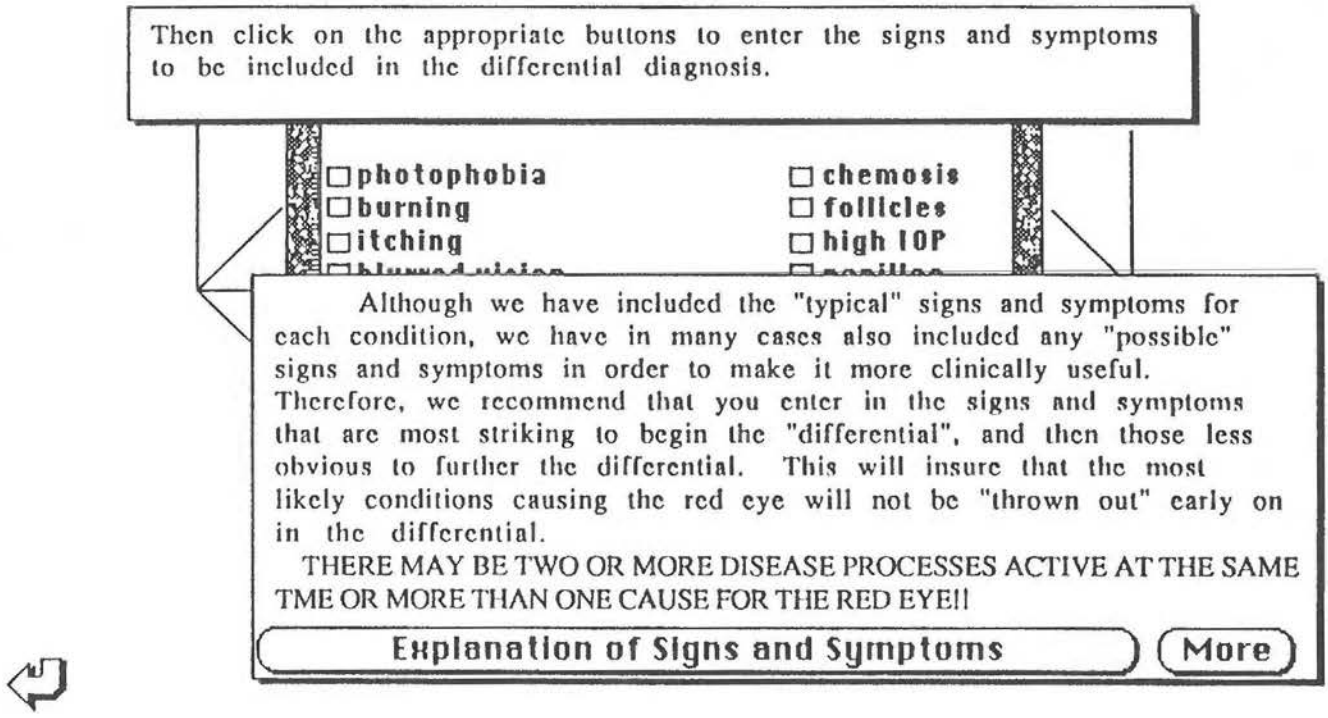
⟵ Gives additional information.

⟵ Takes you to "List of Conditions" covered in this program.

⟵ Takes you back to the previous screen.

Return

Below is an example of what the "Differential Diagnosis" card looks like. To begin, you must decide if pain is to be included in the differential, and if so to what degree. "Click" one of the buttons below.



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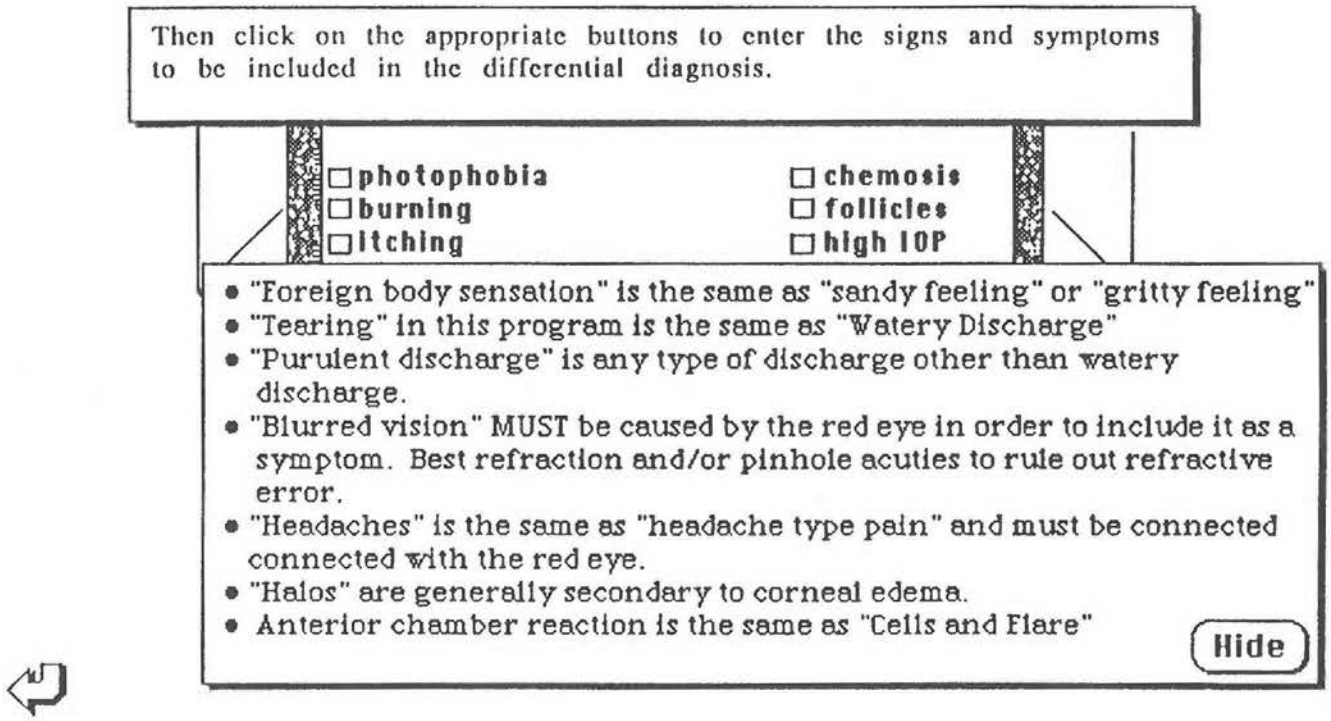
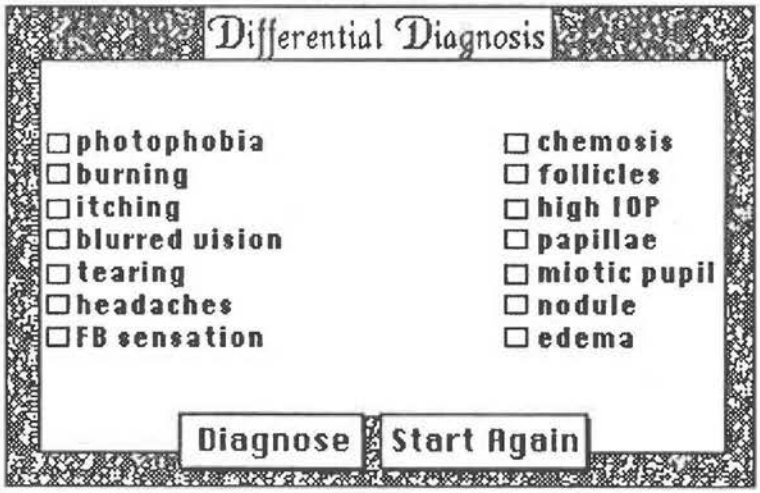




The "Diagnose" and the "Start Again" buttons are two important buttons on this card. Click each one for more specific information.

More

There are two times when you would use this button:

- Obviously if you wanted to start over with a new set of signs or symptoms, this button will erase all of the previously checked signs and symptoms.

- If you make a mistake in entering a sign or symptom, you may click that sign or symptom to "erase" it only if it was the last one that you "clicked". For example, if you have checked tearing, photophobia and blurred vision in that order, you could "click" blurred vision again so that it would not be considered in the differential, but you could not "click" photophobia and erase it since it was not the last one that you that you checked. In that case you must start again.

Hide

Diagnose | Start Again

The "Diagnose" and the "Start Again" buttons are two important buttons on this card. Click each one for more specific information.

More

Differential Diagnosis

The "Diagnose" button allows you to view the conditions under consideration or the "List of Possibilities". If you wish to return to the list of signs and symptoms to further the differential, there will be a button to allow you to go back and enter in more signs or symptoms.

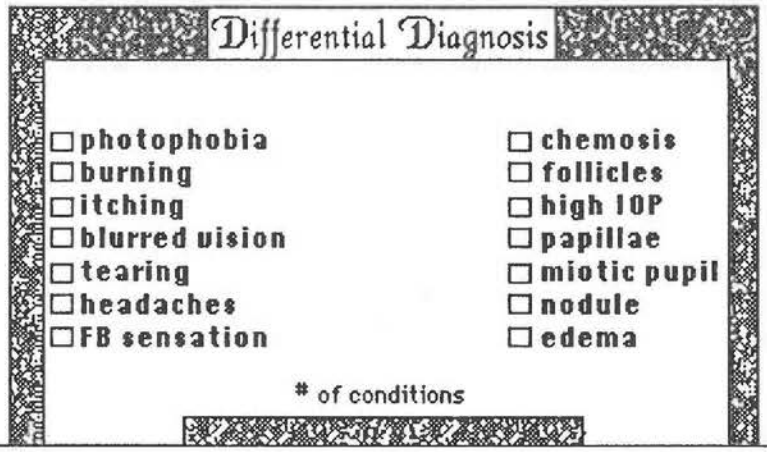
Hide

☐FB sensation ☐edema

Diagnose | Start Again

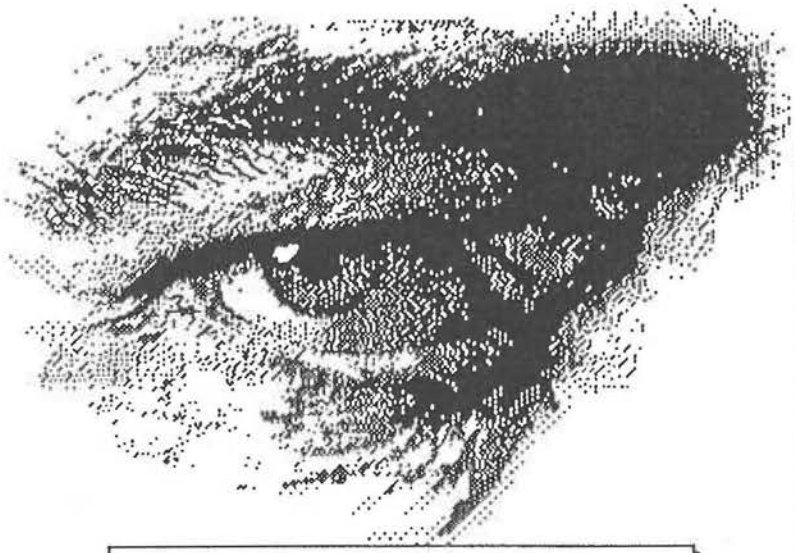
The "Diagnose" and the "Start Again" buttons are two important buttons on this card. Click each one for more specific information.

More



This shows how many conditions present with the signs and symptoms checked above. It is recommended to "Diagnose" when this number is less than five to allow you to see several of the possible causes of the red eye. You may always return to enter another sign or symptom to make the list smaller.

End



Cornea | Trauma

Conjunctiva/Uveal Tract

Episclera/Sclera/Lids/Orbit

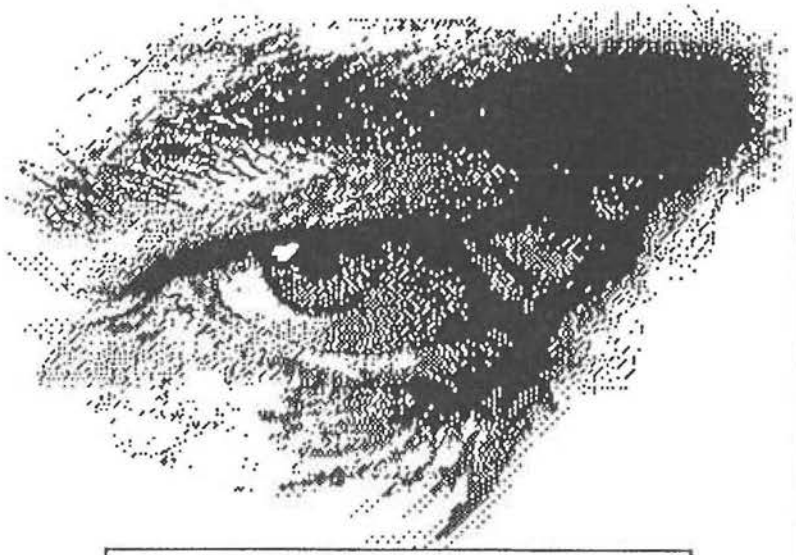
General Red Eye Work up

Conditions Covered in This Program

Trauma:
Click a topic below.

Chemical Burn
Conjunctival Foreign Body
Corneal Abrasion
Corneal Foreign Body
Subconjunctival Hemorrhage
Thermal/UV Keratopathy
Traumatic Iritis

To Main Menu



Cornea | Trauma

Conjunctiva/Uveal Tract

Episclera/Sclera/Lids/Orbit

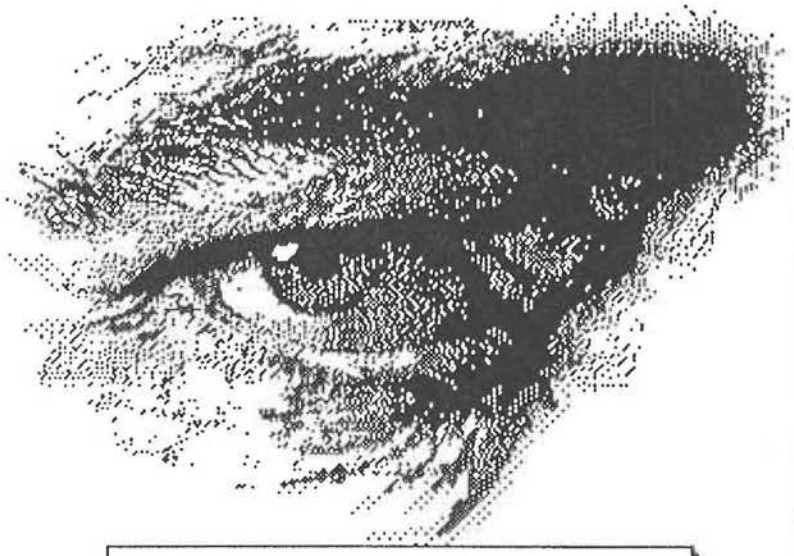
General Red Eye Work up

Conditions Covered in This Program

Cornea:
Click a topic below.

Acute Interstitial Keratitis
Contact Lens Related
Corneal Abrasion
Infectious Corneal Ulcer
Dry Eye
Exposure Keratopathy
Filamentary Keratopathy
Herpes Simplex Keratitis
Herpes Zoster Ophthalmicus
Phlyctenulosis
Pterygium
Recurrent Corneal Erosion
Sup. Punctate Keratopathy
S. L. Keratoconjunctivitis
Trachoma

To Main Menu



Cornea | Trauma

Conjunctiva/Uveal Tract

Episclera/Sclera/Lids/Orbit

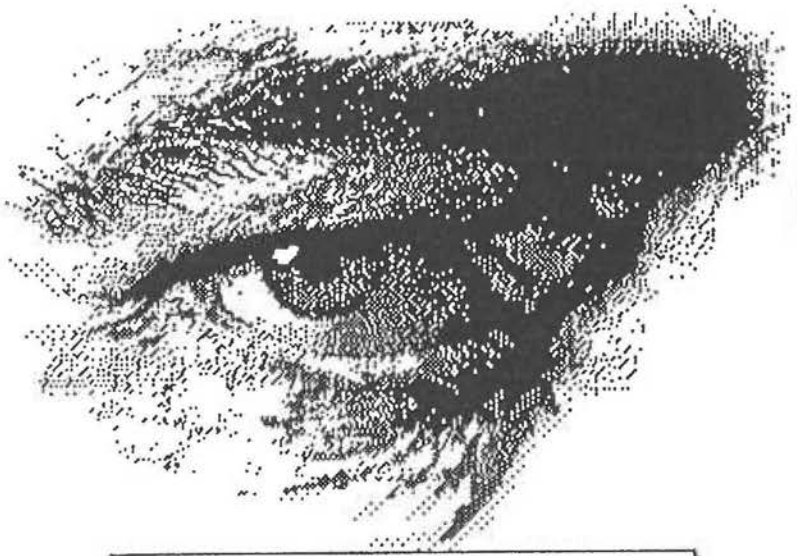
General Red Eye Work up

Conditions Covered in This Program

Episclera/Sclera/Lids/Orbit: Click a topic below.

Blepharitis
Canaliculitis
Contact Dermatitis
Entropian
Episcleritis
Ocular Rosacea
Orbital Cellulitis
Scleritis
Trichiasis

To Main Menu



Cornea | Trauma

Conjunctiva/Uveal Tract

Episclera/Sclera/Lids/Orbit

General Red Eye Work up

Conditions Covered in This Program

Conjunctiva/Uveal Tract: Click a topic below.

Acute Angle Closure Glaucoma
Allergic Conjunctivitis
Anterior Uveitis
Bacterial Conjunctivitis
Chlamydial Conjunctivitis
Epidemic Keratoconjunctivitis
Floppy Eyelid Syndrome
GPC
Hyperacute Conjunctivitis
Ocular Pemphigoid
Parinaud's Conjunctivitis
Stevens-Johnson
Vernal Conjunctivitis
Viral Conjunctivitis

To Main Menu

General Work Up for a Red Eye

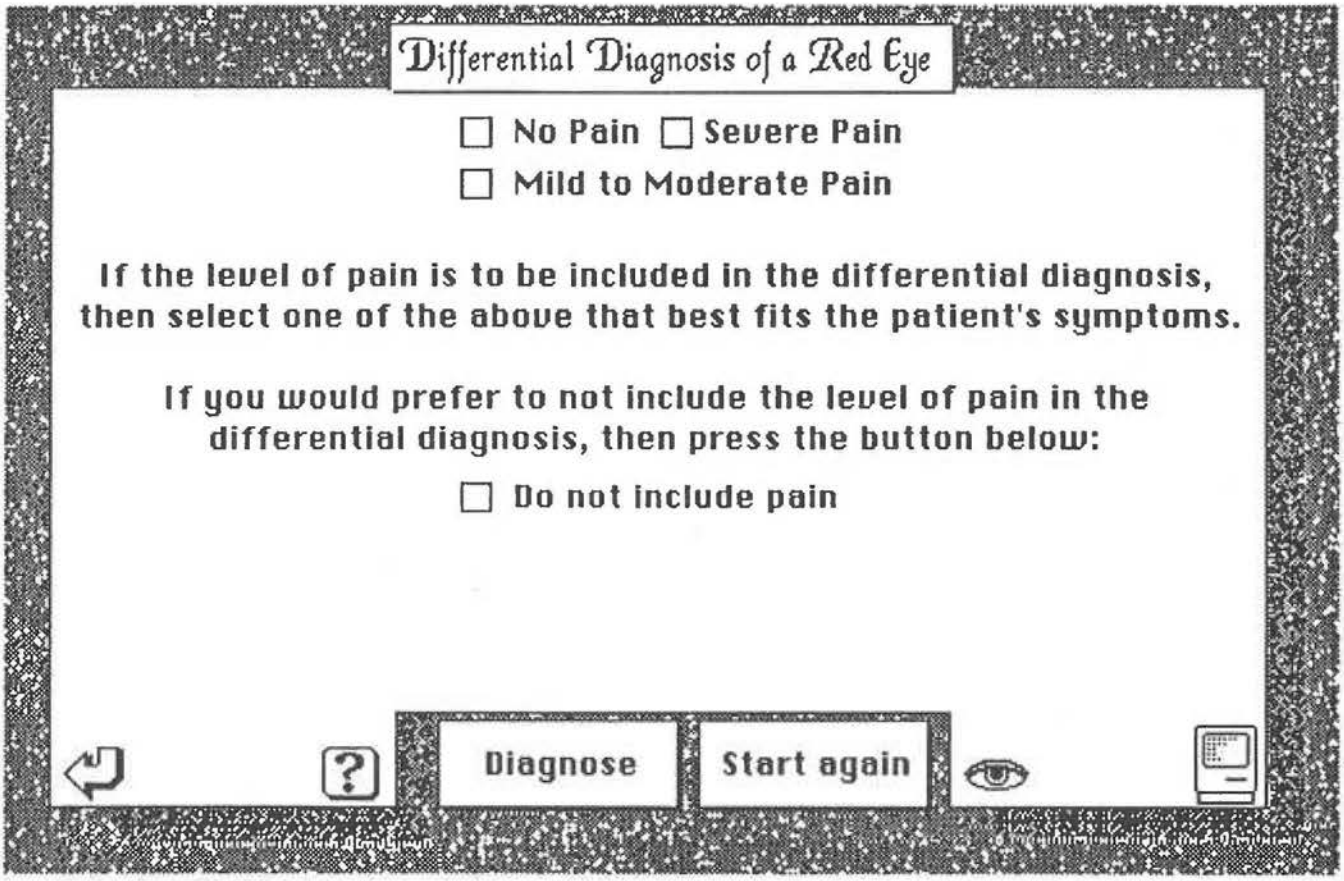
CAREFUL CASE HISTORY:

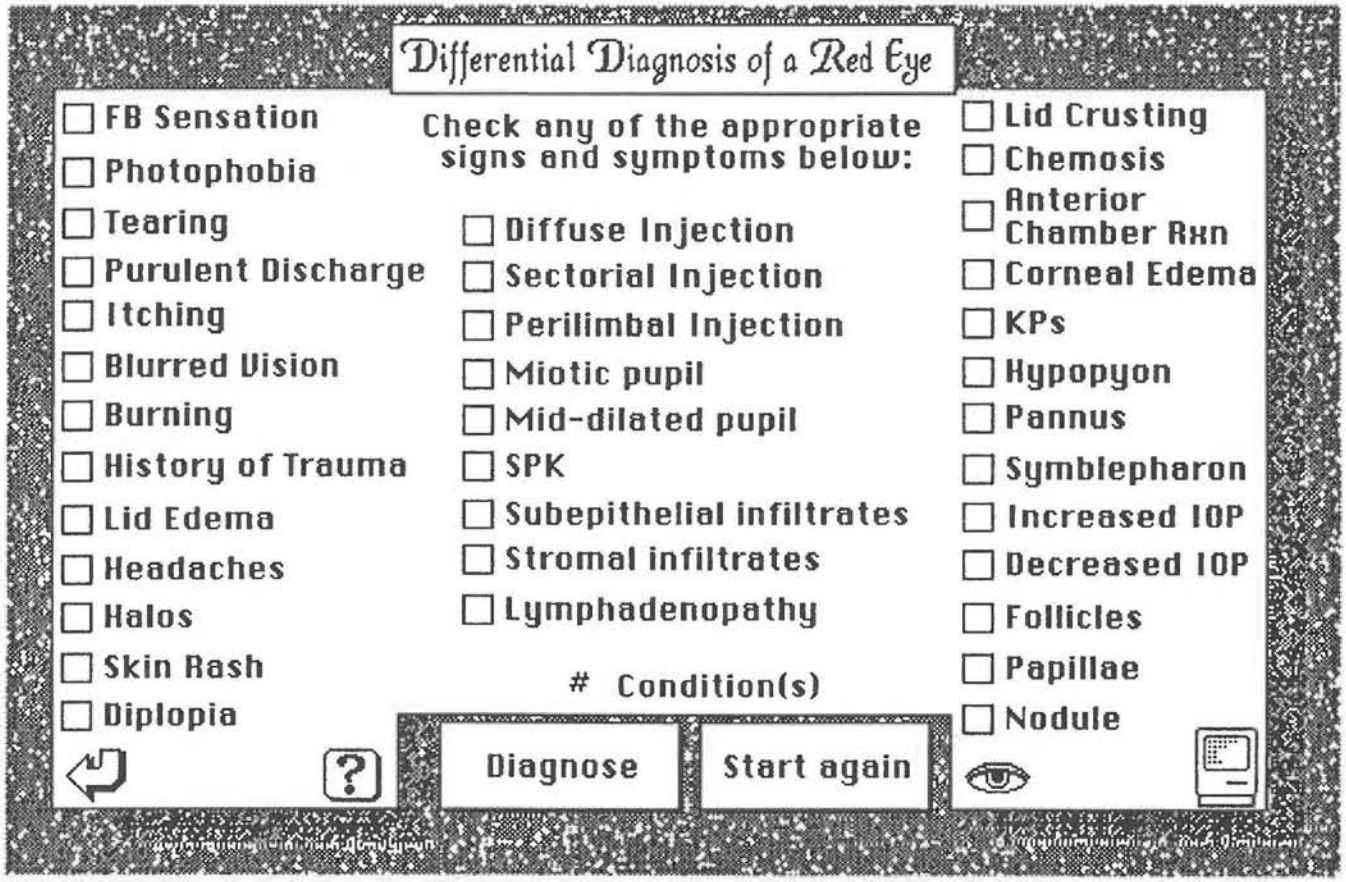
- Statement of chief complaint. Is it vague or specific? Vague is usually not as serious.
- How does it feel? Explore the following:
  - -- Pain (slight/moderate/severe/dull/discomfort), discharge (type and amount) itching, burning, FB sensation, photophobia.
  - -- Time frame (onset, frequency, duration, getting better or worse?)
  - -- Vision affected? Diplopia?
- Trauma? (high speed object/ blunt injury/chemical injury)

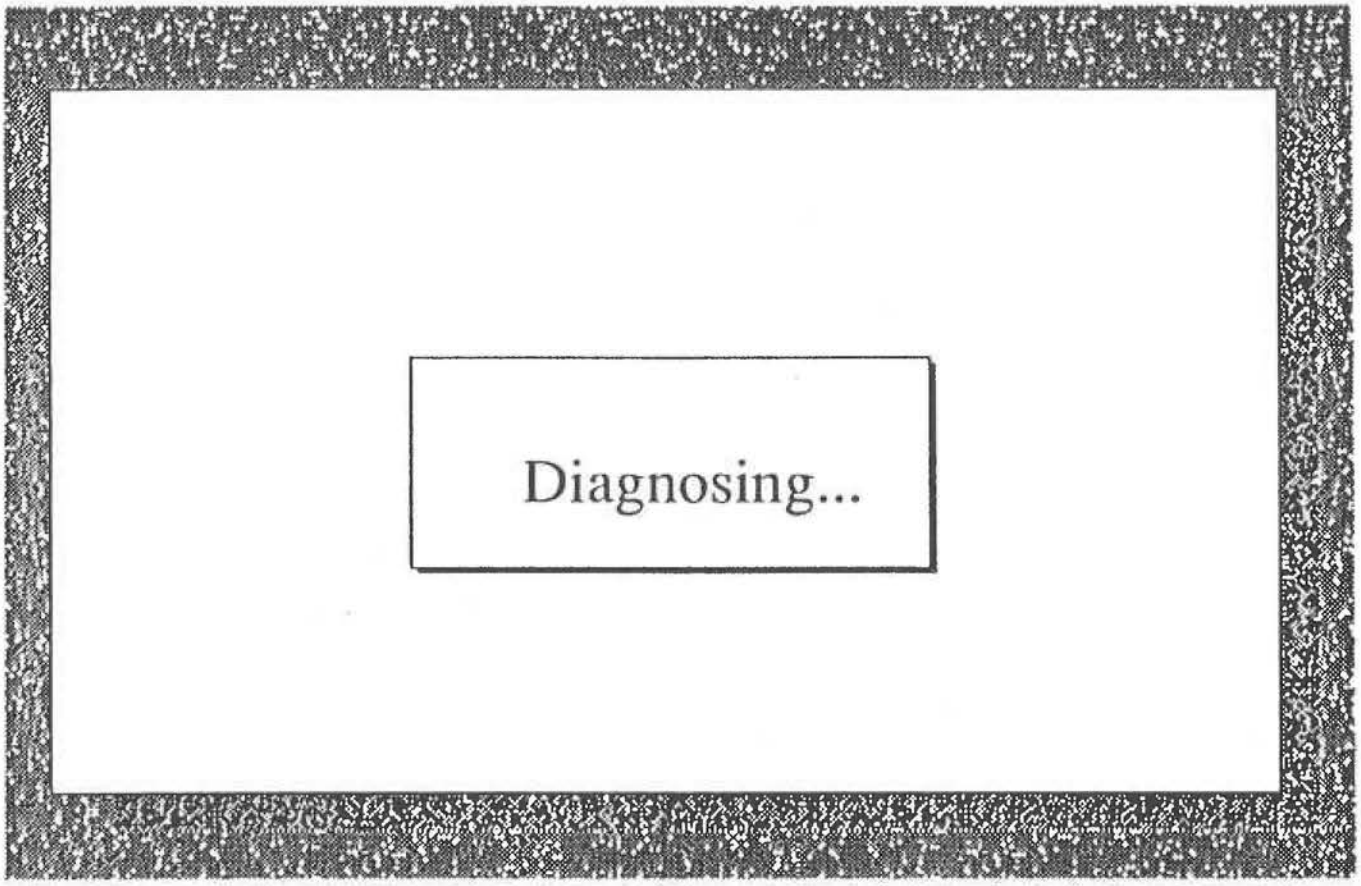
- Patient's and Family's systemic health and ocular health (previous episodes)
- Patient's medications/ allergies (both medical and seasonal)

OBJECTIVE: (Wash hands before and after examining the patient. Do not inocculate yourself or the fellow eye --> use cotton swabs and two flourescein strips if red eye unilateral)

- Visual acuity (if < 20/20 use pinhole) Use topical anesthetic if needed. Must get VA s!!
- External exam with penlight (symmetry, edema, hyperemia, palpate for tenderness)
- Pupils, Versions, Confrontation fields, Preauricular lymph node palpation
- SLE: Lashes/Lids/Conj (papillae, follicles, membrane, evert lid if necessary, chemosis, hyperemia, hemorrhagic), Sclera/Episclera/Cornea (edema, infiltrates, KP's, Anterior Chamber (cells/flare/hyphema/hypopyon/angle), Iris, Lens
- IOP (unless corneal insult , infection, or hyphema)
- Fundus evaluation (DFE indicated in trauma)


Differential Diagnosis of a Red Eye
No Pain
Severe Pain
Mild to Moderate Pain
If the level of pain is to be included in the differential diagnosis, then select one of the above that best fits the patient's symptoms.
If you would prefer to not include the level of pain in the differential diagnosis, then press the button below:
Do not include pain
Diagnose
Start again


Differential Diagnosis of a Red Eye
Check any of the appropriate signs and symptoms below:
FB Sensation
Photophobia
Tearing
Purulent Discharge
Itching
Blurred Vision
Burning
History of Trauma
Lid Edema
Headaches
Halos
Skin Rash
Diplopia
Diffuse Injection
Sectorial Injection
Perilimbal Injection
Miotic pupil
Mid-dilated pupil
SPK
Subepithelial infiltrates
Stromal infiltrates
Lymphadenopathy
Condition(s)
Lid Crusting
Chemosis
Anterior Chamber Rxn
Corneal Edema
KPs
Hypopyon
Pannus
Symblepharon
Increased IOP
Decreased IOP
Follicles
Papillae
Nodule
Diagnose
Start again


Diagnosing...

List of Possibilities

"Click" on a condition to see it's critical sign for a positive diagnosis.

Back to List of Signs and Symptoms

List of Possibilities

"Click" on a condition to see it's critical sign for a positive diagnosis.

Infectious Corneal Ulcer
Recurrent Corneal Erosion
Herpes Simplex Keratitis
S. L. Keratoconjunctivitis
Blepharitis
Viral Conjunctivitis
Vernal Conjunctivitis
Ocular Rosacea
Dry Eye
Epidemic Keratoconjunctivitis

^

Back to List of Signs and Symptoms

Acute Angle Closure Glaucoma

Main Menu

- General Information
- Diagnosis
- DDx
- Work Up
- Treatment
- Follow-up

General Information: click a topic

- Incidence/Etiology
- Predicting potential cases
- Classification & Naming

** A True Ocular Emergency **

Incidence and Etiology of Acute Angle Closure Glaucoma:

- Incidence:
  Depending on the source, incidence can range from 0.06% of the general population to .17% in caucasians more than 40 years old.
- Etiology:
  Results from a decreased aq. humor outflow due to a restricted anterior chamber angle. Most often, the eye has an anatomical predisposition of a shallow anterior chamber, and iris tissue is responsible for the restriction.
- Common causes include: pupillary block (more common in hyperopes), angle crowding (plateau iris), neovascularization, and mechanical closure of angle secondary to anterior displacement of the lens/iris diaphragm.

← Menu Predicting potential cases →

Predicting Potential Cases of PACG:

RISK FACTORS:

a. Narrow angles
   (5% of the population)
   (.65% have angles that are critically narrow)
b. Hyperopia
c. Females > males
d. Whites > blacks
e. Age (mid 40s)
f. Family history

- Typically, an older person with a small globe and a significant narrow anterior chamber angle.

← Incidence and etiology Types of Angle Closure →

Types of Angle Closure Glaucoma:

- Acute (most common):
  Increased IOP, closed angle, mid dilated pupil, corneal edema, perilimbal injection, blurred vision, halos, nausea, aqueous flare, pain of trigeminal distribution (may c/o tooth ache), optic nerve and visual field changes.
- Sub-acute:
  Sector iris atrophy
  Glaukomflecken-->tiny opacities in lens due to increased pressure (50-60 mmHg).
  Pigment dispersion
  Disc and field changes
- Chronic:
  Gradual occlusion begins superiorly
  40-60 mmHg IOP without symptoms

← Predicting potential cases Critical Signs →

To Main Menu For Acute Angle Closure Glaucoma →

Critical Signs for Acute Angle Closure Glaucoma:

- Oval mid-dilated pupil (unreactive to light)
- Closed angle in eye involved (gonioscopy)
- Acutely elevated IOP
- Corneal microcystic edema

← Types of Angle Closure Glaucoma Signs →

Signs of Acute Angle Closure Glaucoma:

- Sudden increase in IOP (60-90 mm Hg)
  -- IOP may be low if the patient is in recovery.
- Dilation of the vessels at the limbus (perilimbal flush)
- "Steamy" cornea (edema)
- Oval mid dilated pupil (4-5mm) that is unreactive to light
  -- most reliable sign
- Cells and flare may be present
  -- If the IOP has been elevated for a prolonged period of time during a previous attack--> gray atrophy of the iris stroma and glaukomflecken.

← Critical Signs Symptoms →

Symptoms of Acute Angle Closure Glaucoma:

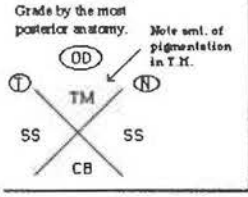
- Significant ocular pain and discomfort
  -- Pain may be moderate, but often is severe
  -- Many times, the pain can involve the whole head which leads the patient to report a brow ache or headache.
- Nausea and often vomiting
- Rapid loss of vision (blurred vision)
- Colored rings around point sources of light (edema of cornea)
- Frontal headaches
- Often there is a history of mild attacks associated with patient fatigue or following a period of sudden emotional stress.

* All symptoms above need not be present *

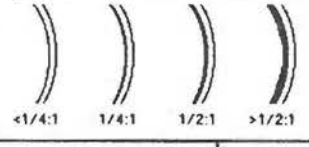
← Signs Gonioscopy →

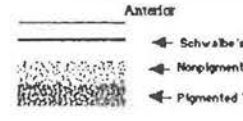
Gonioscopy

Grade by the most posterior anatomy.



Van Herrick: Angles that are estimated to be a quarter of the corneal thickness or less require gonioscopy to determine if subsequent dilation will precipitate angle closure and increase IOP.





Moderately narrow, closure possible. I - C = 20°

- Grade IV angle
- Grade III angle
- Grade II angle
- Grade I angle
- Grade 0 angle

Menu →
Symptoms →
Differential Diagnosis →

Differential Diagnosis

Rule out other types of glaucoma:

- Inflammatory open angle glaucoma (moderate to severe anterior chamber reaction)
- Traumatic (hemolytic) glaucoma (Hx of trauma + RBCs in anterior chamber)
- Pigmentary glaucoma: (angle is open, radial iris transillumination, pigment cells in anterior chamber and on trabecular meshwork)
- Phacolytic glaucoma: (cataract, anterior chamber particles or white flaky material present on pupillary border and anterior lens capsule)
- Combined open and closed angle glaucoma

Also:
- Glaucomatocyclitic crisis: mild cells and flare, fine KPs, open angle, eye not painful, recurrent IOP spikes in one eye usually 40-60 mm Hg

Gonioscopy | Work up

Work-Up for Acute Angle Closure Glaucoma

- Hx: Onset? Medications? Any previous or present symptoms in fellow eye? Recent laser treatment or surgery? Detailed account of activities that preceeded onset of symptoms.
- Visual acuity. Pinhole if < 20/20
- SLE: Examine both eyes especially anterior chamber depth. Look for KPs, posterior synechiae, iris neovascularization, swollen lens, anterior chamber cells/flare.
- IOP
- Gonioscopy of both angles. May want to avoid if diagnosis is firmly established or if there is a great deal of corneal edema.
- Evaluate optic nerve.

Differential Diagnosis | Emergency Treatment

Emergency Treatment of Acute Angle Closure Glaucoma:

Acute angle closure is a true emergency and reqires immediate care! Long term management is surgical.

- Initial Tx to lower the IOP
  - Hyperosmotic (may induce vomiting) (Contraindications)
    -- Glycerol (1 gram/kg of body wt.)--› can cause a problem in diabetics
    -- Isosorbide and Manitol (IV) are also used
  - Anderson's indentation procedure can be an alternative to the oral hyperosmotic. (More on indentation procedure)
  - Beta Blocker (Contraindications)
    -- Timolol 0.5% or Levobunolol 0.5%
  - Carbonic Anhydrase Inhibitors (Contraindications)
    -- Acetazolamide (Diamox)
    -- Methazolamide (Neptazane)

Work up | Specific Tx

Emergency Treatment of Acute Angle Closure Glaucoma:

The following has been effective in treating acute angle closure.
1. Oral hyperosmotic (Osmoglyn 1.0-1.5 mg/kg of body weight) max effect 45-120 min
2. Topical beta blocker (Timoptic 0.5% 1 drop)
3. 500 mg of Diamox (Acetazolamide)

- Wait one hour and reassess with gonioscopy to determine if the angle is open. If IOP is less than 50 mm Hg, use 1 drop of 2% Pilocarpine.
- When pressure is controlled and attack is broken:
  1% Pred acetate qid
  Azetazolamide 500 mg sequel po bid
  Topical beta blocker bid
  Pilocarpine ??
  ** Refer for definitive treatment**
  (When to use Pilocarpine)
- There is a high probability that angle closure may develop in the fellow eye. Treat fellow eye with 0.5% pilocarpine qid if angle narrow until definitive treatment.
- Definitive tx includes: laser/surgical iridectomy or trabeculectomy.

Treatment | Follow up

Follow-Up of Acute Angle Closure Glaucoma:

- After definitive treatment, patients are reevaluated in weeks to months.
- Visual fields and stereo disc photographs are obtained for baseline purposes.
- If a repeat attack occurs after the patient has had the iridotomy, a plateau iris may be present.

Treatment | Menu

Acute Interstitial Keratitis

Main Menu

- General Information
- Signs & Symptoms
- Work-Up
- Treatment
- Follow Up

General Information: Click a topic

- Definition and Incidence
- Etiology

Definition and Incidence of Acute Interstitial Keratitis (IK)

- The term interstitial keratitis refers to the vascularization and nonsuppurative infiltration affecting all or just part of the corneal stroma. Most often, IK is associated with a systemic disease. 90% of cases are secondary to syphilis.
- The stromal opacification is generally silvery looking and often has a patchy or feathery appearance.
- Manifestations of keratitis usually are not apparent until the age of 10 if the cause is congenital syphilis, with the greatest frequency occuring between the ages of 10 and 20.
- Over the years, there have been a reduction in the number of cases of congenital syphilis, and encountering a case of acute interstitial keratits is rare. The most usual presentations of IK are congenital cases encountered during routine exams of adults. Signs of old IK often persist throughout life.
- There is a 3:1 predilection for females.

← Menu Etiology →

Common Etiologies of Interstitial Keratitis

- Congenital Syphilis:
  - In the past, has been responsible for 90% of the cases of diffuse IK, most commonly of the congenital form. The congenital form is usually bilateral (80%).
- Acquired Syphilis:
  - IK secondary to acquired syphilis is commonly uniocular (60%), sectorial, and of a milder form.
  - A positive FTA-ABS test confirms the presence of a previous infection.
- Tuberculosis:
  - IK is often unilateral, involving the peripheral inferior sector of the cornea with central cornea spared. Resolution is less rapid and less complete than that due to syphilis.
- Leprosy:
  - IK is usually a deep infiltration extending from the periphery to the center of the cornea, especially in the upper outer quadrant. Frequently bilateral.
- Cogan's syndrome, herpes simplex, onchocerciasis, mumps, and gold toxicity are among the other systemic conditions known to cause IK.

← Definition and Incidence Critical Signs →

Main Menu for HSV Keratitis →

Critical Signs for Acute Interstitial Keratitis

- Corneal stromal blood vessels and edema.

← Etiology Signs →

Other signs of Acute Interstitial Keratitis

- Anterior chamber cells and flare
- Fine KPs on corneal endothelium
- Indistinct cellular infiltration
- Miosis
- Small stromal opacities
- Conjunctival injection
- The gross vascularization can give the cornea a "salmon patch" appearance

← Critical signs Symptoms →

Symptoms of Acute Interstitial Keratitis

- Pain
- Tearing
- Photophobia
- Red eye
- Blurred vision

← Signs Work up →

Work Up for Acute Interstitial Keratitis

- History: Venereal disease in the mother during pregnancy or in patient. Difficulty hearing or tinnitus?
- External examination: Look for signs of congenital syphilis or leprosy.

  Signs of Syphilis Signs of Leprosy
- Slit lamp examination: Look for signs of congenital syphilis or leprosy.

  Signs of Syphilis Signs of Leprosy
- Dilated fundus examination.
  - Look for the classic "salt and pepper" chorioretinitis or optic atrophy of syphilis. These are signs of an active syphilitic disease

← Symptoms Treatment →

Treatment for Interstitial Keratitis

- Acute disease or old inactive disease
  - Topical cycloplegic tid
  - Topical steroid
    - Pred Forte q 1-6 hours depending on degree of inflammation.
  - Refer to medical internist for FTA-ABS and PPD
    - Underlying cause needs to be treated.

← Work up Follow up →

Follow-Up for Interstitial Keratitis

- Acute disease:
  - Every 3-7 days at first, then see every 2-4 wks.
  - Frequency of steroid administration is slowly reduced as the inflammation subsides.
  - IOP should be monitored closely and lowered with medication when > 30 mmHg.
- Old inactive disease:
  - Routine follow-up every year unless treatment is required for an underlying etiology.

Treatment

Menu

Allergic Conjunctivitis

Main Menu

- Etiology
- Symptoms
- Signs
- Critical Signs
- Differential Diagnosis
- Treatment
- Follow Up

Etiology of Allergic Conjunctivitis

- Immunological responses, known as hypersensitivity reactions, to antigens of exogenous agents can be important causes of conjunctival inflammation. Most hypersensitivity reactions are classified clinically into 2 types based on the time between exposure to the antigen and the appearance of the immunological response:

  (Immediate (Type I) - mediated by serum immunoglobulins (antibodies) which produce an eosinophilic cellular response. This response develops within minutes after exposure to the offending antigen and is characterized by conjunctival hyperemia and chemosis secondary to vascular dilation and serous exudate. An example of Type I is hayfever, with itching being the prominant symptom.

  (Delayed (Type II) - mediated by lymphocytes producing a mononuclear cellular response. This response develops within hours or days after introduction of the antigen and is characterized by the same general signs of chemosis and hyperemia. In many cases, a mild follicular reaction may also be present.

- Allergic conjunctivitis is common in persons aged 20-30 years with a history of atopic conditions, such as hayfever.

Main Menu | Symptoms

Symptoms of Allergic Conjunctivitis

- Itching is the predominant symptom
- Tearing
- Watery discharge
- Photophobia

Etiology | Signs

Signs of Allergic Conjunctivitis

- The vision may fluctuate or remain unaffected
- Pink to red bulbar hyperemic injection
- A "glassy" appearing lustre to the mucous membranes
- Chemosis (subconjunctival infiltration and edema)
  - -- produces mild to dramatic swelling of the bulbar conjunctiva. The conjunctival tissue may elevate and roll over the limbus. This is more common and predominant on the temporal conjunctiva due to the eye rubbing pattern at the outer canthus
- Mucoid (stringy) whitish discharge that may spread across the cornea and ultimately end up in the fornices at the inner canthi
- Lid edema and erythema
- Small "velvety" to "giant" papillary changes on both the upper and lower palpebral conjunctiva
- Frequently, follicular changes are also present

Symptoms | Critical Signs

Main Menu for Allergic Conjunctivitis

Critical Signs of Allergic Conjunctivitis

- Chemosis
- Red edematous eyelids
- Conjunctival papillae
- No preauricular adenopathy

Signs | DDH

Differential Diagnosis of Allergic Conjunctivitis

1. Bacterial conjunctivitis - generally presents as a "meaty" red conjunctivitis that increases toward the fornices. The cornea is typically clear; the hyperemic vessels will blanch with a mild vasoconstrictor; usually a papillary response and a mucopurulent discharge.
2. Viral conjunctivitis - generally presents as a pinkish-purple hyperemia that increases toward the plica. There is typically a fast TBUT, a follicular response, a serous discharge, and enlargement of preauricular node or lymphadenopathy.
3. Chronic conjunctivitis - generally presents as angular injection with inferior hyperemia; a papillary response and no discharge present.
4. Vernal conjuncitivitis - generally presents as a seasonal, recurrent allergy with the presence of cobblestone papillae on the tarsal conjunctiva.

Critical Signs | Treatment

Treatment of Allergic Conjunctivitis

- The general approach to the management to allergic conjunctivitis includes desensitation, pharmacologic agents, and an avoidance to the particular offending antigens.
- Desensitation seems to be of value when allergic conjunctivitis is asociated with pollen, dust, or other airborne allergens.
- Pharmacologic agents used in the treatment of allergic conjunctivitis include the use of topical vasoconstrictors, antihistamines, corticosteroids, and cromolyn sodium.
  - Vasoconstrictors - [e.g., naphazoline (Vasocon A), phenylephrine, or oxymetazoline] (dosage) --> 1 gt. 4 - 6 X daily
    NOTE: long term use of agents with phenylephrine may cause a rebound hyperemic effect that may present a greater problem to the patient.
  - Antihistamines - [e.g., antazoline 0.5% in conjunction with Vasocon A 0.05%] (dosage) --> 1 gt. q 3-4 hours or every 2-3 hours for more severe cases.
  - Corticosteroids - [e.g., prednisolone acetate (Pred forte) 1.0%] (dosage) --> 1 gt. q 2 hours for 3-5 days (Prolonged used of steroids is not recommended because they may lead to ocular infection or glaucoma.)
  - Cromolyn Sodium [e.g., Opticrom 2%/4%] (dosage) --> 1 gt. q 4-6 hours. Use Opticrom 4% in more severe cases.
- Avoiding the specific allergens seems to be quite successful in cases of allergic conjunctivitis which present secondary to ophthalmic preparations and/or cosmetics.

DDH | Follow Up

Follow up for Allergic Conjunctivitis

- In moderate to severe reactions, schedule to patient for a 24 - 48 hour checkup.
- Reschedule beyond the second visit only for those cases that are responding slowly.
- Patients who are on steroid management should be checked every 1 to 2 weeks until the condition resolves. Remember that the steroidal dosage should be tapered accordingly.

Treatment | Main Menu

Acute Anterior Uveitis

Main Menu

- General Information
- Signs & Symptoms
- Etiologic Diagnosis
- DDx of Uveitis
- Work-Up
- Treatment
- Follow-Up

General Information: Click a topic

- Definition and Incidence
- Classification & Naming
- Grading cells

Definition and Incidence of Uveitis

DEFINITION:

- Uveitis is a general term which can be subdivided into iritis, cyclitis, iridocyclitis, and choroiditis. Adjacent areas such as retina, vitreous, sclera and cornea are frequently involved.
- Uveitis is an inflammation of the iris, ciliary body or choroid.
- Cases that are bilateral, recurrent or refractory to treatment need a more extensive diagnostic evaluation --> endogenous origin. Most common ones are listed under etiological diagnosis in the main menu.
  - 50% associated with HLA-B27
- Can be limited to anterior or posterior chamber or can affect both.

INCIDENCE:

- Peak incidence is in the 20-50 yr population.
- There is a marked decrease in incidence in people over the age of 70.
- One study reports incidence at about 12 cases /100,000 anterior uveitis, and 3 cases /100,000 posterior uveitis

Menu | Differentiating Signs

Differentiating signs of Anterior Uveitis

Granulomatous (Most likely chronic)	vs	Nongranulomatous (Most likely acute onset)
1. Large "mutton-fat" KPs		1. Fine KPs
2. Koeppe nodules (frequently)		2. AC cell reaction
3. Busacca nodules (lesions on iris)		3. Usually no nodules
4. Posterior synechiae (fresh)		4. Dense synechiae (fibrous)
5. Generally involve posterior chamber, but can involve anterior chamber.		5. Generally anterior chamber

Definition and Incidence | Common Etiologies

Common Etiologies of Uveitis

- The most common cause of uveitis is unknown for all age groups.
- External causes include: trauma, ocular infection and allergic reactions.
- Internal causes include: systemic diseases and immunologic factors.

More Specifics

Differentiating Signs | Grading Cells/Flare

Etiology	Segment affected:
Ankylosing spondylitis	Anterior only
Bechet's disease	Anterior
Histoplasmosis	Posterior only
Inflammatory bowel	Anterior
Juvenile rheumatoid arthritis	Anterior
Rheumatoid conditions	Anterior
Reiter's syndrome	Anterior
Sarcoidosis	Anterior, Posterior or both
Syphilis	Both segments or posterior only
Toxoplasmosis	Both segments or posterior only
Toxocara	Posterior only
Tuberculosis	Both segments or anterior only

Lab Tests | Consultation | Segment affected | Description | Menu

Other Etiologies to Consider

- Pars planitis
- Fuch's heterochromic iridocyclitis
- Glaucomatocyclitic crisis
- Herpes Simplex/Herpes Zoster
- Rubeola
- Leprosy
- Vogt-Koyanagi-Harada disease
- Sympathetic ophthalmitis

Hide

Grading Cells and Flare

	Trace	1+	2+	3+	4+
CELLS	1-5	5-10	10-15	To many to count	Dense cells
FLARE	none	Just detectable faint haze	Mod. haze Clear iris detail	Marked haze Fuzzy iris	Plastic iritis fibrin clot

COMPLICATIONS INCLUDE:

- Band keratopathy
- Cataracts (especially posterior subcapsular)
- Disc/Macular edema
- Corneal edema (depends on damage to endothelium and height of IOP)
- Secondary glaucoma (debris in meshwork, rubeosis, iris bombe, trabecular sclerosis and trabeculitis)
- Retinal detachment
- Macular surface wrinkling

Common Etiologies | Critical Signs

Main menu for Anterior Uveitis

Critical Sign for Anterior Uveitis

- Cells and flare in the anterior chamber

Grading Cells and Flare | Symptoms

Symptoms of Anterior Uveitis

Symptoms may range from none to severe and are not related to the severity of the uveitis.

- Acute onset of deep ocular pain (dull or pulsating)
  - many times reported as "in or behind" the eyeball
  - increased discomfort with near vision often reported
- Photophobia (a constant symptom and may be the first one)
  - may be mild to severe
- Vision may be normal or decreased (due to cells and flare)
  - may be reported as a "haziness"
- Red eye
- Variable tearing but NO other form of discharge
  - an associated disease entity is probably present if there is a mucoid or purulent discharge.

Critical Signs | Differentiating Signs

Differentiating Signs of Anterior Uveitis

Granulomatous (most likely chronic)	vs	Nongranulomatous (most likely acute onset)
1. Large "mutton-fat" KPs		1. Fine KPs
2. Koeppe nodules (freq.)		2. AC cell reaction
3. Busacca nodules (lesions on iris)		3. Usually no nodules
4. Post. synechiae (fresh)		4. Dense synechiae (fibrous)
5. Generally involving posterior chamber, but can involve ant. chamber.		5. Generally ant. chamber

Symptoms | Signs

Signs of Acute Anterior Uveitis

- Generally unilateral
- Cells & flare in anterior chamber
  - cells --> lymphocytes (white dots)
  - flare --> protein (milkiness or haziness)
- Lid congestion
- Pupil may be miotic
- Circumcorneal hyperemia (perilimbal flush)
- Fine KPs on corneal endothelium
- Lower IOP (occasionally may be elevated)
- Posterior synechiae
- Cells in anterior vitreous may be present
- Pseudoproptosis (photophobic)
- Hypopyon if severe case of anterior uveitis

Signs of Chronic Anterior Uveitis

- Cells & flare in anterior chamber
- Lids not involved
- Occasional fixed pupil
- Circumcorneal hyperemia
- Mutton fat KPs
- Frequent secondary glaucoma
- Dense synechiae
- Iris nodules and granulomas
- Frequent vitreoretinal involvement
- Cystoid macular edema

Differentiating Signs | Differential Diagnosis

Differential Diagnosis of Anterior Uveitis

- Conjunctivitis: See main menu for the different types.
  - Vision is usually not affected and hyperemia is generally confined to medial or lateral angles or is diffuse. There is generally serous or purulent discharge. Pupillary response is normal as well as IOP. No significant photophobia or deep pain.
- Angle Closure Glaucoma: See main menu.
  - There is a marked decrease in vision as well as severe pain, corneal edema, and increased IOP. The pupil is generally mid-dilated and nonreactive.
- Rhegmatogenous Retinal Detachment:
  - Elevated retina with a break. This can release pigment cells into the vitreous or anterior chamber.

Other Less Common Conditions to Consider

Signs | Work up

Work-Up of Anterior Uveitis

- An important part of the work up is to consider the risk factors:
  - Age is important (increases with age).
  - Race: African Americans have 10X the risk for sarcoidosis
  - Sex : This further narrows the possibilities with regard to specific etiologies.
  - Sexual history, personal history, ocular history and systemic diseases are all important considerations.
- Attempt to define the etiology especially if chronic, bilateral or granulomatous. If the history, symptoms, and/or signs strongly suggest an underlying etiology, then the work-up should be tailored accordingly.
- Get a good case Hx.
- Visual acuity (pinhole if < 20/20)
- SLE (conical beam to view cells/flare in anterior chamber) Grading cells/flare
- Check IOP.
- Dilated fundus examination along with vitreous examination (look for cells).

Differential Diagnosis | Treatment

Treatment of Anterior Uveitis

Objectives of treatment:
- To decrease the severity and frequency of the attacks
- To prevent posterior synechiae and development of secondary cataracts
- To prevent damage to iris blood vessels and blood - aqueous barrier

Treatment is usually nonspecific due to unknown etiology.

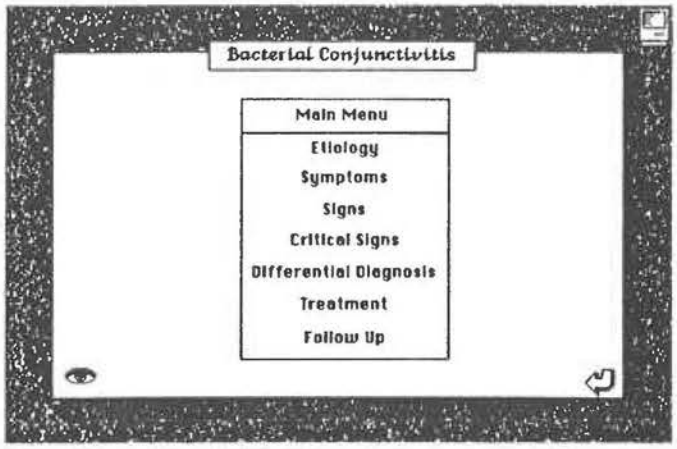
- Cycloplegics:
  SEVERE: Atropine 1% 1 gtt bid or Scopolamine 0.25% 1 gtt bid (Use with caution!)
  MODERATE: Homatropine 5% tid or qid or Cyclopentolate 2% bid - tid
  MILD : Tropicamide 1.0% tid or Cyclopentolate 1% tid
- Topical steroids:
  SEVERE: Prednisolone acetate 1% every hour
  MILD to MODERATE: Prednisolone acetate 1% qid
- Periocular repository steroid should be considered if not responding well to topicals.
- Systemic steroids are then considered if still not responding well, and possibly even systemic immunosuppressive agents. This requires meticulous follow-up by internist and oncologist.

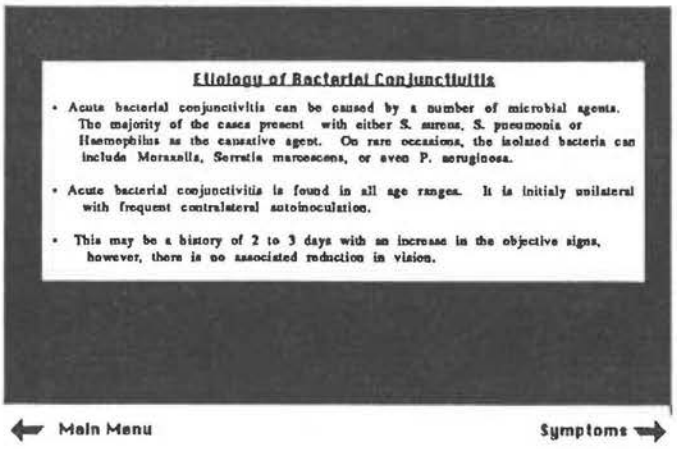
Work up | Follow up

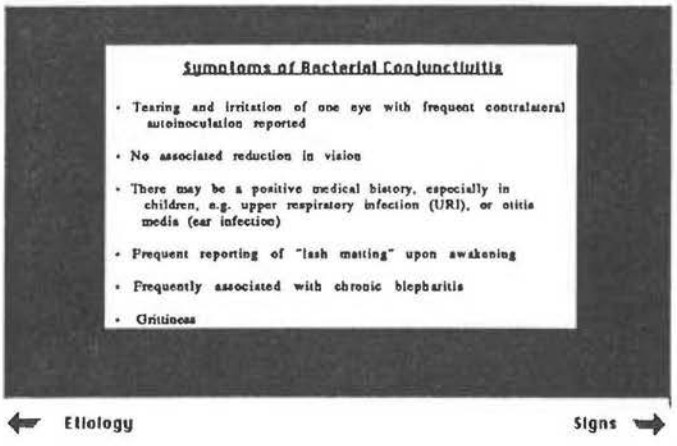
Follow-Up of Anterior Uveitis

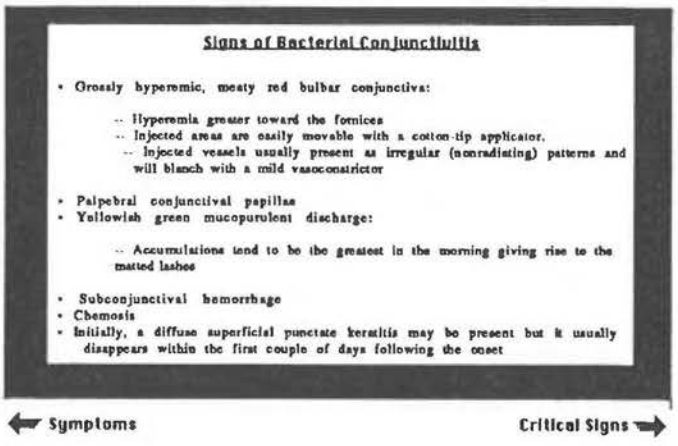
- Follow up depends of severity. It is recommended that the patient be seen every 1-7 days in the acute phase. If the condition is chronic and stable, then the patient can be seen every 1-6 months.
- If the anterior chamber reaction is improving, then the steroid and cycloplegic can be tapered until chamber is free of cells. Flare may still be present.
  -- Steroids can be tapered 1 drop per day for 3-7 days
  -- Cycloplegics used every night until chamber clear and tapered slowly if granulomatous reactions. (There is a higher tendency for posterior synchiae)
- Check vitreous and fundus for all flare ups, when vision is affected or every 3-6 mo.
- Educate and watch for all the complications to steroid use.
- In cases with recurrent uveitis, look for an etiolgy.

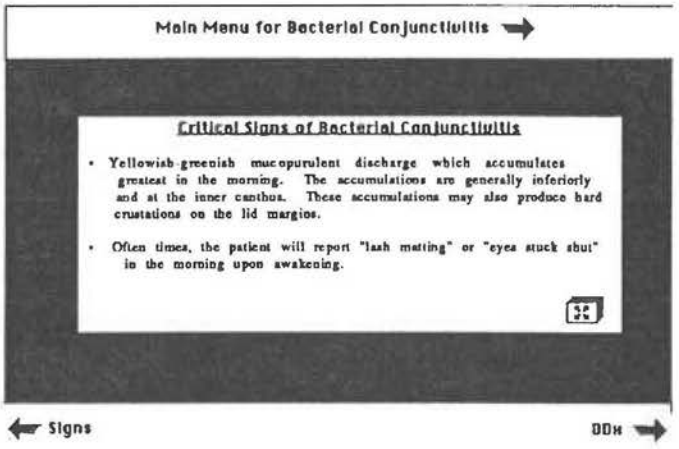
Treatment | Menu

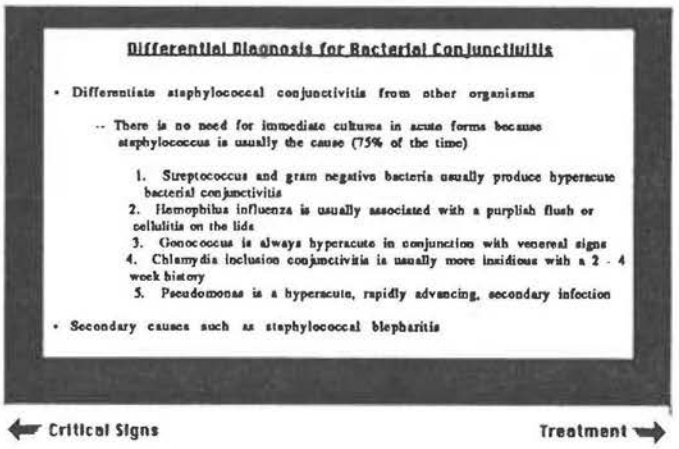

Bacterial Conjunctivitis
Main Menu
Etiology
Symptoms
Signs
Critical Signs
Differential Diagnosis
Treatment
Follow Up

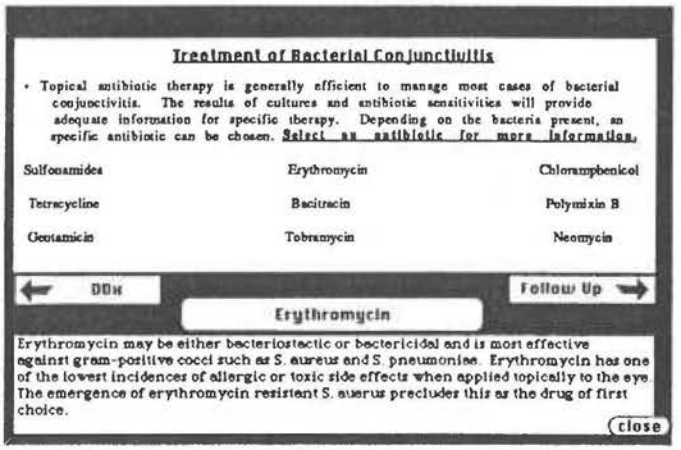

Etiology of Bacterial Conjunctivitis
• Acute bacterial conjunctivitis can be caused by a number of microbial agents. The majority of the cases present with either S. aureus, S. pneumonia or Haemophilus as the causative agent. On rare occasions, the isolated bacteria can include Moraxella, Serratia marcescens, or even P. aeruginosa.
• Acute bacterial conjunctivitis is found in all age ranges. It is initialy unilateral with frequent contralateral autoinoculation.
• This may be a history of 2 to 3 days with an increase in the objective signs, however, there is no associated reduction in vision.
Main Menu
Symptoms

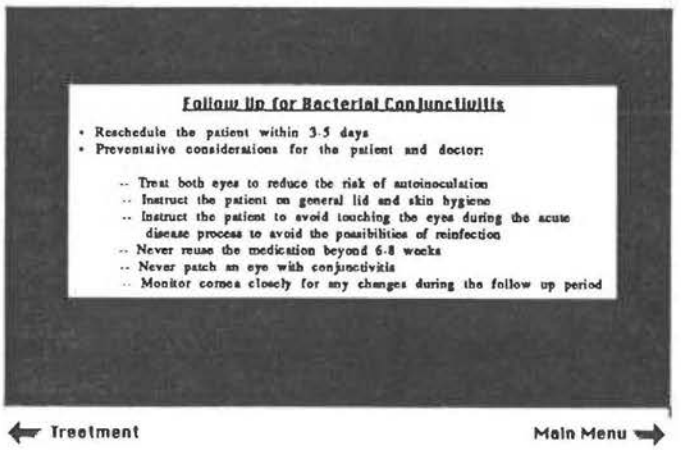

Symptoms of Bacterial Conjunctivitis
• Tearing and irritation of one eye with frequent contralateral autoinoculation reported
• No associated reduction in vision
• There may be a positive medical history, especially in children, e.g. upper respiratory infection (URI), or otitis media (ear infection)
• Frequent reporting of "lash matting" upon awakening
• Frequently associated with chronic blepharitis
• Grittiness
Etiology
Signs

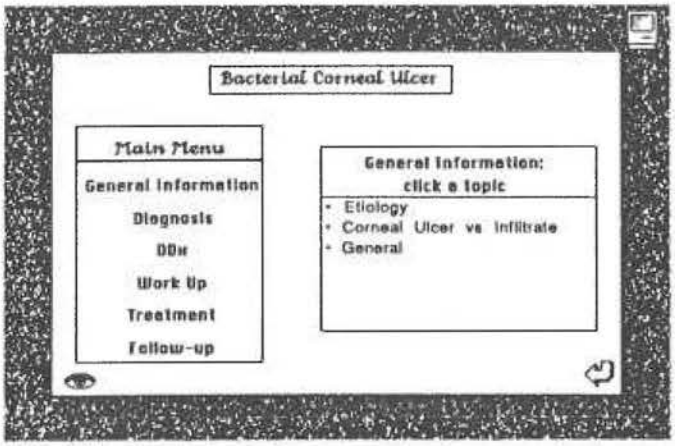

Signs of Bacterial Conjunctivitis
• Grossly hyperemic, meaty red bulbar conjunctiva:
-- Hyperemia greater toward the fornices
-- Injected areas are easily movable with a cotton-tip applicator.
-- Injected vessels usually present as irregular (nonradiating) patterns and will blanch with a mild vasoconstrictor
• Palpebral conjunctival papillae
• Yellowish green mucopurulent discharge:
-- Accumulations tend to be the greatest in the morning giving rise to the matted lashes
• Subconjunctival hemorrhage
• Chemosis
• Initially, a diffuse superficial punctate keratitis may be present but it usually disappears within the first couple of days following the onset
Symptoms
Critical Signs


Main Menu for Bacterial Conjunctivitis
Critical Signs of Bacterial Conjunctivitis
• Yellowish-greenish mucopurulent discharge which accumulates greatest in the morning. The accumulations are generally inferiorly and at the inner canthus. These accumulations may also produce hard crustations on the lid margins.
• Often times, the patient will report "lash matting" or "eyes stuck shut" in the morning upon awakening.
Signs
DDx


Differential Diagnosis for Bacterial Conjunctivitis
• Differentiate staphylococcal conjunctivitis from other organisms
-- There is no need for immediate cultures in acute forms because staphylococcus is usually the cause (75% of the time)
1. Streptococcus and gram negative bacteria usually produce hyperacute bacterial conjunctivitis
2. Hemophilus influenza is usually associated with a purplish flush or cellulitis on the lids
3. Gonococcus is always hyperacute in conjunction with venereal signs
4. Chlamydia inclusion conjunctivitis is usually more insidious with a 2 - 4 week history
5. Pseudomonas is a hyperacute, rapidly advancing, secondary infection
• Secondary causes such as staphylococcal blepharitis
Critical Signs
Treatment


Treatment of Bacterial Conjunctivitis
• Topical antibiotic therapy is generally efficient to manage most cases of bacterial conjunctivitis. The results of cultures and antibiotic sensitivities will provide adequate information for specific therapy. Depending on the bacteria present, an specific antibiotic can be chosen. Select an antibiotic for more information.
Sulfonamides
Erythromycin
Chloramphenicol
Tetracycline
Bacitracin
Polymixin B
Geotamicin
Tobramycin
Neomycin
DDx
Follow Up
Erythromycin
Erythromycin may be either bacteriostactic or bactericidal and is most effective against gram-positive cocci such as S. aureus and S. pneumoniae. Erythromycin has one of the lowest incidences of allergic or toxic side effects when applied topically to the eye. The emergence of erythromycin resistant S. aureus precludes this as the drug of first choice.
close


Follow Up for Bacterial Conjunctivitis
• Reschedule the patient within 3-5 days
• Preventative considerations for the patient and doctor:
-- Treat both eyes to reduce the risk of autoinoculation
-- Instruct the patient on general lid and skin hygiene
-- Instruct the patient to avoid touching the eyes during the acute disease process to avoid the possibilities of reinfection
-- Never reuse the medication beyond 6-8 weeks
-- Never patch an eye with conjunctivitis
-- Monitor cornea closely for any changes during the follow up period
Treatment
Main Menu



Etiology of Infectious Keratitis

Common Pathogens	Gram Stain	Rate of Progression	Key Characteristics
• S. aureus	G+	Usually days	Purulent ropy discharge, indistinct margins
• S. Pneumo	G+	1-2 days	Gray well circumscribed ulcer.
• P. aeruginosa	G-	hours	Greenish mucopurulent discharge/hypopyon
• N. gonorrhoae	G-	12-24 hours	Hyperacute purulence
• H. influenza	G-	?	Often a bluish, purplish preseptal flush
• Moraxella	G-	?	Generally in alcoholic or debilitated persons. Paracentral or perilimbal infection.

Menu
Ulcers vs Infiltrates

Corneal Ulcers vs Infiltrates

- Any corneal break extending from the epithelium through Bowmans and into the stroma is an ULCER.
- Corneal infiltrates may appear as small, whitish opacities in the subepithelial area. They are usually ill-defined with intact, overlying epithelium. They do not stain.
- A corneal stromal infiltrate underlying an epithelial break should be considered an infective ulcer until proven otherwise.
- True differentiation between infectious and non-infectious ulcers requires culturing.

Etiology
General Information

General Information about Corneal Ulcers

- Ulcers may result from hypersensitivity reactions, trauma or infections.
- Central ulcers are often infectious while peripheral ulcers are generally toxic resulting from antigen/antibody reactions to Staph.
- Unchecked corneal ulcers, regardless of where they begin, progress away from the limbus.
- The most common cause of corneal infection in developed countries is herpes simplex.
- Signs, symptoms and diagnosis of corneal ulcers vary greatly for both the contact lens wearer and the nonwearer. Suspect G- organisms in contact lens wearers.

Ulcers vs Infiltrates
Critical Signs

To Main Menu For Corneal Ulcer

Critical Signs for Infectious Corneal Ulcer

- An ulcer exists if the infiltrate is accompanied by an overlying epithelial defect that stains with flourescein.

General Information
Signs

Signs of Infectious Keratitis/Bacterial Corneal Ulcer

- Generally unilateral
- Epithelial defects
- Conjunctival injection (severe crimson red > 180 degrees)
- Chemosis
- Stromal edema and inflammation surrounding the infiltrate
- Mucopurulent discharge
- Anterior chamber reaction
- Blepharospasm
- Corneal thinning
- Papillary changes
- Increased IOP
- Lid edema
- Pupil miotic

• All signs above need not be present •

Critical Signs
Symptoms

Symptoms of Infectious Keratitis/Bacterial Corneal Ulcer

- Significant ocular pain and discomfort which may follow the following sequence
  Irritation--> burning--> tearing--> photophobia--> blurred VA--> foreign body sensation--> dull aching pain
- Discharge
- Blurred vision especially if central
- Red eye
- Photophobia
- Foreign body sensation
- Burning
- Tearing
- May have a history of trauma

• All symptoms above need not be present •

Signs
Making the Diagnosis

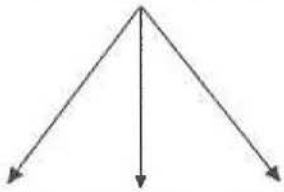
Making the Diagnosis of Infectious Keratitis

- Making the diagnosis can be difficult. You must distinguish microbial keratitis from other types of infiltrative and ulcerative keratitis.
- Since there is no absolute biomicroscopic sign of infection, you must proceed with laboratory investigations if there is any suspicion of microbial keratitis
- After making the diagnosis and performing the proper lab tests, the next three steps are to 1) initiate therapy, 2) modify initial therapy, and (4) terminate therapy.

Symptoms
Differential Dx

Differential Diagnosis

For more information about the following conditions, click on each topic below.



Fungal Keratitis | Acanthamoeba | Herpes Simplex Keratitis

← Making the Diagnosis | Work up →

Work Up for Infectious Keratitis

- Case history: Do they wear contact lenses? If so, how does patient take care of them? Does patient swim with their lenses on ? Any trauma or corneal foreign body? Abrasion with vegetable matter? Any previous corneal disease or systemic illness? Is the patient taking any medication?
- Visual acuity / Pinhole if < 20/20
- SLE: Document size, depth and location of lesion. Is there epithelial loss over the infiltrate? Look for anterior chamber reaction (before flourescein) and check IOP
- If significant discharge, swab palpebral conjunctiva. If you suspect an infectious infiltrate or ulcer, a corneal scraping should be performed.
- Corneal sensitivity testing can help differentiate HSV keratitis. Use a separate wisp for each eye.
- If severe, refer to corneal specialist.

Severity Grade of Keratitis

← Differential Dx | Treatment →

Treatment of Infectious Bacterial Keratitis

The two goals are to eliminate causative agent (bacteria) and suppress inflammatory response.

- Consider and coordinate the most appropriate professionals for care and management. Ulcers and infiltrates are generally treated as bacterial unless there is a high suspicion of fungal, acanthamoeba or HSV keratitis. See DDx card.
- Hospitalization should be considered if there is a severe sight threatening infection, if the patient is unable to comply with the antibiotic therapy due to the frequency of administration, or if systemic antibiotics are needed.
- There is little to gain initiating steroid therapy prior to positive identification of the organism. Corticosteroid therapy is controversial.
- Consult with ophthalmologist.

← Work up | Specific Tx →

Treatment of Infectious Bacterial Keratitis

1. Cycloplegic (i.e. 5% Homatropine q2h)
2. Topical antibiotics (depends on size and severity)

Which antibiotic ??

- If a small nonstaining infiltrate with no anterior chamber reaction or no discharge:
  - Broad spectrum antibiotics (Polymixin B/Bacitracin ung qid)
  - If contact lens wearer, use Tobramycin drops q 2-6 hrs. Also consider adding Tobramycin ointment at night.
- If large staining infiltrate or moderate-severe anterior chamber reaction or discharge:
  - Fortified drops qh. Generally a minimum of two aminoglycosides are used with one of them being either Gentamicin or Tobramycin. This means one drop every 30 minutes.

3. Subconjunctival antibiotics should be considered in severe cases.
4. Oral and IV antibiotics are indicated in ulcers with significant threat of corneal perforation.
5. Oral pain medications are often indicated.

NO CONTACT LENS WEAR AND NO PATCHING IN AN EYE WITH AN INFECTON.

← General Tx | Follow up →

Follow Up of Bacterial Infectious Ulcers

- The patient is seen daily to re-evaluate. The size and depth of infiltrate should be noted along with degree of pain, size of epithelial defect, and anterior chamber reaction. Check IOP.
- If ulcer improves, antibiotic therapy is tapered. Therapy is modified based on culture results.
- If the ulcer has not improved, ulcer should be recultured and hospitalization should be reconsidered. Some suggest reculture within 48 hrs., and every 24 hours after until culture is negative.
- Continue with patient education, and RTC if pain increases or vision decreases.

← Specific Tx | Menu →

Blepharitis

Main Menu

- Etiology
- Description
- Signs
- Symptoms
- Critical Signs
- Treatment
- Follow up

Etiology of Blepharitis

- Chronic blepharitis is a disease that will be commonly seen in the office. Recently, an updated classification of chronic blepharitis based on the clinical signs and symptoms has been presented. The current classification divides chronic blepharitis into three main categories: the first being that of staphylococcal blepharitis, the second one being seborrheic blepharitis, and the third classification being meibomian gland dysfunction.

1. Staphylococcal Blepharitis: This is caused by a chronic staphylococcal infection of the bases of the lashes. The disease generally starts in early childhood and may continue throughout life.
2. Seborrheic Blepharitis: This is a disorder of the glands of Zeis which is frequently associated with seborrheic dermatitis.
3. Meibomian Gland Dysfunction: This is a disorder of the meibomian glands and can be thought of as a posterior form of seborrheic blepharitis.

Main Menu | Description

Description of Blepharitis

1. Staphylococcal Blepharitis: This condition usually presents with a history of waxing and waning signs and symptoms that are usually of shorter duration than those of the other types of blepharitis. The patients are usually younger, with a mean age of 42, compared with 51 years for the other types of blepharitis. It is more common in females, representing 80 percent of the total cases.
2. Seborrheic Blepharitis: This condition usually presents in an older age group with a mean age of 51 years and has a longer duration of symptoms as compared to staphylococcal blepharitis.
3. Meibomian Gland Dysfunction: This condition is distinguished from seborrheic blepharitis by a shorter duration of symptoms at the time of presentation and a more pronounced inflammation of the eyelids.

Etiology | Signs

Signs of Blepharitis

1. Staphylococcal Blepharitis: Clinical features include inflamed eyelids (greater than that of seborrheic blepharitis) erythema and, sometimes, edema along the anterior ciliary portion of the lids. The lid margins are typically involved, including telangiectatic changes. Crusting of the lashes occurs with collarettes surrounding the individual cilia. Anterior and posterior bordeola intermittently occur. Fifteen percent of the cases include bulbar and tarsal conjunctival changes, including injection, and when chronic, papillary hypertrophy of the tarsal conjunctiva. If the response is acute, a follicular response may develop over the inferior tarsal plate.
2. Seborrheic Blepharitis: Clinical features include inflammed eyelids with less frequent exacerbations than staphylococcal blepharitis. The debris deposited on the eyelids has an oily and greasy consistency that is often called "scurf". There is typically also dermatological findings to support the diagnosis.
3. Meibomian Gland Dysfunction: The most prominent feature is diffuse inflammation around the meibomian glands, which are often dilated with retained meibum that is not easily expressed. The orifices of the glands are obstructed and pout with inspissated secretions. The anterior/ciliary aspect of the lids are frequently only minimally involved with a deposition of an oily scurf.

Description | Symptoms

Symptoms of Blepharitis

Staphylococcal Blepharitis:
- Irritation
- Burning
- Itching
- Photophobia

** these symptoms are characteristically worse in the morning **

Seborrheic Blepharitis:
- Irritation
- Burning
- Itching
- Photophobia

** these symptoms are less severe than staphylococcal blepharitis **

Meibomian Gland Dysfunction:
- Stinging
  - Blurred vision secondary to the oil in the precorneal film

Signs | Critical Signs

Main Menu for Blepharitis

Critical Signs for Blepharitis

- Crusty, red, thickened eyelid margins with prominent blood vessels (blepharitis) and/or inspissated oil glands at the eyelid margins (meibomianitis)

Symptoms | Treatment

Treatment for Blepharitis

The treatment for blepharitis is very similar and is routinely administered without the use of bacterial cultures. However, if a certain treatment modality is unsuccessful, a standard work up should involve a bacterial culture and/or smear to assess the infecting organism(s).

	Dosage/Duration	
	Acute presentation	Chronic presentation
1. Skin/scalp hygiene	Daily/ongoing	Daily/ongoing
2. Hot compresses	Every 4 hours	HS/ 3-6 times / week
3. Lid massage	Not indicated	HS/ 3-6 times / week
4. Lid scrubs	HS/ for 1-2 weeks	HS/ for 3 mos. then PRN
5. Lid ointment	q4h / 5-7 days (e.g., gentamicin)	HS/2-4 wk (Bacitracin)

Critical signs | Follow Up

Follow Up Care for Blepharitis

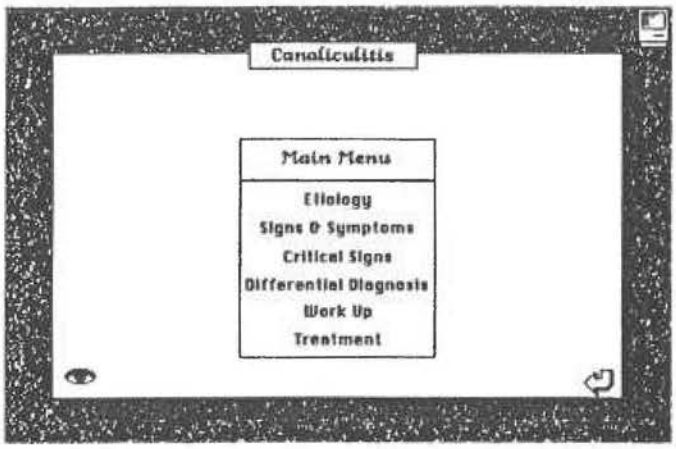
Acute Forms

- Should be stable or improving in 48 to 72 hours.
- Continue treatment for at least 5-7 days.
- If condition is worsening:
  - Reconfirm diagnosis
  - Check patient compliance/reinstruct
  - Re-evaluate for pre-ceptal cellulitis development
  - Consider adding a broad-spectrum oral antibiotic
- Upon resolution, instruct preventative measures

Chronic Forms

- Improvement is usually significant in 4-6 weeks
- Gradually reduce dosages to maintenance levels
- If no improvement:
  - Reconfirm diagnosis
  - Check patient compliance/reinstruct
  - Continue treatment plan
  - Discontinue ointments in squamous forms
- Advise patients on potential chronicity
- Reduce fatty foods in all seborrheic types
- Instruct on ongoing preventative and maintenance therapy

Treatment | Main Menu



Etiology of Canaliculitis

- Canaliculitis is a relatively rare condition occurring in only about 2% of the population with tearing problems. The most likely causative agents are Actinomyces, fungal, viral, herpes and trauma. Recently an allergic etiology has been reported.
- The initial diagnostic suspicion is the consideration of the patient's age. Actinomyces infections are more prominant among patients over the age of 50, whereas herpetic infections have a higher incidence in patients under the age of 20.

← Main Menu Signs & Symptoms →

Signs and Symptoms of Canaliculitis

- Unilateral red eye that has been resistant to antibiotic therapy
- Mild tenderness over the nasal aspect of the upper/lower eyelid
- Epiphoria
- Watery discharge
- Tearing
- Purulent discharge

← Etiology Critical Signs →

Main Menu for Canaliculitis →

Critical signs of Canaliculitis

- "Wrinkle sign" - compression of the medial canthal skin appearing as a wrinkle.
  - -- This sign generally suggests that there may be internal obstruction of the lacrimal drainage system.
  - -- The observation of smooth skin with the ability to advance an instrument to the hard stop of the lacrimal bone generally indicates a patent proximal drainage system.
- Erythematous skin surrounding the punctum

← Signs & Symptoms DDx →

Differential Diagnosis of Canaliculitis

1. Dacryocystitis - usually presents with much more swelling, tenderness, and pain compared to canaliculitis.
2. Nasolacrimal duct obstruction - usually presents with minimal to no erythema or tenderness around the punctum.
3. Conjunctivitis - usually accompanied with conjunctival papillae and/or follicles in conjunction with a discharge.

← Critical Signs Work Up →

Work Up for Canaliculitis

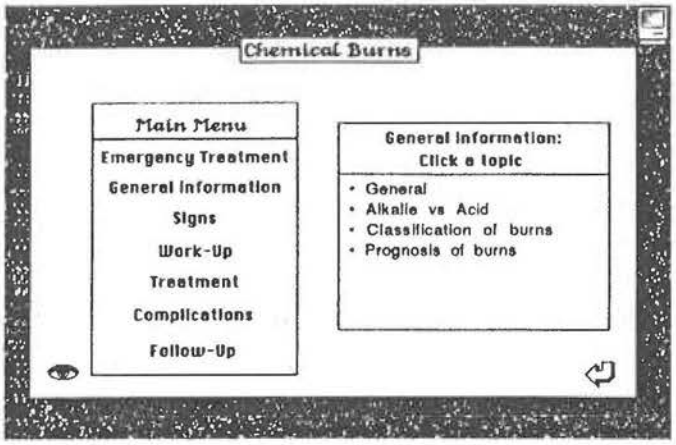
- Gently apply pressure to the lacrimal sac with a cotton swab, rolling it toward the punctum. Observe for any punctal discharge.
- If there is any material expressed, laboratory smears and cultures should be evaluated. Possible smears or stains include: (1) Gram's stain, (2) Giemsa stain, (3) Thioglycolate and Sabouraud's cultures, or (4) a KOH smear if available - (apply 1 drop of 20% KOH on a slide along with a sample of the material expressed.)

← DDx Treatment →

Treatment for Canaliculitis

1. Remove the obstructive concretions and culture
2. Irrigate the canaliculus with 100,000 units/ml of penicillin G solution or a 1% solution of Iodine. (Irrigation should take place while the patient is in the upright position so that the solution will drain out of the nose and not into the nasopharynx.)
   - If the smears reveal a fungus - use nystatin 1:20,000 drops tid in conjunction with a nystatin 1:20,000 solution irrigation several times per week
   - If the smears reveal a herpes virus - use trifluorothymidine 1% drops (e.g., Viroptic) 5 X / day for several weeks
3. Warm compresses applied to the punctal area qid
4. More extensive surgical treatment may be recommended.

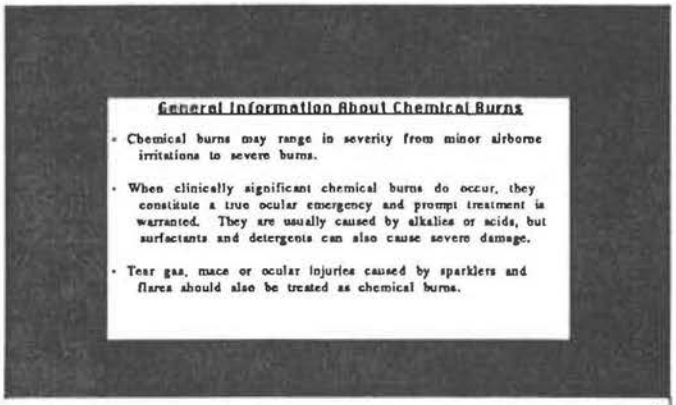
← Work Up Main Menu →



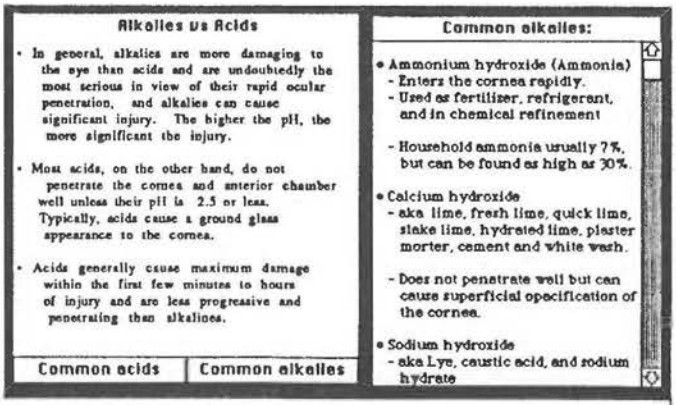
Emergency Treatment For Chemical Burns

- The primary step in management is prompt dilution of the offending agent. Initial irrigation should take place at the sight of injury. If wearing contact lenses, lenses should be removed. If patient has called the office, be sure to get name and phone number, and instruct patient over the phone about irrigation.
- Irrigate the eyes and the ocular surface with copious amounts of water or saline for at least 30 minutes or until pH reaches normal range (7.3-7.7). It may be helpful to place an eyelid speculum and topical anesthetic in eye prior to irrigation if patient is in the office.
- It is essential to irrigate the fornices and remove any caustic material. Do not use acidic solutions to neutralize alkalies or vice versa.
- 5 minutes after irrigation (to allow for equilibration), litmus paper should be touched to the inferior cul-de-sac to check to see that pH is still in the normal range.
- Rapid transport to an emergency facility (Ophthalmologist) is generally necessary. Call ahead so that treatment will be waiting when the patient arrives.
- Emergency treatment may be the most important determinant in the ultimate prognosis of the burn. Generally, subsequent damage is directly proportional to how long the offending agent remained in contact with the tissue.

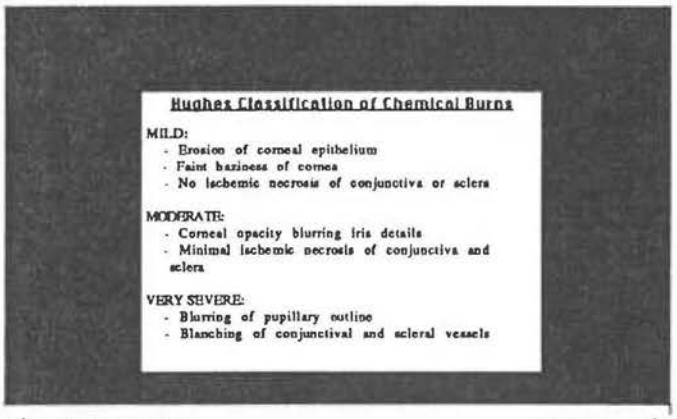
Main menu for Chemical Burns



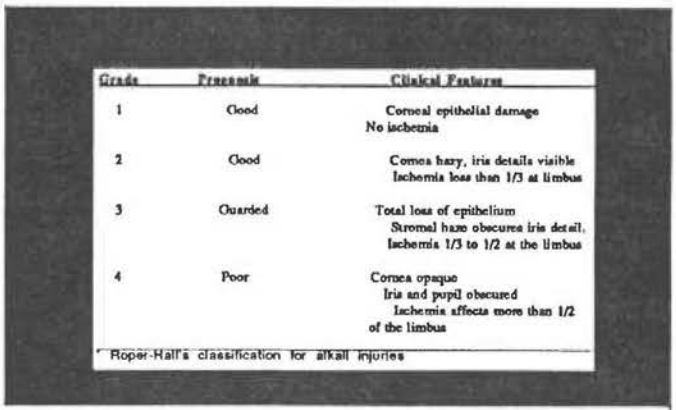
Menu
Alkalies vs Acids



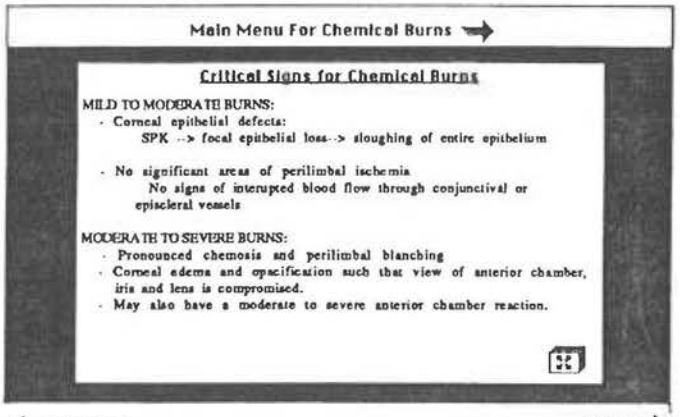
General Information
Classification of Burns



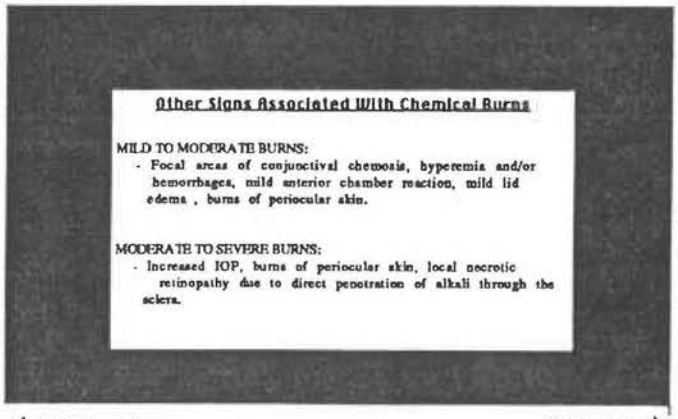
Alkalies vs Acids
Prognosis



Classification of Burns
Critical Signs



Prognosis
Signs



Critical signs
Work up

Work Up of Chemical Burns

- Irrigate eye till ph is neutral.
- History:
  Time of injury? What chemical was the patient exposed to? Duration of exposure before irrigation and duration of irrigation? How much of the chemical got into the eye?
- Visual acuities
- Slit lamp exam with fluorescein (Assess damage)
  - Evert eyelids to search for foreign bodies.
  - Epithelium (intact vs compromised)
  - Corneal stroma (clear vs opacified)
  - Perilimbal vessels (engorged vs blanched)
  - Check IOP if possible

← Signs Treatment →

Treatment (after irrigation) for Chemical Burns

- Treatment is aimed at promoting epithelial healing, avoiding infection, and preventing stromal ulceration. Consult with Ophthalmologist.

1. Antibiotics are essential to prevent secondary bacterial infection. (i.e. Tobramycin drops 0.3% qid / Tobramycin ointment 0.3% qhs)
2. Cycloplegic to reduce pain, inflammation, and prevent synechia (i.e. cyclopentalate 1.0% or scopolamine 2.5% and phenylephrine 2.5%)
3. Hypotensives in presence of increased IOP (i.e. Betoptic 0.5% bid)
4. Corticosteroid use is controversial. Some suggest use for first week in moderate to severe burns. (i.e. Dexamethasone 0.1% qid)
5. Two new treatments under investigation:
   - Ascorbic acid and citric acid 10%
     (decrease incidence of stromal ulceration and perforation)
6. Surgical intervention may be necessary in some severe cases.

← Work up Complications →

Complications of Chemical Burns

Below is a list of conditions commonly associated with chemical burns:

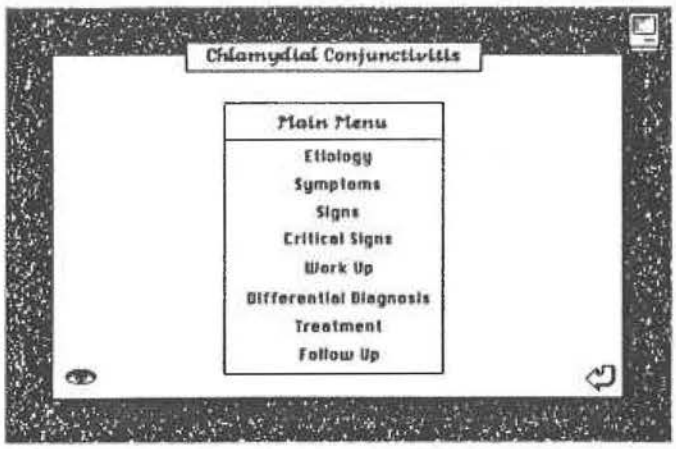
- Chronic anterior uveitis
- Chronic glaucoma
- Entropion
- Infection
- Keratitis sicca
- Neovascularization and pannus
- Perforation
- Phthisis bulbi
- Scarring
- Symblepharon
- Ulceration

← Treatment Follow up →

Follow-Up for Chemical Burns

- Recheck cornea every 24 hours in moderate to severe cases.
- If using steroids, always taper after 5-7 days, especially in alkali burns, to minimize ulceration risks.
- Taper antibiotic but continue until there is no longer any staining.
- Prognosis is good for mild chemical burns with complete resolution in 1 to 2 weeks. Moderate burns may take up to 6 weeks to heal especially if an alkaline burn.
- Prognosis is poor in severe burns with significant risk of secondary ulceration, infection, scarring and perforation.
- Long term therapy depends on the severity of the burn.

← Complications Menu for Chemical Burns →



Follicles - pearly white or gray elevations that is an aggregation of lymphocytes and macrophages; blood vessels can often be noted coursing around the follicles.

Papillae - small conjunctival elevations containing a centrally positioned blood vessel surrounded by inflammatory exudate and cells.

close

Main Menu | Symptoms

Symptoms of Chlamydial Conjunctivitis

- Acute bilateral onset
  - Watering and mucoid discharge
  - Sticking of the lids in the morning
- Foreign body sensation
  - Conjunctival hyperemia
- Swelling of the lids

Etiology | Signs

Signs of Chlamydial Conjunctivitis

- Edematous eyelids
  - Palpable preauricular node
  - Follicular conjunctivitis involving the lower tarsus
  - Visible conjunctival lymphoid follicles
- Fine tarsal papillae
  - Fine and large macropunctate epithelial erosions
  - Subepithelial/stromal infiltrates
- Limbal infiltration
  - Superficial neovascularization / corneal pannus
- Occasional conjunctival chemosis
- Occasional development of anterior uveitis (chronic)

Symptoms | Critical Signs

Main Menu For Chlamydial Conjunctivitis

Critical Signs of Chlamydial Conjunctivitis

- An acute follicular conjunctivitis with a mucopurulent discharge occurring after an incubation time of approximately 4-12 days.
- This disease usually occurs in sexually active young adults but is by no means limited to any specific population.
- The ultimate diagnosis is facilitated by the presence of intraepithelial inclusion bodies apparent in epithelial cells obtained by conjunctival scrapings.

Signs | Diagnosis

Work Up of Chlamydial Conjunctivitis

LABORATORY TESTS USED TO IDENTIFY CHLAMYDIAL INFECTIONS:

1. Giemsa staining of smears
2. Isolation in cell culture
3. Direct fluorescent monoclonal antibodies (DFA)
4. ELISA test
5. Serum antibody levels measured by compliment-fixation (CF) or microimmunofluorescent (MIF) tests.

Critical Signs | DDx

Differential Diagnosis of Chlamydial Conjunctivitis

** Usually the history is the most significant differential. With chlamydia, the patient is typically a sexually active adult with a history, or recent urinary tract involvement.

1. Bacterial conjunctivitis - generally presents as a "meaty" red conjunctivitis that increases toward the fornices. The cornea is typically clear; the hyperemic vessels will blanch with a mild vasoconstrictor; usually a papillary response and a mucopurulent discharge.

2. Viral conjunctivitis - generally presents as a pinkish-purple hyperemia that increases toward the plica. There is typically a fast TBUT; a follicular response, a serous discharge, and enlargement of preauricular node or lymphadenopathy.

Work Up | Treatment

Treatment of Chlamydial Conjunctivitis

SYSTEMIC: It is necessary to use systemic antimicrobial treatment because the infections produced in chlamydia are not limited to the eye.

- For infants, effective therapy is provided by oral erythromycin, 40 mg/kg daily in four divided doses for 2 weeks.
- Adults: Daily administration of tetracycline 250 mg po qid for a duration of 6 weeks. Erythromycin is effective at doses of 250 mg po qid for 6 weeks. Doxycycline, which is better absorbed than tetracycline, is another alternative at a dosage of 300 mg weekly for 3 weeks or 100 mg daily for 1-2 weeks. All systemic medication should be administered to the patient as well as their sexual partners.

OCULAR: Topical rifampin, erythromycin, tetracycline or sulfacetamide ointment have all been used 2-3 X/day for 4 - 6 weeks.

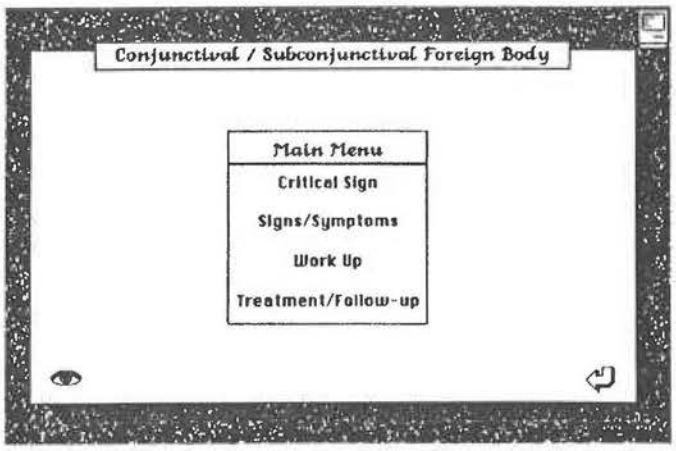
** Avoid tetracycline in pregnant females, mothers who are nursing, and children less than 8 years of age.

DDx | Follow Up

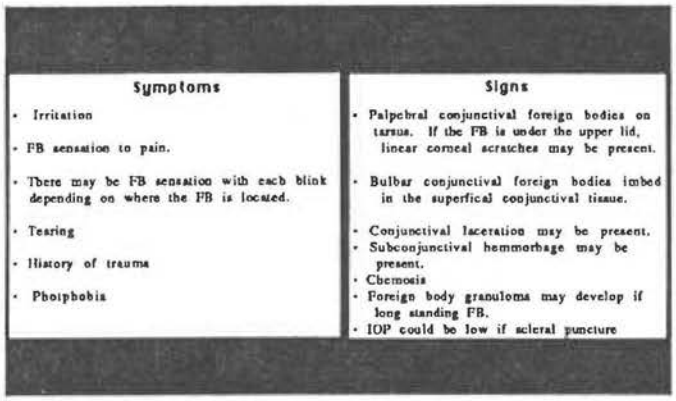
Follow Up for Chlamydial Conjunctivitis

- Patients with ocular manifestations should be examined every 1-3 weeks depending on the severity of the condition. The patient, as well as their sexual partners, should also be evaluated by their physician for other sexually transmitted diseases.
- Clinical signs of chlamydial conjunctivitis may take 2-3 weeks to resolve completely with treatment. Corneal findings such as SPK and subepithelial infiltrates may persist for 6-12 months.
- If the patient is a contact lens wearer, it is very important that the lenses be discontinued until 2-4 weeks following the resolution of the disease.

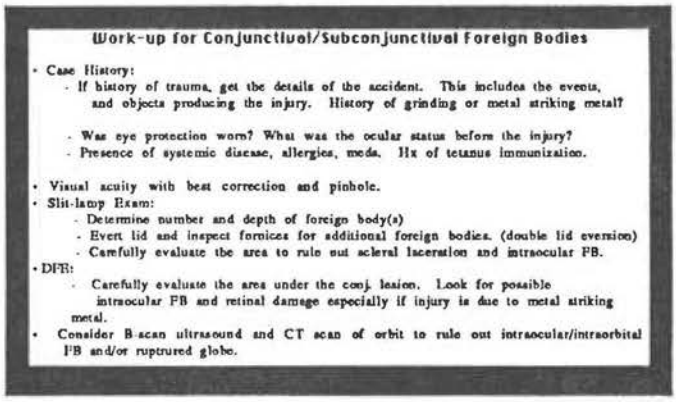
Treatment Main Menu



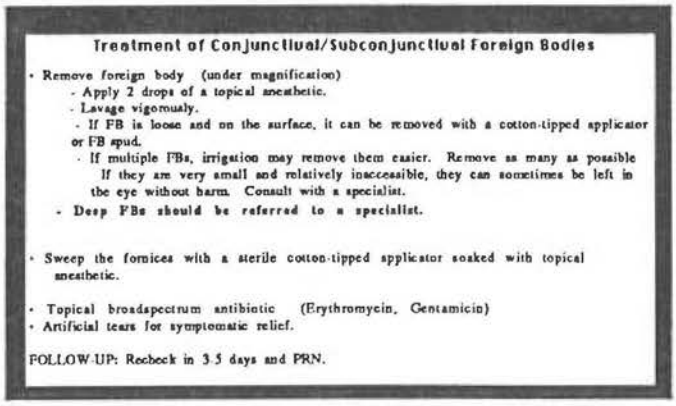
Main menu for Conj. Foreign Body
Signs and Symptoms



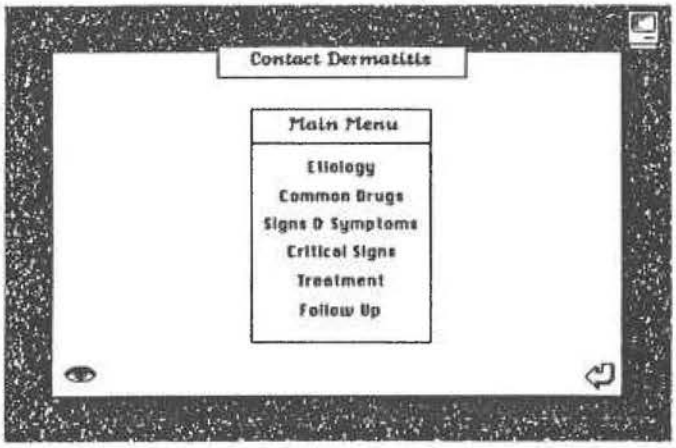
Critical signs
Work-up



Signs
Treatment



Work-up
Menu



Etiology Of Contact Dermatitis

- Contact dermatitis is either classified as primary irritant (nonallergic) or allergic.
- Primary irritant is more common but is seen less often in the office because the onset is relatively rapid (1 - 24 hours) following exposure. It is this rapid onset that allows a self diagnosis and avoidance of the offending irritant.
- Allergic contact dermatitis, on the other hand, is a delayed cell-mediated hypersensitivity reaction that usually involves the eyelid skin and may secondarily involve the conjunctiva. The onset of allergic dermatitis can ocurr anywhere from 12 - 72 hours after the exposure to as long as one year, depending on the potency of the irritant.

Main Menu | Common Agents

Common Drugs or Substances Known to Cause Allergic Dermatitis

- Cosmetics (including nail polish)
- Local anesthetics
- Neomycin
- Tobramycin
- Gentamicin
- Bacitracin
- Benzalkonium chloride
- Thimerosol
- Parabens
- Potassium sorbate
- Atropine
- Timolol
- Phenylephrine
- Lanolin
- Rubber or nickel (eyelash curler)

** NOTE **

Erythromycin has never been reported to cause an allergic contact dermatitis!

Etiology | Signs & Symptoms

Signs and Symptoms of Contact Dermatitis

- Sudden onset of periorbital rash or eyelid swelling
- Itching
- Mild watery discharge
- Diffuse conjunctivitis
- Papillary reaction
- Chemosis
- Crusting of the skin with eruptions may develop when associated with a secondary infection

Common Agents | Critical Signs

Main Menu for Contact Dermatitis

Critical Signs of Contact Dermatitis

- Periorbital edema
- Erythema
- Vesicles
- Thickening or hardening of the skin

Signs & Symptoms | Treatment

Treatment of Contact Dermatitis

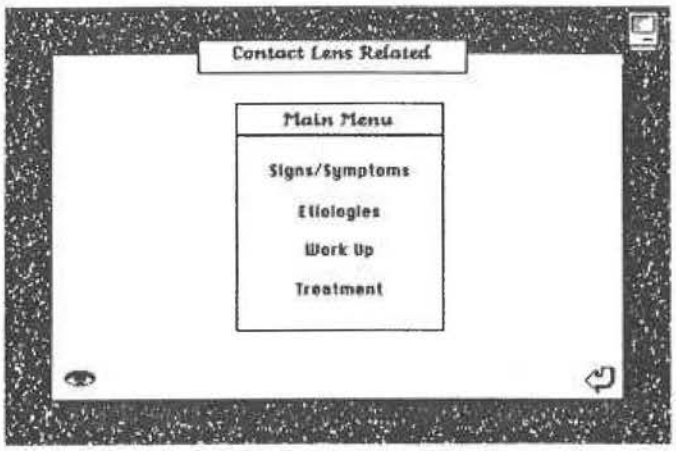
- Eliminate the offending agent.
- Cool compresses 4 - 6 times / day for the more severe cases.
- Oral antihistamines (e.g., Benedryl) 25 - 50 mg po 3 - 4 times / day. Topical antihistamines will have little effect if the ocular involvement is minimal.
- Mild steroid or cream ointment (e.g., 1.0% hydrocortisone) is helpful in dry, subacute, and chronic stages, and should be applied to the periocular area 2 - 3 times / day until the reaction subsides.

Critical Signs | Follow Up

Follow Up for Contact Dermatitis

- Reexamine within one week and taper the steroid cream upon the remission of the symptoms.

Treatment | Main Menu



Signs & Symptoms of Contact Lens Related Conditions

There is a wide range of signs and symptoms depending on the etiology. Any or all of the following may be present.

- Varying degrees of pain
- Photophobia
- Red eye
- Foreign body sensation
- May have blurred vision
- Itching
- Corneal edema
- Corneal infiltrates
- SPK, pseudodendrites
- Corneal ulcer
- Hyperemia (diffuse or perilimbal)
- AC reaction (mild/moderate/severe)
- Discharge

Main Menu For Contact Lens Related Conditions | Etiologies

Etiologies

Assume that the contact lenses are the cause of the red eye until proven otherwise.

- Corneal ulcer or infiltrates:
  - White corneal lesion that may stain with flourescein.
  - Corneal ulcers are the most serious complication associated with contact lenses, and must be ruled out. Think Pseudomonas and Acanthamoeba especially in EW.
  - Corneal infiltrates may be infectious or sterile.
  - Sterile infiltrates in patients wearing contact lenses have a variety of causes

Causes of Infiltrates

- GPC:
  - Large superior tarsal conjunctival papillae along with mucous, itching and lens intolerance

Others to consider

- Hypersensitivity reactions:
  - Staph
  - Preservatives in solution (thimerosal/chlorhexidine etc.). Injection and irritation often developing after lens cleaning or insertion. Diffuse SPK along with bulbar conjunctival follicles and corneal infiltrates may be present.

Signs/Symptoms | Work-up

Other Etiologies to Consider

Also consider the following:

- Contact lens deposits
- Corneal warpage
- Corneal neovascularization
- Corneal abrasion
- Poor contact lens fit
- Lens inside out
- Epithelial thickening due to toxic/ traumatic reaction to contact lenses

Causes of Infiltrative Keratitis

- Staph hypersensitivity
- Solution hypersensitivity
- Contact lens hypersensitivity
- Tight Lens Syndrome
- Other conditions

Solution hypersensitivity:

- Chemical reactions to preservatives in contact lens solutions can be associated with sterile infiltrates.
- The most common causes are thimerosal and chlorhexidine.
- May see pseudodendrites on superior cornea. This is referred to Pseudo-superior Limbic Keratoconjunctivitis. These pseudodendrites do not stain deeply with flourescein or have terminal bulbs.
  -- Differentiate from SLK and HSV keratitis. Both are covered in this program.

Work-Up For Contact Lens Related Problems

- Complete case history:
  - Type of contact lens: RGP, soft (EW or DW), PMMA, disposable?
  - Method and frequency of disinfection. Wearing schedule? Homemade saline??
  - Severity and nature of ocular symptoms. Onset of symptoms.
  - When were lenses last worn?
- SLE (RULE OUT AN INFECTION!!)
  - Evaluate fit and condition of lenses. Deposits?
  - If ulcer or infiltrate is present, note size and staining. Note amount of edema. Check anterior chamber for reaction.
  - Flourescein stain to check for epithelial defects.
  - Evert upper lid to check for follicles/papillae.
  - Check IOP.
- Corneal smears if suspect an infectious ulcer. When acanthamoebe keratitis is suspected use Giemsa stain. Otherwise corneal smears should be made with blood, chocolate or Sabouraud's media.

When to suspect an infectious ulcer?

- In one study of untreated corneal infiltrates associated with contact lens wear, Pseudomonas was the most frequent organism cultured and accounted for half of the culture-positive cases.

Etiologies | Treatment

Treatment

- Treatment depends on the etiology. General therapy is presented below, but for specifics refer to the appropriate section. In all cases, it is probably necessary, to at least, temporarily discontinue contact lens wear.
- The importance of early treatment in corneal infections can not be over emphasized. Thus it is safer to overtreat some suspicious infiltrates as infected ulcers pending results of cultures.
- It is beyond the scope of this program to list all treatment plans for various contact lens related problems. However, therapy for the three conditions below can be seen by "clicking" on one with the mouse.

Corneal Ulcer/Infiltrate
Hypersensitivity reaction
Tight lens syndrome

Work-up | Menu

Treatment for Corneal Ulcer/Infiltrates

If no infection is suspected:
- Discontinue contact lens wear
- Eliminate causative agent
- Tobramycin qid (may add Tobramycin ung hs)
- RTC in 1-3 weeks depending on condition.
  (Lenses can be resumed after condition resolves. Dispense new pair.)

If infection is suspected:
- Discontinue contact lens wear
- Consult with ophthalmologist for appropriate management
- Treatment includes fortified antibiotics and cycloplegics.
- No pressure patch.
- Patient is usually evaluated on a daily basis.
- See section on corneal ulcer for more specifics.

Treatment for Solution Hypersensitivity

- Check the solution that the patient is using for thimerosal, chlorhexidine or potasium sorbate.
- Discontinue lens wear.
- Artifical tears (preservative free) 4-6 X daily until SPK resolved.
- Contact lens wear can be resumed once cornea is clear. Consider dispensing a new pair.
- Change to preservative free system.
- Explain proper lens hygiene including importance of thorough rinsing after use of enzymes. Avoid solutions with preservatives.

Treatment for Tight Lens Syndrome

- Discontinue contact lens wear.
- If an anterior chamber reaction is present, treat with cycloplegic.
- Patient will probably notice photophobia, irritation and tearing for the next few days. Infiltrates will probably last for several weeks.
- RTC in 3-5 days for refitting of contact lens. Fit with a flatter base curve.

Corneal Abrasions

Main Menu

- Critical Sign
- Symptoms
- Signs
- Work Up
- Treatment
- Follow up

Critical Signs for Corneal Abrasion

- Presence of an epithelial defect that stains with fluorescein. This may range from total denuding of the corneal surface to a mild punctate keratitis.
- Corneal abrasions may result from overwear of contact lenses, foreign bodies, fingernail scratches, tree branch scratches, chemicals or a host of other causes.

← Main Menu for Corneal Abrasions Symptoms →

Symptoms of Cornea Abrasions

- Pain (generally severe)
- Photophobia
- Foreign-body sensation
- Tearing
- Blepharospasm

- Motion of the eye and blinking generally increases the pain and foreign-body sensation.
- Generally, there is a history of scratching the eye.

← Critical signs Signs →

Signs of Corneal Abrasions

- Epithelial staining defect with fluorescein
  - A vertical or tracing type linear stain is typical.
- Conjunctival injection
- Lid edema may be present
- Corneal edema
- Lacrimation
- Blepharospasms
- IOP can be decreased
- Mild anterior chamber reaction may be present.

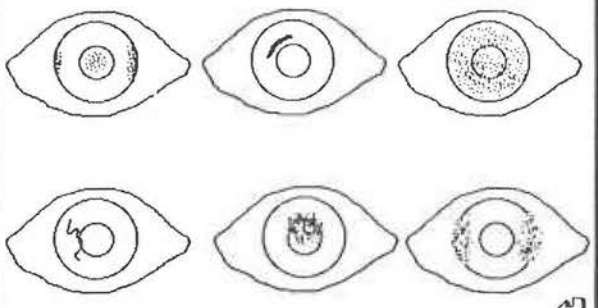
- Contact lens-induced abrasions may take many forms

Corneal Staining Patterns →

← Symptoms Work up →

Common staining patterns with contact lens wear

** Click on cornea for more information**



Work-Up for Corneal Abrasion

- Case History:
  - Generally there is a history of scratching the eye. How? Where and when? Details of accident? Visual status before the accident? What is the offending agent? Does the patient wear contact lenses??

- Visual acuity (with pinhole if < 20/20)
- External Exam:
  - Install a drop of topical anesthetic unless corneal ulcer is suspected.
  - Use bright pen light with oblique illumination (look for shadow on iris)

- Slit-lamp Exam:
  - Check for mild anterior chamber involvement.
  - Use fluorescein and measure size and depth; diagram location and size.
  - Lesions with swollen margins and negative staining should be suspect of viral Etiology.
  - Rule out corneal ulcer, herpes simplex keratitis, contact lens overwear, corneal epithelial dystrophies.

- Upper and lower fornices should be checked to rule out retention of foreign body, Evert of upper lid.

← Signs Treatment →

Treatment of Traumatic Corneal Abrasion

- Obtain ophthalmological consult if stromal involvement or corneal perforation.
- Treatment ranges from patient assurance and no action to topical antibiotics, cycloplegics and patching.
  - -- Lavage (may or may not debride loose epithelial flaps)
  - -- Antibiotic ointment (Gentamicin, Tobramycin , Polymixin B)
  - -- Cycloplegic: Cyclopentolate 1-2% or Homatropine 5% (depends on severity)
  - -- Pressure patch for 24hrs
    - Generally a pressure patch is not applied if the abrasion has a significant risk for infection (scratches from a branch or fingernail).

- Make sure all foreign material has been debrided from the wound.

- Very superficial abrasions may be left untreated or with antibiotic cover. As the abrasion increases in size and depth or as the patient reports discomfort, then patching with an antibiotic cover is indicated. A contact lens is sometimes used as a patch.

- Heat (heating pad or hot water bottle) may be applied throughout the day in half-hour intervals to speed up healing process. Patient should be instructed to remain quiet during the initial 24 hrs. to lessen the chance of disturbing newly formed epithelial cells. Consult patient on healing and RCE

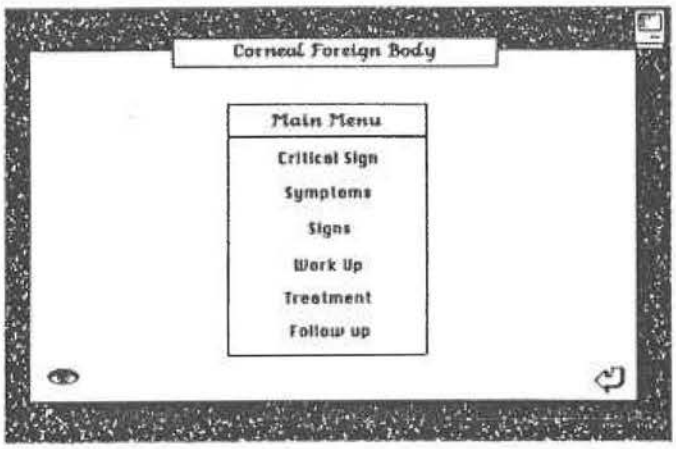
← Work up Follow up →

Follow-Up for Corneal Abrasions

- Re-examination should be daily until epithelium has returned to normal. Large abrasions may take many days to heal. Daily treatment with prophylactic antibiotics, cycloplegics and patching is done until epithelium is healed.

- Many corneas will be adequately repaired within 24 hrs.
- If condition worsens, consult with ophthalmologist.

← Treatment Menu →



Critical Signs for Corneal Foreign Body

- Presence of corneal foreign body, rust ring or both
- Generally, there is an obvious history.
- Some of the more common types of foreign bodies:
  -- metal, bits of rust, cinders, sand, vegetable matter, glass, fiberglass.

Main menu for Corneal Foreign Body
Symptoms

Symptoms of Corneal Foreign Bodies

- Generally there is a history of a foreign body in the eye.
- Foreign body sensation with each blink. Patient's discomfort varies from mild to very severe.
- Tearing
- May have blurred vision
- Photophobia

Critical signs
Signs

Signs of Corneal Foreign Bodies

- Appearance of particle or particles on corneal epitbelium
- Corneal FBs frequently cause track marks, abrasions and ulcers.
- A small infiltrate (Coat's white ring) and corneal edema may surround the foreign body, especially if it has been there > 24 hrs.
- Hemosiderosis (rust ring) may be present if metallic FB
- Conjunctival injection adjacent to limbus closest to the FB
  -- The degree depends upon the material and the time on the cornea.
- SPK (superficial punctate keratopathy)
- Lid edema may be present
- Mild anterior chamber reaction may be present.
- Hypopyon along with an increase in pain if there is a secondary infection developing.

Symptoms
Work-up

Work-up for Corneal Foreign Bodies

- Case history:
  Was the patient wearing protective eyewear? Time of injury? If long standing, look for sequella of secondary reactions.
  -- Did this occur during the course of employment?
  Did the foreign body arise from metal striking metal? Was the object propelled?
  -- If so, look for intraocular foreign body. What type of material?
- Visual acuity with pinhole if < 20/20
- Slit-lamp Exam:
  - Determine epithelial depth (slit lamp optic section)
  - Evert lid and inspect fornices for additional foreign bodies
  - Check for mild anterior chamber involvement.
  - Measure dimensions of infiltrate if present
  - Many times metal foreign bodies will form a rust ring (orange stain)
  - Rule out perforation.
- If injury is due to metal striking metal, dilate and look at retina and vitreous to rule out intraocular foreign body. Also consider x-ray.

Signs
Treatment

Treatment of Corneal Foreign Bodies

- Remove foreign body (using the slit lamp)
  - Apply 2 drops of a topical anesthetic (proparacaine)
  - Remove FB with a FB spud, 25-gauge needle or moistened cotton tipped applicator within the slit lamp beam. (See Principles of Foreign Body Removal from Anterior Segment by Lou Catania for techniques)
  - If multiple FBs, irrigation may remove them easier.
  - **Deep FBs (stromal) should be referred to a specialist**
- Remove rust ring (using slit lamp). Metalic particle will rust within 2-48 hrs.
  - Usually an Algerbrush is effective and will remove the rust ring.
  - If the rust ring is centered in the visual axis and appears deep, it may be safer to leave it and allow the rust to migrate to the surface than attempt to remove it. Consult with ophthalmologist. Do not attempt to remove rust ring if deeper than basement membrane.
- Measure and note size of resultant corneal defect
- Cyclopegic (cyclopentalate 1.0 - 2.0%)
- Antibiotic ointment (erythromycin, Gentamicin)
  - If you suspect an intraocular FB, do not apply ointment. The injury may allow the ointment into the anterior chamber.
- Consider pressure patch for 24 hrs.

Work-up
Contraindicated therapy

Contraindicated Therapy

- No patient with a corneal abrasion or corneal foreign body should be maintained on topical anesthetics.
  -- This will slow down the healing process.
  -- This will ultimately cause the total breakdown of corneal epithelium and stromal, and cause edema and severe pain.
- Do not treat with medication containing topical steroids because it increases the chance of a secondary bacterial, viral or fungal infection.

Treatment
Follow-up

Follow-Up for Corneal Abrasions

- If the resulting corneal defect is small (< 1-2 mm), clean, and noncentral after removal, then:
  -- Remove pressure patch after 24 hrs and treat with topical antibiotics for 3-4 days.
  Examples: Sulfacetamide drops qid or
  Erythromycin ung bid or tid
  -- Follow up PRN
- Follow up in 24 hours to re-evaluate if any of the following are present:
  -- A central or large corneal defect
  -- Mucopurulent discharge and/or infiltrate
  -- Residual rust in the cornea
  -- Anterior chamber reaction

** If there is an infiltrate with a significant anterior chamber reaction, purulent discharge, or extreme redness and pain, then an infection needs to be ruled out and the condition treated more aggressively with antibiotics. Consult with ophthalmologist.

Contraindicated therapy
Menu

Dry Eye

Main Menu

- Anatomy
- Classification
- Symptoms
- Signs
- Critical Signs
- Work Up
- Differential Diagnosis
- Treatment
- Follow Up

Anatomy and Composition of the Tear Film

- The precorneal tear film is basically composed of three layers: 1) a thin, superficial lipid layer; 2) a relatively thick aqueous layer; and 3) an inner mucin layer.
- The superficial lipid layer, which is an oily covering composed of waxy and cholesterol esters, primarily functions to inhibit evaporation of the underlying aqueous layer. It is predominantly secreted by the meibomian glands, with the glands of Zeis performing a minor role.
- The aqueous layer, which lies between the lipid and mucin layers, comprises the vast majority of the tear film thickness. It is secreted primarily by the lacrimal gland, and secondarily by the accessory lacrimal glands of Kraus and Wolfring. It functions to lubricate the conjunctiva and cornea, and to provide cleansing by flushing any debris from the ocular surface.
- The innermost layer of the tear film is the mucin layer which provides a thin hydrophyllic covering to the epithelial surface. Without this coating the corneal surface would repel the aqueous layer, preventing proper spreading of the tear film. The mucin layer is predominantly secreted by the conjunctival goblet cells.

Main Menu | Classification

Classification of Tear Film Abnormalities

- Dry eye syndromes are best divided into five categories. They include:

Choose A Topic Below

- Lipid Abnormalities
- Aqueous Deficiency (KCS)
- Mucin Deficiency
- Lid-surface Abnormalities
- Epitheliopathy

Anatomy | Symptoms

Symptoms of Dry Eye

- Intermittant burning and tearing which is exacerbated by reading, drafts, wind, smoke and fumes
- Grittiness or foreign body sensation
- Itching
- Redness of one or both eyes
- Intermittant blurring of vision - a "film" over the eye
- Photophobia

Classification | Signs

Signs of Dry Eye

- No discharge present but there may be an accumulation of mucoid or lipid buildup in the inferior cul-de-sac
- Excess mucous or debris in the tear film
- Generally a low-grade bulbar hyperemia of angular type
- A nonspecific papillary (palpebral) conjunctivitis
- The lid margin and the inferior tear meniscus may be reduced (0.5mm) or even absent in more severe cases
- Punctate corneal and/or conjunctival staining with rose bengal or NaFl
- Occasional corneal dellen formation
- Positive "tear breakup time" (BUT) - less than 10 seconds on the Schirmer test

Symptoms | Critical Signs

Main Menu for Dry Eye

Critical Signs of Dry Eye

- A scanty tear meniscus seen at the inferior eyelid margin. The meniscus should be at least 1 mm in height with a convex shape
- A decreased tear breakup time (less than 10 seconds)

Signs | Work Up

Work Up for Dry Eye

- SLR using fluorescein stain, examining the tear meniscus and the tear break-up time
- Schirmer testing

Procedure for a tear break-up time:

1. Wet NaFl strip with saline
2. Instill NaFl in the inferior culdesac or on the superior bulbar conjunctiva
3. Do not touch or manipulate the lids
4. Have the patient blink 2 - 3 times
5. Evaluate the NaFl pattern for breakup points after 1 to 2 seconds. A positive breakup time equals random patterns of breakup points in less than 10 seconds but more than 1 second.

Procedure for Schirmer testing:

1. Instill 1 drop of anesthetic in each eye (measures basal secretion only)
2. Place the Schirmer filter paper at the junction of the middle and lateral 1/3 of the lower eyelid for 5 minutes
3. Normal secretion equals wetting of greater than or equal to 10 mm in 5 minutes

Critical Signs | DDH

Differential Diagnosis of Dry Eye

- Herpes simplex keratitis - usually unilateral; the keratitis is often dendritic but may appear similar; the face lesions of rosacea are generally absent
- Trichasis - SPK are typically linear from an eyelash rubbing on the eye
- Blepharitis - presents as erythema, telangiectasias, and crusting of the eyelid margins
- Exposure keratopathy - SPK results from poor eyelid closure with a failure of the lids to cover the entire globe
- Topical drug toxicity - SPK results from drops with preservatives, causing a hypersensitivity reaction
- Ultraviolet burn/photokeratopathy - SPK often seen in welders or from sun lamps
- Contact-lens related problems - SPK from chemical toxicity, tight-lens syndrome, contact-lens overwear, GPC etc.
- Thygeson's superficial punctate keratopathy - bilateral with recurrent SPK in the absence of conjunctival injection
- Foreign body sensation - The SPK are typically linear appearing as fine scratches arranged vertically on the cornea.

Work Up | Treatment

Treatment of Dry Eye

- Local treatment of the dry eye syndrome may aim at increasing the aqueous phase or reducing the outflow of the aqueous phase.

1. Increasing the aqueous phase: Instillation of sodium chloride 0.45%. This treatment "cleanses" the eyes and has few side effects. The only drawback to this method is that it is very short-lived and must be applied frequently.

2. Reducing the outflow of tears: Occlusion of both puncta is required to obtain a good effect. Permanent occlusion by cautery is only indicated in the most desperate cases. Mucomimetics, or artificial tears with methylcellulose (0.5%) or polyvinyl alcohol (1.4%), retain the lacrimal fluid in their meshes, thus retarding the tear flow. The instillation of these agents should be PRN. The only side effect is a plastic-like film that may develop on the palpebral ciliary margins. An alternative to the frequent instillation of a mucomimetic is a drug-release system. A hydroxypropyl cellulose ophthalmic insert (i.e. Lacrisert) can be instilled on the inferior tarsal conjunctiva and has a duration of 8 to 16 hours.

DDx

Follow up

Follow Up for Dry Eye

- If there is no improvement after 2 weeks:

    1. Question the patient on compliance and continue to treat for 2-3 weeks if noncompliant.

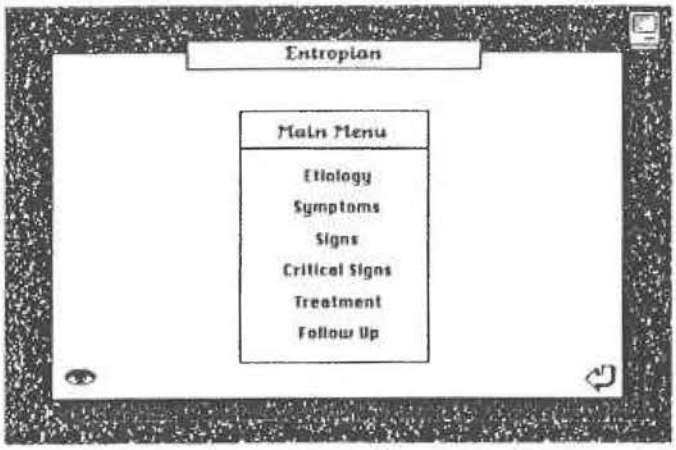
    2. If the patient is compliant, upgrade the treatment.

- If there is improvement both objective and subjective, taper the therapy to a minimal dosage and recheck every 3-6 months or PRN based on the severity.

NOTE: Patients with severe "dry eye" should be discouraged from contact lens wear.

Treatment

Main Menu



Etiology of Entropian

- Involutional (Aging): This usually involves the lower lid and is caused by degenerative changes. With aging, atrophy of the orbital tissues can lead to a relative enophthalmos and a tendency for inward rotation of already attenuated eylid structures.
- Cicatricial: Due to conjunctival scarring in ocular pemphigoid, Steven-Johnsons syndrome, chemical burns, trauma, trachoma, and others.
- Spastic: Due to surgical trauma, ocular irritation, or blepharospasm.
- Congenital: A rare phenomenon that is usually associated with other abnormalities such as tarsal hypoplasia or microophthalmia. It may be confused with epiblepharon (medial lid infolding) which is more common and typically resolves spontaneously.

← Main Menu Symptoms →

Symptoms of Entropian

- Ocular irritation
- Corneal foreign body sensation
- Hyperlacrimation or lid spasms
- Red eye

← Etiology Signs →

Signs of Entropian

- Inferior lid and lashes turned inward, rubbing against the corneal surface
- Inferior (irregular to vertically oriented) foreign body tracking on the cornea
- A spastic lid response upon forced closure of the lids
- Superficial punctate keratitis
- Conjunctival injection

← Symptoms Critical Signs →

Main Menu for Entropian →

Critical Signs for Entropian

- Inward turning of the eyelid margin.

← Signs Treatment →

Treatment of Entropian

1. A surgical procedure is usually indicated

   If surgery is contraindicated or refused:

   (a) Complete epilation
   (b) Low water hydrophilic bandage lenses may help
   (c) An ongoing lubrication for corneal protection and comfort is indicated
   (d) Erythromycin or bacitracin tid for the corneal SPK

2. Everting the eyelid margin away from the globe and taping it in place with adhesive tape may provide temporary relief

← Critical Signs Follow Up →

Follow Up for Entropian

- If the cornea is relatively healthy, the condition does not require urgent attention.
- If the cornea is significantly damaged, aggressive treatment is indicated and follow up should be scheduled more closely until the corneal involvement resolves.

← Treatment Main Menu →

Epidemic Keratoconjunctivitis

Main Menu

- Etiology
- Symptoms
- Signs
- Critical Signs
- Differential Diagnosis
- Treatment
- Follow Up

Etiology of Epidemic Keratoconjunctivitis

- Epidemic keratoconjunctivitis is known to be caused by adenoviruses 8, 19, 21 with the current type 37 predominantly recovered from persons in the United States and Europe with EKC.
- Outbreaks of EKC has been known to occur frequently in outpatient facilities and factory dispensaries.
- The source of the epidemic often goes unrecognized, however, and is frequently attributed to applanation tonometry, slit lamp examination, instillation of eye drops, and in some cases, the practitioner.
- Infected individuals continue to shed the virus for 2 weeks and should be considered infective during this period and encouraged to have limited contact with other individuals.
- The contraction of EKC can be minimized through routine washing of hands between patients and the mechanical wiping and drying of instruments.

Main Menu | Symptoms

Symptoms of Epidemic Keratoconjunctivitis

- A low grade fever
- Foreign body sensation
- Lid swelling
- Blurred vision
- Photophobia
- Watery discharge

Etiology | Signs

Signs of Epidemic Keratoconjunctivitis

- Generally, unilateral at onset
- Marked lid swelling
- Acute follicular conjunctivitis in the lower fornix
- Conjunctival petechial and subconjunctival hemorrhages
- Chemosis
- Edema of the caruncle and semilunar fold
- Preauricular lymphadenopathy
- Pseudomembrane formation is not uncommon in severe cases
- Corneal diffuse SPK
- Discrete elevated epithelial lesions
- Subepithelial infiltrates
- Possible iritis
- Transient vision loss in severe cases

Symptoms | Critical Signs

Main Menu For Epidemic Keratoconjunctivitis

Critical Signs of Epidemic Keratoconjunctivitis

- ** The corneal involvement distinguishes EKC from other forms of adenoviral conjunctivitis
- A diffuse corneal SPK manifesting within the first week of the disease
- Discrete elevated epithelial lesions that stain with fluorescein develop after about 7 days
- Subepithelial opacites begin to form beneath the epithelial opacities between 10 - 14 days.
- The epithelial lesions typically disappear within 4 weeks and the subepithelial opacities normally disappear within 3 - months.

Signs | DDx

Differential Diagnosis of Epidemic Keratoconjunctivitis

1. Viral conjunctivitis - a pinkish purple hyperemia that increases toward the plica. There is typically a fast TBUT; a follicular response; a tearing discharge and enlargement of preauricular node or lymphadenopathy.
2. Allergic conjunctivitis - small "velvety" to "giant" papillary changes on both the upper and lower palpebral conjunctiva with the absence of preauricular lyphadenopathy.
4. Chlamydial conjunctivitis - an acute follicular conjunctivitis with a mucopurulent discharge typically seen in sexually active adults. The definitive diagnosis is made with presence of intracpithelial inclusion bodies apparent in epithelial cells obtained by conjunctival scrapings.
5. Herpes simplex keratitis - usually unilateral; the keratitis is often dendritic but may appear similar; the face lesions of rosacea are generally absent
6. Stevens-Johnson syndrome - a bilateral conjunctivitis with hemorrhagic crusting of the lips and target lesions on the skin which appear as red, central vesicles surrounded by a pale ring which is surrounded by a red ring

Critical Signs | Treatment

Treatment of Epidemic Keratoconjunctivitis

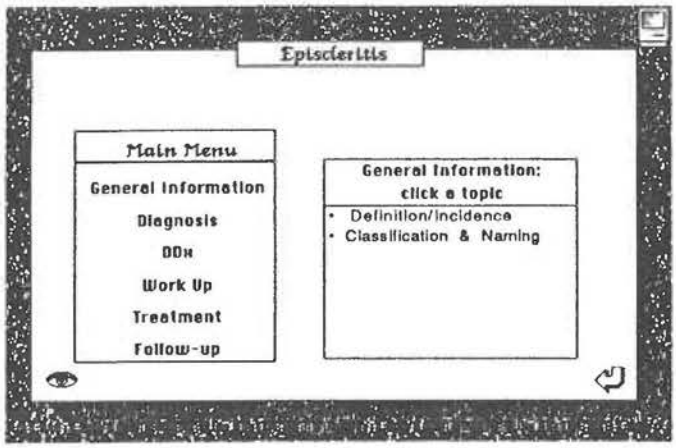
- In most cases, EKC is self-limiting, with an excellent prognosis for complete recovery. Although the follicular conjunctivitis generally runs a course of 7 - 14 days with the corneal involvement subsiding within 3 months, the opacities may persist for up to 2 years. The conjunctival membrane formation can lead to scarring, resulting in a secondary cicatricial entropian.
- During the acute phase of EKC, the treatment is generally supportive with the use of hot compresses, topical astringents, decongestants or lubricants providing relief of the symptoms. Prophylactic use of topical antibiotics is recommended due to the increased incidence of secondary bacterial infections.
- There has been much controversy in the use of topical steroids in the management of EKC. In patients with marked reduction in visual acuities, topical steroids will reduce the opacities and improve vision. However, the subepithelial opacities represent a local immune response to viral infection and, therefore, may suppress the healing process, ultimately prolonging the disease.
- Vidarabine has shown mild benefit, along with Viroptic, as being effective against certain adenoviruses known to cause EKC.

DDx | Follow Up

Follow Up for Epidemic Keratoconjunctivitis

- Due to the self-limiting nature of EKC, the patient should be educated on the duration of the condition with the possibility of aggravation during its course.
- If the treatment regimen consists of topical steroid use, the patient should be monitored more closely with routine intraocular pressure checks due to the nature of the drug. Steroid should be used conservatively with proper tapering upon improvement.
- Due to the contagious nature of EKC, the patient should avoid coming in contact with other people as much as possible.

Treatment | Main Menu



Definition and Incidence of Episcleritis

- Definition:
  Episcleritis is generally a benign inflammation that occurs most often in young adults with a tendency for regression and then recurrence. It can be bilateral or unilateral.
- Incidence:
  - It is relatively common and seems to occur spontaneously.
  - Peak incidence is in the fourth decade and is twice as common in females as in males.

Menu | Classification

Classification of Episcleritis

1. Generally, episcleritis is classified as two types:

- Simple (75% of the cases):
  - Localized discomfort accompanied by variable degrees of lacrimation and photophobia
  - Segmental or diffuse injection and edema of episclera are usually present
  - Grey deposits that appear yellow in red-free light may be present (lymphocytic infiltration)
- Nodular (25% of the cases):
  - Similar in pattern and incidence of simple, but may run a more protracted course
  - A mobile, elevated edematous nodule is present
  - The nodule may be simple or multiple but does not undergo necrosis.
  - Symptoms are generally more intense than with simple.

Definition and Incidence | Critical Signs

Main Menu For Episcleritis

Critical Sign for Episcleritis

- Unilateral or bilateral sectorial redness
  - Injection may be diffuse, but this is uncommon.
  - Episcleral vessels will blanch with topical phenylephrine 2.5% to differentiate from scleral vessels.

Classification | Symptoms

Symptoms of Episcleritis

- Acute onset of redness (may be as little as 1/2 hr.)
  - Generally, redness is sectorial, but can encompass entire anterior portion of the globe.
- Generally, only discomfort is reported (mild pain), but can be absent to severe. Patient may report heat and prickling.
- Pain is usually localized to the eye, but can radiated to the forehead.
  - Eye is rarely tender to the touch.
- Tearing is common, but no ocular discharge.
- Photophobia (mild to moderate)
- Visual acuity is not affected significantly.
- Recurrent episodes of condition are common
- Above symptoms are present in nodular episcleritis but more intense.

Critical Signs | Signs

Signs of Episcleritis

- Sectorial deep injection with apex generally towards limbus (generally unilateral)
- Tenderness over the area of injection
- Episcleral edema
- A nodule which may be moved over the underlying sclera may be present. (nodular episcleritis)

Cornea is rarely affected

Symptoms | Etiology

Etiologies of Episcleritis

Below is a list of some of the systemic causes reported in the literature:

- Idiopathic (most common cause)
- Herpes zoster opthalmicus
- Reumatoid arthritis
- Gout
- Syphilis
- Collagen vascular diseases
  - Polyarteritis nodosa
  - Systemic lupus erythematosus
  - Wegner's granulomatosis
- Trauma
- Giant cell arteritis
- Sarcoidosis
- Tuberculosis
- Thyrotoxicosis

Signs | Rule out

Differential Diagnosis Episcleritis

- Common forms of conjunctivitis:
  - Viral (burning , follicles, preauricular lymphadenopathy)
  - Allergic (allergic Hx, itching, white stringy mucoid discharge)
  - Bacterial (bright red injection, purulent discharge)
- Phlyctenular keratoconjunctivitis
- Scleritis (severe pain, diffuse injection)
- Acne rosacea (chronic recurring corneal irritation, skin lesions)

Etiology | Work up

Work-Up for Episcleritis

- Case History:
  Investigate medical history
  - rash, arthritis, venoreal disease, recent medical history, medical problems
- Visual acuity
- External Exam:
  Look for bluish hue of scleritis in natural light (to rule out scleritis)
- Slit-lamp Exam:
  Determine depth of injected vessels
  - anesthetize with proparacaine and use a cotton-tipped applicator to move conjunctival vessels.

  Check for anterior chamber involvement (absent in simple form) and IOP.
  Check for presence of nodule?
- 1 gt of 2.5% phenylephrine in affected eye should blanch episcleral vessels.
- Refer for diagnostic tests if case history suggests an underlying etiology or if recurrent.

Rule out | Treatment

Treatment for Simple Episcleritis

- Most cases resolve within 3 weeks without complication, and treatment is often not required. An associated uveitis (7%), may be present. Intraocular inflammation should be excluded.

MILD (to relieve symptoms):
- Artificial tears (Refresh) qid , cold packs , topical vasoconstrictor

MODERATE TO SEVERE:
- Same steps as above plus steroid if indicated
- Topical steroid (prednisolone 1% tid or qid) often will relieve the discomfort. It is rare that more frequent topical steroid treatment is needed.

VERY SEVERE CASES:
- Oral nonsteroidal antiinflammatory drugs if topical steroids do not provide relief
  - Ibuprofen 200-600 mg po 3-4X/day or
  - Aspirin 325-1000 mg po 3-4 X/day with food and/or antacids

Possible Complications | Tx for Nodular Episcleritis

Work up | Follow-up

Follow-Up for Episcleritis

- Normal course is usually 10 - 21 days with or without treatment.
- Check weekly if patient is on topical steroids until symptoms have resolved. Also need to check IOP. Once symptoms are resolved, taper steroids.
- If patient is on artificial tears or vasoconstrictor/antihistamines, patient need not be seen for several weeks unless the condition worsens or is still bothering them.
- Be sure to inform patient that episcleritis may recur in the same or fellow eye from a 3 month to 3 year period.
- If more than 3 recurrences, recommend systemic medical workup.

Treatment | Menu

Exposure Keratopathy

Main Menu

- Etiology/Presentation
- Symptoms
- Signs
- Critical Signs
- Work Up
- Differential Diagnosis
- Treatment
- Follow Up

Etiology and Presentation of Exposure Keratopathy

Exposure keratopathy presents clinically with corneal desiccation most notable in the inferior interpalpebral area of the cornea and conjunctiva. It can lead to a frank epithelial defect and a noninfiltrated ulceration.

Common causes are listed below:

- Seventh nerve palsy (orbicularis oculi weakness, e.g. Bell's palsy.)
- Eyelid deformity:
  a. Ectropian or eyelid scarring from trauma
  b. Chemical burn
  c. Herpes Zoster Ophthalmicus
- Exophthalmus
- Nocturnal lagophthalmos (failure to close eyes during sleep)
- Proptosis (e.g. due to an orbital process, such as Grave's disease)
- Postptosis repair or postblepharoplasty procedures

Main Menu | Symptoms

Symptoms of Exposure Keratopathy

- Ocular irritation
- Burning
- Foreign body sensation
- Redness of the eyes

** The symptoms are much worse in the morning due to the length of time that the cornea has been exposed.

Etiology | Signs

Signs of Exposure Keratopathy

- Conjunctival injection
- Corneal infiltrate or ulcer
- Eyelid deformity
- Abnormal eyelid closure with a routine blink, however, many patients are able to force a complete closure of the lids.
- Superficial punctate keratitis in a band region where the cornea has been chronically exposed
- Possible corneal anesthesia
- Epithelial erosion that is greater in the morning

Symptoms | Critical Signs

Main Menu for Exposure Keratopathy

Critical Signs for Exposure Keratopathy

- Inadequate blinking which ultimately leads to a "dry eye"
- SPK in lower one-third of cornea appearing as a horizontal band

Signs | Work Up

Work Up for Exposure Keratopathy

1. Evaluate eyelid closure and the extent of corneal exposure.
2. Slit lamp examination with fluorescein dye to evaluate the tear film and corneal integrity. Look for signs of secondary infection (corneal infiltrate, anterior chamber reaction, or severe conjunctival injection).
3. Rule out: tear dysfunction or an epithelial basement membrane disorder

Critical Signs | DDx

Differential Diagnosis of Exposure Keratopathy

- Herpes simplex keratitis - usually unilateral; the keratitis is often dendritic but may appear similar; the face lesions of rosacea are generally absent
- Dry eye syndrome - SPK results from a poor tear lake or a decreased TBUT
- Blepharitis - presents as erythema, telangiectasias, and crusting of the eyelid margins
- Exposure keratopathy - SPK results from poor eyelid closure with a failure of the lids to cover the entire globe
- Topical drug toxicity - SPK results from drops with preservatives, causing a hypersensitivity reaction
- Ultraviolet burn/photokeratopathy - SPK often seen in welders or from sun lamps
- Contact-lens related problems - SPK from chemical toxicity, tight-lens syndrome, contact-lens overwear, GPC etc.
- Thygeson's superficial punctate keratopathy - bilateral with recurrent SPK in the absense of conjunctival injection
- Foreign body sensation - the SPK are typically linear appearing as fine scratches arranged vertically on the cornea.
- Trichasis - SPK are typically linear from an eyelash rubbing on the eye .

Work Up | Treatment

Treatment of Exposure Keratopathy

1. Correct the underlying disorder
2. Artificial tears (Refresh tears q1-6 hrs)
3. Lubricating ointment (Refresh PM qhs - qid)
4. If severe, tape the eyelids closed at night in conjunction with ointment
5. Application of an HW or disposable soft CL with frequent instillation of lubrication
6. Humidification of the air
7. Swimmers or protective goggles to maintain a moist chamber for the eyes
6. If medical therapy fails, surgical intervention may be needed:
   a. Eyelid reconstruction
   b. Tarsorrhaphy
   c. Orbital decompression for proptosis
   d. Conjunctival flap

DDx | Follow Up

Follow Up for Exposure Keratopathy

- If corneal ulceration is present - re-evaluate every 1-2 days.
- For less severe corneal pathology - re-evaluate every 7-28 days.
- Manage the secondary corneal involvement every 3 months.

Treatment

Main Menu

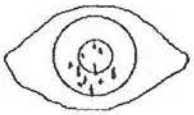
Filamentary Keratopathy

Main Menu

- Critical Sign
- Symptoms
- Signs
- Etiology
- Work Up
- Treatment
- Follow up

Critical Signs for Filamentary Keratopathy

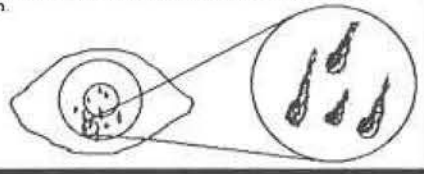
- Short stalks or strands of epithelial cells and mucous which are attached to the anterior surface of the cornea at one end of the strand. Click on cornea for a close up look at the strands.
- Strands usually develop in the lower third of the cornea.
- Strands stain strongly with rose bengal and less strongly with fluorescein.



Main menu for Filamentary Keratopathy → Symptoms

Critical signs for filamentary Keratopathy

- Short stalks or strands of epithelial cells and mucous which are attached to the anterior surface of the cornea at one end of the strand. Click on cornea for a close up look at the strands.
- Strands usually develop in the lower third of the cornea.
- Strands stain strongly with rose bengal and less strongly with fluorescein.



Main menu for Filamentary Keratopathy → Symptoms

Symptoms of Filamentary Keratopathy

- Foreign body sensation or grittiness
- Mild to severe discomfort
- Red eye
- Photophobia
- Blepharospasm and increased blink rate if patient complains of epiphoria and rhinorrhea
- Symptoms range from severe in acute cases to annoying in chronic cases, and onset may be acute, subacute, or insidious.

Critical signs ← → Signs

Other signs of Filamentary Keratopathy

- Conjunctival injection
- Poor tear film
- SPK (superficial punctate keratopathy)
- Small, greyish, subepithelial , granular opacities form beneath the filaments

Symptoms ← → Work-up

Work-Up for Filamentary Keratopathy

- Case History:
  - There are many conditions which can produce filamentary keratopathy (often considered a form of aberrant epithelial healing).
  - Thus, any condition leading to focal epithelial erosions may produce a filamentary keratopathy.
  - Try to ascertain the etiology. → Etiology
- Visual acuity
- Slit-lamp exam:
  - Note size and shape of filaments. Size and shape very widely from 0.5 mm to over 10 mm in length. Initially, they are short and thin and become fatter, longer, and more twisted in time.
  - Strands stain strongly with rose bengal and less strongly with fluorescein.

← Signs → Treatment

Main menu for Filamentary Keratopathy

Common Etiologies of Filamentary Keratopathy

- Dry eye syndrome; in particular keratoconjunctivitis sicca (KCS)
  - -- This is the most common etiology and is found in roughly 15% of patients with rheumatoid disease.
  - -- KCS may also be associated with autoimmune diseases such as Sjogren's.
  - -- The filaments may be distributed diffusely and are often associated with areas that stain with fluorescein. Refer to diagnostic tests in "KCS stack".
- Superior Limbic Keratoconjunctivitis (SLK)
  - -- Superior conjunctival injection and rose bengal staining
  - -- Filaments are often distributed over the superior portion of the cornea.
  - -- See diagnostic tests in "SLK stack".
- Prolonged patching
  - -- Postoperative, corneal abrasions can predispose cornea to filament formation.
- Recurrent corneal erosions
- Following epithelial defects of herpes simplex, herpes zoster or Thygeson's SPK
- Systemic disorders (i.e. diabetes, psoriasis, ectodermal dysplasias)
- Retained foreign body of upper lid
- Trachoma

← Main menu for Filamentary Keratopathy

Treatment of Filamentary Keratopathy

- Initially the eye should be stabilized by eliminating the filaments, with the long term treatment directed towards treating the underlying cause. Keratoconjunctivitis sicca (KCS) and superior limbic keratoconjunctivitis SLK) are the most frequent causes.

Treatment for KCS | Treatment for SLK

- If symptoms are severe or if the above treatment fails, then bandage soft contact lens may be needed. The soft lens protects the epithelium from the lids.
  - Since some bandage lenses have low Dk and limited oxygen permeability, it is recomended that the cornea be evaluated within 24-48 hoours to look for edema or corneal break down. If a lens is needed for several days or weeks, an extended wear lens is recommended.

← Work-up → Follow up

Follow-Up for Filamentary Keratopathy

- Check in 1-4 weeks
- Fortunately, most cases respond to conventional therapy.
  - KCS generally responds well to hypertonic agents and artificial tears.
- For those who do not respond to conventional treatment, may want to try a mucolytic agent (FDA has not approved for ocular use) or a bandage soft contact lens.
  - -- The contact lens usually provides a dramatic clinical improvement, but this is not the best approach for all cases.
- The prevention of further filaments may require long-term use of a tear supplement. Lubrication must be maintained chronically if the underlying condition cannot be eliminated.
- Manage primary condition appropriately and advise of risk of recurrence.

Treatment Menu

Floppy Eyelid Syndrome

Main Menu

- Definition/Incidence
- Signs & Symptoms
- Critical Signs
- Differential Diagnosis
- Work Up
- Treatment
- Follow Up

Definition and Incidence of Floppy Eyelid Syndrome

- Floppy eyelid syndrome refers to the clinical findings of chronic papillary conjunctivitis along with a rubbery, "floppy" eyelid that is easily everted.
- It is an uncommon and frequently unrecognized cause of chronic unilateral or bilateral papillary conjunctivitis.
- The condition is usually bilateral but tends to be worse on the side that is slept on.
- The mechanism is thought to relate to loss of tarsal integrity, causing the eversion of the eyelid during sleep, resulting in the mechanical irritation to the lids and conjunctiva.
- It is commonly seen in middle aged obese men who may complain of a chronic mucoid discharge and spontaneous eversion of the eyelids during sleep.

← Main Menu Signs & Symptoms →

Signs and Symptoms of Floppy Eyelid Syndrome

- A chronically red, irritated eye, often worse upon awakening.
- A mild mucous discharge
- A soft and rubbery tarsal plate
- A superior tarsal papillary conjunctivitis
- Superficial punctate keratitis (SPK)
- The patient is typically obese

NOTE: The symptoms are thought to arise from the spontaneous eversion of the upper eyelid during sleep. The eversion allows the superior palpebral conjunctiva to rub against a pillow or mattress.

← Definition/Incidence Critical Signs →

Main Menu for Floppy Eyelid Syndrome →

Critical signs of Floppy Eyelid Syndrome

- A diffuse papillary conjunctivitis
- An upper eyelid which can be everted easily without the use of a finger or cotton-tipped applicator exerting a counter pressure
- A soft rubbery tarsus that can be folded on itself

← Signs & Symptoms DDx →

Differential Diagnosis:

• Click on a specific disease •

- Vernal conjunctivitis
- Giant papillary conjunctivitis
- Superior limbic keratoconjunctivitis
- Toxic keratoconjunctivitis

Vernal conjunctivitis

Vernal conjunctivitis presents with large conjunctival papillae referred as cobblestone papillae under the upper eyelid. It is generally a seasonal disease and a history of atopic conjunctivitis is sometimes present. A variation to this is limbal vernal which presents with large conjunctival papillae along the limbus. Eversion of the upper eyelid is necessary to make the diagnosis.

close

← Critical Signs Work Up →

Work Up for Floppy Eyelid Syndrome

1. Pull the skin of the upper eyelid toward the patient's forehead and watch to see if the eyelid spontaneously everts or is abnormally lax.
2. Perform a slit-lamp evaluation of the cornea and conjunctiva with fluorescein staining.

← DDx Treatment →

Treatment for Floppy Eyelid Syndrome

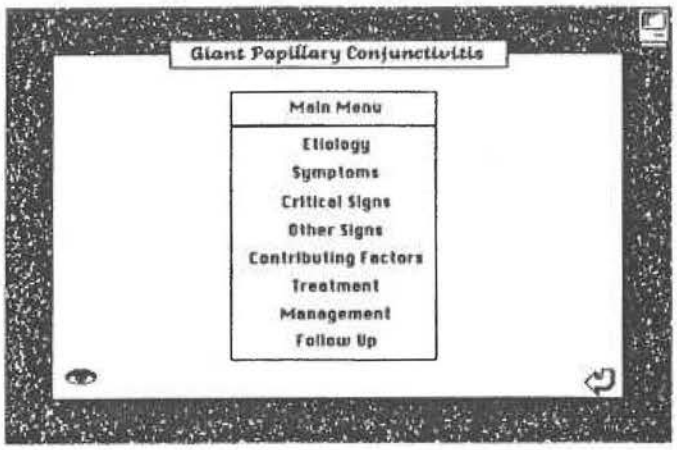
1. Topical antibiotics or lubricants for any mild corneal or conjunctival abnormality (e.g., erythromycin ointment 2-3 X / day for SPK, then Refresh PM ointment qhs when corneal pathology resolves)
2. The eyelids should be taped closed during sleep, or an eye shield can be worn, to prevent the eyelid from rubbing against the pillow or mattress. The patient should be instructed to refrain from sleeping face down on the pillow or mattress.
3. If the case is severe enough, an eye lid tightening surgical procedure can be performed. This is usually the definitive means of treatment.

← Work Up Follow Up →

Follow Up for Floppy Eye Syndrome

The patient should be followed every 2-7 days at first and then every few weeks to months as the condition stabilizes.

← Treatment Main Menu →



Etiology of GPC

- The etiology is not completely understood.
- Giant papillary conjunctivitis is thought to be a specific conjunctival inflammatory reaction to denatured proteins which become adherent to the anterior surface of both rigid and soft contact lenses. There seem to be two possible inflammatory responses:

  (1) Type I - immediate
  (2) Type IV - delayed

- Giant papillary conjunctivitis can also be initiated by mechanical trauma caused by patients rubbing their eyes to relieve the common symptom of itching.

Main Menu | Diagnosis

Symptoms of GPC

- The corresponding symptoms reported by the patient may include:
  - Itching
    - Burning, stinging
    - Foreign body sensation
    - Loose lenses that will not center
      - Mucous discharge (more prominant nasally)
      - Fluctuating vision due to the lens deposits and mucous
      - General lens intolerance (a decreased wear time)
- Obtain a history of the lenses including; details of contact lens use, the age of the lenses, and the cleaning and enzyme regimen used.

Etiology | Critical Signs

Main Menu for GPC

Critical Signs of GPC

- The diagnosis of GPC is made by everting the upper eyelid, especially in patients who wear contact lenses. Carefully inspect the upper palpebral conjunctiva searching for papillary hypertrophy. (?)
- Papillary hypertrophy of the upper tarsal conjunctiva:
  - Macropapillae (0.3 - 1.0 mm in size)
  - Giant papillae (> than 1.0 mm in size)

Diagnosis | Other Signs

Other Signs of GPC

- Contact-lens deposits
- A high riding lens that will not remain centered
- Mild conjunctival injection
- Translucent appearance to the conjunctiva
- Superior punctate corneal staining in more severe cases
- Conjunctival hyperemia and edema
- Ptosis

Associated Allergic Signs
• Chemosis
• Tarsal edema
• Watery discharge
• Sneezing
• Congestion
• Drowziness (drug induced)
• Dry throat, lips and nasal passages
• Nose bleeds

Critical Signs | Contributing Factors

Contributing Factors to GPC

- Lens deposits
- Increased wearing time [e.g., extended wear]
- Older lenses / larger lenses
- Tinted or opaque lenses
- Individual activity of the lens type [e.g., material]
- Atopic history [e.g., excema, allergies, asthma etc.]
- Patients age
- Genetics/predisposition

Other Signs | Treatment

Primary Goal in Treating GPC

- The main goal in the treatment of GPC is to allow continued contact lens wear with the least obstructive therapeutic regimen.
- The treatment requires a systematic approach. The main emphasis in this approach is to stop the inflammatory response in order to make the patient as comfortable as possible.

• Never promise success to the patient!!

Contributing Factors | Treatment

Cromolyn sodium (Opticrom) has been pulled from the market and the chances of it returning are very slim according to the manufactors. However, Levocabastine is a new drug to be used in the treatment of allergic conjunctivitis. It has an antihistamine action that is more effective than Cromolyn sodium in stopping redness, itching, tearing and eyelid and conjunctival swelling. It is not yet approved for topical use by the FDA but is expected to receive approval in 1992.

close

Treatment | Management

Management of GPC

- New contact lenses with the same parameters and design.
- Different soft lenses - (e.g., CSI)
- Disposable lenses:

  OPTIONS:

  -- wear the lenses for 2 weeks and then throw them away.
  -- wear the lenses for 1 week and then throw them away.
  -- clean the lenses every night, enzyme them every 3 days, and then throw them away after 1 week.

- Rigid gas-permeable lenses:

  ADVANTAGES:

  -- less surface to attract deposits
  -- better edges
  -- easier to care for

Treatment | Follow Up

Follow Up for GPC

The patient should be evaluated every 2 - 4 weeks noting their progress. Once the symptoms have been extinguished, slowly taper the use the cromolyn sodium.

NOTE: GPC can also result secondarily to an exposed suture of an ocular prosthesis or post cataract surgery. If this is the case, the suture should be removed and then treated with Opticrom 2% qid until the symptoms have resolved.

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close

Management | Main Menu

Herpes Simplex Keratitis

Main Menu

- General Information
- Diagnosis
- DDx
- Work Up
- Treatment
- Follow-up

General Information: click a topic

- Definition/Incidence
- Primary/Secondary Infection
- When to Consult?

Definition and Incidence of Herpes Simplex Keratitis

- A dendritic,epithelial keratitis or ulceration is normally produced as an acute or chronic disease by infection of herpes simplex virus type 1 (HSV I). HSV -2 can be the cause --> check history. Acute follicular conjunctivitis and skin lesions are commonly present.
- HSV keratitis remains the leading cause of corneal blindness in the U.S. (responsible for more than 1.5 million cases per year).
- Primary infection occurs in 70-80% of the population between the ages of 2-5, and by the age of 15, 90% of the population is infected systemically.
- Infection usually results from contact with infected individuals by mostly saliva and mouth contact, but also from active skin lesions.

Menu | Primary vs Secondary

Primary vs Secondary Infection

- Primary HSV keratitis usually is found in infants and young children, and is rare in adults, although incidence is increasing with increasing HSV-2 infections.
- In the recurrent cases, there is often a history of previous attacks. This is helpful in making a diagnosis.
- Several of the aggravating or inciting factors are as follows:
  - sunlight, trauma, extreme heat or cold, fever, steroids, infectious disease, surgery and epilation.
- This program concentrates mainly on the treatment of the primary infection and suggests consultation for the recurrent form, especially if keratitis is not limited to the epithelium.

Definition and Incidence | When to consult

When to Consult??

- Anytime there is a stromal, disciform or interstitial keratitis, the patient should be under ophthalmological care. If the keratitis is severe, then a corneal specialist should be consulted.
- Primary care in most cases, is adequate if it is a primary HSV infection or a mild to moderate recurrent HSV infection that is restricted to the epithelium.

Primary vs Secondary Form | Critical Signs

Main Menu for HSV Keratitis

Critical Signs for Herpes Simplex Keratitis:

- Single or multiple dendritic ulcers are pathognomonic for HSV keratitis. However, it may present in many forms making it a challenge to diagnose and treat.

Primary Ocular Herpes

- Usually presents as an acute follicular keratoconjunctivitis.
- Regional lymphadenitis with or without a vesicular blepharitis or cutaneous involvement.

Recurrent Ocular Herpes

- May occur as one or a combination of the following:
  - Epithelial infectious ulcers
  - Epithelial trophic ulcers
  - Stromal interstitial keratitis
  - Stromal immune disciform keratitis

When to Consult | Symptoms

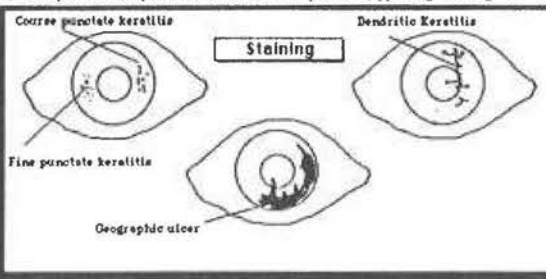
Symptoms of Primary Herpes Simplex Keratitis

PRIMARY INFECTION (uncommon in first 6 months of life):

- Generally infants and young children (ages 5-15)
- Symptoms usually appear 2-12 days after initial contact with infected person. They include:
  - Mild malaise and fever.
  - Lid edema may be present.
  - Red eye, FB sensation and tearing are generally unilateral and rarely severe, but the eye may be sharply painful during the first attack.
  - Skin lesions are common. Chief concern may be the skin lesions next to the eye.
  - May also report a mild photophobia and burning irritation.
  - Generally all ocular symptoms are unilateral.
- Within 2 weeks, 50% of patients will develop corneal lesions (epithelial)
  - FB, photophobia and blurred vision are common.

Critical Signs | Signs | Symptoms of Recurrent Infection

- A fine to course punctate superficial keratitis may be present in the first attack. Fine punctate keratitis--> Course punctate keratitis--> Dendritic keratitis --> Geographic
- Initially, lesions are confined to the epithelium. However, the anterior stroma will inevitably be involved.
- Often, multiple or subepithelial infiltrates are present (appearing within 1-2 weeks).



Signs of Recurrent Infection

RECURRENT HSV KERATITIS
- Almost always associated with a stromal or interstitial form.

- Always unilateral
- Follicular conjunctivitis
- Moderate to severe bulbar hyperemia with occasional conjunctival hemorrhages
- Ipsilateral preauricular lymph node may be slightly enlarged and tender.
- Tearing (serous) discharge (quick tear break up time)
- Dendritic keratitis (more common) or metaherpetic (trophic) keratopathy
- Corneal hypothesia
- Neurotrophic ulcer
- Corneal stromal disease
  - necrotizing interstitial keratitis
  - disciform keratitis

Corneal Lesions | Work-up | Symptoms | Signs for Primary Infection

DDx for Herpes Simplex Keratitis

Rule out the following:

- Herpes Zoster Ophthalmicus
- PCF
- EKC
- Chlamydia
- Recurrent corneal erosion
- Bacterial infections.
- Contact lens related pseudodendrites

All of the above are covered in this program.

Signs | Work up

Work Up for Herpes Simplex Keratitis

- History:
  - Has the patient had any previous episodes?
  - History of a corneal abrasion?
  - Contact lens wearer? What type? What care system?
  - Has the patient recently been on topical or systemic steroids?
  - Any previous nasal, oral or genital sores?
  - Immune deficiency state?
- Visual acuities (pinhole if < 20/20)
- External examination:
  - Skin lesions typically involve the lids and periorbital area. If present, note size and location. Initially, these consist of vesicles which rapidly form superficial crusts and then heal without scarring.

DDx | More

Clinical Features of Primary Herpes Simplex Infection | Menu

Confirmatory Lab Tests if Diagnosis is in Doubt

Work Up for Herpes Simplex Keratitis

- Slit lamp examination with IOP measurements.
  - Look for an acute follicular conjunctivitis.
  - Epithelial and/or stromal involvement?
  - Dendrites? Ulcerative or infiltrative?
    - An ulcerative dendrite will be depressed versus an infiltrative dendrite which is raised. Fluoreacein will undermine a ulcerative dendrite versus a shedding effect over a infiltrative dendrited.
  - Interstitial or disciform keratitis?
  - Anterior chamber reaction? Check before using NaFl.
- Test for corneal hypoesthesia.
  - Hypoesthesia develops and increases with each recurrent attack.
  - Use a separate wisp for each eye.

General Tx

Clinical features of Primary Herpes Simplex Infection | Menu

Confirmatory Lab tests if diagnosis is in doubt

General Treatment for HSV Keratitis

- No steroids in an epithelial keratitis!!
- Skin lesions can be treated in a variety of different ways:
  - mildly abraded with washcloth and treated with Acyclovir ung.
  - alcohol scrubs tid for 14-21 days
  - antibiotic ung (bacitracin or erythromycin)

Tx Skin lesions

- Antiviral therapy (Viroptic) for keratitis/conjunctivitis
  - needs to be tapered due to toxicity but continue several days after epithelial healing. If ineffective after 1 week, switch to another.

Tx of Keratitis

- Supportive systemic therapy - ad lib (aspirin, ibuprofin etc.)
- Hot compresses (ad lib)
- Cycloplegia if indicated (anterior chamber reaction)
- Co-manage with pediatrician or primary physician if primary HSV keratitis
- In recurrent cases consult with ophthalmologist.

Tx for Neurotrophic Ulcer | Tx for Stromal Involvement

Work up | Follow-up

Treatment of Eyelid/Skin Involvement

- Warm compresses to skin lesions tid for 7-14 days
- If eyelid margin is involved:
  - Trifluorothmidine (Viroptic) 1% drops 5X per day or
  - Vidaribine (Vira-A) ointment 5X per day
  - Treat for 7-14 days until resolution.
- Antibiotic ointment if lesions become infected (bacitracin or erythromycin)
- Topical acyclovir ointment tid for 7-14 days
  - Acyclovir is expensive and has not been proven effective. It is not approved for ophthalmic use.

Follow-up

Treatment of Corneal Epithelial Disease

- Antiviral agent:
  - Trifluorothmidine (Viroptic) 1 gt 1% 9X/day or
  - Vidaribine (Vira-A) 3% ointment 5X per day
  - ** Treat for 7-14 days until resolution **
- Cycloplegic (Scopolamine 0.25% tid) if ant. chamber reaction is present.
- If patient is on steroids, have them tapered since this is an epithelial disease.
- Consider gentle debridement of infected epithelium.
  - This may increase the risk for spreading the virus.

Technique for Debridement

Follow-up

Treatment of Neurotrophic Ulcer

- Ocular lubricants for mild punctate epithelial staining: Artificial tears (Cellufresh drops) q 2 hours and Artificial tear ointment (Refresh PM) qhs.
- If a small corneal epithelial defect is present then use erythromycin ointment and pressure patch for 24 hrs. (then continue qid for 4 days or until resolved)
- If corneal ulcer is present, refer to ophthalmologist or corneal specialist.

Follow-up

Treatment of Corneal Stromal Disease

- Patient should be under ophthalmological care.

If mild case

- Cycloplegic if anterior chamber reaction

If severe and/or central, then in addition to cycloplegic:

- Antiviral
  Trifluorothmidine (Viroptic) 1 gt 1% drops tid
- Topical steroids
  Prednisolone acetate 1% qid
  *** Contraindicated in corneal epithelial disease***
- A corneal transplant may be required.

Follow-up

Follow up for Herpes Simplex Keratitis

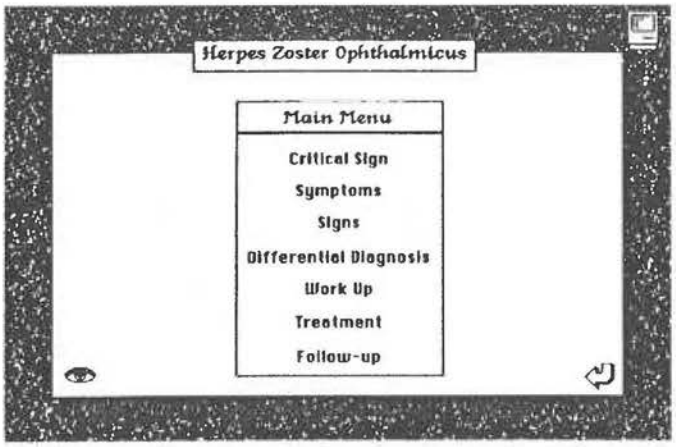
- Patient should be examined in 2-3 days to evaluate response to therapy. Recheck every 3 days until cornea is clear and every 5 days until skin lesions resolve.
- Evaluate the following:
  - Size of epithelial defect and ulcer
  - Corneal thickness and depth to which ulcer is involved. If stroma is involved, it is best to get corneal specialist consultation.
  - Anterior chamber reaction and IOP
- Antiviral medications for corneal dendrites and geographic ulcers should be continued 5-9X daily for 10-14 days. 90% of epithelial dendrites heal within 14 days or less without scarring.
- Topical steroids, if used for stromal disease, are tapered slowly over months to years.
- Prophylactic antiviral agents are used tid. No antiviral is needed when steroid is given once a day or less.

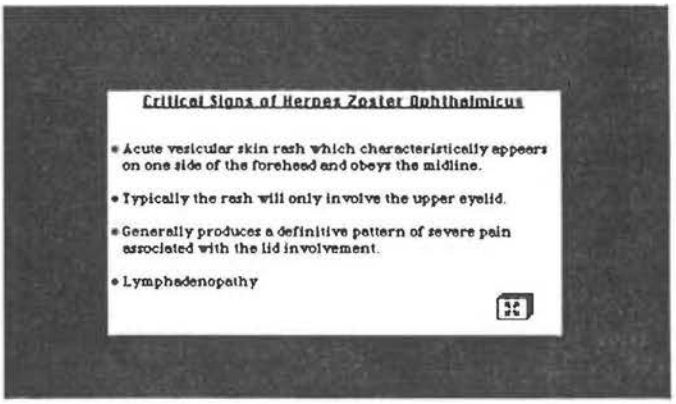
General Tx More

Follow up for Primary Herpes Simplex Keratitis

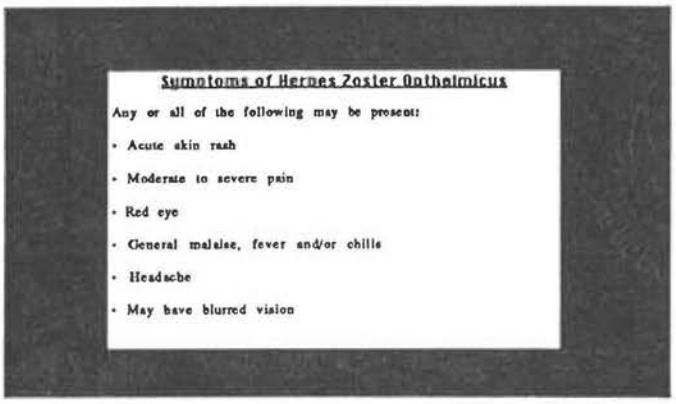
- With prolonged treatment, the antivirals can produce a punctate keratopathy, retardation of epithelial healing, superficial stromal opacification, follicular conjunctivitis, or lacrimal punctal occlusion.
- If epithelial defects do not resolve after several weeks, suspect neurotrophic ulcer or antiviral toxicity. Consult!
  - Generally at this point, antivirals will be stopped.
- Be aware of complications:
  - Bacterial or fungal infection
  - Secondary preseptal cellulitis
  - Stromal involvement
- Advise patient or patient's parents on recurrence risks
- Routine checks or PRN

Menu

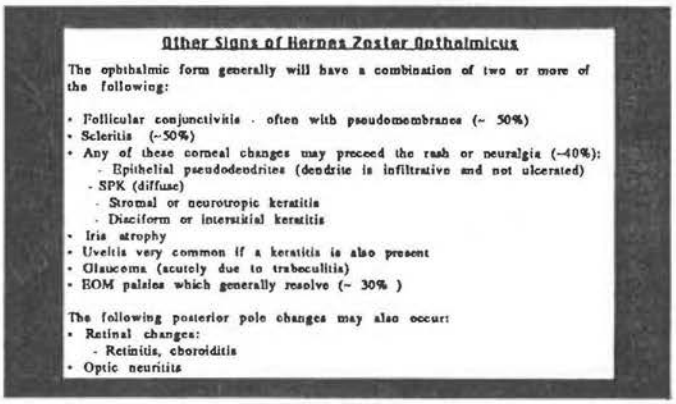




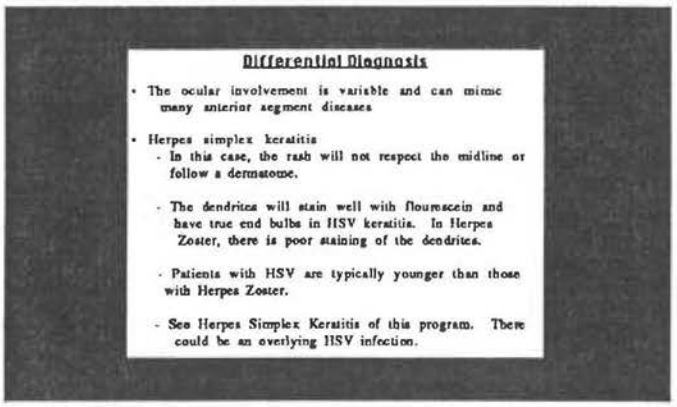
Main Menu for Herpes Zoster Ophthalmicus
Symptoms



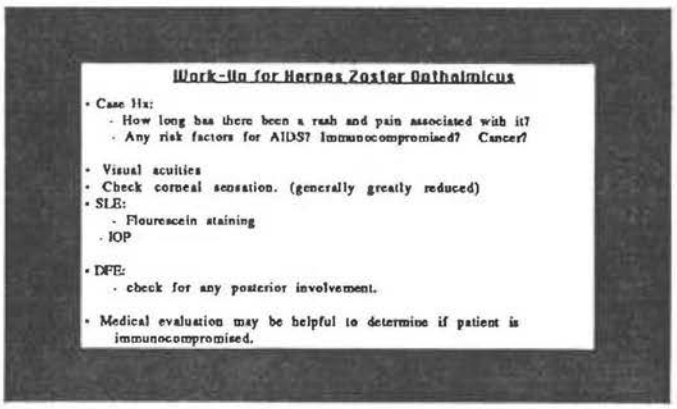
Critical Signs
Signs



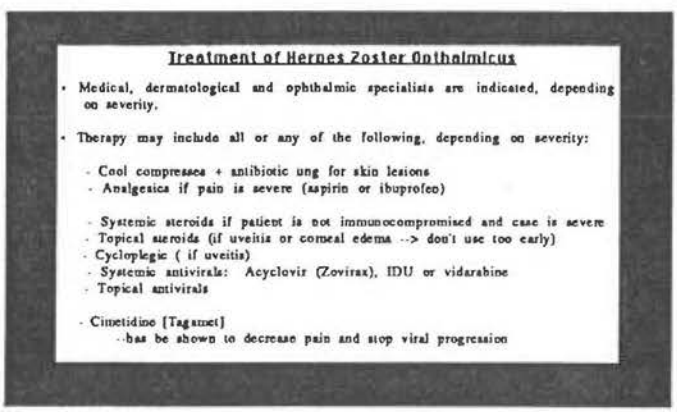
Symptoms
DDx



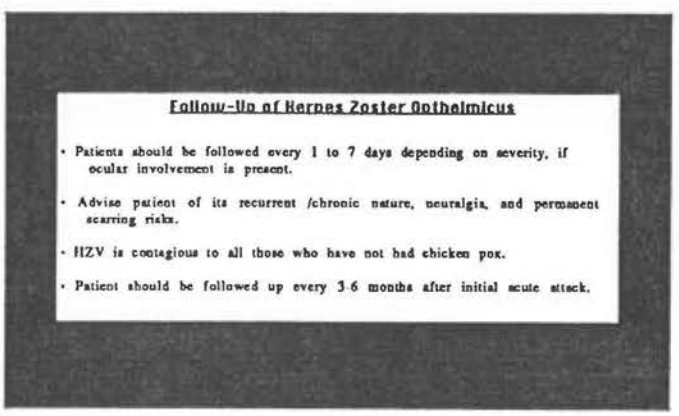
Signs
Work-up



DDx
Treatment



Work-up
Follow-up



Treatment
Menu

Hyperacute Conjunctivitis

Main Menu

- Etiology
- Signs & Symptoms
- Critical Signs
- Work Up
- Differential Diagnosis
- Treatment
- Follow Up

Etiology of Hyperacute Conjunctivitis

Hyperacute conjunctivitis is generally caused by a gram negative diplococci of the Neisseria species. These are aggressively invading bacteria that can produce a severe conjunctivitis that is often bilateral. Often occurring in the child, adolescent, and adult, the conjunctivitis can start as a routine mucopurulent conjunctivitis that can rapidly evolve into a severe inflammation with copius exudate and marked chemosis and lid edema. This clinical appearance requires laboratory confirmation, hospitalization and immediate therapy.

← Main Menu Signs & Symptoms →

Signs and Symptoms of Hyperacute Conjunctivitis

- Copius mucopurulent discharge
  -- generally accumulates in the lower cul-de-sac and overflows at the inner canthus
- Intermittant blurring of vision secondary to the copius discharge
- Conjunctival hemorrhages
  -- these can range from petechiae (small dots) to larger areas of gross sub-conjunctival blood
- Conjunctival papillae
- Chemosis
- True or pseudomembranes may develop in the fornices and/or on the palpebral conjunctiva
- Foreign body sensation
- Lid edema and erythema
- Tenderness of the globe presenting as a throbbing pain
- Frequent follicles and preauricular lymphadenopathy or enlargement may occur, mimicking a viral presentation

← Etiology Critical Signs →

Main Menu for Hyperacte Conjunctivitis →

Critical signs of Hyperacute Conjunctivitis

- Copius mucopurulent discharge

  -- generally accumulates in the lower cul-de-sac and overflows at the inner canthus

- Intermittant blurring of vision secondary to the copius discharge.

← Signs & Symptoms Work Up →

Work Up for Hyperacute Conjunctivitis

- Labortatory workup is indicated:

  -- Conjunctival scrapings for culture and sensitivities: (blood agar, chocolate agar [37°, 10% CO2] and Thayer Martin plate)
  -- immediate Gram stain.

- Always consider the risk of corneal invasion of hyperacute bacteria through an intact cornea.

← Critical Signs DDx →

Differential Diagnosis of Hyperacute Conjunctivitis

- Possible organisms to consider:

1. Streptococcus pneumoniae - usually bilateral, often hemorrhagic, and associated with preseptal cellulitis.
2. Hemophilus influenzae - common in children with a predisposed medical Hx.
3. Neisseria gonorrhoeae - presents as rapid proliferation, extremely purulent discharge, and a positive venereal history.
4. Pseudomonas - often secondary to injury and usually corneal involvement

- Differential consideration for hypersute bacterial conjunctivitis can be accomplished fairly accurately by age alone (rule of fives).

Onset	Possible Organism
0 to 5 days	Gonnococcus
5 days to 5 weeks	Chlamydia
5 weeks to 5 years	Streptococcus or Hemophilus influenzae
5 years and older	Staphylococcus

← Work Up Treatment →

Treatment of Hyperacute Conjunctivitis

- Initiated if the results of the gram stain show gram negative intracellular diplococci or there is a high suspicion of a clinical gonococcus infection. The therapeautic regimen is as follows:

  1. Obtain conjunctival cultures and scrapings. It is very important to institute treatment prior to obtaining the culture results.
  2. Irrigation of the eye with saline qid until the discharge is eliminated.
  3. Topical Bacitracin or Tetracycline ointment qid until resolution.

- If the conjunctival cultures confirm Neisseria species, procede with the following:

  3. Hospitalization is advised and systemic therapy is recommended.
  4. Systemic therapy for the adult consists of three forms:

     - Aqueous crystal penicillin G, 4.8 million units IM in divided doses accompanied by one dose of 1 gm probenecid PO
     - Spectinomycin, 4 gm at one visit, in two divided doses given IM
     - Tetracycline hydrochloride, 1.5 gm IM followed by 500 mg PO qid for 14 days. This mode of treatment is used if the patient is sensitive to penicillin.

← DDx Follow Up →

Follow up for Hyperacute Conjunctivitis

- Reschedule in 24 to 48 hours on the basis of the presenting severity.
- Adjust the initial broad spectrum therapy to a specific drug upon receipt of the laboratory results.
- If the condition is stable or improving within 24 to 48 hours, continue therapy at moderating dosages for a minimum of 10 to 14 days.
- If the condition is unstable or worsening within 24 to 48 hours, adjust the medications, increase the dosages, add oral medication and reconsider the diagnosis.
- Monitor the cornea carefully for any signs of bacterial keratitis.
- Upon resolution of the condition, advise and educate the patient on prevention.

← Treatment Main Menu →

Ocular Pemphigoid

Main Menu

- Definition/Incidence
- Symptoms
- Ocular Signs
- Systemic Signs
- Critical Signs
- Work Up
- Differential Diagnosis
- Treatment
- Follow Up

Definition and Incidence of Ocular Pemphigoid

- Ocular pemphigoid is a subepithelial bullous disease of the aged affecting the mucous membranes leading to shrinkage, scarring, and adhesions. When the conjunctiva is involved, normal tissue is replaced by scar tissue (cicatrization).
- A history of trauma combined with the observation of persistant dry spots on the cornea should alert the clinician of a possible mucin deficiency.
- Ocular pemphigoid may begin with a typical dry eye complaint in the elderly patient. The continual conjunctival shrinkage and scarring can lead to symblepharon formation with an entropian and trichiasis, lagophthalmos and exposure dermatitis, and the inability to elevate the eyes.
- The incidence of ocular pemphigoid is very low (1 in every 20,000 patients). Woman are affected more than men in a ratio of 7:3. The average age at presentation is over 60 years and there is no racial predelection.

Main Menu | Symptoms

Symptoms of Ocular Pemphigoid

- Insidious onset of redness
- Foreign body sensation
- Photophobia
- Tearing

Etiology | Ocular Signs

Ocular Signs of Ocular Pemphigoid

- Superficial punctate keratitis
- Secondary bacterial conjunctivitis
- Corneal ulcers
- Increased intraocular pressure
- Poor tear film
- Entropian
- Trichiasis
- Corneal opacification with pannus and keratinization
- Recurrent corneal erosions
- Corneal neovascularization
- Pseudopterygia
- Obliteration of the fornices and restriction of ocular motility

Symptoms | Systemic Signs

Systemic Signs of Ocular Pemphigoid

- Mucous membrane vesicles of the nose, oral cavity, pharynx, larynx, esophagus, anus, vagina, or urethra
- Denuded epithelium and scarring which can lead to strictures of the esophagus, anus, vagina, or urethra
- A desquamative gingivitis in the mouth is common
- Vesicles and bullae may also be noted on the skin with erythematous plaques or scars near the affected mucous membranes

Ocular Signs | Critical Signs

Main Menu for Ocular Pemphigoid

Critical Signs for Ocular Pemphigoid

- Inferior symblepharon - linear folds of conjunctiva connecting the palpebral conjunctiva of the lower eyelid to the inferior bulbar conjunctiva.

Systemic Signs | Work Up

Work Up for Ocular Pemphigoid

- History: Is the patient on any chronic topical medications?
  Has there been an acute onset of illness in the past?
- Skin and mucous membrane (especially the mouth) examination.
- Slit lamp examination, especially looking for inferior symblepharon. Pull down the patients lower eyelid and have them look up.
- Check the intraocular pressure.
- Dermatology; ear nose and throat; gastrointestinal; and pulmonary consults, if needed.
- Gram's stain and culture of the conjunctiva if a secondary bacterial infection is suspected.
- Consider a conjunctival biopsy for immunofluorescence studies.

Critical Signs | DDx

Differential Diagnosis of Ocular Pemphigoid

1. Stevens-Johnson syndrome - Usually presents as an acute onset of redness often accompanied by fever and malaise. The ocular involvement is similar to that of ocular pemphigoid, presenting with symblepharon and entropian with trichiasis. The differentiating sign with Stevens-Johnson syndrome is that the lips are typically swollen and crusted, and "target lesions" of the skin (red centers surrounded by a pale zone) are often found. Stevens Johnson sydrome is also a self-limiting condition in which the conjunctival shrinkage and symblepharon is static, unlike the chronic progressive course of ocular pemphigoid.
2. Membranous conjunctivitis - usually adenovirus or beta-hemolytic streptococcus that can occur with or without scarring. Symblepharon can follow with severe presentations.
3. Severe chemical burn - chemical burns can generally be elicited through an extensive case history, however, the signs are; epithelial defects ranging from scattered SPK to focal epithelial loss to sloughing of the entire epithelium.
4. Chronic topical medication - e.g., epinephrine, pilocarpine or antiviral agents.

Work Up | Treatment

Treatment of Ocular Pemphigoid

- As in any tear - film deficiency, the treatment of mucin - deficient dry eye with ocular pemphigoid is mainly with artificial tears (e.g., Cellufresh 4 - 10 X / day). Vitamin A drops are extremely beneficial in the promotion of epitbelial growth and differentiation. The main goal is to maintain patient comfort rather than attempting to halt the progression of the disorder. Frequent instillation of an artificial tear ointment at bedtime may be particularly useful in managing mild cases.
- In addition to the basic disease process, secondary bacterial infections (blepharitis) may complicate the clinical problem. In such cases, lid scrubs followed by antibiotic ointments (bacitracin tid) have shown to be effective.
- Timolol has been shown to exacerbate ocular pemphigoid. It is recommended that increases in the IOP levels should be managed with carbonic anhydrase inhibitors.
- Entropian and trichiasis can be corrected with surgical methods in their early stages but special care needs to be taken not to further shorten the already shrunken conjunctiva.
- Systemic steroids (prednisolone 60 mg po q day) for preventing acute exacerbations.

DDH Follow Up

Follow Up for Ocular Pemphigoid

- Every 1 - 2 weeks in acute exacerbations.
- Every 1 - 3 months during remissions.

Treatment Main Menu

Ocular Rosacea

Main Menu

- Definition
- Symptoms
- Signs
- Critical Signs
- Differential Diagnosis
- Work Up
- Treatment
- Follow Up

Definition of Ocular Rosacea

- Rosacea is a common chronic skin disorder of unknown etiology.
- It is more prevelent in females and usually manifests between the ages of 30 to 50 years. It also tends to be more prevelant in the irish population.
- Characteristically, the facial flush areas (forehead, nose, and cheeks) and the V of the neck are involved.
- The prescence of telangiectasia pustules and rhinophyma is diagnostic of rosacea.

← Main Menu Symptoms →

Symptoms of Ocular Rosacea

- Bilateral chronic ocular irritation
- Redness
- Burning
- Foreign body sensation
- Tearing
- Photophobia

← Definition Signs →

Signs of Ocular Rosacea

- Rhinophyma of the nose often ocurrs in the late stages
- Blepharitis
- Telangiectasias of the eyelid margins with inflammation
- Chalazia
- Meibomianitis or styes are common
- Conjunctival injection / episcleritis
- Punctate epithelial erosions
- Peripheral vascularization
- Subepithelial infiltrates with corneal thinning
- Corneal perforation may occur secondary to the infiltration and thinning
- Iritis

← Symptoms Critical Signs →

Main Menu for Ocular Rosacea →

Critical Signs of Ocular Rosacea

- Telangiectasias of the cheeks, nose or forehead
- Pustules, papules, and/or erythema of the cheeks, nose or forehead
- Superficial or deep corneal neovascularization
- Stromal infiltrates may sometimes be seen as an extension of the corneal neovascularization

← Signs DDx →

Differential Diagnosis of Ocular Rosacea

- Herpes simplex keratitis - usually unilateral; the keratitis is often dendritic but may appear similar; the face lesions of rosacea are generally absent
- Dry eye syndrome - SPK results from a poor tear lake or a decreased TBUT
- Blepharitis - presents as erythema, telangiectasias, and crusting of the eyelid margins
- Exposure keratopathy - SPK results from poor eyelid closure with a failure of the lids to cover the entire globe
- Topical drug toxicity - SPK results from drops with preservatives, causing a hypersensitivity reaction
- Ultraviolet burn/photokeratopathy - SPK often seen in welders or from sun lamps
- Contact-lens related problems - SPK from chemical toxicity, tight-lens syndrome, contact-lens overwear, GPC etc.
- Thygeson's superficial punctate keratopathy - bilateral with recurrent SPK in the absense of conjunctival injection
- Foreign body sensation - the SPK are typically linear appearing as fine scratches arranged vertically on the cornea
- Trichasis - SPK are typically linear from an eyelash rubbing on the eye

← Critical signs Work Up →

Work Up for Ocular Rosacea

- External examination:
  - Look for the characteristic telangiectasias, pustules, papules, and/or erythema of the cheeks, forehead or nose. These findings are often subtle and are best seen in natural lighting conditions.
  - Inspect the eyelids for chalazia.
- Slit lamp examination:
  - Look for telangiectasias of the eyelid margins, evaluate the conjunctiva for any injection, and check the cornea for any neovascularization, especially inferiorly.

← DDx Treatment →

Treatment for Ocular Rosacea

1. Tetracycline 250 mg po qid X 3 - 6 weeks. With patients that exhibit a contra - indication to tetracycline, such as a pregnant or nursing mother, erythromycin of the same dosage and duration may be substituted. Once the relief of symptoms becomes prevelant, slowly taper the dose over a couple of weeks.
2. Some patients may also experience an associated blepharitis which can be treated by antibacterial (e.g., bacitracin-polymixin B) lid scrubs at bedtime.
3. Treatment of chalazia:
   - Warm compresses for 15-20 minutes qid in conjunction with light massage over the lesion
   - For more severe cases, consider a topical antibiotic(e.g., bacitracin or erythromycin ung)
4. If small corneal perforations are present, they may be treated with cyanoacrylate while larger ones may require surgery
5. If the SPK stain with fluorescein, smears, cultures, and/or antibiotic treatment may be necessary

← Work Up Follow Up →

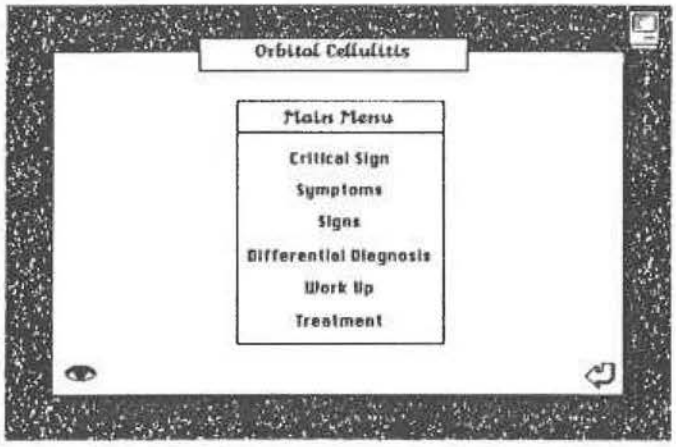
Follow Up for Ocular Rosacea

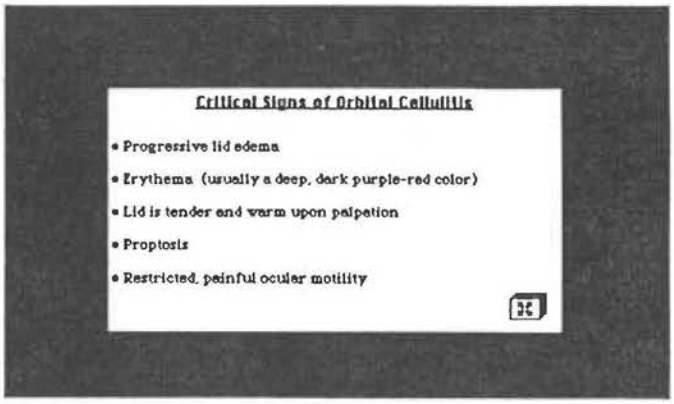
The follow up for Ocular Rosacea is variable depending on the severity of the disease.

- Patients without corneal involvement should be followed every 4-6 weeks.
- Patients demonstrating corneal involvement should be followed more frequently.

** It is important to counsel these patients since this disease may be a chronic condition and some signs of meibomianitis or blepharitis may persist after most of the symptoms are relieved.

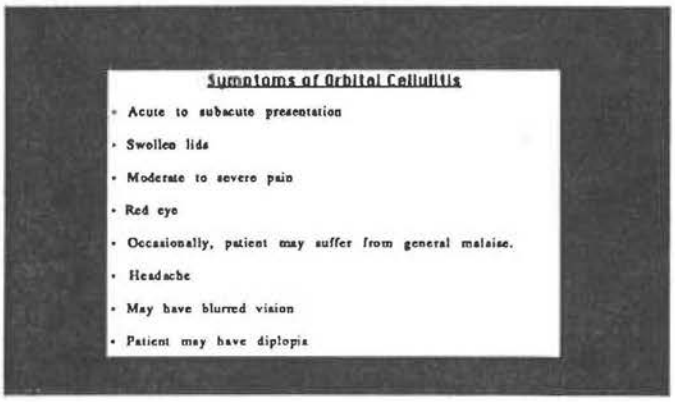
Treatment Main Menu





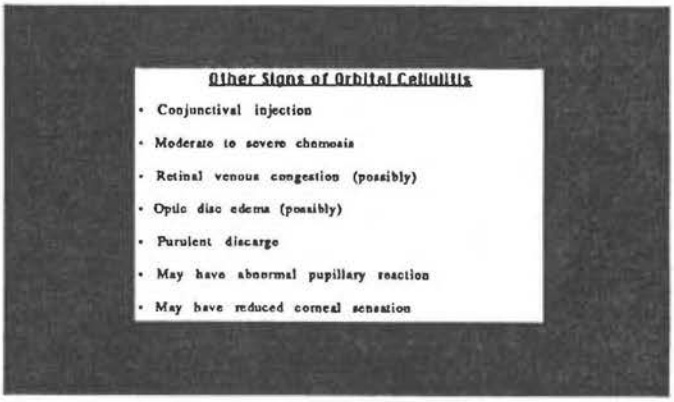
← Main Menu for Orbital Cellulitis

Symptoms →



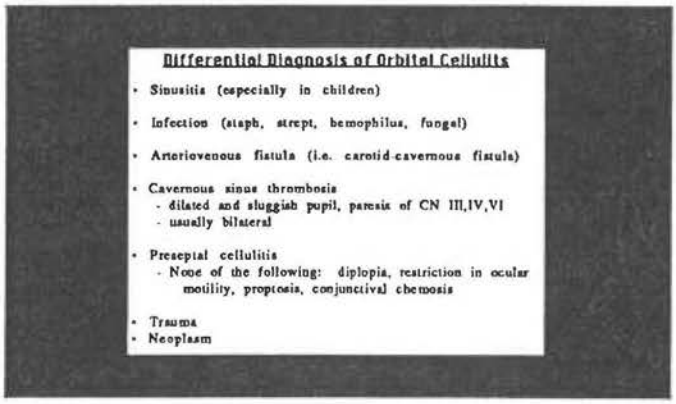
← Critical Signs

Signs →



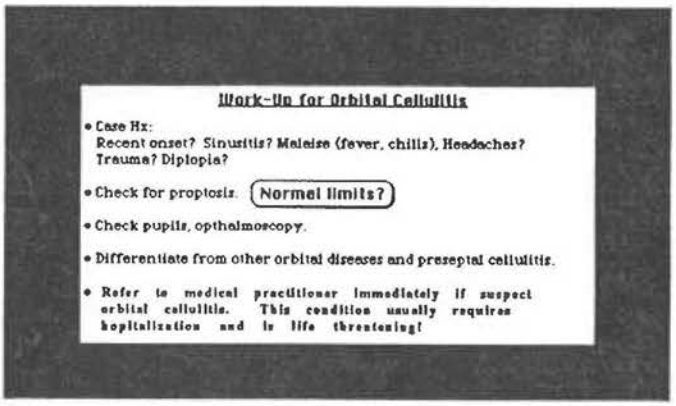
← Symptoms

DDx →



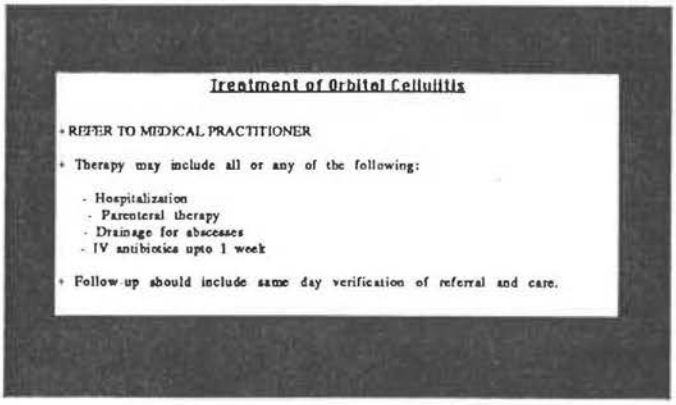
← Signs

Work-up →



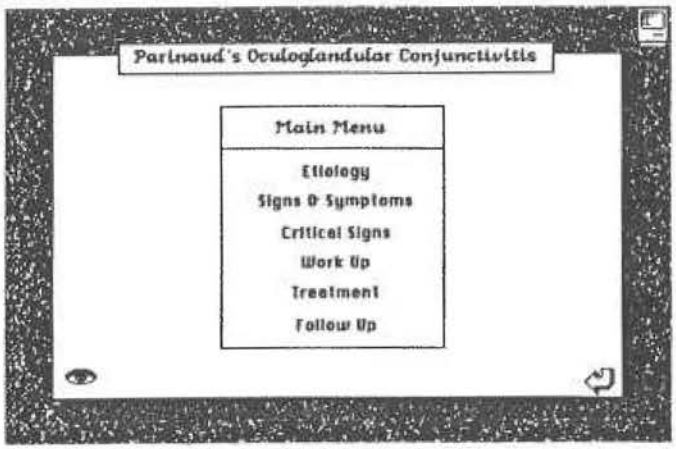
← DDx

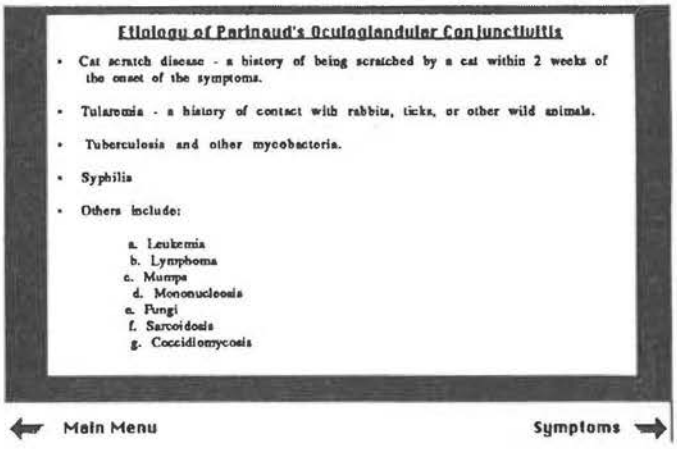
Treatment →

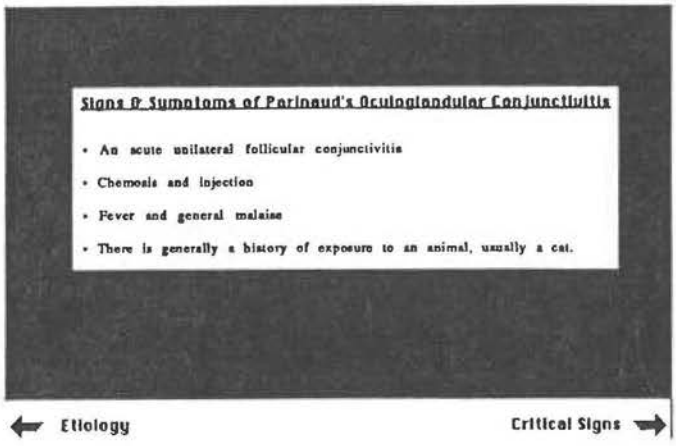


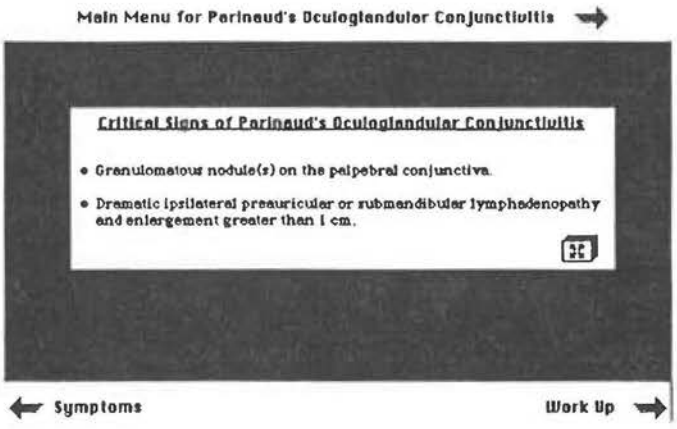
← Work-up

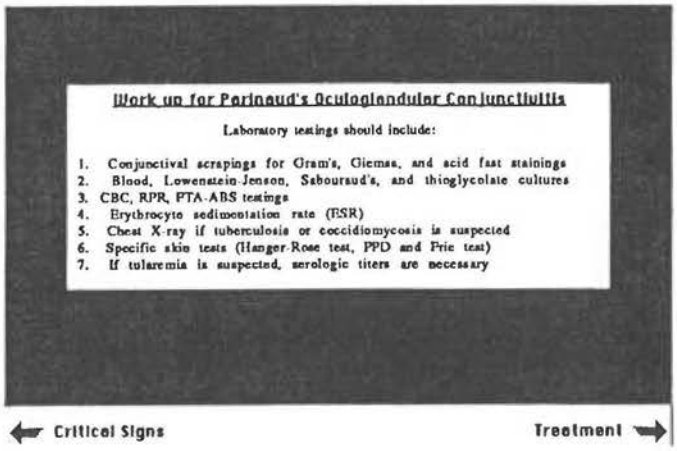
Menu →

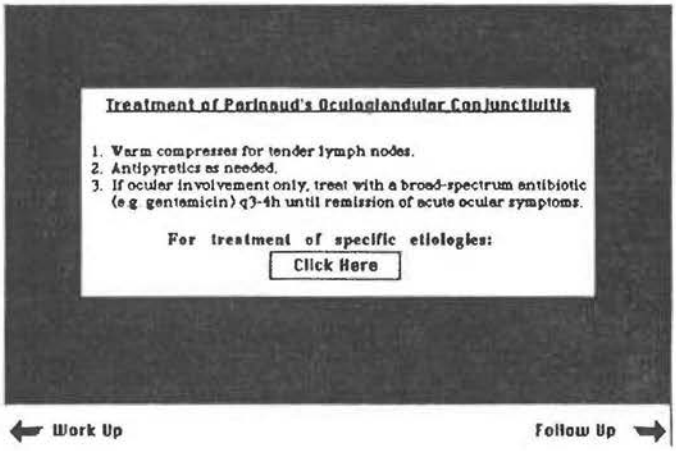

Parinaud's Oculoglandular Conjunctivitis
Main Menu
Etiology
Signs & Symptoms
Critical Signs
Work Up
Treatment
Follow Up

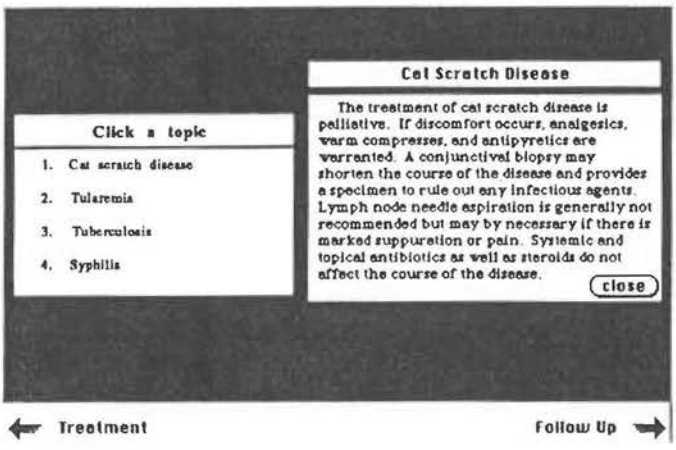

Etiology of Parinaud's Oculoglandular Conjunctivitis
• Cat scratch disease - a history of being scratched by a cat within 2 weeks of the onset of the symptoms.
• Tularemia - a history of contact with rabbits, ticks, or other wild animals.
• Tuberculosis and other mycobacteria.
• Syphilis
• Others include:
a. Leukemia
b. Lymphoma
c. Mumps
d. Mononucleosis
e. Fungi
f. Sarcoidosis
g. Coccidiomycosis
Main Menu
Symptoms

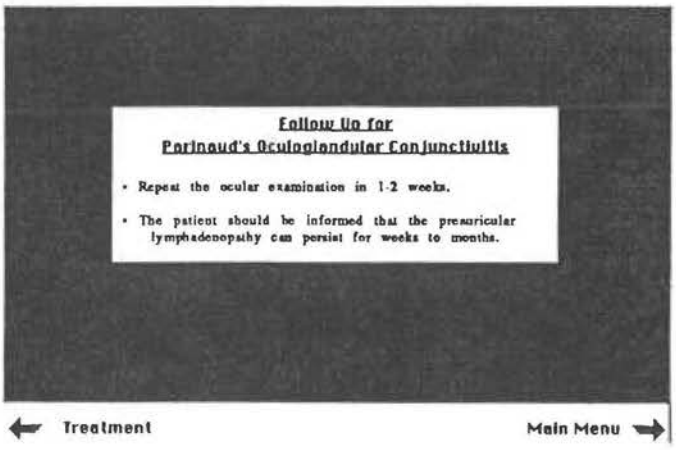

Signs & Symptoms of Parinaud's Oculoglandular Conjunctivitis
• An acute unilateral follicular conjunctivitis
• Chemosis and injection
• Fever and general malaise
• There is generally a history of exposure to an animal, usually a cat.
Etiology
Critical Signs


Main Menu for Parinaud's Oculoglandular Conjunctivitis
Critical Signs of Parinaud's Oculoglandular Conjunctivitis
• Granulomatous nodule(s) on the palpebral conjunctiva.
• Dramatic ipsilateral preauricular or submandibular lymphadenopathy and enlargement greater than 1 cm.
Symptoms
Work Up


Work up for Parinaud's Oculoglandular Conjunctivitis
Laboratory testings should include:
1. Conjunctival scrapings for Gram's, Giemsa, and acid fast stainings
2. Blood, Lowenstein-Jenson, Sabouraud's, and thioglycolate cultures
3. CBC, RPR, FTA-ABS testings
4. Erythrocyte sedimentation rate (ESR)
5. Chest X-ray if tuberculosis or coccidiomycosis is suspected
6. Specific skin tests (Hanger-Rose test, PPD and Prie test)
7. If tularemia is suspected, serologic titers are necessary
Critical Signs
Treatment


Treatment of Parinaud's Oculoglandular Conjunctivitis
1. Warm compresses for tender lymph nodes.
2. Antipyretics as needed.
3. If ocular involvement only, treat with a broad-spectrum antibiotic (e.g. gentamicin) q3-4h until remission of acute ocular symptoms.
For treatment of specific etiologies:
Click Here
Work Up
Follow Up


Click a topic
1. Cat scratch disease
2. Tularemia
3. Tuberculosis
4. Syphilis
Cat Scratch Disease
The treatment of cat scratch disease is palliative. If discomfort occurs, analgesics, warm compresses, and antipyretics are warranted. A conjunctival biopsy may shorten the course of the disease and provides a specimen to rule out any infectious agents. Lymph node needle aspiration is generally not recommended but may by necessary if there is marked suppuration or pain. Systemic and topical antibiotics as well as steroids do not affect the course of the disease.
close
Treatment
Follow Up


Follow Up for
Parinaud's Oculoglandular Conjunctivitis
• Repeat the ocular examination in 1-2 weeks.
• The patient should be informed that the preauricular lymphadenopathy can persist for weeks to months.
Treatment
Main Menu

Phlyctenulosis

Main Menu

- Etiology
- Symptoms
- Critical Signs
- Differential Diagnosis
- Work Up
- Treatment
- Follow Up

Etiology of Phlyctenulosis

Phlyctenular conjunctivitis is an inflammatory condition characterized by the development of a conjunctival or corneal nodule. The presenting nodule is a direct result of a nonspecific delayed hypersensitivity reaction to foreign protein. Phlyctenular conjunctivitis occurs worldwide, typically affecting children, with a higher incidence in females. It is usually unilateral in presentation with an acute or subacute onset of symptoms. Some of the common etiologies are listed below:

- Staphylococcus (often related to blepharitis)
- Tuberculosis
- Acne Rosacea Keratitis
- Other infectious agent elsewhere in the body

Main Menu | Symptoms

Symptoms of Phlyctenulosis

- Itching
- Tearing
- Irritation or pain
- Mild to severe photophobia
- Foreign body sensation
- "Sandy" or "gritty" feeling
- Mucopurulent discharge
- Blepharospasm
- A history of similar episodes

Etiology | Signs

Signs of Phlyctenulosis

- Phlyctenules - localized, superficial, infiltrative reactions.
  - -- characterized by a raised, circumscribed, focal accumulation of infiltrative cells and debris caused by superficial epithelial toxins.
  - -- the lesions are associated with variable degrees of surrounding edema and hyperemia.
  - -- the lesions are characteristically seen on the bulbar conjunctiva or proximal to the limbus. The most common sites for limbal phlyctenules are the inferior circumlimbal areas, especially at the 4 and 8 o'clock positions.
- Bulbar injection - another common sign where the injected vessels create a band of hyperemia that typically points toward the lesion.
  - -- the vessels may overlie the corneal portion of the phlyctenule resulting in superficial pannus.
- Mucopurulent discharge
- SPK - generally in the surrounding areas of the cornea

Symptoms | Critical Signs

Main Menu for Phlyctenulosis

Critical Signs for Phlyctenulosis

- Conjunctival phlyctenule - A small white nodule on the bulbar conjunctiva in the center of a hyperemic area. Often times, this will occur at the limbus.
- Corneal phlyctenule - A small white nodule, initially at the limbus, bordered by dilated blood vessels which migrate toward the center of the cornea producing corneal neovascularization and ulceration. Often bilateral.

Signs | DDx

Differential Diagnosis of Phlyctenulosis

For more information click a topic

- Inflammed pingueculum
- Small pterygium
- Infectious corneal ulcer
- Ocular rosacea
- Herpes simplex keratitis
- Vernal conjunctivitis

Herpes simplex keratitis

Herpes simplex keratitis may present as an SPK, a dendritic keratitis, or a geographic ulcer. The edges of the herpetic lesions are mildly heaped up with swollen epithelial cells which stain with rose bengal while the central ulceration stains well with fluorescein.

Critical Signs | Work Up

Work up for Phlyctenulosis

- History: tuberculosis or any recent infection (e.g. staphylococcal).
- SLE: Inspect the eyelid margins for blepharitis or rosacea.
- PPD (tuberculin skin test).

NOTE: The PPD should be read between 48 and 72 hours after placement. A positive reaction is defined as skin induration (not just erythema) of 10 mm or more.

- Chest X-ray if the PPD is positive or TB is suspected.

DDx | Treatment

Treatment for Phlyctenulosis

- Topical steroids (e.g., prednisolone acetate 1%) in relatively high doses (qid for 3-4 days) to "melt" the infiltrate quickly minimizing the risk of anterior stromal scarring
- Prophylactic antibiotic ung IIS X 5 days (e.g. bacitracin, erythromycin or gentamicin)
- Eyelid hygiene bid to qid
- Artificial tears (e.g. Refresh drops) 4-6 X / day
- If severe photophobia is present a cycloplegic may increase patient comfort.
- If severe blepharitis, use tetracycline 250 mg po qid
  - this therapy is not recommended with pregnant women, nursing mothers, or children younger than 8 years because tetracycline may permanently discolor the teeth
- If PPD or chest x-ray is positive for TB, refer to internist for work up
- If central corneal scarring, penetrating keratoplasty may be of benefit

Work Up | Follow Up

Follow Up care for Phlyctenulosis

- Recheck within 3-5 days depending on the steroid dosage.
- The phlyctenule should show quick response and reversal. If not, increase the dosage of the steroid. Upon improvement, continue the steroid until complete reduction of the raised lesion and total resolution of corneal (anterior stromal) haze is observed. Often, permanent anterior stromal leucomatous hazy scar will persist with or without overlying pannus.
- Maintain antibiotic use for 2-3 weeks after discontinuing the steroid.
- Continue eyelid hygiene indefinately
- Use artificial tears as needed

Treatment

Main Menu

Pterygium

Main Menu

- Symptoms
- Signs
- Critical Signs
- Differential Diagnosis
- Treatment
- Follow Up

Symptoms of a Pterygium

- Ocular irritation
- Conjunctival hyperemia
- Foreign body sensation
- Reduction of vision

← Main Menu Signs →

Signs of a Pterygium

- A thick, fleshy, triangular mass of tissue (yellowish in color) with the apex leading onto the cornea.
- Most commonly found on the nasal aspect of the cornea.
- Frequently a bilateral presentation with varying degree of advancement in each eye.
- A rich surface vascularization resembling pannus.
- Occasionally there is a ferric line (orange-brown) seen at the leading edge known as Stocker's line.
- Associated SPK or a corneal defect are sometimes present.

← Symptoms Critical Signs →

Main Menu For Pterygium →

Critical Signs of a Pterygium

- A wing-shaped fold of fibrovascular tissue arising from the interpalpebral conjunctiva and extending onto the cornea.

← Signs DDx →

Differential Diagnosis of a Pterygium

1. **Conjunctival intraepithelial neoplasia:** Presents as a unilateral jelly-like, velvety or leukoplakic mass, often elevated and vascularized but not in a wing-shaped configuration.
2. **Dermoid:** A congenital white lesion, usually at the inferotemporal limbus. It is occasionally associated with a deformity of the ear - often preauricular skin tags - and/or vertebral skeletal defects [Goldenhar's syndrome].
3. **Pannus:** This commonly presents as blood vessels growing into the cornea, and is often secondary to contact lens wear, trachoma, phlyctenular keratitis, atopic disease, blepharitis, ocular rosacea, herpes keratitis, and others. It is usually at the level of Bowman's membrane with minimal to no elevation.

← Critical Signs Treatment →

Treatment of a Pterygium

1. Protect the eye from sun, dust, and wind (e.g., sunglasses or goggles) as sunlight and chronic irritation are thought to be factors in the growth of pterygiums.
2. Reduce the ocular irritation if present:
   (a) Mild: Artificial tears (e.g., Refresh 4-8 X/day) and/or a mild topical vasoconstrictor (Naphcon A tid - qid)).
   (b) Moderate to severe: A mild topical steroid (e.g., Fluorometholone tid - qid)
3. If a corneal defect is present then apply artificial tears ointment (e.g., Refresh PM) and patch the eye for 24 hours.
4. Surgical removal may be indicated when:
   (a) The lesion is interfering with contact lens wear.
   (b) The patient is experiencing extreme irritation that is unrelieved with the above treatment.
   (c) The pterygium involves the visual axis.

← DDx Follow Up →

Follow Up for a Pterygium

1. Measure and diagram the lesion. Photodocumentation is recommended.
   (a) Recheck first discovered lesions in 6 - 12 months.
   (b) Longstanding lesions are usually stable.
2. If treating with a vasoconstrictor, then the patient should be followed in 2 weeks. The drops may be discontinued when the inflammation has subsided.
3. If treating with a steroid, then follow the patient every 1-2 weeks, monitoring both the inflammation and intraocular pressures. Taper and discontinue the steroid over several days once the inflammation has resolved.

← Treatment Main Menu →

Recurrent Corneal Erosion

Main Menu

- General Information
- Diagnosis
- Work Up
- Treatment/Follow-Up

Diagnosis: click a topic

- Signs/Symptoms
- Critical Signs
- Other signs

General Information About Recurrent Corneal Erosion

- Any damage to the epithelial basement membrane (EBM) or Bowmans layer will disrupt the adhesions between the anterior stroma and the epithelium. Therefore any chronic adverse stimuli with or without trauma to the cornea can result in an unstable corneal epithelium. Corneal dystropies, diabetes and trauma contribute to RCE.

- There is significant value in a positive attitude towards recurrent corneal erosion. It is important to convey to the patient that most of these cases will eventually heal. It will help them feel better about the condition. In severe cases, this is a tough disease to treat.

Menu | Etiologies
Main Menu For Recurrent Corneal Erosion

Etiologies of Recurrent Corneal Erosion

- Anterior corneal dystrophies:

EPITHELIAL BASEMENT MEMBRANE DISORDERS MOST COMMON CAUSE:
  - Map-Dot-Dystrophy/Fingerprint
  - Meesmann's
  - Reis-Buckler's

- Stromal corneal dystrophies:
  - Lattice
  - Macular
  - Granular

The dystrophies tend to be bilateral

- Severe dry eye
- A previous corneal abrasion, chemical/thermal injuries
- Radial K, cataract surgery, retinal surgery
- Diabetic patients
- Bullous keratopathy, Band keratopathy, Salzmann's Nodular Degeneration
- Nocturnal lagophthalmous

Critical Signs for Recurrent Corneal Erosion

- Varies from a corneal abrasion to a localized roughening of the corneal epithelium.

- It is possible that the epithelial changes may resolve within hours of the onset of symptoms so the doctor may not see the abnormality during examination.

General Information | Critical Signs
Etiologies | Signs/Symptoms

Symptoms of Recurrent Corneal Erosion

- Most presentations occur upon wakening
- Recurrent attacks of acute ocular pain or discomfort
- FB sensation ("gritty/sandy" feeling)
- Photophobia
- Tearing -This frequently happens upon awakening or during sleep.
- Burning
- Lid spasms
- Often there is a history of a corneal abrasion in the involved eye.
- May have blurred vision depending on degree and location
- Generally unilateral

Other Signs Associated with Recurrent Corneal Erosion

- Mild case often presents as SPK
- Corneal edema in severe cases (brownish haze --> brawny edema)
- Decreased vision in some cases
- Microcysts, bullae, and filament formation
- Map-dot-fingerprint dystrophy

Work-Up for Recurrent Corneal Erosion

- Case Hx:
  - Recurring nature? Trauma (abrasion)?
  - Recent surgery (Common in postoperative patients -cataracts etc.)?
  - Radial K patients have about a 20% increase in EBM changes.
  - Contact lens wearer? (DW,EW,ROP) Care system used?
  - Medical history (diabetes?) Medications?
  - Family Hx with a similar problem?

- Visual acuity

- SLE:
  - Flourescein staining.
  - Check for corneal dystrophies (generally bilateral and genetic).
  - Check endothelium

Critical signs | Work up
Signs/Symptoms | Treatment

Treatment for Recurrent Corneal Erosion

- Acute mild cases are treated as corneal abrasions.
  - Artificial tears QID + ointment HS (preservative free)
  - Consider antibiotic and cycloplegic depending on presentation.
  - F/U: 1-2 days until epithelium is healed

- Moderate cases (25-50% of cornea involved or patient has had one to two recurrences):
  - Consider debriding epithelium if it is loose or heaped up and not healing.
  - Cycloplegic (Cyclopentolate 2% or Homatropine 2%)
  - Antibiotic ointment (Gentamicin, Tobramycin, Erythromycin)
  - Pressure patch for 24hrs.
    - The above procedure may need to be repeated if large or persistent defects.

  - After epithelial healing is complete:
    - Hypertonic drops q4h for 4-6weeks and hypertonic ointment tid 8-12 weeks
    - Artificial tears to act as a lubricant may help.

  - F/U: Monthly for 4-6 months

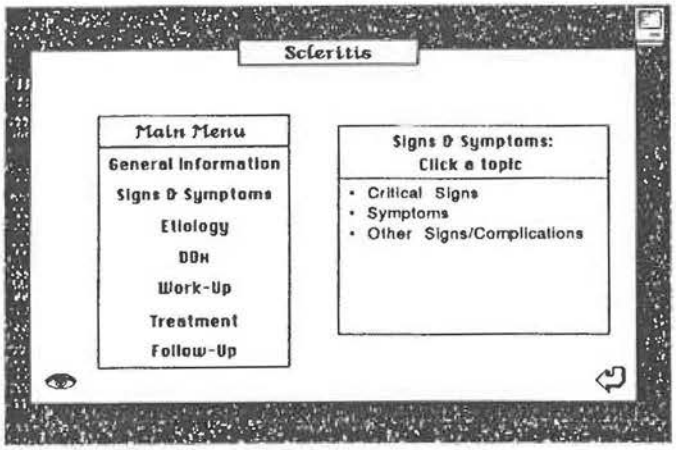
Treatment for Recurrent Corneal Erosion

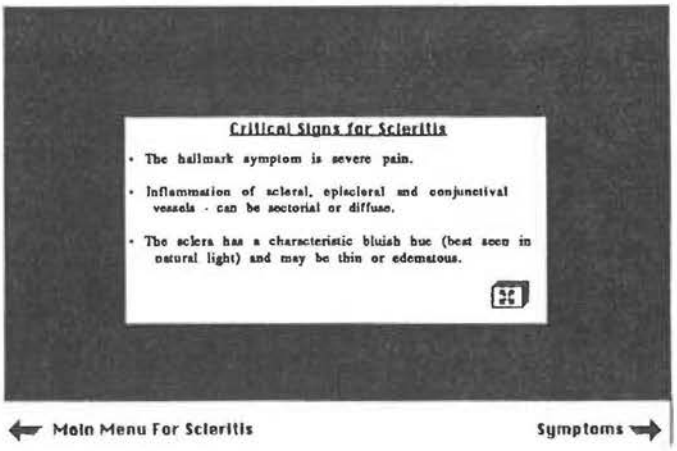
- Severe Cases (> 50% of the cornea involved):
  -Consider debriding the entire area
    --cycloplege + antibiotic ung + patch until healed
  - Consider bilateral patching to totally immobilize the eyes (max 48-72 hrs)
  - Consider a contact lens patch for several months as alternative to bilateral patch.
    -- If inflammatory response, wait until resolved before fitting lens.
    -- The lens should be fit a little loose
    -- The lens should be a thin, high water content lens (low power or plano)
      - B&L collagen degradable lens
    -- Hypertonic drops can be used over the lens (5%NaCl tid/qid)
    -- F/U weekly for the first 3-4 weeks
- In severe, chronically re-eroding corneas, consult with a corneal specialist.

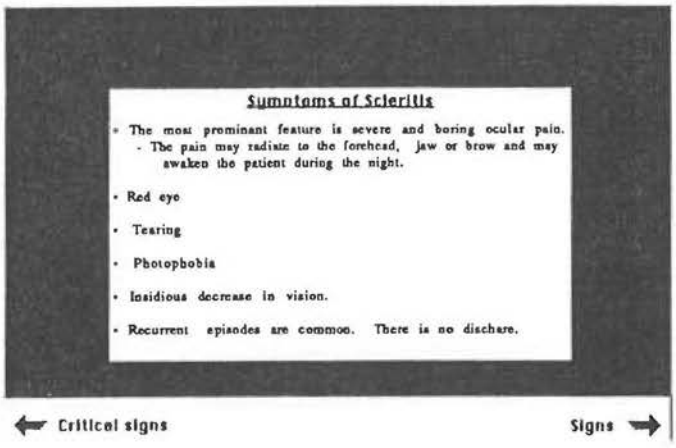
Tx for recalcitrant cases

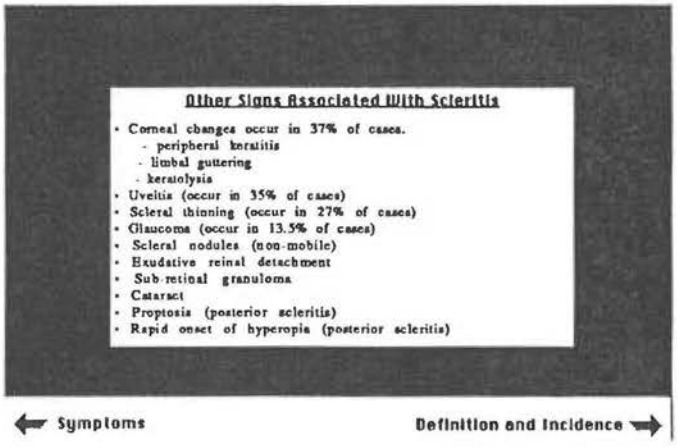
- In all cases, reassure and educate patient of the condition. Vision threatening complications are rare, and most eventually will heal. Discuss chronic nature and importance of long term management and preventive measures.

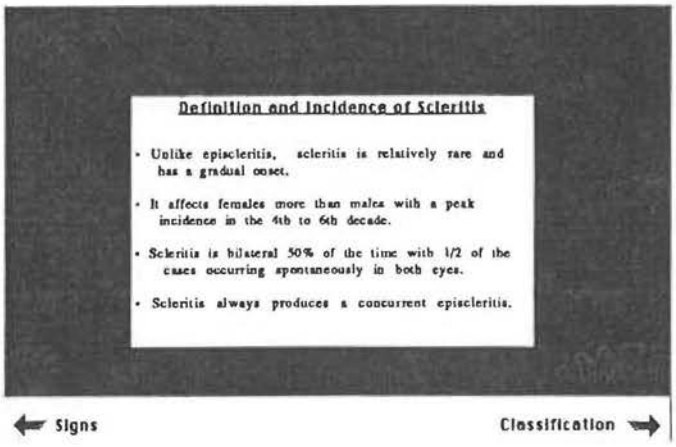
Work up | Treatment for severe cases
Tx for mild/moderate cases | Menu

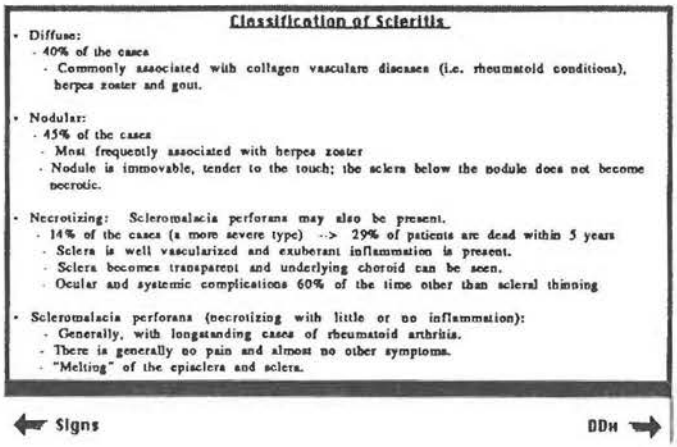

Scleritis
Main Menu
General Information
Signs & Symptoms
Etiology
DDH
Work-Up
Treatment
Follow-Up
Signs & Symptoms:
Click a topic
• Critical Signs
• Symptoms
• Other Signs/Complications

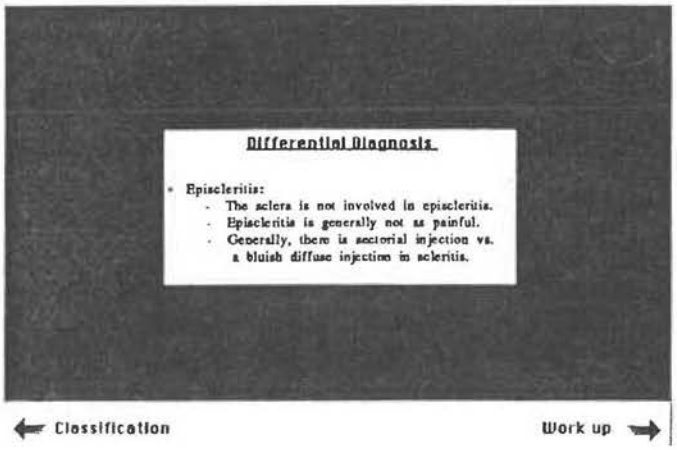

Critical Signs for Scleritis
• The hallmark symptom is severe pain.
• Inflammation of scleral, episcleral and conjunctival vessels - can be sectorial or diffuse.
• The sclera has a characteristic bluish hue (best seen in natural light) and may be thin or edematous.
Main Menu For Scleritis
Symptoms

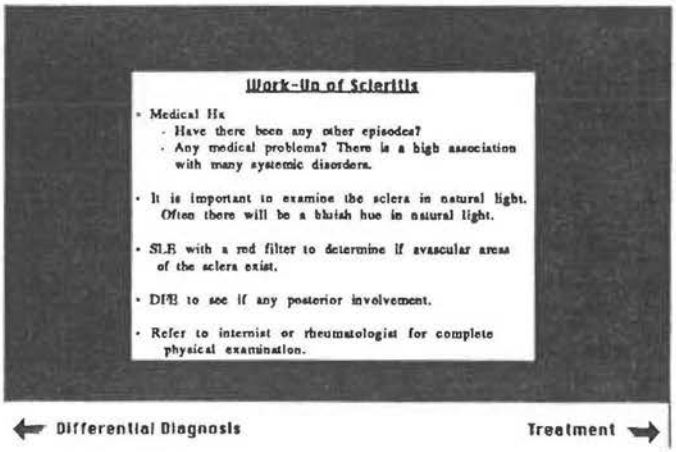

Symptoms of Scleritis
• The most prominant feature is severe and boring ocular pain.
- The pain may radiate to the forehead, jaw or brow and may awaken the patient during the night.
• Red eye
• Tearing
• Photophobia
• Insidious decrease in vision.
• Recurrent episodes are common. There is no discharge.
Critical signs
Signs


Other Signs Associated With Scleritis
• Corneal changes occur in 37% of cases.
- peripheral keratitis
- limbal guttering
- keratolysis
• Uveitis (occur in 35% of cases)
• Scleral thinning (occur in 27% of cases)
• Glaucoma (occur in 13.5% of cases)
• Scleral nodules (non-mobile)
• Exudative reinal detachment
• Sub-retinal granuloma
• Cataract
• Proptosis (posterior scleritis)
• Rapid onset of hyperopia (posterior scleritis)
Symptoms
Definition and Incidence


Definition and Incidence of Scleritis
• Unlike episcleritis, scleritis is relatively rare and has a gradual onset.
• It affects females more than males with a peak incidence in the 4th to 6th decade.
• Scleritis is bilateral 50% of the time with 1/2 of the cases occurring spontaneously in both eyes.
• Scleritis always produces a concurrent episcleritis.
Signs
Classification


Classification of Scleritis
• Diffuse:
- 40% of the cases
- Commonly associated with collagen vascular diseases (i.e. rheumatoid conditions), herpes zoster and gout.
• Nodular:
- 45% of the cases
- Most frequently associated with herpes zoster
- Nodule is immovable, tender to the touch; the sclera below the nodule does not become necrotic.
• Necrotizing: Scleromalacia perforans may also be present.
- 14% of the cases (a more severe type) --> 29% of patients are dead within 5 years
- Sclera is well vascularized and exuberant inflammation is present.
- Sclera becomes transparent and underlying choroid can be seen.
- Ocular and systemic complications 60% of the time other than scleral thinning
• Scleromalacia perforans (necrotizing with little or no inflammation):
- Generally, with longstanding cases of rheumatoid arthritis.
- There is generally no pain and almost no other symptoms.
- "Melting" of the episclera and sclera.
Signs
DDH


Differential Diagnosis
• Episcleritis:
- The sclera is not involved in episcleritis.
- Episcleritis is generally not as painful.
- Generally, there is sectorial injection vs. a bluish diffuse injection in scleritis.
Classification
Work up


Work-Up of Scleritis
• Medical Hx
- Have there been any other episodes?
- Any medical problems? There is a high association with many systemic disorders.
• It is important to examine the sclera in natural light. Often there will be a bluish hue in natural light.
• SLE with a red filter to determine if avascular areas of the sclera exist.
• DFE to see if any posterior involvement.
• Refer to internist or rheumatologist for complete physical examination.
Differential Diagnosis
Treatment

Treatment of Scleritis

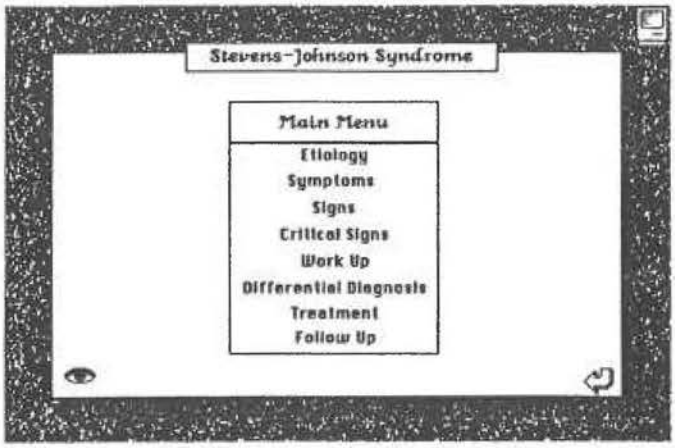
- Refer to Ophthalmologist.
- Steroids
  - Topical steroids increase comfort but many times are not enough. Therefore, systemic steroids are highly recommended especially in a severe or necrotizing scleritis
  - Subconjunctival steroids are not recommended.
- Nonsteroidal anti-inflammatory agents (NSAIA) (e.g. Oxyphenbutazone, Indomethacin, Naproxen)
- Immunosuppressive drugs in severe or unremitting cases.
- Surgery is indicated in some cases.
- No ocular treatment is available for scleromalacia perforans. Refer to rheumatologist.

Work up | Follow up

Follow-up of Scleritis

- Follow-up really depends on the degree of scleritis.
- Generally a decrease in pain indicates a response to the treatment.

Treatment | Menu


Stevens-Johnson Syndrome
Main Menu
Etiology
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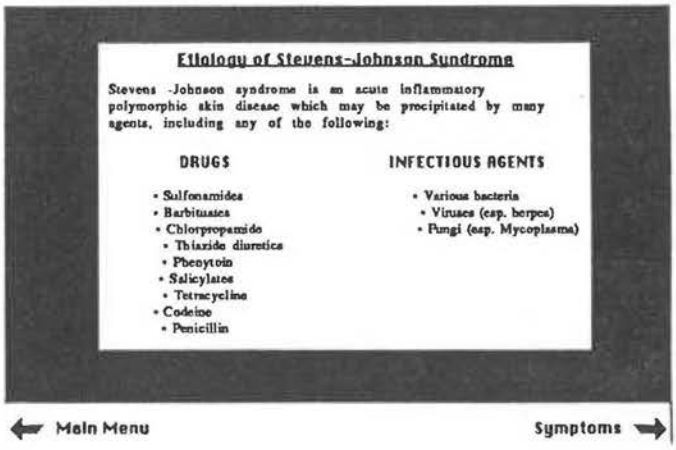
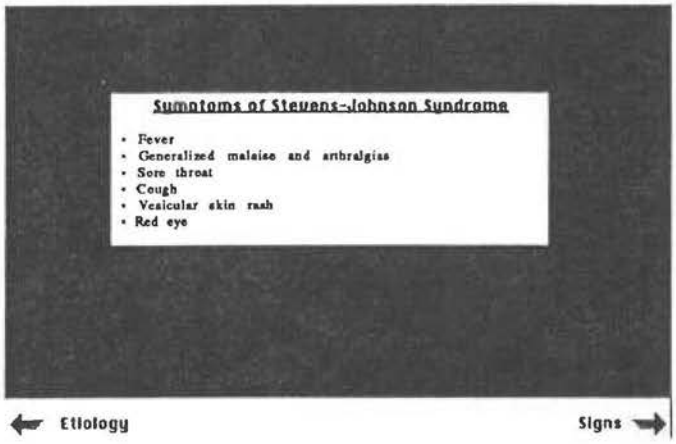
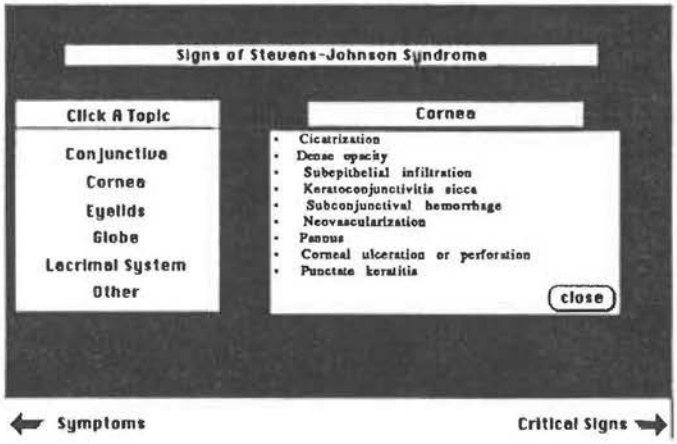
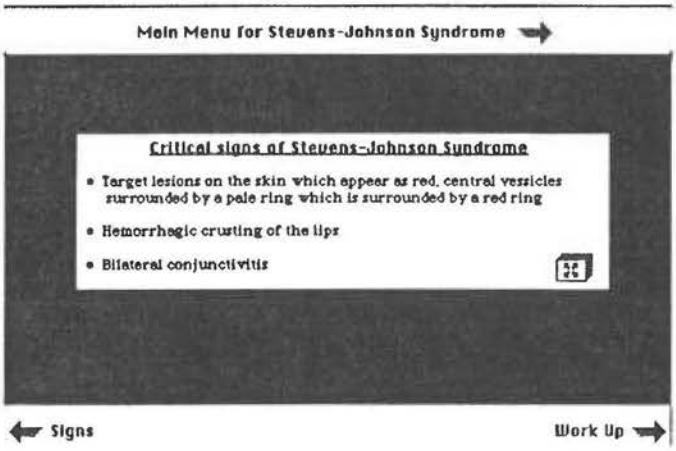
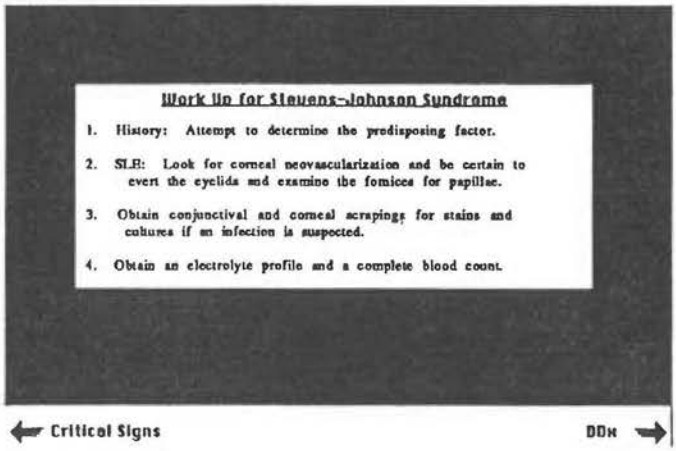
Differential Diagnosis
Treatment
Follow Up

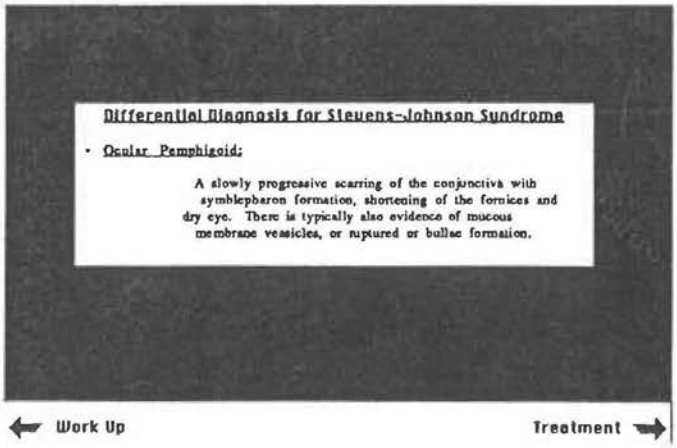

Etiology of Stevens-Johnson Syndrome
Stevens -Johnson syndrome is an acute inflammatory polymorphic skin disease which may be precipitated by many agents, including any of the following:
DRUGS
• Sulfonamides
• Barbiturates
• Chlorpropamide
• Thiazide diuretics
• Phenytoin
• Salicylates
• Tetracycline
• Codeine
• Penicillin
INFECTIOUS AGENTS
• Various bacteria
• Viruses (esp. herpes)
• Fungi (esp. Mycoplasma)
Main Menu
Symptoms

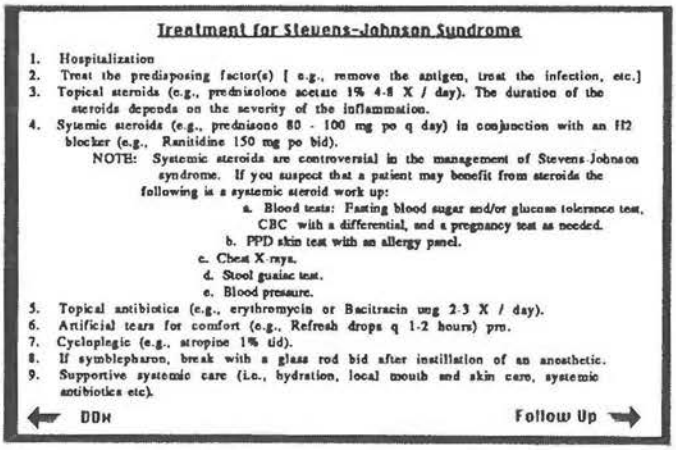

Symptoms of Stevens-Johnson Syndrome
• Fever
• Generalized malaise and arthralgias
• Sore throat
• Cough
• Vesicular skin rash
• Red eye
Etiology
Signs


Signs of Stevens-Johnson Syndrome
Click A Topic
Conjunctiva
Cornea
Eyelids
Globe
Lacrimal System
Other
Cornea
• Cicatrization
• Dense opacity
• Subepithelial infiltration
• Keratoconjunctivitis sicca
• Subconjunctival hemorrhage
• Neovascularization
• Pannus
• Corneal ulceration or perforation
• Punctate keratitis
close
Symptoms
Critical Signs


Main Menu for Stevens-Johnson Syndrome
Critical signs of Stevens-Johnson Syndrome
• Target lesions on the skin which appear as red, central vesicles surrounded by a pale ring which is surrounded by a red ring
• Hemorrhagic crusting of the lips
• Bilateral conjunctivitis
Signs
Work Up


Work Up for Stevens-Johnson Syndrome
1. History: Attempt to determine the predisposing factor.
2. SLE: Look for corneal neovascularization and be certain to evert the eyelids and examine the fornices for papillae.
3. Obtain conjunctival and corneal scrapings for stains and cultures if an infection is suspected.
4. Obtain an electrolyte profile and a complete blood count.
Critical Signs
DDx


Differential Diagnosis for Stevens-Johnson Syndrome
• Ocular Pemphigoid:
A slowly progressive scarring of the conjunctiva with symblepharon formation, shortening of the fornices and dry eye. There is typically also evidence of mucous membrane vesicles, or ruptured or bullae formation.
Work Up
Treatment

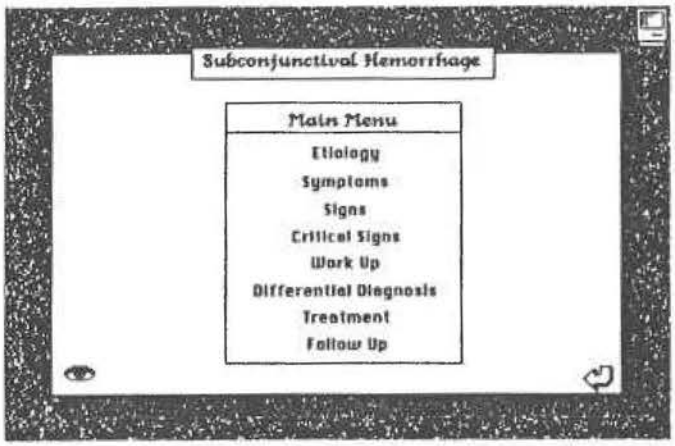

Treatment for Stevens-Johnson Syndrome
1. Hospitalization
2. Treat the predisposing factor(s) [e.g., remove the antigen, treat the infection, etc.]
3. Topical steroids (e.g., prednisolone acetate 1% 4-8 X / day). The duration of the steroids depends on the severity of the inflammation.
4. Systemic steroids (e.g., prednisone 80 - 100 mg po q day) in conjunction with an H2 blocker (e.g., Ranitidine 150 mg po bid).
NOTE: Systemic steroids are controversial in the management of Stevens-Johnson syndrome. If you suspect that a patient may benefit from steroids the following is a systemic steroid work up:
a. Blood tests: Fasting blood sugar and/or glucose tolerance test, CBC with a differential, and a pregnancy test as needed.
b. PPD skin test with an allergy panel.
c. Chest X-rays.
d. Stool guaiac test.
e. Blood pressure.
5. Topical antibiotics (e.g., erythromycin or Bacitracin ung 2-3 X / day).
6. Artificial tears for comfort (e.g., Refresh drops q 1-2 hours) prn.
7. Cycloplegic (e.g., atropine 1% tid).
8. If symblepharon, break with a glass rod bid after instillation of an anesthetic.
9. Supportive systemic care (i.e., hydration, local mouth and skin care, systemic antibiotics etc).
DDx
Follow Up

Follow Up for Stevens-Johnson Syndrome

- The patient should be followed daily while in the hospital, with monitoring for the development of an infectious corneal ulcer or elevation in the intraocular pressure. When the acute phase has resolved, the patient should be seen on a weekly outpatient basis with monitoring for any long term ocular complications, such as scarring, that may arise.
- Topical steroid and antibiotic treatment should be maintained for at least 48 hours after the acute phase has resolved. The steroids should then be tapered accordingly.
- If the conjunctiva has been severly scarred, the use of artificial tears and lubricating ointment may need to be used indefinitely.
- If trichiasis develops, cryotherapy or surgical repair may be indicated.
- Consider keratoprosthesis if the eye has been badly scarred but still shows signs of visual potential.

Treatment

Main Menu



Etiology of Subconjunctival Hemorrhages

A subconjunctival hemorrhage is painless and one of the most common ocular presentations. There are many etiologies which are listed below:

1. Valsalva-like maneuvers: (e.g., coughing, sneezing, straining, vomiting, constipation etc.)
2. Traumatic: The hemorrhage may be isolated or associated with a retrobulbar hemorrhage or a ruptured globe
3. Systemic hypertension that is not under good control
4. A bleeding disorder or menstruation
5. Idiopathic

Main Menu | Symptoms

Symptoms of Subconjunctival Hemorrhages

- Ocular irritation
- A rapid onset appearance of "blood" on the eye typically leads to an immediate patient concern
- The patient may or may not elicit a positive history as to the etiology of the hemorrhage

Etiology | Signs

Signs of Subconjunctival Hemorrhages

- Loose blood in the bulbar subconjunctival spaces which is usually unilateral but can present bilaterally.
- Flat sheaths of uniform red blood without vessel patterns (one may see streaks as the blood spreads).
- Blood will typically accumulate more toward the limbus, yet there is usually a clear space denoting a visible border between the blood and the cornea.
- The spread of the blood can occur in any direction, to any degree during the first few hours or days.
- Over an average of 7-21 days the blood will turn orange, to pink and back to white. Rarely will permanent blood staining persist.

Symptoms | Critical Signs

Main Menu for Subconjunctival Hemorrhage

Critical Signs for Subconjunctival Hemorrhages

- Blood underneath the conjunctiva, often in a sector of the eye.
- Following trauma, the entire view of the sclera may be obstructed.

Signs | Work Up

Work Up for Subconjunctival Hemorrhages

1. Rule out a history of trauma (e.g., ocular, head or to the eyes).
2. Rule out local inflammatory disease (e.g., hyperacute hemorrhagic conjunctivitis).
3. Rule out associated systemic diseases (e.g., cardiovascular or blood dyscrasias, febrile diseases or leukemia).
4. Always measure the blood pressure in patients presenting with subconjunctival hemorrhages simply because the condition of hypertension is commonly overlooked.
5. If the patient has recurrent subconjunctival hemorrhages or a history of bleeding/clotting problems, a bleeding time, PT, PTT, and CBC with differential(platelets) should be obtained and a medical consultation considered.

Critical Signs | DDx

Differential Diagnosis for Subconjunctival Hemorrhages

1. Kaposi's sarcoma - presents as a red or purple lesion beneath the conjunctiva which is usually slightly elevated. These patients should be evaluated for AIDS.
2. Other conjunctival neoplasms - e.g. lymphomas, with secondary hemorrhages

Work Up | Treatment

Treatment of Subconjunctival Hemorrhages

1. Attempt to determine the etiology through a careful history. Refer to a general medical practitioner if indicated.
2. Reassure the patient as to the self-limiting nature of the hemorrhage. Explain a slow resolution (by color) over a period of 7 to 21 days.
3. Artificial tears (e.g., Cellufresh) may be prescribed if mild ocular irritation is present.
4. Alternating hot and cold packs may aid in the reabsorption of the loose blood but this is probably more a placebo than therapy.

DDx | Follow Up

Follow up for Subconjunctival Hemorrhages

- This condition usually clears spontaneously within 1-2 weeks. Patients are advised to return if the blood does not fully resolve, or if they suffer a recurrence.
- For recurrent presentations - recheck at 3 to 6 months.
- If there are more than 2 recurrances within 1 year, a full medical workup by a physician is indicated for hypertension or a bleeding diathesis.

Treatment

Main Menu

Superficial Punctate Keratopathy

Main Menu

- General Information
- Critical Signs
- Signs/Symptoms
- Etiology
- Work Up
- Treatment

General Information

- Historically, the nomenclature has been very confusing. Below are some of the different terms used:

Punctate Epithelial Erosions (PEE):

- Fine focal defects which are generally depressed. These will stain with fluorescein and rose bengal.

Punctate Epithelial Microcysts (PEM):

- Can occur in isolated groups or confluently.

Punctate Epithelial Keratopathy (PEK):

- Round lesions which may be very small or very large.
- These represent accumulations of epithelial cells which are often surrounded by an inflammatory cell infiltrate.
- These stain poorly with fluorescein but well with rose bengal.

More ➡

⬅ Main Menu

General Information

- Most spotty lesions on the cornea are superficial, but there are variations in size, location and distribution. The clinician must determine the location (epithelial, sub-epithelial or stromal).

- Size
  - Small lesions are generally epithelial (can not be seen w/o slit lamp).
  - Larger lesions may be epithelial or subepithelial.

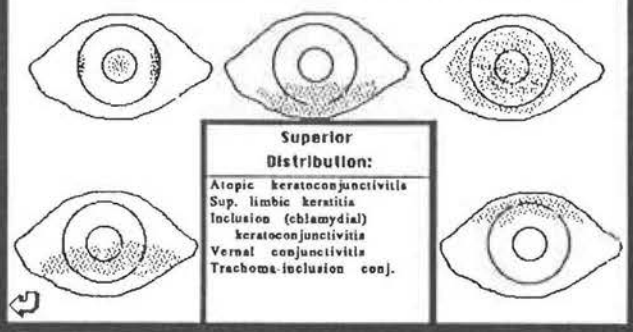
- Staining patterns
  - The distribution of the epithelial/subepithelial lesions is of diagnostic value.

Staining patterns ➡

Common Etiologies ➡

⬅

Click of cornea for possible etiology



Superior Distribution:

- Atopic keratoconjunctivitis
- Sup. limbic keratitis
- Inclusion (chlamydial) keratoconjunctivitis
- Vernal conjunctivitis
- Trachoma-inclusion conj.

Common Etiologies of Punctate Epithelial Erosions (PEE)

- Thermal/UV keratopathy (photokeratopathy)
- Trauma (non specific response to injury)
- Mild chemical injury

It is common to find punctate epithelial erosions as part of the picture with almost every form of superficial corneal disturbance, but these are particularly striking in the following:

- Dry eye syndrome
- Staph blepharokeratoconjunctivitis
- Neuroparalytic keratitis
- Topical drug toxicity
- Foreign body under the lid
- Exposure keratopathy
- Contact lens related disorders

The position of staining will help determine the etiology.

⬅ ➡

Common Etiologies of Punctate Epithelial Microcysts (PEM)

- These can occur in isolated groups or confluently.

- If localized:
  1. May be an area of epithelial healing.
  2. Superficial dystrophic process associated with recurrent erosion.

⬅ ➡

Common Etiologies of Punctate Epithelial Keratopathy (PEK)

The conditions in italics are not covered in this program.

FINE PUNCTATE KERATOPATHY:

- Staph Blepharokeratoconjunctivitis
- Viral keratitis
- Chlamydial
- *Molluscum contagiosum*
- Dry eye syndrome
- Exposure keratopathy

COURSE PUNCTATE KERATOPATHY:

- *Adenovirus*
- Herpes simplex keratitis
- Herpes zoster keratitis
- *Vaccinia*

⬅ Critical signs ➡

Main menu for Superficial Punctate Keratitis ➡

Critical Signs for Superficial Punctate Keratopathy

- Small pinpoint epithelial defects which stain with rose bengal or fluorescein.
- There is most likely an underlying cause.

⬅ Etiologies Signs/Symptoms ➡

Signs and Symptoms of SPK

- Conjunctival injection
- Small pinpoint epithelial defects which stain with fluorescein or rose bengal

- Some pain depending upon etiology
- Foreign body sensation
- Photophobia
- Red eye

- Corneal edema and infiltration can be present but are generally limited to the anterior stroma.

← Critical signs

Work up →

Work-Up for Superficial Punctate Keratopathy

- Since SPK is nonspecific, the goal of the workup is to find out the etiology.

- Case History:
  - Is patient a contact lens wearer? Is there a history of trauma?
  - Is the patient using any eyedrops? Is there any discharge or eyelid matting?
  - Any associated symptoms may help establish etiology (allergic itiching, viral burning, inflammatory pain).

- Slit-lamp Exam: Use fluorescein. Staining patterns →
  - Look at staining pattern. May also want to use rose bengal.
  - Look for follicular / papillary response in both upper and lower lids (evert).
  - Look at eyelid closure. May be worthwhile to evert upper lid to search for FB.
  - Evaluate tear film.
  - If SPK is accompanied by infiltration or significant ocular anterior chamber reaction, infection must be excluded, diagnosed and treated.
  - Inspect contact lenses for fit and for defects, deposits etc.

- Look for accompanying signs in the lids and conjunctiva ! !

← Signs/Symptoms

Treatment →

Treatment of Superficial Punctate Keratopathy

Non-Specific Treatment of SPK

- Antibiotic (therapeutic or prophylactic) -› gentamicin or tobramycin
  - Prophylactic dose (bid for 2-3 days)
  - Therapeutic dose (minimum qid for 5-7 days; depends on severity)
  - Erythromycin ointment can also be used

- Non preserved lubricants are valuable (Celiufresh)

- Hypertonic drops or ointments can be used to reduce secondary epithelial edema.

- Cycloplegics in moderate to severe cases to reduce risk or secondary anterior chamber reaction.

Non specific Tx for non-contact lens wearer →

Non specific Tx for contact lens wearer →

- See appropriate section to treat underlying cause.

← Work up

Menu →

Non-Specific Treatment for Non Contact Lens Wearer

MILD:
- Artificial tears qid (i.e. Refresh)
- May add a lubricating ointment at bedtime (i.e. Refresh PM)
- Return if symptoms worsen or do not improve.

MODERATE TO SEVERE:
- Antibiotic ointment (i.e. erythromycin ointment)
- Cycloplegics
  - Tropicamide 1% or cyclopentolate 2%
- Pressure patch for 24 hours

- After patch is removed:
  - Continue antibiotic ointment 2-3 X / day for 4 days

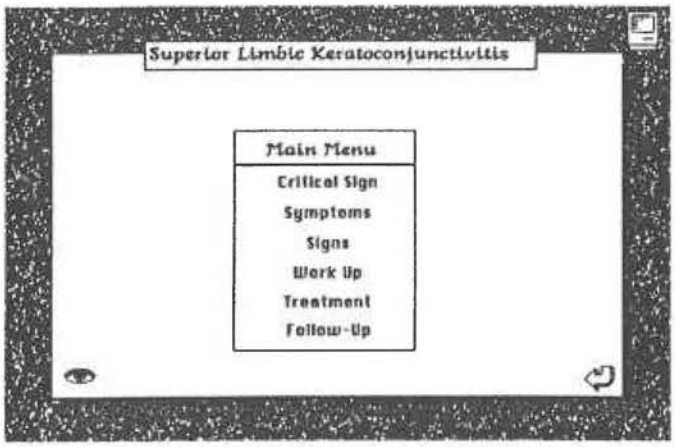
FOLLOW UP:
- Generally told to return if symptoms worsen or do not improve. Follow up based on underlying cause.

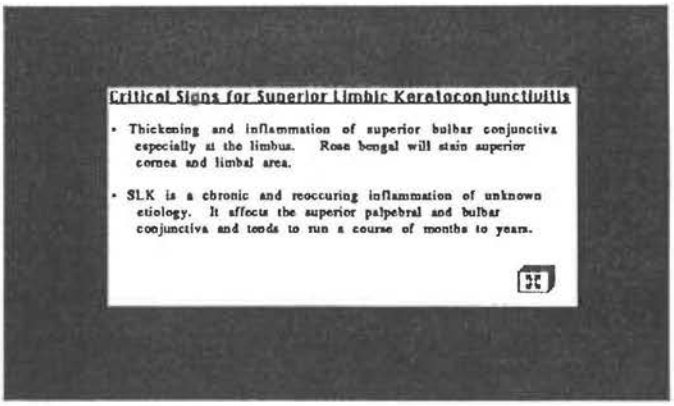
Non-Specific Treatment for Contact Lens Wearer

MILD CASE:
- Artificial tears qid (i.e. Refresh)
- Lenses may or may not be worn, depending on the symptoms and the degree of SPK.
- Should be rechecked within a few days to a week, depending on the symptoms and degree of SPK

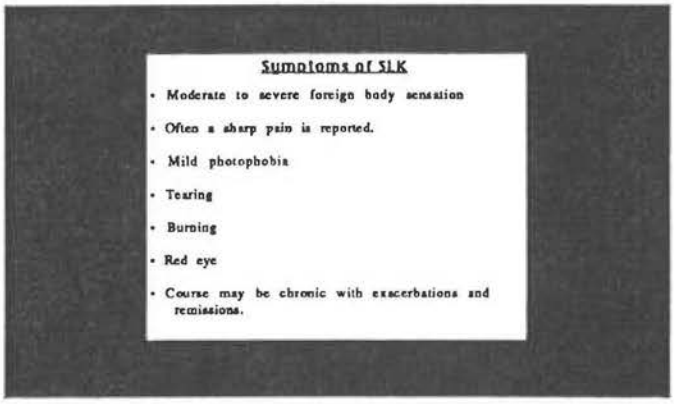
MODERATE TO SEVERE CASE:
- Discontinue contact lens wear
- Tobramycin drops 4-6 X daily and Tobramycin ung qhs
- Consider cycloplegic for pain (Homatropine)

FOLLOW UP:
- Patient should be followed daily until significant improvement is seen.
- Patient should not wear lenses until condition resolves.
- Discontinue antibiotic when SPK resolves.
- If contact lenses are thought responsible , habits or lenses should be changed. See specific treatment of contact lens problems.

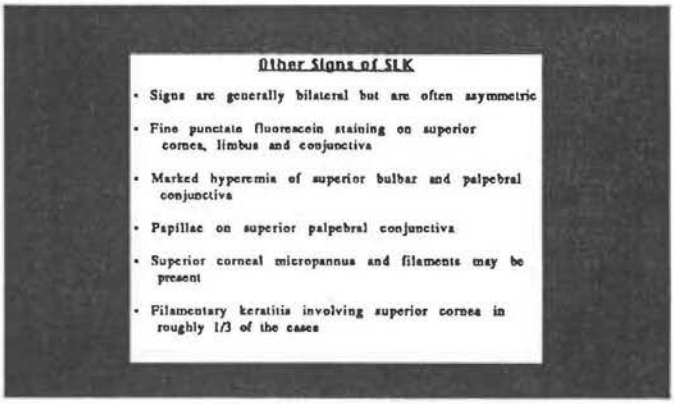




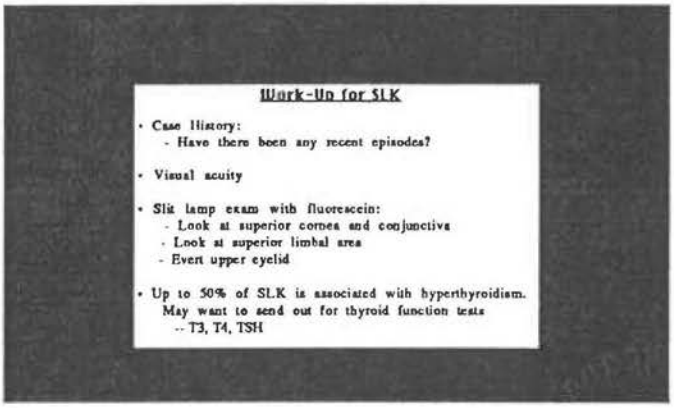
Main Menu For SLK | Symptoms



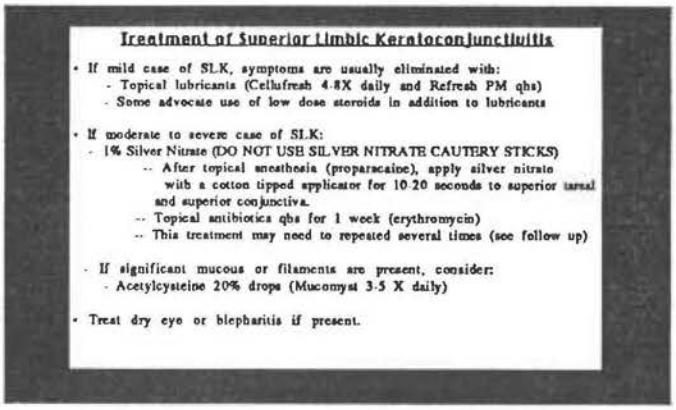
Critical signs | Signs



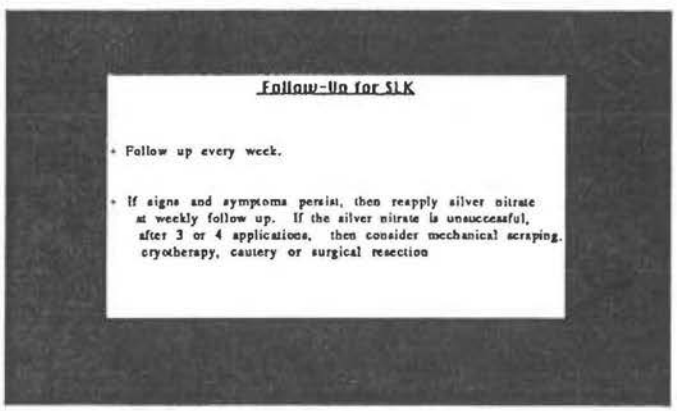
Symptoms | Work-up



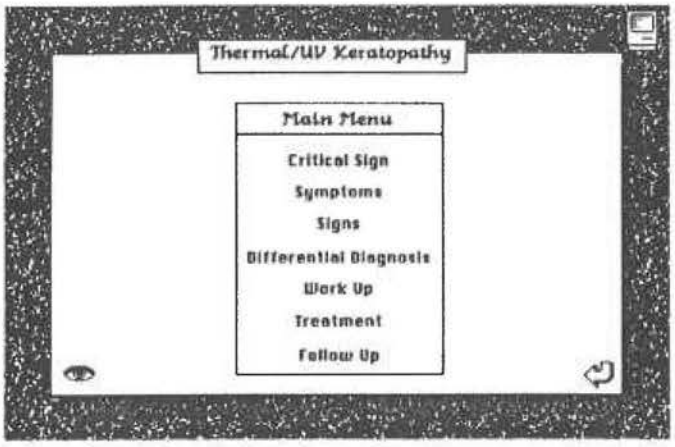
Signs | Treatment

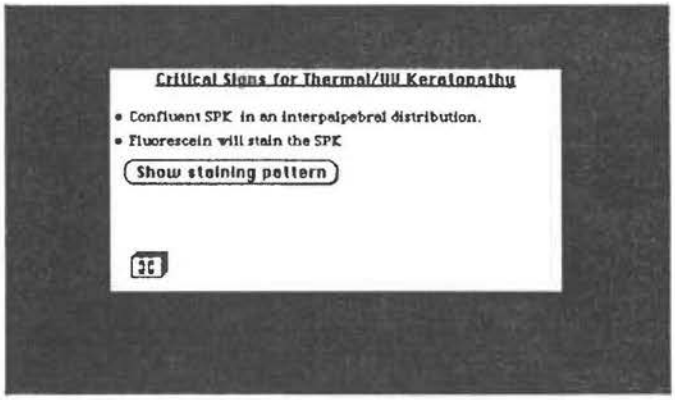


Work-up | Follow up

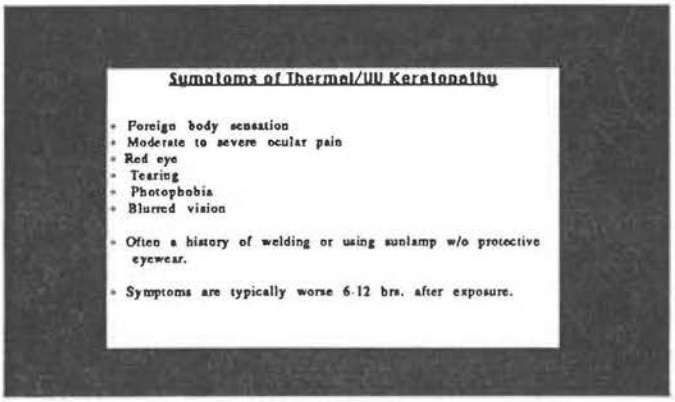


Treatment | Menu

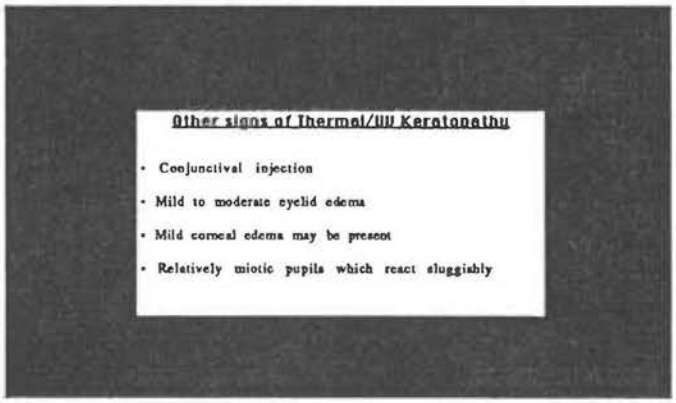




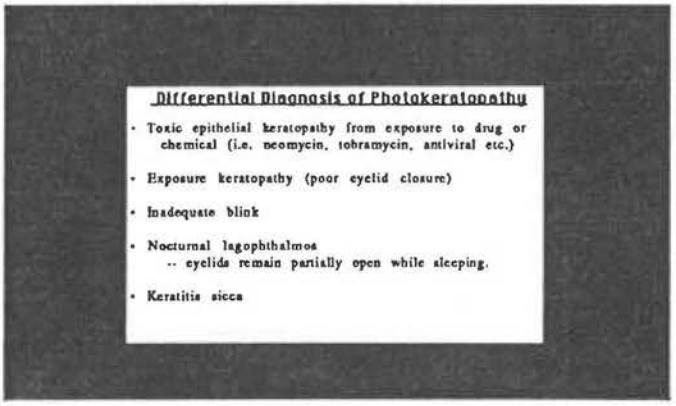
← Main menu for Thermal/UV Keratopathy Symptoms →



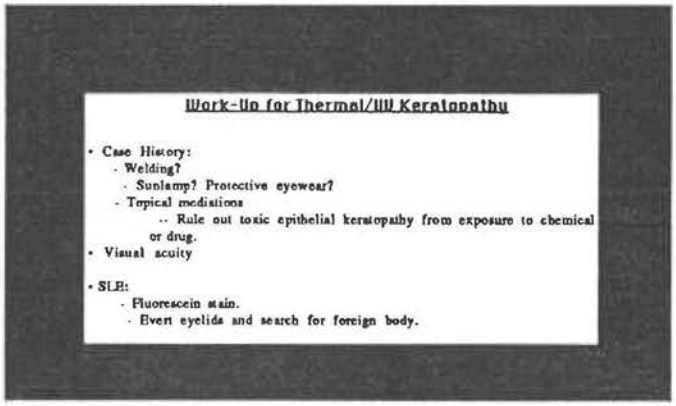
← Critical Signs Signs →



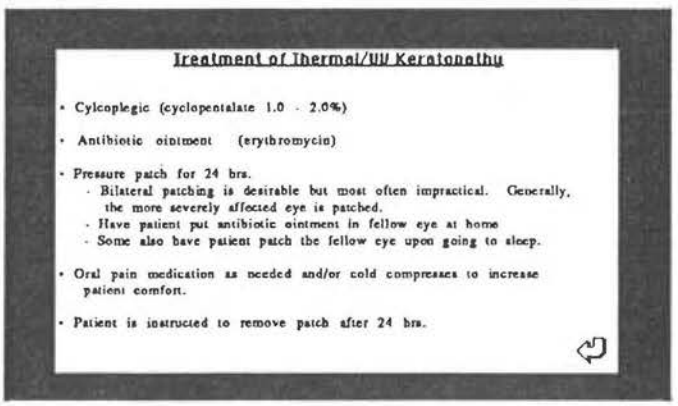
← Symptoms DDH →



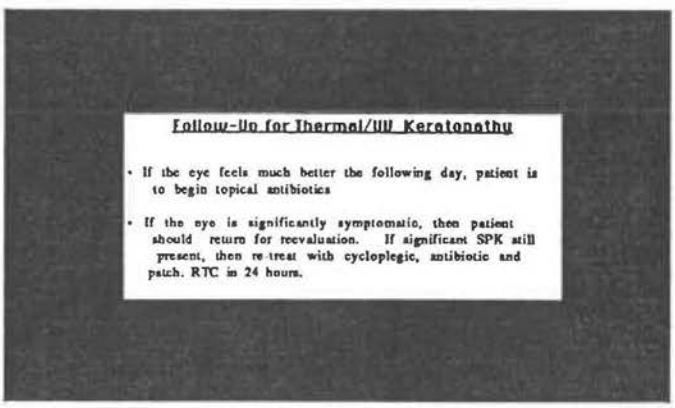
← Signs Work-up →



← DDH Treatment →



← Work-up Follow up →



← Treatment Menu →

Trachoma

Main Menu

- Etiology
- Symptoms
- Signs
- Critical Signs
- Classification
- Differential Diagnosis
- Treatment
- Follow Up

Etiology of Trachoma

- The causative organism of trachoma is Chlamydial trachomatis.
- Trachoma tends to be an endemic in underdeveloped countries and among certain ethnic groups.
  a. Asia
  b. Africa
  c. South America
  d. American Indians
- Trachoma is generally spread by direct contact (hands, sexual, or cervical).

Main Menu | Symptoms

Symptoms of Trachoma

- Tearing
- Photophobia
- Mucous discharge
- Pain
- Lid swelling

Etiology | Signs

Signs of Trachoma

- Chronic follicular conjunctivitis
- Lid edema
- Chemosis
- Hyperemia
- Papillary hypertrophy may mark the follicular response
- Linear conjunctival scarring (Arlt's lines)
- Epithelial keratitis
- Marginal and central infiltrates
- Superficial vascularization / Pannus
- Shallow corneal ulcers
- Limbal swelling and follicles
- Herbert's pits
- Trichiasis and entropian in the later stages

All the signs of trachoma most often affect the upper half of the cornea and conjunctiva but are not limited to this area.

Symptoms | Critical Signs

Main Menu for Trachoma

Critical Signs of Trachoma

- Chronic follicular conjunctivitis that, usually, initially affects the upper tarsal plate
- Limbal follicles and Herbert's pits are pathognomonic of trachoma

Signs | Classification

MacCallan Classification of Trachoma

- Stage I - The upper tarsal plate demonstrates immature follicles without evidence of conjunctival scarring
- Stage IIA - The upper tarsal plate demonstrates mature follicles with moderate papillary hypertrophy
- Stage IIB - The papillary hypertrophy obscures the follicular response
- Stage III - Conjunctival scarring develops with continued follicular hypertrophy
- Stage IV - The follicular hypertrophy on the upper tarsal plate is replaced by cicatricial scarring

Critical Signs | DDx

Differential Diagnosis of Trachoma

1. Bacterial conjunctivitis - generally presents as a "meaty" red conjunctivitis that increases toward the fornices. The cornea is typically clear; the hyperemic vessels will blanch with a mild vasoconstrictor; usually a papillary response and a mucopurulent discharge.
2. Chlamydial conjunctivitis - generally sexually transmitted typically seen in teenagers and young adults. A history of vaginitis, cervicitis, or urethritis may be noted. Chlamydial conjunctivitis presents as inferior tarsal conjunctival follicles in conjunction with tiny gray-white subepithelial infiltrates. It is generally diagnosed with conjunctival scrapings and a Giemsa stain. The stain shows basophilic bodies in the epithelial cells, polymorphonuclear leukocytes, and lymphocytes.

Classification | Treatment

Treatment of Trachoma

- All cases of trachoma, mild to severe, should be treated with oral tetracycline (or erythromycin) 250 mg for 3 to 6 weeks.
- Topical ointments (tetracycline, erythromycin or sulfonamides) for 2 to 3 months have shown some success, but are not the treatment of choice.
- The family members and intimate associates should also be treated with topical ointments bid for 1 to 2 months.
- In cases with active corneal involvement, topical steroids may be indicated. However, the prognosis for response is guarded.
- If there are any suggestive venereal signs or symptoms present, refer for a medical evaluation.

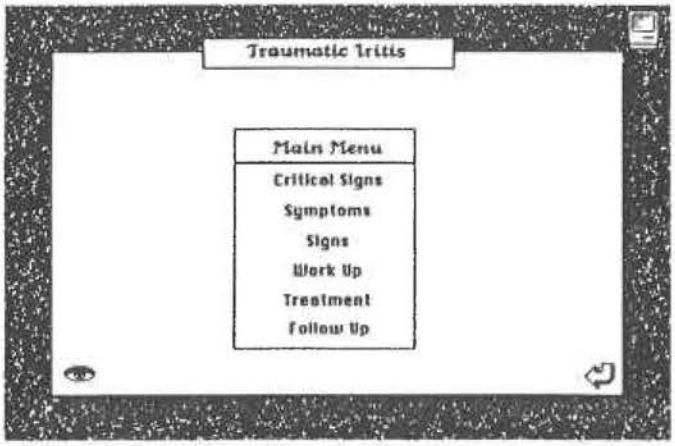
DDx | Follow Up

Follow Up for Trachoma

- Follow the patient every 2 to 3 weeks in the beginning, and then as needed.
- An active disease usually takes about 3 to 6 months to run its course (with or without treatment).
- Most cases result in some degree of conjunctival and corneal scarring, and the patient should be informed about the prognosis.
- Careful instructions should be given to patients under care and, also, ones at a high risk (exposed, endemic, or ethnic populations) of acquiring trachoma. These instructions should include hygiene and the monitoring for early signs.

Treatment

Main Menu


Traumatic Iritis
Main Menu
Critical Signs
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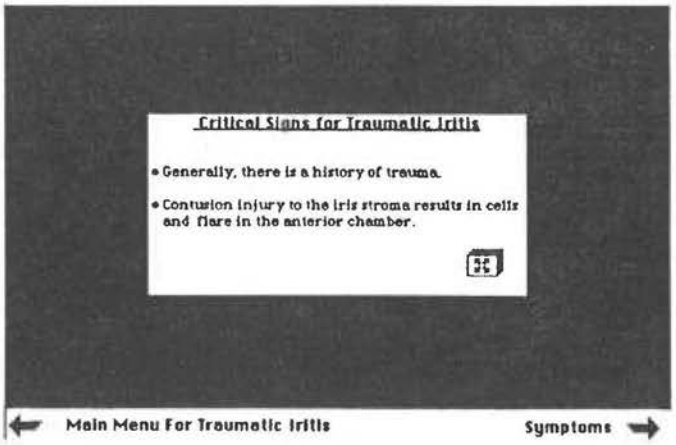
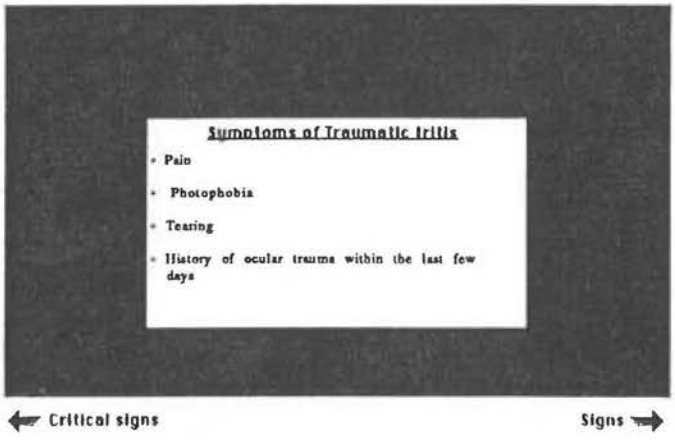
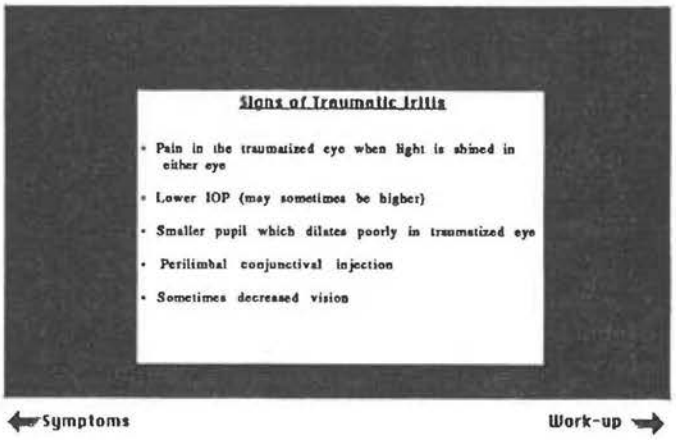
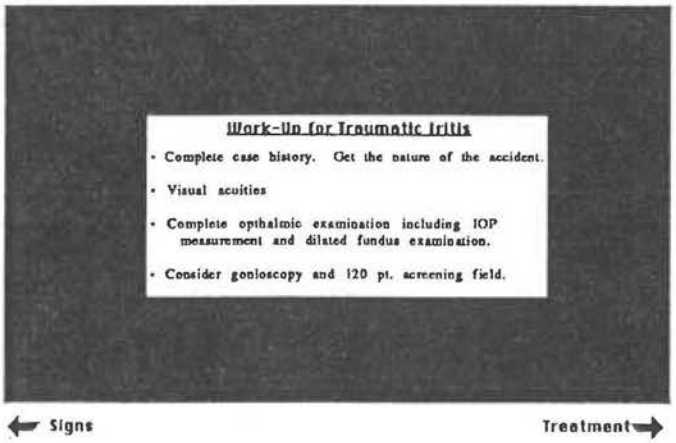
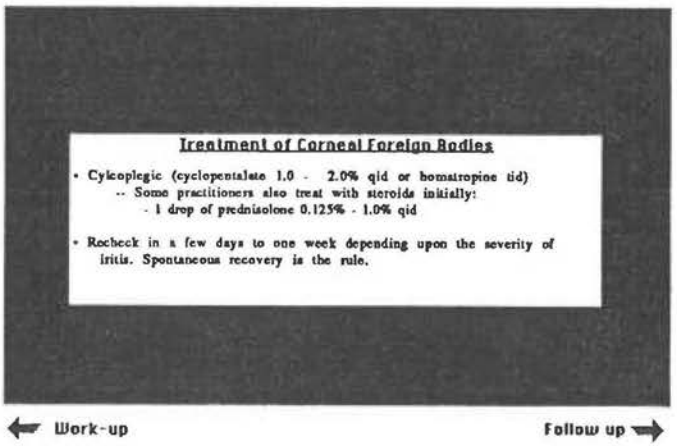
Treatment
Follow Up

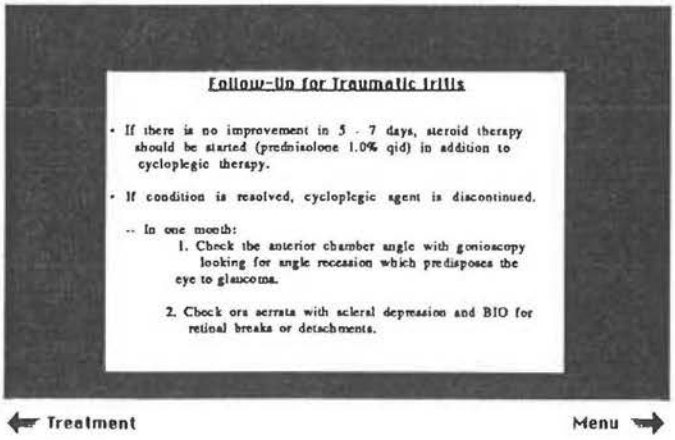

Critical Signs for Traumatic Iritis
• Generally, there is a history of trauma.
• Contusion injury to the iris stroma results in cells and flare in the anterior chamber.
Main Menu For Traumatic Iritis
Symptoms

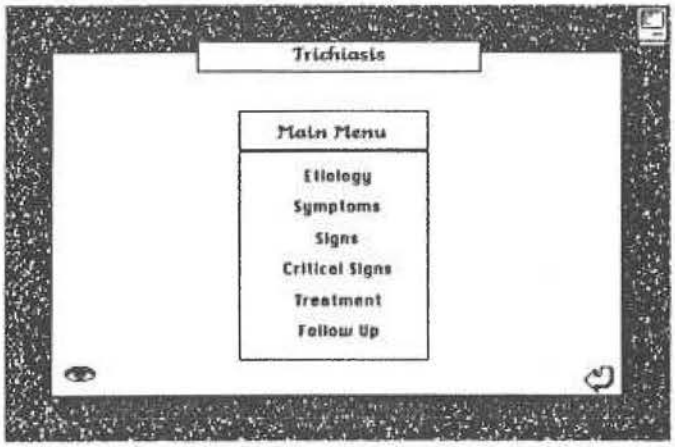

Symptoms of Traumatic Iritis
• Pain
• Photophobia
• Tearing
• History of ocular trauma within the last few days
Critical signs
Signs


Signs of Traumatic Iritis
• Pain in the traumatized eye when light is shined in either eye
• Lower IOP (may sometimes be higher)
• Smaller pupil which dilates poorly in traumatized eye
• Perilimbal conjunctival injection
• Sometimes decreased vision
Symptoms
Work-up


Work-Up for Traumatic Iritis
• Complete case history. Get the nature of the accident.
• Visual acuities
• Complete opthalmic examination including IOP measurement and dilated fundus examination.
• Consider gonioscopy and 120 pt. screening field.
Signs
Treatment


Treatment of Corneal Foreign Bodies
• Cyloplegic (cyclopentalate 1.0 - 2.0% qid or homatropine tid)
-- Some practitioners also treat with steroids initially:
- 1 drop of prednisolone 0.125% - 1.0% qid
• Recheck in a few days to one week depending upon the severity of iritis. Spontaneous recovery is the rule.
Work-up
Follow up


Follow-Up for Traumatic Iritis
• If there is no improvement in 5 - 7 days, steroid therapy should be started (prednisolone 1.0% qid) in addition to cycloplegic therapy.
• If condition is resolved, cycloplegic agent is discontinued.
-- In one month:
1. Check the anterior chamber angle with gonioscopy looking for angle recession which predisposes the eye to glaucoma.
2. Check ora serrata with scleral depression and BIO for retinal breaks or detachments.
Treatment
Menu



Etiology of Trichiasis

- Epiblepharon: Congenital versus acquired
- Chronic blepharitis: Thickened erythematous, inflammed eyelid margins with excess secretions and telangiectatic blood vessels running across them
- Entropian: Inward turning of the eyelid margin. May be due to Steven-Johnsons syndrome, ocular pemphigoid, chemical burns, trachoma, or others
- Idiopathic presentation

Main Menu | Symptoms

Symptoms of Trichiasis

- Ocular irritation
- Foreign body sensation
- Frequent hyperlacrimation and epiphoria
- Red eye

Etiology | Signs

Signs of Trichiasis

- Inferior lid involvement more often than superior
- Misdirected eyelash or lashes
- Inferior vertical or regular foreign body tracking
- Superficial punctate keratitis
- Conjunctival injection
- Corneal abrasion

Symptoms | Critical Signs

Main Menu for Trichiasis

Critical Signs for Trichiasis

- Fine filamentary lashes or complete lash(es) turned inward rubbing against the inferior corneal surface.

Signs | Treatment

Treatment of Trichiasis

1. Remove the misdirected lashes.
   a. A few misdirected lashes can be removed at the slit lamp with fine cilia forceps (recurrance is common; 2-4 weeks in youths and 4-6 weeks in adults).
   b. Diffuse, severe or recurrent trichiasis, the misdirected lashes can sometimes be removed as above, however, definitive therapy generally requires electrolysis, cryotherapy, or surgery.
2. Treat the SPK with antibiotic ointment (e.g., erythromycin or bacitracin tid) for several days.
3. Treat any active or chronic marginal lid disorders.

Critical Signs | Follow Up

Follow Up for Trichiasis

- The patient can usually self-manage the condition with proper instruction.
- The patient should return if corneal irritation reoccurs.

Treatment | Main Menu

Vernal Conjunctivitis

Main Menu

- Etiology
- Symptoms
- Signs
- Critical Signs
- Differential Diagnosis
- Treatment
- Follow Up

Etiology of Vernal Conjunctivitis

Vernal conjunctivitis is a seasonally recurrent, bilateral inflammation of the conjunctiva usually presenting in warmer weather. It may occur in one of two forms:

- A palpebral form which is distinguished by cobblestone papillae on the tarsal conjunctiva which may be associated with sheild ulcers of the superior cornea.
- A limbal form which occurs with papillary hypertrophy on the limbal conjunctiva associated with white, chalky concretions known as Trantas' dots near the limbus. Limbal vernal is more prevelant in black patients.

There is usually a family or personal history of allergies, with young males between the ages of 12 and 30 being at the highest risk.

← Main Menu Symptoms →

Symptoms of Vernal Conjunctivitis

- Intense itching
- Watery discharge
- Tearing
- Photophobia
- Foreign body sensation
- Burning
- Mucous discharge
- Blurred vision

← Etiology Signs →

Signs of Vernal Conjunctivitis

- Vernal conjunctivitis is mostly bilateral in presentation and is frequently associated with secondary lid conjestion and a pseudoptosis.
- The discharge characteristically consists of thick, ropy, whitish-yellowish strands of dense mucous that may cover the superior tarsus and conjunctiva, spread onto the cornea, and accumulate at the inner canthus.
- Generalized palpebral/perilimbal hyperemia
- Diffuse papillary ("cobblestone") conjunctivitis on the superior tarsal plate.
- The most common corneal involvement is superior SPK scattered in the upper one-third corneal region.
- With limbal involvement, the most frequent sign are Tranta's dots. Tranta's dots are most frequently found on the superior limbus and appear as puffy, white, round dots about 1 - 2 mm in size. They may be flat or slightly raised (in the more advanced disease).

← Symptoms Critical Signs →

Main Menu for Vernal Conjunctivitis →

Critical Signs of Vernal Conjunctivitis

- Large conjunctival papillae, commonly referred to as "cobblestone papillae" under the upper eyelid. Eversion of the upper eyelid is necessary to make the diagnosis. A variation to this is limbal vernal which presents with large conjunctival follicles along the limbus.
- Thick, ropy, whitish-yellowish strands of dense mucous may cover the superior tarsus and conjunctiva, spread onto the cornea, and accumulate at the inner canthus.

← Signs Work Up →

Differential Diagnosis of Vernal Conjunctivitis

- Atopic keratoconjunctivitis - a year-round allergy
- SLK (superior limbic keratoconjunctivitis) - usually a milder, less symptomatic presentation.
- GPC - also less symptomatic and more related to a cause (e.g., contact lenses).
- Infectious causes of a seasonal nature or itching.

← Critical Signs Treatment →

Treatment of Vernal Conjunctivitis

- All forms of vernal conjunctivitis respond dramatically to steroids. The recommended concentration and dosage is 1% prednisolone (or an equivalent) q2-4h for 3-7 days. The duration may have to be prolonged in more severe cases.
  - -- Taper the steroids to the lowest maintenance dose (e.g., 1 gtt 3 X per week) and continue this maintenance dose for 4-6 weeks.
  - -- With the long term use, steroids can lead to the development of cataracts or an elevation of the intraocular pressure. These must both be monitored if topical steroids are used.
- Topical antibiotics (e.g., erythromycin ung or sulfacetamide drops qid).
  - -- a bandage hydrophilic lens is recommended for prophylaxis
- Cycloplegic agent (e.g., homatropine 5% tid).
- Cromolyn sodium (Opticrom) 4% qid may be introduced during the tapering of the steroids but it is not effective as initial therapy.
- Cool compresses qid.

← Work Up Follow Up →

Follow Up for Vernal Conjunctivitis

- Milder forms of vernal conjunctivitis usually respond very quickly and completely to steroids in 1 week and may or may not need maintenance regimens.
- Limbal vernal does not require maintenance regimens with steroid usage.
- Patients with more severe forms should be carefully advised of the chronic nature of their disease with remissions and exacerbations over an extended period of years (generally 5-10).
- Advise patients in remission to report symptoms upon exacerbation or to be rechecked on an annual basis.

← Treatment Main Menu →

Viral Conjunctivitis

Main Menu

- Etiology
- Symptoms
- Signs
- Critical Signs
- Differential Diagnosis
- Treatment
- Follow Up

Etiology of Viral Conjunctivitis

- Viruses are single-cell organisms smaller than bacteria which are made up of single nucleic acid (DNA or RNA) strands that can grow or multiply independently.
- Some of the viruses that have been known to infect the eye are as follows:

DNA Virus	RNA Virus
Adenovirus	Coxsackie
Cytomegalovirus	Enterovirus 70 - (Hemophilus conjunctivitis)
Epstein-Barr (mononucleosis)	Influenza
Herpes simplex	Rubeola (measles)
Molluscum contagiosum	Mumps
Verrucae (papilloma)	Newcastle disease
Vaccinia	Poliovirus
Variola (small pox)	Rabies
Varicella (herpes zoster)	Rhinovirus
HIV (AIDS)	Rubella

Main Menu — Symptoms

Symptoms of Viral Conjunctivitis

- Tearing and irritation of one eye with frequent contralateral autoinoculation reported
- Watery discharge
- Photophobia
- A history of onset is typically 3-7 days
- There may be a prodromal medical history, especially in children, e.g., upper respiratory infection (URI), otitis media (ear infection), a low grade fever, or most commonly - pharyngoconjunctival fever (PCF)
- Occassionally, there is a previous history of conjunctivitis
- A mild fluctuation in visual acuity (transient)

Etiology — Signs

Signs of Viral Conjunctivitis

- Red edematous eyelids with a purplish-pinkish bulbar hyperemia.
- The injection usually begins at the inner canthus and slowly spreads laterally to involve the entire bulbar conjunctiva (vessels may blanch with a vasoconstrictor).
- Membrane/pseudomembrane formation in the inferior cul-de-sac.
- The most common discharge is that of a serous (teary, watery) variation.
- Follicular changes - pale mounds of infiltrative cellular accumulation on the palpebral conjunctiva of varying diameter.
  - -- Follicular changes in the lower cul-de-sac of children are considered to be normal in quiet eyes.
  - -- Follicular changes on the superior tarsal plate (in children or adults) is usually not considered to be normal.
- Chemosis
- Subconjunctival hemmorhages
- Occasional preauricular lymphadenopathy (tenderness on palpation).
- Sub-epithelial corneal infiltrates may develop several weeks after the initial onset.
- Stromal infiltrates which may persist for months to years following the infection.
- There is typically a quick NaFl tear break-up time which can ultimately produce secondary corneal SPK which may stain with rose bengal.

Symptoms — Critical Signs

Main Menu for Viral Conjunctivitis

Critical sign of Viral Conjunctivitis

- A follicular response of the superior tarsal conjunctiva.

Signs — Differential Diagnosis

Differential Diagnosis for Viral Conjunctivitis

- There are four specific etiologies that need to be ruled out when considering viral conjunctivitis as a potential diagnosis.

Choose A Topic Below

- Pharyngoconjunctival Fever (PCF)
- Epidemic Keratoconjunctivitis (EKC)
- Herpes Simplex Virus (HSV)
- Chlamydial Conjunctivitis

Critical Signs — Treatment

Treatment for Viral Conjunctivitis

- There is no prescription cure for viruses of any kind.
- Antiviral agents are not effective for adenoviruses.
- Prophylactic antibacterial use is questionable as to the value and efficiency.
- Steroid and steroid combination drugs should be limited to the more severe presentations.

Options:

1. Ocular lubricants (mucomimetics) to supplement and protect the tear film from dessication.
2. Topical vasoconstrictors/antihistamine preparations (e.g., Naphcon A) will improve the appearance and may reduce the symptoms.
3. Warm/cool compresses several times / day for 1-2 weeks.

Differential Diagnosis — Follow Up

Follow Up for Viral Conjunctivitis

- Instruction of preventative measures plays a key role in the reduction of spreading the viral infection from one eye to the other.
- It is very important to advise the patient that "This may get worse before it gets better".
- Carefully instruct the patient on the contagious nature of some viruses

1. Recheck acute presentations approximately 5-8 days after the first signs of the infection are reported.
   - If nonspecific adenovirus, the condition will have improved.
   - If virulent adenovirus (e.g., EKC), it will worsen and the cornea usually becomes involved.
2. Educate the patient on the potential dormant nature of ocular viruses and the potential for intermittent mild to moderate exacerbations of signs and symptoms.

Treatment — Main Menu

Conclusion

This software is designed to aid both the student and clinician in the differential diagnosis of a red eye. There are four main sections to the program:

- Main Menu
- A list of all conditions covered in the program which are separated by tissue involved. This can serve as a menu providing rapid transference from one disease to another.
- Differential Diagnosis: By entering signs and symptoms, the computer will come up with a "List of Possibilities" for the etiology of the red eye.
- 45 "Disease Stacks": These stacks contain information about each condition including a treatment and follow-up regimen.

Since ocular conditions do not always present with the same signs and symptoms, we have programmed each condition with the "TYPICAL" signs and symptoms. As students, we have limited clinical experience and have relied heavily on textbooks and our two advisors for the information contained in this program.

We have tried to be as specific as possible in describing the management of those conditions that are commonly treated by optometrists in states with therapeutic laws. Or treatment regimens are general for those conditions that are best treated by other health care professionals. Keep in mind that most therapeutic modalities described here are not the only ways to treat that particular condition but are ones that were commonly given in the references we used. Therefore, they are guidelines and not absolutes. It is beyond the scope of this program to list all of the contraindications and side effects of the drugs listed here. Please consult the Physicians Desk Reference if questions exist and to keep abreast of revised recommendations.

We realize that with a project of this magnitude and our limited clinical experience, errors and omissions may exist. Again we have strived to be as complete and concise as possible, but we recommend that you use this only as a guide and not as the sole source in treating conditions, especially those that you are not familiar with treating. Therefore, we do not imply or accept professional liability for the treatment of those conditions included in this software.

Although every possible cause for a red eye has not been included, we hope that this program is helpful to all that use it, and we welcome any suggestions or corrections so that we can include them in the next version.

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