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A world wide web informational reference source for viral ocular disease

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A world wide web informational reference source for viral ocular disease

Abstract

This World Wide Web page is a quick reference source for anyone wishing to research ocular viral disease. Doctors and medical students will find the web site helpful to aid in the diagnosis and treatment of ocular viral disease. Topics within the site are cross-linked and presented in subjective, objective, assessment, plan format (SOAP). Photographs are included as are references for further study.

Degree Type Thesis

Degree Name Master of Science in Vision Science

Committee Chair Kenneth Eakland

Subject Categories Optometry

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A WORLD WIDE WEB

INFORMATIONAL REFERENCE SOURCE

FOR

VIRAL OCULAR DISEASE

by

Kandi S. Epps, BS

Gerrie L. Lubben, BS

Michele M. Orr, BS

Nathan L. Scott

A thesis submitted to the faculty of the College of Optometry Pacific University Forest Grove, Oregon for the degree of Doctor of Optometry December, 1998 PAULINIC UNIVERSITY LIBRAL FOREST GROVE, OREGON

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Kenneth Eakland, OD

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A World Wide Web informational reference source for viral ocular disease

Authors:

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Michele M. Orr, BS

Nathan L. Scott

Advisor:

Kenneth Eakland, PD

Biographies

Kandi Epps is a candidate for an OD degree at Pacific University College of Optometry in May of 1999. She graduated from Iowa State University in 1992 with a degree in Animal Ecology. After working on the Missouri River for the state of Montana, she returned to pursue optometry. After graduation, she plans to stay in the western half of the nation to practice.

Gerrie Lubben is a candidate for an OD degree at Pacific University College of Optometry in May of 1999. She graduated from the University of South Dakota in 1994 with a Bachelor of Science in psychology and worked as a commercial optician until entering PUCO in 1995. Her plans are to become an associate at a private practice in the upper Midwest following graduation, and eventually establish her own behavioral optometry practice

Michelle Orr is a candidate for an OD degree at Pacific University College of Optometry in May of 1999. She graduated from Portland State University with a BS in Biology in 1993. She then completed research and coursework in human cytogenetics at Portland State University and Oregon Health Sciences University. She then worked as an optometric technician prior to entering optometry college. She plans to practice in the Pacific Northwest after graduation.

Nathan L. Scott is a second year optometry student. Prior to college, he worked in retail management. He graduated from Centralia College in 1996 with Associate of Arts and Associate of Science – Pre-Optometry degrees. He is the recipient of the King County Optometric Society Scholarship. He plans to return to Centralia, Washington upon licensure and practice with an emphasis on vision therapy. He also plans to be very involved in VOSH activities.

Abstract

This World Wide Web page is a quick reference source for anyone wishing to research ocular viral disease. Doctors and medical students will find the web site helpful to aid in the diagnosis and treatment of ocular viral disease. Topics within the site are cross-linked and presented in subjective, objective, assessment, plan format (SOAP). Photographs are included as are references for further study.

Acknowledgements

The authors thank Dr. Kenneth Eakland for his assistance in developing this project.

Additionally, the authors extend thanks to Mike Geraci and Jonathan Molitor for their technical assistance.

Key Words

World Wide Web, virus, optometry, ocular disease

Introduction

The World Wide Web (WWW) has become a valuable resource for obtaining information on a wide variety of topics, including health and medical treatments. An ocular disease web site was created for use by doctors and medical students to aid in clinical diagnosis and treatment; bacterial conjunctivitis was the template, completed in a previous thesis. This project is an expansion of that, and further topics will be added.

The viral web site discusses ocular viral diseases, their associated signs and symptoms, and their treatments. Photographs are included to facilitate learning. Rather than being a comprehensive resource, the viral web site is designed to allow quick access to specific disease topics. Topics within the site are cross-linked and presented in subjective, objective, assessment, plan format (SOAP).

Methods

Microsoft Word[®] 97 was used to create the text. Photographs were obtained from PUCO's Ocular Disease and Special Testing (ODST) database. The photographs were scanned then edited using Adobe Photoshop[®] 4.0. A web page template designed by Pacific University Information Services specifically for Pacific University web pages was used. The text and photographic information was assimilated into the template using Claris Homepage[®].

Forty-six individual pages are inter-linked between each other. Twenty-six pages relate to the diagnosis and treatment of ocular viral diseases. Antiviral drugs, differential diagnosis, references and a general discussion about viruses each have their own page. Information contained within each page was gathered from medical books, medical journals and class notes from Pacific University College of Optometry professors. All of the pages contain detailed text following the subjective, objective, assessment, plan format (SOAP). Photographs of the disease conditions are linked to their respective ocular disease. Since, graphics are slow to download from the internet the pictures are displayed on a linked page.

After acceptance, the completed project is tentatively scheduled to be uploaded to the PUCO web site in January 1999.

Discussion

The web site can be accessed by visiting PUCO's home page at www.opt.pacificu.edu using a web browser, such as Microsoft's Internet Explore. Once at PUCO's homepage, click on the projects link which takes the user to PUCO's faculty and student project page. Once there, clicking on the viral ocular disease link takes the user to the home page for viral ocular disease. The home page for viral ocular disease is nothing more than a welcome area and starting point for users. From this page the user decides what specific page within the ocular viral disease web site they wish to explore. By placing the cursor on the link they wish to explore and clicking the mouse, the user accesses the specific site chosen.

There are forty-six separate pages within the ocular viral disease web site. Maneuvering between pages is made easy with common links in each site. In the past, if a user had visited several linked pages s/he was forced to go back to the main index page by using the back button on the web browser. This method is time consuming because the user must revisit the linked web pages used getting to the last visited page. However, the ocular disease web site uses common links in every page allowing the user to randomly choose the next page they wish to visit. This feature allows an individual to customize their search for information.

Common to each page within the site are:

- Header image denoting the user is at the PUCO Ocular Viral Web Site
- Title of the web page below the header image
- Links for the ocular viral web pages
- Links to some of Pacific University's web sites
- References that also link to the reference page
- See Figure 1 (pages 3 & 4)

Figure 1

OCULAR VIRAL WEB SITE

Back to

College of Optometry Home

Faculty and Student Projects

Ocular Viral Disease Welcome Page

Ocular Viral Disease

Acute Hemorrhagic Conjunctivitis

Acute Retinal Necrosis Syndrome

Adenoviral Conjunctivitis and Keratoconjunctivitis EKC - Epidemic Keratoconjunctivitis ECE -Pharynocconjunctival Pavar

Varicella

HSV - Herpes Simplex

Primary Opular HSV -Epithewal Keratilis and Slephantis

Herces Simplex Kerauris

Recurrent HSV Epitheriai Keratitis

Metaneroetic Disease Tropolo Keratitis

Stromal HSV Keratitis / Chronic Interstitial Keratitis

HSV Necrotizing Stromat

Disciform HSV Keratitis

HZC - Herbes Zoster Dontnaimicus

HV - Human Immunodefectency Virus

HIV - Ocular Manifestations

<u>CMV - Cytomedalovirus</u> <u>Retinitis</u>

Molluscum Contegiosum

Figure 1 (continued)

Newcastle's Disease

Parinaud's Oculogiangular Fever

Rucella

Rubeola

Thysegon's Superficial Punctate Keratopathy SPK)

Vaccinia

. errucae / Virai Warts

Chemotherapy

Antiviral Drugs

Miscellaneous

<u>Vinuses - A Generol</u> Discussion

Differential Diagonais

References

Main Links

Main Links Home Weicome Admissions Academic Programs Stucent Life News and Events Alumni and Friends Scecial Programs Campus Resources

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Twenty-six of the pages relate to the diagnosis and treatment of viral ocular diseases. Their format follows the SOAP format. The subjective findings are listed with objective findings, assessment and plan following.

The antiviral drug page contains information about the development of antiviral drugs and patient education regarding their use, or lack thereof. The page also addresses treating viral infections with antibiotics. Next, the antiviral drugs are listed. Following each drug is a list of currently known clinical uses, form(s) of availability, side effects, contraindications.

There is a page devoted to the discussion of viruses generally. The page includes information regarding :

- Structure and Chemical Composition
- Genetic Composition
- Classification of Viruses
- Viral Replication
- Resistance
- Control of Viral Disease
- Antiviral Drugs

There are sixteen pages with photographs of ocular viral disease. The photograph pages are linked to their respective disease pages. The differential diagnosis site has an alphabetical listing of ocular diseases, including bacterial, with detailed criteria for differentiation. The reference page lists all the references used to gather information for this web site.

Conclusion

The ocular viral disease web site is designed to provide information about ocular viral disease for anyone, but especially doctors and medical students. The web site is specific enough to serve as an aid in the diagnosis and treatment of ocular viral disease.

Ocular viral diseases do not always presents with the classic signs and symptoms. Information in the web site relied mostly on medical textbooks, medical journals and class notes from PUCO professors. References are included for these sources and serve as a starting point for more in depth research. All treatment options and protocols are presented to represent the standard of care. As always, the diagnosis and final treatment protocol is determined by the practitioner.

Treatment options given in the ocular viral disease web site are not within the scope of practice for optometrists in all states. The ultimate responsibility for proper and legal treatment of an ocular viral disease lies with the optometrist or other medical professional. The therapeutic treatments listed in the web site are

the most common, but certainly are not conclusive. They are recommendations and guides, but not absolutes.

It is beyond the scope of this project to list in detail all known information pertaining to the symptoms, signs, pharmacology, diagnostic techniques and differential diagnoses for ocular viral disease. The authors strongly recommend that practitioners consult additional resources if more information is needed on a topic.

The authors realize the magnitude of the project may produce errors, omissions and unintentional additions. Even though accuracy and completeness have been striven for, we recommend that this project be used in conjunction with additional sources of information and references when diagnosing and/or treating diseases. The authors do not imply or accept professional liability for the treatment of diseases included in the web site.

Although it is not possible for all information concerning ocular viral disease to be included, it is hoped that this web site is helpful to all who use it and we encourage any corrections or constructive criticism for future updates.

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Appendix One

Structural Outline of the Web Site

- 1) Main Page
 - a) Purpose of web site
 - b) Instructions for use
 - c) Links
 - d) Authors
 - e) Release of liability
- 2) Ocular Viral Disease pages
 - a) Acute hemorrhagic conjunctivitis
 - i) Subjective
 - ii) Objective
 - iii) Assessment
 - iv) Plan
 - b) Acute retinal necrosis syndrome
 - i) Subjective
 - ii) Objective
 - iii) Assessment
 - iv) Plan
 - c) Adenoviral conjunctivitis and keratoconjunctivitis
 - i) General information
 - ii) Epidemic keratoconjunctivitis
 - (1) Subjective
 - (2) Objective
 - (3) Assessment
 - (4) Plan
 - iii) Pharyngoconjunctival fever
 - (1) Subjective
 - (2) Objective
 - (3) Assessment
 - (4) Plan
 - d) Varicella
 - i) General information
 - ii) Systemic manifestations and treatment
 - iii) Ocular manifestations and treatment
 - e) Herpes simplex virus
 - i) General information
 - ii) Etiology
 - iii) Sources of infection
 - f) Primary ocular herpes simplex virus epithelial keratitis and blepharitis
 - i) Subjective
 - ii) Objective
 - iii) Assessment
 - iv) Plan

- g) Herpes simplex keratitis
 - i) General information
- h) Recurrent ocular herpes general
 - i) Recurrent herpes simplex virus cytology
- i) Recurrent herpes simples virus epithelial keratitis
 - i) Subjective
 - ii) Objective
 - iii) Assessment
 - iv) Plan
- j) Metaherpetic disease / trophic keratitis
 - i) Subjective
 - ii) Objective
 - iii) Assessment
 - iv) Plan
- k) Stromal herpes simplex virus / chronic interstitial keratitis
 - i) General information
 - ii) Subjective
 - iii) Objective
 - iv) Assessment
 - v) Plan
- I) Herpes simplex virus necrotizing stromal keratitis
 - i) Subjective
 - ii) Objective
 - iii) Assessment
 - iv) Plan
- m) Disciform herpes simplex keratitis
 - i) Subjective
 - ii) Objective
 - iii) Assessment
 - iv) Plan
- n) Herpes zoster ophthalmicus
 - i) General information
 - ii) Subjective
 - iii) Objective
 - iv) Assessment
 - v) Plan
- o) Human immunodefeciency virus
 - i) General information
- p) Human immunodefeciency virus ocular manifestations
 - i) Subjective
 - ii) Objective
 - iii) Assessment
 - iv) Plan
- q) Cytomegalovirus retinitis
 - i) Subjective
 - ii) Objective

- iii) Assessment
- iv) Plan
- r) Molluscum contagiosum
 - i) Subjective
 - ii) Objective
 - iii) Assessment
 - iv) Plan
- s) Newcastle's disease
 - i) Subjective
 - ii) Objective
 - iii) Assessment
 - iv) Plan
- t) Parinaud's oculoglandular fever
 - i) Subjective
 - ii) Objective
 - iii) Assessment
 - iv) Plan
- u) Rubella
 - i) General information
 - ii) Systemic manifestations
 - iii) Ocular manifestations
 - iv) Plan
- v) Thysegon's superficial punctate keratopathy
 - i) General
 - ii) Subjective
 - iii) Objective
 - iv) Assessment
 - v) Plan
- w) Vaccinia
 - i) General
 - ii) Photograph
- x) Vurrucae / viral warts
 - i) Subjective
 - ii) Objective
 - iii) Assessment
 - iv) Plan
- 3) Antiviral drug page
 - a) Idoxuridine
 - i) Clinical uses
 - ii) Availability
 - iii) Side effects
 - iv) Contraindications
 - b) Vidarabine
 - i) Clinical uses
 - ii) Availability
 - iii) Side effects

- iv) Contraindications
- c) Trifluridine
 - i) Clinical uses
 - ii) Availability
 - iii) Side effects
 - iv) Contraindications
- d) Acyclovir
 - i) Clinical uses
 - ii) Availability
 - iii) Side effects
 - iv) Contraindications
- e) Ganciclovir
 - i) Clinical uses
 - ii) Side effects
- f) Foscarnet
 - i) Clinical uses
 - ii) Side effects
- g) Zidovudine
 - i) Clinical uses
 - ii) Side effects
- h) Didanosine
 - i) Clinical uses
 - ii) Side effects
- i) Zalcitabine
 - i) Clinical uses
- j) Cidofovir
 - i) Clinical uses
- 4) Viruses A General Discussion Page
 - a) Structure and chemical composition
 - b) Genetic composition
 - c) Classification of viruses
 - d) Viral replication
 - e) Resistance
 - f) Control of viral disease
 - g) Antiviral drugs
 - h) Differential diagnosis page
- 5) Differential Diagnosis Page
 - a) Alphabetical listing of common ocular diseases with detailed criteria for differentiation
- 6) Reference page
- 7) Photograph pages
 - a) Chickenpox
 - b) CMV retinitis
 - c) Corneal infiltrate
 - d) Dendrites
 - e) Subepithelial infiltrates

- f) Geographic ulcers
- g) Herpes zoster ophthalmicus
- h) Injection
- i) Molluscum contagiosum
- j) Papillae
- k) Pseudomembrane
- I) Rubella
- m) Rubeola
- n) Subepithelial opacities
- o) Subconjunctival hemorrhages
- p) Thysegon's

Appendix Two

Complete copy of Ocular Viral Disease Thesis as printed form Netscape Navigator®

OCULAR VIRALWEB SITE

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<u>purrent HSV Epithelial</u>

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omal HSV Keratitis /

Welcome to Pacific University College of Optometry Ocular Viral Web Site

This site is designed for use by anyone, but especially doctors and medical students, seeking information about ocular viral disease.

To help you navigate the web site please read the following:

The <u>viral general discussion</u> page is a good place to start if you are not familiar with viruses.

The pages that discuss specific viral diseases are patterned after the subjective, objective, assessment and plan format (SOAP). Links to pictures are included.

A listing of <u>antiviral drugs</u> is shown in the antiviral drug page. Each drug has currently known clinical uses, forms of availability, side effects and contraindications listed.

The <u>differential diagnosis</u> page has diseases with their common signs and symptoms listed to help successfully diagnosis or differentiate between viral diseases and common diseases mistaken for viral diseases.

The <u>reference page</u> lists the sources for the information contained within this web site. Additionally, at the bottom of each page is a list of references that also serve as a link to the reference page.

Use the links on the left side of this page or below to start researching ocular viral

pronic Interstitial eratitis

SV Necrotizing Stromal aratitis

sciform HSV Keratitis

20 - Herpes Zoster ohthalmicus

V - Human imunodefeciency Virus

V - Ocular anifestations

<u>MV - Cytomegalovirus</u> atinitis

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Ocular Viral Disease

Acute Hemmorhagic Conjunctivitis Acute Retinal Necrosis Syndrome

Adenoviral Conjunctivitis and Keratoconjunctivitis EKC - Epidemic Keratoconjunctivitis PCF - Pharyngoconjunctival Fever

Varicella HSV - Herpes Simplex Virus

Primary Ocular HSV - Epithelial Keratitis and Blepharitis

Herpes Simplex Keratitis Recurrent HSV Epithelial Keratitis

Metaherpetic Disease / Trophic Keratitis Stromal HSV Keratitis / Chronic Interstitial Keratitis

HSV Necrotizing Stromal Keratitis Disciform HSV Keratitis

HZO - Herpes Zoster Ophthalmicus HIV - Human Immunodefeciency Virus

HIV - Ocular Manifestations CMV - Cytomegalovirus Retinitis

Molluscum Contagiosum Newcastle's Disease Parinaud's Oculoglandular Fever

Rubella Rubeola

Thysegon's Superficial Punctate Keratopathy (SPK)

Vaccinia Verrucae / Viral Warts

Chemotherapy

Antiviral Drugs

Miscellaneous

Viruses - A General Discussion Differential Diagnosis

References

Authors

Kandi Epps, Gerrie Lubben, Michele Orr and Nathan L. Scott

RELEASE OF LIABILITY

The authors have strived to be as complete as possible in designing this site, but realize that with a project of this magnitude that errors and omissions may exist. The information contained in this site is for informational and educational purposes only, and should in no way be used as a sole source for the treatment of any condition. We sincerely hope the information is helpful and welcome suggestions and corrections.

in Links

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/ - Cytomegalovirus nitis

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ACUTE HEMORRHAGIC CONJUNCTIVITIS

Subjective

- Caused by enterovirus 70 and coxsackie virus AZ4
- Rare, affects low socioeconomic status, poor hygiene, crowded living conditions
- · Highly contagious, incubation period 8-48 hrs
- Rapid unilateral to bilateral presentation
- Self-resolves in 5-7days
- Pain
- Photophobia
- Red eyes
- Puffy eyelids
- Excessive tearing

Objective

- Bilateral conjunctival injection [see picture], subconjunctival hemorrhages [see picture], dot homorrhages to diffuse spreading hemorrhages
- Lid edema
- Profuse serous discharge
- Palpebral follicles
- Eptihelial keratitis may be seen (rare)
- Preauricular lymphadenopathy
- Generalized myalgias in 25%
- Resolution of the conjunctivitis within 5-7 days
- Motor paralysis of the lower extremities may appear after resolution of the conjunctivitis

Assessment

• R/O other infectious causes, EKC

Plan

No treatment necessary due to rapid self-resolution

References 54, 35, 31, 53

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ciform HSV Keratitis

<u>) - Herpes Zoster</u> <u>halmicus</u>

<u>- Human</u> <u>uunodefeciency</u> Virus

- Ocular

ACUTE RETINAL NECROSIS SYNDROME

Subjective

- · Mild to moderate ocular pain
- Foreign body sensation
- Red eye
- Pain on rotation
- Haziness
- Floaters
- Peripheral vision problems
- Central vision loss later
- · Herpes-type virus implicated, w/immunogenetic predisposition
- Affects 20-60 year olds
 - o HSV in younger pts
 - o VZV in older pts

Objective

- Bilateral involvement 33% of the time
- Mild to moderate conjunctival injection
- Episcleritis
- Chemosis
- Lid edema
- Subconjunctival hemorrhages [see picture]
- Mild proptosis
- Anterior granulomatous uveitis
- Possible hypopyon
- Elevated IOP's
- Retinal and choroidal vasculitis
 - o narrowing and sheathing of the larger arteries
 - o phlebitis unusual
- Retinal necrosis
 - o confluent areas of whitening which obscure underlying choroidal detail
 - o starts in periphery then moves geographically towards post pole
- Vitritis
 - o vitreous cells must be present for a diagnosis of ARNS
 - Optic neuritis
 - Exudative retinal detachment
 - Retinal and disc neovascularization
 - Vitreous hemorrhage

- anifestations
- <u>MV Cytomegalovirus</u> etinitis
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- Macular edema
- Vitreous traction
- Vitreous opacification
- Retinal holes/tears at junction of normal to abnormal retina
- Atrophic RPE in a scalloped pattern
- Occur in up to 75% of pts
 - o rhegmatogenous RD
 - o proliferative vitreoretinopathy
- Patients: <33% pts lose vision to 20/200 or worse (with intervention)
- Associated systemic conditions:
 - o HZ skin lesions
 - o HS skin ulcers
 - o acute varicella
 - o aphthous ulcerations
 - o low-grade fever
 - o mild HA
 - o sinus pain
 - nuchal rigidity
 - o viral encephalitis

Assessment

- · Self-limiting process in 6-12 wks
- Assumed that most ARNS cases due to HSV or VZV
- Diagnostic vitrectomy if signs/symptoms are equivocal
- Diagnostic tests if using acyclovir:
- o CBC
 - o creatinine
 - o blood urea nitrogen
 - o liver fx tests
 - Diagnostic tests if using systemic corticosteroids
 - o tuberculin test
 - o chest X-ray
 - Other possible diagnostic tests
 - o HIV titer
 - o RPR
 - o FTA-ABS
 - o ACE
 - o gallium scan
 - o toxo titers
 - o lumbar puncture
 - o CT or MRI
 - FA: shows early blockage of choroidal fluorescence and cut-off of blood flow
 - o acute/convalescent titers to HSV1, HSV2, VZV, CMV

Plan

- Retinal consult
- IV acyclovir x 5-10 days, then oral acyclovir (400-600 mg) 5x/day x 6 wks or foscarnet for acyclovir-resistant herpesviruses
- Antithrombotic, aspirin 125-650 mg 1-2x/day
- High dosage corticosteroids questionable efficacy
- Prophylactic photocoagulation posterior to active area
- Surgical reattachment of retina
- Scleral buckle
- Pars plana vitrectomy

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ciform HSV Keratitis

<u>) - Herpes Zoster</u> hthalmicus

<u>- Human</u> nunodefeciency Virus

- Ocular nifestations

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ADENOVIRAL CONJUNCTIVITIS and KERATOCONJUNCTIVITIS

General Information

- · Can be caused by several adenoviral serotypes
- EKC and PCF are highly contagious for 2 weeks
 - o follow sterile procedures: glove up, sterilize equipment, wash hands o patient education
 - limit exposure to others while ocular discharge is present (stay home from work and school)
 - wash hands frequently
 - don't share towels
 - don't shake hands
 - don't touch anything to your eyes
 - □ symptoms may worsen before they improve

Jump to EKC or PCF

EPIDEMIC KERATOCONJUNCTIVITIS (EKC)

Subjective

- Caused by adenovirus serotypes 3, 8, 19, or 37
- Can affect any age group
- Highly contagious
- No systemic symptoms
- Frequently occurs in the fall
- FB sensation
- Photophobia
- Pharyngitis
- Watery eyes
- Red conjunctiva
- Itchy eyes

Objective

- Bilateral but asymmetric follicular conjunctivitis with preauricular lymphadenopathy
- If severe, may see subconjunctival hemorrhages, chemosis, and pseudomembranes
- Watery, serous discharge

- suloglandular Fever
- ibella
- ibeola
- ysegon's Superficial inctate Keratopathy PK)
- accinia
- rrucae / Viral Warts
- nemotherapy
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- Purplish-pink bulbar conjunctiva
- · Swelling and erythema of the eyelids
- · Follicles may produce rugae-like folds in the lower cul-de-sac
- Associated keratitis in 80% of cases:
 - o Stage 1
 - a. within 7-10 days of onset
 - b. diffuse punctate epithelial keratitis
 - c. may self-resolve in 14 days or progress to Stage 2
 - o Stage 2
 - a. white subepithelial opacities develop beneath the epithelial lesions [see picture]
 - b. possible to the adenovirus
 - c. can be associated with mild uveitis
 - o Stage 3
 - a. anterior stromal infiltrates which may persist for months to years

Assessment

 R/O other causes of red eye: allergic, bacterial, foreign body, episcleritis, sleritis, anterior uveitis, and other viral, including PCF, HSV, HZO, acute hemorrhagic latrogenic or history of contact with infected individuals

Plan

- Mainly supportive therapies: lubricants (4-8 times daily for 1-3 weeks), cool compresses several times daily for 1-3 weeks, vasoconstrictor/antihistamine (ex. Naphazoline) gid if itching is severe, and topical NSAIDS
- Historically, antivirals have been ineffective, but phase-3 clinical trials indicate cidofovir may prove useful against adenoviral conjunctivitis
- If visual acuity is reduced or pseudomembranes are present, corticosteroids (ex. FML or Pred Mild) can be used qid for 1 week then slowly tapered
 - use steroids only if herpes simplex has been ruled out (note that the steroid will not shorten the course of the infection)
 - o remove pseudomembrane prior to initiating steroid therapy to prevent symblepharon
- F/U 1-3 weeks or PRN

Jump top of page, EKC, or PCF

PHARYNGOCONJUNCTIVAL FEVER (PCF)

Subjective

- Caused by adenovirus serotypes 3, 4, or 7
- More common in children
- Highly contagious
- Photophobia
- FB sensation
- Sneezing, sniffling, and low-grade fever precedes ocular involvement by 1-2 weeks
- Watery eyes

- Red conjunctiva
- Itchy eyes
- Abrupt onset, usually unilateral with involvement of fellow eye in 1-2 days via autoinoculation

Objective

- Upper respiratory involvement
- · Follicular conjunctivitis with preauricular lymphadenopathy
- If severe, may see subconjunctival hemorrhages, chemosis, and pseudomembranes
- · Watery, serous discharge
- Purplish-pink bulbar conjunctiva
- · Swelling and erythema of the eyelids
- · Follicles may produce rugae-like folds in the lower cul-de-sac
- Associated keratitis: small epithelial infiltrates in 30% of cases [see picture]

Assessment

 R/O other causes of red eye: allergic, bacterial, foreign body, episcleritis, sleritis, anterior uveitis, and other viral, including EKC, HSV, HZO, acute hemorrhagic latrogenic, contact with infected individuals, hot tubs or swimming pools

Plan

- Mainly supportive therapies: lubricants (4-8 times daily for 1-3 weeks), cool compresses several times daily for 1-3 weeks, vasoconstrictor/antihistamine (ex. Naphazoline) qid if itching is severe, and topical NSAIDS
- Historically, antivirals have been ineffective, but phase-3 clinical trials indicate cidofovir may prove useful against adenoviral conjunctivitis
- F/U 1 week or PRN

Jump top of page, EKC, or PCF

References

54, 13, 24, 52, 44, 3, 6, 45, 10, 27, 29, 37, 30, 51, 43, 23, 7, 9, 48, 2, 20, 25, 39

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VARICELLA

General Information

- VZV: the varicella zoster virus (herpes virus varicellae) which causes both varicella (chickenpox) in childhood and herpes zoster (shingles) in adulthood
- Virus remains latent in trigeminal ganglion or in sensory ganglia of spinal cord
- 20% develop Herpes zoster due to either reactivation of latent virus or reexposure to it
 - o if it originates from trigeminal ganglion, HZO occurs
 - Nearly everyone contracts varicella eventually, often by age 9yrs in populous societies
 - o recent development of Varicella vaccine, although HZO may still occur
 - VZV is present in respiratory secretions and cutaneous lesions
 - Incubation period of 10-21 days
 - Has systemic and ocular manifestations

Systemic Manifestations and Treatment

- Fever and malaise
- Mucocutaneous exanthem
- · Maculopapulovesicular rash, especially on face and trunk rather than extremities
- Mucosal vesicles less common
- · Vesicles rupture and crust after 1 wk and are no longer contagious
- · Scarring is unusual without a secondary bacterial infection
- More severe in infants and adults
- Treatment: analgesics, antipyretics, antipruritics, and general hygiene measures o AVOID aspirin

Ocular Manifestations and Treatment

- Eyelid vesicles
- Nonspecific papillary conjunctivitis
- · Pocks on the conjunctiva, limbus, peripheal cornea
 - o round, focal accumulations of inflammatory cells, occasionally associated with superficial ulceration and nearby hemorrhages
 - o conjunctival lesions are harmless, but corneal ones can produce scarring and vascularization
 - o usually occur during active varicella
 - may harbor live virus
 - o Treatment (corneal) with topical antiviral (ex, trifluridine)
 - also develop wks or mths later as sterile, phlyctenule-like immunologic reactions
 - last for 1-2 wks and resolve spontaneously
 - o Treatment (corneal) with topical corticosteroids
- Punctate or dendritic epithelial keratitis
 - o occurs typically, but rarely, during active varicella
 - o proliferation of VZV in corneal epithelium

ibella

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- therapeutic SCLs
 - avoid steroid use if possible

resembles herpes simplex disciform keratitis
 lasts for 2-5 mths (vs. 5-10 mths for HSV)

o resembles herpes simplex keratitis

o heaped epithelial dendrites (vs. ulcerated dendrites in HSV)

o less responsive to antiviral therapy but recommended with debridement

o corneal hypoesthesia

o less severe corneal scarring

□ mild topical ab

a hypertonic agents

cycloplegics
 lubricants

Disciform keratouveitis

o Treatment :

- If congenital: o microphthalmos
 - o chorioretinitis
 - o cataracts
 - o microcephaly
 - o deafness
 - o cardiac anomalies

References 6, 10, 28, 54, 52, 24, 44, 13, 8

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<u>- Human</u> <u>unodefeciency</u> Virus

- Ocular

HERPES SIMPLEX VIRUS

General Information

- Most common virus in humans
- Leading infectious cause of blindness (>1.5 million cases of blindness per yr)
 - Highest risk of primary infection from age 6 mo. to 5 -10 yrs
 - o 70% kids infected by age 5 yrs
 - o 90% of these are subclinical
 - o 70% immune to HSV by age 15-25 yrs
 - o 90% immune to HSV by age 60 yrs
 - Recurrence occurs in 25% of HSV infections
 - o 33% recurs once or more per yr
 - o 67% recurs once or more per 2 yrs
 - o after 1st recurrence: 50% risk of recurrence
 - o after 2nd recurrence: 75% risk of recurrence
 - o after 3rd recurrence: 100% risk of recurrence
 - Males recur 50% more than females
 - Climate may be a factor
 - · Recurrence can present ocularly in spite of non-ocular primary infection

Etiology

- Primary infection
- · Recurrent infection (after primary attack)
- Transmission from symptomatic or asymptomatic carrier
- Ocular
 - o primary epithelial (infectious) disease
 - o recurrent epithelial (infectious) disease
 - o metaherpetic (postinfectious iinflammatory) disease
 - o stromal (sterile immune) disease
 - o disciform (sterile disease of questionable etiology)

Sources of infection

- HSV-1 (90% of oral lx)
- HSV-2 (90% of genital Ix)
- · Transmission is through direct contact

anifestations <u>AV - Cytomegalovirus</u> <u>stinitis</u> <u>alluscum Contagiosum</u> <u>swcastle's Disease</u> <u>rinaud's</u> <u>suloglandular Fever</u> <u>ibella</u> <u>ibeola</u> <u>ysegon's Superficial</u> <u>nctate Keratopathy</u> <u>accinia</u> <u>rrucae / Viral Warts</u> <u>semotherapy</u>	 o mostly mouth and saliva o contact with active skin lesions (vesicles) o incubation period of ~1 wk Most common site of 1x is mucocutaneous border of lips o cold sores and fever blisters Inciting factors for recurrence Sunlight Mild trauma Extreme heat or cold Fever Steroids (topical or systemic) Infectious disease (systemic or ocular) Poor general health Surgery Epilation Immunosuppressive agents Menstruation Psychiatric disturbances 	
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PRIMARY OCULAR HSV / EPITHELIAL KERATITIS AND BLEPHARITIS

Subjective

- Usually infants and young children (ages 5-15)
- · Occasional history of 2 day to 2 wk contact with infected host
- Fever or malaise, mild to moderate
- Unilateral red eye, rarely severe
- Blurred vision
- Mild FB sensation, grittiness
- Mild photophobia
- Burning irritation
- Pain
- Tearing
- Skin rash
- Irritibility
- Possibly no symptoms

Objective

- Skin lesions (vesicles or pustules) [see pictures]
 - o mostly mucocutaneous border of lips
 - o often adjacent to lid margins (single or groups)
 - o often buried between lashes
 - o may be found elsewhere on face, lips, in nose, on trunk
 - o progress to crusting
- Lids mildly to moderately swollen, lower more than upper
 - o ulcerative blepharitis
- Regional lymphadenopathy (mild to severe)
 - o ipsilateral preauricular node
 - o often face and neck adenopathy
- Conjunctival injection and edema [see picture]
- Follicles
 - o often in fornices, rarely at limbus
- Watery discharge
- Fine to coarse SPK may appear within 2 wks
 - o may be greater inferiorly
 - o fine stains poorly with NaFI
 - o stellate keratitis may appear
 - o coarse SPK may progress to dendrites stain well with Rose bengal
- Subepithelial infiltrates may appear within 2-3 wks [see picture]
 - o central or peripheral
- More severe cases
 - o bulbar conjunctival hemorrhages
 - o pseudomembranes
 - o phylectenule-like lesions on globe
- Retinitis
 - o rare
 - o bilateral in neonates with severe systemic HSV infection
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Assessment

- 1. Staph infection
- papillae present with Staph, follicles with HSV
- 2. Impetigo
 - adenopathy too pronounced
- 3. PCF
 - corneal involvement too severe
 skin lesions
- 4. Chlamydia
 - no mucopurulence
- 5. EKC
 - wrong age group no rule of 8's
 - a systemic involvement
- 6. Herpes Zoster
 - pain not as severe
 - vesicles to pustules to ulcerations
 - midline not respected
- 7. Molluscum Contagiosum
- 8. Vaccinia

Plan

- Co-manage with PCP
- Culture in doubtful cases
- Mild abrasion of skin lesions with washcloth
 - o Acyclovir ung
 - o q4h x 21 days
 - o alternate tx: alcohol scrubs
 - o tid x 14 to 21 days
 - o supportive systemic tx: Acyclovir, aspirin, ibuprofen, cold packs
 - o oral Acyclovir (200mg): Sig 1 cap q4h (5x/day) x 7-10 days
 - o Tylenol with codeine #3 (30mg): 1-2 caps/tabs q4h or PRN
- Cycloplege if ciliary spasm is present: Cyclopentalate 1% or 2% tid
- Viroptic drops if keratitis present
 - o SPK: q4h x 1 wk or dendrite(s): q2h x 1 wk
 - o qid x 2nd wk
 - o tid x 3rd wk
 - o bid x 4th wk
- If no keratitis, optional tx:
 - o ocular lubricants
 - o prophylactic AB
- Sunglasses
- Educate on recurrence risks and "triggers"
- F/U
- o every 3 days until cornea clears
- o every 5 days until skin lesions resolve
- o look for:
- o bacterial/fungal superinfection
- o preseptal cellulitis
- o stromal involvement
- o avoid steroids
- o corneal specialist consult
- o annual check of eyes and lids or PRN

References - for all HSV information 6, 10, 28, 54, 52, 24, 44, 13, 8

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HERPES SIMPLEX KERATITIS

General Information

- By early adulthood neutralizing antibodies are present up to 90% population
- Primary infection is subclinical in 85-90% of cases and frequently goes undiagnosed
- Usually self-limiting but occasionally fatal
- Virus remains latent in trigeminal and/or superior cervical ganglia
- Estimated 300,000+ ocular cases diagnosed/yr in US
- Approx 0.15% of US population with history of external ocular HSV infection
- Transmission occurs via direct contact or contaminated secretions o tears, saliva, respiratory and genital secretions
- Incubation period of HSV-1 is 3-9 days
- · Herpetic infection more common in patients with atopic disease
- · Rarely with significant stromal involvement

Recurrent ocular herpes - general

- Cornea is principal target tissue
- Males infected 2:1 over females
- Many triggers, both endog/exogenous eg, strong sunlight, fever, menstruation, psychiatric disturbances
- Attacks more freq. in autumn and winter
- Most commonly dendritic, ameboid, or punctate epi keratitis
- w/repeated attacks stromal keratitis and assoc. uveitis may appear
- Disciform keratitis or more heavily infiltrated stromal keratitis may dvp w/o apparent preceding epi herpetic keratitis
- Stromal keratitis: permanent structural damage to cornea and rest of eye affects vision
- Resistant to tx
- Initial episode has 25% risk of recurrence w/in 2 yrs
- Second episode has 43% risk
- Bilateral in ~11% cases

Recurrent HSV - cytology

- ix may occur as a result of reactivation of the virus in latently infected cells;
- Traditionally thought to be caused by reactivation of the virus in the trigeminal ganglion and then transferred to corneal epi cells and keratocytes. If favorable conditions exist in the epi, viral replication and cell lysis occur, producing clinical disease.
- Main cell type present in recurrent inflam process is monocyte (PMN in primary)

References - for all HSV information 6, 10, 28, 54, 52, 24, 44, 13, 8

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Ocular ifestations

RECURRENT HSV EPITHELIAL KERATITIS

Subjective

- Possibly asymptomatic
- History of previous herpetic attacks
 - o ocular
 - o cold sores/fever blisters
 - o mouth ulcerations
 - o skin lesions
 - o periocular
 - o mouth or skin
 - o genital region
- Stress factors
- Corneal pain/irritation/foreign body sensation
 - o more severe in initial recurrences with increasing hypoesthesia
- Variable photophobia
- Burning
- Redness
- Variable VAs
- Tearing

Objective

- Unilateral
- Follicular conjunctivitis
- Moderate to severe bulbar hyperemia
- Occasional bulbar conjunctival hemorrhages
- Ocassional pseudomembranes [see picture]
- Ipsilateral preauricular lymphadenopathy
- Photophobia and ciliary spasm may be severe
- Quick TBUT
- Keratitis
- Secondary anterior uveitis without KPs
- Dendrites (75-80%) [see pictures]
 - o slightly depressed fine linear branching patterns with terminal end bulbs 0.1-1.0mm wide and up to 10mm long
 - o single or multiple o surrounded by SPK
 - o central or peripheral
 - o cells lining edge of ulcer are laden with virus and stain with rose bengal
 - o necrotic cells in ulcer bed stain with NaFI

<u>IV - Cytomegalovirus</u>

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- o frequent stromal edema around dendrite
- o after several days infiltrates may appear in underlying stroma
- Early on, lesion confined to epithelium but with time anterior stroma is involved
- Lesions may occur on any area of cornea but recurrences often affect same area as previous attacks
- Ciliary injection can be intense and out of proportion to symptoms
- Vesicles or ulcers may develop on lids, face, mouth and nose, or elsewhere
- Superficial vascularization not uncommon
- Dendrite may desquamate into larger geographic ulcer [see picture]
 - o often occurs after steroid use0.
 - o stromal involvement may increase
 - o uveitic reaction may become more intense
 - o eventually stromal keratouveitis may dominate

Assessment

- · Check corneal sensitivity before instilling topical anesthetic
- DDX primary vs recurrent herpes
- R/O bacterial and other possible infectious keratitis and viral keratitis o R/O CL-related pseudodendrites
 - o no skin involvement
 - o no branching of dendrite
 - o no terminal end bulbs
 - o stain minimally
- True dendritic ulcer is pathognomonic of HSV
- All other forms of dendriform keratitis are infiltrative keratitis
 - o RCE
 - o keratitis sicca
 - o Theodore's SLK
 - o Thygeson's SPK
 - o atopic keratoconjunctivitis
 - o vernal keratoconjunctivitis
 - o herpes zoster ophthalmicus
- Ulcerative dendrite depressed while infiltrative is raised
- Giemsa stain with scrapings of corneal or skin lesion
 - o multinucleated giant cells
- ELISA testing
- Viral culture

Plan

- Goals: to reduce risk of significant stromal involvement and minimize scarring
 o epithelial lesions can heal without scar formation
 - o superficial scarring occurs with stromal involvement
 - o with repeated scarring, visual loss is permanent
- Optional debridement (may increase risk of spreading virus)
- Viroptic drops q1-2 hrs for 48-72 hrs and
 - o Vidarabine ung (Vira A) hs with frequent reinstillation during the night
 - reduce drops to q3-4 hrs (with Vira A hs) after 3-5 days or on dendrite regression
 - o c/t for 14-21 days or until rose bengal and NaFI are negative
 - o then taper to qid or tid during last week
- Or rarely (due to toxicity) idoxuridine ung 5x daily or idoxuridine gtt qh
- Lesions which occur within 2 mm of limbus are more resistant to antiviral treatment than central lesions
- · Watch for antiviral toxicity (usually at 5-8 days) may take weeks to resolve
 - o follicles
 - o chemosis
 - o fine/coarse SPK
 - o retarded epithelial healing

- Cycloplege/dilate if indicated
- NSAIDs, especially in first 48 hrs
- Cold packs
- Ocular lubes
- Topical antibiotic drops as prophylactic/therapeutic agents ?????
- Sunglasses
- · Rarely patching for relief
- NO STEROIDS in epithelial keratitis
- F/U
- o every 24-48 hrs x 1st week
- o every 3-5 days x 2nd week
- o weekly for 2-3 wks after resolution
- o every 6 mths x 1st year
- o annually or PRN
- Steroid-enhanced ulcers should be seen daily until definite healing begins
- Debrided cases should be seen every 2-3 days until healed
- o repeated debridement is sometimes necessary
- Watch for formation of metaherpetic ulcers
 - o loss of dendritic shape to become round
 - o with possible deeper, indolent ulcers
 - o considerable stromal edema or infiltrate
- Counsel on recurrent nature of disease

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ciform HSV Keratitis

<u>) - Herpes Zoster</u> <u>halmicus</u>

<u>- Human</u> nunodefeciency Virus

- Ocular nifestations

<u>/ - Cytomegalovirus</u> <u>nitis</u>

METAHERPETIC DISEASE/TROPHIC KERATITIS

Subjective

- History of epithelial HSV
- Active dendriform epithelial involvement or inactive recurrent disease

Objective

- Metaherpetic (trophic) keratopathy
 - o chronic "sterile" indolent ulceration from dendriform keratitis (immune) due to persistent defects in epithelial basement membrane
 - o anterior stromal damage
 - o postinfectious inflammatory response
- Oval, ameboid, or geographic epithelial ulcer 2-8mm
- · Edges are raised, thickened, rolled or heaped gray borders
- Often vertically oriented
- Stains brilliantly with rose bengal
- Anterior stromal infiltration and edema
 - o below and larger than overlying dendrite
 - o may produce collagenolytic scarring and corneal melting
- Neovascularization

Assessment

- DDX from dendritic form by
 - o shape/edges
 - o diffuse/deeper stromal response
 - o resistance to antivirals
- DDX from stromal form by
 - o persistent NaFI and rose bengal staining
 - o chronic epithelial involvement

Plan

- Debridement
- Same primary care as epithelial HSV disease
- · Various classes of antiviral drugs at full strength for full cycles and recycles
- Bandage soft CL

	 Intermittent pressure patching 		
bliuscum Contagiosum	 Heavy lubricant therapy 		
wcastle's Disease	 Acetylcysteine (Mucomyst) for corneal melting 		
	 Cyanoacrylate glues 		
irinaud's	 Investigational antiviral agents 		
uloglandular Fever	 Surgery (last resort) o conjunctival flap o keratoplasty 		
		ibeola	o guarded to poor prognosis
		vesser's Cursticial	• F/U
inclate Keratonathy	o every 24-48 hrs x 1st week		
PK)	o every 3-5 days x 2nd week		
<u>iccinia</u> irrucae / Viral Warts nemotherapy	o weekly for 2-3 wks post resolution		
	o every 6 months x 1 year		
	o annually or PRN		
	 Counsel on recurrent nature of disease o patient should RTC with any "red eye," even without pain 		
		tiviral Drugs	
		scellaneous	Deferences for all HSV information
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STROMAL HSV KERATITIS/CHRONIC INTERSTITIAL KERATITIS

General Information

 Onset is important because deeper ocular structures are involved, vision is threatened, and morbidity is significantly increased

Subjective

- History of recurrent epithelial or stromal keratitis
- Occurs in 3% of dendritic ulcers
- Corneal pain often absent or minimal
- · Watering eye
- Deep throbbing HA pain from secondary keratouveitis
- Vision reduced, often substantially
- Insidious onset
 - o acute epithelial response may follow stromal response

Objective

- Intensity varies
- Runs a chronic, indolent course for many months
- Stromal infiltration with an intact epithelium and mild to moderate edema
 o deep to full-thickness infiltrates
 - o may resemble bacterial or fungal disease
 - o infiltration exceeds edema
- · Patchy infiltrative response resulting in permanent scarring
- · Resolution often leads to formation of a dense, white, vascularized scar
- Superficial and deep stromal vessels often accompany infiltrate
 - o active or ghost neo vessels
- Limbal vasculitis: edematous, hyperemic reaction; focal or multiple quadrants
- Ulcerative necrosis
 - develops as creamy homogeneous stromal breakdown to abscess
 stromal thinning
 - a loada ta cornaal mali
 - o leads to corneal melting
- Wessely rings often seen in anterior stroma
 - partial/complete ring of infiltrate surrounding main stromal lesion and separated by clear zone of cornea
- Epithelial edema diffuse and bullous
 - o ground-glass appearance
 - o recurrent bullae over stromal lesion, breaking down to form ulcers

olluscum Contagiosum

wcastle's Disease

- irinaud's suloglandular Fever
- ibella
- ibeola
- vsegon's Superficial Inctate Keratopathy PK)
- iccinia
- rrucae / Viral Warts

nemotherapy

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- Punctate erosions stain with rose bengal and NaFI
- KPs some becoming pigmented
- Guttata common but reversible
- Fibrin plaques on endothelium adjacent to stromal lesion
- Keratouveitis
 - o trabeculitis with IOP increase (severe cases)
 - o RBCs may appear among cells and flare
 - o varying in degree to hypopyon
- Posterior synechiae
- Rubeosis iridis
 - Loss of corneal substance minor faceting to gross thinning or perforation
 - Descemetocele formation (advanced cases)
 - Hard white/yellowish lipid deposits may appear in last healing stages

Assessment

- Deep keratitis in nonsyphilitic patients is presumed HSV stromal keratitis
 o FTA-ABS in inactive cases
 - o FTA-ABS and VDRL in active cases
- R/O
- o Herpes zoster ophthalmicus
- o Cogan's interstitial keratitis
- o Staph keratitis
- o stromal corneal dystrophies
- o neo and thinning are not present in dystrophy
- o systemic disease
- o Epstein-Barr virus
- o mumps
- o collagen vascular disease
- o bacterial, fungal, acanthamoebal disease

Plan

- Goals
 - o minimize permanent ocular damage from recurrent episode
 - o avoid iatrogenic disease
 - o counter the socioeconomic effects of a chronic, debilitating disorder
- Counsel on recurrent nature, guaranteed scarring, cosmetic and functional
- Loss, extensive ongoing steroid therapy
- Mild stromal keratitis not involving visual axis:
 - o topical lubricants
 - o cycloplegics
- If keratitis progresses with increasing infiltration and vascularization, if visual
 - axis is threatened, or if symptoms cannot comfortably be controlled:
 - o initiate corticosteroids with topical antiviral cover
 - o prednisolone acetate (0.125% to 1%) 4-8 x daily
 - o taper steroid every 1-3 wks over several wks to mths
- trifluridine bid to qid taper and watch for toxicity
 Prophylactic antibiotic therapy is unnecessary
- If epithelium ulcerates, steroid should be reduced or discontinued
- A replaced on the second should be reduced of discontinued
 A replaced on the second should be reduced of discontinued
- Oral acyclovir used in severe stromal disease unresponsive to topical antiviral/steroid mgmt, or when topical antivirals are discontinued due to toxic epitheliopathy
- Refer to appropriate corneal specialist
- Specialty care may include
 - o topical steroids
 - o antivirals
 - o cycloplegics
 - o prophylactic ABs
 - o oral acyclovir
 - o penetrating keratoplasty

· Guarded to poor prognosis, especially with neovascularization moving centrally

References - for all HSV information 6, 10, 28, 54, 52, 24, 44, 13, 8

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/ - Cytomegalovirus nitis

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castle's Disease

naud's loglandular Fever

HSV NECROTIZING STROMAL KERATITIS

Subjective

History of previous attacks

Objective

- Believed to be caused by a direct viral infection of stroma with subsequent host immune response
- Dense yellow-white infiltration within corneal stroma, and necrosis [see picture]
- In mild cases infiltrates may be localized
- In severe cases stromal abscess may occur
 - o necrotic, cheesy-white infiltrate occupying entire corneal thickness
- Overlying epithelium often breaks down over stromal infiltrate
- Edema, ulceration, and stromal neovascularization develop
- Wessely ring may be present
- Uveitis usually present and can be severe
 - o retrocorneal membrane
 - o hypopyon
 - o synechiae formation
 - o secondary glaucoma
 - o secondary cataract
- Stromal perforation may occur
- Bacterial or fungal superinfection may occur

Assessment

Plan

· Culture if bacterial, fungal, or acanthamoebal infection is of concern

Antivirals

- o topical trifluridine 5-9x daily
- Topical steroids may be added with caution at low dosage if no virus is present
- Monitor frequently for complications:
 - o HSV epithelial keratitis
 - o avoided by using topical antiviral cover
 - o indolent ulcers (not containing actively replicating viral particles)
 - o reflect underlying stromal inflam.or antiviral toxicity in epi disease
 - tx is to not stop steroid use but to increase dose along with antiviral therapy
 - o patching/bandage CL may help healing
 - o stromal ulcers
 - epithelium may break down over a dense stromal infiltrate forming an ulcer, deepening, producing a descemetocele, which may progress to corneal perforation

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- o uveitis is more severe with possible hypopyon
- o secondary glaucoma
- o from trabeculitis
- o secondary infection
- o signaled by rapid increase in inflam signs in cornea, with ulceration and hypopyon
- o secondary cataract
- o effects of continued disease and steroid administration
- If ulceration progresses, discontinue topical steroids and prescribe oral prednisone (0.5 - 1mg/kg/day)
- . The role of oral acyclovir is unknown at this time

References - for all HSV information 6, 10, 28, 54, 52, 24, 44, 13, 8

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luscum Contagiosum

vcastle's Disease

<u>naud's</u> loglandular Fever

DISCIFORM HSV KERATITIS

Subjective

- No history of epithelial HSV although SPK or dendrite may be present
- Stromal disease occasionally associated (active or inactive)
- Tearing
- Photophobia
- Mild, dull orbital pain
- · Recurrent episodes leading to corneal hypoesthesia
- Red-eye concerns
- Acute onset of blurred vision

Objective

- · Often central, edematous, round stromal disc with well-defined borders
- · May arise with or without previous corneal ulceration
- Non-infiltrative
- Type IV immune response
- Wessley ring may be present around disc
- · Fine, granular infiltrates may be elsewhere in stroma
- Moderate keratouveitis with fine KPs on endothelium in just the involved area
- No necrosis
- No neovascularization
- In severe forms stromal edema is more pronounced with folds in Descemet's, focal bullous keratopathy, uveitis, and development of superficial and deep vascularization
- Bullae may rupture with ulceration and subsequent necrosis and melting of cornea with iritis

Assessment

- · Disciform keratitis generally considered herpetic
- R/O
 - o herpes zoster
 - o bacterial causes (ulcer)
 - o chemical keratitis
 - o systemic causes
 - o vaccinia
 - o mumps
 - o varicella
- Plan
 - Counsel on recurrent nature, guaranteed scarring with decreased cosmesis and vision, need for extensive steroid therapy
 - Highly sensitive to topical corticosteroids so use lowest dose necessary

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- o prednisolone 0.125% to 1% bid to gid
- o avoid if visual axis not involved

Prophylactic topical antiviral therapy

- 3% acyclovir ung
- Cycloplegic
- Topical lubricants
- Sunglasses
- Refer to corneal specialist
- Specialty care may include
 - o topical steroids
 - o antivirals
 - o cycloplegics
 - o heat o hypertonics
 - o hypertonics
 - o penetrating keratoplasty
- Ongoing co-management with specialist
- Watch for complications
 - o indolent ulcer overlying area of disciform keratitis
 - o 2° fungal/bacterial infection
 - o increased amt of infiltrate
 - o development of hypopyon
 - o 2° glaucoma
 - o development into stromal keratouveitis
- Prognosis guarded to poor, highly unpredictable, unremitting, and invariably destructive

References - for all HSV information 6, 10, 28, 54, 52, 24, 44, 13, 8

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- Human nunodefeciency Virus

<u>- Ocular</u> ifestations

HERPES ZOSTER OPHTHALMICUS

General Information

 Results from activation of latent varicella zoste virus in trigeminal ganglion with neuronal spread of virus through the first (ophthalmic) division of the nerve

Subjective

- Usually history of VZV (chicken pox) and/or HZV (shingles)
- More common in ages 50-70s
- Red facial rash
- · Neuralgic pain precedes vesicles by a few days
- · Vesicle and pustule formation healing as crusts in 2nd week
- Red eye
- Eye pain
- HA
- Fever, malaise
- Blurred vision
- Underlying immunosuppressive disease may be present in younger patients
 o associated with AIDs and ARNS
 - o malignancy
 - o trauma

Objective

- STAGE 1: acute lesions that develop within 3 wks of rash [see picture]
 - o lids and adnexa
 - vesicular eruption following distribution of affected dermatome (5th CN)
 - nasociliary branch involv often associated with ocular complications
 - Hutchinson's Sign
 - unilateral, not crossing midline
 - usually forehead and upper lid only
 - □ lower eyelid, cheek and jawline less commonly
 - D blepharitis
 - secondary Staph infection
 - erythema
 - Iid edema
 - vesicular lip eruption

<u>vIV - Cytomegalovirus</u>

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periorbital edema

canaliculitis

dacryoadenitis

ptosis

o conjunctiva

□ follicular conjunctivitis - rare

papillary conjunctivitis [see picture]

mucopurulent vesicular conjunctivitis

petechial hemorrhagic conjunctivitis

chemosis

o cornea

keratitis
 SPK - 50% cases

- coarse puntate keratitis
- corneal "pseudodendrites" or "microdendrites"

peripheral mucus deposits

elevated appearance

stain poorly with NaFI but well with rose bengal

u wiped easily from corneal surface

nummular lesions - 33% cases

white to brown multiple fine granular deposits

surrounded by halo of stromal haze

interstitial keratitis

fascicular vascularizing keratitis

serpiginous ulceration

□ disciform lesions - 5% cases

central cornea

preceded by nummular keratitis

associated with iritis and fine KPs

a without treatment the inflammation becomes chronic

neurotrophic keratitis

corneal hypoesthesia

calcific band keratopathy

lipid keratopathy

corneal edema

peripheral corneal ulceration

a epithelial inclusion cysts

o sclera and episclera

episcleritis - 1/3 cases

scleritis - uncommon

o iris and uvea

iritis - ipsilateral in 40% cases

iridocyclitis

may include hypopyon

iris vasculitis and ischemia

sector iris atrophy, distorted pupil and loss of pigmented border

anterior segment necrosis

choroiditis

o lens

cataract

2° to inflammation or steroids

o anterior chamber angle and ciliary processes

trabeculitis

□ glaucoma - 10% cases

2° to trabeculitis or steroids

hypotonia

□ phthisis bulbi - rare, 2° to severe ischemia of CB

o vitreous

vitritis

□ vitreous hemorrhage

o retina

retinitis - rare

□ yellow exudates, hemorrhages, vascular sheathing

neuroretinitis

thrombophlebitis

- ARN
- RD, exudative or rhegmatogenous
- perivasculitis and arteritis
- macular edema
- o pupil
 - Adie's tonic pupil
 - Horner's syndrome
 - Argyll Roberton pupils
- o optic nerve
 - □ optic neuritis 1% cases
 - retrobulbar neuritis
 - optic atrophy
 - a papillitis and papilledema
 - neuroretinitis
- o EOMs
 - isolated CN palsies from viral spread in cavernous sinus 3rd > 4th > 6th

 - □ spontaneous recovery within 6 mo
 - myositis
 - ptosis
 - orbital apex syndrome
 - diplopia
- o brain
 - cephalagia
 - hypesthesia
 - anesthesia dolorosa
 - postherpetic neuralgia
 - contralateral hemiplegia very rare and usu mild
 - zosteriform temporal arteritis and angiitis
 - □ facial palsy
 - D CVAs
 - Guillain-Barre syndrome
 - encephalitis very rare
- Stage 2: chronic lesions that may last up to 10 yrs
 - o "punched-out" scars
 - o associated hyper/hypopigmentation
 - o ptosis from lid scarring
 - o defective lid function leading to exposure keratopathy
 - o trichiasis
 - o madarosis
 - o poliosis
 - o ectropion
 - o entropion
 - o lid-notching
 - o punctal stenosis and epiphora
 - o mucus-secreting conjunctivitis
 - o lipid-filled granulomas under tarsal conjunctiva
 - o submucosal conjunctival scarring
 - o chronic scleritis leading to scleral atrophy
 - o keratitis
 - nummular
 - disciform
 - a neurotrophic
 - □ mucous plaques 5% cases
 - usually occur between 3-6 mths and last 3 mths
 - ciliary injection
 - on surface of diffusely swollen corneal epithelium
 - diffuse stromal haze
 - a easily removed
 - stain with NaFI and especially rose bengal
 - o Neuralgia 7% cases
 - constant or intermittently severe and stabbing
 - may be worse at night
 - aggravated by touch and heat
 - □ usually improves slowly in time

n may lead to depression

- Stage 3: recurrent lesions 10 yrs-post acute attack
 - frequently precipitated by sudden withdrawal/reduction of topical corticosteroids
 - o episcleritis
 - o scleritis
 - o nummular, disciform, and mucous plaque keratitis
 - o iritis
 - o glaucoma

Assessment

- Any facial rash with severe uveitis should indicate HZO
- R/O HSV
- Test corneal sensation
- Check for nummular corneal lesions which may last for mths
- Transilluminate iris for signs of atrophy
- Examine hairline scalp for evidence of postherpetic scarring and pigmentation

Plan

- Oral acyclovir (Zovirax)
 - o 800mg 5x daily x 7-10 days
 - o when given at acute vesicular cutaneous stage
 - may shorten duration and extent of disease
 - may reduce pain during eruptive phase
 - may reduce ocular complications
 - incidence and duration of ant uveitis
 - □ may reduce post-herpetic neuralgia (questionable)
- Famcyclovir
 - o 500mg 3x daily x 7 days
 - o better compliance than acyclovir
 - o faster resolution of postherpetic neuralgia
- IV acyclovir for immunocompromised pts
- Oral corticosteroids
 - prednisone 60mg po x 3 days, then 40mg x 3 days, then 20mg x 4 days, d/c
 - o may decrease postherpetic neuralgia
 - o posterior segment involvement
 - o severe cutaneous eruption
- Anti-ulcer therapy
 - o cimetidine 400mg po bid
- Adults withskin rash >3-5 days or no active lesions, or children:
 - o warm compresses to periocular skin tid
 - o bacitracin ung to lesions bid
- Children with systemic spread
 - o hospitalize
- Bacitracin ung to skin lesions bid or
- Topical cutaneous AB-corticosteroid preparations (Neo-Cortef ung or Terra-Cortril spray)
 - o 3x daily until all crusts have separated
 - o to avoid bacterial superinfection
 - o calamine and starch powder should be avoided
- Warm compresses to periocular skin tid
- Topical ocular steroids
 - o prednisolone acetate 1% gid
 - o anterior segment inflammation
 - o withheld when epithelial keratitis is present
 - o often continue for several mths then tapered gradually
- Topical ocular antivirals have little/no effect on HZO
- · Cool compresses for conjunctival involvement

- Ocular lubricants
 - o Refresh Plus gtt 6-10x daily o Refresh PM ung qhs
- Cycloplegic
 - o Cyclogel 1% 4x daily
- Possibly debridement
- F/U
- o frequently for first several wks
- o bimontly for 6 mths
- o trimonthly for 6 mths
- o yearly or PRN

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naud's loglandular Fever

HUMAN IMMUNODEFICIENCY VIRUS (HIV)

General Information

- HIV-associated illnesses
 - AIDS-related complex (ARC)
 - acute, transient mononucleosis-like syndrome
 - o persistent generalized lymphadenopathy
 - o chronic fever, weight loss, diarrhea
 - o AIDS is diagnosed when an "indicator disease" is present such as
 - 1. Pneumocystis carinii pneumonia
 - 2. Kaposi's sarcoma
 - 3. HIV encephalopathy.
- Cranial nerve palsies
- Unilateral or bilateral facial palsy
 - o occurs at the time of HIV seroconversion
 - may spontaneously resolve
 - o occurs in advanced HIV
 - search for other etiologies
 - 1. meningeal lymphoma
 - 2. herpes zoster
 - 3. cryptococcal meningitis
- Transmission
 - o through sexual intercourse
 - o receipt of contaminated blood products
 - o congenitally from an infected mother
- Groups at high risk
 - o homosexual and bisexual males
 - o IV drug abusers
 - o hemophiliacs
 - o heterosexual partners of HIV-infected persons
 - HIV has been isolated from
 - o tears
 - o cornea
 - o aqueous humor
 - HIV has not been implicated directly in development of ocular disease

References

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- o vitreous
- o retina

bella

beola

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OCULAR MANIFESTATIONS OF HIV

Cinically apparent ocular lesions can be seen in as many as 94% of HIV patients

Subjective

- Rarely visual loss
- History of HIV infection

Objective

- Bilateral
- Associated acute angle-closure glaucoma
 - o choroidal cyclocongestive type
 - o choroidal effusions
 - myopic shift
 - angle shallowing
 - elevated IOPs
- Bilateral overnight blindness
- Associated optic neuropathies
 - o syphilitic optic perineuritis
 - o CMV papillitis or neuroretinitis
 - o syphilis
 - o hepatitis B
 - o ARN Syndrome
 - rapidly progressing
 - with little vitreous reaction
 - due to VZV
- Papilledema
- Telangiectasia
- Vitritis
- Culture-negative conjunctivitis and keratoconjunctivitis
- Culture-negative peripheral corneal melting
- Orbital pseudotumor
 - o responsive to oral corticosteroid treatment
- Microvasculopathy: seen in 89-100% of AIDS patients
 - o retina
 - cotton-wool spots
 - most common retinal manifestation of AIDS
 - occur in at least 2/3 of patients with AIDS
 - often ONH and along major vascular arcades

IV - Cytomegalovirus

Iluscum Contagiosum

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occur and regress without visible sequelae over 4-6 wks

- retinal hemorrhages
 - classic Roth spot appearance
 - peripheral blot and posterior flame-shaped hemorrhages
- microaneurisms
- retinal vasculitis
- a ischemic maculopathy
- macular edema and exudates
- o conjunctiva
 - dilated capillaries at limbus
 - □ microaneurysms
 - isolated vascular segments
 - vessel segments of irregular caliber
 - sludging of blood flow
 - o optic nerve
 - ischemic optic neuropathy
- Opportunistic ocular infections
 - o always associated with disseminated disease
 - o extensive and severe may be 1st sign of a disseminated infection
 - o retinal and choroidal pathogens
 - cytomegalovirus
 - only common ocular infection
 - □ 25% of AIDS patients
 - may be an early sign of AIDS, but usually a later manifestation
 - poor prognostic sign for patient survival
 - □ cryptococcus neoformans
 - toxoplasma gondii
 - I 1-3% ocular infection in AIDS patients
 - retinochoroiditis (ocular toxoplasmosis)
 - intracranial lesions more common
 - lesions: single or multifocal, uni- or bilateral, discrete or diffuse
 - possible prominent vitreal and anterior chamber reactions
 - lesions respond to continual antiparasitic therapy
 - Mycobacterium avium complex
 - Herpes simplex virus
 - Herpes zoster virus
 - Histoplasma capsulatum
 - Candida albicans
 - chorioretinitis rarely
 - Pneumocystis carinii
 - Treponema pallidum
 - Iuetic retinitis
 - extensive, deep retinal, homogeneous yellow
 - lesions
 - resolve w/penicillin therapy, but commonly recur
 - optic neuritis
 - o ocular surface/external pathogens
 - Herpes zoster
 - may be 1st manifestation of HIV infection
 - indicates increased risk of eventual development of AIDS
 - Herpes simplex
 - epithelial lesions may be severe and prolonged
 - CMV
 - may infect the conjunctiva
 - Candida albicans
 - □ mucocutaneous infections are common
 - corneal ulcers without preexisting trauma
 - Molluscum contagiosum
 - Chlamydia trachomatis, serotype L2 (lymphogranuloma venereum)
- Neoplasms
 - o Kaposi's sarcoma
 - eyelids and lid margin

- □ initial site for development in 4% of cases
- as tumor enlarges, may cause entropion or trichiasis
- surface may ulcerate and become infected, placing eye at risk
- conj involved in 20% of AIDS patients with Kaposi's
 - bright red subconjunctival mass
 - □ small tumors resemble subconjunctival hemorrhages
 - □ often inferior cul de sac
- □ slow-growing, usually asymptomatic
- □ large tumors may require excision
- a orbit involved infrequently
- □ multifocal tumor so metastasis occurs
- irradiation causes regression with little risk to eye
- □ treatment directed towards chemotherapeutic agents
- o Burkitt's lymphoma
 - orbit
 - □ proptosis and lid swelling
 - ocular motility disturbances may be 1st manifestation
 - responsive to irradiation and chemotherapy; recurrences common
 - may be related to infection with Epstein-Barr virus
- Neuro-ophthalmic abnormalities
 - o secondary to intracranial tumors and malignancies
 - o cranial nerve palsies
 - o visual field defects
 - o pupillary abnormalities
 - o papilledema
 - o optic atrophy
 - o neurological exam with CT scan used when unexplained visual disturbances arise

Assessment

- HIV testing requires patient consent
- FA
- o microaneurysms
- o telangiectases
- small areas of capillary nonperfusion corresponding to the cotton-wool spots
- Serologic testing is available for antibodies against
 - o herpes simplex virus
 - o herpes zoster virus
 - o CMV
 - o Epstein-Barr virus
- ELISA
 - o positive results are confirmed by a Western blot test

Plan

- Zidovudine (AZT) effective against HIV
 - o systemic treatment
 - o resolution of vitritis
- For glaucoma
 - o cycloplegia
 - o aqueous suppressants (acetazolamide and timolol)

References 21, 8, 33, 22

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vcastle's Disease

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CYTOMEGALOVIRUS RETINITIS (CMV)

Subjective

- History of immunocompromise
 - o 2/3 of patients diagnosed with AIDS have CMV retinitis
 - o cytotoxic chemotherapy
 - o post-organ transplantation

Objective [see pictures]

- Unilateral or bilateral
- White opacifications resembling cotton wool spots o located in deep retina and RPE
- Full thickness retinal necrosis and edema
 o geographical yellow-white granular lesions
- · Starts at periphery or posterior pole and lesions coalesce
- Retinal hemorrhages and occlusive vasculitis at advancing border
 o branch retinal artery occlusions possible
- Retinal vascular sheathing, usually periphlebitis
- · Retinal NFL hemorrhages in necrotic area
- · Spreads to involve entire retina
- · Invariably progresses to total retinal atrophy
- · Optic nerve may be involved
 - CMV inclusions have been found in all layers of the retina and in glial cells of the optic nerve.
- Rhegmatogenous or exudative retinal detachment may occur
- Vitreal cells may occur

Assessment

- · Serologic testing is available for antibodies against CMV
 - R/O
 HIV retinitis
 - o Hiv retinitis
 - o cotton-wool spots due to other systemic diseases

Plan

- IV Dihydroxypropoxymethylguanine can cause regression in some cases
- Acyclovir is rarely effective against CMV

References 28, 14, 17, 21

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MOLLUSCUM CONTAGIOSUM

Subjective

- All ages, especially children
- Mildly irritating lump on eyelid
- Mildly contagious, commonly via autoinoculation
- Cosmetic concern

Objective [see picture]

- Multiple
- May be on lid and elsewhere on body
- Pale, waxy, pearly, elevated, round umbilicated nodule with a central core
- Cheesy material may be expressed from the core (beware of contagious viral particles)
- Ocular irritation may result from secondary conjunctivitis and keratitis

Assessment

 R/O other lumps and bumps o basal cell carcinoma

Plan

- If quiet, leave alone
 - If center is discharging cheesy or waxy material, express:
 - 1. clean surface with alcohol wipe
 - 2. loosen central core material with sharp curetting instrument
 - beware of contagious viral particles
 - Educate about contagious nature and recurrence (same lesion may require multiple treatments)

- F/U one week or PRN

- 3. squeeze out contents with fingers or cotton swabs 4. reclean surface with alcohol wipes

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NEWCASTLE'S DISEASE

Subjective

Transmitted by birds, esp. chicken droppings

Objective

- Bilateral but asymmetric follicular conjunctivitis with preauricular lymphadenopathy
- If severe, may see subconjunctival hemorrhages [see picture], chemosis, and pseudomembranes
- Watery, serous discharge
- Purplish-pink bulbar conjunctiva
- Swelling and erythema of the eyelids
- · Follicles may produce rugae-like folds in the lower cul-de-sac
- Can have an associated keratitis

Assessment

Determine history of contact with birds

Plan

- Remove infectious source and educate patient
- Self-limiting

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References

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PARINAUD'S OCULOGLANDULAR FEVER/CONJUNCTIVITIS

Subjective

- Rare condition
- Most common causes: cat-scratch fever, tularaemia, sporotrichosis, tuberculosis, syphilis, and lymphogranuloma venereum
- Systemic involvement: fever, malaise, HA

Objective

- Acute and unilateral
- Granulomatous conjunctivitis accompanied by large follicles with yellowish cores
- Extensive ipisilateral preauricular lymphadenopathy

Assessment

- Determine association with cats
- · Consider systemic involvement

Plan

- Treatment varies according to cause
- Cat-scratch fever requires systemic treatment with antibiotics and non-steroidal anti-inflammatories (aspirin)
- If no systemic involvement, treat with broad-spectrum antibiotic q4h until remission

References 32, 28, 6, 21, 15

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/castle's Disease

<u>naud's</u> Ioglandular Fever

Plan

RUBELLA (CONGENITAL RUBELLA SYNDROME)

General Information

- 15% of women of child bearing age are susceptible
- Respiratory transmission
- Natural immunity following infection is long lasting
- Risk of fetal infection due to maternal viremia: o approx 50% in first 8 weeks
 - o 33% weeks 9-12
 - o 10% weeks 13-24
- · Most ocmmon cause of congenital cataract
- Incidence is declining due to immunization and monitioring of maternal antibodies during pregnancy

Systemic Manifestations

- Spontaneous abortion and stillbirths common
- Congenital heart defects 70%
- Bilateral nerve deafness
- Microcephaly
- Intrauterine growth retardation
- Mental retardation
- Hypotonia
- Hepatosplenomegaly
- Thrombocytopenia purpura
- Pneumonitis

Ocular Manifestations

- · Cataracts in 50% due to infection in the first 6 weeks
- Microopthalmos in 15%
- Retinopathy
 - o involvement of RPE cells
 - o most commonly involves the macula
 - o retina has a salt and pepper appearance [see picture]
 - o vision usually unaffected
- Glaucoma in 10%
- Corneal haze
- Strabismus
- Nystagmus
- Optic atrophy
- Extreme refractive error
- Iritis
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Plan

RUBEOLA (MEASLES)

Subjective

- · Infectious disease of childhood caused by a paramyxovirus
- Incubation period of 10-14 days
- Fever
- Cough
- Light sensitivity
- Red eyes
- Discharge from the eyes
- Brownish pink rash

Objective [see pictures]

- History of fever
- · Conjunctivitis with mucopurulent discharge and keratitis
- Several days later, Koplik's spots start on the forehead and spread to the rest of the body, may appear on the conjunctiva and caruncle
- Secondary bacterial infection may develop
- Other ocular signs: strabismus, cellulitis, retinal edema, dacryocystitis, dacryoadenitis
- · Complications in immunocompromised:
 - o neurologic; diffuse cerebral involvement multiple focal or diffuse lesions, cerebellar syndrome, spinal syndrome, optic neuritis
 - o congestion of the optic disc, may be associated with retrobulbar neuritis
 - o vascular constriction resembling CRA occlusion may be seen acutely
 - o dilation of retinal veins, pigmentary retinopathy
 - o subacute sclerosing panencephalitis (SSPE)
 - cerebral involvement, convulsive disorders, coma, opisthotonos, mutism
 - o 75% of SSPE have ocular signs: pigmentary macular changes, temporal pallor or edema of the optic disc, optic atrophy, cortical blindness, papilledema

Assessment

- R/O other causes of red eye, especially HSV, HZO
- · History of contact with measles in the past 10-14 days

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No treatment for measles or SSPE

o supportive only unless secondary infection exists

· Live attenuated vaccine gives permanent protection, may prevent disease if given within 48 hrs of exposure

References 54, 40, 41, 49

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THYGESON'S SUPERFICIAL PUNCTATE KERATITIS

General

- Rare, chronic, bilateral occurence; may be episodic with remissions and exacerbations
- · Possibly higher incidence in women than men, esp. young adults age 15-40
- Etiology unknown, viral suggested
- Non-contagious

Subjective

- May be asymptomatic
- · Symptoms usually remit and exacerbate over short periods of 4-6 week duration
- Disease may self-resolve over a 4-7 year period
- FB sensation
- Tearing/lacrimation
- Photophobia
- Slightly decreased vision
- Burn/itch
- White eye

Objective

- White eye--no associated conjunctival, AC, or eyelid inflammation
- Bilateral but asymmetric
- Multiple gray-white, coarse, granular, intraepithelial lesions--resemble snowflakes [see pictures]
- The infiltrates are raised, of variable size, number between 12-20, and stain negatively with halos around negative staining lesions
- · Lesions may change location, size, and shape
- Corneal sensitivity usually normal

Assessment

- R/ O causes of punctate epithelial keratopathy: viral, toxic, bacterial, chlamydial, exposure, dry eye, trauma, epithelial dystrophy
- If patient wears CLs, consider lens-associated causes of SPK: material, solution, mechanical, physiological

olluscum Contagiosum

wcastle's Disease	Plan
irinaud's suloglandular Fever ibella ibeola ivsegon's Superficial inctate Keratopathy PK) iccinia	 Treatment is supportive Mild to moderate: lubricants Moderate to severe: lubricants, topical steroids (q2-4h for 10-14 days, discontinue if no response in 3-4 weeks), prophylactic gentamicin or tobramycin drops bid to qid, bandage contact lens Idoxuridine may cause long-lasting subepithelial opacities 1% trifluridine used 8-10 times daily for 14 days and then tapered may decrease symptoms F/U: weekly while undergoing therapy; if in remission, follow every 3-12 months
rrucae / Viral Warts nemotherapy tiviral Drugs iscellaneous uses - A General scussion ferential Diagnosis ferences	References 54, 3, 28, 46, 47, 18, 5, 11

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VACCINIA

General

- Autocontamination of the eye from smallpox vaccination
- · Caused lid pustules and ulcers, follicular conjunctivitis, keratitis
- · Smallpox has been eradicated so historical interest only

References





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VERRUCAE/VIRAL WARTS

Subjective

- Common benign skin tumor that can be located on any part of the body, commonly localized
- Contagious and can be autoinoculated by hands and fingers
- Slow-growing
- Asymptomatic
- Cosmetic concern

Objective

- Single or multiple nonsecreting warts
- Coloration:
 - Gray to brown to yellow
- Shapes:
 - o Verrucae planar: flat, round; most common
 - o Verrucae vulgaris: "raised, irregular mass on a broad base"
 - o Verrucae digitata: pedunculated; cauliflower-like projections on a stalk
- May shed viral particles which may cause FB sensation and mild secondary viral conjunctivitis (with possible corneal involvement)

Assessment

- R/O other lumps/bumps
 - o papilloma (nonviral)
 - o molluscum contagiosum
 - o neoplasia

Plan

- Resolution is usually spontaneous
- Advise on contagious nature of warts and hand/finger contact

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- · If causing patient no distress, do nothing
- If removal is required or requested:
 - o referral for excision
 - o chemical removal: bichloroacetic acid
 - o laser removal
 - o cryo removal (hyphercator)
- Educate patient on recurrent nature of verrucae
- F/U recheck annually or PRN

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ANTIVIRAL DRUGS

The development of antiviral drugs has been slow due to the fact that viruses depend on host cells for multiplication. It is difficult to develop antiviral drugs that are toxic only to the virus, leaving the host cell unaffected. Most antiviral drugs are antimetabolites: they inhibit viral nucleic acid synthesis.

Antibiotics will have no direct effect on viral infections. They may be used judiciously if increased susceptibility to bacterial infection is suspected, but do not use antibiotics unless you have good reason. Educate your patients!

IDOXURIDINE

Clinical Uses

- Primary and recurrent herpes simplex keratitis, and primary herpetic conjunctivitis
 - o in a primary corneal infection, in the absence of corneal ulceration administer until follicular conjunctivitis or periocular skin lesions resolve
 - for primary herpetic conjunctivitis, use antivirals prophylactically to prevent corneal involvement:
 - D 0.1% solution hourly during the day and every 2 hours at night
- Does not eradicate the latent virus in the trigeminal ganglion so it will not eliminate the recurrence of herpetic keratitis
- Ineffective against HSV infection of the skin
- Continue therapy 3-5 days after corneal healing is complete and there is no FI staining
- Limit therapy to less than 21 days to decrease the risk of corneal toxicity
- May cause long-standing subepithelial opacities in patients with Thygeson's

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SPK

• Reepithelialization time for dendritic and geographic ulcers is btwn 6 and 7 days

Availability

0.1% solution (Herplex by Allergan) for topical ocular use

Side Effects

- It does affect the metabolism of normal cells and toxic effects include superficial punctate keratopathy, corneal filaments, indolent ulceration, "ghost" dendrites, and retardation of corneal stromal wound healing, conjunctival cicatrization
- Also irritation, stinging, burning, lacrimation, conj. hyperemia, corneal erosion, edema, follicular conjunctivitis

Contraindications

- Allergy to the drug
- Some strains of HSV are resistant
 - o if no healing occurs after 14 days of use, switch drugs

VIDARABINE

Clinical Uses

- Used on vaccinia, HSV, primary and recurrent herpes simplex keratitis, CMV, VZV
 Primary clinical indication is dendritic or geographic enithelial keratitis caused by
- Primary clinical indication is dendritic or geographic epithelial keratitis caused by HSV
 - 3% ung applied 5xdaily for maximum of 21 days, continuing treatment 3-5 days after the cornea has healed
- Reepithelialization time for dendritic and geographic ulcers is btwn 6 and 7 days (p285)
- Effective alternative when IDU is not effective for HSK
 - Can be used in conjunction with trifluridine for HSK
 - o apply ung at nighttime to increase corneal contact time

Availability

• 3% ung (Vira-A by Parke Davis)

Side Effects

- · Stinging, burning, irritation, lacrimation, conjunctival hyperemia
- Follicular conjunctivitis, superficial punctate keratitis, corneal edema, corneal erosion, trophic epithelial defects, delay of corneal wound healing, and lacrimal punctal occlusion
- Less toxic and less likely to cause adverse reactions than idoxuridine

Contraindications

• Hypersensitivity, intolerance

TRIFLURIDINE

Clinical Uses

- Drug of choice for HSK
- Primary and recurrent epithelial keratitis
- 1% solution 9x daily for 14 days or until re-epithelialization; then reduce to 1 drop every 4 hours during wake time for 7 more days
- Avoid 21 days continuous administration due to ocular toxicity

Availability

• 1% solution (Viroptic by Burroughs Wellcome)

Side Effects

- Burning or stinging, conjunctival hyperemia and chemosis, corneal erosion and edema, keratitis sicca, delayed corneal wound healing, elevated IOP, ptosis, lacrimal punctal occlusion, conjunctival scarring
- Compared with idoxuridine and vidarabine, causes the least amount of local irritation and toxicity

Contraindications

• Allergy, intolerance

ACYCLOVIR

Clinical Uses

- Highly selective
 - does not disrupt DNA synthesis or replication of normal cells therefore nontoxic
- Genital Herpes
 - o oral acyclovir decreases duration of viral shedding from the lesion, decreases healing time of lesions, and decreases the severity of symptoms
 - o effectively suppresses recurrences during treatment; recurrence resumes after cessation of treatment, thus no permanent effect
- HZO
 - o decreases the incidence and severity of secondary ocular inflammatory disease like episcleritis, scleritis, keratitis, and anterior uveitis
 - o not approved for HZO but still used
 - 800 mg orally 5 times a day for 7-10 days within 72 hours of onset of vesicles
- Varicella or Herpes Simplex (ARN; PORN)
 - o IV 14mg/kg every 8 hours
 - o adequate hydration essential

Side Effects

- Overall, a remarkably safe drug
 - o oral
 - nausea, vomiting, diarrhea, HA, skin rash; less frequently anorexia, edema, leg pain, fatigue, sore throat, paresthesia, lymphadenopathy

o ung

a extremely low incidence

□ superficial punctate keratipahy, burning/stinging

Contraindications

- Hypersensitivity, intolerance
- Renal insufficiency patients should be monitored carefully, esp. if high doses are used (is this for oral or IV or ung???)

GANCICLOVIR (COP 288; ODF 109,309)

Clinical Uses

- CMV retinitis in AIDS patients and prevention of CMV retinitis in transplant patients
- No permanent effect
- IV 5mg/kg every 12hrs for 14-21 days; maintenance 5mg/kg every day or 6mg/kg 5 days per week
- Intravitreal implant (Vitrasert [ODF 109]) available for patients who cannot tolerate IV therapy
- · Maintenance oral doses: oral capsule (Cytovene) 1000mg 3 times/daily
- AIDS patients show regression or disappearance of the exudative, hemorrhagic, and periphlebitic lesions of CMV retinitis; reactiviation occurs in 27-50% despite maintenance doses; ret. det. also occur in 15-20%

Side Effects

- Retinal detachments
- Bone marrow suppression, resulting in neutropenia, thrombocytopenia, granulocytopenia

FOSCARNET

Clinical Uses

- CMV retinitis in AIDS patients who are unresponsive to or intolerant of ganciclovir
- IV: 60mg/kg every day; maintenance 90-120 mg/kg every day

Side Effects

 Fever, GI problems, renal toxicity; changes in serum calcium, phosphorus, and magnesium, leading to seizures

ZIDOVUDINE (AZT)

Cliniocal uses

- Oral administration for AIDS
- Reduces risk of opportunistic infections
- CMV retinitis

Side Effects

Bone marrow hypoplasia, increased vulnerability to bacterial infections

DIDANOSINE

Clinical Uses

Advanced AIDS patients who are intolerant or unresponsive to zidovudine

Side Effects

 Painful peripheral neuropathy, acute pancreatitis, hyperuricemia, rash, increased levels of hepatic transaminases; pigmentary retinal lesions

ZALCITABINE

Clinical Uses

Advanced AIDS patients

CIDOFOVIR (ODF 309)

Clinical Uses

- CMV retinitis
- IV 5mg/kg once per week for 2 weeks then 5mg/kg every 2 weeks; IV saline and oral probenecid given before and after each dose; dosage reduction required with increased serum creatinin

References 14, 4, 3, 13, 50, 16

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VIRUSES - A GENERAL DISCUSSION

Viruses are obligate intracellular parasites, meaning viruses lack the cellular components needed to acquire nutrients, produce energy and synthesize proteins. Viruses are not capable of independent metabolism. Viruses must invade a host cell in order to gain the components needed for almost all biological functions required for their survival.

Viruses are among the smallest biological entities. They range in size from 20 to 40nm, comparatively much smaller than the typical bacterium. A virusí size prevents it from being filtered using standard bacteriological filters. Before the advent of electron microscopes viruses were unable to be seen.

Jump to: <u>Structure and Chemical Composit Genetic Composition</u> <u>Classification of Viruses Viral Replication Viral Detection and Cultivation</u> <u>Resistance Control of Viral Disease Antiviral Drugs</u>

Structure and Chemical Composition

Viruses, in their simplest form, are strands of nucleic acid surrounded by a protective protein coat called a capsid. The capsid is made up of repeating protein units called protomers. Together the nucleic acid core and the capsid are called a nucleocapsid. In some instances, the nucleocapsid affects infectivity. Some forms of viruses use their proteins to slightly modify the host cell membrane to form an envelope. An entire infectious virus is termed a virion.

For non-enveloped viruses the host recognition sites are located on the nucleocapsid. The protein and nucleocapsid compose the virion. In contrast, enveloped viruses have their host recognition sites on the envelope. In this instance, the nucleic acid, capsid and envelope compose a virion.

The shape of non-enveloped viruses is determined by the manner in which the protomers are arranged around the nucleic acid core. Envelopes alter the appearance of a virion. Enveloped viruses may not have one common shape due to the fluidity of the envelope Enveloped viruses can be pleomorphic (have the ability to assume different shapes).

Jump to:

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Genetic Composition

Viruses are unique in that they posses only a single type of nucleic acid, either deoxyribonucleic acid (DNA) or ribonucleic acid (RNA), but never both. The nucleic acid of some viruses may be single-stranded while others may be double stranded. This results in four possible configurations:

1. Double-stranded DNA

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2. Single-stranded DNA

- 3. Double-stranded RNA
- 4. Single-stranded RNA

Classification of Viruses

Viruses are classified using set criteria. Classification is based upon the following order of importance:

- 1. Type of nucleic acid
- 2. Single- or double-strandedness of the nucleic acid
- 3. Capsid morphology
- 4. Size
- 5. Symmetry
- 6. Number of capsomers
- 7. Presence or absence of envelope
- 8. Host range

Jump to:

Structure and Chemical Composit Genetic Composition Classification of Viruses Viral Replication Viral Detection and Cultivation Resistance Control of Viral Disease Antiviral Drugs

Viral Replication

Viruses cannot reproduce via fission rather, they must rely on a host cell to produce new virions. Productive infection occurs when a virus replicates inside a host cell then releases progeny (offspring) virions. Progeny are released when the host cell is lysed, obviously resulting in host cell death. Other viruses can infect and produce progeny without destroying the host cell. Even more, there are some viruses that produce no virions when they infect host cells. Instead they integrate with the host cell's chromosomes then replicate.

Viral productive infection involves five steps:

1. Attachment

The virus must attach itself to a susceptible cell. This step requires specific interaction between the attachment site on the surface of the virus and the receptor site on the surface of the host cell.

2. Penetration and uncoating

Some enveloped viruses enter the cell via fusion with the host cell membrane. Most non-enveloped and enveloped viruses penetrate the host cell by receptor-mediated endocytosis, a type of phagocytosis.

Viral nucleic acid must be released from the capsid for reproduction to occur. The uncoating process accomplishes this. While, uncoating is a poorly understood process, most viruses are uncoated as they escape from the vesicle that results after receptor-mediated endocytosis.

3. Synthesis of viral components

At this stage the viral nucleic acid competes with the cell's chromosomes for control of the host cell's biological components. For production of progeny, the virus must replicate its chromosomal information and produce the structural

proteins for its capsid and envelop (if it has one).

Of special interest is the virus that causes AIDS. It is a single stranded RNA virus and is classified as a retrovirus. Retroviruses manufacture a double-stranded DNA copy of their RNA genome. This DNA copy integrates into the host cell chromosome and from there produces more viral genome (RNA). This inverted flow of genetic information from RNA to DNA requires a unique enzyme contained within the capsid called reverse transcriptase. Many of the AIDS therapies target reverse transcriptase.

4. Assembly of viral components

Assembly of viral nucleic acid occurs in one of two ways. The nucleic acid is packaged in a pre-assembled capsid or the nucleic acid associates with the capsid proteins during the capsid assembly process.

5. Release of progeny virus

Some host cells burst during viral release. However, most enveloped viruses leave the host cell undamaged by a process called budding. The latter process called exocytosis resembles a reversed version of penetration and results in more viruses potentially being produced because the still infected host cell is left functioning.

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Viral Detection and Cultivation

Cell cultures are the growth of possible host cells on artificial media. A virus that infects a single cell may ultimately form a plaque (a zone of clearing). Viral infection of cell cultures may induce several other morphological changes. For example, fusion of cells into larger cells with multiple nuclei, clumping of cells or development of inclusion bodies. These changes are referred to as cytopathic effects (CPEs) and are easily seen under a light microscope. CPEs are usually characteristic of a specific virus.

Some enveloped viruses may be identified via hemadsorption, the attachment of red blood cells to the surface of virus-infected cells.

Sometimes living experimental animals must be used to culture viruses

Hemagglutination, the clumping of red blood cells, can detect the presence of the influenza virus and others.

Using PCR (polymerase chain reaction), viral DNA that has integrated with its host cell chromosomes can be amplified. This technique has been used to detect papillomavirus DNA in genital lesions and HIV in newborns.

The most useful test for viral detection is the serological reaction between the antigens of the virus and an antibody of known viral specificity. As an example, if a virus obtained from an infected patient reacts with a herpes-specific body, the virus is now known.

Resistance

Interferons play a major role in viral resistance in the human immune response. Interferons are rapidly produced by virus infected cells and diffuse to non-viral infected cells. Interferons stimulate the non-viral infected cells to produce enzymes with antiviral activity. Interferons do not protect the host cell that was originally infected, however.

Additionally, the immune system can detect virus-infected cells and destroy them before progeny virions can be formed and released. The skin lesions of chicken pox are localized areas where virus-infected cells have been lysed. Viral infection also stimulates that body to produce antibodies that react with viral surface antigens and prevent attachment to potential host cells. People who recover from a viral disease are often naturally immunized to the virus that caused the disease.

Control of Viral Disease

Vaccination is the most important method of preventing the infection and subsequent spread of viral diseases. Harmless, attenuated viral variants stimulate immunity without the danger of disease.

Antiviral Drugs

Since the virus is developing inside the host cell, it is very difficult to produce drugs that are more toxic to virally infected cells while leaving the host cell undamaged. Antiviral drugs mostly attempt to selectively inhibit nucleic acid synthesis of viral infected cells.

Jump to: <u>Structure and Chemical Composit Genetic Composition</u> <u>Classification of Viruses Viral Replication Viral Detection and Cultivation</u> <u>Resistance Control of Viral Disease Antiviral Drugs</u>

References 55, 56, 58

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Differential Diagnosis of Red Eye

Acanthamoeba - severe ocular pain, redness, photophobia over a period of several weeks. much more pain than would be expected for presentation. Epithelial and subepithelial infiltrates, pseudodendrites on cornea, corneal stromal ring infiltrate, eyelid swelling, conjunctival injection, cells and flare, corneal ulceration may occur later, little discharge.

Acne rosacea - typically middle age, chronic recurring bilateral corneal irritation, diffuse bulbar and circumcorneal injection, occasionally tearing/purulence, chronic blepharoconjunctivitis, skin lesions, possibly red swollen nose.

Acute angle closure glaucoma - pain, blurred vision, colored halos around lights, frontal headache, nausea and vomiting, closed angle in the involved eye, increase in IOP with microcystic corneal edema, conjunctival injection, fixed, mid-dilated pupil, shallow anterior chamber.

Adenoviral conjunctivitis - recent upper respiratory infection, or contact with someone with red eye. First one eye then contralateral eye a few days later. Tarsal conjunctival follicles, watery mucuos discharge, red and edematous eyelids, palpable preauricular node, pinpoint subconjunctival hemorrhages, membrane, pseudomembrane, subepithelial infiltrates, may develop several weeks after onset. If associated with pharyngitis and fever = pharyngoconjunctival fever. If associated with large subconjunctival hemorrhage = acute hemorrhagic conjunctivitis.

Allergic conjunctivitis - itches, pink/purple injection, white stringy mucoid discharge, chemosis, allergic history, seasonal, hyperemia greater further from limbus.

Anterior uveitis - deep pain, photophobia, no discharge, circumcorneal/perilimbal flush, cells/flare, no relief of pain with topical anasthetic, no direct corneal involvement.

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Bacterial conjunctivitis - irritation, foreign body sensation, bright red injection, purulent/mucopurulent discharge, papillae, lids and lashes matted in morning, hyperemia greater further from limbus.

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Blepharitis/meibomianitis - crusty, red, thickened eyelid margins with prominent blood vessels and/or meibomian gland stasis. Itching, burning, foreign body sensation, tearing.

Canaliculitis - tearing or discharge, red eye, mild tenderness over nasal aspect of upper or lower lid. Erythematous punctum, erythema of surrounding skin, mucopurulent discharge or concretions from punctum with pressure over the lacrimal sac.

Chlamydia - stringy mucous discharge or eyelid sticking, red eye, ocular irritation of longer than 4 weeks duration, superficial punctate keratitis, inferior tarsal follicles, superior corneal pannus, palpable preauricular, gray-white subepithelial infiltrates peripherally. Sexually transmitted, typically teenagers and young adults. History of vaginitis, cervicitis or urethritis possible.

Contact dermatitis - sudden onset, periorbital rash or eyelid swelling, mild watery discharge, periorbital edema, erythema, vesicles, scaly skin, conjunctivitis.

Dacryocystitis - pain, redness, swelling inferior lid (nasal), tearing, mucoid or purulent discharge from punctum with pressure over the lacrimal sac, fever, may be recurrent, fistula formation, lacrimal sac cyst, or mucocele possible with chronic cases.

Episcleritis - hot, gritty, mild/moderate pain on palpation and lateral gaze, watery discharge, focal hyperemia and infiltration of the bulbar conjunctiva, wedge shaped sectorial salmon pink injection decreasing toward limbus, no corneal involvement, no/minimal anterior chamber reaction, typically young adults, may be recurrent.

Foreign body reaction - foreign body sensation, tearing, blurred vision, photophobia, history of foreign body in eye. Conjunctival injection, eyelid erythema, mild anterior-chamber reaction, superficial punctate keratitis, possibly small infiltrate surrounding the foreign body.

Fungal keratitis - red eye, mild to severe pain, photophobia, decreased vision, discharge, corneal ulcer that results from trauma with vegetative material. Raised dirty serpiginous ulcer with ragged leading edge. Posterior corneal infiltrates and iritis common. Infiltrates have feathery borders, may be surrounded by satellite lesions. Can occur without corneal trauma in immunocompromised.

Giant papillary conjunctivitis - itching, decreased lens-wearing time, increased lens awareness, excessive lens movement, giant papillae on the superior tarsal conjunctiva.

Herpes simplex - Unilateral, follicular conjunctival reaction, may be recurrent, may have skin vesicles along eyelid margin or around eyes, may have palpable preauricular node. May have dendritic keratitis. Dendrites have terminal bulbs, arboral pattern. Edges of herpetic corneal lesions have a heaped up appearance and stain well with rose bengal, central ulceration stains well with flouroscein.

Herpes zoster - Vesicular lesions of face in trigeminal nerve distribution, does not cross midline. Epithelial dendrites present on cornea, do not have terminal bulbs, do not stain well with flouroscein.

Infectious corneal ulcer - red eye, mild to severe pain, photophobia, decreased vision, discharge, focal white opacity in the corneal stroma with overlying epithelial defect. Epithelial defect stains with flouroscein, shows stromal loss. Conjunctival injection, corneal thinning, stromal edema and inflammation surrounding the infiltrate, folds in Descemet's, anterior chamber reaction, hypopyon, mucopurulent discharge, upper eyelid edema, posterior synechiae, hyphema, and glaucoma in severe cases.

Keratitis - superficial pain, pain relieved with topical anesthetic, corneal staining.

Molluscum contagiosum - dome shaped, multiple, umbilicated, shiny nodules on the eyelid or eyelid margin. Follicular conjunctival response, corneal pannus.

Pinguecula / pterygium - irritation, redness, decreased vision, may be asymptomatic. Pterygium; wing- shaped fold of fibrovascular tissue arising from the interpalpebral conjunctiva and extending into the cornea, may have an iron line (Stocker's line) adjacent to it in the cornea. Pingueculum; yellow-white flat or slightly raised conjunctival lesion, usually in the interpalpebral fissure adjacent to the limbus, but not involving the cornea.

Parinaud's oculoglandular conjunctivitis - red eye, mucopurulent discharge, foreign body sensation, fever, rash, follicular conjunctivitis. Granulomatous nodules on the palpebral conjunctiva, visibly swollen preauricular or submandibular node on same side. Commonly, history of being scratched or licked by a cat within past 2 weeks.

Phlyctenular keratoconjunctivitis - corneal irritation, localized pannus, watering, leash of vessels at phlycten, phlycten invades cornea.

Superior limbic keratoconjunctivitis - red eye, foreign body sensation, pain, tearing, photophobia, may be chronic, thickening and inflammation of the superior bulbar conjunctiva, particularly at the limbus, papillae on superior palpebral conjunctiva, superior punctate staining with flourescein, superior micropannus and filaments, usually bilateral.

Superficial punctate keratitis - pain, photophobia, red eye, foreign body sensation, small pinpoint corneal epithelial defects (stain with flouroscein), conjunctival injection, watery or mucoid discharge.

Scleriis - general malaise, severe pain, deep blue/purplish injection all the way to fornices, diffuse hyperemia, decreased corneal sensitivity, sclerosing stromal keratitis, probable uveitic response with cells/flare, high correlation with systemic connective

tissue disease.

Subconjunctival hemorrhage - red eye, mild irritation. Blood underneath the conjunctiva, often in a sector .

Trachoma - history of exposure to endemic area (N. Africa, Middle East, India, Southeast Asia). Superior tarsal follicles, mild superficial punctate keratitis, pannus, purulent discharge and tender preauricular node. Similar appearance to Chlamydia initially, but not sexually transmitted.

Trichiasis - ocular irritation, FB sensation, tearing, red eye. Misdirected eyelashes rubbing against the globe.

Toxic reaction (to drops) - inferior papillary reaction, may see a follicular response, inferior superficial punctate keratitis and discharge.

Vernal keratoconjunctivitis - itching, thick ropy discharge, seasonal (spring/summer), recurrent, young patients (usually male). Large conjunctival papillae (tarsal) or along the limbus, superior corneal shield ulcer, limbal or palpebral Horner-Trantas' dots (raised white dots), superficial punctate keratitis.

Viral conjunctivitis - burning, pink/purple injection toward plica, tearing, palpebral follicles, palpable preauricular lymph node, hyperemia greater further from limbus.

References 6,10,38,52,44,26

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