CHAPTER 26
INFECTIONS OF THE SKIN & EYES

WHY IS THIS IMPORTANT?

Skin and eyes are in contact with potentially pathogenic organisms all the time.

OVERVIEW
OVERVIEW

- Many infections can occur on the skin.
  - The skin is always exposed to pathogens.
  - Soft tissue below the skin is a breeding ground for infections.
- Eyes are open to the outside world.
  - Infections here extremely dangerous.
    - Proximity to the nervous system
    - Potential for loss of vision

ANATOMY OF THE SKIN

- Largest organ in the body.
- Barrier between our body and the outside
  - First line of defense against invading microorganisms
- Outer layer (epidermis) comes into direct contact with the environment.
  - Constant shedding of cells keeps pathogens from successfully attaching to the skin
  - Several mechanical mechanisms discourage pathogens.
    - Production of perspiration flushes pathogens away
    - Sebum is a natural antibacterial substance.
      - Produced by the sebaceous glands
    - In spite of these, skin is often breached by trauma.
      - Wounds, abrasions, punctures, and bites
    - When this occurs, pathogens can get into the underlying tissue.
      - Infection of the skin caused by bacteria, viruses, and fungi.
SUMMARY OF SKIN INFECTIONS

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<th>Infections</th>
<th>Organisms Causing the Infection</th>
<th>Characteristics of the Infection</th>
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<tr>
<td>Bacterial</td>
<td>Staphylococcus aureus</td>
<td>Skin abscesses</td>
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<td>Fungal</td>
<td>Scalp ringworm</td>
<td>Scalp lesions</td>
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<tr>
<td>Viral</td>
<td>Herpes simplex virus</td>
<td>Vesicles on skin</td>
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<tr>
<td>Parasitic</td>
<td>Schistosoma mansoni</td>
<td>Skin nodules</td>
</tr>
</tbody>
</table>

Types of skin infections:
- Macules
- Papules
- Vesicles
- Pustules

BACTERIAL INFECTIONS OF THE SKIN

<table>
<thead>
<tr>
<th>Infections of the skin</th>
<th>Organisms</th>
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<td>Maculopapular rash</td>
<td>Staphylococcus aureus</td>
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<tr>
<td>Wet nodule</td>
<td>Pseudomonas aeruginosa</td>
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<tr>
<td>Ulcerated</td>
<td>Proteus mirabilis</td>
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</tbody>
</table>

Infections of soft tissues:
- Methicillin-resistant Staphylococcus aureus (MRSA)
- Clostridium perfringens
- Gas gangrene
**BACTERIAL INFECTIONS OF THE SKIN**

- Bacteria can infect skin and soft tissue just beneath the skin.
  - Soft tissue infections accompany a break in the skin.
- Soft tissue contains blood supply.
  - Rich environment for pathogen growth.
  - Increases the possibility of systemic infection.

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**BACTERIAL INFECTIONS OF THE SKIN**

- Surgical procedures breach the skin.
  - Wound infections are important problem in hospital settings.
- Bite of dog or cat introduces organisms.
  - Can result in serious infection.

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**BACTERIAL INFECTIONS OF THE SKIN**

- Intact skin is relatively dry.
- Surface of the skin usually has a pH of 5.0-6.0.
  - Too acidic for many pathogens.
- Skin’s normal flora is an additional barrier.
  - Coagulase-negative staphylococci and other Gram-positive organisms.
Sebum can be converted to free fatty acids by normal flora.
- Inhibit growth of pathogens
- Bacteria enter the skin via minor abrasions, hair follicles, surgical, or traumatic wounds.
- Fasciitis – infection of fasciae
  - Very difficult to treat, especially necrotizing fasciitis

Patients with bacteremia can develop necrotizing fasciitis.
- Streptococci reach fasciae
- Settle in small hematomas or in bruised areas
- Once bacteria enter the fasciae, they spread rapidly.
- Resulting inflammatory response affects the neurovascular bundles.
  - Thrombosis of these vessels compromises blood supply and nerves.
  - Area rapidly becomes necrotic.
- Infection can be so rapid surgical removal of the tissue is the only option.
BACTERIAL INFECTIONS OF THE SKIN

- Surface of the skin penetrated by ducts, hairs, and sweat glands.
- Microbial invasion can occur along any route.
  - Especially through any ducts that are obstructed

FOLLICULITIS: Pathogenesis

- Minor infection of hair follicles
  - Usually caused by *S. aureus*
  - Associated with sweat gland activity
- Most often seen in areas where sweat glands predominate.
  - Neck, face, axillae, and buttocks

FOLLICULITIS: Pathogenesis

- Blockage of the gland predisposes to folliculitis.
- Serious infections result in boils (furuncles).
  - Localized region of pus surrounded by tissue inflammation
FOLLICULITIS: Pathogenesis

- Antibiotics cannot penetrate a boil.
  - Difficult to treat
- Draining the abscess initial step.
- If body defenses do not wall off the infection:
  - Neighboring tissues become infected.
    - Enlarged infected region – a carbuncle
  - Extensive damage and fever.

FOLLICULITIS: Pathogenesis

- Folliculitis can also be caused by *Pseudomonas aeruginosa*.
- Increase in these infections due to hot tubs and whirlpools
  - Temperature results in large numbers of pathogens
  - Causes large areas of folliculitis
  - Symptoms normally subside when the insult is discontinued

ACNE: Pathogenesis

- Very common skin infection
  - Affects 17 million people in US
    - 85% are teenagers
- Three categories:
  - Comedonal acne
  - Inflammatory acne
  - Nodular cystic acne
ACNE: Pathogenesis

- Comedonal acne:
  - Inflammation of the hair follicles and sebaceous glands of the face
  - Glands become plugged by shedding skin cells and sebum.
    - Whiteheads (comedos) appear on the skin.
    - Blockage protrudes through the skin.
    - Blackheads (comedones) appear.

ACNE: Pathogenesis

- Inflammatory acne:
  - Caused by *Propionibacterium acnes*
    - Predominant anaerobe of the skin
    - Metabolizes the glycerol component of sebum
    - Causes free fatty acids to form
    - Presence of fatty acids initiates inflammatory response.

ACNE: Pathogenesis

- Inflammatory acne:
  - Neutrophils secrete enzymes that damage the wall of the follicles.
    - Causes pustules and papules
  - Primary cause is hormonal influence on the secretion of sebum.
    - Increased during puberty
    - Usually resolves spontaneously in adulthood
**ACNE: Pathogenesis**

- Nodular cystic acne:
  - Characterized by formation of cysts
    - Filled with pus
    - Leave prominent scars on the skin

**FOLLICULITIS AND ACNE: Treatment**

- Folliculitis treated locally by drainage without antibiotics.
- All three types of acne can usually be effectively treated with topical drying agents.

**ERYSIPelas: Pathogenesis**

- Characterized by a rapidly spreading infection of the deeper layers of the dermis
- Always caused by group A streptococci
- Symptoms include:
  - Edema of the skin marked by erythema and pain
  - Systemic infection with lymphadenopathy and fever
- Can progress to:
  - Septicemia
  - Local necrosis of the skin
  - Serious and requires immediate treatment
**ERYSIPelas:**

**Treatment**

- Penicillin and streptomycin are effective.

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**SCALDED SKIN SYNDROME**

- Salient sign is blistering and peeling off of large sheets of skin.
- Caused by two exotoxins secreted by certain strains of *S. aureus* – exfoliatins.
  - Gene for one located on the bacterial chromosome
  - Gene for the other located on a plasmid
- Normally restricted to infants
  - Can occur in adults, especially in late stages of toxic shock syndrome

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**SCALDED SKIN SYNDROME**

[Image of skin condition]
SCALDED SKIN SYNDROME: Pathogenesis

- Exotoxins are transported through the blood to distal sites.
  - Cause the upper layers of the skin to separate and peel off
- First sign is a reddened area, usually around the mouth.
  - Soon spreads forming large, soft vesicles over the whole body

SCALDED SKIN SYNDROME: Pathogenesis

- Top layer of skin peels away
  - Exposed dermal layer looks scalded
- Condition is only temporary.
  - Skin will regenerate in 7-10 days.
- Accompanying high fever throughout infection

SCALDED SKIN SYNDROME: Treatment

- Good immune response to this infection.
  - Recurrence is unlikely.
- Most bacteria responsible are sensitive to penicillin.
  - Cephalosporins are effective for penicillin-resistant strains.
GAS GANGRENE
(CLOSTRIDIUM PERFRINGENS)

- Gangrene – tissue necrosis resulting from an obstructed blood supply.
- Bacteria responsible for the infection release gases.
- Caused by Clostridium perfringens.
- Usually associated with deep tissue wounds.

GAS GANGRENE:
Pathogenesis

- C. perfringens is a Gram-positive anaerobic, spore-forming rod.
- Spores are introduced into dead tissue.
  - Blood circulation has been impaired.
  - Environment is perfect for anaerobic growth.
- Spores germinate and bacteria multiply
  - Produce toxins which destroy tissue surrounding the already-dead tissue
- Destruction expands the anaerobic environment
  - Infection spreads
GAS GANGRENE: Pathogenesis

- Onset of gas gangrene is sudden.
  - 12 - 48 hours after initial injury.
  - Bacteria grow and produce hydrogen gas.
    - Causes breaks in the tissue – crepitant tissue.

- Movement of the affected area causes snap, crackle, and popping sounds.
- Also a foul smell
  - Obvious that infection has set in
- Infection is accompanied by high fever, massive tissue destruction, shock, and blackened skin.
- If not treated, gas gangrene is lethal.
GAS GANGRENE: Treatment

- Treatment in hyperbaric chambers is effective.
  - Kills the anaerobic pathogens
  - Penicillin and clindamycin should also be given.

CUTANEOUS ANTHRAX

- *Bacillus anthracis* causes inhalation anthrax – a serious respiratory infection.
- Can also cause less harmful infections in the skin
  - Cutaneous anthrax is one.

CUTANEOUS ANTHRAX: Pathogenesis

- First signs usually appear 2 - 5 days after anthrax spores have been inoculated into an opening in the skin.
  - Most often the forearm or hand
  - Initial lesion is a papule.
  - Looks like an insect bite
CUTANEOUS ANTHRAX: Pathogenesis

- Papule progresses through vesicular and ulcerative stages over 7-10 days.
- Forms a black eschar surrounded by edema.
- Symptoms are normally mild.
  - Lesions typically heal slowly after the scab falls off.
- Infection can become systemic.
  - Progresses to massive edema and toxemia
  - Can be fatal

CUTANEOUS ANTHRAX: Treatment

- Antibiotics have little effect.
- *Bacillus anthracis* is susceptible to ciprofloxacin.

VIRAL INFECTIONS OF THE SKIN

- Same barrier constraints that apply to bacteria apply to viruses.
  - Needs to be an entry point
- Several common viral infections manifest their signs on the skin after systemic infection.
MEASLES

- Extremely contagious infection
- Caused by single-stranded RNA virus
- Leading cause of vaccine-preventable disease worldwide
- Common forms include:
  - Rubeola (lasts 5 days)
  - Hard measles (lasts 7-18 days)

MEASLES

- Measles virus can produce severe infection in children.
  - High fever, widespread rash, and transient immunosuppression
- Usually occurs in preschool children
  - Those who have not yet been vaccinated (MMR vaccine).
- Only one serotype of measles
  - Shows some antigenic drift

MEASLES: Pathogenesis

- 9-11 days after exposure:
  - Infection begins in respiratory tract.
  - Cough, runny nose, and fever.
- Initial signs followed by:
  - Viremia
  - Lymphatic spread of the virus throughout the body
    - Lymph tissue, bone marrow, and skin

Extremely contagious infection
Caused by single-stranded RNA virus
Leading cause of vaccine-preventable disease worldwide
Common forms include:
  - Rubeola (lasts 5 days)
  - Hard measles (lasts 7-18 days)
MEASLES: Pathogenesis

- Virus can be present in the blood during first week of illness.
  - Viruria can persist for up to four days after rash.
- Immunity is suppressed.
  - Susceptibility to bacterial superinfections
- Koplik’s spots appear on the mucous membrane of cheeks
  - 1-3 days after respiratory signs and before the skin rash.

MEASLES: Pathogenesis

- Characteristic red skin rash appears 1 day after the Koplik’s spots.
- Significant numbers of virions are found in:
  - Koplik’s spots
  - Areas around rash
- Lymphadenopathy is common.
MEASLES:
Pathogenesis

- Measles can be very severe in immunosuppressed individuals.
  - Can be lethal
- 15-25% mortality rate in developing countries.
- Up to 15% of cases of measles have complications.
  - Otis media, sinusitis, mastoiditis, pneumonia, and sepsis
  - 1 in every 1000 cases develop encephalitis
    - Permanent nerve damage or death

MEASLES:
Treatment

- No therapy other than supportive care.
- Close observation is required for potential complications.
- Very effective vaccine (part of MMR) is routinely given to children.

RUBELLA (GERMAN MEASLES)

- Very mild or asymptomatic infection
  - Low-grade fever, lymphadenopathy, and faint macular rash.
- Very serious in pregnant women
  - Can cause congenital abnormalities in fetus.
- Usually seen in the spring
- Infected individual contagious for 7 days before and 7 days after appearance of rash.
RUBELLA: Pathogenesis

- Virus enters through the respiratory tract.
- Spreads to the blood, lymph organs, and skin
- Viremia is seen up to 8 days before the rash.
- Viral shedding in oropharynx up to 8 days after.
- Transplacental transfer of virions can occur in pregnancy.

RUBELLA: Treatment

- No specific therapy.
- Live attenuated vaccine (part of MMR) is recommended in the first year of life.

SMALLPOX (VARIOLA)

- Infection caused by a DNA poxvirus.
- Two forms of smallpox:
  - Variola major – mortality rate 20% or higher
  - Variola minor – mortality rate 1%
SMALLPOX (VARIOLA)

- Smallpox has effectively been eradicated from the entire world.
  - Last victim in Somalia in 1977
- Only reservoir is humans.
  - Should be no more cases
- Stocks of smallpox virus mean further infections are possible
  - Decreased herd immunity to smallpox increases the possibility

SMALLPOX: Pathogenesis

- Dominant feature is the appearance of papulovesicular rash and pustules.
- Incubation period is usually 12-14 days.
  - Can be 4-5 days
  - Abrupt onset of fever, chills, and muscle aches
- Rash appears 3-4 days later.
  - Papulovesicles most prominent on the head and extremities.
  - Become pustular over 10-12 days.
- Death from smallpox results from:
  - Overwhelming virus infection
  - Bacterial superinfection
**SMALLPOX:**

**Treatment**

- Virus has potential as a bioweapon.
  - Many countries stockpiling vaccine
- No infected population
  - Difficult to test potential antibiotics

**CHICKENPOX AND SHINGLES**

- Caused by the varicella-zoster virus.
- 90% of US population infected by age 10.
  - Mortality rate very low
- Infection has two clinical manifestations:
  - Chickenpox
  - Shingles

**CHICKENPOX AND SHINGLES:**

**Pathogenesis**

- Virus is spread through secretions of the respiratory tract.
- Infection occurs in upper respiratory tract, lymph nodes.
- Causes vesicular rash
  - Usually initially on back of the head and ears
  - Then on the face, trunk, and proximal extremities
- Commonly involvement of mucous membranes and fever early in infection.
- Irritating, itchy lesions can appear.
- Secondary viremia includes infection of the skin.
CHICKENPOX AND SHINGLES: Pathogenesis

- Latent form of the virus resides in the dorsal root ganglia.
- After latent virus is reactivated:
  - Multiplies in a sensory neuron
  - Travels down to the skin
- Shingles rash comprises vesicles similar to those seen in chickenpox.

- Shingles vesicles are localized in distinct areas of the body.
  - Usually around the waist
  - Can be on the upper chest and back
- Reactivation increases in frequency with advancing age.
- Lesions are very painful.
  - Appear several days to several weeks after pain
  - Can persist for months
- Can be multiple organ involvement in immunocompromised patients
  - Significant mortality rate
CHICKENPOX AND SHINGLES: Treatment

- Acyclovir and famciclovir reduce fever and skin lesions.

HERPES SIMPLEX TYPE I (HSV-1)

- HSV-1 causes above-the-waist infections.
  - HSV-2 causes below-the-waist infections.
- Latent infection
  - Signs appear when virus is reactivated.
- Virus found worldwide.
  - 90% of population in developing countries have antibodies.
- Humans are the only reservoir.

HSV-1: Pathogenesis

- After initial infection:
  - Syncytia develop
  - Degeneration of epithelial cells
  - Necrosis at the infection focus
  - Inflammatory response
  - Infiltration by neutrophils then mononuclear cells
HSV-1:
Pathogenesis

- Virus spreads either interneuronally or intraneuronally.
- Intraneural spread
  - Virus can hide from the immune response and lie latent for years.
- Latent virus resides on the trigeminal, superior cervical, and vagus nerve ganglia.
  - Reactivation stimuli are not yet understood.
- HSV-1 infection is often asymptomatic.

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HSV-1:
Pathogenesis

- Principal sign is grouped or single vesicular lesions that become pustular
  - Coalesce to form multiple ulcers
- Can be painful ulcerative lesions on the tongue, gums, and pharynx
  - Usually persist for 5-12 days
- Latent reactivation causes painful lesions on or near lips – cold sores
  - Can last for as long as 7 days

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HSV-1:
Pathogenesis

- [Image of herpes lesions]

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**HSV-1: Pathogenesis**

- Sometimes infects the fingers, around the nails
  - Usually because of a break in the skin
  - Causes formation of painful pustular lesions
- Virus can also infect the eye.
  - Most common cause of corneal damage and blindness in developed world.
  - Dendritic ulcerations in the conjunctiva and cornea cause scarring.

**HSV-1: Treatment**

- Most effective treatment is the nucleoside analog acyclovir.
  - Reduces primary infection and can suppress recurrence.
- Immunocompromised may harbor resistant HSV.
  - Foscarnet can be used.

**WARTS**

- Small growths on the skin or mucous membranes.
  - Respiratory tract, genital tract, and interior of the mouth
- Caused by human papillomavirus (HPV)
- HPV infection is life-long:
  - Warts can return even after removal
  - Virus is still associated with the tissue
WARTS

- Warts vary in appearance, location, and pathogenicity
  - Some are small and self-limiting
  - Others are large but benign
  - Others are malignant
- Some strains of HPV cause cervical cancer.
- Warts are larger and occur more frequently in the immunodeficient.

WARTS: Pathogenesis

- Transmitted by direct contact between humans and by fomites.
- Genital warts can be sexually transmitted.
  - Incubate for 8-20 months before visible signs develop

WARTS: Pathogenesis

© CDC/Susan Lindsley
WARTS:
Pathogenesis

- Dermal warts form after virus gains entry through broken skin.
  - Incubates for 1-4 weeks
  - Virus infects epithelial cells
    - Proliferation of these cells forms the warts
  - Occurs at the boundary between the dermis and the epidermis
  - Usually only one at most few during an outbreak.

TREATMENT OF WARTS

- Can be spontaneous regression
- No satisfactory treatment of warts
  - Growth can be removed using liquid-nitrogen cryotherapy.
  - Antimetabolites can block HPV infection.
- Fungi are always present on the skin
  - Rarely bother us
- Compromise of an individual’s health causes opportunistic infections
- As with bacteria and viruses, unbroken skin is a barrier.

CUTANEOUS CANDIDIASIS

- Caused by *Candida albicans*
  - Part of the normal flora, oropharyngeal, gastrointestinal, and genitourinary tracts
- Can grow in multiple morphological forms
  - Most often seen as a yeast
  - Capacity to form hyphae
    - Strongly associated with pathogenicity
CANDIDIASIS: Pathogenesis

- Hyphae invade deep into tissues.
  - Form strong attachments to human epithelial cells
  - Secrete proteinases and phospholipases
  - Digest epithelial cells and facilitate tissue invasion
- C. albicans surface proteins:
  - Bind C3 receptors.
  - Prevent opsonization.

Compromise of T-cell function or overuse of antibiotics allows C. albicans to increase in numbers
- Results in local and invasive infection
- Indwelling catheters and chemotherapy also advance invasion

Infections usually occur in folds of skin.
- Two wet skin surfaces are opposed to each other
- Cause diaper rash
- Initial lesions are erythematous papules.
- Thrush can develop in infants and patients with immunodeficiencies.
CANDIDIASIS: Pathogenesis

- Infection usually confined to chronically irritated areas.
- Mucocutaneous candidiasis can be seen in immunocompromised patients.
  - Infections of the hair and skin fail to heal and require therapy.
  - Results in considerable discomfort
  - Can cause disfigurement with extensive areas of lesions.

CANDIDIASIS: Treatment

- *Candida albicans* is usually susceptible to nystatin, fluconazole, and azole antibiotics.
  - Deep tissue infections also require amphotericin B.
- Measures should be taken to decrease moisture.
- Fluconazole is best treatment for mucocutaneous candidiasis.
DERMATOPHYTOSIS

- Dermatophytes are fungi that are pathogenic to the skin.
  - Cause cutaneous mycoses
  - Result in slow, progressive eruptions of the skin
    - Unsightly but not painful or life-threatening.
- Different forms are classified according to the inflammatory response.
- All forms typically involve erythema, induration, itching, and scaling.

DERMATOPHYTOSIS

- Dermatophytosis on the scalp – tinea capitis
- Dermatophytosis in the groin – tinea cruris (jock itch)
- Dermatophytosis on the feet – tinea pedis (athlete’s foot)
DERMATOPHYTOSIS

- Ecological and geographic differences in different dermatophyte infections.
- Many domestic cats and dogs act as reservoirs.
- Human-to-human transfer requires close contact.
  - Dermatophytes poorly infective
  - Very low virulence
- Infection usually seen in families, barber shops, and locker rooms.

DERMATOPHYTOSIS: Pathogenesis

- All three forms of tinea begin when dermatophyte hyphae contact minor traumatic skin lesions.
- Stratum corneum is penetrated by hyphae.
  - Dermatophytes proliferate in the keratinized layers of the skin.
  - Aided by production of protease enzymes.
- Course of the infection depends on anatomical location, moisture, and the rate skin cells are shed.
  - Speed and strength of inflammatory response has a role.
  - Faster skin is shed, less time it takes to get over the infection
  - Inflammation can increase shedding rate.
  - Immunosuppression increases the length of the infectious period.
DERMATOPHYTOSIS: Pathogenesis

- Invasion of deep tissues is rare.
- Most infections are self-limiting.
  - Can become chronic if the inflammatory response is poor
- Infections can also affect the nails and hair follicles.
  - Plugs follicles, causing hair to become brittle.

DERMATOPHYTOSIS: Treatment

- Most resolve without therapy.
- If not, topical agents like tolnaftate, allylamines, and azoles are used.
- Extensive infections in the nail beds require systemic therapy with griseofulvin or itraconazole for weeks or months.
- Tinea capitis also requires systemic therapy.

PARASITIC INFECTIONS OF THE SKIN

- There are several common parasitic infections of the skin.
- As with other infections, the skin must be broken.
CUTANEOUS LEISHMANIASIS

- Zoonotic parasitic infection seen tropical and subtropical rodents.
  - Common in central Asia, India, Middle East, South and Central America
- Humans contract it when they enter areas where the rodents live.
- Infection is vector-transmitted from rodent to human.
  - Sand flea is the vector.
- Can also be transmitted through domestic dogs
  - Dog fleas are the vector.

CUTANEOUS LEISHMANIASIS: Pathogenesis

- Lesions appear on the extremities or the face weeks to months after the bite.
  - Appear as itchy pustules
  - Accompanied by lymphadenopathy
- Pustules ulcerate after a few months.
  - Can be several of these lesions on the body
**CUTANEOUS LEISHMANIASIS: Pathogenesis**

- Lesions heal spontaneously in 5-12 months.
  - Leaves depigmented scars
- Lesions on ear can cause destruction of the pinna.
- Patients with AIDS have multiple nonhealing lesions.

**CUTANEOUS LEISHMANIASIS: Treatment**

- If no involvement of mucous membranes then no treatment required.
- Amphotericin B, ketoconazole, itraconazole are used for more severe cases.

**PEDICULOSIS**

- Pediculosis – parasitic infection by lice
  - Can occur on the head or body
  - Often in the genital region – crabs
- Body lice can transmit epidemic typhus.
- Outbreaks of head lice are frequently seen in schools.
PEDICULOSIS: Pathogenesis

- Lice require blood.
- Feed several times a day.
- Itching is a reaction against the saliva of the biting louse.
- Scratching can result in secondary bacterial infection.
- Head louse has specialized legs to grasp hair shaft, usually at the base.

PEDICULOSIS: Pathogenesis

PEDICULOSIS: Treatment

- Usually involves combing out the lice and nits in combination with non-prescription drugs.
- Increasing resistance to these drugs
- Topical ointments such as lindane and malathion are effective but toxic.
INFECTION OF THE EYES

- Affected by infections in the same manner as the skin.
- Eyes are a privileged site.
  - Essentially protected from the immune system.
- Mechanical barriers such as tears protect the eye from infecting organisms.

INFECTION OF THE EYES

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<th>Causing Organism</th>
<th>Infection Characteristics</th>
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Table 3.1: Infections of the Eyes © Garland Science

CONJUNCTIVITIS AND OTHER EYE INFECTIONS

- Eye infections are usually painful.
- There is also potential for vision loss.
- Conjunctivitis is the most common infection.
  - Occurs in all age groups
  - Easily spread if a contaminated hand rubs the eyes
CONJUNCTIVITIS AND OTHER EYE INFECTIONS

- Parts of the eye other than the conjunctiva also become infected:
  - Cornea infection – keratitis
  - Anterior and posterior chambers
  - Orbital sinuses can also be involved
    - Can be life-threatening
    - Close proximity to the central nervous system

CONJUNCTIVITIS AND OTHER EYE INFECTIONS

- Contact lenses washed in contaminated water
  - Increase in numbers of cases of lens keratitis
- Aggressive keratitis caused by *Pseudomonas aeruginosa* occurs in hospitals.
  - Painful
  - Loss of vision can occur
- Eyelid abscesses (styes) are fairly common problems.
- Infection of the entire eyelid is also possible.
- Lacrimal gland and duct can be infected.

TRACHOMA

- Leading eye infection in underdeveloped countries.
  - An estimated 500 million people are affected.
  - Approximately 10 million have been blinded by it.
- Caused by *Chlamydia trachomatis*
- Spread to the eyes by hands, fomites, and flies.
- Essentially a chronic conjunctivitis.
  - Causes scarring, corneal ulceration, and eventual vision loss
RIVER BLINDNESS

- Caused by the parasite *Onchocerca volvulus*.
- Affects 20 million people worldwide.
- Spread by blood-sucking blackfly
  - Transmits parasite to the eye
- Parasite invades the anterior chamber
  - Causes corneal ulceration, fibrosis, and blindness

EYE INFECTIONS: Treatment

- Topical eye drops and ointments containing erythromycin or gentamicin are effective against acute bacterial conjunctivitis.
- Fluoroquinolones can be used for eye infections caused by *Pseudomonas*.
- Quinolones such as ciprofloxacin useful for all types of eye infection.

NEONATAL EYE INFECTIONS

- Neonatal gonorrheal ophthalmia:
  - Serious conjunctivitis caused by *Neisseria gonorrhoeae*.
  - Contracted as infant passing down the birth canal.
- *Chlamydia trachomatis* can also infect the eyes of newborns.
- Both infections cause large amounts of pus to form in the eyes.
  - Causes ulceration and scarring of the cornea if not treated
- Common practice to treat eyes of newborn infants with erythromycin.
NEONATAL EYE INFECTIONS

LOAIASIS

Eye infection caused by the parasitic worm *Loa loa*.
- Found in African rain forests
- Transmitted to humans by the deer fly
- Acquires the parasite from infected humans

Loa loa matures in its vector – the deer fly.
- Worm migrates to the mouth of the fly.
  - Transmitted to a human when the fly bites
- Microfilariae migrate through the subcutaneous tissue.
  - Cause inflammation
  - Settle in the cornea and conjunctiva
- Worms can grow to more than an inch in length.