

Disclosures

Advisory Board

- Bristol Myers Squibb -Psoriasis
- Incite -Vitiligo
- JDNPPA -Dermatology
- UCB -Psoriasis

Speaker Panel

- American Association of Nurse Practitioners Dermatology
- Dermatology Nurses' Association -Dermatology
- MauiDerm NP+PA -Dermatology
- Nurse Practitioner Association for Continuing Education -Dermatology

Objectives

Upon completion of this presentation the participant will

- Understand the anatomy and physiology of the skin
- Relate general concepts of cutaneous Basic Science
- Construct a differential diagnoses and management approach to Infections, Infestations and Bites

- >700 million outpatient skin-related visits
- 52.9% of all skin-related visits were addressed by non-dermatologists:
 - family practitioners (20.5%)
 - pediatricians (11.3%)
 - internists (9.2%)
 - general surgeons (3.4%)
 - otolaryngologists (1.0%)
 - emergency medicine physicians (0.2%)



Wilmer EN, Gustafson CJ, Ahn CS, Davis SA, Feldman SR, Huang WW. Most common dermatologic conditions encountered by dermatologists and nondermatologists. Cutis. 2014 Dec;94(6):285-92. PMID: 25566569.

- contact dermatitis
- infectious skin diseases:
 bacterial (cellulitis/abscess),
 viral (warts, herpesvirus),
 fungal (tinea), yeast (candida)
- 3. acne, epidermoid cysts
- benign tumors, actinic keratoses, non-melanoma skin cancers
- ~10% referred to dermatology for management



Wilmer EN, Gustafson CJ, Ahn CS, Davis SA, Feldman SR, Huang WW. Most common dermatologic conditions encountered by dermatologists and nondermatologists. Cutis. 2014 Dec;94(6):285-92. PMID: 25566569.

- atopic dermatitis, acne vulgaris, cellulitis/abscess, verruca vulgaris, and benign skin lesions
- diagnosis and management can be managed by PCPs
- diagnosis, management, referral of serious skin diseases by nondermatologists can be life saving



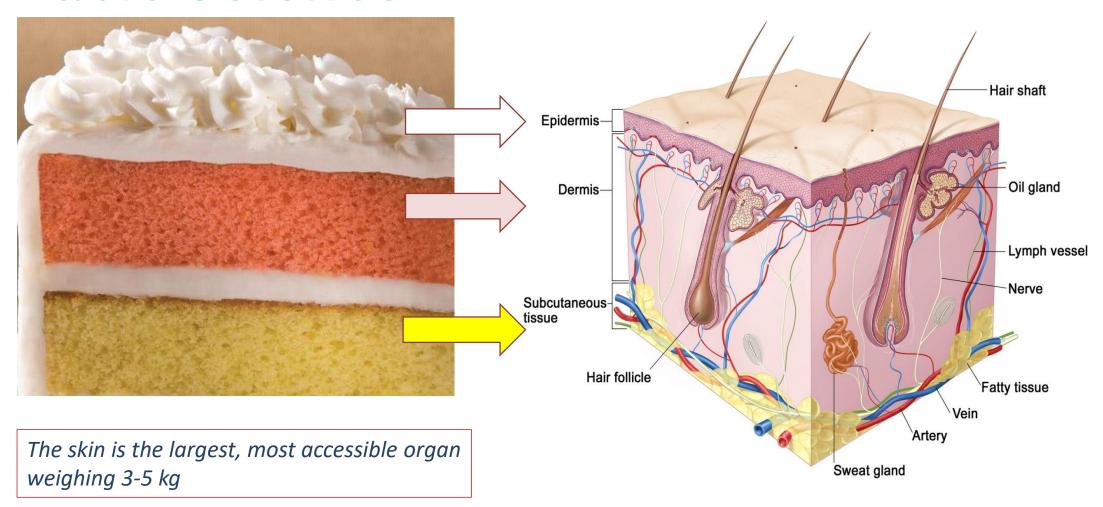
Etcheverria, Eden MPAS, PA-C. Recognizing and treating five common dermatologic conditions seen in primary care. Journal of the American Academy of Physician Assistants 33(11):p 33-37, November 2020. | DOI: 10.1097/01.JAA.0000718288.06130.dd

Grada A, Muddasani S, Fleischer AB Jr, Feldman SR, Peck GM. Trends in Office Visits for the Five Most Common Skin Diseases in the United States. J Clin Aesthet Dermatol. 2022 May;15(5):E82-E86. PMID: 35642232; PMCID: PMC9122273.

leading dermatologic diagnoses across all specialties

	African Amer	Hispanic	Asian/Pacific Is	Caucasian
	Eczema/Dermatitis	Eczema/Dermatitis	Eczema/Dermatitis	Acne
	Acne	Acne	Acne	Eczema/Dermatitis
	Fungal Infection	Cyst	Atopic Derm	Actinic Keratoses
	Cyst	Warts	Urticaria	Warts
	Cellulitis/Abscess	Cellulitis/Abscess	Psoriasis	Cellulitis/Abscess
Referral to Derm	28.5%	23.9%	36.7%	43.2%

Davis SA, Narahari S, Feldman SR, Huang W, Pichardo-Geisinger RO, McMichael AJ. Top dermatologic conditions in patients of color: an analysis of nationally representative data. J Drugs Dermatol. 2012 Apr;11(4):466-73. PMID: 22453583.



Epidermis

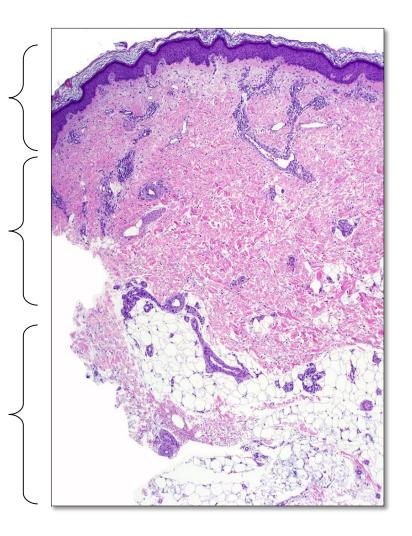
keratinocytes hold moisture & provide protective outer layer

Dermis

fibroblasts, collagen, elastic fibers & ground substance provide tensile strength also blood vessels, nerves, adnexal structures (hair, oil glands, lymph vessels)

Subcutis

or panniculus provides fat for energy and insulation



5. Stratum corneum _____

desquamating keratinocytes, filaggrin & lipids provide hydrophobic barrier against trauma and infection

3. Stratum granulosum -

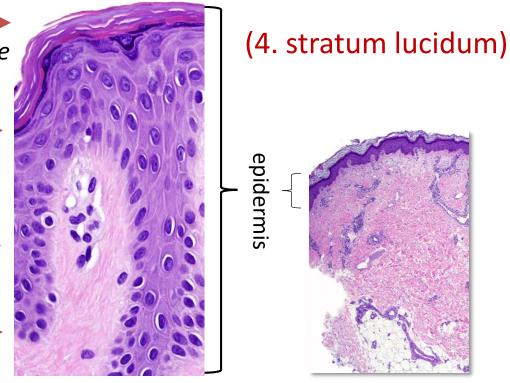
lipids secreted into extracellular space btw KC forms a water barrier that keeps water in the skin

2. Stratum spinosum

spiny desmosomal junctions hold the keratinocytes together providing epidermal integrity

1. Stratum basale

where the keratinocytes develop



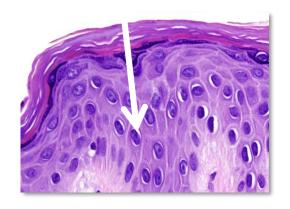
epidermal stem cells "keratinocytes" migrate from basal cell layer up to and through the stratum corneum over 28 days

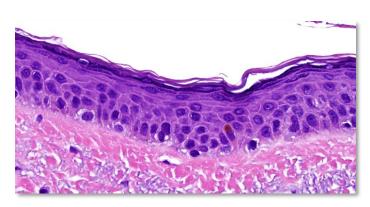
3 main cell types make up the epidermis

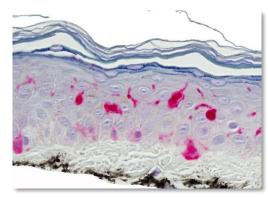
Keratinocytes

Melanocytes









Keratinocytes are held together by desmosomes creating "brick & mortar" epidermal structure

breakdown of this structure: atopic dermatitis



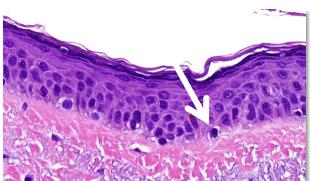
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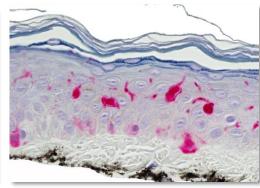
Keratinocytes

Melanocytes

Langerhans cells







Melanocytes

pigment-producing cells transfer their pigment "melanin" to keratinocytes as melanosomes clusters of melanocytes = moles mass of malignant melanocytes = melanoma



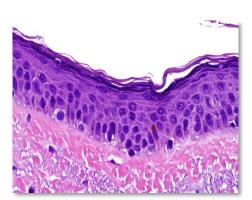
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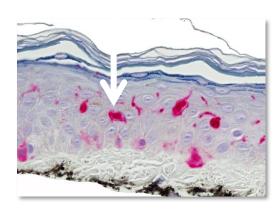
Keratinocytes

Melanocytes

Langerhans cells







Langerhans cells are antigen presenting cells important in the induction of delayed-type hypersensitivity



Allergic Contact Dermatitis

Dermoepidermal junction

- allows the epidermis and dermis to adhere to one another
- inflammatory conditions:
 Lichen Planus
- autoantibodies:Bullous Pemphigoid



Epidermolysis Bullosa

Dermis: flexible, tough 1-4 mm support structure contains

Collagen fibers

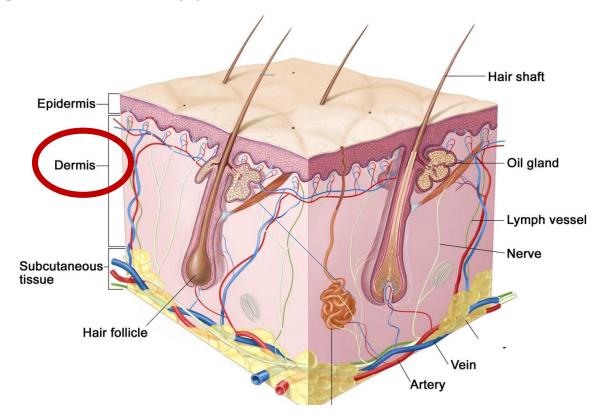
tensile strength

Elastic fibers

resiliency

Ground substance

diffusion



hair follicles sweat glands blood vessels nerves lymphatic vessels

Dermal Cells:

Fibroblasts, Histiocytes & Mast Cells

Fibroblasts

- instrumental in wound healing & scar formation
- Dysfunction from uncontrolled synthesis & excessive deposition of collagen at sites of prior dermal injury: keloids

Langerhans, neutrophils, lymphocytes, monocytes, mast cells

- immediate-type hypersensitivity immune reactions
- Dysfunction of mast cells can result in urticaria
 -a hypersensitive vascular reaction of the skin





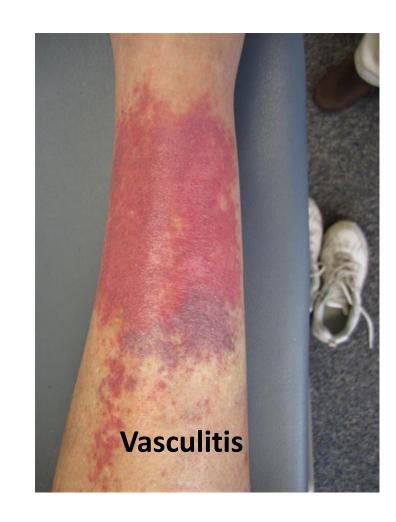
Dermis:

Blood Vessels

- **≻**temperature
- **≻**oxygen
- **>** nutrients

Nerves

- **≻**touch
- **>** pain
- **≻**itch





Dermis: hair follicles

- 100,000 scalp hairs preserve heat, tactile sensation
- each hair grows for 3 yrs, rests for 3 mos, then alternates between growth and rest for 3 wks
- hair growth is generally random, telogen effluvium happens if those growth cycles become synchronized -postpartum women
- 50% have androgenic hair loss due to conversion of testosterone to DHT resulting in miniaturization of the hair follicles



Female Pattern Hair Loss

Dermis:

Sebaceous (oil) glands

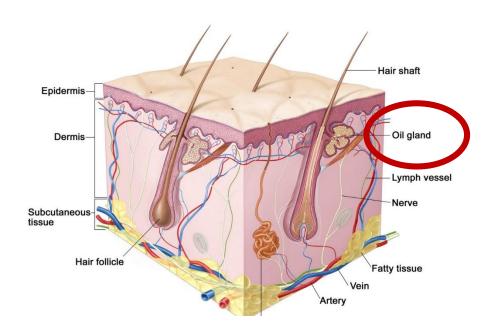
- lubricate the hair follicle
- can also be involved in diseases like acne

Eccrine glands

- produce sweat to help with electrolyte balance and thermoregulation as evaporation of sweat from the skin cools the body
- Acetylcholine triggers the release of sweat from the eccrine glands which can be blocked with botulinum toxin type A (Botox®)in the setting of excess sweating "hyperhidrosis"

Apocrine glands

- located in the axillary and anogenital areas
- secrete a very thick type of sweat which causes body odor when combined with bacteria

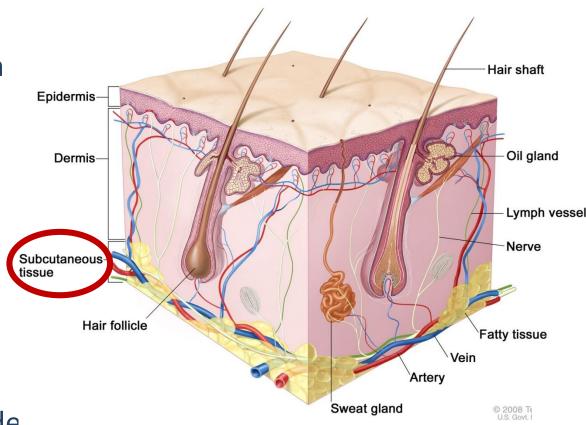


Subcutis

fat layer separates dermis from deeper fascia & muscles

- insulates
- supplies energy
- cushions & protects
- allows for skin mobility over underlying structures

Disorders in the subcutis include lipoatrophy and panniculitis



Ethnic Dermatology

Genetic variations lead to disease susceptibility among different racial groups

 Atopic Dermatitis, dermatomyositis, hidradenitis suppurativa, keloids, lupus, psoriasis > pts with SOC

Vitamin D

 calcium homeostasis, bone metabolism, skin cellular proliferation, barrier maintenance, immune functions

Ceramide level

- major lipid constituent enhances skin barrier function
- deficiencies: increase risk of psoriasis, atopic derm



Taylor SC-Kyei A. Defining skin of color. In: Taylor SC, Kelly AP, eds. Dermatology for skin of color 2nd ed. New York, NY: McGraw-Hill; 2016:9-15.

Sangha AM. Dermatological Conditions in Skin of Color—Physiological Nuances in Skin of Color and Their Clinical Implications. *J Clin Aesthet Dermatol.* 2023;16(2 Suppl 1):S12–S13.

Cutaneous Infections Bacterial Infections

Bacterial Folliculitis

- S. Aureus- most common
- Gram negative long term abx

Topical

- chlorhexidine wash
- mupirocin, clindamycin

Systemic

- guided by culture
- consider MRSA



Pseudomonas Folliculitis

- exposure to contaminated pool, whirlpool, hot tub, water slide, loofah
- very itchy hives with central pustules
- often under bathing suit area
- hours to days after exposure
- infection is self-limited
- 5% acetic acid (white vinegar) wet compresses
- topical: clindamycin
- resistant: oral ciprofloxacin



Impetigo

- highly infectious superficial epidermal infection
- S. aureus, strep pyogenes, combination
- face and extremities

Nonbullous Impetigo (70%)

 small 2-4 mm red macules that evolve into superficial vesicles, pustules, erosions with moist, honey-colored crusts



Bullous impetigo

Bullous impetigo (30%)

- vesicles \rightarrow large flaccid bullae, children, elders
- poor hygiene, warm, moist climates
- self inoculation from nasal or perineal carriage
 Risk
- post-strep glomerulonephritis; rheumatic heart

Ecthyma

- deeper impetigo causing erosion/ulceration into dermis
 Rx
- localized: mupirocin or retapamulin BID x 5 days
- generalized: dicloxacillin, cephalexin
- MRSA: doxy, clinda, sulfamethoxazole/trimethoprim



Erysipelas

- Infection of superficial dermal lymphatics
- β-hemolytic streptococci
- fiery red, hot, swollen plaque
- face, legs, ears, buttocks
- onset acute, spreads rapidly



Cellulitis

- pink to red, warm, swollen, tender
- infection of dermis and sq tissue
- *S. aureus* (purulent)
- streptococci (non-purulent)

Risk Factors

 barrier disruption, tinea pedis, inflammatory disorders, edema, immunosuppression

Erysipelas / Cellulitis Treatment

- rest, elevation
- penicillin VK
- cephalexin 250-500mg qid
- dicloxacillin 500mg qid
- clindamycin 300-450mg tid
- duration x 5 days (minimum)

Cellulitis

Chronic Recurrences

- 14% in one year; 45% in three years
- Treatment: parallels acute episode
- treat underlying tinea, lower leg edema

Prophylactic antibiotics reduce recurrences

- penicillin V 250-500 mg orally BID
- benzathine penicillin G 1.2-2.4MU IM Q4wk
- cefadroxil 500 mg orally BID if MSSA
- cephalexin 500 mg orally QID if MSSA
- TMP-SMX; DS tablet orally BID if MRSA



N Engl J Med. 2013;368(18):1695-1703 Cochrane Database Syst Rev 2017; 6:CD009758.

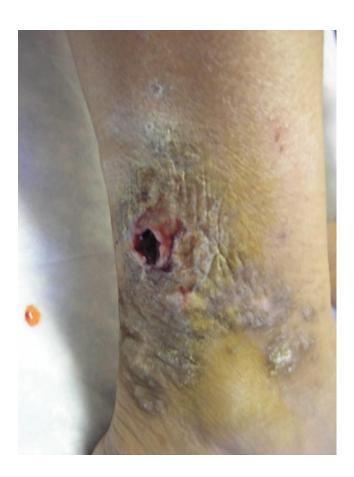
Furuncle

Furuncle

- deep, tender abscess or boil, single draining point
- favoring hair bearing areas; S. aureus common Carbuncle
- 2+ confluent furuncles; multiple draining points

Management

- | & D
- culture
- oral abx only if systemic s/s (fever, chills, leukocytosis)
- cover for MRSA if failed abx, immunocompromised
- trimethoprim-sulfamethoxazole DS, doxycycline, minocycline, clindamycin
- if recurrent in same location, consider Hidradenitis Suppurrativa



Staphylococcal Scalded Skin Syndrome

- desquamation of skin from Staph
- often follows localized infection (URI, ears, conjunctiva, perineum)
- <5 yr immunocompromise, renal failure
- erythematous rash with diffuse large flaccid, vesicles and bullae filled with cloudy/yellow exudate
- oral mucosa usually NOT involved
- Ddx: SJS/TEN
- Rx: systemic abx, IVF, supportive care



Cutaneous Infections Fungal Infections

Pityrosporum Folliculitis

- Pityrosporum (Malassezia)
- •itchy monomorphic follicular papules and pustules
- upper trunk, forehead
- sweating or humidity
- •oral antibiotics, corticosteroids
- •Rx: topical: azoles, selenium sulfide, ciclopirox, sulfacetamide sulfur
- oral: fluconazole, itraconazole*



Itraconazole carries a Black Box Warning for Congestive Heart Failure, Cardiac Effects and Drug Interactions

Tinea Versicolor

- Malassezia globosa
 - hyper-/hypo-pigmented
 - pink, copper, pale brown/tan, atrophic
- recurrent, scaly, itchy rash; trunk, neck, UEs
- colonization of scalp
- Rx: topical antifungals –azoles
- shampoos: ketoconazole, selenium sulfide, ciclopirox
- wash: sulfacetamide sulfur
- 2nd line: oral fluconazole, itraconazole



Dermatophytes

- T. rubrum infection of skin, hair, nails
- human, animal, soil or autoinoculation
- Itching, burning, pink, annular plaque with central clearing as infection spreads outward
- advancing scaly border
- KOH testing "ziti & meatballs"
- topical antifungals
 - imidazoles (econazole, ketoconazole)
 - allylamines (naftifine, terbinafine)
 - benzylamine (lotrimin)

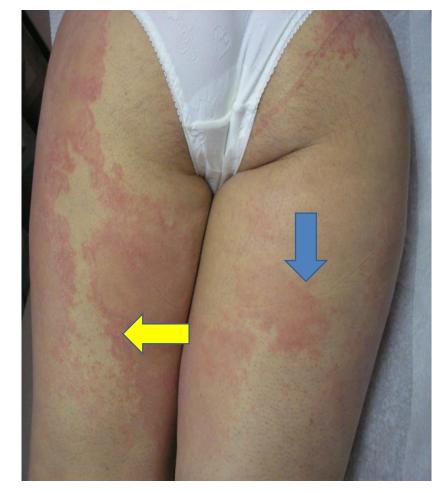




Dermatophytes



Disseminated Tinea Corporis



TMVII: new-cause-sexually-transmitted-fungal-infection



https://www.mdedge.com/edermatologynews/article/271563/infectious-diseases/new-cause-sexually-transmitted-fungal-infection-reported-msm

Tinea Capitis

- T. Tonsurans, +/- scale, lymphadenopathy
- alopecia
- "black dots" from broken hairs

Deep Fungal Infection

- if pustules and recent topical corticosteroid use at site of skin infection, think Majocchi granuloma (deep follicular tinea)
- Rx: systemic antifungals
 - griseofulvin, terbinafine
 - -azoles (ketoconazole, itraconazole, fluconazole)





Tinea Cruris

- +/- mixed candida & dermatophytes
- sharply demarcated erythematous or tan eruption with raised scaly borders
- vesicles with severe inflammation
- usually extremely pruritic; scrotum spared,
 unlike candidiasis where scrotum involved
- epidemics (sports, prisons), foot to groin
- topical treatment: imidazole agents



Tinea Pedis

(Athlete's Foot)

- T. rubrum, T. mentagrophytes, E. floccosum
- can be asymptomatic
- "2 feet, 1 hand"

Types

- moccasin
- vesicular/bullous
- interdigital
 - 3rd and 4th toe web spaces most common
 - maceration
 - risk for secondary bacterial infection





Tinea Unguium

(Onychomycosis)

- distal thickening, yellowing of toe/finger nail plate
- concerning in diabetics, immunosuppressed pts
- topical therapy
 - ciclopirox 8% sol/laquer
 - efinaconazole 10% or tavaborole 5% solution
- oral therapy* (hx, monitoring, AEs)
 - terbinafine: 6 wks fingernails, 12 wks toenails
 - fluconazole: 3-6 mo fingernails, 6-12 mo toenails



Intertriginous Candidiasis

(Intertrigo)

- red, moist, well demarcated, confluent plaques with satellite pustules and papules at skin folds
- no improvement with barrier creams
- KOH preparation or fungal culture
 Treat with –azole antifungals;
 miconazole, ketoconazole, fluconazole
 nystatin (cream/powder), zinc oxide paste

Candida Balanitis is most common in older, uncircumcised men presents as redness with white exudate edema of the glans penis recurrent infection can lead to phimosis or ureteral meatus stenosis



Cutaneous Infections Viral Infections

Herpes Simplex Virus

- ➤ HSV-1: peri-oral, HSV-2: genital
- >cluster of vesicles, pustules or erosions on erythematous base with scalloped borders
- > crust over and resolve in 2-6 wks
- >virus lies dormant in nerve roots
- ➤ ultraviolet radiation, laser, stress, trauma, immunosuppression reactivates intermittent recurrence in the same location, i.e., "cold sores"
- ➤ Rx for HSV-1: acyclovir ointment



Primary Varicella Virus

(chickenpox)

respiratory droplets or direct contact prodrome of fever, chills, malaise, aches very itchy "dew drop on a rose petal" at scalp, face, then trunk & extremities highly contagious (until all are crusted over) course usually self-limited and benign -unless pregnancy or immunocompromised



Herpes zoster virus

- reactivation of latent varicella zoster virus (VZV)
 infection. Reactivation: immunosuppression, certain
 medications, other infections, physical/emotional stress
- 1- to 3-day prodrome of burning pain or paresthesias, erythematous papules and vesicles in the affected dermatome
- Labs: Polymerase chain reaction (PCR)-based testing detects VZV DNA with high sensitivity within 24 hours.
 Skin lesions at all stages may yield a positive result (including vesicles, ulcers, crusts)
- Tzanck smear, direct fluorescent antibody (DFA) testing, and viral culture have a lower degree of sensitivity



HVZ Management

- Aluminum acetate or Burow's soaks can help to alleviate cutaneous symptoms. Patients routinely require oral analgesia. Consider NSAIDs and opioids, including controlled-release oxycodone in patients not rapidly responding to the initial regimen.
- Antiviral therapy (acyclovir and pro-drug forms), if administered in the first 72 hours (and possibly up to 7 days) after symptom onset, can shorten the length and severity of the acute episode and may help to decrease the likelihood of developing postherpetic neuralgia. Take into account the risk factors for developing postherpetic neuralgia. A single published study supports the use of amitriptyline (25 mg daily) as an adjunct to an antiviral agent in acute herpes zoster, to decrease pain associated with subsequent postherpetic neuralgia. Another study showed that gabapentin with valacyclovir prevented postherpetic neuralgia more effectively than valacyclovir alone. Observe for and promptly treat any secondary bacterial infection.
- Antivirals: Acyclovir 800 mg by mouth every 4 hours for 7-10 days (frequently given 5 times daily)
- Famciclovir 500 mg by mouth every 8 hours for 7 days
- Valacyclovir 1000 mg by mouth every 8 hours for 7 days. Note: valacyclovir should not be used in immunosuppressed patients.
- Foscarnet 40 mg/kg intravenously (IV) every 8 hours for 10 days for cases resistant to acyclovir. Given as a 1-hour infusion.
- Skin lesions may continue to form for up to 1 week after the initiation of antiviral therapy. Some experts will recommend continuing antiviral therapy in immunocompromised patients until all lesions have crusted. While rare, acyclovir-resistant VZV has been reported in the immunocompromised population, especially in those with HIV. Any atypical lesions or failed response from use of acyclovir should be evaluated for drug resistance. If this occurs, alternative therapies such as IV foscarnet or cidofovir may be considered.
- Patients with active vesicular lesions can spread the infection. The following consultations may be needed: dermatology, neurology, infectious disease, and/or ophthalmology (herpes zoster ophthalmicus).

HVZ Management (2)

- Zoster may be accompanied by acute pain. Postherpetic neuralgia, pain and neuropathic symptoms that persist in a dermatome >1 mo after resolution of the rash. Risk factors include older age, female sex, presence of a prodrome, greater rash severity, and acute pain. Post-herpetic neuralgia can be intractable and debilitating, and prevention is an important goal.
- associated: CVA, peripheral motor neuropathies, neurogenic bladder, diaphragmatic paralysis, postherpetic abdominal asymmetry, herpes zoster encephalitis (10%-20% mortality rate), 2ndary bacterial superinfection including necrotizing fasciitis
- Immunocompromised pts: higher risk of disseminated zoster, persistent ulcers, chronic hyperkeratotic zoster
- Involvement of a thoracic dermatome may simulate acute myocardial infarction. Involvement of the ophthalmic division of the trigeminal nerve (to include the tip of the nose, "Hutchinson sign") should trigger emergent referral to an ophthalmologist. Disseminated zoster, >20 vesicles outside of the primary and adjacent dermatomes, is seen chiefly in immunocompromised pts
- Prophylactic vaccination: The CDC Advisory Committee on Immunization Practices recommends use of Shingrix that is more effective than the older Zostavax vaccine. Shingrix is approved for adults ≥50 years. The panel also recommends that patients previously vaccinated with the Zostavax vaccine be revaccinated with Shingrix. The vaccination decreases risk for both herpes zoster and postherpetic neuralgia. An ongoing study has shown high levels of immunity for at least 7 years post-vaccination.
- Prophylaxis in immunocompromised patients: Recommend acyclovir prophylaxis early in the period immediately following transplant when patients are the most immunosuppressed and are at highest risk for complications of zoster. Although there are no established criteria, transplant specialists recommend 3-6 months of acyclovir as prophylaxis in this population. Among individuals with AIDS, however, antiviral prophylaxis with acyclovir is not recommended.

Arthropod Infestations

Cutaneous Larva Migrans

- parasitic infestation of the epidermis caused by larvae of hookworms that infect domestic dogs and cats or humans
- acquired by walking barefoot on soil or sand contaminated with dog or cat feces containing the larvae.
- larvae penetrate the epidermis and undergo a prolonged migration through the epidermis in a snake-like fashion.
- migration begins ~4 days after entry, progress 1-2 cm/day
- pruritic, edematous, erythematous, vesicles and bullae
- albendazole 400 mg po daily for 3-7 days, or ivermectin 200 μ g/kg by po daily for 1-2 days (both contraindicated in pregnancy)



Pediculosis (Lice) (capitis, corporis, pubis)

- Multiple erythematous papules concentrated in areas covered by clothing: the groin, axillae, trunk, buttocks
- closely examine the pt's clothing seams for lice, nits, feces
- Wood's lamp may be helpful, as lice and nits will fluoresce yellow-green.
- Wash all clothing and linens with hot water and dry using high heat.
- Discard or avoid using heavily infested items for 2 weeks (seal in plastic bags), if feasible. Iron furniture seams with a hot iron.
- 5% permethrin cream or lotion —or- 0.5% malathion lotion head-to-toe for 8-14 hours —or- oral ivermectin



Pediculosis- Management

- Capitis: OTC pediculicides highly resistant, retreat in 8-10 days
 - Pyrethrins w/piperonyl butoxide (RID, Pronto) ≥ 2 years or older
 - Permethrin 1% (Nix) ≥ 2 months or older
- Corporis: Prescription therapies
 - Malathion 0.5% gel/lotion (Ovide) ≥ 6 years or older
 - Spinosad 0.9% cream ≥ 6 months or older
 - Ivermectin 1% cream (Soolantra), 0.5% lotion (Sklice) ≥ 6 years or older

Permethrin 5% cream to entire body for 8-10 hrs – single application

- Pubis: Pyrethrins w/piperonyl butoxide (RID, Pronto): ≥ 2 years or older
- Permethrin 1% (Nix): ≥ 2 months or older
 - Ensure skin is cool/dry
 - Apply to all suspect areas
 - Wash after 10 minutes
 - Remove nits with nit comb, tweezers, or fingernails
 - Retreat in 10 days

Leone, P. A. (2007, April 1). Scabies and pediculosis: an update of treatment regimens and general review. Clinical Infectious Diseases, 44, S153-S159.

Scabies

- intensely pruritic eruption caused by the mite *Sarcoptes scabiei* transmitted via direct person-to-person contact of all ages and socioeconomic groups.
- typical infestation 10-20 mites. widespread and highly pruritic hypersensitivity reaction develops 2-6 weeks after initial infestation.
- in the elderly, the institutionalized, immunosuppressed or neurologic dysfunction, the mite burden is much higher, with thousands to millions of mites
- Burrows with small erythematous papules, are seen mostly on the flexor wrists, around the axillae and areolae, interdigital web spaces, umbilicus, genital, buttocks
- often spares the head and neck areas.



Scabies - Management

- Wood's Lamp: "dot" "line"
- Burrows may be more easily identified by applying ink from a fountain or marking pen to a suspected burrow, then wiping any excess away with an alcohol pad after a minute or two. The ink will penetrate the burrow, making it more visible.
- Perform a scabies prep. Place a small amount of mineral oil on the skin area to be tested, on a #15 blade, and on a
 microscope slide. Take the blade and gently remove the terminal end of the burrow where you see the tiny black speck.
 Apply this scraping to a glass slide, cover with a cover slip, and examine under the microscope for the presence of the mite
 or its ova or fecal pellets, known as scybala.
- In cases of crusted scabies, add a few drops of 10% potassium hydroxide (KOH) solution to the skin scraping to break down the excess keratin. Scales will typically contain many mites.
- Patients should be instructed to launder bed linens, towels, and clothing used in the last 72 hours prior to treatment in hot water and dry on high heat. Items that cannot be laundered can be sealed in air-tight plastic bags for 10-14 days. All carpets and upholstered furniture should be thoroughly vacuumed
- CDC 2021 Recommended Regimens:
 Permethrin cream (5%) applied to all areas of the body from the neck down and washed off after 8-14 hours OR
- Ivermectin 200 μg/kg orally, repeated in 2 weeks –OR- Ivermectin 1% lotion applied to the entire body from the neck down. Leave on for 8-14 hours. Repeat 7 days later if symptoms persist.
- Permethrin is effective and safe, and it is less expensive than ivermectin. Ivermectin has limited ovicidal activity therefore, a second dose of ivermectin should be administered 14 days after the first dose. Ivermectin should be taken with food

Scabies - Management

• CDC 2021 Alternative Regimen: Lindane (1%) 1 oz of lotion (or 30 g of cream) applied in a thin layer to all areas of the body from the neck down and thoroughly washed off after 8 hours. Lindane is an alternative regimen because it can cause toxicity; it should only be used if the patient cannot tolerate the recommended therapies or if these therapies have failed. Lindane should not be used immediately after a bath or shower, and it should not be used by persons who have extensive dermatitis or children aged younger than 10 years. Seizures and aplastic anemia has been reported.

Special Considerations – Pregnant or Lactating Patients:

Ivermectin likely poses a low risk however, due to limited data, permethrin is the preferred treatment.

Special Considerations – HIV Infection, immunosuppressed:

Pts should receive the same treatment regimens as those who are HIV negative but are at increased risk for crusted scabies. Consider consultation with a specialist.

Crusted Scabies:

Crusted (Norwegian) scabies is an aggressive infestation that usually occurs in immunodeficient, debilitated, or malnourished persons. Combination treatment is recommended with a topical scabicide, (5% topical benzyl benzoate or 5% topical permethrin cream; full-body application to be repeated daily for 7 days then twice weekly until cure), and treatment with oral ivermectin 200 μ g/kg on days 1,2,8,9, and 15. Lindane should be avoided because of the risks for neurotoxicity with heavy applications or denuded skin.

Pruritus: Antihistamines for pruritus is an important adjunctive treatment:

Flea Bites

- bloodsucking parasitic insects that live on pets, livestock, and humans. species most likely to bite humans is the cat flea or the rat flea
- 1-4 mm, blackish-brown. wingless, capable of jumping to a height of several meters. feed only on blood and capable of living long periods without feeding.
- legs and the ankles arranged in a linear or clustered pattern "breakfast, lunch, dinner pattern"
- bites, which are felt immediately, become increasingly irritated and may remain sore for as long as a week. Itching may be generalized or just at the site of the bites
- transient wheal, papular urticaria or bullae, recurrent or chronic, prolonged symptoms that may last for years



Flea Bites - Management

- Burow's solution compresses applied to the bites to relieve the itching
- Calamine lotion (to which 0.25% menthol and 0.5% phenol may be added by the pharmacist)
- mid-potency topical corticosteroids BID: triamcinolone cream, ointment; mometasone cream, ointment; fluocinolone cream, ointment
- oral antihistamines may be prescribed for the relief of pruritus

Spider Bites

- spiders have fangs that deliver venom via a small hole at the tips. Clinical manifestations cause skin lesions to systemic illness and, in rare cases, even death.
- Black widow spider: found in woodpiles. characteristic red hourglass figure
- bite is often very painful. Systemic symptoms: HTN, tachycardia, palpitations, diaphoresis, anxiety, shortness of breath, hyperthermia or hypothermia, excessive salivation, nausea, vomiting, abdominal pain
- Brown recluse spider: found in Midwest and Southern states, identified by the characteristic violin-shaped figure
- pain, swelling, bullae, and ischemia develop minutes to hours later. may eventually ulcerate and become necrotic and gangrenous, DIC may occur

 Photo Courtesy V.Lazareth NP





Spider Bites - Management

- Look closely for 2 small puncta, the fang marks of the spider.
- Ddx: cellulitis or furunculosis.
- Treatment includes:
 Collection and identification of spider, if possible.
- Wound irrigation.
- Rest, cold compresses, elevation of the affected extremity.
- Symptomatic treatment as indicated.
- Tetanus prophylaxis as indicated.
- Conservative local debridement of clearly necrotic tissue.
- Antivenom as indicated.

Lyme Disease

• immune-mediated inflammatory disease resulting from infection with the spirochete *Borrelia burgdorferi*. A red edematous plaque initially develops at the site of a tick bite. The lesion slowly expands outward for 3-6 weeks and may develop central clearing, forming a characteristic targetoid, or "bull's-eye," lesion.



- New England, Midwest, west coast. blacklegged tick or deer tick. Mice and deer
- spring and summer months. risk of infection is strongly linked to the amount of time spent in wooded or rural areas.
- spirochete disseminates to the nervous system, heart, joints, and other organs. Pts may develop acute neurologic abnormalities, atrioventricular block, or myocarditis.
- Untreated patients may progress to late-stage disease that manifests with arthritis, mild encephalopathy, neuropathies.

Lyme Disease Testing & Stages

CDC-recommended two-tier serologic testing:

Enzyme immunoassay (EIA) for detection of IgM and IgG antibodies. If there is a positive or equivocal result, complete step 2.

• Western blot (immunoblot) detects IgM and IgG antibodies to multiple individual components of the spirochete. Of note, a negative Western blot should supersede a positive EIA test and the testing is considered negative.

Considerations in testing early disease:

IgM antibodies appear within 1-2 weeks while IgG antibodies appear 2-6 weeks following the appearance of EM. Therefore, in early cases of EM, serological testing will be falsely negative and treatment should be initiated.

• Stage 1 - Early Localized (days to weeks following tick bite):

EM at the site of the tick bite develops in ~60%-90% of patients. There may or may not be systemic symptoms. False negative serological testing is common.

Stage 2 - Early Disseminated (weeks to months following tick bite):

Multiple widespread skin lesions represent dissemination of the infection with or without systemic symptoms. ~10%-15% of patients develop neurologic features (meningitis, cranial or peripheral neuropathies, facial nerve palsy. ~5% of patients may experience cardiac manifestations (AV block, myocarditis). Serological testing is usually positive, but false negatives may still occur.

• Stage 3 - Late (months to few years):

Untreated cases can also lead to: Chronic arthritis (typically knees), Mild encephalopathy with subtle cognitive deficits, Axonal polyneuropathies. Serological testing is virtually always positive.

• "Chronic" Lyme Disease or Post-Lyme Disease Syndrome:

Despite extensive study of this entity, numerous expert reviews have concluded that no consistent, reproducible syndrome or evidence of persistent infection resulting in these vague symptoms exists. However, primary reinfection has been demonstrated in individuals in endemic areas.

Lyme Disease - Management

- Infectious Diseases Society of America Guidelines Prophylaxis: doxycycline 200 mg in a single dose within 72 hours of tick bite
- Early Localized Disease: Doxycycline 100 mg by mouth every 12 hours for 14-21 days

 Pregnant patients: Amoxicillin 500 mg by mouth every 8 hours for 14-21 days.

 Alternative regimens: Cefuroxime axetil 500 mg by mouth every 12 hours for 14-21 days, OR Azithromycin 500 mg every 24 hours for 7-10 days, OR Clarithromycin 500 mg every 12 hours for 14-21 days, OR Erythromycin 500 mg every 6 hours for 14-21 days.
- Mild Early Disseminated Disease or Chronic Disease: Doxycycline 100 mg by mouth every 12 hours for 14-28 days.
 Pregnant patients: Amoxicillin 500 mg by mouth every 8 hours for 14-28 days.
 Alternative regimen: Cefuroxime axetil 500 mg by mouth every 12 hours for 14-28 days.
- Severe Early Disseminated Disease or Chronic Disease: Ceftriaxone 2 g intravenously (IV) every 24 hours for 14-28 days Alternative parenteral regimens: Cefotaxime 2 g IV every 8 hours for 14-28 days (duration dependent upon severity of infection), OR Penicillin G 18-24 million units IV daily, in divided doses every 4 hours, for 14-28 days (duration dependent upon severity of infection).

Rocky Mountain Spotted Fever

- 20%-30% fatal without treatment
- American dog and Rocky Mountain wood ticks. throughout the contiguous US, esp Arkansas, Missouri, NC, OK, TN. >90% April -Sept.
- Early: high fever, severe headache, myalgias, nausea, vomiting. Late: rash, photophobia, confusion, ataxia, seizures, cough, dyspnea, arrhythmias, jaundice, severe abdominal pain.
- The rash may not be apparent until 2-5 days after onset of fever. It begins as 1-5 mm macules, typically on the soles, ankles, palms, wrists, and forearms, spreading centripetally to the trunk. necrotic ulcerations and distal gangrene can be seen, resulting in amputations.
- A palm and sole petechial eruption in the setting of high fever, myalgias, and headache is characteristic of RMSF.



RMSF Testing & Treatment

- Serology: Indirect immunofluorescence assay (IFA) and ELISA
- Skin biopsy with direct immunofluorescence may help confirm diagnosis in the acute setting with a sensitivity of 70%.
- Immunohistochemical staining (IHC), polymerase chain reaction (PCR), and electron microscopy (EM) may aid in diagnosis.
- Treatment should be initiated immediately, prior to diagnostic confirmation, with high clinical suspicion.
- Doxycycline is the drug of choice for treatment of all tick-borne rickettsial disease in children and adults. Clinical response time is typically within 24-72 hours.
- Treat for at least 3 days after the fever subsides and when evidence of clinical improvement is noted. Standard duration of treatment is 7-14 days. Severe or complicated disease might require longer treatment courses.
- In the early / acute phase of illness, it may not be possible to distinguish between RMSF and meningococcemia. When in doubt, treatment for both diseases should be started as soon as possible.

Dengue Fever

- surging worldwide; particularly in the Americas. Puerto Rico declared a public health emergency in March 2024. cases increasing in FL, TX, CA
- spread by a type of warm weather mosquito that is expanding its geographic reach because of climate change. May experience headache, fever and flu-like symptoms. Severe cases can involve cause serious bleeding, shock and death.
- Repeated infections can be especially dangerous. There are four types
 of dengue virus. When someone is first infected, their body builds
 antibodies against that type but if they get infected with another type,
 the antibodies from the first infection may fail to neutralize the second
 type and can actually help the virus enter immune cells and replicate.
- There is no widely available medicine for treating dengue infections. Vaccine use has been sporadic due to restrictions and other issues

https://www.wbur.org/news/2024/06/28/dengue-fever-massachusetts-worldwide



Bites

Animal Bites

- 3%-18% of animal bites become infected. dog bites about 60% of animal bites, and 10%-20% by felines.
- Dog bites are more common in the summer months. In adults, the bites tend to be on the extremities. In children, bites are more commonly on the face
- Cat bites are more common in women and elderly individuals. Infection tends to develop approximately 8 hrs post injury.
- pain, cellulitis, purulent discharge
- regional adenopathy and fever, and tenosynovitis or osteomyelitis can develop if the wound is in close proximity to deeper structures.



Rabies Prophylaxis

- Ascertain whether attack was provoked or unprovoked, if the pet's rabies vaccination status is known, and if the pet can be apprehended.
 - If there is suspicion for rabies, the local health department should be notified. If rabies vaccination status is unknown and culprit animal can be captured and observed for 10 days for signs of rabies, no further testing is warranted provided normal animal behavior.
- If the animal appears to be rabid, initiation of post exposure prophylaxis is indicated with 20 IU/kg of rabies immunoglobulin, and the 4-dose regimen of human diploid rabies vaccine should be given on days 0, 3, 7, and 14 at separate sites to prevent neutralization of rabies vaccine by immunoglobulin.
- Elevation of the affected area is necessary for adequate lymphatic drainage, and if not performed properly, this can lead to treatment failure. A sling may be needed for upper extremity wounds.

 Follow-up should occur 24-48 hours after initial encounter to assess the wound site.
- Tetanus vaccine / tetanus immunoglobulin should be given based on CDC guidelines.
- Pain out of proportion to wound severity near a bone or joint is concerning for osteomyelitis.

The US Centers for Disease Control and Prevention (CDC) offers guidelines at: https://www.cdc.gov/rabies/medical_care/index.html.

Human Bites

- Human bites on the hands can occur as self-inflicted wounds, as accidents, or as a result of violence
- most severe human bites typically occur as "fight bites," with clenched fist injuries in which the patient's metacarpophalangeal (MCP) joint makes contact with someone's teeth.
- Risk factors: visibly dirty wound, delayed presentation, additional bite wounds, immunocompromised, hx of diabetes, asplenia, PVD
- Infectious symptoms may occur within 12 hrs of the bite injury



Human Bites - Pearls

- Bite wounds may be small, so perform a careful physical examination and consider the possibility of additional fractures and injuries.
- Focus on obtaining a thorough history to understand the injury mechanism, given that patients may be unwilling to reveal the cause of their injuries.
- Identify the time between injury and presentation to the hospital for treatment.
- Bite marks in young children may be signs of abuse and should be measured. Intercanine measurements over 3 cm are likely caused by a bite from an adult.
- Signs of acute infection include swelling, purulent discharge, fluctuance, pain, and fever.
- In a suspected fight, look for a wound over the dorsal MCP joint.
- The neurovascular status of the hand is typically preserved but perform a focused inspection of the hand if a fight bite injury is suspected.
- Evaluate extension at the MCP joint to assess the proximal extensor compartment and examine the proximal interphalangeal (PIP) joint via the Elson test for distal function.

Human Bites Testing

Radiographs:

X-rays should be used in all clenched fist injuries, penetrating scalp wounds in pediatric cases, and in wounds with visible tooth fragments or other foreign bodies.

Laboratory tests:

In patients with an obvious infection, obtain intraoperative tissue samples for Gram stain as well as aerobic and anaerobic cultures. In human bites, hepatitis B virus, hepatitis C virus, HIV, and syphilis status should be evaluated in the biter, and postexposure prophylaxis (PEP) should be used where appropriate.

- Alert the testing laboratory regarding the sample source because of the known difficulty in culturing oral flora such
 as Eikenella and Pasteurella.
- Tissue cultures in acute bite injuries with no signs of infection are rarely performed.
- Check ESR, C-reactive protein (CRP), and WBC in patients with delayed presentation for medical attention if there is a suspected infection.

• Initial management:

Most patients can be managed in the ED with irrigation, simple debridement, and prophylactic (IV+po) antibiotics.

• Operative repair of bite wounds to the hand is indicated in patients with tendon damage, bone penetration, and extensive wound contamination + P.T.

Bites - Management

- A comprehensive therapeutic approach of animal/human bites includes meticulous wound care, antimicrobial therapy, and vaccination.
- Wound Management
 Irrigation with copious amounts of normal saline should follow initial wound culture and Gram stain.
- Débridement of affected tissue.
- Primary wound closure should only be used for noninfected wounds; otherwise, approximate the edges of the wounds with adhesive tape.
- Closed fist injuries from humans should be allowed to heal via secondary intention.
- Radiograph of the affected area to check for retained tooth or nail.
- Elevation and immobilization of the wound should be stressed.

Bites - Management

Antimicrobial Therapy

Prophylactic antibiotics for 3-5 days should be given under the following circumstances: Deep puncture wounds

- Moderate to severe wounds with crush injury
- Wounds in hands or in close proximity to bone
- Wounds in immunocompromised hosts
- Wounds requiring surgical repair
- Treatment for cat and dog bites can include the following, with emphasis on broad coverage of anaerobic and gram-negative coverage. Of note, *Eikenella corrodens* and *Pasteurella* spp. have resistance to first-generation cephalosporins (such as cephalexin), dicloxacillin, erythromycin, and clindamycin.

Therapy for dog and cat bites can be divided into enteral vs. parenteral depending on the severity of the wound.

Enteral antibiotics:

Enteral antibiotics for 10-14 days are recommended when the wound appears without signs of a systemic inflammatory response, and the patient is otherwise stable.

First-line therapy: Amoxicillin-clavulanic acid 875/125 mg by mouth twice daily.

Second-line therapy in cases with penicillin allergy:

Clindamycin 300 mg by mouth 3 times daily plus either levofloxacin 500 mg by mouth daily or trimethoprim-sulfamethoxazole 2 double-strength tablets by mouth twice daily, or Doxycycline 100 mg by mouth twice daily, or Moxifloxacin 400 mg by mouth daily.

Bites - Management

Parenteral antibiotics:

Parenteral antibiotics are indicated when the patient has fever, sepsis, expanding cellulitis, inability to tolerate oral antibiotics, or a crush injury, or they are an immunocompromised host.

First-line therapy:

Ampicillin-sulbactam 3 g every 6 hours, or

- Piperacillin-tazobactam 4.5 g every 8 hours.
- Second-line therapy: Third-generation cephalosporin and metronidazole 500 mg IV every 8 hours.

Once patient has shown signs of stabilizing, convert to an enteral regimen to complete a total of 10-14 days.

Wounds leading to tenosynovitis will require 3-4 weeks of therapy.

Wounds leading to osteomyelitis will require 4-6 weeks of therapy.

Hospitalization may be required in cases of severe fever, sepsis, spreading cellulitis, substantial edema or crush injury, loss of function of affected area, or immunocompromised states.

Vaccination

Approach to tetanus and rabies prophylaxis is outlined in the section of Management Pearls.

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