CHAPTER 11

TREATMENT ALGORITHMS FOR DERMATOLOGIC CONDITIONS

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Abstract

Dermatologic treatment algorithms are organized by etiology and clinical morphology. The algorithm for common skin infections is etiology-specific. Bacterial infections like cellulitis are treated with systemic antibiotics targeting Gram-positives, whereas superficial impetigo responds to topical mupirocin. Fungal infections (tineas) are managed with an algorithm progressing from topical azoles or terbinafine to oral terbinafine or fluconazole for extensive disease or onychomycosis. Viral infections like HSV require episodic or suppressive oral antivirals (e.g., valacyclovir). Autoimmune skin disorder algorithms aim for inflammation control. Plaque psoriasis follows a stepwise algorithm from high-potency topical corticosteroids and vitamin D analogs, to phototherapy, and finally to systemic agents or biologics for moderate-to-severe disease. Bullous pemphigoid management prioritizes high-potency topical or systemic corticosteroids. Dermatitis algorithms are centered on barrier repair and inflammation suppression. Atopic dermatitis is managed with a foundational algorithm of emollients, followed by topical corticosteroids or calcineurin inhibitors. Skin cancer algorithms are dictated by tumor type and risk. Basal cell and squamous cell carcinomas are treated with an algorithm based on location and risk, primarily surgical excision or Mohs micrographic surgery. Melanoma management is a stage-dependent algorithm involving wide local excision, sentinel node biopsy for staging, and systemic immunotherapy or targeted therapy for advanced, metastatic disease.

Keywords: *Dermatitis, Psoriasis, Skin Infections, Skin Cancer, Melanoma, Treatment Algorithm*

Learning Objectives

After completion of the chapter, the learners should be able to:

- Identify first-line topical treatments for psoriasis, atopic dermatitis, and acne vulgaris.
- Explain the stepwise treatment algorithm for plaque psoriasis, from topical agents to phototherapy and systemic biologics.
- Select the correct antibiotic algorithm for a patient with non-purulent cellulitis versus a purulent skin abscess.
- Differentiate the surgical and medical management algorithms for basal cell carcinoma (BCC) versus invasive cutaneous melanoma.
- Justify the choice of Mohs micrographic surgery over standard excision for a non-Dermatologic skin cancer in a high-risk area.

COMMON SKIN INFECTIONS

The management algorithms for common skin infections are stratified by the causative organism: bacterium, fungus, or virus. An accurate diagnosis based on clinical presentation and, if necessary, microscopy or culture, is the first step in the algorithm.

Pathophysiology

Cellulitis (Non-purulent)

Cellulitis is an acute, spreading infection of the dermis and subcutaneous tissues. The pathophysiology begins with a breach in the skin barrier (e.g., a fissure, insect bite, tinea pedis, or trauma). This breach allows bacteria, most commonly Group A *Streptococcus* (e.g., *S. pyogenes*) or *Staphylococcus aureus*, to bypass the epidermis and proliferate. The bacteria release toxins and enzymes (like hyaluronidase and streptokinase) that break down the extracellular matrix, allowing the infection to spread rapidly along fascial planes. The host's potent inflammatory response, characterized by vasodilation and neutrophil infiltration, is responsible for the clinical signs: intense, warm erythema, edema, and pain.

Tinea Corporis (Ringworm)

Tinea corporis is a superficial fungal infection of the glabrous (non-hairy) skin caused by dermatophytes (e.g., *Trichophyton*

rubrum). The pathophysiology begins when fungal arthrospores adhere to the stratum corneum (the outermost dead layer of skin). The fungus then germinates, and its hyphae spread *radially* through the stratum corneum, digesting keratin as a food source. The fungus *only* lives in this dead keratin layer. The characteristic "ringworm" appearance (an annular plaque with central clearing and a scaling, active border) is a direct result of this growth pattern. The inflammation and scaling at the border represent the active, advancing front of the fungus, while the "central clearing" represents the area where the immune system has begun to clear the infection.

Diagnosis and Classification

- **Bacterial:** Infections like impetigo present with honey-crusted plaques. Cellulitis and erysipelas present as spreading, tender, erythematous, and edematous skin. A purulent focus (abscess or furuncle) suggests a staphylococcal, often MRSA, etiology.
- **Fungal (Dermatophytosis/Tinea):** These infections are classified by location (e.g., *tinea corporis* - body, *tinea pedis* - foot, *tinea cruris* - groin, *onychomycosis* - nail). The classic presentation is an annular (ring-shaped), scaling plaque with central clearing. Diagnosis is confirmed by KOH microscopy of skin scrapings, which reveals septate hyphae.
- **Viral:** Herpes Simplex (HSV) presents as grouped vesicles on an erythematous base, progressing to pustules and erosions. Varicella-Zoster Virus (VZV) reactivation (shingles) causes a dermatomal vesicular eruption. Human Papillomavirus (HPV) causes verrucae (warts).

Differential Diagnosis

The differential for bacterial cellulitis includes stasis dermatitis, which is often bilateral and lacks the acute, tender warmth of infection. Tinea corporis can mimic nummular eczema or psoriasis. Herpes simplex can be mistaken for bacterial impetigo or fungal infections, especially *tinea faciei*.

Treatment Algorithm

Bacterial Infections

- Impetigo: The algorithm for limited disease is topical mupirocin. For widespread infection, oral antibiotics (e.g., cephalexin) are required.
- Non-purulent Cellulitis/Erysipelas: The algorithm targets *Streptococcus*. Oral cephalexin, dicloxacillin, or clindamycin is first-line.
- Purulent Cellulitis/Abscess: This algorithm must cover MRSA. The primary treatment for an abscess is incision and drainage. Antibiotics (e.g., trimethoprim-sulfamethoxazole or doxycycline) are added based on severity.

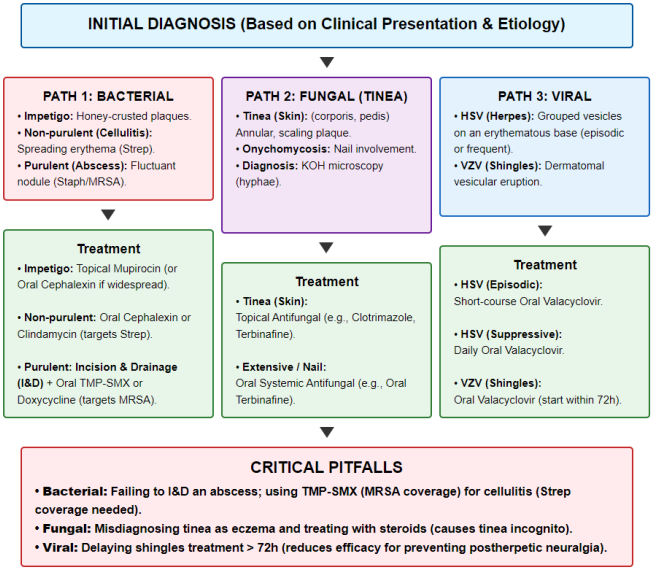

Figure 11.1: Common Skin Infections - Management Algorithm

Fungal Infections

- Tinea (Skin): The first-line algorithm for localized *tinea corporis*, *cruris*, or *pedis* is a topical antifungal cream (e.g.,

clotrimazole, ketoconazole, terbinafine) applied daily for 2-4 weeks.

- Extensive Tinea or Onychomycosis: If the infection is widespread, fails topical therapy, or involves the nails, the algorithm shifts to oral systemic therapy. Oral terbinafine is first-line for onychomycosis and most tineas.

Table 11.1: Treatment Algorithm for Common Skin Infections

Infection Type	Example(s)	Presentation	First-Line Treatment Algorithm
Bacterial	Impetigo	Honey-colored crusts.	**Topical:** Mupirocin. **Oral:** Cephalexin (if widespread).
Bacterial	Cellulitis	Spreading, warm, tender erythema.	**Oral:** Cephalexin (covers *Strep*). Add TMP-SMX (if MRSA suspected).
Fungal	Tinea Pedis / Corporis	Annular, scaling plaque, central clearing.	**Topical:** Terbinafine or Clotrimazole cream.
Viral	Herpes Zoster (Shingles)	Dermatomal, vesicular rash.	**Oral Antiviral:** Valacyclovir (start within 72h).

Viral Infections:

- HSV (Oro-labial/Genital): The algorithm is based on frequency and severity.
  - Episodic: For acute outbreaks, a short course of oral valacyclovir, acyclovir, or famciclovir is initiated

within 72 hours of onset.

- Suppressive: For patients with frequent recurrences, a lower-dose daily antiviral is used for long-term suppression.

Varicella-Zoster (Shingles)

The algorithm is oral valacyclovir or acyclovir initiated within 72 hours of rash onset to reduce the duration of rash and the risk of postherpetic neuralgia

Cellulitis

Monitoring and Follow-Up

Clinical monitoring is paramount. The aim is to **mark the borders of the erythema** with a skin marker at the time of diagnosis. This provides an objective measure of progression. The patient should show signs of clinical improvement (e.g., reduction in fever, pain, and, crucially, no further spread of the erythema) within 24-48 hours of starting effective antibiotics. Outpatients should have a follow-up check (in-person or phone) at 48 hours. Inpatients require daily monitoring of vital signs and the infection site. Failure to improve warrants investigation for a resistant organism (MRSA), a deeper infection (abscess, necrotizing fasciitis), or an incorrect diagnosis (e.g., stasis dermatitis).

Long-Term Management / Secondary Prevention

The primary long-term management is **risk factor reduction**. For patients with recurrent cellulitis, the algorithm focuses on identifying and managing the portal of entry. This includes:

1. **Treating Tinea Pedis:** This is a very common and often-overlooked entry point. All patients with cellulitis of the leg should have their feet inspected and treated for athlete's foot.
2. **Lymphedema Management:** Patients with chronic edema or lymphedema (e.g., post-surgery or due to venous insufficiency) require meticulous skin care, emollients, and often compression therapy to reduce the "culture medium" of static lymph fluid.
3. **Prophylactic Antibiotics:** For patients with 3-4

episodes per year despite other measures, low-dose daily oral penicillin or erythromycin may be considered.

Patient Counseling Points

1. **Elevation:** "The most important non-antibiotic treatment is to elevate your leg above the level of your heart. This uses gravity to help drain the swelling, which reduces pain and helps the antibiotic get to the infection."
2. **Warning Signs:** "We have marked the edge of the infection. If you see this redness spreading *past* the line, or if you develop a high fever, severe pain, or any blisters, you must come back to the emergency room immediately. This could be a sign of a more dangerous infection."
3. **Find the Cause:** "This infection got in through a small crack in your skin. We must find it. This often means treating any athlete's foot you might have between your toes, or using lotion daily to prevent dry, cracked skin."

Ring worm

Monitoring and Follow-Up

Monitoring is clinical. The goal is the resolution of erythema, scaling, and pruritus. Patients are instructed to apply topical antifungal cream (e.g., clotrimazole, terbinafine) not just to the ring, but to at least 2 cm of normal-looking skin *beyond* the active border. A common error is stopping treatment as soon as the rash looks better; treatment must be continued for 1-2 weeks *after* the rash has completely resolved to eradicate subclinical spores. Failure to improve on topical therapy warrants a re-evaluation of the diagnosis or consideration of oral antifungal therapy.

Long-Term Management / Secondary Prevention

Prevention is focused on hygiene and moisture control. This includes drying the skin thoroughly after bathing, especially in

skin folds. Patients should be advised to avoid sharing towels or clothing. In athletes (e.g., wrestlers, *tinea corporis gladiatorum*), this involves showering immediately after practice and not sharing equipment. Any household pets, particularly new kittens, should be checked by a veterinarian as they can be asymptomatic carriers of dermatophytes (e.g., *Microsporum canis*).

Patient Counseling Points

1. "**This is Not a Worm":** "The name 'ringworm' is very misleading. This is not a worm; it is a common, superficial fungal infection of the skin, just like athlete's foot. It is very treatable."
2. **How to Apply Cream:** "You must apply the cream not just to the red ring, but to a border of normal skin around it. Keep applying the cream for at least one full week *after* the rash is completely gone to prevent it from coming back."
3. **Avoid Steroids:** "Do not put any steroid cream (like hydrocortisone) on this. A steroid will make the rash *look* less red, but it is 'food' for the fungus and will make the infection spread and drive deeper into the skin. This is a very common mistake."

Common Pitfalls in Management

A common pitfall is misdiagnosing tinea corporis as nummular eczema and prescribing topical steroids alone; this can worsen the infection (*tinea incognito*). In bacterial infections, a critical error is failing to cover MRSA in a purulent infection or not performing an incision and drainage on a fluctuant abscess. In shingles, delaying antiviral therapy beyond the 72-hour window significantly reduces its efficacy in preventing postherpetic neuralgia.

Case Study

A 48-year-old male with Type 2 Diabetes presents to urgent care with a 2-day history of a rapidly expanding, red, hot, and tender rash on his left lower leg. The exam shows a well-demarcated, erythematous plaque with no central purulence,

fluctuance, or abscess. He has a fever of 101.1°F.

Discussion

This is the classic presentation of **non-purulent cellulitis**. The algorithm is bifurcated from purulent SSTIs (like an abscess). For non-purulent cellulitis, the primary pathogen is assumed to be *Streptococcus*, and the treatment algorithm must cover it.

Treatment Algorithm

1. **Diagnosis:** Non-purulent cellulitis (a bacterial infection).
2. **Algorithm (Non-Purulent):** The primary treatment is antibiotics. Drainage is not possible.
3. **Therapy:** The antibiotic choice must cover *Streptococcus*.
    - **Action:** Start **Cephalexin 500 mg QID for 7 days**. (Note: TMP-SMX or Doxycycline, which cover MRSA, would be *incorrect* first-line monotherapy here as they have poor *Strep* coverage).
4. **Counseling:** You draw a line around the border of the erythema and instruct the patient to elevate his leg. He is told to return immediately if the redness spreads past the line or his fever persists.

Outcome

The patient is started on Cephalexin and his symptoms begin to improve within 36 hours. You also treat his tinea pedis ("athlete's foot"), which was the likely portal of entry for the bacteria.

AUTOIMMUNE SKIN DISORDERS

Autoimmune skin disorders are chronic, inflammatory conditions where the immune system attacks components of the skin. Management algorithms are typically stepwise, aiming to control inflammation and induce remission.

Psoriasis

Pathophysiology

Psoriasis is a chronic, immune-mediated inflammatory disease with a strong genetic predisposition. The pathophysiology is a profound dysregulation of the skin's immune system, primarily driven by the **IL-23/Th17 axis**. In psoriasis, a trigger (e.g., infection, trauma) causes dendritic cells to release Interleukin-23 (IL-23). IL-23 activates T-helper 17 (Th17) cells, which in turn release a cascade of pro-inflammatory cytokines, especially **IL-17** and **TNF-α**. These cytokines signal to the keratinocytes (skin cells) to **hyperproliferate**. Normally, keratinocytes mature and shed over ~28 days. In psoriasis, this process is accelerated to ~3-5 days. This rapid turnover and incomplete maturation are what create the characteristic, thick, silvery-scaled plaque.

Diagnosis and Differential

Psoriasis, most commonly plaque psoriasis, is a chronic inflammatory disease presenting as well-demarcated, erythematous plaques with silvery scale, typically on extensor surfaces (elbows, knees), the scalp, and the lumbosacral area. Diagnosis is clinical. The differential includes seborrheic dermatitis (greasier scale, in scalp and face), nummular eczema (coin-shaped, very pruritic), and tinea corporis.

Treatment Algorithm

The psoriasis algorithm is stepwise, based on disease severity (Body Surface Area, BSA, and quality of life impact):

Mild-to-Moderate (e.g., < 10% BSA):

- **First-line:** High-potency topical corticosteroids (e.g.,