

Project: Ghana Emergency Medicine Collaborative

Document Title: Skin Emergencies

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Table 118-1 Definitions of Skin Lesions

LESION	APPEARANCE
Macule	Flat; color differs from surrounding skin
Patch	A macule with surface changes (i.e., scale or wrinkling)
Papule	Elevated skin lesion <0.5 cm in diameter
Plaque	Elevated skin lesion >0.5 cm in diameter; without substantial depth
Nodule	Elevated skin lesion >0.5 cm in diameter and depth
Cyst	Nodule filled with expressible material
Vesicle	Blisters <0.5 cm in diameter filled with clear fluid
Bullae	Blisters >0.5 cm in diameter filled with clear fluid
Pustule	Vesicle filled with cloudy or purulent fluid
Crust	Liquid debris that has dried on the skin surface; usually moist and yellowish brown
Scale	Visibly thickened stratum corneum; usually white
Lichenification	Epidermal thickening characterized by visible and palpable skin thickening and accentuated skin markings
Induration	Dermal thickening that <i>feels</i> thick and firm
Wheal	Papule or plaque of dermal edema; often with central pallor and irregular borders
Erythema	Red appearance of skin caused by vasodilation of dermal blood vessels; blanchable
Purpura	Red appearance of skin caused by blood extravasated from disrupted dermal blood vessels; nonblanchable
Macular purpura	Flat, nonpalpable
Papular purpura	Elevated, palpable

Lesion Definition

Perspective

- Skin conditions and related complaints \approx 4-12% of all US emergency department visits.
- Three important factors:
 - onset and evolution of the skin problem
 - associated symptoms
 - prior treatment
- Cutaneous eruptions can be manifestations of primary dermatologic disease or can signal underlying systemic illness.

Growths vs. Rashes

- Growths
 - Epidermal, pigmented, and dermal or subcutaneous proliferative processes.
- Rashes
 - Two groups depending on whether epidermis is involved.
 - (1) Lesions and rashes with epidermal involvement include eczematous rashes, scaling as well as vesicular, papular, pustular, and hypopigmented rashes.
 - (2) Rashes without epidermal involvement include erythema, purpura, and induration.

Diagnosis

- Occasionally, configuration disease-specific
 - Morphology of the primary lesion is usually given more diagnostic weight
- Many skin diseases have preferential areas of involvement
 - Location may aid in diagnosis
- Nikolsky's Sign
 - *When lateral pressure on un-blistered skin causes the epidermis to slide off, with separation of large sheets of epidermis from underlying dermis*



Source Undetermined



Source Undetermined



Case #1

- 72 year old male presents with a one day history of lip swelling. He denies breathlessness and otherwise has no complaints. He also denies any change in medications, consumption of new foods or environmental exposures.
 - He has hypertension controlled by a medication whose name he cannot recall that was initiated 8 years ago.
 - ***Diagnosis/Disposition?***



Source Undetermined



Hypersensitivity Reactions

- Type I (immediate hypersensitivity)
 - IgE (and IgG4) mediated ; -- most allergic/anaphylactic reactions in humans.
- Type II (cytotoxic)
 - antibody-mediated cytotoxic reaction; complement-fixing IgG (or IgM) engages cell-bound antigen, activating the classic complement pathway, leading to the fixation of membrane attack complexes and cell lysis.
- Type III (immune complex)
 - IgG or IgM complex mediated. Circulating soluble antigen-antibody immune complexes migrate from the circulation to deposit in the perivascular interstitial space, thereby activating the complement system.
- Type IV (delayed hypersensitivity)
 - T cell mediated; no documented relationship to the pathogenesis of anaphylaxis.

Hypersensitivity Reactions

Table 5 - Comparison of Different Types of hypersensitivity

characteristics	type-I (anaphylactic)	type-II (cytotoxic)	type-III (immune complex)	type-IV (delayed type)
antibody	IgE	IgG, IgM	IgG, IgM	None
antigen	exogenous	cell surface	soluble	tissues & organs
response time	15-30 minutes	minutes-hours	3-8 hours	48-72 hours
appearance	weal & flare	lysis and necrosis	erythema and edema, necrosis	erythema and induration
histology	basophils and eosinophil	antibody and complement	complement and neutrophils	monocytes and lymphocytes
transferred with	antibody	antibody	antibody	T-cells
examples	allergic asthma, hay fever	erythroblastosis fetalis, Goodpasture's nephritis	SLE, farmer's lung disease	tuberculin test, poison ivy, granuloma

Urticaria & Angioedema



Heckat, [Wikimedia Commons](#)



- Urticaria (hives)
 - reaction that consists of papules or wheals that are non-pitting, edematous, pruritic, slightly erythematous, raised circular or annular, and range in size from millimeters to several centimeters.
 - Centers are usually clear , borders can be serpiginous.
 - Erythema due to dilation of blood vessels in the dermal layer of the skin and the edematous wheals are due to transudation from these blood vessels.
 - Urticaria favors the extremities and trunk and is usually transient, with crops of hives appearing/resolving spontaneously in a matter of hours.

Angioedema

- Similar to urticaria
 - But involves the deeper dermal and subcutaneous tissue.
 - (+) Urticaria = indicates a mast cell component to the reaction.
 - (-) Urticaria = kinin-related.
- Commonly involves the face, mouth, lips, tongue, extremities, and genitalia
- Acute
 - Episodes that last < 6 weeks (90%)
- Chronic
 - persist longer than 6 weeks (10%).



Source Undetermined

Angioedema

- (+) Urticaria (mast cell degranulation)
 - IgE mediated, antihistamines (*both H1 and H2*) 1st Line
 - Adrenaline can be considered for moderate to severe cases caution in patients older than 35 years to ↓ risk of precipitating an acute coronary syndrome.
 - Steroids may help in preventing rebound Sx
- (-) Urticaria (10%)
 - usually kinin-related and causes include hereditary angioedema (HAE), acquired C1 inhibitor deficiency (ACID), and ACE inhibitors. ↑↑ bradykinin levels.
 - Treatment is mostly supportive, fewer Rx options
 - **Active airway management** is the mainstay of treatment
 - ?Racemic Adrenaline (Epinephrine) aerosol for airway edema
 - ?FFP (contains C1 Inhibitor)
 - *inhibition of ACE, one of the main inactivators of bradykinin, results in increased bradykinin levels. Rare reports of inciting worsened Sx*



Source Undetermined



ACE Inhibitor-Induced



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Source Undetermined

- Incidence of 0.1-0.7% and has a predilection for the tongue, lips, and laryngeal soft tissue.
- The highest incidence occurs in the first month of therapy,
 - *But can occur as many as 10 years after therapy is started.*
- Specific risk factors include African ancestry, smoking, older age, and female gender
 - some risk associated with DM.
- Pathophysiology
 - Prevention of bradykinin and substance P metabolism, both of which are potent mediators of tissue inflammation.
 - Most patients who develop angioedema on ACE inhibitors should be able to tolerate angiotensin receptor blocker drugs (ARBs).

Case #2

- 10 year old male with a history of recent TMP/SMX Rx for a peri-rectal abscess (*resolved*) presents with complaint of rash located primarily on the anterior thoraco-abdominal surface and palms.
 - ***Diagnosis/Disposition?***




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Source Undetermined

Erythema Multiforme



Source Undetermined 

- Acute, usually self-limited disease precipitated by a variety of factors.
 - Sudden appearance of skin lesions that are erythematous or violaceous macules, papules, vesicles, or bullae.
 - Distribution often symmetrical, most commonly involving the soles and palms, the backs of the hands or feet, and the extensor surfaces of the extremities.
 - Stevens-Johnson Syndrome (a severe form) is occasionally fatal.
 - characterized by bullae, mucous membrane lesions, and multisystem involvement

- Initial Lesion → Target Lesion
 - Three zones of color is the hallmark
 - Central dark papule or vesicle that is surrounded by a pale zone and halo of erythema
 - Commonly found on the hands or wrist.



Source Undetermined



Etiologies

- Most common precipitating factors
 - Exposure to medications ($\leq 10\%$)
 - NSAIDs, sulfonamides, multiple anti-epileptics
 - *Antibiotics are responsible for the majority of cases*
 - HSV Infection
 - lesions appear approximately 10 days after an outbreak
 - Other
 - Hepatitis and influenza A.
 - Less common causes
 - fungal diseases, such as dermatophytosis, histoplasmosis, and coccidioidomycosis, and bacterial infections, especially streptococcal infections and tuberculosis.
 - Various collagen vascular disorders have been known to precipitate erythema multiforme, particularly rheumatoid arthritis, systemic lupus erythematosus, dermatomyositis, and periarteritis nodosa.
 - Pregnancy and various malignancies have also been associated with erythema multiforme.

Management

- Address underlying cause
- Mild forms (*Discharge*)
 - Resolve spontaneously in 2-3 weeks
 - Oral antivirals (acyclovir, valacyclovir, or famciclovir) may be used to prevent both HSV (oral and genital) and EM
 - With the distinctive clinical findings and no systemic symptoms, patients can be discharged home.
- Severe cases (*Admit*)
 - May last up to 6 weeks
 - IV hydration, local skin care, systemic analgesia, and systemic corticosteroid therapy (80-120 mg of prednisone daily in divided doses)
 - Bullous Lesions
 - treated with the application of wet compresses soaked in a 1 : 16,000 solution of potassium permanganate or a 0.05% silver nitrate solution several times a day.

Complications

- Stevens-Johnson Syndrome
 - Infection and fluid loss.
 - Renal involvement and pneumonia are rare.
 - Severe conjunctivitis
 - may result in corneal scarring and blindness.

Case #3

- 44 year old male reports worsening rash over the last 2 days with a 5 day history of sore throat, fever, myalgia.
 - Has not taken antibiotics but has been using ibuprofen.
 - ***Diagnosis/Disposition?***



Source Undetermined



Stevens-Johnson Syndrome/Toxic Epidermal Necrolysis

- Life-threatening, reactive diseases that represent two ends of a continuum
 - SJS
 - <10% TBSA epidermal detachment and
 - TEN
 - >30% TBSA epidermal detachment.
 - Overlap occurs in 10% to 30% TBSA.
- Two or more mucosal sites are usually affected.
 - The overall mortality of TEN approaches 30%.

SJS/TEN

- Prodromal Symptoms
 - Malaise, rhinitis, sore throat, body aches, and fever.
 - Followed by the abrupt development of a macular rash that may or may not appear as target lesions.
 - Mucous membrane involvement may precedes rash in TEN
 - *Macular exanthem usually starts centrally and then spreads to the extremities*
- Bullae form within the rash and large sheets of epidermis separate from the dermis.
 - Involved skin is exquisitely tender to palpation.
 - ***Nikolsky Sign (+)***

Progression

- Mucous membrane involvement develops
- Conjunctival and corneal involvement may lead to permanent scarring and blindness.
 - Full thickness of epidermis involved
- Mortality rate 15-20%
 - as high as 50% in elderly patients
- Life-threatening metabolic derangements, sepsis, respiratory failure, and gastrointestinal hemorrhage may occur and are compounded by underlying comorbidities.

Causes

- Non-staph TEN usually assoc with drug exposure
- The Main Culprits
 - Over 200 medications
 - Long-acting sulfa drugs, penicillin, quinolones, tetracycline, aspirin, barbiturates, phenytoin, phenobarbital, carbamazepine, allopurinol, and non-steroidal anti-inflammatory drugs (NSAIDs)
 - TEN has occurred after vaccination and immunization against poliomyelitis, measles, smallpox, diphtheria, and tetanus

SJS



Source Undetermined



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Source Undetermined

TEN



Source Undetermined

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Bullous SJS/TEN



Source Undetermined

Treatment

- Supportive Care
 - Discontinue offending agent, fluid replacement, electrolyte balance, ophthalmologic assessment and aggressive infection control
 - Systemic corticosteroids remain controversial
 - *no benefit and associated with increased mortality secondary to infection*
- Admission to a Burn ICU
 - Fluid replacement
 - Per Parkland Formula with Ringer's Lactate
 - Goal UOP 0.5cc/kg/hr
 - Wound Care (*no Silvadine = Sulfa*)
 - Infection control
 - IVIG per local protocol
 - Fas ligand (FasL) expression hypothesized as the “death ligand” induced for abnormal apoptosis of epidermal cells
 - ↑ in SJS/TEN patients
 - Pooled human immunoglobulin contain anti-Fas antibodies that have been shown *in vitro to impede apoptosis when pre-incubated with keratinocytes*

Pearls

1. The hair-bearing scalp is spared even in severe disease.
2. Neutropenia is associated with a poor prognosis.
3. Cross reactions within anticonvulsants are common. The first 8 weeks of treatment have the highest risk of TEN and previous anticonvulsant therapy portends a tenfold increased risk.
4. Patients with previous SJS/TEN to one medication class are higher risk for developing SJS/TEN to other medication classes.

Case #4

- 32 year old female with no past history presents with 5 days of fever, rash, vomiting and new altered mental status/profound hypotension.
 - Last menstrual period was 7 days ago
 - Intubated at outside hospital and transferred with possible sepsis...
 - *Diagnosis/Disposition?*



Source Undetermined



Source Undetermined



Toxic Shock Syndrome

- Acute febrile illness characterized by a diffuse desquamating erythroderma.
- Classically composed of high fever, hypotension, constitutional symptoms, multi-organ involvement and rash

BOX 135-2**DEFINITION OF STREPTOCOCCAL TOXIC SHOCK SYNDROME**

Must meet criteria from both 1 and 2 below:

1. Isolation of group A *Streptococcus* from:
 - a. A normally sterile site such as blood or cerebrospinal fluid is a definite case.
 - b. A normally nonsterile site such as sputum or skin lesion is a probable case.
2. Hypotension and at least two of the following:
 - a. Renal impairment
 - b. Coagulopathy
 - c. Liver involvement
 - d. Adult respiratory distress syndrome
 - e. Generalized erythematous macular rash that may desquamate
 - f. Soft tissue necrosis

Causes

- Notoriety in the early 1980s because of association with tampon
 - Overall incidence of toxic shock syndrome has since decreased
 - Has been seen in both men and children as well as after nasal packing placement for epistaxis
- Linked to exotoxin-producing *S. aureus*.
- Most cases of non-menstrual TSS occur in the post-operative setting.

- In menstruating women with toxic shock syndrome
 - *S. aureus* is isolated more than 90% of the time.
- Clinical manifestations
 - Due to the exfoliative exotoxin produced by *S. aureus*.
 - Is the same exotoxin produced in bullous impetigo
 - TSS ← from systemic circulating exotoxin
 - Bullous impetigo ← direct *S. aureus* inoculum.
 - The exotoxin in both cases has exquisite specificity in causing loss of desmosome-mediated cell adhesion within the superficial epidermis only.

TSS Diagnosis

- Requires the presence of
 - (1) fever of at least 38.9°C
 - (2) hypotension, SBP <90 mmHg
 - (3) skin rash
 - Diffuse, blanching, non-pruritic macular erythroderma rash
 - Desquamation 1-2 weeks after onset of illness
 - *esp. Palms/Soles*
 - (4) involvement of ≥ 3 organ systems.
- Systemic involvement may include the gastrointestinal (GI) tract, muscular system, or central nervous system (CNS) and laboratory evidence of renal, hepatic, or hematologic dysfunction

BOX 135-1**TOXIC SHOCK SYNDROME: CRITERIA FOR DIAGNOSIS**

Fever of 38.9°C (102°F) or higher

Rash (diffuse macular erythema) that resembles the rash of scarlet fever

Desquamation of skin 1 to 2 weeks after onset of disease

Hypotension (systolic blood pressure <90 mm Hg, orthostatic drop of 15 mm Hg or more, or orthostatic dizziness or syncope)

Clinical or laboratory abnormalities in at least three organ systems:

Gastrointestinal: nausea and vomiting, diarrhea

Muscular: myalgia or creatine phosphokinase at least two times normal level

Mucous membrane: vaginal, oropharyngeal, or conjunctival hyperemia

Renal: blood urea nitrogen or creatinine level at least twice normal or pyuria greater than five cells per high-power field

Hepatic: bilirubin, serum transaminases at least twice normal level

Hematologic: thrombocytopenia, less than 100,000/mm³

Neurologic: disorientation or altered consciousness without focal findings

Reasonable evidence for the absence of other cause of illness

Multi-System Issues

- ≥ 3 three of the following
 - GI
 - Profuse diarrhea/vomiting at illness onset
 - MSK
 - Sever myalgia *or* $>$ two-fold increase in CPK
 - Mucosal Inflammation
 - Conjunctival, vaginal, pharyngeal hyperemia
 - Renal
 - \uparrow BUN or creatinine $>$ two times upper limit
 - Hepatic
 - TBili or LFTs $>$ two times the upper limit
 - Hematologic
 - Thrombocytopenia (PLT $<100,000/\text{mm}^3$)
 - CNS
 - Disorientation, confusion, hallucination

TSS Physical Findings

- Subjective
 - Headache, myalgias, arthralgia, alteration of consciousness, nausea, vomiting, and diarrhea
- Objective
 - Rash
 - Diffuse, blanching, macular erythroderma → desquamation
 - Accompanying nonexudative mucous membrane inflammation is common.
 - Pharyngitis
 - Sometimes accompanied by a “strawberry tongue”
 - Other
 - Conjunctivitis, or vaginitis may be seen.
 - As a rule, the rash fades within 3 days of its appearance.
 - This is followed by a full-thickness desquamation, most commonly involving the hands and feet.

Investigations

- Basic Labs
 - FBC, PT/PTT, LFTs, Renal Panel, Blood Cultures
 - ABG
 - Urinalysis
 - Creatinine Phosphokinase
 - Consider LP
- Imaging
 - CXR
- Pelvic Exam – *TSST-1 exotoxin assay*
 - Commonly negative

Management

- Hypotension
 - 4-20L IVF
 - What is the appropriate fluid choice?
 - What may be required if the patient receives 20L of RL?
 - Vaso-active medications prn
- Infection Management
 - Remove tampon (or nasal/wound packing)
 - *Clindamycin*:
 - potent suppressor of bacterial toxin synthesis
 - Other Antibiotic Therapy not clearly indicated unless source is clear
 - If needed then lactamase resistant penicillin (nafcillin or oxacillin)
 - OBGYN-guided drainage if abscess source found

Management

- ICU Care: Early goal-directed therapy for sepsis
 - Restoration and maintenance of adequate central venous pressure and mean arterial pressure, maintaining central venous oxygenation, and achieving adequate urine output
 - May consist of central venous pressure monitoring to direct aggressive fluid resuscitation and the use of alpha- and beta- adrenergic vasoactive agents.
 - Intravenous antibiotics that cover for penicillinase-producing staphylococci, such as nafcillin or oxacillin, can be used when there is low probability for MRSA.
 - Clindamycin and Vancomycin = MRSA coverage.

What about Strep?

- Mid-1980s
 - Streptococcal toxic shock–like syndrome described worldwide in younger otherwise healthy patients with serious soft tissue infections.
 - 1987: two cases of shock due to isolated *S. pyogenes* soft tissue infections reported and postulated a toxin responsible (Cone)
 - 1989: further characterized syndrome as involving otherwise healthy young patients who presented in shock or progressed to a shock state within 4 hours of admission (Stevens)
 - Although these cases are predominantly caused by Lancefield group A strains, other groups have also been shown to cause streptococcal toxic shock syndrome.

Virulence

- Attributed to
 - Surface proteins, toxin production, host factors.
 - Strains that produce M protein associated with overwhelming infection.
 - M protein binds to complement control factors and other host proteins to prevent activation of the alternate complement pathway and thus evade phagocytosis and killing by polymorphonuclear neutrophil leukocytes.
 - Extracellular toxins, including super antigenic streptococcal pyrogenic exotoxins, contribute to tissue invasion and initiate the cytokine storm believed to be responsible for illnesses such as necrotizing fasciitis and the highly lethal streptococcal toxic shock syndrome

Mechanism

- Toxins act as Super-Antigens
 - Activate a large population of T cells, bypassing the antigen presentation phase and liberating massive amounts of cytokines, tumor necrosis factor alpha, interleukin-1, and interleukin-6.
 - Cytokines induce the clinical signs of fever, hypotension, shock, rash, and ultimately multi-organ failure.
 - *Lack of host antibodies to the surface proteins* and toxins predispose to infection and increased virulence.

Management

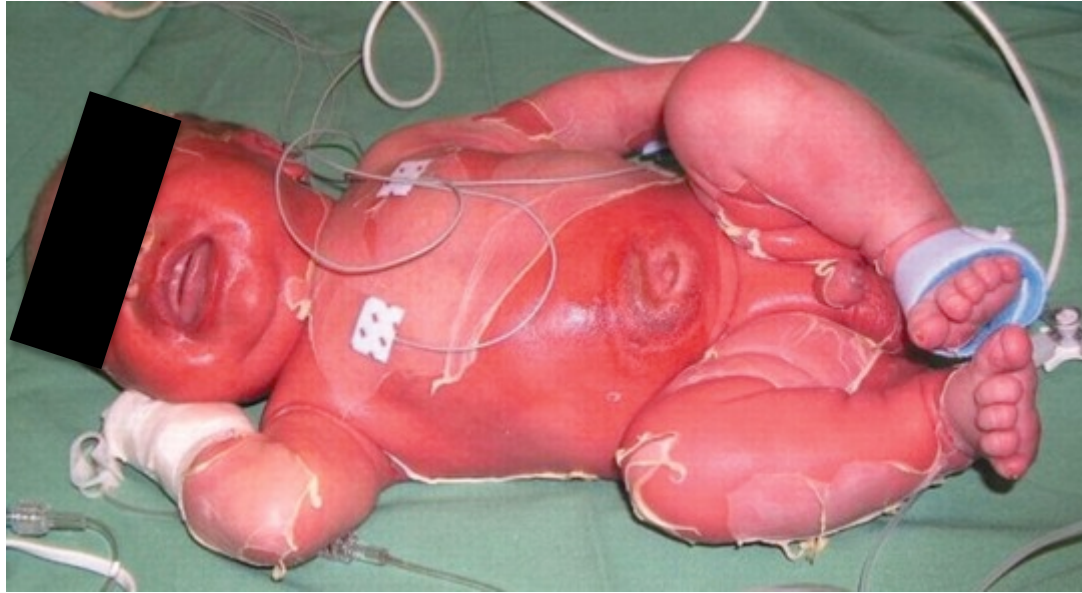
- Early goal-directed therapy for sepsis as described above for staphylococcal toxic shock syndrome is a mainstay of treatment.
- Even with appropriate antibiotic therapy and intensive supportive care, the mortality rate for this disease is 30-70%.

Antibiotics

- Choices
 - Clindamycin, erythromycin, or ceftriaxone + clindamycin.
 - Penicillin is only moderately effective against the large inoculum of slow-growing *Streptococcus* seen in necrotizing fasciitis or myositis.
- Clindamycin
 - Thought to work by two mechanisms:
 - (1) effective killing of *S. pyogenes* organisms
 - (2) decreasing production of extracellular products that play a role in the pathogenesis of systemic toxicity and/or tissue destruction.
- Resistance?

Some clindamycin-resistant strains of *S. pyogenes*, so presumptive selection clindamycin and a β -lactam antibiotic would be prudent in the initial treatment

Case #5



Source Undetermined

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- 4 month old male is brought to A&E with report of several days of fever, cough, congestion and new rash
 - *Disposition/Diagnosis?*

Staph Scalded Skin Syndrome

- Generally occurs in children ≤ 6 years old
- Caused by phage group 2 exotoxin-producing staphylococci (*Staphylococcus aureus*).
- Prodrome
 - *fever, malaise, and irritability following an upper respiratory infection with pharyngitis or conjunctivitis.*
- Rash
 - Begins with erythema and crusting around the mouth, then faint, diffuse erythematous macular rash spreads peripherally, followed by flaccid bulla formation and desquamation in 2-3 days.
 - *exfoliation over large surface area \rightarrow fluid and electrolyte losses.*
 - Mucous membranes are usually not involved.
 - ***After desquamation, lesions dry up with clinical resolution in 3-7 days.***

Management

- Technically, most patients will recover without antibiotics
- Most group 2 toxin-producing organisms are penicillin resistant.
- Current Recommendations
 - IV therapy with 50-100 mg/kg of nafcillin daily or oral cloxacillin 50mg/kg/day or dicloxacillin (vs clindamycin)
- *Admission usually necessary, especially infants.*
 - *Requires careful attention to fluid and electrolyte losses and the prevention of secondary infection of the denuded skin.*
 - *Requires effective management of pain*

Pearls

- The fluid in the bullae of staphylococcal scalded skin syndrome is sterile.
 - The toxin is produced at a remote site and delivered to the skin via the bloodstream.
- Infants with involvement of large areas of the body surface are at increased risk for hypothermia.
- Corticosteroids are contraindicated in the treatment of staphylococcal scalded skin syndrome.

Case #6

- 35 year old female presents three days after having undergone I&D of right axillary abscess. Her wound was packed and she was discharged without antibiotics. She returns today for a wound check and dressing change. She reports increased pain at the site and is febrile in triage.

— ***Diagnosis/Disposition?***



Source Undetermined



Hidradenitis Suppurativa

- Disease of Apocrine Sweat Glands
 - Recurrent abscess formation in the axillae and groin resembles localized furunculosis.
 - Tends to be recurrent and may be extremely resistant to therapy
 - Treatment
 - Incision & Drainage/Packing
 - Anti-staphylococcal antibiotics
 - Theatre/OR-drainage *if refractory to A&E treatment*

Case #7

- 21 year old female presents with several days of worsening external vaginal pain, swelling and fever without discharge.

– ***Diagnosis/Disposition?***



Source Undetermined



Bartholin's Cyst Abscess

- Obstructed Bartholin duct
 - Flora usually mixture of aerobic and anaerobic flora from the vagina, with *Chlamydia trachomatis* and *N. gonorrhoeae* cultured $\approx 10\%$ of the time.
 - Patient usually has a painful cystic mass on the inferior lateral margin of the vaginal introitus.
 - Signs and symptoms are usually localized, but septic shock can occur in rare cases.

Management

- Incision & Drainage
 - Drained from mucosal rather than cutaneous surface.
 - Simple incision and drainage carries a high risk of recurrence, so the cavity should be packed open.
 - Word Catheter
 - 10-Fr balloon catheter (*specifically designed*)
 - After incision and drainage, catheter is inserted and inflated with 2-5 mL of water or saline
 - Remains for 4-6 weeks so that a sinus will have time to form.
 - Sitz Baths
 - Warm H₂O “hip bath” helps keep area clean and draining.
 - Marsupialization
 - More complex treatment option, involving incision and drainage of the cavity followed by suturing of the walls of the cyst to the skin

INCISION AND DRAINAGE

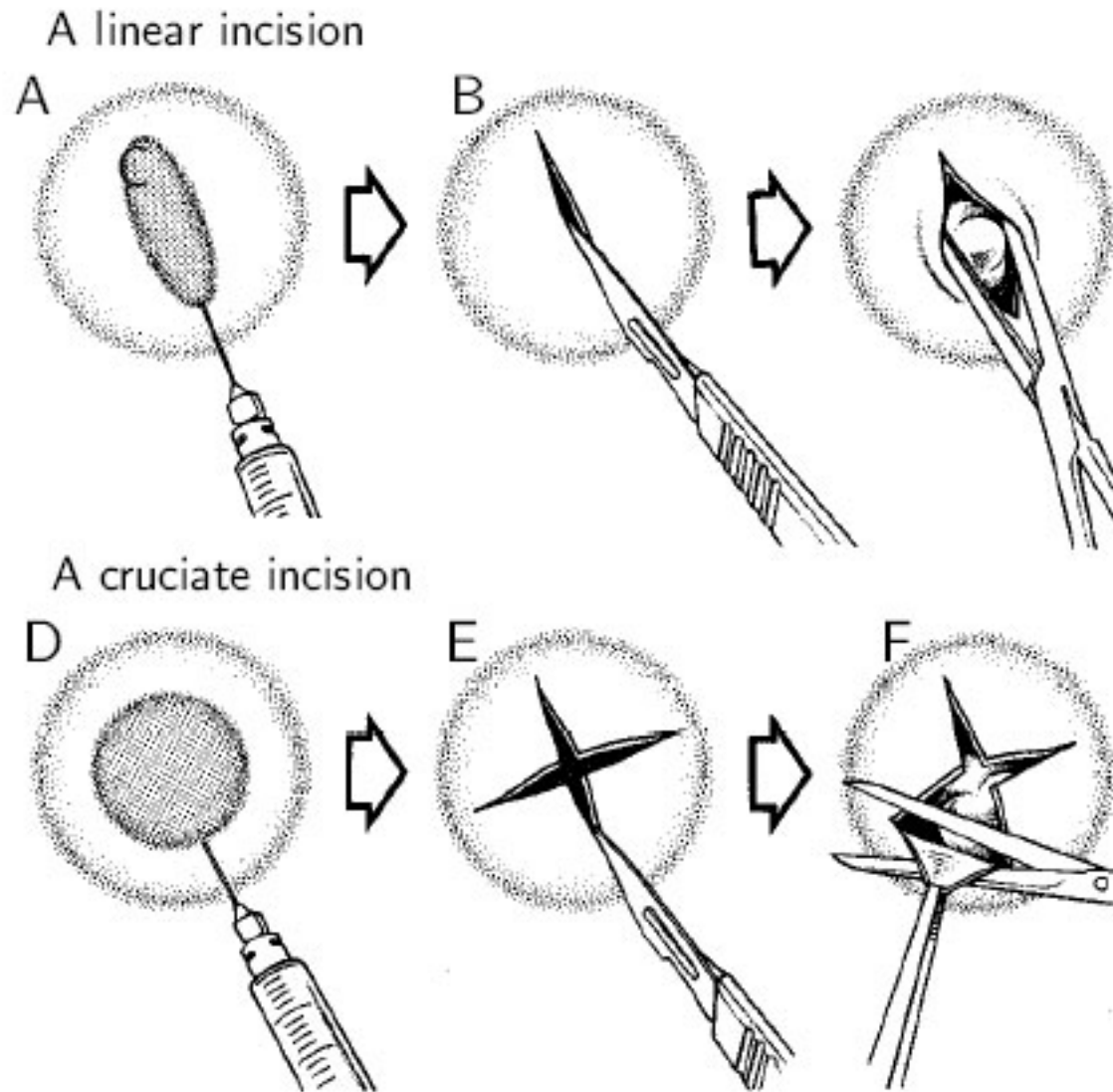


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Case #8

- 5 year old boy with eczema complains of gluteal muscle pain after falling on a fence >48 hours ago but has no wounds or deficits and is otherwise well per his parents.
 - On exam you notice...
 - ***Diagnosis/Disposition?***



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Source Undetermined



Source Undetermined

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Source Undetermined

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Bullous Impetigo

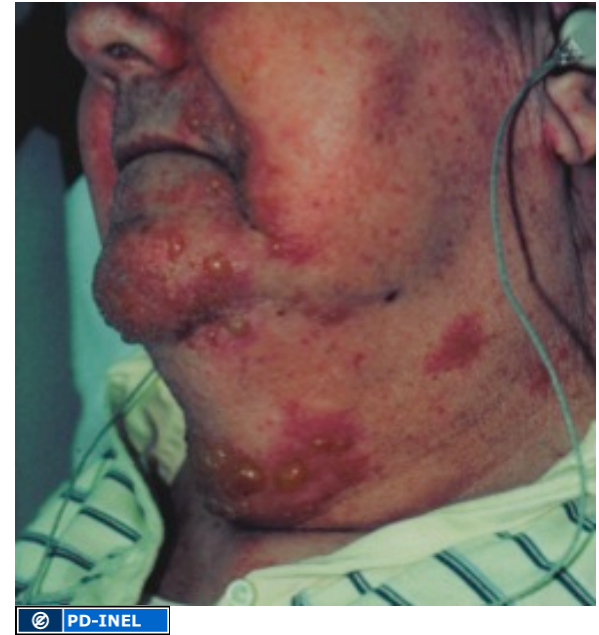
- Focal bacterial infection of the superficial skin
 - *Streptococcus pyogenes* (group A-hemolytic *Streptococcus*) and *Staphylococcus aureus*.
- *Most commonly affects children 2-5 years of age and usually involves the face and extremities.*
- *Lesions*
 - thin-walled, 1-2-cm bullae. When these rupture, they leave a thin serous crust and remnant of the blister roof at the rim of the crust.
 - The face, neck, and extremities are most often affected.
 - Gram's stain of the fluid from a bulla reveals gram-positive cocci. Cultures are positive in 95% of cases.
- *The lesions may spread rapidly by autoinoculation secondary to scratching and coalesce to form larger areas of infection.*
 - *e.g., Super-infection of eczema sites*

Management

- Good hand washing and personal hygiene
 - Emphasized to the patient and family
 - Highly contagious and spread by direct contact
- Treatment based on extent of lesions
 - Oral penicillinase-resistant semi-synthetic penicillin such as dicloxacillin, 250 mg four times a day for 5 to 7 days for adults, or erythromycin, 250 mg four times a day in adults or 30 to 50 mg/kg/day in children.
 - If the infection is limited to a small area, mupirocin 2% ointment three times a day may be applied.
 - Without treatment, impetigo heals within 3 to 6 weeks

Case #9

- 60 year old Greek male with Rheumatoid Arthritis and Hypertension presents with mouth blisters for several months.
 - Blisters have now extended to his neck/face over the last 4 days. Some of them have sheared with minimal pressure



Source Undetermined

– ***Diagnosis/Disposition?***

Pemphigus Vulgaris

- Bullous disease, affecting both sexes equally.
- Most common in patients 40 to 60 years old.
- Mostly prevalent in people of Jewish, Mediterranean, or South Asian descent.
- Mortality rate before the use of steroids was approximately 95%.
- Cause largely unknown
 - Some studies suggest autoimmune mechanism.
 - Associated in a few instances with Rx use
 - penicillamine , captopril

Pemphigus Vulgaris

- Mucous membrane lesions occur before skin lesions
 - 50-60% of patients have oral lesions that typically antedate the cutaneous lesions by several months.
 - Most common site is in the mouth, especially the gums and vermillion borders of the lips.
 - Oral lesions are bullous but commonly break, leaving painful, denuded areas of superficial ulceration
- Initially small, flaccid bullae that break easily, forming superficial erosions and crusted ulcerations.
- Any area of the body may be involved.
- **(+)Nikolsky's Sign**
 - Blisters may be extended or new bullae may be formed by firm tangential pressure of a finger on the intact epidermis

Management

- Pain control and local wound care are essential components of therapy.
- Once the diagnosis is made, treatment with oral glucocorticoids
 - initial doses: Prednisone (or equivalent) 100-300 mg qD
- Despite localization to the skin and mucous membranes, death the rule before steroid use
 - Related to *uncontrolled spread of the disease, secondary infection, dehydration and thromboembolism.*

Case #10

- A 6 year boy and his 15 year old Rugby-playing brother presents with several days of a painful raised rash. Their mother is concerned that they have a bacterial infection.

– ***Diagnosis/Disposition?***



Source Undetermined



Source Undetermined



Herpes Simplex Virus

- Usually localized in a non-dermatomal distribution.
- HSV-1 (≈“above the waist”)
 - Primarily affects non-genital sites
 - Mouth is the most common site of HSV-1 infections.
 - Children are affected more commonly than adults.
 - Small clusters of vesicles appear but are soon broken, leaving irregularly shaped, crusted erosions.
 - severity of gingivostomatitis varies from the presence of small ulcers to extensive ulceration of the mouth, tongue, and gums accompanied by fever and cervical lymphadenopathy. The infection may be so severe that oral fluid intake is difficult, and dehydration may result.
 - Healing typically occurs in 7 to 14 days, unless a secondary infection with streptococci or staphylococci occurs.

Non-Genital Herpes Simplex

- *Herpetic whitlow*
 - *Painful HSV infection of the distal finger characterized by edema, erythema, vesicles, pustules on erythematous base.*
 - *Fever, lymphangitis, and regional adenopathy*
- *Herpes gladiatorum*
 - *Spreads via direct skin-to-skin contact in sports such as rugby and wrestling.*
 - *May become latent, and tends to recur at the site of the primary lesion.*
 - *Cutaneous lesions of the primary infection may occur alone or with fever, lymphadenopathy, myalgia, pharyngitis, malaise, blepharitis, or keratoconjunctivitis.*

Progression

- Incubation may take 2-14 days.
- Symptoms
 - Stinging, burning, and pruritus at the inoculation site typically precede the eruptions.
- Diagnosis
 - Made on identification of the characteristic lesions most easily during the vesicular phase.
 - Later, crusted and dried vesicular fluid may be confused with impetigo.
 - Viral cultures taken from scraping the base of an unroofed vesicle may confirm the diagnosis.
 - Tzanck smear may provide more immediate confirmation.

Management

- Oral antivirals (Acyclovir, Famcyclovir) in addition to analgesics/antipyretics
 - To be most effective, antivirals must be started within 72 hours of the eruption and should be initiated if prodromal symptoms (burning, stinging) occur.
 - Topical antibiotic ointments help prevent secondary infection and speed healing.
 - *Do not I&D digital Whitlow Lesions*

Case #11

- 42 year old female with a known (+)retroviral screen and anti-retroviral Rx non-compliance, complains of several days of a burning over her left forehead. As her vitals are normal and there is no evidence of rash she is discharged home with clinic follow-up.

- One week later she returns and now has an painful facial rash.

– *Diagnosis/Disposition?*



Herpes Zoster



Mikael Häggström, [Wikimedia Commons](#)

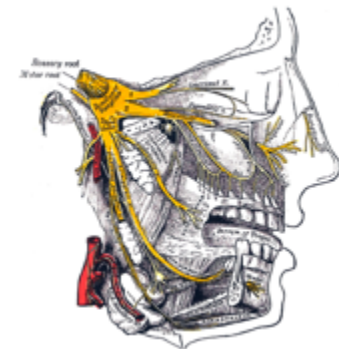
 PD-Self

- Varicella-Zoster Virus
 - Occurs exclusively in individuals who have previously had chickenpox.
 - Before the rash appears, the patient typically develops pain in a dermatomal distribution.
 - Pain precedes eruption by 1-10 days
 - Rash consists of grouped vesicles on an erythematous base involving one or more dermatomes.
 - Thorax = most cases
 - Trigeminal distribution next most



 PD-INEL

Source Undetermined



Lipothymia, [Wikimedia Commons](#)

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Progression

- Caused by a reactivation of latent varicella-zoster virus present since the initial infection with chickenpox.
 - During the latent period between the two illnesses, the virus is thought to reside in dorsal root ganglion cells.
- Very low mortality rate and rarely life threatening, even when dissemination to the visceral organs occurs.
 - Early systemic corticosteroid therapy may shorten the duration of post-herpetic neuralgia but does not lessen the severity of pain.
 - Antiviral Tx (acyclovir, famciclovir, vidarabine, foscarnet, valacyclovir, and interferon- α), has been shown to be effective for immunocompromised patients.
- Complications
 - CNS involvement, ocular infection, and neuralgia as well as rare meningoencephalitis, myelitis, and peripheral neuropathy

Herpes Zoster Ophthalmicus

- Ocular complications
 - Occur in 20-70% of cases involving the ophthalmic division of the trigeminal nerve.
 - Severity varies from mild conjunctivitis to panophthalmitis
 - Eye involvement produces anterior uveitis, secondary glaucoma, and corneal scarring.
- Hutchinson's Sign
 - Correlation between eye involvement and vesicles located at the tip/side of nose
 - Patch innervated by the infratrochlear and external nasal branches of the nasociliary nerve (also innervates cornea)
 - *The prognostic value of the Hutchinson sign has been validated in one study.*

Immunocompromise

- More severe in immunosuppressed patients, especially those with AIDS, Hodgkin's disease, or other lymphomas
- Cutaneous dissemination occurs more commonly in these patients than in the general population.
- Visceral and CNS dissemination is also more likely to occur in these patients.

Case #12

- 65 year old man with poorly-controlled DM and multiple prior pilonidal abscesses presents with 3 days of scrotal pain & swelling that started after he noticed a small, painful “spider bite” in the right gluteal fold.
- Transferred with Foley catheter from an outside hospital due to perineal involvement

– *Diagnosis/Disposition?*



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Source Undetermined

Necrotizing Fasciitis

- Usually occurs in men in the lower extremities and may evolve after only minimal local trauma.
- Type I
 - Polymicrobial and involves non- group A streptococci plus anaerobes.
 - Abdomen and perineum
- Type II
 - Group A beta-hemolytic streptococci
 - Extremities
 - Streptococci endotoxin → separation of the dermal connective tissue, resulting in continued inflammation and necrosis.
 - Streptococcal necrotizing fasciitis is frequently associated with streptococcal toxic shock syndrome.

Progression

- Early clinical findings similar most infected wounds
 - Involved site quickly becomes erythematous, tender, and edematous with formation of necrotic patches/ bullae (days 2-4) productive of sero-sanguinous fluid
 - Fever usually present
 - Deep structures and muscles are not involved.
 - Hypotension, tachycardia, leukocytosis, and hypocalcemia ensue with systemic toxicity out of proportion to the clinical findings
- X-ray → reveal gas in the tissues, but tissue gas is not a universal finding
 - MRI better, but due to rapidly progressive and frequently fatal outcome of this condition, delaying treatment to wait for imaging is not justified even when available immediately



kidney, ureter and bladder showed mottled air density over the pelvic region and perirectal area.

Management

- Surgical debridement along fascial planes
- Fluid and critical care resuscitation
- Parenteral antibiotics against *S. aureus*, *Streptococcus*, gram-negative organisms, and anaerobes
 - Eventually directed by Gram stain/culture

Fournier's Gangrene

- Insidious necrotizing subcutaneous infection of the perineum that occurs primarily in men usually 20-50 years of age
 - Usually involves the penis or scrotum.
- Pain or pruritus in the genitalia is followed by fever, chills, and impressive perineal swelling
- Inflammation may involve the entire abdomen, back, and thighs.
 - Frequently crepitus on palpation, indicating subcutaneous gas.
- Systemic Symptoms
 - Nausea and vomiting, changes in sensorium, and lethargy.
 - Eventually, gangrenous areas demarcate and become less painful as destruction and sloughing of sensory nerves occur. The tissue breaks open, sloughs, and gives off an overwhelming feculent odor.

- Most common causal factors
 - Infection or trauma to the perianal area
 - *including anal intercourse, abscess/scratches, chemical or thermal injury, and diabetes.*
 - Local trauma and perianal disease precede approximately one third of all cases.
 - Cultures demonstrate bacteria of the distal colon, with a complex picture of aerobic and anaerobic bacteria.
 - *B. fragilis* the predominant anaerobe
 - *E. coli* the predominant aerobe.

- Necrosis Mechanism
 - Bacterial invasion of the subcutaneous tissues → obliteration of the small branches of the pudendal arteries that supply the perineal or scrotal skin, resulting in acute dermal gangrene.
- This combination of erythema, edema, inflammation, and infection in a closed space stimulates anaerobic growth.
- Emergency Management
 - Antibiotic therapy against anaerobes and gram-negative enterics and wide incision and drainage of the area to remove all the necrotic tissue as soon as possible.
 - *Mortality rate is approximately 3-38%.*

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