



Chapter 110 – Dermatologic Presentations

Episode overview

1. List five broad categories of rashes
2. Describe the primary skin lesion types (table)
 - a. Bonus: What are the secondary skin lesions (show notes only)
3. List systemic diseases that present with cutaneous signs for each of the following locations (table):
 - a. Generalized rash
 - b. Head and neck
 - c. Hands
 - d. Legs
 - e. Palms and Soles
4. Describe the various presentations of tinea and their treatment
5. List 8 RFs for candida infections
6. Describe the stepwise management of diaper dermatitis (list 4)
7. Describe the distribution of Pityriasis rosea
8. Describe the management of atopic dermatitis
9. Describe the management of impetigo & folliculitis
10. List 6 RFs of C.A.-MRSA and 4 oral Abx treatments
11. Describe the presentation and management of:
 - a. Staph Scalded Skin
 - b. TSS
12. List 10 causes of EM / SJS / TEN
13. Describe presentation of EM + SJS/TEN. Differentiate between TEN and SJS
14. List 6 broad categorical causes of urticaria
15. Describe the typical features for each of the following:
 - a. Measles
 - b. Rubella
 - c. Roseola Infantum
 - d. Erythema Infectiosum
 - e. Scarlet Fever
16. Describe treatment of poison ivy
17. Describe presentation and treatment of Pediculosis + Scabies
18. List 10 causes of Erythema Nodosum
19. List a 6 ddx for vesicular lesions
20. List 4 lesions with a positive Nikolsky's sign
21. List 4 complications of HSV infection
22. List 5 complications of Varicella + describe the management of an exposure during pregnancy
23. List 5 complications of Zoster + differentiate between Ophthalmicus and Oticus
24. What is the treatment of herpes zoster?



Wisecracks

1. List 5 causes of desquamating lesion
 2. List 5 palm and sole rashes
 3. List 10 maculopapular rashes
 4. List 1 low, medium and high potency topical steroid
 5. Identify the following rashes: erythema migrans, erythema marginatum, erythema multiforme, erythema nodosum, meningococcemia
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Key concepts

- You have to be able to describe the lesion(s) to diagnosis and manage it
- Key steps: accurate history, physical examination, including lesions and distribution, and appropriate diagnostic tests.
- Incision and drainage may be adequate therapy for simple abscesses**.
- Antibiotics to cover MRSA are appropriate for most skin and soft tissue infections.
- Tinea capitis requires 4 to 8 weeks of systemic antifungal treatment.
- Onychomycosis requires long-term systemic treatment.
- Newer nonsedating antihistamines are a useful alternative to older sedating ones to control pruritus and histamine-mediated rashes while allowing the patient to remain active.
- Scabies infestations should be diagnosed clinically and treated expeditiously even without definitive proof of the infestation.
- Medication reactions are common and may results from any medication, typically within 4 to 21 days after taking the medication.
- Rashes that are associated with mucosal lesions, blisters, or desquamating skin are often caused by significant soft tissue infections, drug eruptions, or immune disorders.
- Patients with Stevens-Johnson syndrome (<10% TBSA) and toxic epidermal necrolysis require inpatient treatment, preferably in a burn unit.
- Cutaneous signs of systemic disease may include pruritus, urticaria, erythema multiforme, erythema nodosum, pyoderma gangrenosum, and others.
- Physicians should be familiar with one or two topical steroid preparations of low, medium, and high potency and their appropriate therapeutic use.
 - Hydrocortisone 0.1% lotion - low/mid
 - Hydrocortisone valerate 0.2% ointment - mid
 - Betamethasone dipropionate 0.05% cream - high (more potent than beta.val)
- Life-threatening conditions at risk for dehydration and infection require inpatient treatment - the rest should be managed as outpatients!

Rosen's In Perspective

So, you walk up to that chart and read the CC...."rash"...your heart sinks and all you remember is that maculopapular is the only piece of dermatology knowledge you have...

Let's build on some dermatology knowledge before going back to this case...



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So, your skin has three layers:

1. Epidermis
 - a. Keratinocytes
 - b. Melanocytes
 - c. Langerhans cells
2. Dermis
 - a. Connective tissue, fat, blood vessels, nerve endings, immune cells
3. Subcutaneous layers

Here's a better approach:

Make sure you ask about new foods, meds, soaps, pets, jewelry etc.

(See Box 110.1 in Rosen's for original diagram)

Approach to the management of the unknown rash

- 1) Time of onset
- 2) Historical features
- 3) Medical history
- 4) Primary lesion
- 5) Secondary lesion
- 6) Distribution of the lesions
- 7) Systemic illness
- 8) Diagnostic tests
- 9) Category of rash
 - a. Infectious
 - b. Immune
 - c. Vascular
 - d. Allergic
 - e. Malignancy
- 10) Treatment

Core questions

1) List five broad categories of rashes

- Infectious
- Allergic
- Autoimmune
- Vascular
- Malignancy-related



2) Describe the primary skin lesion types (table)

The primary skin lesions result directly from the disease process.

Primary Lesions (For original table, see Rosen's Table 110.1)

Lesion	Description	Size
Macule	Flat circumscribed pigmented area	<0.5cm in diameter
Patch	Flat circumscribed pigmentation area	>0.5cm in diameter
Papule	Elevated, solid, palpable lesion, variable colour	<0.5cm in diameter
Plaque	Elevated, solid, palpable lesion, variable colour	>0.5cm in diameter
Nodule	Solid, palpable, subcutaneous lesion	>0.5cm in diameter
Abscess	Erythematous, fluctuant, tender, fluid-filled nodule	Any
Tumour	Solid, palpable, subcutaneous lesion	>0.5cm in diameter
Vesicle	Elevated, thin walled, circumscribed, clear fluid-filled lesion	>0.5cm in diameter
Pustule	Elevated, circumscribed, purulent, fluid-filled lesion	Any
Bulla	Elevated, thin walled, circumscribed, fluid-filled lesion	>0.5cm in diameter
Petechiae	Flat, erythematous or violaceous non-blanching lesions	<0.5cm in diameter
Purpura	Erythematous or violaceous non-blanching lesions, may be palpable	>0.5cm in diameter

Secondary lesions result from secondary factors: scratching, healing, infections.

- a. Bonus: What are the secondary skin lesions (show notes only). See Table 110.2 in Rosen's 9th edition

Secondary lesion	Description
Scale	Thickened area of keratinised epithelium
Crust	Dried area of plasma proteins, resulting from inflammation
Fissure	Deep cracks in skin surfaces, extending into dermis
Erosion	Disruption of surface epithelium, usually linear, traumatic
Ulcer	Deep erosion extending into dermis
Scar	Dense collection of collagen, a result of healing after trauma or procedures
Excoriation	Linear erosions typically secondary to scratching or rubbing
Infections	Bacterial, viral, fungal or protozoal infection, caused by breaks in dermal-epidermal junction, often erythematous
Hyperpigmentation	Increase in melanin containing epidermal cells
Lichenification	Abnormally dense layer of keratinised epidermal cells



3) List systemic diseases that present with cutaneous signs for each of the following locations (table):

(See table 110.4 in Rosen's 9th edition)

- a. Generalized rash
- b. Head and neck
- c. Hands
- d. Legs
- e. Palms and Soles
 - i. Infectious
 1. Secondary syphilis
 2. Hand foot and mouth disease (coxsackie)
 3. RMSF
 4. Kawasaki syndrome
 5. Staph. Aureus endocarditis
 6. Measles
 7. Dengue
 8. Acute meningococcemia
 - a. Meningococcal disease may manifest as one of three syndromes: meningitis, bacteremia, or bacteremic pneumonia.
 - ii. Drug induced
 1. Chemotherapy drug: Acral erythema (e.g. due to doxorubicin)
 - a. Hand-foot skin reaction
 2. Erythema multiforme

4) Describe the various presentations of tinea and their treatment

Tinea refers to superficial dermatophytic infection of the skin, hair, and/or nails, usually by the Trichophyton organism.

Tinea Corporis	Tinea refers to superficial dermatophytic infection of the skin, hair, and/or nails , usually by the Trichophyton organism.	<p>Infections of the body, groin, and extremities usually respond to topical antifungal agents.</p> <p>A number of effective topical antifungal agents are available, including clotrimazole, haloprogin, miconazole, tolinaftate, terbinafine, naftifine, and others.</p> <p>Two or three daily applications of the cream form of any of these preparations result in healing of most superficial lesions in 1 to 3 weeks.</p>
Tinea Capitis	Tinea capitis is a fungal infection of the scalp. The most common	Treatment should be with a systemic antifungal agent, such as



	<p>organisms include <i>Microsporum</i> and <i>Trichophyton</i> species.</p> <p>Hair loss is the result of hyphae growing within the hair shaft, rendering it fragile, so that the hair strands break off 1 to 2 mm above the scalp.</p> <p>May develop into a kerion</p>	<p>terbinafine or griseofulvin. Therapy should be given for 4 to 6 weeks.</p> <p>The patient should be referred for outpatient follow-up with primary care within 4 weeks. Alternative therapy includes fluconazole or itraconazole for 4 to 6 weeks.</p>
Kerion	<p>A kerion is a fungal infection affecting hair follicles that is characterized by intense inflammation, and a boggy, erythematous mass, typically affecting the scalp...may contain frank pus.</p>	<p>Kerions are treated the same as tinea capitis, with systemic antifungal agents for 6 to 8 weeks. If bacterial superinfection exists, an antibiotic is added.</p> <p>Surgical drainage of kerions is not helpful and should be avoided.</p>
Tinea Pedis Tinea manuum (hands)	<p>commonly referred to as athlete's foot, presents with scaling, maceration, vesiculation, and fissuring between the toes and on the plantar surface of the foot.</p>	<p>topical antifungal agents, such as terbinafine twice daily for 2 to 4 weeks; miconazole cream, powder, or spray twice daily for 2 to 4 weeks; and clotrimazole cream, solution, or lotion twice daily for 2 to 4 weeks.</p>
Tinea Versicolor	<p>or pityriasis versicolor, is a superficial fungal infection caused by genus <i>Malassezia</i>. Superficial hypopigmented or hyperpigmented patches occur mainly on the chest and trunk but may extend to the head and limbs.</p>	<p>Tinea versicolor may be treated with topical antifungal agents, such as 2.5% selenium sulfide shampoo, imidazole creams, and ketoconazole cream or foam. Systemic therapy may be indicated, such as oral ketoconazole.</p>
Tinea Unguium (onychomycosis)	<p>Tinea unguium may be caused by dermatophytes, candida, or other fungal species. Paronychia or untreated tinea pedis may be predisposing factors. Onychomycosis presents with toenails or</p> <p>fingerails that are thickened, opaque, cracked, or destroyed.</p>	<p>Involve-ment of one or two nails may be treated with topical antifungal agents. More extensive infection requires systemic therapy with an antifungal agent, such as terbinafine or itraconazole.</p>

5) List 8 RFs for candida infections

Many conditions predispose to infection, including:

diabetes mellitus, HIV infection, pregnancy, obesity, smoking, malnutrition, malignancy, or treatment with corticosteroids, antibiotics, or



immunosuppressive agents.

6) Describe the stepwise management of diaper dermatitis (list 4)

“Contact dermatitis is an inflammatory reaction of the skin to a chemical, physical, or biologic agent, which acts as an irritant or allergic sensitizer.

The primary lesions of contact dermatitis are papules, vesicles, and bullae on an erythematous base. Streaky, linear, intensely pruritic lesions are characteristic. A pattern in the region in contact with the allergen is typical.” – Rosen’s 9th edition

Stepwise Ladder:

- Avoid the irritant or allergen (can be difficult because the reaction can be delayed!)
- Frequent diaper changes, lots of “air” time
- Barrier pastes/ointments: avoid any with fragrances/preservatives
 - Vaseline [white petrolatum], Desitin, Triple Paste, A&D Ointment, and Balmex
 - Sucralfate for recalcitrant cases
- Consider topical antifungal agents (e.g. clotrimazole)
- Consider topical breast milk
- Consider low-mid potency steroid cream
- *Oozing or vesicles should be treated with cool wet compresses of Domeboro or Burow’s solutions (aluminum acetate).*
- *Topical baths, available over the counter, may also be comforting.*
- *Systemic antihistamines, such as hydroxyzine and diphenhydramine, may help control pruritus; nonsedating antihistamines are preferred for use during the day.*
- *If present, secondary bacterial infection must also be treated.*

Know your ddx:

- Cutaneous candidiasis
- Contact dermatitis
- Atopic dermatitis
- Tinea cruris
- Intertrigo
- HSV
- Folliculitis

7) Describe treatment of poison ivy

“Allergic contact dermatitis is a form of delayed hypersensitivity mediated by lymphocytes sensitized by the contact of the allergen to the skin. The common Toxicodendron species, including poison ivy, oak, and sumac can produce a severe reaction.

Toxicodendron species often result in vesicular or bullous eruptions. Oozing, crusting, scaling, and fissuring may be found along with lichenification in chronic lesions. The



distribution of the eruption depends on the specific contact and may be localized, asymmetric linear, or unilateral.

Use treatment regimens for contact dermatitis, a course of systemic corticosteroids may be indicated to treat severe Toxicodendron-associated dermatitis.

Patients should be counseled to wash all clothes or items that might have contacted the plant because the irritant plant oil can persist.” – Rosen’s 9th edition

8) Describe the distribution of Pityriasis rosea

Pityriasis rosea is a mild skin eruption predominantly found in children and young adults. The etiology is unknown.

Clinical presentation includes multiple pink or pigmented oval papules or plaques 1 to 2 cm in diameter on the trunk and proximal extremities. A history may reveal an initial larger patch (“herald patch”) that precedes the widespread eruption.

The lesions are parallel to the ribs, forming a Christmas tree–like distribution on the trunk and extremities.

“In children, papular or vesicular variants of the disease may occur.

Pityriasis rosea is self-limited, resolving in 8 to 12 weeks. Recurrences are rare. Treatment should include supportive care, including alleviation of pruritus. Topical zinc oxide and calamine lotion are useful for pruritus. If the disease is severe or widespread (eg, vesicular PR), topical or oral steroids may be used. No restriction of activity or isolation is indicated.” – Rosen’s 9th edition

9) Describe the management of atopic dermatitis

“Atopic dermatitis is a common dermatologic condition often referred to as eczema or chronic dermatitis.

The course of atopic dermatitis involves remissions and exacerbations. More than 90% of patients have the onset of atopic dermatitis before 5 years old.

*Atopic dermatitis is an inflammatory skin condition. **Diagnostic criteria include itchy skin plus three or more of the following:***

- 1. **history of flexural involvement,***
- 2. **generalized dry skin in the past year,***
- 3. **history of asthma or hay fever,***
- 4. **onset of rash before 2 years old, and***
- 5. **flexural dermatitis.***



The itching may be focal or generalized, is worse during the winter, and is triggered by increased body temperature and emotional stress.

Treatment should be aimed at control of inflammation, dryness, and itching. **Management includes a careful review of daily skin care with patients or caregivers.**” – Rosen’s 9th edition

General recommendations for all patients include avoidance of nonspecific skin irritants, nonessential toiletries, and detergents.

- Skin dryness is treated with lubricating ointments such as Vaseline or 10% urea in Eucerin cream (not lotion).
 - Treatment of exudative areas includes the application of wet dressings, which are useful for their moisturizing, anti inflammatory, and antipruritic actions. Two or three layers of gauze soaked in Burow’s solution should be applied for 15 to 20 minutes four times a day for exudative lesions.
 - Antihistamines may be helpful in reducing the pruritus and are also useful for their sedative and soporific effects, although there is no convincing evidence that H1 antihistamines decrease itching in patients with atopic eczema.
- Topical corticosteroids are the cornerstone of therapy and are often best prescribed in ointment form. Approximately 80% of patients have improvement of symptoms with topical steroid treatment. When the dermatitis is severe, the application of a fluorinated corticosteroid ointment such as half-strength betamethasone valerate is recommended to affected areas three times a day.
 - Fluorinated corticosteroids should not be used on the face, because they can produce cutaneous atrophy.
- extremely severe disease may require systemic steroids. Ultraviolet B treatment is moderately effective. Cyclosporine and other immunosuppressant agents are being used with some promising benefit.

Inpatient admission is a consideration for those patients who have generalized erythema and exfoliation (erythroderma) or intractable itching in that skin breakdown and severe secondary bacterial or viral skin infections may occur.

10) Describe the management of impetigo & folliculitis

- Infection of the skin caused by staph. Aureus or beta-hemolytic strep. (More surrounding erythema)
 - MRSA may also be involved
- Usually in children
- Bullous impetigo is another common form: the toxin released from staphylococcus causing 1-2 cm bullae
 - Ddx: HSV, inflammatory fungal infection, contact dermatitis, pemphigus vulgaris.



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- Treatment: (usually a self limited dz in 3-6 wks.)
 - PO abx for severe or multiple lesions
 - Cephalexin
 - MRSA:
 - Septra
 - Doxy
 - Clindamycin
 - Bullous impetigo:
 - Erythromycin or azithromycin
 - Topical:
 - Mupirocin
- Complication: post-pyoderma glomerulonephritis

Folliculitis:

- Inflammation of the hair follicle (staph. Aureus)
 - But be aware of “hot tub folliculitis post-hot tub/swimming pool” due to pseudomonas
- Little pustule with a central hair.
- Ddx:
 - Acne, keratosis Pilaris, fungal infection, hidradenitis suppurativa
- Treatment:
 - Iodine or chlorhexidine wash daily for a few weeks
 - Warm compresses prn
 - If systemic or extensive may consider abx

Another side pearls on a string....:

- Cellulitis : on ultrasound shows as cobblestoning with fine areas of reticular hypoechoic stranding
- Abscess will appear as a cystic hypoechoic fluid-filled cavity.
 - Recent literature does support the addition of TMP-SMX to incision and drainage for mild abscesses.
- Carbuncle: large abscess in the thick inelastic skin on the **back of the neck, back, thighs.**
 - Produce severe pain and fever.
 - Treatment:
 - Rule out septicaemia
 - Incision and drainage - if fluctuant
 - Abx if septic or evidence of cellulitis

11) List 6 RFs of C.A.-MRSA and 4 oral antibiotics treatments

Note: you can't really clinically differentiate an MRSA lesion from an MSSA lesion...

Risk factors for MRSA (community acquired) :



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- Previous MRSA
- Close contact with a person known to be MRSA +ve
- Inmates or resident or guard of a correctional facility
- Sports team contact/involvement
- MSM
- Native/aboriginal communities
- IVDU
- Cosmetic body shaving

(From Rosen's and Uptodate)

Note that health care associated MRSA is a different beast! (Risk for that includes hospitalization, long term care, recent sx or hemodialysis, HIV infection, prior Abx use).

LIST in text:

- When to treat MRSA abscess with abx?
 - Cellulitis
 - Systemic illness
 - Comorbidities
 - Immunocompromise
 - Extremes of age
 - Abscess in an area difficult to drain
 - Septic phlebitis
 - Abscess in a difficult to treat/access location
 - Lack of response to I&D alone

PO abx:

- TMP-SMX (usually one double strength tab BID)
- Doxycycline
- Clindamycin
- Minocycline
- Linezolid (special use only! Very \$\$, risk of heme side effects)

12) Describe the presentation and management of:

a. Staphylococcal Scalded Skin Syndrome

- This is a dx in kids < 6 yrs.
- *caused by an infection with phage group 2 exotoxin-producing **staphylococci**.*
- *The illness begins with erythema and crusting around the mouth. The erythema then spreads down the body, followed by **bulla formation and desquamation**. **Mucous membranes are usually typically involved**. After desquamation occurs, the lesions dry up quickly, with clinical resolution in 3 to 7 days.*
- *Most group 2 toxin-producing organisms are penicillin resistant. Although most patients will recover without antibiotic treatment, IV therapy with nafcillin,*



cephalexin, or dicloxacillin is recommended.14 Clindamycin, vancomycin, or linezolid may be considered in cases of suspected MRSA.

b. TSS

- Formerly “tampon” disease. But in fact it is known to occur in men and children! It is a life threatening septic shock syndrome preceded by nonspecific infectious symptoms.
- *Toxic shock syndrome (TSS) is an acute febrile illness characterized by a diffuse desquamating erythroderma.* This is due to an exotoxin producing Staph. Aureus.
- *Clinical presentation may include high fever, hypotension, constitutional symptoms, multiorgan involvement, and rash.* May be associated with menstruation.
 - Associated settings: *can occur in any patient population!*
 - Associated with menstruation > 50%
 - Post op, post partum, post infectious (osteomyelitis/arthritis), sinusitis, PTA, abscess
 - TSS is caused by S. aureus or group A streptococcus, also called Streptococcus pyogenes.
- Diagnosis of TSS requires the presence of
 - (1) temperature of at least 38.9° C;
 - (2) hypotension, with a systolic blood pressure of 90 mm Hg or less;
 - (3) rash (diffuse, blanching, macular erythroderma → followed by full thickness desquamation)
 - (4) involvement of at least three organ systems. Systemic involvement may include the gastrointestinal tract, muscular system, or CNS and laboratory evidence of renal, hepatic, or hematologic dysfunction.

13) List 10 causes of EM / SJS / TEN

These are all examples of drug reactions. Many medications have the potential to produce a drug reaction. Patients at higher risk of drug reactions include those with immunodeficiency (elderly), certain infections, and genetic predisposition.

Drug reactions:

Benign	Serious!
morbilliform rash, urticaria, or fixed drug eruption, blistering dermatoses, erythema nodosum,	vasculitis, angioedema, anaphylaxis, Stevens-Johnson syndrome, toxic epidermal necrolysis, drug-induced lupus, lichenoid drug eruptions, psoriasiform drug eruptions, drug-induced neutrophilic dermatoses (ie, Sweet’s syndrome, erythema nodosum, and pyoderma



	gangrenosum), and cutaneous lymphoma-like drug reactions
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Some causes of EM / SJS / TEN

- **EM:**
 - drug reaction;
 - viral infections, especially hepatitis and influenza A;
 - fungal diseases, such as dermatophytosis, histoplasmosis, and coccidioidomycosis;
 - bacterial infections, especially streptococcal infections and tuberculosis.
 - Various collagen vascular disorders,
 - Pregnancy and various malignant neoplasms
 - unknown in approximately 50% of cases.
- **SJS/TENS**
 - Medications
 - sulfa drugs, nonsteroidal antiinflammatory drugs (NSAIDs), penicillin, aspirin, barbiturates, phenytoin, carbamazepine, or allopurinol.
 - Infection
 - Malignancy
 - Idiopathic (30-50% of CASES!)

14) Describe presentation of EM + SJS/TEN. Differentiate between TEN and SJS.

Most reactions occur within 4-21 days of taking a drug.

Stevens-Johnson syndrome and toxic epidermal necrolysis are considered a continuous spectrum of the same disease, an immune-complex–mediated hypersensitivity reaction. There is an SJS/TENS overlap when 10-30% BSA is involved.

From Rosen's 9th edition

<u><i>Erythema multiforme</i></u> “a central, dark papule or vesicle that is surrounded by a pale zone, a halo of erythema, and is commonly found on the hands or wrists.”	<u><i>Stevens-Johnson syndrome</i></u> is considered a minor form of toxic epidermal necrolysis with less than 10% body surface area (BSA) involved.	<u><i>Toxic epidermal necrolysis</i></u> Mortality may be up to 30%
Hypersensitivity reaction: causes: drug reaction; viral infections, especially hepatitis and influenza A; fungal diseases, such as dermatophytosis, histoplasmosis, and coccidioidomycosis; and bacterial infections, especially	Medications sulfa drugs, nonsteroidal antiinflammatory drugs (NSAIDs), penicillin, aspirin, barbiturates, phenytoin, carbamazepine, or allopurinol. Infection	Toxic epidermal necrolysis may be caused by medications, infection, malignancy, or idiopathic (30% to 50% of cases)



streptococcal infections and tuberculosis. Various collagen vascular disorders, Pregnancy and various malignant neoplasms have also been associated with erythema multiforme. The etiology is unknown in approximately 50% of cases.	Malignancy Idiopathic (30-50% of CASES!)	
Differential diagnosis includes urticaria, scalded skin syndrome, pemphigus, and pemphigoid and viral exanthems.		
acute, usually self-limited disease. It is characterized by skin lesions that are erythematous or violaceous macules, papules, vesicles, or bullae. Their distribution is often symmetric , most commonly involving the soles and palms, the backs of the hands or feet, and the extensor surfaces of the extremities. <u>The presence of lesions of the palms and soles is particularly characteristic. The target lesion with three zones of color is the hallmark of erythema multiforme</u>	commonly begins with prodromal symptoms, such as fever, malaise, rhinitis, sore throat, and myalgias. These are followed by the abrupt development of a macular rash that may appear as target lesions. The extremities are commonly involved, although any area may be affected. The exanthem becomes confluent, and dermal-epidermal dissociation ensues; Nikolsky's sign (denudation with shear stress) is present, and the skin is commonly painful to the touch. Mucous membrane involvement may occur with erythema, blistering, sloughing, or necrosis	Similar initial presentation to SJS but with RAPID systemic involvement The main feature of non-staphylococcal-induced toxic epidermal necrolysis, or Lyell's disease, is the separation of large sheets of epidermis from underlying dermis. The cornea may become involved and can lead to permanent blindness. Risk factors for poor prognosis include age older than 40 years old, underlying malignancy, heart rate more than 120, initial percentage of epidermal detachment more than 10%, BUN level more than 10 mmol/L, serum glucose level more than 14 mmol/L (or 252 mg/dL), and bicarbonate level less than 20 mmol/L.
Treatment begins with treatment of the underlying cause. Mild forms with no systemic symptoms, lesions limited to extremities, and no mucous membrane involvement typically resolve spontaneously in 2 or 3	The treatment of Stevens-Johnson syndrome and toxic epidermal necrolysis includes discontinuation of the offending agent and supportive care,	Morbidity and mortality are often related to infection and dehydration. High-dose IVIG may be administered to patients with severe toxic epidermal



<p>weeks.</p> <p>Patients with lesions on the trunk and patients who are immunocompromised, especially those with multiple lesions, require a course of systemic steroids for 14 to 21 days with a taper and urgent dermatology referral.</p> <p>Patients with mucous membrane involvement, systemic symptoms, or vesicle formation raise concern for Stevens-Johnson syndrome.</p>	<p>including hydration, prevention of secondary infection, and expert wound management (at a burn centre).</p>	<p>necrolysis or Stevens-Johnson syndrome. Plasmapheresis is considered in consultation with a specialist.</p>
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15) List 6 broad categorical causes of urticaria

This is a review from Episode 119!

Urticaria appears as papules or wheals that consist of central swelling with surrounding reflex erythema, and it is associated with itching or a burning type sensation → lasting less than 24 hours.

This can occur in isolation (mast cell degranulation) or due to systemic disease (histamine, bradykinin, etc).

- Drug induced (often nonimmunologic)
 - ASA
 - Penicillin
 - Narcotics
- Food related
- Skin irritants:
 - Foods, clothing fabrics, animal/plants, chemicals, cosmetics, cold/heat
 - Physical urticaria: Dermatographism
 - Cholinergic urticaria: induced by exercise, heat, or emotional stress. It may be associated with pruritus, nausea, abdominal pain, and headache.
- Inhalation irritants
 - Pollens, mold, dander, dust
- Stings/bites
- Systemic disease:
 - systemic lupus erythematosus, lymphoma, carcinoma, hyperthyroidism, rheumatic fever, and juvenile rheumatoid arthritis



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- Cold urticaria may also be associated with underlying illness, such as cryoglobulinemia, cryofibrinogenemia, syphilis, and connective tissue disease.
- Infectious
 - Rhinovirus, rotavirus, hepatitis, mono
 - Candida, dermatophytes, bacteria,

So to recap:

- Local irritants (from any route) - drug, chemical, temperature, pressure, bites
- drug eruption, exanthems, erythema multiforme, erythema marginatum, and juvenile rheumatoid arthritis, systemic infections

16) Describe the typical features for each of the following:

- a. Measles
- b. Rubella (german measles)
- c. Roseola Infantum
- d. Erythema Infectiosum

This is a great order to think through: because these first four are listed in order of decreasing severity/complication risk!

Roseola infantum "Sixth disease"	Erythema infectiosum "Fifth disease"	MEASLES	GERMAN MEASLES Aka: Rubella
Benign; spread by saliva Due to herpes virus 6 Mild	Parvovirus B19 Mild	Viral illness, droplet. Isolation impractical	Viral, droplet Should be isolated at home
fever and a skin eruption. Well children 6 months to 3 years old. May occur post febrile seizure The rash typically appears with defervescence. The lesions are discrete pink or rose-colored macules or maculopapules 2 or 3 mm in diameter that blanch on pressure and rarely coalesce	fever in 10% to 15% of patients, and a characteristic rash. Arthralgia and arthritis occur commonly in adults but rarely in children. The rash is intensely red on the face and gives a "slapped-cheek" appearance with circumoral pallor. A reticular maculopapular eruption, which may be noted on the arms, moves caudally to the trunk, buttocks, and thighs. The rash may recur with changes in	contagious from 5 days prior to onset of symptoms until 5 to 6 days after the onset of dermatologic involvement. Maculopapular erythematous lesions involve the forehead and upper neck and spread to involve the face, trunk, arms, and finally the legs and feet. Koplik spots begin to disappear coincident with the appearance of the rash. Complications may include otitis media, encephalitis, and pneumonitis. Otitis media is the most common	Fever, skin eruption, lymphadenopathy. Rash appears post 5 days of maximal infectivity. The major complications of rubella include encephalitis, arthritis, and thrombocytopenia. The most severe complication is fetal damage. No treatment is required in most cases of rubella. Antipyretics are usually adequate for the treatment of headache, arthralgias, and painful



	temperature and exposure to sunlight. The incubation period is usually between 4 and 14 days. The infection is benign and requires supportive care only.	complication. Encephalitis occurs in approximately 1 in 1000 cases of measles and carries 15% mortality. Measles pneumonia may also be life-threatening. Treatment: supportive, Vitamin A supplements	lymphadenopathy
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17) Scarlet Fever

- From group A strep. Infection.
- Sudden onset fever, chills, malaise, and sore throat, followed within 12 to 48 hours by a distinctive rash that begins on the chest and spreads rapidly, usually within 24 hours.
- skin has a rough sandpaper-like texture because of the multitude of pinhead-sized lesions. The pharynx is injected, and there may be erythematous lesions or petechiae on the palate. After the resolution of symptoms, desquamation of the involved areas occurs and is characteristic of the disease.
- Erythema marginatum may be seen in 10% of cases** and presents with annular erythematous lesions that may be transient and reappear over days, weeks, or months
- Complications include the development of a streptococcal infection of lymph nodes, tonsils, middle ear, and respiratory tract. Late complications include rheumatic fever and acute glomerulonephritis.
- Treatment is with oral penicillin VK or IM benzathine penicillin (given as Bicillin C-R). In patients allergic to penicillin, treatment may be initiated with erythromycin, other macrolides, or a cephalosporin.

18) Describe presentation and treatment of Pediculosis + Scabies

“Scabies, the human itch mite, from the Latin word *scabere*, to scratch, is a human skin infestation caused by the penetration of the obligate human parasitic mite *Sarcoptes scabiei*-*var hominis* into the epidermis.



The pruritus is typically worse at night. Clinical findings include small (<5 mm) papules or pustules and small raised or flattened burrows. In the ED, treatment should be instituted based on a clinical suspicion of the diagnosis of scabies.

First line treatment of scabies is topical permethrin 5% cream (whole body) and repeatFd in 1-2 weeks. Items that cannot be washed and or dry-cleaned can be decontaminated by sealing the items in an airtight container for at least 72 hours.

Pediculosis may affect the scalp hair (pediculosis capitis, caused by the mite *Pediculus humanus capitis*), body (pediculosis corporis, caused by *Pediculus humanus corporis*), or genitalia (pubic lice, caused by *Phthirus pubis*). Infestation is typically associated with significant pruritus.

The diagnosis of pediculosis is made by identification of lice or nits on the hair shaft

Therapy for pediculosis should be initiated with a pediculicide, such as permethrin 1% (Nix, Lyclear), which is effective in 90% of cases. Permethrin should be applied to the dry scalp and hair and remain for 10 minutes. Treatment should be repeated in 1 week to kill any newly hatched lice.

Spinosad 0.9% suspension (Natroba) is a new agent with demonstrated pediculicidal efficacy.

Hats, hair brushes and combs, and linens as well as clothing should be treated. Items should be boiled or washed and dried at high temperatures. Floors and furniture should be vacuumed.” – Rosen’s 9th edition

*“Bed bugs (*Cimex lectularius*) appear brown, approximately 5 to 6 mm in length.*

Bed bugs are found not only in linens, but on furniture, luggage, and in walls, baseboards, and buildings.

They often feed on humans at night with a painless bite. Clinical presentation may appear as erythematous welts, macules, papules, urticaria, purpura, vesicles, or bullae, with intense pruritus. The distribution is often over uncovered areas, such as arms, legs, and shoulders. Lesions resolve spontaneously in 1 to 2 weeks. Symptomatic treatment should be undertaken with antihistamines and topical corticosteroids.

A patient with a scaly, persistent pruritic eruption should be treated with permethrin 5% cream, ivermectin, or crotamiton.” – Rosen’s 9th edition

19) List 10 causes of Erythema Nodosum

“Erythema nodosum is an inflammatory reaction of the dermis and adipose tissue that presents with painful erythematous or violaceous subcutaneous nodules. These painful nodules occur most commonly over the anterior tibia but may also be seen on the arms or



body. Fever and arthralgia of the ankles and knees may precede the rash.” – Rosen’s 9th edition

Causes:

- Drug reaction
- Autoimmune
 - Sarcoidosis
 - ulcerative colitis
 - regional enteritis
- Infectious
 - Coccidioidomycosis
 - histoplasmosis, tuberculosis
 - infections with streptococci
 - Yersinia enterocolitica
 - Chlamydia
- Pregnancy
- Idiopathic

“Elevation of the legs, and wearing of elastic stockings reduce pain and edema. Aspirin in a dosage of 650 mg every 4 hours or other NSAIDs may also afford some relief. Erythema nodosum is a self-limited process that usually resolves in 3 to 8 weeks. Patients with severe pain may be treated with potassium iodide daily for 3 or 4 weeks.” – Rosen’s 9th edition

20) List a 6 ddx for vesicular lesions

- Herpes simplex virus
- Varicella zoster virus (either varicella form or zoster form)
- Contact dermatitis
- Allergic dermatitis
- Cutaneous candida
- Dermatitis herpetiformis

...cause is very dependent on location of the vesicles!

21) List 4 lesions with a positive Nikolsky’s sign

This sign is positive if with gentle rubbing the skin sloughs off (the top layer of the epidermis).

- SJS
- TENS
- Pemphigus Vulgaris
- staphylococcal scalded skin syndrome

.....and a subset of patients with bullous pemphigoid



22) List 4 complications of HSV infection

“HSV-1 primarily affects nongenital sites, whereas lesions caused by HSV-2 are found predominantly in the genital area and are typically transmitted primarily by sexual contact.

- Secondary HSV generalization (in patients with atopic dermatitis)
- Secondary bacterial infection
- Dehydration (gingivostomatitis - in children with HSV1)
- Cervical carcinoma (HSV 2 associated infections)
- Death (mucocutaneous herpes infection in immunocompromised patients (including neonates!) can become generalized and then disseminated to internal organs)

Recommended treatment for a first clinical episode of genital herpes is with acyclovir, famciclovir, or valacyclovir. These agents reduce the duration of viral shedding, accelerate healing, and shorten the duration of symptoms, but they do not prevent recurrent episodes. Immunocompromised patients may need IV therapy.” – Rosen’s 9th edition

23) List 5 complications of Varicella + describe the management of an exposure during pregnancy

Let’s walk down a little review here....”Varicella-zoster virus (VZV) infection causes two clinically distinct forms of disease: varicella (chickenpox) and herpes zoster (shingles). Varicella is generally a mild, self-limited illness in healthy children, but can occasionally lead to serious morbidity during pregnancy.” – Uptodate

“Varicella, or chickenpox, is an infection caused by the varicella-zoster virus. After an incubation period of 14 to 21 days, the illness begins with a low-grade fever, headache, and malaise. The exanthem coincides with these symptoms in children and follows them by 1 or 2 days in adults.

The vesicle of varicella is 2 or 3 mm in diameter and surrounded by an erythematous borderdrying of the vesicle begins centrally, producing umbilication. The dried scabs fall off in 5 to 20 days. The hallmark of varicella is the appearance of lesions in all stages of development in one region of the body. Extensive eruptions are often associated with a high and prolonged fever.” – Rosen’s 9th edition

Complications:

- encephalitis or meningitis,
- Pneumonia (Varicella pneumonia occurs more commonly in adults than in children)
- staphylococcal or streptococcal cellulitis,
- Thrombocytopenia
- arthritis,
- hepatitis,
- glomerulonephritis.



One way to think through this: essentially any organ system can get infected. Think head to toe: CNS, Lungs, Heart, GI organs, Heme, and then secondary superinfection.

Oral acyclovir may be effective if it can be started within 24 hours of development of rash for patients with chronic respiratory or skin disease. Usually, it is self-limited and treatment is symptomatic.

Oral acyclovir may be effective if it can be started within 24 hours of development of rash for patients with chronic respiratory or skin disease.

Exposure during pregnancy:

This isn't directly covered in Rosen's....so,

“Significant exposure to varicella infection, which is highly contagious, is defined as household contact, face to face contact with an index case for five minutes, or sharing the same hospital room with a contagious patient.

(Herpes zoster is much less contagious and usually requires close contact or exposure to open cutaneous lesions for transmission to occur: Treatment (if indicated) is oral acyclovir).

*Immunoprophylaxis for the prevention of maternal varicella infection — Passive immunization with VZV-specific antibodies reduces the risk of varicella infection and also attenuates the severity of infection in those who seroconvert. The US Advisory Committee on Immunization Practices recommends VariZIG, a varicella-zoster immune globulin preparation, in **all nonimmune pregnant women** who have been exposed to persons with VZV [should be given within 10 days of exposure - enough time to check whether the pregnant patient is immune]. Postexposure prophylaxis is not needed among women who were immunized with varicella vaccine in the past.” – Uptodate*

24) List 5 complications of Zoster + differentiate between Ophthalmicus and Oticus

“Herpes zoster, or “shingles,” is an infection caused by the varicella-zoster virus. It occurs exclusively in individuals who have previously had chickenpox. Peaks in those 50-70 yrs old.

Dermatomal pain may precede the eruption by 1 to 10 days and is variable in intensity; it may be described as sharp, dull, or burning in quality. The rash consists of grouped vesicles on an erythematous base involving one or several dermatomes. The thorax is involved in most cases, and the trigeminal distribution is the next most commonly involved region.



Herpes zoster has a very low mortality rate and is rarely life-threatening, except when dissemination to the visceral organs occurs [be worried about immunocompromised patients - AIDS, hodgkin's disease, lymphomas].

Complications include CNS involvement, abdominal organ involvement, ocular infection, and neuralgia. Meningoencephalitis, myelitis, and peripheral neuropathy have been reported. – Rosen's 9th edition

Ophthalmicus	Oticus
<p><i>Ocular complications occur in 20% to 70% of cases involving the ophthalmic division of the trigeminal nerve.</i></p> <p><i>The severity varies from mild conjunctivitis to panophthalmitis, which threatens the eye. Corneal dendritic lesions may be visible on fluorescein examination. Eye involvement may produce anterior uveitis, secondary glaucoma, and corneal scarring. There is a close correlation between eye involvement and vesicles located at the tip of the nose (Hutchinson's sign).</i></p>	<p>Ramsay Hunt syndrome: (Herpes zoster oticus) — The major otologic complication of VZV reactivation is the Ramsay Hunt syndrome, which typically includes the triad of ipsilateral facial paralysis, ear pain, and vesicles in the auditory canal and auricle.</p> <p>The facial paralysis seen in Ramsay Hunt syndrome is felt generally to be more severe than Bell's palsy attributed to HSV, with increased rates of late neural denervation and a decreased probability of complete recovery (UTD)</p>

25) What is the treatment of herpes zoster?

Antiviral medications are indicated, especially within <72 hours of onset of rash, to decrease the duration of symptoms and associated pain. Antiviral therapy may be initiated with acyclovir, famciclovir, or valacyclovir. [Uptodate recommends continuing administration of antiviral therapy as long as new lesions are appearing].

Doses to know:

- **Valacyclovir:** 1000 mg three times daily for seven days
- **Famciclovir:** 500 mg three times daily for seven days
- **Acyclovir:** 800 mg five times daily for seven days (this is the GO-TO drug for PREGNANT women with herpes zoster)

Supportive care is important for pain and pruritus control.

IV administration of acyclovir may be of some benefit in the treatment of severe ocular herpes zoster.

Treatment includes mydriasis and the application of topical corticosteroids. **Unlike the situation with herpes simplex conjunctivitis, eye involvement caused by herpes zoster does not appear to be exacerbated by corticosteroids.**



Postherpetic neuralgia may occur in 15% of patients and is more commonly in the elderly. Treatments may include opioids, amitriptyline, topical capsaicin, topical lidocaine, topical or oral gabapentin.

The varicella vaccine has been shown to boost immunity against herpes zoster virus (shingles) and is recommended for patients 60 years old and older. It reduces the occurrence of shingles and also slightly reduces pain compared with no vaccination in those who ultimately develop shingles.

Wisecracks

1. List 5 causes of desquamating lesion

- Kawasaki's disease
- Scarlet fever
- Staphylococcus scaled skin syndrome\
- Toxic Shock Syndrome
- TENS/SJS

2. List 5 palm and sole rashes

a. Palms and Soles

i. Infectious

1. Secondary syphilis
2. Hand foot and mouth disease (coxsackie)
3. RMSF - rocky mountain spotted fever
 - a. Give doxy!
4. Kawasaki syndrome
5. Staph. Aureus endocarditis
6. Measles
7. Dengue
8. Acute meningococcemia
9. crusted scabies

ii. Drug induced

1. Chemotherapy drug: Acral erythema (e.g. due to doxorubicin)
 - a. Hand-foot skin reaction
2. Erythema multiforme

3. List 10 maculopapular rashes

- Viral
 - Erythema infectiosum
 - Roseola infantum
 - Rubella
 - Measles
- Bacterial
 - Almost any bacterial infection!

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- *Bartonella henselae* (cat scratch disease)
- *Bartonella quintana* (trench fever)
- *Borrelia burgdorferi* (Lyme disease)
- *Neisseria gonorrhoeae* (gonorrhea)
- *Neisseria meningitidis* (meningococemia)
- *Pseudomonas aeruginosa*
- *Rickettsia akari* (rickettsialpox)
- *Rickettsia prowazekii* (epidemic/louse-borne typhus)
- *Rickettsia rickettsii* (RMSF-early lesions)
- Drug related
 - Drug induced exanthem
 - EBV + amoxicillin
 - CMV + antibiotics
 - HHV 6 + anticonvulsants
- Idiopathic
 - Pityriasis rosea

In the pediatric population, 72% of cases of fever and rash are caused by viruses, and 20% are caused by bacteria. The classic viral exanthems are rubeola (measles), rubella (German measles), herpesvirus 6 (roseola), parvovirus B19 (erythema infectiosum or fifth disease), and the enteroviruses (echovirus and coxsackievirus).

Viral exanthem / illness table

Roseola infantum “Sixth disease”	Erythema infectiosum “Fifth disease”	MEASLES	GERMAN MEASLES Aka: Rubella
Benign; spread by saliva Due to herpes virus 6 Mild	Parvovirus B19 Mild	Viral illness, droplet. Isolation impractical	Viral, droplet Should be isolated at home
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4. List 1 low, medium and high potency topical steroid

“Topical steroids have several mechanisms of action, including antiinflammatory effects, antiproliferative effects on fibroblasts and collagen, reduction of leukocyte adhesion to capillaries, reduction of capillary wall permeability, reduction of complement components, and histamine antagonism.

Adverse effects may include skin atrophy, striae, acneiform lesions, pigment changes, telangiectasia, hypothalamic-pituitary axis suppression from systemic absorption, and exacerbation of certain conditions, such as fungal infections and viral infections.” – Rosen’s 9th edition

- Hydrocortisone 0.1% lotion - low/mid
- Hydrocortisone valerate 0.2% ointment - mid
- Betamethasone dipropionate 0.05% cream - high (more potent than beta.val)

SELECTED FEVER AND RASH EMERGENCIES

- Meningococcal infection
- Bacterial endocarditis
- Rocky Mountain spotted fever
- Necrotizing fasciitis
- Toxic shock syndrome
- Miliary tuberculosis

5. Identify the following rashes:

Please see the Wikipedia links for more information

- **Erythema Migrans**

- Source: [Here](#)
- Target Lesion from Stage 1 of lyme disease.
- Causative agent: *Borrelia* infections



- **Erythema Marginatum**

- Source: [Here](#)
- Erythematous rings seen w/ Acute **rheumatic fever** secondary to group A strep or sometimes with **hereditary angioedema** (bradykinin based)



- **Erythema Multiforme**

- Source: [Here](#)
- Old school definition minor (not life threatening) or major (can be life threatening)
- Associated with post infection or drug administration
- Thought to be IgM bound complex deposition in vasculature, causing badness.
- Now thought to be on spectrum with SJS and TEN
- Ie EM or SJS < 10% TBSA then grey zone of SJS / TEN 10-30% then TEN >30%
- Conflicting definitions exist

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- **Erythema Nodosum**

- Source: [here](#)
- Inflammatory conditions of the subcutaneous fat tissue
- Typical age group 12-20
- Many causes! Eg: Strep infection, Drugs, Pregnancy, Tuberculosis, Sarcoidosis, IBD



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- **Meningococemia (This is BAD!)**

- Source: [here](#)
- *Neisseria meningitidis* very very bad



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