Rash of the Day: A Derm/ID Intermezzo

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Introductory remarks

• No conflicts of interests for this presentation
39 F with 3 month h/o weight loss, fevers over the past 2 months, has received antibiotics, has murmur but negative blood cultures.
Most likely diagnosis?

1. Cholesterol emboli 0%
2. HACEK organism leading to IE 0%
3. IE with partially treated organism 0%
4. Something else entirely 0%

✓
Sub-acute bacterial endocarditis

→ Septic embolization from the source

**Osler nodes**
- Painful, erythematous, fingertips

...splinter hemorrhages, conjunctival petechiae, Roth spots

**Janeway lesions:**
- Nontender hemorrhagic macules and papules on palms
THANK YOU MARIO!

BUT OUR PRINCESS IS IN ANOTHER CASTLE!
Raynaud’s Phenomenon

• Intermittent arteriolar vasospasm digits

• Primary: no ischemic injury or associated dz
  – Most common healthy adolescent/young women
  – Numbness, digital pallor, cyanosis, sharp bordered redness

• Secondary: vasospasm can = ischemic injury, underlying dz
  – Scleroderma, ~20% SLE, RA, cryoglobulinemia, Buerger’s, etc.
  – Asymmetrical, pain with ulcers and necrosis, dilated nailfold capillaries
CREST

- **Calcinosi**s cutis: hard, white skin nodules hands or elbows (rare)
- **Raynaud** phenomena: often 1st symptom to develop
- **Esophageal** dysfunction: common
- **Sclerodactyly**: often with ulcerations and/or scarring fingertips
- **Telangiectasia**: face, fingers, palms and mucous membranes

- ↑ risk: Choctaw Native Americans, Japanese descent
  - Also: pulmonary HTN, autoimmune hepatitis, biliary cirrhosis, and sicca syndrome. Anti-Scl-70 present in 30%
42 M chronic MJ user, repeatedly admitted with episodes of nausea and vomiting found to have this rash on exam.
What’s the underlying diagnosis?

1. Livedo reticularis    0%
2. Porphyria          0%
3. Erythema ab igne ✓ 0%
4. Cutis marmorata    0%
Cannabinoid hyperemesis

- Reported in 2004 Allen et al, in South Australia
  - Long term marijuana users
  - Intractable cyclical vomiting unresponsive to antiemetics and abd pain
  - Learned compulsive bathing behavior

- Nausea/vomiting: type 1 cannabinoid receptors GI nerve plexus have inhibitory effect GI motility (?)

- Compulsive bathing: thermoregulatory role (hypothalamic) endocannabinoids may be responsible (?)

2. J Am Board Fam Med 2010;23:790-793
Erythema ab igne or “redness from fire”
Erythema ab igne\textsuperscript{1-3}

- Repeated exposures to moderate heat
  - Mild localized erythema $\rightarrow$ Reticulate erythema $\rightarrow$
    Hyperpigmentation $\rightarrow$ Telangiectasias $\rightarrow$ Scaling $\rightarrow$ Atrophy

- Historically (incidence $\downarrow$ as central heating use $\uparrow$)
  - Shins those who worked by open fires or coal stoves.
  - Back/Abd if Tx chronic pain w hot H2O bottles or heating pads.
Dermite des chaufferettes – ‘heating unit dermatitis’

• Also called “thermal keratoses” due to histologic similarity to actinic keratosis.
  – Suggests heat can induce clonal mutation same as UV light does.

• Rare cases of squamous cell CA and Merkel cell CA have been reported.
  – Thermally induced cancers usually SCCs, can occurs even after 30 years!

• Tx → Interrupt contact with heat source!
EAI vs Livedo reticularis¹-³

• Non-blanchable
• Fixed
• More brownish

37 F admitted with history of polysubstance abuse has neutropenia and painful, necrotic rash – Diagnosis?
Why the rash?

1. IVDU $\rightarrow$ HCV $\rightarrow$ Cryoglobulinemia

2. Cocaine use (Risk ESRD $\uparrow$ x 3$^1$) $\rightarrow$
   ESRD $\rightarrow$ calciphylaxis

3. Warfarin skin necrosis

4. Cocaine $\rightarrow$ levamisole toxicity $\rightarrow$
   retiform purpura

0%

1
Levamisole retrospective...

- **Levamisole** – veterinary antihelminthic discovered 1960s
  - Immunomodulatory effects led to use in nephritic syndrome, colon cancer and rheumatoid arthritis
  - Withdrawn US market 1999
  - Recent use as cutting agent in cocaine preparation; detected 69% cocaine seized US border control in July 2009.

- **Role in cocaine uncertain**
  - Cutting (adding volume), enhancing effect, tracer for distribution?
  - Adverse effects levamisole in cocaine 1st reported 2008, Northern Alberta, Canada.

1. Clinical pharmacology and therapeutics, vol 88:3; September 2010
2. Annals of Internal Medicine, 1 June 2010, vol 152:11
3. Arch Dermatol/vol 146:11, November 2010
Levamisole

• Agranulocytosis
  – Mechanism unknown, was 1\textsuperscript{st} seen 1977 (levamisole for RA)
  – Agranulocytosis was seen in 2.5-13\% of folks using levamisole clinically
  – More commonly associated HLA-B27 genotype; P-ANCA (+)
  – Severe neutropenia (lowest ANC <100 cells/mm\textsuperscript{3}); plus fever, rash, malaise or sore throat

• Retiform purpura
  – 1\textsuperscript{st} reported in 1978 (levamisole treated RA), earlobe vasculitis in children treated for nephritic syndrome
  – Purpuric lesions preferentially involving earlobes,
  – Histology: vasculopathic pattern range from leukocytoclastic and thrombotic vasculitis to vascular occlusive disease sans true vasculitis
Levamisole and cocaine

- Red macules/papules → Purpura → Bullae → Necrosis → Eschars

- Suspect: Retiform purpura (ears, etc.) + neutropenia + UDS cocaine
Late retiform purpura

- Crusting eschars
• 42M with chronic lymphedema (OSA/pulmonary HTN) has slowly resolving cellulitis.

• Vancomycin changed to piperacillin/tazobactam

• Now, rapidly spreading erythema, ↑ WBCs, and fever (38.5F).
Patient greets you with a handshake and a smile – what’s going on?

1. Acute generalized exanthematous pustulosis (AGEP) 0%
2. Generalized pustular psoriasis (Von Zumbusch) 0%
3. Necrotizing fasciitis 0%
4. Staphylococcal toxic shock syndrome 0%
Acute generalized exanthematous pustulosis (AGEP)

- Eruption commonly related to drugs (90%).
  - Antibiotics, diltiazem, nifedipine, etc.

- 80% related to antibiotics (penicillins, macrolides 62% in 1 series); listed as delayed reaction to β-lactams.
  - Post mercury ingestion
  - Post viral illness (enterovirus, adenovirus, EBV, CMV, HBV)

- Drug specific T cells (and keratinocytes) secrete large amounts IL-8 (explains neutrophilia).
AGEP

- Acute onset 1-2 days after drug exposure.
- Pustules (> 100) on erythematous base, begin face or intertriginous areas.
- Fever > 38 C and neutrophilia.
- May have: edema face, target lesions, purpura, vesicles, erosions mouth and tongue.
Diagnosis AGEP

Skin Biopsy
→ Here spongiform pustules with neutrophils in superficial epidermis.

- Patch tests and lymphocyte transformation tests (LTT; in vitro host T cells) test for T cell immunity vs drugs.

- LTT sensitivity 74% (skin tests 62%), specificity 85%.

AGEP → 72 hours later.

- Resolves with skin desquamation 4-10 days after stopping drug.
43 F with T 101F; symmetric, diffuse rash; nausea, vomiting, leukocytosis, and ↑ AST/ALT, 4 wks after stent placement.
What is the most likely scenario?

1. New sexual partner, secondary syphilis 0%
2. Recent tick bite, rocky mountain spotted fever (R. rickettsii) 0%
3. Clopidogrel after new stent, now HSS/DRESS 0%
4. Meningococcemia and purpura fulminans 0%
Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS)

- Nomenclature in evolution (anticonvulsants, pseudolymphoma)
  - Literature search 2010 still shows use various terms: AHS, DIHS, HSS, DRESS and DIDMOHS
  - Most recently HSS/DRESS (Hypersensitivity syndrome/DRESS)

- Broad clinical spectrum, long latency (1-8 wks after drug start)
  - Fever, rash (> 80%), LAD, ↑ WBCs (↑ eos 60%-70%), ↑ LFTs
  - Rash: urticated or erythematous maculopapular eruption starts face, spreads downward symmetrically. LAD.
  - Facial edema often mistaken for angioedema is typical
  - Internal organ issues: liver, hematologic or renal most commonly affected

DRESS triad = Fever + Rash + Internal organ involvement

• Mortality ~ 10%, most from liver failure
• Current Tx with corticosteroids

Most common drugs include:
• Anti-convulsants (phenytoin, carbamazepine, phenobarbital, lamotrigine)
• Antibiotics (sulfonamides, minocycline, metronidazole)
• Abacavir; allopurinol; clopidogrel; ticlopidine...
Selfies

- Selfie: a self-portrait photograph, typically taken with a hand-held digital camera or camera phone.

- First ever selfie, a daguerreotype by Robert Cornelius 1839

- Added to the Oxford English Dictionary in November 2013
“My son and his college friends took a hike outside St Louis over the weekend, and one of the young men has developed an impressive "rash" on his bilateral thighs, began 2 days post the hike trip, non-pruritic, associated with myalgias”
Other data:
Sore throat last week; No headache, no fever and VSS
Does not recall tick bites; Others on hike wore shorts too, no skin lesions

What is the diagnosis?

1. Contact dermatitis 0%
2. Rocky mountain spotted fever 0%
3. Henoch-Schonlein purpura 0%
4. Meningococcemia 0%
5. Arcanobacterium haemolyticum 0%
Henoch-Schonlein Purpura

• Clinical features/epidemiology
  – IgA mediated small vessel venulitis, majority children, may affect adults
  – May follow: vaccinations, drug reaction, infection (URI, pharynx, otitis)

• Presentation
  – Fever, palpable purpura extremities and buttocks, may be urticarial early
  – Others: proteinuria/hematuria, arthralgia, abdominal pain, GI bleeding, intussusception, orchitis

• Prognosis/Tx
  – Most excellent prognosis, older children/adults ↑ nephritis
  – Systemic steroids, immunosuppressive agents (azathioprine, etc.)
Henoch-Schonlein Purpura

• Syndrome typically involves children, adults may be affected
  – Triad: abdominal pain, arthralgia, palpable purpura (LCV) caused by circulating IgA immune complexes
  – Present often as palpable purpura on lower limbs, thighs, buttocks
  – Other skin lesions: targetoid, SC nodules, itchy urticaria, LE edema

• Often follow infections and drug therapy
  – Infections: Strep throat, others [bacteria (S. aureus, H. pylori), virus (hepatitis, HIV), parasites (T. canis, amebiasis)]
  – Drugs: PCNs, macrolides, cocaine, etc.
Parent is concerned as mass develops in 13 year old female. Soccer player, denies injury. She grabs cellphone and sends a selfie...

1. Acral lentiginous melanoma 0%
2. Pyogenic granuloma 0%
3. Squamous cell carcinoma 0%
4. Glomus tumor 0%
5. Melanonychia 0%
Pyogenic granuloma

• Rapidly growing, benign vascular growths
  – **Misnomer** as neither pyogenic, nor granulomatous!
  – Presentation: solitary red papule/nodule (skin, face, gingiva), friable and ulcerates easily, grows fast (weeks)

• Associations
  – Medications: retinoids, PIs, some chemotherapeutic drugs
  – Pregnancy: 2\textsuperscript{nd} or 3\textsuperscript{rd} trimester, gingival lesions (granuloma gravidarum)
  – Trauma: at its site (physical, laser Tx port-wine stain, after removal)

• Treatment:
  – Removal, recurrences common; eliminate inciting factor; give birth!
Pyogenic granuloma – patient on ART
Glomus tumor

- Rare, small, benign hamartomas with vascular and smooth muscle elements
- Solitary form: most common in adults and adolescents, UEs, especially nail beds
- Multiple form: inherited, Aut Dom, from few to hundreds, usually in legs, children
- Discrete blue-red papules-nodules, 1 to several CMs in size, tender/painful especially in cold. Firm, tender blue papules, may also be found beneath nail plate
Longitudinal melanonychia

Subungual melanoma

Pigment

Pigmentary lines are lighter

Diffuse discoloration

Distribution

Constant diameter

Wider proximal, thinner distal

Nails affected

Multiple

Single

Acral lentiginous melanomas: < 5% melanoma subtypes; but occurs in all races and is the most common subtype in darker complected individuals.
THE POPE NOW POSES FOR SELFIES TOO:

On August 28th, 2013, a teenager on a pilgrimage took a selfie with Pope Francis, and it was officially deemed the first "Papal Selfie."
53 F with chronic LE edema due to venous insufficiency develops RLE cellulitis. Treatment with TMP/SMZ started, now AKI, LGIB and...

<table>
<thead>
<tr>
<th></th>
<th>1. Necrotizing fasciitis</th>
<th>0%</th>
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<tbody>
<tr>
<td></td>
<td>2. Leukocytoclastic vasculitis</td>
<td>0%</td>
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<tr>
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<td>3. Staphylococcal toxic shock syndrome</td>
<td>0%</td>
</tr>
<tr>
<td></td>
<td>4. Erythema multiforme</td>
<td>0%</td>
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</table>
Leukocytoclastic vasculitis (LCV)

- Commonest form of vasculitis
  - Most commonly associated drugs and infections, 40% cause unknown
  - May be limited to skin, but systemic manifestations 15-50% (joints, kidneys, GI tract)

- Skin manifestations polymorphic – palpable purpura #1
  - Urticarial, vesicular or bullous, ulceroinfarctive, nodular, pustular, etc.
  - Affects: LE (#1), buttocks, UE, trunk, face (especially in very sick pts)
  - Frequently accompanied edema lower legs, single vs recurrent
Leukocytoclastic vasculitis (LCV)

• Not a disease entity but a vascular reaction pattern
  – Due to circulating immune complexes ($\geq 81\%$) from myriad causes
  – LCV often co-exists with other vasculitis. Adequate biopsy includes deep dermis and SC tissue containing large vessels

<table>
<thead>
<tr>
<th>Category</th>
<th>Cause</th>
</tr>
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<tbody>
<tr>
<td>Infection</td>
<td>Bacterial (Strep, Mycoplasma, TB, leprosy), viral (HCV, HIV, CMV)</td>
</tr>
<tr>
<td>Drugs</td>
<td>Antibiotics (Sulfonamides, PCNs), NSAIDs, others</td>
</tr>
<tr>
<td>Associated Dz</td>
<td>Paraneoplastic (LK, lymphoma), CTD (SLE, RA), vasculitis (Churg-Strauss, Wegener’s, cryoglobulinemia), sarcoidosis, etc.</td>
</tr>
<tr>
<td>Others</td>
<td>Chemicals (insecticides), foreign proteins (serum sickness), in 40% no underlying cause identified</td>
</tr>
</tbody>
</table>
Vasculitis

• Diagnosis – biopsy?
  – Most histological forms are not specific; biopsy within 48H lesions best
  – Specific Dx requires clinical, histological and serological correlation

• What can your pathologist do for you?
  – Confirm presence of vasculitis
  – Describe nature inflammatory infiltrate, type/size of involved vessels

<table>
<thead>
<tr>
<th>Size</th>
<th>Sample vessels</th>
<th>Sample diseases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Large</td>
<td>Aorta + largest branches</td>
<td>GCA, Takayasu</td>
</tr>
<tr>
<td>Medium</td>
<td>Main visceral (renal, coronary, etc.)</td>
<td>PAN, Kawasaki</td>
</tr>
<tr>
<td>Small</td>
<td>Venules, capillaries, arterioles</td>
<td>HSP, cryoglobulinemia, MPA</td>
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</tbody>
</table>
21 M heterosexual, very worried about new onset penile lesions. History of unprotected sex with new partner.
What would you suspect?

1. Genital herpes 0%
2. Latex allergy (condom) 0%
3. Syphilis 0%
4. Psoriasis
5. Reactive arthritis 0%
These aren't the droids you're looking for.
The answer was elsewhere...
Genital Psoriasis

• Can cause significant distress, often confused with STI
  – 2-5% psoriasis sole presentation is genital
  – Clinical diagnosis, look for other findings (nails, joints, etc.)
  – Classic, inverse and generalized pustular psoriasis may affect genitals

• Koebner phenomenon may cause flare of genital lesions after intercourse, contact urine/feces or tight clothes.

• Differential includes: balanitis, dermatophyte infections, Reiter’s syndrome (circinate balanitis).

Psoriatic involvement penis

• Penile psoriasis – Often no scaling due to maceration
  – Thin, pale, erythematous plaques with light scale on penis (glans or under foreskin). Not associated with itching or burning.
  – Ask for family history of psoriasis, recent new medications (BB).

• Inverse psoriasis – axillary, groin, intergluteal cleft
  – Bright red plaques with little scale, lack sharp borders and may itch.
  – Psoriatic scale not as prominent on male genitalia due to moisture and maceration. Lesions aggravated by trauma, localized fungal infections.
26 M with AIDS and skin lesion with pearly borders with visible vasculature developing over 3 months.
What is the most likely diagnosis?

1. Pyogenic granuloma 0%
2. Basal cell carcinoma 0%
3. Molluscum contagiosum 0%
4. Squamous cell carcinoma 0%
Squamous cell carcinoma

• Majority on chronically sun-damaged skin
  – Scalp, neck, pinna, lips
  – Sun damage: solar elastosis, actinic keratosis
    and solar lentigines commonly seen.

• Often as: erythematous, hyperkeratotic,
  papule/nodule that may ulcerate
  – Over time, depressed center
  – New masses within scars or chronic ulcers
    suspicious

• Lesions head, neck, oral = ↑ risk metts
Risk factors - SCC

• Chronic sun exposure
• Fair skin and blue eyes
• Family history of SCC
• Scaring processes (chronic ulcers, burns, hidradenitis)
• Ionizing radiation
• Immunosuppression
• Certain HPV subtypes
• Chemical carcinogens

• Several genetic syndromes
  – Xeroderma pigmentosum
  – Oculocutaneous albinism
  – Epidermodyplasia verruciformis
  – Epidermolysis bullosa
  – KID syndrome (Keratitis-ichthyosis-deafness)
49 M arrives with back pain and fever. Dr. Penedo notes subtlety pictured on exam.
What would you most suspect?

1. Behcet’s with uveitis 0%
2. Hyphema suggestive of ocular trauma 0%
3. Sarcoid with uveitis 0%
4. Hypopyon suggestive endogenous endophthalmitis 0%
Endophthalmitis

• Hallmark: progressive vitritis
  – Massive inflammation vitreous cavity with inflammatory cells, primarily neutrophils. Progressive intraocular inflammation with loss of vision, pain and hypopyon.

• Exogenous – Endophthalmitis vitrectomy study (EVS)
  – Infecting organisms gain entry into the eye by direct inoculation – surgery, penetrating trauma, or contiguous spread from adjacent tissues. After Sx 1-2 wks, CNS (70%), Staph (10%), other GPCs. Chronic – P. acnes.

• Endogenous
  – Infectious agents hematogenously disseminated to the eye from a distant focus of infection

Endogenous endophthalmitis

• Microorganisms bloodstream cross blood-ocular barrier
  – Rare, 2-8% endophthalmitis cases
  – Often risk factors present: DM, HIV, IVDU, HD, CA, cardiac dz, immunosuppressive therapy or indwelling catheter.

• Presentation includes potential systemic symptoms and:
  – ↓ vision, eye pain, hypopyon, conjunctival injection, corneal edema, vitritis, poor fundus view secondary to inflammation.

• Management
  – Requires both local and systemic measures
## Endophthalmitis management

<table>
<thead>
<tr>
<th></th>
<th>Exogenous</th>
<th>Endogenous</th>
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</thead>
<tbody>
<tr>
<td>Vitreous culture</td>
<td>Standard</td>
<td>Essential</td>
</tr>
<tr>
<td>Intravitreal antibiotics</td>
<td>Standard</td>
<td>If progressive or severe</td>
</tr>
<tr>
<td>Vitrectomy</td>
<td>Standard</td>
<td>Favored some data, not studied in EVS</td>
</tr>
<tr>
<td>Systemic antibiotics</td>
<td>Not as sole therapy</td>
<td>Essential</td>
</tr>
<tr>
<td>Corticosteroids</td>
<td>Controversial</td>
<td>Controversial</td>
</tr>
</tbody>
</table>

I met two veterans, men of steel;
One came from Iraq, one hailed from Mobile;
Their ulcers did not respond to my pills;
I wonder what clues, their histories reveal...
What are the most likely diagnoses...

1. Pyoderma gangrenosum and necrobiosis lipoidica 0%
2. Cutaneous leishmaniasis and Blastomycosis 0%
3. MRSA furunculoses 0%
4. Squamous cell carcinoma and calciphylaxis 0%
Old World Cutaneous Leishmaniasis

• Endemic S. Europe, M. East, Africa, China, parts India, former USSR
  – Obligate intracellular parasites
    Leishmania genus – in via sandfly bites

• Over weeks to months, lesions
  – Papular → nodular → ulcerative
  – Ascend lymphatic chain (sporotrichoid)
  – Gradually enlarge or coalesce
  – Large plaques have central ulceration or crusting with raised indurated border
North American Blastomycosis

- Fungal soil organism in N. America

- Initial flu-like illness with indolent pulmonary infection
  - Resolve ~ 50% or may progress
  - Localized lung dz or extra-pulmonary after dissemination other organs – most commonly skin and bones

- Skin lesions
  - Ulcers - necrotic edges, granulation tissue at base
  - Verrucous skin lesions, crusted or purplish hue
  - Abscesses
41 M, neutropenic for 22 days after chemotherapy, CVL in place, now fever and lesion below.
Which would you favor as an etiology?

1. Pseudomonas aeruginosa 0%
2. Aspergillus 0%
3. Mucormycosis 0%
4. All are possibilities / can cause such lesions 0%
Necrotic skin lesions

- Multiple etiologies including
  - Bacterial P. aeruginosa and others → Ecthyma gangrenosum
  - Angioinvasive fungi → Mucor, Aspergillus, others

- May represent potentially life threatening emergencies

- Angioinvasive

![Diagram of blood vessels with labels tunica adventitia, tunica media, tunica intima]
Ecthyma gangrenosum

*Pseudomonas aeruginosa*: Common, but not exclusive!
Invasive aspergillosis in BMT
Fusarium
73 M with a history of asthma, arrives with 6 month history of progressive, pruritic rash.
What is the most likely diagnosis?

1. Psoriasis 0%
2. Darier’s disease 0%
3. Atopic eczema 0%
4. Crusted scabies 0%

✓ 4. Crusted scabies
Scabies vs. crusted scabies*
*No Norwegians were made fun of during the preparation of this table.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Scabies</th>
<th>Crusted scabies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Involved population</td>
<td>Anyone</td>
<td>Elderly, AIDS, mentally challenged, DM, immunocompromised</td>
</tr>
<tr>
<td>Amount of mites</td>
<td>Dozens</td>
<td>Thousands to millions</td>
</tr>
<tr>
<td>Clinical presentation</td>
<td>Itching nodules, burrows</td>
<td>Itching (50%), crusting, may be erythrodermic</td>
</tr>
<tr>
<td>Affected areas</td>
<td>Trunk/extremities, genital areas, web spaces, wrists/ankles</td>
<td>Whole body</td>
</tr>
</tbody>
</table>

Crusted scabies

- Therapy: evidence from case reports/series.
- Permethrin (topical for index patient) and ivermectin (PO) now and in 1 week.

Bonus
34 M on chronic steroids myasthenia gravis with these lesions on his arm.
What do these lesions most likely represent?

1. Sweet’s syndrome
2. Folliculitis – most likely MRSA
3. Cutaneous T cell lymphoma
   (lymphomatoid papulosis type)
4. Majocchi’s granuloma

✓ 4. Majocchi’s granuloma
Majocchi’s granuloma or nodular granulomatous perifolliculitis

• Described 1883 Domenico Majocchi women with frequent skin trauma to their lower extremities.
  – Same organisms responsible tinea corporis and pedis (Trichophyton rubrum, T. mentagrophytes or Epidermophyton floccosum) and others.

• Occlusion/trauma skin (ex: shaving), use of topical steroids on unsuspected tinea and immunocompromised states = ↑ risk

• As of 2011, 22 cases in solid organ transplant literature
  – Presents as subacute to chronic nodular lesions, progressively ↑ in size and number, mainly affect lower extremities.

Majocchi’s granuloma (nodular granulomatous perifolliculitis)

- Dermatophyte invades hair follicle → granulomas and/or suppuration
- Look for cluster of pustules/papules/nodules within erythematous scaling plaque. May resemble bacterial furuncle or carbuncle
- Can occur throughout body but spare palms/soles/mucous membranes.
- Tx: systemic antifungals (terbinafine, itraconazole, etc.)
Thank you all for your kind attention.