10+ Essential skin diseases for non-dermatologist

Common skin infections

July 3, 2014

Suthinee Rutnin, MD

Division of Dermatology, Department of Internal Medicine, Faculty of Medicine, Ramathibodi Hospital, Mahidol university

Objectives

- Diagnosis and management of common bacterial infection:
  - Staphylococcal & Streptococcal infection
  - Corynebacterial infection
  - Pseudomanas infection
- Diagnosis and management of common fungal infection:
  - Tinea versicolor
  - Dermatophytosis
  - Mucocutaneous candidiasis

Objectives

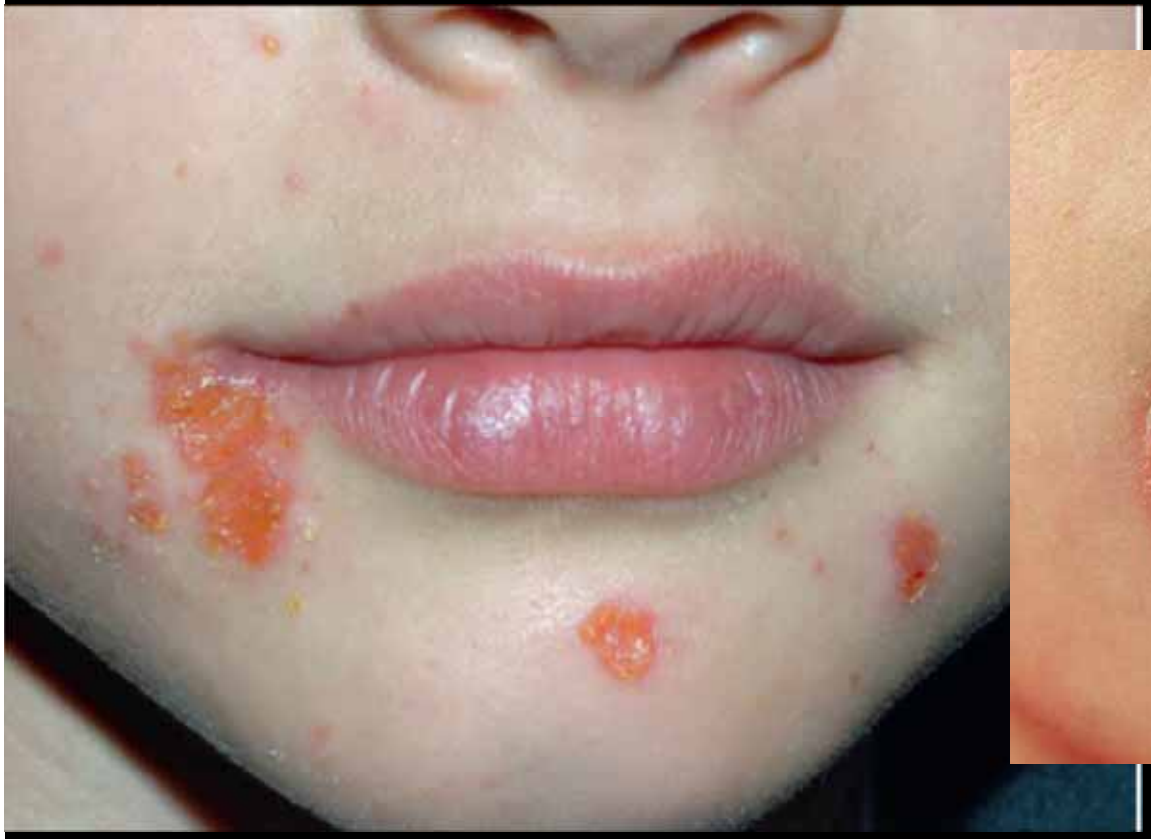
- Diagnosis and management of common viral infection:
  - Herpes infection
    - HSV
    - Varicella
    - Herpes zoster

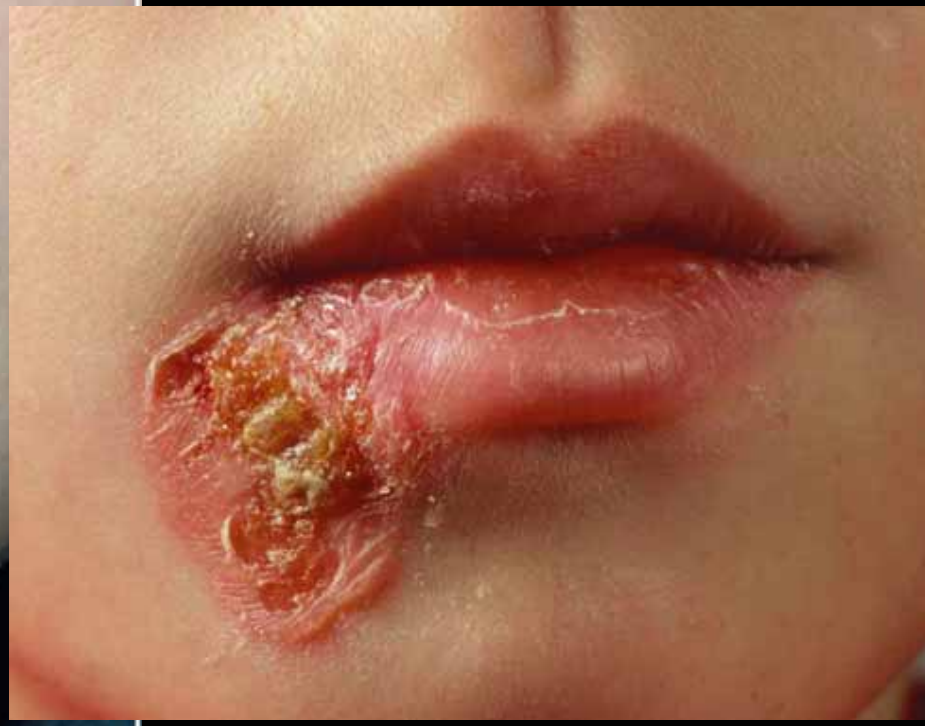
Bacterial infection

- 20% of outpatient dermatology visits: bacterial skin infections
- Staphylococci and streptococci: majority of bacterial infection
- An increase in the prevalence of community-associated methicillin-resistant *Staphylococcus aureus* (CA-MRSA)

Staphylococcal & Streptococcal infection

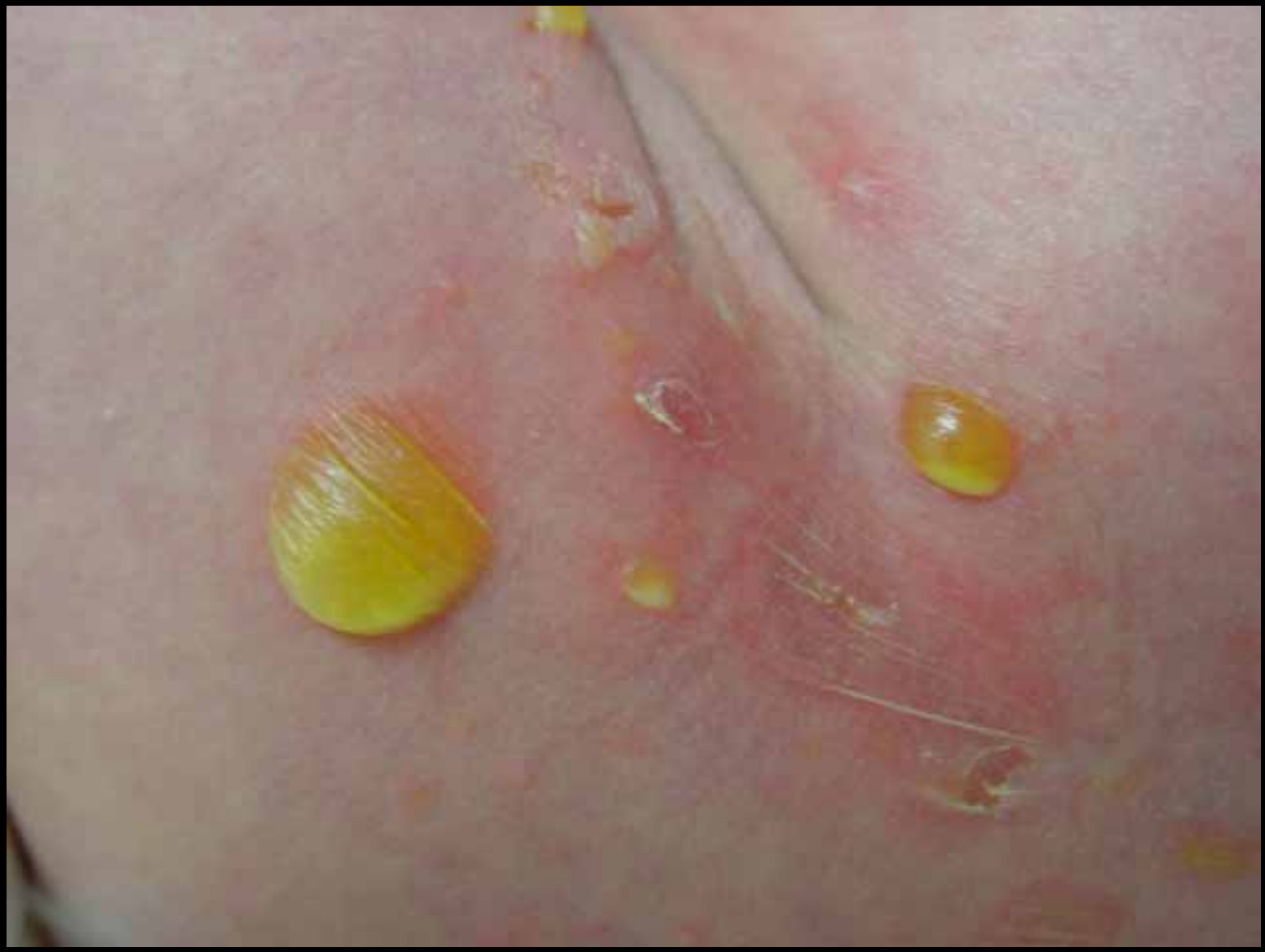
Impetigo





honey-colored crusting mostly caused by staphylococcus aureus, and lesser by group A βhemolytic streptococci

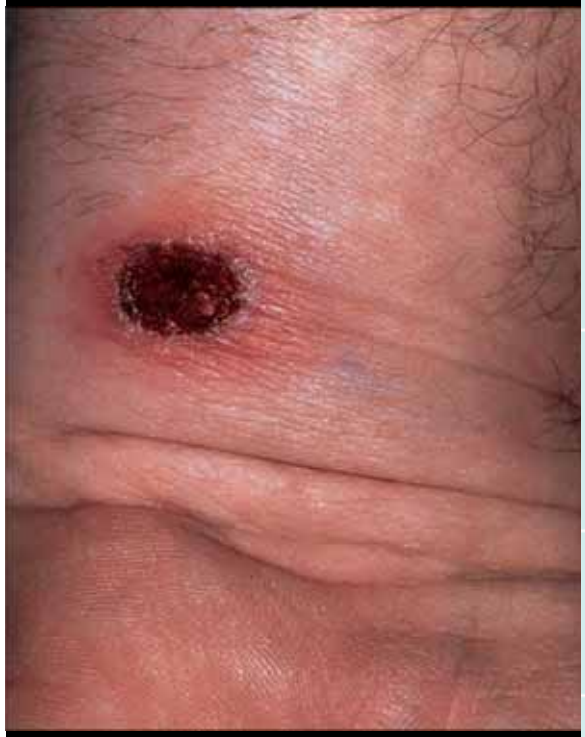
Bullous impetigo



Characteristic features of Bullous and Non bullous impetigo

	Non-bullous impetigo	**Bullous impetigo**
Epidemiology	~70% of all cases of impetigo Children most often affected	Less common Often occurs in the neonatal period
Clinical lesions	Early: single 2–4 mm erythematous macule evolving to transient vesicle & pustule Late: superficial erosion with a typical "honey-colored" yellow crust	Early: small vesicles enlarge into 1–2 cm superficial bullae Late: flaccid, transparent bullae measuring up to 5 cm in diameter
Distribution	Face (around the nose and mouth) and extremities	Face, trunk, buttocks, perineum, axillae and extremities
Clinical course	Usually resolves within 2 weeks without scarring if untreated	Usually resolves in 3–6 weeks without scarring if not treated
Complica-tions	**5% of impetigo caused by S. *pyogenes* results in acute post-streptococcal GN (APSG)** Risk of APSG is not altered by treatment with antibiotics Not link to a risk of rheumatic fever	**In infants/young children and adults with immunodeficiency or renal failure**, exfoliative toxin may disseminate and cause **staphylococcal scalded skin syndrome**

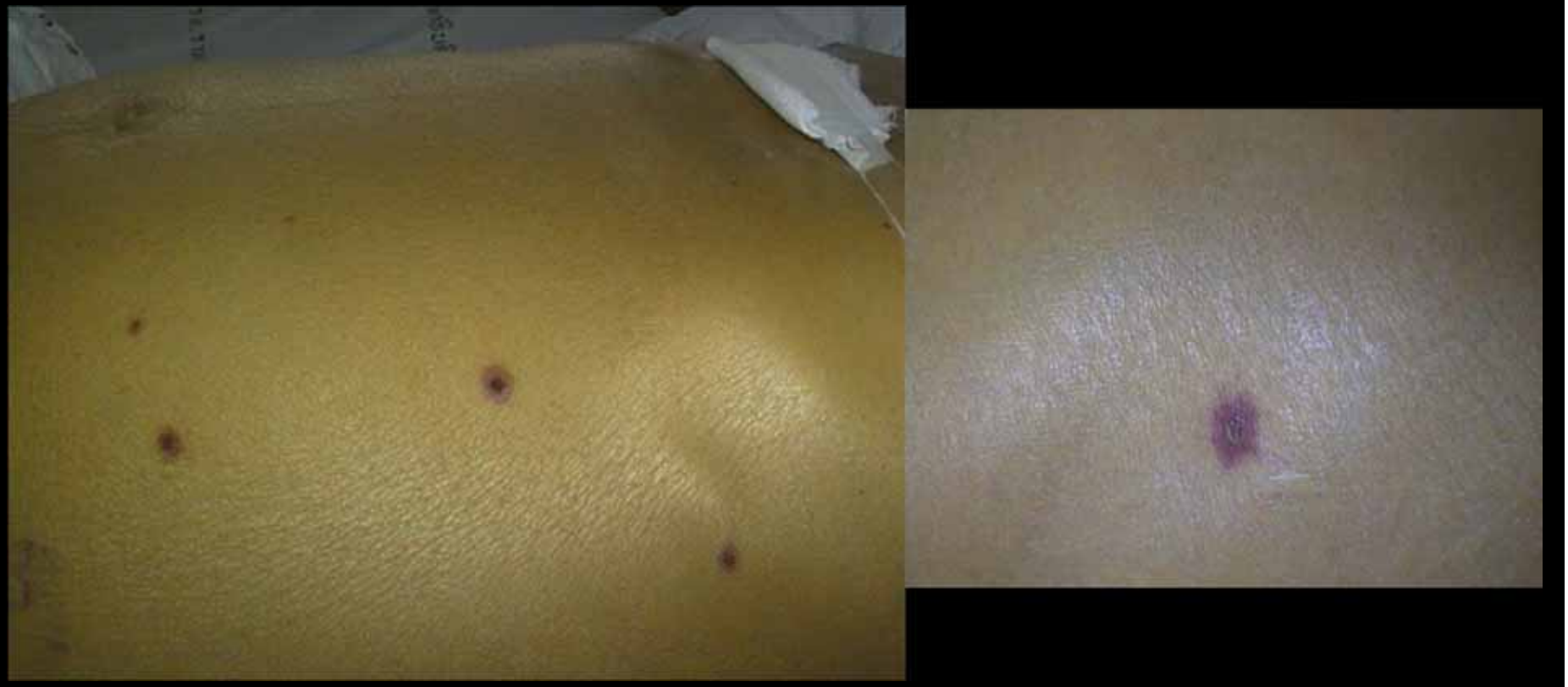
Ecthyma



Punch out ulcer
with hemorrhagic crust

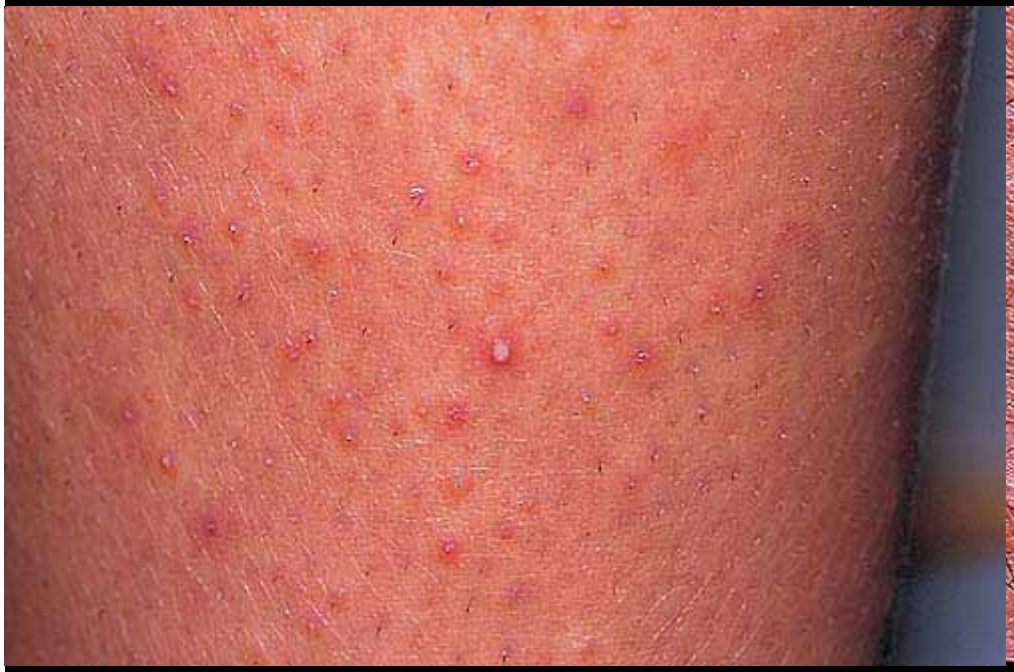
CLINICAL FEATURES OF ECTHYMA	
Clinical findings	• Fewer than 10 lesions are typically seen, most commonly on the lower extremities • An initial vesiculopustule enlarges (0.5–3 cm in diameter) over the course of several days, and develops a hemorrhagic crust • The ulcer has a "punched-out" appearance and a purulent, necrotic base • Lesions are slow to heal and produce scarring
Risk factors	• Young age (children), lymphedematous limbs, poor hygiene, neglect (including the elderly), immunosuppression, scratching (e.g. of insect bites), trauma
Complications	• Lesions are often contaminated with staphylococci • Systemic symptoms and bacteremia are rarely seen • Cellulitis and osteomyelitis are extremely infrequent
Diagnosis	• Clinical appearance • Culture of moist, purulent base; skin biopsy with deep-tissue Gram stain and culture occasionally required
Differential diagnosis	• Ecthyma gangrenosum • Ulcers due to vasculitis, vasculopathies, other causes

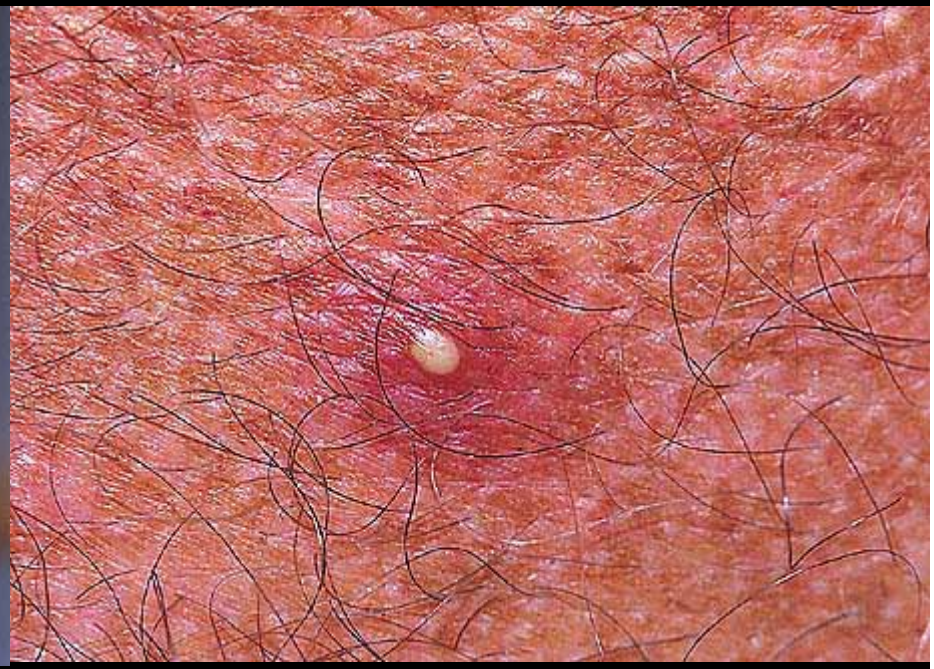
Ecthyma gangrenosum



gangrenous ulcer with a central black/gray eschar surrounded by an erythematous halo

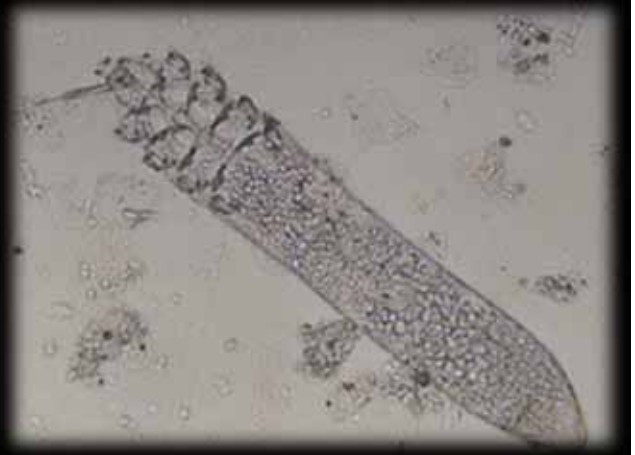
Folliculitis



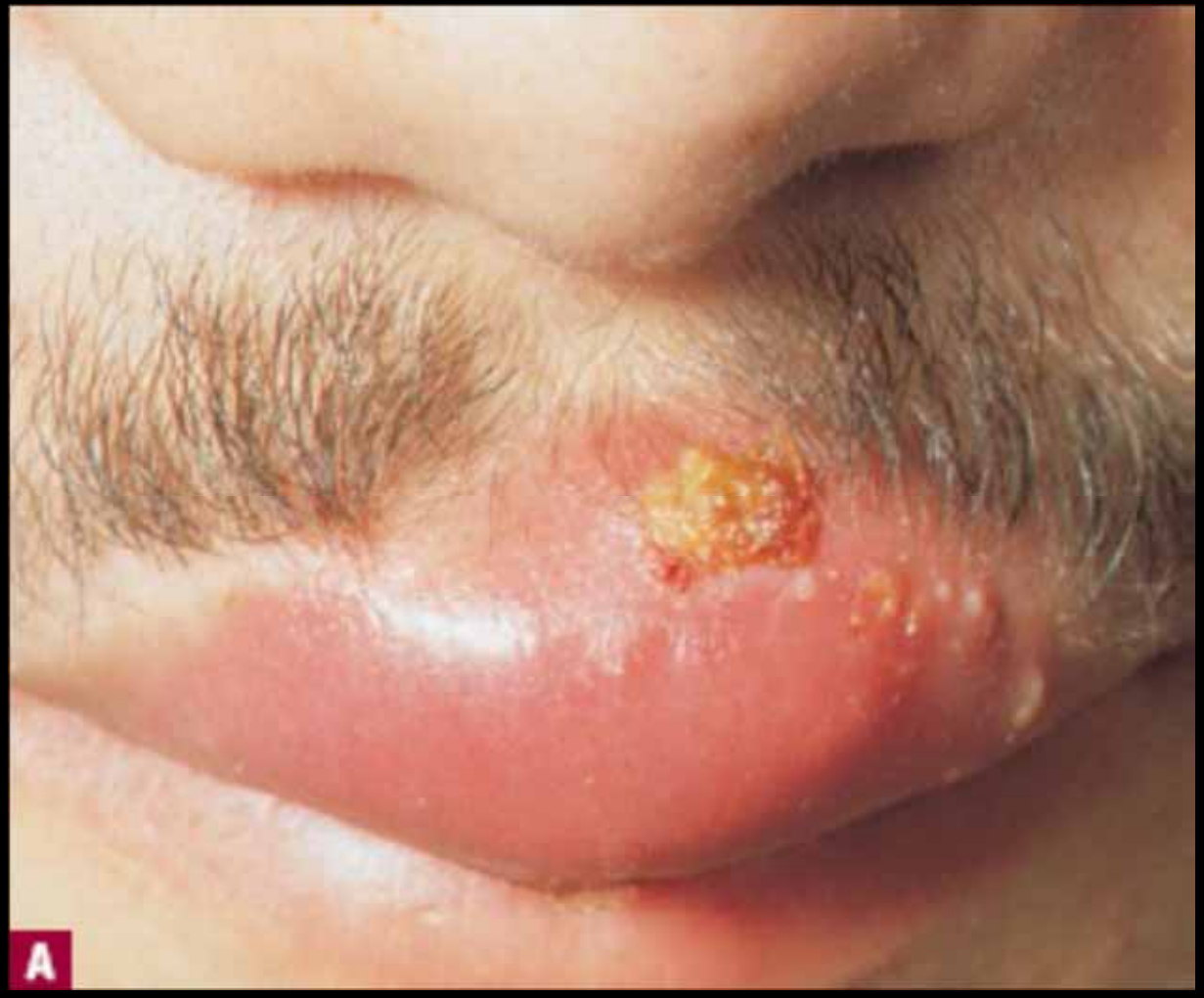


Infectious folliculitis

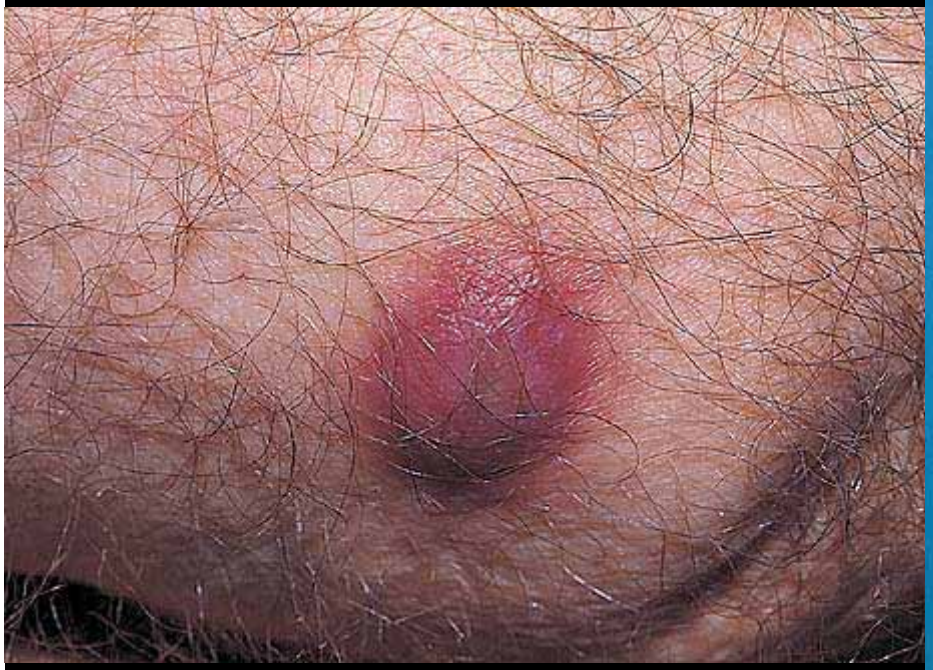
- Bacterial folliculitis: Staphylococcus, Pseudomonas, Gram negative, Syphilitic folliculitis
- Fungal folliculitis: Dermatophyte, candida, pityrosporum
- Viral folliculitis: Herpes simplex
- Infestration: Demodisidosis

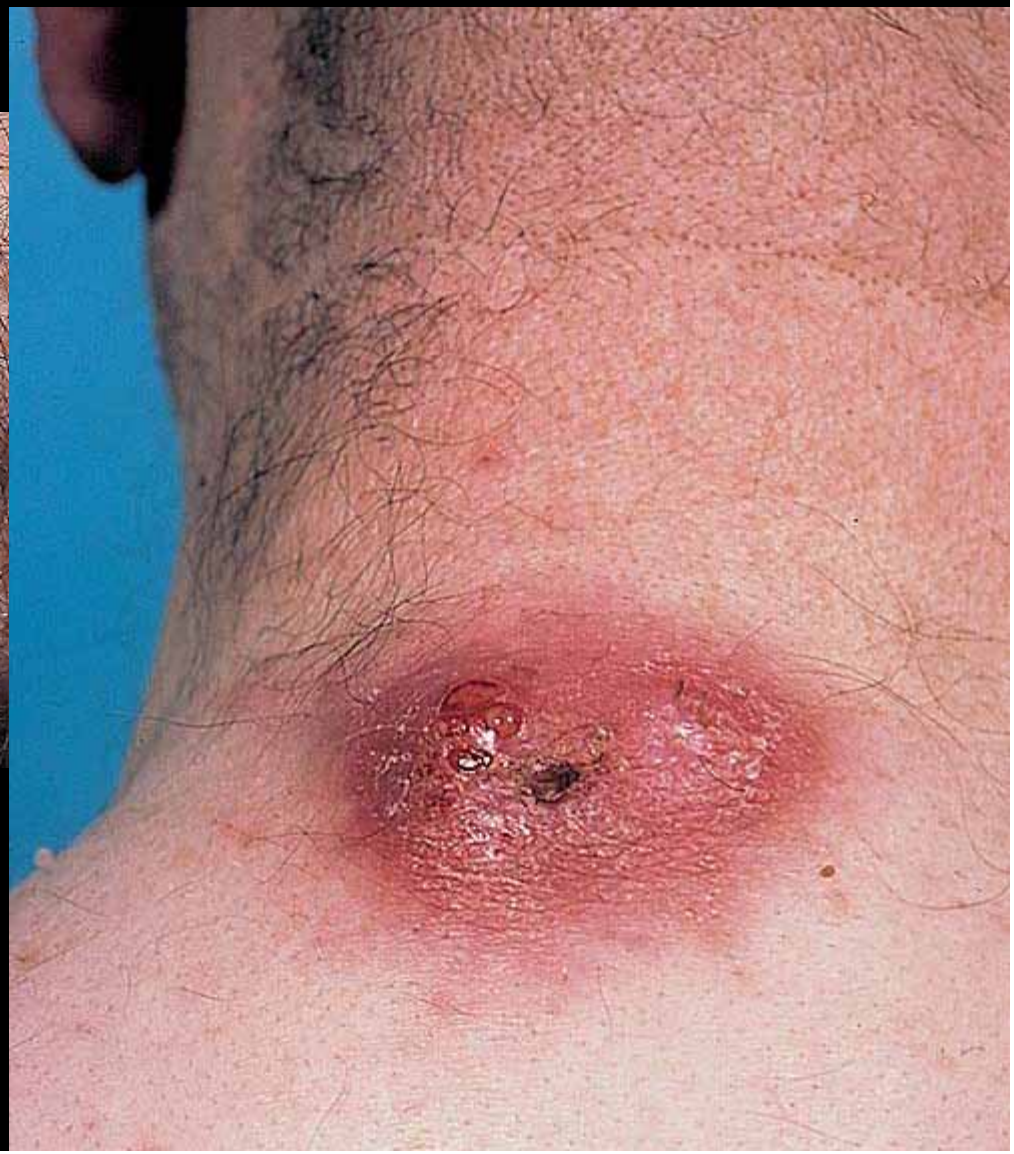


Furuncle



Furuncle/Carbuncle





Topical, oral or intravenous therapy

- The extent of skin involvement
- The presence of complications (e.g. cellulitis, lymphangitis)
- Comorbid conditions (e.g. atopic dermatitis, varicella)
- The patient's immune status
- Local drug-resistance patterns (e.g. the prevalence of community-associated methicillin-resistant *S. aureus* (CA-MRSA)

Impetigo/Ecthyma

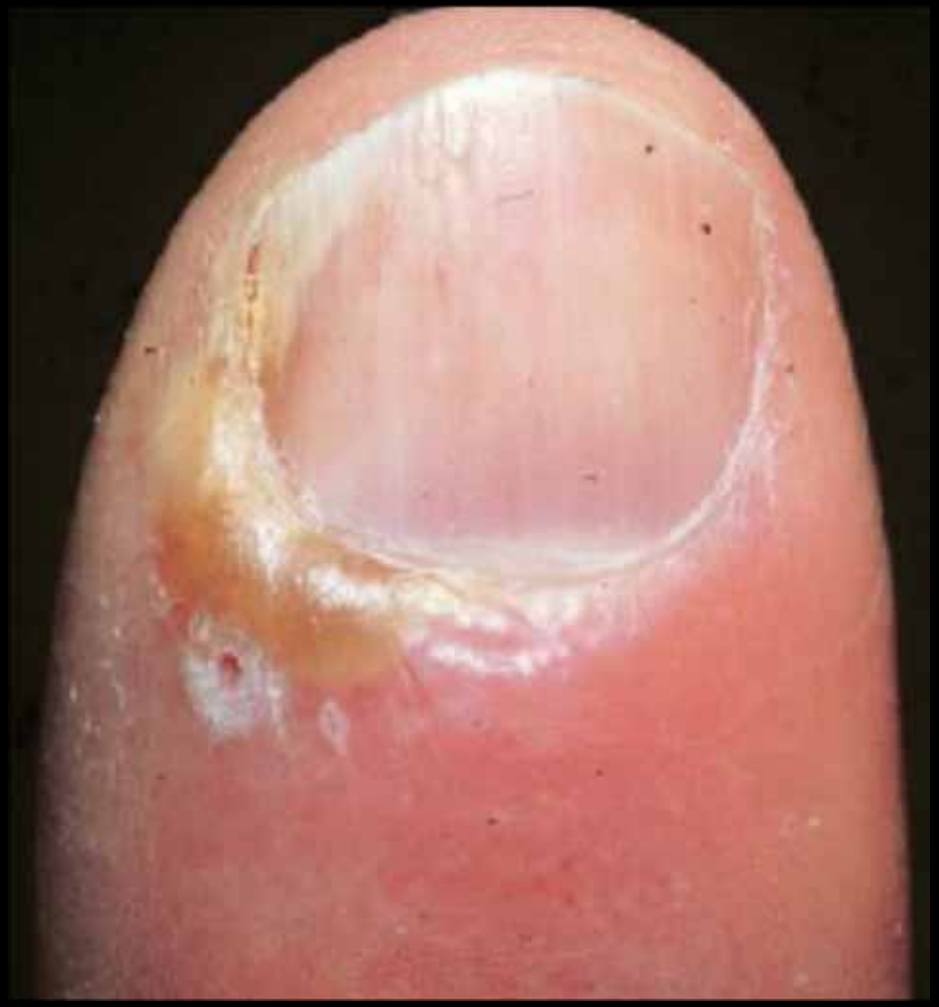
	Uncomplicated		Complicated	
	First line	Second line	First line	Second line
Impetigo	-2% Mupirocin ointment -2% fusidic acid cream	-β-lactamase-resistant penicillin -1 th generation cephalosporin -Clindamycin -Macrolide	IV ceftriaxone (daily dosing)	-IV ampicillin/sulbactam -IV cefuroxime
Ecthyma	First line -β lactamase-resistant penicillin (e .g . Dicloxacillin) -First-generation cephalosporin (e .g . Cephalexin)			

CA-MRSA

Minor infection		Severe infection		
First line	Second line	First line	Second line	Third line
Trimethoprim–sulfamethoxazole† Minocycline† Doxycycline† Clindamycin	Linezolid	Vancomycin	Linezolid Daptomycin Telavancin	Quinupristin-dalfopristin Tigecycline

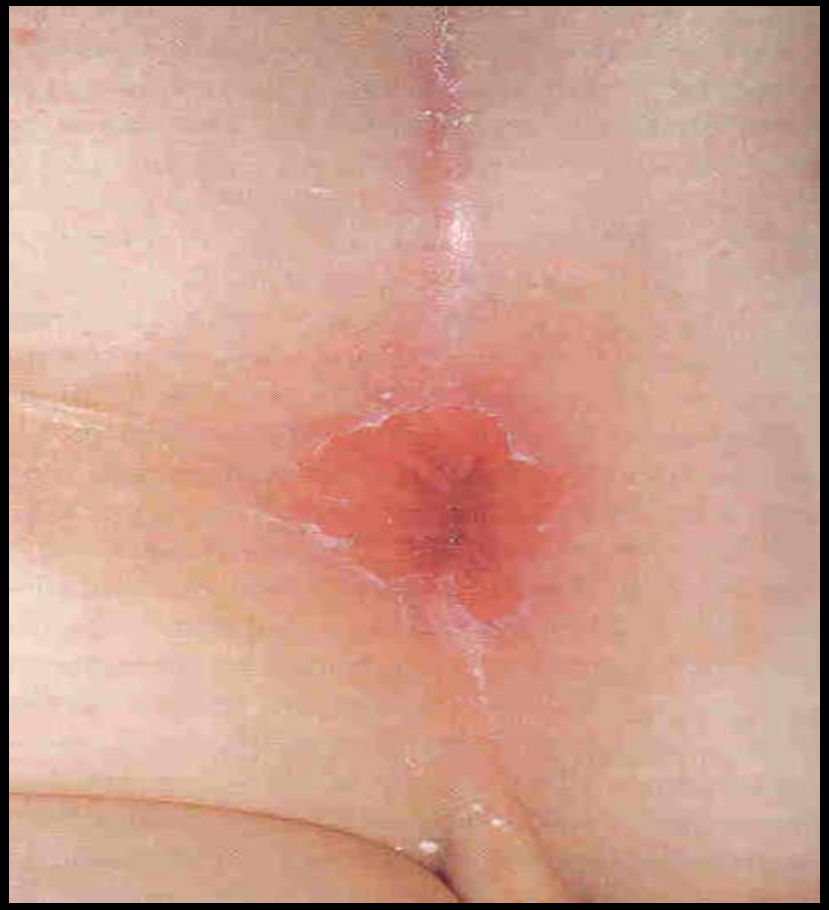
†Do not provide coverage of group A streptococci; if coverage of the latter and CA-MRSA is desired, can be combined with a β-lactam.

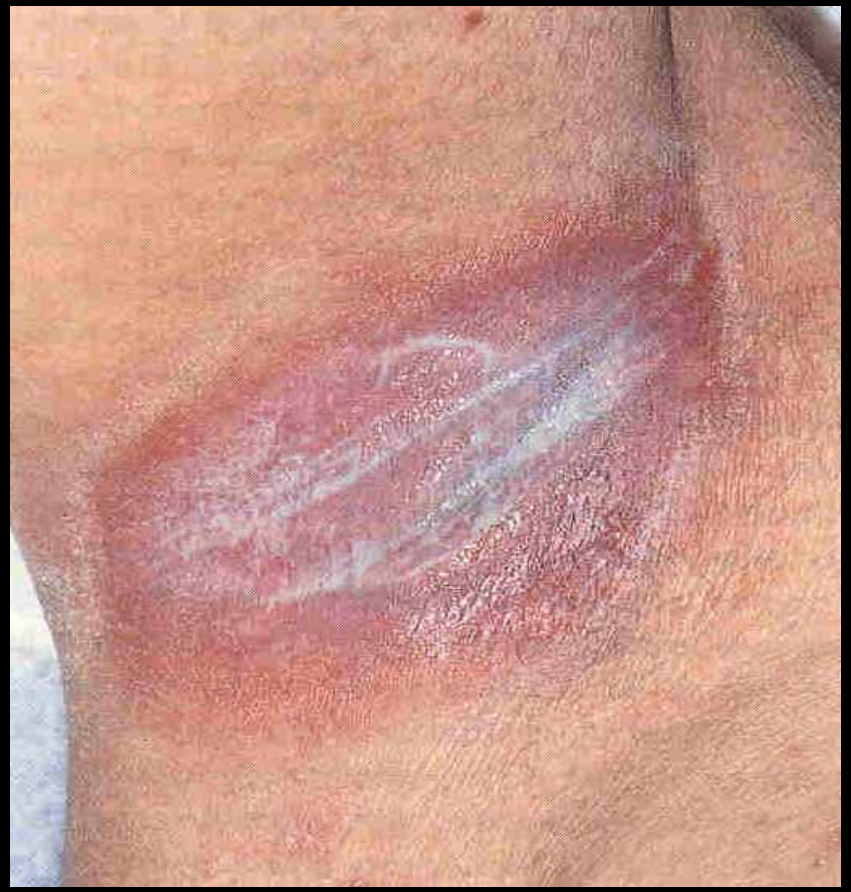
Acute paronychia



Blistering distal dactylitis

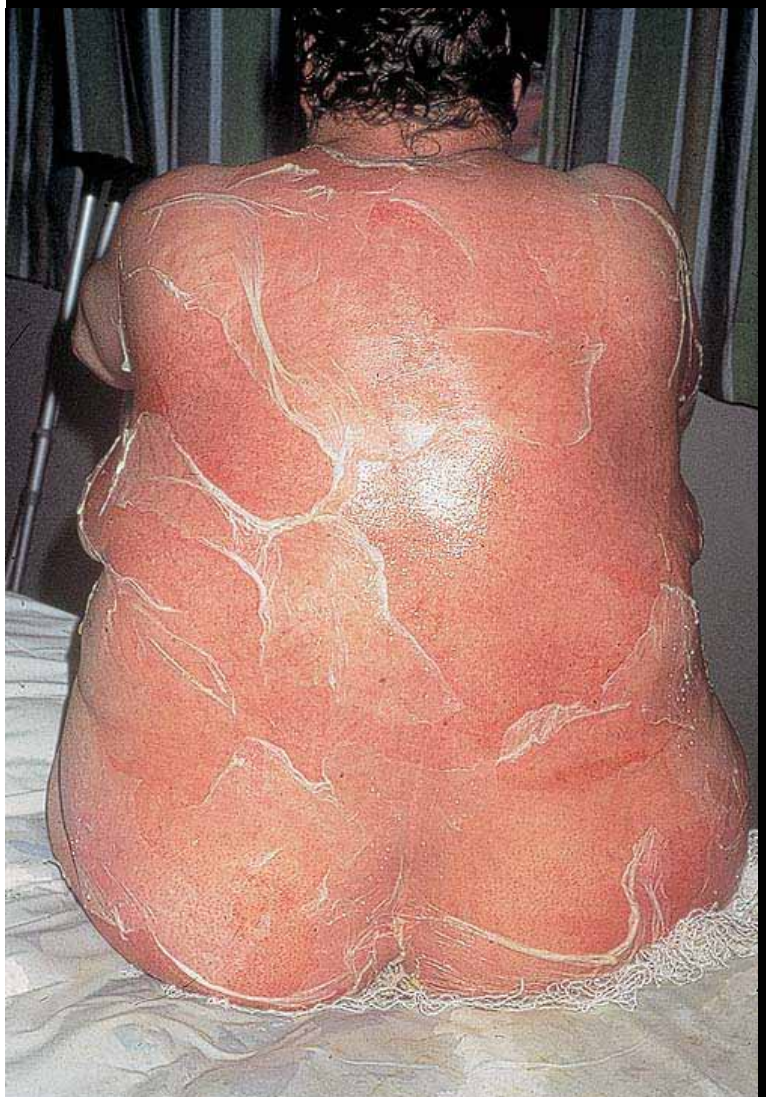
Streptococcal intertrigo

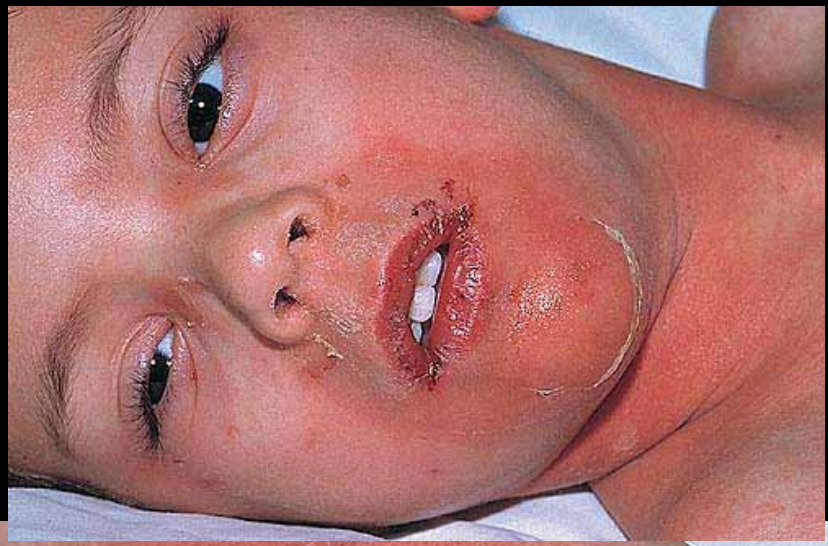


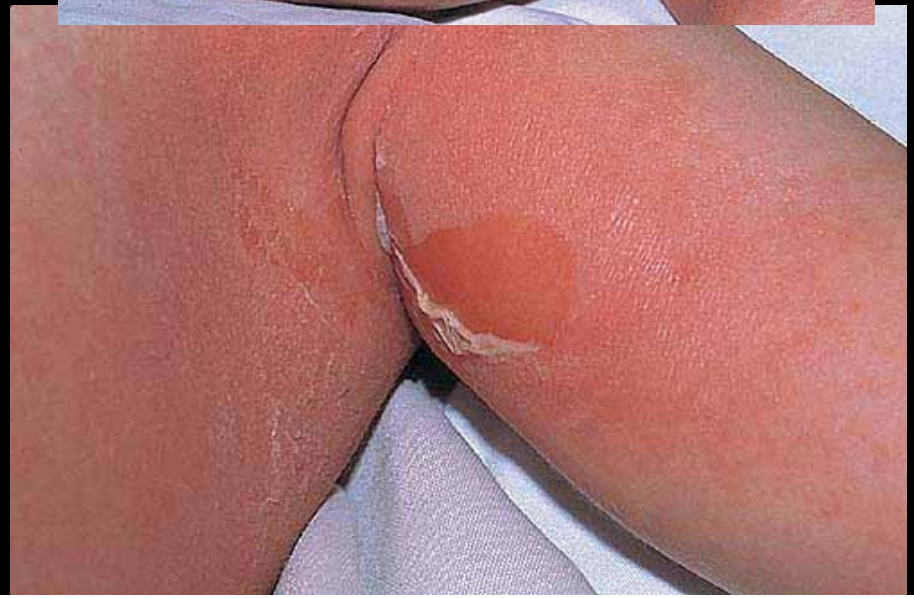


Acute lymphangitis

Staphylococcal scalded-skin syndrome







Staphylococcal scalded-skin syndrome

Prodome

- Fever of malaise, fever, irritability

Exanthem

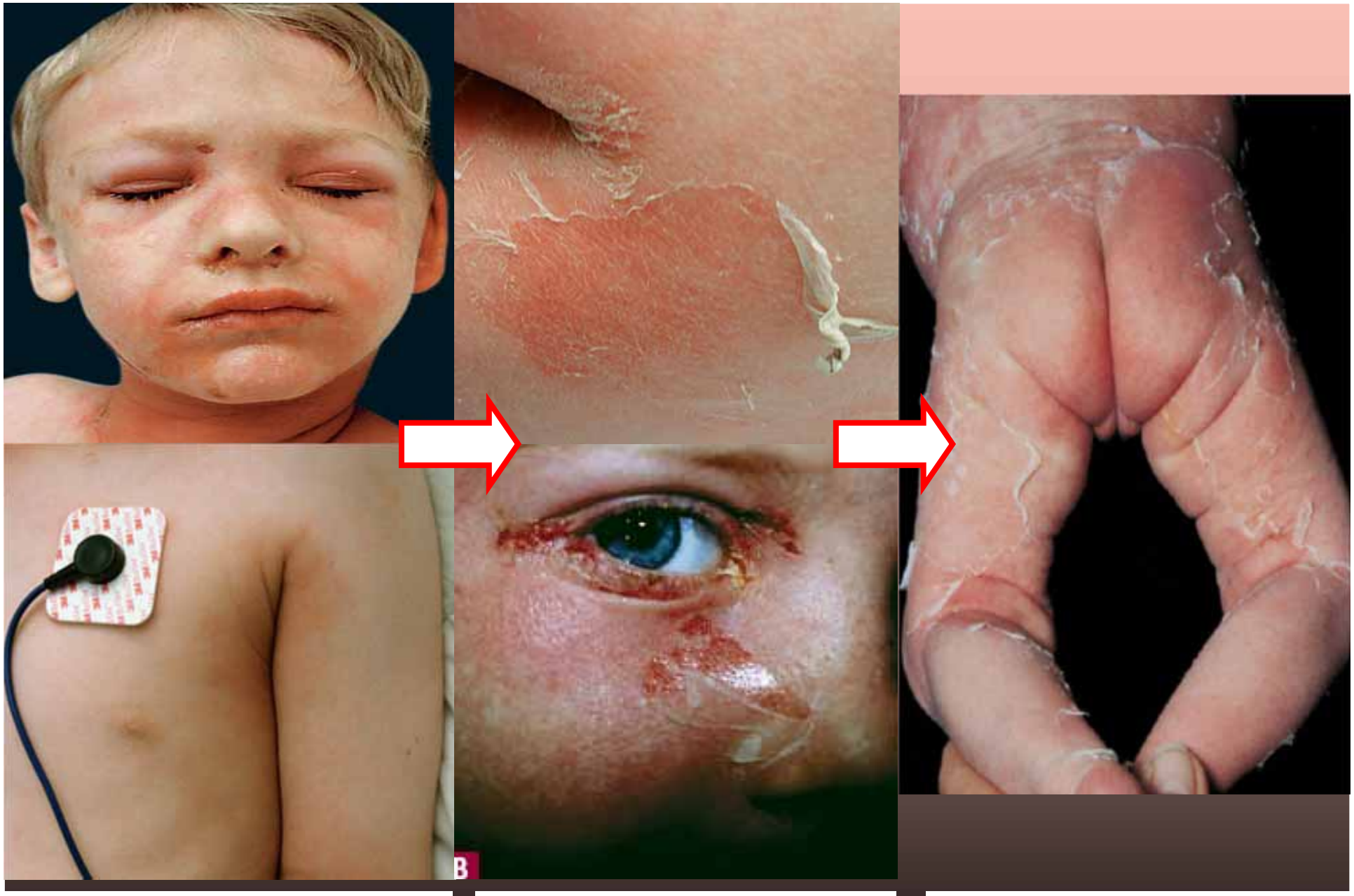
- Generalized exanthem & cutaneous tenderness
- Erythema typically first appear on head, intertriginous sites spread to generalizeation within 48 hours

Denudation

- Skin wrinkled forming blister classically flexoral area
- Patient characteristic periorificial crusting & radial fissuring

Desquamate

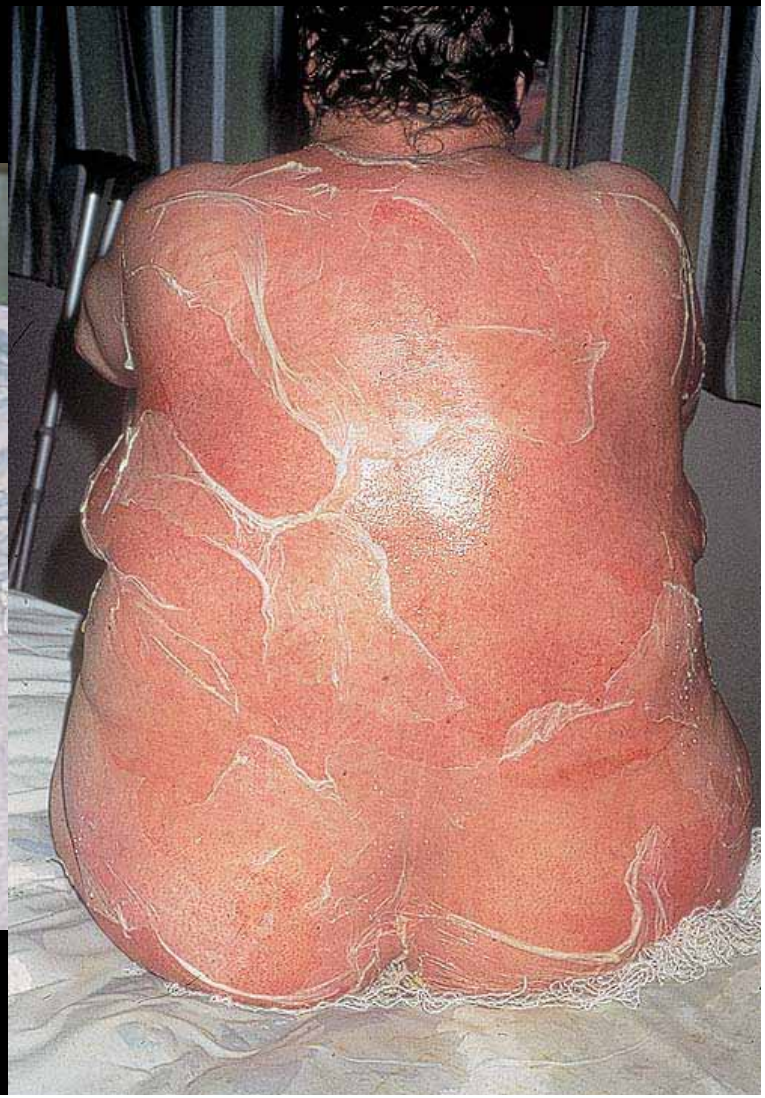
- Scaling & Desquamation continue for the rest 3-5 days
- Followed by re-epithelization without scarring in 1-2 weeks after receiving proper Rx


Generalized exanthem
Cutaneous tenderness
Blistering ,denudation
Nikolsky sign +
Generalized
desquamation

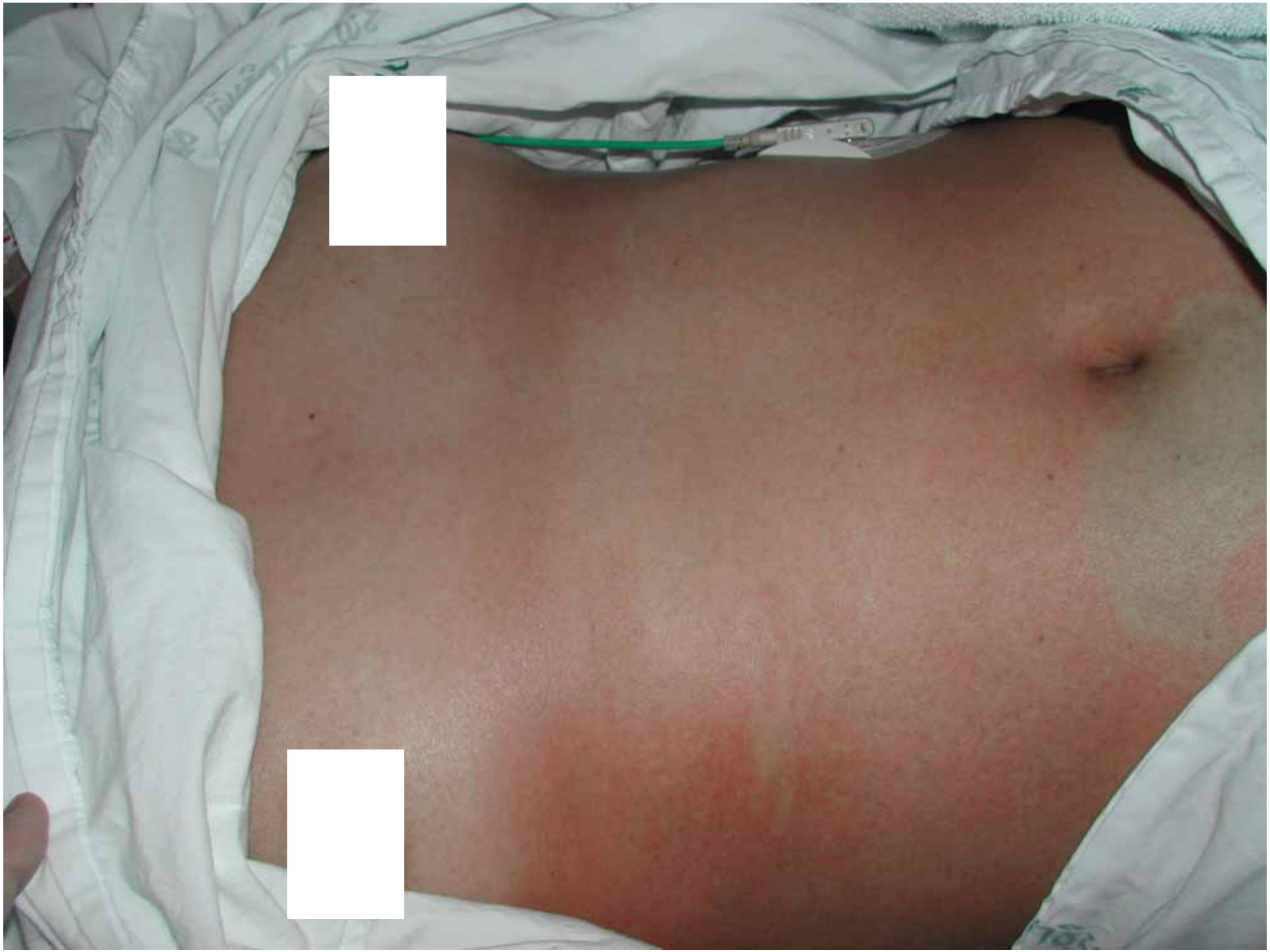
Comparison between TEN AND SSSS

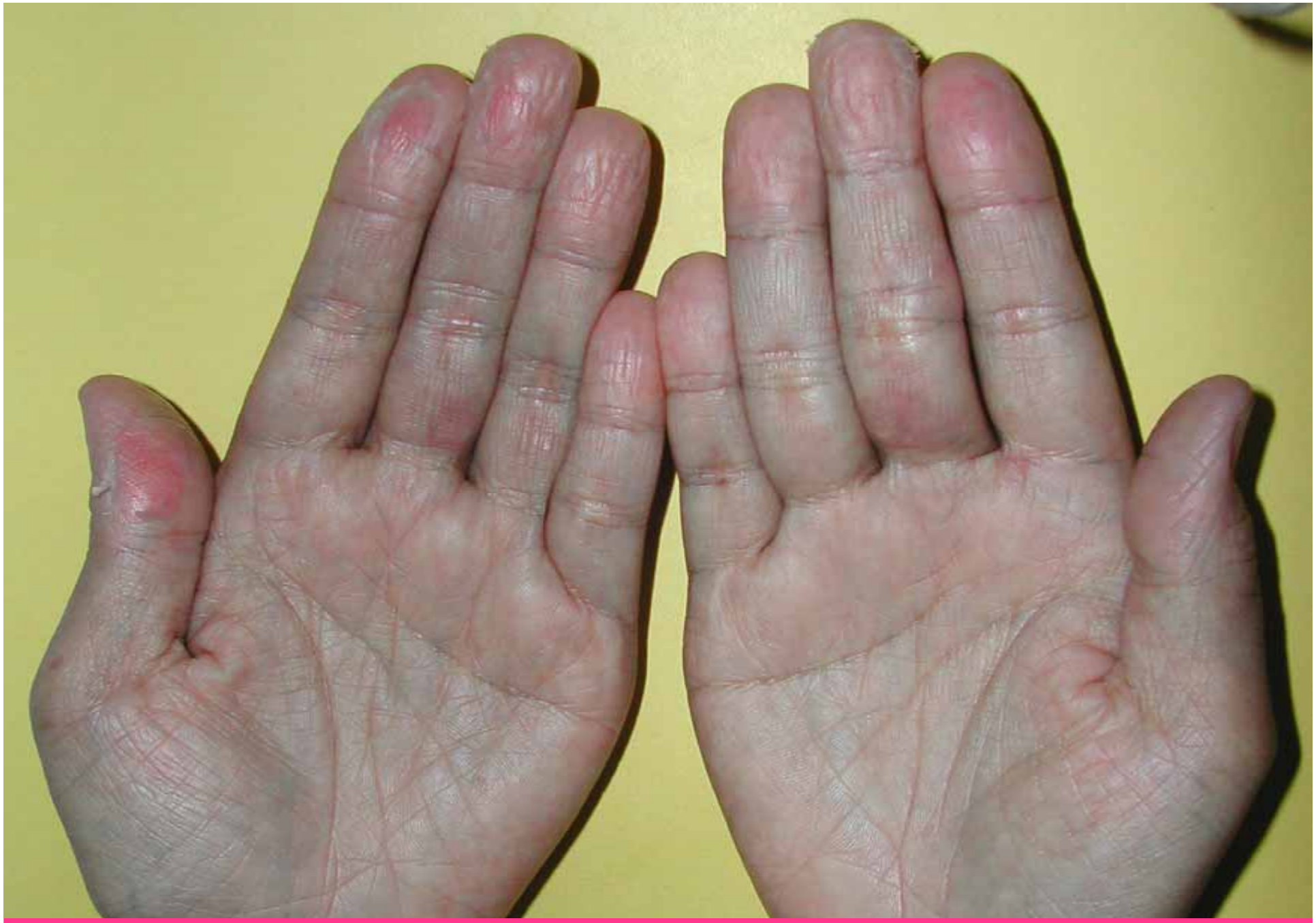
	TEN	SSSS
Cause	Usually drug-induced	Toxin-producing *S. aureus* infection
Age	Adults	Infants and young children
Histology	Dermo–epidermal separation; dermis has a dense inflammatory infiltrate	Granular layer split in epidermis; dermis lacks inflammatory infiltrate
Distribution of rash	Areas of sparing present	Generalized
Mucous membranes	Involved	Uninvolved
Nikolsky's sign	In some areas, difficult to elicit	Present in seemingly uninvolved skin
Face	Lip and mucous membrane redness, edema	Perioral crusting and fissuring with mild facial swelling and erosions
Treatment	Standard burn treatment, IVIG, corticosteroids (controversial)	Antibiotics (-lactamase resistant) and supportive care

TEN vs SSSS

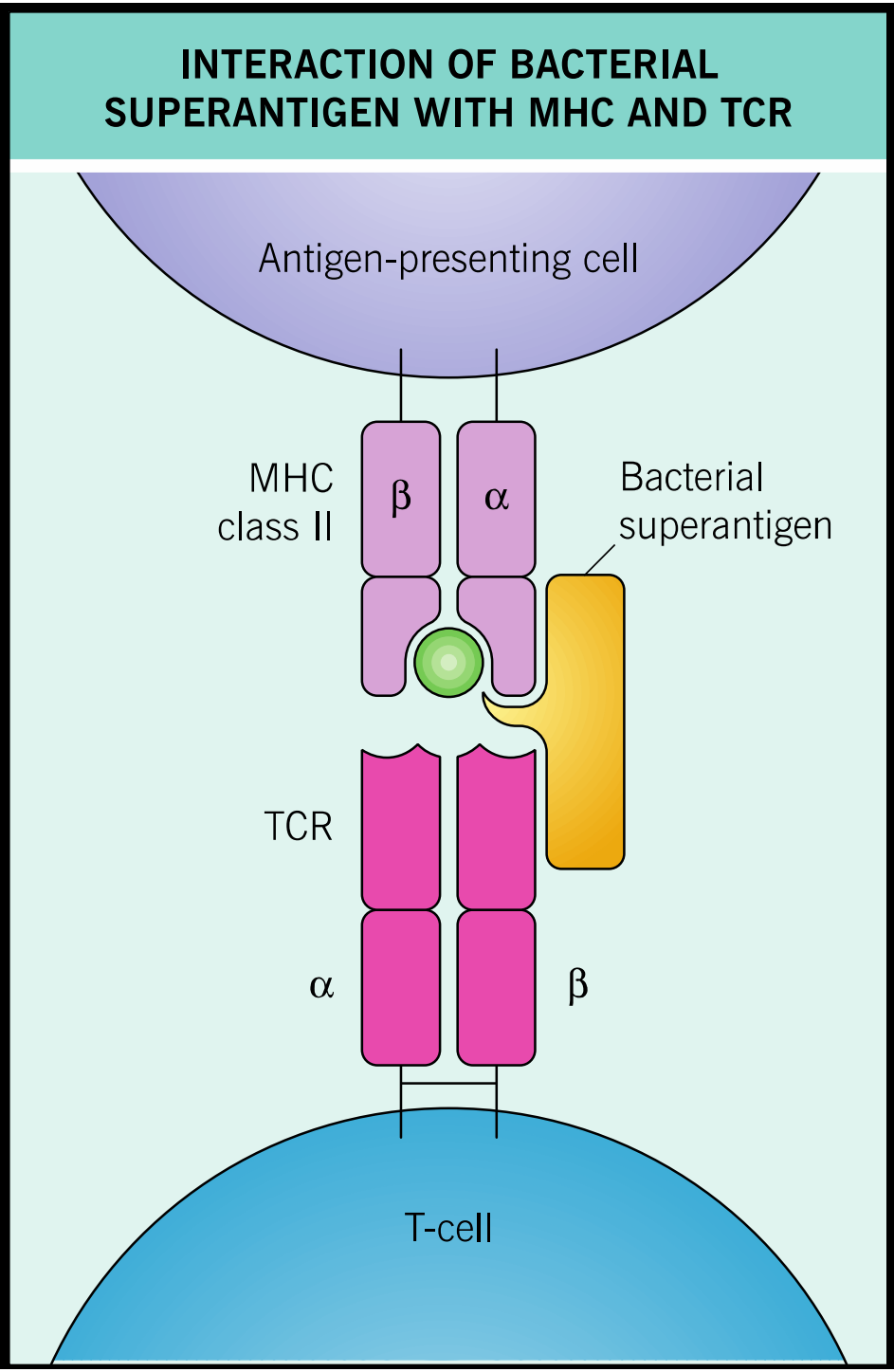


Toxic shock syndrome





6 days latter



- Superantigen:
  - bind MHC class II of APC and vβ of TCR
  - Produce mosaic release of cytokines, chemokines, clonal T cell expansion

Toxic shock syndrome

CASE DEFINITIONS FOR THE TOXIC SHOCK SYNDROMES

TOXIC SHOCK SYNDROME

- Fever: temperature >102°F (or >38.9°C)
- Rash: diffuse macular erythroderma
- Desquamation: 1–2 weeks after the onset of illness (especially palms and soles)
- Hypotension: systolic blood pressure <90 mmHg for adults (<5th percentile for children)
- Involvement of three or more of the following organ systems:
  - Gastrointestinal
  - Muscular
  - Central nervous
  - Renal
  - Hepatic
  - Mucous membranes (erythema)
  - Hematologic (platelets <100000/mm^3)
- Negative results for the following tests (if done):
  - Blood and cerebrospinal fluid cultures (blood culture may be positive for *Staphylococcus aureus*)
  - Serologic tests for Rocky Mountain spotted fever, leptospirosis, measles

Toxic shock syndrome

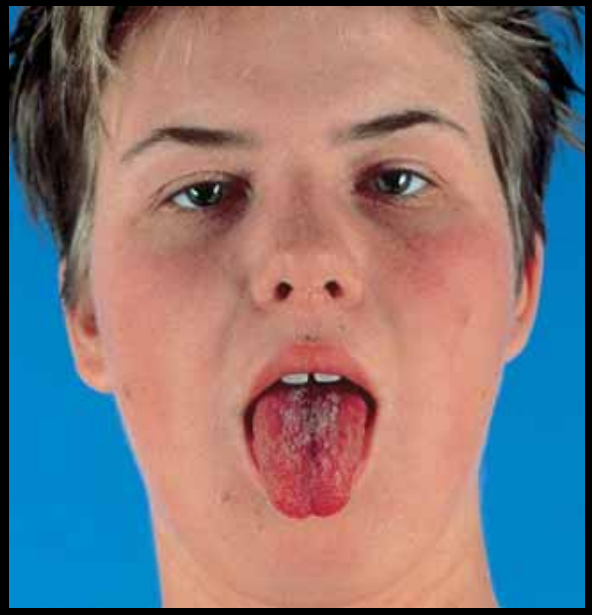
	Staphylococcal	Streptococcal
Typical patient	Young (15–35 years) and healthy	Young (20–50 years) and healthy
Diffuse macular erythroderma	Very common	Less common
Vesicles and bullae	Rare	Uncommon (5%)
Localized extremity pain	Rare	Common
Soft tissue infection	Rare	Common
Hypotension	100%	100%
Renal impairment	Common	Common
Predisposing factors	Surgical packing, surgical meshes, abscesses, contraceptive sponge, tampon	Lacerations, bites, bruises, varicella
Positive blood cultures	<15%	>50%
Mortality	<3%	30–60%

Scarlet fever

Streptococcal scarlet fever

Fever, exudative pharyngitis, strawberry tongue,
headache, abdominal pain, vomit

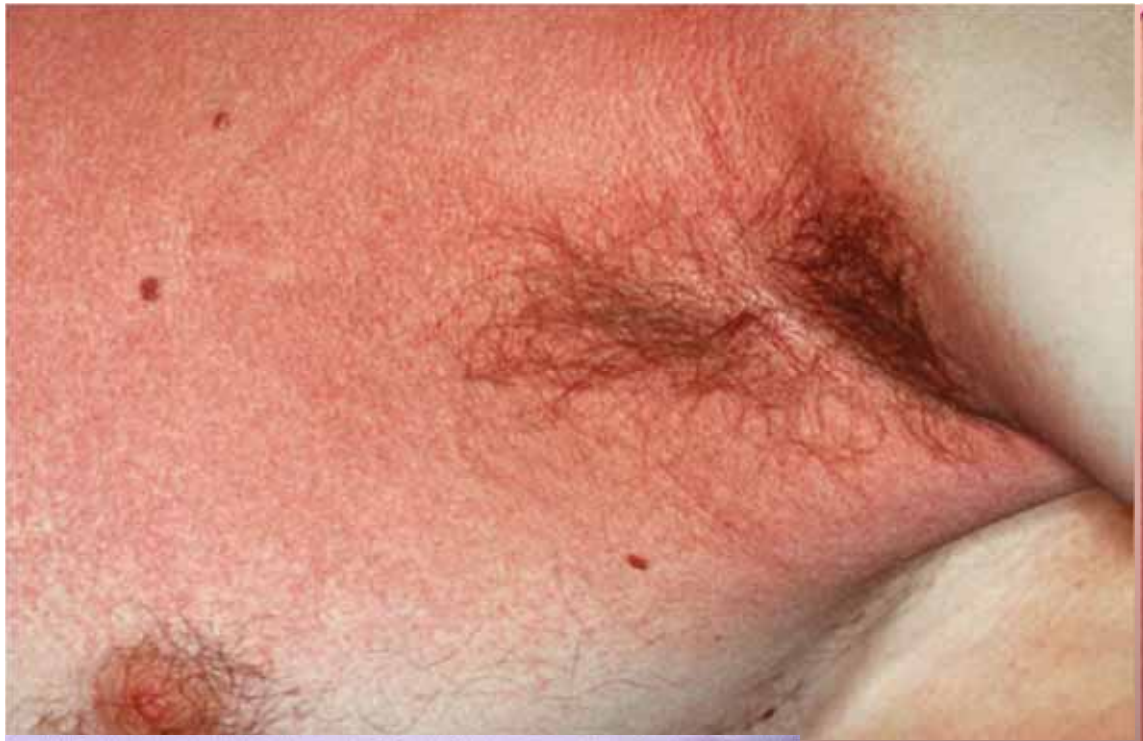
1-2 days

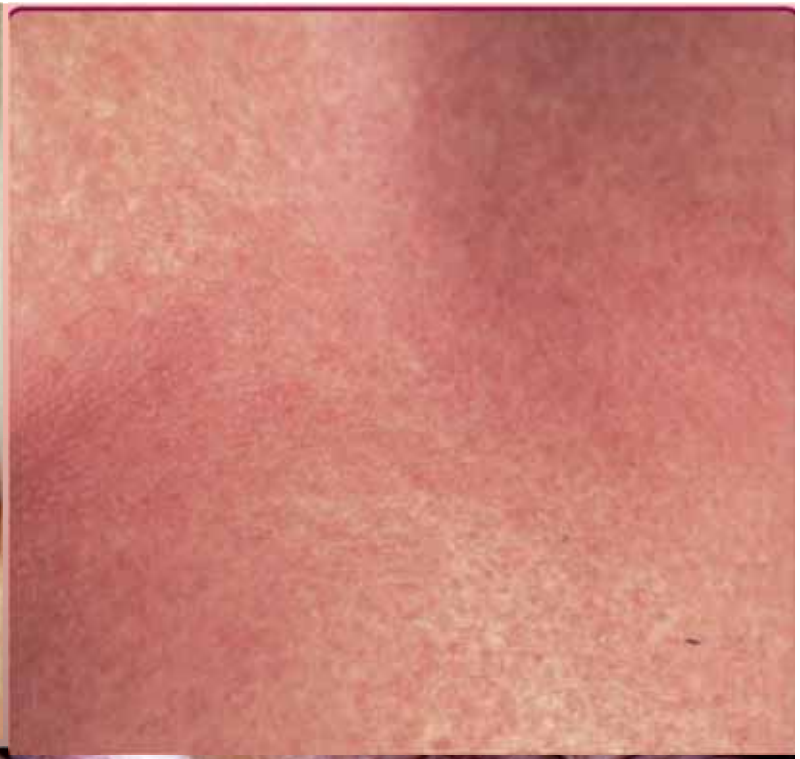


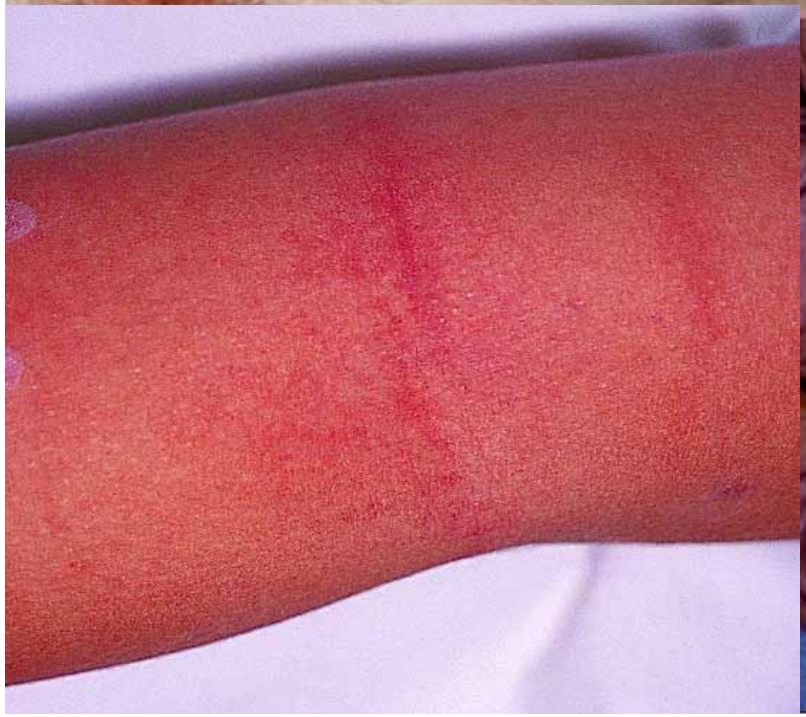
Generalized exanthem
Sand paper rash, Pastia sign

3-4 days

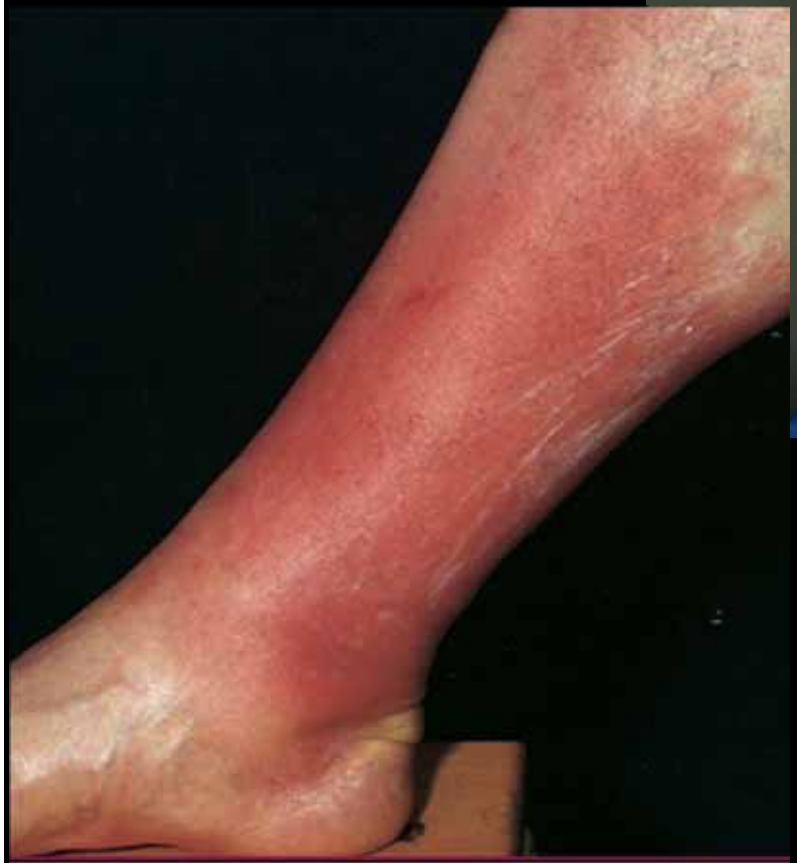
Desquamative phase
usaully starting on the face,
peeling of palm & finger occur approximately 1 wk later

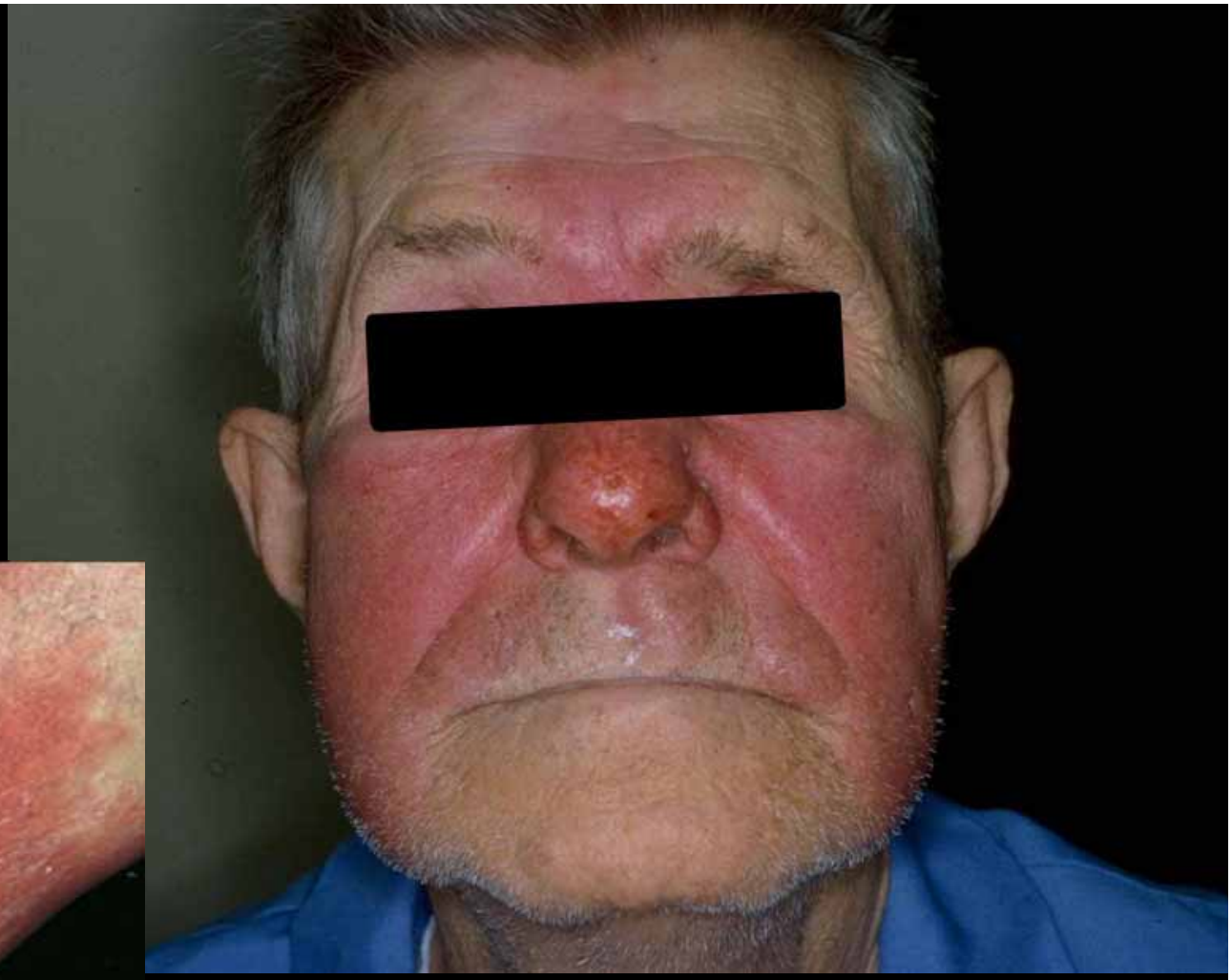




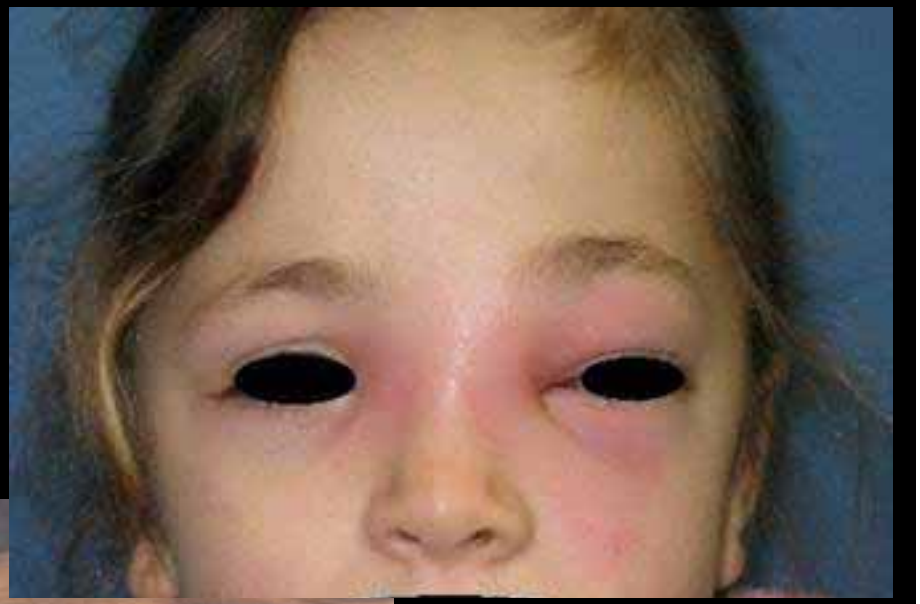


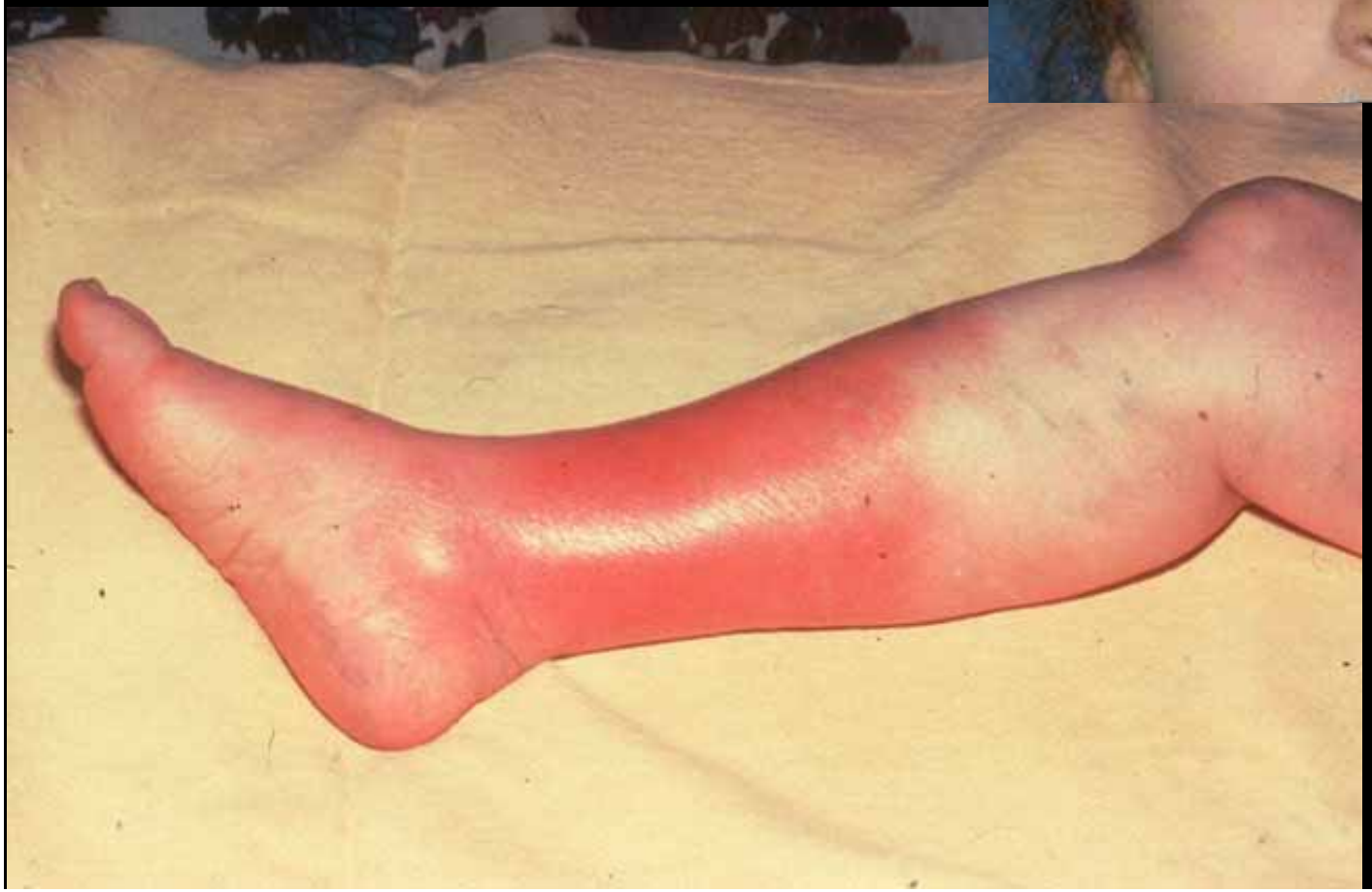
Erysipelas



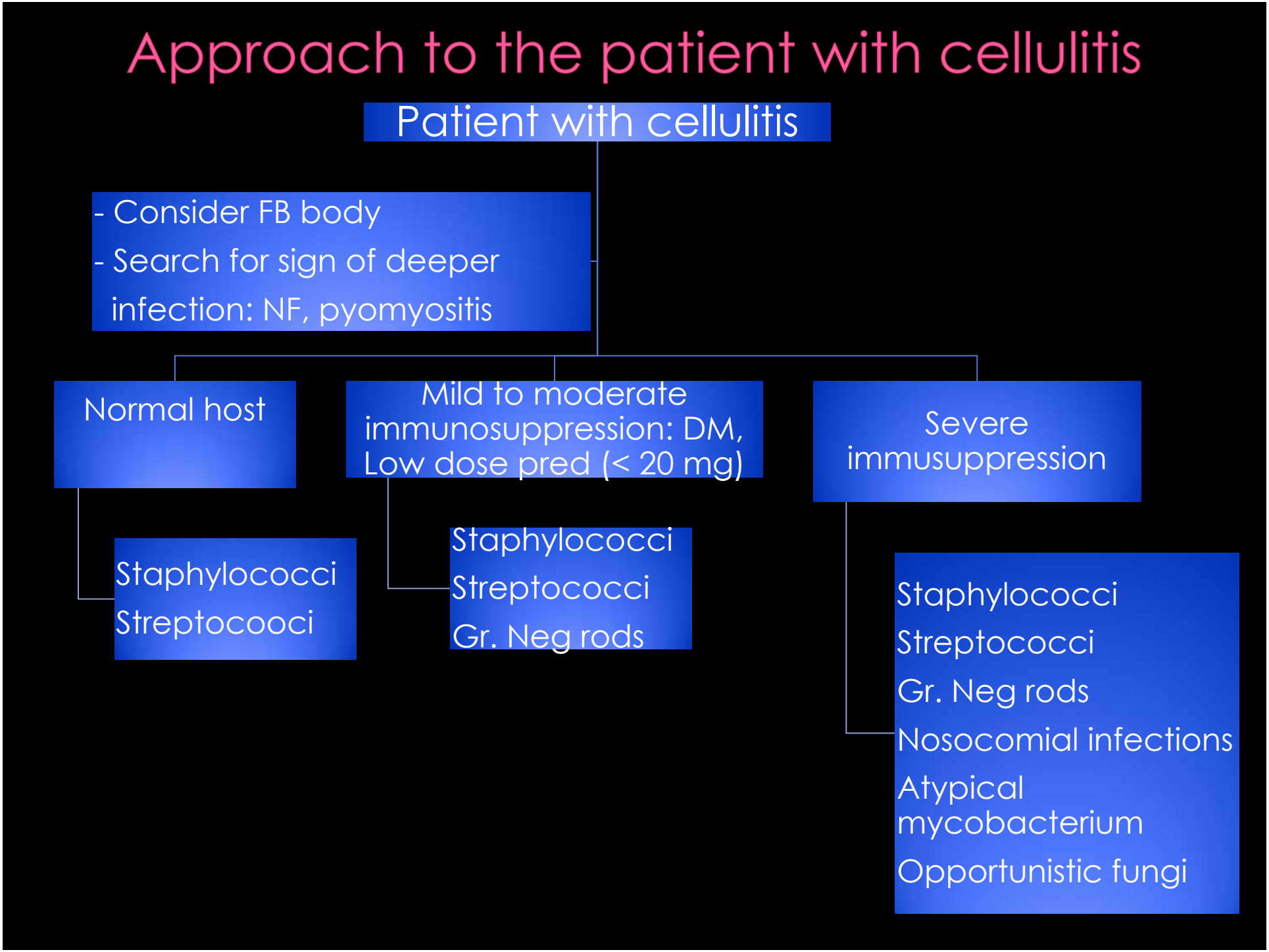


Cellulitis

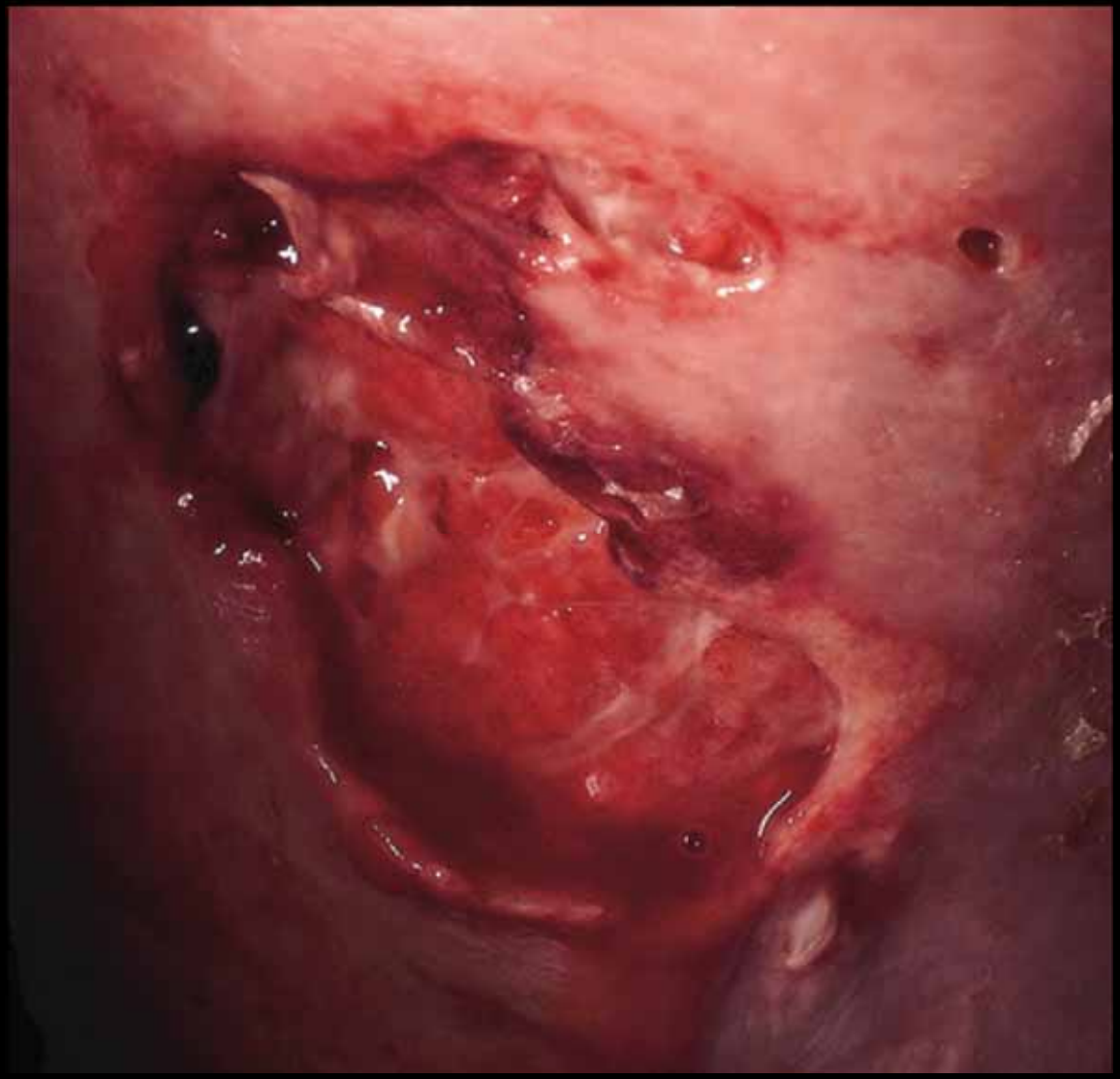




Approach to the patient with cellulitis

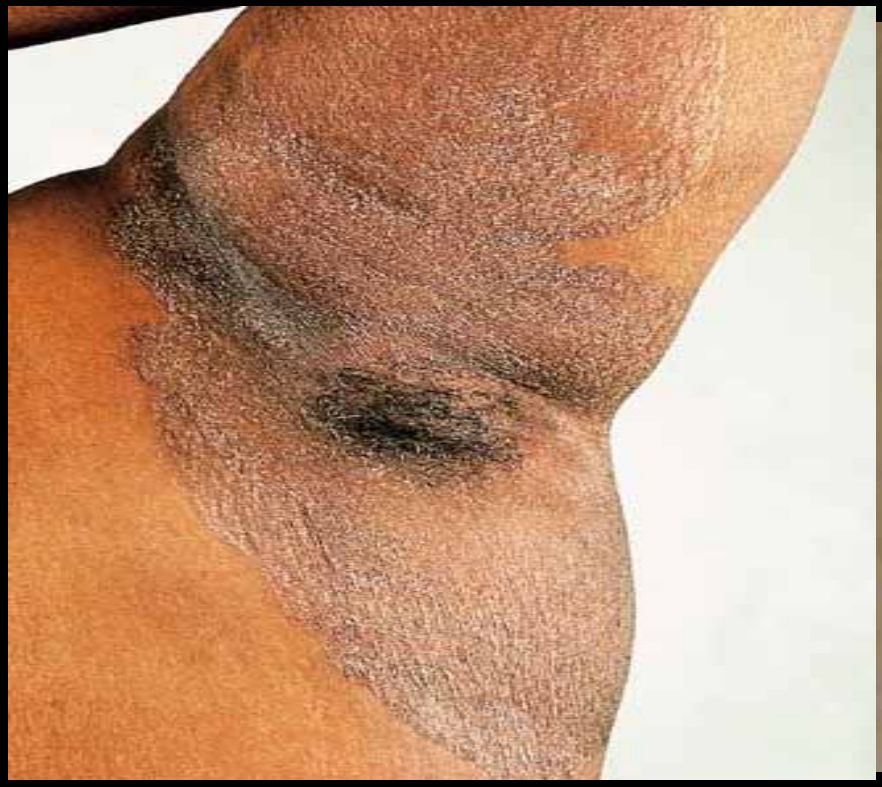


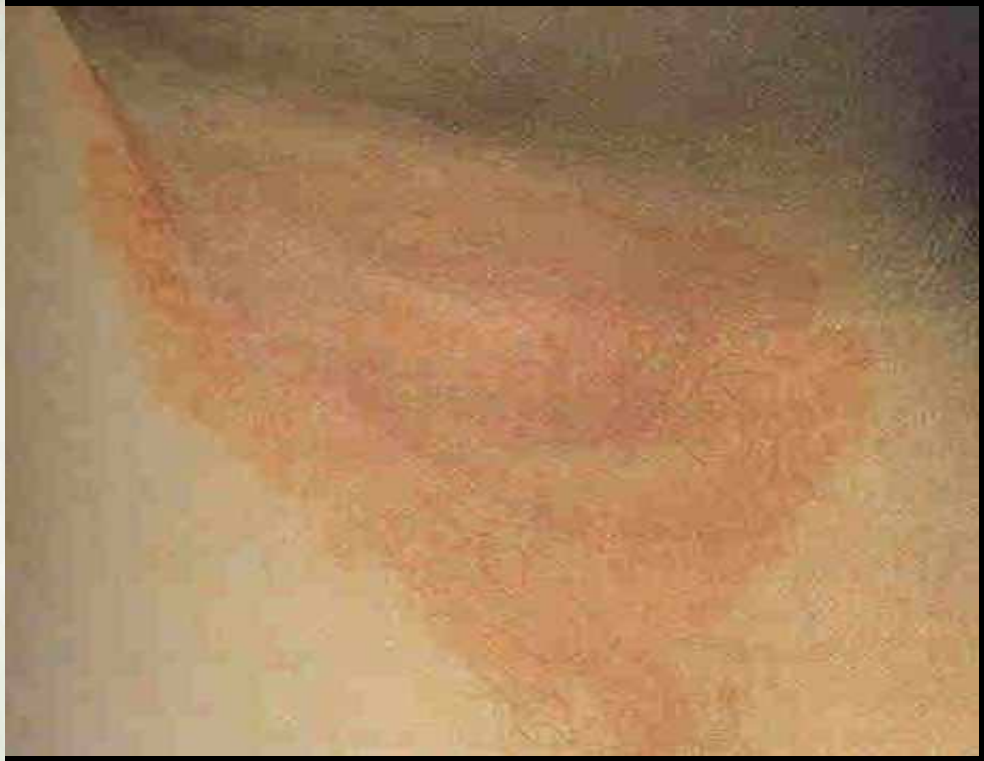
Necrotizing fasciitis



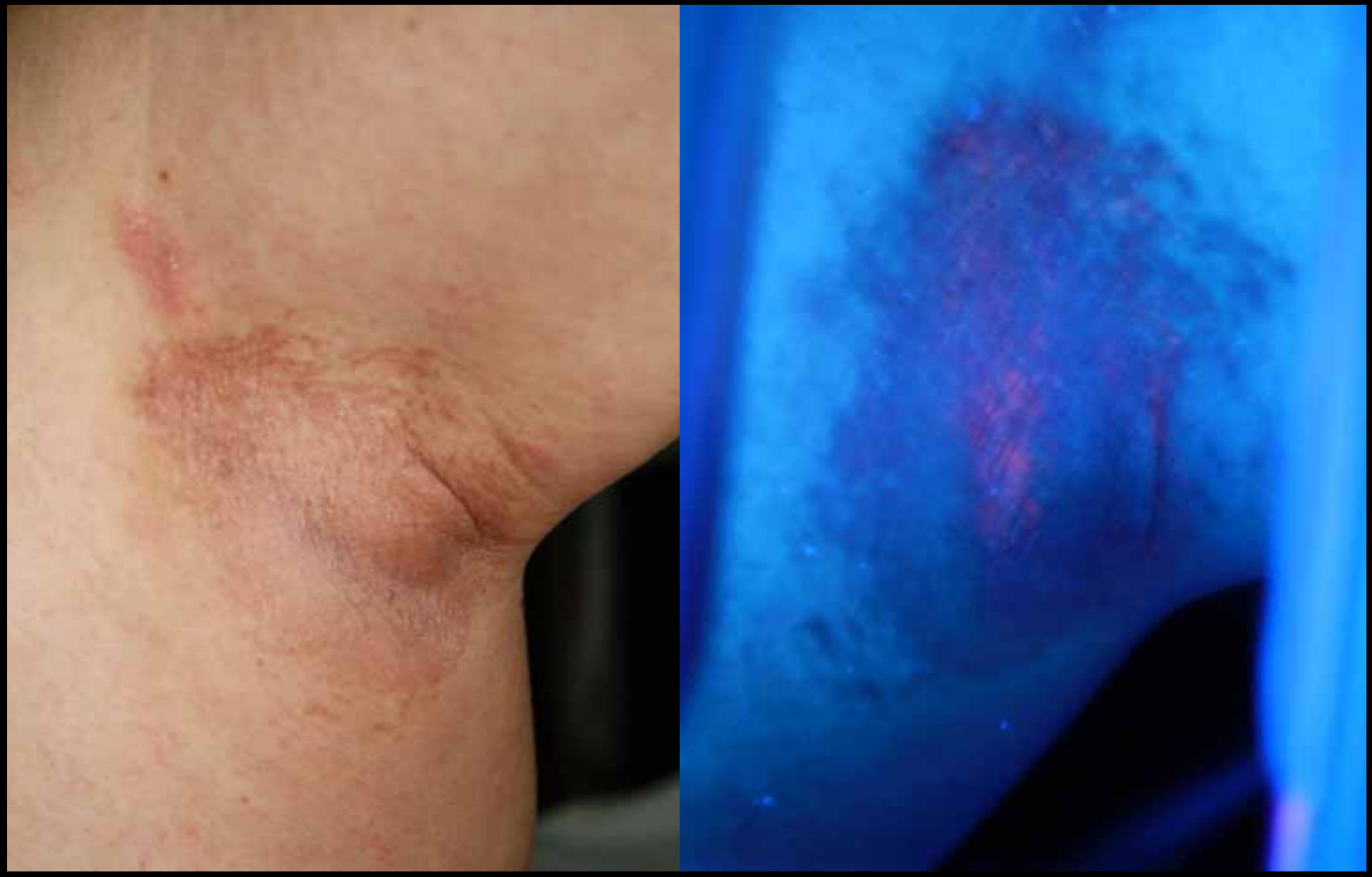
Corynebacterial infection

Erythrasma



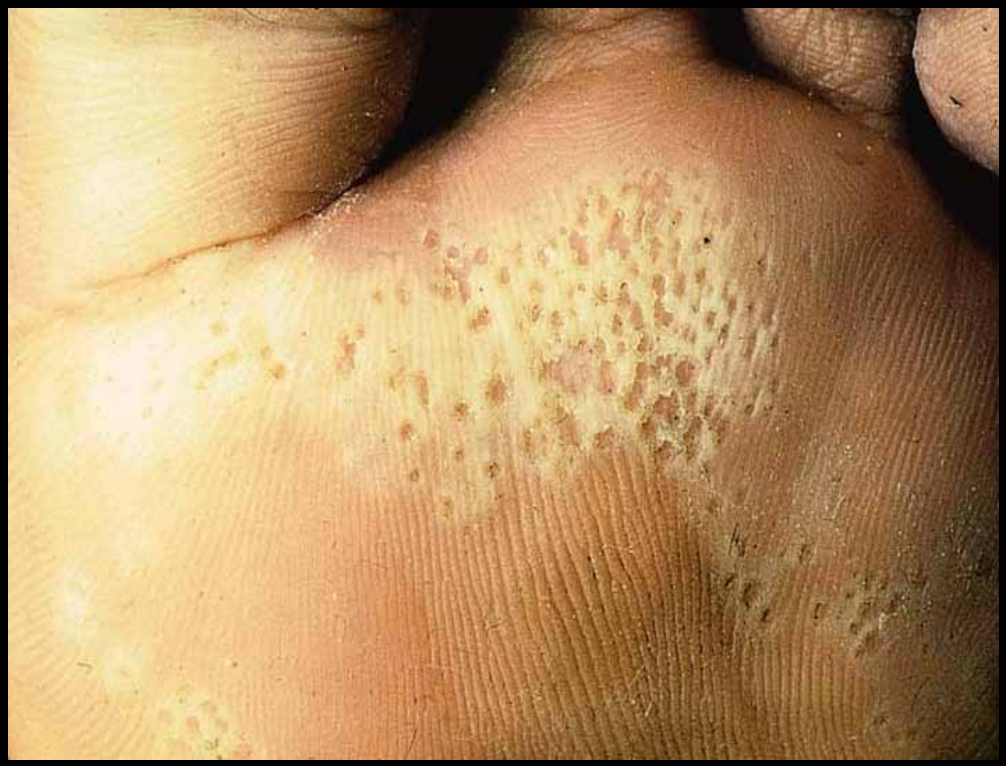


Corynebacterium minutissimum



coral red by Coproporphyrin III

Pitted keratolysis

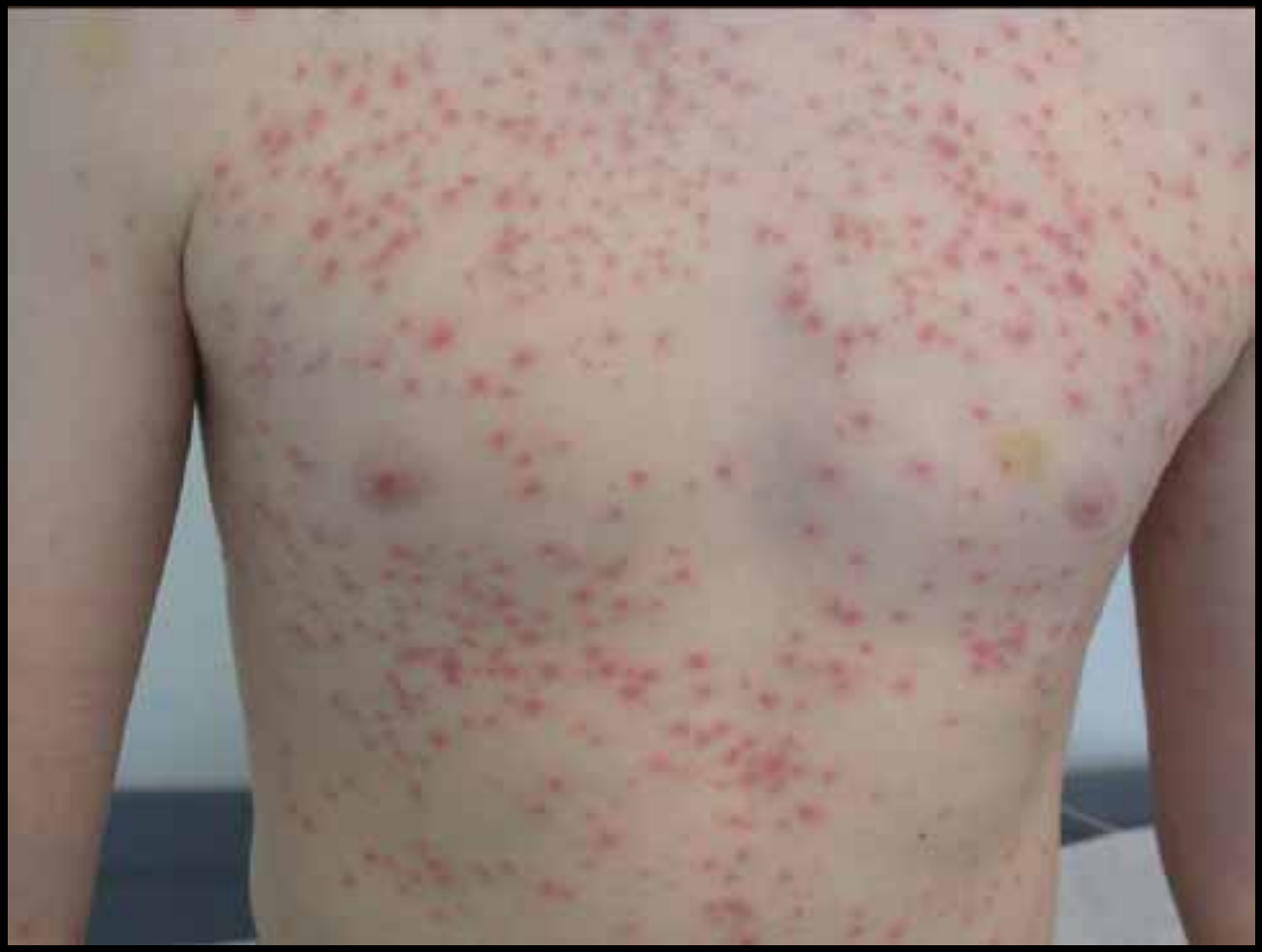


Micrococcus sedentarius, Corynebacterium species, Dermatophilus congolensis, Actinomycosis spp., Streptococcus spp

Rx : 5% BP ,topical clindamycin ,erythromycin, 20% aluminium chloride

Pseudomanas infection

Pseudomanas infection



Hot tub folliculitis

Green nail

Fungal infection

Cutaneous fungal infections

- Divided into 3 types

ORGANIZATION OF CUTANEOUS MYCOSES	
Superficial	Involve stratum corneum, hair and nails
Subcutaneous	Involve dermis or subcutaneous tissue Often due to implantation
Systemic ("deep") "True" pathogens	Involve dermis or subcutaneous tissue. Skin involvement usually reflects hematogenous spread or extension from underlying structures
Opportunistic	Primary or secondary skin lesions in immunocompromised hosts

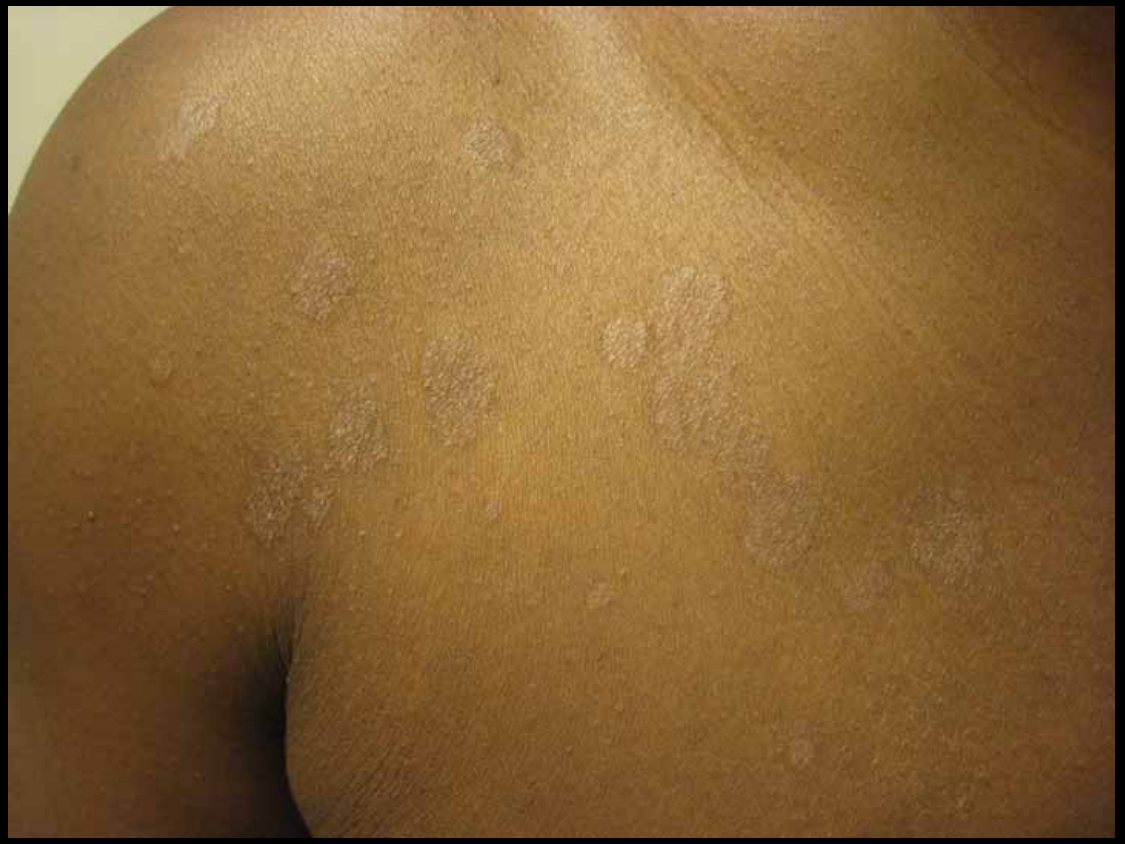
Superficial mycosis

- Classified into 2 types

SUPERFICIAL MYCOSES OF THE SKIN		
	Cutaneous disorder	Pathogens
Minimal. If any, inflammation	Pityriasis(tinea) versicolor Tinea nigra Black piedra White piedra	Malassezia furfur, M. globosa Hortaea werneckii Piedraia hortae Trichosporon beigelii
Inflammatory response common	Tinea capitis, barbae, faciei, corporis, cruris, manuum, pedis Cutaneous candidiasis	Trichophyton, Microsporum, Epidermophyton spp. Candida albicans, other Candida spp.

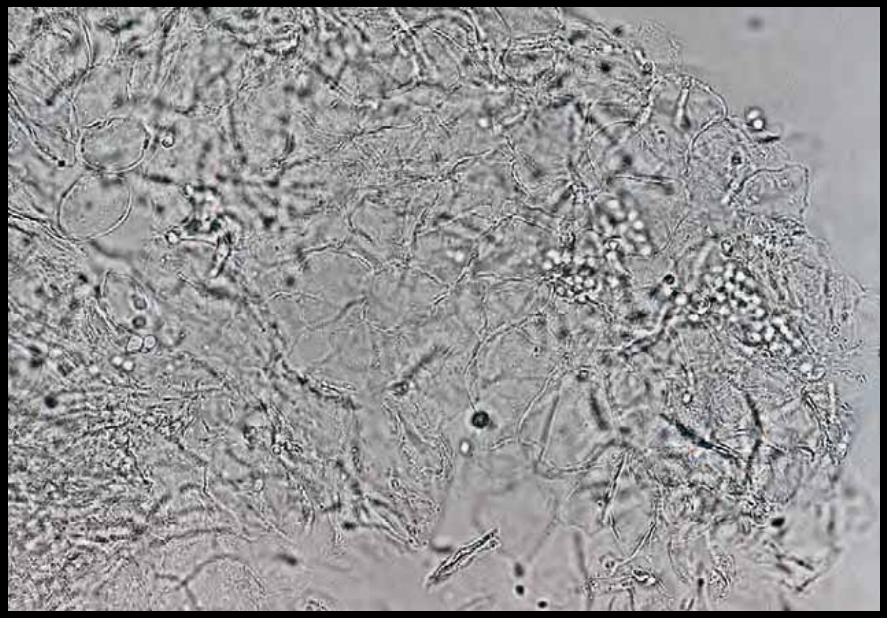
Case 1

A 35 year-old man with 2 week history of skin lesion on trunk

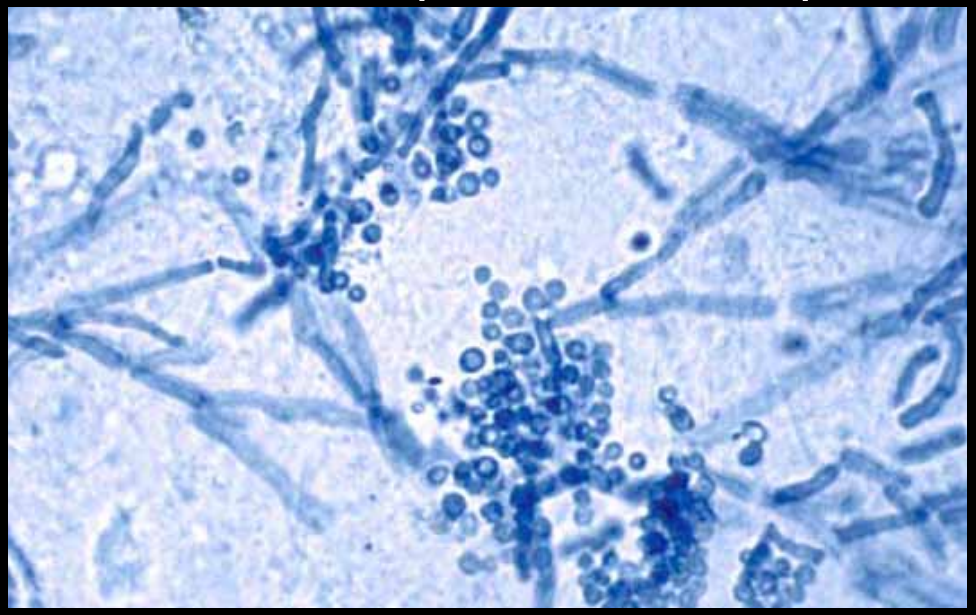


Tinea versicolor

KOH



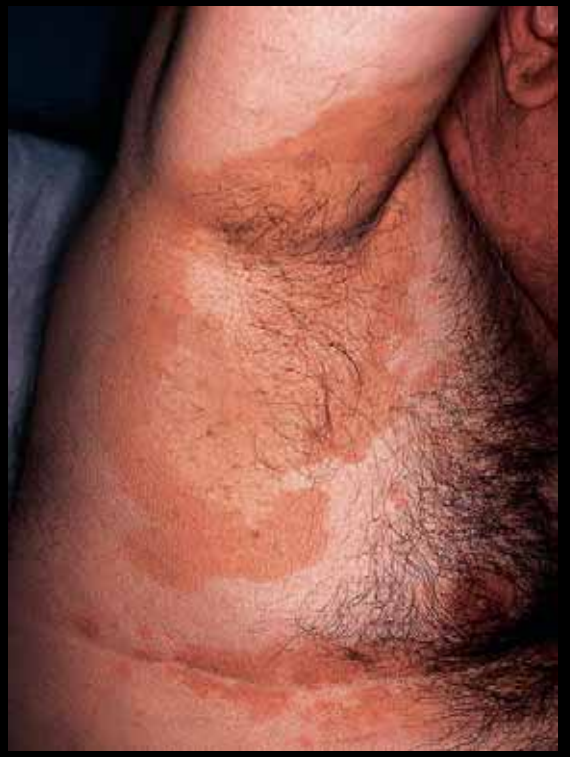
Scottape technique



Spagetti and meatball

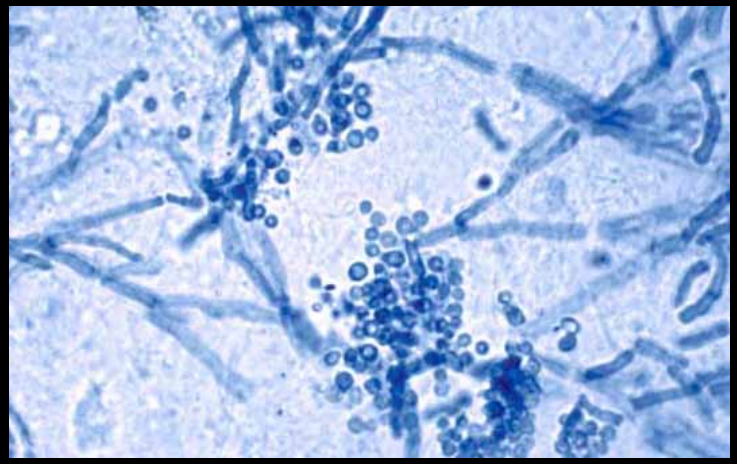
Pityriasis (Tinea) Versicolor

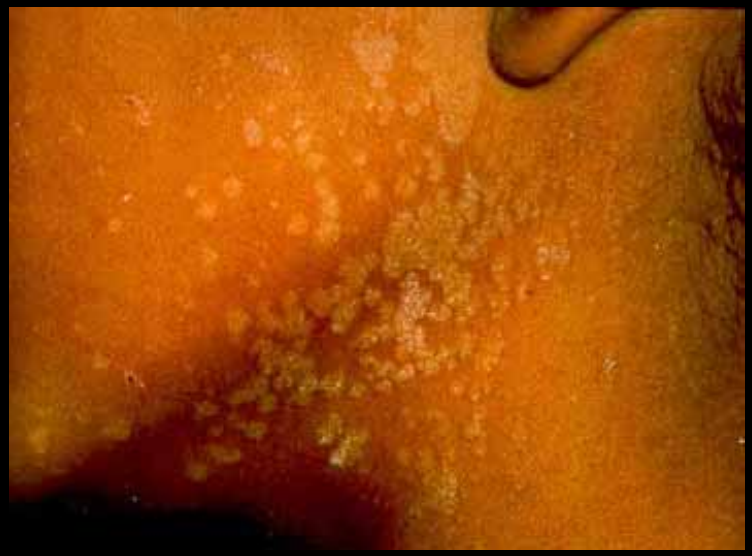
- Causative organism: Malassezia sp. (furfur, globosa)
- Epidemiology: Worldwide, + humid, moist, warm
- Clinical features
  - Multiple oval to round patches or thin plaques with mild, fine scale
  - Favored sites: seborrheic area (upper trunk, shoulder)
  - Less frequently, on face (esp. children), scalp, anticubital fossa
  - The most common colors are brown (hyperpigmanted) and whitish-tan (hypopigmented), occasionally pink color (inflammation)



Pityriasis (Tinea) Versicolor

- Wood lamp: Bright yellow
- KOH and scottape technique
  - Yeast form and short mycelium
- Treatment:
  - Topical: selenium sulfide, ketoconazole
  - Systemic: azoles

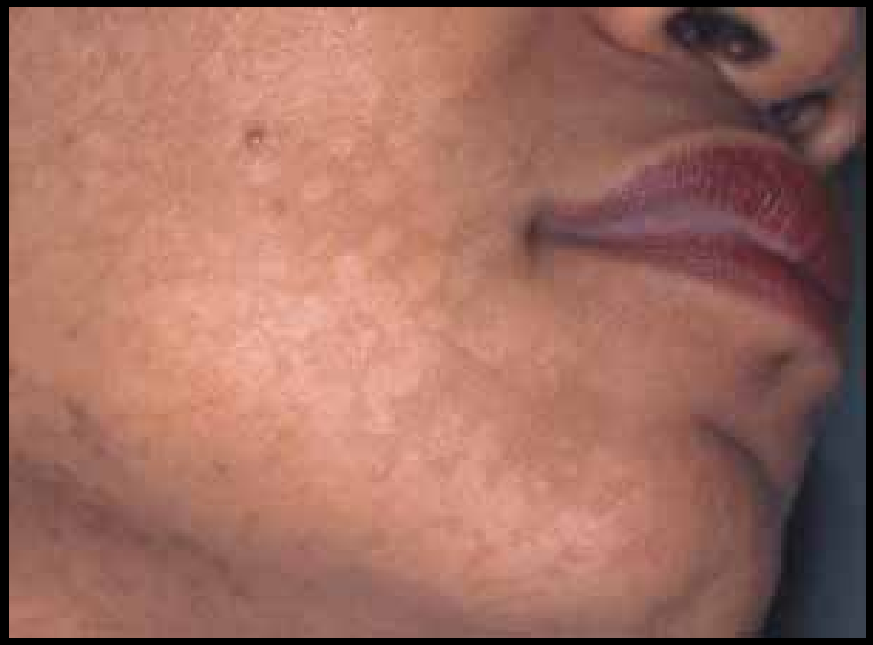




Treatment of Tinea versicolor

- Systemic azoles:
  - ketoconazole: 400mg single dose
  - Fluconazole: 400mg single dose, or 300mg single dose with possible second dose in 1 wk
  - Itraconazole: 200mg/Dx1wk
- Patients at high risk for recurrence may be helped by
  - Using ketoconazole shampoo once weekly as a body cleanser.
  - Once-monthly dosing of oral ketoconazole (400 mg), fluconazole (300 mg) or itraconazole (400 mg).

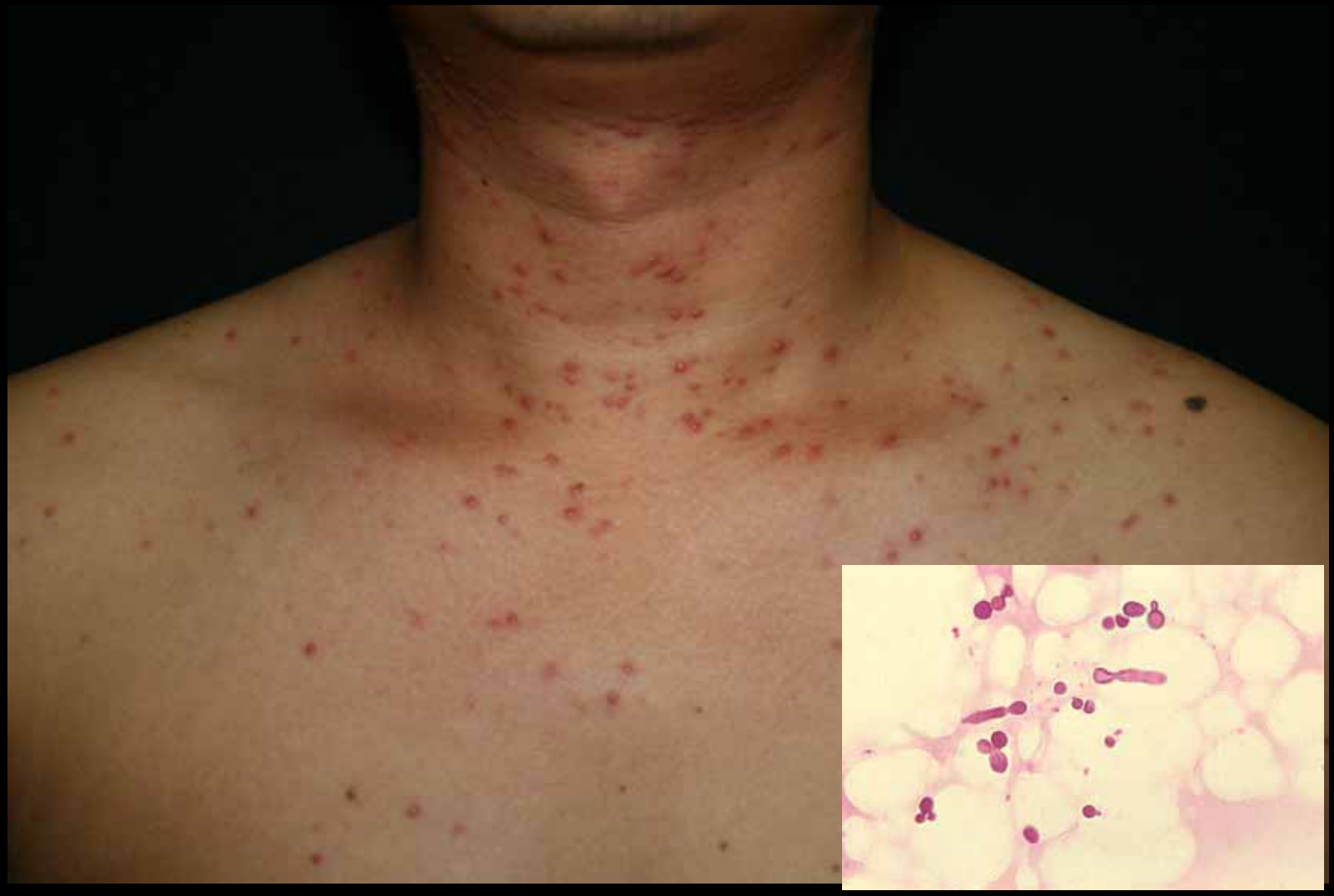
This topical medication is derived from the organism that causes this skin lightening condition.



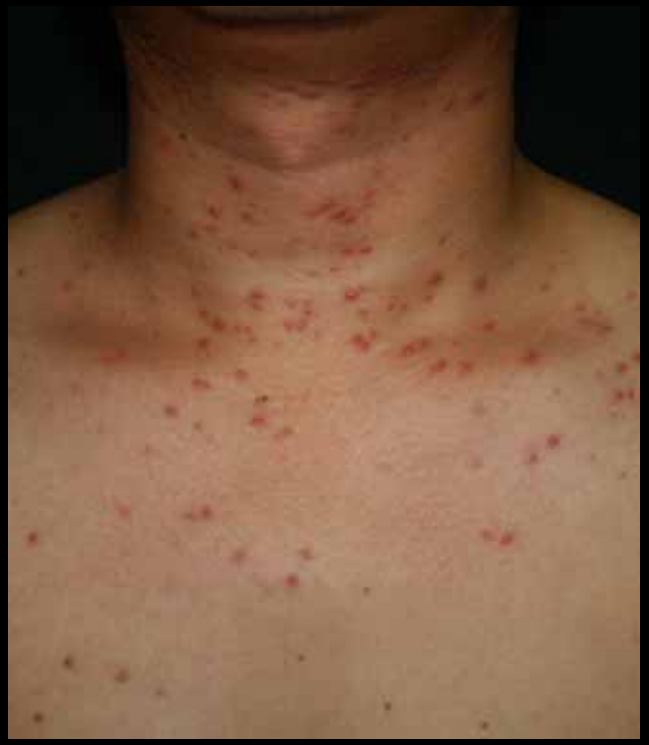
A. Hydroquinone
B. Azelaic acid
C. Hydrocortisone
D. Tretinoin
E. Mequinol

Decreased pigmentation may be secondary to the inhibitory effects of dicarboxylic acids on melanocytes (these acids result from metabolism of surface lipids by the yeast)

Pityrosporum folliculitis



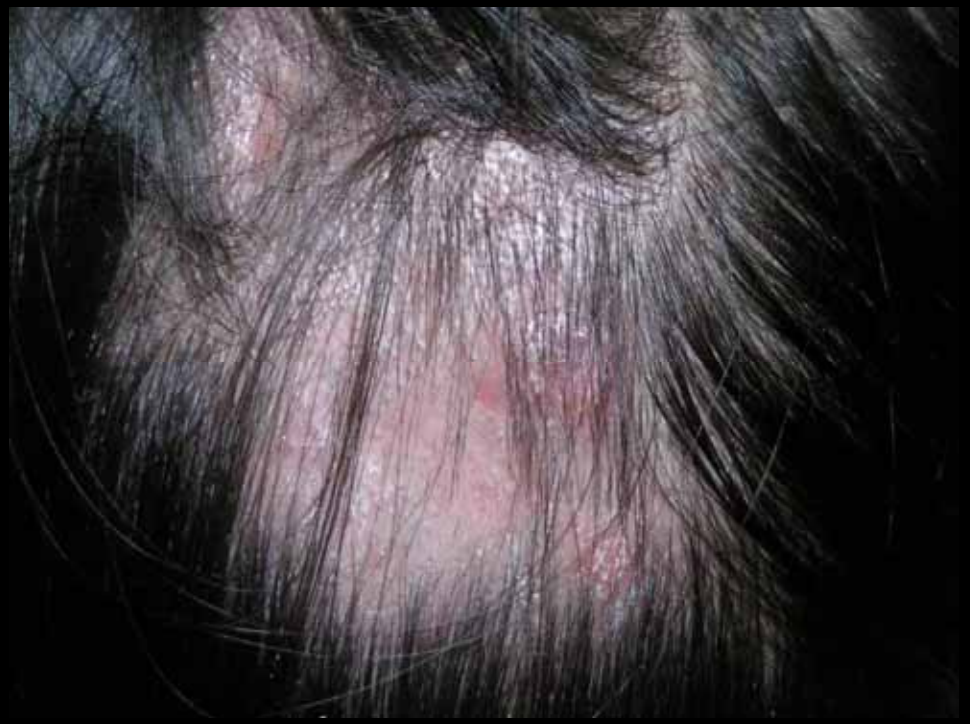
Pityrosporum folliculitis



- Most commonly seen in young women
- Characterized by pruritic, monomorphic follicular papules and pustules on the upper trunk, arms, neck and, occasionally face.
- Due to excessive growth of M. furfur and M. globosa within the follicle.
- Only yeast forms are observed

Case 2

- A 7 year-old girl with Progressive hair loss for 3 months



What is your diagnosis?

Tinea capitis
(Inflammatory type)

What is (are) your further management ?

1

2

3

4

⊙ KOH (scrapping scale from scalp) *

Branching septate hyphae

◉ Wood lamp

What is (are) the causative organism ? *

Green fluorescence

Yellow green

Ectothrix

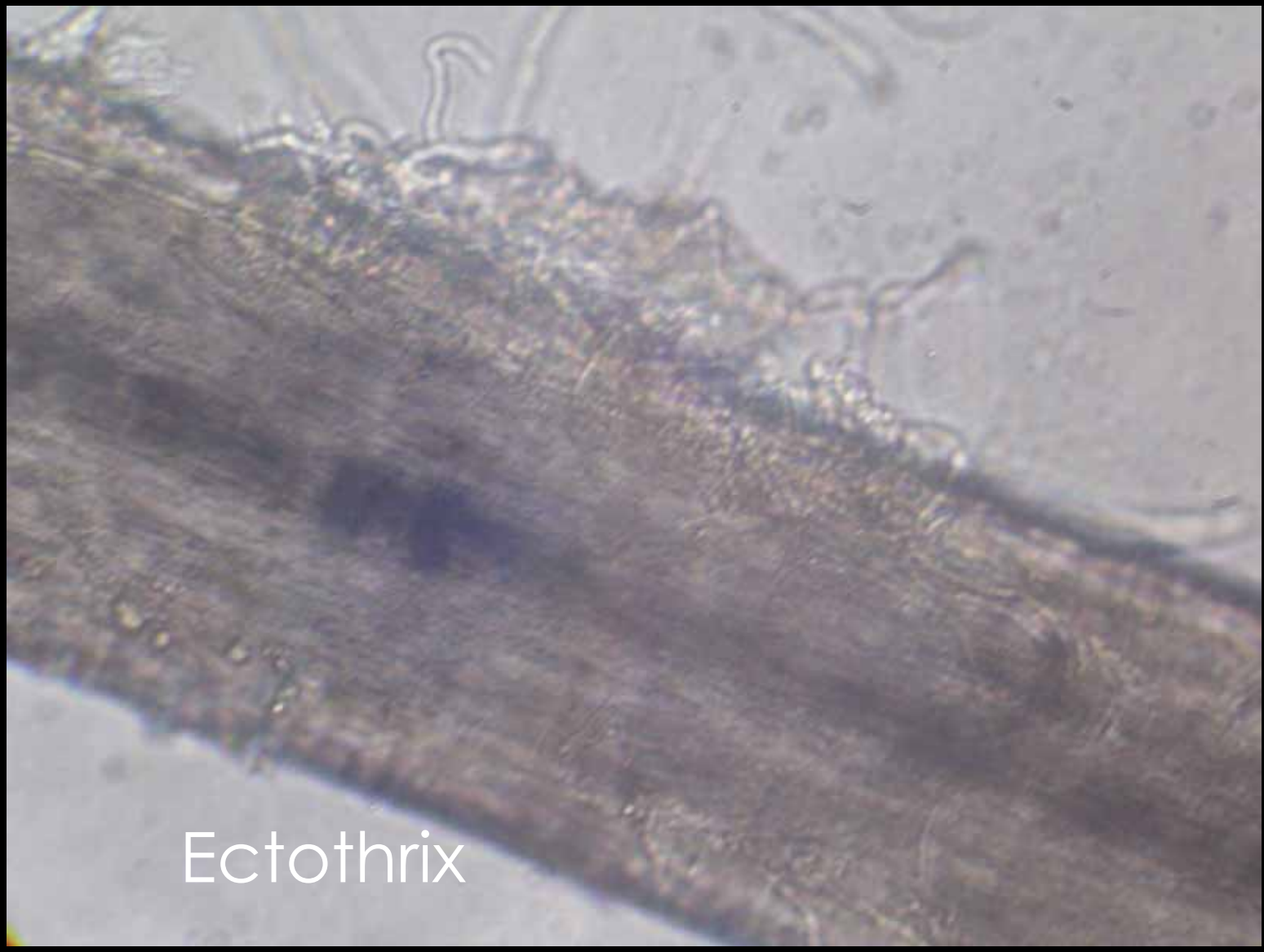
M. audouinii
M. canis
M. ferrugineum

Dull green

Endothrix

T schoenleinii

- **Microscopic examination from hair** *



Fungal culture *

M. canis

Dermatophytosis

- Dermatophytes – fungus those has ability to form molecular attachments to keratin and use it as a source of nutrients
- In 1934, Emmon classified to 3 genus
  - Epidermophyton, microsporum, Trichophyton
- Classification by ecology
  - Geographic organisms
    - Grow in soil and only sporadically infect humans
    - Usually inflammatory type
    - M. gypseum is the most common geophillic pathogen

Dermatophytosis

- Zoophilic species
  - Found on animals but are also transmitted to human sporadically
- Anthropophilic species
  - Humans as a host
  - Often epidermic in nature
  - Human to human transmission by direct contact or fomite
  - Can cause inflammatory or noninflammatory type depend on virulence of organism and host status

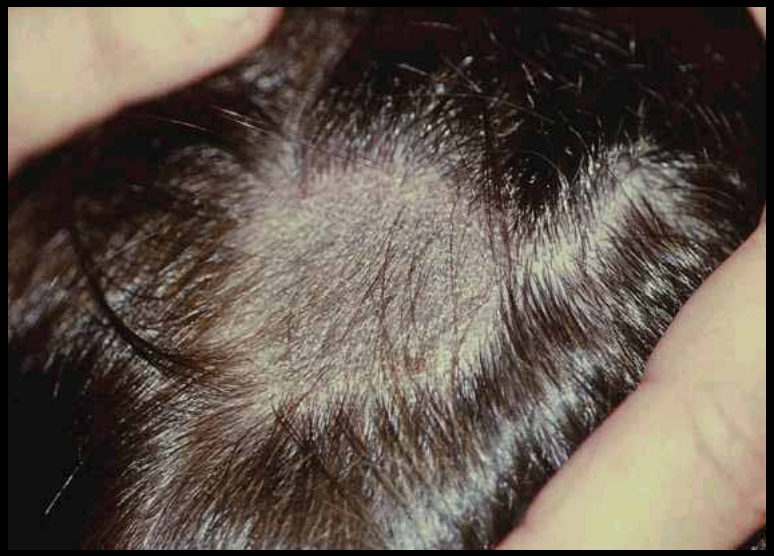
Tinea capitis

- Caused by any dermatophytes except

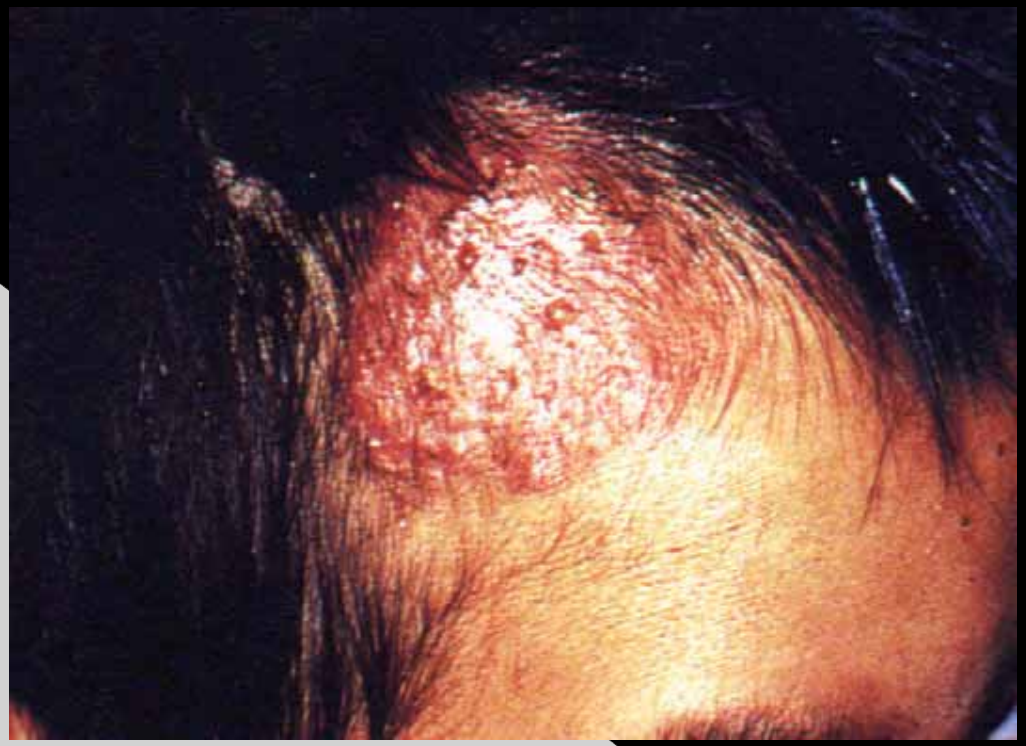
  E. floccosum, T. mentagrophyte

- Epidermiology
  - Unknown incidence
  - Most common in chidren 4-13 year
  - Most common causative agents
    - Worldwide: M. Canis
    - USA: T. tonsulans

The 4 Clinical manifestations of Tinea capitis

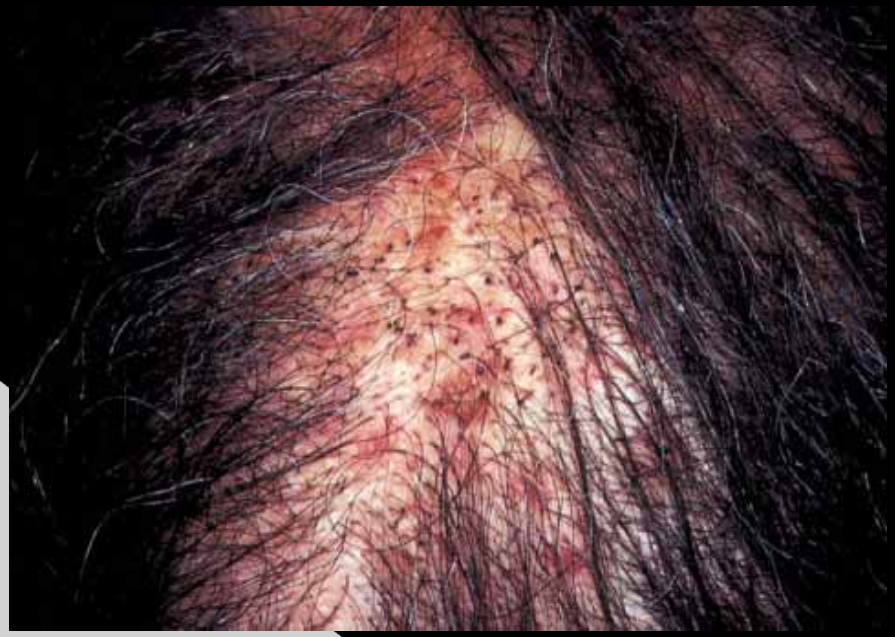


1. Noninflammatory, human or epidermic type
   - Most common – anthropophilic ectothrix
     ex. *M. audouinii, M. ferrugineum*
   - Clinical manifestation
     - Begin as a small erythematous papule surrouding a hair shaft with scaling.
     - Affected hair turn gray , lusterless , and break off.
     - One or more well demarcated scaling patch on the occiput or posterior neck.



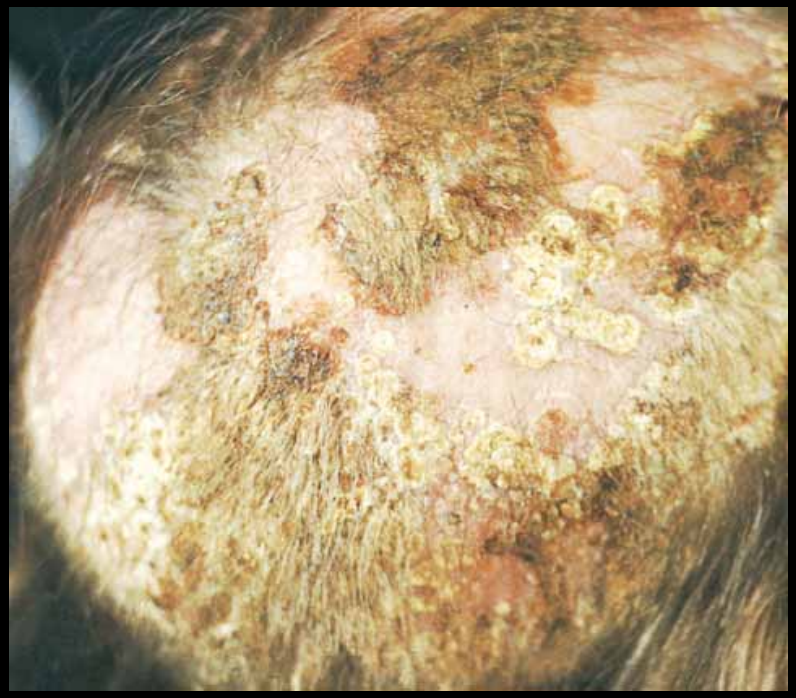
2. Inflammatory type

- Common in zoophilic or geophillic pathogen
- Most common organisms – *M. canis and M. gypsium*
- Clinical manifestation
  - Pustular folliculitis to kerion
  - Can cause scarring alopecia
  - Usually pruritic, may associated with pain, cervical, lymphadenopathy and fever



3. “ Black dot ” tinea capitis

- Caused by anthropophilic endothrix
- *T. tonsulans, T. violaceum*
- Clinical manifestation
  - Hair broken at the level of the scalp leave behind group of black dots
  - Diffuse scaling is usually present but inflammation varies (minimal inflam. to kerion)
  - Affected area – usually multiple or polygonal with poorly demarcated, finger-like margin.



4. Tinea favosa or favus (Latin, “honeycomb”)

- **The most common cause – *T.schoenleinii***
- Usually acquired before adolescence and extent to adulthood
- Associated with *malnutrition and poorly hygiene*
- Now seen almost exclusively in *Africa, the Middle East, and parts of South America*
- Characterized by thick yellow crust (scutula) within hair follicle which lead to scarring alopecia

Lab investigation

1.Skin scraping in KOH

Septate and branching hyphae

2.Microscopic examination

Hair plucked examination (low power)

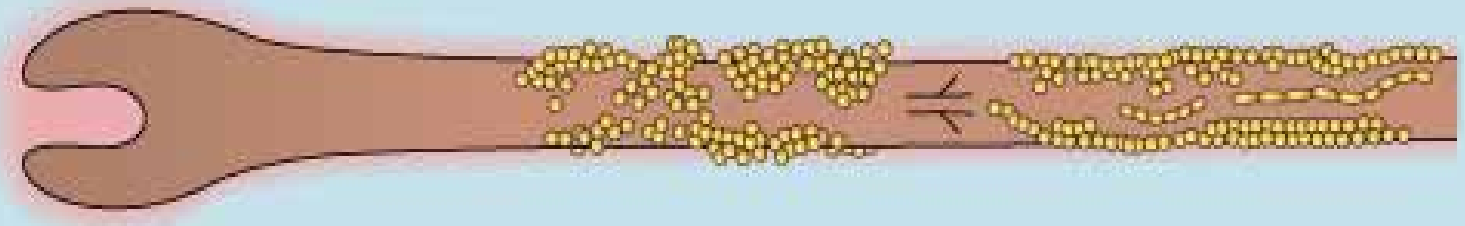
3 possible patterns of infections :

- Ectothrix
- Endothrix
- Favus

THE THREE PATTERNS OF HAIR INVASION AND THE CAUSATIVE DERMATOPHYTES

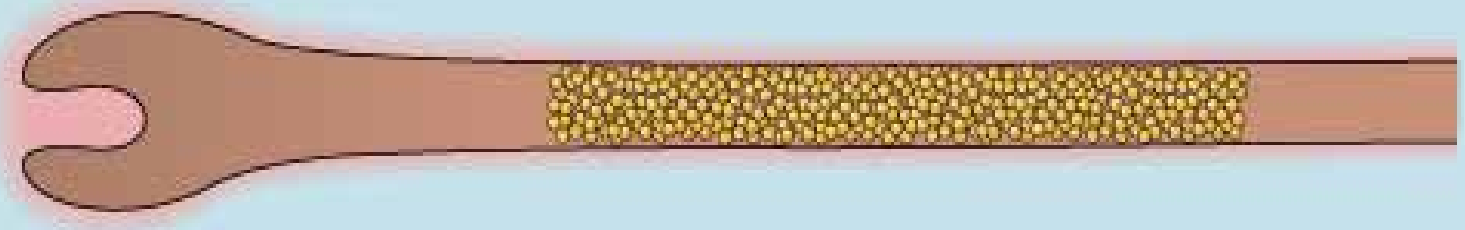
Ectothrix

*M. canis**
*M. audouinii**
*M. ferruginosum**
*M. distortum**
M. gypseum
T. rubrum (occasionally)



Endothrix

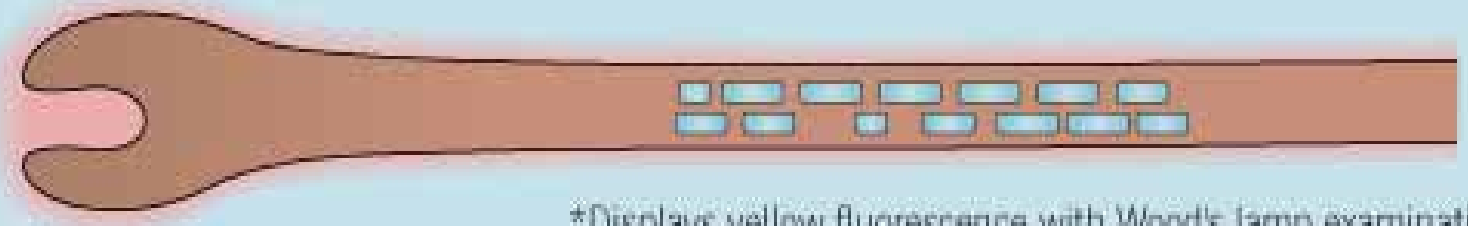
T. tonsurans
T. violaceum
T. soudanense
T. gourvilli
T. yaoundei
T. rubrum (occasionally)



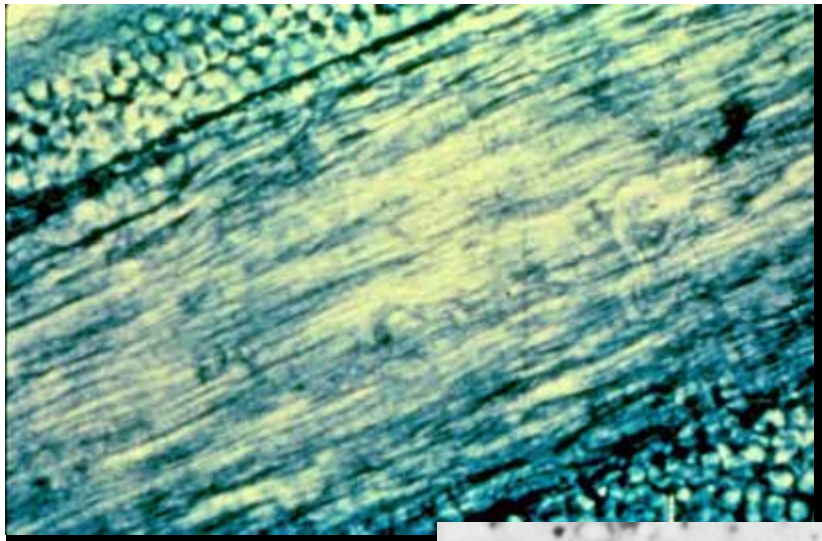
Arthroconidia
Hyphae and air spaces

Favus

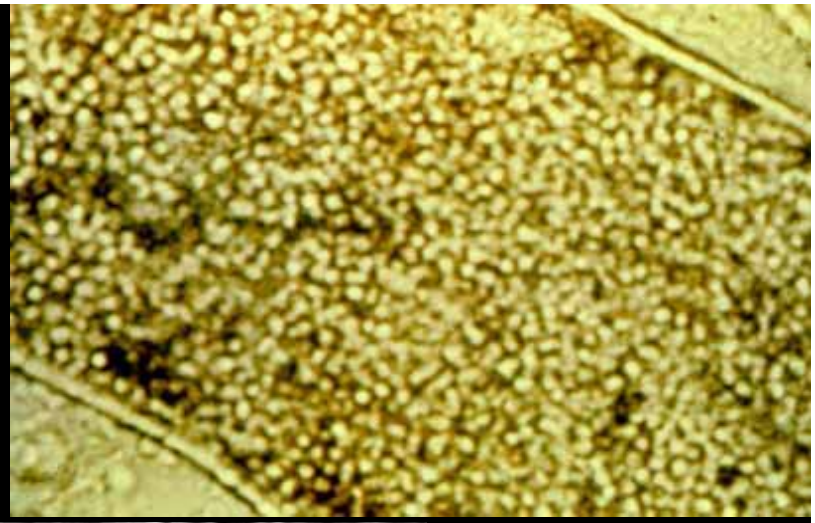
*T. schoenleinii**



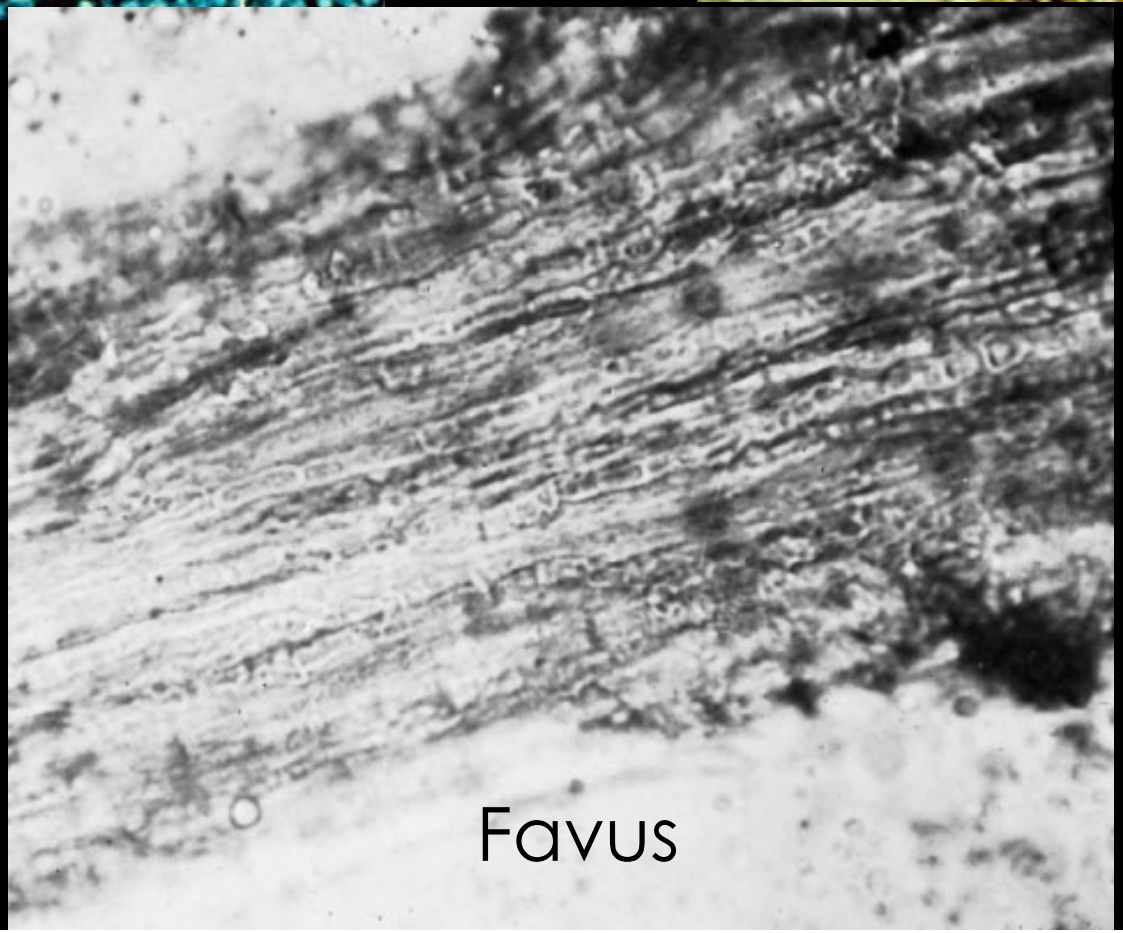
*Displays yellow fluorescence with Wood's lamp examination



Ectothrix

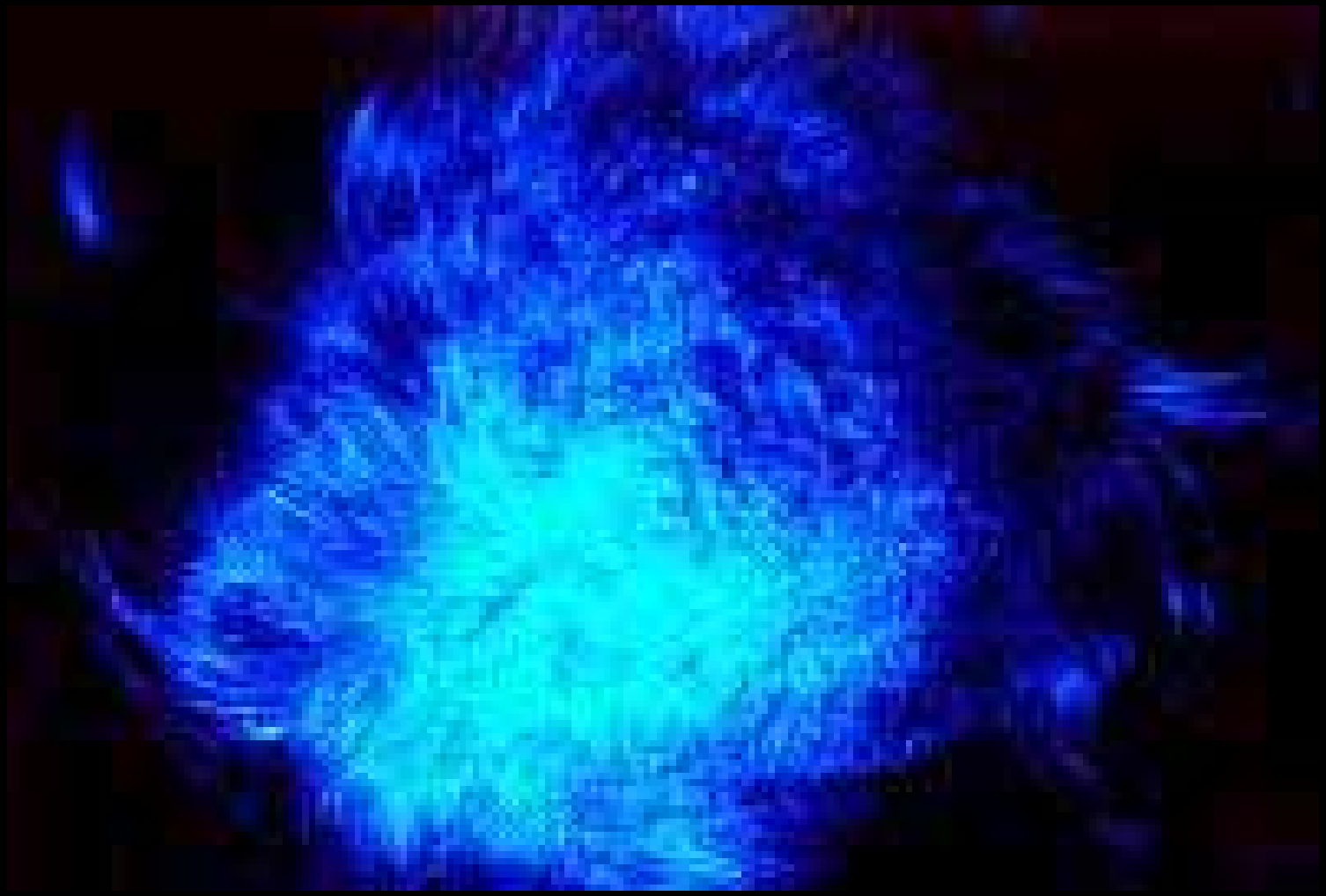


Endothrix



Favus

green fluorescence



4.Culture

- Most common media for isolation of fungus is

Sabouraud's dextrose agar

- The addition of cyclohexamide and chloramphinical is for inhibition of contaminant saprobes and bacteria.

Treatment

Systemic Rx	Griseofulvin (microsized)	Fluconazole	Itraconazole	Terbinafine
Dose	0.5-1g/d 10-25mg/d Fatty meal	6mg/kg/d	3-5mg/kg/d	3-6mg/kg/d
Duration	6-8wks	3-6wks	4-8wks	3-4wks
Disad-vantage	Poor compliance Photosensitivity GI side effect	GI side effect-uncommom, hepatitis-reported	Possible GI upset Peripheral edema Hepatitis (=fluco)	GI side effect Rare hepatitis TCA toxicity (inh cyP2D6)

Adjuvant treatment

- Markedly inflammation – oral steroid
  - Decrease incidence of scarring
  - No evidence for difference in cure rate
  - Relieve pain and swelling
  - Dose 1mg/kg each morning 10-15 days

- Household transmission – prevent by Rx of infected family member or animals and disinfection of environment
  - 2% keto. Shampoo or 2.5% selenium sulfide
  - All household members 3 times/wk

Treatment of this patient

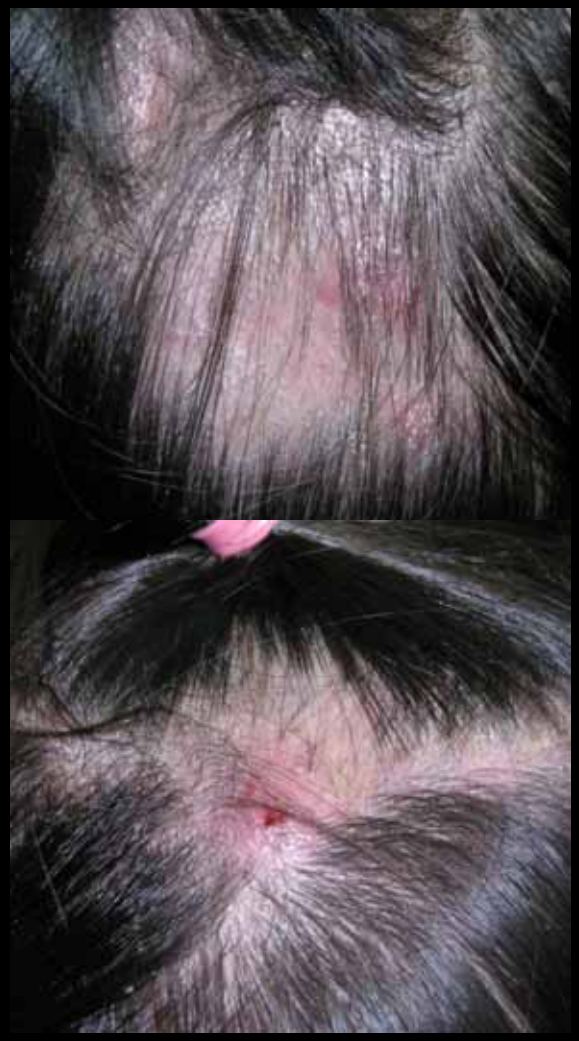
- Griseofulvin(500) 20mg/kg/d
  - 1 tab oral OD with fatty meal

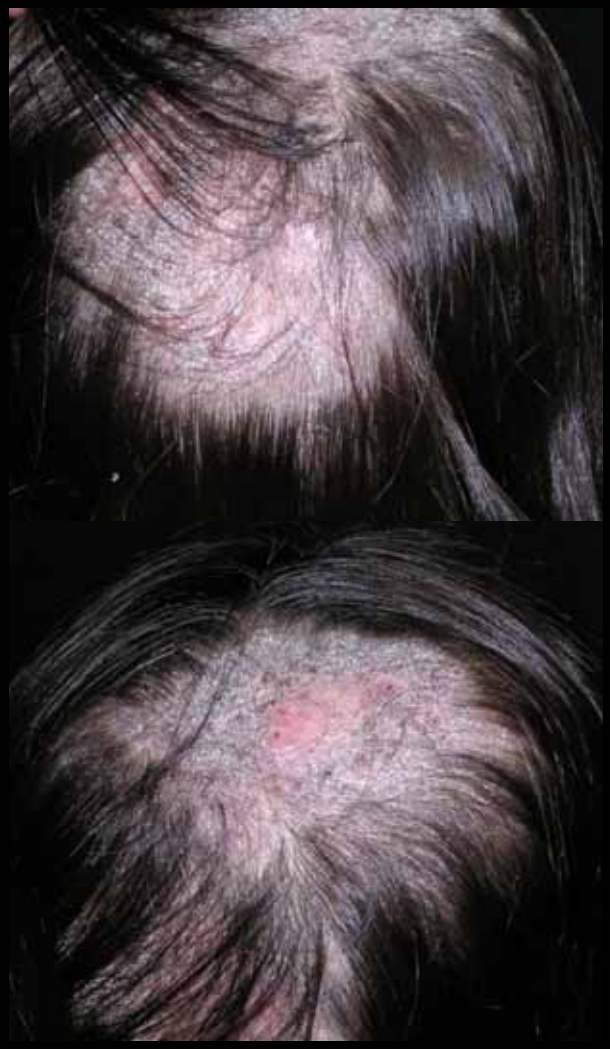
- Applying ketoconazole shampoo for the patient,her families and her 2 dogs

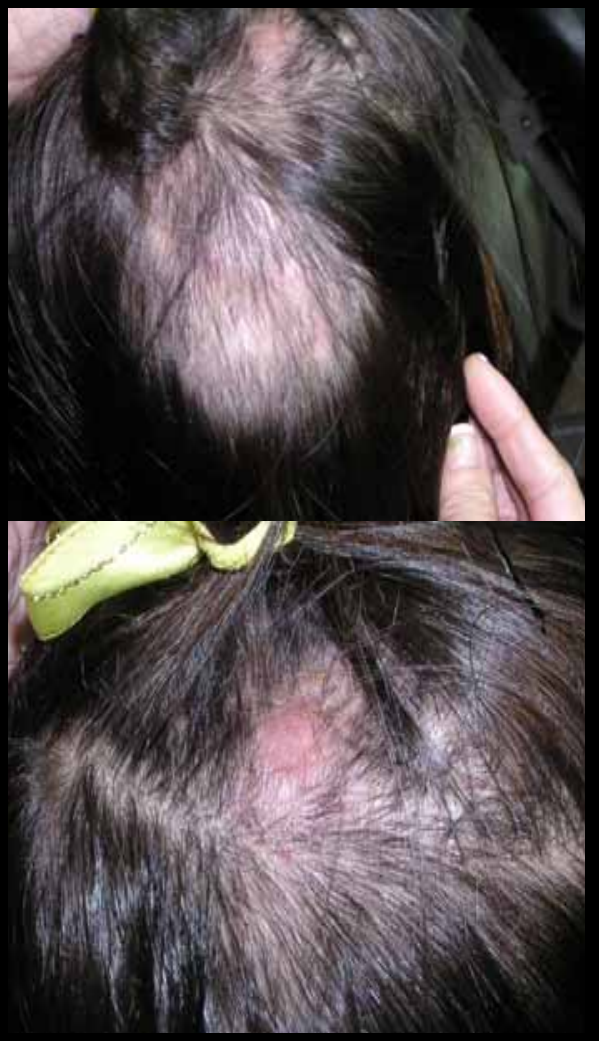
Before

2 WK

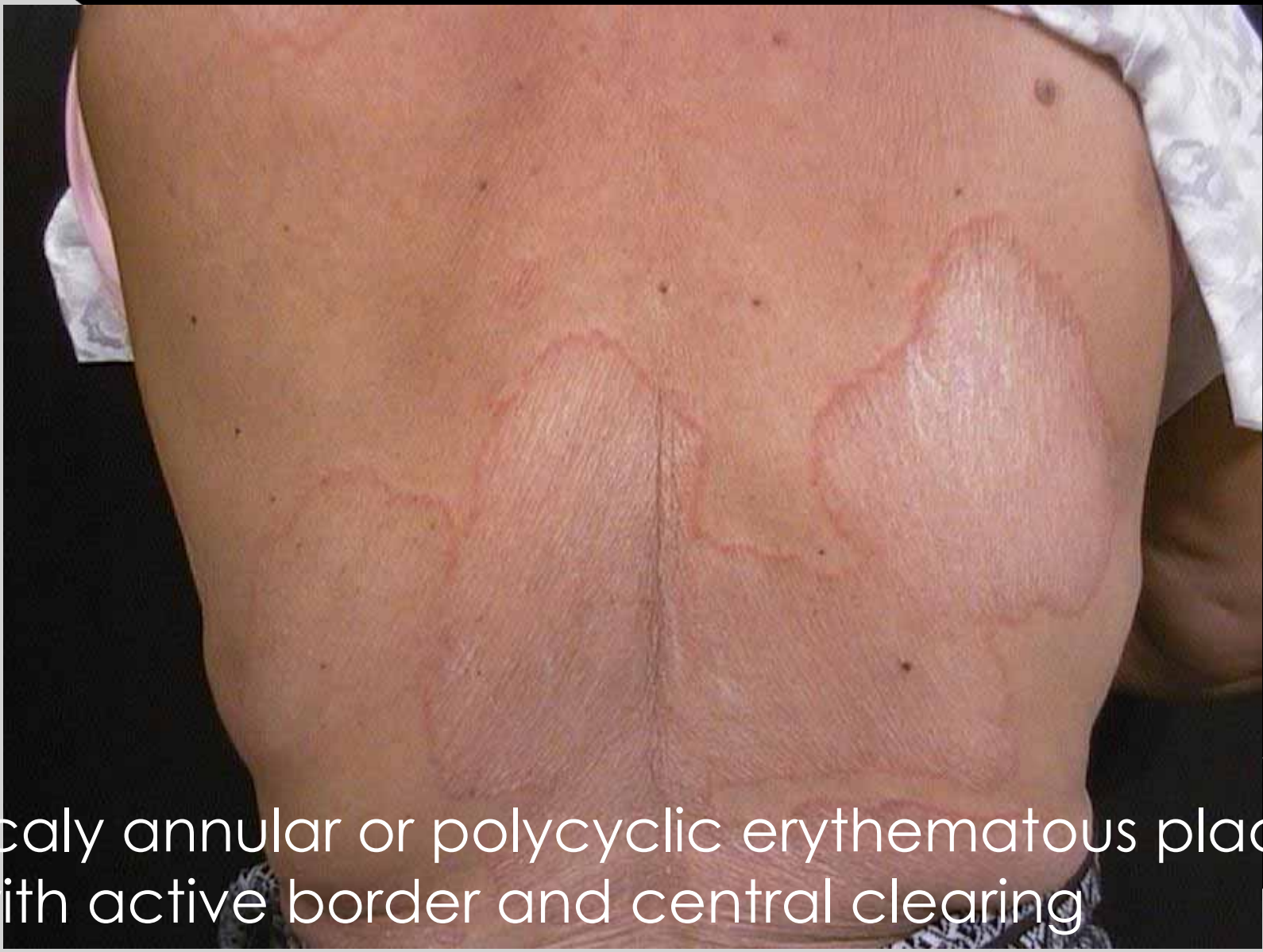
4 WK





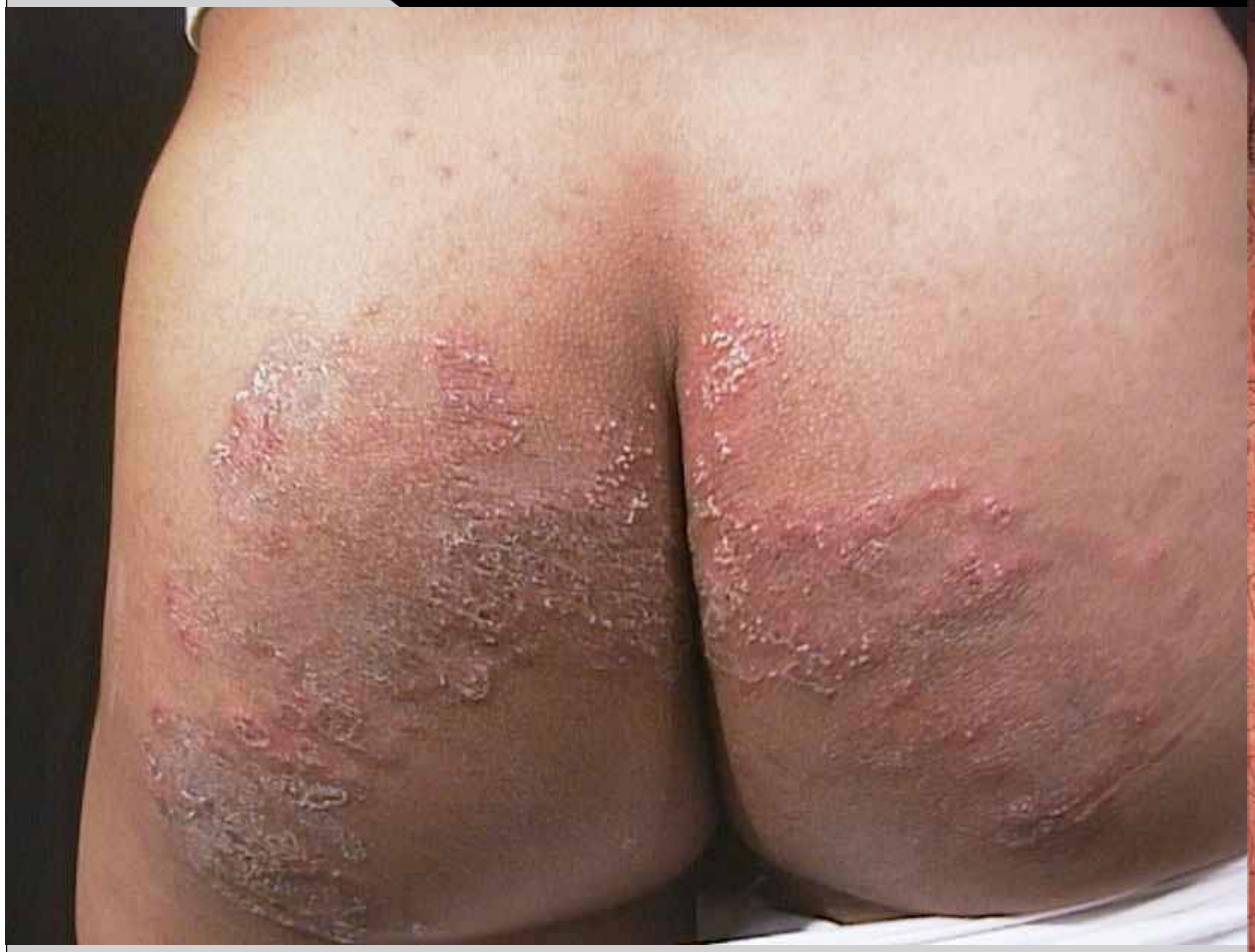


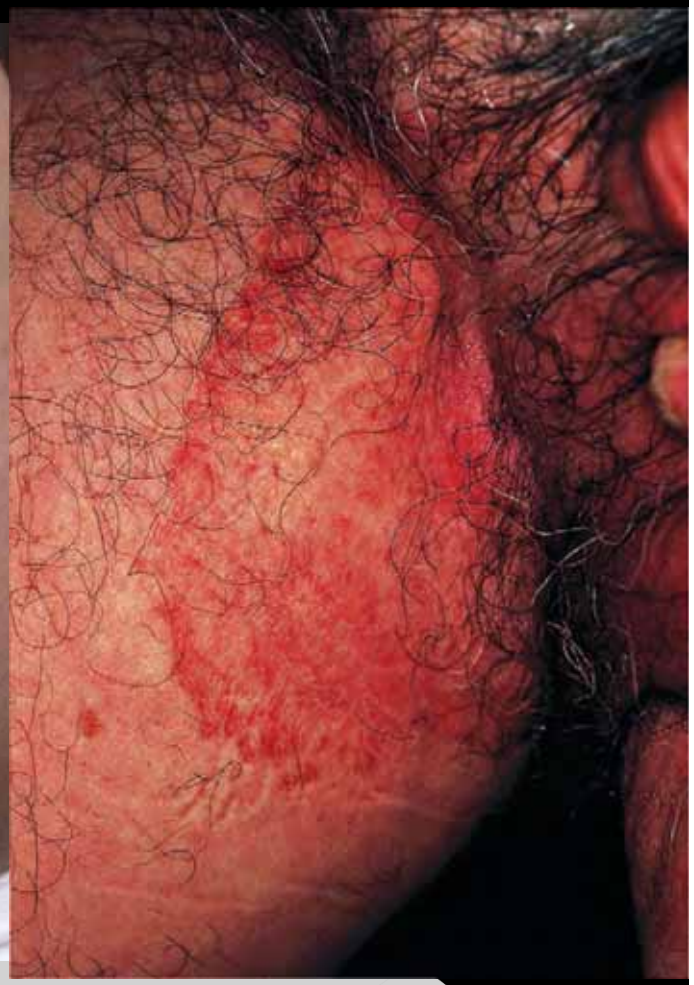
Tinea corporis



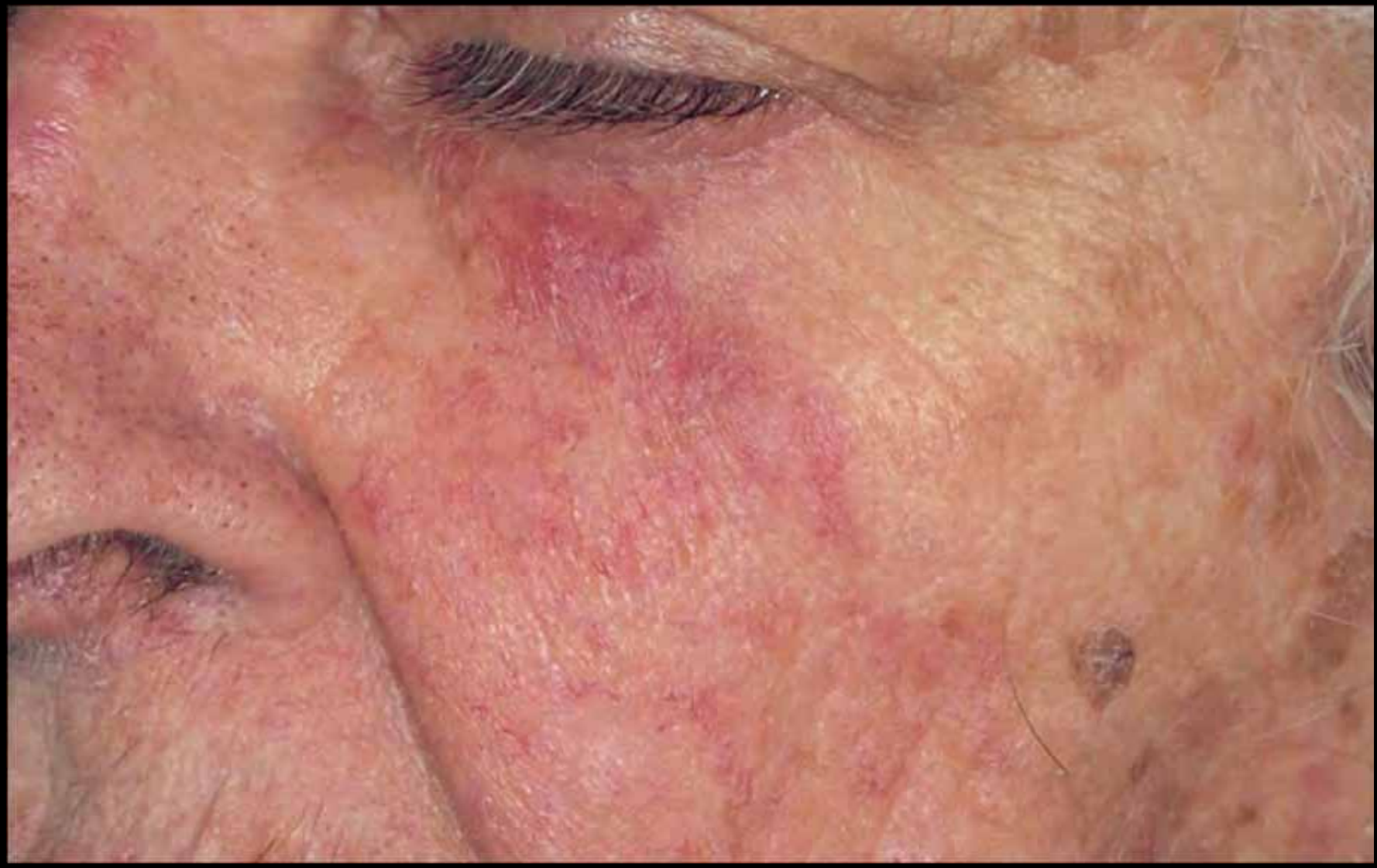
Scaly annular or polycyclic erythematous plaque with active border and central clearing

Tinea cruris

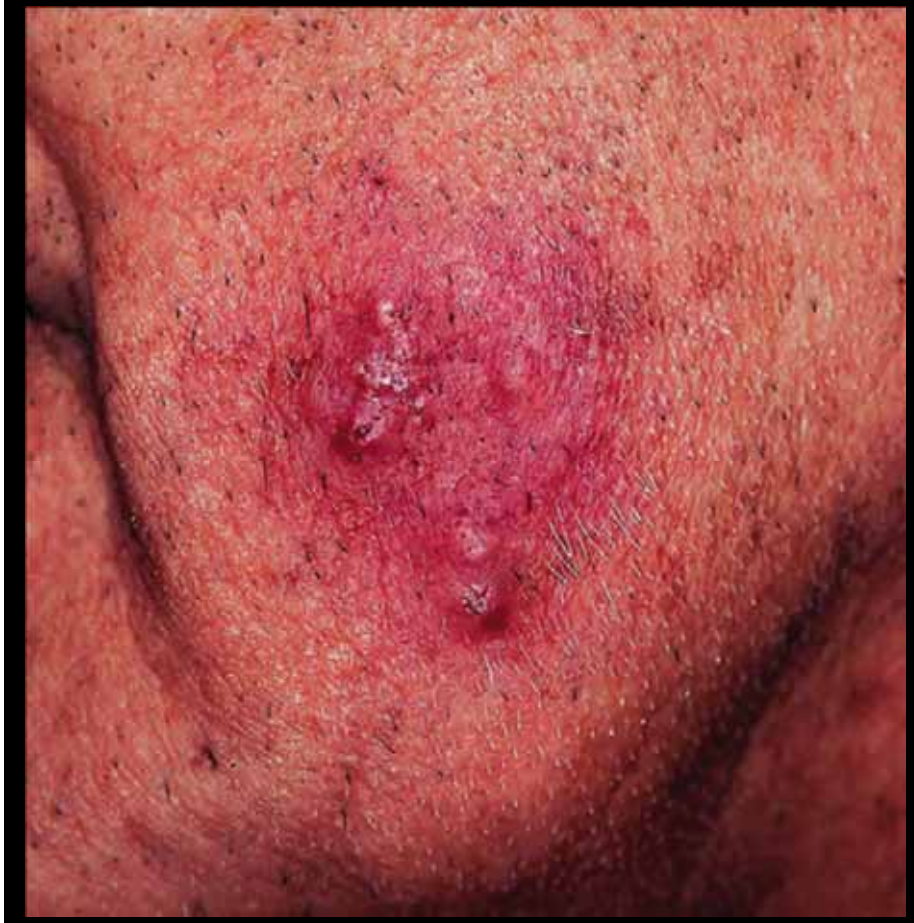


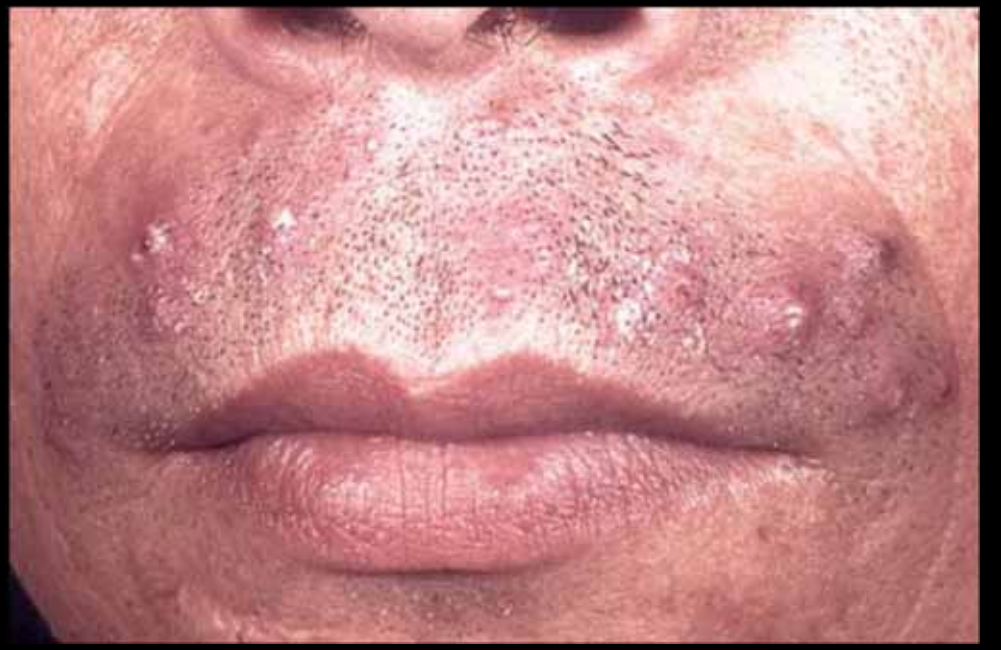


Tinea faciei

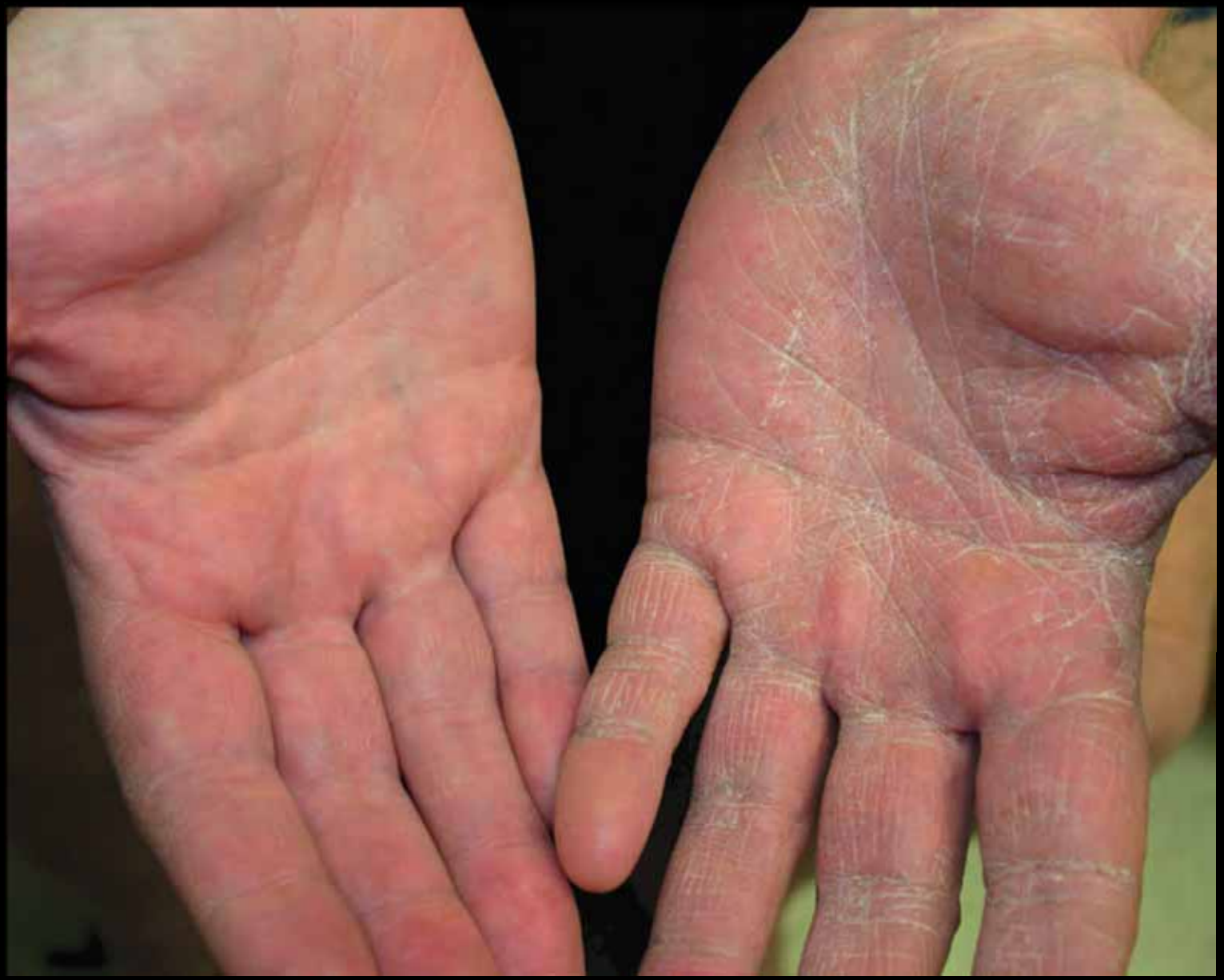


Tinea barbae



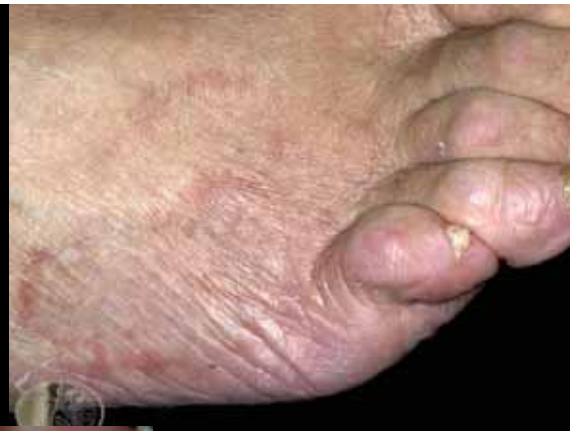


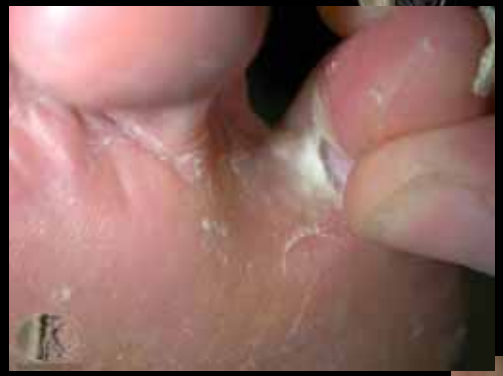
Tinea manuum

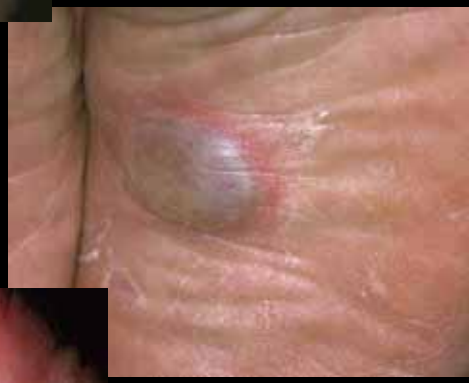


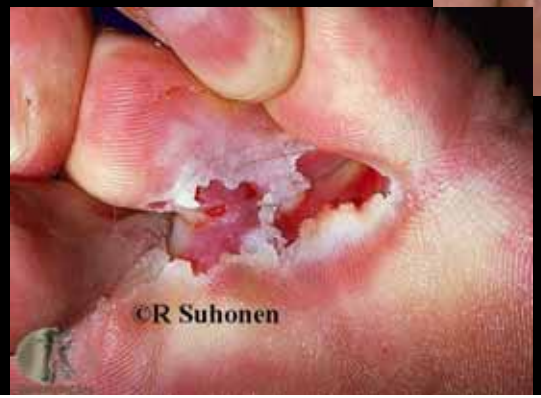
Tinea pedis

- 4 Types
  - Moccasin
    - T. rubrum
  - Interdigital
    - Most common
    - T. mentagrophyte (var interdigitale)
  - Inflammatory (vesicular/bullous)
    - T. mentagrophyte
  - Ulcerative
    - T. rubrum

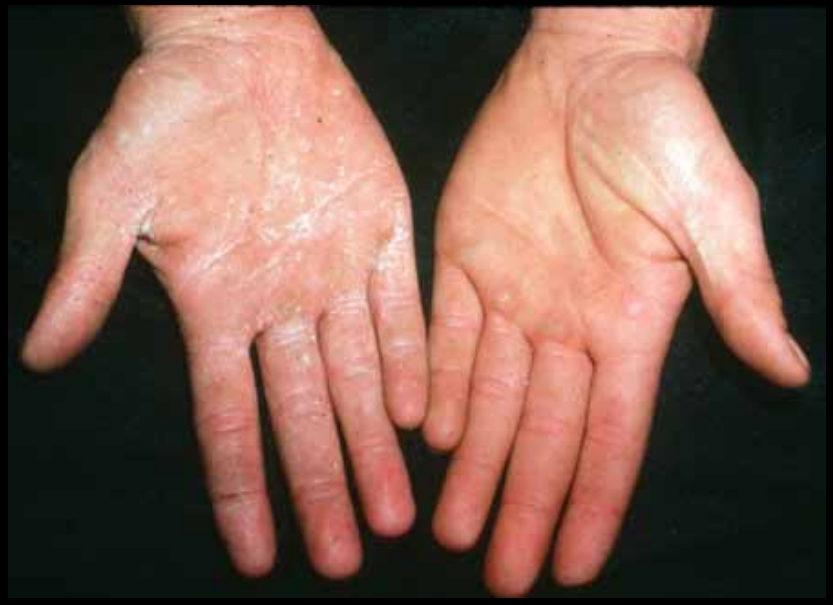


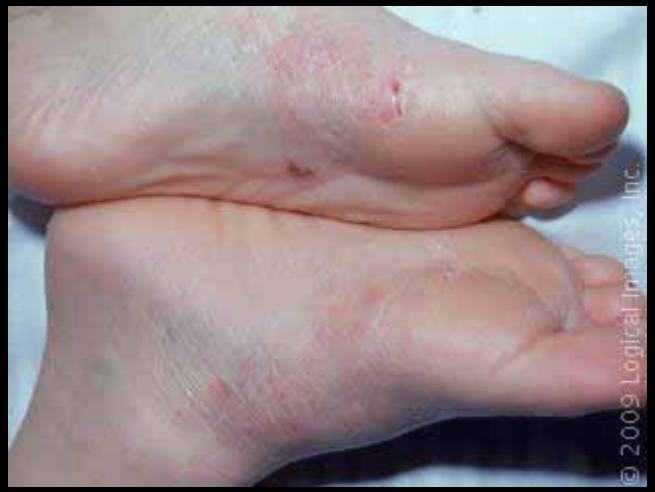




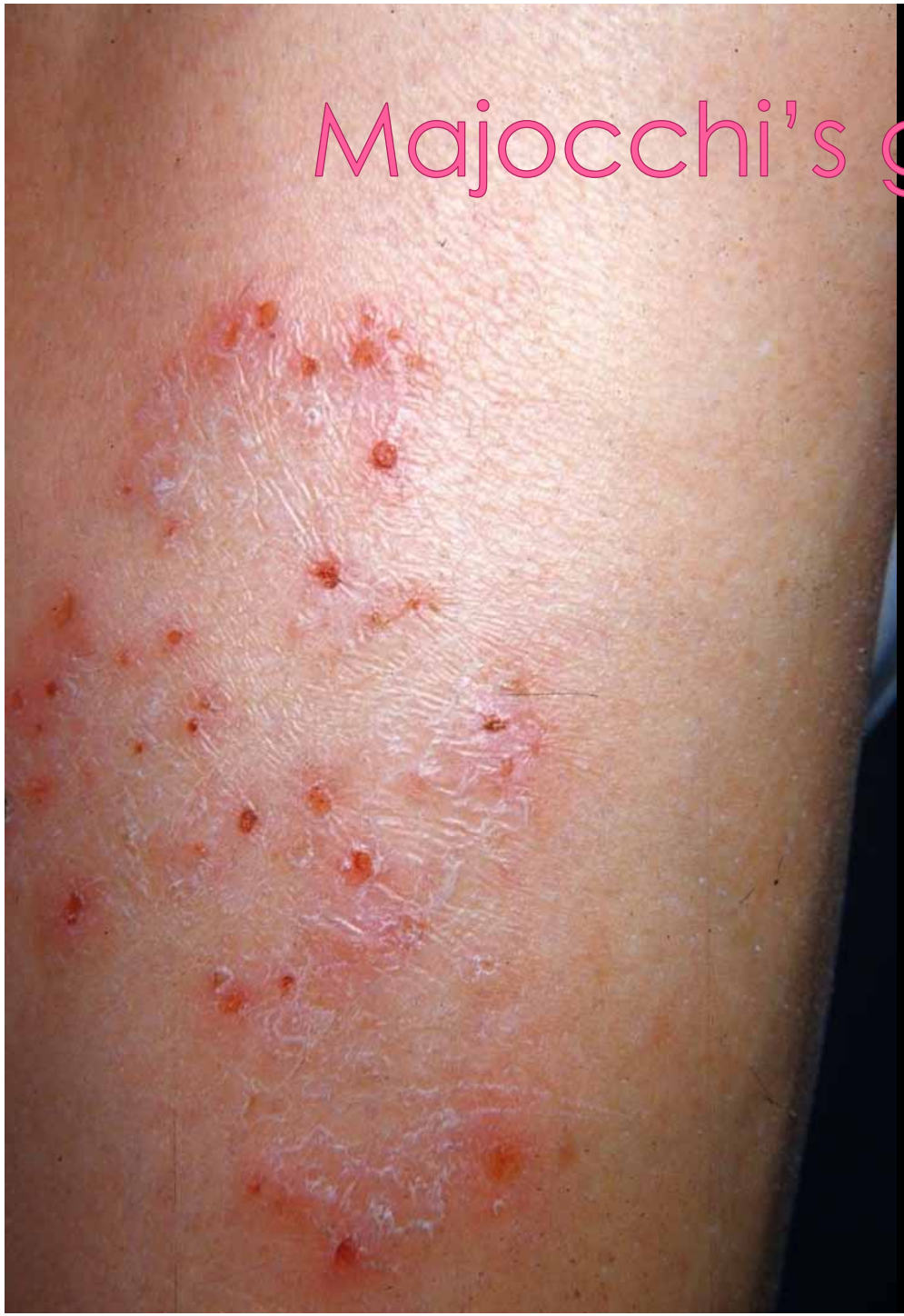


Two foot one hand





Majocchi's granuloma



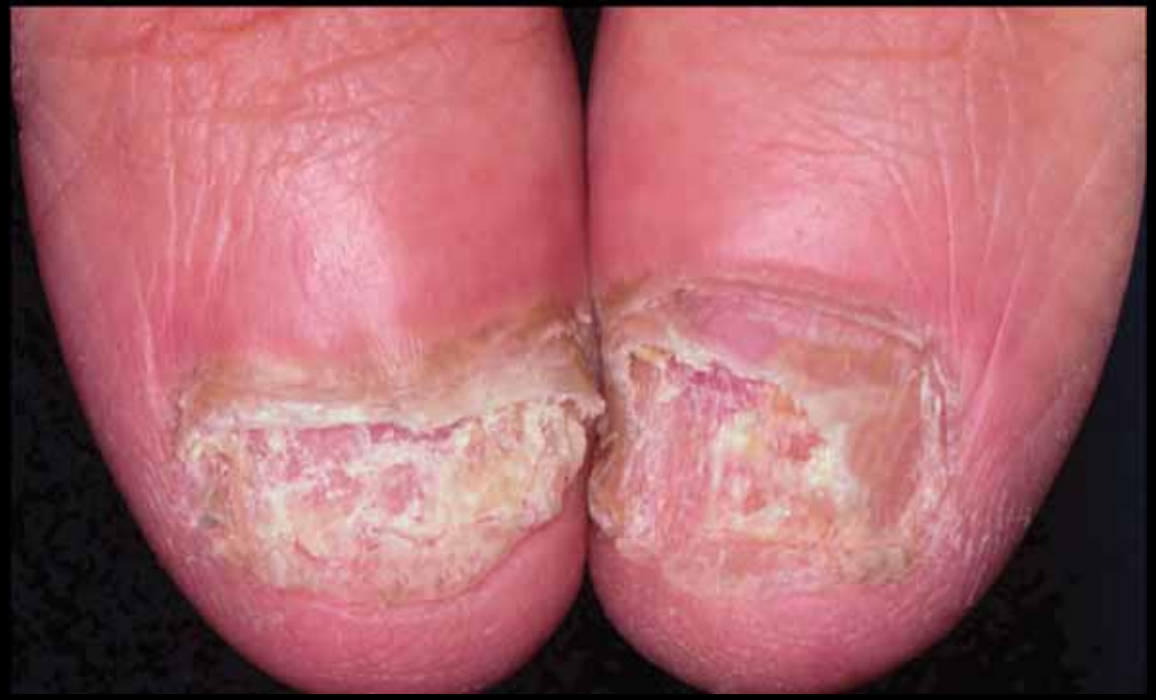
Majocchi's granuloma
- Infection of hair follicle
- Usually KOH negative
- Tx with oral antifungals

Treatment of dermatophytosis

- Topical imidazoles
- Systemic antifungal medication

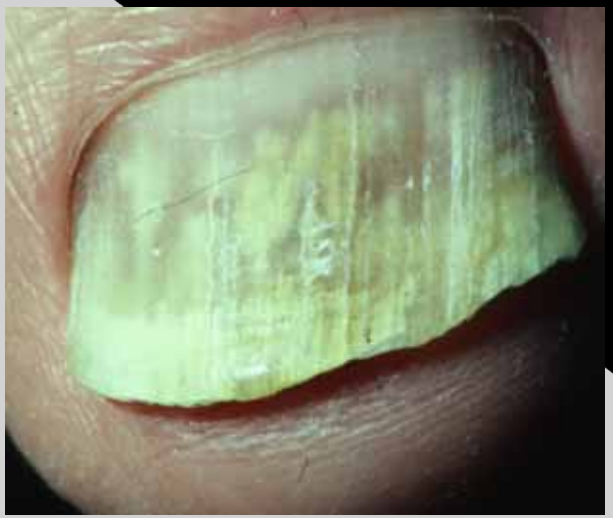
Systemic Rx	Griseofulvin (Micronized)	Fluconazole	Itraconazole	Terbinafine
Tinea pedis/manuum	7.5-1 g/d 10-20mg/d Fatty meal x4 wks	150–200 mg/wk 6 mg/kg/wk x4-6wks	200–400 mg/day 3–5 mg/kg/day (max 400 mg) × 1wk	250 mg/day 125mg<25kg 187.5mg25-35kg 250mg>35kg x2wk
Tinea corporis/cruris Extensive	0.5-1 g/d 10-20mg/d Fatty meal x2-4 wks	150–200 mg/wk 6 mg/kg/wk X2-4wks	200–400 mg/day 3–5 mg/kg/day (max 200 mg) × 1wk	250 mg/day 125mg<25kg 187.5mg25-35kg 250mg>35kg x1wk

Tinea ungium

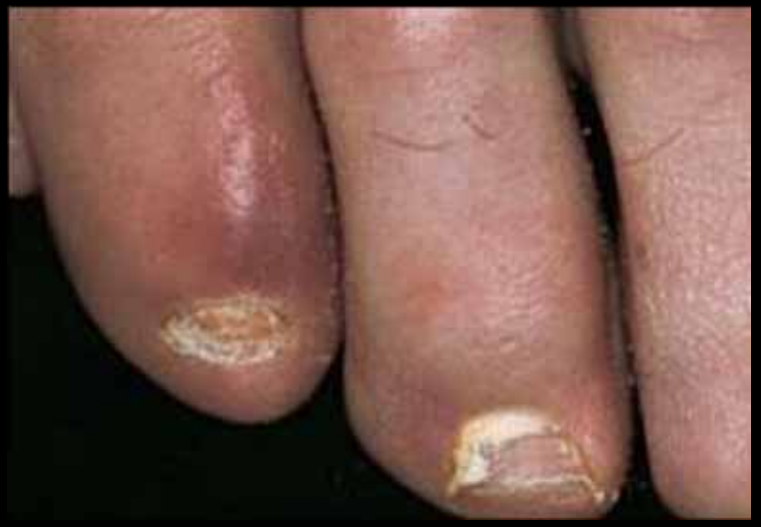


- Onychomycosis is a term used to encompass all fungal infections of the nail and includes those due to dermatophytes as well as non- dermatophytes.

Onychmycosis – 4 Types



Distal-lateral subungual onychomycosis (DSO) T. rubrum

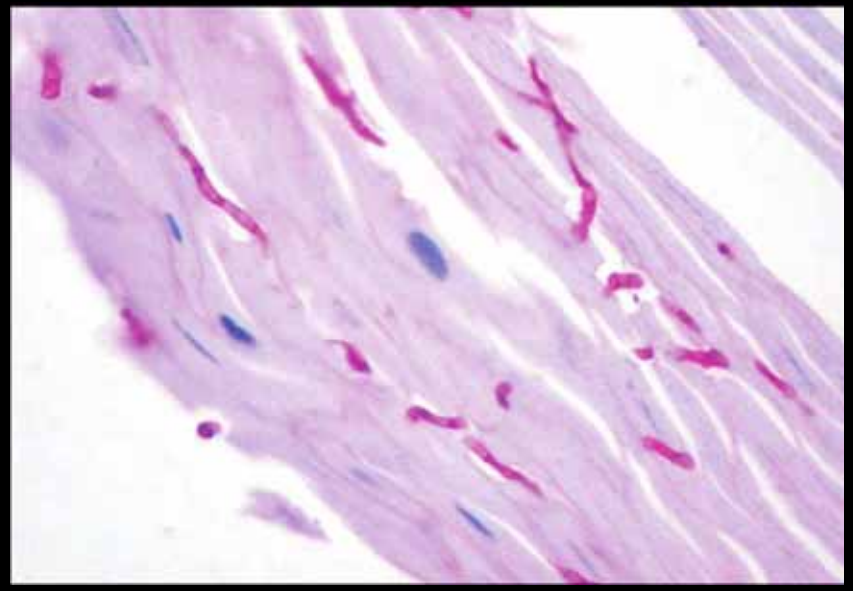


Proximal subungual onychomycosis (PSO)T. rubrum

White superficial onychomycosis (WSO)T. mentagophyte

Candida onychomycosis

Lab investigation for onychomycosis



- KOH
- Nail clipping: sensitivity of 80% to >95% (compared to 35–60% for culture alone)
- Fungal culture

Treatment of onychomycosis

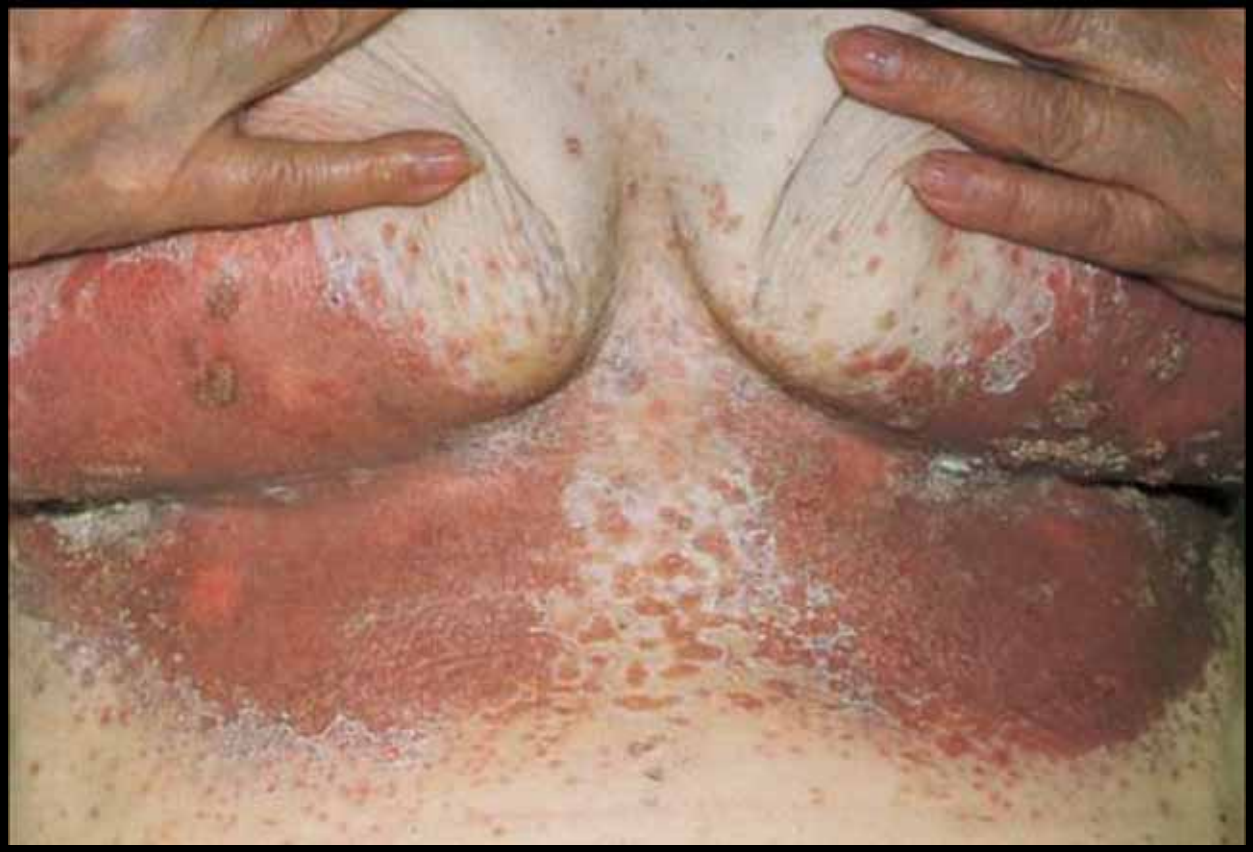
Systemic Rx of Tinea ungium	Griseofulvin (Micronized)	Fluconazole	Itraconazole	Terbinafine
Fingernail	No longer use for this indication	Adult: 150–200 mg/wk X 6 mos Chlidren: 6 mg/kg/wk X3-4mos	200 mg/day × 6 weeks or Pulse RX: 200 mg bid × 1 wk/mo for 2 consecutive months	250 mg/day x6wk 62.5mg<20kg 125mg20-40kg 250mg>40kg x6wk
Toenail		Adult: 150–200 mg/wk X 9 mos Chlidren: 6 mg/kg/wk X5-7mos	200 mg/day × 12 weeks or Pulse RX: 200 mg bid × 1 wk/mo for 3–4 consecutive months	250 mg/day x12wk 62.5mg<20kg 125mg20-40kg 250mg>40kg x12wk

Tips for Dermatophyte treatment

- Systemic tretment should be prescribed in
  - Tinea capitis
  - Onychomycosis
  - Majoccho's granuloma

Case 3

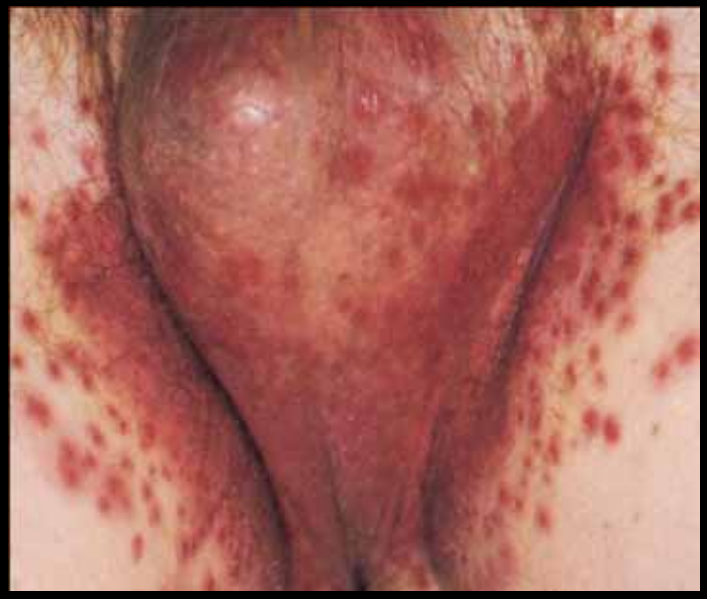
- A 60 year-old woman with icthy rash submammary area for 3 weeks

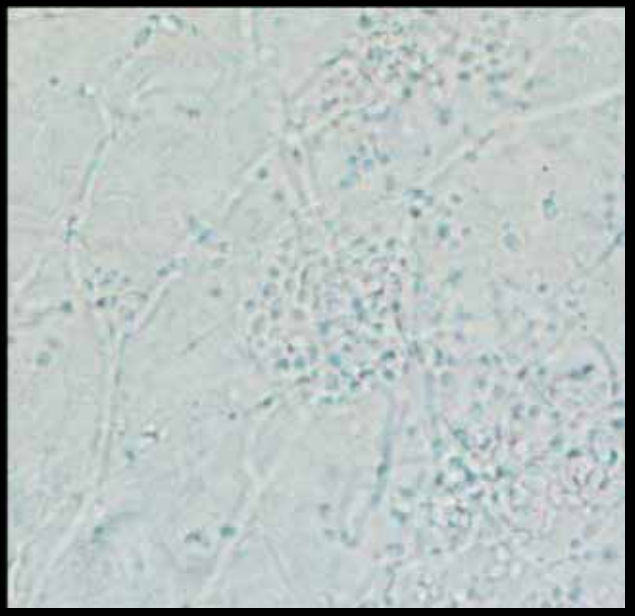


Cutaneous candidiasis

KOH : Pseudohyphae with budding yeast

Cutaneous candidiasis



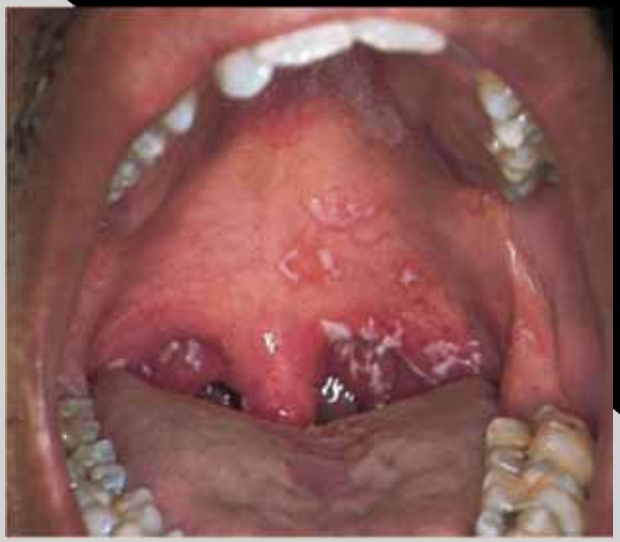


- Candida albicans accounts for 70-80% of all candida infection
- Erythematous marcerated patches with satellite vesiculopustules on intertriginous area

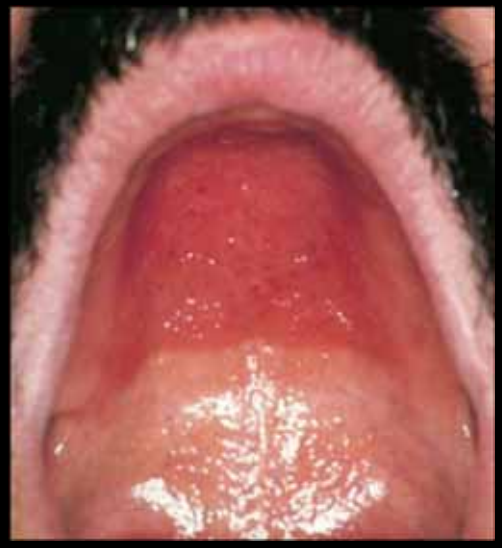
Treatment of mucutaneous candidiasis

- Candidal intertrigo
  - Topical imidazoles or cyclopirox BID for 1-2 weeks or until resolved
  - Systemic agents for recalcitrant or severe cases
    - Fluconazole: 50-100 mg OD x14 days
      or 150 mg/wk x 2-4 weeks
    - Itraconazole 200 mg BID X14 days

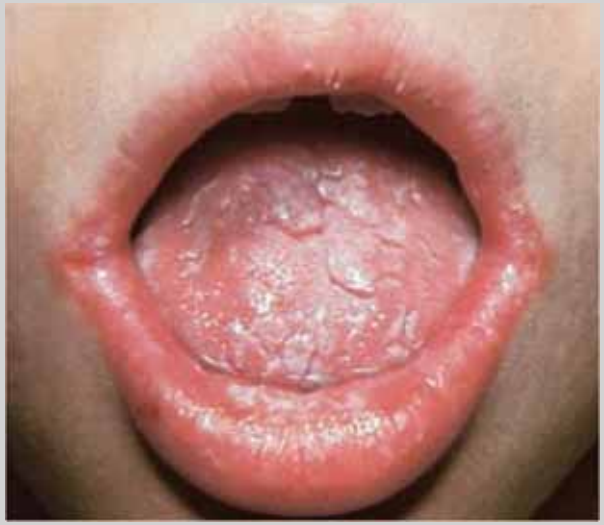
Oral candidiasis: 4 types



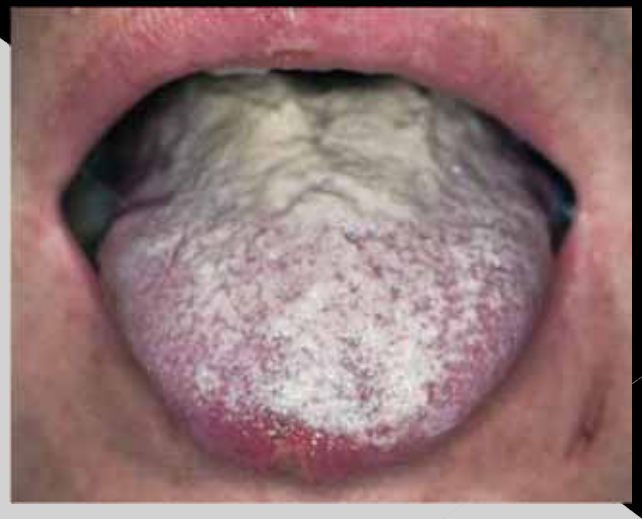
Pseudomenbranous



Atrophic



Perleche



Hyperplastic

Treatment of mucocutaneous candidiasis

- Oropharyngeal candidiasis
  - Nystatin 100 000 units/ml suspension:
    - Children and adults – 4–6 ml swish and swallow qid
    - Infants – 2 ml (1 ml inside each cheek) qid
  - Clotrimazole* 10 mg troche five times daily
  - Fluconazole 200 mg po on day 1, then 100–200 mg po daily
  - *Continue treatment for 7–14 days after clinical resolution*

Viral infection

Herpes simplex infection

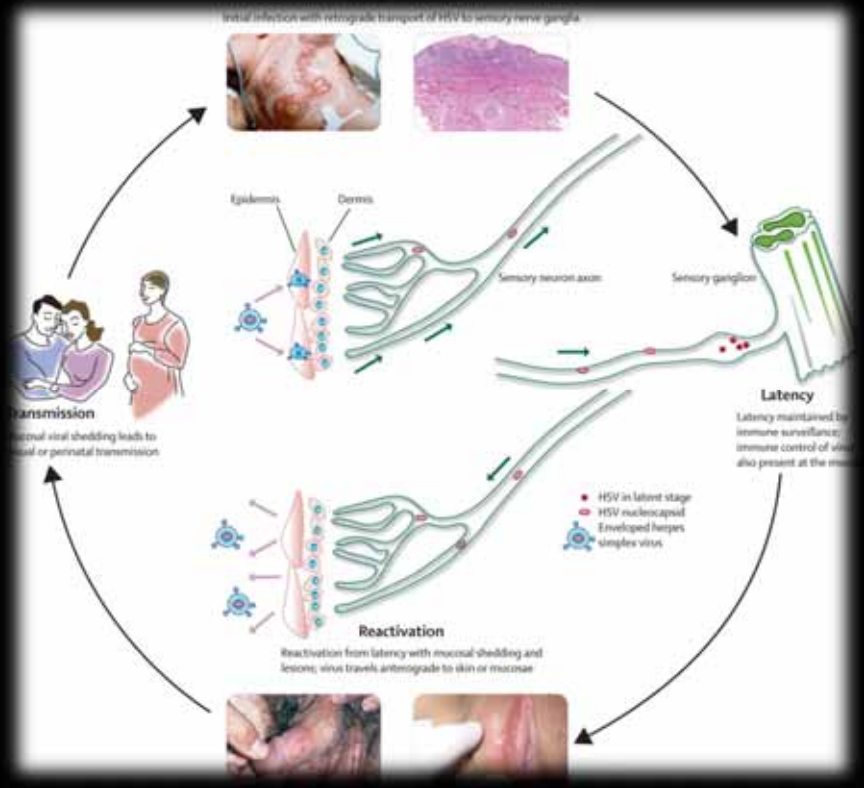
- Epidemiology: 2 types of HSV
- HSV-1 mostly associated with orofacial disease
- HSV-2 usually associated with genital and perigenital infection (70-90%)
- Transmission:
  - HSV-1- direct contact with contaminated saliva or secretions
  - HSV-2 – sexual contact

Herpes simplex infection

- Incidence :
  - primary HSV1 infection is greatest in childhood
  - Most adults are seropositive for HSV-1
  - HSV-2 infection correlates with sexual behavior

Herpes simplex infection

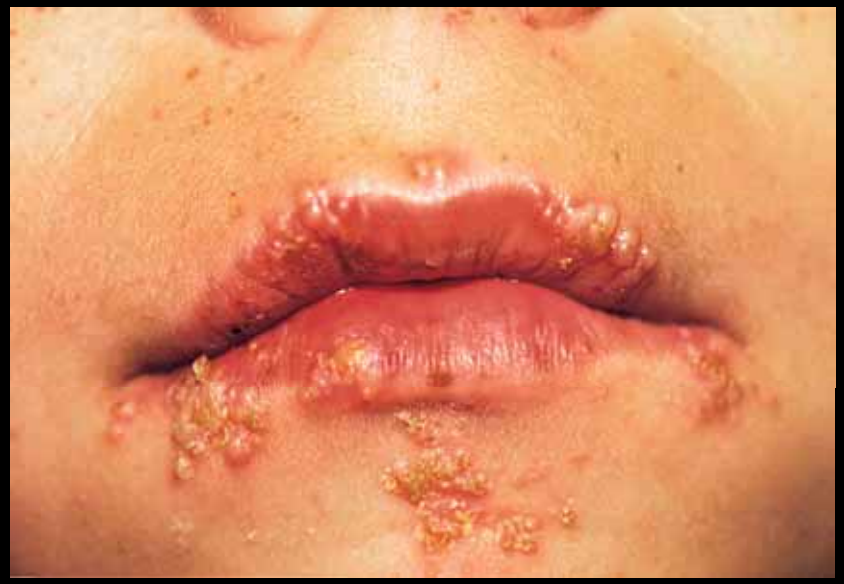
- Etiology and Pathogenesis
- HSV infection stages :
  - Primary infection
  - Latency
  - Reactivation resulting in recurrent infection

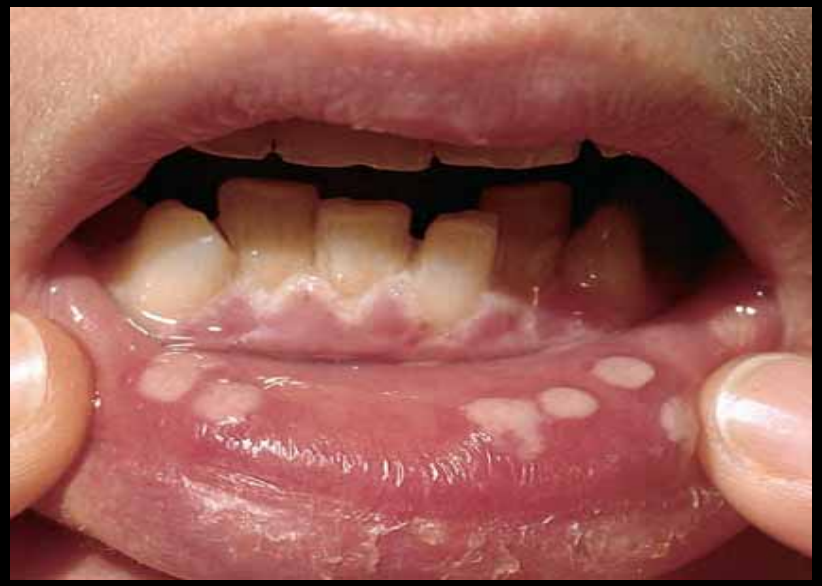


• Virus replication in skin or mucosa → virus infects local nerve endings → ascends to sensory nerve ganglia which become latent → reactivation

Clinical findings – orofacial infection

"primary infection"

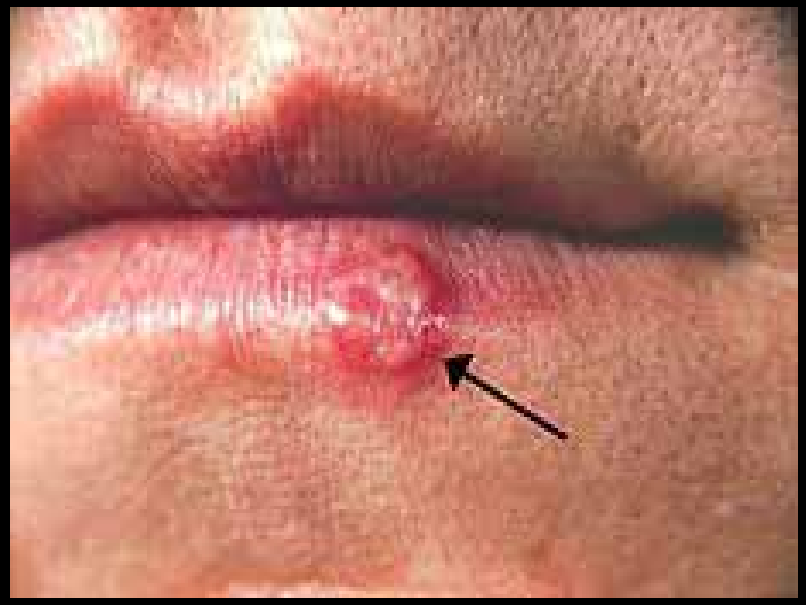




- Prodrome : fever, tender lymphadenopathy, localized pain or burning
- Painful, groups of vesicles on erythematous base progress to pustules and erosions with characteristic scalloped primary sites = mouth and lips, others = buccal mucosa and gingiva

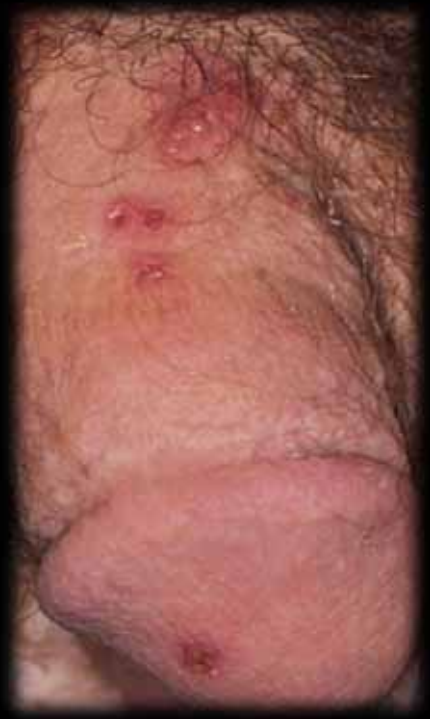
Clinical findings – orofacial infection

“recurrent infection”

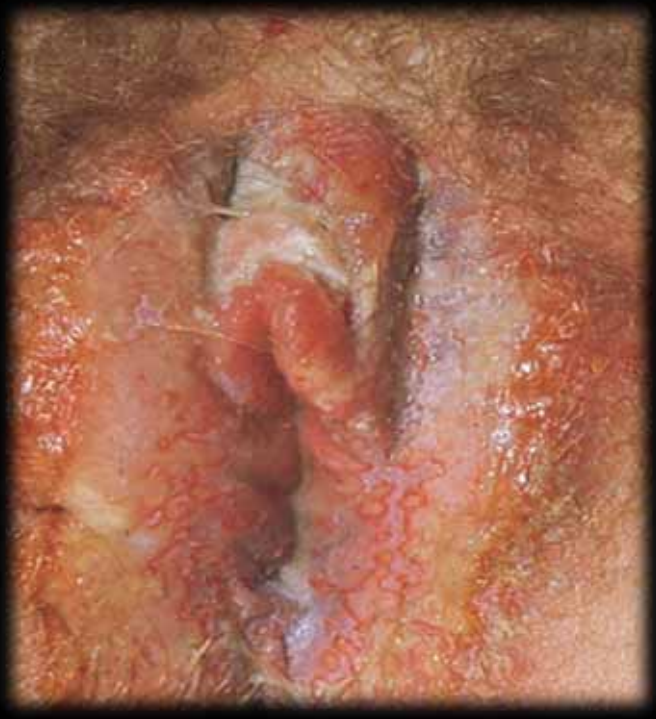


- Prodromal symptoms (pain, burning, itching) in 45-60%
- Cluster of tiny vesicles on erythematous base and ulcers
- Sites: perioral facial area : mainly lips (outer 1/3 of lower lip
- Resolved within 5-15 days

Clinical findings – genital infection



"primary infection"

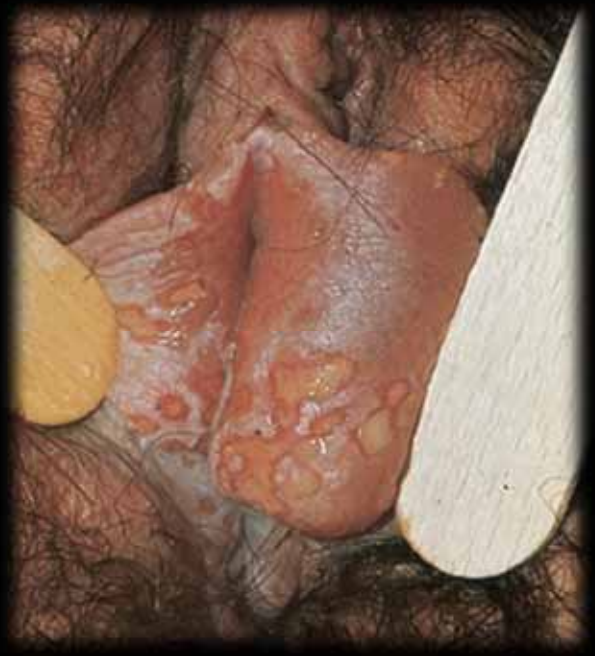


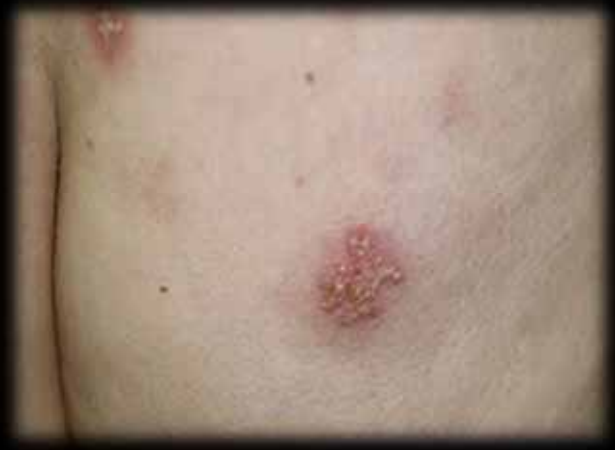
- Fever, inquinal lymphadenopathy, dysuria, discharge
- Extensive genital lesions in different stages of evolution (vesicle, pustule, ulcer)
- Sites: men : glans penis, penile shaft

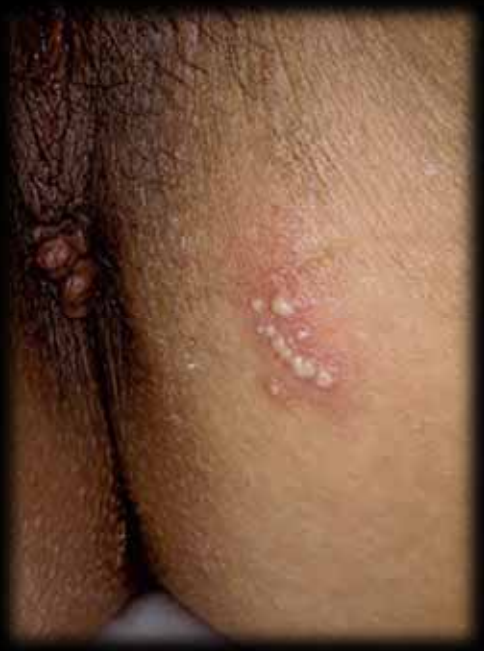
  women : vulva, perineum, buttock, vagina, cervix

Clinical findings – genital infection

"recurrent infection"

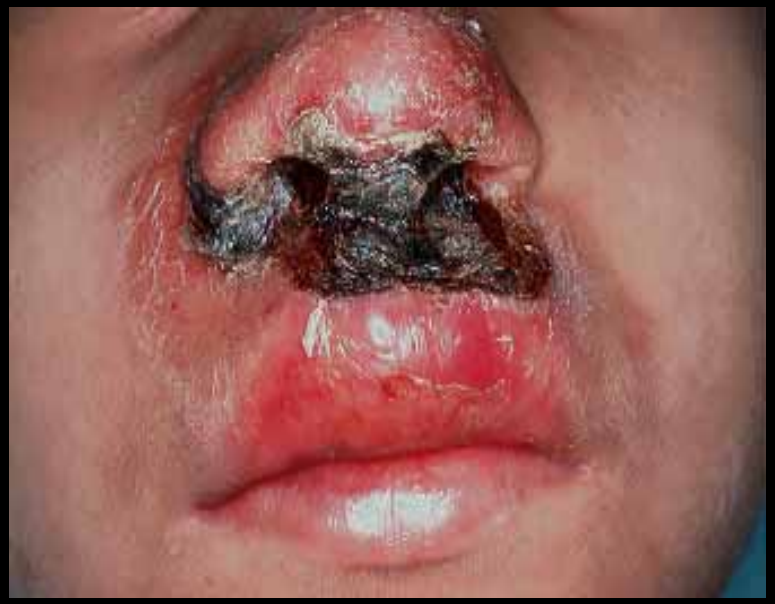


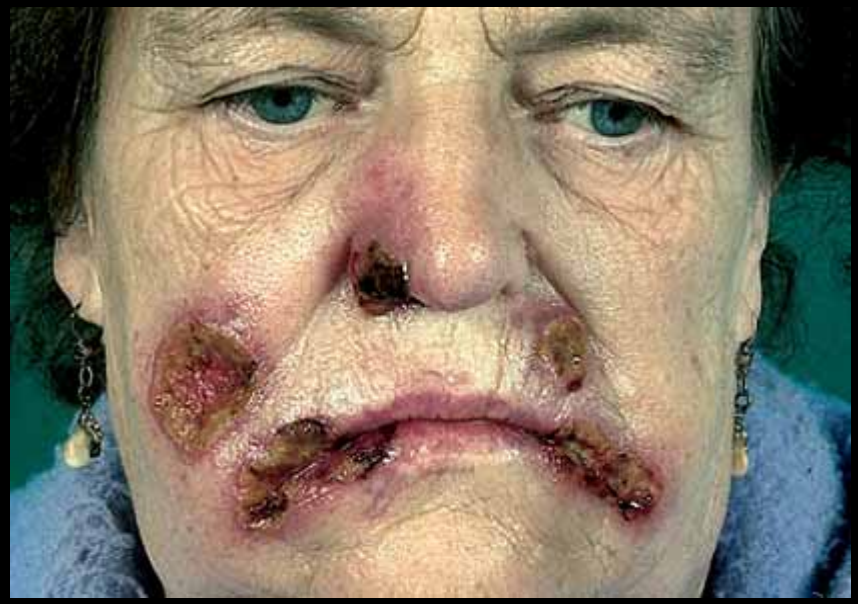




- Multiple small grouped vesicular lesions
- Genital area or perigenital areas (abdomen,groin,buttocks, thighs)
- Prodrome → outbreak in same site or change location
- Heal in 6-10 days

Clinical findings – immunocompromised host





- More severe, more extensive, more recurrent and difficult to treat
- Multiple sites or disseminated infection
- Atypical lesions : hemorrhagic, necrotic lesions, verrucous, exophytic

Most common presentation = chronic enlarging ulcerations

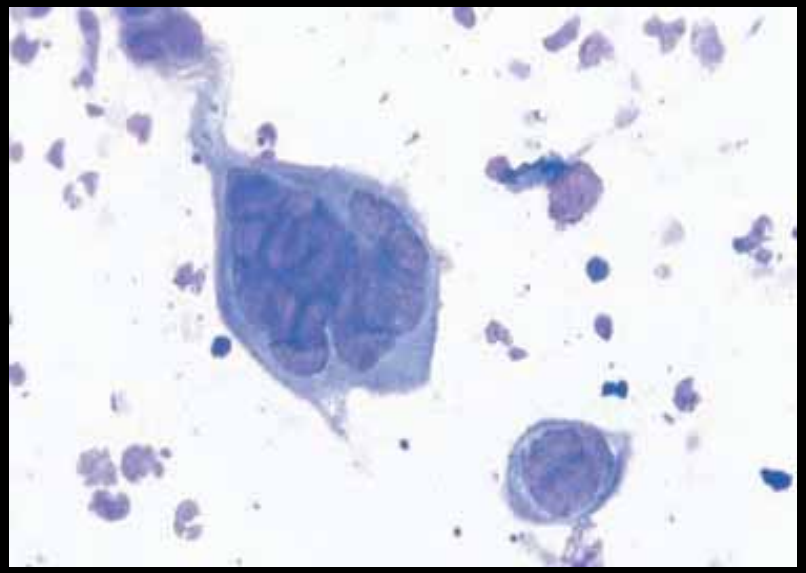
Laboratory tests

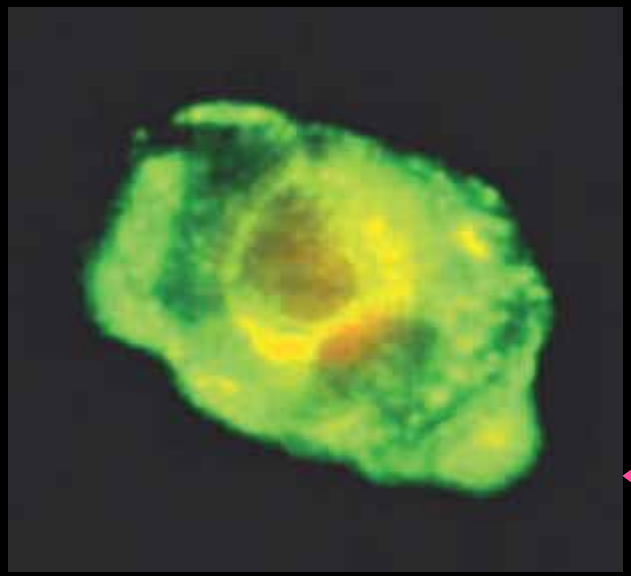
Tzanck smear : scraping base of freshly ruptured vesicle → Wright stain

- multinucleated giant cells
- rapid diagnosis, less sensitivity (~40%)

DFA (direct fluorescent antibody staining of lesion scarping)

- lower sensitivity than viral culture, but greater than Tzanck smear





Laboratory tests

Viral culture

- sensitivity 60-70%
- prove positive after 48-96 hours of inoculation
- high yield in vesicular stage, 1^0 infection and immunocompromised host

PCR assay to detect HSV DNA

- most sensitive → preferred method of diagnosis
- useful for detection in late-stage ulcers

HSV antibody detection (serology)

- differentiate 1^0infection from recurrent infection
- mean to seroconversion = 3-4 weeks

Management of primary genital infection

- Oral acyclovir, famciclovir and valacyclovir all speed the resolution but not decrease subsequent recurrence (HSV establish latent infection days before symptom evolves)
- Duration : 7-10 days or clinical resolution occurs

Recommended Regimens*

Acyclovir 400 mg orally three times a day for 7–10 days

OR

Acyclovir 200 mg orally five times a day for 7–10 days

OR

Famciclovir 250 mg orally three times a day for 7–10 days

OR

Valacyclovir 1 g orally twice a day for 7–10 days

*Treatment can be extended if healing is incomplete after 10 days of therapy.

www.cdc.gov/std/herpes

Management of recurrent genital infection

- Oral acyclovir, famciclovir and valacyclovir all speed the resolution
- Duration : 5-10 days or clinical resolution occurs

Recommended Regimens*

Acyclovir 400 mg orally three times a day for 5 days

OR

Acyclovir 800 mg orally twice a day for 5 days

OR

Acyclovir 800 mg orally three times a day for 2 days

OR

Famciclovir 125 mg orally twice daily for 5 days

OR

Famciclovir 1000 mg orally twice daily for 1 day

OR

Famciclovir 500 mg once, followed by 250 mg twice daily for 2 days

OR

Valacyclovir 500 mg orally twice a day for 3 days

OR

Valacyclovir 1 g orally once a day for 5 days

www.cdc.gov/std/herpes

Management of recurrent genital infection

- Long-term suppressive therapy in attacks ≥ 6 times per year
- Duration : 1 year and reassess the need to resume

Recommended Regimens*

Acyclovir 400 mg orally twice a day

OR

Famiciclovir 250 mg orally twice a day

OR

Valacyclovir 500 mg orally once a day*

OR

Valacyclovir 1 g orally once a day

* Valacyclovir 500 mg once a day might be less effective than other valacyclovir or acyclovir dosing regimens in patients who have very frequent recurrences (i.e., ≥10 episodes per year).

www.cdc.gov/std/herpes

Management of primary orofacial infection

- Oral acyclovir within 3 days of onset decreases illness duration
- Famciclovir and valacyclovir may be equally effective,
  but no studies and not approved for use in children
- Duration : 7-10 days or clinical resolution occurs

Recommended Regimens*

Acyclovir 400 mg orally three times a day for 7–10 days

OR

Acyclovir 200 mg orally five times a day for 7–10 days

OR

Famciclovir 250 mg orally three times a day for 7–10 days

OR

Valacyclovir 1 g orally twice a day for 7–10 days

*Treatment can be extended if healing is incomplete after 10 days of therapy.

Management of recurrent orofacial infection

- Oral acyclovir, famciclovir and valacyclovir afford benefit
- Duration : 4-5 days or until lesions are healed
- Long-term suppressive treatment is controversial

Recommended Regimens

Topical 1% penciclovir cream q 2 hrs while awake: RX of choice

Topical 10% docosanol cream five times a day

Acyclovir 400 mg orally five times a day for 5 days

Famciclovir 500 mg orally three time a day for 5 days

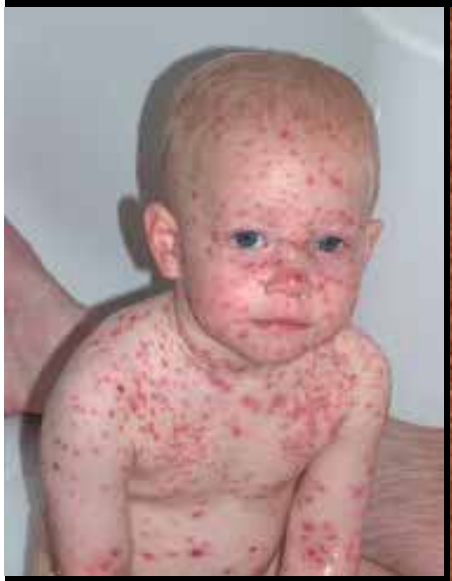
Famciclovir 1,500 mg single dose

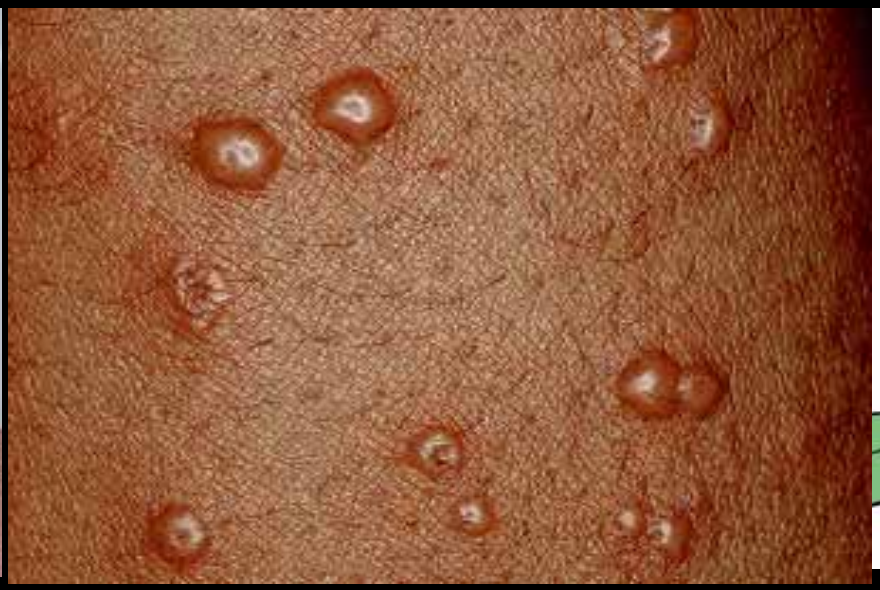
Valacyclovir 2,000 mg orally twicw a day for 1 day

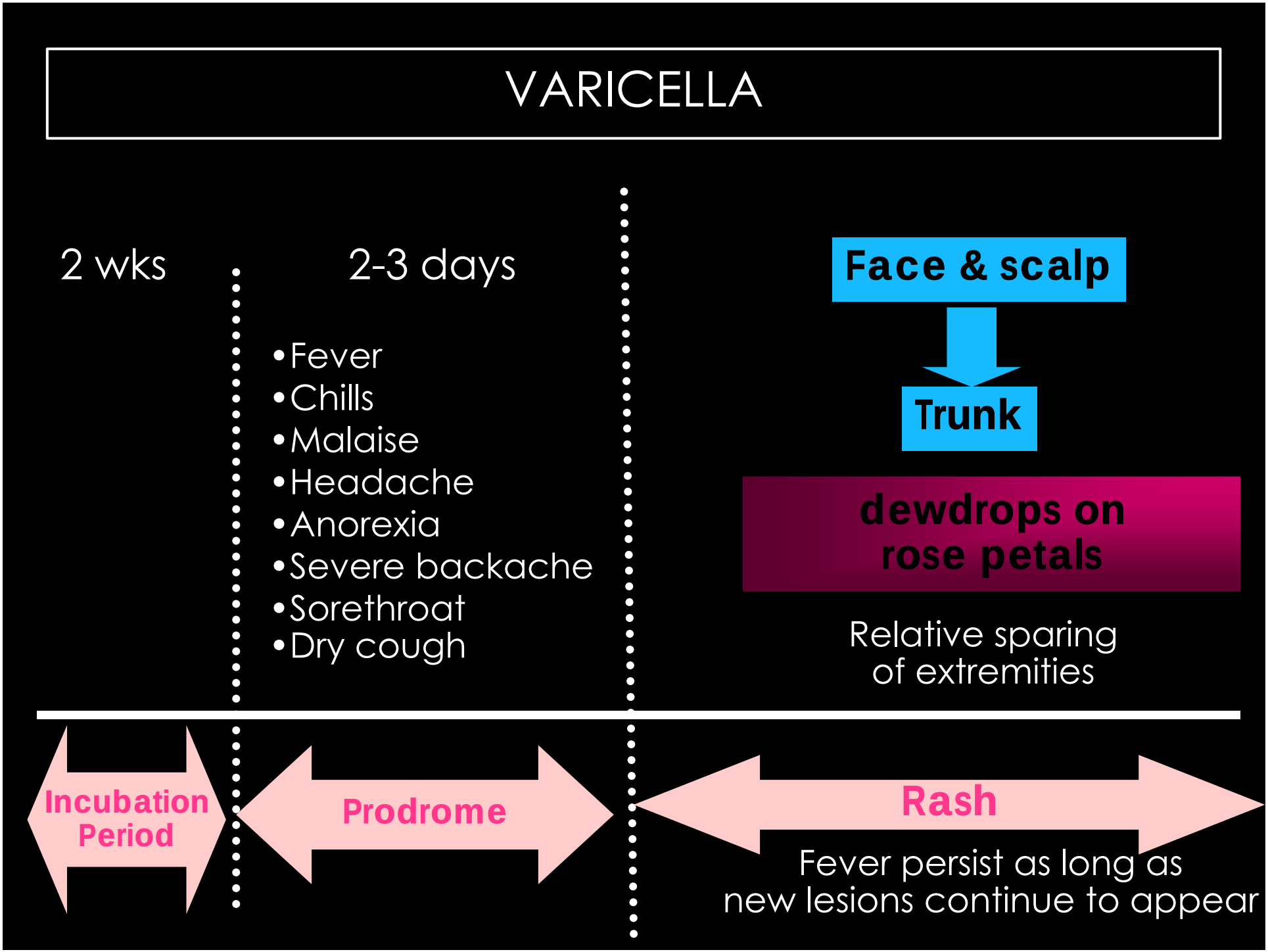
Varicella infection

VARICELLA

- Highly contagious disease caused by VZV
- Transmission : direct contact
  : airborne transmission
- Contagious period : 1-2 days before rash until the crops of vesicles has crusted






VARICELLA
2 wks
2-3 days
•Fever
•Chills
•Malaise
•Headache
•Anorexia
•Severe backache
•Sorethroat
•Dry cough
Face & scalp
Trunk
dewdrops on rose petals
Relative sparing of extremities
Incubation Period
Prodrome
Rash
Fever persist as long as new lesions continue to appear

VARICELLA

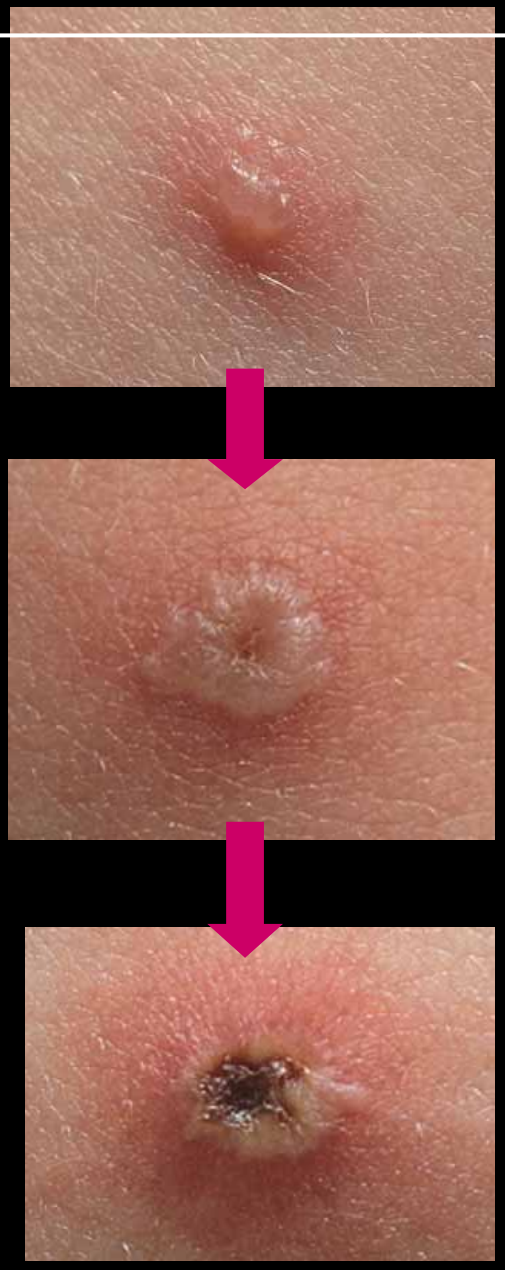
All stages of development is hallmark

Rose-colored
Macules & papules

vesicles

pustules

crusts



COMPLICATION OF VARICELLA

- Complications are more common in adults
- In children : most common complication is 2nd bacterial infection

  others – bacterial pneumonia, otitis media, suppurative meningitis
- In adults : major complication is primary varicella pneumonia
- In immunocompromised patients :

  - extensive rash, often with hemorrhagic or purpuric lesions
  - longer period of new vesicle formation
  - visceral dissemination

  (lung, liver and CNS are common)

LABORATORY TESTING

Tzanck smear

Viral culture

- sensitivity 30-60%, take 1 week time
- high yield in new vesicle containing clear fluid (lower yield in pustule)

DFA (direct fluorescent antibody staining of lesion scarping)

- more sensitive and faster than viral culture

PCR assay to detect VZV DNA

- most sensitive → preferred method of diagnosis
- idenify wild-type and vaccine strain

VZV antibody detection (serology)

- retrospective diagnosis, lacks sensitivity and specificity

TREATMENT OF VARICELLA

Antiviral Treatment of Varicella in the Normal and Immunocompromised Host

Patient Group	Regimen
Normal[a]	
Neonate	Acyclovir 10 mg/kg or 500 mg/m^2 every 8 h for 10 days
Child (2 to <18 years of age)	Symptomatic treatment alone, or Valacyclovir 20 mg/kg every 8 h for 5 days[b] (not to exceed 3 g/day) or Acyclovir 20 mg/kg po four times a day × 5 days (not to exceed 3200 mg/day)
Adolescent (≥40 kg) or adult, especially with mild immune compromise (e.g., use of inhaled glucocorticoids)	Valacyclovir 1 g po every 8 h for 7 days or Famciclovir 500 mg po every 8 h for 7 days or Acyclovir 800 mg po five times a day for 7 days
Pneumonia	Acyclovir 10 mg/kg IV every 8 h × 7–10 days[b]
Pregnancy	Routine use of acyclovir is not recommended. If there are complication (e.g., pneumonia) treat pneumonia as per recommendation above.
Immunocompromised	
Mild varicella or mild compromise	Valacyclovir 1 g po every 8 h for 7–10 days or Famciclovir 500 mg po every 8 h for 7–10 days or Acyclovir 800 mg po five times a day for 7–10 days
Severe varicella or severe compromise	Acyclovir 10 mg/kg IV every 8 h for 7–10 days
Acyclovir resistant (advanced AIDS)	Foscarnet 40 mg/kg IV every 8 h until healed

Herpes zoster infection

HERPES ZOSTER

- 20% in healthy adults, and 50% in immunocompromised patients
- Less contagious than varicella (transmission rate 15%, while 70% in varicella)
- Transmission by direct contact

  airborne transmission

  only in disseminated herpes zoster

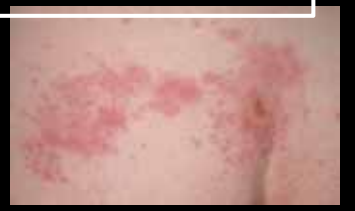
  localized herpes zoster in immunocompromised host
- Strong risk factors :
  - older age
  - CMI dysfunction (HIV, BMT, leukemia/lymphoma, use CMT or steroid)

HERPES ZOSTER

- Pain
- Paresthesia
- Itching
- Tingling
- Burning

Several days

Dermatome innervation

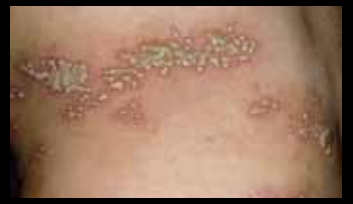


Erythematous macules & papules

12-24 hrs

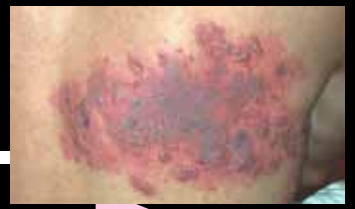
vesicles

3rd day



pustules

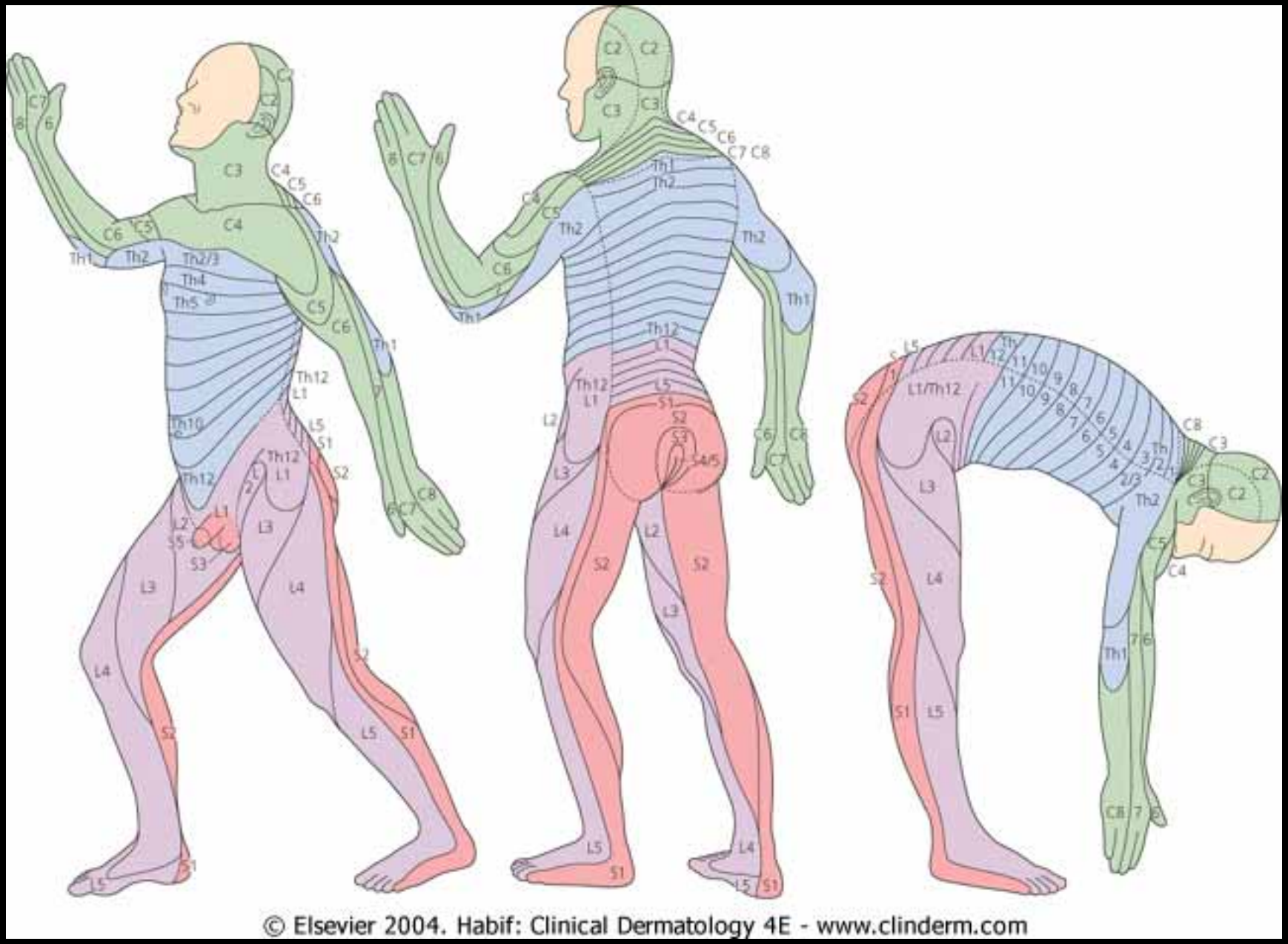
7-10 days



Persist 2-3 wks **Dry & form crusts**

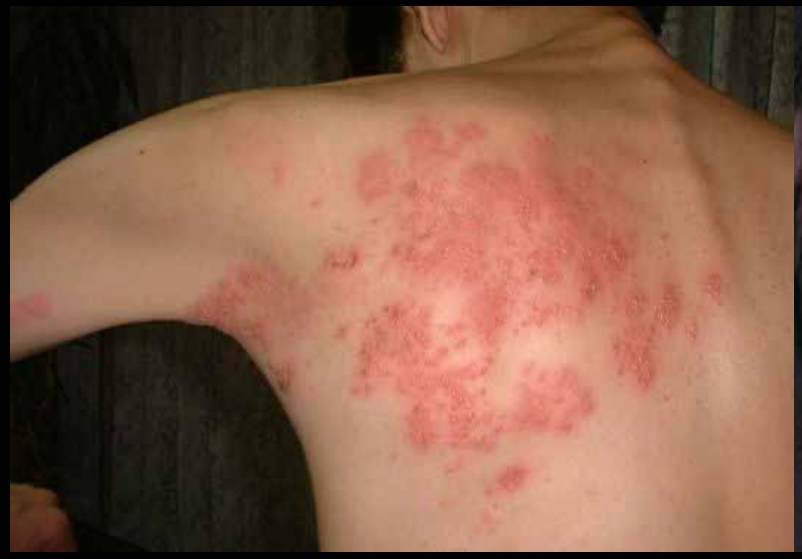
Prodrome

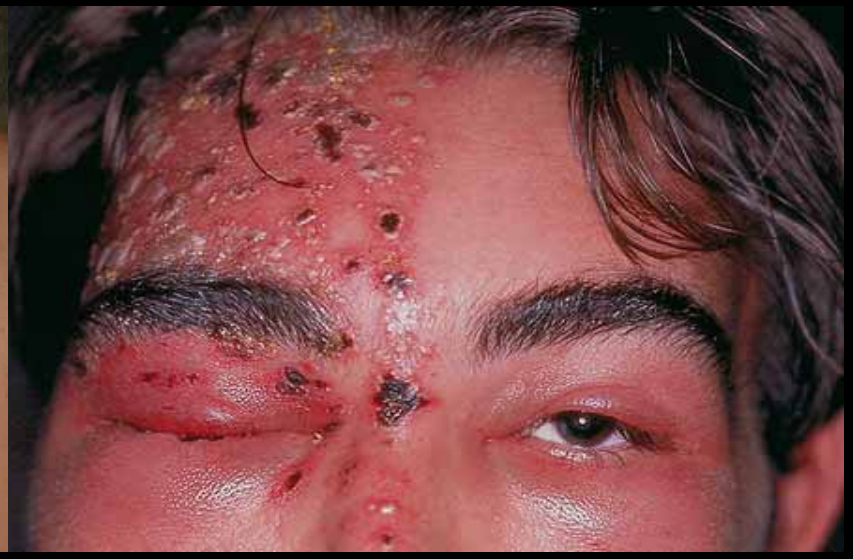
Rash



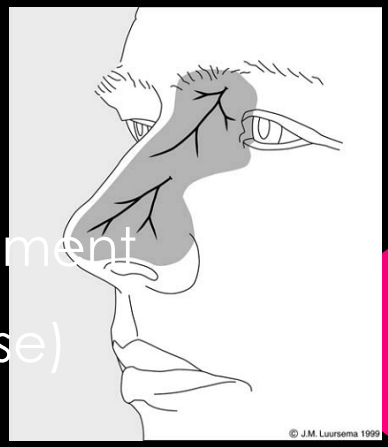
CLINICAL FINDING OF HERPES ZOSTER

- Most common in trunk from T3 to L2 and trigeminal nerve (esp. V1)



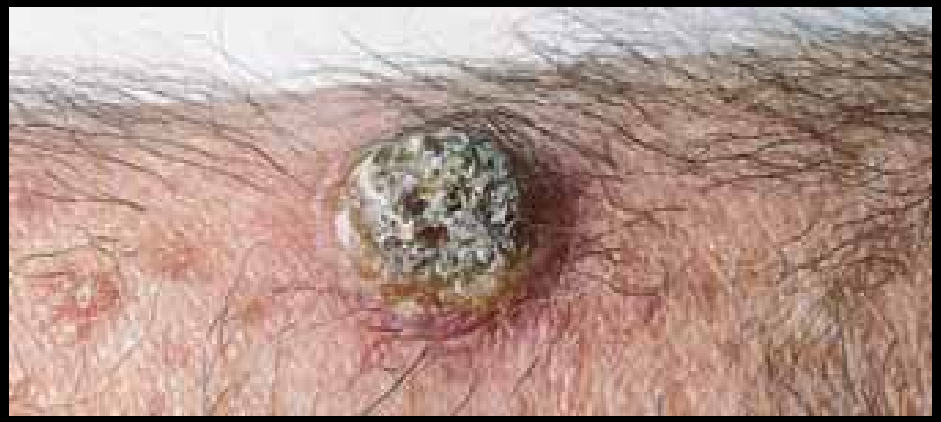


- Ophthalmic zoster occurs in 10-15%
  - 20-70% have eye involvement
  - "hutchinson's sign" → nasociliary branch involvement (lesions at tip and side of nose) → risk for eye involvement



HERPES ZOSTER IN IMMUNOCOMPROMISED HOST

- Severe and multiple recurrence
- Cutaneous dissemination (>20 vesicles outside 1^0 & adjacent dermatomes)
- Visceral dissemination
  (lung, liver, brain)
- Necrosis of skin and scarring
- Chronic verrucous, hyperkeratotic lesions
  (caused by acyclovir-resistant VZV)



COMPLICATION OF HERPES ZOSTER

Postherpetic neuralgia (PHN) : most common complication

- overall incidence 8-15%
- most significant risk factor = age
- other risk factors: prodrome pain, severe pain in acute phrase severity, ophthalmic zoster
- remit spontaneous over several months

Cutaneous dissemination and superficial gangrene with scarring

in HIV, malignancy esp. lymphoma, immunosuppressive therapy

Ocular complications: acute retinal necrosis

Visceral complications: pneumonitis, hepatitis, meningoencephalitis

LABORATORY TESTING

Tzanck smear

Viral culture

- sensitivity 30-60%, take 1 week time
- high yield in new vesicle containing clear fluid (lower yield in pustule)

DFA (direct fluorescent antibody staining of lesion scarping)

- more sensitive and faster than viral culture

PCR assay to detect VZV DNA

- most sensitive → preferred method of diagnosis
- idenify wild-type and vaccine strain

VZV antibody detection (serology)

- retrospective diagnosis
- lacks sensitivity and specificity

TREATMENT OF HERPES ZOSTER

- VZV is 10 fold less sensitive to acyclovir than HSV → require higher dosage
- Famciclovir and valacyclovir are preferred for VZV infection
- Topical antiviral Rx lacks efficacy in VZV infection and not recommended
- In normal patients :
  - Rx within 72 hours of rash onset (up to 7 days affords benefit)
  - if >72 hours, Rx in (1) zoster involve cranial nerve, esp. CN V1
    (2) continue to have new vesicles

Recommended Regimens

Valacyclovir 1 gm orally three time a days for 7 days

OR

Famciclovir 500 mg orally three time a day for 7 days

OR

Acyclovir 800 mg orally five times a day for 7 days

TREATMENT OF HERPES ZOSTER

In immunocompromised host :

- Mild compromise, including HIV-1 infection
  - Famciclovir 500 mg PO every 8 h for 7–10 days or
  - Valacyclovir 1 g PO every 8 h for 7–10 days or
  - Acyclovir 800 mg PO 5 times a day for 7–10 daysa
- Severe compromise
  - Acyclovir 10 mg/kg IV every 8 h for 7–10 days
- Acyclovir resistant (e.g., advanced AIDS)
  - Foscarnet 40 mg/kg IV every 8 h until healed

TREATMENT OF HERPES ZOSTER

- PHN : randomized controlled trials showed effective treatment in

  (1) topical : 5%lidocain patch, 8% capsaicin patch

  (2) systemic : gabapentin (Neurontin®),

  pregabalin (Lyrica®)

  TCAs(prefer nortriptyline rather than amitryptyline

(fewer cardiac side effect)

  opioid, tramadol

In conclusion

- Common bacterial infection:
  - Staphylococcal & Streptococcal infection
  - Corynebacterial infection
  - Pseudomanas infection
- Superficial mycosis:
  - Tinea versicolor
  - Dermatophytosis
  - Mucocutaneous candidiasis

In conclusion

- Diagnosis and management of common viral infection:
  - Herpes infection
    - HSV
    - Varicella
    - Herpes zoster

THANK YOU