UMY UNIVERSITAS MUHAMMADIYAH YOGYAKARTA

INFEKSI BAKTERI PADA KULIT

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DERMATOVENEREOLOGI Dep. FKIK UMY

Do’a belajar

Asyhadu anlaa ilaaha illalloh
wa asyhadu anna Muhammadan rasuulullloh

Rodliitu billaahi robbaa
wa bil-islaami diinaa
wa bi Muhammadin nabiyyaw wa rosuulaa

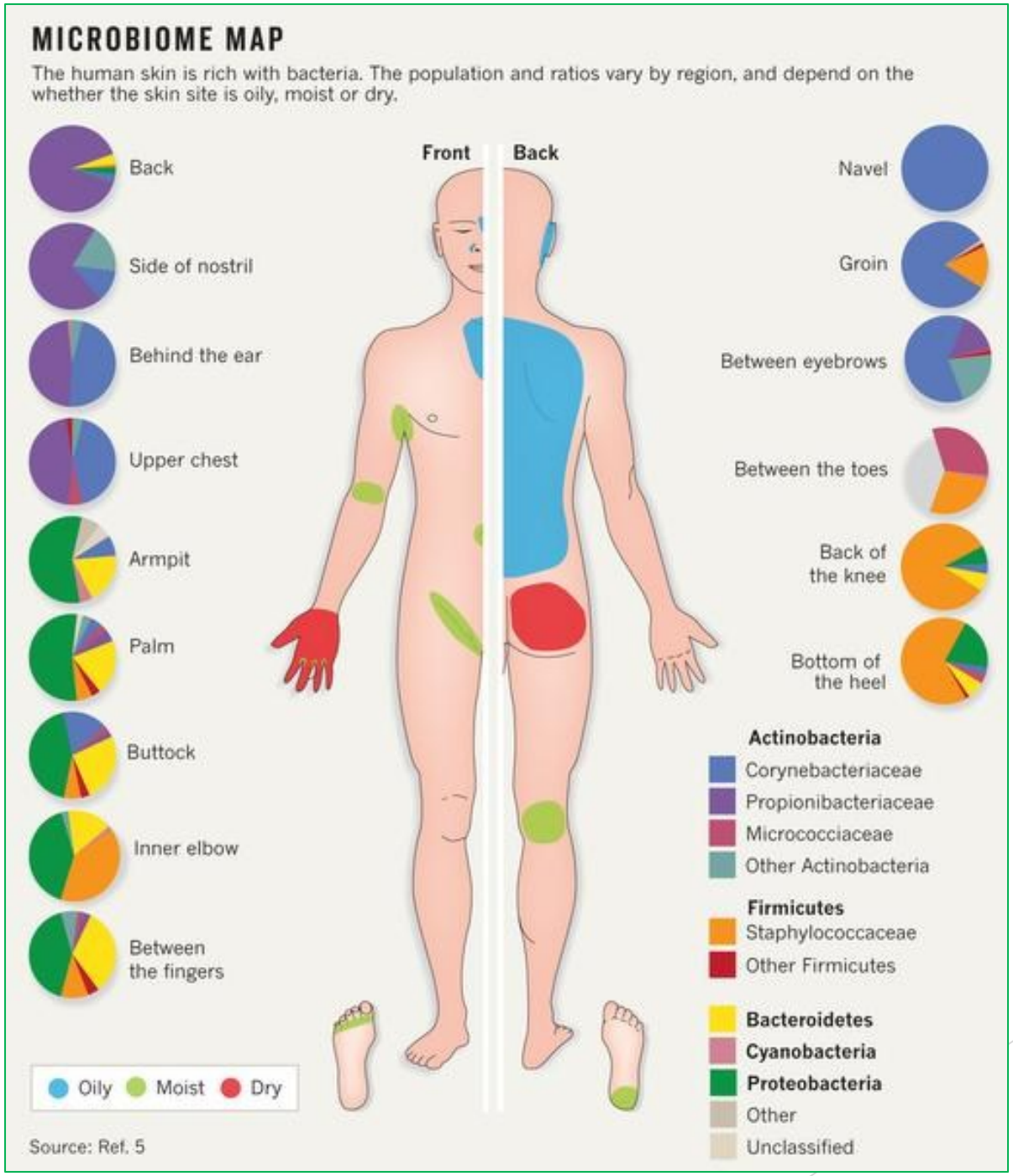
Robbi zidnii ‘ilmaa
warzuqnii fahmaa
Aamiin....

Tujuan Belajar

1. Mengetahui jenis penyakit infeksi bakteri pada kulit (SKDI 2012)
2. Mengetahui faktor penyebab dan faktor risiko serta patogenesis
3. Memahami cara penegakan diagnosis
4. Memahami cara melakukan (pengambilan sampel) dan interpretasi hasil pemeriksaan penunjang
5. Memahami cara penatalaksanaan

Jenis Infeksi Bakteri di Kulit

1. Infeksi primer
2. Infeksi sekunder
3. Erupsi/manifestasi infeksi primer di organ lain
4. Reaksi kulit (hipersensitivitas/alergi) terhadap infeksi di organ lain


MICROBIOME MAP
The human skin is rich with bacteria. The population and ratios vary by region, and depend on the whether the skin site is oily, moist or dry.
Back
Side of nostril
Behind the ear
Upper chest
Armpit
Palm
Buttock
Inner elbow
Between the fingers
Front
Back
Navel
Groin
Between eyebrows
Between the toes
Back of the knee
Bottom of the heel
Oily
Moist
Dry
Actinobacteria
Corynebacteriaceae
Propionibacteriaceae
Micrococciaceae
Other Actinobacteria
Firmicutes
Staphylococcaceae
Other Firmicutes
Bacteroidetes
Cyanobacteria
Proteobacteria
Other
Unclassified
Source: Ref. 5

Faktor yg berpengaruh terhadap Infeksi Bakteri di Kulit

Portal of Entry –

disruption of skin barrier.

Natural Resistance of The Skin –

Free fatty acid, Linoleic acid; Sphingosine, Glucosylceramides, Hexadeconic acid.

Antimicrobial peptides (AMP) in the lamellar bodies → insertion of bacterial membrane:

Cathelicidins & Defensins

Host inflammatory responses–

AMP;

Toll like receptor;

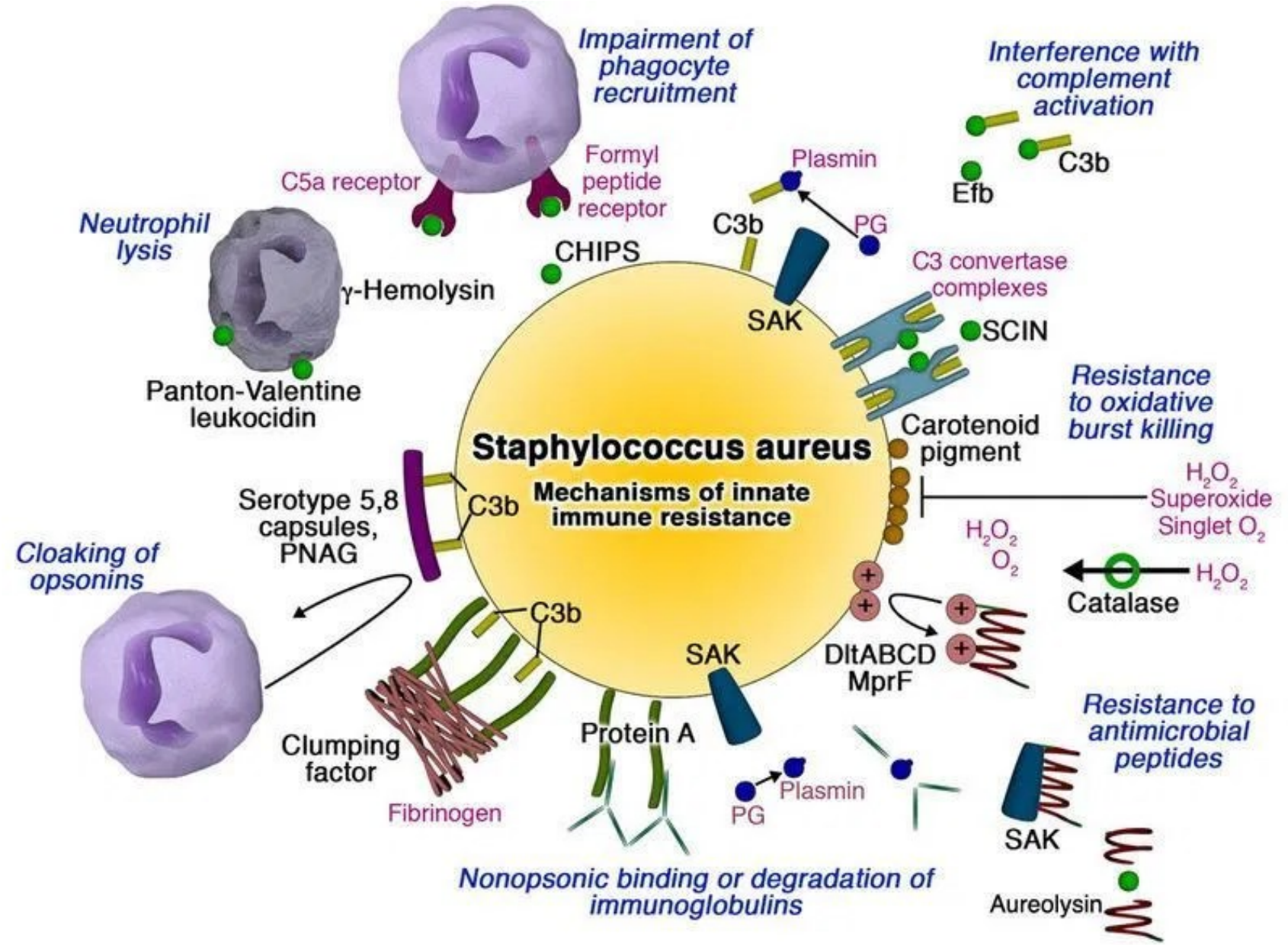
Complement : mannin-binding lectin.

Pathogenicity of Microorganism -

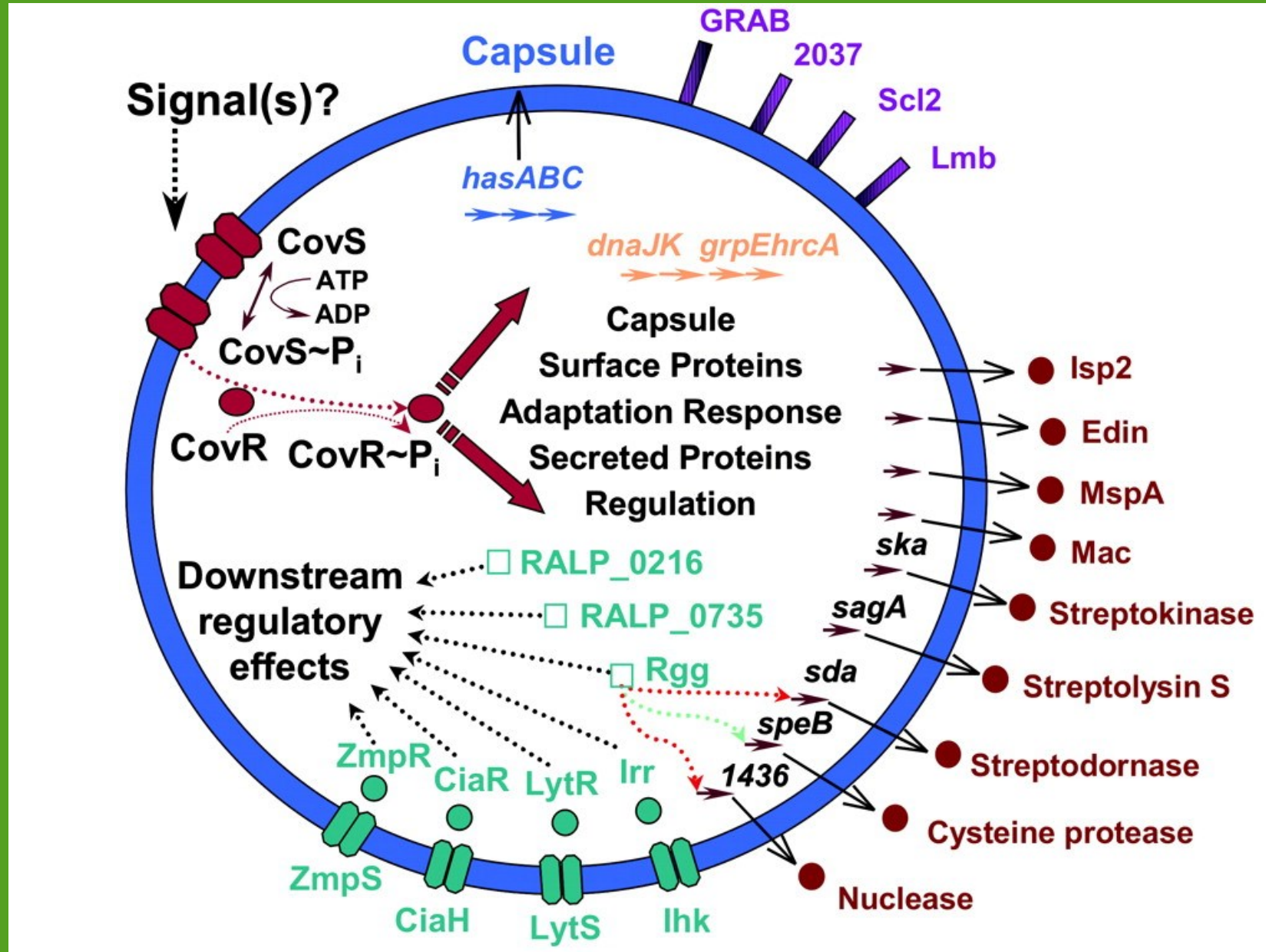
Cytotoxin & enzymes → direct invasion & lysis of protein, receptor.

Aureoly-sin A, Staphylokinase, Protease, α toxin, etc.

M protein, Streptokinase, Streptolysin, etc.


Impairment of phagocyte recruitment
C5a receptor
Formyl peptide receptor
Neutrophil lysis
γ-Hemolysin
Panton-Valentine leukocidin
CHIPS
Plasmin
C3b
PG
SAK
Interference with complement activation
C3b
Efb
C3 convertase complexes
SCIN
Resistance to oxidative burst killing
Carotenoid pigment
H_2O_2
Superoxide
Singlet O_2
H_2O_2
O_2
H_2O_2
Catalase
DltABCD
MprF
Staphylococcus aureus
Mechanisms of innate immune resistance
Serotype 5,8 capsules, PNAG
C3b
Cloaking of opsonins
C3b
SAK
Protein A
Clumping factor
Fibrinogen
Plasmin
PG
Resistance to antimicrobial peptides
SAK
Aureolysin
Nonopsonic binding or degradation of immunoglobulins

Streptococcus sp.



Capsule
- antiphagocytic factor

Surface Proteins
- environmental sensors
- protease modulation
- proteases
- adhesins

Adaptation Response
- chaperones
- transport proteins
- amino acid degradation
- nucleotide metabolism

Secreted Proteins
- cytolysins
- host modulation
- antiphagocytic factors
- nucleases

Regulation
- nutrient adaptation
- PG modifications
- virulence attenuation

PREDISPOSING FACTORS

- Chronic *S. aureus* carrier state (nares, axillae, perineum, vagina)
- Diabetes mellitus
- Obesity
- Poor hygiene
- Bactericidal defects (e.g., chronic granulomatous disease)
- Chemotactic defects
- Hyper-IgE syndrome (Job's syndrome)
- HIV disease, especially MRSA infection

BOX 176-1 INFECTIONS AND TOXIN SYNDROMES INVOLVING THE SKIN AND SOFT TISSUES CAUSED BY *STAPHYLOCOCCUS AUREUS*

SITES OF COLONIZATION (CARRIER STATE)

- Anterior nares
- Throat
- Axillae, perineum
- Hands
- Involved skin in individuals with atopic dermatitis

SITES OF COLONIZATION IN NEONATES (AND SITES OF INFECTION)

- Skin
- Umbilicus
- Circumcision site
- Conjunctivae

SUPERFICIAL PYODERMAS

- Primary pyodermas
  - Skin
    - Impetigo
    - Bullous impetigo
    - Erythema
    - Botryomycosis
  - Hair follicles
    - Superficial folliculitis (follicular or Bockhart impetigo)
    - Folliculitis (sycosis barbae)
    - Furuncle (boil)
    - Carbuncle
  - Intertriginous sites
    - Perianal dermatitis
  - Digital infections
    - Paronychia
    - Blistering distal dactylitis
  - After skin disruption
    - Trauma (physical, thermal)
    - Foreign body (intravascular catheter, prosthetic device)
- Secondary pyodermas
  - Impetiginization of dermatoses such as atopic dermatitis, herpes simplex (superinfection)
  - Pyodermas associated with systemic disease
  - Job syndrome
  - Chédiak–Higashi syndrome
  - Chronic granulomatous disease

INVASIVE INFECTIONS

- Lymphangitis, lymphadenitis
- Erysipelas
- Cellulitis
- Streptococcal gangrene
- Pyomyositis
- Bacteremia, septicemia

METASTATIC SKIN INFECTIONS ASSOCIATED WITH BACTEREMIA (OFTEN *S. aureus* ACUTE INFECTIONS ENDOCARDITIS)

- Abscesses (superficial and deep)
- Septic vasculitis (pustular purpura)

PURPURA FULMINANS

- Disseminated intravascular coagulation associated with staphylococcal bacteremia
- Meningococcemia-like syndrome

STAPHYLOCOCCAL TOXIN-ASSOCIATED SYNDROMES

- Staphylococcal scarlet fever
- Staphylococcal scalded-skin syndromes
- Staphylococcal toxic-shock syndrome

BOX 176-8 INFECTIONS AND TOXIN SYNDROMES CAUSED BY GROUP A *STREPTOCOCCI*

SUPERFICIAL PYODERMAS

- Nonintertriginous skin
  - Impetigo
  - Ecthyma
  - Blistering distal dactylitis
- Intertriginous skin
  - Perianal streptococcal cellulites
  - Streptococcal vulvovaginitis
  - Streptococcal intertrigo

INVASIVE INFECTIONS

- Acute lymphangitis
- Erysipelas
- Cellulitis
- Streptococcal gangrene
- Bacteremia, septicemia

TOXIN-ASSOCIATED SYNDROME

- Scarlet fever
- Streptococcal toxic-shock-like syndrome
- Streptococcal gangrene

NONSUPPURATIVE COMPLICATIONS

- Rheumatic fever
- Glomerulonephritis

OTHER ASSOCIATED CUTANEOUS REACTION PATTERNS

- Erythema nodosum
- Erythema multiforme
- Guttate-pattern psoriasis
- Vasculitis

SKDI 2012

Infeksi Bakteri		
8	Impetigo	4A
9	Impetigo ulseratif (ektima)	4A
10	Folikulitis superfisialis	4A
11	Furunkel, karbunkel	4A
12	Eritrasma	4A
13	Erisipelas	4A
14	Skrofuloderma	4A
15	Lepra	4A
16	Reaksi lepra	3A
17	Sifilis stadium 1 dan 2	4A

Infeksi Bakteri di SNPPDI 2019

11	Impetigo bullosa dan krustosa	4
12	Ektima	4
13	Folikulitis superfisialis	4
14	Paronikhia piogenik -	4
15	Furunkel, karbunkel	4
16	Folikulitis profunda	2
17	Selulitis	3A
18	Ulkus piogenik	2
19	Eritrasma	3A
20	Erisipelas	3A
21	TB kutis (termasuk skrofuloderma)	3A
22	Lepra tanpa komplikasi	4
23	Reaksi lepra	3A
24	Sifilis primer dan laten	4
25	Sifilis sekunder dan sifilis dengan penyulit	3A
26	Scarlet fever	2

INFEKSI (PRIMER) BAKTERI PADA KULIT

- Klasifikasi berdasar penyebab :
  - Staphylococcus sp. → exfoliative toxin → desmoglein
  - Streptococcus sp. → Streptolysin → cytolisis

- Klasifikasi berdasar manifestasi klinis (struktur kulit yg terinfeksi) - Pyoderma :

1. Impetigo bulosa & krustosa
2. Ektima
3. Folikulits
4. Furunkel
5. Karbunkel

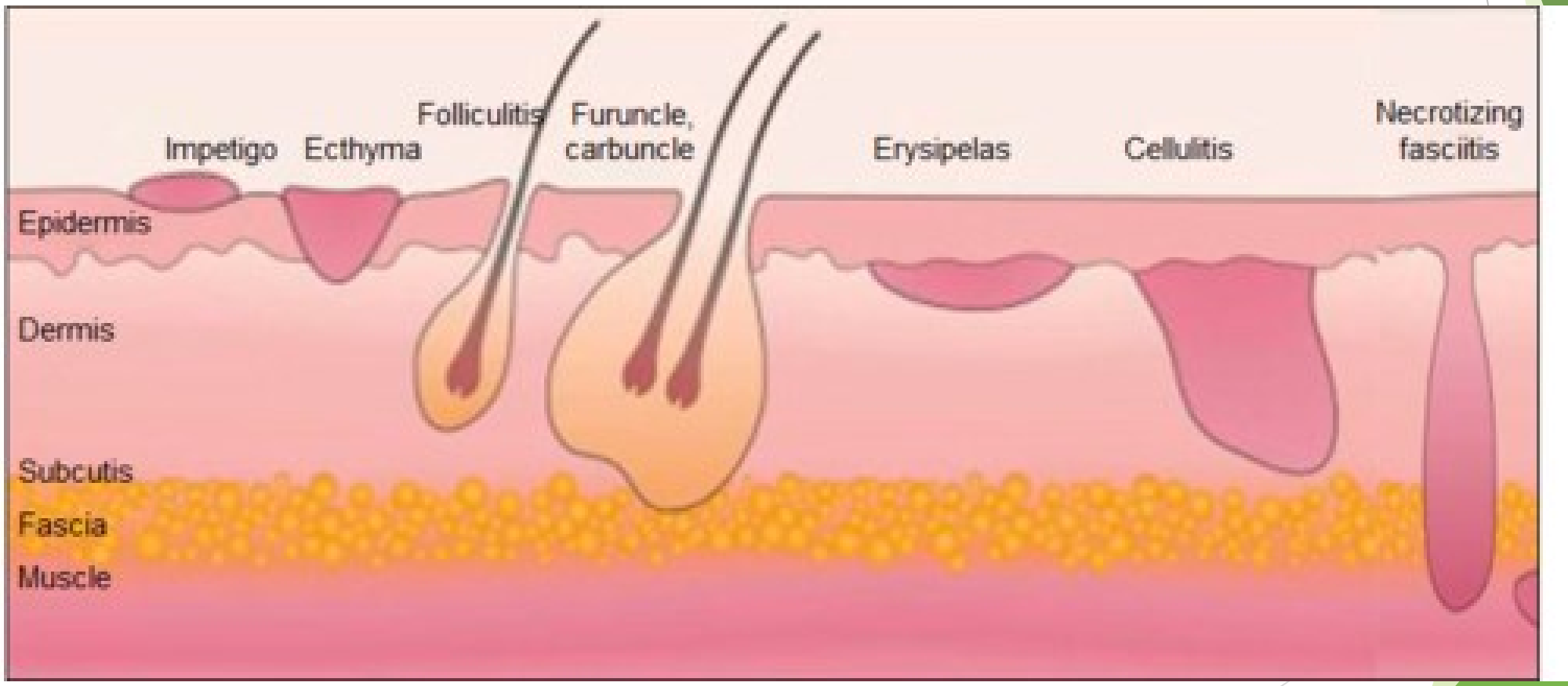
C. minutissimun :
1. Erythrasma

Digital infection :
1. Paronychia

Invasive infection :
1. Erysipelas
2. Cellulitis

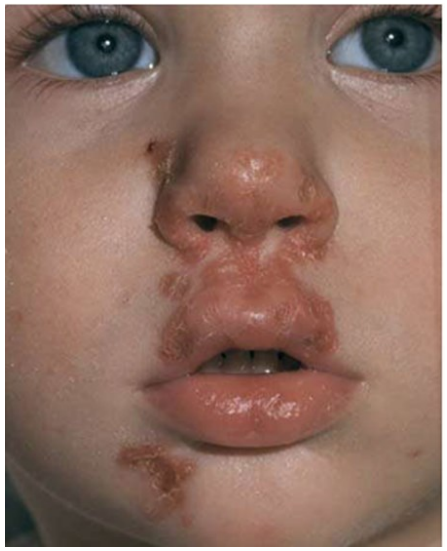
Staph toxin-associated syndromes:
1. Scarlet fever

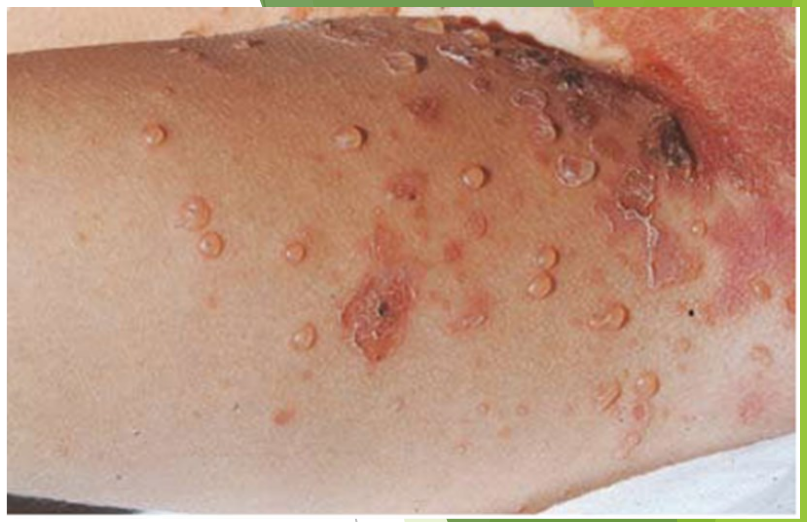
Map of Pyoderma



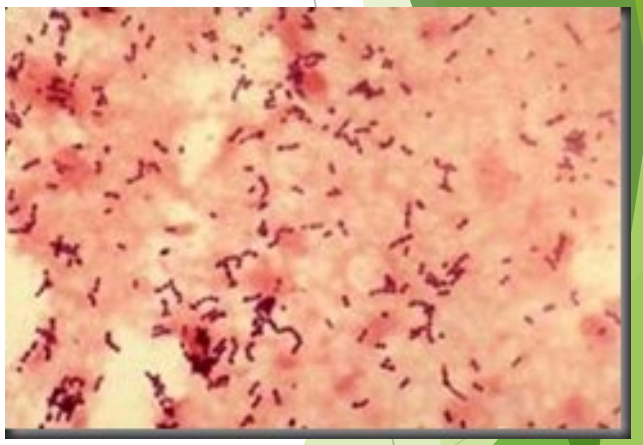
- Impetigo nonbulosa (Impetigo krustosa)
  - Predileksi: daerah wajah, terutama di sekitar nares dan mulut.
  - Lesi awal berupa makula atau papul eritematosa yang secara cepat berkembang menjadi vesikel atau pustul yang kemudian pecah membentuk krusta kuning madu (*honey colour*) dikeliling eritema. Lesi dapat melebar sampai 1-2 cm, disertai lesi satelit di sekitarnya.
  - Rasa gatal dan tidak nyaman dapat terjadi.
- Impetigo bulosa
  - Predileksi: daerah intertriginosa (aksila, inguinal, gluteal), dada dan punggung.
  - Vesikel-bula kendur,dapat timbul bula hipopion.
  - Tanda Nikolsky negatif.
  - Bula pecah meninggalkan skuama anular dengan bagian tengah eritematosa (kolaret) dan cepat mengering.
- Ektima
  - Merupakan bentuk pioderma ulseratif yang disebabkan oleh *S. aureus* dan atau *Streptococcus* grup A.
  - Predileksi: ekstremitas bawah atau daerah terbuka.
  - Ulkus dangkal tertutup krusta tebal dan lekat, berwarna kuning keabuan.

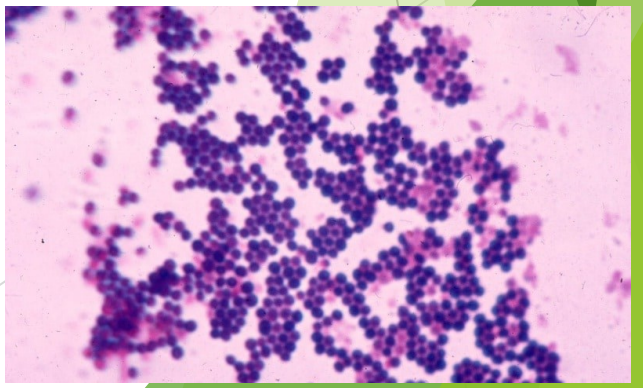
Impetigo → epidermis

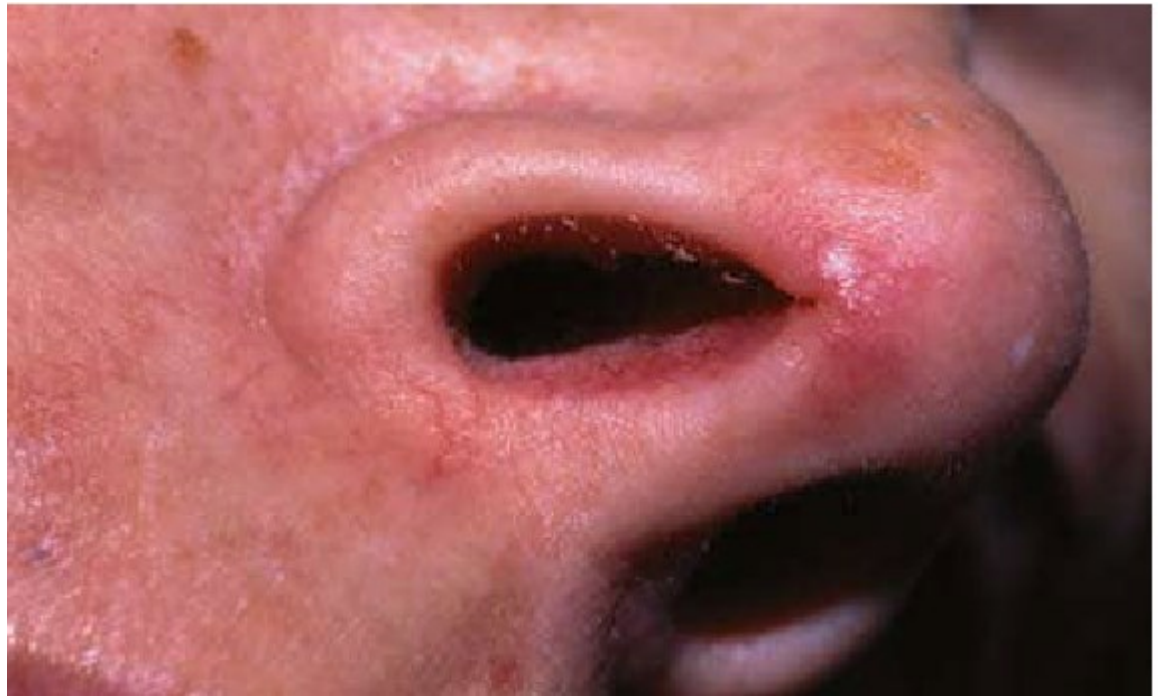




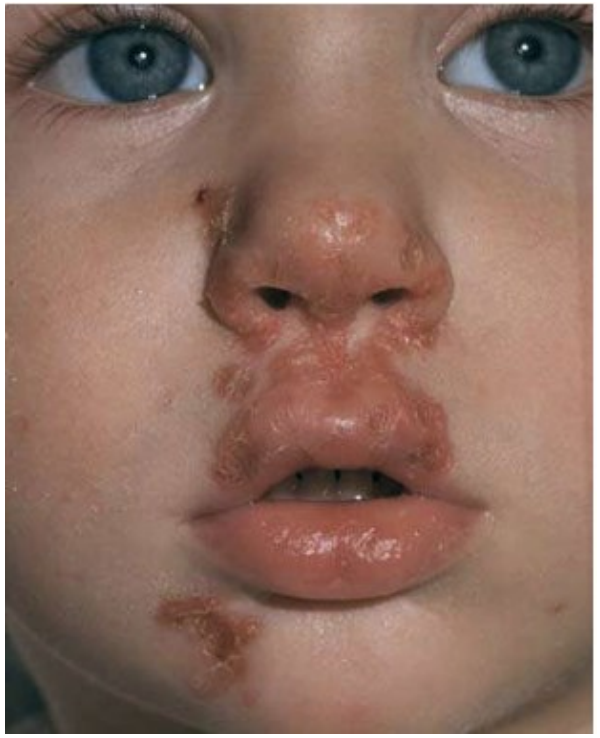
- Epidemiologi:
  - Bayi dan anak
- Ax. Asimtomatik ?
- Pemeriksaan fisik
  - Impetigo krustosa : erosi dg krusta kekuningan
  - Impetigo bulosa :vesikel, bula, erosi
  - Gx sistemik (-)
- Pemeriksaan penunjang
  - Sampel kulit : eksudat/pus → pengecatan Gram: kokus ungu berderet (Steptococcus sp) atau bergerombol (Staphylococcus sp.)

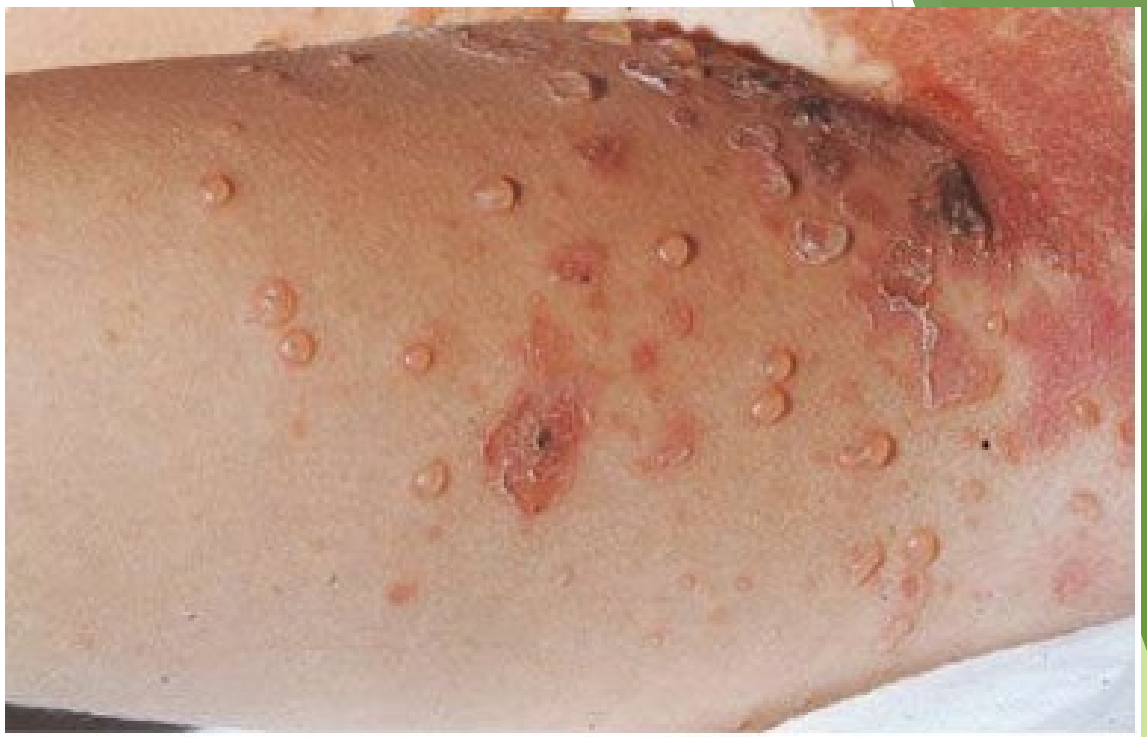




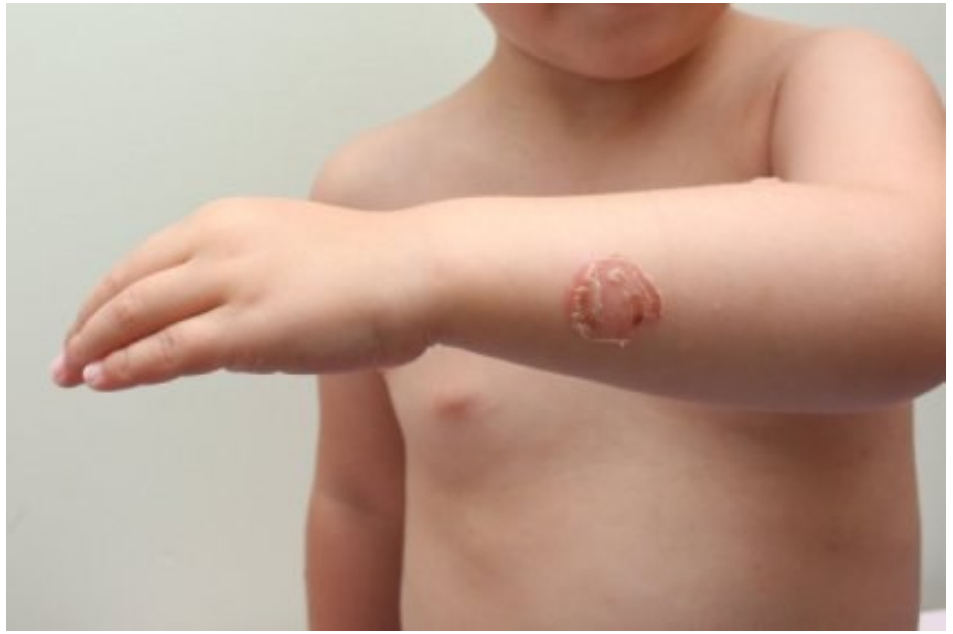


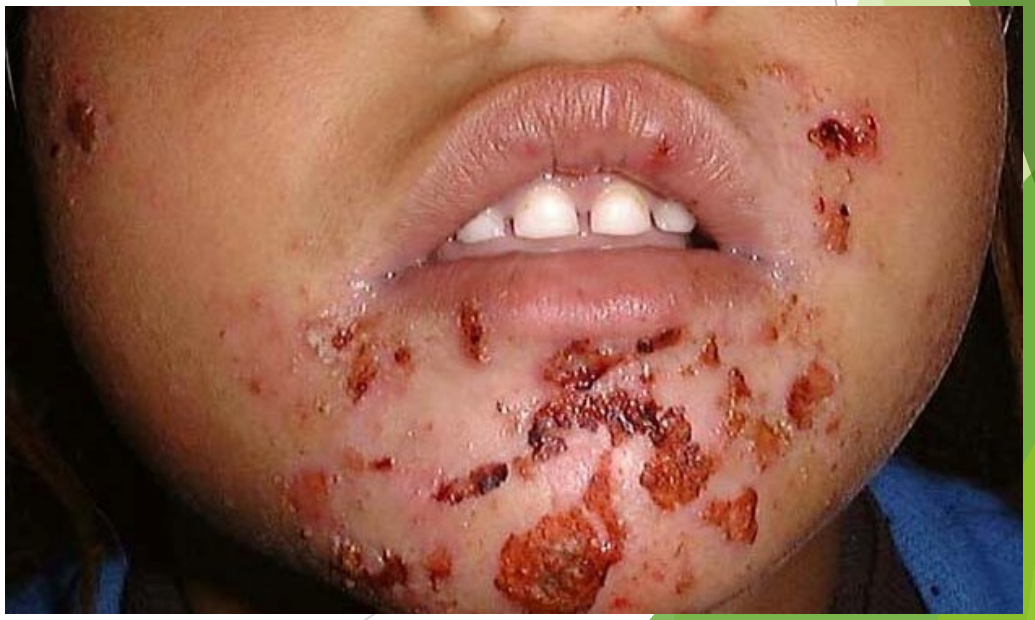
- Impetigo: *S. aureus nasal colonization Colonization of the nares in usually asymptomatic. This patient had* ***tenderness and erythema*** *of the skin adjacent to the nares* indicative of superficial infection rather than colonization.





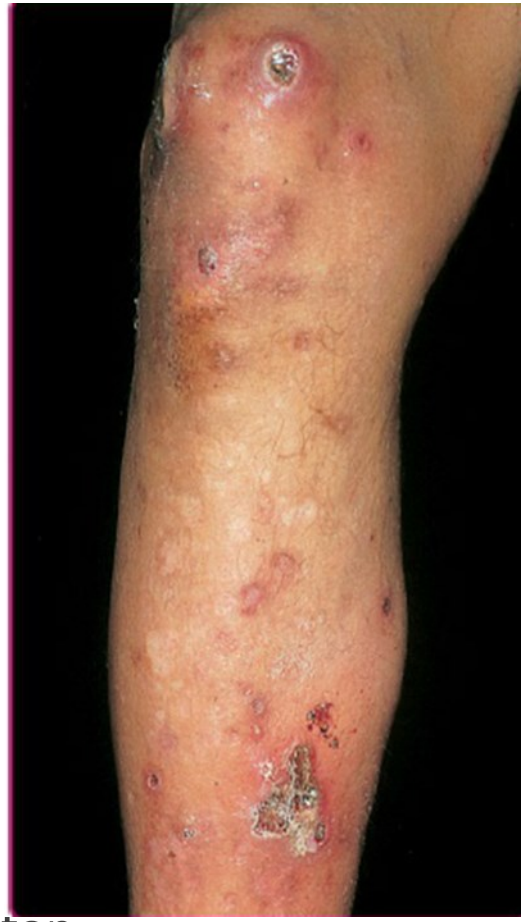
- Impetigo: variable pruritus, especially associated with atopic dermatitis.
- Ecthyma: pain, tenderness.

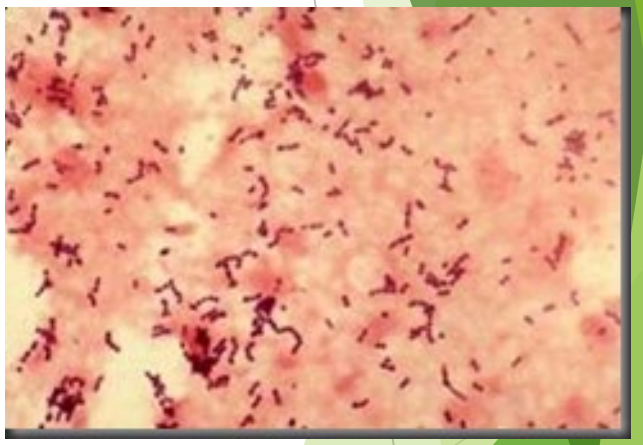


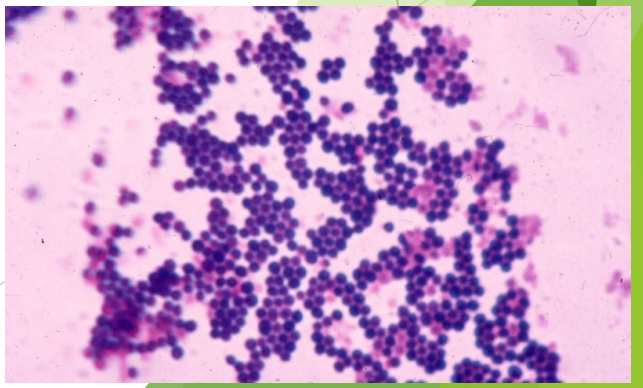


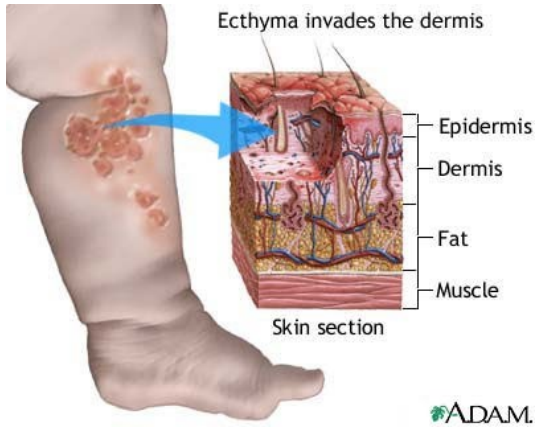
Ektima → epidermis & dermis

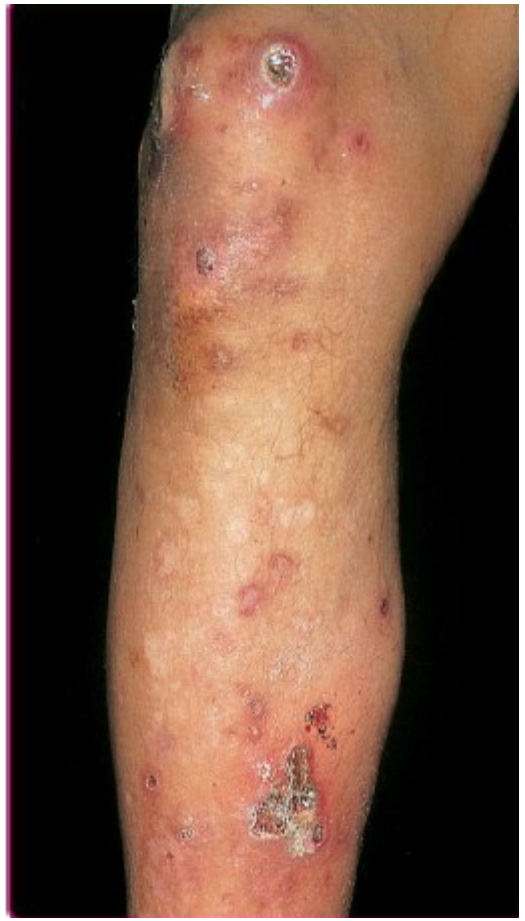
- Epidemiologi:
  - Anak & Dewasa
- Ax. Gatal & nyeri
- Pemeriksaan fisik
  - Ulkus, pustulasi, kulit sekitar eritem, odem
  - Krusta coklat - hitam
  - Gx sistemik: demam+-, pembesaran limfonodi
- Pemeriksaan penunjang
  - Sampel kulit : eksudat/pus → pengecatan Gram: kokus ungu berderet atau bergerombol

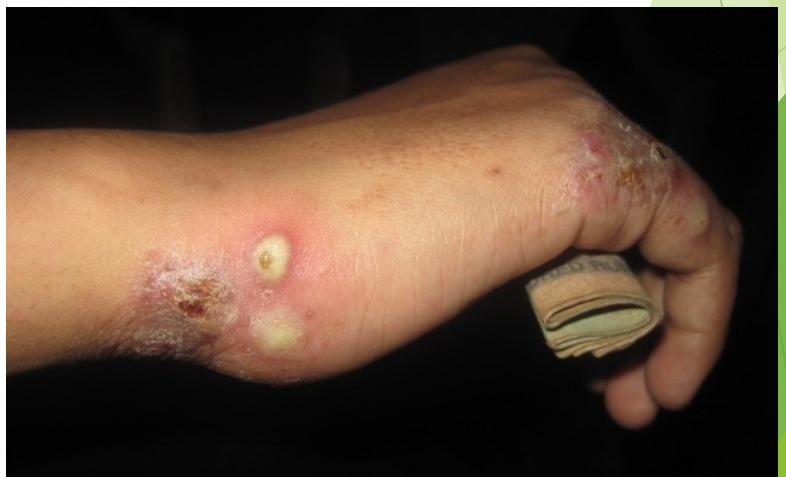




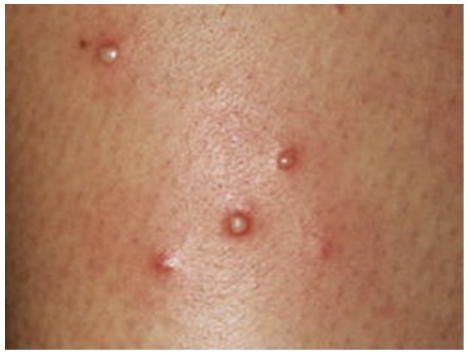


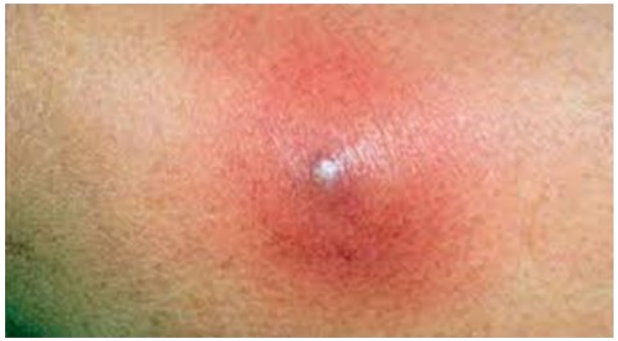

Ecthyma invades the dermis
Epidermis
Dermis
Fat
Muscle
Skin section
A.D.A.M.

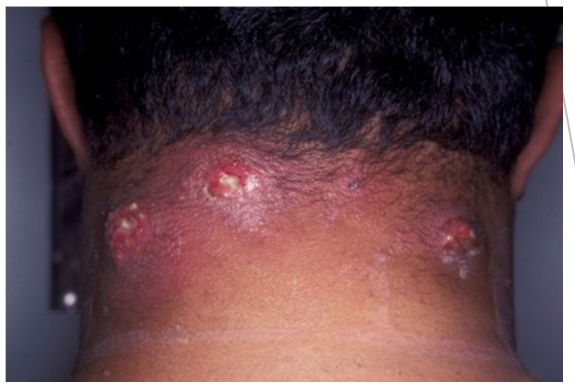




Folikulitis, Furunkel, Karbunkel → Folikel rambut; Abses





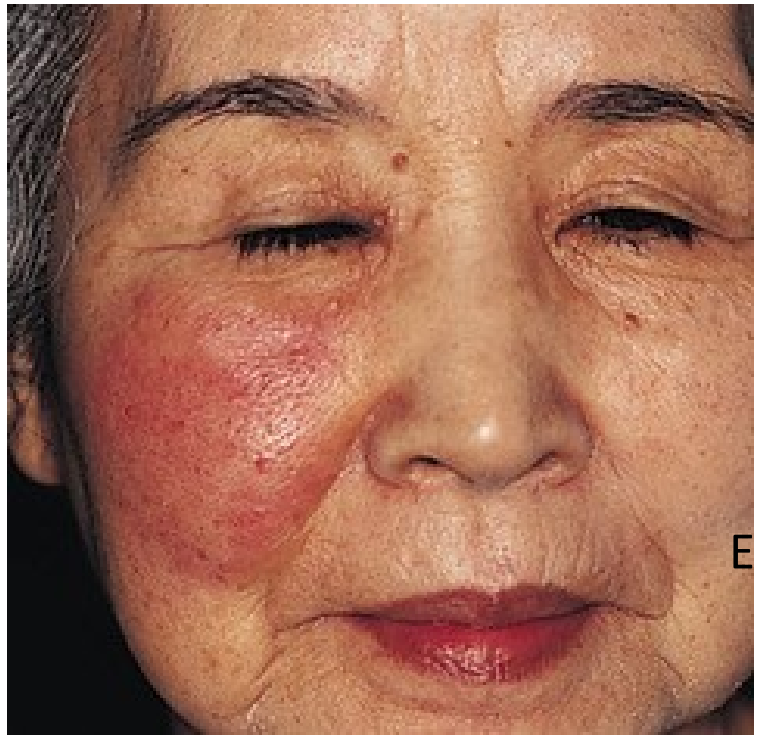


- *A furuncle is an acute, deep-seated, red, hot,* tender nodule or abscess that evolves from a staphylococcal folliculitis.
- A *carbuncle is a deeper infection composed of interconnecting abscesses usually arising* in several contiguous hair follicles.
- An *abscess is an acute or chronic localized inflammation, associated with a collection of pus and tissue destruction*.
- Ax. Gatal – nyeri
- Pemeriksaan fisik
  - Gejala sistemik : demam
  - Papul folikuler – nodul eritem, punctum (soliter – multiple), odem, nyeri tekan+

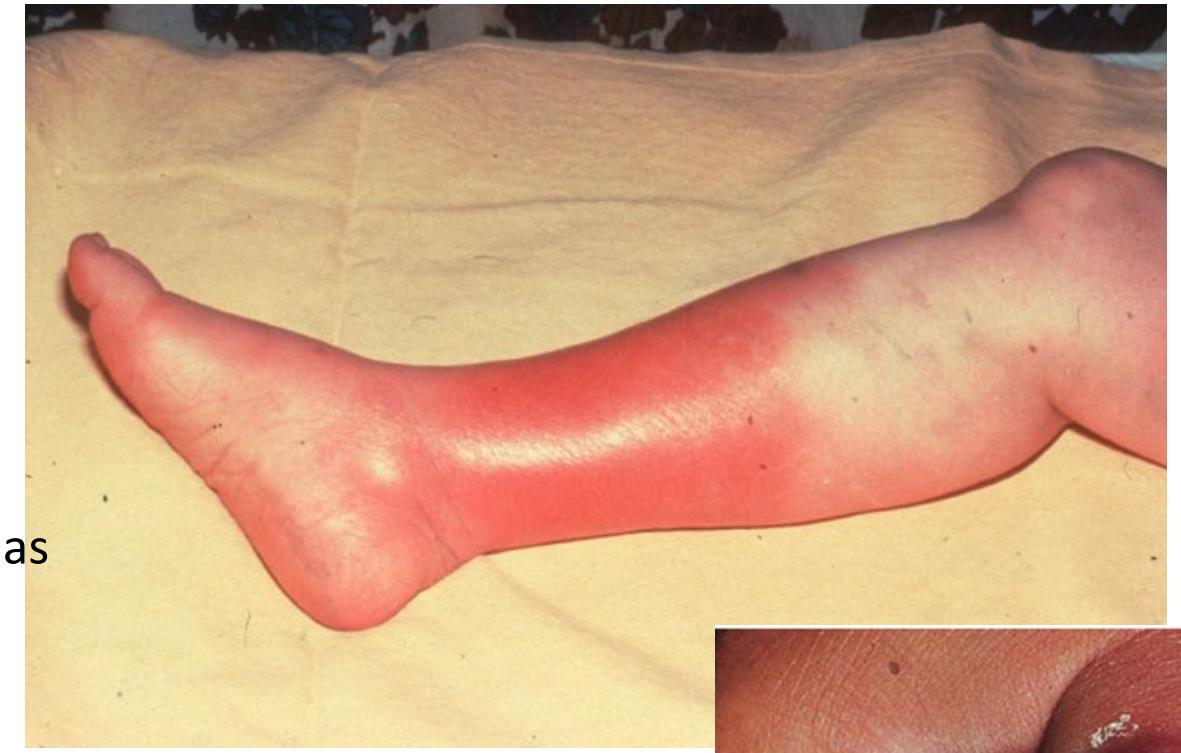
- Folikulitis
  Merupakan salah satu bentuk pioderma pada folikel rambut.
  Dibedakan menjadi 2 bentuk:
  - Folikulitis superfisialis (impetigo Bockhart/impetigo folikular)
    Predileksi: skalp (anak-anak), dagu, aksila, ekstremitas bawah, bokong (dewasa).
    Terdapat rasa gatal dan panas.
    Kelainan berupa pustul kecil *dome-shaped*, multipel, mudah pecah pada folikel rambut.
  - Folikulitis profunda (*sycosis barbae*)
    Predileksi: dagu, atas bibir.
    Nodus eritematosa dengan perabaan hangat, nyeri.
- Furunkel/karbunkel
  - Merupakan infeksi pada folikel rambut dan jaringan sekitarnya.
  - Predileksi: daerah berambut yang sering mengalami gesekan, oklusif, berkeringat, misalnya leher, wajah, aksila, dan bokong.
  - Lesi berupa nodus eritematosa, awalnya keras, nyeri tekan, dapat membesar 1-3 cm, setelah beberapa hari terdapat fluktuasi, bila pecah keluar pus.
  - Karbunkel timbul bila yang terkena beberapa folikel rambut. Karbunkel lebih besar, diameter dapat mencapai 3-10 cm, dasar lebih dalam. Nyeri dan sering disertai gejala konstitusi. Pecah lebih lambat, bila sembuh dapat meninggalkan jaringan parut.

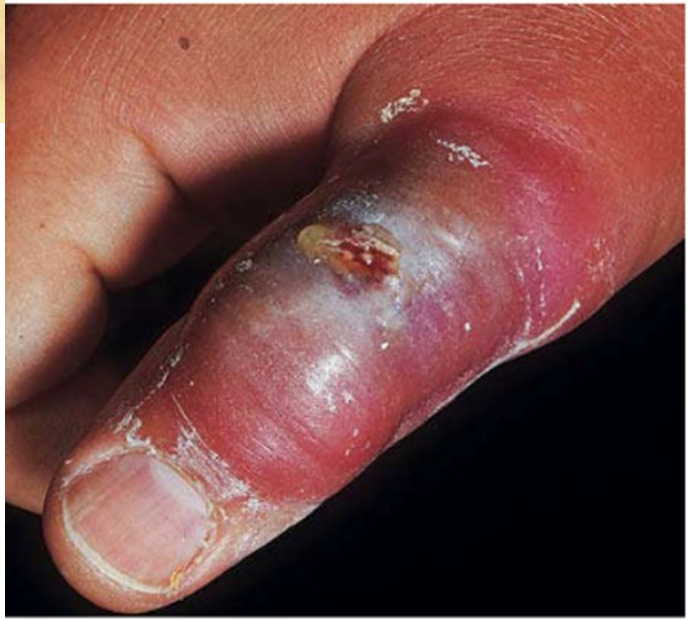
PIODERMA PROFUNDA

- Terdapat gejala konstitusi dan rasa nyeri.
- Terdiri atas:
  - Erisipelas: lesi eritematosa merah cerah, infiltrat di bagian pinggir, edema, vesikel dan bula di atas lesi.
  - Selulitis: infiltrat eritematosa difus.
  - Flegmon: selulitis dengan supurasi.
  - Abses kelenjar keringat: tidak nyeri, bersama miliaria, nodus eritematosa bentuk kubah.
  - Hidradenitis: nodus, abses, fistel di daerah ketiak atau perineum.
  - Ulkus piogenik: ulkus dengan pus.

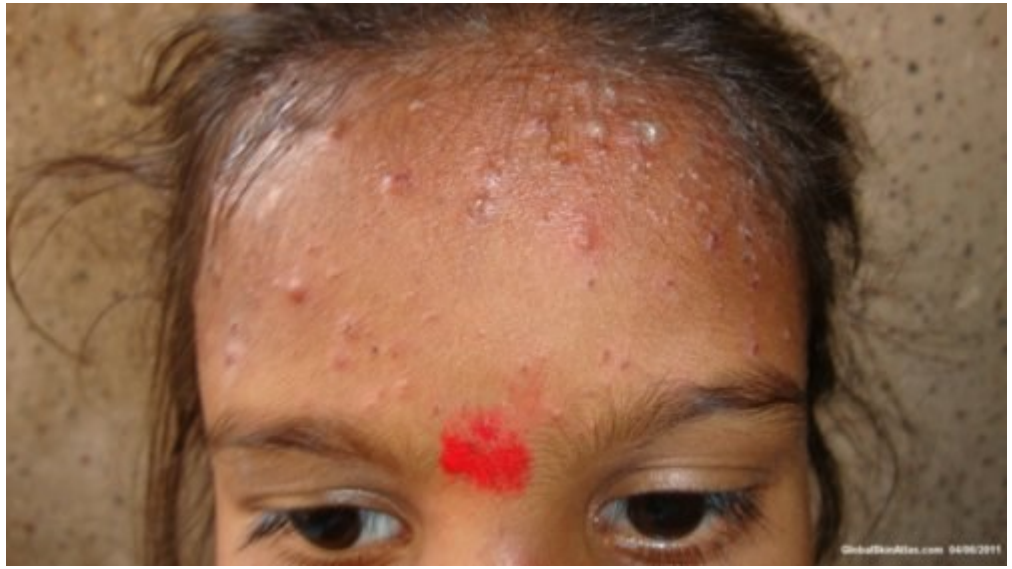


Erisipelas

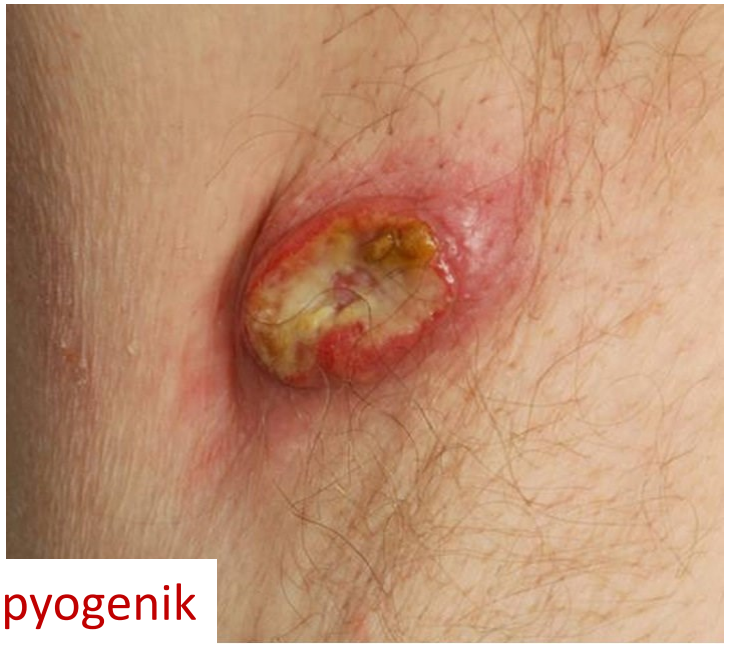




Abses kelenjar keringat

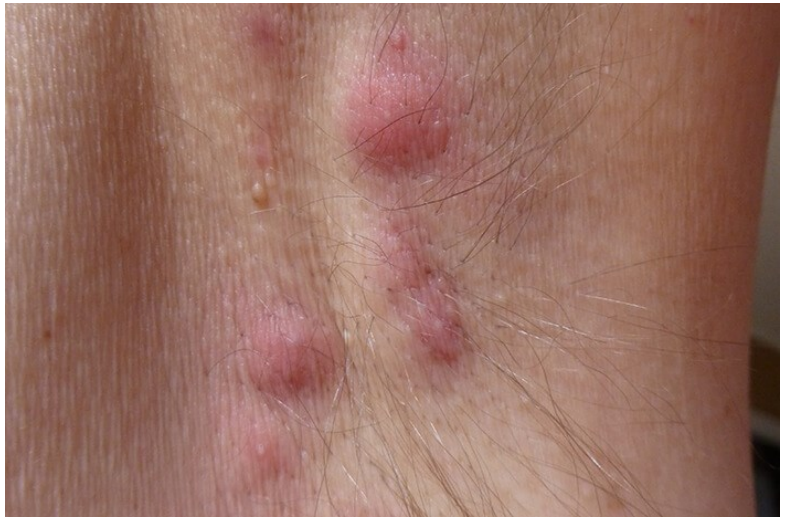


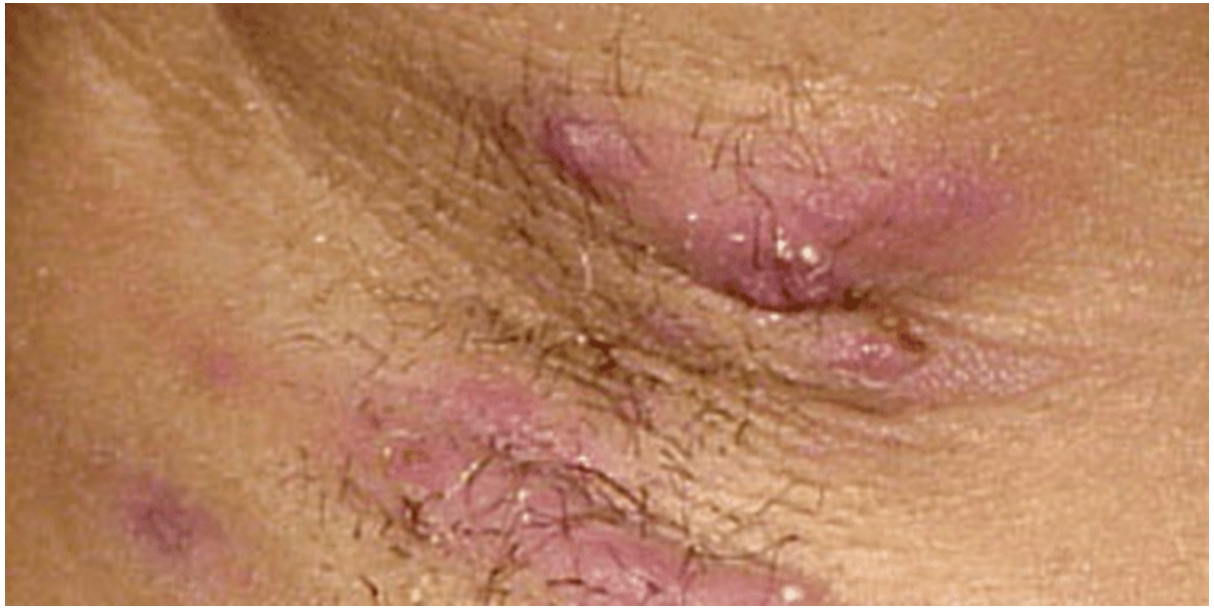
Selulitis



Ulkus pyogenik

Hidradenitis supurativa (kel. apokrin





KOMPLIKASI

Komplikasi[8-11]

- Impetigo non-bulosa: glomerulonefritis akut
- Ektima: ulserasi dan skar
- Komplikasi lainnya yang jarang: sepsis, osteomielitis, artritis, endokarditis, pneumonia, selulitis, limfangitis, limfadenitis, *toxic shock syndrome*, *Staphylococcal scalded skin syndrome, necrotizing fasciitis*.

DIFERENSIAL DIAGNOSIS

Diagnosis Banding[6,8-11]

1. Impetigo nonbulosa: ektima, dermatitis atopik, dermatitis seboroik, dermatitis kontak alergi, skabies, tinea kapitis
2. Impetigo vesikobulosa: dermatitis kontak, *Staphylococcal scalded skin syndrome*, pemfigoid bulosa, pemfigus vulgaris, eritema multiforme, dermatitis herpetiformis
3. Ektima: impetigo nonbulosa
4. Folikulitis: tinea barbae, tinea kapitis, folikulitis keloidal (*acne keloidal nuchae*), folikulitis pitirosporum, "*Hot tub*" folikulitis, folikulitis kandida
5. Furunkel, karbunkel: akne kistik, kerion, hidradenitis supurativa
6. Selulitis/erisipelas: dermatitis kontak, dermatitis stasis, necrotizing fasciitis, tuberkulosis kutis verukosa, infeksi mikobakterium atipik, mikosis profnda, leismaniasis, *deep vein thrombosis*, limfedema, vaskulitis leukositoklastik, pioderma ganggrenosum, gout, *paget disease*
7. Hidradenitis: skrofuloderma

PEMERIKSAAN PENUNJANG

Pemeriksaan Penunjang

Bila diperlukan[6-11]

1. Pemeriksaan sederhana dengan pewarnaan Gram. (A,2)
2. Kultur dan resistensi spesimen lesi/aspirat apabila tidak responsif terhadap pengobatan empiris. (A,2)
3. Kultur dan resistensi darah, darah perifer lengkap, kreatinin, *C-reactive protein* apabila diduga bakteremia. (A,2)
4. Biopsi apabila lesi tidak spesifik. (A,2)

PENATALAKSANAAN

Non medikamentosa

1. Mandi 2 kali sehari dengan sabun
2. Mengatasi/identifikasi faktor predisposisi dan keadaan komorbid, misalnya infestasi parasit, dermatitis atopik, edema, obesitas dan insufisiensi vena. (A,2)

Medikamentosa

Prinsip: pasien berobat jalan, kecuali pada erisipelas, selulitis dan flegmon derajat berat dianjurkan rawat inap. Terdapat beberapa obat/tindakan yang dapat dipiih sesuai dengan indikasi sebagai berikut:

1. Topikal
   - Bila banyak pus atau krusta: kompres terbuka dengan permanganas kalikus 1/5000, asam salisilat 0,1%, rivanol 1‰, larutan povidon iodine 1%; dilakukan 3 kali sehari masing-masing ½-1 jam selama keadaan akut. (D,5)
   - Bila tidak tertutup pus atau krusta: salep/krim asam fusidat 2%, mupirosin 2% (A,1). Dioleskan 2-3 kali sehari, selama 7-10 hari.
2. Sistemik: minimal selama 7 hari

   Lini pertama:
   - Kloksasilin/dikloksasilin**: dewasa 4x250-500 mg/hari per oral; anak-anak 25-50 mg/kgBB/hari terbagi dalam 4 dosis (A,1)
   - Amoksisilin dan asam klavulanat: dewasa 3x250-500 mg/hari; anak-anak 25 mg/kgBB/hari terbagi dalam 3 dosis (A,2)
   - Sefaleksin: 25-50 mg/kgBB/hari terbagi dalam 4 dosis. (A,2)

   Lini kedua:
   - Azitromisin 1x500 mg/hari (hari 1), dilanjutkan 1x250 mg (hari 2-5) (D,5)
   - Klindamisin 15 mg/kgBB/hari terbagi 3 dosis. (A,2)
   - Eritromisin: dewasa 4x250-500 mg/hari; anak-anak 20-50 mg/kgBB/hari

Penyebabnya MRSA:

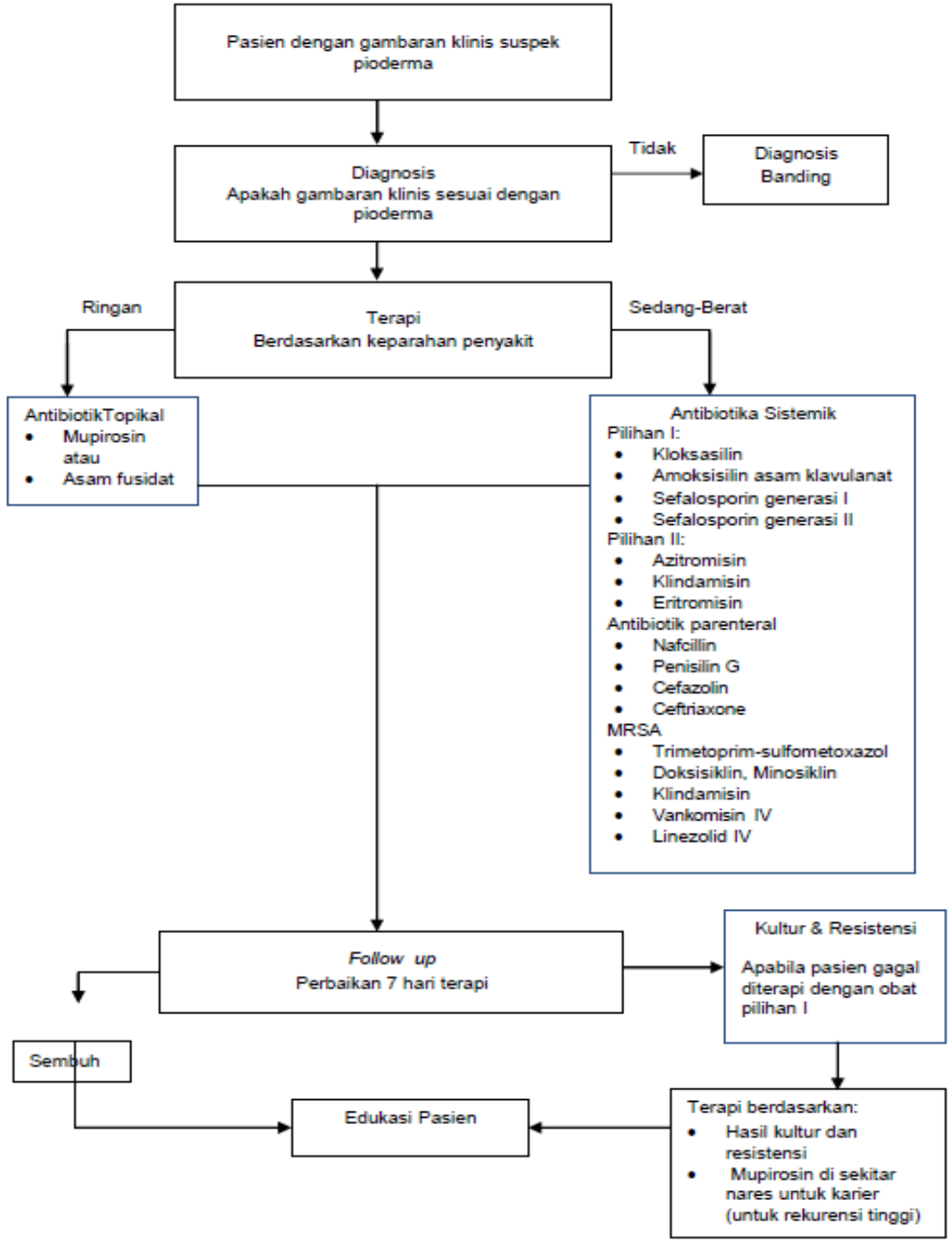
- Trimetoprim-sulfometoxazol 160/800 mg, 2 kali sehari. (A,2)
- Doksisiklin, minosiklin 2x100 mg, tidak direkomendasikan untuk anak, usia 8 tahun. (A,2)
- Klindamisin 15 mg/kgBB/hari terbagi 3 dosis. (A,2)

Kasus yang berat, disertai infeksi sitemik atau infeksi di daerah berbahaya (misalnya maksila), antibiotik diberikan parenteral.

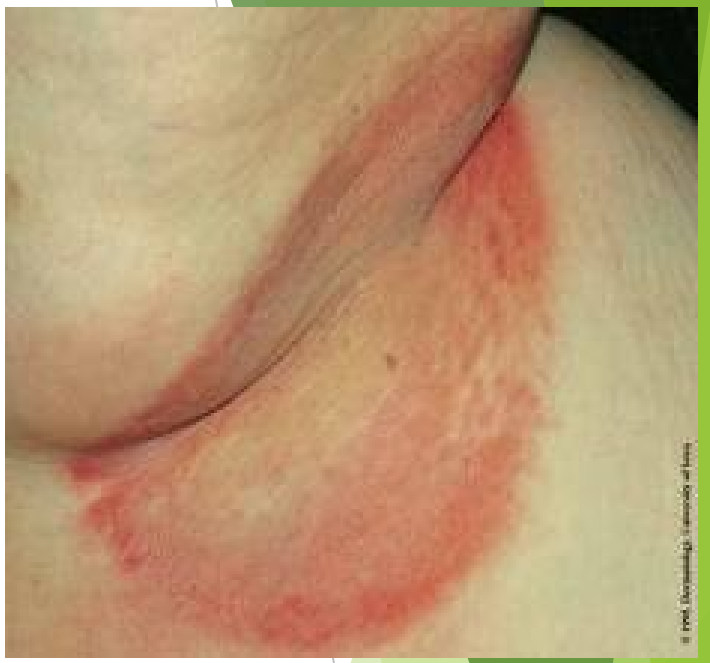
- Nafcillin 1-2 gram IV tiap 4 jam, anak 100-150 mg/kgBB/hari terbagi dalam 4 dosis. (A,2)
- Penisilin G 2-4 juta unit IV tiap 4-6 jam, anak: 60-100.000 unit/kgBB tiap 6 jam. (A,2)
- Cefazolin IV 1 gram tiap 8 jam, anak: 50 mg/kgbb/hari dibagi dalam 3 dosis. (A,2)
- Ceftriaxone IV 1-2 gram ,1 kali/hari. (A,2)
- Apabila terdapat/dicurigai ada *methycillin resistant Staphylococcus aureus* (MRSA) pada infeksi berat: vankomisin 1-2 gram/hari dalam dosis terbagi atau 15-20 mg/kgBB setiap 8-12 jam intravena, selama 7-14 hari (A,1). Anak: vankomisin 15 mg/kgBB IV tiap 6 jam. (A,2)
- Linezolid 600 mg IV atau oral 2 kali sehari selama 7-14 hari (A,1), anak-anak 10 mg/kgBB oral atau intravena tiap 8 jam. (A,2)
- Klindamisin IV 600 mg tiap 8 jam atau 10-13 mg/kgBB tiap 6-8 jam. (A,2)
- Kasus rekuren, diberikan antibiotik berdasarkan hasil kultur dan resistensi.

Tindakan[7,9]

Apabila lesi abses besar, nyeri, disertai fluktuasi, dilakukan insisi dan drainase. (A,1)


Pasien dengan gambaran klinis suspek pioderma
Diagnosis
Apakah gambaran klinis sesuai dengan pioderma
Tidak
Diagnosis Banding
Terapi
Berdasarkan keparahan penyakit
Ringan
Sedang-Berat
AntibiotikTopikal
Mupirosin
atau
Asam fusidat
Antibiotika Sistemik
Pilihan I:
Kloksasilin
Amoksisilin asam klavulanat
Sefalosporin generasi I
Sefalosporin generasi II
Pilihan II:
Azitromisin
Klindamisin
Eritromisin
Antibiotik parenteral
Nafcillin
Penisilin G
Cefazolin
Ceftriaxone
MRSA
Trimetoprim-sulfometoxazol
Doksisiklin, Minosiklin
Klindamisin
Vankomisin IV
Linezolid IV
Follow up
Perbaikan 7 hari terapi
Kultur & Resistensi
Apabila pasien gagal diterapi dengan obat pilihan I
Sembuh
Edukasi Pasien
Terapi berdasarkan:
Hasil kultur dan resistensi
Mupirosin di sekitar nares untuk karier (untuk rekurensi tinggi)

Eritrasma



- Infeksi kulit yg disebabkan C. minutissimum
- Ax. Rasa nyeri panas seperti terkena cabe
- Px. Fisik:
  - Lipatan (intertriginous) : Patch eritem dg maserasi, kecoklatan - merah terang, dg skuamasi ringan

Px Lampu Wood : merah bata (coral red) – porfirin

Tx. Topikal : eritromisin, klindamisin, mikonazol

Sistemik : eritromisin 4x250 mg, claritromisin 1 gr

SCARLET FEVER

- Syndrome:
  1. exudative pharyngitis,
  2. fever,
  3. scarlatiniform rash (sandpaper texture).
- Cause by pyrogenic exotoxin of Group A Streptoccoccus / Staphylococcus sp.
- Kasus jarang.
- Inkubasi : 12 jam – 5 hari
- Erupsi kulit terjadi 1-2 hari setelah gx sistemik, mulai dari leher – badan – ekstremitas.
- Lesi berupa patch petekie/ purpurik (Pastias's sign), ok. Vasa darah yg fragil.
- Lesi memudar 3-4 hari kemudian, diikuti pengelupasan kulit, dan berakhir dalam 1 bulan.
- Tx. antibiotik

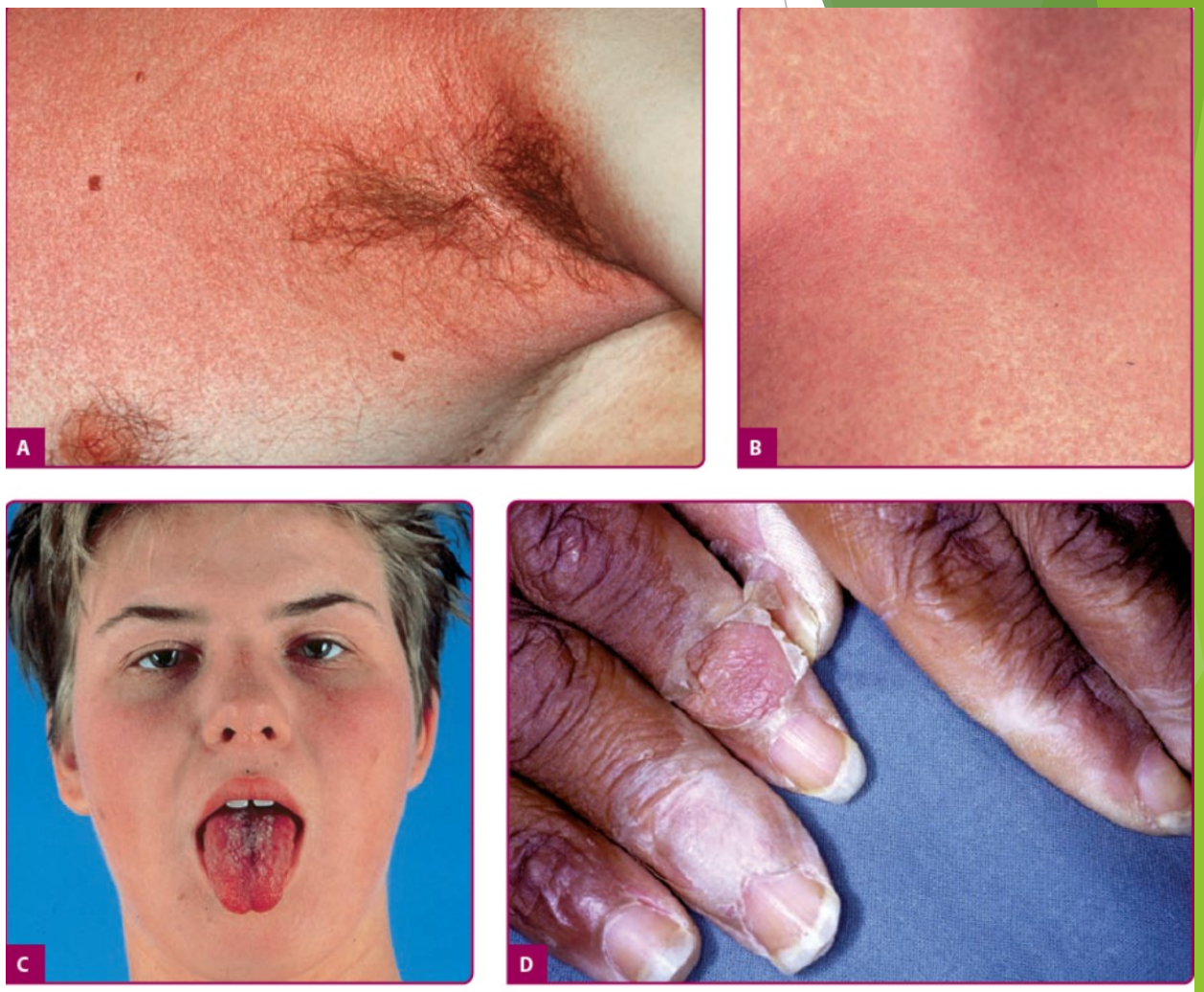


Figure 177-9 Scarlet fever. **A.** Exanthematous rash with a sandpaper texture in the axilla. **B.** Exanthematous rash with a sandpaper feel on the chest. **C.** Perioral pallor and strawberry tongue. **D.** Poststreptococcal desquamation.

Paronikia

DEFINITION

Paronychia is an inflammatory process of the nail fold (Fig. 21.3A). Acute paronychia is most often the result of bacterial infection, commonly from *Staphylococcus aureus*. Chronic paronychia is usually caused by *Candida albicans*. The predisposing factor in the production of chronic paronychia is trauma or maceration producing a break in the seal (cuticle) between the nail fold and the nail plate. This break produces a pocket that holds moisture and promotes the growth of microorganisms.

TABLE 21.1 Nail Disorders

		Physical Examination			Laboratory Test	
	Frequency (%)[a]	**Pits**	**Brown Stains**	**Differential Diagnosis**	**KOH**	**Culture**
Fungal infection	0.4	Absent	Present	Psoriasis Trauma Aging Secondary to eczema	Positive	Positive
Psoriasis	<0.1	Present	Present	Fungus Trauma Aging Secondary to eczema Alopecia areata (pits)	Negative	Negative
Paronychia	0.3	Absent	Absent	Herpes simplex	Negative Positive	Bacterial *Candida albicans*

KOH, Potassium hydroxide.
[a] Percentage of new dermatology patients with this diagnosis seen at the Hershey Medical Center Dermatology Clinic, Hershey, PA.

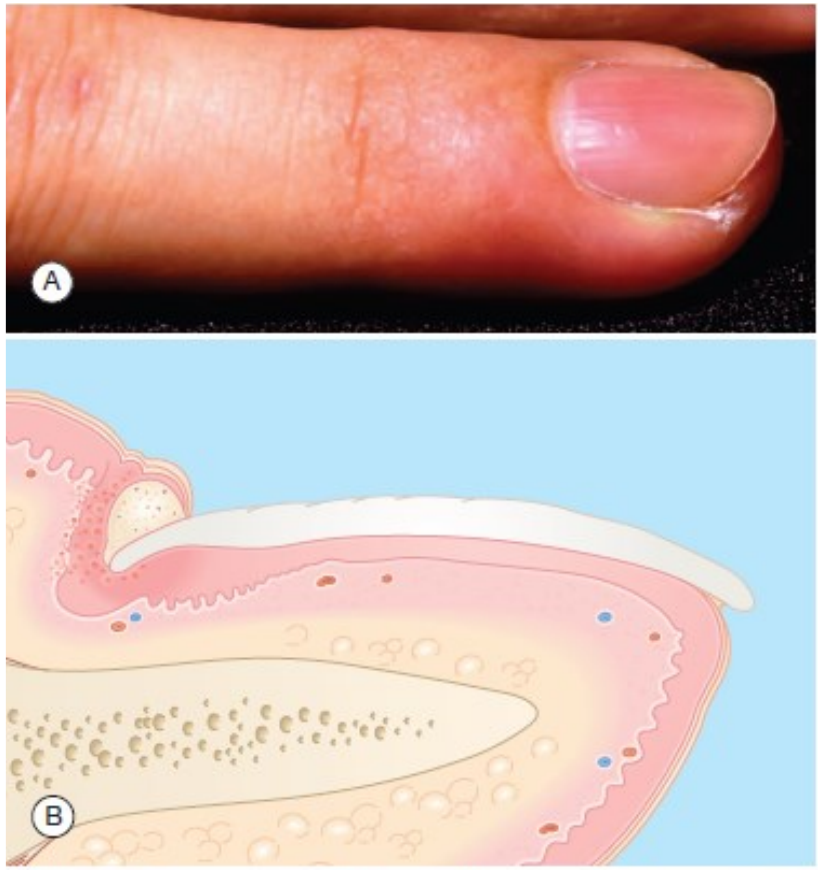


FIGURE 21.3 **A. Acute paronychia** – erythematous, swollen nail fold. **B. Chronic paronychia** – edema and inflammation, deformed cuticle with pocket with pus and candidal hyphae.

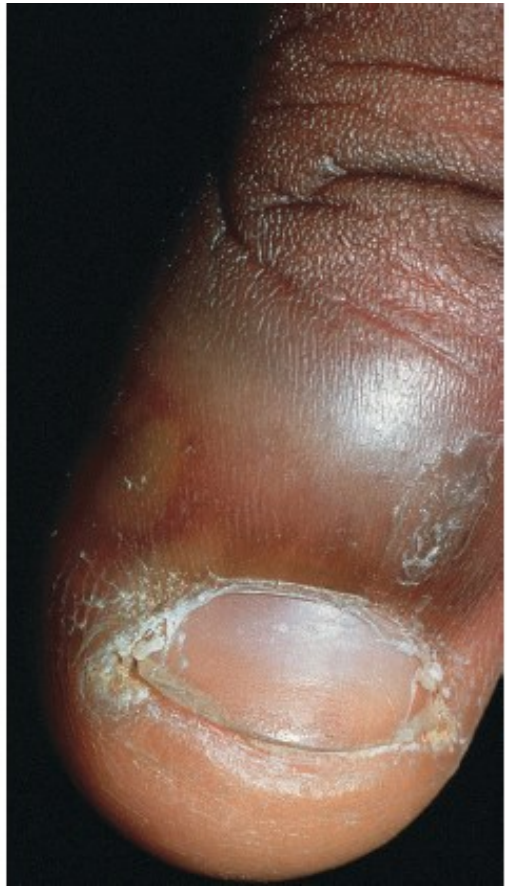


Fig. 14-6
Staphylococcal paronychia.

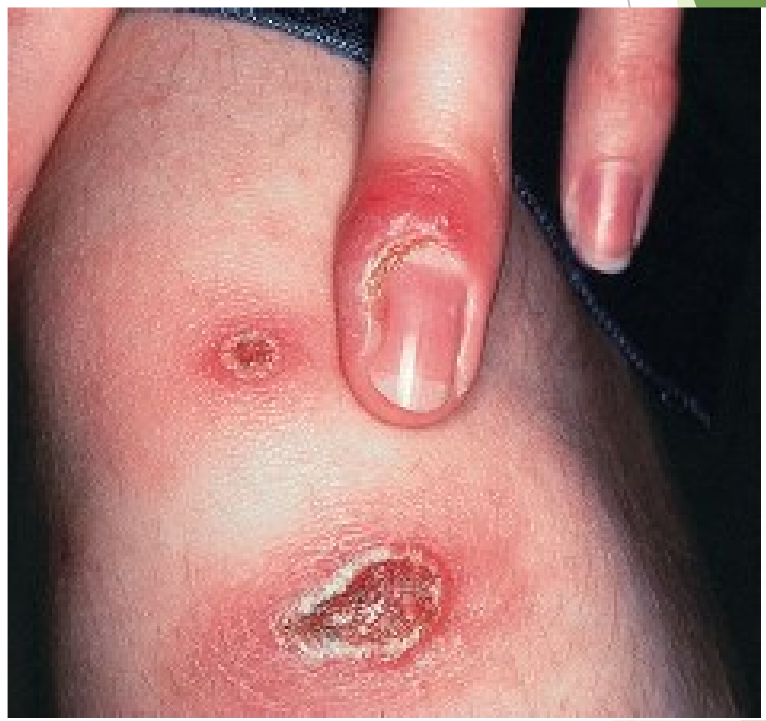


Fig. 14-7
Streptococcal paronychia and impetigo.

Therapy for Paronychia

Acute

- Cephalexin: 25–50 mg/kg daily in oral suspension, 500 mg b.i.d.
- Erythromycin: 30–50 mg/kg daily in oral suspension, 500 mg b.i.d.
- Dicloxacillin: 500 mg b.i.d.

Chronic

- Avoid trauma, water, and irritants
- Clotrimazole plus betamethasone dipropionate cream b.i.d.

Acute paronychia should be incised and drained when it is fluctuant. Appropriate antibiotic therapy for the causative agent should be instituted. In most cases, this therapy consists of cephalexin, erythromycin, or dicloxacillin.

Chronic paronychia requires the avoidance of prolonged exposure to wetness. Wearing gloves is mandatory, preferably cotton under rubber or vinyl gloves. Frequent washings and manicuring should be avoided. An anticandida and antiinflammatory topical preparation such as clotrimazole plus betamethasone dipropionate applied twice daily, is helpful.

Therapy for Paronychia

In Grown Toe Nail

DEFINITION

Ingrown toenail occurs when the lateral portion of the nail plate grows into the lateral nail fold, resulting in an inflammatory response.

INCIDENCE

Ingrown toenail is a fairly common occurrence, with the great toenails most commonly affected.

HISTORY

Pain and swelling are the symptoms that cause patients to seek medical attention. Usually, the problem has been present for weeks or months with an acute flare, which may signal a secondary infection.

PHYSICAL EXAMINATION

The lateral nail fold is red, swollen, and usually has weeping granulation tissue (Fig. 21.2). The nail plate is penetrating into the lateral nail fold.

COURSE AND COMPLICATIONS

Ingrown toenail is a chronic process, which causes pain and swelling that interfere with ambulation. Occasionally, cellulitis of the toe can be a complication.

PATHOGENESIS

The ingrown nail plate acts as a foreign body, causing an inflammatory reaction in the lateral nail fold.

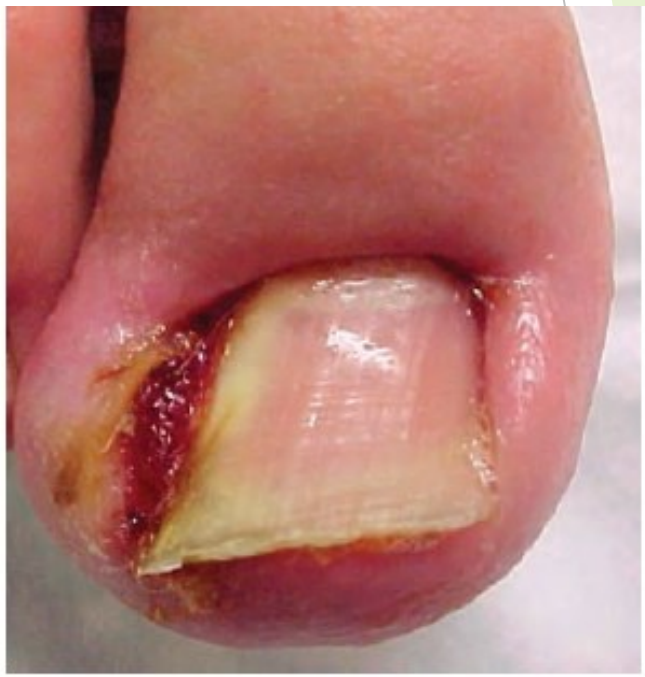


FIGURE 21.2 **Ingrown toenail** – red, swollen, lateral nail fold with granulation tissue.

Etiology

- Trimming toenails improperly: Cutting the toenail rounded, V shape or too short will cause bulging of the soft tissue and the possibility to leave a nail spur that is difficult to remove, resulting in an inflammatory reaction with pressure necrosis. The proper way to trim the toenail is to cut it straight across beyond the nail bed. [5]
- Poorly fitting shoes: The nail plate can be forced out of the nail groove by footwear that has a toe box that is too small for the forefoot. The constant pressure on the nail bed and nail groove results in breakage that starts an inflammatory process and eventually results in an ingrown nail.
- Nail plate abnormality: Increased curvature of the nail plate, as in pincer nail, may develop into an ingrown nail. [5] Deformities that result from prior trauma or underlying bone pathology may predispose to ingrown nails.
- Excessive sweating: It was noted that ingrown nails are common among teenagers and soldiers, in whom excessive sweating is present, which results in softening of the nail fold. With the participation in sports, nail spicules may develop and can easily pierce the adjacent softened nail fold.
- Obesity causing deepening of the nail groove
- Drugs (eg, antiviral therapy for HIV disease): Indinavir has been reported to have an association with an increased incidence of ingrown nails. [6] Cyclosporine, docetaxel, oral antifungals, and retinoids can cause excess nail fold granulation tissue and eventual ingrown nail development. [7, 8, 9]

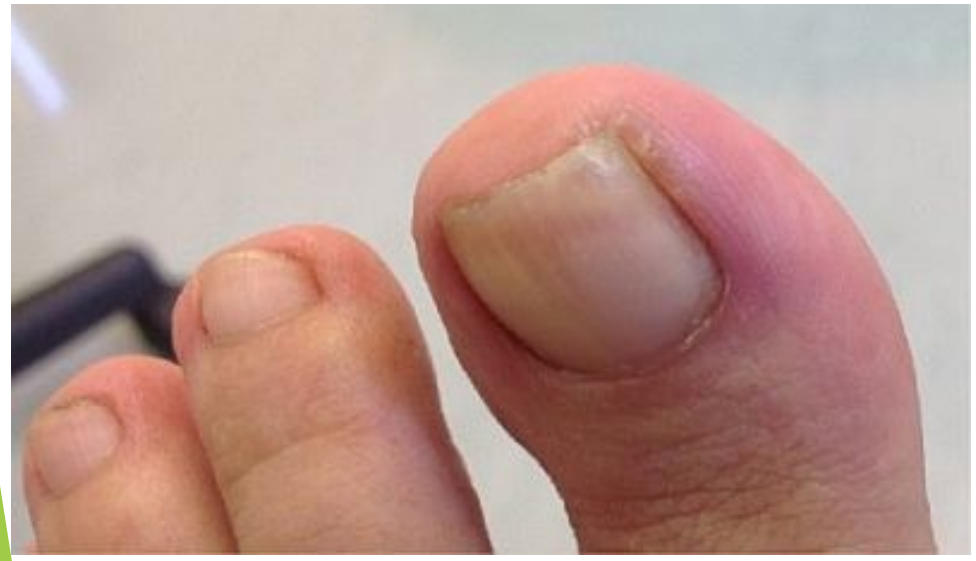
Etiology

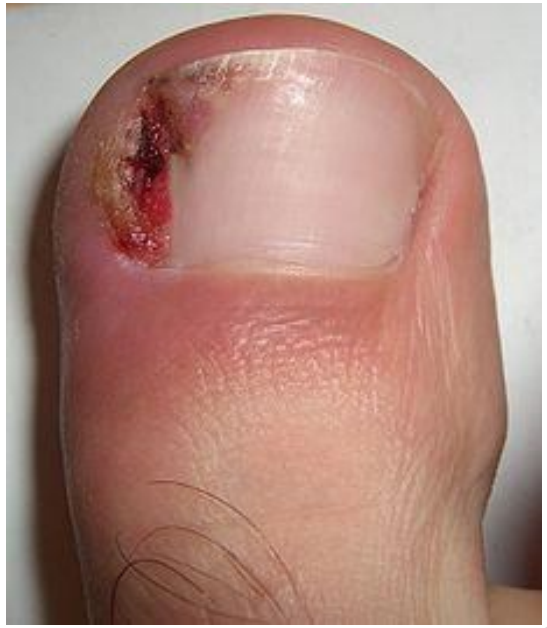
- Generalized joint hypermobility: Joint hypermobility through changes in foot biomechanics and gate affection increases medial midfoot pressure and loading during walking, and, as the first metatarsophalangeal joint bears the highest pressure, an ingrown toenail in the big toe may develop. [10]
- Onychomycosis: This infection may result in brittle nails, which may form nail spicules and pierce the adjacent nail fold.
- Heredity: Some people are genetically predisposed to inwardly curved nails, with distortion of one or both nail margins.
- Pathological hallux interphalangeal angle (≥14.5): This was correlated with the development of ingrown hallux nail and may act as a predisposing factor. [11]
- Paronychia with sporangium formation: This was reported to cause an ingrown nail. [12]
- Hematopoietic stem cell transplantation: Children with hematopoietic stem cell transplantation have a higher incidence of ingrown nails and were found to have the aggressive forms, with more than 50% of patients having nail edge and bilateral great toe involvement, as well as recurrence in 37.5%. [13]
- Nail consistency: Young male runners who have a hard nail consistency were found to have a higher incidence of ingrown nail. [14]
- Diabetes: The prevalence of ingrown nails was found to be higher in diabetic patients, suggesting the role of diabetic vasculopathy in the development and evolution of ingrown nails. [15]

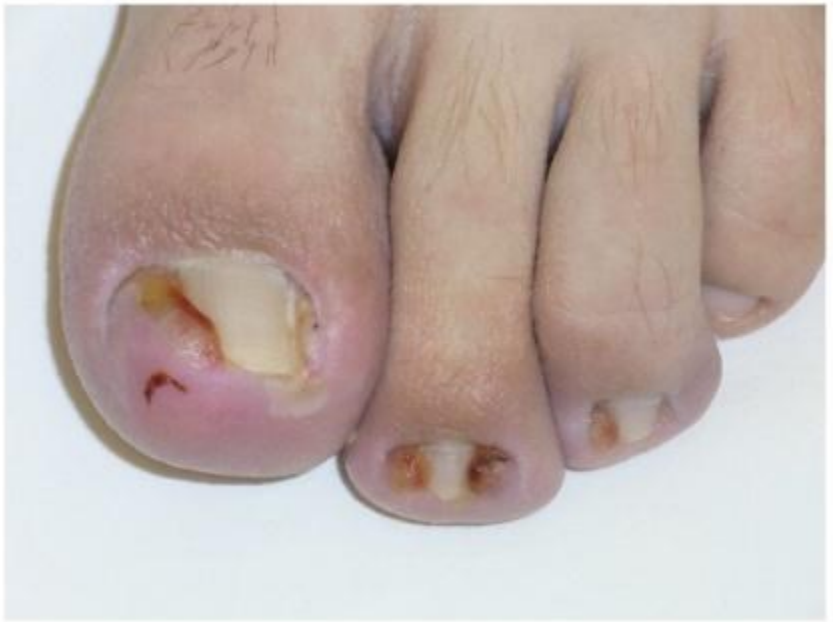
Stages of Ingrown Nail

Accordingly, ingrown nail has been divided into the following three stages [20] :

- Stage 1: Mild erythema edema and pain with pressure
- Stage 2: Significant erythema, edema, local infection, and discharge
- Stage 3: Granulation tissue formation and hypertrophy of the lateral wall besides the significant erythema, edema, and discharge (see image below)







Therapy for In Grown Nail = Onychocryptosis = Unguis incarnatus

Therapy for Ingrown Toenail

Initial

- Well-fitting shoes
- Trim nail plate straight across
- Topical antiseptics
- Cotton pledget insertion

Alternative

- Nail avulsion with matrix destruction

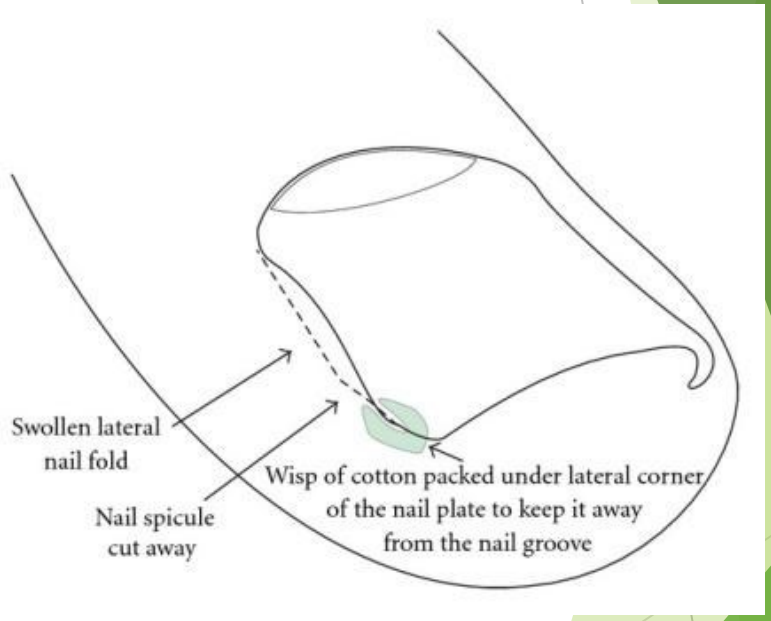
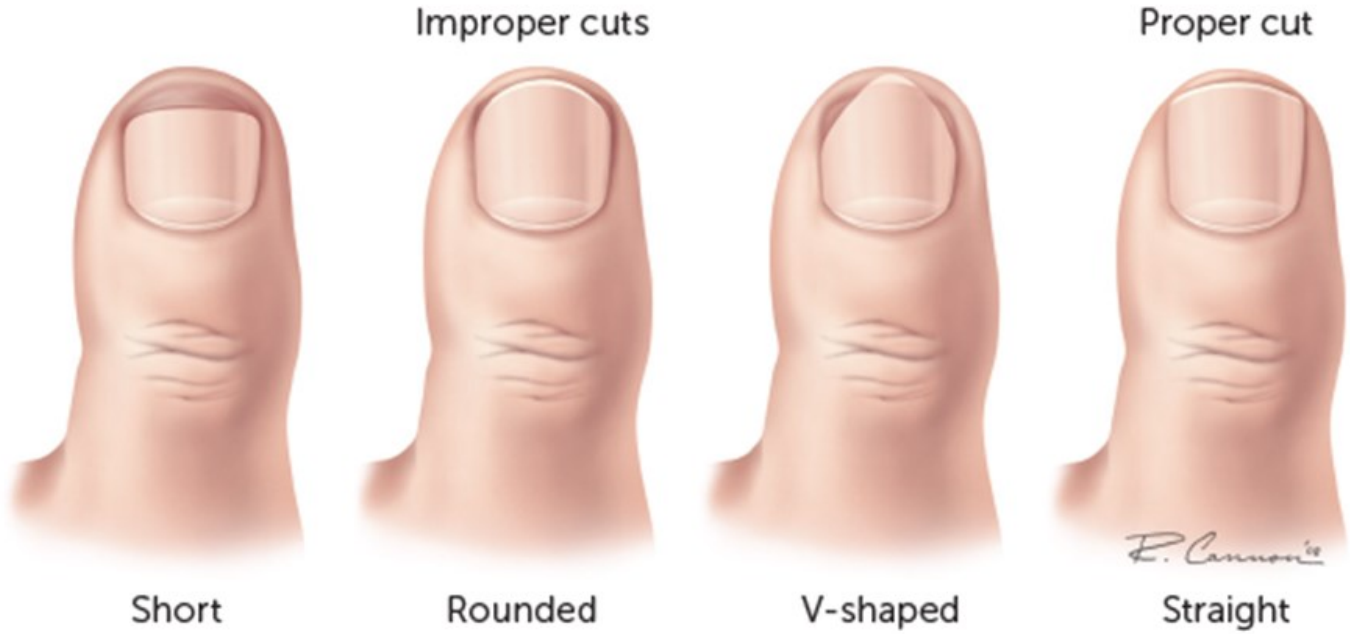


FIGURE 1



Examples of improper and proper toenail trimming. Toenails should be cut straight across, and the corners should not be rounded off.

Reprinted with permission from Heidelbaugh JJ, Lee H. Management of the ingrown toenail. Am Fam Physician. *2009;79(4):303.*

SIFILIS

Definisi

Penyakit infeksi menular seksual yang disebabkan oleh *Treponema pallidum*, bersifat kronis, sejak awal merupakan infeksi sistemik, dalam perjalanan penyakitnya dapat mengenai hampir seluruh struktur tubuh, dengan manifestasi klinis yang jelas namun terdapat masa laten yang sepenuhnya asimtomatik, mampu menyerupai berbagai macam penyakit, dapat ditularkan kepada janin dalam kandungan, dan dapat disembuhkan.

Sifilis dapat diklasifikasikan menjadi sifilis didapat dan sifilis kongenital. Sifilis didapat terdiri atas stadium primer, sekunder, dan tersier, serta periode laten di antara stadium sekunder dan tersier.

T. Pallidum masuk ke tubuh melalui mukosa/kulit yg tidak intak (hubungan seksual), inkubasi 10-90 hari, terjadi luka asimtomatik, sembuh sendiri, memasuki masa laten, perluasan infeksi secara hematogen sampai ke kulit dan organ tubuh lain sehingga terjadi hepatitis, tulang, persendian, ginjal (nefritis), SNC (Sifilis Sekunder).

Jika tidak diobati, Lesi berkembang/meluas ke sistem kardiovaskular dan SNC dalam bberapa tahun, sehingga terjadi komplikasi (sifilis tersier)

Kriteria Diagnostik

Klinis

Stadium I (Sifilis primer) – ICD10: A51

- Ulkus tunggal, tepi teratur, dasar bersih, terdapat indurasi, tidak nyeri; terdapat pembesaran kelenjar getah bening regional.
- Lokasi: di tempat kontak dengan lesi infeksius pasangan seksual. Pada laki-laki sering didapatkan di penis (terutama di glans penis atau sekitar sulkus koronarius) dan skrotum; pada perempuan didapatkan di vulva, serviks, *fourchette*, atau perineum. Namun dapat pula ulkus tidak tampak dan tidak disadari oleh pasien.

Stadium II (Sifilis sekunder) – ICD10: A51.3

- Terdapat lesi kulit yang polimorfik, tidak gatal dan lesi di mukosa, sering disertai pembesaran kelenjar getah bening generalisata yang tidak nyeri (limfadenopati).

Stadium laten – ICD10: A53.0

- Tidak ditemukan gejala klinis pada pasien, namun tes serologi sifilis (TSS) reaktif, baik serologi treponema maupun nontreponema.

Stadium III (Sifilis tersier) – ICD10: A52

- Didapatkan gumma, yaitu infiltrat sirkumskrip kronis yang cenderung mengalami perlunakan dan bersifat destruktif. Dapat mengenai kulit, mukosa dan tulang.

Diagnosis Banding

1. Sifilis primer: herpes simpleks, ulkus piogenik, skabies, balanitis, LGV, karsinoma sel skuamosa, penyakit Behcet, ulkus mole
2. Sifilis sekunder: erupsi obat alergik, morbili, pitiriasis rosea, psoriasis, dermatitis seboroik, kondilomata akuminata, alopesia areata
3. Sifilis tersier: sporotrikosis, aktinomikosis, tuberkulosis kutis gumosa, keganasan

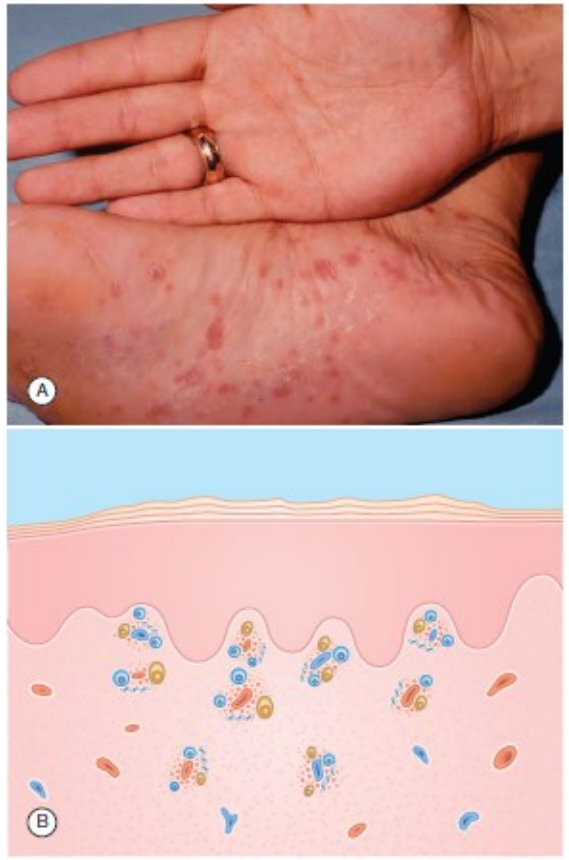


FIGURE 9.19 **Secondary syphilis. A.** Characteristic plantar and palmar, "ham"-colored, slight scaling patches and macules. **B.** Epidermis – slight hyperkeratosis. Dermis – perivascular infiltrate with lymphocytes, plasma cells, and spirochetes (with silver stain).

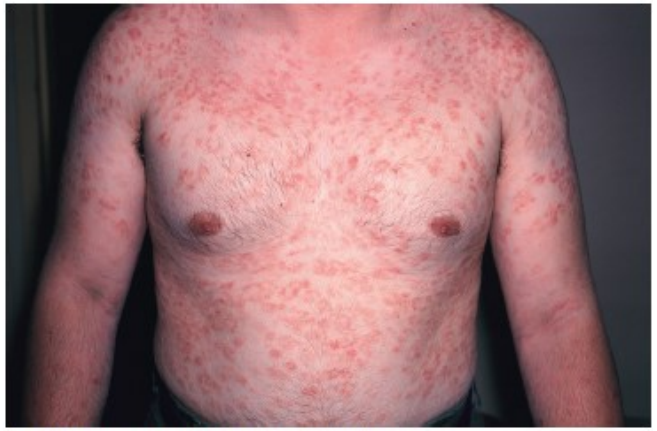


Fig. 18-4 Secondary syphilis, lichenoid lesions.

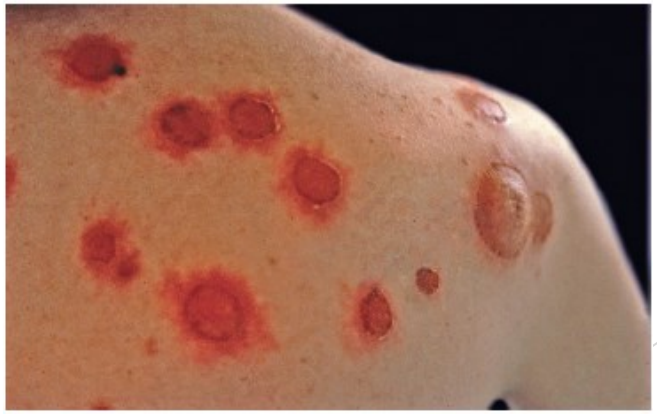


Fig. 18-5 Secondary syphilis.

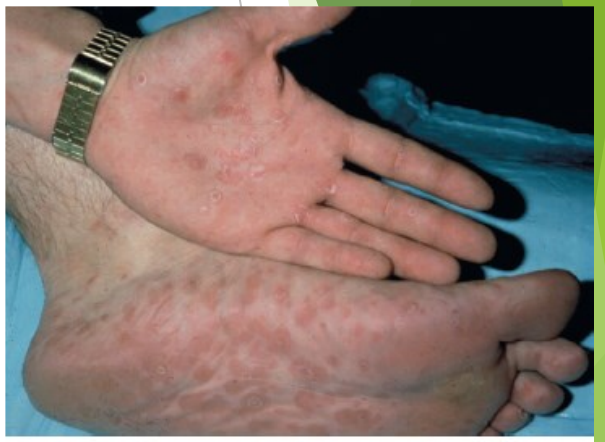


Fig. 18-6 Secondary syphilis; red, flat-topped papules of the palms and soles.

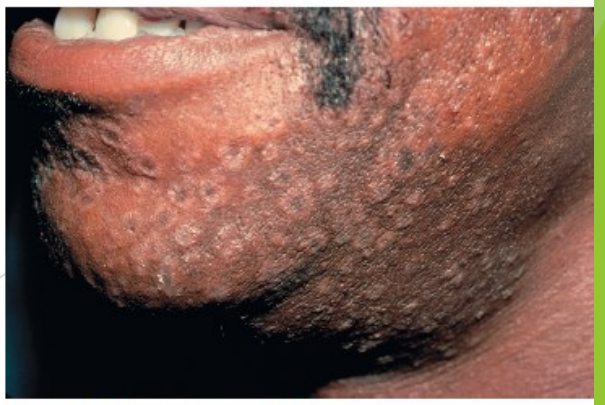


Fig. 18-7 Annular secondary syphilis.

TABLE 3.4 Regional Diagnoses

Growths	Rashes
Scalp	
Nevus	Seborrheic dermatitis (dandruff)
Seborrheic keratosis	Psoriasis
Pilar cyst	Tinea capitis Folliculitis
Face	
Nevus	Acne
Lentigo	Acne rosacea
Actinic keratosis	Seborrheic dermatitis
Seborrheic keratosis	Contact dermatitis (cosmetics)
Sebaceous hyperplasia	Herpes simplex
Basal cell carcinoma	Impetigo
Squamous cell carcinoma	Pityriasis alba
Flat wart	Atopic dermatitis
Nevus flammeus	Lupus erythematosus
Trunk	
Nevus	Acne
Skin tag	Tinea versicolor
Cherry angioma	Psoriasis
Seborrheic keratosis	Pityriasis rosea
Epidermal inclusion cyst	Scabies
Lipoma	Drug eruption
Basal cell carcinoma	Varicella
Keloid	Mycosis fungoides
Neurofibroma	Secondary syphilis
Genitalia	
Wart (condyloma acuminatum)	Herpes simplex
	Scabies
Molluscum contagiosum	Psoriasis
Seborrheic keratosis	Lichen planus Syphilis (chancre) Contact dermatitis

Growths	Rashes
Groin (inguinal)	
Skin tag	Intertrigo
Wart	Tinea cruris
Molluscum contagiosum	Candidiasis Pediculosis pubis Hidradenitis suppurativa Psoriasis Seborrheic dermatitis
Extremities	
Nevus	Atopic dermatitis
Dermatofibroma	Contact dermatitis
Wart	Psoriasis
Seborrheic keratosis	Insect bites
Actinic keratosis	Erythema multiforme
Xanthoma	Lichen planus (wrists and ankles) Actinic purpura (arms) Stasis dermatitis (legs) Vasculitis (legs) Erythema nodosum (legs)
Hands (palmar)	
Wart	Essential dermatitis Atopic dermatitis Psoriasis Tinea manuum Erythema multiforme Secondary syphilis
Feet (dorsal)	
Wart	Contact dermatitis (shoe)
Feet (plantar)	
Wart (plantar)	Contact dermatitis (shoe)
Corn	Tinea pedis
Nevus	Essential dermatitis
	Psoriasis
	Atopic dermatitis

TABLE 9.1 Scaling Papules, Plaques, and Patches[a]

	Frequency (%)[b]	Etiology	Physical Examination: Appearance of Lesions	Physical Examination: Characteristic Distribution	Differential Diagnosis	Laboratory Tests
Lupus, discoid	0.2	"Autoimmune"	Red to *purplish* papules and plaques with adherent scale and *follicular plugging*; older lesions atrophic	Sun-exposed areas favored	Psoriasis Lichen planus Subacute cutaneous lupus erythematosus	Biopsy with immunofluorescence; antinuclear antibodies
Fungus	2.5	Infection (dermatophyte)	*Annular* patches with elevated borders surmounted by scale	Anywhere	(See Table 9.2)	Potassium hydroxide preparation; fungal culture
Mycosis fungoides	0.2	Neoplastic (lymphoma)	*Yellowish-red* or *violaceous*, irregularly shaped patches and plaques with only slight scale	*Asymmetric*; girdle area is often the first area involved	Psoriasis Parapsoriasis Eczema Erythroderma	Biopsy
Pityriasis rosea	1.1	Human herpesvirus 6 and 7	Tannish-pink *oval* papules and patches with delicate *collarette of scale*; rash preceded by *herald patch*	"Christmas tree" pattern on trunk; spares face and distal extremities	Secondary syphilis Tinea corporis Lichen planus Pityriasis lichenoides chronica Guttate psoriasis	
Psoriasis	5.2	Unknown	Erythematous plaques with *silvery scales*	Anywhere; scalp, elbows, knees, and *intergluteal cleft* are favored locations; nails often involved	Seborrheic dermatitis Tinea cruris Candidiasis Intertrigo Pityriasis rosea Tinea corporis Dermatitis T-cell cutaneous lymphoma Onychomycosis	
Secondary syphilis	<0.1	Infection (spirochete)	*Red–brown* or *copper-colored* scaling papules and plaques, sometimes annular in shape	Generalized; *palms* and *soles* often included; mucous membranes sometimes involved	Pityriasis rosea Viral exanthem Drug eruption Sarcoidosis	Serologic test for syphilis

[a] See also discussions of seborrheic dermatitis (Chapter 8), lichen planus (Chapter 11), and tinea versicolor (Chapter 13).
[b] Percentage of new dermatology patients with this diagnosis seen in the Hershey Medical Center Dermatology Clinic, Hershey, PA.

Pemeriksaan Penunjang

Tabel 1. Pemeriksaan penunjang sifilis

	Sifilis primer	Sifilis sekunder	Sifilis laten
RPR atau VDRL	Dapat reaktif atau non reaktif	Reaktif, titer tinggi	Reaktif
TPHA	Reaktif	Reaktif	Reaktif

Penatalaksanaan

Obat pilihan:

Benzil benzatin penisilin G (BBPG), dengan dosis:

1. Stadium primer dan sekunder: 2,4 juta Unit, injeksi intramuskular, dosis tunggal (A,1)[2,3-6]
   Cara: satu injeksi 2,4 juta Unit IM pada 1 bokong, atau 1,2 juta Unit pada setiap bokong.
2. Stadium laten: 2,4 juta Unit injeksi intramuskular, setiap minggu, pada hari ke-1, 8 dan 15 (B,2)[2,3]

Sesudah diinjeksi, pasien diminta menunggu selama 30 menit.

Obat alternatif: bila alergi terhadap penisilin atau pasien menolak injeksi atau tidak tersedia BBPG:

1. Doksisiklin 2x100 mg oral selama 14 hari untuk stadium primer dan sekunder (B,3) atau selama 28 hari untuk sifilis laten.[2,3] (B,3)
   Doksisiklin 2x100 mg oral selama 30 hari untuk stadium primer dan sekunder atau lebih dari 30 hari untuk sifilis laten.[4] (D,5)
2. Eritromisin 4x500 mg oral selama 14 hari untuk ibu hamil dengan sifilis stadium primer dan sekunder, atau 30 hari untuk sifilis laten (*very low quality evidence, conditional recommendation*)[3]
   Eritromisin 4x500 mg oral selama 30 hari untuk ibu hamil dengan sifilis stadium primer dan sekunder, atau lebih dari 30 hari untuk sifilis laten.[4] (D,5)

Evaluasi terapi: evaluasi secara klinis dan serologi dilakukan pada bulan ke-1, 3, 6, dan 12.

Kriteria sembuh: titer VDRL atau RPR menurun 4 kali lipat dalam 6 bulan setelah pengobatan.

Edukasi

1. Sedapat mungkin pasangan seksual ikut diobati
2. Konseling/edukasi:
   - Mengenai penyakit sifilis, cara penularan, pencegahan, dan pengobatan
   - Risiko mudah tertular HIV perlu dilakukan KTIP (Konseling dan tes HIV atas inisiatif petugas kesehatan)
   - Konseling umum: lihat halaman 378

Konseling Umum Bagi Pasien Infeksi Menular Seksual (IMS)

Konseling bagi pasien IMS merupakan peluang penting untuk dapat sekaligus memberikan edukasi tentang pencegahan infeksi HIV pada seseorang yang berisiko terhadap penyakit tersebut.[1]

Beberapa pesan tentang IMS yang perlu disampaikan:[1]

1. Mengobati sendiri cukup berbahaya
2. IMS umumnya ditularkan melalui hubungan seksual
3. IMS adalah ko-faktor atau faktor risiko dalam penularan HIV
4. IMS harus diobati secara paripurna dan tuntas
5. Pasangan seksual perlu diperiksa dan diobati
6. Kondom dapat melindungi diri dari infeksi IMS dan HIV
7. Tidak dikenal adanya pencegahan primer terhadap IMS dengan obat
8. Komplikasi IMS dapat membahayakan pasien

Rincian penjelasan kepada pasien IMS:

1. Kemungkinan risiko tertular hepatitis B, hepatitis C, sifilis, dan IMS lainnya[1]
2. Tawarkan pemeriksaan serologis sifilis[1]
3. Konseling dan tes HIV (KT HIV) dengan prinsip dasasr 5 C (*informed consent, confidentiality, counseling, correct test results, connections to care treatment and prevention services*):
   - Menggunakan alur layanan:
     - Konseling dan tes secara sukarela (KTS): konseling pretes-tes darah/*rapid diagnostic test*-konseling pasca tes) atau
     - Konseling dan tes atas inisitatif petugas kesehatan (KTIP): pemberian informasi tentang HIV/AIDS-tes darah
   - Hasil tes darah HIV non-reaktif diberikan informasi tentang: masa jendela, pencegahan penularan, risiko penularan HIV dari ibu ke anak, perencanaan kehamilan/keluarga berencana (KB) dan anjuran konseling/edukasi/tes darah pada pasangan
   - Semua hasil tes darah reaktif dirujuk ke tim perawatan, dukungan dan pengobatan (PDP) di fasilitas layanan kesehatan terdekat
   - Semua kasus IMS, hepatitis B, hepatitis C, ibu hamil, pecandu napza/IDU, wanita penjaja seks (WPS), *lesbian gay bisexual transgender* (LGBT) dan pasangannya wajib dilakukan KT HIV.[2]

Terimakasih
Selamat Belajar

Skin microbiome map