

Infection

Cutaneous infections: mechanisms, morphology and clinicopathologic recognition

Viral • bacterial • superficial fungal infections

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Learning objectives

By the end of this lecture, students should be able to:

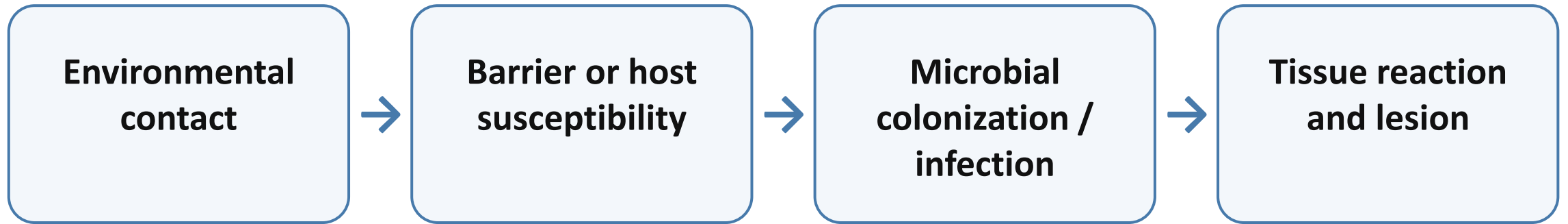
- Classify selected skin infections by pathogen group and epidermal compartment.
- Link pathogen-specific mechanisms to characteristic visible and microscopic lesions.
- Distinguish warts, molluscum, impetigo and superficial mycoses using diagnostic clues.
- Select an appropriate microscopic or microbiologic approach when required.

Why does the skin remain vulnerable?

A formidable barrier is continuously exposed to the environment.

- Frequent trauma provides opportunities for microbial access to susceptible tissue.
- The epidermis is in continuous contact with microorganisms, parasites and insects.
- Some infections remain local; Host immune competence helps determine their impact.
- The disease pattern depends on the infectious agent and the tissue response.

From exposure to cutaneous disease



The same exposure may have very different consequences depending on the host and the depth and type of tissue involved.

Immunocompromise changes the risk

A seemingly trivial skin infection can become dangerous.

- That ordinarily limited cutaneous infections can become life-threatening in immunocompromised hosts.
- Assessment therefore includes immune status, lesion burden and evidence of extension.
- The severity of illness is not determined by lesion size alone.

Anatomic location helps organize infections

Stratum corneum

Living epidermis

Dermis

Subcutis

Dermatophytes

Predominantly cornified surface

HPV / molluscum

Epidermal epithelial lesions

Impetigo

Superficial intraepidermal pustules

A practical overview of pathogen groups

Group	Representative lesion	Mechanistic emphasis
Papillomavirus	Verrucae (warts)	Viral-driven epidermal proliferation
Poxvirus	Molluscum contagiosum	Cytoplasmic inclusions within epidermal cells
Bacteria	Impetigo	Neutrophilic pustules and surface crust
Dermatophytes / yeast	Tinea syndromes / versicolor	Involvement of cornified tissue or surface

These are representative skin-predominant infections, not an exhaustive infectious-disease taxonomy.

Clinical morphology is a diagnostic language

A descriptive lesion pattern can narrow the differential.

- Rough papules suggest a proliferative epidermal process.
- Umbilicated papules raise suspicion for molluscum contagiosum.
- Pustules that rupture into honey-colored crust point toward impetigo.
- An expanding scaly border suggests a superficial dermatophyte infection.

Four patterns: One compact differential

Observed pattern	Priority exemplar	Distinctive tissue clue
Rough, keratotic papule	Verruca vulgaris	Papillomatosis + koilocytosis
Smooth, centrally umbilicated papule	Molluscum	Cytoplasmic molluscum bodies
Crusted erosion or pustule	Impetigo	Subcorneal neutrophilic pustule
Scaly, often annular plaque	Tinea corporis	Hyphae in cornified keratin

Direct infection versus reactive skin findings

A cutaneous finding does not always mean local microbial proliferation.

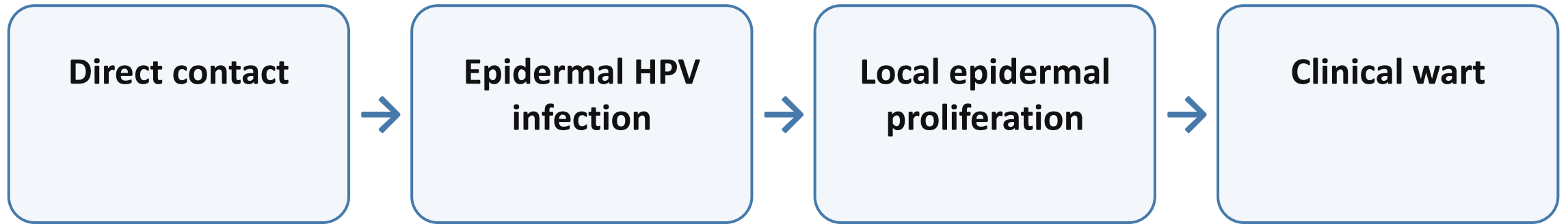
- Warts, molluscum, impetigo and superficial mycoses represent processes with primary cutaneous manifestations.
- An infection elsewhere may provoke a cutaneous immune reaction; The skin finding is then not simply a local focus of microbial growth.
- Herpes simplex, herpes zoster, viral exanthems and deep fungal diseases are additional infection-related patterns addressed in other Robbins sections.

Verrucae are HPV-induced proliferations

The primary lesion is a virally driven epidermal growth.

- Human papillomaviruses produce benign-appearing warts at characteristic anatomic sites.
- Verrucae are particularly common in children and adolescents, but occur at any age.
- The clinical subtypes differ by surface morphology and location.
- Histopathology reflects keratinocyte proliferation and viral cytopathic change.

Transmission of cutaneous warts



Autoinoculation can move infection from one cutaneous site to another; Direct person-to-person contact is also described.

Warts often regress without intervention

Natural history is usually self-limited.

- That many verrucae regress spontaneously over approximately 6 months to 2 years.
- A persisting papule therefore need not indicate invasive neoplasia.
- Clinical context and histology are important for lesions with atypical morphology.

HPV types are biologically heterogeneous

Not all HPV genotypes have equivalent tissue or oncogenic effects.

- More than 150 papillomavirus types are discussed.
- Different types are associated with different forms of cutaneous and anogenital warts.
- Site, viral genotype and host background jointly affect clinical interpretation.

Selected HPV genotype associations

HPV types	Association in Robbins	Interpretive point
6, 11	Predominant types in anogenital warts	Typically low-risk associations
16	Genital carcinoma in situ and bowenoid papulosis	High-risk HPV association
5, 8	Epidermodysplasia verruciformis-associated lesions	Unusual host and lesion context

HPV 6 and 11: Anogenital warts

Anogenital site and verrucous architecture are linked.

- Anogenital warts are predominantly associated with HPV types 6 and 11.
- Condyloma acuminatum appears as a soft, tan, cauliflower-like growth.
- Typical anogenital distribution includes genital and perianal mucocutaneous sites.

HPV 16 and high-risk intraepithelial lesions

A subset of HPV infections is linked to neoplastic change.

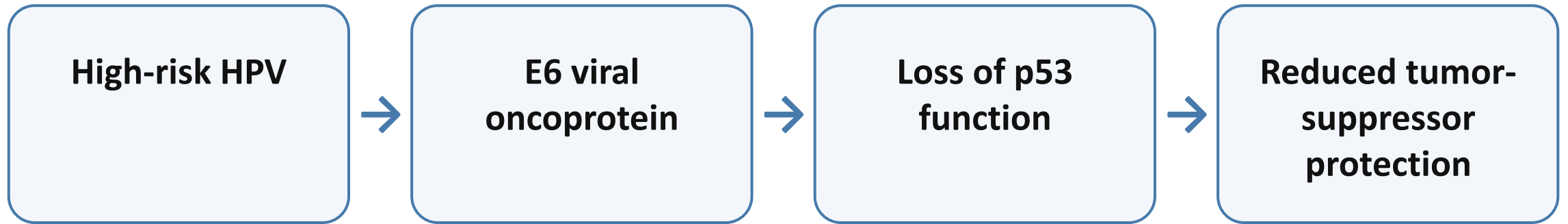
- HPV 16 with genital squamous carcinoma in situ and bowenoid papulosis.
- Bowenoid papulosis may show carcinoma-in-situ histology despite an often regressive clinical course.
- Do not equate ordinary verruca vulgaris with an HPV-associated neoplastic lesion.

HPV 5 and 8: Unusual host association

Epidermodysplasia verruciformis provides a distinct context.

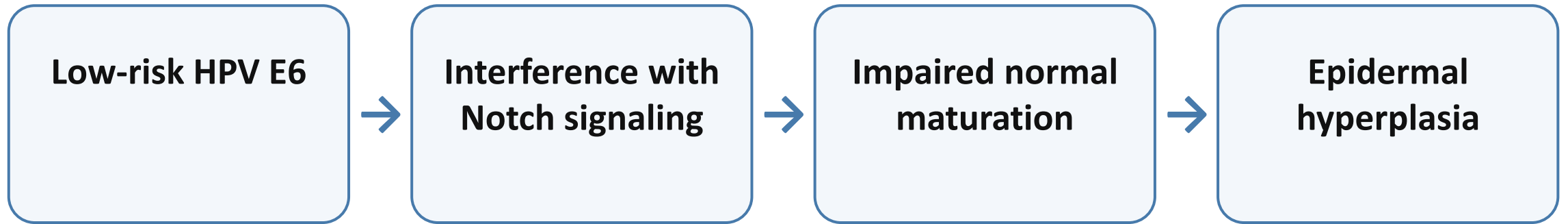
- Patients may develop numerous flat wart-like lesions containing HPV genomes.
- Some lesions can progress to squamous cell carcinoma.
- This uncommon setting should not be generalized to ordinary self-limited warts.

Oncogenic HPV mechanism emphasized by Robbins



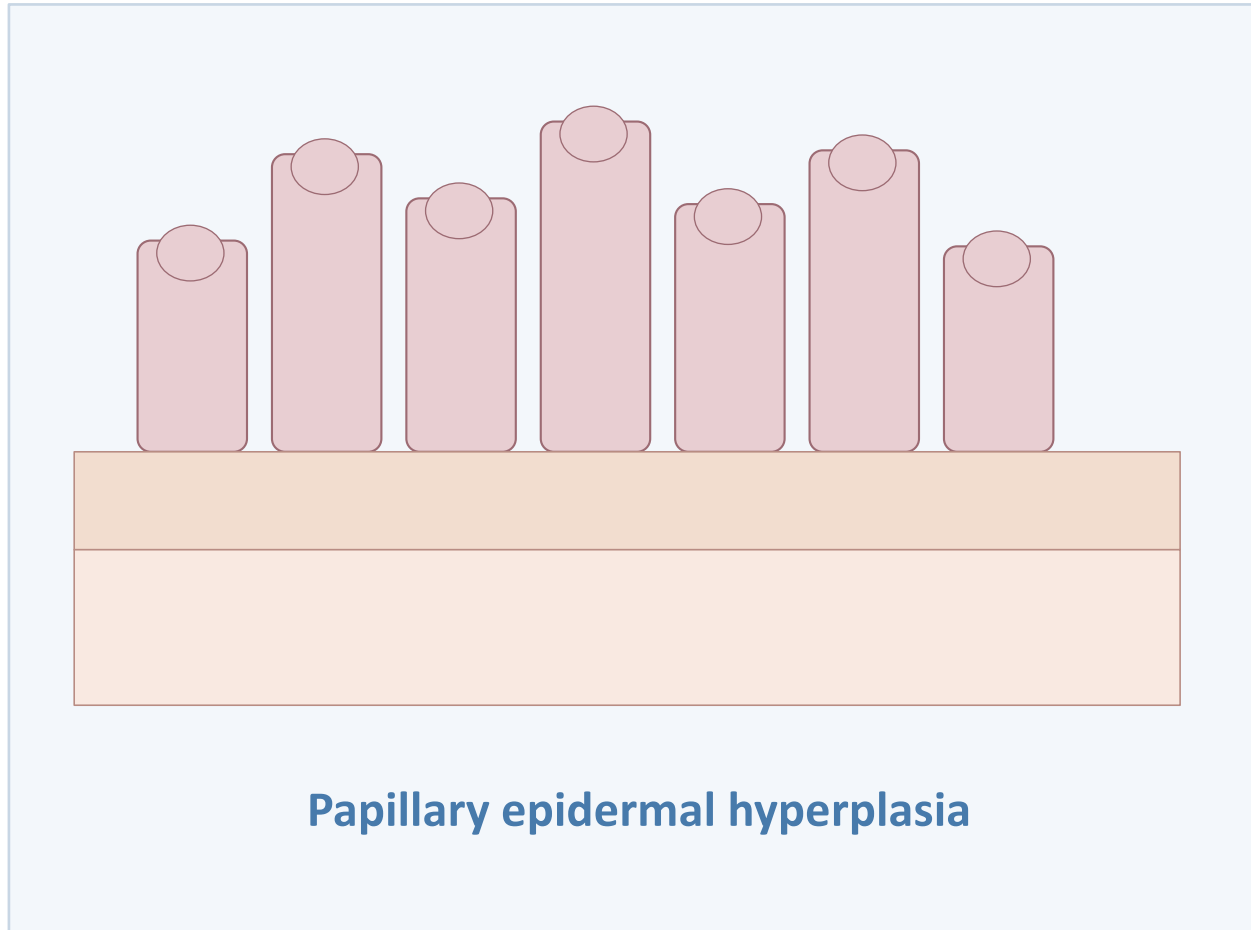
This selected mechanism explains a cancer association in high-risk HPV infection; It does not define the behavior of every cutaneous HPV subtype.

Low-risk HPV: Altered keratinocyte differentiation



Interference with Notch-dependent differentiation as a likely contributor to wart hyperplasia.

Verruca vulgaris: Prototypical surface pattern



- Often a rough, pebble-like papule.
- Common on the hands, including dorsal and periungual areas.
- Color varies with background skin pigmentation.

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Flat warts differ from common warts

Clinical designation depends on contour and distribution.

- Verruca plana tends to form small, slightly elevated, smooth, flat papules.
- Typical locations include the face and dorsal surfaces of the hands.
- The lack of an obvious rough surface does not exclude HPV infection.

Palmar / plantar warts versus common warts

Palmar or plantar verrucae

- Arise on palms or soles.
- Rough, scaly lesions; May resemble a callus.
- Can reach approximately 1–2 cm in the source description.

Verruca vulgaris

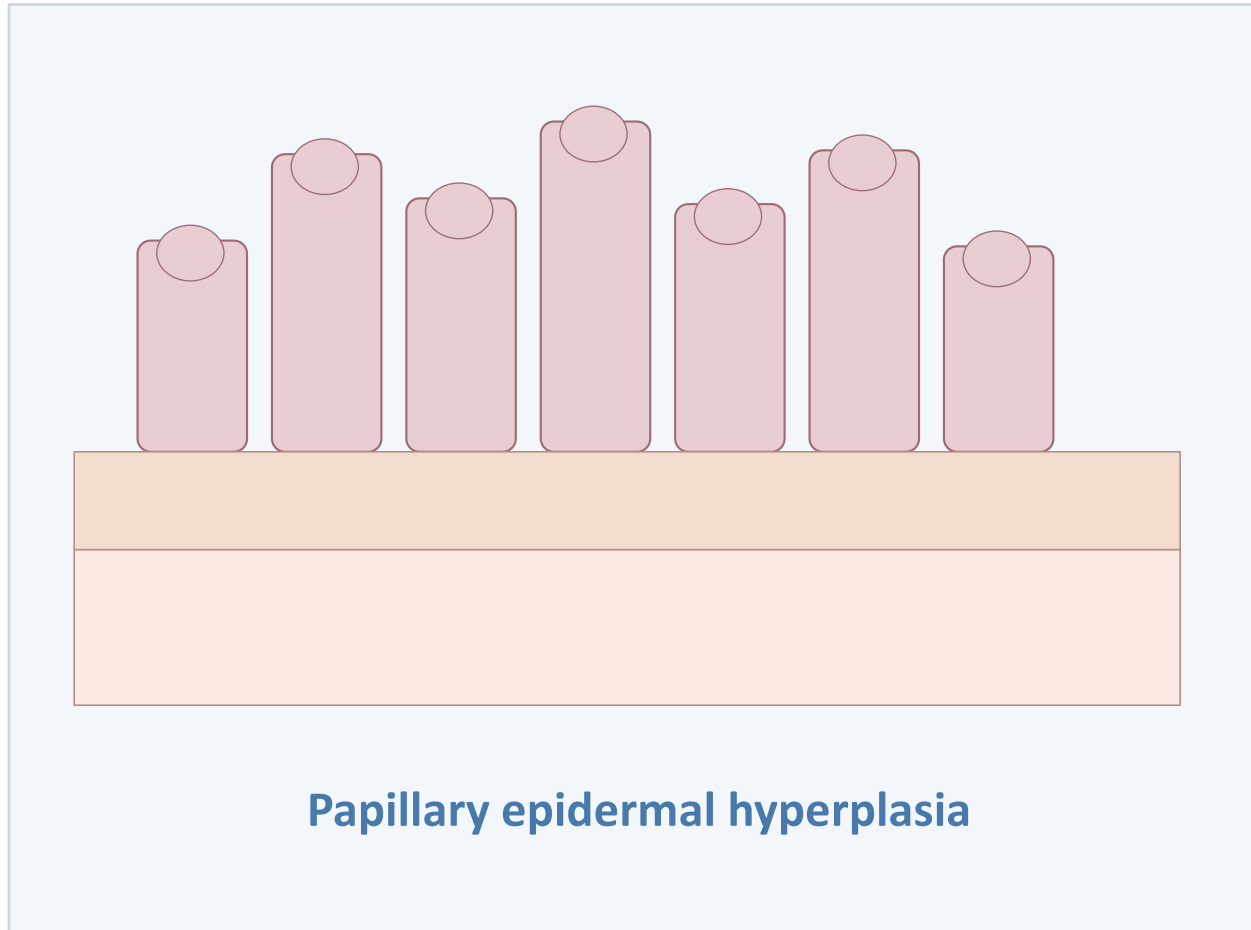
- Commonly occurs on dorsal hands and near nails.
- Rough, pebble-like surface.
- Frequently small papules measuring about 0.1–1 cm.

Condyloma acuminatum: Clinical architecture

Exophytic anogenital warts can become sizable.

- Lesions are soft, tan, papillary or cauliflower-like masses.
- Involvement may include genital, perianal, urethral and rectal locations.
- Microscopic changes may be subtler than in common cutaneous verrucae.

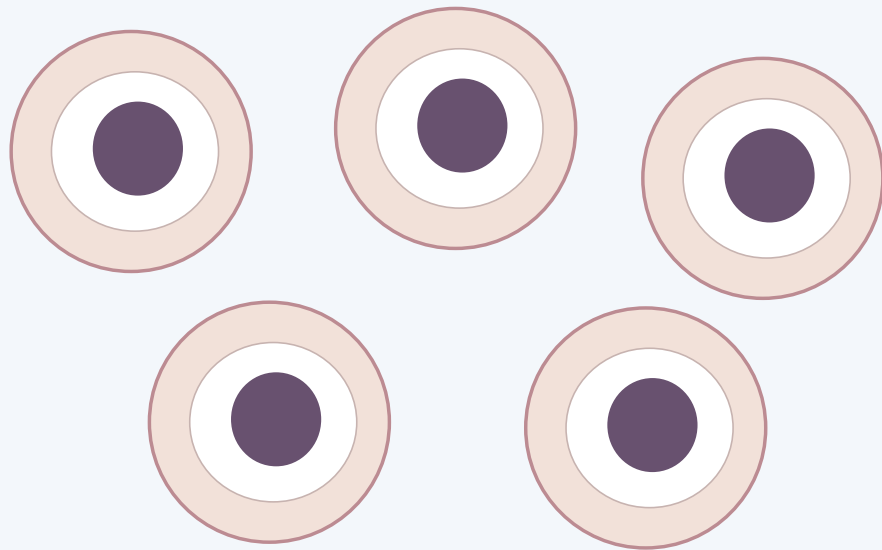
Verrucae: Papillomatous epidermal architecture



- Epidermal hyperplasia can be undulant, papillomatous or spire-like.
- The proliferating squamous epithelium creates the rough surface.
- Architectural changes must be integrated with cellular viral effects.

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Koilocytosis: Cytopathic evidence



Pale perinuclear halo + altered nucleus

- Superficial epidermal cells show conspicuous perinuclear pallor.
- The halo surrounds an altered infected nucleus.
- Koilocytosis supports HPV-associated epithelial change in context.

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Other HPV cytopathic features

The diagnosis is more than a single perinuclear halo.

- Prominent condensed keratohyalin granules.
- Jagged eosinophilic intracytoplasmic keratin aggregates can occur.
- Infected nuclei contain virions demonstrable ultrastructurally.
- Condylomata may have less obvious cytopathic alteration than common warts.

HPV typing is not synonymous with morphology

Molecular detection answers a different question.

- In situ hybridization or polymerase chain reaction can determine HPV type.
- The routine wart pattern is initially a clinicopathologic diagnosis.
- Molecular genotype becomes pertinent when the clinical question concerns viral association or risk context.

Molluscum: A poxvirus infection of epidermis

The lesion is typically benign and self-limited.

- Molluscum contagiosum is caused by a poxvirus, not a papillomavirus.
- Person-to-person direct contact is a usual route of spread.
- Children and young adults are common affected groups.
- The lesion exhibits highly characteristic intracytoplasmic inclusions.

Poxvirus architecture distinguishes the pathogen

The infectious agent is unusually large for a virus.

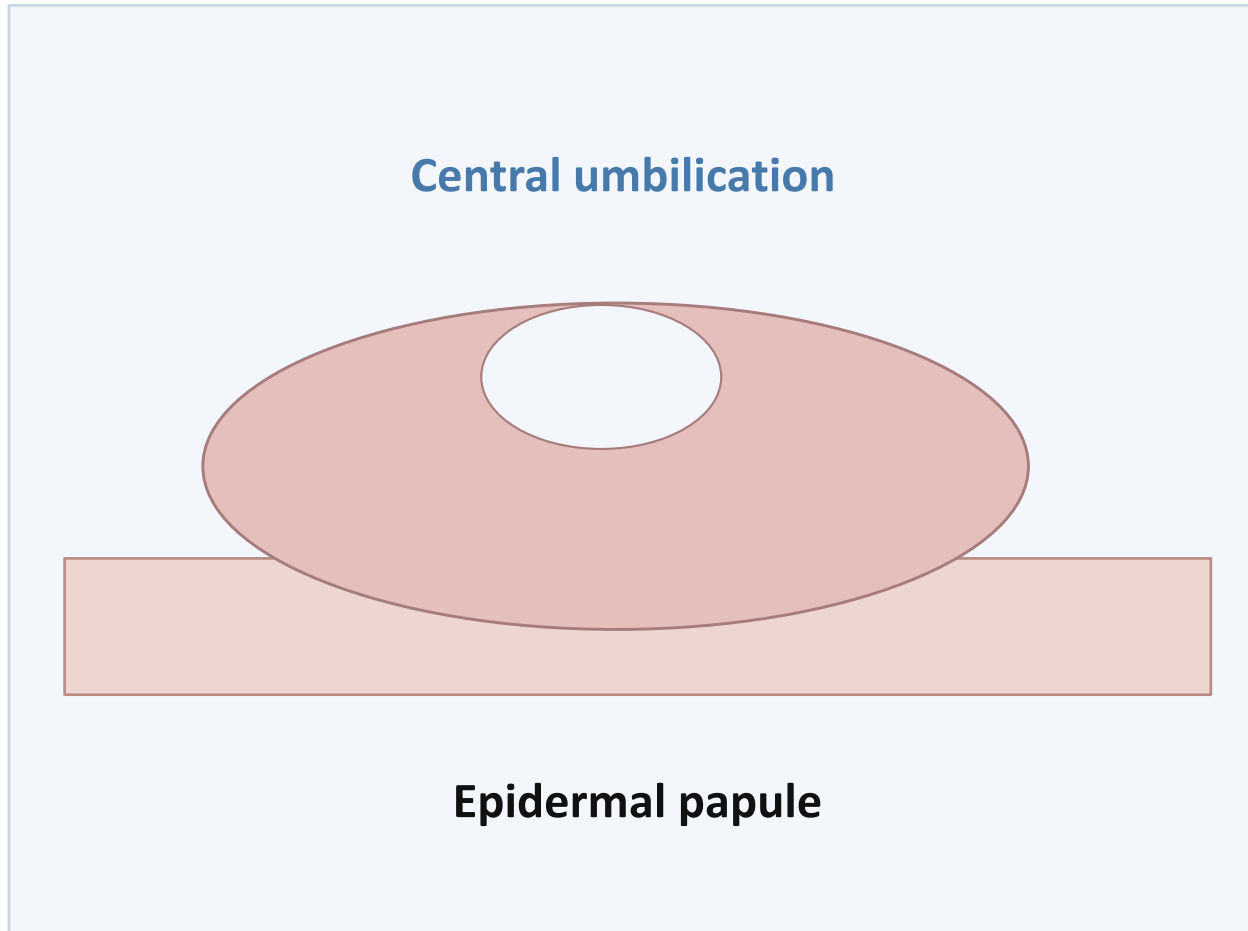
- A brick-shaped virion with a dumbbell-shaped DNA core.
- Its maximal dimension is given as approximately 300 nm.
- These ultrastructural features identify the virus family; They are not needed to diagnose a routine papule clinically.

Clinical hallmark: The umbilicated papule

Morphology can be more decisive than symptoms.

- Firm pink or skin-colored papules show a central umbilication.
- Lesions may be pruritic and are usually multiple.
- The trunk and anogenital areas are common distributions.
- Typical papules around 0.2–0.4 cm; Larger lesions are unusual.

Molluscum: Central umbilication



- A depressed center is characteristic of the clinical papule.
- A curd-like core may be expressed from the depression.
- Surface appearance should be correlated with tissue morphology.

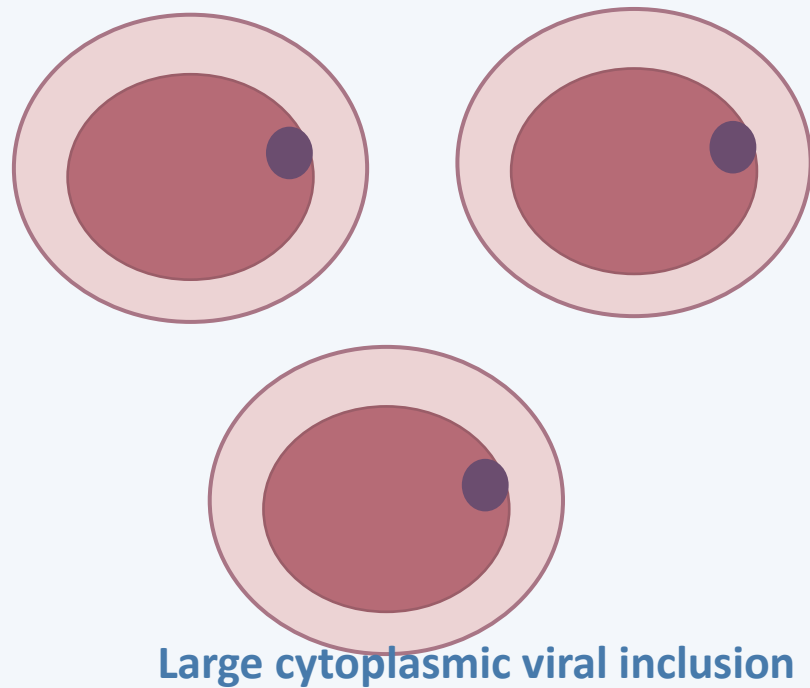
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Histologic configuration: A cup-shaped lesion

Molluscum causes a distinctive growth pattern of epidermis.

- The affected epithelium forms a cup-like focus of verrucous hyperplasia.
- Keratinocytes accumulate large intracytoplasmic viral inclusions.
- The virus-containing structures are concentrated in upper epidermal layers.

Molluscum bodies: An inclusion signature



- Ellipsoid homogeneous inclusions occupy the cytoplasm.
- They may enlarge to approximately 35 μm account.
- Numerous viral particles are present within the inclusions.

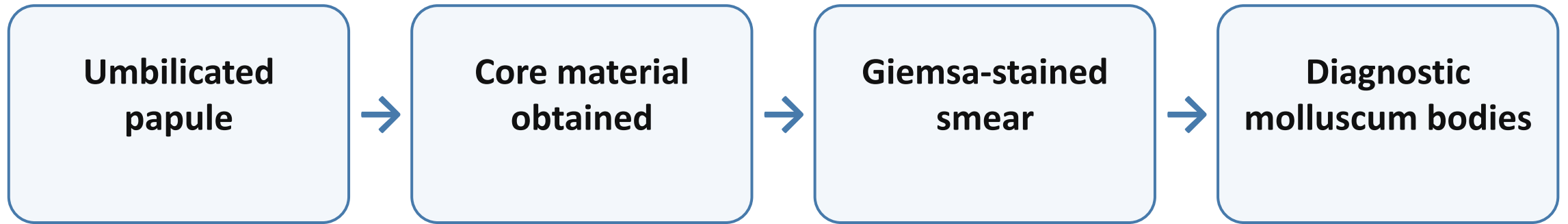
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Molluscum bodies change staining appearance

Their position in the epidermis affects H&E color.

- Eosinophilic inclusions in the stratum granulosum.
- At the stratum corneum, the inclusions can acquire a pale blue appearance.
- The same diagnostic structure is therefore recognizable in more than one staining state.

From molluscum lesion to cytologic confirmation



The textbook specifically describes a smear of expressed core material as a way to display inclusion bodies.

Verruca versus molluscum

Feature	Verruca (HPV)	Molluscum (poxvirus)
Surface	Rough / papillomatous in common wart	Often smooth, centrally umbilicated
Architectural clue	Papillary epidermal hyperplasia	Cup-like epidermal hyperplasia
Signature cytopathy	Koilocytosis	Intracytoplasmic molluscum bodies
Molecular group	Human papillomavirus	Poxvirus

Impetigo is a superficial bacterial infection

Its dominant lesion is pustular and crusted.

- Impetigo is highly contagious and classically affects children.
- Exposed sites, particularly the face and hands, are frequent locations.
- The primary histologic event involves the superficial epidermis.
- Deep dermal involvement is generally absent in uncomplicated lesions.

Contemporary etiology

Historical organisms and present patterns should not be conflated.

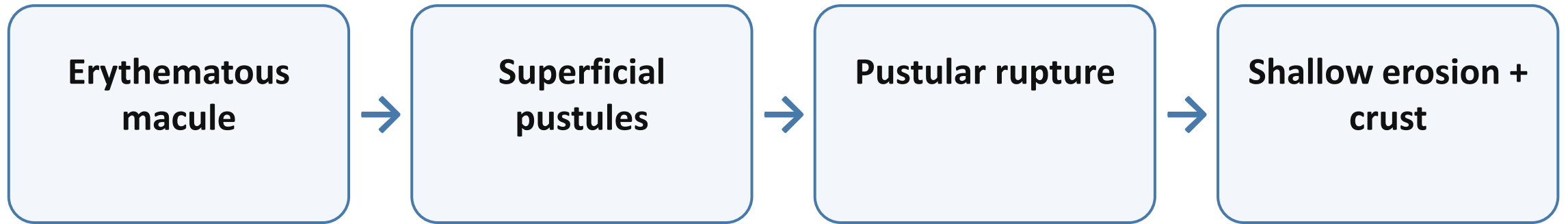
- Older descriptions linked nonbullous impetigo largely to group A β -hemolytic streptococci.
- Bullous impetigo was classically associated with *Staphylococcus aureus*.
- *S. Aureus* now commonly causes both forms.

Nonbullous versus bullous impetigo

Feature	Nonbullous (contagiosa)	Bullous form
Typical morphology	Smaller pustules and erosions	Larger fragile blisters / pustules
Clinical context	Often on exposed sites	Especially encountered in children
Agent in Robbins	<i>S. aureus</i> frequently implicated	<i>S. aureus</i> implicated
Basic tissue plane	Very superficial epidermis	Very superficial epidermis

The source distinguishes forms chiefly by lesion size, not by an entirely different infected organ.

Impetigo lesion evolution



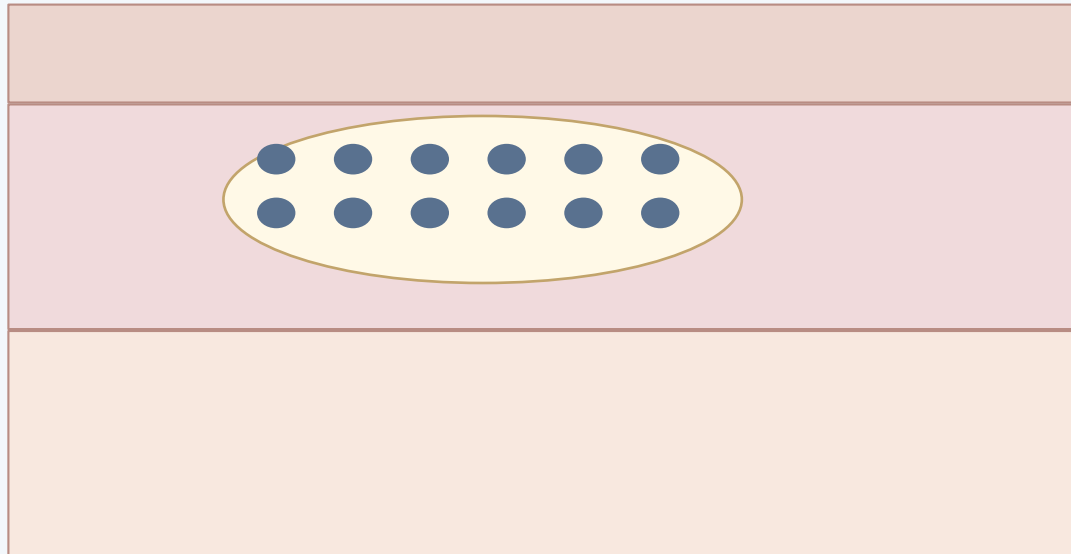
Exuded serum, inflammatory cells and debris dry on the surface; New lesions may appear around the edges.

Why is the crust honey-colored?

A visible sign reflects epidermal exudation.

- Bacteria provoke epidermal injury and a local innate inflammatory response.
- Pustular contents include serum proteins and neutrophils.
- After rupture, serous material and cellular debris dry to form a superficial crust.
- The crust is a consequence of tissue reaction, not a pigmented microbial colony.

Impetigo: Subcorneal pustule



Neutrophil-rich subcorneal pustule

- Neutrophils accumulate immediately beneath the stratum corneum.
- Serum and inflammatory cells occupy a superficial pustule.
- Special stains may demonstrate bacteria in the lesion.

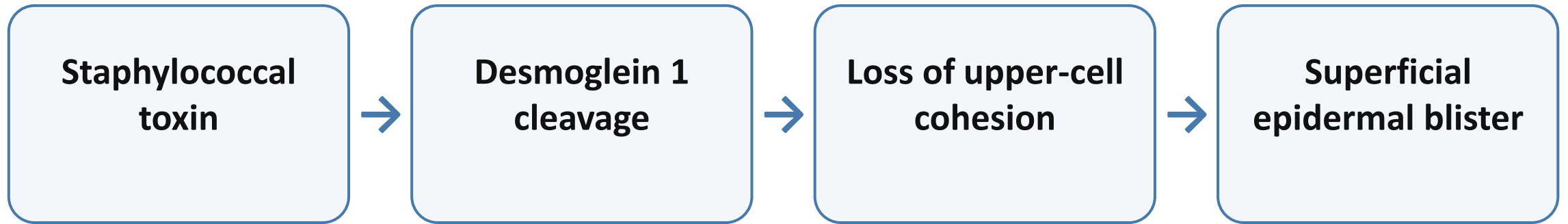
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The bacterial response remains epidermal

Innate immunity can create much of the lesion morphology.

- Microbial presence triggers an acute inflammatory response in the epidermis.
- Reactive epithelial change and mild superficial dermal inflammation accompany pustules.
- The superficial location explains the propensity for crusting and healing without scarring.

Bullous impetigo: Desmoglein 1 injury



Desmoglein 1 is an intercellular adhesion protein; Cleavage creates a high epidermal plane of separation.

One target, two diseases: Dsg1

Feature	Bullous impetigo	Pemphigus foliaceus
Primary mechanism	Bacterial toxin cleaves desmoglein 1	Autoantibodies compromise desmoglein 1
Disease class	Superficial infection	Autoimmune blistering disease
Shared morphology	Superficial epidermal separation	Superficial epidermal separation

Similar blister planes do not prove identical pathogenesis.

Why uncomplicated impetigo does not scar

Healing potential reflects the depth of damage.

- The virtual absence of substantial dermal involvement.
- Epidermal repair can proceed after microbial elimination.
- Deep dermal destruction is not a typical feature of uncomplicated superficial impetigo.

Recognition of impetigo: The combined clues

No single feature should be interpreted in isolation.

- Look for clustered pustules, shallow erosions and adherent yellow-gold crust.
- Consider exposed skin sites and interpersonal spread.
- A subcorneal neutrophilic pustule strongly supports the characteristic tissue reaction.
- Remember that similar-appearing superficial blisters may have noninfectious causes.

Superficial fungal infections favor keratinized sites

Anatomic restriction is foundational.

- The Robbins section contrasts superficial fungi with deep infections of the dermis or subcutis.
- Dermatophyte infections involve cornified skin, hair or nails.
- Different sites produce different tinea names and clinical appearances.

Depth matters in dermatophytosis

Stratum corneum

Living epidermis

Dermis

Subcutis

Dermatophytes

Predominantly cornified surface

HPV / molluscum

Epidermal epithelial lesions

Impetigo

Superficial intraepidermal pustules

Where dermatophytes come from

Transmission and local milieu affect susceptibility.

- Dermatophytes can be associated with soil and animals in the source account.
- Exposure to infected animals is a recognized risk for some tinea corporis lesions.
- Heat, humidity, friction and maceration favor selected distributions.
- Persistent infection in one keratinized site can coexist with spread to others.

Dermatophytosis: The anatomic nomenclature

Name	Location	Characteristic association
Tinea capitis	Scalp / hair	Patchy scale and hair loss
Tinea barbae	Beard region	Adult male beard-area infection
Tinea corporis	Glabrous skin	Expanding scaly plaque
Tinea cruris	Inguinal / upper thigh	Warm, moist fold distribution
Tinea pedis	Feet / toe webs	Scale, erythema and maceration

Tinea capitis: Hair-bearing scalp disease

The hair unit makes the presentation distinctive.

- Scalp dermatophytosis is described mainly in children.
- Patchy lesions may produce mild erythema, scale and crust.
- Associated hair loss is a clinically informative site-specific clue.

Tinea barbae: Beard-area infection

The same family of organisms presents differently by site.

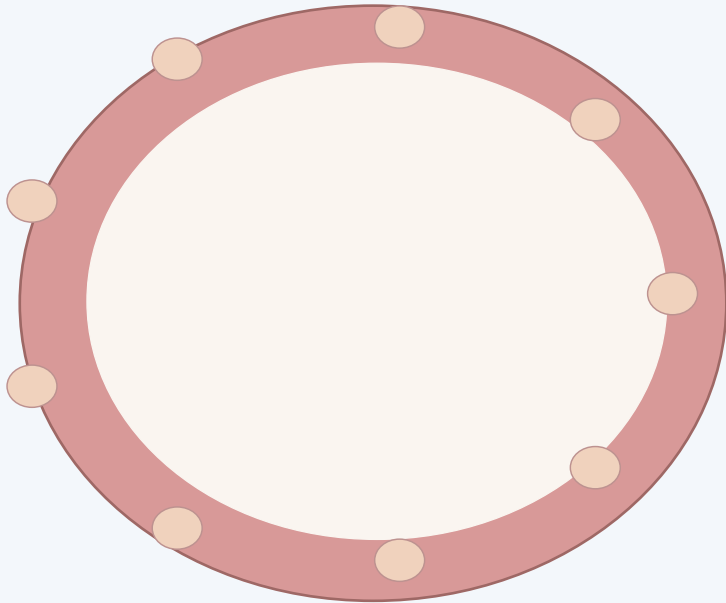
- Dermatophytes can involve the beard region.
- The disease is comparatively uncommon in the Robbins survey.
- Its recognition requires distinguishing a keratin-related infection from other follicular lesions.

Tinea corporis: An expanding annular lesion

The active edge often provides the key morphological clue.

- Typical disease is a round, slightly erythematous plaque.
- The peripheral border is raised and scaly.
- Heat, humidity, exposure to animals and another dermatophyte focus are described risk contexts.

Tinea corporis: The border is diagnostic



Expanding raised scaly edge

- An outwardly expanding scaly edge defines the lesion.
- The center may appear less active than the advancing margin.
- A ring-like lesion is a clinical pattern, not proof of an organism without context.

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Tinea cruris: Macerated inguinal skin

The local environment shapes disease distribution.

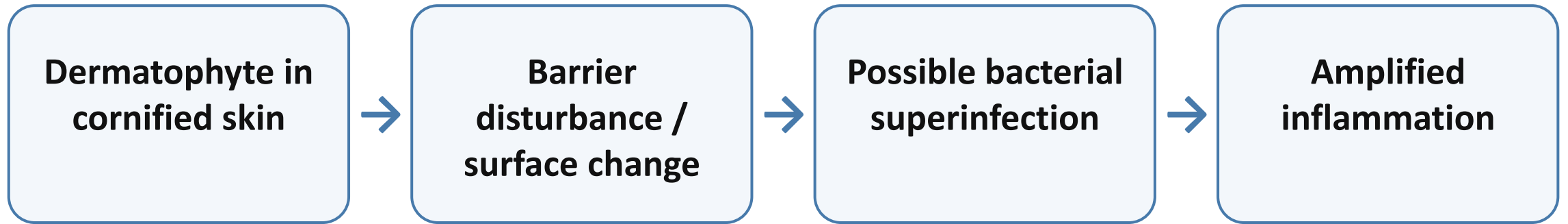
- The process commonly affects the upper inner thighs and groin.
- Heat, friction and maceration are predispositions.
- The lesions appear as moist, erythematous patches with raised scaly borders.

Tinea pedis: Interdigital scaling

Toe webs are a common starting location.

- Scaling and erythema frequently localize initially to interdigital spaces.
- The source notes that superimposed bacterial infection can drive a substantial component of inflammation.
- Not every inflamed area in tinea pedis is a direct effect of dermatophyte growth.

Tinea pedis: Mixed microbial contributions



Primary dermatophytosis from inflammation attributable to secondary bacteria at some sites.

Nail involvement: Onychomycosis

Keratinized nail tissue creates a different visible lesion.

- Dermatophyte infection may involve nails directly or extend from skin infection.
- The nail plate can become thickened, discolored and deformed.
- The gross nail changes indicate a differential diagnosis; Species assignment requires appropriate material.

Tinea versicolor is not a dermatophytosis

The causative organism belongs to a different fungal group.

- Tinea versicolor to *Malassezia furfur*, a yeast.
- The upper trunk is a typical distribution.
- Patchy macules of variable coloration have fine peripheral scaling.
- Do not classify every condition called “tinea” as a dermatophyte infection.

Dermatophyte infection versus tinea versicolor

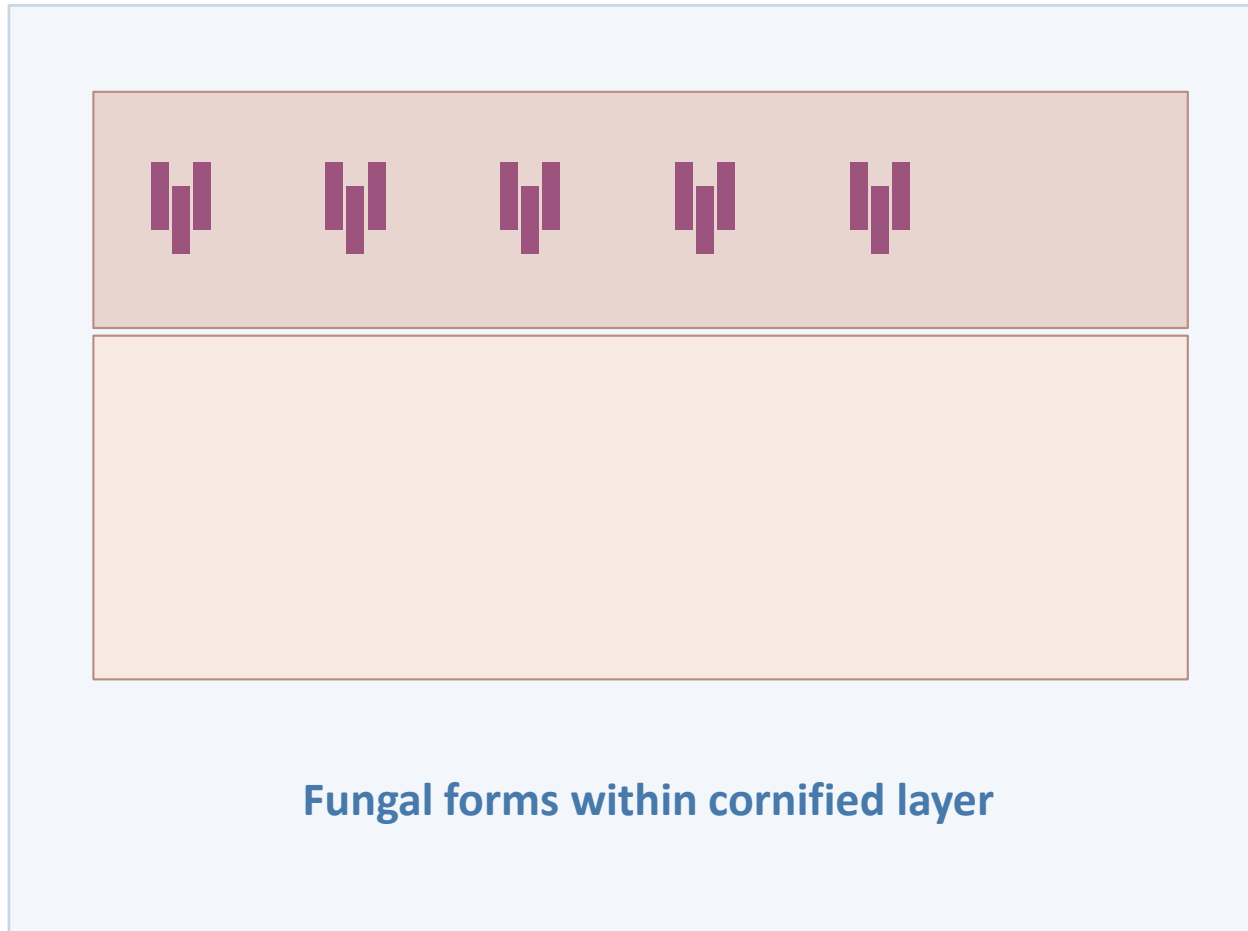
Feature	Dermatophyte disease	Tinea versicolor
Agent	Dermatophyte fungi	Malassezia furfur (yeast)
Distribution	Site-specific skin, hair, nails	Most often upper trunk
Lesion pattern	Scale; annular edge in tinea corporis	Varicolored macules with fine scale
Classification	Dermatophytosis	Superficial yeast-associated eruption

Dermatophyte histology is not always specific

Host reaction and superinfection modify the slide.

- Routine sections may show a mild spongiotic or eczematous reaction.
- Intraepidermal neutrophils can accompany infection.
- Morphology varies with organism, host response and bacterial coinfection.
- The organism may be inconspicuous on routine H&E sections.

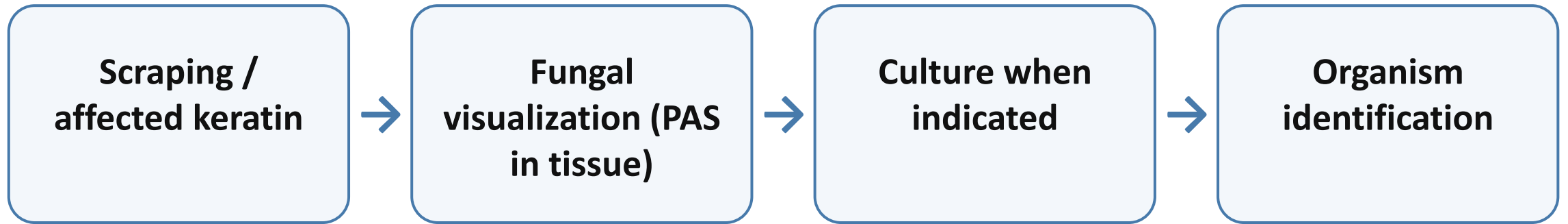
Fungal structures are found in cornified tissue



- Look in anucleate stratum corneum or other keratinized specimens.
- Hyphae can be highlighted by periodic acid–Schiff (PAS).
- The conceptual diagram indicates location, not a diagnostic photomicrograph.

Original schematic; Not a clinical image or photomicrograph

Confirmatory fungal examination



Culture of material scraped from affected cornified skin, hair or nails as a route to species identification.

Histologic signatures: A comparative map

Infection	Architecture / level	Key feature
Verruca	Papillomatous epidermal expansion	Koilocytosis
Molluscum	Cup-like epidermal hyperplasia	Large cytoplasmic inclusions
Impetigo	Subcorneal pustule	Neutrophils; bacteria may be demonstrable
Dermatophytosis	Cornified layer involvement	Hyphae, enhanced by PAS

Clinical surface patterns: Rapid recognition

Surface sign	Most relevant exemplar	Interpretation
Rough keratotic papules	Common wart	Papillomatous proliferation
Dome-like umbilicated papules	Molluscum	Poxvirus inclusion-producing lesion
Honey-colored crust	Impetigo	Ruptured superficial pustule
Raised scaly annular border	Tinea corporis	Keratin-involving fungal infection

A rational approach to tissue confirmation

Sample and stain for the question being asked.

- HPV-associated lesions are interpreted by papillary architecture and koilocytosis; Typing is an additional molecular question.
- Molluscum bodies are visible in lesion histology or potentially in a smear of core material.
- Impetigo is supported by a subcorneal neutrophilic pustule and organism demonstration when needed.
- Dermatophytosis is evaluated in cornified material, with PAS or culture as applicable.

Case discussion: Centrally umbilicated papules

A school-age child has several firm, skin-colored papules on the trunk. Each has a small central depression. One papule yields a curd-like central core.

Diagnostic question

Which infectious lesion best explains the surface morphology, and what cellular inclusion would you seek?

Discussion: Link the clinical clue to its tissue-level signature.

Case discussion: Superficial golden crust

A child develops a red macule that evolves into small pustules. Several rupture, leaving shallow erosions with adherent yellow-gold crust on exposed facial skin.

Diagnostic question

What is the most likely process, and where should neutrophils be found histologically?

Discussion: Link the clinical clue to its tissue-level signature.

Case discussion: Enlarging scaly plaque

A patient presents with a rounded erythematous plaque on the trunk. The edge is elevated and scaly, with gradual outward extension.

Diagnostic question

Which infectious group is most likely, and what tissue compartment should be evaluated?

Discussion: Link the clinical clue to its tissue-level signature.

Case discussion: A rough papule on the hand

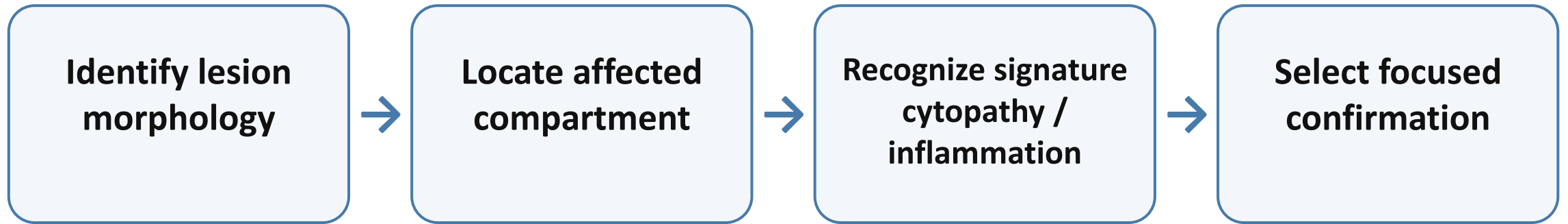
A young patient has a longstanding rough papule near a fingernail. The surface is irregular and pebble-like.

Diagnostic question

What infection is likely, and what viral cytopathic change should be sought?

Discussion: Link the clinical clue to its tissue-level signature.

Integrating agent, lesion and tissue signature



Reliable dermatopathologic inference combines clinical architecture, host context and the correct microscopic level.

Final synthesis: Four mechanisms, four visual signatures

The clinically visible lesion arises from a specific tissue process.

- HPV: Epidermal hyperplasia plus koilocytic cytopathy → verrucous papule.
- Poxvirus: Abundant cytoplasmic viral inclusions → umbilicated papule.
- Impetigo: Superficial neutrophilic injury, sometimes toxin-mediated splitting → pustule and crust.
- Superficial fungi: Growth in cornified tissue → scale, annularity or nail/hair changes.

Primary reference

Textbook material underlying this lecture

- Robbins, Cotran & Kumar. Pathologic Basis of Disease. 11th edition (2026). Chapter 25: The Skin.
- Relevant textbook sections: Infection; Verrucae (Warts); Molluscum Contagiosum; Impetigo; and Superficial Fungal Infections.