

Seborrheic Dermatitis

A clinicopathologic lecture

Morphology • Pathogenesis • Differential diagnosis

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What this lecture will establish

- Recognize characteristic distribution and lesion morphology
- Explain multifactorial pathogenesis without assuming a single cause
- Interpret acute and chronic histologic patterns
- Differentiate close mimics and justify management principles

An epidermal disease, not sebaceous-gland inflammation

Sebum-rich skin

Inflamed epidermis

Association ≠ identity

A frequent chronic inflammatory dermatosis

- Involvement of up to 5% of the general population
- Often recurrent, especially in adulthood
- Usually recognized from clinical pattern rather than laboratory assays

Three diagnostic anchors

Typical sites: Scalp, central face, ears, presternal chest

Clinical appearance:
Erythematous-yellow base
with greasy scale

Histology: Spongiosis,
acanthosis, and follicular
parakeratosis

The regional distribution is not random

Scalp
Dandruff / scale

Face
Glabella · nasolabial folds

Ear
Canal · postauricular

Trunk
Presternal chest

Folds
Axillae / intertriginous

Why location provides diagnostic leverage

- Multiple involved sebaceous-rich sites strengthen the clinical impression
- Examination should extend beyond the most symptomatic area
- Atypical distribution increases the importance of mimics

Scalp disease includes dandruff

- Dandruff is a common clinical expression on the scalp
- Loose scale may dominate when erythema is subtle
- Scalp inspection should include hairline and postauricular skin

Facial localization follows a characteristic map

Central forehead
Glabella and eyebrows

Central face
Nasolabial folds

Lower face
Beard / moustache

Periorbital margin
Possible eyelid margin involvement

The ears require active inspection

- External auditory canal can be affected
- Retroauricular erythema and scale may be easily overlooked
- Fissures are particularly relevant behind the ears

The chest may show conspicuous scaling

- Presternal skin is a textbook-predilection site
- Adult disease may involve anterior chest and upper trunk
- Distribution and scale quality are often more useful than color alone

Intertriginous disease extends the phenotype

- Axillary, inframammary, and groin folds may be involved
- Fold erythema can have less conspicuous scale
- Consider Candida and other intertriginous dermatoses when morphology differs

Infantile seborrheic dermatitis: Cradle cap

- Greasy adherent scalp scale is a classic infant presentation
- Can extend to axillary and diaper folds
- Infants are often less troubled by pruritus than children with atopic dermatitis

Infant and adult disease differ in course

Infantile

Cradle cap; Folds may be involved

Often self-limited

Gentle scale management

Adult

Scalp, face, ears, chest

Chronic relapsing tendency

Maintenance may be needed

Clinical color varies with skin pigmentation

- Erythema may appear pink, red, or less obvious on darker skin
- Scale, distribution, and symptoms remain diagnostically useful
- Avoid restricting recognition to one photographed skin tone

Pruritus is variable rather than obligatory

- Scalp discomfort and itching may occur
- Minimal itch does not exclude active facial disease
- Marked itch or exudation should prompt a wider differential

The etiology is multifactorial and unresolved

No single necessary-and-sufficient mechanism has been established

Sebum, skin-resident yeast, epidermal barrier, and host response interact

Associations should not be presented as proven causal sequences

Sebum: A permissive environment, not a diagnosis

- Increased sebum production is a possible contributor
- Androgen-dependent changes may influence sebaceous activity
- Oily skin alone does not explain who develops dermatitis

The acne comparison tests the sebum hypothesis

Increased sebum

Different inflammatory outcomes

Natural counterexample to sufficiency

Parkinson disease provides clinical insight

- Seborrheic dermatitis is more frequent in Parkinson disease
- The Robbins account links dopamine deficiency with increased sebum
- This observation supports—but does not prove—a sebum contribution

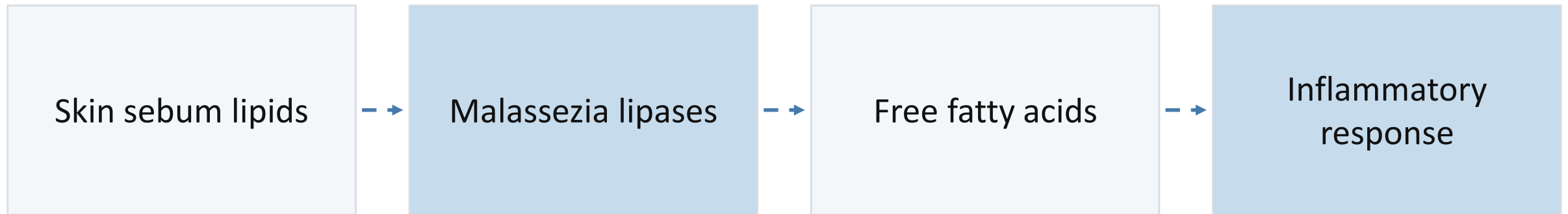
Levodopa response is an association, not a therapy rule

- In Parkinson disease, levodopa may reduce oiliness
- Improvement of dermatitis has been observed in parallel
- Do not extrapolate levodopa as seborrheic dermatitis therapy

Malassezia is a plausible inflammatory cofactor

- Malassezia yeasts may colonize sebaceous-rich skin
- The organism is often part of the normal cutaneous microbiota
- Presence alone does not demonstrate pathogenic causation

The proposed Malassezia–lipase mechanism



Proposed associations; Not a demonstrated linear pathway

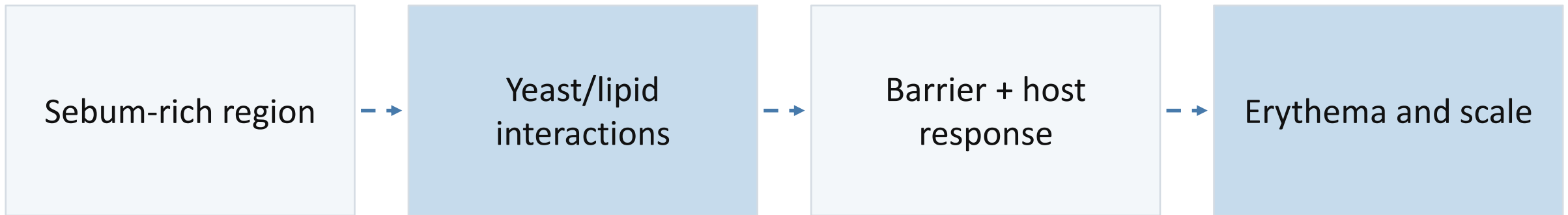
Barrier integrity helps explain individual susceptibility

- Differences in epidermal barrier function may modify exposure
- Altered barrier lipids could facilitate irritant responses
- Barrier impairment complements rather than replaces the sebum–yeast model

Host immune response shapes clinical severity

- Inflammation is not explained by yeast quantity alone
- Host cutaneous immune responses vary
- The same ecological exposure may yield different phenotypes

A defensible integrated pathogenesis model



Proposed associations; Not a demonstrated linear pathway

Severe HIV-associated disease is a clinical clue

- Severe, treatment-refractory disease occurred with low CD4 counts
- The frequency of this severe phenotype declined with effective ART
- Severe atypical disease warrants assessment of clinical context

Clinical phenotype: Color, texture, and contour

Erythematous-yellow base

Macules or papules

Greasy scale

Crusting

What “greasy scale” adds to the examination

- Scale character is one clue, not an isolated diagnostic test
- Interpret scale together with site and erythematous base
- Psoriasis and eczema may overlap clinically

Retroauricular fissuring can be diagnostically useful

- Fissures may develop behind the ears
- Check both sides of the postauricular crease
- Fissures also require assessment for irritation or infection

Histology evolves as lesions persist

Earlier

Intercellular epidermal edema

More spongiotic pattern

Inflammatory scale may be focal

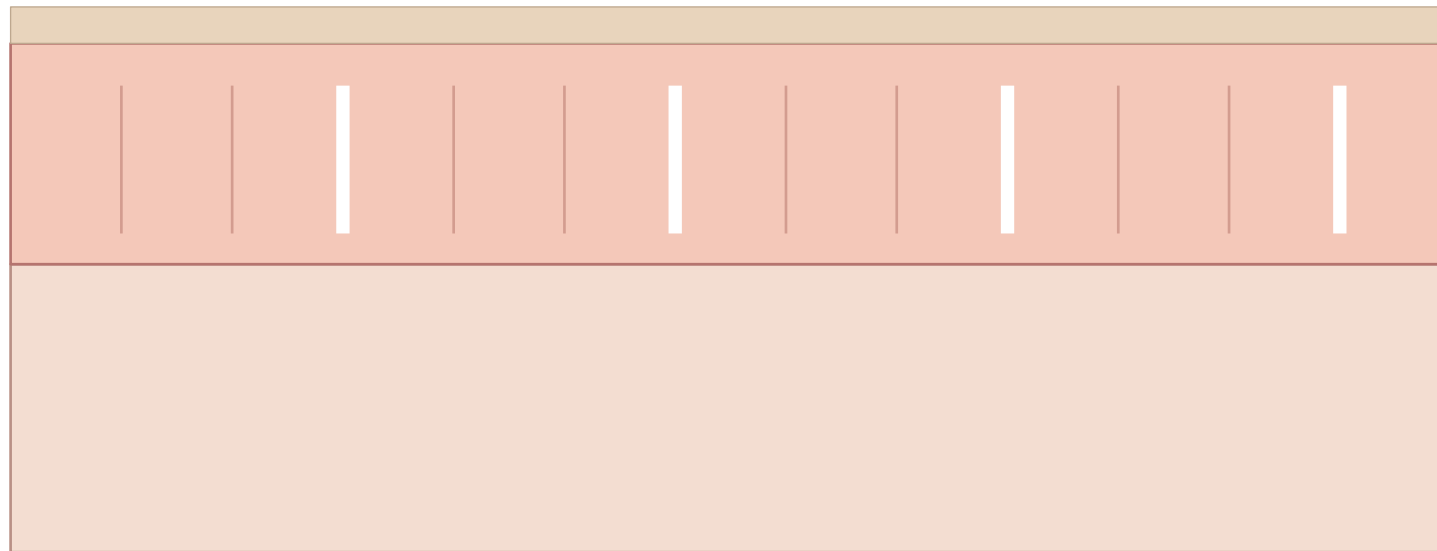
Later

Reactive epidermal thickening

More acanthotic pattern

Overlap with psoriasis can increase

Spongiosis means intercellular epidermal edema



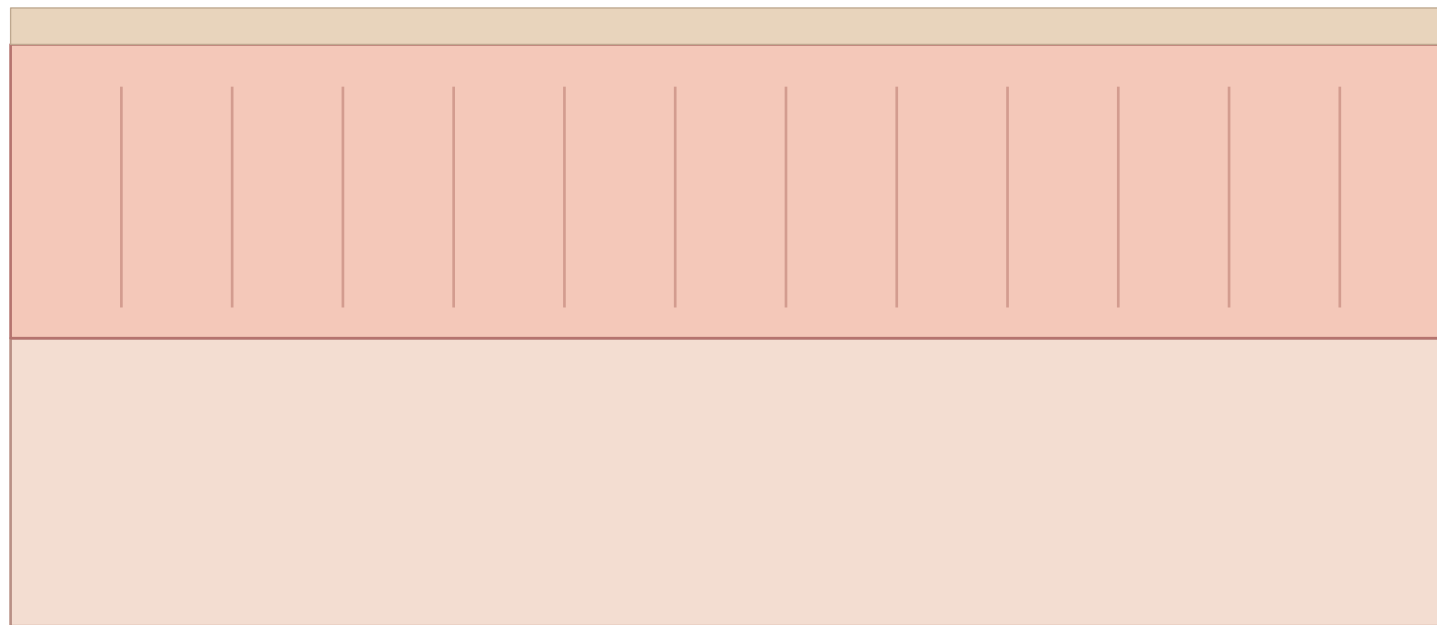
Intercellular edema

Spongiotic reaction pattern

Characteristic of earlier lesions

Schematic illustration — not a histologic specimen

Acanthosis reflects reactive epidermal thickening



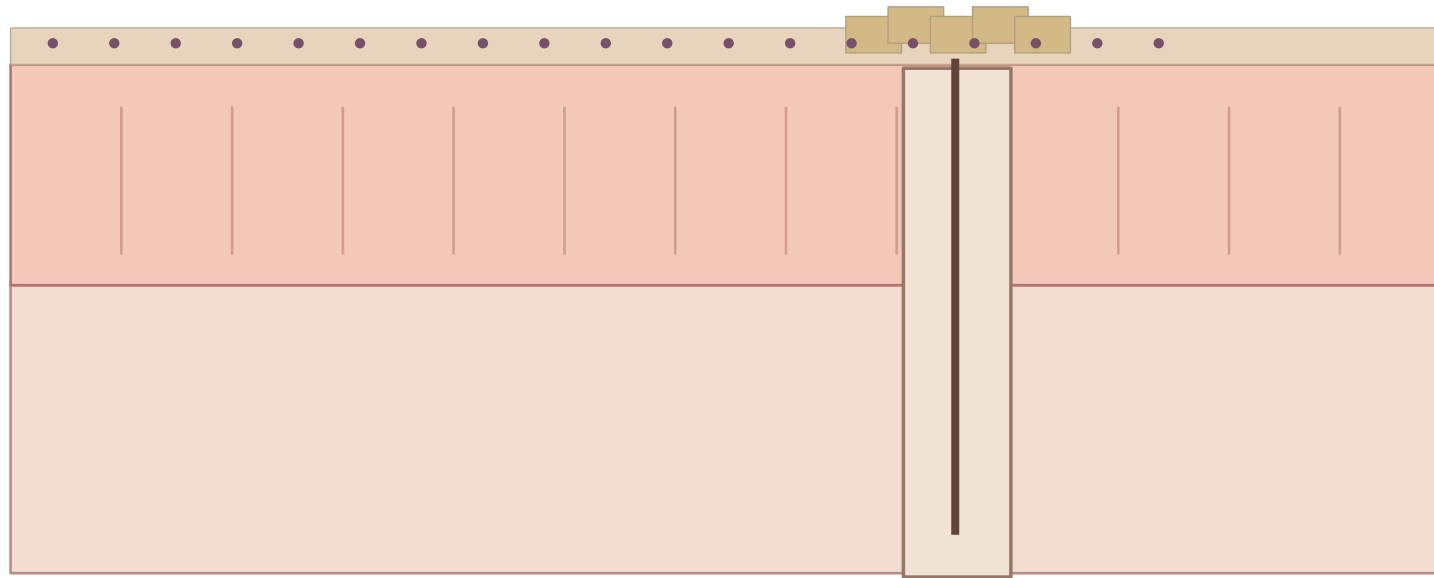
Expanded viable epidermis

Reactive thickening

More prominent in chronic lesions

Schematic illustration — not a histologic specimen

Parakeratosis records altered cornification



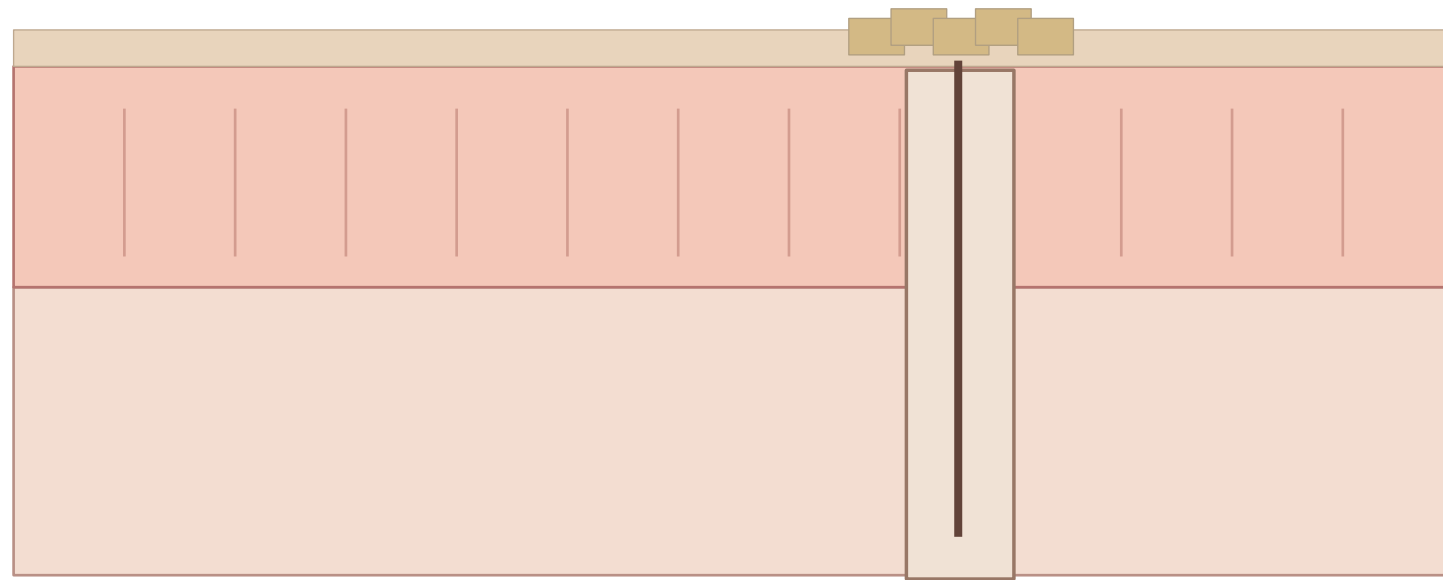
Retained nuclei in surface
scale

Altered keratinization

May localize to follicular
openings

Schematic illustration — not a histologic specimen

Follicular lipping is a characteristic clue



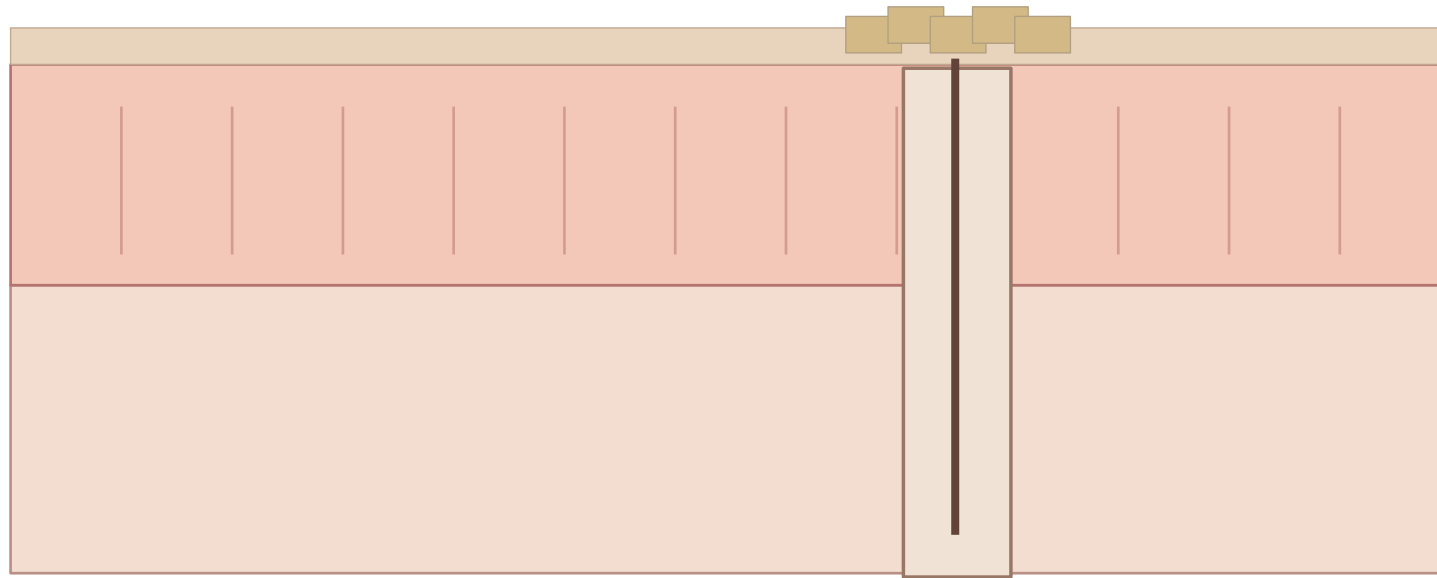
Parakeratotic mound

Follicular opening (ostium)

Neutrophils / serum may be admixed

Schematic illustration — not a histologic specimen

The follicular ostium is the diagnostic landmark



Follicular infundibulum

Surface scale at the ostium

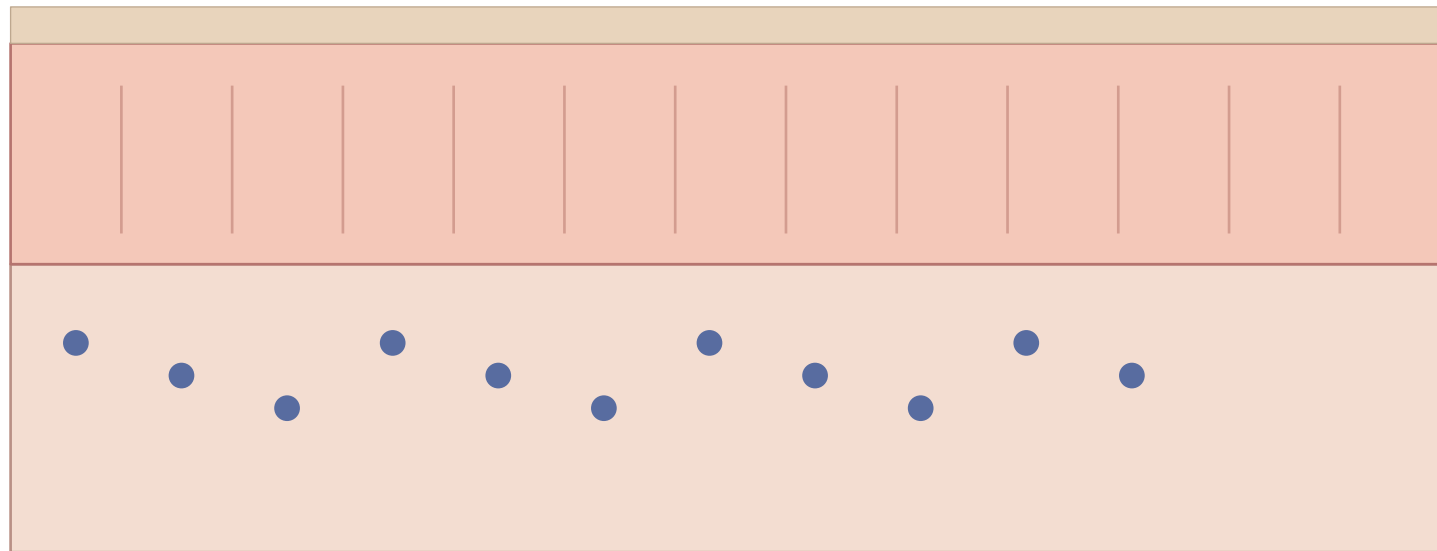
Assess adjacent epidermis

Schematic illustration — not a histologic specimen

Neutrophils and serum may enter surface crust

- Follicular parakeratotic mounds can contain neutrophils
- Serous exudate contributes to crust formation
- Neutrophils alone are not specific: Distribution matters

Dermal inflammation is predominantly superficial



Superficial dermis

Perivascular inflammatory
cells

Epidermal changes remain
essential

Schematic illustration — not a histologic specimen

Integrated biopsy interpretation

Reaction pattern

Follicular relationship

Inflammatory distribution

Why scalp psoriasis is the closest mimic

- Both can create scaling and erythematous scalp plaques
- Both may show acanthosis and parakeratosis
- Location alone is insufficient in isolated scalp disease

Morphologic comparison with psoriasis

Seborrheic dermatitis

Follicular lipping / plugging

Spongiosis, especially early

Greasy or bran-like scale

Psoriasis

Regular elongated rete ridges

Suprapapillary thinning

Characteristic neutrophilic microabscesses

Scalp biopsy evidence must be interpreted cautiously

- A comparative study included 20 seborrheic dermatitis and 15 psoriasis cases
- Follicular plugging and shoulder parakeratosis favored dermatitis
- Its small, selected cohort does not yield a universal diagnostic rule

Immunohistochemistry is not a routine separator

- A published comparative scalp study tested several IHC markers
- These stains did not improve differentiation in that cohort
- Use clinicopathologic correlation rather than an invented immunophenotype

Sebopsoriasis describes overlap—not a unique stain

- Clinical features may overlap between the two entities
- Consider typical lesions at other body sites
- Do not force a categorical diagnosis when evidence is mixed

Contact and atopic eczema are important mimics

- All can show spongiotic inflammatory changes
- Exposure distribution and clinical history are central
- A biopsy pattern of spongiosis alone cannot specify etiology

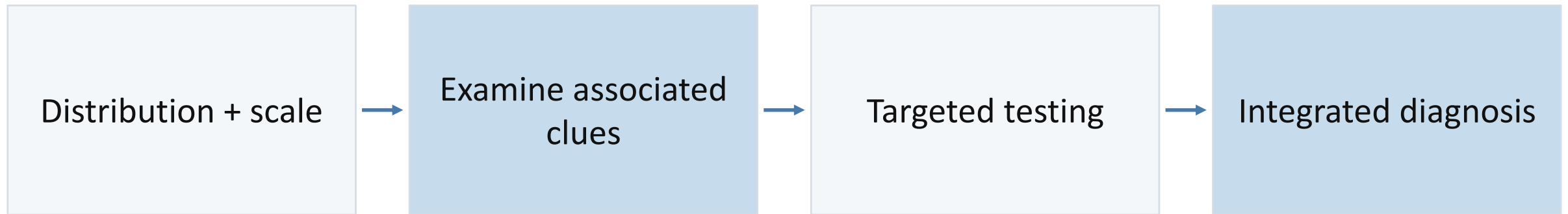
Tinea demands a different diagnostic pathway

- Scaly disease may reflect a dermatophyte infection
- Suspected fungal infection may warrant direct mycologic testing
- Do not use *Malassezia* colonization as a surrogate for dermatophyte diagnosis

Other facial and fold mimics require attention

- Rosacea may produce central facial erythema
- Candida can affect moist skin folds
- Contact dermatitis can follow topical products or devices

A focused differential diagnosis workflow



Diagnosis is usually clinical

- Characteristic location and lesion appearance are central
- The chronic or relapsing pattern provides context
- Histology is an adjunct for uncertain or atypical cases

When a biopsy adds value

- Unexpected distribution or morphology
- Poor response that raises an alternative diagnosis
- Need to separate overlapping inflammatory reaction patterns

Why a yeast-positive scraping is not definitive

- Malassezia can exist on uninvolved normal skin
- Microscopic detection does not automatically establish disease
- Clinical inflammatory phenotype is the decisive context

Therapy targets more than one disease component

Antifungal

Anti-inflammatory

Scale control / maintenance

Antifungals have controlled-trial support

- Cochrane 2015 reviewed 51 trials involving 9,052 participants
- Topical ketoconazole and ciclopirox outperformed vehicle in relevant comparisons
- Most studies had short follow-up; Sustained control remains less well studied

Antifungal evidence is not absolute

- Substantial between-study differences affect certainty
- Claims of superiority among individual antifungals are limited
- Short-term improvement should not be misrepresented as permanent cure

Anti-inflammatory therapy reduces active inflammation

- Topical corticosteroids can relieve erythema and scaling
- Calcineurin inhibitors offer a nonsteroidal option for selected facial disease
- Most randomized anti-inflammatory trials were short term

The scalp often needs a vehicle-specific regimen

- Medicated shampoos can deliver antifungal or keratolytic agents
- Short anti-inflammatory courses may control exacerbations
- Regimen selection depends on scalp tolerability and recurrence

Face and ear treatment requires restraint

- Topical antifungal creams are commonly used
- Limit corticosteroid exposure on thin facial skin
- Consider nonsteroidal anti-inflammatory alternatives when frequent therapy is required

Gentle infant management is distinct

- Regular gentle washing loosens scalp scale
- Avoid traumatic scale removal
- Escalate or reassess if presentation is extensive or atypical

Relapsing disease requires follow-up planning

- Explain the chronic or recurrent nature in adults
- Adjust maintenance treatment according to distribution and flares
- Reconsider diagnosis if the expected response is absent

Clinical exercise: Greasy scale in typical sites

A patient has scaling of the scalp, glabella and nasolabial folds
Examination reveals yellowish scale over erythematous skin

Which distribution and morphologic findings support the diagnosis?

Discussion prompt; Explanation in presenter notes

Clinical exercise: A refractory widespread eruption

A patient has extensive seborrheic-pattern dermatitis resistant to treatment
The clinical history includes possible immunosuppression

What associated conditions and diagnostic mimics require assessment?

Discussion prompt; Explanation in presenter notes

Histology exercise: Ostial parakeratosis

Scalp biopsy has spongiosis, follicular parakeratotic mounds and a superficial infiltrate
Which constellation favors seborrheic dermatitis?

What histologic features would redirect toward psoriasis?

Discussion prompt; Explanation in presenter notes

Seborrheic dermatitis: Integrated synthesis

- A chronic inflammatory epidermal disorder with sebaceous-rich topography
- Sebum, Malassezia, barrier and host response are interacting contributors
- Follicular lipping and stage-dependent spongiosis/acanthosis support the biopsy
- Clinical context remains essential for diagnosis and treatment

Selected references

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