
Acute Eczematous Dermatitis

A clinicopathologic lecture

From antigen exposure to spongiotic inflammation

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Learning objectives

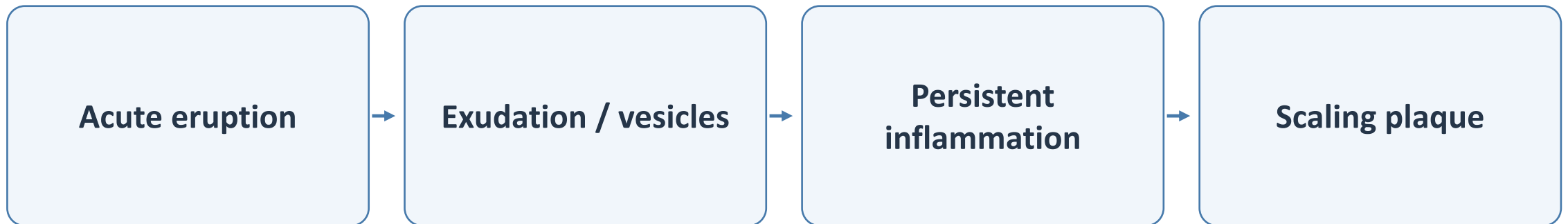
- Classify major initiating contexts for eczematous dermatitis
- Trace allergic contact sensitization and re-exposure
- Explain epidermal spongiosis and vesicle formation
- Connect lesion evolution with histopathology and management

Eczema is a clinicopathologic reaction pattern

- Acute eczematous dermatitis is among the common inflammatory skin disorders
- “Eczema” describes an acute papulovesicular, often weeping presentation
- Multiple distinct initiating factors can converge on spongiotic epidermal injury

The phase of disease changes the phenotype

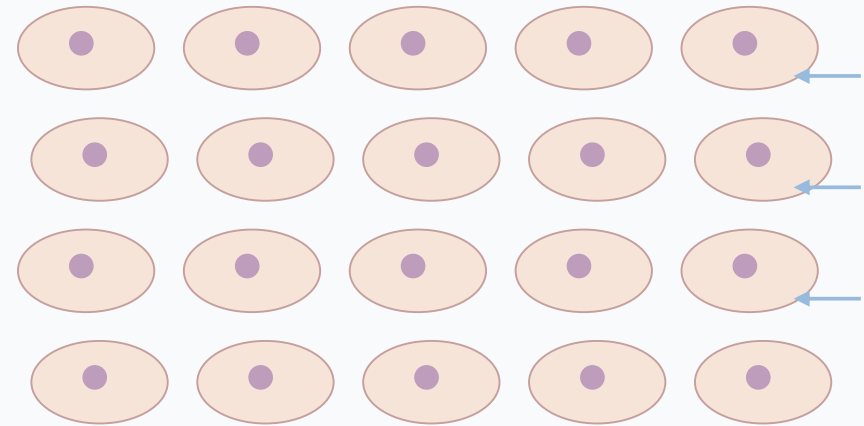
- Acute: Erythema, edema, papulovesicles, oozing and crusts
- Persistent lesions: Reactive epidermal thickening and increased scale
- A single eruption may exhibit features of more than one phase



Spongiosis defines the acute tissue response

- Spongiosis means edema within intercellular spaces of epidermis
- Fluid separates keratinocytes, particularly in the stratum spinosum
- Progressive spongiosis can culminate in intraepidermal vesicles

EPIDERMAL INTERCELLULAR EDEMA

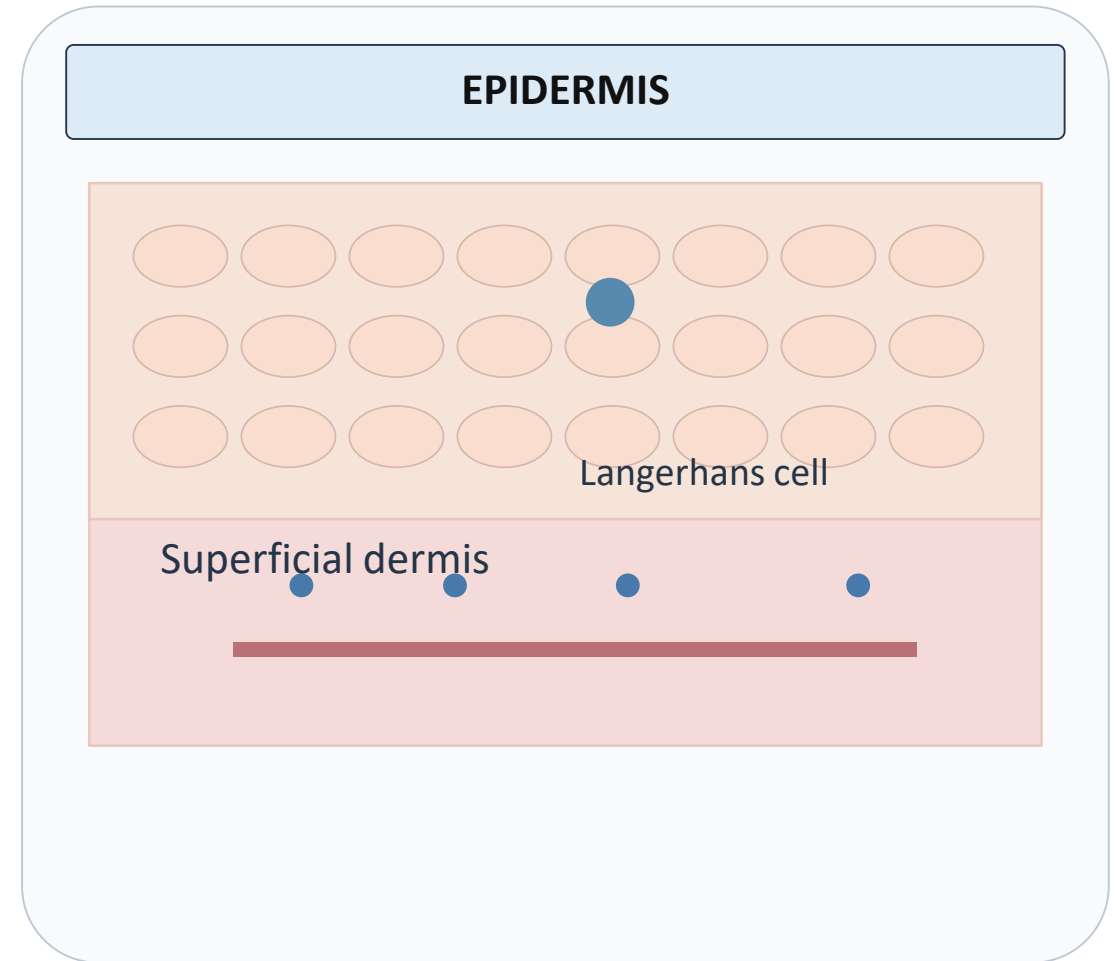


Fluid widens spaces

Schematic

The epidermis and dermis play different roles

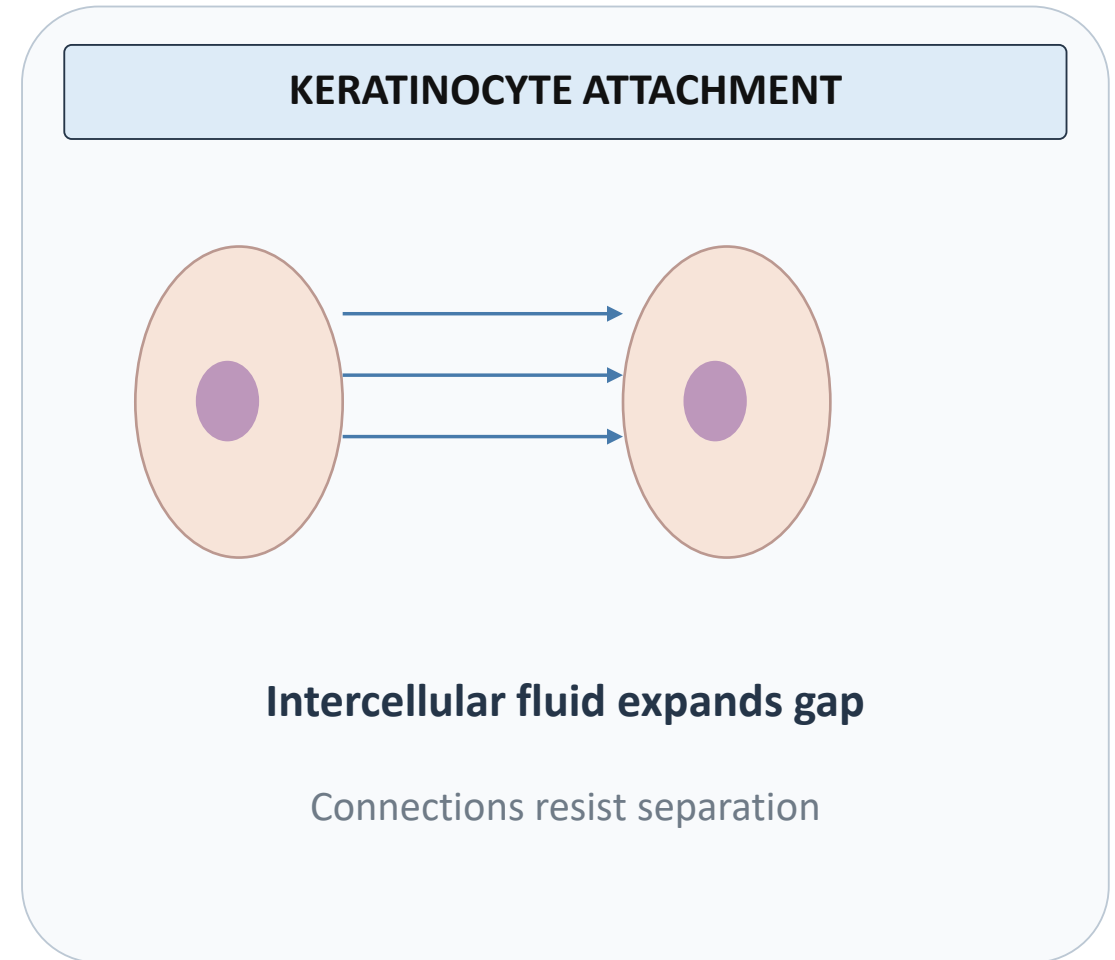
- Epidermis: Keratinocytes, desmosomes and intraepidermal dendritic cells
- Papillary dermis: Vessels and sites of early inflammation and edema
- Eczematous inflammation links dermal recruitment to epidermal change



Schematic

Keratinocyte adhesion normally resists separation

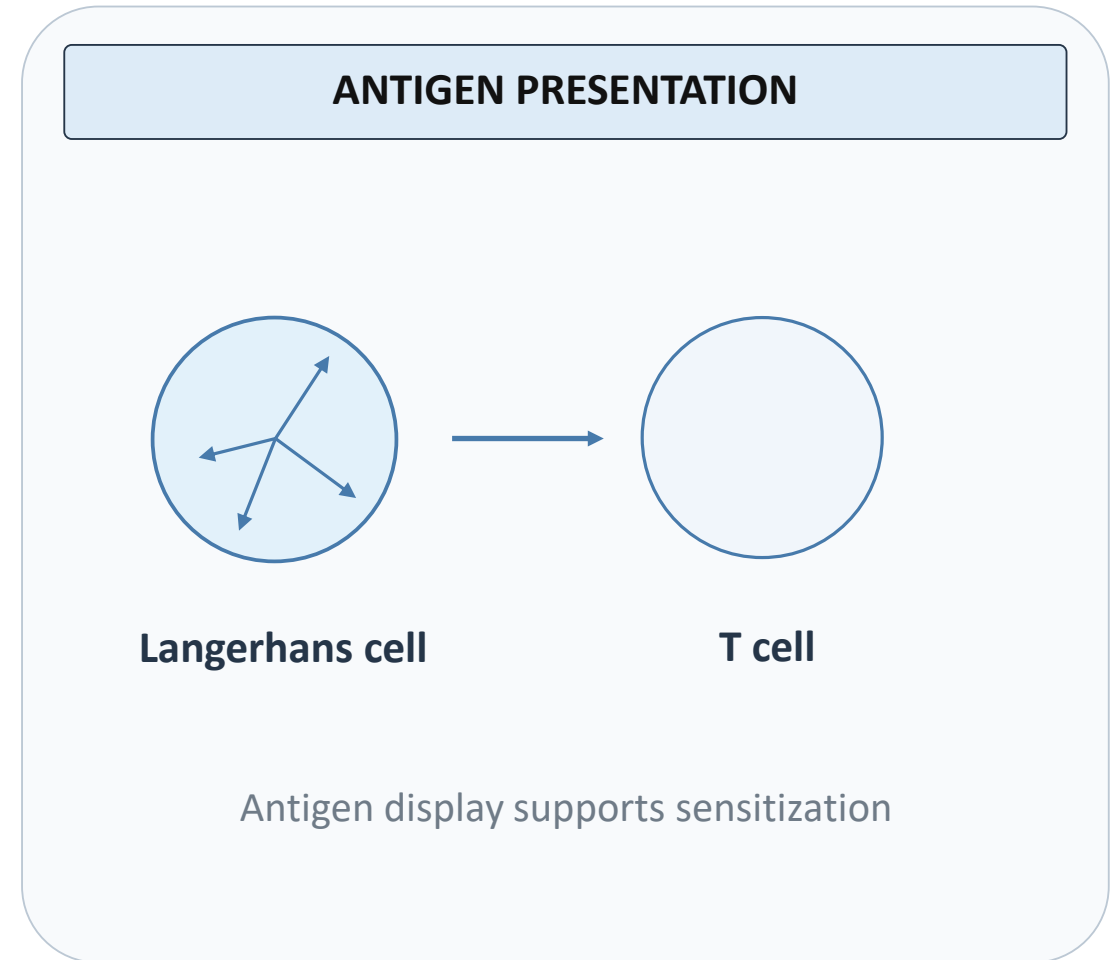
- Desmosomes connect adjacent keratinocytes and help maintain skin integrity
- Keratin forms a durable barrier in the epidermis
- Interstitial epidermal fluid mechanically widens spaces between cells



Schematic

Langerhans cells connect skin to adaptive immunity

- Intraepidermal Langerhans cells capture and process antigens
- They migrate toward regional lymph nodes for T-cell presentation
- This function is central to allergic contact sensitization



Schematic

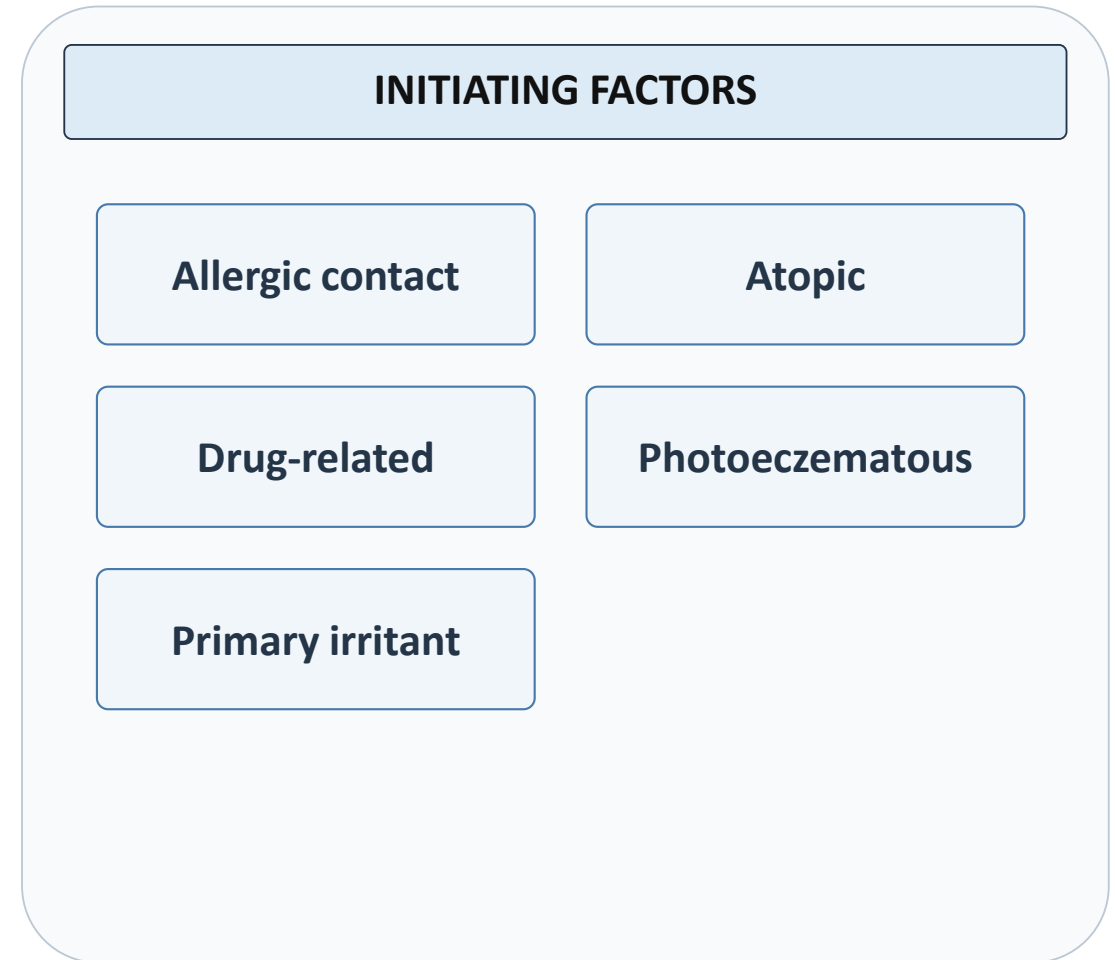
Skin-homing T cells return to sites of antigen

- Activated lymphocytes can express cutaneous lymphocyte-associated antigen (CLA)
- Chemokine receptors aid localization to cutaneous inflammation
- On re-exposure, memory T cells accumulate at the site of antigen



Five initiating contexts are recognized

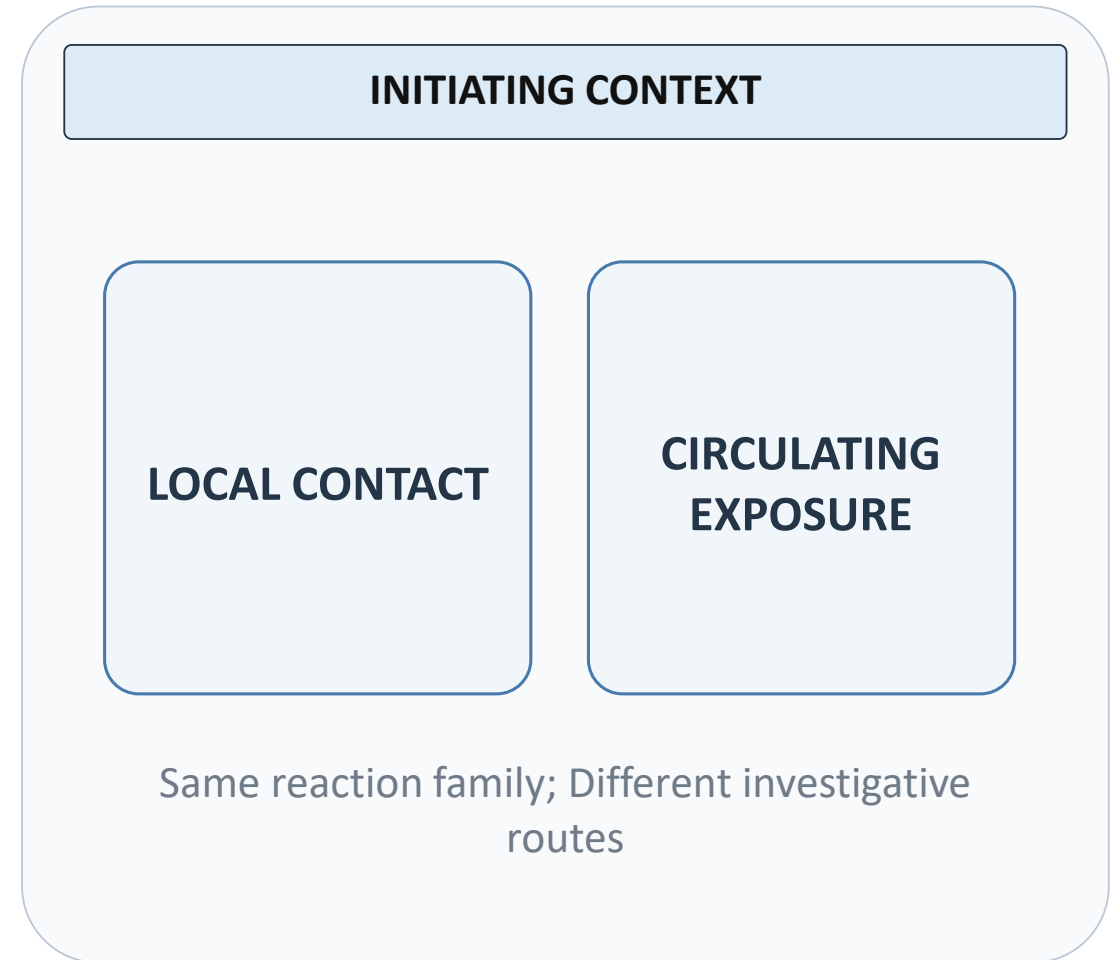
- Allergic contact dermatitis; Atopic dermatitis
- Drug-related eczematous dermatitis;
Photoeczematous dermatitis
- Primary irritant dermatitis



Schematic

External versus internal antigen exposure

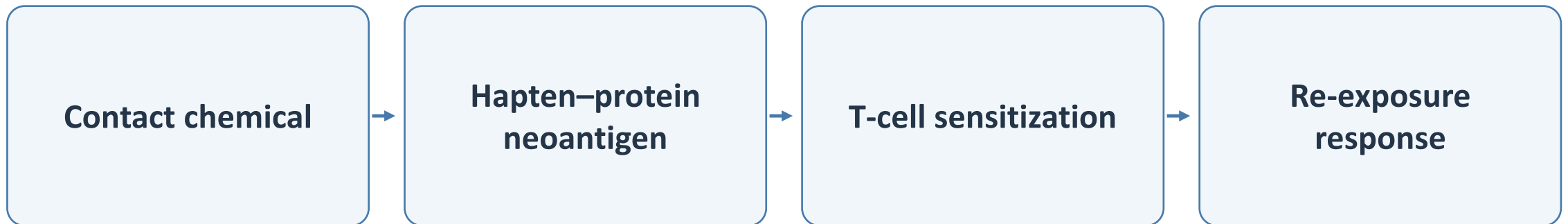
- “Outside”: Contact with a locally applied antigen, such as poison ivy
- “Inside”: A circulating antigen associated with an ingested agent
- This exposure distinction directs the clinical history; It does not determine histology by itself



Schematic

Allergic contact dermatitis: Acquired reactivity

- A contact chemical can modify self proteins, producing a new antigenic target
- Sensitization generates effector and memory T cells
- Re-exposure elicits localized delayed inflammatory injury



Schematic

Primary irritant dermatitis is a distinct initiating category

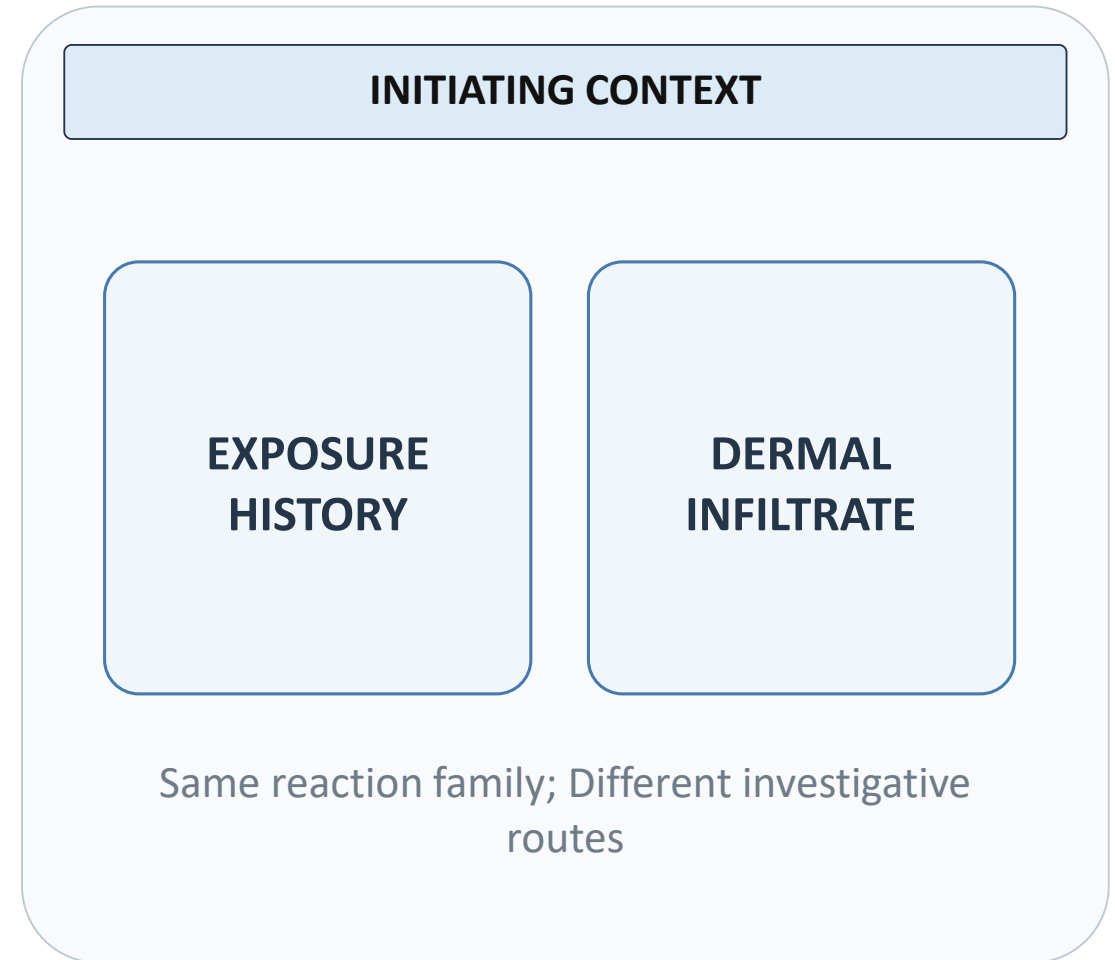
- Primary irritant dermatitis separately from allergic contact disease
- An irritant history is not proof of hapten-specific memory T-cell sensitization
- Interpret a spongiotic biopsy with the exposure pattern, not the label alone

Atopic dermatitis enters the same morphologic spectrum

- Atopic dermatitis is one of the classified eczematous disorders
- Spongiosis and chronic epidermal reaction can occur across eczematous subtypes
- Clinical context is necessary to separate atopic from contact-related eczema

Some ingested drugs produce an eczematous eruption

- The provoking agent may be circulating rather than applied directly
- Biopsies from certain drug reactions may contain eosinophils
- Drug exposure and chronology matter more than one histologic clue



Schematic

Photoeczematous dermatitis adds a light-related context

- Photoeczematous dermatitis is a recognized initiating category
- Ultraviolet exposure can affect antigen properties and epidermal immune cells
- Assess the distribution and exposure history rather than assuming one mechanism

A shared pattern does not imply identical pathogenesis

Identifiable

Epidermal intercellular edema

Papulovesicular eczema pattern

Not established by morphology alone

Exact environmental or systemic trigger

Specific antigen or sensitization status

Read as a diagnostic comparison, not as an exclusive criterion.

Poison ivy demonstrates florid contact eczema

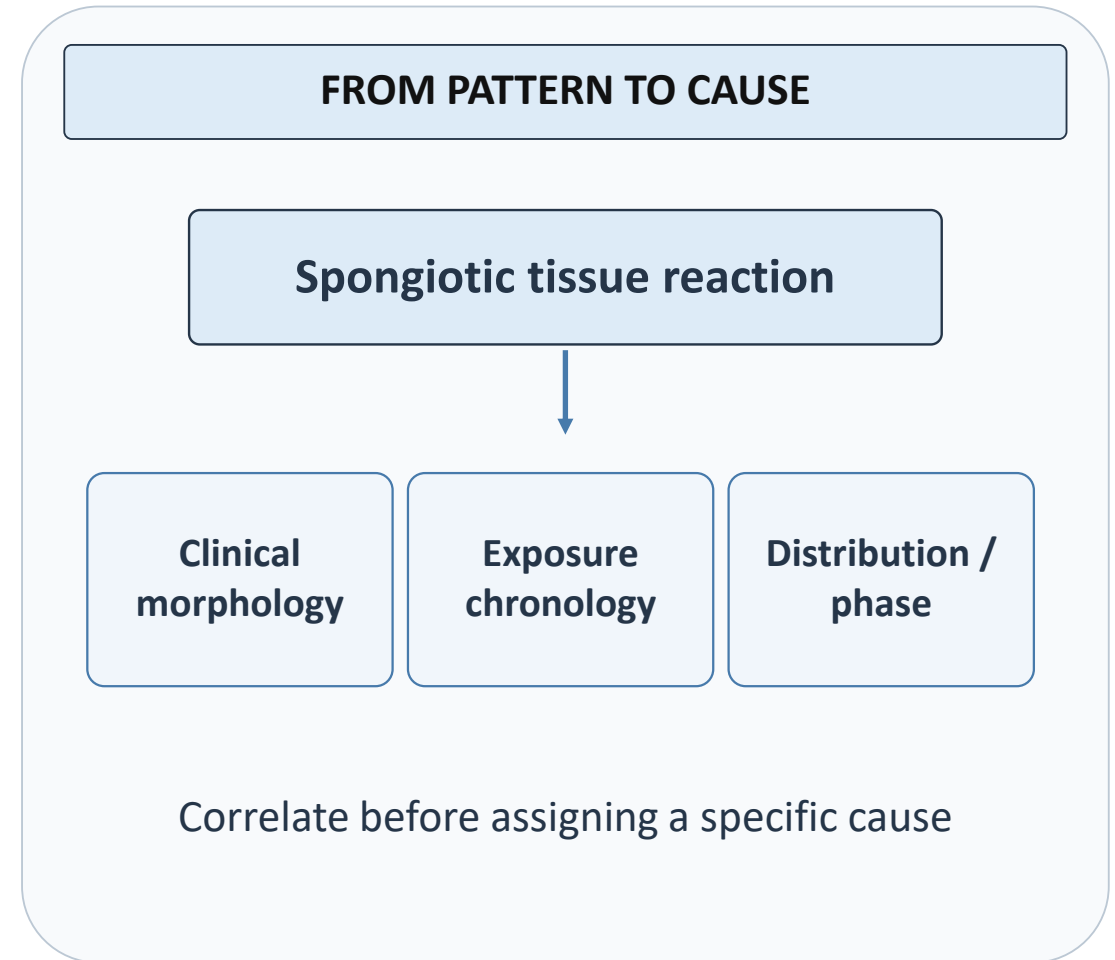
- Urushiol from poison ivy/oak provides a classic contact antigen
- Pruritic, edematous, oozing plaques may form after contact
- Both vesicles and larger bullae can occur in a pronounced reaction

Patterned distribution can reveal contact exposure

- Nickel-associated dermatitis related to a necklace
- The eruption follows a site of contact instead of requiring generalized disease
- Distribution is a diagnostic clue and should be interpreted with timing and history

Exposure site helps contextualize histology

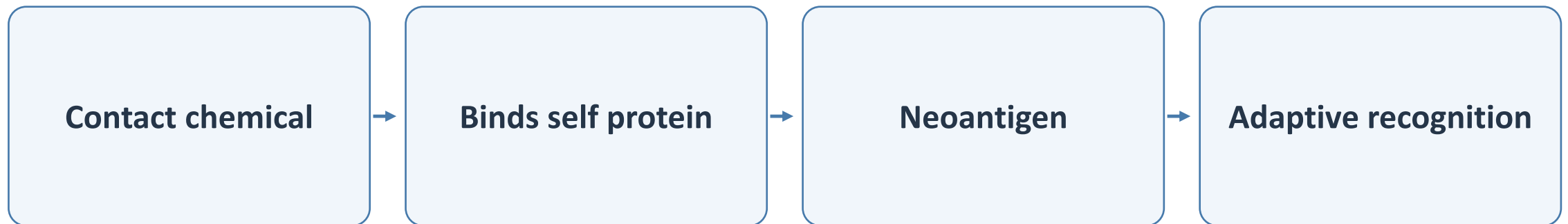
- Examine where the rash begins and whether it matches a plausible contact area
- Distinguish direct exposure from ingestion and other initiating contexts
- Combine site, time course and morphology to refine the differential



Schematic

Contact chemicals can behave as haptens

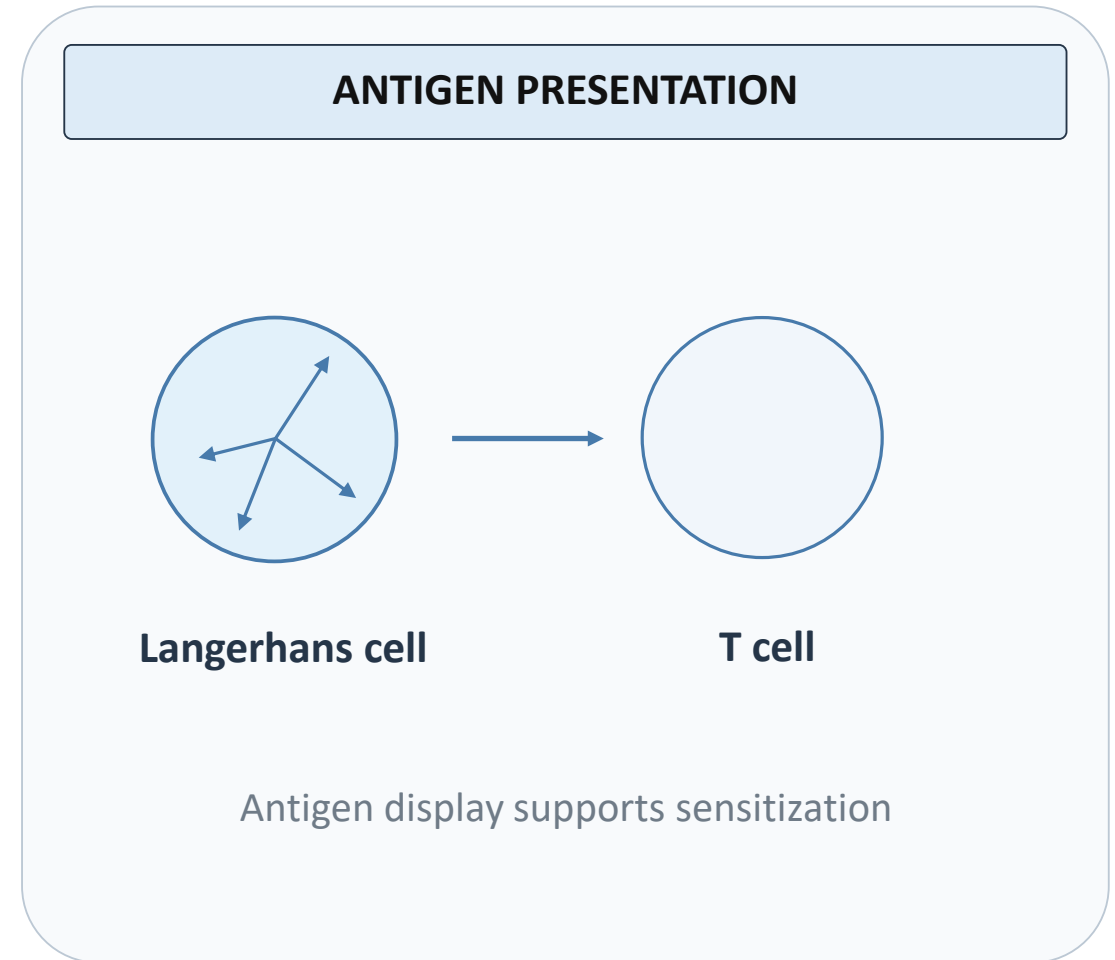
- Small chemicals may react with endogenous skin proteins
- The resulting altered proteins can function as neoantigens
- This explains how a local exposure can become immunologically recognizable



Schematic

Antigen capture occurs within the epidermis

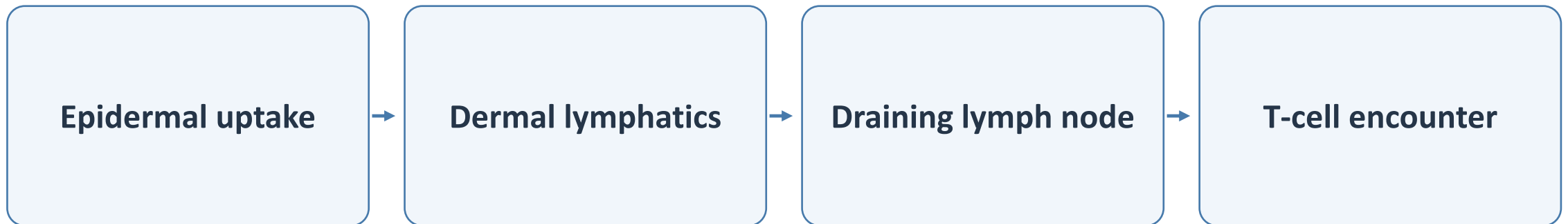
- Langerhans cells reside in the epidermis where exposure occurs
- They internalize and process contact-associated neoantigens
- Cutaneous antigen capture initiates a regional immune response



Schematic

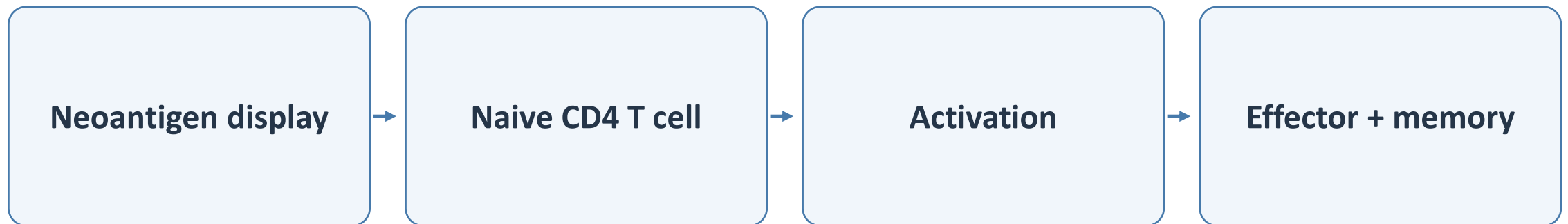
Migration carries antigen to the lymph node

- Antigen-bearing Langerhans cells enter dermal lymphatics
- They reach draining regional lymph nodes
- An encounter outside the epidermis is needed to establish T-cell sensitization



Naive CD4 T cells are activated by presentation

- Antigen presentation stimulates naive CD4 T lymphocytes
- Activated cells differentiate into effector and memory populations
- This creates capacity for a response on later exposure



Schematic

Immunologic memory explains re-exposure responses

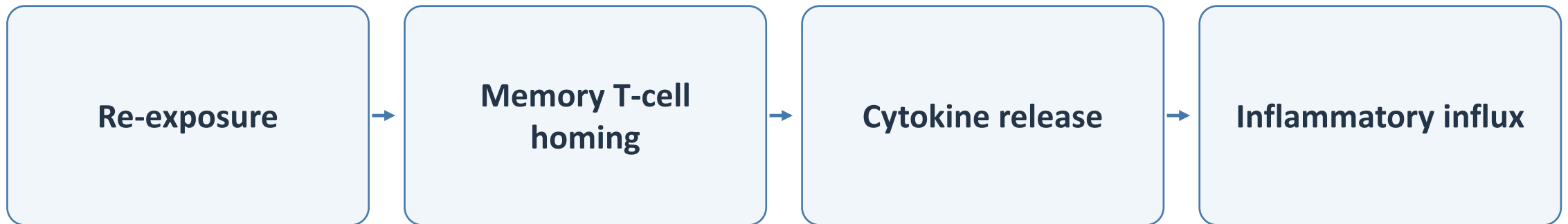
- After initial sensitization, memory cells persist
- Re-exposure engages a prepared cutaneous immune system
- The episode can therefore be amplified relative to first contact



Schematic

Antigen re-exposure recruits memory T cells

- Memory T cells recognizing antigen localize to involved skin
- Skin-homing surface molecules help direct recruitment
- Inflammatory mediators from these cells recruit other leukocytes

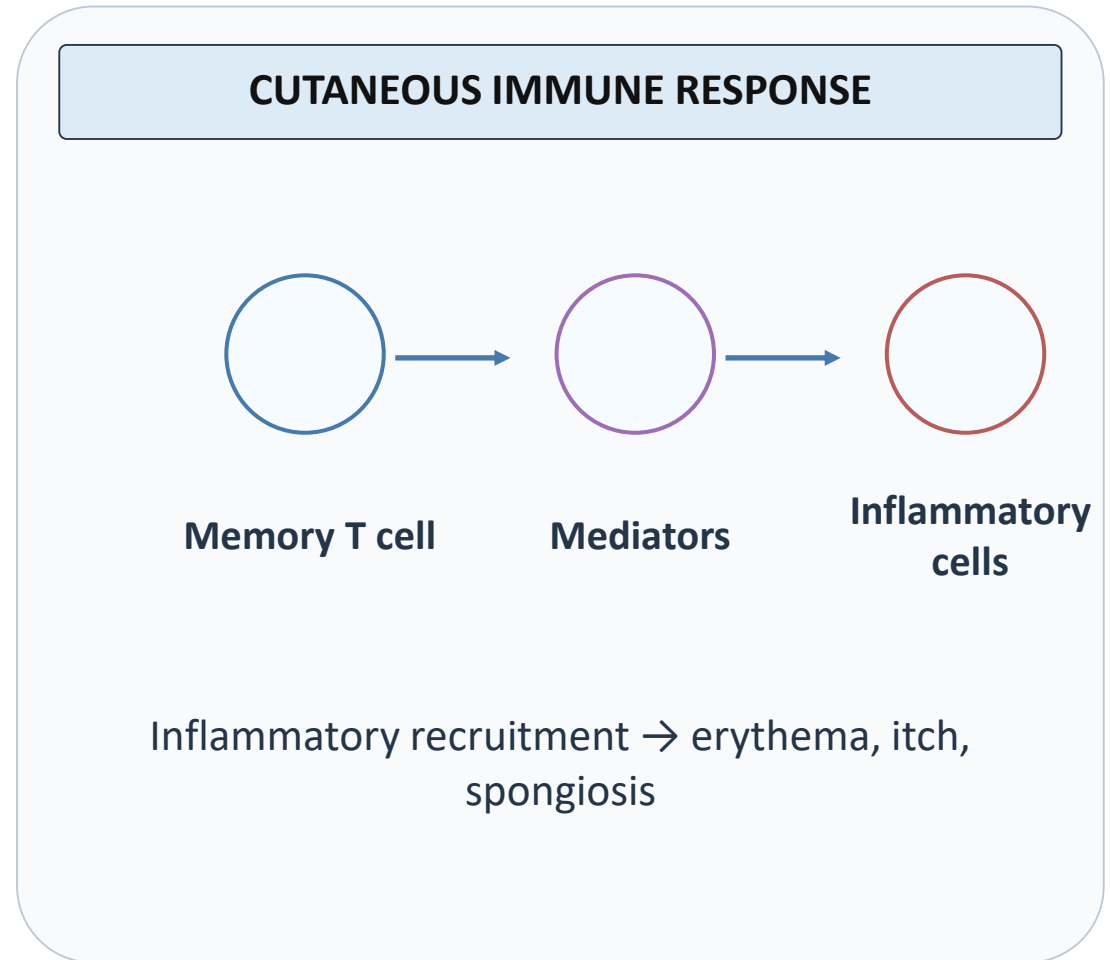


CLA and chemokine receptors guide tissue localization

- Cutaneous lymphocyte-associated antigen marks a skin-homing T-cell phenotype
- Robbins also cites chemokine receptors CCR4 and CCR10 in the skin immune system
- Homing signals provide spatial specificity to the inflammatory response

Cytokines translate recognition into tissue injury

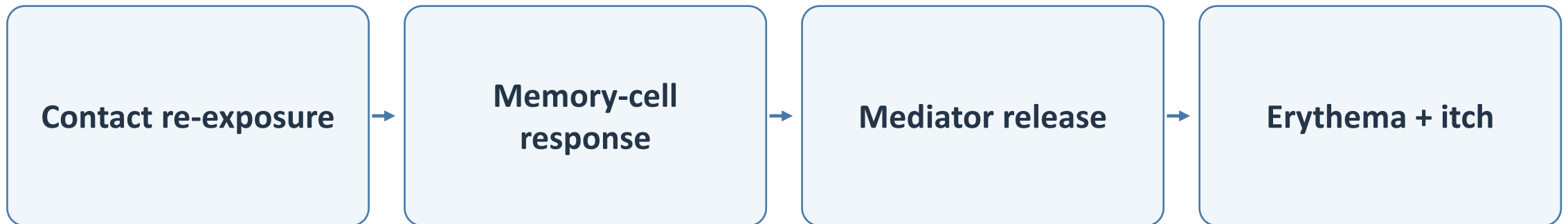
- Recruited and resident T cells release cytokines and chemokines
- These mediators attract additional inflammatory cells
- Dermal inflammation and epidermal edema produce the visible eruption



Schematic

Erythema and pruritus can develop within a day

- The re-exposure response developing within approximately 24 hours
- Erythema and itch mark the acute spongiotic phase
- The exact clinical timeline must still be assessed in each patient



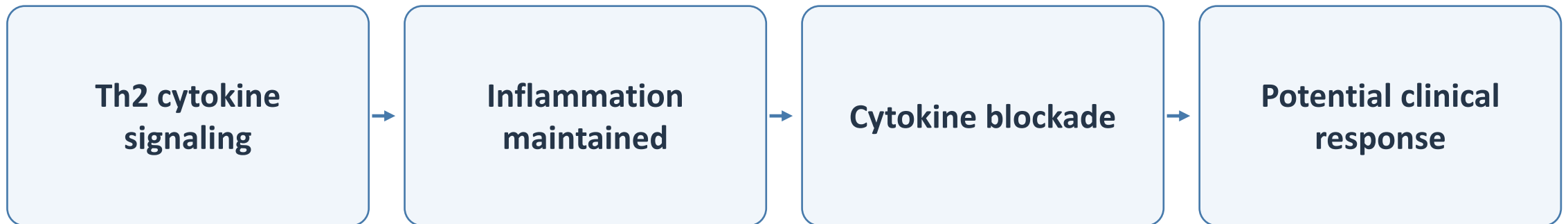
Ultraviolet light has opposing immune effects

UV effect on cells	UV effect on antigen
Langerhans-cell injury	Altered antigen structure
Reduced sensitization capacity	Potentially increased sensitivity

Read as a diagnostic comparison, not as an exclusive criterion.

IL-4 and IL-13 provide therapeutic context

- Severe eczema to Th2-derived cytokines IL-4 and IL-13
- Biologic agents blocking cytokine action can benefit severe disease
- This treatment observation is not a universal diagnostic test for eczema subtype

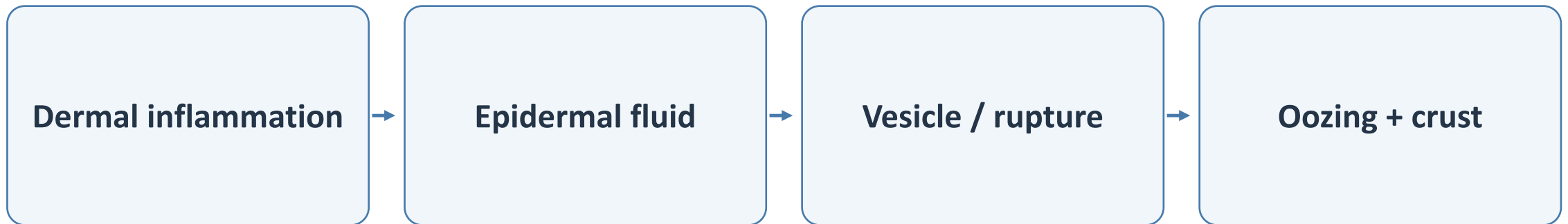


The visible acute lesion is papulovesicular

- An erythematous background accompanies small raised papules and vesicles
- Lesions are often pruritic and may develop exudate
- Crusting follows when exudate dries at the surface

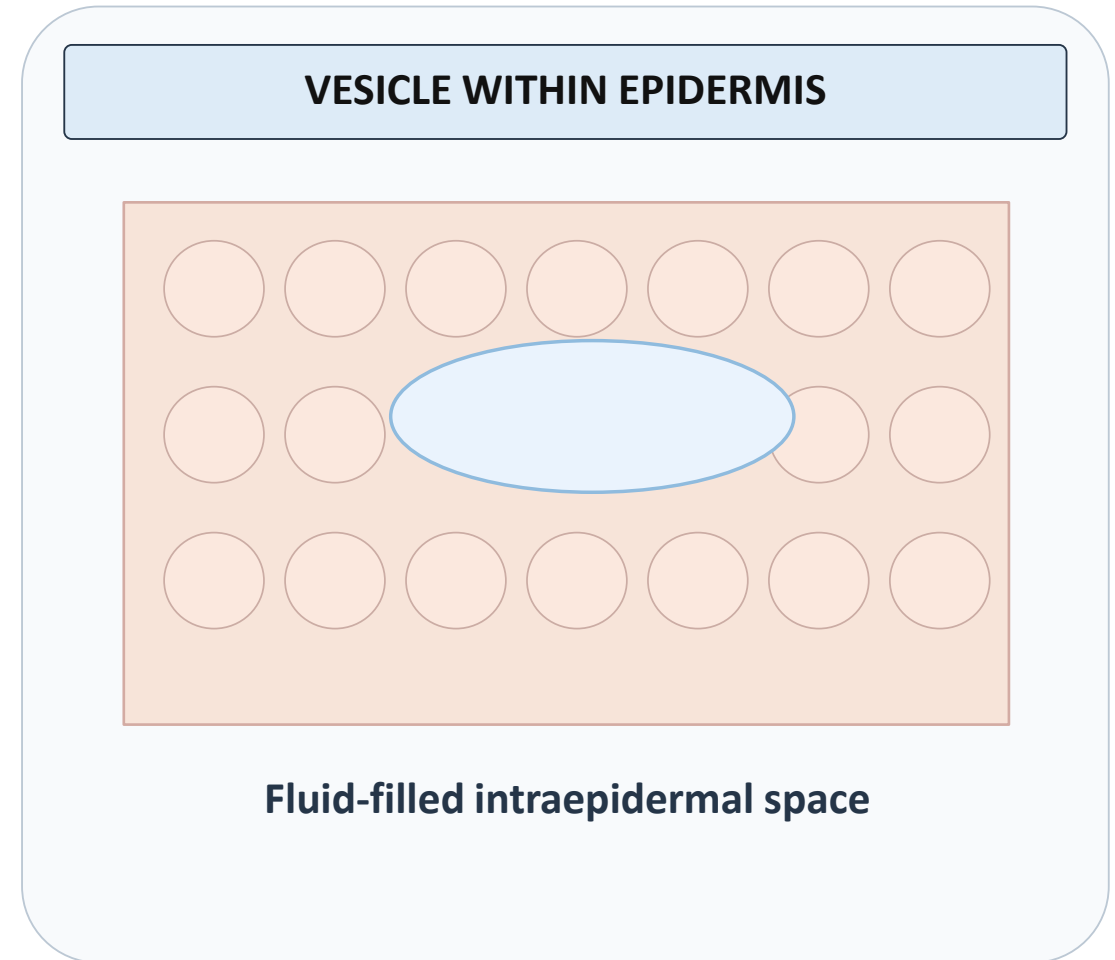
Pruritus, edema and oozing co-occur

- Inflammation drives erythema and itch
- Fluid accumulation expands the epidermal intercellular spaces
- Surface rupture of vesicles produces an oozing clinical appearance



Vesicles and bullae express the same process

- Fluid accumulation can coalesce into intraepidermal cavities
- Small spaces become microvesicles, with larger blisters in florid eruptions
- The size of a blister does not identify the initiating antigen



Schematic

Crust can reflect dried exudate or infection

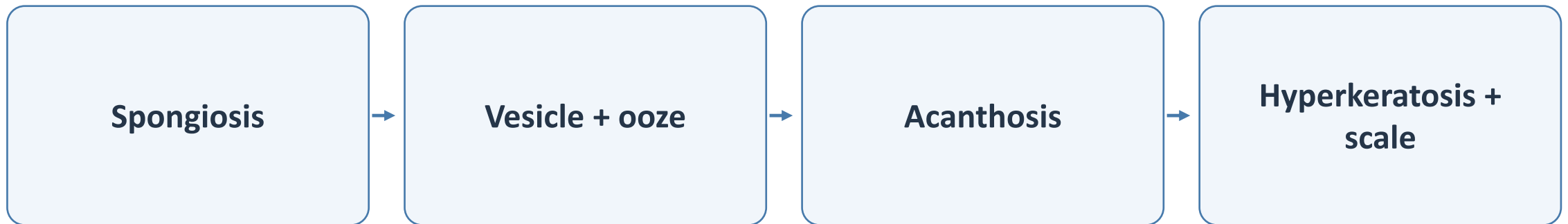
- Acute eczema often has crusted papulovesicles
- Secondary bacterial infection can produce yellow crust—impetiginization
- A yellow crust requires clinical assessment rather than automatic attribution

Excoriated, oozing skin is vulnerable to infection

- Eczematous lesions can become secondarily infected
- Superinfection changes the surface appearance and clinical priorities
- Distinguish primary inflammation from a superimposed infectious component

Long-standing eczema becomes less wet

- Persistent lesions gradually lose prominent vesiculation and oozing
- The surface becomes increasingly scaly and thickened
- Morphologic chronicity may obscure the original acute mechanism

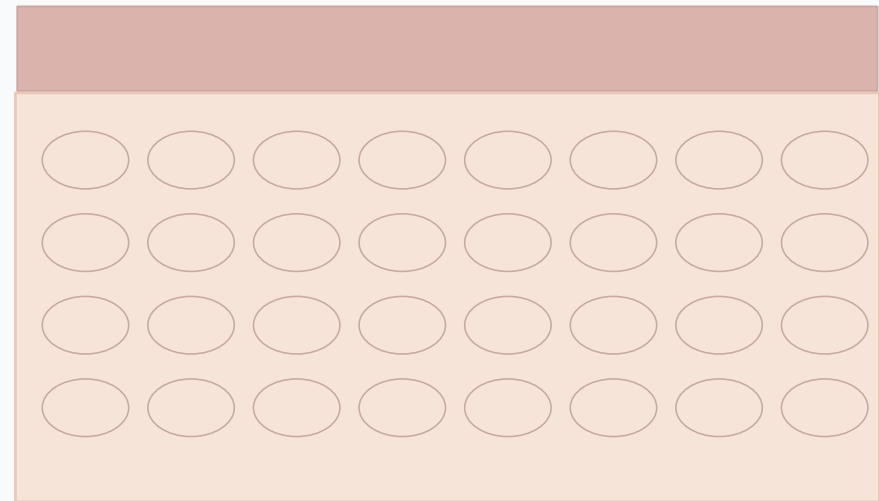


Acanthosis is a reactive epidermal response

- Acanthosis refers to increased thickness of the viable epidermis
- It as lesions persist
- It explains the raised plaque component of long-standing eczema

PERSISTENT EPIDERMAL RESPONSE

Thickened stratum corneum



Acanthosis: Increased viable epidermal thickness

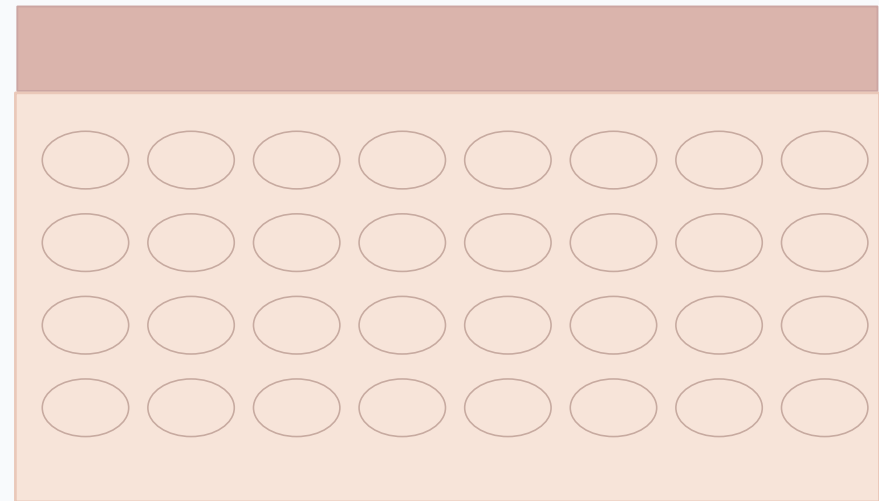
Schematic

Hyperkeratosis produces a drier, scaly surface

- Hyperkeratosis means excess stratum corneum thickness
- With chronicity, lesions become progressively hyperkeratotic
- A thick scaly plaque can represent previously vesicular eczema

PERSISTENT EPIDERMAL RESPONSE

Thickened stratum corneum



Acanthosis: Increased viable epidermal thickness

Schematic

Pigmentation can alter clinical recognition

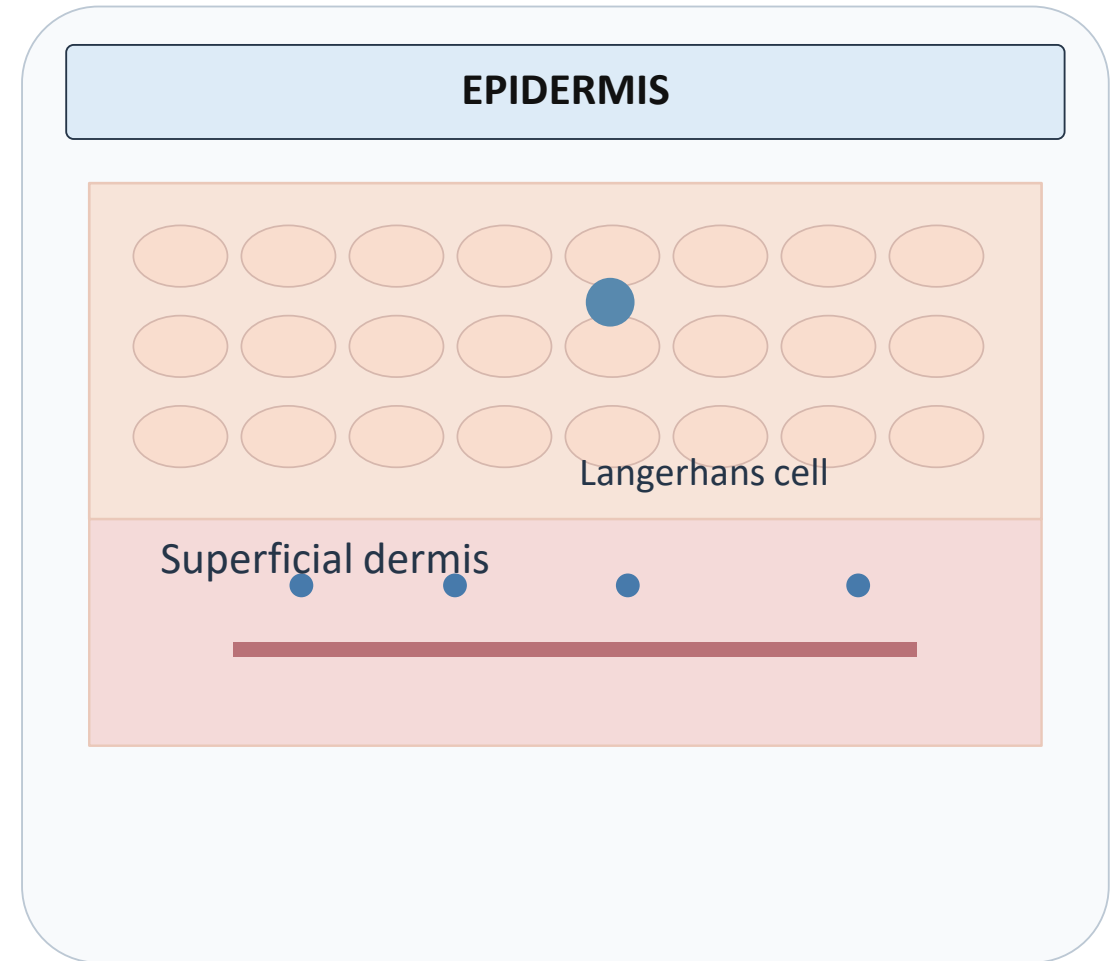
- Eczematous dermatitis with hyperpigmentation in darker skin
- The appearance of erythema varies with skin pigmentation
- Interpret the eruption through morphology, pruritus and exposure history, not redness alone

Postinflammatory hypopigmentation may occur

- The textbook also notes hypopigmentation after inflammation in darker skin
- Pigment change can persist beyond the most active inflammatory lesion
- Do not equate a pigmentary change with active vesiculation

The histologic sequence begins in the dermis

- Earliest lesions feature superficial perivascular lymphocytes
- Papillary dermal edema and mast-cell degranulation accompany inflammation
- Epidermal spongiosis then becomes conspicuous



Schematic

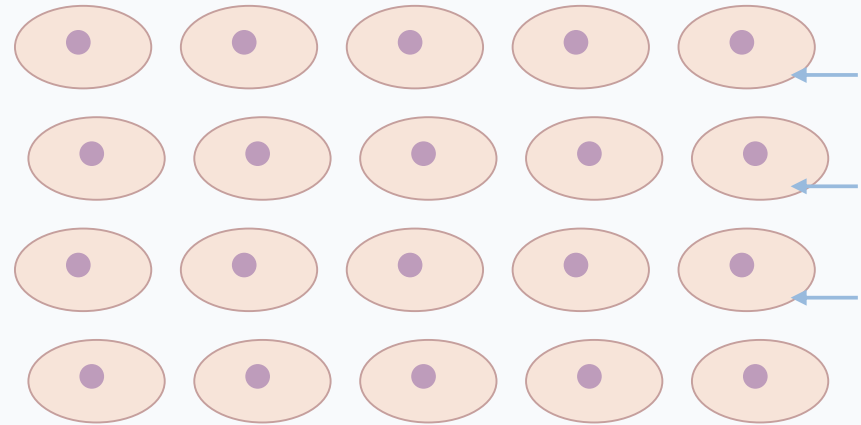
Papillary dermal edema precedes overt vesicles

- Early inflammatory fluid increases in superficial dermal tissue
- Dermal vascular and immune events occur before or with epidermal changes
- A very early biopsy may show less developed epidermal spongiosis

Spongiosis is intercellular—not intracellular—edema

- Fluid collects between epidermal keratinocytes
- Intercellular spaces expand while neighboring cells remain linked at points
- The characteristic pattern is most apparent in the stratum spinosum

EPIDERMAL INTERCELLULAR EDEMA

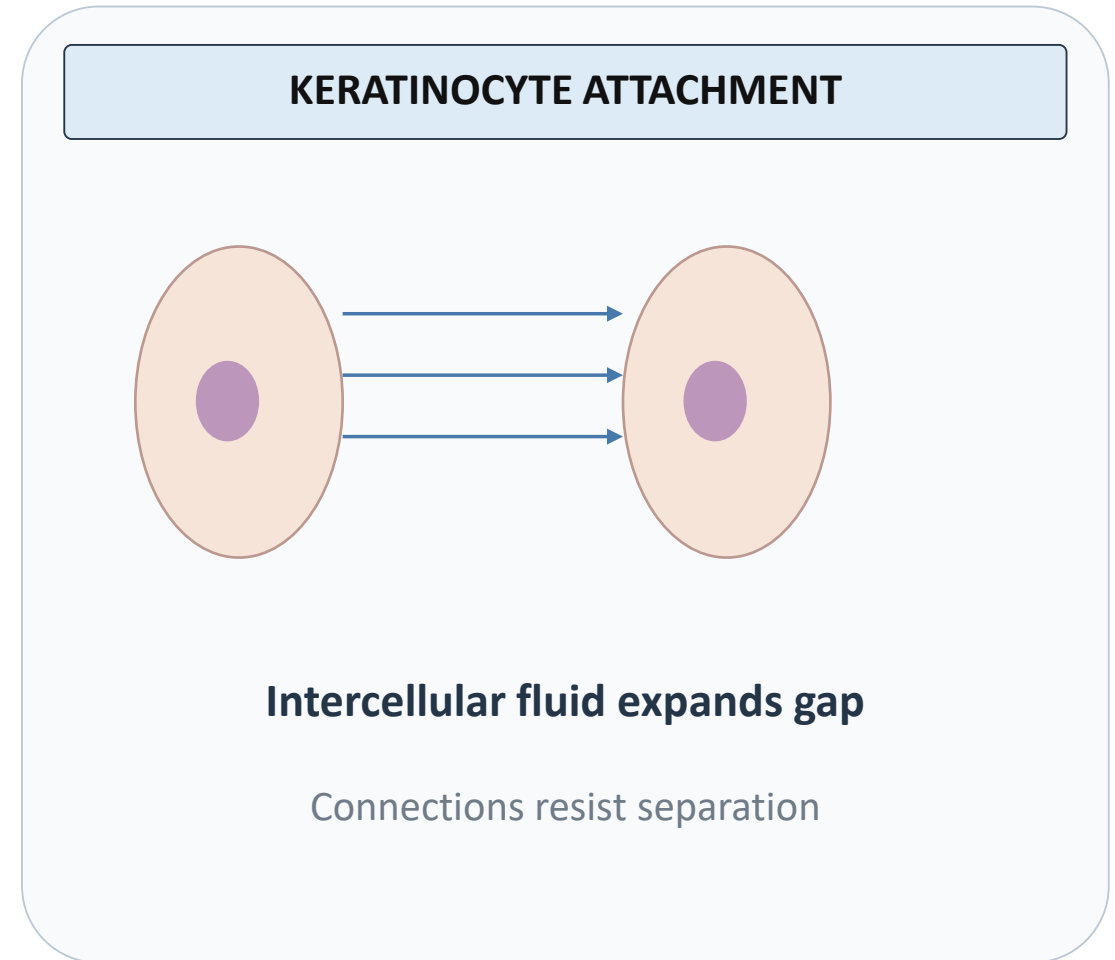


Fluid widens spaces

Schematic

Desmosomes are stressed as the spaces enlarge

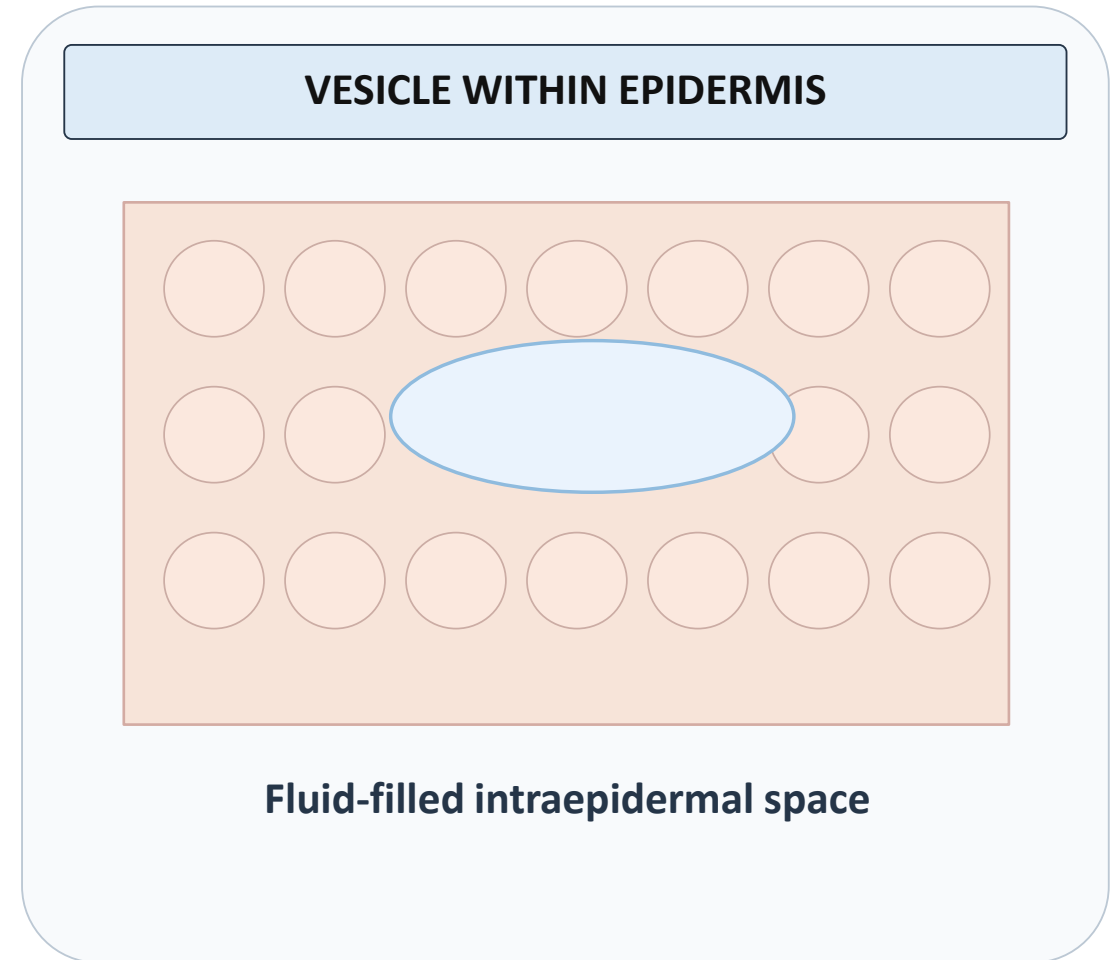
- Progressive fluid accumulation splays apart keratinocytes
- Connections between cells may appear stretched or disrupted
- Mechanical separation helps generate small epidermal cavities



Schematic

Intraepidermal microvesicles result from coalescence

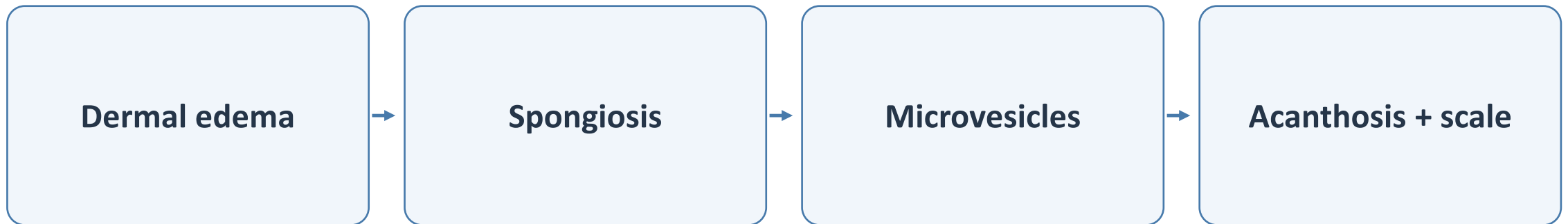
- Edema-induced epidermal spaces can merge
- Focal detachment or shearing forms microvesicles within the epidermis
- These microscopic cavities underlie clinical vesiculation



Schematic

Histology changes along a continuous timeline

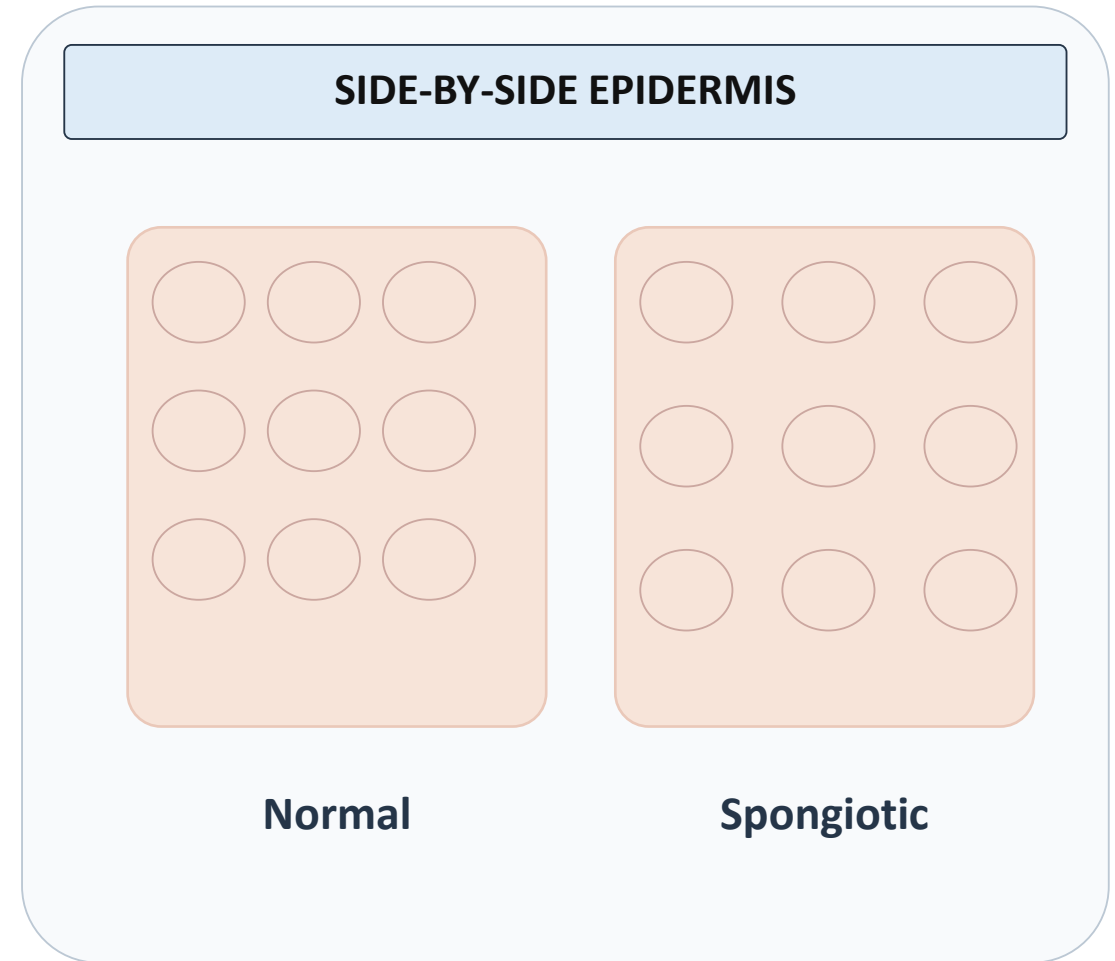
- First: Superficial dermal edema and perivascular cells
- Next (within 24–48 hours): Epidermal spongiosis and microvesicles
- Later: Abnormal scale, acanthosis and hyperkeratosis



Schematic

Normal versus spongiotic epidermis

- Normal: Densely apposed keratinocytes with narrow intercellular spaces
- Spongiotic: Expanded spaces and residual intercellular links
- Severe spongiosis can progress beyond separation to true vesicles



Schematic

Certain drug eruptions suggest a deeper infiltrate

Histologic clue	Clinicopathologic question
Eosinophils with perivascular cells	Recent or ongoing drug exposure?
Superficial + deep involvement	Does chronology support the agent?

Read as a diagnostic comparison, not as an exclusive criterion.

Contact eczema often favors superficial mononuclear cells

- Contact-related eczema typically shows superficial mononuclear inflammation
- This pattern with eosinophil-rich drug-associated reactions
- Infiltrate distribution helps prioritize—but does not finalize—the etiology

Inflammatory cells provide clues, not certainty

Histology can support

Spongiosis and phase

Infiltrate depth / composition

Degree of epidermal change

Histology alone cannot prove

Exact contact allergen

Drug causality

A unique eczema subtype

Read as a diagnostic comparison, not as an exclusive criterion.

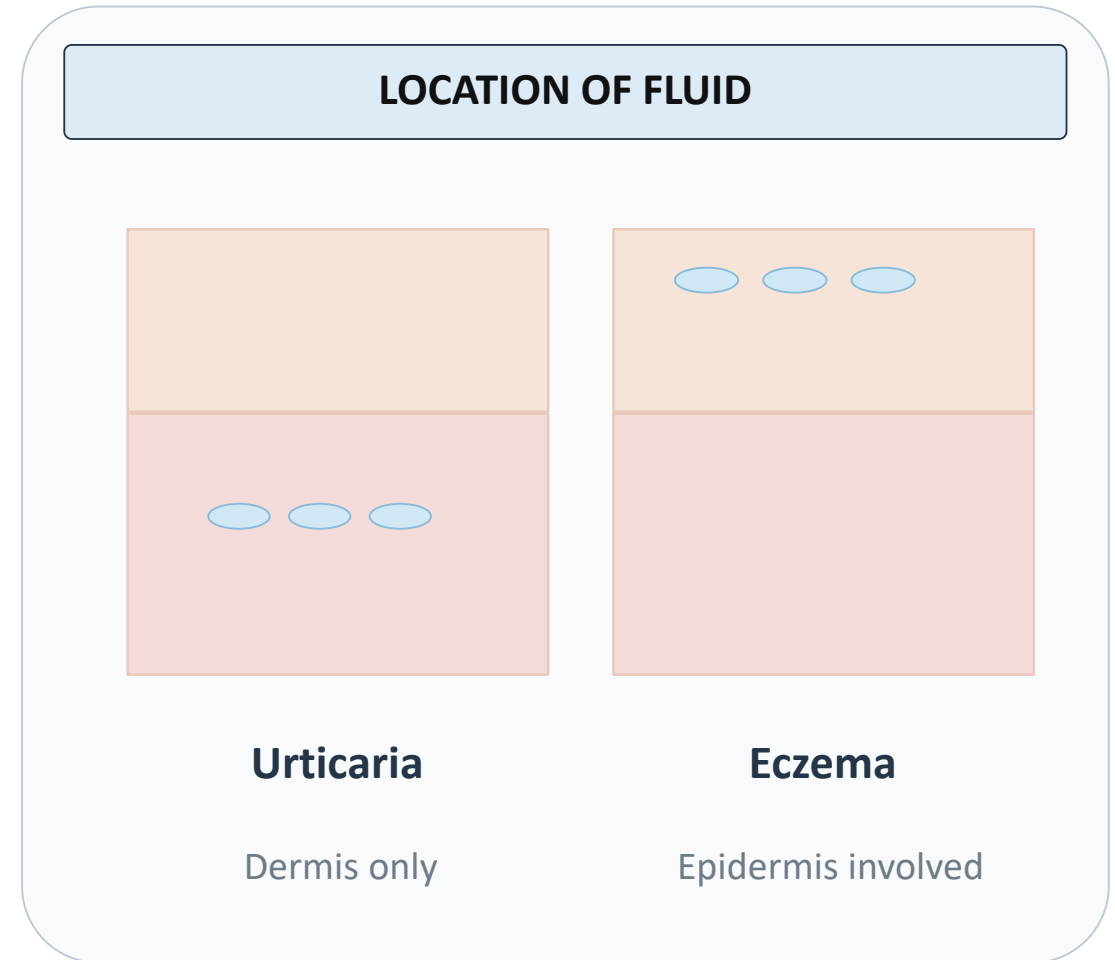
Urticaria favors dermal edema without spongiosis

Urticaria	Eczematous dermatitis
Dermal edema / wheals	Epidermal spongiosis
Epidermis generally normal	Papulovesicles and crusting
Mast-cell hyperpermeability	Often T-cell–linked inflammation

Read as a diagnostic comparison, not as an exclusive criterion.

The distribution of edema separates two patterns

- In urticaria, edema is concentrated in superficial dermis
- In eczema, fluid also extends into the epidermis
- This anatomical distinction explains wheals versus vesicles



Schematic

Interface injury is not spongiotic dermatitis

Eczematous pattern

Spongiosis ± microvesicles

Intercellular epidermal fluid

Eczematous eruption

Erythema multiforme pattern

Interface dermatitis

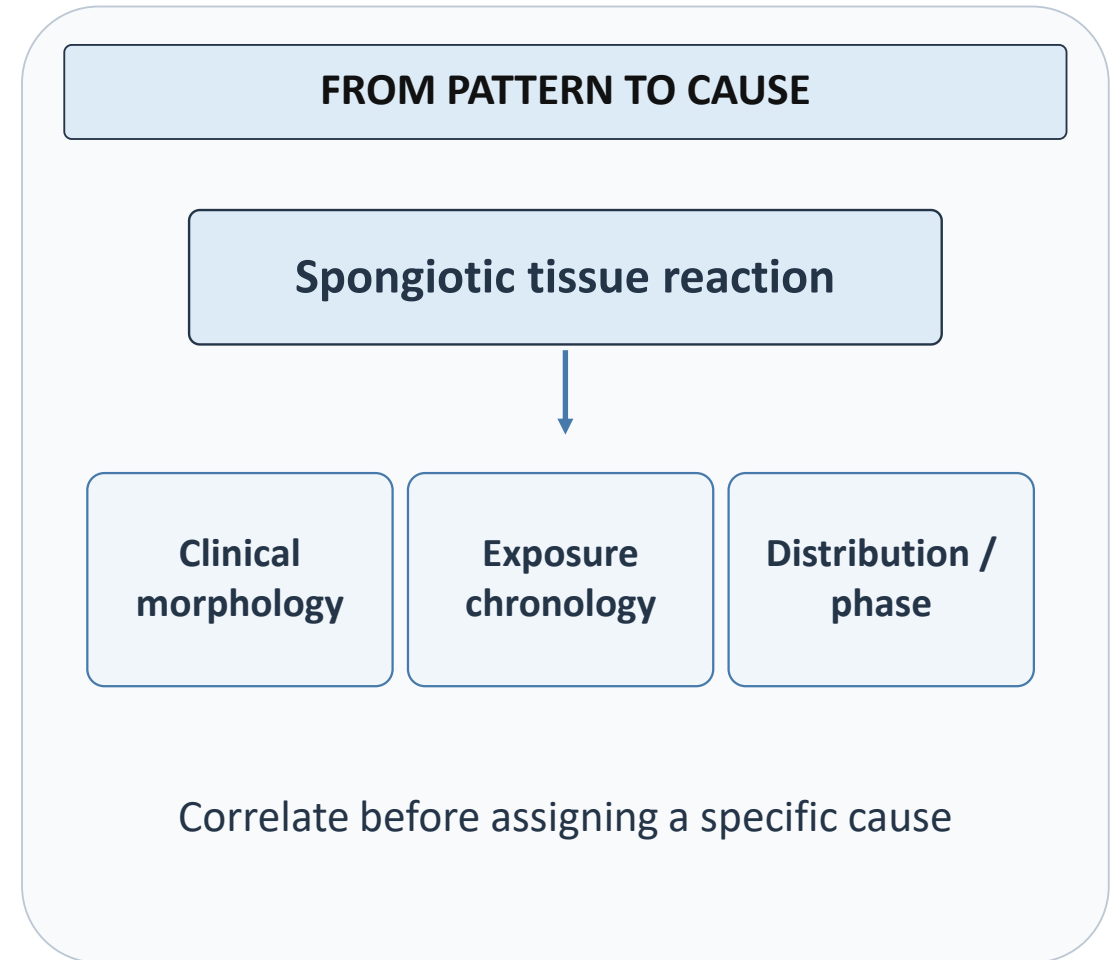
Junctional keratinocyte injury

Often targetoid lesions

Read as a diagnostic comparison, not as an exclusive criterion.

A diagnosis begins with the eruption's morphology

- Are there papulovesicles, surface exudate, crusts or plaques?
- Is the eruption acute, recurrent or persistent?
- Is there a contact-pattern distribution or possible systemic exposure?



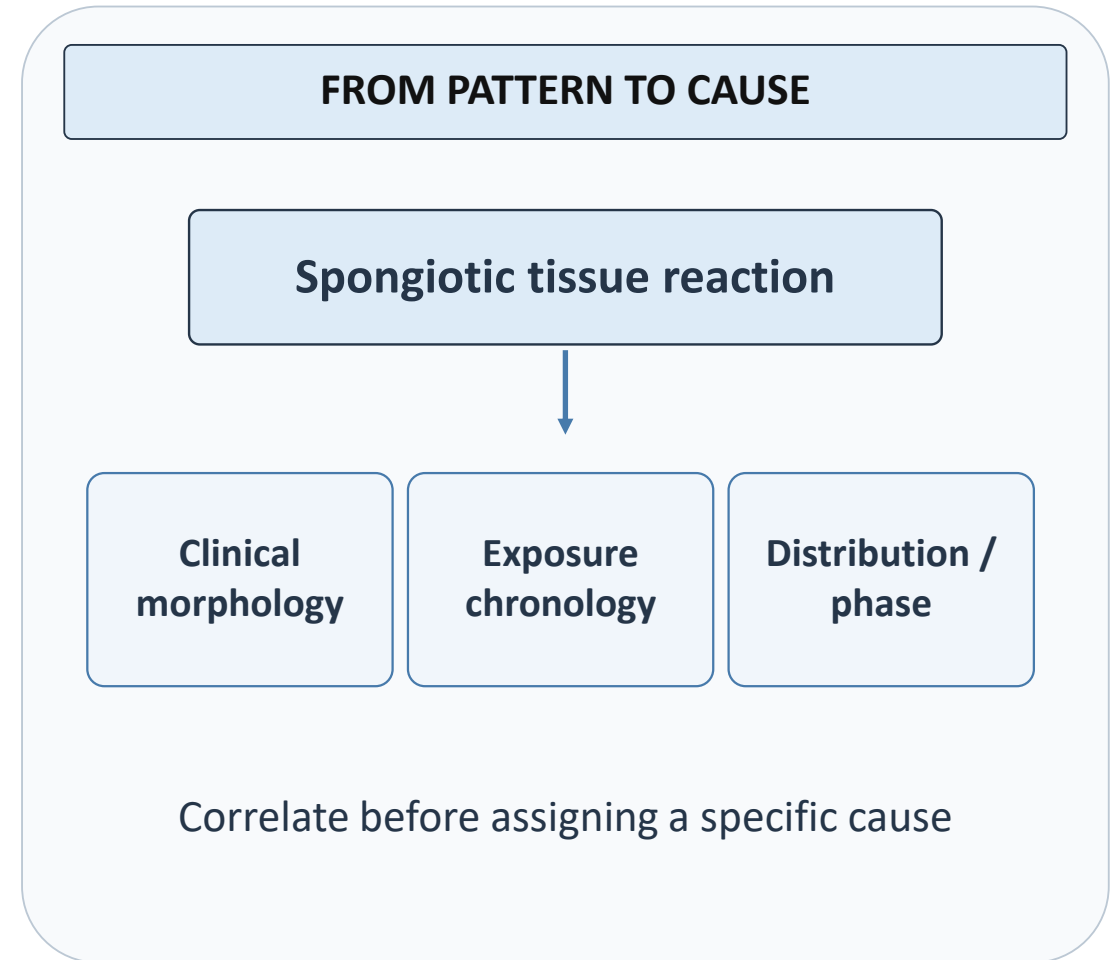
Schematic

Clinical history is essential for the etiologic label

- Ask about locally applied substances and contact patterns
- Review ingested medications or other circulating exposures
- Assess relation to light exposure and the clinical setting of atopy

The biopsy establishes pattern and activity

- Document epidermal spongiosis, vesiculation and reactive thickening
- Assess papillary dermal edema and the depth of inflammatory infiltrate
- Interpret stage-dependent findings with the eruption and exposure history



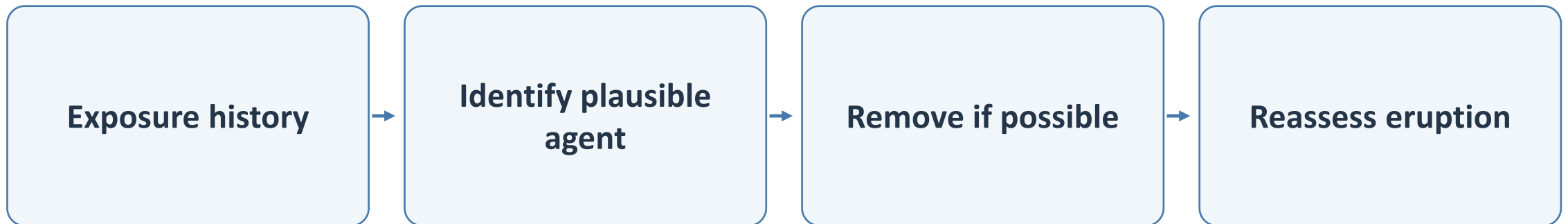
Schematic

Clinicopathologic correlation prevents overcalling

- “Spongiotic dermatitis” is a useful histologic description
- The differential remains broad across the listed eczema contexts
- A specific causal diagnosis needs corroborating clinical information

Avoidance addresses the initiating factor

- Treatment starts by identifying potentially removable offending substances
- External or ingested exposures should be examined when supported by history
- Avoidance may prevent additional immune stimulation



Topical corticosteroids interrupt inflammation

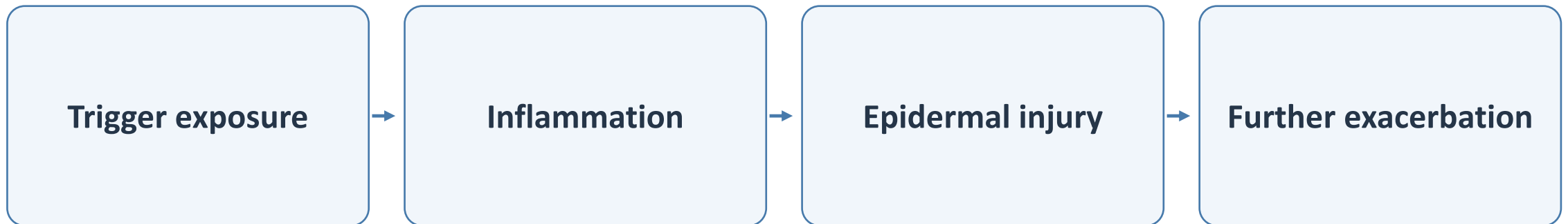
- Mild-to-moderate eczema is commonly treated with topical anti-inflammatory agents
- Corticosteroids blunt the inflammatory response during flares
- Their benefit as palliative rather than causal eradication

Severe disease may respond to cytokine blockade

- Biologic approaches that inhibit cytokine action in severe eczema
- IL-4 and IL-13 are identified as relevant Th2-derived mediators
- Specific treatment selection is outside a morphology-only diagnosis

Uncontrolled flares can sustain more inflammation

- Continued inciting exposure can drive a recurrent response
- Anti-inflammatory treatment interrupts an acute exacerbation
- Combine suppression of the eruption with efforts to identify its cause



Case: Pruritic, oozing plaques after plant exposure

- A patient develops intensely pruritic, edematous plaques after outdoor plant contact
- Some lesions exhibit vesicles and serous exudate
- Which exposure mechanism and epidermal feature are most likely?

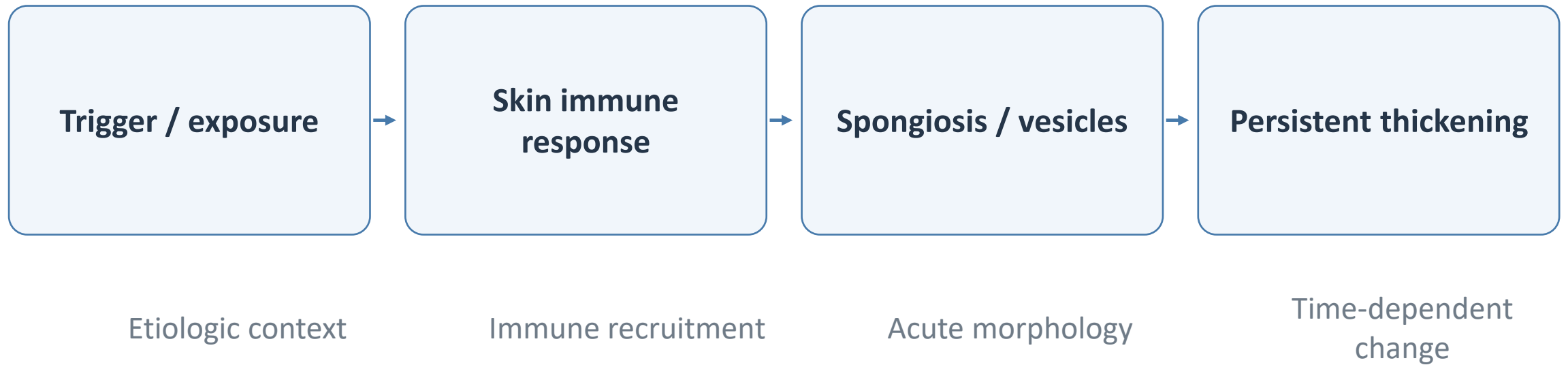
Case: A rash follows a newly ingested medication

- An eczematous eruption occurs after exposure to an ingested medication
- Biopsy: Spongiosis and perivascular inflammatory cells including eosinophils
- What does this finding contribute—and what does it not prove?

Case: Transient wheals without epidermal change

- A patient has pruritic edematous wheals
- Biopsy shows superficial dermal edema but a normal epidermis
- Which mechanism is favored over eczematous dermatitis?

Synthesis: Exposure, immunity and tissue reaction



Primary reference and scope

- Robbins, Cotran & Kumar: Pathologic Basis of Disease, 11th ed. (2026)
- Chapter 25, The Skin: pp. 1028–1029 and 1047–1050
- Mechanism contextualized from the textbook's Chapter 6 discussion of T-cell–mediated reactions
- Original published micrographs were not embedded; morphological visuals here are editable schematics