

Other Types of Panniculitis

Patterns, causes, and pathological mimics

A clinicopathologic approach to deep subcutaneous inflammation

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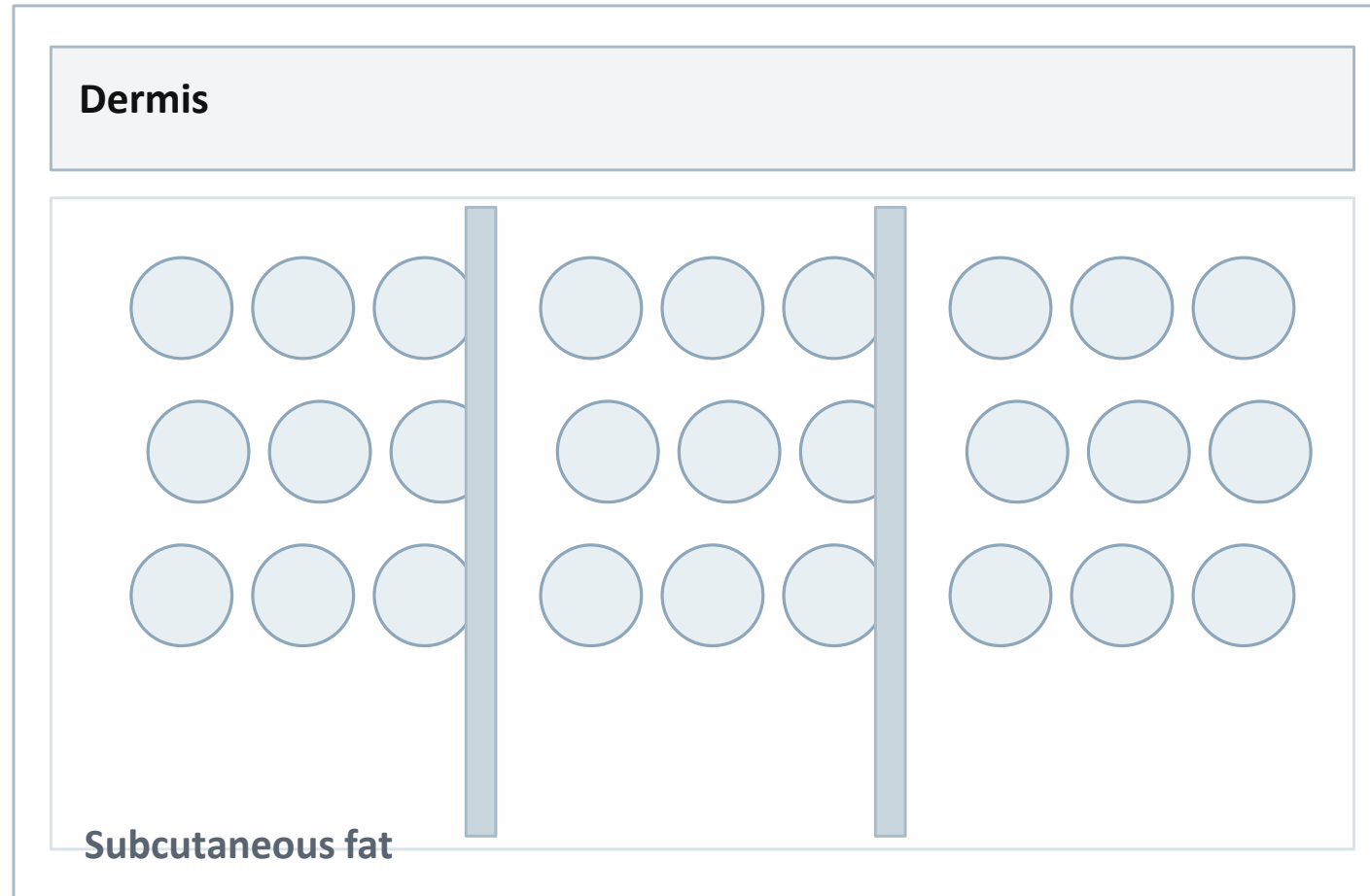
Learning outcomes

- Localize panniculitis to fat septa or fat lobules.
- Distinguish nonvasculitic from vasculitic inflammation.
- Recognize four less-common etiologic settings.
- Explain why lymphoma can imitate inflammatory panniculitis.

What this lecture includes

- Four explicitly named entities: Weber–Christian disease, factitial panniculitis, T-cell lymphoma mimics, and lupus-associated panniculitis.
- Necessary anatomical and clinical comparisons with erythema nodosum and erythema induratum.
- Interpretative questions derived from the textbook descriptions—not additional unverified diagnostic criteria.

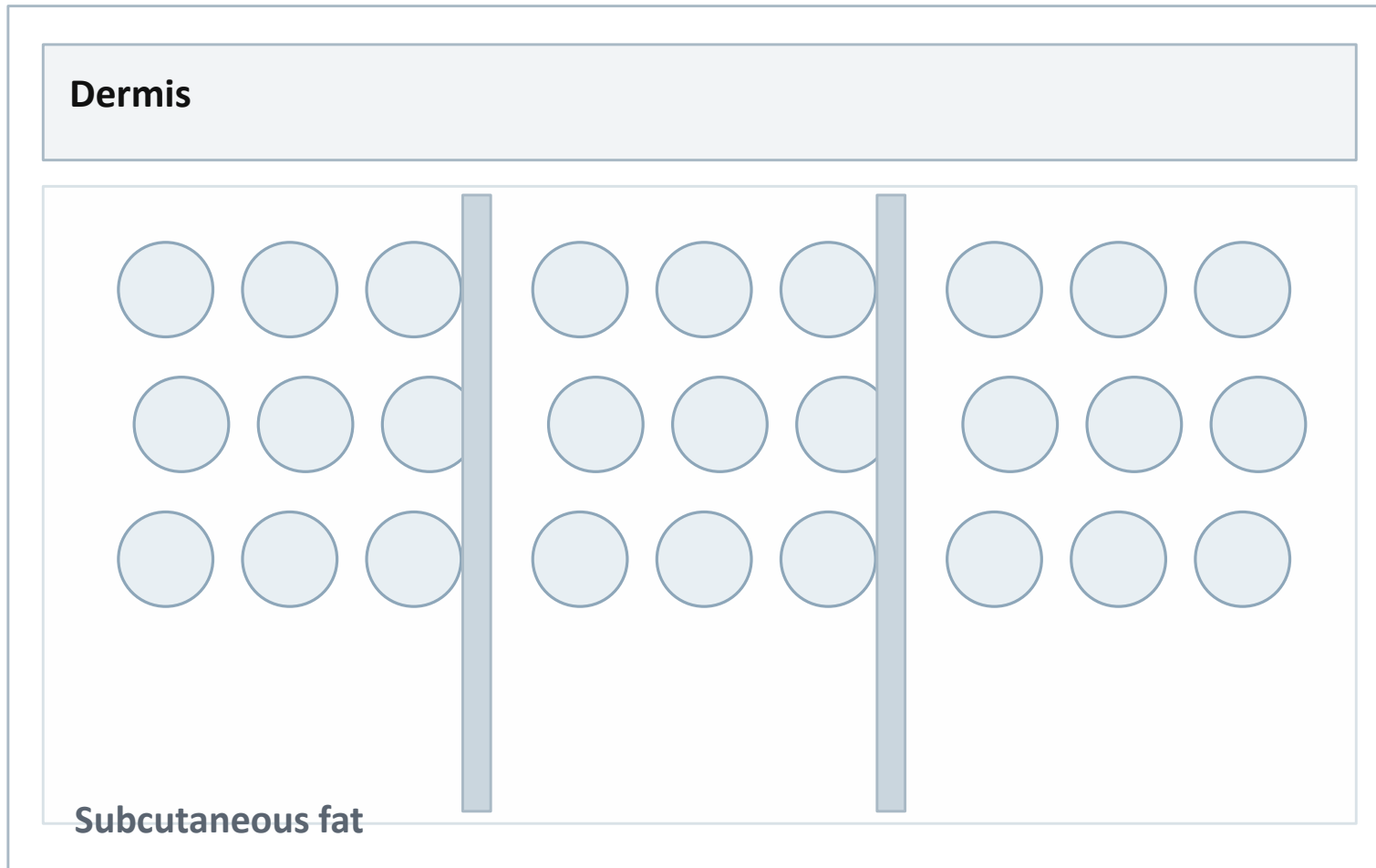
Panniculitis involves the subcutaneous adipose tissue



Epidermal or superficial dermal abnormalities do not define the central site of panniculitis.

SCHEMATIC

Two major anatomical patterns: Septal and lobular



Septal

Connective tissue between
adipose lobules

Lobular

Fat lobules themselves

A pattern is not yet an etiologic diagnosis

- The anatomical pattern describes where inflammation is concentrated.
- Vasculitis indicates inflammatory injury to blood vessels and is a separate observation.
- Clinical context and possible underlying processes must still be considered.
- A malignancy may produce a similar inflammatory appearance.

Four questions for a panniculitis specimen

Compartment

Is inflammation
mainly septal or
lobular?

Vessels

Is a vasculitic
process described
or absent?

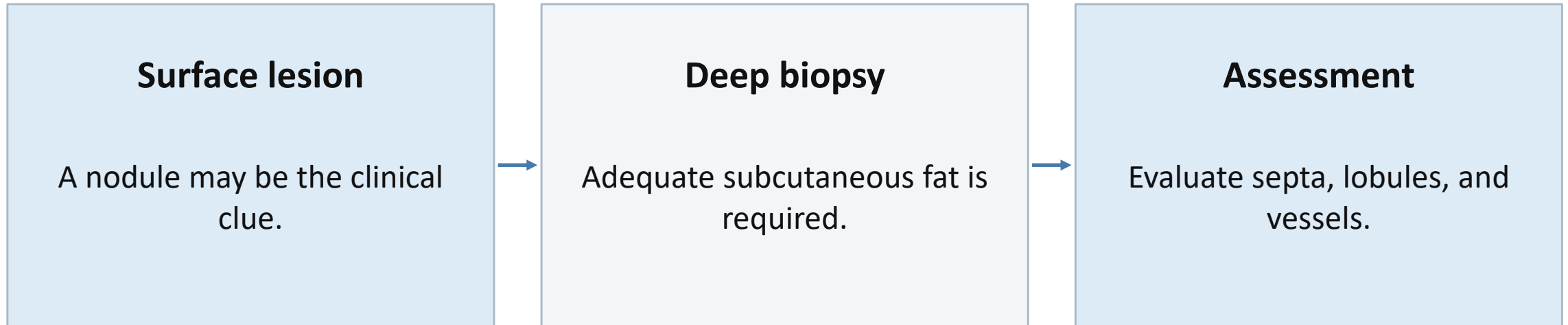
Injury

Are necrosis and
other tissue effects
identified?

Context

Is a trigger, systemic
disease, or mimic
known?

Why sampling the subcutis matters



Robbins explicitly stresses a generous deep wedge of subcutis for diagnosing erythema nodosum.

Classification axes serve different purposes

Morphologic description

- Septal or lobular localization
- Vasculitis: Present or absent
- Necrosis and inflammatory cell types

Etiologic interpretation

- Immune-associated inflammation
- Factitial injury or injected substance
- Lymphoma producing a mimic

Why erythema nodosum is an important comparator

- Erythema nodosum as the most common panniculitis.
- It frequently occurs on the lower legs and typically presents subacutely.
- Its defining histologic pattern is septal inflammation without vasculitis.

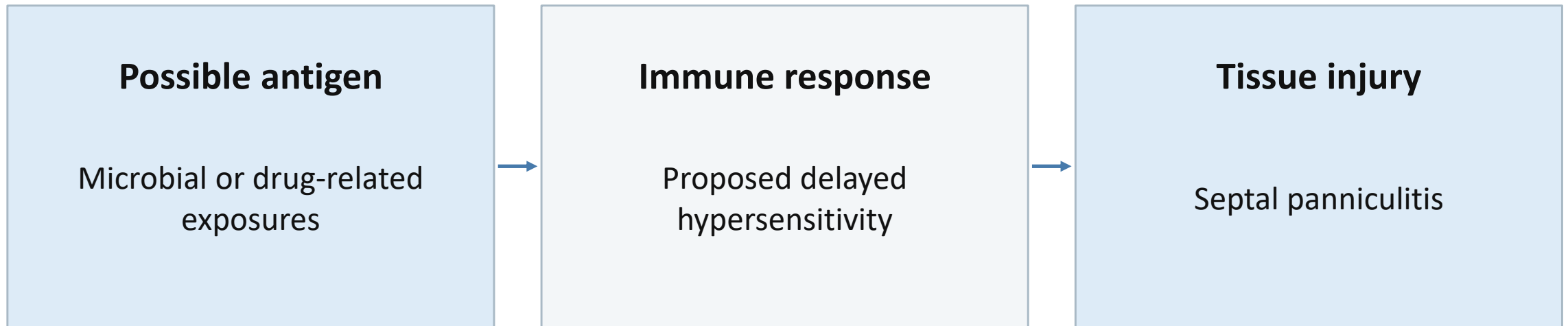
Erythema nodosum: Clinical phenotype

- Poorly defined, exquisitely tender erythematous plaques or nodules.
- They may be more readily palpable than visible.
- Fever and malaise can accompany the skin findings.

Erythema nodosum: Associated settings

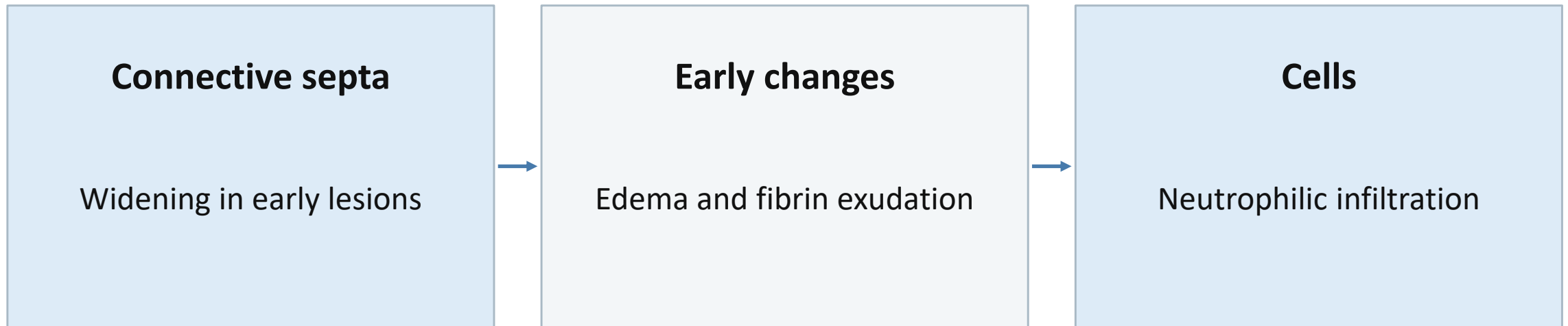
- Reported associations include infections and several drugs.
- Sarcoidosis, inflammatory bowel disease, and certain neoplasms are also associated.
- In many patients, the specific cause remains unidentified.

Erythema nodosum: Mechanism is not fully resolved



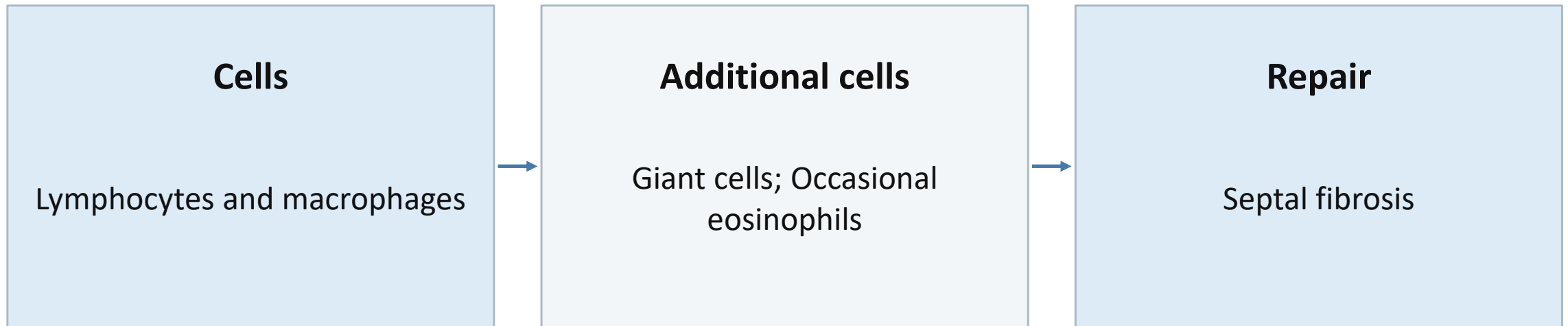
Immune complexes have been implicated in some cases; The persisting uncertainty.

Erythema nodosum: Early septal morphology



This is a septal-centered pattern, not a primary lobular process.

Erythema nodosum: Later changes



Histologic appearance varies with the age of a lesion.

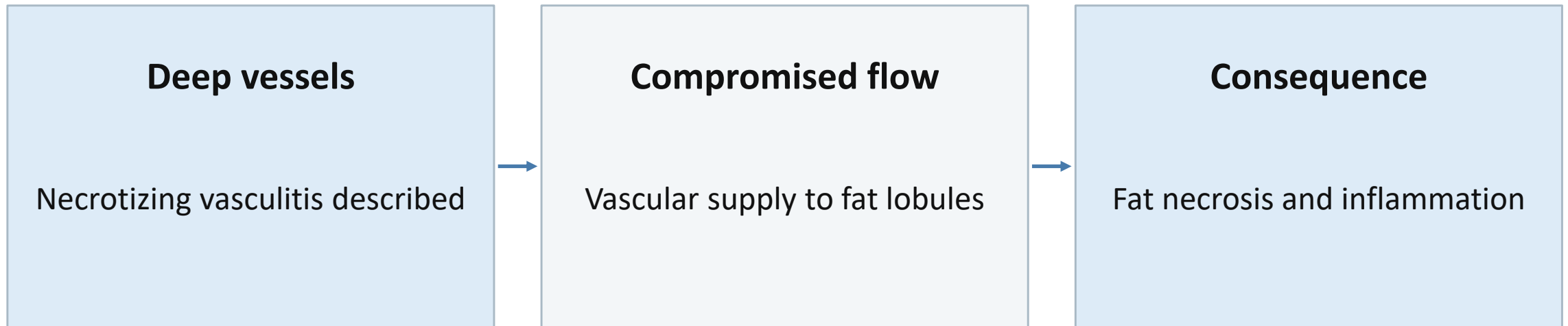
Erythema nodosum: Vasculitis is absent

- The reference description explicitly states that vasculitis is not present.
- This negative finding supports comparison with vasculitic panniculitides.
- It must be interpreted alongside the positive septal features and the clinical presentation.

Erythema induratum: Clinical clues

- An uncommon panniculitis, described particularly in adolescent and menopausal females.
- Typically presents as a slightly tender erythematous nodule.
- The nodule commonly progresses to ulceration.

Erythema induratum: Vessel-centered mechanism



Primary deep-vessel vasculitis as the prevailing interpretation.

Erythema induratum: Characteristic morphology

- Lobular granulomatous inflammation.
- Zones of caseous necrosis in fat lobules.
- Early necrotizing vasculitis in deep dermal and subcutaneous small- to medium-sized vessels.

Erythema nodosum versus erythema induratum

Feature	Erythema nodosum	Erythema induratum
Main location	Septa	Lobules
Vasculitis	Not present	Necrotizing vasculitis
Selected findings	Edema, fibrin, evolving fibrosis	Granulomas and caseous necrosis
Typical course	Bruise-like resolution; No scar	Nodule often ulcerates

The four “other” entities require different reasoning

- Weber–Christian disease is described as a relapsing, nonvasculitic lobular form.
- Factitial panniculitis is defined by deliberate physical or injected injury.
- T-cell lymphomas can infiltrate fat and mimic panniculitis.
- Systemic lupus erythematosus may cause inflammation of the subcutis.

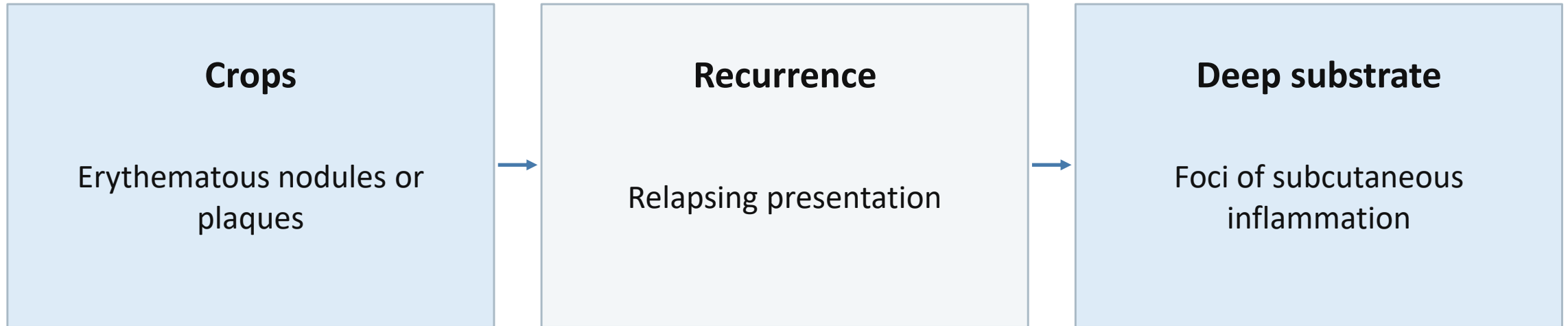
Weber–Christian disease: Source-defined identity

- Also termed relapsing febrile nodular panniculitis.
- Described as a rare lobular, nonvasculitic panniculitis.
- Occurs in children and adults.

Weber–Christian disease: Clinical distribution

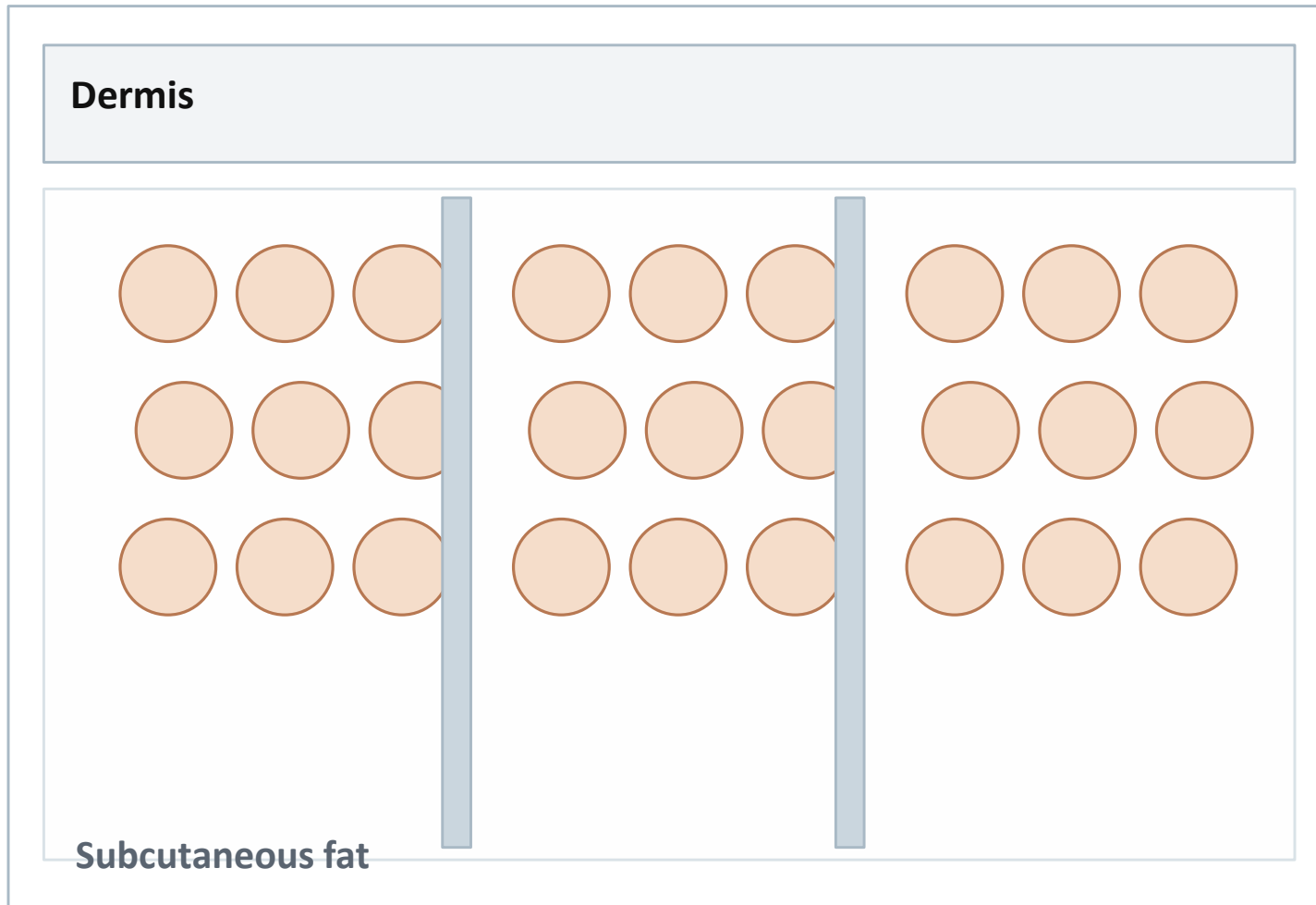
- Crops of erythematous plaques or nodules.
- Predominant involvement of the lower extremities.
- The source describes a relapsing presentation rather than a single fixed lesion.

Weber–Christian disease: Episodic lesion pattern



“Relapsing febrile nodular panniculitis” is the textbook synonym; The excerpt gives no specific fever frequency.

Weber–Christian disease is lobular, not septal



The fat lobule is the central compartment in the Robbins description.

Schematic anatomy, not a patient specimen

Weber–Christian disease: Absence of vasculitis

- The term nonvasculitic is part of the source definition.
- This contrasts with the deep necrotizing vasculitis of erythema induratum.
- Anatomical location and vascular status must both be described.

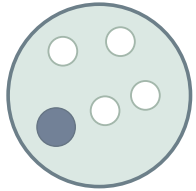
Weber–Christian disease: Foamy macrophage aggregates

- Deep-seated inflammatory foci contain aggregated foamy macrophages.
- These macrophages form part of a broader mixed inflammatory population.
- A macrophage-rich lobular infiltrate is descriptive, not sufficient by itself to specify etiology.

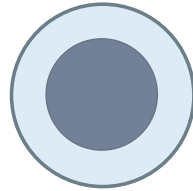
Weber–Christian disease: The associated cell mixture

- Foamy macrophages coexist with lymphocytes.
- Neutrophils are also included in the infiltrate.
- Giant cells are part of the source description.

Mixed inflammatory cells in Weber–Christian disease

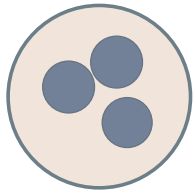


**Foamy
macrophages**

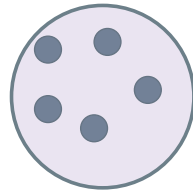


Lymphocytes

Schematic cell mixture — not histology



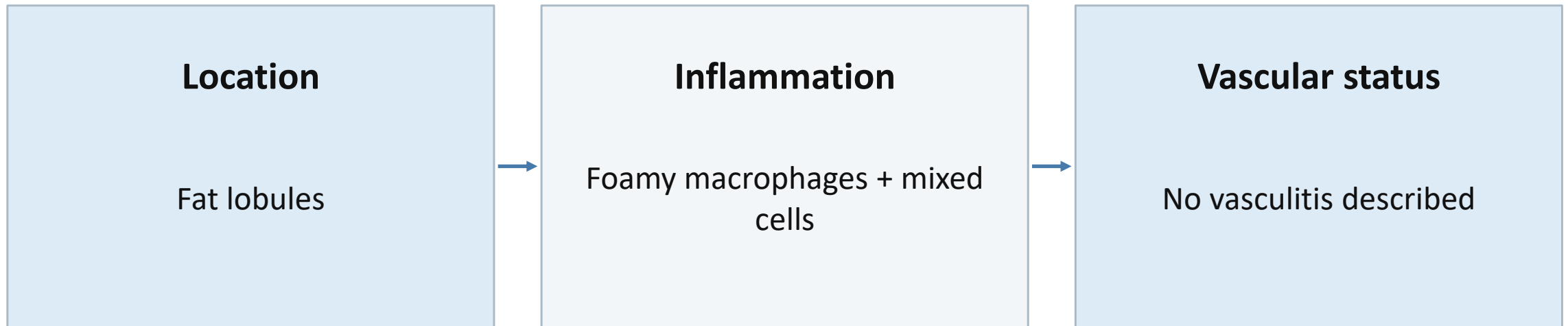
Neutrophils



Giant cells

These populations together in deep lobular inflammatory foci.

Integrated description of Weber–Christian disease



Combine clinical relapses and lower-extremity nodules with the histologic pattern.

Weber–Christian disease versus erythema nodosum

Weber–Christian

- Relapsing crops of nodules or plaques
- Lobular panniculitis
- Nonvasculitic; Foamy macrophages and mixed cells

Erythema nodosum

- Exquisitely tender nodules
- Septal panniculitis
- No vasculitis; Stage-related septal fibrosis

Weber–Christian disease versus erythema induratum

Weber–Christian

- Fat-lobule involvement
- Nonvasculitic
- Mixed inflammatory infiltrate

Erythema induratum

- Fat-lobule involvement
- Deep necrotizing vasculitis
- Granulomas and caseous necrosis

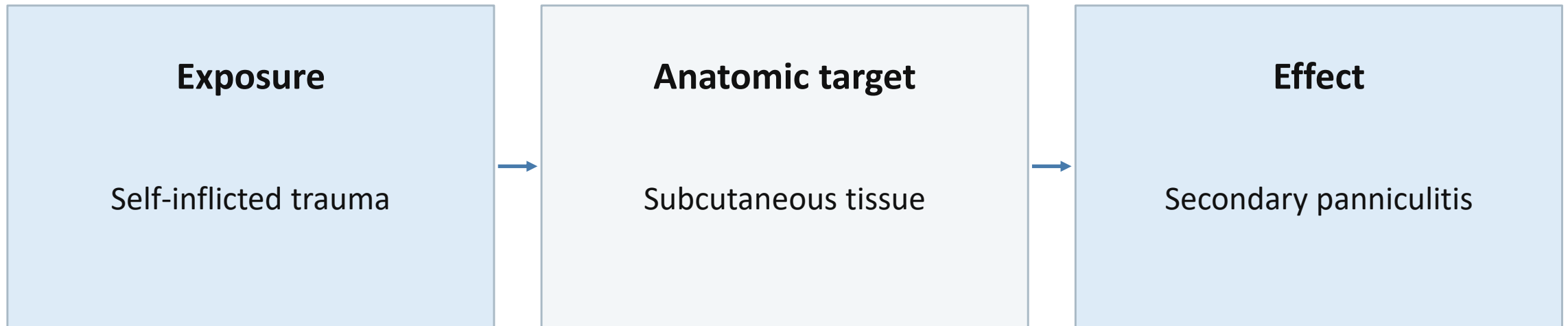
Weber–Christian disease: Limit the inference

- The excerpt does not supply a specific initiating cause.
- No diagnostic biomarker, staging system, or laboratory cutoff is provided.
- The named microscopic pattern should not be expanded into unverified molecular mechanisms.

Factitial panniculitis: Definition

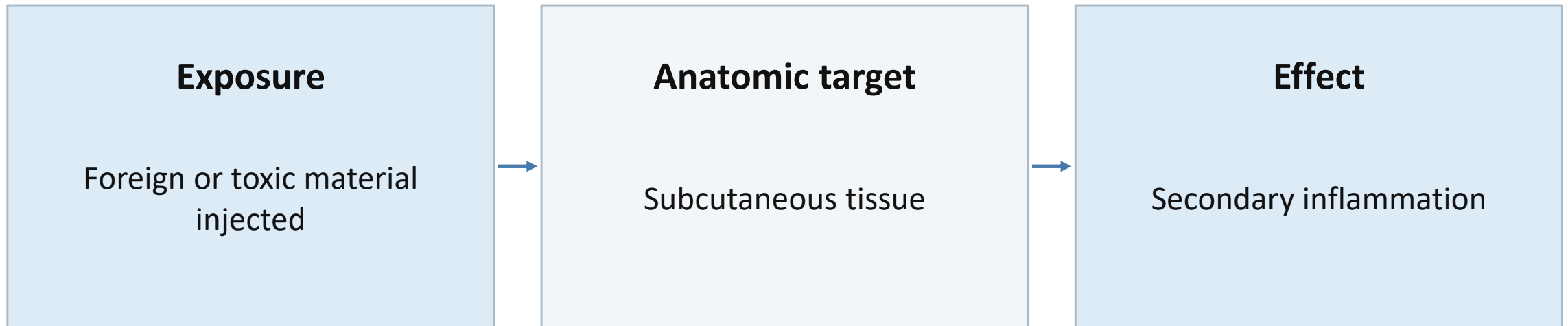
- A secondary form of panniculitis.
- Caused by self-inflicted trauma or injection of foreign or toxic material.
- The causal history—not merely septal/lobular anatomy—is central to the designation.

Factitial panniculitis: Trauma pathway



This is an etiologic linkage reported; No specific biopsy cell pattern is stated.

Factitial panniculitis: Injection pathway



The source does not specify any particular substance or histologic signature.

Factitial panniculitis: What the name tells us

Established from the excerpt

- The process is secondary
- Trauma or injection is causal
- Foreign or toxic materials may be involved

Not specified in the excerpt

- A pathognomonic cell type
- A defined vasculitic status
- Any laboratory confirmation threshold

Avoid inferring factitial causation from morphology alone

- The excerpt defines the exposure-based cause, but gives no unique morphology.
- A compatible inflammatory biopsy is not itself proof of self-injury.
- Establishing a causal history requires evidence beyond a nonspecific pattern.

Application: Evidence for factitial panniculitis

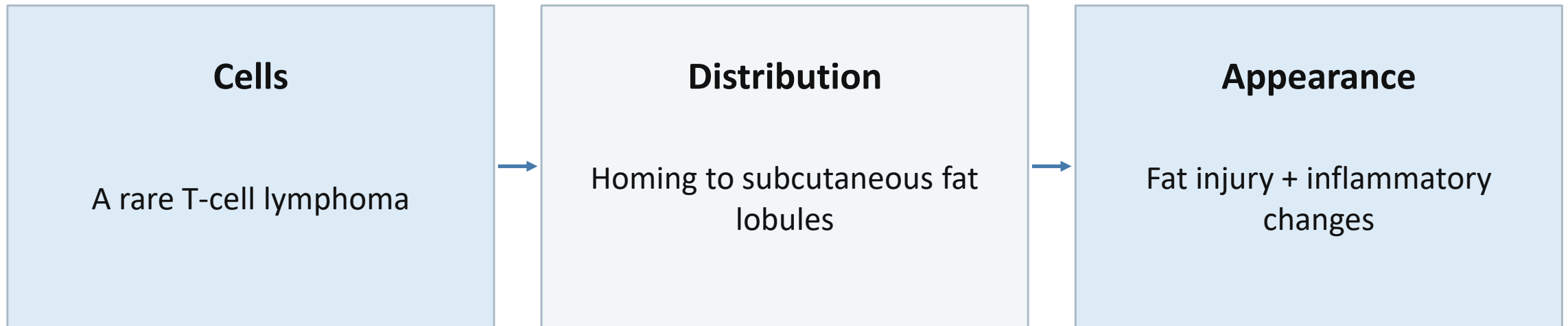
CLINICAL / PATHOLOGY SCENARIO

A patient has nodular subcutaneous inflammation and a verified history of repeated injections of a foreign material at the same locations.

REASONING

The injection exposure supports a secondary, factitial etiology. The excerpt does not justify assigning a unique septal/lobular or cellular pattern.

T-cell lymphoma may mimic panniculitis

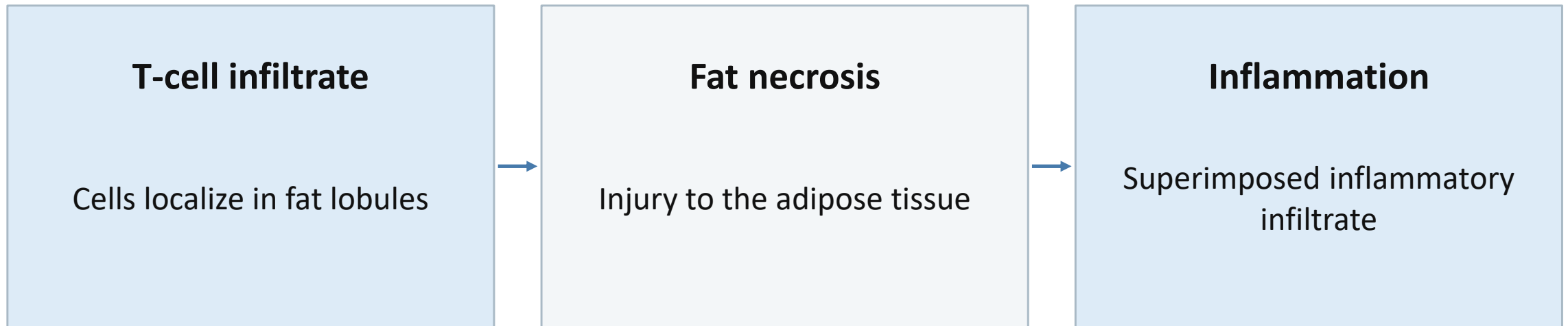


The source directly describes an inflammatory mimic of a malignant infiltrate.

T-cell lymphoma: Preferential fat-lobule localization

- Rare T-cell lymphomas with a tendency to home to fat lobules.
- Their presence produces a lobule-centered process.
- The appearance may overlap with non-neoplastic lobular panniculitis.

T-cell lymphoma: Resulting tissue injury



The necrosis and accompanying inflammation produce the panniculitis-like mimic.

Why panniculitis-like lymphoma is an important mimic

- The inflammatory component can dominate the perceived pattern.
- A tissue reaction that resembles panniculitis is not necessarily non-neoplastic.
- The textbook explicitly identifies lymphoma as a cause of deceptive lobular inflammation.

T-cell lymphoma mimic versus Weber–Christian disease

Lymphoma mimic

- Lymphomatous T cells target fat lobules
- Fat necrosis develops
- Superimposed inflammation imitates panniculitis

Weber–Christian description

- Lobular nonvasculitic panniculitis
- Foamy macrophages and mixed inflammatory cells
- Relapsing crops of lower-leg lesions

Do not convert a mimic into a benign diagnosis

- “Panniculitis-like” describes morphology, not behavior.
- The differential diagnosis must preserve the possibility of neoplasia.
- Robbins provides no immunophenotypic panel or molecular test in this short section.

Application: The malignant mimic

CLINICAL / PATHOLOGY SCENARIO

A biopsy shows a lobule-centered process with fat necrosis and inflammation; A rare T-cell lymphoma is also being considered.

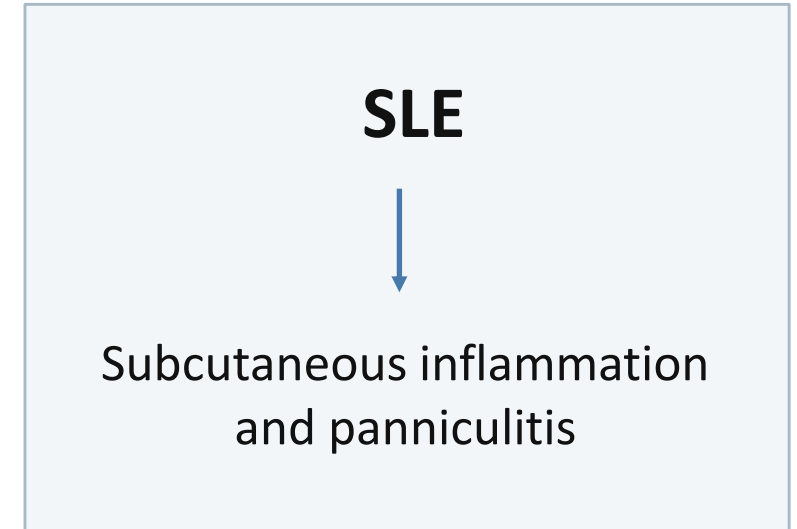
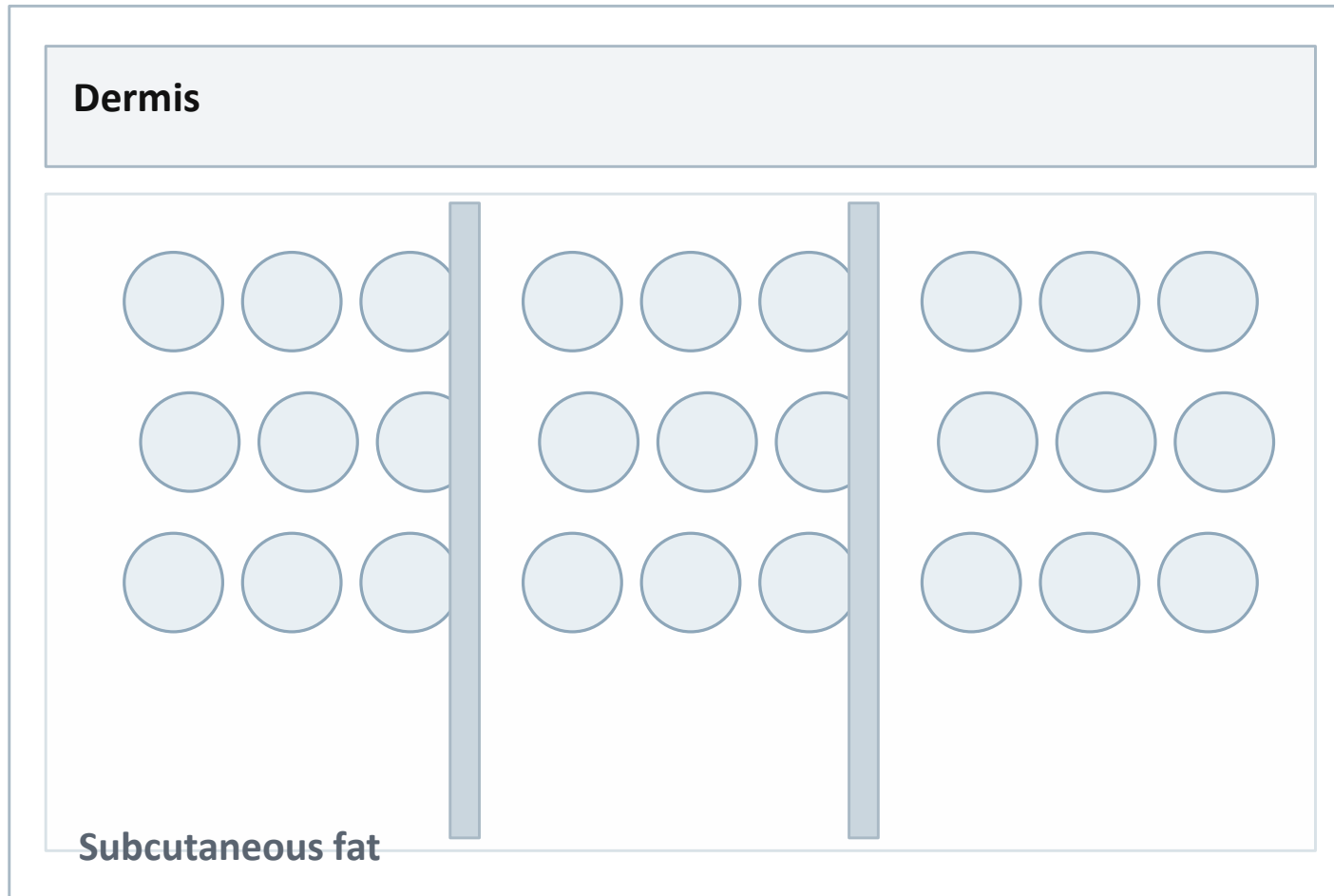
REASONING

The morphologic description alone does not exclude lymphoma. Robbins specifically warns that a fat-homing T-cell lymphoma can mimic panniculitis.

Systemic lupus erythematosus and panniculitis

- Systemic lupus erythematosus can occasionally be associated with subcutaneous inflammation.
- The involvement may present as an associated panniculitis.
- The short Robbins subsection does not give an entity-specific cellular or vascular profile.

Lupus-associated panniculitis involves the subcutis



Schematic only

Lupus-associated panniculitis: Limits of this excerpt

- No frequency, preferred body site, or distinctive microscopic pattern is specified.
- No lupus panniculitis-specific immunohistochemical finding is listed.
- The claim supported here is the clinical association with SLE and inflammation of subcutis.

Why the systemic disease context matters

- A subcutaneous inflammatory nodule can be considered in the setting of known SLE.
- The systemic context gives an etiologic lead, not a standalone tissue diagnosis.
- Robbins does not claim all panniculitis in patients with SLE is caused by lupus.

Application: Panniculitis with known SLE

CLINICAL / PATHOLOGY SCENARIO

A patient with systemic lupus erythematosus develops a subcutaneous inflammatory process. Which Robbins association is relevant?

REASONING

SLE is explicitly listed as a condition that can occasionally cause subcutaneous inflammation and an associated panniculitis. No further histologic specificity is justified from the excerpt.

Integrated comparison of the four named entities

Entity	Source-defined mechanism / context	Anatomy / injury
Weber–Christian	Relapsing febrile nodular form	Lobular; Nonvasculitic; Mixed cells
Factitial	Self-inflicted trauma or injection	Secondary inflammation; Pattern unspecified
T-cell lymphoma	Lymphoma homes to fat	Lobular; Necrosis; Inflammation mimic
SLE-associated	SLE with subcutis inflammation	Panniculitis; Pattern unspecified

Different causes can converge on a nodule

Inflammatory form

Relapsing
nonvasculitic
lobular panniculitis

External injury

Trauma or injected
material

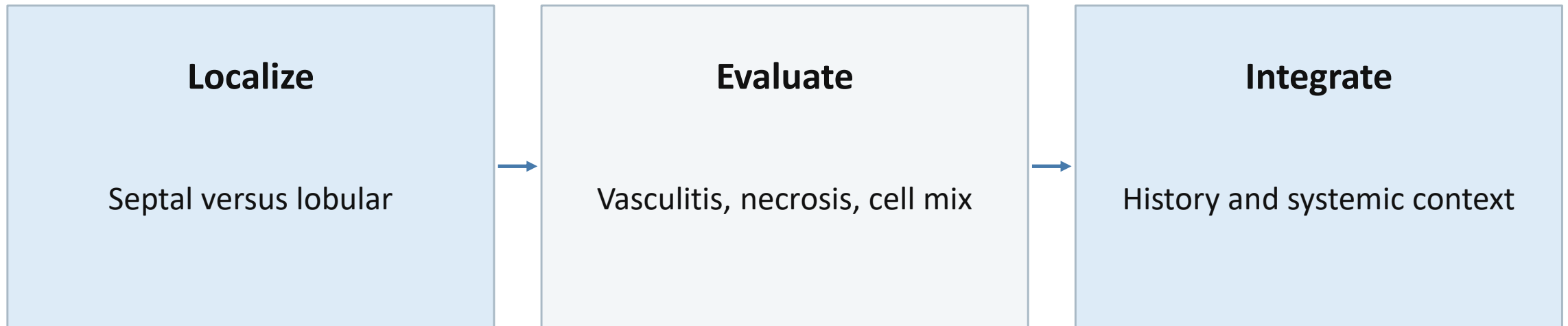
Neoplastic mimic

T-cell lymphoma
producing fat
necrosis

Systemic disease

SLE-associated
subcutaneous
inflammation

Morphology-first interpretation of panniculitis



Do not treat a pattern as a complete etiologic diagnosis.

Case comparison: Septal versus lobular

CLINICAL / PATHOLOGY SCENARIO

Two biopsies show subcutaneous inflammation. One is septal with fibrosis; The other is lobular, nonvasculitic, and rich in foamy macrophages.

REASONING

The first better fits the later erythema nodosum pattern. The second resembles the Weber–Christian description; Location and cell composition provide discrimination.

Case comparison: Vascular involvement

CLINICAL / PATHOLOGY SCENARIO

Two lobular lesions are considered. One has deep necrotizing vasculitis and caseous necrosis; The other is defined as nonvasculitic with a mixed inflammatory infiltrate.

REASONING

The first favors the Robbins description of erythema induratum; The second is compatible with the Weber–Christian description. Vasculitis is the crucial distinguishing axis.

Three common interpretive pitfalls

- Calling every lobular inflammatory process an idiopathic panniculitis despite a possible lymphoma mimic.
- Attributing factitial causation without supporting exposure evidence.
- Claiming specific lupus panniculitis histology that is not supplied by this source.

Integrated knowledge check

CLINICAL / PATHOLOGY SCENARIO

A recurrent lower-extremity nodular process is lobular and nonvasculitic; Foamy macrophages, lymphocytes, neutrophils, and giant cells are described. Which source-defined entity does this match?

REASONING

Weber–Christian disease (relapsing febrile nodular panniculitis). The description combines recurrence, localization, absence of vasculitis, and the mixed-cell pattern.

Clinicopathologic synthesis

- Localize disease to the subcutaneous septa or lobules.
- Ask whether vasculitis, fat necrosis, and distinct inflammatory populations are reported.
- Recognize injury, malignancy, and systemic disease as alternative etiologic contexts.
- Preserve uncertainty when the source supplies only an association or descriptive pattern.