

Fat Necrosis

Breast pathology · morphology and clinical mimics

A clinicopathologic approach to a benign lesion that can resemble carcinoma

Dr Abu Suraih Sakhri

MBBS, MD (Pathology)

www.abusuraihsakhri.com

Learning objectives

- Recognize the clinical presentations that make fat necrosis resemble carcinoma.
- Explain the time-dependent inflammatory and reparative response in injured breast fat.
- Identify the gross and microscopic features of evolving fat necrosis.
- Distinguish major inflammatory mimics described in the breast chapter.
- Apply morphology and clinical context to short diagnostic problems.

Why fat necrosis matters

Biology

Fat destruction followed by inflammation, fibrosis and sometimes calcification.

Presentation

A palpable mass, skin change, or mammographic abnormality may raise concern for cancer.

Pathologic task

Recognize repair while ensuring a suspicious lesion is not incorrectly dismissed.

Position among inflammatory breast disorders

Inflammatory disorders of the breast



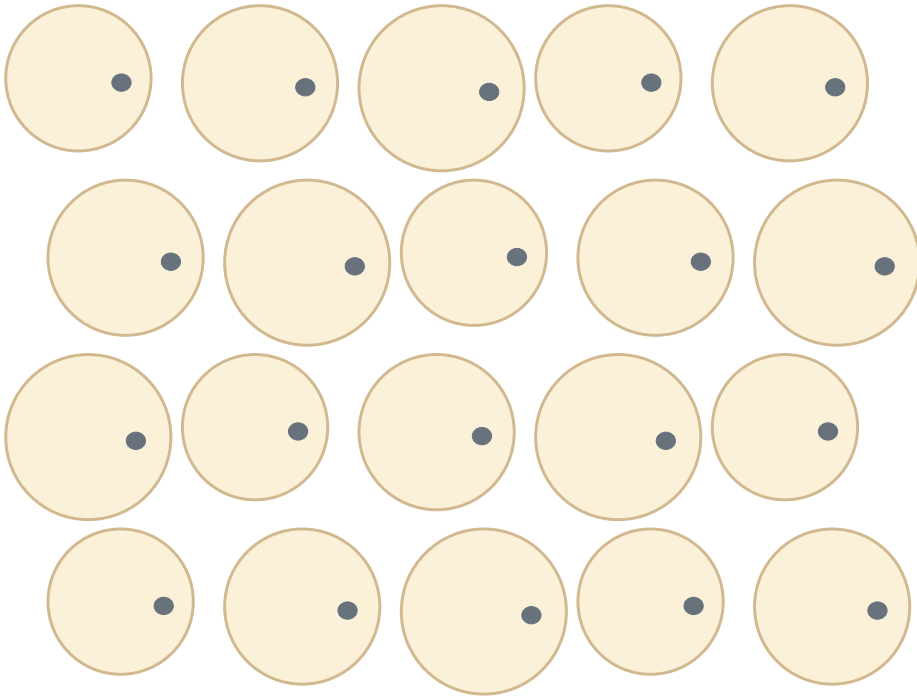
Fat necrosis: Predominantly adipose injury



Other mimics: Duct ectasia, squamous metaplasia, mastopathies

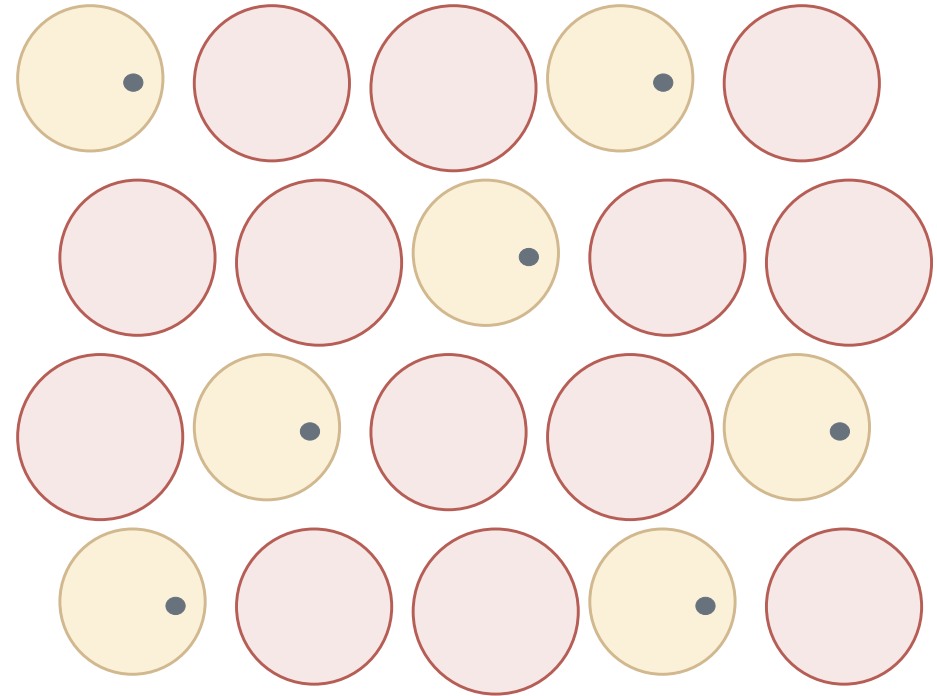
What is necrotic breast fat?

Preserved adipose tissue



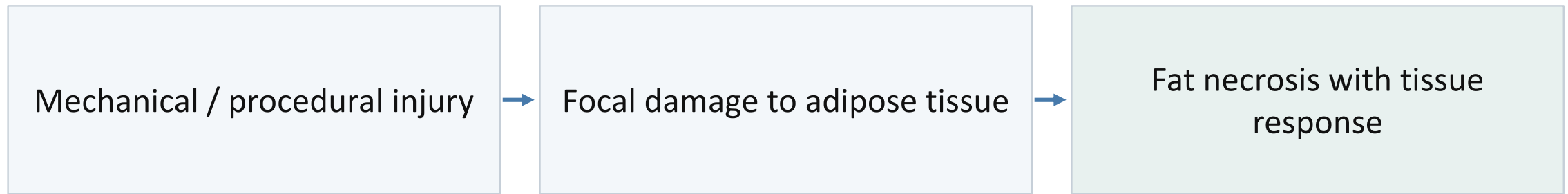
Schematic: Intact adipocyte contours and peripheral nuclei

Injured adipose tissue



Schematic: Ghost outlines, disrupted fat and cellular response

Antecedent injury: Trauma or surgery



An antecedent injury may be recognized, but a specific event is not always recalled.

A trauma history is not obligatory

- A history of breast trauma or surgery in about half of affected females.
- The remainder may not report a recognizable antecedent event.
- Therefore, absence of known trauma does not exclude fat necrosis.
- History modifies suspicion; It does not establish the diagnosis.

Painless palpable mass

Clinical trigger

A newly detected breast lump prompts assessment.

Potential confusion

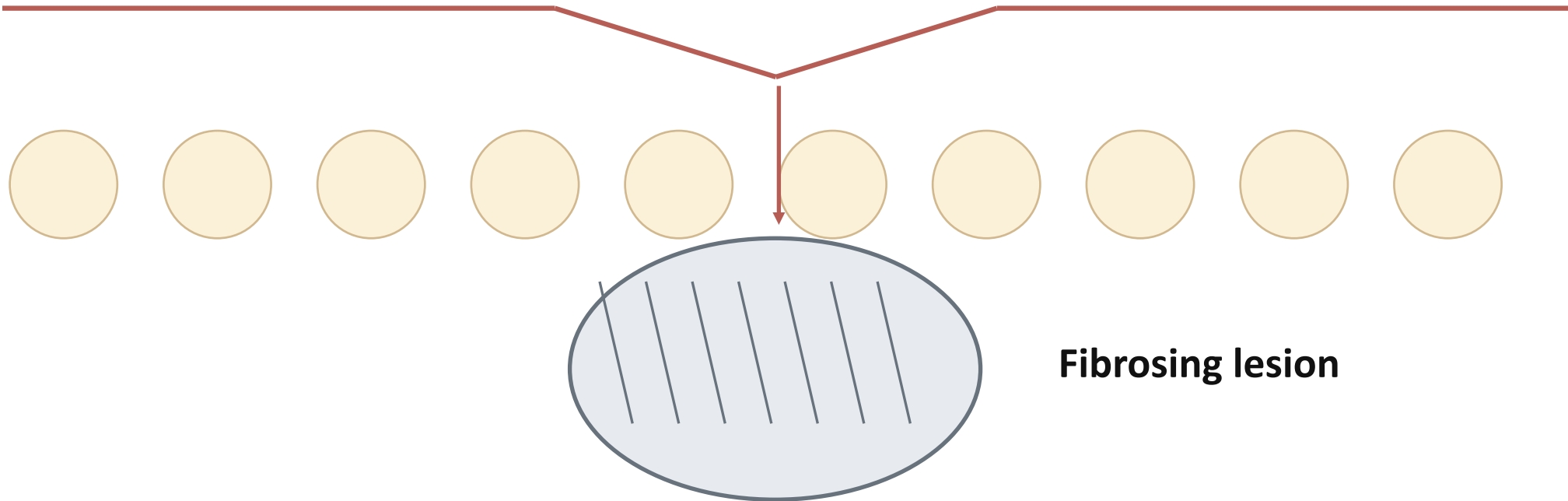
A firm benign inflammatory lesion can resemble an invasive tumor.

Interpretive point

Correlate the mass with the pathologic and radiologic findings.

Skin thickening and retraction

Skin dimple / retraction



Schematic: Fibrosis can tether overlying skin

Mammographic densities and calcifications

Inflammation / scarring



Tissue density



Mammographic abnormality

Fat injury / repair

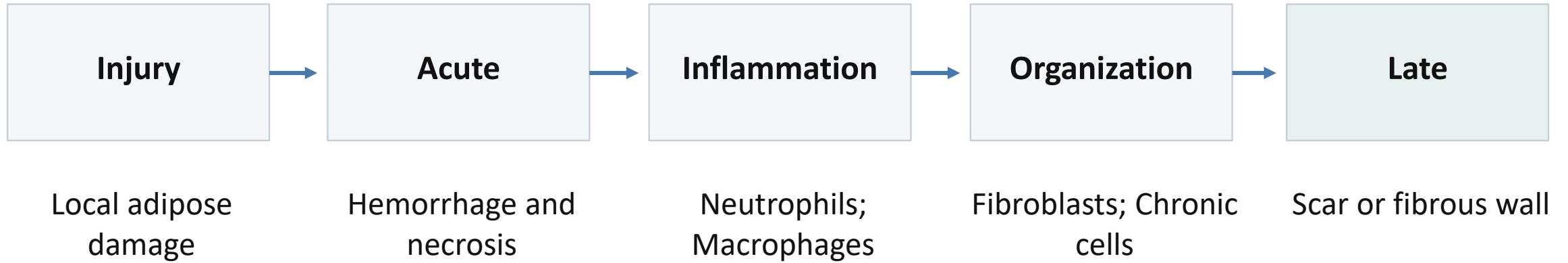


Calcification



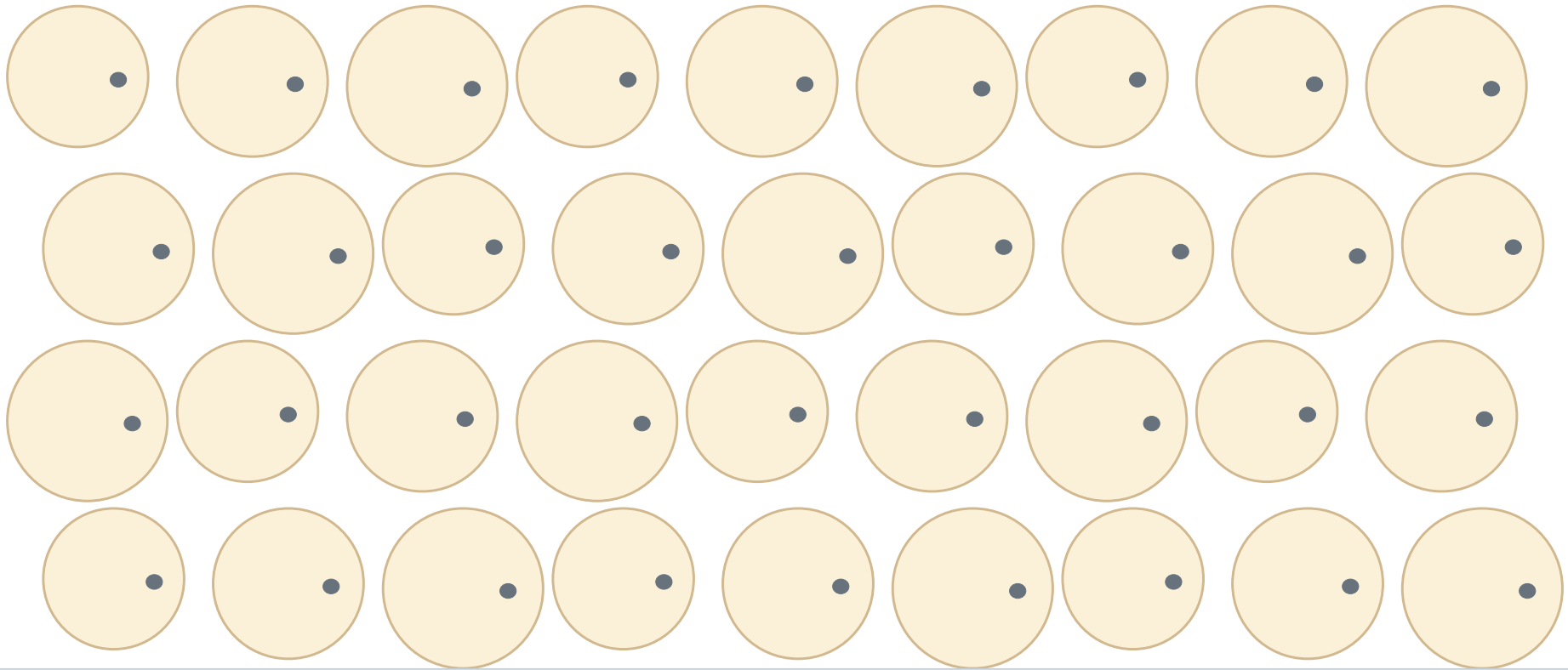
Mammographic abnormality

One lesion, several appearances over time



Normal adipose lobules: A reference schematic

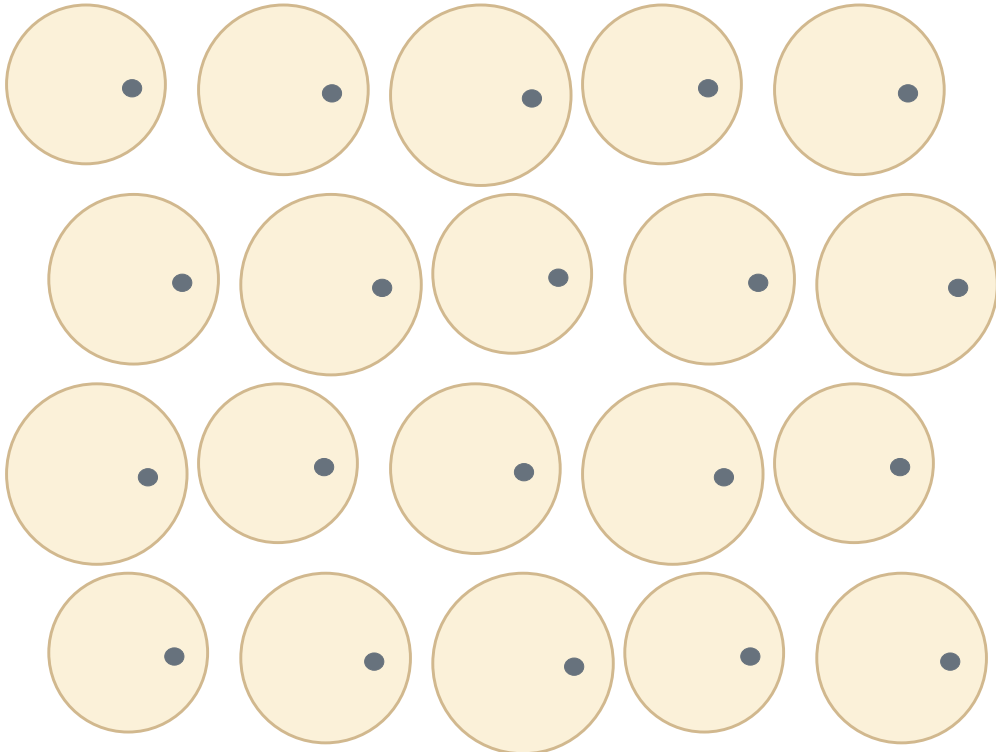
Intact fat cells



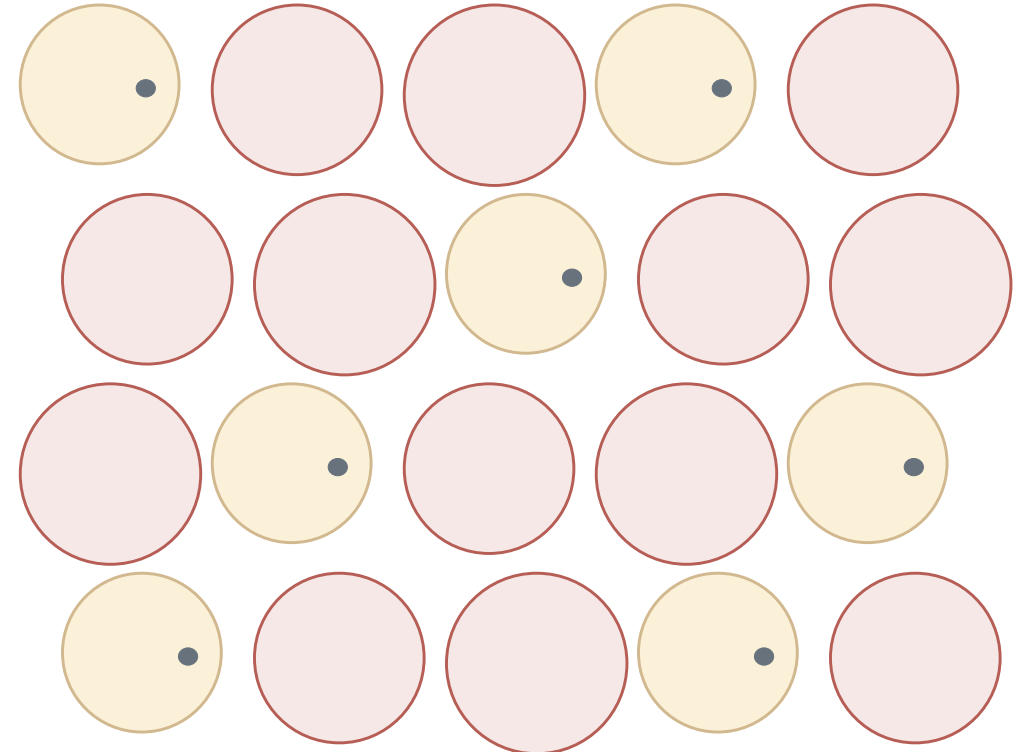
Normal adipose tissue provides a comparison for recognizing ghost fat cells and an inflammatory response.

Injured adipose tissue: Structural disruption

Compare cells at the injury interface



Uninjured reference (schematic)

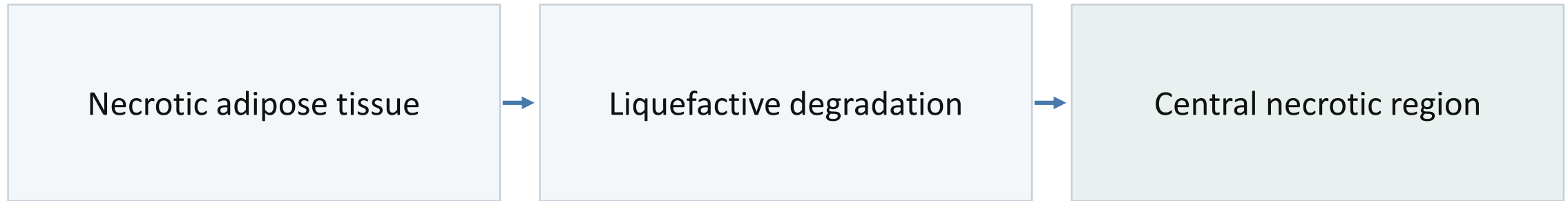


Necrotic fat (schematic)

Acute fat necrosis can be hemorrhagic

Tissue damage	Hemorrhage	Later clue
Necrosis arises in a locally injured region of adipose tissue.	Blood may accompany the earliest stage of fat injury.	Hemosiderin can remain as evidence of previous bleeding.

Liquefactive change in acute fat necrosis



Central liquefactive fat necrosis in acute breast lesions.

Neutrophils participate in early inflammation

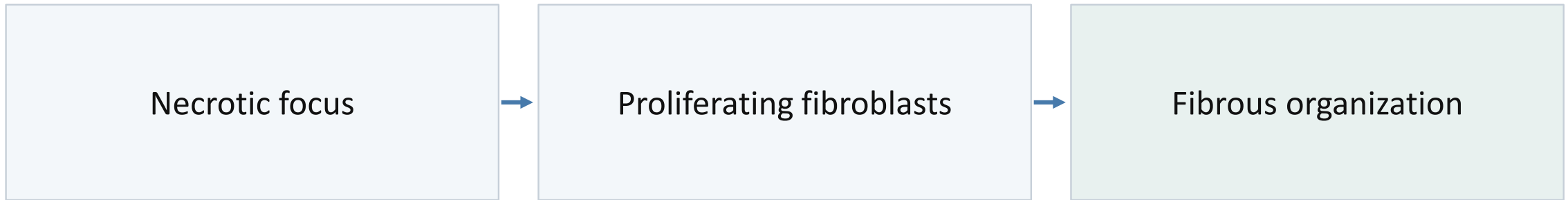
Location	Meaning	Interpretive caution
Present around areas of necrotic fat in acute lesions.	Reflect an acute inflammatory reaction to tissue injury.	Neutrophils alone do not define an infectious abscess.

Macrophages participate in fat clearance



Macrophages accompany neutrophils in the acute lesion.

Fibroblasts enter during organization

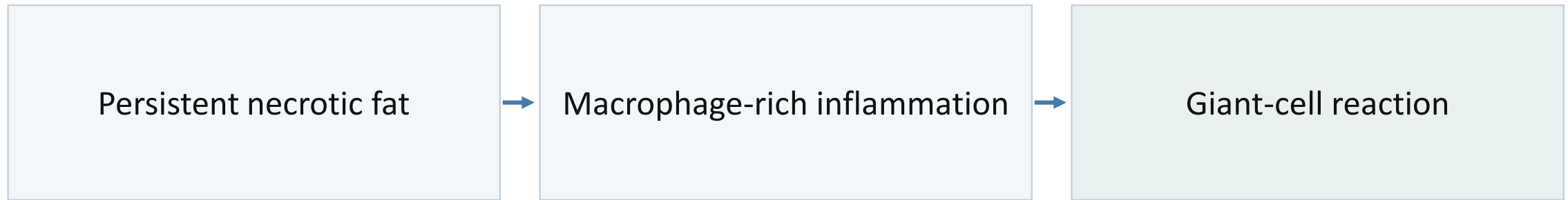


The transition toward fibrosis helps explain why a necrotic focus can become a firm mass.

Chronic inflammatory cells surround the injury

Spatial pattern	Cellular component	Clinical consequence
The response develops around the damaged central region.	Chronic inflammatory cells accompany the fibroblastic response.	Organization can persist as a palpable abnormality.

Multinucleated giant cells mark a later response

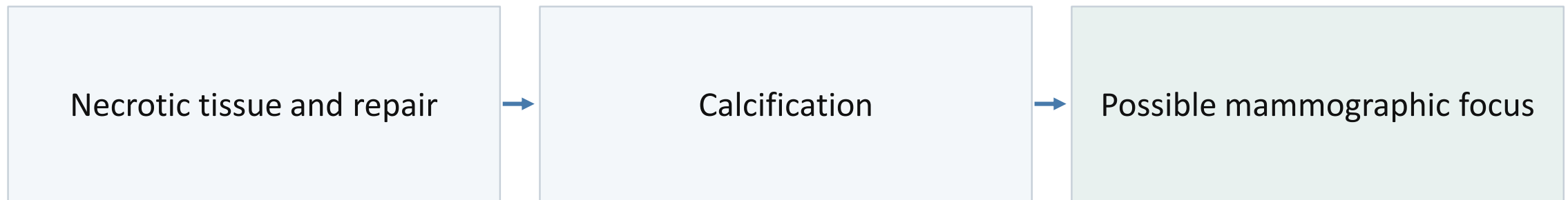


Giant cells occur as the lesion evolves; Their presence alone does not imply a specific infection.

Hemosiderin signals previous hemorrhage

Origin	Later finding	Interpretation
Earlier bleeding within an injured region.	Hemosiderin appears as the inflammatory process evolves.	Supports tissue injury; It is not a cancer-specific finding.

Calcifications arise during lesion evolution

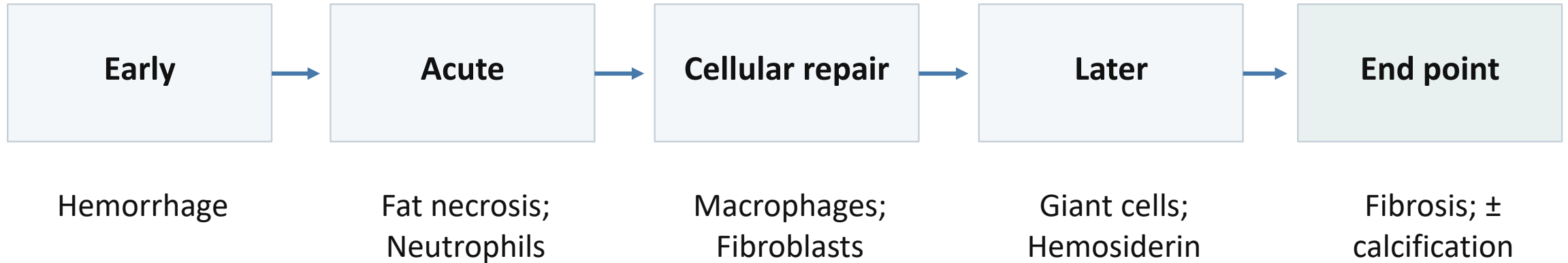


Calcifications are a common source of overlap with imaging findings of breast carcinoma.

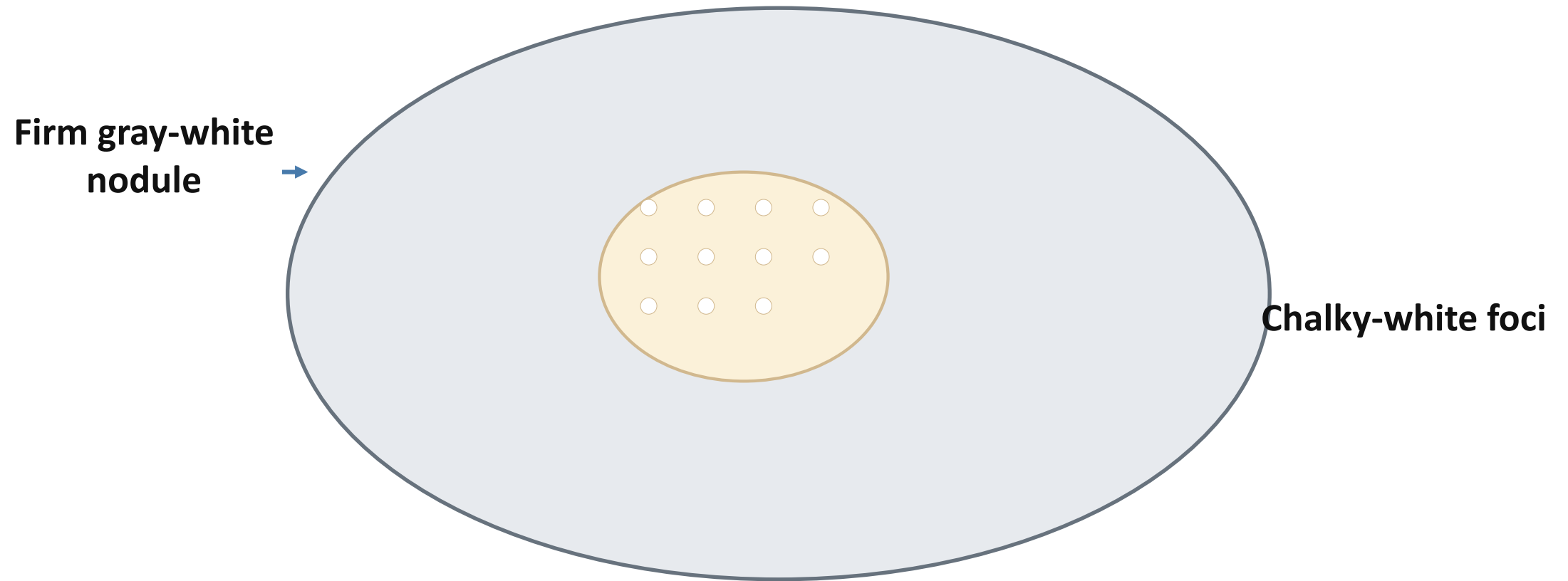
Scar formation is a common endpoint

Sequence	Outcome A	Outcome B
Inflammation and fibroblast proliferation organize the injured region.	The necrotic lesion can be replaced by scar tissue.	The damaged area may instead be walled off by fibrous tissue.

Histologic evolution: Integrate the sequence



Gross morphology: Firm gray-white nodule



Schematic illustration — not a gross specimen

Small chalky-white foci within a nodule

Macroscopic background	Internal appearance	Interpretation
An ill-defined, firm, gray-white breast nodule.	Small chalky-white foci may be present.	The appearance reflects a necrotic and reparative lesion, not a tumor phenotype.

Calcified fat: What saponification means

General pathology

Fatty acids can form calcium soaps; This produces chalky deposits in enzymatic fat necrosis.

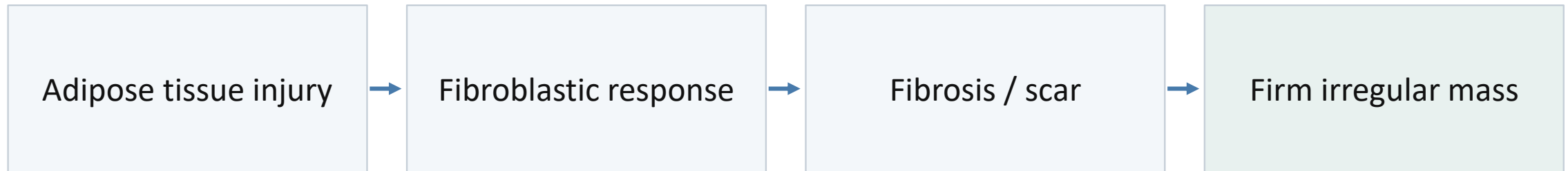
Breast lesion

Chalky gross foci and later calcifications in healing breast fat necrosis.

Distinction

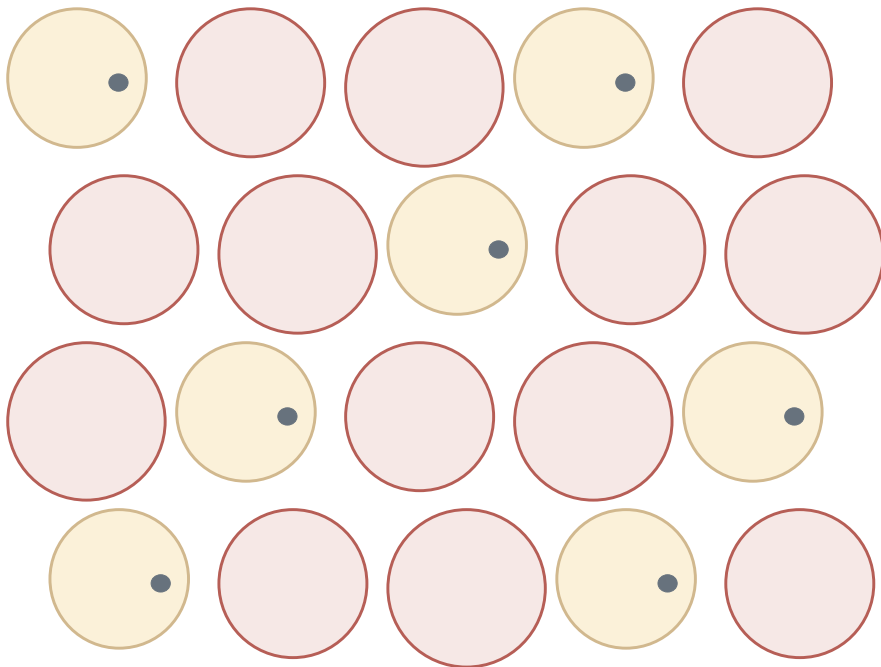
Mechanistic example in pancreatitis is not the same as the usual breast injury context.

Why the nodule can feel irregular and firm

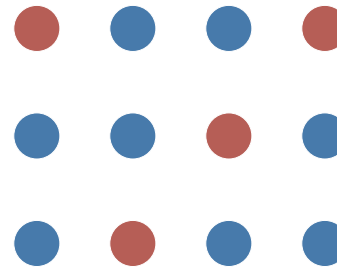


Palpation cannot reliably identify the tissue type responsible for the mass.

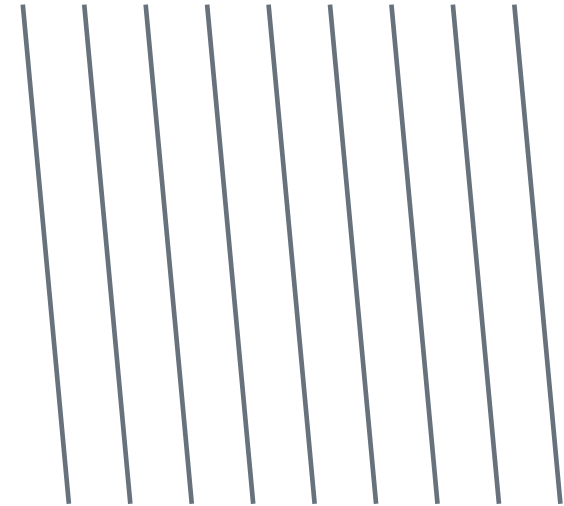
Low-power pattern: Necrosis plus repair



Necrotic fat (schematic)



Inflammatory cells (schematic)



Fibrosis (schematic)

Microscopic checklist for fat necrosis

- Central necrotic adipose tissue; Early areas may be liquefactive and hemorrhagic.
- An inflammatory response with neutrophils and macrophages.
- Peripheral fibroblast proliferation and chronic inflammatory cells.
- Later giant cells, hemosiderin and/or calcification.
- Fibrous encapsulation or replacement by scar.

Necrotic adipocytes versus viable adipocytes

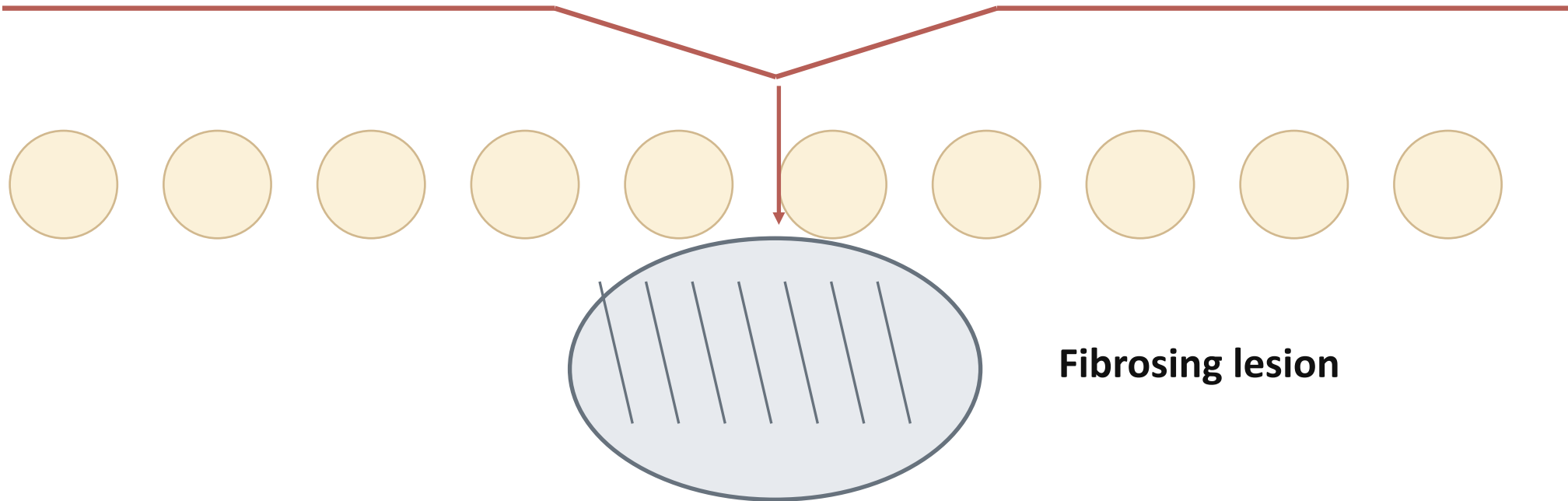
Feature	Viable adipose tissue	Necrotic fat region
Contours	Organized adipocytes	Residual or disrupted adipocyte outlines
Nuclear detail	Cell nuclei are present	Necrotic cells lose viability and nuclear detail
Surroundings	Usual stromal framework	Inflammatory cells and evolving repair
Later changes	No injury-associated scar	Possible giant cells, calcium and fibrosis

Inflammatory cell patterns must be staged

Phase	Reported cellular response	Interpretive consequence
Acute	Neutrophils and macrophages	Recent damage with active inflammation
Organizing	Fibroblasts + chronic inflammatory cells	Formation of reparative stroma
Evolved	Giant cells; $\pm$ hemosiderin	Response to persistent debris / prior bleed
Healed	Fibrosis $\pm$ calcification	Scarred lesion may persist as mass

Fibrotic contraction produces skin retraction

Skin dimple / retraction



Schematic: Fibrosis can tether overlying skin

Calcification is a morphologic and imaging bridge

Histology	Imaging	Diagnostic implication
Calcifications may appear in the developing necrotic lesion.	Mammographic calcifications are a possible mode of detection.	Their presence cannot, by itself, prove benignity or malignancy.

Why imaging and palpation can mimic carcinoma

Observed finding	Possible origin in fat necrosis	Why confusion occurs
Palpable mass	Fibrotic replacement of injured fat	A carcinoma can also be firm
Skin change	Fibrous traction / scarring	Carcinoma may cause retraction
Density	Inflammation and fibrosis	Mammography detects density, not histology
Calcification	Evolving calcified necrotic focus	Some carcinomas also calcify

Trauma history: Helpful but incomplete

History element	What it supports	What it cannot establish
Recent breast trauma	A plausible cause of focal injury	That every abnormality is benign
Previous surgery	A possible injury mechanism	That recurrent malignancy is excluded
No remembered injury	Does not rule out fat necrosis	Does not prove carcinoma

A benign lesion may warrant diagnostic assessment

Clinical or imaging abnormality



Determine whether the features can be explained by fat necrosis



If findings remain suspicious or discordant: Further assessment

What a representative tissue sample should show

- The sampled abnormality contains necrotic adipose tissue and a coherent inflammatory/reparative response.
- The composition can vary markedly with lesion age.
- Fibrosis, hemosiderin, calcification and giant cells support an evolving injury pattern.
- Sampling only scar may not capture an earlier necrotic center.
- Interpret morphology in relation to the lesion that was sampled.

Pitfall: An old scar may dominate the specimen

Early lesion

Necrosis, hemorrhage and acute inflammatory cells are conspicuous.

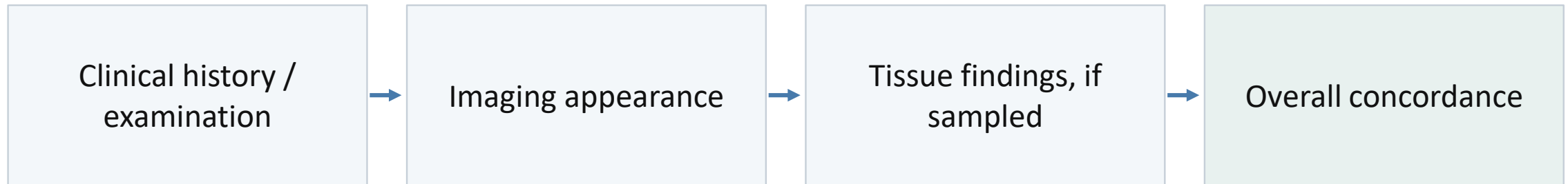
Late lesion

Dense collagen and calcification can predominate.

Sampling pitfall

A small sample may not represent the entire pathologic process.

Interpret breast lesions by concordance



A lesion that mimics carcinoma requires an explanation coherent across available evidence.

Duct ectasia: Another cancer mimic

Clinical location	Characteristic clue	Morphologic basis
Typically a periareolar mass.	Thick white nipple secretions can occur.	Dilated ducts with retained secretions, inflammation and fibrosis.

How duct ectasia produces an irregular mass



In duct ectasia the organizing process is centered on ducts rather than injured fat.

Squamous metaplasia: A distinct subareolar process

Primary abnormality	Consequence	Clinical phenotype
Keratinizing squamous metaplasia extends into lactiferous ducts.	Keratin plugging, duct dilation, rupture and granulomatous inflammation.	Painful erythematous subareolar mass; Recurrent disease may fistulize.

A recurrent subareolar fistula points away from fat necrosis

Finding	Fat necrosis	Squamous metaplasia
Typical presentation	Painless palpable mass often possible	Painful erythematous subareolar mass
Etiologic association	Trauma or surgery in some cases	Marked association with smoking
Key morphology	Necrotic fat and repair	Keratinizing duct epithelium with keratin debris
Characteristic complication	Fibrosis / retraction	Recurrent subareolar fistula

Lymphocytic mastopathy: Clinical profile

Presentation	Associations	Clinical significance
One or more hard palpable masses or mammographic densities.	Described with type 1 diabetes or Hashimoto thyroiditis.	Its major consequence is confusion with breast carcinoma.

Lymphocytic mastopathy: Stromal and duct features

- The stroma is densely collagenized and can impede needle sampling.
- Atrophic ducts and lobules remain within the collagenized tissue.
- Basement membranes may be thickened.
- Lymphocytic inflammation accompanies the epithelial structures.
- The pattern differs from the necrotic adipose tissue of fat necrosis.

Granulomatous mastitis: Broad etiologic context

Systemic disorders

Granulomatosis with polyangiitis and sarcoidosis are examples.

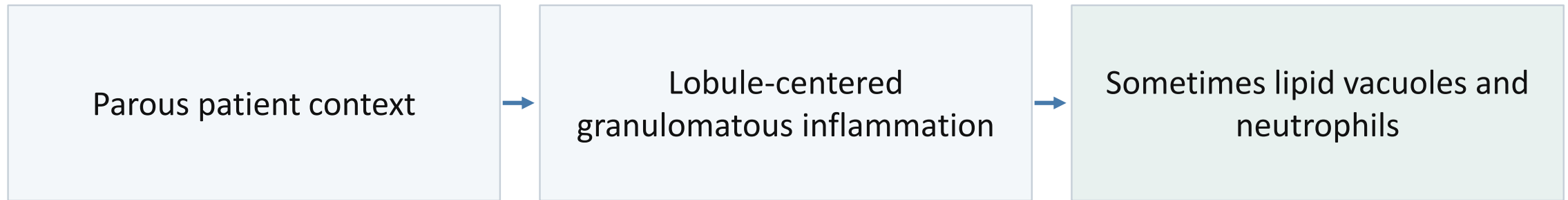
Infectious disease

Tuberculosis and localized infections can produce breast granulomas.

Localized lobular pattern

Granulomatous lobular mastitis is centered on lobules.

Granulomatous lobular mastitis: Defining pattern



A granulomatous reaction to lipid must be located and interpreted in its tissue context.

Cystic neutrophilic granulomatous mastitis

Morphologic clue	Microbiologic association	Relationship
Neutrophils surround lipid vacuoles within granulomatous inflammation.	Often associated with lipophilic Corynebacteria.	Overlap with granulomatous lobular mastitis.

Infections can also produce breast granulomas

- Mycobacterial or fungal breast infections are uncommon.
- They occur particularly in immunocompromised patients or adjacent to foreign objects.
- Breast prostheses and nipple piercings are contexts.
- A granulomatous response alone does not establish its etiology.

Inflammatory breast mimics: Distinguishing features

Entity	Clinical or anatomic clue	Morphologic center
Fat necrosis	Mass, skin change, density or calcification	Necrotic adipose tissue; Staged repair
Duct ectasia	Periareolar mass, white secretion	Dilated duct; Inspissated debris and fibrosis
Squamous metaplasia	Painful recurrent subareolar abscess / fistula	Keratin-plugged lactiferous duct
Lymphocytic mastopathy	Hard masses; Autoimmune disease association	Densely collagenized stroma and lymphocytes
Granulomatous mastitis	Parous context for lobular form	Lobule-centered granulomatous inflammation

Treatment differs according to the cause

Antibiotics

Relevant when a bacterial process is involved.

Steroids

Sometimes used for granulomatous inflammatory disease.

Surgery

May be indicated for specific structural lesions, e.g. Recurrent keratinizing duct disease.

Always consider an underlying carcinoma

Overlapping signs

Mass, retraction, density and calcification occur in benign lesions.

Important alternative

Inflammatory carcinoma may also mimic nonneoplastic breast inflammation.

Implication

Benign pathology must explain the clinical and imaging abnormality.

A conceptual pathway for an inflammatory breast lesion

Describe location, symptoms and imaging appearance



Determine whether the focus is fat, duct, lobule or collagenized stroma



Identify specific injury / inflammation / infection clues



Resolve possible malignancy mimic by evidence concordance

Case 1: A mass after local injury

Presentation

A patient notices a painless breast lump after a remembered blunt injury. Tissue examination shows necrotic adipose tissue, macrophages and reparative fibrosis.

What process most coherently explains the findings?

Interpretation

Breast fat necrosis.

The antecedent injury plus necrotic fat and organization form a coherent lesion pattern.

Case 2: Calcifications after surgery

Presentation

A prior surgical site develops a firm nodule and mammographic calcifications. The excised focus has giant cells, hemosiderin and scar around necrotic fat.

Why are the imaging and histologic findings linked?

Interpretation

Evolving fat necrosis with reparative calcification.

Calcification and fibrosis are expected later components of the lesion.

Case 3: Periareolar mass with secretion

Presentation

An older multiparous patient presents with a periareolar mass and thick white nipple discharge. Histology shows dilated ducts containing inspissated secretions.

Which inflammatory disorder is better supported?

Interpretation

Mammary duct ectasia.

The process is centered on dilated ducts, not a primary necrotic focus in adipose tissue.

Case 4: Recurrent painful subareolar disease

Presentation

A patient who smokes develops repeated painful subareolar inflammation and a draining fistula. Ducts contain keratin plugs and squamous metaplasia.

What diagnosis accounts for recurrence?

Interpretation

Squamous metaplasia of lactiferous ducts.

Retained abnormal keratinizing epithelium drives recurrent duct obstruction and inflammation.

Case 5: A hard mass in type 1 diabetes

Presentation

A patient with type 1 diabetes has a hard breast mass. Histology shows dense collagen, atrophic lobules, thickened basement membranes and lymphocytes.

How does this differ from fat necrosis?

Interpretation

Lymphocytic mastopathy.

Dense collagenization with duct-lobular changes is the defining pattern; Necrotic fat is not.

Case 6: Lobule-centered granulomas

Presentation

A parous patient has granulomatous inflammation centered on mammary lobules, with lipid vacuoles and accompanying neutrophils.

Which adjacent disorder belongs in the differential?

Interpretation

Granulomatous lobular mastitis.

The distribution and granulomatous morphology point to a lobular inflammatory process.

Synthesis: Four essential diagnostic links

Cause and context	Evolution	Recognition	Safety
Recognized trauma / surgery may be present, but not in every case.	Necrotic fat → inflammation → fibroplasia → scar or fibrous wall.	Hemorrhage, cells of injury response, giant cells, hemosiderin and calcifications vary by stage.	Clinical and mammographic resemblance to carcinoma requires concordant interpretation.

Selected source literature

- Robbins, Cotran & Kumar. Pathologic Basis of Disease. 11th ed. Elsevier; 2026. Chapter 23, The Breast, p. 947 (primary source).
- Robbins, Cotran & Kumar. Pathologic Basis of Disease. 11th ed. Chapter 2 (general mechanism of enzymatic fat necrosis; separate context).
- Kerridge WD, et al. Fat necrosis of the breast: a pictorial review with histopathologic correlation. Radiol Res Pract. 2015;2015:613139.
- Source clarification: original chapter PDFs and licensed diagnostic photomicrographs were not available for direct image-level verification in this build.