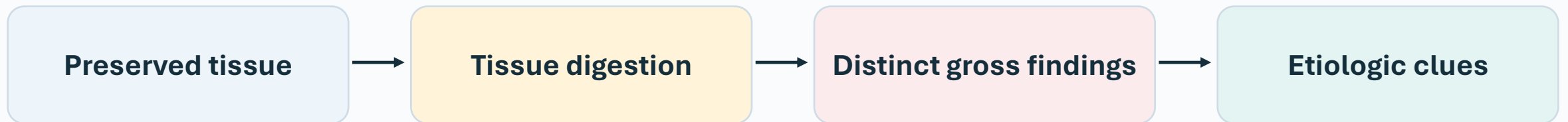


Patterns of Tissue Necrosis

Six morphologic patterns — causes, gross findings, histology, and clinical interpretation



Dr Abu Suraih Sakhri

MBBS, MD (Pathology)

www.abusuraihsakhri.com

Learning outcomes

- Name and distinguish six morphologic patterns of tissue necrosis.
- Predict dominant necrotic appearance from the mechanism and organ involved.
- Recognize tissue architecture, nuclear loss, inflammatory debris, and mineral deposition.
- Link kidney infarction, cerebral infarction, tuberculosis, pancreatitis, and vascular injury to their typical patterns.
- Avoid interpreting a descriptive necrosis pattern as an etiologic diagnosis in isolation.

Tissue necrosis is a collective process

A tissue becomes necrotic when large numbers of its cells die.

- A myocardial infarct is a tissue-level consequence of many myocardial cell deaths.
- Individual-cell changes are interpreted within tissue architecture and inflammatory response.
- Gross and microscopic patterns offer clues to causation, not definitive proof of an etiology.

Six recognized descriptive patterns

Coagulative

Preserved tissue framework

Liquefactive

Enzymatic dissolution

Gangrenous

Clinical limb designation

Caseous

Granular necrosis in granuloma

Fat

Lipolysis and calcium soaps

Fibrinoid

Eosinophilic vascular-wall
injury

What determines the dominant appearance?

Protein denaturation

Preservation of cell outlines and tissue framework despite cell death.

Enzymatic digestion

Proteolysis of dead cells and dissolution of tissue architecture.

Site and insult

CNS ischemia, infection, pancreatic enzymes, and vascular immune injury favor different appearances.

Evolution over time

Inflammation, phagocytosis, and calcification modify the initial lesion.

The central morphologic opposition

Protein / enzyme denaturation



Architecture persists



COAGULATIVE

Predominant enzymatic digestion



Framework dissolves



LIQUEFACTIVE

These are dominant tissue-level morphologies, not mutually exclusive molecular pathways.

Necrotic cytoplasm on H&E

Increased eosinophilia

Loss of basophilic RNA and accumulation of denatured proteins increase affinity for eosin.

Homogeneous / glassy quality

Loss of intracellular glycogen and structural changes reduce normal granular detail.

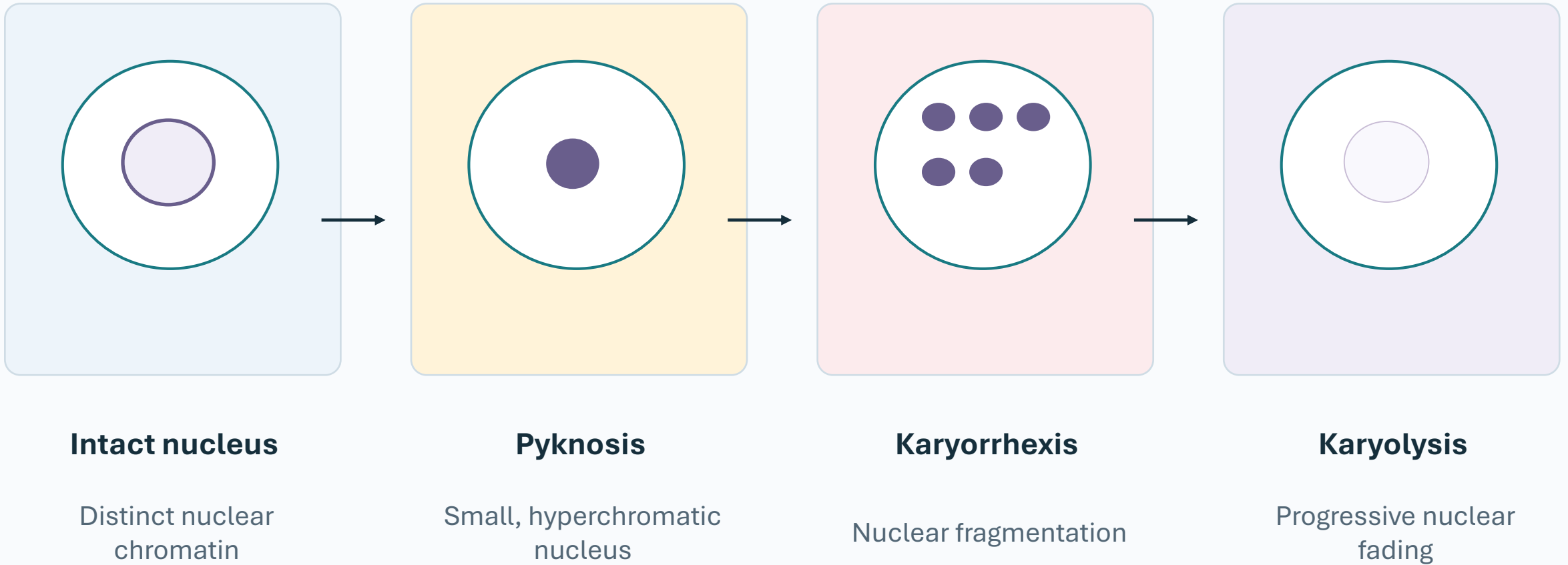
Moth-eaten vacuolation

Digestive enzymes destroy organelles and create a vacuolated cytoplasm.

Myelin figures

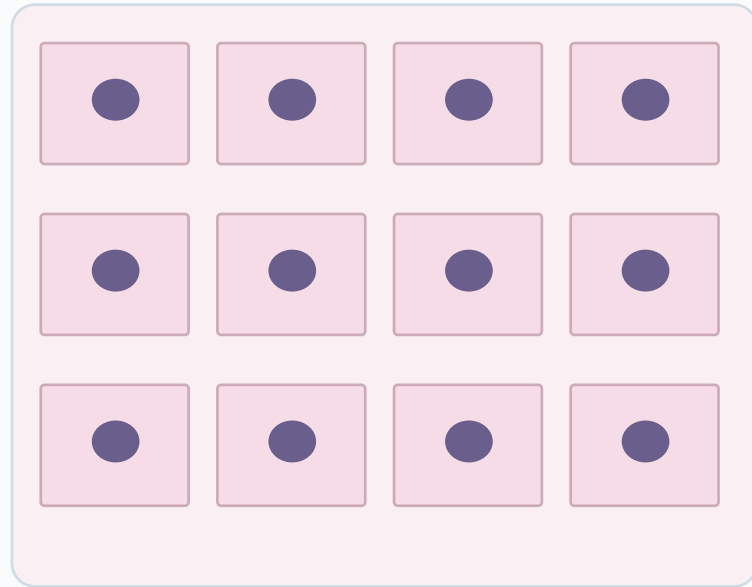
Whorled phospholipid debris may be phagocytosed, degraded, or associated with calcium deposition.

Nuclear changes in necrotic cells

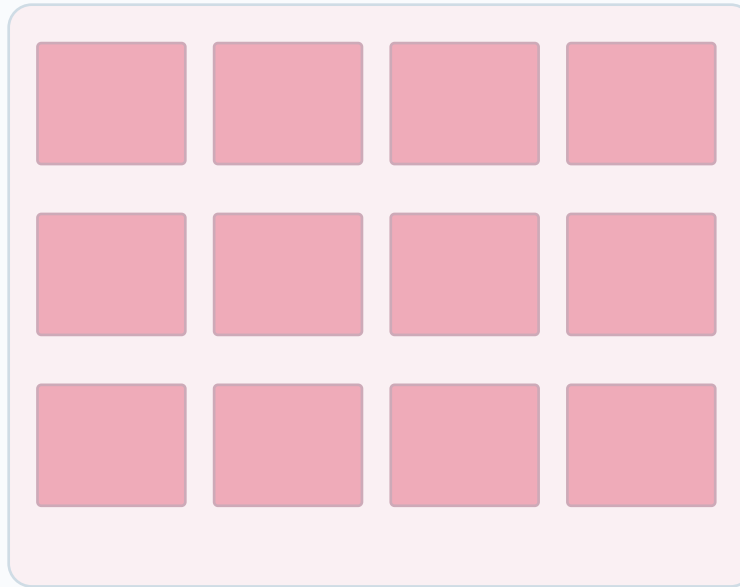


Schematic nuclear appearances; All three nuclear endpoints need not be present simultaneously.

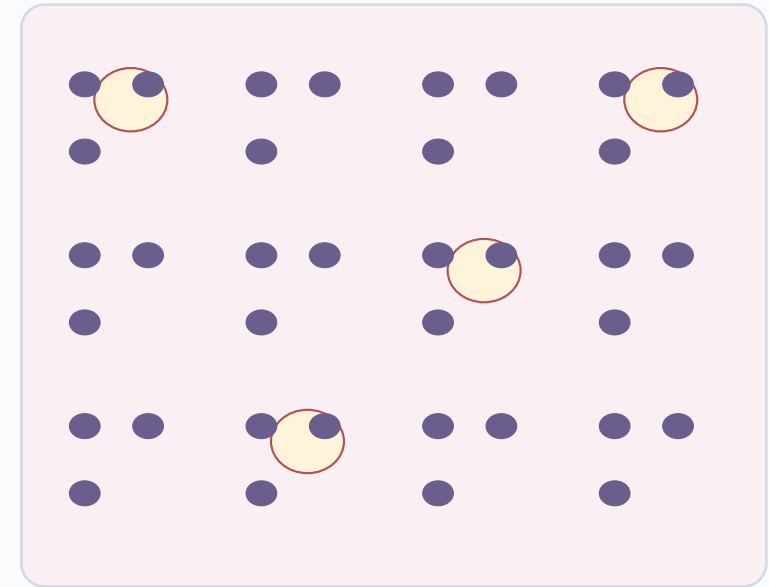
Cell outlines versus tissue dissolution



Viable tissue



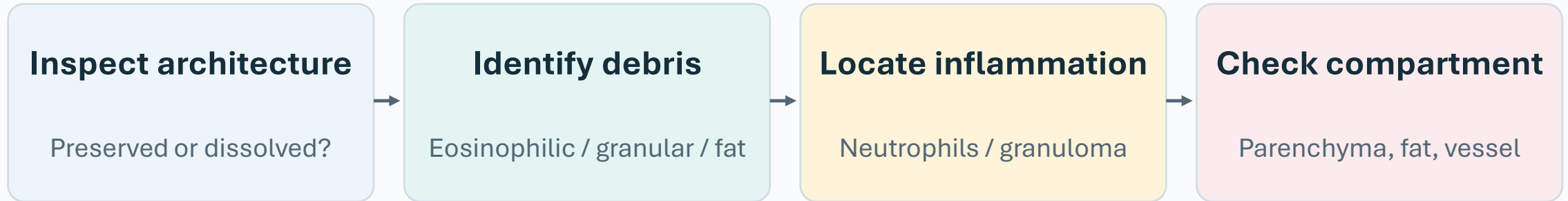
Coagulative necrosis



Liquefactive necrosis

Schematic H&E-like fields: Not a photomicrograph; Tissue architecture is illustrated conceptually.

At the microscope: A systematic approach



- Combine morphology with site, clinical setting, and relevant organism or vascular studies.

An organ distribution is often diagnostic context

Setting	Dominant pattern	Reason to consider it
Solid-organ ischemic infarct	Coagulative	Protein denaturation; Tissue preserved
CNS infarct	Liquefactive	Digestion and softening
Suppurative infection	Liquefactive	Leukocyte-associated digestion
Tuberculous focus	Caseous	Necrotizing granulomatous process
Acute pancreatitis with fat injury	Fat	Lipase action and calcium soaps

Molecular cause and gross label are different

Biologic events

- Loss of cell viability and membrane function.
- Enzyme activity and protein damage.
- Leukocyte recruitment and clearance.

Descriptive labels

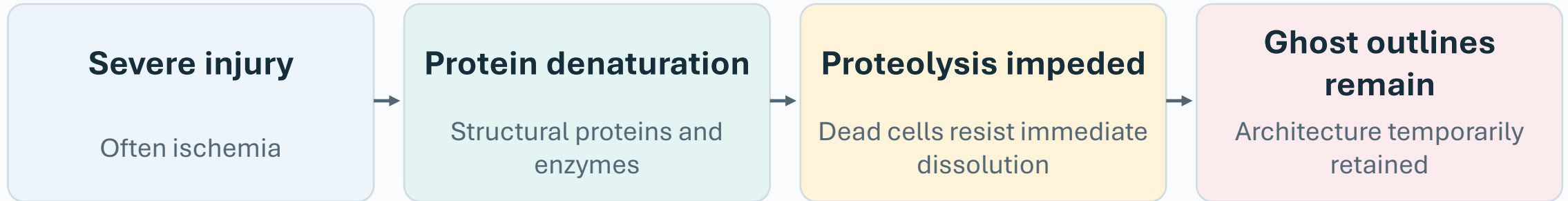
- Firm infarct; Soft suppurative cavity.
- Cheeselike focus; Chalky fat deposits.
- Necrotic limb; Bright pink vessel wall.

Coagulative necrosis: Definition

Dead cells persist with their basic tissue architecture recognizable.

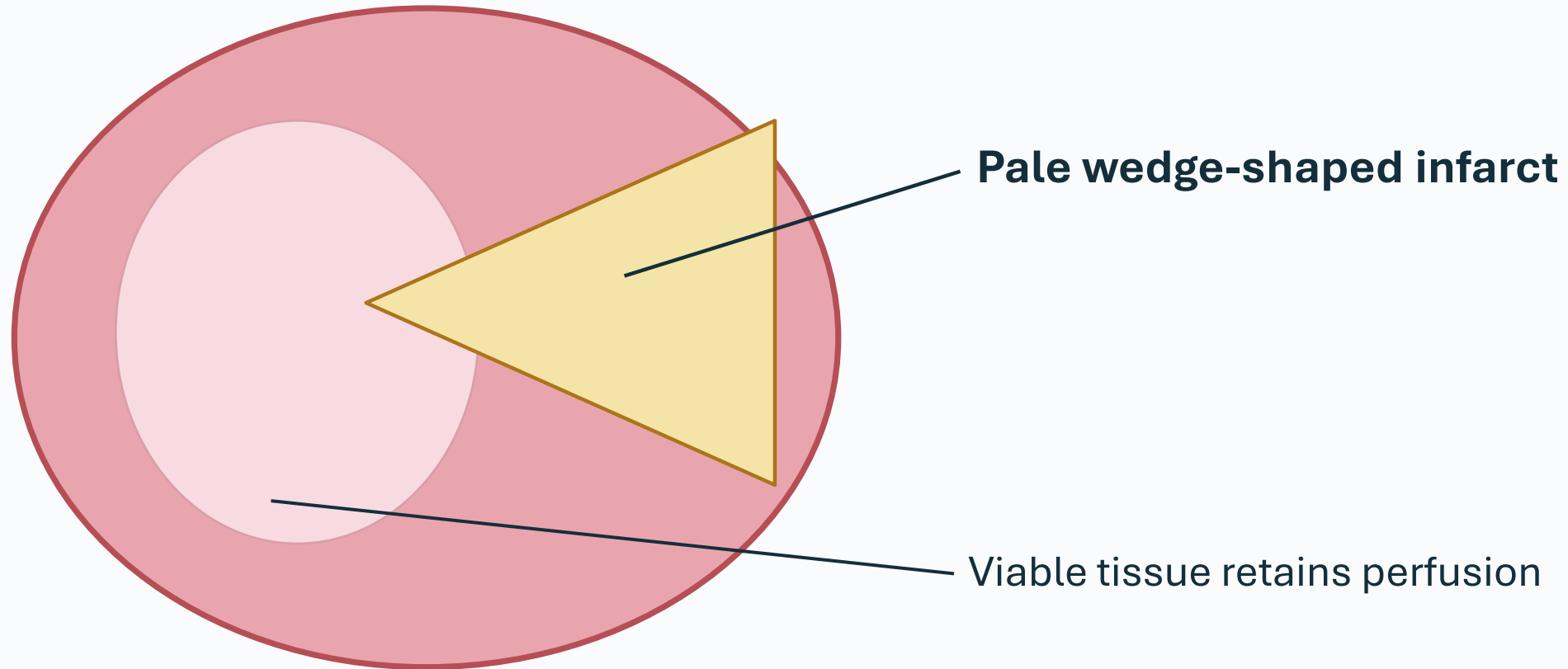
- A firm gross texture is characteristic.
- Cellular outlines can remain visible for at least several days.
- Ultimately leukocytes and lysosomal enzymes remove the dead material.

Why coagulative architecture persists



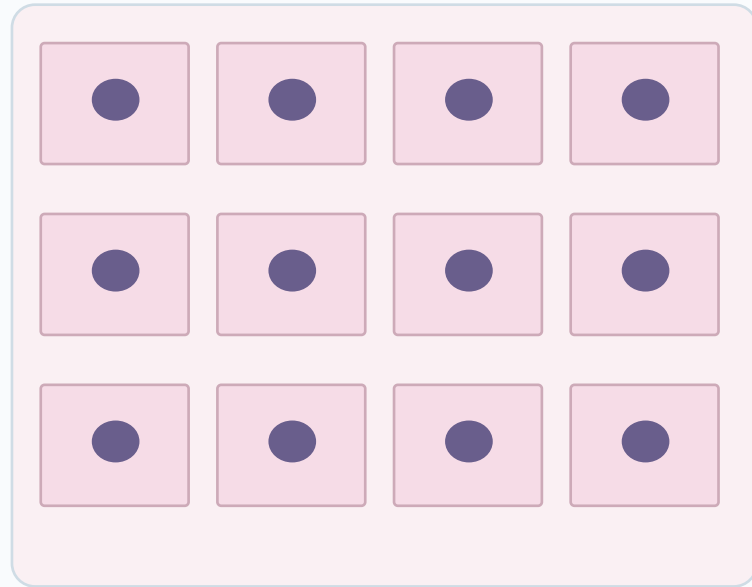
Kidney infarct: Conceptual gross map

Schematic gross section — not a specimen photograph

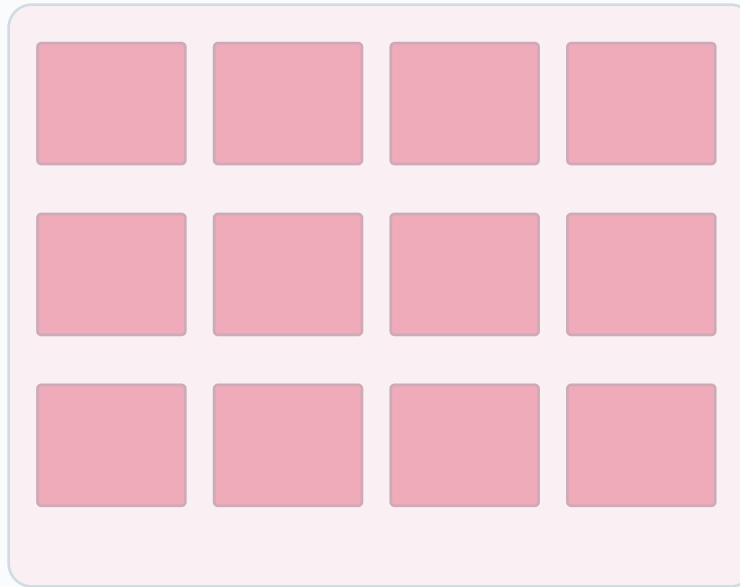


Ischemia in many solid organs → coagulative necrosis

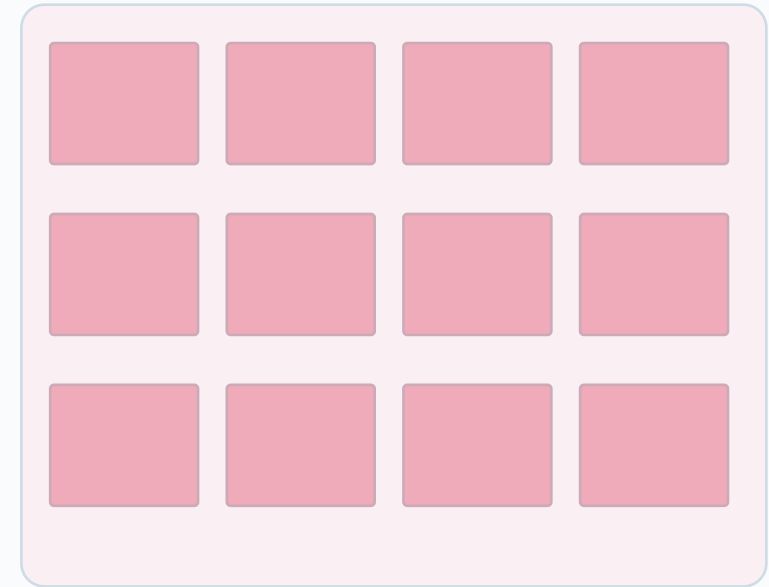
Coagulative necrosis at tissue scale



Viable parenchyma



Necrotic ghost outlines



Preserved overall framework

Schematic H&E-like fields: Not a photomicrograph; Tissue architecture is illustrated conceptually.

Histologic clues to coagulative injury

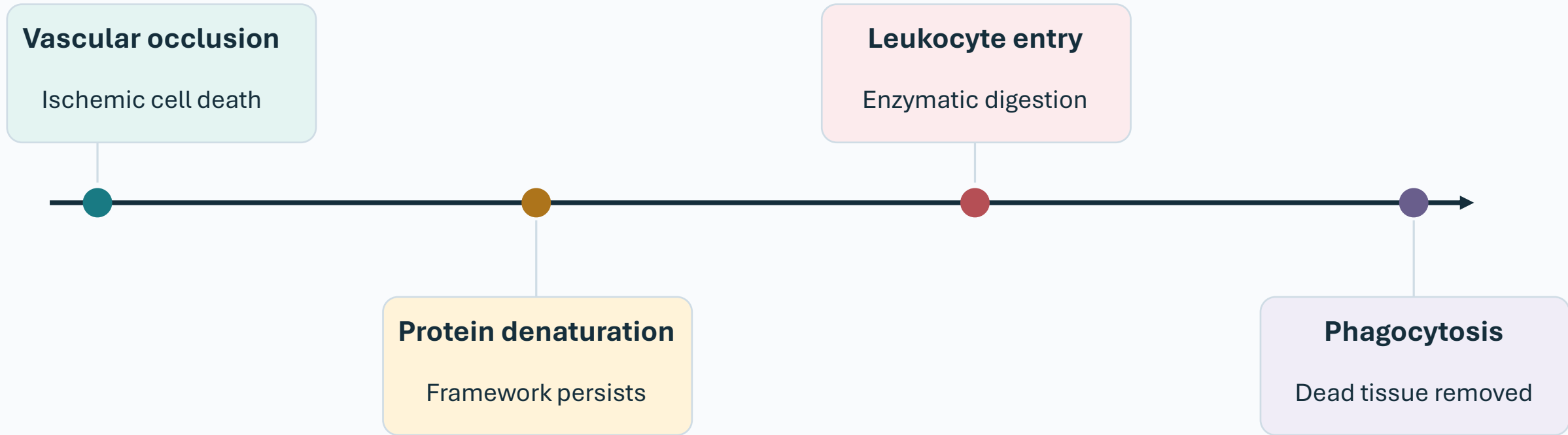
- Tubules or cardiac fibers can remain anatomically recognizable despite cell death.
- Cytoplasm becomes more intensely eosinophilic.
- Nuclei are lost or show necrotic alterations.
- The necrotic territory can recruit an inflammatory infiltrate at its boundary.
- A section at one time point cannot establish the exact onset of ischemia.

Ischemia usually produces coagulative necrosis

Vessel obstruction typically produces coagulative necrosis in solid organs other than the brain.

- Localized ischemic tissue necrosis is termed an infarct.
- The anatomic distribution and perfusion territory support vascular causation.
- The CNS is the principal textbook exception described in this section.

Coagulative morphology evolves during clearance



The sequence is schematic; Exact timing varies with organ, reperfusion, and the host response.

Firm texture: What the gross appearance tells us

Gross impression

- Firm tissue with preserved structural continuity.
- In an infarct, a localized area may appear pale.
- Boundaries may become more apparent with inflammatory response.

Microscopic confirmation

- Cell outlines remain discernible.
- Nuclei fade, fragment, or disappear.
- Inflammatory cells clear dying tissue over time.

A key distinction: Infarct is not synonymous with liquefaction

Term	What it denotes	What it does NOT establish
Infarct	Localized ischemic necrosis	A single universal necrosis pattern
Coagulative	Tissue outline preservation	Specific organ or vascular lesion alone
Liquefactive	Dead-tissue digestion	Necessarily an infectious source

Case: Renal arterial obstruction

A wedge-shaped pale lesion is found in the kidney supplied by an occluded arterial branch. Histology shows intact tubular outlines, markedly eosinophilic cytoplasm, and fading nuclei.

Which pattern best fits the reported architecture?

- Coagulative necrosis.
- Liquefactive necrosis.

Renal infarct: Interpretation

Coagulative necrosis is the expected renal infarct pattern.

- The decisive clue is preservation of tubular architecture despite dead cells.
- The wedge-shaped vascular distribution supports an ischemic infarct.
- The lesion is not defined solely by the presence of eosinophilic cytoplasm.

Liquefactive necrosis: Definition

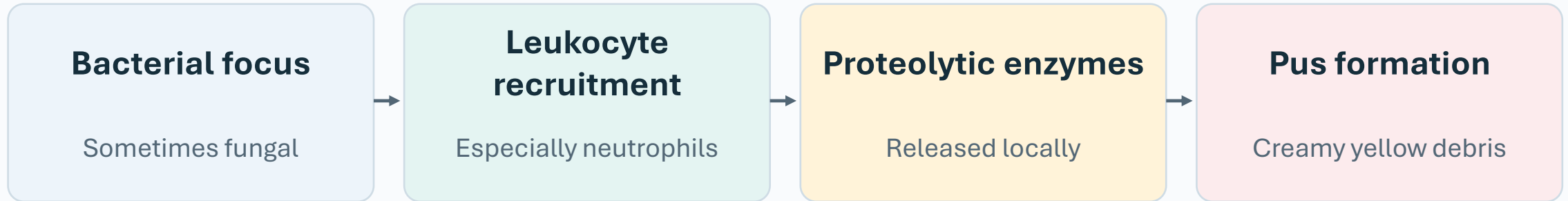
Enzymatic digestion converts dead tissue into a viscous liquid mass.

- Cell and tissue outlines are lost.
- The mechanism contrasts with persistence of architecture in coagulative necrosis.
- Typical settings are suppurative infections and CNS ischemic injury.

How digestion predominates

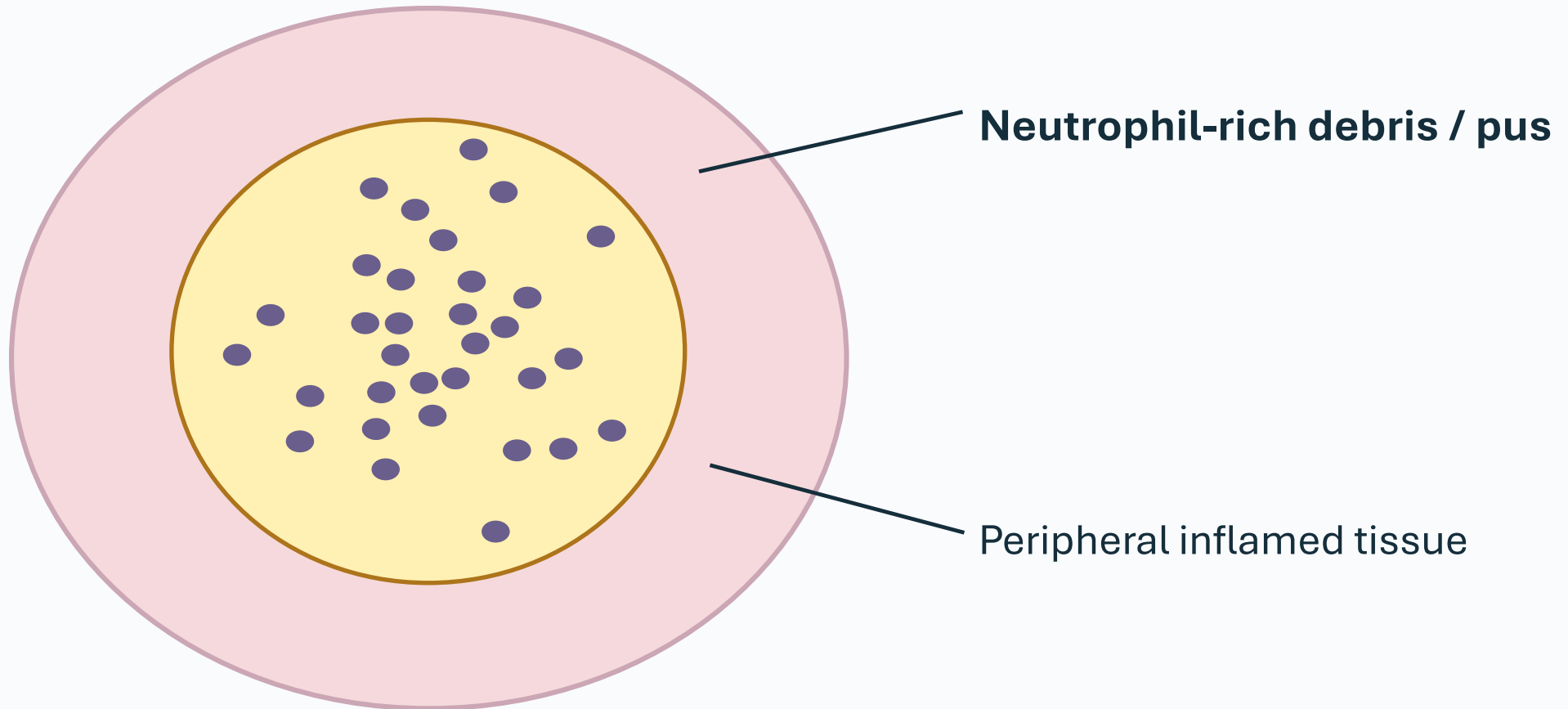


Suppurative infection: A focused mechanism



An abscess is an example of liquefaction

Schematic infectious abscess — not clinical microscopy



Bacterial (and some fungal) infections recruit leukocytes and degradative enzymes.

Why pus often appears yellow

Leukocytes

Large numbers of inflammatory cells and their breakdown products contribute to creamy yellow material.

Digested tissue

Cellular remnants lose original tissue organization.

Variable microbial burden

The finding is associated with infection but is not an organism identification method.

Diagnostic consequence

Microbiology and site-specific evaluation determine the cause rather than color alone.

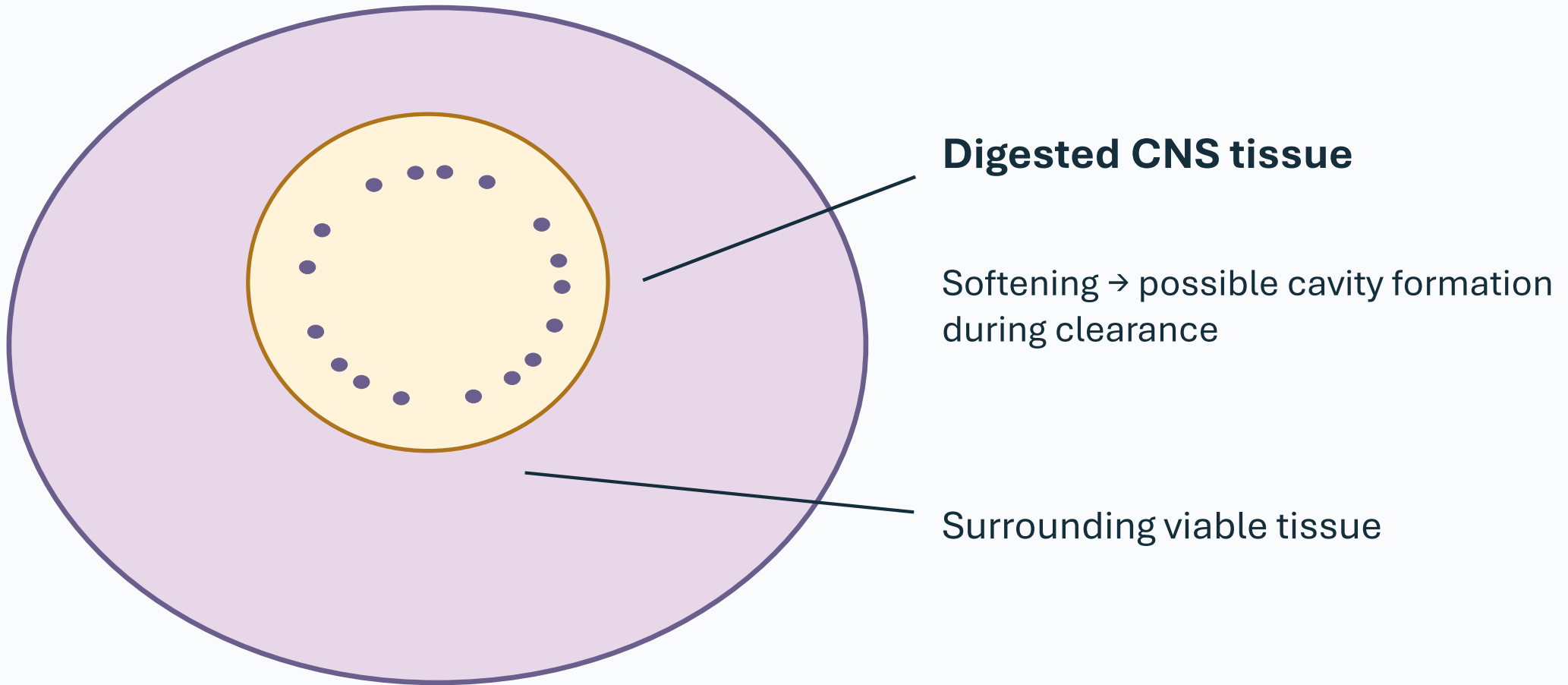
Liquefaction is not synonymous with infection

Hypoxic cell death in the CNS commonly becomes liquefactive, without requiring microbes.

- The reason for the CNS preference is not fully understood.
- The injured CNS region can soften as tissue undergoes digestion.
- Clinical correlation and imaging distinguish ischemia from infection.

Cerebral infarct: Conceptual progression

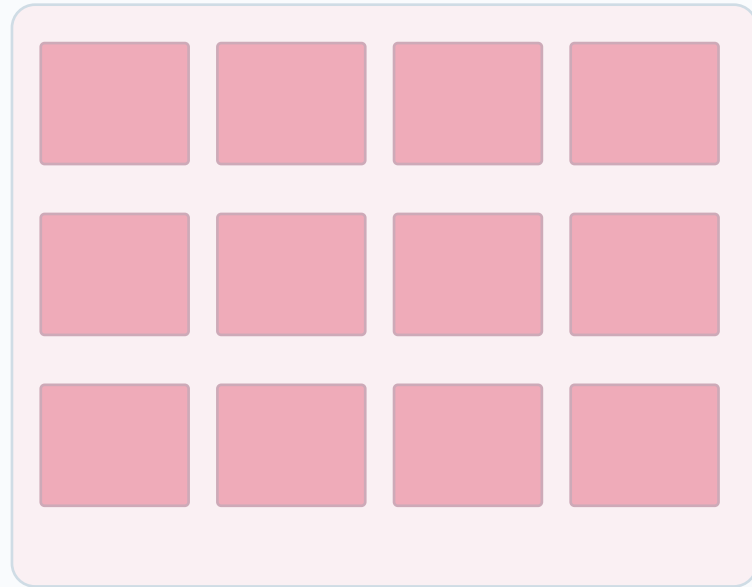
Schematic CNS infarct evolution — not radiology or gross pathology



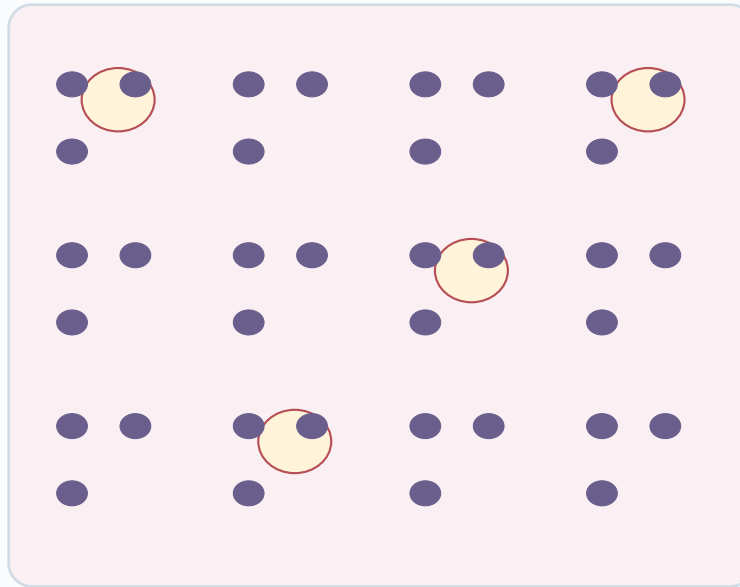
Same insult, different organ morphologies

Organ / setting	Typical necrosis pattern	Interpretive emphasis
Kidney, blocked artery	Coagulative	Tubules retain ghost outlines
Heart, blocked artery	Coagulative	Myocyte outlines persist initially
Brain, ischemic injury	Liquefactive	Dissolution / softening of tissue
Bacterial abscess	Liquefactive	Digestive enzymes and pus

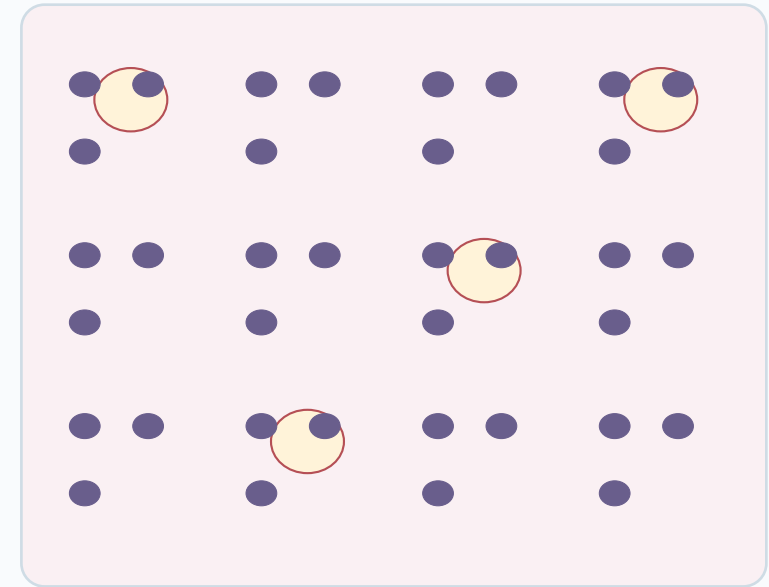
Microscopy: Dissolved versus preserved framework



Coagulative reference



Digestive destruction



Leukocyte-rich debris

Schematic H&E-like fields: Not a photomicrograph; Tissue architecture is illustrated conceptually.

CNS infarct versus abscess

Ischemic CNS necrosis

- Hypoxic death in central nervous system.
- Liquefaction can follow without infection.
- Organ context and vascular distribution matter.

Suppurative necrosis

- Bacterial and occasional fungal foci.
- Leukocytes release digestive enzymes.
- Pus may accumulate in the lesion.

Case: Brain tissue softening

A patient develops an ischemic cerebral infarct. At pathologic examination, the affected area is softened, with dissolution of the dead cerebral tissue.

What pattern is expected in the central nervous system?

- Liquefactive necrosis.
- Coagulative necrosis as in a renal infarct.

Cerebral infarct: Interpretation

Liquefactive necrosis is typical of ischemic CNS tissue death.

- A necrotic cerebral focus commonly loses tissue architecture by digestion.
- The liquefactive appearance does not itself mean that bacteria are present.
- Robbins explicitly identifies the CNS as an exception to the usual ischemic coagulative pattern.

Gangrenous necrosis: A clinical label

Gangrene is not a unique microscopic form of cell death.

- It usually describes ischemic necrosis affecting a limb or a similar anatomical part.
- Underlying injury often shows coagulative necrosis across several tissue planes.
- Superinfection can add prominent liquefaction: Wet gangrene.

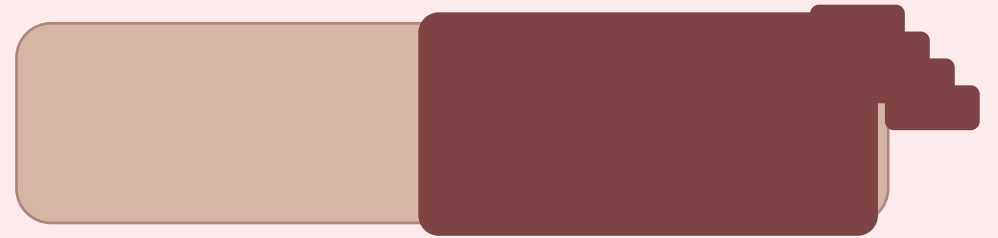
The anatomy behind the term

Clinical designation, not a distinct microscopic mechanism



Dry gangrene

Loss of blood supply

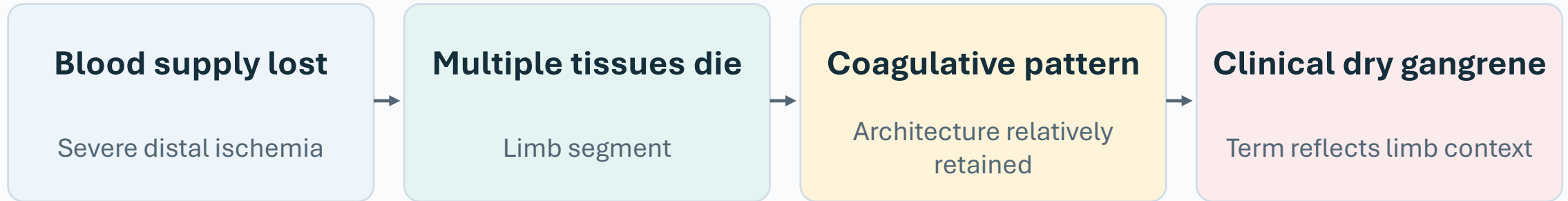


Wet gangrene

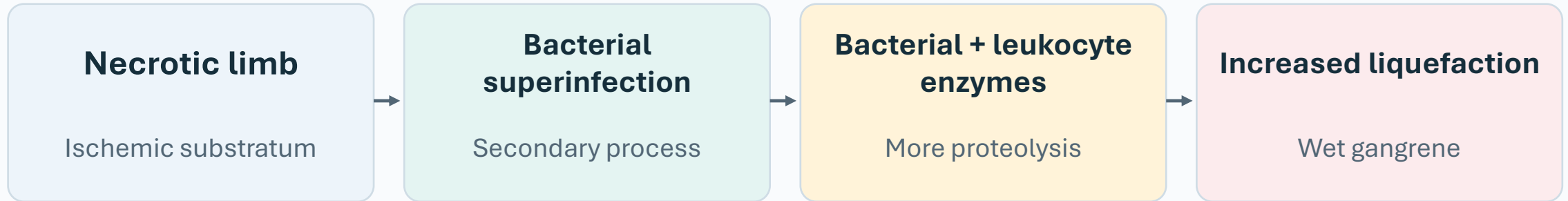
Ischemic death + bacterial infection

Schematic distal limb segment; No patient photograph.

Dry gangrene: Ischemia predominates



Wet gangrene: Infection changes the picture



Dry versus wet gangrene

Feature	Dry gangrene	Wet gangrene
Principal setting	Severe ischemic tissue death	Ischemia with superimposed infection
Dominant necrotic process	Usually coagulative	Greater liquefactive component
Tissue context	Often distal limb / multiple tissue planes	Infected necrotic limb / region
Key interpretation	Perfusion failure	Perfusion failure plus infection

Gangrene is not a histologic diagnosis by itself

What the term conveys

- A clinically recognized devitalized region.
- Often an ischemic limb segment.
- Potentially includes several tissue compartments.

What needs separate assessment

- Nature and extent of vascular compromise.
- Evidence of infection or suppuration.
- Tissue viability at the margins.

Why the “wet” qualifier matters

Second mechanism

Bacterial and recruited leukocyte enzymes increase digestion beyond the ischemic lesion.

Multiple compartments

The process is not limited to one microscopic cell type.

Clinical consequence

The adjective highlights possible superinfection rather than a new fundamental death pathway.

Interpretive limit

Neither gross discoloration nor the word gangrene identifies the specific infectious organism.

Case: Necrotic lower leg with infection

An ischemic lower leg has already become necrotic. A bacterial infection supervenes and the lesion develops pronounced tissue liquefaction.

What makes the process “wet gangrene”?

- Additional bacterial and leukocytic enzymatic digestion.
- Exclusive mitochondrial apoptosis without inflammation.

Gangrene case: Interpretation

Wet gangrene adds infection-associated liquefaction to ischemic necrosis.

- The ischemic substratum commonly displays a coagulative component.
- The term describes an anatomic and clinical scenario, not a seventh pathway of cellular death.
- The possibility of infection must be assessed independently of appearance.

Caseous necrosis: Distinctive appearance

Caseous necrosis produces friable, white, cheeselike necrotic material.

- Classically associated with tuberculous infection.
- Microscopy shows amorphous granular debris with loss of recognizable tissue structures.
- The necrosis may be surrounded by epithelioid histiocytes in a granuloma.

Gross caseation: Descriptive clues

White to yellow-white

The pale color is a gross descriptive cue rather than a specific chemical test.

Friable / crumbly

The surface differs from the firm architecture of coagulative necrosis.

Loss of recognizable tissue

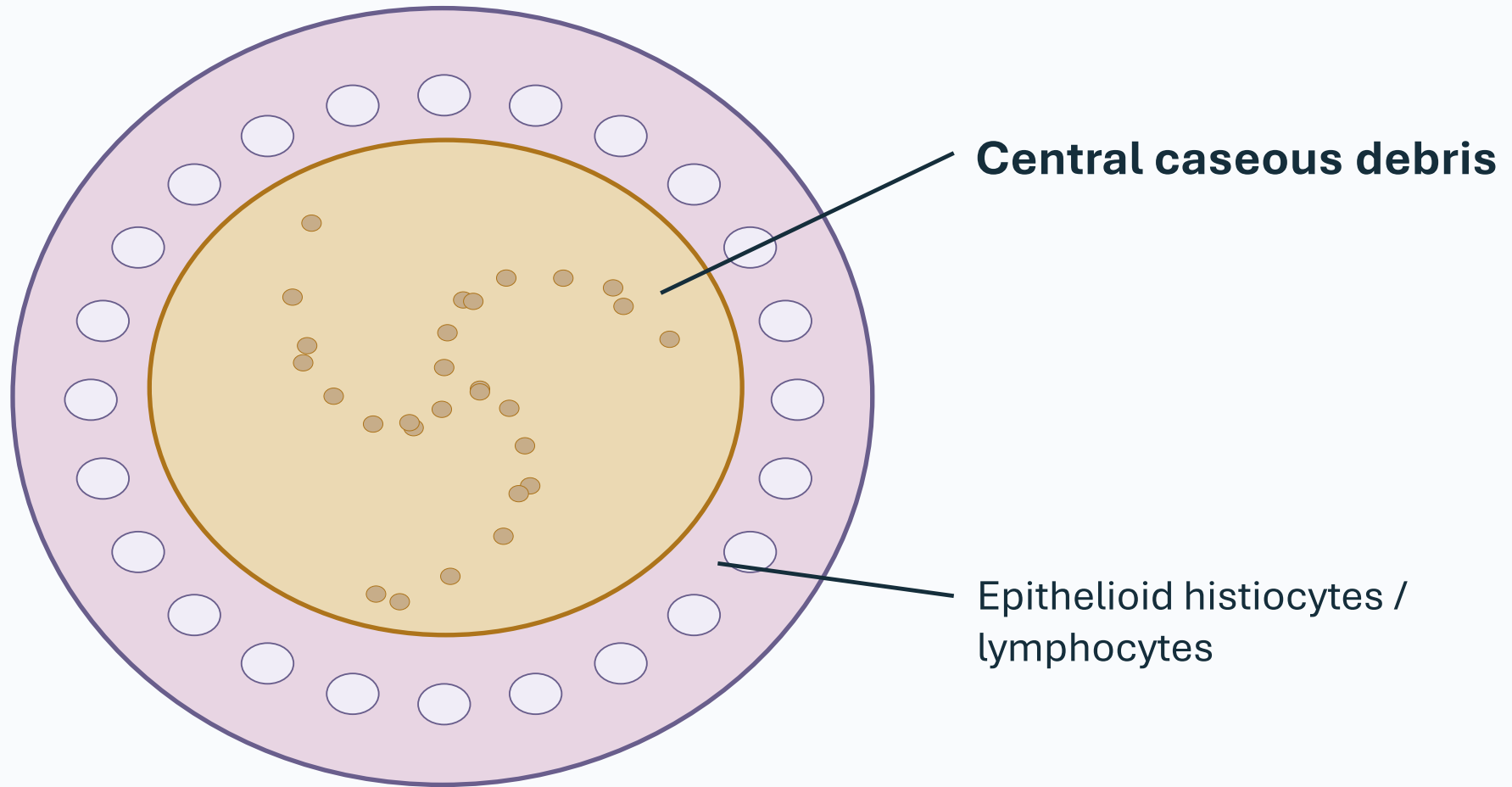
Destroyed cells merge into a granular necrotic mass.

Context

A necrotizing granuloma supports an infectious differential, especially tuberculosis.

A caseating granuloma: Architecture

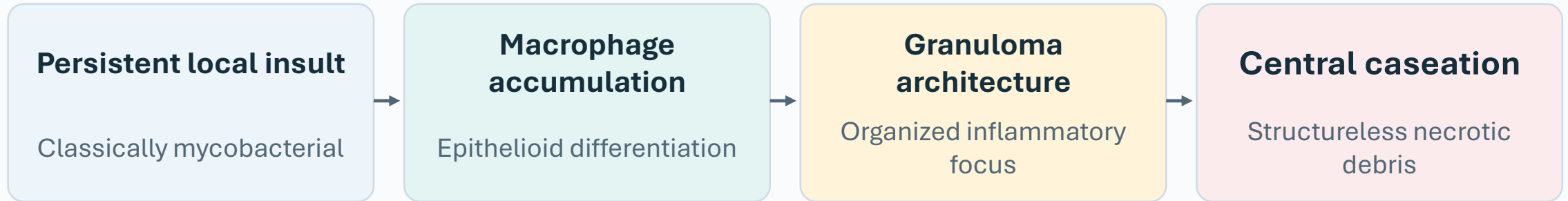
Schematic caseating granuloma — not a histologic section



Caseous necrosis under the microscope

- The center is structureless and composed of fragmented or lysed cells.
- Amorphous granular eosinophilic necrotic debris replaces recognizable structures.
- A surrounding zone of epithelioid histiocytes indicates granulomatous inflammation.
- Ancillary organism testing may be required to determine the etiology.

Why a granuloma matters



Caseation is a clue, not microbiologic proof

Morphology can support

- Necrotizing granulomatous inflammation.
- A tuberculosis-centered differential in an appropriate setting.
- Selection of microbiologic / special-stain studies.

Morphology alone cannot confirm

- Species of causative organism.
- Active infection status and viable organisms.
- A single etiologic diagnosis in every case.

Caseous versus coagulative necrosis

Feature	Caseous	Coagulative
Gross	White, friable, cheeselike	Firm, organized tissue
Microscopic architecture	Amorphous granular debris	Ghost cellular outlines persist
Typical context	Tuberculous granuloma	Solid-organ ischemia
Inflammatory organization	Epithelioid granulomatous rim	Leukocyte clearance of dead tissue

Case: Lung lesion with granulomatous rim

A pulmonary focus contains soft, friable white necrotic material. Histology shows amorphous granular debris circumscribed by epithelioid histiocytes.

What pattern is present, and what association must be considered?

- Caseous necrosis; Tuberculosis is an important possibility.
- Coagulative necrosis; Isolated arterial occlusion is obligatory.

Caseating lung lesion: Interpretation

Caseous necrosis in a granulomatous inflammatory setting.

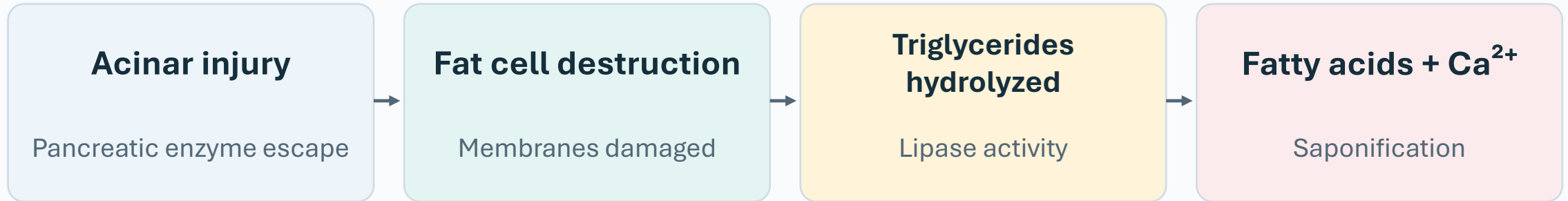
- Friable gross debris and amorphous microscopic necrosis support the pattern.
- Tuberculosis is a classic association.
- Additional studies establish the organism; This morphology is not a standalone microbiologic diagnosis.

Fat necrosis: Destruction of adipose tissue

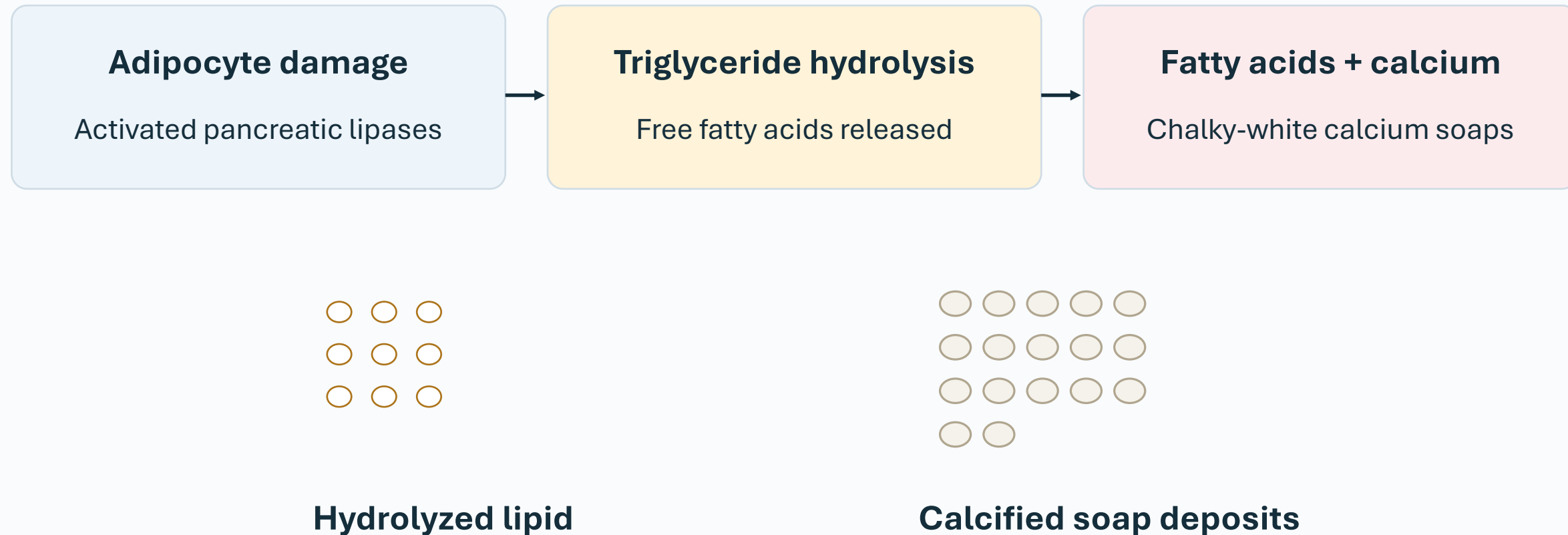
Activated pancreatic lipases can destroy fat around the pancreas and in the peritoneal cavity.

- Damaged pancreatic acinar cells permit enzyme leakage.
- Triglycerides are hydrolyzed to fatty acids.
- Released fatty acids bind calcium to produce grossly visible chalky deposits.

Pancreatic enzyme escape: The sequence



The chemistry of fat saponification



Schematic pathway: Calcium deposition occurs in damaged fat, not in simple fatty change.

Fat necrosis: Key gross characteristic

Chalky-white deposits

Grossly evident calcium soaps form in areas of damaged peripancreatic / peritoneal fat.

Localized fat destruction

Lipase injury involves adipose structures beyond the pancreatic parenchyma.

Inflammation

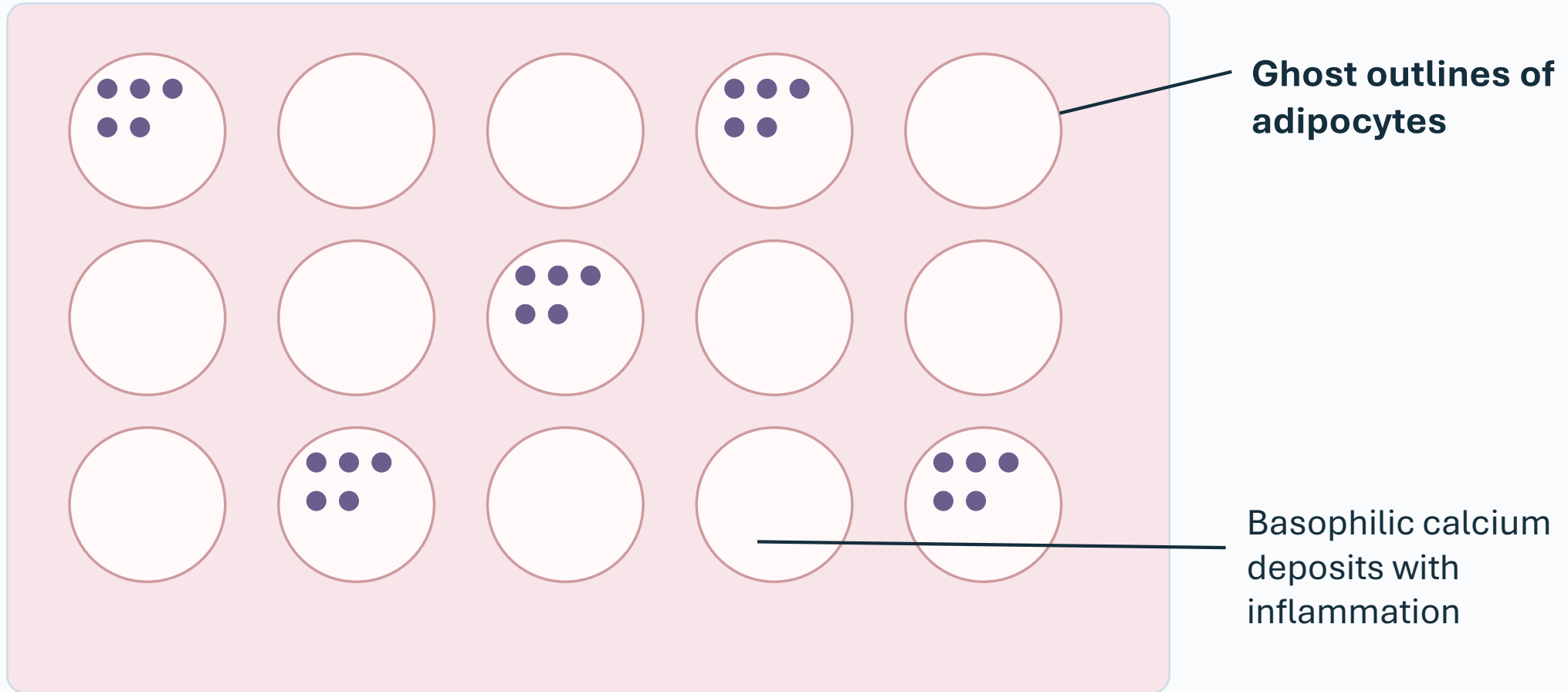
Local reactions accompany necrotic adipocyte breakdown and calcium deposition.

Clinical context

In severe acute pancreatitis, this morphology provides a conspicuous clue to enzymatic fat damage.

Fat necrosis: Histologic pattern

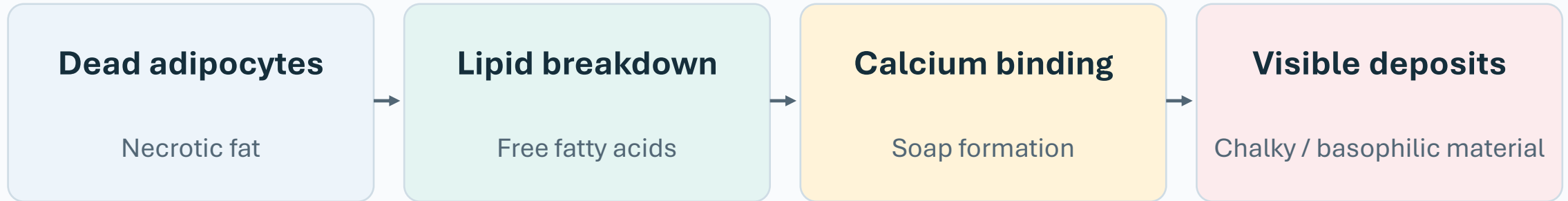
Schematic H&E fat necrosis — not a photomicrograph



The histologic triad to recognize

Finding	Interpretation
Shadowy adipocyte outlines	Necrotic fat cells remain recognizable as empty ghosts
Basophilic calcium deposits	Local calcium soap deposition after fatty-acid formation
Inflammatory reaction	Host response to fat-cell destruction

Fat necrosis and dystrophic calcification



Fat necrosis $\neq$ fatty change

Fat necrosis

- Destruction of fat cells.
- Release of fatty acids and calcium soap formation.
- Inflammatory reaction and necrotic adipocyte outlines.

Fatty change (steatosis)

- Triglyceride accumulation within viable parenchymal cells.
- Classically described in liver injury contexts.
- Does not by itself imply fat-cell death or saponification.

Enzymatic fat necrosis is a tissue clue

- Recognize that the damaged substrate is fat, not just pancreatic acinar tissue.
- Connect fat-cell membranes, triglycerides, released fatty acids, and calcium soaps.
- Interpret basophilic calcific deposits with adipocyte ghost outlines.
- Avoid confusing small lipid vacuoles in viable hepatocytes with dead adipose tissue.

Case: Acute pancreatitis and mesenteric chalk

During surgery for severe acute pancreatitis, multiple chalky-white foci are observed in peritoneal fat. Histology shows outlines of dead adipocytes and basophilic deposits.

Which pathogenesis best accounts for the finding?

- Pancreatic lipase-mediated fat injury with calcium saponification.
- A purely reversible intracellular triglyceride accumulation.

Pancreatic fat necrosis: Interpretation

Enzymatic fat necrosis with calcium soap formation.

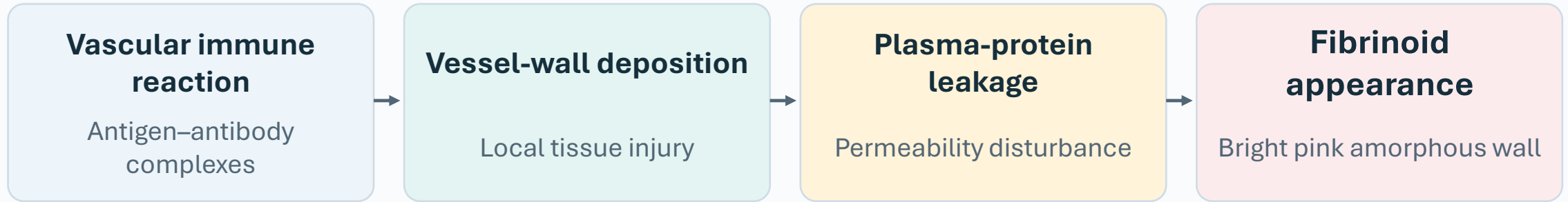
- Activated pancreatic lipases hydrolyze adipocyte triglycerides.
- Free fatty acids combine with calcium to create chalky deposits.
- Adipocyte shadows plus basophilic calcium are characteristic histologic clues.

Fibrinoid necrosis: Vascular-wall damage

Fibrinoid necrosis describes a striking bright-pink, amorphous lesion of vessel walls.

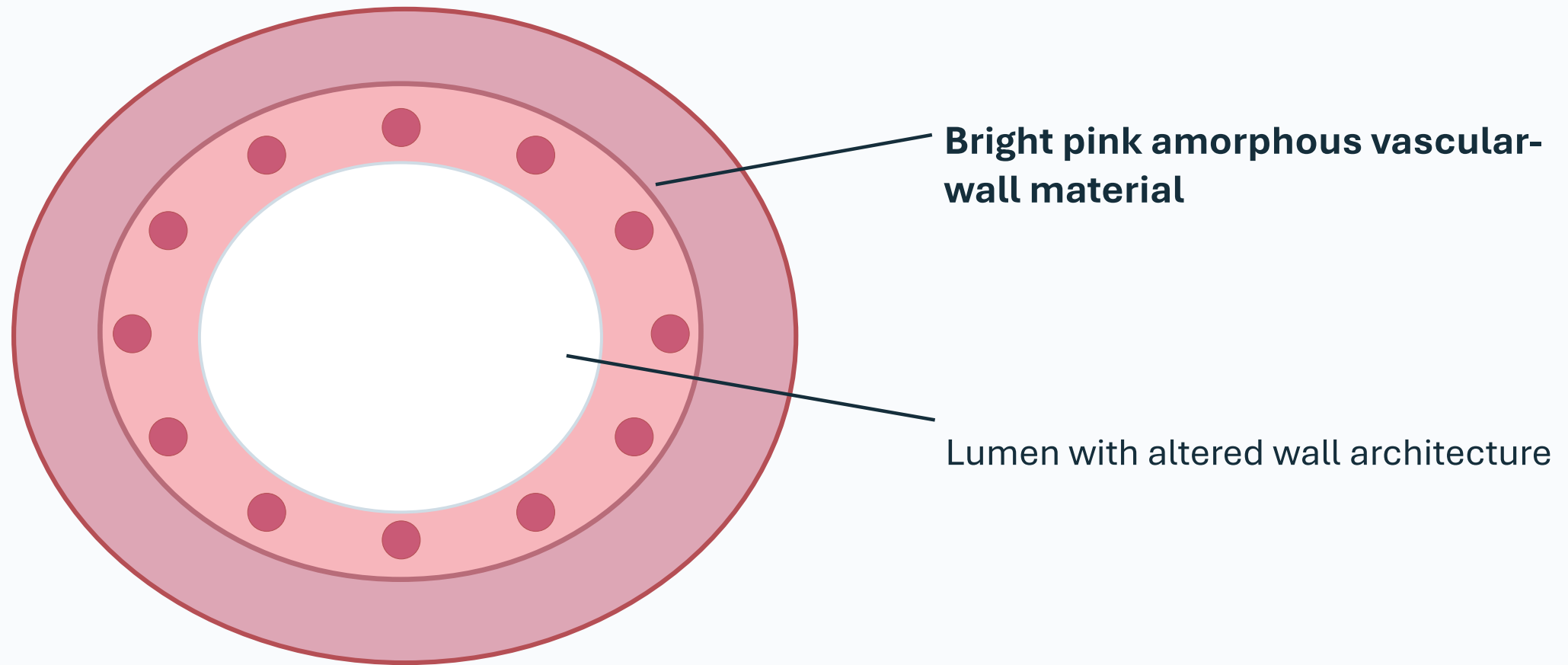
- Vascular immune reactions as a typical setting.
- Antigen–antibody complexes may accumulate in arterial walls.
- Leaked plasma proteins contribute to fibrin-like eosinophilic material.

Immune-mediated vascular injury



Where to look: Vessel wall, not parenchymal core

Schematic vessel-wall injury — not a histologic micrograph



Actual fibrinoid necrosis is interpreted with vessel injury and clinical context.

Fibrinoid necrosis at H&E

- Examine injured vessels for intensely eosinophilic material involving the wall.
- The material is amorphous, with a fibrin-like microscopic appearance.
- Immune deposits and leaked plasma proteins contribute to this appearance.
- The pattern belongs to vascular damage and requires relevant clinical and histologic context.

Why “fibrinoid” is a morphologic term

Descriptive resemblance

“Fibrinoid” refers to a bright pink, fibrin-like H&E appearance.

Not a molecular assay

The term does not by itself demonstrate pure fibrin as the only constituent.

Anatomical location

Interpretation depends on the material being in damaged vessel walls.

Causal possibilities

Immune-complex reactions as a common context.

Fibrinoid versus other eosinophilic necrosis

Pattern	Dominant location	Recognition clue
Fibrinoid	Vessel wall	Amorphous bright pink, fibrin-like material
Coagulative	Parenchymal infarct	Preserved dead tissue framework
Caseous	Necrotizing granuloma	Granular acellular center + epithelioid rim

Case: Immune injury in an artery

A small artery affected by an immune-mediated disorder has a circumferential, bright-pink amorphous lesion of its wall, with local inflammation.

Which descriptive type of necrosis is most appropriate?

- Fibrinoid necrosis.
- Fat necrosis with pancreatic saponification.

Fibrinoid vascular lesion: Interpretation

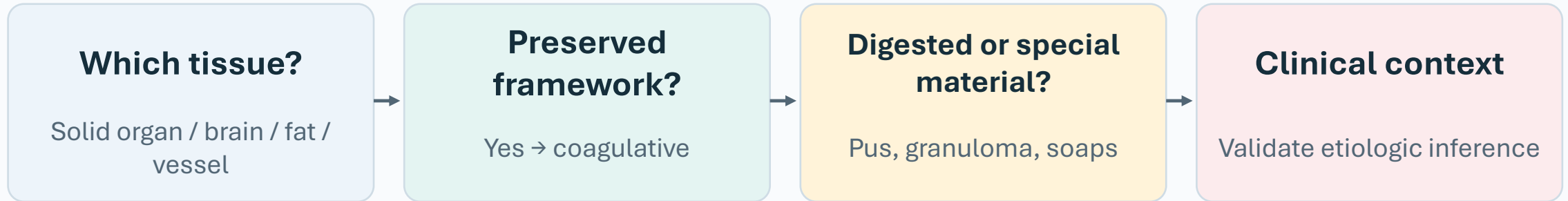
Fibrinoid necrosis describes the eosinophilic vascular-wall damage.

- The crucial compartment is the wall of the affected artery.
- Immune-complex deposition and plasma-protein escape are common associated processes.
- Etiologic diagnosis still requires broader vascular and clinical assessment.

All six patterns: Morphology and setting

Pattern	Morphologic hallmark	Classic example
Coagulative	Firm; Architecture retained	Renal / myocardial infarct
Liquefactive	Digested; Viscous debris	Cerebral infarct or abscess
Gangrenous	Necrotic limb; $\pm$ infection	Ischemic lower limb
Caseous	Friable debris in granuloma	Tuberculosis
Fat	Adipocyte ghosts; Calcium soaps	Acute pancreatitis
Fibrinoid	Pink amorphous vessel wall	Immune vascular injury

Pattern recognition: Organ, then architecture



A pattern is not proof of one cause

Coagulative → think ischemia

Check vascular distribution and organ findings;
Architecture alone does not establish a particular artery.

Liquefactive → infection OR CNS ischemia

Do not equate liquefaction automatically with a bacterial process.

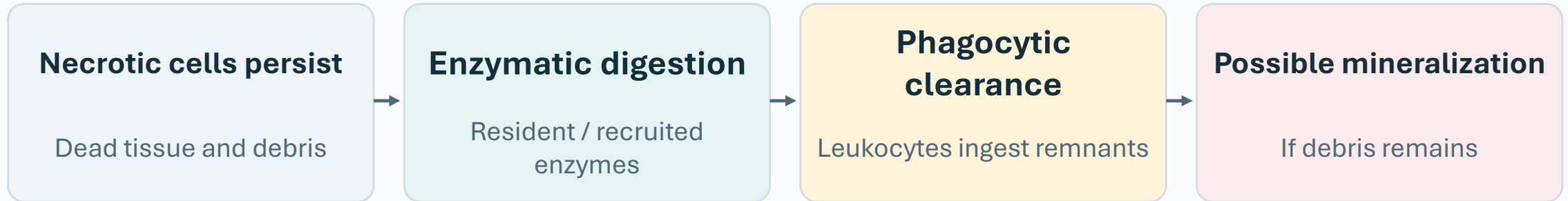
Caseous → consider tuberculosis

The granulomatous necrosis guides testing but does not independently identify the organism.

Fibrinoid → assess vascular injury

Evaluate immune and other vessel-wall insults in context.

What happens to necrotic debris?



Dystrophic calcification: An endpoint, not a type

Early injury signature

- Coagulative, liquefactive, and other patterns describe dominant morphology.
- Their causes vary with organ and insult.
- Debris is frequently cleared by host cells.

If necrotic remnants persist

- Calcium and other minerals may deposit.
- Deposits can follow multiple forms of dead-tissue injury.
- Fat saponification is a specific calcium soap mechanism.

Knowledge check: Tissue preservation

An infarcted kidney retains tubule outlines despite nuclear loss. The pattern is:

(A) Coagulative necrosis.

(B) Liquefactive necrosis.

(C) Caseous necrosis.

(D) Fibrinoid necrosis.

Knowledge check: Tissue preservation

Coagulative necrosis preserves outlines of dead renal tissue.

- The architecture persists because protein and enzyme denaturation impedes rapid breakdown.
- Solid-organ ischemia is the classic cause.
- The ischemic area may subsequently be cleared by leukocytes.

Knowledge check: Match the mechanism

Which finding most strongly supports pancreatic enzymatic fat necrosis?

(A) Calcium soaps and ghost adipocytes.

(B) Preserved renal tubules with vanished nuclei.

(C) Bright pink immune-damaged vessel walls.

(D) Caseating granuloma with central granular debris.

Knowledge check: Fat injury

Lipase-mediated hydrolysis and calcium soaps are characteristic.

- Fat-cell destruction releases triglycerides for enzymatic hydrolysis.
- Fatty acids bind calcium, producing chalky deposits.
- Histology shows necrotic adipocyte outlines and basophilic calcium.

Summary: Six patterns, three interpretive rules

- Architecture retained → think coagulative; Tissue liquefied → think enzymatic digestion.
- CNS ischemia and suppurative infection are different causes of liquefactive necrosis.
- Gangrene is a clinical label; Wet gangrene adds infection-associated liquefaction.
- Caseous granulomas, chalky fat necrosis, and fibrinoid vessel injury have distinctive tissue compartments.
- Use morphology to generate and prioritize diagnoses—not to replace etiologic testing.

Textbook source and visual provenance

- Robbins, Cotran & Kumar: Pathologic Basis of Disease. 11th ed. (2026), Chapter 2, “Patterns of Tissue Necrosis,” pp. 38–40.
- Included adjacent cell-level morphologic material (“Morphology,” pp. 38–39).
- Six-pattern description, organ examples, and calcium deposition concepts follow the primary Robbins excerpt.
- All displayed pathology-like tissue drawings are original, explicitly labeled schematics; they are NOT photomicrographs.
- No copyrighted Robbins figure or other externally sourced histologic specimen is reproduced.