

Osteonecrosis

(Avascular Necrosis)

Etiology • Morphology • Clinicopathologic consequences

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Learning objectives

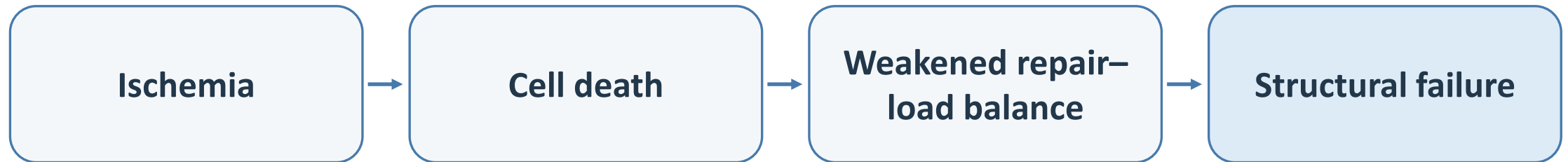
- Define bone infarction and identify its two principal anatomic patterns.
- Explain the routes from ischemia to cell death, repair, collapse, and arthritis.
- Correlate morphology with pain, imaging, staging, and management decisions.

Osteonecrosis is infarction of bone and marrow

Bone infarction = death of bone and marrow after loss of perfusion

- Vascular insufficiency causes ischemic necrosis of osseous tissue and its marrow.
- The lesion may occupy trabecular marrow spaces or lie directly beneath a joint surface.
- The anatomic location determines much of the clinical course.

Why a bone infarct is mechanically important



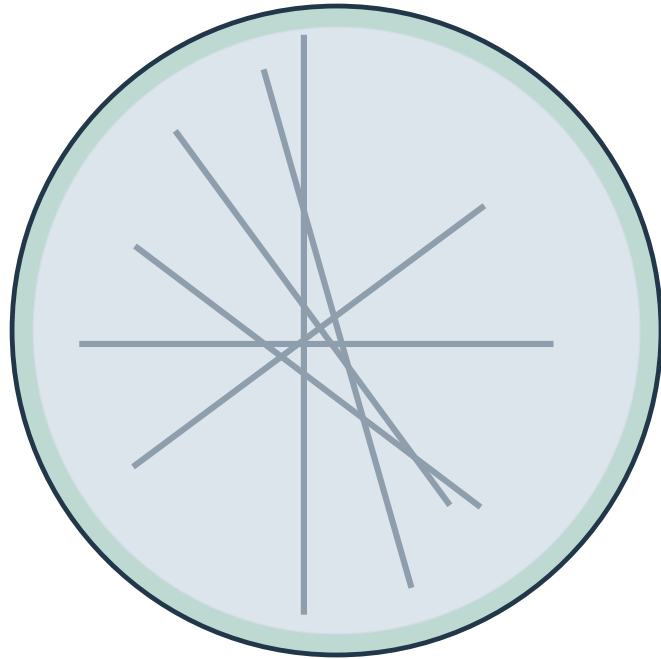
Conceptual causal pathway

Viability and structural integrity are different properties of bone.

Two osteonecrosis patterns

Feature	Medullary infarct	Subchondral infarct
Site	Trabecular bone and marrow	Immediately beneath articular cartilage
Typical structural outcome	Cortex spared	Risk of surface collapse
Clinical tendency	Often silent	Often painful

Anatomic framework of the femoral head

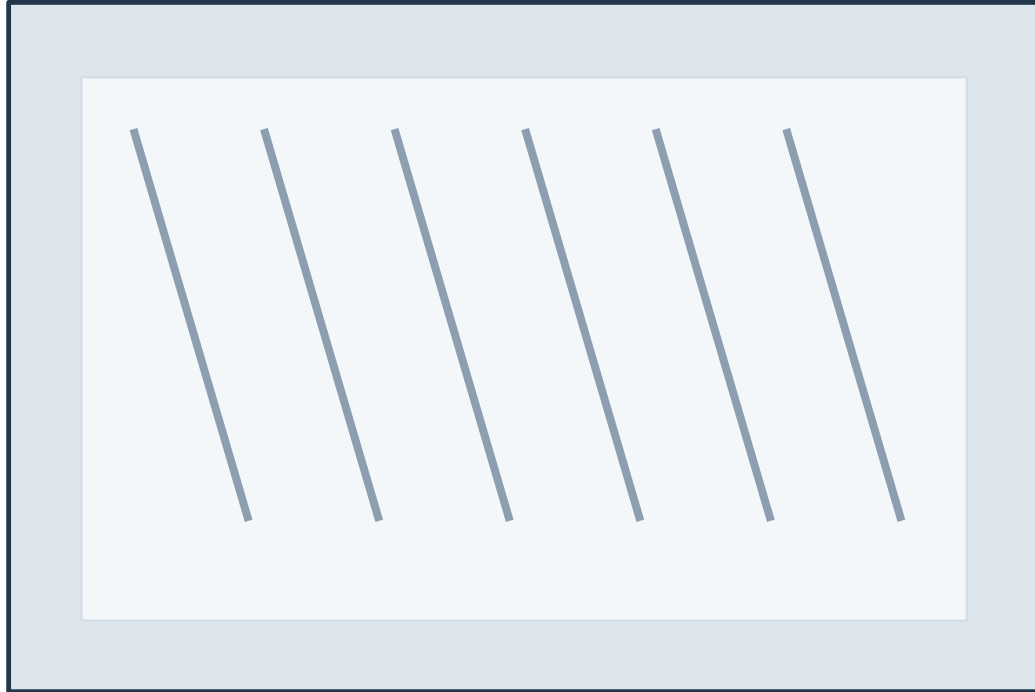


Articular cartilage

Subchondral trabeculae

Cancellous marrow

Perfusion and collateral protection differ by compartment



Compartment schematic

Cortical shell

Collateral periosteal circulation

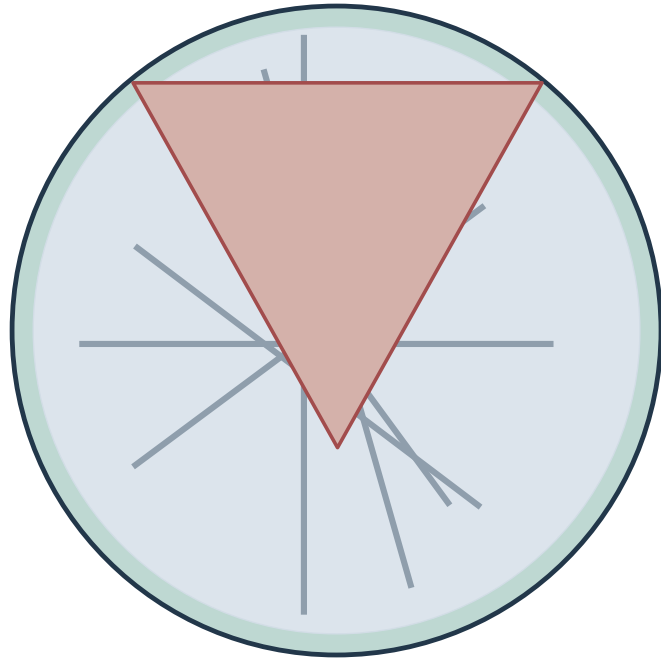
Trabecular marrow

Local intraosseous vascular supply

Ischemic territory

Marrow necrosis with cortex spared

Cartilage can survive while subchondral bone dies



Cartilage remains nourished

Bone below may be necrotic

Subchondral support is vulnerable

Risk factors converge on impaired bone perfusion

LOCAL VASCULAR

Trauma / fracture

Vasculitis

DRUGS / THERAPY

Corticosteroids

Radiation

SYSTEMIC

Sickle cell crisis

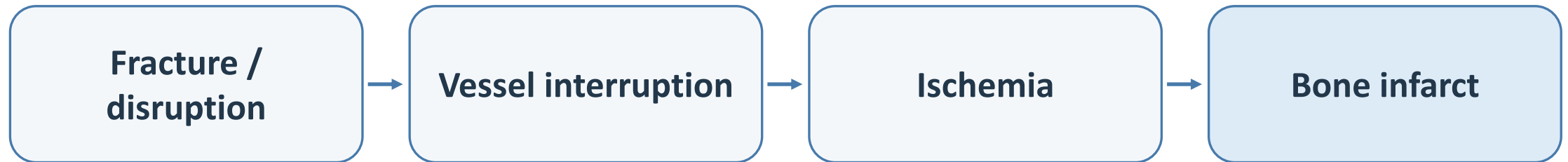
Vascular compromise

UNASSIGNED

No known cause

Idiopathic group

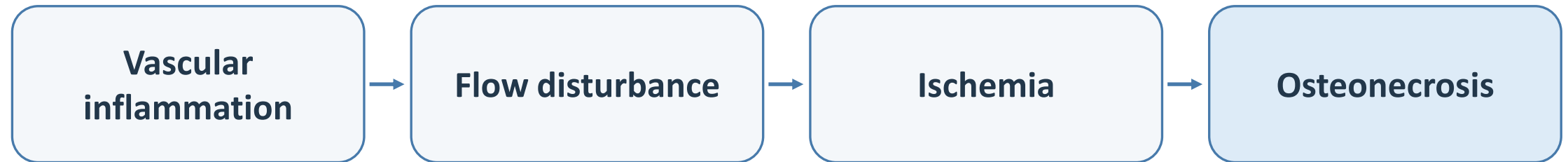
Trauma: Mechanical interruption of vessels



Conceptual causal pathway

A fracture can directly disrupt osseous vessels.

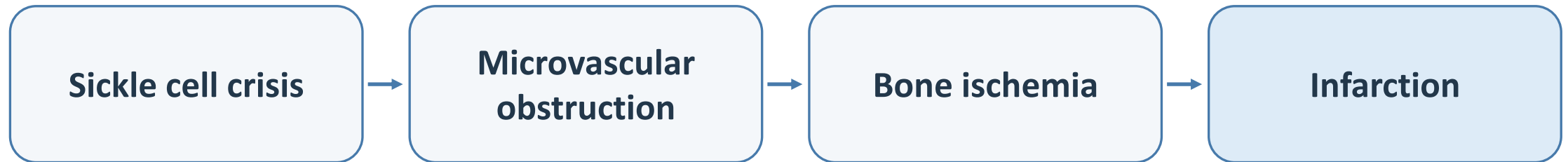
Vasculitis: Vascular injury without a fracture



Conceptual causal pathway

Inflammatory damage can compromise the lumen or vessel wall.

Sickle cell disease: A systemic ischemic setting



Conceptual causal pathway

Sickle cell crises are recognized predisposing conditions.

Corticosteroids: An important nontraumatic association

- Corticosteroid exposure is included among drug-related risk factors.
- A clear fracture history is not required for an ischemic lesion.
- The mechanism of glucocorticoid-associated osteonecrosis is not fully established.

Radiation: Therapy-associated vascular vulnerability

- Radiation is listed among conditions predisposing to bone ischemia.
- Prior irradiation should be considered in the exposure history.
- The morphologic endpoint can resemble infarcts arising from other causes.

Idiopathic osteonecrosis

No identified cause $\neq$ no ischemic mechanism

- Some infarcts have no recognized predisposing cause.
- The textbook estimates that approximately one quarter are designated idiopathic.
- “Idiopathic” describes the limits of etiologic attribution—not a different morphology.

Osteonecrosis of the jaw: Separate clinical context

- The Robbins section notes a rare jaw complication of prolonged bisphosphonate treatment.
- Medication-related jaw osteonecrosis should not be equated automatically with classic femoral-head infarction.
- Its diagnostic framework is outside the present bone-infarct morphology sequence.

Etiologic classification: Known versus unknown

Pathway	Recognized factor	Shared endpoint
Local vascular	Fracture; Vasculitis	Reduced perfusion
Systemic / exposure	Steroids; Sickle cell; Radiation	Reduced perfusion
Unassigned	Idiopathic	Bone infarction

Three routes to an ischemic infarct

Mechanical disruption

Vessel cut / disrupted

Thrombotic occlusion

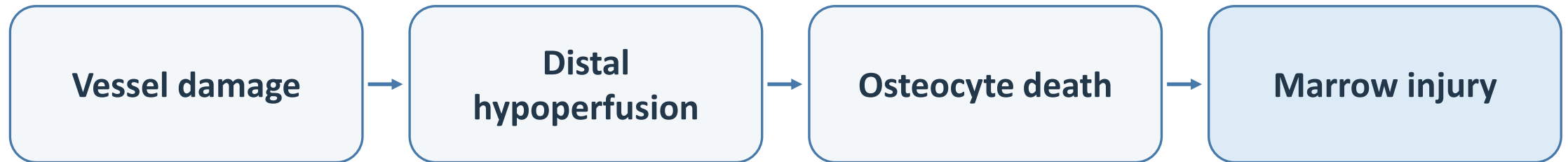
Lumen obstructed

External compression

Flow impeded from outside

Tissue ischemia → bone and marrow necrosis

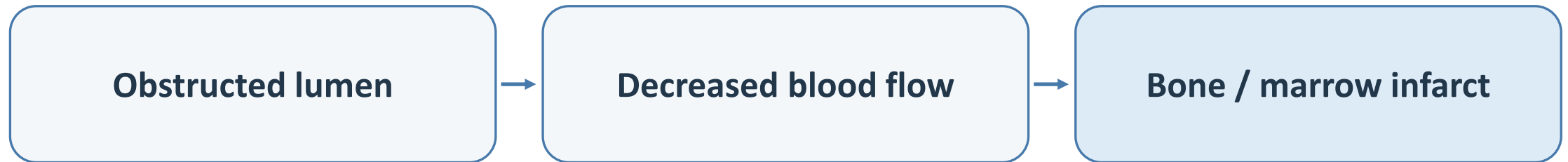
Mechanical disruption: Loss of continuity



Conceptual causal pathway

An injured vessel fails to deliver blood to downstream bone.

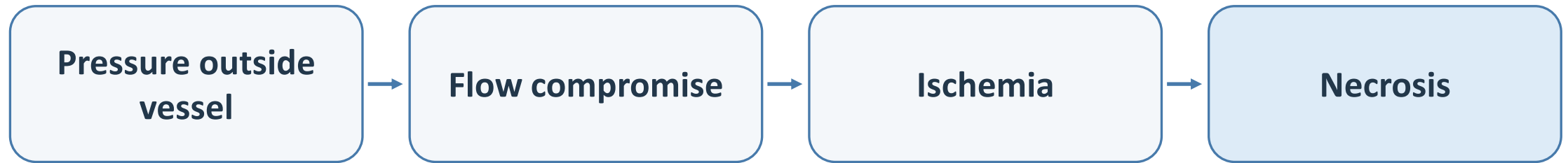
Thrombotic occlusion: Loss of luminal patency



Conceptual causal pathway

A vessel can remain anatomically intact yet become blocked.

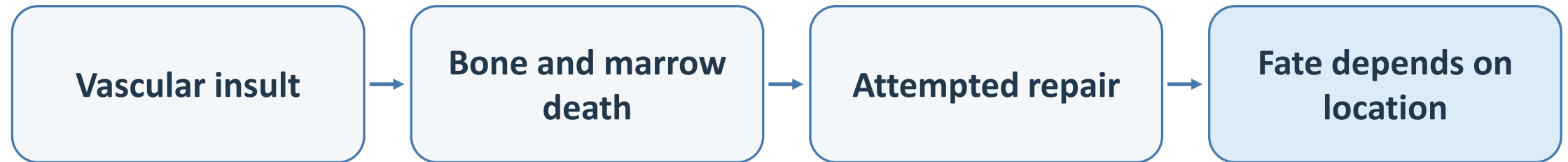
Extravascular compression: Impeded flow



Conceptual causal pathway

External pressure can compromise perfusion through a vessel.

Different causes, identical final tissue injury



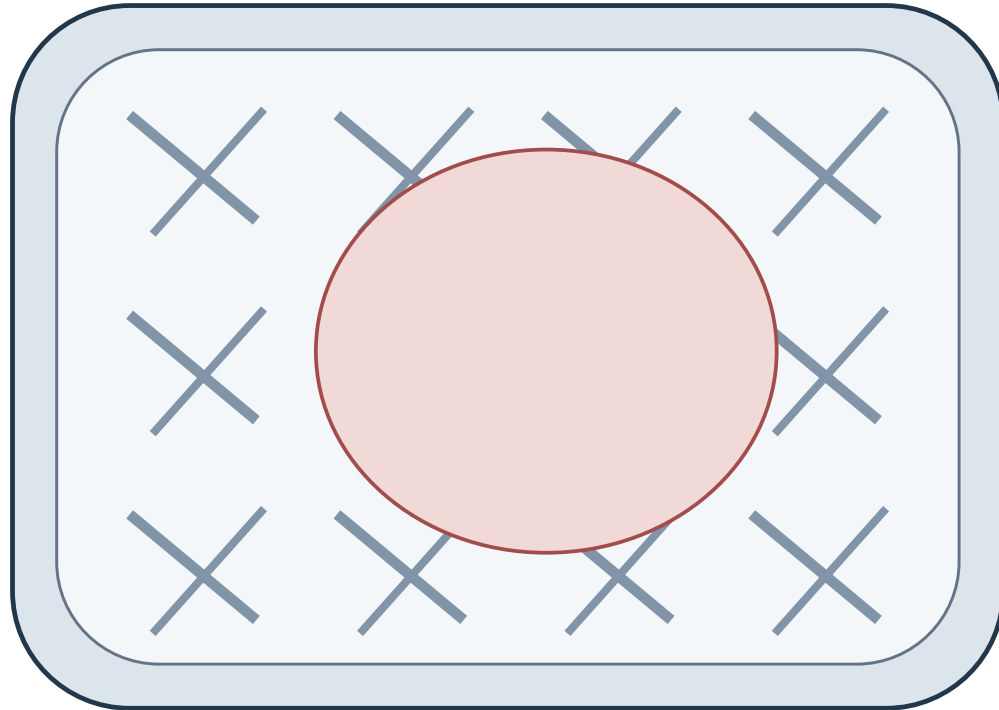
Conceptual causal pathway

Etiologic agents differ upstream of ischemia.

The biological lesion and the mechanical lesion differ

Biologic event	Structural event	Clinical result
Loss of viability	Architecture may persist	May be occult
Slow repair	Compression microfractures	Pain with loading
Failure of support	Articular surface collapse	Secondary arthritis

Medullary infarcts are geographic lesions



Schematic cross-section

Regional marrow + trabecular infarct

Preserved cortical rim

Why the cortex survives a medullary infarct



Compartment schematic

Cortical shell

Collateral periosteal circulation

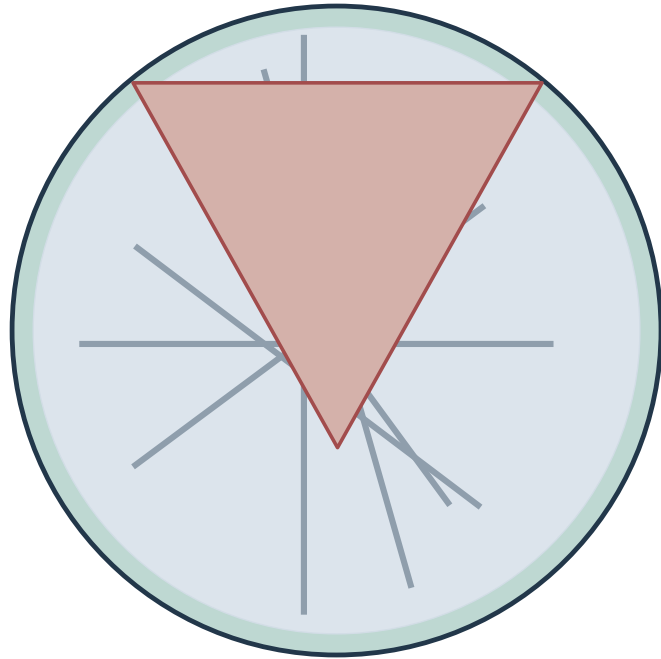
Trabecular marrow

Local intraosseous vascular supply

Ischemic territory

Marrow necrosis with cortex spared

Subchondral infarcts have a wedge-like geometry

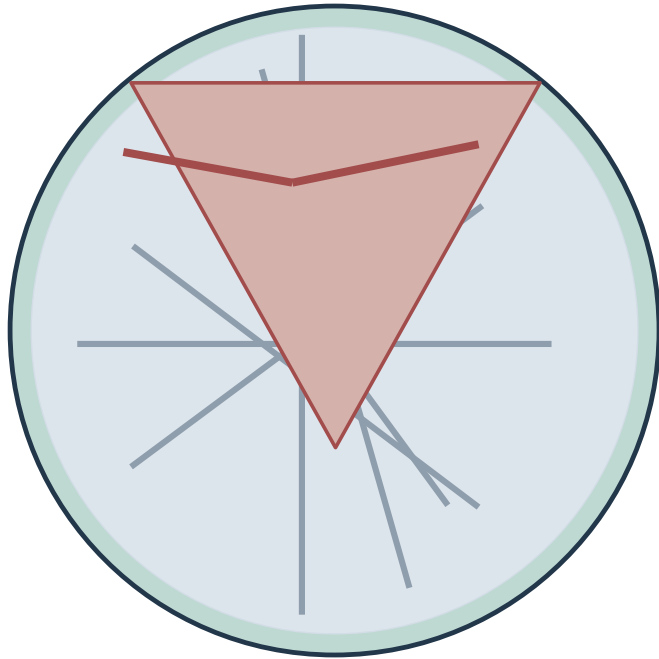


Articular cartilage

Wedge of subchondral necrosis

Broad base near joint surface

The gross femoral-head lesion

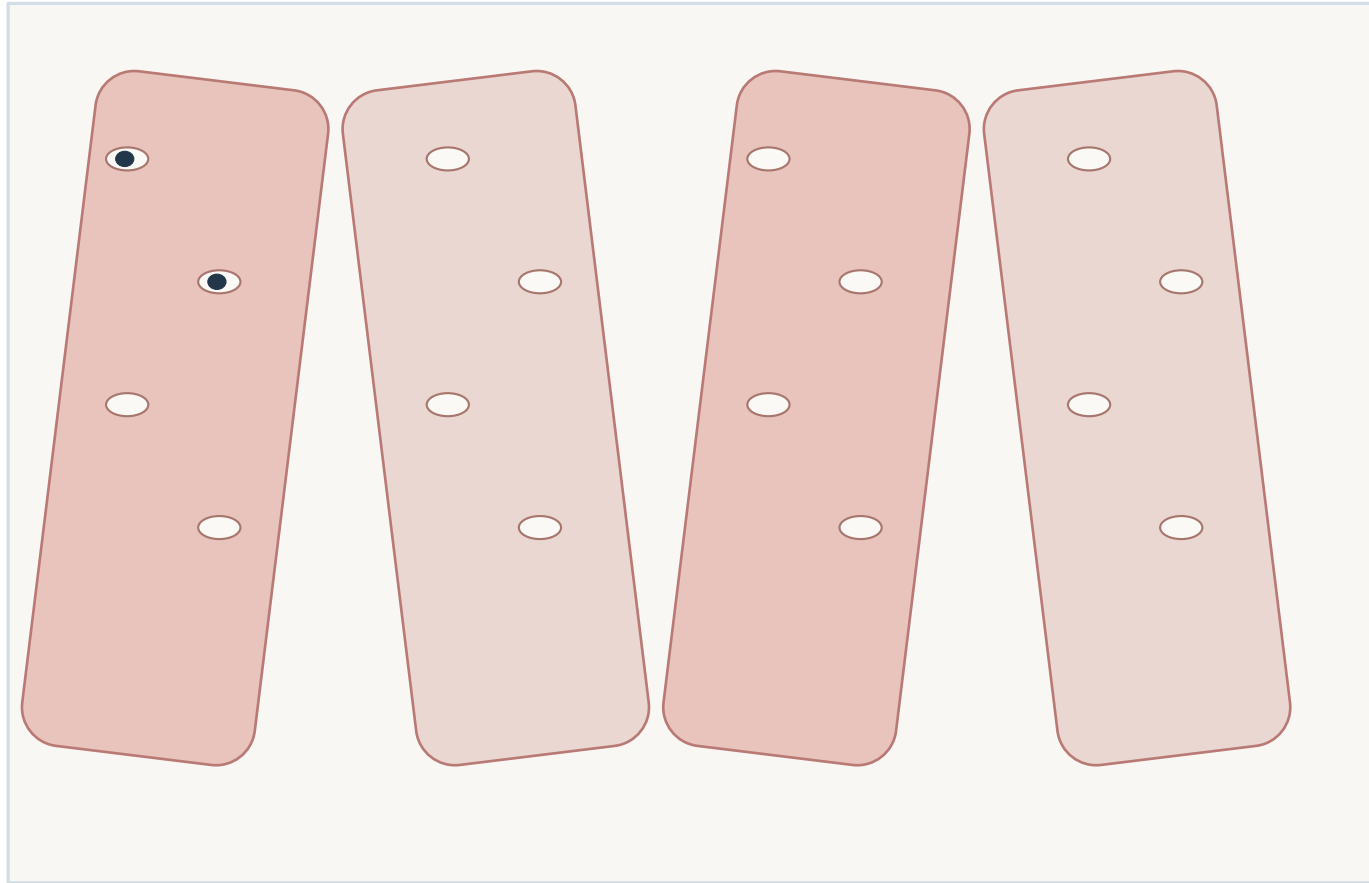


Pale wedge-like infarct

Compression fracture line

Gross morphology schematic

Microanatomy: Osteocyte lacunae become empty



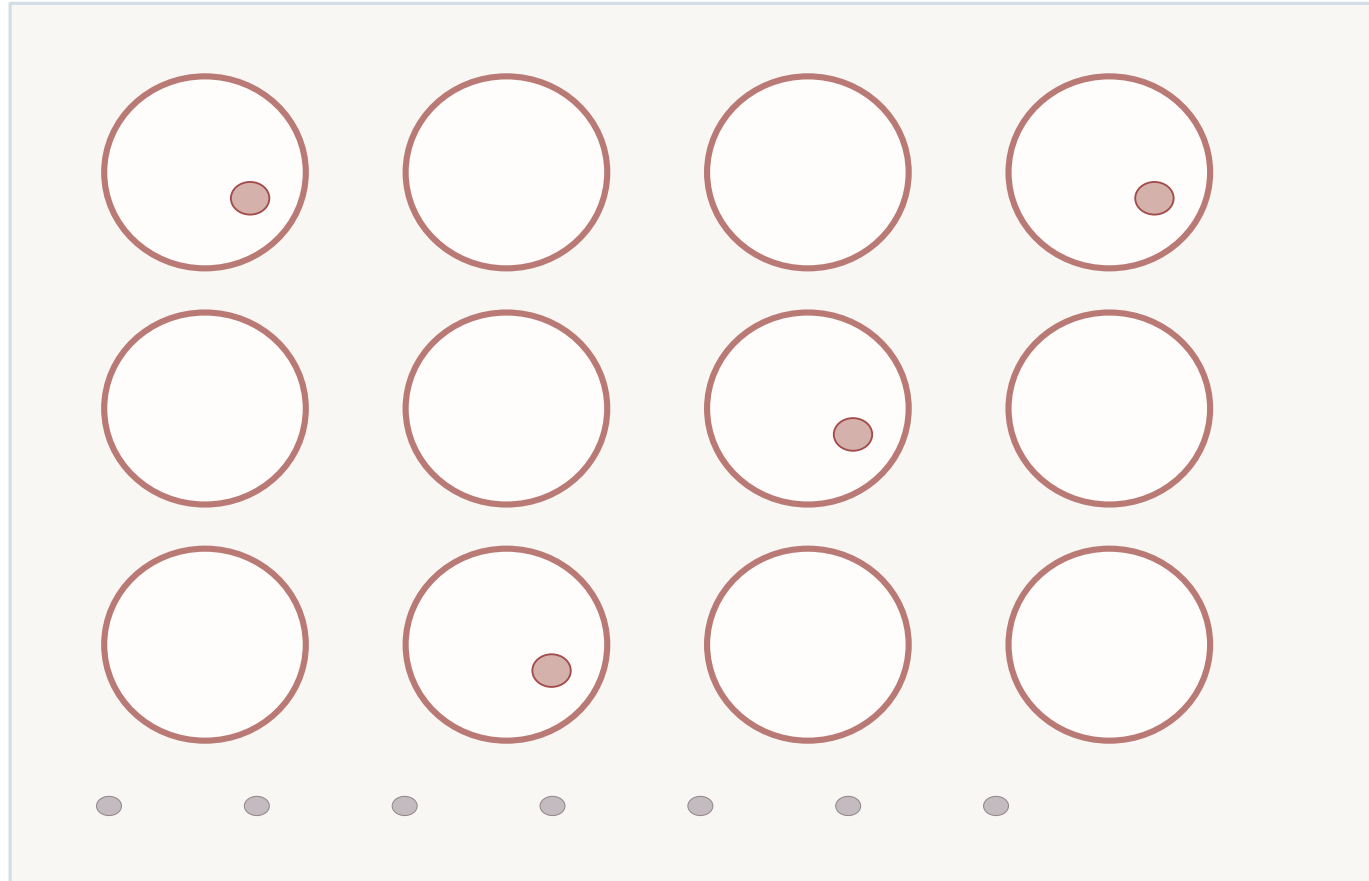
Empty osteocyte lacunae

Loss of nuclei in dead bone

Trabecular mineral matrix persists

Schematic illustration — not a clinical/histologic specimen

Marrow adipocytes also undergo necrosis



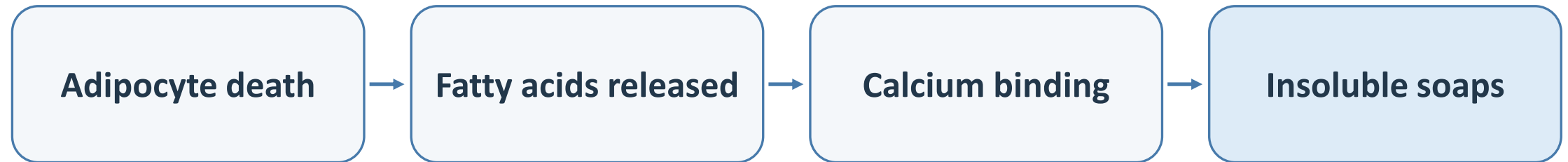
Adipocytes in marrow spaces

Cell membrane injury and death

Fatty acids → calcium soaps

Schematic illustration — not a clinical/histologic specimen

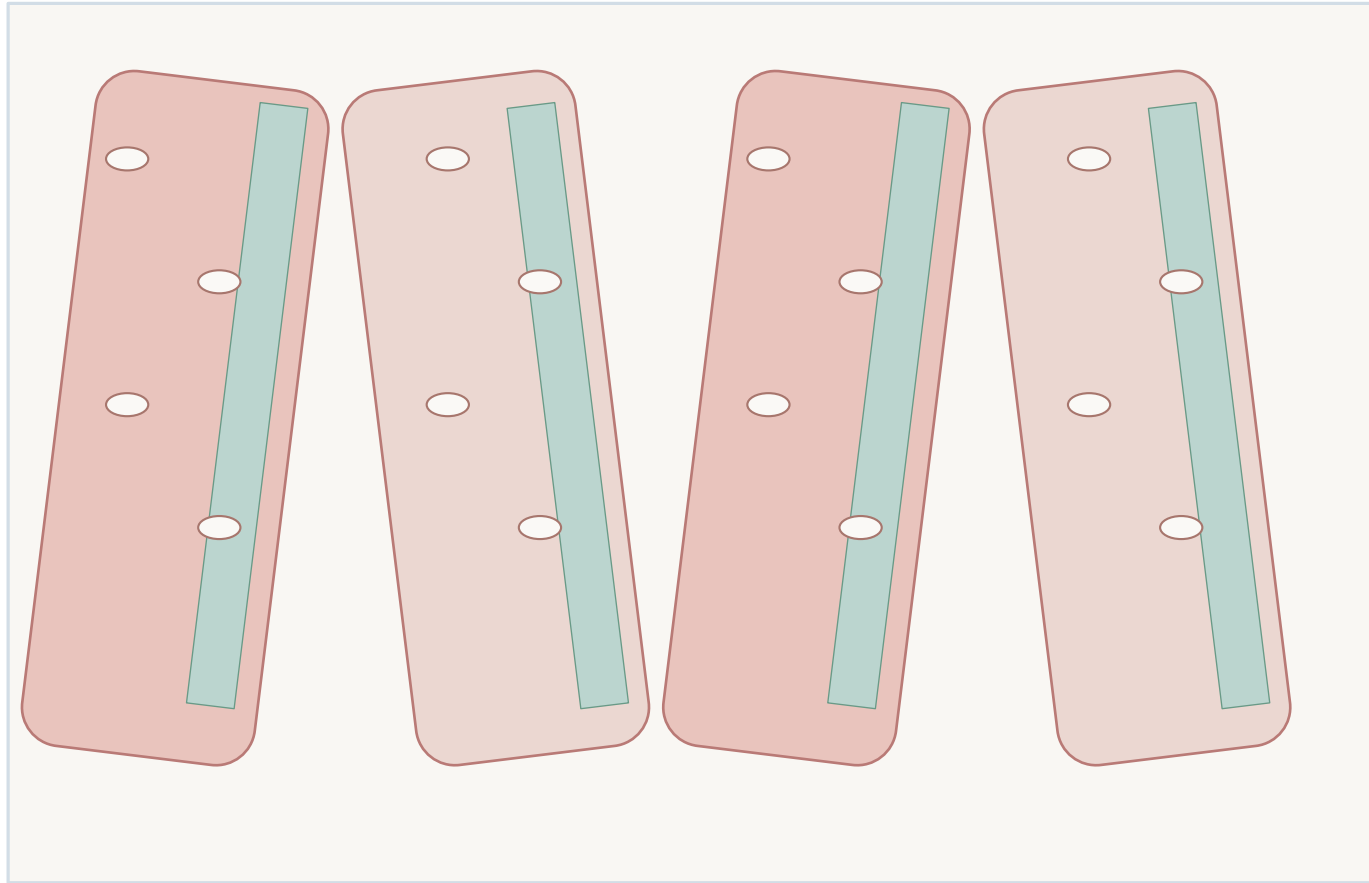
Fat necrosis produces calcium soaps



Conceptual causal pathway

Fatty acids are released from necrotic marrow adipocytes.

What remains after the cells die?



Persistent dead trabeculae

Empty osteocyte lacunae

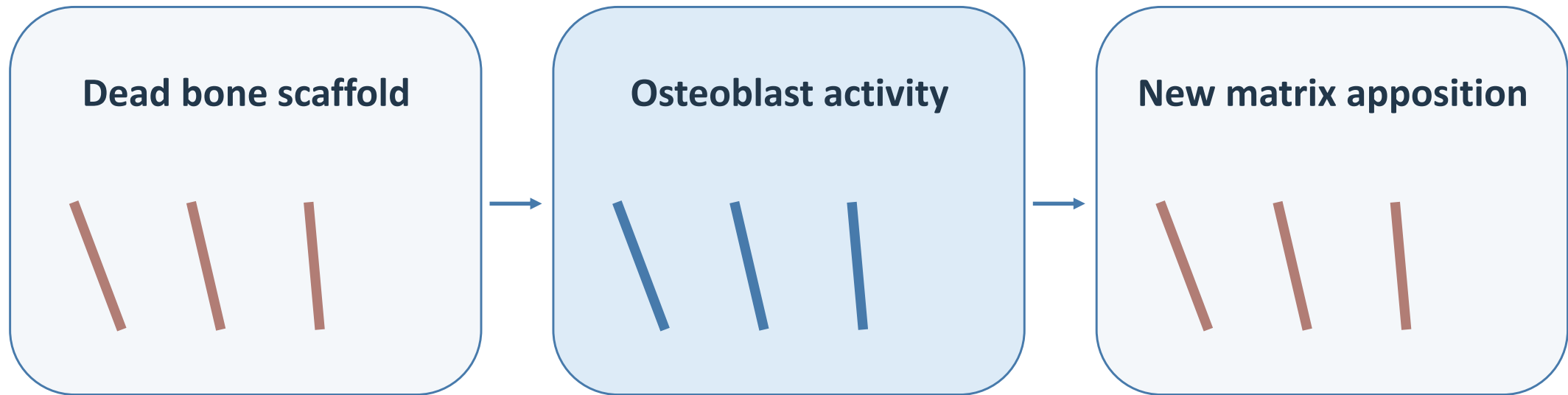
New bone apposed on scaffold

Schematic illustration — not a clinical/histologic specimen

Microscopic recognition: Paired tissue compartments

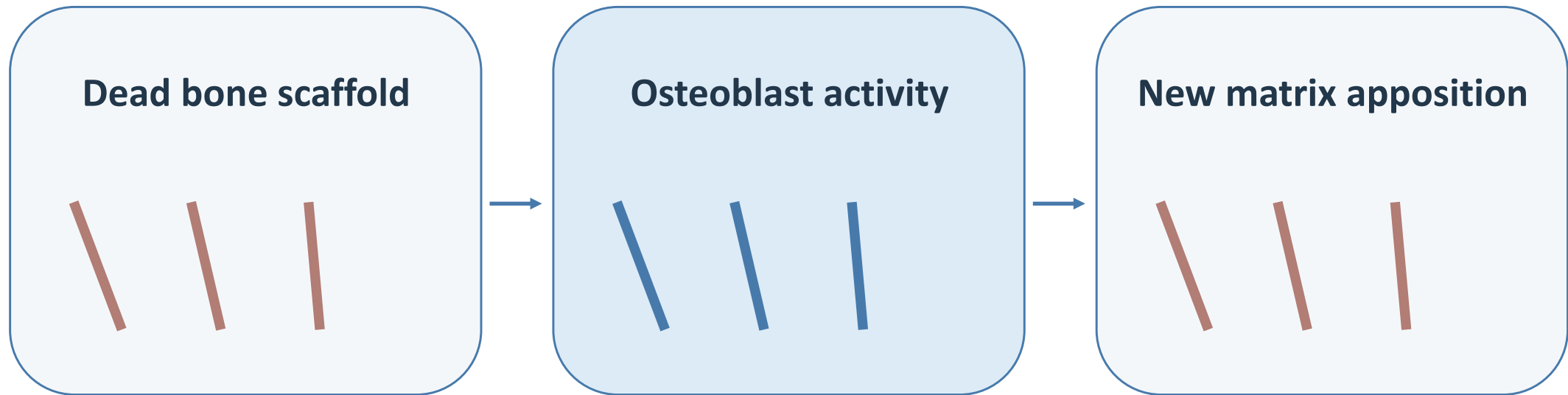
Tissue	Key observation	Meaning
Trabecular bone	Empty osteocyte lacunae	Death of osteocytes
Marrow fat	Necrotic adipocytes	Marrow injury
Infarct repair	New bone and resorption	Reaction to dead scaffolding

Dead trabeculae form the initial repair scaffold



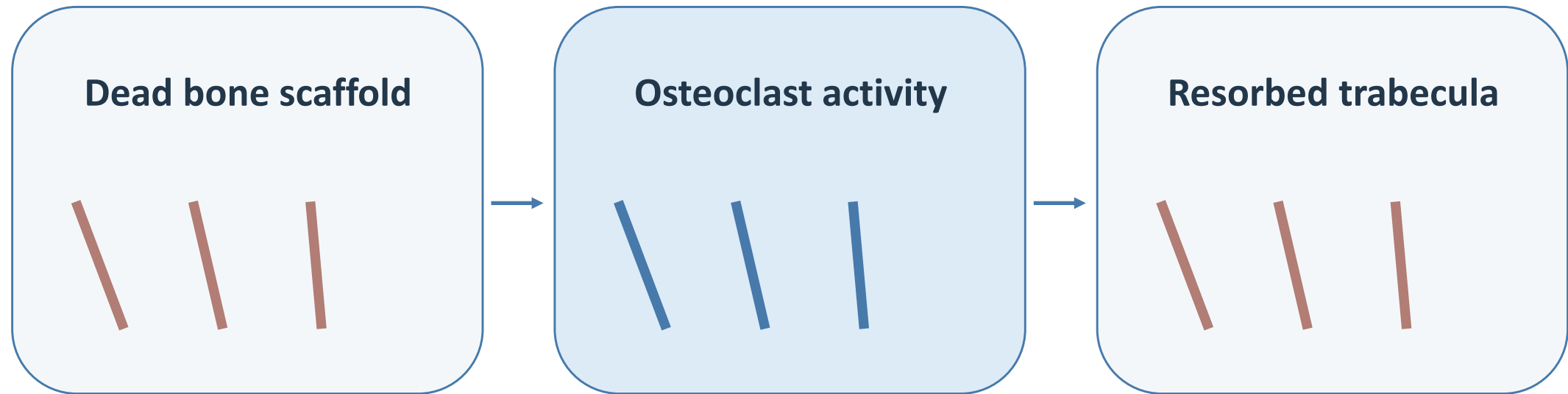
Conceptual histologic repair — all elements editable

Osteoblasts deposit new bone on old trabeculae



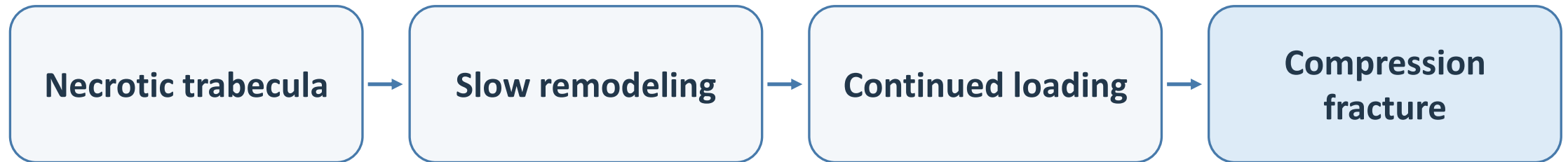
Conceptual histologic repair — all elements editable

Osteoclasts resorb necrotic trabeculae



Conceptual histologic repair — all elements editable

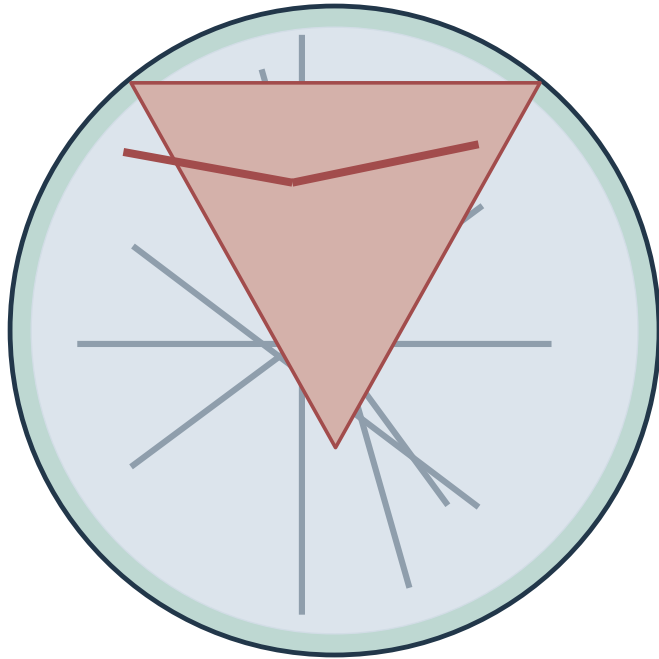
Repair is not equivalent to immediate load-bearing recovery



Conceptual causal pathway

The remodeling process consumes time while the joint remains mechanically loaded.

Why subchondral infarcts collapse

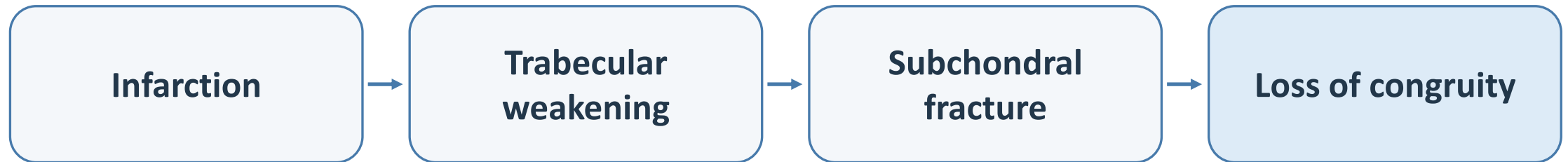


Subchondral compression fracture

Dead support under load

Slow remodeling

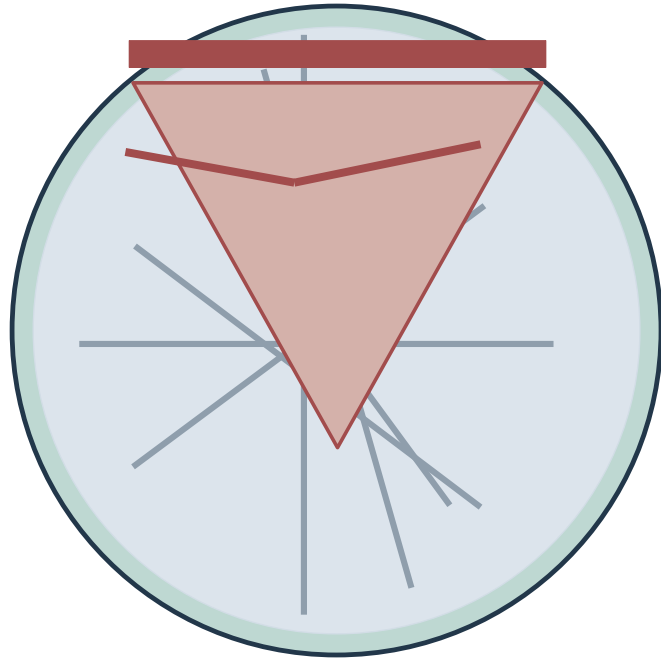
The subchondral fracture is a biomechanical turning point



Conceptual causal pathway

A fracture changes the lesion from cellular necrosis to structural failure.

Articular cartilage fails secondarily

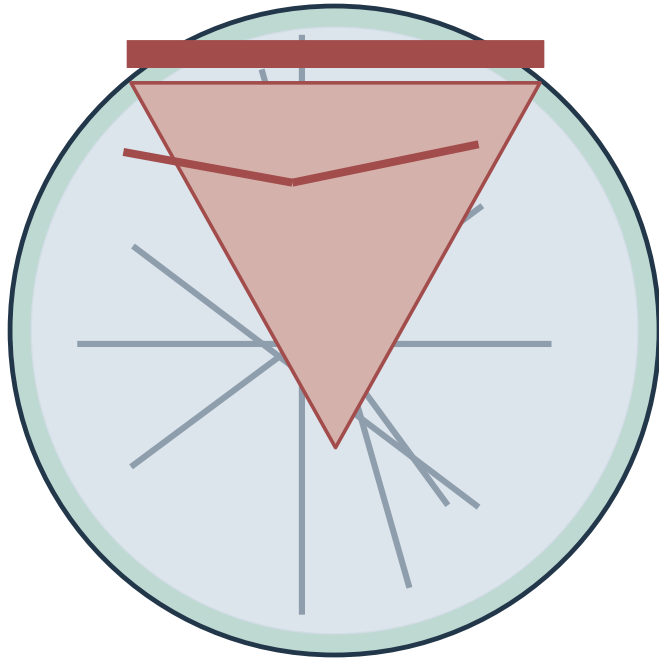


Loss of articular support

Surface deformation

Secondary cartilage damage

Secondary osteoarthritis is the late joint outcome

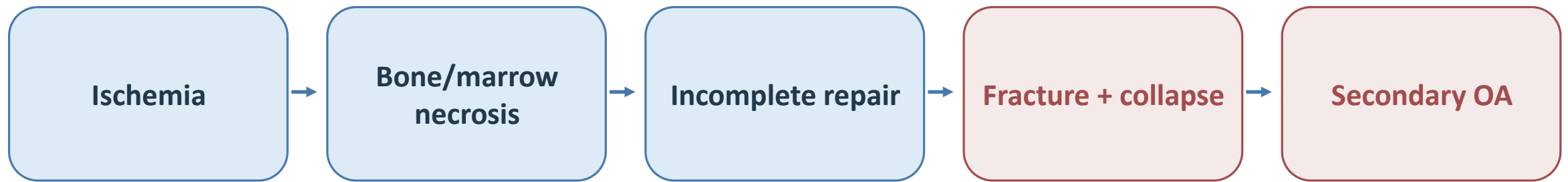


Distorted joint surface

Cartilage loss

Secondary osteoarthritis

Morphologic sequence: Ischemia to arthritis



Sequence is conceptual; Durations vary by patient and lesion

Medullary versus subchondral: Why outcomes diverge

Parameter	Medullary	Subchondral
Configuration	Geographic trabecular focus	Triangular subarticular focus
Cortex / cartilage	Cortex spared	Cartilage initially viable
Typical consequence	Usually silent	Collapse → osteoarthritis
Why different?	Less direct effect on joint surface	Load-bearing plate loses support

Clinical expression depends on infarct location

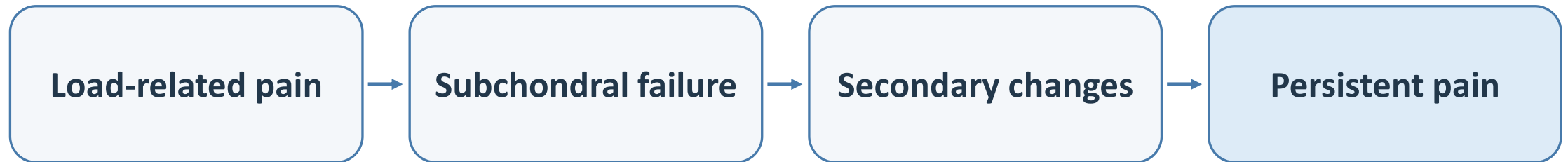
Pattern	Anatomic condition	Expected presentation
Medullary	Away from articular support	Often silent
Subchondral	Beneath load-bearing cartilage	Pain; Risk of collapse
Collapsed	Irregular joint surface	Secondary OA phenotype

Early subchondral pain is activity related

Load-dependent symptoms connect structure to function

- Pain may initially appear with weight-bearing activity.
- A vulnerable load-bearing segment is being mechanically stressed.
- Early clinical severity does not establish the full extent of necrosis.

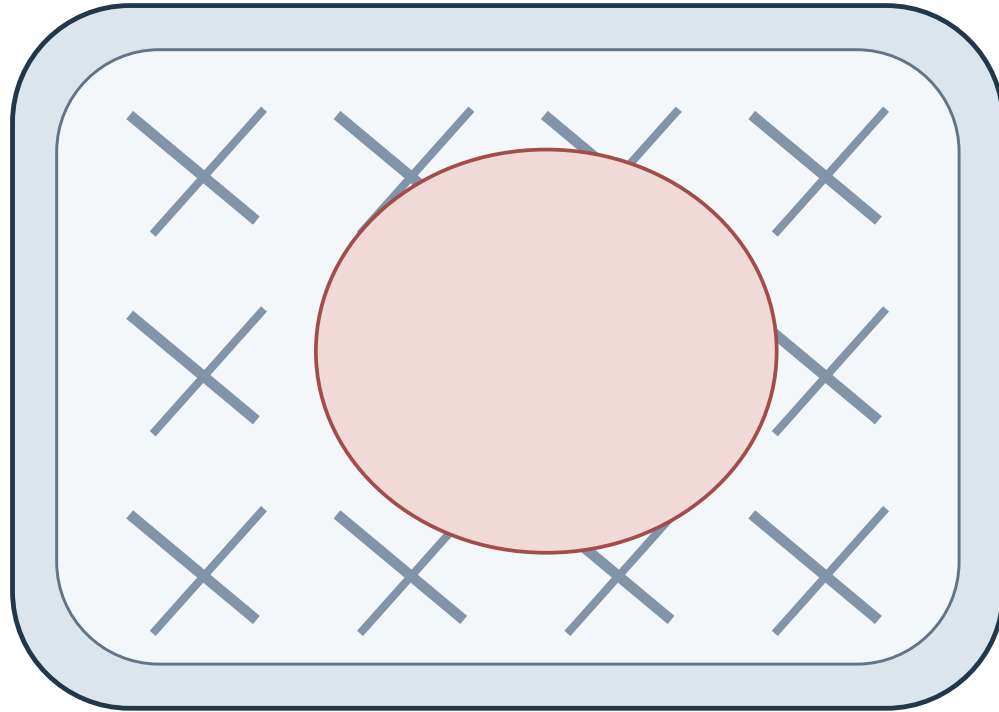
Pain may become constant with progression



Conceptual causal pathway

Fracture and secondary joint changes can make pain persistent.

Medullary infarcts may remain undetected clinically

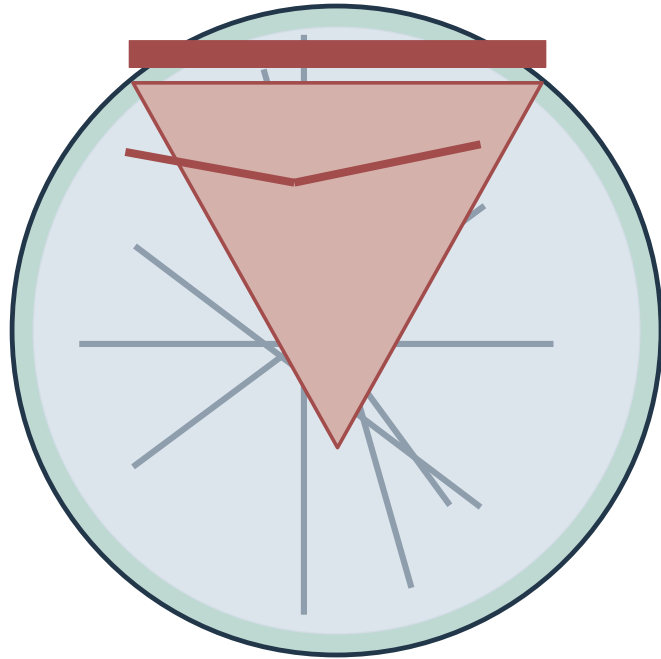


Schematic cross-section

Regional marrow + trabecular infarct

Preserved cortical rim

Femoral-head osteonecrosis: Clinical integration



Loss of articular support

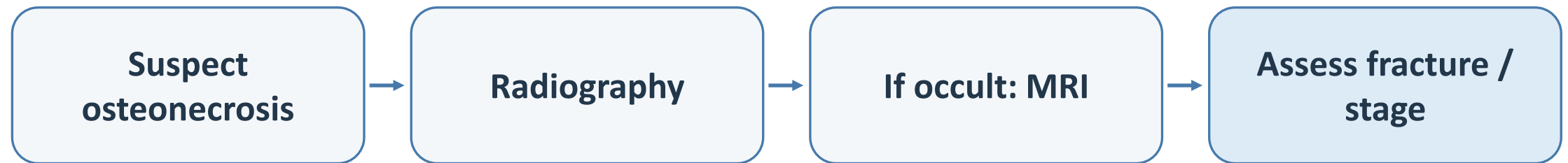
Surface deformation

Secondary cartilage damage

A relevant history narrows the differential

- Ask about fracture or dislocation, corticosteroid exposure, and systemic disorders.
- Radiation and vascular inflammatory disease may be relevant.
- Risk-factor absence does not exclude idiopathic osteonecrosis.

Suspected femoral-head osteonecrosis: Imaging workflow

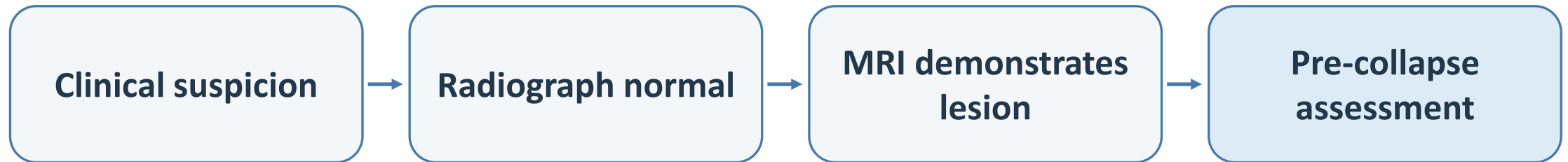


Conceptual causal pathway

Plain radiographs: Useful for structural change

- Late changes can include a sclerotic margin or focal osseous abnormality.
- A subchondral crescent sign reflects fracture.
- Radiography may be normal before overt structural change appears.

MRI: Detecting the pre-collapse lesion



Conceptual causal pathway

MRI can show an osteonecrotic area before radiographs become abnormal.

2019 revised ARCO classification: Four stages

I

**X-ray normal
MRI positive**

II

**Bone changes
No fracture**

III

**Fracture
± depression**

IV

**Secondary
Osteoarthritis**

2019 revised ARCO staging; For osteonecrosis of the femoral head

ARCO stage I: Occult on the radiograph

Stage I: X-ray normal; MRI / bone scan positive

- Plain radiographs do not yet show abnormalities.
- MRI or bone scintigraphy identifies the lesion.
- This is a pre-collapse imaging category.

ARCO stage II: Abnormal bone but no fracture

Stage II: X-ray abnormal; No fracture or flattening

- Radiographs show sclerotic, osteopenic, or cystic change.
- Neither subchondral fracture nor femoral-head flattening is established.
- Stage II remains within the pre-collapse spectrum.

ARCO stage III: Fracture has developed

Stage III is defined by fracture; IIIA and IIIB use 2-mm depression

- Radiography or CT demonstrates fracture in the subchondral or necrotic zone.
- Stage IIIA: Femoral-head depression ≤ 2 mm.
- Stage IIIB: Femoral-head depression > 2 mm.

ARCO stage IV: Secondary osteoarthritis

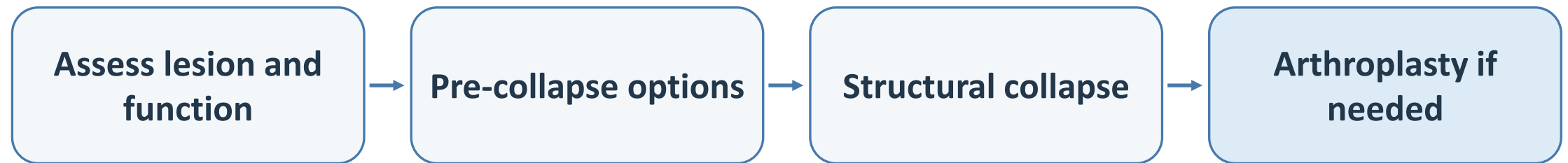
Stage IV: Radiographic osteoarthritis

- Radiographs demonstrate an osteoarthritic joint.
- Joint-space narrowing and acetabular change may accompany destruction.
- This category represents an advanced articular consequence.

Staging records the major pathologic transition

Stage	Radiograph / CT	Major distinction
I	X-ray normal	MRI / scan positive
II	Bone changes, no fracture	Pre-collapse
III	Fracture $\pm$ depression	Subchondral failure
IV	Osteoarthritic joint changes	Secondary joint degeneration

Management responds to structure, symptoms, and stage



Conceptual causal pathway

Conservative strategies: Mechanical symptom control

- Reducing weight-bearing can lessen stress on vulnerable trabeculae.
- Immobilization or activity modification is included's management spectrum.
- These approaches do not guarantee reversal of established infarction.

Hip-preserving surgery is stage dependent

- Core decompression is a possible early-stage, pre-collapse procedure.
- The procedure seeks to reduce intraosseous pressure and facilitate revascularization.
- Outcomes depend on lesion characteristics and whether collapse has occurred.

Advanced collapse may require joint replacement

- Femoral-head deformation compromises load-bearing congruity.
- Secondary osteoarthritis can create marked pain and loss of function.
- Hip arthroplasty is among surgical options for advanced disease.

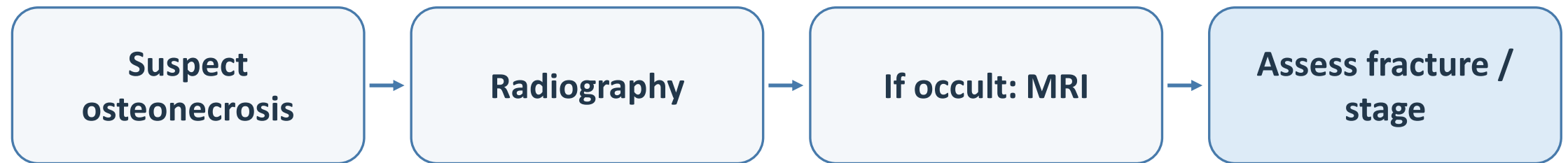
Case: Pain after prolonged corticosteroid treatment

CLINICAL PROBLEM

- A 46-year-old develops gradually increasing groin pain without remembered trauma.
- The patient has received long-term systemic corticosteroids.

? Plain radiographs are unrevealing. What study best evaluates suspected early osteonecrosis?

Case reasoning: Detect disease before collapse



Conceptual causal pathway

Case: Subchondral fracture and deformation

CLINICAL PROBLEM

- A patient with established femoral-head osteonecrosis develops persistent pain.
- CT shows fracture in the necrotic zone and 3-mm head depression.

? Which 2019 ARCO stage best describes these findings?

Case reasoning: ARCO IIIB

I

**X-ray normal
MRI positive**

II

**Bone changes
No fracture**

III

**Fracture
± depression**

IV

**Secondary
Osteoarthritis**

2019 revised ARCO staging; For osteonecrosis of the femoral head

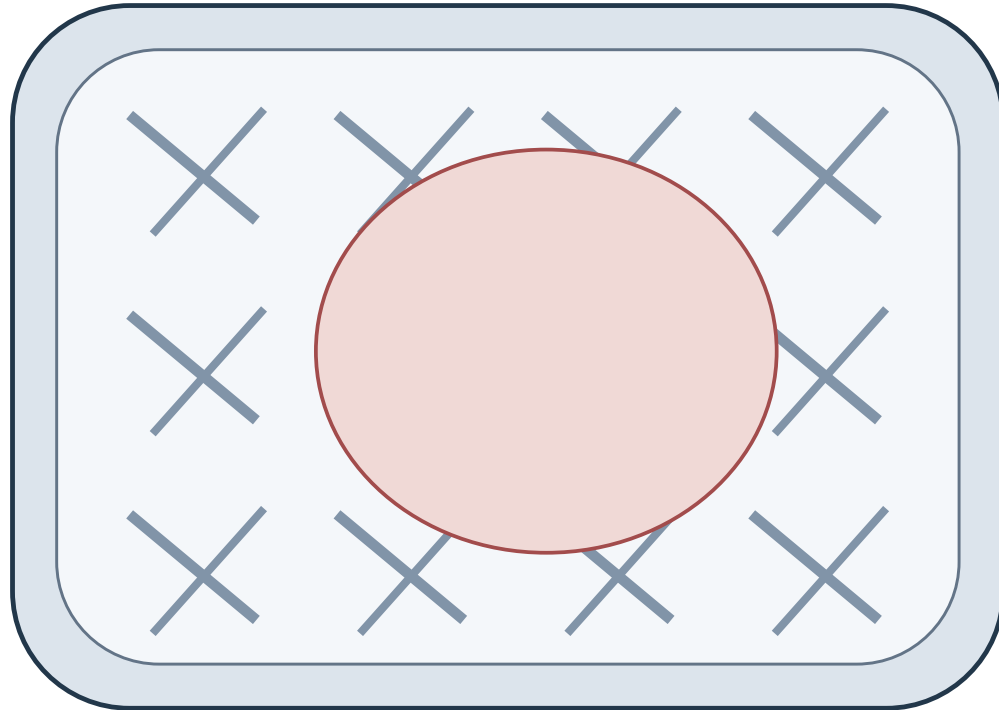
Case: Incidental medullary bone infarction

CLINICAL PROBLEM

- An incidental geographic lesion involves trabecular marrow within a long bone.
- The cortical shell is preserved and there is no nearby articular collapse.

? What morphology-based clinical tendency is expected?

Case reasoning: A medullary infarct may be silent

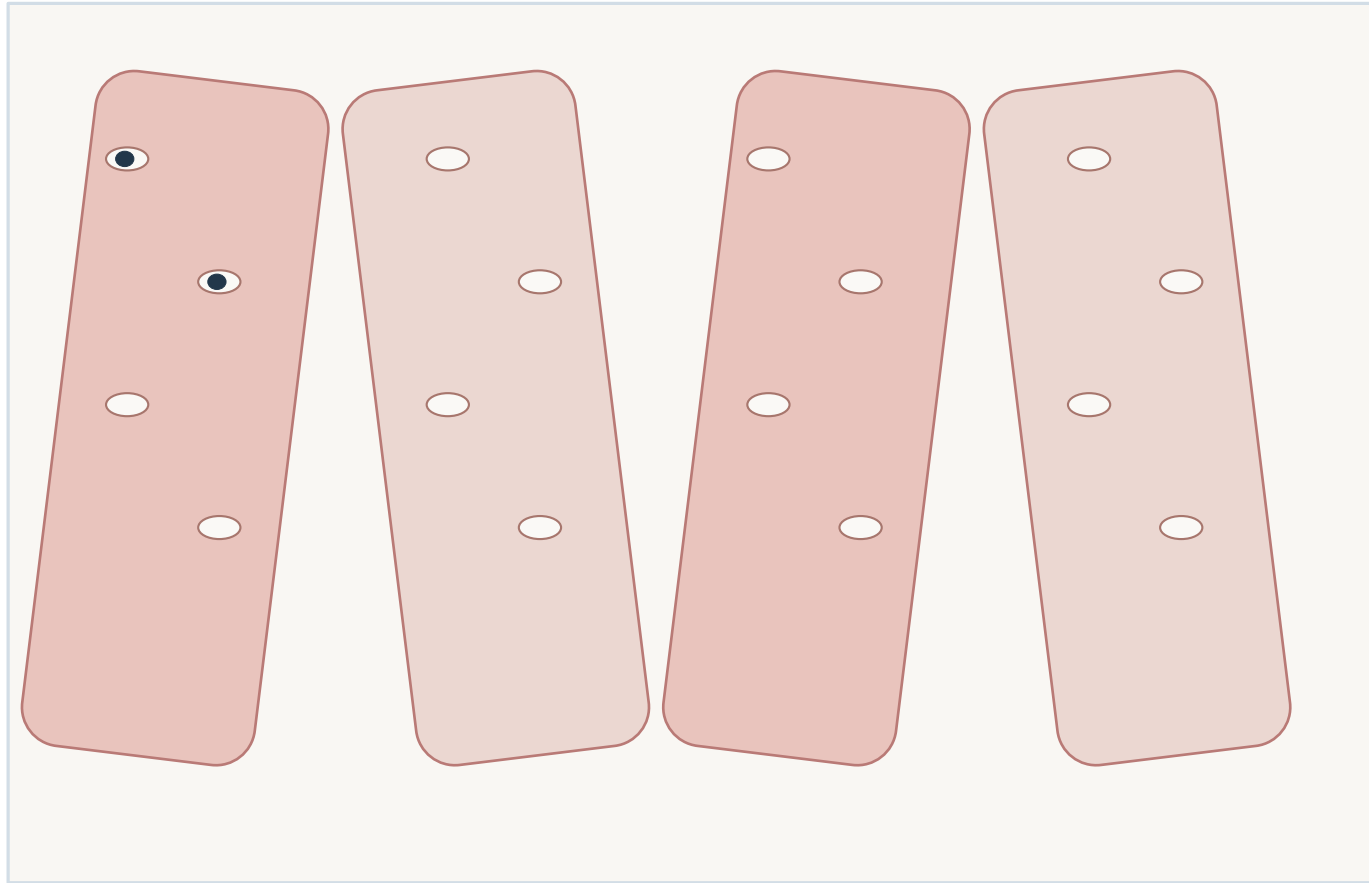


Schematic cross-section

Regional marrow + trabecular infarct

Preserved cortical rim

Microscopic challenge: Identify necrotic bone



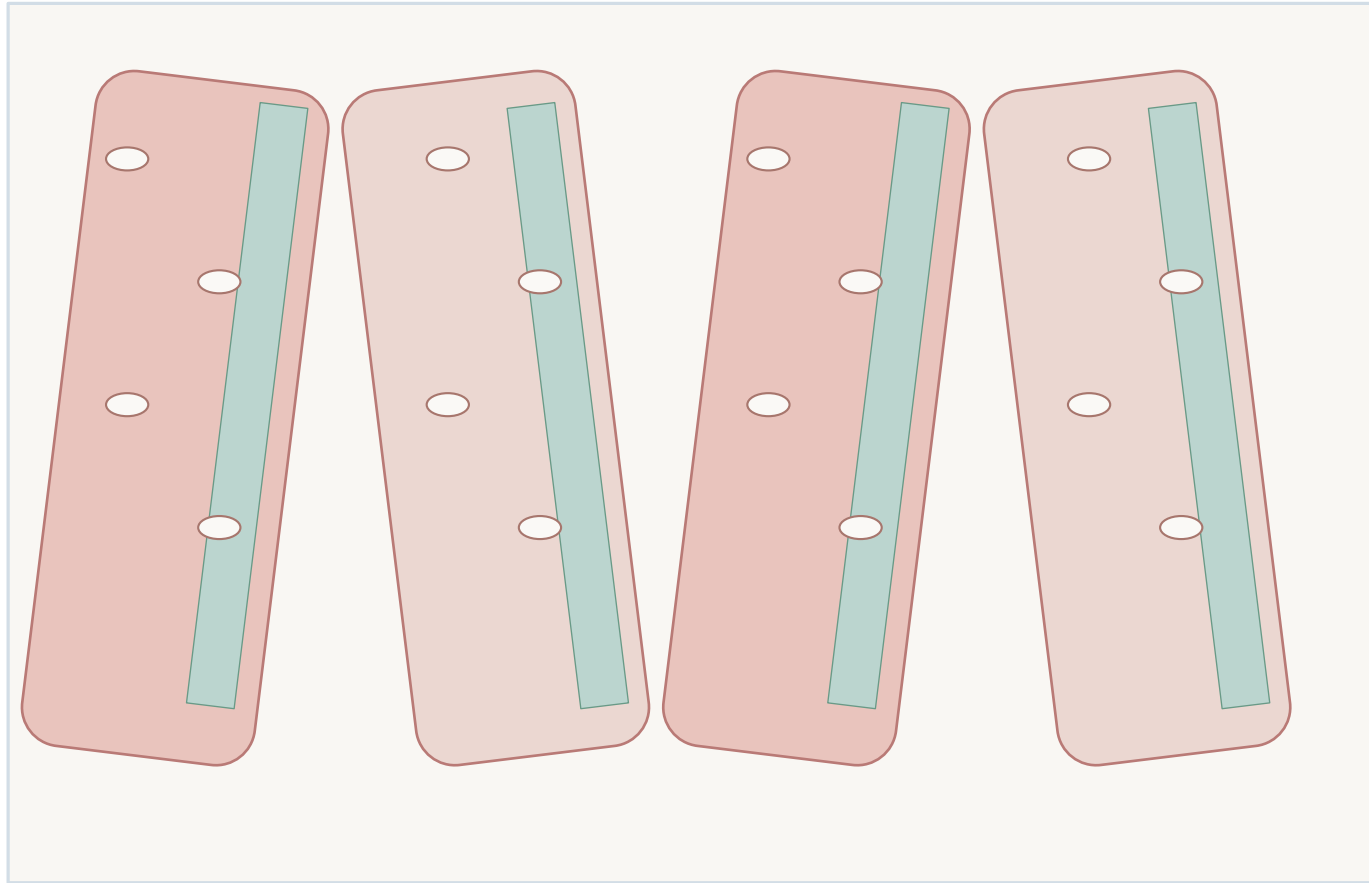
Empty osteocyte lacunae

Loss of nuclei in dead bone

Trabecular mineral matrix persists

Schematic illustration — not a clinical/histologic specimen

Microscopic answer: Two compartments have died



Persistent dead trabeculae

Empty osteocyte lacunae

New bone apposed on scaffold

Schematic illustration — not a clinical/histologic specimen

Osteonecrosis: Five clinicopathologic rules

- Bone and marrow infarction is the defining lesion.
- Vascular compromise has traumatic, thrombotic, and compressive routes.
- Medullary lesions often spare cortex; Subchondral lesions threaten the joint.
- Empty lacunae and marrow fat necrosis support morphology.
- Slow repair plus loading can lead to collapse and secondary osteoarthritis.

Source references and visual provenance

- Robbins, Cotran & Kumar, Pathologic Basis of Disease, 11th ed. (2026): Chapter 26, pp. 1075–1077.
- AAOS OrthoInfo: Osteonecrosis of the Hip; ACR Appropriateness Criteria: Osteonecrosis (2022).
- Yoon et al. J Arthroplasty. 2020;35:933–940. DOI: 10.1016/j.arth.2019.11.029.
- Anatomic and histologic drawings are original editable schematics—not diagnostic specimens.