

Pyogenic Osteomyelitis

Etiology · pathogenesis · morphology · clinicopathologic correlation

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Learning objectives

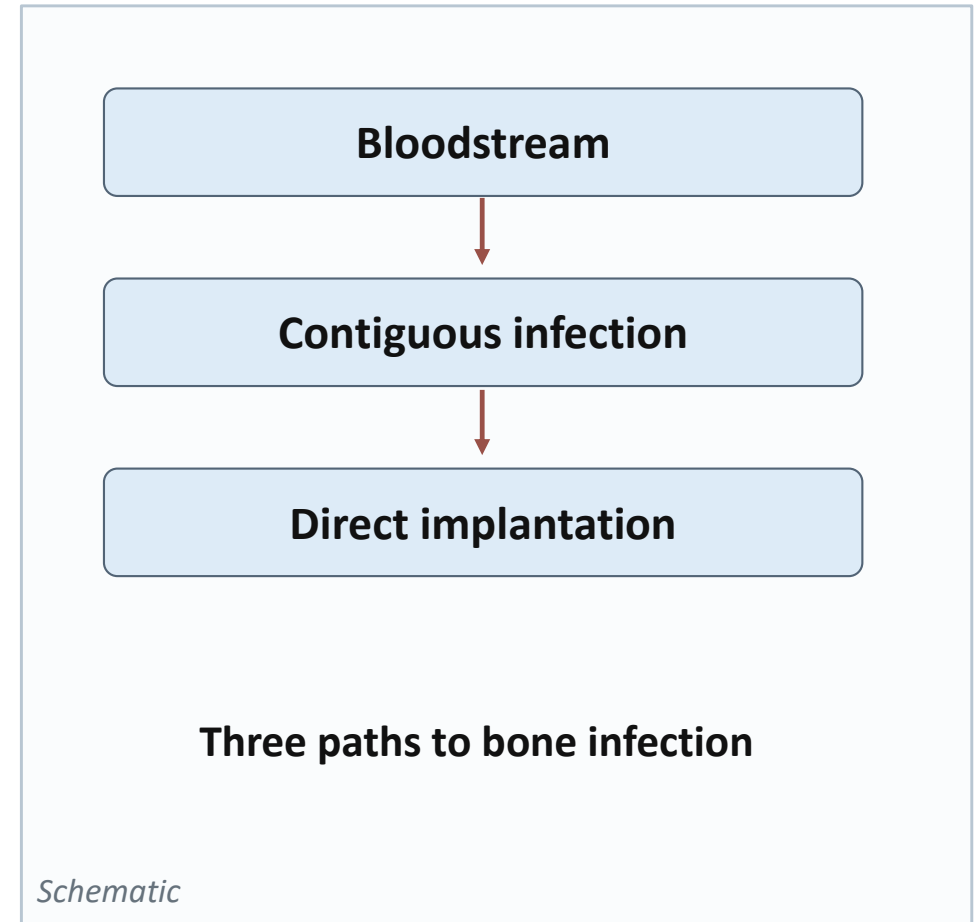
- Distinguish hematogenous, contiguous and directly implanted bone infections.
- Relate age and clinical setting to organisms and sites of infection.
- Explain acute suppuration, necrosis, sequestrum and involucrum.
- Interpret clinical findings, imaging and microbial sampling.
- Anticipate chronic complications and key diagnostic distinctions.

Osteomyelitis affects bone and marrow

- Osteomyelitis is inflammation of bone and marrow, virtually always due to infection.
- Pyogenic osteomyelitis is almost always bacterial.
- Infection may be part of a systemic process or present as a solitary primary focus.
- The local response combines inflammation, tissue necrosis and reactive bone changes.

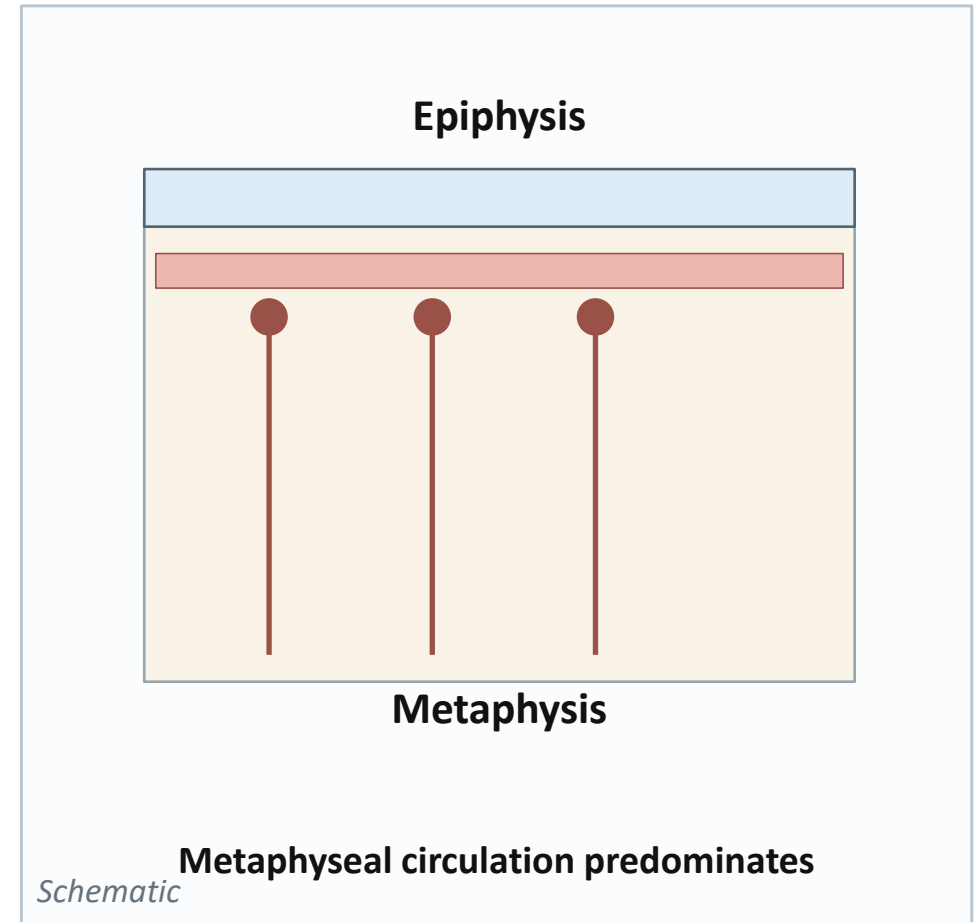
Three routes to bone infection

- Hematogenous spread: Organisms seed bone from the bloodstream.
- Contiguous extension: An adjacent infectious focus enters bone.
- Direct implantation: Organisms enter through open injury or an orthopedic procedure.
- The route changes the likely patient population and microbiology.



Hematogenous spread in children

- Most osteomyelitis in otherwise healthy children is hematogenous.
- Long bones are frequent targets.
- An initiating bacteremia may follow trivial mucosal injury or minor skin infection.
- A seemingly minor antecedent infection can precede a serious bone focus.



Contiguous spread versus implantation

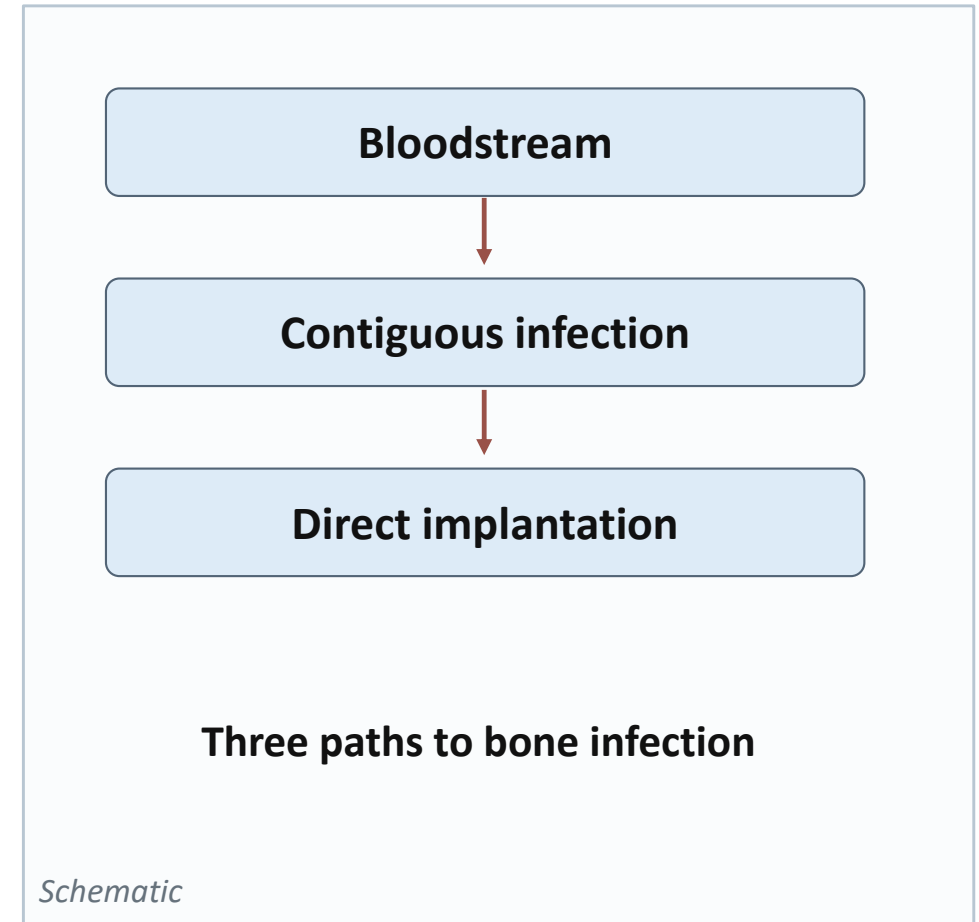
Route	Entry mechanism	Typical context
Contiguous spread	Infection extends from adjacent tissues	Diabetes-associated foot infection
Direct implantation	Organisms reach bone at time of disruption	Open fracture; Orthopedic surgery
Hematogenous spread	Bacteremia seeds osseous vessels	Otherwise healthy children

Age changes the dominant context

Population	Predominant clinical setting	Important concern
Children	Bloodborne infection; Long bones	Metaphyseal seeding
Adults	Open fractures, operations, diabetes	Direct or contiguous infection
Infants	Epiphyseal infection may reach a joint	Septic arthritis; Cartilage loss

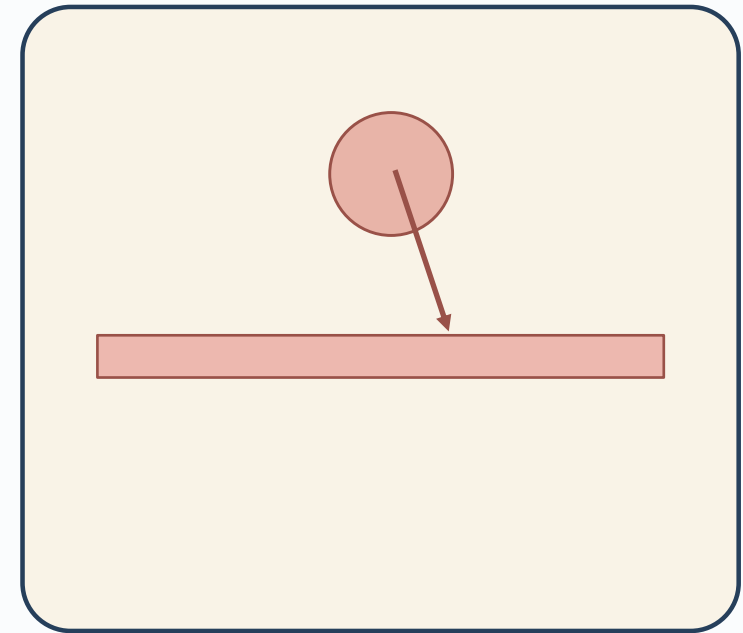
Diabetes-associated foot infection

- Diabetes as an adult predisposition to foot infections involving bone.
- The clinical context differs from otherwise healthy childhood hematogenous disease.
- Adjacent infected tissue provides a plausible route of extension to bone.
- The anatomic site and route guide microbiologic suspicion.



Infant epiphyseal infection threatens joints

- In infants, infection may involve the epiphysis.
- The nearby joint can become infected: Septic or suppurative arthritis.
- Destruction of articular cartilage can produce permanent disability.
- This is a major age-specific complication of bone infection.



Epiphyseal focus → adjacent joint

Schematic

The organism depends on the setting

- Staphylococcus aureus predominates among culture-positive pyogenic cases.
- Neonates have distinct likely organisms.
- Sickle cell disease shifts organism risk.
- Direct spread, surgery and open injury can yield mixed bacterial infection.

S. Aureus

Neonate: GBS / E. Coli

Sickle cell: Salmonella

Open injury: Mixed flora

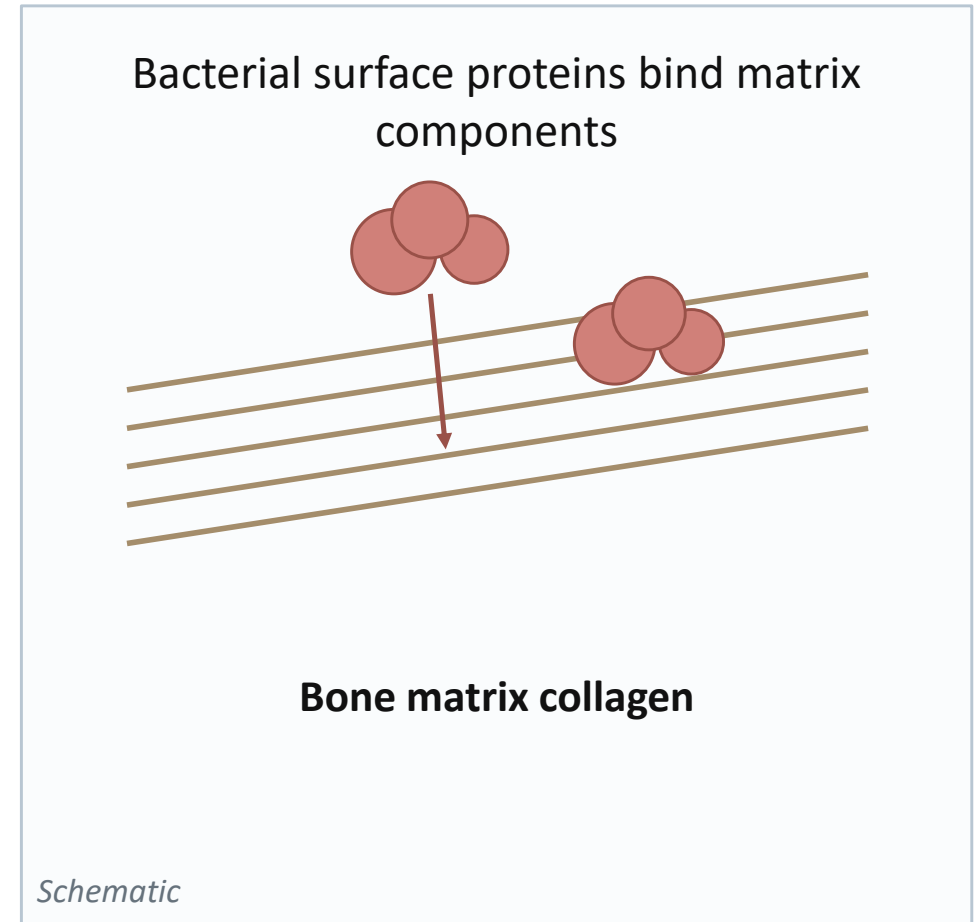
Schematic

Staphylococcus aureus predominates

- 80%–90% of culture-positive pyogenic osteomyelitis to S. Aureus.
- The denominator is culture-positive cases—not all suspected episodes.
- Staphylococcal surface proteins help bind the organism to bone matrix.
- Clinical setting still matters when considering alternative organisms.

Staphylococcal adherence to matrix

- Staphylococcal cell wall proteins bind bone matrix components.
- Collagen is one of the matrix ligands named.
- Attachment favors bacterial localization on a permissive bone surface.
- This links a bacterial factor with osseous tropism.



Neonatal pathogen spectrum

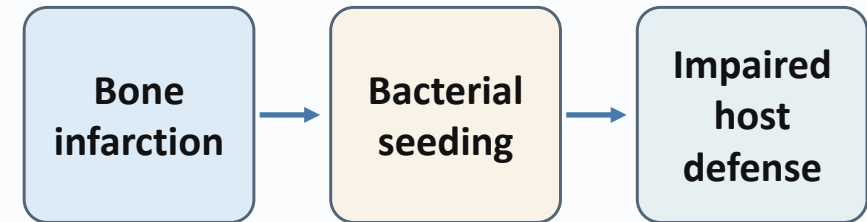
- During the neonatal period, group B streptococci are important pathogens.
- Escherichia coli is another likely neonatal cause.
- Adult and older-child assumptions should not be transferred automatically to newborns.
- Age provides information before culture results are available.

Older children: Gram-positive pathogens

- In older children, gram-positive organisms are the most likely causes.
- *S. Aureus* remains particularly important.
- The common hematogenous presentation is often localized to a long bone.
- Microbial confirmation should still be pursued when feasible.

Why sickle cell disease predisposes

- Bone infarcts create areas of osteonecrosis susceptible to bacterial seeding.
- Impaired splenic function weakens the immune response.
- Both damaged tissue and altered host defense contribute.
- This risk context also changes the expected microbiology.



Sickle cell anemia combines tissue injury and susceptibility to infection

Schematic

Sickle cell disease: Organism clue

- Salmonella and other gram-negative bacteria as frequent pathogens in sickle cell disease.
- S. Aureus is important in osteomyelitis generally, but the risk setting modifies the differential.
- A bone pain episode cannot be interpreted from pain alone; Infection and infarction require consideration.
- Use clinical assessment and microbiological evidence to discriminate.

Polymicrobial pyogenic infection

- Mixed bacterial infection is common after direct spread.
- Orthopedic surgery is another setting in which multiple organisms may be involved.
- Open fractures create an opportunity for direct environmental inoculation.
- Do not assume every adult case has a single staphylococcal pathogen.

Host setting directs microbiology

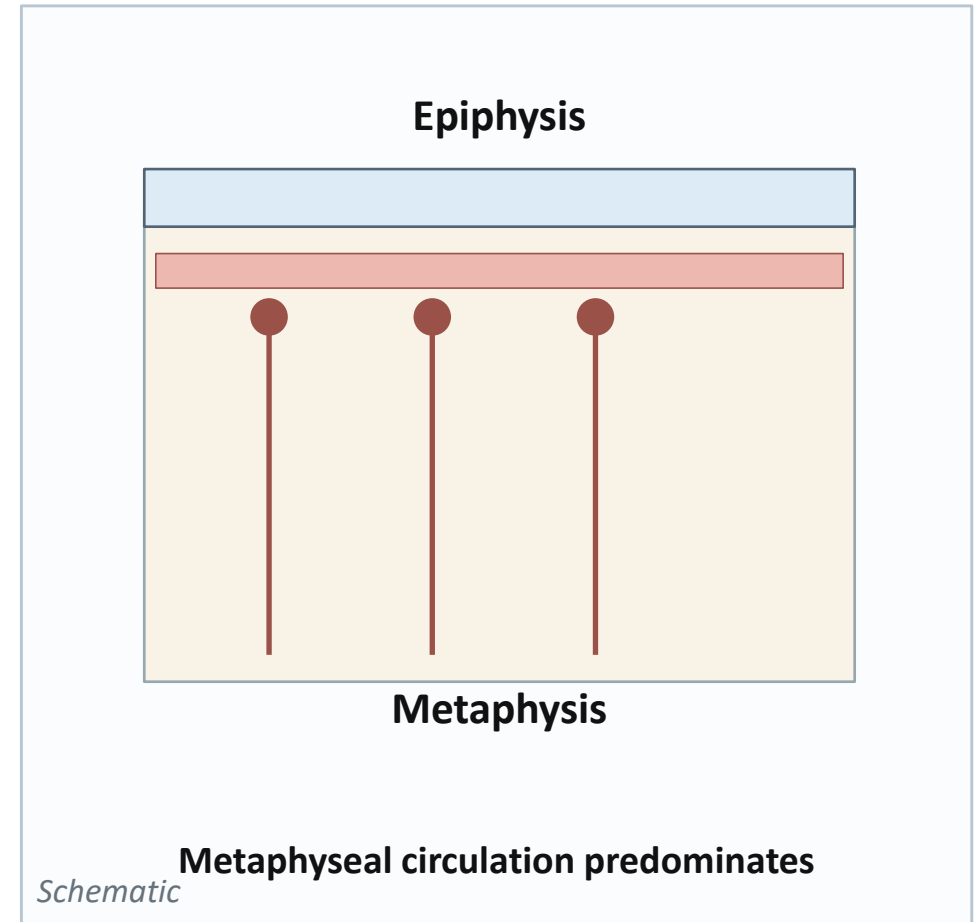
Setting	Organisms emphasized	Mechanistic context
Culture-positive pyogenic disease	S. Aureus (80%–90%)	Matrix adherence
Neonate	Group B streptococci; E. Coli	Age-dependent distribution
Sickle cell disease	Salmonella; Other gram-negative bacteria	Infarction + splenic dysfunction
Open fracture / surgery	Frequently mixed organisms	Direct implantation

Organism identification is imperfect

- Microbiological cultures are obtained whenever possible.
- The chapter reports identifiable organisms in approximately half of patients.
- Negative or unrevealing culture does not by itself exclude bone infection.
- Robbins anticipates that organism-specific DNA testing may improve identification; No performance figures are supplied.

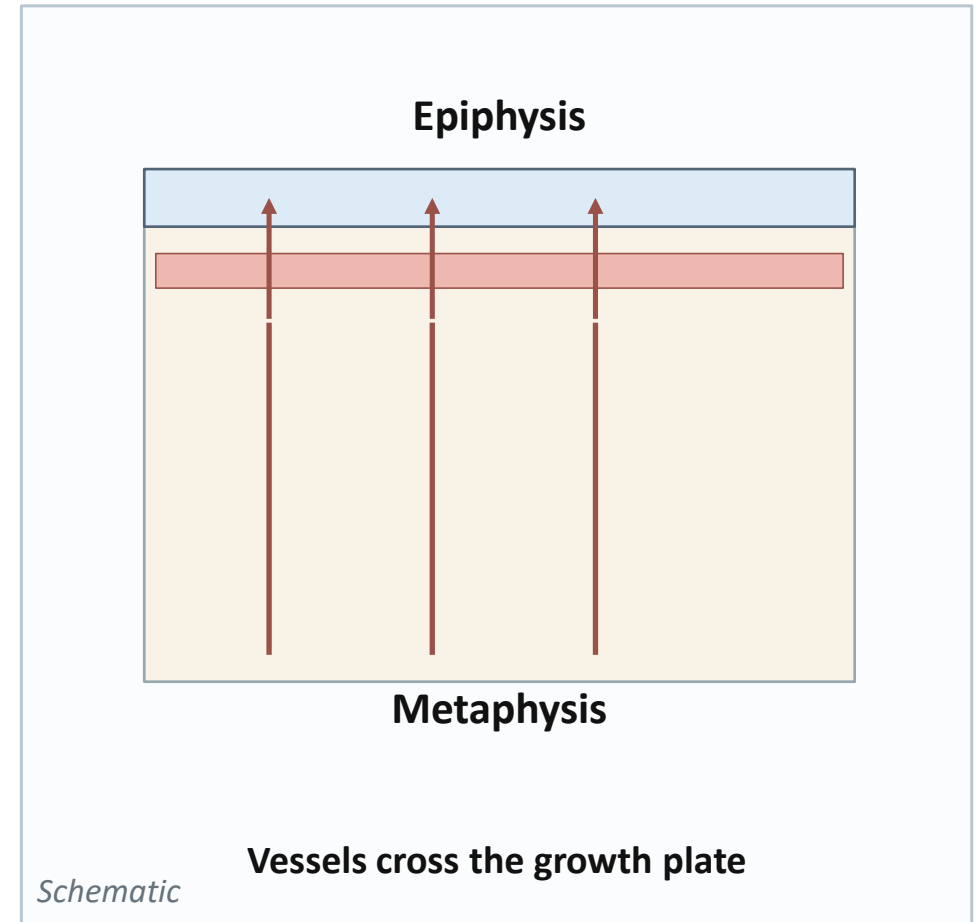
Age-dependent bone vascular supply

- The osseous circulation changes during skeletal growth.
- Vascular architecture determines where hematogenous organisms lodge.
- Metaphyseal and epiphyseal communication differs before and after physeal closure.
- The expected lesion site is consequently age dependent.



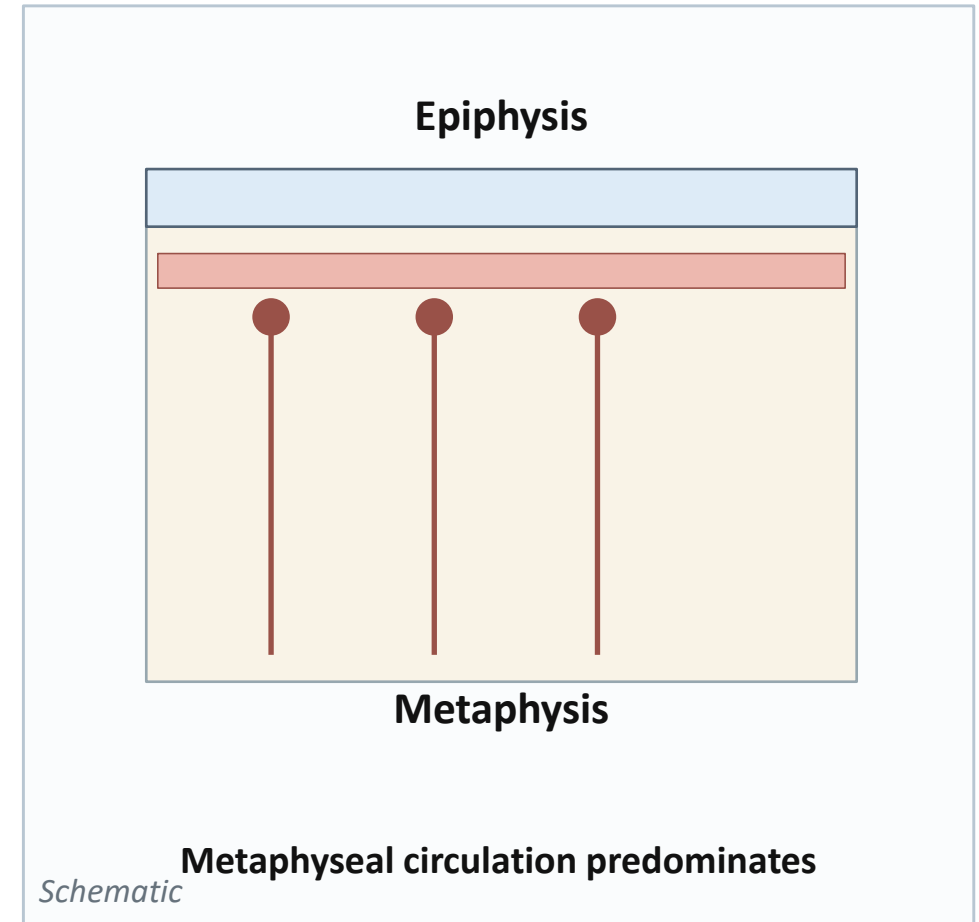
Neonates: Vessels cross the physis

- Neonatal metaphyseal vessels penetrate the growth plate.
- Both metaphysis and epiphysis may be involved.
- The epiphyseal location helps explain risk of adjacent joint infection.
- A neonatal focus need not remain confined to the metaphysis.



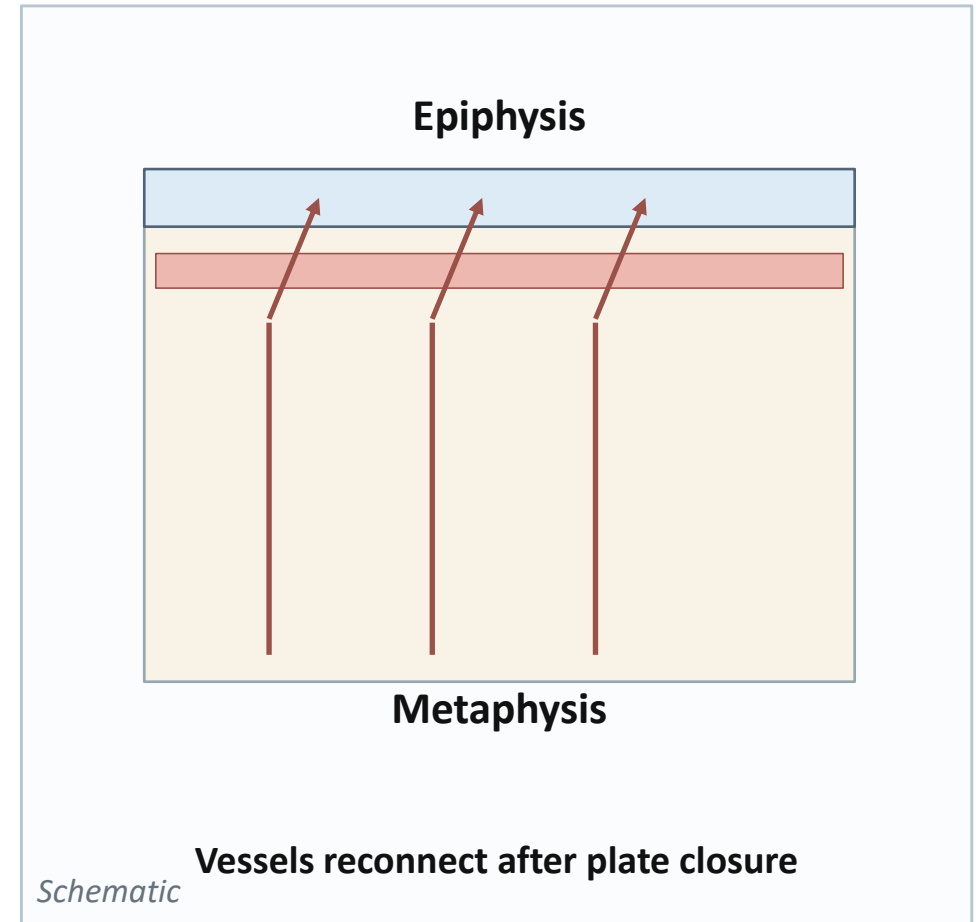
Older children: Metaphyseal localization

- After neonatal vascular remodeling, metaphyseal infection is typical in older children.
- The growth plate limits the direct route found in the neonate.
- This anatomic pattern is particularly relevant to long-bone hematogenous seeding.
- Age and site should be interpreted together.



Adults: Post-physeal communication

- Growth plate closure alters metaphyseal–epiphyseal circulation.
- Anastomoses provide a route for bacterial spread across these regions.
- Epiphyseal and subchondral involvement becomes more common.
- Adult anatomy differs from the usual metaphyseal pattern of older children.

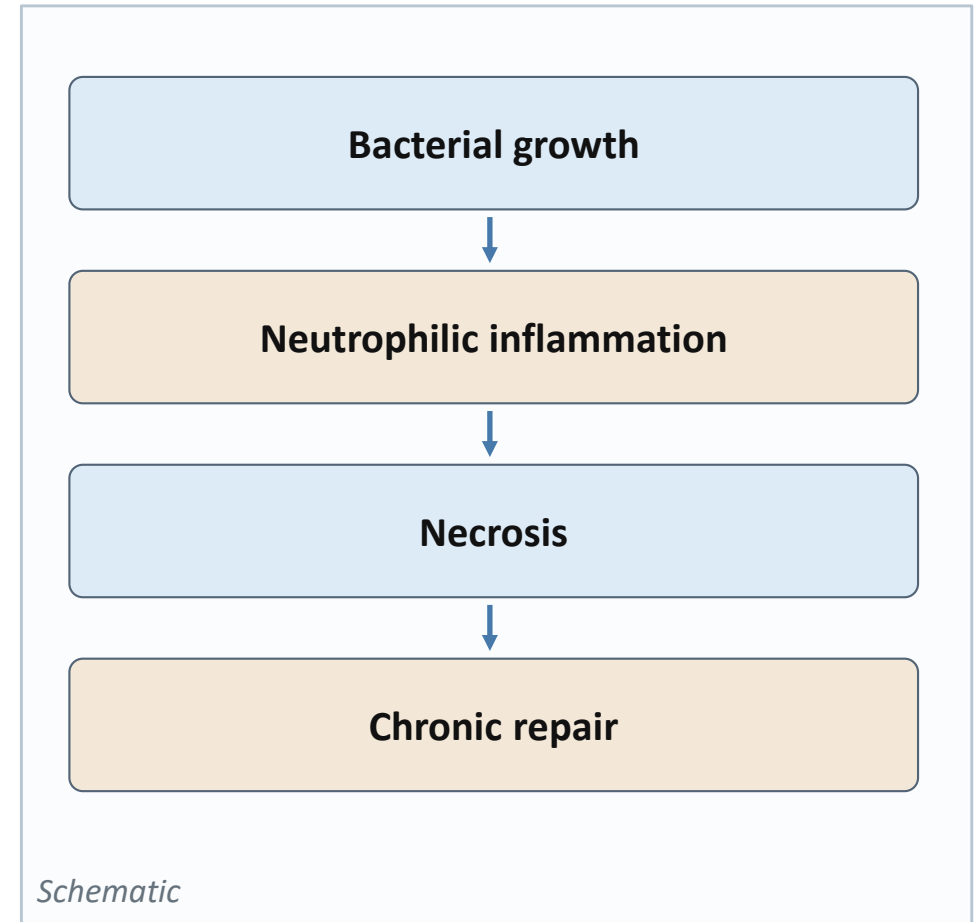


Age–location relationship

Age group	Vascular relationship	Distribution
Neonate	Metaphyseal vessels traverse plate	Metaphysis, epiphysis or both
Older child	Distinct metaphyseal circulation	Metaphysis typical
Adult	Metaphyseal–epiphyseal anastomoses	Epiphyseal / subchondral more common

Inflammation evolves over time

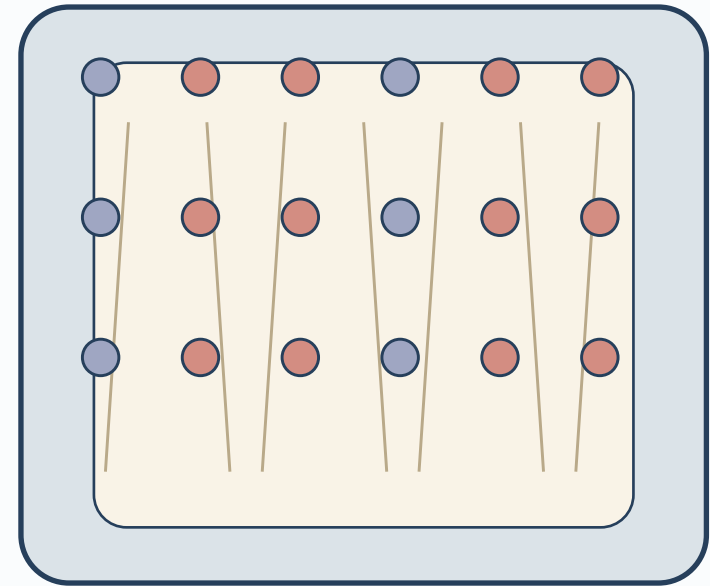
- Acute infection begins with bacterial multiplication.
- Neutrophilic inflammation causes early suppurative tissue injury.
- Bone and marrow necrosis can develop rapidly.
- Chronic inflammation and reactive repair become prominent as the process evolves.



Early suppurative inflammation

- Bacterial proliferation evokes a neutrophil-rich inflammatory response.
- Purulent exudate accumulates in infected marrow spaces.
- Neutrophil-dominated inflammation is an acute-stage clue.
- The inflammatory process can extend beyond its initial medullary focus.

Neutrophil-rich infiltrate (schematic)

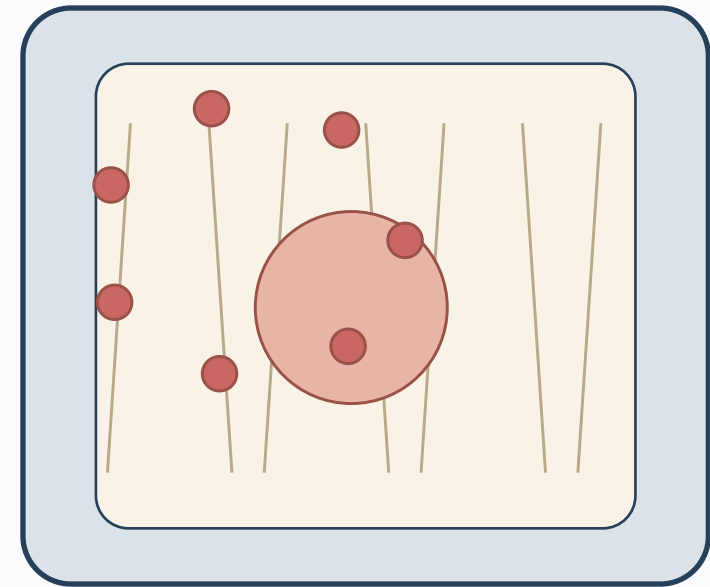


Schematic

Bone and marrow necrosis

- Necrosis of bone cells and marrow within about 48 hours.
- Infection and inflammation damage tissues and impair local viability.
- Devitalized bone is a substrate for persistent infection.
- Necrosis becomes central to chronic morphologic complications.

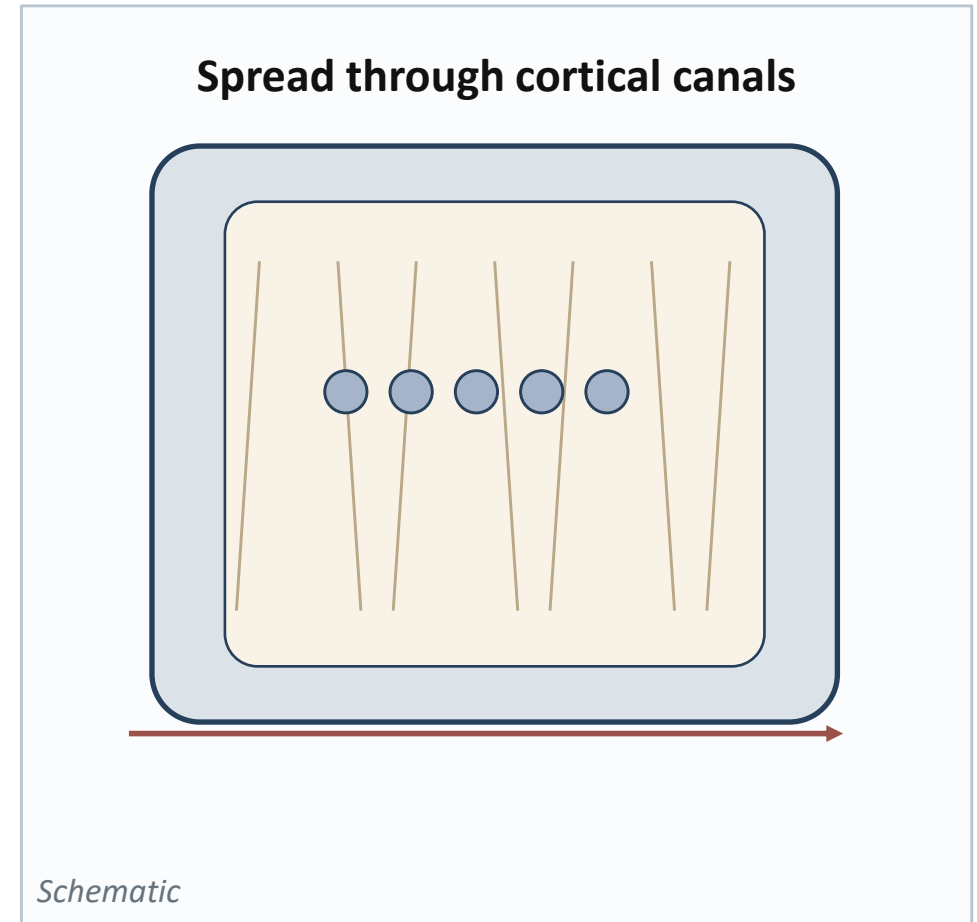
Suppurative focus



Schematic

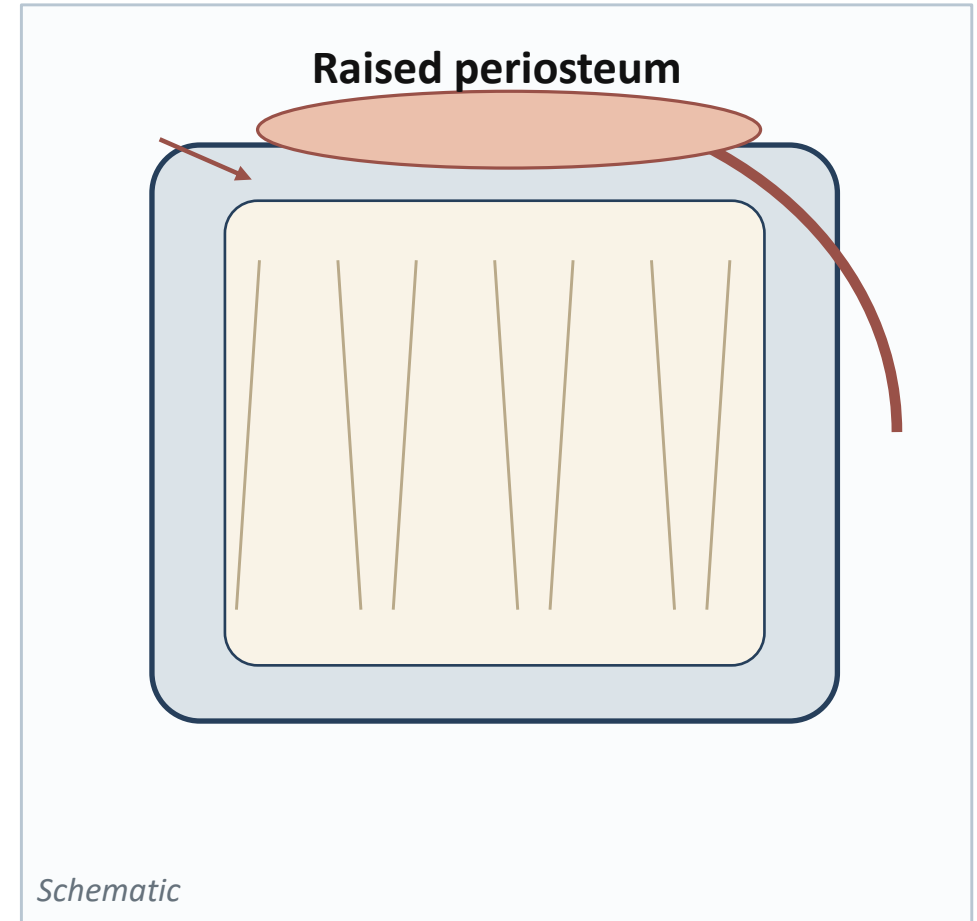
Spread through Haversian canals

- Bacteria and inflammation extend through intracortical Haversian canals.
- The process can reach the outer cortex and periosteum.
- An intramedullary infection is therefore not necessarily contained within marrow.
- Cortical spread links deep infection to periosteal abscess formation.



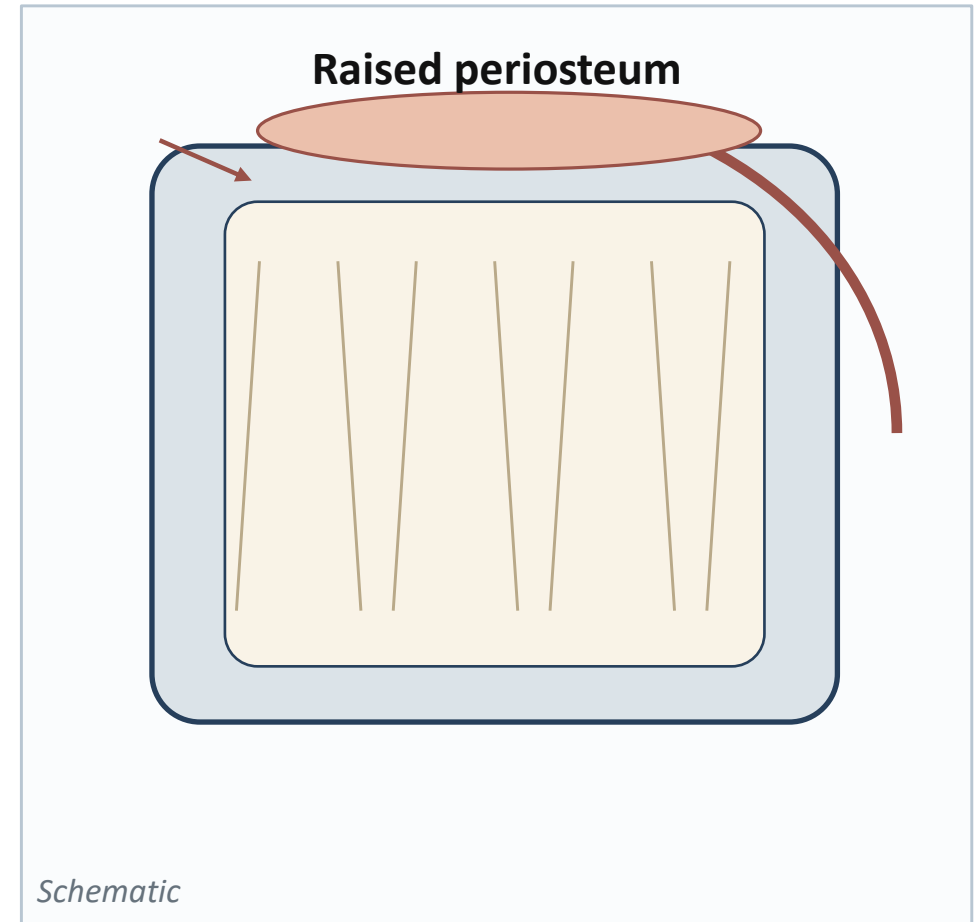
Why the pediatric periosteum matters

- The periosteum is comparatively loosely attached to the cortex in children.
- Suppuration can lift it away from bone.
- A dissecting subperiosteal collection may spread along the cortical surface.
- Separation jeopardizes a component of cortical blood supply.

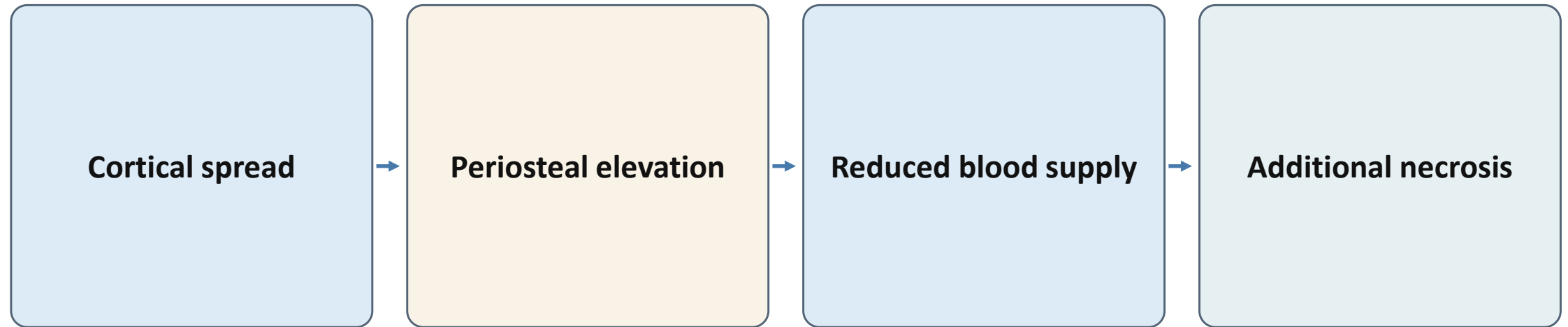


Subperiosteal abscess formation

- Purulent material accumulates beneath detached periosteum.
- The abscess may track longitudinally along the bone surface.
- The lifted periosteum restricts its normal vascular contribution.
- Consequent ischemia aggravates bone necrosis.



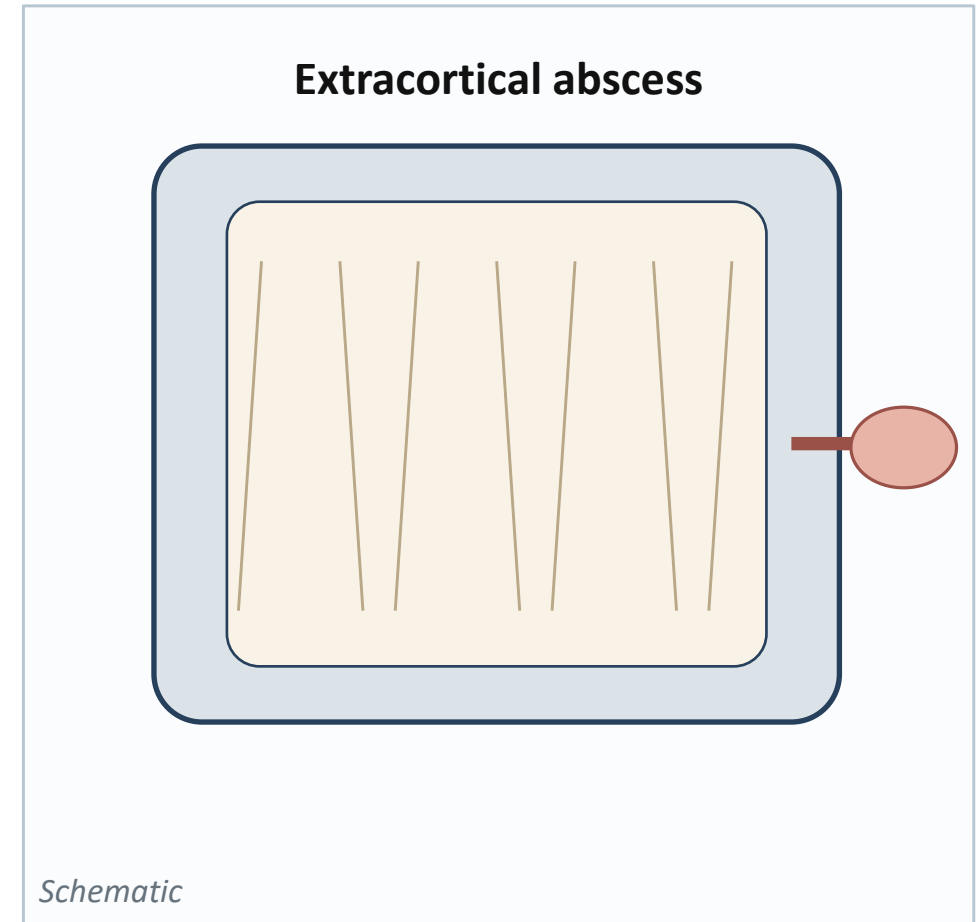
Vascular injury amplifies necrosis



The inflammatory process and ischemic injury reinforce one another.

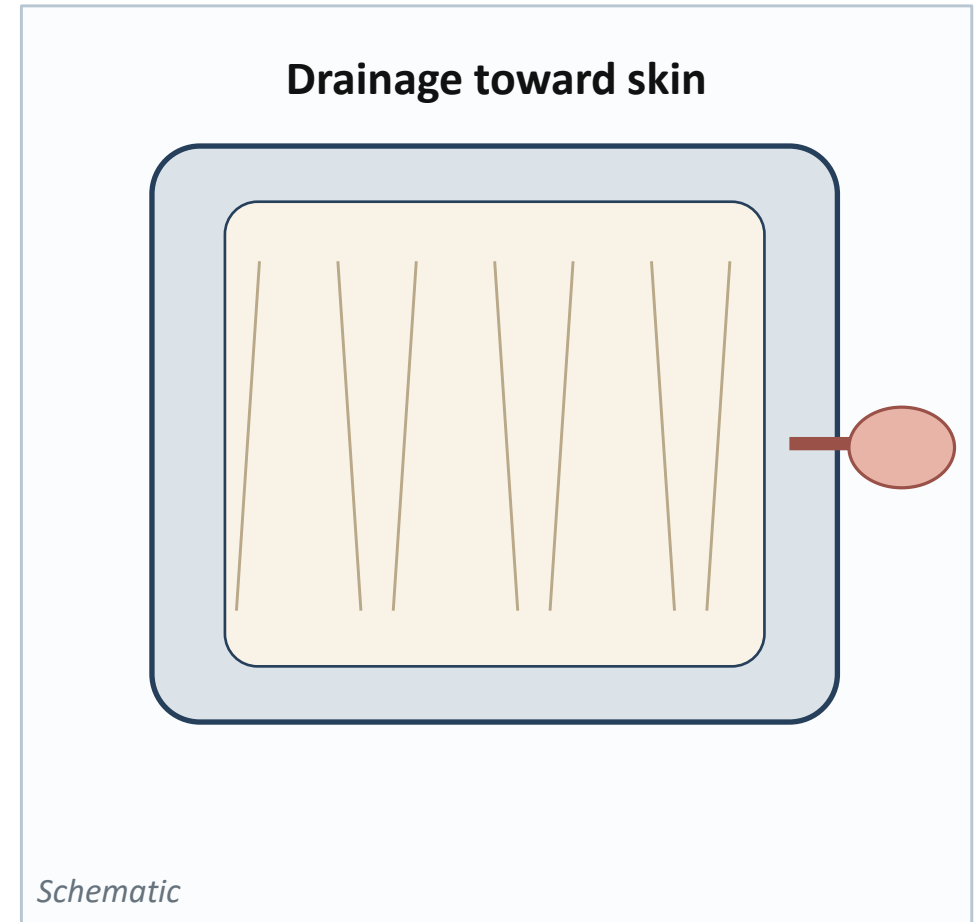
Infection can reach soft tissues

- A subperiosteal abscess can extend beyond periosteal confines.
- Soft tissue abscesses may form after periosteal rupture.
- The extramedullary collection is a consequence of cortical and periosteal extension.
- Recognition of spread matters for drainage planning.



Formation of a draining sinus

- A deep soft tissue abscess may channel toward the skin.
- An external sinus creates a route for chronic drainage.
- The tract is a morphologic sign of extension beyond the bone.
- Long-standing draining tracts have important late complications.

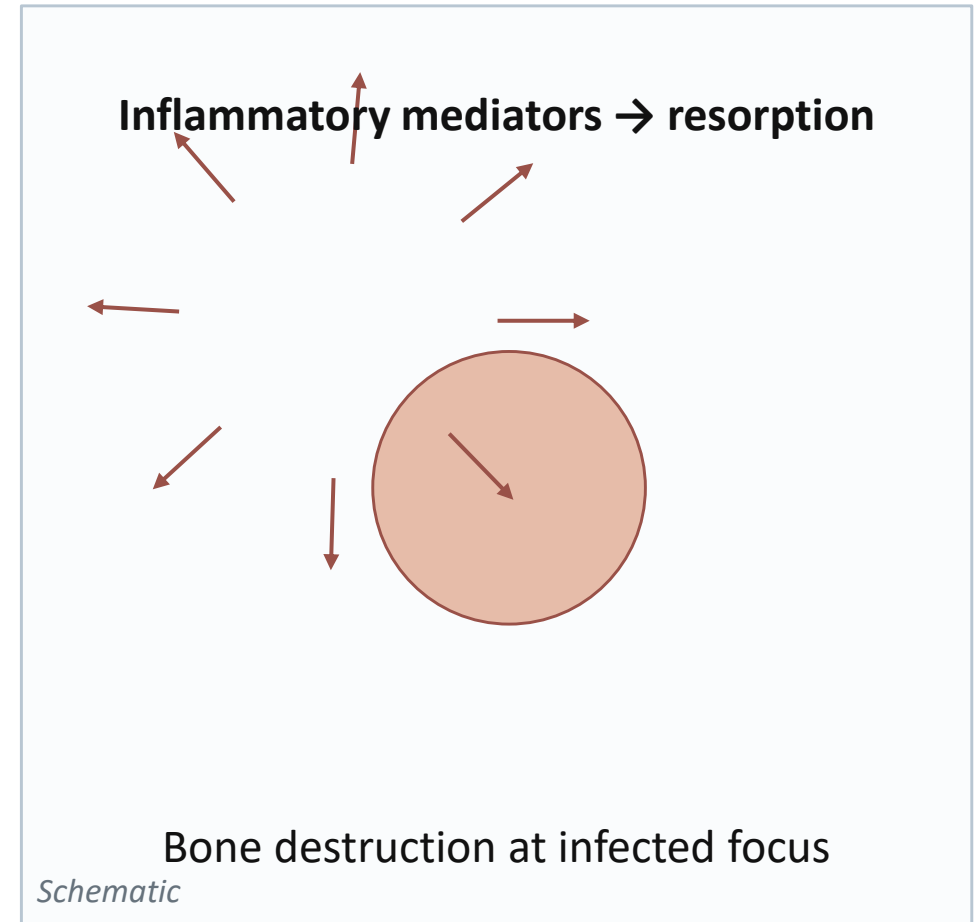


The first week: Chronic response emerges

- Chronic inflammatory cells are recruited as acute inflammation evolves.
- Their cytokines promote bone resorption and tissue remodeling.
- Fibrous tissue begins to grow into the affected region.
- Reactive bone may be laid down at the focus periphery.

Cytokine-associated bone resorption

- Chronic inflammatory cells release cytokines into the infected region.
- These signals to bone resorption.
- Resorption contributes to osseous structural loss.
- Destruction and repair may coexist in a chronic lesion.

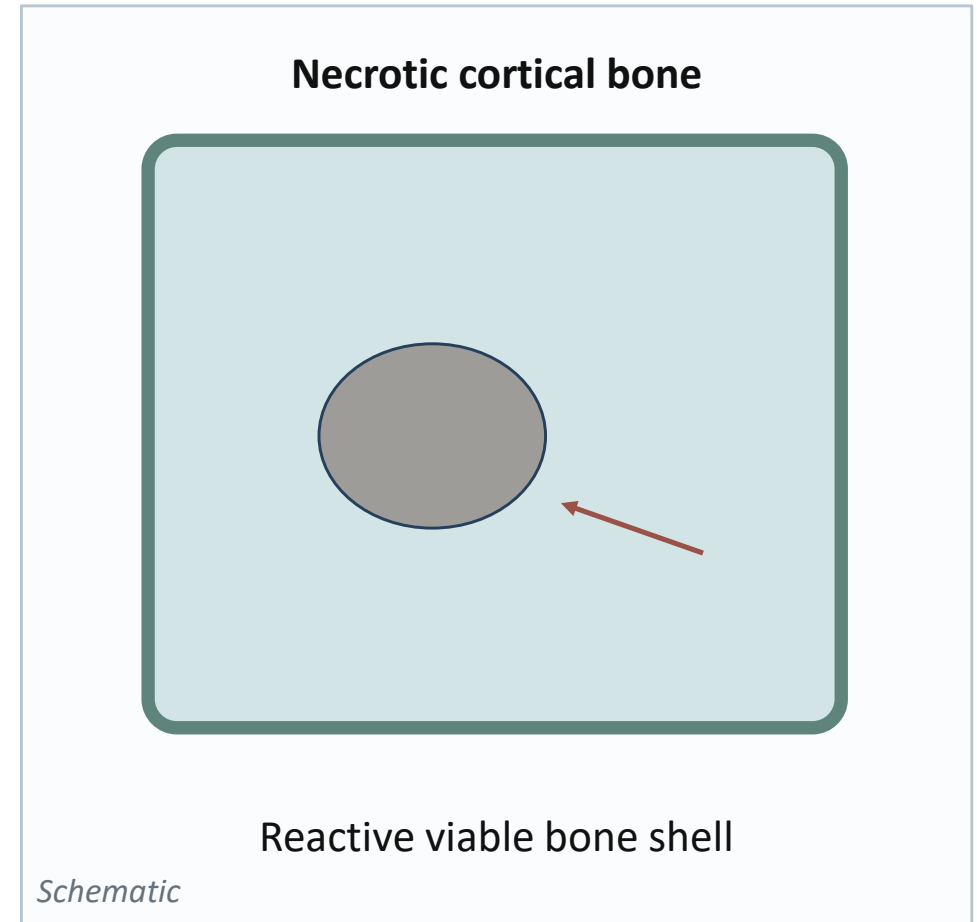


Fibrous tissue ingrowth

- A healing response follows tissue destruction.
- Fibrous tissue grows into affected marrow as inflammation persists.
- Marrow fibrosis is a common histologic feature of chronic disease.
- Fibrosis does not imply eradication of the underlying infection.

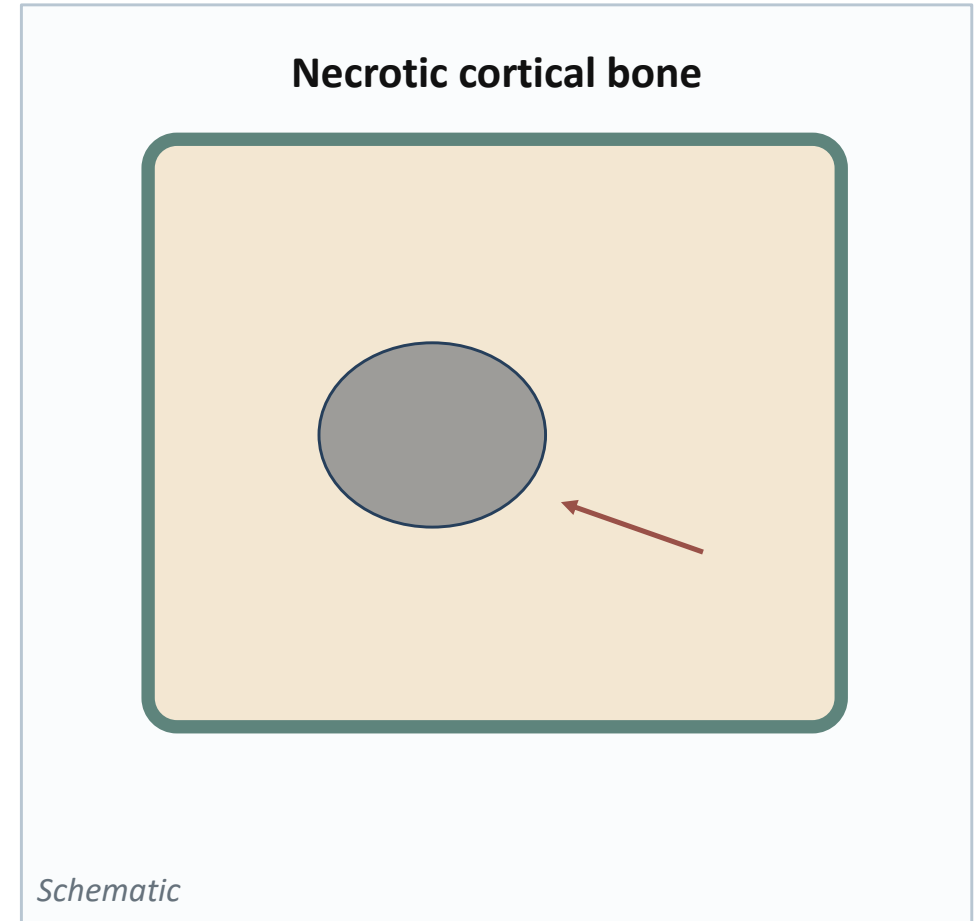
Reactive new bone at the periphery

- Repair-related new bone forms around infected, damaged tissue.
- Reactive bone formation may accompany ongoing resorption.
- The peripheral shell can encircle devitalized bone.
- This reaction provides the anatomical basis of an involucrum.



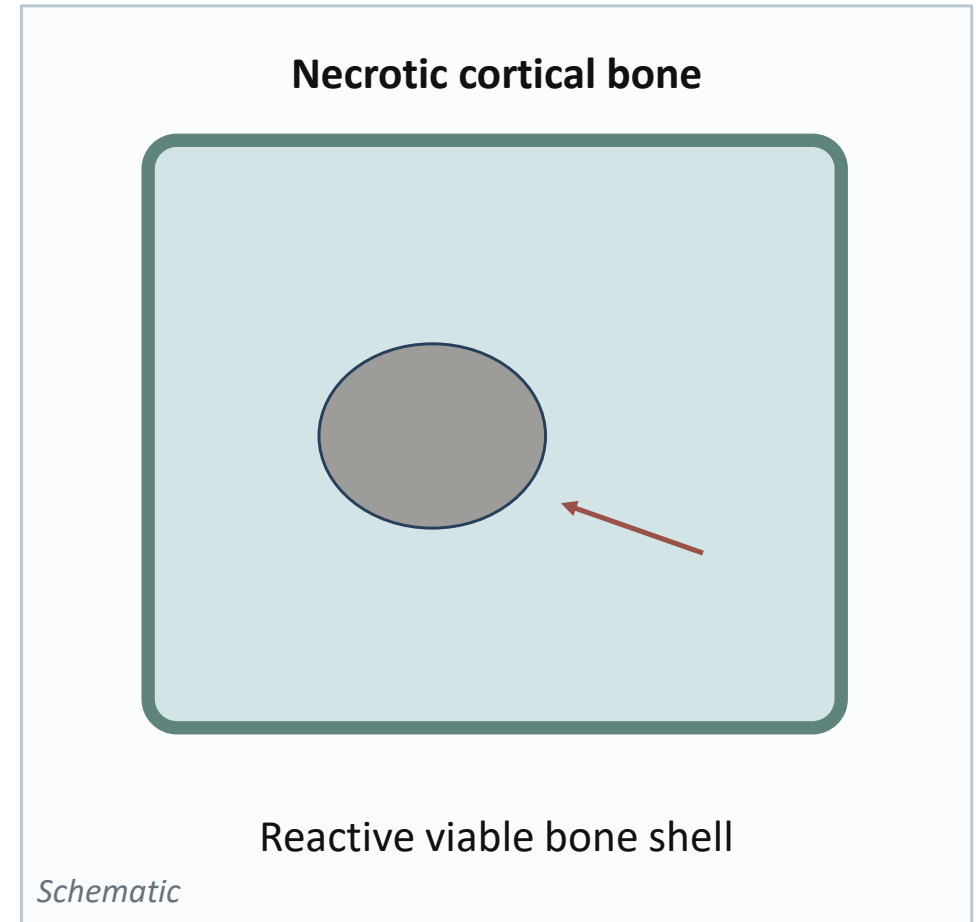
Sequestrum: Devitalized bone

- A sequestrum is a fragment or region of dead bone.
- It remains within an infected or previously infected osseous focus.
- Necrotic tissue is separated from viable reparative bone.
- The finding is characteristic of established chronic osteomyelitis.



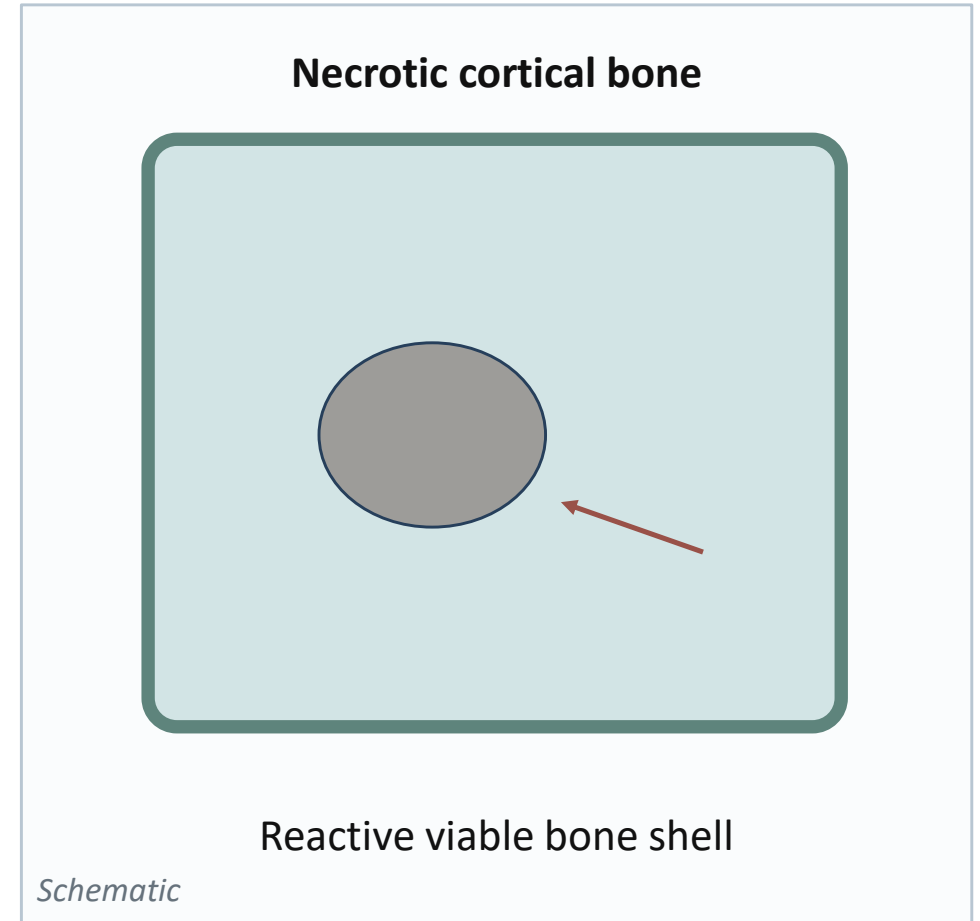
Involucrum: Reactive viable bone shell

- Involucrum is new living bone formed around devitalized infected bone.
- It represents reactive peripheral bone deposition.
- The sequestrum–involucrum relationship is central to chronic gross morphology.
- The shell may contain a route for a draining sinus.



Sequestrum and involucrum together

- The inner sequestrum is devitalized native bone.
- The outer involucrum is newly formed viable bone.
- The residual drainage path can traverse this shell.
- The conceptual cross-section is schematic; It is not a specimen photograph.



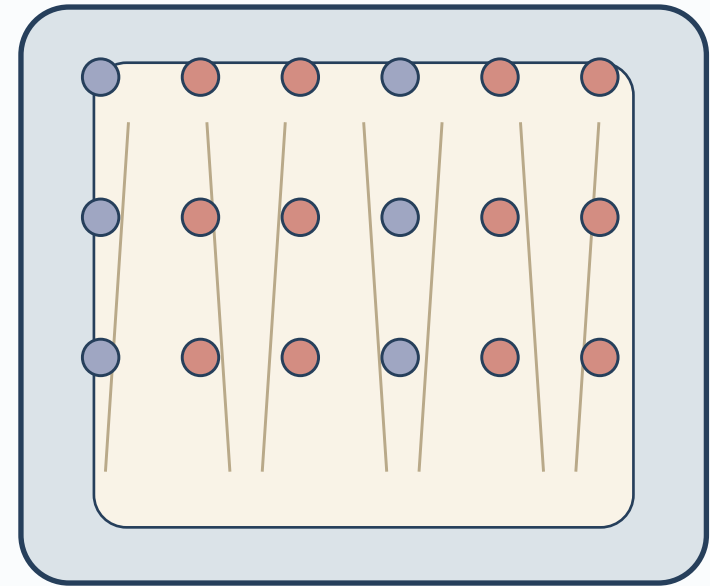
Stage-associated morphology

Stage	Dominant lesion	Supporting tissue response
Acute	Bacterial growth; Neutrophils	Bone / marrow necrosis
Evolving	Periosteal extension; Abscess	Osteolysis; Fibrous ingrowth
Chronic	Sequestrum; Chronic infiltrate	Marrow fibrosis; Reactive bone

Acute histology: What matters

- Neutrophils dominate the acute inflammatory infiltrate.
- Acute marrow injury and bone cell necrosis may coexist.
- Microscopic findings depend on disease stage and sampling location.
- The diagram is illustrative, not an H&E image.

Neutrophil-rich infiltrate (schematic)

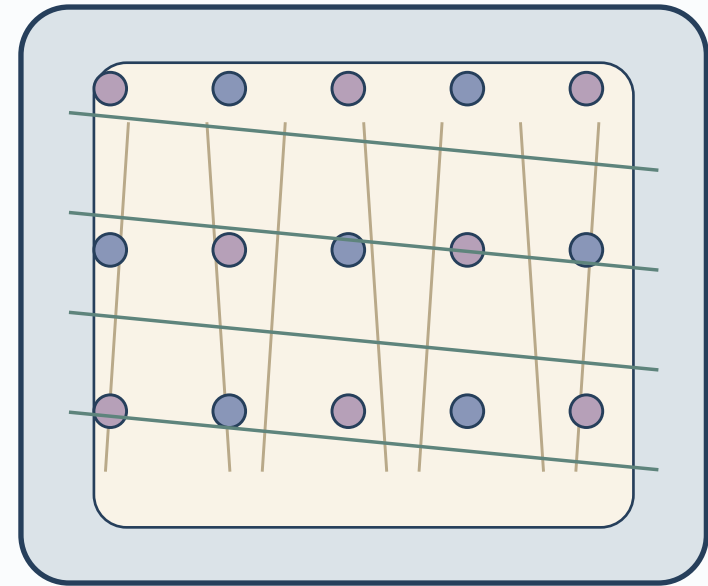


Schematic

Chronic histology: Mixed repair and injury

- Marrow fibrosis is a typical chronic change.
- Lymphocytes and plasma cells form a chronic inflammatory infiltrate.
- Sequestrum may be present alongside reactive new bone.
- The histologic pattern varies with location and disease duration.

Lymphocytes / plasma cells + fibrosis



Schematic

Acute systemic presentation

- Hematogenous disease may cause malaise, fever and chills.
- Leukocytosis can accompany the systemic inflammatory response.
- Intense throbbing pain may localize to the involved region.
- A severe systemic syndrome is only one clinical presentation.

Systemic

Fever · malaise · chills

Local

Intense pain

Laboratory

Leukocytosis

Schematic

Subtle presentations also occur

- Some infants present with unexplained fever rather than clear focal complaints.
- Adults may report only localized bone pain.
- Absence of a dramatic febrile illness does not rule out osteomyelitis.
- Clinical interpretation depends on age and site of infection.

Infant

Unexplained fever

Child

Acute fever + bone pain

Adult

Localized pain possible

Schematic

Age-specific clinical patterns

Population	Possible presentation	High-priority issue
Infant	Unexplained fever	Adjacent joint spread
Child	Acute systemic illness with bone pain	Long-bone hematogenous focus
Adult	Localized pain, sometimes without marked systemic features	Injury / procedure / diabetes context

Plain radiographs: Destructive response

- A lytic focus of bone destruction.
- Associated reactive bone formation may be visible.
- The film depicts structural consequence, not the microbial species.
- Radiographic information must be integrated with clinical and microbiological findings.

MRI is emphasized for detection

- MRI as more sensitive and specific for detecting osteomyelitis changes than plain radiographs.
- MRI helps characterize a suspected infection when bone disease is not fully defined by radiography.
- Imaging may support suspicion and lesion localization.
- MRI alone does not identify the pathogen.

Imaging and culture answer different questions

- Radiographs reveal lysis and reactive bone.
- MRI more sensitively detects changes of osteomyelitis.
- Biopsy and microbial culture are used to identify the organism.
- Structural evidence and etiologic evidence are complementary.

Radiograph

Lysis + reactive bone

MRI

**More sensitive /
specific**

Biopsy / culture

**Pathogen
identification**

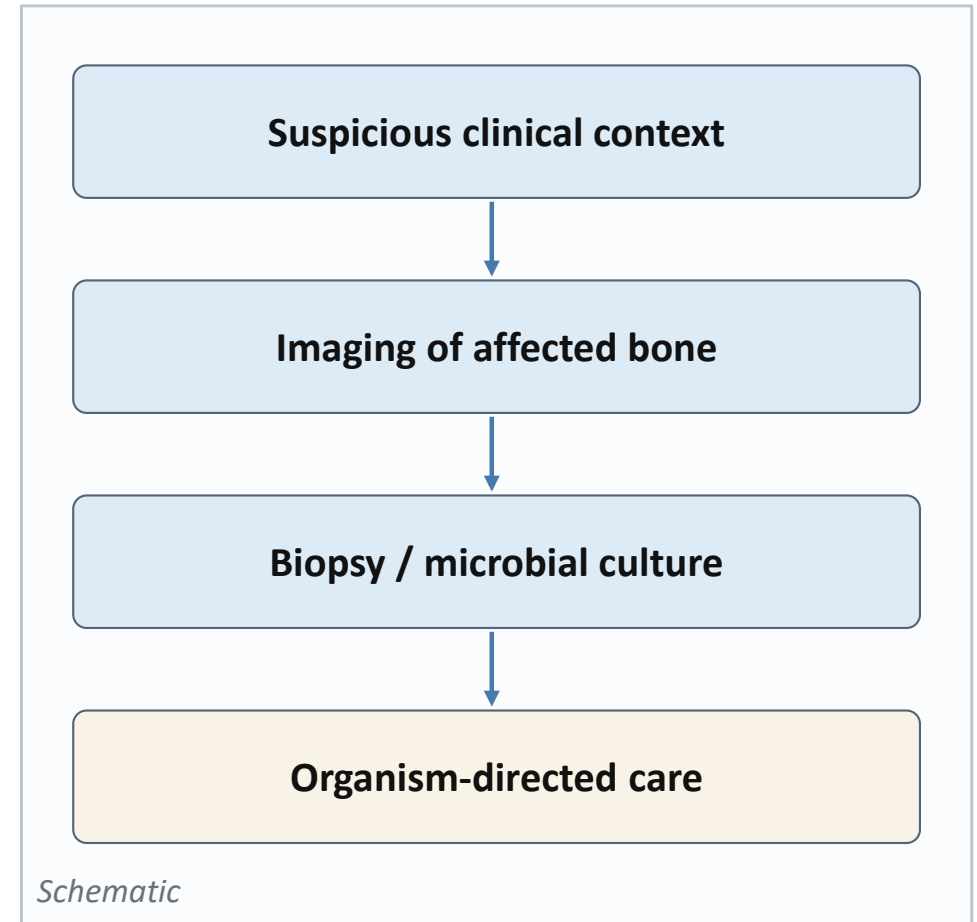
Schematic

Biopsy and microbial culture

- Microbiological cultures should be obtained whenever possible.
- The chapter emphasizes biopsy and microbial culture for pathogen identification in most instances.
- Identification is incomplete in a substantial portion of cases.
- The culture result supports etiologic classification rather than merely visual diagnosis.

Diagnostic reasoning: Integrated approach

- Assess age, route of infection and host vulnerability.
- Recognize local symptoms, systemic illness or subtle presentations.
- Use imaging to characterize osseous changes.
- Obtain suitable material for microbiologic identification when possible.



Antibiotics and surgical drainage

- Antibiotics combined with surgical drainage are usually curative.
- Management must address both causative bacteria and collections of infection.
- Necrotic bone or inadequately drained infection can impede resolution.
- The text does not specify antimicrobial regimens or treatment durations.

Why some acute infections persist

- The chapter reports failure of acute infection to resolve in 5%–25% of cases.
- Delayed diagnosis increases the risk of persistence.
- Extensive necrosis and inadequate antibiotics or debridement are adverse factors.
- Weakened host immunity further reduces the chance of uncomplicated resolution.

Delay in diagnosis

Extensive necrosis

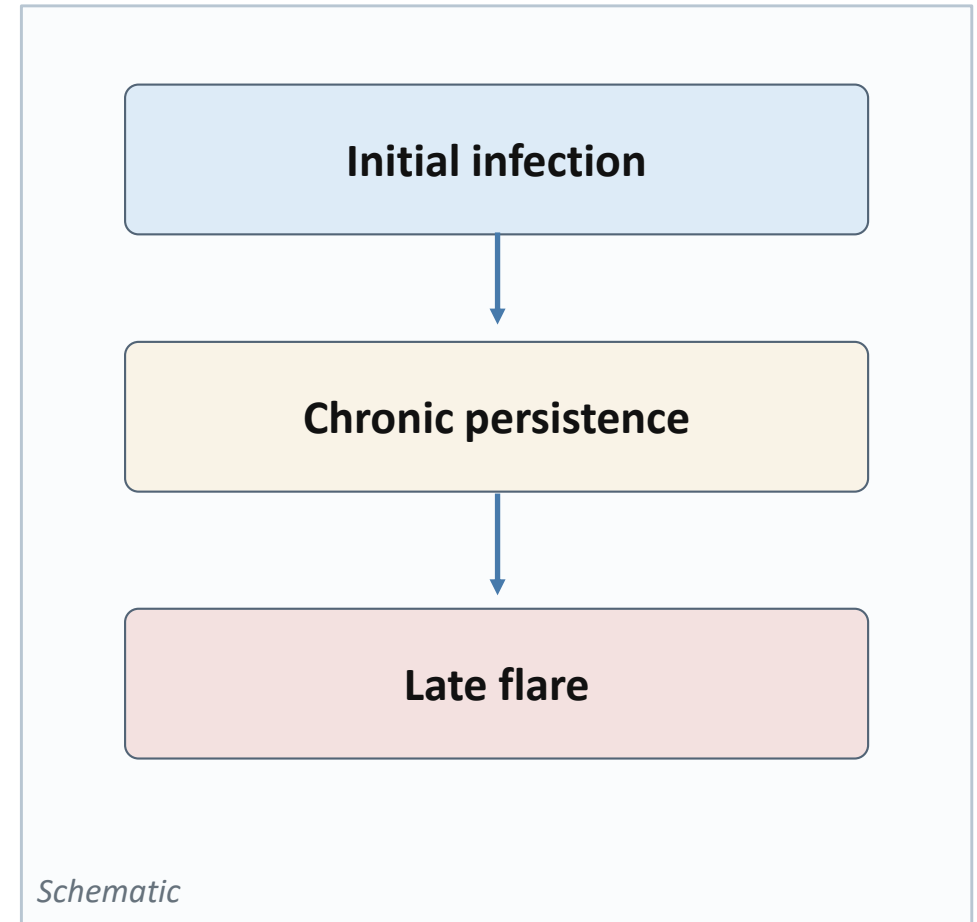
Inadequate antimicrobials / debridement

Weakened immunity

Schematic

Chronic disease may flare after dormancy

- Chronic osteomyelitis may remain quiescent for years.
- Spontaneous flares can interrupt prolonged clinical dormancy.
- Persistent devitalized bone and altered local tissue responses help explain chronicity.
- A quiet interval does not establish that a chronic focus is eradicated.



Pathologic fracture as a complication

- Osteolysis, structural destruction and tissue necrosis can weaken bone.
- Compromised osseous integrity may culminate in a pathologic fracture.
- The complication reflects local mechanical consequences of chronic infection.
- Clinical risk depends on the location and extent of damaged bone.

Sepsis, endocarditis and amyloidosis

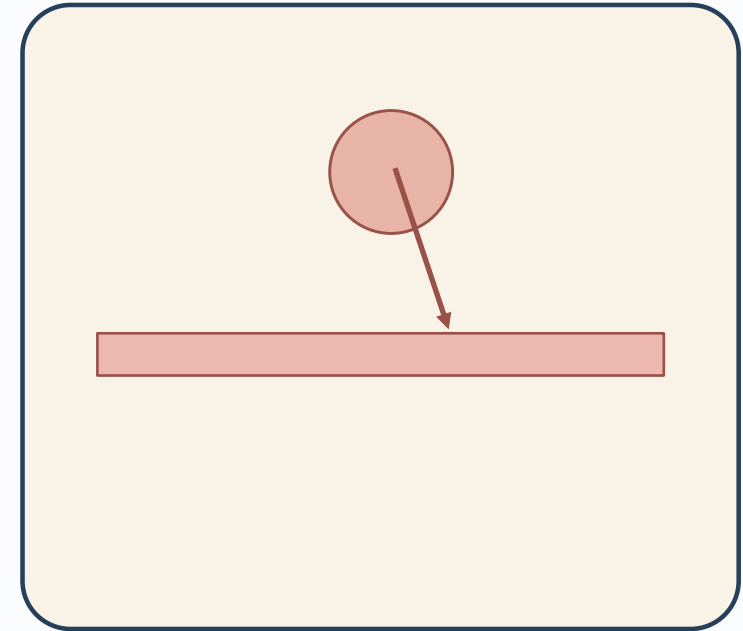
- Chronic osteomyelitis can have systemic complications.
- Sepsis and endocarditis are listed among these complications.
- Secondary (reactive) amyloidosis is another recognized late consequence.
- These outcomes are clinically distinct from local bone destruction.

Malignancy in chronic osteomyelitis

- Squamous cell carcinoma arising within draining sinus tracts.
- Sarcoma may arise in infected bone.
- These are complications of long-standing chronic disease, not routine acute findings.
- A new destructive or proliferative change in a chronic lesion warrants careful assessment.

Septic arthritis in infants

- Epiphyseal bone infection can spread to the joint.
- Suppurative arthritis can destroy articular cartilage.
- Permanent disability is a possible consequence.
- Neonatal circulation makes this an important anatomic teaching point.



Epiphyseal focus → adjacent joint

Schematic

Local and systemic consequences

Category	Complication	Pathologic connection
Local	Abscess / draining sinus	Periosteal or soft tissue extension
Mechanical	Pathologic fracture	Loss of viable bone
Articular	Septic arthritis (infants)	Epiphysis-to-joint spread
Systemic	Sepsis; Endocarditis; Amyloidosis	Persistent infectious / inflammatory disease
Neoplastic	Sinus SCC; Bone sarcoma	Long-standing chronic lesion

Case: Febrile child with focal bone pain

An otherwise healthy school-age child develops fever, malaise and intense localized pain over a long bone after a minor skin infection. Imaging raises concern for osteomyelitis.

What route and typical osseous site are most plausible?

Case analysis: Pediatric hematogenous focus

- A preceding skin lesion can provide an entry point for transient bacteremia.
- Hematogenous infection is typical in otherwise healthy children.
- The metaphysis is the expected location in an older child.
- Biopsy and culture are useful for identifying the pathogen.

Case: Persistent pain after open fracture

An adult with a previously open tibial fracture has localized pain and repeated purulent drainage. Imaging shows destructive and reactive bone changes.

Which route and chronic morphologic findings should be considered?

Case analysis: Chronic post-traumatic infection

- Direct implantation after an open fracture is an important adult mechanism.
- Mixed bacterial infection is possible in this setting.
- Sequestrum, involucrum and a draining sinus support chronic osteomyelitis.
- Persistence should prompt attention to necrosis and adequacy of local drainage.

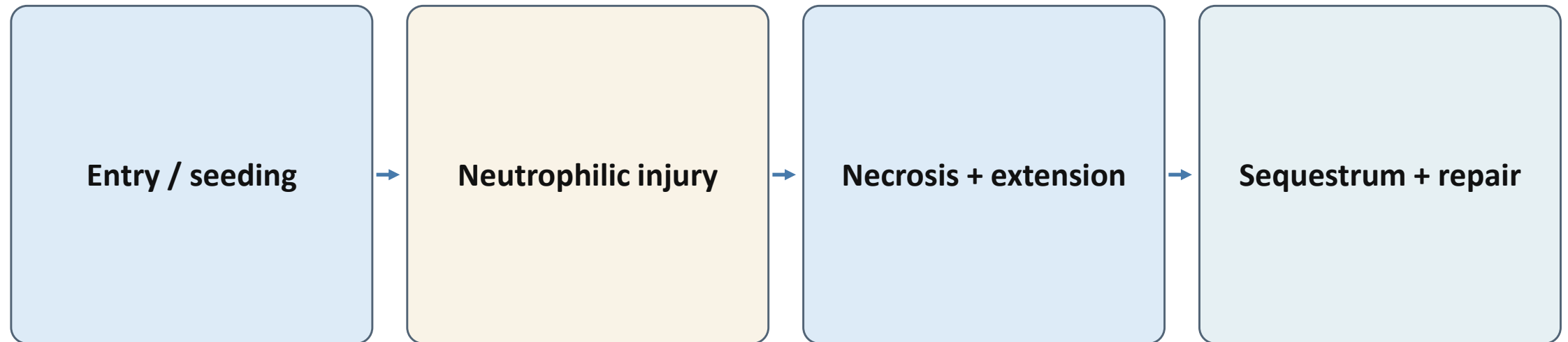
Pyogenic versus mycobacterial disease

Feature	Pyogenic osteomyelitis	Mycobacterial osteomyelitis
Common pattern	Suppuration / neutrophils	Caseating granulomas
Course	May be acute, chronic or recurrent	Often indolent; Can be destructive
Important site	Long bones in healthy children	Spine commonly involved (Pott disease)
Microbiology	Often S. Aureus in culture-positive cases	Mycobacterial infection

Separate infarction from infection

- Osteonecrosis means ischemic death of bone and marrow; It is not synonymous with pyogenic infection.
- Osteomyelitis denotes an inflammatory process, usually infectious.
- Bone infarction is relevant as a nidus for seeding in sickle cell disease.
- The two conditions can overlap clinically, but their initiating injury differs: Ischemia versus infection.

Integrated mechanism: From seeding to chronicity



Anatomic route, age-related circulation and host factors shape the sequence.

Five clinicopathologic conclusions

- Entry route and patient age strongly influence pathogen and site.
- Acute pyogenic infection is neutrophil-rich and can rapidly necrose bone.
- Periosteal elevation worsens vascular injury; Chronic lesions may form sequestrum and involucrum.
- Imaging localizes damage, while biopsy and culture guide etiologic identification.
- Chronic disease can relapse and cause local, systemic and rare neoplastic complications.

Primary reference

- Robbins, Cotran & Kumar. Pathologic Basis of Disease. 11th edition (2026).
- Chapter 26: Bones, Joints, and Soft Tissue Tumors.
- Pyogenic Osteomyelitis, pp. 1076–1077; surrounding Osteomyelitis morphology and clinical features.
- All anatomical illustrations in this lecture are original editable schematics, not specimen photographs.