

Infectious Arthritis

Mechanisms, morphology, clinicopathologic patterns

Suppurative · Mycobacterial · Viral · Lyme

Dr Abu Suraih Sakhri

MBBS, MD (Pathology)

www.abusuraisakhri.com

Learning outcomes

- Trace how microorganisms reach synovial joints and explain the significance of cartilage injury.
- Distinguish acute suppurative, mycobacterial, viral, and Lyme patterns using source-supported features.
- Interpret the diagnostic value of joint aspirate, infection context, and characteristic histopathology.

Infectious arthritis: The central definition

Infection involving articular structures

Infectious agents can reach the joint through blood, direct introduction, or spread from adjacent infected tissues.

One parent category, four major patterns

Suppurative

Acute bacterial

Often rapid, painful
monoarthritis

Mycobacterial

Chronic tuberculosis

Granulomas, pannus,
eventual fusion

Viral / Lyme

Viral: Acute to subacute

Lyme: Episodic large-joint
arthritis

Three routes by which joints become infected

Hematogenous
Dissemination



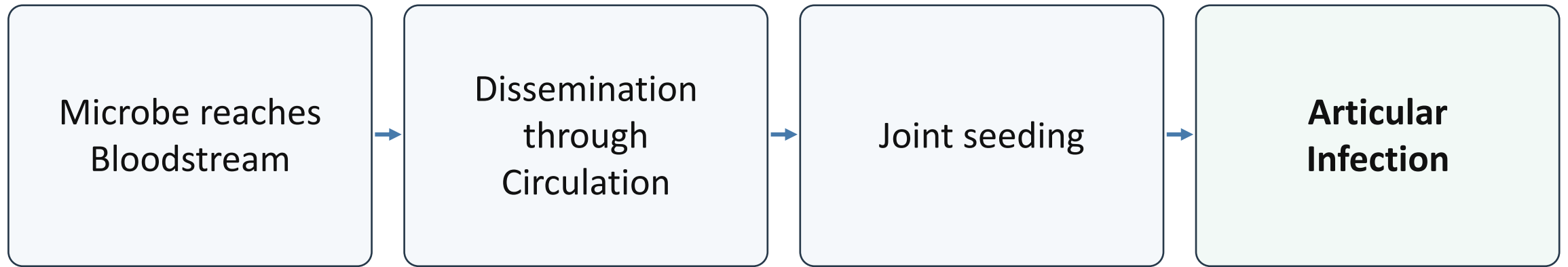
Direct
Inoculation



**Contiguous spread
From nearby infection**

Different routes converge on infection of articular structures.

Hematogenous route: Conceptual sequence



The diagram illustrates route of entry, not a molecular-level mechanism.

Direct inoculation versus contiguous spread

Direct inoculation

Organism is introduced directly into articular structures

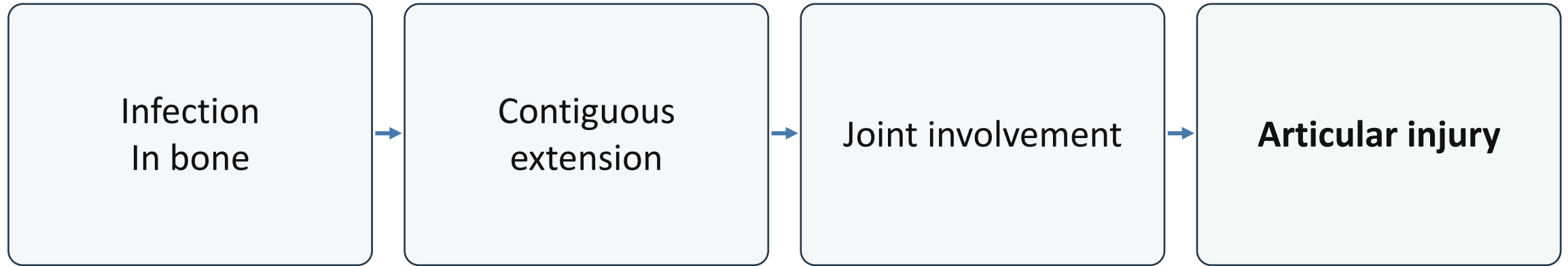
Route defined by local entry

Contiguous spread

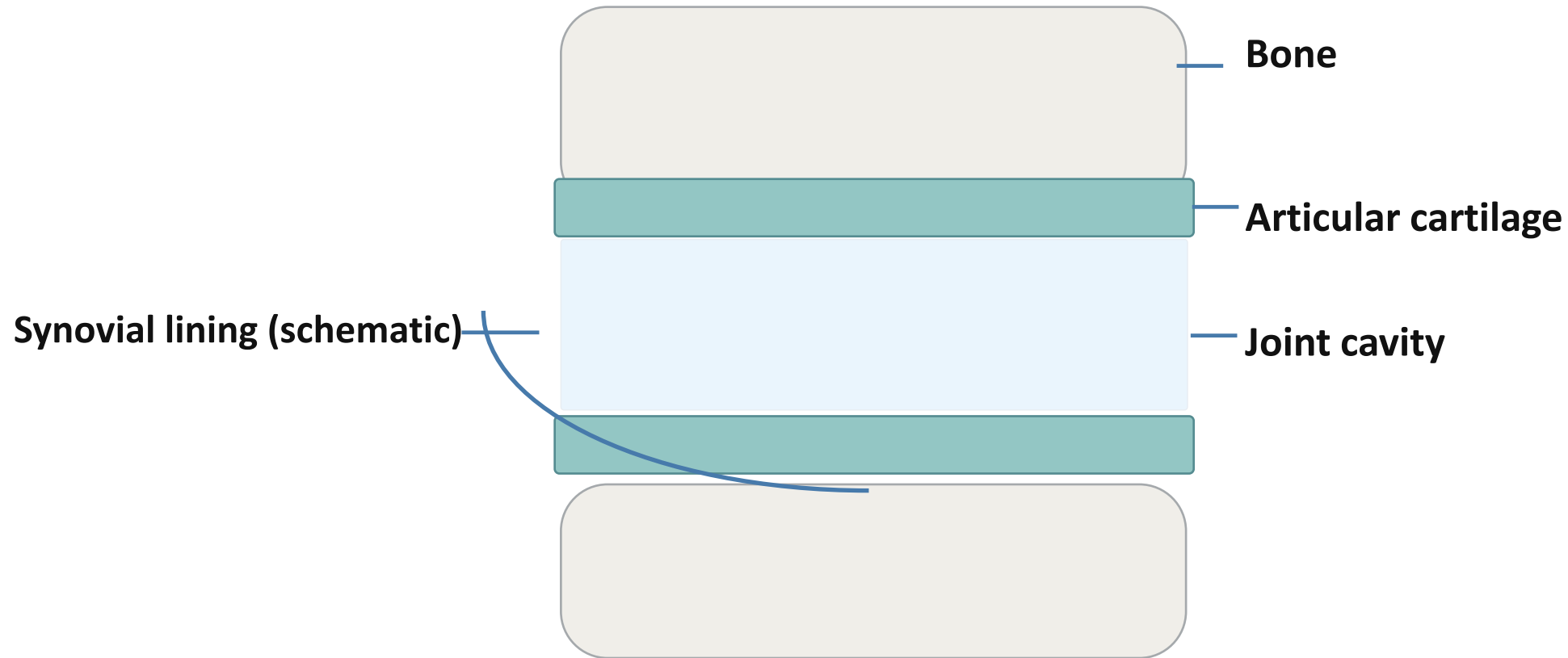
An infected nearby site involves the joint

Soft-tissue abscess or osteomyelitis

Neighboring osteomyelitis can involve the joint

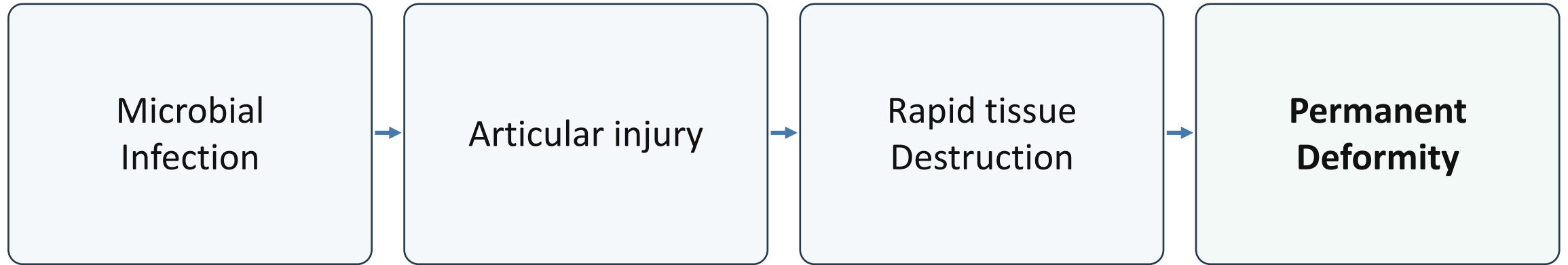


Why articular cartilage is vulnerable



Schematic. Cartilage has limited regenerative capacity compared with bone.

Joint infection can create lasting disability



The ultimate outcome depends on course and effective recognition/treatment.

Clinical tempo helps organize the differential

Abrupt

Suppurative bacterial arthritis

Acute pain, swelling, immobility

Insidious

Tuberculous arthritis

Gradually progressive joint pain

Variable / Episodic

Viral: Acute/subacute

Lyme: Migratory or recurring

The infectious arthritis evaluation framework

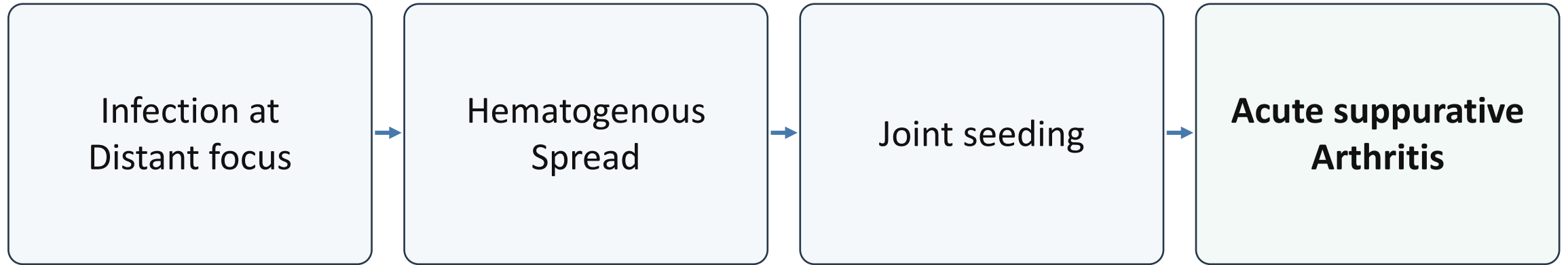
- Determine onset: Sudden acute, insidious progressive, or intermittent episodes.
- Identify joint pattern: Single joint versus limited or migratory large-joint involvement.
- Integrate exposure, immune status, and evidence of a causative organism.

Suppurative arthritis: An acute bacterial process

Rapid, destructive infection of a synovial joint

The etiologic organism varies with age, underlying disease, exposure, and anatomic site.

Bacteremia is the usual route in acute suppuration



The organism depends on clinical setting

- The anatomic site and patient context influence the responsible bacterium.
- Neonatal infections have distinctive routes and organisms.
- Host defense defects and medical conditions create pathogen-specific susceptibility.

Changing pathogen distribution after vaccination

Earlier burden

Haemophilus influenzae was historically an important cause

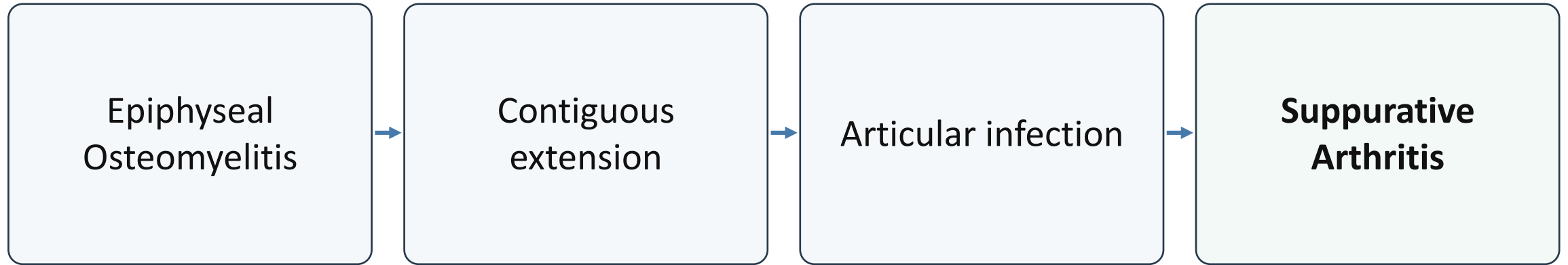
After effective vaccination

The incidence of H. Influenzae arthritis has declined

Current U.S. Setting

Staphylococcus aureus is most common

A neonatal pathway: Adjacent bone infection



This contiguous route in neonates.

Illustrative age- and host-linked pathogen clues

All age groups (U.S.)

Staphylococcus aureus

Leading bacterial cause

Neonates

Group B Streptococcus

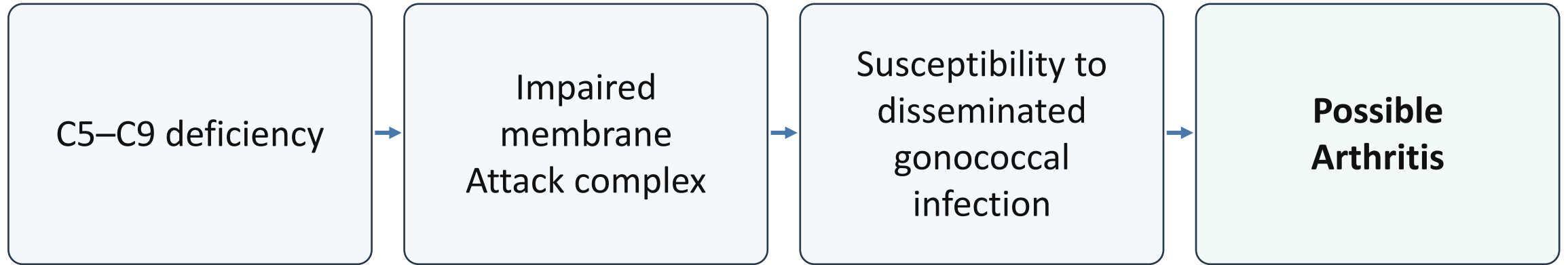
May accompany adjacent
epiphyseal infection

Sickle cell disease

Salmonella

Heightened infection
susceptibility

Terminal complement deficiency and gonococcal spread



Additional predisposing conditions

- Congenital or acquired immunodeficiencies.
- Debilitating illnesses and preexisting chronic arthritis.
- Trauma involving the joint and intravenous substance use.

Risk factor and pathogen clues

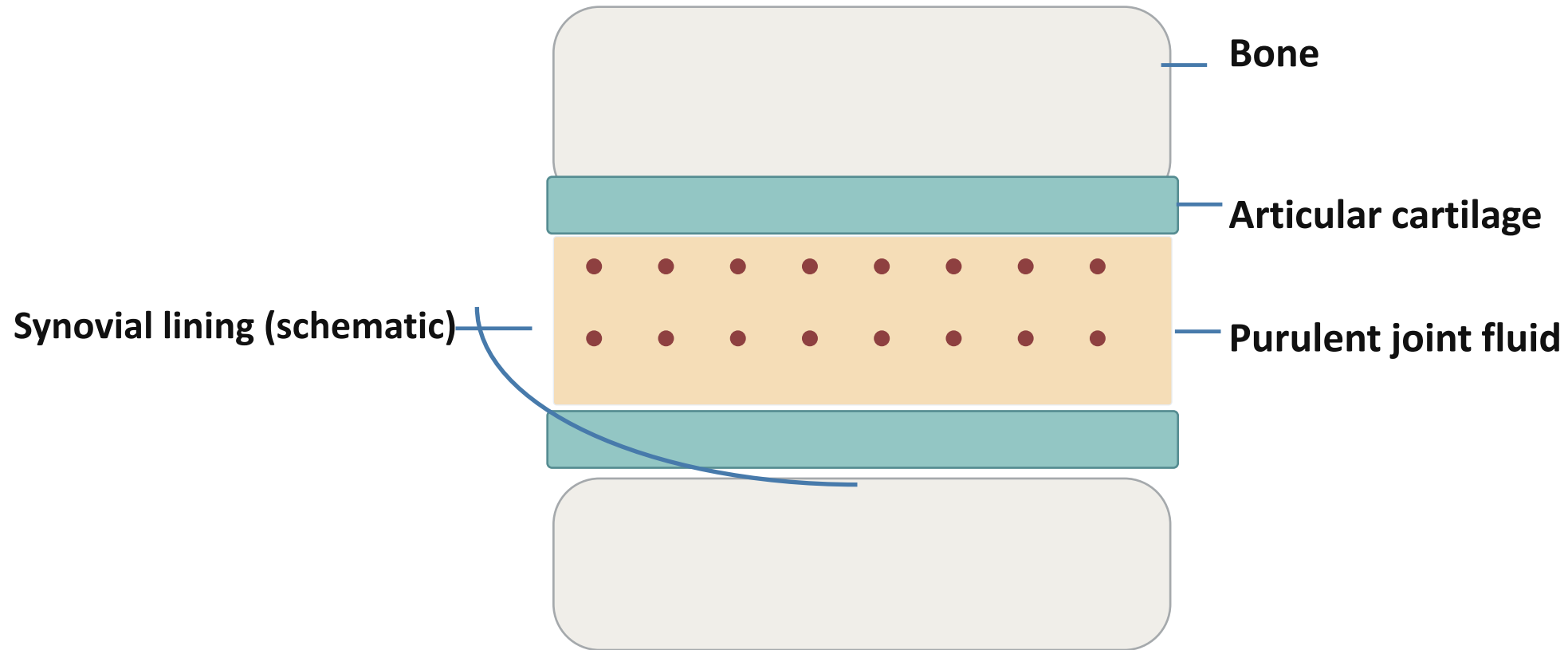
Clinical setting	Source-linked organism or pattern
General U.S. bacterial arthritis	Staphylococcus aureus most common
Neonatal infection	Group B Streptococcus; possible osteomyelitis spread
Sickle cell disease	Salmonella susceptibility
Complement C5–C9 deficiency	Disseminated gonococcal infection / arthritis
Intravenous substance use	Greater axial joint involvement

Classic acute presentation

Sudden pain, swelling, and limited joint motion

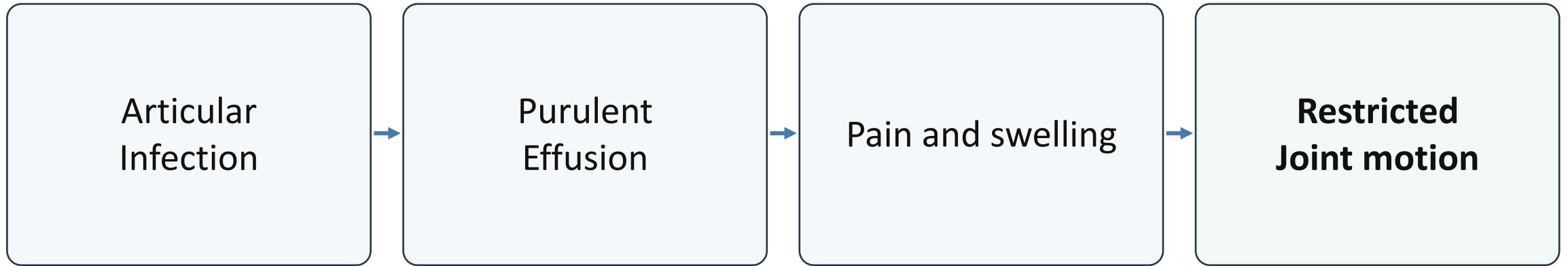
The rapid clinical onset is a major distinction from insidious tuberculous arthritis.

Suppuration within the joint cavity



Conceptual purulent effusion. Not a clinical or histologic specimen.

Inflammation explains the acute syndrome



Clinical sequence is a mechanistic teaching synthesis, not a source-derived quantitative pathway.

Systemic inflammatory signs are common

- Fever is a frequent accompanying feature.
- Leukocytosis is often observed.
- C-reactive protein is commonly elevated.

Typical appendicular joint involvement

Lower extremity

Knee

Hip

Upper extremity

Shoulder

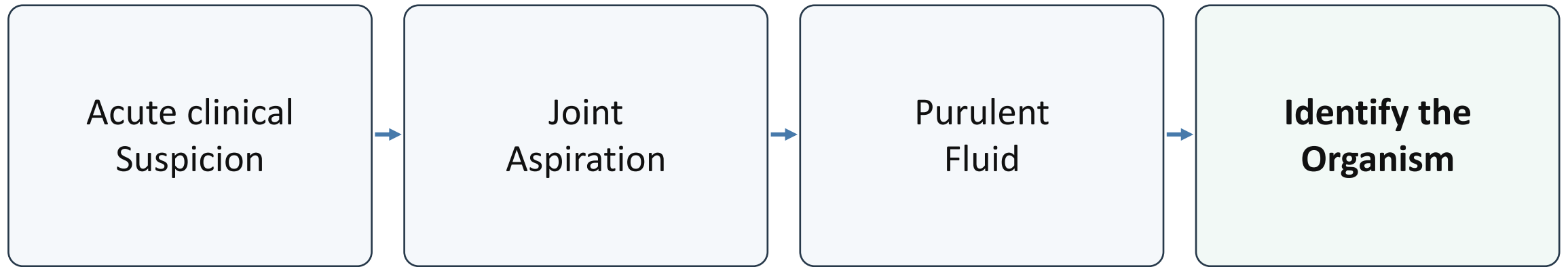
Elbow and wrist

Other listed site

Sternoclavicular joint

An important possible
location

The diagnostic hinge: Aspiration of an affected joint

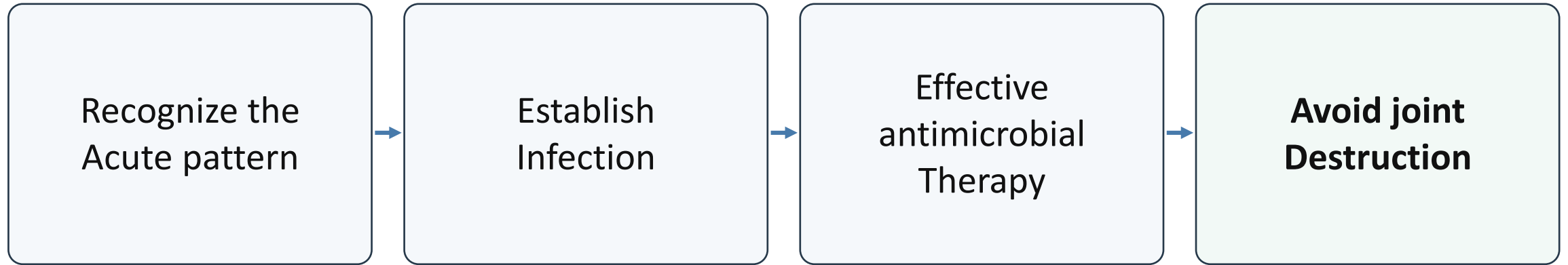


Source states joint aspiration is diagnostic if purulent fluid yields an identifiable organism.

Microbiologic attribution matters

- A purulent aspirate provides evidence of suppuration.
- Identifying the infectious organism establishes the etiologic diagnosis in the setting described.
- Neither leukocytosis nor raised CRP substitutes for joint-level organism identification.

Why early diagnosis changes the outcome



Robbins specifically states prompt recognition and effective antimicrobial treatment can prevent destruction.

Abrupt versus indolent joint infection

Suppurative

Sudden painful swollen joint

Restricted motion; Acute inflammation

Mycobacterial

Insidious, progressively painful joint

Granulomas; Pannus; Late joint-space loss

Tuberculous arthritis: The chronic pattern

Progressive joint infection caused by *Mycobacterium tuberculosis*

It primarily in adults.

Tuberculous organisms reach joints in two ways



An alternative route is direct extension from adjacent osteomyelitis.

Contiguous tuberculosis route

Adjacent tuberculous
Osteomyelitis



Spread into joint



**Chronic granulomatous
Arthritis**

Mycobacterial arthritis: Cumulative tissue damage

Insidious
Onset

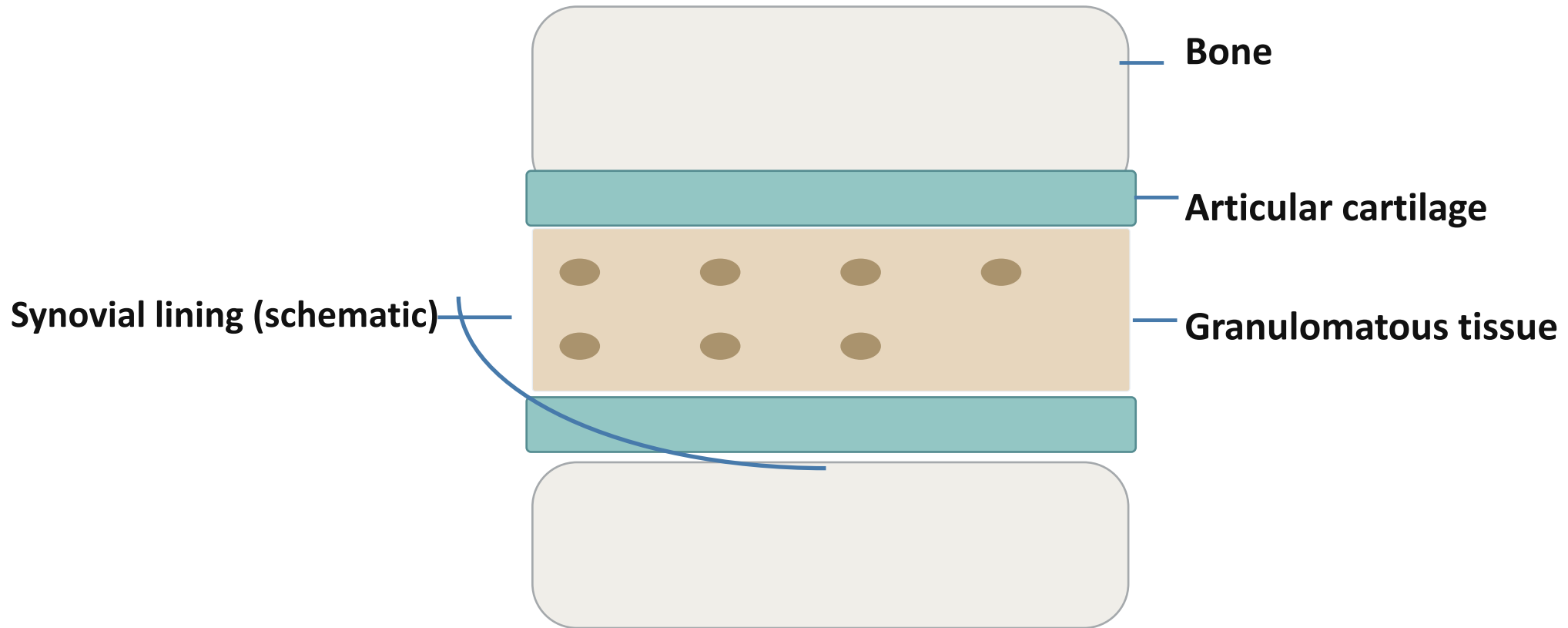
Synovial
Pannus

Caseating
Granulomas

Fibrous
Ankylosis

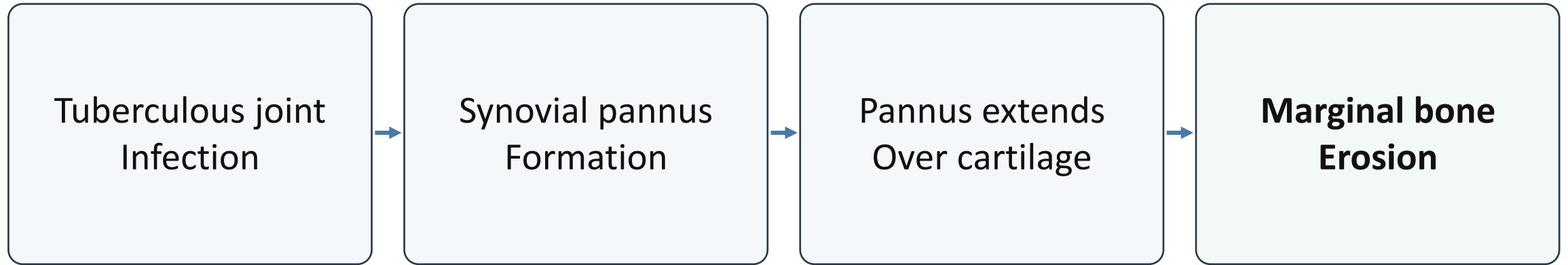


Mycobacterial articular damage

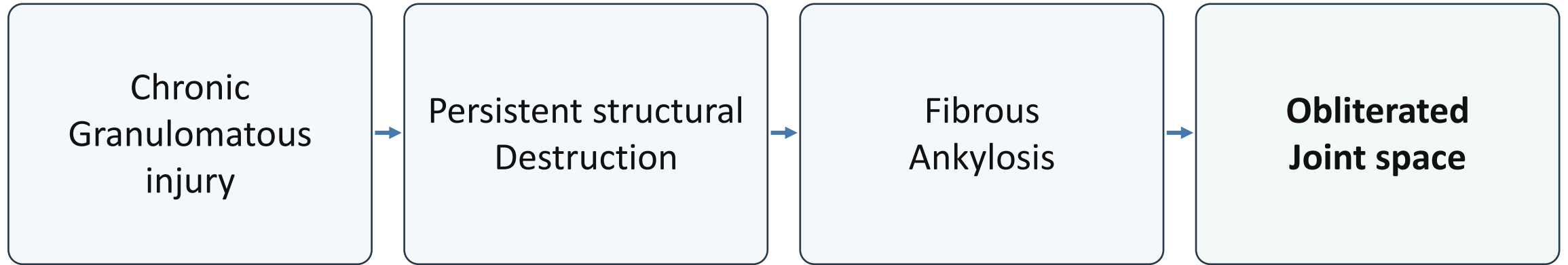


Schematic representation of granulomatous tissue, not an H&E photomicrograph.

Synovial pannus can advance over cartilage



Late tuberculosis can obliterate the joint space



Weight-bearing joints in mycobacterial arthritis

Most frequent

Hip

Weight-bearing joint

Next

Knee

Weight-bearing joint

Then

Ankle

Weight-bearing joint

Distinguishing bacterial and tuberculous morphology

Acute bacterial

Purulent joint aspirate

Acute inflammation and rapid clinical onset

Tuberculous

Confluent caseating granulomas

Pannus, bone erosion and fibrous ankylosis

Viral arthritis spans more than one mechanism

**Direct viral joint infection or infection-triggered
autoimmunity**

The arthritis may be acute or subacute.

Viral causes named by Robbins

Viral group / agent

Alphaviruses

Parvovirus B19

Other agents

Rubella

Epstein–Barr virus

Hepatitis viruses

Hepatitis B virus

Hepatitis C virus

Two explanations for viral joint symptoms

Direct involvement

The virus infects the joint

Examples: Rubella and some alphaviruses

Immune-mediated response

Viral infection triggers an autoimmune reaction

Joint symptoms need not imply direct viral invasion

Lyme arthritis: A spirochetal joint infection

Borrelia burgdorferi transmitted by Ixodes ticks

Lyme disease follows a multiorgan course and may later involve joints.

The geographic context

United States

New England

Mid-Atlantic states

Additional region

Upper Midwest

Range described as expanding

Lyme disease: Multisystem sequence

Initial skin
Infection

Subsequent
Articular episodes

Dissemination to
Other organs



Early-treated versus untreated Lyme disease

With timely treatment

Arthritis now affects fewer than 10% of infected patients

Textbook reports many infections are treated earlier

Without timely treatment

Up to 80% may develop migratory arthritis

Episodes can persist for weeks or months

Lyme arthritis: Characteristic distribution

Most characteristic

Knee

Large joint

Other common sites

Shoulder and elbow

Large joints

Additional site

Ankle

Large joint

Lyme arthritis can recur at new sites

Initial episode
1–2 joints

New joint
Episodes later

Symptoms for
weeks
To months



Diagnostic evidence in Lyme arthritis

- Anti-Borrelia antibodies are the usual basis of diagnosis.
- Borrelia DNA may be detected using PCR on synovial fluid.
- The reported approximate synovial-fluid PCR detection rate is 70% in patients with Lyme arthritis.

Antibiotic therapy changes Lyme outcome

Cure in approximately 90%

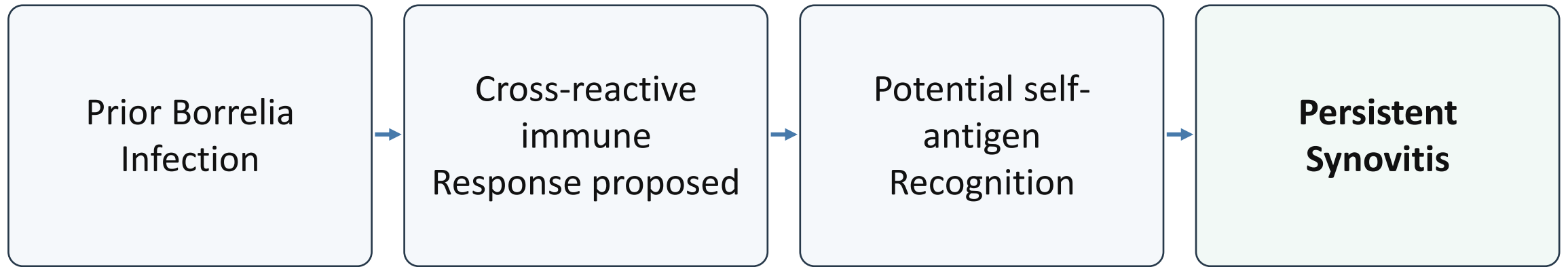
This statement concerns therapy active against *Borrelia*; This pathology lecture does not prescribe regimens.

Persistent arthritis need not imply ongoing detectable infection

Postinfectious / antibiotic-refractory Lyme arthritis

The organism may not be detected in joint fluid, even by PCR, in such cases.

Proposed immune mechanism after Borrelia clearance

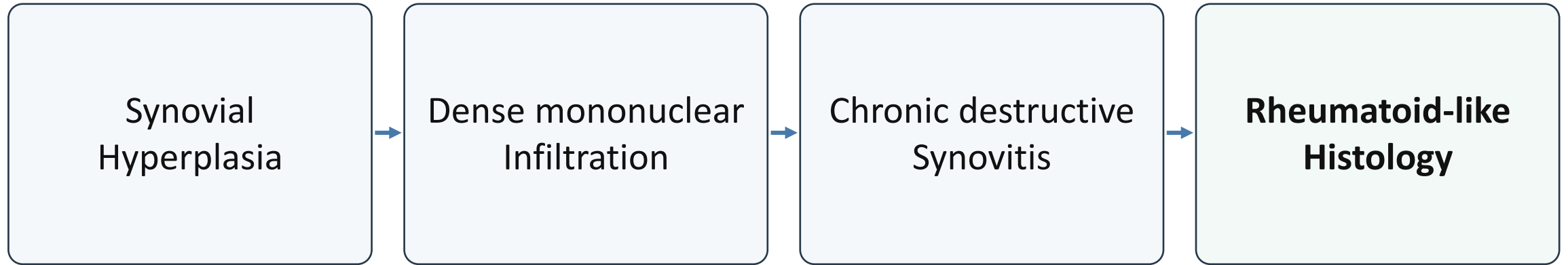


This is a proposed explanatory mechanism, not established in every patient.

Histology of Lyme-associated synovitis

- Synoviocyte hyperplasia and fibrin deposition are described.
- Mononuclear inflammation includes a prominent CD4⁺ T-cell population.
- Arterioles may exhibit concentric “onion-skin” wall thickening (obliterative endarteritis).

Why chronic Lyme synovitis can mimic rheumatoid arthritis



Severe Lyme synovitis can resemble rheumatoid arthritis histologically.

Infectious arthritis: Organizing clinical patterns

Syndrome	Tempo	Distinguishing clue
Acute suppurative	Sudden	Purulent aspirate with identifiable organism
Mycobacterial	Insidious	Caseating granulomas; pannus; ankylosis
Viral	Acute / subacute	Direct viral infection or triggered immunity
Lyme	Episodic / migratory	Large joints; anti-Borrelia antibodies

Infectious versus reactive arthritis

Infectious arthritis

Infectious agent may seed articular tissues

Suppurative, mycobacterial, viral, Lyme are considered here

Reactive arthritis

Arthritis follows genitourinary or gastrointestinal infection

The associated postinfectious syndrome is a separate Robbins topic

Infectious versus crystal-induced arthritis

Infectious

Microorganisms involve the joint

Organism-directed diagnosis is important

Crystal-induced

Deposition of urate, calcium pyrophosphate,
or other crystals

Crystal-driven inflammation can damage
cartilage

Case discussion: Sudden monoarthritis

A patient develops abrupt severe knee pain, swelling and restricted motion, accompanied by fever and a raised inflammatory response.

Which finding would make the suppurative diagnosis etiologically specific?

Case reasoning: Acute suppurative pattern

The critical source-supported diagnostic step

- Aspiration yielding purulent articular fluid strongly supports acute suppuration.
- Identifying the organism in the aspirate establishes the infectious etiology in the Robbins description.
- Rapid recognition and effective antimicrobial treatment can avert destructive outcomes.

Case discussion: Indolent hip disease

An adult with gradually progressive hip pain develops chronic joint damage. Synovial tissue demonstrates confluent caseating granulomas.

Which infectious arthritis pattern explains the combination?

Case reasoning: Mycobacterial arthritis

How morphology and tempo align

- Tuberculosis can reach the joint from a visceral source or adjacent osteomyelitis.
- Caseating granulomas with pannus and marginal erosion are characteristic source findings.
- Long-standing disease may produce fibrous ankylosis and joint-space obliteration.

Case discussion: Episodic large-joint synovitis

A patient from a region associated with Lyme disease develops recurrent episodes involving one or two large joints. Synovium shows chronic inflammation.

Which source-based evidence supports a *Borrelia*-associated explanation?

Case reasoning: Lyme arthritis

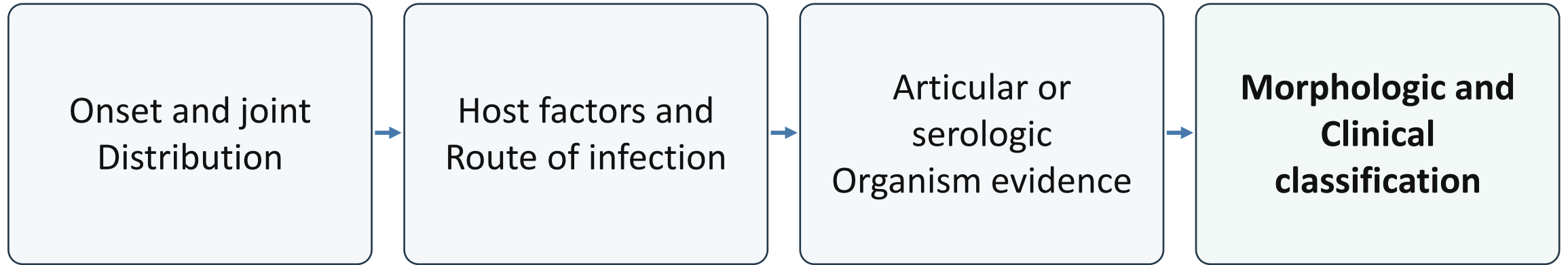
Combine history with joint-specific tests

- Anti-Borrelia antibody testing is the usual diagnostic basis.
- Synovial-fluid PCR can detect Borrelia DNA, but a negative result does not by itself resolve persistent post-treatment disease.
- Chronic synovitis can resemble rheumatoid arthritis; Morphology alone may be insufficient.

Morphologic pattern and likely infectious category

Morphologic or clinical finding	Category to consider
Purulent aspirate; abrupt swollen monoarthritis	Acute suppurative arthritis
Caseating granulomas and synovial pannus	Mycobacterial arthritis
Acute or subacute viral-associated symptoms	Viral arthritis
Chronic CD4 ⁺ -rich synovitis with obliterative endarteritis	Lyme arthritis (compatible, not exclusive)

Clinicopathologic synthesis



Teaching synthesis; No single universal test or cutoff is implied.

Essential conclusions

- All three entry routes may seed joints; Cartilage destruction can leave permanent disability.
- Acute purulent arthritis requires prompt recognition and etiologic identification.
- Tuberculosis, viral infection, and Lyme disease produce distinctive chronicity, mechanisms, and diagnostic clues.

Primary reference and lecture scope

Robbins, Cotran & Kumar: Pathologic Basis of Disease

11th edition (2026) · Chapter 26: Bones, Joints, and Soft Tissue Tumors

Infectious Arthritis; Suppurative Arthritis; Mycobacterial Arthritis; Viral Arthritis; Lyme Arthritis

Comparative context: Reactive Arthritis and Crystal-Induced Arthritis