Diagnostic Imaging

Musculoskeletal Non-Traumatic Disease

SECOND EDITION

Manaster

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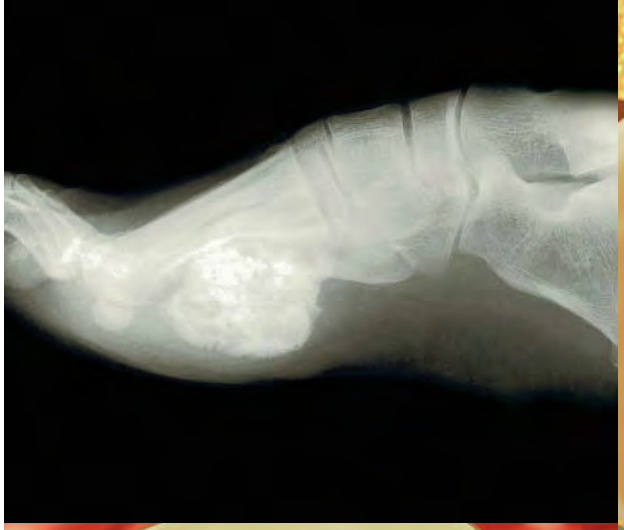

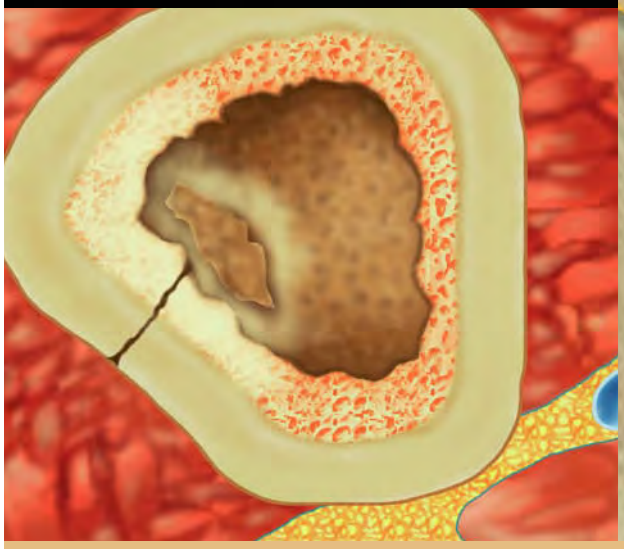

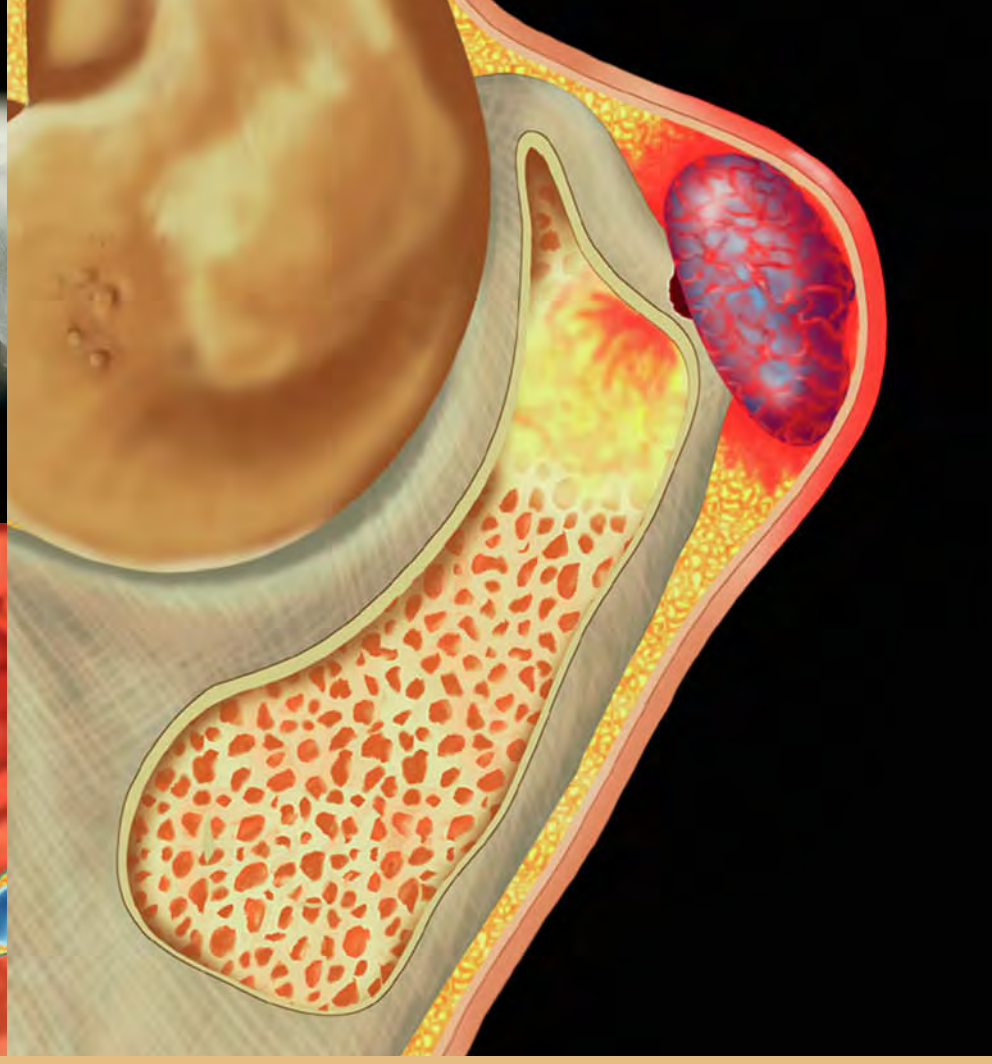

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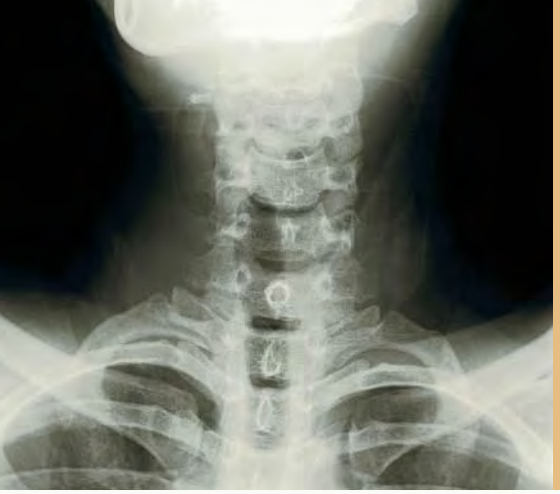

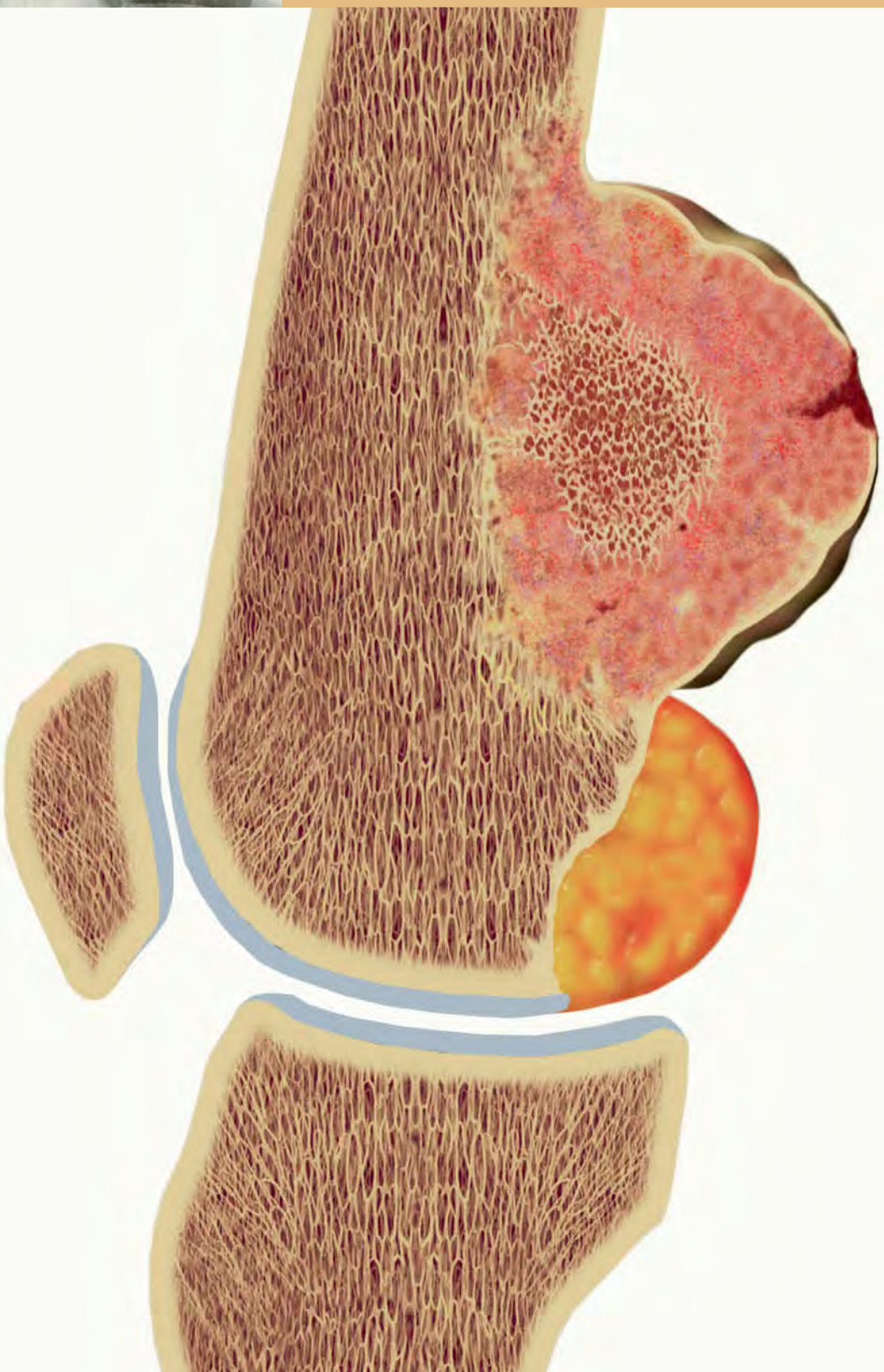

Diagnostic Imaging

Musculoskeletal Non-Traumatic Disease

SECOND EDITION

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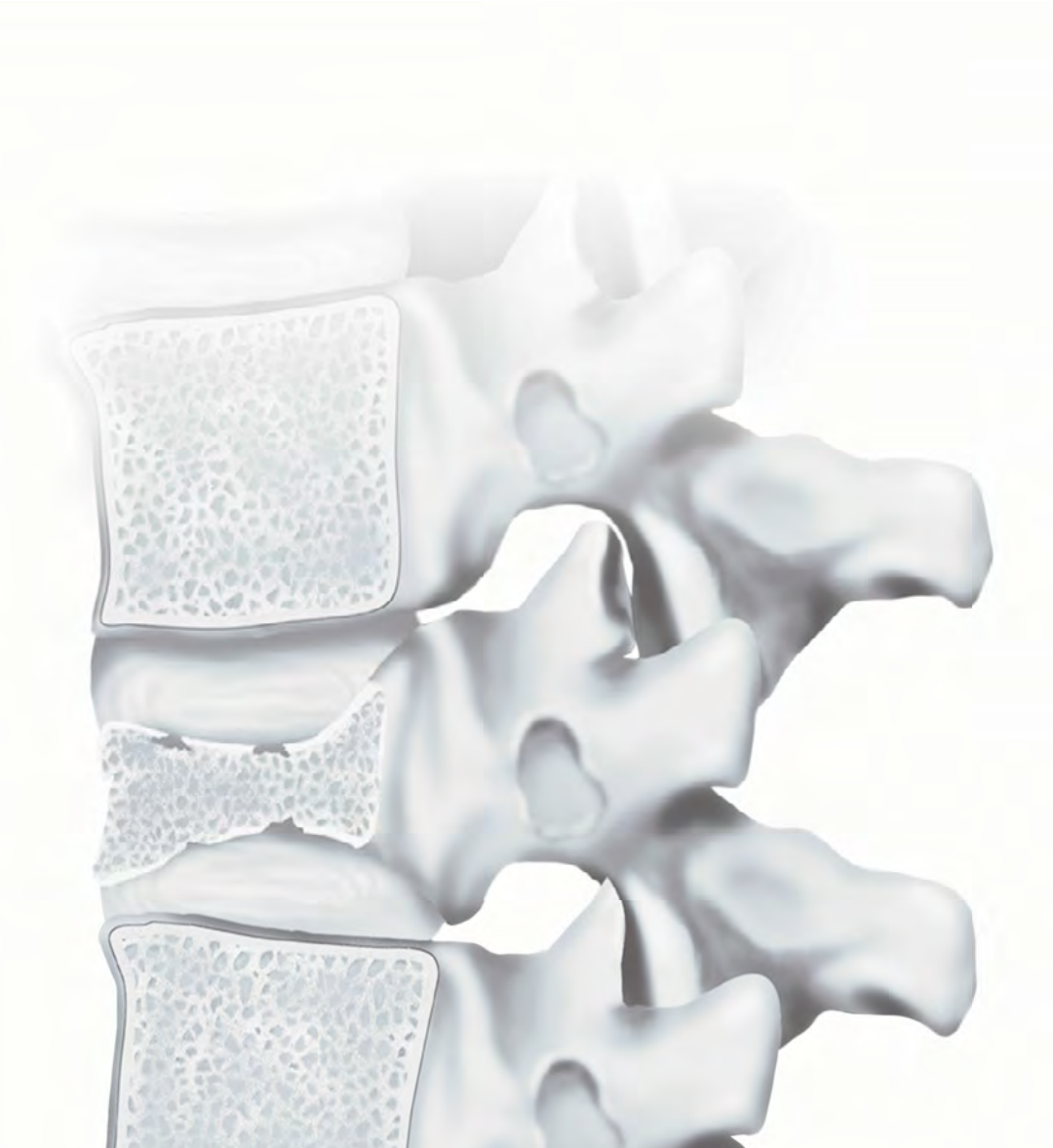
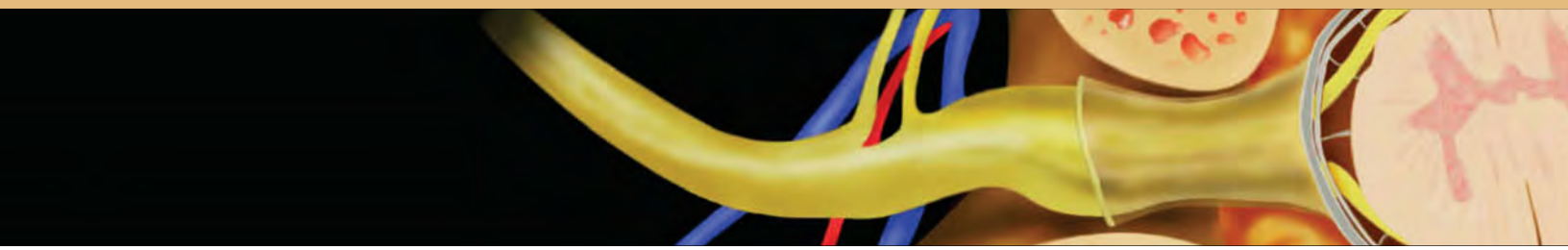
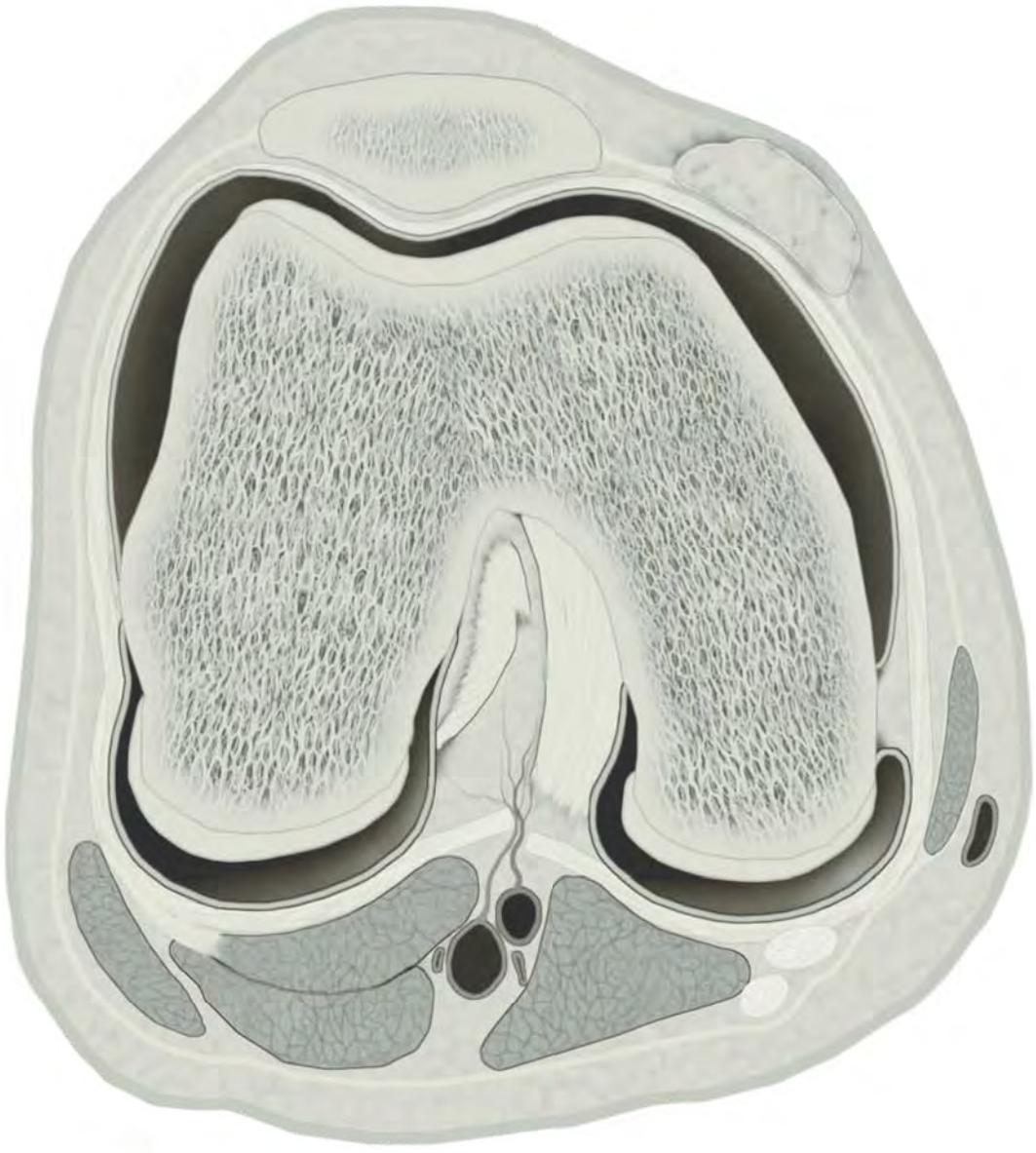
Dedication

This book is dedicated to all my family members, colleagues, and students. I have learned much from each of you and treasure the association.

BJM







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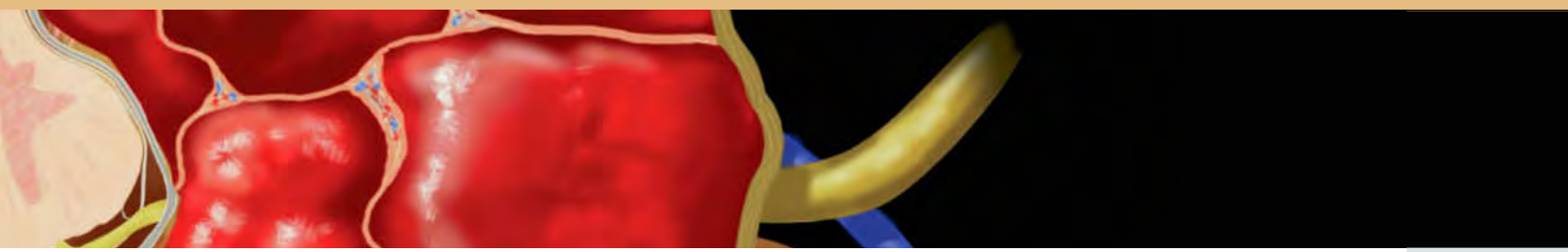

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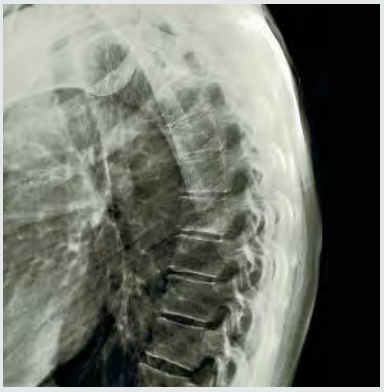

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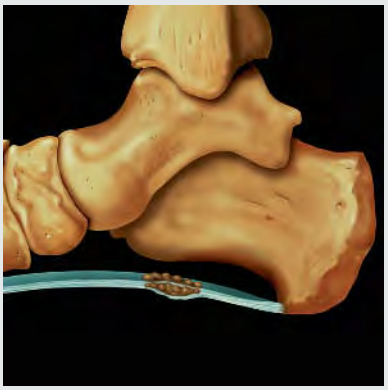

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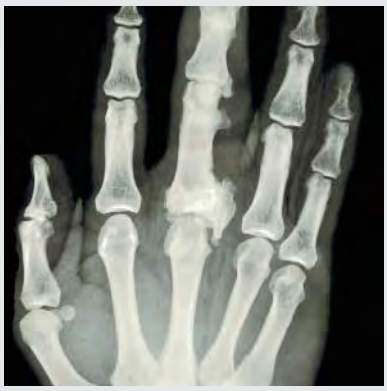

Preface

We are delighted to present the second edition of *Diagnostic Imaging: Musculoskeletal: Non-Traumatic Disease*. Along with its companion book, *Diagnostic Imaging: Musculoskeletal: Trauma*, we offer complete coverage of the extensive topic of musculoskeletal imaging in the standard *Diagnostic Imaging* text format. More than 1,000 pages and thousands of images provide a detailed understanding of arthritis, osseous tumors, soft tissue tumors, metabolic bone disease, infectious processes, systemic diseases contributing to osseous abnormalities, drug and nutritional abnormalities contributing to bone disease, and congenital/developmental musculoskeletal abnormalities. One might expect to find the topics of orthopedic implants and "hardware" in either the traumatic or non-traumatic disease books; we have chosen to include these topics in the present book.

As with the initial edition, we have kept the authoring team small. This maintains the advantage of consistent quality throughout the book and ensures that there is no duplication of topics or information, while being certain of complete coverage of topics.

We have maintained the signature *Diagnostic Imaging* format of bulleted text and Key Facts boxes. Prose introductions to major sections offer the author's approach to difficult topics and are worth an initial perusal. Tabular offerings as well as graphics enhance understanding of some topics and may serve as quick references.

The clinical images have been updated for this edition and, as with the initial edition, are seen in far larger numbers than can be accommodated in most textbooks. Since many, if not most, musculoskeletal diseases rarely have a single imaging presentation, we have included the variations in appearance

and have not wasted space showing similar-appearing cases. We have also enlarged the ebook gallery for many of the topics, showing additional cases that will help the reader appreciate the variability of these disease processes.

Text and references have been updated for this edition. Some new topics have been added, particularly in the section of Drug-Induced and Nutritional MSK Conditions. The authors hope and expect that you will find our offering valuable in your practice.

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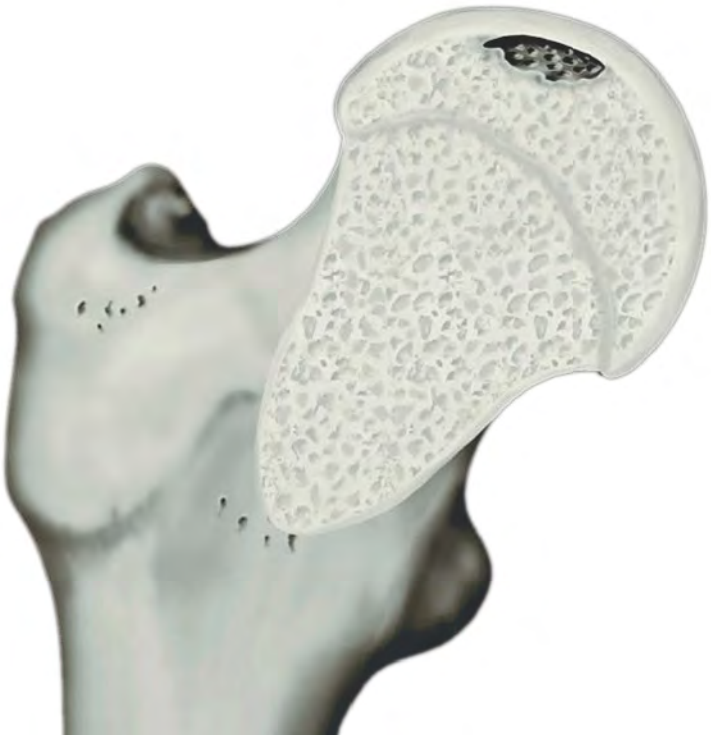

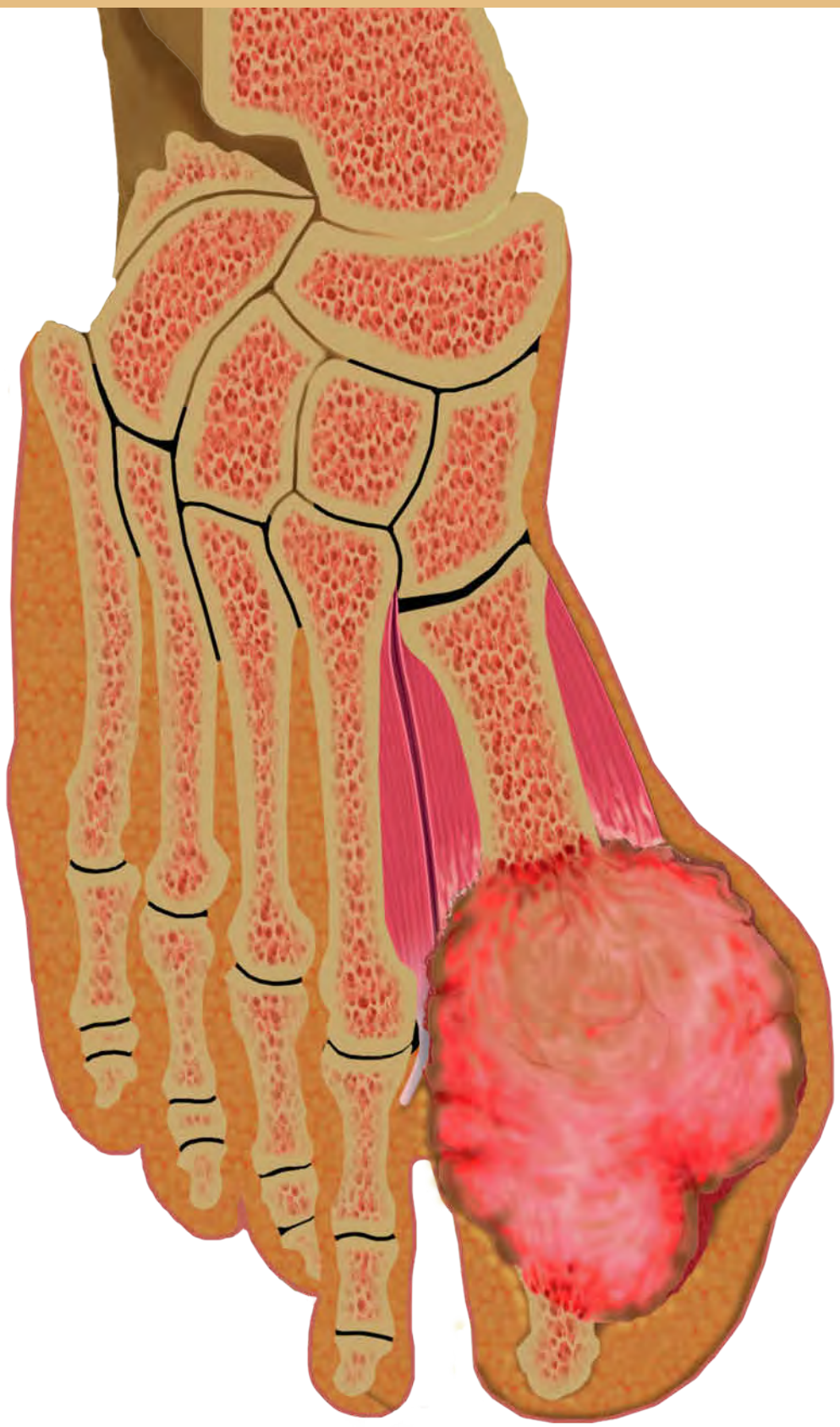

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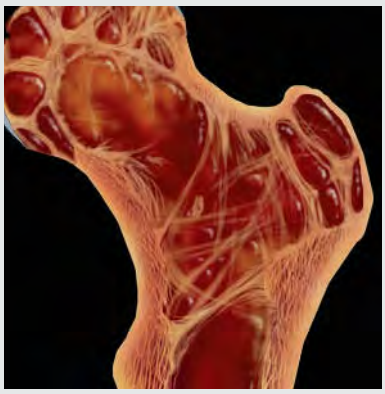

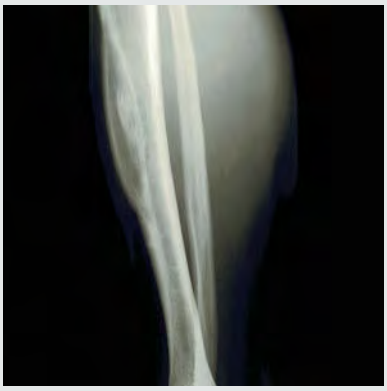

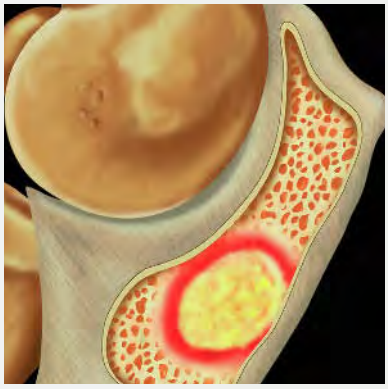

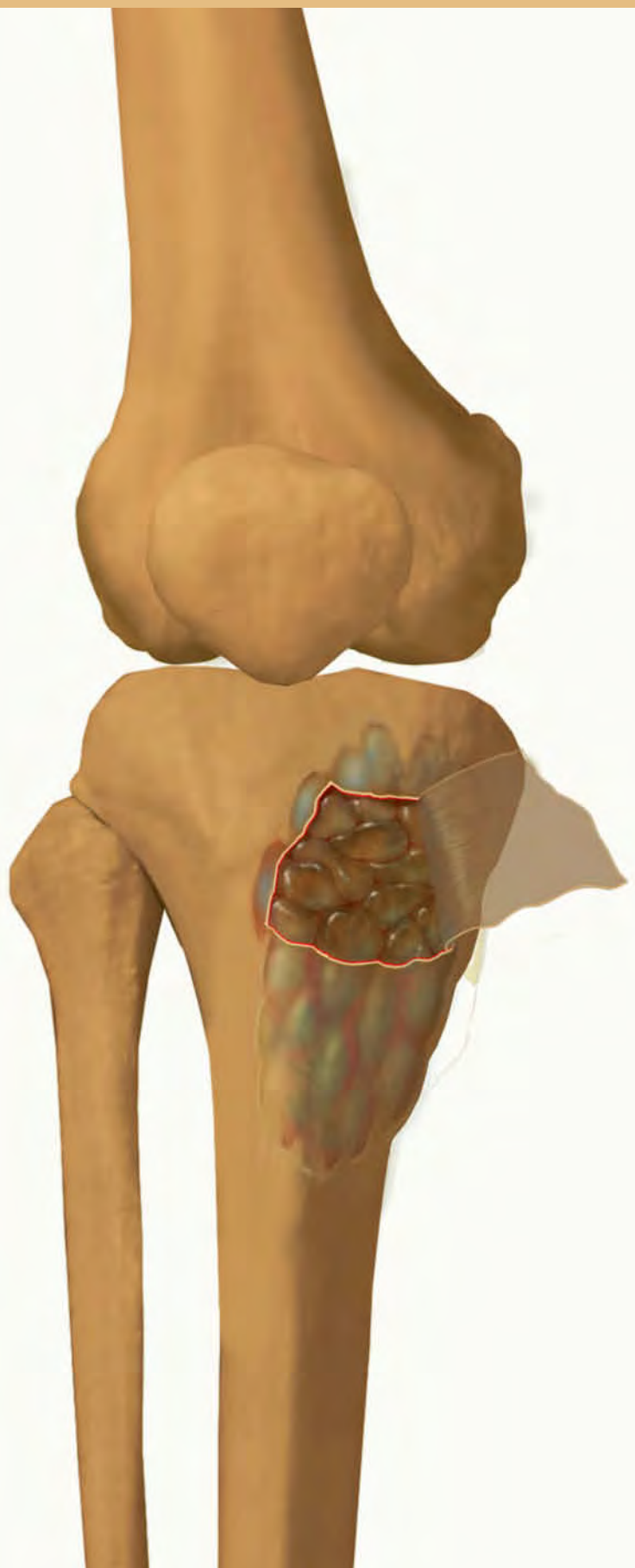

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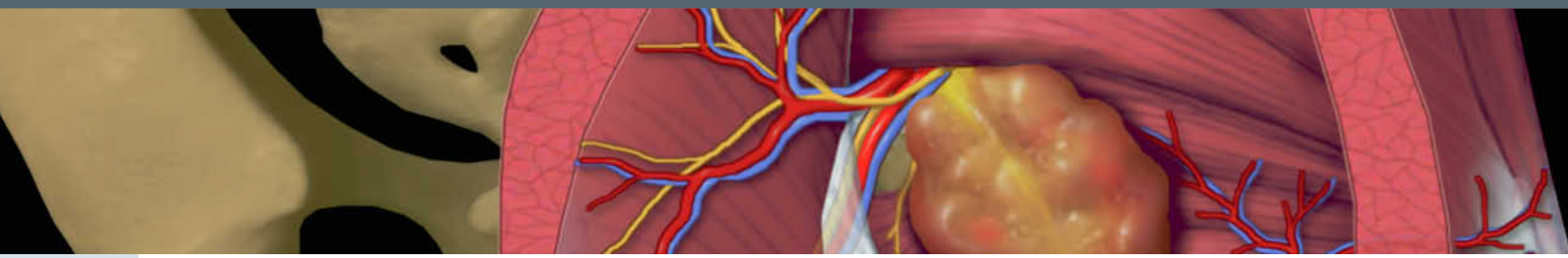

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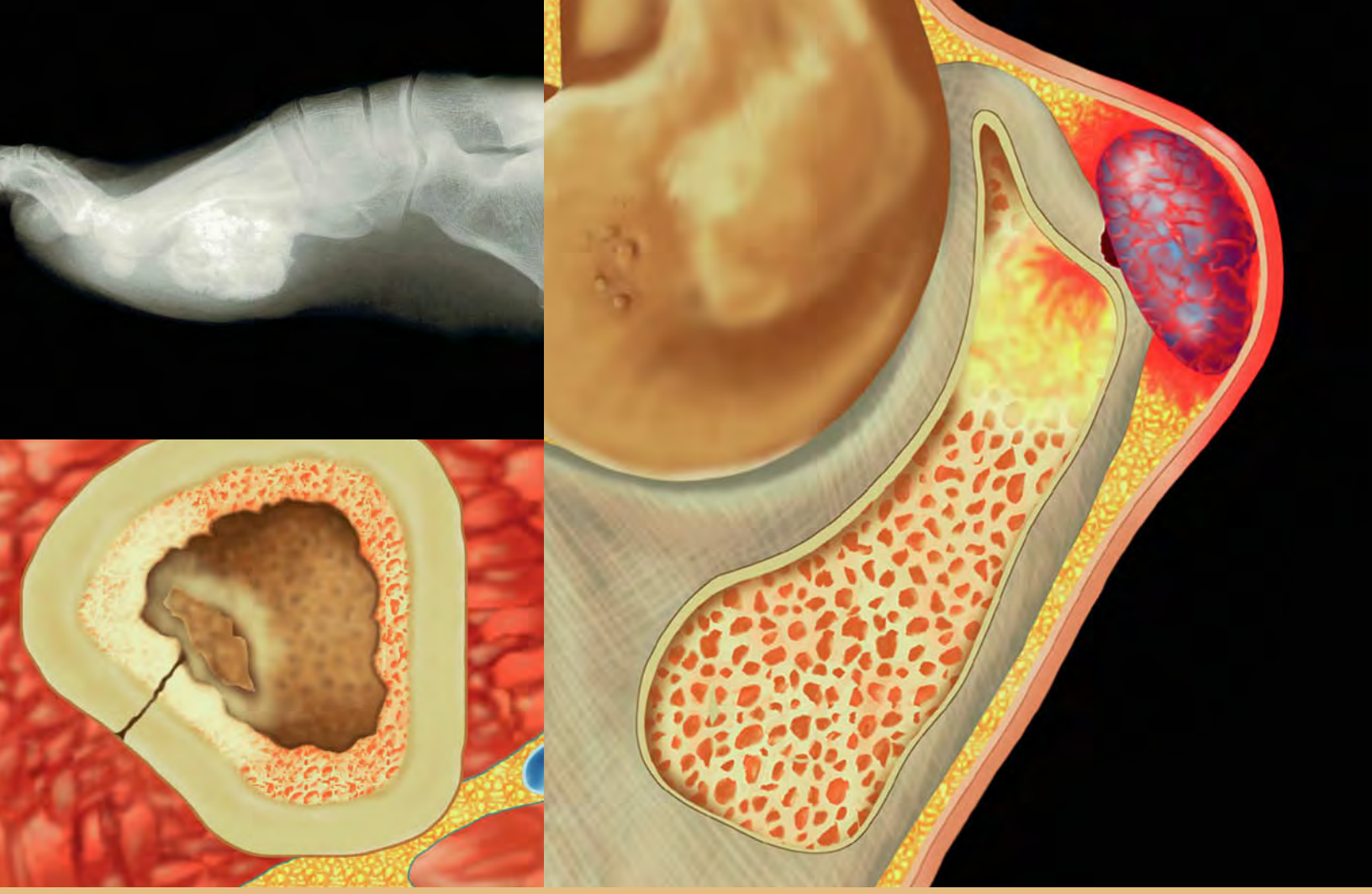

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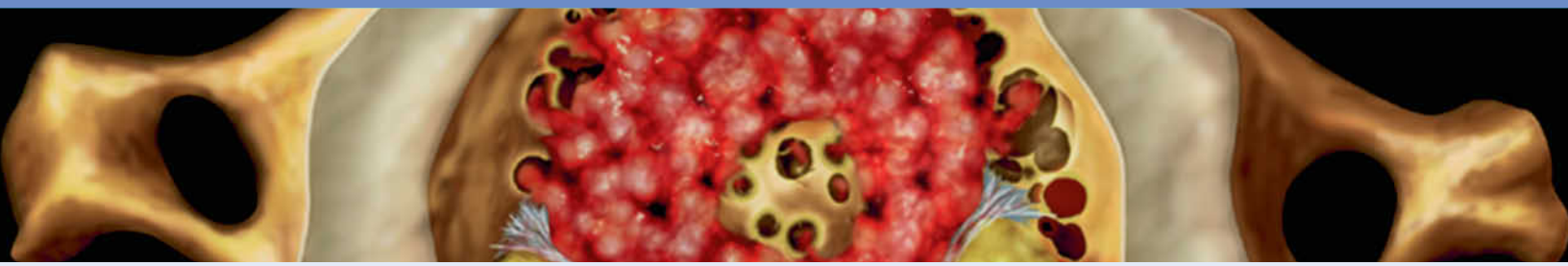
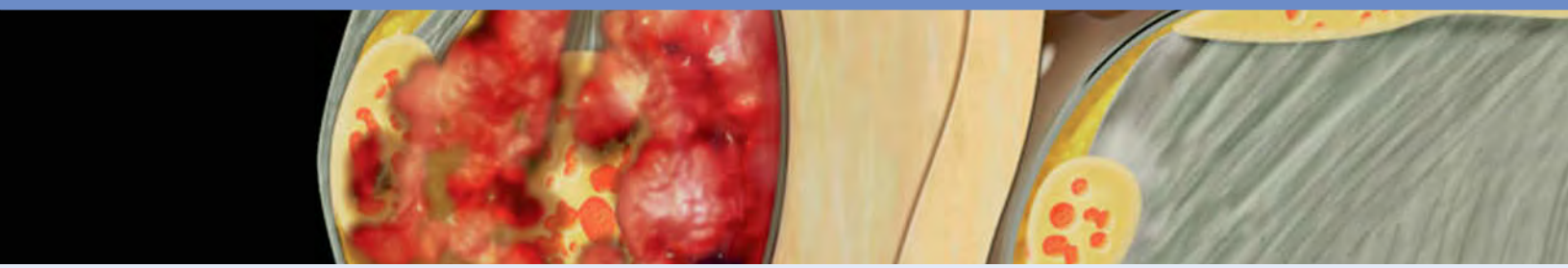
SECTION 1

Arthritis





Due to Biochemical Disorders or Depositional Disease

Miscellaneous Joint Disorders

Classic Appearance of Arthritic Processes

When an arthritic process is well established in a particular patient, it will usually achieve a typical appearance, which allows diagnosis by means of imaging. At such a moderately early or mid stage of disease, radiographs are usually sufficient to make the correct diagnosis. The diagnosis usually depends on the location of the joint abnormalities and a host of other radiographic characteristics.

Location of involved joints can often eliminate some diagnoses and raise the probability of others. For example, distal interphalangeal joint disease is commonly seen in psoriatic arthritis, osteoarthritis, and erosive osteoarthritis. However, it is not seen in rheumatoid arthritis until extremely late in the disease; thus RA should not be considered in an early arthritis. Similarly, a disease involving the sacroiliac joints would raise the possibility of ankylosing spondylitis, inflammatory bowel disease arthritis, psoriatic spondyloarthropathy, chronic reactive arthritis, osteoarthritis, and DISH. Common locations of joint involvement are illustrated in diagrammatic fashion in this section. Note the joints that are involved earliest and most commonly are distinguished from those involved less frequently or in end-stage disease.

While the location of the joints involved certainly contributes to establishing a list of reasonable diagnoses, the lists can be relatively long, as in the examples above. There are several other parameters that are useful in honing that list to a single diagnosis that are outlined in the tables that follow. Further explanation regarding some of these parameters may be helpful, as follows.

Age and gender may be the easiest parameters to apply. There are a minimal number of arthritic processes that affect children (juvenile inflammatory arthritis, hemophilic arthropathy, inflammatory bowel disease arthropathy, and septic joint) and teenagers (in addition to those affecting children, early onset adult rheumatoid arthritis and ankylosing spondylitis). Some diseases are gender specific (hemophilic arthropathy and hemochromatosis), while others are found in one gender far more frequently (gout, ankylosing spondylitis, chronic reactive arthritis in males, and rheumatoid arthritis in females).

One of the most important parameters is the **character of the process**. Some arthritides are **purely erosive**; rheumatoid arthritis is the hallmark for this group. Others are **purely bone-forming** (also termed "productive"). This bone formation may appear in the form of osteophytes (as in osteoarthritis), enthesopathy or ligamentous ossification (as in ankylosing spondylitis, DISH, and OPLL), or periositis (as in psoriatic arthritis, chronic reactive arthritis, and juvenile idiopathic arthritis). Other processes may be **mixed**, sometimes starting with erosions but progressing to osteophytes (as in pyrophosphate arthropathy or gout) or starting with periostitis and progressing to mixed erosions and osteophytes (as in psoriatic arthritis or chronic reactive arthritis). These processes tend to be distinctive for each type of arthritis by the time they are well established; between evaluating the character of the process and its primary location in an individual, the diagnosis can usually be secured.

Bilateral symmetry of an arthritic process can be a useful characteristic. Rheumatoid arthritis is especially well known for appearing bilaterally symmetric. Note that rheumatologists do not require specific joints of specific digits to qualify the arthritis as symmetric. For example, 5th PIP left hand and 3rd PIP right hand would be considered symmetric disease simply because of PIP involvement of each hand. Note also that bilateral symmetry may not be present in early stages of arthritic disease, even in rheumatoid arthritis. Similarly, while we usually think of the sacroiliitis of ankylosing spondylitis as being bilaterally symmetric, in its early stages the symmetry is often strikingly absent. Therefore, useful generalizations regarding bilateral symmetry are most often made in the mature stages of the disease process. However, rigid application of "rules" of symmetry should be avoided when evaluating early arthritis.

Soft tissue swelling can be the key to finding the earliest changes of arthritis on a radiograph. The sausage digit may lead to the discovery of subtle periostitis, even in the absence of joint space narrowing or erosions. Swelling around a metacarpophalangeal joint may lead to closer examination of a metacarpal head showing cortical indistinctness or the dot-dash pattern of early inflammatory disease. Be sure to window every image to evaluate the soft tissues, as these abnormalities can lead to closer examination of adjacent joints.

Soft tissue masses are not frequently seen in conjunction with arthritic processes. However, they may lead to specificity in diagnosis. Gouty tophi, seen as a mass containing a variable degree of dense tissue, can be diagnostic. As another example, soft tissue nodules, combined with acroosteolysis and interphalangeal joint erosions, leads to the rare diagnosis of multicentric reticulohistiocytosis.

In differentiating between the ligamentous ossification of DISH/OPLL, osteophytes of spondylosis deformans, syndesmophytes of ankylosing spondylitis, and paravertebral ossification of psoriatic arthritis and chronic reactive arthritis, the **character of paravertebral ossification** can often suggest the correct diagnosis. However, as with other parameters, it is important to note that mature paravertebral ossification in each of these entities may all have a similar appearance. True osteophytes may bridge across the disc space and give the appearance of the flowing ligamentous ossification of DISH. Mature ankylosing spondylitis has much bulkier syndesmophytes than the thin vertical ones depicted in early disease.

Subchondral cysts are seen in virtually all arthritic processes and therefore are rarely useful in differentiating among them. However, occasionally the subchondral cysts are so large that this characteristic becomes useful in diagnosis. Particularly large subchondral cysts in a setting that otherwise resembles rheumatoid arthritis lead to the diagnosis of robust rheumatoid arthritis. Very large cysts are also noted in pyrophosphate arthropathy and pigmented villonodular synovitis. Osteoarthritis and gout may also produce very large subchondral cysts.

Bone density must always be interpreted within the context of patient age and gender. An elderly female will usually have diffuse osteoporosis, with or without superimposed rheumatoid arthritis (classically described as causing juxtaarticular, followed by diffuse osteoporosis). Thus, though we state that normal bone density is a characteristic of osteoarthritis and gout, in an older patient those arthritic processes may be seen in the presence of diffuse osteoporosis. Another example that may cause confusion is the young adult with end-stage renal disease and a renal

transplant. Erosive disease in these patients is likely to be gout or amyloid. However, the bone density will be decreased due to both their renal osteodystrophy and likely use of steroids for their transplant. In this case, gout should be suggested to explain erosive disease, despite the bone appearing osteoporotic. Focal osteoporosis can also be helpful in identifying joints with active inflammation, as the hyperemia from the inflammatory process leaches the calcium from the bone.

The **pattern and timing of cartilage destruction** may be another useful parameter. Some arthritides, such as gout, classically cause prominent erosions before significant cartilage destruction, while most inflammatory arthritides, such as rheumatoid arthritis, result in early marginal erosions but also relatively early cartilage destruction. The pattern of cartilage destruction also distinguishes the inflammatory arthropathies, where it is uniform throughout the joint as opposed to the more focal cartilage destruction seen in the weight-bearing portions of the joint in osteoarthritis.

Adjacent calcific or ossific densities may be particularly helpful in diagnosis. Chondrocalcinosis is not unique to pyrophosphate arthropathy but is most frequently seen in that disease. The presence of chondrocalcinosis should also raise the question of traumatic osteoarthritis and hemochromatosis. Calcifications in gouty tophi are usually unique in their appearance. Calcific or ossific bodies in synovial chondromatosis are different from the osseous debris seen with a Charcot joint. Therefore, the character of adjacent calcific or osseous densities may be useful in the diagnostic process.

Ankylosis of the peripheral joints is most commonly seen in psoriatic arthritis and juvenile idiopathic arthritis. It is commonly found in the spine of patients suffering from spondyloarthropathies (most frequently ankylosing spondylitis), DISH, and juvenile idiopathic arthritis. Other more rare arthritic processes may show ankylosis as well. On the other hand, ankylosis in cases of rheumatoid arthritis is exceedingly rare. Do not be fooled by a surgical arthrodesis in a patient with severe rheumatoid arthritis. Arthrodesis is often attempted to stabilize the digits in this disease, and may mimic ankylosis.

Early Appearance of Arthritic Processes

We are now diagnosing arthritic processes at an earlier stage, prior to any radiographic change. This ability is essential, since early application of disease-modifying drug therapy may halt joint destruction. The benefit of early diagnosis is obvious, yielding longer patient productivity and decreasing the need for arthroplasty. However, the diagnosis may be difficult with subtle or absent radiographic findings and relies on MR or ultrasound. Early tenosynovitis and joint effusions may be identified on ultrasound, and MR may demonstrate tenosynovitis, effusion, and bone marrow edema long before actual erosions are seen in rheumatoid arthritis. Inflammatory change at vertebral body corners may be identified on MR, indicating early spondyloarthropathy. Even more subtle may be the enthesitis and adjacent marrow edema found in early ankylosing spondylitis, which are often found at the "corners" of the image (interspinous ligaments, iliac spine, greater trochanter) and are easily overlooked. Close attention should be paid to these locations, even when evaluating a "routine lower back pain" spine MR exam.

Late Appearance of Arthritic Processes

End-stage arthritic processes may have a classic appearance. Classic changes are often seen in the deformities and erosive change in rheumatoid patients or in the postural changes with vertebral column fusion in ankylosing spondylitis patients. However, at times an arthritic process, particularly when ineffectively treated, may attain a potentially confusing nonstandard appearance. An example of this is the rheumatoid patient who has failed drug therapy, resulting in an arthritis mutilans appearance of the hands (remember that pencil-in-cup and arthritis mutilans are not exclusively seen in psoriatic arthritis). Another example is the Native American ankylosing spondylitis patient who is treated without the use of Western medications and may present not only with the spondyloarthropathy expected in ankylosing spondylitis, but also with erosive disease involving all the peripheral joints, including hands and feet. Finally, the classic disease process that may be confusing is end-stage gout, which, if misdiagnosed or undertreated, may result in spectacular erosive disease at unexpected locations. It is important to remember that gout can look like anything and can be located at any joint!

Coexistence of Arthritic Processes

It is not unusual for two of the more common arthritic processes to coexist, particularly in the elderly patient. This may be confusing initially but can be worked out through understanding the prevalence of the diseases in the patient population, as well as by paying attention to the appearance and location of the abnormalities present. The most common combination is a new onset of rheumatoid arthritis superimposed on osteoarthritis. In this case, the osteoarthritis is usually well-established, involving the 1st carpometacarpal and interphalangeal joints in classic fashion, but there is new inflammatory change seen in the metacarpophalangeal joints. The elderly patient may also develop pyrophosphate arthropathy, superimposed over osteoarthritis or rheumatoid arthritis. The patient with a diabetic Charcot joint may develop superimposed septic arthritis. Keeping these possibilities in mind is useful to the interpreter, as the pattern of disease may not be classic.

Conclusion

There are many subtleties involved at specific joint locations in specific diseases, which cannot be discussed in such a broad introduction. These will be covered in proper detail in the individual sections that follow.

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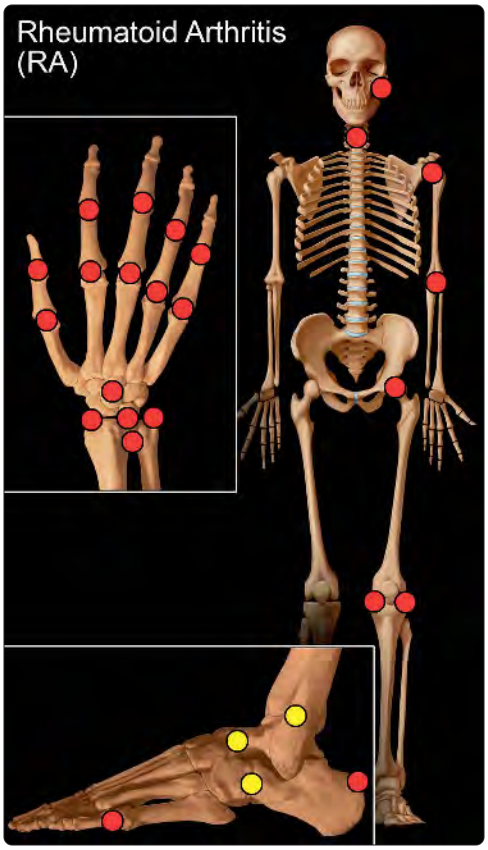
Characteristics of Arthritic Processes

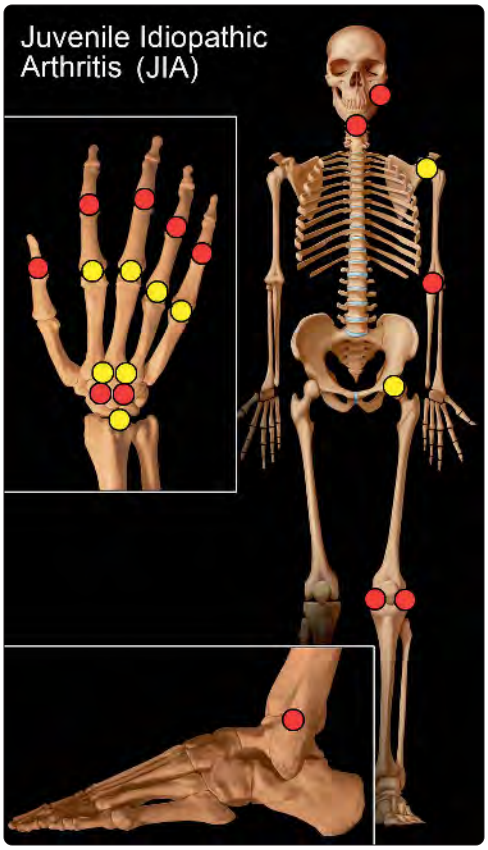
Arthritis Type	Gender	# of Joints	Symmetry	Bone Density	Character	Cartilage Destruction
RA	M < F (1:3)	Polyarticular	Yes, by end stage	Density ↓	Erosive	Early, diffuse
Robust RA	M > F	Polyarticular	Yes, by end stage	End stage ↓	Erosive	Early, diffuse
JIA	M < F (1:4-5)	Pauci- or polyarticular	Generally no	End stage ↓	Erosive	Early, diffuse
Hemophilia	M only	Pauciarticular	No	Normal	Erosive	Early, diffuse
Adult Still disease	M = F	Polyarticular	Generally no	End stage ↓	Erosive	Early, diffuse
MCRH	M < F	Polyarticular	Yes	Related to age	Erosive	Early, diffuse
Osteoarthritis	M < F	Polyarticular	Often	Normal	Produces bone	Early, focal
DISH/OPLL	M > F (2:1)	Nonarticular	Nonarticular	Normal	Produces bone	None
AS/IBD arthritis	AS: M > F (2.5-5:1); IBD: M = F	Polyarticular	Yes, by end stage	Mid to end stage ↓	Mixed	Mid stage, diffuse
PSA/CRA/HIV	PSA: M = F; CRA/HIV: M > F (5-6:1)	Polyarticular	Generally no	Normal	Mixed	Mid stage, diffuse
Gout	M > F (9:1)	Polyarticular	No	Normal	Mixed	Late disease
Pyrophosphate	M < F (1:2-7)	Polyarticular	Generally no	Related to age	Mixed	Mid stage, diffuse
Hemochromatosis	M only	Polyarticular	Generally no	Normal	Produces bone	Late disease
Amyloid	M > F	Pauciarticular	No	Density ↓	Erosive	Early, focal
PVNS	M < F (1:2)	Monoarticular	No (single joint)	Normal	Erosive	Late, focal
PSC	M > F	Monoarticular	No (single joint)	Normal	Erosive	Late, focal
Charcot	M = F, relates to etiology	Mono- or pauciarticular	No	Not unless diabetic	Destructive	Early, diffuse
Septic arthritis	M = F	Monoarticular	No (single joint)	Normal	Erosive	Early, diffuse

Characteristics of Arthritic Processes (Continued)

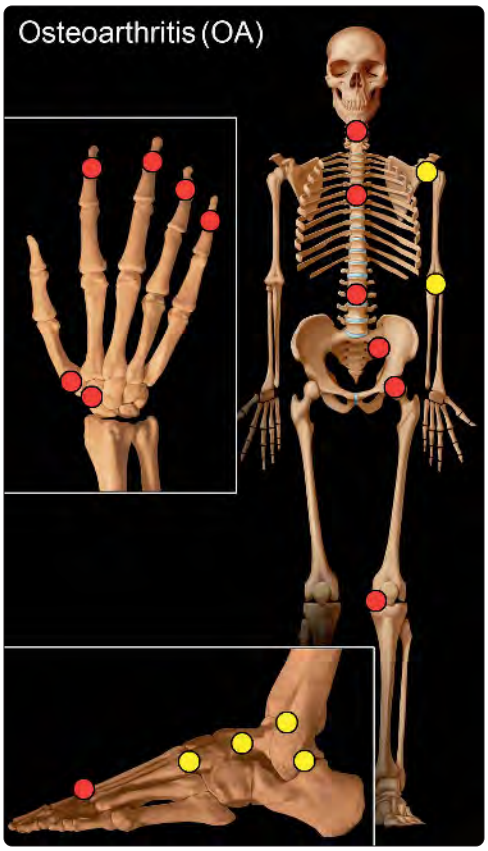
Arthritis Type	Subchondral Cysts	Enthesopathy	Periostitis	Adjacent Density	Ankylosis	Soft Tissue Masses
RA	Yes	No	No	No	No	Rheumatoid nodules
Robust RA	Yes, large	No	No	No	No	Rheumatoid nodules
JIA	Yes	No	Yes, early	No	Yes	No
Hemophilia	Yes	No	No	No	No	No
Adult Still disease	Yes	No	No	No	Yes	No
MCRH	Yes	No	No	No	No	Nodules
Osteoarthritis	Yes	Yes	No	Rare chondrocalcinosis	No	Heberden nodes
DISH/OPLL	No	Yes, prominent	No	No	Yes	No
AS/IBD arthritis	Yes	Yes, prominent	No	No	Yes	No
PSA/CRA/HIV	Yes	Yes	Yes, prominent	No	Yes	No
Gout	Yes	No	No	Yes, tophus	No	Tophi
Pyrophosphate	Yes, large	No	No	Chondrocalcinosis	No	No
Hemochromatosis	Yes	No	No	Chondrocalcinosis	No	No
Amyloid	Yes, large	No	No	No	No	Amyloid nodules
PVNS	Yes, large	No	No	No	No	No
PSC	Yes	No	No	Calcified bodies	no	Rare extraarticular
Charcot	Yes	No	Occasionally	Osseous debris	No	Large fluid collections
Septic arthritis	If chronic	No	Yes	No	Rare	No

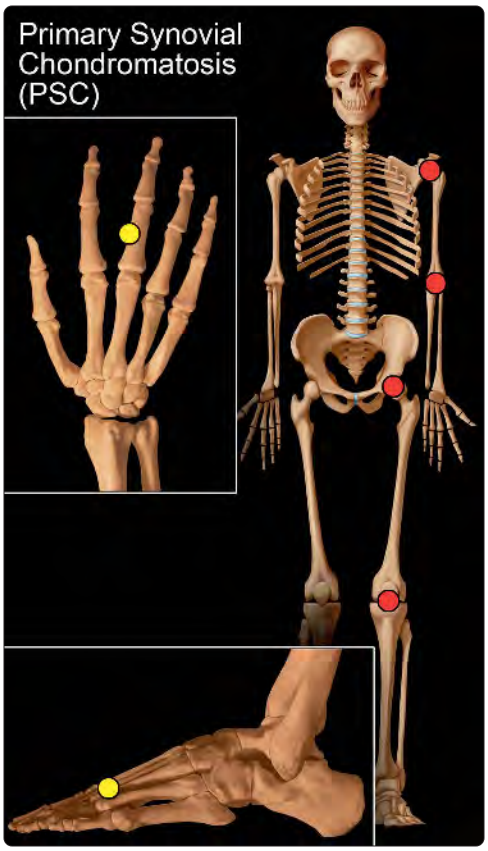
Rheumatoid arthritis = RA, juvenile idiopathic arthritis = JIA, multicentric reticulohistiocytosis = MCRH, diffuse idiopathic skeletal hyperostosis = DISH, ossification posterior longitudinal ligament = OPLL, ankylosing spondylitis = AS, inflammatory bowel disease = IBD, psoriatic arthritis = PSA, chronic reactive arthritis = CRA, pigmented villonodular synovitis = PVNS, and primary synovial chondromatosis = PSC.





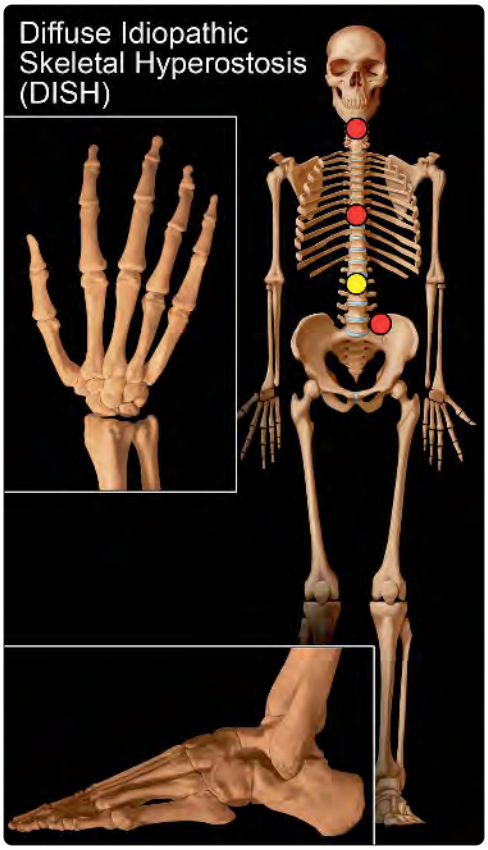
(Left) *RA distribution (common: Red; less common: Yellow) is shown. The hand is the hallmark of the disease, with distal radioulnar joint, radiocarpal joint, intercarpal joint, MCP & PIP joint involvement. Retrocalcaneal and 5th MTP involvement is most common in the foot, with other ankle/hindfoot joints involved less commonly. Hip, knee, shoulder, elbow, temporomandibular, and cervical involvement are common as well.* **(Right)** *JIA distribution is shown. The knee, ankle, and elbow are most frequently involved. In the hand, pericapitate and proximal interphalangeal joints are most frequent, followed by radiocarpal, carpometacarpal, and metacarpophalangeal joints. Cervical spine and temporomandibular involvement are common, while the shoulder and hip are less so.*

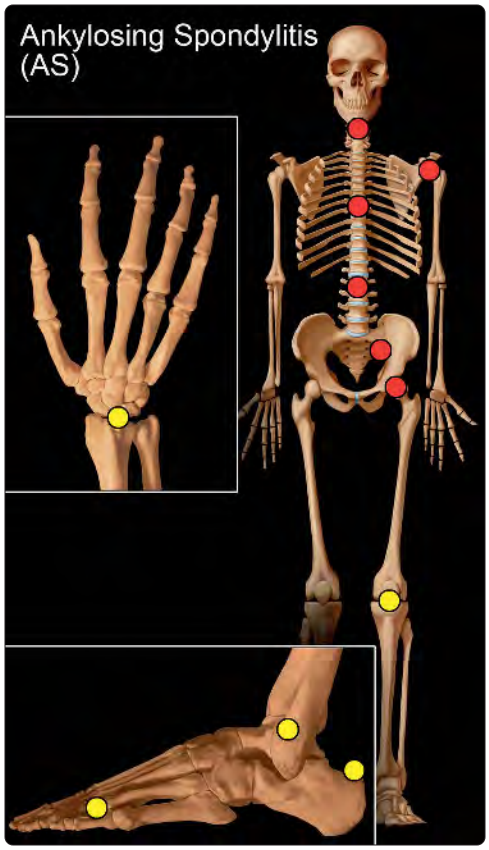




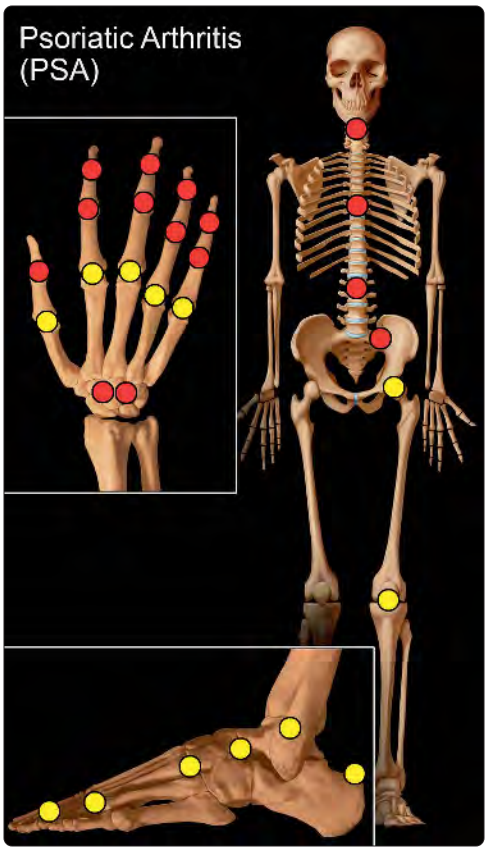
(Left) *OA distribution (common: Red; less common: Yellow) is shown. Hands show common involvement of the 1st carpometacarpal and scapho-trapezio-trapezoid joints, as well as the distal interphalangeal joints. Foot involvement is most frequent at the 1st metatarsophalangeal joint, with the ankle, subtalar, talonavicular, and tarsometatarsal joints less frequently involved. Hip and knee OA are common, while shoulder and elbow are less common. All elements of the spine are commonly affected.* **(Right)** *PSC distribution is shown. Knee involvement is most common, followed by elbow, shoulder, and hip. Axial involvement is virtually never seen. The rare process tenosynovial chondromatosis affects the hands and feet.*

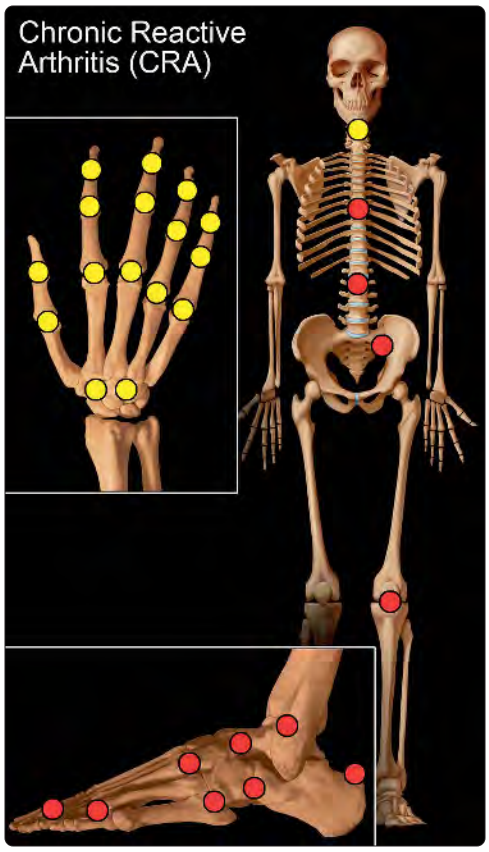
(Left) *DISH and ossification of posterior longitudinal ligament (OPLL) distribution (common: Red; less common: Yellow) are shown. These processes are shown together since they have considerable overlap in distribution, with OPLL predominating in the cervical spine and DISH predominating in the thoracic spine. The nonsynovial portions of the sacroiliac joints (upper 1/2 to 2/3) are affected in DISH.* **(Right)** *AS & inflammatory bowel disease spondyloarthropathy (IBD) distribution are shown. These are shown together; their distribution is identical. All elements of the spine may be involved, along with the sacroiliac joints and large proximal joints (hips, shoulders, and less commonly, knees). With advanced disease, the wrist and ankles may be affected.*

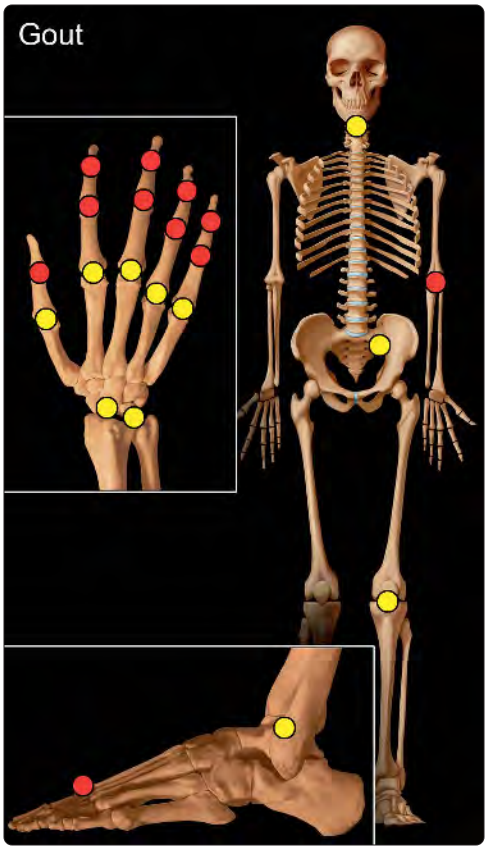


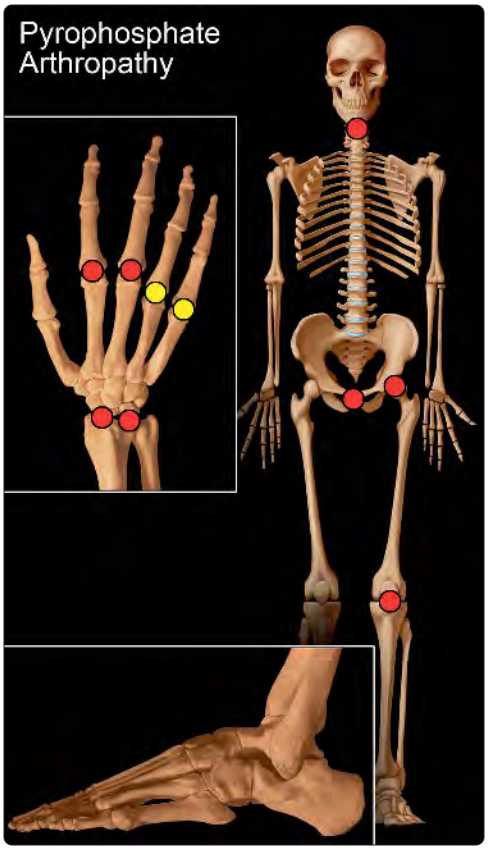


(Left) *PSA distribution (common: Red; less common: Yellow) is shown. The spondyloarthropathy involves all the elements of the spine as well as the sacroiliac joints. The hands show the most frequent peripheral joint involvement, especially in the pericapitate and IP joints. Less frequently, the lower extremities may be involved (foot, ankle, knee, hip).* **(Right)** *CRA distribution is shown. The spondyloarthropathy involves all the elements of the spine as well as the sacroiliac joints. This axial distribution is identical to that of psoriatic arthritis. The feet show the most frequent peripheral joint involvement, with the retrocalcaneal, hindfoot, midfoot, and forefoot all at risk. Knee involvement is also seen. Hand and wrist involvement is considerably less frequent, seen either in advanced disease or sporadically.*

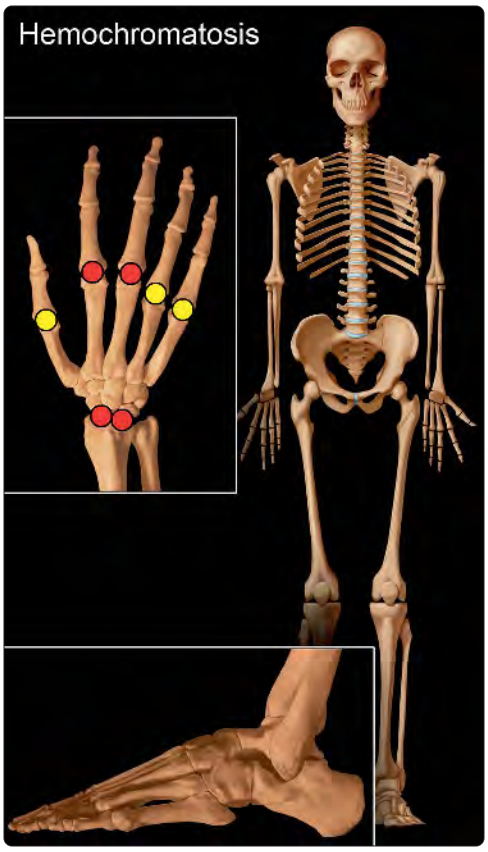


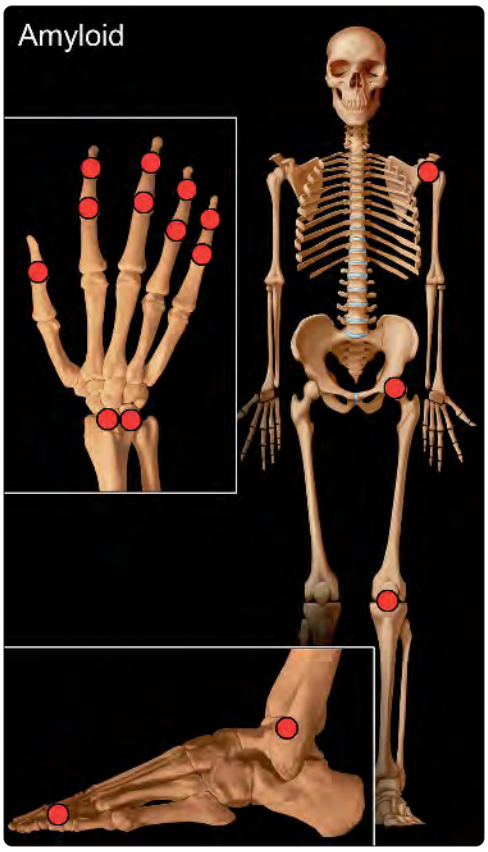






(Left) *Gout distribution (common: Red; less common: Yellow) is shown. Involvement of the 1st MTP joint is the hallmark of the disease, though other foot & ankle joints may be affected. In the hand, the IP joints are much more frequently affected than the MCP or carpals. Of the more proximal joints, the elbow is more frequently involved than the knee. Axial involvement of the C spine & SI joints is uncommonly seen.* **(Right)** *Pyrophosphate arthropathy distribution is shown. The wrist shows a specific predilection for radiocarpal involvement, often leading to SLAC deformity, while the MCP joints are affected in the hand (2nd & 3rd earlier and more frequently than 4th & 5th). The knee is commonly involved, as are the hips & symphysis pubis. The upper elements of the C spine are often affected as well.*





(Left) *Hemochromatosis distribution (common: Red; less common: Yellow). Disease affecting the wrist shows a distinct predilection for the radiocarpal joint. In the hand, the metacarpophalangeal joints are distinctively involved; the 2nd and 3rd are found to be abnormal both earlier and more severely than the 1st, 4th, and 5th. Note that this distribution is similar to that of pyrophosphate arthropathy in the wrist and hand. The remainder of the skeleton is only rarely affected.* **(Right)** *Amyloid distribution. The large proximal joints (shoulder, hip, and knee) are particularly prone to involvement. In the hand, any joint may be involved, but the interphalangeal joints and radiocarpal joints are more frequently abnormal. Ankle and foot interphalangeal joints may be affected as well.*

Rheumatoid Arthritis of Axial Skeleton

KEY FACTS

TERMINOLOGY

- Chronic progressive systemic inflammatory disease in which joints are primary target

IMAGING

- Purely erosive disease, most frequently involving C1-C2
- Radiographic findings
  - Dens erosions
  - Atlantoaxial subluxation
  - Atlantoaxial impaction: May be unilateral or bilateral
    - If unilateral, collapse results in torticollis
  - Subaxial subluxation
  - Sternoclavicular joints involved in 30% of patients with RA but difficult to visualize on radiograph
  - Osteoporosis
- Radiographs must include lateral flexion-extension
- CT: Additive to radiographs
  - Extent of erosive disease more apparent
  - Atlantoaxial (AA) impaction well shown
- MR: Additive to radiographs
  - Pannus, usually around odontoid, distinctly seen
  - Cord compression and damage directly visualized

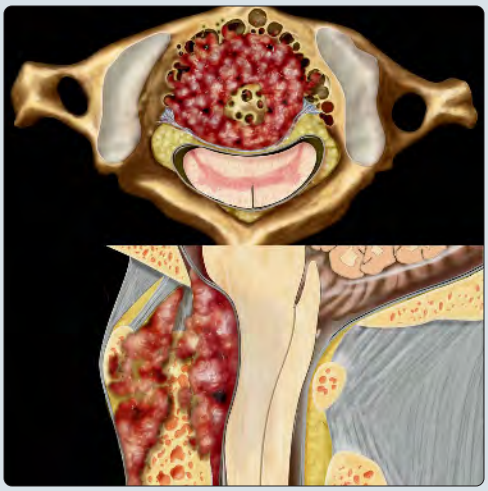
CLINICAL ISSUES

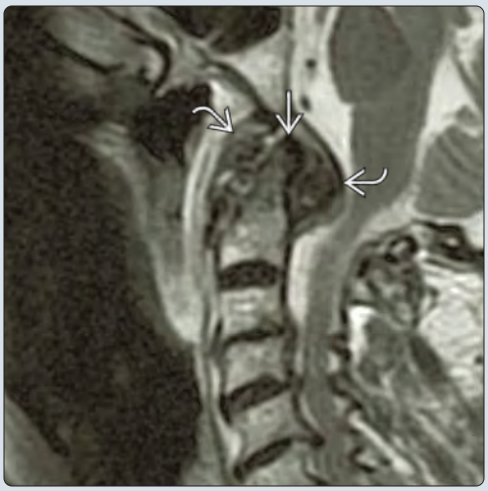
- Patients with axial disease rarely show associated symptoms until very late
  - Cord symptoms with AA impaction
  - Cord symptoms with > 9 mm AA subluxation
  - Unilateral C1-C2 facet disease → painful torticollis
- Patients with axial disease virtually always have significant peripheral disease (hands/feet) as well

DIAGNOSTIC CHECKLIST

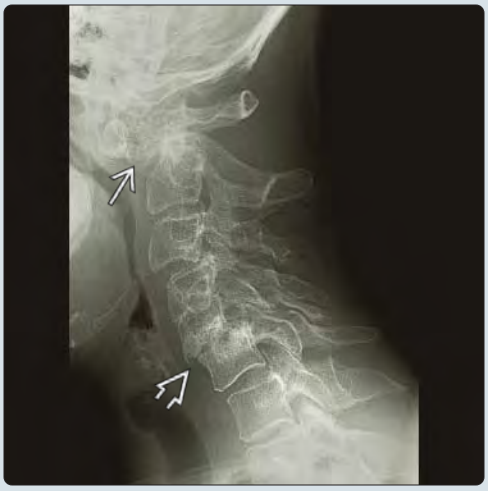
- Watch location of anterior arch of atlas relative to odontoid to evaluate for AA impaction
  - Anterior arch should align with upper portion of dens
- Remember AA subluxation may be underestimated on neutral lateral radiograph and CT

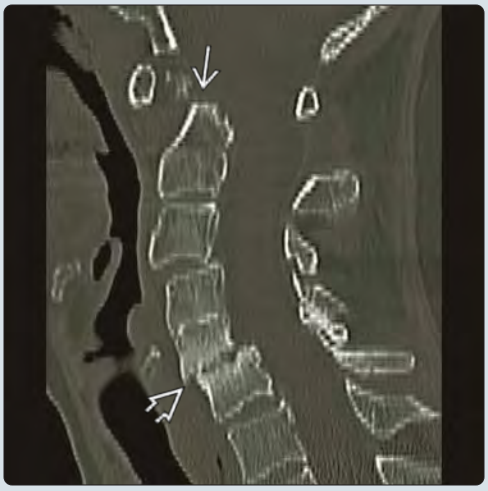
(Left) *Graphic in axial and sagittal planes through the atlantoaxial level of the cervical spine demonstrates the inflammatory pannus surrounding the odontoid process that frequently occurs in patients with rheumatoid arthritis (RA). Note the erosion of the odontoid, as well as focal compression of the spinal cord.* **(Right)** *Sagittal T2WI MR shows extensive erosive changes of the odontoid process, as well as a large amount of pannus with effacement of the thecal sac and posterior displacement of cord.*





(Left) *Lateral x-ray shows severe (> 9 mm) atlantoaxial (AA) subluxation and impaction. Note the disruption of the spinolaminar line at C1-C2. Many of the facets are eroded, and abnormal motion of osteoporotic bone results in endplate destruction and subluxation at the C5-C6 level.* **(Right)** *Sagittal CT in the same patient emphasizes the severe odontoid erosion and AA subluxation with impaction. There is no soft tissue swelling at C5-C6, indicating that the disc space loss is mechanical rather than infectious.*





TERMINOLOGY

Abbreviations

- Rheumatoid arthritis (RA)

Definitions

- Chronic progressive systemic inflammatory disease in which joints are primary target
  - Atlantoaxial subluxation = C1-C2 subluxation = atlantoaxial (AA) subluxation
  - Atlantoaxial impaction = cranial settling = C1-C2 impaction = AA impaction

IMAGING

General Features

- Best diagnostic clue
  - Purely erosive disease, most frequently involving C1-C2 articulation
- Location
  - C1 and C2 facets, uncovertebral joints, dens, peridens bursa
  - Subaxial (below C2) involvement of cervical facets and uncovertebral joints
  - Sternoclavicular joints involved in 30% of patients with RA but difficult to visualize on radiograph
  - Thoracolumbar spine and sacroiliac joints may have microscopic involvement not manifested on radiographs
- Size
  - Ranges from mild pannus formation to significant instability with cord involvement

Radiographic Findings

- Dens erosions
  - Due to inflammatory pannus in surrounding bursa
  - Seen on lateral or open mouth odontoid views
- AA subluxation
  - Due to disruption of transverse ligament by inflammatory pannus
  - Normal distance between inferior margin of anterior arch of atlas and dens < 4 mm
    - AA subluxation generally not symptomatic until distance reaches 9 mm
  - Evaluated on lateral radiograph
    - May not be apparent without flexion-extension views
      - Flexion generally shows maximal subluxation
      - Extension may indicate pannus preventing full reduction
- AA impaction
  - Due to erosions and collapse of facet joints at C1-C2
    - With bilateral facet collapse, dens may protrude through foramen magnum and cause cord symptoms
  - Evaluated on lateral radiograph
    - Anterior arch of atlas should normally be adjacent to upper portion of dens
      - Anterior arch location adjacent to body of dens or body of C2 indicates AA impaction
    - Impaction of dens through foramen magnum may not be easily seen due to overlying mastoids on lateral radiograph
      - If impaction suggested by position of anterior arch of atlas relative to C2, CT will show extent of process
- Unilateral AA impaction
  - Due to erosions and collapse of single facet joint at C1-C2
    - Unilateral collapse results in acute torticollis
- Subaxial subluxation
  - Due to combination of ligament instability, facet and uncovertebral joint erosive disease, and osteoporosis
    - Erosions and ligament disruption allow abnormal motion across vertebral body endplates → subluxation
    - Uncovertebral joint pannus, combined with underlying osteoporosis and abnormal motion → endplate destruction
  - Seen on lateral radiograph as stair-step deformity
    - Associated endplate destruction may suggest infection
  - Fusion in adult RA extremely uncommon but may occur following endplate destruction
- Osteoporosis

CT Findings

- Extent of erosive disease more apparent
- May see pannus around dens
- Coronal and sagittal reformats show extent of facet and uncovertebral erosions
- AA impaction may be much more evident
  - Projection of dens > 5 mm above Chamberlain line (extends from hard palate to opisthion on sagittal)
  - Intersection of dens with Wackenheim line (extends along clivus on sagittal)
- Note that AA subluxation may be reduced (and therefore underestimated) on CT since patient is supine in scanner

MR Findings

- T1WI
  - Pannus is mass-like low signal
- T2WI
  - Pannus has heterogeneous low and high signal
  - High signal synovial fluid in facets and uncovertebral joints
  - High signal erosions and marrow edema
- STIR
  - Same as T2WI, but abnormal cord signal may be more apparent
    - Contusion or syrinx
- T1WI C+ FS
  - Increased sensitivity in diagnosis of early RA
  - Pannus shows avid enhancement
  - Early erosions enhance

Imaging Recommendations

- Best imaging tool
  - Radiographs, with lateral flexion-extension
  - CT: Additive to radiographs
    - Erosion extent better visualized
    - AA impaction well shown
  - MR: Additive to radiographs
    - Pannus distinctly seen, usually around odontoid
    - Cord compression and damage directly visualized

DIFFERENTIAL DIAGNOSIS

DDx of Atlantoaxial Subluxation

- Seronegative spondyloarthropathies
  - Subluxation may be seen in ankylosing spondylitis, enteropathic spondylitis, psoriatic spondylitis, or chronic reactive spondylitis
  - Syndesmophytes indicate spondyloarthropathy
  - Vertebral body or facet fusion indicate spondyloarthropathy; fusion occurs extremely rarely in adult RA
- Juvenile idiopathic arthritis
  - Often have fused levels, with waisting of hypoplastic bodies

DDx of Endplate Abnormalities

- Disc space infection
  - Single level more suggestive of infection
  - Normal bone density at adjacent levels suggests infection
- Spondyloarthropathy of hemodialysis
  - Endplate destruction, often with listhesis
  - May be multilevel
  - Generally does not involve C1-C2

PATHOLOGY

General Features

- Etiology
  - Unknown etiology for RA
  - Pathophysiology presumed to relate to persistent immunologic response of genetically susceptible host to some unknown antigen
- Genetics
  - Genetic predisposition
    - Concordance in monozygotic twins (25%)
    - 1st-degree relatives develop RA at rate 4x that of general population
  - Still, individual not likely to have affected family member

Gross Pathologic & Surgical Features

- Synovial lining is hypertrophic, edematous
- Joint distension, bone erosion, cartilage destruction

Microscopic Features

- Organized accumulations of CD4 helper T cells, antigen presenting cells, lymphoid follicles
- Large amounts of immunoglobulin produced, including rheumatoid factor
- Angiogenesis in synovium

CLINICAL ISSUES

Presentation

- Most common signs/symptoms
  - Patients with axial disease rarely show associated symptoms until very late
    - Cord symptoms with AA impaction
    - Cord symptoms with significant AA subluxation (> 9 mm)
    - Painful torticollis with unilateral C1-C2 facet collapse
  - Patients with axial disease virtually always have significant peripheral disease (hands/feet) as well

Demographics

- Age
  - Peak age of onset: 4th-5th decades
- Gender
  - M:F = 1:3
- Epidemiology
  - RA in 1% of worldwide population
    - 5% in some Native American populations
  - 50% of RA patients have cervical spine involvement

Natural History & Prognosis

- With severe disease, may develop radiculopathy or cervical myelopathy
- ↑ morbidity and mortality if patient has craniocervical junction instability

Treatment

- Treatment of RA: Generally combination, aimed at pain relief while escalating therapy rapidly to suppress disease prior to joint destruction
  - Nonsteroidal antiinflammatory drugs
    - Symptomatic relief; do not alter disease process
  - Glucocorticoids (oral or intraarticular)
    - Controls inflammation rapidly; allows time for slower-acting drugs to take effect
  - Disease-modifying antirheumatic drugs
    - Suppresses joint destruction (e.g., methotrexate, sulfasalazine, antimalarials, gold)
  - Biologics: Anti-TNF-α drugs, antiinterleukin-1
    - Role of cytokines (especially tumor necrosis factor-α and interleukin-1) in pathophysiology of RA now recognized
- Treatment of symptomatic cervical spine instability
  - Transoral odontoidectomy
  - Posterior fusion occiput-C1-C2
  - Laminectomy and facet fusion/stabilization
- Some believe liberal surgical treatment of C1-C2 instability may improve morbidity and mortality

DIAGNOSTIC CHECKLIST

Image Interpretation Pearls

- Watch location of anterior arch of atlas relative to odontoid to evaluate for AA impaction
- Remember AA subluxation may be underestimated on neutral lateral radiograph and CT

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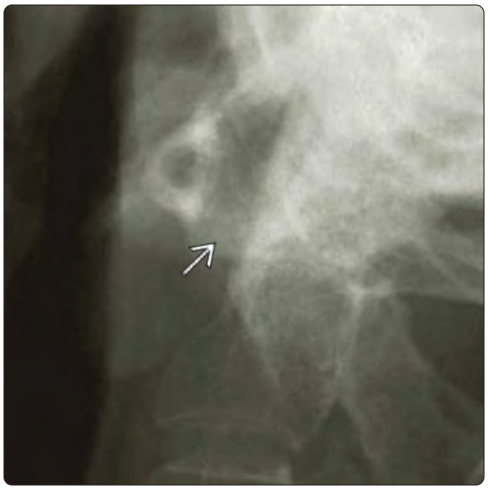
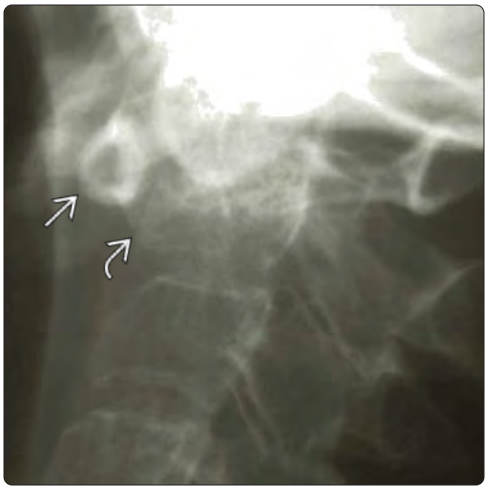
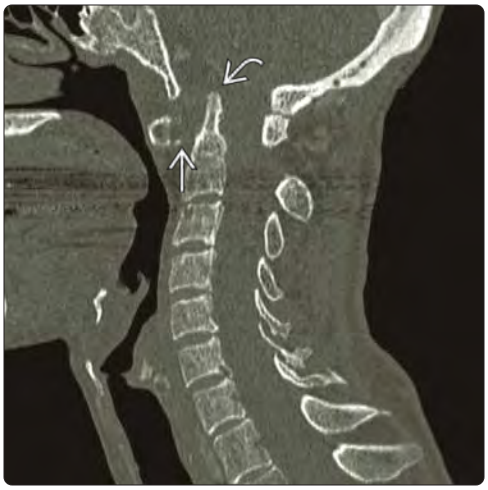
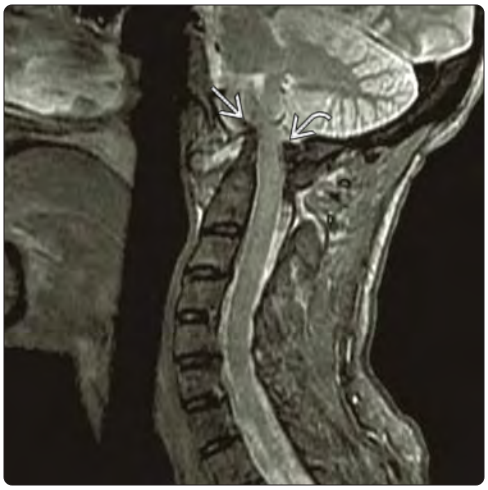


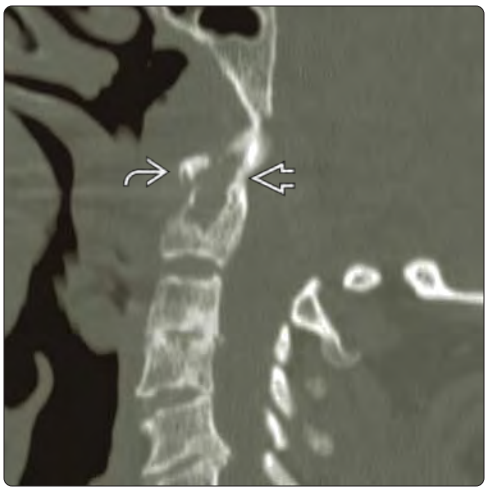


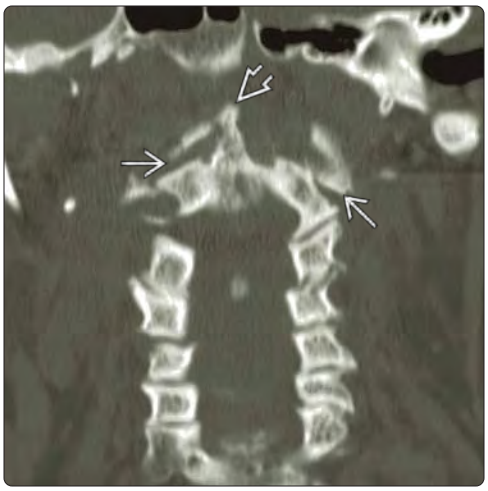
(Left) *Lateral radiograph shows AA subluxation. Even more importantly, the anterior arch of the atlas is in a low position relative to the odontoid. This indicates AA impaction. The actual impaction is difficult to visualize radiographically because of superimposed mastoid processes.* **(Right)** *Lateral radiograph of the same patient 1 year later shows the anterior arch of the atlas located at the level of the body of the odontoid. The AA impaction is severe.*





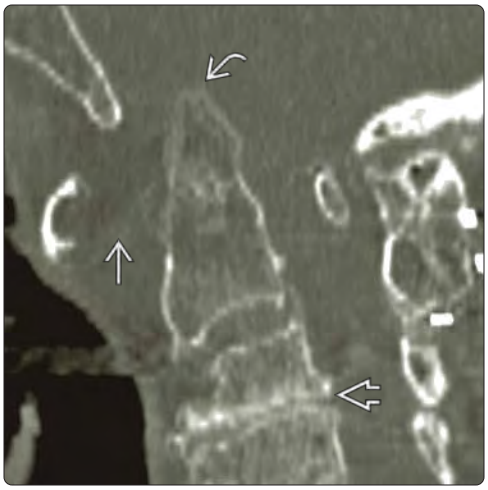
(Left) *Sagittal CT shows a typical pattern of cranial settling in RA due to AA impaction. There is upward translocation of the dens with respect to the foramen magnum; Wackenheim clival line is abnormal. Dens erosion and AA subluxation are noted as well.* **(Right)** *Sagittal STIR MR of the same patient shows the impacted dens position and narrowing of the subarachnoid space at the foramen magnum with cord compression between the odontoid and opisthion.*

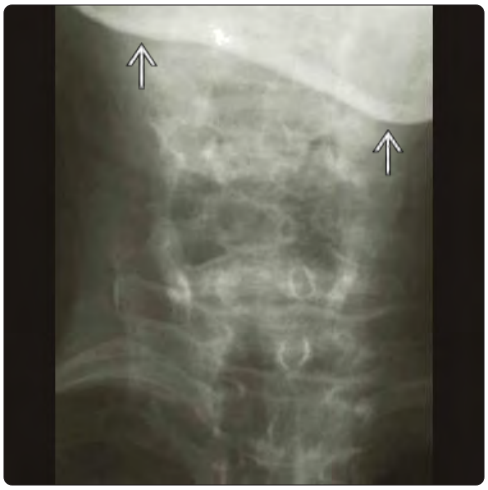




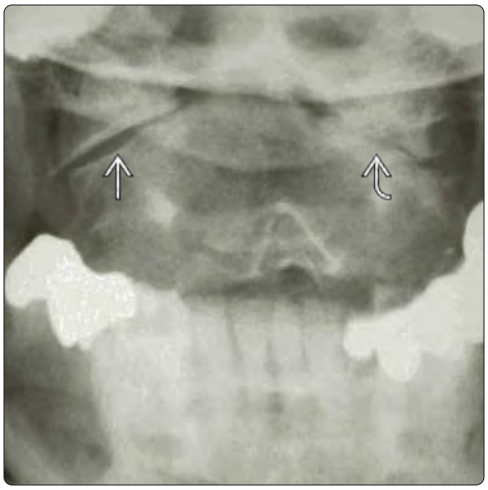
(Left) *Sagittal NECT shows severe erosive changes of C1 and odontoid process. The location of the remnant of anterior arch of C1 opposite the body of C2 indicates AA impaction.* **(Right)** *Coronal NECT shows the impacted tip of the eroded odontoid. It also demonstrates the erosions and collapse of the lateral masses (facets) at C1-C2. Compare this to the facets of the subaxial spine, which are normal. It is the collapse of the lateral masses that results in AA impaction.*

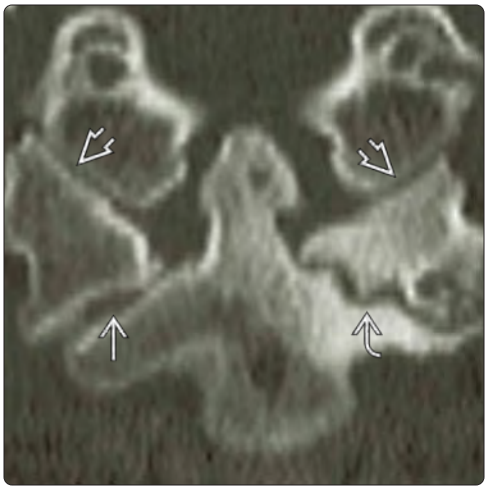
(Left) *Sagittal bone CT in a patient with RA shows erosions at multiple levels in the cervical spine, causing instability at occiput-C1, AA subluxation, AA impaction (cranial settling), as well as endplate erosions and uncovertebral erosions at lower levels. Such diffuse involvement is common.* **(Right)** *AP radiograph in a patient with RA shows angulation of the mandible. The cervical spine is straight; this mandibular tilt should suggest unilateral AA impaction.*



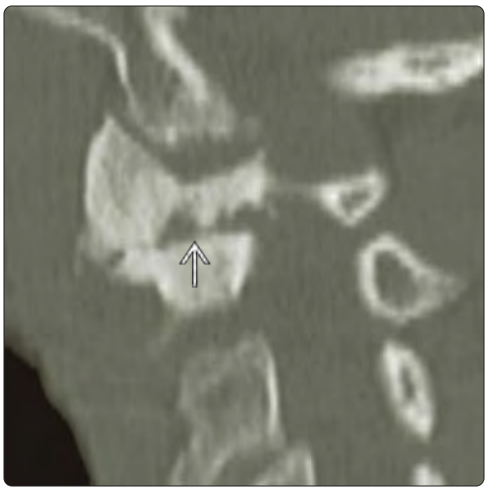


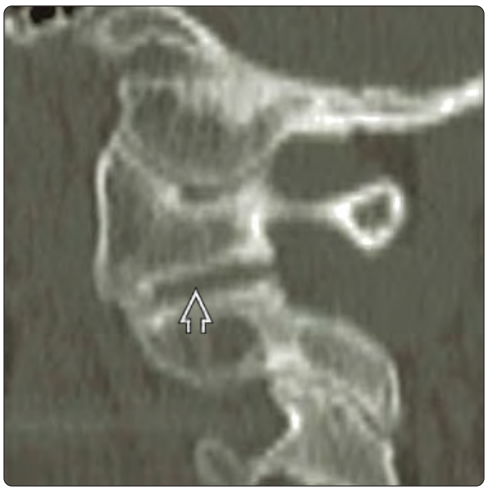
(Left) *Open mouth odontoid radiograph in an RA patient with acute torticollis shows a normal right C1-C2 facet but eroded left C1-C2 facet. This discrepancy may result in unilateral collapse of this joint and associated painful torticollis.* **(Right)** *Coronal bone CT of the same patient confirms the erosions and collapse of the left C1-C2 facet compared with the normal right C1-C2 facet. Note that the occiput-C1 facets are normal as well.*

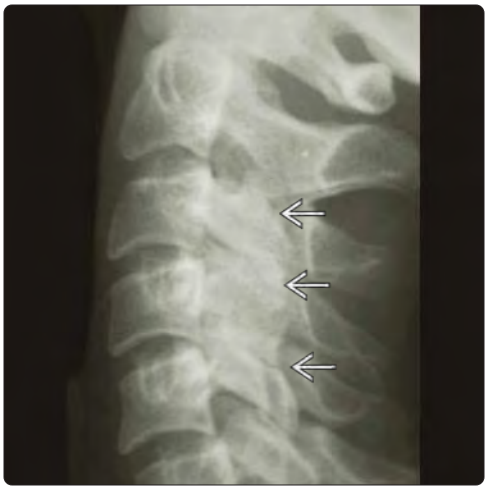


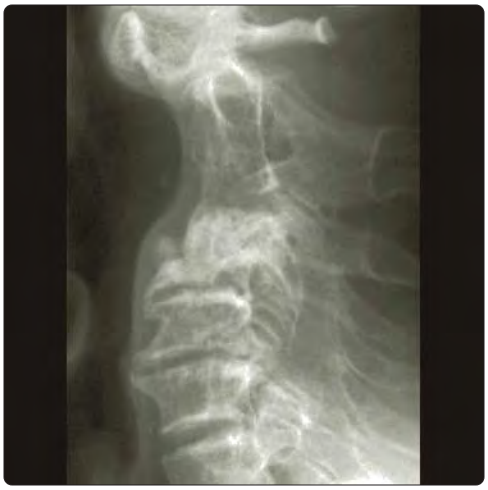


(Left) *Sagittal bone CT in this same RA patient shows the erosions and collapse of the left C1-C2 facet.* **(Right)** *The normal right C1-C2 facet joint in this same patient is shown for comparison. Facet joints are at risk for erosive disease at any location of the spine. However, C1-C2 facets seem particularly at risk. If both C1-C2 facets erode and collapse, the patient may develop AA impaction. With unilateral collapse, the patient develops a painful torticollis.*

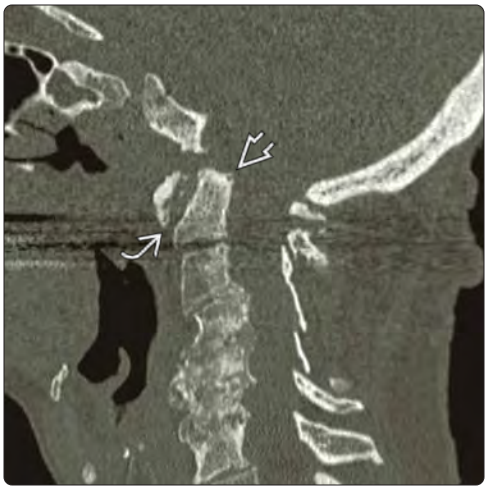


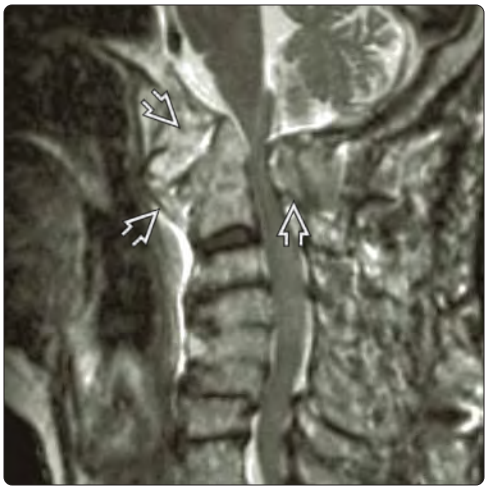




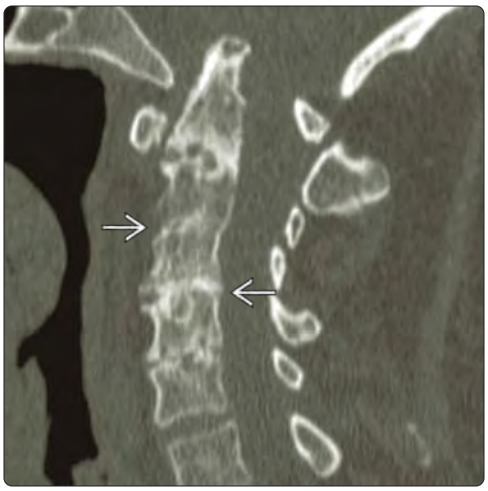


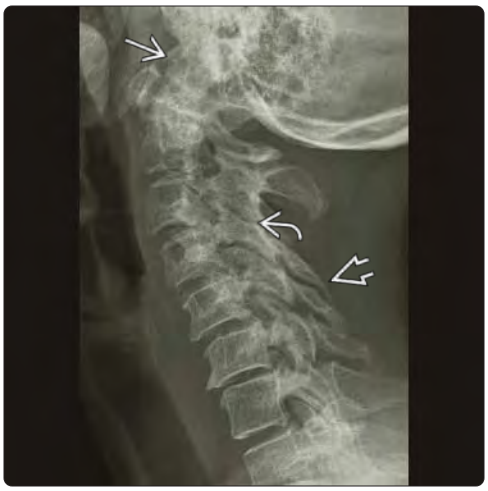
(Left) *Lateral radiograph in an RA patient with normal AA structures shows erosive change of multiple facet joints. This has not yet resulted in abnormal alignment or endplate destruction.* **(Right)** *Lateral radiograph in a patient with RA shows severe erosive disease, resulting in AA subluxation, facet erosions, and presumed ligamentous disruption. The combination leads to malalignment and subsequent endplate mechanical erosions and disc destruction.*





(Left) *Sagittal NECT shows C1-C2 impaction. The odontoid process is eroded. The subaxial spine shows marked diffuse disc and endplate degeneration due to a combination of ligamentous disruption and facet/uncovertebral joint erosions.* **(Right)** *Sagittal STIR MR shows pannus formation causing severe cord compression. C1-C2 subluxation and impaction are prominent. Multilevel subluxations of the subaxial cervical spine reflect facet and uncovertebral involvement.*





(Left) *Sagittal CT shows RA C1-C2 impaction. Note the fusion at several vertebral bodies. Fusion in RA is uncommon but may occur at the site of these mechanical erosions and disc degeneration.* **(Right)** *Lateral x-ray shows mild AA subluxation and severe AA impaction. Note also the eroded facets at multiple levels as well as the thinned spinous processes, typical of RA. The patient has mild stair-step subluxations of the vertebral bodies secondary to a combination of abnormal motion and osteoporosis.*

Rheumatoid Arthritis of Shoulder and Elbow

KEY FACTS

TERMINOLOGY

- Chronic progressive systemic inflammatory disease in which joints are primary target

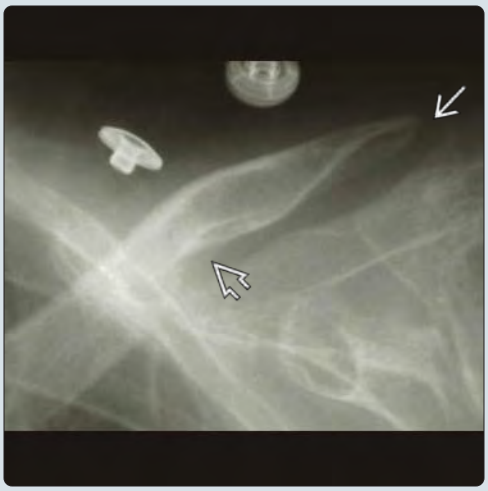
IMAGING

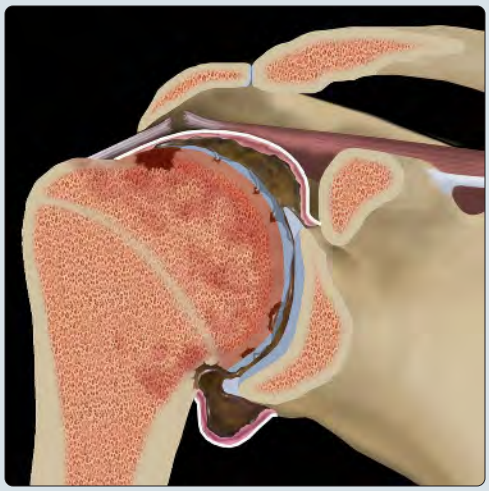
- Purely erosive arthropathy
- Uniform cartilage narrowing
- Osteoporosis
- Glenohumeral joint
  - Largest and earliest erosions at margin
  - Eventually, erosions uniformly involve humeral head and glenoid
  - Subchondral cysts may be large, but underlying osteoporosis may mask their size
  - Elevation of humeral head due to rotator cuff tear
  - Hatchet-like mechanical erosion at medial surgical neck of humerus
  - Swelling of joint may be prominent due to decompression of synovial fluid through rotator cuff tear (RCT) into subacromial/subdeltoid bursa
- Elbow joint
  - Effusion (elevated anterior and posterior fat pads)
  - Olecranon bursitis common in rheumatoid arthritis (RA)
  - Erosions uniform throughout joint
- MR in RA
  - Thickened, low signal, avidly enhancing pannus and synovium
  - Low signal rice bodies within effusion
  - Subchondral marrow edema
  - RCT, partial or complete
  - Decompression of synovial effusion well seen

DIAGNOSTIC CHECKLIST

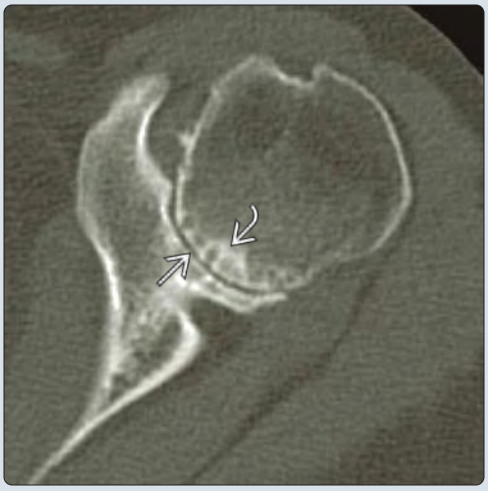
- Other causes of synovitis, especially infection if monoarticular

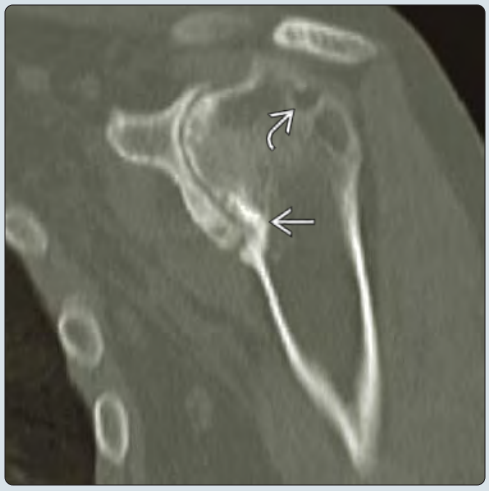
(Left) *AP radiograph shows erosions of the distal end of the clavicle ⇨ as well as at the coracoclavicular ligament insertion site of the clavicle ⇨, typical sites of erosions in rheumatoid arthritis (RA).* **(Right)** *Coronal graphic shows advanced RA of the shoulder. Thickened synovium lining the capsule is distended by effusion. Cartilage is thinned uniformly. Large marginal erosions are seen where bone is not covered by cartilage, and smaller subchondral erosions are present. Marrow edema and rotator cuff tear complete the picture.*





(Left) *Axial bone CT demonstrates the typical uniform glenohumeral cartilage loss ⇨ that is seen in RA, along with humeral head erosions and subchondral cysts ⇨.* **(Right)** *Coronal bone CT shows marginal erosion at the humeral head ⇨. The head is subluxated superiorly secondary to chronic rotator cuff tear. This results in a mechanical erosion of the osteoporotic bone at the surgical neck of the humerus ⇨; this puts the patient at additional risk of fracture.*





TERMINOLOGY

Abbreviations

- Rheumatoid arthritis (RA)
- Rotator cuff tear (RCT)

Definitions

- Chronic progressive systemic inflammatory disease in which joints are primary target

IMAGING

General Features

- Best diagnostic clue
  - Purely erosive arthropathy
  - Uniform cartilage narrowing
  - Osteoporosis
- Location
  - Earliest changes are in synovium, followed by cartilage and bone

Radiographic Findings

- Acromioclavicular (AC) joint
  - Erosions on both sides of joint
  - End-stage penciling of clavicle into thin point
  - May resorb clavicle at insertion of coracoclavicular ligaments
- Glenohumeral joint
  - Uniform cartilage narrowing
  - Erosions
    - Largest and earliest at margin (junction of cartilage-covered humeral head and greater tuberosity)
    - Eventually, erosions uniformly involve humeral head and glenoid
    - End-stage destruction of entire head and glenoid
  - Subchondral cysts may be large, but underlying osteoporosis may mask their size
  - Elevation of humeral head due to RCT
    - With chronicity, head seems to articulate with underside of acromion, molding acromion into concavity
  - Hatchet-like mechanical erosion at medial surgical neck of humerus
    - Due to chronic elevation of humeral head and consequent rubbing of osteoporotic humeral neck against inferior glenoid
    - Increases risk of insufficiency fracture across surgical neck
  - Swelling of joint may be prominent due to decompression of synovial fluid through RCT into subacromial/subdeltoid bursa
- Elbow joint
  - Effusion (elevated anterior and posterior fat pads)
  - Swelling about joint
    - Over olecranon: Olecranon bursitis common in RA
    - Elsewhere due to decompression of synovial fluid through capsule or into bicipital radial bursa
  - Erosions uniform throughout joint
    - Equally involving capitellum, trochlea, ulna, radial head/neck
    - End-stage uniform destruction of osseous structures

CT Findings

- Same osseous findings as noted on radiograph
  - Erosions and subchondral cysts more apparent
- Inflamed synovium enhances

MR Findings

- T1WI
  - Thickened low signal pannus
  - Low signal effusion, erosions, and subchondral cysts
  - If superimposed amyloid, deposits are low signal
    - Intraarticular or within thickened tendons
- Fluid-sensitive sequences
  - Effusion is high signal
  - Thickened low signal pannus lines synovium
  - Low signal rice bodies within effusion
  - Erosions and subchondral cysts high signal
  - Subchondral marrow edema
  - RCT, partial or complete
  - Decompression of synovial effusion well seen
    - Shoulder: Generally through RCT into subacromial/subdeltoid bursa
    - Elbow: Into bicipital radial bursa or into adjacent soft tissues
  - If amyloid deposits present, they remain low signal
  - ↑ signal ulnar nerve from elbow impingement
- T1WI FS + contrast
  - Avid enhancement of thick pannus along synovium
  - Subchondral cysts and erosions have high signal surrounding low signal fluid contents

Imaging Recommendations

- Best imaging tool
  - Radiographs generally make diagnosis
  - MR often adds information
    - Integrity of rotator cuff
    - Early (preradiographic) diagnosis: Synovitis
    - Early osseous destructive phase: More accurate evaluation of erosions and subchondral cysts

DIFFERENTIAL DIAGNOSIS

Septic Arthritis

- In differential of early RA
- Enhancing synovitis, effusion
- Marrow edema or early erosions

Charcot, Neuropathic

- In differential of late shoulder RA
- Destruction of humeral head and glenoid
- Large effusion, decompressing into subdeltoid via RCT
- Presence of osseous debris and lack of pannus helps to differentiate
- Generally unilateral; etiology is syringomyelia

Spondyloarthropathies

- Ankylosing spondylitis and enteric spondylitis often involve large proximal joints (shoulder, hip)
- Early in disease, may appear purely inflammatory, with synovitis and erosions
- Later in disease, generally mixed erosive/productive
- Spine/SI joint involvement differentiates from RA

PATHOLOGY

General Features

- Etiology
  - Unknown etiology
  - Pathophysiology presumed to relate to persistent immunologic response of a genetically susceptible host to some unknown antigen
- Genetics
  - Genetic predisposition
    - Concordance in monozygotic twins: 25%
    - 1st-degree relatives develop RA at rate 4x that of general population
  - Individual not likely to have affected family member
- Associated abnormalities
  - Subcutaneous rheumatoid nodules in 30%
    - Extensor surfaces (ulna, Achilles) and digits
  - Amyloid deposition
  - Thoracic: Pleural effusions, rheumatoid nodules, rarely interstitial fibrosis
  - Vasculitis
  - Felty syndrome: RA + splenomegaly + leukopenia
  - Sjögren syndrome: RA + keratoconjunctivitis + xerostomia
  - ↑ risk of lymphoma
  - Lifetime of systemic inflammation may contribute to ↑ risk of cardiovascular disease, renal disease, and infection
  - ↑ mortality rate, reduced survival by 10-18 years

Gross Pathologic & Surgical Features

- Synovial lining is hypertrophic, edematous
- Joint distension, osseous erosion, cartilage destruction
- Rice bodies: Detached synovial villi

Microscopic Features

- Organized accumulations of CD4 helper T cells, antigen presenting cells, lymphoid follicles
- Large amounts of immunoglobulin produced, including rheumatoid factor
- Angiogenesis in synovium

CLINICAL ISSUES

Presentation

- Most common signs/symptoms
  - Symmetric polyarthritis, especially small joints
  - Constitutional symptoms of fatigue, low-grade fever
  - Usually presents over course of weeks or months; occasionally fulminant disease
- Other signs/symptoms
  - Shoulder/elbow
    - Pain, swelling, ↓ range of motion
    - RCT symptoms

Demographics

- Age
  - Peak age at onset: 3rd-5th decades
- Gender
  - M:F = 1:3
- Epidemiology
  - RA in 1% of worldwide population
    - 5% in some Native American populations
  - Shoulder involved in 60% of patients with RA
  - Acromioclavicular involved in 50% of patients with RA
  - Elbow involved in 50% of patients with RA

Natural History & Prognosis

- Up to 60% go into remission
  - With aggressive multidrug Rx, most improve
- With severe erosions and RCT, poor prognosis for shoulder function

Treatment

- Treatment of RA: Generally combination of drugs, aimed at pain relief while escalating therapy rapidly to suppress disease prior to joint destruction
  - Nonsteroidal antiinflammatory drugs
    - Symptomatic relief; do not alter disease progress
  - Glucocorticoids (oral or intraarticular)
    - Controls inflammation rapidly; allows slower acting drugs to take effect
  - Disease-modifying antirheumatic drugs
    - Suppresses joint destruction (e.g., methotrexate, sulfasalazine, antimalarials, gold)
  - Biologics: Anti-TNF-α drugs, antiinterleukin-1
    - Role of cytokines (especially tumor necrosis factor-α and interleukin-1) now recognized
- Surgical treatment
  - Synovectomy may arrest/delay progression
  - Shoulder
    - Poor prognosis for RCT repairs
    - Arthroplasty: Often reverse shoulder device is chosen because of rotator cuff tear
      - RA patients at particular risk for periprosthetic fracture, especially of acromion
  - Elbow
    - Radial head resection → symptomatic relief
    - Arthroplasty often fails (loosening, periprosthetic fracture) due to thin osteoporotic bone

DIAGNOSTIC CHECKLIST

Consider

- Other etiologies of synovitis, especially infection if monoarticular

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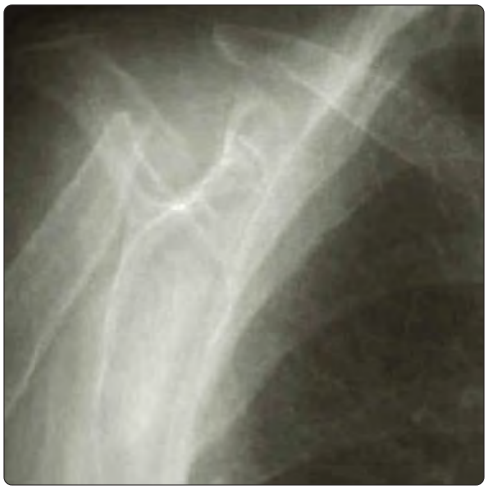
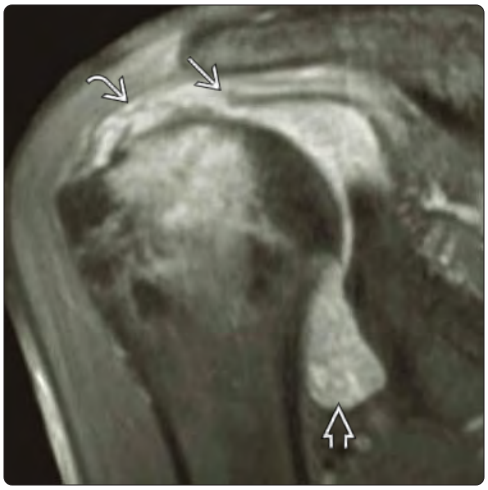
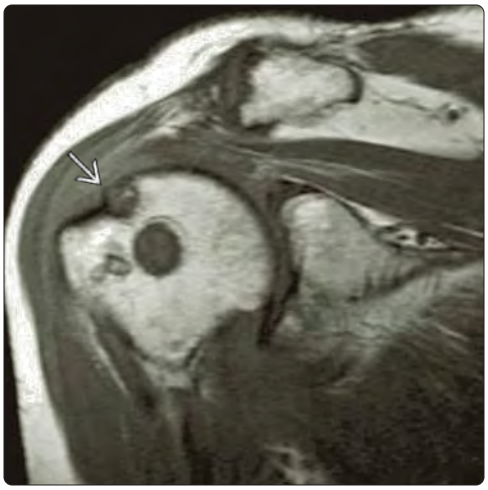
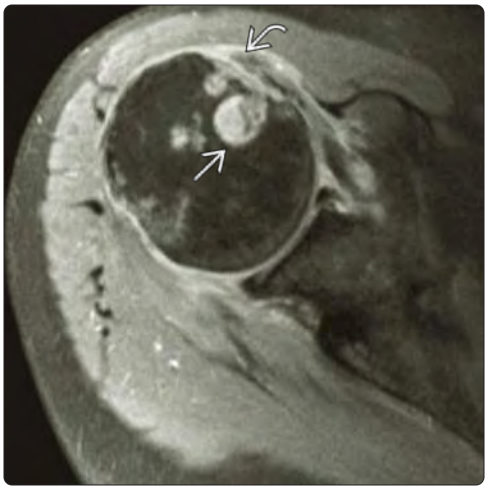


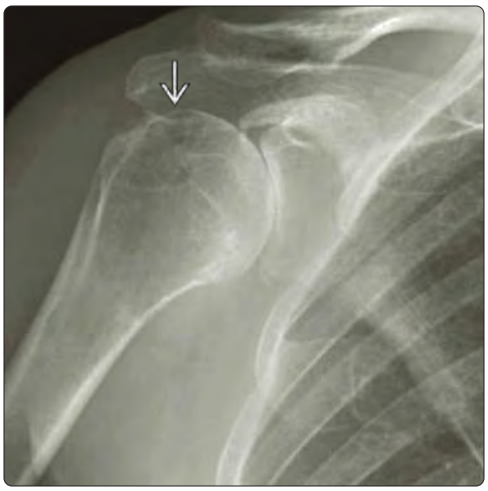


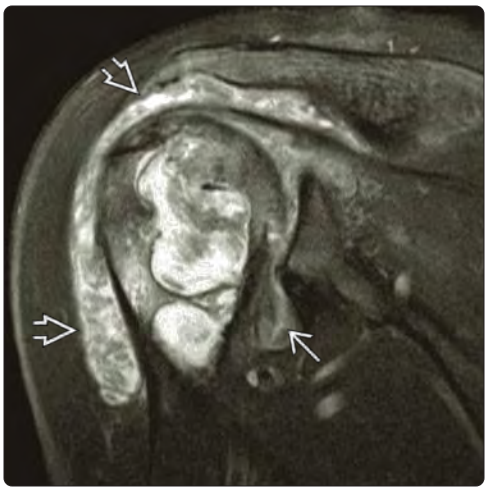
(Left) *AP radiograph shows a case of severe, long-term RA, with erosions of the clavicle, glenoid, and humeral head.* **(Right)** *Coronal PD FS MR shows the retracted infraspinatus tendon, part of the chronic rotator cuff tear that is usually seen with advanced RA. There is mild edema in the humeral head. The glenohumeral joint is distended, and low signal synovitis fills the axillary bursa and extends across the rotator cuff tear into the subacromial bursa. Note the humeral head elevation.*





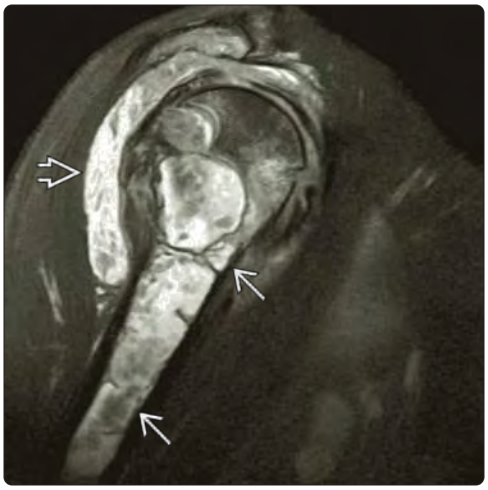
(Left) *Coronal T1WI MR shows typical findings of RA in the shoulder. There are low signal subchondral cysts, as well as a marginal erosion of the humeral head.* **(Right)** *Axial PD FSE FS MR in the same patient shows subchondral cysts as well as a marginal erosion. Note the thin and disrupted subscapularis tendon, with fluid seen both in the glenohumeral joint and subdeltoid bursa. It seems remarkable that the radiograph in this case appeared normal.*

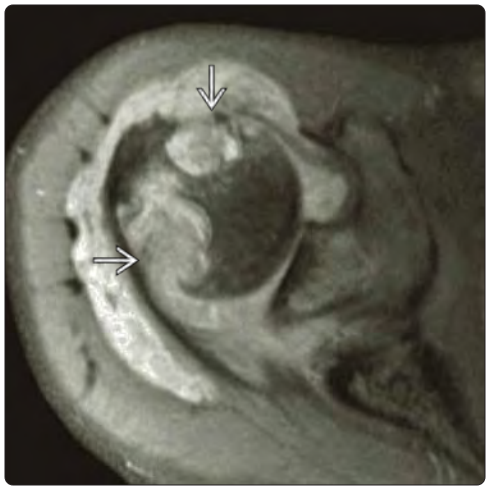




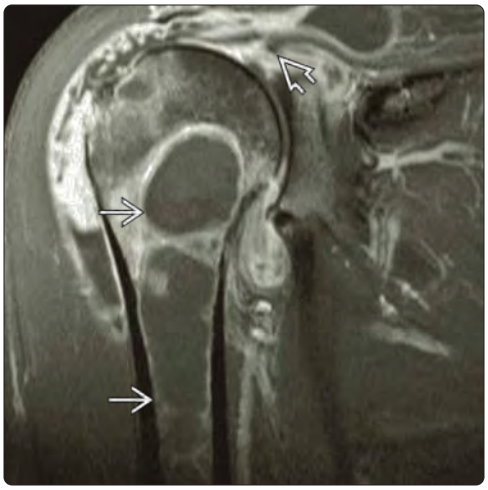
(Left) *AP radiograph in a patient with RA shows only osteopenia and a small marginal erosion. The osteopenia seen on radiographs in patients with RA often disguises the full extent of erosive disease and subchondral cysts.* **(Right)** *Coronal T2WI FS MR in the same patient shows tremendous synovitis in both the glenohumeral joint and subacromial/subdeltoid bursa. The diffuse nodular low signal masses within the fluid are synovitis.*

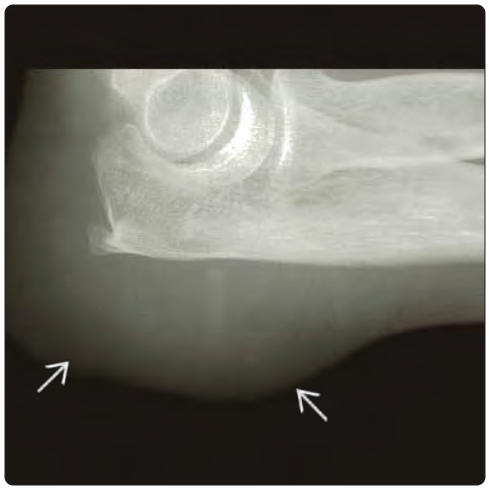
(Left) *Sagittal T2WI FS MR in the same patient emphasizes the tremendous synovitis in both the glenohumeral joint and subacromial/subdeltoid bursa. Both the synovitis and extent of subchondral cysts extending down the marrow are well depicted.* **(Right)** *Axial PD FS MR in the same patient shows the size of the erosions. It is quite remarkable that the radiographs showed only osteopenia and a small erosion, even in retrospect. This humeral metadiaphysis is at risk for fracture.*



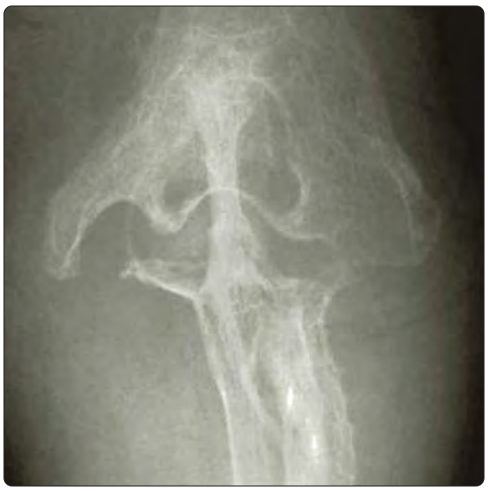


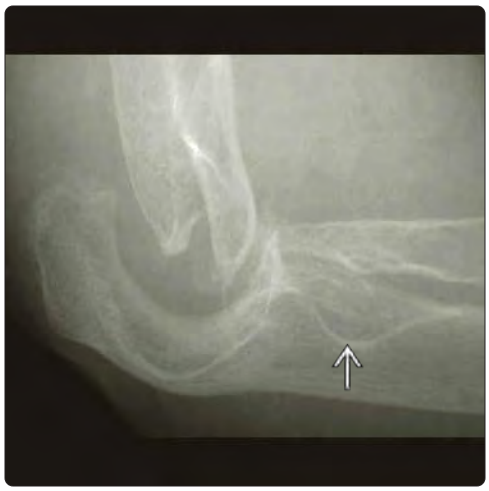
(Left) *Coronal T1 FS postcontrast MR in the same patient confirms that the marrow abnormality represents erosions and subchondral cysts, with low signal fluid surrounded by enhancing synovitis. A rotator cuff tear is seen as well, with retraction of supraspinatus.* **(Right)** *Lateral radiograph of the elbow shows diffuse osteopenia and soft tissue swelling over the olecranon. The location is typical for olecranon bursitis, secondary to RA in this case.*

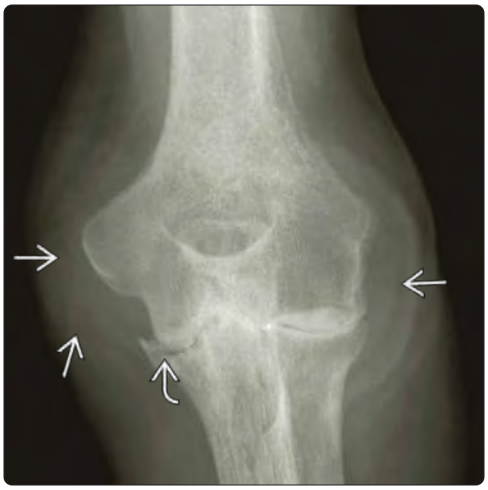


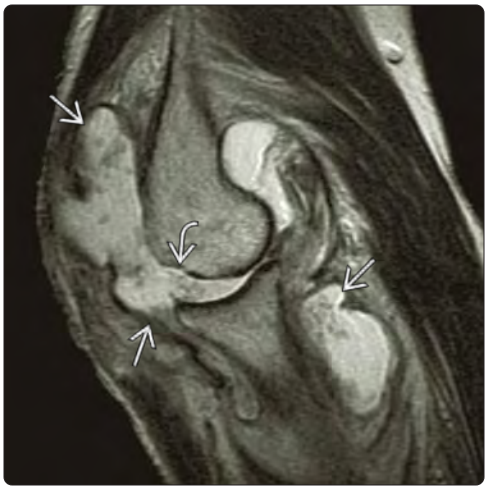


(Left) *Anteroposterior radiograph shows a classic case of severe and long-term RA. There is symmetric erosive disease of the distal humerus, proximal radius, and proximal ulna, along with osteopenia.* **(Right)** *Lateral radiograph confirms the erosions, as well as a mechanical erosion at the proximal shaft of the ulna where the remnant of radial head has been rubbing. The symmetry of the process and purely erosive nature make the diagnosis RA.*

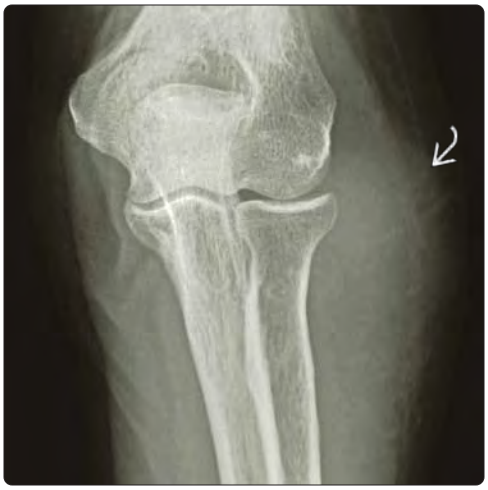


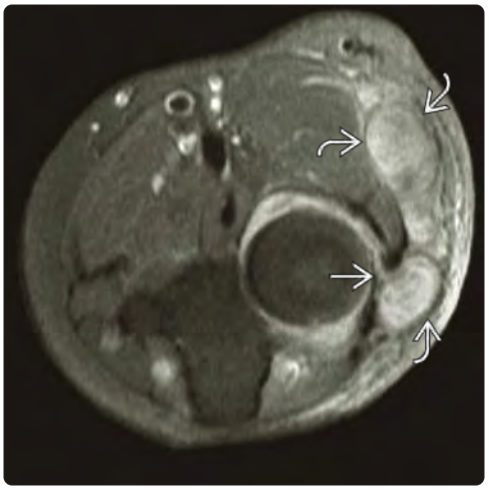




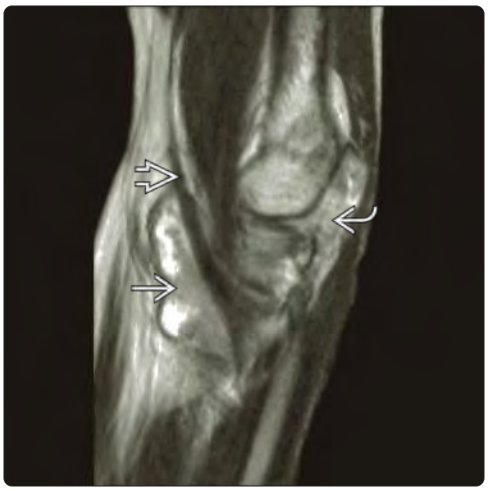


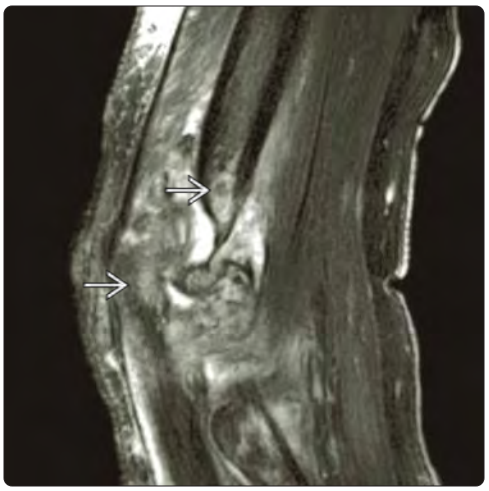
(Left) *AP radiograph shows extensive uniform thinning of cartilage throughout the elbow and subchondral erosions at the coronoid. There is extensive soft tissue swelling and diffuse osteopenia. No productive change is seen; the findings are typical of RA.* **(Right)** *Sagittal T2FS MR in the same case of RA shows complete loss of cartilage and thinning of cortex at the capitellum. The fluid is all contained within an extremely distended joint and contains low signal material that has been termed rice bodies.*





(Left) *AP radiograph shows a lateral soft tissue mass but no other abnormalities in a patient with RA.* **(Right)** *Axial PD FS MR performed to evaluate the soft tissue mass in the same patient shows synovitis and fluid surrounding the radial neck. There is a thin neck of fluid extending from the joint effusion to the mass. This proves that the mass is simply fluid from the joint, which has decompressed into the soft tissues laterally, as happens in restricted joints with active synovitis.*





(Left) *Sagittal T2WI MR shows bicipital radial bursitis related to RA. Lobular-enhancing synovitis is seen in the bicipital radial bursa around the biceps tendon. Fluid and synovitis are also seen in the elbow joint.* **(Right)** *Sagittal T2WI MR in a patient with RA shows joint destruction, large effusion, and abnormal bone marrow signal. These findings could be due simply to advanced RA, but infection must be considered and was proved surgically.*

Rheumatoid Arthritis of Wrist and Hand

KEY FACTS

IMAGING

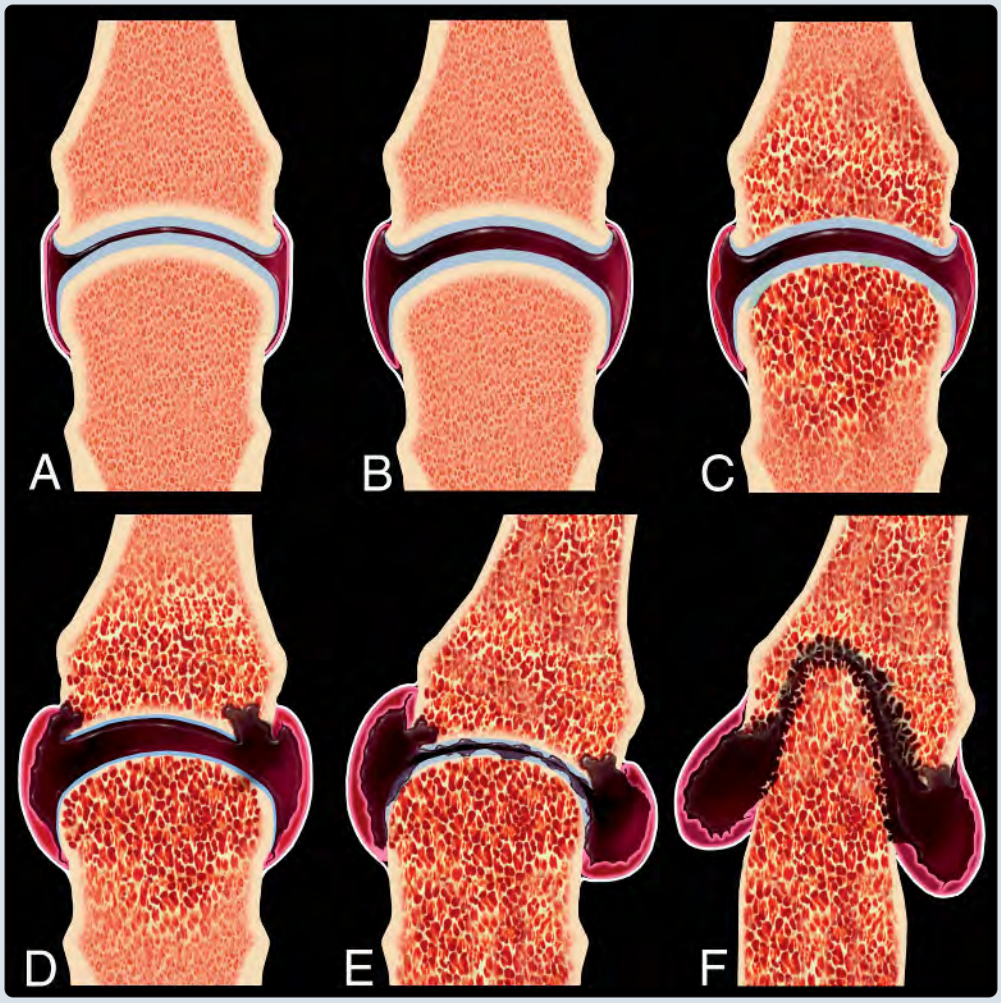
- Erosions
  - Earliest osseous pattern is loss of cortical distinctness, followed by dot-dash pattern of cortical loss
  - Marginal erosions tend to occur early in portion of bone which is within capsule but not covered by cartilage
  - Direct subchondral erosions
  - Late aggressive disease: Pencil-in-cup appearance in phalanges
- Ulnar styloid may show capping: Only site of productive change in RA
- Malalignment due to ligament/tendon disruption
- MR: Pannus: Thick, nodular low signal synovium outlined by effusion
  - Marrow edema: Subchondral high signal
  - Thickened, avidly enhancing synovium outlines low signal effusion and erosions
  - Tenosynovitis may be earliest soft tissue abnormality, though nonspecific
- US: Excellent for early effusions in small joints
  - Tenosynovitis and tendon rupture seen directly

CLINICAL ISSUES

- RA in 1% of worldwide population
  - 5% in some Native American populations
- Female > male (3:1)
- Carpus involved in 80% patients with RA
- MCPs involved in 85% patients with RA
- Hand PIP involved in 75% patients with RA

DIAGNOSTIC CHECKLIST

- Earliest RA may be monostotic or asymmetric
  - Must differentiate from septic joint
- Use sites of focal soft tissue swelling to guide you to subtle osseous findings on radiograph
- Watch for cortical indistinctness and dot-dash pattern for earliest radiographic signs of erosion



PA graphic of a PIP joint shows progressive destruction of the joint. (A) is normal, with intact cortex, cartilage, bone density, and capsule. (B) shows early disease, with only synovitis and effusion. (C) shows juxtaarticular osteopenia, with cortex becoming indistinct, the dot-dash pattern. (D) shows thinning of cartilage and marginal erosions in the portion of bone which is intracapsular but not protected by cartilage. (E) shows progression of osteopenia and subchondral erosions extending through cartilage defects. (F) shows arthritis mutilans, with pencil-in-cup deformity, seen in end-stage disease.