Radiology Overview of Arthritic Processes

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DISCLOSURES

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Classification of Arthritides

Non-Inflammatory
- Degenerative Joint Disease

Inflammatory
- Seropositive Arthritide:
  - Rheumatoid Arthritis

- Erosive Osteoarthritis

- Seronegative Arthritides:
  - Psoriatic Arthritis
  - Reactive Arthritis (Reiter’s Disease)
  - Ankylosing Spondylitis
  - Enteropathic

Metabolic
- Gout
- CPPD Disease

Neuropathic
- Neuropathic Joint Disease

Miscellaneous
- DISH (Diffuse Idiopathic Skeletal Hyperostosis)
- PHO (Pulmonary Hypertrophic Osteoarthropathy)
- PVNS (Pigmented Villonodular Synovitis)

Collagen Vascular Disorders
- SLE
- Scleroderma
Degenerative Joint Disease
If we evaluate the x-ray before performing our physical exam, we would expect to find decreased ROM of the 1st MPJ left with possible crepitus & pain associated with dorsiflexion of the hallux. We would likely note a palpable exostosis/spur at the dorsal surface of the 1st MPJ as well as POP to that dorsal flag.
Degenerative Joint Disease

In a **Normal Joint:**
Chondrocytes regulate the extracellular matrix so that the synthesis and degradation of their structural components remain balanced.
Degenerative Joint Disease

In Degenerative Arthritis:
The degradation and repair processes become unbalanced, with abnormal production of metalloproteases, collagenase, cytokines, and growth factors. This results in weakening & biochemical breakdown/damage of the cartilaginous structure.

- Softening & fibrillation of the cartilage
- Thickening of the joint capsule & synovial hypertrophy
Degenerative Joint Disease

Changes in the proteoglycans make the cartilage less resistant to compressive forces in the joint & more susceptible to the effects of stress. The remaining fibers are placed under additional strain, eventually leading to mechanical failure.
Degenerative Joint Disease

Formation of subchondral cysts with sclerotic margins
Degenerative Joint Disease

Asymmetric narrowing of 1st MPJ (R>L).
Degenerative Joint Disease
Rheumatoid Arthritis
Rheumatoid Arthritis

- A chronic, systemic inflammatory disorder that may affect many tissues and organs, but principally attacks synovial joints.

- **Age of onset: 40 – 60 yoa**
  - Peak Incidence: 40 – 50 yoa, males = females
  - Between 20 – 40 yoa, females 3:1 males

- **Lab Findings:**
  - Seropositive for rheumatoid factor antibodies: 70-90%
  - Positive for Anti-CCP (Cyclic Citrullinated Peptide)

- **Distribution:**
  - C-spine
  - Bilateral joint involvement
  - Symmetrical joint space narrowing
Rheumatoid Arthritis

Bilateral Joint Involvement
With Symmetrical Joint Space Narrowing
Rheumatoid Arthritis

Earlier Stage of RA with Marginal Erosions
Rheumatoid Arthritis

- **Forefoot** is the most common site for radiographic changes
  - Forefoot is the initial site of radiographic change in 15% of patients with RA
  - Of the FF joints, the 1st and 5th MPJs are most often affected first

- **Erosive processes** favor the medial-plantar aspect of the 1st-4th MPJs, medial aspect of the hallux IPJ, & medial/lateral aspects of the 5th MPJ
Rheumatoid Arthritis

Fibular Deviation of the Toes
**RA: Soft Tissue Manifestation**

**Rheumatoid Nodule:**

- Extra-articular subcutaneous lesion/mass
- 20%-30% of RA patients will develop nodules
- Occur almost exclusively in patients who are rheumatoid factor positive
- Usually located on extensor surfaces of the arms & elbows
- Can develop at pressure points on the feet & knees
Rheumatoid Arthritis
Later Stage of RA with Fibular Deviation at the MPJs & Joint Destruction

Licked Candy Stick Appearance
Narrowing of the ankle, STJ, and TN joint is a radiographic feature seen towards the end of the “early radiographic changes period” and continues on in the later stages of RA. Notice the disuse osteopenia/deossification & cortical thinning, as well as the presence of pseudocysts at the talar head/neck.
Rheumatoid Arthritis

In this x-ray, we see absent joint spaces throughout the lesser tarsus (signifying ankylosis) with the exception of the narrowed CC joint.
Metabolic Arthritides
Gouty Arthritis
Gouty Arthritis

- In-borne error of purine metabolism
  - Hyperuricemia

- Clinical features
  - Age: 40 – 50 yoa
  - Gender: Male 20:1 Female

- Lab findings
  - Elevated serum uric acid
    - 10% uricosuric
  - Polarizing microscopy
    - Negatively bi-refringent urate crystals
    - Needle-shaped & blue in color when oriented perpendicular to the compensator
Gouty Arthritis
Gouty Arthritis

Asymmetric Joint Involvement
Gouty Arthritis

- **Primary gout** (hormonal/genetic factors cause metabolic abnormalities)
  - Hyperuricemia due to:
    - **Over-production** of uric acid due to error in purine metabolism
    - **Under-excretion** of urates by kidneys

- **Secondary gout**
  - Hyperuricemia precipitated by drug therapy or a medical condition
    - Drug therapy: **SPEED**- Salicylates, Pyrazinamide, Ethambutol, Ethanol, Diuretics (ie. thiazide - decrease excretion of uric acid)
    - Hyperparathyroidism – increases serum Ca which impairs renal excretion of urates
Gouty Arthritis

Asymptomatic Hyperurecemia:
- Elevated urate levels in patients with no known hx. of gout or renal disease, no known disorders/drugs which elevate uric acid levels.

![The Danger of Hyperuricemia](chart.png)
Gouty Arthritis

Tophi with Extensive Erosions & Joint Destruction seen with Chronic Gout
Gouty Arthritis

Tophi
Gouty Arthritis

Tophi forming away from the 1st MPJ
Gouty Arthritis
Inflammatory Seronegative Arthritides
Psoriatic Arthritis
Psoriatic Arthritis

**Incidence:**
- 7 - 15% of patients with psoriasis will develop psoriatic arthritis
- 80% in those exhibiting nail changes

**Clinical features:**
- Age: 20 – 50 yoa
- No gender predilection

**Lab findings:**
- HLA-B27 antigen
  - 25%-75% SI involvement
  - 30% with peripheral joint involvement (hands)

**Distribution:**
- Asymmetrical joint involvement (typically DIPJs)
- Unilateral joint involvement
Psoriatic Arthritis

Asymmetrical & Unilateral Joint Involvement
Psoriatic Arthritis

Take Home Point:
Erosions & Bone Formation
Psoriatic Arthritis

- **Soft-tissue changes**
  - Inflammatory synovitis leads to symmetrical soft-tissue edema around involved joint, similar to RA
  - However, in PA the edema extends beyond joint creating a **sausage-like appearance of the digit**
Psoriatic Arthritis

Periostitis
Psoriatic Arthritis

Acro-osteolysis
Psoriatic Arthritis

**Erosions** (continued)

- Typically occur simultaneously at medial/lateral margins & progress across entire articular surface
- With progression, bony ends can become tapered like a “pencil point”
  - Cup and saucer
  - Pencil in cup
  - Mortar and pestle
  - Whittling
- Telescoping appearance - “Main-en-lorgnette”
  - Seen in severe PA/arthritis mutilans
Psoriatic Arthritis

“Pencil Point” Metatarsals
Psoriatic Arthritis

IPJ Whiskering:

- Resembles stubble of new beard growth
- Spiculated appearance that radiates away from the bone margin
- Ill-defined sclerosis often accompanies whiskering
Reactive Arthritis
(Reiter’s Disease)
Hans Conrad Julius Reiter.**Hans Conrad Julius Reiter**

(February 26, 1881 – November 25, 1969) was an infamous German physician who was convicted of war crimes for his medical experiments at the Buchenwald concentration camp. He wrote a book on "racial hygiene" called Deutsches Gold, Gesundes Leben - Frohes Schaffen. Reiter was born in Reudnitz, near Leipzig in the German Empire. He studied medicine at Leipzig and Breslau (now Wrocław), and received a doctorate from Tübingen on the subject of tuberculosis. After receiving his doctorate, he went on to study at the hygiene institute in Berlin, the Pasteur Institute in Paris and St. Mary's Hospital in London, where he worked with Sir Almroth Wright for two years.[1] Reiter was also known for implementing strict anti-smoking laws in Nazi Germany.
Reiter was a member of the *Schutzstaffel* during World War II and participated in medical experiments performed by the Nazis. After the Nazis were defeated, he was arrested by the Red Army in Soviet Union-occupied Germany and tried at Nuremberg. During his detention, he admitted to knowledge of involuntary sterilization, euthanasia, and the murder of mental hospital patients in his function as the gatherer of statistics and acting as “quality control” officer, and to helping design and implement an explicitly criminal undertaking at Buchenwald concentration camp, in which internees were inoculated with an “experimental” typhus vaccine, resulting in over 200 deaths. He gained an early release from his internment, possibly because he assisted the Allies with his knowledge of germ warfare.
Reactive Arthritis

An aseptic, peripheral, idiopathic disease complex preceded by a history of diarrhea or sexual contact followed by:

- Conjunctivitis
- Urethritis
- Polyarthritis
- Mucocutaneous lesions
Reactive Arthritis

Saying:

“Can’t See, Can’t Pee, Can’t Climb a Tree”

- Feet are affected in 84-93% of patients suffering from Reactive Arthritis/Reiter’s Disease
- At presentation the knee & ankle are most commonly affected
Reactive Arthritis

Asymmetrical & Unilateral Joint Involvement with Lower Extremity Predilection
Reactive Arthritis

Clinical features:
- Symptoms are mild and spontaneously regress
- Etiology unknown but attributed to gonococcal infection or chlamydia
- Clinical diagnosis

Two types:
- **Endemic** – venereal/genitourinary. (Males 20-30)
- **Epidemic** – post-dysenteric -Salmonella infection/food poisoning/drinking contaminated water.
  (Women/Children)
Reactive Arthritis

Clinical features (continued):

- Prior history of diarrhea or sexual exposure 3-11 days prior to onset of classic triad:

  - Conjunctivitis
  - Urethritis
  - Polyarthritis

- Develops within a month of initial infection

- Balanitis and keratoderma blennorhagica are also consistent findings

- Triad can occur in any sequence and not all may be manifested
Reactive Arthritis

Diaphyseal Periosteal Reaction of Phalanges
Reactive Arthritis

Calcaneal Periostitis

Retrocalcaneal Bursitis leading to bone erosion
Reactive Arthritis
CONCLUSION

There are many different forms of Arthritis.
The foot and ankle are commonly affected by these disorders.
Many of these Arthritic processes lead to marked pain and destruction of bone.
Some may be associated with other disease processes.
Radiographs are a crucial component in the diagnosis of these arthritic diseases.
THANK YOU!

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Radiographic Evaluation of the Arthritides
Ankylosing Spondylitis
Ankylosing Spondylitis

- Affects articulations, ligaments and tendons of pelvis and spine

**Clinical features**
- Age: 15 – 35 yoa
- Gender: Male 10:1 female
- Often seen in conjunction with Irritable Bowel Disease

**Lab findings**
- HLA-B27 – 90%

**Distribution**
- Low back, pain may be uni- or bi-lateral
- Thoraco-lumbar spine initially
Ankylosing Spondylitis

Bilateral, Symmetrical Joint Involvement Originating at the:

Axial Skeleton
(SI Joint & Spine)

Appendicular Skeleton
(Hips, Shoulders, Knees, Hands & Feet)
Ankylosing Spondylitis
Ankylosing Spondylitis

Shiny Corner Sign:

- Also known as a Romanus Lesion
- Early spinal finding in AS
- Represents small erosions at the superior and inferior endplates (corners on lateral radiograph) of the vertebral bodies, with surrounding reactive sclerosis
- Eventually squaring of the anterior margin of the vertebral body occurs
Ankylosing Spondylitis

Bony Ankylosis
Ankylosing Spondylitis

**Radiographic Features:**

- Non-descript changes in LE
- SI joint – enthesopathy
- Spine – syndesmophytes (bamboo spine)
- Osteopenia
- Erosions
- Reactive sclerosis
- Bony ankylosis
**Ankylosing Spondylitis**

Annulus fibrosus + Ossification = Syndesmophyte

- **Annulus Fibrosus**: ring of ligament fibers that surround/protection the inner core of the disc and connect the vertebrae to one another.
- **Sharpey’s fibers**: outer portion of the annulus fibrosus.
- **Syndesmophytes**: ossification of Sharpey’s fibers, which radiographically appear as thin vertical outgrowth of bone that extends across the margin of the intervertebral disc. Typically form at the anterior and lateral aspects of the spine, particularly near the thoracolumbar junction.
Ankylosing Spondylitis

Bamboo Spine
Syndesmophyte

Osteophyte
Ankylosing Spondylitis

Ossification of the Longitudinal Ligaments Connecting Adjacent Vertebrae Are Called:

SYNDESMOPHYTES
Ankylosing Spondylitis

Normal SI Joint

Serrated “Postage Stamp” Erosions
(typically iliac side first due to thinner cartilage)

Ankylosis of SI Joint with Osteopenia
Ankylosing Spondylitis

Normal SI Joint

Fused SI Joint
Ankylosing Spondylitis
Chondrocalcinosis
Chondrocalcinosis

- **Gout-like symptoms with CPPD crystals**
- **Clinical features**
  - Age: >30 yoa, peak at 6th decade
  - 50% of people older than 85 years have radiographic evidence of **chondrocalcinosis**
  - Gender: Males = females
  - Can simulate acute gout attack- erythema, edema, fever, increased calor at the joint, & pain
- **Lab findings**
  - Elevated ESR
  - Polarizing microscopy – **weak positive** birefringence of the rod/rhomboid shaped crystals
Chondrocalcinosis

Potential sites for calcification in CPPD
Chondrocalcinosis
Chondrocalcinosis

Articular Deposition

Meniscal Deposition
Chondrocalcinosis

**Distribution**
- Knee
- Wrist
- Hand
- Ankle
- Symphysis pubis
- Elbow
Chondrocalcinosis

Pubic Symphysis
CPPD Deposition
Rheumatoid Arthritis

Rheumatoid arthritis (late stage)

- Boutonniere deformity of thumb
- Ulnar deviation of metacarpophalangeal joints
- Swan-neck deformity of fingers

Boutonniere Deformity

Swan-neck Deformity
Rheumatoid Arthritis

Late Radiographic Changes:
- **SWAN-NECK DEFORMITY**
  - Flexion of the DIPJ and hyperextension of the PIPJ
  - Seen in SLE and scleroderma
Rheumatoid Arthritis
Rheumatoid Arthritis

Flexion of the DIPJ

Hyperextension of the PIPJ
Rheumatoid Arthritis

Late Radiographic Changes

– BOUTINNEIRE DEFORMITY

- Flexion of the PIPJ and hyperextension of the DIPJ
- Named because of the appearance of the fingers placing a carnation in a button hole
- Common in RA but also seen in SLE and Jaccoud’s (post-rheumatic fever) arthritis
Rheumatoid Arthritis

Flexion of the PIPJ

Hyperextension of the DIPJ
Rheumatoid Arthritis

Late Radiographic Changes:

- **MAIN EN LORGNETTE DEFORMITY**
  - AKA “Opera Glass Hand”
  - Seen in advanced RA, advanced PA, & erosive osteoarthritis
  - Develops as a result of:
    - Shortening of several proximal phalanges secondary to compressive erosions & destruction of bony ends
    - MCPJ dislocations causing a “telescoping” & retraction of the fingers
Rheumatoid Arthritis

MCPJ Dislocation

Telescoping of the Phalanges
Rheumatoid Arthritis

Dorsal Dislocation of MPJs
Rheumatoid Arthritis

Bony Ankylosis of MPJs
Rheumatoid Arthritis
Rheumatoid Arthritis

Pathological Fractures Secondary To The Osteopenia Not Uncommon
State the diagnosis for each of the following X-rays.
State the diagnosis for each of the following X-rays.

- **GOUT**
- **RA**
- **PA**
State the diagnosis for each of the following X-rays.
State the diagnosis for each of the following X-rays.

- DJD
- GOUT
- RA
What do you think is going on with these two different radiographs?
What do you think is going on with these two different radiographs?

Detritic Synovitis from Silastic Hemi-Implant

Septic Arthritis
Of the arthritides that we have discussed thus far, which one results in the most “bone production mineralization”?
Osteoarthritis (DJD)
Which of the arthritides that we have discussed results in the least “bone production/mineralization”?
Rheumatoid Arthritis
Match the term with its corresponding arthritide(s):

1) Heberden’s Node   A) PA
2) Ivory Phalanx      B) RA
3) Gull Wing Sign    C) OA
4) Romanus Lesion    D) Erosive OA
5) Swan-neck Deformity E) Gout
6) Fluffy Heel Spur  F) AS
7) Martel’s Sign     G) Reiter’s Dz
1C, D
2A
3D
4F
5B
6G
7E
Neuropathic Arthritis
Neuropathic Joint Disease
Neuropathic Joint Disease

Destructive articular disease process associated with conditions that cause sensory & autonomic neuropathy.

Pathophysiology:
- Neurotraumatic Theory (Volkman & Virchow 1886): Bony destruction is attributed to loss of pain sensation & proprioception combined with repetitive, mechanical minor trauma to the foot.
- Neurovascular Theory: Suggests that joint destruction is secondary to an autonomically stimulated vascular reflex that causes hyperemia & periarticular osteopenia.
- Pro-inflammatory Cytokines Theory: Activation of cytokines like tumor necrosis factor alpha & interleukin-1B result in increased expression of RANKL which causes osteoclast maturation and ultimately local osteolysis.
Neuropathic Joint Disease

**Pathophysiology (Biochemical):**

- Physiologic balance between pro- & anti-inflammatory cytokines is lost
- Increase in proinflammatory cytokines like TNF, IL-1B, IL-6
- Prolonged inflammatory response triggers the RANKL pathway
- RANKL pathways increase osteoclast production/activity, which induces bone lysis & destruction
Neuropathic Joint Disease

Lab findings:
- Negative except for etiologic factor
Neuropathic Joint Disease

Clinical features:

- Vary widely depending of the stage of the disease
  - Mild swelling/no deformity to significant swelling/moderate deformity
- Signs of inflammation such as:
  - Profound unilateral swelling
  - Increase in local skin temperature (3-7 degrees greater than the non-affected foot’s skin)
  - Erythema
  - Joint effusion
- Joint instability & crepitation (Loose bag of bones)
- Loss of protective sensation, DTRs, ataxia
- Pain (severity significantly less than what would be expected based on the clinical/radiographic findings)
Neuropathic Joint Disease

Charcot Arthropathy:
- Diabetes is the most common cause
- 1 out of 700 patients with diabetes will develop Charcot joint

<table>
<thead>
<tr>
<th>Causes of Neurotrophic Arthropathy</th>
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<tbody>
<tr>
<td>CONGENITAL</td>
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<tr>
<td>Congenital indifference to pain</td>
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<tr>
<td>Dysautonomia</td>
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<td>Spina bifida vera (meningocele, etc.)</td>
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<td>ACQUIRED</td>
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<tr>
<td>Alcoholism</td>
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<td>Amyloidosi s</td>
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<td>Charcot-Marie-Tooth disease</td>
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<td>Diabetes mellitus</td>
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<td>Leprosy, yaws</td>
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<td>IATROGENIC</td>
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<td>Phenylbutazone</td>
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<td>Steroids</td>
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<td>Multiple sclerosis</td>
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<td>Syringomyelia</td>
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<td>Trauma</td>
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<td>Tumor</td>
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Neuropathic Joint Disease
Neuropathic Joint Disease

Distribution:
- Hypotrophic form (Atrophic) – non-weightbearing joints (forefoot)
- Hypertrophic form – weightbearing joints (Ankle, rearfoot, midfoot)

Radiographic features:
- Atrophic form
  - Usually localized to the forefoot (metatarsal heads/shafts)
  - Bone resorption
    - Osteolysis is more pronounced in atrophic charcot than with psoriatic arthritis
  - “Licked candy stick”
  - Diabetic osteolysis
Neuropathic Joint Disease

“Licked Candy Stick” Appearance (Atrophic Form)
Neuropathic Joint Disease

Osteolysis of the distal metatarsals and phalanges with tapering results in a pencil-like appearance in the late stage of diabetic neuropathy.
Eichenholtz Classification for Hypertrophic Charcot:

- 3 stages

**Destruction/Fragmentation** (Acute Charcot)- characterized by inflammation & joint effusion. Capsular/ligamentous structures become lax and subsequently periarticular fracture/erosions & joint dislocation occurs.

**Coalescence** (Subacute Charcot)- resorption of fine bone debris & coalescence of larger boney fragments. Bone becomes sclerotic and joint stability starts to increase.

**Reconstruction** (Chronic Charcot)- associated with re-stabilization of the foot with continued ankylosis, remodeling/rounding of bone, formation of pseudarthroses, & reduction in sclerosis.
Neuropathic Joint Disease

Fragmentation
Neuropathic Joint Disease

Coalescence

Reconstruction
Neuropathic Joint Disease

Radiographic features:

- **Hypertrophic** form – 6 **D**s (3 “De-” & 3 “Dis-”)
  - Destruction
  - Density increases
  - Debris production
  - Dislocation
  - Disorganization
  - Distension of joint
Neuropathic Joint Disease

Increased Density & Debris (Hypertrophic Form)
Neuropathic Joint Disease

Fragmentation & Destruction With Talar Dislocation (Hypertrophic Form)
Neuropathic Joint Disease

Disorganization of the ankle & subtalar joints (Hypertrophic Form)
Neuropathic Joint Disease
Miscellaneous Arthritides
Diffuse Idiopathic Skeletal Hyperostosis
Overview:

- Ligamentous/Tendinous ossification and calcification at their respective attachment sites to bone (entheses)
- Also known as a diffuse variant of Forestier’s Disease (OALL: ossification of the anterior longitudinal ligament) or senile ankylosing spondylitis
- Spinal & extra-spinal articulations
- 12% middle aged and elderly
Diffuse Idiopathic Skeletal Hyperostosis (DISH)

Clinical features:

- Age: >50 yoa (AS 15-35 y/o)
- Gender: males predominant (like AS)
- 20% have concurrent diabetes; dyslipidemia & hyperuricemia also commonly occur simultaneously
- Obesity (high BMI or large waist circumference) is a common feature of many patients with DISH
- May exhibit postural abnormalities & limitation in spinal mobility similar to AS
- Dysphagia is common because calcification of the anterior longitudinal ligament & extraspinal ligaments, may impinge on esophageal space
Diffuse Idiopathic Skeletal Hyperostosis (DISH)

- Typically affects the lower thoracic spine, but the cervical & lumbar spine might also be involved.
- Left side of the spine is usually spared.
  - Due to pulsating aorta.
  - Finding is supported by the fact that individuals with DISH & situs inversus (organs on reverse side of body) have sparing of the right side of the spine b/c the aorta is located on the right side.
Diffuse Idiopathic Skeletal Hyperostosis (DISH)

**Lab findings:**
- HLA-B8 antigen 40% (AS positive for HLA-B27)

**Distribution:**
- Cervical, thoracic and lumbar spine

**Radiographic features:**
- Spinal
  - Flowing hyperostoses over 4 contiguous vertebral bodies
  - Anterior longitudinal ligament (PLL in 50% - cervical)
  - Preservation of normal disk height with absence of vertebral body squaring/shiny corner sign (differentiates DISH from AS)
  - Absence of bony ankylosis of facet joints (differentiates DISH from AS)
  - Absence of sacroiliac erosion, sclerosis, or bony fusion, although narrowing and sclerosis of facet joints are acceptable (differentiates DISH from AS)
Diffuse Idiopathic Skeletal Hyperostosis (DISH)

- DISH is characterized by ‘flowing’ ossification of the anterior longitudinal ligament.
- Ossification is separated from the anterior aspect of the vertebral body by a thin radiolucent line.
- Anterior ossifications often meet without fusion, thereby allowing continued motion of the spine.
Diffuse Idiopathic Skeletal Hyperostosis (DISH)

Lateral radiograph of the thoracic spine shows flowing ossification of the anterior longitudinal ligament of several thoracic vertebral bodies. The disc spaces and vertebral body heights are normal.
Diffuse Idiopathic Skeletal Hyperostosis (DISH)

Radiographic features (continued):

- Extra-spinal
  - Can occur at any tendinous or ligamentous insertion
  - Roughening at bony attachment (whiskering)
  - Ossification of ligament or tendon
  - Normal joint space preserved
  - Hypertrophy/enlarged sesamoids
  - Hypertrophy/broadening of the distal phalangeal tufts (arrowheading)
Diffuse Idiopathic Skeletal Hyperostosis

Enthesopathy at the iliolumbar ligament insertions (black arrow), anterior superior iliac spines (white arrow), and hamstring tendon (yellow arrow) attachments bilaterally.

Note that the Sacro-iliac joint is spared.
Diffuse Idiopathic Skeletal Hyperostosis (DISH)

Exuberant ossification at sites of tendon, ligamentous, or joint capsule insertion (entheses) is strongly suggestive of the diagnosis.
Diffuse Idiopathic Skeletal Hyperostosis (DISH)

Hypertrophic new bone formation at the metacarpals & distal phalangeal tufts.
Pulmonary Hypertrophic Osteoarthropathy
Pulmonary Hypertrophic Osteoarthropathy (PHO)

**Overview:**

- A clinical syndrome caused by intrathoracic neoplasm or infection (i.e. bronchogenic carcinoma) that produces the following triad of symptoms:
  - Digital clubbing
  - Symmetrical arthritis
  - Linear periostitis
(PHO)

- AKA:
  - Hypertrophic Osteoarthropathy (HOA)
  - Primary Hypertrophic Osteoarthropathy (PHO)
  - Pachydermoperiostosis

“Pulmonary” dropped because the clinical findings may occur in several non-pulmonary diseases & even may occur without any underlying illness
PHO

Clinical features:
- Age: 40 – 60 yoa
- Gender: males predominant

Lab features:
- Cause dependent
- Elevated ESR

Distribution:
- Upper and lower extremities
  - Changes are most commonly observed in the tibia, radius, ulna, fibula, and femur
PHO

**Location**
- Radius & Ulna
- Tibia & Fibula
- Proximal Phalanges
- Femur
- Metacarpals & metatarsals
- Humerus & Distal Phalanges
- Pelvis

**Most Common**
- Bilateral/symmetric involvement

**Least Common**
PHO

Radiographic/Clinical features:

- Joint effusion

No specific joint disease
Radiographic/Clinical features:

- Digital clubbing

  - Bulbous enlargement of finger tips and rounding of nail plate
  - Clubbed portions consist of excessive collagen fiber deposition, accumulation of interstitial edema, & vascular hyperplasia between the nail matrix and the distal phalanx
  - AKA: Hippocratic finger; Watchglass nails, Drumstick fingers
  - Considered the oldest sign in clinical medicine-2500 yrs ago Hippocrates noted this finding on a patient suffering from empyema
Pulmonary Hypertrophic Osteoarthropathy (PHO)
Radiographic features:

- Periostitis of long bones
  
  "Double stripe sign" on bone scans

  - Symmetric diffusely increased uptake along cortical margins of diaphysis and metaphysis of tubular bones

Metaphyseal and diaphyseal regions typically involvement

  - Typically begins at the diaphysis of long bones & then extends into the metaphyseal region

  - Single periosteal proliferation: appears as a continuous thin line of sclerotic new bone separated from the cortex by a radiolucent space

  - Laminated periosteal proliferation: looks like onion skin in that it exhibits multiple layers/strips of new bone that eventually thicken and fuse with the cortex
PHO
PHO

- Periosteal new bone formation

Abnormal long bone uptake of radiotracer

Anterior and posterior views
Linear Periostitis:
Subperiosteal edema elevates the periosteum & osteoid matrix is deposited beneath it. As this mineralizes, a new layer of bone is formed, and eventually the distal long bones may become sheathed with a cuff of new bone.
Pulmonary Hypertrophic Osteoarthropathy (PHO)

Linear Periostitis
Pulmonary Hypertrophic Osteoarthropathy (PHO)

Bronchogenic Carcinoma:
Begins as a small focus of atypical epithelial cells within the bronchial mucosa that grows and spreads to regional lymph nodes & organs like the liver, brain, and bone.
Pigmented Villonodular Synovitis
Pigmented Villonodular Synovitis (PVNS)

**Overview:**
- Localized, invasive nodular mass arising from synovial lining, tendon sheath, ligaments, or bursae
  - Locally destructive
  - Does not metastasize

**Clinical features:**
- Age: young adults
- Gender: males predominant
- Typically monoarticular arthritis in nature affecting the knee, hip, ankle or hand
- Decreased ROM/locking/catching of the involved joint
- Insidious progression with acute episodic attacks of pain & swelling
- Synovial fluid is typically hemorrhagic & dark brown in color
Pigmented Villonodular Synovitis

Radiographic features:
- Soft-tissue mass with bony erosions/lysis
- Pressure erosions well-demarcated with sclerotic borders (saucerization)
- Calcification unusual
- Typically monoarticular, but when polyarticular it will affect the midfoot due to its unique synovial compartmentalization
Pigmented Villonodular Synovitis

Monoarticular & Asymmetric Involvement
Pigmented Villonodular Synovitis

Extrinsic bone erosions in elbow
Pigmented Villonodular Synovitis

The hyperplastic synovium invades the subchondral bone & produces cysts and erosions.
The nodular mass may have many synovial protrusions that affect joints, bursae, and tendon sheaths. It has a typical yellow-brown appearance due to excessive deposits of hemosiderin, fibrous stroma & lipids.
Pigmented Villonodular Synovitis
Arthritides associated with Collagen Vascular Diseases
Systemic Lupus Erythematosus
Systemic Lupus Erythematosus

Overview:
– Autoimmune, connective tissue disorder characterized by inflammation & vasculitis with multiple organ system involvement

Clinical features:
– Gender: Females (13:1 ratio)
– Age: 20 – 40 yoa

Lab findings:
– Elevated ESR and ANA
Systemic Lupus Erythematosus

Radiographic features:
- Most prominent x-ray features in hands
- Juxta-articular osteopenia & soft tissue edema
- Affects the small joints of the hands, wrists, knees, and shoulder
- Polyarthritis (80%):
  - Symmetric
  - Non-erosive
  - Non-deforming
Systemic Lupus Erythematosus

Radiographic features:

- **Reversible** subluxations, dislocations, deformities are due to ligamentous instability & laxity of the supporting structures

- Calcification & atrophy
  - Muscle atrophy & contractures cause the reversible deformities to become fixed/rigid

- 10% will have irreversible deformities:
  - Ulnar drift at the metacarpophalangeal joints
  - Swan neck deformity (like RA)
  - Boutonniere deformity (like RA)
Systemic Lupus Erythematosus
Systemic Lupus Erythematosus

Swan-neck and boutonniere deformities of the hand

Reversible when placed against x-ray cassette
Systemic Lupus Erythematosus

AVN attributable to SLE and/or steroid therapy. 5-50% of SLE patients develop osteonecrosis of weight-bearing joints. Note the subchondral fractures, density changes, & joint surface disruption of the femoral head.
Scleroderma
Scleroderma

Overview:
- Systemic inflammatory connective tissue disorder affecting skin, lungs, GI, heart, kidneys and musculoskeletal system

Clinical features:
- Females 30 – 60 yoa
- CREST (Calcinosis Cutis; Raynaud’s Phenomenon; Esophageal Dysmotility; Sclerodactyly; Telangiectasia)
Scleroderma

Radiographic features:

- **Hands** most commonly involved
- Soft-tissue atrophy & calcification
- Arthritis is initial symptom in $2/3$ of patients, often preceding skin changes
  - Resembles RA but is less destructive/erosive
  - Symmetric joint space narrowing with marginal erosions (9-15%)
  - Acro-osteolysis
  - Juxta-articular osteopenia
  - Contractures are generally dermatogenic induced due to sclerotic changes of the overlying skin or surrounding connective tissue
Scleroderma

Calcinosis cutis
Scleroderma

Acro-osteolysis
Scleroderma

Raynaud’s Phenomenon:

Triphasic color changes of pallor, cyanosis, and erythema (white/blue/red) represent phases of vasoconstriction, slow blood flow, and reperfusion.

Color changes extend proximally from the tips of digits to various levels, with a well-demarcated border.
Scleroderma

Sclerodactyly:
Initial stage of the disease involves swelling of the fingers. Later, as the connective tissue becomes fibrotic, while skin on the fingers and toes becomes hard & shiny. The fingers can become difficult to bend and can form contractures due to the severe tightening of the skin.
Scleroderma

Telangiectasias

Dilation of small vessels and capillaries cause flat red marks to appear on the skin.
In Summary:

- Remember your 6 broad arthritide categories.
- It is important to differentiate degenerative from inflammatory causes of joint space narrowing.
- **Degenerative arthritis** is characterized by subchondral cysts, osteophytosis, subchondral sclerosis, & asymmetric joint space narrowing.
- **Inflammatory arthritis** is characterized by bone erosions, osteopenia, soft-tissue swelling, & uniform joint space narrowing.
- Inflammation that involves multiple joints in a proximal distribution of the hands or feet **without** bone proliferation suggests Rheumatoid Arthritis.
- Inflammation that involves multiples joints in a distal distribution of the hands or feet **with** bone proliferation suggests a Seronegative Spondyloarthropathy.
The End