Radiologic Manifestations of Avascular Necrosis in Adults

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Our Patient

38 year old female with a history of avascular necrosis of the left hip. She has had a left total hip arthroplasty and a renal transplant and presents with worsening right hip pain for many weeks.
What is Avascular Necrosis?

• Also called: avascular necrosis, aseptic necrosis, osteonecrosis and ischemic necrosis
• Etiology is not completely understood
• Generally accepted as compromise of bone vasculature with subsequent death of bone and marrow cells
• Often leads to mechanical failure and joint destruction
• An estimated 10,000 to 20,000 new cases are diagnosed each year in U.S.
• 10% of all total hip replacements
• Male to female ratio, 5-8:1
• Average age of onset is between 30 - 40
Etiologies and Associations

- **Causes can be classified into traumatic and non-traumatic**

- **Traumatic**
  - Fracture, dislocation or fracture-dislocation
  - Minor blunt trauma

- **Non-traumatic**
  - Corticosteroids (iatrogenic/hypersecretion)
  - Pancreatitis
  - Systemic Lupus Erythematosis
  - Alcohol use
  - Intravascular coagulation
  - Sickle cell disease
  - Caisson disease
  - Chronic renal failure
  - Tumors (lymphoma, leukemia)
  - Arteritis
  - Radiation
  - Gaucher’s disease
  - Thrombophlibitis
  - Smoking
  - Organ transplantation
  - Hyperuricemia (gout)
  - Hyperlipidemia
  - Pregnancy
  - Infection/idiopathic
  - Embolism
Etiologies and Associations

- **Mnemonic: PLASTIC RAGS**
  - Pancreatits/pregnancy
  - Lupus
  - Alcohol
  - Steroids
  - Trauma
  - Idiopathic/Infection
  - Caisson disease
  - Radiation
  - Arteritis
  - Gaucher’s disease
  - Sickle cell disease

- 60-90% of cases are reportedly due to corticosteroid or alcohol
- Hyperlipidemia demonstrated in 80% with idiopathic AVN
- Children:
  - Legg-Calvé-Perthes Disease - AVN of the femoral head leading to impaired epiphyseal growth
  - Panner’s Disease - AVN of the capitellum of the humerus
Clinical Manifestations

• Insidious onset
• Weight-bearing and motion-induced pain
• Rest pain occurs in 2/3 of patients, night pain in approximately 1/3 of patients
• Patients may have pain and limited ROM
• Most commonly in epiphyseal cavities
  – Femoral head (anterolateral), femoral condyles (medial), humeral head
  – Epiphyseal ends of long bones covered by articular cartilage - limited arterial access/venous outflow
• Body of the talus, carpal scaphoid and lunate are less commonly involved
Pathogenesis

• Underlying cause: **vascular compromise** of sinusoids of the trabecular bone leading to necrosis

• Current theorized mechanisms:
  – Mechanical disruption of arterial supply
  – Embolism
  – Elevated intraosseous pressure/encroachment
  – Vasculitis/vessel injury
  – Venous obstruction
Pathogenesis: Mechanical

- Disruption of vasculature to bone with limited or no collateral circulation leads to necrosis
- Most commonly occurs in the femoral neck
  - Intracapsular fractures
  - Dislocations
  - Impaction
- AVN can occur in as little as 8 hours after insult
Pathogenesis: Embolism

- Poor collateral flow makes a bone vulnerable to embolic events
- Believed to be the mechanism behind AVN associated with:
  - Sickle cell disease/hemoglobinopathies
  - Caisson disease (dysbaric) - nitrogen emboli
  - Fat embolism (pancreatitis, alcoholism)
Pathogenesis: Encroachment

- An elevation in intramedullary pressure may cause external compression of intraosseous vessels
  - Gaucher’s disease: abnormal accumulation of sphingolipids within macrophages due to β-glucocerebrosidase deficiency compressing sinusoids
  - Intraosseous hemorrhage (hemophilia)
  - Tumor
  - Steroids: has been associated with increased fat cells leading to vessel compression - 2-4% to more than 25% incidence
Pathogenesis: Vessel Injury

- Injury to the vessels themselves can cause occlusion and the development of AVN
  - Radiation
  - Systemic lupus erythematosus - 5-6% incidence
  - Vasculitis
Pathogenesis: Venous Obstruction

- Back pressure from venous obstruction may exceed arterial perfusion pressure resulting in ischemia
  - Pregnancy: mass effect from gravid uterus can impair venous drainage
  - Joint effusions
  - Steroids
Pathogenesis

• Model: femoral head
• AVN usually develops in fatty marrow - less vascular
  – Epiphysis is predominantly fatty marrow
• Collateral vascular supply of femoral head is relatively limited
• After the initial insult, marrow elements and cortex die
  – Hematopoietic cells most susceptible (6-12 hours)
  – Osteocytes, osteoblasts, osteoclasts (12-48 hours)
  – Fat cells (2-5 days)
• Inflammatory response to necrosis (weeks to months)
  – Cuff of hyperemia, inflammatory cell infiltrate and fibrosis (granulation tissue) surrounds necrosis - “reactive interface”
  – Results in varying degrees of hypoxic injury
Pathogenesis

• Inflammatory cuff leads to reactive revascularization
  – Osteoclastic resorption of necrotic bone and osteoblastic bone deposition

• Resorption leads to loss of normal structural integrity leading to **microfractures** of the subchondral trabeculae

• Physiologic stress fractures *cannot be repaired*

• **Articular collapse** occurs due to subchondral weakness

• Ultimately **degenerative joint disease** due to articular disruption

• Process does **NOT affect articular cartilage**
  – Derives nutrients from synovial fluid

• Commonly bilateral at time of diagnosis
Pathogenesis

- (V) viable bone
- (R) reactive interface
- (I) ischemic zone
- (N) necrotic zone

Pathogenesis

- Intramedullary necrosis (bone infarct) of metadiaphyseal marrow cavity often considered separate entity
  - Sickle cell disease
  - Gaucher disease
Pathogenesis

- Geometry of convex surfaces of joints (e.g. femoral head) leads to increased incidence of AVN
  - Shape leads to increased intramedullary pressure on convex side
  - Greater chance of occluding vasculature

http://www.rad.washington.edu/mskbook/osteonecrosis.html
Staging

• Modified Ficat and Arlet Staging System

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<tr>
<th>STAGING OF OSTEONECROSIS OF THE FEMORAL HEAD</th>
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• Typical progression begins with subtle sclerosis of bone
• At femoral head, this is followed by a characteristic subchondral lucency (the “crescent sign”)
• Collapse of the bone (flattening of the femoral head)
• Finally by narrowed joint space and osteoarthritic changes in the opposing bones of the joint
Imaging Modalities

- **Radiograph**: first line
  - Fast, inexpensive but *not sensitive*
  - AP, frog-leg lateral

- **Scintigraphy (bone scan)**: useful in the early diagnosis
  - Technetium$^{99m}$-methylene diphosphonate shows decreased/absent activity in early disease (stage I) surrounded by increased activity
  - **High sensitivity but low specificity**
    - Infection, myeloma, metastases, hemangioma, radiation
  - Eventually becomes a “hot lesion” due to revascularization in later stages
    - Transition can lead to false-negatives (6-10%)

- **CT**: generally not used in the imaging of AVN
  - Less sensitive than both scintigraphy and MR
Imaging Modalities

• **MR:** primary test for evaluation of AVN, especially when radiographs are equivocal
  - **Sensitivity approaches 100%**
    - Can detect early lesions, as young as 2 weeks
  - Multiplanar, able to visualize effusions, edema and cartilage better, no ionizing radiation
  - Classic pathognomonic “**double-line**” sign on T2W images
    - High signal intensity rim inside a low signal margin surrounding the osteonecrotic lesion
    - Corresponds to sclerotic bone (low signal) around granulation tissue (high signal)
    - Often “serpiginous” appearance
Imaging Modalities

- MR classification system based on appearance of central lesion

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<th>MR CLASSIFICATION OF OSTEONECROSIS</th>
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- General chronologic progression from stage A to stage D
- T1W, STIR commonly used today but sequences vary from site to site
  - Coronal and axial
- Role of Gd enhancement still under investigation
  - May increase sensitivity
  - May help differentiate TBME from AVN
Imaging Modalities

- Normal hip radiograph
Imaging Modalities

- Normal coronal T1W
Imaging Modalities

• Normal axial T1W
Imaging Modalities

- Normal coronal STIR
Imaging Modalities

• Normal axial STIR
Radiological Differential Diagnosis

- Transient osteoporosis of the hip - uncommon, idiopathic, self-limited condition
  - Appears similar to AVN on MR (edema, local osteopenia) but resolves in 6-12 months
  - Common in pregnant women and middle-aged men

- Transient bone marrow edema syndrome - diffuse edema in the absence of osteopenia
  - Possibly earliest manifestation of AVN
  - Does not demonstrate double-line sign on T2W

- Osteochondritis dissecans - Believed to be distinct
  - Fragmentation/separation of part of articular surface
  - Typically presents in childhood/adolescence, more common in boys

- Subchondral cysts, osteoarthritis
Back to Our Patient

- 38 yo female with h/o AVN is s/p L-THA and renal transplant with worsening R hip pain for many weeks

No definite abnormality
Our Patient

- Stage 0/I disease based on normal radiograph and abnormal MR

Coronal T1W

Focal low signal intensity regions consistent with edema or fibrosis
Our Patient

• Stage 0/I disease based on normal radiograph and abnormal MR

Coronal STIR

Corresponding focal high signal intensity regions consistent with edema or granulation tissue
Our Patient

• Compare T1W images with STIR and normal
AVN of the Femoral Head

- Blood supply to femoral head is largely through medial circumflex femoral artery
- Small contribution by artery of the ligamentum teres
- Inadequate to supply femoral head in the absence of MCFA supply
- Up to 25% of dislocations, especially if complicated by fracture (75%)

Radiological Stages of AVN

- 43 yo male with hip pain and questionable history of alcoholism

Region of subchondral lucency

Stage II disease

Courtesy of Ferris Hall, MD
Radiological Stages of AVN

- 57 yo female with h/o sickle cell disease

Crescent line: subchondral fracture in absence of normal microfracture repair

Stage III disease
Radiological Stages of AVN

- 30 yo male with h/o sarcoid and steroid therapy

Segmental flattening

Stage IV disease
Radiological Stages of AVN

- 31 yo male with h/o renal failure, s/p pelvic radiation

More severe cystic and sclerotic changes with flattening and evidence of fracture
AVN of the Femoral Head

• 37 yo male with h/o HIV and steroid therapy

Minimal abnormalities
AVN of the Femoral Head

- 37 yo male with h/o HIV and steroid therapy

- Classically, “double line” sign seen on T2W images
- Geographic appearance on MR is an early finding

Courtesy of Ferris Hall, MD
Patient #2

- 72 yo female with h/o ovarian cancer, s/p pelvic radiation, p/w hip pain
AVN of the Femoral Head

- Bone scan of this patient was consistent with at least stage II disease

Bilateral increased Tc\textsuperscript{99m} uptake
AVN of the Femoral Head

- Early (stage I) bone scans reveal a photopenic region representing necrosis surrounded by increased uptake representing hyperemia and reactive bone formation.

Patient #3

- 37 yo male with h/o HIV p/w shoulder pain

Irregular subchondral lucencies
Patient #3

- 37 yo male with h/o HIV p/w shoulder pain

Coronal T1W

Crescentric region consistent with segmental fractures and edema
AVN of the Humeral Head

- Main blood supply: anterior circumflex humeral artery
- The arcuate artery arises from ascending branch of anterior humeral circumflex artery as it penetrates bone
  - gives branches to lesser and greater tuberosities and perfuses the entire epiphysis of the humeral head
- The posterior circumflex artery supplies only a small area in posteroinferior aspect of the humeral head
- Fracture or occlusion of the arcuate artery can lead to AVN
AVN of the Humeral Head

AVN of the Humeral Head

- 56 yo female with h/o SLE and steroid therapy

Subchondral lucency consistent with AVN

Courtesy of Ferris Hall, MD
Patient #4

- 47 yo male with h/o wrist trauma

Disuse osteopenia of “normal” bone from pain of injury

Avascular proximal pole with normal appearing calcification

Courtesy of Ferris Hall, MD
AVN of the Scaphoid

• Usually secondary to trauma and fracture
• Also known as Preiser’s Disease when idiopathic.
AVN of the Scaphoid

- 15% of scaphoid fractures
- Radiologic evidence does not develop for 2-4 weeks

Patient #5

- 31 yo male with wrist pain and instability

Coronal T1W

Low signal intensity focus consistent with edema or fibrosis
Patient #5

- 31 yo male with wrist pain and instability

Coronal STIR

High signal intensity focus consistent with edema
Patient #5

- 31 yo male with wrist pain and instability

Sagittal T1W

Low signal intensity focus
Kienbock’s Disease

• Believed to result from interruption of blood supply to the lunate bone with subsequent avascular necrosis
• Commonly observed in manual laborers between 20-40
• Risk factors
  – The ulna is shorter than the radius
    • Accentuated stresses across wrist leading to ligamentous disruption
  – Inadequate or vulnerable vasculature
• May be bilateral
• Staged from grade I to grade IV depending upon the severity of the problem
• May progress to collapse and fragmentation
Patient #6

- 36 yo female with h/o ulcerative colitis and steroid therapy, p/w knee pain

Subtle lytic lesion in medial condyle

Courtesy of Ferris Hall, MD
Patient #6

- 36 yo female with h/o ulcerative colitis and steroid therapy, p/w knee pain

Sagittal T1W

- Low signal rim consistent with edema or fibrosis
- Low signal core consistent with edema or fibrosis
Patient #6

- 36 yo female with h/o ulcerative colitis and steroid therapy, p/w knee pain

Sagittal STIR

High signal rim consistent with edema

Low signal core consistent with fibrosis
AVN of the Distal Femur

• Commonly presents in females over 60 years of age
• Slight flattening of femoral condyle with decreased signal intensity on T1W and increased signal intensity on T2W
• Occurs more commonly in the medial condyle, presumably due to increased weight bearing
AVN of the Distal Femur

- Classically, “double line” sign seen on T2W images.
- Sclerotic bone around granulation tissue (low intensity around high intensity)
Patient #7

- 31 yo male with h/o ankle fracture-dislocation, s/p ORIF

Disuse osteopenia of head of talus and remaining foot

Fracture

Sclerotic body of talus

Courtesy of Ferris Hall, MD
Patient #7

- 31 yo male with h/o ankle fracture-dislocation, s/p ORIF
AVN of the Talus

Fracture of talar neck

Usual cause is impact on anterior margin of tibia due to forceful dorsiflexion

AVN of the Talus

- Radiologic evidence of AVN may take up to 3 months
- More commonly occurs at talar body (as seen in this patient)
- **Hawkins sign** - subchondral radiolucent band in proximal talus - sign of intact vascular supply after fracture

Summary

• AVN can be post-traumatic or due to a variety of systemic diseases (or idiopathic)
  – Mechanical, embolic, intraosseous pressure, venous, vasculitis

• Bone infarct (metadiaphyseal) often associated with systemic disease (Gaucher, sickle cell)

• AVN (epiphyseal) associated factors:
  – Fragile vascular supply (e.g. scaphoid, talus, lunate)
  – Joint convexity (e.g. femoral head)
  – Trauma (e.g. femoral neck)
  – Systemic disease/iatrogenic

• Radiograph, scintigraphy, MR studies used to diagnose
  – AP, frog-leg lateral radiograph - first line
    • Crescent sign, articular flattening, focal sclerosis/lucency
  – Scintigraphy sensitive but not specific
    • Low uptake surrounded by high uptake early; hot lesion late
  – MR is highly sensitive and very specific
    • “Double line” sign, serpiginous geographic appearance
References

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