Avascular Necrosis of the Foot

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Avascular Necrosis: Pathophysiology

• Ischemia to the bone from oxygen depletion and resultant cell death.
• Repair: revascularization, resorption and reossification.
• Subchondral fractures at the margin of the repair interface.
• Bone collapse and related joint collapse and osteoarthritis.
Commonly Affected Bones of the Foot

• Talus
• Navicular
• 1\textsuperscript{st} and 2\textsuperscript{nd} Metatarsals
• 1\textsuperscript{st} Metatarsal Sesamoids
Talus

• Why is it prone to AVN?
• Major weight bearing bone of the foot. 60-80% of its surface is articular.

Fig. 485. – The left talus. Lateral aspect. http://www.prohealthsys.com/anatomy/grays/osteo/tarsals.php
Vascularity of the Talus

• Artery to the sinus tarsi from:
  - the peroneal and anterior tibial arteries,
  - the deltoid branch from the posterior tibial artery,
  - the artery of the tarsal canal from the posterior tibial artery.

• Body of talus: Chief supply is from artery of the tarsal canal.
Plain Radiographic Appearance of AVN: Talus
CT: Ischemia Versus AVN: Talus

• Look for increased density.

CT of AVN: Talus

- Inflammatory bowel disease on prednisolone

MRI of AVN: Talus

- CT; T1, STIR, Gadolinium T1 fat sat

Pitfalls: Hawkins Sign

- Seen 6-8 weeks after injury. Thin subchondral area of radiolucency involving the entire talar dome and lateral talar gutter. Signifies talar viability and excludes future development of AVN.
Pitfalls: Look at Subtalar Joint

- Subchondral collapse can occur at the subtalar aspect.

Kohler’s Disease: AVN of the Navicular

- Flattening, sclerosis and irregular rarefaction.
Kohler’s Disease: 1908

• Self limiting AVN of the navicular.
• Age: 4-7 years, more common in boys.
• Etiology uncertain: Temporary ischemia or ossification (Last bone in the foot to ossify). Repetitive compressive forces.
• Pain, swelling and a limp. Bilateral in 1/3rds, may be asymptomatic in the other foot.
• Bone resumes normal appearance in about 2 years with conservative management. Rest and 6-8 weeks cast.
Freiberg’s Disease: AVN of the 2nd Metatarsal Head: 1914
Why 2\textsuperscript{nd} Metatarsal?

- Longest metatarsal. Can affect third too.
- Etiology uncertain. Prone to greater compressive forces. Seen more in teenage girls, (f:m: 5:1) dancers and runners- repetitive stress, trauma, improper shoes.
- 13-18 years; usually unilateral.
- Affects vascularity of the epiphysis with articular cartilage loss, reduced vascularity to subchondral bone, subchondral fracture, collapse and fragmentation.
- Pain, swelling and a palpable lump. Unilateral in 90%.
- ACR appropriateness criteria: Plain radiographs- sclerosis, flattening, widening and sclerosis of metatarsal head and arthrosis.
- MRI in early phase or CT.
- Management is either conservative or grafts, prosthesis or arthrodesis.
MRI Appearance

- MRI: Well before plain radiographs show abnormality: diffuse marrow edema, low T1 serpiginous signal, osteochondral fragmentation.
MR Appearances

- T2 STIR and T1.
Vascular Disruption of 1st Metatarsal Head

- **Vascularity of the metatarsal heads:** Dorsal metatarsal arteries, arise from the dorsalis pedis artery, and the plantar metatarsal arteries, branches of the posterior tibial artery. Anastomose forming an arterial network around the metatarsal heads with nutrient arteries traversing the metaphyseal cortex to supply the subchondral bone.

- **Surgical procedures** such as metatarsal head osteotomies, in which extensive capsular stripping may result in damage to the medial and lateral head vessels, trauma, metatarsal shaft fractures, or vasculopathy may also represent potential causes for disruption of the tenuous blood supply to the metatarsal heads.

- **Freiberg’s infraction** and metatarsal head subchondral fractures occurring in adults have the same pathogenesis.

- **Primary lesion may initiate as a subchondral fissure** most commonly involving the dorsal aspect of the metatarsal head. This fissure may lead to disruption of the epiphyseal vascular supply, evolving to ischemic bone necrosis with subsequent repair or collapse.

- **Five stages of anatomic changes** have been described, with progression or consolidation at any stage. No sequelae when consolidation takes place at an early stage, whereas flattening or arthrosis may be seen when consolidation occurs at later stages.
Complications

• Premature closure of growth plates.
• Loose bodies.
• Secondary osteoarthritis.
AVN of Other Metatarsals

• Following proximal osteotomy of the 1\textsuperscript{st} metatarsal for hallux valgus:

• Other metatarsal heads also affected by AVN, 3\textsuperscript{rd}, 4\textsuperscript{th} and rarely the fifth: Trauma is the initiating factor.
Avascular Necrosis of the Sesamoids of the 1st Metatarsal

• Originally described by Axel Reander in 1924.
• Sesamoid of the flexor hallucis brevis. Medial is bi or multipartite in 10-33 %, larger than the lateral. Ossification occurs in the 8th year.
• Vascularity is through 1, 2 or 3 vessels and is reduced in the tibial sesamoid in women.
• Cause not fully understood. Chronic micro trauma which disturbs the blood flow to a bone leading to necrosis.

Role of 1st Metatarsal Sesamoids

• Role of the sesamoids:
• Absorbing weight-bearing pressure, reducing friction, and protecting tendons.
• The functional complexity and anatomic location of these small bones make them vulnerable to injury from shear and loading forces.
• Injury to the hallucal sesamoids can cause incapacitating pain, which can be devastating to an athlete.
• Often overlooked.
Avascular Necrosis of Sesamoid: Plain Radiographs: Fragmentation, Demineralization and Stippled Sclerosis.

Taren Cardona. Surgical Excision of Painful Fibular Sesamoid. The Foot and Ankle Online Journal, ISSN 1941-6806
CT of Medial Sesamoid: Stress Fracture Versus AVN
Pitfalls: Bipartite/ fracture/ AVN
MRI: Avascular Necrosis of Sesamoid

- To distinguish AVN from stress fractures.
- AVN: Low T1 and T2 signal.
- Stress fractures: Bone marrow edema on T2 without the low signal.
- Sesamoiditis, stress fractures are in fact AVN.
Avascular Necrosis of Sesamoid

• History of point pain, repetitive chronic stress, rarely a result of acute trauma.
• MRI in the early stages: Low T1 and T2 signal indicating ischemia. Later fragmentation and collapse.
• Treated conservatively for 6 months: activity modification, splinting, orthotics, NSAID’s. Bone grafting and partial or complete excision.
Overview of AVN of the Foot

• Atraumatic avascular necrosis (AVN) is an unusual pathology to the foot.
• Risk factors include the use of corticosteroids, smoking, alcohol, rheumatologic disorders, hematologic disorders, and metabolic disorders.
• Opposite side for comparison. Look for multifocal AVN if there are predisposing causes.
• MRI if plain radiographs are negative.
Thank you