HIV-Associated Avascular Necrosis

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Disclaimer

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A Case

- 62F with well-controlled HIV (CD4 1047/41%, VLUD) on TLD presents to establish care after moving to this area.
- Diagnosed 10 years ago on routine screening, started on EFV/TDF/FTC and then transitioned to DTG/TDF/3TC some years ago
- Notes that she has had chronic L hip pain for several years, which developed after a fall, but previously had been active and working as a caregiver
- Has been gradually worsening, now to an unbearable degree
- In the past week, she has been unable to ambulate ☹

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Imaging

- **XR Pelvis:** There is deformity and sclerosis of the left femoral head. Findings may reflect avascular necrosis of head due to prior fracture deformity vs sequela of septic joint, or may be from superimposed overhanging osteophytes.
- **CT Pelvis:** Severe left hip osteoarthritis. Left femoral head collapse secondary to avascular necrosis.

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Learning Objectives

- Review epidemiology of HIV-associated avascular necrosis.
- Understand the potential mechanisms for increased risk in PWH.
- Recognize how to diagnose HIV-associated avascular necrosis.
- Discuss management options.

Background

- First described in PWH in 1990 via case reports
- Now recognized to be far more common
  - Yearly incidence of symptomatic osteonecrosis:[1]
    - General population: 0.003 to 0.006 cases per 100 person-years
    - PWH: 0.03 to 0.37 cases per 100 person-years
  - 2001 natural history study at NIH showed 4.4% prevalence in asymptomatic PWH[1]
  - 2017 EuroSIDA multinational prospective cohort study showed 0.6% prevalence[2]
  - 2025 retrospective study in S.Africa showed that of 1400 patients s/p elective primary THA at a tertiary care center (2015-2021), 11% were for HIV-associated AVN[3]
- Occurs earlier in PWH
- Less commonly responsive to conservative measures in PWH

1 - Morse CG et al, CID 2007; 2 - Borges AH et al, CID 2017; 3 - Geldenhuys DB et al, SICOT-J 2025

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A Refresher on AVN

- Also called osteonecrosis, aseptic necrosis, ischemic necrosis
- Bone death occurs due to interrupted blood supply
- Mechanism still poorly understood and controversial
  - Genetic predisposition? Metabolic factors? Mechanical stresses?
  - Interrupted blood supply → Adjacent hyperemia to attempt repair → Reactive angiogenesis → Impairment of angiogenesis leads to demineralization, trabecular thinning, collapse
- Can be precipitated by direct damage to bone vasculature (e.g. femoral neck fracture) or marrow (e.g. radiation)
- Other less direct mechanisms poorly understood

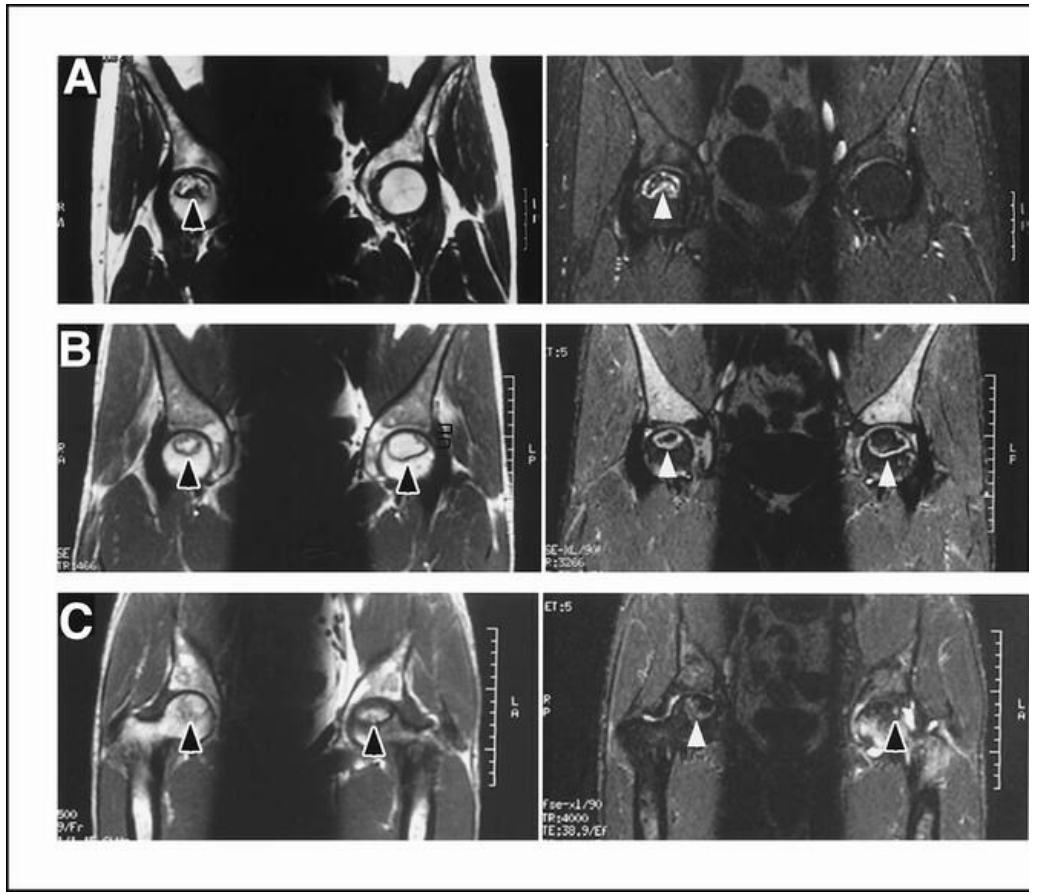
Risk Factors and Pathogenesis

- **Modifiable factors:**
  - Excess alcohol intake
  - Cigarette smoking
  - Testosterone therapy?[4]
  - Decompression disease
- **Other disease states:**
  - Glucocorticoid usage
  - Sickle cell hemoglobinopathies
  - Dyslipidemia?
- **HIV:**
  - History of AIDS-defining conditions
  - Lower CD4
  - *Lipid dysfunction?*
  - *Inflammatory pathways?*
- **ART:**
  - TDF/FTC especially if >1y[2,5]
  - Maybe past exposure to early PIs?[2,6]

2- Borges AH et al, CID 2017; 4 - Vabre C et al, Eur J Clin Pharm 2023; 5 - Bayard C et al, OFID 2017; 6 - Permpalung N et al, Int J Antimicrob Agents 2014

Management of HIV AVN

- **Symptomology**
  - Gradual onset of hip or groin pain
  - Worse with weight bearing
  - Can be bilateral and multifocal in PWH
  - Common sites: hip, shoulder
- **Imaging**
  - XR may be normal, especially if early
  - MRI is gold standard for diagnosis
  - Both sides should be imaged even if no sx
- **Management**
  - Joint replacement (total hip arthroplasty) is needed in many cases



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