

WHEN CHRONIC PAIN CLOUDS ACUTE DIAGNOSIS: A CASE OF HIP OSTEONECROSIS IN A MEDICALLY COMPLEX PATIENT

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Background

Avascular necrosis (AVN) is a progressive disorder of ischemic osteocyte death leading to structural collapse, most commonly affecting the hip [1]. Risk factors include corticosteroids, alcohol, and systemic disease [2]. MRI enables early diagnosis before joint failure [3], when joint-preserving measures remain effective [4]. Chronic pain may mask evolving pathology; clinicians should suspect AVN when new or worsening symptoms arise following recent therapies, particularly in high-risk patients, to avoid delayed recognition and irreversible joint damage.

Materials and Methods

1. Relevant patient consent was obtained for the publication of this case report.
2. As the single case report is devoid of patient identifiable information, it is exempt from IRB review requirements as per University of California, Los Angeles IRB Review Exemption policy.

Case Report

A 78-year-old man with chronic pain and prostate cancer presented with new-onset, debilitating left hip pain impairing ADLs. His longstanding pain was multifactorial, radiculopathy, SI joint dysfunction, piriformis syndrome, and hip tendinopathy/arthropathy, and had been managed with physical therapy, non-opioid adjuvants, and serial injections. Eight months prior, he developed pancytopenia and was started on systemic corticosteroids for presumed autoimmune cytopenia, followed by rituximab and ongoing tapering. A bone marrow biopsy revealed evolving myelodysplastic syndrome. Soon thereafter, his hip pain acutely worsened. MRI demonstrated a confluent marrow lesion in the proximal femur with surrounding edema (Figure. 1), and CT-guided biopsy confirmed steroid-associated osteonecrosis complicated by *Pseudomonas aeruginosa* infection. He completed 6 weeks of IV antibiotics and remains on oral suppression due to neutropenia.

Case Report. CONT

Electrodiagnostic studies revealed chronic bilateral L5–S1 radiculopathy. Serial lumbar MRIs showed progressive L4–5 canal narrowing. Early hip MRI had shown only tendinopathy. Despite extensive steroid exposure (>12 g prednisone equivalents), AVN was initially masked by chronic symptoms and attributed to baseline pathology. This case underscores the need to re-evaluate worsening musculoskeletal pain in high-risk patients, even in the context of presumed chronic disease, to avoid delayed recognition of dual pathology involving osteonecrosis and opportunistic infection.

Discussion

Patients with chronic musculoskeletal pain are prone to diagnostic overshadowing, where new symptoms are misattributed to baseline pathology [5]. In this case, evolving hip pain from steroid-induced AVN was initially attributed to longstanding radiculopathy and sacroiliitis, delaying appropriate targeted evaluation. Glucocorticoids remain the leading non-traumatic AVN trigger [6], and immunosuppression may predispose to pathogens like *Pseudomonas aeruginosa* [7, 8]. Early imaging, biopsy, and multidisciplinary care are essential in high-risk patients with atypical pain progression.

Figure.



Figure 1. Serial hip MRIs without contrast. **A)** Coronal view from initial scan (10 months prior) showing no acute pathology. **B)** Coronal view from repeat study demonstrating confluent marrow signal abnormalities in the left proximal femur (yellow arrows), along with surrounding intramuscular edema and soft tissue inflammation (red arrows). **C)** Corresponding axial view confirming extent of involvement.

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