CLINICAL VIGNETTE

Gout of the Ordinary: A Case of Clavicular Gout in HIV

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Introduction

Gout is a common form of inflammatory arthritis, occurring in in roughly 4% of adults in the United States.[1] Acute gout flares are caused by an inflammatory response to intraarticular depositions of monosodium urate crystals, which accumulate due to chronically elevated serum urate levels.[1] These episodes classically come on rapidly, are intensively painful, and most often involve the lower extremities and/or peripheral joints.[1] Gout is largely known to occur more frequently among men, certain ethnic groups (e.g. Pacific Islanders), and patients with other comorbid conditions (e.g. hypertension, chronic kidney disease, diabetes, obesity). The association of hyperuricemia with human immunodeficiency virus (HIV) infection and several anti-retroviral medications used to treat HIV is less well known.[1-3] We present an atypical case of primary gout, occurring in the sternoclavicular (SC) joint of a patient with well-controlled HIV on anti-retroviral therapy.

Case Summary

A 69-year-old male with HIV well-controlled on comprehensive antiretroviral therapy (cART) with fixed dose combination dolutegravir sodium, abacavir sulfate, and lamivudine presented to his primary care provider with 6 weeks of worsening pain in his right SC joint. The patient noted clavicular bony enlargement over 1 year, but only started to have pain in the area in the preceding 6 weeks, which prompted him to seek treatment. He denied any fevers, chills, night sweats, unintentional weight loss, or prior history of joints swelling. He noted that the pain was mildly improved with tramadol and acetaminophen at home prior to presenting.

Physical exam revealed bony prominence of the right SC joint with associated tenderness to palpation, but no overlying erythema or warmth. Initial laboratory testing was notable for an elevated serum erythrocyte sedimentation rate of 52 mm/hr, normal uric acid level, and normal white blood cell count. His CD4 count was 1419 cells/mm^3 with an undetectable serum HIV RNA level. Computed tomography (CT) scan without contrast revealed asymmetric increased subcortical sclerosis and cystic change about the right SC joint with no significant effusion or soft tissue edema or inflammation. Diclofenac sodium 1% topical gel was prescribed for pain control and a magnetic resonance imaging (MRI) study was ordered to further evaluate the lesion. The MRI detailed joint effusion, bone marrow edema, and bone erosions about the SC joint concerning for inflammatory arthritis or osteomyelitis. The patient subsequently underwent a CT-guided arthrocentesis, which aspirated 0.5cc of serosanguineous fluid. Synovial fluid cultures for bacteria, fungi, and acid-fast bacilli yielded no growth. Polarized light microscopy demonstrated extracellular birefringent crystals consistent with urate crystals, confirming a diagnosis of gout.

The patient was subsequently started on oral colchicine for an acute gout flare with some improvement in pain. Shortly after, he received an intralesional injection of triamcinolone acetonide 20mg and 0.5cc of 1% lidocaine, which significantly reduced the size and tenderness of the SC joint. On follow up 5 months later, his SC joint pain and swelling had completely resolved with no further episodes of gout.

Discussion

While most acute episodes of gout occur in the lower extremities and peripheral joints, gout can occur in a number of atypical locations including the axial skeleton, nose, eyes, and viscera.[1] Classically, gout first presents as an acute flare that is self-limiting over 1-2 weeks.[1] These episodes are often demarcated by rapid onset over 1 day and joint symptoms of pain, warmth, erythema, and swelling.[1] However, chronic gouty arthritis can occur in the absence of previous flares and lead to joint pain and destruction, as seen in our patient.[1] While elevations in serum uric acid, ESR, C-reactive protein (CRP), and WBC count can suggest an acute gout flare, they are nonspecific and are often normal.[1] The gold standard for gout diagnosis depends on synovial fluid analysis with confirmation of negatively birefringent needle-shaped crystals via polarizing light microscopy.[1]

Higher than expected frequencies of hyperuricemia and gout have been documented in patients with HIV.[2,3] Hyperuricemia and subsequent gout attacks in HIV is thought to be due to increased cellular turnover and upregulation of pro-inflammatory cytokines.[2-4] Additionally, many antiretroviral therapy agents have been associated with increased serum urate levels including didanosine, stavudine, zalcitabine, and

ritonavir.[2,3] While this patient was on fixed dose combination dolutegravir sodium, abacavir sulfate and lamivudine when he initially presented, during the first 6 months of SC joint swelling, he was on a fixed dose combination of lopinavir and ritonavir along with fixed dose combination of tenofovir disoproxil fumarate and emtricitabine. It is possible that his prior regimen, which included ritonavir, pre-disposed him to both hyperuricemia and gout. Interestingly, poorly controlled HIV and AIDS has been associated with hypouricemia due to malnutrition and increased reno-tubular losses.[4] Several case reports have reported the development of gout as part of an immune reconstitution inflammatory syndrome (IRIS) after starting antiretroviral therapy.[5] While IRIS did not likely play a role in our patient's case, this demonstrates yet another link between HIV, antiretroviral therapy, and gout.

Conclusion

Gout can occur in a variety of atypical locations, including the axial skeleton. Although serologic markers may provide some clue to an acute flare, these can often be normal. The gold standard for diagnosing gout remains arthrocentesis with direct visualization of urate crystals using polarizing light microscopy. Given the association between hyperuricemia and HIV and certain anti-retroviral medications, clinicians should include gout in the differential for patients with HIV presenting with arthralgia and/or joint swelling. Furthermore, clinicians should take into consideration other patient risk-factors for gout when choosing anti-retroviral therapy in HIV patients.

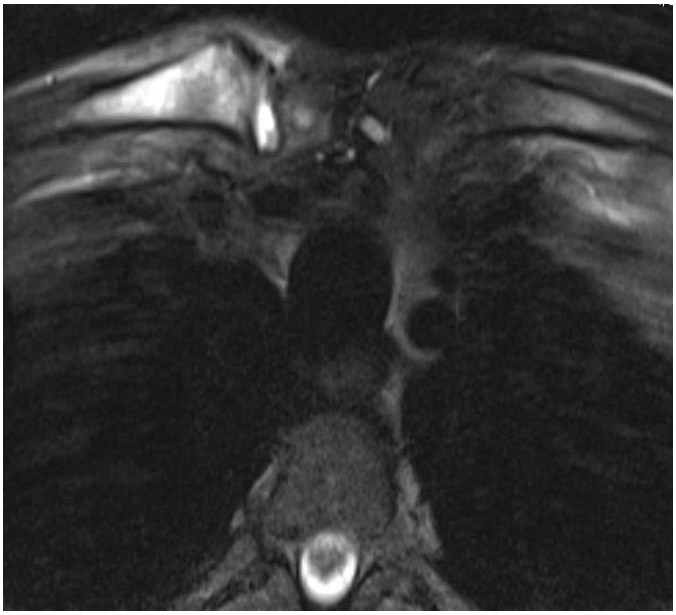

Figure 1. CT scan demonstrating subcortical sclerosis and cystic change about the right sternoclavicular joint.

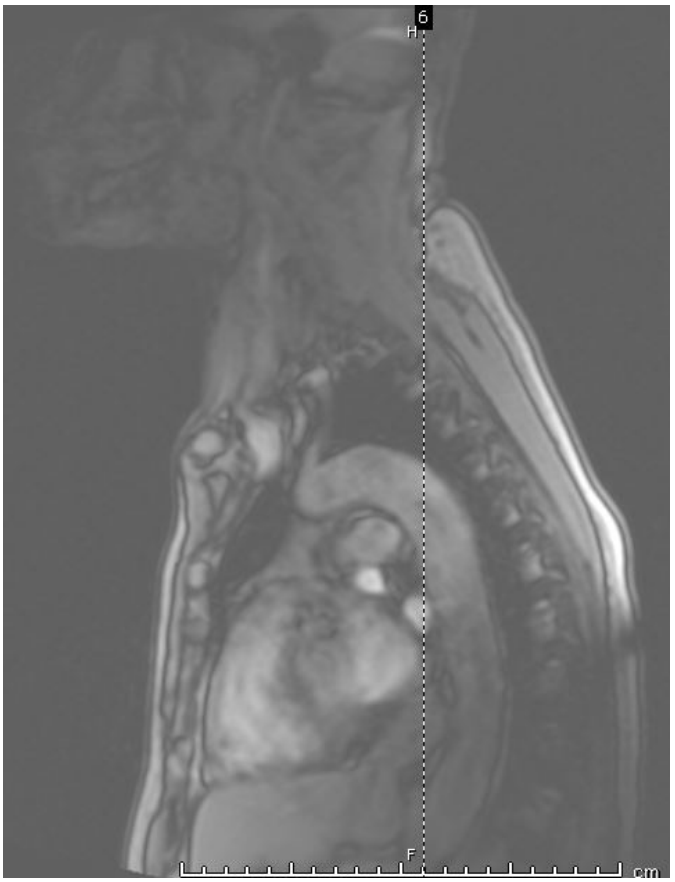

Figure 2. MRI showing joint effusion, bone marrow edema, and bone erosions about the right sternoclavicular joint.

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