CLINICAL VIGNETTE

Atypical First Presentation of Gout as Flexor Tenosynovitis

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Case Presentation

A 56-year-old female with alcoholic cirrhosis and CKD presented to the emergency room with new pain and swelling in her right hand that developed the night prior and progressively worsened with difficulty using her hand. She denied prior episodes, recent trauma or new physical activity. She did report cleaning her fish tank the previous day, without any hand wounds or skin injury. At initial evaluation, she had a temperature of 100.4°F and mild tachycardia, but otherwise normal vital signs. Physical exam was notable for erythema, edema, and tenderness to palpation over her right 3rd metacarpophalangeal (MCP) joint with associated inability to make a fist. Labs were significant for a relative leukocytosis of WBC 7.2 10E3/uL from baseline of 4.0 10E3/uL and elevated ESR of 35 mm/h. She was admitted and CT of the hand showed a well-corticated 3 by 5 mm lucent lesion in the ulnar aspect of the head of the third metacarpal thought to be degenerative, and diffuse soft tissue swelling around the third finger concerning for cellulitis with no drainable fluid collection. In consultation with the surgery and infectious diseases, she was empirically treated with intravenous vancomycin, cefepime and metronidazole to include coverage for fresh water associated organisms.

Despite broad-spectrum antibiotics and hand elevation, her symptoms and exam only marginally improved. An MRI on hospital day 2 revealed tenosynovitis of the extensor tendons of the right hand, most prominently in the 3rd digit. As the clinical presentation and course were atypical for infectious tenosynovitis, surgical treatment was deferred, and other etiologies were pursued. Bedside ultrasound of the joint revealed monosodium urate crystal deposition and admission uric acid level was elevated at 8.9 mg/dL, suggestive of gout as the etiology of her hand pain. She received oral prednisone and colchicine, leading to rapid improvement of her symptoms, exam findings, and narcotic requirements. She was unable to tolerate colchicine due to severe diarrhea, and was discharged on prednisone with rheumatology follow-up for consideration of long-term uric acid lowering therapies.

Discussion

Tenosynovitis refers to inflammation of the tendon and surrounding synovial sheath with the underlying etiology ranging from relatively benign such as repetitive use to potentially limb threatening acute infections.[1,2] Given potential severe morbidity, infectious tenosynovitis should be strongly considered at presentation. Infectious tenosynovitis can result from direct inoculation, diffusion from a nearby infection, or hematogenous spread. Individuals who are immunocompromised are at higher risk, as well as those with history of smoking, diabetes, chronic alcohol abuse, or substance use. Physical exam may reveal fever, edema of the digits, pain with passive extension, pain with palpation of the cul-de-sac of the sheath, lymphadenopathy amd lymphangitis. Management consists of early surgical debridement or washout in addition to appropriate antibiotics.[2] Surgery can lead to functional sequelae and significant morbidity necessitating a careful discussion of the risks and benefits based on clinical suspicion, especially as delay of necessary surgery can lead to necrosis of the tendons and loss of hand function. In patients that do not respond to appropriate treatment of presumed infectious tenosynovitis, investigation of other potential etiologies should be pursued.

Gout is an inflammatory arthritis characterized by the deposition of monosodium urate crystals in cartilage, bone, and periarticular tissue of peripheral joints leading to episodes of severe joint pain. While gout is common, affecting 1-2% of the population, involvement of the hand is much less common and presents variously as nerve entrapment, dermatitis, tophaceous infiltration of tendons, joint contractions, skin ulcerations, draining sinuses, and/or tenosynovitis.[3,4] Among these, gouty tenosynovitis is particularly rare. One series describes 3 patients with hand masses who underwent surgical exploration and debridement with microscopic analysis of the aspirate showing uric acid crystals.[4] Another report details a patient who was initially misdiagnosed with infectious tenosynovitis, and underwent urgent ultrasound-guided aspiration of the joint, revealing uric acid crystals and no organisms.[5] A third reports coexistent infectious tenosynovitis and gout with the patient initially undergoing surgery and treatment with broad-spectrum antibiotics. The diagnosis of gout was only entertained after the patient failed to improve with antibiotics.[6]

The gold standard for diagnosis of gout is finding negatively birefringent monosodium urate crystals under polarizing microscopy from joint aspiration.[7] Ideally, joint aspirate can evaluate for deposition disease such as gout and pseudogout as well as for infectious etiologies with bacterial stain and culture. Unfortunately, our patient did not have a drainable synovial fluid collection. Hyperuricemia and bedside ultrasound can be useful in cases without definitive fluid analysis. Hyperuricemia is a serum urate level greater than 6.8 mg/dL (404 mmol/L) and is associated with an elevated risk of gout, though it does not

necessarily correlate with acute gout flares.[7] In a cohort study, the annual rate of gouty arthritis was 4.9% in patients with serum urate levels greater than 9.0 mg/dL and only 0.5% in patients with serum urate levels between 7.0 and 8.9 mg/dL, compared to 0.1% in patients with serum urate levels less than 7.0 mg/dL.[8] Bedside ultrasound can be suggestive of gout with a sensitivity of 76% and specificity of 84%.[9] Our patient had both hyperuricemia and findings of crystal disease on bedside ultrasound. Once we felt infection was unlikely, we treated her for presumed gout with prednisone with subsequent rapid improvement.

Conclusion

Initial evaluation and treatment of tenosynovitis should strongly focus on infectious etiologies given the significant potential morbidity associated with this diagnosis. Treatment includes broad antibiotics and surgical consultation for potential early debridement and washout. In cases without expected improvement and no clear infectious source, alternative diagnoses should be considered, such as acute gouty tenosynovitis even in patients without a history of gout. Our patient was treated for infectious tenosynovitis without significant improvement, and subsequent bedside ultrasound findings and hyperuricemia were supportive of gout as the underlying etiology, which was further supported by her rapid improvement with corticosteroid therapy. While joint aspiration is preferred, it is not always obtainable, and bedside ultrasound and serum urate levels may be useful in the diagnosis of gout.

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