CLINICAL VIGNETTE

An Atypical, Yet Not Uncommon Patient with Acute Gouty Foot Pain

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The patient is a thirty eight year old male presenting with pain in his right foot. The pain started acutely 3 days prior and was severe, rated 10/10. He limped into the office with an obvious antalgic gait. There was no trauma prior to the onset and no prior episodes of pain. He has no known drug or food allergies, and takes only prn inhaled steroids and antihistamines for mild seasonal allergies. He is physically active and exercises three times a week. He works at a restaurant and family history is significant for his father with hypertension and gout.

Physical exam included normal vital signs and BMI of 25. The foot exam showed mild edema and erythema over the lateral right fifth metatarsal-phalangeal area. Flexion, abduction, and extension of the joint were somewhat difficult due to severe pain. The history and exam were suggestive of gout and no fracture and no signs of degenerative joint disease or erosions were seen on foot films. Despite the lack of pain at the first metatarsal phalangeal joint, a clinical diagnosis of gout was made without attempts to aspirate the joint. The patient was informed of the diagnosis, educated on a low purine diet, and started on colchicine.

As this was his first gout attack, urate lowering therapy was not initiated. However, after finding an elevated uric acid level and three more attacks in the next six months, low dose allopurinol was started at 100mg daily. He admitted to not being able to resist his favorite seafood and the occasional steak with wine, which seemed to consistently trigger the attacks. Three weeks after he started allopurinol, he got another attack. This was likely related to initiation of uric acid lowering therapy. He was started on low dose colchicine and his allopurinol dose was titrated to 200mg over the next six months without subsequent attacks. However he felt tired all the time with mild nausea and some diarrhea which he attributed to his medications. He read about allopurinol reactions in certain groups such as Han Chinese for which was the ancestry of one of his grandparents. He was switched to febuxostat, a newer urate lowering drug and xanthine oxidase inhibitor. (1) He reported no new side effects nor gout attacks over the next 3 months. Uric acid levels were initially over 10 mg/dL, and decreased to 7. Eventually a goal of <5 was established. Uric acid levels >7 are arbitrarily considered hyperuricemia although the normal range is 4-7. The recommended gout target is <6 although some studies suggest the lower the better in terms of reducing the number of acute flare ups. (2) As febuxostat may cause elevation of LFT's, they were checked at baseline and periodically while also following uric acid. One review article reported febuxostat was "noninferior to allopurinol and generally well tolerated," (3) and justified the trial in this patient with risk for allopurinol hypersensitivity syndrome. Additionally, although it was unclear if the colchicine or the allopurinol or both were responsible for his initial symptoms, the patient's demographic background increased his risk for the rare hypersensitivity syndrome, also increased in Koreans and Thais.

The diagnosis of gout was established clinically with an initial period of uncertainty given the lack of gold standard diagnosis (from joint aspiration revealing negatively birefringent crystals), atypical, though common location of joint pain for gout, in an otherwise healthy young patient. However the overwhelming clues pointing towards gout included rapid onset, unilateral monoarticular nature, and diet related triggers. This case is a good illustration of how a good history and exam can usually establish the diagnosis, with imaging and blood tests only helping to confirm. It also reflects the current research that urate crystals form at a variety of unexpected sites, often without clear inflammatory sequelae. More studies on the effects of diet, metabolism, genetics, and inflammatory responses are needed to help guide clinical approaches and set targets in the therapy for gout.

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Submitted April 18, 2017