



Chronic Tophaceous Gout

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Abstract

A condition of purine metabolism, gout is characterised by sporadic bouts of acute inflammation, pain, swelling and redness of joint. It is usually preceded by hyperuricemia related to increased uric acid synthesis or reduced excretion which leads to monosodium urate deposits in soft tissues and synovial joints, known as tophi.

Keywords: Hyperuricemia; tophi; gout.

Introduction

A number of factors can contribute to gout, including male gender, obesity, age between 30 and 50, kidney disease, and genetics¹. Drugs like aspirin, hydrochlorothiazide, diuretics, organic acids, etc. may also contribute to decreased kidney clearance. Purine-rich food items like meat and seafood are another well-known risk factor. Alcohol has been well known for precipitating uric acid crystals and precipitating acute gout attacks. Gout can occasionally be a symptom of tumour lysis syndrome, which is primarily brought on by purines released during the death of tumour cells².

In addition to causing joint inflammation, hyperuricemia can also result in kidney or bladder stones, metabolic disease, nephropathy, and cardiovascular disease. Stone formation and nephropathy are caused by the crystallisation of

uric acid in the urinary lumens³. Synergistic effects can also result from concurrent hypercalcemia.

Nephropathy and cardiovascular diseases like hypertension, heart failure, and coronary disease are primarily caused by the deposition of monosodium urate, which causes endothelial dysfunction and a pro-inflammatory response^{2,3}.

Tophus is created when monosodium urate crystals and by-products of chronic inflammation cluster together⁵. Leukocyte chemotaxis causes neutrophils to attempt to phagocytose the crystals, but when they do, the crystals puncture them and release damaging enzymes into the joint space, causing further inflammation^{3,7}.

Case Report

A 50 year old male with a 5-6 year history of gout diagnosis and subsequent non adherence to

medications presented to us on outpatient basis. Our patient had been advised to take febuxostat 40 mg PO every day. However, the patient was non-compliant and instead resorted to traditional medicine. He was noted to have multiple hard swellings, there was no personal history of alcohol use, or family history of gout, however there was a history of purine rich diet intake present. Upon physical examination three hard swellings were noted, one in pulp of right thumb, one in right middle finger proximal interphalangeal joint and another in big toe area at base of first metatarsal (Figure 1). He is newly diagnosed with hypertension and hypothyroidism during this outpatient visit. Patient has a BMI of 30 kg/m². Laboratory workup revealed elevated serum uric acid (8.7 mg/dl; normal =3.5-7.2) with a normal serum creatinine (1.15 mg/dl; normal =0.72-1.25) and serum urea (27 mg/dl; normal=10.0-50.0). Radiograph of right hand (figure 2) showed soft tissue swelling and deposits of radiolucent substance in pulp of right thumb. Radiograph of foot showed soft tissue swelling and destruction of first metatarsophalangeal joint, distal end of 1st meta tarsal and 1st proximal phalanx with classic punched out erosions. The patient was treated on outpatient basis with allopurinol (100 mg/d).



Figure 1: Hard swelling with subdermal whitish deposits in pulp of right thumb, another one in right middle finger proximal interphalangeal joint and another in big toe area at base of first metatarsal.



Figure 2: Soft tissue swelling and deposits of radiolucent substance in pulp of right thumb



Figure 3: Radiograph of foot showed soft tissue swelling and destruction of first metatarsophalangeal joint, distal end of 1st meta tarsal and 1st proximal phalanx with classic punched out erosions

Discussion

Gout and hyperuricemia are becoming more common in developing nations, which is likely a result of population ageing, alcohol consumption, hypertension, obesity, metabolic syndrome, and diuretic use. Long-term hyperuricemia and urate crystal deposition in various tissues cause gout, a disorder of purine metabolism². Every patient experiences hyperuricemia at some point during their illness. However, at the time of an acute attack, serum uric acid levels can be normal or low, and people with asymptomatic hyperuricemia may never experience a clinical event brought on by urate crystal deposition^{3,6,8}. In the initial stage, other joints are less frequently affected than the first metatarsophalangeal joint². The midtarsi,

ankles, knees, and arms are the next most common localizations.

In the synovial fluid and other tissues, deposits of monosodium urate crystals (MSU) cause gout, which is linked to hyperuricemia³. Immune activation follows crystal deposition. Tophi are subcutaneous nodules in and around joints or soft tissues made up of crystal aggregates. Tophi can develop in soft tissue, osseous tissues, ligaments, and various organs⁸. The helix of the ears, the fingers, toes, wrists, knees, the olecranon bursae, the Achilles tendons, the sclerae, and the cardiac valves are common sites for tophi to develop^{2,4}. The overlying skin may become ulcerated as a result of superficial tophi⁵.

Urate crystal deposits that are encircled by a pronounced inflammatory response of macrophages, lymphocytes, and large foreign body giant cells are histopathological features^{3,7}. A distinguishing feature of urate crystals is their birefringence. Although the generalised form of tophaceous gout has become less common over the past few years, the disease still exists, most likely because of underdiagnosis and non-compliance to treatment^{5,6}. Large tophi, which are uncommon in chronic gout, were present in our case. Oral colchicine and/or non-steroidal anti-inflammatory drugs are the first-line treatments for an acute flare⁹. It is also possible to use systemic or intra-articular corticosteroids, which are equally effective but have more side effects¹⁰. Interleukin-1 inhibitors are still being researched and are not currently prescribed for a gout attack that is acute. The first-line treatment for lowering serum uric acid is allopurinol. As urate-lowering treatments, probenecid, colchicine, and other xanthine oxidase inhibitors like febuxostat are also possible^{2,9}.

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