# Polyarticular Symmetric Tophaceous Joint Inflammation as the Initial Presentation of Gout

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**A 76-year-old woman suffered from bilateral distal index finger pain and swelling. The patient had been initially treated with antibiotics for herpetic whitlow complicated by a secondary bacterial infection. Gout was diagnosed through clinical history, physical examination and identification of monosodium urate crystals in the joint aspirate. Gout appearing as polyarticular, symmetric tophi involving the periungual region and distal interphalangeal joint has not been previously described. (Am J Emerg Med 1990;8:43-45. © 1990 by W.B. Saunders Company.)**

Gout has been known since antiquity, and was described in medieval times as *Gutta,* meaning "disfluxion of the humours."[1] The first article describing this disease entity was published by Garrod in 1876.[1] In 1892, Osler characterized gout as "a nutritional disorder associated with an excess formation of uric acid".[2] The classic presentation of gout is a disease affecting middle to older aged men, with a recurrent monoarthritis of the metatarsophalangeal joint.[3,4]

Although gout has a 95:5 male predominance, it is also described in women.[5] This clinical presentation typically occurs in elderly postmenopausal women with degenerative joint disease who are maintained on diuretics. Furthermore, in women it can manifest as a polyarticular upper extremity pattern.[6]

However, Yu,[5] in a review of 23 cases of gout in women, found none that appeared as polyarticular disease. Raddatz et al[7] examined 41 men with polyarticular gout and reported a 12% incidence of polyarticular disease on first appearance. The distal interphalangeal joint was involved in 3% of the cases reported; however, none had bilateral symmetric disease.[7] This case of polyarticular, symmetric periungual, and distal interphalangeal joint inflammation is unique as an initial presentation of tophaceous gout.

## CASE REPORT

A 76-year-old woman came in to the emergency department of Presbyterian-University Hospital with a complaint of bilateral index finger pain and swelling. This condition had initially been diagnosed as herpetic whitlow with secondary bacterial infection. The patient had begun taking erythromycin and had been instructed to use warm compresses three times a day. On the return visit to the medical clinic, the patient demonstrated no clinical improvement in the condition after 1 week. The patient was referred to the emergency department for surgical consultation and possible incision and drainage of the presumed infected areas.

The patient had a medical history with sick sinus syndrome, degenerative joint disease, and hypertension. Her only medication included hydrochlorothiazide. Neither the patient nor the medical records revealed any medical history or family history of gouty arthritis. The current illness had lasted for 1 week and included no fever or other systemic symptons. The patient denied the use of alcoholic beverages, including moonshine. Furthermore, there was nothing in the patient's history to suggest recent or remote lead intoxication.

Physical examination showed a temperature of 36.6°C, a pulse of 72/min, a respiratory rate of 20/min, and a blood pressure of 166/84 mm Hg. The general physical examination results were unremarkable with the exception of a II/VI systolic ejection murmur. The musculoskeletal examination showed bilateral, symmetric distal index finger erythema, swelling, and tenderness associated with nodular yellow periungual deposits without warmth (Fig 1). Heberden's nodes, along with other stigmata of osteoarthritis, were apparent but not involved in the inflammatory process. There was slight restriction of mobility in the distal interphalangeal joints, but the other joints in the hand, wrist, and fingers had a normal range of motion. The neurovascular examination of the digits was also normal.

Laboratory profile showed a hemoglobin of 15.5 g and a white blood cell count of 15,600 with 76 segmented neutrophils and no bands. The blood chemistries included Na, 139 mEq/L; K, 3.5 mEq/L; Cl, 95 mEq/L; $HCO_3$, 30 mEq/L; blood urea nitrogen (BUN), 36 mg/dL; and creatinine, 2.2 mg/dL. The serum uric acid level was 12.8 mg/dL.

Roentgenograms of the hands demonstrated bilateral subchondral cysts with overhanging margins localized to the distal interphalangeal joint suggestive of gouty arthritis (Fig 2). Needle aspiration of these lesions produced fluid that was negative for bacteria or leukocytes on light microscopy. Polarized microscopy revealed yellow, negatively birefringent, needle-shaped crystals that were identified as monosodium urate. A sample of the joint aspirate was sent for culture to exclude infection.

The physical findings, laboratory profile, and joint fluid analysis provided the diagnosis of tophaceous gout. The patient was treated with indomethacin, colchicine and instructed to discontinue her hydrochlorothiazide. She was referred for outpatient rheumatologic evaluation, whereby the diagnosis of gout was later confirmed.

## DISCUSSION

Gout is a relatively common disease with an incidence of 2.7% to 9.7%[8]; approximately 5% of this population is composed of women.[9] The Framingham study, in a prospective analysis of 5,000 patients for coronary artery disease, found a 1.5% prevalence of gout (2.8% male, 0.4% female).[10] The male predominance of gout is well established, with the disease affecting women in only 5.1% of the cases in 19 series reported since 1900.[6]

Gout is a phenomenon of middle age, with the first attack occurring at an average age of 48.7 years (47.7 in men and 54.1 in women).[6] A family history of gout occurs in 85% of those patients with gout.[11] In elderly women, however, a

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Manuscript received February 2, 1989; revision accepted March 28, 1989.

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*Key Words:* Polyarticular, tophaceous gout.



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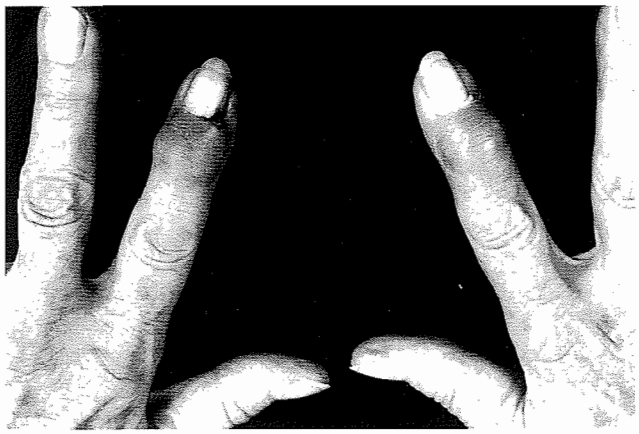

FIGURE 1. Bilateral distal index finger erytherma and swelling.

family history of gout is present in only 7.5% of patients.[11] Associated medical conditions occurring in the patient with gout include hypertension (73%), renal insufficiency (52%), diabetes mellitus (36%), and coronary artery disease (32%).[11]

Gout in women usually occurs later in life, and most often occurs in those patients with preexisting osteoarthritis, renal insufficiency, or on diuretic therapy.[6] Examination of these predisposing factors indicates that gout is associated with osteoarthritis of the distal interphalangeal joint, clinically known as Heberden's nodes. This predisposition is the result of the decreased temperature, pH, and trauma associated with the peripheral extremities encouraging crystal deposition.[12] Interestingly, the patient described in this case did not have her osteoarthritic joints involved in this first presentation of gout.

Diuretic therapy, also strongly associated with gouty arthritis in elderly women, is reported in the range of 95% to 100% of cases.[11,13] Urate excretion is decreased in the presence of thiazide diuretics. The mechanism is enhanced proximal tubule reasbsorption, secondary to volume depletion and reduced distal tubule secretion, mediated by the aldosterone/angiotensin system.[14] Renal insufficiency is associated with, but not causally related to, gout. Berger and Yu separately concluded that uncomplicated gout has no effect on renal function.[15,16]

Gout is categorized as primary when a known metabolic defect exists or when gout is the main manifestation, or as secondary when an unknown metabolic defect exists or gout is acquired.[17] The etiologic classification of hyperuricemia includes increased production due to gout, Lesch-Nyhan syndrome, or lymphoproliferative disorder, or decreased elimination secondary to intrinsic renal disease. Decreased urate excretion can occur secondary to competition with organic acids such as thiazide diuretics, lactate, or ketones.[17]

The diagnosis of gout begins with the triad of acute monoarthritis, hyperuricemia, and a satisfactory response to colchicine therapy.[18] Gout is diagnosed on presentation in only 50% of the cases; the most common incorrect diagnosis is infection.[7] A comprehensive means of diagnosis has been established by the American Rheumatism Association Subcommittee for the classification of gout based on analysis of 700 patients (Table 1).

The first criterion necessary for the diagnosis of gout is the presence of monosodium urate crystals in synovial fluid.n In 1679, Antonj Van Leeuwenhoek first discovered these crystals under the microscope.[1,19] Examination of fluid aspirated from our patient's periungual region showed the rod-like or needle-shaped, yellow, negatively birefringent crystals characteristic of gouty synovial fluid. The visualization of negative elongation, or yellow appearance of parallel crystals with a first-order red compensator and polarized microscopy is diagnostic of monosodium urate crystals and gout.[20] The absence of leukocytes on the Gram stain of this patient's synovial fluid is difficult to explain since urate crystals are leukotactic and an inflammatory response would be expected in the setting of urate crystal–induced synovitis.

Secondly, the presence of tophi, although not routinely seen, are highly suggestive of gout. The tophus consists of a core of monosodium urate crystals surrounded by inflammatory cells.[1] These are rare at the time of first presentation and usually suggest previous attacks of gouty arthritis.[1,21] Tophi are rarely seen in women, with an incidence of only 5%.

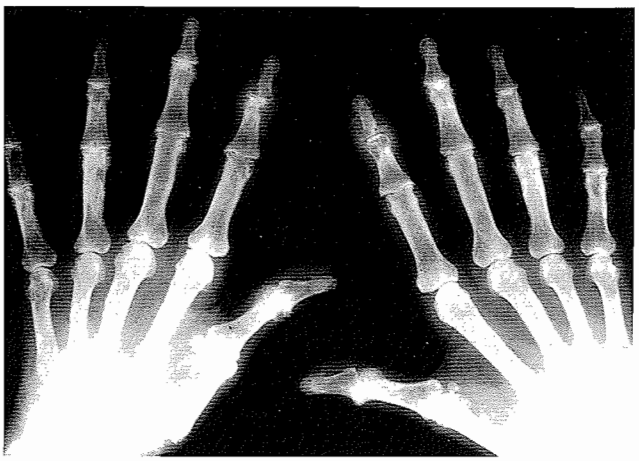

FIGURE 2. Bilateral subchondral cysts with overhanging margins localized to the distal interphalangeal joint.

**TABLE 1.** Diagnostic Criteria for Gouty Arthritis

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| Uric acid crystals in joint fluid |
| Tophus with uric acid crystals |
| Six clinical, laboratory, and radiological phenomena associated with gout: |
| Greater than one attack of arthritis |
| Maximum inflammation within 24 h |
| Monoarthritis |
| Redness over joint |
| Unilateral first metatarsal phalangeal |
| Painful or swollen first MTP |
| Unilateral tarsal joint attack |
| Tophus |
| Hyperuricemia |
| Asymmetric joint swelling on roentgenography |
| Subcortical cysts without erosion on roentgenography |
| Monosodium urate crystals in joint fluid |
| Negative cultures of joint fluid |

Adapted from Wallace SL et al.[24]

Radiological criteria are also used in the diagnosis of gout. Soft tissue calcification is often visible as eccentric asymmetric nodular deposits.[18] Specifically, sharply defined marginal erosions of the subchondral bone with overhanging margins appear in gouty arthritis.[8,22] These radiological findings were evident in our patient.

The clinical presentation of gout can be confirmed by the measurement of serum uric acid levels. Hyperuricemia, as defined by a serum uric acid level of 7.0 mg/DL or more, is manifested in 5% of the population.[18] The male predominance of gout is believed to be due to the protective effect of estrogen in women, because estrogen will cause an increase in uric acid excretion.[23] This will result in a serum uric acid level of 6.0 mg/DL or less in most female patients.[9] Our patient had a uric acid level of 12.8 mg/dL during her presentation.

## CONCLUSION

This case is submitted as a unique case of gout in an elderly woman. The symmetric polyarthritis with tophi involving the distal interphalangeal joint and adjacent periungual soft tissue as the initial manifestation of gout is unusual, and represents the first such case reported. This symptom complex has been rarely described, but always with a prior history of gouty arthritis.[6,11] Our patient demonstrated several of the diagnostic criteria and clinical characteristics of gout, but the condition was initially misdiagnosed as an infectious process. We present this case as an example of how an occult disease process can be diagnosed by careful reveiw of patient history, physical examination, and directed laboratory profile in a busy emergency department.

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