Chronic Tophaceous Gout Treated Mistakenly as Rheumatoid Arthritis for One Year

Hasan Ulusoy[1], Gurkan Akgol[1], Arif Gulkesen[2], Ayhan Kamanli[1]

ABSTRACT

Gout is a common disorder characterized by hyperuricemia and the deposition of monosodium urate crystals in various tissues. Clinical manifestations of gout include attacks of acute inflammatory arthritis, a chronic destructive arthropathy known as chronic tophaceous gout, and non-articular accumulation of monosodium urate crystals crystals suchs as tophi and kidney stones. Acute gouty arthritis usually begins with one joint in the lower limbs, such as first metatarsophalangeal joint, midtarsi, ankles and knees. Subsequent attacks frequently last longer than does the first attack, affect several joints, and are prone to spread to the upper limbs, especially to small joints of the hands. Although acute gouty arthritis is familiar for most physicians chronic gouty arthritis which affects small joints of the hands can be difficult to distinguish from other common interphalangeal arthropathies such as rheumatoid arthritis, psoriatic arthritis and erosive osteoarthritis. Because of very similar presentations, an awareness of the differential diagnosis, as well as a combination of clinical, radiographic, and laboratory findings is necessary to differentiate these diseases. Here we describe a case of chronic tophaceous gout who had been treated mistakenly as rheumatoid arthritis for one year.

Key words: *Gout, gouty arthritis, chronic tophaceous gout, interphalangeal arthropathies*

Bir Yıl Romatoid Artrit Olarak Takip Edilen Kronik Tofüsllü Gut

ÖZET

Gut hiperürisemi ve monosodyum ürat kristallerinin çeşitli dokularda çökmesi ile karakterize yaygın bir hastalıktır. Gutun klinik görünümleri akut inflamatuvar artrit ataklarını, kronik tofüslü gut olarak bilinen kronik destrüktif bir artropatiyi ve monosodyum ürat kristallerinin tofüs ve böbrek taşları gibi eklem dışı birikimlerini içerir. Akut gut artriti sıklıkla birinci metatarsofalangeal, midtarsal, ayak bileği ve diz eklemi gibi alt ekstremite eklemlerinden birinde başlar. Sonraki ataklar sıklıkla ilk atağa göre daha uzun sürer ve birden fazla eklemi etkiler, üst ekstremite ve özellikle elin küçük eklemlerine yayılma eğilimindedir. Akut gut artriti çoğu hekim için tanıdık olsa da, elin küçük eklemlerini etkileyen kronik gut artritinin romatoid artrit, psöriatik artrit ve eroziv osteoartrit gibi diğer yaygın interfalangeal artropatilerden ayırımı zor olabilmektedir. Oldukça benzer sunumları nedeniyle, ayırıcı tanının bilincinde olanın yanı sıra, klinik, radyolojik ve laboratuvar bulguların kombinasyonu bu hastalıkları ayırabilmek için gereklidir. Biz bu yazıda bir yıldır yanlışlıkla romatoid artrit olarak tedavi edilmiş bir kronik tofüslü gut olgusu sunuyoruz.

Anahtar kelimeler: *Gut, gut artriti, kronik tofüslü gut, interfalangeal artropatiler*

INTRODUCTION

Gout is a heterogeneous disorder characterized by hyperuricemia and the deposition of monosodium urate (MSU) crystals in and around the joints (1). It affects 1-2% of adults in developed countries, where it is the most common inflammatory arthritis in elderly men (2). Clinical manifestations of gout include attacks of acute inflammatory arthritis, a chronic destructive arthropathy (chronic tophaceous gout) and non-articular accumulation of MSU crystals suchs as tophi and kidney stones (1,3).

[1]*Division of Rheumatology, Department of Physical Medicine and Rehabilitation, Firat University, Faculty of Medicine, Elazıg, Turkey,* [2]*Department of Physical Medicine and Rehabilitation, Elazıg Training and Research Hospital of the Ministry of Health, Elazıg, Turkey*

Correspondence: *Uzm. Dr. Hasan Ulusoy*
Firat Universitesi, Tip Fakultesi, Fiziksel Tip ve Rehabilitasyon AD, Romatoloji BD 23119 Elazig, Turkey
E-mail: ulusoyh@mynet.com

Received: 14.10.2010, Accepted: 18.10.2010

Although the initial attack of arthritis is monoarticular and usually affects the first metatarsophalangeal (MTP) joint, polyarticular involvement becomes much more frequent as time goes on, especially in the chronic tophaceous gout stage. With diffuse and symmetric involvement of small joints in the hands and feet, chronic tophaceous gout can be confused with other common interphalangeal arthropathies such as rheumatoid arthritis (RA), psoriatic arthritis (PsA) and erosive osteoarthritis (EOA) (1,4,5). Here we describe a case of chronic tophaceous gout who had been treated mistakenly as RA for one year.

CASE

A 66-year-old man presented with pain and swelling episodes of wrists, metacarpophalangeal (MCP) and proximal interphalangeal (PIP) joints in both hands for three years. He also described painful swelling and redness attacks at the dorsum of the feet for over six years. These painful episodes had been relieving with non-steroidal anti-inflammatory drugs (NSAIDs) within two days. In an out patient clinic, the patient had been diagnosed as RA and treated with methotrexate 12.5 mg/week and sulfasalazine 2 g/day for one year. Despite decreasing, his complaints had repeated sometimes in the small joints of the hands. On initial examination, tenderness and swelling were found in the wrists, MCP and PIP joints (Figure 1). Strikingly, an olecranon bursitis and the tophaceous deposits located within the skin overlying this bursa were shown on the left elbow (Figure 2). Hand radiographs showed the typical features of chronic gout in the MCP and PIP joints, including extra-articular erosions with overhanging edges, spared joint spaces and relative lack of periarticular osteopaenia (Figure 3). Laboratory findings showed a normal erythrocyte sedimentation rate (ESR) (10 mm/h; normal <20) and C-reactive protein (CRP) level (4.8 mg/l; normal <5 mg/l). The patient was negative for rheumatoid factor (RF), anti-cyclic citrullinated peptide antibodies (anti-CCP) and antinuclear antibodies (ANA). C3 and C4 complement levels were within normal limits. Although the serum uric acid level was slightly elevated (7.7 mg/dl; normal <7 mg/dl), 24-hour urinary excretion of uric acid was markedly elevated (1048 mg; normal <750 mg). Abdominal ultrasound did not reveal any urate stone in the kidneys. According to these clinical, radiological and laboratory findings he was diagnosed with chronic tophaceous gout and other possible rheumatic diseases which could affect small joints in the hands were excluded. Thereafter, the patient was prescribed a medical treatment consisted of allopurinol (300 mg/day) and colchicine (1.5 mg/day). Methotrexate and sulfasalazine doses were gradually tapered and eventually completely stopped. Six months after initiation of allopurinol, there is a considerable decreasing in the excretion of uric acid in twenty-four hour urine (from 1048 mg to 386 mg). No evidence of any collagen tissue disease has been shown during clinical and laboratory follow-up for six months.

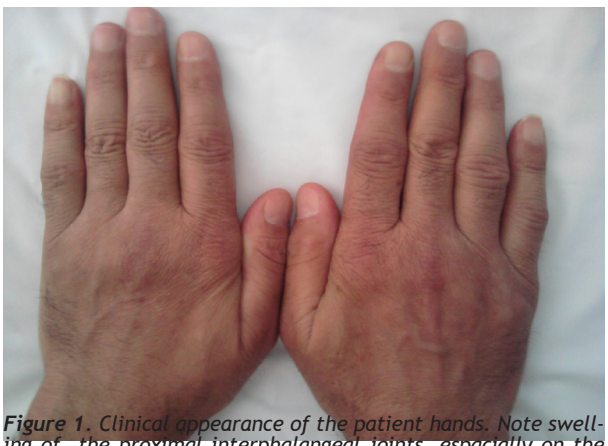

***Figure 1**. Clinical appearance of the patient hands. Note swelling of the proximal interphalangeal joints, espacially on the second, fourth and fifth fingers of right hand, on the second and fifth finger of left hand.*

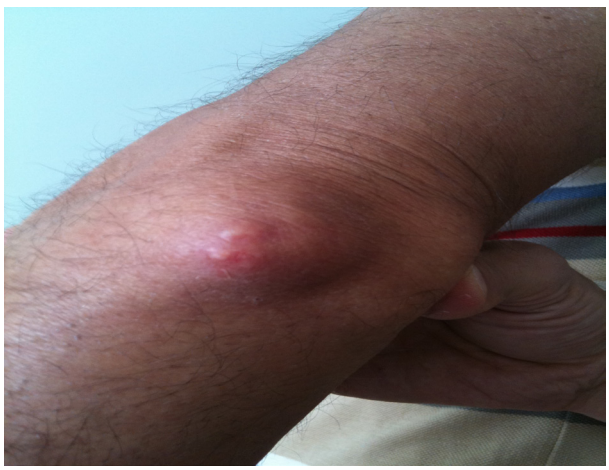

***Figure 2**. Deposits of uric acid (tophi) within the skin overlying the olecranon process.*

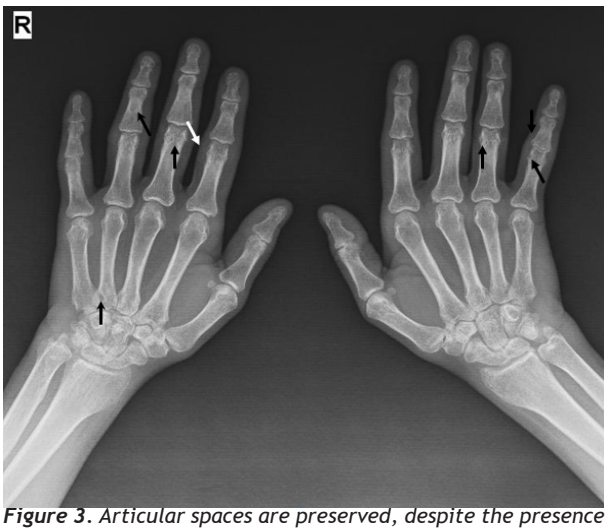

***Figure 3**. Articular spaces are preserved, despite the presence of osseous erosions. Soft tissue swelling of proximal interphalangeal joints is evident. Extra-articular erosions at the ulnar aspects of second finger of right hand (white arrow). Note overhanging margins associated with these erosions in this finger. Extra-articular cysts (black arrows) in the proximal phalanx of third finger, in the middle phalanx of fourth finger, in the forth metacarpal of right hand. Subtle cysts at the fifth and fourth proximal interphalangeal joints of left hand (black arrows).*

***Table 1**. Criteria for the classification of primary gouty arthritis of American College of Rheumatology.*

A. The presence of characteristic urate crystals in the joint fluid
or
B. A tophus proved to contain urate crystals by chemical means or microscopy
or
C. The presence of 6 of the following 12 findings listed below:
1. More than one attack of acute arthritis*
2. Maximal inflammation developed within 24 hours*
3. Attack of monoarticular arthritis*
4. Joint redness observed*
5. First metatarsophalangeal joint painful or swollen
6. Unilateral attack involving first metatarsophalangeal joint
7. Suspected tophus*
8. Hyperuricemia*
9. Asymmetric swelling within a joint
10. Subcortical cysts without erosion*
11. Negative culture of joint fluid during acute attack

DISCUSSION

Gout is probably the best understood and most manageable of all common systemic rheumatic diseases. It is a disorder of purine metabolism and results from long-standing hyperuricaemia and urate crystal deposition in various tissues (6,7). The natural history of articular gout is typically composed of three periods: asymptomatic hyperuricaemia, episodes of acute gouty arthritis with asymptomatic intervals, and chronic gouty arthritis (1). Acute gouty arthritis usually begins with one joint in the lower limbs (85-90% of cases). The first MTP joint is the most frequent initial point (60% of cases), which is classically termed podagra. The next most frequent locations are the midtarsi, ankles, knees and arms. The initial attack is rarely polyarticular (3-14% of cases) (1,2). Subsequent attacks frequently last longer than does the first attack, affect several joints, and spread to the upper limbs, especially to small joints of the hands. In untreated patients chronic tophaceous gout may develop, which is characterised by chronic destructive polyarticular involvement and tophi (7,8). If small joints of the hands is affacted chronic tophaceous gout can be confused with other more common interphalangeal arthropathies. Although the definite diagnosis of gouty arthritis rests upon the presence of MSU crystals in synovial fluid or tophus aspirate, analysis of clinical, radiological and laboratory findings are the keys to differentiate of this disease from other erosive interphalangeal arthropathies such as RA, PsA, EOA, chronic renal diseases and endocrine diseases (9). Although our patient did not approve of aspiration of tophi, the presence of elevated serum uric acid level, raised 24-hour urinary excretion of uric acid, tophus formations and characteristic radiological appearances allowed to diagnose the patient with chronic gouty arthritis. Moreover, these findings have met the criteria for the classification of primary gouty arthritis of American College of Rheumatology (Table 1) (10).

The prevalence of gout is much higher in men than in women and rises with age (11). Epidemiologically, our patient who was a 66-year-old man was prone to develop gout. Hyperuricaemia is the key predictor for development of gout (9). Both serum level and 24-hour urinary excretion of uric acid were higher than normal values in the current patient. Renal mechanisms are responsible for hyperuricaemia in about 90% of individuals because impaired excretion of renal uric acid is the main mechanism underlying the rise in the urate pool (12). Renal function tests were normal in this patient. Patients who overproduce uric acid represent less than 10% of those with gout (1). Diseases associated with enhanced turnover of nucleic acid and hyperuricaemia, such as myelo-

proliferative and lymphoproliferative disorders, haemolytic anaemia, and psoriasis were not present in the patient. Gout may be associated with use of several drugs, including diuretics, low-dose salicylates, ciclosporin, tacrolimus, levodopa, ethambutol and teriparatide (13). The patient has not been using any of these drugs. Two inherited enzyme abnormalities which result in hyperuricaemia, gouty arthritis, nephrolithiasis and neurological manifestations are hypoxanthine-guanine phosphoribosyl transferase deficiency and phosphoribosylpyrophosphate synthetase superactivity. Clinical symptoms are present early in life in all patients with these enzyme abnormalities (2). First complaint had started in 60 years old in our patient. Tophi¬¬¬¬, MSU crystals surrounded by chronic mononuclear and giant-cell reactions, are frequently seen in the helix of the ear, over the olecranon processes, on the Achilles tendons, around the finger joints, around the knees, and within the pre-patellar bursae. Other rare locations are the spinal colon, eyes, breast, vocal cords, heart, and colon (1,2,8). There were tophus formations over the olecranon process in the current patient.

Generally acute gouty arthritis does not show abnormal findings on plain radiography, apart from non-specific soft-tissue swelling. By contrast, chronic gouty arthritis might show characteristic features, mainly the consequences of tophus infiltration into bone. Bone erosions are key features and are at first extra-articular. They are typically punched out, occurring along the long axis of the bone, with overhanging edges and sclerotic rims. The joint space is very well preserved until late stages in the course of disease (14). Although there was a three years duration of interphalangeal joints involvement in our patient, the joint spaces were well preserved. In addition, extra-articular erosions with overhanging edges and bone cysts were shown. These radiographic appearances were consistent with chronic gouty arthritis.

Although various clinical and radiological clues exist, gouty arthritis is especially confused with RA. Important radiographic features distinguishing gouty arthritis from RA are the presence of marginal erosion, joint space narrowing and marked periarticular osteopenia in the latter (15). In addition to the radiographic findings, the conjunction of both a negative RF and anti-CCP test made the diagnosis of RA unlikely in this patient.

Interphalangeal joints involvement is a common feature of PsA, but psoriatic skin and nail abnormalities, family history of psoriasis, sacroiliac and spinal radiographic changes are additional features which help in differentiating PsA from gouty arthritis. We should note that all of these were negative in our patient. Additionally, similarly to RA, PsA causes marginal erosions giving “mouse ear” appearance in the interphalangeal joints (16,17).

In daily practice, EOA is an other frequently encountered interphalangeal arthropathy. Radiological features of EOA include joint space narrowing, subchondral sclerosis, marginal osteophytes, and erosions, which begin at the central portion of the joint and give characteristic patterns of the affected joints, known as “gull-wing” and “saw-tooth” deformities (17). Hand radiographs of our patient did not reveal any of these appearances.

Other diseases in which it is possible to find interphalangeal joints involvement resembling gouty arthritis of the hands include hypothyroidism, hyperparathyroidism, chronic renal failure, and frostbite. In our patient, all of these were excluded by clinical and laboratory findings.

Standard management of acute gouty arthritis consists of rest, application of ice to the affected joint, and prescription of colchicine, NSAIDs, or both (18). Prednisolone shows the same efficacy as NSAIDs for management of acute gouty arthritis and it may be used instead of colchicine and NSAIDs (19). Anakinra, an antagonist of interleukin-1β, seems to be effective for acute gouty arthritis and could become an alternative for patients in whom NSAIDs, colchicine, or corticosteroids are contraindicated (20). Gout is not always a progressive disease, thus urate-lowering therapy is not recommended after one acute attack. Urate-lowering therapy is indicated for patients with recurrent gout attacks, chronic arthropathy, tophi, and gout with uric acid stones (18). Prevention of acute flares, which can be induced by dissolution of intra-articular crystal deposition, is advised during the first 3-6 months of urate-lowering therapy and can be achieved with colchicine 1 mg per day, or small doses of NSAIDs (2). Allopurinol lowers uricaemia through inhibition of xanthine oxidase activity, and is used as first-line urate-lowering therapy. Febuxostat is a novel xanthine oxidase inhibitor approved for management of gout in patients with allopurinol intolerance (21). Uricosuric agents (probenecid, sulfinpyrazone, benzbromarone) can also be used as second-line therapy for patients with underexcretion of uric acid (2). Inevitably, every patient should be informed about the disease, its curable nature, the targets of drug therapy, how to prevent and handle flares, and the importance of lifestyle and dietary factors.