

# Coexisting Ankylosing Spondylitis and Gouty Arthritis

## Ankilozan Spondilit ve Gut Artriti Birlikteliği

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### Summary

Coexisting ankylosing spondylitis (AS) and gouty arthritis have rarely been reported in the literature previously. We present a 43-year-old male patient with AS who had been diagnosed as having gout based on some clinical and laboratory findings. In this paper, the clinical features of both rheumatic diseases are discussed in light of the recent literature. The gouty arthritis should be considered in the differential diagnosis of acute peripheral arthritis in patients with AS. *Türk J Phys Med Rehab 2011;57:111-3.*

**Key Words:** Anklosing spondylitis, gouty arthritis

### Özet

Ankilozan spondilit (AS) ve gut artriti birlikteliği daha önce literatürde nadiren bildirilmiştir. Biz, klinik ve laboratuvar bulguları ışığında gut tanısı konulan, AS'li 43 yaşındaki erkek hastayı sunduk. Bu yazıda bu iki romatizmal hastalığın klinik özellikleri güncel literatürler ışığında tartışılmıştır. Gut artriti, AS'li hastalarda akut periferik artrit için ayırıcı tanıda göz önünde bulundurulmalıdır. *Türk Fiz Tıp Rehab Derg 2011;57:111-3.*

**Anahtar Kelimeler:** Ankilozan spondilit, gut artriti

### Introduction

Gout and ankylosing spondylitis (AS) are both common rheumatic diseases. In addition, gout is a relatively frequent disease and it seems likely that it would occur in some patients with rheumatologic disease including AS. In fact, gouty arthritis is uncommon in systemic lupus erythematosus, systemic scleroderma and rheumatoid arthritis (RA) (1-5). Moreover, coexistent gouty arthritis and AS have rarely been reported since Wong's report (6). To our knowledge, only four articles of AS coexisting with gout have been published in the literature (6-9). It prompts us to report another case of gouty arthritis in a patient with AS. In this paper, we present a patient with AS who had been diagnosed to have gouty arthritis according to some clinical and laboratory findings.

### Case

A 43-year-old Caucasian man has had axial AS for 9 years. He had typical advanced radiological changes with ankylosis of his

spine and sacroiliac joints (Figure 1). The patient had limited range of motion of the cervical and lumbar spine and restricted movement of the chest wall. He had no history of peripheral joint involvement, uveitis, other extraarticular features, or trauma. He had used several non-steroidal anti-inflammatory drugs (NSAIDs) intermittently and sulphasalazine 2 g/day for pain and disease control for many years. Symptoms had been partially relieved. During the course of disease, acute-phase reactants (erythrocyte sedimentation rate -ESR, C-reactive protein) were in normal limits with NSAIDs and sulphasalazine therapy.

In December 2007, he was admitted to our hospital, complaining of pain and swelling in his right great toe which started suddenly at night and woke him up. He had no history of medication except for NSAIDs and sulfasalazine. He had no family history for hyperuricemia, gout or AS. On questioning, he described protein-rich diet regimen and he had no alcohol consumption. On physical examination at admission, the patient had painful swelling, warmth and dusky redness in the right first metatarsophalangeal (MTP) joint. Painful swelling of the right

ankle was also noted. His cardiovascular, pulmonary and abdominal examinations were normal. The chin to manubrium sternal distance was measured as 5 cm, occiput to wall distance as 9 cm, chest expansion as 2 cm, hand to floor distance as 5 cm, dorsal Schober as 1 cm, and the modified lumbar Schober as 3 cm. Enthesopathic points were not sensitive to palpation. No extraarticular involvement was detected. Patient's body mass index was 22.7 kg/m<sup>2</sup>.

Laboratory data included ESR 68 mm/hr (1-20), white blood cell count 8.9 K/uL (4.0-11.0), C-reactive protein 28.2 mg/dL (0.00-0.50), uric acid 7.8 mg/dL (2.3-7.5), creatinine 0.9 (0.4-1.3) mg/dL and HLA-B27 positivity. Cholesterol and triglyceride levels were in normal range. The rest of his laboratory values were within normal limits. Soft tissue abnormalities were seen on X-ray (Figure 2). In addition, an attempt to aspirate the right ankle joint failed to yield any fluid. The venous Doppler ultrasonography of the right lower extremity was performed for differential diagnosis, the result of which was normal. Although monosodium urate (MSU) crystals in synovial fluid were not observed under polarizing microscope, the typical clinical and laboratory findings supported a diagnosis of acute gouty arthritis according to preliminary criteria for diagnosis of acute gout (10).

The patient was treated for acute gouty arthritis with oral colchicine 4 g/day and indomethacin 100 mg/day. Dietary regimen including a low-purine diet and maintaining adequate fluid intake and rest of the right lower extremity were added to the treatment. Clinical symptoms of acute gouty arthritis relieved in the first week of the treatment. Colchicine therapy was continued with a dose of 1mg/day for prophylaxis. Physical modalities were applied to the cervical and lumbar regions and an exercise program was also administered. During 12-month follow-up, AS was in remission and gout attacks have not been repeated. There were no complications related to the use of medication.

## Discussion

AS is a chronic inflammatory disease of the spine and sacroiliac joints affecting primarily young individuals. Peripheral joints can become involved and a number of extraarticular manifestations can occur. Genetic factors are thought to play a significant role in the development of AS (11). On the other hand, gout is a MSU crystal deposite disease with characteristic clinical manifestations. Peripheral joints, particularly those of the lower



Figure 1. Bilateral grade 4 sacroiliitis.

extremities, are affected. In men, the initial episode is usually monoarticular. The first MTP joint is initially involved in approximately half of all men with gout. Very rarely, the spine and sacroiliac joints are affected (7,12,13).

The coincidence of gouty arthritis and other rheumatic diseases is rare. Coexisting gout and RA is relatively uncommon (2,4). Gout, occasionally occurs in patients with systemic lupus erythematosus (1,5) and systemic scleroderma (3); however, the incidence is unknown. AS and gout are two distinct rheumatic diseases that can occur concurrently. To our knowledge, only four articles of AS coexisting with gouty arthritis have been reported in the literature (6-9). These few cases suggest a negative association between these two diseases (6-8). However, in contrast to this hypothesis, it has been assumed in a study (9) that the coexistence of AS and gout is more common than previously believed. Gout and AS share a few clinical characteristics, including male predominance, genetic predisposition, familial aggregations, predisposition to involvement of the joints and entheses of lower limbs, excellent response to some NSAIDs such as indomethacin, and frequent development of renal disorders (11,12,14-16).

Involvement of foot or tarsal joint has been reported; however, first MTP joint involvement has not been mentioned in AS. Therefore, gout also should be included in differential diagnoses, especially when the first MTP joint is affected. In Ho et al.'s (9) series, it was found that 61.5% of subjects had first MTP joint involvement. During long-term follow-up for axial AS, our patient was also diagnosed as having gouty arthritis due to acute first MTP joint involvement. Differentiating peripheral arthritis or enthesopathies of AS from gouty arthritis and from gout affecting tendons or ligamentous insertions is occasionally difficult, especially, when the course of AS arthritis becomes acute or the



Figure 2. No abnormality except soft tissue swelling.

course of gout becomes chronic (9,17). In addition, in the differential diagnosis of the first MTP joint arthritis (podagra), inflammatory arthritis such as RA, gout, pseudogout, etc., septic arthritis and degenerative arthritis (osteoarthritis, hallux valgus, hallux rigidus) should be taken into account (13). In our patient, we ruled out these diseases regarding the clinical findings such as male gender, age over 40 years old, unilateral first MTP joint involvement, sudden onset of symptoms especially at night, redness observed over joints, laboratory findings including elevated serum uric acid level and acute-phase reactants, and radiological findings.

Clinical, radiological and laboratory criteria are helpful in the absence of MSU crystals in the aspirated joint fluid or tophus. The slow onset of symptoms in an arthritis case excludes the diagnosis of gout. In addition, the severity of pain can also be an indicator, as pain of gouty arthritis is described as overwhelming. Redness, warmth and swelling accompany the pain in gout (13). Also, our patient had painful swelling, warmth and dusky redness in the first MTP joint, which all lead to the diagnosis of gouty arthritis (10). During the 12-month follow-up, gouty attacks have not been repeated in our case due to early diagnosis, colchicine prophylaxis and compliance to dietary regimen.

Several medications such as diuretics, low-dose aspirin, and anti-tuberculosis drugs have been associated with the development of hyperuricemia and gout. These drugs can impair uric acid excretion (9). Sulphasalazine has been reported to ameliorate AS activity, particularly improving the peripheral arthritis symptoms (18,19) but it has not been mentioned to be associated with hyperuricemia. In order to investigate whether such relationship exists, the pharmacokinetics of sulphasalazine was explored. It is formed by sulphapyridine, which is readily absorbed, and 5-aminosalicylic acid (5-ASA), of which only 25% is absorbed and then excreted in urine as acetyl-5-ASA (9,17). As is known, salicylic acid is uricosuric at doses over 4-5 g/day, whereas lower doses can cause hyperuricemia via urate retention (20,21). To obtain clear and conclusive information about effects of sulphasalazine on urate excretion, further research is necessary. In our patient, sulphasalazine was prescribed at a daily dose of 2 g, and the dosage was not changed after the diagnosis of gout was established. Our review of the literature showed that all reports of gouty arthritis accompanying AS had successfully been treated by colchicines, as in our case (6-8).

Since no underlying disease can be identified in the majority of cases, secondary gout is quite rare (13). To our knowledge, only one report has been published of a patient with AS who also had primary gout (7). In our patient, the diagnosis of secondary gout was ruled out as in that previous report. The other cases of coexistent gout and AS were of secondary gouty arthritis (6,8,9). In a report published in 1994, a case was described of a 71-year-old male patient with AS in whom episodes of gouty arthritis started to occur during chronic renal insufficiency. This patient, who suffered from long-standing AS, developed oligoarthritis affecting his left first MTP joint and right knee (6). Another paper describes the case of a patient with AS who experienced episodes of gouty arthritis and chondrocalcinosis articularis. In this report, gouty arthritis was confirmed by the presence of MSU crystals in the synovial effusion obtained from the right knee and Baker's cyst, and by the extremely high level of urates, indicating hyperuricemia (8). In Ho et al.'s (9) series, it was found that 61.5% of subjects had

first MTP joint involvement, 27.7% had chronic renal insufficiency, 89.2% were hyperuricemic at onset of acute peripheral arthritis, and 93.9% were positive for HLA-B27 antigen. In another case, which reported coexistence of AS and primary gout, HLA-B27 positivity, elevated C-reactive protein and borderline values of the uric acid had been found (7). Similarly, our HLA-B27-positive patient with AS also had hyperuricemia and elevated acute-phase reactants.

We suggest that although the coincidence of gout and AS is rare, the gouty arthritis should be considered in the differential diagnosis of acute peripheral arthritis in patients with AS. Early diagnosis and proper treatment can prevent further complication.

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