

Eklem Dekstrüksiyonları Olan Son Evre Bir Gut Artriti Vakası

A Case Of Final Stage Gout Arthritis With Joint Destruction

Öz

Gut, antik çağlardan bu yana bilinen ve "kralların hastalığı" veya "zengin insanların hastalığı" olarak da isimlendirilen bir durumdur. Eklemde ve yumuşak dokuda monosodyum ürat kristallerinin birikmesiyle oluşur. Diyet, obezite, alkol tüketimi, ilaçlar, genetik bilinen başlıca hazırlayıcı nedenlerdir. Orta yaşlı erkeklerde ve menopoz sonrası kadınlarda sık görülür. Tedavinin tam olarak uygulanmadığı durumlarda, tofus, eklem hasarı, böbrek taşları, sinir sistemi patolojisi ve son dönem böbrek yetmezliği gelişebilir. Tofus tüm eklemlerde, özellikle el ve ayak eklemlerinde görülebilir. Özellikle tedavide geç kalındığında veya bu hastadaki gibi ilaç tedavisine uyumun sağlanamadığı durumlarda, vücudun atipik bölgelerinin (olekranon, aşil) biriktiği ve eklem deformasyonları yapabileceği unutulmamalıdır.

Abstract

Gout is a condition even known from ancient times and also called as "the disease of kings" or "rich man's disease". It is formed by the accumulation of monosodium urate crystals in the joints and soft tissue. Diet, obesity, alcohol consumption, drugs, genetics are major known predisposing causes. It is common in middle-aged men and postmenopausal women. In cases which the treatment cannot be performed completely, even tophus, joint damage, kidney stones, nervous system pathology and end-stage renal failure may develop. Tophus can be seen in all joints, but especially in the hand and foot joints. It should be kept in mind that especially in cases who is late for the treatment or when compliance with drug treatment cannot be achieved, as in this patient, atypical areas of the body (olecranon, achilles) may accumulate and perform joint deformations.

Introduction

It is one of the most common causes of inflammatory arthritis in the world. It occurs when monosodium urate crystals exceed insoluble amounts in blood ($\geq 408 \mu\text{mol} / \text{L}$: 6-8 mg / dl) and accumulate (1). in joints and soft tissue. The accumulated crystals initiate the inflammatory process. By the time, these deposits form lesions called the tophus. Its prevalence is increasing (2). Mostly the cause of uric acid synthesis and excretion impairment is obesity, excessive uric acid intake, alcohol, use of drugs that affect

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uric acid excretion especially thiazide diuretics, myeloproliferative diseases and at a much lower rate, genetic reasons may be responsible (3). It has different prevalence in various countries. The most important affecting factors are; environmental factors, obesity and genetics (4). The incidence in the UK is between 2.5-3 per 1000 person (5). It is more common especially in middle-aged men (6). It is generally observed in males between the ages of 30-60, but it has been observed that the elevation of uric acid levels start during puberty (7). The incidence of the disease is increased in postmenopausal women (8). The prevalence of gout increases due to increased metabolic syndrome in the elderly, chronic heart failure, increased use of diuretics in the treatment of hypertension and the use of low-dose aspirin (9). Hyperuricemia progresses and gout occurs in 4 stages; hyperuricemia and MSU deposits without gout, crystal storage without gout symptoms, acute gout exacerbation with crystal storage, and lastly, final stage gout with characteristic tophus, chronic gout arthritis and radiological erosions (10). Success in gout treatment depends on educating the patient about gout and, his active participation to the treatment. In the acute period: rest, cold administration, nonsteroidal anti-inflammatory agents, colchicine and steroids are administered, but the efficacy of treatment is associated with early treatment (11). Treatment of gout and acute exacerbation is still insufficient (12). EULAR 2016 is recommended for the treatment of gout. Treatment options include drugs that lower uric acid levels (xanthine oxidase inhibitors; Allopurinol, Febuxostat), or increase uric acid excretion (probenecid, lecinurad, benzbromarone) and drugs that convert urate to more easily soluble allantoin (Pegloticase) in the blood (13,14).

Case

Our 56-year-old male patient had complaints of pain, swelling, fever, redness and inability to step, on his right toe which started 34 years ago. The doctors gave him simple painkillers. Our patient applied to people who use traditional medicine during these episodes, since the pain and swelling did not disappear. When pain was relieved after painful periods, he did not look for a treatment. As he continued to consume alcohol and feed on a diet rich in red meat, attacks repeat and swelling began to form in his aching areas. 10-12 years ago, he consulted to another doctor in a nearby city at the time of the attack and he was diagnosed with Gout. Colchicine and allopurinol were recommended. Allopurinol was cut off when he developed allergic complaints. He didn't use colchicine regularly. Since our patient did not pay attention to his alcohol consumption and diet, these pain and swelling spread to his hands and body. He had complaints of stiffness and pain in his joints. Our patient, who had been receiving antihypertensive treatment for about 14 years, was taking Lercanidipine 20 mg / day. In addition, stent was implanted in 4 coronary arteries 5 years ago

due to AMI. There was no other known illness. In the family history; his uncle had similar complaints and swelling on his body. He passed away without any diagnosis and treatment.

In the physical examination, his blood pressure was 135/85 mm/Hg, BMI was calculated as 24.7 kg / m², and there were diffuse tophuses in his body. Especially there were deformed tophus in her feet and hands. In addition, there were tophuses in the olecranon and achilles tendon which we are not used to see (Figures 1-99 are both visual and comparative x-rays).

Laboratory findings were: Leukocyte; $6.7 \times 10^9/L$, Hemoglobin: 15.93 g/dl; Platelet: 194.000/mm³; Uric acid 9.2 mg/dl; CRP: 30.6/dl; Alanintransaminaz: 13I/L; Aspartatransaminase: 22.9 IU/L; Cholesterol: 244 mg/dl; LDL Cholesterol: 126 mg/dl; Uric acid: 9.2 mg/dl (laboratory normal value 3.5-7.2 mg/dl) Fasting blood sugar: 100 mg/dl; HbA1c: 5.78. Cord vocals of our patient were evaluated. (Picture 10). Cord vocals were free, moving. Urine tests were natural. Increased soft tissue density (probably due to MSU accumulation), over-hanging margins of the tophuses and joints were observed in joint films. Statin was started because of coronary risk. In addition, he was referred to a rheumatologist for planning alternative therapies due to his uric colysis hypersensitivity.

Discussion

Gout may occur due to a purine metabolic disorder or a purine-rich diet, as well as hypertension, obesity, metabolic syndrome, chronic renal failure, alcoholism, and certain medications (thiazide diuretics, low-dose aspirin, cyclosporine, pyrazinamide). MSU crystals can accumulate in joints and soft tissue to form tophuses. Gout also increases the risk of ischemic heart disease independent of other factors. It has also been suggested that there may be inflammatory processes and immobilization or other mechanisms involved (15).

In this case, it is important he was late in diagnosis and treatment. However, the lack of patient compliance in treatment is another condition that affects poor prognosis. In our case, olecranon, achilles, hands and feet are important in terms of changes due to accumulation of severe tophuses. Nowadays, it is an important case that it is very difficult to come across a patient with gout who may have joint deformity and loss of function. His treatment with Allopurinol was discontinued because of skin symptoms such as rash, itching and redness. Skin manifestations have been found to be 2% in allopurinol treatment in approximately 3 weeks, requiring discontinuation of treatment, because fatal side effects may occur if taken again (16,17). The disease progressed due to the inability to adapt to treatment and diet. Febuxostat treatment was not considered because of coronary ischemic heart disease.

Picture 1: Gout tophuses and joint deformation



Picture 2: Bilateral Hand X-rays



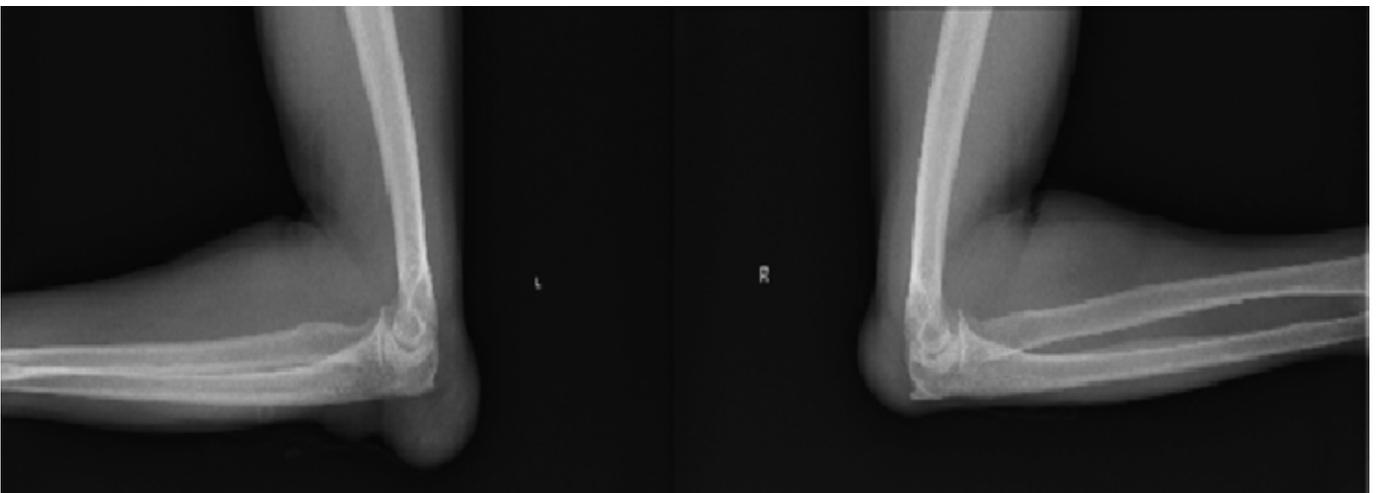
Picture 3: Tophuses on Olecranon



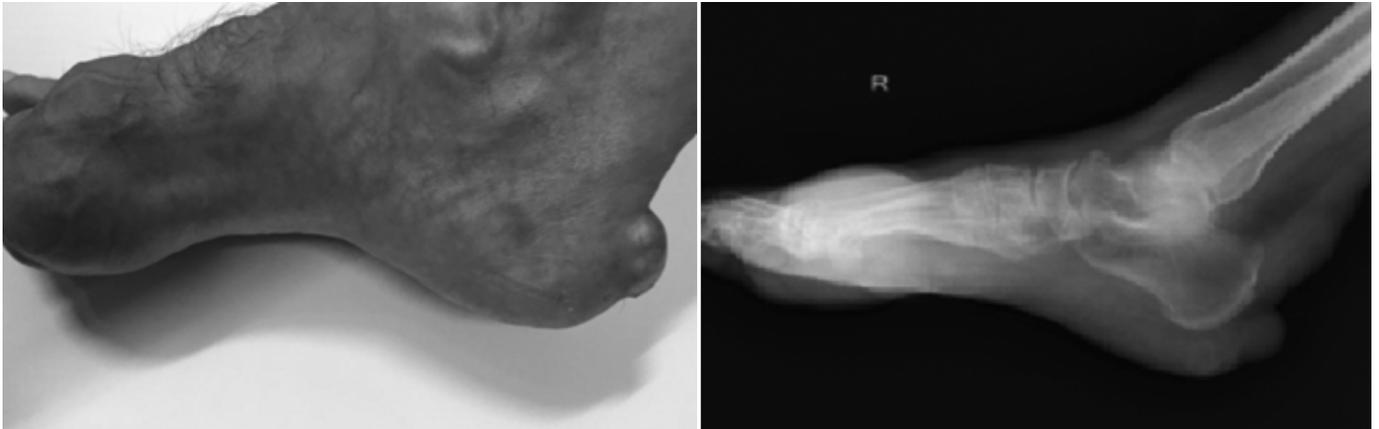
Picture 4: X-ray of the forearm and elbow



Picture 5: X-ray of the elbow joint



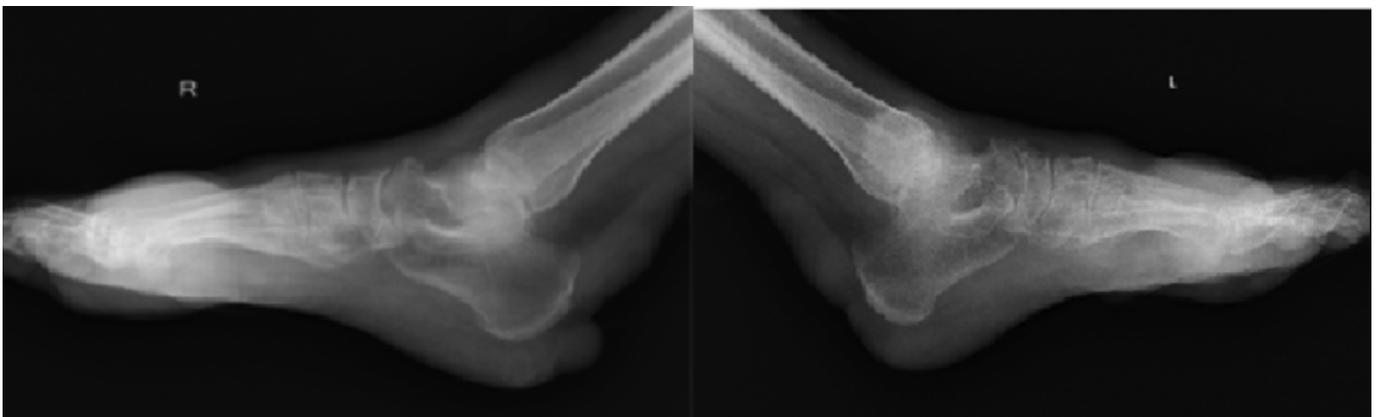
Picture 6: Radiograph of lateral foot and Tophus in Achilles tendon



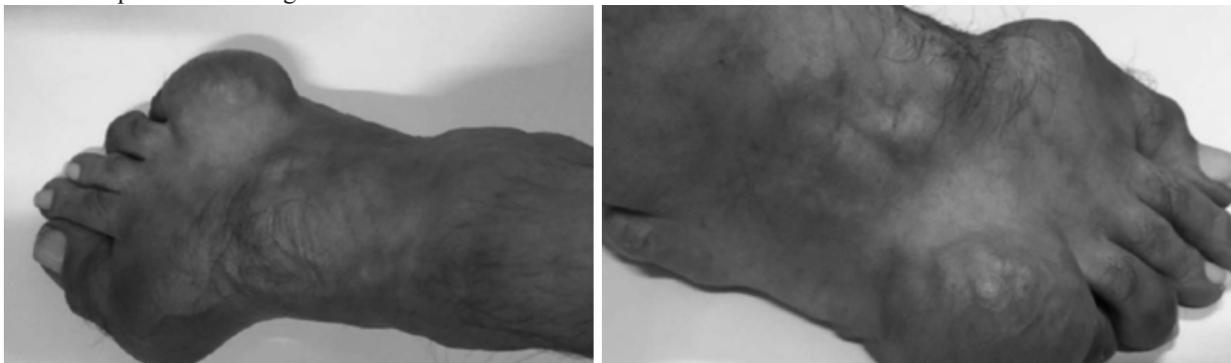
Picture 7: Tophuses and foot radiography of bilateral foot joints



Picture 8: Radiographs of the lateral legs and increase in radiography brightness at Achilles tendon and soft tissue due to MSU deposit.



Picture 9: Tophuses on the right foot malle



Picture 10: Cord vocals



Medical treatment alone is not sufficient in the treatment of gout. Patient participation should also be ensured. In other words, the patient should be fully informed about the disease and its causes and should be actively involved in the treatment. Otherwise, our chances of success are low. In case such patients are detected, they should be called to the outpatient clinic and their controls should be done frequently. Although our patient has coronary heart disease, he should not receive statin treatment, and new gout drugs should be actively tried in the treatment. However, the use of statin and colchicine should be carefully monitored for potential neurotoxic and muscular toxicity (13).

Conclusion

Gout Treatment is made by using medicines in combination with diet and regular control of complications. The most crucial point in the treatment is the awareness of the patient about his disease. In other words, the patient's education is important due to the fact that he knows the complications that may develop and does not interrupt his diet and treatment even during the periods of well-being. Regular doctor checks are also among the most important factors in completing the treatment.

The fact that, we lost time until the diagnosis made in our case and the treatment could not be regulated due to drug allergy after the diagnosis which caused the complications to be more severe. If a good dialogue was established and the patient was called regularly, perhaps severe complications would have been prevented. After the patient was diagnosed, he did not go to the doctor's controls when his acute painful seizures passed, and his alcohol and protein-rich diet also contributed to the progress of the complications.

In summary, the patient and physician should always establish the best dialogue and contribute to the awareness of the patient about his illness. Otherwise, complications

will develop faster and treatment will be much more difficult and complicated.

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