Case Report

Intraarticular Tophi in a Joint Without a Previous Gouty Attack

KUANG-HUI YU

ABSTRACT. Subcutaneous tophi are usually a late clinical manifestation of gout. However, intraarticular tophi may develop very early, since crystal shedding has been presumed to precipitate an acute gouty attack. There is little direct evidence of intraarticular tophi before the initial gouty attack. We describe a patient who had gout for 3 years without subcutaneous tophi. Whitish intraarticular deposits, presumably representing urate tophi, were noted during right knee arthroscopy for a posterior cruciate ligament tear. This observation illustrates that tophi deposition may occur early, even in previously unaffected joints. (J Rheumatol 2003;30:1868–70)

Key Indexing Terms:
GOUT ARTHROSCOPE TOPHI INTRAARTICULAR

Gout is a disease of purine metabolism or renal excretion of uric acid. It is considered to have 4 different phases characterized by asymptomatic hyperuricemia, recurrent attacks of acute arthritis, intercritical gout, and chronic tophaceous gout[1,2]. Subcutaneous tophi are usually a late clinical manifestation. Subcutaneous tophi developing before gouty attack is very rare, but has been reported[3-5]. Intraarticular tophi are instead pathogenetically present before a gouty attack, which is induced by crystal shedding. Intraarticular tophi in previously unaffected knee joints have been reported in patients with long history of gout[6] and with subcutaneous tophi[7]. This article describes a patient with a 3 year history of gout who did not have subcutaneous tophi, but in whom whitish intraarticular deposits, presumably representing urate tophi, were noted during right knee arthroscopy for posterior cruciate ligament tear. The knee joint had no previous gouty attack.

CASE REPORT

A 20-year-old male patient was involved in a traffic accident in June 1999 that resulted in a right tibia-fibular fracture and right hip fracture-dislocation. The fractures were treated surgically with a favorable outcome. But he subsequently experienced weakness of the right leg after he started ambulation, especially when going up or down stairs. There was no acute inflammation episode of the knee joint. Positive posterior draw test was demonstrated on examination and he received an arthroscopic examination in October 2000. Posterior cruciate ligament rupture was found and reconstruction was performed. The arthroscopic examination incidentally found multiple whitish, small, discrete deposits of tophi over the synovial and cartilaginous surface (Figure 1). The radiograph of the right knee showed no calcification.

He had had gouty arthritis for 3 years, but had never experienced a knee joint attack. Initially the attacks involved the first metatarsophalangeal joint, and later involved either the right or the left ankle joint. Attacks usually subsided within a couple of days. Synovial fluid aspirates from the inflamed joint revealed intracellular monosodium urate crystal. Laboratory examination revealed serum urate 11.0 mg/dl and creatinine 1.0 mg/dl, while urinary uric acid excretion was 825 mg/day. There were no subcutaneous tophi, nor was there a history of urolithiasis. The patient's father also had gout, suffering his first attack at 48 years. The patient received allopurinol treatment and had no further gouty attacks or knee symptoms and arthroscopy was not repeated.

DISCUSSION

Gout is a clinical syndrome resulting from urate crystal deposition, and tophi may appear in subcutaneous tissue or within the joint. Subcutaneous tophi usually are a late manifestation[8]. Hench reported that the interval from the first gouty attack to the beginning of chronic arthritis or visible tophi was 3 to 42 years, with an average of 11.6 years[9]. However, synovial tophi have been observed microscopically early during the first gouty attack[10].

The events leading to the initial crystallization of monosodium urate in a joint after many years of asymptomatic hyperuricemia are not completely understood. It has been proposed that gouty attacks are initialized by de novo urate crystallization or shedding of the preformed urate crystal from synovium or cartilage into the joint space, causing a foreign body inflammatory reaction[11,12]. It is difficult to prove de novo crystallization in the joint space, but indirect evidence of crystal shedding has been reported. Pascual found that 36 of 37 synovial fluid aspirates obtained during intercritical periods yielded urate crystals if the aspirated knee had previously been inflamed. In comparison, the yield was only 22% if there was no history of prior acute attack in the aspirated knee[13]. Rouault, *et al*[14], Agudelo, *et*

From the Division of Rheumatology, Allergy, and Immunology, Chang Gung Memorial Hospital, Tao-Yuan, Taiwan.

K.H. Yu, MD, Attending Physician.

Address reprint requests to Dr. K.H. Yu, Division of Rheumatology, Allergy, and Immunology, Chang Gung Memorial Hospital, 5 Fu-Shin Street, Kuei-Shan, Tao-Yuan, Taiwan. E-mail: gout@adm.cgmh.org.tw

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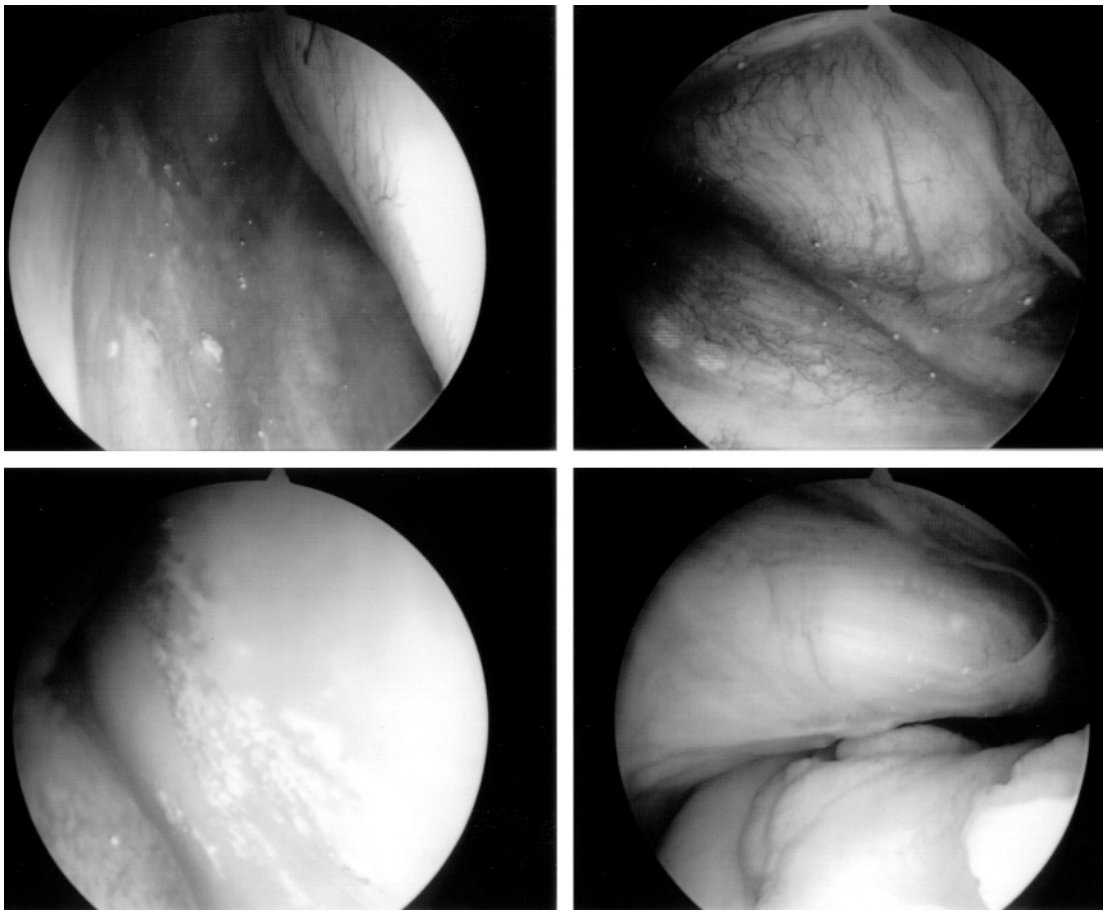

Figure 1. Multiple intraarticular small, whitish, discrete deposits of tophi over the synovial and cartilaginous surface of right knee joint.

al[15], and Bomalaski, *et al*[16] also found monosodium urate crystal in asymptomatic joints of patients with gout. Further, synovial urate microtophi have been observed in asymptomatic joints of patients with long history of gout[6,7]. These findings corroborate that clinically inapparent crystal aggregates are likely to be present in most gout patients by the first attack of gouty arthritis.

The differential diagnosis of whitish intraarticular crystal deposits includes urate (tophi), calcium pyrophosphate, dicalcium phosphate dihydrate, hydroxyapatite, octacalcium phosphate, tricalcium phosphate, and oxalate. We found no calcification on radiograph of the right knee. Unfortunately, it was not possible in this case to confirm the presence of urate crystals in the whitish intraarticular deposits through crystal analysis of arthroscopic scrapings from the deposits or in the return washout fluid from the right knee.

This case demonstrated that whitish intraarticular deposits, presumably urate tophi, may rarely occur in seemingly unaffected knee joints of patients with interval gout. This observation illustrates that tophi deposition may occur very early and supports the theory of crystal shedding in the pathogenesis of gouty attacks.

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