

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 reported³⁻⁵. 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 gout⁶ and with subcutaneous tophi⁷. 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 manifestation⁸. 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 years⁹. However, synovial tophi have been observed microscopically early during the first gouty attack¹⁰.

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 knee¹³. 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, Kwei-Shan, Tao-Yuan, Taiwan. E-mail: gout@adm.cgmh.org.tw

Submitted April 17, 2002; revision accepted January 8, 2003.

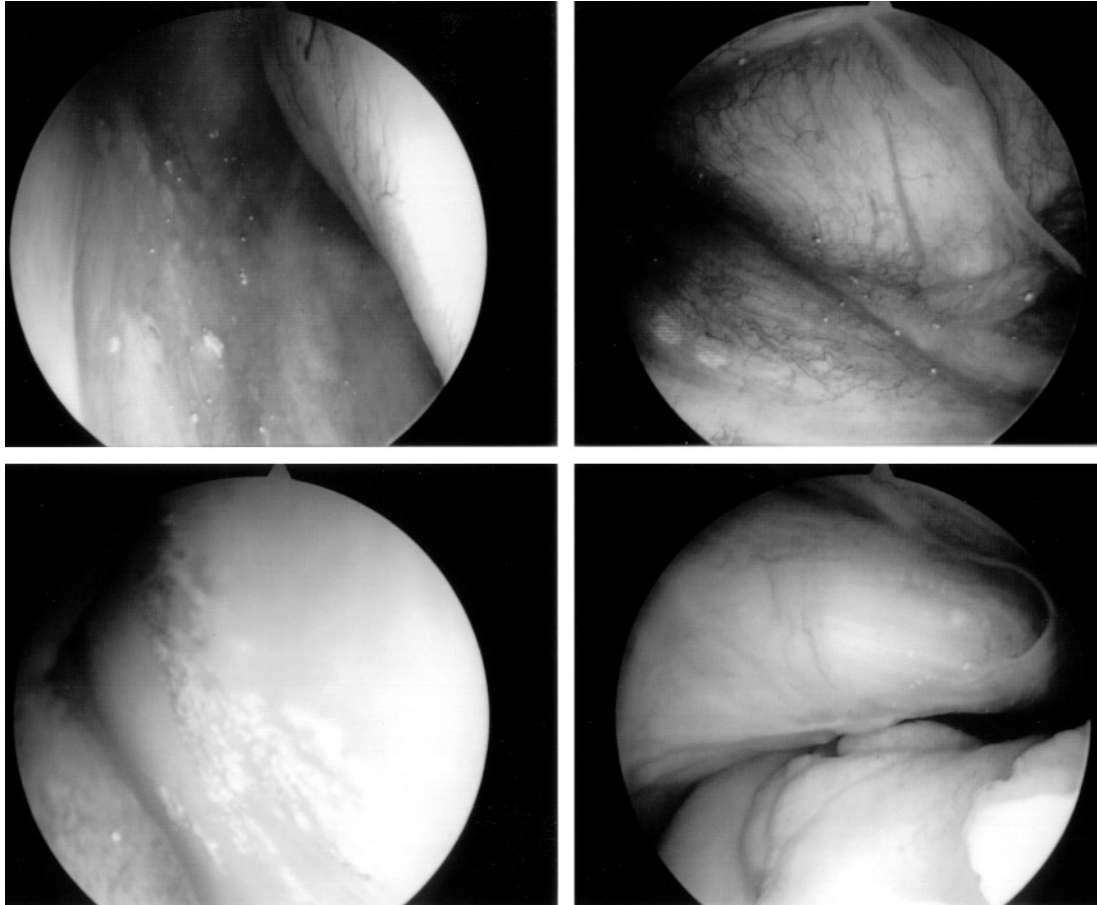


Figure 1. Multiple intraarticular small, whitish, discrete deposits of tophi over the synovial and cartilaginous surface of right knee joint.

*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.

REFERENCES

1. Becker MA, Levinson DJ. Clinical gout and the pathogenesis of hyperuricemia. In: Koopman WJ, editor. Arthritis and allied conditions. 13th ed. Philadelphia: Williams & Wilkins; 1997:2041-71.
2. Kelley WN, Wortmann RL. Gout and hyperuricemia. In: Kelley WN, Harris ED, Ruddy S, Sledge CB, editors. Textbook of rheumatology. 5th ed. Philadelphia: W.B. Saunders; 1997:1313-51.
3. Hollingworth P, Scott JT, Burry HC. Nonarticular gout: hyperuricemia and tophus formation without gouty arthritis. *Arthritis Rheum* 1983;26:98-101.
4. Varga J, Giampaolo C, Goldenberg DL. Tophaceous gout of the spine in a patient with no peripheral tophi: case report and review of the literature. *Arthritis Rheum* 1985;28:1312-5.
5. Shmerling RH, Stern SH, Gravallesse EM, Kantrowitz FG. Tophaceous deposition in the finger pads without gouty arthritis. *Arch Intern Med* 1988;148:1830-2.
6. Kennedy TD, Higgins CS, Woodrow DF, Scott JT. Crystal

- deposition in the knee and great toe joints of asymptomatic gout patients. *J R Soc Med* 1984;77:747-50.
7. Gordon TP, Bertouch JV, Walsh BR, Brooks PM. Monosodium urate crystals in asymptomatic knee joints. *J Rheumatol* 1982;9:967-9.
 8. Gutman AB. The past four decades of progress in the knowledge of gout, with an assessment of the present status. *Arthritis Rheum* 1973;16:431-45.
 9. Hench PS. The diagnosis of gout and gouty arthritis. *J Lab Clin Med* 1936;220:48-55.
 10. Agudelo CA, Schumacher HR Jr. The synovitis of acute gouty arthritis: A light and electron microscopic study. *Hum Pathol* 1973;4:265-79.
 11. Terkelaub R. Pathogenesis and treatment of crystal-induced inflammation. In: Koopman WJ, editor. *Arthritis and allied conditions*. 13th ed. Philadelphia: Williams & Wilkins; 1997:2085-102.
 12. Bennett RM, Lehr JR, McCarty DJ. Crystal shedding and acute pseudogout: An hypothesis based on a therapeutic failure. *Arthritis Rheum* 1976;19:93-7.
 13. Pascual E. Persistence of monosodium urate crystals and low-grade inflammation in the synovial fluid of patients with untreated gout. *Arthritis Rheum* 1991;34:141-5.
 14. Rouault T, Caldwell DS, Holmes EW. Aspiration of the asymptomatic metatarsophalangeal joint in gout patients and hyperuricemic controls. *Arthritis Rheum* 1982;25:209-12.
 15. Agudelo CA, Weinberger A, Schumacher HR Jr, Turner R, Molina J. Definitive diagnosis of gout by identification of urate crystals in asymptomatic metatarsophalangeal joints. *Arthritis Rheum* 1979;22:559-60.
 16. Bomalaski JS, Lluberas G, Schumacher HR Jr. Monosodium urate crystals in the knee joints of patients with asymptomatic nontophaceous gout. *Arthritis Rheum* 1986;29:1480-4.