

Case Report

Recurrence of Gout After Total Knee Arthroplasty

Phil Blyth, BHB, MBChB, and V. S. Pai, MCh(Orth), MS(Orth)

Abstract: The existence of gout in a total knee arthroplasty (TKA) may be more common than is currently recognized. We report 2 cases of acute recurrence of gout in knees after TKA, 1 with coexistent infection. **Key words:** cemented total knee arthroplasty, gout, urate crystals, infection.

Total knee arthroplasty (TKA) is a common operation for all types of arthritis, including chronic gouty arthritis. The ability of gout to mimic infection and a failure to look actively for the presence of urate crystals are 2 possible reasons for the lack of recognition of gout in a TKA. The inflammatory response to the presence of urate crystals may be responsible for some cases of aseptic loosening. To the best of our knowledge, the presence of gout in a TKA has been described only once previously [1]. This article describes 2 cases of the recurrence of gout after TKA: 1 patient with coexistent sepsis and 1 patient who developed a further attack of gout.

Case Report 1

A 58-year-old Maori man with a 30-year history of gout affecting his hands, elbows, and knees had a right TKA (Cemented Miller Galante, Zimmer) in May 1996. Four months later, he experienced increasing knee pain and had difficulty walking. He assumed it was his normal gout and self-prescribed diclofenac 75 mg twice a day and colchicine 0.6 mg

3 times a day. He became increasingly feverish and unwell, however.

On examination, the patient's temperature was 37.9°C, and pulse was 110 beats/min. His knee was hot and red with a tense effusion. Lymphadenopathy was not evident. The range of flexion was between 5° and 30°. Radiographs showed a well-fixed TKA with no evidence of loosening. Laboratory findings included a white blood cell count of $12.8 \times 10^9/L$ with 98% neutrophils, and the erythrocyte sedimentation rate was 88 mm/h. Blood urate level was high at 0.91 mmol/L (normal, 0.2–0.4 mmol/L), and blood cultures grew *Staphylococcus aureus*. A 30-mL knee aspirate of frank pus was positive for urate crystals, and a Gram's stain showed gram-positive cocci, which subsequently grew *S. aureus*. Treatment was started with intravenous flucloxacillin 1 g 4 times per day. An emergency arthroscopic washout was performed with 4 L of normal saline. After this, the patient developed septic shock with acute respiratory failure, high-output renal failure, and metabolic acidosis requiring ventilation and inotropic support.

Despite 2 washouts in the following 4 days, the patient's toxic symptoms deteriorated. The patient was returned to the operating room on the fifth day after admission for incision, drainage, radical debridement with removal of the prosthesis, and

From Hawkes Bay Regional Hospital, Hastings, New Zealand.
Submitted June 29, 1998; accepted September 3, 1998.
Reprint requests: Dr. Phil Blyth, BHB, MBChB, Hawkes Bay Regional Hospital, Private Bag, Hastings, New Zealand.
Copyright © 1999 by Churchill Livingstone®
0883-5403/99/1403-0018\$10.00/0

insertion of an antibiotic-laden polymethylmethacrylate spacer.

For a further 10 days after the removal of the prosthesis, the patient continued to have a fever of greater than 38°C. On weaning sedation, it was found that he had developed a moderately severe sensory and motor peripheral neuropathy, with generalized weakness in both arms and legs and some numbness in both feet, suggestive of critical illness neuropathy.

The patient's recovery was further complicated by an attack of gout with swelling in wrists, knees, and ankles. His trachea was finally extubated on day 76. A knee aspirate after this showed blood clots with urate crystals; no bacteria were seen, and the culture was sterile. Flucloxacillin was changed to dicloxacillin 500 mg 4 times a day orally.

Three months postadmission, the patient was transferred to the rehabilitation unit and was able to attend to simple grooming tasks independently. Further intensive physiotherapy is planned with consideration of revision knee arthroplasty or arthrodesis in 2 months.

Case Report 2

A 52-year-old Maori man with a 20-year history of gout affecting his wrists, elbows, and knees had had an uncemented right TKA (Duracon, Howmedica, USA) for progressive arthritis. The operation had proceeded uneventfully, and postoperatively he was given cefuroxime 750 mg 3 times a day for 48 hours.

On the third postoperative day, the patient spiked a temperature of 38.6°C. On examination, his knee was warm, swollen, erythematous, and tender. A complete blood count showed a white cell count of $8.5 \times 10^9/L$ with 65% neutrophils. Both a urine specimen and a chest radiograph showed no abnormality. A knee aspirate showed red blood cells greater than $1000/mm^3$, occasional polymorphonuclear cells, and no bacteria or subsequent growth. The specimen was not examined for crystals. The patient was recommenced on intravenous cefuroxime 750 mg 3 times a day.

On the fourth day, the patient's general condition deteriorated; his knee became warmer, more swollen, and painful. There was no discharge from the wound, and blood cultures yielded no growth. In addition, he developed painful swelling in his wrist and metacarpophalangeal joints. A second aspiration of the knee joint revealed red cells greater than $1,000/mm^3$ and occasional polymorphonuclear cells, and it was positive for urate crystals. He was started

on naproxen 750 mg immediately, followed by 250 mg 3 times a day and colchicine 1.2 g then 0.6 mg 3 times a day. The following day, his temperature returned to normal. By day 7, the gout in his hand had resolved, and the colchicine was withdrawn. He continued to mobilize and, with all signs of inflammation settling, was discharged.

Five weeks later, the patient was re-referred by his physician with a 1-day history of fever, pain, and swelling. On examination, his temperature was 37.5°C, and his pulse rate was 107 beats/min. His knee had a 15° fixed flexion deformity with further flexion of 80°. The knee joint was hot and erythematous with a moderate effusion. The metatarsophalangeal joints of his right foot were also tender. His leukocyte count was $6.7 \times 10^9/L$ with neutrophils 44%, and his erythrocyte sedimentation rate was 60 mm/h. Aspirate from the knee confirmed a recurrent attack of gout, and subsequent culture was negative. The patient improved with colchicine 1.2 g followed by 0.6 mg 3 times a day and naproxen 250 mg 3 times a day and was commenced on long-term allopurinol 300 mg daily. Twelve months later, he remained almost asymptomatic.

Discussion

The prevalence of gout varies from approximately 0.1% in Europe [2] to 14% in the adult male Maori population of New Zealand [3]. It is well known that the typical acute attack may be precipitated by events such as a surgical procedure [4]. The high incidence of gout in the Maori population may explain the presentation of 2 cases of gout after TKA in our department in a year. Williamson et al. [1] reported a case of acute gouty arthropathy in the knee after a TKA and suggested the importance of differentiating gout from infection. They also suggested that in 5% to 15% crystals may be absent in the synovial fluid. When the synovial fluid is negative, a synovial biopsy may confirm diagnosis.

Case 1 illustrates the importance of keeping the differential diagnosis open because it shows it is possible to have urate crystals in the presence of sepsis. Had the treatment of the patient's infection been delayed any further, there may have been a fatal outcome. It is not clear whether the formation of crystals predisposed the joint to infection or whether the crystals precipitated as a result of a pH change in synovial fluid after infection.

Case 2 suggests that an acute attack of gout can mimic infection, and the diagnosis can be missed unless the synovial fluid is examined for crystals. The attack usually peaks 24 to 48 hours after first

onset, then subsides within 7 to 10 days; this may be similar to the time course of a treated infection.

It is not possible to differentiate accurately between acute gout and infection on clinical grounds alone. Both conditions cause a painful, erythematous, warm, and swollen joint. Biochemical markers, such as a raised erythrocyte sedimentation rate or serum urate, may be found in both instances. Consequently, the diagnosis of gout relies on synovial fluid or tissue examination under a polarized microscope. Because the presence of gout in a TKA is relatively rare, laboratories may not routinely examine the fluid with polarized light unless specifically requested.

We believe that gout in TKAs is more widespread than is currently recognized. There may be many more patients with suspected infections of TKAs that are actually attacks of gout, and some cases of aseptic loosening may be caused by chronic gouty inflammation [5]. The recognition of gout is essential for prolonging prosthetic joint survival in a patient with this condition. We strongly recom-

mend the examination of synovial fluid for urate crystals to avoid missing the diagnosis of gout.

Acknowledgment

The authors are grateful to Mrs. Sue Roberts for her help in preparing the manuscript.

References

1. Williamson SC, Roger DJ, Petrera P: Acute gouty arthropathy after total knee arthroplasty: a case report. *J Bone Joint Surg Am* 76:126, 1994
2. Harris CM, Lloyd DC, Lewis J: The prevalence and prophylaxis of gout in England. *J Clin Epidemiol* 48:1153, 1995
3. Klemp P, Stansfield SA, Castle B, et al: Gout is on the increase in New Zealand. *Ann Rheum Dis* 56:22, 1997
4. Scott JT, Higgins CS: Diuretic induced gout: a multifactorial condition. *Ann Rheum Dis* 51:259, 1992
5. Ortman BL, Pack LL: Aseptic loosening of a total hip prosthesis secondary to tophaceous gout: a case report. *J Bone Joint Surg Am* 69:1096, 1987