

## An Atypical Case of Gout- Induced Arthropathy After a Total Knee Arthroplasty.

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### Abstract

#### Background

Gout is a rare replacement joint that can present as an acutely or chronically painful knee or hip. It can be challenging to differentiate between gout and acute infection of a replaced joint as both presentations are very similar.

**Case description:** A 66-year-old female with a past medical history of hypertension, diabetes, heart failure with reduced ejection fraction (10 to 15 %), chronic obstructive pulmonary disease, and gout presented to the emergency department with right-knee pain for 2 days. She denied any trauma to the knee and reported worsening swelling and tenderness of the knee. She did write an episode of gout in the right toe approximately 6 months prior. She had a bilateral total knee replacement 9 years earlier. Knee arthrocentesis was positive for urate crystals. She was treated with oral glucocorticoids once a septic joint was ruled out. Her symptoms improved, and she was discharged to follow up with her primary care physician.

#### Discussion/Conclusion

Gout can occur in a prosthetic joint. It is rare, and only a handful of published case reports exist for gouty arthritis in a replaced joint; however, the condition may be underreported or misdiagnosed. Gouty arthritis is a manifestation of metabolic disease and can reactivate at any point after surgery. Although rare in a replaced joint, it is essential to rule out gout and test for crystals in a replaced joint presenting with septic arthritis–type symptoms.

**Keywords:** gout, septic arthritis, total knee replacement, arthritis, swelling.

### Introduction

Gout is a joint inflammatory arthropathy that affects more than 8 million Americans and is responsible for approximately 7 million ambulatory visits at an annual cost of \$1 billion [1].

Gout occurs earlier in life in men than in women and rarely occurs in childhood. Its incidence and prevalence appear to be increasing worldwide [2].

While gout is rare in a replaced joint, it can present as an acutely or chronically painful knee or hip [3]. Differentiating gout and acute infection of a replaced joint can be difficult, given that their

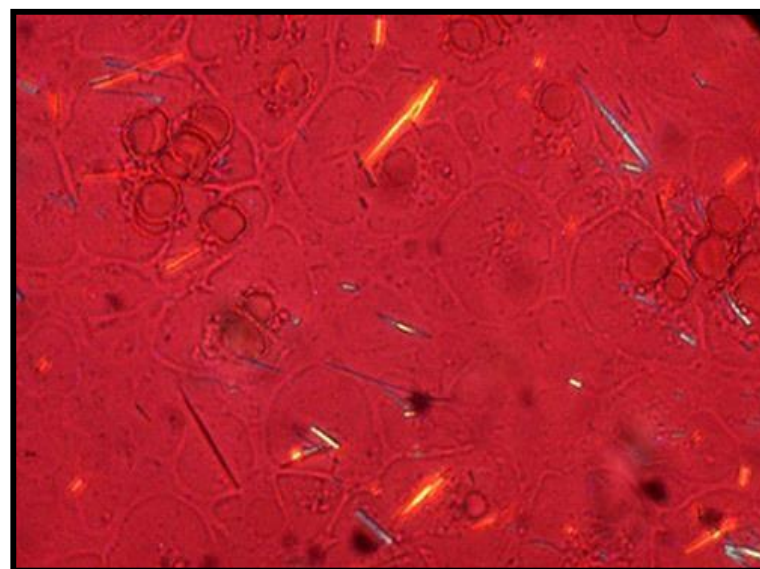
presentations are similar (**Figure 1A, Figure 1B**). A thorough history, physical exam, and laboratory results are essential for a correct diagnosis.

Both gout and joint infection can present with a rapid onset of joint pain, swelling, erythema, and constitutional symptoms, including fever and malaise [4]. Both conditions often have laboratory findings that include an elevated white blood cell (WBC) count, erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP) level.



**Figure 1A.** Knee affected by gout [5].

**Figure 1B.** Knee affected by septic arthritis [6].



**Figure 2.** Synovial fluid from a patient with gout [7].

The polarized light study makes the definitive diagnosis of gout of the synovial fluid showing negatively birefringent, needle-shaped crystals [4] (**Figure 2**).

Gout must be identified correctly to avoid diagnosing a gouty joint as a septic joint, where there may be an indication for operative involvement and possible removal of the common [8].

## Case Description

### Presentation and Medical History.

The patient is a 66-year-old African American female with a medical history of significant hypertension, diabetes, heart failure with an ejection fraction of 10 to 15 %, and chronic obstructive pulmonary disease; she presented to the emergency department with right-knee pain for 2 days. She denied any trauma to the knee, fever, chills, and shortness of breath. She reported progressive worsening of swelling and tenderness of the knee and similar symptoms of swelling, pain, and passion in her right toe approximately 6 months before this episode when she was diagnosed with gout. It is unclear the treatment she received for her initial gout episode. She denied any recent knee

### Physical Examination

The physical exam of the patient's right knee showed significant tenderness on palpation, mild edema, and erythema, as shown in

This corresponding case report is that of a 66-year-old female who presented with right-knee pain with physical findings suggestive of either a septic joint, given her history of bilateral total knee arthroplasty, or the unlikely diagnosis of gout in a replaced joint.

pain before the current outbreak; she had a bilateral total knee replacement 9 years before this episode at age 57 and had been pain-free since that time.

The patient reported current medications of 20 mg of Torsemide and 40 mg of Furosemide daily for heart failure per her primary care physician (PCP). Chart review revealed that she had been switched from 40-mg Furosemide to 20-mg Torsemide, but unfortunately, she had misunderstood the directions provided by her physician and had been taking both diuretics for more than a year week up to her current presentation at the emergency room.

**Figure 3.** She could not bear weight on the knee, and a complete knee exam was limited by pain.



**Figure 3.** Exam findings of the patient upon presentation; warm, swollen, tender, and erythematous area circled in red (photographs taken July 2019 by authors at Emory University Hospital Midtown).

**Radiographic Study:** Plain radiographs of the right knee (**Figure 4**) showed an intact prosthesis with no acute abnormalities or complications.



**Figure 4:** Radiographic findings of the right knee show the intact prosthesis

### Laboratory findings

Laboratory findings for the patient in the emergency department were significant for mild leukocytosis, elevated ESR, troponins, and creatinine (**Table 1**).

**Table 1:** Laboratory findings

|                      |                              |
|----------------------|------------------------------|
| WBC count            | 10.8 × 10 <sup>9</sup> /L    |
| Uric acid            | 12.6 mg/dL                   |
| ESR                  | 110 mm/hr                    |
| Nucleated cell fluid | 19,650 cells/mL              |
| Lymphocytes          | 73 × 10 <sup>9</sup> cells/L |
| Neutrophils          | 13 /mm <sup>3</sup>          |
| Eosinophil           | 0 cells/mcL                  |
| Body fluid           | Cloudy                       |
| Creatinine           | 2.45 mg/dL                   |
| Glucose              | 109 mM                       |



Knee arthrocentesis was completed in the emergency department and at the time of admission for synovial-fluid WBC count, culture, Gram stain, crystals, and uric acid, with the results pending. The differential diagnosis on admission was septic arthritis vs. gout arthropathy. The synovial-fluid analysis ultimately did reveal monosodium birefringent crystals—effectively diagnosing gout.

### Hospital Course.

On admission, the patient presented with an acute kidney injury given her creatinine, which was elevated from baseline (1.14 mg/dL). Because of her acute kidney injury, she was treated with oral glucocorticoids (40-mg Prednisone once daily for 5 days), and nonsteroidal anti-inflammatory drugs (NSAIDs) were held. She also received physical therapy and showed improvement under the treatment plan.

### Discussion

Gout results from the precipitation of monosodium urate crystals in a joint space that triggers immune activation, releasing several inflammatory cytokines and activating neutrophil recruitment [9]. The common area can be irreversibly damaged over time from these

She was discharged on the third day after admission with 2 days of 40 mg of Prednisone to complete her treatment dose. On discharge, she had regained almost full mobility of her knee and near complete resolution of her pain. She was scheduled for a close follow-up with her PCP after discharge.

crystal depositions. There can also be the formation of subcutaneous nodules in the joint space made up of monosodium urate crystals in a matrix of mucopolysaccharides, proteins, and lipids, known as tophi, as shown in **Figure 5**.



**Figure 5.** Large tophus and multiple superficial tophi of the knee in a patient with uncontrolled gout [10].

Gout can also be characterized by extracellular fluid urate saturation reflected in the blood by hyperuricemia with a serum plasma urate greater than 6.8 mg/dL, the approximate limit of urate solubility [11]. Hyperuricemia is necessary but not enough for developing urate crystal deposition disease and should be distinguished from gout, the clinical syndrome.

Most hyperuricemic individuals never experience a clinical event resulting from urate crystal deposition.

**Gout can manifest in various ways:** Gout flares (recurrent flares of inflammatory arthritis), tophaceous deposits, uric acid nephrolithiasis, chronic nephropathy (often due to comorbid states) and chronic arthropathy.

Although rare, gout in a prosthetic joint should be considered when presented with symptoms suggesting septic arthritis. A detailed history is critical in differentiating the conditions.

Septic arthritis is synonymous with an infection in a joint caused by microorganisms, usually bacteria [12]. Most commonly, septic arthritis arises via hematogenous seeding. Bacteremia is more likely

to localize in a joint with pre-existing arthritis (such as rheumatoid arthritis, osteoarthritis, gout, pseudogout, and Charcot arthropathy), particularly if associated with synovitis [13]. Patients with septic arthritis usually present acutely with a single swollen and painful joint (i.e., monoarticular arthritis). Joint pain, swelling, warmth, and restricted movement occur in 80 % of patients with septic arthritis [14].

Septic arthritis occurs because of hematogenous seeding. Blood cultures are positive in approximately 50 % of cases [15]. Laboratory findings such as elevated WBC count, ESR, and CRP are common but nonspecific [16].

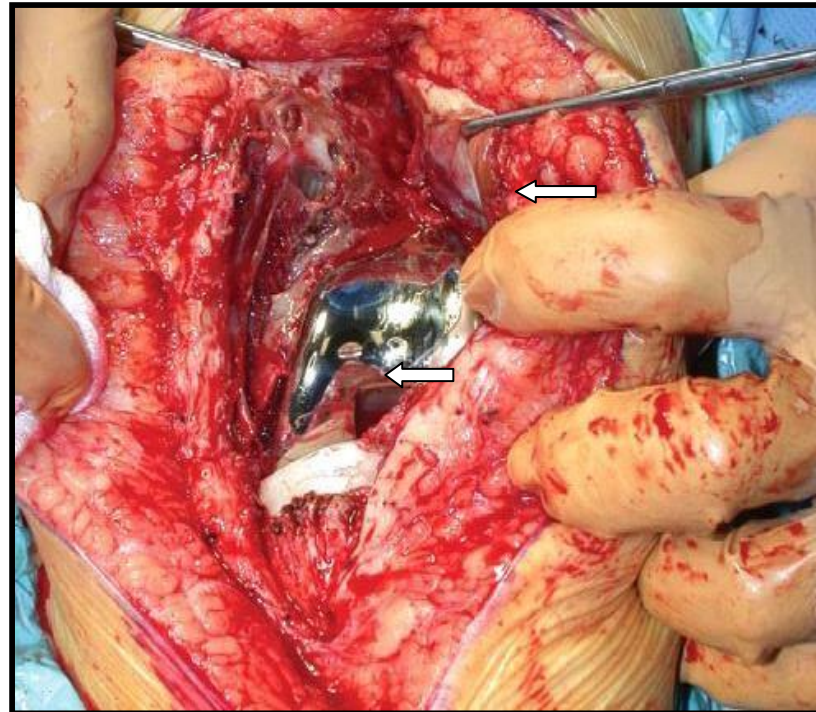
Septic arthritis may be definitively established in positive synovial fluid by Gram stain and culture [17]. In patients with purulent synovial fluid (WBC count of 50,000 to 150,000 cells/microliter, mostly neutrophils) but negative synovial-fluid cultures, a presumptive diagnosis of septic arthritis may be made.

As mentioned above, both conditions can present with a rapid onset of joint swelling, erythema, and pain. Recent infection, dental work, or colonic/genitourinary procedures suggest septic arthritis. At the

same time, the current treatment of malignancy that can lead to tumor lysis syndrome, diuretic therapy, renal insufficiency, hemolytic anemia, asymptomatic hyperuricemia, and initial gout attack increases the risk of gout development in the patient [3].

The presence of needle-shaped, negatively birefringent monosodium urate crystals on polarized light microscopic examination are diagnostic for gout; hence, synovial-fluid analysis is the gold standard for diagnosis.

Salin et al. described a case of a woman in her 50s who had a total knee replacement of the left knee and presented some years later with severe unrelenting pain and a suspicious laboratory profile [18]. Because of a presumptive diagnosis of an acute infection, she underwent an arthrotomy that showed chalky white deposits confirmed to be gout, as shown in (Figure 6) [18].



**Figure 6.** Inflamed synovium is laden with gout crystals at the incision and debridement (arrows pointing to gout crystals) [18].

Diagnosis of gout is usually clinical, based on the rapid development of monoarticular arthritis marked by swelling and redness, with the most involved joint being the first metatarsophalangeal. Other joints affected include the midtarsal joints, ankles, knees, fingers, wrists, and elbows. There can also be deposition of urate crystals throughout the body (vertebrae, skin, soft tissues), mimicking other disease states.

First-line treatment for gout is NSAIDs, with Indomethacin being traditionally primary; however, there has been no evidence that it is more effective than any other NSAID [19]. Any oral NSAID may be given at adequate dosing, titrated as tolerated, and continued for 1 or 2 days after relief of symptoms.

Treatment of acute gout should commence within 24 hours of symptom onset to achieve rapid and complete resolution. Oral corticosteroids, intravenous corticosteroids, and NSAIDs are equally effective in treating acute flares of gout [20].

Another treatment option for acute gout is Colchicine. However, it may be less effective in treating acute flares when given beyond 72 to

96 hours after symptom onset because of its lack of analgesic properties. Common adverse effects include nausea, vomiting, and diarrhea. Colchicine should be used cautiously in patients with hepatic or renal impairment [19].

Certain patients may not be able to tolerate NSAIDs secondary to renal impairment, gastrointestinal dysfunction, or a host of other issues. In those cases, corticosteroids are an appropriate alternative [21].

In some instances, when gout is limited to a single joint, intra-articular corticosteroid injection may be preferable to systemic corticosteroids because of their lower adverse effect profile. Discontinuation of corticosteroid therapy for acute gout may increase the risk of rebound flares. Preventive treatment and initiation of a tapered course of corticosteroids over 10 to 14 days may be considered after the resolution of symptoms to reduce the risk of a rebound flare [22].

## Conclusion

In our case report, the gout episode was likely secondary to an accidental over-diuresis, as the patient had been taking two different loop diuretics for more than 1 week.

Gout can occur in a prosthetic joint, although it is rare, and only a handful of published case reports exist for gouty arthritis in a replaced knee joint; the condition may be underreported or misdiagnosed. This case report highlights the need for an increased index of suspicion in

cases like this to avoid misdiagnosis, which can lead to significant consequences. A misdiagnosis such as septic arthritis could be made, given that signs and symptoms of gout in a replaced joint may suggest septic arthritis. Unnecessary complications could occur with misdiagnosis, potentially including surgery or adverse effects from a broad spectrum of antibiotics.

In this case, the appropriate tests and subsequent management were carried out, leading to effective treatment and eventual resolution of the patient's symptoms.

## Declarations

**Ethics approval and consent to participate:** Not applicable

## Consent for publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

**Availability of data and Material:** Not applicable

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