



A Case of Triple Arthritis: Tuberculosis, MRSA and Gout in One Knee

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Authors' contributions

This work was carried out in collaboration between all authors. Author JT managed the literature searches and wrote the first draft of the manuscript. Authors MJA and AM reviewed the manuscript. Author AM served as an adviser. All authors read and approved the final manuscript.

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Case Study

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ABSTRACT

Aim: To report a case of concurrent tuberculous and bacterial arthritis in chronic tophaceous gout.
Presentation of the Case: A 59-year old Filipino male patient consulted for sensorial changes with four-month progressive swelling of the right knee draining non-foul smelling yellowish discharge with chalky material. He eventually had arthrocentesis of right knee. Blood cultures were all negative. Acid fast bacilli were present both in the synovial fluid and endotracheal aspirate. Endotracheal aspirate grew *Mycobacterium tuberculosis*. Synovial fluid grew methicillin-resistant *Staphylococcus aureus* (MRSA). He was treated with prednisone, clindamycin, colchicine, febuxostat, tramadol, and anti-tuberculous medications. He failed to undergo surgery. Patient eventually had fatal myocardial infarction.
Discussion: To our knowledge, this is the first report of concurrent tuberculous and bacterial (MRSA) arthritis in gout.

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Conclusion: This case highlights the possible differential diagnoses especially infections among patients with chronic tophaceous gout. Concurrent tuberculosis and bacterial arthritis can complicate the management of these patients. Hence, microbiologic and crystal analysis of synovial fluid is a very important part of the work-up to tailor the early, specific treatment for these patients.

Keywords: *Mycobacterium tuberculosis; methicillin-resistant Staphylococcus aureus; septic arthritis; gout; case report.*

1. INTRODUCTION

Gout is a metabolic disease that leads to hyperuricemia which causes precipitation of urate crystals in joints causing severe pain and swelling. Precipitation of urate crystals may also happen in the kidneys thereby causing nephrolithiasis and worse, complicating to chronic kidney disease if left untreated. In chronically untreated or undertreated gouty patients, periarticular tophi can form causing disability and/or disfigurement. These tophi may sometimes rupture expressing chalky white discharge.

Tuberculosis is a major public health concern in the Philippines [1]. The tubercle which can be transferred through cough droplets infects the lungs. TB can also affect the lymph nodes, spine, pleura, genitourinary and gastrointestinal organs. Rarely, it affects the joints [2-4].

Septic arthritis is the purulent infection of the joint causing severe swelling, pain, and disability. It is an important medical emergency with high morbidity and mortality [5]. Septic arthritis is most commonly caused by bacteria and rarely by mycobacteria, viruses, fungi, and parasites. While case reports and series have been published on concomitant TB and gouty arthritis [6-8] or concomitant bacterial and gouty arthritis [9-23], to our knowledge there is no published literature on concomitant tuberculous and bacterial arthritis among patients with gout.

2. REPORT OF A CASE

We describe a 59-year old Filipino male with chronic tophaceous gout diagnosed twelve years prior to consult. He was previously on colchicine, diclofenac, methylprednisolone and allopurinol with gouty attacks about three times a year affecting the knees, elbows, and first metatarsophalangeal joints of feet. He had no previous arthrocentesis done nor intraarticular steroid injections prior to this admission.

He presented with a four-month history of bilateral knee pain, swelling, and warmth with the right knee being worse. He denied any history of trauma to his knees. He took diclofenac with slight relief of symptoms. Left knee improved but right knee increased in swelling and started draining non-foul-smelling yellowish fluid with chalky material. He then developed two-week history of progressive disorientation. On the day of admission, he developed dysgeusia and worsening of sensorium. He had no fever, cough, chest pain, dyspnea, vomiting, or diarrhea. He had a history of passing out stone in the urine. He had no other comorbidities except he was diagnosed with chronic kidney disease stage 5 a month ago and given sodium bicarbonate, febuxostat, colchicine, ferrous sulfate, folic acid, potassium citrate, and amlodipine. He had no known exposure to tuberculosis. He was a previous smoker and alcohol beverage drinker.

On examination, he was drowsy with a blood pressure of 90/60 mmHg, 86 beats/minute of heart rate, respiratory rate 20 cpm, temperature 36.5°C, oxygen saturation 99% on room air. He had anicteric sclerae, no neck vein engorgement, clear breath sounds, distinct heart sounds, no murmur, apex beat at fifth intercostal space midclavicular line, normoactive bowel sounds, nontender and soft abdomen, and no bipedal edema. He had multiple tophi over extremity joints with warm and tender knees. Right knee was very swollen with draining sinus from a fistula. The hemoglobin was 82 g/L (reference range, 120 to 180), hematocrit was 29 (reference range, 40 to 54), platelet count was 420,000 (reference range, 150,000 to 450,000), white-cell count was 1256 (reference range, 4500 to 11,000), with 73% neutrophils (reference range, 50 to 70), 20% lymphocytes (reference range, 20 to 50), and 4% monocytes (reference range, 2 to 9); blood levels of blood urea nitrogen was 24.7 mmol/L (reference range, 2.9 to 9.3), and creatinine was 12.68 mg/dL (reference range, 0.64 to 1.27). Blood cultures were negative. Synovial fluid grew methicillin-resistant *Staphylococcus aureus* (MRSA) sensitive to

clindamycin, erythromycin, linezolid, tetracycline, cotrimoxazole; and tested positive for acid fast bacilli (AFB). Endotracheal aspirates tested positive for AFB with auramine staining with fluorescence microscopy and grew *Mycobacterium tuberculosis*. Urine and stool AFB studies were negative.

He underwent hemodialysis and blood transfusions. He was treated with clindamycin, rifampicin, pyrazinamide, and isoniazid when the synovial fluid analysis came out. Patient was also on colchicine, low-dose prednisone, febuxostat, and tramadol to control gouty attacks. Eleven days after the diagnosis of bacteriologically confirmed TB arthritis, patient demised from myocardial infarction which precipitously presented with sudden dyspnea, hypotension, acute pulmonary congestion, and decrease in sensorium.

3. CASE DISCUSSION

Gout is a type of inflammatory arthritis due to hyperuricemia from an increased body pool of urate. It presents usually with episodic acute and chronic arthritis from the deposition of monosodium urate crystals in joints with or without the formation of periarticular tophi. Uric acid may also deposit in the kidney interstitium causing nephrolithiasis. In the Philippines, the prevalence of gout ranges from 0.13% to 1.6% [24-25]. Progression to tophi formation usually happens in 75% of patients with untreated gout for at least 20 years [26].

Although the patient has chronic gout, he was also suspected of a concomitant septic process due to the presence of worsening sensorium, persistence of swelling of the right knee despite steroids and allopurinol, hemodynamic instability, and a draining sinus which could be an access for infections.

Bacterial arthritis is the purulent infection of a joint causing intense swelling, warmth, and tenderness by bacteria. *Staphylococcus aureus* is the most common isolate followed by *Streptococcus spp.* Previous studies report on the increasing rates of methicillin-resistant *S. aureus* (MRSA) especially among intravenous drug users, the elderly, and in relation to orthopedic procedures [27]. Chronic and immunosuppressive diseases, such as diabetes, leukemia, cirrhosis, granulomatous diseases, cancer and hypogammaglobulinemia are risk factors for bacterial arthritis [28-30]. Moreover, underlying joint diseases such as rheumatoid

arthritis, hip and/or knee prosthesis and joint surgery predispose patients to septic arthritis [28]. While rheumatoid arthritis carries a more potent risk for septic arthritis compared to other joint problems, gout, pseudogout and osteoarthritis are also associated with increased risk [31]. Bacterial arthritis carries a 2%-10% mortality rate [32-34], hence prompt and effective treatment is essential in bacterial arthritis to prevent irreversible joint destruction and even death [5]. Concomitant bacterial and gouty arthritis is rare [9-23].

The patient's synovial fluid grew MRSA similar to two retrospective studies in Taiwan reporting *Staphylococcus aureus* as the most common causative microorganism among patients with concomitant bacterial and gout arthritis [9-10]. The knee joint was the most common site of involvement as in this patient. Possible mechanism of concomitant gouty and bacterial arthritis include the decrease in pH brought about by increased lactic acid production from neutrophilic invasion of the joint thereby decreasing the urate solubility hence the formation of its crystals [21]. Moreover, changes caused by the septic process (e.g. plasma proteins and polysaccharides) could further the precipitation of crystals [35]. The most common mechanism especially among patients with well-established tophaceous gout is by subcutaneous tophi rupture with secondary wound infection which must be the mechanism in our patient [9-10].

Tuberculosis (TB) is a major public health concern in the Philippines with a national incidence of 133 new smear-positive cases per 100,000 population in 2004 [1]. Pulmonary tuberculosis is the most common site of infection occasionally affecting the lymph nodes, spine, genitourinary system, pleura, meninges, and breasts. The joints are rarely affected [2]. Two percent of all cases of tuberculosis affect the bone and joint [3]. The most common peripheral joints affected are the weight-bearing joints especially the knees and the hips [3,36]. Concomitant TB and gouty arthritis is rare with a few case reports published [6-8]. In the Philippines, two cases were reported presenting with recurrent knee pain and swelling which has the same presentation in our patient [7].

This patient presented with concomitant TB and bacterial arthritis with chronic tophaceous gout. To our knowledge, this is the first report of these conditions concurrently in one joint.

4. CONCLUSION

This case highlights the need to explore other causes of joint swelling in a known case of chronic tophaceous gout. Other than the crystal-induced arthritis, concomitant septic arthritis can complicate these patients. Septic arthritis can be tuberculous, bacterial or both. Hence, the need for microbiologic and crystal analysis of synovial fluid cannot be overly emphasized to tailor the specific management for these patients.

CONSENT

It is not applicable.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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