Coexistence of oligoarticular gout and *Mycobacterium kansasii* joint and bursal infection in a patient with an orthotopic heart transplant

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ABSTRACT

Mycobacterium kansasii *septic arthritis is rare, most often occurring in immunosuppressed patients including those with organ transplants. We present a case of oligoarticular* M. kansasii *infection in bilateral ankles, knee, and bilateral olecranon bursae in coexistence with tophaceous gouty arthropathy in a heart transplant patient. There are no reports of* M. kansasii *infection occurring in joints also affected by tophaceous gout. We contend that gouty arthropathy may alter the joint in such a way as to increase the risk of development of this infection in patients already at risk. In transplant patients presenting with a history and pattern of arthritis consistent with gout, a sufficient level of suspicion should be maintained for this infectious complication, even if monosodium urate crystals are seen on joint aspiration.*

Introduction

Mycobacterium kansasii septic arthritis is a rarely described occurrence, most frequently seen in immunosuppressed populations such as patients with AIDS (1). It is also an uncommon complication of organ transplant and can present as pulmonary, cutaneous, or disseminated disease. Septic arthritis in renal transplant patients has been described. We present a case of *Mycobacterium kansasii* septic arthritis and olecranon bursitis in a patient with tophaceous gout and an orthotopic heart transplant.

Case presentation

A 42-year-old man with an orthotopic heart transplant performed for idiopathic dilated cardiomyopathy and a history of chronic kidney disease presented to our hospital with pneumonia and oligoarticular arthritis.

The patient had a history of gout, which was manifested by podagra several years prior to admission and prior to the transplant. Fourteen months prior to admission, while in the cardiac care unit on a ventricular assist device, he was seen by the rheumatology team for severe acute wrist pain. His left wrist was aspirated and monosodium urate crystals were identified. The patient was treated with a course of prednisone and improved. His serum uric acid was measured at 13.9 mg/dL. He was started on allopurinol and colchicine but did not follow up with rheumatology for further management and repeat serum urate measurements.

He had received the heart transplant approximately 12 months prior to this admission. His course was complicated by early grade 2R rejection, which was treated with two rounds of pulse corticosteroids as well as thymoglobulin. He responded to this therapy and had no further complications or episodes of rejection. He remained on prednisone 5 mg daily, tacrolimus 2-4 mg twice daily, and mycophynelate mofetil 1000 mg twice daily for the transplant. He continued to take prophylactic colchicine and allopurinol 300 mg daily. Over the next year he had several flares of gout that did not require admission to the hospital. These were treated successfully with prednisone tapers and had involved his ankle, great toe, knee, and finger.

Approximately 1 month prior to admission he began to experience mild bilateral ankle pain that did not cause him to seek medical attention. Two weeks later he developed progressive dyspnea, fever, and productive cough, which prompted his admission.

He presented with the complaint of shortness of breath, productive cough, fever, weight loss, and night sweats. He was diagnosed with left middle and left lower lobe pneumonia. Staining of the sputum revealed 4+ acid-fast bacilli, which were later identified as *Mycobacterium kansasii*. He was started on multidrug therapy with isoniazid, pyrazinamide, ethambutol, and rifabutin. The colchicine and allopurinol were held on admission due to mild elevation in the serum creatinine that resolved with hydration.

During hospitalization he complained of increasing bilateral ankle and left elbow pain. Initially, this pain severely limited his activity and, on admission day 6, rheumatology was consulted for management of presumed polyarticular gout. The patient complained of pain in both of his ankles, the right knee, and the left olecranon bursa. He was noted to have swelling, warmth, and tenderness in his ankles and right knee. He was

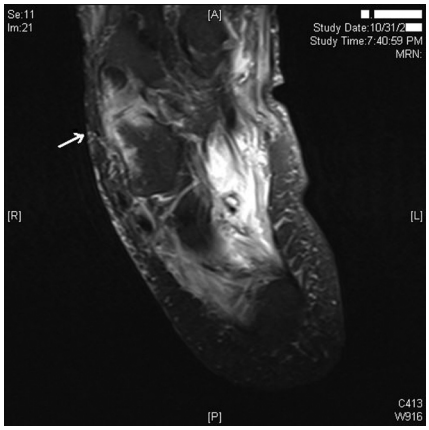

Fig. 1. Synovial enhancement and osteomyelitis about the tarsal-fourth metatarsal joint.

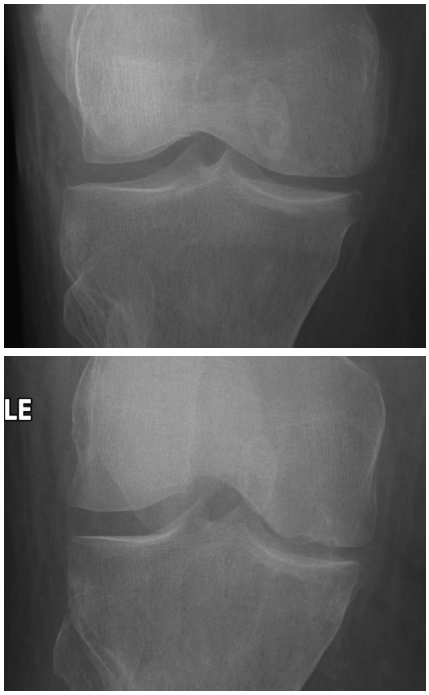

Fig 2. Interval development of mild cortical irregularity involving the medial femoral articular surface after 40 days of hospitalization.

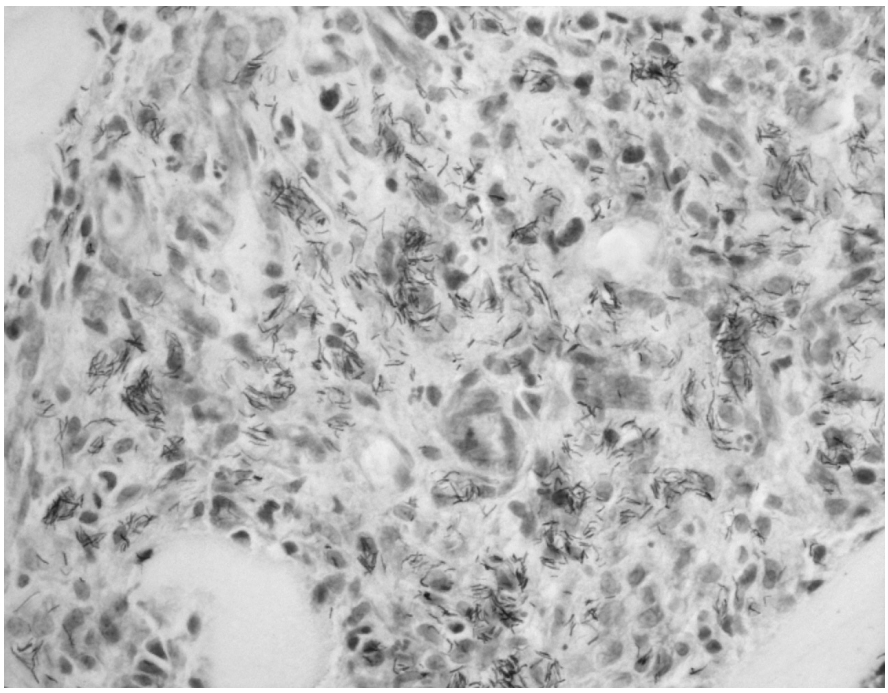

Fig. 3. Histopathological staining of synovial tissue from the right ankle shows extensive chronic inflammation composed of numerous histiocytes; however, no granulomas were identified. Gram and Grocott stains were performed and were negative for bacterial and fungal organisms. However, given the patient's history of mycobacterial infection (*M. kansasii*), acid-fast staining was performed and shows numerous intracellular acid-fast positive bacilli (40X magnification).

also noted to have bilateral olecranon tophi with severe swelling and tenderness on the left and mild tenderness and swelling on the right. A serum uric acid was measured and was 11.6 mg/dL.

Bloody and tophaceous appearing fluid was aspirated from the right ankle and microscopy confirmed tophaceous material and demonstrated numerous intracellular monosodium urate crystals. A stain of the fluid from the ankle also revealed numerous acid-fast bacilli that were later identified as *Mycobacterum kansasii*. The right ankle was re-aspirated on hospital day 9 and, in addition, the left ankle, right knee, and bilateral olecranon bursae were also aspirated to evaluate for infection. Approximately 30 mL of tophaceous fluid was aspirated from the left olecranon and 3 mL from the right olecranon. The white blood-cell count from the left olecranon was approximately 40,000 whilte-blood-cells per high power field. A small amount of fluid was aspirated from the ankles and knee. This was not sent for cell count. Numerous acid-fast bacilli were again noted in the right ankle as well as in both olecranon bursae. Staining and culture of fluid from the right knee and left ankle did not reveal acid-fast bacilli. All joints were noted to have numerous monosodium urate crystals on microscopy.

A non-contrast MRI of the right ankle revealed synovial enhancement and effusion thought to be most consistent with septic arthritis involving the tibiotalar joint with an associated collection superficial to the lateral malleolus. There were also findings suspicious for osteomyelitis involving the dorsal aspect of the middle cuneiform as well as synovial enhancement and osteomyelitis about the tarsal-fourth metatarsal joint (Fig. 1). Non-contrast MRI of the left ankle also revealed enhancement surrounding the tibiotalar joint. There was also synovial enhancement with associated osteomyelitis of the calcaneus and cuboid, as well as abnormal signal within the lateral talus. MRI of the right knee revealed mild thickening and enhancement of the synovium without changes suggestive of osteomyelitis. A repeat film of the right knee, taken for persistent knee swelling 40 days after initial assessment, demonstrated interval development of a mild cortical irregularity involving the medial femoral articular surface (Fig. 2).

The joint pain improved on multi-drug therapy, daily colchicine, and low-dose prednisone (10mg). Orthopaedic consultation was obtained for drainage of the collection noted on MRI. He was taken to the operating room for drainage of the collection and debridement and washout of the right ankle. Synovial tissue sent to pathology revealed extensive chronic inflammation composed of numerous histiocytes without granulomas and stained positive for acid-fast bacilli (Fig. 3). Blood cultures eventually grew *M. kansasii*.

Discussion

M kansasii most frequently causes pulmonary disease and cervical lymphadenitis. There have been approximately 50 reported cases of *M. kansasii* septic arthritis (1). The most appreciated risk factors for the development of this infection include HIV and AIDS, trauma to the joint, corticosteroid injections, and systemic immunosuppressive therapy (1). *M. kansasii* septic arthritis has been reported in renal transplant patients (1, 2). The infection has been described in a number of joints but has been most frequently noted in the knee, ankle, wrist, and elbow. Olecranon bursa involvement has also been described (3). The disease usually presents as an indolent monoarthritis, however, polyarthritis has been previously described (4).

The average interval between the onset of symptoms and the diagnosis of infection is considered to be quite long, approximately 14 months. However, shorter intervals have also been observed (1). Joint infection with *M. kansasii* is most often diagnosed by synovial biopsy and culture. In only about 15% of patients is staining and culture of the synovial fluid alone adequate to make the diagnosis as it was in this case (1). Osteomyelitis of adjacent bone is frequently noted in mycobacterial infection of the joint as it was in this case (5, 6).

In contrast to other mycobacterial species, the environmental source for *M. kansasii* is primarily in tap water. It has been reported to survive for up to 12 months in this environment (7). How this relates to the mechanism of infection remains unclear.

In our patient there was septic involvement of bilateral ankles, the right knee, and both olecranon bursae. To our knowledge, there are no reports of *M kansasii* infection in association with tophaceous gouty arthropathy. In each joint that stained positive for the organism, gouty tophi or monosodium urate crystals were also appreciated on microscopy. We would contend that chronic joint damage and inflammation associated with gouty arthropathy might result in an increased risk of infection with this organism within these joints. An increased level of suspicion for mycobacterial infection should be maintained by the physician treating gouty arthritis in patients with organ transplants who may be at risk for this infection. In our patient, staining of the synovial fluid was enough to make the diagnosis whereas synovial biopsy is usually necessary. Might it be that the environment present in a gouty joint increases the sensitivity of acid-fact staining for this organism? Gouty tophi may themselves provide a nidus for infection in patients with inadequately managed gout such as in our patient.

Out patient had antecedent tophaceous gout that may have been a risk factor for the development of this infection. Many transplant patients will develop hyperuricemia and gout secondary to calcineurin-inhibitor use although this is less frequently observed with tacrolimus. However, despite the high incidence of gout in this population, care should be taken to consider mycobacterial infection as an uncommon cause of oligoarticular arthritis in a distribution also consistent with gout. Therapy with allopurinol with the specific goal of normalization of the serum urate concentration should be undertaken. Untreated hyperuricemia and gout may even result in an increased mortality (8). Due to a lack of appreciation of this problem, our patient was seriously undertreated for tophaceous gout and this may have contributed to abnormal joints and the subsequent development of oligoarticular infection. This case underscores the idea that chronic inflammation and joint damage due to gout may contribute to an increase risk of infection in that joint, especially in the immunosuppressed (9).

Management should generally include antibiotic therapy, in some cases combined with surgical debridement and synovectomy as well as removal of osteitic bone when necessary. Frequently patients achieve a cure, but this can take many months of multidrug therapy (1).

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