Mycobacterium kansasii causing chronic monoarticular synovitis in a patient with HIV/AIDS

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ABSTRACT

Mycobacterium kansasii is a nontuberculous mycobacterium that primarily causes pulmonary disease in AIDS patients, however it has also been known, rarely, to result in skeletal infection. When skeletal infection occurs, the time from onset of symptoms to diagnosis is up to 5 years in previously reported cases. We describe a 48-year-old woman with HIV/AIDS who presented with chronic, isolated left knee pain and swelling of over two decades which had recently worsened. Radiographs and magnetic resonance imaging demonstrated marked subarticular erosions, synovial thickening, and bone marrow edema, which had progressed compared with prior imaging done seven years earlier. Synovial biopsy grew Mycobacterium kansasii. Following the presentation of our case, clinical and imaging findings, including the differential diagnosis, of monoarticular arthritis caused by Mycobacterium kansasii are reviewed and discussed.

CASE REPORT

CASE REPORT

A 48-year-old woman with human immunodeficiency virus/acquired immune deficiency syndrome (HIV/AIDS) presented to our emergency department with chronic left knee pain and swelling which had worsened over the past month. According to the history, 22 years prior to this presentation, at the age of 26, while living in the southeastern United States, the patient was involved in a motor vehicle accident. The patient was dragged by a car, which injured her knee and created an open sore. This healed without her seeking medical treatment. Ever since that time, she experienced waxing and waning knee pain which she self-treated with over-the-counter pain medications. At age 33, the patient was diagnosed with HIV and had subsequently poor medication compliance. At age 41, plain radiographs of the left knee performed in the emergency department showed a small subarticular lucency of the medial tibia and a suprapatellar joint effusion (Fig. 1). Magnetic resonance imaging (MRI) performed three months later revealed generalized extensive synovial thickening and moderate joint effusion (Fig. 2). Clinical follow-up of these imaging studies, if any, is not documented in our records.

At the current emergency department visit, the patient's left knee pain was exacerbated by ambulation and associated with joint stiffness. There were no fevers or chills. On exam the patient had left knee joint swelling with a large palpable effusion, erythema, and mild warmth with limited extension due to pain. Erythrocyte sedimentation rate was significantly elevated to 143 mm/hr (normal range: 0-20 mm/hr).

Radiographs of the knee at this visit demonstrated joint space narrowing and marginal erosions (Fig. 3). A large effusion was also seen with apparent thickening of the synovium. Aspiration of the joint was performed. Synovial fluid gram stain and culture were negative.

The patient was prescribed non-steroidal anti-inflammatory drugs. An MRI was subsequently performed which showed extensive subarticular erosions, synovial thickening, and a joint effusion (Fig. 4). The clinical differential diagnosis at this point was inflammatory arthropathy, likely sero-negative spondyloarthropathy.

The patient was referred to rheumatology for evaluation. C-reactive protein and erythrocyte sedimentation rate were elevated at 19.0 mg/L (normal range: <5.0 mg/L) and 120 mm/hr, respectively, while antinuclear antibody, rheumatoid factor, and Lyme titer were negative. CD4 count was 279 cells/mcl (normal range: 500-1500 cells/mcl), with the patient's nadir of 147 cells/mcl fulfilling the diagnosis of AIDS. Her adherence to highly active antiretroviral therapy in the HIV clinic was poor. These findings made rheumatologic etiology or Lyme disease less likely but were more concerning for opportunistic infection. Synovial biopsy was performed and showed granulomatous synovitis with negative Gram and acid-fast stains. Initial Quantiferon Gold was negative but repeat testing two months later became positive. Mycobacterial culture from the synovial biopsy grew Mycobacterium kansasii (M. kansasii) three months after biopsy. The patient then began treatment with a four-drug antimycobacterial regimen: azithromycin, rifabutin, ethambutol and isoniazid/pyridoxine. She had a marked decrease in pain and swelling after 4 months of therapy. At a 6-month follow-up appointment, the patient had stopped all her medications due to depression and had developed increasing left knee pain and swelling. She was placed back on the four-drug regimen and has since been lost to follow-up for 4 months.

DISCUSSION

Etiology & Demographics

Atypical mycobacteria rarely develop in healthy individuals and had originally been considered saprophytes; it was only in the 1950's that these mycobacteria were recognized as human pathogens [1]. M. kansasii was first described in 1953 [2] presenting in one patient as pulmonary disease and in another as disseminated infection. Due to the appearance of colonies in culture, it was initially termed the "yellow acid fast bacillus". It is considered to be an atypical mycobacterium along with Mycobacterium marinum, M. avium, M. abscessus, and others.

M. kansasii may be found in water, cattle, and pigs [1]. It has been isolated from tap water in areas where the disease exists but not from soil or natural water sources and is able to live for up to 12 months in tap water [3]. It is not transmissible from person to person. In the United States, along with other atypical mycobacteria, M. kansasii is more common in the South.

Infection with M. kansasii, as with other nontuberculous mycobacterial infections, is more common in patients with HIV and especially in those with lower CD4 counts (AIDS) [3, 4]. The lungs are the most common site of infection with M. kansasii, and it is the second most common non-tuberculous mycobacterial cause of pulmonary disease. Disseminated disease can occur in patients with very low CD4 counts [3]. Musculoskeletal involvement by M. kansasii is rare, with tendon sheath or septic arthritis being the most common sites of infection [5]. Despite this, it is the most common nontuberculous cause of mycobacterial osteomyelitis and single joint synovitis [1, 5], with Mycobacterium avium being another cause of atypical mycobacterial synovitis and osteomyelitis [6, 7]. Risk factors for synovitis and osteomyelitis include HIV/AIDS, systemic M. kansasii infection, recent steroid therapy, and trauma to the affected joint [5]. The incidence of skeletal infection with M. kansasii is not known due to lack of systemic reporting, however it is quite rare. In one case series and review of prior case reports, by Bernard et al., 50 reported cases were collected from 1963 to 1998 [5]. In these reviewed 40 cases and in their own 10 cases, it was found that of those patients with systemic disease (over half of whom had HIV), 46% had disseminated M. kansasii and/or polyarthritis, and 17% had arthritis only in the knee. In the patients without systemic diseases, 39% were associated with trauma and 39% were associated with recent local steroid therapy [5]. There was no age predilection in this case series and review, with the range of 13-82 years and a mean age in the mid-40s, however a further more recent case is that of a child developing ankle M. kansasii infection under the age of 5 [8]. A PubMed search of published cases from 1999 through 2014 revealed an additional 23 cases, 12 of which are of tenosynovitis [9-31].

Clinical & Imaging findings

Patients with M. kansasii septic arthritis usually present with swelling and chronic pain of the affected joint, but without fever. Synovial biopsy is usually required for diagnosis, with the mean time between the onset of a patient's symptoms and diagnosis 14 months (range 1-60 months) [5].

Radiographically, early atypical mycobacterial joint infection can show no bony findings but only joint effusion with soft tissue swelling [5, 1]; this was the case for our patient at the time of her first radiographs. Computed tomography (CT) may demonstrate synovial thickening along with the radiographic findings noted, although this was not performed in the case of our patient. A tiny medial tibial marginal erosion was also noticed which fits mycobacterial infection but at this early stage, with no other bony findings, is nonspecific. Upon progression, infection spreads into the synovium manifesting as thickened synovium and osteopenia [1], the former of which was seen in our patient on the MRI that was performed three months after the first knee radiograph.

Marginal and subchondral erosions become more prominent on radiograph and CT upon progression of atypical mycobacterial arthritis. Phemister's triad of subchondral osteoporosis, peripheral marginal erosions, and gradually progressing articular cartilage loss (manifested as joint space narrowing) is often seen in mycobacterial joint infections [1,

32]. Our patient demonstrated progression on plain radiograph upon her return to the emergency department 7 years following the first radiographs. More pronounced marginal erosions, intercondylar notch erosions, osteopenia and mild joint space narrowing are seen, along with a larger joint effusion. On MRI, low to intermediate signal intensity on T1-weighted sequences and high signal on fluid-sensitive sequences is typically seen in the joint, in areas of bony erosions, and in patchy areas of adjacent bone marrow [1, 33, 34]. Heterogeneous signal is seen in nodular, thickened synovium, which also demonstrates avid post-contrast enhancement [33]. These findings were demonstrated on our patient's MRI which was performed shortly after the previously described radiograph.

Treatment & Prognosis

M. kansasii infection, including septic arthritis, is treated with tailored antimycobacterial antibiotics [3], which differs from the regimen commonly used for Mycobacterium tuberculosis, hence the importance of distinguishing between the two. Pharmacotherapy commonly lasts 18 months. Additionally, surgical debridement is often employed [5, 3]. For those patients with M. kansasii septic arthritis who are otherwise healthy, antibiotic treatment is curative in the vast majority of cases, however in those with other systemic diseases (including AIDS, rheumatoid arthritis [RA], and renal transplant) the outcomes are more mixed [5].

Differential Diagnosis

Atypical mycobacterial arthritis must be distinguished from crystal deposition diseases, pyogenic or fungal infection, pigmented villonodular synovitis (PVNS), RA, and amyloid arthropathy, which can all have similar imaging features.

Gout and calcium pyrophosphate dehydrate crystal deposition disease are diseases characterized by crystal deposition in synovial joints and other soft tissues. In gout, uric acid crystals are deposited in the synovial joints causing gouty arthritis; in more advanced disease, tophi can form in soft tissues. Gout has classic radiographic findings of well-circumscribed erosions with overhanging edges and soft tissue tophi. CT demonstrates similar findings to radiograph, with the addition of tophi seen in the erosions [35]. On MRI, tophi are seen, which are generally of intermediate signal intensity on T1-weighted images, of heterogeneous intensity on T2-weighted images, and heterogeneously enhancing on post-contrast images. Bone marrow edema can be seen adjacent to intraosseous tophi. Soft tissue swelling is usually seen as well. Mass-like synovial thickening (pannus) and synovial effusion are also often present [36]. Tophi or pannus with adjacent bone marrow edema can resemble the nodular thickening of atypical mycobacterial infection, however mycobacterial infection lacks the prominent, well-defined marginal erosions seen in gout. Clinically, gout in the joint can usually be distinguished from mycobacterial arthritis by the presence of urate crystals in joint aspirate.

In calcium pyrophosphate dehydrate crystal deposition disease, crystals are deposited primarily in the soft tissues of the joint. Radiographs of the knee often, but not always, show calcification of cartilage, synovium, joint capsule, tendons and ligaments, with a predilection for the menisci. Subchondral cysts can be seen. Marked loss of joint space at the patellofemoral compartment out of proportion with that at the tibiofemoral compartments has also been described. CT demonstrates similar findings as those seen on radiograph, but with more sensitivity. On MRI, linear areas of hypointensity can be seen in the hyaline cartilage, representing calcification [37, 38]. Calcification in the articular soft tissues is usually seen on radiographs before any significant internal derangement of the joint occurs; this is not the case in atypical mycobacterial infection.

Pyogenic septic arthritis is a clinical diagnosis based on physical exam and joint fluid aspirate. However if performed, imaging can demonstrate non-specific findings such as joint effusions and synovial thickening. Complications can be seen on MRI, such as adjacent osteomyelitis or abscesses [39]. MRI in fungal arthritis can reveal synovial proliferation and adjacent T1-weighted bone marrow hypointensity representing osteomyelitis, if present [40].

PVNS is a benign neoplasm involving either the synovium or the surrounding extraarticular structures. On plain radiograph, diffuse intraarticular PVNS (that involving the synovium) usually exhibits itself as a joint effusion, soft tissue swelling, and extrinsic bony erosion, but with lack of osteopenia or joint space loss. On CT, diffuse thickening of the soft tissues around the joint or soft tissue masses are often seen. These may measure increased density relative to the surrounding soft tissues due to hemosiderin deposition. MRI typically demonstrates a joint effusion and diffuse, nodular, synovial thickening of intermediate to low signal intensity on T1-weighted sequences and low signal intensity on T2-weighted sequences. Hemosiderin deposits in the synovial thickening reveal themselves as susceptibility artifact on gradient-echo sequences. Any bony erosion seen is caused by the extensive nodular thickening of synovium [41]. Findings in atypical mycobacterial arthritis on radiographs and MRI which are not seen in, and can help distinguish it from, PVNS are: bony erosion in the absence of significant mass effect from the synovial thickening; osteopenia in the periarticular bone; patches of signal intensity on fluid sensitive sequences and post-contrast enhancement in the bone marrow which are both indicative of edema and inflammation.

RA is a chronic systemic inflammatory condition, which principally affects the joint synovium. It typically manifests as a polyarticular arthritis, involving the hands before disease in other joints is severe [42]. Radiographically, RA shows joint effusion, joint space narrowing, osteopenia, subcortical cysts, and erosions. CT demonstrates the same findings as radiograph, with the addition of more clearly seen synovial thickening. On MRI, effusion, thickening and enhancement of synovium (representing synovitis and hyperemia, respectively), bone marrow edema, and erosions can be seen. In later stage disease, massive erosions and further bone destruction can often become evident [43]. Several of these findings can also be found in atypical mycobacterial arthritis, however, in mycobacterial arthritis significant synovitis, nodular thickening, and permeative-appearing bone marrow extension of disease can be seen without prominent joint space

narrowing. In mycobacterial infection, joint space narrowing occurs more slowly and is seen later than in RA, because the contact and pressure in the joint helps to protect the surfaces from mycobacterial involvement, whereas the free articular cartilage surfaces, where the mycobacterium are able to attach, are the first to be destroyed [44].

Amyloidosis includes several disease entities all of which involve the deposition of insoluble fiber-like protein in tissues. Amyloid arthropathy results from deposition of this protein into and around the joints, usually bilaterally, with the most frequent joints involved being the shoulders, hips, wrists, and knees. Nodules created from soft tissue amyloid deposition can be seen and arise most often in the elbows, hands, and wrists. Radiographic and CT findings of amyloid arthropathy include: joint effusions, subchondral cysts, marginal erosions, periarticular osteopenia, asymmetric soft tissue masses, and relative sparing of the joint space. The subchondral cysts can be large with sclerotic margins, and pathologic fractures are possible due to these lesions. On MRI, soft tissue amyloid deposition usually exhibits low T1-weighted and T2-weighted signal, whereas bony involvement is similarly low on T1-weighted sequences but can be low to heterogeneously high on T2-weighted sequences. Synovial and tendon thickening can be seen-the latter of which occurs mostly in the shoulder. The findings of joint effusion, marginal erosions, osteopenia, synovial thickening and joint space preservation can be seen in amyloid arthropathy and atypical mycobacterial arthritis, however the typically monoarticular distribution and high signal intensity on fluid-sensitive MRI sequences as well as avid post-contrast enhancement of mycobacterial infection help to distinguish it from amyloid arthropathy [45, 46].

TEACHING POINT

M. kansasii monoarticular synovitis and osteomyelitis are a rare manifestation of atypical mycobacterial disease, more commonly seen in AIDS patients, which is often reported following trauma to the involved joint, but which may have an incubation period of several years. Atypical mycobacterial infection should be considered in the differential diagnosis for any chronic monoarticular arthritis demonstrating imaging findings of nodular synovial thickening, bony erosions, and synovial effusion.

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FIGURES

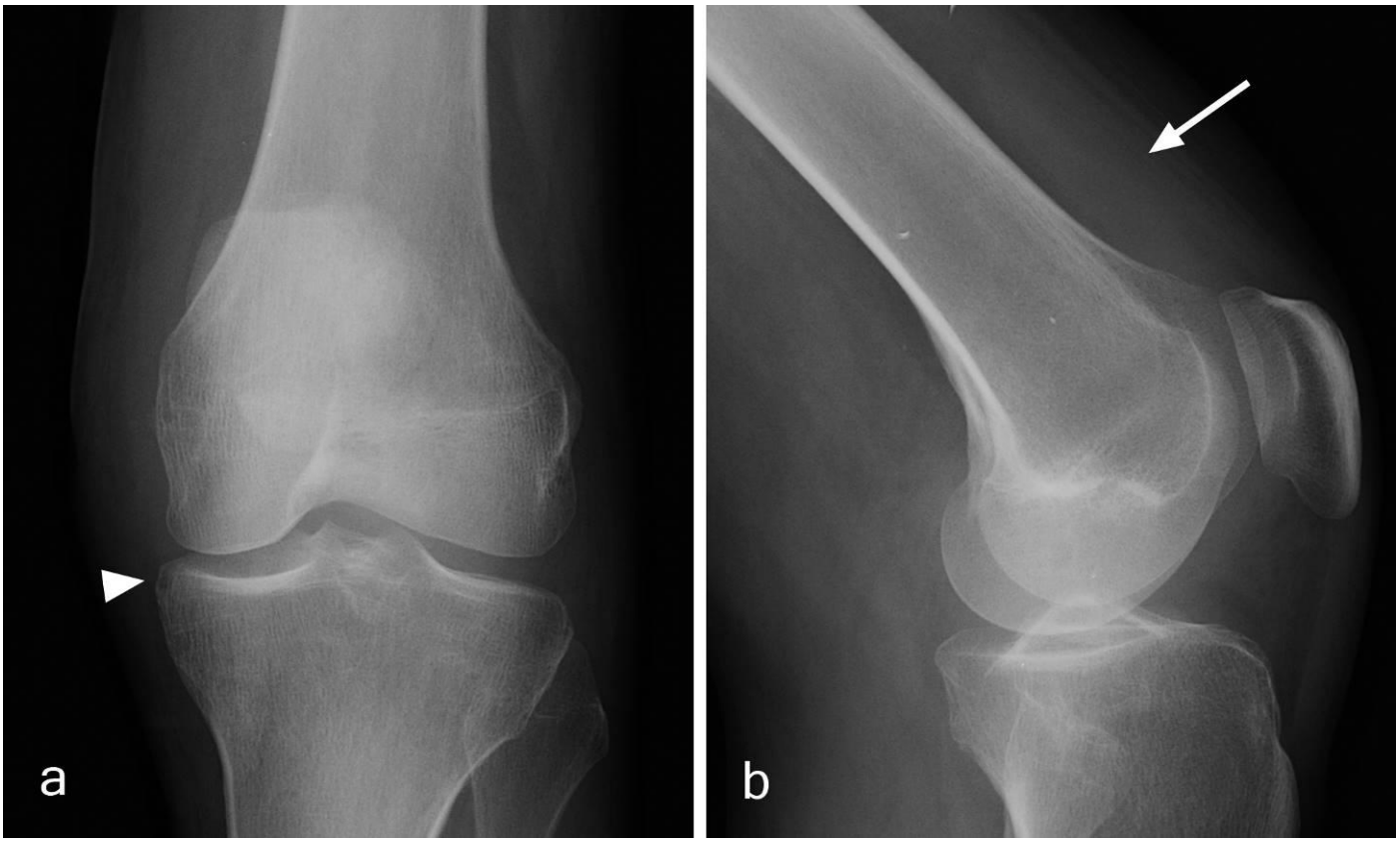

Figure 1: 41-year-old woman with Mycobacterium kansasii synovitis and osteomyelitis of the knee.
Findings: Anterior-posterior (a) and lateral (b) radiographs of the left knee performed on the patient's initial visit to the emergency department demonstrate a tiny medial marginal tibial erosion (arrowhead), and synovial effusion (arrow).
Technique: kVp and mAs were not recorded.

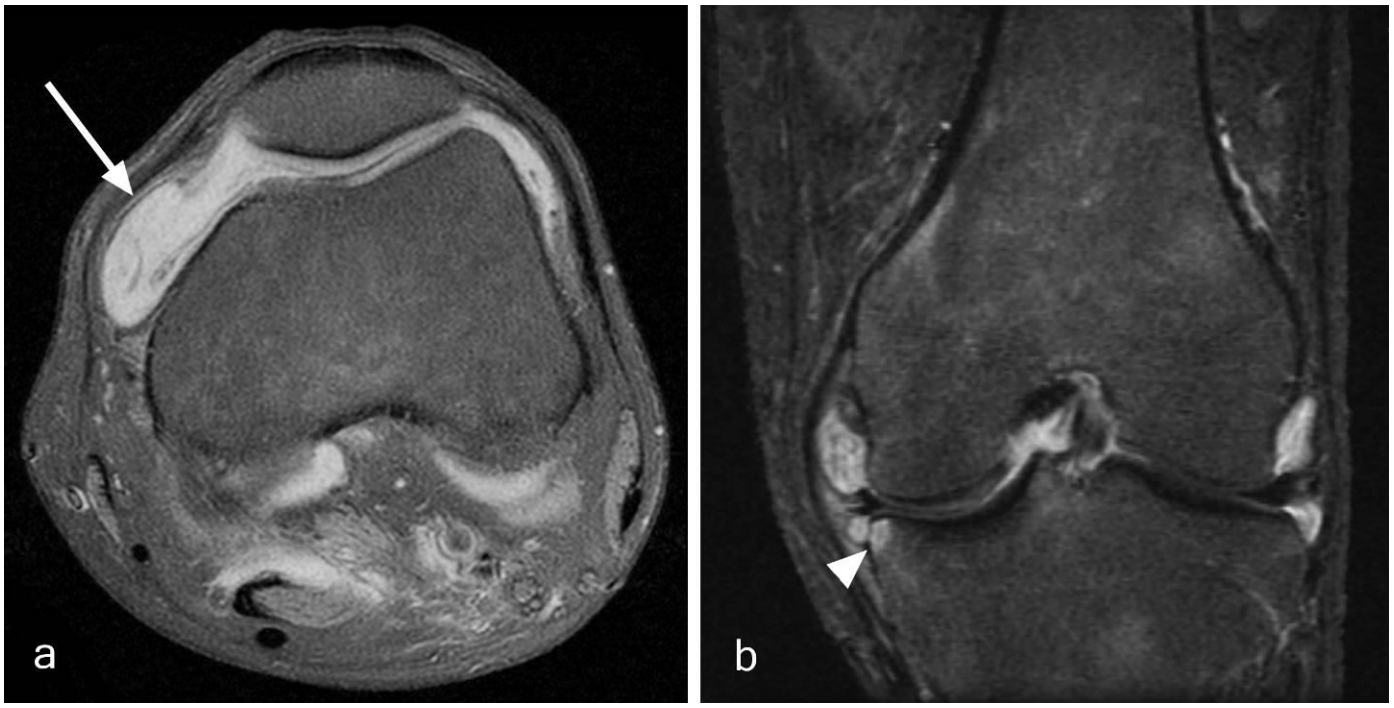

Figure 2: 41-year-old woman with Mycobacterium kansasii synovitis and osteomyelitis of the knee. The MR examination of the left knee was performed three months after the initial radiographs (Figure 1).
Findings: Non-contrast axial fat-suppressed proton density (PD) (a) and coronal short tau inversion recovery (STIR) (b) images of the left knee demonstrate synovial thickening with effusion (arrow) and hyperintense STIR signal in the bone marrow of the medial tibia (arrowhead) in the area of the tiny erosion seen on the radiographs from three months earlier.
Technique: MR was performed on a GE® 1.5 T magnet. The parameters are as follows: a: PD (TR: 2,250; TE: 24.24), slice thickness 4 mm; b: STIR (TR: 3,600; TE: 30.64), slice thickness 4 mm.

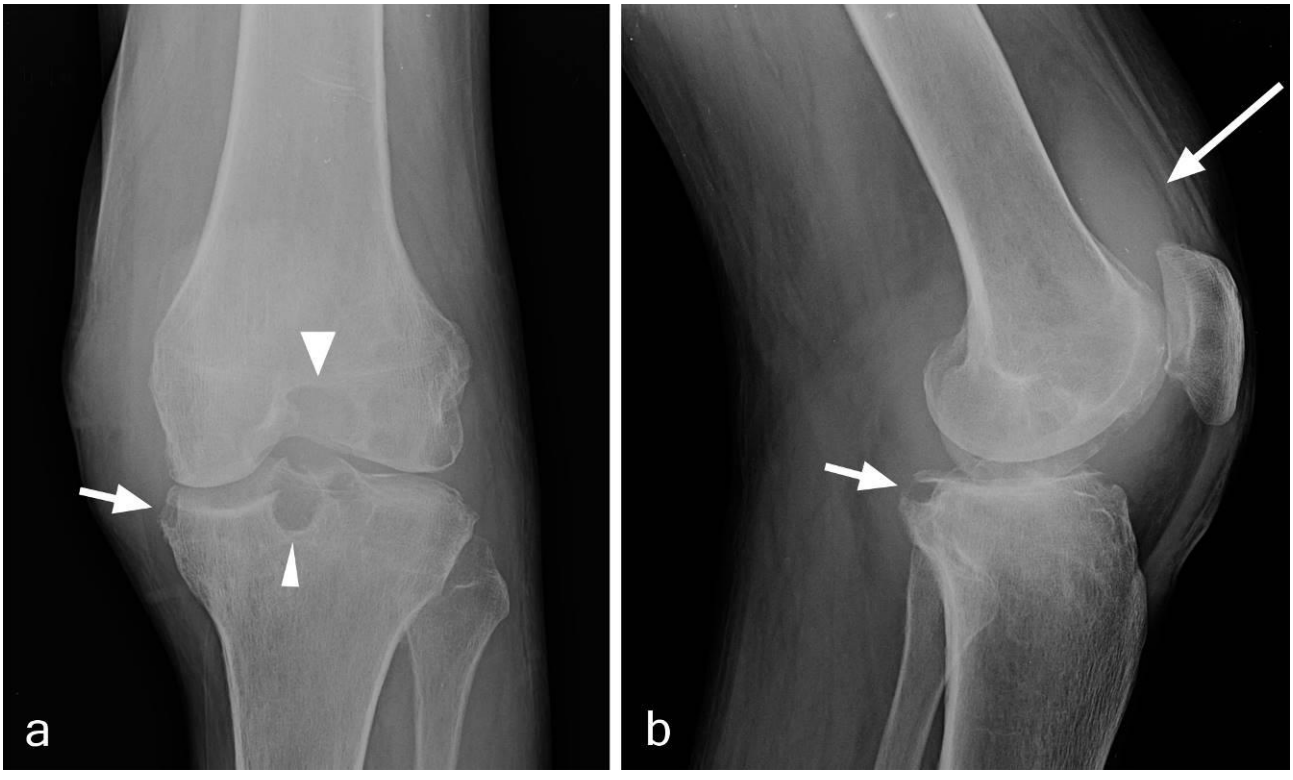

Figure 3: 48-year-old woman with Mycobacterium kansasii synovitis and osteomyelitis of the knee.
Findings: Anterior-posterior (a) and lateral (b) radiographs of the left knee performed seven years after those of figure 1 demonstrate joint space narrowing, synovial effusion (long arrow), subchondral lucencies, intercondylar notch widening (wide arrowhead), an erosion adjacent to the tibial spine (narrow arrowhead), marginal erosions (short arrows), and soft tissue swelling around the medial aspect of the knee.
Technique: kVp and mAs were not recorded.

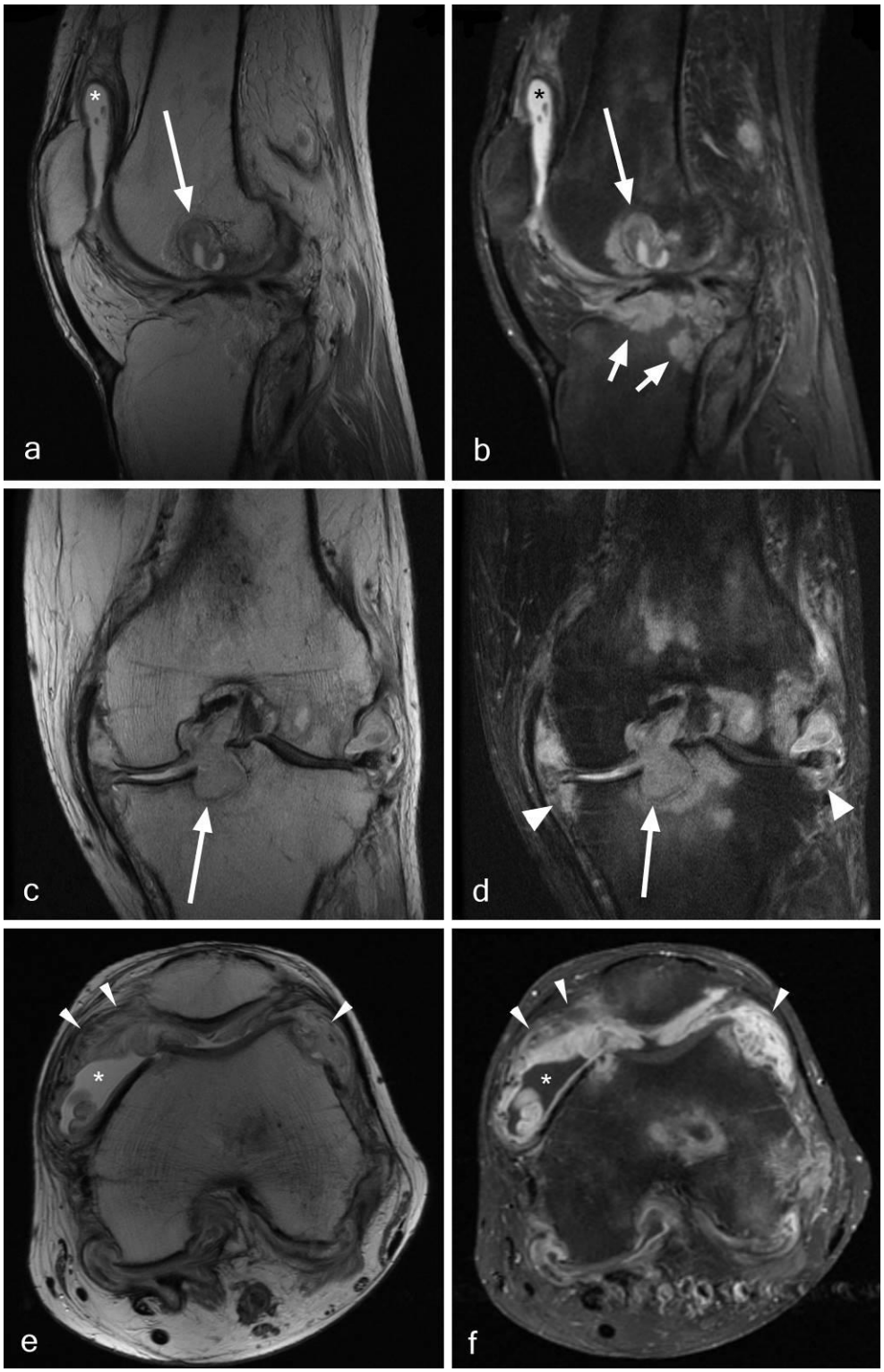

Figure 4: 48-year-old woman with Mycobacterium kansasii synovitis and osteomyelitis of the knee. MR examination of the left knee was performed seven years after the initial MRI (Figure 2). Intravenous gadolinium contrast was administered prior to figure 4f.

Findings: Sagittal, coronal, and axial PD (a, c, e), sagittal and coronal PD with fat suppression (b, d), and axial post-contrast T1-weighted fat-suppressed (f) images demonstrate large subarticular erosions (long arrows) with heterogeneously decreased signal on proton density (a) and increased signal on fluid sensitive images (b, d) compared to bone marrow, as well as marginal erosions with fluid sensitive hyperintensity (wide arrowheads d). There is nodular synovial thickening with contrast enhancement (narrow arrowheads e, f), and a joint effusion (asterisks a, b, e, f). Bone marrow edema is also seen (short arrows b).

Technique: MR was performed on a GE® 1.5 T magnet. The parameters are as follows: a: PD (TR: 4000; TE: 36.864), slice thickness 3 mm; b: PD with fat suppression (TR 3600; TE 45.6), slice thickness 4mm; c: PD (TR 4000; TE 35.865), slice thickness 3mm; d: PD with fat suppression (TR 4000; TE 35.856), slice thickness 3mm; e: PD (TR 4000; TE 35.736), slice thickness 3.5 mm; f: T1-weighted sequence with fat suppression after administering 5 mL Gadavist® gadolinium intravenous contrast (TR 733.336; TE 14), slice thickness 3mm.

Diagnosis	Radiograph/CT	MRI
Mycobacterium kansasii synovitis/ osteomyelitis	• Early: soft tissue swelling with synovial effusion. No bony findings. • Late: thickened synovium with effusion. • Osteopenia, peripheral marginal and subchondral erosions, with gradual joint space loss.	• Low to intermediate signal intensity on T1-weighted images and high signal intensity on fluid-sensitive sequences in the joint, in areas of bony erosion and in patchy areas of adjacent bone marrow. • Thickened synovium with avid contrast enhancement.
Gout	• Well-circumscribed marginal erosions with overhanging edges. • Soft tissue tophi. • Additionally on CT: tophi within the erosions.	• Tophi with intermediate signal intensity on T1-weighted images and heterogeneous signal intensity on T2-weighted images with heterogeneous post-contrast enhancement. • Bone marrow edema often adjacent to intraosseous tophi. • Mass-like synovial thickening and synovial effusions.
Calcium pyrophosphate dehydrate crystal deposition disease	• Calcification of articular soft tissues, e.g. cartilage, synovium, tendons and ligaments—particularly menisci in the knee. • Marked patellofemoral compartment joint space loss.	• Linear areas of hypointensity in hyaline cartilage representing calcification.
Pyogenic/fungal infection	• Synovial effusion +/- thickening.	• Synovial effusion and thickening/proliferation. • Complications such as adjacent osteomyelitis or abscesses.
Pigmented villonodular synovitis	• Synovial effusion, soft tissue swelling, extrinsic bony erosion with lack of osteopenia or joint space loss. • Additionally on CT: diffuse periarticular soft tissue density or masses, with increased density due to hemosiderin deposition.	• Synovial effusion. Diffuse, nodular synovial thickening of intermediate to low signal intensity on T1-weighted images and low signal on T2-weighted images. • Susceptibility artifact in the synovial thickening on gradient-echo images, which represents hemosiderin deposits.
Rheumatoid arthritis	• Synovial effusion, early joint space narrowing, osteopenia, subcortical cysts, erosions. • Additionally on CT: synovial thickening.	• Synovial effusion. Thickening and enhancement of the synovium. • Bone marrow edema and erosions.
Amyloid arthropathy	• Synovial effusion, joint space preservation, marginal erosions, osteopenia, asymmetric soft tissue masses, subchondral cysts (can be large, with sclerotic margins).	• Synovial effusion. • Synovial thickening. • Periarticular osseous erosions filled with tissue of low T1-weighted signal but low to heterogeneously high T2-weighted signal. • Soft tissue masses with low T1- and T2-weighted signal. • Tendon thickening (only present in shoulder).

Table 1: Differential diagnosis table for Mycobacterium kansasii knee synovitis and osteomyelitis.

Etiology	A joint infection caused by an atypical mycobacterium which can be found in tap water or livestock, and enters the joint by either direct inoculation from trauma or from systemic spread of the organism
Incidence	Rare, only 73 published cases from 1963 to 2014
Gender ratio	No gender predilection
Age predilection	Can occur at any age, with cases in the literature ranging from ages <5-82
Risk factors	HIV/AIDS; systemic Mycobacterium kansasii infection; immunosuppression; trauma to the joint; corticosteroid injection to the joint
Treatment	Tailored antimycobacterial antibiotic therapy with or without surgical debridement
Prognosis	Very good in otherwise healthy patients; mixed response in those with systemic diseases
Findings on imaging	*Radiography/CT:* • Early: soft tissue swelling with synovial effusion. Often no bony findings. • Late: thickened synovium with effusion. Osteopenia, peripheral marginal and subchondral erosions, with gradual joint space loss. *MRI:* • Low to intermediate signal intensity on T1-weighted images and high signal intensity on fluid-sensitive sequences in the joint, in areas of bony erosion and in patchy areas of adjacent bone marrow. • Thickened synovium with avid contrast enhancement.

Table 2: Summary table for Mycobacterium kansasii synovitis and osteomyelitis.

ABBREVIATIONS

AIDS = acquired immune deficiency syndrome
CT = computed tomography
HIV = human immunodeficiency virus
M. kansasii = Mycobacterium kansasii
MR = magnetic resonance
MRI = magnetic resonance imaging
PD = proton density
PVNS = pigmented villonodular synovitis
RA = rheumatoid arthritis
STIR = short tau inversion recovery
TE = echo time
TR = repetition time

KEYWORDS

Mycobacterium kansasii septic arthritis and osteomyelitis; Mycobacterium kansasii; septic arthritis; synovitis; osteomyelitis; atypical mycobacterium; atypical mycobacterial synovitis and osteomyelitis; HIV; AIDS; knee; radiograph; MRI

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