

## Case Report

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# Nontuberculous Mycobacterial Tenosynovitis in the Hand: Two Case Reports with the MR Imaging Findings

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Nontuberculous mycobacterial infections can cause destructive tenosynovitis of the hand. We report on and discuss the clinical course and distinctive radiologic findings of two patients with hand tenosynovitis secondary to *M. marinum* and *intracellulare* infection, which are different from those of the nontuberculous mycobacterial infections reported in the previous literature.

**Index terms:** *Mycobacteria, atypical; Tenosynovitis; Hand; Radiography; Magnetic Resonance Imaging*

## INTRODUCTION

Nontuberculous mycobacteria (NTM) are ubiquitous organisms that are frequently present in the environment (soil, water) and in animal reservoirs (1, 2). NTM infections in adult immunocompetent hosts are uncommon, and the lungs are most commonly infected in 90% of the cases (1, 3). The hand and wrist are the most frequently reported sites of NTM tenosynovitis because of their abundance of synovial fluid and tissue combined with a higher probability of penetrating injury at those sites. However, there are only rare reports of NTM infections of the musculoskeletal system (1, 3). Consequently, clinical awareness of this disease has been poor because of the difficulty of making the diagnosis, and the radiographs obtained in the early stage and even

in the advanced stages can be normal. Thus, this can cause delayed treatment. NTM infections are now being seen with increasing frequency as a cause of localized soft-tissue infection, and especially in the hand (4, 5). The radiologic features of NTM tenosynovitis have been characterized by exuberant tenosynovitis that rarely involves the underlying muscles and bony structures with preserved joint spaces and without tendon tear even in the long-standing phase (6-8). However, we encountered two distinctive cases of NTM tenosynovitis in the hand, which caused tendon disruption and bony involvement.

To heighten the awareness of this infection so that delays in making the diagnosis and administering proper treatment can be avoided, we present here two cases of NTM tenosynovitis of the hand and we review the radiological characteristics and discuss the differential points from the previous radiologic findings reported in the medical literature.

## CASE REPORTS

### Case 1

A 59-year-old male, a fisherman, was referred to a physician in August 2007 because of swelling, erythema, ulceration, and pain in the right hand. In November 2006,

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**Fig. 1.** 59-year-old male, fisherman, with swelling, erythema, ulceration and pain in right hand.  
**A.** Radiograph shows diffuse soft tissue swelling of second and third right fingers without bone erosions. **B.** Axial T2-weighted MR image shows tenosynovitis around second and third fingers with soft tissue edema. Extensor tendon of second digit is focally disrupted (arrow). **C.** Axial fat-suppressed T1-weighted image with gadolinium enhancement shows diffuse enhancement along extensor tendon sheaths with tear of sheath at second finger (arrow). Adjacent bone marrow enhancement is also noted (arrowhead).

he received shallow penetrating injury on his right third finger by a fish bone while he was pulling up a net. After that, his third finger was swollen and pain then developed. He had visited a local clinic and took antibiotics for about eight months. However, his symptoms were aggravated. He had no relevant medical history, with the exception of inactive pulmonary tuberculosis that was completely treated in 2003.

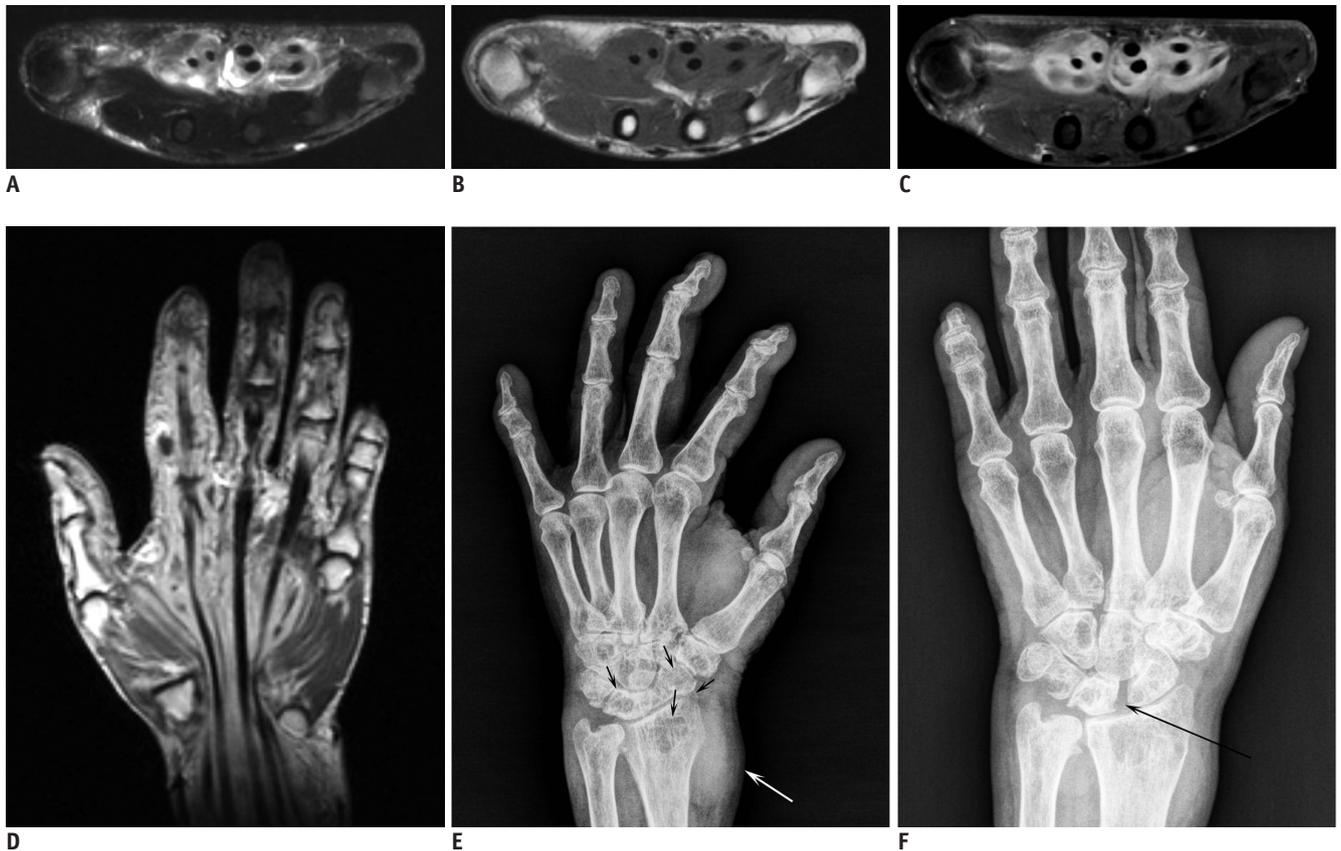
The patient was afebrile and in good general health. Swelling, erythema, tenderness and ulceration were noted in the dorsum of the right hand, and an abscess-like pocket in the palm of the hand was observed. He had been evaluated at the emergency care unit 48 hours earlier, and culture tests of the soft tissue and acid-fast stains were done at that time. Polymerase chain reaction (PCR) analysis for mycobacteria was not performed.

The blood tests showed an erythrocyte sedimentation rate of 84 mm/hr (normal values, 3-20 mm/hr) and the C-reactive protein level was 2 mg/L (normal values, 0.1-5 mg/L); the general hematologic and biochemical parameters were in the normal ranges. Culture tests showed there was no growth in the soft tissue, but the acid-fast stains were positive. Chest radiographs showed no active lung lesion and hand radiographs showed increased soft tissue density of the second and third right finger, without joint or bone erosions (Fig. 1). Magnetic resonance imaging (MRI) of the hand showed a proliferative tenosynovitis of the extensor

tendon sheaths and some flexor muscles in mainly the second and third fingers without definite abscess formation. Increased signal intensity of the tendon in the second digit on the T2 weighted sequence and diffuse enhancement along the tendon sheath were noted on the fat-suppressed T1-weighted image with gadolinium enhancement. The extensor tendon of the second digit was focally disrupted, so the margin of the tendon was not clearly delineated. Moreover, there was focal signal change of the bone marrow adjacent to the disrupted tendon (Fig. 1).

He was admitted to the hospital for further management, and antituberculous treatment was first started under the impression of tuberculous tenosynovitis. Finally, tenosynovectomy of the tendon sheaths of the second and third fingers was performed and the biopsy material was sent for microbiological and pathological analysis. During the operation, disruption of the extensor tendon in the second digit and soft tissue necrosis around the second and third digits were found. The histology revealed non-caseous granuloma. Acid-fast staining was positive and the culture and PCR analysis for mycobacteria were negative. A few weeks later, atypical acid-fast bacilli could be grown and they were identified as *Mycobacterium marinum*. No other clinical manifestations of atypical mycobacterial infection could be identified.

The patient was prescribed of 500 mg clarithromycin, 800 mg of ethambutol and 750 mg of rifampicin daily for 16



**Fig. 2.** 63-year-old woman with volar side pain of left wrist.

**A.** Axial fat-suppressed T2-weighted MR image shows thickening and increased signal intensity of flexor tendon sheaths. **B, C.** Precontrast (**B**) and postcontrast (**C**) T1-weighted MR images show diffuse peripheral enhancement, suggesting exuberant tenosynovitis. Flexor tendons are intact and no signal changes of bone marrow are noted. **D.** Coronal T1-weighted image with gadolinium enhancement shows tenosynovitis from wrist to distal portion of second digit. **E.** Radiograph (oblique view) obtained four years after initial visit shows bony erosions along margin of left radiocarpal bones with osteoporotic change (black arrows) and round mass at volar aspect (white arrow). **F.** Radiograph (posteroanterior view) obtained four years after initial visit shows widening of scapholunate joint space (black arrow).

months. He was evaluated every three months. The lesion is currently stable and no signs of infection have been detected, but he still has intermittent pain on his finger.

### Case 2

A 63-year-old woman presented with pain at the volar side of the left wrist. She had undergone surgery for carpal tunnel syndrome of the left wrist in 2006 at another hospital. After the surgery, she reported to us that she had a fluid collection in the operation site after the surgery. She had received revision surgery four times during one year. Tissue culture and PCR analysis for mycobacteria were performed, but the results were all negative. The patient was afebrile and in good general health.

Radiographs and MRI of the left hand were obtained one year after the initial surgery. The chest radiographs was normal and the hand radiographs revealed no bony abnormality. However, MRI of the hand showed a

proliferative tenosynovitis of the flexor tendon sheath along the tracts of the flexor digitorum tendons involving from the wrist and mainly to the distal portion of the second digit. This lesion showed diffuse peripheral enhancement. The flexor tendons were intact and there were not any definite signal changes of the bone marrow (Fig. 2). At that time, her wrist pain was tolerable, so she did not receive any specific medical treatment and she'd just taken painkiller for three years.

As time went by, volar side wrist pain and a restricted range of motion of the left wrist had developed. She finally visited our hospital for further management and evaluation in 2010. On physical examination, there was 4 cm palpable mass-like lesion in the volar side of the left wrist, and tenderness on that lesion was also present. The blood tests showed an erythrocyte sedimentation rate of 27 mm/hr and a C-reactive protein level of 1.06 mg/L; the general hematologic and biochemical parameters were in the

normal ranges. A radiograph revealed bony erosions along the margin of the left radiocarpal bones with osteoporotic change and the distance between the scaphoid and lunate was widened over 4 mm. On the oblique view, there was a 4 cm round bulging mass at the volar aspect (Fig. 2). MRI was not performed at that time.

Thus, considering her slowly progressing clinical history and the image findings such as multiple erosive bone lesions with a bulged soft-tissue lesion, tuberculous arthritis with an abscess pocket was the most possible diagnosis even though the tuberculosis previous tests were negative. Under the impression of tuberculous arthritis, she underwent tenosynovectomy and mass excision. The biopsy material from the soft tissue was sent for microbiological and pathological analysis. During the operation, the clinician found a capsule containing a high viscosity yellowish content, but there was no pus. An abscess in the proximal scapholunate ligament, as well as chronic inflamed soft tissue along the flexor tendons and synovium, were also found.

The acid-fast stain, the culture and PCR analysis for mycobacteria were negative. The histology revealed fibroadipose tissue with chronic inflammation. A few weeks later, atypical acid-fast bacilli were grown and these were identified to be *Mycobacterium intracellulare*.

The patient was prescribed of the same medication as the first patient, and she is currently being followed, but she still suffers from wrist pain.

## DISCUSSION

Nontuberculous mycobacteria tenosynovitis is a rare condition, but tenosynovitis of the hand is a typical clinical manifestation of this malady (2). Various slow and fast growing mycobacteria have been isolated with *Mycobacterium marinum* (fish tank finger) and *Mycobacterium kansasii* being the most frequent causes of NTM tenosynovitis (3, 9). Less frequently, *M. avium* complex, *M. szulgai*, *M. terrae*, *M. fortuitum*, *M. chelonae*, *M. abscessus*, *M. malmoense* and *M. xenopi* are found (1, 3). These infections mostly appear to be the result of previous trauma, surgical procedures, corticosteroid injection or non-apparent inoculation (water contamination). Some, but not all of these patients, have underlying diseases that cause various degrees of immunosuppression. Hematogenous spread may occur from a primary focus in the lungs. The gastrointestinal tract may also be a portal of entry for

atypical mycobacteria because some of these organisms may colonize the mouths of normal individuals (1-3).

The clinical manifestations and radiographic and histologic features of bone, joint and soft tissue infections by NTM are usually indistinguishable from those of tuberculosis. They are all diseases characterized by a gradual and insidious onset with slowly progressive enlargement of the involved tendon, followed by pain and limitation of motion. The symptoms are so mild that the disease is often well advanced before the patient seeks treatment (10, 11). The imaging appearances of NTM tenosynovitis in the medical literature were frequently reported as exuberant tenosynovitis, which rarely involves the underlying muscles and bony structures, with preserved joint space and without tendon tear even in the long-standing phase (6-8).

Yet in our first case, the extensor tendon of the second digit was focally disrupted and marrow change of bone adjacent to the disrupted tendon was noted on MRI. Because he had a shallow penetrating injury on the third finger, the disrupted tendon and marrow change of the bone was not directly caused by the shallow penetrating injury itself. Also, in our second patient, even though misalignment of an adjacent joint space and erosion/destruction of the articular margin were not obviously detected on the initial radiography, eventually marginal bone erosions with osteoporosis of the radiocarpal bones and scapholunate joint widening were present in the long-standing phase. Based on the imaging findings of our cases, we found that the imaging findings of our cases did not correspond with those of the majority of the previous reports regarding NTM tenosynovitis. Of course, there is no case report in the literature showing such aggressive imaging findings. Thus, we assume that NTM tenosynovitis can behave aggressively enough to cause tendon tear and bony involvement.

Nevertheless, the imaging of NTM infections is nonspecific in the early stages as well as in the late stages and the imaging may resemble other granulomatous or mycobacterial infections. Thus, to determine the final diagnosis, it is useful to perform open biopsy and appropriate atypical mycobacterial cultures so the optimal combination therapy and duration of treatment can be administered.

Our case highlights NTM tenosynovitis as an important part of the differential diagnosis of atypical arthritis, which can lead to disruption of musculoskeletal structures such as tendon tear, bone marrow involvement and misalignment of the articular space. To the best of our knowledge, this is

the first report showing the distinctive imaging features of NTM tenosynovitis in the late stage, and these features are different from those of the previously reported cases of NTM tenosynovitis.

Even though these infections are often indolent and they are persistent, they may cause significant local tissue damage. Thus, timely detection of NTM infection followed by treatment with an appropriate combination of antibiotics is important to prevent permanent disability of the infected site.

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