—Case Reports—

Purulent Flexor Tendon Rupture of the Hand due to *Mycobacterium abscessus* Infection: A Case Report and Review of the Literature

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Mycobacterium abscessus infection of the upper extremities is uncommon. However, *M abscessus* can cause severe chronic tenosynovitis, and delayed diagnosis may result in poor outcomes. We describe an unusual clinical case of purulent flexor tendon synovitis followed by subcutaneous tendon rupture due to *M abscessus* infection in a patient with diabetes mellitus. A 76-year-old man presented to our hospital with painful, erythematous swelling over his left fourth finger. On physical examination, the left fourth finger was swollen and reddish, with persistent exudate from the surgical scar. The left elbow was also swollen and reddish with persistent discharge, which was consistent with olecranon bursitis. The patient was unable to flex his left fourth finger, and the passive range of motion of the finger was also restricted. The physical examination findings and patient history suggested purulent flexor tendinitis. His infection healed after radical debridement of necrotic tissue and administration of antibiotics effective against *M abscessus*. Third-stage flexor reconstruction restored the function of the fourth finger. The combination of surgical debridement and chemotherapy was the most effective treatment for mycobacterial tenosynovitis. This case shows that *M abscessus* can cause chronic severe purulent tenosynovitis and flexor tendon rupture after tendon surgery. Although early diagnosis and combination treatment with debridement and chemotherapy might improve outcomes by limiting the severity and duration of damage to the flexor synovial system, late-presenting patients require combined radical debridement of necrotic tissue and aggressive chemotherapy followed by staged flexor tendon reconstruction.
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Key words: flexor tendon rupture, *Mycobacterium abscessus*, non-tuberculous mycobacterial infection, purulent tenosynovitis, staged reconstruction

Introduction

Mycobacterium abscessus is a rapidly growing *Mycobacterium* species found in soil, plants, and aqueous environments[1–4]. *M abscessus* has been associated with chronic lung infections, endocarditis, keratitis, and disseminated disease in immunocompromised hosts and occasionally causes post-traumatic infection of the skin, soft tissue, and long bones[1]. Although *M abscessus* is an uncommon cause of infection in the upper extremities, it can have a chronic, destructive course that results in extensive necrotic tenosynovitis[4–7]. We present a rare case of necrotic flexor tendon rupture of the left fourth finger due to *M abscessus* infection in a 76-year-old man with diabetes mellitus and review published reports of similar cases.

Case Report

A 76-year-old man presented to our hospital with painful, erythematous swelling over his left fourth finger. His

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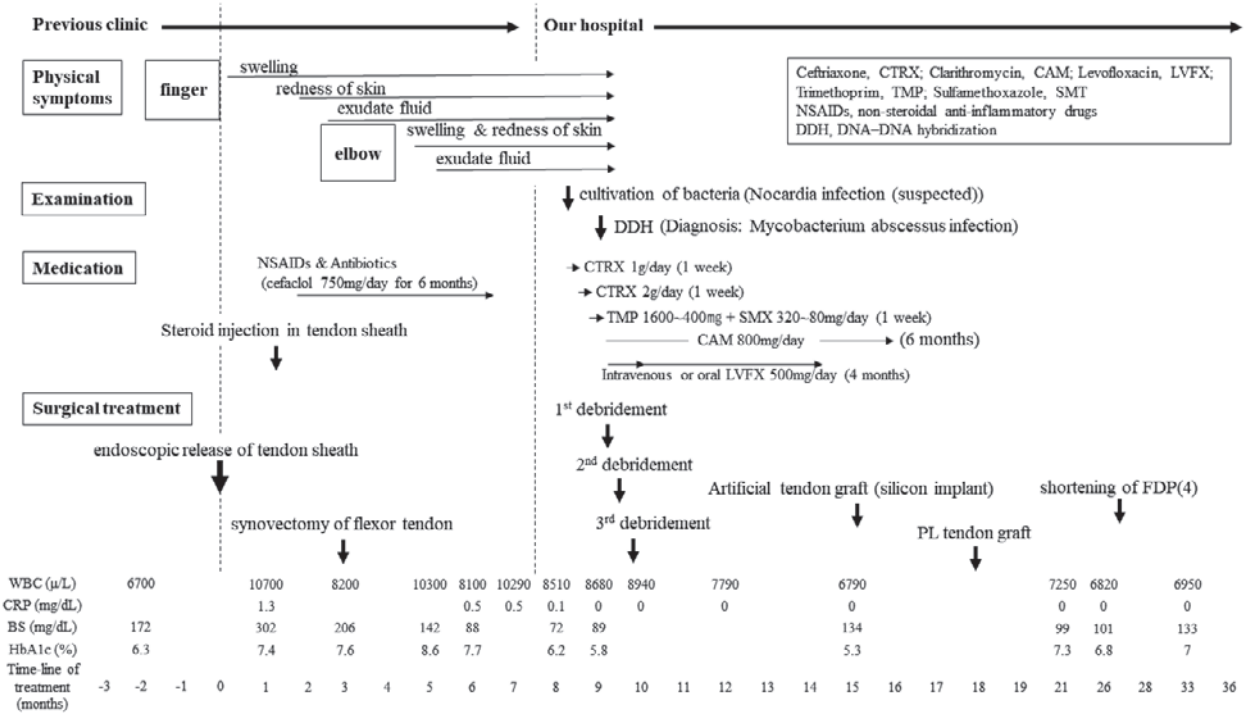

Fig. 1 Timeline of clinical course.

treatment timeline is shown in **Figure 1**. He was a retired businessman who had occasionally played golf, but pain and snapping of his left fourth finger had impaired his ability to perform daily life activities. He had a history of diabetes mellitus for more than 5 years, and his blood glucose level was approximately 200 mg/dL without medication for diabetes mellitus. He had not received any injected corticosteroid for his fingers. The trigger finger of his left fourth finger was treated via endoscopic release of the tendon sheath at another clinic. Four weeks after the first operation, the fourth finger was still swollen and was injected with corticosteroids. The swelling of the fourth finger temporarily improved but recurred 8 weeks postoperatively. Purulent synovitis was suspected, and the patient was prescribed cefaclor (750 mg/day) and an NSAID (loxoprofen sodium hydrate 180 mg/day). Eleven weeks postoperatively, synovectomy of the flexor tendon was performed, but erythema and swelling at the surgical site gradually increased, extending from the left palm to the palmar aspect of the distal phalange of the left fourth finger. The patient presented at our hospital 7 months postoperatively for investigation of his left upper extremity.

On physical examination, the left fourth finger was swollen and reddish, with persistent exudate from the surgical scar. The left elbow was also swollen and reddish with persistent discharge, which was consistent with olecranon bursitis. The patient was unable to flex his left fourth finger, and the passive range of motion of the finger was also restricted. His white blood cell count was 10.3×10^3/L, C-reactive protein level was 1.2 mg/L, HbA1c was 6.2%, and blood glucose level was 72 mg/dL.

The physical examination findings and patient history suggested purulent flexor tendinitis and pyogenic bursitis of the elbow joint. Several samples of the exudate from the scar on the left fourth finger and bursitis fluid of the left elbow were collected with swabs or sterilized tubes and sent to the microbiology laboratory for pathogen detection and identification. Ceftriaxone 1 g/day was administered initially but was increased to 2 g/day because of the persistent exudate.

Magnetic resonance imaging at 7.5 months after endoscopic release of the tendon sheath revealed swelling of the left fourth finger and thumb, with high signal intensity on an enhanced T1-weighted image (**Fig. 2A-F**). There was a hypointense area around the flexor tendons on enhanced T1-weighted imagery, indicating the presence of a liquid reservoir and abscess formation.

Initial swab results revealed a bacillus positive for Kinyoun acid-fast stain. The culture results suggested infection due to *Nocardia* species, and oral trimethoprim 1,600 mg daily and sulfamethoxazole 320 mg daily were administered. However, after a few days, because of dis-

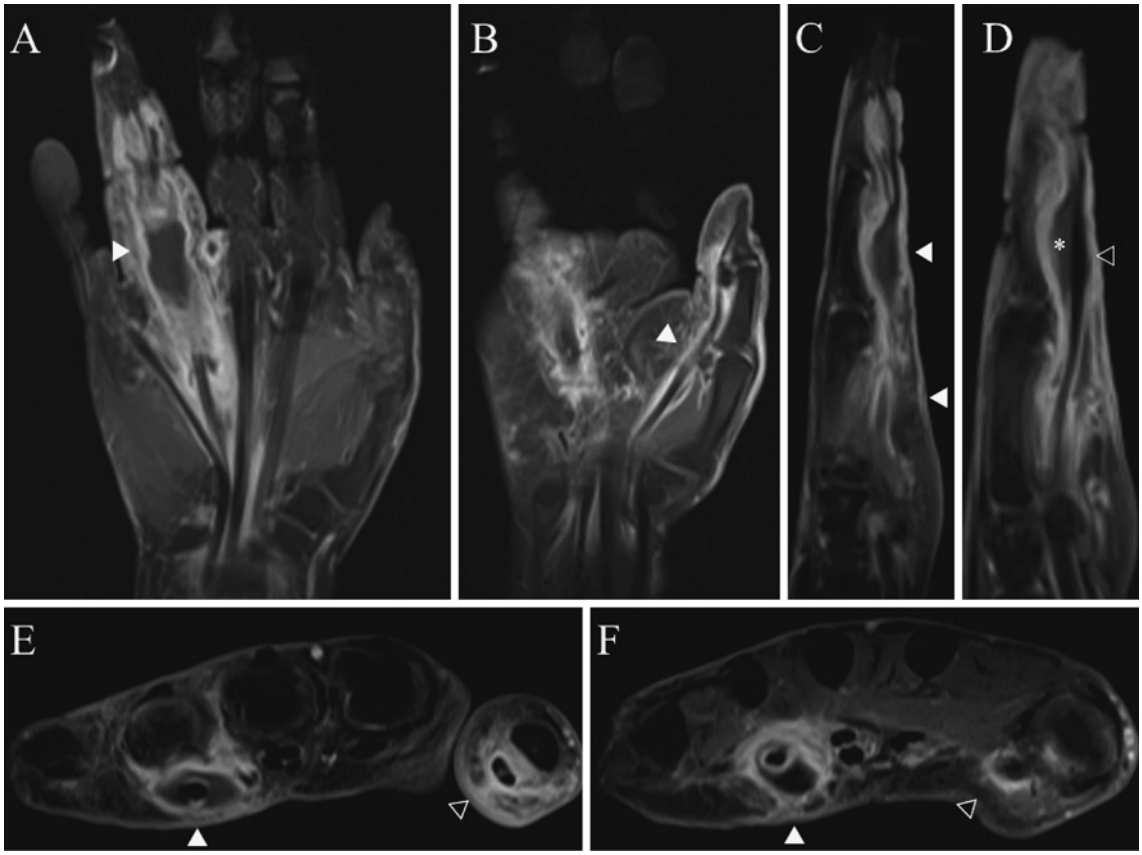

Fig. 2 Magnetic resonance image of the left hand showing enlargement and swelling of the left fourth finger and thumb, with high signal intensity on enhanced T1-weighted images.
(A, B) Coronal view showing an enlarged and swollen fourth finger (white arrowhead) and thumb (white arrowhead) with high signal intensity on enhanced T1-weighted images. (C, D) Sagittal magnetic resonance image showing the ruptured flexor digitorum superficialis of the fourth finger and enlarged flexor digitorum profundus on enhanced T1-weighted images. (C) The flexor digitorum superficialis (white arrowhead), which had low signal intensity, was ruptured and enlarged by pyogenic synovium. (D) The flexor digitorum profundus (black arrowhead), which had low signal intensity, was barely continuous with the surrounding diffuse low-intensity area, which indicates pyogenic synovium (asterisk). Axial view showing the flexor tendons of the fourth finger (white arrowhead) and thumb (black arrowhead) surrounded by enhanced infectious synovitis in the distal palm (E) and carpal tunnel (F) on enhanced T1-weighted images.

turbance of renal function, the doses of oral trimethoprim and sulfamethoxazole were decreased to 400 mg and 80 mg per day, respectively. One week after the culture test, DNA-DNA hybridization[2] revealed *M abscessus* infection. Because the previous diagnosis of infection due to *Nocardia* species was shown to be incorrect, sulfamethoxazole was withdrawn. There was no improvement in the swelling of the left elbow or fourth finger. Moreover, the left thumb gradually became swollen. The patient provided written informed consent for surgery and publication of his anonymized images in this case report.

At 8 months after endoscopic release of the tendon sheath, surgery was performed to treat the purulent tenosynovitis and bursitis. Preoperative photos showed swollen and reddish skin of the left fourth finger (**Fig. 3A-C**) and left elbow (**Fig. 3D**). A zigzag incision was made on the volar aspect of the left fourth finger through the distal phalangeal joint to the distal forearm. This revealed turbid fluid around the flexor tendon, with pyogenic tenosynovitis extending from the distal interphalangeal joint to the midpalmar region (**Fig. 4A**). The flexor tendon of the thumb was surrounded by turbid pyogenic synovium but was not necrotic (**Fig. 4B**). The pulley of the flexor tendon of the fourth finger had disappeared, and the flexor digitorum superficialis was necrotic and ruptured (**Fig. 4C**). The flexor digitorum profundus of the fourth finger was barely continuous, necrotic, and surrounded by abscesses (**Fig. 4D**). Radical debridement of the necrotic tissue, including the pyogenic synovium, tendon sheath, flexor pulley, and necrotic flexor tendons,

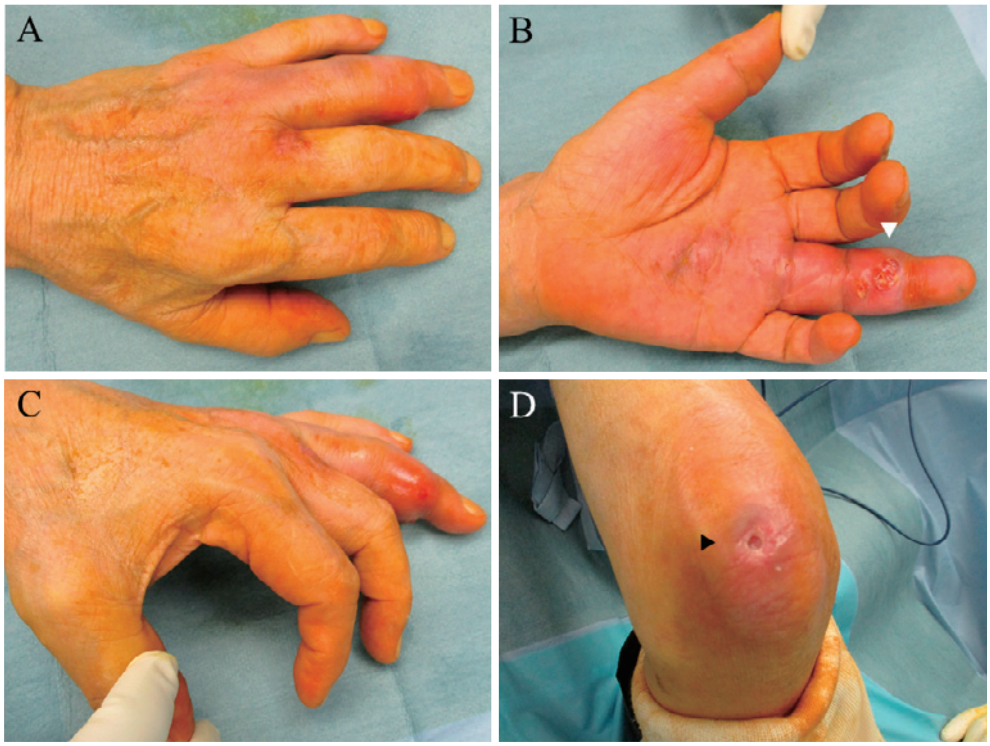

Fig. 3 Purulent synovitis of the left fourth digit of a 76-year-old man.
Preoperative dorsal (A), palmar (B), and lateral (C) clinical photographs showing the swollen and reddish skin of the fourth finger (white arrowhead). (D) Preoperative clinical photograph showing the swollen and reddish skin of the posterior elbow with exudate from a fistula (black arrowhead).

was performed. The flexor tendon of the thumb was preserved. Tissue samples were sent for histological and microbiological analysis, including acid-fast bacilli smear and culture.

Postoperatively, oral clarithromycin and intravenous levofloxacin were administered for 3 weeks. Histology showed granulomatous lesions with multinucleated giant cells. Stains for bacteria, fungi, and mycobacteria were negative. The bacillus was positive for Kinyoun acid-fast stain, and *Mycobacterium* infection was suspected. A tuberculosis molecular study was negative for *Mycobacterium tuberculosis*, but isolated colonies of *M abscessus*, which was sensitive to clarithromycin and levofloxacin, grew after prolonged incubation. After 2 planed debridement operations, the antibiotics and surgery achieved a good response, with resolution of swelling and improvement of the wound condition; wound healing was subsequently uneventfully completed in 3 weeks. Four weeks after the first debridement, the patient was discharged, and passive range of motion of the finger was started. He received combination therapy with oral levofloxacin hydrate and clarithromycin for 3 months. After that, clarithromycin monotherapy was administered for an additional 2 months. At 6 months after the first debridement in our hospital, he had recovered a full range of total passive motion. Furthermore, his white cell count was 6.7×10^3/L, C-reactive protein level was <0.05 mg/L, HbA1c was 5.3%, and blood glucose level was 134 mg/dL.

Eight months after the first debridement, an artificial tendon was placed and the A2 and A4 flexor pulleys were reconstructed with Leeds-Keio artificial ligament II (Yufu Itonaga Co., Ltd.; Tokyo, Japan) through an incision from the distal phalangeal joint to the distal forearm. Three months after placement of the artificial tendon, the artificial tendon was replaced with an autografted palmaris longus tendon, which was used as a long graft to reconstruct the flexor tendon of the fourth digit. The distal end of the palmaris longus was sutured to the distal phalangeal base with suture anchors. Subsequently, the proximal end of the grafted palmaris longus was connected to the flexor digitorum profundus of the third finger with interlacing sutures at the distal forearm. Although active range of motion exercises of the left fingers were performed, the left fourth finger had a residual flexion lag. Additional surgery was performed to adjust

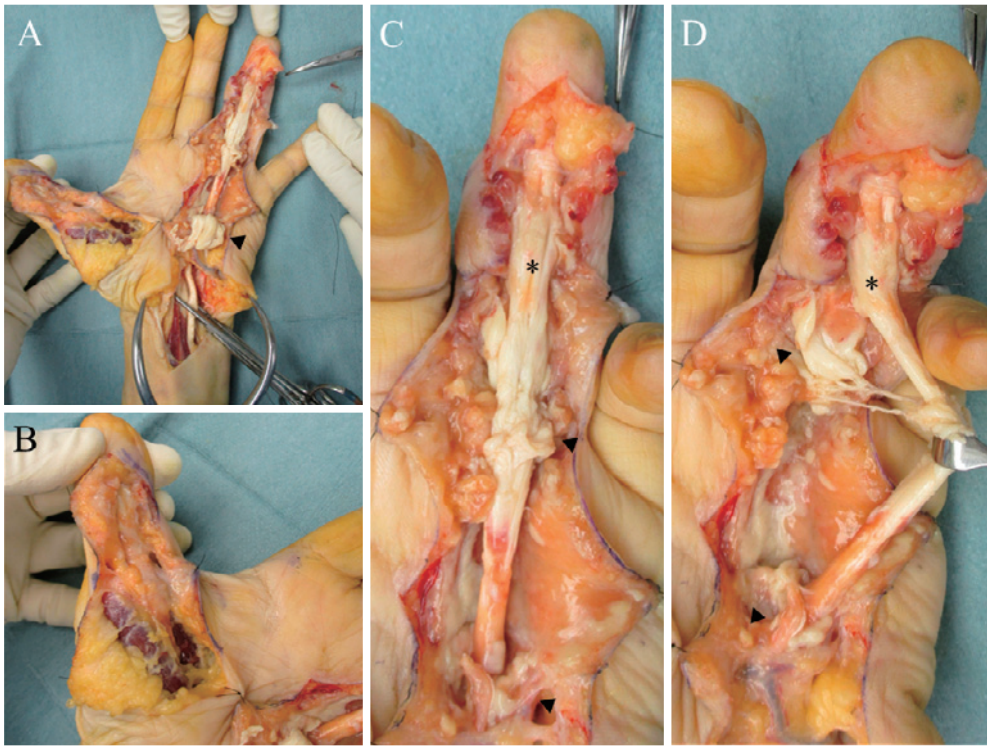

Fig. 4 Perioperative photographs of the left hand.
(A, C, D) Exposure of the fourth tendon sheath revealed inflamed synovium around the ruptured flexor digitorum superficialis (black arrowhead) and necrotic flexor digitorum profundus (asterisk) of the left fourth finger. The flexor tendon of the thumb was surrounded by turbid pyogenic synovium but was not necrotic (B).

the tension of the flexor of the fourth finger at 9 months after the PL tendon graft. Under local anesthesia, the reconstructed flexor of the fourth finger was exposed and found to be redundant in the distal forearm. The proximal stump of the palmaris longus was resutured to the long flexor digitorum profundus with a slightly tighter tension than that of the third finger. Active range of motion exercises of the left fingers were performed after the additional operation.

There was no evidence of local recurrence after 3.5 years of follow-up. The white blood cell count was 7.9 × 10^3/L and C-reactive protein level was <0.05 mg/L. At final follow-up, the total active range of motion of the fourth finger was 190° (92% of the contralateral finger). The ranges of motion of the distal interphalangeal and proximal interphalangeal joint on the fourth finger were 70° and 50°, respectively (**Fig. 5A-D**). The pulp-palm distance was 21 mm (contralateral pulp-palm distance 10 mm). The grasp strength was 17 kg (77% of the unaffected hand). The wrist reached a full range of motion without pain, and there was full active extension of the fourth digit. The final evaluation of the fourth finger range of motion was 69%, which was classified as fair using the original Strickland classification. There was no residual pain and the patient was able to play golf. Regarding patient-based evaluation scores, the Disabilities of the Arm, Shoulder and Hand Score and The Hand20 were 17 and 27.5, respectively.

Discussion

M abscessus hand infections are uncommon and usually occur after a medical intervention or in immunocompromised patients[1–4]. To our knowledge, there are only 5 reported cases in the English literature of *M abscessus* infection causing tendinitis or synovitis of the flexor tendon of the hand (**Table 1**)[4,7–9]. Moreover, our case is the first of purulent flexor tendon rupture due to *M abscessus* infection. The affected patients had a history of contaminated injection, trauma caused by fish or shrimp, or surgical wound resulting in chronic granulomatous flexor tenosynovitis. The course is usually chronic and virulent, with gradual progression of local erythematous swelling and tenderness that may persist for several months.

Although definitive diagnosis of *M abscessus* infection requires isolation of the pathogen from tissue culture, conventional *Mycobacterium* culture usually needs several

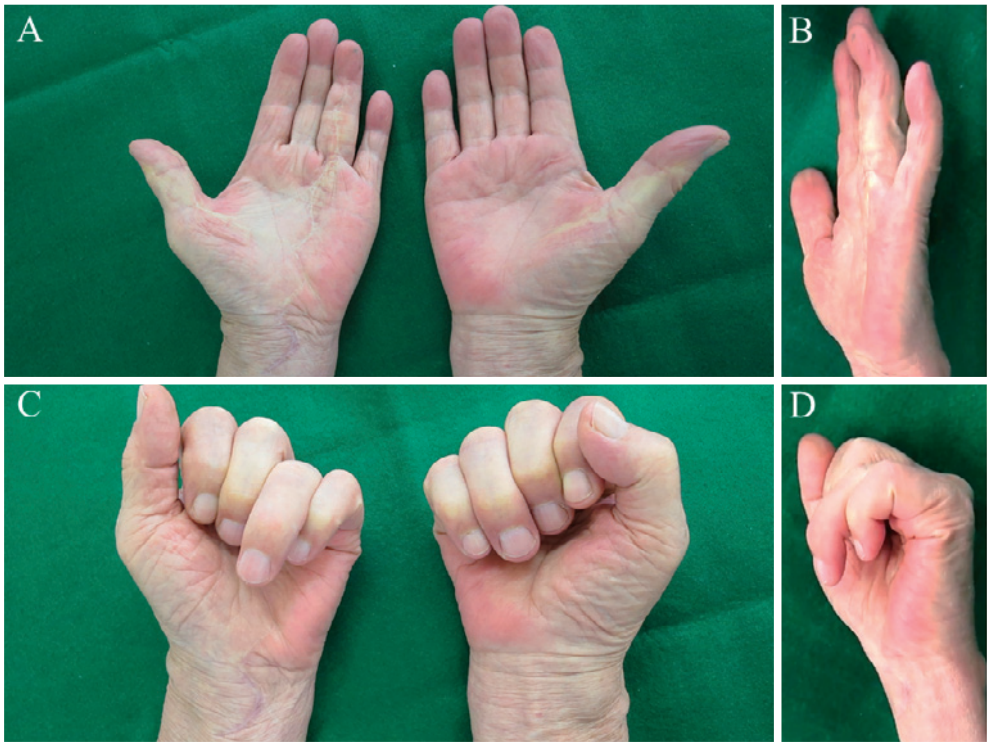

Fig. 5 Photograph of the left hand at final follow-up.
The bilateral digits in full extension (A) and full flexion (C). Lateral view of the left fingers in full extension (B) and full flexion (D).

weeks to identify the exact species and gather drug-sensitivity data. Moreover, although Kinyoun acid-fast stain is a useful procedure to detect acid-fast species of the bacterial genera *Mycobacterium* and *Nocardia* and the apicomplexan genus *Cryptosporidium*, discriminating *Mycobacterium* from *Nocardia* and *Cryptosporidium* is impossible. To overcome this limitation, several commercial non-radiolabeled DNA probes are available for identification of certain *Mycobacterium* species. In our case, DNA-DNA hybridization was used to identify *M abscessus*. Because an accurate diagnosis is critical for implementing an effective treatment plan, early differentiation of *Mycobacterium* species is essential.

Soft tissue infections caused by *M tuberculosis* and those caused by non-tuberculous mycobacteria have similar characteristics, but the treatment regimens differ. Abscess formation with substantial necrotic granulomatous tissue usually requires surgical debridement. In addition to debridement, adequate antimycobacterial medication is crucial. Furthermore, treatment of non-tuberculous mycobacteria is difficult, as these organisms are not susceptible to most anti-tuberculous drugs, and multi-agent drug therapy is often necessary[6]. Treatment of *M abscessus* infection depends on antimicrobial sensitivities and clinical appearance. *M abscessus* infections are susceptible to monotherapy with clarithromycin, but this leads to rapid development of resistance[10]. Thus, for soft tissue infections caused by non-tuberculous mycobacteria, macrolide-based therapy with parenteral medications (amikacin, cefoxitin, or imipenem) is suggested[6]. Although the optimal duration of antibiotic administration for hand infections caused by non-tuberculous mycobacteria is unknown, a minimum of 6 to 9 months is recommended[6].

Our case shows that *M abscessus* can cause chronic severe purulent tenosynovitis and flexor tendon rupture after tendon surgery. Initially, positivity for Kinyoun acid-fast stain suggested purulent tendinitis of the left fourth finger and elbow bursitis due to infection by a *Nocardia* species, and sulfamethoxazole was administered. However, isolated colonies of *M abscessus* were identified after prolonged incubation. Thus, sulfamethoxazole was replaced with a combination of clarithromycin and levofloxacin. The infection healed after radical debridement of necrotic tissue and administration of antibiotics effective against *M abscessus*. Staged flexor reconstruction restored the function of the fourth finger. Although early diagnosis and aggressive treatment might improve outcomes by limiting the severity and duration of damage to the flexor synovial system, late-presenting patients re-

Table 1 Summary of known published reports of tendinitis or synovitis of the flexor tendon caused by *Mycobacterium abscesses* infection

Authors	Year	No. of cases	Age (years) /sex	Finger (side)	Occupation	Underlying diseases	Precipitating factor	Specific cause	Interval from onset to diagnosis (months)	Surgery	Chemotherapy / duration of chemotherapy (months)
Zenone T. et al.[6]	1998	1	44/F	Second (right)	Unknown	Anorexia nervosa, neutropenia, CD4+ lymphocytopenia	Local cortisone injection	No	5	None	CAM and CPFX (12)
Kang G.C.[7]	2010	2	57/F	Second (right)	Fishmonger	None	Fish handling	No	3	Synovectomy of the flexor	CPFX and AZM (6)
			61/M	Third (left)	Frozen fish handler	None	Fish handling	Surgery, release of the tendon sheath	3	Synovectomy of the flexor	CAM (9)
Park J.W. et al.[4]	2014	1	53/M	No data	No data	None	Trauma	Abrasion	2	Synovectomy of the flexor	EMB, RFP, and CAM (4)
Zheng S.[9]	2018	1	70/F	Third (right)	Housewife	None	Trauma	Scalding injury	5	Debridement	CAM and CLF (4)
Present case	2019	1	69/F	Fourth (left)	Retired businessman	Diabetes mellitus	Iatrogenic	Surgery, release of the tendon sheath	7	Removal of necrotic tissue followed by tendon reconstruction	CAM (6) and LVFX (4)

AZM, azithromycin; CLF, clofazimine; CAM, clarithromycin; CPFX, ciprofloxacin; EMB, ethambutol; LVFX, levofloxacin; MIN, minocycline; RFP, rifampin; STR, streptomycin

quire combination therapy followed by staged reconstruction of the flexor tendon. The combination of surgical debridement and chemotherapy is the most effective treatment for mycobacterial tenosynovitis.

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