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Rare Complications in Crohn's Disease: Diagnostic and Therapeutic Challenge of Dermal Abscesses Caused by Nontuberculous Mycobacteria Following Infliximab Therapy, and Fluoroquinolone-Related Achilles Tendon Rupture

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Patient: Male, 57-year-old
Final Diagnosis: Dermal abscesses caused by *Mycobacterium mucogenicum*
Symptoms: Dermal abscesses
Clinical Procedure: —
Specialty: Infectious Diseases

Objective: Unusual clinical course

Background: *Mycobacterium mucogenicum* is a rapidly growing nontuberculous mycobacterium that rarely causes cutaneous disease. Patients receiving tumor necrosis factor–alpha (TNF-α) inhibitors are at increased risk for opportunistic and granulomatous infections, including nontuberculous mycobacteria. To our knowledge, this is the first reported case of a cutaneous infection caused by *M. mucogenicum* in a patient receiving infliximab for inflammatory bowel disease (IBD).

Case Report: We describe a 57-year-old man with Crohn's disease on infliximab who presented with dermal abscesses, ulcerations, and lymphangitis. The patient initially received empiric levofloxacin therapy and developed an early Achilles tendon rupture within 4 days, likely related to underlying enthesiopathy and fluoroquinolone exposure. Microbiologic evaluation confirmed *M. mucogenicum*. He was treated with trimethoprim-sulfamethoxazole and clarithromycin, while infliximab was continued due to active disease. The patient showed clinical improvement with resolution of systemic symptoms and progressive healing of skin lesions. Antimicrobial therapy was continued for 6 months with sustained response.

Conclusions: This case highlights the importance of early recognition of nontuberculous mycobacterial infections in patients receiving TNF-α inhibitors. Combined therapy with clarithromycin and trimethoprim-sulfamethoxazole was safe and effective despite continued infliximab use. Our case suggests that fluoroquinolones should be used with caution in patients with IBD and suspected enthesiopathy due to the potential risk of tendon rupture.

Keywords: Abscess • Inflammatory Bowel Disease • Opportunistic Infections • *Mycobacterium mucogenicum* • Fluoroquinolones Adverse Effects • Tendon Rupture

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Introduction

Mycobacterium mucogenicum is a rapidly growing nontuberculous mycobacteria (NTM), first identified in 1982 as *M. chelonae*-like organism (MCLO) during an outbreak of peritonitis from 2 dialysis units [1]. It is a Gram-positive curved bacillus that is acid and alcohol fast, grows optimally at 28-37°C, and produces smooth, mucoid colonies in 2-4 days on Middlebrook 7H10 or Trypticase soy agar. In 1995, the species was named *M. mucogenicum* due to the mucoid nature of its colonies [1]. The organism can survive in tap water due to its ability to tolerate disinfectants and chlorination [2] and can easily contaminate central venous catheters (CVCs) and infect posttraumatic wounds due to the production of protective mucoid biofilm. Hence, patients with an indwelling CVC need to take precautions while taking a bath in tap water.

Patients being treated with TNF-α inhibitors (infliximab, etanercept) are also at increased risk for infections with granulomatous organisms including tuberculosis (TB)-causing mycobacteria and NTM since TNF-α inhibitors compromise granuloma formation, apoptosis, and the cytotoxic effect of macrophages against these pathogens [3]. Management of opportunistic infections in patients with inflammatory bowel disease (IBD) poses a therapeutic dilemma, particularly when balancing the risk of uncontrolled intestinal inflammation against progression of infection during immunosuppression. Only a single case of *M. mucogenicum* infection has been reported in the setting of treatment with etanercept [4] and has never been reported with infliximab use. We report the first case of *M. mucogenicum* skin infection in the setting of infliximab usage for IBD.

In addition, fluoroquinolones, frequently used empirically for skin and soft tissue infections, are associated with tendinopathy and tendon rupture, particularly involving the Achilles tendon. Patients with IBD may have underlying enthesiopathy as an extra-intestinal manifestation, potentially increasing susceptibility to tendon injury [5,6]. Our patient also developed an early complication of Achilles tendon rupture following 4 days of levofloxacin therapy in the setting of preexisting undiagnosed Crohn's-related enthesiopathy. This case highlights the complex interplay between infection, immunosuppression, and medication-related adverse effects in patients with IBD.

Case Report

A 57-year-old White male patient with a history of type 2 diabetes mellitus was initially diagnosed with ulcerative colitis following colonoscopy and histopathology in 2019. The patient remained in remission for 5 years, having alternate day bowel movement without blood or mucus with lialda (mesalamine) 2.4 g daily as oral formulation and enema. In April 2024, he developed an ileal abscess, leading to a diagnosis of Crohn's disease. This required a distal ileum resection, followed by a long Hartman's pouch and end ileostomy. In June 2024, he was initiated on induction therapy with intravenous infliximab at a dose of 5 mg/kg and received 3 doses by the end of July 2024. Prior to the third infusion, his trough infliximab level was very low (1.9). Two weeks after the third dose, he developed flu-like symptoms followed by a small papule on the medial aspect of the right thigh that slowly progressed to a papule and ruptured after applying salve, to become a fairly large, dime-sized ulcerated area (**Figure 1A**). The ulcer was painful, and had erythematous margins and purulent discharge and did not respond to oral antibiotics including clindamycin and doxycycline. Subsequently, the patient developed multiple small pruritic pustules distributed over the face, forehead, upper extremities, groin, and genital region, with varying stages of evolution including papules, pustules, and ulcerative lesions. Most of the lesions on his face resolved spontaneously. He also had constitutional symptoms including intermittent pyrexia, fatigue, and increased ileostomy output. He also lost 20 pounds in 2 weeks and complained of pain in the right Achilles tendon. There was no history of arthralgias or bug bite. Due to these symptoms, a fourth dose of infliximab was withheld.

On examination, the patient was tachycardic and had splinter hemorrhages on his nail beds. There was a small, non-tender papule at the tip of the penis, and no lymphadenopathy was noted. The patient had multiple active ulcers on the anterior right shin (**Figure 1C**), lateral right ankle, and right forearm. Healing nodular lesions were observed on the left side of the face. A painful ulcerated lesion on the medial aspect of the right thigh exhibited purulent drainage and surrounding erythema (**Figure 1A**). Initial laboratory investigations (**Table 1**) were significant for leukocytosis (21 900/cc), thrombocytosis (531 000/cc), elevated C-reactive protein (CRP) (18 mg/L), and elevated erythrocyte sedimentation rate (ESR) (29 mm/hr). Antinuclear antibody (ANA), antineutrophil cytoplasmic antibody (ANCA), and rheumatoid factor results were negative. Blood cultures were sterile after 5 days and 2 wound cultures from the ulcers showed no growth. Workup for sexually transmitted infections including chlamydia, gonorrhea, syphilis, and HIV was also negative. There was no evidence of deep vein thrombosis on lower extremity doppler ultrasound (**Table 2**). Considering the possibilities of pyoderma gangrenosum, ecthyma gangrenosum, and fungal or mycobacterial infection, multiple biopsies from the lesions, including the lesion from the uninvolved edge of the large thigh lesion, were taken and sent for histopathology tests, including acid-fast (AFB) staining for mycobacteria, fungal staining, and culture. Broth microdilution culture-based testing for antibiotic sensitivity was also conducted. Pyoderma gangrenosum was considered given the patient's IBD history; however, absence of characteristic undermined violaceous borders and lack of pathergy response

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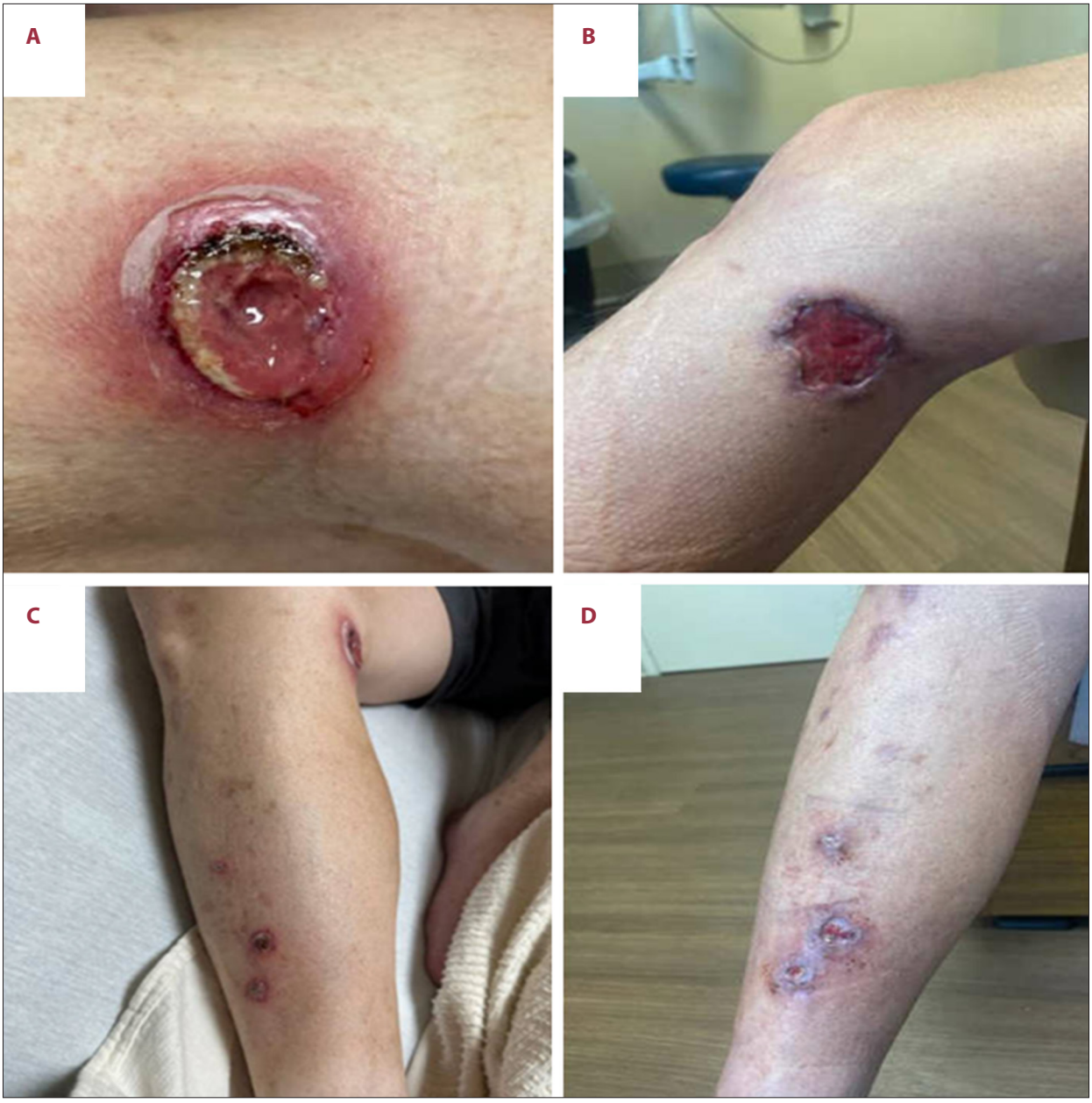

Figure 1. Effect of treatment on right thigh and shin ulcers. Ulcer on the right medial thigh pre-treatment (**A**) and post-treatment (**B**). Ulcer on the right shin pre-treatment (**C**) and post-treatment (**D**).

prompted further microbiologic investigation. Ecthyma gangrenosum was considered less likely due to sterile blood cultures and absence of *Pseudomonas* growth. Negative fungal stains and cultures further narrowed the differential diagnosis toward atypical mycobacterial infection.

The patient was continued on broad-spectrum antibiotic therapy with IV cefepime, metronidazole, and vancomycin for a total of 4 days. He was then de-escalated to oral levofloxacin and doxycycline at the time of discharge, with a consideration of transition to high-dose topical steroids, in addition to infliximab for the management for severe pyoderma gangrenosum, if the workup for fungal, mycobacterial, and other bacterial etiologies was negative. The patient was also continued on daily medi-honey dressing and wound care follow-up.

During outpatient follow-up, the patient developed a painful partial tear in his left Achilles tendon, probably related to the combination of IBD-associated enthesiopathy and levofloxacin. This occurred 4 days after levofloxacin initiation. MRI of the ankles confirmed severe tendinosis in bilateral ankles and a partial longitudinal tear in the left ankle Achilles' tendon.

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Table 1. Laboratory investigations at admission, discharge, and follow-up.

Investigation	At admission	At discharge following 4 days of IV antibiotics	Follow-up after 4 weeks of bactrim and clarithromycin	Follow-up after 3 months of bactrim and clarithromycin
Hb	13.9	10.9	11.9	12.8
WBC	21.9	12.8	9.8	7.7
Platelet count	531	379	289	235
ESR	29			
CRP	18		1.2	2
Creatinine	1.14	0.72	1.27	1.25
eGFR	75	>90	66	67
AST	11	12	18	19
ALT	10	7	18	20
ALP	103	87	110	110
Sodium	130	136	137	
Potassium	5.2	4.1	5.1	
Calcium	10.1	8.9	9.2	
Albumin	3.8	3.2	3.7	4.1
Lactic acid	1.4			
Magnesium	2	1.7		
Phosphorus	2.8	2		
Urine analysis	Normal			
Urine drug screen	Negative			
Urine microalbumin/creatinine ratio	103			
HbA1c	5.8			

IV – intravenous; Hb – hemoglobin; WBC – white blood cells; ESR – erythrocyte sedimentation rate; CRP – C-reactive protein; eGFR – e glomerular filtration rate; AST – aspartate transaminase; ALT – alanine transaminase; ALP – alkaline phosphatase; HbA1c – glycated hemoglobin.

Culture from a biopsy of the right forearm lesion grew *Sphingomonas* species, which were susceptible to cefepime, ceftazidime, ciprofloxacin, gentamicin, levofloxacin, piperacillin-tazobactam, and trimethoprim-sulfamethoxazole. Given the absence of progressive lesions attributable to *Sphingomonas* and clinical improvement following targeted antimycobacterial therapy, this organism was considered a likely colonizer rather than the primary pathogen.

AFB culture from the biopsy of the right thigh ulcer grew *M. mucogenicum/phocaium* that were sensitive to cefoxitin, imipenem, moxifloxacin, clarithromycin, amikacin, trimethoprim-sulfamethoxazole, and linezolid. They showed intermediate sensitivity to ciprofloxacin, and were resistant to tobramycin and doxycycline. Histopathology demonstrated mixed acute and chronic inflammatory infiltrates with focal granulomatous inflammation and dermal necrosis. Special stains (AFB and Fite) highlighted rare acid-fast bacilli within dermal tissue, supporting the diagnosis of atypical mycobacterial infection.

A final diagnosis of mycobacterial dermal abscesses and ulcerations with lymphangitis in the setting of active Crohn's disease with enthesiopathy was considered. The patient was started on trimethoprim-sulfamethoxazole, double strength, twice a day, and clarithromycin 500 mg twice a day. Infliximab infusions were reinitiated at an increased dose of 10 mg/kg

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Table 2. Microbiology, immunology, and imaging workup results.

Investigation	Results
Rapid influenza A and B/COVID/RSV	Negative
Superficial swab aerobic/anaerobic cultures	No growth at 5 days
Legionella urine Ag	Negative
Urine chlamydia and gonorrhea	Negative
Syphilis screen- RPR	Non-reactive
HIV Ag/Ab	Non-reactive
Blood cultures x 2	Sterile at 5 days
ANA	Negative
ANCA (MPO, PR-3)	Negative (1.3, 1.2)
RA factor	<3.5
Hepatitis C antibody	Non-reactive
HBsAg	Non-reactive
Surgical pathology (right knee lesion)	Acutely inflamed benign skin with granulation tissue and necrosis
Biopsy specimen stains for fungal and AFB	Negative for fungal organisms and acid-fast bacilli
Aerobic culture	*Sphingomonas* – susceptible to cefipime, ceftazidime, gentamicin, ciprofloxacin, levofloxacin, piperacillin-tazobactam, trimethoprim-sulfamethoxazole
Fungal culture	Negative
AFB culture	*Mycobacterium mucogenicum/phocaium*
AFB susceptibility rapid-grower	Susceptible to cefoxitin, imipenem, moxifloxacin, clarithromycin, amikacin, TMP/SMX, linezolid. Intermediate sensitivity to ciprofloxacin. Resistant to tobramycin, doxycycline
Chest X-ray	Normal
CT lung cancer screening	Multiple pulmonary nodules
CT lower extremity	Superficial ulcer/laceration of the subcutaneous soft tissue of the posterior medial proximal left leg. No evidence of underlying abscess or organized fluid collection
CT abdomen pelvis	No abscess or fistula seen
Venous doppler	No evidence of deep vein thrombosis
2D echocardiography	Echogenicity noted on the non-coronary cusp- pulmonary valve, likely focal calcification vs vegetation vs mass
Transesophageal echocardiogram	Normal, no vegetation/mass/thrombus

COVID – corona virus disease; RSV – respiratory syncytial virus; Ag – antigen; Ab – antibody; RPR – rapid plasma reagent; ANA – anti-nuclear antibody; ANCA – anti neutrophil cytoplasmic antibody; MPO – myeloperoxidase; PR-3 – proteinase 3; RA factor – rheumatoid arthritis factor; HBsAg – hepatitis B surface antigen; AFB – acid-fast bacilli; TMP/SMX – Trimethoprim/sulfamethoxazole; CT – computed tomography.

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after a duration of 8 weeks with an objective to attain remission. Infliximab therapy was resumed prior to completion of antimycobacterial therapy after multidisciplinary risk–benefit discussion, for the following reasons: the infection was localized, appropriate dual antimicrobial therapy had been initiated, and the patient exhibited worsening Crohn's-related symptoms. Continuation of biologic therapy was deemed necessary to prevent severe IBD flare and further systemic compromise. The patient continued to have significant pain in his Achilles tendon that resolved after reinitiating the infliximab at 10 mg/kg. He became afebrile, with resolution of his other symptoms and semisolid bowel movements from his ileostomy, which was his baseline. After 4 weeks of trimethoprim-sulfamethoxazole and clarithromycin, the wounds started to heal with healthy granulation tissue (**Figure 1D**).

Chronologically, initial skin lesion development occurred 2 weeks after the third infliximab dose. The patient received empiric intravenous antibiotics during hospitalization, then transitioned to oral levofloxacin pending culture results, and developed Achilles tendon symptoms within 4 days of levofloxacin exposure. Microbiologic diagnosis of *M. mucogenicum* was established from biopsy cultures, and targeted antimycobacterial therapy was subsequently switched to trimethoprim-sulfamethoxazole and clarithromycin once *M. mucogenicum* susceptibility results became available, with subsequent significant clinical improvement.

During follow-up, all the skin lesions started to show signs of healing. The last lesion to heal was the one on the right medial aspect of the thigh (**Figure 1B**), which healed completely by 3 months. At the last follow-up visit, the patient showed complete resolution of his enthesiopathy-related ankle pain and he was able to ambulate.

As the patient was continued on infliximab infusions, dual therapy for *Mycobacterium* infection with trimethoprim-sulfamethoxazole and clarithromycin was planned to be continued for a prolonged course of 6 months.

Discussion

M. mucogenicum can cause a wide range of infections, including infections of the bloodstream, liver, respiratory system, peritoneum, skin and soft tissue, and central nervous system, as well as osteomyelitis, in both immunocompromised and immunocompetent hosts [2,7]. Cutaneous manifestations range from localized abscesses and ulcerations to nodular lesions with lymphangitic spread, particularly in immunocompromised hosts.

There have been various outbreaks of *M. mucogenicum* involving CVC- and hemodialysis-related infections, as well as bacteremia in bone marrow transplant patients due to water contamination [7]. Common underlying conditions associated with *M. mucogenicum* infection are malignancies, end-stage renal disease, cirrhosis, autoimmune diseases, sickle cell disease, and thalassemia [8]. Since *M. mucogenicum* forms biofilms on CVCs, management of CVC-associated infection includes the removal of the CVC along with long-term administration of antibiotics [8].

Choice of antibiotics and duration of therapy is mostly based on expert opinion as there are no standard guidelines. Like other rapidly growing NTM, *M. mucogenicum* is resistant to isoniazid and rifampin; however, it is susceptible to amikacin, cefoxitin, clarithromycin, ciprofloxacin, and imipenem, and shows variable susceptibility to doxycycline [2]. A few studies have shown good clinical outcomes with a combination of antibiotics for a duration of 4 weeks. Further, amikacin in combination with clarithromycin and imipenem or a combination of clarithromycin and a fluoroquinolone are associated with lower relapse rates. Hence, they can be used as an empiric regimen while awaiting sensitivity results [8].

We used levofloxacin empirically in our patient while awaiting sensitivity results. Our patient had IBD-associated enthesiopathy and developed a partial tear of his Achilles tendon after only 4 days of levofloxacin treatment. IBD-associated enthesiopathy arises from immune-mediated inflammation at tendon insertion sites, involving cytokines such as TNF-α and IL-23. This inflammatory milieu may weaken tendon integrity. Superimposed fluoroquinolone exposure may further impair collagen synthesis and increase matrix degradation, lowering the threshold for tendon rupture.

Fluoroquinolone-associated tendon tear most commonly involves the Achilles tendon (90%) and is bilateral in 44% of cases [5,6]. The onset of fluroquinolone-associated tendon tear is widely variable, with symptoms starting as early as 2 hours of the first dose or may be delayed for 6 months after discontinuing the treatment [5,6]. The median duration of onset of tendon tear is 6-8 days, with 85% of cases happening within the first month and 50% of cases reported after discontinuing the fluroquinolones [5,6]. Importantly, 20-30% of patients with fluoroquinolone-associated tendon tear have a history of use of an oral corticosteroid [5,6].

The mechanism of fluroquinolone-associated tendon tear remains unclear. There are multiple mechanisms that have been proposed without any definite evidence. The most clinically relevant mechanisms include direct tenocyte toxicity, oxidative stress–mediated collagen degradation, mitochondrial dysfunction, and dysregulation of matrix metalloproteinases (MMPs) resulting in reductions in both the diameter and amount of type I collagen fibrils, degenerative changes in tenocyte cells,

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with vacuole formation, organelle dilation, and apoptosis, all of which impair tendon remodeling [9].

Fluoroquinolones, being a DNA gyrase inhibitor, probably directly injure the tendons of weight-bearing joints by increasing necrosis and cellular apoptosis. Further, fluoroquinolones may also indirectly injure the tendons by release of nitric oxide and oxygen-derived free radicals which cause the degradation of collagen. Another possible mechanism is mitochondrial toxicity via inhibition of DNA topoisomerase II. Fluroquinolones, being chelators of cations, may also lead to depletion of divalent ions important for integrin receptor signaling [10]. One more mechanism proposed is alteration in collagen fibrils due to changes in regulation of MMPs [11]. Other risk factors predisposing patients to tendon rupture are age >60 years, non-obese individuals, diabetes, male sex, use of glucocorticoids, and renal failure [10,11]. Our patient was male, older than 60 years, and diabetic. Considering these risk factors in the setting of IBD-associated enthesiopathy, discontinuation of fluroquinolones and reinitiation of infliximab helped resolve the tendon tear without any surgical intervention in our patient.

We propose another mechanism of fluroquinolone-induced tendon rupture. Transforming growth factor-beta (TGF-β) plays an important role in collagen synthesis, extracellular matrix production, and regulation of MMPs, which are essential for tendon remodeling and healing. TGF-β also promotes the expression of tissue inhibitors of metalloproteinases (TIMPs), thereby maintaining a balance between matrix degradation and repair.

Some experimental studies suggest that fluoroquinolones may influence pathways involving TGF-β and MMP activity. For example, Huang et al demonstrated suppression of TGF-β–induced MMP-9 production in cancer cells, while Bourikas et al reported immunomodulatory effects of ciprofloxacin mediated through TGF-β1 signaling [12,13]. Additionally, prior studies have shown that TGF-β1 can enhance collagen and TIMP production while reducing MMP activity, supporting its role in extracellular matrix stability [10]. Furthermore, Mousavizadeh et al demonstrated that oxidized low-density lipoprotein (LDL) impairs TGF-β activity in human tendon cells, highlighting the vulnerability of this pathway in tendon biology [14].

However, these findings are derived from heterogeneous and largely non-tendon-specific models, and a direct mechanistic link between fluoroquinolones and TGF-β–mediated tendon injury has not been clearly established. Therefore, while disruption of TGF-β–MMP signaling may represent a potential contributing mechanism, this relationship remains speculative. Further experimental studies are needed to better understand the role of these pathways in fluoroquinolone-associated tendon injury.

TNF is a potent immune inflammatory modulator that interacts with various infectious and non-infectious stimuli. Its roles in the pathogenesis of mycobacteria have been clearly defined, and include upregulation of inflammatory cytokines and chemokines, inhibition of apoptosis of mononuclear cells and macrophages, and suppression of regulatory T-lymphocytes (T-regs). TNF plays a key role in formation and maintenance of structural integrity of granulomas that contain mycobacterial organisms (TB or NTM) that elude destruction by an early immune response. In the presence of TNF-α inhibitors, the granulomatous immune response is jeopardized and this predisposes the patient to a 2- to 7-fold increased risk of reactivation of latent TB and other granulomatous infections like NTM, including *M. mucogenicum* [3]. Usually, this results in an atypical course, with 50% extra-pulmonary and 10% disseminated TB infection as seen in HIV-positive patients [3].

Biologics including infliximab have a significant role in controlling intestinal as well as extra-intestinal manifestations of IBD. However, there is no literature related to the use of infliximab in the presence of NTM infection. In most published reports, etanercept, a TNF-α inhibitor, was discontinued after the diagnosis of NTM infection and was usually not restarted [4]. There is also no uniform timeline of when TNF-α inhibitors can be restarted [3]. We did not discontinue infliximab as our patient had significant enthesiopathy and acute exacerbation of IBD symptoms in the form of increased ileostomy drainage, fever, night sweats, and weight loss. Compared with previously reported cases of NTM infections in patients receiving TNF-α inhibitors, in which biologic therapy was discontinued, our case is unique in that infliximab was continued with close monitoring and concurrent targeted antimicrobial therapy, resulting in a favorable outcome [4]. Our report shows that biologics can be safely continued in the presence of NTM infection as long as the infection is treated with appropriate antibiotics for a prolonged period.

Management of skin infection with lymphangitis due to *M. mucogenicum* was also challenging, with limited antibiotic options. *M. mucogenicum* is a fastidious organism that needs 7-10 days to grow on AFB cultures. We managed our patient with trimethoprim-sulfamethoxazole and clarithromycin for a prolonged duration of 6 months. Our patient tolerated the regimen well and all his skin lesions healed successfully.

The usual timeline described in the literature suggests that patients generally develop NTM infections after 11-12 weeks of taking TNF-α inhibitors, but our patient developed an NTM infection after only 8 weeks [3]. The usual NTM infections associated with taking TNF-α inhibitors include bacteremia and pulmonary infections. Only 1 patient with rheumatoid arthritis has been described in the literature with *M. mucogenicum* skin infection, while receiving etanercept [4].

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Long-term therapy with trimethoprim-sulfamethoxazole and clarithromycin is usually safe, with few adverse effects like rash, nausea, vomiting, and diarrhea. However, there can be serious adverse effects like organ dysfunction and *C. difficile* colitis that may be life-threatening. As clarithromycin can interact with statins, statins are preferably withheld during clarithromycin therapy.

According to recent guidelines for NTM management, combination therapy guided by susceptibility testing is recommended to reduce relapse and resistance. Alternative regimens may include macrolide-based therapy combined with amikacin or imipenem depending on susceptibility patterns. Resistance patterns vary geographically, underscoring the importance of culture-directed therapy [15]. This report is limited by its nature as a single case study, which restricts generalizability. Further studies are needed to better understand this rare clinical condition.

Conclusions

This case underscores the importance of early diagnosis of opportunistic infections, including mycobacterial infections, in immunosuppressed patients receiving infliximab. Careful balancing of targeted antimicrobial therapy with continued immunosuppression may be feasible in selected patients with localized NTM infection. Further studies are needed to determine the optimal timing of immunosuppression and the appropriate duration of antibiotic therapy for definitive cure. The combined antibiotic regimen, with clarithromycin and trimethoprim-sulfamethoxazole for treating disseminated mycobacterial dermal abscesses with lymphangitis, was observed to be safe and effective. Additionally, our case emphasizes the need for cautious use of fluoroquinolones in patients with IBD and enthesiopathy due to potential increased risk of tendon injury. Future research should explore and investigate the mechanisms underlying fluoroquinolone-associated tendon injury. Further studies and accumulation of similar cases are needed to better inform clinical decision-making in such complex scenarios.

Department and Institution Where Work Was Done

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Patient Permission/Consent Declarations

Written and informed consent was obtained from the patient.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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