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CASE REPORT

Nontuberculous mycobacterial myositis in dermatomyositis with long-term use of immunosuppressant: a case report

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Abstract

Inflammatory myopathies, such as polymyositis and dermatomyositis, are systemic inflammatory disorders that affect skeletal muscles and internal organs. The treatment of inflammatory myopathies usually involves long-term use of high doses of steroids and/or immunosuppressants, making patients susceptible to opportunistic infections. Unfortunately, infections are a leading cause of morbidity and mortality in patients with inflammatory myopathies. Musculoskeletal nontuberculous mycobacterial infections are rare. Nontuberculous mycobacterial infections are easily overlooked owing to their rarity, leading to delayed diagnosis and treatment, indolent clinical course, and difficulty isolating the pathogen. Nontuberculous mycobacterial infections are a growing health concern because of their increasing incidence and the need for prolonged treatment. In patients with connective tissue diseases, immunosuppressant use may lead to an increased risk of nontuberculous mycobacterial infection with a poor prognosis, which highlights the need for early diagnosis and treatment. Herein, we report the case of a 59-year-old man diagnosed with dermatomyositis, who had prolonged use of immunosuppressants and developed a disseminated soft tissue infection in both thighs caused by *Mycobacterium abscessus*. Multimodal images were obtained using magnetic resonance imaging, ultrasonography, and computed tomography. A strong suspicion of possible combined opportunistic infections and appropriate staining is essential in diagnosing nontuberculous mycobacterial myositis.

Keywords Nontuberculous mycobacteria infection · Myositis · Inflammatory myopathy · Opportunistic infection

Introduction

Inflammatory myopathies are systemic inflammatory disorders that affect the skeletal muscles and internal organs [1]. Dermatomyositis is the most common idiopathic inflammatory myopathy [2]. The diagnosis is based on clinical features, including typical skin manifestations supported by serology and confirmed by histopathology [3]. MRI findings can range from extremely subtle superficial tissue involvement (skin or subcutaneous fat) to extensive muscle and myofascial involvement. Inflammation usually involves the proximal muscles symmetrically and shows high signal intensity within the muscles and their fascia on fat-suppressed fluid-sensitive sequences and gadolinium-enhanced T1-weighted images. Treatment included steroid therapy or immunosuppressants. Infectious complications are reported in up to 26% of patients [1, 4, 5].

Although rare, musculoskeletal nontuberculous mycobacterial (NTM) infections are currently a growing health concern owing to their increasing incidence and the need for prolonged treatment [6]. However, NTM infections are easily overlooked, leading to delayed diagnosis and treatment owing to their rarity, indolent clinical course, and difficulty in isolating the pathogen [7, 8]. In patients with connective tissue diseases, immunosuppressant use may increase the risk of NTM infections with a poor prognosis, highlighting the need for early diagnosis and treatment [9]. The most common site of NTM infection is the hand/wrist, followed by the knees, spine, and feet [10, 11]. However, reports on the MRI findings of NTM myositis are scarce, and concomitant inflammatory myositis can be confounded by the underlying disease. Therefore, a strong suspicion of possible

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combined opportunistic infections is essential for diagnosing NTM myositis in imaging studies, especially in patients with inflammatory myopathies.

Here, we report the case of a 59-year-old man diagnosed with dermatomyositis, who had prolonged use of immunosuppressants and developed a disseminated soft tissue infection in both thighs caused by *Mycobacterium abscessus*. Multimodal images were obtained using magnetic resonance imaging, ultrasonography, and computed tomography.

Case report

A 59-year-old man with dermatomyositis and vasculitis is referred to the radiology department for an ultrasound of multiple palpable masses. A detailed timetable for the patient's clinical course and treatment is listed in Table 1. Two years ago, he was diagnosed with polychondritis and interstitial lung disease due to cytoplasmic antineutrophil cytoplasmic antibodies (c-ANCA) positive vasculitis in an outside hospital (Table 1). After diagnosis, he was treated with steroids and immunosuppressive drugs such as mycophenolate mofetil (MMF) for recurrent multiple skin ulcers, arthralgia, and interstitial lung disease. One year later, c-ANCA turned negative, but anti-Sm (antibody against the Smith antigen) and anti-Ro antibodies (anti-Sjögren's-syndrome-related antigen A autoantibodies) were positive. Digital angiography at the time showed steno-occlusion of the proper palmar digital arteries of both hands, consistent with vasculitis.

One year and 5 months after the initial diagnosis, he developed symmetric proximal muscle weakness in his upper and lower extremity and neck with Gottron-like skin rash and dysphagia while continuing immunosuppressant therapy with steroids and MMF. Muscle biopsy showed endomysial, perimysial, and epimysial inflammatory cell infiltrates, consistent with inflammatory myopathy. According to the European Alliance of Associations for Rheumatology (EULAR)/American College of Rheumatology (ACR) classification criteria for adult and juvenile idiopathic inflammatory myopathies, he was classified as definite dermatomyositis. He was treated with MMF and steroids for dermatomyositis, but there was no improvement. Therefore, he was given a combination of immunosuppressive drugs such as methotrexate, azathioprine, tacrolimus, and cyclosporine but also did not improve.

Three months after the muscle biopsy, he performed MRI of both thighs at the outside hospital. MRI showed multifocal patchy high signal intensity in the fat-suppressed T2 weighted images and enhancement in the muscles and along the myofascial plane of all three compartments of both thighs with mild fatty atrophy change of muscles (Fig. 1). All these findings were thought that active inflammation

Table 1 A chronological representation of a patient's diagnosis, events, and treatment

Time	First visit	1 year	1 year and 5 months	1 year and 8 months	1 year and 10 months	2 years
Diagnosis/event	c-ANCA (+) vasculitis, polychondritis, interstitial lung disease	c- ANCA (-), anti-SM/Ro (+), DSA: steno-occlusion of the palmar digital arteries in both hands	Diagnosis of dermatomyositis	MRI of both thighs	Visit our rheumatology clinic	USG for multiple palpable masses, confirmed as NTM myositis
Treatment	Steroid, mycophenolate mofetil (MMF)		Methotrexate, azathioprine, tacrolimus, cyclosporine		Methylprednisolone,IVIG	Treatment of NTM

c-ANCA cytoplasmic antineutrophil cytoplasmic antibodies, *anti-Sm* antibody against the Smith antigen, *anti-Ro antibodies* anti-Sjögren's-syndrome-related antigen A autoantibodies, *DSA* digital angiography, *IVIG* intravenous immunoglobulin, *USG* ultrasonography, *NTM* non-tuberculous mycobacteria

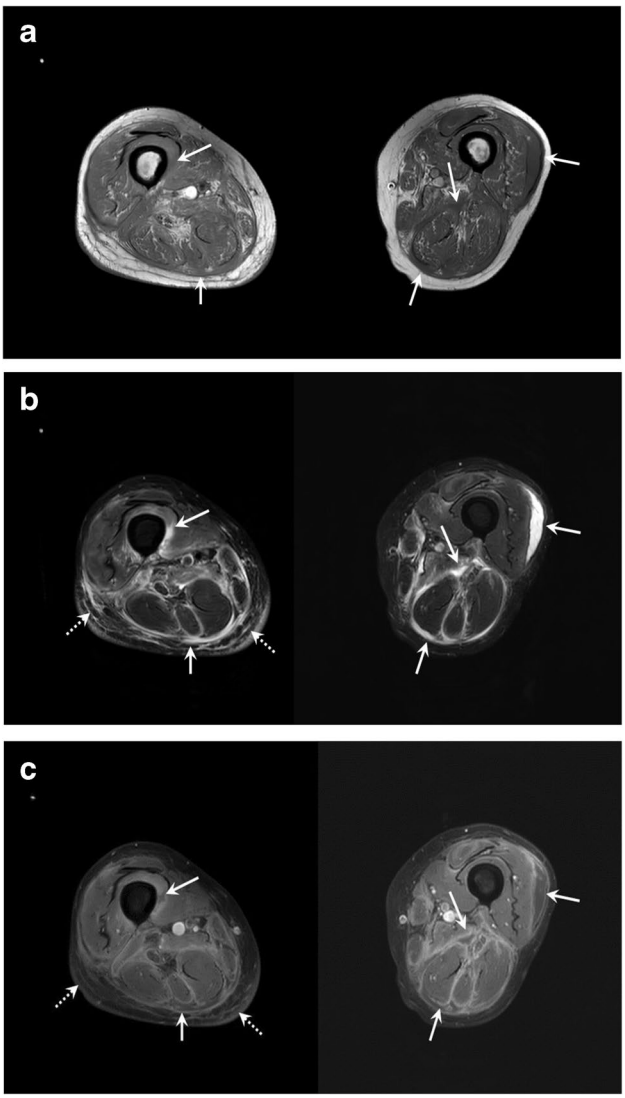

Fig. 1 Axial T1 (T1WI, **a**), fat-suppressed T2 (T2WI, **b**), and contrast-enhanced fat-suppressed T1 (CET1, **c**) weighted fast spin echo sequence of both thighs of 59-year-old man with dermatomyositis. MRI revealed multifocal patchy abnormal signal intensity with an iso to low signal on T1WI (**a**) and high signal intensity on T2WI (**b**) and CET1 (**c**) along the myofascial plane throughout the whole compartment muscles of both thighs symmetrically. Note the high signal intensity (arrows) in the intermuscular spaces on T2WI (**b**). CET1 (**c**) shows multiple peripheral rim-enhancing lesions (arrows) at the vastus intermedius muscle of the right thigh, intermuscular spaces of both thighs, subfascial region beneath the tensor fascia lata of the left thigh, and beneath deep peripheral fascia of the posterior compartment on both thighs. Lattice-like high signal intensities (dashed arrows) were observed in the posterior subcutaneous fat layer of the right thigh on T2WI (**b**) and CET1 (**c**)

and chronic changes in dermatomyositis at that time. In the right thigh, a multifocal lattice-like high signal intensity in T2 weighted image was observed in the subcutaneous fat layer, suggesting the involvement of dermatomyositis (Fig. 1). However, there were multiple areas of high signal intensity in T2 weighted images and low signal intensity in T1 weighted image with peripheral enhancement beneath the tensor fascia lata of the left thigh, vastus intermedius of the right thigh, and intermuscular spaces of both thighs, representing abscess or thick fluid collection, which was unusual for dermatomyositis but were initially overlooked as fascial involution of dermatomyositis (Fig. 1). Among them, lesions in the vastus intermedius muscle and the intermuscular spaces between the medial and posterior compartments of the right thigh showed peripheral high signal intensity on T1 weighted images and heterogeneous high and low signal intensity on T2 weighted image with enhancement but was also overlooked (Fig. 2).

He was transferred to our rheumatologic clinic 5 months after diagnosis of dermatomyositis due to poor treatment response. Laboratory examinations at the referral time revealed mild leukocytosis (WBC count $10.9 \times 103/mm^3$), mild elevation of erythrocyte sedimentation rate (ESR, 57 mm/h), serum aldolase (9.2 U/L), lactate dehydrogenase (LDH, 372 U/L), and serum c-reactive protein (CRP, 4.35 mg/dl). In contrast to the initial results, serum autoantibody tests, including anti-Jo-1, anti-Sm, anti-PR3, and anti-MPO tests, conducted during hospitalization showed negative results. He did not have a fever. The serum creatine kinase (CK) and myoglobin levels were within the normal range. Immunosuppressive therapy with methylprednisolone and intermittent intravenous immunoglobulin (IVIG) was also administered at our rheumatology clinic. His symptoms improved temporarily after IVIG but soon returned.

Two months after being treated at our hospital, multiple palpable masses were developed on his both thighs. Ultrasonography revealed multifocal heterogeneous echogenicity in subcutaneous fat, indicating panniculitis in the palpable areas of the right thigh (Fig. 3). In another palpable area of the left thigh, ultrasonography revealed a thick hypoechogenic area beneath the tensor fascia lata where the peripheral enhancing area was observed in the prior outside MRI (Fig. 3). Ultrasonography-guided gun biopsies were performed for the thick hypoechoic area beneath the tensor fascia lata. The pathological report revealed necrosis, granulation tissue, and acid-fast bacilli, accompanied by chronic granulomatous inflammation (Fig. 4). Two weeks later, follow-up ultrasonography was requested for the palpable masses in the right thigh that developed fluctuations. In the follow-up ultrasonography, the previously noted heterogeneous echogenic lesion was interchanged with an abscess containing floating debris, and aspiration was performed (Fig. 3). Real-time polymerase chain reaction (PCR) was performed on the aspirated abscess, and NTM infection was identified, and *Mycobacterium abscessus* was cultured from both thighs. Antibiotic medication such as oral clarithromycin, imipenem, and amikacin was initiated, and his general weakness improved after a month. A follow-up MRI of both

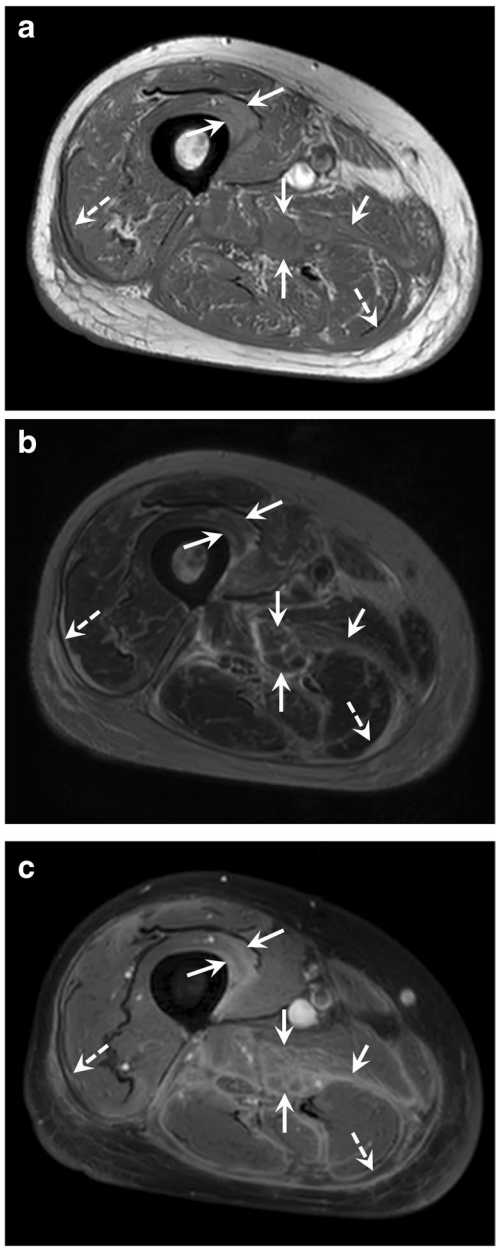

Fig. 2 Another axial images for right thigh, proximal level to Fig. 1 with magnification. There are peripheral high signal intensity (arrows) on T1 weighted image (**a**) and heterogeneous high and low signal intensity (arrows) on T2 weighted image (**b**) with enhancement in the vastus intermedius muscle and the intermuscular spaces between medial and posterior compartment of the right thigh, representing abscess containing paramagnetic materials periphery. Liquefied abscess or fluid collection (dashed arrows) also seen as areas of low signal intensity on T1 weighted image (**a**), high signal intensity on T2 weighted image (**b**) with thin smooth peripheral enhancement

thighs was recommended to determine the extent and distribution of the NTM infection, but the patient declined due to financial reasons. Therefore, a contrast-enhanced CT for both lower extremities was performed instead. Although the patient's symptoms improved, CT (Fig. 5) revealed multiple peripheral enhancing abscesses in the subcutaneous fat layer of the right thigh and beneath the tensor fascia lata. Surgical irrigation and debridement were performed, and the patient's symptoms gradually improved.

Discussion

In our case, the patient was diagnosed as vasculitis and dermatomyositis and finally complicated with NTM myositis. NTM myositis in our case showed bilateral symmetric high signal intensity in the muscle and along the myofascial plane on T2 weighted images, with imaging findings similar to those of dermatomyositis resulting in delayed diagnosis. The fact that the patient had previously been diagnosed with dermatomyositis on biopsy also led to a bias in diagnosis.

Dermatomyositis and polymyositis are proximal myopathies characterized by bilateral symmetrical inflammation of the proximal muscles of the extremities. Dermatomyositis shows a patchy involvement in contrast to the diffuse patterns of polymyositis [12]. MRI is considered the most effective diagnostic modality for accurately depicting the extent and intensity of muscular abnormalities. In the evolution of dermatomyositis, fasciitis may represent an early lesion because the fascial microvasculature is the primary target tissue of inflammatory cell infiltration, and the inflammation progresses from the fascia to the muscle [12]. Therefore, the affected muscle shows symmetric high signal intensity along the myofascial plane on the fluid-sensitive sequence and fat-saturated contrast-enhanced T1-weighted image [12]. Subcutaneous tissue involvement is also a characteristic finding in dermatomyositis. Subcutaneous fat shows a reticular pattern of involvement on MRI that is attributed to edema caused by perivascular inflammation. Treatment often involves long-term administration of high doses of glucocorticoids or immunosuppressants. This treatment approach renders patients particularly vulnerable to opportunistic infections. Previous reports indicate that opportunistic infections occur in 10.9–21.3% of patients with polymyositis and dermatomyositis [4, 5]. A meta-analysis of mycobacterial infections in patients with inflammatory myopathies found a higher proportion of case reports of tubercular muscle involvement, which is an otherwise rare site [13]. They hypothesize that this higher proportion of TB myositis case reports in patients with inflammatory myopathy could be because of the seeding of TB bacilli in inflamed/necrotic muscle tissue in patients with inflammatory myopathy [13]. The muscle may act as a nidus for bacillary growth and later development of tubercular muscle disease in susceptible hosts [13]. Although NTM infection is much rarer than TB, the same pathophysiology could explain why the NTM infection in our case occurred in the

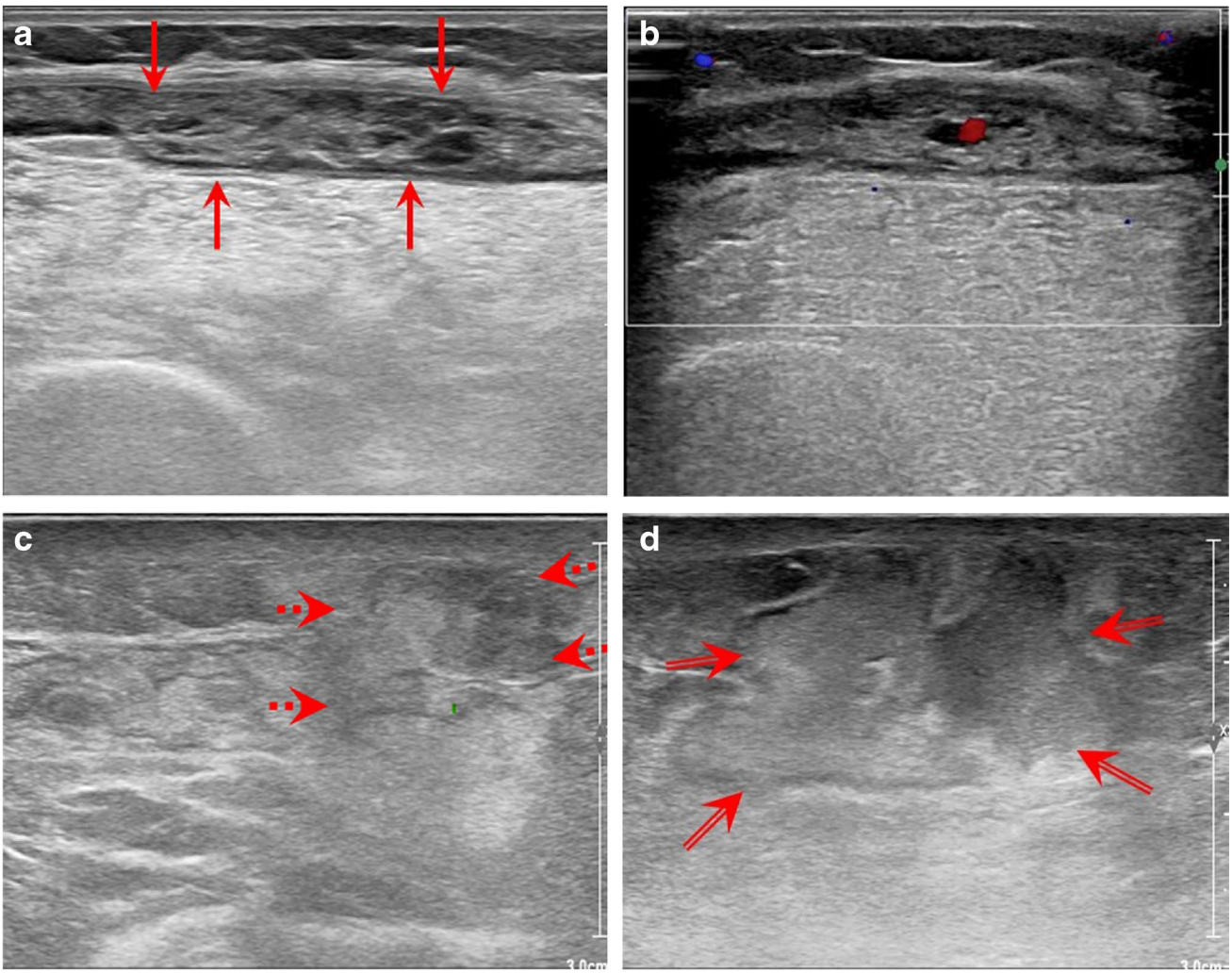

Fig. 3 Ultrasonography images of the left thigh (**a**, **b**) and the right thigh (**c**, **d**). Heterogeneous hypoechogenicity (arrows in **a**) is observed beneath the tensor fascia lata of the left thigh, with increased vascularity in the Doppler image (**b**). In the right thigh, multifocal localized swelling and heterogeneous echogenic infiltration (dashed arrows in **c**) were observed in the subcutaneous layer of the right thigh, which was interpreted as panniculitis (**c**). Two weeks later, these lesions became hypoechoic abscesses with innumerable floating debris and increased in size (double arrows in **d**)

bilateral symmetric proximal muscles along the myofascial plane and overlying subcutaneous fat layer, which are typical impacted sites of dermatomyositis.

Conversely, NTM infections can be misdiagnosed as inflammatory myopathy, both clinically and radiologically. Shipman et al. reported a disseminated *Mycobacterium avium* infection masquerading as longstanding polymyositis [14]. In their case, the MRI was interpreted as having an asymmetric involvement of inflammatory myopathy, and the biopsy results diagnosed erythema nodosum or polymyositis-associated panniculitis. Subsequently, the biopsy results were re-evaluated and confirmed as mycobacterial infections using acid-fast staining. Because NTM infections have a relatively indolent clinical course and nonspecific clinical symptoms, especially in their early phase, distinguishing between an NTM infection and inflammatory myopathy is difficult.

In our case, the pathological report revealed necrosis, granulation tissue, and acid-fast bacilli, accompanied by chronic granulomatous inflammation, and *Mycobacterium abscessus* was cultured from both thighs which is no doubt for the NTM infection. However, questions may arise regarding whether NTM myositis is an opportunistic infection associated with dermatomyositis or if the NTM infection alone was present from the outset and erroneously diagnosed as dermatomyositis. Additionally, myositis-specific antibodies, including anti-Jo 1, which are known to increase in dermatomyositis, were all negative in our case. Serum CK levels, typically utilized for diagnosing and monitoring patients with inflammatory myopathy, were not elevated. We

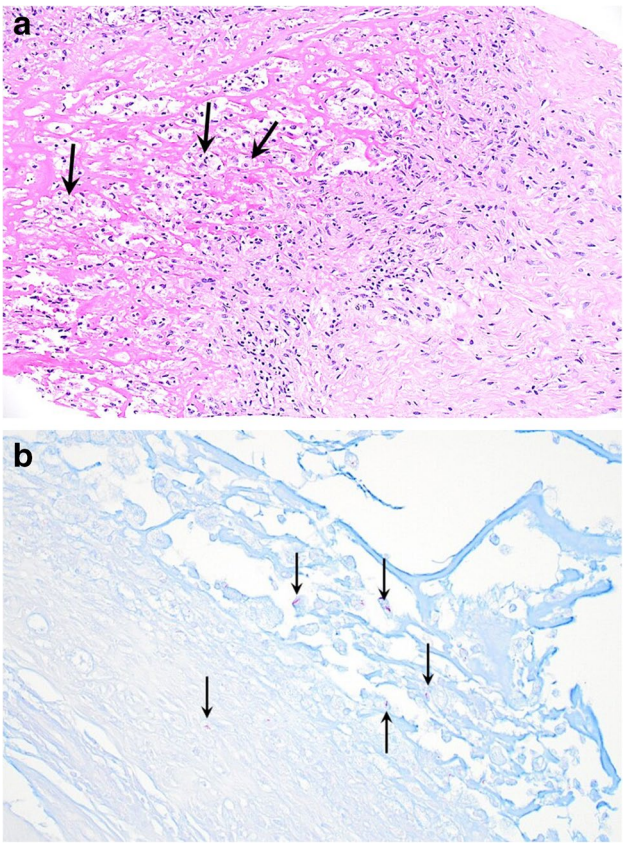

Fig. 4 Histopathology showed poorly formed granuloma with necrosis and foamy histiocytes (arrows in **a**) infiltration (**a**, H&E stain, ×200). At Ziehl–Neelsen stain (**b**, ×400), many acid-fast bacilli (arrows in **b**) are noted in necrotic tissue and cytoplasm of foamy histiocytes

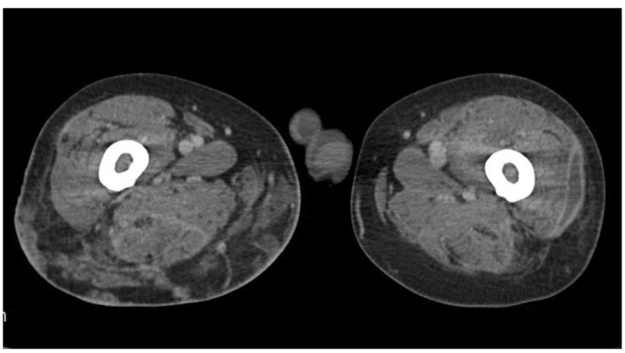

Fig. 5 Contrast-enhanced CT of both thighs reveals peripheral enhanced subfascial fluid collection beneath tensor fascia lata of the left thigh and multiple peripheral enhanced subcutaneous abscess at the right thigh

did observe mild leukocytosis and slightly elevated serum ESR, LDH, and CRP levels. However, these findings can be seen in cases of inflammatory myositis both with and without infection. Nevertheless, in our case, it is plausible to infer that NTM appeared as an opportunistic infection associated with dermatomyositis. Firstly, the Gottron-like skin rash observed in our patient is pathognomonic for dermatomyositis [15]. Secondly, symmetric progressive weakness of the upper and lower extremities, as well as the neck, is a hallmark clinical characteristic of dermatomyositis [15]. Due to these two reasons, our patient was classified as having definite dermatomyositis based on the EULAR/ACR classification criteria for adult and juvenile idiopathic inflammatory myopathies, even excluding the muscle biopsy results [16]. Thirdly, although various myositis-associated or myositis-specific autoantibodies, such as anti-Jo-1, have been identified in dermatomyositis patients, a significant proportion of these patients test negative for such autoantibodies [17]. According to a previous study, 45.9% of inflammatory myopathy patients tested negative for both myositis-specific and myositis-associated autoantibodies [17]. The documented frequency of anti-Jo-1 positivity varies from 4 to 22% across literature [18]. Myositis-specific autoantibodies are not mandatory for diagnosing dermatomyositis but contribute to its phenotypical features and subclassification [19]. Lastly, a subset of dermatomyositis patients, especially those under long-term treatment like our patient, present with normal serum CK levels. In such individuals, the serum aldolase level might serve as an alternative biomarker to serum CK levels [20]. It is noted that serum aldolase typically rises in conditions like dermatomyositis, polymyositis, and muscle dystrophy but remains usual in infectious myositis [20]. In our case, the elevated serum aldolase is more indicative of inflammatory myositis rather than infection.

Little is known about the imaging findings of NTM myositis owing to its rarity. Based on previous case reports, the imaging findings of NTM myositis on CT and MRI have shown skin thickening, diffuse muscle edema, and intramuscular fluid collection or abscess formation in the affected muscle, which appear as low and high signal intensities on T1WI and T2WI, respectively, with peripheral enhancement [21–24]. Consistent with previous studies, some areas with peripheral enhancing lesions, representing abscesses, were also present in our case [21, 24]. In addition, some of the abscesses showed high signal intensity on T1-weighted images, heterogeneous signal intensity on T2 weighted images, and contrast enhancement in the periphery. These findings are known to be due to the paramagnetic materials including oxygen free radicals and macrophage sequestration iron from microvascularization in the wall of the abscess [25]. These findings are commonly observed in TB abscesses, but some studies have reported them in pyogenic bacterial abscesses as well [26].

The condition that requires differentiation in our case is other opportunistic infections that can accompany dermatomyositis. A systematic review revealed that TB is a more common pathogen for opportunistic infection than NTM in

patients with inflammatory myopathy [13]. Along with the indolent clinical course of TB myositis, its imaging findings are also analogous to those of NTM myositis [25]. As such, a tissue biopsy culture is often necessary to differentiate between them. Imaging studies on TB myositis have shown that it typically forms intramuscular abscesses in multiple muscles and are accompanied by cellulitis, similar to our case [26]. Bacterial pyomyositis and fasciitis, including necrotizing fasciitis, can involve multiple muscles and fasciae, but bilateral symmetric involvement is atypical [26, 27]. Compared to indolent infections like TB and NTM, the abscess wall is thick and irregular [25–27]. The swelling of the overlying subcutaneous fat layer is more pronounced than in TB, with severe clinical symptoms and a much faster progression [25–27]. Viral myositis can manifest as streaky or patchy muscle infiltration with abnormally high T2-weighted signals and either heterogeneous or diffuse muscle enhancement [26, 27]. The extent and distribution of viral myositis can vary, but it might be bilateral and/or symmetric, making it challenging to distinguish from the active inflammation of dermatomyositis based solely on imaging findings [26, 27]. While viral myositis can progress to rhabdomyolysis, abscess formation, akin to TB or bacterial pyomyositis, is rare [26, 27]. Fungal infections have been reported in immunocompromised individuals, often as opportunistic infections of the lung or esophagus. Two case reports described multiple microabscesses with a background of diffuse muscular MRI signal abnormalities consistent with inflammation [28, 29]. Except for opportunistic infections, exacerbation of dermatomyositis should also be considered as a differential diagnosis. Several studies have reported that patients with dermatomyositis might experience extensive fascial and subcutaneous edema [30, 31]. In this case, it is very difficult to differentiate between simple edema and abscess in the fascial space on T2-weighted images alone. However, as shown in our case, the presence of peripheral high signal intensity on T1-weighted image or post-contrast rim enhancement suggesting infection is an important point for differentiation [25–27].

The prognoses of NTM infections, particularly among immunocompromised individuals, are relatively poor [9]. In soft tissue and musculoskeletal infections, rapid-growing NTM, such as *Mycobacterium abscessus* complex, *Mycobacteroides chelonae*, and *Mycobacterium fortuitum*, are frequently identified and associated with disseminated infections among patients who are immunocompromised [8, 11, 32]. Combination antibiotic treatment is necessary for these rapidly growing NTM infections, highlighting the importance of early diagnosis for NTM infections [32, 33]. However, the diagnoses of NTM infections are usually delayed, with a mean duration between symptom onset and diagnosis ranging from approximately 10 to 20 months [7, 8, 11]. The primary reason for diagnostic delays is considered to be the non-specific and indolent clinical symptoms of NTM. Therefore, in such cases, image studies including US, CT, and MRI can help raise awareness of the possibility of NTM infection. On imaging, we believe that intramuscular fluid collection and abscess formation in the affected muscle can be a diagnostic clue for differentiating NTM infections from inflammatory myopathy or for detecting superimposed NTM infections. Careful image interpretation with a strong suspicion of possible combined opportunistic infections and recommending appropriate staining is essential in diagnosing NTM myositis.

Data availability The datasets used and/or analysed during the current study available from the corresponding author on reasonable request.

Declarations

Ethics approval This case report was performed in accordance with the ethical standards of our institutional research committee and with the 1964 Declaration of Helsinki and its later amendments or comparable ethical standards. Institutional review board approval was obtained for this study, and informed consent was waived due to its retrospective nature.

Conflict of interest The authors declare no competing interests.

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