

A Case Report of Refractory *Mycobacterium wolinskyi* Knee Infection in a Metabolic Syndrome Patient: mNGS Diagnosis and Pharmacist-Guided Therapy

Min Shao^{1,*}, Lijia Ni^{2,3,*}, Liang Jiang⁴, Jingyi Hou⁴, Sicheng Xu⁴, Yin Lin⁵, Xiaoying Xie⁶

¹Department of Pharmacy, Liwan Central Hospital of Guangzhou, Guangzhou, 510000, People's Republic of China; ²Department of Clinical Laboratory, Sun Yat-Sen Memorial Hospital, Sun Yat-Sen University, Guangzhou, 510120, People's Republic of China; ³Institution of Antibiotic, Sun Yat-Sen Memorial Hospital, Sun Yat-Sen University, Guangzhou, 510120, People's Republic of China; ⁴Department of Orthopedic Surgery, Sun Yat-Sen Memorial Hospital, Sun Yat-Sen University, Guangzhou, 510120, People's Republic of China; ⁵Department of Pharmacy, Sun Yat-Sen Memorial Hospital, Sun Yat-Sen University, Guangzhou, 510120, People's Republic of China; ⁶Department of Clinical Laboratory, The Seventh Affiliated Hospital of Sun Yat-Sen University, Shenzhen, 518106, People's Republic of China

*These authors contributed equally to this work

Correspondence: Xiaoying Xie; Yin Lin, Email xiexy5@mail.sysu.edu.cn; linylin@mail.sysu.edu.cn

Abstract: *Mycobacterium wolinskyi* (*M. wolinskyi*), which is a rare rapidly growing mycobacterium (RGM), and the infections it causes are predominantly linked to surgery or invasive procedures. We detailed a case of refractory surgical site infection (SSI) caused by *M. wolinskyi*. The causative pathogen was identified by metagenomic next-generation sequencing (mNGS) analysis, 16S rRNA and *rpoB* gene sequencing. What renders this case particularly remarkable is the complexity introduced by a series of antibiotic-induced adverse effects, which seem to be deeply intertwined with the patient's underlying metabolic syndrome. With the meticulous pharmaceutical guidance provided by the clinical pharmacist, the patient experienced a substantial improvement in his knee joint infection.

Keywords: *Mycobacterium wolinskyi*, surgical site infection, metabolic syndrome, mNGS

Introduction

Mycobacterium wolinskyi (*M. wolinskyi*) is a rapidly growing non-tuberculous mycobacterium (NTM) within the *Mycobacterium smegmatis* group,¹ which was first isolated in 1999.¹ As an emerging opportunistic pathogen, it primarily affects immunocompromised hosts²⁻⁴ or individuals undergoing invasive procedures.⁵⁻⁷ In contrast to more common mycobacterial species such as *Mycobacterium abscesses* and *Mycobacterium avium* Complex (MAC), *M. wolinskyi* is categorized as a rare pathogen, with fewer than 30 cases documented globally to date. Most of these cases have been reported in developed countries including the United States, Japan, France, and Korea, while sporadic cases have also been identified in developing countries such as India and Brazil. However, no such cases have been recorded on the Chinese mainland. The scarcity of reported clinical infection cases may be attributed to its relatively recent initial isolation and the requirement for sequencing-based identification, as conventional identification systems (Vitek 2 automated platform and MALDI-TOF MS, bioMérieux, France) usually failed to identify it.⁸⁻¹¹

Clinically, *M. wolinskyi* infections are strongly associated with invasive medical procedures^{5,6,10} or foreign body implants,^{8,12,13} manifesting as recalcitrant skin and soft tissue, bone and joint, or even systemic infections such as bloodstream infections. The treatment of *M. wolinskyi* infections typically involves a combination of surgical intervention and prolonged antibiotic therapy. The antibiotic therapy management is complicated by intrinsic multidrug resistance, requiring tailored regimens guided by drug susceptibility testing. Although amikacin, fluoroquinolones, and tetracyclines

are commonly used empirically,¹⁴ growing evidence indicates significant strain-specific variations in resistance patterns, highlighting the necessity for targeted therapeutic strategies.

Notably, comorbidities such as metabolic syndrome—characterized by chronic inflammation and altered pharmacokinetics¹⁵—may exacerbate antimicrobial toxicity, a dimension poorly explored in existing *M. wolinskyi* literature. Here, we present the first case of postoperative *M. wolinskyi* knee infection complicated by metabolic syndrome-driven antibiotic hypersensitivity, where metagenomic next-generation sequencing (mNGS) enabled rapid pathogen identification, and clinical pharmacist-led therapeutic drug monitoring (TDM) mitigated adverse drug reactions (ADRs). The rapid mNGS diagnosis of rare pathogens and pharmacist-guided antibiotic regimens set our report apart from previous studies.

Case Presentation

A 41-year-old female with metabolic syndrome (BMI 25.1 kg/m², HbA1c 6.9%, hypertension, hyperlipidemia) underwent left knee anterior cruciate ligament (ACL) reconstruction and meniscus repair on April 26, 2023, following trauma. On postoperative day 20 (May 16), purulent discharge (gray to yellowish) emerged from the incision, accompanied by erythema, swelling, and pain. To address the infection, the patient underwent the first revision surgery and debridement and was administered clindamycin 300 mg q8h iv.gtt for a duration of 12 days. However, the treatment was unsuccessful, prompting escalation to cefoperazone/sulbactam 3 g q8h and levofloxacin 500 mg qd iv.gtt for one week, which also proved ineffective. Seeking for further evaluation and management, the patient was admitted to our hospital on June 13th, 2023.

Diagnostic Journey

Inflammatory markers revealed the presence of mild inflammation (N% 72.9%, CRP 22.9 mg/L, ESR 41 mm/h). Magnetic resonance imaging (MRI) of the left knee joint indicated a minor accumulation of suprapatellar bursa effusion and periarticular soft tissue edema (Figure 1). Both laboratory and imaging findings indicated an inflammatory process. Clinically, the patient presented with persistent knee joint erythema, swelling, and pain accompanied by incision site drainage; however, the causative pathogen remained unidentified. Following microbiological recommendations, synovial fluid specimens were inoculated into aerobic and anaerobic blood culture bottles. Concurrently, empirical antibiotic therapy with linezolid (600 mg q12h iv.gtt) was initiated immediately after specimen collection. On June 21th, 2023, the patient underwent a second revision surgery for anterior cruciate ligament reconstruction with debridement, during which a tissue specimen was obtained for mNGS analysis — an intervention aimed at enhancing early definitive pathogen identification.

Microbiology

The aerobic blood culture bottle demonstrated positivity following a 4-day incubation period. Subsequent subculture on Columbia blood agar plates at 35°C for equivalent duration yielded small, white opaque, rough-textured colonies (Figure 2A). The colonies maintained their achromic characteristics despite prolonged incubation (Figure 2B). Microscopic examination revealed Gram-positive bacilli (Figure 2C) with notable acid-fast staining properties (Figure 2D). Conventional identification systems (Vitek 2 automated platform and MALDI-TOF MS, bioMérieux, France) failed to achieve species-level classification. Notably, mNGS analysis of intraoperative tissue samples detected *M. wolinskyi* genomic sequences: at a sequencing depth of 1×, 11 reads per million (RPM) were obtained, with a relative abundance of 2%. To verify species concordance between the cultured isolate and mNGS findings, targeted molecular identification was performed through amplification and bidirectional sequencing of the 16S rRNA and *rpoB* genes (Conlight Medical Laboratory). BLAST alignment revealed 100% 16S rRNA sequence identity (GenBank accession: NR_042922.1) and 99.08% *rpoB* homology (GenBank accession: AY262743.2) with *M. wolinskyi* strain ATCC 700010. Antimicrobial susceptibility profiling, conducted via broth microdilution and E-test methodologies, was interpreted per CLSI M24-A2 guidelines (Table 1), incorporating breakpoints established for rapidly growing mycobacterium (RGM).

Multidisciplinary Treatment Challenges

On 29 June 2023, based on published therapeutic protocols for *M. wolinskyi* infections and the antimicrobial susceptibility profile (Table 1), the antimicrobial stewardship team implemented a triple therapy regimen: amikacin 600 mg qd,

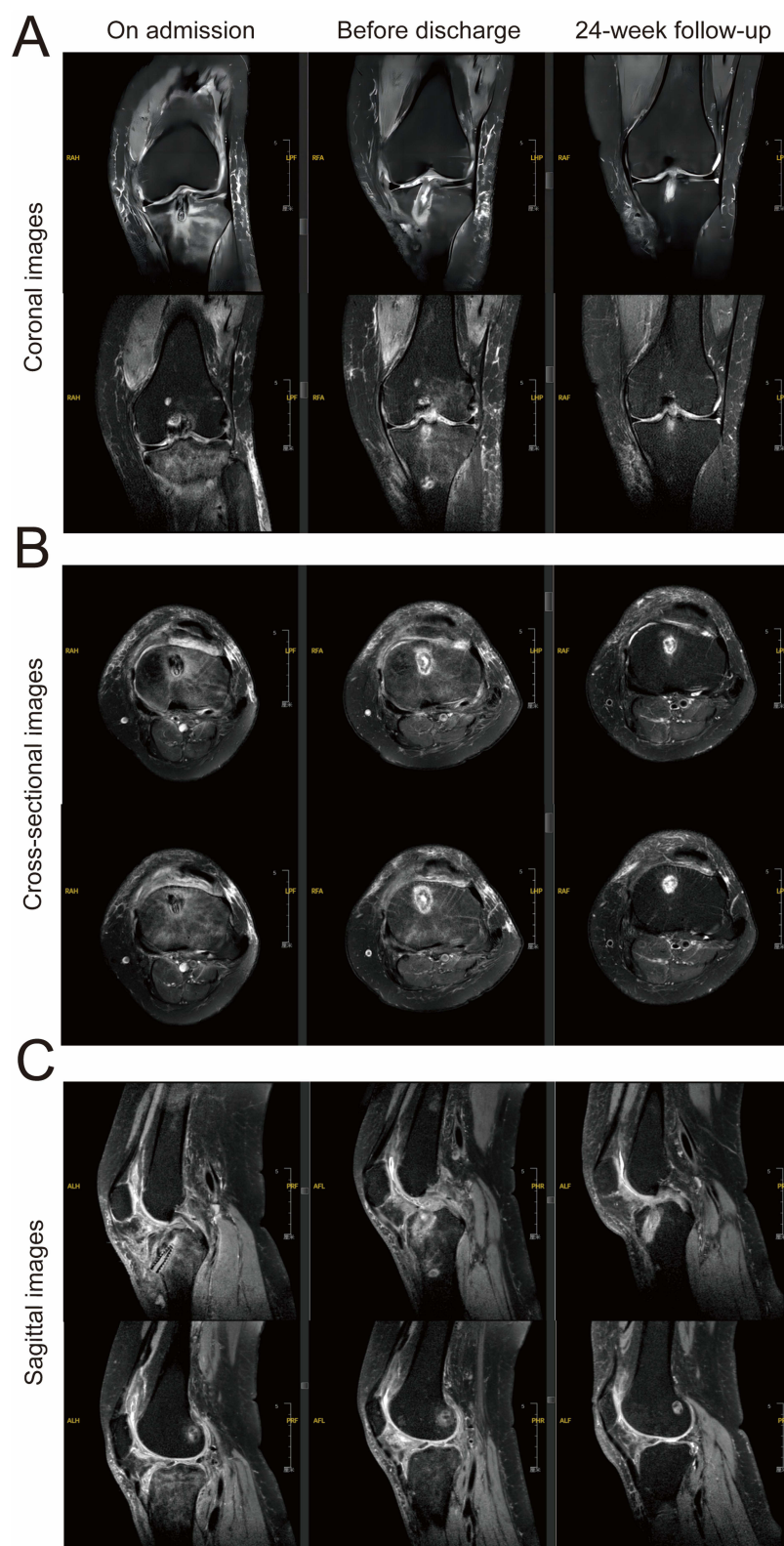


Figure 1 Magnetic resonance imaging of the left knee joint at different stages. **(A)** The coronal images of the left knee joint. **(B)** The cross-sectional images of the left knee joint. **(C)** The sagittal images of the left knee joint.

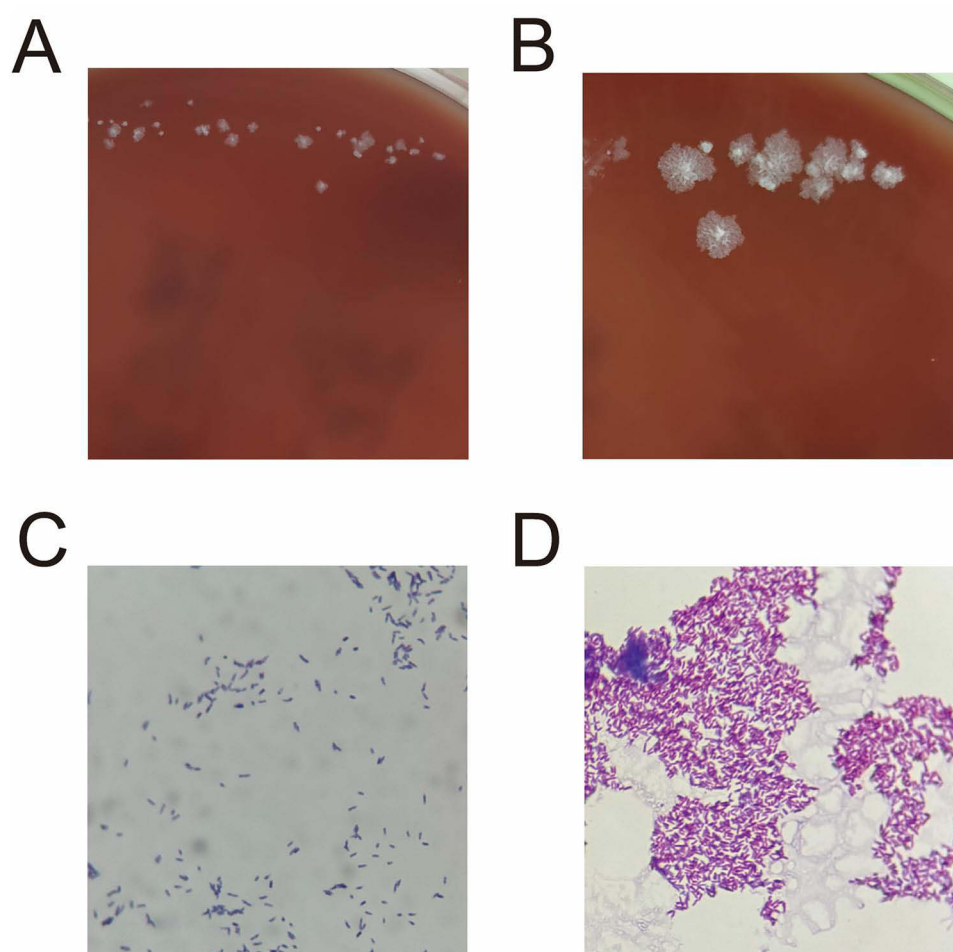


Figure 2 The colony morphology and microscopic morphology of *Mycobacterium wolinskyi*. (A) The colony morphology after four days of cultivation on the Columbia blood plate. (B) The colony morphology after eight days of cultivation on the Columbia blood plate. (C) Gram staining of colonies revealing Gram-positive bacilli ($\times 1000$). (D) Acid-fast staining of colonies revealing red bacilli ($\times 1000$).

imipenem 1 g q12h, and linezolid 600 mg iv.gtt q12h. Within 48 hours of initiation, the patient developed ototoxicity (tinnitus) and gastrointestinal intolerance (vomiting), accompanied by TDM revealing supratherapeutic linezolid trough concentrations (9.04–10.93 $\mu\text{g/mL}$; target $<7 \mu\text{g/mL}$). Concurrent hematological monitoring showed progressive anemia (Hb 117 g/L to 96 g/L) and thrombocytopenia ($312 \times 10^9/\text{L}$ to $170 \times 10^9/\text{L}$), prompting protocol-driven dose adjustments: amikacin reduction to 400 mg daily and linezolid conversion to oral administration.

Persistent elevation of linezolid serum trough concentration (15.8 $\mu\text{g/mL}$ on 2 July 2023) necessitated further dose reduction to 300 mg PO q12h, achieving target therapeutic levels (5.63 $\mu\text{g/mL}$) by 10 July 2023. Nevertheless, the progressive decline in hemoglobin levels to 87 g/L confirmed linezolid-induced hematotoxicity, prompting the initiation of vitamin B6 therapy (100 mg IV qd). This was followed by subsequent hematological recovery, with hemoglobin levels reaching 106 g/L and platelet counts reaching $202 \times 10^9/\text{L}$ by July 31, 2023.

Serial MRI evaluation after 21 days of intensive therapy revealed marked resolution of left knee septic arthritis (Figure 1, panels before discharge). The antimicrobial regimen was subsequently adjusted to minocycline (100 mg PO q12h) in combination with dose-optimized linezolid (300 mg PO q12h) and continued imipenem. However, the emergence of vestibular toxicity (vertigo, tinnitus, emesis) necessitated replacement of minocycline with moxifloxacin (400 mg PO qd). Post-discharge continuation therapy included: linezolid (300 mg PO q12h), moxifloxacin (400 mg PO qd), and vitamin B6 (30 mg PO q8h), sustained for a total duration of 24 weeks. At the 6-month follow-up, the patient exhibited significant improvement in clinical symptoms, and radiological findings corroborated ongoing joint repair (Figure 1, panels 24-week follow-up).

Table 1 The Results of Antimicrobial Susceptibility Testing of *Mycobacterium wolinskyi* Isolated in Our Case

Antibiotics	Sensitivity
Piperacillin/Tazobactam	R
Cefotaxime	R
Ceftazidime	R
Imipenem	S
Ciprofloxacin	S
Amikacin	S
Trimethoprim / Sulfamethoxazole	R
Vancomycin	R
Minocycline	S
Tobramycin	R
Linezolid	S
Clarithromycin	R
Moxifloxacin	S
Doxycycline	R

Abbreviations: S, susceptible; R, resistant.

Due to the patient's refractory SSI, as well as the multiple revisions of the antibiotic treatment regimen necessitated by metabolic syndrome, the diagnostic and therapeutic process was complex and extended over a period of more than eight months. To enhance clarity and facilitate understanding of the patient's clinical course, we employed a Gantt chart to visually represent the timeline of the diagnostic and therapeutic interventions (Figure 3).

Discussion

Our report presents a case of refractory surgical site infection (SSI) caused by *M. wolinskyi* which is a rare bacterial pathogen. The patient had been suffering from the infection for one month before presenting to us. During this period, she underwent debridement surgery and received multiple courses of antibiotic therapy. However, the treatment outcomes were unsatisfactory. At this stage, the isolation and identification of the critical pathogen, along with its antimicrobial susceptibility results, were of crucial importance for the successful anti-infective treatment. Thus, the clinician consulted

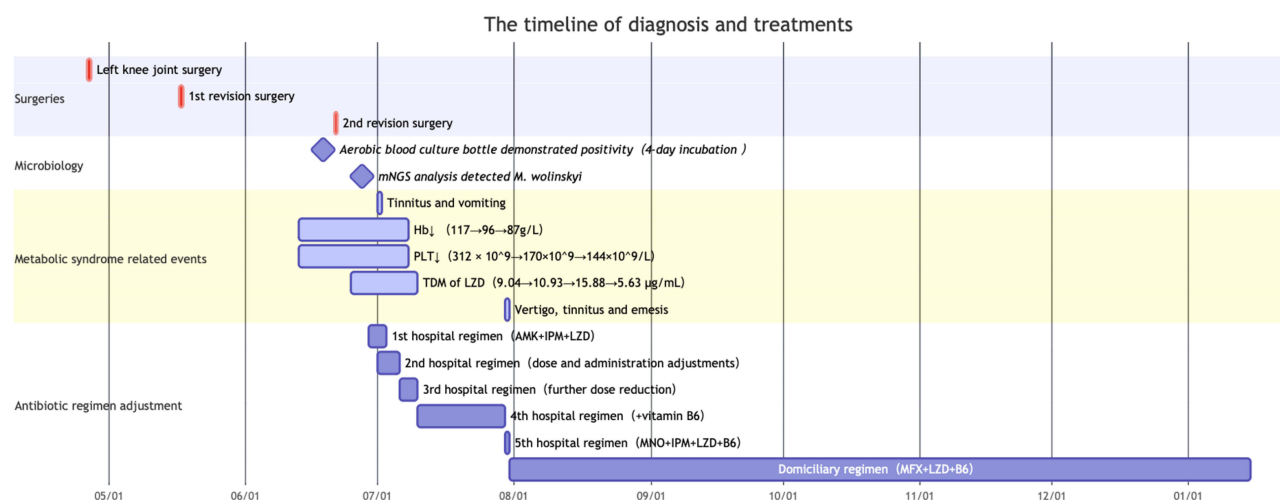


Figure 3 The Gantt chart illustrates the detailed diagnosis and treatment timeline for our case.

Abbreviations: mNGS, metagenomic next-generation sequencing; *M. wolinskyi*, *Mycobacterium wolinskyi*; Hb, hemoglobin; PLT, platelets; TDM, therapeutic drug monitoring; LZD, Linezolid; AMK, Amikacin; IPM, Imipenem; MNO, Minocycline; MFX, Moxifloxacin.

the laboratory microbiologist to catch the pathogen. Considering the possibility of a rare bacterial infection, the microbiologist proposed that the joint fluid should be injected into blood culture bottles for both aerobic and anaerobic culture, and the infected tissue could be sent for mNGS analysis. These two steps were critical for achieving successful pathogen isolation during the initial culture attempt, whereas other reported cases required multiple rounds of culturing.^{12,13,16–20} This approach offers a practical reference protocol for the etiological diagnosis of refractory infections, meriting widespread implementation.

However, difficulties were encountered in identifying the bacteria. Both conventional biochemical reactions (Vitek 2 automated system) and protein mass spectrometry (MALDI-TOF) failed to identify the pathogen, as previously reported.^{8–11} Subsequently, universal primers targeting the 16S rRNA gene were used in an attempt to amplify and sequence the pathogen; however, this effort also proved unsuccessful due to overlapping sequencing peaks, a phenomenon that had not been previously reported in the literature. Fortunately, mNGS analysis obtained 11 RPM of *M. wolinskyi* in the tissue sample which was collected during the debridement surgery. Thereupon, targeted primers for 16S rRNA (F: 5'-GAGAATTCGTGCTTAACACATGCAAGTCG-3', R: 5'-ATGGATCCGTGAGATTTACGAACAACGC-3') and *rpoB* (F: 5'-CGACCACTTCGGCAACCG-3', R: 5'-TCGATCGGGCACATSCGG-3') were designed, and satisfactory identification rates (100% and 99.08%) were obtained. The diagnostic challenges we encountered underscore the critical role of molecular diagnostic tools—such as 16S rRNA, *hsp65*, and *rpoB* gene sequencing, whole-genome sequencing, and mNGS—in enabling the rapid and accurate identification of difficult-to-diagnose NTM species in clinical settings. Moreover, real-time nanopore sequencing has shown increasing potential in rapidly identifying both drug-susceptible and drug-resistant *Mycobacterium tuberculosis*.²¹ This technology may offer comparable diagnostic advantages for rare NTM infections when conventional methodologies prove inadequate.

M. wolinskyi belongs to the *M. smegmatis* group,¹ which is susceptible to ethambutol but resistant to clarithromycin, helping to distinguish it from other RGM.^{3,16} Resistance to clindamycin may be related to the *erm* (38) gene.^{22,23} Currently, there is no established standardized antimicrobial regimen for treating *M. wolinskyi* infections. Accordingly, clinical pharmacists tailored the antibiotic regimen based on AST results of the isolated strain (Table 1). However, during treatment, the patient experienced multiple antibiotic adverse effects, including amikacin-induced tinnitus and vomiting, linezolid-related myelosuppression, and minocycline-associated vestibular dysfunction, which significantly impaired treatment adherence. With the guidance of pharmaceutical experts and careful TDM throughout the treatment, the patient's knee joint infection demonstrated a favorable response. However, contrary to our case, the occurrence of antibiotic adverse effects has been rarely reported in the published *M. wolinskyi* infection cases. Ariza-Heredia et al⁵ reported leukopenia with linezolid, Karakala et al¹² noted a sulfa allergy, Fujikura et al²⁰ observed thrombocytopenia, and Muranaka et al⁸ described nausea with moxifloxacin. What makes our patient so distinctive might be associated with her metabolic syndrome (diabetes, hyperlipidemia and hypertension)²⁴ combined with autoimmunity and rheumatic disease (gout). Alterations in the immune-metabolic cross-talk contribute to the development of autoimmune diseases and metabolic syndrome,²⁵ resulting in metabolic dysfunction and immunosuppression in the patient. Consequently, the patient was infected with the opportunistic bacterium *M. wolinskyi* and had adverse reactions to multiple antibiotics.

The high cost and specialized technical requirements of mNGS may limit its widespread integration into clinical practice. Furthermore, there remains a paucity of long-term efficacy data for pharmacist-guided antibiotic regimens in managing infections caused by rare pathogens.

Conclusion

Our case contributes to the broader understanding and improved management of NTM infections in patients with metabolic syndrome. Diagnosing and managing infections caused by rare pathogenic bacteria requires a multidisciplinary approach that integrates the expertise of microbiologists, pharmacologists, infectious disease specialists, and surgeons. Widespread adoption of advanced laboratory techniques—such as 16S rRNA and *rpoB* gene sequencing, mNGS, and therapeutic drug monitoring of blood concentrations—can significantly enhance both diagnostic accuracy and treatment efficacy for challenging pathogens in similar complex infectious cases.

Data Sharing Statement

The raw data presented in this article will be made available by the authors without undue reservation. For data inquiries, please contact the last corresponding author (xiexy5@mail.sysu.edu.cn).

Ethics Approval and Consent to Participate

This was a retrospective case report study conducted in a tertiary hospital and was approved by the Clinical Research and Ethics Committee of the Sun Yat-Sen Memorial Hospital (No. SYSKY-2024-521-01). The patient provided consent for the publication of the case details.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors report no conflicts of interest in this work.

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