International Medical Case Reports Journal

Dovepress
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CASE REPORT

Mycobacterium abscessus Cervical Lymphadenitis Mimicking Tuberculosis in China: A Case Report

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Abstract: *Mycobacterium abscessus* (Mab) is recognized as the most pathogenic mycobacterium affecting humans, comprising three phylogenetic subspecies: *M. abscessus subsp. abscessus, M. abscessus subsp. bolletii*, and *M. abscessus subsp. massiliense*. Its clinical manifestations are varied and frequently subject to misdiagnosis. Mab exhibits both intrinsic and acquired resistance to numerous antibiotics, which results in limited therapeutic options and poor clinical outcomes. This report details a case of cervical lymphadenitis caused by Mab, initially misdiagnosed as tuberculosis. During anti-tuberculosis therapy, the patient's symptoms initially improved but subsequently deteriorated. Diagnosis of Mab infection was confirmed through high-throughput sequencing. However, due to the delay in accurate diagnosis and treatment missteps, the patient ultimately succumbed to a central nervous system (CNS) Mab infection. Consequently, early and precise diagnosis is imperative for the effective management of Mab infections.

Keywords: tuberculosis, *Mycobacterium abscessus*, diagnosis

Introduction

The incidence of human nontuberculous mycobacterial (NTM) infections has increased markedly in recent years. In Germany, the application of machine learning algorithms to health claims data revealed a significant underestimation of NTM infection cases. The study found that the actual prevalence and incidence rates in 2021 were fivefold and ninefold higher, respectively, than previously reported, highlighting the growing burden of NTM infections, which often go undiagnosed.[1–3] Mab is the most pathogenic rapidly growing mycobacterium affecting humans, leading to pulmonary and extrapulmonary infections, such as lung, skin, soft tissue, CNS infections, as well as bacteremia, particularly in immunosuppressed individuals. This organism is notorious for its resistance to many antibiotics and its ability to form biofilms. The pathophysiology of Mab infections is complex and involves various host and microbial factors. The rising incidence of Mab infections and the emergence of strains with heightened virulence pose significant public health concerns.[3]

Mab is typically transmitted through contaminated water, dust, and inhaled or ingested aerosols.[4] Its enhanced growth and survival in the presence of particulates such as kaolin, halloysite, silicon dioxide, and house dust suggest that contaminated fomites may also serve sources of transmission, underscoring the role of the environment in Mab spread.[5] Consequently, the frequency and clinical presentation of Mab infections vary geographically due to environmental differences and population-specific susceptibilities.

Members of the Mab complex, particularly *M. abscessus subsp. abscessus*, are frequently misidentified as other bacterial infections, leading to delayed diagnosis and treatment.[6] The occurrence of Mab cervical lymphadenitis is indeed

https://doi.org/10.2147/IMCRJ.S600141
Received: 14 February 2026
Accepted: 1 April 2026
Published: 14 April 2026

a rare clinical phenomenon. This rarity is underscored by the limited number of documented cases and the challenges associated with its diagnosis. One case that highlights the diagnostic complexity involved is that of an immunocompetent female with Mab cervical lymphadenitis, which was initially suspected to be malignancy.[7] The clinical presentation can closely resemble more common conditions, such as tuberculosis or even malignancies. In this study, we present a case of Mab cervical lymphadenitis mimicking tuberculosis, which led to death from central nervous system infection in China.

Case Presentation

A 23-year-old female patient from Xinxiang, Henan Province, China, presented with right cervical lymph node enlargement for one month (Figure 1A). The affected area did not exhibit tenderness or mobility and had a well-defined boundary. The local physician administered antibiotic treatment to the patient, but symptomatic improvement was not evident, which the specific plan is unknown. The patient was healthy, with no history of allergies, no family history, no long-term medication use, and no history of immunosuppressive agent use. The patient was a local college student with no travel history outside China.

Cervical computed tomography (CT) revealed right cervical lymph node lesions, indicating a tuberculosis infection (Figure 1B). Serum tuberculosis antibody test results were positive. The erythrocyte sedimentation rate increased, but T-SPOT.TB (T-SPOT) was negative. Chest CT and pulmonary function tests revealed no obvious abnormalities. Examinations of *Aspergillus* galactomannan, β-D-glucans, respiratory viruses, complete blood count, tumor markers, autoimmune disease antibodies, and enzyme-linked immunosorbent assay (ELISA) tests for human immunodeficiency virus (HIV) were negative. Acid-fast staining and GeneXpert MTB/RIF tests of the puncture fluid were also negative.

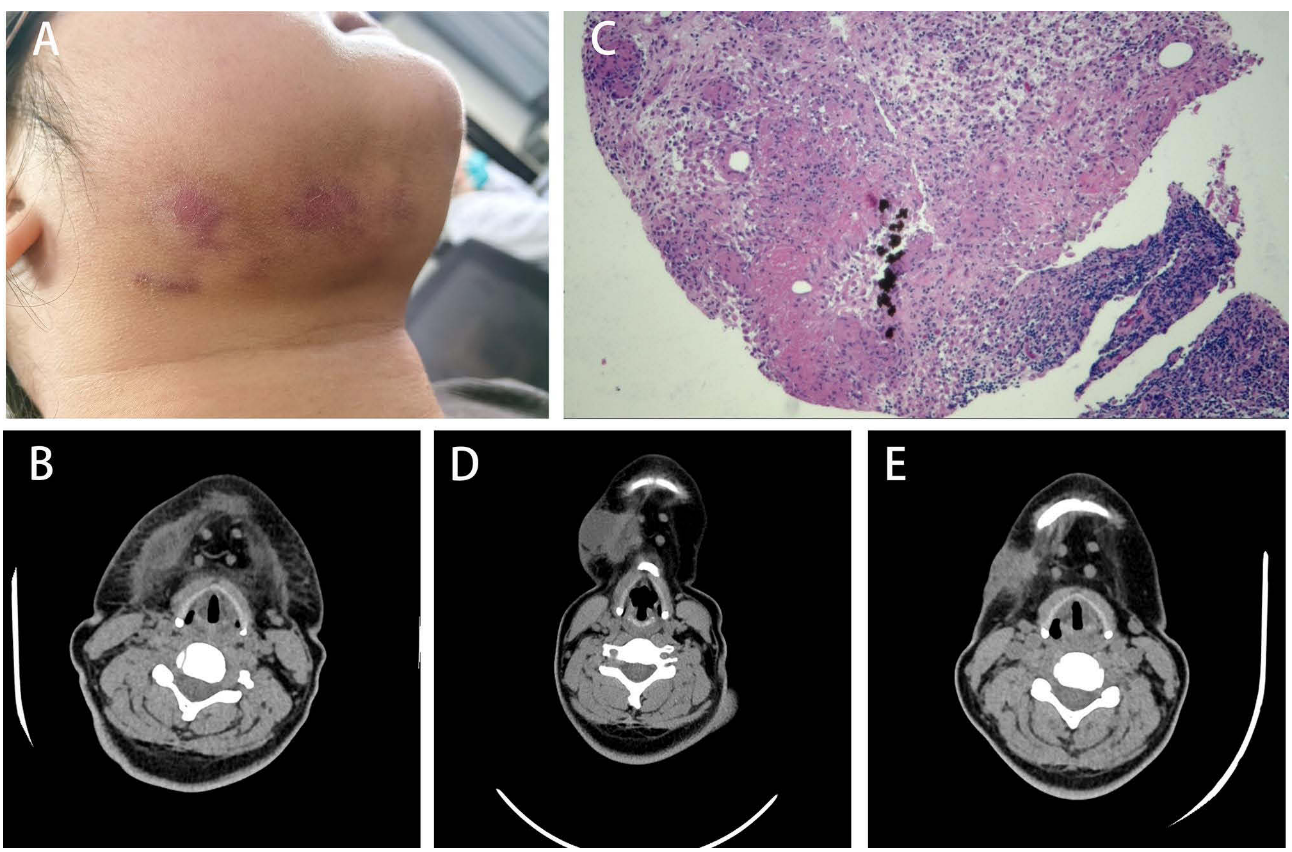

Figure 1 Right submaxillary lesions and pathological changes. (**A**) The right cervical lymph node enlargement. (**B**) Cervical CT revealed multiple nodular and clustered slightly low-density shadows with blurred boundaries and partial fusion into a mass was observed in the right submandibular region, reflecting tuberculosis infection. (**C**) The results of cervical lymph node puncture revealed focal caseous necrosis and epithelial-like cells, and tuberculosis was not excluded. (**D**) Cervical CT revealed multiple round low-density shadows, larger lesions than before, and more local soft tissue density shadows in the right submandibular region. (**E**) Cervical CT revealed a noticeable resolution of the right submandibular lesion and significantly reduced soft tissue density shadow.

Nevertheless, tuberculosis could not be excluded, as cervical lymph node puncture displayed focal caseous necrosis and epithelioid cells (Figure 1C). Initially, a four-drug regimen (daily standard weight-based dosing of rifampicin [600 mg], ethambutol [1000 mg], isoniazid [300 mg], and pyrazinamide [1500 mg]) was prescribed for suspected tuberculosis of the cervical lymph nodes.

After two months of tuberculosis treatment, the patient complained of improved symptoms. However, the patient's neck swelling symptoms worsened during the third month of anti-tuberculosis treatment. Cervical CT revealed re-expansion of the right submaxillary lesions (Figure 1D). The patient may have been misdiagnosed. Mab was identified in the cervical lymph node pus using high-throughput sequencing. The patient was switched to rifabutin, linezolid, amikacin, and clarithromycin for the treatment of nontuberculous mycobacteria for two months.

Cervical CT revealed resolution of the right submaxillary lesions (Figure 1E). However, the patient developed a worsening headache. After eight weeks, cerebrospinal fluid culture showed Mab. With the diagnosis of invasive CNS infection caused by Mab, antimicrobial therapy was adjusted to include tigecycline, linezolid, amikacin, and clarithromycin. However, the patient died from irreversible CNS involvement.

Discussion

The misdiagnosis of Mab as TB is a significant clinical challenge, particularly in regions where TB is endemic. This misdiagnosis often arises due to the overlapping clinical and radiological features of NTM infections and TB, as well as the limitations of conventional diagnostic methods. For instance, a study conducted in Mali highlighted the prevalence of NTM infections among patients initially treated for TB, emphasizing the need for high-throughput sequencing to accurately differentiate between these infections.[8] Similarly, the challenges in distinguishing between NTM and TB were underscored in a case report in which a patient was misdiagnosed with TB for three years before NTM was correctly identified using high-throughput sequencing.[9]

The diagnostic conundrum is further complicated by the fact that NTM, including Mab, can present with non-specific symptoms that mimic TB, such as chronic cough and fever. The clinical manifestations of Mab infection include abscesses, panniculitis, nodules, cellulitis, ulcers, and sinus tract drainage.[10] Diagnosis relies on isolating the organism from clinical specimens; however, Mab is challenging to detect using Gram staining and standard cultures. This was illustrated in a case in which Mab was initially misdiagnosed as TB, leading to ineffective treatment until the correct diagnosis was made through high-throughput sequencing.[11] The importance of accurate species identification is under-scored by studies emphasizing the role of high-throughput sequencing in distinguishing NTM from TB, thereby preventing misdiagnosis and ensuring appropriate treatment.[12,13]

The Ziehl-Neelsen staining technique is a cornerstone in the detection of acid-fast bacilli. The diagnostic insights rely on the presence of a sufficient bacterial load for visualization. However, in the context of Mab, which may present with atypical clinical manifestations and lower bacterial loads, the sensitivity of Ziehl-Neelsen staining can be suboptimal.[14] Coupled with the long turnaround time for antimicrobial susceptibility testing, these challenges frequently delay accurate identification and timely initiation of appropriate therapy. This limitation underscores the importance of high-throughput sequencing, which offers enhanced sensitivity and specificity. It allows for the rapid and precise identification of specific mycobacterial DNA sequences, which might not be detectable through traditional staining methods.[15]

The process of Mab infection shares remarkable similarities with that of *M. tuberculosis* infection, including granuloma formation and persistent infection. After colonization, macrophages and neutrophils are recruited to phago-cytose the bacteria, and the innate immune response initially contains the infection. However, Mab can resist phagosomal defenses and induce the generation of inflammatory cytokines, such as tumor necrosis factor (TNF), which promotes further immune-cell recruitment and granuloma formation.[16] During persistent infection, Mab undergoes an irreversible transition from a smooth (S) to a rough (R) variant, leading to granuloma breakdown and the formation of large extracellular bacterial cords.[17] The biological and environmental triggers driving this transition remain unknown.

Mab exhibits two phenotypically distinct morphotypes, S and R, defined by the presence or absence of glycopeptidolipids (GPLs) in the cell wall.[18] Both variants can originate from the same isolate, coexist within the host, and evolve differently in response to host immunity, leading to distinct clinical outcomes and disease manifestations.[19] The transition of Mab from a smooth (S) to a rough (R) morphotype is a critical factor in its pathogenicity, particularly in cases of lymphadenitis. This transition is characterized by a reduction in GPL production, which is well-documented as a key factor in the increased virulence associated with the R morphotype. The study titled "Loss of LpqM proteins in *Mycobacterium abscessus* is associated with impaired intramacrophage survival" provides insights into the genetic and molecular mechanisms underlying this transition.[20]

Treatment of Mab infections presents its own set of challenges due to the organism's intrinsic resistance to many antibiotics commonly used for TB. Current treatment recommendations for Mab involve a combination of antibiotics, including macrolides like clarithromycin and aminoglycosides such as amikacin. The resistance to clarithromycin is a growing concern, as demonstrated by studies showing inducible resistance in Mab isolates.[21] During treatment, this patient's clinical symptoms improved, and cervical lymph node enlargement was reduced, however, she died a of CNS infection in the later stage, which may be related to inducible resistance and the S-to-R transition of the Mab. Various pathogenic factors confer upon Mab an exceptional ability to produce diverse infection patterns. Most of these determinants are related to the cell envelope structure and overall bacterial composition. The capacity of Mab to shift into more virulent morphotypes enables immune evasion and promotes systemic dissemination. Its distinctive mycobacterial outer membrane (MOM) and multiple efflux pumps create a robust barrier against both host defenses and many antibiotics. Moreover, the ESX-4 Type VII secretion system equips Mab with mechanisms to modulate host immunity, driving more aggressive infection progression.[22] Therefore, it is necessary to pay greater attention to the cases of cervical lymph node involvement combined with central nervous system infection caused by Mab, and further explore the mechanisms by which *Mycobacterium abscessus* invades the central nervous system.

This study is subject to several limitations. Firstly, there was a lack of timely consideration by clinicians regarding the differential diagnosis between Mab and tuberculosis infections. Secondly, the initial treatment regimen for patients involved anti-tuberculosis therapy, which proved ineffective in controlling the condition and subjected patients to potential adverse effects of anti-tuberculosis medications, including hepatic and renal impairments. Thirdly, delays in the treatment of patients with CNS infections exacerbated their clinical conditions. Furthermore, numerous aspects of this organism's infection mechanisms remain insufficiently understood, necessitating further research for comprehensive characterization.

Conclusion

In summary, this is an uncommon instance of Mab infection resembling tuberculosis. This article seeks to enhance comprehension of Mab diseases and address these neglected and significant global threats. It is crucial not only to prevent incorrect TB treatment but also to direct effective management of Mab infections. Developing new antibiotics and treatment plans, along with implementing systematic diagnostic protocols, is vital to tackle the increasing challenge of Mab infections in clinical settings.

Abbreviations

Mab, *Mycobacterium abscessus*; NTM, Nontuberculous mycobacteria; CT, Computed Tomography; TB, Tuberculosis; T-SPOT, T-SPOT.TB; ELISA, Enzyme-Linked Immunosorbent Assay; CNS, Central Nervous System; TNF, Tumor necrosis factor; GPLs, Glycopeptidolipids; S, Smooth; R, Rough; MOM, Mycobacterial outer membrane; HIV, Human Immunodeficiency Virus; ESX-4, Type VII secretion system-4.

Data Sharing Statement

Available upon request. The data is available from the correspondent author 1.

Ethics Approval

This investigation was reviewed and approved by the Institutional Review Board of The First Affiliated Hospital of Xinxiang Medical University.

Consent for Participation and Publication

Informed consent for participation was obtained from the patient during the hospitalization. Informed consent for publication was acquired from the patient during the hospitalization. Publishing the case details did not require institutional approval.

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.

Funding

There is no funding to report.

Disclosure

The authors report no conflicts of interest in this work.

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