Infection and Drug Resistance

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CASE REPORT

Mycobacterium arosiense Infection in a Patient with CD209 Mutation: A Rare Case Report

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Abstract: We report a case of an adult male with a history of recurrent cough and chest pain for five years. *Mycobacterium arosiense* was identified in his alveolar lavage fluid, and whole-exome sequencing revealed heterozygosity for CD209 in this patient. After 17 months of combined antibiotic therapy, the patient recovered completely.

Keywords: Mycobacterium arosiense, CD209 mutation, pneumonia

Case Introduction

A 26-year-old male, as an ordinary employee working in a corporate office, presented with a five-year history of recurrent cough and chest pain. Over the past two years, he had received intermittent treatment with various antibiotics, including moxifloxacin, imipenem cilastatin, and piperacillin tazobactam, with temporary improvement followed by relapse. A chest computed tomography (CT) scan revealed no abnormal lung lesions. Bronchoscopy showed patchy necrotic material covering the mucous membranes of the main trachea and bronchi. Tuberculosis skin tests and T-SPOT assays were negative. Most blood markers were within normal limits, and multiple microbiological tests on sputum, urine, stool, blood, and pharyngeal swabs were negative for bacteria and viruses. Next-generation sequencing (NGS) of bronchoalveolar lavage fluid identified *Pseudomonas aeruginosa* (sequence number 128043, relative abundance 99.24%) and *M. arosiense* (sequence number 268, relative abundance 0.21%). Based on these findings, the patient was diagnosed with bronchial infection due to *M. arosiense* and *P. aeruginosa* colonization and treated with rifampin, ethambutol, moxifloxacin, and azithromycin. His symptoms resolved, and he remained asymptomatic for over one year after completing 17 months of therapy. The microbiological cure was confirmed by NGS of bronchoalveolar lavage fluid after 3 years of therapy. Six bronchoscopies were performed to monitor endotracheal and endobronchial lesions (Figure 1).

Discussion

M. arosiense is a slow-growing, yellow-pigmented, scotochromogenic nontuberculous mycobacteria (NTM) species.[1] It was first reported in 2008 in a young boy with hereditary partial gamma interferon receptor alpha-1 deficiency and osteomyelitic bone lesions.[1] Clinically, *M. arosiense* is rarely encountered, and its identification often relies on advanced molecular techniques like metagenomic NGS (mNGS), which can detect all microorganisms in a sample.[2]

In vitro studies show that *M. arosiense* is sensitive to clarithromycin, rifamycins, amikacin, moxifloxacin, linezolid, and clofazimine but resistant to isoniazid, fluoroquinolones, and streptomycin.[1] The young patient improved significantly after receiving a combination of rifampin, ethambutol, moxifloxacin, and azithromycin.

Received: 7 October 2025
Accepted: 8 December 2025
Published: 16 December 2025

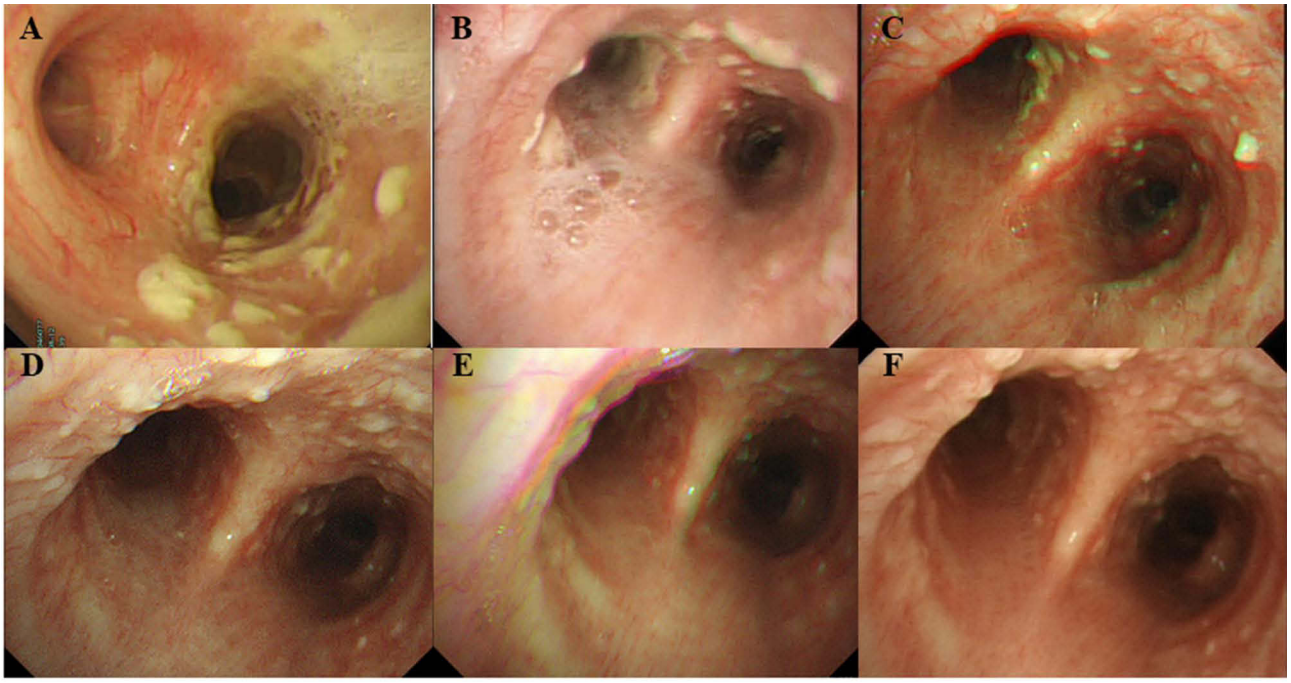

Figure 1 Bronchoscopy of the patient during the therapy. (**A**) Bronchoscopy before therapy; (**B**) After 2 months of therapy; (**C**) After 4 months of therapy; (**D**) After 9 months of therapy; (**E**) After 17 months of therapy; (**F**) After 3 years of therapy.

CD209, an autosomal dominant gene located on chromosome 19p13.3, encodes Dendritic Cell-Specific ICAM3-Grabbing Non-integrin (DC-SIGN), a C-type lectin expressed on dendritic cells and alveolar macrophages.[3] DC-SIGN binds various ligands, including pathogens like Mycobacterium tuberculosis, HIV-1, and Dengue virus.[4,5] It plays a critical role in Mycobacterium tuberculosis infection by facilitating internalization and immune suppression.[6–8] The patient's whole-exome sequencing revealed a heterozygous CD209 mutation (NM_021155.3:c.220C > T [p.Gln74*]), classified as a variant of uncertain significance.[9,10] While CD209 promoter polymorphisms have been associated with infectious disease susceptibility, the relationship between CD209 mutations and NTM infections remains unclear.

This is the first reported case of pulmonary *M. arosiense* infection with a confirmed CD209 mutation. Previous cases have not explored genetic factors. However, there were also several limitations in this study. First, the sensitivity results of *M. arosiense* were lack. In addition, the exact functional impact of the CD209 mutation in relation to *M. arosiense* infection remains unclear. Therefore, further genetic testing in refractory NTM cases is warranted to investigate potential links between genetic mutations and specific infections.

Conclusion

This case highlights a rare instance of *M. arosiense* pneumonia in a patient with a CD209 mutation. While the patient responded well to antibiotic therapy, the role of the CD209 mutation in susceptibility remains uncertain. Further studies are needed to clarify the relationship between genetic factors and NTM infections.

Data Sharing Statement

This paper does not report data generation or analysis.

Ethics Approval and Consent to Participate

This study was conducted following the Declaration of Helsinki and obtained approval from the clinical research ethics committee of The First Affiliated Hospital, Zhejiang University School of Medicine [No. 20241303]. The patient gave written informed consent for his personal and clinical details along with any identifying images to be published in this study. The institutional approval was not required to publish the case details.

Consent to Publish

All authors have seen and approved the content.

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

This work was supported by the National Key R&D Program of China (2022 YFC2304505 and 2023YFC2306800) and Department of Health of Zhejiang province (2023XY037). The funder had no role in study design, data collection and analysis, decision to publish or preparation of the manuscript.

Disclosure

The authors report no conflicts of interest in this work.

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