**Case Report** **OPEN ACCESS**

# Mycobacterium gordonae pulmonary infection in an immunocompetent adult

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## Abstract

**Context**: Nontuberculous mycobacteria are non-communicable organisms and currently there are over 125 species. Nontuberculous mycobacteria are usually recovered from the environment and can cause disease through respiratory, cutaneous, parenteral and gastrointestinal exposure. **Case Report**: We describe a case of a young, immunocompetent patient that developed symptomatic *Mycobacterium gordonae* pulmonary infection. A computed tomography of the chest revealed hilar lymphadenopathy and nodular densities on the left side. Definitive diagnosis was made by a culture of a transbronchial biopsy. Treatment consisted of a short course of rifampin and ethambutol, which resulted in a clinical and radiographic improvement. **Conclusions**: *Mycobacterium gordonae* is capable of causing clinically significant disease in both immunocompetent and immunosuppressed individuals.

**Keywords**: Nontuberculous mycobacteria, rifampin, cure, contaminant

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## Introduction

Of all the mycobacteria species, *Mycobacterium gordonae* (*M. gordonae*) is the least pathogenic and its isolation is typically regarded as a contaminant. The organism is ubiquitous and it is most commonly isolated from soil and water. Nosocomial transmission has also been reported due to tap water used for rinsing of medical instruments and dye added to topical anesthetic used in bronchoscopy [1]. Despite its non-virulent nature, there have been reports of clinically significant disease caused *by M. gordonae* [3]. We report a case of symptomatic pulmonary infection in a young, immunocompetent woman. *M. gordonae* pulmonary disease occurring in a patient without a previous pulmonary abnormality is extremely uncommon.

## Case Report

A 26 year old female with no previous medical conditions presented to the emergency room for acute worsening of a non-productive cough, dyspnea and chest tightness. The patient denied any fevers, night sweats, weight loss, hemoptysis, and exposure to sick contacts or a history of cigarette smoking. She stated that her symptoms developed approximately 3 months prior to presentation. The patient had been empirically treated as an outpatient by her primary care physician with albuterol and singulair with no improvement.

On admission vital signs were stable and physical examination was unremarkable. Laboratory data including comprehensive metabolic panel and complete blood count with differential was unrevealing. Contrasted computed-tomography (CT) of the chest revealed left sided hilar adenopathy and several nodular densities throughout the left lower lobe.

Sputum for acid-fast smear and culture were negative. Tuberculin skin test was non-reactive and a blood test for HIV was negative. Bronchoscopy with bronchoalveolar lavage and a transbronchial biopsy was preformed. The biopsy revealed acid fast bacilli and necrotizing granulomatous inflammation. The patient was started on anti-tuberculous medication consisting of rifampin, isoniazid, pyrazinamide and ethambutol. Rifampin was discontinued after only one week because the patient developed fever and a diffuse rash. The symptoms resolved after the rifampin was stopped.

Two months after starting TB therapy, the cultures from the tissue biopsy grew *M. gordonae*. At this time the patient was completely asymptomatic and a follow up CT chest revealed significant improvement. The patient was observed off of all therapy and continued to do well clinically. A repeat CT scan of the chest was done 6 months after initial diagnosis and showed complete resolution of the adenopathy and only one nodule was present in the left lower lobe.

# Discussion

*M. gordonae*, formerly called *Mycobacterium aquae*, is slow growing mycobacteria, usually requiring up to 3 weeks to reach mature growth [2]. The organism grows best at a temperature range of 35-37 °C and can be recovered from pipelines, fresh water and laboratory faucets [2]. *M. gordonae* is typically thought to be a contaminant; however there are numerous reports of the organism causing disease particularly in immunosuppressed individuals. Corticosteroid therapy, HIV infection, malignancy, organ transplant recipients, and those in extreme of age groups are all underlying conditions and patient populations in which the bacteria have caused significant disease [3]. There have only been rare reports of immunocompetent individuals such as our patient that have developed symptomatic disease [4].

Infection involving the peritoneum, soft tissue, cornea, genitourinary system and disseminated disease has been described; but pulmonary infection is the most likely site of symptomatic disease [3, 5]. In a review of 24 cases of infection with *M. gordonae*, 8 patients had lung involvement [3]. Unilateral pulmonary involvement was present in 6 of the cases. All of the patients had at least one antecedent pulmonary abnormality including a history of infection with tuberculosis, pulmonary malignancy, chronic bronchitis and smoking. Symptomatic pulmonary infection in individuals with no pulmonary abnormalities as seen in our patient is unusual.

The presence of clinical symptoms and radiographic abnormalities are required for the diagnosis of *M. gordonae* pulmonary infection [2]. Common symptomatic manifestations include cough, weight loss, dyspnea, hemoptysis and fever [3]. A variety of radiographic finding are possible including pulmonary nodules, cavities, infiltrates, bronchiectasis and consolidation. In fact, one study concluded that regardless of the NTM species, the most common pulmonary findings on a CT scan were small bilateral nodules or branching centrilobular lesions [6]. In addition to clinical symptoms and radiographic abnormalities, positive cultures from sputum, bronchial wash or transbronchial or lung biopsy are necessary [2]. Positive cultures of sputum and bronchial wash should be interpreted with caution since these cultures are more likely to represent contamination in the absence of clinical and radiographic evidence. Commercial DNA probes to rapidly identify *M. gordonae* are available.

The treatment regimen for *M. gordonae* infection is not well defined but antimicrobial agents that have the most consistent in vitro activity include ethambutol, rifamycins, clarithromycin, linezolid and the flouroquinolones [2]. In addition, the organism is sometimes sensitive to cycloserine, trimethoprim and sulfamethoxazole [7]. *M. gordonae* is resistant to isoniazid [7]. Duration of therapy is also ill defined and in one review ranged from 9 to 22 months with a median of 15 months [3]. Cure is seldom achieved even with effective treatment but the disease can be stabilized and result in symptomatic improvement [7]. In our patient rifampin and ethambutol provided effective coverage against the organism, but treatment with rifampin was discontinued after only one week due to allergy development and treatment with ethambutol was continued for 2 months. Despite the short treatment course and the use of only ethambutol as effective coverage, our patient achieved symptomatic improvement and almost complete resolution of the pulmonary lesions 6 months after initial diagnosis.

Although commonly regarded as a contaminant, *M.gordonae* is capable of causing significant infections in both immunocompromised and immunocompetent hosts and its isolation should not completely be dismissed without further evaluation.

# References

1. Wallace RJ, Brown BA, Griffith DE. Nosocomial outbreaks/pseudo-outbreaks caused by non-tuberculous mycobacteria. Annu Rev Microbiol 1998; 52:453-490.
2. Griffith D, Aksamit T, Brown-Elliott B et al.An official ATS/IDSA Statement: Diagnosis, treatment and prevention of nontuberculous mycobacterial diseases. Am J Respir Cirt Care Med 2007; 175: 367-416.
3. Weinberger M, Berg SL, Feurstein IM, Pizzo PA, Witebsky FG. Disseminated infection with Mycobacterium gordonae: Report of a case and critical review of the literature. Clin Infect Dis 1992; 14:1229-1239.
4. Resch B, Eber E, Beitzke A. Bauer C, Zach M. Pulmonary infection due to Mycobacterium gordonae in an adolescent immunocompetent patient. Respiration 1997; 64: 300-303.
5. Pinho L, Santos J, Oliveira G, Pestana M. Mycobacterium gordonae urinary infection in a renal

transplant recipient. Transpl Infect Dis 2009; 11:253-256.
6. Jeong YJ, Lee KS, Koh WJ, Han J, Kim TS, Kwon OJ. Nontuberculous mycobacterial pulmonary infection in immunocompetent patients: comparison of thin section CT and histopathologic findings. Radiology 2004; 231: 880-886.
7. Pozniak A, Bull T. Recently recognized mycobacteria of clinical importance. J Infect 1999; 38: 157-161.