Case Report

Pulmonary infection with caseating mediastinal lymphadenitis caused by *Mycobacterium gordonae*

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**ABSTRACT**

It is often difficult to discern true mycobacterial infection from colonization due to *Mycobacterium gordonae* since this organism is ubiquitous and is commonly an innocuous saprophyte. This study reports a rare case of caseating hilar adenopathy and pulmonary disease caused by *M. gordonae* in a patient with chronic obstructive pulmonary disease (COPD) and rheumatoid arthritis (RA) on maintenance steroids and methotrexate. Pathologic exam and cultures of lymph node excision biopsy and bronchoalveolar lavage (BAL) confirmed the diagnosis.

Triple antimycobacterial therapy with azithromycin, ethambutol and rifabutin was administered. The patient had significant clinical and radiologic improvement and follow-up cultures confirmed microbiologic cure.

*Mycobacterium gordonae* can be a rare cause of significant pulmonary infection, and positive sputum or BAL cultures for *M. gordonae* should not be automatically discarded and considered as nonpathogenic contaminants or colonizing organisms, especially in immunocompromised hosts with comorbidities. A detailed review of the case and relevant literature is provided.

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**Case report**

A 54-year-old white male former plumber was referred for infectious diseases and pulmonary evaluation because of weight loss, worsening productive cough of grayish white sputum, night sweats for several months, and newly noted mediastinal adenopathy. He had a history of oxygen-dependent COPD and disabling rheumatoid arthritis for which he continues on maintenance prednisone 20 mg PO daily and methotrexate 15 mg PO weekly. He smoked one
pack per day for most of his adulthood and quit smoking one month prior to his presentation. On examination, he appeared to be frail and weighed 53 kg. He was tachypneic with a respiratory rate of 23 per minute, pulse of 88 per minute, and a blood pressure of 115/70. His lung exam revealed diminished breath sounds with scattered bilateral rhonchi and basilar crackles. The remainder of his exam was unremarkable except for warm wrist and ankle joints with typical rheumatoid arthritis deformities of his hand joints.

Laboratory work-up revealed a white blood cell count of 11,700/mm$, with 78.6% neutrophils, 18.4% lymphocytes, and 3% monocytes; hemoglobin of 13.3 g/dl; and a platelet count of 380,000/mm$. A comprehensive metabolic panel was within normal limits and his HIV antibody was negative. Computed tomography (CT) scan of the chest showed mediastinal adenopathy in the pretracheal and subcarinal regions, measuring 2.1 × 1.5 and 1.4 × 3.5 cm, respectively, with concomitant interstitial infiltration and honey-combing of both lungs (Fig. 1). CT scanning also showed evidence of changes compatible with the diagnosis of COPD and interstitial lung disease based on the presence of emphysematous bullae and evidence of interstitial lung fibrosis.

He underwent bronchoscopy that showed no evidence of endobronchial lesions. Bronchoalveolar lavage, brushings and transbronchial biopsies were obtained from the right lower lobe anterior segment. He also underwent mediastinal lymph node biopsy by video assisted thoracoscopic surgery. Pathologic exam of the resected lymph node showed caseating granulomatous inflammation (Fig. 2) with well-defined, supplicative, necrotic centers surrounded by palisading epithelioid macrophages (Fig. 3). Mycobacterial cultures of the resected caseating granulomatous lymph node tissue and bronchoalveolar lavage both grew a mycobacterium that formed pigmentation both in the presence and absence of light. This was identified as Mycobacterium gordonae, and the identification was confirmed by the Tennessee State Laboratory. Neither the local nor State laboratory was able to perform genotyping or antimicrobial susceptibility of the isolated organism. The patient was treated with triple antimycobacterial therapy including azithromycin, ethambutol and rifabutin for 18 months with microbiologic cure. Prednisone at 20 mg PO daily that he had received for 2 years before the first positive culture was tapered down to 5 mg after improvement of his infection.

Follow-up sputum cultures collected on multiple subsequent visits revealed no further growth of M. gordonae. Sputum conversion time was approximately 2 months after the start of triple antimycobacterial therapy. His pulmonary symptoms improved, and his CT scan of the chest showed radiologic improvement nine months after initiation of therapy. He regained 30 lb during the first year of therapy.

Discussion

Mycobacteria are facultative intracellular pathogens that are capable of residing in mononuclear phagocytes [1]. Recent advances in techniques such as DNA probes and the

Fig. 1 – Mediastinal adenopathy in the pretracheal and subcarinal regions, measuring 2.1 × 1.5 and 1.4 × 3.5 cm, respectively, with interstitial infiltration and honey-combing of both lungs.

Fig. 2 – Sections of lymph nodes show multiple well-defined, discrete granulomas of variable sizes and different stages. (Hematoxylin-Eosin, original magnification >100).

Fig. 3 – Higher magnification shows well-defined necrotizing granuloma of Mycobacteria gordonae. Palisading epithelioid macrophages are seen surrounding supplicative necrotic center. (Hematoxylin-Eosin, original magnification >200).
recognition of the highly conserved 16-S ribosome RNA gene sequence with hypervariable regions in non-tuberculous Mycobacteria have led to a dramatic increase in the identification of Mycobacteria species [2]. M. gordonae, named after the American bacteriologist Ruth Gordon [3], also formerly called Mycobacterium aquae, or the tap water bacillus, is a common contaminant of water and raw milk, and it may be present in the soil. It is generally considered saprophytic and rarely causes significant lung disease. Isolation of this organism in sputum cultures is not generally considered clinically significant [3–5].

M. gordonae belongs to the Runyon group II, which includes the scotochromogen mycobacteria that produce pigments in the dark. Mycobacterium scrophulaceum is a member of the same group. There are two subgroups among scotochromogens [6], – a Tween hydrolyzing and a non-hydrolyzing group – that can be differentiated by immunodiffusion [7]. M. gordonae is nitrate reductase negative [7] and has a positive Tween hydrolysis test [8].

The American Thoracic Society (ATS) and Infectious Disease Society of America (IDSA) have issued an official statement on the diagnosis, treatment, and prevention of non-tuberculous mycobacterial diseases in 2007 [9]. Diagnosis should be based on the presence of pulmonary symptoms with appropriate exclusion of other etiologies in addition to radiologic and microbiologic evidence of disease caused by non-tuberculous mycobacteria. Radiologic evidence of disease includes nodular or cavitary opacities on chest radiograph or a high resolution CT scan (HRCT) that shows multifocal bronchiectasis with multiple small nodules. Microbiologic confirmation involves positive culture results from at least two separate expectorated sputum samples, positive culture results from at least one bronchial wash or lavage, or transbronchial or other lung biopsy with mycobacterial histopathologic features (granulomatous inflammation or acid fast bacilli) and a positive culture for non-tuberculous mycobacteria or biopsy showing mycobacterial histopathologic features (granulomatous inflammation or acid fast bacilli) and one or more sputum or bronchial washings that are culture positive for non-tuberculous mycobacteria [9]. The ATS/IDSA statement further stresses that these criteria fit best with Mycobacterium avium complex (MAC), Mycobacterium kansasii, and Mycobacterium abscessus, while there is not enough known about most other non-tuberculous Mycobacteria to be certain that these diagnostic criteria are universally applicable for all other non-tuberculous species [9].

Although rare, invasive lung disease caused by M. gordonae has been documented in medical literature [5,10,11]. Pneumonitis, interstitial fibrosis, interstitial nodular infiltrates and, rarely, cavitation have all been caused by this organism [4,10,12]. Aguado and colleagues [5] reported a patient with Hodgkin’s disease whose lung biopsy revealed multiple caseating granulomas with a large number of acid fast bacilli on stain, and an abundant tissue culture of growth of M. gordonae. Marchevsky et al. [10] reported growth of M. gordonae with evidence of tissue invasion in lung biopsies of 3 patients with interstitial fibrosis and organizing pneumonia among 40 case series with non-tuberculous mycobacterial lung disease. Similarly, a 26-year-old female patient had left-sided hilar adenopathy and nodules in the left lower lobe. Transbronchial biopsy revealed acid fast bacilli and necrotizing granulomatous inflammation. Two months after starting tuberculosis therapy, the cultures from the tissue biopsy grew M. gordonae. Follow-up CT chest revealed significant improvement with complete resolution of her lung disease [11].

M. gordonae was also reported as an opportunistic pathogen in patients with AIDS with an unexplained pulmonary infection [13]. Besides AIDS, underlying immune suppression, advanced structural lung disease, such as emphysema and pneumoconiosis, alcoholism, chronic lung disease, diabetes mellitus, and malignancy have all been reported as risk factors for invasive infections with M. gordonae [13,14].

Extrapulmonary infections with M. gordonae including skin infection, septic arthritis, osteomyelitis, olecranon bursitis, tenosynovitis, peritonitis, and prosthetic valve infection have also been reported [15–18]. Pseudoinfection due to M. gordonae can occur with contaminated ice, aerosol therapy and dye used during bronchoscopy [19]. In some institutions, contamination of clinical specimens by M. gordonae is a significant ongoing problem. However, this institution’s microbiology laboratory has not encountered problems with this organism contaminating clinical specimen cultures.

Infections caused by M. gordonae have responded well to multiple antimycobacterial agents including rifabutin, ethambutol, clarithromycin, azithromycin, linezolid and fluoroquinolones such as levofloxacin [20–22]. In vitro resistance to isoniazid and pyrazinamide is likely [23]. Several documented cases of M. gordonae-associated pulmonary disease cleared with appropriate therapy [24–26].

It is known that rheumatoid arthritis may have extra-articular manifestations, and rheumatoid lung nodules are the most common pulmonary manifestations of RA. RA by itself may cause granulomas in the mediastinal lymph nodes. Rheumatoid nodules in mediastinal lymph nodes are extremely uncommon [27]. This specific patient had pathology revealing granulomas with caseating necrosis, a finding not typical in the setting of RA.

It is believed that tuberculosis infection is extremely unlikely in this patient. While the PPD testing was negative, clearly this has poor sensitivity in the setting of an immunocompromised individual. Interferon gamma release assays could have been employed, but similarly lose sensitivity with an immunocompromised individual. In addition, this patient had multiple cultures and biopsies performed that did not yield M. tuberculosis.

Caseating mediastinal lymphadenitis with concomitant lung disease caused by M. gordonae infection has not been reported before. This reported case and reviewed literature illustrates the role of M. gordonae as an emerging pathogen, especially in immunocompromised hosts. Positive clinical cultures for this organism should be carefully reviewed and acted upon according to the clinical context.

**Disclaimer**

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Declaration of conflict of interest
None.

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