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A rare case of lung abscess caused by *Mycoplasma pneumoniae* in an adult with chronic obstructive pulmonary disease

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Abstract

Background: Lung abscess is a severe pulmonary infection usually caused by anaerobic or pyogenic bacteria. *Mycoplasma Pneumoniae (MP),* a common cause of community-acquired pneumonia, rarely leads to lung abscess, especially in adults.

Case presentation: We report a 74-year-old male with Chronic Obstructive Pulmonary Disease (COPD) who presented with chest pain, productive cough, and dyspnea. Chest CT revealed a cavitary lesion with an air–fluid level in the right lower lobe. Despite broad-spectrum antibiotics, the lesion progressed. Conventional cultures were negative, but targeted Next-Generation Sequencing (tNGS) of bronchoalveolar lavage fluid identified 29,507 sequence reads of MP, confirming the diagnosis of MP-associated lung abscess. Antimicrobial therapy was switched to levofloxacin, leading to clinical improvement and marked radiological resolution.

Conclusion: This case represents one of the first documented adult cases of MP-induced lung abscess, with COPD as a predisposing factor. It underscores the importance of considering MP in culture-negative lung abscesses, highlights the diagnostic value of tNGS, and emphasizes that early targeted therapy can achieve favorable outcomes in such rare and severe presentations.

Keywords: *Mycoplasma pneumoniae;* Lung abscess; Chronic Obstructive Pulmonary Disease (COPD); targeted Next-Generation Sequencing (tNGS); Atypical pathogens.

Introduction

Lung abscess is a life-threatening pulmonary infection characterized by localized necrosis of the lung parenchyma and subsequent cavity formation [1]. It typically arises as a complication of aspiration, bronchial obstruction, or hematogenous dissemination of infection [2]. The most frequently implicated pathogens are anaerobic bacteria, particularly *Streptococcus species* and *Bacteroides fragilis* [3]. In patients with underlying structural lung disease or impaired immunity, however, less common organisms may also contribute to abscess formation [4].

Mycoplasma Pneumoniae (MP) is a well-recognized cause of acute respiratory tract infections, including tracheobronchitis and Community-Acquired Pneumonia (CAP) [5]. It accounts for approximately 12-35% of CAP cases, with cyclical epidemics occurring every 4-6 years [6]. Transmission is facilitated in crowded settings, and most infections manifest as mild-to-moderate atypical pneumonia [7]. Nevertheless, MP is capable of causing severe pulmonary complications, such as Necrotizing Pneumonia (NP), as well as a wide spectrum of extrapulmonary manifestations involving the skin, cardiovascular, neurological, and hematological systems [8].

While necrotizing pneumonia has occasionally been reported as a complication of MP infection, progression to a frank lung abscess is exceedingly rare [9]. Published cases are predominantly restricted to pediatric patients, with very few well-documented reports in the adult population [10-12]. This rarity may be explained by the pathogen's unique biological characteristics, including the absence of a cell wall and limited capacity for direct tissue destruction, which contrasts with classical pyogenic bacteria [13]. As such, when MP infection leads to extensive parenchymal necrosis and cavity formation, it represents an unusual and diagnostically challenging scenario [1].

Patients with chronic lung diseases, such as Chronic Obstructive Pulmonary Disease (COPD), may be particularly vulnerable to severe or atypical infections due to impaired mucociliary clearance, chronic airway inflammation, and frequent use of corticosteroids or antibiotics [14]. These factors may predispose to unusual presentations and complicate the clinical course [15].

Herein, we describe what we believe to be the first documented case of an MP-associated lung abscess in an adult patient with COPD. This report highlights the importance of considering atypical pathogens in the differential diagnosis of lung abscess, especially when conventional broad-spectrum antibiotics fail, and underscores the role of molecular diagnostic tools such as targeted Next-Generation Sequencing (tNGS) in establishing a timely and accurate diagnosis.

Case presentation

A 74-year-old male with a 10-year history of COPD presented to our hospital with chest pain persisting for more than 10 days. The pain was dull in nature, worsened with coughing, and accompanied by progressive dyspnea, chest tightness, and a productive cough yielding whitish purulent sputum. He denied hemoptysis, gastrointestinal complaints, recent travel, or exposure to sick contacts. He was a former smoker with a 40 pack-year history and reported no alcohol abuse or immunosuppressive medication use. Initial symptomatic treatment at a local clinic provided no relief, prompting referral for further evaluation.

On admission, his vital signs were as follows: temperature, 37.5°C; pulse, 126 beats/min; respiratory rate, 18 breaths/min; and blood pressure, 85/62 mmHg. Oxygen saturation on room air was 90%. Physical examination revealed decreased breath sounds bilaterally without adventitious rales, a regular cardiac rhythm without murmurs, and no abdominal tenderness or peripheral edema.

Laboratory investigations showed leukocytosis (WBC 10.19×10^9/L, neutrophils 9.21×10^9/L), markedly elevated procalcitonin (16.33 ng/mL) and C-reactive protein (211.76 mg/L), hypoalbuminemia (29.7 g/L), and mildly elevated creatinine (136.7 μmol/L). Autoimmune serologies were negative. Viral panels (including EBV, CMV, HSV, adenovirus, RSV, influenza A/B), as well as serological tests for *Mycoplasma pneumoniae* and *Chlamydia pneumoniae,* were negative. Multiple sets of blood cultures and throat swabs yielded no bacterial growth.

Chest CT at presentation demonstrated a cavitary lesion with an air-fluid level in the right lower lobe, consistent with lung abscess (Figure 1). Given these findings, bacterial lung abscess was initially suspected. The patient was empirically started on intravenous linezolid (600 mg every 12 h) plus imipenem/cilastatin (1 g every 8 h). Despite this regimen, he continued to experience intermittent fever, productive cough, and dyspnea. A repeat chest CT obtained 10 days later revealed progressive enlargement of the cavitary lesion with increased air-fluid level (Figures 2 & 3). The small volume of pleural effusion precluded percutaneous drainage.

Further microbiological evaluation was pursued. Sputum cultures were noncontributory but raised the possibility of *Mycoplasma pneumoniae.* Diagnostic bronchoscopy revealed inflamed bronchial mucosa and purulent secretions within the right lower lobe bronchus. Bronchoalveolar Lavage Fluid (BALF) was obtained and subjected to tNGS), which identified 29,507 sequence reads uniquely mapping to *Mycoplasma pneumoniae*, thereby confirming the diagnosis of MP-associated lung abscess.

Given the rising prevalence of macrolide-resistant MP strains, the antibiotic regimen was switched to intravenous levofloxacin (500 mg once daily). Following treatment adjustment, the patient's clinical condition improved significantly, with defervescence, reduced cough and sputum production, and progressive resolution of dyspnea. Inflammatory markers decreased markedly (PCT from 16.33 to 0.28 ng/mL; CRP from 211.76 to 9.7 mg/L). The patient was discharged on oral moxifloxacin (400 mg once daily for 14 days). At the 6-week follow-up, chest CT demonstrated substantial absorption of the cavitary lesion with near-complete resolution of the air-fluid level (Figure 4). The patient remained asymptomatic and stable during subsequent outpatient visits.

Discussion

Lung abscess is a severe form of pulmonary infection most often caused by anaerobes and facultative anaerobes following aspiration or bronchial obstruction [16,17]. MP, in contrast, is generally regarded as an atypical pathogen of CAP, usually leading to mild or moderate disease [18-20]. Although MP is responsible for 12-35% of CAP cases [21], the development

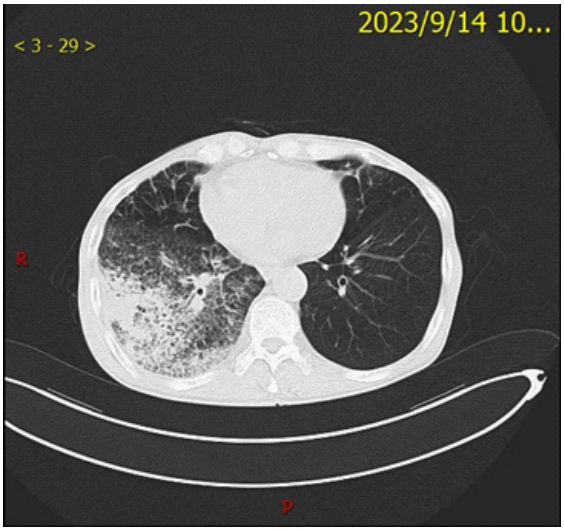

Figure 1: Chest CT on admission showed consolidation in the right lower lobe without evidence of air bronchogram.

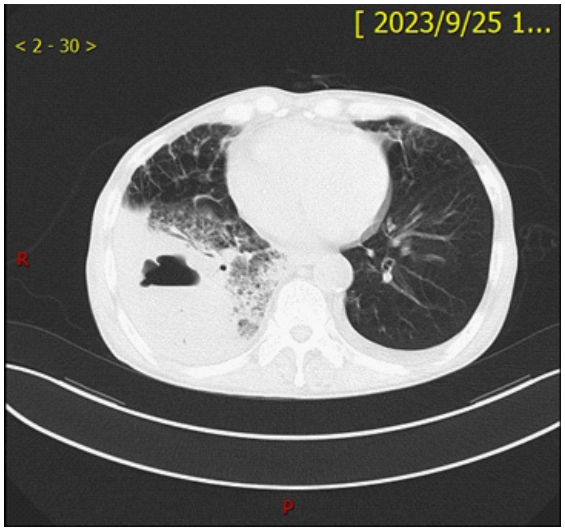

Figure 3: On day 11 of illness, chest CT demonstrated enlargement of the cavity with an air-fluid level.

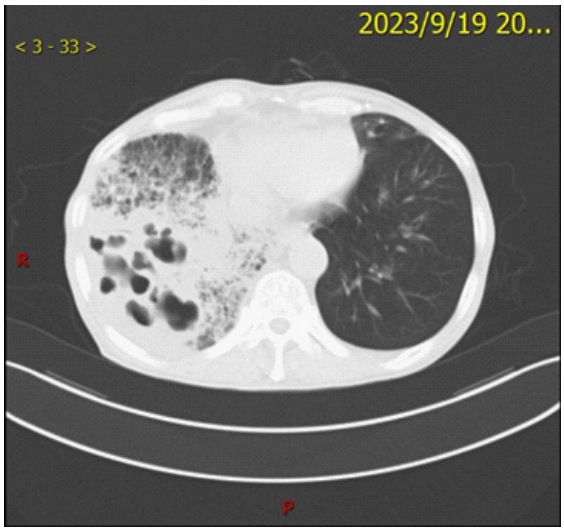

Figure 2: On day 5 of illness, chest CT revealed cavity formation within the consolidation in the right lower lobe, with a relatively smooth inner wall.

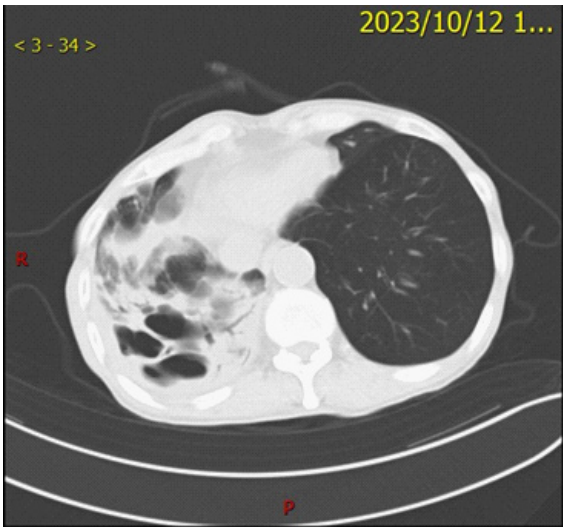

Figure 4: On day 28 of illness, chest CT showed marked improvement in both the consolidation and cavity of the right lower lobe.

of lung abscess is extremely uncommon, particularly in adults [22]. To date, most reported MP-associated lung abscesses have occurred in children [2,23,24], and our report may represent one of the first well-documented cases in an adult with underlying COPD [25,26].

This case highlights several important aspects of the interaction between host factors and MP pathogenicity. First, COPD significantly increases susceptibility to severe pulmonary infections due to structural airway remodeling, impaired mucociliary clearance, and frequent use of corticosteroids, which together weaken local immune defenses [26,27]. These factors likely facilitated the unusually aggressive course of MP infection in our patient, resulting in tissue necrosis and abscess formation. Second, although MP lacks classical virulence factors such as toxins, it induces intense host inflammatory responses [28,29]. Cytokines including TNF-α, IL-6, and IL-18 contribute to epithelial destruction and necrosis, which may explain the extensive parenchymal damage observed in this case [30]. Third, while bacterial coinfections can aggravate lung injury, repeated cultures of sputum and bronchoalveolar lavage in our patient remained negative, suggesting MP alone was sufficient to cause abscess formation.

Another noteworthy feature of this case is the diagnostic challenge. Conventional methods such as culture and serology are either insensitive or time-consuming, making early recognition of MP-related complications difficult [24,31]. In our patient, tNGS provided a rapid and definitive diagnosis, allowing targeted therapy to be initiated in a timely manner. This emphasizes the value of molecular diagnostics when dealing with atypical or unexplained pulmonary infections.

From a therapeutic standpoint, our patient responded favorably to macrolide therapy combined with drainage and supportive measures. Given MP's intrinsic resistance to β-lactams, empirical treatment with standard broad-spectrum antibiotics may be ineffective [31,32]. This case therefore underlines the importance of considering MP as a potential cause of lung abscess in adults with COPD when initial treatments fail, especially in the absence of conventional pathogens [33,34].

In summary, our report expands the spectrum of clinical manifestations of MP infection, demonstrating that it can rarely lead to lung abscess even in adults. The coexistence of COPD may predispose to this unusual presentation, and rapid molecular diagnosis is essential for guiding appropriate therapy. Clinicians should maintain vigilance for MP in atypical lung abscess cases, particularly in patients with chronic lung disease.

Conclusion

This case represents a rare instance of Mycoplasma pneumoniae-associated lung abscess in an adult with COPD. While MP is typically linked to mild or moderate atypical pneumonia, it can occasionally lead to severe parenchymal destruction, particularly in patients with underlying chronic lung disease. Our findings underscore three key points: (1) clinicians should consider MP as a potential etiology in refractory lung abscesses when routine cultures are negative; (2) rapid molecular diagnostics, such as tNGS, are invaluable for timely identification of uncommon pathogens; and (3) early targeted antimicrobial therapy can achieve favorable outcomes even in severe presentations. Increased clinical awareness of such rare complications may facilitate earlier recognition and improve management strategies in similar patients.

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