Computed tomographic findings of macrolide-resistant Mycobacterium massiliense pulmonary disease and changes after antibiotic treatment

Hyun Jung Yoon, MDab, Myung Jin Chung, MDa,*, Won-Jung Koh, MDc, Byung Woo Jhun, MDb, Seong Mi Moon, MDb

Abstract
The purpose of this study was to present the computed tomographic (CT) findings of lung abnormalities in macrolide-resistant Mycobacterium massiliense pulmonary disease and its changes in follow-up CT after antibiotic treatment. Chest CT scans of patients with macrolide-resistant M. massiliense pulmonary disease (n = 19) were retrospectively reviewed. Patients were treated with multidrug therapy, and sputum examinations were performed. Follow-up CT scans obtained during antibiotic treatment after detection of macrolide resistance were also reviewed, if available (n = 13). The CT scores at detection of macrolide resistance and at the last follow-up periods were also compared.

Of all patients with macrolide-resistant M. massiliense pulmonary disease, 2 (11%) patients achieved sputum culture conversion during the follow-up period. The most common CT findings of M. massiliense pulmonary disease at detection of macrolide resistance were bronchiectasis and broncholiths (n = 19, 100%), followed by consolidation (n = 16, 84%), cavities (n = 11, 58%), and nodules (n = 6, 32%). On the last follow-up CT, overall CT scores were increased in 8 (62%) of 13 patients, and total mean CT score was significantly increased (P = .021). For each CT pattern, the cavity showed the greatest increase in CT score (P = .027), followed by bronchiectasis (P = .038).

Common CT findings of macrolide-resistant M. massiliense pulmonary disease were similar to those of pulmonary disease caused by other species of nontuberculous mycobacteria at presentation. However, in macrolide-resistant M. massiliense pulmonary disease, serial CT scans showed deterioration with cavitory and bronchiectatic change in most patients despite multidrug antibiotic therapy.

Abbreviations: AFB = acid-fast bacillus, CT = computed tomographic, IQR = interquartile range, MAC = Mycobacterium avium complex, NTM = nontuberculous mycobacteria.

Keywords: computed tomography, drug resistance, macrolides, Mycobacterium massiliense, nontuberculous mycobacteria

1. Introduction
Nontuberculous mycobacteria (NTM), a ubiquitous mycobacteria that causes chronic human pulmonary infection, has been reported to be increasing worldwide.[1,2] Mycobacterium avium complex (MAC), Mycobacterium abscessus, and Mycobacterium kansasii are the most frequent causes of NTM pulmonary disease.[3,4] M. abscessus is a rapidly growing mycobacterium and the most common cause of rapidly growing mycobacterial pulmonary disease. Currently, M. abscessus can be divided into 3 subspecies: M. abscessus subspecies abscessus (hereafter referred to as M. abscessus), M. abscessus subspecies massiliense (hereafter referred to as M. massiliense), and M. abscessus subspecies bolletii (hereafter referred to as M. bolletii).[5,6] The most common subspecies is M. abscessus (45–65%), followed by M. massiliense (20–55%) and M. bolletii (1–18%).[7]

The response rates for macrolide-based antibiotic therapy are much higher among patients with M. massiliense pulmonary disease than for those with M. abscessus pulmonary disease.[8–15] However, acquired macrolide resistance can develop during macrolide-containing antibiotic treatment of M. massiliense pulmonary disease and is conferred by mutations in the drug-binding receptor rrl gene for 23S rRNA, at nucleotide positions 2058 and 2059.[16–19] Therefore, an awareness of macrolide-resistant M. massiliense pulmonary disease is very important for the diagnosis and management of this disease.[4]

Although a recent study reported clinical characteristics of macrolide-resistant M. massiliense pulmonary disease and the treatment outcomes of affected patients,[20] there has been no report regarding the computed tomographic (CT) imaging findings of macrolide-resistant M. massiliense pulmonary disease.
and changes in follow-up CTs of affected patients. One previous study presented CT findings of *M. massiliense* pulmonary disease, but all *M. massiliense* strains described were susceptible to macrolides.[21] Thus, the purpose of our study was to demonstrate CT findings of lung abnormalities at the time of diagnosis of macrolide-resistant *M. massiliense* pulmonary disease and its serial changes on follow-up CT after treatment with antibiotic therapy.

2. Materials and methods

This retrospective study was approved by the institutional review board of the Samsung Medical Center (IRB file No. 2018-08-001) and informed consent was waived for the use of patients’ medical data due to the retrospective nature of this study.

2.1. Patients and diagnoses

All patients diagnosed with macrolide-resistant *M. massiliense* pulmonary disease at Samsung Medical Center between September 2005 and October 2015 were screened and their medical records reviewed. A total of 19 patients with macrolide-resistant *M. massiliense* pulmonary disease for whom CT scans at the time of detection of macrolide resistance were included. The patients fulfilled the diagnostic criteria for NTM pulmonary disease according to the guidelines of the American Thoracic Society and Infectious Diseases Society of America.[11] All patients were administered antibiotic therapy after macrolide-resistance detection for the disease. Fifteen patients who were described in the recently published article by Choi et al.[20] were included in our study. Clinical data for the 15 patients was also included in a previously published article by our institution.[20]

Sputum smears and cultures of acid-fast bacillus (AFB) were regularly obtained during the follow-up period.[22] NTM species were identified by polymerase chain reaction (PCR)-restriction fragment length polymorphism analysis of the rpoB gene or reverse-blot hybridization of rpoB.[18,10] Drug susceptibility testing was performed at the Korean Institute of Tuberculosis, using the broth microdilution method.[23]

2.2. Treatment and evaluation of treatment outcomes

For the initiation phase of treatment of macrolide-susceptible *M. massiliense* pulmonary disease, patients were hospitalized for 2 or 4 weeks and received oral macrolide and/or fluoroquinolone, along with intravenous amikacin and cefoxitin (or imipenem). After discharge, the patients underwent a 2-drug oral regimen consisting of oral macrolide and/or fluoroquinolone for the continuation phase of treatment of *M. massiliense* pulmonary disease in our institution.[10]

For the treatment of macrolide-resistant *M. massiliense* pulmonary disease, a standardized treatment protocol was not established in our institution. Patients with mild symptoms when macrolide resistance was detected received oral antibiotics at the outpatient clinic. Patients with severe symptoms were hospitalized and received intravenous amikacin and cefoxitin for 2 to 4 weeks. For the oral antibiotics, treatment with a macrolide was continued for all patients and additional drugs (such as a fluoroquinolone, doxycycline, linezolid, clofazimine, or inhaled amikacin) were used which were guided by drug susceptibility results and patient tolerance.[4,20,24] Sputum culture conversion after the detection of macrolide-resistant *M. massiliense* pulmonary disease was assessed; conversion was defined as 3 consecutive negative cultures, with the time of conversion defined as the date of the first negative culture.[6,10]

2.3. CT acquisition

All CT examinations were performed using various helical CT scanners (Aquilion 64, Toshiba Medical System, Tokyo, Japan; LightSpeed 16, LightSpeed VCT and Discovery CT750 HD, GE Healthcare, Waukesha, WI; Brilliance-40, Philips Medical Systems, Cleveland, OH; SOMATOM Definition Flash, Siemens, Forchheim, Germany). CT scans were obtained from the lung apices to the level of the middle portion of both kidneys. All CT data were reconstructed using a high-spatial-frequency algorithm. The CT images were obtained using the following parameters: collimation, 1.25 or 0.625 mm; field of view, 36 cm; beam pitch, 1.35 or 1.375; gantry speed, 0.5 or 0.6 s/rotation; 120 kVp; 150–200 mA; and reconstruction interval, 12.5 mm. The image data were reformatted with a 2.5-mm section thickness for transverse images and a 2.0-mm section thickness for coronal images. The reconstructed images were then interfaced directly with a picture archiving and communication system (Centricity 2.0, GE Healthcare, Mt. Prospect, IL), which displayed all image data on 2 monitors (1536 × 2048 matrix, 8-bit viewable gray scale, and 60-ft-Lambert luminescence). Both mediastinal (width, 400 HU [Hounsfield unit]; level, 20 HU) and lung (width, 1500 HU; level, −700 HU) window images were viewed on these monitors.

2.4. CT interpretation

Two chest radiologists jointly assessed the CT images, and decisions on CT findings were reached by consensus (with 5 and 24 years of experience in chest CT interpretation, respectively). The presence of all parenchymal abnormalities in each lobe (6 lobes: right upper lobe, right middle lobe, right lower lobe, upper division of left upper lobe, lingular division of left upper lobe, and left lower lobe) was recorded. Each lung lobe was evaluated for the presence and extent of parenchymal abnormalities, including bronchiectasis, cellular bronchiolitis (small centrilobular nodules <10 mm in diameter and branching nodular structures [i.e., tree-in-bud sign]), nodules (10–30 mm in diameter), air-space consolidation (lobar [consolidation of 10–20 mm in diameter with a polygonal shape], segmental, or peribronchial), and cavities. The laterality (unilateral or bilateral) and location of lung lesions was also analyzed. A total of 114 lung lobes in 19 patients (6 lobes per patient) with macrolide-resistant *M. massiliense* pulmonary disease were evaluated for the presence of lung lesions. Thelast follow-up CT scans were available in 13 patients, which meant the 13 patients had >1 follow-up CT scan preceded by the CT scan obtained at the time of macrolide-resistance detection, and they were also assessed in the same manner. Additionally, the time interval between the CT scan date when macrolide-resistance was detected (time point A) and the last follow-up CT scan date (time point B) was recorded.

After the pattern and distribution of the parenchymal abnormalities seen at CT were analyzed, the diseases were classified into 3 forms: fibrocavitary form (previously called the upper lobe cavitary form), nodular bronchiectatic, and...
unclassifiable. The fibrocavitary form was defined as when a cavity (or cavities) was present in the upper lobes with findings of emphysematous change in the middle and lower lung zones with or without a volume decrease of the upper lobes and apical pleural thickening.[25,26] The nodular bronchiectatic form was defined as when bilateral bronchiectasis and cellular bronchiolitis were present mainly in the right middle lobe and lingular division of the left upper lobe, irrespective of the presence of cavities in both lungs. However, in this form, there was neither upper lobar volume loss nor emphysematous change in the remaining lungs.[25,26] When the disease did not belong to either the upper lobe cavitary or the nodular bronchiectatic form, it was deemed unclassifiable. In this form, multifocal lobular or segmental consolidation or consolidation along the bronchovascular bundles might be seen.

2.5. CT scoring

The CT scores in terms of the severity of lung involvement in macrolide-resistant M massiliense pulmonary disease (Table 1) were calculated by adopting the previously published scoring system proposed by Kim et al.[21] A total score of 30 was allocated for the overall extent of a lung lesion in each patient. Scores were given by considering the presence, severity, and extent of bronchiectasis, cellular bronchiolitis, cavities, nodules, and consolidation in both lungs. For cavities, the diameter, wall thickness, and extent were evaluated. The mean overall CT score for each pattern of parenchymal abnormality was defined as the sum of score of the 19 patients divided by the total number of patients. The available 13 patients’ last follow-up CT scans were also scored and recorded.

2.6. Statistical analysis

Data are presented as the median and interquartile range (IQR) for continuous variables and as the frequency and percentage for categorical variables. CT scores of total and each parenchymal abnormality between the 2 time points were compared within the 13 patients who had follow-up CT scans as pairwise comparisons using a Wilcoxon signed rank test. A P-value of <.05 was considered to indicate a significant difference. Data were analyzed using IBM SPSS Statistics for Windows (version 18.0; IBM, Armonk, NY).

3. Results

3.1. Clinical characteristics and treatment outcomes

Of the 19 patients with macrolide-resistant M massiliense pulmonary disease, 5 patients were men and 14 patients were women, and median age was 57 years (IQR: 53–67 years). For antibiotic therapy after macrolide-resistance detection for the disease, the median period of treatment was 28 months (IQR: 12–39 months). Negative sputum conversion and its maintenance for >12 months were accomplished in only 2 patients (11%); follow-up periods after the detection of macrolide resistance were 47 and 55 months, respectively). Surgical resection was performed for 2 patients during follow-up after the detection of macrolide resistance. Therefore, the 2 patients were excluded from CT score comparison analysis. Of the 2 patients who achieved negative sputum conversion, 1 patient was who had no change in the overall CT score at the last follow-up CT and the other one was who underwent surgical resection after the detection of macrolide resistance.

3.2. CT Findings at time of the detection of macrolide resistance

Of 19 patients, 10 (53%) patients had the nodular bronchiectatic form, 7 (37%) had the fibrocavitary form, and 2 (10%) had the unclassifiable form. The pattern of the parenchymal findings including the frequency, laterality, and location of the lung lesions are summarized in Table 2. The most common CT findings at time of the detection of macrolide resistance were bronchiectasis and bronchiolitis (n = 19, 100%) (Fig. 1), followed by consolidation (n = 16, 84%), cavities (n = 11, 58%; Fig. 2), and nodules (n = 6, 32%). Cellular bronchiolitis and bronchiectasis were bilateral in distribution in 89% of patients, and they involved more than two-thirds of all lung lobes.

The CT scores recorded by both observers are shown in Table 3. In all 19 patients of macrolide-resistant M massiliense pulmonary disease, bronchiectasis and cellular bronchiolitis has relatively higher scores (4.9 and 4.9, respectively) than those of other disease patterns.

3.3. Changes in the last CT findings after treatment

In comparing CT scores between the time point A and B in the 13 patients who had follow-up CT scans, 8 (62%) of 13 patients’ scores increased, 2 (15%) decreased, and 3 (23%) had no change in the overall CT score at the last follow-up CT (time point B). The total mean CT score was significantly increased at time point B (P = .021). In CT patterns, cavities showed the greatest increase in score (P = .027; Fig. 1), followed by bronchiectasis (P = .038; Fig. 2). Cellular bronchiolitis and consolidation showed a slight increase in score, but these increases were not statistically significant (P = .581 and .763, respectively). Nodules showed no change in score. The median time interval between the time points A and B was 24 months (IQR: 10–37 months).
4. Discussion

There have been several studies regarding CT imaging findings of NTM pulmonary diseases caused by *M abscessus* and *M massiliense* or MAC. Considerable overlap exists in the imaging findings among those diseases.\(^{[21,25,27,28]}\) Kim et al.\(^{[21]}\) demonstrated the predominant CT findings of macrolide-susceptible *M massiliense* to be bilateral bronchiectasis and cellular bronchiolitis or upper lobe cavities combined with consolidations. However, there has been no previously published report regarding the CT imaging findings of macrolide-resistant *M massiliense* pulmonary disease in detail.

In this study, we investigated CT imaging findings of 19 patients with macrolide-resistant *M massiliense* pulmonary disease as well as its final changes in comparable 13 patients upon follow-up. Similar to the above-mentioned Kim et al study,\(^{[21]}\) the most common CT findings at presentation in macrolide-resistant *M massiliense* pulmonary disease in our study were bilateral cellular bronchiolitis and bronchiectasis (n=19, 100%). Cavities were noted in 11 (58%) patients. Among the 19 patients, the nodular bronchiectatic form is more frequent than the fibrocavitary form; this result was also similar to the previous study by Kim et al.\(^{[21]}\) Ten (53%) patients had the nodular bronchiectatic form, 7 (37%) had the fibrocavitary form, and 2 (10%) had the unclassifiable form in our study. However, in comparison to the previous report, macrolide-resistant *M massiliense* pulmonary disease had a greater tendency to include cavities than macrolide-susceptible *M massiliense* lung disease (11 [58%] vs 45 [44%]), and to be the fibrocavitary form (7 [37%] vs 8 [24%]).\(^{[21]}\) From these comparisons, we could assume that CT findings of *M massiliense* at the time of detection of macrolide-resistance have a tendency to present cavitary change, because macrolide resistance could develop after long-term

![Figure 1](image-url)

**Figure 1.** Serial CT scans of *M massiliense* pulmonary disease at time points A and B in a 66-year-old man. (A) Scans obtained at time point A show bronchiectasis and bronchiolitis mainly in both upper lobes. (B) Scans obtained at time point B (38 months after time point A) show interval progression of bronchiectasis with wall thickening of dilated bronchi (severity) in both upper lobes. The number (extent) of involved lobes was also increased. Multifocal peribronchial consolidations were increased or newly appeared in both lower lung zones. Total (severity, extent, and mucus plugging) scores for bronchiectasis, cellular bronchiolitis, and consolidation were 5, 6, and 1, respectively, for time point A and 7, 6, and 2, respectively, for time point B. CT = computed tomographic.
antibiotic therapy which included macrolide in patients with *M massiliense* pulmonary disease.

*M abscessus* pulmonary disease has been shown to have unsatisfactory clinical and radiographic treatment success rates (25–42%; 8, 28). In contrast, *M massiliense* pulmonary disease reported high negative sputum conversion rates and radiographic improvement rates after antibiotic therapy[8,10,21,29]; this may be because *M abscessus* has inducible macrolide resistance, but inducible resistance is not found in *M massiliense*, which has a partially deleted, nonfunctional *erm* (41) gene.[16] However, once macrolide-resistant is detected, the expected course of disease changes dramatically. Choi et al[20] reported that the treatment outcomes of macrolide-resistant *M massiliense* pulmonary disease were very poor after multidrug antibiotic treatment. In the report, only one (7%) of 15 patients had a favorable outcome, and the 5-year mortality rate after the development of macrolide resistance was high (33%). Another previous report suggested that susceptibility to macrolide was the only significant independent predictor of a favorable microbiological response in *M abscessus* and *M massiliense* pulmonary disease[30,31]. Our study also showed low negative sputum conversion rate (11%).

In our study, the total mean CT score was significantly increased at final follow-up CT scans (P = .021) despite long-term antibiotic treatment, and cavities showed the greatest increase in mean score (P = .027), followed by bronchiectasis (P = .038). This implies that the phenotype of macrolide-resistance *M massiliense* disease changes.

![Figure 2](image)

Figure 2. Serial CT scans of *M massiliense* pulmonary disease at time points A and B in a 46-year-old woman. (A) Scans obtained at time point A show bronchiectasis and cavities (arrows) in the right upper lung zone. (B) Scans obtained at time point B (36 months after time point A) show interval progression of cavities (arrows). Total scores (diameter, wall thickness, and extent) for cavitory lesions were 4 (score of 1, 2, and 1, respectively) for time point A and 7 (scores of 3, 3, and 1, respectively) for time point B. CT = computed tomographic.

### Table 3

| Mean CT score | Bronchiectasis (9 points) | Bronchiolitis (6 points) | Cavity (9 points) | Nodules (3 points) | Consolidation (3 points) | Total |
|---------------|---------------------------|--------------------------|------------------|--------------------|-------------------------|-------|
| All 19 patients (time point A) | 4.9 | 4.9 | 2.9 | 0.3 | 1.3 | 14.6 |
| Comparison test (n = 13) | | | | | | |
| Time point A | 5.1 | 5.1 | 2.2 | 0.3 | 1.4 | 14.0 |
| Time point B | 5.7 | 5.2 | 3.5 | 0.3 | 1.5 | 16.2 |
| P-value | .038 | .581 | .027 | 1 | .763 | .021 |

Bold represents statistically significant, P < .05.
pulmonary disease showed deterioration in their CT findings, with gradual changes in cavities and bronchiectasis during antibiotic treatment. Because patients with macrolide-resistant M. massiliense pulmonary disease show a poor response to antibiotic therapy in the sputum and on imaging studies, accurate and timely detection of macrolide-resistance in M. massiliense pulmonary disease during treatment is necessary.

Author contributions

Conceptualization: Hyun Jung Yoon, Myung Jin Chung, Won-Jung Koh.
Data curation: Hyun Jung Yoon, Myung Jin Chung, Won-Jung Koh, Byung Woo Jhun, Seong Mi Moon.
Formal analysis: Hyun Jung Yoon, Myung Jin Chung, Byung Woo Jhun, Seong Mi Moon.
Funding acquisition: Won-Jung Koh.
Investigation: Hyun Jung Yoon, Won-Jung Koh.
Methodology: Hyun Jung Yoon, Myung Jin Chung, Won-Jung Koh, Byung Woo Jhun, Seong Mi Moon.
Supervision: Myung Jin Chung, Won-Jung Koh.
Writing – original draft: Hyun Jung Yoon.
Writing – review & editing: Myung Jin Chung, Won-Jung Koh, Byung Woo Jhun, Seong Mi Moon.

Myung Jin Chung orcid: 0000-0002-6271-3343.

References

[1] Prevots DR, Marras TK. Epidemiology of human pulmonary infection with nontuberculous mycobacteria: a review. Clin Chest Med 2013;36:13–34.
[2] Stout JE, Koh WJ, Yew WW. Update on pulmonary disease due to nontuberculous mycobacteria. Int J Infect Dis 2016;45:123–34.
[3] Griffith DE, Aksamit T, Brown-Elliott BA, et al. An official ATS/IDSA statement: diagnosis, treatment, and prevention of nontuberculous mycobacterial diseases. Am J Respir Crit Care Med 2007;175:367–416.
[4] Haworth CS, Banks J, Capstick T, et al. British Thoracic Society guidelines for the management of non-tuberculous mycobacterial pulmonary disease (NTM-PD). Thorax 2017;72:1–64.
[5] Griffith DE, Brown-Elliott BA, Berwell JI, et al. Mycobacterium abscessus. “Pleased to meet you, hope you guess my name…”. Ann Am Thorac Soc 2015;12:436–9.
[6] Tortoli E, Kohli TA, Brown-Elliott BA, et al. Emended description of Mycobacterium abscessus, Mycobacterium abscessus subsp. abscessus and Mycobacterium abscessus subsp. bolletii and designation of Mycobacterium abscessus subsp. massiliense comb. nov. Int J Syst Evol Microbiol 2016;66:4471–9.
[7] Koh WJ, Stout JE, Yew WW. Advances in the management of pulmonary disease due to Mycobacterium abscessus complex. Int J Tuberc Lung Dis 2014;18:1141–8.
[8] Koh WJ, Jean K, Lee NY, et al. Clinical significance of differentiation of Mycobacterium massiliense from Mycobacterium abscessus. Am J Respir Crit Care Med 2011;183:405–10.
[9] Roux AL, Catherinot E, Soumier N, et al. Comparing Mycobacterium massiliense and Mycobacterium abscessus lung infections in cystic fibrosis patients. J Cyst Fibros 2015;14:63–9.
[10] Koh WJ, Jeong BH, Jeon K, et al. Oral macrolide therapy following short-term combination antibiotic treatment of Mycobacterium massiliense lung disease. Chest 2016;150:1211–21.
[11] Park J, Cho J, Lee CH, et al. Progression and treatment outcomes of lung disease caused by Mycobacterium abscessus and Mycobacterium massiliense. Clin Infect Dis 2017;64:301–8.
[12] Koh WJ, Jeong BH, Kim SY, et al. Mycobacterial characteristics and treatment outcomes in Mycobacterium abscessus lung disease. Clin Infect Dis 2017;64:309–16.
[13] Diel R, Ringhausen F, Richter E, et al. Microbiological and clinical outcomes of treating non-Mycobacterium avium complex nontuberculous mycobacterial pulmonary disease: a systematic review and meta-analysis. Chest 2017;152:120–42.
Pasipanodya JG, Ogbonna D, Ferro BE, et al. Systematic review and meta-analyses of the effect of chemotherapy on pulmonary Mycobacterium abscessus outcomes and disease recurrence. Antimicrob Agents Chemother 2017;61:e01206–17.

Kwak N, Dalcolmo MP, Daley CL, et al. Mycobacterium abscessus pulmonary disease: individual patient data meta-analysis. Eur Respir J 2019;54:piu 1801991.

Bastian S, Veziris N, Roux AL, et al. Assessment of clarithromycin susceptibility in strains belonging to the Mycobacterium abscessus group by erm(41) and rrl sequencing. Antimicrob Agents Chemother 2011;55:775–81.

Maurer FP, Ruegger V, Ritter C, et al. Acquisition of clarithromycin resistance mutations in the 23S rRNA gene of Mycobacterium abscessus in the presence of inducible erm(41). J Antimicrob Chemother 2012;67:2606–11.

Shallom SJ, Moura NS, Olivier KN, et al. New real-time PCR assays for detection of inducible and acquired clarithromycin resistance in the Mycobacterium abscessus Group. J Clin Microbiol 2015;53:3430–7.

Mougari F, Amarsy R, Veziris N, et al. Standardized interpretation of antibiotic susceptibility testing and resistance genotyping for Mycobacterium abscessus with regard to subspecies and erm41 sequence. J Antimicrob Chemother 2016;71:2208–12.

Choi H, Kim SY, Lee H, et al. Clinical characteristics and treatment outcomes of patients with macrolide-resistant Mycobacterium massiliense lung disease. Antimicrob Agents Chemother 2017;61:e02189.

Kim HS, Lee KS, Koh WJ, et al. Serial CT findings of Mycobacterium massiliense pulmonary disease compared with Mycobacterium abscessus disease after treatment with antibiotic therapy. Radiology 2012;263:260–70.

Koh WJ, Kwon OJ, Jeon K, et al. Clinical significance of nontuberculous mycobacteria isolated from respiratory specimens in Korea. Chest 2006;129:341–8.

Woods GL, Brown-Elliott BA, Convile P, et al. CLSI Standards: Guidelines for Health Care Excellence. Susceptibility Testing of Mycobacteria, Nocardiae, and Other Aerobic Actinomycetes. Wayne (PA): Clinical and Laboratory Standards Institute.

Jhun BW, Yang B, Moon SM, et al. Amikacin inhalation as salvage therapy for refractory nontuberculous mycobacterial lung disease. Antimicrob Agents Chemother 2018;62:e00011.

Chung MJ, Lee KS, Koh WJ, et al. Thin-section CT findings of nontuberculous mycobacterial pulmonary diseases: comparison between Mycobacterium avium-intracellulare complex and Mycobacterium abscessus infection. J Korean Med Sci 2005;20:777–83.

Song JW, Koh WJ, Lee KS, et al. High-resolution CT findings of Mycobacterium avium-intracellulare complex pulmonary disease: correlation with pulmonary function test results. AJR Am J Roentgenol 2008;191:W160.

Han D, Lee KS, Koh WJ, et al. Radiographic and CT findings of nontuberculous mycobacterial pulmonary infection caused by Mycobacterium abscessus. AJR Am J Roentgenol 2003;181:513–7.

Fujuchi S, Matsumoto H, Yamazaki Y, et al. Analysis of chest CT in patients with Mycobacterium avium complex pulmonary disease. Respiration 2003;70:76–81.

Lyu J, Kim BJ, Kim BJ, et al. A shorter treatment duration may be sufficient for patients with Mycobacterium massiliense lung disease than with Mycobacterium abscessus lung disease. Respir Med 2014;108:1706–12.

Lyu J, Jang HJ, Song JW, et al. Outcomes in patients with Mycobacterium abscessus pulmonary disease treated with long-term injectable drugs. Respir Med 2011;105:781–7.

Jeon K, Kwon OJ, Lee NY, et al. Antibiotic treatment of Mycobacterium abscessus lung disease: a retrospective analysis of 65 patients. Am J Respir Crit Care Med 2009;180:896–902.

Sato M, Hiyama T, Kaito K, et al. Usefulness of F-18 FDG PET/CT in the assessment of disseminated Mycobacterium avium complex infection. Ann Nucl Med 2009;23:757–62.

Demura Y, Tsuchida T, Usada K, et al. Usefulness of 18F-fluorodeoxyglucose positron emission tomography for diagnosing disease activity and monitoring therapeutic response in patients with pulmonary mycobacterioses. Eur J Nucl Med Mol Imaging 2009;36:632–9.