


Bronchiectasis and Emphysema in a Non-Smoking Patient with Prior TB Infection - A Case Report

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ABSTRACTS

Background: Bronchiectasis is a chronic lung disease characterized by permanent bronchial dilation and is commonly associated with previous respiratory infections. Structural airway damage may persist even after the primary infection has resolved, leading to chronic respiratory symptoms and progressive lung impairment. Tuberculosis (TB) is recognized as one of the major causes of post-infectious bronchiectasis, particularly in regions with a high prevalence of TB. **Case:** A 66-year-old female with a long history of tuberculosis presented with yellow sputum for two months. Chest CT scan showed fibrotic lines in both lungs, signs of bronchiectasis in the form of tram lines, and pulmonary emphysema. Previous chest x-rays revealed peribronchial and parenchymal infiltrates with a flattened diaphragm and emphysematous lung appearance. **Discussion:** Bronchiectasis with emphysema or chronic obstructive pulmonary disease (COPD) often occurs in the elderly population. A history of TB is a major risk factor for bronchiectasis occurring concurrently with COPD. Chronic inflammation due to TB infection can trigger further parenchymal damage manifesting as bronchiectasis and emphysema. **Conclusion:** This case report highlights the importance of radiological imaging in evaluating chronic respiratory symptoms in patients with a previous history of TB.

Keywords: Bronchiectasis, case report, emphysema, tuberculosis.

ABSTRAK

Latar Belakang: Bronkiektasis merupakan penyakit paru kronis yang ditandai dengan dilatasi bronkus permanen dan sering berkaitan dengan infeksi saluran pernapasan sebelumnya. Kerusakan struktural pada jalan napas dapat menetap bahkan setelah infeksi primer telah sembuh, sehingga menimbulkan gejala respirasi kronis dan penurunan fungsi paru secara progresif. Tuberkulosis (TB) dikenal sebagai salah satu penyebab utama bronkiektasis pascainfeksi, terutama di wilayah dengan prevalensi TB yang tinggi. **Kasus:** Perempuan berusia 66 tahun dengan riwayat TB lama dengan keluhan batuk berdahak kuning selama 2 bulan. *CT scan* toraks menunjukkan garis fibrotik pada kedua paru, tanda bronkiektasis berupa *tram line*, dan emfisema paru. Rontgen toraks sebelumnya menunjukkan infiltrat peribronkial dan parenkimal dengan diafragma mendatar dan gambaran efisematoid paru. **Diskusi**: Bronkiektasis dengan emfisema atau penyakit paru obstruktif kronik (PPOK) sering pada usia lanjut. Riwayat TB merupakan faktor risiko utama bronkiektasis bersamaan dengan PPOK. Inflamasi kronis akibat infeksi TB dapat memicu kerusakan parenkim lebih lanjut yang memanifestasikan bronkiektasis dan emfisema. **Simpulan:** Studi kasus ini menyoroti pentingnya pencitraan radiologis pada pasien dengan riwayat TB. **Barlaam Bagus Purwaka, Petra Gusti Parikesit, Merari Panti Astuti. Bronkiektasis dan Emfisema pada Pasien Tidak Pernah Merokok dengan Riwayat Infeksi TB - Laporan Kasus.**

Kata Kunci: Bronkiektasis, laporan kasus, emfisema, tuberkulosis.

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INTRODUCTION

Bronchiectasis is a chronic lung disease characterized by permanent dilatation of the bronchi. Clinically, it presents with cough syndrome, sputum production and recurrent exacerbations.[1] The most common cause of bronchiectasis is a previous respiratory infection. Respiratory infections can weaken the function of the mucociliary transport mechanism resulting in mucus build-up in the bronchi.[2]

Bronchiectasis was first described by Laennec in 1819 as a suppurative lung disease with a heterogeneous phenotype.[2] The exact prevalence of bronchiectasis is difficult to determine; in Western countries it is estimated to be 1.3% population.[3] In the US and Europe, prevalence increases with age (> 65 years) and is more common in women. In the US, the average annual prevalence was approximately 700 per 100,000 people among Medicare beneficiaries between 2012 and 2014.[4] The prevalence of bronchiectasis is higher in women and highest in Asian populations.[3] A prospective Korean study revealed that approximately one-fifth of all

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bronchiectasis patients had a history of TB. Patients with post-TB bronchiectasis are known to have severe radiological grade and worse lung function than other causes of bronchiectasis.[5]

CASE

A 66-year-old woman with persistent cough in the past 4 days accompanied by fever. The patient has a history of complete TB therapy 22 years ago. She also has a history of allergy to ciprofloxacin, levofloxacin, and cotrimoxazol. On physical examination, saturation was 89% and auscultatory lung sounds were vesicular in both lungs. Blood pressure 100/70 mmHg, pulse rate 79 x/min, and temperature 37.8° C. Body weight 36.5 kg. Chest x-ray showed coarse bronchovascular scars, air bronchogram (+), with right-sided peribronchial and parenchymal infiltrates more prominent with a flattened diaphragm and pulmonary emphisematoid features; COPD with exacerbation was suspected **(Figure 1)**. Sputum examination resulted in *Candida sp* and *Gram-positive cocci*. Sputum *bakteri tahan asam* (BTA) examination was negative.

Two months later, the patient came to the pulmonary outpatient clinic for control. She complaints of coughing yellow phlegm with a thick and sometimes watery consistency accompanied by a runny nose since the last 2 months. Fever and shortness of breath were denied. Physical examination revealed vesicular sounds in both lungs. Vital signs examination showed blood pressure 100/60 mmHg, pulse frequency 78 x/min, temperature 36.6°C, and saturation 94%. Thoracic CT scan revealed fibrotic lines in both lungs with signs of bronchiectasis in the form of tram lines and pulmonary emphysema **(Figure 2).**

The clinical picture leads to bronchiectasis accompanied by pulmonary emphysema. At the last clinic visit, the patient was given lansoprazole 30 mg, combination inhaler containing tiotropium bromide and olodaterol 2.5 mcg each, and codeine 10 mg.

DISCUSSION

In this study, we present the emphysematous bronchitis in the setting of post-tuberculosis bronchiectasis of a 66-year-old non-smoking woman treated for TB 22 years ago, emphasizing the distinctive interaction between chronic inflammatory lung injury and structural airway remodelling. She had a productive cough with yellow sputum for two months. The chest x-ray revealed bronchovascular scarring, air bronchogram and parenchymal infiltrates, which is consistent with emphysema. In the absence of smoking and other risk factor, post-TB chronic sequelae were identified as the primary culprit. Although post-TB bronchiectasis is extensively reported, its simultaneous occurrence with emphysema in non-smoker female is relatively rare clinical manifestation,[6] highlighting the necessity for increased awareness and additional study on post-TB chronic pulmonary sequelae.

The pulmonary mechanism in this patient explaining the associated appearance of bronchiectasis and emphysema could potentially be elucidated through the sequelae of chronic post-TB inflammation. Following TB infection, neutrophils, lymphocytes and macrophages continue to exert inflammatory responses even after receiving microbiological cure following TB infection. These immune inflammatory cells secrete destructive mediators, include lysosome enzymes, antimicrobial peptides and undergo reacting oxygen species associated from Mycobacterium tuberculosis phagocytosis, contributing extracellular matrix destruction, prevention of the inflammation resolution finally causing fibrotic as well as airway remodelling.[7] Structural airway damage due to bronchial stenosis is further aggravated by extrinsic pressure of enlarged peribronchial lymph nodes and the direct address of endobronchial TB.[8] As the integrity of the bronchial wall deteriorates, mucociliary clearance is compromised and more mucus collects, providing a comfort place for bacterial infection. Impaired mucociliary clearance, which facilitates chronic infection and progressive airway inflammation and destruction forms a vicious cycle is the pathophysiology of bronchiectasis.[9] These mechanisms account for how a solitary post-infectious inflammatory insult from TB can be correlated with structural lung pathologies.

COPD is characterized by a mix of clinical and

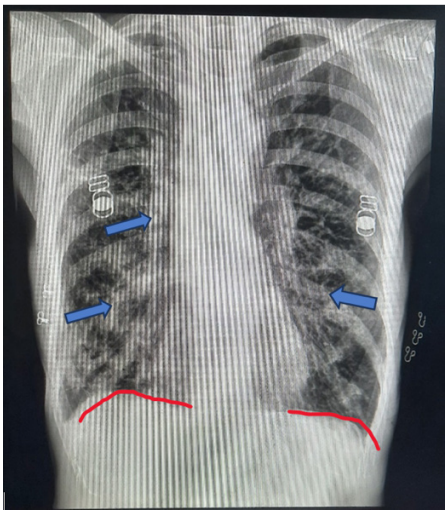

Photo documentation by the Radiology Department of Bethesda Hospital.

Figure 1. Gross bronchovascular pattern, air bronchogram (+), peribronchial and parenchymal infiltrates with the right side being more prominent (blue arrow) with a flattened diaphragm suggestive of pulmonary emphysematoid (red line).

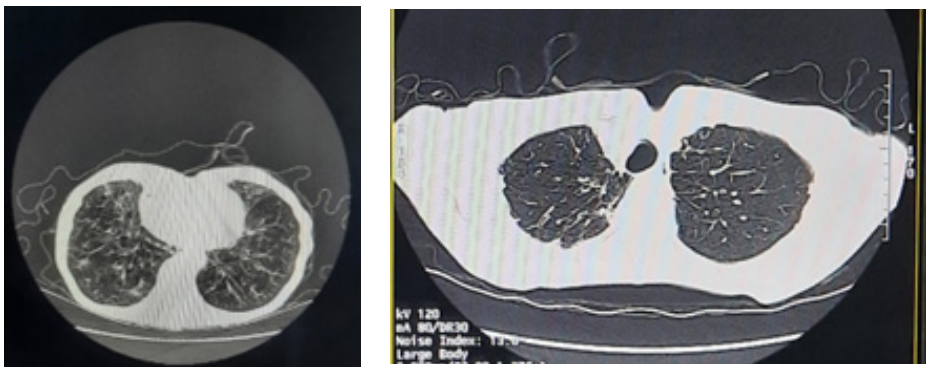

Photo documentation by the Radiology Department of Bethesda Hospital.

Figure 2. Axial CT scan of the lung window 10 May 2024. (a) Axial inferior lung (b) Axial apical lung. Inhomogeneous lung parenchymal structure with fibrotic streaks (yellow arrows) in both lungs accompanied by tram lines (blue arrows).


physiological assessments, with spirometry as the gold standard confirmatory test. COPD should be considered in any patient who presents with dyspnea, chronic cough and/or sputum production and/or a history of exposure to risk factors but spirometry showing a post-bronchodilator FEV1/FVC ratio of less than 0.7 is required for diagnosis according to GOLD 2025 guidelines,[10] while bronchiectasis is determined through morphological evaluation; yet, both conditions lead to analogous lung function impairments and nonspecific respiratory symptoms. The most common characteristic of bronchiectasis with COPD is in older patients with increased sputum production and an increased episodes of exacerbations.[11] The diagnosis of bronchiectasis in this patient was made on the basis of radiological findings as there were normal vesicular breath sounds by auscultation. This is in concordance with the literature, whereby the findings on chest radiography are usually inconclusive and images can be normal in bronchiectasis, thus, high-resolution CT is now considered the diagnostic gold standard.[12] In this case, lung function testing with spirometry was not performed and is a limitation in formally diagnosing COPD. However, the radiologic features of emphysema noted along with clinical presentation and past history of TB infection were suggestive of diagnosis, hence LABA/LAMA combination therapy was started.

Smoking has been established as a major risk factor in patients with COPD, and it has even been suggested that smoking is also a risk factor for tuberculosis.[13] However, this case was a never-smoker. About half COPD cases worldwide are attributed to non-tobacco-related risk factors, including air pollution, occupational exposures, poorly controlled asthma, environmental tobacco smoke, infectious diseases, and low socioeconomic status, with variations depending on geographical region.[14] Nevertheless, this case is a housewife seldom subjected to vehicular air pollution or refuse incineration, and she has no prior history of asthma. Another study estimates that 10% patients with emphysema have seldom or never smoked.[15] In comparison to smokers with COPD, never-smokers exhibit comparatively moderate chronic respiratory symptoms and minimal or no emphysema. In never-smoker patients with severe emphysema, it is essential to examine other potential etiologies, such as alpha-1-antitrypsin deficiency (AATD), a genetic condition marked by diminished AAT levels. The absence of AAT permits neutrophil elastase to deteriorate lung elastin, resulting in alveolar injury. This damage results in irreversible alveolar expansion, the defining characteristic of emphysema.[16] Our hospital cannot measure AAT levels.

A factor for developing bronchiectasis with emphysema is a history of tuberculosis infection. Evidence suggests that post-tuberculosis pulmonary inflammation may mask smoking-related lung function decline.[13] The pathophysiology of lung function decline after pulmonary tuberculosis treatment is speculative. One line of evidence suggests that lung damage is associated with bronchial stenosis as a consequence of extrinsic pressure from enlarged peribronchial lymph nodes as well as a consequence of endobronchial tuberculosis involvement.[17] In addition, there is an increase in metalloproteinase enzyme activity which may lead to long-term lung damage.[14] In another study, pulmonary TB was a risk factor for bronchiectasis co-occurring with COPD. Previous history of TB associated with bronchiectasis is generally more severe with varied clinical symptoms. The potential effect of progression from TB to bronchiectasis with COPD is mediated by chronic inflammation. This chronic inflammatory response may persist in patients with TB even after microbiological cure.[5]

The patient's most common clinical complaint was chronic and recurrent cough. Coughing is the most effective mechanism to clear airway secretions in bronchiectasis. This case developed bronchiectasis with signs of emphysema after completion of TB therapy. The pathogenesis of bronchiectasis was proposed by Cole with a picture of a pathogenic vicious circle. In pulmonary infections, there is a strong inflammatory response involving neutrophils, lymphocytes and macrophages accompanied by inflammatory products from microorganisms such as proteases, collagenases and free radicals. The inflammatory response weakens the bronchial wall as it loses its elastic elements. Inflammatory mediators will also impair mucociliary clearance as cilia movement is impaired and mucus clearance is inhibited. Bacterial infections are more likely to occur due to the accumulation of mucus in the bronchi. This vicious cycle will continue if there is no therapy to interrupt this process **(Figure 3)**.[18]

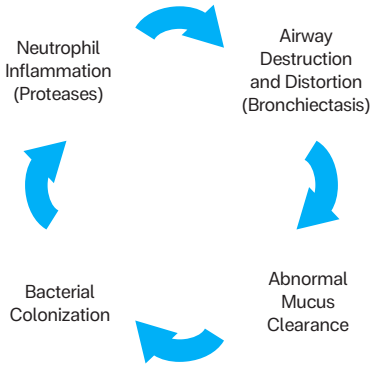

Figure 3. The vicious circle of bronchiectasis.[18]

In our case, emphysema was seen on both the patient's x-ray and CT. The x-ray showed peribronchial and parenchymal infiltrates and a flattened diaphragm with pulmonary emphysematoid formation. On CT of the patient's lung, the parenchymal structure was inhomogeneous with a centrilobular emphysema appearance. Emphysema with bronchiectasis had no difference in prevalence in patients with or without TB. However, patient with previous TB had a higher prevalence of extensive centrilobular emphysema than panlobular or bulbous types in other studies with HRCT (high resolution computed tomography) examination.[5] In addition, infiltrates in the lung indicate inflammation or infection. The existence of HRCT makes the observation and classification of emphysema easier and more accurate.[5] The infiltrate features in this study suggest that the infection occurred in the peribronchial and parenchymal lung.

Due to the high incidence of tuberculosis in Indonesia,[19] bronchiectasis caused by tuberculosis is common and needs to be carefully assessed.[20] The CT image in this case showed an inhomogeneous parenchymal structure with fibrotic lines in both lungs. The tram lines appeared as dilated bronchi.[1]

Tuberculosis can be a secondary complication in cases of post-tuberculous bronchiectasis due to endogenous reactivation caused by

malnutrition and lung damage or exogenous reactivation.[20] Although there was no information regarding the patient's height and BMI, the patient's weight was enough to indicate that the patient was not well nourished. It should be noted that one of the signs of tuberculosis is weight loss.[21] At the last visit, this patient was diagnosed with bronchiectasis accompanied by emphysema with a history of TB.

In this patient, sputum examination was performed for distinct two complementary reasons. First, her past history of prior TB infection, together with her underweight status, both are well known risk factors for reactivation of TB. One was sputum acid-fast bacilli (BTA) examination, which would be relevant for excluding active TB reinfection.[22] The second reason is that in patients with bronchiectasis it should very be recommended to obtain sputum culture both in stable state and exacerbation state before initiation of antibiotics, so that the causative organism can be determined to choose what kind of antibiotic should administered.[23] In this case, *Candida sp.* was identified on sputum culture. and Gram-positive cocci which enlightened the clinical management, emphasizing emerging isolates that underlined the significance of microbiological evaluation among post-TB bronchiectasis patients.

This case was prescribed with combination inhaler, which contains tiotropium bromide (LAMA) and olodaterol (LABA). Bronchodilator therapy is the recommended treatment of choice for chronic obstructive pulmonary disease (COPD), as endorsed by the Global Initiative for Chronic Obstructive Lung Disease (GOLD).[24] Initial treatment typically involves the use of short-acting beta-agonists (SABA) as a reliever for COPD patients. However, high-frequency use of SABA has been associated with an increased risk of exacerbations.[25] When LABA and LAMA are used in combination, their bronchodilatory effects are enhanced, leading to improved treatment outcomes.[24]

Lansoprazole (PPI) was prescribed for our patient primarily for gastric protection against medication-induced gastroduodenal irritation. In addition to gastric protection, PPIs may also provide added protection in the context of concurrently diagnosed COPD since gastroesophageal reflux disease (GERD) is known as an independent risk factor for recurrent exacerbation of COPD. The study conducted by Kikuchi (2018) found that treatment with lansoprazole was associated with a decrease in exacerbations of COPD which might be attributed to the some protective effect against the viral infections known to trigger exacerbation.[26]

CONCLUSION

Bronchiectasis with emphysema is considered to have a close relationship with a history of tuberculosis although further research needs to be done.

CONFLICT OF INTEREST

The authors declare no conflict of interest, financial or otherwise in this manuscript

AUTHOR CONTRIBUTION

All authors contributed to the study conception and design. The first draft was written by BBP and PGP. All authors commented on previous versions of the manuscript. All authors read and approved the final manuscript. The supervision was held by MPA.

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