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Case Report

# Computed tomography scan imaging in case of destroyed lung post-tuberculosis

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## A B S T R A C T

**Background:** Destroyed lung is a radiological term describing severe and irreversible damage to the lung parenchyma, most commonly resulting from TB. This condition poses significant clinical challenges and requires advanced imaging for timely diagnosis and appropriate management.

**Case Presentation:** This report presents three cases illustrating the clinical and radiological features of destroyed lung secondary to tuberculosis. The first case involves a 45-year-old woman with a prior history of TB who presented with facial swelling, dyspnea, and chronic cough; imaging revealed extensive cavitary lesions and destruction of the left lung. The second case describes a 25-year-old man with active TB who experienced worsening shortness of breath and night sweats; CT imaging showed a left fluidopneumothorax, multiple bullae, and bilateral cavitary lesions, indicating destroyed lung with secondary infection. The third case features a 46-year-old woman with chronic respiratory symptoms and right-sided pleural effusion; imaging demonstrated right lung destruction, bilateral fibrotic changes, and pulmonary artery hypertension.

**Conclusion:** Destroyed lung remains a significant post-tuberculosis complication requiring prompt diagnosis and multidisciplinary management. Radiologists play a critical role in identifying characteristic imaging findings and reviewing prior imaging to support clinicians in developing appropriate treatment strategies.

## INTRODUCTION

Destroyed lung refers to extensive and irreversible parenchymal damage characterized by reduced ventilation-perfusion ratio, commonly caused by chronic inflammatory conditions—most notably pulmonary tuberculosis (TB). Other contributing conditions include bronchiectasis, emphysema, aspergilloma, and recurrent pneumonia. Destroyed lung can lead to life-threatening complications such as massive hemoptysis, empyema, fungal superinfections, sepsis, and compensatory hyperinflation of the contralateral lung. Imaging modalities like chest X-ray and computed tomography (CT) remain critical in both diagnosis and monitoring, with pneumonectomy reserved for advanced or intractable cases.[1]

Tuberculosis remains a major global health concern caused by Mycobacterium tuberculosis, typically presenting with fever, night sweats, weight loss, and hemoptysis. Although effective treatment exists, management is complicated by the rise of multi-drug-resistant TB (MDR-TB), TB-HIV co-infection, and under-resourced healthcare systems.[2]

In 2020, the World Health Organization (WHO) reported that 43% of new TB cases occurred in the South-East Asia region. Indonesia ranked second globally, accounting for 8.5% of global TB cases, with an estimated 845,000 new cases in 2019 and over 92,000 TB-related deaths among HIV-negative individuals. The national strategy targets TB elimination by 2035 and eradication by 2050.[3]

Despite the pivotal role of microbiological testing, early radiological detection is essential, especially when bacterial loads are low or sputum samples are negative. CT imaging enables identification of cavitary lesions, bronchiectasis, and destruction patterns, offering critical guidance for treatment decisions. Although several studies have addressed the clinical features and management of destroyed lung, a significant gap remains in the literature regarding integrated diagnostic-therapeutic approaches, particularly in complex cases involving MDR-TB and HIV co-infection.[2]

This report aims to illustrate the diagnostic value of CT imaging in a post-tuberculosis destroyed lung case, emphasizing the role of integrated clinical decision-making. By presenting a real-world case scenario, we highlight the

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challenges and strategies in managing such complex cases and provide insights into optimizing diagnosis and care pathways. This case contributes to the growing body of evidence needed to refine clinical guidelines for destroyed lung management.

## CASE PRESENTATION

### *Case 1*

A 45-year-old woman was admitted with complaints of progressive swelling of the face, upper, and lower extremities for the past two weeks. She also reported shortness of breath on exertion that subsided with rest and a productive cough persisting for approximately one week before admission. Her medical history revealed a prior episode of TB in 2003, confirmed by microbiological testing and treated for six months, after which she was declared cured.

On physical examination, she appeared dyspneic and exhibited signs of generalized edema. Bronchoscopic evaluation demonstrated extraluminal compression of the left main bronchus and its branches, in addition to hypersecretion of mucus in both the left and right main bronchi. A conventional chest radiograph revealed irregular pleural thickening on the right, elevation of the right hilum, and opacities in the left hemithorax, leading to mediastinal and cardiac shift toward the left (Figure 1) left bronchus. Multiple cavities were seen in the right upper lung zone, along with fibroinfiltrates and consolidations, indicating post-TB sequelae with suspicion of a destroyed lung on the left side.

Further evaluation with CT of the thorax, both with and without contrast, revealed collapse of the left upper lobe with accompanying consolidation, shifting the trachea, heart, and mediastinal structures toward the left hemithorax. Bronchiectasis was evidenced by the presence of signet ring and tram track signs in the left bronchus, while the right upper lobe and left lower lobe also showed bronchial dilatation and surrounding consolidation (Figure 2). Additional findings included centrilobular emphysema in the anterior segment of the right lower lobe, fibrotic bands in the right middle and lower lobes, and multiple calcified nodules in both lungs. A CT angiography (CTA) identified dilation of the pulmonary trunk and right pulmonary artery, with a pulmonary artery-to-ascending aorta (PA/Ao) ratio greater than 1, suggesting the presence of pulmonary hypertension.

Based on clinical, radiological, and bronchoscopic findings, the patient was diagnosed with a destroyed lung on the left side, complicated by cylindrical atelectasis in the right upper and left lower lobes, centrilobular emphysema in the right lower lobe, post-inflammatory fibrosis, and possible secondary infection (Figure 3). Medical management focused on infection control, pulmonary support therapy including bronchodilators and mucolytics, and monitoring of pulmonary artery pressures. Surgical intervention was not immediately pursued.

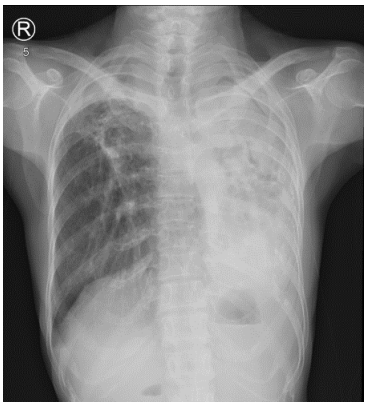

**Figure 1.** There is an opacity in the left hemithorax accompanied by retraction of the trachea, heart, and mediastinum towards the left side.

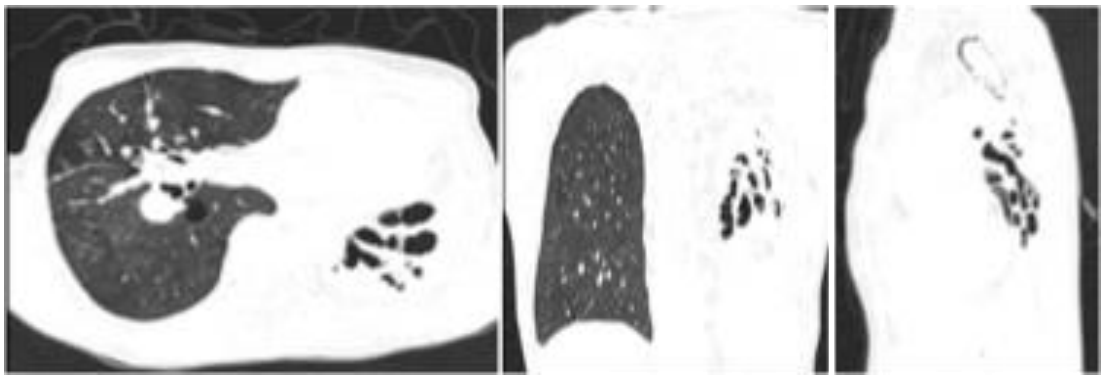

**Figure 2.** There is dilation of the left bronchus with a signet ring sign and tram track sign, accompanied by sur-rounding consolidation and collapse of the left upper lobe, which pulls the trachea, heart, and mediastinal structures towards the left side.

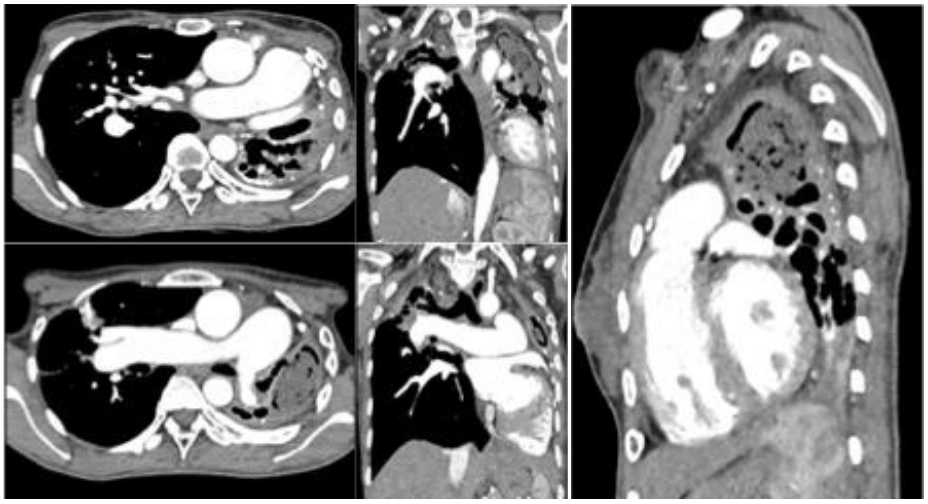

**Figure 3.** There is a noticeable narrowing of the diameter of the left pulmonary artery.

The patient's symptoms stabilized following conservative medical management, and she was discharged with a plan for regular outpatient follow-up to monitor respiratory function and detect early signs of deterioration or complications such as right heart strain. Long-term goals included symptom control, infection prevention, and evaluation for potential surgical intervention should pulmonary function decline further.

### *Case 2*

A 25-year-old man presented with worsening shortness of breath that had been occurring intermittently over the previous three months. His symptoms had acutely deteriorated in the five hours before admission and did not improve with rest or changes in position. He also reported a dry cough lasting approximately three months and night sweats for the past two months. There was no reported hemoptysis or weight loss. A bacteriological examination of sputum confirmed a diagnosis of active pulmonary tuberculosis.

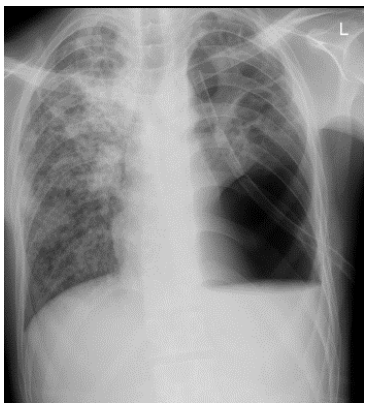

**Figure 4**. There is an avascular lucent lesion in the middle to lower zone of the left lung, with collapse of the left lower lobe, and a homogeneous pleural effusion forming an air-fluid level at the base of the left he-mithorax.

On physical examination, the patient exhibited signs of respiratory distress. A chest X-ray demonstrated an avascular lucent lesion in the middle to lower zone of the left lung, with collapse of the left lower lobe (Figure 4). There was also a homogeneous pleural effusion forming an air-fluid level at the base of the left hemithorax. The right lung showed multiple cavities and fibroinfiltrates extending from the upper to the lower zones. Additionally, multiple pulmonary nodules with surrounding consolidation and several thin-walled bullae were seen in the left lung. These findings were suggestive of active post-TB sequelae with superimposed secondary infection, multiple bullae, and fluidopneumothorax on the left side (Figure 6).

A subsequent CT scan of the thorax with and without contrast confirmed the presence of heterogeneous fluid density in the left pleural cavity (Figure 5), along with intrapleural air creating a well-defined air-fluid level. This combination was consistent with empyema and pneumothorax, causing compression and collapse of the left lung with a slight shift of the trachea and mediastinal structures toward the right. The scan also revealed cylindrical bronchiectasis and bullae in the left lung, as well as multiple cavities in the right upper lobe. These were accompanied by tree-in-bud opacities in the right upper, middle, and superior segments of the lower lobes, indicating active endobronchial spread of infection. There was also fluid collection in the right pleural cavity, suggestive of bilateral involvement.

The diagnosis was established as destroyed lung on the left side due to active tuberculosis, complicated by bullous lung disease, empyema, pneumothorax, and secondary bacterial infection. The patient was started on anti-tuberculosis pharmacotherapy and supportive medical treatment. A follow-up chest X-ray performed several days later showed persistent pneumothorax and no significant improvement in the number of bullae in the left lung or right upper lobe.

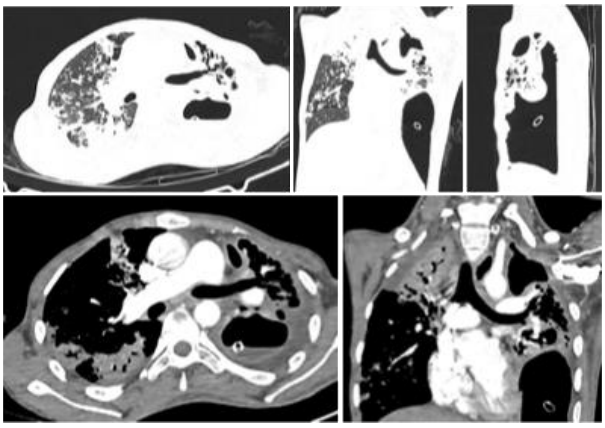

**Figure 5.** The CT scan shows heterogeneous fluid density in the left pleural cavity accompanied by air density within it, forming an air-fluid level, which causes compression collapse of the left lung lobe, with minimal devi-ation of the trachea, mediastinal structures, and heart towards the right side.

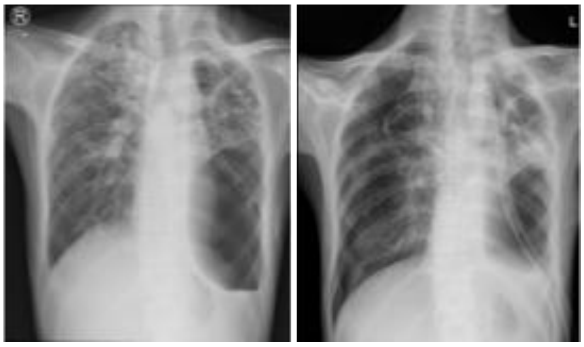

**Figure 6.** The left image shows multiple cavities and bullae in the left lung and right upper lobe, as well as the presence of left fluidopneumothorax. In the right image, taken post-bullectomy, multiple bullae are still visible in the right upper lobe, along with an effusion in the left lung that appears to be increasing.

The patient's condition remained relatively stable under medical management, and he was referred to a regional hospital in Sumba for continued anti-tuberculosis therapy, monitoring, and consideration of surgical options such as bullectomy or pleural drainage if his condition did not improve. The primary goals of treatment were to stabilize respiratory function, manage complications, and limit further lung destruction.

### *Case 3*

A 46-year-old woman presented with intermittent episodes of shortness of breath and palpitations, particularly during the night, for approximately one month prior to admission. She also complained of a productive cough with yellowish sputum but denied hemoptysis. Her medical history included a previous diagnosis of pulmonary tuberculosis, although the details of diagnosis and treatment were not

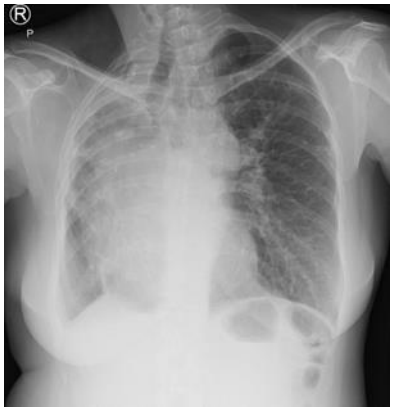

**Figure 7.** There are multiple cavities with consolidation and fibrocalcification, along with a homogeneous pleural effusion at the base of the right hemithorax, causing retraction of the trachea, heart, and mediastinal structures towards the right side.

clearly documented. On clinical examination, the patient was tachypneic with mild desaturation on room air. A chest radiograph revealed a homogeneous pleural effusion occupying the right hemithorax and causing widening of the pleural space (Figure 7). Additional findings included fibrocalcific consolidation in the right lung and in the upper zone of the left lung. These abnormalities were associated with a retraction of the trachea, heart, and mediastinum toward the right side. The radiographic findings were consistent with post-tuberculosis sequelae, including pleural thickening, cavitary disease, and suspected destroyed lung on the right side.

To further assess the extent of damage, a contrast-enhanced CT scan of the thorax and CT pulmonary angiography (CTPA) were performed. CT imaging demonstrated fibroinfiltrates and calcified nodules in the right upper and lower lobes (Figure 8). Ground-glass opacities accompanied by calcified nodules were identified in the left upper and lower lobes. Multiple cavities were noted in the lingular segment of the left lung, and cystic bronchiectasis was evident in the right lower lobe. Additionally, cylindrical and traction bronchiectasis were present in the right middle lobe. Irregular pleural thickening was observed in both hemithoraces, more pronounced in the superior aspect of the right pleura and the superolateral portion of the left pleura. No enhancement was observed post-contrast. Partial right lung collapse was also present, along with significant retraction of the mediastinal structures toward the right side.

CTPA revealed dilation of the pulmonary trunk and both the right and left pulmonary arteries, with the pulmonary trunk-to-ascending aorta (PA/Ao) ratio exceeding 1. This radiological finding, in conjunction with the pulmonary damage, was indicative of developing pulmonary artery hypertension secondary to extensive post-TB parenchymal destruction.

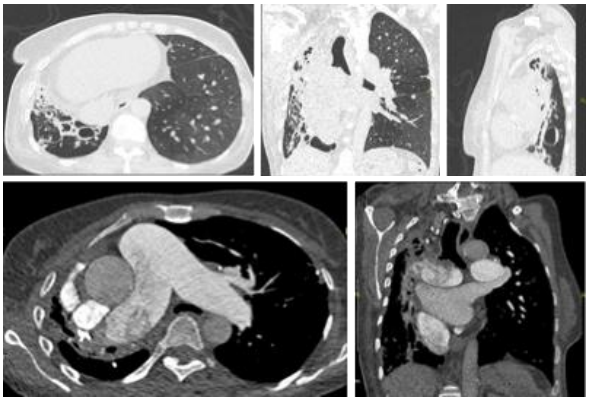

**Figure 8.** The CT examination reveals fibroinfiltrate and calcified nodules in the right upper to lower lobes, ground-glass opacity accompanied by calcified nodules in the left upper to lower lobes, along with dilation of the pulmonary trunk and right and left main pulmonary arteries

The patient was diagnosed with destroyed lung on the right side secondary to old pulmonary tuberculosis, complicated by pulmonary artery hypertension, cystic and traction bronchiectasis, and cavitary lesions in both lungs. Management was directed toward controlling respiratory symptoms, reducing pulmonary artery pressure, and preventing recurrent infections. Pharmacological therapy included bronchodilators, antibiotics, and supportive oxygen therapy as needed.

The patient's condition was stabilized, and she was scheduled for regular outpatient follow-up with periodic echocardiography and imaging to monitor the progression of pulmonary hypertension and assess the potential need for further interventions. The expected outcome was to improve symptom control, preserve remaining pulmonary function, and prevent decompensation of cardiopulmonary status.

## DISCUSSION

The term destroyed lung describes irreversible and extensive damage to pulmonary parenchyma, typically resulting from chronic inflammatory conditions, most commonly TB. This condition is radiologically characterized by a severely reduced ventilation-to-perfusion ratio due to cavitary destruction, fibrosis, bronchiectasis, and lung volume loss.[4] Although TB is the most frequent etiology, other contributing factors include bronchiectasis, emphysema, aspergilloma, and recurrent pneumonia.[5] Several risk factors have been identified, such as advanced age, prior TB treatment, malnutrition, bronchiectasis, and respiratory failure, highlighting the multifactorial nature of disease progression.[6]

Anatomically, TB-related lung damage is often unilateral, predominantly affecting the upper lobes. The left lung may be more susceptible due to the narrower, longer, and more angulated left main bronchus, making it prone to obstruction, collapse, and post-obstructive sequelae such as atelectasis and infection.[7,8] Clinical manifestations of destroyed lung are often nonspecific and include chronic cough, sputum production, dyspnea, fever, and hemoptysis. In advanced cases, severe complications such as respiratory failure, septicemia, empyema, or massive hemoptysis may develop.[9,10]

Chest radiographs and CT imaging are pivotal in diagnosing destroyed lung. Radiographic findings include unilateral opacification, shift of mediastinal structures, fibrotic bands, calcifications, and cavitary lesions. CT provides greater detail, revealing bronchiectasis (signet ring/tram track signs), atelectasis, emphysema, and pleural changes.[11,12] Differentiating destroyed lung from total lung collapse or chronic pleural disease is essential for appropriate management.[13]

### *Case 1: Destroyed Lung with Contralateral Compensatory Changes and Bronchiectasis*

In the first case, a 45-year-old woman presented with a history of TB diagnosed in 2003, treated over six months, and declared cured. Nearly two decades later, she developed progressive respiratory symptoms, including dyspnea and productive cough. Radiographic and bronchoscopic evaluations revealed a severely collapsed left upper lobe with mediastinal shift and evidence of extensive bronchial distortion. CT imaging confirmed classic features of destroyed lung: collapsed lobe, bronchiectasis characterized by tram track and signet ring signs, and surrounding parenchymal consolidation. These findings are consistent with long-standing post-TB sequelae. Additionally, the right lung showed compensatory changes including centrilobular emphysema in the right lower lobe and fibrosis in the middle and lower lobes, suggesting structural remodeling and hyperinflation to compensate for the destroyed left lung.

Multiple calcified granulomas were observed in both lungs, indicating prior granulomatous infection. The presence of bronchiectasis alongside centrilobular emphysema and fibrotic bands reflects chronic airway remodeling, which has been associated with prior cavitary TB and a history of delayed diagnosis or suboptimal treatment.[14–17] Pulmonary artery dilation with a PA/Ao ratio >1 observed in the CTA raised concerns for secondary pulmonary hypertension—an increasingly recognized consequence of destroyed lung, particularly in those with chronic hypoxemia. This case emphasizes the need for long-term pulmonary follow-up in TB survivors, even in patients deemed microbiologically cured, to detect late complications such as pulmonary artery hypertension, chronic infection, and respiratory insufficiency.[18–20]

### *Case 2: Destroyed Lung Complicated by Empyema, Pneumothorax, and Loculated Pleural Effusion*

The second case involved a 25-year-old man with active TB confirmed through bacteriological sputum testing. The patient presented with chronic symptoms of shortness of breath and dry cough, acutely worsening prior to admission. Chest imaging and CT scans revealed a complex presentation, including heterogeneous fluid collections in the left pleural cavity with intrapleural air, forming a distinct air-fluid level—hallmarks of empyema and pneumothorax. These complications resulted in compression collapse of the left lung and mild mediastinal shift. The tree-in-bud appearance in the contralateral lung and multiple cavities suggested active endobronchial spread of TB, a recognized radiological sign of bronchogenic dissemination.[21,22]

Empyema thoracis, or infected pleural fluid, typically occurs when TB spreads from the lung parenchyma into the pleural space. It is often associated with a delayed or inadequate response to anti-TB therapy.[23] The presence of bullae and multiple cavities increased the risk of spontaneous pneumothorax—a well-documented complication in cavitary TB.[24,25] Furthermore, the detection of loculated effusions in the right pleura, despite the disease being predominantly left-sided, indicates extensive pleural involvement. This complexity may necessitate surgical interventions such as tube thoracostomy or decortication, especially in cases unresponsive to antibiotics or standard anti-TB therapy.[26]

This case illustrates the importance of comprehensive imaging and clinical monitoring in TB patients with atypical or rapidly worsening respiratory symptoms. Early identification of empyema and pneumothorax can facilitate timely surgical consultation and avoid progression to respiratory failure.

### *Case 3: Destroyed Lung with Pulmonary Hypertension and Bilateral Fibrobronchiectatic Changes*

In the third case, a 46-year-old woman presented with exertional dyspnea, palpitations, and chronic cough. Imaging revealed extensive bilateral pulmonary damage consistent with destroyed lung syndrome, particularly on the right side. CT scans showed fibroinfiltrates and calcified granulomas in both lungs, cavitary lesions in the lingular segment, and widespread bronchiectasis, including cystic and traction forms. The radiological presence of calcifications and cavities indicates a chronic, possibly recurrent or incompletely treated, TB infection.[26]

Of particular concern was the dilatation of the pulmonary trunk and both main pulmonary arteries, yielding a PA/Ao ratio >1, which strongly suggests pulmonary hypertension. Pulmonary artery hypertension in TB is often a secondary phenomenon resulting from chronic hypoxic vasoconstriction, obliterative vasculopathy, or mechanical compression by fibrotic or calcified lymph nodes.[28–31] The shift of mediastinal structures toward the right and partial lung collapse reflect extensive volume loss and architectural distortion. Bronchiectasis, especially when associated with volume loss and fibrotic retraction, contributes to ventilation-perfusion mismatch, chronic hypoxia, and ultimately right heart strain.

Management of such a complex presentation requires a multidisciplinary approach, including pulmonologists, infectious disease specialists, and cardiologists. Medical management of pulmonary hypertension, along with aggressive infection control and airway clearance therapy, is essential. This case underlines the severe long-term consequences of pulmonary TB and the necessity of considering vascular complications in patients with chronic post-TB pulmonary damage.

## CONCLUSIONS AND RECOMMENDATION

Destroyed lung is a severe complication of chronic pulmonary inflammation, most commonly caused by tuberculosis. It results in irreversible structural damage, including bronchiectasis, fibrosis, and lung volume loss. Radiological imaging, particularly CT scans, is essential for accurate diagnosis and management planning.

To improve outcomes, early detection and long-term follow-up in post-TB patients are crucial. Healthcare systems must enhance access to advanced imaging and adopt multidisciplinary approaches. Future research should focus on innovative diagnostic tools and targeted therapies to prevent progression and optimize patient care.

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