SAR Journal of Medicine

Abbreviated Key Title: *SAR J Med*
Home page: https://sarpublication.com/journal/sarjm/home
DOI: https://doi.org/10.36346/sarjm.2025.v06i05.002

OPEN ACCESS

ISSN 2707-773X (P)
ISSN 2709-6920 (O)

Case Report

'Destroyed Lung' as Post Tuberculosis Lung Disease: Does it Preventable or Evolved as a Natural Trend of Advanced Pulmonary Tuberculosis?

Shital Patil [1*], Rajeshwari Pawar [2], Satyam Wande [2], Apurva Kelkar [2], Rudra Gaikwad [2], Gajanan Gondhali[3]

[1]Professor, Thoracic Oncology and Pulmonary Medicine, Venkatesh Chest Hospital and Critical Care Centre, Latur, Maharashtra, India
[2]Junior Resident, Internal Medicine, MIMSR Medical College, Latur, Maharashtra, India
[3]Professor, Internal Medicine, MIMSR Medical College, Latur, Maharashtra, India

***Corresponding Author:** Shital Patil
Professor, Thoracic Oncology and Pulmonary Medicine, Venkatesh Chest Hospital and Critical Care Centre, Latur, Maharashtra, India

Article History: | Received: 07.09.2025 | Accepted: 01.11.2025 | Published: 04.11.2025 |

Abstract: ***Background*:** Pulmonary tuberculosis (TB) continues to be a major global health problem, with post-tuberculosis lung disease (PTLD) emerging as an underrecognized sequela. Among its various manifestations, "destroyed lung" represents a progressive, debilitating outcome characterized by irreversible structural damage, recurrent infections, and significant impairment of quality of life. Despite successful completion of anti-tuberculosis therapy (ATT), patients often suffer long-term pulmonary morbidity. This case series highlights two patients with advanced PTLD presenting as destroyed lung, emphasizing the natural history, diagnostic challenges, and therapeutic outcomes. ***Case Presentation*:** The first case involves a 48-year-old male with a history of delayed TB diagnosis for three years, during which he was empirically treated for pneumonia, typhoid, and jaundice by various practitioners. Eventually, he was diagnosed with pulmonary TB and treated for eight months at a government center, with treatment deemed successful. However, after eight years of treatment completion, he presented to our center with recurrent respiratory symptoms. Bronchoscopy revealed a necrotic mucosal lumen with suppurative secretions in the left main bronchus extending into both upper and lower divisions. The bronchial anatomy was markedly distorted with dilated, ectatic bronchi. Bronchoalveolar lavage (BAL) cultures demonstrated colonization with *Pseudomonas aeruginosa* and *Klebsiella pneumoniae*. BAL GeneXpert MTB/RIF was negative, excluding active TB. He was managed with meropenem, amikacin, nebulized bronchodilators, and supportive care, showing marked clinical improvement after 14 days. The second case is a 51-year-old male with a delayed diagnosis of TB for two years. He too had initially been misdiagnosed and treated empirically for non-specific febrile illnesses. Eventually, he completed one year of ATT at a government center and was declared cured. Ten years later, he presented with persistent and recurrent pulmonary symptoms. Bronchoscopic evaluation revealed necrotic mucosa with purulent secretions in the right main bronchus, involving upper, middle, and lower divisions. The bronchial tree was distorted with extensive ectatic changes. BAL culture grew *Pseudomonas aeruginosa* and *Klebsiella pneumoniae*, while GeneXpert MTB/RIF was negative. He was treated with the same regimen as the first case and demonstrated a favorable outcome after two weeks of therapy. ***Discussion*:** Both patients highlight the long-term sequelae of delayed TB diagnosis and management. Even after microbiological cure, chronic structural lung damage predisposed them to persistent colonization with pathogenic organisms, recurrent infections, and functional impairment. These cases underline that destroyed lung syndrome is largely preventable with early recognition of TB, timely initiation of ATT, and structured follow-up care. Importantly, both cases reinforce that post-tuberculosis sequelae require comprehensive management beyond TB treatment completion, integrating bronchodilator therapy, infection control, and long-term pulmonary rehabilitation. ***Conclusion*:** Destroyed lung as a PTLD manifestation represents the culmination of delayed diagnosis and chronic lung injury rather than treatment failure or reactivation. Early detection, timely treatment, and structured long-term surveillance could prevent this disabling outcome. These cases emphasize the urgent need for public health strategies addressing PTLD to reduce the burden of preventable chronic respiratory morbidity.

Keywords: Pulmonary Tuberculosis, Radiological Stigma, ATT, Destroyed Lung, Sequel.

Citation: Shital Patil, Rajeshwari Pawar, Satyam Wande, Apurva Kelkar, Rudra Gaikwad, Gajanan Gondhali (2025). 'Destroyed Lung' As Post Tuberculosis Lung Disease: Does it Preventable or Evolved as a Natural Trend of Advanced Pulmonary Tuberculosis? *SAR J Med*, *6*(5), 64-72.

INTRODUCTION

Tuberculosis (TB) is a chronic infectious disease that severely affects the health of millions of people each year and is a major public health problem worldwide [1]. TB-related stigma has become a formidable challenge for TB prevention and control [2]. However, there is also a growing awareness of the need to address the stigma related to TB, a major social problem [3]. TB-related stigma has been identified as a major obstacle to patients seeking medical care and completing a full course of treatment [4].

Pulmonary tuberculosis can have diverse presentations ranging from cavitation, consolidation, tumorous lesions, coin lesions, lower lung filed tuberculosis and endobronchial and miliary nodules [6-14]. Similarly, non-tuberculous pathologies can present with abnormalities such as consolidations, nodules, cavitations mimicking tuberculosis [14-19]. Bronchoscopy is a very crucial interventional pulmonology technique in evaluating these cases [20-27]. High risk factors for tuberculosis would be advanced age, malnutrition, pregnancy, steroids exposure, diabetes mellitus and immunosuppression [6-28]. Tuberculosis in advanced stage may cause cardiac dysfunction and systemic effects which will have poor outcome if timely treatment not received [20-31]. Final outcome in delayed treatment initiation may lead to destroyed lung as post tuberculosis sequel and proportionate number of cases may have lung function abnormalities irrespective of radiological outcome [18-24]. Tuberculosis may be misdiagnosed due to confusing or overlapping clinical and radiological features in high burden setting like India. [25-28] In children extrapulmonary tuberculosis can present like tuberculous meningitis and with or without mediastinal adenopathy which is usually missed due to vague presentations like bronchitis and asthma and cases with failure to thrive [29-35]. Destroyed lung is now very well defined in published literature with defining radiological criteria, and we have evaluated our cases in line with published criteria.

CASE SUMMARY

Case 1

A 48-year-old male presented with recurrent cough, purulent sputum, and dyspnea, eight years after completing anti-tuberculosis treatment (ATT). His medical history revealed a delayed diagnosis of pulmonary tuberculosis for nearly three years, during which he was repeatedly treated empirically as pneumonia, typhoid, and jaundice by multiple practitioners. Eventually, he was diagnosed and received 8 months of directly observed therapy at a government facility and was declared treatment completed. Chest x-ray PA showing cavitation, bronchiectasis and pleuroparenchymal fibrosis with complete mediastinal shift to left side [Image 1].

On current evaluation, physical examination revealed coarse crepitations over the left hemithorax. Laboratory findings demonstrated leukocytosis (total leukocyte count 14,200/µL), raised CRP (48 mg/L), and microbiological evidence of infection. Chest radiography and CT thorax indicated unilateral lung destruction with bronchiectasis. Flexible bronchoscopy revealed a necrotic mucosal lumen with copious purulent secretions in the left main bronchus, extending to both upper and lower lobe divisions. The bronchial architecture appeared distorted with markedly dilated ectatic bronchi [Image 2].

Bronchoalveolar lavage (BAL) culture yielded *Pseudomonas aeruginosa* and *Klebsiella pneumoniae*, both susceptible to carbapenems and aminoglycosides. GeneXpert MTB/RIF was negative, ruling out active or recurrent TB. He was managed with intravenous meropenem (1 g every 8 hours) and amikacin (750 mg once daily), along with nebulized bronchodilators. Over 14 days of inpatient care, his symptoms resolved with marked clinical improvement. He was discharged on inhaled bronchodilator therapy and advised long-term follow-up for monitoring bronchiectasis-related complications.

Case Summary 2

A 51-year-old male, previously treated for pulmonary TB, presented with recurrent productive cough, purulent sputum, and breathlessness of one-month duration. He had a history of undiagnosed pulmonary TB for two years, initially treated empirically as pneumonia, typhoid, and jaundice before receiving 12 months of ATT under government supervision and declared cured. Chest x-ray PA showing cavitation, bronchiectasis and pleuroparenchymal fibrosis with complete mediastinal shift to right side [Image 3].

Ten years after completion of therapy, he developed recurrent symptoms and presented to our center. On examination, he had bilateral coarse crackles more prominent over the right lung. Laboratory investigations showed leukocytosis (TLC 13,800/µL), elevated CRP (52 mg/L), and purulent sputum positive for bacterial growth. Radiological evaluation confirmed right-sided destroyed lung changes with bronchiectasis.

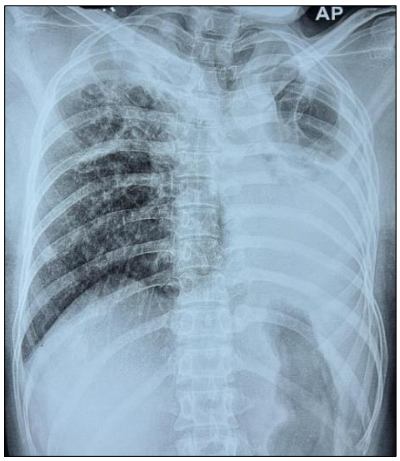

Image 1: Chest x-ray PA showing cavitation, bronchiectasis and pleuroparenchymal fibrosis with complete mediastinal shift to left side

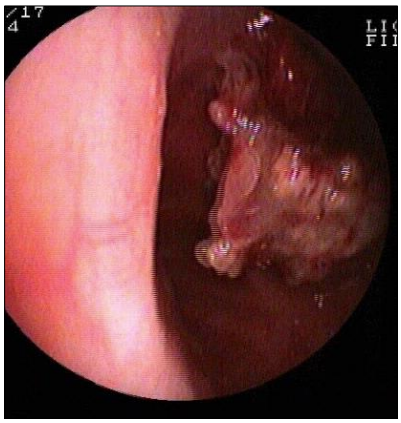

Image 2: Bronchoscopic image showing necrotic mucosal slough with broncholith

Bronchoscopy demonstrated necrotic mucosal lumen with copious suppurative secretions originating from the right main bronchus and extending to upper, middle, and lower bronchial divisions [Image 4]. The airway architecture was grossly distorted with dilated ectatic bronchi. BAL fluid cultures grew *Pseudomonas aeruginosa* and *Klebsiella pneumoniae*, sensitive to carbapenems and aminoglycosides. GeneXpert MTB/RIF was negative, excluding TB reactivation.

He was treated with intravenous meropenem and amikacin for 14 days, alongside nebulized bronchodilators. He showed significant symptomatic improvement, with resolution of sputum purulence and reduction in dyspnea. He was counseled about long-term sequelae of TB and advised regular follow-up to monitor and prevent further complications.

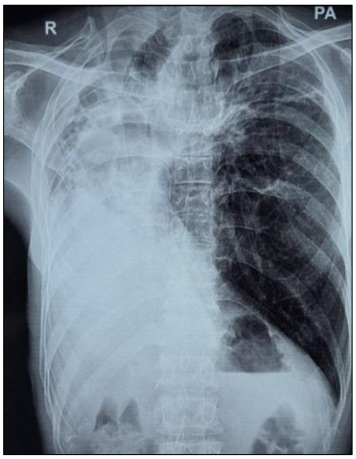

Image 3: Chest x-ray PA showing cavitation, bronchiectasis and pleuroparenchymal fibrosis with complete mediastinal shift to right side

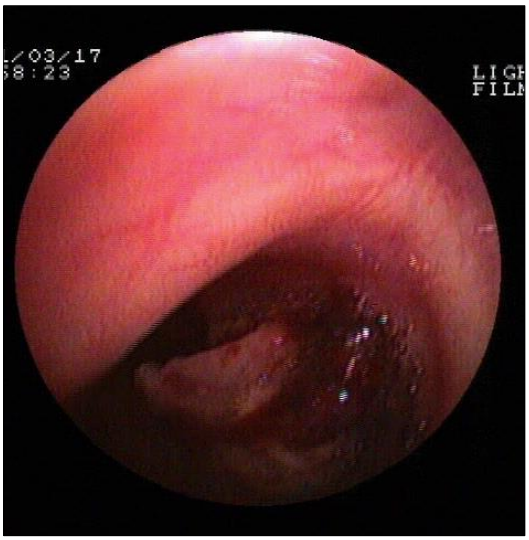

Image 4: Bronchoscopic image showing necrotic mucosal slough with hyperemic mucosa

Both the Patients Were Discharged to Home with Following Protocol:

- Dry powder inhaler of salmeterol plus fluticasone 250 microgram two times.
- Dry powder inhaler of Tiotropium 18 microgram one time.
- Tablet acebrophylline 200 mg bed time daily.
- Tablet N-acetyl cysteine 600 mg one time daily in glass of water.
- Postural drainage for sputum clearance and chest physiotherapy and breathing exercises with incentive spirometry.
- High protein diet and regular walk for maximum tolerance.
- Influenza vaccination annually.
- Pneumococcal vaccine every five years.
- High protein diet and pulmonary rehabilitation with breathing exercise.

DISCUSSION & LITERATURE OF REVIEW

A third of the world's population is infected with Mycobacterium tuberculosis (MTB), and over 9 million new cases of tuberculosis (TB) are reported annually [1]. However, up to half of TB survivors have some form of persistent pulmonary dysfunction despite microbiologic cure. Pulmonary dysfunction, ranging from minor abnormalities to severe breathlessness, can increase the risk of death from respiratory causes.

Definitions for Processes Contributing to Lung Remodelling During Pulmonary Tuberculosis (TB) and Pulmonary Impairment after TB- [36]

1. Pulmonary cavitation- Process by which normal pulmonary tissue is obliterated, becoming gas-filled spaces or cavities in the lung. This process initially involves caseous necrosis of lipid pneumonia lesions, producing caseous pneumonia. During caseation, alveolar cells and septa are destroyed along with neighbouring vessels and bronchi. Cavities form when these regions of caseous pneumonia liquefy, fragment and are released upon coughing.
2. Pulmonary fibrosis- Results from long-term lung tissue injury that is characterised by excessive extracellular matrix deposition in the lung. Replacement of normal lung parenchyma with collagenous tissue results in architectural changes in the lung, such as thickening and stiffening of the lung walls.
3. Bronchiectasis- Manifests as irreversible bronchial dilatation and thickening of the bronchial wall. Elastic and muscular components of the bronchial wall are destroyed in bronchiectasis. Bronchial dilatation associated with bronchiectasis in TB may be due to multiple factors, including traction from surrounding tissue fibrosis, caseous necrosis that makes its way into the bronchi, and elevated luminal pressure due to coughing. Bronchiectasis can also predispose to recurrent exacerbations of purulent sputum production and possibly bacterial pneumonia in subsequent years.
4. Pulmonary impairment after TB- A broad term we use in this review to refer to lung dysfunction that includes airflow obstruction, restrictive ventilatory defects and impaired gas exchange. Pulmonary impairment after TB is probably downstream of a wide variety of lung remodelling events, some of which are described above. Given the lung's considerable reserve, these structural changes may manifest as symptoms and pulmonary disability over a period of time.

Destroyed Lung [36]

The expression of "destroyed lung" is, now, accepted to designate the large destructions of the lung, secondary to pulmonary and essentially infectious diseases, the cure of which is obtained but with important sequelae. The main cause remains tuberculosis, cured by

chemotherapy. Some large pulmonary suppurations, treated by antibiotics, can lead to the same sequelae. These "destroyed lungs" can keep an asymptomatic form. But often, about ten years after the initial disease, they cause several troubles such as progressive dyspnea leading to irreversible respiratory insufficiency, repeated pulmonary infectious episodes and haemoptysis, the risk of which is increased by aspergillosis. The radiological aspect of these "destroyed lungs" is made of opacities with multiple cavities or with one unique large cavity.

Destroyed lung is defined as combination of pleural and parenchymal lung destruction with cavitation, bronchiectasis, loss of lung volume and mediastinal herniation to diseased side.

Components of Destroyed Lung- [36]

1. Pulmonary Cavitations
2. Cystic bronchiectasis
3. Loss of lung volume
4. Pleuroparenchymal fibrosis
5. Unilateral near complete lung parenchymal abnormalities with combinations of above findings
6. Contralateral lung parenchymal compensatory hyperinflation manifested as emphysema
7. Pull of contralateral lung and mediastinal structures to diseased side radiologically documented as mediastinal herniation.

Possible Clinical Outcomes in TB [37]

There are several possible clinical outcomes, depending primarily on the host immune defense mechanism.

1. In the presence of an excellent host defense, the patient may have no residual pulmonary findings and no clinical finding other than a positive tuberculin skin test.
2. In a complete failure of host defense mechanism, the patient may develop miliary tuberculosis.
3. When the host defense mechanism is fairly adequate, the patient may develop small foci of metastatic tubercle bacilli in the apex of the lung or perhaps in the kidney or bone. These lesions are walled off by the host defense mechanisms and may lie dormant for months or years; they may reactivate, possibly as a result of concurrent inhibition of the host cell-mediated immune defense (e.g. , by steroid therapy, debilitating illness, malignancy, or old age).
4. If the patient has a poor initial immune response, primary infection may develop directly into some manifestation of secondary tuberculosis such as apical disease, lymphadenitis, or bone or renal tuberculosis.

Radiological Classification of the Extent of Disease [38-42]

1. **Minimal:** Lesions those are slight to moderate density but which do not contain demonstrable cavitation. Involve a small part of one or both lungs, but total extent should not extend the volume of lung on one side that occupies the space above 2nd chondrosternal junction and spine of 4th or body of 5th thoracic vertebra
2. **Moderately Advanced:** Lesions may be present in one or both lung but the total extend should not exceed the following- disseminated lesions of slight to moderate density that may extend throughout the total volume of one lung or the equivalent in both lungs, dense and confluent lesions limited to one third the volume of one lung, total diameter of cavitation if present must be less than 4cm.
3. **Far Advanced:** Lesions more extensive than moderately advanced.

Walgreen timetable for active tuberculosis case findings in comparison with actual time trends is still valid and is comparable with histopathological examinations is a marker from primary infection to disease occurrence in the susceptible host [39].

Determining Factors for Tuberculosis-Related "Destroyed Lung" [43-50]

1. **Delayed Diagnosis and Prolonged Active Disease Duration** — longer untreated or suboptimally treated periods allow progressive parenchymal necrosis, cavitation and loss of lung architecture that culminate in a destroyed lung.
2. **Incomplete, Irregular or Prolonged/Incorrect Anti-Tuberculosis Therapy (Poor Adherence / Default / Inadequate Regimen)** — partial treatment and treatment interruptions permit ongoing tissue destruction and relapse/complications.
3. **Large Cavitary Disease and Extent of Radiological Involvement at Presentation** — extensive cavitation, multilobar involvement and pleural disease strongly predict eventual structural loss and poorer outcomes.
4. **Post-TB Bronchiectasis and Chronic Airway Remodelling** — bronchiectasis is a common PTLD phenotype and acts as the anatomic substrate for recurrent infection, progressive bronchial dilation and localized destruction.
5. **Recurrent or Persistent Secondary Bacterial (And Fungal) Infections** — colonization/infection with organisms such as *Pseudomonas* and *Klebsiella* perpetuates inflammation, suppuration and structural damage.
6. **High Mycobacterial Burden / Smear Positivity at Baseline** — heavier initial bacterial load and delayed bacteriological conversion associate with worse structural outcomes.
7. **Host Factors — Older Age, Comorbid Lung Disease (COPD, Prior Lung Injury),**

Malnutrition, Immunosuppression (including HIV) — these reduce pulmonary reserve, impair healing and raise risk of progressive destruction.

8. **Tobacco Smoking and Harmful Alcohol Use** — increase risk of severe disease, poorer treatment response, and long-term structural impairment.
9. **Delayed Access to Quality Healthcare / Misdiagnosis as Other Febrile Respiratory Illnesses** — health-system and patient delays that lead to late TB recognition are repeatedly associated with advanced disease at presentation.
10. **Biologic/Inflammatory Milieu and Biomarkers (Excessive Protease Activity, Persistent Inflammation)** — ongoing inflammatory and proteolytic processes in healed TB scars can continue to remodel and erode lung tissue. Emerging biomarker studies and mechanistic reviews support this pathway.

Preventable vs. Inevitable Sequelae Debate

A key question is whether destroyed lung is preventable or represents an inevitable natural progression of advanced TB. Evidence suggests that timely diagnosis, early initiation of effective ATT, and proper adherence significantly reduce the risk of severe structural lung damage. In both cases presented, there was a substantial diagnostic delay due to misdiagnosis as pneumonia, typhoid, or jaundice, a common scenario in primary care settings. These delays allowed TB to progress unchecked, leading to extensive parenchymal destruction. Thus, while destroyed lung may evolve as a natural outcome in advanced or extensive TB, it is largely preventable with robust public health measures, early case detection, and adequate treatment.

Microbiological Patterns in Destroyed Lung

Secondary bacterial colonization is a hallmark of destroyed lung. *Pseudomonas aeruginosa* and *Klebsiella pneumoniae* thrive in necrotic and ectatic bronchi, producing recurrent purulent infections. These organisms are associated with biofilm formation and multidrug resistance, complicating therapy. In both cases, leukocytosis, elevated CRP, and purulent sputum correlated with culture positivity for these organisms, indicating infective exacerbations. Literature supports that post-TB bronchiectasis frequently harbors Gram-negative bacilli, necessitating targeted antimicrobial therapy guided by culture sensitivity. Importantly, both our patients responded to intravenous carbapenems and aminoglycosides, consistent with the therapeutic approach in severe exacerbations.

Quality of Life and Comorbidity Burden

Destroyed lung has profound implications for patient quality of life. Recurrent infections, breathlessness, and reduced exercise tolerance contribute to chronic morbidity. Social and economic burdens are also significant, particularly for working-age patients like the two presented. The psychological impact of chronic symptoms after being declared "cured" of TB cannot be underestimated. These patients represent a growing population of "post-TB lung disease" (PTLD), now increasingly recognized as a distinct clinical entity requiring structured follow-up beyond microbiological cure.

CONCLUSION

Destroyed lung as a manifestation of post-tuberculosis lung disease represents a preventable yet disabling outcome of delayed diagnosis and advanced pulmonary involvement. Both cases in this series highlight that, despite microbiological cure with standard antituberculosis therapy, structural lung damage can persist and evolve into chronic bronchiectasis with recurrent bacterial colonization. This condition significantly impairs quality of life and increases healthcare burden. Early recognition of tuberculosis, timely initiation of appropriate therapy, and structured post-treatment follow-up are crucial to prevent such sequelae. Furthermore, comprehensive management of PTLD should extend beyond TB cure, incorporating bronchoscopy-based evaluation, targeted antimicrobial therapy, bronchodilators, and pulmonary rehabilitation to optimize long-term outcomes.

Key Learning Points from This Case Series Are

1. **Destroyed Lung is a Sequela of PTLD** – It results from chronic, irreversible lung damage after tuberculosis, not necessarily from treatment failure or reactivation.
2. **Delayed Diagnosis is a Key Risk Factor** – Prolonged misdiagnosis and empirical treatment for unrelated febrile illnesses can lead to extensive pulmonary destruction before TB is recognized.
3. **Microbiological Cure Does not Equal Structural Cure** – Even after successful completion of ATT, patients may develop long-term complications due to permanent anatomical distortion.
4. **Secondary Infections are Common** – Colonization with bacteria such as *Pseudomonas* and *Klebsiella* is frequent in destroyed lung and worsens morbidity.
5. **Bronchoscopy and BAL are Essential Tools** – They help in assessing bronchial anatomy, ruling out TB reactivation (GeneXpert), and identifying secondary bacterial colonization.
6. **Timely Antibiotic Therapy Improves Outcomes** – Prompt institution of broad-spectrum antibiotics, bronchodilators, and supportive care can control exacerbations and prevent deterioration.
7. **Prevention is Possible** – Early detection and initiation of ATT, combined with regular follow-up, can significantly reduce the risk of destroyed lung development.
8. **Quality of Life Remains Impaired** – Even with successful management of recurrent infections, patients often have long-term functional limitation, underscoring the importance of preventive strategies and pulmonary rehabilitation.

Several Key Lessons from the Presented Cases:

1. **Delayed Diagnosis is the Primary Driver –** Both patients had prolonged untreated TB due to misdiagnosis. This highlights the need for stronger TB screening programs at primary and secondary healthcare levels.
2. **Destroyed Lung is Largely Preventable –** Had TB been diagnosed earlier, extensive parenchymal destruction could have been avoided.
3. **Secondary Bacterial Colonization Dictates Morbidity –** Even after microbiological cure, colonization by *Pseudomonas* and *Klebsiella* led to recurrent infections and impaired quality of life.
4. **Targeted Therapy is Effective –** Both patients achieved significant improvement with culture-guided antibiotics, underscoring the importance of microbiological confirmation.
5. **TB Programs Must Evolve –** Beyond ensuring cure, there is a need to integrate long-term follow-up and pulmonary rehabilitation into TB control strategies.

REFERENCES

1. World Health Organization. Global tuberculosis report 2019. Geneva: WHO; 2019.
2. Datiko DG, Jerene D, Suarez P. Stigma matters in ending tuberculosis: Nationwide survey of stigma in Ethiopia. BMC Public Health. 2020;20(1):190.
3. Craig GM, Daftary A, Engel N, O'Driscoll S, Ioannaki A. Tuberculosis stigma as a social determinant of health: a systematic mapping review of research in low incidence countries. Int J Infect Dis. 2017; 56:90–100.
4. Murray EJ, Bond VA, Marais BJ, Godfrey-Faussett P, Ayles HM, Beyers N. High levels of vulnerability and anticipated stigma reduce the impetus for tuberculosis diagnosis in Cape Town, South Africa. Health Policy Plan. 2013;28(4):410–418.
5. Shital Patil, and Halkanche Gajanan, "Cavitary Lung Disease: Not Always due to Tuberculosis! Primary Lung Cancer with Smear Positive Pulmonary Tuberculosis- A Case Report." American Journal of Medical Case Reports, vol. 2, no. 8 (2014): 164-166.
6. Shital P, Anil J, Sanjay M, Mukund P (2014) Tuberculosis with Diabetes Mellitus: Clinical-Radiological Overlap and Delayed Sputum Conversion Needs Cautious Evaluation-Prospective Cohort Study in Tertiary Care Hospital, India. J Pulm Respir Med 4: 175
7. Patil Shital, and Laxman Kasture, 'Tennis Racket cavity' on Chest Radiograph: Strong Predictor of Active Pulmonary Tuberculosis! – A Case Report." American Journal of Medical Case Reports, vol. 2, no. 9 (2014): 167-169
8. Patil Shital, C R Choudhary, Laxman Kasture, and Ayachit Rujuta, "Endobronchial Tuberculosis Presenting as a Post-obstructive Pneumonia, Para-hilar Mass Lesion in Chest Radiograph and 'Tumorous' Endobronchial Lesion during Bronchoscopy: A Case Report." American Journal of Infectious Diseases and Microbiology, vol. 3, no. 5 (2015): 147-151.
9. Shital Patil, Jayashree Dahiphale, Vipul Raka, Sanika Narkar, Shubham Choudhari, Gajanan Gondhali (2023). "Coin Lesion" in Chest Radiograph Presenting as Round Pneumonia with Eccentric Cavitation in HRCT Thorax: Tuberculosis or Malignancy-A Real Puzzle! South Asian Res J Med Sci, 5(2): 33-40.
10. Shital Patil, Shubham Choudhari, Jayashree Dahiphale, Jayashree Dahiphale, Sanika Narkar, Vipul Raka, Gajanan Gondhali (2023). Cavitating Consolidation with Acute Febrile Respiratory Illness & Sister Cavities' Without Typical Constitutional Symptoms in Pulmonary Tuberculosis: A Rare But Possible! South Asian Res J Med Sci, 5(2): 41- 52.
11. Shital Patil, Mazhar Mirza, Gajanan Gondhali (2023). Satellite Nodules with Pericavitary Consolidation Presenting As Black Hole in the Starry Sky Pattern' in HRCT Thorax: A Strong Predictor of Active Pulmonary Tuberculosis!. South Asian Res J Med Sci, 5(4): 92-100.
12. Shital Patil, Mazhar Mirza, Swati Patil (2023). Fissural Pleural Effusion Presenting As Pseudotumor Lung, Phantom Tumour or Round Pneumonia' In Primary Pulmonary Tuberculosis: Bronchoscopy is Point of Care Test in Presence of Satellite Nodules in Chest Imaging! South Asian Res J App Med Sci, 5(4), 51-59.
13. Shital Patil, Jayashree Dahiphale, Vipul Raka, Sanika Narkar, Shubham Choudhari, Gajanan Gondhali (2023). Stepladder Lung Cavities" with "Starry Sky Pattern" in HRCT Thorax with Constitutional Symptoms: A Strong Predictor of Active Pulmonary Tuberculosis, SAR J Med, 4(2), 32-42.
14. Patil S, Narwade S, Mirza M. Bronchial wash Gene Xpert MTB/RIF in lower lung field tuberculosis: Sensitive, superior, and rapid in comparison with conventional diagnostic techniques. J Transl Intern Med 2017; 5: 174-85.
15. Patil S, Patil D, Khule S. Implant tuberculosis: A rare but possible cause in nonhealing surgical wound!. J Assoc Chest Physicians 2023;11:174-9.
16. Patil S, Ray S, Anjan A. Endobronchial tuberculosis with paratracheal adenopathy misdiagnosed and treated as bronchial asthma in a 12-year-old female child: Bronchoscopy is the point-of-care test! Res Rev Pediatr 2023;24:22-4.
17. Patil, S., and Gondhali, G. (2018). Short course of high dose steroids used for non-pulmonary indication like anaphylaxis caused flare up of tuberculosis & presenting as acute pulmonary tuberculosis with pleural effusion: a case report. European Journal of General Medicine, 15(1), pp. 37-42.
18. Shital Patil, Mazhar Mirza. Tuberculous Lymphadenitis of Hilar Lymph Nodes as a Cause of

Right Middle Lobe Syndrome: A Case Report. Respir Case Rep. 2018; 7(2): 90-96
19. Patil S, Patil R. "Fleeting pulmonary infiltrates in allergic bronchopulmonary aspergillosis" Misdiagnosed as tuberculosis. Int J Mycobacteriol 2018; 7:186-90.
20. Patil S, Jadhav A. Short course of high-dose steroids for anaphylaxis caused flare up of tuberculosis: A case report. J Transl Int Med 2019; 7: 39-42
21. Patil S, Gondhali G. Pulmonary Melioidosis Masquerading as Tuberculosis: A Case Report. Electron J Gen Med. 2021;18(5): em310.
22. Patil S, Gondhali G. COVID-19 pneumonia with pulmonary tuberculosis: Double trouble. Int J Mycobacteriol 2021; 10:206-9
23. Shital, P., & Mirza, M. (2018). Laryngeal & Lower lung field tuberculosis in pregnancy: A case report. Electronic Journal of General Medicine, 15(2), em06
24. Patil SV, Toshniwal S, Acharya A, Gondhali G. Cardiac dysfunction in active pulmonary tuberculosis: Mysterious facts of TB's pandora. Electron J Gen Med. 2023;20(2): em452.
25. Patil S, Gondhali G, Bhadake M. Disproportionate tachycardia and tachypnea in pulmonary tuberculosis: A marker of concurrent cardiac dysfunction. J Assoc Pulmonologist Tamilnadu 2022; 5:124-9.
26. Shital Vishnu Patil, Ganesh Narwade, Gajanan Gondhali Cardiac Dysfunction in Active Pulmonary Tuberculosis: Double Trouble!! European Respiratory Journal 2020 56: 1604
27. S. Patil, R. Patil, G. Gondhali; Cardiac Dysfunction in Active Pulmonary Tuberculosis: Underestimated, Missed Routinely and Have Impact on Clinical Outcome! Prospective Study of 600 Cases in Tertiary Care Setting in India Am J Respir Crit Care Med 2020;201: A5435
28. Shital Patil, Sanika Narkar, Vipul Raka, Jayashri Dahiphale, Shubham Choudhari, Gajanan Gondhali (2023). 'Destroyed lung' as Post Tuberculosis Sequel: A Preventable Stigma of 'disease of concern' of Millennium! Saudi J Med, 8(3): 112-119.
29. Patil S, Patil R, Jadhav A. Pulmonary functions' assessment in post-tuberculosis cases by spirometry: Obstructive pattern is predominant and needs cautious evaluation in all treated cases irrespective of symptoms. Int J Mycobacterial 2018; 7:128-33.
30. Patil SV, Toshniwal S, Gondhali G. Cavitating lung disease is not always due to tuberculosis! Wegener's granulomatosis with mycetoma with deep vein thrombosis lower limb: Case report with review of literature. Electron J Gen Med. 2023;20(1): em425.
31. Patil S, Gondhali G. Bronchus sign on HRCT thorax: presenting sign of Wegener granulomatosis with lung involvement – misdiagnosed as TB in presence of acino-nodular pattern on imaging. J Assoc Chest Physicians 2022; 10:105-11.
32. Patil S, Patil D. Wegener's granulomatosis mimicking like pulmonary tuberculosis and presenting as cavitating lung disease with mycetoma: A case report with review of literature. Muller J Med Sci Res 2022; 13:103-9.
33. Patil S, Gondhali G, Patil D. Chronic febrile respiratory illness with acinonodular consolidations as presenting feature of granulomatosis with polyangiitis: A case report with review of literature. J Assoc Pulmonologist Tamilnadu 2022; 5:116-20.
34. Shital Patil, Sonal Ray, Akhilesh Anjan (2023). Tuberculous Meningitis in Male Child and Cavitary Pulmonary Tuberculosis in Mother: Concurrent Familial Infective Disease as Evidence of Recent Transmission from Mother to Baby!. Saudi J Med, 8(5): 217-224.
35. Patil S, Ray S, Anjan A. Endobronchial tuberculosis with paratracheal adenopathy misdiagnosed and treated as bronchial asthma in a 12-year-old female child: Bronchoscopy is the point-of-care test! Res Rev Pediatr 2023; 24:22-4.
36. Shital Patil, Sanika Narkar, Vipul Raka, Jayashri Dahiphale, Shubham Choudhari, Gajanan Gondhali (2023). 'Destroyed lung' as Post Tuberculosis Sequel: A Preventable Stigma of 'disease of concern' of Millennium!. Saudi J Med, 8(3): 112-119.
37. Ravimohan S, Kornfeld H, Weissman D, Bisson GP. Tuberculosis and lung damage: from epidemiology to pathophysiology. Eur Respir Rev. 2018 Feb 28;27(147):170077.
38. Crofton and Douglas disease of Respiratory System 5th edition part 1, 518
39. Aditi Gatagat, Shital Patil, Sachin Tote (2023). Cavitary Lung Disease with Constitutional Symptoms Misinterpreted & Treated as a Bronchial Asthma and Enteric Fever for a Long Period: What are Timings from Infection to Cavitation in Tuberculosis? Revisits Wallgren's timetable !; SAR J Med Case Rep, 4(5), 51-62. DOI: 10.36346/sarjmcr.2023.v04i05.001
40. Patil S, Narkar S, Raka V, Dahiphale J, Choudhari S, Gondhali G. 'Destroyed lung' as post tuberculosis sequel: A preventable stigma of 'disease of concern' of millennium!. *Saudi J Med*. 2023;8(3):112-9
41. Abhishek Vairagade, Omprakash Bhangdiya, Shital Patil (2024). 'Destroyed Lung': A Marker of Sequel of 'Far Advanced' Radiological Type of Pulmonary Tuberculosis', *SAR J Med*, *5*(3), 69-79.
42. Liu L, Gao L, Zhang X, He J, Zhang Y, Wang Y, et al. Risk factors of tuberculosis-destroyed lung in patients with pulmonary tuberculosis and structural lung disease. Ther Clin Risk Manag. 2024;20:73-83.
43. Gai X, He L, Xu Y, Chen H, Wang J. Risk factors and biomarkers for post-tuberculosis lung disease: a prospective cohort study. BMJ Open. 2023;13(5):e065990.
44. Akalu TY, Gebremeskel GG, Tesfaye YG. Risk factors associated with post-tuberculosis sequelae: a

systematic review. EClinicalMedicine. 2024;69:102251.
45. Ruan H, Li Y, Wang Y, Chen Z, Zhang J, Zhou H. Long-term follow-up of patients with tuberculosis-destroyed lung: clinical features and outcomes. BMC Pulm Med. 2022;22:154.
46. Kim WY, Jung JY, Park MS, Kim YS, Kim SK, Chang J, et al. Predicting mortality in patients with tuberculous destroyed lung: a retrospective cohort study. Respir Med. 2018;135:86-91.
47. Perumal R, Abdool-Gaffar MS, Guler R, Andries A, Moodley S. The hidden epidemic of post-tuberculosis bronchiectasis. ERJ Open Res. 2023;9(3):00123-2023.
48. Ivanova O, Vassall A, Migliori GB, D'Ambrosio L, Centis R, Jaramillo E, et al. Post-tuberculosis lung impairment: a systematic review and meta-analysis. Eur Respir Rev. 2023;32(168):220180.
49. Malefane L, Ndlovu H, Oni T, Chisale M, Chuturgoon AA. Post-tuberculosis lung disease and inflammatory role players: mechanistic perspectives. Am J Respir Cell Mol Biol. 2024;70(2):123-35.
50. Faruqi AA, Kizilbash QA, Shaikh A, Javed R, Khan O. A case report of destroyed lung syndrome. Cureus. 2023;15(6):e39754.