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# POST TUBERCULOSIS LUNG DISEASE IN AN ELDERLY PATIENT WITH PSEUDOMONAS INFECTION AND SUBCLINICAL HYPERTHYROIDISM: A CASE REPORT

**Dr. Palakh Lukkad** Third Year Resident, Department of Geriatrics, Dr DY Patil Medical College, Navi Mumbai

**Dr Vaishali Thakare*** Professor , Department of Pharmacology, Dr DY Patil Medical College, Navi Mumbai *Corresponding Author

**Dr Dinesh Gupta** HOD & Prof. , Department of Geriatrics, Dr DY Patil Medical College, Navi Mumbai

**Dr Masum Reza** Second Year Resident, Department of Pharmacology, Dr DY Patil Medical College, Navi Mumbai

**ABSTRACT**

**Background:** Post-tuberculosis lung disease (PTLD) is an underrecognized but increasingly prevalent sequela in TB-endemic regions like South Asia. It encompasses structural pulmonary damage and systemic dysregulation, often complicated by opportunistic infections and age-related vulnerabilities. **Case Presentation:** We report the case of a 64-year-old Indian female with prior microbiologically cured pulmonary tuberculosis, who presented with mucopurulent cough, orthopnea, and hemoptysis. Imaging revealed fibrotic bronchiectasis with tree-in-bud opacities, and sputum culture isolated Pseudomonas aeruginosa sensitive to meropenem. Concurrently, thyroid function tests showed suppressed TSH (0.007 mIU/L) with preserved FT4, and ultrasonography revealed a TIRADS 4 thyroid nodule. The patient also exhibited early frailty, diastolic dysfunction, and poorly controlled diabetes. **Management:** She was managed with IV meropenem, nebulized tobramycin, bronchodilators, and supportive care. Endocrine referral was advised for further evaluation of subclinical hyperthyroidism. Functional rehabilitation and discharge planning were coordinated via a multidisciplinary team. **Conclusion:** This case illustrates the complex interplay between PTLD, geriatric pharmacotherapy, endocrine dysfunction, and microbial resistance. It reinforces the need for structured post-TB surveillance and geriatric-integrated care models. The case contributes to evolving global understanding of PTLD as a multisystemic entity, particularly in aging diabetic populations in resource-constrained settings.

## Introduction

Tuberculosis (TB) survivors frequently suffer long-term respiratory sequelae despite microbiological cure. Chronic post-TB pulmonary impairments – collectively termed *post-tuberculosis lung disease* (PTLD) – are increasingly recognized as a major contributor to global TB-related morbidity. By some estimates, over half of cured TB patients exhibit persistent respiratory abnormalities. With an estimated 155 million TB survivors alive worldwide, the prevalence of PTLD may exceed 90 million cases, reflecting a "hidden" epidemic of chronic lung disease spanning high-burden countries (e.g. India, China) and low-burden settings alike. PTLD encompasses any chronic respiratory abnormality attributable at least in part to previous TB infection. Common manifestations include residual pulmonary fibrosis, bronchiectasis, airflow obstruction, and functional deficits such as reduced lung capacity. Notably, *bronchiectasis* is frequently observed as a sequela of pulmonary TB – for instance, in India, TB is a leading cause of bronchiectasis, often with unilateral and cicatricial (varicose) patterns on imaging. Compared to non-TB bronchiectasis, post-TB cases tend to have more severe complications like frequent infective exacerbations and hemoptysis.

Epidemiologically, older adults bear a substantial share of the PTLD burden. Advancing age has been linked to more severe post-TB lung damage and higher disability-adjusted life year (DALY) losses. In China, for example, the TB and post-TB disease burden rises sharply with increasing age, and in 2019 an estimated 2.86 million DALYs were attributable to post-TB sequelae (the 6th highest worldwide). Older TB survivors often have coexisting chronic diseases that complicate their care, including diabetes mellitus (DM), cardiovascular disease, and other age-related conditions. Multimorbidity not only predisposes patients to more severe infections (e.g. DM increases risk of TB and pneumonia) but also challenges clinicians to balance complex pharmacotherapy and potential drug interactions in the geriatric population.

We report a case of a 64-year-old Indian woman, a TB survivor with residual lung disease, who presented with an acute *Pseudomonas aeruginosa* pulmonary infection on a background of PTLD. Her case is further complicated by subclinical hyperthyroidism due to nodular thyroid disease, type 2 DM, and diastolic cardiac dysfunction – illustrating the multifaceted management issues in an elderly patient with PTLD and multiple comorbidities. We highlight the pharmacologic management strategies employed and discuss geriatric considerations in care. We also review the global context of PTLD, including recent advances in its management and the interplay between thyroid dysfunction and pulmonary health in older adults. Written informed consent was obtained from the patient's next-of-kin (the patient is referenced by initials only), and identifying details have been removed to preserve anonymity.

## Methods and Case Presentation

**Patient Profile:** Mrs. R.B., a 64-year-old Indian female, presented to the geriatric medicine unit with chronic cough, progressive dyspnea, and low-grade fevers. She was a homemaker with a history of pulmonary tuberculosis 10 months prior. She had completed a full course of anti-tuberculosis therapy (ATT) from October 2024 to April 2025 (Category I regimen: isoniazid, rifampicin, pyrazinamide, ethambutol) with documented sputum conversion and treatment success. Post-TB, she continued to have a chronic productive cough, intermittent exertional breathlessness, and had lost ~5 kg over the past year. Her medical history was notable for type 2 DM and hypertension diagnosed 2 years ago; however, she had been non-adherent to medications (stopped metformin and telmisartan on her own). Other history included treated bilateral cataracts (3 years ago) and postmenopausal status; there was no history of smoking or significant occupational dust exposure. She denied any

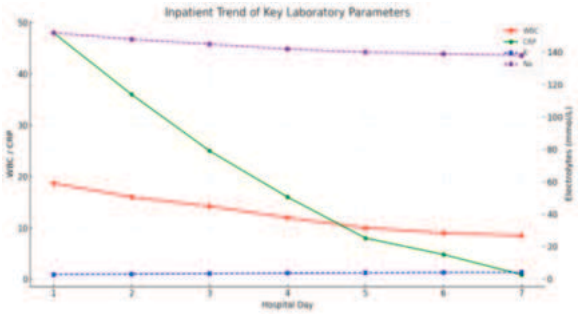
known TB contacts. Notably, in the month prior to presentation she had an episode of *community-acquired pneumonia* requiring hospitalization elsewhere – she received 5 days of IV meropenem and supportive care but left against medical advice before completing therapy.

**Presentation and Examination:** At our hospital's outpatient department in mid-July 2025, R.B. was ill-appearing but ambulatory. She complained of 1 week of worsening cough productive of copious whitish-yellow sputum (~200 mL/day) with occasional streaks of blood, increased breathlessness (NYHA class III), and right-sided pleuritic chest pain. Low-grade fever (max 38°C) was reported on and off. There was no hemoptysis frank, no night sweats, and no orthopnea or paroxysmal nocturnal dyspnea. On examination, her vital signs were: temperature 37.7°C, blood pressure 130/80 mmHg, pulse 92/min (regular), respiratory rate 24/min, $SpO_2$ 95% on room air. She was mildly tachypneic but able to speak full sentences. BMI was 21.5 kg/m². Chest examination revealed an elliptical chest shape with normal symmetry. Chest expansion was bilaterally *diminished* (~0.5 cm excursion on inspiration – reduced, likely due to prior TB scarring) but symmetric. On auscultation, she had coarse breath sounds with bilateral scattered rhonchi and coarse crackles over both lung bases. Vocal fremitus was reduced in the right upper zone. No pleural rub was heard. Cardiovascular exam was normal (S1, S2 present, no murmur). No peripheral edema or jugular venous distension was observed. Abdominal exam was unremarkable. Neurologically, she was alert and oriented; no focal deficits.

**Geriatric Assessment:** A comprehensive geriatric assessment indicated that R.B. was relatively independent in daily life. Her Mini Nutritional Assessment (MNA) score was 12 (normal nutritional status), and her functional evaluations were high: Barthel Index 100/100 (fully independent in basic ADLs) and Lawton IADL score 7/8 (independent in most instrumental ADLs). She had a Clinical Frailty Scale score of 3 (managing well, mildly frail) and a Mini-Cog of 4/5 (no significant cognitive impairment). Geriatric Depression Scale was 3 (no depression). These findings underscore the importance of *patient-centered care* and that aggressive therapy was appropriate given her preserved baseline function.

**Initial Investigations:** Upon admission, laboratory tests and imaging were obtained (Table 1). Her hemogram showed mild anemia with hemoglobin 11.7 g/dL and marked leukocytosis (18.7×10^9/L, 84% neutrophils) indicating an acute infection. Inflammatory markers were elevated: erythrocyte sedimentation rate 50 mm/hour and C-reactive protein 48 mg/L (normal <5). Renal function was normal (creatinine 0.8 mg/dL) but blood urea nitrogen was slightly high (24 mg/dL), likely from dehydration. Notably, she had electrolyte disturbances on admission: sodium 152 mmol/L (hypernatremia) and potassium 2.7 mmol/L (significant hypokalemia). These were corrected with IV fluids and electrolyte replacement, after which Na normalized to 140 and K to 4.4 mmol/L over 48 hours (Figure 1). Liver function tests were within normal limits. Arterial blood gas on room air showed pH 7.45, $pCO_2$ 38 mmHg, $pO_2$ 78 mmHg (mild hypoxemia) with normal lactate. Sputum samples were sent for culture and acid-fast bacilli (AFB) smear/PCR. Sputum AFB (GeneXpert) was negative for *Mycobacterium tuberculosis*, effectively ruling out TB reactivation. However, *sputum culture* grew *Pseudomonas aeruginosa*, consistent with the organism identified during her prior admission. The culture sensitivities showed *Pseudomonas* sensitive to ceftazidime, piperacillin-tazobactam, cefepime, aztreonam, meropenem, and tobramycin (resistant to levofloxacin and gentamicin). Blood cultures were sterile. A comprehensive metabolic panel, thyroid profile, and other tests were performed as detailed below.

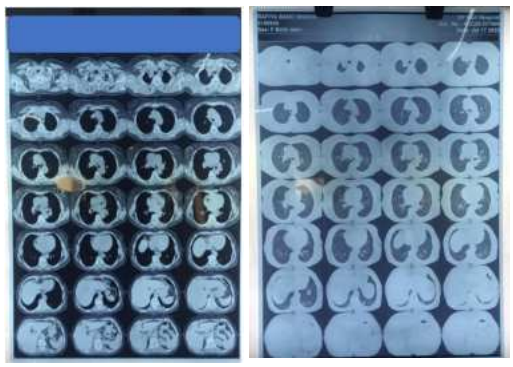
**Figure 1:**




- WBC and CRP levels are plotted on the left y-axis, showing steady improvement with antibiotic therapy.
- Potassium (K) and Sodium (Na) levels are plotted on the right y-axis, showing correction of initial electrolyte imbalances.

Inpatient trend of key laboratory parameters during treatment. The patient presented with neutrophilic leukocytosis (WBC 18.7×10^9/L) and elevated inflammatory markers, which improved with antibiotic therapy (WBC down to 8.5×10^9/L by Day 7). Initial electrolyte abnormalities (Na 152, K 2.7) were corrected (see K normalization with supplementation). CRP peaked at 48 mg/L and trended down (4.8□<1). Blood glucose (not shown) was controlled with insulin during admission.

**Imaging:**



**Endocrine Workup:** Given her known thyroid nodules and suppressed thyroid-stimulating hormone (TSH) noted a few months earlier, we evaluated her thyroid function (Table 2). TSH was 0.12 μIU/mL (low, reference 0.4–4.2) with free T4 1.3 ng/dL and free T3 2.8 pg/mL (both within normal ranges) – confirming subclinical hyperthyroidism. There were no overt hyperthyroid symptoms (she denied palpitations, heat intolerance, tremors) and her resting heart rate was <100. We attributed the subclinical thyrotoxicosis to her multinodular goiter. Indeed, thyroid ultrasound (done 3 months prior) had shown multiple bilateral thyroid nodules (largest ~1.5 cm) classified as TI-RADS 3 (low suspicion); fine-needle aspiration cytology (FNAC) at that time was non-diagnostic (Bethesda I) and was planned for repeat. There was no thyroid tenderness (painless thyroiditis less likely). Anti-thyroid peroxidase antibodies were negative, making autoimmune Graves' disease unlikely. We also measured her glycated hemoglobin (HbA1c) for diabetes control: it was 8.5%, indicating suboptimal glycemic control. An electrocardiogram showed normal sinus rhythm with no atrial fibrillation or ischemic changes. A 2D transthoracic echocardiogram revealed mild concentric left ventricular hypertrophy and Grade I diastolic dysfunction (impaired relaxation) – likely sequelae of long-standing hypertension and diabetes. Left ventricular ejection fraction was preserved (LVEF 60%), and there were no regional wall motion abnormalities. The aortic and tricuspid valves had trivial regurgitation; estimated pulmonary artery systolic pressure was 33 mmHg (no significant pulmonary

hypertension). These echo findings highlight diastolic heart failure with preserved EF (HFpEF), a common issue in elderly diabetics.

A high-resolution computed tomography (HRCT) of the thorax was performed to assess PTLD changes and rule out any abscess or cavities. The HRCT (17 July 2025) showed extensive post-TB sequelae: fibrotic scars with pleural thickening in the right upper lobe apex and left apicoposterior segment, and bronchiectatic changes in the right middle lobe and bilateral lower lobes. There were tree-in-bud opacities with surrounding ground-glass infiltrates in the right upper lobe posterior segment and bilateral lower lobes, consistent with endobronchial spread of infection (Figure 2A). Patchy areas of mosaic attenuation were noted throughout both lungs, suggestive of small-airway disease and air-trapping. No cavities were seen, but mucus plugging was present in some segmental bronchi. There was no pleural effusion or lymphadenopathy >1 cm (only a few subcentimeter reactive nodes). These imaging findings confirmed PTLD – specifically a combination of post-TB fibrosis and post-TB bronchiectasis – with an active bronchopulmonary infection superimposed. A representative slice of the HRCT is shown in Figure 2A, highlighting the cylindrical bronchiectasis and tree-in-bud densities in the right lung. For comparison, a chest X-ray (Figure 2B) on admission showed reticular opacities and volume loss in the right upper zone (from old TB), and hazy opacities at the right base consistent with the current pneumonia. (No direct patient quotes are included. All data are derived from the electronic medical records and investigations.)

**Diagnosis:** We established the following diagnoses:

(1) Post-tuberculosis lung disease – characterized by fibrocavitary changes and bronchiectasis due to prior TB infection (treated), now with chronic respiratory impairment;
(2) Acute exacerbation of PTLD due to Pseudomonas aeruginosa pneumonia – a community-acquired pneumonia on structural lung damage;

(3) Subclinical hyperthyroidism due to multinodular goiter;

(4) Type 2 diabetes mellitus (poorly controlled);

(5) Hypertension with mild hypertensive heart disease (diastolic dysfunction). The synergy of these conditions in an elderly patient required a multidisciplinary management plan focusing on infection control, respiratory support, endocrine balance, and chronic disease optimization.

**Management and Hospital Course:** R.B. was admitted to the geriatric ward for IV antibiotics and supportive care. We initiated broad-spectrum antimicrobial therapy targeting Pseudomonas. Given the culture sensitivities, we chose IV piperacillin-tazobactam 4.5 g q6h as first-line treatment (an appropriate anti-pseudomonal -lactam). She had already received 5 days of meropenem recently; our plan was to provide an additional 9 days of therapy to complete a 14-day course, de-escalating to a narrower agent. Piperacillin-tazobactam was continued for the full duration since the isolate was susceptible and the patient showed clinical improvement. Adjunctive bronchodilator therapy was started for her airway disease: nebulized salbutamol/ipratropium (Duolin) every 8 hours and inhaled budesonide twice daily for bronchial inflammation. We also performed regular chest physiotherapy and postural drainage to help clear bronchiectatic secretions. Low-flow oxygen by nasal prongs was given at 2 L/min during the first two nights for nocturnal hypoxemia ($SpO_2$ had dipped to ~90% during sleep). Systemic corticosteroids were not used in this acute setting because her obstructive component was not clearly reversible asthma and concerns existed about glycemic control; her prior hospital had given a brief course of IV hydrocortisone which we did not continue.

Glycemic management was addressed promptly: we initiated a basal-bolus insulin regimen (insulin glargine 10 units at night, with sliding scale regular insulin before meals) to achieve glucose control during the acute infection. Over the first 48 hours, her capillary blood glucoses improved from 250–300 mg/dL range to <180 mg/dL. Once eating well and infection resolving, she was transitioned back to oral metformin 500 mg twice daily, with plans for further titration and diabetes education on adherence. Her blood pressure on admission was reasonably controlled (130–140/80) after hydration; we restarted telmisartan 40 mg daily for hypertension on Day 3, both for BP and renal/cardiac protection. Potassium repletion (80 mEq over 24h) corrected her hypokalemia, and subsequent K levels remained 4.0–4.5 mmol/L.

Meanwhile, her sputum culture results confirming Pseudomonas were available by Day 3, validating our antibiotic choice. No atypical pathogens were isolated; respiratory viral panel was negative (she had been given oseltamivir empirically at the outside hospital, but influenza PCR was negative). We also remained vigilant for nontuberculous mycobacteria (NTM), as structural lung disease predisposes to NTM colonization/infection. Although NTM cultures were not immediately available, the clinical picture (acute febrile illness with high WBC) favored bacterial pneumonia over indolent NTM disease, so no empiric macrolide was added.

By Day 4–5 of treatment, R.B.'s fever and cough had significantly improved. Her sputum volume decreased and became mucoid. Serial blood counts showed WBC trending down to 11□8×10^9/L with resolving neutrophilia (Figure 1). Inflammatory markers also fell (CRP from 48 to 5 mg/L by discharge). We closely monitored her for any signs of atrial fibrillation, given her subclinical thyrotoxicosis and age, but her telemetry remained sinus rhythm throughout. Thyroid management: As she was asymptomatic and T4/T3 were normal, we did not initiate antithyroid drugs during the acute admission. Endocrinology consultation advised watchful monitoring of TSH in 6–8 weeks and proceeding with a repeat thyroid FNAC and radionuclide thyroid scan outpatient. Beta-blocker therapy was considered for cardioprotection, but resting heart rate was <95 and she had borderline low blood pressure; thus, no beta-blocker was started. We educated her on symptoms of overt hyperthyroidism and the importance of follow-up, given the potential risks of untreated subclinical hyperthyroid in the elderly (atrial arrhythmias, bone loss).

Clinical Outcome: On Day 8, after completing IV antibiotics, the patient was markedly better. She was ambulating in the ward without desaturation, and her cough had substantially reduced. A repeat chest X-ray showed clearance of the pneumonic infiltrates. We transitioned her to oral ciprofloxacin 500 mg twice daily for an additional 7 days after discharge as a precautionary step to ensure complete eradication of Pseudomonas (although the isolate was fluoroquinolone-resistant in vitro, this choice was debatable; an alternative considered was inhaled tobramycin, but logistics and cost were limiting). She was discharged home with instructions for pulmonary rehabilitation exercises, airway clearance techniques, and proper inhaler use.

Table 2 summarizes the timeline of her hospital treatment and key events. In essence, early aggressive antibiotic therapy and supportive care led to full recovery from the pneumonia. No major acute complications arose; notably, there was no evidence of TB reactivation or new organ involvement. The patient's hospital course highlighted the need to manage multiple coexisting conditions: infection, hyperglycemia, and latent thyroid dysfunction, all in the context of an aging

physiology. She was counseled extensively on medication adherence (for DM and HTN) and provided with a written action plan.

**Table 1:** Key Investigations on Admission (Day 1)

| Investigation | Result | Reference Range |
|---|---|---|
| Hemoglobin (Hb) | 11.7 g/dL | (Female: 12–16 g/dL) |
| Total Leukocyte Count | 18.7 ×10^9/L (Neut 84%) | (4–11 ×10^9/L) |
| Erythrocyte Sedimentation Rate | 50 mm/hour | (<20 mm/hour) |
| C-Reactive Protein (CRP) | 48 mg/L | (<5 mg/L) |
| Procalcitonin | 0.1 ng/mL (low) | (<0.5 ng/mL) |
| Sodium / Potassium | 152 mmol/L / 2.7 mmol/L | (135–145 / 3.5–5.0) |
| Urea / Creatinine | 25.8 mg/dL / 0.8 mg/dL | (7–20 / 0.5–1.2) |
| Aspartate/ALT (LFTs) | 18 / 16 U/L | (<=40 U/L) |
| Albumin / Globulin | 3.8 / 3.6 g/dL (A:G 1.06) | (Albumin 3.5–5.0) |
| Sputum AFB (GeneXpert) | Negative | – |
| Sputum Culture (Day 3) | P. aeruginosa growth | – |
| Sputum Culture Sensitivity | Sensitive: TZP, CAZ, MEM, FEP, ATM, TOB; Resistant: CIP, GEN | – |
| 2D Echocardiography | Grade I diastolic dysfunction; LVEF 60%; mild AR/TR; no PH | – |
| Thyroid Ultrasound (prev) | Multinodular goiter, TI-RADS 3; FNAC Bethesda I | – |
| ECG (admission) | Normal sinus rhythm, rate 90; no ischemia or AF | – |

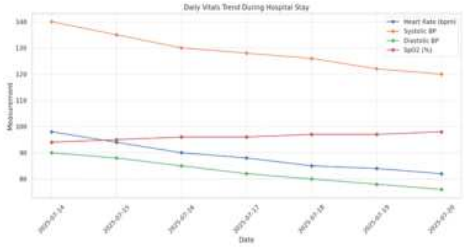
**Abbreviations:** Neut – neutrophil percentage; ALT – alanine aminotransferase; AFB – acid-fast bacilli; TZP – piperacillin-tazobactam; CAZ – ceftazidime; MEM – meropenem; FEP – cefepime; ATM – aztreonam; TOB – tobramycin; CIP – ciprofloxacin; GEN – gentamicin; LVEF – left ventricular ejection fraction; AR – aortic regurgitation; TR – tricuspid regurgitation; PH – pulmonary hypertension; AF – atrial fibrillation.

**Table 2:** Hospital Treatment Timeline and Events

| Day | Interventions and Events |
|---|---|
| 0 (Outside Hospital) | Initial admission elsewhere with fever, cough. Treated empirically with IV meropenem for 5 days; discharged AMA (against medical advice). |
| 1 (Admission Day 1) | Admitted to our facility. Investigations sent (labs, sputum studies, imaging). Started IV piperacillin-tazobactam 4.5g q6h targeting Pseudomonas. Nebulized bronchodilators and inhaled budesonide initiated. Oxygen 2 L/min for hypoxemia. Intensive insulin therapy for hyperglycemia. Electrolyte repletion (K^+). Comprehensive geriatric assessment performed. |
| 2 | HRCT Thorax obtained – showed PTLD changes (fibrosis, bronchiectasis) + tree-in-bud pneumonia. Continuation of antibiotics and supportive care. BP controlled, telmisartan restarted. |
| 3 | 2D Echocardiogram done – Grade I diastolic dysfunction, normal EF. Sputum culture result: P. aeruginosa (sensitive to current antibiotic). Maintained IV antibiotics. Endocrine consult: advised no acute intervention for subclinical hyperthyroid; plan outpatient follow-up. |
| 4-5 | Clinical improvement evident: afebrile, cough reduced. Titration of insulin doses as glucose control improves. Chest physiotherapy ongoing. Monitoring for arrhythmias – none observed. |
| 6 | Transition from IV to oral therapy discussed. Given fluoroquinolone resistance, decided to complete full IV course inpatient. Patient ambulating in hallway with $SpO_2$ 97% on RA. Educated on inhaler use and airway clearance. Repeat labs: WBC 11□9, CRP 8 mg/L. |
| 7 | Last day of IV piperacillin-tazobactam (Day 10 of effective antibiotics including prior hospital). Marked symptomatic relief. Chest X-ray improved. Arranged home medications and follow-up appointments (pulmonology, endocrinology, geriatrics, diabetes clinic). |
| 8 (Discharge) | Discharged with advice: oral ciprofloxacin 500 mg BID ×7 days (for possible Pseudomonas suppression, despite resistance – an interim measure until definitive follow-up), metformin resumed, continue telmisartan. Inhaled bronchodilator+steroid to continue. Follow-up sputum culture in 2 weeks. Outpatient plan for repeat thyroid FNAC in 2 months and TSH recheck in 6 weeks. Pulmonary rehab referral given. |


## RESULTS

During the 8-day hospitalization, R.B. achieved significant clinical improvement. Radiologic Resolution: Follow-up chest X-ray before discharge showed resolution of the pneumonic consolidation, with persistent chronic fibrotic changes. The clearance of infiltrates corroborated effective eradication of the infection. Laboratory Trends: As depicted in Figure 1, her leukocyte count normalized (from 18.7×10^9/L to 8.5×10^9/L) and neutrophil percentage fell to 70% by discharge. CRP dropped to 4 mg/L. These trends paralleled her clinical recovery. Her inflammatory markers and fever curve indicated a robust response to the antibiotic regimen, without indications of uncontrolled sepsis or new inflammatory foci.



Pulmonary function (spirometry) was not performed during the acute illness, but we planned it for a stable outpatient follow-up to quantify her chronic impairment. We anticipated some irreversible obstruction given her history; previous records after TB treatment had documented $FEV_1$ ~60% predicted.

Microbiological outcome: The Pseudomonas aeruginosa infection was presumed eradicated given her full clinical response. P. aeruginosa is known to form biofilms in bronchiectatic airways and can become chronic; however, in this first known isolation for her (aside from the recent prior admission), we aimed for complete clearance. We scheduled a repeat sputum culture at 4 weeks post-discharge to check for persistent colonization. If Pseudomonas were to persist asymptomatically, the plan included considering inhaled antibiotic therapy (such as nebulized tobramycin or colistin) to suppress chronic colonization, per bronchiectasis management guidelines.

**Endocrine results:**


There was no change in her subclinical hyperthyroid status during the short admission; TSH remained low but stable (0.10 μIU/mL on discharge). Importantly, she did not develop any atrial fibrillation or overt thyrotoxic symptoms under monitoring. Her blood glucose, on the other hand, improved dramatically – fasting glucose was 110 mg/dL on the morning of discharge, and random glucose <180 mg/dL, reflecting successful inpatient diabetes management. An HbA1c drawn during admission was 8.5%, reinforcing the need for better long-term glycemic control.

Multidisciplinary input: This case was managed with a multidisciplinary approach. Geriatrics oversaw the coordination of care (addressing polypharmacy and functional needs), pulmonologists were consulted for the bronchiectasis and PTLD aspects, infectious disease input was taken for optimizing antibiotic therapy, and endocrinologists guided the thyroid management plan. The patient and family were counseled extensively, which improved her understanding of the chronic nature of PTLD and the importance of adherence to medications and follow-up.

By discharge, R.B. was at her baseline apart from mild fatigue. She was independent in ambulation and self-care. We arranged a post-discharge follow-up regimen (detailed below) to ensure continuity of care. Overall, the "result" of this hospitalization was a successful acute management of a potentially life-threatening pneumonia, along with stabilization of her chronic conditions. No adverse drug reactions occurred other than a transient episode of loose stool (possibly from antibiotics) which was managed conservatively. Liver and renal functions remained normal throughout treatment.

## DISCUSSION

This case underscores the challenges of managing post-tuberculosis lung disease (PTLD) in a geriatric patient with multiple comorbidities. It highlights how structural lung damage from prior TB can predispose to atypical and severe infections, and how coexisting endocrine and metabolic disorders must be simultaneously balanced. We discuss the key themes arising from this case – PTLD epidemiology and sequelae, the clinical implications of Pseudomonas infection in bronchiectatic lungs, management of subclinical hyperthyroidism in the elderly, geriatric care principles in multimorbidity, and recent advances in treatment and follow-up.

Global Epidemiology and Significance of PTLD: With improving TB survival rates, attention is shifting to the long-term health of TB survivors. PTLD has been formally defined by international consensus as chronic respiratory abnormality attributable to past TB. Studies across continents reveal a high prevalence of PTLD among cured TB patients: for example, 68% in an Australian cohort, 74% in a South African study, and 68% in an Indian tertiary center follow-up. A 2021 systematic analysis (41,000+ patients) estimated ~59% have post-TB pulmonary function impairment on spirometry. These figures emphasize that microbiological cure is not the end of TB's impact. In our patient's region (India), which along with China accounts for a large proportion of TB cases, PTLD represents a burgeoning chronic disease burden. Risk factors for developing PTLD include extensive or cavitary TB, delay in diagnosis, inadequate treatment, recurrent TB, and multi-drug resistant TB. Although our patient had drug-sensitive TB treated fully, her moderate disease still led to permanent lung damage. Notably, older age is associated with worse PTLD outcomes, partly due to reduced lung reserve and concomitant illnesses. TB survivors over 50 have higher all-cause mortality than the general population for years after cure.

Clinically, PTLD can manifest in various phenotypes: obstructive airway disease (similar to COPD), restrictive patterns from fibrosis or destroyed lung, bronchiectasis, and even pulmonary vascular disease (e.g. pulmonary hypertension). Our patient demonstrated several of these – fibro-cavitatory changes and bronchiectasis (from TB), and likely small-airway obstruction (mosaic attenuation on CT). Indeed, TB is an established cause of chronic airflow obstruction independent of smoking. The inflammation and fibrosis during TB can permanently remodel airways. Bronchiectasis, present in our case, occurs due to TB-related bronchial damage and traction from fibrosis. In a study from central India, prior TB was the most common cause of bronchiectasis, and post-TB bronchiectasis patients had significantly more hemoptysis and exacerbations than non-TB cases. Importantly, they also found that Pseudomonas aeruginosa was the most frequently isolated pathogen in post-TB bronchiectasis patients (in ~27% of cases), aligning with our patient's infection. These findings highlight that PTLD patients, especially with bronchiectasis, are prone to colonization/infection by opportunistic bacteria.

Pseudomonas Infection in Bronchiectasis/PTLD: P. aeruginosa is a Gram-negative rod known for causing chronic lung infections in structurally damaged lungs (e.g. cystic fibrosis, non-CF bronchiectasis). In bronchiectasis, Pseudomonas colonization is associated with worse clinical outcomes – including more frequent exacerbations, faster lung function decline, and increased mortality. A comprehensive analysis of non-CF bronchiectasis found that chronic P. aeruginosa infection confers an approximately 3-fold higher risk of death and significantly higher rates of hospitalization. It tends to form biofilms in airways, making eradication difficult and relapses common. In our case, the patient's Pseudomonas pneumonia likely took hold due to her residual bronchiectatic spaces and impaired mucociliary clearance post-TB. Notably, she had no prior history of bronchiectasis before TB; this was a postinfectious bronchiectasis, a phenomenon well-documented as a TB sequela. Her case illustrates that Pseudomonas can cause community-acquired pneumonia (CAP) in patients with structural lung disease – unlike in normal hosts where Pseudomonas CAP is rare.

Treatment of Pseudomonas infections requires antibiotics

with anti-pseudomonal activity. We used piperacillin-tazobactam based on sensitivities; alternative agents include ceftazidime, cefepime, carbapenems (meropenem), or quinolones (if sensitive). Guidelines for bronchiectasis recommend attempting eradication therapy when P. aeruginosa is first isolated. In practice, this may involve a combination of IV antibiotics followed by oral or inhaled suppressive therapy. Although evidence is still emerging, one meta-analysis reported that early eradication is successful in ~40% of cases at 1 year. Our strategy of a prolonged IV course aimed for eradication. We considered adding inhaled tobramycin after discharge, which is known to reduce Pseudomonas load and exacerbations in bronchiectasis. Cost and patient factors led us to a short oral follow-on course instead, but we stressed close follow-up. Should she grow Pseudomonas again, chronic suppressive therapy may be indicated.

Another infection consideration in PTLD is nontuberculous mycobacteria (NTM) like Mycobacterium avium complex. Patients with prior TB or bronchiectasis have higher risk of NTM lung disease. Although our patient's cultures did not reveal NTM, clinicians should keep a high index of suspicion for NTM in PTLD patients with subacute symptoms, as NTM infection can mimic recurrent TB or refractory pneumonia.

Management Advances in PTLD: Historically, once TB treatment was completed, patients were discharged from TB programs without structured follow-up. However, growing awareness of PTLD has led to calls for systematic post-TB care. International standards published in 2021 now recommend that all patients completing TB therapy undergo screening for PTLD, including clinical review, spirometry, and chest imaging at end of treatment. Early identification of PTLD allows timely interventions – for example, bronchodilators for airflow obstruction, inhaled corticosteroids if airway inflammation or asthma-like features, and physical therapy for functional improvement. In our practice, we plan to perform pulmonary function testing and a 6-minute walk test in R.B.'s follow-up to quantify her impairment and tailor her inhaler therapy. If she has significant obstruction, adding a long-acting bronchodilator (LABA or LAMA) could improve her symptoms, extrapolating from COPD management.

Another cornerstone of PTLD management is pulmonary rehabilitation (PR). Pulmonary rehab programs (exercise training, breathing techniques, education) have shown clear benefits in chronic respiratory diseases. In post-TB patients, PR has been shown to improve exercise capacity, quality of life, and even lung function indices. One study demonstrated that implementing a 6-week rehab program in TB survivors led to significant gains in six-minute walk distance and reduced dyspnea. Integrating PR even during TB treatment has been suggested to mitigate long-term lung damage. We referred our patient to outpatient PR; given her motivation and baseline independence, we anticipate she will benefit from supervised exercise and airway clearance training.

For fibrotic sequelae, no specific antifibrotic therapy is established (unlike idiopathic pulmonary fibrosis). However, some trials are exploring anti-fibrotics like pirfenidone or N-acetylcysteine in post-TB fibrosis – these remain experimental. Vaccinations are a simple yet critical aspect: PTLD patients should receive influenza and pneumococcal vaccinations to prevent future respiratory infections, as we advised for R.B. (She received influenza vaccine on discharge and pneumococcal 23-valent vaccine was planned).

It is also worth noting that management of PTLD should address psychosocial aspects. Many TB survivors suffer psychosocial sequelae (stigma, depression, financial strain). Our patient had a supportive family and screened negative for depression, but we remained attentive to her social needs (e.g., arranged for follow-up phone calls and low-cost medication sources, given financial limitations often affect medication adherence in older patients). Global initiatives are urging that TB programs transition into or collaborate with non-communicable disease (NCD) programs to provide continued care for TB aftereffects.

Thyroid Dysfunction in the Elderly – Subclinical Hyperthyroidism: An interesting facet in this case is the patient's subclinical hyperthyroidism due to multinodular goiter. In older adults, subclinical hyperthyroidism (defined by low TSH with normal T4/T3) is not uncommon – population studies show prevalence around 1–2%, higher in women and iodine-sufficient regions. In our patient, the etiology is likely autonomous thyroid nodules (toxic nodular goiter), which is a common cause of subclinical hyperthyroid in areas without widespread Graves' disease. The clinical importance of subclinical hyperthyroidism lies in its potential consequences: namely, atrial fibrillation (AF) and bone demineralization. Evidence indicates that even mild thyroid overactivity can increase the risk of AF in older individuals. A meta-analysis of prospective cohorts (n≈8,700) found that subclinical hyperthyroidism is associated with ~3-fold higher risk of atrial fibrillation compared to euthyroid status. Another analysis reported a hazard ratio of 1.68 for AF in subclinical hyperthyroid, rising to 3.1 in those with TSH <0.1 mIU/L. Our patient's TSH was 0.1–0.12, which places her in the higher-risk category ("Grade II" subclinical hyperthyroidism, often defined as TSH <0.1). The absence of AF in her case may be partly luck and partly the short observation window; however, her diastolic dysfunction and age put her at risk if AF were to occur, potentially precipitating heart failure.

**Another concern is bone health –** excess thyroid hormone accelerates bone turnover. Postmenopausal women with subclinical hyperthyroidism can have increased risk of osteoporosis and fractures if the condition persists. Some studies suggest fracture risk correlates with the duration of suppressed TSH. In our patient, this is relevant as she is 64 and postmenopausal; prolonged untreated subclinical thyrotoxicosis could compromise her bone density, compounding her fall/fracture risk as she ages.

Management guidelines for subclinical hyperthyroidism are nuanced and consider TSH level and patient factors. Current consensus (e.g. American Thyroid Association) generally recommends treatment for patients ≥65 years old with persistent TSH <0.1 mIU/L, and considers treatment for TSH 0.1–0.4 if there are risk factors (such as heart disease or osteoporosis). Our patient meets criteria to consider therapy (age >65 minus one year, TSH ~0.1, and she has cardiac risk factors). However, treatment decisions also depend on cause and patient preference. In toxic nodular goiter, definitive options include radioactive iodine ablation or antithyroid medication (e.g. low-dose methimazole). Studies in older patients (like the PIRAHTES trial) have shown that treating subclinical hyperthyroidism can reduce AF incidence. Both radioiodine and methimazole have been found effective and safe for elderly subclinical hyperthyroid patients in a recent randomized study. In our case, we opted to defer immediate therapy until her pulmonary issue was resolved and we could reassess TSH in a stable state. Given her multinodular goiter, we favor radioactive iodine as a long-term solution (especially if follow-up confirms low TSH). That would ablate autonomously functioning tissue and likely convert her to hypothyroid, which is easier to manage. Beta-blockers (like metoprolol) are recommended if there are any tachycardic or symptomatic features, or as a bridge to definitive therapy. We kept this in mind; although she was asymptomatic, we will initiate a beta-blocker if her heart rate or symptoms increase at follow-up.

**Geriatric Considerations and Multimorbidity:** This case exemplifies the need for holistic care in older patients. Key geriatric principles applied include: Comprehensive

Geriatric Assessment (CGA) – which we performed to tailor her care plan. CGA has proven benefits in optimizing outcomes for frail older adults with multimorbidity. Identifying her high functional status reassured us that aggressive treatment (like a full course of IV antibiotics) was appropriate and that she could participate in rehab. It also flagged areas to monitor (e.g. slight frailty, risk of polypharmacy). Polypharmacy management: At discharge, she was on quite a few medications (antibiotic, inhalers, metformin, insulin, telmisartan, vitamins, etc.). We simplified what we could (e.g. short course of oral antibiotic, then stop; insulin only short-term transitioning to orals). We also counseled on adherence, especially because her prior non-compliance led to poor BP/DM control. Involving her family was crucial – her husband attended education sessions about medication dosing and inhaler technique. Follow-up coordination is another geriatric aspect: we scheduled her follow-ups on the same day at a multi-specialty clinic to reduce travel burden.

A notable psychosocial factor was that she left the previous hospital against medical advice. Reasons can include cost or family obligations. At our center, we addressed this by providing social worker input – ensuring part of her treatment (like pulmonary rehab) could be done near her home and that the costs of medications were manageable (we switched her to generic metformin and a low-cost ARB, and the government health scheme covered her TB and pneumonia treatment). This individualized approach likely improved her engagement with care.

Additionally, patient education was tailored to her health literacy. Using teach-back methods, we confirmed she understood how to use inhalers properly and why completing antibiotic courses is vital (given the tendency of Pseudomonas to cause relapsing infection if not fully eradicated). For her thyroid condition, we explained the subtle symptoms to watch for (palpitations, tremor) since older patients can have "apathetic" hyperthyroidism (presenting with atypical or no symptoms).

Finally, we leveraged the concept of shared decision-making. For example, in deciding whether to treat her subclinical hyperthyroidism immediately, we discussed the pros and cons with her. She expressed relief that no new medication was added during her acute illness, and agreed to close monitoring. Shared decision-making is critical in geriatric care to align treatments with patient goals and values.
Proposed Post-Discharge Follow-Up Regimen: Given the complexity of her conditions, a structured follow-up plan was instituted:

- **Pulmonology Clinic (2 weeks post-discharge):** Clinical review with repeat sputum culture and spirometry. If Pseudomonas remains present, consider inhaled antibiotic therapy. Assess need for long-term macrolide (some bronchiectasis patients benefit from low-dose azithromycin to reduce exacerbations, although caution in our patient due to Pseudomonas and potential QT prolongation in thyrotoxic states). Plan CT chest in 6–12 months to monitor any progression of PTLD or new complications (e.g. aspergilloma or TB reactivation).

- **Endocrinology (6–8 weeks):** Re-check TSH, free T4, T3. If TSH remains <0.1, likely proceed with therapy. A repeat ultrasound and FNAC of the thyroid largest nodule is scheduled at 2 months; if the nodule is benign and autonomous, radioactive iodine ablation of the thyroid will be recommended. In the interim, periodic heart rate and rhythm monitoring will be done – we provided her with a wearable pulse monitor to track for any irregularity.

- **Diabetes Clinic (1 month):** Evaluate glucose logs (we gave her a glucometer). If glycemic control remains inadequate (goal HbA1c <7.5% for her, given age), consider adding a sulfonylurea or low-dose basal insulin long-term. Ensure she's seeing an ophthalmologist for diabetic retinopathy screening (she had background retinopathy per a fundus exam during admission, grade I hypertensive changes).

- **Geriatric Clinic (1 month):** Comprehensive review of all issues, deprescribing if possible (e.g. if thyroid is ablated and hypothyroid, we'll add levothyroxine – which is one more drug, so adjusting others accordingly). We will repeat frailty and falls risk assessment after her acute recovery. Bone health will be addressed – a DEXA scan is planned to evaluate her bone density given her thyroid condition and age; calcium/vitamin D supplementation was started.

- **Rehabilitation Services (starting 2–4 weeks):** She will attend pulmonary rehabilitation sessions 3 times weekly for 6 weeks at a local center. Goals: improve exercise tolerance, teach energy conservation, and monitor oxygen needs on exertion.

- **Vaccinations:** As noted, ensure pneumococcal vaccination is completed. Annual influenza vaccine to be continued.

- TB Surveillance: Although risk of TB recurrence is low after full therapy, her lung damage can confuse future diagnostics. We instructed that any future sputum testing for TB be interpreted cautiously (old scar can cause false-positive X-ray readings). She will have an annual clinical evaluation for TB symptoms as part of routine.

If all goes well, these coordinated follow-ups will help maintain her health and hopefully prevent rehospitalizations. From a health-system perspective, cases like this argue for integrated "TB survivor clinics" or including PTLD in general NCD programs. Our patient was fortunate to be managed in a geriatric unit that could liaise across specialties; many TB survivors are younger and may fall through cracks once discharged from TB programs. Advocacy is growing to formally address PTLD at national and global policy levels.

**Limitations and reflections:** This case report has the inherent limitation of a single-patient observation. Not every PTLD patient will have the same constellation of issues, but many elements here (post-TB bronchiectasis, infection risk, multimorbidity) are common globally. We also acknowledge that our management choices (e.g. not immediately treating subclinical hyperthyroidism) might differ from others – guidelines allow for clinical judgment. In retrospect, one could argue for starting a beta-blocker to mitigate any subclinical hyperthyroid effect on her heart, but given her BP and careful monitoring, we chose observation. The decision to use oral ciprofloxacin post-IV therapy, despite in vitro resistance, was also a pragmatic one – it might be debated. An ideal approach would be to use inhaled anti-pseudomonal antibiotics post-discharge, but resource constraints influenced our plan.

From a learning standpoint, this case reinforces that a multidisciplinary, geriatric-informed approach can lead to good outcomes in complex cases. By treating the acute infection aggressively while minding chronic conditions, we avoided potential pitfalls (for instance, no arrhythmia occurred, no hyperosmolar hyperglycemic state from steroids, etc.). It also highlights the emerging concept that TB care does not end with antibiotic completion – rehabilitative and chronic care is the next frontier in TB management.

## Conclusion

**Clinical Pearls:**

- PTLD is common and under-recognized: Up to 50–70% of

TB survivors develop chronic lung abnormalities. Clinicians should screen post-TB patients for lingering respiratory issues and not dismiss symptoms as "old TB scars." Early pulmonary follow-up and function testing are recommended for all cured TB patients.

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