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The Triple Burden: A Case of Very Severe Chronic Obstructive Pulmonary Disease at the Intersection of Tobacco Smoke, Occupational Dust, and Post-Tuberculosis Sequelae

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ABSTRACT

Introduction: Chronic Obstructive Pulmonary Disease (COPD) is a heterogeneous condition with multiple etiologies. The confluence of tobacco smoke, occupational hazards, and post-tuberculosis lung disease (PTLD) presents a significant diagnostic and therapeutic challenge, often resulting in a severe clinical phenotype.

Case Illustration: We present the case of a 56-year-old male construction worker with a significant smoking history and a history of cured pulmonary tuberculosis, who presented with an acute exacerbation of dyspnea. Physical examination revealed signs of severe airflow obstruction, including tachypnea, pursed-lip breathing, and a barrel chest. Diagnostic workup, including a Pulmonary Update in Medical Assessment (PUMA) score of 8 and a COPD Assessment Test (CAT) score of 36, indicated a high

disease burden. Post-bronchodilator spirometry confirmed very severe, irreversible airflow obstruction (Forced Expiratory Volume in 1 second [FEV1] 23% predicted; FEV1/Forced Vital Capacity [FVC] ratio 0.337). Chest radiography demonstrated emphysematous changes superimposed on fibrotic sequelae from prior tuberculosis.

Discussion: The patient's profound respiratory impairment is attributed to a "triple hit" pathophysiology. Smoking-induced emphysema, chronic inorganic dust exposure from his 40-year occupation, and tuberculosis-induced structural damage—including fibrosis and potential bronchiectasis—have synergistically contributed to his very severe airflow limitation. This case exemplifies the distinct entity of tuberculosis-associated COPD, which is characterized by more severe and less reversible obstruction compared to smoking-induced disease alone.

Conclusion: This case highlights the critical need for clinicians to recognize the cumulative impact of multiple risk factors in COPD. A comprehensive history, including past infections and occupational exposures, is paramount for accurate diagnosis and for tailoring management, which must include aggressive non-pharmacological interventions like pulmonary rehabilitation.

Keywords: Chronic Obstructive Pulmonary Disease (COPD), Post-Tuberculosis Lung Disease (PTLD), Occupational Lung Disease, Construction Worker, Spirometry, Case Report.

INTRODUCTION

Background: The Converging Global Epidemics

Chronic Obstructive Pulmonary Disease (COPD) represents a monumental global health challenge. In 2019, it was responsible for an estimated 3.3 million deaths worldwide, with a global prevalence of 212.3 million cases (Venkatesan, 2022). Projections indicate a continued rise, with the number of affected individuals expected to approach 600 million by 2050. While cigarette smoking remains the most widely recognized etiological agent, the understanding of COPD has evolved significantly. The Global Initiative for Chronic Obstructive Lung Disease (GOLD) 2024 report defines COPD as a "heterogeneous lung condition characterized by chronic respiratory symptoms (dyspnea, cough, expectoration and/or exacerbations) due to abnormalities of the airways (bronchitis, bronchiolitis) and/or alveoli (emphysema) that cause persistent, often progressive, airflow obstruction" (Global Initiative for Chronic Obstructive Lung Disease, 2024; Agusti et al., 2023). This modern definition explicitly acknowledges the multifactorial nature of the disease, accommodating contributions from environmental pollution, occupational exposures, and infectious diseases (Mannino and Buist, 2007).

Concurrently, the world is home to a vast and growing population of tuberculosis survivors, estimated at 155 million globally (Allwood et al., 2021). A substantial proportion of these individuals, potentially as high as 50%, suffer from the long-term respiratory sequelae known as Post-Tuberculosis Lung Disease (PTLD) (Allwood et al., 2021). PTLD is a spectrum of chronic pulmonary abnormalities that persist after the successful bacteriological cure of tuberculosis, encompassing structural damage such as fibrosis, bronchiectasis, and cavitation (van Zyl-Smit, Allwood and Stick, 2020; Meghji et al., 2021). This condition is a major, yet often under-recognized, cause of chronic respiratory disability, increased hospitalization rates, and mortality (Meghji et al., 2021; Raff and Kirenga, 2023). The strong epidemiological link between a history of tuberculosis and the subsequent development of fixed airflow obstruction establishes PTLD as a

critical non-smoking risk factor for COPD (Amaral et al., 2015; Mbongue et al., 2023). The convergence of these global epidemics—tobacco use, occupational hazards, and the legacy of tuberculosis—creates a complex clinical landscape where patients may present with severe respiratory disease driven by multiple, overlapping pathogenic mechanisms.

Objectives

The primary objectives of this case report are threefold:

1. To present a detailed clinical account of a patient with very severe COPD resulting from the combined effects of long-term tobacco use, chronic occupational dust exposure as a construction worker, and sequelae of a prior pulmonary tuberculosis infection.
2. To analyze the distinct pathophysiological contributions of each etiological factor—tobacco-induced emphysema, occupational pneumoconiosis, and post-tuberculosis structural damage—to the patient's severe and irreversible airflow obstruction.
3. To discuss the diagnostic and therapeutic challenges posed by this complex, mixed-etiology phenotype, referencing current GOLD guidelines for COPD management and emerging international standards for the assessment of PTLD (Global Initiative for Chronic Obstructive Lung Disease, 2024; Agusti et al., 2023).

Benefits

This report aims to provide significant clinical and educational benefits. First, it serves as a clinical exemplar to heighten awareness among pulmonologists, internists, and primary care physicians about the severe respiratory consequences of PTLD, particularly when compounded by other common risk factors like smoking and occupational exposures. Second, it underscores the absolute necessity of obtaining a comprehensive patient history that extends beyond smoking status to include a detailed inquiry into past infectious diseases and a granular, lifelong occupational exposure history. By doing so, this report advocates for a more holistic diagnostic approach that can improve accuracy and guide more effective, personalized management for patients with complex

respiratory diseases.

Hypothesis

The central hypothesis of this report is that the patient's exceptionally severe airflow obstruction (GOLD Stage 4) and profound symptom burden are not attributable to a single cause but are the result of a synergistic "triple-hit" pathogenic mechanism. It is posited that the chronic inflammation and parenchymal destruction from decades of smoking, the fibrotic and pneumoconiotic effects of long-term occupational dust inhalation, and the destructive, scarring legacy of tuberculosis have converged within the patient's lungs. This convergence has created a uniquely severe and poorly reversible obstructive phenotype that is more profound than would be expected from any single etiological factor acting in isolation.

Research Gap

While the individual associations between smoking and COPD, occupational dust and COPD, and tuberculosis and COPD are well-established in the medical literature, a significant gap exists. There is a paucity of detailed case reports and clinical studies that specifically characterize the presentation, physiological profile, and management of patients who exist at the confluence of all three major risk factors. This patient sub-phenotype, representing a "perfect storm" of pulmonary insults, is likely under-recognized and under-reported. This report aims to begin filling that gap by providing a granular, evidence-based analysis of this specific clinical entity, offering insights into its unique pathophysiology and clinical course.

Novelty

The novelty of this case report lies in its detailed deconstruction of a tripartite etiology for very severe COPD. By systematically integrating clinical data with pathophysiological principles from three distinct but intersecting fields—tobacco-related lung disease, occupational medicine, and infectious disease sequelae—it offers a holistic and nuanced perspective on a complex clinical

entity. This approach moves beyond a siloed view of risk factors to present an integrated model of cumulative and synergistic lung damage. The case is not merely an anomaly but a potential harbinger of a growing clinical challenge, as the global population of aging smokers, occupationally exposed workers, and tuberculosis survivors continues to expand and overlap, demanding a more sophisticated and comprehensive approach from clinicians.

CASE ILLUSTRATION

Patient Demographics and Presenting Complaint

Mr. A, a 56-year-old male, presented to the Puskesmas Mpunda, a primary care clinic in Bima City, with a chief complaint of progressively worsening shortness of breath over the preceding two days.

History of Present Illness

The patient described his dyspnea as constant and unremitting, accompanied by audible wheezing and a productive cough yielding whitish sputum. The symptoms were significantly exacerbated by minimal physical activity, such as walking within his home, and were severe enough to frequently disrupt his sleep. He denied orthopnea or paroxysmal nocturnal dyspnea. There was no associated fever, night sweats, or drastic weight loss related to this acute presentation.

Past Medical and Social History

The patient's history was remarkable for multiple significant risk factors for chronic lung disease.

- **Prior Respiratory Diagnoses:** He was first diagnosed with COPD in 2023. His maintenance therapy at the time of presentation consisted of oral medications: salbutamol 4 mg, theobron 130 mg, and acetylcysteine 200 mg.
- **Tuberculosis History:** In April 2024, approximately one year after his initial COPD diagnosis, he was hospitalized at Bima Regency General Hospital with severe dyspnea, hemoptysis, and a

significant weight loss of 5 kg over one month. He was diagnosed with pulmonary tuberculosis and successfully completed a standard 6-month course of Category I anti-tuberculosis therapy (OAT). He was subsequently declared cured based on clinical and microbiological criteria.

- **Occupational History:** The patient had worked as a construction worker continuously since the age of 16, a total duration of 40 years. His daily work involved direct and prolonged exposure to a variety of airborne pollutants, including cement dust, concrete dust, silica, and other inorganic dusts. He reported inconsistent use of personal protective equipment, such as respiratory masks, throughout his career. This long-term, high-intensity exposure is a critical etiological factor, as inorganic dust exposure in construction workers is strongly associated with increased mortality from COPD, independent of smoking status (Torén, Järvholm and Bergdahl, 2004).
- **Smoking History:** He was a long-term, heavy smoker, having started at age 17 and quit at age 55 (a 38-year duration). He typically consumed one to one-and-a-half packs of local *kretek* (clove) cigarettes daily, which corresponds to 16–24 cigarettes per day. This calculates to a substantial smoking history of approximately 38 to 57 pack-years, placing him in the highest risk category for smoking-induced lung disease.
- **Comorbidities:** The patient had no known history of hypertension, diabetes mellitus, cardiovascular disease, liver disease, or malignancy.

Physical Examination Findings

On presentation, the patient was in moderate respiratory distress, though he was conscious, alert, and fully oriented (Compos Mentis).

- **Vital Signs:** Blood Pressure was 130/80 mmHg, Heart Rate was 108 beats per minute (tachycardia), Respiratory Rate was 32 breaths per minute (tachypnea), Body Temperature was 36.8°C (afebrile), and peripheral oxygen saturation (SpO2) was 92% on ambient air (hypoxemia).
- **Anthropometrics:** His weight was 51 kg and height was 175 cm, yielding a Body Mass Index

(BMI) of 16.7 kg/m², classifying him as underweight.

- **Head and Neck:** Examination revealed no conjunctival pallor or scleral icterus. He exhibited prominent pursed-lip breathing, a physiological maneuver to increase end-expiratory pressure and prevent small airway collapse. The Jugular Venous Pressure (JVP) was estimated at 5-2 cmH_2O. There was no palpable cervical lymphadenopathy.
- **Thorax and Lungs:** The chest examination was highly suggestive of severe airflow obstruction and hyperinflation. Inspection revealed a classic barrel chest deformity with visibly widened intercostal spaces. On palpation, tactile vocal fremitus was symmetrically decreased across both hemithoraces. Percussion of the chest wall yielded a hyperresonant note bilaterally. Auscultation confirmed a markedly prolonged expiratory phase. Diffuse bilateral wheezing was present, along with coarse crackles (*ronki basah kasar*).
- **Cardiovascular, Abdominal, and Extremities:** Examinations of the cardiovascular, abdominal, and peripheral systems were all within normal limits.

Diagnostic Workup and Results

A series of diagnostic tests were performed to quantify the severity of his disease and rule out alternative diagnoses.

- **Clinical Assessment Scores:**
  - **PUMA Questionnaire:** The patient's score was 8 out of a possible 9. The PUMA questionnaire is a validated screening tool for identifying patients at high risk for COPD. A score of ≥6 is considered a strong positive screen, with studies showing that higher scores are more likely to be associated with an advanced GOLD stage of disease (Au-Doung et al., 2022). A score of 8 placed him in the highest risk stratum, strongly predicting significant underlying obstructive lung disease.
  - **COPD Assessment Test (CAT):** The patient's CAT score was 36. The CAT is an 8-item questionnaire that measures the health status and quality of life impairment in COPD patients. Scores range from 0 to 40, with a score of 31–40 indicating a "very high impact"

(Jones et al., 2009). A score of 36 suggests that the disease profoundly affects every aspect of his daily life, correlating with feelings of never having good days and being unable to perform even simple tasks.

- **Pulmonary Function Testing:**
  - **Peak Expiratory Flow (PEF):** At the primary care clinic, his PEF was measured at 202 L/minute. This value is less than 50% of the predicted value for his age and height, indicating severe airflow obstruction.
  - **Spirometry:** Formal spirometry was conducted at the referral pulmonology clinic to confirm the diagnosis and stage its severity. The results, both before and after the administration of a short-acting bronchodilator, are summarized in Table 1. The post-bronchodilator FEV1/FVC ratio of 0.337 unequivocally confirms the presence of severe, fixed (irreversible) airflow obstruction, the hallmark of COPD (Global Initiative for Chronic Obstructive Lung Disease, 2024). The post-bronchodilator FEV1 of only 23% of the predicted value classifies his disease as GOLD Stage 4 (Very Severe) (Global Initiative for Chronic Obstructive Lung Disease, 2024). The minimal improvement in FEV1 post-bronchodilator (+0.06 L) underscores the irreversible nature of his condition.

Parameter	Pre-Bronchodilator	Post-Bronchodilator	Change	Interpretation
FVC (L)	1.41	1.72	+0.31 L	Severely reduced vital capacity
FEV1 (L)	0.52	0.58	+0.06 L	Very severely reduced expiratory volume

Parameter	Pre-Bronchodilator	Post-Bronchodilator	Change	Interpretation
FEV1 (% Predicted)	21%	23%	+2%	Consistent with GOLD Stage 4 (Very Severe)
FEV1/FVC Ratio	0.369	0.337	-	Confirms severe, fixed/irreversible obstruction

Table 1: Pre- and Post-Bronchodilator Spirometry Results

- **Imaging:**
  - **Anteroposterior Chest Radiograph:** The chest X-ray (provided in the case data) revealed multiple pathological findings consistent with his complex history. There were clear signs of pulmonary hyperinflation, including flattened diaphragms and increased radiolucency of the lung fields, classic for emphysema. Superimposed on these changes were prominent, irregular interstitial markings and fibrotic stranding, particularly in the perihilar and upper lung zones. These findings are highly characteristic of the fibrotic sequelae of healed pulmonary tuberculosis. The radiograph thus provides a compelling visual representation of the patient's dual lung pathology, where emphysematous destruction coexists with post-infectious fibrosis (van Zyl-Smit, Allwood and Stick, 2020; Meghji et al., 2021).

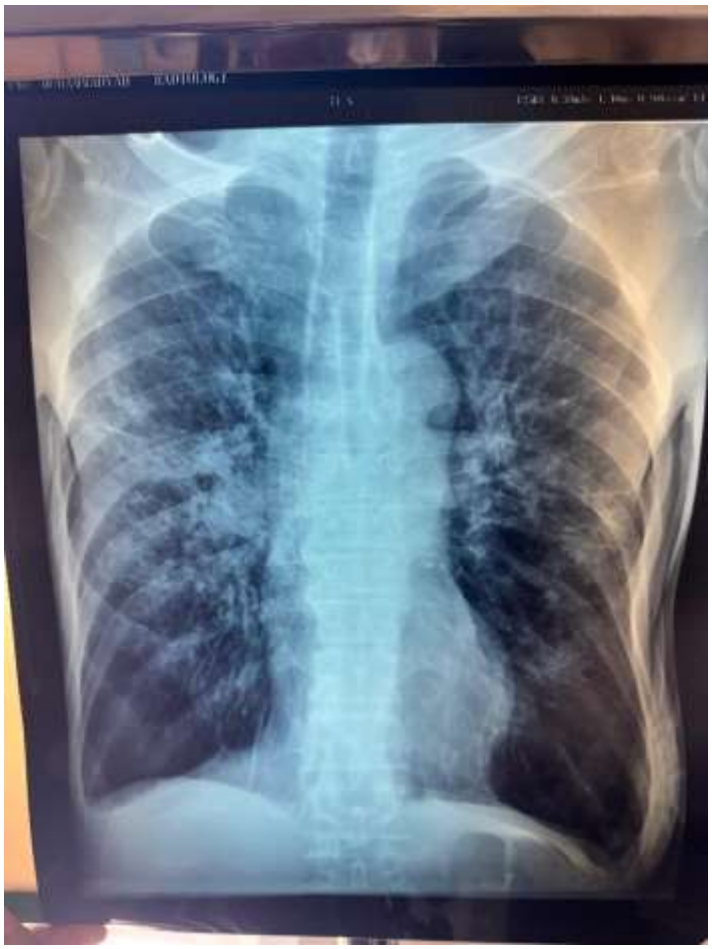

Figure 1. Anteroposterior Chest Radiograph

- **Laboratory and Microbiology:**
  - **Complete Blood Count:** Showed a hemoglobin of 12.1 g/dL, hematocrit of 36.2%, platelet count of 312,000/mm³, and a white blood cell count of 7,236/mm³. The differential count was unremarkable, with an eosinophil count of 1%. The absence of polycythemia, despite chronic hypoxemia, was noted.
  - **GeneXpert MTB/RIF (TCM):** A repeat sputum test was performed to rule out reactivation of tuberculosis as a cause for his deterioration; the result was negative.
  - **Random Blood Glucose:** 82 mg/dL, ruling out hyperglycemia.

Clinical Course, Diagnosis, and Management

At the primary care clinic, the patient was stabilized with supplemental oxygen at 2 L/minute via nasal cannula. He received three sequential nebulizations with a combination of a short-acting beta-agonist and a short-acting muscinarinic antagonist (ipratropium bromide + salbutamol sulfate) at 20-minute intervals. Following this initial treatment, his SpO2 improved modestly to 93% and he reported a subjective reduction in his sensation of breathlessness.

Given the severity of his underlying disease and the acute exacerbation, he was referred to the Pulmonology Clinic at Bima Regency General Hospital for specialist evaluation and long-term management. Based on the comprehensive clinical picture, the constellation of risk factors, and the definitive spirometry results, a final diagnosis was established: **COPD, GOLD Stage 4, Group E, in acute exacerbation, with a mixed etiology of tobacco smoke, occupational dust exposure, and post-tuberculosis sequelae.**

The patient's initial maintenance therapy of only oral bronchodilators was recognized as critically suboptimal for his disease severity. This reflects a common and significant gap in the translation of evidence-based guidelines into primary care, where the severity of COPD can be underestimated and the cornerstone of modern therapy—long-acting inhaled bronchodilators—is often underutilized. The failure to initiate appropriate guideline-directed inhaled therapy after his initial diagnosis in 2023 may have contributed to a lack of disease control and potentially accelerated his functional decline, culminating in this severe presentation.

His long-term management plan was revised accordingly. He was discharged on a combination inhaler containing a long-acting beta-agonist and an inhaled corticosteroid (Salmeterol/Fluticasone), prescribed at a dose of two puffs twice daily. He was scheduled for regular monthly follow-up at the pulmonology clinic to monitor his progress and adjust therapy as needed. Crucially, he was also given a referral for a comprehensive pulmonary rehabilitation program to address his profound functional limitations.

DISCUSSION

Diagnosis Confirmation and Severity Stratification according to GOLD 2024

The diagnosis of COPD in this patient is unequivocally confirmed by post-bronchodilator spirometry, which demonstrated an FEV1/FVC ratio of 0.337, well below the diagnostic threshold of <0.70 (Global Initiative for Chronic Obstructive Lung Disease, 2024). The severity of his condition can be stratified using the comprehensive framework provided by the GOLD 2024 report (Global Initiative for Chronic Obstructive Lung Disease, 2024).

- **Severity of Airflow Limitation:** The patient's post-bronchodilator FEV1 was only 23% of the predicted value. According to the GOLD classification of airflow limitation severity, an FEV1 <30% predicted places a patient in **GOLD Stage 4: Very Severe Airflow Limitation** (Global Initiative for Chronic Obstructive Lung Disease, 2024). This represents the most advanced stage of physiological impairment.
- **Symptom Burden and Exacerbation Risk (ABE Assessment Model):** The GOLD strategy has moved beyond FEV1 alone to incorporate symptom burden and exacerbation history for guiding therapy. This is known as the "ABE" assessment model. The patient's CAT score of 36 is far above the threshold of ≥10, indicating a very high symptom burden. His hospitalization in April 2024 for what was ultimately diagnosed as tuberculosis, but presented as a severe respiratory decompensation, qualifies as a high-risk exacerbation event (≥1 hospitalization in the past year). A patient with a high symptom burden and a high risk of future exacerbations is classified as **Group E** (Global Initiative for Chronic Obstructive Lung Disease, 2024). This classification is critical as it identifies him as being at the highest risk for poor outcomes and places him in the therapeutic category requiring the most aggressive management strategies.

Deconstructing the Pathophysiology: A Tripartite Etiology

The profound respiratory failure in this patient cannot be attributed to a single cause. Rather, it is the culmination of three distinct, yet synergistic, pathological processes that have unfolded over

decades.

The Role of Tobacco Smoke

The patient's extensive smoking history of 38 to 57 pack-years is a potent driver of COPD. Chronic inhalation of cigarette smoke triggers a cascade of inflammatory responses in the lung, characterized by the infiltration of neutrophils, macrophages, and lymphocytes. This chronic inflammation leads to a protease-antiprotease imbalance, where enzymes like neutrophil elastase overwhelm the lung's protective antiproteases (e.g., alpha-1 antitrypsin), resulting in the progressive destruction of alveolar walls and the development of emphysema. Concurrently, oxidative stress from the countless free radicals in tobacco smoke damages cellular structures and perpetuates the inflammatory cycle, while also contributing to mucus hypersecretion and small airway remodeling (chronic bronchitis). This mechanism alone is sufficient to cause severe COPD.

The Impact of Occupational Exposure

For 40 years, the patient worked in an environment laden with inorganic dusts, a known and significant cause of occupational lung disease. The American Thoracic Society estimates that workplace exposures may be responsible for as much as 14% of all COPD cases (Occupational Cancer Research Centre, n.d.). Construction workers are at particularly high risk due to exposure to cement dust, concrete, silica, and asbestos fibers (Occupational Cancer Research Centre, n.d.). A large cohort study of Swedish construction workers demonstrated that exposure to inorganic dust was associated with a significantly increased risk of mortality from COPD (Hazard Ratio 1.10). The effect was even more dramatic among never-smokers (HR 2.30), highlighting dust as a potent, independent pathogen (Torén, Järvholm and Bergdahl, 2004). These inhaled particles incite a chronic inflammatory and fibrotic response in the lungs, leading to small airways disease (industrial bronchitis) and parenchymal fibrosis (pneumoconiosis), which contribute to fixed airflow obstruction and reduced lung compliance. This occupational exposure added a substantial burden of non-emphysematous, fibrotic lung injury to his underlying smoking-related damage.

The Legacy of Tuberculosis (Post-Tuberculosis Obstructive Pulmonary Disease - PTOPD/SOPT)

Tuberculosis is not merely a transient infection; it is an architectural catastrophe for the lungs. The host's immune response to *Mycobacterium tuberculosis*, while necessary for containment, results in granuloma formation and caseous necrosis. The subsequent healing process is often imperfect and pathologically exuberant, leading to permanent structural damage. This damage, collectively known as PTLD, frequently includes dense parenchymal fibrosis, scarring, volume loss, and the formation of traction bronchiectasis, where scarred lung tissue pulls airways open, distorting their anatomy and impairing mucus clearance (van Zyl-Smit, Allwood and Stick, 2020; Amaral et al., 2015; Mbongue et al., 2023). These structural changes create regions of severe, fixed airflow limitation and ventilation-perfusion mismatch.

Crucially, studies comparing TB-associated COPD to smoking-induced COPD have identified key differences. TB-associated disease is often characterized by significantly lower FVC and post-bronchodilator FEV1, and a markedly reduced response to bronchodilators, indicating a more profound and irreversible component of obstruction (Mbongue et al., 2023). The patient's history of hemoptysis and significant weight loss during his active TB infection suggests extensive parenchymal disease, a known predictor for the development of severe post-TB sequelae and subsequent lung function impairment (Mbongue et al., 2023).

The Patient's Unique Clinical Phenotype: A "Triple Hit" Model

The exceptional severity of this patient's condition at the relatively young age of 56 is best explained by a synergistic "triple hit" model, where each pathological process amplified the damage caused by the others.

1. **First Hit (Smoking):** Decades of heavy smoking established the initial foundation of lung injury. It caused widespread chronic inflammation, initiated emphysematous destruction of the lung parenchyma, and impaired mucociliary clearance, creating a lung environment that was

highly susceptible to further insults.

2. **Second Hit (Tuberculosis):** The pre-existing, smoking-induced lung damage likely increased the patient's susceptibility to developing a severe form of pulmonary tuberculosis. The TB infection then acted as an acute, focal, and devastating insult. It superimposed areas of intense destruction, cavitation, and subsequent dense fibrosis onto the diffusely damaged, emphysematous lung. This created a hybrid pathology of both parenchymal loss (emphysema) and parenchymal scarring (fibrosis), leading to severe, fixed obstruction and architectural distortion, including probable traction bronchiectasis.
3. **Third Hit (Occupational Dust):** For 40 years, the patient inhaled a continuous stream of inorganic dust into lungs already ravaged by smoking and scarred by tuberculosis. This chronic, low-grade inflammatory stimulus acted as an accelerant. It likely exacerbated the existing inflammatory pathways, promoted further fibrotic remodeling in both the airways and interstitium, and contributed significantly to small airways disease. This final, prolonged insult effectively "cemented" the irreversible nature of his airflow obstruction.

This step-wise accumulation of damage from three distinct sources provides a compelling explanation for his clinical outcome: a very severe, poorly reversible obstructive physiology that is far more profound than what would be anticipated from smoking, occupational exposure, or prior tuberculosis alone.

Rationale for the Therapeutic Strategy

The management plan for this patient must address his high symptom burden, high exacerbation risk, and severe underlying physiological impairment, consistent with his classification as GOLD Group E.

- **Pharmacological Management:** The GOLD 2024 guidelines recommend initiating therapy with a long-acting beta-agonist and long-acting muscarinic antagonist (LABA+LAMA) combination for Group E patients (Global Initiative for Chronic Obstructive Lung Disease,

2024; Agusti et al., 2023). The addition of an inhaled corticosteroid (ICS) to create triple therapy (LABA+LAMA+ICS) is recommended for patients with a blood eosinophil count ≥300 cells/μL or a history of asthma. The patient's discharge medication was a LABA/ICS combination (Salmeterol/Fluticasone). While his eosinophil count was low (1%), this choice can be justified in the context of his extreme symptom severity and high exacerbation risk. However, the addition of a LAMA to achieve triple therapy would be a primary consideration at his first follow-up visit to maximize bronchodilation, as this is the cornerstone of treatment for severe COPD.

- **Non-Pharmacological Management:** For a patient with such severe functional limitations, non-pharmacological interventions are arguably more important than medication.
    - **Pulmonary Rehabilitation:** The referral for pulmonary rehabilitation is the single most critical and effective intervention. Comprehensive pulmonary rehabilitation programs, which include structured exercise training, disease education, and psychosocial support, have been proven to improve dyspnea, increase exercise capacity, enhance quality of life, and reduce hospitalizations in patients with severe COPD (Global Initiative for Chronic Obstructive Lung Disease, 2024; Agusti et al., 2023). Furthermore, recently published international clinical standards for PTLD specifically advocate for pulmonary rehabilitation as a core component of management for symptomatic survivors (Raff and Kirenga, 2023).
    - **Smoking Cessation:** Although the patient had recently quit, continuous reinforcement and support for this cessation are vital to slow the rate of further decline in lung function.
    - **Vaccination:** To mitigate the risk of future exacerbations, which could be life-threatening in his condition, it is imperative that he receive vaccinations against influenza, pneumococcus, and COVID-19, as recommended by global guidelines (Global Initiative for Chronic Obstructive Lung Disease, 2024; Agusti et al., 2023).

CONCLUSION

Concluding Summary

This report details the case of a 56-year-old male with very severe (GOLD 4, Group E) COPD. His profound and largely irreversible airflow obstruction is the cumulative result of a triple burden of lifelong risk factors: a heavy smoking history, four decades of occupational exposure to inorganic dust as a construction worker, and the destructive structural sequelae of a prior pulmonary tuberculosis infection. The patient's clinical presentation, physiological derangements, and radiological findings coalesce to form a compelling picture of mixed-etiology COPD. This case serves as a stark illustration of how the convergence of multiple distinct pathogenic pathways—inflammatory, destructive, and fibrotic—can synergize to produce an exceptionally severe clinical phenotype.

Recommendations

Based on the analysis of this complex case, the following recommendations are proposed:

- **For Clinical Practice:** Clinicians must maintain a high index of suspicion for COPD in all patients with a history of tuberculosis, regardless of their smoking status. An integral part of any respiratory assessment must be a comprehensive, lifelong occupational history. In line with emerging international standards, routine post-bronchodilator spirometry should be strongly considered for all tuberculosis survivors at the completion of their treatment to establish a pulmonary function baseline and screen for the presence of PTLD (Meghji et al., 2021; Raff and Kirenga, 2023). Early identification of airflow obstruction in this high-risk group can facilitate timely intervention.
- **For Public Health:** Public health initiatives must adopt a multi-pronged approach. Primary prevention efforts should continue to focus on aggressive smoking cessation campaigns and the enforcement of workplace safety regulations to minimize hazardous dust exposure in industries like construction. Simultaneously, secondary prevention strategies should be developed,

including active case-finding programs for COPD that target high-risk populations, such as known tuberculosis survivors and workers in hazardous occupations.

- **For Future Research:** This case highlights the need for further investigation into the COPD-PTLD overlap phenotype. Future research should aim to better characterize the clinical, physiological, and radiological features of this patient population. Studies exploring the specific molecular and cellular mechanisms of synergy between tobacco smoke, inorganic dust, and post-tuberculosis lung damage are needed. Ultimately, prospective clinical trials are required to determine the optimal pharmacological and non-pharmacological therapeutic strategies for this complex and highly morbid patient group.

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