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COPD in a Construction Worker with Heavy Smoking History: Application of the PERDOKI Seven-Step Work-Relatedness Assessment

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Abstract

Chronic obstructive pulmonary disease (COPD) is a multifactorial disease in which tobacco smoking remains the dominant risk factor, while occupational exposure to vapours, gases, dusts, and fumes may contribute to disease development and progression. This case report describes a 65-year-old male construction labourer with a 48-pack-year smoking history and more than 30 years of unprotected occupational exposure to cement dust, sand, road dust, and probable respirable crystalline silica. He presented with worsening dyspnea and a productive cough consistent with an acute exacerbation of COPD. Spirometry showed airflow obstruction with FEV1/FVC of 59% and FEV1 of 60% predicted, corresponding to GOLD 2 airflow obstruction if confirmed by post-bronchodilator spirometry. Chest radiography demonstrated emphysematous changes, including bilateral hyperinflation, flattened diaphragms, attenuated vascular markings, and a vertical cardiac silhouette. Using the seven-step work-relatedness assessment of the Indonesian Association of Occupational Medicine Specialists (PERDOKI), occupational exposure was interpreted as a possible contributing factor rather than a definitive or sole cause, given the patient's substantial smoking history and the absence of quantitative exposure monitoring. Management combined guideline-based COPD pharmacotherapy, smoking abstinence, workplace exposure reduction, respiratory protection, work modification, pulmonary rehabilitation referral, and periodic occupational health surveillance. This case highlights the importance of systematic occupational history taking in COPD patients, especially among construction workers with dual exposure to smoking and mineral dust.

Keywords: COPD, Construction Worker, Silica Exposure, Cement Dust, Work-Relatedness, Smoking

1. Introduction

Chronic obstructive pulmonary disease (COPD) is a major global cause of morbidity and mortality. The World Health Organization reported that COPD was the third leading cause of death worldwide and caused approximately 3.23 million deaths in 2019.[1] More recent regional evidence from the Global Burden of Disease (GBD) 2021 study shows that COPD remains a substantial problem in Asia, causing approximately 2.89 million deaths and 60.51 million disability-adjusted life-years (DALYs) in 2021.[2] For Indonesia, recent nationally representative spirometry-based prevalence estimates remain limited; however, the high prevalence of tobacco exposure among Indonesian men and continued exposure to air pollution and workplace dust support the relevance of COPD prevention and occupational risk assessment.[2]

Tobacco smoking is the most established risk factor for COPD, but it does not fully explain all cases. The Global Initiative for Chronic Obstructive Lung Disease (GOLD) defines COPD as a heterogeneous lung condition caused by abnormalities of the airways and/or alveoli, usually resulting from significant exposure to noxious particles or gases and influenced by host factors.[3] Occupational exposure to vapors, gases, dusts, and fumes is increasingly recognized as an important contributor to COPD. Recent reviews estimate that workplace exposure accounts for approximately 14% of the population-attributable fraction of COPD, with a higher attributable fraction reported among never-smokers.[4] The ATS/ERS statement on nonmalignant respiratory diseases similarly emphasizes that occupational exposure contributes to COPD and chronic bronchitis at the population level.[5]

Construction work is particularly relevant because workers may inhale mixed inorganic dusts from cement, sand, concrete, road dust, stone cutting, demolition, and other activities that may generate respirable crystalline silica. A systematic review reported that COPD occurs more frequently among construction workers than among workers without construction dust exposure.[6] Updated analyses among older construction workers also demonstrated an increased COPD risk compared with non-construction workers.[7] Silica exposure is biologically plausible as a contributor to chronic bronchitis, emphysema, small-airway disease, and fixed airflow obstruction, even without classic radiological silicosis.[8,9] Cement dust and other inorganic particles may further contribute to respiratory symptoms and reduced lung function.[4]

The diagnostic challenge becomes more complex when a patient has both heavy smoking and long-term occupational dust exposure. In such cases, a categorical conclusion that COPD is caused by occupation alone may be inappropriate. However, failure to assess occupational exposure may lead to under-recognition of work-related COPD, delayed workplace intervention, and ongoing lung function decline.[4] The gap addressed by this case report is the limited practical illustration of how occupational-medicine reasoning can be applied to COPD cases in Indonesian construction workers when objective dust measurements are unavailable, and smoking is a major competing risk factor. Therefore, this case report aims to describe a COPD case in a construction worker with a heavy smoking history and to demonstrate how the PERDOKI seven-step work-relatedness assessment can support a balanced conclusion regarding possible occupational contribution without overstating causality.[10,11]

2. Case Presentation

A 65-year-old male construction laborer presented with worsening shortness of breath that began one day before hospital admission and a productive cough for one week. The sputum had changed from white to thick yellow and was accompanied by malaise and mild fever. He denied chest pain, significant weight loss, and known contact with individuals with tuberculosis. He had previously been diagnosed with COPD, but he was not receiving regular maintenance treatment at the time of presentation. No drug allergy or significant family history of respiratory disease was reported. Information on the exact duration of chronic respiratory symptoms before this exacerbation, the

number of prior exacerbations, previous hospitalizations, prior inhaler use, oxygen saturation, arterial blood gas analysis, complete blood count, and inflammatory markers was not available in the source clinical record and is therefore acknowledged as a limitation of this case report.

The patient had a major smoking history of approximately 48 pack-years, having smoked two packs per day for 48 years before stopping one year before presentation. Occupationally, he had worked as a construction laborer since 1989, accumulating more than 30 years of experience. His routine tasks included mixing cement, laying bricks, installing roofs, carrying materials, and working near road dust. A typical workday lasted approximately nine hours with a one-hour break. The work environment involved repeated exposure to cement dust, sand, road dust, outdoor heat, ultraviolet radiation, and ergonomic hazards such as heavy lifting, repetitive movement, and awkward posture. The patient reported that he had not consistently used respiratory protective equipment during his employment. Quantitative personal dust monitoring and cumulative exposure estimates were not available.

On physical examination, the patient was hemodynamically stable, with blood pressure of 110/70 mmHg, heart rate of 84 beats per minute, respiratory rate of 22 breaths per minute, and body temperature of 36.7°C. Lung auscultation revealed vesicular breath sounds with coarse rhonchi and prolonged expiration bilaterally. Other systemic examinations were unremarkable.

Spirometry demonstrated an FEV1 of 60% predicted, FVC of 71% predicted, and FEV1/FVC ratio of 59%, indicating an obstructive ventilatory pattern. Based on GOLD criteria, an FEV1 of 60% predicted would be compatible with GOLD 2 moderate airflow obstruction if the obstruction is confirmed using post-bronchodilator spirometry.[3] The term “restrictive component” was avoided because restriction cannot be established from simple spirometry alone without total lung capacity measurement. No bronchodilator reversibility test was documented; therefore, asthma-COPD overlap could not be fully assessed.

Chest X-ray showed emphysematous changes, including bilateral hyperinflation, increased retrosternal airspace, hyperlucency, flattened diaphragms, attenuated peripheral vascular markings, and a vertical cardiac silhouette.

These findings were consistent with COPD/emphysematous change. No radiological evidence suggestive of active pulmonary tuberculosis, focal pneumonia, pleural effusion, or overt pulmonary fibrosis was reported in the available record. However, high-resolution computed tomography was not performed, limiting the ability to evaluate emphysema distribution, occult interstitial lung disease, or pneumoconiosis-related abnormalities.

Based on the clinical presentation, smoking history, spirometry obstruction, and radiographic emphysematous changes, the patient was diagnosed with an acute exacerbation of COPD with GOLD 2 airflow obstruction when interpreted from available spirometry. Occupational exposure to cement dust, sand, road dust, and probable respirable crystalline silica was considered a possible contributing factor rather than a definitive or exclusive cause, given the patient’s heavy smoking history and the absence of quantitative exposure assessment.

Management included pharmacological therapy with tiotropium 18 mcg once daily, budesonide/formoterol 160/4.5 mcg twice daily, and salbutamol as needed for symptom relief. Non-pharmacological management emphasized continued smoking abstinence, avoidance of dust exposure, adequate rest, vaccination against influenza and pneumococcal infection, pulmonologist referral, and functional capacity assessment using the six-minute walk test. Pulmonary rehabilitation was recommended because it can improve exercise tolerance and symptom control in COPD patients.[3]

From an occupational health perspective, the patient was considered fit for work with conditions. Recommended workplace interventions included dust suppression by wet methods, use of pre-mixed materials where feasible, improved ventilation, task modification to reduce heavy lifting and roofing activities, scheduled rest breaks, and mandatory respiratory protection. A properly fitted particulate respirator, such as an FFP2/FFP3 or equivalent respirator, depending on exposure assessment and local standards, was recommended. Regular occupational health follow-up, including annual spirometry, symptom monitoring, and reassessment of fitness for work, was also advised.

Table 1. Timeline of Clinical and Occupational Exposure History Relevant to COPD Diagnosis

Time/Period	Clinical and Occupational Event	Relevance to Diagnosis
1989 onward	Began work as a construction labourer with repeated exposure to cement dust, sand, road dust, and physically demanding tasks.	Long latency and cumulative inhalational exposure support the plausibility of occupational contribution.
1989–presentation	Worked approximately nine hours daily with limited or absent respiratory protective equipment.	Suggests prolonged unprotected exposure; quantitative dust measurement was unavailable.
48 years before cessation	Smoked approximately two packs per day, equivalent to 48 pack-years.	Major non-occupational COPD risk factor and important confounder in causal attribution.
One year before presentation	Stopped smoking.	Supports risk-reduction efforts but does not eliminate prior cumulative smoking risk.
One week before admission	Productive cough with sputum changing from white to thick yellow, malaise, and mild fever.	Compatible with an infective or inflammatory trigger of COPD exacerbation.
One day before admission	Worsening dyspnoea.	Reason for acute presentation.
At presentation	Spirometry: FEV1/FVC 59%, FEV1 60% predicted, FVC 71% predicted; chest X-ray with emphysematous changes.	Supports COPD with moderate airflow obstruction if post-bronchodilator obstruction is confirmed.

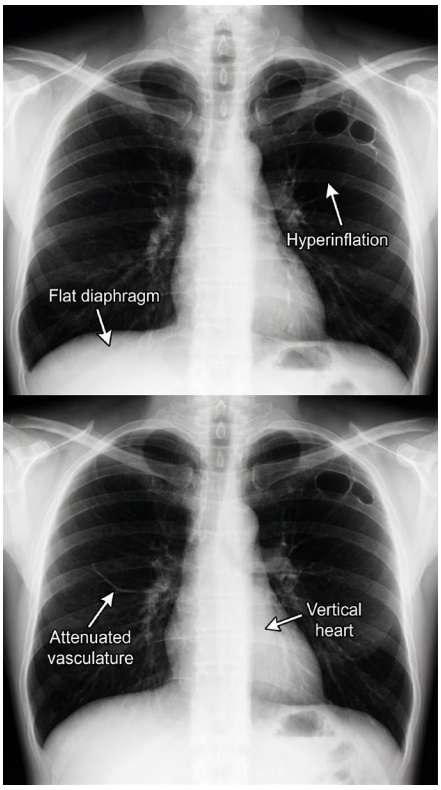

Figure 1. Chest X-ray (PA view) demonstrating emphysematous changes. Annotated findings include bilateral hyperinflation, increased retrosternal airspace, hyperlucency, flattened diaphragms, attenuated vascular markings, and a vertical cardiac silhouette.

PERDOKI refers to Perhimpunan Spesialis Kedokteran Okupasi Indonesia, the Indonesian Association of Occupational Medicine Specialists. In Indonesian occupational medicine practice, the PERDOKI seven-step approach is used to structure the assessment of whether a disease is work-related or may be classified as an occupational disease.[10,11] In this case report, the framework was used as a work-relatedness assessment rather than as definitive proof of occupational causation. This distinction was important because the patient had two major exposure domains: heavy tobacco smoking and long-term occupational mineral dust exposure. The framework allowed a cautious conclusion that occupational exposure may have contributed to disease development or progression, without overstating causality.

Table 2. Application of the PERDOKI Occupational Disease Diagnostic Steps in the COPD Case Assessment

PERDOKI Step	Case-Based Assessment	Interpretation
1. Establish clinical diagnosis	Symptoms, physical examination, spirometry, and chest X-ray supported COPD with acute exacerbation.	COPD diagnosis was clinically plausible; post-bronchodilator confirmation was not documented.

2. Identify occupational exposure	More than 30 years of construction work with cement dust, sand, road dust, probable silica, and inadequate respiratory protection.	Exposure was relevant and prolonged.
3. Assess the relationship between exposure and disease	COPD developed after decades of work and smoking exposure.	The temporal relationship was compatible with chronic occupational contribution.
4. Review epidemiological evidence	Literature supports increased COPD risk among construction workers and workers exposed to VGDF, silica, and inorganic dust.[4,5,6,7,8]	External evidence supports plausibility.
5. Evaluate individual susceptibility/risk	Age and 48-pack-year smoking history were major risk factors.	Smoking was a strong confounder and likely primary contributor.
6. Consider non-occupational causes and differential diagnosis	No reported TB contact, weight loss, focal pneumonia, pleural effusion, or overt fibrosis on X-ray; asthma-COPD overlap could not be excluded without reversibility testing.	Alternative diagnoses were partly addressed but not fully excluded due to limited investigations.
7. Determine work-relatedness	Work exposure was considered a possible contributing factor, not a sole or definitive cause.	A balanced work-relatedness conclusion was most appropriate because smoking was a major confounder, and objective exposure assessment was unavailable.

3. Discussion

This case demonstrates a frequent but challenging scenario in occupational medicine: COPD in a worker with both heavy smoking and long-term workplace dust exposure. The patient's 48-pack-year smoking history represents a strong causal risk factor. Therefore, it would be inappropriate to claim that occupational exposure was the sole cause of COPD. Nevertheless, dismissing the workplace contribution would also be inappropriate because construction-related exposure to cement dust, sand, road dust, and respirable crystalline silica is biologically plausible and epidemiologically supported.

The central issue in this case is attribution. COPD has no clinical phenotype that reliably distinguishes occupational COPD from smoking-related COPD. The ATS statement notes that occupational COPD is rarely diagnosed because COPD develops slowly, is multifactorial, and many affected workers have simultaneous exposure to cigarette smoke and workplace irritants.[12] This diagnostic uncertainty is directly relevant here. The PERDOKI framework, therefore,

supports a probabilistic conclusion: occupational exposure was a possible contributing factor, while smoking remained a major competing and potentially synergistic risk factor. Systematic reviews of occupational COPD also support the role of occupational exposures and job-exposure assessment approaches in COPD risk attribution.[13,14]

Construction workers are an important group for COPD surveillance, as increased COPD mortality has been reported among construction workers exposed to inorganic dust.[15] A systematic review concluded that COPD occurs more frequently among construction workers than among workers not exposed to construction dust.[6] A later analysis among older construction workers found significantly increased odds of COPD compared with non-construction workers.[7] The relevant exposure is not a single agent but a mixture of inorganic dusts and irritants. Cement handling, brick work, demolition, road dust, and roof work may generate respirable particles. Silica-containing dust is particularly important because epidemiological and pathological evidence links silica exposure to chronic bronchitis, emphysema, small-airway disease, and airflow obstruction even in the absence of radiological silicosis.[8,9] Cement dust exposure has also been associated with airway symptoms, lung-function changes, and broader respiratory health hazards among exposed workers.[16,17]

Smoking and occupational dust exposure may interact biologically. Smoking induces airway inflammation, oxidative stress, impaired mucociliary clearance, and parenchymal destruction. Occupational mineral dust may further stimulate macrophage activation, neutrophilic inflammation, cytokine release, protease-antiprotease imbalance, and airway remodelling.[18] In exposed workers, these mechanisms may accelerate the decline of FEV1 and increase susceptibility to exacerbations. In this case, the patient's emphysematous chest X-ray findings and obstructive spirometry are consistent with COPD, while the long latency of exposure supports biological plausibility. However, without serial spirometry, HRCT, quantitative dust exposure data, or biomarker evidence, the relative contribution of smoking and work cannot be precisely quantified.

The available spirometry showed FEV1/FVC of 59% and FEV1 of 60% predicted, indicating obstruction. Under GOLD, the diagnosis of COPD requires post-bronchodilator FEV1/FVC <0.[7] in an appropriate clinical context, and airflow obstruction severity is graded by post-bronchodilator FEV1.[3] Therefore, the patient may be described as having COPD with GOLD 2 airflow obstruction if the result represents post-bronchodilator spirometry. Because bronchodilator reversibility testing was not documented, the diagnosis should be framed cautiously. This limitation is important because asthma-COPD overlap and partially reversible obstruction cannot be completely excluded. This cautious interpretation is consistent with Indonesian PDPI guidance, which also emphasizes spirometry-based diagnosis and structured clinical management of COPD/PPOK.[19]

The imaging findings supported emphysematous change. Hyperinflation, increased retrosternal airspace, hyperlucency, flattened diaphragms, attenuated vascular markings, and a vertical cardiac silhouette are compatible with COPD/emphysema. Nonetheless, chest radiography is less sensitive than HRCT for

characterizing emphysema, airway disease, pneumoconiosis, or early interstitial abnormalities, including silica-related lung disease.[9] Therefore, HRCT would be useful if clinically indicated, especially in a construction worker with probable silica exposure and chronic respiratory disease.

Management should address both disease control and exposure control. Guideline-based COPD therapy, smoking abstinence, vaccination, inhaler technique education, and pulmonary rehabilitation are central to clinical care.[3,19] However, occupational COPD management also requires prevention of ongoing exposure. Workplace interventions should follow the hierarchy of controls: elimination or substitution where possible, engineering controls such as wet methods and local exhaust ventilation, administrative controls such as task rotation and rest breaks, and personal respiratory protection as the last line of defence.[20,21] In this case, continued construction work may be permissible only with exposure reduction, appropriate respirator selection and fit, avoidance of high-dust tasks, and periodic occupational health surveillance.

The medico-legal implication is that work-relatedness should be expressed in cautious but clinically meaningful terms. A conclusion of "possible occupational contribution" is more defensible than a definitive causal statement because heavy smoking is a strong confounder, and quantitative exposure data were unavailable. This balanced approach helps protect the patient from further exposure while avoiding overstatement in a single case report.

This case report has several limitations. First, no quantitative occupational exposure assessment was available, so cumulative silica or cement dust exposure could not be estimated. Second, the patient's heavy smoking history made it impossible to separate the independent effect of smoking from occupational exposure. Third, post-bronchodilator spirometry and bronchodilator reversibility testing were not documented, limiting assessment of asthma-COPD overlap and formal GOLD confirmation. Fourth, oxygen saturation, arterial blood gas analysis, complete blood count, C-reactive protein, sputum microbiology, and prior exacerbation frequency were not available. Fifth, HRCT and lung volume testing were not performed, limiting evaluation for emphysema distribution, pneumoconiosis, occult fibrosis, and true restriction. Finally, longitudinal spirometry was unavailable, so the rate of lung function decline could not be assessed.

4. Conclusion

This case highlights the importance of routine occupational history taking in all COPD patients, particularly those working in high-dust industries such as construction. In a patient with a 48-pack-year smoking history, occupational exposure should not be overstated as the sole cause of disease. However, more than 30 years of unprotected exposure to cement dust, sand, road dust, and probable silica represents a biologically plausible contributor to COPD development or progression. The PERDOKI seven-step work-relatedness assessment provides a structured method for evaluating possible occupational contribution while acknowledging diagnostic uncertainty. Clinically, the key lesson is that COPD management should combine smoking cessation, guideline-based pharmacotherapy, vaccination, pulmonary rehabilitation, and

systematic workplace intervention to prevent further decline in lung function and protect worker health.

5. Consent for Publication

Written informed consent was obtained from the patient for the publication of clinical details and any accompanying images.

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