



Occupational Community-Acquired Pneumonia in a Fitter-Splicer with a Significant Smoking History: A Case Report and Expanded Review of the Literature

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ABSTRACT

Introduction: Community-acquired pneumonia (CAP) is a primary driver of global morbidity and mortality, with its incidence heavily influenced by host and environmental factors. While the roles of age, comorbidities, and smoking are well-established, the contribution of occupational exposures—particularly the complex and synergistic hazards faced by industrial workers—remains a critical and often under-appreciated component of risk assessment. This report presents an in-depth analysis of a fitter-splicer, a profession with a unique combination of respiratory hazards, who developed CAP, providing a platform to explore the intricate relationship between workplace environment, lifestyle factors, and infectious disease susceptibility.

Case Illustration: A 50-year-old male with a 30-pack-year smoking history presented with a one-week history of a worsening

productive cough, persistent fever, right-sided pleuritic chest pain, and a significant episode of hemoptysis. Physical examination revealed focal findings over the right lower lung, including decreased tactile fremitus, diminished breath sounds, and localized rhonchi, indicative of parenchymal consolidation. Laboratory investigations showed marked neutrophilic leukocytosis and a profoundly elevated C-reactive protein level (165 mg/L). A chest radiograph confirmed dense consolidation in the right lower lobe, consistent with lobar pneumonia. Despite being stratified as low severity by both the CURB-65 and Pneumonia Severity Index (PSI) scores, he was hospitalized due to his clinical presentation and occupational context. He was managed with a dual antibiotic regimen of intravenous ceftriaxone and oral levofloxacin. The patient demonstrated a favorable clinical response and was discharged after a nine-day hospital stay.

Discussion: This case serves as a paradigm for the critical interplay between a patient's multifaceted occupational exposures and his extensive smoking history. The role of a "fitter-splicer" is deconstructed to reveal a dual-threat exposure profile: inhalation of metal fumes and inorganic dusts (e.g., silica) from fitting, grinding, and welding, combined with exposure to chemical vapors and organic dusts from conveyor belt splicing and vulcanization processes. These exposures are known to synergistically impair pulmonary immune defenses. This chronic occupational insult, compounded by decades of smoking-induced lung damage, created a state of profound susceptibility to infection. This decision represents a significant deviation from current evidence-based guidelines, which now strongly recommend against their routine use in non-severe CAP, reserving

them for patients with severe disease characterized by a hyperinflammatory state. The differential diagnoses of tuberculosis and underlying malignancy are explored in detail, emphasizing the necessity of post-discharge radiological follow-up in this high-risk patient.

Conclusion: This case underscores the clinical imperative to obtain a detailed occupational and environmental history in all patients presenting with CAP. It vividly demonstrates how combined industrial exposures and smoking can dramatically amplify pneumonia risk by overwhelming the lung's clearance and immune mechanisms. Furthermore, it highlights the urgent need for robust preventive strategies in high-risk occupations, including the stringent implementation of the hierarchy of controls in the workplace, targeted and accessible smoking cessation programs, and the proactive, guideline-driven administration of pneumococcal vaccination to this vulnerable population.

Keywords: Community-Acquired Pneumonia; Occupational Health; Pneumoconiosis; Fitter; Welding; Smoking; Risk Factors.

INTRODUCTION

Background

Community-acquired pneumonia (CAP) remains a formidable public health challenge, representing a leading cause of hospitalization and mortality from infectious disease worldwide (Johansson et al., 2020; Jain et al., 2015). The etiological landscape of CAP is broad, with *Streptococcus pneumoniae* persisting as the most frequently identified bacterial cause, responsible for up to 50% of cases (Welte et al., 2012; Said et al., 2013; van der Poll & Opal, 2017). This is complemented by a range of "atypical" bacteria such as *Mycoplasma pneumoniae* and *Legionella pneumophila*, and an increasing recognition of respiratory viruses, including influenza and human rhinovirus, as significant pathogens (Jain et al., 2015; Said et al., 2013).

While well-established host factors like advanced age, smoking, alcohol abuse, and the presence of comorbidities (e.g., COPD, diabetes, heart failure) are known to predispose individuals to CAP, the role of the work environment is a crucial, yet often under-recognized, determinant of risk (Almirall et al., 2017; Torres et al., 2013). A substantial body of evidence links chronic occupational inhalation of inorganic dusts (e.g., silica, coal dust), metal fumes (from welding), gases, and chemical vapors to an elevated incidence of respiratory infections, particularly lobar pneumonia (Torén et al., 2020; Wergeland and Iversen, 2001; Palmer et al., 2011). Workers in heavy industries such as mining, construction, and metal fabrication are exposed to a complex milieu of airborne particulates (Hopkins Medicine, 2023; Szram et al., 2013). These exposures can initiate a spectrum of occupational lung diseases (pneumoconioses) and, more fundamentally, compromise the lung's innate and adaptive immune defenses, thereby creating a permissive environment for pathogenic colonization and the development of severe infection (Barnes et al., 2025; Hubbs et al., 2020; Palmer et al., 2003).

Research Gap and Novelty

The association between welding—a process that generates significant metal fume—and an increased risk of pneumococcal pneumonia is well-documented, with some studies showing a six-fold increased likelihood of this specific infection (Coggon et al., 1994; Torén et al., 2020; Wong et al., 2010; Palmer & Cosgrove, 2012). However, the specific risk profile for workers with the job title "fitter-splicer" is not clearly defined in the existing literature. This profession entails a unique and potentially more complex set of respiratory hazards. It combines exposures from metal fitting, grinding, and welding with those from conveyor belt splicing activities, which may involve processes like rubber vulcanization and the use of various solvents and adhesives, releasing a complex mixture of chemical fumes and organic dusts (KSK, 2023; MTP, n.d.; IARC, 1982; Szewczyńska et al., 2002).

This case report seeks to address this gap by providing a detailed clinical narrative and an expanded discussion of a fitter-splicer who developed CAP. The novelty of this report lies in its comprehensive analysis of the patient's dual occupational hazards in synergy with his significant smoking history, offering a model for understanding multifactorial risk in industrial workers. Furthermore, the patient's management provides a valuable opportunity to discuss contemporary and sometimes controversial aspects of CAP treatment (Metlay et al., 2025; Dequin et al., 2023).

Objectives

The primary objectives of this report are:

1. To present a comprehensive clinical account of the diagnosis, management, and clinical course of a 50-year-old male fitter-splicer with CAP.
2. To conduct a focused review of the literature to elucidate the pathophysiological mechanisms by which his specific occupational exposures and long-term smoking may have synergistically increased his susceptibility to pneumonia.
3. To highlight the importance of primary prevention strategies, including occupational health measures and vaccination, in high-risk industrial populations.

CASE ILLUSTRATION

Patient Information

A 50-year-old Indonesian male, employed as a fitter-splicer in a mining operation, presented to a company clinic located in the Southwest Maluku Regency. He reported a significant smoking history of 10 cigarettes per day since the age of 20, corresponding to a 30-pack-year exposure. He denied any history of excessive alcohol consumption. His past medical history was unremarkable for tuberculosis, hypertension, diabetes mellitus, or other chronic cardiopulmonary or renal diseases. He engaged in regular exercise twice per week.

Clinical Presentation and Initial Findings

The patient's chief complaint was a productive cough that had persisted for one week and had become progressively more severe and frequent. He reported a notable episode of coughing reddish-tinged sputum (hemoptysis). This was accompanied by a continuous fever for the three days preceding his presentation. The onset of the cough preceded the fever. He also described associated right-sided pleuritic chest pain and epigastric discomfort. He explicitly denied dyspnea, nausea, or vomiting.

Upon initial physical examination, the patient appeared moderately ill but was fully conscious, alert, and oriented (Glasgow Coma Scale score of 15). His vital signs on admission were recorded as: blood pressure 100/80 mmHg, heart rate 98 beats per minute, respiratory rate 24 breaths per minute, body temperature 37.2°C, and peripheral oxygen saturation (SpO₂) of 96% while breathing ambient air. The cardiovascular examination was normal, with regular S1 and S2 heart sounds and no murmurs or gallops. The abdominal examination revealed increased bowel sounds and mild tenderness to palpation in the epigastric region.

The respiratory examination yielded significant findings highly suggestive of a focal parenchymal consolidation process. Palpation of the chest wall revealed diminished tactile vocal

fremitus over the right lower lung field. On auscultation, vesicular breath sounds were audibly decreased in the right lung base, and adventitious sounds in the form of rhonchi were present on the right side but absent on the left (+/-).

Diagnostic Assessment

A series of diagnostic tests were performed to confirm the diagnosis and assess the severity of the illness.

Laboratory Findings: Initial blood work revealed a significant inflammatory response. The complete blood count (CBC) showed marked leukocytosis with a neutrophilic predominance, These markers were trended throughout the hospitalization and showed a progressive decline, correlating with the patient's clinical improvement (Table 1).

Table 1: Laboratory Findings on Admission and During Hospitalization.

Parameter	Reference Range	Day 1 (13/06)	Day 2 (14/06)	Day 6 (18/06)	Day 9 (21/06)
White Blood Cell (WBC)	3.5-10.0 x10 ³ /μL	33,9	24,1	11.8	9.5
Neutrophils (%)	35-80	91.9	82	75	68
Lymphocyt	15-50	4.1	12	18	25

es (%)					
Hemoglobin (Hgb)	11.5-16.5 g/dL	14.4	13.9	13.5	13.8
Hematocrit (Hct)	35.0-55.0 %	38.8	41	40	41
Platelets (PLT)	130-400 x10 ³ /μL	227	245	260	275
Blood Urea Nitrogen (BUN)	7-20 mg/dL		19	-	-
Creatinine (Cr)	0.6-1.2 mg/dL		1.1	-	-

Radiographic and Electrocardiographic Findings: An initial anteroposterior (AP) chest radiograph was obtained. The imaging revealed features characteristic of lobar pneumonia, specifically a dense, homogeneous opacity consistent with consolidation in the right lower lobe. This opacity obscured the right hemidiaphragm, a classic example of the "silhouette sign," and air-filled bronchi were visible within the consolidation (air bronchograms), confirming an alveolar filling process (Radiopaedia, 2023a; Radiology Assistant, n.d.). A follow-up chest radiograph performed on day 6 of hospitalization showed partial resolution of the infiltrate. An initial

electrocardiogram (ECG) was unremarkable, showing a normal sinus rhythm without evidence of acute ischemia.



Figure 1. An initial Anteroposterior (AP) Chest Radiograph

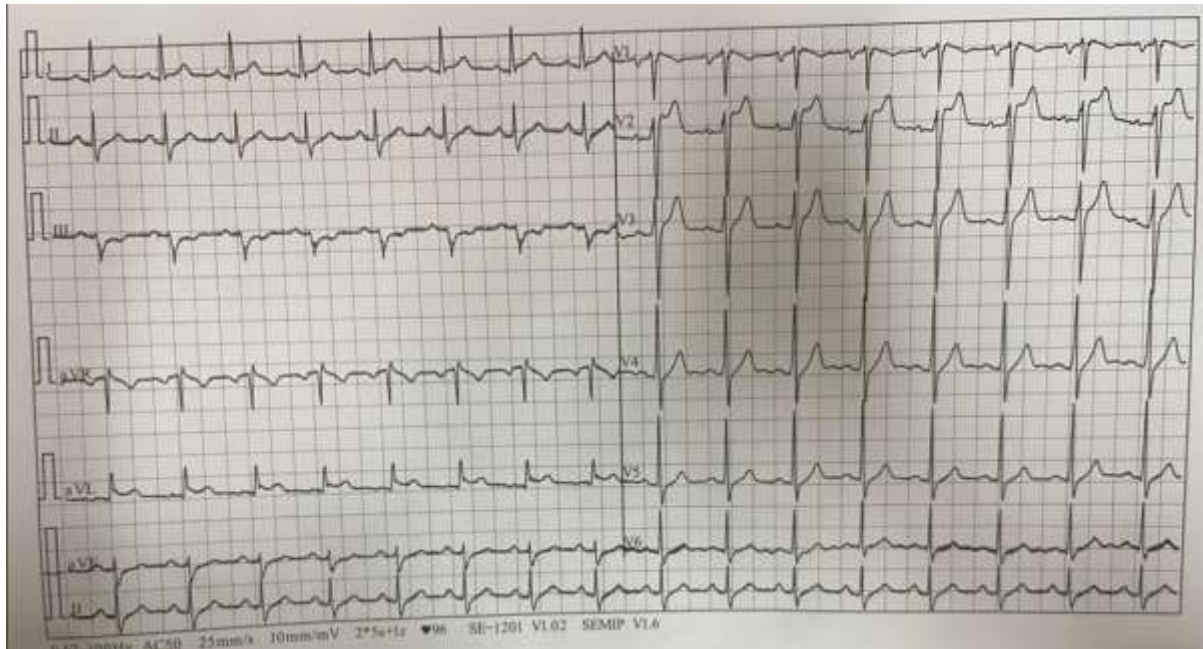


Figure 2. Electrocardiogram

Severity Assessment: To objectively stratify the patient's risk of mortality and guide the site-of-care decision, the CURB-65 and Pneumonia Severity Index (PSI) scores were calculated based on admission data. Both scores classified the patient's pneumonia as low severity, suggesting that outpatient management could have been considered under different circumstances (Table 2).

Table 2: Pneumonia Severity Assessment on Admission (CURB-65 and PSI Scores).

Score Component	Patient Value	Points Awarded
CURB-65 Score		
Confusion	No (Compos Mentis)	0

Urea (BUN > 19-20 mg/dL)	No (18 mg/dL)	0
Respiratory Rate (≥ 30 /min)	No (24/min)	0
Blood Pressure (SBP <90 or DBP ≤ 60)	No (100/80 mmHg)	0
Age (≥ 65 years)	No (50 years)	0
Total CURB-65 Score		0
<i>30-day Mortality Risk: 0.6-1.5% (Low Risk)</i>		
Pneumonia Severity Index (PSI) (Fine et al., 1997)		
Age	50 years	+50
Male Sex	Yes	(Age in years)
Coexisting Illnesses	None	0

Respiratory Rate $\geq 30/\text{min}$	No (24/min)	0
SBP < 90 mmHg	No (100 mmHg)	0
Temperature <35°C or 40°C	No (37.2°C)	0
Altered Mental Status	No	0
Pulse $\geq 125/\text{min}$	No (98/min)	0
Arterial pH <7.35	Not measured (assumed normal)	0
BUN $\geq 30 \text{ mg/dL}$	No (18 mg/dL)	0
Sodium <130 mEq/L	Not measured (assumed normal)	0
Glucose $\geq 250 \text{ mg/dL}$	Not measured (assumed normal)	0
Hematocrit <30%	No (42%)	0

PaO ₂ < 60 mmHg or SpO ₂ < 90%	No (SpO ₂ 96%)	0
Pleural Effusion	Not specified (assumed no)	0
Total PSI Score		50
<i>Risk Class: I (Mortality Risk: 0.1%)</i>		

Therapeutic Management and Hospital Course

The patient was admitted with working diagnoses of suspected pneumonia and dyspepsia syndrome. A multi-modal treatment plan was initiated, comprising ¹:

- **Fluid Resuscitation:** Intravenous Ringer's Lactate solution at 20 drops per minute.
- **Antimicrobial Therapy:** A dual antibiotic regimen of intravenous Ceftriaxone (2 grams every 12 hours) and oral Levofloxacin (750 mg once daily).
- **Supportive and Symptomatic Care:** Supplemental oxygen via nasal cannula at 2 L/min, intravenous Paracetamol (1 gram every 8 hours) for fever, intravenous Ranitidine (25 mg every 12 hours) and oral Sucralfate syrup for dyspepsia, and oral Acetylcysteine (200 mg three times daily) as a mucolytic. Intravenous Ketorolac was administered as needed for chest pain.

The patient's clinical course was closely monitored. On the second day of hospitalization, he developed nausea and reported a streak of blood in his sputum. By day four, his fever and chest pain had resolved, and his cough began to show signs of improvement. However, on day six, he experienced a transient recurrence of chest pain and chills, with a minor temperature elevation to 37.1°C. This prompted a repeat CBC and chest radiograph. Following this episode, his condition

improved steadily. By day eight, the rhonchi on auscultation had completely resolved. He was deemed clinically stable and was planned for discharge.

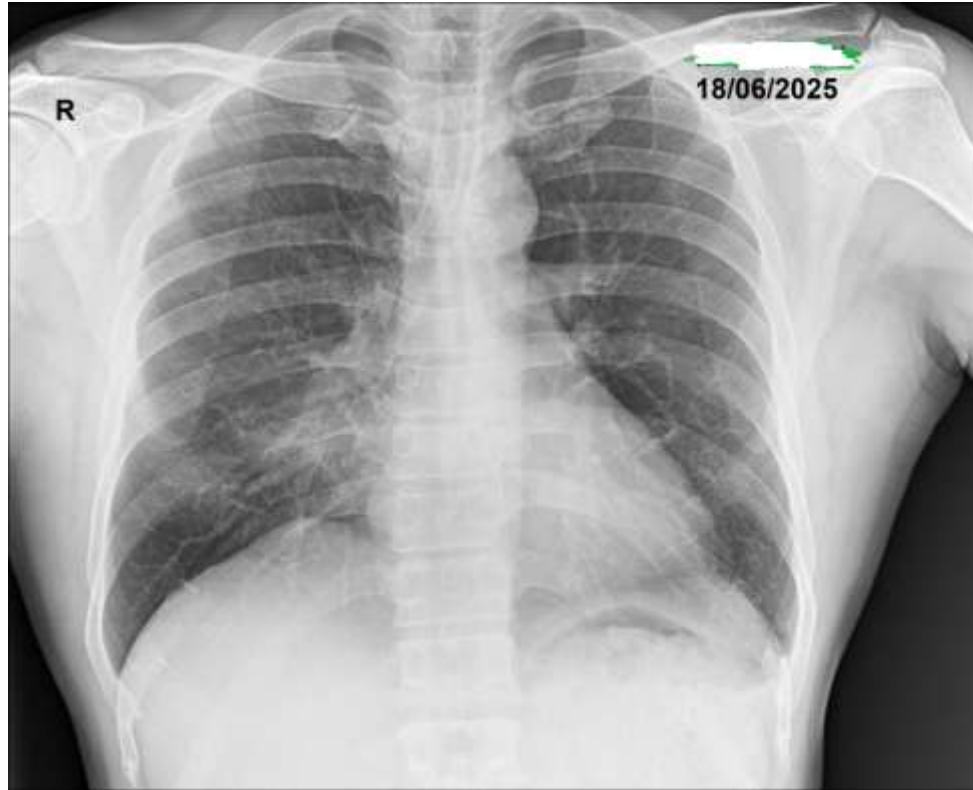


Figure 3. Anteroposterior (AP) Chest Radiograph at 18 June 2025

Outcome and Discharge

The patient was discharged on the ninth hospital day in a stable and improved condition. All intravenous medications were discontinued. He was provided with a prescription for a course of oral medications, including Azithromycin 500 mg once daily (to complete a course of atypical coverage), Paracetamol for analgesia, Acetylcysteine, Omeprazole, and Sucralfate syrup. He was counseled on the critical importance of completing the full course of antibiotics, the need for a follow-up appointment to ensure complete clinical and radiological resolution, and was advised to

discuss occupational health risks and smoking cessation strategies.

DISCUSSION

Diagnostic Confirmation and Etiological Considerations

The diagnosis of CAP in this patient was firmly established based on the classic triad of clinical symptoms (fever, productive cough), focal findings on chest examination (decreased breath sounds and rhonchi localized to the right lung), and definitive radiographic evidence of a new pulmonary infiltrate (Metlay et al., 2019; AAFP, 2006). The supporting laboratory data, including a marked leukocytosis with neutrophilia, further substantiated an acute bacterial infectious process (Ho et al., 2008; Falk and Fahey, 2009).

Although specific microbiological testing to identify the causative pathogen was not performed—a common scenario where an etiologic agent is not found in up to 50% of CAP cases—the clinical and radiological presentation of lobar consolidation strongly points towards *Streptococcus pneumoniae* as the most probable pathogen (Welte et al., 2012; Radiopaedia, 2023b). Nonetheless, other potential pathogens, including *Haemophilus influenzae* and atypical organisms such as *Mycoplasma pneumoniae* or *Legionella pneumophila*, remain important considerations in the differential diagnosis and provide the rationale for initiating broad-spectrum empiric antibiotic therapy (Mandell et al., 2007; Medscape, 2024).

The Nexus of Occupation, Smoking, and Pneumonia Susceptibility

A central and instructive aspect of this case is the patient's occupational role as a "fitter-splicer," which implies a complex, multi-source exposure profile that is likely more hazardous than a single exposure type. Deconstructing this job title reveals a convergence of well-established respiratory risks. The "fitter" component in an industrial or mining context typically involves the assembly, installation, and maintenance of pipe systems and metal structures (KSK, 2023; MTP, n.d.). Such work inherently involves activities like cutting, grinding, and welding, which are known

to generate respirable metal fumes (containing iron, manganese, zinc) and inorganic dusts, such as respirable crystalline silica (RCS), from nearby concrete or stone (Breathe Freely, 2021; Breathe Freely, 2019). The "splicing" component, particularly in settings with conveyor systems, often refers to the hot vulcanization of rubber belts, a process that releases a complex mixture of chemical fumes, vapors, and organic dusts (IARC, 1982; Szewczyńska et al., 2002). Consequently, this patient was likely exposed to a dual assault on his respiratory system: metallic/inorganic dusts and organic/chemical fumes.

The pathophysiological mechanism linking these exposures to increased pneumonia risk involves a profound impairment of local pulmonary host defenses. Chronic inhalation of metal fumes, especially those rich in iron, has been shown to paradoxically blunt the local inflammatory response of alveolar macrophages while simultaneously providing a rich nutrient source for bacteria, thereby increasing susceptibility to infection (Palmer et al., 2003; Szram et al., 2005). Concurrently, inorganic dusts like silica are cytotoxic to alveolar macrophages, overwhelming their phagocytic capacity and triggering a chronic inflammatory cascade that can lead to fibrosis (silicosis) and a well-documented increase in susceptibility to infections, including both pneumonia and tuberculosis (Leung et al., 2012; Hopkins Medicine, 2023).

The patient's 30-pack-year smoking history acts as a powerful and synergistic effect modifier. Smoking does not simply add to the occupational risk; it multiplies it through a mechanism of "particle overload" (Barnes et al., 2025). The lung's mucociliary escalator and alveolar macrophages have a finite capacity for clearing inhaled particulates. The constant burden from occupational dusts already strains this system. The addition of the massive particulate load from cigarette smoke, which independently paralyzes cilia and damages macrophages, leads to a saturation of these clearance pathways (Barnes et al., 2025). This results in the prolonged retention of both toxic occupational dusts and tobacco smoke components within the lung parenchyma. This retained material incites a state of chronic, low-grade inflammation and fundamentally cripples the innate immune system's ability to mount an effective and timely response to an acute challenge,

such as the micro-aspiration of pathogenic bacteria like *S. pneumoniae*, thus dramatically increasing the likelihood of clinical pneumonia developing (Nuorti et al., 2000; Feldman and Anderson, 2020).

Analysis of Therapeutic Strategy

Empiric Antibiotic Selection: The patient was managed with a dual antibiotic regimen of intravenous ceftriaxone and oral levofloxacin. This combination provides robust, broad-spectrum coverage against the most common CAP pathogens, including drug-resistant *S. pneumoniae*, as well as atypical pathogens (File, 2004; Mandell et al., 2007). While standard guidelines for hospitalized patients with non-severe CAP often recommend either respiratory fluoroquinolone monotherapy or a combination of a beta-lactam plus a macrolide, the chosen regimen of a beta-lactam plus a fluoroquinolone is a potent and acceptable alternative (File, 2004; Sligl et al., 2024). The patient's positive clinical response, with resolution of fever and improvement in symptoms, validates the efficacy of this empiric choice.

Navigating the Clinical Course and Differential Diagnoses

The patient's initial presentation with hemoptysis, combined with his profound risk factors of long-term smoking and occupational dust exposure, appropriately raised suspicion for two critical differential diagnoses: pulmonary tuberculosis (TB) and an underlying lung malignancy.¹

Tuberculosis: TB must be considered in any patient with pneumonia, particularly in endemic regions or in individuals with risk factors for impaired immunity, such as silicosis, which is a known complication of his occupational exposures (Leung et al., 2012; Jolobe, 2012). Although the patient's rapid response to broad-spectrum antibiotics made active TB less likely, a definitive workup would typically involve collecting serial sputum samples for acid-fast bacilli (AFB) smear, culture, and nucleic acid amplification testing (NAAT). The absence of typical radiographic features of post-primary TB, such as upper-lobe infiltrates or cavitation, also argued against this diagnosis (Jeong and Lee, 2015; NYC DOHMH, 2022; Aggarwal et al., 2014).

Lung Malignancy: Non-resolving or slowly resolving pneumonia is a classic presentation for an endobronchial malignancy causing post-obstructive pneumonitis (Feinsilver et al., 2003; Medscape, 2023). Given the patient's age and extensive smoking history, his pre-test probability for lung cancer is substantial. While his clinical improvement with antibiotics makes an acute infectious process the primary diagnosis, it does not exclude the presence of an underlying lesion that precipitated the infection. The transient worsening of his condition on day 6 is characteristic of slowly resolving pneumonia. Therefore, a critical component of his long-term management is post-discharge follow-up imaging. A repeat chest radiograph or, preferably, a chest CT scan at 6 to 8 weeks post-treatment is essential to ensure complete radiological resolution of the infiltrate and to definitively rule out an occult malignancy that may have been masked by the acute inflammatory process (Feinsilver et al., 2003; Kuru and Kalkan, 2024; Mayo Clinic, 2022).

Preventive Medicine and Occupational Health Implications

This case serves as a poignant example of a likely preventable disease and highlights critical opportunities for primary prevention.

- **Occupational Safety:** Employers are mandated to implement the hierarchy of controls to minimize worker exposure to hazardous dusts and fumes. This should include engineering controls like local exhaust ventilation at the source, administrative controls such as job rotation to limit exposure duration, and the provision of appropriate, fit-tested personal protective equipment (PPE), such as NIOSH-approved respirators, as part of a comprehensive written respiratory protection program (OSHA, n.d.; OSHA, 2022).
- **Smoking Cessation:** The powerful synergistic effect between smoking and occupational exposures underscores the urgent need for accessible and effective smoking cessation programs specifically targeted at industrial workers.
- **Vaccination:** There is compelling evidence and formal recommendations from public health bodies, such as the Department of Health in England, for offering the 23-valent pneumococcal polysaccharide vaccine (PPV23) to all welders (Palmer and Cosgrove, 2012; OHSAS, n.d.;

Cummins et al., 2019). The underlying rationale—that chronic metal fume exposure impairs pulmonary defenses against pneumococcus—applies directly to this fitter-splicer. A key recommendation stemming from this case is the logical extension of this vaccination guidance to include other industrial trades with similar high-intensity exposures to metal fumes and inorganic dusts. This simple, cost-effective intervention has the potential to prevent severe illness and mortality in this high-risk population.

CONCLUSION

This report details a case of community-acquired pneumonia in a 50-year-old fitter-splicer, where the patient's clinical course was profoundly influenced by the synergistic interplay of his specific occupational exposures and a long-standing smoking habit. The complex exposure profile associated with industrial fitting (metal dust) and splicing (chemical fumes) likely resulted in a significant, chronic impairment of his pulmonary immune defenses, creating a state of heightened vulnerability to respiratory infection.

Ultimately, this case serves as a powerful reminder for clinicians to maintain a high index of suspicion for occupational etiologies in all patients presenting with pneumonia and to meticulously document occupational, environmental, and smoking histories. It underscores the paramount importance of primary prevention in industrial settings, which must include a multi-pronged approach of stringent exposure controls, robust smoking cessation support, and the targeted, proactive use of preventive measures like pneumococcal vaccination for at-risk workers. Ensuring complete radiological follow-up in such high-risk patients is also crucial to rule out underlying pathologies like lung cancer.

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