



PAKISTAN  
CHEST SOCIETY  
STRIVING FOR PULMONARY CARE

CLINICAL PRACTICE  
GUIDELINES

# OCCUPATIONAL LUNG/PLEURAL DISEASES

PAKISTAN CHEST SOCIETY-2026



Guidelines on

# Occupational Lungs / Pleural Diseases

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2026



PAKISTAN  
CHEST SOCIETY  
STRIVING FOR PULMONARY CARE

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## Preface

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The Industrial Revolution has led to human exposure to newer substances, which may be a risk factor for occupational lung diseases. Occupational lung diseases (OLD) are a broad group of work-related diseases occurring in a variety of occupations and presenting with a wide range of diseases, from asthma to more severe conditions like pulmonary fibrosis and mesothelioma. These diseases are a significant but often under-recognized and preventable cause of morbidity and mortality worldwide. Since the symptoms can be easily mistaken for those of non-occupational lung diseases, identifying the work-related cause is crucial for clinicians, public health professionals, and policy makers. This information enables them to formulate effective preventive, diagnostic, treatment and compensation strategies for workers. So OLD should be considered in the differential diagnosis of almost every case of a chronic pulmo-pleural disease, thus highlighting the importance of a proper occupational history. Aim: the primary aim of the guidelines is to take care of the need of medical students, general practitioners, postgraduate trainees, clinicians and occupational health regulators in prevention, diagnosis, management and compensation of workers with OLD.

### **Prof. Mohammad Yousaf Khan (Chairman)**

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## Message by the President Pakistan Chest Society

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Occupational lung diseases continue to pose a significant yet under-recognized health burden in Pakistan. These guidelines highlight the importance of occupational exposure assessment, early diagnosis, preventive strategies, and appropriate management. PCS reaffirms its commitment to promoting workplace respiratory health through education, advocacy, and standardized clinical practice.



### **Prof. Shereen Khan**

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President  
Pakistan Chest Society

## Message by the Chairman

### Guideline Committee, Pakistan Chest Society

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It is my privilege to present the **Guidelines for the Management of Occupational Lung Diseases** developed by the Pakistan Chest Society. Occupational lung diseases represent an important yet frequently underrecognized cause of respiratory morbidity in Pakistan, particularly among workers exposed to dust, industrial chemicals, fumes, and other hazardous agents in sectors such as mining, construction, manufacturing, and agriculture. Chronic occupational exposure can lead to a range of



respiratory conditions including pneumoconiosis, occupational asthma, hypersensitivity pneumonitis, and chronic obstructive pulmonary disease. These guidelines have been developed by the Working Group for Occupational Lung Diseases under the chairmanship of Prof. Mohammad Yousaf Khan and after reviewing current international evidence and adapting it to the local occupational and healthcare context. The document provides practical recommendations on risk identification, early diagnosis, clinical evaluation, and evidence-based management of common occupational lung diseases, while emphasizing the importance of preventive strategies and workplace safety. By promoting a standardized approach to recognition and management, these guidelines aim to improve early detection, enhance patient care, and reduce the burden of occupational respiratory diseases in Pakistan.

## Prof. Muhammad Ashraf Jamal

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# Pakistan Chest Society

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# Occupational Lung Diseases

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# Introduction

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Author: Dr. Mohammad Yousaf Khan

## **Common Occupational Lung Diseases**

Some of the common occupational lung diseases discussed in the guidelines include:

**Hypersensitivity pneumonitis:** An allergic reaction to a specific substance, such as a chemical, protein, or organism.

**Occupational asthma:** an immune response to a sensitizing agent cause airflow limitation and shortness of breath.

**Asbestosis:** A lung disease caused by inhaling asbestos fibres.

**Mesothelioma:** A lung disease that can cause pleural effusion and thickening.

Coal worker's pneumoconiosis: Also known as black lung disease, this lung disease is caused by inhaling coal dust.

**Silicosis:** A lung disease caused by inhaling silica, a mineral found in sand, quartz, and other rocks.

**Bagassosis:** An interstitial lung disease, a type of hypersensitivity pneumonitis, caused by inhalation of the fibrous cane-sugar residue, bagasse.

**Byssinosis:** Also known as brown lung disease, this lung disease is caused by inhaling dust from hemp, flax, and cotton processing.

**Aluminosis:** A rather uncommon lung disease caused by exposure to aluminium dust and fumes. It is clinically important because exposure to aluminium dust may induce a wide range of pulmonary pathologies, including pulmonary granulomatosis, pulmonary fibrosis, pulmonary alveolar proteinosis, and desquamative interstitial pneumonia.

## **Legislative framework for employees' safety and health from occupational health diseases**

Pakistan has a number of laws that address occupational safety and health, including the Factories Act, 1934, the Mines Act, 1923, and the Dock Labourers Act, 1934. Factories Act, 1934, requires factories to maintain clean and safe working conditions and adequate ventilation, lighting, and sanitation facilities. It also requires machinery to be properly maintained and guarded.

**Mines Act, 1923** focuses on safety measures in the mining sector.

**Dock Labourers Act, 1934** protects the safety of workers employed in dock areas.

### **Other Related Laws:**

Workmen Compensation Act, 1923

Khyber Pakhtunkhwa Workers' Compensation Act, 2013

Sindh Workers Compensation Act, 2016

**Occupational safety and health legislation by provinces:** Sindh (2017), Punjab (2019), Khyber Pakhtunkhwa (2022), and Balochistan (2022). Occupational safety and health standards are a set of rules, guidelines, and measures that aim to protect workers' physical safety, health, and social and economic well-being.

### **Mechanisms for implementing laws and regulations regarding workers' safety at the workplace**

Mechanisms for enforcement of compliance with the laws and regulations include the following:

1. System for supervision and inspection of workers
2. Number of inspectors, inspection visits, and reports
3. Support mechanism for progressive improvement of occupational safety and health in accordance with the national and provincial laws and regulations

### **Current situation of occupational lung diseases in Pakistan**

In the twentieth century, occupational lung diseases were the problem commonly faced by workers in developed countries. In developing countries like Pakistan, workers still face unregulated and unprotected exposure to occupational hazards. The difference now is that developing country workers are being exposed when risk and preventive measures are established, health surveillance strategies are well known, laws and regulations exist, and mechanisms for implementation are there. The problem is that most developing countries are complacent to the rule of law and good governance regarding workers' safety and health.

# Prevention

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Author: Dr. Saadia Ashraf

Occupational lung diseases (OLDs) remain a significant yet preventable cause of morbidity and mortality worldwide. In Pakistan, rapid industrialization, informal labor structures, and weak enforcement of occupational health laws have created conditions where preventable respiratory diseases continue to affect large numbers of workers.

This chapter provides comprehensive preventive strategies for major occupational lung diseases:

- Work-related asthma and COPD
- Hypersensitivity pneumonitis (HP)
- Silicosis
- Coal workers' pneumoconiosis (CWP)
- Asbestosis and mesothelioma
- Byssinosis (cotton dust disease)
- Bagassosis (sugarcane fibre hypersensitivity)
- Aluminosis

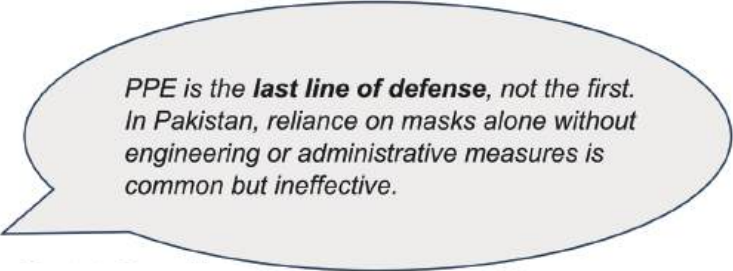
Emphasis is placed on:

1. **The hierarchy of controls** as a framework.
2. **Common, feasible, and cost-effective measures** in resource-limited settings.
3. **Integration of prevention into public health surveillance** and existing health structures.
4. **Contextual realities in Pakistan**, where enforcement and resources are limited.

## The Hierarchy of Controls

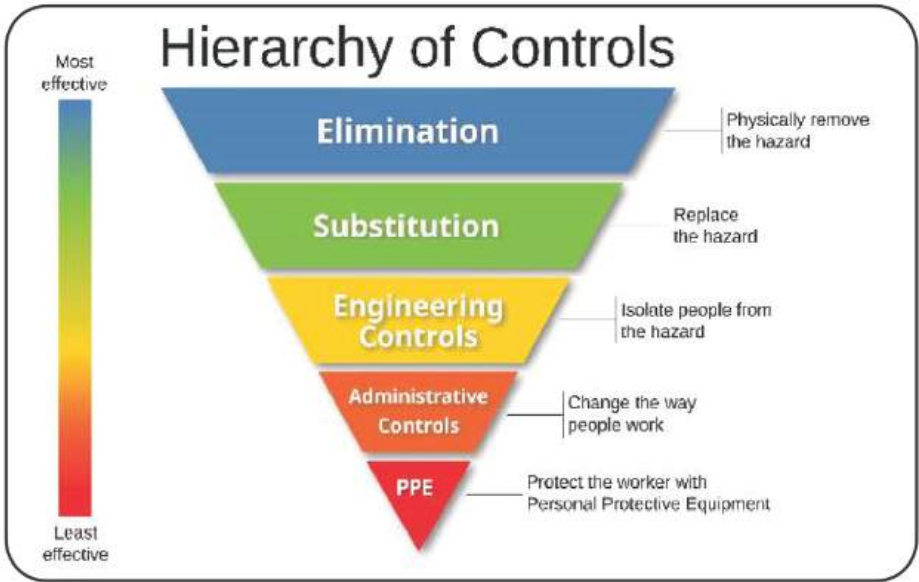
**Elimination** → **Substitution** → **Engineering** → **Administrative** → **PPE.**

- **Elimination:** Remove hazardous processes altogether (e.g., banning asbestos use).
- **Substitution:** Replace with safer materials (e.g., synthetic fibers for cotton, sandblasting alternatives).
- **Engineering controls:** Local exhaust ventilation (LEV), wet suppression, enclosure.
- **Administrative controls:** Rotating tasks, limiting shifts in dusty environments, enforcing wet cleaning.
- **Personal Protective Equipment (PPE):** Respirators, protective clothing.



*PPE is the **last line of defense**, not the first. In Pakistan, reliance on masks alone without engineering or administrative measures is common but ineffective.*

**Figure 1** Hierarchy of Controls Pyramid.



**General Preventive Approaches**

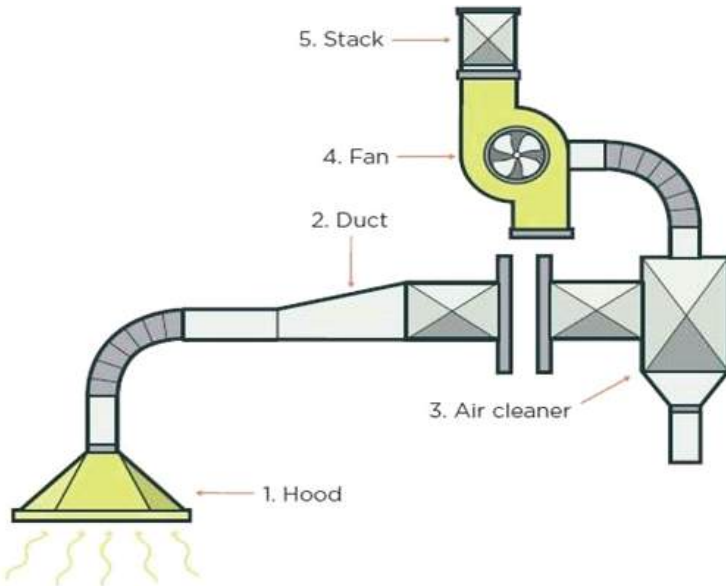
**Engineering Controls**

**Wet suppression:** Continuous water spray at drilling, cutting, or grinding points.

**Local exhaust ventilation (LEV):** Capture hoods near dust/fume sources.

**Enclosure:** Sealing high-dust operations.

**Cross ventilation:** Simple roof vents or side openings in brick kilns, cotton mills, stone cutting yards.



Basic components of a Local Exhaust Ventilation (LEV) system: hood, ducting, fan, and filter.

## Movable Exhaust Hoods

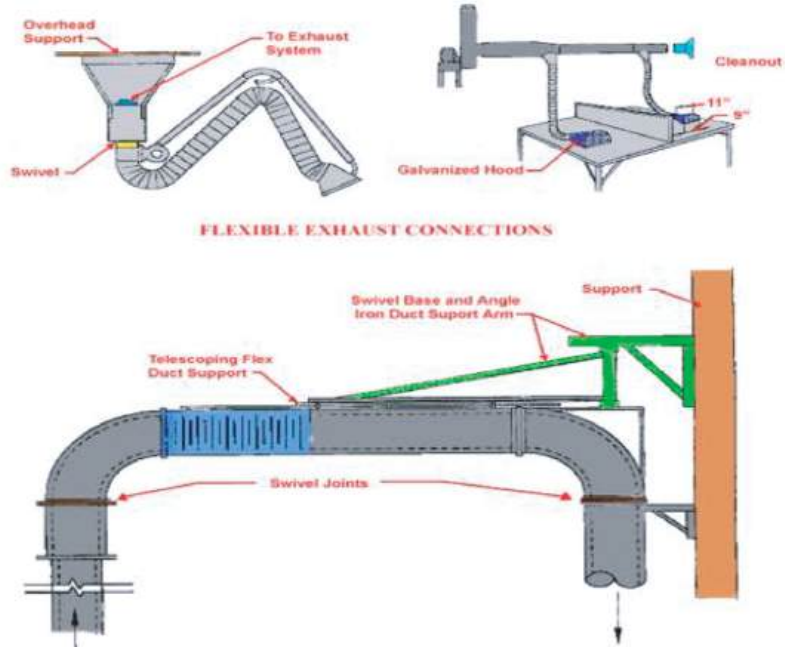


Figure 2 Examples of ventilation strategies.

### Administrative Controls

- Dusty tasks scheduled during non-peak hours.
- Rotating workers between low- and high-exposure tasks.
- Prohibiting dry sweeping.
- Training programs and signage with pictograms for workers with limited literacy.

### Personal Protective Equipment (PPE)

- **Filtering facepiece respirators (N95, P2, P3):** Minimum for dusty workplaces.
- **Reusable elastomeric half-mask respirators:** Cost-effective long-term.
- **Powered air-purifying respirators (PAPR):** For very dusty environments.
- **Fit testing & seal checks** essential for effectiveness.

## TYPES OF RESPIRATORY PROTECTION

 <p><b>Elastomeric Half Facepiece Respirators</b> are reusable and have replaceable cartridges or filters. They cover the nose and mouth, and provide protection against gases, vapors, or particulates equipped with the appropriate cartridge or filter.</p>	 <p><b>Elastomeric Full Facepiece Respirators</b> are reusable and have replaceable cartridges, cartridges, or filters. The facepiece covers the face and eyes, which offers eye protection.</p>	 <p><b>Filtering Facepiece Respirators</b> are disposable half facepiece respirators that filter out particles such as dusts, mists, and fumes. They do NOT provide protection against gases and vapors.</p>	 <p><b>Powered Air-Purifying Respirators (PAPR)</b> have a battery-powered blower that pulls air through a filter. They, cartridges, or canisters. They provide additional airflow against gases, vapors, or particles when equipped with the appropriate cartridge, canister, or filter. Loose fitting PAPRs do not require fit testing as it can be used with facial hair.</p>
 <p><b>Supplied-Air Respirators</b> are connected to a separate source that supplies clean compressed air through a hose. They are not lightweight, need airtight seals, and are used in extremely dangerous to life and health (IDLH).</p>	 <p><b>Self-Contained Breathing Apparatus (SCBAs)</b> are used for entry into or escape from environments contaminated by low IDH. They contain their own breathing air supply and can be either open circuit or closed circuit.</p>	 <p><b>Combination Respirators</b> can be either a supplied air SCBA respirator or a supplied air air-purifying respirator. The P2/3A type has a self-contained air supply if primary source fails or it can be used in IDH environments. The air-purifying type offers protection using both a supplied air flow &amp; an air-purifying component and cannot be used for entry into IDH environments.</p>	

**Table 1 : Respirator types, protection factors, suitability.**

Respirator Type	Filter/Cartridge	Assigned Protection Factor (APF*)	Typical Use Cases	Limitations / Caveats
<b>Filtering Facepiece Respirator (FFR)</b> (e.g., N95, N99, N100; P95, P100)	Disposable filter media	10	Moderate dust (e.g., cotton mills, bagasse, construction dust)	Must be fit-tested; poor seal with facial hair; not reusable; not effective against gases/vapors
<b>Elastomeric Half-Mask Respirator</b>	Replaceable P100 filters	10	Higher dust exposure (e.g.,	Requires cleaning & storage; heavier than FFR
<b>Full-Facepiece Elastomeric Respirator</b>	Replaceable P100 or combination cartridges	50	High dust, asbestos removal, welding fumes	Provides eye protection; communication difficult; heavier
<b>Powered Air-Purifying Respirator (PAPR)</b>	High-efficiency filters (HEPA/P100)	25–1000 (depending on configuration)	Very high dust environments, silicosis risk industries	Expensive; requires battery charging/maintenance
<b>Supplied-Air Respirator (SAR) or SCBA</b>	External air supply / compressed cylinder	≥1000	Extreme hazard situations (e.g., firefighting, confined spaces, asbestos abatement)	Costly, heavy, requires training; not suitable for routine use in Pakistan's informal sector
Respirator Type	Filter/Cartridge	Assigned Protection Factor (APF*)	Typical Use Cases	Limitations / Caveats
			stone cutting, flour milling)	

\*APF = *Assigned Protection Factor* as defined by OSHA/NIOSH (represents the workplace level of respiratory protection a respirator is expected to provide when properly fitted and used).

In resource-limited Pakistani settings, **reusable elastomeric half-mask respirators** with P100 filters are the most **cost-effective and sustainable option**, compared with disposable N95s that are often reused

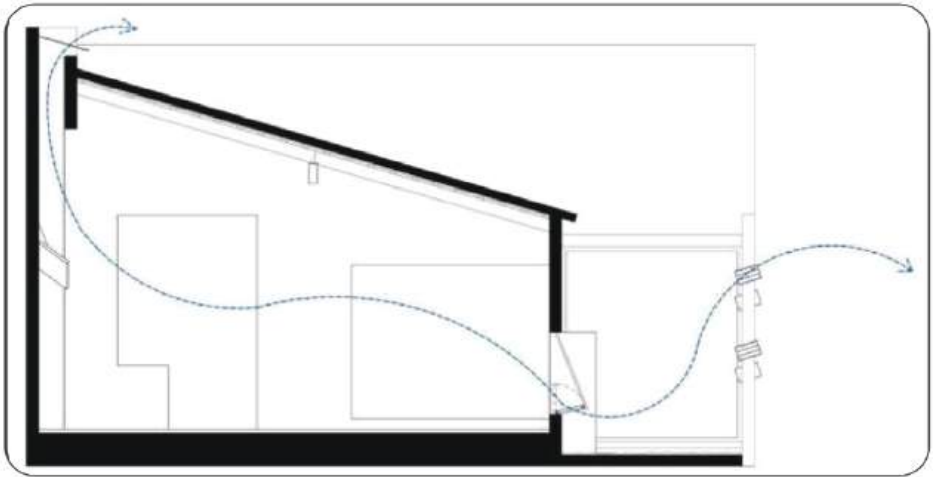
## Disease-Specific Preventive Strategies

### 1. Work-related Asthma & COPD

- Elimination/substitution of sensitizers (e.g., low-VOC paints, enzyme substitutes).
- Enclosures for spray-paint booths.
- Adequate ventilation in welding and spray painting.
- Worker education on early reporting of symptoms.
- Pre-employment and periodic spirometry.

### 2. Hypersensitivity Pneumonitis (HP)

- Moisture control in cotton, bagasse, hay storage.
- Enclosure and Local Exhaust Ventilation in grain processing mills.
- Substitution of microbial-contaminated materials.
- PPE during high-exposure tasks (filtering respirators).



**Figure 3:** Storage ventilation schematic for preventing mold growth.

### 3. Silicosis

- Wet drilling and on-tool misting.
- LEV capture hoods in stone cutting and construction.
- No sandblasting with silica sand (promote steel grit substitutes).
- Periodic chest radiography (ILO classification).
- Symptom screening integrated into TB programs.

*Silicosis prevention in Pakistan requires urgent banning of dry stone cutting and promotion of wet methods. "No dry stone cutting" should be enforced nationally.*

### 4. Coal Workers' Pneumoconiosis (CWP)

- Dust suppression using water sprays in mining.
- Ventilation shafts and mechanical exhausts.
- Continuous Personal Dust Monitors (CPDM) for high-risk mines.
- Annual chest X-ray and spirometry.

## 5. Asbestosis and Mesothelioma

- Ban on import and use of asbestos (lagging, brake linings).
- Safe asbestos removal protocols.
- Use of high-efficiency respirators (PAPR, full-face).
- Surveillance: annual chest radiograph, HRCT if abnormal.

## 6. Byssinosis (Cotton Dust Disease)

- Humidification and ventilation in cotton mills.
- Dust suppression at carding machines.
- Task rotation to limit exposure.
- Spirometry every 6–12 months.

## 7. Bagassosis

- Prevent damp storage of sugarcane bagasse.
- Improve bagasse ventilation.
- Health surveillance for HP symptoms.
- PPE during handling.

## 8. Aluminosis

Enclosure and LEV in aluminum processing.

Avoid dry grinding/polishing.

PPE with high-efficiency filters.

### Surveillance and Screening

*Surveillance by hazard (baseline, periodic, referral triggers).*

- **Silica:** Spirometry + chest X-ray every 6-12 months.
- **Coal dust:** Annual chest X-ray and spirometry.
- **Asbestos:** Radiography every 12 months, HRCT for suspected disease.
- **Cotton dust:** Spirometry every 6-12 months.

### Common and Doable Strategies in Resource-Limited Settings

1. **Wet sweeping/mopping instead of dry sweeping.**
2. **Locally fabricated LEV systems** using exhaust fans and ducting.
3. **Bucket and hand-sprayer wetting** for drilling/cutting stones.
4. **Reusable elastomeric respirators** instead of disposable N95s.
5. **Community spirometry camps** using portable devices.
6. **TB van linkages** for chest radiographs.
7. **Posters with pictograms** for illiterate workers.
8. **Peer safety champions** in small workshops.

*In Pakistan, the biggest gains can be made by enforcing just three measures:  
(1) Banning dry sweeping  
(2) Mandating wet stone cutting  
(3) Introducing reusable respirators*

## Policy and Legal Framework

- **ILO Conventions** on Occupational Safety Health and silicosis
- **Pakistan Factories Act (1934)** - outdated and poorly enforced.
- Need for **national occupational exposure limits (OELS)**
- aligned with american conference of governmental industrial hygienists (ACGIH).
- **Integration with TB programs** due to silico-TB overlap.
- **National surveillance registry** for OLDs.

## Summary

Occupational lung diseases are **preventable but neglected** in Pakistan.

The Pakistan Chest Society recommends:

- Implementation of the **hierarchy of controls**.
- Emphasis on **engineering and administrative measures**, supported by PPE.
- Adoption of **simple, low-cost interventions** feasible in resource-limited settings.
- Integration with TB programs for surveillance.
- Stronger **policy enforcement** and **ban on asbestos and dry stone cutting**.

**Quick Reference:** *Preventive Measures at a Glance.*

Disease	Key Hazard	Top Preventive Measures
Silicosis	Respirable crystalline silica	Wet drilling, LEV, reusable respirators, TB linkage
CWP	Coal dust	Water sprays, ventilation shafts, annual surveillance
Asbestosis	Asbestos fibers	Material ban, safe removal, full-face respirators
Byssinosis	Cotton dust	Humidification, dust suppression, spirometry
Bagassosis	Moldy sugarcane fiber	Ventilation, storage management, PPE

## References:

1. World Health Organization. Occupational health: A manual for primary health care workers. Geneva: WHO; 2001.
2. International Labour Organization. Safety and health in mines convention (No. 176). Geneva: ILO; 1995.
3. National Institute for Occupational Safety and Health (NIOSH). Workplace safety and health topics. Centers for Disease Control and Prevention; 2023.
4. Pakistan Chest Society. Guidelines for the management of silicosis patients in a primary care setting. Peshawar: PCS; 2024.
5. Occupational Safety and Health Administration (OSHA). Respirable crystalline silica standard. Washington DC: OSHA; 2016.
6. Parkes WR. Occupational lung disorders. 3rd ed. London: Butterworth-Heinemann; 1994.
7. Hnizdo E, Vallyathan V. Chronic obstructive pulmonary disease due to occupational exposure to silica dust: A review of epidemiological and pathological evidence. *Occup Environ Med.* 2003;60(4):237–43.

- 8.** Sigsgaard T, Nowak D, Annesi-Maesano I. Occupational asthma: diagnosis, management and prevention. *Eur Respir J.* 2019;53:1802318.
- 9.** International Commission on Occupational Health (ICOH). Global elimination of silicosis. ICOH Statement; 2015.
- 10.** Nafees AA, Fatmi Z, Kazi A, et al. Lung function decline among textile workers exposed to cotton dust: A 5-year follow-up. *Occup Environ Med.* 2013;70(8):575–9.

# Hypersensitivity Pneumonitis (Extrinsic Allergic Alveolitis)

Author: Dr. Nousheen Iqbal

## Definition

Hypersensitivity Pneumonitis (HP) is also known as extrinsic allergic alveolitis. It is typically an immune-mediated disease that manifests as ILD in susceptible individuals after exposure to an identified or unidentified factor including, but not limited to, agricultural dusts, bioaerosols, and certain reactive chemical species. First National ILD Registry of Pakistan published in 2021 in which HP ILD was reported in 17.7% with an avian exposure in 79.5% of HP patients, while 86.8% were females.<sup>1</sup>

## Subtypes

HP has been classified previously as acute, subacute, or chronic depending upon the frequency, duration, and intensity of exposure and illness duration. But ATS / JRS / ALAT guideline on HP suggest classification into fibrotic and non-fibrotic HP. (2) Fibrotic (i.e., mixed inflammatory plus fibrotic or purely fibrotic) or nonfibrotic (i.e., purely inflammatory). Usually in Fibrotic HP patients have chronic history with low to unidentifiable exposure or have past exposure. On other hand, Non-Fibrotic HP history is usually short with high intensity or chronic exposure. Patients present with dyspnea, cough, fever, malaise and/or acute respiratory failure.<sup>5</sup>

## Causes

HP develops in vulnerable individuals after recurrent exposure to one or more agents as shown in (Table 1). Inciting agents include microbial, plant, avian or animal-based proteins, or inorganic low-molecular weight chemical agents that combine with host proteins to form haptens. Exposure can occur at home, workplace or recreational environment.<sup>4</sup>

Sources	HP diseases
Fungi/Moulds/Yeast	Farmer's lung
Moldy hay	Humidifier lung
Contaminated water/misting fountains/humidifiers and air conditioning system	Malt worker's lung
Contaminated plant material (moldy barley)	Woodworker's lung
Contaminated sawdust, moldy wood	Indoor air alveolitis (domestic HP)
Contaminated houses (flooded), systems, upholstered furniture, potted domestic ventilation and cooling flowers	Compost lung
Compost	

<p>Contaminated stucco</p> <p>Moldy cork</p> <p>Aspergillus enzyme in baking agents</p> <p>Organic wastes</p> <p>Cheese casings</p> <p>Moldy thatched roof</p> <p>Red wood dust</p>	<p>Stucco worker's lung</p> <p>Suberosis</p> <p>Baker's lung</p> <p>Waste sorter's lung</p> <p>Cheese washer's lung</p> <p>Thatched roof lung</p> <p>Sequoiosis</p>
<p><b>Edible mushrooms</b></p> <p>Mushrooms growing</p>	<p>Mushrooms grower's lung</p>
<p><b>Bacteria</b></p> <p>Contaminated machine fluid, metalworking fluids</p> <p>Sewage treatment plants</p> <p>Detergents, washing powders, biological cleaning agents</p> <p>Moldy molasses, bagasse dust</p>	<p>Machine operator's lung</p> <p>Sewage worker's pneumonitis</p> <p>Detergent workers alveolitis</p> <p>Bagassosis</p>
<p><b>Animal fur dust</b></p> <p>Avian droppings, serum, feathers: Parakeets, canaries, budgerigars, pigeons, parrots, chicken, turkeys, geese, ducks, wild birds, pheasants</p> <p>Feather beds, pillows, duvets</p> <p>Cow Milk</p> <p>Fish Feed (Daphnia, meat, mosquito larvae)</p> <p>Oyster shell powder</p> <p>Dust from silkworm larvae and cocoon</p> <p>Contaminated grain or flour</p>	<p>Furrier's lung</p> <p>Bird fancier's disease, bird breeder's disease, pigeon breeder's lung, chicken breeder's lung</p> <p>Feather duvet lung</p> <p>Heiner syndrome</p> <p>Fish feed alveolitis</p> <p>Shellfish alveolitis, oyster shell HP, mollusk shell HP</p> <p>Silkworm rearer's lung</p> <p>Corn (wheat) weevil lung</p>
<p><b>Plant Proteins</b></p> <p>Esparto grass</p> <p>Flour dust (wheat, rye, oats, maize)</p> <p>Legumes (soya) flour dust</p> <p>Paprika dust</p> <p>Wood particles (Cabreuva, cedar, pine, mahogany, umbrella pine)</p>	<p>Esparto lung, plasterer's lung</p> <p>Flour dust alveolitis</p> <p>Soya dust alveolitis</p> <p>Paprika splitter's lung</p> <p>Wood fiber alveolitis</p>

<p><b>Chemicals</b>  Polyurethane foams, spray paints, elastomers, glues, adhesives, mattress, car parts, shoes,</p>	<p>Acid anhydride alveolitis</p>
<p>imitation leather, rubber products, chipboard, elastic synthetic fibers, electrical insulations, Hardener for epoxy resin.</p> <p>Dental materials, lacquer, resin, glues  Copper sulfate Bordeaux mixture  Degreasing agents, cleaning agents, extraction agents</p> <p>Coolant fluid in laser hair removal devices  Polyester powder (powder paints)  Laboratory reagent, chromatography</p>	<p>Methacrylate  Vineyard sprayer's lung  Chemical alveolitis</p> <p>Hair remover lung  Painter's lung  Chemical alveolitis</p>
<p><b>Pharmaceutical Agents:</b>  Lenalidomide  Parvastatin  Venlafaxine  Temozolide  Penicillins, Cephalosporins  Methotraxate  a - IFN</p> <p><b>Metals:</b>  Zinc fumes  Zircon  Batteries, computers, neons  Hard Metals, Alloys</p>	<p>Drug Induced HP</p> <p>Zinc fumes alveolitis  Zirconium alveolitis  Beryllium HP  Giant Cell pneumonitis</p>

**Pathophysiology**

An immune reaction develops after exposure to the inciting agent in susceptible individuals which included both humoral (i.e., antigen-specific IgG antibodies) and T-helper cell type 1 (Th1) cellular immune responses.<sup>5,6</sup> The pathogenesis of pulmonary fibrosis is not fully understood in HP. However, it is believed that due to recurrent alveolar epithelial injury following repeated exposure leads to fibroblast activation and proliferation, the accumulation of extracellular matrix, and the eventual destruction of the lung architecture leads to pulmonary fibrosis.

**Symptoms and Signs**

Mostly patients present with dyspnea and cough, with examination findings of mid-inspiratory squeaks. Some patients may have constitutional symptoms like flu, fever, weight loss, malaise, chest tightness and wheezing and crackles on examination.<sup>7</sup>

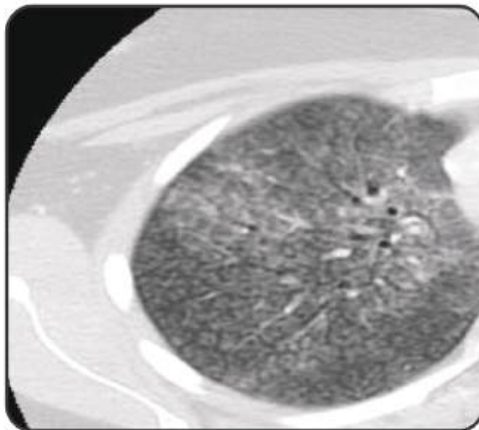
## Diagnostic Workup

The diagnosis of HP requires a multidisciplinary approach, which includes clinical, radiographic (HRCT) findings, in some cases bronchoalveolar lavage (BAL) lymphocytosis and histopathologic data.<sup>5,6</sup> According to the CHEST guidelines 2021, a confident diagnosis of HP can be made in patients who have an identified exposure and a typical HP pattern on a high-resolution computed tomography (HRCT) scan.<sup>4</sup> We strongly recommend HRCT chest in every patient with suspected HP.

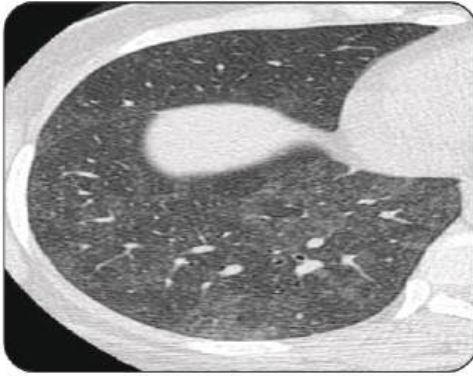
## 1. HRCT Findings in HP

HRCT chest plays a vital role in the diagnosis of HP. HRCT findings include Ground glass haze, air trapping, Centrilobular nodules to pulmonary fibrosis. The reaction to inhaled antigens leads to bronchiolocentric inflammation, which can be seen as ill-defined centrilobular ground-glass nodules on HRCT chest. (Figure 1a) Diffuse interstitial inflammation leads to an increase in lung density with visibility of vessels and bronchial walls and is called ground glass haze. (Figure 1b). In HP, lobular areas with ground glass are frequently intermixed with lobules of normal appearance, leading to a patchwork of lobules with differing density, which is called mosaic attenuation. (Figure 1c) Narrowing of the small airways leads to air trapping on expiratory scans. A patchwork of lung lobules with normal density, with lobules with ground-glass attenuation and lobules with decreased density and decreased vessel size due to air-trapping is called the “three-density pattern” and is the most specific sign for HP on HRCT. This pattern is more accentuated on expiratory scans.<sup>6</sup>

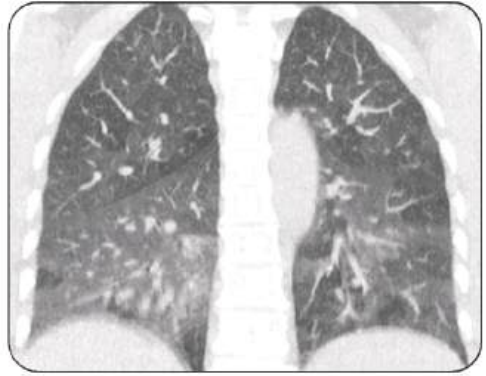
In Fibrotic HP, HRCT chest showed bilateral, peribronchovascular interstitial thickening, traction bronchiectasis, honeycombing with ground glass opacities, mosaic attenuation (three-density sign), diffuse or mid-to-upper zone distribution. (Figure 2 a and 2b) In Non-Fibrotic HP, HRCT chest showed bilateral, diffuse mosaic attenuation (including the three-density sign), ground glass opacities, features of small airways disease (small centrilobular ill-defined nodules; gas trapping on expiration).<sup>3</sup>



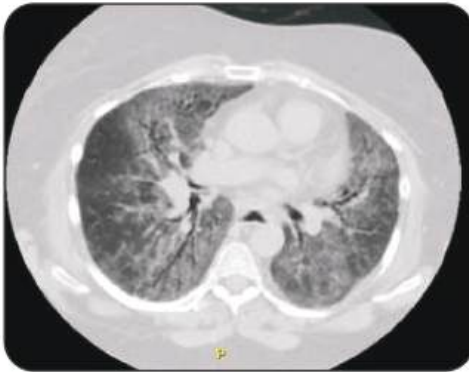
**Figure 1(a) showing centrilobular nodules with ground glass haze. Patient had HP secondary to Pigeon Fancier's Lung.**



**Figure 1b: Ground glass haziness**



**Figure 1c ; Mosaic attenuation along with ground glass haziness.**



**Figure 2a: HRCT chest showing bilateral, peribronchovascular interstitial thickening, traction bronchiectasis, mild honeycombing with ground glass opacities, mosaic attenuation (three-density sign).**



**Figure 2b: HRCT chest showing septal thickening, mosaic attenuation and honeycombing.**

## 2. Bronchoalveolar Lavage and Lung Biopsy

Bronchoalveolar lavage analysis and lung biopsy can be undertaken where diagnostic confidence remains low despite clinical history and HRCT. The ATS/JRS/ALAT guidelines,<sup>2</sup> suggest a confident diagnosis of HP also requires evidence of BAL lymphocytosis (BAL lymphocyte count >30%). Transbronchial lung biopsy (TBLB) or transbronchial lung cryobiopsy (TBLC) (depends on local expertise) or surgical Lung biopsy considered in Non-Fibrotic HP cases if diagnosis is uncertain.<sup>2</sup> However, Chest guidelines 2021 not routinely recommend BAL lymphocytosis if there is exposure history, clinical context and HRCT typical of HP due to low sensitivity and specificity to rule in or out in Fibrotic HP.<sup>4</sup> It may help to rule out infection particularly in non-fibrotic HP. Both ATS/JRS/ALAT and Chest guidelines do not recommend routine lung biopsy. Decision should only be made after multidisciplinary discussion based on risk benefit for individual patient. On Lung Biopsy pathologic findings characteristic of HP typically includes a combination of cellular and/or fibrosing interstitial pneumonia with bronchiolocentric accentuation, poorly formed non-necrotizing granulomas with or without giant cells, with or without peribronchiolar metaplasia, and/or small foci of organizing pneumonia.<sup>4</sup>

## OCCUPATIONAL (WORK-RELATED) HYPERSENSITIVITY PNEUMONITIS SCREENING QUESTIONNAIRE FOR CLINICIANS

While there is no single standardized or universally agreed-upon questionnaire for screening work-related hypersensitivity pneumonitis (HP), clinicians use a comprehensive history-taking approach, often guided by detailed lists of potential exposures. These questions aim at identifying potential triggers and patterns of symptoms as they evolve over time. The following questionnaire presents common questions and exposure categories to be used by clinicians while screening for HP.

### Work-Related HP Screening Questions

**1. Symptom Onset & Timing (Symptoms at Work)**

Do your symptoms (cough, shortness of breath, fever, chills, etc.) occur or worsen while you are at work?

**2. Symptom Improvement**

Do your symptoms improve or go away on weekends, days off, or during vacations (away from work), and return upon your return to work?

**3. Occupational Environment (Other Workers Affected)**

Are other people around you at work experiencing similar respiratory complaints or illnesses?

**4. Ventilation**

What type of ventilation system is used in your workplace (e.g., general ventilation or localized extraction)?

**5. On-site Observations**

Have you noticed any mould growth, significant dust accumulation, or pest problems (rodents, cockroaches) in your work area?

**6. Specific Exposures & Materials (Farming/Agriculture)**

Do you have regular exposure to farming, farm animals, hay, or damp straw/silage?

**7. Birds/Animals**

Are you exposed to pigeons, other birds, parakeets, chinchillas, or other animals/animal proteins?

**8. Fungi/Mould/Bacteria**

Are you exposed to areas with known mould, mushroom farming/preparation, or contaminants in humidifiers/air conditioners?

**9. Dusts (Organic/Inorganic)**

Do you work with industrial dusts, flours, wood dust, textiles, or chemical sprays/aerosols (e.g., metalworking fluids)?

**10. Chemicals**

Do you work with specific chemicals like isocyanates, pesticides, or components used in printing/bookmaking/upholstery?

**11. Vocational Exposure/Hobbies**

Do any of your hobbies involve raising animals, gardening, or exposure to significant dusts or chemicals?

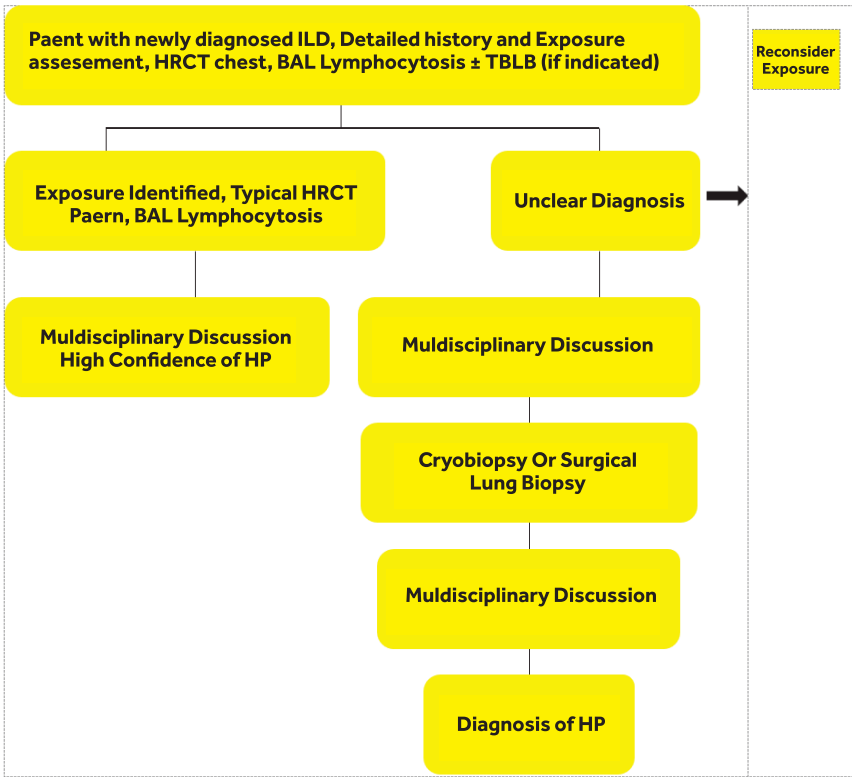
**12. Home Environment**

Do you use a humidifier or hot tub/jacuzzi regularly at home?

Do you notice mould at home?

A full clinical assessment involves integrating the exposure history with clinical findings, imaging, spirometry and lab tests.

Figure 1: Algorithm Adapted from ATS/JRS/ALAT Guidelines.<sup>2</sup>



### 3. Serology

Despite being a commonly used adjunct in the diagnostic workup of HP, serum antigen-specific IgG/IgA testing has several important limitations that restrict its diagnostic utility e.g; non-standardized antigen preparations, heterogeneous immunoassay methodologies, variable cutoff thresholds and limited population validation.

#### Clinical Implications:

- A positive antigen-specific IgG or IgA result indicates exposure but is not diagnostic of HP in isolation.
- Conversely, a negative test does not exclude HP, particularly if the relevant antigen is not included in the test panel.
- Due to cross-reactivity and lack of specificity, these assays are best used to support a diagnosis when integrated with:
  - Detailed exposure history
  - High-resolution chest CT findings
  - BAL lymphocytosis
  - Histopathology when indicated

For patients with suspected HP, chest guidelines<sup>4</sup> suggest do not depend solely on serum antigen-specific immunoglobulin G (IgG) or immunoglobulin A (IgA) testing to confirm or rule

out the diagnosis of HP. Similarly, patients with suspected HP, performing antigen-specific inhalation challenge testing to support the diagnosis of HP is not routinely recommended due to reasons stated above.

## **Treatment**

We recommend following Treatment Strategies:

### **1. Antigen Avoidance:**

The cornerstone of treatment is identification and elimination of the inciting antigen which improves the outcomes in patients with HP, but this can be difficult to achieve in practice sometimes.<sup>4</sup> This may involve changing of occupation, and/or work or home environments.

### **2. Corticosteroids:**

Steroids can reduce inflammation, especially in acute cases or when symptoms are severe. We recommend dose of 0.5-1 mg/kg/day for 1-2 weeks in acute HP or 4-8 weeks for subacute/chronic HP followed by a gradual taper to off or maintenance dosage of approximately 10 mg/day. Patient should be evaluated for complications of steroids (e.g., diabetes, hypertension, cataract formation, osteoporosis). Bone mineral-preserving therapies and pneumocystis prophylaxis is strongly recommended.

### **3. Immunosuppressants:**

If prolonged corticosteroid treatment is required, or if side effects are intolerable, addition of corticosteroid-sparing drugs may be considered (e.g., azathioprine, and mycophenolate mofetil). The side effects should be monitored by liver function tests and complete blood count for transaminitis and leukopenia, respectively.

### **4. Antifibrotic Medications:**

For progressive lung fibrosis, nintedanib or pirfenidone may be used.

### **5. Pulmonary Rehabilitation:**

It can help improve breathing and overall fitness, like exercise endurance, capacity, and quality of life.

### **6. Oxygen Therapy:**

Provide supplemental oxygen to maintain adequate oxygen saturation in hypoxic patients.

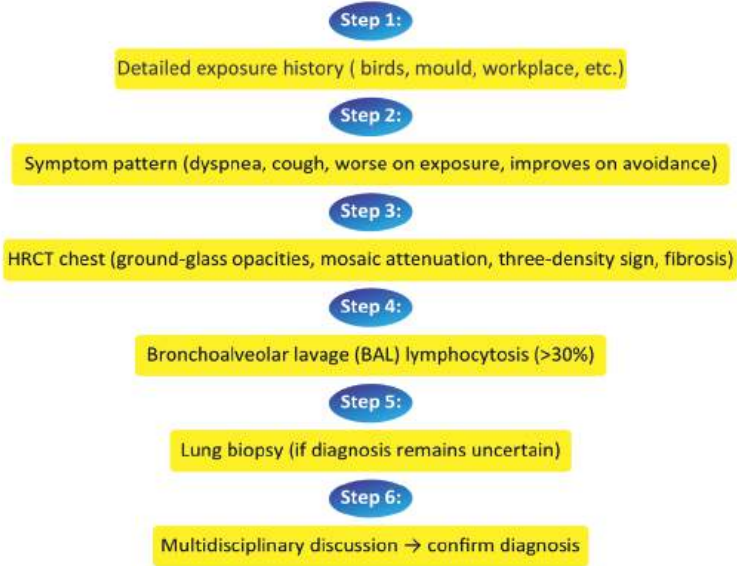
### **7. Lung Transplantation:**

Lung transplantation may be considered in patients with >50% risk of death from their disease within 2 years, and >80% likelihood of 5-year post-transplant survival provided there is adequate graft function if transplanted. (8) Retrospective cohort studies of transplanted HP patients have shown survival rates of 85%–89%, 75%–84% and 70%–84% at 1, 3 and 5 years post-transplant, respectively.<sup>9,10</sup>

Other management includes, smoking cessation, vaccinations and appropriate treatment of comorbid conditions (e.g., coronary artery disease, sleep apnea, obesity, GERD, pulmonary hypertension); disease education, symptom management (e.g., cough) and palliative care when appropriate.

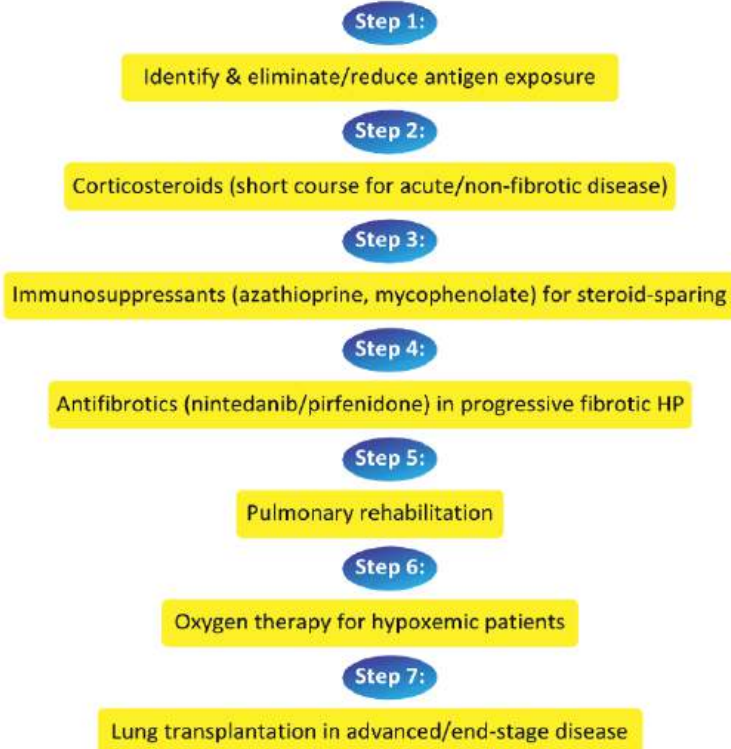
## Algorithm: Hypersensitivity Pneumonitis (HP)

### Diagnosis



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### Management



## Conclusion

HP is typically an immune-mediated disease that manifests as ILD in susceptible individuals after exposure to an identified or unidentified factor. Currently large number of patients are being reported as HP due to avian exposure in Pakistan. HP is broadly classified as fibrotic and non fibrotic HP. A confident diagnosis of HP can be made in patients who have an identified exposure and a typical HP pattern on a high-resolution computed tomography (HRCT) scan. Bronchoalveolar lavage analysis and lung biopsy can be undertaken where diagnostic confidence is low. Multidisciplinary discussion helps in making diagnosis and invasive testing decisions if needed.

## References

1. Zubairi ABS, Ansarie M, Mahmud T, Ashraf S, Rao NA, Javaid A, Shaheen Z, Khan S, Khan A. National Registry of Interstitial Lung Disease from Pakistan. *Cureus*. 2021 Apr 25;13(4):e14684. doi: 10.7759/cureus.14684. PMID: 34055529; PMCID: PMC8149777.
2. Raghu G, Remy-Jardin M, Ryerson CJ, et al. Diagnosis of hypersensitivity pneumonitis in adults. An official ATS/JRS/ALAT clinical practice guideline. *Am J Respir Crit Care Med* 2020; 202:e36-e69. doi:10.1164/rccm.202005-2032ST
3. Barnes H, Corte TJ, Keir G, Khor YH, Limaye S, Wrobel JP, et al. Diagnosis and management of hypersensitivity pneumonitis in adults: A position statement from the Thoracic Society Australia and New Zealand. *Respirology*. 2024; 29(12): 1023-1046. <https://doi.org/10.1111/resp.14847>
4. Fernández Pérez ER, Travis WD, Lynch DA, et al. Executive summary diagnosis and evaluation of hypersensitivity pneumonitis: CHEST guideline and expert panel report. *Chest* 2021; 160:595-615. doi:10.1016/j.chest.2021.03.067
5. Selman M, Buendía-Roldán I. Immunopathology, diagnosis, and management of hypersensitivity pneumonitis. *Semin Respir Crit Care Med*. 2012 Oct;33(5):543-54. doi: 10.1055/s-0032-1325163. Epub 2012 Sep 21. PMID: 23001807.
6. Hamblin M, Prosch H, Vašáková M. Diagnosis, course and management of hypersensitivity pneumonitis. *Eur Respir Rev*. 2022 Feb 9;31(163):210169. doi: 10.1183/16000617.0169-2021. PMID: 35140104; PMCID: PMC9488722.
7. Lacasse Y, Selman M, Costabel U, Dalphin JC, Ando M, Morell F, et al.; Group. Clinical diagnosis of hypersensitivity pneumonitis. *Am J Respir Crit Care Med* 2003;168:952-958.
8. Leard LE, Holm AM, Valapour M, Glanville AR, Attawar S, Aversa M, et al. Consensus document for the selection of lung transplant candidates: an update from the International Society for Heart and Lung Transplantation. *J Heart Lung Transplant*. 2021; 40: 1349-1379
9. Nosotti M, Leiva-Juarez M, D'Ovidio F, Van Raemdonck D, Ceulemans L, Keshavjee S, et al. Survival after lung transplantation for chronic hypersensitivity pneumonitis: results from a large international cohort study. *Transpl Int*. 2022; 35:10450.
10. Kern RM, Singer JP, Koth L, Mooney J, Golden J, Hays S, et al. Lung transplantation for hypersensitivity pneumonitis. *Chest*. 2015; 147: 1558-1565.

# Occupational Asthma

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Author: Dr. Ibrahim Ali Khan

Occupational asthma (OA) is a form of work-related asthma caused or worsened by exposures in the workplace. It presents with variable airflow obstruction, airway hyperresponsiveness, and airway inflammation attributable to specific occupational agents. OA accounts for an estimated 10–25% of adult-onset asthma cases and is an important, preventable cause of chronic respiratory morbidity. This guideline provides an evidence-based framework for recognition, diagnosis, management, and prevention of occupational asthma.

## Definition

Occupational asthma (OA) is characterized by variable airflow obstruction, heightened airway responsiveness, and airway inflammation directly attributable to exposures in the workplace and not due to environmental stimuli encountered outside of work. It is potentially preventable and may be reversible if recognized early and exposure discontinued.

## Types of the Disease

- 1 Sensitizer-Induced Occupational Asthma:
  - Results from repeated exposure to a specific allergen (IgE-mediated or non-IgE-mediated).
  - Develops after a latency period lasting from weeks to several years.
- 2 Irritant-Induced Occupational Asthma (Reactive Airways Dysfunction Syndrome – RADS):
  - Triggered by a single or repeated high-level exposure to an irritant.
  - Lacks a latency period; symptoms develop within 24–48 hours.
- 3 Work-Exacerbated Asthma:
  - Pre-existing asthma aggravated by workplace exposures such as dust, fumes, or cold air.

## Epidemiology & Burden

Occupational asthma contributes substantially to adult-onset asthma. Under-recognition and inadequate surveillance systems, particularly within informal labor sectors, lead to underestimation of its true burden. Estimated prevalence in high-risk sectors (regional data):

- Textile workers: 8–12%
- Flour mill workers: 5–10%
- Healthcare workers: 4–7%

Higher prevalence is reported in industries with high exposure to sensitizers (e.g., isocyanates, flour dust). Males predominate in heavy industrial exposures; females are more affected in textile and healthcare settings.

## Etiology & Risk Factors

Occupational asthma results from exposure to a wide range of sensitizing and irritant agents. Risk factors include:

- High-intensity or prolonged exposure to sensitizers (e.g., isocyanates, flour, animal dander).
- Single or multiple high-level irritant exposures (for RADS).
- Lack of engineering controls, inadequate PPE, and poor ventilation.
- Smoking and atopy may increase susceptibility.

## Sources of the Causative Agents

Source	Agents
Organic dusts	Flour, cotton, grain dust, animal dander
Chemicals	Isocyanates, formaldehyde, glutaraldehyde
Fumes/Gases	Welding fumes, chlorine, ammonia
Biological agents	Fungal spores, mites, enzymes
Drugs/Enzymes	Antibiotics (penicillin), amylase

Occupation	Agent / Exposure
Bakers, Flour mill workers	Flour, mold, grain dust
Textile workers	Cotton dust, dyes, formaldehyde
Welders, Metal workers	Chromium, nickel, welding fumes
Healthcare workers	Latex, disinfectants (glutaraldehyde)
Agricultural workers	Pesticides, animal dander, hay dust
Painters	Isocyanates, solvents
Brick kiln workers	Smoke, inorganic dusts

## Occupations/Environments Associated with Exposure

### Brief Pathophysiology

Sensitizer-Induced OA: Repeated exposure → IgE or T-cell mediated immune response → airway inflammation → bronchial hyperresponsiveness → variable airflow obstruction.

Irritant-Induced OA (RADS): Single high-dose irritant exposure → direct epithelial injury → non-specific airway hyperreactivity → persistent symptoms.

Work-Exacerbated Asthma: Exacerbation of pre-existing asthma by workplace triggers through enhancement of baseline airway inflammation.

### Symptoms & Signs

Typical symptoms (worse during work shifts or workdays):

- Wheeze
- Dry cough
- Chest tightness
- Breathlessness
- Night-time symptoms and variability with work exposure

Signs may include prolonged expiration and wheeze on auscultation; examinations may be normal between exposures.

## Diagnostic Workup

### Clinical Evaluation:

1. Comprehensive occupational history including tasks, agents, duration, and temporal relation of symptoms to work.
2. Correlation of symptom patterns with work schedule.
3. Inquiry about similar symptoms in coworkers or workplace clusters.

### Objective Testing

Test	Purpose
Spirometry with bronchodilator	Baseline airflow limitation and reversibility
Serial Peak Expiratory Flow Rate (PEFR)	Compare work vs off-work; $\geq 2$ weeks recommended
Methacholine challenge	Detect bronchial hyperresponsiveness
Skin prick / specific IgE (RAST)	Detect specific sensitization to workplace allergens
Chest X-ray	Exclude alternate diagnoses
Specific inhalation challenge (if available)	Gold standard diagnostic test in specialized centres

## Management

### A. General Measures:

- Immediate cessation or reduction of exposure; reassignment to low-exposure roles if necessary.
- Smoking cessation.
- Optimize workplace ventilation and engineering controls.

### B. Pharmacological Therapy:

Drug/Intervention	Purpose
Inhaled corticosteroids (ICS)	Long-term control; reduce airway inflammation
LABA + ICS	Moderate-to-severe cases requiring combination therapy
Leukotriene receptor antagonists	Add-on therapy for symptom control
Short-acting beta-agonists (SABA)	Relief of acute symptoms

### C. Environmental/Occupational Measures:

- Eliminate or reduce exposure at source.
- Use PPE and provide worker training.
- Reassign workers to lower-exposure tasks where feasible.
- Substitute hazardous substances with safer alternatives when possible.

### D. Education & Prevention:

- Train workers on hazard recognition and avoidance.
- Implement regular medical screening for at-risk workers.
- Ensure compliance with occupational safety legislation and guidelines.

## Occupational (Work-Related) Asthma Screening Questionnaire for Clinicians

The following questions help identify potential cases of work-related asthma. A positive response to these questions indicates the need for further evaluation by a specialist.

### 1-Asthma Symptoms

- Have you ever been diagnosed with asthma?
- Do you currently experience symptoms such as wheezing, coughing, chest tightness, or shortness of breath?

### 2-Work Relationship

- Did your asthma symptoms start at work?
- Do/did your asthma symptoms worsen at work?
- Do/did your asthma symptoms worsen on your first day back to work after a break (e.g., "weekend, holiday")?
- Do/did your asthma symptoms worsen throughout the work week?
- Are/were chest symptoms (cough/wheeze/chest tightness/shortness of breath) less on days off work and/or holidays?

### 3-Occupational History

- What type of industry do you work in?
- Are you exposed to dusts, gases, or chemical fumes in your workplace?
- Are others at work experiencing similar symptoms?

Healthcare providers use patient responses to develop an initial assessment and determine if a referral for objective testing (such as serial peak expiratory flow (PEF) measurements, spirometry or specific inhalation challenges) is required.

### Algorithm: Occupational Asthma Diagnosis

**Step 1:** Occupational history

**Step 2:** Symptom timing (worse on Mondays/workdays)

**Step 3:** Spirometry (obstruction ± restriction)

**Step 4:** Radiology (if advanced disease suspected)

**Step 5:** Exclude differentials

**Step 6:** Apply Schilling/WHO grading

## Management

- Step 1:** Confirm diagnosis
- Step 2:** Remove or reduce exposure
- Step 3:** Initiate bronchodilators
- Step 4:** Add inhaled corticosteroids (ICS) if persistent symptoms
- Step 5:** Pulmonary rehabilitation
- Step 6:** Oxygen therapy in advanced disease
- Step 7:** Refer for specialist care if needed

### Key Recommendations:

- Early recognition of workplace exposure and symptom-onset patterns.
- Use of detailed occupational history, spirometry, and serial peak flow or challenge testing for diagnosis.
- Removal from exposure and exposure control as primary management strategies.
- Pharmacotherapy (inhaled corticosteroids + LABA) for symptomatic control and prevention of long-term impairment.
- Regular medical surveillance and worker education in high-risk industries.

### References

1. Global Initiative for Asthma (GINA). Global Strategy for Asthma Management and Prevention. 2024.
2. Tarlo SM, Malo JL. An official American Thoracic Society Statement: Occupational contribution to the burden of airway disease. *Am J Respir Crit Care Med.* 2008;178(8):693-698.
3. Canadian Centre for Occupational Health and Safety (CCOHS). Asthma, Work-related. 2025.
4. Memon Y, et al. Prevalence of occupational asthma among textile workers in Karachi. *J Pak Med Assoc.* 2014;64(3):309-314.
5. Fishwick D, Sen D, Barber CM. Occupational asthma: Identification, management, and prevention. *Clin Med (Lond).* 2015;15(6):573-577.
6. Khan A, et al. Occupational lung diseases in Pakistan: Current situation and challenges. *Pak J Chest Med.* 2020;26(1):1-8.

# Asbestos Related Lung/pleural Diseases: An Overview

Author: Dr. Mohammad Yousaf Khan

Asbestos is a generic name for a variety of silicate minerals that occur naturally in a fibrous form. Asbestos was considered a miracle mineral in the past, being resistant to fire, heat, and corrosion. Moreover, it is strong, durable, flexible and inexpensive. It has been used in the manufacturing of over 3000 factory applications and many products. Asbestos has been declared as Group 1A carcinogen by the WHO in 1987. Moreover, asbestos can cause both benign and malignant diseases of the lungs and pleura. A relatively intense exposure is needed for lung diseases (asbestosis and lung cancer) compared to pleural diseases. This prompted occupational health experts to formulate guidelines about clinical, radiological, functional and pathological assessment of asbestos-related diseases.

As shown in Table 1, the asbestos minerals can be divided into two distinct groups, serpentine and amphiboles.<sup>1,2,3</sup>

**Table 1. Types of Asbestos**

Serpentine (Spiral Fibres)	Purpose
Chrysotile (White Asbestos)	Crocidolite (Blue Asbestos) Amosite (Brown Asbestos) Tremolite Actinolite Anthophyllit

## Difference Between Chrysotile and Amphibole Asbestos: Characteristics & Half-lives of Fibres

Chrysotile is a magnesium silicate. Larger and longer fibers of chrysotile are often curved. Chrysotile is relatively unstable in the lung. It fragments readily into short fibres and fibrils that are easily phagocytized by macrophages. Chrysotile is particularly unstable in acid environments, such as the macrophage phagolysosome, and dissolves by progressive removal of both magnesium and silicon from the fiber. The clearance half-life in humans is in the range of a few weeks to a few months. However, some chrysotile fibres persist, perhaps because they are sequestered in fibrous tissue and thus protected from dissolution.<sup>1,2,3</sup>

The amphiboles are a large mineral group which cleaves into very long, thin, fibres. As opposed to chrysotile, amphiboles are stable within the lung, they do not fragment and are not sensitive to inflammatory macrophage and chemical attack. Thus, clearance half-lives for amphiboles are much longer than for chrysotile and extend on decades

## Importance of Fibre Type, Size And Durability

In addition to fibre size, the persistence of a fibre within the lung is a crucial determinant of its pathogenicity. Although most focus has been on the induction of mesothelioma, durable

amphibole fibres induce more fibrosis (asbestosis) than do chrysotile fibers. Asbestosis appears at much lower fibre burdens in individuals with amphibole exposure than in individuals with chrysotile exposure. The persistence of amphibole fibers in the lung is believed to be the reason workers with asbestosis due to amphibole exposure are more likely to see progression of their disease than workers with asbestosis due to chrysotile exposure.<sup>2,3,4</sup>

### **Fibre Count & Asbestos Related Diseases & Grades of Asbestosis**

Subjects with asbestosis have higher asbestos fiber counts than those with pleural plaques or mesothelioma. Amphibole, but not chrysotile, asbestos fibre counts also have a correlation with the grades of asbestosis. This is not surprising because chrysotile fibres clear relatively rapidly from the lung, whereas amphibole fibres tend to persist for many years.<sup>2,3,4</sup>

### **Forms of Asbestos-exposure And Importance of Background Environmental Asbestos**

Asbestos exposure has 3 main forms. It can occur through work, bystander exposure, or general community exposure. The risk of disease is higher for people who are directly exposed to asbestos at work.

Asbestos is ubiquitous and is always likely to be found in the lungs of the general population. It is, therefore, important that results found in a specific case be compared with those for the background population. For this reason, control ranges need to be established for each laboratory performing these analyses.<sup>2,3,4</sup>

### **Commonly Used Asbestos-Containing Products**

Manufacturers commonly use asbestos in the following products:

- Products containing asbestos cement: Pipes, shingles, clapboards, sheets
- Vinyl-asbestos floor tiles
- Asbestos paper in filtration and insulation products
- Material in brake linings and clutch facings
- Textile products: Yarn, felt, tape, cord, rope
- Spray products used for acoustic, thermal, and fireproofing purposes

### **Occupations Associated with Highest Risk for Asbestos Exposure**

Occupations that were traditionally associated with highest risk of exposure include construction workers (insulators, pipefitters, carpenters, electricians, roofers, drywall workers), maintenance workers, shipyard workers and Navy personnel, boilermakers and furnace workers, auto brake mechanics, and individuals involved with asbestos mining and processing.

### **The Basic Assessment for Differentiating Asbestos From Non-Asbestos Exposures**

The basic assessment of the type of dust includes differentiating ferruginous asbestos bodies from pseudo asbestos bodies. Ferruginous bodies are formed on the transparent, fibrous cores typical of asbestos bodies, while pseudo asbestos bodies are formed on non-asbestos minerals, such as carbon, iron, aluminum, chromium oxide, talc, and glass.<sup>2,3</sup>

### **Factors Determining Asbestos Body Formation**

Ferruginous body formation varies according to a number of factors including fiber type, length, concentration, and quantity of iron within the lung. Thus, the quantification of asbestos bodies may not accurately reflect the total concentration of mineral fibres in the lungs.<sup>2</sup>

### **Importance of Tissue Selection For Fibre Count**

Both formalin-fixed and fresh lung tissue can be used for these procedures. Within one laboratory, different samples from the same individual may differ 3-fold and up to 10-fold in very small samples. Consequently, analyses of transbronchial samples should only be undertaken in exceptional circumstances and the results interpreted with considerable caution. Optimal samples for mineral analysis comprise 2-cm<sup>3</sup> block from 3 anatomic sites in the contralateral lung (in tumor cases) or either side (in nontumor cases), usually from the apical upper, apical lower, and basal segments of the lung. To be representative, the samples should not contain tumors or large areas of pleural fibrosis. Mineral fibre analysis can also be carried out on bronchoalveolar lavage samples.

# Asbestosis

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## **Definition**

Asbestosis is defined as diffuse pulmonary fibrosis caused by the inhalation of excessive amounts of asbestos fiber dust. Pathologically, both pulmonary fibrosis of a particular pattern and evidence of excess asbestos in the lungs must be present. Clinically, the disease usually progresses slowly, with an average latent period of more than 15 years from initial exposure to onset of symptoms.

## **An Overview**

All types of asbestos have been implicated in the causation of asbestosis to a lesser or greater degree. There appears to be a dose-response relationship between the concentration of fibres in the lung and the severity of fibrosis. Asbestosis typically occurs in individuals with prolonged and heavy exposure to asbestos, and the disease may progress even after exposure has ceased. The diagnosis of asbestosis is usually based on exposure history, clinical findings, and radiographic features. Biopsy is seldom required, but if thought ethically acceptable, it may be helpful when differential diagnostic considerations can not be resolved on clinical or radiographic grounds alone.

Accelerated asbestosis: Asbestosis typically has a latency period of 15 years or more from initial exposure to diagnosis. However, some of the early cases evolve much more rapidly due to high exposures. Such cases generally exhibited small fibrotic lungs without honeycomb changes but with numerous asbestos bodies in histologic sections.

Accelerated asbestosis is the term used for such cases. Although accelerated asbestosis is now rarely encountered in industrialized nations, such disease is likely to develop if asbestos dust control is neglected, which is especially the case in developing countries.

## **Clinical Presentation of Asbestosis**

It ranges from asymptomatic disease, identified on radiologic investigation or by histologic assessment of lung parenchyma remote from a resected lung cancer to a symptomatic and progressive disease. The clinical features are identical to those encountered with other forms of diffuse interstitial lung disease, such as usual interstitial pneumonia (UIP). Its usual presentation is an insidious onset of breathlessness and dry cough. There may also be chest pain related to associated disorders, such as benign asbestos pleuritis or, in patients with severe breathlessness, respiratory muscle fatigue from the increased respiratory work required to inflate fibrotic lung. Bibasal dry crackles. Finger clubbing or central cyanosis are late features and may or may not be present.

## **Diagnostic Criteria for Asbestosis**

### **The Diagnostic Gold Standard**

The diagnostic gold standard is the histopathological examination of lung tissue (diffuse fibrosis and at least 2 asbestos bodies per  $\text{cm}^3$  of tissue). However, most cases of asbestosis are diagnosed on clinical and radiologic grounds, without histologic examination, from the following findings:<sup>2,3</sup>

1. A history of moderate to heavy asbestos exposure, typically occupational and often protracted for many years. In general, when the cumulative exposure has been substantial to heavy, the likelihood of clinical asbestosis and its severity are correspondingly greater, with a shorter latency interval between the commencement of exposure and the subsequent symptomatic onset of the disease.
2. Clinical signs of interstitial fibrosis in the form of end-inspiratory crackles on auscultation of the lung fields, especially in the lower zones.
3. Detection of reticular, linear, diffuse opacities in the lower zones of the lung fields on radiologic examination.
4. Classically, restrictive impairment of lung function.
5. Usually, but not always, associated parietal pleural fibrous plaques and/or diffuse pleural fibrosis.

**Criteria 1 and 3 are obligatory for the clinical diagnosis, which is further supported by criterion 5.**

### **Pulmonary Function Tests**

Pulmonary function tests usually demonstrate a restrictive impairment of lung function with a reduction of lung volume and forced vital capacity, diminished carbon monoxide diffusing capacity (DLCO), and often hypoxemia on arterial gas analysis.

Of greater significance is longitudinal assessment of lung volumes, with a greater than average, age-related decline for 3 to 5 years (with at least 3 serial measurements). A reduction in DLCO during serial measurements appears to be a more useful assessment, but it simply reflects a loss of parenchymal function by emphysema. Coexistent airflow obstruction with reduced forced expiratory volume in 1 second (FEV1) is often due to concomitant tobacco smoking. Mild airflow obstruction independent of smoking, as shown by a reduced FEV1, can be seen in some workers.<sup>2,3</sup>

### **Radiological Diagnosis Chest X-Ray (CXR)**

CXR is available worldwide and there is an accepted CXR-based classification scheme for the evaluation of the pneumoconiosis. The CXR findings are normal in 10% to 18% of patients with biopsy-proven asbestosis, and at least east 10% of pleural disease is not identified on CXR. Despite these problems associated with the chest radiograph, it is still useful as a diagnostic tool in patients with characteristic, extensive disease and a confirmed history of exposure.

Asbestosis typically presents with small, irregular opacities at the lung bases. As the disease progresses, these opacities coalesce and become coarser, ultimately leading to a honeycomb pattern of small cysts. Association with pleural plaques increases the specificity of the diagnosis. Extensive pleural disease is readily identified on chest radiography. However, the pleural density in such cases prevents accurate assessment of the underlying lung parenchyma. This problem, along with poor interobserver agreement between expert readers in patients with minimal disease, has highlighted the benefits of high-resolution computed tomography (HRCT).<sup>2,3</sup>

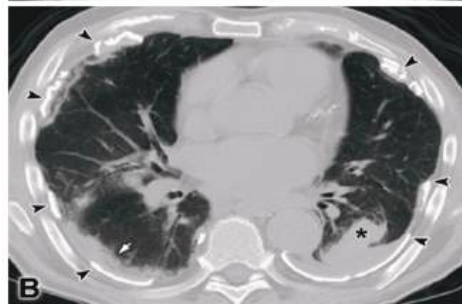
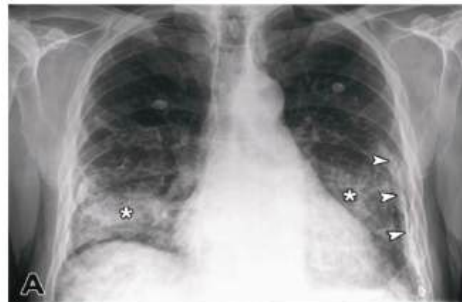


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Posterior-anterior chest radiograph with limited view of the right lower lobe in a patient with asbestosis demonstrating small, irregular opacities. There is a calcified pleural plaque (arrowhead) projecting within the central tendon of the right hemidiaphragm.<sup>2</sup>

A, Posterior-anterior (PA) chest radiograph demonstrates extensive, calcified pleural plaques in both profile (arrowheads) and en face (asterisks).

B, Axial chest computed-tomography slice through the lower lobes demonstrates a variety of parenchymal pathology not visualized on the PA chest radiograph, including round atelectasis (asterisk), calcified pleural plaques (arrowheads), and fibrosis (arrow).



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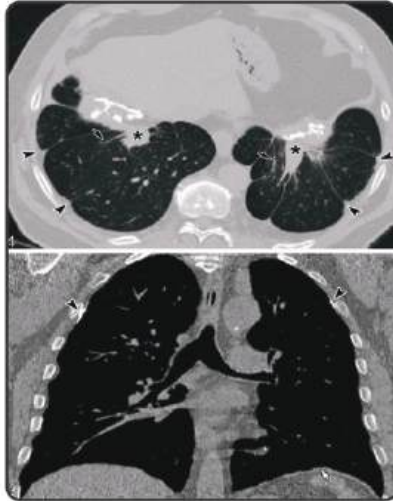
### Computed Tomography (CT)

Conventional CT and HRCT are more sensitive and specific than chest radiography in the diagnosis of both parenchymal and pleural diseases related to asbestos. HRCT is most useful when the chest radiographic findings are normal or minimally abnormal.

HRCT findings associated with asbestosis include peripheral intralobular and interlobular lines, ground-glass attenuation, and honeycombing. Diffuse fibrotic involvement of the visceral pleura is seen as widespread pleural thickening on computed tomography and is associated with parenchymal bands and rounded atelectasis. Parietal pleural involvement shows well-circumscribed areas of pleural thickening and correlates with the presence of

pleural plaques. Despite the increased sensitivity of HRCT compared with CXR, it is important to recognize that the finding of a normal examination by computed tomography does not exclude the presence of asbestosis.<sup>2,3</sup>

HRCT is an important tool for quantification of fibrosis as well as for clarifying the role of emphysema in dyspneic cigarette smokers who have a history of asbestos exposure. HRCT has the ability to quantify the contribution that asbestosis, diffuse pleural thickening, and emphysema makes in the physiological deficit and dyspnea suffered by an individual patient.



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Axial chest computed tomography at the level of the dome of the diaphragm in a patient with diffuse visceral pleural fibrosis shows bilateral pleural thickening (arrowheads), parenchymal bands (arrows), and areas of round atelectasis (asterisks). Coronal reconstruction of axial high-resolution computed tomography data demonstrates subtle, parietal pleural plaques, both calcified (arrowheads) and noncalcified (arrow)<sup>2</sup>

### **Bronchoalveolar Lavage**

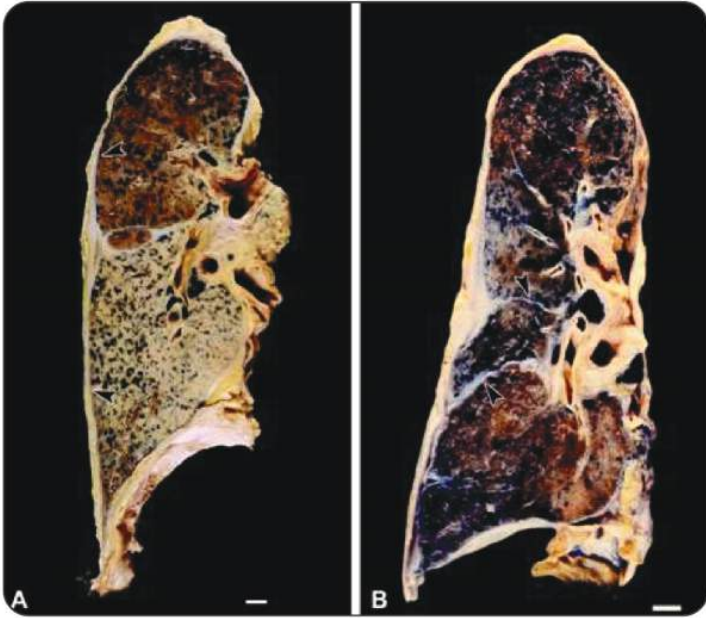
Bronchoalveolar lavage is potentially useful when the clinico radiologic diagnosis is uncertain. The demonstration of asbestos fibers and/or asbestos bodies can help support the diagnosis but is not necessary.<sup>23</sup>

### **Pathology of Asbestosis**

#### **Gross Pathology**

The disease ranges in severity from subtle findings that are not visible macroscopically to advanced diseases characterized by small, fibrotic lungs with honeycombing. The changes tend to be more severe in the lower lobes and periphery of the lung. The honeycomb changes seen in advanced cases is represented by subpleural cysts measuring up to 15 mm in diameter. Airways are generally unremarkable, although there may be areas of traction bronchiectasis in severely fibrotic areas. Lymph nodes may be enlarged. A minor degree of diffuse, visceral pleural fibrosis is commonly seen. Most cases of asbestosis are associated

with parietal pleural plaques.<sup>2</sup>

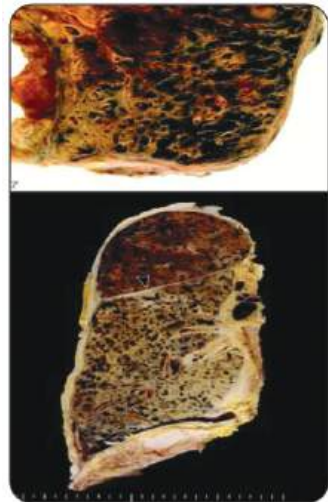


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- A. Right lung of a patient who was a crocidolite asbestos sprayer demonstrates advanced asbestosis that is most severe in the lower lobe. In addition, there is marked thickening of the visceral pleura (arrowheads).
- B. Right lung of a 71-year-old patient with asbestosis who was an amosite and crocidolite asbestos sprayer. There is thickening of the visceral pleura that extends into the fissures (arrowheads).<sup>2</sup>

Top figure: This example of asbestosis shows honeycomb changes, with cysts up to about 15 mm in diameter. There is also diffuse visceral pleural fibrosis with blunting of the costophrenic angle.

Bottom figure: In this patient with advanced asbestosis, there is diffuse visceral pleural fibrosis surrounding the lung and extending into the fissure (arrowhead).<sup>2</sup>



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### Sampling Techniques for Histologic Examination

Sampling technique for histologic examination of cases of asbestosis is determined by the specimen used for histopathology. For autopsy specimens, minimal sampling should include blocks from the peripheral and central portions of each lobe of both lungs. For pneumonectomy specimens, samples should include peripheral and central blocks from each lobe. For lobectomy specimens, at least one peripheral and one central section should be sampled. Areas immediately adjacent to tumors should be avoided.<sup>2</sup>

### Histologic Diagnosis

The histopathological diagnosis of asbestosis requires an appropriate pattern of interstitial fibrosis plus the finding of asbestos bodies. Both components must be present. Fibrosis in asbestosis lacks any significant degree of inflammation, and is collagenous rather than fibroblastic.<sup>2</sup>

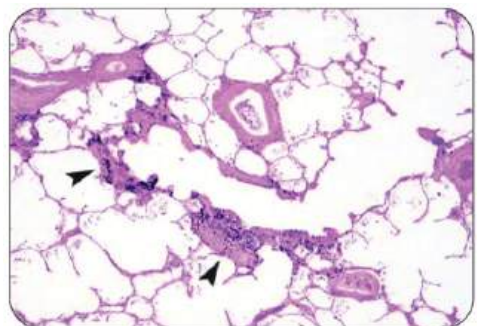
### Histological Pattern of Interstitial Fibrosis in Asbestosis

In early asbestosis, the fibrosing process is limited to the walls of alveoli immediately around the bronchioles and is called peribronchiolar pattern. From this the fibrosis extends and ultimately links adjacent bronchioles. At this time, the predominantly peribronchiolar pattern of fibrosis may be lost.

Initially, the disease is predominantly subpleural, but as it advances, the fibrosis extends inward a considerable distance from the pleura. At that stage, a variety of morphologic patterns may be seen. Some cases resemble usual interstitial pneumonitis (UIP), whereas others are more like fibrotic, nonspecific interstitial pneumonia (NSIP), and still others do not match any other form of interstitial fibrosis. Fibroblast foci are seen only occasionally. If these foci of immature fibrosis are at all conspicuous, another diagnosis (such as UIP) should be considered. Honeycombing may be seen in advanced cases, but it is seldom as severe as in UIP.

**Asbestos Airway Disease:** Asbestos exposure may at time be associated with fibrosis of the walls of the respiratory bronchioles which is sometimes incorrectly referred to as peribronchiolar fibrosis and, therefore, is liable to be confused with the fibrosis of the alveolar walls around the bronchioles, which is truly peribronchiolar. A better term in such cases is bronchiolar wall fibrosis. If bronchiolar wall fibrosis is associated with asbestos bodies, the term asbestos airway disease is preferred. These are the cases where an obstructive pattern is seen on spirometry.<sup>2,3</sup>

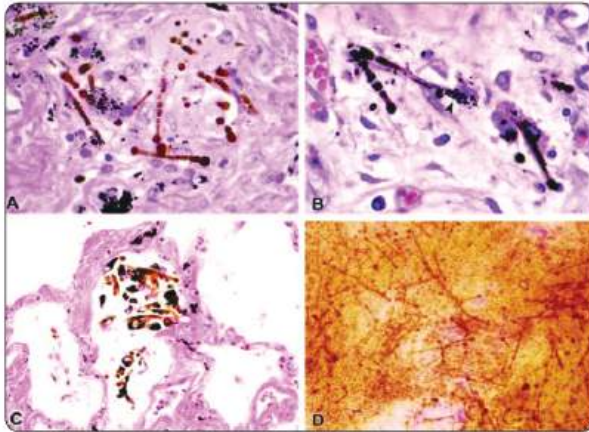
Asbestos airways disease consists of bronchiolar wall fibrosis (arrowheads) without fibrotic thickening of alveolar septa. This pattern should not be referred to as asbestosis (hematoxylin-eosin, original magnification  $\times 70$ ).<sup>2</sup>



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## Asbestos Bodies

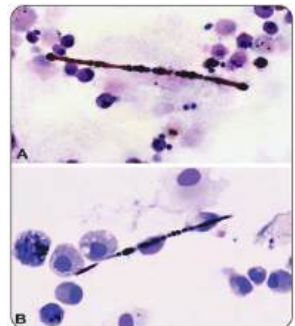
The second feature necessary for a histologic diagnosis of asbestosis is the finding of asbestos bodies. Asbestos bodies are golden-brown, beaded, or dumbbell-shaped structures with a thin, translucent core. They are formed from the deposition of an iron-protein-mucopolysaccharide coating on the surface of an inhaled asbestos fibre by alveolar macrophages. In asbestosis, these bodies are typically found embedded within fibrous tissue, but they may also be observed within alveolar spaces or within the cytoplasm of macrophages or multinucleate giant cells. Asbestos bodies may also be observed within hilar lymph nodes, but that does not constitute asbestosis. The finding of asbestos bodies alone is insufficient for a histologic diagnosis of asbestosis and indicates only asbestos exposure.



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- A. In this case of asbestosis from a crocidolite sprayer, asbestos bodies are embedded within the fibrous tissue. They consist of golden-brown, beaded structures with a thin, translucent core.
- B. Asbestos bodies in this case of asbestosis are embedded within fibrous tissue and are associated with pigment deposition (arrowhead).
- C. Asbestos bodies in this case from an amosite asbestos miner are lying free within the alveolar space.
- D. Numerous asbestos bodies are present within this sputum cytology sample from an insulator (hematoxylin-cosin, original magnifications  $\times 440$  [A and B] and  $\times 150$  [C]; Papanicolaou stain, original magnification  $\times 150$  [D]).<sup>2</sup>

A and B, Asbestos bodies with beaded, ferruginous coating and thin, translucent core are present within bronchoalveolar lavage fluid (Wright-Giemsa, original magnifications  $\times 440$  [A] and  $\times 880$  [B]).<sup>2</sup>



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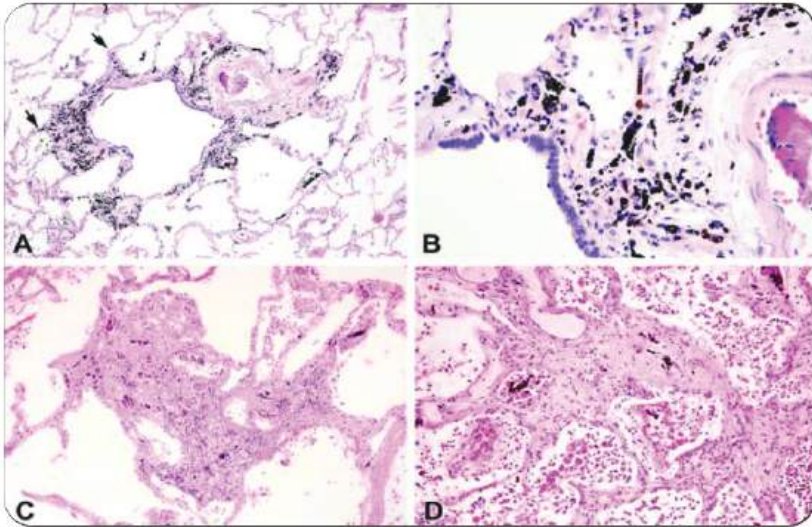
### Asbestos Fibre Analysis for The Diagnosis Of Asbestosis

In difficult cases, fibre analysis may be necessary to determine the etiology of the fibrotic process. Fibre analysis should be considered as an adjunctive technique in the assessment of asbestosis and cannot substitute the histopathologic diagnosis of asbestosis. Patients with asbestosis have in excess of a million fibres per gram of dry lung tissue.<sup>2,4</sup>

### Histologic Grading Scheme for The Extent and Severity of Asbestosis

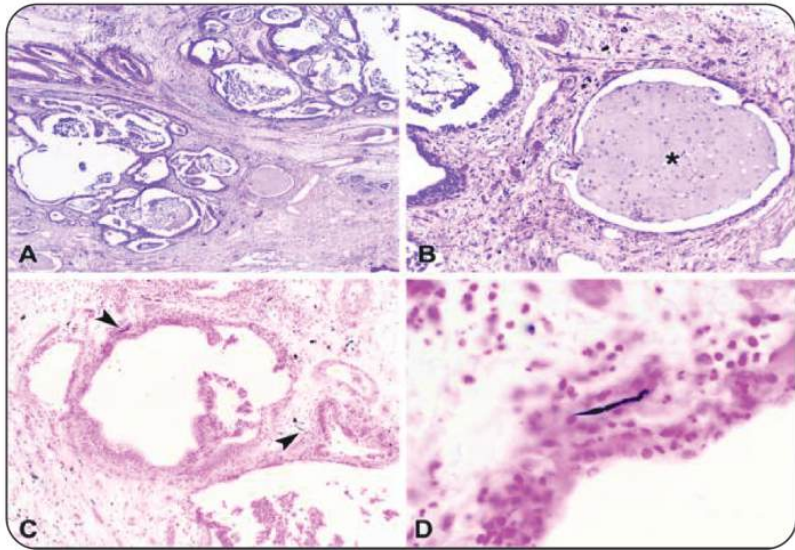
number of schemes have been proposed. These may be of particular value in epidemiologic studies. A 12-point grading scheme is proposed by the College of American Pathologists and National Institute for Occupational Safety (CAP-NIOSH). A modified, 4-point grading system of histologic criteria, to describe severity and extent of asbestosis includes:

- Grade I involving alveolar walls of respiratory bronchioles and the alveolar ducts;
- Grade II involving greater proportions of acini;
- Grade III involving the whole acinar structure;
- and Grade IV when honeycomb remodeling and large (up to 1 cm) dilated spaces are grossly visible in the lung parenchyma.<sup>2,4</sup>



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A, Grade 1 asbestosis consists of fibrosis involving the bronchiolar wall and extending to the first layer of alveoli (arrows). B, Higher magnification shows asbestos bodies embedded within the fibrous tissue. C, Grade 2 asbestosis consists of fibrosis involving more distant alveolar walls but which spares at least some alveoli between adjacent bronchioles. D, Grade 3 asbestosis consists of fibrosis involving all alveoli between 2 adjacent bronchioles (hematoxylin-eosin, original magnifications  $\times 60$  [A and C],  $\times 300$  [B],  $\times 125$  [D]).<sup>2</sup>



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A Low-power view shows fibrotic lung with honeycomb changes. B, Mucous-filled honeycomb cysts (asterisk) are present in this case of grade 4 asbestosis. C, At this magnification, asbestos bodies are just visible (arrowheads). D, Higher magnification shows the upper asbestos body from C (hematoxylin-eosin, original magnifications  $\times 15$  [A], and  $\times 70$  [B]; Perliron, original magnifications  $\times 70$  [C] and  $\times 440$  [D]).<sup>2</sup>

#### How To Use Biopsy/Cytology Options Available for The Diagnosis of Asbestosis

Asbestosis is usually diagnosed histologically in lobectomy or pneumonectomy specimens re-sected because of cancer or at autopsy. If a clinical diagnosis cannot be made, lung biopsy is needed. Video-assisted thoracoscopic surgery is required as transbronchial biopsies rarely provide adequate tissue for histopathology. However, the finding of asbestos bodies or asbestos fibres in bronchoalveolar lavage samples may also assist in the diagnosis of asbestosis when quantitative methods are employed.

### Histologic Differential Diagnosis of Asbestosis

Distinction between idiopathic pulmonary fibrosis (IPF) and asbestosis can be difficult. The most common pattern of IPF is UIP. This is characterized by histological heterogeneity, represented by densely hyalinized areas of fibrosis, alternating with areas showing fibroblastic foci, and yet other areas consist of nearly healthy lung. Honeycomb changes are frequently found in UIP. While some cases of asbestosis resemble UIP, others resemble fibrotic NSIP. In general, the presence of asbestos bodies permits the distinction of asbestosis from other interstitial lung disorders. The presence of frequent fibroblast foci is against a diagnosis of asbestosis. Pleural plaques provide evidence of asbestos exposure (Table 2).<sup>2</sup>

**Table 2. Differential Diagnostic Features for Asbestosis and Idiopathic Interstitial Pneumonias**

Histologic Feature	Asbestosis	NSIP	UIP
Distribution	Peribronchiolar with subpleural accentuation	Diffuse	Subpleural accentuation. lower lung zone
Honeycomb changes	Uncommon except in advanced cases	Uncommon	Common
Fibrotic Foci	Rare	Inconspicuous	Conspicuous
Asbestos bodies	Frequent	Absent	Absent
Inflammation	Minimal	Variable	Minimal, typically localized to honeycomb foci
Pleural fibrosis	Common	Uncommon	Uncommon

Abbreviations: NSIP, nonspecific interstitial pneumonia; UIP, usual interstitial pneumonia.

### Recommendations for Evaluation of Asbestosis Employment History

A detailed employment history should be obtained (consistent recommendation, high quality evidence).<sup>3</sup>

- Year of first exposure, duration and end of exposure.
- Type of exposure: occupational, domestic or environmental.
- Intensity of exposure: exposure is significant when high concentration of asbestos is breathed in or when direct exposure to asbestos is for more than 6 months (8 hours a day and 40 hours a week) (consistent recommendation, very low quality evidence). The risk of exposure covers an area of 300 to 2200m, depending on wind direction.
- Type of asbestos at workplace.
- Smoking history.

## **Radiological Imaging**

### **Chest X-ray (CXR):**

CXR is the basic investigation for the diagnosis of asbestos related diseases (consistent recommendation, moderate quality evidence).<sup>3</sup>

The International Labour Office (ILO) classification of CXR is useful for systematically describing and recording radiographic abnormalities in the chest, seen not only in asbestosis but also in other Pneumoconiosis.<sup>3</sup>

### **High Resolution Computed tomography (HRCT):**

HRCT is useful, in the diagnostic work up for asbestos related disease, in the following circumstances:

- Borderline lung fibrosis on CXR.
- In case of discrepancy between pulmonary function tests showing restrictive pattern and CXR reported normal.
- Widespread pleural thickening obscuring lung fields.<sup>2,3</sup>

## **Radiological Characteristics of Asbestosis and Associated Pleural Fibrosis**

### **Asbestosis:**

- Irregular reticular opacities predominantly in the lung bases.
- Parenchymal bands.
- Round atelectasis.
- Honeycombing.
- Pleural plaques.
- Long dense or linear opacities in the diaphragmatic pleura, commonly bilateral and maybe calcified.

### **Diffuse pleural thickening:**

Pleural thickening extending to a quarter of the chest wall, commonly unilateral, involves posterior basal pleura. It may be calcified and associated with parenchymal bands and round atelectasis.<sup>2,3</sup>

## **Lung Function Tests**

Lung function testing, when used in the context of health surveillance, helps in the early diagnosis and follow-up of asbestos related diseases. It is indispensable for assessing a worker's capacity for work (consistent recommendation, high quality evidence).<sup>3</sup>

### **Spirometry:**

As an initial procedure, Forced Expiratory Volume in 1 second (FEV1), Forced Vital Capacity (FVC) and FEV1/FVC measurement can detect and quantify obstructive ventilatory defects and alert to a restrictive ventilatory defect.

### **Lung Volumes:**

Measurement of lung volumes is mandatory for diagnosing a restrictive ventilatory defect. A reduction in the Total Lung Capacity (TLC) can be due to asbestosis as well as diffuse visceral pleural thickening.

### **Diffusion Capacity (DLCO):**

DLCO is very sensitive in detecting early pulmonary fibrosis of asbestosis but has low specificity.

Pleural plaques alone are not responsible for any reduction in lung function.

### **Mineralogical Assessment for Asbestosis**

Asbestos bodies (AB) detection:

Asbestos can be detected in respiratory samples on optical microscopy as AB. Specific processing is required for detection of asbestos by AB counts in lung tissue or bronchoalveolar lavage (consistent recommendation, high quality evidence). Asbestos exposure is significant if AB counts is more than 1000AB per gram of dry lung tissue or 1AB per millilitre of bronchoalveolar lavage samples.<sup>2,3,4</sup>

### **Asbestos fibre detection:**

Asbestos fibre counts are done by electron microscopy. Asbestos fibre counts in excess of a million per gram of dry lung tissue indicates significant asbestos exposure. Fibre analysis is done in conjunction with the histopathological examination to identify the number and type of asbestos fibres.<sup>2,3,4</sup>

### **What Is The Criteria for Attributing Diseases to Asbestos**

Attributing diseases to asbestos exposure may have diagnostic, legal and financial implications. The intensity and duration of asbestos exposure must meet certain criteria to be labelled as a cause of the diseases.

Assessment of intensity and Duration of Asbestos Exposure

Asbestos fibers per milliliter per year (fibers/ml/y) is a unit used to measure a person's cumulative exposure to asbestos over their working life. This unit is used to estimate the risk of developing asbestosis or lung cancer.<sup>3,4,5</sup>

### **Definitions**

Fibers/ml: The average number of asbestos fibers in a millilitre of air inhaled by a person

Working year: Typically 8 hours per day, 5 days per week, 48 weeks per year

Fibers/ml/y: The cumulative exposure to asbestos over a working life

The Helsinki criteria states that a person's exposure to asbestos must be at least 25 fibers/ml years to be considered as the cause of asbestosis or lung cancer. This means that a person could be exposed to 25 fibers/ml for one year, or 5 fibers/ml for five years, to meet the criteria.<sup>3,4,5</sup>

### **An Overview of The Latest Recommendations for Managing Asbestosis**

The latest recommendations for managing asbestosis primarily focus on preventing further asbestos exposure, quitting smoking, getting vaccinated against common respiratory infections, and utilizing pulmonary rehabilitation to manage symptoms as currently no treatment can reverse the disease; if symptoms become severe, antifibrotic agents and oxygen therapy are considered. Further treatment options like lung transplant is for extreme cases.

### **Key points to remember:**

#### **Avoid further asbestos exposure:**

This is the most crucial step, as any additional exposure can worsen the condition.

**Smoking cessation:**

Smoking and asbestosis are independent and synergistic risk factors for lung cancer. Thus, smoking significantly increases the risk of lung cancer in individuals with asbestos exposure and so quitting smoking is essential.

**Inhaled bronchodilator therapy:**

Asbestosis with an obstructive component may be helped by inhaled bronchodilators.

**Vaccinations:**

Get vaccinated against influenza and pneumococcal pneumonia to reduce the risk of lung infections.

**Pulmonary rehabilitation:**

An exercise program designed to help manage breathing difficulties and maintain activity levels as well as improve lung function.

**Medical monitoring:**

Regular checkups with a healthcare professional to monitor symptoms by radiographic examination and lung function tests.

**Supplemental oxygen:**

Assess patient's oxygen status at rest and with exercise. If hypoxic, advise supplemental oxygen.

**Antifibrotic agents:**

Asbestosis resembles IPF histologically, so antifibrotic agents (for example, pirfenidone and nintedanib) used in other interstitial lung diseases may be effective in slowing down the decline in FVC in progressive asbestosis.

**Lung transplant:**

People with advanced diseases may be eligible for lung transplantation.

**Prognosis of Asbestosis**

Asbestosis typically progresses slowly over many years. Many people have mild symptoms and do well, whereas some develop progressive shortness of breath. A few develop respiratory failure and heart failure.

People with asbestosis have a significantly increased risk of lung cancer. Additionally, smoking increases lung cancer risk in people exposed to asbestos.<sup>1</sup>

**ASBESTOS-RELATED ISSUES IN PAKISTAN****1-Sources of asbestos:**

Among the many sources of asbestos exposure, currently the known sources of asbestos exposure in Pakistan are through:

Environmental exposures in mining and quarrying of asbestos in the north-western districts of the Khyber Pakhtunkhwa (KP) province.<sup>7</sup>

Ship breaking and recycling in Gadani, Balochistan province.

## **2-Prevention at workplace:**

Preventive measures at the workplace are almost non-existent. It is due to lack of awareness, about the risks at workplace and non-implementation of legislation regarding safety of workers, amongst the employers and the workers.

## **3. No asbestosis reported from the asbestos-region of the KP province.**

Despite high number of biopsy-proven mesothelioma cases due to environmental exposure to asbestos in the north-western districts of the KP province, there are hardly any cases of asbestosis. This could be either due to asbestosis being misdiagnosed (as IPF or fibrotic NSIP) or due to low intensity exposure, sufficient to cause pleural disease but not asbestosis (high intensity exposure is needed to cause asbestosis and lung cancer as compared to pleural diseases).

Specific request made to the laboratory to look for asbestos bodies in the biopsy specimens may help get the diagnosis of asbestosis.

## **References:**

1. Asbestosis. (n.d.). Cleveland Clinic.

<https://my.clevelandclinic.org/health/diseases/22245-asbestosis>

2. Roggli, V. L., Gibbs, A. R., Attanoos, R., Churg, A., Popper, H., Cagle, P., Corrin, B., Franks, T. J., Galateau-Salle, F., Galvin, J., Hasleton, P. S., Henderson, D. W., & Honma, K. (2010). Pathology of asbestosis: An update of the diagnostic criteria Report of the Asbestosis Committee of the College of American Pathologists and Pulmonary Pathology Society. *Archives of Pathology & Laboratory Medicine*, 134(3), 462-480. <https://doi.org/10.5858/134.3.462>

3. Roza, C. D., Cruz Carmona, M. J., Álvarez, R. F., Sancho, J. F., Martínez, B. M., & González, C. M. (2017). Recomendaciones sobre el diagnóstico y manejo de la enfermedad pleural y pulmonar por asbesto. *Archivos de Bronconeumología*, 53, 437-442. <https://share.google/5p9sKi6irLuxi0VHs>

4. Roggli, V., Gibbs, A. R., Attanoos, R., Churg, A., Popper, H., Corrin, B., Franks, T., Galateau-Salle, F., Galvin, J., Hasleton, P., & Honma, K. (2016). Pathology of asbestosis: An update of the diagnostic criteria-Response to a critique. *Archives of Pathology & Laboratory Medicine*, 140(9), 950-952. <https://doi.org/10.5858/arpa.2015-0503-SA>

5. Consensus report. (1997). Asbestos, asbestosis, and cancer: The Helsinki criteria for diagnosis and attribution. *Scandinavian Journal of Work, Environment & Health*, 23(4), 311-316.

6. Miedema, J. R., Moor, C. C., Veltkamp, M., Baart, S., Lie, N. S. L., Grutters, J. C., Wijsenbeek, M. S., & Mostard, R. L. M. (2022). Safety and tolerability of pirfenidone in asbestosis: A prospective multicenter study. *Respiratory Research*, 23(1), 139. <https://doi.org/10.1186/s12931-022-02062-2>

7. Khan, M. Y., Iqbal, Z., & Imran, M. (2018). Environmental asbestos and pleural malignant mesothelioma in the north-west of Pakistan: A serious health hazard with potentially fatal outcome. *MOJ Current Research & Reviews*, 1(3), 124-127. <https://medcraveonline.com/MOJCRR/environmental-asbestos-and-pleural-malignant-mesothelioma-in-the-north-west-of-pakistan-a-serious-health-hazard-with-potentially-fatal-outcome.html>

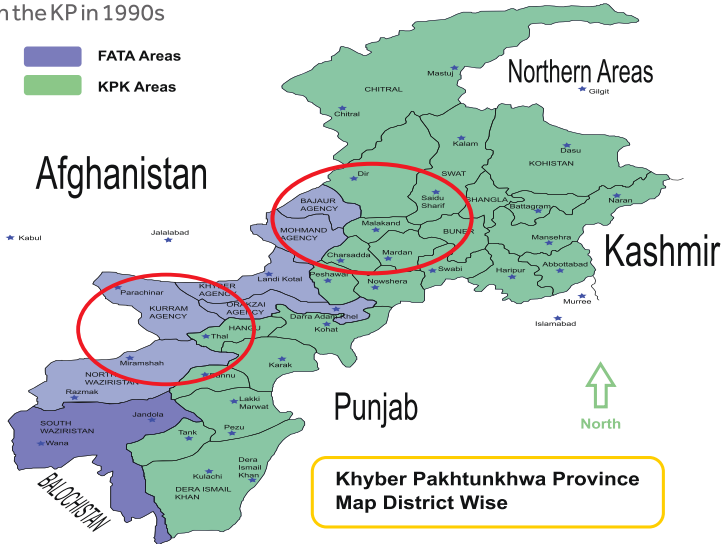
# Mesothelioma

Author: Dr. Zafar Iqbal

Mesothelioma is a cancer of the mesothelium, the thin lining of the body cavities that also covers the major internal organs of the body. The rarity of this tumour and its strong association with asbestos exposure make it a 'signal tumour,' that is, it is considered an epidemiological marker for exposure to asbestos. Owing to its initial nonspecific manifestations, mesothelioma is usually diagnosed at an advanced stage, and the tumour has limited therapeutic options. Its rarity and the variations in its geographic distribution are a major constraint in the field of research related to its therapeutics and prognostics.

## Epidemiology

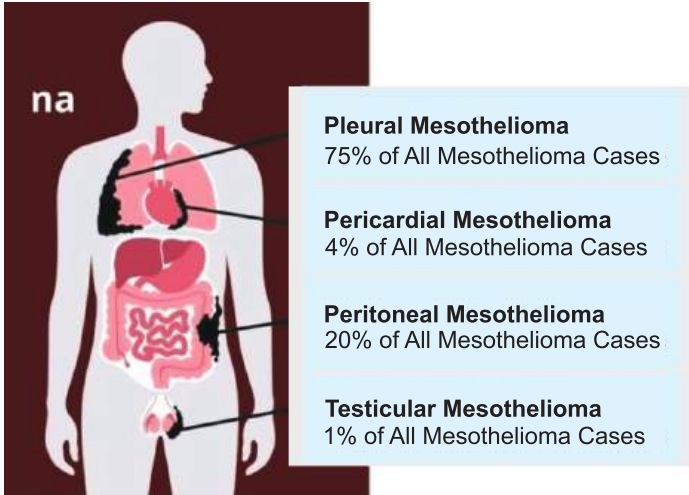
The predominant cause of malignant mesothelioma is inhalational exposure to asbestos, responsible for an estimated 80% to over 90% cases, with latency period of 30 to 40 years. Risk for developing mesothelioma increases with age with people over 60 having a ten times higher risk than those under 40. The incidence is more than 40 times higher for people who are occupationally exposed to asbestos than for the general population. 5-year survival rate for pleural mesothelioma is around 5% to 10%, highlighting its aggressive nature and challenges in treating this form of cancer. The male to female ratio is 2.7:1. Prevalence in Pakistan is high, particularly in the north western, Khyber Pakhtunkhwa (KP) province, initially reported in the KP in 1990s



Agencies: Mohmand, Bajaur, Kurram & North Waziristan;  
Districts: Charsaddah, Malakand, Dir, Nowshera, Mardan, Swabi

**Figure 1** Map showing areas of KP (formally known as North West Frontier) in Pakistan asbestos occurrence in red circle. (Source: physical maps of Pakistan Ezilon map).

## Types of Mesothelioma



Source : 2025 MesotheliomaVets.com

### Pathophysiology

Asbestos is a mineral found deep underground in rock and soil deposits. The mineral occurs as a bundle of spindly fibres that might be inhaled or ingested and then retained in the body tissues. After several years of retained fibres that cause irritation, scarring, and damage to the cell DNA, mesothelioma may begin to form. Both the amphibole and serpentine classes of the mineral are harmful to human health. The amphiboles can stay in the lungs longer, making exposure to amphiboles result in a higher chance of mesothelioma development than the serpentine class.

During the long latency period of malignant mesothelioma, a myriad of pathogenic events may occur that can contribute to the development of the disease. After asbestos fibers are inhaled deeply into the lung and penetrate pleural space, prolonged cycles of tissue damage, repair, and local inflammation are initiated following the interaction of asbestos fibers with mesothelial cells. That, in turn, leads to carcinogenesis.

Reactive oxygen species (ROS) induced by asbestos fibers with their exposed surface lead to DNA damage and stimulate a signal transduction cascade. Macrophages phagocytize asbestos fibers but are unable to digest them, thus resulting in the production of abundant ROS. These events activate MAP-kinase signaling pathways through the epithelial growth factor (EGF) receptor. As a result, several of the induced transcription factors are highly expressed in mesothelioma.

Asbestos fibers can absorb different proteins and chemicals to the broad surface of asbestos, with the accumulation of hazardous molecules, including carcinogens, as a consequence. Once inside, asbestos fibers bind to important cellular proteins. The subsequent deficiency of these proteins can also be detrimental for normal mesothelial cells.

Studies have also identified other comparable silicate minerals with similar health effects. These asbestiform minerals include winchite and richterite that can also cause mesothelioma.

**Figure 3:** Histological Types

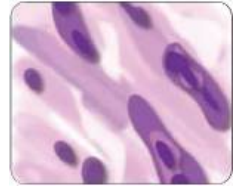
**Epithelioid Mesothelioma**

Clearly defined clump of cells  
Pink cytoplasm  
Easier to identify and remove during surgery  
Cell type with best prognosis



**Sarcomatoid Mesothelioma**

Overlapping, irregular shape  
Enlarged, elongated structure  
Metastasize and spread quickly Most challenging prognosis of the three types of mesothelioma cells



**Biphasic Mesothelioma**

Combination of epithelioid and sarcomatoid cell types  
Prognosis varies depending on cell prevalence  
Second most common cell type diagnosis  
Cells clump together in separate groups



Source: 2025 MesotheliomaGuide.com

**SYMPTOMS & SIGNS**

Developing mesothelioma or other asbestos-related disease can sometimes take over 20 years after the initial exposure. Symptoms can often be mistaken for other common and less-severe illnesses. Because of the long latency period, seniors, including veterans, are the most affected group.

**Pleural Mesothelioma**

If the tumors form in the pleural space (tissue lining of the lungs), symptoms can be:

- Chronic cough and hoarseness
- Difficulty breathing or shortness of breath
- Issues swallowing (the sensation of food getting stuck in the throat)
- Pain in the side of the chest or lower back

**Peritoneal Mesothelioma**

Cancerous cells that form in the tissue lining the abdomen may cause the following:

- Abdominal pain
- Constipation
- Fluid or swelling in the abdomen
- Nausea and vomiting

### Pericardial Mesothelioma

The rarest site of mesothelioma involves tumors growing in the tissue linings of the heart.

Symptoms can involve:

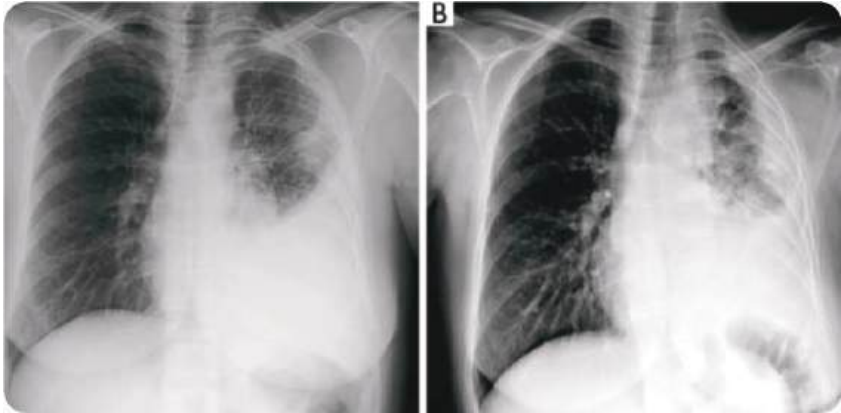
- Chest pain
- Irregular heartbeat
- Heart murmur
- Shortness of breath

### Diagnostic Work Up

Chest X-Ray (CXR)

Typical findings on CXR include Pleural effusion (Most likely unilateral exudative or haemorrhagic in nature, rich in mesothelial cells with frozen hemi thorax), Loss of volume, Nodular pleural thickening, Irregular fissure thickening, Or a localized mass lesion. The presence of pleural plaques may alert the clinician to prior asbestos exposure, even in the absence of known exposure. However, chest radiograph appearances are generally insensitive and nonspecific and further imaging is usually required

Figure 4: CXR showing Pleural Effusion and Thickening



Source :ISSN: 1755-5191 Diagnostic imaging and workup of malignant pleural mesothelioma

### CT-Chest

CT-Chest continues to be the mainstay imaging technique for the initial assessment of MPM and plays a primary role in structuring the subsequent diagnostic and staging evaluation as well as therapeutic decision-making process. Technical CT factors are very important for reaching the correct diagnosis. Pleural enhancement, Infiltration of chest wall, mediastinum or diaphragm. Nodular or mediastinal pleural thickening, Inter-lobar fissure nodularity.

**FIGURE 5. Malignant pleural mesothelioma.** Axial contrast-enhanced CT image demonstrates circumferential irregular pleural thickening (white arrow) and mediastinal lymphadenopathy

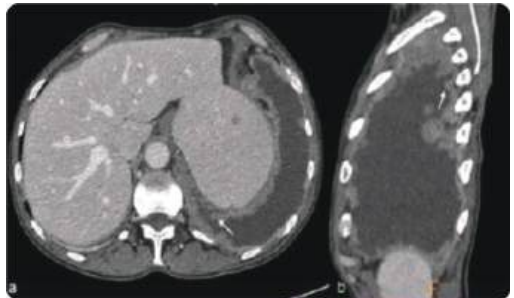


**FIGURE 6. Malignant pleural mesothelioma.** Axial contrast-enhanced CT image demonstrates right subtle circumferential pleural thickening (white arrow) and ipsilateral volume loss.

Source : ISSN: 1755-5191 Diagnostic imaging and workup of malignant pleural mesothelioma



**FIGURE 7. Malignant pleural mesothelioma.** Axial (a) and sagittal reformatted (b) contrast-enhanced CT images showing extensive nodular pleural thickening (white arrows) and a large-sized pleural effusion.



Source : ISSN: 1755-5191 Diagnostic imaging and workup of malignant pleural mesothelioma

## Common Immunohistochemical Stains Used To Differentiate Malignant Mesothelioma From Metastatic Adenocarcinoma

Malignant Mesothelioma	Adenocarcinoma
CK5/6 Calretinin D2-40 WT-1	TTF-1 Napsin A Ber-EP4 B72.3 CEA Leu-M1 MOC-31 Claudin-4

To establish the diagnosis at least 2 positive mesothelioma and 2 negative carcinoma markers must be present

**Molecular Markers;** Fluorescence In Situ Hybridization (FISH) testing is highly specific tool to detect p16 deletion used for differentiation between benign versus malignant mesothelial proliferations.

### Role of Thoracoscopy & Image-Guide Biopsy

Pleural fluid cytology and non-image-guided Abrams or Cope biopsies have sensitivities of approximately 30% for detecting malignant mesothelioma, and thoracoscopic biopsy has a sensitivity of approximately 90%. The difference between these two probably relates to obtaining adequate tissue. The use of immunohistochemical stains allows a firm diagnosis to be made from relatively small samples.

### Staging

#### Primary tumor

**T0** No evidence of primary tumor

**T1** Tumor limited to the ipsilateral parietal pleura with or without mediastinal pleura and with or without diaphragmatic pleural involvement

**T1a** No involvement of the visceral pleura

**T1b** Tumor also involving the visceral pleura

**T2** Tumor involving each of the ipsilateral pleural surfaces (parietal, mediastinal, diaphragmatic, and visceral pleura) with at least one of the following:

- Involvement of the diaphragmatic muscle
  - Extension of tumor from the visceral pleura into the underlying pulmonary parenchyma
- T3** Locally advanced but potentially resectable tumor; tumor involving all of the ipsilateral pleural surfaces (parietal, mediastinal, diaphragmatic, and visceral pleura) with at least one of the following:
- Involvement of the end thoracic fascia
  - Extension into the mediastinal fat
  - Solitary, completely resectable focus of tumor extending into the soft tissue of the chest wall
  - Non-transmural involvement of the pericardium

**T4** Locally advanced, technically unresectable tumor; tumor involving all of the ipsilateral pleural surfaces (parietal, mediastinal, diaphragmatic, and visceral pleura) with at least one of the following:

- Diffuse extension or multifocal masses of tumor in the chest wall, with or without associated rib destruction

- Direct diaphragmatic extension of the tumor to the peritoneum
- Direct extension of the tumor to the contralateral pleura
- Direct extension of the tumor to a mediastinal organ
- Direct extension of the tumor into the spine
- Tumor extending through to the internal surface of the pericardium with or without a pericardial effusion or tumor involving the myocardium

**Regional lymph nodes**

**NX** Regional lymph nodes cannot be assessed

**N0** No regional lymph node metastases

**N1** Metastases in the ipsilateral bronchopulmonary or hilar lymph node

**N2** Metastases in the subcarinal or in the ipsilateral mediastinal lymph node, including the ipsilateral internal mammary and peridiaphragmatic nodes

**N3** Metastases in the contralateral mediastinal, contralateral internal mammary, ipsilateral, contralateral supraclavicular lymph nodes

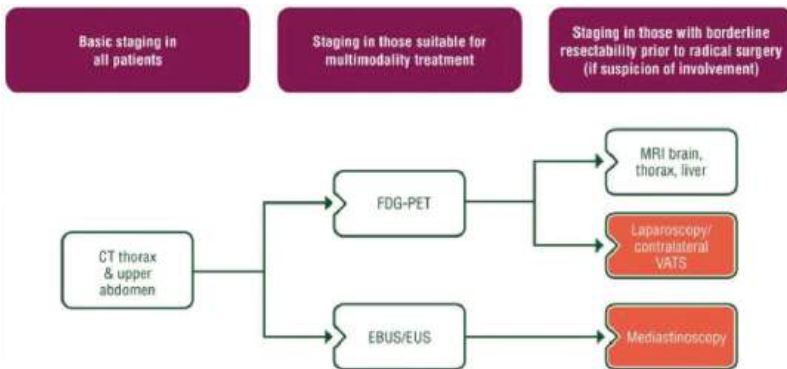
**Distant metastases**

**M0** No distant metastases

**M1** distant metastases

Stage grouping			
Stage	T	N	M
IA	T1a	N0	M0
IB	T1b	N0	M0
II	T2	N0	M0
III	T1,T2	N1	M0
	T1,T2	N2	M0
	T3	N0,N1,N2	M0
IV	T4	Any N	M0
	Any T	N3	M0
	Any T	Any N	M1

**Staging**



## Treatment Options For Mesothelioma

- **Pleurodesis** uses chemicals or drugs to produce a scar in the space between the layers of the pleura. Fluid is first drained from the space using a catheter or chest tube and the chemical or drug is put into the space. The scarring stops the build-up of fluid in the pleural cavity.
- **Pleurectomy And Decortication** is done to remove part of the covering of the lungs and lining of the chest wall and part of the outer surface of the lungs.
- **Extrapleural Pneumonectomy** is surgery to remove one whole lung and part of the lining of the chest wall, the diaphragm, and the pericardium.
- **Chemo-Radio Therapy** is administered as adjuvant therapy to some patients after surgery.

**Immunotherapy drugs** used to treat advanced mesothelioma include:

1. ipilimumab
2. nivolumab

Other immunotherapy drugs, such as durvalumab, are being studied in malignant mesothelioma.

**Targeted therapy** uses drugs or other substances to identify and attack specific cancer cells. Bevacizumab is a targeted therapy drug used to treat malignant mesothelioma. Other targeted therapies, such as ramucirumab, are being studied in malignant mesothelioma.

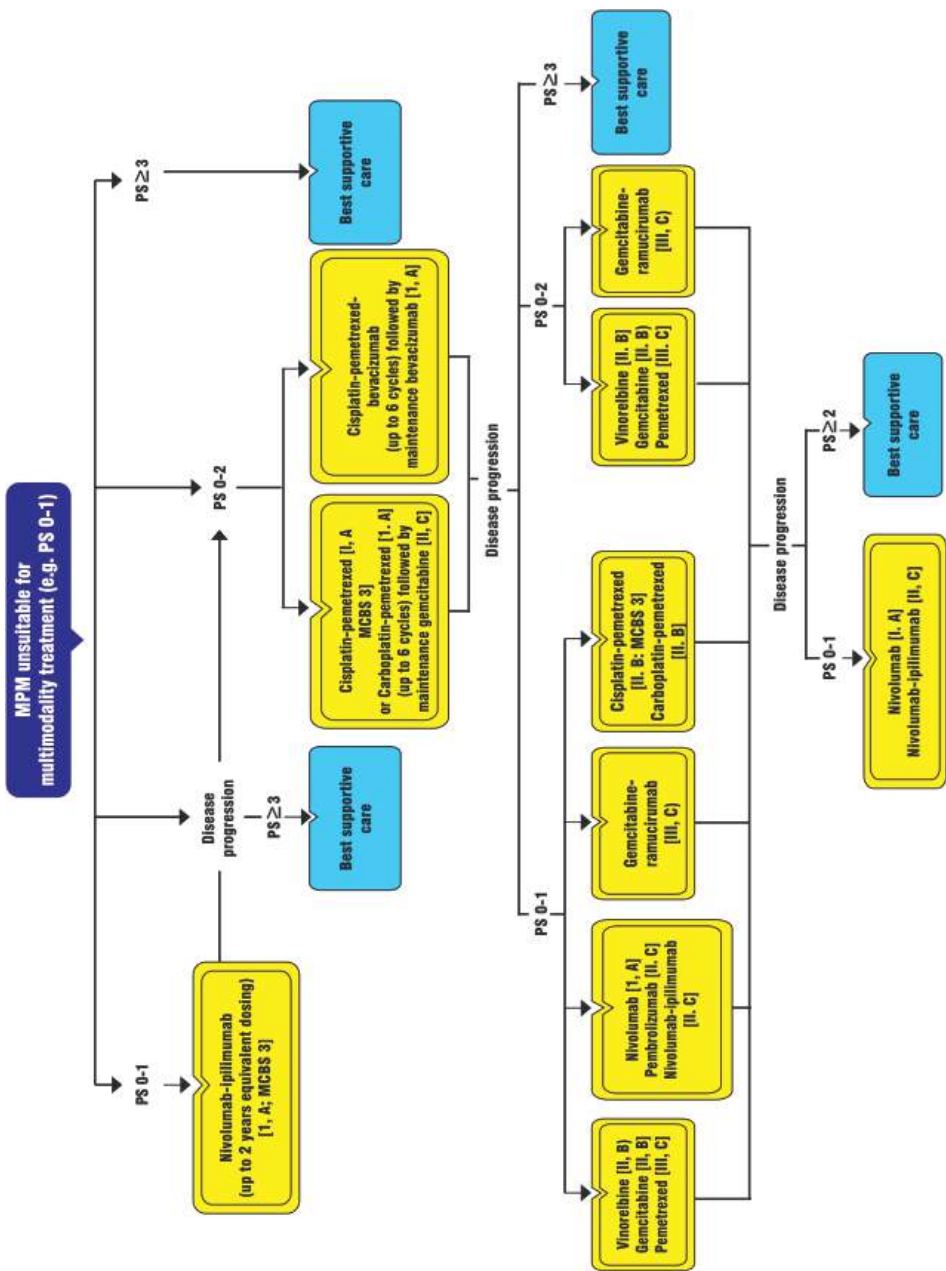
## Treatment

MPM staged and determined suitable for multimodality treatment

Consideration of VATS pleurodesis

Macroscopic complete resection<sub>a</sub>  
with/without induction  
Platinum-pemetrexed ChT<sub>b</sub>

Completion of up to 6 cycles of ChT  
(Platinum-pemetrexed)



Source; Malignant pleural mesothelioma: ESMO Clinical Practice Guidelines, 2022

**References:**

- 1.Environmental asbestos and pleural malignant mesothelioma in the north-west of Pakistan; a serious health hazard with potentially fatal outcome ;Mohammad Yousaf Khan, Zafar Iqbal, Muhammad Imran, *MOJ Curr Res & Rev.* 2018;1(3):124-127. DOI: 10.15406/mojcrr.2018.01.00019
- 2.Pleural mesothelioma Brierley, J. · Gospodarowicz, M.K. · Wittekind, C. *TNM Classification of Malignant Tumours* John Wiley & Sons, Inc, Chichester, West Sussex/Hoboken, New Jersey, 2017; 113-114 Google Scholar
- 3.First-line nivolumab plus ipilimumab in unresectable malignant pleural mesothelioma (CheckMate 743): a multicentre, randomised, open-label, phase 3 trial *Lancet.* 2021; 397:375-386
- 4.Radiological data in malignant pleural. Mesothelioma: a pilot study. Zafar Iqbal, Mohammad Yousaf Khan, Jamshed Alam, Zia Ullah

# Coal Worker's Pneumoconiosis (CWP)

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Authors: Dr. Saadia Ashraf & Dr. Shahid Salam

Coal Worker Pneumoconiosis (CWP), also known as black lung disease, is a progressive and preventable occupational lung disease caused by prolonged exposure to coal dust. It's one of the most common conditions affecting coal workers across the world and more specifically in Pakistan, particularly those involved in underground mining or those working with high concentrations of coal dust (brick factories). CWP is a significant global health concern, with high rates reported in countries where coal mining is prevalent, such as Pakistan, China, and India.

This guideline will provide an in-depth understanding of CWP, focusing on its epidemiology, causes, signs and symptoms, diagnosis, and treatment options. Understanding of these aspects is very important for healthcare professionals involved in the care of workers exposed to coal dust and for those in occupational safety management roles to reduce exposure.

## Epidemiology

CWP is one of the oldest known occupational diseases, with documented cases dating back to the 18th century. However, its prevalence remains high in coal mining regions, particularly among older miners who have experienced long-term exposure to dust.

In Pakistan, where coal mining is a significant industry, CWP has become a great public health concern. The epidemiology of CWP in Pakistan is affected by several factors, including the widespread use of manual mining techniques, poor working conditions, and inadequate protective measures for miners.

1. **Prevalence:** The global prevalence of CWP varies based on geographic region, industry practices, and regulatory oversight. A 2019 study by the CDC found that approximately 1 in 5 coal miners with over 25 years of exposure to coal dust showed signs of CWP.<sup>1</sup> In Pakistan, a study conducted in 2017 showed that up to 30% of coal miners in some regions of Pakistan could develop CWP due to prolonged exposure to coal dust without sufficient protective measures. The prevalence of CWP in Pakistan is high, especially in Balochistan, Khyber Pakhtunkhwa, and parts of Sindh.
2. **At-Risk Population:** Coal miners, particularly those engaged in underground mining, are at the highest risk for developing CWP. Miners often work in hazardous conditions, with exposure to coal dust levels far exceeding occupational safety limits. Workers involved in manual or small-scale mining, where safety measures are typically not enforced, are more susceptible to respiratory diseases.
3. **Age and Duration of Exposure:** The development of CWP is strongly associated with the duration of exposure to coal dust. The risk of disease increases significantly in

miners who have worked for 15 years or more. The disease typically manifests in miners who are over the age of 50, although early onset can occur in individuals with severe and prolonged exposure to high levels of dust.<sup>1,7</sup>

4. **Gender and Occupation:** CWP predominantly affects male miners, as the coal mining workforce has historically been male-dominated. However, as gender roles in mining have evolved, there has been a gradual increase in the number of female workers exposed to coal dust especially in the bricks factories where coal is used as the main source of fuel, potentially altering the demographic trends in the future.<sup>5</sup>
5. **Geographic Variation:** The disease remains highly prevalent in areas with ongoing coal mining operations. In the Pakistan, miners in regions such as Khyber Pakhtunkhwa and Balouchistan experience some of the highest rates of CWP. Similarly, countries like China and India, where coal mining is a major industry, report a high burden of the disease.<sup>9</sup>

### Causes

CWP is caused by the inhalation of coal dust, consisting of small particles that can penetrate into the lungs. With the passage of time, they accumulate and lead to inflammation, fibrosis, and scarring of lung tissue.

**1. Coal Dust:** The primary cause of CWP is prolonged exposure to coal dust, which contains a mixture of carbon, silica, and other minerals. The size of coal dust particles is critical in determining their harmful effects. Only particles that are small enough (respirable dust) can reach the alveoli, where they cause the greatest damage. Continuous exposure to high concentrations of dust is necessary for the development of CWP.<sup>1</sup>

**2. Silica Dust:** Coal miners are often also exposed to silica dust, especially in mines where both coal and silica-rich rocks are extracted. Silica is a known carcinogen and contributes significantly to the severity of CWP. Studies have shown that miners exposed to both coal dust and silica are more likely to develop more severe forms of the disease, such as PMF.<sup>8,9</sup>

**3. Duration and Intensity of Exposure:** The risk of developing CWP increases with the duration of exposure. Miners who have worked in high-dust environments for 10 to 20 years are at higher risk, with the disease becoming more likely after 25 years of exposure. However, high-intensity exposure over shorter periods can also lead to the development of the disease, particularly when effective dust control measures are lacking.<sup>7</sup>

**4. Environmental and Occupational Factors:** The amount of dust generated depends on several factors, including the type of mining operation, the mining method, and the level of ventilation. Underground mining, especially in older mines with poor ventilation, poses the highest risk for coal dust exposure. Inadequate dust control measures and poor use of personal protective equipment further contribute to the risk of developing CWP.<sup>5,9</sup>

**5. Genetic Factors:** Although environmental factors primarily cause CWP, genetic susceptibility may influence an individual's risk. Some workers may have a heightened inflammatory response to coal dust, making them more likely to develop CWP even after

relatively short periods of exposure.<sup>6</sup>

### **Pathogenesis**

The pathogenesis of CWP involves a complex interplay of environmental factors (such as coal dust) and the body's immune and inflammatory responses.

#### **1. Inhalation of Coal Dust:**

**Primary Exposure:** CWP begins when a person inhales coal dust particles, which are composed primarily of fine particles of carbon and silica, along with other minerals found in coal. These particles can be inhaled deeply into the lungs, particularly during mining operations.

**Particle Size:** The dust particles responsible for CWP are usually small enough to penetrate the respiratory system and reach the alveoli. Particles with a diameter of less than 10 micrometers (PM10) are particularly dangerous because they can bypass the natural defences of the upper respiratory tract and lodge in the lower respiratory system.

**2. Lung's Initial Response to Dust:** Once coal dust particles are inhaled, they are deposited in the alveoli. The body's immune response begins with the activation of macrophages. **Coal Dust Uptake:** Macrophages phagocytise the coal dust, but due to the inert nature of coal particles, these cells cannot completely eliminate them. The dust particles accumulate in the macrophages, leading to the release of inflammatory mediators.

#### **3. Inflammatory Response:**

**Cytokine Release:** The accumulation of coal dust in macrophages triggers the release of pro-inflammatory cytokines, such as tumor necrosis factor (TNF), interleukins (e.g., IL-1, IL-6), and other chemokines. These cytokines recruit additional immune cells (neutrophils and lymphocytes) to the site of deposition.

**Oxidative Stress:** Coal dust particles can generate free radicals and reactive oxygen species (ROS), which cause oxidative stress, further damaging the lung tissue and enhancing the inflammatory response.

**Fibrosis Development:** Chronic inflammation and tissue injury activate fibroblasts (cells responsible for collagen production), leading to the deposition of extracellular matrix proteins like collagen. This causes fibrosis (scarring) in the lungs, particularly around the dust particles, which is a hallmark of CWP.

#### **4. Formation of Coal Macules and Nodules:**

**Coal Macules:** In the early stages of CWP, small areas of blackened tissue called coal macules are formed. These are characterized by a collection of coal dust and inflammatory cells (mainly macrophages). Coal macules do not initially impair lung function.

**Progression to Coal Nodules:** Over time, continued exposure and the inflammatory response cause the macules to evolve into larger lesions known as coal nodules. These nodules are composed of fibrous tissue and macrophages and are surrounded by a fibrotic reaction. They may eventually lead to progressive massive fibrosis (PMF) in severe cases.

#### **5. Progressive Massive Fibrosis (PMF):**

**Fibrotic Lung Damage:** In the advanced stages of CWP, the accumulation of fibrous tissue in the lungs leads to the formation of large fibrotic masses or conglomerates. This condition is known as progressive massive fibrosis (PMF).

**Impaired Gas Exchange:** As fibrosis progresses, the lung architecture is severely altered, and the surface area for gas exchange is reduced. This results in impaired oxygen uptake, leading to respiratory symptoms such as shortness of breath, cough, and reduced exercise tolerance.

#### **6. Immunologic and Genetic Factors:**

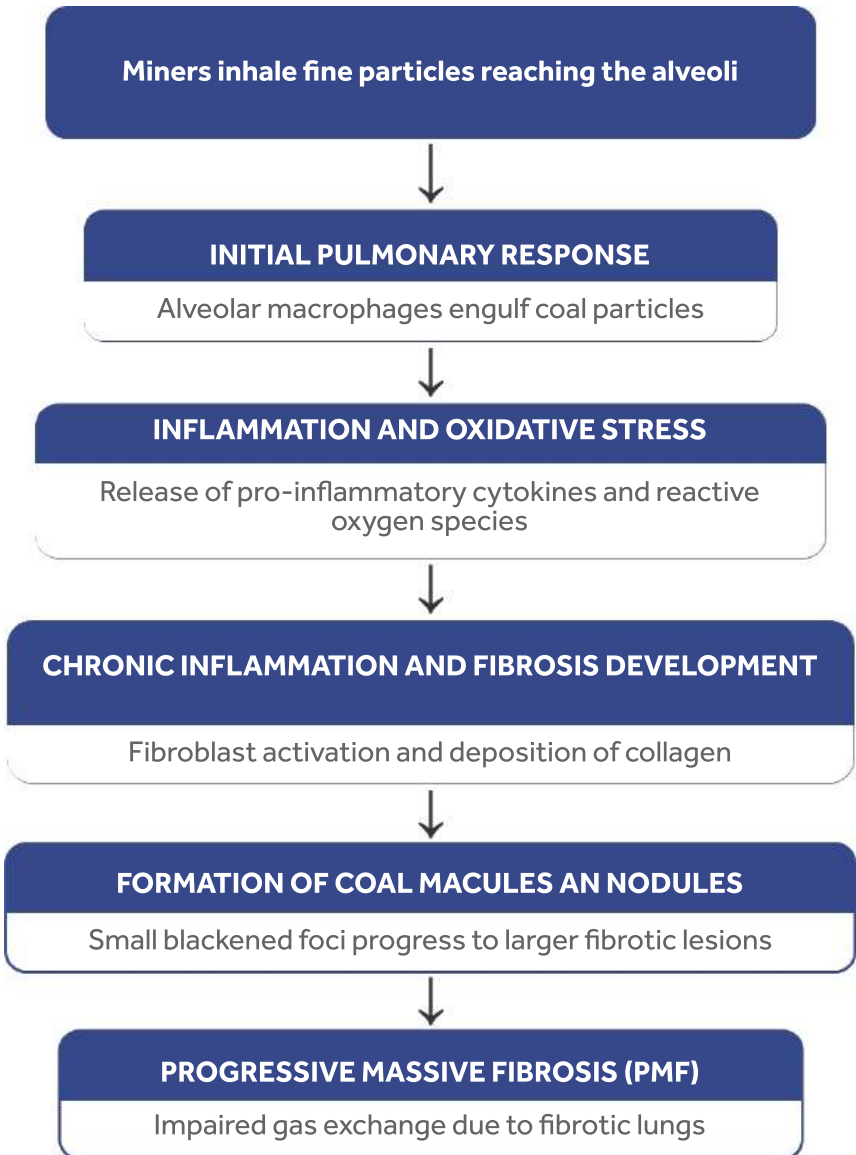
**Immune System Dysfunction:** The immune response to coal dust is variable between individuals. In some, a chronic inflammatory response is mounted, leading to tissue damage and fibrosis, while others may not develop the disease despite similar exposure levels.

**Genetic Predisposition:** Some genetic factors may influence susceptibility to CWP. Variations in genes that regulate inflammation, immune responses, and lung repair mechanisms may predispose certain individuals to develop more severe forms of the disease.

#### **7. Additional Factors Contributing to Pathogenesis:**

**Silica Dust Exposure:** Many coal mines also contain silica dust, which exacerbates the disease. Silica is a more potent inducer of fibrosis compared to coal dust alone. Silicosis, a similar pneumoconiosis caused by silica exposure, often coexists with CWP in miners, leading to a more aggressive disease course.

**Co-occurrence of Other Respiratory Conditions:** Miners are at an increased risk of developing other chronic lung conditions, such as chronic obstructive pulmonary disease (COPD), bronchitis, and emphysema, which can further worsen the respiratory symptoms in CWP.



## Signs and Symptoms

The signs and symptoms of CWP can develop gradually over time and are often mild at first. As the disease progresses, it can lead to significant impairment of lung function and overall health.

### 1. Early Symptoms:

Chronic cough is one of the earliest signs of CWP is a persistent cough, which may initially be dry but can become productive with time as the disease progresses. Dyspnea initially occurs during physical exertion and gradually worsens, even during rest, as lung function deteriorates. Fatigue and reduced stamina are common in the early stages and are often attributed to the gradual decrease in oxygenation due to impaired lung function. Wheezing may be present as a result of airway narrowing caused by fibrosis and inflammation.

### 2. Progressive Symptoms:

As the disease advances, patients experience more severe shortness of breath, even at rest. This can significantly impair their ability to perform daily activities and work. Pain or tightness in the chest may occur due to inflammation and scarring of lung tissue. Cyanosis may occur in advanced stages of the disease. Clubbing is a late sign.

### 3. Progressive Massive Fibrosis (PMF):

In severe cases, the formation of large fibrotic masses leads to PMF. This is characterized by extensive scarring and lung destruction, leading to respiratory failure, severe hypoxia, and the need for oxygen therapy. PMF is associated with a significant increase in morbidity and mortality among coal miners.

## Diagnosis

The diagnosis of Coal Worker Pneumoconiosis is based on a combination of occupational history, clinical examination, imaging studies, and pulmonary function tests.

### 1. Occupational History:

A comprehensive occupational history is essential for diagnosing CWP. Healthcare providers should inquire about the duration, type, and intensity of coal dust exposure, including whether the individual worked in underground or surface mining operations, and if they were involved in high-risk tasks such as drilling or cutting coal.<sup>1,6</sup>

### 2. Clinical Examination:

During physical examination, the clinician may detect signs of chronic lung disease, such as crackles on auscultation, wheezing, or reduced breath sounds. In later stages, signs of respiratory failure, including cyanosis and use of accessory muscles of respiration, may be evident.<sup>6</sup>

### 3. Chest X-ray:

Chest radiographs remain the most widely used diagnostic tool for CWP. Radiographic findings include small opacities (ranging from 1-10 millimeters) and nodules that represent the accumulation of coal dust in the lungs. As the disease progresses, larger opacities and the formation of PMF may become visible.<sup>5</sup> The ILO classification system is commonly used to assess the severity of findings on chest X-rays.<sup>9</sup>

### 4. CT Scans:

High-resolution computed tomography (CT) scans are more sensitive than traditional X-rays and can detect early signs of CWP, including small nodules, emphysema, and fibrosis that may not be visible on standard X-rays. CT scans are particularly useful for evaluating PMF and other complications of CWP.

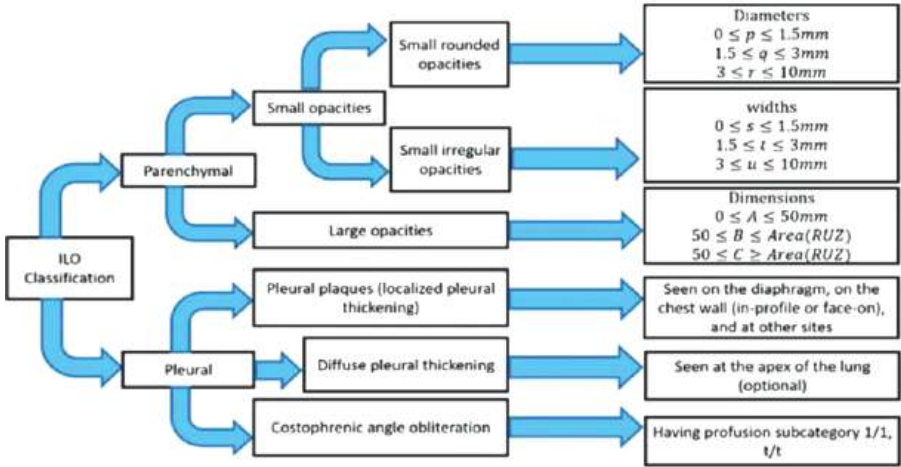
### 5. Pulmonary Function Tests (PFTs):

These tests assess the functionality of the lungs and can detect the presence of obstructive or restrictive patterns. In CWP, restrictive patterns with decreased lung volumes, such as total lung capacity and forced vital capacity, are commonly observed.

**6. Sputum Analysis:**

Although not routinely used in diagnosis, sputum analysis can be performed to detect the presence of coal dust particles or inflammatory markers.

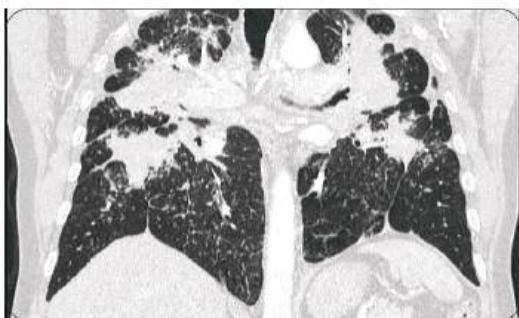
**ILO Classification of CWP**



Miliary Shadow in a patient with CWP



CT scan of a patient with CWP (Miliary pattern)



PMF on CT Chest in patient with CWP



PMF in patient with CWP

### Treatment

There is no cure for Coal Worker Pneumoconiosis, and treatment is primarily focused on managing symptoms, slowing disease progression, and improving the quality of life.

**1. Prevention:** The most effective approach to CWP is prevention. This includes reducing dust exposure in coal mines through the use of dust control measures, ventilation systems, and personal protective equipment, such as respirators. Regular monitoring of dust levels and health screening for miners are essential.<sup>1,7</sup>

### 2. Medical Management:

- **Bronchodilators:** They may have some improvement in the lung functions and symptoms of patients with CWP as most patients are smokers as well and have concurrent COPD.
- **Corticosteroids:** In cases with significant inflammation, corticosteroids may help reduce lung inflammation and improve respiratory function. The dose of steroid is 0.5-1mg/dl.
- **LTOT:** For patients with severe hypoxia, supplemental oxygen is necessary to maintain adequate oxygen levels and prevent complications such as cor-pulmonale.

**3. Pulmonary Rehabilitation:** Pulmonary rehabilitation is an essential component of care for CWP patients. It includes exercise training, nutritional support, and educational programs to help improve overall lung function, decrease symptoms, and increase physical capacity. It is

particularly beneficial for those with advanced disease,

#### **4. Management of Complications:**

- Infections: Respiratory infections are common in CWP patients due to compromised lung defences. Antibiotics or antiviral treatments may be prescribed if an infection is suspected.<sup>6</sup>
- Progressive Massive Fibrosis (PMF): PMF requires a more intensive management approach, including oxygen therapy, pulmonary rehabilitation, and, in extreme cases, lung transplantation if lung function deteriorates to the point of respiratory failure.<sup>10</sup>

#### **References**

1. Centers for Disease Control and Prevention (CDC). (2020). Coal Workers 'Pneumoconiosis (Black Lung Disease). National Institute for Occupational Safety and Health (NIOSH). Retrieved from <https://www.cdc.gov/niosh/topics/cwp>
2. International Labour Organization (ILO). (2000). ILO Classification of Radiographs of Pneumoconioses. 3rd edition. World Health Organization.
3. Swan, P. E., & Ainsworth, A. (2007). Coal Worker's Pneumoconiosis: A Global Perspective. *International Journal of Environmental Research and Public Health*, 4(2), 125-134. <https://doi.org/10.3390/ijerph4020125>
4. Baker, W. R., & Morrow, W. J. (2004). Coal Workers 'Pneumoconiosis: The Continuing Challenge. *Occupational Medicine*, 54(3), 181-186. <https://doi.org/10.1093/occmed/kqh042>
5. Hedlund, J., & McGowan, S. (2006). The Role of Chest X-ray in the Diagnosis of Coal Workers 'Pneumoconiosis: A Review. *Chest*, 130(5), 1571-1578. <https://doi.org/10.1378/chest.130.5.1571>
6. American Lung Association. (2015). Coal Workers 'Pneumoconiosis (Black Lung Disease). Retrieved from <https://www.lung.org/lung-health-diseases/lung-disease-lookup/coal-workers-pneumoconiosis>
7. Ruffin, R. E., & Powers, M. L. (2001). Coal Workers 'Pneumoconiosis: Epidemiology, Clinical Course, and Management. *Current Opinion in Pulmonary Medicine*, 7(2), 108-113. <https://doi.org/10.1097/00063198-200103000-00002>
8. National Institute for Occupational Safety and Health (NIOSH). (2019). Progressive Massive Fibrosis (PMF) in Coal Miners. Retrieved from <https://www.cdc.gov/niosh/topics/cwp/pmf.html>
9. George, P. M., & Page, C. P. (2016). Management of Coal Workers 'Pneumoconiosis: Current and Future Treatment Options. *Respiratory Medicine*, 115, 16-23. <https://doi.org/10.1016/j.rmed.2016.04.003>
10. Finkelstein, M. M. (2003). Dust Exposure and Risk of Pneumoconiosis in Coal Miners: A Review of Epidemiological Studies. *European Respiratory Journal*, 21(6), 1101-1109. <https://doi.org/10.1183/09031936.03.00055402>

# Silicosis

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Author: Dr. Muhammad Azam Mushtaq

Silicosis has been recognized for centuries as one of the most important occupational lung diseases. It remains a global health issue despite being entirely preventable. With increasing industrialization, especially in mining, stone crushing, ceramics, construction, and glass production, silicosis continues to cause significant morbidity and mortality. These guidelines are tailored for use in low- and middle-income countries (LMICs) with a high burden, while incorporating best practices from developed countries.

## Epidemiology & Burden

Globally, millions of workers remain at risk of silica exposure. Prevalence varies depending on industrial practices and dust control measures. In Pakistan, silicosis is particularly prevalent in Punjab's stone-crushing and brick kiln industries, Sindh's industrial zones, KP's mining regions, and Balochistan's coal and phosphate mines. Documented deaths exceed hundreds annually, though underreporting is likely. Internationally, silicosis continues to burden India, China, and parts of Africa where occupational health standards remain weak.

## Etiology & Risk Factors

Silicosis results from inhalation of respirable crystalline silica particles (quartz, cristobalite, tridymite). Occupational risk factors include:

- Mining (stone, sand, coal, granite)
- Construction (cutting, drilling, grinding concrete, brick, marble)
- Manufacturing (ceramics, pottery, glass)
- Sandblasting

Host risk factors: duration of exposure, poor ventilation, lack of PPE, concurrent TB, and smoking.

## Pathophysiology

When inhaled, silica particles deposit in alveoli where they are engulfed by macrophages.

This triggers release of cytokines and growth factors, leading to inflammation, fibroblast activation, and fibrosis. Progression occurs from nodular lesions to coalescent nodules and massive fibrosis. Acute silicosis results from overwhelming exposure causing alveolar proteinosis.

## Histological Classification

Histological classifications of silicosis are primarily based on the severity and pattern of fibrosis in the lung tissue. The disease is typically divided into three main types acute, accelerated, and chronic with the chronic form further subdivided into simple and complicated.

### **1. Acute silicosis (silicoproteinosis)**

This is the most aggressive but rarest form, resulting from a high-intensity, short-term exposure to fine silica dust.

**Microscopic features:** The histological findings resemble pulmonary alveolar proteinosis. The alveolar spaces are filled with an eosinophilic, finely granular, lipoproteinaceous material mixed with foamy macrophages.

**Silicotic nodules:** may be poorly formed or even absent in this type. Outcome: It has a rapid onset and high mortality rate.

### **2. Chronic silicosis**

The most common type, developing after decades of low-to-moderate exposure. It presents in two forms:

#### **(a) Simple silicosis Microscopic features:**

Characterized by the presence of mature silicotic nodules in the lung parenchyma.

#### **Silicotic nodule:**

A well-defined, rounded nodule with a classic "whorled" pattern of hyalinized collagen fibers.

#### **Peripheral inflammation:**

The nodule is surrounded by a cellular periphery of dust-laden macrophages and lymphocytes.

#### **Silica particles:**

Birefringent silica crystals can be seen within the nodule when viewed under polarized light microscopy.

#### **Gross features:**

Discrete, palpable, hard nodules, typically distributed in the upper lobes.

#### **Lymph nodes:**

Hilar and mediastinal lymph nodes frequently contain silicotic nodules and may exhibit characteristic "eggshell" calcification.

#### **(b) Complicated silicosis (Progressive massive fibrosis, PMF)**

#### **Microscopic features:**

This is the result of the coalescence of individual silicotic nodules to form large, conglomerate masses of dense scar tissue.

#### **Central necrosis:**

The fibrous masses may show central necrosis, potentially leading to cavitation, often from superimposed infection like tuberculosis.

#### **Distortion:**

Significant architectural distortion of the lung parenchyma is present.

#### **Gross features:**

Large, firm, blackened fibrous masses measuring more than 1 cm in diameter, most commonly in the upper lobes.

#### **Appearance:**

They migrate toward the hilum, causing compensatory emphysema in the surrounding lung tissue.

### 3. Accelerated silicosis

#### Onset:

A more rapidly progressive form than chronic silicosis, developing within 5 to 10 years of exposure to high levels of silica.

#### Microscopic features:

The histological appearance is similar to chronic silicosis but with faster development of fibrosis.

#### Progression:

It often progresses more rapidly to PMF



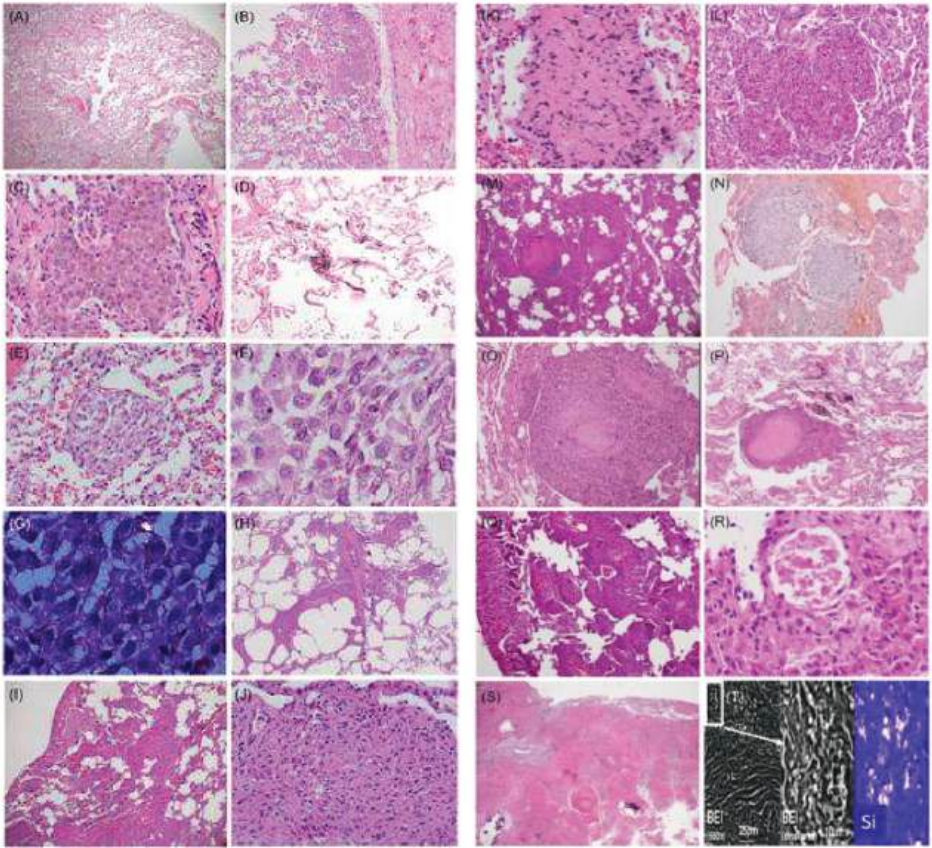
Left: gross scarring of predominantly upper lobe

Right: Coalescent Fused Silicotic Nodule have a Whorled and Hyalinized Appearance.

Crawford JA, Sanyal S, Burnett BR, Wiesenfeld SL, Abraham JL.

Accelerated silicosis in sandblasters: pathology, mineralogy, and clinical correlates. *Am J Ind Med.*

2024; 67: 179-199. doi:10.1002/ajim.23561



Left) (A) nearly normal lung (B) subpleural and focal airspace filling with pigmented macrophages (C) detail of airspace macrophages, showing fine dust content (D) focal dust macular lesion with mild centrilobular emphysema (E) focal interstitial cellular granuloma with normal surrounding lung. (F and G) Detail of macrophages with fine opaque (F) and birefringent particles noted with PLM (G). (H) Patchy interstitial infiltration by dusty macrophages (I) more diffuse interstitial infiltration by dusty macrophages (J) detail of interstitial dusty macrophages with very fine particles.

Crawford JA, Sanyal S, Burnett BR, Wiesenfeld SL, Abraham JL. Accelerated silicosis in sandblasters: pathology, mineralogy, and clinical correlates. *Am J Ind Med.* 2024; 67: 179-199. doi:10.1002/ajim.23561

(Right) (K) interstitial fibrotic dusty nodule. (L) Interstitial aggregates of macrophages with mild fibrosis, not yet recognizable as developing silicotic nodule. (M) Developing silicotic nodules in diffuse interstitial cellularity. (N) Two developing cellular silicotic nodules with mild fibrosis. (O) Small developing silicotic nodule with cellular periphery and small nodular center. (P) More developed silicotic nodule and adjacent foci of dusty macular lesions and perivascular dust. (Q) Diffuse interstitial macrophages with small focus of PAP (arrow). (R) Focal area of PAP in cellular interstitial lesion. (S) PMF with confluent silicotic nodules and focal calcification. (T) Left: SEM image of silicotic nodule, higher magnification of boxed area (arrow) is shown in center frame, showing brighter inorganic dust particles, Right: silicon X-ray distribution map reveals location of silica particles.

## Clinical Presentation

Symptoms develop insidiously and vary with disease type:

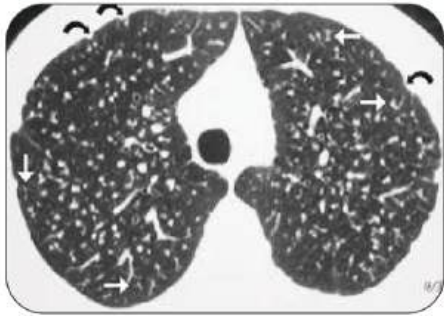
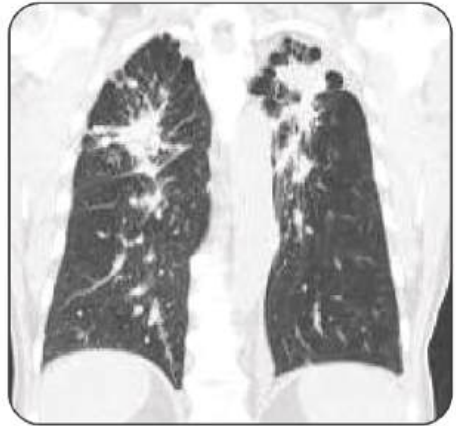
- **Chronic silicosis:** Dyspnea on exertion, chronic cough, sputum, fatigue.
- **Accelerated silicosis:** More severe symptoms develop within 5-10 years.
- **Acute silicosis:** Rapid onset dyspnea, weight loss, hypoxemia, often fatal.

**Complications:** Tuberculosis (silico-TB), lung cancer, COPD, cor pulmonale.

## Diagnosis

Diagnosis requires correlation of exposure history, radiology, and lung function:

- **History:** Duration, intensity, and type of exposure.
- **Imaging:** Chest X-ray showing nodules, coalescent masses, or progressive massive fibrosis (PMF). HRCT provides greater sensitivity.



- **Pulmonary Function Tests:** Restrictive, obstructive, or mixed pattern; reduced DLCO.
- **Exclusion of other causes:** TB, sarcoidosis, hypersensitivity pneumonitis.

Silicosis should be suspected in any worker with respiratory symptoms and occupational exposure to silica dust.

## Disease Severity & Grading

Severity is classified by radiological findings and lung function:

- Mild: Small nodules, FEV1 >80%.
- Moderate: Coalescent nodules, FEV1 60-79%.
- Severe: Progressive massive fibrosis, FEV1 <60%, hypoxemia.

The ILO International Classification of Radiographs of Pneumoconioses is recommended for standardized grading.

## Management

There is no curative therapy; management focuses on exposure cessation, symptom relief, and prevention of complications:

### A. Exposure Control:

- Immediate removal from further silica exposure.

### B. Medical Management:

- Bronchodilators and inhaled corticosteroids for airway symptoms.
- Oxygen therapy for hypoxemia.
- Treatment of infections (particularly TB).

### C. Pulmonary Rehabilitation:

- Exercise training, breathing techniques, education.

### D. Advanced Options:

- Lung transplantation in selected cases.

### E. Referral Criteria:

- Rapidly progressive disease, FEV1 <60%, PMF, or suspected lung cancer → specialist care.

## Prevention & Occupational Health

### Primary prevention:

- Engineering controls: Wet cutting methods, ventilation, dust suppression.
- PPE: N95 or higher respirators.
- Dust monitoring: Regular workplace RCS monitoring.
- Worker education and training.

### Secondary prevention:

- Annual spirometry and chest X-rays.
- Pre-placement and periodic medical exams.

### Tertiary prevention:

- Compensation schemes, rehabilitation, registry reporting.

## Prognosis & Complications

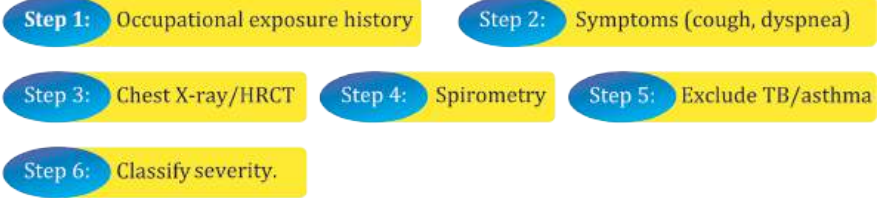
Prognosis depends on exposure cessation and disease stage. Chronic silicosis may remain stable for years, whereas accelerated and acute forms progress rapidly with poor outcomes. Complications include recurrent infections, silico-TB, lung cancer, COPD, and cor pulmonale.

## Recommendations

1. All individuals with history of silica exposure and respiratory symptoms should undergo chest imaging and spirometry.
2. GPs should refer patients with FEV1 <80% predicted, progressive symptoms, or abnormal imaging to pulmonologists.
3. Industries must adopt engineering controls, PPE, and dust monitoring per WHO/ILO standards.
4. National governments should establish silicosis registries and compensation schemes.

## Algorithm

### Diagnosis:



### Management:



## References

1. WHO: Elimination of Silicosis - Global Strategy
2. ILO: Guidelines for the Diagnosis and Classification of Pneumoconioses
3. ATS Statement on Occupational Lung Diseases
4. Indian Council of Medical Research (ICMR): Silicosis Guidelines
5. Pakistan Bureau of Statistics: Industrial workforce data
6. HRCF & Dawn reports on silicosis burden (2022-2023)

# Bagassosis

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Author: Dr Khalid Waheed

Bagassosis is a rare form of hypersensitivity pneumonitis (HP), also known as extrinsic allergic alveolitis, caused by the inhalation of dust from bagasse, the fibrous residue left after the extraction of juice from sugarcane. This dust, when contaminated with thermophilic microorganisms such as *Thermoactinomyces sacchari*, can trigger an immunologically mediated inflammatory response in the alveoli and interstitial tissues of the lungs. Though uncommon, bagassosis remains an important occupational lung disease, particularly in countries with a significant sugarcane processing industry. Being one of the largest sugar manufacturing country and still the presence of industries in the unorganized sector, it is one of unnoticed diseases in some part of our country.

There is no specific, recent data on the exact number of cases in Pakistan, research suggests it's a rare disease and is now almost non-existent due to preventive measures. Historically, cases were more common in sugar factory workers, but advancements in dust control and bagasse processing have significantly reduced its incidence. Bagassosis was initially more prevalent in sugar factories, especially during the manual processing of bagasse when dust levels were higher. The use of dust control measures (wetting bagasse, improving ventilation, using masks), as well as advancements in machinery and processing methods, have dramatically decreased the incidence of bagassosis.

## **Etiology and Pathogenesis**

### **Causative Agents**

The primary etiological factor in bagassosis is the inhalation of moldy bagasse dust containing thermophilic actinomycetes, particularly *Thermoactinomyces sacchari* and *Thermoactinomyces vulgaris*. Other implicated organisms include *Aspergillus* species and various thermophilic fungi and bacteria.

### **Source of Exposure**

Bagasse is used industrially in:

- Paper and pulp manufacturing
- Biofuel and biomass production
- Animal feed preparation
- Construction materials like particleboard

Microbial contamination occurs when bagasse is stored in damp, warm conditions, which promotes the proliferation of thermophilic organisms.

### **Occupational Risk and Environmental Exposure**

Occupational groups at risk include:

- Sugarcane mill workers
- Workers in paper, pulp, and particleboard industries using bagasse
- Biofuel plant workers
- Animal feed processors
- Agricultural workers handling stored bagasse

## Pathophysiology

Bagassosis is an entity caused by an allergic reaction to bagasse dust which is a major remaining product of the sugarcane industry. This product is used in the manufacture of pulp, paper industries, as biofuel or in lacquer ware crafts.<sup>1,2</sup> The size of the bagasse dust particle ranges from 0.08 to 9  $\mu\text{m}$ . This is significant because the dust particles of size 0.4  $\mu\text{m}$  can reach the respiratory bronchioles and cause either sensitization, alveolitis, or subsequent fibrosis depending on the duration of exposure, with the influence of individual's genetic and environmental factors.<sup>1,3,4</sup>

Bagassosis is a classic example of hypersensitivity pneumonitis resulting from an exaggerated immune response to inhaled organic antigens.

## Immunological Mechanisms

1. Type III hypersensitivity: Immune complex formation leads to complement activation and neutrophilic inflammation.
2. Type IV hypersensitivity: A delayed-type response mediated by sensitized T cells.

## Histopathology

- Alveolitis with mononuclear infiltrates
- Non-caseating granulomas
- Interstitial fibrosis in chronic stages

Prolonged antigen exposure results in chronic inflammation, progressive fibrosis, and a restrictive ventilatory defect.

## Clinical Features

### Acute Presentation

Occurs 4-8 hours post-exposure:

- Fever, chills, malaise
- Dry cough evolving into productive cough
- Dyspnea, chest tightness
- Myalgia and fatigue

Symptoms typically remit on cessation of exposure (weekends, vacations).

### Chronic Presentation

Results from repeated or continuous exposure:

- Progressive exertional dyspnea
- Chronic cough with sputum
- Weight loss, fatigue
- Digital clubbing and signs of cor pulmonale in advanced disease

### Physical Examination

- Fine, end-inspiratory crackles (velcro-like), especially at lung bases
- Occasional wheezing

## Diagnostic Evaluation

### History and Clinical Suspicion

A detailed occupational history is pivotal. Correlation of symptoms with exposure supports diagnosis.

### **Pulmonary Function Tests (PFTs)**

- Restrictive pattern: ↓ TLC, ↓ FVC
- Normal or ↑ FEV1/FVC ratio
- ↓ DLCO due to impaired gas exchange

### **Radiological Imaging**

- **Chest X-ray:** Diffuse reticulonodular opacities,
- **High-Resolution CT (HRCT):**
  - Ground-glass opacities mainly in middle and lower zones
  - Centrilobular nodules
  - Mosaic attenuation
  - Interstitial fibrosis in chronic cases

### **Bronchoalveolar Lavage (BAL)**

- Lymphocytosis (>30%)
- Possible presence of mast cells and neutrophils in acute settings

### **Serologic Tests**

- Precipitin (IgG) antibodies against *Thermoactinomyces sacchari*

### **Histopathology**

Indicated in uncertain cases:

- Interstitial inflammation
- Non-caseating granulomas
- Fibrosis in chronic stages

### **Differential Diagnosis**

- Farmer's lung
- Bird fancier's lung
- Chronic obstructive pulmonary disease (COPD)
- Sarcoidosis
- Idiopathic pulmonary fibrosis (IPF)

### **Management**

#### **Avoidance of Exposure**

The cornerstone of treatment:

- Immediate cessation of exposure to bagasse dust
- Reassignment to non-exposed areas in the workplace

#### **Pharmacological Therapy**

- **Corticosteroids:** Prednisone (0.5-1 mg/kg/day) for 2-4 weeks, followed by tapering
- **Bronchodilators:** For relief in cases with bronchospasm

#### **Supportive Therapy**

- Supplemental oxygen for hypoxemia
- Pulmonary rehabilitation in chronic disease

#### **Occupational Health Measures**

- Engineering controls: Effective dust extraction and ventilation
- Use of personal protective equipment (PPE)

- Pre-employment and periodic health screening
- Medical surveillance of at-risk workers

### **Prognosis**

- **Favorable** if identified early and exposure is discontinued
- **Poorer outcomes** with delayed diagnosis and continued exposure, leading to irreversible fibrosis and respiratory failure
- Corticosteroid therapy improves symptoms and prevents progression in early stages

### **Prevention**

#### **Workplace Interventions**

- Dust control systems and proper ventilation
- Regular equipment maintenance to prevent dust buildup

#### **Personal Protection**

- Mandatory use of masks and respirators
- PPE training and compliance

#### **Health Education and Surveillance**

- Worker education on risks and early symptoms
- Routine health monitoring including lung function tests

### **Conclusion**

Bagassosis, although rare, remains a significant occupational lung disease in regions with extensive sugarcane processing. Awareness among clinicians and industrial stakeholders is critical for early recognition, diagnosis, and prevention. A multidisciplinary approach involving occupational health, pulmonology, and industrial hygiene is essential for the effective management and control of this disease.

## References

1. Madu A. Bagassosis. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 [updated 2023 Jul 10; cited 2025 Oct 28]. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK534874/>
2. Institute of Cost and Management Accountants of Pakistan (ICMAP). Synopsis of Sugar Industry in Pakistan. Karachi: ICMAP; 2023. Available from: [https://www.icmap.com.pk/downloads/Synopsis\\_of\\_Sugar\\_Industry.pdf](https://www.icmap.com.pk/downloads/Synopsis_of_Sugar_Industry.pdf)
3. Arun T, Sinha B. Bagassosis - a rare cause of hypersensitivity pneumonitis: a case report and review. *Indian J Occup Environ Me* 2020;24(1):63-6.
4. Barnes H, Troy L, Patel K, Hsu M, Glaspole I, Lancaster LH, et al. Hypersensitivity pneumonitis: current concepts in diagnosis and management. *J Clin Med*. 2022;11(2):393. doi:10.3390/jcm11020393.
5. Romeo L, Cavalcanti M, Lo Presti A, Beltrano C, Santagata M, Ponticello A, et al. Respiratory health effects and immunological response to exposure to bagasse dust in sugarcane industry workers. *Int Arch Occup Environ Health*. 2009;82(1):41-8.
6. Bakr WMK, Ezz El-Din A, El-Sayed NM. Concentration and size distribution of inhalable and respirable dust among sugar industry workers. *Egypt J Occup Med*. 2015;39(1):85-98.
7. Nafees AA, Fatmi Z, Kadir MM, Sathiakumar N. Respiratory symptoms and lung function among sugar mill workers in Pakistan: a cross-sectional study. *Int J Occup Environ Health*. 2008;14(2):119-26-
8. Munir A, Malik SA, Ahmad M. Occupational health status in sugar industry of Pakistan. *Pak J Life Soc Sci*. 2012;10(2):128-31.
9. Riaz M, Saleem A, Fatima H. Assessment of occupational health and safety practices in Noon Sugar Mill, Bhalwal, Pakistan. *J Biodivers Environ Sci (JBES)*. 2017;11(1):97-104.
10. Sforza GGR, Marinou A. Hypersensitivity pneumonitis: a complex lung disease. *Eur Respir Rev*. 2017;26(144):170056. doi:10.1183/16000617.0056-2017.

# Byssinosis

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Author: Dr. Istikhar Ali Sajjad

Byssinosis, is a heterogeneous disease affecting lungs caused by exposure to raw, nonsynthetic textile material during their manufacturing process. Most common being the cotton but also caused by exposure to flax, jute or hemp dust.

Byssinosis has been recognized as an occupational disease since the 18th century. The term "byssinosis" was coined in 1970 to describe the disease. In the 1960s and 1970s, studies in the United States and the United Kingdom demonstrated a clear link between cotton dust exposure and respiratory disease. This disease complex is also known as "cotton worker's Lung" "Brown Lung disease" and Monday morning fever (attributed so, as workers get symptoms of fever and cough on Monday after not having work on weekend).

## Epidemiology:

Byssinosis is a significant occupational health concern in Pakistan, particularly in the textile industry. Studies have shown that 20-30% of textile workers in Pakistan suffer from byssinosis. A study<sup>1</sup> conducted in Pakistan showed that 26.6% of the workers had symptom of frequent sputum production and 16.7% had frequent cough. While in China a cohort study from 1981 to 1996 showed 15.3% workers reported byssinosis as compared to 7.6% at baseline. While talking about the developed world, the Center of Disease Control and Prevention (CDC) data showed decline in number of deaths referred to Byssinosis between 1979 and 2014.<sup>3</sup> the incidence is still on raise in developing countries like India, Pakistan, Nepal, Bangladesh and Sri Lanka. The prevalence of byssinosis is higher in smaller, unregistered textile mills, where workers are often exposed to high levels of cotton dust without adequate protective measures.

## Causes and Risk Factors

The primary cause of byssinosis is inhaling cotton dust, which contains tiny particles of cotton fibers, dust, and other contaminants. Workers in the textile industry, particularly those involved in carding, spinning, and weaving, are at highest risk of developing byssinosis. Other risk factors include:

1. **Duration and intensity of exposure:** Workers with longer duration and higher intensity of exposure to cotton dust are more likely to develop byssinosis.
2. **Concentration of cotton dust:** Higher concentrations of cotton dust in the work environment increase the risk of developing byssinosis.
3. **Poor ventilation:** Inadequate ventilation in the work environment can lead to higher concentrations of cotton dust and increased risk of byssinosis.
4. **Smoking:** Smoking can exacerbate the effects of cotton dust exposure and increase the risk of developing byssinosis.
5. **Gene Environment Interaction:** Workers with an A allele of TNF-308G/A polymorphism have a greater FEV1 decline with endotoxin exposure.<sup>4</sup>

### **Pathogenesis:**

It is now thought that the cotton dust directly causes the disease and some believe that the causative agents are endotoxins that come from the cell walls of gram-negative bacteria that grow on the cotton. Endotoxins are an independent risk factor for decline in FEV1 and FVC 2. Although bacterial endotoxin is a likely cause, the absence of similar symptoms in workers in other industries exposed to endotoxins makes this uncertain. However the evidence supporting role of endotoxins in pathogenesis of Byssinosis is that endotoxins are detected in cotton dust. Many animal and human studies support that endotoxins produces airway inflammation and ambient concentrations of endotoxins correlate with expiratory flow rate reduction.

The steps in pathogenesis are as follows

1. **Inhalation of cotton dust/ endotoxins:** Cotton dust, containing tiny fibers and endotoxins, is inhaled into the lungs.
2. Activation of immune cells: Immune cells, such as macrophages and T-lymphocytes, recognize cotton dust as a foreign substance and become activated.
3. **Release of inflammatory mediators:** Activated immune cells release inflammatory mediators, such as cytokines and chemokines, which recruit more immune cells to the lung.
4. **Inflammation and airway damage:** The influx of immune cells and inflammatory mediators leads to inflammation and damage to the airways, including the bronchioles and alveoli.
5. **Airway obstruction:** Chronic inflammation and airway damage lead to airway obstruction, characterized by narrowing of the airways and increased airway resistance.
6. **Fibrosis and lung scarring:** Prolonged exposure to cotton dust can lead to fibrosis and lung scarring, further impairing lung function.

### **Clinical presentation:**

The main distinction of byssinosis from other respiratory diseases comes from the initial exposure. Patients who have byssinosis have typically been exposed to cotton or just dust for an extended period of time and experience symptoms of chest tightness and coughing. The symptoms of byssinosis can vary depending on the severity of the disease and the individual's overall health.

Typical symptoms include chest tightness, cough, breathlessness, and wheezing, especially on the first working day after a weekend break ('Monday Fever' phenomenon). Systemic symptoms like fatigue and low-grade fever may also occur. On examination, clinicians may find tachypnea, accessory muscle use, wheezes, rhonchi, and inspiratory crackles in advanced cases.

### **Severity Assessment:**

Byssinosis severity is graded using the Schilling criteria, which assesses the presence and intensity of chest tightness/breathlessness on return to work after a weekend off, with grades ranging from no symptoms to chronic lung impairment.

Following is the Schilling criteria for byssinosis severity grading:

**Grade 0:** No symptoms of chest tightness or breathlessness on the first day back at work.

**Grade 1/2:** Occasional chest tightness or breathlessness on some of the first days back at work.

**Grade 1:** Chest tightness or breathlessness on most of the first days back at work.

**Grade 2:** Chest tightness or breathlessness on the first and other days of the work week.

**Grade 3:** Grade 2 plus chronic lung impairment, indicated by a forced expiratory volume in 1 second (FEV1) less than 80% of the predicted value or Radiological evidence of fibrosis

### **WHO grading system:**

The World Health Organization (WHO) also proposed a modification of the Schilling classification that incorporates spirometry.<sup>9</sup>

1. No effect: FEV1 = 80% of predicted value
2. Mild to moderate effect FEV1 60%-79% of predicted value
3. Severe effect: FEV1 less than 60% of predicted value

## **INVESTIGATIONS**

### **1. Pulmonary Function Tests:**

In Byssinosis, pulmonary function tests (PFTs) before, after and in between work shifts may be helpful. PFTs typically show:<sup>10</sup>

1. Reduced forced expiratory volume (FEV1): Decreased FEV1 indicates airway obstruction.
2. Reduced forced vital capacity (FVC): Decreased FVC suggests restrictive lung disease.
3. Increased residual volume (RV): Elevated RV indicates air trapping.
4. Decreased forced expiratory flow (FEF) rates: Reduced FEF rates, such as FEF25-75, indicate small airway obstruction.
5. Normal or decreased diffusing capacity (DLCO): Decreased DLCO suggests impaired gas exchange.

These changes in PFTs are consistent with a mixed obstructive-restrictive pattern, which is characteristic of Byssinosis.

Additionally, PFTs may also show:

- Reversible airway obstruction: Improvement in FEV1 and FVC after bronchodilator administration.
- Expiratory flow limitation: Reduced flow rates during forced expiration.

These findings help diagnose and monitor the progression of Byssinosis.

### **2. Radiological Findings:**

Chest X-rays are often not sensitive enough to detect early changes in Byssinosis. X-ray findings may be used to rule out other conditions, such as pneumonia or lung cancer.

In Byssinosis, chest X-rays may appear:

1. **Normal in early stages:** Early Byssinosis may not show significant X-ray changes.
2. **Hyperinflation:** Increased lung volumes and flattened diaphragms may be seen.
3. **Peribronchial thickening:** Thickening of bronchial walls may be visible.
4. **Volume loss and fibrosis:** X-rays may show signs of reticulations volume loss and fibrosis in advance cases.

HRCT chest findings may show normal HRCT or findings similar to hypersensitivity pneumonitis. Which may show GGO, Centrilobular micronodularity, reticulations predominantly in upper and mid zones or head cheese sign.

### **Pulmonary Rehabilitation**

Pulmonary rehabilitation is a comprehensive program that helps improve lung function, overall health, and quality of life. The program typically includes:

1. **Exercise Training:** Regular exercise helps improve lung function and overall fitness.
2. **Education:** Patients learn about their condition, symptoms, and management strategies.
3. **Breathing techniques:** Patients learn breathing techniques to help manage symptoms.
4. **Support:** Patients receive support and guidance from healthcare professionals.

### **Personal Protective Equipment**

Using personal protective equipment (PPE) can help reduce exposure to cotton dust. PPE includes:

- **Respirators:** Respirators can filter out dust particles, reducing exposure.
- **Masks:** Masks can also help reduce exposure, although they may not be as effective as respirators.

### **Symptom Monitoring:**

Regular monitoring of symptoms and lung function is crucial in managing Byssinosis. This includes:

1. **Spirometry:** Regular lung function tests help monitor disease progression.
2. **Symptom tracking:** Patients track their symptoms, allowing for early detection of changes.

### **Prevention:**

Prevention is key in managing Byssinosis. Strategies include:

1. **Engineering controls:** Improving ventilation and reducing dust levels can minimize exposure.
2. **Worker Education:** Educating workers about risks and prevention strategies can help reduce exposure.
3. **Regular health surveillance:** Monitoring workers' health and lung function can help detect early signs of disease.

### **Prognosis:**

The prognosis for Byssinosis varies depending on the severity of the disease and the effectiveness of management. With early diagnosis and management, symptoms can be alleviated, and disease progression can be slowed. A longitudinal study of textile workers found byssinosis to be associated with increased cancer risk, especially GIT related carcinomas.<sup>11</sup>

In conclusion, managing Byssinosis requires a comprehensive approach, focusing on removing the individual from exposure, alleviating symptoms, and preventing further lung damage. By working together, healthcare professionals, employers, and individuals can help reduce the burden of Byssinosis.

## References:

1. Nafees AA, Fatmi Z, Kadir MM, Sathiakumar N. Pattern and predictors for respiratory illness and symptoms and lung function among textile workers in Karachi, Pakistan. *Occup Environ Med.* 2013;70(2):99-107.
2. Christiani DC, Wang XR, Pan LD, et al. Longitudinal changes in lung function and respiratory symptoms in cotton textile workers. A 15 years follow up study. *Am J Respir Crit Care Med.* 2001;163(4):847-853.
3. National institute for Occupational Safety and Health. Byssinosis: number of deaths, crude and age-adjusted death rates, U.S. residents age 15 and over, 1979-2014. [www.cdc.gov/eWorld/Data/918](http://www.cdc.gov/eWorld/Data/918). Accessed March 28, 2022.
4. Zhang H, Hang J, Wang X, et al. TNF polymorphisms modify endotoxins exposure-associated longitudinal lung function decline. *Occup Environ Med.* 2007;64(6):409-413.
5. Patel, Pujan H.; Yarrarapu, Siva Naga S.; Anjum, Fatima (2022), "Byssinosis", StatPearls, Treasure Island (FL): StatPearls Publishing, PMID 30137833, archived from the original on 2022-08-04, retrieved 2022-02-10
6. Catellan RM, Olenchock SA, Kinsley KB, Hankinson JL. Inhaled edotoxin and decreased spirometric values. An exposure-response relation for cotton dust. *N Engl J Med.* 1987;317(10):605-610.
7. Schilling RS. Respiratory disability in ex-cotton workers. *Br J Ind Med.* 1987;44:571-572.
8. Kilburn K. recommended-health-based occupational exposure limits for selected vegetable dusts. *World Health Organ Tech Rep ser.* 1983;684:1-78.
9. Nafees, Asaad Ahmed; Matteis, Sara De; Burney, Peter; Cullinan, Paul (2022-01-24). "Contemporary Prevalence of Byssinosis in Low- and Middle-Income Countries: A Systematic Review". *Asia-Pacific Journal of Public Health.* 34 (5): 483-492. doi:10.1177/10105395211073051. hdl:10044/1/96110. ISSN 1010-5395. PMID 35073782. S2CID 246277619. Archived from the original on 2022-02-10. Retrieved 2022-02-10.
10. Khan AJ, Nanchal R. Cotton dust lung disease. *Curr Opin Pulm Med.* 2007;13(2):137-141.
11. Fang SC, Mehta AJ, Hang JQ, et al. cotton dust, endotoxin and cancer mortality among the Shanghai textile workers cohort: a 30-yr analysis. *Occup Environ Med.* 2013;70(10):722-729.

# Lung Diseases Due To Aluminium Dust Exposure

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Aluminium production and use both result in the generation of various particles, fumes, gases, and airborne materials with the potential for inducing a wide range of lung pathology. Occupational exposure to aluminium dust is increasingly recognized as a cause of chronic respiratory disease. Workers in aluminium production, polishing, welding, bauxite mining, and metalworking are particularly at risk. This guideline synthesizes current evidence for recognition, diagnosis, management, and prevention of aluminium dust-related lung disease, providing clinicians with a structured approach.

## Epidemiology & Burden

Several reports of interstitial lung disease attributable to aluminium exposure have appeared in the medical literature over the past 70 years. Globally, millions of workers in aluminium production and processing are exposed to respirable dust. In South Asia, exposure is common in unregulated industries such as polishing, cookware production, and recycling plants. Data are limited, but case series from Germany, China, and Canada have reported aluminosis and asthma among exposed workers. In Pakistan, occupational surveillance is weak, and cases are likely underdiagnosed or misclassified as COPD or asthma.

## Etiology & Risk Factors

Diseases arise from inhalation of fine aluminium particles and aluminium oxide. Risk factors include:

**Occupations:** smelting, mining, welding, polishing, recycling

**Duration and intensity of exposure**

**Lack of dust control and PPE**

**Smoking** (synergistic with chronic bronchitis and lung cancer)

**Co-exposure** to silica, asbestos, or other industrial dusts

## Pathophysiology

Aluminium dust triggers macrophage activation, reactive oxygen species (ROS) generation, and fibrotic pathways leading to restrictive and obstructive defects. Hence, the following disease patterns can result from aluminium dust exposure.

1. **Aluminosis or Shaver's disease:** was first described in workers exposed to aluminium and silica. Aluminosis is an interstitial lung fibrosis characterized by upper lung predominance, peripheral emphysema, and frequent rupture of emphysematous blebs causing pneumothorax. This pattern of pulmonary fibrosis is also called aluminium-lung.
2. **Asthma and Chronic Bronchitis:** Airway inflammation and hyperresponsiveness triggered by aluminium dust exposure leads to asthma which ultimately ends up in chronic bronchitis.
3. **Hypersensitivity alveolitis:** Results from immune-mediated interstitial inflammation.
4. **Pulmonary Alveolar Proteinosis (PAP):** Aluminum-induced Interstitial Lung Disease can present as PAP characterized by the accumulation of a lipoproteinaceous material in the alveoli.

5. **Desquamative Interstitial Pneumonia (DIP):** Aluminium exposure can present as DIP, characterized by intra-alveolar accumulation of macrophages.
6. **Mediastinal lymphadenopathy:** Aluminium dust exposure can present predominantly with mediastinal lymphadenopathy, with evidence of aluminium particles in the mediastinal lymph nodes. It may also be associated with pneumonitis, with biopsies showing granulomas similar to those seen in sarcoidosis and chronic beryllium disease. The disease differs from sarcoidosis by the identification of aluminium within the granulomas.
7. **Lung cancer:** chronic inflammation and genotoxic effects of aluminium compounds.

Given commonly occurring concurrent exposure to other fibrogenic fibres in work environments, fibrogenic lesions observed in aluminum-exposed workers have been attributed to exposure to a mixture of other dusts such as silica and asbestos rather than aluminum alone. However, analysis of tissues or fluids of the lungs of workers exposed to aluminium dust showed high concentrations of aluminium fibers, raising the possibility of aluminum-induced fibrosis.

#### **Clinical presentation:**

Presentations vary by disease type:

**Aluminosis:** exertional dyspnea, dry cough, basal crackles, and clubbing

**Chronic bronchi:** productive cough for  $\geq 3$  months/year for 2 consecutive years

**Asthma:** episodic wheeze, cough, chest tightness, reversible obstruction

**Alveolitis:** acute/subacute dyspnea, fever, inspiratory crackles

**Lung cancer:** weight loss, hemoptysis, persistent cough

#### **Diagnosis**

##### **A. Occupational History:**

Job type, duration, intensity of aluminium exposure, ventilation, PPE use, and co-exposure.

##### **B. Clinical Evaluation:**

Detailed symptom analysis

Examination: crackles, wheeze, clubbing

##### **C. Investigations:**

**Spirometry:** Obstructive (asthma/bronchitis) or restrictive (aluminosis/fibrosis)

**Chest X-ray:** interstitial opacities

**HRCT:** nodules, fibrosis, ground-glass opacities characterized by upper lung predominance

**Bronchoscopy/BAL:** rule out alternative diagnoses

**Biopsy:** only if diagnosis uncertain

##### **D. Differential Diagnosis:**

COPD, asthma, IPF, silicosis, asbestosis, hypersensitivity pneumonitis

##### **Disease Severity & Grading**

Severity can be classified by clinical, functional, and radiological criteria:

**Mild:** FEV<sub>1</sub>  $\geq 80\%$ , minimal symptoms, HRCT shows early fibrosis

**Moderate:** FEV<sub>1</sub> 60–79%, exertional dyspnea, interstitial opacities

**Severe:** FEV1 <60%, advanced fibrosis, hypoxemia, cor pulmonale

**Management**

**A. General Measures:**

Immediate removal from aluminium dust exposure

Smoking cessation

**B. Pharmacological Therapy:**

Bronchodilators and inhaled corticosteroids (asthma/bronchitis)

Anti-fibrotics (pirfenidone, nintedanib) in progressive fibrosis (experimental)

Systemic steroids for hypersensitivity pneumonitis (short course) Chemotherapy/radiotherapy for lung cancer (oncology referral)

**C. Supportive Care:**

Pulmonary rehabilitation Oxygen therapy in hypoxemia Vaccinations: influenza, pneumococcal

**D. Referral:**

Progressive disease, FEV1 <60%, suspected malignancy → specialist care

**Prevention & Occupational Health**

Prevention strategies include:

Engineering controls: dust suppression, wet methods, exhaust ventilation PPE: respirators (N95 or higher)

Workplace dust monitoring and threshold limit values

Health surveillance: pre-placement and annual medical exams

Worker education and training

National registries and compensation schemes

**Prognosis & Complications**

Prognosis varies:

**Aluminosis:** progressive, may lead to respiratory failure

**Chronic bronchitis/asthma:** variable, improved with exposure cessation **Alveolus:** can improve if diagnosed early and exposure avoided

**Lung cancer:** poor prognosis, late diagnosis common

Complications include cor pulmonale, recurrent infections, respiratory failure, and malignancy

**Algorithms**

**Diagnosis:**

**Step 1:** Occupational history

**Step 2:** Symptoms

**Step 3:** Spirometry

**Step 4:** Chest imaging (CXR/HRCT)

**Step 5:** Exclude differentials

**Step 6:** Classify phenotype.

## Management:

**Step 1:**

**Confirm**

**Step 2:**

**Remove from exposure**

**Step 3:**

**Symptom-based therapy (ICS/LABA, antibiotics, steroids)**

**Step 4:**

**Pulmonary rehabilitation**

**Step 5:**

**Oxygen therapy**

**Step 6:**

**Oncology referral if malignancy suspected.**

## Key Recommendations:

- Early recognition of occupational exposure and symptom onset
- Use of detailed exposure history, spirometry, and imaging as primary diagnostic tools
- Classification of disease phenotypes (aluminosis, bronchitis, asthma, fibrosis, cancer)
- Stepwise management including exposure cessation, pharmacotherapy, pulmonary rehabilitation, and referral when indicated
- Strong emphasis on prevention: dust suppression, PPE, surveillance, and regulatory compliance
- Clear referral criteria for advanced disease and suspected malignancy

## References:

- 1.WHO: Aluminium exposure and health risks–Global Strategy.
- 2.ATS/ERS Occupational Lung Disease Guidelines.
- 3.ILO: Dust control in aluminium industries.
- 4.German case series on aluminosis (BMC Pulm Med, 2019).
- 5.BMJ Best Practice: Aluminium-induced lung disease.
- 6.Occupational Medicine Texts–Aluminium exposure and respiratory outcomes.
- 7.Vahlensieck M, Overlack A, Muller KM. Computed tomographic high-attenuation mediastinal lymph nodes after aluminum exposition. *Eur Radiol.* 2000;10:1945–1946
8. Miller RR, Churg AM, Hutcheon M, et al. Pulmonary alveolar proteinosis and aluminum dust exposure. *Am Rev Respir Dis.* 1984;130:312.
- 9.Herbert A, Sterling G, Abraham J, et al. Desquamative interstitial pneumonia in an aluminium welder. *Hum Pathol.* 1982;13:694–699.
- 10.Voisin C, Fisekci F, Buclez B, et al. Mineralogical analysis of the respiratory tract in aluminium oxide-exposed workers. *Eur Respir J.* 1996;9: 1874–1879.

# Medico-legal Aspects Of Occupational Lung Disease

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## Introduction

The purpose of medical guidelines is to provide evidence-based recommendations for the investigation and management of medical conditions.

In the case of this present document, respiratory diseases which occur because of an individual's occupation. Guidelines set out the standards to be achieved; they identify "best practice", based primarily on the medical literature, but also upon the experience and judgement of specialists in the field, where the medical literature is lacking. Guidelines can help identify those areas in which the evidence is insufficient for firm recommendations, and therefore the areas in which further research would be of value. Importantly, guidelines must take into account what is practical in the medical and societal setting in which they are to be implemented: guidelines cannot simply be a statement of "the ideal".

This chapter, on the medicolegal aspects of occupational lung disease, inevitably must be different from the other chapters in this publication. It cannot guide doctors and other healthcare professionals as to what their own "best practice" should be, because so much is out of their control. Doctors (and others) can be guided as to how to diagnose and treat (for example) mesothelioma arising from asbestos exposure, or hypersensitivity pneumonitis due to occupational exposures. Preventing these conditions from arising in the first place is a different matter: it involves government's action, and the attitudes of employers. Once the condition does arise and is diagnosed, the matter of whether or not compensation is available for those damaged by their work is also outside the control of doctors; it depends again upon government and the Courts.

Accordingly, this chapter cannot set out an account of precisely what must be done, from the compensation point of view by a doctor faced with an individual with an occupational lung disease. Nor will it advise what the ideal legal framework would be for prevention of those diseases: that ideal framework does not exist anywhere in the world, and it never will. Where progress has, though, been made in both prevention and compensation, in all countries, it has been attributed to the efforts of informed doctors and dedicated healthcare professionals, accompanied by pressure from workers themselves, in particular by Trade Unions. Accordingly, members of the Pakistan Chest Society have an important role to play in shaping the future especially in the field of prevention of occupational lung disease. Finally, it is important that we stress that this chapter is written by respiratory specialists, and not by lawyers. The views expressed are therefore from the, medical perspective, and they must not be interpreted as being definitive statements of the law.

### **Occupational lung disease and the medico-legal process: the principal aspects.**

Readers of these guidelines may find it helpful to consider, first, the broad legal aspects of occupational lung disease, and a very simple outline of the legal system may assist.

The Islamic Republic of Pakistan has what is referred to as a common law system. This means that laws are derived not only from the Constitution of 1973, and from Statutes, these being the laws that are enacted by the legislature. A common law system means that, in addition, the decisions of Judges in previous similar cases are important in determining how a new case will be decided. The higher the status of the Court making the earlier decision, the greater the influence on all future similar cases. Laws enshrined in the Constitution, and in Statutes, will though always take precedence over decisions of a Court.

Importantly, in Pakistan, the common law model is modified by incorporation of with the Federal Shariat Court intended to ensure that laws are aligned with Islamic principles. Finally, some parts of the law are determined at a national level and others are devolved to the provinces i.e. there is a federal system, as in (for example) the states of Germany, and the states of the USA.

It will accordingly be seen that the legal system in Pakistan is complex, as indeed it is in many other countries. When thinking about occupational lung disease, though, a simple way of looking at these legal matters is to divide the issue into two parts:

First, what are the obligations upon employers to protect their workforce?

Second, what rights to support and compensation do workers have if they are in fact damaged?

### **The duties of employers**

In theory, there are extensive legal protections for workers in Pakistan. The Constitution of 1973 is the Supreme law of the land, and amongst its provisions are ones which make it clear that employers have a duty to provide a safe workplace. The Factories Act 1934, with its amendments, includes a number of specific requirements regarding ventilation, temperature, cleaning and disinfection, lighting, provision of drinking water and sanitation. Of particular relevance to occupational lung diseases are restrictions on exposure to fumes and dust. Penalties are specified in law: the Penal Code originated in 1860 and was adopted as the Pakistan Penal Code at independence in 1947. With its later amendments, it sets out the penalties that may be imposed upon employers if they cause death or injury amongst the workforce.

The Labor Policy (2010, and its predecessor Policies) aims to help industrial relations between employers and the workforce, and obviously the results of effective negotiations between these two groups would be to improve working conditions and safety, and reduce the risk of industrial accidents and disease, including lung diseases. The intention of the 2010 policy is to have conditions in accordance with International Labour Standards, as set out by the International Labour Organisation (ILO). The Pakistan Occupational Health and Safety Act 2018 builds on earlier legislation, and gives details of wide-ranging measures which should be implemented in workplaces.

It is, though, very important to understand the limitations of what at first sight appears to be a reasonably comprehensive set of protections for workers. First, it is no use having rules if the opportunities to enforce them are limited. Pakistan has a well-organized factory inspectorate, but as in many and probably most other countries, it is under resources given

the scale of the task it faces. Second, the protective legislation is often industry-specific. The Factory Act, for instance, applies to “manufacturing facilities”, and not to (for instance) the construction industry and agriculture.

Third, while it may be possible to inspect and try to enforce standards on large companies, it may be unrealistic to expect the same level of worker protection to be present in very small-scale workplace settings. Of course, in an ideal world those levels of protection would indeed be exactly the same, wherever an employee is working, but in practice this is unlikely to be achievable. Finally, most occupational lung diseases arise from long-term, indeed sometimes lifelong, exposures and their prevention means attention to working conditions over the course of years or even decades.

### **The rights of workers to support and compensation**

Pakistan was one of the earliest countries in the world to enshrine in law the rights of workers to compensation. The fundamental legislation is over 100 years old: the Workmen’s Compensation Act 1923 Act (now often known as the Employees Compensation Act). This set down in law the duty of certain classes of employers (note, not all employers) to compensate a worker who was injured by an accident. It will be seen from the wording that those passing that Act had particularly in mind workers sustaining a traumatic injury, or being killed, during the course of their work. Indeed, the supplementary Schedule to the Act contains tables of the type of injury that would be liable for compensation.

How applicable, though, is this to occupational lung disease? Respiratory specialists will readily see that injury by “an accident” does not fit easily with what we have learnt about occupational lung disease during this past century. Of course, there are some patients whose problems we can trace back to a single acute “injury”, one example would be where accidental exposure to chlorine gas led to the development of acute irritant-induced asthma (also often known as Reactive Airways Dysfunction Syndrome – RADS). These cases though are rare, and much more commonly we as practising chest specialists are faced with patients whose problems have arisen in consequence of long term exposures at work: presenting after months to years (occupational asthma), or even after employment has ceased (some cases of silicosis and asbestosis; mesothelioma).

Fitting cases such as these into the definition “injury by an accident” has of course been resisted very strongly by those responsible for payment of compensation. In practice, payments for compensation are usually not made by the employers (or previous employers) themselves, but by their insurers. Of course, large (often multinational) insurance companies have teams of lawyers whose job is to prevent payouts of compensation unless it is absolutely necessary to do so, and occupational lung diseases are a prime example of what insurers call ‘long-tail’ claims, where symptoms and diagnosis may not occur for many years, or even decades, after the relevant exposure. Major legal cases have been fought on this very point in many countries around the world.

In some countries, things have been made easier for those with occupational lung diseases (and occupational diseases affecting other organs) by the introduction of Government compensation schemes. These are particularly important in countries which have a so-called civil law system, such as Scandinavian countries and others in continental Europe. This introduces a further subject for discussion: should compensation be from the State (meaning the nation), or from the employer? When there are difficulties obtaining

compensation from the employer (or its insurers), State compensation is an alternative route. This means that in theory, an individual with an occupational lung disease could have two routes to compensation: from the State, or from his/her previous employer.

In the legal systems of countries like Germany and France, and the Scandinavian countries, State compensation is good, and that available by taking a previous employer to Court is limited. In common law legal systems, like the United Kingdom and the USA, both routes can be taken, and most of those with the types of occupational lung diseases that we see will pursue State compensation and a claim against previous employers (or, rather, their insurers). The second of these will, if successful, give greater compensation than that available from the State but in most countries any State compensation given will then be claimed back by the State from the employer's award of damages.

**What is the position in Pakistan?** The basic position is that the right to claim compensation from an employer is set out in law, and if the claim is successful then it is the duty of the employer to pay it. The Workmens' Compensation Act and its subsequent legislation clearly set out those rights and duties. There is, though, no comprehensive State (ie taxpayer) funded compensation scheme. The Pakistan legal system (like that in Britain, the USA, Australia and many other countries) is a common law adversarial system. The two parties in this example, the person with lung disease and the employer or its insurer are adversaries, they fight each other, and we have all heard the phrase 'he is fighting the case against him in Court'. In Pakistan, the Judge does not (or is not meant to) decide what the result should be. He or she looks at the arguments and evidence put forward by both sides and decides who has put forward the stronger case; in other words, who has won the battle.

The backlog of cases (all cases, not just for lung disease compensation) in the Courts in Pakistan is very long, and measured in years. It will be clearly seen that the Claimant with lung disease may have a very big battle to achieve their compensation. This is all the more the case if an employer does not have the money to pay, or says it does not have the money. Reference has been made above to insurance companies. In some countries Employers Liability Insurance is compulsory, even for employers with only one or two employees. In Pakistan, Employers' Liability insurance is not universally compulsory, but only in specific situations, particularly in the construction and manufacturing industries. Most small employers will not have such insurance, so the potential to claim from them is less still. In addition, the application of even the 1923 Act to agriculture is limited, despite this area involving up to 45% of the workforce in Pakistan. Death during work on what are described as "mechanised farms" is in theory covered, but it will clearly be seen that a patient with hypersensitivity pneumonitis/extrinsic allergic alveolitis from work in agriculture has effectively no chance of compensation for loss of their livelihood.

The compensation that is available is still very much based upon the "death or traumatic injury" model, set out in the 1923 Compensation Act. Consider, for instance, mining. In 2024-2025, for example, Khyber Pakhtunkhwa province made 74 death grants to the families of deceased mineworkers and 27 grants to workers permanently disabled. Similar grants have been made in some other coal-mining provinces. There are also, though, some encouraging signs in Khyber Pakhtunkhwa in that there has been some growth over the past few years in grants for non-fatal pulmonary disease in miners. This is welcome progress, but it will again be seen that this is very much industry specific, and we are a very long way indeed from a

Pakistan-wide scheme of compensation for occupational lung disease.

### **What is the way forward?**

The discussions above give a picture of the present position which might be thought of as being not particularly encouraging, but Pakistan shares such problems with a very large number of other countries. At the beginning of this chapter, we stated that we would not set out “guidelines” in the conventional sense. We do, though, conclude by making a series of suggestions which we believe form the basis for a way forward.

Members of the Pakistan Chest Society spend the great majority of their professional lives diagnosing and treating lung diseases, including occupational lung diseases. Our role, though, is in prevention as well as diagnosis and treatment, and this has been well demonstrated over the past decades in the field of smoking and lung health. We believe that the same attention should now be applied to the prevention of occupational lung diseases.

At the same time, we should be assessing and carefully documenting those patients in whom these diseases are already present, and this has two benefits. First, it means that there is the opportunity to improve assessment of the number of cases of occupational lung disease. Second, if in the years to come the payment of compensation does become easier, then there will be clear documentation in the records of those patients who might be eligible for such compensation.

Accordingly, we hope that the recommendations we make will stimulate further discussion amongst members of the Pakistan Chest Society. They have all arisen from our reflections upon the medico-legal aspects of occupational lung disease, but it will be seen that they involve considerations of the quality of patient care, the education of respiratory specialists, and the role of the Society in influencing the attitudes not only of employers but also the legislature at Provincial and at State level. All are interlinked – this is not merely a matter of ‘What compensation is available?’

1. All new patients presenting to a chest specialist should have their occupation recorded. If no longer working, their previous jobs should be listed along with potential adverse exposures.
2. In all patients in whom an occupational lung disease is suspected, this potential diagnosis should be recorded in the medical notes. If the diagnosis is then confirmed, this should again be noted.
3. A record should be made of the extent of respiratory disability. The Respiratory Disability Rating Scale, appended to these Guidelines, is a very simple symptoms-based assessment which does not require separate time to be put aside for assessment, nor the use of expensive equipment. If compensation does become available, then the degree of disability may be relevant, and use of the Scale is a simple way of charting the impact of disease progression.
4. Patients in whom an occupational lung disease is diagnosed should have this carefully explained to them, along with an explanation of how they might be able to minimise further risks. It is understood that many of them might not be able to make major changes, particularly with smaller employers and/or when there is no realistic alternative employment, but all possible efforts should be made.
5. The ready availability of video conferencing (e.g. Teams, Zoom, WhatsApp video) enables the setting up of occupational lung disease groups by respiratory specialists on a district and/or provincial basis, and also at the level of the nation as a whole. They

should meet at intervals to discuss patients who are difficult diagnostically, and to identify emerging disease patterns. As just one example of this, the increasing manufacture and use of artificial ('engineered') stone is leading to an epidemic of accelerated silicosis in young people in many countries, and Pakistan has many companies involved in this activity. Further, since there appear to be significant province by province differences in the approach to compensation, the joint experience of members of the group will be invaluable in building up a more detailed picture of what is available (and, sadly, usually not available) to a patient.

6. Pakistan has long had the legal framework in place for protection of employee health, but it is limited in the occupational settings to which it applies, and it still has largely a "death or acute injury" based approach. This chapter commenced by dividing consideration of the medico-legal aspects of occupational lung disease into two parts: the duties of employers, and the availability of compensation for patients. We believe that there is a vital role to be adopted by members of the Pakistan Chest Society in the first of these, in campaigning for the better protection of the respiratory health of the workforce. A wider range of industries and employments should be included, and emerging diseases as well as long-recognised ones must be considered.
7. It is in the interests of employers to have a healthy workforce, and this is the case whether they are large established industries or are very small employers, although both may be concerned about the potential costs of protecting against occupational lung diseases. The Pakistan Chest Society should work alongside colleagues in other organisations, such as the Pakistan Occupational Hygiene Association, in identifying simple interventions which would, if they were implemented, have an effect in reducing the burden of occupational lung diseases.
8. The Society should consider the introduction of a postgraduate diploma in occupational lung diseases, with those obtaining the qualification being recognised as specialists at district, division and provincial levels, according to the degree of their later experience. Clearly, such a step would require discussions with the College of Physicians and Surgeons of Pakistan.
9. In countries where the incidence of tobacco-related disease and death is falling, this has not come about because of better medical treatments, and nor indeed because of compensation for those affected. It is the result of legal changes, often implemented by Governments who were initially reluctant to do so, but who had to eventually give way to pressure from informed medical professionals and a public which had been educated as to the hazards. Occupational exposures are a cause of preventable lung disease, in exactly the same way that tobacco is a cause. The journey to reduce the burden of occupational disease is a long and hard one, and complete success will never be achieved in any country. Continuing dialogue between members of the Pakistan Chest Association, and interaction with those outside it, will though be vital for progress in this country.

# Medico-legal Aspects Of Occupational Lung Disease

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## SUMMARY

### Introduction

- Medical guidelines provide evidence-based recommendations for investigating and managing diseases.
- These guidelines focus on respiratory diseases caused by occupational exposures and:
  - Set standards of care and identify best practices
  - Highlight areas where evidence is lacking and where research is needed
  - Must consider what is practical and feasible in the local medical and societal context
- The medicolegal aspects of occupational lung disease differ from clinical guidelines.
  - Doctors can guide diagnosis and treatment
  - Prevention requires government and employer responsibility
  - Compensation depends on laws and courts, not doctors
- Progress in prevention and compensation on enti comes from:
  - Informed doctors and healthcare workers
  - Advocacy from workers and trade unions
  - This chapter is written by physicians, **not lawyers**, and should be interpreted as medical guidance rather than legal advice.

### Occupational Lung Disease and the Medico -Legal Process

- Pakistan follows a common law system (Constitution, statutes, and case law)
- Modified by Sharia law through the Federal Shariat Court
- Federal and provincial laws both apply (federal system)
- Two key issues:
  - Employers' obligations to protect their workforce
  - Workers' rights to support and compensation if harmed

### Duties of Employers

- Constitution of 1973: employers must provide safe workplaces.
- Factories Act 1934: specific requirements for:
  - Ventilation, temperature, cleaning, lighting, sanitation
  - Restrictions on fumes and dust exposure
- Penal Code (1860, adopted 1947) penalties for causing death or injury to workers.
- Labor Policy 2010: aims for compliance with International Labour Organisation (ILO) standards.
- Pakistan Occupational Health and Safety Act 2018: expands workplace safety measures

**Limitations:**

- Weak enforcement due to limited resources
- Many laws are industry - specific, excluding construction and agriculture
- Difficult to monitor small workplaces compared to large companies
- Most occupational lung diseases arise from long-term exposures, requiring decades of prevention measures.

**Rights of Workers to Support and Compensation**

- Pakistan was an early adopter of worker compensation laws.
- **Workmen's Compensation Act 1923**
  - Requires certain employers to compensate workers injured by accidents Originally focused on traumatic injuries and death, not long term diseases
  - Problems with occupational lung diseases:
  - Diseases typically develop years after exposure.
  - Employers/insurers resist claims, especially for long-tail diseases (e.g., silicosis, mesothelioma).
- **In other countries:**
  - Civil law systems (e.g., Scandinavia): strong state compensation schemes
  - Common law systems (e.g., UK, USA): workers may seek both state and employer based compensation
- **In Pakistan:**
  - Compensation rights exist but are limited
  - No comprehensive state-funded scheme
  - The legal process is adversarial; the claimant must fight the employer or insurer in court.
  - Severe backlog of court cases → delays of years
  - Employer insolvency or lack of compulsory insurance further limit compensation
  - Agriculture is poorly covered, despite 45% of the workforce being in this sector

**Current practice examples:**

- Khyber Pakhtunkhwa (2024–25):
  - 74 death grants for deceased mineworkers
  - 27 grants for permanently disabled miners
  - Some growth in non-fatal pulmonary disease grants → but industry-specific only

**The Way Forward**

- Despite challenges, progress is possible with collective efforts.
- Physicians' role should extend beyond diagnosis to prevention and advocacy.
- Accurate patient documentation is essential for:
  - Better disease surveillance
  - Supporting future compensation claims

### **Recommendations:**

1. Record occupation and potential exposures for all new patients
2. Document suspected or confirmed occupational lung disease in medical notes
3. Record disability using simple tools (e.g., Respiratory Disability Rating Scale)
4. Clearly explain diagnosis and prevention strategies to patients
5. Use videoconferencing to form provincial/national groups of specialists to:

Discuss difficult cases

Track emerging disease trends

Share experience on compensation variations across provinces

6. Advocate for improved protective legislation and broader industry coverage
7. Collaborate with organisations (e.g., Pakistan Occupational Hygiene Association) to identify cost-effective prevention strategies
8. Consider introducing a postgraduate diploma in occupational lung diseases, recognised at district, division, and provincial levels
9. Learn from anti-tobacco efforts:

Prevention is achieved through legal and social change, not just medical care

Long-term advocacy and public awareness are essential to reduce disease burden

