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Understanding Pneumoconiosis, A Misdiagnosed Subtype of Diffuse Interstitial Lung Disease

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Abstract

Pneumoconiosis is a lung disease caused by inhalation of organic or nonorganic airborne dust and fibers for an extended period. The clinical presentation can vary depending on the dust type and the disease's severity. Common symptoms include cough, shortness of breath, chest tightness, and wheezing. Pneumoconiosis is one of the most common occupational diseases in the world. The most frequent types include asbestosis, silicosis, anthracosis, berylliosis, siderosis, and talcosis. It is important to note that there are other types of pneumoconiosis. However, due to their exceptionally rare occurrence, they are not addressed in this review. Diagnosis of pneumoconiosis typically involves a thorough medical history, physical examination, and imaging tests such as chest X-rays and CT scans. Pulmonary function tests may also be used to assess lung function. The prognosis of pneumoconiosis depends on several factors, including the type and severity of the disease, as well as the patient's age, overall health, and exposure to the causative dust. In some cases, the condition may progress and lead to severe respiratory impairment, while in other cases, the symptoms may be mild and manageable with treatment. Treatment strategies are mainly tailored to avoid further exposure to inhalants, smoking suspension, pulmonary rehabilitation, and symptomatic treatment. Patients with end-stage disease may be candidates for lung transplants. This narrative review article presents an overview of the most common types of pneumoconiosis.

Keywords: Diffuse interstitial lung disease; Pneumoconiosis; Anthracosis; Silicosis; Talcosis

Abbreviation: DILD: Diffuse Interstitial Lung Disease; US: United States; BAF: Bronchial Anthracofibrosis; TB: Tuberculosis; COPD: Chronic Obstructive Pulmonary Disease; CT: Computed Tomography; CDC: Centers for Disease Control and Prevention; DLCO: Diffusion Capacity Of Carbon Monoxide; HRCT: High-Resolution Computed Tomography; WHO: World Health Organization; VAT: Video-Assisted Thoracoscopy; BAL: Bronchoalveolar Lavage; PFT: Pulmonary Function Testing; FEV1: Forced Expiratory Volume In The First Second; FVC: Forced Vital Capacity; MPM: Malignant Pleural Mesothelioma; CI: Confidence Interval; CBD: Chronic Beryllium Disease; MCH: Major Histocompatibility Complex; BeLPT: Beryllium Lymphocyte Proliferation Test; ATS: American Thoracic Society; JRS: Japanese Respiratory Society; ALAT: Asociacion Latino Americana de Thorax; CHEST: American College Of Chest Physicians; HP: Hypersensitivity Pneumonitis; UIP: Usual Interstitial Pneumonia; VATS: Video-Assisted Thoracic Surgery; HEPA: High Efficiency Particulate Air; OSHA: Occupational Safety and Health Administration; PEL: Permissible Exposure Limits; NIV: Non-Invasive Ventilation

Introduction

Pneumoconiosis is defined as any lung disease caused by inhaling organic or nonorganic airborne dust and fibers [1,2]. The primary types of pneumoconiosis are asbestosis, silicosis, and coal workers' pneumoconiosis (caused by asbestos fibers, silica dust, and coal mine dust, respectively). Other forms of the disease

can be caused by inhaling dust containing aluminum, barium, iron, talc, mica, and mixed-dust pneumoconiosis, among others [3]. Pneumoconiosis is one of the most common occupational diseases in the world. There is evidence of a downward prevalence since 2015 reported by the Global Burden Disease studies. Also, in the United States, pneumoconiosis deaths have decreased from 2,738 deaths in 1999 to 1632 in 2018. All the pneumoconiosis decreases except those attributed to inorganic dust [4,5]. Recently there has been an alarmingly re-emerged pneumoconiosis worldwide and even in the United States, countries with highly developed healthcare systems and standards of workplace safety and efficient reduction of exposure to particles [5-7].

Pneumoconiosis results from the accumulation of fine inhaled particles that enter the lung, escape mucociliary clearance, and deposit in the terminal alveoli. Macrophages take up particles via phagocytosis and release inflammatory cytokines, lysosomal enzymes, and free radicals. After the inflammatory process, the fibrotic process initiates via stimulation of fibrocytes, with an overproduction of fibronectin and collagen, resulting in scar formation [1]. There are also some independent factors that predispose to this disease, such as being male, smoking, age, duration, and quantity of exposure [8]. The diagnosis is made clinically with three major criteria: Exposure to inhalants, characteristic chest X-ray, and the absence of an illness that may be mistaken for pneumoconiosis background of long-term exposure to dust inhalants in addition to radiological evidence of pulmonary fibrosis [1,5,9].

The symptomatology patients present in these diseases are nonspecific and overlap with other pulmonary diseases. Patients may have shortness of breath, decreased exercise tolerance, gradual onset of a nonproductive cough, tachypnea, end-inspiratory crackles, or might be asymptomatic. There is a need to complete the diagnosis and evaluation with a CT scan and pulmonary function test. They may show a typical restrictive pattern with chronic airflow obstruction [1,5]. The treatment for pneumoconiosis is mainly to avoid further exposure to inhalants, smoke tobacco suspension, pulmonary rehabilitation, and symptomatic treatment. Patients with end-stage disease may be candidates for a lung transplant [5,9,10]. This narrative review aims to provide a comprehensive overview of the most common types of pneumoconiosis to better understand the overall approach to these complex and potentially fatal conditions.

Anthracosis

Anthracosis, also known as black lung disease, is a medical condition characterized by the deposition of coal dust or carbon particles in the lungs [11]. It is commonly observed in individuals exposed to coal dust for prolonged periods, such as coal miners, industrial workers, smokers, and individuals living in areas with high levels of air pollution [11,12]. The deposition of coal dust in the lungs can cause inflammation and scarring, leading to respiratory symptoms such as coughing, shortness of breath, and chest pain. The severity of anthracosis depends on the duration and intensity of exposure to coal dust and individual susceptibility. The prevalence of anthracosis in the general population has been estimated to range from 3.1% to 20% [13]. However, it is difficult to accurately assess the frequency since bronchoscopy is required to diagnose anthracosis and cannot be performed on the general population [3,4].

Anthracosis was previously most prevalent in coal workers, but new reports now mostly report this disease in farmers (40%) and rural dwellers (60%) [11]. The prevalence of anthracosis varies depending on the population studied and the level of exposure to coal dust. In coal miners, the prevalence can range from 1% to 95%, depending on the duration and intensity of exposure [15,16]. In non-coal miners, the prevalence is generally lower but still significant. Anthracosis is more common in men than women, likely due to the higher proportion of men working in coal mining and other high-risk occupations [16]. The disease is also more prevalent in older individuals, as the effects of long-term exposure to coal dust accumulate over time. In a meta-analysis, the mean age of patients affected with anthracosis was 63±3.8 years, which shows that these patients are significantly older than non-anthracosis subjects (52±6.4 years) [17].

Anthracosis typically begins in the respiratory bronchioles. Microscopic examination of lung tissue has shown carbon-like particles in the cytoplasm of macrophages within the bronchial wall and free particles in the mediastinal lymph nodes. Submucosal fibrosis may also be observed in the bronchial wall, although the epithelial lining is usually intact. Lobectomy studies have revealed fibrosis of the bronchi and reactive hyperplasia with anthracotic pigmentation as the primary histopathological findings [16,18,19]. Perforation of anthracotic lymph nodes into the bronchial lumen may lead to anthracosis with retracted mucosa. Bronchial cytology has demonstrated macrophage-containing anthracotic nodules in 71% of patients [19]. Associated pathologies, such as tuberculosis or cancer, have distinct histopathology distinguishing them from anthracosis.

Anthracosis should be included in the differential diagnosis of conditions such as COPD, tuberculosis, lung cancer, mucormycosis, and amyloidosis. The presence of chronic dyspnea and/or cough in an elderly non-smoking individual that worsens in winter suggests anthracosis [18]. Wheezing detected during lung auscultation is a favorable indication of bronchial anthracofibrosis (BAF) [20]. If there is an obstructive lung disease shown in spirometry with high attenuation lymph node or bronchial calcification, especially in patients who also have mass lesions (or atelectasis), it strongly supports the diagnosis of anthracosis [21]. Moreover, smooth bronchial narrowing with enlarged calcified lymph nodes is a helpful indicator for differentiating BAF from lung cancer [12,21,22]. However, bronchoscopy should always be performed to confirm the diagnosis in all cases, and bronchoscopy specimens

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should be evaluated for TB. If radiological findings show a mass lesion, open lung biopsy, transthoracic lung biopsy, or advanced bronchoscopic techniques may be required to exclude TB or malignancy.

Progressive massive fibrosis should also be considered a differential diagnosis, but a history of work-related dust exposure and more diffuse lung involvement can help differentiate it from pneumoconiosis. Anthracosis may lead to complications such as enlarged mediastinal lymph nodes, vocal cord paralysis, or broncho-lithiasis. The occurrence of these complications is sometimes associated with tuberculosis [23]. The prognosis of anthracosis can vary depending on the severity and duration of the disease, as well as other underlying respiratory conditions. Prognosis is generally fair in mild cases where the accumulation of coal dust in the lungs is minimal and there are no other respiratory symptoms or complications [16,17]. With appropriate treatment and avoidance of further exposure to coal dust, individuals with mild anthracosis can expect to have normal lung function and an average life expectancy [16,18]. However, the prognosis may be poorer in more severe cases, where there is significant scarring and inflammation in the lungs.

Advanced anthracosis can lead to COPD, which can cause long-term respiratory impairment and increase the risk of respiratory infections, heart disease, and lung cancer [11,15]. A recent study described the clinical course of 280 BAF subjects. The study showed that subjects who suffered only from BAF or a combination of BAF and malignancy had significantly lower survival rates than subjects with BAF and TB, acute exacerbation of airway disease, or pneumonia. This study's leading causes of death were malignancy, infection, cardiac disease, trauma, acute exacerbation, and hemoptysis [17]. Preventive measures, such as improving workplace ventilation and reducing exposure to coal dust, have effectively reduced the incidence of anthracosis [20,23]. However, the disease remains a significant public health concern in specific populations, and continued efforts are needed to minimize exposure and improve treatment and management of the disease.

Silicosis

Silicosis is a form of pneumoconiosis resulting from inhaling silica dust. Silica is a mineral found in rocks, sand, and soil and is commonly encountered in mining, construction, and sandblasting industries [24]. Inhaling silica dust can cause inflammation and scarring in the lungs, leading to the formation of nodules and fibrous tissue. Over time, this can impair lung function and cause respiratory symptoms such as coughing, shortness of breath, and chest pain [24,25]. Silicosis is a preventable disease, and measures such as adequate ventilation, respiratory protection, and limiting exposure to silica can help prevent its development [25]. An estimated 23 million workers in China, 11.5 million in India, 3.2 million in Europe, and 2 million in Brazil are at risk of silica exposure, leading to silicosis's potential development [26]. In the United States alone, it is estimated that 2.3 million workers are exposed, with a majority of the silicosis cases occurring in the manufacturing and construction industries [26,27].

Job activities with high silica exposure include cutting, sawing, grinding, drilling, and crushing various materials such as stone, rock, concrete, brick, block, and mortar and abrasive blasting with sand [25,27]. Interestingly, even young adults who work in jobs such as health care and social assistance have been found to have developed silicosis, despite these industries being less commonly associated with silica exposure [25,28]. Although silicosis has traditionally been considered a disease that develops over a long time (usually over 15-20 years), recent cases of silicosis among denim sandblasters and engineered stone workers have shown that it can occur much more quickly, presenting with inflammation and other systemic symptoms, and even leading to increased mortality among younger workers [28]. Therefore, silicosis can be categorized as acute, accelerated, and chronic (simple and complicated). However, the Centers for Disease Control and Prevention (CDC) now use newer definitions that categorize silicosis as either acute (silicoproteinosis) or nodular (including accelerated and chronic forms of silicosis) [29].

Silicoproteinosis is a clinical presentation of acute silicosis that resembles primary pulmonary alveolar proteinosis [30]. This type of silicosis typically occurs within a few weeks to years after exposure to high silica concentrations. It may be a response to lung injury from inorganic particulate exposure. Acute silicosis cases have been reported in various occupations, including tombstone sandblasters, brick masons, and denim sandblasters [28,30]. Patients with acute silicosis commonly report respiratory and systemic symptoms, such as shortness of breath, cough, chest tightness, fevers, headaches, and unintentional weight loss, which can be mistaken for lung infections. Lung function in acute silicosis shows a spirometric reduction of forced vital capacity and forced expiratory volume in 1 second [26,31,32]. Chest imaging demonstrates bilateral patchy centrilobular nodularity, ground glass opacities, and interlobular septal thickening. The prognosis of acute silicosis is poor, and patients may progress to chronic respiratory failure, cor pulmonale, and death. Although treatment with systemic steroids and whole lung lavage have been reported, their effectiveness in reducing morbidity or mortality has not been systematically evaluated [32].

The onset of cough and difficulty breathing due to chronic silicosis usually occurs between 10 and 25 years after exposure. Chronic silicosis is divided into two categories based on chest imaging results: simple and complicated [31,33]. In simple silicosis, bilateral reticulonodular opacities are present in the upper lobes, with occasional lower lobe linear interstitial opacities, along with possible pleural thickening or effusion. Complicated silicosis, also known as progressive massive fibrosis, is diagnosed with large opacities seen on chest radiographs or conglomerate opacities on chest HRCT scans [33]. Lung function varies depending on the stage of the disease and may show mixed obstructive and

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restrictive ventilatory abnormalities, with restrictive defects in advanced cases. The diffusion capacity of carbon monoxide (DLCO) may be normal in mild diseases but typically worsens over time, especially in smokers [33,34]. Emphysema may be present and can correlate with reduced lung function parameters. Biopsy and autopsy specimens show silicotic nodules, silica particles, and interstitial fibrosis [35].

Simple silicosis can progress to complicated silicosis, which has a poor prognosis due to worsening lung function and an increased risk of tuberculosis or nontuberculous mycobacteria infection. Treatment is supportive, with supplemental oxygen for hypoxia, prompt treatment of infections, recommended vaccinations, and pulmonary rehabilitation [29,31,35]. The primary differentiation between accelerated and acute or chronic silicosis is determined by the time it takes to develop, with accelerated disease manifesting within 5 to 10 years of exposure [35,36]. Diagnostic assessments based on lung histopathology and chest imaging are significant because they exhibit features of silicoproteinosis, a defining feature of acute silicosis and silica nodules, birefringent silica particles, and interstitial fibrosis that are characteristic of chronic silicotic lung disease [28,32]. In addition, disease progression is faster compared to chronic silicosis.

In summary, chronic silicosis is the most common form and usually occurs after prolonged exposure to low levels of silica dust. Accelerated silicosis develops after a higher level of exposure to silica over a shorter period of time, while acute silicosis is a rare but severe form that can develop after extremely high levels of exposure to silica over a period of weeks or months [36]. The prognosis of silicosis varies depending on the severity of the disease and the length of exposure to silica. In general, the disease progresses slowly and may take years to decades to manifest symptoms [35,36]. There is no cure for silicosis, so treatment is mainly supportive, focusing on managing symptoms and preventing complications. Preventive measures such as reducing exposure to silica and early detection through regular health surveillance are crucial in improving the prognosis of the disease [36].

Asbestosis

Asbestosis is an interstitial lung disease caused by the inhalation of asbestos fibers. Asbestos fibers have been historically chosen for construction, shipping, mining, and aerospace engineering commercial use because of their high electrical and thermal resistance and low cost. Asbestos fibers are mineral silicates (mainly hydrated magnesium silicates) and are classified into two main categories based on their shape, i.e., serpentine and amphibole [37]. Chrysolite, being more flexible, curvy, and soluble, settles in the upper part of the respiratory tract. The mucociliary function is more prominent in the upper respiratory tract, so chrysotile fibers are easily removed. Amphiboles (crocidolite, amosite, tremolite, and anthophyllite) are straight, stiff, more brittle fibers. They are more toxic than serpentine fibers, which are less soluble and straight. They usually align with the airstream and reach deeper into the lungs and the interstitium by penetrating the epithelium [38].

The World Health Organization (WHO) estimates that 125 million people worldwide are exposed to asbestos in the workplace. Over 100,000 people die yearly from asbestos-related lung cancer, mesothelioma, and asbestosis [38]. A substantial amount of asbestos remains in buildings and eventually will be removed, either during remediation or renovations, or demolition. It has been estimated that approximately 1.3 million construction and general industry workers are potentially exposed to asbestos during maintenance activities or remediation of buildings containing asbestos [39]. Since the early 1940s, millions of American workers have been exposed to asbestos. Health hazards from asbestos fibers have been recognized in workers exposed in the shipbuilding trades, asbestos mining and milling, manufacturing of asbestos textiles and other asbestos products, insulation work in the construction and building trades, and various other trades. Demolition workers, drywall removers, asbestos removal workers, firefighters, and automobile workers also may be exposed to asbestos fibers.

Studies evaluating the cancer risk experienced by automobile mechanics exposed to asbestos through brake repair are limited, but the evidence suggests there is no safe level of asbestos exposure [40,41]. Asbestosis can be challenging to diagnose due to clinical similarity to other types of DILDs. The onset of the symptoms can vary between 10 to 40 years after initial exposure. It usually starts with progressively worsening dyspnea and persistent dry cough. Subsequently, patients develop chest discomfort, digital clubbing, and symptoms of cor pulmonale. Auscultation might reveal bibasilar rales at lower lateral and basal areas [42]. Localized chest pain and its radiation to the shoulder denotes pleural involvement, the hallmark of asbestosis [43]. Productive cough and wheezing are unusual and often associated with smoking. Constitutional symptoms are rare, and weight loss and hemoptysis raise suspicion of lung tumors [43]. The diagnosis of asbestosis is mainly clinical.

History of exposure is the single most crucial factor. However, various tests might be needed to accurately diagnose chest X-rays, chest high-resolution computed tomography (HRCT), pulmonary function tests, bronchoscopy with biopsy, bronchoalveolar lavage, and lab analysis [44]. Chest X-ray is the least expensive and invasive method. It is characteristic of progressive restrictive pulmonary disease with interstitial fibrosis. It shows small bilateral parenchymal opacities with a multinodular or reticular pattern that blurs the diaphragm and heart border creating the "shaggy heart" sign [45]. The lungs will be affected bilaterally in advanced asbestosis, giving them a honeycomb appearance [46]. However, X-ray has low sensitivity, especially in the early stages of

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the disease. Therefore, an HRCT scan is the preferred option, as it provides greater detail and has increased sensitivity for the early stages of asbestosis [40,47]. Tomography findings of early stages consist of ground-glass opacities with peri bronchial, intralobular, and interlobular septal fibrosis, subpleural dot-like structures, and subpleural lines of varying lengths parallel to the pleura. Coarse parenchymal bands are often contiguous with the pleura [42,48]. HRCT of asbestosis may mimic idiopathic pulmonary fibrosis; however, it can be distinguished by the star point of fibrosis. In asbestosis, fibrosis begins centrally and spreads peripherally in a centrifugal pattern.

In contrast, idiopathic pulmonary fibrosis starts peripherally at the bases and advances centrally and upwards in a centripetal pattern [44]. In a study by Morgan A. on deposition and clearance of asbestos in rats, autoradiographs of lung sections showed that alveolar deposition was initially uniform but changed to subpleural foci of fibrosis in a couple of months, these foci served as centers for the development of nodular fibrosis, thus sustaining the theory of centrifugal progression of the disease [49,50]. In the advanced stages of the disease, there are two patterns of parenchymal changes: atelectatic induration fibrosis and honeycombing [43,51]. The first pattern has a rapid evolution and is associated with higher exposure. It is marked by collapse and collagenous fibrosis filling the alveolar lumens [5]. Honeycombing is less frequent, usually between 17-32% on HRCT [42,48]. Biopsy is undoubtedly the most specific diagnostic tool. It has proven the diagnostic of asbestosis in exposed patients despite normal HRCT [52]. A lung biopsy can be performed via endobronchial, transbronchial, and video-assisted thoracoscopic (VAT) approaches.

The VAT-guided biopsy is minimally invasive, with fewer complications, high sensitivity, and specificity. However, it has a higher cost and expertise requirement [53]. Transbronchial biopsy has an increased risk of bleeding and associated complication, therefore is not a preferred choice. The endobronchial biopsy has a high diagnostic value and low risk of complications, thus making it the preferred choice for most centers [53]. The macroscopic view shows peri bronchial fibrosis. The microscopy reveals distinct asbestos bodies coated with iron-containing proteinaceous material and diffuse pulmonary interstitial fibrosis. The patchy characteristic of interstitial fibrosis differentiates asbestosis from idiopathic pulmonary fibrosis IPF [44]. Bronchoalveolar lavage (BAL) has a limited role due to the high incidence of false-negative results due to the short half-life of chrysotile fibers [54]. Additionally, it may show false-positive results in patients with previous asbestos exposure, despite normal imagistic tests [55].

Therefore, BAL is usually reserved for patients with abnormal HRCT, where it is not sufficiently diagnostic, and differential diagnosis with malignancy, infection, or hypersensitivity pneumonitis needs to be done [18]. An essential role in the initial evaluation of dyspnea is attributed to pulmonary function testing (PFT). It should include spirometry, lung volumes, and carbon monoxide (DLCO) diffusing capacity. PFTs in asbestosis show reduced lung volumes, particularly the vital and total lung capacity, decreased pulmonary compliance, absence of airflow obstruction by spirometry, and decreased DLCO [46,56]. FEV1/FVC ratio is either normal or increased. The presence of airway obstruction denotes simultaneous exposure to cigarette smoke [57]. The earliest changes in asbestos-exposed patients are reductions in the DLCO and the presence of exertional hypoxemia, which subsequently progresses to hypoxemia at rest [58]. Lab analysis is nonspecific and usually shows elevated C-reactive protein, erythrocyte sedimentation rate, rheumatoid factor, and antinuclear antibodies.

The prognosis for asbestosis varies significantly by individual. It usually is determined by the amount of asbestos exposure, the duration of that exposure, and lifestyle factors such as smoking, age, and general health [59]. Cigarette smoking is associated with the acceleration of pulmonary fibrosis progression [60]. There are two main complications of asbestosis: respiratory failure and malignancy. Asbestosis progresses slowly, usually leading to respiratory failure [61]. The odds of developing respiratory failure increase with the abovementioned factors, with symptoms of dyspnea and imagistic changes such as diffuse pleural thickening and honeycombing on HRCT. PFTs are pivotal in predicting the risk and the survival rate [62]. Finally, the most common malignancy associated with asbestosis is malignant mesothelioma-a rare neoplasm that develops from the mesothelial surface of the pleural cavity, peritoneal cavity, tunica vaginalis, or pericardium [63].

The most common type is malignant pleural mesothelioma (MPM). Patients with MPM usually present with chest pain, dyspnea, cough, and night sweats. Additionally, studies showed that asbestos is involved in developing bronchogenic carcinoma and lung cancer [64,65]. Unequivocally, the incidence of lung cancer is higher in asbestos-exposed smokers than exposed nonsmokers. A recent meta-analysis on the development of lung cancer in asbestos-exposed population showed odds ratios of 1.70 (95% CI 1.31-2.21) among asbestos-exposed nonsmokers, 5.65 (95% CI 3.38-9.42) among smokers without asbestos exposure, and 8.70 (95% CI 5.8-13.10) among asbestos-exposed smokers [64]. Asbestos exposure has also been attributed to developing laryngeal, oropharyngeal, esophageal, kidney, and biliary system malignancies [66].

Berylliosis

Berylliosis, or chronic beryllium disease (CBD), is a granulomatous condition resulting from exposure to beryllium. Beryllium exposure is the main contributing factor, and industries such as metal machine shops, electronics, defense, and beryllium extraction are among those with high levels of exposure [67]. Other sectors, such as ceramics, automotive, aerospace, jewelry

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making, dental/alloy appliances, and computers, may also pose a risk. In addition, specific individuals may have a genetic predisposition to developing severe CBD. Approximately 2-5% of beryllium-exposed workers develop CBD [68]. Stopping exposure to beryllium does not necessarily halt the progression to CBD. However, unless individuals live in close proximity to industrial sites, it is unlikely for the general population to develop acute or CBD, as ambient air levels of beryllium are typically very low (<0.05ng/m^3) [69].

Exposure to beryllium can occur through inhalation of beryllium fumes or dust, as well as skin exposure [68,69]. While the organic forms of beryllium are quickly excreted from the body, the insoluble inorganic particles can remain present for several years. Beryllium exposure can trigger a cell-mediated immune response, where T-cells become sensitized to beryllium. With each subsequent exposure, this response begins the accumulation of macrophages and CD4+ helper T-lymphocytes in the lungs [70]. As the reaction progresses, macrophages, CD+4 T-lymphocytes, and plasma cells group together to form noncaseating granulomas, which can ultimately lead to lung fibrosis. Current literature indicates a genetic component to beryllium sensitivity, with beryllium-exposed workers who have a mutation at the HLA-DPB1 Glu69 position showing a greater prevalence of beryllium sensitization and CBD [68,70,71]. The HLA-DPB1 gene plays a crucial role in the function of MCH class II molecules on antigen-presenting cells [71]. Beryllium and beryllium compounds are classified as category 1 carcinogens, known to cause cancer in animals and humans [70,72].

The clinical presentation and symptoms of chronic beryllium disease are not specific. There can be a delay of three months to 40 years between exposure to beryllium and the onset of symptoms [72]. Common symptoms include fever, night sweats, weight loss, dry cough, and fatigue [69]. Noncaseating inflammatory granulomas can develop due to continued exposure to beryllium, also observed in other chronic diseases such as sarcoidosis and tuberculosis [73,74]. CBD can lead to restrictive lung disease resulting in decreased diffusion capacity, and in rare cases, granulomas can form in other organs like the liver. During the physical examination, lymphadenopathy, crackles, rash, and hepatosplenomegaly may be observed [74].

Berylliosis diagnosis involves establishing beryllium sensitivity, which is done by performing a beryllium lymphocyte proliferation test (BeLPT) using peripheral blood or bronchoalveolar lavage (BAL) fluid [75]. Lymphocytes are cultured with beryllium sulfate, and a high number of abnormal cells are counted [74,75]. Those with two abnormal BeLPTs with peripheral blood or one abnormal and one borderline result are considered beryllium sensitized. Also, those with one abnormal BeLPT with fluid from a bronchial alveolar lavage are considered sensitized. Bronchoscopy with BAL is performed for patients with a positive BeLPT to obtain cell counts [76]. A tissue biopsy is obtained from bronchoscopy to fulfill the final criterion for CBD diagnosis: the presence of granulomatous inflammation on lung biopsy [75,76]. The imaging findings of berylliosis are also not specific. In the early stages of the disease, radiography findings are typically normal [76].

However, interstitial fibrosis, pleural irregularities, hilar lymphadenopathy, and ground-glass opacities have been reported in the later stages [6,7,10]. CT scans commonly reveal parenchymal nodules in the early stages. Ground-glass opacities were more frequently observed in berylliosis than in sarcoidosis [67,69,70]. Hilar lymphadenopathy, interstitial pulmonary fibrosis, and pleural thickening are typical later-stage findings. Other tests that may be used to diagnose berylliosis include arterial blood gas, pulmonary function tests, spirometry, and DLCO levels [70,75]. The differential diagnosis for berylliosis includes sarcoidosis, idiopathic pulmonary fibrosis, hypersensitivity pneumonitis, asthma, and other granulomatous lung diseases like histoplasmosis, tuberculosis, and silicosis [67,69]. It is estimated that approximately 6% of patients diagnosed with sarcoidosis may have CBD [68].

The prognosis of berylliosis can vary depending on the severity of the disease and the promptness of diagnosis and treatment. Sometimes, the condition may resolve independently without causing significant long-term damage to the lungs or other organs [69,73,77]. However, in other cases, berylliosis can progress and lead to chronic lung disease and respiratory failure (mortality rates for CBD range from 5% to 38%) [67,72]. Patients sensitized to beryllium, but not CBD, do not need to be treated but should receive regular check-ups. These patients have a higher risk of developing CBD compared to non-sensitized individuals [71,76]. The prognosis is generally better for individuals who receive early and appropriate treatment, including avoiding further exposure to beryllium and using immunosuppressive drugs to manage the inflammatory response [77]. However, even with treatment, some individuals may experience ongoing lung impairment and reduced quality of life. Therefore, close monitoring and management of symptoms are essential for individuals with berylliosis to minimize complications and optimize long-term outcomes.

Hyper-Sensitivity Pneumonitis

Hypersensitivity pneumonitis is a complex interstitial lung disease characterized by an exaggerated response to an inhaled antigen in susceptible individuals [78]. A joint task force including the American Thoracic Society/Japanese Respiratory Society/ Asociacion Latino Americana de Thorax (ATS/JRS/ALAT) defines the entity as an inflammatory and/or fibrotic disease affecting the lung parenchyma and small airways [79]. It typically results from an immune-mediated reaction provoked by an overt or occult inhaled antigen in susceptible individuals [79,80]. On a molecular level, the Presence of major histocompatibility II is associated with hypersensitivity pneumonitis, and the expression of MUC5B allele

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rs35705950 is linked to a greater extent of radiographic fibrosis [78]. Traditionally hypersensitivity pneumonitis was classified as acute, subacute, and chronic [78-80].

The recent diagnostic guideline published by the American college of chest physicians (CHEST) and ATS/JSR/ALAT classifies hypersensitivity pneumonitis (HP) as fibrotic and nonfibrotic based on the presence or absence of fibrosis on a high-Resolution computed tomography scan (HRCT) [79-81]. The new classification guideline prioritizes fibrosis and antigen classification as the main prognostic factor [81]. It further explains that chronic hypersensitivity pneumonitis does not always follow acute disease, and not all patients with chronic hypersensitivity pneumonitis present with fibrosis on HRCT [79,81]. Non Fibrotic hypersensitivity pneumonitis is characterized by pure inflammation, while the fibrotic subset could be a mixture of fibrosis and inflammation or purely fibrotic [78,79,81]. In the absence of an unidentified antigen exposure with typical features of hypersensitivity, pneumonitis is described as cryptogenic HP or HP of undetermined cause [78,79].

An insurance claim-based retrospective cohort analysis following a ten-year study period identified the mean age of disease presentation as 52 years, with 58% in women. The 1-year prevalence rates for hypersensitivity pneumonitis ranged from 1.67 to 2.71 per 100,000 persons, and 1-year cumulative incidence rates ranged from 1.28 to 1.94 per 100,000 persons [79,82,83]. The prevalence increased with age, ranging from 0.95 per 100,000 among 0- to 9-year-olds to 11.2 per 100,000 among those 65 years and older [83]. Higher rates are observed among specific at-risk groups, up to 4.95 per 100,000 among bird breeders [82]. The estimated prevalence of farmer's lung ranges from 1% to 19% of exposed farmers, the prevalence of pigeon breeder's lung is from 6% to 20% of exposed individuals, and the prevalence of budgerigar's lung is from 1% to 8% of exposed individuals [84]. Older age, male sex, and fibrosis were associated with higher mortality rates [83].

Insidious HP has been reported among a few exposed groups, such as lifeguards, automobile workers exposed to polyurethane, and office workers exposed to contaminated humidifiers [79]. HP risk factors include exposure to environmental or occupational inducers [85]. Over 50 recognized inciting agents are summarized as contaminating microorganisms, animal proteins, and inorganic chemicals that have been etiologically linked to HP [79,80]. Viral infections may increase hypersensitivity to environmental antigens and stimulate the release of inflammatory cytokines. At the same time, tobacco smoking has an impact on immune reactivity and drives the pathogenic pathway toward fibrotic disease [78]. Farming and grain processing, indoor air and water pollution with birds, and dust from other animals are associated with HP [8]. In Japan, HP is associated with bacteria and yeast in homes during the hot and humid summer [80].

Clinical presentation varies, but common symptoms include shortness of breath, cough, mid-expiratory squeaks, chills, low-grade fever, weight loss, wheezing, body ache, and malaise [79,80,82,86]. Acute onset of symptoms is associated with a shorter but higher exposure to substances that cause HP, while chronic symptoms are associated with repeated low exposure to inciting agents [80]. Patients with a nonfibrotic subtype tend to have an acute onset of symptoms, and an identifiable antigen is mostly present, while the insidious presentation is associated with fibrotic HP [79,80]. The onset of symptoms can be sudden within hours to days to weeks, or insidiously, over months to years [79,82]. The symptoms experienced may be episodic or relapsing in association with antigen exposure [79,82,86].

Diagnosing hypersensitivity pneumonitis involves a multidisciplinary approach and should be part of the differential list for newly diagnosed interstitial lung disease [79,81]. CHEST and ATS/JTS/ALAT have published expert diagnostic guidelines to aid the diagnosis of the disease [79,81]. The diagnostic guideline published by ATS/JRS/ALAT emphasizes the importance of exposure identification, imaging pattern, and bronchoalveolar lavage (BAL) lymphocytosis/ histopathological finding [79]. According to ATS/JRS/ALAT, HP can be diagnosed with high confidence in patients in whom an exposure has been identified and who have a typical HP pattern on HRCT and have BAL lymphocytosis; such patients do not require additional testing [79]. CHEST guidelines and expert panel report state that HP can be diagnosed if there is a presence of HRCT findings characteristic of HP with clinical findings to support the diagnosis [78,81]. CHEST also recommends against not using BAL fluid analysis to confirm the diagnosis of HP in patients with compelling exposure history within the appropriate clinical context and a chest HRCT pattern typical for HP [81]. Both expert guidelines identify the use of HRCT to establish the presence of fibrosis [78,79,81]. The search for exposure can be done using a basic questionnaire or specific IGg testing [78]. Lung biopsy is done in patients suspected of HP. However, for whom a confident diagnosis cannot be made based on the available data, obtaining lung tissue is recommended if warranted based on the risk: benefit for the individual patient [78,81].

The prognosis of hypersensitivity pneumonitis is unpredictable, with clinical courses that may range from clinical improvement to progressive decline and death from respiratory failure [78,79]. Patients with fibrosis have a worse prognosis, and those with usual interstitial pneumonia (UIP)-like patterns have reduced survival [79,80]. The extent of fibrosis and the presence of honeycombing and bronchiectasis is associated with mortality [78]. Patients with non-fibrotic disease respond well to treatment, and full recovery is possible if the inciting factor is avoided [79,80]. The inability to remove inciting factor, failure to identify inciting factor, and persistent exposure are associated with lower prognosis [79,86]. Cigarette smoking, lack of BAL lymphocytosis,

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lower baseline vital capacity, and male sex are associated with poor prognosis [78-80].

Siderosis

Pulmonary siderosis is a rare occupational disease that occurs due to chronic inhalation of iron compounds. Zenker first applied the term "Siderosis" in 1866 for the description of a pathological condition of the lung caused by extended continued inhalation of the dust of iron or iron oxide. The fibrotic reactions in the lung due to iron and steel fumes are termed siderofibrosis [87,88]. The occupations that lead to the development of pulmonary siderosis include mining, welding, steel manufacturing, iron oxide manufacturing, grinding wheel manufacturing, and silver jewelry manufacturing. Unfortunately, most affected people are neither aware of occupational diseases associated with their occupation nor of using any protective gear [87,89,90]. Despite striking radiological and histopathological features, pulmonary siderosis has traditionally been classified as a 'benign pneumoconiosis' because of the absence of associated symptoms, functional impairment, pulmonary fibrosis, or predisposition to tuberculosis [87,88,91].

Doig and McLaughlin first described 'welders' siderosis' in 1936 when they carried out a prospective study examining the clinical and chest radiological characteristics of 16 electric arc welders [89]. All but one of these original subjects were followed for 9 years: four of these demonstrated progressive radiographic reticular changes, nine showed no radiographic changes, and in two men (both of whom had spent significantly less time welding), there was evidence of at least partial resolution of the initial radiographic opacities [92]. All subjects, however, remained in good health, concluding that siderosis (in its pure form) was not associated with respiratory symptoms or functional impairment. This view was supported by subsequent pathological investigations of the lungs of subjects occupationally exposed to iron oxide fumes, which did not demonstrate any evidence of pulmonary fibrosis [92]. In the Meyer E. et al. study of four patients exposed to iron oxide fumes, the pathological examination did not show evidence of pulmonary fibrosis [93]. McLaughlin et al. described that exposure to iron oxide produces X-ray changes but no lung fibrosis after 40 years of exposure [87].

Some authors, however, have reported respiratory difficulties or histological findings of emphysema or pulmonary parenchymal fibrosis in welder's lung disease. Buckell et al. studied siderosis in iron turners and grinders and suggested lung fibrosis after >20 years of exposure [94]. Funahashi et al. in a study of ten symptomatic welders, the pathological examination showed that some degree of parenchymal fibrosis was present. In 50%, this fibrosis was considered moderate to pronounced [95]. Symptomatic pulmonary siderosis with interstitial fibrosis, even with progressive massive fibrosis, consolidation, and usual interstitial pneumonia pattern, has been reported recently [87,95-97]. However, it takes years of exposure for a patient to become symptomatic, and rapid development of symptomatic disease within a year after exposure has been reported. In addition, high-intensity, brief exposure to iron dust can also result in future symptoms [98]. It is important to realize that exposure to silica or asbestos is not uncommon in many jobs involving iron exposure, thus giving rise to mixed dust fibrosis or to asbestosis, which has associated morbidity and complications [99].

Pulmonary siderosis is diagnosed by identifying a patient's exposure history and chest radiographic findings and confirming iron accumulation by iron dyeing the bronchoalveolar lavage fluid or biopsy specimen. Recently, because of the development of radiological technology, radiological findings have played an important role in the differential diagnosis of the disease [98]. Typical radiological findings of pulmonary siderosis are poorly-defined centrilobular micronodules, branching linear structures, and ground-glass attenuation. However, atypical radiologic findings rarely appear, which may be misdiagnosed as other diseases [100]. In addition, with the cessation of exposure, the radiographic opacities may gradually disappear [99]. At the time of welding, the aspirated iron oxide is stored in alveolar macrophages and then transferred to the lung parenchyma. Subsequently, macrophages are distributed through the blood and peri bronchial lymphatic vessels, forming small nodules. Because of these pathophysiological characteristics, typical CT findings of pulmonary siderosis may appear [98].

In 1995, Akira reported that the most common CT findings, present in 15 of 21 arc welders studied, were ill-defined micronodules diffusely distributed in the lung [100]. Emphysema was observed in 7 cases and was the predominant CT finding in 3 cases. In addition, a honeycomb pattern was found in 3 cases, presenting the predominant CT finding in all 3 cases. The CT appearance of this honeycomb pattern resembled that observed in usual interstitial pneumonia [101]. These chest CT findings are further supported by Han et al. who reported that the most frequent thin-section CT findings in patients with welder's lung disease were poorly defined centrilobular micronodules (55.6%), branching linear structure (33.3%) and ground-glass attenuation (11.1%) without zonal predominance [100]. All of these findings represent macrophage accumulation in alveolar space. However, in addition to these typical imaging findings, pulmonary siderosis can appear in unusual forms. Other chest CT findings in welder's lungs included localized, stable tuberculous lesions, bronchial dilatation or wall thickening, localized emphysema, non-calcified mediastinal lymphadenopathy, subsegmental atelectasis, or tracheal dilatation [100]. Another study of CT features of uncommon pneumoconiosis showed centrilobular ill-defined micronodules' most common radiological finding in 15/21 cases, while only 3 cases showed honeycombing [101].

In 2004, since the report of a case of pulmonary siderosis with typical imaging findings in Korea, Kinosita et al. reported

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a case of pulmonary siderosis suspected of lung cancer. In that case, pulmonary siderosis showed a 3-cm-sized pulmonary nodule in radiological findings but was confirmed by histological examination. In 2011, there was a pulmonary siderosis with atypical imaging findings in Korea. In that case, chest CT showed a 1.3 × 1.5cm sized mass in the left upper lobe and multiple ill-defined irregular nodules in both lung fields. Thus, similarly, that pathologic lesion was misdiagnosed as metastatic lung cancer. As described above, all the pulmonary siderosis cases with atypical imaging findings included mass-like lesions or large nodules [102]. The histopathological diagnosis requires a lung biopsy. The standard methods for lung biopsy are transbronchial lung biopsy via bronchoscopy, imaging-guided lung biopsy, or surgical lung biopsy. Akar E et al. in a study of seven cases diagnosed by video-assisted thoracic surgery (VATS), showed 100% diagnostic yield with no complication [90]. Transbronchial lung biopsy also showed good diagnostic yield without any complication in a few reported cases [87,103].

Talcosis

Pulmonary talcosis is a rare but debilitating form of pneumoconiosis that is difficult to diagnose due to its nonspecific signs and symptoms. The causative agent, talc, is a magnesium silicate mineral found in various household products such as ceramics, paper, oral medications, and cosmetics such as baby powder [104]. The disease most commonly affects men in their 40s [105]. Although found in everyday household products, talc is overlooked as a cause of pneumoconiosis. Long-term exposure to talc inhalation is known to cause talcosis, talc-silicosis, and talc-asbestosis in industrial workers. A less common cause is pulmonary injury from talc emboli in intravenous drug users that self-inject talc-containing oral tablets. Talcosis caused by cosmetic face powder is even rarer [106]. The patient's presentation can range from asymptomatic to fulminant disease. Initial diagnosis is difficult due to nonspecific complaints, including progressive exertional dyspnea and cough. Misdiagnosis or improper management can cause disease progression to chronic respiratory failure, emphysema, pulmonary arterial hypertension, and cor pulmonale [105].

A history highlighting occupational exposure or intravenous drug abuse in the past provides an initial clue to the diagnosis. Chest X-ray may show a pulmonary fibrosis-type picture. Computerized tomography (CT) scan of the chest on initial evaluation may reveal nonspecific diffuse centrilobular ground-glass nodules bilaterally. Characteristic findings of pulmonary talcosis on high-resolution computed tomography (HRCT) include small centrilobular nodules associated with heterogeneous conglomerate masses containing high-density amorphous areas. Histopathological tests such as bronchoalveolar lavage and transbronchial biopsies are needed for tissue diagnosis. The presence of negatively birefringent, needle-shaped particles of talc seen within the giant cells and in the areas of pulmonary fibrosis under polarized light confirms the diagnosis [104,105]. The natural history of pulmonary talcosis is slowly progressive similar to other forms of pneumoconiosis, even after exposure has ceased [106].

Treatment Options for Pneumoconiosis

Since most forms of pneumoconiosis are irreversible, management must be directed towards preventive measures to stop these diseases' development and subsequent complications. If patients develop these conditions, healthcare workers can direct their efforts toward preventing the progression of the disease and symptomatic therapy to alleviate the effects of these diseases on the body [107]. Management often depends on many factors- the type of pneumoconiosis, total cumulative lifetime exposure to irritants, radiographic changes, smoking status, and life expectancy. The primary avenue of preventing the development of pneumoconiosis involves lifestyle modifications. Some maladaptive lifestyle habits that can cause the development of pneumoconiosis are- elimination of exposure to the causative agent(s) (talc/silica/coal/grains/beryllium etc.), cessation of smoking, preventive vaccination against respiratory pathogens, and education of the general public at large, and at-risk population about this class of conditions, and how to manage/avoid them [108]. Exposure prevention involves many measures - clear demarcation of residential settlements and industries, use of workplace safety equipment such as respirators, masks, HEPA, or air particulate filters, and vaccination against respiratory pathogens [109].

Occupational measures such as adherence to Occupational Safety and Health Administration (OSHA) permissible exposure limits (PEL) for particulate matter and toxic pollutants can reduce the long-term harmful effects of certain industrial occupations without disrupting occupational avenues for industry and manufacturing-dependent industries populations. Vaccination against respiratory pathogens can prevent the development of lower respiratory pathologies, creating environments favorable to the development and progression of pneumoconiosis and avoiding the worsening of pre-existing interstitial lung disease. Conditions such as Hypersensitivity Pneumonitis can be managed by avoiding known triggers, such as birds and animals, or using protective equipment and countermeasures, such as air purifiers for use by farmers and agriculturists in the event that they can't avoid the triggering agent due to occupational reasons [110].

Medical management is primarily centered around symptomatic management and improvement of functional status- to alleviate the impact of pneumoconiosis and DILD on activities of daily living and quality of life. Systemic glucocorticoid therapy in oral formulations such as prednisolone has been found to improve radiographic and symptomatic status by arresting and possibly reducing inflammation and scarring associated with these conditions [107]. Bronchodilator medications in the form

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of Theophylline and sympathomimetic drugs can also improve functional ability by enhancing airflow- given that the primary deleterious effect of pneumoconiosis is impaired oxygenation and respiration. Supportive therapy in the form of supplemental low-flow oxygen using nasal prongs/NIV masks/home-based or portable oxygen cylinders can become necessary in cases with significant worsening of FEV1 and DLCO to maintain a cardiorespiratory status compatible with life. In addition, resting hypoxemia or exertion-induced desaturation are pertinent reasons to start supplemental oxygen. Additional management techniques, such as chest physiotherapy, can be utilized to avoid over-dependence on medications and drugs. These involve humidification therapy, education about postural drainage, and even manual techniques in vibrations and compression to alleviate breathing difficulties.

In addition, forced expiratory courses such as huffing and coughing can be taught to better facilitate the drainage of secretions and improve alveolar ventilation. In advanced-stage diseases among younger patients and patients with good surgical prognosis, lung transplantation can improve a patient's quality of life. Lung transplantation is reported to be associated with significant reductions in mortality rates among patients with reasonably advanced staged disease [108]. Novel treatments for pneumoconiosis include cytokine inhibitor drugs that target pro-inflammatory cytokines, antioxidant medications, inhalational polymers, and intra-tracheal mononuclear cell implantation [109,110]. However, these modalities require rigorous testing and standardization before being deployed as effective treatments on a large scale. Lastly, conditions such as asbestosis can be associated with malignancy (i.e., carcinoma of the lung mass itself or pleural malignancies like mesothelioma). Treatment of such complications depends on the stage and extent of the disease [111-113]. However, it can vary from drug-based chemotherapy and adjuvant radiotherapy to surgical intervention or combination [107].

Conclusion

Pneumoconiosis involves a broad spectrum of lung diseases caused by inhaling mineral dust, often due to certain occupations. The primary characteristics are chronic inflammation of the lungs and gradual scarring, which may eventually result in respiratory or heart failure and death. Pneumoconiosis is a global health concern due to its widespread occurrence. The high incidence and mortality rates are largely attributed to inadequate occupational protection and the absence of early diagnostic techniques and effective treatments. In this narrative review, we have provided an overview of the current clinical status and future research directions for pneumoconiosis. The lack of effective dust prevention measures, early diagnostic methods, and disease-specific treatments remain significant challenges in the management of pneumoconiosis.

This lung disease continues to be a global threat, underscoring the critical need for extensive research into its underlying mechanisms. It should be noted that further studies are still needed to better understand, diagnose, and treat this condition. These studies presumably could be focused on investigating new treatment options, developing more accurate diagnostic tests, and identifying new risk factors for the development of pneumoconiosis. Ultimately, these efforts could lead to better outcomes for patients with this condition and help prevent future pneumoconiosis cases.

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