Literature Review

Current Lung Asbestosis Approach for Diagnosis, Not Just Histopathology: A Literature Review

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Abstract

Asbestosis is characterized by diffuse interstitial fibrosis in the lungs, which is caused by breathing asbestos fibers from the crystalline or amphibole groups. The diagnosis of asbestosis, a form of pneumoconiosis, is one of the seven steps in identifying an occupational lung disease. Because there is no known cure for this condition, early detection, prevention, and education of workers and anybody in their proximity who has a risk of asbestos fiber exposure is critical. Clinical symptoms of asbestosis include weight loss, decreased appetite, and dyspnea during exertion. Clubbing fingers, cyanosis, and tachypnea are all symptoms of severe asbestosis. Bronchoalveolar lavage (BAL), histology, CT scans, HRCT, and respirometry can all help with the diagnosis. The "shaggy heart border sign" on a chest X-ray, along with the asbestos body observed in the BAL, is a reliable indicator of asbestosis. Because of the dismal prognosis and lifelong consequences, prevention is essential.

Keywords: asbestosis, asbestos body, diagnosis

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INTRODUCTION

Asbestos is a natural mineral used as an insulation material and as a fire retardant, which was used in many homes that were built before 1977 (such as pipes, ceilings, and floor tiles).[1] Due to health concerns and various fibrotic lung diseases related to asbestosis, asbestos had been prohibited by many nations.[2] However, despite prohibition, data from 1990 to 2017 shows that the incidence of asbestosis is constantly rising.[3,4]

Workers who are exposed to asbestos fibers at high intensities are more likely to get asbestosis. In 2017, 3400 asbestos-related fatalities were reported worldwide. The incidence of asbestosis was 15.7% of pneumoconiosis overall in 2017.[4] Between 1990 and 2017, the percentage of people with asbestosis climbed by 116.6% globally, including notable increases in Western Europe (16.8%), the Americas (20%), and Australia (31.7%).[4]

According to estimates from the World Health Organization (WHO), 125 million persons globally were exposed to asbestos particles at work in 2018.[5] The International Labor Organization (ILO) also supports that data by revealing the number of deaths linked to asbestos exposure increased in 2018; between 107,000 and 112,000 persons died from lung cancer, mesothelioma, and asbestosis.[6] Among the 160 asbestosis cases in Birmingham in 2017, research revealed that 77 individuals (44%) worked in the industrial sector, particularly in construction.[7]

Although asbestos is no longer widely utilized and is prohibited in some wealthy nations, it is nevertheless used in underdeveloped nations. Currently, China, Brazil, Kazakhstan, and Russia supply roughly 90% of the world's asbestos. China and India utilize around half of the world's asbestos, with Brazil, Indonesia, and Russia coming in second and third, respectively.[8] In 2019, the United Kingdom General Practice Research Database reported that Italy had an average of 1148 cases of asbestosis,

with a hospitalization rate of 25.2 per 100,000 people.[9]

In addition to asbestosis, asbestos exposure may increase the risk of mesothelioma and bronchogenic carcinoma.[10] According to a study by Suraya et al at Persahabatan Hospital Jakarta, individuals who were exposed to asbestos fibers had a twofold increased risk of lung cancer.[11] Pulmonary fibrosis, a condition where asbestos fibers continue to build up, can impair the lung's capacity to exchange gases.

The prolonged interval between asbestos exposure and the onset of clinical symptoms of the disease results in delayed diagnosis and treatment. There is now no known most efficient treatment to eradicate asbestos fibers' damaging effects on the alveoli. Treatments aim to keep the illness from getting worse while also helping to lessen symptoms that have already started.

ETIOLOGY AND PATHOGENESIS

The silica minerals known as asbestos fibers, which belong to the crystalline category, are the main cause of asbestosis. There are two main categories of asbestos fibers: serpentine and amphibole.[12] Chrysotile, or white asbestos, is the sole kind of asbestos that belongs to the serpentine group. On the other hand, the amphibole group includes the minerals tremolite, amosite, actinolite, anthophyllite, and crocidolite, as shown in Figure 1.

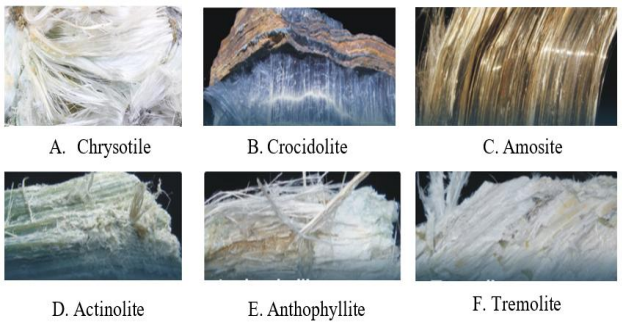

Figure 1. Types of asbestos fibers that are dangerous to the body[5]

The fiber's surface, size, and chemical makeup all have an impact on how cells react, which determines how poisonous and pathogenic the fiber is.[4] Because amosite and crocidolite are larger than chrysotile, they have a greater capacity to induce inflammation, cell proliferation, and the release of oxidants from alveolar macrophages, all of which contribute to their fibrogenicity. More than 1000 asbestos fibers per milligram of lung tissue are thought to be a sign of asbestos fiber exposure.[12]

Patients with asbestosis have concentrations of asbestos fibers in their lung tissue that are greater than 50,000/g and occasionally as high as millions or hundreds of millions.[12] There are three primary ways that people might be exposed to asbestos fibers: directly, indirectly, and via the community.

The first kind is direct exposure, which is typical for those who come into close contact with asbestos fibers, such as those in the mining, shipbuilding, and aviation sectors. The second kind of exposure is indirect exposure, which is prevalent among families of manufacturing workers who engage in trades like painting, masonry, and electrical work. The third kind of asbestos exposure is the most prevalent among the general people, who often visit locations like paint companies that use chemicals, landfills, and road surfaces. Populations with high levels of direct asbestos fiber exposure, such as those in the first exposure group, are most likely to be exposed, but the chance of exposure declines for those in the second and third exposure groups.[13]

Large-sized asbestos fibers are retained in the nose and eliminated via the nasal mucociliary system, marking the beginning of the pathogenesis phase of asbestosis. The size of each asbestos fiber, the level of exposure, and the asbestos fiber's surface all affect how well it penetrates the lung. When asbestos fibers are less than 5 μm in size, they can penetrate the upper airway with 80% penetration and remain in the lower airway, particularly in the respiratory bronchioles, with the remaining 10–20% remaining in the upper airway. Tiny, respirable fibers with diameters less than 0.4 μm and lengths less than 10 μm will gather in the distal alveoli.[13,14]

The mucociliary system will remove asbestos fibers that have accumulated in the respiratory bronchioles. However, if the mucociliary system is unable to remove the asbestos fibers, type 1 alveolar

epithelial cells will carry the fibers into the interstitial tissue, where they will eventually flow to the pleural glands or hilus. A group of cells known as alveolar macrophages, pulmonary epithelium, mesothelium, endothelium, and fibroblasts are particularly vulnerable to the harmful effects of asbestos fibers. Asbestos fibers in the alveolar and peribronchial ducts surrounding the terminal bronchioles are not destroyed by alveolar macrophage buildup, which leads to the first lung lesions.[12,14]

Alveolar macrophages will be activated by asbestos fibers, causing lung tissue damage and drawing neutrophils from the circulation to the injured area. Localized creation of collagenase unique to neutrophils will ensue from this. Because asbestos fibers are toxic, fibroblast activation and an accumulation of alveolar macrophages that are unable to remove asbestos fibers will cause interstitial tissue to thicken. Fibrosis initially appears in the lower lobe pulmonary bronchioles next to the visceral pleura; in more advanced stages, more extensive interstitial fibrosis will manifest. The predominant pathogenetic result of asbestosis is believed to be interstitial fibrosis, resulting from the accumulation and dispersion of asbestos fibers that originate in the parenchyma of the lung.[12]

When asbestos fibers are affixed to the alveolar surface epithelium, the immune system produces reactive oxygen species (ROS) through trauma. Type 1 alveolar cells will be damaged by reactive oxygen species, and this damage will result in the production of fibroblast growth factor beta-2 (FGF-2), one of the factors involved in fibrosis. When macrophages phagocytize asbestos particles, they release inflammatory mediators such as interleukins and tissue necrosis factor (TNF). These mediators have a crucial role in inducing the release of more inflammatory cells, such as myofibroblasts and lymphocytes, which doubles the amount of fibrosis.[14]

Fibrosis may also worsen due to macrophage production of insulin-like growth factor and platelet-derived growth factor (PDGF). The destruction of matrix glycoproteins by the macrophage-produced plasminogen activator results in increased fibrosis and injury to interstitial tissue. Prolonged inflammation triggers the mitogen-activated protein kinase (MAPK) pathway, which in turn triggers transcription factors including nuclear factor kappa beta (NF-κβ) and activator protein-1 (AP-1), as well as promoter gene activity. Autoantibody synthesis is correlated with exposure to amphibole fibers. A positive antinuclear antibody test result for autoantibodies increases the likelihood of interstitial tissue deterioration and pleural abnormalities.[14]

Fibrosis and cellular responses have the power to start and encourage the development of cancer.10 Because asbestos fibers are genotoxic, they can harm DNA, interfere with gene transcription, and alter protein expression—all of which are critical for the processes of cell division, inflammation, and death. The composition and chemical characteristics of asbestos fibers, the duration of exposure, free radicals, and cigarette smoke are all factors that affect how pathogenic asbestos fibers are. Asbestos fibers release reactive nitrogen species (RNS) in addition to ROS. Both ROS and RNS can modify a variety of macromolecules, such as ribonucleic acid (RNA), DNA, lipids in cell membranes, and signal transduction proteins, which can lead to cytotoxicity, cancer, and malfunction in cells.[12,14]

By first harming the pulmonary epithelium, species produced from cigarette smoke can damage alveolar epithelial cells and exacerbate DNA damage. The ROS, NF-kB, transcription factor-activated protein-1 (AP-1), pulmonary epithelium, and other factors contribute to the exaggeration of inflammation during the fibrosis process' inflammatory response to asbestos fibers. Tumor necrosis factor-a (TNF-α), transforming growth factor B (TGF-B), platelet-derived growth factor (PDGF), and IL-8 are growth factors and cytokines that contribute to the pathophysiology of pulmonary fibrosis. These factors and cytokines amplify the lesion, promote fibroblast proliferation, and increase collagen accumulation. The quantity of fibers that build up in the lung determines the severity of pulmonary fibrosis.[13]

In the beginning, asbestosis is thought to be an alveolar process that will progress to the interstitial space in the lung. An ongoing inflammatory process

in the interstitial space causes pro-inflammatory agents to constantly interact to stimulate the fibroblasts at the site of the lesion. The initial characteristics of asbestosis include foci fibrosis in the bronchiolus respiratorius and ductus alveolaris, according to the amplification of asbestos fibers that are usually seen in the lower, middle, and subpleural regions. This process will continue unabated, resulting in a case of irreversible damage.[14] The pathology of asbestosis may be seen in Figure 2.

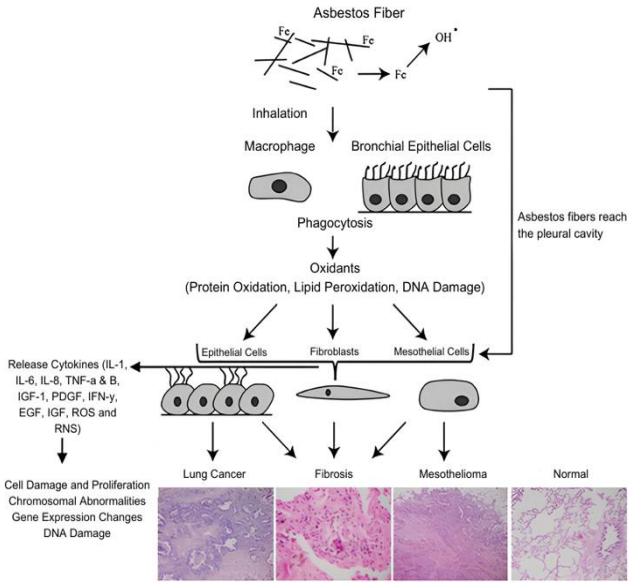

Figure 2. Pathophysiology of Lung Asbestosis[15]

DIAGNOSIS

The approach to enforcing asbestosis as an occupational disease uses seven steps to determine occupational disease. The determination of the seven steps is by the Regulation of the Minister of Health (*Permenkes*) of the Republic of Indonesia Number 56 of 2016 concerning the implementation of occupational disease services.

The seven steps of occupational disease diagnosis include establishing a clinical diagnosis, determining the exposure experienced by workers in the workplace, determining the relationship between exposure and clinical diagnosis, determining the magnitude of exposure, determining individual factors that play a role, determining exposure outside the workplace, and determining the diagnosis of occupational disease, as shown in Figure 3.

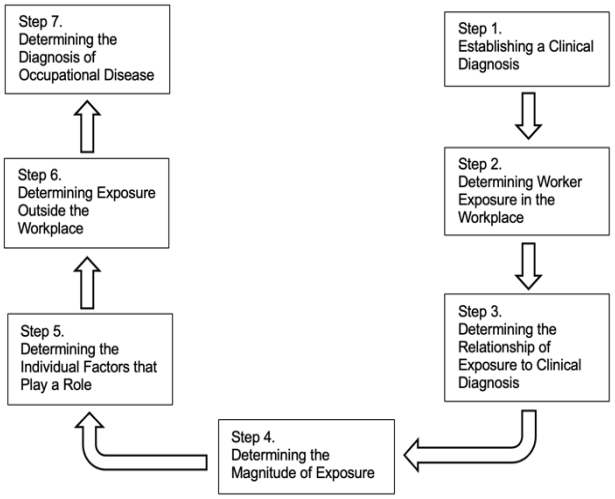

Figure 3. Seven Steps of Occupational Lung Disease Diagnosis[16]

Asbestosis is diagnosed clinically using a combination of physical examination, history collection, and ancillary testing. Work history is the most crucial historical question to pose. The history of one's occupation is crucial to the diagnosis and treatment plan. Jobs where there is a chance of being exposed to asbestos fibers directly or indirectly are linked to an increased risk of asbestosis. The most vulnerable occupations to asbestos fiber exposure are those that involve mining and grinding. Workers in the aircraft sector, including mechanics, are also susceptible to asbestosis.[15]

Other workers, such as building construction, shipping, steam engine operators, heating technicians, painters, building supervisors, and building maintenance workers, are also at risk even though they are not directly exposed to asbestos fibers.[15] The important exposure history to ask about includes the duration, onset, type, and intensity of asbestos fiber exposure received by the patient. Exposure history to asbestos fibers helps in excluding various types of interstitial lung diseases such as coal pneumoconiosis, silicosis, hypersensitive pneumonitis, idiopathic pulmonary fibrosis disease, and other interstitial lung diseases. Clinical symptoms appear after a history of exposure to asbestos fibers for approximately 10–20 years.[15]

Initial clinical symptoms may include shortness of breath at work and then progressively worse even when not at work. Shortness of breath is followed by a persistent dry cough. Advanced and progressive

pulmonary fibrosis causes increasingly severe shortness of breath accompanied by a productive cough.[14] Clinical symptoms that appear will be more frequent and more severe in patients who have a history of smoking. Patients sometimes present with complaints of discomfort or chest heaviness. In patients with advanced asbestosis that progresses to lung cancer, complaints of shortness of breath will be more severe and may be accompanied by hemoptysis. Other symptoms include decreased appetite and weight loss.[15,17]

Abnormalities in the physical examination that are first encountered are rhonchi in the posterior basal lung, while in advanced asbestosis, decreased and faint breath sounds can be found in lung percussion.[15] Tachypnea, cyanosis, and finger tapping are found if the disease is more advanced.[12] Further examinations that can be done to support the diagnosis are spirometry, chest X-ray (CXR), computed tomography scan (CT scan), high-resolution CT scan (HRCT), bronchoalveolar lavage (BAL), and histopathology. The following will describe the various advanced and supporting examinations to facilitate the diagnosis of asbestosis.[12,15]

According to the 2014 Helsinki guidelines, the diagnosis of asbestosis can be strengthened by a history of exposure to asbestos fibers and three types of supporting examinations that support asbestosis in addition to anamnesis and physical examination. The supporting examination in question is based on the results of BAL, CXR, and thoracic CT scans.[17]

Based on BAL results, asbestosis can be established if there is evidence of >0.1 million asbestos fibers (>5 μm)/g dry lung or >1 million asbestos fibers (>1 μm)/gr dry lung measured using an electron microscope and has met the standards of a qualified laboratory, or if there is evidence of >1000 asbestos bodies/g dry lung (100 asbestos bodies/g wet lung or >1 asbestos body/ml) from BAL fluid seen using a light microscope and meets the standards of a qualified laboratory.[17]

A thoracic CT scan examination was performed if there was a thoracic photograph according to ILO detected 0/1, 1/0, or if there was a discrepancy between the findings of pulmonary function results stating restriction while the results of the radiology department stated normally; extensive pleural changes greatly hampered the visibility of radiographs to see the state of the lung parenchyma so that it was considered to be a supporting examination of thoracic CT scan. The "0/1" indicates that there are very few tiny opacities in the lungs, which are hardly perceptible and on the verge of being categorized as a category 1 finding, which would indicate a somewhat greater level of asbestosis.[17]

Criteria that support asbestosis from thoracic CT scan results are the sum of ≥2-3 bilateral irregular opacities in the lower lobe based on film or there is a bilateral honeycombing picture ≥2 based on the International Classification of high-resolution CT for Occupational and Environmental Respiratory disease classification (ICOERD) system.[17]

Histopathological confirmation remains indispensable for suspicion of malignancy due to asbestos fiber exposure and exclusion of differential diagnosis. The flow of definitive diagnosis of asbestosis remains based on assessment of a long history of asbestos fiber exposure, sufficient time between exposure and onset of disease, persistent bilateral crackles on inspiration, clubbing fingers on hands and feet, restriction pattern on pulmonary function examination, and pulmonary fibrosis on thoracic photograph or HRCT.[18,19]

LUNG EXAMINATION

A typical picture of lung function abnormalities in asbestosis patients can be seen from the results of spirometry examination in the form of restriction abnormalities with a decrease in forced vital capacity (FVC), total lung capacity (TLC), and a decrease in lung diffusion capacity which can be assessed from the diffusing capacity of the lung for carbon monoxide (DLCO) examination.[8,12]

Spirometry examination does not show obstruction abnormalities in workers with asbestosis, but obstruction abnormalities can be found in patients with a history of smoking. Restrictive abnormalities are characteristic of asbestosis, as asbestos fibers

can trigger an inflammatory response and fibrosis formation in the respiratory and terminal bronchioles.[15]

A cohort study conducted by Yang et al on 281 asbestos insulator workers found a decrease in FVC along with forced expiratory volume in the first second (FEV_1) and TLC along with the increasing severity of the asbestosis.[18] Research conducted by Barnikel et al, which assessed lung function in asbestosis patients, observed that about 44 of 56 patients (78.6%) had a decrease in FVC ≤100 ml/year, 12 patients (21.4%) with a decrease in FVC ≥100 ml/year, and as many as 4 patients (7.1%) had a decrease in FVC ≥200 ml/year.[15] A study by Bledsoe et al in Boston of 62 asbestosis cases obtained that among those who had CXR interpretation of ILO ≥1, 51 cases were smokers, and only 1 person had no smoking history.[20]

The study by Bledsoe et al also describes the CXR, lung function, and histopathology of asbestosis patients in 47 patients. The results of the pulmonary function examination revealed that as many as 20 patients (42.5%) had obstructive patterns, 15 patients (32%) had restrictive patterns, and 12 patients (25.5%) with normal results. In this study, no mixed lung function results were found in the patients.[20]

The next examination of lung function abnormalities in lung diffusion capacity using the DLCO tool. Abnormalities in the interstitial space will cause diffusion disorders if it contain substances that cause diffusion to be blocked. The DLCO examination, in addition to assessing pulmonary diffusion disorders, can also see hypoxemia during exercise, which is found in asbestosis patients.[12,15]

Decreased DLCO is a more reliable early indicator of asbestosis than decreased TLC and FVC from spirometry findings. The DLCO test is not specific for measuring asbestosis, although it is a relatively sensitive criterion for detecting interstitial lung disease in its early stages.[8,17] The DLCO test is sensitive for diagnosing diffuse interstitial pulmonary fibrosis; however, it is less specific because low DLCO can be produced by various interstitial lung disorders. DLCO has a sensitivity of 85%; however, its specificity for diagnosing asbestosis is uncertain. The DLCO examination is also used to monitor the progression of the disease and assess the patient's response to medication.[15]

RADIOLOGICAL FINDINGS

The CXR of asbestosis patients show irregular fine ridges mainly scattered in the posterior, basal, and subpleural areas of the lung. Diffuse reticulonodular infiltrates can also be found in the lateral basal areas of the lung. In advanced disease states, a honeycomb appearance can be found in the lower lobes accompanied by bilateral pleural thickening and calcification. Uneven lesions in the diaphragmatic area also indicate an advanced disease course. Along with the enlargement of the lesion, this will make the heart border blurred, known as the shaggy heart border sign, and begin to appear pleural thickening accompanied by bilateral pleural calcification.[12,16]

According to the International Labor Organization (ILO) classification, the technique of reading asbestosis thoracic radiographs included in the pneumoconiosis group consists of assessing the quality of CXR, lung parenchymal abnormalities, pleural abnormalities, and other abnormalities. The assessment of the quality of CXR can be interpreted as good, acceptable without technical defects that interfere with the classification of thoracic radiographs in pneumoconiosis, acceptable with technical defects but still adequate for the classification of thoracic radiographs of pneumoconiosis, and unacceptable. Lung parenchymal abnormalities can be assessed using indicators based on small opacity and large opacity lesion images.[16]

Small opacity lesions are assessed based on profusion or density, zone, shape, and size. The division of categories and subcategories of density is divided into category 0 with subcategories 0/-, 0/0, and 0/1 with the interpretation that there are no small ridges or densities less than category 1. Category 1 with subcategories 1/0, 1/1, and 1/2 with the interpretation that there are relatively few small ridges. Category 2 with subcategories 2/1, 2/2, and

2/3 has the interpretation that there are several small ridges; the lung pattern is not very clear. Category 3 with subcategories 3/2, 3/3, and 3/+ with the interpretation of many small ridges, partial or complete lung scars are not clear.[16]

Lung parenchymal lesion location zones were divided into 3, consisting of the upper 1/3, middle 1/3, and lower 1/3 of the lung. Then small opacities were categorized by shape and size. The size of small opacities was classified as p (≤1.5 mm), q (1.5-3 mm), or r (3-10 mm). Irregular small opacities were classified as s, t, or u with the same size as small round opacities. A profusion of small opacities was grouped into a 4-point major category scale (0-3), with each major category divided into 3 subcategories, giving a 12-point scale between 0/- and 3/+.[16]

Major opacities were categorized into categories A, B, and C. Category A has dimensions (≤50 mm), category B (>50 mm), and category C is 1 large ridge that exceeds the equivalent area of the right upper zone or several large ridges combined exceeding the right upper zone. Abnormalities in the pleura are assessed based on the location, whether in the diaphragm or chest wall, the width of the pleural thickening <1/4 of the chest wall or >1/4 of the chest wall, <1/2 or >1/2 of the chest wall, whether there is calcification or not, blunt or acute costophrenic angle, whether there is diffuse pleural thickening or not, whether there is expansion and symbols.[16]

Diagnosis of asbestosis will be facilitated with the help of supporting examinations such as CT scan or HRCT. The image of asbestosis from HRCT has characteristics such as thickening of intralobular lines, interlobular lines, subpleural curved lines, irregular nodules at the base, ground-glass opacity, and honeycombing.[15] CT scan examination of advanced asbestosis shows a picture of peripheral bands, thickening of interlobular septa, and ectasis rings, especially at the basal lung.[12,15] Figure 4 shows a radiological picture of both thorax photos and CT scans found in asbestosis cases.

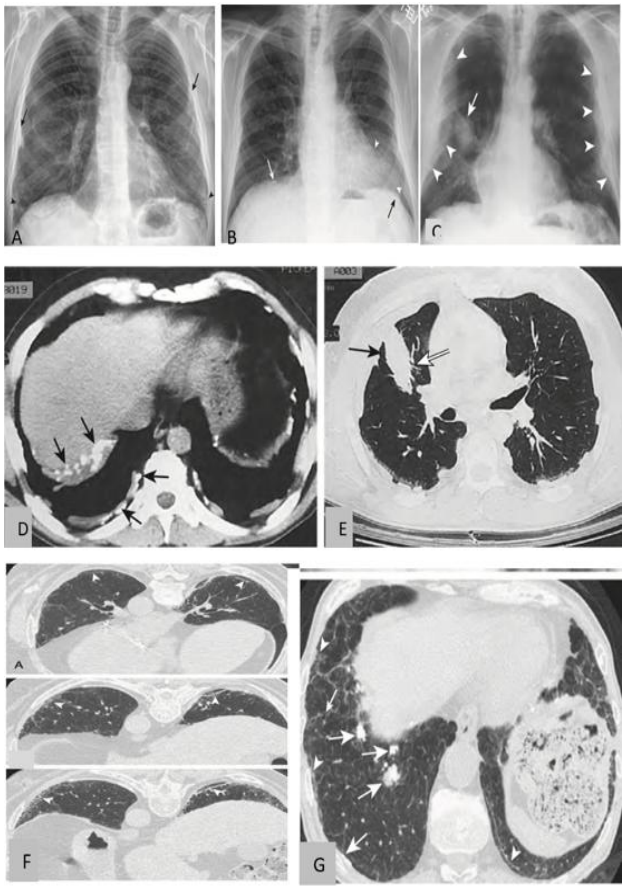

Figure 4. Radiologic findings in a case of Asbestosis. (A) PA thorax photograph showing bibasilar reticular opacity consistent with fibrosing lung disease with bilateral pleural plaque calcification; (B) "Shaggy heart border sign" with bilateral pleural plaque calcification; (C) Thorax image of subpleural plaques with well-demarcated lesions in the dextral medial lobe area; (D) CT-Scan of the thorax of an asbestosis patient with pleural plaques; (E) Some of the plaques are calcified; (F) Axial-prone HRCT shows bilateral subpleural reticular and linear opacities; (G) Pleural calcification, peripheral bands, and reticular opacity with mild bronchiectasis suggestive of pulmonary fibrosis disease[15]

BRONCHOALVEOLAR LAVAGE (BAL)

BAL examination can be used to support the diagnosis of asbestosis. The pathognomonic characteristic of asbestosis that can be seen from the BAL examination is the asbestos body. The asbestos body is an asbestos fiber with a membrane composed of protein and iron. The fibers are coated with protein and iron. The asbestos body is formed more in amphibole than chrysotile. The results of the BAL examination, in addition to looking for asbestos bodies, can see an increase in the number of cells, especially macrophages, neutrophils, and eosinophils, which can distinguish acute or chronic phase infections.[15]

The increased number of macrophages is consistent with the important role of alveolar macrophages in the pathogenesis of asbestosis. Asbestos bodies can also be found from lung tissue biopsy and sputum cytology. The increase in asbestos bodies is related to the degree of damage in the histopathological results of asbestosis.[15]

Asbestos bodies in BAL fluid with levels >1/ml or >1000/g indicate a positive asbestos fiber exposure. A study conducted by Sartorelli et al on 73 workers showed lymphocytosis above normal values in the acute phase of asbestosis.[21] In the chronic phase, the dominant cells found were increased macrophages and neutrophils.[15]

In asbestosis patients with a history of smoking, there may be an increase in eosinophils. An increased CD4/CD8 ratio in lymphocytic alveolitis is also seen in asbestosis. Abnormalities can be found on BAL examination even though clinically and radiologically there is no evidence of asbestosis.[15]

Apart from BAL results, asbestos bodies can be found in lung tissue biopsy, sputum cytology, and bronchial lavage. Figure 5 shows a picture of asbestosis from histopathologic examination showing lesions consisting of foci of peribronchial fibrosis with chronic interstitial inflammation, and macrophage accumulation accompanied by proliferation of type II pneumocytes. Figure 5 shows an asbestos body as seen using light microscopy.[12]

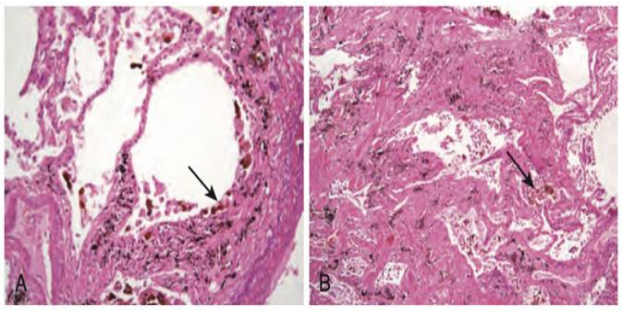

Figure 5. (A) Asbestos body with early fibrosis with preserved lung parenchyma architecture. (B) diffuse interstitial fibrosis with destruction of wall structure with honeycombing[15]

TREATMENT

Management of asbestosis is palliative depending on the symptoms, and to date, there is no specific treatment that is effective for asbestosis. Supportive therapy is an option. Once the diagnosis is made, patients should avoid asbestos exposure to avoid further damage. Education and knowledge about the long-term effects of asbestos exposure should be provided to workers exposed to asbestos fibers. Other workers who have not shown symptoms of asbestosis should be examined for early diagnosis. Smoking is one of the factors that can aggravate and accelerate the worsening of asbestosis disease, so quitting smoking as soon as possible can help reduce the worsening of existing disease.[15]

Medicamentous therapy aims to reduce symptoms and progression of existing disease. Drugs that can be given include corticosteroids, immunosuppressives, antineoplastics, and antibiotics. Corticosteroids are given with the aim of suppressing acute and chronic inflammatory processes, thus reducing lung damage, although the success rate is still low. Corticosteroids also suppress the release of ROS and inflammatory mediators during the inflammatory process. Prednisolone is a type of corticosteroid that can be given at a dose of 0.5–1 mg/kgBW/day for 4–12 weeks.[13]

Monitoring for tapering off is done when the patient's condition is stable and improving. The dose can be reduced to 0.25–0.5 mg/kg in the next 4–12 weeks. Colchicine is one of the drugs that can be administered with an antifibrosis effect.[10] Antibiotic therapy is given clinically if there are signs of secondary infection. The influenza vaccine is intended to prevent respiratory infections caused by *pneumococcus* and *Haemophilus influenza*.[12] Oxygen therapy is given to patients suffering from hypoxia during rest or activity.[13]

Pulmonary rehabilitation and the management of underlying diseases that aggravate asbestosis can improve the quality of life of patients even if the course of the disease is already progressive. The surgical action that can be performed in this case of asbestosis is thoracotomy by performing a decortication of subpleural fibrosis. Decortication has a risk of increased incidence of postoperative pulmonary atelectasis. Pleurectomy can be performed as an alternative, but if the production of pleural fluid on the pleural effusion is still ongoing, then the action is palliative to evacuate the pleural

fluid, thus reducing the shortness of breath felt by the patient. A pulmonary transplant is a choice when all the other therapies and procedures fail.

CONCLUSION

Establishing the diagnosis of asbestosis is still difficult to do initially due to clinical symptoms that resemble other pneumoconiosis. The history of asbestos fiber exposure in terms of exposure, amount, intensity, and position of workers becomes crucial. BAL and thoracic HRCT scan play a major role in distinguishing asbestosis from other types of pneumoconiosis.

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CONFLICT OF INTEREST

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