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REVIEW OF CURRENT AND EMERGING ANTIFIBROTIC THERAPY FOR ASBESTOSIS AND SILICOSIS

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ABSTRACT

Asbestosis and silicosis are progressive occupational lung diseases driven by asbestos fiber and respirable crystalline silica inhalation, resulting in chronic inflammation and irreversible pulmonary fibrosis. While prevention and early diagnosis remain paramount due to the lack of curative options, current supportive therapies offer limited efficacy. This review examines their pathogenesis, immune mechanisms, and management strategies, with emphasis on antifibrotic agents like pirfenidone and nintedanib, alongside emerging therapies such as PDE4B inhibitors (e.g., nerandomilast) and stem cell approaches. Recent clinical data suggest slowed fibrosis progression, but occupational-specific RCTs are needed to optimize safety, efficacy, and biomarkers in high-risk cohorts like construction workers.

KEYWORDS: Asbestosis; Silicosis; Antifibrotic therapy; Pirfenidone; Nintedanib; Interstitial lung disease.

INTRODUCTION

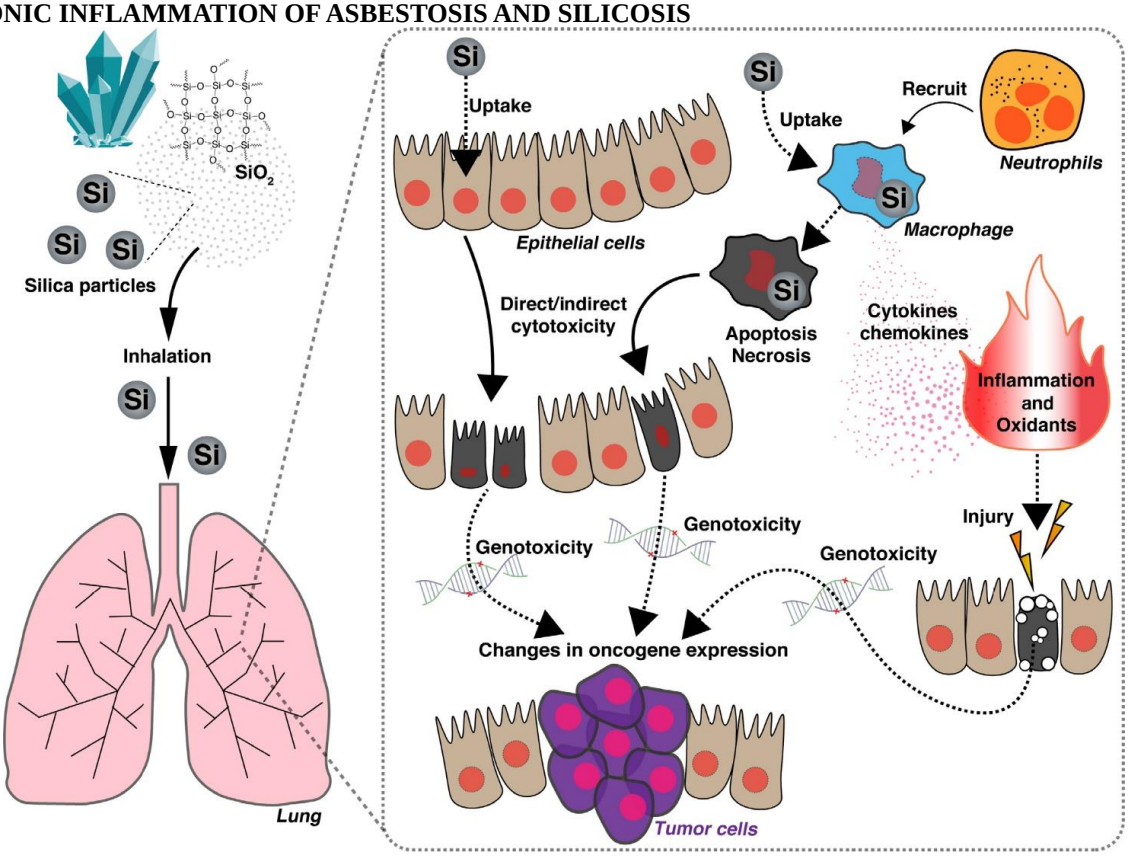
Numerous respiratory ailments can arise as a result of workplace exposures. Some of these occupational illnesses also have causes that are not related to work, such as asthma, chronic obstructive pulmonary disease (COPD), and lung cancer.[1,2] Other respiratory diseases associated with occupational settings, like silicosis and asbestosis, result from specific exposures.[3] Diseases such as silicosis and asbestosis provide a distinct opportunity for monitoring the population levels of occupational diseases since they are primarily caused by workplace exposures.[4] Fibrosis is the hallmark of silicosis, a lung condition brought on by breathing in respirable crystalline silica (RCS).[4,5] In the past, silicosis was common among stonemasons and miners, but it has lately been noted in contemporary manufacturing and construction settings.[6] Another occupational lung disease associated with fibrosis is asbestosis, which is brought on by prolonged exposure to asbestos, a term that refers to six naturally occurring silicate fibers.[2,3]

ETIOPATHOGENESIS OF ASBESTOSIS AND SILICOSIS

MECHANISM

It is commonly known that silicosis, a kind of pneumoconiosis, is caused by occupational and environmental exposure to silica particles.[4,5] Bilateral diffuse interstitial fibrosis of the lungs brought on by asbestos fiber inhalation is known as asbestosis.[3][2][7] When silica particles have a diameter of less than 5 µm—small enough to enter the distal airways—they are referred to as "respirable" and can reach the alveoli.[4] Neutrophils, T-lymphocytes, and mast cells are among the immune system cell types that gather in BAL and/or interstitial areas; fibrosis is linked to rats exposed to silica or asbestos.[8][9]

CHRONIC INFLAMMATION OF ASBESTOSIS AND SILICOSIS



SILICOSIS IMMUNE CELL

Immune cells such as macrophages, neutrophils (NEUTs), mast cells (MCs), dendritic cells (DCs), T cells, and B cells are all stimulated by silicon dioxide (SiO2). Immune cells control processes linked to lung fibrosis and silicosis via a number of molecular mechanisms.[9][6] The regulation of inflammation brought on by silicosis involves both the innate and adaptive immune systems.[5][9]

MACROPHAGES

M1 macrophages are activated in the early phases of silicosis, which mainly causes inflammation by producing the pro-inflammatory IL-1β and IL-6 cytokines.[9] Afterwards, M2 macrophages are stimulated to release TGF-β and IL-10, two pro-fibrotic cytokines that aid in tissue repair.[9][6]

NEUTROPHILS

NEUTs are essential for the innate immune system's fight against infections because they immediately destroy them through phagocytosis and degranulation.[9] When exposed to silica, neutrophils release a variety of cytokines and neutrophil extracellular traps (NETs), which promote the synthesis of downstream factors and draw additional neutrophils or other leukocytes to the lung.[9][6]

CURRENT THERAPEUTIC APPROACHES

According to an NIH workshop study, corticosteroids or immunosuppressants have historically been used to treat IPF, silicosis, and asbestosis, with depressing outcomes in terms of morbidity and death.[10] Mineral-induced lung illness can be prevented or lessened by controlling occupational exposure to minerals and removing symptomatic individuals from the job.[7][11]

ANTIFIBROTIC THERAPY

Antifibrotic therapy, which has shown the most promise, is utilized for managing different kinds of interstitial diffuse lung diseases.[12] Silica particles provoke inflammatory responses in lung tissue and stimulate fibroblast proliferation, leading to pulmonary fibrosis.[5][6] Antifibrotic medications possess anti-inflammatory properties and restrict the growth of fibroblasts, rendering them suitable for the treatment of silicosis.[10][13] Clinical research indicates that pirfenidone and nintedanib, two antifibrotic medications authorized for managing diffuse interstitial lung diseases, are generally well tolerated by patients and help slow the deterioration of lung function.[14][12][15] These medications decrease the activity of growth factors that contribute to the development and progression of fibrosis.[16]

Pirfenidone

Pirfenidone, a compound derived from pyridine, functions as an immunosuppressant that possesses both antifibrotic and anti-inflammatory properties. It can block the process of fibrosis by modulating or inhibiting factors such as fibroblast growth factor, connective tissue growth factor, TGF-β, oxidative agents, and pro-inflammatory elements in the lungs and kidneys.[16] In

rats, pirfenidone diminished inflammation and alveolar injury caused by silica exposure; lowered the concentrations of TNFα, IL-1β, and IL-6 in lung tissue; reduced the expression of collagen I and vimentin; and inhibited the expression of TGFβ1, Smad2/3, and EMT.[10][13]

Nintedanib

Nintedanib, which is a tyrosine kinase inhibitor, is utilized in the clinic for the treatment of fibrotic lung conditions such as idiopathic pulmonary fibrosis and chronic interstitial lung diseases, with the aim of reducing the decline in lung function and the likelihood of pulmonary exacerbations.[12][15] The treatment of mouse models of lung fibrosis induced by silica and bleomycin with the tyrosine kinase inhibitor nintedanib resulted in the inhibition of PDGF receptor activation, decreased fibroblast growth, and halted the transformation of fibroblasts into myofibroblasts; it also caused a reduction in the quantities of lymphocytes and neutrophils found in the bronchoalveolar lavage fluid (BALF); in addition, it lowered the levels of IL-1β, keratinocyte chemoattractant (KC), tissue inhibitor of metalloproteinase-1 (TIMP-1), and collagen.[6][13] A research experiment demonstrated that the intratracheal administration of nintedanib as a nanosuspension exhibited significant antifibrotic effects in a mouse model of silicosis, while avoiding both local and systemic safety issues.[6]

CONCLUSION

Asbestosis and silicosis are primarily preventative occupational diseases rather than curable ailments because they are mostly permanent and do not respond to current medical therapies. Despite long-standing awareness of their hazards, the majority of current cases are a reflection of past exposure control failures. New instances will keep appearing if workplace exposures are not consistently and strictly controlled (e.g., dust suppression, protective measures). Through early and accurate diagnosis, required reporting, and efficient worker education, doctors play a crucial preventative role in reducing the burden of disease.

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