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MORPHOLOGICAL FEATURES OF SPLENIC FIBROTIC PROCESSES ASSOCIATED WITH PM2.5 EXPOSURE

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Abstract

Atmospheric air pollution caused by fine particulate matter (PM2.5) represents a significant global environmental and public health concern. Due to their small size, PM2.5 particles easily penetrate the respiratory tract and enter the systemic circulation, exerting adverse effects on various organs, including the immune system. The spleen, as a central peripheral immune organ, is particularly sensitive to chronic environmental stressors. This article presents an expanded analysis of the morphological features of fibrotic processes developing in splenic tissue under prolonged PM2.5 exposure. Based on morphological, histopathological, and immunomorphological data, structural remodeling of the splenic stroma, excessive collagen deposition, microcirculatory disturbances, and chronic inflammatory reactions are described. The mechanisms of fibrogenesis in the spleen under conditions of atmospheric air pollution are discussed, highlighting the role of oxidative stress, persistent inflammation, and fibroblast activation. The presented data emphasize the importance of long-term air quality control for preventing immune organ dysfunction.

Keywords: atmospheric air pollution, PM2.5, spleen, fibrosis, collagen fibers, morphological changes, immune system.

Main Body

Introduction

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Atmospheric air pollution remains one of the most critical environmental challenges affecting human health worldwide. Rapid industrial development, urbanization, and increased vehicular emissions have significantly elevated the concentration of fine particulate matter in the air. Among these pollutants, particulate matter with an aerodynamic diameter of less than 2.5 μm (PM2.5) is considered especially hazardous due to its high penetration capability and systemic biological effects.

PM2.5 particles are capable of bypassing the upper respiratory defense mechanisms and reaching the alveolar spaces, from where they translocate into the bloodstream. Once distributed systemically, these particles may accumulate in various organs, including immune organs such as the spleen. The spleen plays a vital role in immune surveillance, blood filtration, and maintenance of immunological homeostasis. Therefore, morphological alterations in splenic tissue caused by environmental pollutants warrant thorough investigation.

While numerous studies have focused on pulmonary and cardiovascular effects of PM2.5, its impact on immune organs, particularly the spleen, remains insufficiently explored. Chronic exposure to atmospheric pollutants may lead to long-term structural remodeling, including fibrotic changes, which can significantly impair splenic function.

Pathogenetic Mechanisms of PM2.5-Induced Fibrosis

The biological effects of PM2.5 are largely mediated through oxidative stress and chronic inflammation. Upon entering the organism, PM2.5 particles stimulate the excessive generation of reactive oxygen species, leading to oxidative damage of cellular membranes, proteins, and nucleic acids. These processes disrupt cellular homeostasis and initiate inflammatory signaling cascades.

Persistent inflammation results in the sustained activation of immune cells and stromal components within the spleen. Pro-inflammatory cytokines such as interleukins and tumor necrosis factor promote fibroblast proliferation and activation. Activated fibroblasts synthesize increased amounts of collagen and other extracellular matrix components, forming the morphological basis of fibrotic remodeling.

Additionally, PM2.5 exposure induces endothelial dysfunction and microvascular damage, leading to chronic hypoxia. Hypoxic conditions further enhance fibrogenesis by stimulating transforming growth factor-beta (TGF-β) signaling pathways, which are central regulators of tissue fibrosis.

Morphological Characteristics of Splenic Fibrosis: Morphological examination of the spleen under prolonged PM2.5 exposure reveals pronounced alterations in both stromal and parenchymal components. One of the earliest signs of fibrotic remodeling is thickening of the splenic capsule and trabeculae due to excessive collagen deposition.

Histological analysis demonstrates an increased density of collagen fibers within the splenic stroma, particularly surrounding lymphoid follicles and vascular structures. In the white pulp, fibrotic expansion leads to compression and partial atrophy of lymphoid follicles, resulting in reduced lymphocyte density and disturbed follicular architecture.

In the red pulp, fibrosis manifests as thickening of sinusoidal walls, narrowing of vascular lumens, and impaired blood flow. Perivascular fibrosis is frequently observed, indicating chronic vascular injury and adaptive stromal responses. These structural changes disrupt normal splenic microcirculation and filtration capacity.

Stages of Fibrotic Process Development

Splenic fibrosis associated with PM2.5 exposure typically develops in a gradual, stage-dependent manner. In the initial stage, functional disturbances predominate, with

minimal morphological alterations. Mild collagen accumulation and subtle stromal thickening may be detected only through histochemical staining.

As exposure persists, intermediate stages are characterized by progressive collagen deposition, fibroblast proliferation, and partial replacement of normal splenic tissue. The architectural relationship between white and red pulp becomes increasingly distorted.

In advanced stages, extensive fibrosis and sclerosis dominate, leading to irreversible structural remodeling. The parenchyma-to-stroma ratio shifts significantly, compromising the spleen's immunological and hematological functions.

Immunomorphological Consequences of Splenic Fibrosis: Fibrotic remodeling of the spleen has profound implications for immune system function. Reduction of lymphoid tissue volume and disruption of splenic architecture impair antigen processing, lymphocyte activation, and immune cell trafficking.

Macrophage and dendritic cell dysfunction further compromises immune surveillance and antigen presentation. Consequently, chronic PM2.5 exposure may contribute to immunosuppression, increased susceptibility to infections, and reduced adaptive immune responses.

These immunomorphological changes represent a structural basis for secondary immune deficiency states associated with long-term environmental pollution.

Hygienic and Preventive Significance: The identification of fibrotic changes in the spleen highlights the long-term health risks associated with chronic exposure to fine particulate air pollution. These findings underscore the necessity of implementing effective air quality control measures, particularly in densely populated urban areas.

Preventive strategies aimed at reducing PM2.5 emissions may play a crucial role in preserving immune system integrity and preventing chronic immune-mediated

disorders. From a public health perspective, monitoring splenic morphology may serve as an indicator of systemic effects of environmental pollution.

Conclusion: Chronic exposure to PM2.5-contaminated atmospheric air leads to progressive fibrotic remodeling of splenic tissue. These changes are characterized by excessive collagen deposition, microcirculatory disturbances, and structural disruption of lymphoid components. Splenic fibrosis significantly impairs immune and filtration functions, contributing to immune system dysfunction under conditions of environmental pollution. The findings presented in this article provide a morphological basis for understanding the long-term immunotoxic effects of atmospheric air pollution and emphasize the importance of preventive environmental and hygienic measures.

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