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Pneumonitis: A Concise Clinical Review

***Beatrice V, Patch W, Wilson J**
Department of Hematology, Oslo University Hospital,Norway

***Corresponding Author:** Beatrice V, Department of Hematology, Oslo University Hospital,Norway

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Abstract

Pneumonitis represents a complex spectrum of inflammatory lung conditions distinct from infectious pneumonia, encompassing hypersensitivity reactions, drug-induced injury, radiation damage, and chemical aspiration. This article provides a comprehensive overview of pneumonitis, exploring its pathophysiology, clinical presentation, diagnostic approaches, and currentmanagement strategies. With increasing recognition of immunotherapy-related pneumonitis and advances in antifibrotic therapies, understanding this condition has never been more critical for clinicians. The article synthesizes current evidence from international guidelines and recent clinical trials to present a cohesive framework for diagnosis and treatment

Introduction

Pneumonitis, derived from the Greek pneumōn (lung) and -itis (inflammation), refers broadly to inflammation of lung tissue not caused by infectious organisms. This distinction from pneumonia is crucial, as management approaches differ fundamentally. The condition represents an exaggerated immune response or direct toxic injury to the pulmonary parenchyma, particularly affecting the alveoli and interstitial spaces.

The clinical significance of pneumonitis has grown substantially in recent years due to several factors: the increasing use of immunomodulatory cancer therapies that can trigger immune-related adverse events, greater recognition of occupational and environmental causes, and improved diagnostic capabilities with high-resolution imaging. Chronic pneumonitis, if unrecognized or untreated, can progress to irreversible pulmonary fibrosis, respiratory failure, and death, making early diagnosis and intervention paramount.

This review aims to provide clinicians with a comprehensive understanding of pneumonitis, incorporating the latest classification systems, diagnostic algorithms, and evidence-based treatment approaches

Pneumonitis is best understood as a clinical syndrome with multiple potential triggers rather than a single disease entity. The major etiological categories include hypersensitivity pneumonitis, drug-induced pneumonitis, radiation-induced pneumonitis, and aspiration pneumonitis

Hypersensitivity Pneumonitis

Hypersensitivity pneumonitis (HP) is an immune-mediated interstitial lung disease caused by repeated inhalation of environmental antigens in sensitized individuals.

Common antigens include:

- **Molds and bacteria**: Thermophilic actinomycetes from moldy hay ("farmer's lung"), Aspergillus species, and contaminated humidifiers or hot tubs
- **Animal proteins**: Avian proteins from bird droppings and feathers ("bird fancier's lung"), animal dander
- **Chemical sensitizers**: Isocyanates in paints and foams, metalworking fluids

Drug-Induced Pneumonitis

Common culprits include:

- **Chemotherapeutic agents**: Bleomycin, methotrexate, cyclophosphamide
- **Immunomodulatory drugs**: Immune checkpoint inhibitors (anti-PD-1/PD-L1 antibodies), interferon therapies
- **Cardiovascular drugs**: Amiodarone, certain antiarrhythmics
- **Antibiotics**: Nitrofurantoin, sulfonamides

Radiation-Induced Pneumonitis

Patients receiving thoracic radiation therapy for breast, lung, or esophageal cancer, as well as those undergoing whole-body irradiation prior to bone marrow transplantation, are at risk for radiation pneumonitis. Symptoms typically emerge 1-3 months after treatment completion, though later presentations occur.

Risk factors include radiation dose and volume, concurrent chemotherapy (particularly with immune checkpoint inhibitors), older age, and pre-existing lung disease . The pathophysiology involves direct DNA damage, oxidative stress, and a cytokine-mediated inflammatory cascade that can extend beyond the radiation field.

Aspiration Pneumonitis

Aspiration of gastric contents or other irritant substances triggers a chemical burn of the airways and lung parenchyma. Gastric acid causes rapid bronchoconstriction, atelectasis, and edema, which may resolve spontaneously or progress to acute respiratory distress syndrome. Bacterial superinfection occurs in approximately 25% of cases.

Pathophysiology

Immune-Mediated Mechanisms

In hypersensitivity pneumonitis, repeated antigen exposure in susceptible individuals triggers a complex immune response involving both innate and adaptive immunity. The initial phase involves antigen presentation by alveolar macrophages to CD4+ T lymphocytes, leading to Th1 and Th17 polarization. These cells release cytokines including interferon-gamma, interleukin-17, and tumor necrosis factor-alpha, recruiting additional inflammatory cells and promoting granuloma formation.

Direct Toxic Injury

Aspiration of gastric acid causes immediate chemical burns to the alveolar epithelium and capillary endothelium, increasing vascular permeability and triggering complement activation. The resulting inflammatory exudate fills air spaces, impairing gas exchange. Unlike immune-mediated forms, the initial injury is non-specific, though subsequent inflammation may amplify damage.

Laboratory Investigations

Initial laboratory studies may reveal non-specific markers of inflammation including elevated erythrocyte sedimentation rate and C-reactive protein . Complete blood count may show neutrophilia, lymphocytosis, or eosinophilia depending on the underlying cause.

Pulmonary Function Testing

Spirometry typically reveals a restrictive pattern with reduced forced vital capacity (FVC) and preserved or reduced forced expiratory volume in one second (FEV1)/FVC ratio. Mixed obstructive-restrictive patterns occur occasionally. Diffusing capacity for carbon monoxide (DLCO) is often reduced, reflecting impaired gas exchange. Serial testing is essential for monitoring disease progression and treatment response.

Conclusion

neumonitis encompasses a diverse group of inflammatory lung conditions with distinct etiologies but shared pathophysiological features. The recognition that chronic inflammation can lead to irreversible fibrosis has shifted therapeutic paradigms toward earlier, more aggressive intervention. Recent advances in classification—particularly the distinction between fibrotic and non-fibrotic hypersensitivity pneumonitis—provide a framework for prognosis and treatment selection.

Successful management requires a systematic diagnostic approach, meticulous exposure assessment, and individualized treatment plans incorporating antigen avoidance, immunosuppression, and emerging antifibrotic therapies. Multidisciplinary collaboration between pulmonologists, radiologists, pathologists, and occupational medicine specialists optimizes outcomes. As our understanding of underlying immune mechanisms deepens, more targeted therapies will likely emerge, offering hope for patients with this challenging group of diseases

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