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Mini Review

Emerging Therapeutic Strategies for Drug-induced Pulmonary Injury: A Mini Review

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Abstract

Drug-induced pulmonary injury (DIPI) represents a significant, though often under-recognized, complication of pharmacotherapy. With the increasing global use of chemotherapeutic agents, immunomodulators, and novel biologics, the reported incidence of DIPI is rising, thereby posing significant diagnostic and management challenges. This mini-review outlines the mechanisms, clinical manifestations, and current treatment approaches for DIPI, while highlighting recent advances in biomarker discovery and targeted interventions. This review emphasizes early detection and personalized management to mitigate morbidity and mortality associated with this condition.

Introduction

Drug-induced pulmonary injury (DIPI) encompasses a spectrum of pulmonary pathologies resulting from adverse reactions to medications. These injuries may range from transient infiltrates to progressive fibrosis, often mimicking idiopathic or autoimmune pulmonary conditions. The growing arsenal of therapeutic agents, particularly in oncology and rheumatology practices, has substantially expanded the list of drugs implicated in DIPI, making it a critical area of clinical concern [1].

Recent studies estimate that drug-induced pulmonary injury accounts for approximately 3% - 5% of interstitial lung disease cases, with higher incidence reported among patients receiving antineoplastic agents and immune checkpoint inhibitors.

Mechanisms and clinical spectrum

The pathogenesis of DIPI is multifactorial and remains complex, involving direct cytotoxic drug effects and immune-mediated inflammatory responses, oxidative stress and genetic predisposition [2,3]. Common injury patterns include:

- Interstitial pneumonitis and fibrosis (e.g., commonly associated with bleomycin and immune checkpoint inhibitors)
- Organizing pneumonia (e.g., frequently linked to amiodarone and rituximab)
- Alveolar hemorrhage (e.g., potentially induced by anticoagulants or immunosuppressants)
- Pulmonary hypertension (e.g., related to anorexigens)

The clinical presentation is often nonspecific, with symptoms such as cough, dyspnea, and radiographic abnormalities which can often delay diagnosis. High-resolution computed tomography (HRCT) and bronchoscopic evaluation remain cornerstone diagnostic tools for evaluation in clinical practice, though histopathology is definitive.

Notably, although the pathological patterns of DIPI are heterogeneous, immune-mediated mechanisms appear to predominate with newer biologics and immune checkpoint inhibitors, whereas direct cytotoxic injury remains more characteristic of traditional chemotherapeutic agents. This distinction has important therapeutic implications, as immune-mediated injury may respond more favorably to immunosuppressive strategies, while cytotoxic injury often requires early recognition and irreversible drug cessation to prevent progressive fibrosis.

Molecular mechanisms underlying drug-induced pulmonary injury

At the molecular level, drug-induced pulmonary injury is frequently mediated by dysregulated inflammatory signaling pathways. Key cytokines implicated include tumor necrosis

factor-α (TNF-α), interleukin-6 (IL-6), and transforming growth factor-β (TGF-β), which contribute to alveolar inflammation, endothelial injury, and fibrotic remodeling. Immune checkpoint inhibitors may amplify T-cell–mediated lung injury through enhanced immune activation, whereas cytotoxic agents induce oxidative stress and direct epithelial cell damage. Understanding these pathways may facilitate the development of targeted therapeutic strategies.

Current management strategies

Initial management primarily involves discontinuation of the suspected offending agent and supportive care. Systemic corticosteroids are widely used for immune-mediated injury, though the evidence remains largely observational [4,5] (Table 1). Emerging therapeutic approaches include:

- **Biomarker-guided therapy:** Serum markers (e.g., KL-6, SP-D) as well as genomic profiling may enable earlier and more accurate risk stratification and early intervention.
- **Antifibrotic agents:** Pirfenidone and nintedanib, currently approved for idiopathic pulmonary fibrosis, are currently under investigation for drug-induced fibrosis.
- **Immunomodulation:** Tailored regimens for steroid-refractory cases, including agents such as mycophenolate mofetil or rituximab, have demonstrated preliminary promise."

Future directions

Advances in pharmacogenomics and artificial intelligence offer the potential to better predict individual susceptibility to DIPI-related lung injury, including machine-learning models that analyze electronic health records and imaging data to identify early radiographic patterns of drug-induced pneumonitis. Large-scale collaborative registries and robust post-marketing surveillance are essential to refine risk-benefit profiles of newer drugs. Multidisciplinary teams integrating pulmonology, pharmacology, and radiology may enhance patient outcomes through early recognition and protocol-driven management.

Table 1: Common Causative Drugs, Pulmonary Injury Patterns, and Management Approaches in DIPI

Drug class	Example agents	Common injury pattern	General management
Chemotherapeutic agents	Bleomycin, methotrexate	Interstitial pneumonitis, fibrosis	Drug discontinuation, corticosteroids
Immune checkpoint inhibitors	Nivolumab, pembrolizumab	Pneumonitis	Corticosteroids, immunosuppression
Antiarrhythmics	Amiodarone	Organizing pneumonia	Drug withdrawal, steroids
Immunosuppressants	Rituximab	Alveolar hemorrhage	Supportive care, immunomodulation

Practical takeaways for clinicians

Clinicians should maintain a high index of suspicion for DIPI in patients presenting with new respiratory symptoms while receiving high-risk medications. Early drug discontinuation, timely imaging, and prompt initiation of corticosteroids when indicated are critical to improving outcomes. Multidisciplinary collaboration is essential for accurate diagnosis and optimal management.

Conclusion

Drug-induced pulmonary injury represents a growing clinical challenge requiring heightened clinical vigilance. While cessation of the causative drug and corticosteroids form the cornerstone of current therapeutic management, personalized approaches based on mechanistic insights are gradually emerging. Future research should prioritize predictive biomarkers and targeted therapies to reduce the burden of DIPI in an era of rapidly expanding pharmacotherapy.

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