Interstitial Lung Disease in Systemic Lupus Erythematosus: Mechanisms, Diagnosis and Treatment Strategies

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Short Communication

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ABOUT THE STUDY

Interstitial Lung Disease (ILD) is a significant pulmonary complication of Systemic Lupus Erythematosus (SLE), a chronic autoimmune disorder. It refers to a group of lung conditions characterized by inflammation and scarring of the lung interstitium, impairing gas exchange and causing progressive respiratory dysfunction. The presence of ILD in SLE patients is associated with increased morbidity and mortality.

Mechanisms of interstitial lung disease in SLE

Autoimmune inflammation: SLE is characterized by the production of autoantibodies that target various tissues, including the lungs. Immune complexes formed by these autoantibodies deposit in the lung interstitium, triggering inflammatory responses. The release of pro-inflammatory cytokines, such as Tumour Necrosis Factor Alpha (TNF-α) and Interleukin-6 (IL-6), exacerbates the immune response, leading to tissue damage.

Type III hypersensitivity: In SLE, type III hypersensitivity reactions also play a role in lung injury. The deposition of immune complexes in pulmonary tissues leads to complement activation and inflammation, which contributes to the development of ILD.

Fibrosis: Chronic inflammation in the lungs results in fibrosis, a process where normal lung tissue is replaced by scar tissue. Fibrosis leads to the thickening of the alveolar walls, reducing the surface area for gas exchange and resulting in reduced lung function. This scarring may progress over time, leading to irreversible pulmonary damage.

Vascular injury: Pulmonary vasculitis is another contributing factor in SLE-related ILD. Inflammation of the small blood vessels in the lungs can further damage the pulmonary parenchyma, leading to interstitial fibrosis and compromised lung function.

Diagnosis of ILD in SLE

Clinical symptoms: Patients with ILD in SLE often present with progressive may be indicative of ILD when they persist or worsen over time.

Pulmonary Function Tests (PFTs): PFTs are essential in assessing lung function in SLE patients. Restrictive patterns, characterized by decreased Forced Vital Capacity (FVC) and reduced Diffusion capacity of the Lung for Carbon monoxide (DLCO), are commonly seen in ILD. These tests provide valuable information on the severity of lung involvement.

High-Resolution Computed Tomography (HRCT): HRCT is a critical imaging modality for diagnosing ILD in SLE. It can reveal characteristic findings such as ground-glass opacities, reticular patterns and honeycombing, which are indicative of interstitial lung changes.

Bronchoscopy and lung biopsy: In some cases, bronchoscopy with Broncho-Alveolar Lavage (BAL) may be performed to rule out infections or malignancy. A lung biopsy, though invasive, may be necessary to confirm the diagnosis and assess the extent of tissue damage.

Treatment strategies for ILD in SLE

Corticosteroids: Corticosteroids are the mainstay of treatment for ILD in SLE. High doses are typically used during acute flare-ups to control inflammation. However, long-term use is limited due to side effects such as osteoporosis, hypertension and infection.

Immunosuppressive agents: For patients with moderate to severe ILD, immunosuppressive drugs such as azathioprine, mycophenolate mofetil and cyclophosphamide are commonly used. These medications help control autoimmune activity and reduce inflammation in the lungs.

Biologic therapies: Biologic agents like rituximab and tocilizumab have shown promise in managing refractory ILD in SLE. Rituximab targets B cells, while tocilizumab inhibits IL-6, both of which are involved in the inflammatory pathways of ILD.

Oxygen therapy: In patients with significant hypoxemia, supplemental oxygen may be necessary to improve oxygen saturation, especially during physical activity or sleep.

CONCLUSION

Interstitial lung disease in systemic lupus erythematosus is a significant cause of morbidity and mortality, requiring early detection and appropriate management. The mechanisms of ILD in SLE include immune-mediated inflammation, fibrosis and vascular involvement. Diagnosis is based on a combination of clinical evaluation, pulmonary function tests, HRCT imaging and serological markers. Treatment strategies, such as corticosteroids, immunosuppressive agents, biologic therapies and supportive care, aim to reduce inflammation, prevent fibrosis and improve lung function. Timely intervention and close monitoring are essential for improving the quality of life and prognosis of patients with ILD in SLE.

REFERENCES

1. Mageau A, et al. Epidemiology of interstitial lung disease in systemic lupus erythematosus in France: A nation-wide population-based study over 10 years. Resp. 2022;27:630-634.
2. Hannah JR, et al. Pulmonary complications of systemic lupus erythematosus. Inseminars in respiratory and critical care medicine. Semin Respir Crit Care Med. 2019;40:227-234.
3. Kim JS, et al. Thoracic involvement of systemic lupus erythematosus: clinical, pathologic and radiologic findings. J Comput Assist Tomogr. 2000;24:9-18.
4. Richter P, et al. Interstitial Lung Disease in Systemic Lupus Erythematosus and Systemic Sclerosis: How Can We Manage the Challenge? Int J Mol Sci. 2023;24:9388.
5. Yaling CH, et al. Association of interstitial lung disease with clinical characteristics of Chinese patients with systemic lupus erythematosus. Arch Rheumatol. 2020;35:239.
6. Deak M, et al. Non-thromboembolic risk in systemic lupus erythematosus associated with antiphospholipid syndrome. Lupus. 2014;23:913-918.
7. Fenlon HM, et al. High-resolution chest CT in systemic lupus erythematosus. Am J Roentgenol. 1996;166:301-307.
8. Amarnani R, et al. Lupus and the lungs: the assessment and management of pulmonary manifestations of systemic lupus erythematosus. Front Med. 2021;7:610257.
9. Kobe H, et al. Acute onset systemic lupus erythematosus interstitial lung disease: A case report. Respir Med Case Rep. 2021;32:101329.
10. Chen J, et al. Biologics-induced interstitial lung diseases in rheumatic patients: Facts and controversies. Expert Opin Biol Ther. 2017;17:265-283.