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Perspective

A Systemic Lupus Erythematosus and Lung Therapy Study

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DESCRIPTION

Systemic Lupus Erythematosus (SLE) is a complicated autoimmune disease with multiorgan manifestations, including pleuropulmonary involvement (20-90%). Although the precise mechanism of pleuropulmonary involvement in SLE is unknown, systemic type 1 interferons, circulating immune complexes, and neutrophils appear to have crucial functions. Lupus pleuritis, pleural effusion, acute lupus pneumonitis, shrinking lung syndrome, interstitial lung disease, Diffuse Alveolar Haemorrhage (DAH), pulmonary arterial hypertension, and pulmonary embolism are the eight types of pleuropulmonary involvement. The mortality rate for DAH is high (68-75%). Chest X-ray (CXR), Computed Tomography (CT), Pulmonary Function Tests (PFT), bronchoalveolar lavage, biopsy, technetium-99 m hexamethylprophylene amine oxime perfusion scan, and F-fluorodeoxyglucose positron emission tomography are all diagnostic tools for pleuropulmonary involvement in SLE. High-resolution CT, CXR, and PFT are methods for detecting pleuropulmonary involvement in SLE. Specific therapy for pleuropulmonary involvement in SLE are unknown. Immunosuppressive therapies such as corticosteroids and cyclophosphamide, on the other hand, are commonly utilized. Rituximab has also been shown to be effective in three of the eight different types of pleuropulmonary involvement: lupus pleuritis, acute lupus pneumonitis, and shrinking lung syndrome. SLE is a complicated autoimmune illness with multiorgan involvement that might include pulmonary involvement, arthritis, photosensitive rashes, glomerulonephritis, and cytopenia. SLE is a highly variable disease with a poorly understood pathogenesis. The absence of pathognomonic molecular markers or vague constitutional symptoms causes a delay in SLE diagnosis and therapy. SLE is challenging to diagnose due to its complicated clinical features and uncertain pathogenesis. In 2019, the European League against Rheumatism (EULAR)/American College of Rheumatology (ACR) developed new SLE classification criteria, which included one obligatory entry criterion (positive Antinuclear Antibody (ANA) followed by additional weighted criteria grouped into seven clinical (constitutional, hematologic, neuropsychiatric, mucocutaneous, serosal, musculoskeletal, and renal) and three immunologic antiphospholipid antibodies.

The current EULAR/ACR criteria include pleural effusion. Pleuropulmonary symptoms, including pleural effusion, are clinical criteria and are common in SLE. Many SLE patients (50-70%) develop pulmonary problems ranging from asymptomatic pleural effusion to life-threatening alveolar haemorrhage and 4-5% have pulmonary signs as presenting symptoms. Pulmonary symptoms in children are less common than in adults, although they can be serious and fatal SLE complications. Many investigations have found that patients with SLE can have modest or nonexistent pulmonary impairment, indicating a subclinical disease. According to one multiethnic US cohort research (LUMINA XLVIII), 7.6% and 11.6% of patients had irreversible lung impairment 5 and 10 years after SLE diagnosis, respectively aging, pneumonitis, and anti-Rib Nucleoprotein (anti-RNP) antibodies are all associated with the onset of persistent lung illness.

Patients with pleuropulmonary symptoms showed significantly poorer survival rates than those without (82.2% *vs* 95.6%, p=0.030) in another retrospective cohort study. Hence, SLE-related pleuropulmonary symptoms in children and adults with SLE are potentially fatal. An in-depth study of SLE-related pleuropulmonary symptoms will aid in early identification and prevent the disease from worsening. As a result, the purpose of this study was to discuss the epidemiology, pathogenesis, diagnosis, and therapy of SLE-related lung disorders, such as infection and drug-induced lung injury.

Both clinical and laboratory settings, geographic and racial distributions appear to influence the prevalence and severity of SLE. Individuals of African or Asian descent had around two to three times the incidence and prevalence rates of other ethnicities in the United States and the United Kingdom. SLE is more common among Asians, Black Americans, Caribbeans, and Hispanic Americans than in Caucasians. Asian and African-descent Europeans have similar results, although Africa has the lowest rate of SLE. Sex is the most major risk factor for SLE, with 90% of SLE patients being female in most studies. Hormones play a role in the onset of SLE *via* an unknown mechanism. Although sex hormones have negligible effects on children, the women-to-men ratio of patients with SLE is 3:1, but it ranges from 7 to 15:1 in adults, especially in women of childbearing age. Because postmenopausal women have lower levels of sex harmones,

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the women-to-men ratio in elderly individuals is reduced to 8:1. SLE can occur at any age, with roughly 20% of cases detected during the first 20 years.

It is extremely rare before the age of five, but its prevalence rises during the first decade. Adult SLE prevalence and incidence rates are significantly greater. The women-to-men ratio and disease activity are lower in late-onset SLE patients, although organ damage accumulation is greater, leading to higher mortality. A considerable number of SLE patients have pulmonary involvement. The most common signs of lung illnesses are pleurisy, diffuse alveolar haemorrhage, shrinking lung syndrome, interstitial lung disease, and pulmonary arterial hypertension. Some patients develop life-threatening consequences, such as pulmonary bleeding. As a result, lung involvement in SLE patients should be evaluated and treated as soon as possible. CXR, HRCT, and PFT are suggested as diagnostic tests. Physicians and patients are less aware of the respiratory complications of SLE. Nevertheless, precise diagnostic criteria for SLE lung involvement remain elusive. As a result, more emphasis should be placed on active surveillance and care of SLE pulmonary symptoms.