Section: General Medicine

Original Research Article

# PULMONARY MANIFESTATIONS IN PATIENTS OF SYSTEMIC LUPUS ERYTHYMATOSUS

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## ABSTRACT

**Background:** Systemic lupus erythematosus (SLE) is a chronic autoimmune disorder affecting multiple organ systems, with pulmonary manifestations contributing significantly to morbidity and mortality. Pulmonary complications such as pleuritis, interstitial lung disease (ILD), pulmonary hypertension, and pleural effusion are common and may occur independently of disease activity. This study aimed to assess the pulmonary manifestations in patients with SLE and to analyze their correlation with disease duration. **Materials and Methods:** A cross-sectional study was conducted in the Department of Medicine, RIMS Imphal, from January 2023 to March 2025. A total of 131 SLE patients (≥18 years), diagnosed according to the Modified SLICC criteria, were included. Patients with pre-existing pulmonary conditions or pregnancy were excluded. Data collection involved clinical history, physical examination, laboratory tests, pulmonary function tests (PFTs), and high-resolution CT (HRCT) thorax. Statistical analyses were performed using SPSS version 26, with significance set at $p < 0.05$. **Result:** Of the 131 participants, 99.2% were female, with the majority in the 31–40 years age group. Renal involvement was observed in 38.17% of patients, and all tested positive for ANA and anti-dsDNA. Restrictive patterns on PFTs were seen in 61.8% of cases. Radiological findings included pulmonary consolidation (most frequent), pleural effusion, bronchial wall thickening, pulmonary infiltrates, and ground-glass opacities. While pulmonary consolidation was commonly observed across all disease durations, pleural effusion and dyspnea showed no significant association with disease duration. However, pulmonary edema was more frequent within the first five years of illness, and the difference was statistically significant. Overall, no consistent correlation was found between disease duration and most pulmonary manifestations. **Conclusion:** Pulmonary involvement is highly prevalent in SLE, with consolidation and restrictive lung patterns being the most frequent findings. The majority of manifestations did not correlate with disease duration, suggesting that pulmonary complications may arise at any stage of illness. Early and routine pulmonary screening, irrespective of disease duration, is crucial for timely detection and management. Further longitudinal studies are required to clarify the pathophysiological mechanisms and optimize clinical guidelines for pulmonary involvement in SLE.

## INTRODUCTION

Systemic lupus erythematosus (SLE) is a chronic, multi-system autoimmune disease characterized by the production of autoantibodies and immune complex formation, leading to inflammation and damage in various organs. The disease can affect multiple systems, including the skin, kidneys, cardiovascular system, and the central nervous system. SLE primarily affects women of childbearing age, with a female-to-male ratio of approximately 9:1. The incidence varies globally, but it is higher in populations of African, Hispanic, and Asian descent. Although the exact etiology remains unclear, genetic, environmental, and hormonal factors are believed to contribute to disease pathogenesis. The clinical manifestations of SLE can be diverse and range from mild to life-threatening, often complicating early diagnosis and management.[1] Among the various organ systems affected in SLE, the pulmonary system is frequently involved, with manifestations ranging from mild, subclinical conditions to severe, life-threatening complications. Pulmonary involvement is noted in approximately 50% of SLE patients during the course of the disease, and it is associated with increased morbidity and mortality. Common pulmonary manifestations in SLE include pleuritis, interstitial lung disease (ILD), pulmonary hypertension (PH), and pulmonary embolism. These conditions can lead to significant clinical impairment and impact the patient's quality of life. The inflammatory processes underlying pulmonary involvement in SLE are complex, involving both the innate and adaptive immune responses, with an emphasis on vascular inflammation, autoantibody production, and immune complex deposition.[2] Pleuritis, which is inflammation of the pleura, is one of the most common pulmonary manifestations in SLE, often presenting with pleuritic chest pain and respiratory distress. The prevalence of pleuritis varies but can affect up to 40% of SLE patients. It may present with or without accompanying pleural effusion, which can exacerbate respiratory symptoms. Another significant manifestation is interstitial lung disease (ILD), which encompasses a variety of pulmonary disorders characterized by inflammation and fibrosis of the interstitial spaces of the lungs. ILD in SLE can present as diffuse alveolar damage, organizing pneumonia, or non-specific interstitial pneumonia.[3] The pathogenesis of ILD in SLE involves immune-mediated damage to lung tissue, resulting in fibrosis and impaired gas exchange. Pulmonary hypertension, which occurs due to the narrowing of pulmonary arteries and subsequent increased pulmonary vascular resistance, is another important manifestation. It is associated with poorer prognosis and is often seen in patients with long-standing disease, particularly those with concurrent renal or cardiac involvement.[4] The mechanisms behind pulmonary manifestations in SLE are multifactorial. The autoimmune response plays a central role, with the production of autoantibodies, including antinuclear antibodies (ANA), anti-dsDNA antibodies, and antiphospholipid antibodies, which can mediate tissue damage through immune complex deposition. This leads to endothelial injury, inflammation, and, in some cases, thrombosis, further contributing to pulmonary dysfunction. Additionally, chronic inflammation, a hallmark of SLE, can lead to vascular damage and fibrosis, exacerbating pulmonary involvement. The severity of pulmonary manifestations in SLE is often linked to disease activity, with patients experiencing more severe pulmonary involvement during periods of disease flares. However, some pulmonary manifestations may occur independently of disease activity, which underscores the complexity of managing these conditions in SLE.[5] The clinical relevance of early diagnosis and management of pulmonary complications in SLE cannot be overstated. Pulmonary manifestations can significantly impact the quality of lifeand are associated with high rates of morbidity and mortality, particularly when complications such as pulmonary fibrosis or pulmonary hypertension develop. Early identification of these complications is critical to prevent irreversible damage and improve clinical outcomes. Pulmonary complications in SLE often require multidisciplinary management, involving rheumatologists, pulmonologists, and cardiologists. The development of effective screening methods, including pulmonary function tests, high-resolution CT scans, and echocardiography, has helped in the early detection of these complications, although there is still a need for more precise and reliable diagnostic tools.[6] Despite the advancements, there are significant gaps in our understanding of the underlying pathophysiology and optimal management strategies. The rationale for conducting this crosssectional study lies in the need for further investigation into the prevalence, types, and clinical significance of pulmonary manifestations in SLE patients. Although pulmonary involvement is common, the heterogeneity of symptoms and the complexity of pathophysiological mechanisms pose challenges in understanding the full scope of pulmonary complications. This study aims to provide a comprehensive analysis of pulmonary manifestations in SLE, which could inform better diagnostic and therapeutic strategies, ultimately improving patient outcomes.

The primary objective of this study was to assess the pulmonary manifestations in patients diagnosed with Systemic Lupus Erythematosus (SLE) and to analyze the correlation between pulmonary findings and the duration of the disease. Given that pulmonary complications are one of the leading causes of morbidity and mortality in SLE patients, this study sought to identify patterns of pulmonary involvement, determine the significance of specific pulmonary symptoms, and compare the findings with previously established studies on SLE-related


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pulmonary manifestations. By understanding the pulmonary impact of SLE, clinicians can improve diagnostic accuracy and tailor treatment strategies accordingly. Furthermore, identifying key pulmonary symptoms early can aid in reducing disease progression and associated complications. The study also sought to evaluate how different radiological and serological markers influence pulmonary manifestations in SLE patients. A thorough assessment of these factors is essential in formulating better clinical guidelines.

## MATERIALS AND METHODS

**Aim**

The study aims to bridge the knowledge gap regarding the interplay between disease duration and lung involvement.

**Materials and Methods**

**Ethical statement**

The cross-sectional study was carried out in accordance of the Declaration of Helsinki 1964 and its later amendments. Written consent was taken from all participants. All study procedure was approved by the Research Ethics Board of the hospital.

**Study Design and Settings**

A cross-sectional study was conducted from January 2023 to March 2025 in the Medicine Department RIMS, Imphal among patients above 18 years of age diagnosed with Systemic Lupus Erythematous according to the Modified SLICC Criteria. The study was initiated after acquiring ethical approval (No. A/206/REB-Comm(SP)/RIMS/2015/1013/44/2023) from the research ethics board of the institute. Patients with known bronchial asthma, chronic obstructive pulmonary diasease, occupational lung disease, sarcoidosis and pregnant women were excluded from the study. Consecutive sampling technique was used to reach the sample size of 131, calculated from a study by Alamoudi SB et al.[7] A detailed clinical history and physical examination, with particular emphasis on disease activity and duration, were conducted for all participants, along with routine laboratory investigations, relevant serological tests, chest radiography, pulmonary function tests (PFTs), and high-resolution computed tomography (HRCT) of the thorax. The diagnosis of systemic lupus erythematosus (SLE) was established according to the Modified Systemic Lupus International Collaborating Clinics (SLICC) criteria. Data were collected using a structured proforma, supplemented with information extracted from patient case records in the department. Demographic details, including age, sex, occupation, and place of residence, were recorded. Following informed consent, HRCT thorax findings and PFT results were systematically documented for further analysis.[8]

**Inclusion Criteria**

Patients were classified as having systemic lupus erythematosus (SLE) according to the Modified SLICC criteria. Clinical criteria included: (1) acute cutaneous lupus, (2) chronic cutaneous lupus, (3) oral ulcers (palatal), (4) non-scarring alopecia (diffuse thinning or hair fragility with broken hairs), (5) synovitis involving two or more joints, defined by swelling/effusion or tenderness with ≥30 minutes of morning stiffness, (6) serositis, (7) renal involvement, (8) neurologic involvement, (9) haemolytic anaemia, (10) leukopenia (<4000/mm³ at least once), and (11) thrombocytopenia (<100,000/mm³ at least once).

Immunological criteria included: (1) antinuclear antibodies (ANA) above the laboratory reference range, (2) anti-dsDNA antibodies above the laboratory reference range (except ELISA, which required levels ≥2× reference), (3) anti-Smith antibody positivity, (4) antiphospholipid antibody positivity, (5) low complement levels (C3/C4), and (6) a positive direct Coombs test in the absence of haemolytic anaemia.

For classification, patients were required to meet at least four criteria, including one clinical and one immunological, or have lupus nephritis as the sole clinical criterion in the presence of ANA or anti-dsDNA antibodies.

**Statistical Analysis**

Collected data were compiled and analyzed using SPSS (IBM, version 26). Results were expressed as mean, standard deviation, and percentages. Statistical significance was tested using $\chi^2$ or Fisher's exact test, with a p-value $< 0.05$ considered significant.

## RESULTS

In the current study, one hundred and thirty one (131) patients with Systemic Lupus Erythematosus (SLE) who fulfilled the inclusion and exclusion criteria were evaluated. Majority (99.2%) of the respondents were female. As shown in table 1, majority of the patients fall within the 31-40 age group (25.95%), followed by patients of 51 to 60 years of age group (23.6%). Only 7.6% of the participants were in the age group of 11 to 20 years of age.

Table 2 presents the other baseline characteristics of patients with pulmonary manifestations in Systemic Lupus Erythematosus (SLE). Acute cutaneous lupus (34.35%) and chronic cutaneous lupus (32.06%) were observed in a subset of patients. Oral ulcers (29.01%), non-scarring alopecia (25.95%), synovitis (24.43%), and serositis (25.19%) were also noted. Renal involvement was present in 38.17% of patients. All patients were positive for antinuclear antibody (ANA) and anti-dsDNA, while 40.46% showed anti-Smith antibody positivity. Additionally, 48.09% had low complement levels (C3/C4), indicating immune dysregulation. These findings highlight the varied clinical spectrum of SLE, emphasizing its systemic nature and the frequent occurrence of renal and serological abnormalities in affected patients. The duration of illness was less than 6 months in 34 (25.9%) patients, 6 months to 1 year in 34 (25.9%) patients, 1 year to 5 years in 34 (25.9%) patients and


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> 5 years in 29 (22.3%) patients as shown in Fig 1. In this study, around two-third (61.8%) of the patients had Restrictive pattern on PFT, while around one-third (38.2%) of the patients had constrictive pattern on PFT as shown in Fig 2.

As shown in Table 3, Pleuritic Chest Pain (PCP) was observed in a small subset of SLE patients across all illness durations, with a slightly higher occurrence in those with longer disease duration (1–5 years and 6 months–1 year: 3 cases each). There was no significant association between occurrence of pleuritic chest pain and duration of illness in SLE patients. This suggests that PCP occurrence is not dependent on the duration of SLE and may be influenced by other factors such as immunosuppressive therapy, disease severity, or individual susceptibility rather than disease duration alone. Similarly, dyspnea was observed across different illness durations, with a slightly higher occurrence in patients with SLE for more than 5 years (6 cases) but the difference was not significant. This implies that dyspnea in SLE may be influenced by multiple factors, such as pulmonary involvement (e.g., lupus pneumonitis, pleuritis, interstitial lung disease), disease activity, or medication effects, rather than just the duration of the disease. Other symptoms like cough, hemoptysis were also seen in few patients with SLE as shown in table 3 and these symptoms were not associated with duration of illness. pleural effusion was observed in patients across different illness durations, with the highest number of cases in the 6 months–1 year group (4 cases), followed by <6 months (3 cases), 1–5 years (2 cases), and >5 years (1 case). No significant association was seen between duration of illness and pleural effusion in SLE patients. This suggests that pleural effusion occurs independently of disease duration and may be influenced more by active disease, serositis, systemic inflammation, or other complications rather than the length of time a patient has had SLE.

Pulmonary consolidation was commonly observed across all illness durations, with the highest occurrence in the 1–5 years group (18 cases), followed by <6 months (14 cases), 6 months–1 year (13 cases), and >5 years (12 cases). No statistically significant association between duration of illness and pulmonary consolidation in SLE patients. Bronchial wall thickening and pulmonary infiltrates were also observed in equal distribution among all illness duration groups as shown in table 3.

Pulmonary cavity and pleural thickening were observed in few patients across different illness durations, with no significant association between pulmonary cavity and pleural thickening with disease duration. Pulmonary edema was more frequently observed in patients within the first five years of illness rather than those with longer disease duration and the difference was found to be statistically significant. Atelectasis, Lymphadenopathy, Interlobar Sepatal thickening, ground glass opacity and bronchiectasis were seen across all illness duration groups with no clear pattern linked to disease duration as shown in table 3.

**Table 1: Baseline Characteristics of Patients with Pulmonary Manifestations in SLE (N=131)**

| Characteristics | | No. of participants, n | Percentages (%) |
|---|---|---|---|
| Gender | Male | 1 | 0.8 |
| | Female | 130 | 99.2 |
| Age in years | 11 to 20 years | 10 | 7.6 |
| | 21 to 30 years | 29 | 22.1 |
| | 31 to 40 years | 34 | 25.9 |
| | 41 to 50 years | 27 | 20.6 |
| | 51 to 60 years | 31 | 23.6 |

**Table 2: Baseline Characteristics of Patients with Pulmonary Manifestations in SLE (N=131)**

| Characteristics | | No. of participants, n | Percentages (%) |
|---|---|---|---|
| Acute Cutaneous Lupus (ACL) Status | Absent | 86 | 65.6 |
| | Present | 45 | 34.4 |
| Chronic Cutaneous Lupus (ACL) Status | Absent | 89 | 67.9 |
| | Present | 42 | 32.1 |
| Oral Ulcers (Palate) involvement | Absent | 93 | 70.9 |
| | Present | 38 | 29.1 |
| Non-Scarring Alopecia | Absent | 97 | 74.1 |
| | Present | 34 | 25.9 |
| Synovitis | Absent | 99 | 75.6 |
| | Present | 32 | 24.4 |
| Serositis | Absent | 98 | 74.8 |
| | Present | 33 | 25.2 |
| Renal Involvement | Absent | 81 | 61.8 |
| | Present | 50 | 38.2 |
| Antinuclear Antibody (ANA) Positivity | Yes | 131 | 100 |
| Anti-dsDNA positively | Yes | 131 | 100 |
| Anti-Sm Antibody Positivity | No | 78 | 59.5 |
| | Yes | 53 | 40.5 |
| Low Complement Levels (C3/C4) | No | 68 | 51.9 |
| | Yes | 63 | 48.1 |


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**Table 3: Association between signs and symptoms of SLE patients and duration of illness (N=131)**

| Sl. no. | Characteristics | Duration of Illness, n(%) | | | | P value |
|---|---|---|---|---|---|---|
| | | < 6 months | 6 months to 1 year | 1 year to 5 years | > 5 years | |
| 1. | Pleuritic Chest Pain | 2 (5.8) | 3 (8.8) | 3 (8.8) | 2 (6.9) | 0.959 |
| 2. | Dyspnea | 2 (5.8) | 2 (5.8) | 1 (2.9) | 6 (20.7) | 0.056 |
| 3. | Cough | 3 (8.8) | 2 (5.8) | 4 (11.8) | 1 (3.4) | 0.624 |
| 4. | Hemoptysis | 0 (0.0) | 2 (5.8) | 6 (17.6) | 4 (13.8) | 0.056 |
| 5. | Pleural Effusion | 3 (8.8) | 4 (11.8) | 2 (5.8) | 1 (3.4) | 0.624 |
| 6. | Pulmonary Consolidation | 14 (41.2) | 13 (38.2) | 18 (52.9) | 12 (41.4) | 0.627 |
| 7. | Bronchial wall thickening | 7 (20.6) | 8 (23.5) | 7 (20.6) | 9 (31.0) | 0.746 |
| 8. | Pulmonary infiltrates | 6 (17.6) | 12 (35.3) | 12 (35.3) | 5 (17.2) | 0.150 |
| 9. | Pulmonary cavity | 1 (2.9) | 1 (2.9) | 3 (8.8) | 0 (0.0) | 0.306 |
| 10. | Pulmonary edema | 2 (5.8) | 4 (11.7) | 8 (23.5) | 0 (0.0) | 0.017 |
| 11. | Pleural thickening | 1 (2.9) | 1 (2.9) | 0 (0.0) | 1 (3.4) | 0.777 |
| 12. | Atelectasis | 11 (32.3) | 15 (44.1) | 11 (32.3) | 13 (44.8) | 0.566 |
| 13. | Lymphadenopathy | 10 (29.4) | 14 (41.2) | 9 (26.5) | 15 (51.7) | 0.143 |
| 14. | Interlobar Septal Thickening | 12 (35.3) | 15 (44.1) | 14 (41.2) | 12 (41.4) | 0.900 |
| 15. | Ground Glass Opacity | 8 (23.5) | 10 (29.4) | 12 (35.3) | 11 (37.9) | 0.603 |
| 16. | Bronchiectasis | 10 (29.4) | 13 (38.2) | 12 (35.3) | 11 (37.9) | 0.866 |

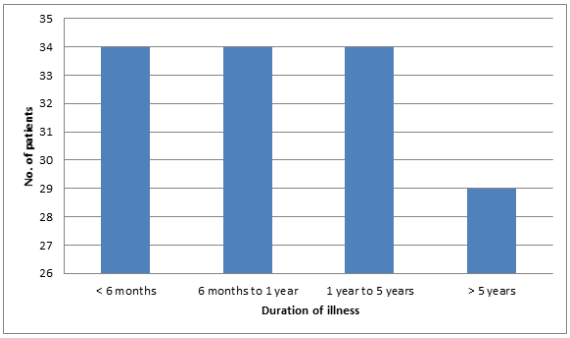


**Figure 1: Duration of Illness in SLE Patients (N=131)**

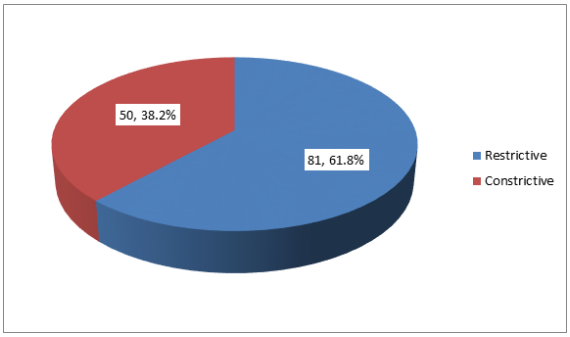


**Figure 2: Distribution of the patients by pulmonary function test (N=131)**

## DISCUSSION

### Autoimmune Markers and Serological Findings

#### ANA and Anti-dsDNA Positivity

All patients in the present study tested positive for ANA and anti-dsDNA antibodies. This observation is supported by the study by Siegel and Sammaritano (2024), which found that ANA is nearly 100% sensitive for SLE diagnosis, while anti-dsDNA positivity correlates strongly with disease activity and nephritis.[9] The universal presence of these markers in the study population reaffirms their diagnostic and prognostic significance in SLE. ANA positivity remains a cornerstone in diagnosing SLE and its systemic involvement, particularly in pulmonary pathology. The correlation between anti-dsDNA levels and disease activity suggests its potential utility in monitoring treatment responses. Elevated anti-dsDNA levels have also been associated with lupus nephritis, which indirectly contributes to pulmonary edema. Regular assessment of these serological markers may help tailor treatment strategies and predict pulmonary involvement. The findings validate the inclusion of ANA and anti-dsDNA testing in routine diagnostic protocols for SLE patients.

#### Anti-Sm and Low Complement Levels (C3/C4)

The study found that 40.46% of patients were positive for anti-Sm antibodies, while 48.09% had low complement levels. Similar results were reported by Di Bartolomeo et al. (2021), who found that low complement levels are indicative of active lupus and are often associated with lupus nephritis and pulmonary involvement.[10] Low complement levels, particularly C3 and C4, serve as markers of immune complex deposition, a process implicated in pulmonary damage in SLE. The presence of anti-Sm antibodies, although less common, is highly specific for SLE and correlates with more severe disease manifestations. The study's findings highlight the necessity for complement level monitoring to assess disease progression and treatment effectiveness. Reduced complement levels may serve as an early indicator of impending flare-ups, particularly those affecting pulmonary tissues. Integrating complement testing with radiological findings may improve predictive accuracy in diagnosing SLE-associated lung complications.

### Pulmonary Symptoms and Radiological Findings

#### Dyspnea and Cough

Dyspnea was significantly associated with disease duration in this study (p=0.05). This finding is supported by Brady et al. (2021), who reported that dyspnea is a primary symptom of lupus pneumonitis, interstitial lung disease, and pleuritis, often worsening with disease progression.[11] Similarly, Shin et al. (2022) found that persistent dyspnea in SLE patients is a strong predictor of underlying pulmonary pathology, requiring early intervention.[12] The progression of dyspnea in SLE patients may be


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attributed to ongoing inflammation, leading to fibrosis and restrictive lung disease over time. Chronic hypoxia resulting from these conditions can further exacerbate disease severity and lead to cardiovascular complications. Given these findings, clinicians should incorporate routine pulmonary function tests and imaging studies to assess lung involvement. Early recognition of dyspnea can aid in the timely administration of immunosuppressive therapy, potentially mitigating disease progression. Additionally, lifestyle modifications such as pulmonary rehabilitation and oxygen therapy may improve the quality of life in affected patients.

**Pulmonary Edema and Hemoptysis**

Pulmonary edema (p=0.017) and hemoptysis (p=0.05) were significantly associated with disease duration. These findings align with De Zorzi et al. (2022), who reported that pulmonary edema in SLE often results from lupus-associated vasculopathy, cardiac dysfunction, or nephrotic syndrome.[13] Hemoptysis, often indicative of diffuse alveolar hemorrhage (DAH), was also found to increase with disease duration, consistent with findings by Zamani et al. (2021).[14] The underlying mechanisms leading to pulmonary edema in SLE may involve increased vascular permeability due to immune complex deposition. Long-standing nephropathy in lupus patients may also contribute to fluid overload, exacerbating pulmonary congestion. The presence of hemoptysis suggests significant alveolar damage, necessitating urgent medical intervention. Given the severity of these manifestations, early echocardiographic evaluation and close monitoring of renal function are recommended. Therapeutic strategies such as corticosteroids and plasma exchange may be considered in severe cases to reduce inflammation and prevent irreversible lung damage.

**Ground Glass Opacity (GGO) and Interstitial Lung Disease (ILD)**

Ground glass opacity (GGO) was observed across all disease stages but did not show statistical significance with disease duration. Amarnani et al. (2021) similarly noted that GGO is an early radiological sign of ILD in SLE but may not always correlate with disease duration.[15] Brady et al. (2021) reported that GGO is a key finding in lupus pneumonitis and early ILD and requires careful monitoring to prevent progression to fibrosis.[11] The presence of GGO may indicate an active inflammatory process within the alveolar structures, which, if left untreated, may lead to fibrotic lung changes. Since ILD remains a major concern in SLE patients, high-resolution CT scans should be routinely used for early detection. The absence of statistical significance suggests that ILD can develop at any disease stage, reinforcing the need for lifelong monitoring. Treatment options, including corticosteroids and antifibrotic agents, should be explored to slow down disease progression. Given its clinical implications, regular pulmonary assessments and patient education regarding symptom recognition are essential.

**Pleural Effusion and Bronchiectasis**

Pleural effusion and bronchiectasis were not significantly associated with disease duration in this study. However, Palmucci et al. (2022) found that pleural effusion is a frequent manifestation in SLE, primarily due to serositis, and is often recurrent in patients with active disease.[16] Similarly, Baisya et al. (2021) identified bronchiectasis as a rare but potential sequela of chronic pulmonary inflammation in SLE.[17] Pleural involvement in SLE is typically associated with persistent inflammation, leading to pleural thickening and fibrosis. Bronchiectasis, on the other hand, may develop as a long-term complication due to repeated infections and chronic inflammation. The lack of statistical significance does not exclude the clinical relevance of these manifestations, warranting periodic chest imaging. Given their impact on respiratory function, patients with recurrent pleural effusion should undergo thoracentesis and pleural biopsy when necessary. Bronchiectasis management should focus on airway clearance techniques and antibiotic prophylaxis to prevent exacerbations.

**Pulmonary Cavity**

Pulmonary cavities were rare and not statistically significant in this study. Brady et al. (2021) described pulmonary cavitation in SLE as an uncommon but severe complication often linked to infections, necrotizing pneumonia, or vasculitis.[11] Pulmonary cavitation is a radiological finding characterized by air-filled spaces within lung tissue, which can arise from infectious or autoimmune causes. In SLE, vasculitic damage to pulmonary vessels can lead to necrosis and subsequent cavitation, posing a risk for secondary infections such as fungal or bacterial superinfections. Patients with pulmonary cavities should undergo further evaluation with bronchoscopy and microbiological analysis to rule out opportunistic infections, particularly in immunosuppressed individuals. Although the prevalence of cavitary lesions is low in SLE, their presence often signifies a severe disease course requiring aggressive immunosuppressive and antimicrobial therapy. Additionally, distinguishing lupus-related cavitation from tuberculosis or malignancies is crucial, as the management strategies for these conditions differ significantly.

Given the overlap between infectious and inflammatory causes of consolidation, SLE patients presenting with this radiological feature should undergo thorough clinical evaluation, including inflammatory markers, autoimmune panels, and cultures to guide appropriate management. Regular imaging follow-ups are also necessary to monitor resolution or progression of consolidation and to differentiate it from fibrotic lung disease. The role of immunomodulatory therapy in preventing recurrent episodes of air space consolidation should be further investigated to improve patient outcomes.

**Pulmonary function tests (PFTs)**


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Pulmonary function tests revealed a restrictive pattern in 61.8% of the patients, while 38.2% had a constrictive pattern, further emphasizing the significant impact of SLE on pulmonary function in our study. Systemic Lupus Erythematosus (SLE) is recognized as being linked to restrictive lung diseases.

**Clinical Implications**

The findings of this study emphasize the significant burden of pulmonary manifestations in SLE patients and the need for early detection and intervention. The presence of dyspnea, pulmonary edema, hemoptysis, and ground glass opacity suggests that pulmonary complications can develop at any stage of the disease, reinforcing the need for regular pulmonary evaluations. Early identification of these manifestations allows for timely treatment with immunosuppressive agents, reducing the risk of disease progression and long-term complications. The presence of autoimmune markers such as ACL, anti-dsDNA, and low complement levels further highlights the role of systemic inflammation in pulmonary pathology, suggesting that aggressive immunosuppression may be necessary in high-risk patients. Moreover, the study underscores the importance of distinguishing SLE-related pulmonary complications from infections and other autoimmune lung diseases to ensure targeted treatment strategies.

**Future Implications**

Routine pulmonary screening should be an integral part of the clinical management of patients with systemic lupus erythematosus (SLE). Regular chest radiographs, pulmonary function tests, and high-resolution CT scans are essential for detecting early lung involvement before irreversible damage occurs. Early intervention is crucial, as prompt recognition and treatment of respiratory symptoms such as dyspnea, hemoptysis, and pulmonary edema with corticosteroids and immunosuppressive agents can help prevent disease progression. A multidisciplinary approach involving rheumatologists, pulmonologists, and radiologists is recommended to ensure accurate diagnosis and effective management of pulmonary complications. In addition, patient education plays a vital role; individuals should be counseled to recognize early respiratory symptoms and seek timely medical attention to reduce the risk of severe complications. Structured smoking cessation programs should also be integrated into routine care, given the negative impact of smoking on pulmonary outcomes in SLE patients. From a research perspective, further studies are warranted to evaluate the role of biologic therapies, antifibrotic agents, and novel immunosuppressive regimens in preventing and managing lung damage. Future investigations should also focus on identifying reliable biomarkers to predict pulmonary complications and guide individualized treatment strategies. Advances in imaging, including artificial intelligence-based diagnostic tools, may further improve early detection and classification of pulmonary abnormalities. Longitudinal studies exploring genetic and environmental risk factors could provide insights into disease mechanisms and support the development of personalized therapeutic approaches. Additionally, interventions such as pulmonary rehabilitation and lifestyle modifications should be studied to improve long-term outcomes and quality of life for patients with SLE-related pulmonary involvement.

**Limitations**

Despite the significant findings, this study has certain limitations. The cross-sectional design limits the ability to establish causal relationships between disease duration and pulmonary manifestations. A larger sample size with long-term follow-up is required to confirm these associations and evaluate disease progression over time. Additionally, the study was conducted in a single center, which may limit its generalizability to broader populations. The reliance on radiological findings without histopathological confirmation may also introduce diagnostic variability. Furthermore, the impact of treatment regimens on pulmonary outcomes was not extensively analyzed, highlighting the need for future studies focusing on therapeutic response. Potential confounding factors such as medication use, comorbidities, and environmental exposures were not comprehensively assessed, which may have influenced the observed findings.

## CONCLUSION

The findings of this study emphasize the significant burden of pulmonary manifestations in Systemic Lupus Erythematosus (SLE) and their association with disease duration. Pulmonary edema was identified as the most common pulmonary symptoms, with statistical significance observed in their correlation with disease duration. The presence of autoimmune markers, including ANA, anti-dsDNA, and low complement levels, suggests that systemic inflammation plays a critical role in the development of pulmonary complications. Radiological findings, including ground glass opacity, pleural effusion, and interstitial lung disease, reinforce the need for routine pulmonary screening in SLE patients to facilitate early diagnosis and timely intervention.

In conclusion, this study highlights the high prevalence of pulmonary manifestations in SLE patients and underscores the importance of early recognition and intervention. By implementing routine screening, adopting a multidisciplinary approach, and optimizing treatment strategies, clinicians can mitigate the impact of pulmonary complications and improve overall patient outcomes. Future advancements in diagnostics and therapeutics hold promise for reducing disease burden and enhancing the long-term prognosis of SLE patients with pulmonary involvement.


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