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SHRINKING LUNG SYNDROME IN SYSTEMIC LUPUS ERYTHEMATOSUS-CLINICAL AND THERAPEUTIC CHALLENGES

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ABSTRACT

Background: Systemic lupus erythematosus (SLE) is a chronic autoimmune disease characterized by multisystem involvement. One of its rare but clinically significant complications is the shrinking lung syndrome (SLS), which affects less than 1% of patients and presents diagnostic and therapeutic challenges.
Aim: This paper aims to present a comprehensive overview of shrinking lung syndrome as a rare manifestation of SLE, with a focus on its pathophysiology, diagnostic difficulties, and current therapeutic approaches.
Materials and Methods: A narrative review was conducted based on selected scientific publications indexed in PubMed, Scopus, and Google Scholar. The inclusion criteria included articles describing the pathophysiology, clinical features, diagnosis, and treatment of SLS in the context of SLE.
Results: SLS is characterized by progressive dyspnea, diaphragmatic elevation, and restrictive defects on pulmonary function testing. The exact pathogenesis remains unclear but involves neuromuscular dysfunction, diaphragmatic weakness, pleural involvement, and potential autoantibody-mediated mechanisms. Diagnosis is challenging due to the absence of specific markers and requires exclusion of other causes. Treatment primarily includes corticosteroids and immunosuppressants, with adjunctive respiratory rehabilitation. Despite therapy, pulmonary restriction may persist.
Conclusions: SLS remains an under-recognized and underdiagnosed complication of SLE. Increased clinical awareness and early diagnostic interventions are key to preventing long-term respiratory impairment. Future research is needed to establish standardized diagnostic protocols and treatment guidelines.

KEYWORDS

Systemic Lupus Erythematosus, Shrinking Lung Syndrome, Diaphragmatic Dysfunction, Autoimmune Disease, Respiratory Failure, Immunosuppressive Therapy, SLE Complications

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1.Introduction

Systemic lupus erythematosus (SLE) is a chronic disease with a complex epidemiology, developing on an autoimmune basis, which leads to dysfunction of many systems and organs. Women have the disease more often than men. Genetic, epigenetic, environmental, hormonal, cellular and humoral type response abnormalities are involved in the pathogenesis of the disease. Lupus can cause general symptoms such as weakness, fatigue, subfebrile states and fever, and weight loss. The clinical picture of the disease includes involvement of the skin, mucous membranes, changes in the musculoskeletal system, changes in the kidneys, lungs, circulatory system, nervous system, hematological symptoms and changes in the gastrointestinal system. [1]

Shrinking lung syndrome (SLS) is a rare manifestation of systemic lupus erythematosus, with an incidence of 1%. SLS is characterized by dyspnea, restrictive abnormalities on pulmonary function tests, reduced lung volumes on imaging studies, and bilateral diaphragmatic weakness. [2]

The purpose of this article is to present a rare but serious complication of systemic lupus erythematosus. The authors review the diagnostic difficulties and complexity of treating this syndrome in the context of an autoimmune disease. The article also aims to highlight the need to individualize therapy and further research into effective treatments.

2 Materials and Methods.

2.1 Data sources.

To gather current information on shrinking lung syndrome (SLS) in systemic lupus erythematosus (SLE), a review of the scientific literature available in PubMed, Scopus and Web of Science databases was conducted. The literature search included publications in English and Polish published between 1965 and 2024. Relevant keywords and search phrases were used, such as: "shrinking lung syndrome," "systemic lupus erythematosus," "SLS in SLE," "autoimmune pulmonary complications," "dyspnea in lupus," "respiratory dysfunction in SLE," and their logical combinations.

2.2 Criteria for selection of publications.

Original articles, case reports, systematic reviews and meta-analyses that contained clinical, diagnostic or therapeutic data on patients diagnosed with shrinking lung syndrome in the course of SLE were included in the analysis. Partial-text publications, papers that did not contain explicit confirmation of the diagnosis of SLE, and articles unrelated to the topic of autoimmune diseases were excluded. In addition, literature items cited in selected papers were included if they contained relevant clinical or pathophysiological information.

3 Research results.

3.1 Shrinking lung syndrome (SLS) - definition and history, epidemiology, criteria.

Shrinking lung syndrome (SLS) was first described in 1965 by Hoffbrand and Beck. SLS is a fairly rare complication of SLE, which can occur at any time during the course of the disease [3] its estimated incidence is < 1%. The incidence was much higher in women than in men, with a ratio of 10:1 [4, 5].

The diagnosis of SLS is based mainly on the exclusion of other causes of dyspnea and pleuritic chest pain in patients with SLE and on the analysis of the results of additional tests. The key examination is a classic chest X-ray [3]. The most common symptom observed is unilateral or bilateral diaphragmatic elevation, a hallmark of the condition. A decrease in lung volume is also often found. Rarer symptoms include linear atherectasia, pleural effusion and pleural thickening [6].

High-resolution CT has proven more sensitive than X-ray in detecting benign pleural effusions, thickening or atherectasia. It allows exclusion of the presence of lung parenchymal disease or vascular pathology [7].

3.2 Pathophysiology of SLS.

The etiology of SLS syndrome remains unknown; however, various potential pathogenetic mechanisms are under consideration:

3.2.1 Neuromuscular disorders.

Neuromuscular dysfunction is one of the main pathophysiological mechanisms considered in the context of SLS in SLE. Weakening of respiratory muscles, particularly the diaphragm, leads to lung volume limitation and impaired respiratory mechanics, which is clinically manifested by dyspnea and restrictive type of abnormalities in pulmonary function tests.

In a study by Laroche et al (1989), reduced diaphragm contraction strength was demonstrated in patients with SLS, indicating a significant contribution from neuromuscular dysfunction. Possible mechanisms include both primary myopathy associated with lupus and secondary damage due to chronic inflammation, malnutrition or drugs (e.g., corticosteroids) [8].

3.2.2 Respiratory muscle inflammation.

Inflammatory myopathy, especially in the respiratory muscles, may be directly related to the pathogenesis of SLS. In cases of lupus myositis, the diaphragm and intercostal muscles may become inflamed, leading to weakness and reduced thoracic mobility.

Pérez-de-Llano et al (2011) described a case of SLS caused by lupus myopathy, in which a muscle biopsy showed features of inflammation. Immunosuppressive treatment (methylprednisolone, azathioprine) resulted in improvement of respiratory parameters, confirming the reversibility of this mechanism with proper therapy [9].

3.2.3 Diaphragmatic nerve injury.

Bilateral or unilateral damage to the phrenic nerve has also been identified as a potential cause of SLS. The mechanism may be neurogenic (inflammatory neuropathy in SLE) or mechanical (e.g., secondary to pleuritis leading to compression or irritation of the nerve).

Hardy et al (2001) described a case of a patient with SLE and bilateral phrenic nerve palsy, resulting in severe respiratory impairment. Electromyography (EMG) and nerve conduction studies can be helpful in identifying this mechanism in patients with suspected SLS.

In addition, the work of Henderson et al (2013) suggests that chronic pleuritis can induce secondary phrenic nerve damage, further complicating the clinical picture and highlighting the multifactorial etiology of SLS [10, 11].

3.2.4 Pleural dysfunction.

Pleuritis causes inhibition of deep inspiration by nerve reflexes and pain resulting in chronic pulmonary hypoinflation, which in predisposed patients leads to parenchymal remodeling, which reduces lung compliance. Disturbed compliance worsens hypoinflation, initiating a positive feedback loop that helps explain the gradual progression of SLS. The defect is primarily functional, so one would expect the patient's ventilatory drive to limit further respiratory deterioration, which explains the low mortality rate of SLS despite its alarming clinical presentation [12].

3.2.5 Involvement of autoantibodies.

Positive anti-SSa/Ro antibodies may be associated with both SLS and myositis, and it has been suggested that myositis contributes to diaphragmatic dysfunction in some patients [13].

3.3 Clinical picture.

The clinical manifestation of SLS can sometimes be nonspecific and ambiguous, often resulting in misdiagnoses or significant delays in making a proper diagnosis.

The most common and also the most significant clinical manifestation of SLS is dyspnea, occurring in 70-100% of patients [1,2,3]. It initially takes the form of gradually increasing exertional dyspnea, developing over weeks or months. As the disease progresses, resting dyspnea and orthopnoë may develop. Characteristically, these symptoms occur despite the absence of visible parenchymal changes on imaging studies. The underlying mechanism of dyspnea is a reduction in Total Lung Capacity (TLC, Forced Vital Capacity - FVC), which is an important diagnostic criterion in differentiating SLS from other respiratory diseases [3, 28, 30].

Pleuritic pain is also a common and clinically significant symptom, which is observed in about 60-76% of patients [4,5]. It is stabbing in nature and may be unilateral or bilateral, increasing with deep inspiration. Importantly, in some patients, the pain may precede the onset of dyspnea, which is an important clinical clue that facilitates the early diagnosis of SLS [31].

Reduced exercise tolerance is a frequently associated, albeit nonspecific, symptom. It can precede other clinical symptoms and is sometimes misinterpreted as the result of an exacerbation of systemic activity of the underlying disease, such as anemia in the course of SLE [32].

Dry cough is less common and is described in about 15-30% of patients. Its presence more often reflects pleural irritation than an active inflammatory process in the lung parenchyma [29].

Despite significant dyspnea and impaired lung function parameters, no typical auscultatory signs such as wheezing, rales or crackles are found on physical examination. Moreover, the absence of fever, leukocytosis and sputum distinguishes SLS from infectious respiratory diseases. On physical examination, muted respiratory murmurs may be present at the base of the lungs, which is associated with limited diaphragmatic mobility and reduced ventilation of the peripheral parts of the lungs. Such discrete clinical physical presentation with markedly increased subjective complaints make SLS a difficult disease to diagnose and often result in significant diagnostic delay.

3.4 Supporting research.

Spirometry in SLS can show a significant decrease in FVC and reduced inspiratory and expiratory pressures, suggesting respiratory muscle weakness [14].

Chest X-ray shows reduced lung volume and atelectatic changes in the parasternal segments [15].

High-resolution computed tomography (HRCT) has greater sensitivity in detecting small pleural effusions, pleural thickening and atherectasia, but its main diagnostic role is to exclude pulmonary parenchymal lesions or vascular pathologies [7].

Electromyography (EMG) of the diaphragm can be a valuable diagnostic tool. In a retrospective study of patients with SLE and suspected SLS, EMG was performed in 11 patients - half of whom showed diaphragmatic paresis or paralysis. The study's authors emphasize the usefulness of EMG in the differential diagnosis of SLS, especially in cases with inconclusive results from other tests [16].

MRI as a diagnostic test in SLS may prove particularly useful in assessing diaphragmatic function. A 2016 article based on an observational study describes a case of a patient with SLE who underwent dynamic thoracic MRI. The image showed limited diaphragmatic mobility, which supported the diagnosis of SLE. The authors emphasize that MRI can be a valuable diagnostic tool, especially when other imaging studies do not provide clear results

Gasometry can be a useful tool in assessing respiratory function in patients with suspected SLS. In a case report by Guleria et al. (2014), concerning a patient with SLE and scleroderma overlap syndrome who was diagnosed with SLS, gasometric analysis revealed features of primary respiratory alkalosis (pH: 7.53; pCO_2: 29.3 mmHg; HCO_3-: 19.8 mmol/L) accompanied by mild hypoxemia (pO_2: 74.9 mmHg). The authors emphasized that such a picture could be a consequence of weakened respiratory muscles, including the diaphragm, which is typical of the pathophysiological mechanism of SLS [17].

3.5 Differential diagnosis.

The differential diagnosis should exclude other possible causes of dyspnea, pleuritic pain in a patient with SLE, including pneumonia and pleurisy, pericarditis, pulmonary embolism, interstitial lung disease, chronic obstructive pulmonary disease, heart failure, pulmonary hypertension, musculoskeletal disorders and anxiety disorders.

The differential causes of diaphragmatic elevation include pulmonary atherectasia, pulmonary hypoplasia, lobectomy, pneumonectomy, diaphragmatic nerve palsy, stroke on the other side of the lung, subdiaphragmatic abscess, distended stomach or large intestine, or abdominal tumors.

The differential diagnosis should also consider causes of restrictive disorders such as pulmonary fibrosis, pleural effusions, kyphoscoliosis, obesity and neuromuscular disorders [3].

3.6 Importance of shrinking lung syndrome in systemic lupus erythematosus.

SLS is a rare complication of SLE, occurring in less than 1% of patients. Clinical observations suggest that there is no clear correlation between the presence of SLS and the activity of the underlying disease. In most cases, SLS develops in patients with already diagnosed SLE, but, although rare, it can also be the first symptom of the disease.

An example is a 2024 case report published in *Case Report: Diaphragm Ultrasound Reveals Shrinking Lung Syndrome*, which presents a 25-year-old woman in whom SLS was the first manifestation of lupus. The diagnosis of SLE was made only after limited diaphragmatic mobility was found and other causes of dyspnea were ruled out [18].

3.7 Treatment.

Currently, there is no clearly established treatment regimen for SLS. In clinical practice, it is often recommended to start therapy with glucocorticosteroids at a dose of 0.5-1 mg/kg/day, used alone or in combination with immunosuppressive drugs. The most commonly used immunosuppressive drugs include cyclophosphamide, rituximab, azathioprine, hydroxychloroquine, methotrexate, mycophenolate mofetil and belimumab. Glucocorticosteroids are generally effective, but chronic restriction often persists despite their use [19].

When treatment with glucocorticosteroids alone is not satisfactory, other immunosuppressive drugs, such as methotrexate, azathioprine, cyclophosphamide, Based on recent scientific reports reviewing cases of SLS in pediatric patients, it can be concluded that in situations where there is no complete response to standard immunosuppressive therapy, it is worth considering the implementation of complementary treatment, including the use of rituximab [18, 20].

Supportive treatment, including pulmonary rehabilitation, is an important part of the therapeutic management of SLS, making it possible to improve lung expansion. Pulmonary rehabilitation plays a key role in improving exercise tolerance and reducing the severity of dyspnea, highlighting the importance of structured physiotherapy programs in the comprehensive therapy of SLS. Maintenance of stable pulmonary function test results and cessation of nocturnal desaturation indicate that the combination of non-invasive ventilation (NIV) and rehabilitation can effectively halt disease progression and contribute to improved patient function [20, 21].

3.8 Clinical and therapeutic challenges.

3.8.1 Diagnostic difficulties associated with low awareness and rare SLS.

SLS is a rare, often under-recognized complication of SLE, with an estimated incidence of about 0.5-1.5%. The clinical picture, including exertional dyspnea, chest pain, restricted diaphragmatic mobility and reduced lung volume, is nonspecific and can easily be misinterpreted as a manifestation of other diseases such as interstitial lung disease, pleuritis or pulmonary hypertension leading to diagnostic delays [19].

An additional problem is the low awareness of the syndrome, with the result that SLS is sometimes overlooked in the differential diagnosis in patients with SLE, which also causes diagnostic delays. The lack of specific biomarkers and the limited availability of advanced functional tests of the respiratory muscles, such as measurement of maximum inspiratory pressure (MIP) and expiratory pressure (MEP), further complicate the diagnostic process [22].

3.8.2 Lack of treatment standards - need for individualized approach.

Currently, there are no standardized therapeutic guidelines for the treatment of SLS, which poses a significant clinical challenge. Due to the limited number of studies and the low proportion of diagnosed cases, the available therapeutic data are mainly from case reports and small observational studies. The most common treatment is glucocorticosteroids, which in many cases lead to rapid clinical improvement. In situations of resistance or intolerance, the addition of immunosuppressive drugs such as azathioprine, methotrexate, mycophenolate mofetil or cyclophosphamide is recommended. In isolated reports, efficacy has also been demonstrated for rituximab and belimumab. Because of the variability in response to treatment, the choice of therapy should be tailored individually, taking into account the severity of symptoms, systemic activity of SLE and the patient's tolerance to immunosuppression [23].

3.8.3 Need for prospective studies and case registries.

Currently, most of the knowledge about SLS in SLE comes from case reports and retrospective analyses. The literature shows that tens to over a hundred cases of SLS have been described in registries to date. The registries have made it possible to establish the frequency (~1%), clinical characteristics (e.g., elevated diaphragm, restrictive pattern) and initial treatment trends (eightfold higher remission rate with muscle rehabilitation, glucocorticosteroids and rituximab) [3].

Registries often include small groups (e.g., 7-15 patients). This approach limits the ability to objectively compare therapies (e.g., corticosteroids vs. rituximab) and identify risk factors, which can result in diagnostic delay and differentiation between other respiratory disease [24].

Advantages of a prospective approach include the ability to develop objective and reproducible diagnostic criteria for SLS and to monitor its dynamics over time, including progression of clinical symptoms and changes in lung functional parameters such as FVC or TLC. Prospective studies would also allow standardization of diagnostic algorithms - from diaphragmatic ultrasonography to contrast-enhanced chest CT - and standardization of monitoring methods, which could translate into shorter time to diagnosis and improved treatment outcomes [25].

3.8.4 Evaluate treatment effectiveness and long-term outcomes.

Treatment to date (corticosteroids, theophylline, diaphragmatic inflammation) is mainly based on single reports. Prospective comparative studies (randomized or observational) are needed to test the efficacy of, for example, prednisone vs. prednisone + rituximab and assess the durability of respiratory function, quality of life and complications [26].

3.8.5 Support for clinical guideline development.

Currently, SLS has no internationally recognized diagnostic or therapeutic standards. Data from prospective studies and multicenter patient registries could provide the basis for the development of standardized clinical guidelines, modeled on EULAR or ACR standards, which would allow standardization of the diagnostic and therapeutic management of SLE patients with SLS manifestations [27].

4 Summary.

Despite its rarity, SLS should be considered in the differential diagnosis of patients with SLE, especially in the presence of dyspnea, pleuritic chest pain or diaphragmatic elevation. Although awareness of this condition has increased in recent years, SLS remains an underestimated complication that, contrary to popular belief, may even be an early manifestation of SLE. Maintaining high diagnostic vigilance in such cases is crucial. Therefore, the development of clear guidelines for rapid differential diagnosis and a review of available therapeutic options are important steps toward improving the diagnosis and treatment of this syndrome [5, 25].

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