Journal of Clinical & Experimental Pathology

Christopher, J Clin Exp Pathol 2022, 12:5

Commentary Open Access

Significance of Interstitial Lung Disease in Myositis

Lisa Christopher*

Department of Molecular Genetics, Northwestern University Feinberg School of Medicine, Chicago, USA

Corresponding author: Lisa Christopher, Department of Molecular Genetics, Northwestern University Feinberg School of Medicine, Chicago, USA, E-mail: christopherlisa@northwetern.edu

Received: 21-Oct-2022, Manuscript No. JCEP-22-82959; **Editor assigned:** 24-Oct-2022, PreQC No. JCEP-22-82959 (PQ); **Reviewed:** 09-Nov-2022, QC No. JCEP-22-82959; **Revised:** 17-Nov-2022, Manuscript No. JCEP-22-82959 (R); **Published:** 24-Nov-2022, DOI:10.4172/2161-0681.22.12.423.

Citation: Christopher L (2022) Significance of Interstitial Lung Disease in Myositis. J Clin Exp Pathol. 12: 423.

About the Study

With an incidence ranging from 19.9% to 42.6%, Interstitial Lung Disease (ILD) is a frequent consequence of Polymyositis (PM) and Dermatomyositis (DM). In 7.2% to 37.5% of patients, ILD is the presenting symptom and has been documented to occur before clinical myopathy symptoms. ILD can still arise at any stage of myositis, with a median duration to emergence of 16.9-18 months, even in the face of aggressive immunosuppression. A research found that up to a third of patients experienced a later deterioration of Pulmonary Function Tests (PFTs), which is not unusual for ILD that is initially stable or improving on immunosuppression.

ILD, while not being included in the recently approved ACR/EULAR diagnostic criteria for Idiopathic Inflammatory Myopathies (IIM), is the primary cause of hospitalization and death in individuals with PM/DM, with reported mortality rates ranging from 7.5% to 44%. As a result, treatment options must frequently revolve around lung-specific medicines. Myositis-ILD patients exhibit unique diagnostic and therapeutic problems that are best addressed through multidisciplinary collaborations with skilled rheumatologists and pulmonologists.

However, neither rheumatologists nor pulmonologists have agreed upon a comprehensive set of categorization criteria due to the heterogeneity of both myositis and ILD. The initial foundation for categorizing the IIMs was provided in 1975 by the Bohan and Peter Classification. Since then, several categorization and diagnostic criteria, including the inclusion of myositis-specific antibodies, have proposed revisions to those first put out by Bohan and Peter. The European League Against Rheumatism and the American College of Rheumatology both accepted a validated categorization criteria for myositis in 2017. Even though these criteria represented a major improvement over those from 1975, ILD was not included in the criteria and only anti-Jo1 of the recognized Myositis-Specific Antibodies (MSAs) included.

In this study presented a new categorization approach for IIM based on clinical findings and the addition of a larger MSA panel. In their study, IIM could be classified into four broad groups, one of which was made up mostly of patients with anti-synthetase syndrome and positive for either the anti-Jo1 or anti-PL-7 antibody. Every patient in this cluster was said to have pulmonary involvement, and the authors concluded that including MSAs into myositis categorization looked to be more advantageous than morphologic characteristics derived from muscle biopsy.

Myositis specific antibodies and ILD

With an average incidence of 20% and 29%, respectively, in this disease group, antisynthetase antibodies are the most prevalent autoantibodies observed in individuals with either DM or PM. Eight anti-synthetase antibodies against the aminoacyl-tRNA synthetase enzyme are currently identified (ARS-Abs). Even though the term "anti-synthetase syndrome" has historically been used to describe patients who have tested positive for one of these antibodies, some recent literature challenges this usage. In fact, it is not unusual for clinical features thought to be indicative of the so-called "syndrome" to be minimal or absent at different stages of the illness. Additionally, even with the presence of antibodies unrelated to ARS myositis, the syndrome's distinctive symptoms could still be present. When ILD in myositis is not detected or treated early, it might have a negative consequence.

In a newly diagnosed myositis patient, physicians should have a high degree of suspicion for concomitant pulmonary involvement and a low threshold for doing serial PFTs or CT imaging, especially if autoantibodies with a documented ILD link are present. Patients with established myositis-ILD benefit from co-management by pulmonary and rheumatology teams due to their clinical complexity. While immunosuppressive medications used to treat myositis-ILD are comparable to those used to treat isolated myositis, in quickly decompensating individuals with underlying lung involvement, a more aggressive treatment strategy with higher dose regimens or combination therapy is more usual. Solid data supporting the use of specific immunosuppressive drugs is currently absent, and the significance of anti-fibrotic treatment in patients with progressive illness is still being explored. The clinical studies necessary to improve this discipline will necessitate collaboration between rheumatologists and pulmonologists.