CT characterization of idiopathic inflammatory myopathy-associated interstitial lung disease: frontiers to strengthen diagnostic accuracy

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We read with great interest the article by Oliveira Filho et al. entitled "Clinical, functional, and computed tomographic characterization of idiopathic inflammatory myopathy-associated interstitial lung disease: a retrospective cohort study."[1] The study provides valuable insights into the epidemiological, clinical, and imaging features of this rare condition. Nevertheless, we would like to highlight several methodological and interpretive points that we believe could refine the conclusions.

First, although lung biopsy is often unnecessary in cases with clear clinicoradiological concordance such as nonspecific interstitial pneumonia patterns, nearly half of the patients in the study presented with an indeterminate pattern. In such cases, histopathological confirmation is strongly recommended in order to improve diagnostic accuracy.[2] The absence of biopsy in this large subgroup limits pathological characterization and may affect treatment decisions.

Second, the cohort was predominantly composed of antisynthetase syndrome patients, with other idiopathic inflammatory myopathy (IIM) subtypes being underrepresented. This limits the generalizability of the findings to the wider IIM population. Moreover, no significant association was reported between antibody profiles and functional or respiratory outcomes. This could have provided additional prognostic information.

Third, the time frame for assessing changes in FVC was not specified. In progressive fibrosing interstitial lung disease, a ≥ 10% annual decline is considered clinically meaningful and prognostically relevant. Without standardized intervals and clear thresholds, interpretation of FVC change becomes challenging—especially since FVC is effort-dependent and may decline due to muscular weakness rather than interstitial disease progression.[3] The use of DL_{CO} could have provided a more reliable measure of interstitial involvement.

Fourth, the diagnosis of pulmonary hypertension relied solely on echocardiography and radiological findings. The gold standard, right heart catheterization, was not performed, which may have resulted in underdiagnosis.[4] The discussion statement "We found no cases of pulmonary arterial hypertension" is therefore methodologically uncertain and likely reflects the limitations of noninvasive assessment in patients with fibrotic lung disease.

Fifth, it is unclear how HRCT scans were reviewed. The methods section suggests that they were evaluated by two of the authors of the study, but a consensus approach or interobserver agreement was not mentioned. Such details are important to ensure reproducibility.

Finally, although the authors reported improvement in symptoms such as dyspnea and cough, this appears to have been based on patient self-report without validated scales. Given that treatment was individualized, the relationship between therapy and symptom or functional improvement remains difficult to assess.

We commend the authors for assembling a well-characterized cohort and for providing long-term follow-up data. We hope that our considerations will encourage further research to strengthen diagnostic accuracy, standardize functional assessment, and refine prognostic evaluation in IIM-associated interstitial lung disease.

AUTHOR CONTRIBUTIONS

KBA: literature review and writing of the original draft. AME: conceptualization, project administration, and manuscript review.

CONFLICTS OF INTEREST

None declared.

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Authors' Reply

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We thank the authors for their thoughtful correspondence designated "CT characterization of idiopathic inflammatory myopathy-associated interstitial lung disease: frontiers to strengthen diagnostic accuracy," which includes comments regarding our manuscript "Clinical, functional, and computed tomographic characterization of idiopathic inflammatory myopathy-associated interstitial lung disease: a retrospective cohort study," as well as for the opportunity to clarify several methodological points. We herein explain the comments made by the authors point-by-point.

1. Lung biopsy in connective tissue disease (CTD)-related interstitial lung disease (ILD) is infrequently performed at our center because, in routine practice, histology usually does not modify management, especially when the diagnosis of a CTD is confirmed, even when there is an indeterminate pattern on CT scans. This approach—also acknowledged among the limitations of our study—reflects contemporary practice in autoimmune ILD.[1,2]
2. We agree with the authors that antisynthetase syndrome is over-represented in our cohort. This bias occurred due to convenience sampling in a referral ILD clinic, and inclusion criteria were restricted to participants with data available for longitudinal analysis in this retrospective design—points we have explicitly recognized as a selection bias limiting generalizability of the findings to the wider idiopathic inflammatory myopathy (IIM)-related ILD group. We did not evaluate the association of antibody profile with clinical and functional outcomes due to missing data regarding these serum exams.
3. We agree with the authors that the absence of DL_{CO} and respiratory muscle strength metrics (e.g., PI_{max}/PE_{max}) may influence interpretation of FVC. We decided not to apply the International Myositis Assessment & Clinical Studies (IMACS) or the current ATS/ERS recommendations that define clinically meaningful (e.g., ≥10%) annual decline, because annual, standardized spirometry results were not consistently available due to the retrospective nature of the study. We therefore reported change between the first and last available tests and recognize the effort-dependence of FVC as a limitation.
4. We agree that right-heart catheterization remains the gold standard for confirming pulmonary hypertension (PH). In our cohort, however, this invasive test was not performed due to the clinical judgment of the health care team, mainly because it was considered unlikely to change management decisions or was limited by the functional status of the patients. This reflects the inherent constraints of a retrospective study and underscores some of its methodological limitations. In line with the 2022 ESC/ERS pulmonary hypertension guideline,[3] our surveillance used recommended screening tools—transthoracic echocardiography and CT measures (e.g., main pulmonary artery diameter/PA:A ratio). Although suspected PH was noted by echocardiography in a subset of patients, we found no features suggesting group-1 PH (pulmonary arterial hypertension; PAH); most suspicions were compatible with PH associated with parenchymal lung disease (group 3).
5. HRCT examinations were reviewed by two thoracic radiologists with expertise in ILD, and, in cases of disagreement, adjudicated by an ILD pulmonologist—thereby ensuring a consensus-based final interpretation.
6. Symptom data necessarily relied on patient reports because of the retrospective chart-review design. Validated scales to assess symptoms were not routinely applied in our center. Dyspnea was graded using the mMRC scale, which was the instrument routinely applied in our service.

We appreciate the colleagues' engagement and believe these clarifications strengthen the interpretation and context of our findings.

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