HHS Public Access

Author manuscript

Eur Respir J. Author manuscript; available in PMC 2017 July 12.

Published in final edited form as:

Eur Respir J. 2016 December ; 48(6): 1813–1814. doi:10.1183/13993003.01841-2016.

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We appreciate the letter of correspondence by A.S. Jee and colleagues highlighting the need to standardise the rheumatologic classification criteria utilised in the diagnosis of a defined connective tissue disease (CTD) when evaluating patients with interstitial lung disease (ILD) for autoimmune features and CTD. This was prompted by our recent publication in the *European Respiratory Journal* [1]. A.S. Jee and colleagues note that the interstitial pneumonia with autoimmune features (IPAF) cohort in our study contained only one subject with an anti-tRNA synthetase antibody and speculate that patients at our centre with positive antisynthetase antibodies and ILD were diagnosed with an antisynthetase syndrome rather than IPAF [2]. This observation prompted A.S. Jee and colleagues to highlight the numerous criteria that exist for the diagnosis of the idiopathic inflammatory myopathies: polymyositis, dermatomyositis and antisynthetase syndrome and propose that these entities be defined uniformly in the evaluation of patients with ILD. This approach would more precisely distinguish IPAF patients from those with CTD-ILD with potential improvement in the accuracy and prognostic ability of the IPAF criteria.

As noted by A.S. Jee and colleagues, for patients in our ILD registry with an anti-tRNA synthetase antibody, multidisciplinary evaluation yielded a clinical diagnosis of antisynthetase syndrome in all but one patient. An additional patient had evidence of myositis with elevated serum aldolase and creatine kinase levels with a positive anti-Ku antibody, but met IPAF criteria through the clinical (Raynaud's) and morphologic domain (interstitial lymphoid aggregates with germinal centres on surgical lung biopsy) because the anti-Ku antibody is not part of the IPAF criteria. At our institution, our rheumatology colleagues have a heightened awareness of occult CTD presentations in patients with ILD and together we often discern subtle physical findings, such as mild mechanics hands. This more sensitive assignment of a diagnosis of an idiopathic inflammatory myositis in our patients aligns with our practice of aggressively treating ILD in patients with a "myositis" phenotype because of the possibility that ILD in this setting may respond to immunosuppressive treatment and may progress rapidly without such therapy [3, 4].

An additional point to consider in the evaluation of the IPAF criteria is centre-specific practice patterns in serologic evaluation. Prior to the publication of the IPAF criteria, we had

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Conflict of interest: Disclosures can be found alongside this article at erj.ersjournals.com

not routinely sent a full myositis panel on all ILD patients, electing to send only the anti-Jo-1 antibody except in patients where an inflammatory myositis was suspected. Our practice is now evolving with one author (J.M. Oldham) sending a full myositis panel and anti-melanoma differentiation-associated gene 5 antibody in all patients with nonspecific interstitial pneumonitis (NSIP) and the others (M.E. Strek and R. Vij) assessing these antibodies in most ILD patients, even those with usual interstitial pneumonia (UIP) morphology with positive results in a small minority of patients. None of us have yet to incorporate the anti-PM-Scl antibody into our clinical practice, which is an antibody we have not seen our rheumatology colleagues use and is a test that is more difficult for us to perform as it must be sent elsewhere for assay.

Finally, A.S. Jee and colleagues call for uniformity can be interpreted more broadly in the implementation of the rest of the IPAF criteria including decisions about what constitutes diffuse lymphoplasmacytic infiltrates or the presence of multi-compartment thoracic disease [5]. In our study, when determining whether ILD patients met IPAF multi-compartment criteria, we excluded patients with a history of tobacco use from evaluation for intrinsic airways disease, since we could not be certain airway involvement would be “unexplained” in this setting. This decision will need to be revisited as additional centres assess the IPAF criteria in their ILD cohorts.

While the IPAF research criteria are an important first step in identifying features of autoimmunity that might affect prognosis or impact treatment, we believe that there remains great heterogeneity within patients that meet criteria for IPAF, as our study demonstrated. For example, a young African–American non-smoking woman with a positive antisynthetase antibody and otherwise unexplained NSIP and organising pneumonia may have a different ILD phenotype, prognosis and response to treatment than an older Caucasian cigarette smoking man with a positive rheumatoid factor and UIP on surgical lung biopsy who meets IPAF criteria through either a diffuse lymphoplasmacytic infiltrate or unexplained multi-compartment involvement of the airways, pulmonary vasculature or pleural or pericardial abnormalities. In addition, as many patients with NSIP and organising pneumonia are treated with immunosuppressive therapy if there is any suggestion of an autoimmune phenotype, the performance of the IPAF criteria in patients with UIP and unclassifiable ILD may be most important of all [6].

In summary, we agree with A.S. Jee and colleagues that evaluation of the patient with interstitial pneumonitis requires a multidisciplinary collaboration including rheumatology, with uniformity and standardisation in CTD definitions when applying the IPAF criteria. We are excited and inspired by the recognition, research and dialogue the formulation and publication of the IPAF criteria has generated. We look forward to the day when all patients will have a validated assessment of the contribution of autoimmunity to their ILD with a resulting diagnosis that accurately reflects their prognosis and response to treatment.

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