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Correspondence to: Leandro G. Fritscher, MD, Avenida Ipiranga, 6690/501, Porto Alegre, RS, Brazil; e-mail: leandro.fritscher@pucrs.br

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A Clue to Diagnosing Connective Tissue Disease-Associated Interstitial Lung Disease

To the Editor:

In a recent issue of CHEST (August 2010), Fischer and coworkers discussed the dilemmas surrounding the classification of a patient with interstitial pneumonia (IP) whose clinical features suggested an associated connective tissue disease (CTD) but did not provide a clear diagnosis of CTD-associated interstitial lung disease (ILD) on the basis of current rheumatologic classification systems. Undeniably, current rheumatologic classification schemes are limiting because they do not allow CTD designation when IP is the lone manifestation. We agree with the authors that the detection of occult CTD in patients presenting with IP is optimized by multidisciplinary collaboration. It is very difficult to differentiate idiopathic pulmonary fibrosis from CTD-ILD before the appearance of their systematic manifestations. However, several autoantibodies have been identified as being of diagnostic significance for CTD and may serve as clues for a new CTD-ILD classification.

We recently reported an elderly woman who presented with organizing pneumonia; anticyclic citrullinated peptide (anti-CCP) antibody positivity was the first manifestation of rheumatoid arthritis (RA). Although she did not exhibit articular symptoms initially, representative RA manifestations developed 8 months later. Thus, anti-CCP antibody positivity may be a good indicator for RA-ILD diagnosis. Anti-CCP antibodies are reportedly the best predictors of RA activity; they can be detected very early in the disease and have been reported to predict erosive RA development. ILD as the first presentation of CTDs, particularly in RA, is rare; therefore, the examination of these antibodies may be important for the differential diagnosis of CTD-ILD.

We also agree with the authors that specific antibodies are integral in CTD-ILD assessment, and practitioners should apply certain indicators in addition to antinuclear antibody and rheumatoid factor for more effective screening for CTD. In particular, anti-Scl-70, anti-tRNA synthetase antibodies (eg, Jo-1, PL-7, and PL-12), anti-Ro (SS-A), antinucleosome protein, and anti-CCP antibodies are highly specific to CTDs. The selection of the autoantibodies is critical for CTD-ILD diagnosis. The disease-specific antibodies should be examined extensively and carefully.

Although the symptoms and extrapulmonary manifestations are not always specific for CTDs, an initial basic examination is important. The diagnostic and therapeutic outcomes are the ultimate determinants for the differential diagnosis and the further refinement of actual diagnosis. Nonetheless, several autoantibodies specific for CTDs are currently the strongest marker of the diseases and should be used wisely and actively for the early diagnosis and correction of the diagnosis of CTD-ILD.

Shinji Teramoto, MD, FCCP
Tokyo, Japan
Kosaku Komiya, MD
Oita, Japan
Shunsuke Akashi, MD
Masahiro Kawashima, MD
Tokyo, Japan

Affiliations: From the Department of Respiratory Medicine (Drs Teramoto, Akashi, and Kawashima), National Hospital Organization, Tokyo National Hospital; and the Department of Respiratory Medicine (Dr Komiya), Oita University Hospital.

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Correspondence to: Shinji Teramoto, MD, FCCP, Department of Respiratory Medicine, National Hospital Organization, Tokyo National Hospital, 3-1-1 Takeeda, Kiyose, Tokyo, Japan; e-mail: shinjit-ty@umin.ac.jp

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REFERENCES