Anti-Jo1 Antibody in Polymyositis/dermatomyositis Is Still Closely Associated with Lung rather than Joints

To the Editor:

We read with interest the paper by Klein, *et al*[1] containing detailed data about arthritis in patients with idiopathic inflammatory myopathy (IIM). The authors reported that arthritis was a common feature of IIM that appeared not only at the onset of disease, but also might precede muscular manifestation and occur at any time. Especially noteworthy, 27 out of 29 anti-Jo1–positive patients (93.1%) had arthritis, and this finding led authors to confirm a strong association between arthritis and anti-Jo1. Traditionally, anti-Jo1 is known to be associated with interstitial pulmonary disease and polymyositis (PM)/dermatomyositis (DM), often manifesting the pulmonary symptom. It had occasionally been reported in arthritis in patients with PM/DM, but there were not enough occurrences to treat the relationship with anti-Jo1. In that context, the authors' 93.1% was an exceptional result considerably distinguished from previous data.

We investigated 23 patients with PM/DM found to have anti-Jo1 using the double immunodiffusion (DID) method. We reviewed medical records retrospectively, including age, sex, diagnosis, disease duration, and results of autoimmune target test. Simple chest radiographs and chest high-resolution computed tomography (when examined) were reviewed for lung involvement. Joint involvement was defined when any radiologic lesion was found around the joints, including soft tissue swelling at any time during the followup period. PM/DM was diagnosed by the criteria of Bohan and Peter[2]. The control group for comparison consisted of 28 patients with PM/DM who had not been found to have anti-Jo1 using DID. Our study was approved by our institutional review board.

We ascertained a substantial number of joint symptoms in patients with PM/DM (13/23, 56.5%), but did not find a strong association between anti-Jo1 and arthritis ($p > 0.05$; Table 1). The correlation of anti-Jo1 with interstitial pulmonary diseases was reconfirmed as already known (20/23, 87.0%, $p < 0.05$). Not surprisingly, both involvement of lung and joints was observed in the majority of patients with PM/DM, and patients with anti-Jo1 showed significantly higher extramuscular manifestation (lung and/or joints) than did controls (22/23, 95.7%, $p = 0.007$).

Table 1. Distribution of lung and joint involvement according to positivity of anti-Jo1 antibodies in patients with PM/DM. Anti-Jo1 was detected by double immunodiffusion method (IT-ENA, ImmunoThink Co.) and reaffirmed by the autoimmune target test (IT-AIT, ImmunoThink Co.) with specific immunofluorescence pattern (cytoplasmic fine granular). Values are n (%) unless otherwise specified.

Characteristics	Patients	
	With Anti-Jo1, n = 23	Without Anti-Jo1, n = 28
Involved organs*		
Lung + joints	11 (47.8)	8 (28.6)
Lung only	9 (39.1)	9 (32.1)
Joints only	2 (8.7)	1 (3.6)
None	1 (4.3)	10 (35.7)
Disease duration, yrs, mean ± SD	7.39 ± 5.4	8.8 ± 2.9
Age, yrs, mean ± SD	47.9 ± 9.4	42.4 ± 14.7
Sex, f/m	18/5	24/4

* Lung involvement regardless of arthritis was 20 (87.0%) and 17 (60.7%) in patients with and without anti-Jo1, respectively ($p = 0.037$, chi-square test). Joint involvement regardless of lung lesion was 13 (56.5%) and 9 (32.1%) in patients with and without anti-Jo1, respectively ($p = 0.080$, chi-square test). PM: polymyositis; DM: dermatomyositis.

Klein, *et al* reported lung involvement only in whole patients with IIM (37/106, 34.9%), so they were unable to estimate how much lung involvement was in anti-Jo1–positive patients and compare the incidence with other results including ours. We could not interpret whether the cohort reflected the known lung correlation with anti-Jo1.

These differences may have resulted because the patient groups had varying IIM characteristics, or because of ethnic difference. But we want to put a higher priority on the methodological difference rather than previously described reasons. The authors' methods to identify anti-Jo1 were a commercialized line immunoassay (LIA), Western blot, and in-house–made 35S radioimmunoprecipitation, and all of these methods are more sensitive than DID. Using these methods may cause 93% joints involvement, which is different from our results and other studies. (Considering the difference of sensitivity between methods, even if all anti-Jo1–negative patients in our data are supposed to be positive, lung involvement is still markedly dominant from joints involvement.)

At this point, we need to remember that the method that found the anti-Jo1 in patients first was DID[3]. Antiextractable nuclear antigen test has been steadily developed since DID was introduced, but at the same time, its status as a marker antibody positioned by DID has been continuously threatened by other sensitive methods more recently developed[4]. As for radioimmunoprecipitation, it is recognized as the gold standard but is not acceptable for routine use because it is time-consuming and cumbersome to process, and the recent trend is to avoid radioisotopes. LIA or ELISA are highly sensitive but low in specificity and make it difficult to interpret clinical correlations[5]. In contrast, the DID method is antibody dose–dependent and able to detect antibodies over a significant level, and therefore it is clearly helpful in patient diagnosis even with lower sensitivity. In addition, it is important to remember that almost all the clinical significance of autoantibodies has been established by DID.

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