Response to: 'Anti-Ro52 autoantibodies are associated with interstitial lung disease and more severe disease in patients with juvenile myositis' by Sabbagh *et al*

We read with great interest the article by Sabbagh *et al*¹ published in the *Annals of the Rheumatic Diseases*. We congratulate the authors for their accuracy and the exhaustive data presented, which clearly demonstrate the importance of anti-Ro52 autoantibodies in disease monitoring, management and prognosis in patients with juvenile myositis. We would like, however, to highlight some key points.

First, in this study,¹ the patients with juvenile myositis including juvenile dermatomyositis (JDM), juvenile polymyositis (JPM) and juvenile connective tissue disease–myositis (JCTM) overlap are a highly heterogeneous population. In particular, JCTM added a great heterogeneity to the study population. In reality, there has been still controversy on considering CTM as a distinct entity or rather a complication related to other connective tissue disease itself.^{2–3} A recent study³ showed a distinct entity of CTM compared with PM and DM, featuring more extramuscular involvement and more severe infections. Especially, the prevalence of interstitial lung disease (ILD) in patients with CTM (48%) was significantly higher than that in those with PM (35%) and DM (30.8%). The results of the study by Sabbagh *et al* indicated that the positive rate of anti-Ro52 autoantibodies in patients with JCTM was higher than that in those with PM and DM. However, the positive rate is also highly heterogeneous among patients with different JCTM, varying from 0 (juvenile systemic sclerosis) to 29% (juvenile idiopathic arthritis).¹ Furthermore, in subgroup analyses of this study, there was a significant difference regarding the prevalence of ILD between patients with and without anti-Ro52 autoantibodies only in JDM group, but no difference in JPM or JCTM groups. As such, anti-Ro52 autoantibodies seemed to be only associated with JDM, but not with JPM or JCTM. In addition, we consider it necessary to validate the association between anti-Ro52 autoantibodies and ILD and other clinical features separately in DM patients by univariate and multivariate analyses, given too few numbers of patients with JCTM or PM. If so, the results will be more significant for clinical practice.

Second, in this study,¹ length of follow-up, as one of confounding factors, was included in multivariate analyses using linear or logistic regression to test the independent association of anti-Ro52 autoantibodies with clinical features of juvenile myositis. Although it was not detailed enough about the time when various clinical features occurred (at illness onset or diagnosis or during follow-up), we presume that authors investigated the association between anti-Ro52 autoantibodies and clinical features at illness onset or diagnosis. In this case, length of

follow-up may have little impact on the results and should not be used as a confounder. In contrast, delay to diagnosis should be considered. If so, we wonder whether the close association between anti-Ro52 autoantibodies and ILD or other clinical features has been remained.

Third, the number of anti-Ro52-positive patients is small and even. Single-digit in some subgroup analyses, which could lead to a low statistical power.

In conclusion, considering high heterogeneity of myositis, the association between anti-Ro52 autoantibodies and clinical features in patients with juvenile myositis needs to be discussed in more detail based on classification of myositis. The importance of anti-Ro52 autoantibodies in disease monitoring, management and prognosis in JDM patients deserves high attention, while the significance of these autoantibodies for JPM and JCTM has remained to be verified.

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