To the Editor:

We thank Dr. Pipitone, et al for their interest1 in our article2. Pipitone, et al reported high sensitivity of active myositis by 18F fluorodeoxyglucose positron emission tomography (FDG-PET) imaging3 compared with that in our study, when they defined active myositis based on the ratio of standardized uptake values of proximal muscles to that of the liver. Moreover, they reported that the PET sensitivity was decreased to a value similar to ours when they used the definition of active myositis in our study, a method comparing the uptake of muscle to the liver qualitatively, not quantitatively.

These findings indicate the importance of methods defining activity of myositis and we agree that a method for grading proposed by Walter, et al4 might be too strict to define activity of myositis.

It is to be clarified what methods and definitions are suitable for management of myositis in clinical practice. Moreover, the clinical significance of PET examination in measuring disease activity of myositis remains to be determined. Is PET imaging, an expensive and radiation-exposing examination, superior to other methods measuring disease activity including serum creatine kinase (CK) levels and magnetic resonance imaging examinations? Can PET provide information on myositis that could not be obtained by other methods, although PET is a good tool for detecting hidden malignancies?

It is also important to clarify what conditions the PET imaging reflects: cell infiltration, muscle cell response to injury, or regeneration of muscles. Pipitone, et al reported no correlation of FDG muscle uptake with disease duration, CK levels, muscle strength, or magnetic resonance imaging scores5. We showed only a tendency in patients with the FDG uptake to have myositis with endomysial cell infiltration6. In addition, cell infiltration into distal muscles as well as proximal ones had been reported5; significant FDG uptake in the distal muscles was not observed in our study.

To answer these questions, further research (e.g., a longitudinal study and a study including patients with noninflammatory myopathy such as muscle dystrophy as controls) is required.

TAKAYOSHI OWADA, MD, PHD; REIKA MAEZAWA, MD; KAZUHIRO KURASAWA, MD, PHD. Clinical Immunology, Dokkyo Medical University, Mibu, Tochigi, Japan. Address correspondence to Dr. K. Kurasawa, 880 Kita-Kobayashi, Mibu, Tochigi 321-0293, Japan. E-mail: kurasawa@dokkyomed.ac.jp

REFERENCES

J Rheumatol 2013;40:1; doi:10.3899/jrheum.121243