

Is ¹⁸F Fluorodeoxyglucose Positron Emission Tomography Useful to Assess Activity of Myositis?

To the Editor:

We read with great interest the paper by Owada, *et al*¹ that evaluated the role of ¹⁸F fluorodeoxyglucose positron emission tomography (FDG-PET) in assessing activity of myositis. The authors found that FDG-PET was highly specific (97%) in discriminating patients with myositis from unaffected controls. However, the sensitivity of FDG-PET was only 33%. In contrast, in a study published in 2012², we found that FDG-PET was 75% sensitive and 100% specific in distinguishing patients with active myositis from unaffected controls.

How can these discordant findings be reconciled? The main reason for such discrepancies lies in the different methods used to define active disease. Owada, *et al* considered FDG-PET positive for active myositis if muscle FDG uptake was greater than liver uptake, according to the method proposed by Walter, *et al* to assess activity of large-vessel vasculitis³. In contrast, we calculated the ratio of the average FDG proximal muscle to liver maximum standardized uptake values (SUVmax muscle/SUVmax liver) in patients with myositis and controls, respectively, and chose the cutoff value that yielded the best accuracy in discriminating patients from controls. Had we resorted to the same approach used by Owada, *et al*, FDG-PET sensitivity would have dropped to 27% (a value remarkably similar to that found by Owada, *et al*), despite the fact that all our patients had active myositis. Thus, using the grading proposed by Walter, *et al* may unduly decrease the sensitivity of FDG-PET in identifying active myositis.

In both studies, specificity of FDG-PET was determined using controls without muscle disease. Therefore, as Owada, *et al* remarked, it remains unclear whether muscle FDG uptake is specific for myositis. Preliminary observations from our group have revealed that muscle FDG uptake is similar in patients with myositis and in those with other myopathies, including human immunodeficiency virus-associated myopathy, paraneoplastic myopathy, necrotizing myopathy, and inclusion body myositis (unpublished data). Limited data from the literature also support the view that FDG-PET may disclose abnormal FDG in affected muscles from patients with myopathies different from myositis, including necrotizing and sarcoid myopathy^{4,5}. These findings may thus suggest that FDG-PET is not specific to myositis, in agreement with the concept that PET simply detects areas of increased cell metabolism regardless of its cause.

We fully concur with Owada, *et al* that FDG-PET can be very helpful in revealing extramuscular manifestations in myositis, including interstitial lung disease and hidden tumors. However, the definition of cancer-associated myositis according to the modified Bohan and Peter criteria (namely myositis associated with cancer within 1 year of the diagnosis of myositis)⁶ is probably too restrictive, because the risk of developing a tumor in myositis remains elevated beyond 5 years after the diagnosis of myositis⁷.

We agree with Owada, *et al* that FDG-PET is a useful investigation to assess patients with myositis, but its sensitivity can be significantly increased without incurring loss of specificity. Further research is required to better define the role of FDG-PET in the investigation of patients with myositis and in monitoring disease activity.

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