

**Comment on:
18F-fluorodeoxyglucose PET/
CT uptake characteristics
in ICI-induced polymyalgia
rheumatica compared to
idiopathic polymyalgia
rheumatica**

Sir,

I read with great interest the article on immune checkpoint inhibitor-mediated polymyalgia rheumatica (ICI-PMR) in patients with cancer compared with those with primary/idiopathic PMR (1). ICI-PMR is characterised by lower overall levels of extra-articular inflammation (1). Compared with patients with idiopathic PMR, those with ICI-PMR had lower 2-[18F]fluoro-2-deoxy-D-glucose (FDG) uptake on positron-emission tomography/computed tomography (PET/CT) at various sites, including the shoulder bursae and tendons, and the spinous and pelvic girdles. If there is no or minimal evidence of inflammation on FDG-PET/CT, why definitively adopt a diagnosis of PMR-like or ICI-PMR?

Moreover, ICI-PMR patients had a significantly higher prevalence of clinically confirmed peripheral arthritis, as evidenced by joint aspiration and/or ultrasound imaging. They also had elevated C-reactive protein (CRP) levels comparable to those of patients with idiopathic PMR. If so, why not adopt a rheumatoid arthritis (RA)-like diagnosis?

Furthermore, ICI-PMR was defined as inflammatory girdle pain after ICI initiation, with elevated acute-phase reactants and/or ultrasound evidence of proximal articular or extra-articular inflammation (1). Idiopathic PMR diagnoses had to meet the 2012 ACR/EULAR criteria (2), whereas only 60% of ICI-PMR fulfilled these criteria (1). So, why adopt a PMR-like diagnosis in these patients?

In their article, Bouchut *et al.* studied 18F-FDG uptake in 20 musculoskeletal regions of interest (ROIs) on PET/CT and found it lower at the ischiatic tuberosities, greater trochanters, anterior pubic symphysis, iliopsoas bursae, sternoclavicular joints, and interspinous bursae than in patients with idiopathic PMR (1). Although FDG-PET/CT is used for anatomical localisation, it has limited utility for monitoring and detecting relapse because uptake often persists despite clinical remission and is typically affected by steroid exposure, even after its withdrawal (3). Recovery from steroid

effects may take 3 weeks or longer (4). Therefore, I wonder whether FDG-PET/CT is a valid tool for characterising ICI-mediated rheumatism. Moreover, a similar study suggests that ICI-PMR could be treated with lower doses and a shorter taper than idiopathic PMR (5). In that study, FDG-PET/CT scans were obtained at the time of ICI-PMR or primary PMR diagnosis, before glucocorticoid therapy initiation (5), thereby avoiding steroid-related factors that might affect imaging sensitivity. ICI-PMR patients showed no arthritic pattern and had fewer constitutional symptoms (*e.g.*, weight loss), lower inflammation (ESR, CRP, and morning stiffness), and fewer inflammatory sites, mainly soft-tissue lesions, on FDG-PET/CT compared to primary PMR (5). This ICI-related low-grade inflammation, which necessitates low-dose steroid therapy, appears to represent a distinct PMR-like disease. It remains debated whether ICI-PMR is a unique disease or a subset of primary PMR (5-7).

Subtyping of PMR is feasible based on clinical features, as there are no imaging, pathologic, or serologic biomarkers. Clinical diagnosis of PMR is supported by laboratory findings, sometimes combined with imaging (*e.g.*, ultrasound, magnetic resonance imaging, and PET/CT) (8). In the article by Bouchut *et al.*, PET/CT scans were used, and it remains unclear how much PET/CT contributes to identifying ICI-PMR as a subset of PMR (1, 4). Primary/idiopathic PMR may present in four subtypes. The classic form of PMR is characterised by pain and stiffness in the proximal muscles of the shoulders, neck, and hip girdles, worse after rest, with elevated acute-phase reactants such as CRP and ESR and is often accompanied by constitutional symptoms. A subtype that involves additional soft-tissue and extra-articular inflammation, such as bursitis and tenosynovitis, which can be identified by ultrasonography (2) and by FDG-PET/CT (1, 5, 8). PMR sometimes shows more arthritic features and is seronegative. It occasionally shows more vasculitic features (8). Further investigation is warranted; hence, ICI-PMR remains unclassified among these primary PMR subsets and appears to be a distinct PMR-like entity within the ICI-mediated rheumatism.

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