Response to: Correspondence on "Immune checkpoint inhibitor-induced inflammatory arthritis persists after immunotherapy cessation" by Braaten *et al*

We were interested to read the letter by Ceccarelli *et al* regarding their experience with Immune checkpoint inhibitor (ICI)-induced inflammatory arthritis (IA) at Sapienza University.[1] Their findings support that ICI-induced IA is a heterogeneous disease with differing outcomes. The differences in the cohorts studied may also give us insight into the risk factors for persistence in ICI-induced IA. The authors point out one main difference between the cohorts, type of ICI therapy. Indeed, combination anti-cytotoxic T-lymphocyte-associated protein 4 (CTLA-4)/anti-programmed cell death protein-1 (PD-1) therapy was an independent risk factor for persistent IA in our cohort,[2] and their study included only patients on anti-PD-1 agents. There are several other relevant differences. First, the patients had a shorter duration of ICI use before IA was diagnosed and corticosteroids were started as compared with our study. Duration of ICI therapy was also an independent risk factor for IA persistence in our cohort.[2] Second, all patients in the study were evaluated deliberately for IA which likely led to earlier diagnosis and potentially milder disease. Disease activity is not specifically reported, but the higher incidence of IA (9.7%) than in any previously published studies suggests that milder disease was included.[1] The need for multicentre, international efforts to characterise longitudinal outcomes for ICI-induced IA is apparent.

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