Background: Immune checkpoint inhibitors (ICI) have changed the treatment landscape of many cancer types, but are also associated with development of immune-related adverse events, including de novo sarcoid-like reactions. However, little is known about the use of ICI therapy in patients with preexisting sarcoidosis as patients with preexisting autoimmune diseases have been systematically excluded from clinical trials of ICI therapy due to concerns of heightened toxicities. Emerging research suggests that ICI therapy can be considered in some patients with autoimmune diseases.1

Objectives: To determine the risk of sarcoidosis exacerbation or flare in patients with preexisting sarcoidosis receiving ICI therapy.

Methods: We conducted a retrospective cohort study of patients seen at The University of Texas MD Anderson Cancer Center between 2016-2019. Patients were included in the cohort if they received one of 7 ICI therapies (ipilimumab, nivolumab, pembrolizumab, durvalumab, avelozulab, or cemiplimab) and had an International Classification of Disease version 10 code of sarcoidosis (D86.7). Patients were included if the medical record documented a history of sarcoidosis, “probable” if a history of biopsy proven sarcoidosis was mentioned, and “definitive” if histological evidence was available. Frequency of flares and outcomes of patients after receiving ICI were collected.

Results: During the study timeframe a total of 32 patients with preexisting sarcoidosis received ICI therapy. Nine patients (28%) had a definitive diagnosis of sarcoidosis, 12 (37%) had a probable diagnosis and 11 (33%) had a possible diagnosis of sarcoidosis. The mean time between diagnosis of sarcoidosis and initiation of ICI therapy was 13 years (range: <1 to 51 years). Twenty-seven patients (84%) received monotherapy and five patients (16%) received combination or sequential ICI therapy. Of the 32 patients, one patient with a 20-year history of sarcoidosis, never treated, developed a clinically symptomatic exacerbation of sarcoidosis one month after the initial dose of atezolizumab, with increased hilar nodules on imaging, skin nodules, arthritis and uveitis. Biopsy of a lymph node showed non-necrotizing granulomas, and biopsy of the skin panniculitis. The patient also developed colitis thought to be an immune-related adverse event. Atezolizumab was discontinued after 3 doses. Patient was treated with prednisone and azathioprine.

Conclusion: Patients with a remote history of stable sarcoidosis at the time of ICI therapy infrequently develop a flare of their sarcoidosis. The risk of flares in patients with active sarcoidosis requiring immunosuppression at the time of ICI initiation is unknown.

References: