declared, Charles Masson: None declared, Divi Corne: None declared, Jean-Jacques Dubost: None declared, Laurent Lagrue: None declared, Sebastien Ottaviani: None declared, Franck Grados: None declared, Rakiba Belkhir: None declared, Olivier Chauvet: None declared, Philippe Goupille-Grangereau: research support from: AbbVie, Amgen, Biogen, BMS, Celgene, Chugai, Lilly, Janssen, Medac, MSD France, Nordic Pharma, Novartis, Pfizer, Sanofi and UCB, Consultant of: AbbVie, Amgen, Biogen, BMS, Celgene, Chugai, Lilly, Janssen, Medac, MSD France, Nordic Pharma, Novartis, Pfizer, Sanofi and UCB, Speakers bureau: AbbVie, Amgen, Biogen, BMS, Celgene, Chugai, Lilly, Janssen, Medac, MSD France, Nordic Pharma, Novartis, Pfizer, Sanofi and UCB, Jean-Charles Dubost: None declared, Laurent Marguerie: None declared, Sebastien Ottaviani: None declared, Franck Grados: None declared, Rakiba Belkhir: None declared, Jacques Masson: None declared, Divi Corne: None declared, Jean-Jacques Dubost: None declared, Laurent Lagrue: None declared, Sebastien Ottaviani: None declared, Franck Grados: None declared, Rakiba Belkhir: None declared, Olivier Chauvet: None declared, Philippe Goupille-Grangereau: research support from: AbbVie, Amgen, Biogen, BMS, Celgene, Chugai, Lilly, Janssen, Medac, MSD France, Nordic Pharma, Novartis, Pfizer, Sanofi and UCB, Consultant of: AbbVie, Amgen, Biogen, BMS, Celgene, Chugai, Lilly, Janssen, Medac, MSD France, Nordic Pharma, Novartis, Pfizer, Sanofi and UCB, Christelle Sordet: None declared, Bruno Fauret Grant/research support from: AbbVie, Lilly, MSD, Pfizer, Consultant of: AbbVie, Biogen, BMS, Boehringer Ingelheim, Celgene, Lilly, Janssen, Medac MSD France, Nordic Pharma, Novartis, Pfizer, Roche, Sanofi Aventis, SOBI and UCB, Peggy Philipp: None declared, Muriel Pierpont: None declared, Bernard Combe Grant/research support from: Novartis, Pfizer, Roche-Chugui, Consultant of: AbbVie, Gilead Sciences, Inc., Janssen; Eli Lilly and Company; Pfizer; Roche-Chugui; Sanofi, Speakers bureau: Bristol-Myers Squibb; Gilead Sciences, Inc.; Eli Lilly and Company; Merck Sharp & Dohme; Pfizer; Roche-Chugui; UCB, Olivier Lobatte Consultant of: BMS, Gilead, Medimmune, Novartis, Pfizer, Servier, UCB, Gaetane Nocturne: None declared \n
Disclosure of Interests: None declared

DOI: 10.1136/annrheumdis-2020-eular.5054

Immunology in rheumatic disease

OP0126 IS IMMUNE CHECKPOINT INHIBITORS THERAPIES SAFE AND EFFECTIVE FOR PATIENTS WITH CANCER AND PREEXISTING AUTOIMMUNE DISEASE? W. Xie1, H. Huang1, Z. Zhang1. 1 Peking University First Hospital, Department of Rheumatology and Clinical Immunology, Beijing, China

Background: Immune checkpoints inhibitors (ICIs) are associated with frequent immune-related adverse events (irAEs). Most patients with preexisting autoimmune diseases have been universally excluded from clinical trials and ICIs are not recommended for patients with cancer and PAD due to the unknown safety. In this study, we aim to evaluate the safety and efficacy of ICIs in patients with PAD and cancer.

Objectives: Systematic searches were performed of PubMed, EMBASE, and the Cochrane library from inception through September 2019 for observational studies reporting safety and efficacy data among ICIs-treated patients with cancer and PAD. A random effects meta-analysis was performed to calculate pooled incidence rates of PAD flare, irAEs and response.

Methods: Systematical search of PubMed, EMBASE and Cochrane Library plus a hand search of conference proceedings were performed for observational studies that reported cancer incidence in patients with RA treated with biologics or tocatafinib with active comparator of conventional DMARDs (csDMARDs) or TNFi. The pooled relative risk (RR) and 95% confidence interval (CI) were calculated with fix-effects or random-effects model.

Results: A total of 19 ICI-treated patients with PAD in 14 publications were finally identified. In the random effects meta-analysis, pooled incidence of PAD flares, de novo irAEs or both of any grade was 60% (95% CI 52%-68%). Viewed separately, there were 219 and 206 patients experiencing PAD exacerbation and de novo irAEs of any grade, yielding a pooled incidence of 35% (95% CI 29%-41%) and 33% (95% CI 24%-42%) respectively. Of these, most of flare and de novo irAEs were graded as mild (grade 1-2) (pooled proportion: 82%, 95%CI 72%-91%; 65%, 95%CI 54%-76%; respectively), Rheumatoid arthritis was associated with a trend to higher flare occurrence compared with another individual PADs (RR=1.25-1.88). With respect to efficacy, 136 patients showed complete or partial response, corresponding to a pooled response rates of 30% (95% CI 22%-39%). There were no statistical differences between patients with and without immunosuppressive therapy at ICI start regarding flare (RR: 1.08, 95% CI 0.72-1.62), but a trend towards lower response rates was observed in patients with baseline immunosuppressants (RR: 0.58, 95% CI 0.26-1.33).

Conclusion: Immune toxicities are frequent in ICIs-treated patients with PAD but often mild and manageable without discontinuing therapy. Rheumatoid arthritis is associated with a trend toward more flares. ICI treatment are effective and not absolute contraindication in PAD patients, but close monitoring and multidisciplinary collaboration should be contemplated, especially for those concomitantly receiving immunosuppressant or having rheumatoid arthritis.

References:
**Results:** Thirty-four seropositive RA, 12 seronegative RA and 34 HC s were included. The immune cell subsets which showed correlation with DAS28-ESR (r > 0.2 or r < -0.2) were activated CD4 T cells (r = 0.31), CD45RO+ memory CD4+ T cells and amongst this population, activated/proliferated cells in FlowJo version 10. A manual gating strategy was used to identify CD45RO+ (memory) CD4+ T cells and amongst this population, activated/proliferated cells percentage of activated/proliferated cells from stimulated wells divided by the replicate wells were harvested, pooled and stained for surface markers and via-tin staining. The Immune cell subsets which showed correlation with DAS28-ESR (r> 0.2 or r> -0.2) were activated CD4 T cells (r= 0.31), HLA-DR+Th1 cells (r= 0.18), HLA-DR+Th1-17 cells (0.22), CD3+CD8+effector T cells (r= 0.25), CD3+CD8+effector memory T cells (r= 0.26), plasma cells (r= 0.40) and CD14++CD16+intermediate monocyte (r= 0.23). The proportions of HLA-DR+Th1 cells (2.3% vs. 5.7%), HLA-DR+Th1-17 cells (0.7% vs. 2.2%), Th17 cells (1.7% vs. 2.0%), HLA-DR+Th1+ cells (0.02% vs. 0.1%), CD3+CD8+effector memory T cells (16.6% vs. 25.7%), plasma cells (0.04% vs. 0.17%) were statistically higher in the patients with RA compared to HCs. While the proportion of Tph cells showed weak correlation with DAS28-ESR (r = 0.18), that was extremely higher in RA (0.08% vs. 0.25%). Interestingly, when assessing the correlations with the disease activity in seropositive and seronegative RA separately, the proportions of Tph cells (r = 0.52) and HLA-DR+Tph cells (r = 0.50) were highly reflected in seropositive RA, but not in seronegative RA. Regarding the disease activity after the MTX treatment, the change of proportion of Tph cells between week 0 and 52 significantly reflected the change of DAS28-ESR (r = 0.75, p= 0.025), but not HLA-DR+Tph cells because of the non-specific reduction by the MTX treatment. Rather, HLA-DR+Tph cells significantly reflected the change of DAS28-ESR while receiving the MTX treatment (r = 0.76, p= 0.021).

**Conclusion:** HLA-DR+ Tph cells and HLA-DR+Th1 cells highly reflected the disease activity of seropositive RA. However, after the treatment, the proportion of HLA-DR+Tph cells decreased independent from the disease activity, and that of HLA-DR-Th1 cells more accurately reflected the change of the disease activity during treatment.

**References:**