were associated with high serum levels of inflammatory markers IL-6 (over 5.5 pg/ml, p=0.0006) and IL-1b (over 4 pg/ml, p=0.0005), however the level of these markers was not affected by the smoking status of the patients. We found 14 times lower sPD-L1 levels in smoking RA patients that did not receive TNF-inhibitors (p=0.0092), but treatment with TNF-inhibitors normalised levels of sPD-L1.

Furthermore, aCCP positivity in RA patients was associated with higher levels of sPD-L1 (p=0.0036). We speculate that the presence of antibodies might influence the levels of sPD-L1 through the stimulation of Fc-receptors expressed by PD-L1 producing cells. In PBMC depleted of T cells, we saw that smokers had lower mRNA expression of the stimulatory FcγRIIia (p=0.028) and predominance of the inhibitory FcγRIIb/FcγRIIia ratio (p=0.0004).

Conclusions Smoking decreases the serum levels of the inflammation limiting protein sPD-L1, but levels were restored by treatment with TNF-inhibitors. aCCP positive RA patients had higher levels of sPD-L1, possibly due to activation of Fc-receptors expressed by PD-L1 producing cells.

REFERENCE

Disclosure of interest None declared

P024 COMPARISON OF CCP2 AND CCP3 ASSAYS IN A LARGE COHORT OF ESTABLISHED RHEUMATOID ARTHRITIS AND CONTROLS

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Introduction Rheumatoid Factor (RF) and anti-citrullinated protein antibodies (ACPA) are important serological marker in the diagnosis of rheumatoid arthritis (RA) and are part of the classification criteria. ACPA are generally detected using anticitrullinated peptide (CCP) antibody assays. The first generation of the CCP test uses a peptide derived from the filaggrin protein as the antigen, whereas, the second and third generation CCP (CCP2, CCP3) are based on peptides specifically designed and optimised (mimotopes) to detect ACPA, thereby enhancing the immunoreactivity of the citrulline-containing epitope.

Objectives The goal was to compare the performance of CCP2 and CCP3 assays.

Methods 1655 samples including 968 RA patients and 687 controls (450 ankylosing spondylitis (AS) and 237 psoriatic arthritis (PsA) patients), all derived from the Swiss Clinical Quality Management in Rheumatic Diseases Foundation (SCQM) were included. ACPA were determined by CCP2 ELISA (Eurodiagnostica, Sweden), CCP3 ELISA (QUANTA Lite CCP3 IgG) and CCP3 CIA (QUANTA Flash CCP3 IgG) (both Inova Diagnostics, US). RF IgM was measured by ELISA (QUANTA Lite RF IgM, Inova Diagnostics, US).

Results The CCP2 ELISA showed a high sensitivity (71.1%) and a moderately high specificity (86.9%) with a corresponding Odds ratio (OR) of 16.3 (95% CI: 12.5 to 21.1). The two CCP3 assays showed lower sensitivities (61.8% for the ELISA and 61.4% for the CIA), but significantly higher specificities (98.4% and 98.5% respectively), resulting in much higher predictive values, with OR of 99.3 (95% CI: 54.4 to 181.2) and 107.5 (95% CI: 57.4 to 210.5), respectively. When compared at the same specificity (95%), the sensitivities were 61.3% for the CCP2 ELISA, 68.1% for the CCP3 ELISA and 66.1% for the CCP3 CIA. When multi-parametric analyses were performed by combining ACPA with RF IgM, combining different markers resulted in higher OR than the individual markers. The combination of CCP3 and RF IgM resulted in a higher OR (OR=187.0, 510/1655) than the combination of CCP2 with RF IgM (OR=36.7, 565/1655). The addition of CCP2 to the combination of CCP3 and RF IgM resulted in a lower OR (OR=175.0, 494/1655).

Conclusions CCP3 showed a better overall performance than CCP2 in this cohort of RA and controls, when analysed individually as well as in combination with RF IgM.

Disclosure of interest None declared