Abstract AB0205 – Table 1. Demographics and clinical characteristics (n=42)

Age (years)	59±9	DMARDs and Biologics	
Sex (W/M)	32/10	MTX	15 (36%)
Smoking (%)	12 (29%)	LEF	19 (45%)
RF(+)	31 (74%)	ADA	5 (12%)
AntiCCP(+)	27 (64%)	IFX	9 (21%)
DAS28-ESR	3.1±1.0	ETA	10 (24%)
Corticosteroids	28 (67%)	ABA	13 (31%)
		TOC	2 (5%)
		TOF	2 (5%)
		RTX	20 (50%)

Abstract AB0205 Table 2. Thoracic HRCT findings and self-reported questionnaires in RA patients

Warrick Score (n=15)	
Alveolitis	2.6±1.2
Fibrosis	12±6.3
Total	15±7
Leicester Cough Questionnaire score (n=42)	18±4
Modified Borg Scale	1.8±2.1
SF-36	53±17

Conclusions: In this study, we could not show any relationship between self-reported questionnaires and thoracic HRCT findings, except a weak association of the presence of parenchymal lesions with SF-36 scores. Alveolitis and/or fibrosis on thoracic HRCT were found to be associated with lower DLCO. DLCO was shown to be negatively correlated with SF-36 scores. SF-36 might be included in the detection of pulmonary evaluation in RA patients. The relationship between thoracic HRCT findings and self-reported questionnaires in RA necessitates further studies.

Disclosure of Interest: None declared

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AB0206 EXPRESSION OF INFLAMMATORY GENES AND THE IL1B GENE ASSOCIATION WITH THE SEVERITY OF RHEUMATOID ARTHRITIS IN TAMIL NADU POPULATION

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Background: Rheumatoid Arthritis (RA) is a multifactorial complex and chronic inflammatory disease associated with progressive joint destruction, disabling and systemic complications. The prevalence is about 0.5%–1% worldwide and 0.9% in India. Genetic factors are recognised to have substantial effect on the susceptibility to RA.

Objectives: The present study aims to investigate the inflammatory caspase genes (*CASP5* and *CASP8*) as well as proinflammatory cytokine interleukin-1β (IL-1β) in RA patients. Hence the study was designed to explore the possible association of inflammatory genes in Tamil Nadu population.

Methods: We conducted a study involving 55 RA patients and equal number of normal healthy controls and performed gene expression analysis in *CASP5* and *CASP8* genes. We also carried out genotyping of IL-1β gene using PCR-RFLP. For gene expression study, the mRNA levels of inflammatory genes were assessed using qPCR and the inflammatory marker levels (IL-1β) were estimated by ELISA.

Results: The gene expression analysis of RA patients showed activation of *CASP5* and *CASP8* compared to the healthy individuals. The inflammatory marker levels in the serum showed significantly higher levels (23.35±2.12 pg/mL) $p<0.05$ in RA patients compared to the control subjects. The homozygous and heterozygous mutant variants of *IL1B* were observed to be higher in the RA patients (OR=2.1, $p<0.01$).

Conclusions: Thus the results of our study suggests that, the mutant alleles of *IL1B* was associated with RA susceptibility which in turn has direct association with the increased levels of serum IL-1β in RA patients. In addition, the activation of inflammatory genes supports the role of inflammasome in the development of RA in Tamil Nadu population.

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AB0207 CALPROTECTIN (S100A8/9) PLASMA LEVELS DECREASE AFTER ABATACEPT THERAPY AND CORRELATE WITH DISEASE ACTIVITY IN PATIENTS WITH RHEUMATOID ARTHRITIS

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Background: Calprotectin (S100A8/9) is a damage-associated molecular pattern molecule that is involved in the early phase of tissue injury. It is found mainly in circulating neutrophils, monocytes and macrophages of rheumatoid arthritis (RA) synovial tissue where it acts as a chemoattractant and induces production of proinflammatory cytokines. Several studies have reported its association with clinical disease activity and radiographic damage in patients with RA.

Objectives: The aim of our study was to analyse the plasma levels of calprotectin in patients with established RA after the abatacept treatment compared with healthy individuals, and to examine their potential association with disease activity and treatment response.

Methods: The plasma levels of calprotectin were determined by ELISA (BÜHL-MANN Laboratories AG) in 40 patients with established RA before and 3 months after initiation of abatacept treatment, and in 30 age-/sex-matched healthy subjects. Disease activity was evaluated by 28-joint Disease Activity Score (DAS28). The CRP levels and erythrocyte sedimentation rate (ESR) were determined by routine laboratory techniques. Data are presented as median ± IQR.

Results: Calprotectin levels at baseline were significantly higher in patients with established RA than in healthy individuals (1925 [741; 4093] vs. 506 [302; 754] $p<0.0001$; ng/ml). After 3 months of therapy, the levels significantly decreased (from 1925 [741; 4093] to 1569 [695; 3115] $p=0.045$; ng/ml). Calprotectin baseline levels significantly correlated with CRP, ESR and DAS28 at baseline ($r=0.57$, $p=0.0003$; $r=0.56$, $p=0.0003$; $r=0.40$, $p=0.014$, respectively), with change in DAS28 over 3 months ($r=-0.54$, $p=0.001$) and with change in CRP over 3, 6, 12 months ($r=-0.56$, $p=0.0006$; $r=-0.61$, $p=0.001$; $r=-0.71$, $p=0.0001$, respectively). Calprotectin levels at month 3 significantly correlated with ESR at month 3 ($r=0.43$, $p=0.013$), CRP levels at month 3 and 6 ($r=0.39$, $p=0.27$ and $r=0.43$, $p=0.024$, respectively) and with change in CRP over 12 months ($r=0.45$, $p=0.023$). Change in calprotectin levels over 3 months correlated with the change in DAS28 over 3 months ($r=0.39$, $p=0.028$) and with change in CRP over 3, 6 and 12 months ($r=0.48$, $p=0.004$; $r=0.38$, $p=0.028$; $r=0.60$, $p=0.0009$, respectively).

Conclusions: We demonstrate here decrease in plasma levels of calprotectin after 3 months of abatacept therapy in patients with established RA, its association with disease activity and disease activity improvement over time.

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AB0208 UNFAVOURABLE CARDIOVASCULAR RISK PROFILE IN MALE PATIENTS WITH RHEUMATOID ARTHRITIS OF LOW DISEASE ACTIVITY

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Background: Rheumatoid arthritis (RA) is associated with the increased cardiovascular (CV) morbidity and mortality, mostly due to accelerating atherosclerosis. Both traditional and non-traditional factors seem to contribute to the excess of CV risk. Data in literature indicate a positive association between RA activity and the extent of CV disease (CVD) risk, suggesting a dominant effect of systemic inflammation. It is reported, that low disease activity is sufficient to achieve a protective effect against CVD and that atherosclerosis is not accelerated in RA of low activity or remission.

Objectives: The goal of the study was to assess CV parameters in female and male patients with RA of low disease activity in comparison with healthy controls.

Methods: The study was conducted in 70 patients with low RA activity, without known CVD (54 women, 16 men) and 33 healthy volunteers (18 women, 15 men). Patients underwent standard physical examination, assessment of disease activity in 28 joints (DAS28) and laboratory measurements including amino-terminal pro-brain natriuretic peptide (NT-proBNP). The following procedures were performed both in RA patients and controls: blood pressure (BP), carotid intima