Results: We enrolled 412 patients, of whom 70 were reRA and 102 taRA; 240 patients fulfilled neither definition. As can be seen in table 1, reRA patients were more frequently female (92.9 vs. 70.6%, $p<0.001$), younger (44.37 vs. 51.14 years, $p=0.002$), and had higher CDAI levels at first presentation (26.06 vs. 15.39, $p<0.001$); time to first DMARD treatment was significantly longer for reRA than taRA (3.17 vs. 1.45 years, $p=0.001$). In a multivariate analysis, treatment delay also showed statistical significance ($p=0.007$). After matching reRA with taRA patients for the date of their initial presentation at our clinic, treatment delay was significantly longer univariately ($p=0.013$) and adjusted for other significant predictors ($p=0.027$). As our matching allowance for calendar year was ± 1 year, we could only use 50 (of 70) identified reRA patients from the cohort study approach. Based on the significant predictors, a discriminative matrix model could be constructed (figure 1).

Characteristics	reRA vs. non-reRA n=342			
	Univariate		Multivariate	
	OR (95% CI)	Sig. (p)	OR (95% CI)	Sig. (p)
Time to First Treatment	1.15 (1.07; 1.24)	<0.001	1.12 (1.03; 1.21)	0.007
Female gender	4.57 (1.78; 11.72)	0.002	3.07 (1.14; 8.23)	0.026
Age at Symptom Onset	0.98 (0.97; 1.00)	0.048	0.99 (0.96; 1.01)	0.181
CDAI	1.06 (1.03; 1.08)	<0.001	1.06 (1.03; 1.08)	0.001

		reRA vs. non-reRA				
Gender	Female	10.6 (2.8; 44.3)	36.6 (18.4; 59.7)	51.8 (29.9; 72.9)	>2 years	Time to First Treatment
		3.4 (0.8; 25.3)	15.2 (6.1; 33.3)	25.1 (11.2; 47.0)	>1; ≤2 years	
		2.1 (0.5; 20.3)	10.8 (4.6; 23.3)	18.6 (9.0; 34.6)	≤1 year	
	Male	3.1 (0.5; 33.0)	13.2 (3.5; 45.9)	22.1 (6.6; 60.6)	>2 years	
		0.9 (0.1; 25.3)	4.6 (1.0; 24.9)	8.3 (2.0; 36.0)	>1; ≤2 years	
		0.6 (0.1; 19.9)	3.0 (0.7; 18.4)	5.8 (1.5; 26.8)	≤1 year	
		Disease Activity (CDAI)				
		REM/LDA (≤ 10) MDA (> 10, ≤ 22) HDA (> 22)				

*Colour scheme: blue: 0–5%; green: 5.1–15%; orange: 15.1–25%; red: >25% predicted probability of reRA; results are estimated for patient presenting in the year 2010

Abstract SAT0100 – Figure 1. Comparing reRA vs non-reRA in a cohort study (logistic regression model) and predicting the probability of reRA including all selected baseline risk factors* in a matrix risk model

*Colour scheme: blue: 0%–5%; green: 5.1%–15%; orange: 15.1%–25%; red: >25% predicted probability of reRA; results are estimated for patient presenting in the year 2010

Conclusions: Our data suggest that delay to initial treatment affects the long-term course of RA. Earlier treatment initiation thus may change the severity of RA.

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SAT0101 ATLANTOEPITROPHIC MAGNETIC RESONANCE IMAGING INVOLVEMENT IN EARLY RHEUMATOID ARTHRITIS

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Background: The involvement of the cervical spine in rheumatoid arthritis (RA) can be essential regarding prognosis and mortality. The early detection of a cervical spine involvement in RA is essential to avoid possibly fatal complications.

Objectives: To assess the involvement of the atlantoaxial joint in patients with early rheumatoid arthritis (ERA) to evaluate the role of magnetic resonance imaging (MRI) in depicting this early joint involvement and to establish a risk-profile for the individual patient.

Methods: Fifty patients (13 men and 37 women), mean age of 58.2 years (range 36–79 years) with clinical and laboratory evidence of ERA (mean disease duration <12 months) were included in our study. MRI of the atlantoaxial joint was performed in all patients. The MRI features were correlated with clinical, radiological and biochemical variables. All patients underwent radiographic examination of the hands, wrists and feet. The assessment of the structural damage was carried out by two experienced readers, according to simple erosion narrowing scores (SENS).

Results: Twelve (24%) of the 50 patients with early ERA had positive MR findings. In all cases the MR showed pannus surrounded the dens, with additional erosions in 8 patients, bone marrow oedema at atlantoaxial in 9 patients and an abnormal cervico-medullary angle (<135°), in 2 patients. Compared with patients without cervical involvement, these 12 patients showed significantly higher anti-CCP antibodies (ACPA) titre [mean 200.25 UI (SD 262.44) vs. mean 22.05 (SD

40.21) ($p<0.001$); higher swollen joint count (SJC) [mean 13.66 (SD 3.39) vs mean 8.65 (SD 3.38) ($p<0.001$); higher Ritchie Articular Index (RAI) [mean 33.25 (SD 5.94) vs. mean 20.86 (SD 4.22) ($p=0.047$); higher GH [mean 69.58 (SD 13.49) vs. mean 45.92 (SD 9.55) ($p<0.001$); higher Disease Activity Score (DAS) in 44 joints level [mean 5.72 (SD 0.44) vs. mean 4.52 (SD 0.53) ($p<0.001$); higher Health Assessment Questionnaire Disability Index (HAQ-DI) [mean 1.55 (SD 0.37) vs. mean 1.09 (SD 0.33) ($p<0.001$), and higher simple erosion narrowing (SENS) scores [mean 15.83 (SD 4.52) vs mean 7.71 (SD 3.43) ($p<0.001$). Multivariate analysis showed meaningful relationship between ACPAs, high level of DAS and the presence of hand and wrist erosive lesions (SENS) with cervical involvement.

Conclusions: Our results showed that ERA patients with higher disease activity and advanced peripheral erosiveness are indicators of higher risk of early involvement of the atlantoaxial inflammatory synovitis. In daily clinical practice the MRI of cervical spine it should be proposed in patients with prognostic factors of unfavourable disease evolution, even if asymptomatic.

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SAT0102 PRISTANE-INDUCED ARTHRITIS IN DARK AGOUTI RAT: A NEW ANIMAL MODEL TO STUDY CARDIOVASCULAR DYSFUNCTION IN RHEUMATOID ARTHRITIS

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Background: Rheumatoid arthritis (RA) is characterised by an increased cardiovascular (CV) mortality. Animal models provide the opportunity to study CV features in RA, however, most used animal models develop a “monophasic” arthritis, making those models inappropriate for long-term studies on CV impairments.

Objectives: The aim of this study was to characterise vascular function and cardio-metabolic parameters in the “chronic” model of pristane-induced arthritis (PIA) in Dark Agouti (DA) rats.

Methods: 80 rats DA received an intradermal injection of 150 µL of pristane (PIA) or of saline solution (controls) at day 0. Arthritis score was daily followed. Acetylcholine (ACh) and sodium nitroprusside (SNP)-induced vasorelaxation were studied in macrovascular (aortic rings pre-contracted with serotonin) and in microvascular levels (mesenteric artery segments pre-contracted with phenylephrine) at day 28 (acute phase) and day 120 (chronic phase). Radiographic score, circulating markers of inflammation, lipid and glucose levels were also measured.

Results: PIA rats developed an acute arthritis phase from day 13 to day 50 followed by a remission phase, then by a chronic arthritis phase from day 70 to day 120. Radiographic score was higher in chronic than in acute phase in PIA ($p<0.001$). Levels of IL-6, total cholesterol and triglycerides were higher in PIA than in controls at both phases ($p<0.001$) whereas plasma myeloperoxidase activity and glycaemia were unchanged. Adiponectine levels were lower in PIA compared to controls in acute ($p<0.001$) but not in chronic phase. ACh-induced vasorelaxation in macrovascular bed was significantly reduced in PIA compared to controls in acute ($p<0.05$) but not in chronic phase. Furthermore, an altered ACh-induced vasorelaxation was shown in microvascular bed in PIA in chronic ($p<0.001$) but not in acute phase. No altered SNP-induced vasorelaxation was observed between groups at both phases in both vascular beds. Endothelial function (EF) negatively correlated with arthritis score ($p<0.001$), IL-6 ($p<0.05$) and total cholesterol ($p<0.05$) levels in macrovascular but not in microvascular bed. No correlation was found between EF and myeloperoxidase activity, adiponectin and triglycerides levels in both vascular beds.

Conclusions: PIA model shares several features of the CV alterations in RA: an endothelial dysfunction at the micro- and macrovascular level with independency of course among these vascular beds, a link between inflammation and macrovascular endothelial dysfunction, associated with low lipid levels. These data suggest that this model would be very useful for long-term pharmacological studies as well for deciphering the complex pathophysiology of increased CV risk in RA.

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