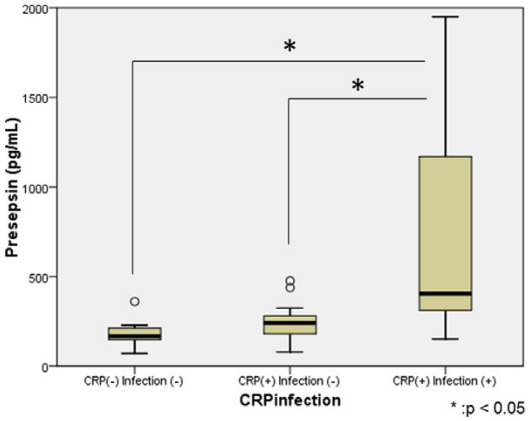
Results: Eighty-four patients with CTDs were enrolled, including 42 patients with rheumatoid arthritis (RA). The level of procalcitonin was evaluated in all patients, and the level of presepsin was in 48 patients. Thirty-six patients were classified in infection group; 38 patients in the CRP-positive non-infection group; and 10 patients in CRP-negative non-infection group. The level of presepsin was significant higher in infection group than CRP-positive non-infection group (693 +/- 577 pg/mL vs. 250 +/- 101 pg/mL, $p<0.01$) (Fig. 1). Among the patients with RA, the level of presepsin was significant higher in infection group than non-infection group (809 +/- 637 pg/mL vs. 233 +/- 135 pg/mL, $p<0.01$). AUCs of procalcitonin (0.823) and presepsin (0.821) showed similar diagnostic value. The cut-off value of presepsin and procalcitonin were 265 pg/mL and 0.16 ng/mL, respectively (sensitivity: 78.3% and 82.6%, specificity: 76.0% and 76.0%).

【Fig. 1】The level of presepsin in each group



Conclusions: Procalcitonin and presepsin may be of diagnostic value for bacterial infection in patients with CTDs, especially may distinguish bactrial infection from active phase in patients with CTDs.
Disclosure of Interest: None declared
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SAT0668 ASSESSMENT OF INTRACRANIAL VESSELS AND VASCULAR LESIONS IN RHEUMATOID ARTHRITIS. A DETAILED TRANSCRANIAL DOPPLER, CAROTID ULTRASOUND AND BRAIN MRI STUDY

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Background: Stroke has been associated with rheumatoid arthritis (RA). Vascular physiology should be assessed in the preclinical vascular state.
Objectives: We assessed RA patients and healthy controls by transcranial Doppler (TCD), carotid ultrasonography and brain MRI. We wished to determine preclinical pathophysiological changes in the cerebral vasculature.
Methods: Altogether 63 female RA patients and 60 age-matched controls underwent TCD assessment of the medium cerebral (MCA), basilar and vertebral arteries. Pulsatility (PI), resistance (RI) indices and circulatory reserve capacity (CRC) were determined. The presence of carotid plaques and intima-media thickness (cIMT) were also determined. Intracerebral vascular lesions were investigated by brain MRI. RA subsets include MTX- and biologic-treated patients.
Results: MCA PI and RI values at rest and after apnea are significantly increased in the total RA population vs controls. MCA PI (r) and RI (r) is also lower in biologic-treated patients. MCA CRC was also impaired and basilar artery PI was higher in RA. More RA patients had carotid plaques and had increased cIMT. Correlation analysis suggested multiple associations between right and left TCD parameters. There may be an association of TCD and carotid features with cerebral atrophy and age. Disease duration, disease activity and anti-CCP may influence left MCA PI and RI, as well as CRC. Lp(a) may also influence the development of carotid plaques.
Conclusions: This may be the first study to show increased distal MCA and basilar artery occlusion in RA as determined by TCD. RA patients also exert CRC defect. We also confirmed increased carotid plaque formation, increased cIMT. Biologics may beneficially influence some parameters in the intracranial vessels.
Disclosure of Interest: None declared
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SATURDAY, 17 JUNE 2017

Epidemiology, risk factors for disease or disease progression

SAT0669 HOW DO WE USE BIOLOGICS IN PATIENTS WITH A HISTORY OF MALIGNANCY? AN ASSESSMENT OF TREATMENT PATTERNS USING SCANDINAVIAN REGISTERS

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Background: Immune competence is of importance for the occurrence and outcome of malignancies. Robust data on risk of relapse of previous cancer following treatment with biological immune-modulators are scarce. Most treatment guidelines caution about their use in patients with a history of cancer, leaving rheumatologists with the decision whether a potential treatment benefit may offset any potential risk of cancer relapse.
Objectives: To assess the overall use of biologics and the relative use of different biological drugs in RA patients with a history of cancer.
Methods: As part of a Nordic collaboration, and using data from the ARTIS (Sweden), ROB-FIN (Finland), and ICEBIO (Iceland) biologics registers, we identified all patients with RA who initiated a first ever biological treatment 2010 through 2014. Through linkage to the national cancer registers, we identified those patients who had a history of any invasive malignancy (including squamous cell skin cancer) either within the five years preceding start of biological treatment ("recent history of malignancy") or more than five years before start of biological treatment ("non-recent history malignancy).
Results: The age- and gender distributions were similar across countries and drugs. Initiators of non-TNFi biologics were older than TNFI-initiators; the median age at start was the highest for rituximab. Out of a total of 8065 bio-initiations, 6% occurred in individuals with a history of cancer (2% with a cancer within 5 years, and 4% with a cancer more than 5 years before treatment start. Whereas there was little variation (around 5%) across TNFi initiators, the proportion of patients with a history of cancer at treatment start was higher among rituximab initiators, in part explained by age (Table). There were only small variations across country (not shown).
Conclusions: In Sweden, Finland and Iceland, one out of 20 biologics-initiators (and almost one out of five rituximab initiators) have a history of an invasive cancer, underscoring the need for more data on benefit/risks in this treatment context. The higher proportion in rituximab initiators is partly explained by differences in age at treatment start and reflects the preference for rituximab by clinicians for treatment of patients with history of cancer.
Disclosure of Interest: K. Chatzidionysiou Consultant for: Roche, Pfizer, Abbvie, Elli-Lilly, UCB, K. Aaltonen: None declared, D. Nordström Speakers bureau: AbbVie, BMS, Lilly, MSD, Novartis, Pfizer, Roche, UCB, B. Gudbjornsson Speakers bureau: Actavis, Celgene, MSD, Pfizer, G. Grondal: None declared, A. Geirsson: None declared, L. Steingrimsdottir: None declared, T. Frisell: None declared, J. Askling Grant/research support from: AbbVie, Eli Lilly, Janssen, Merck, Pfizer, Roche, UCB, Samsung
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Abstract SAT0669 – Table 1

	Etanercept	Infliximab	Adalimumab	Certolizumab	Golimumab	Rituximab	Abatacept	Tocilizumab	All
Total number of pts	2072	1538	1236	1036	795	883	281	224	8065
Age at treatment start (Swedish pts)	57	58	57	57	57	66	62	61	
Age at treatment start (Finnish pts)	55	50	54	55	54	67	58	54	
Age at treatment start (Icelandic pts)	52	53			53	66			
Total number of pts with cancer <5 yrs before start	41	20	9	18	15	76	6	12	197
Total number of pts with cancer ≥5 yrs before start	70	48	34	36	19	87	19	10	323
Total number of pts with a history of cancer	111	68	43	54	34	163	25	22	520
Proportion of pts with history of cancer <5 yrs before start	2%	1%	1%	2%	2%	9%	2%	5%	2%
Proportion of pts with history of cancer ≥5 yrs before start	3%	3%	3%	3%	2%	10%	7%	4%	4%
Proportion of pts with any history of cancer	5%	4%	3%	5%	4%	18%	9%	10%	6%