

of BASDAI and ASDAS-CRP, absence of previous infection had a strong relation with low levels of SIgA.

**Conclusions:** SIgA serum level were the only one serologic maker, which had an inverse correlation with all clinical activity variables of disease, previous infection and some specific antibodies associated with intestinal mucosal infection, suggesting a protective role of this molecular shape of IgA that is characteristic of mucosal immune responses

**References:**

[1] Mantis NJ. *Mucosal Immunol* (2011) 4:603–11.

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**AB0202 LOW DOSE ACETYSALICYLIC ACID AS PRIMARY PROPHYLAXIS OF CARDIOVASCULAR EVENTS IN RHEUMATOID ARTHRITIS. A LONGITUDINAL, RETROSPECTIVE STUDY**

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**Background:** Cardiovascular events (CV) i.e. acute myocardial infarction and stroke are recognized as a leading cause of mortality in patients with Rheumatoid Arthritis (RA) [1,2]. Acetylsalicylic acid (ASA) is known to be associated with a significant decrease in the incidence of CV events in patients at high risk for atherosclerosis like patients with diabetes [3] and has been recently reported to play a primary prophylactic role of CV events in Systemic Lupus Erythematosus by our team [4].

**Objectives:** To investigate the so far unexplored role of ASA in reducing CV morbidity in RA.

**Methods:** We analysed patients admitted to our Outpatient clinic from January to December 2015. Out of 199, 155 patients, who had been followed from January 2000 and had not experienced any CV event at the first visit, were enrolled. The incidence of CV morbidity was recorded at December 2016.

**Results:** The 155 patients had been followed-up for a median of 8 years (range 1–15 years). Out of them, 111 patients had been treated with ASA, that we currently administer to patients undergoing steroid treatment. During the 15-years of follow up, 5 CV events (2 cerebrovascular, 3 acute myocardial infarction) had occurred (Incidence rate 3.93/1000 person/year). Interestingly, only 1 CV event had occurred in ASA treated patients (Incidence rate 1.12/1000 person/year) with respect to 4 in the non-ASA group (44 patients) (Incidence rate 10.48/1000 person/year) ( $p=0.0146$ ).

**Conclusions:** Our study has several limitations including the low number of patients and CV events. Nevertheless, it might suggest a primary prophylactic role of ASA in RA, that awaits to be investigated in large controlled prospective studies.

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**AB0203 EXPRESSION LEVELS OF SELECTED GENES CAN PREDICT THE INDIVIDUAL RHEUMATOID ARTHRITIS PATIENT RESPONSE TO TUMOR NECROSIS FACTOR ALPHA BLOCKER TREATMENT**

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**Background:** Rheumatoid arthritis (RA) patients have many therapeutic options. However, there are limited tools to predict the individual patient's response to therapy. The GeneFrón personal diagnostic kit (IFR 300) has been developed based on analysis of large databases to select interferon stimulated gene (ISG) expressions which could predict response to a biologic agent

**Objectives:** This study aims to evaluate the ability of the GeneFrón diagnostic kit to predict the individual RA patient response to TNF $\alpha$  blockers.

**Methods:** Two separate analyses were performed, one retrospective and one prospective analysis. The response of 61 RA patients reported in 2 published

data sets was analyzed retrospectively utilizing the GeneFrón kit. In addition, 18 patients with RA were assessed prospectively, before and 3 months after starting treatment with a TNF $\alpha$  blocker. Clinical assessment included swollen and tender joint counts, patient and physician assessments of disease activity. Patients' blood samples were obtained before administration of the TNF $\alpha$  blocker and were analyzed utilizing the GeneFrón diagnostic kit which measures expression levels of selected genes by quantitative real time PCR.

**Results:** GeneFrón kit analysis of retrospective data correctly predicted the response to a TNF $\alpha$  blocker in 53 of 61 RA patients (accuracy - 86.8%). In the prospective analysis 6 patients achieved a moderate EULAR response, 6 achieved a good EULAR response and 6 did not respond. According to the EULAR moderate response, the GeneFrón diagnostic kit predicted the response correctly in 16 of 18 patients (accuracy-89%, sensitivity -100%, specificity - 67%). According to the EULAR good response, the kit predicted the response correctly in 15 of 18 patients (accuracy - 83.3%, sensitivity - 100%, specificity - 75%).

**Conclusions:** The GeneFrón diagnostic kit predicted the response to TNF $\alpha$  blockers in a high percentage of RA patients assessed either retrospectively or prospectively in a real life setting. This personal diagnostic kit has the ability to guide selection of a suitable biological drug for the individual RA patient

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**AB0204 DISEASE FACTORS ASSOCIATED TO ABNORMAL INTIMA-MEDIA THICKNESS IN MEXICAN MESTIZO RHEUMATOID ARTHRITIS PATIENTS**

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**Background:** Atherosclerotic cardiovascular disease (ASCVD) is the main mortality cause in patients with rheumatoid arthritis (RA) (1). It has been proven that the carotid intima-media thickness (CIMT) measured with carotid duplex ultrasonography (US) is an important ASCVD predictor with a measurement  $\geq 0.9$  mm (2–4).

**Objectives:** To characterize the disease factors related with abnormal carotid duplex US findings in Mexican mestizo patients with RA.

**Methods:** In a cross-sectional setting, we enrolled consecutive RA patients. Patients with overlap syndromes, personal history of ASCVD, dyslipidemia and previous use of any statin were excluded. A board-certified radiologist performed a bilateral carotid duplex US to all patients. Abnormal CIMT was defined as  $\geq 0.9$  mm (hypertrophy  $\geq 0.9 - 1.2$  mm and carotid plaque  $\geq 1.2$  mm). A clinical history and blood tests were performed at the time of the patient's visit. Disease activity was measured with Disease Activity Score using 28 joints-C-reactive protein (DAS28-CRP).

**Results:** We enrolled 57 patients. Demographic characteristics are shown in table 1. A total of 30 (52.2%) patients had an abnormal CIMT. US findings are shown in table 2. A significant correlation between abnormal CIMT and RA disease duration ( $p=0.04$ ), as well as between the former and anti-cyclic citrullinated peptide antibodies (ACPA) positivity ( $p=0.033$ ) was found.

Table 1. Demographic and disease characteristics

Variable	Results
Female gender, n (%)	54 (94.7)
Age (years), mean $\pm$ SD	56 $\pm$ 9.9
Disease duration (years), mean $\pm$ SD	12.4 $\pm$ 8.3
BMI (kg/m <sup>2</sup> ), mean $\pm$ SD	28.22 $\pm$ 4.9
Smoking status, n (%)	5 (8.77)
DAS 28- CRP, mean $\pm$ SD	3.33 $\pm$ 1.19
Disease Activity, n (%)	
Remission	17 (29.8)
Low	11 (19.3)
Moderate	25 (43.8)
Severe	4 (7.1)
Positive Anti-CCP, n (%)	44 (77.19)
Positive RF, n (%)	51 (89.47)

BMI: Body Mass Index.

Table 2.- Carotid Doppler ultrasound findings.

Ultrasound findings, n (%)	Disease duration	Disease Activity	Positive Anti-CCP	
			Positive Anti-CCP	Positive RF
Abnormal CIMT	32 (56.14)	<b>0.022</b>	0.57	<b>0.034</b>
Plaque	11 (19.29)	0.33	0.75	0.67
Hypertrophy	21 (36.84)	0.52	0.59	0.54

**Conclusions:** There is a strong relationship between CIMT and the chronic inflammatory process of RA, as well as ACPA positivity. These results might be influenced by the high mean disease duration of our patients. Prospective studies that evaluate CIMT among disease duration intervals are necessary to support these findings.

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**AB0205 CAROTID INTIMA-MEDIA THICKNESS LINKED TO THE PRESENCE OF CARDIOVASCULAR RISK FACTORS IN MEXICAN MESTIZO PATIENTS WITH RHEUMATOID ARTHRITIS**

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**Background:** Rheumatoid Arthritis (RA) is associated to subclinical atherosclerosis. Traditional risk factors for cardiovascular outcomes do not explain completely the higher risk, which could be caused by chronic systemic inflammation.

**Objectives:** The aim of this study is to relate abnormal carotid intima-media thickness (CIMT) to the presence of cardiovascular risk factors.

**Methods:** Observational cross-section design. We included patients who fulfilled the 1987 ACR and/or 2010 ACR/EULAR classification criteria for RA, 40 to 75 years old, with no personal history of atherosclerotic CV disease. A board-certified radiologist performed carotid duplex ultrasounds. Patients were distributed in two groups according to the absence (Group 1) or presence (Group 2) of traditional risk factors for cardiovascular disease (smoking status, dyslipidemia, high blood pressure and diabetes).

**Results:** A total of 82 patients were included. Demographic characteristics for each group are shown in Table 1. Ultrasound findings are shown in Table 2. CIMT alterations were more common in Group 2 (66.7%) than in Group 1 (38.7%), with statistical significance ( $p=0.013$ ). Presence of carotid plaque was more common in Group 2 (27.5%) than in Group 1 (16.1%), shown clinical relevance, although did not shown statistical significance ( $p=0.18$ ).

Table 1. Demographic characteristics

Variable	Total	Group 1 (n=31) (CVRF-)	Group 2 (n=51) (CVRF+)	P
Patients, n (%)	82 (100)	31 (37.80)	51 (62.20)	–
Female gender, n (%)	77 (93.9)	29 (93.5)	48 (94.1)	0.0917
Age (years), mean ± SD	57±9.96	51.90±8.43	59.82±9.69	<b>0.001</b>
Disease duration (years), mean ± SD	12.45±8.39	11.74±8.76	12.89±8.32	0.554
BMI (kg/m <sup>2</sup> ), mean ± SD	28.22±4.9	29.05±5.09	27.72±4.92	0.248
Smoking status, n (%)	8 (9.75)	–	8 (15.68)	–
Diabetes, n (%)	13 (15.85)	–	13 (25.49)	–
HBP, n (%)	28 (34.14)	–	28 (54.90)	–
Dyslipidemia, n (%)	8 (9.75)	–	8 (15.68)	–

CVRF: Cardiovascular Risk Factors, HBP: High Blood Pressure.

Table 2.- Carotid ultrasound findings

	Total n=46	Group 1 (CVRF -) n=12	Group 2 (CVRF +) n=34	p
Abnormal CIMT, n (%)	46 (56.1)	12 (38.7)	34 (66.7)	<b>0.013</b>
Plaque, n (%)	19 (23.2)	5 (16.1)	14 (27.5)	0.18
Bilateral, n (%)	12 (14.6)	1 (3.2)	11 (21.6)	<b>0.02</b>
Hypertrophy, n (%)	28 (34.1)	7 (22.6)	21 (41.2)	0.068
Bilateral, n (%)	12 (14.6)	2 (6.5)	10 (19.6)	0.091

CVRF: Cardiovascular Risk Factors

**Conclusions:** In this cohort of Mexican patients with RA, we demonstrate relation between the presence of alterations in CIMT (carotid hypertrophy and carotid plaque) and risk factors for cardiovascular disease, which can be enhanced by intrinsic risk of RA. These findings reaffirm the importance of global health assessment in patients with RA to reduce morbidity.

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**AB0206 INTERLEUKIN-17 AND CC-CHEMOKINE LIGAND 20 ARE NOT USEFUL MARKERS OF RHEUMATOID ARTHRITIS ACTIVITY IN PATIENTS UNDERGOING BIOLOGIC TREATMENT**

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**Background:** Interleukin-17 (IL-17) and IL-17-induced CC-chemokine ligand 20 (CCL20) are increasingly implicated in the pathogenesis of rheumatoid arthritis (RA). A correlation has been reported to exist between serum levels of IL-17 and CCL20 and the disease activity following biologic treatment [1]. However, such an effect has not been universally demonstrated [2].

**Objectives:** The aim of the present study was to investigate if serum IL-17 and CCL20 reflect activity of the disease and whether they could be of prognostic value for predicting therapeutic response to biologic therapy in RA.

**Methods:** Thirty RA patients qualified to receive biologic treatment were prospectively assessed before and 12 weeks of therapy with either TNF $\alpha$  inhibitors or anti-IL-6 receptor antibodies. Serum concentrations of IL-17 and CCL20 were measured with high sensitivity ELISA with estimated detection levels 0.01 pg/ml and 0.47 pg/ml, respectively. Successful response to therapy was defined by the EULAR criteria.

**Results:** The patient baseline characteristics were summarized in Table 1.

Table 1. Patient characteristics: data presented as the mean  $\pm$  SD or %

Age (years)	54.3 $\pm$ 11.5
Sex (% of male)	4 (13%)
Disease duration (years)	9.6 $\pm$ 6.0
Prednisone (<5 mg/24h) (%)	17 (57%)
NSAIDs (%)	22 (73%)
Methotrexate (%)	12 (40%)
Biologic treatment (%)	– TNF $\alpha$ inhibitors (adalimumab, certolizumab, golimumab, infliximab): 22 (73%)
	– IL-6R blocker (tocilizumab): 8 (27%)

Twelve weeks of biologic treatment resulted in a significant improvement in the majority of the patients with only 2 patients (7%) identified as non-responders. The favorable response to therapy was reflected both by clinical and standard biochemical criteria (Table 2). However, the mean serum concentrations of IL-17 and CCL20 did not change significantly over the course of therapy and they did not correlate with the disease activity, response to therapy, the type of biologic intervention and other medication used.

Table 2. Selected parameters before and after treatment: data presented as the median (interquartile range)

	Before treatment (n=30)	After 12-weeks of treatment (n=30)	P-value (Wilcoxon test)
DAS28 (ESR)	5.53 (5.26–6.08)	3.23 (2.19–3.75)	<0.001
ESR (mm/h)	24.0 (16.0–32.0)	10.0 (4.0–20.0)	<0.001
CRP (mg/l)	7.37 (2.3–15.8)	0.8 (0.1–5.5)	<0.001
WBC (10 <sup>9</sup> /l)	9.0 (7.6–9.9)	7.8 (6.6–9.6)	0.014
IL-17 (pg/ml)	0.65 (0.00–5.81)	0.53 (0.00–4.45)	0.355
CCL20 (pg/ml)	22.0 (18.0–28.0)	22.5 (20.0–31.0)	0.322

Serum IL-17 and CCL20 levels showed no correlation with DAS28, and standard inflammatory markers.

**Conclusions:** Serum levels of IL-17 and CCL20 did not parallel changes in the clinical status and standard biochemical parameters in patients undergoing biologic treatment for RA. Thus, the measurement of IL-17 and CCL20 in serum does not seem to provide additional information that would help to monitor the response to biologic treatment in RA more effectively.

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