population census of the National Institute of Statistics, with an estimated prevalence of RA of 0.5% (women: 0.8%, men:0.2%). Crude and adjusted rates of the solid tumors were calculated. The trend was analyzed by Generalized Linear Model (GLM).

**Results:** 338,343 RA hospital admissions were detected in the study period, being 18,401 (5.4%) due to solid tumors. The main clinical-demographic characteristics are shown in the next table.

<table>
<thead>
<tr>
<th>Total</th>
<th>Women</th>
<th>Men</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>18,401 (5.4)</td>
<td>8,689 (3.8)</td>
<td>9,712 (8.6)</td>
</tr>
<tr>
<td>Age, mean (SD)</td>
<td>69.98 (11.21)</td>
<td>68.62 (12.3)</td>
<td>71.20 (9.99)</td>
</tr>
<tr>
<td>In-hospital extus n (%)</td>
<td>2455 (13.34)</td>
<td>1035 (11.9)</td>
<td>1420 (14.62)</td>
</tr>
<tr>
<td>Charlson Index, mean (SD)</td>
<td>5.72 (2.95)</td>
<td>5.63 (2.95)</td>
<td>5.80 (2.95)</td>
</tr>
<tr>
<td>Stay, mean (SD)</td>
<td>10,94 (11.6)</td>
<td>10,76 (11.6)</td>
<td>11,11 (11.77)</td>
</tr>
</tbody>
</table>

The solid tumor adjusted rate during the study period was 647.53/10^5 inhabitants/yr (366.97/10^5 in women and 1792.99/10^5 in men; relative risk men:women =4.8). This rate increased from 305.65/10^5 in 1999 to 993.19/10^5 in 2015 (from 814.06/10^5 in 1999 to 2535.5/10^5 in 2015 in men; from 181.68/10^5 in 1999 to 607.71/10^5 in 2015 in women). The annual age-adjusted rate increased significantly: 7.37% (6.52% in men and 8.02% in women; p<0.001).

**Conclusions:** There was an increasing incidence of hospital admissions due to solid tumors in RA in Spain during the period 1999-2015. An annual rate increase of 7.37%, is estimated.

**Disclosure of Interest:** None declared

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**Figure 1.** Trends in disease activity scores over 8 years by four indices in mean and adjusted mean (AM) methods. (A) Trends based on DAS28-CRP and DAS28-ESR in two methods (B) Trends based on SDAI and CDAI in two methods

**REFERENCES:**


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**Disclosure of Interest:** None declared

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**SAT0115**

**TRENDS IN THE ACTIVITY OF RHEUMATOID ARTHRITIS AS THE CONSEQUENCE OF TREAT-TO-TARGET STRATEGY: EIGHT-YEAR DATA FROM 2009 TO 2016**

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**Background:** In past decades, treatment of rheumatoid arthritis (RA) has advanced greatly, driven largely by the advent of new medications and treat-to-target (T2T) strategy, but the secular trends in the activity and remission of RA over past years and the efficacy of T2T strategy are not fully validated in large population in real life practice.

**Objectives:** To investigate the trends in the activity of RA over past 8 years and evaluate the value of T2T strategy in daily practice.

**Methods:** All the medical records of RA patients from 2009 to 2016 were retrospectively reviewed. Disease activity scores at obtained visits were measured by DAS28-CRP, DAS28-ESR, SDAI and CDAI. To display trends over years, both methods (B) Trends based on SDAI and CDAI in two methods

**Results:** In total, 1,001 patients with 6,944 clinical visits were included. Over eight-year period, significant improvements were witnessed in disease activity and remission rate, measured by all four indices (p<0.0001). More patients achieved lower disease activity and higher remission rates after T2T adherence in 2011 compared to those in the years of 2009 and 2010 (P<0.0001). Moreover, sub-cohort study revealed that more patients (49.3%>73.2% vs. 19.1%>34.5%)

**OR=2.4-3.0) achieved remission with a shorter median time compared with the non-T2T period group (p<0.001), particularly in DAS28-CRP (21 vs. >52 weeks), DAS28-ESR (37 vs. >52 weeks).

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**SAT0116**

**THE STUDY OF BASELINE SERUM SICAM-1 AND CXCL13 LEVELS IN PREDICTING RESPONSE TO TUMOR NECROSIS FACTOR-A INHIBITOR THERAPY IN PATIENTS WITH RHEUMATOID ARTHRITIS**

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**Background:** TNF-α inhibitors are not effective for each patient, leading to poor response as well as financial burden. There is an urgent need for biomarkers to assist us in individualized treatment. However, reliable biomarkers that predict therapeutic response to TNF-α inhibitors are still lacking.

**Objectives:** To investigate whether baseline serum soluble intercellular adhesion molecule-1 (sICAM-1) and C-X-C motif chemokine ligand 13 (CXCL13) could serve as biomarkers to predict therapeutic response to TNF-α inhibitor therapy in RA patients.

**Methods:** RA patients were enrolled from the 12-week TNF-α inhibitor clinical trial in our center between October 2014 and October 2017. 20 age- and gender-matched healthy controls were also recruited. Serum samples at baseline and week 12 were collected from RA patients, then serum levels of sICAM-1 and CXCL13 were measured by enzyme-linked immunosorbent assay. Clinical and laboratory data were recorded from baseline to week 12. RA patients were classified into responders and non-responders at week 12 according to EULAR response criteria.

**Results:** 51 RA patients were enrolled in this study. Serum levels of sICAM-1 and CXCL13 in RA patients were significantly higher than healthy controls (p<0.01 and p<0.001 respectively). Serum sICAM-1 and CXCL13 levels were higher in seropositive RA patients (p=0.012 and p=0.005 respectively). Baseline serum levels of sICAM-1 and CXCL13 were correlated with changes in ESR, DAS28-ESR, DAS28-CRP, SDAI and CDAI. Baseline serum sICAM-1 levels were higher in responders to TNF-α inhibitor therapy at week 12 by EULAR response criteria (p=0.010). However, there was no significant difference in CXCL13 levels. In addition, serum sICAM-1 and CXCL13 levels were decreased after treatment in
responders (p<0.001 and p<0.001 respectively), nevertheless, non-responders showed a rising trend (p=0.086 and p=0.051 respectively). Binary logistic regression model revealed that baseline serum sICAM-1 levels had a positive effect on response to therapy. ROC curve analysis for predictive ability of baseline serum sICAM-1 showed an area under the curve (AUC) of 0.775 (p=0.010).

**Conclusions:** Serum sICAM-1 and CXCL13 levels were elevated in RA patients, and they were higher in seropositive patients than in seronegative patients. Elevated baseline serum sICAM-1 levels were associated with favorable response to TNF-α inhibitor therapy. The decrease of serum sICAM-1 levels after treatment in responders was consistent with their therapeutic response. Thus, baseline serum sICAM-1 could be a predictive biomarker for TNF-α inhibitor therapy in RA patients. There was a lack of reliable evidence that baseline serum CXCL13 had predictive ability, possibly due to different mechanisms of action or small sample size.

**REFERENCES:**


**Acknowledgements:** PUCRP 201305

**Disclosure of Interest:** None declared

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**SATURDAY, 16 JUNE 2018**

**Rheumatoid arthritis - comorbidity and clinical aspects**

**SAT0117**

**HIGH URIC ACID AS A RISK FACTOR FOR CARDIOVASCULAR DISEASES IN RHEUMATOID ARTHRITIS PATIENTS**

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**Background:** Rheumatoid arthritis (RA) patients have an increased risk of cardiovascular diseases (CVD). It is unclear whether an elevated serum uric acid (UA) further increases that risk.

**Objectives:** We study CVD and their risk factors in association with UA in RA patients.

**Methods:** Adult patients who satisfied the ACR classification criteria for RA from The Kuwait Registry for Rheumatic Diseases (KRRD) from four major hospitals were evaluated from February 2013 through May 2017. Patients with recorded UA were identified and CVD and their risk factors were studied in those patients. To optimize classifier number and prediction accuracy, hierarchical cluster analysis for multiple factors were performed, which indicated nine possible independent CVD risk factors. A binary logistic regression was conducted to examine their significant association with CVD and the independence of UA as a risk factor.

**Results:** A total of 564 RA patients with available UA were identified, 353 (62.6%) females. Mean age was 50.8±11.5 years and disease duration 10.5±2.9 years. Mean UA was 271±78 μmol/L. Of those patients, 31 (5.5%) were reported to have CVD. UA was significantly correlated to the presence of CVD (r=0.262, p<0.011). Logistic regression model indicated a 10% increase of CVD with every 10 μmol/L increase in UA. A correlation matrix between UA and other risk factors showed a significant association between high uric acid and a younger age at RA diagnosis (r=0.262), hyperlipidemia (r=0.191) and diabetes mellitus (r=0.244).

**Conclusions:** Our study suggests that UA may be an independent risk factor for CVD and is associated with the presence of other risk factors. UA should be measured and carefully approached in RA patients.

**Disclosure of Interest:** None declared

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**SAT0118**

**ASSOCIATION OF RENIN-ANGIOTENSIN SYSTEM IMBALANCE WITH SUBCLINICAL ATHEROSCLEROSIS AND DISEASE ACTIVITY IN RHEUMATOID ARTHRITIS**

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**Background:** Rheumatoid arthritis (RA) is an independent risk factor for cardiovascular disease (CVD). The renin-angiotensin system (RAS) is a hormonal cascade with important role in hydroelectrolytic homeostasis, blood pressure and regulation of cardiovascular remodeling. Angiotensin II (Ang II) acts as a proinflammatory mediator (1).

**Objectives:** To investigate the association of serum levels of RAS components with the presence of subclinical atherosclerosis using carotid ultrasonography in women with RA.

**Methods:** Women with RA according to ACR/EULAR 2010 or ACR 1987 criteria and without clinical ischemic CVD were included. Disease activity was assessed using the DAS28. The presence of atherosclerotic plaques and the thickness of the medium-internal complex (EMI) of the arterial wall in the common carotid artery were evaluated by ultrasonography. Serum levels of angiotensin (Ang II), Ang-(1-7), angiotensin converting enzyme (ECA) and ECA II were determined by enzyme immunoassay.

**Results:** 50 women with RA, mean age 48.2 years (±7.32), mean duration of disease of 15.35 years (±8.56), DAS28 of 4.02 (±4.11) and CDAI of 14.23 (±11.53) were included. Seven patients presented altered EMI, eight had atherosclerotic plaque. The prevalence of risk factors for CVD was: 12% of smoking, 12% of family history of premature CVD, 46% of arterial hypertension, 10% of diabetes, 62% of dyslipidemia, 94% of abdominal obesity and 46% of metabolic syndrome. The control group consisted of 30 healthy women, mean age of 46.3 years (±7.72). RA patients had a higher serum concentration of Ang II (p<0.01), Ang-(1-7) (p<0.01), and ACE (p<0.01) than the control group (table 1). There was a negative correlation between ECA II and EMI (p=0.041, rho 0.290). EMI correlated positively with age (p=0.022, rho 0.324), disease duration (p=0.012, rho 0.315) and overall Framingham risk (p=0.008, rho 0.368) and Ang II correlated positively with DAS28 (p=0.034, rho 0.301) and CDAI (p=0.040, rho 0.291).

**Table 1.** Comparison between plasma concentrations of RAS biomarkers in patients with rheumatoid arthritis (RA) and the control group (CG).

<table>
<thead>
<tr>
<th>RAS Biomarkers (pg/mL)</th>
<th>RA (n = 50) Mean±SD</th>
<th>CG (n = 30) Mean±SD</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ang II</td>
<td>407.60±278.68</td>
<td>198.77±105.48</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Ang-(1-7)</td>
<td>162.06±234.58</td>
<td>36.94±61.36</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>ACE</td>
<td>266.21±103.64</td>
<td>222.69±147.81</td>
<td>0.17</td>
</tr>
<tr>
<td>ACE II</td>
<td>71.86±27.32</td>
<td>153.57±225.04</td>
<td>0.17</td>
</tr>
</tbody>
</table>

RA = rheumatoid arthritis; CG = control group; SD = standard deviation; Ang = angiotensin, ACE =angiotensin converting enzyme. 1Mann-Whitney Test.

**Conclusions:** Imbalance of RAS components, especially Ang II and ECA II, may be associated to CVD in RA patients. Ultrasonography of the carotid arteries can identify patients that could benefit from ECA blockade.

**REFERENCE:**


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**Disclosure of Interest:** None declared

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**SAT0119**

**PHYSICAL ACTIVITY IN TUNISIAN ADULTS WITH RHEUMATOID ARTHRITIS**

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**Background:** Physical activity (PA) is associated with multiple health-related benefits among the general population and adults with chronic diseases like...