SOLUBLE CD163 AS A POTENTIAL BIOMARKER IN SYSTEMIC SCLEROSIS

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Background: Recent accumulating evidences indicate a crucial role of macrophage lineage in the pathogenesis of fibrotic diseases including systemic sclerosis (SSc). CD163 is a surface marker expressed by M2 macrophages that accumulate during the healing phase of acute inflammation. It is actively released from the plasma membrane in response to certain inflammatory stimuli and enters the circulation in its soluble form (sCD163).

Objectives: In this study, we aimed to evaluate the performance of serum and urinary sCD163 concentrations as possible biomarker in SSc.

Methods: Urine and serum samples were obtained from SSc patients, fulfilling the 2013 ACR/EULAR classification criteria for SSc, and age- and sex-matched controls. Serum and urinary sCD163 concentrations were measured by commercially available ELISA kit (R and D systems) and evaluated for their significance in comparison to controls. Statistical analysis was carried out using Mann-Whitney U test and the relationship between parameters was statistically examined by Spearman’s rank test.

Results: Two hundred and three SSc patients were included, 163 (80%) were female, with a mean ± standard deviation (SD) age of 59±13 years and a mean ± SD disease duration of 12±9 years. Eighty-one (41%) patients had diffuse cutaneous SSc and mean ± sCD163 mRSS was 6.6±7.7. Lung fibrosis on imaging was observed in 33% of the patients, 7% had pulmonary arterial hypertension, 44% had history of digital ulcers and 41% were taking immunosuppressive therapy. Control group consisted of 47 age- and sex-matched patients with non-inflammatory cutaneous SSc and mean ±SD mRSS was 6.6±7.7. Lung fibrosis on imaging was also observed in 33% of the patients in the control group. Serum sCD163 levels were significantly higher in SSc patients compared with controls (mean ±SD: 529±251 vs 385±113 ng/ml; p<0.001). Urinary sCD163 concentrations in SSc patients were also higher than those in controls, but this did not reach significance (236.9±498 vs 176.2±173 ng/ml; p=0.580).

When looking at the subsets according to skin disease or disease duration, no difference could be identified. Furthermore, when the organ involvements were investigated, no subpopulation could be identified as having higher concentrations.

Conclusions: To our knowledge this is the first evaluation of both serum and urinary sCD163 levels in SSc. Our results show a significant difference for sera values that should be prioritised for further studies as compared to urinary concentrations conversely to what has been described in lupus. Our results further support that the M2 macrophages/CD163 signalling system may play a role in the pathogenesis of SSc. However, further studies are required to address the exact role of CD163 in the pathogenesis of SSc and to determine whether it could help in the risk-stratification of the patients in this heterogeneous disease.

Disclosure of Interest: None declared

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INTERLEUKIN-10 INDUCTION PREVENTS VASCULAR INFLAMMATION IN A MURINE MODEL OF KAWASAKI DISEASE

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Background: Kawasaki disease (KD), which is a common paediatric heart disease, is characterised by coronary vasculitis and subsequently aneurysm formation. Although the administration of intravenous immunoglobulin (IVIG) is effective for reducing aneurysm formation, approximately 10%–20% of patients are resistant to this therapy. Therefore, additional therapeutic approaches for treating the IVIG-resistant patients need to be developed.

Methods: To induce the expression of IL-10 in vivo, Adeno-associated virus (AAV) vectors encoding IL-10 were injected into DBA/2 mice. After the induction of IL-10, the mice were treated intraperitoneally with CAWS to induce vasculitis. Cardiac functions by echocardiography, inflammation and fibrosis by histological analyses, gene expression of inflammatory cytokines and fibrosis-related factors in the heart, and infiltrating cells by flow cytometry were assessed to evaluate the effects of IL-10.

Results: In vitro study, bone marrow-derived macrophages (BMDM) were stimulated with CAWS in presence or absence of IL-10. TNF-α and IL-6 produced by the BMDM and Dectin-2 expressions on the BMDM were assessed.

Conclusions: Our study has shown that IL-10 may have therapeutic application in the prevention of coronary vasculitis and aneurysm formation, and provided new insights into the mechanism underlying the pathogenesis of KD.

Disclosure of Interest: None declared


LOWER LEVELS OF INTERFERON GAMMA COMPARED FROM PATIENTS WITH ENTHESITIS RELATED ARTHRITIS SECRETE HIGHER LEVELS OF IL23 AND LOWER LEVELS OF INTERFERON GAMMA COMPARED TO HEALTHY CONTROLS

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Background: Enthesitis related arthritis (ERA) is the subtype of juvenile idiopathic arthritis most closely related to adult spondyloarthritis (SpA).

Conclusions: In ERA, increased circulating levels of sCD163 and activation of the CD163/Robo4 antiangiogenic axis may contribute to peripheral microangiopathy since the very early phase of the disease.

Disclosure of Interest: None declared

NSAID or corticosteroids resulted in resolution of the disease flare. In one severe case canakinumab (anti-interleukine-1β antibody) was successfully used.

**Abstract OP0098 – Table 1**

<table>
<thead>
<tr>
<th>NLRP12 mutation</th>
<th>ID, sex</th>
<th>NLRP12 related symptoms</th>
<th>Comorbid disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>p.H304Y</td>
<td>PK, f</td>
<td>PF, 1/week</td>
<td>CVID, AIHA, pancytopenia, S, ILD Severe CD, food and drug allergy</td>
</tr>
<tr>
<td>p.Ala5Glu</td>
<td>HT, m</td>
<td>PF, eye pain</td>
<td>Meningitis, septic shock, otitis,</td>
</tr>
<tr>
<td>p.Arg723Gln</td>
<td>BA, f</td>
<td>PF, A</td>
<td>Bacterial meningitis</td>
</tr>
<tr>
<td>p.Val936Leu</td>
<td>QA, f</td>
<td>PF (infections)</td>
<td>Recurrent purulent sinusitis</td>
</tr>
<tr>
<td>p.R738fs</td>
<td>SD, m</td>
<td>1–4d of PF, cold-induced, 3–4/ year, pneumonia, febrile seizures, brain oedema</td>
<td></td>
</tr>
</tbody>
</table>

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**OP0099 REMISSION IN JIA CAN BE PREDICTED FROM ONE YEAR FOLLOWING DIAGNOSIS IN A UK MULTICENTRE INCEPTION COHORT**

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**Background:** One of the primary aims for children and young people with JIA is remission. Early improvement in disease likely predicts greater chance of remission in the longer-term.

**Objectives:** To assess the association between change in disease activity over the first year of disease and later occurrence of remission.

**Methods:** Children and young people with oligoarticular, RF-negative or RF-positive polyarticular JIA recruited to the Childhood Arthritis Prospective Study prior to January 2014 were selected for analysis. Two definitions of remission based on clinically inactive disease at two consecutive follow-ups were explored: 1) Wallace’s preliminary criteria and 2) the clinical Juvenile Arthritis Disease Activity Score in 10 joints (cJADAS10). Association between changes in JIA core outcome variables and pain over the first year of disease and later occurrence of remission were analysed using logistic regression models, adjusting additionally for age, gender, symptom duration and predictor value at presentation. Oligoarticular and polyarticular JIA were modelled separately. Multiple imputation accounted for missing data.

**Results:** A total of 1045 children were included. Of these, 70% were female and the majority had oligoarticular JIA (67%). The median age at presentation was 7 years (IQR 3, 11). Within these years, 25% achieved remission according to Wallace’s criteria and 39% according to cJADAS10. More children with oligoarticular JIA achieved remission than those with polyarticular JIA (table 1).