SOLUBLE CD163 AS A POTENTIAL BIOMARKER IN STIMULATED MONOCYTE-DERIVED MACROPHAGES

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 as potential biomarkers. 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 ±SD 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 controls. Serum and urinary sCD163 concentrations were measured by commercially available ELISA kit (R and D systems) and evaluated for their significance as potential biomarkers. Statistical analysis was carried out using Mann-Whitney U test and the relationship between parameters was statistically examined by Spearman’s rank test.

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 sCD163 levels that should be prioritised for further studies as compared to urinary concentrations conversely to what has been described in lupus. Our results further support the M2 macrophages/CD163 signalling system may play a role in the pathogenesis of SSSc. 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

DOI: 10.1136/annrheumdis-2018-eular.4610

OP0096

ADENO-ASSOCIATED VIRUS VECTOR-MEDIATED 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.

For 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

DOI: 10.1136/annrheumdis-2018-eular.2090