prevalence of pulmonary involvement, including interstitial lung disease (ILD), in patients with primary SS widely varies from 8% to 20%.¹ ²Patients with SS with ILD have high morbidity and a fourfold increase in mortality.³

Objectives: Krebs von den Lungen-6 (KL-6) is a mucin-like, high-molecular-weight glycoprotein; it is expressed in regenerating type II pneumocytes. Serum KL-6 is highly associated with the activity of ILD in patients with radiation pneumonitis, idiopathic pulmonary fibrosis, hypersensitivity pneumonitis, sarcoidosis, rheumatoid arthritis, polymyositis, dermatomyositis and systemic sclerosis.⁴⁻⁸ The increase in serum KL-6 level might reflect the increase in the number of regenerating type II pneumocytes secondary to pulmonary damage.⁸ We hypothesise that an early pulmonary damage occurs before clinical or radiological evidence of ILD in patients with SS.

Methods: In this retrospective case-control study, patients who were diagnosed with primary SS and fulfilled the American-European Consensus Group Criteria for Sjögren's Syndrome were included. Clinical information, laboratory results on inclusion, images and pulmonary functions test results were recorded via electronic medical records review. Pulmonary radiography including chest X-ray and chest computed tomography was reviewed by a chest physician.

Results: Of the 39 patients with SS, 21 (53.85%) developed ILD at the end of follow-up. The follow-up period was 2.65 ± 1.88 years. The time to diagnosis of ILD was 2.72 ± 1.74 years in the ILD group. The serum KL-6 level was 1920.10 ± 1974.26 U/ml in the ILD group and 894.11 ± 788.53 U/ml in the non-ILD group ($p = 0.001$). The diffusing capacity of the lungs for carbon monoxide was 70.76 ± 19.63 mmHg and 91.88 ± 12.02 mmHg in the ILD group and non-ILD group, respectively ($p < 0.001$).

Conclusion: Serum KL-6 level is significantly higher in patients with primary SS with developing ILD and may represent an early non-radiographic pulmonary damage. Serum KL-6 can be a valuable biomarker in predicting the development of ILD in patients with primary SS.

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FRI0229

SOLUBLE CD163 IS A BIOMARKER FOR ACCELERATED ATHEROSCLEROSIS IN SLE PATIENTS AT APPARENT LOW RISK FOR CARDIOVASCULAR DISEASE

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Background: Cardiovascular disease (CVD) due to accelerated atherosclerosis is the leading cause of death in SLE. Probably because they do not incorporate important risk factors such as overweight, chronic kidney disease or systemic inflammation, models for predicting CVD established in the general population (such as Framingham model) underestimate the cardiovascular risk in SLE. Identification of biological markers for CVD may help to classify high-risk subjects and set up targeted prevention in SLE.

Objectives: Our study aimed to determine whether sCD163, a soluble macrophage marker upregulated in numerous inflammatory disorders, might be predictive of accelerated atherosclerosis associated with SLE

Methods: Carotid ultrasound was prospectively performed in 63 consecutive SLE patients asymptomatic for cardiovascular disease (CVD) and 18 volunteer health-workers. Ultrasound was performed at baseline and during follow up. Serum level of sCD163 was determined at baseline using ELISA. The primary outcome was the presence of a carotid plaque. Factors associated with carotid plaques were identified through multivariate analysis.

Results: Despite a low risk for cardiovascular events according to Framingham score in both groups ($2.1\% \pm 3.8$ in SLE vs $2.1\% \pm 2.9$ in controls; $p=0.416$), ultrasound study at baseline showed a carotid plaque in 23 (36.5%) SLE patients versus 2 (11.1%) controls ($p=0.039$). Multivariate analysis showed that SLE status increased the risk for carotid plaque by a factor of 9 ($p=0.017$). In SLE patients, sCD163 level was high ($483.7\text{ng/ml} \pm 260.8$ versus $282.1\text{ng/ml} \pm 97.5$ in controls; $p<0.001$) and independently associated with carotid plaques as assessed by stratification based on sCD163 quartile values ($p=0.009$), receiver operating characteristic (ROC) ($p=0.001$) and multivariate analysis ($p=0.015$). Eventually, sCD163 at baseline was associated with the onset of carotid plaque during follow up (3 ± 1.4 years) in SLE patients who had no carotid plaque at first evaluation ($p=0.041$)

Conclusion: sCD163 is associated with progressing carotid plaque in SLE and may be a useful biomarker for accelerated atherosclerosis in SLE patients at apparent low risk for CVD

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FRI0230

CLINICAL AND ECHOCARDIOGRAPHIC CHARACTERISTICS OF MYOCARDIAL INJURY IN SYSTEMIC LUPUS ERYTHEMATOSUS, CLASSIFIED ACCORDING TO CARDIAC MAGNETIC RESONANCE CRITERIA

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Background: Lupus myocarditis (LM) occurs in 5-10% of patients with systemic lupus erythematosus (SLE). Subclinical myocardial inflammation occurs in 37% at post mortem.¹ Echocardiographic strain (STE) supports subclinical myocardial dysfunction in SLE.² Tissue characterisation by cardiac magnetic resonance (CMR) identifies myocardial inflammation, necrosis and/or fibrosis, detecting clinical and subclinical myocardial injury (MIN) in SLE. It's the non-invasive gold standard for diagnosing myocarditis (all types) based on the Lake Louise criteria (LLC).³