prevalence of pulmonary involvement, including interstitial lung disease (ILD), in patients with primary SS widely varies from 8% to 20%.[1,2]Patients 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% ± 3.8 in SLE vs 2.1% ± 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 (483.7ng/ml ± 260.8 versus 282.1ng/ml ± 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 RESONANCECRITERIA

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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]

Objectives: Determine prevalence of MIN in SLE (LLC).
Compare clinical and echocardiographic (echo) features of patients with and without MIN.
Identify echo predictors of MIN.
Methods: A prospective crossectional study was done at Tygerberg Hospital, Western Cape, South Africa. Adult inpatients, fulfilling the 2012 SLICC criteria were screened. Echo analyses included STE and regional function (wall motion score (WMS)). Patients were grouped according to evidence of MIN (absent criteria [AC]; single criterion [SAC]; fulfilling LLC), comparing clinical, laboratory and echo data. Logistic regression and ROC were used to determine predictors of MIN.
Results: 49/106 SLE patients screened were included (Figure 1). 46.9% of patients had MIN (≥1 criterion): 12.2% fulfilled LLC for LM and 34.7% had a SAC. SLE disease activity (SLEDAI) (p=0.022) was higher in patients fulfilling LLC, but not in the SAC group. A clinical and echo diagnosis of LM was made in all patients fulfilling LLC, in 17.6% of patients in the SAC group and none in the AC group (Table 1). Anti-DsDNA and anti-B2GP1 were more frequently positive in SAC vs the AC group (p=0.054 and 0.081). WMS was higher in LLC and SAC groups (p=0.006;p=0.083) with mid and basal STE more impaired in patients with MIN (p=0.047;p=0.043). LVID and mid STE score combined was the best predictor of MIN (Table 2; Figure 2).
Conclusion: CMR evidence of MIN is common in SLE, even in the absence of clinical myocardial dysfunction or high lupus activity. Impaired echo regional and global function occurs more frequently in patients with MIN. STE combined with LVID predicts MIN detected by CMR and has potential as a cost effective screening tool. CMR is limited by a high exclusion rate in SLE, mainly due to renal impairment.

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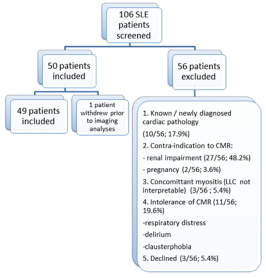


Figure 1

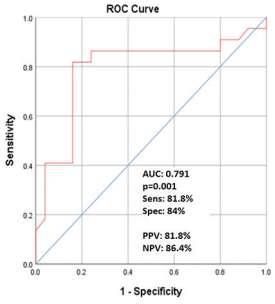
Table 1

	AC n=26	LLC n=6	p	SAC n=17	p
			Med (IQR)		
Age	27 (21-35)	27 (23-28)	1	29 (23-36)	0.542
SLE duration, days	114(6-1366)	35(31-44)	0.724	955(7-2101)	0.486
SLEDAI	13(9-15)	22(16-26)	0.022	12(9-20)	0.813
Antiphospholipid syndrome	2 (8)	0	0.483	4 (24)	0,085
			n (%)		
Female	24 (92)	5 (83)	0.497	14(82)	0.319
Cardiac risk factors	8 (31)	1 (17)	0.489	6 (35)	0.757
Nephritis	9(35)	3(50)	0.483	3(18)	0.225
Clinical LM	0	6(100)	<0.001	3(18)	0.026

Table 2

Logistic regression			
		Univariate	
Echo variable	**OR**	**95% CI**	**p**
LVID	2.6	0.8-8.4	0.109
LV ejection fraction	0.9	0.8-0.9	0.018
Wall motion abnormality	4.2	1.3-13.9	0.018
GLS	1.3	1-1.6	0.049
STE score Basal	1.6	0.9-2.7	0.068
Mid	1.9	1.2-3.2	0.006
Apical	1	0.9-3.9	0.083
		Multivariate	
LVID	3.5	0.9-13.3	0.070
Mid STE score	2.1	1.2-3.5	0.008

Figure 2



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I have received a donation form ROCHE in 2018 for upgrading of our outpatient clinical area in Tygerberg Hospital., Phillip Herbst: None declared, Alfonso JK Pecoraro: None declared, Christelle Ackerman: None declared, Anne-Marie du Plessis: None declared, Helmuth Reuter Speakers bureau: Received Honorarium as speaker at regional meetings, South Africa, Anton F Doubell: None declared
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FRI0231

IN PATIENTS WITH UCTD, IFN ACTIVITY IDENTIFIES PATIENTS THAT PROGRESS TO DEFINITIVE CTD CLASSIFICATION CRITERIA

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Background: Undifferentiated connective tissue disease (UCTD) is a term used to describe individuals with positive autoantibodies and symptoms but do not meet criteria for an established autoimmune CTD (AI-CTD). The prognosis of UCTD is unclear, could result in disease stability, remission or progression. As this condition is understudied, there is an unmet need for biomarkers to stratify those who progress to allow for a more aggressive therapy to be employed. IFN plays a major role in the pathogenesis of various AI-CTDs. However there are limited data on its role in AI-CTD.
Objectives: To determine whether IFN assays can predict progression from UCTD to meeting classification criteria of established AI-CTDs.
Methods: A prospective observational study was conducted on 43 consecutive patients with UCTD; as defined by Mosca criteria [1]. Progression was defined by meeting 2012 ACR/SLICC SLE, 2016 ACR/EULAR Primary Sjogren's, Bohan-Peter, 2013 ACR/EULAR Scleroderma, or 2006 Sydney Antiphospholipid syndrome criteria. For the interferon scores, RNA was extracted from PBMCs and a custom Taqman array was used to measure expression of 30 interferon stimulated genes (ISGs) as previously described [2]. A two-score system of ISGs (IFN-Score-A and Score-B), was calculated without the knowledge of participants' clinical status.
Results: Mean (range) age of the cohort was 48 (39-58) years; Female 36 (84%); mean disease duration 5.5 years; concomitant DMARDs including anti-malarials 38 (88%), concomitant corticosteroid 2(5%) and internal organ involvement 9 (21%). At the follow-up visit, 9/43 (21%) met established classification criteria for AI-CTD [SLE=6; IIM=2; and SSc=1]. Comparing the IFN scores between those who progressed (n=9) vs those remained undifferentiated (n=34); the IFN-Score-A was higher in the progressor group; fold difference (FD) 2.76 (95% CI 1.11-6.88); p=0.030. While for IFN-Score-B, there was a trend to association for higher expression in the progressor group vs remained undifferentiated; FD 1.74 (0.53-3.21); p=0.077.
Conclusion: IFN assays can identify progression to classifiable CTD within cohorts of UCTD. There is a potential role for the use of IFN biomarkers in the prognostication of patients with UCTD.