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

Clemence David¹, Rachid Abbas², Brigitte Escoubet¹, Benedicte Giroux Leprieur², Anne Boutten¹, Thomas Papo³, Monique Dehoux¹, Karim Sacre¹, ¹Université Paris Diderot, Paris, France; ²Institut Gustave Roussy, Villejuif, France; ³Université Paris XIII, Bobigny, France

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. 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 (483.7 ng/ml ± 260.8 versus 282.1 ng/ml ± 97.5 in controls; p<0.01) 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.

Disclosure of Interests: None declared, Rachid Abbas: None declared, Brigitte Escoubet: None declared, Benedicte Giroux Leprieur: None declared, Anne Boutten: None declared, Thomas Papo: None declared, Monique Dehoux: None declared, karim sacre Grant/research support from: GSK


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

Riette du Toit¹, Philipp Herbst², Alfonso J.K. Pecoraro³, Christelle Ackerman³, Anne-Marie du Plessis¹, Helmut Reuter⁴, Anton F. Doubell², ¹Stellenbosch University, Department of Radiology, Cape Town, South Africa; ²Stellenbosch University, Department of Cardiology, Cape Town, South Africa; ³Stellenbosch University, Department of Radiodiagnosis, Cape Town, South Africa; ⁴Stellenbosch University, Division of Clinical Pharmacology, Cape Town, South Africa

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

Disclosure of Interests: None declared


REFERENCES: