with current DUs was 3-fold lower than DAVIX of patients without DUs (0.18 vs 0.63 p=0.0093). Further, DAVIX of patients with positive history of DUs was 50% lower than in patient with a negative history (median 0.34 vs 0.64, p=0.0052). In patients without current DUs, DAVIX of patients who developed new DUs within 12 months of follow-up was 3-fold lower than in patients who didn’t develop DU (0.21 vs 0.65, p=0.0156). ROC curve analysis indicated that DAVIX threshold <0.49 conferred a 4 times higher risk of developing new DUs (67%) compared to overall risk of our population 17.6%.

Conclusion: Outcome measures of vascular involvement in SSCs are scanty. We demonstrated that DAVIX is a promising and feasible surrogate outcome measure of neointima proliferation in SSC and a useful imaging biomarker of vascular disease activity. The predictive value of DAVIX for the future onset of DU could be employed as a useful stratification tool in clinical trials. The value of DAVIX in predicting the worsening of PROs and clinical parameters in overall patients, may offer insights on the role of vascular disease activity in the overall progression of SSC.

References:


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THU0342

DECLINE IN SUBCLINICAL SYSTEMIC SCLEROSIS PRIMARY HEART INVOLVEMENT ASSOCIATES WITH POOR PROGNOSTIC FACTORS AND ACTIVE INTERSTITIAL LUNG DISEASE

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Background: Primary systemic sclerosis heart involvement (pSSc-HI) is described in the majority of SSC patients when sensitive methods such as cardiovascular magnetic resonance (CMR) are used. The natural history of these subclinical findings are unknown.

Objectives: To evaluate for interval change in subclinical pSSc-HI, the association between change in CMR abnormalities and disease phenotype and whether disease modifying antirheumatic (DMARD) and/or vasodilator treatment influence the CMR course.

Methods: SSC patients, fulfilling the 2013 ACR/EULAR criteria, with no cardiovascular (CV) disease, diabetes and no more than 2 CV risk factors had two CMRs performed (V1 & V2; minimum 1 year apart). A 3T CMR with late gadolinium enhancement (LGE), T1 mapping for extracellular volume (ECV) of diffuse fibrosis quantification and stress perfusion was undertaken.

Results: 31 SSC patients were evaluated, with median (IQR) follow up (between the 2 CMR scans) of 33 (17, 37) months. Median (IQR) age was 52 (47,60), 32% had diffuse cutaneous SSC, 52% interstitial lung disease (ILD), 29% Scl70+. 4/31 patients had a non-isoaemic LGE pattern suggesting focal fibrosis at V1, with no change in the pattern, distribution, or median (IQR) LGE scar mass between V1 and V2 [1.88 (1.01, 6.34) vs 1.70 (1.21, 4.18)]. At V2, 2 additional patients showed focal fibrosis, of which one had an episode of clinically diagnosed myocarditis. No significant change in ECV, T1 native, myocardial perfusion reserve (MPR) or left ventricle (LV) volumes and function were noted at V2 compared with V1 (p>0.01).

SSc patients with either increase in pre-existing LGE scar mass (n=1) or new fibrosis were all dCSSc, with ILD, 2 Scl70+. A reduction in forced vital capacity and total lung capacity associated with a reduction in LV ejection fraction (LVEF) (rho=0.413, p=0.021; rho=0.335, p=0.07) and MPR (rho=0.543, p=0.007; rho=0.627, p=0.002).

Patients receiving DMARD treatment had higher baseline LV end-diastolic volume compared to those with no DMARD treatment [mean (SD) 78 (19) vs 69 (10), p=0.167]. A decrease in LV stroke volume and an increase in T1 native at V1 vs V2 was noted for those on DMARD [mean (SD) 49 (8) vs 46 (8), p =0.023; 1208 (65) vs 1265 (56), p=0.008 respectively] (Figure 1). No significant change in CMR measures in those receiving vasodilator or angiotensin-converting-enzyme inhibitor treatment was noted (p>0.01).

Conclusion: This first, pilot longitudinal study of CMR-defined subclinical pSSc-HI suggests largely stable appearances with follow-up. Progression of new focal fibrosis and decline in LV function and MPR, where observed, associated with poor prognostic factors of SSC and ILD progression. Consistent with this, individuals on DMARD appeared to show interval decline. Larger longitudinal studies are warranted to confirm these findings and inform on utility of CMR monitoring of subclinical pSSc-HI in poor prognosis SSC.

References:


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THU0343

AUTOANTIBODIES CAN PARTLY PREDICT SEVERITY OF DAMAGE BUT NOT EXTENT IN PATIENTS WITH IDIOPATHIC INFLAMMATORY MYOSITIS

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Background: Patients with idiopathic inflammatory myopathies (IIM) might suffer from irreversible damage once inflammation has decreased. Autoantibodies are found in up to 80% of patients with IIM and are coupled with specific clinical features. Whether autoantibodies can be used as biomarkers to predict patterns of damage in IIM remains unknown.

Objectives: To investigate the association between autoantibodies and organ damage in patients with IIM using longitudinal national register data.

Methods: Data were retrieved from the electronic Swedish Rheumatology Quality Register (SRQ). Patients (n=302) with a clinical diagnosis of IIM (2017-2020). The study used a longitudinal approach.”

Figure 1. Mean (SD) of T1 native, LSVI/BSA, LVEF, and LVEDV/BSA at V1 compared to V2 in those with and without DMARD treatment.BSA, body surface area; DMARD, disease modifying antirheumatic drugs; EDV, end-diastolic volume; SV, stroke volume; LV left ventricular; EF, ejection fraction.

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