autoantibody profile including myositis specific autoantibodies (MSAs) and myositis associated autoantibodies (MAAs).
Results: Compared to HCs, patients with IIMs more frequently had prolongation of QTc (p=0.037) and QRS (p=0.031). All patient groups had significantly longer QTc and QRS duration than HCs. In multivariate regression analysis of patients with IIMs, increased CRP (p=0.006) was associated to increased QTc. Pooled data for patients with IIMs and HCs showed an association between diagnosis of IIM and increased QTc duration (p<0.001). An association between presence of any MAA and QRS duration appeared in patients with IIMs (p=0.019). In pooled data for patients with IIMs and HCs, diagnosis of IIM was associated with QRS duration (p=0.001).

Table: Multivariate regression analyses of explanatory factors for QTc duration (ms)

Risk factor	OR	95% CI		P-value	OR	95% CI		P-value
Gender, male	0.46	0.28	0.75	0.002	0.46	0.29	0.72	0.001
Age at inclusion, per year	1.02	0.99	1.04	0.068	1.02	1.00	1.04	0.047
Hypertension, no vs. yes	1.63	0.93	2.86	0.086	1.58	0.97	2.60	0.069
Former/present smoker, no vs. yes	1.35	0.84	2.17	0.213	1.57	1.02	2.41	0.041
Increased CRP, no vs. yes	2.17	1.25	3.75	0.006	1.98	1.15	3.40	0.013
BMI, kg/m^2				NS				NS
Dyspnea (NYHAII-IV), no vs. yes				NS				NS
Presence of MSAs/MAAs, no vs. yes				NS	NA	NA	NA	NA
HAQ (Health Assessment Questionnaire), 0–3				NS	NA	NA	NA	NA
IIM, no vs. yes	NA	NA	NA	NA	4.11	2.21	7.63	<0.001

Conclusions: Patients with IIMs, no matter of clinical subgroup, had a higher occurrence of cardiac abnormalities detected by ECG than HCs. Increased CRP and presence of any MAA were associated with increased QTc and QRS duration, respectively. These results support our notion of possible associations between inflammation and autoimmunity and cardiac affection in patients with IIMs. There is now a pressing need to set up a larger prospective study to validate the present findings.
Disclosure of Interest: None declared
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SAT0499 ASSOCIATION BETWEEN SYSTEMIC SCLEROSIS AND OTHER SYSTEMIC AUTO-IMMUNE DISEASES: STUDY IN TWO UNIVERSITY HOSPITALS COHORTS

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Background: Association between systemic sclerosis (SSc) and another autoimmune systemic disease (AISD) in the same patient seems to be more frequent than each disease's prevalence would explain.
Objectives: The aim of our work was to describe patients presenting an overlap syndrome from 2 french cohorts and to compare their characteristics with patients presenting SSc alone.
Methods: This was a retrospective observational study conducted in two french auto-immune diseases reference centre (Strasbourg and Bordeaux). Patients responding to the 2013 ACR-EULAR scleroderma classification criteria for SSc were screened for concomitant AISD. Patients satisfying 2010 ACR-EULAR diagnostic criteria for rheumatoid arthritis (RA) and/or 2016 ACR-EULAR classification criteria for Gougerot-Sjögren syndrome (GSS) and/or 2012 SLICC systemic lupus erythematosus (SLE) classification criteria were included in our study. Patient, disease, and treatment characteristics were retrospectively retrieved from medical records and were compared to a SSc control cohort for Bordeaux University Hospital.
Results: A population of 534 SSc patients was studied. Thirty-four (6.4%) patients were identified as having overlap syndrome. There was 21 (62%, prevalence 3.9%) patients with RA, 14 (41%, prevalence 2.6%) with GSS and 4 (12%, prevalence 0.7%) with SLE (5 patients had 2 AISD).
There were 24 (71%) patients with limited cutaneous SSc. Median Rodnan was 6 (extreme 0–42), 13 (38%) patients had interstitial pneumonia and 9 (26%) presented lung fibrosis. Three patients had pulmonary arterial hypertension (PAH) confirmed with catheterism. Seventeen patients (50%) had anti-centromere Ab, 11 (32%) had anti-Scl70 Ab whereas none had anti-RNA-polymerase III Ab (investigated in 24 patients).
Concerning RA patients, 17 (81%) were ACPA-positive and 17 (81%) had erosive disease. Only 6 (29%) were in remission according to Boolean criteria and 13 (62%) had a DAS28-CRP≤3.2 suggesting difficult-to-treat RA. Patients with GSS all presented sicca syndrome, 8 (57%) had a Chisholm grade ≥3 on accessory salivary gland biopsy and 12 (86%) had positive anti-SSA Ab. In patients with SLE, 3 (75%) had positive anti-DNA Ab and one had a grade IV kidney disease. Three (75%) patients with SLE had a SLICC organ-damage score ≥5 suggesting severe SLE.
Compared with our control cohort, patients with overlap syndrome had higher frequency of corticosteroid, immunosuppresive and biologic therapy use (85.3% vs 45%; 70.6% vs 31.3%; 52.9 vs 3.8%; p<0.0001 for all comparisons).
Conclusions: Association of SSc and another auto-immune systemic disease is present in more than 6% of patients. These patients might have a more severe disease than usual SSc patients requiring prompt diagnosis and adequate treatment.
Disclosure of Interest: None declared
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SAT0500 MYOCARDIAL INVOLVEMENT AT MAGNETIC RESONANCE IN PATIENTS WITH SYSTEMIC SCLEROSIS AND MINOR ARRHYTHMIAS: ASSOCIATION WITH CLINICAL FEATURES AND IMPACT OF TREATMENT

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Background: Systemic sclerosis (SSc) is an autoimmune fibrotic disease characterised by variable clinical manifestations based on the predominant organ involvement. With the exceptions of acute fulminant myocarditis with pericardial effusion and heart failure, myocardial involvement in SSc is clinically silent until arrhythmias appear, while representing the major cause of sudden death or atrioventricular (AV) blocks in SSc. There are no reliable screening algorithm to discriminate myocardial involvement nor established treatments.
Objectives: To identify early/clinically silent myocardial involvement in SSc and define a possible treatment.
Methods: We used Holter electrocardiography (ECG) to investigate our cohort of patients with SSc (n=221, all fulfilling the ACR classification criteria) for ventricular ectopic beats (VEB)/AV blocks/unexplained tachycardia as sensitive signs of myocardial involvement. In 24 patients (women 23%–95.8%, anti-centromere-ACA 16%–66.6%, anti-topoisomerase-aScl70 3%–12.5%, anti-RNA polimerase III 3%–12.5%, anti-Ku 2%–8.3%, limited skin disease-lSSc 12%–50%, diffuse skin disease 5%–20.8%, *sine scleroderma* 7%–29.2%, median age 66.5 years, IQR 57.8–72.5), never treated with anti-fibrotic agents, we performed heart magnetic resonance (hMR) searching for myocardial oedema (on T2 STIR sequences, using the signal ratio between myocardium and skeletal muscle with a cut-off value of ≥1.9 for oedema) or fibrosis (presence of any late gadolinium enhancement (LGE) with intramyocardial or subepicardial pattern). Patients with myocardial oedema at hMR were treated with mycophenolate mofetil (MFM) 2 g/day or with azathioprine (AZA) 100 mg/day and underwent a control hMR after six months.
Results: In 10/24 (42%) of the SSc cases with Holter ECG alterations (8 VEB, 1 tachycardia, 1 type II AV block) we observed SSc myocardial involvement at hMR. In more detail, 6 patients had myocardial oedema at T2 STIR sequences (including cases as follows: 1 aScl70-positive with lSSc and lung fibrosis, 2 ACA-positive with lSSc, 3 ACA-positive *sine scleroderma*), and 4 only fibrosis (1 Scl70-positive with lSSc and lung fibrosis, 1 ACA-positive with lSSc, 1 ACA-positive *sine scleroderma,* 1 anti-Ku-positive *sine scleroderma*). At 6 months of medical treatment, myocardial oedema disappeared in 3 patients treated with Aza and in 1 treated with MFM, the additional 2 patients are still receiving MFM treatment at the time of analysis.
Conclusions: We observed that 42% of patients with SSc and minor arrhythmias on 24 hour Holter ECG have clinically silent myocardial involvement at hMR; those with potential reversible disease (oedema rather than fibrosis) favourably respond to immunosuppressants. Detectable serum ACA and the absence of skin involvement are over-represented in this subgroup of patients.
Disclosure of Interest: None declared
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SAT0501 EARLY VERSUS LATE-ONSET SYSTEMIC SCLEROSIS: ARE THERE CLINICAL AND IMMUNOLOGICAL DIFFERENCES?

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Background: The clinical course of Systemic Sclerosis (SSc) depends on subtype, organ involvement and age. Peak age at onset of SSc is between 30 and 50