intestinal) and time since non-Raynaud phenomenon. Clinical characteristics and prevalence of auto-antibodies within clusters was evaluated. We assessed whether adding autoantibody status as additional factor improved clinical subsetting.

Results: Of 407 SSc patients included, 371 patients (91%) fulfilled ACR/EULAR 2013 criteria. Prevalence of anti-centromere (ACA) was 37%, anti-topoisomerase (ATA) 24%, anti-RNAPIII 5%, anti-fibrillarin 4%, anti-Pm/Sci 5%. Cluster analysis identified clinically recognisable clusters of SSc patients based on absence of skin involvement (cluster 1), peripheral vascular involvement (cluster 2), fibrotic complications (cluster 4), and severe vascular complications (pulmonary arterial hypertension [PH], renal crises; cluster 5). Except for cluster 4, where ATA was dominant, in all clusters ACA was the most prevalent auto-antibody. No cluster associated with any of the more rare SSc specific auto-antibodies. Adding of auto-antibodies resulted in increased clinical overlap, with a frequency of PH between 6 – 8% and a frequency of digital ulcers between 24 – 39% in clusters 2,4 and 5.

Table 1. Clustering of SSc patients based on clinics

	Cluster 1 (n=66)	Cluster 2 (n=50)	Cluster 3 (n=129)	Cluster 4 (n=81)	Cluster 5 (n=81)
Age in years, mean (SD)	53 (14)	54 (14)	54 (14)	53 (16)	62 (12.4)
Female, % (n)	90 (60)	86 (43)	85 (110)	72 (58)	74 (60)
Time since non-Raynaud, median (range)	3 (-3-14)	10 (-3-44)	2 (0-20)	3 (0-27)	13 (0-46)
dcSSc, % (n)	0 (0)	2 (1)	17 (22)	65 (53)	25 (20)
Mean mRSS (SD)	0 (0)	4 (3)	4 (3)	14 (11)	4 (6)
PAH, % (n)	3 (2)	4 (2)	0 (0)	5 (4)	20 (16)
DLCO, %predicated, mean (SD)	70 (18)	66 (16)	70 (15)	64 (16)	54 (18)
History of renal crisis, % (n)	0 (0)	0 (0)	0 (0)	3 (2)	17 (14)
Digital ulcers, % (n)	15 (10)	98 (49)	4 (5)	28 (23)	6 (5)
Anti-topoisomerase, % (n)	5 (3)	15 (7)	22 (28)	48 (37)	24 (19)
Anti-centromere, % (n)	62 (39)	48 (23)	41 (53)	8 (6)	30 (24)

Conclusions: Based on clinical data alone, relevant subgroups in SSc can be distinguished. Adding antibody status to cluster analyses increases overlap of clinical features between subgroups. These findings show that solely presence of SSc specific antibodies is of limited clinical relevance. For optimal risk stratification more complex serological findings as antibody titers might be additive.

Disclosure of Interest: None declared

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SAT0351 DIFFERENCES AMONG PATIENTS WITH INTERSTITIAL LUNG DISEASE ACCORDING TO THEIR SYSTEMIC SCLEROSIS SUBCLASSIFICATION

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Background: Systemic Sclerosis (SSc) has been widely studied from a purely global standpoint, but only a few trials have analysed patients with interstitial lung disease (SSc-ILD) as well.

Objectives: 1. Describe the clinical features of a cohort of patients with SSc and SSc-ILD. 2. Determine whether there are disparities among different types of SSc-ILD and their progression in patients with either limited (lcSSc) or diffuse involvement (dcSSc). 3. Ascertain whether there are disparities among different types of SSc-ILD and their progression according to the SSc-specific autoantibody (AAb).

Methods: Retrospective study of a cohort of patients with ILD-SSc controlled during an SSc consultation. The following variables were collected: sex, age, SSc and ILD progress in years, type of SSc and ILD, smoking, digital ulcers (DU), pulmonary hypertension, digestive disorders, cancer, SSc treatment, corticosteroid doses and lastly, lung function tests upon diagnosis, at treatment onset, and 24 months later. Additionally, a record was kept on the types of AAb present in every SSc. Qualitative and quantitative variables were compared in relation to the clinical and immunological sub-classification. Chi-square and Student's T Tests were performed. A p-value <0.05 was considered significant.

Results: out of 266 patients with SSc, data from 47 patients with ILD-SSc were gathered; 89.4% were female, with an age range of 66.09±15.1 years old, and 9.85±10.2 and 4.38±9.24 years of progression of their SSc and ILD respectively. 33 out of 47 sustained lcSSc, and both Sci-70/ATA (29.8%) and ACA (26.1%) were the most frequently found AAb. Non-specific interstitial pneumonia (NSIP) was the most common ILD radiological pattern (76.6%). Most patients with SSc underwent treatment (51.1%), 24% with mycophenolate mofetil (MMF); 36.2% of the patients had been concomitantly administrated corticosteroids with a mean prednisone dose of 15.73±10.3 mg/d.

Upon comparing patients with lcSSc and dcSSc, prevalence of DU was higher in those with dcSSc (p<0.01), MMF was less frequently used (p<0.02), rituximab was more usually employed (p<0.03), and they presented worse values of FEV1/FVC ratio after 24 months of treatment (p<0.03). No differences were observed as to either type of ILD or progression. However, when variables were analysed regarding AAb in SSc, patients with ACA presented both fewer DU (p<0.02) and NSIP pattern (p<0.02), and more frequent compromise of the small airway (p<0.01), they were younger and thus, they had had shorter progression

of the disease. ILD diagnosis was made significantly earlier in those patients with RNA polymerase, and later in those with anti-U1RNP. No AAb was observed associated with neoplasia. Considering the types of ILD, patients with NSIP pattern were younger (p0.054) and presented worse spirometric values.

	ILD-SSc (n=47)	lcSSc (n=33)	dcSSc (n=14)	P (lcSSc vs dcSSc)	P (ACA vs no ACA)	P (ATA vs no ATA)	P U1RNP vs nonU1RNP	P RNApol vs nonRNApol
Age ± SD (years)	66.09± 14.1	67.09± 14.04	63.71±15.67	Ns	<0.05	Ns	Ns	Ns
Progress in years SSc ± SD	9.85± 10.29	8.81±15. 04	12.31±9.59	Ns	<0.04	Ns	<0.02	Ns
Evolution in years ILD ± SD	4.38± 9.24	4.11±9.7 2	5.02± 8.29	Ns	Ns	Ns	<0.0001	<0.005
NSIP (%)	36(76.6)	24 (72.72)	12(85.71)	Ns	<0.02	Ns	Ns	Ns
IPF (%)	3 (6.4)	1 (3.03)	2(14.28)	Ns	Ns	Ns	Ns	Ns
LIP (%)	2 (4.3)	2 (6.06)	0	Ns	Ns	Ns	Ns	Ns
Small airway compromise(%)	4 (8.5)	4(12.12)	0	Ns	<0.005	Ns	Ns	Ns
PH (ultrasound)	3 (6.4)	3 (9.09)	0	Ns	Ns	Ns	Ns	Ns
Digital ulcers (%)	17 (36.2)	7 (21.21)	10(71.42)	<0.01	<0.02	<0.01	Ns	Ns
Neoplasia (%)	5 (10.6)	3 (9.09)	2(14.28)	Ns	Ns	Ns	Ns	Ns
Immunosuppressive treatment (%)	24 (51.1)	18 (54.54)	6(42.85)	Ns	Ns	Ns	Ns	Ns
Use of cyclophosphamide (%)	0 (17)	4 (12.12)	4(28.57)	Ns	Ns	Ns	Ns	Ns
Use of MMF (%)	16 (34)	15 (45.45)	1(7.14)	<0.02	Ns	Ns	Ns	Ns
Use of azathioprine (%)	1(24)	0	1 (7.14)	Ns	Ns	Ns	Ns	Ns
Use of rituximab (%)	3 (6.4)	0	3 (21.42)	<0.03	Ns	<0.03	Ns	Ns
Use of corticoids (%)	17 (36.2)	13 (39.39)	4 (28.57)	Ns	Ns	Ns	Ns	Ns
Doses of corticoids ± SD (mg/d)	15.73± 10.31	14.42± 9.79	20± 12.24	Ns	<0.02	Ns	Ns	Ns
%FVC ±SD upon diagnosis of SSc	82.29± 14.6	84.5± 13.41	77.89±16.67	Ns	Ns	Ns	Ns	<0.001
%FVC±SD upon diagnosis of ILD	77.8± 16.09	80.29± 10.13	71.92±13.96	Ns	Ns	Ns	Ns	Ns
%FVC±SD 24 months after diagnosis of ILD	77.65± 20.94	79.85± 22.6	73.45±17.58	Ns	Ns	Ns	Ns	Ns
%FEV1/FVC±SD upon diagnosis of SSc	83.78± 14.07	85.11± 15.71	81.11±10.21	Ns	Ns	Ns	Ns	Ns
%FEV1/FVC±SD upon diagnosis of ILD	87.79± 16.06	89.56± 16.85	83.69±13.79	Ns	Ns	Ns	Ns	Ns
%FEV1/FVC±SD 24 months after diagnosis of ILD	93.25±1 8.93	96.71±1 7.24	81.8± 17.83	<0.03	Ns	0.08	Ns	Ns
%DLCO±SD upon diagnosis of SSc	69.76± 17.8	70.93± 18.46	67.67±17.42	Ns	Ns	Ns	Ns	Ns
%DLCO±SD upon diagnosis of ILD	63.28± 18.31	65.69± 18.75	57.92±16.75	Ns	Ns	Ns	Ns	Ns
%DLCO 24 months after diagnosis of ILD	61.31± 17.47	63± 16.74	58.45±15.68	Ns	Ns	Ns	Ns	Ns

Conclusions: In terms of ILD-SSc patient stratification, sub-classification by AAb appears to be more specific than the clinical sub-classification. ACA is related to less frequency of NSIP pattern. Unlike what has been described for SSc from a global point of view, in patients with ILD-SSc no association between AAb and neoplasia could be established.

Disclosure of Interest: None declared

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SAT0352 MORTALITY IN IDIOPATHIC INFLAMMATORY MYOPATHY- RESULTS FROM A SWEDISH NATIONWIDE POPULATION-BASED COHORT STUDY

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Background: Little is known about mortality in idiopathic inflammatory myopathies (IIM) compared to the general population, especially the risk development since diagnosis. In a recently published study, the 5-, and 10-year mortality was lower for IIM than previously reported, suggesting improved outcomes in recent years.

Objectives: To assess the mortality following IIM diagnosis in a nationwide population-based cohort of IIM patients diagnosed 2002–2011 compared to the general population.

Methods: We used nationwide fully covering health care registers to identify all individuals who were treated by a rheumatologist, neurologist, or dermatologist for IIM and who had a listing of IIM at ≥two visits within 1–12 months between 2002 and 2011, general population comparators, and death during follow up. We calculated mortality rates with 95% confidence intervals (CI) and compared the mortality in IIM to that in the general population using Cox proportional hazards models adjusted for age, sex, year of diagnosis, and residential area.

Results: During a median follow up of 4 years, 228 (31%) of the 733 IIM patients and during a median follow up of 6 years, 888 (12%) of the 7,340 general population comparators died. This corresponded to a crude mortality of 61/1,000 person-years in IIM, and 20/1,000 person-years in the comparators. The cumulative mortality at 1 year after diagnosis was 10% in IIM and 1% in the general population, at 5 years it was 24% in IIM and 7% in general population, and at 10 years the proportion was 31% and 12%. The overall hazard ratio (HR) (95% CI) of death comparing the NPR cohort and its comparator, was 3.5 (3.0–4.0). When restricting the outcome to cardiovascular disease-, cancer-, infection-, or pulmonary disease- specific death we noted increased mortality from all outcomes in IIM compared to the risk in the general population. When we stratified on time since diagnosis we noted an increased absolute and relative risk of death in the first year of diagnosis in particular for pulmonary disease and cancer, whereas cardiovascular mortality was a major cause of death also after 10 years after IIM diagnosis (table).

Conclusions: IIM patients are still at increased risk of death. The highest mortality