Table 1. Siena Myositis Ultrasound Grading Scale (SMUGS).

	Grey-scale edema	Grey-scale atrophy	Power Doppler
0	Normal muscle echotexture with hyperechoic septa and hypoechoic muscle fibers, conserved thickness.	Normal muscle echotexture, with hyperechoic septa and hypoechoic muscle fibers, conserved thickness.	No PD signal.
1	Focal hypoechoic areas, where septa are less evident. Conserved thickness.	Focal heterogeneously hyperechoic areas, where septa are thicker and more evident, and muscle fibers are thinner. Conserved muscle thickness.	One or two PD signals in at least one muscle (PD vascular spots, small vessels of homogenous diameters, vessel diameters approximately not superior to fibrous intramuscular septa)
2	Diffuse and heterogeneous hypo echogenicity (rectus femoris as hypoechoic or more than vastus intermedius), septa diffusely less evident. Conserved thickness.	Diffuse and heterogeneously hyperechoic muscle, with thicker septa and thinner muscle fibers. Reduced muscle thickness.	More than 2 PD signals for each muscle (as vascular spots, small vessels of homogenous diameters, vessel diameters approximately not superior to fibrous intramuscular septa).
3	Diffuse and heterogeneous hypo echogenicity (rectus femoris as hypoechoic or more than vastus intermedius), septa diffusely less evident. Increased thickness (rectus femoris became thicker than vastus intermedius).	Diffuse and heterogeneously hyperechoic muscle, with thicker septa and thinner muscle fibers. Reduced muscle thickness.	More than 2 PD signals for each muscle with larger diameter of the vessel (at least superior to fibrous intramuscular septa), or vessels with different diameters or branched vessels.

Figure 1. Different PD findings (clockwise) in longitudinal anterior scans of the thigh: PD 3 in a patient with a recent diagnosis of anti-Mi2 DM; PD 2 in the same patient after one month of treatment with steroids and Methotrexate; PD 1 in a patient affected by anti-SAE DM, with a suspected disease flare; PD 0 in a patient affected by an advanced polymyositis diagnosed in 2000, currently not in treatment.

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POS0833

A RETROSPECTIVE COHORT STUDY IN CHINESE PATIENTS WITH ADULT POLYMYOSITIS AND DERMATOMYOSITIS: RISK OF COMORBIDITIES AND SUBCLASSIFICATION USING MACHINE LEARNING

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Background: Idiopathic inflammatory myopathy (IIM), also known as myositis, refers to a group of heterogeneous disorders including polymyositis (PM), dermatomyositis (DM), inclusion body myositis and immune-mediated necrotising myopathy. Phenotype, pathogenesis, and prognosis vary due to multi-organ involvement and comorbidities. With the clinical application of MSAs, a new classification system for myositis was explored to reduce confusion between subgroups. But it is far from showing the full picture of myositis due to high heterogeneity. Therefore, it is necessary to systematically evaluate the relevant risk factors of myositis for ILD, other rheumatic diseases, and malignancy for better clinical vigilance. And further exploring the subclassification of myositis is critical.

Objectives: To identify the risk factors in Chinese patients with adult polymyositis and dermatomyositis for their comorbidities and explore a subclassification system.

Methods: Clinical records of 397 patients with idiopathic inflammatory myopathies were retrospectively reviewed. Logistic regression was used to identify potential risk factors for interstitial lung disease (ILD), other rheumatic diseases, and malignancy after bivariate analysis. Hierarchical clustering and decisional tree were utilized to identify subgroups and explore a subclassification system.

Results: A total of 119 polymyositis and 191 dermatomyositis patients were included. Anti-PM/ScI, anti-Ro52, anti-aminoacyl-tRNA synthetase and anti-MDA5 (adjusted odds ratios (AOR)=4.779, 1.917, 5.092 and 7.714 respectively) antibodies were risks ($p<0.05$), whereas overlapping malignancy was protective (AOR=0.107; $p=0.002$) for ILD across polymyositis, dermatomyositis and the total group. In subgroup models, Raynaud's phenomenon, arthralgia and semi-quantitative anti-nuclear antibody (AOR=51.233, 4.261, 3.047 respectively) were risks for other overlapping rheumatic diseases ($p<0.05$). For overlapping malignancy, male and anti-TIF1 γ antibodies (AOR=2.533, 16.949) were risks ($p<0.05$), whereas disease duration and combination of ILD (AOR=0.954, 0.106) were protective in the total group ($p<0.05$); while anti-NXP2 antibodies were identified as risk factors (AOR=73.152; $p=0.038$) in polymyositis. Hierarchical clustering suggested a subclassification with 6 subgroups: malignancy overlapping dermatomyositis, classical dermatomyositis, polymyositis with severe muscle involvement, dermatomyositis with ILD, polymyositis with ILD, and overlapping of myositis with other rheumatic diseases according to the characteristics of grouped patients. Accuracy of the classification and regression trees model was 0.768 (95% CI 0.711 to 0.819) on training set and 0.633 (95%CI 0.499 to 0.754) on test set.

Conclusion: Accompanying ILD, other rheumatic diseases and malignancy are strongly associated with clinical manifestation and myositis-specific or myositis-associated autoantibodies among Chinese polymyositis and dermatomyositis patients. The subclassification system proposed a more precise phenotype defining toward stratified treatments.

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POS0834

LONG-TERM OUTCOME OF SSC ASSOCIATED ILD: IMPROVED SURVIVAL IN PPI TREATED PATIENTS

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Background: Gastroesophageal reflux disease (GERD) occurs frequently in patients with systemic sclerosis (SSc) and SSc-associated interstitial lung disease (SSc-ILD). PPI use has been shown to improve survival in patients with idiopathic pulmonary fibrosis, whereas to date there are no data on the use of PPI in SSc-ILD.

Objectives: This study was aimed to assess whether use of PPI is associated with progression of SSc-ILD and survival.

Methods: We retrospectively analysed 1931 patients with SSc and SSc-ILD from the German Network for Systemic Sclerosis (DNSS) database (2003 onwards). Kaplan-Meier analysis compared overall survival (OS) and progression-free survival (PFS) in patients with vs. without GERD (SSc and SSc-ILD), and PPI vs. no PPI use (SSc-ILD only). Progression was defined as a decrease in either % predicted forced vital capacity $\geq 10\%$ or single-breath diffusing capacity for carbon monoxide $\geq 15\%$, or death.

Results: GERD was not associated with decreased OS or PFS in patients with either SSc or SSc-ILD. In patients with SSc-ILD, PPI use was associated with improved OS vs. no PPI use after 1 year (98.4% [95% confidence interval: 97.6–99.3]; n=760 vs. 90.8% [87.9–93.8]; n=290) and after 5 years (91.4% [89.2–93.8]; n=357 vs. 70.9% [65.2–77.1]; n=106; p<0.0001). PPI use was also associated with improved PFS vs. no PPI use after 1 year (95.9% [94.6–97.3]; n=745 vs. 86.4% [82.9–90.1]; n=278) and after 5 years (66.8% [63.0–70.8]; n=286 vs. 45.9% [39.6–53.2]; n=69; p<0.0001).

Conclusion: GERD had no effect on survival in SSc or SSc-ILD. PPIs improved survival in patients with SSc-ILD; however, controlled, prospective trials are needed to confirm this finding.

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POS0835

DECLINE IN FORCED VITAL CAPACITY (FVC) IN SUBJECTS WITH SYSTEMIC SCLEROSIS-ASSOCIATED INTERSTITIAL LUNG DISEASE (SSc-ILD) IN THE SENSICIS TRIAL VERSUS HYPOTHETICAL REFERENCE SUBJECTS WITHOUT LUNG DISEASE

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Background: In the randomized SENSICIS trial in subjects with SSc-ILD, nintedanib reduced the rate of decline in FVC over 52 weeks (mL/year) by 44% compared to placebo. Healthy individuals have varied FVC depending on age, sex, ethnicity and height; expected values can be determined using internationally recognised reference equations.

Objectives: To provide further context to the FVC declines observed in the SENSICIS trial, we compared the decline in FVC observed in subjects with SSc-ILD in the SENSICIS trial with the decline in FVC that would be expected in hypothetical subjects without ILD matched for age, sex, ethnicity and height.

Methods: The SENSICIS trial enrolled subjects with SSc-ILD aged ≥ 18 years with first non-Raynaud symptom ≤ 7 years before screening, extent of fibrotic ILD $\geq 10\%$ on HRCT, FVC $\geq 40\%$ predicted and DLco 30–89% predicted. Baseline FVC (mL) and changes in FVC (mL) at week 52 were assessed in the nintedanib and placebo groups, with missing values at week 52 imputed using predictions from the primary analysis model (random slope and intercept model). Changes in FVC in the SENSICIS trial were compared to values in hypothetical healthy reference subjects matched to the SENSICIS subjects for age, sex, ethnicity and height. FVC values in these healthy reference subjects were derived from the equations published by the European Respiratory Society Global Lung Function Initiative in 2012, which were derived from data from over 70,000 subjects.¹

Results: In the nintedanib and placebo groups of the SENSICIS trial, respectively, mean (SD) time since onset of first non-Raynaud symptom was 3.5 (1.6) and 3.5 (1.8) years. In the nintedanib group, mean (SD) FVC at baseline was 2460 (737) mL, compared with 3403 (787) mL in the healthy reference subjects. In the placebo group, mean (SD) FVC at baseline was 2544 (817) mL compared with 3516 (887) mL in the healthy reference subjects. The difference in the change from baseline in FVC at week 52 between the nintedanib-treated subjects in the SENSICIS trial (n=287) and the healthy reference subjects was 26.6 mL ([95% CI: 1.2, 52.0]; p=0.04). The difference in the change from baseline in FVC at week 52 between the placebo-treated subjects in the SENSICIS trial (n=286) and the reference subjects was 77.5 mL ([95% CI: 51.4, 103.7]; p<0.001) (Figure 1).

Conclusion: Subjects with SSc-ILD who participated in the SENSICIS trial had marked lung function impairment at baseline compared with healthy matched reference subjects, despite a mean duration of SSc of 3.5 years. Over 52 weeks, the decline in FVC in subjects with SSc-ILD who received placebo was 4-fold greater than in healthy reference subjects. Subjects with SSc-ILD who were treated with nintedanib had a decline in FVC that was only slightly greater than the decline observed in the matched healthy subjects. These data support the clinical relevance of the reduction in the rate of FVC decline provided by nintedanib in patients with SSc-ILD.

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