Methods: We constructed two cohorts from the Renji SSc registry. In the first cohort, SSc patients receiving IGU were observed prospectively with effectiveness and safety. In the second cohort, we picked up all the DU patients with at least a 3-month follow-up to investigate the prevention of IGU on ischemic DU.

Results: 1) IGU was a plausible alternative treatment for SSc with acceptable tolerance. 91.3% (21/23) of the SSc patients were disease worsening-free during IGU treatment (median follow-up: 61 weeks). 2) We unexpectedly discovered that IGU was protective against ischemic DU. Although with a limited patient number, 72.7% (8/11) of IGU-treated patients had no new DU occurrence in a median follow-up of 39 weeks; further in the second DU cohort (Table 1), the protection of IGU was still true for new DU occurrence (adjusted RR = 0.25, 95% CI, 0.05-0.94, adjusted OR = 0.07, 95% CI, 0.01-0.49)(Figure 1).

Conclusion: Our study indicates IGU as a possible alternative treatment for SSc. Beyond expectation, this study for the first time describes IGU as preventive against ischemic DU occurrence and merits further investigation.

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Figure 1. Forest plot displaying the association of the outcome (occurrence of ischemic DUs) with IGU treatment.

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POS1268 TYPE 2 (TH2) CYTOKINES AND SCLEERODERMA INTERSTITIAL LUNG DISEASE (SSC-ILD)

Keywords: Systemic sclerosis, Cytokines and chemokines, Lungs

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Background: Systemic sclerosis (SSc) is an autoimmune disease characterized by immune system dysregulation, endothelial dysfunction and fibrosis of skin and internal organs. Interstitial lung disease (ILD) is common in SSc and about 80% of patients will develop ILD during the disease course [1]. Although most patients have a stable or slowly progressive disease, showing a slow decline in lung function or a minimal increase in the extent of pulmonary fibrosis by high resolution computed tomography (HRCT), 25-30% of them will ultimately progress to respiratory failure or death [2]. Of the SSc-related deaths, 35% were attributed to ILD [3]. The underlying pathophysiological mechanisms of SSc-ILD is still not completely understood [2]. The pathogenesis of SSc-ILD is initially characterized by an injury to alveolar epithelial and vascular endothelial cells which promotes the recruitment of inflammatory cells and the production of profibrotic mediators, in the attempt to repair the damage [4,5]. In this early inflammatory phase, profibrotic type 2 (Th2) cytokines, such as IL-4, IL-5, IL-13, play a key role [6,7].

Objectives: To identify trajectories, clinical correlates and associations of functional disability in SSc.

Methods: Australian Scleroderma Cohort Study participants meeting ACR/EULAR criteria for SSc recruited within 5 years of SSc onset were included. Participants needed to have ≥2 HAQ DI scores available within 10 years of SSc onset. Group based trajectory modelling (GBTM) was used to identify the number and shape of trajectories involving repeated measures. Survival analyses were used to identify trajectories, clinical correlates and associations of functional disability in SSc.

Results: Two HAQ DI trajectory groups: low/stable disability (n=221, 52%), and high/increasing disability (n=205, 48%; Figure 1). Participants with high/increasing disability were older (p=0.01) and more likely to have dcSSc (p<0.01), PAH (p<0.01) and multiple cardiovascular risk factors (p<0.01; Table 1). High/increasing disability was associated with markers of poor hand function such as digital ulcers and synovitis (p<0.01), upper and lower GI involvement (both p<0.01) and proximal muscle weakness and atrophy (both p<0.01). Use of immunosuppression (p<0.001), including prednisolone (p<0.001), was more frequent in those with high/increasing disability. Those with high/increasing disability had worse survival after adjusting for age, sex, dcSSc, PAH and ischaemic heart disease (HR 1.9, 95% CI 1.0-3.8, p=0.05), as did those with increasing baseline HAQ DI scores (HR 1.8, 95%CI 1.2-2.7, p<0.01).

Conclusion: Two trajectories of functional disability in SSc were identified. Those with poorer function had a distinct clinical phenotype and survival compared to those with less functional disability. Further work is required to identify if amelioration of disease-specific features associated with higher HAQ DI scores results in a commensurate improvement in physical function.

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