

EUSTAR at EULAR 2026



EULAR 2026 Highlights Brochure London, UK | 3-6 June 2026



By EUSTAR's Young Investigators group

EDITORIAL LETTER

From Organ-Based Management to Precision Medicine in Systemic Sclerosis

The scientific programme of EULAR 2026 clearly demonstrated that systemic sclerosis (SSc) is entering a new era. Rather than focusing on individual organ manifestations, many presentations converged on a broader concept of disease heterogeneity, emphasizing that molecular mechanisms, clinical phenotypes, and longitudinal disease trajectories should increasingly guide both patient stratification and therapeutic decision-making. Throughout this meeting, precision medicine emerged not as a future aspiration, but as an evolving clinical reality.

Several sessions highlighted the transition from traditional clinical classification toward biomarker- and mechanism-based disease stratification. Novel molecular signatures, single-cell and spatial transcriptomic analyses, advanced imaging techniques, and emerging serum biomarkers are beginning to redefine how we identify patients at risk for disease progression and organ involvement. Equally important, these advances are creating opportunities to select patients more appropriately for targeted therapies and future clinical trials.

A second recurring theme was the importance of detecting organ involvement before irreversible damage occurs. Across pulmonary, cardiac, vascular, gastrointestinal and musculoskeletal manifestations, speakers consistently emphasized the value of longitudinal surveillance, multimodal imaging, and risk-adapted screening strategies. Cardiac magnetic resonance imaging, lung ultrasound, capillaroscopy, high-frequency skin imaging, and refined risk prediction tools all illustrated how earlier recognition of subclinical disease may improve clinical outcomes.

The meeting also reflected the rapidly evolving therapeutic landscape in SSc. While current management remains largely phenotype- and organ-driven, several presentations showcased therapies targeting shared molecular pathways across multiple organs. Emerging approaches, including CAR-T cell therapy, PDE4B inhibition, modulation of interferon and complement signalling, and novel pulmonary vascular therapies such as sotatercept, illustrated the growing shift from symptom control toward disease modification and tissue regeneration.

Finally, several presentations highlighted the growing importance of harmonized outcome measures, standardized imaging protocols, and international collaborative registries such as EUSTAR. These initiatives are strengthening both clinical research and everyday practice by enabling more consistent patient assessment, facilitating multicentre studies, and accelerating the translation of scientific discoveries into patient care.

The EUSTAR Young Investigators Group prepared this Highlights Brochure to summarize the scientific advances presented during EULAR 2026 that we believe are most likely to influence future research and clinical practice. We hope this overview provides readers with a concise synthesis of the meeting's key messages while reflecting the remarkable progress currently being made toward more personalized, mechanism-based care for patients with systemic sclerosis.

Jelena Colic, MD, PhD
Editor

EUSTAR at EULAR 2026



CLINICAL MANIFESTATIONS AND DISEASE COURSE IN SYSTEMIC SCLEROSIS

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By EUSTAR's Young Investigators group



CLINICAL MANIFESTATIONS AND DISEASE COURSE IN SYSTEMIC SCLEROSIS

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SESSION 2 – CLINICAL ABSTRACT SESSIONS | Thursday, 4 June 2026

OP179 – Clinical Value of Modified EUSTAR Disease Activity Index to Stratify for Clinical Outcome: A Longitudinal Observational Analysis of the EUSTAR Database | Tania Santiago (Portugal)

This study highlights the clinical value of the 2023 modified EUSTAR Disease Activity Index (mEDAI) as a feasible tool for predicting future disease burden in systemic sclerosis (SSc). In a large EUSTAR cohort, high baseline mEDAI identified patients with greater multisystem involvement at one year and earlier development of key organ complications, supporting its use beyond a cross-sectional assessment of disease activity. Importantly, the presentation also emphasized that SSc progression is not uniform over time: vascular manifestations may continue to accumulate, ILD and other fibrotic outcomes remain clinically relevant across disease stages, and inflammatory activity may show late reactivation. Together, these findings support dynamic, phenotype-based follow-up strategies and suggest that mEDAI may improve patient selection and enrichment in clinical trials.

Key Highlights

- High baseline modified EUSTAR Disease Activity Index may predict future organ involvement.
- SSc progression follows distinct vascular, fibrotic and inflammatory trajectories.
- mEDAI may support follow-up planning and clinical trial enrichment.

OP0212 – Validation of the ILD-RISC Score in Special Populations from the EUSTAR Registry: Tailored Cut-Off Values for Non-Caucasian Ethnicities, Pulmonary Hypertension and Very Early Pre-SSc | Maria Iacovantuono (Italy)

Analysis from EUSTAR database supports a more individualized approach to ILD screening in SSc. Although ILD-RISC is a practical tool to identify patients who may benefit from HRCT, this study shows that one universal threshold may not perform equally across distinct populations. By validating the score in non-Caucasian patients, patients with pulmonary hypertension, and very early pre-SSc, the work highlights the value of adapting risk thresholds to patient phenotype. Tailored cut-offs may preserve adequate sensitivity while improving specificity and reducing unnecessary HRCT scanning during follow-up. These findings refine ILD surveillance by balancing early detection, imaging burden, and patient-specific risk.

Key Highlights

- ILD-RISC may support individualized ILD screening in SSc.
- Tailored cut-offs improve subgroup-specific risk stratification.
- Optimized thresholds may reduce unnecessary HRCT use.

OP0215 – Primary Cardiac Involvement in Early Systemic Sclerosis: Baseline Profile of the Patients in the SOLAR Registry | *Shirkhan Amikishiyev* (Turkey)

Primary cardiac involvement remains one of the most feared complications of SSc, yet it is frequently overlooked during the early phases of disease. This large registry analysis highlights that clinically recognized cardiac involvement is already present in a subset of patients within the first three years after diagnosis and tends to cluster with markers of severe multisystem disease, including diffuse cutaneous involvement, pulmonary arterial hypertension, myositis, overlap features, and hypertension. Importantly, new cases continued to emerge during follow-up, underscoring the dynamic nature of cardiac disease in SSc. Rather than supporting a one-time screening approach, these findings reinforce the need for longitudinal cardiovascular surveillance, particularly in patients with high-risk clinical phenotypes. The study also raises the possibility that current real-world practice may underestimate the burden of subclinical cardiac involvement.

Key Highlights

- Cardiac involvement appears early in the SSc disease course.
- High-risk phenotypes show clustering of cardiac manifestations.

Serial cardiac assessment may be more informative than a single baseline evaluation. OP0216 – The Involvement of the Temporomandibular Joint in Patients with Systemic Sclerosis: A Clinical and Ultrasound Study | *Alessia Laneri* (Italy)

This study highlights an often-neglected manifestation of SSc: temporomandibular joint (TMJ) involvement. Ultrasound revealed a striking burden of structural and inflammatory abnormalities, many of which were clinically silent and would have remained undetected through routine examination alone. The association between active synovitis, microstomia, and impaired mouth opening suggests that TMJ inflammation may contribute directly to functional disability. Equally intriguing is the potential link between TMJ abnormalities and dysphagia in patients without documented oesophageal disease, raising the possibility that orofacial dysfunction contributes to swallowing difficulties more than previously appreciated. These findings position ultrasound as a practical tool for identifying early TMJ pathology and preventing progressive orofacial impairment.

Key Highlights

- TMJ abnormalities are frequently present despite absent symptoms.
- Ultrasound detects disease beyond routine clinical examination.
- Orofacial dysfunction may contribute to dysphagia in selected patients.

OP0217 – Features of Pulmonary Veno-Occlusive Disease Complicate the Course of Precapillary Pulmonary Hypertension in Systemic Sclerosis: A EUSTAR Cohort Study | Nicola Farina (Italy)

This EUSTAR analysis identifies pulmonary veno-occlusive disease (PVOD) features as markers of a distinct and particularly vulnerable subgroup of patients with SSc-associated precapillary pulmonary hypertension. Although uncommon, PVOD characteristics were associated with more severe haemodynamic impairment, worse functional status, and a substantially higher risk of pulmonary hypertension progression. Importantly, readily available clinical variables, including anti-centromere antibody positivity, elevated uric acid levels, and higher pulmonary artery pressures, helped identify patients more likely to have PVOD features. The study reinforces the value of careful HRCT assessment in patients undergoing evaluation for pulmonary hypertension and supports a more individualized approach to risk stratification. Recognition of PVOD features may become increasingly relevant as treatment decisions in SSc-associated pulmonary vascular disease continue to evolve.

Key Highlights

- PVOD features define a high-risk pulmonary vascular phenotype.
- HRCT findings provide clinically relevant prognostic information.
- ACA positivity and elevated uric acid may raise suspicion for PVOD.

Overall Take-Home Messages

- Precision risk stratification is becoming central to systemic sclerosis management.
- Longitudinal surveillance outperforms single baseline assessment for detecting progressive organ involvement.
- Advanced imaging enables earlier detection of subclinical disease and supports individualized clinical decision-making.
- Clinical phenotyping identifies patients at highest risk for severe cardiopulmonary and musculoskeletal complications.

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NEW APPROACHES TO SCLERODERMA DIAGNOSIS AND STRATIFICATION

Chosen and written by
Merve Eriksson

By EUSTAR's Young Investigators group

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SESSION 3 – SCLERODERMA DISEASE STRATIFICATION WITH MECHANISTIC INSIGHTS | Saturday, 6 June 2026

OC 1 – Scleroderma Disease Stratification with Mechanistic Insights | Shervin Assassi (USA)

Disease stratification in systemic sclerosis (SSc) is moving beyond traditional clinical subsets toward a dynamic, mechanism-based approach. The prognostic significance of RNA polymerase III antibody positivity varies according to disease stage, with patients demonstrating either progression or regression of skin fibrosis depending on disease duration. Observational data suggest that an early inflammatory phenotype may precede subsequent improvement in skin involvement, supporting early immunomodulatory treatment in selected patients to prevent irreversible complications such as joint contractures.

The presentation also emphasized the importance of recognising early non-Raynaud manifestations, including puffy fingers and diffuse hand swelling, which may be misclassified as seronegative rheumatoid arthritis. Anti-topoisomerase I positivity remains strongly associated with interstitial lung disease (ILD), even in patients with limited cutaneous disease, while biomarkers such as KL-6 may further refine ILD risk prediction and help identify patients at increased risk of disease progression.

Key Highlights

- Disease duration modifies the prognostic significance of RNA polymerase III antibody positivity.
- Early inflammatory disease may represent a therapeutic window for immunomodulatory treatment.
- KL-6 shows promise as a prognostic biomarker for SSc-ILD progression.

OC 2 – Novel Approach to Assessing Scleroderma Skin Involvement Using SFDI | Andreea Bujor (USA)

Spatial frequency domain imaging (SFDI) was presented as a promising non-invasive approach for quantifying skin involvement in systemic sclerosis (SSc). By projecting patterned light onto the skin and measuring tissue reflectance, SFDI generates quantitative maps of tissue optical properties that may detect pathological changes before they become clinically apparent. SFDI measurements correlated with the modified Rodnan skin score (mRSS) and demonstrated a dose-response relationship with disease severity. Importantly, the technique was able to detect subclinical skin involvement, suggesting potential value for earlier diagnosis and more sensitive longitudinal monitoring. Its convergent validity with high-frequency ultrasound and histological markers of fibrosis further supports its potential as an objective biomarker of skin involvement. With validation in larger cohorts, SFDI could become an important outcome measure in clinical trials, where objective, reproducible, and sensitive assessment of skin fibrosis remains an unmet need.

Key Highlights

- SFDI offers quantitative, non-invasive assessment of skin involvement.
- The method may detect subclinical disease not captured by clinical scoring.
- SFDI could become a sensitive outcome measure for SSc trials.

OC 4 – Clinical Implications: Diagnostic Work-Up and Application of EULAR Recommendations | Christopher Denton (UK)

This session emphasized the importance of comprehensive baseline assessment and proactive monitoring in systemic sclerosis (SSc). Earlier and more systematic screening has enabled improved detection of interstitial lung disease (ILD) and pulmonary arterial hypertension (PAH), contributing to better survival. Autoantibody profiling provides important prognostic information and helps identify patients at increased risk of specific organ complications. Organ involvement accumulates over time, with ILD remaining a major contributor to morbidity, particularly in diffuse cutaneous SSc. Importantly, limited cutaneous SSc, despite being the most common disease subset, carries a substantial burden of gastrointestinal, vascular, and pulmonary complications. Treatment decisions for skin and lung involvement should integrate disease duration, cutaneous subset, and autoantibody profile.

Key Highlights

- Earlier detection of ILD and PAH is associated with improved survival.
- Autoantibody profiling helps predict organ-specific complications.
- Limited cutaneous SSc carries a substantial burden of organ complications.

Overall Take Home Messages

- SSc diagnosis and stratification are evolving toward a biomarker- and mechanism-based approach.
- Early risk stratification and proactive screening enable timely, personalized management.
- Emerging biomarkers, including KL-6, together with novel imaging techniques, may improve early detection and disease monitoring.
- Objective imaging tools such as SFDI may strengthen future skin fibrosis clinical trials.

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NEW TARGETS IN SYSTEMIC SCLEROSIS

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Claudia Iannone

SESSION 4 – BASIC AND CLINICAL ABSTRACT SESSIONS | Friday, 5 June 2026

OP0305 – Multiorgan Single-Cell Profiling Identifies Shared Interferon and Alternative Complement Activation Across Tissues in Systemic Sclerosis | *Cristian Iperi* (Switzerland)

Although systemic sclerosis (SSc) is a multi-organ disease, current therapies remain organ-specific, and a shared molecular target across organs could enable disease-wide treatment. Using multiorgan single-cell profiling, this study showed that type I and II interferon (IFN) signalling and alternative complement activation form a shared signature across SSc-affected tissues: lung, skin, PBMCs and synovium, rather than being organ-restricted. The pathways converged on a common, metabolically active fibroblast state, pointing to a conserved pathology across tissues. These findings position IFN- and alternative complement-targeted therapies as candidates for systemic, rather than organ-by-organ, intervention, and provide a rationale for rethinking current SSc treatment strategies.

Key Highlights

- Type I/II interferon and alternative complement form a shared, systemic signature across SSc organs.
- The IFN and alternative complement pathways converge on a common metabolically active fibroblast state across tissues. Findings support systemic, pathway-targeted therapy over organ-by-organ SSc treatment.

OP0307 – Human Lung Organoids as a Model of SSc-ILD | *R. Neelagar* (Germany)

This study established and validated SSc patient iPSC-derived human lung organoids (HLOs) as a preclinical model of SSc-associated interstitial lung disease (SSc-ILD). The organoids reproduced the relevant distal lung compartments, containing alveolar (AEC1/AEC2) and distal bronchial epithelial cells alongside PDGFR α + fibroblasts. Exposure to profibrotic stimuli — TGF β or SSc patient serum — drove

fibroblast-to-myofibroblast differentiation, with increased α SMA and excessive extracellular matrix deposition (COL1A1, FN1, collagen I), accompanied by transcriptomic programmes of epithelial injury and proinflammatory signalling. The antifibrotic agent nintedanib attenuated these responses. Single-cell analysis showed SSc-HLOs recapitulated fibroblast subpopulations described in SSc-ILD, including a proliferative myofibroblast subset, with reduced, injury-primed AEC2 precursors. Together these findings support SSc-HLOs as a promising, patient-derived platform for mechanistic study and drug testing in SSc-ILD.

Key Highlights

- SSc-iPSC-derived lung organoids reproduce the epithelial–mesenchymal compartments of SSc-ILD.
- TGF β or SSc serum drives myofibroblast differentiation and excess ECM deposition.
- Nintedanib attenuates profibrotic responses, supporting HLOs for preclinical drug testing.

OP0309 – Single-Cell Profiling Identifies PDE4B as a Disease-Relevant Target in Systemic Sclerosis Across Tissues and Cell Subsets | Astrid Hofman (Switzerland)

PDE4B inhibition has recently proven effective in idiopathic pulmonary fibrosis and progressive pulmonary fibrosis through endothelial-stabilizing, immunomodulating and anti-fibrotic effects, making its tissue distribution in SSc clinically relevant. This work provides the first tissue-spanning, single-cell overview of PDE4B in SSc. PDE4B dysregulation localized primarily to the immune compartment — T cells, B cells and monocytes — across lung, skin and PBMCs. Notably, the activated FAP⁺ fibroblasts that drive fibrosis almost uniformly co-expressed PDE4B in SSc skin. By recasting PDE4B as a shared immune target that also captures the activated fibroblasts central to fibrosis, the study strengthens the rationale for trialling PDE4B inhibitors in SSc.

Key Highlights

- PDE4B is upregulated across immune cells in affected organs and activated fibroblasts in SSc skin.
- It marks both immune drivers and the FAP⁺ fibroblasts central to fibrosis.
- Findings strengthen the rationale for trialling PDE4B inhibitors in SSc.

OP0309 – Proteome-Wide Autoantibody Landscape and Pathogenic Role of Anti-CCR8 Autoantibodies in Systemic Sclerosis: Post-Hoc Analysis of a B-cell Depletion Trial | Kazuki Matsuda (Japan)

Classical SSc autoantibodies aid diagnosis but are largely unaffected by rituximab and do not predict treatment response. Profiling antibodies against more than 13,000 antigens, this post-hoc analysis of a B-cell depletion trial identified a dynamic, rituximab-sensitive autoantibody landscape. Unlike the static classical autoantibodies, anti-CCR8 shifted with treatment; functionally, it blocked CCL1-driven regulatory T-cell recruitment and worsened dermal fibrosis in vivo. This offers a mechanism for the local regulatory T-cell deficiency seen in fibrotic lesions and indicates that some autoantibodies actively contribute to impaired immune regulation rather than acting as passive markers. Anti-CCR8 and companion signatures could predict rituximab response and stratify patients, while the CCR8–Treg axis emerges as a precision-immunotherapy target.

Key Highlights

- Proteome-wide profiling reveals a dynamic, rituximab-sensitive autoantibody landscape in SSc.
- Anti-CCR8 blocks CCL1-driven Treg recruitment and aggravates dermal fibrosis in vivo.

- Autoantibody signatures may predict rituximab response and guide patient stratification.

OP0310 – CD19-CAR T-Cell Therapy Induces Regenerative Skin Remodelling in Diffuse Cutaneous Systemic Sclerosis | A. Rius Rigau (Germany)

This proof-of-concept study examined skin tissue changes before and after CD19-CAR T-cell therapy in patients with refractory diffuse cutaneous SSc, using serial forearm biopsies analysed by histology, bulk RNA sequencing, spatial transcriptomics and imaging mass cytometry. Deep, broad B-cell depletion produced sustained structural regeneration of fibrotic skin: increased dermal papillae, recovery of rete ridges and reduced collagen fibre alignment, changes not seen with standard-of-care. Tissue improvements paralleled durable reductions in mRSS. Molecular profiling showed downregulation of B-cell signatures and enrichment of vascular development, angiogenesis and epidermal differentiation pathways, with prominent vascular regeneration, improved Raynaud's phenomenon and increased capillary density driven by VEGF- and IGF-associated signalling. These findings provide first evidence that deep B-cell depletion may reverse established structural skin pathology, extending beyond immunomodulation to genuine tissue regeneration.

Key Highlights

- CD19-CAR T-cell therapy induced sustained regenerative remodelling of fibrotic SSc skin.
- Structural recovery (dermal papillae, rete ridges) paralleled durable mRSS reductions.
- Vascular regeneration and improved Raynaud's phenomenon were associated with VEGF/IGF-driven angiogenic signalling.

Overall Take-Home Messages

- Resolving the molecular and immunological drivers of SSc across tissues is opening the door to systemic, mechanism-based therapy.
- Shared molecular pathways may enable mechanism-based therapies across multiple SSc organs Patient-derived lung organoids and deep B-cell-depleting CAR T-cell therapy point toward reversible, regenerative models of SSc.
- Molecular and serological signatures may improve patient stratification and accelerate precision clinical trials in SSc.

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TAKING CARE OF SYSTEMIC SCLEROSIS

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SESSION 6 – CLINICAL POSTER TOURS | Friday, 5 June 2026

PO 321 – Points to Consider for Reporting Outcome Measures in Systemic Sclerosis Interventional Studies on Digital Ulcers: An Initiative from the World Scleroderma Foundation Digital Ulcer Ad Hoc Committee | Corrado Campochiaro (Italy)

Digital ulcers (DU) are a major source of pain, disability, and reduced quality of life in systemic sclerosis (SSc), yet clinical research in this area has long been hampered by inconsistent definitions, outcome measures, and reporting standards. This international World Scleroderma Foundation initiative represents an important step toward harmonizing the design and reporting of interventional studies. The key advance is the establishment of a consensus framework that standardizes how DU are defined, assessed, and reported across trials. Adoption of these standards aims to improve comparability between studies, strengthen evidence synthesis, and accelerate the development of more effective, patient-centred treatment strategies, ultimately facilitating the translation of research findings into clinical practice.

Key Highlights

- **Consensus recommendations standardize the definition, assessment, and reporting of SSc digital ulcers.**
- **Harmonized outcome measures will improve comparability across DU clinical trials.**
- **Standardized reporting may strengthen evidence generation and support the development of better DU therapies**

PO 322 – Rapidly Progressing Versus Stable Phenotype in Patients with Systemic Sclerosis-Associated Pulmonary Arterial Hypertension | Katya Dolnikov (Israel)

In this multicentre cohort study, Dolnikov et al. investigated disease trajectories among patients with systemic sclerosis-associated pulmonary arterial hypertension (SSc-PAH) classified as low or intermediate-low risk at diagnosis. Among 114 patients, 86 were classified as low or intermediate-low

risk; of these, 43% progressed to higher-risk status or died within 5 years. Progressors showed a distinct baseline profile, with shorter disease duration at PAH diagnosis, higher mPAP, shorter 6MWD and more frequent pericardial effusion. Reduced baseline DLCO was the only independent predictor of progression, suggesting that impaired gas transfer may identify lower-risk patients at increased risk of subsequent deterioration.

Key Highlights

- Lower-risk SSc-PAH is not always stable and may follow a progressive trajectory.
- Baseline DLCO may help identify lower-risk patients at risk of later deterioration.
- Close monitoring remains important to enable timely intervention in lower-risk SSc-PAH.

PO 324 – Systemic Sclerosis Post-Diagnosis: Real-World Data on Disease Evolution, Treatment Patterns, and Healthcare Resource Utilisation in the US | *Silvia Bellando Randone* (Italy)

This real-world study provides an overview of the healthcare utilization of patients newly diagnosed with SSc in a cohort from the United States. The findings highlight a substantial burden of multi-organ disease that emerges early and persists over time, reinforcing the need for continuous multidisciplinary care. Notably, diagnosis did not appear to translate into sustained intensification of specialist care or use of disease-modifying therapies. Instead, management remained largely focused on symptom control, while engagement with specialist services and diagnostic testing declined over time. This study identifies key unmet needs and provides a strong rationale for more coordinated and specialist-driven models of care in SSc.

Key Highlights

- Persistent multi-organ burden with early onset and long-term continuity in SSc.
- Significant gap between disease complexity and specialist follow-up.
- Unmet needs in monitoring, treatment, and multidisciplinary care coordination.

PO 329 – Immunosuppressants Prevent Digital Ulcers in Systemic Sclerosis: Evidence from a EUSTAR Effectiveness Study | *Nicola Farina* (Italy)

In this EUSTAR study, Farina et al. explored whether immunosuppressive therapy is associated with a reduced risk of new or recurrent DU in SSc. Among 7154 patients without current DU at a reference visit, 16.6% developed new or recurrent DU during a median 2.5 years of follow-up. Previous DU remained the main risk factor, with older age, higher mRSS, low BMI, male sex, and anti-Scl70 or anticentromere positivity also associated with risk. Exposure to both csDMARDs and bDMARDs was associated with fewer future DU, independent of vasoactive medication and previous DU history, with specific signals for MMF, MTX and CYC.

Key Highlights

- Immunosuppressive therapy was associated with a reduced risk of new or recurrent digital ulcers beyond vasoactive therapy.
- MTX, MMF and CYC showed significant protective associations against future digital ulcers.
- Real-world data support prospective studies of immunomodulatory strategies for digital ulcer prevention.

Overall Take-Home Messages

- Standardizing digital ulcer outcomes in SSc improves study comparability and accelerates the development of effective therapies.

- Lower-risk SSc-PAH is not always stable and requires close monitoring for timely intervention.
- Persistent multi-organ burden in SSc reflects major gaps in long-term specialist care and management.
- Real-world EUSTAR data link immunosuppression with lower future DU risk beyond vasoactive therapy.

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PRIMARY HEART INVOLVEMENT IN SYSTEMIC SCLEROSIS

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PRIMARY HEART INVOLVEMENT IN SYSTEMIC SCLEROSIS

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Margarida Lucas Rocha

Session 7 – PRIMARY HEART INVOLVEMENT IN SYSTEMIC SCLEROSIS | Saturday, 6 June 2026

Pathogenesis of Primary Heart Involvement in Systemic Sclerosis | *Andrea-Hermina Györfi* (Germany)

Primary heart involvement (pHI) remains one of the most severe yet under-recognised manifestations of systemic sclerosis (SSc). This session highlighted how myocardial inflammation, microvasculopathy and fibrosis act as interconnected drivers of cardiac damage and poor outcomes. Increasing degrees of myocardial inflammation and fibrosis were consistently associated with higher rates of major cardiac events, while SSc myocarditis emerged as a histopathologically distinct entity from other autoimmune myocarditides, characterised by greater myocardial fibrosis, higher rates of heart failure and increased cardiac mortality.

Novel translational studies provided important insights into the underlying disease mechanisms. Spatial transcriptomics analyses identified distinct cardiac fibroblast populations occupying specific myocardial niches with specialised functional roles, suggesting that different fibroblast subsets contribute to distinct patterns of fibrosis and clinical phenotypes. A unique stromal-myeloid axis involving C1q/CD163-positive macrophages appears to play a central role in disease progression, reinforcing fibroblast heterogeneity as a promising avenue for targeted therapeutic strategies.

Key Highlights

- Myocardial inflammation and fibrosis predict adverse cardiac outcomes.
- SSc myocarditis differs from other autoimmune myocarditis entities.

- Distinct fibroblast subsets may drive specific fibrotic pathways and clinical phenotypes.

All Faces of Primary Heart Involvement in Systemic Sclerosis | Carina Mihai (Switzerland)

This session reinforced the concept that myocardial inflammation, fibrosis and microvasculopathy are interconnected drivers of cardiac damage and adverse outcomes, with increasing degrees of inflammation and fibrosis associated with higher rates of major cardiac events and mortality. Cardiac magnetic resonance (CMR) was highlighted as the cornerstone modality for detecting subclinical cardiac involvement. Beyond conventional late gadolinium enhancement (LGE), which identifies focal non-ischaemic myocardial fibrosis, quantitative techniques including T1 and T2 mapping and extracellular volume (ECV) assessment enable detection of diffuse fibrosis, active inflammation and microvascular dysfunction.

The updated Lake Louise Criteria further support multiparametric CMR for diagnosing myocarditis. Importantly, the World Scleroderma Foundation/Heart Failure Association definition of SSc-pHI provides a framework for distinguishing SSc-related cardiac disease from alternative cardiac and non-cardiac causes. EUSTAR data identified age, male sex, arthritis, muscle involvement, tendon friction rubs, and anti-topoisomerase I antibodies as major risk factors, while elevated troponin, CRP, arrhythmias, and interstitial lung disease may represent clinical red flags warranting comprehensive cardiac assessment.

Key Highlights

- CMR enables early detection of inflammation, fibrosis and microvasculopathy.
- Clinical and serological risk factors support targeted cardiac screening.
- Cardiac involvement remains a major determinant of survival in SSc.

Overall Take Home Messages

- Cardiac involvement in SSc is heterogeneous, frequent and often underdiagnosed.
- Early identification of high-risk patients is crucial to improve outcomes.
- CMR plays a central role in detecting subclinical disease.
- Cardiac involvement remains a major determinant of survival in SSc.

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DIFFICULT TO TREAT GASTROINTESTINAL MANIFESTATIONS IN SYSTEMIC SCLEROSIS

Chosen and written by
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DIFFICULT TO TREAT GASTROINTESTINAL MANIFESTATIONS IN SYSTEMIC SCLEROSIS

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Janina Auth

SESSION 8 – DIFFICULT TO TREAT GASTROINTESTINAL MANIFESTATIONS IN SYSTEMIC SCLEROSIS | Saturday, 6 June 2026

GASTRIC ANTRAL VASCULAR ECTASIA IN SYSTEMIC SCLEROSIS

Case Presentation | Artur Ejsmont (Poland)

A 51-year-old woman with diffuse cutaneous systemic sclerosis, diagnosed three months earlier, and a history of anemia and erosive erythematous gastroscopy was referred for further assessment. On admission, severe anemia and iron deficiency were detected; cardiac and pulmonary involvement were excluded. Initial treatment with mycophenolate mofetil (MMF), followed by cyclophosphamide, failed to control disease progression, with worsening skin involvement. Tocilizumab was therefore initiated seven months after first presentation. Haemoglobin continued to decline, prompting endoscopy: colonoscopy was unremarkable, whereas gastroscopy revealed severe gastric antral vascular ectasia (GAVE) with dilated, fragile vessels. Argon plasma coagulation (APC) was performed to reduce bleeding risk, alongside intravenous immunoglobulins (total 110 g), methylprednisolone (250 mg) and transfusion of irradiated leukocyte-depleted red blood cells. Repeat gastroscopy and APC followed after seven days, after which haemoglobin increased and stabilized. Maintenance comprised methylprednisolone 4 mg daily, proton pump inhibitors and iron supplementation; methotrexate was added one month later and tocilizumab switched to subcutaneous administration. Eighteen months

later, skin involvement had improved, haemoglobin and iron levels remained normal, and follow-up gastroscopy showed complete resolution of GAVE.

Case Discussion | Otylia Kowal-Bielecka (Poland)

The reported prevalence of GAVE in systemic sclerosis (SSc) ranges from 0–22%, with higher rates in cohorts undergoing gastroscopy independent of symptoms, suggesting GAVE is frequently subclinical. The most common manifestations are anemia and iron deficiency; differential diagnoses include chronic inflammation, iron deficiency, acute bleeding, malnutrition, and scleroderma renal crisis. Assessment of iron metabolism can aid evaluation. Endoscopy remains the gold standard, revealing characteristic longitudinal folds with dilated vessels radiating from the pylorus toward the antrum; histopathological confirmation is not required, and negative biopsy does not exclude the diagnosis. Due to limited evidence, the 2023 EULAR recommendations provide no specific guidance for GAVE. Management of symptomatic GAVE typically includes endoscopic interventions such as argon plasma coagulation, radiofrequency ablation or band ligation, with embolization, radiotherapy and surgery reserved for refractory cases. Supportive measures include iron supplementation, transfusions and proton pump inhibitors. Evidence for immunosuppression remains limited, although prior immunosuppression has been associated with lower odds of GAVE. Studies also suggest increased rates of scleroderma renal crisis and malignancy among SSc patients with GAVE.

Key Highlights

- GAVE is an important, but often subclinical, vascular GI manifestation in SSc patients.
- Anemia and iron deficiency should raise awareness of GAVE in SSc patients.
- Endoscopic evaluation seems reasonable in SSc patients with unexplained anemia and risk factors.

SMALL INTESTINAL BACTERIAL OVERGROWTH IN SYSTEMIC SCLEROSIS

Case Presentation | Marco Minerba (UK)

A 62-year-old man with limited systemic sclerosis (ACA+ and Ro52+), characterised by Raynaud's phenomenon with digital ulcers, telangiectasias, calcinosis and gastroesophageal reflux disease, and receiving esomeprazole, famotidine and sildenafil, was referred to a tertiary centre for progressive dysphagia, worsening diarrhea and bloating, new-onset fecal incontinence and unintentional weight loss. Investigations demonstrated extensive gastrointestinal involvement, including esophageal dysmotility, hiatal hernia with Barrett's esophagus and slow intestinal transit. Anorectal assessment revealed reduced resting pressure on manometry and thinning of both internal and external anal sphincters on endoanal ultrasound. Small intestinal bacterial overgrowth (SIBO) was presumed to contribute to diarrhea and fecal incontinence, and the patient was treated with cyclical rifaximin (550 mg twice daily for one week per month) and probiotics, with limited benefit. He was being considered for prokinetic therapy when he experienced a major SSc flare with marked skin progression (modified Rodnan Skin Score 11 to 28), requiring initiation of mycophenolate mofetil. Initial gastrointestinal adverse effects were observed but subsequently resolved.

Case Discussion | Francesco Del Galdo (UK)

SIBO is among the most frequent expressions of gastrointestinal dysmotility in SSc, with a pooled prevalence of approximately 34–35% (roughly ten-fold that of the general population). Disease-related

risk factors include longer disease duration, older age and weight loss. Diagnosis remains a key limitation: hydrogen and methane breath testing is the most validated and feasible modality in SSc, but its low sensitivity and specificity, together with method-dependent prevalence estimates, render the evidence base of low reliability. Fecal calprotectin was discussed as a promising but not-yet-validated non-invasive adjunct. Treatment evidence is limited and largely uncontrolled: cyclical rifaximin is the mainstay and outperforms rotating antibiotics for eradication (77.8% vs 44.8%), while adding a probiotic improves eradication and symptom control. The discussion reframed SIBO not as an isolated infection but as one component of a complex, heterogeneous GI dysmotility syndrome: multimodal assessment with MR enterography and bowel US supported the hypothesis that segmental volume loss of the small intestine may drive SIBO. This imaging phenotyping may explain the limited durability of antimicrobial therapy and could identify candidates for motility-directed or anatomically targeted interventions.

Key Highlights

- SIBO is associated with gastrointestinal and extra-intestinal manifestations in systemic sclerosis.
- Longer disease duration, weight loss and higher UCLA GIT scores are associated with SIBO.
- Rifaximin and probiotics may improve SIBO.

Overall Take Home Messages

- GI involvement in SSc is challenging, as treatments may worsen or trigger gastrointestinal symptoms.
- Treatment recommendations are limited; further controlled trials with GI-related outcomes are needed.
- Multidisciplinary management is important to adequately treat GI symptoms in SSc patients.

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LUNG SCAN IN ILD

Chosen and written by
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Lucy Thornton

By EUSTAR's Young Investigators group

LUNG SCAN IN ILD

Chosen and written by:



Seda Colak



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SESSION 9 – HANDS-ON WORKSHOP | Thursday, 4 June 2026

Lung Scan in ILD | *Andrea Delle Sedie* (Italy)

This hands-on workshop highlighted lung ultrasound (LUS) as a valuable point-of-care tool in rheumatology, particularly for connective tissue disease-associated interstitial lung disease. Its main value lies in providing a rapid, clinic-based assessment that can be performed within five minutes using simplified protocols. The session focused on two key abnormal findings: B-lines and pleural-line abnormalities. According to the OMERACT definition, B-lines are vertical hyperechoic reverberation artefacts arising from the pleural line, extending to the bottom of the screen without fading and moving synchronously with lung sliding. Pleural abnormalities include irregularity and thickening, with irregularity defined as loss of pleural-line regularity, which may be focal, diffuse or nodular. Clinically, LUS may support screening of asymptomatic patients, help identify those requiring HRCT confirmation, and offer a repeatable method to monitor disease evolution. Counting B-lines remains central, while pleural-line assessment may improve interpretation and diagnostic confidence.

Overall Take Home Messages

- LUS may support ILD screening directly in rheumatology clinics.
- Abnormal LUS should guide, not replace, HRCT-based assessment.
- Pleural-line changes add value beyond B-line counting alone.
- Standardised definitions are essential for wider clinical use.

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CAPILLAROSCOPY

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CAPILLAROSCOPY

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Ritasman Baisya



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SESSION 10 – CAPILLAROSCOPY SKILLS SESSION | Wednesday, 3 June 2026

This session, led by Vanessa Smith and Carina Mihai, emphasised the role of nailfold capillaroscopy (NFC) in SSc and related disorders, underscoring its value in evaluating Raynaud's phenomenon and early recognition of SSc. Key NFC parameters include capillary density, dimension, morphology and microhaemorrhages. Abnormal features include density below 7/mm and giant capillaries over 50 µm. Normal morphologies are hairpin, tortuous, crossing or convex; abnormal morphologies are concave or unlisted.

Scleroderma stages are defined as early (normal density, giant capillaries, normal morphology), active (density 4–6/mm, giant capillaries, abnormal morphology), and late (fewer than 3/mm, no giant capillaries, increased abnormal morphology). The EULAR/Scleroderma Clinical Trials Consortium consensus acknowledges that normal individuals may show non-specific abnormalities, such as low density or capillary diameter of 20–50 µm.

The Le Roy criteria identify a 65.9% five-year risk of SSc in Raynaud's patients with specific NFC findings. The VEDOSS criteria and a fast-track algorithm, enabling rapid, reproducible interpretation based on capillary density and giant capillary presence, were also discussed, with a scleroderma-pattern NFC plus SSc-specific autoantibodies reaffirmed as a strong predictor of progression. A hands-on workshop and a 10-year reflection from the EULAR Study Group on Microcirculation in Rheumatic Diseases concluded the session.

Overall Take Home Messages

- Density, dimension, morphology and microhaemorrhages are the key NFC parameters to assess.
- In healthy individuals, NFC may show non-specific findings such as low density or capillary diameter of 20–50 µm.
- The scleroderma pattern is classified as early, active or late, with giant capillaries (>50 µm) as a hallmark feature.
- A fast-track algorithm enables reliable differentiation of scleroderma from non-scleroderma patterns in practice.

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THE VASCULAR CONTINUUM IN SYSTEMIC SCLEROSIS

Chosen and written by
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THE VASCULAR CONTINUUM IN SYSTEMIC SCLEROSIS



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SESSION 11 – MEET THE EULAR EXPERT: SCLERODERMA | Thursday, 4 June 2026

In this interactive session, prof Francesco Del Galdo provided an updated perspective on vascular disease in systemic sclerosis (SSc). Clinical manifestations traditionally considered separately, including Raynaud's phenomenon (RP), digital ulcers (DU), pulmonary arterial hypertension (PAH), and cardiac involvement, may represent different pathological expressions of a vascular continuum, in which clinical symptoms emerge only after years of progressive vascular remodelling and tissue injury.

Biological disease activity often precedes diagnosis by many years, and complications such as DU and PAH may reflect the cumulative consequences of longstanding vascular damage rather than the onset of disease itself. The speakers also highlighted that vascular and fibrotic pathways coexist throughout the disease course, with individual patients exhibiting varying contributions from each domain. This concept reinforces the heterogeneity of SSc and supports a personalized approach to disease assessment and management.

The session reinforced a treat-to-target approach for RP, emphasizing that worsening symptoms should be interpreted within the broader clinical context, as environmental factors and psychological stress may influence symptom burden independently of disease activity. For patients with refractory RP and DU, botulinum toxin was highlighted as a promising local therapeutic option.

Regarding PAH, substantial pulmonary vascular damage is often already present at diagnosis, supporting early combination therapy and risk-based follow-up. Emerging therapies such as sotatercept represent promising advances, with the potential to restore dysregulated signalling pathways central to PAH pathogenesis.

Overall Take Home Messages

- Different vascular manifestations may represent a pathogenic continuum in SSc.
- Treat-to-target approaches and individualized principles are being increasingly applied in SSc.
- Complex manifestations, such as PAH, require multidisciplinary management.

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What's New in Systemic Sclerosis?

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By EUSTAR's Young Investigators group



What's New in Systemic Sclerosis?

Chosen and written by:



Rahma A. Elziaty



Cristina-Andreea Vrancianu

HOT Session | | Friday, 5 June 2026

SCLEROSIS: DECIPHERING PATHOGENESIS, UNVEILING COMORBIDITIES, AND PAVING THE WAY FOR TARGETED TREATMENT | *Oliver Distler* (Switzerland)

Systemic sclerosis treatment is entering a new phase in which disease-modifying strategies are moving beyond conventional organ-based management. While treatment decisions are currently guided primarily by clinical phenotype and organ involvement, emerging data suggest that molecular profiling may help explain the marked heterogeneity in treatment response observed across patients.

The central theme of the session was the pursuit of precision medicine. Despite advances in molecular profiling, biomarkers, and imaging, no validated biomarker currently exists for routine treatment selection. Data from the SENSIS and INBUILD trials showed that neither inflammatory markers nor HRCT patterns such as UIP reliably predict response to nintedanib. Instead, treatment decisions should be guided by risk stratification, extrapulmonary manifestations, and matching the patient's phenotype to the population studied in clinical trials.

The evolving therapeutic landscape offers new hope. Nerandomilast, a PDE4B inhibitor, demonstrated promising efficacy in progressive fibrosing ILD and may simultaneously target inflammation, fibrosis, and vasculopathy. In addition, early studies of CAR-T cell therapy and B-cell/plasma-cell targeting strategies have shown encouraging results, supporting a future shift toward mechanism-based and potentially disease-modifying therapies in systemic sclerosis.

Overall Take Home Messages

- No validated biomarker is currently available to guide treatment selection in SSc.
- Organ involvement, risk stratification and phenotype-guided therapy remain the cornerstone of clinical decision-making.
- Molecular subsetting may help identify patients most likely to benefit from specific therapies.
- PDE4B inhibition represents a promising new approach for SSc-associated interstitial lung disease.
- CAR-T therapy highlights the potential of B-cell targeting as a disease-modifying strategy in systemic sclerosis.

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BASIC SCIENCE INSIGHTS INTO SYSTEMIC SCLEROSIS

Chosen and written by
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BASIC SCIENCE INSIGHTS INTO SYSTEMIC SCLEROSIS

Chosen and written by:



Davide Mohammad Reza Beigi

BASIC POSTER TOURS: BASIC SCIENCE INSIGHTS INTO SYSTEMIC SCLEROSIS | Wednesday, 3rd June 2026

POS0011 - Multi-Omics Reveals a Vascular-Immune Niche Characterizing Regressive Skin Fibrosis in Systemic Sclerosis | Astrid Hofman (Switzerland)

This poster highlighted skin fibrosis regression as a biologically informative process that may give insights into SSc pathogenesis. The study compared regressors and non-regressors in terms of serum proteomics, PBMC profiling and skin transcriptomics to identify vascular and immune features associated with different trajectories of skin disease. The key conceptual advance is that fibrosis regression seems to be characterized by a systemic reduction in monocyte extravasation and a dampened inflammatory profile. These findings may help refine molecular stratification of patients with cutaneous SSc and support tissue-informed target discovery.

Key Highlights

- Skin fibrosis regression may reflect defined vascular-immune states
- Endothelial injury and perivascular immunity remain central in SSc skin

POS0012 - Spatial transcriptomics identifies phenotypic shifts in fibroblasts upon CD19-CAR T-cell therapy in systemic sclerosis skin | Yi-Nan Li (Germany)

This longitudinal spatial-transcriptomic study provided mechanistic insight into tissue remodeling after deep B-cell depletion with CD19-CAR T cells. Beyond clinical improvement, the relevant message was the observed shift in fibroblast phenotypes, including changes in myofibroblast populations and inflammatory extracellular-matrix programs. These findings suggest that immune-directed therapies

may influence fibroblast biology within the skin microenvironment, supporting the concept that effective immune targeting can be associated with downstream tissue remodeling in SSc.

Key Highlights

- B cell depletion with CD19-CAR T therapy may modify pathogenic fibroblast states
- Immune targeting can reshape the fibrotic skin niche

POS0015 - Sema4A neutralization reduces fibrosis and inflammation in a mouse model of systemic sclerosis and a 3D model of immunity-driven fibrosis | Jaime Marty Lobo (Spain)

This poster was translationally attractive because it identified Sema4A as a mediator linking immune activation and fibrosis potentially involved in SSc.

Il Lavoro mostra come questo marcatore sia aumentato nel siero di pazienti con SSc e che la sua neutralizzazione è in grado di ridurre la 3D contraction di matrice con fibroblasti indotta da PMBC e siero di pazienti SSc oltre che marker di fibrosi nel SSc blemocine modello murino. Although still preclinical, these data support Sema4A as a candidate therapeutic axis in immune-driven fibrotic remodeling and provide a rationale for further validation in SSc-associated organ fibrosis.

Key Highlights

- Sema4A is elevated in SSc patients compared to HC
- Sema4A neutralization reduced fibrosis across experimental models

POS0016 - Spatial Profiling of Scleroderma Renal Crisis Reveals Fibrotic Remodeling and Complement Activation in the Kidney | Minrui Liang (China)

This study brought spatial biology to scleroderma renal crisis, a rare but severe SSc complication that remains mechanistically underexplored comparing SSc biopsies with lupus nephritis and healthy controls samples. Spatial profiling of SSc kidney biopsies revealed a unique coordinated fibrotic remodeling, endothelial dysfunction, complement activation and immune-cell accumulation across renal compartments. The identification of complement-related pathways within tissue niches is particularly relevant, as it may help explain microangiopathic injury and generate tissue-informed therapeutic hypotheses for a complication with limited targeted treatment options.

Key Highlights

- SRC shows spatially coordinated fibrosis and complement activation
- Spatial profiling may reveal targetable renal disease niches

Overall Take Home Messages

- Basic SSc research is moving from single markers to spatially defined pathogenic niches and multi omic analysis
- Endothelial injury remains central to skin fibrosis and renal crisis
- Immune targeting may reshape fibroblast states, not only suppress inflammation
- 3D and spatial models are generating new therapeutic hypotheses in SSc

-EULAR 2026 Highlights Brochure-

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