Oral presentation 2

時　間：113 年 12 月 14 日(星期六) 08：45-09：15

地　點：新竹喜來登大飯店 3 樓梅花桐花百合廳

座長/Moderator	基隆長庚紀念醫院 吳詹永嬌 醫師
08:45 - 08:57	**The Risk of Systemic Sclerosis, Pulmonary Artery Hypertension, and Interstitial Lung Disease in Patients with Anti-centromere Protein A and/or Anti-Ro52 Antibodies: A Retrospective Cohort Study** Yao-Chia Yang[1], Tien-Ming Chan[1,2] 楊曜嘉 [1]，詹天明 [1,2] Affiliations 1. Division of Rheumatology, Allergy and Immunology, Chang Gung Memorial Hospital, 5, Fu-Hsing Street, Taoyuan, 333, Taiwan. 林口長庚紀念醫院風濕免疫科 2. Department of Internal Medicine, College of Medicine, Chang Gung University, Taoyuan 33305, Taiwan 長庚大學醫學院
08:57 - 09:00	Q & A
09:00 - 09:12	**Dermal cellular senescence and endothelial-to-mesenchymal transition responses to treatment in systemic sclerosis** Yu-Hsiang Chiu[1, 2], Marijke van Dijk[3], Roel Goldschmeding[3], Jeska de Vries-Bouwstra[4], Jacob M. van Laar[1], Julia Spierings[1] 裘喻翔 [1,2] 1 Department of Rheumatology and Clinical Immunology, University Medical Center Utrecht, Utrecht, the Netherlands 2 Division of Rheumatology/Immunology/Allergy, Department of Medicine, Tri-Service General Hospital, National Defense Medical Center, Taipei, Taiwan 三軍總醫院風濕免疫過敏科 3 Department of Pathology, University Medical Center Utrecht, Utrecht, the Netherlands 4 The Department of Rheumatology, Leiden University Medical Center, Leiden, the Netherlands
09:12 - 09:15	Q & A

The Risk of Systemic Sclerosis, Pulmonary Artery Hypertension, and Interstitial Lung Disease in Patients with Anti-centromere Protein A and/or Anti-Ro52 Antibodies: A Retrospective Cohort Study

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Abstract

Background

Among patients suspected of having systemic sclerosis (SSc), anti-centromere protein A antibody (Anti-CENP-A) is frequently tested. However, the relationship between these antibodies and overlapping syndrome, interstitial lung disease (ILD), pulmonary artery hypertension (PAH), and cancer, particularly in combination with Anti-Ro52 antibodies, remains unclear.

Methods

Patients suspected of having SSc were examined at Linkou Chang Gung Memorial Hospital from January 1, 2022, to December 31, 2022, with observation until December 31, 2023. We retrospectively extracted data from patients tested for Anti-CENP-A and analyzed their characteristics and the relationship between Anti-CENP-A and autoimmune diseases, ILD, PAH and cancer. Outcomes were also evaluated based on the presence or absence of Anti-Ro52 antibodies.

Results

Total 564 patients were included, with 112 patients tested positive for Anti-CENP-A. Among the 112 patients, 33 were diagnosed with SSc (29.5% vs. 13.5% in negatives). Patients with positive Anti-CENP-A demonstrated a higher risk of PAH compared to those with negative results (11.6% vs. 1.6%, $p<0.001$). No significant difference was observed concerning ILD and cancer. Subgroup analysis revealed a higher risk of ILD among double-positive Anti-CENP-A and Anti-Ro52 patients, compared to those with positive Anti-CENP-A and negative Anti-Ro52 (20.7% vs. 3.6%, $p=0.009$).

Conclusion

Anti-CENP-A antibody is a significant biomarker for SSc. In patients suspected of having SSc, positive Anti-CENP-A antibody may indicate a higher risk of PAH. ILD and cancer risks were not increased. The concomitant presence of Anti-Ro52 and Anti-CENP-A may refine ILD risk assessment. Further studies with larger populations may be warranted.

Dermal cellular senescence and endothelial-to-mesenchymal transition responses to treatment in systemic sclerosis

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Abstract

Background:
Cellular senescence and endothelial to mesenchymal transition (EndMT) are profibrotic pathogenesis involved in systemic sclerosis (SSc), but how they respond to treatment is elusive.

Methods:
Forearm skin biopsies were analysed from diffuse cutaneous SSc patients undergoing autologous haematopoietic stem cell transplantation (aHSCT) or cyclophosphamide pulse (CYC) treatment in the ASTIS trial at baseline and 6 months. The severity of fibrosis, inflammation, senescence, EndMT and tissue remodelling were examined in histopathology.

Results:
Fourteen pairs of skin biopsies were evaluated. Decrease in modified Rodnan skin score (mRSS) was more pronounced in aHSCT treated patients compared to CYC at 6 months (median change -14 [IQR -16–-9] versus -6 [IQR -9–-4], respectively, $p = 0.028$). Histologically, expression of uPAR on fibroblasts, P21 on vessels and EndMT decreased after treatment in both groups, yet the reduction was more pronounced in the aHSCT group. Poor skin response was associated with a high baseline CTGF score on fibroblasts and a low baseline P21 score on vessels, odds ratio (OR) 1.43 and 0.41, respectively. Furthermore, poor response was also seen in patients with a rise in CTGF on fibroblasts (OR 1.29) and P21 on vessels (OR 3.02) after treatment, $p < 0.001$.

Conclusions:
Both aHSCT and CYC for dcSSc reduced skin thickening clinically and attenuated profibrotic processes. EndMT and uPAR were associated with fibro-remodelling activity, whereas senescence, CTGF, uPAR and vascularity may predict treatment response.