# Poster Round
# 海報目錄

時　間：114 年 11 月 23 日(星期日)10:40-11:05
地　點：台中林酒店 3F 世紀廳海報區
主持人：盧俊吉醫師

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| 編號 | 題目 | 作者 |
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| TCR 56 | First Clinical Report of Anti-RuvBL1/2 Autoantibody-positive Patients in Taiwan<br>台灣Anti-RuvBL1/2 自體抗體陽性病患臨床報告 | 張詩欣, 李如璧, 賴怡樺, 汪政宏, 藍忠亮* |
| TCR 57 | Decoding the AC-4 ANA Pattern: A Distinct Serological Signal for Autoimmunity Risk Stratification<br>解碼AC-4 ANA型態：自體免疫風險分層的獨特血清學訊號 | 詹天明*蕭朝陽,黃思偉,尤瀚華,謝孟儒,蔡昀臻,李蒔青,楊曜嘉,趙祥元,許鐘元,蘇昱日,林科名,林靖麒, 羅緯麟, 淑芬,余光輝 |

**海報摘要 TCR39**

**"Interpreting Myositis Autoantibody Line-Blot Immunoassays in Real-World Settings: Implications for Diagnostic Accuracy for Inflammatory Myopathies"**

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**廣泛運用抗肌炎抗體線性免疫檢測於發炎性肌炎診斷之臨床預測意義**

賴佩幸, 鄭喬峯, 李岱儒, 張庭暐, 高瑞鴻, 林冠言, 白紹玉, 呂政勳, 沈玠妤, 李克仁, 藍鼎淵, 謝松洲

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**Background**

Myositis autoantibody line blot immunoassay (LIA) is widely adopted beyond rheumatology. We explored its predictive value in real-world settings.

**Methods**

A retrospective study of consecutive patients received myositis LIA via *EUROLINE Autoimmune Inflammatory Myopathies 16 Ag kit* in National Taiwan University Hospital from April 2021 to March 2023.

The LIA testing indications were categorized into: *suspected IIM* (presenting multiple clinical domain manifestations), *single clinical domain* (*pulmonary, muscular, joint, skin, cardiovascular*) and *autoimmune work-ups*. We analyzed autoantibody positive predictive value (PPV) based on testing indications. All analyses were performed using R software.

**Results**

A total of 677 patients were included. Among the 162 patients with IIM, the most common subtypes were dermatomyositis (45.1%), anti-synthetase syndrome (27.8%) and polymyositis (14.8%). LIA demonstrated overall sensitivity 80.2% and specificity 66.4%. *Suspected IIM* exhibited the highest PPV (95.9%) than *single clinical domain* or *autoimmune work-ups* (Figure 1).

While PPV of most autoantibodies were high for *suspected IIM* (Figure 2A), autoantibodies with higher PPV for *pulmonary manifestations* (Figure 2B) were multiple coexisting anti-aminoacyl-tRNA synthetase antibodies, TIF1r and MDA5. Autoantibody PPV was low for cases tested for *joint, skin manifestations* and *autoimmune work-ups* (Figure 2C). We proposed an algorithm for interpreting myositis LIA for the expanded clinical context (Figure 3).

**Conclusion**

Pre-test clinical assessment is valuable under wider clinical applications as the diagnostic accuracies increase with pre-test probability. Autoantibody PPV varies with specific autoantibodies and clinical Indications. Further studies are necessary to elucidate whether autoantibody positivity in single domain manifestations would predict disease course.

| | Suspected IIM | Muscular | Pulmonary | Joint | Skin | Autoimmune |
|---|---|---|---|---|---|---|
| PPV (%) | 95.9 | 45.2 | 31.5 | 25.0 | 13.1 | 3.8 |
| LR+ | 4.78 | 3.48 | 2.78 | 1.67 | 2.07 | 2.46 |

**Figure 1. Myositis Autoantibody Line Blot Immunoassay (LIA) Clinical Performances, Stratified by Testing Indications. Heatmap demonstrating positive predictive value and positive likelihood ratio of myositis LIA, stratified by testing indications.**
PPV: positive predictive value. LR+: positive likelihood ratio.

(2A)

| | TIF1r | Mi2 | MDA5 | Ku | PMScl | Jo1 | SRP | PL7 | SAE | OJ | Ro52 | EJ | NXP2 | PL12 | MASyS | MMSA | MMAA |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Overall PPV | 100 | 91 | 100 | 85.7 | 100 | 100 | 100 | 100 | 100 | 100 | 66.7 | 100 | 100 | N/A | 100 | 100 | 100 |
| Low Positivity | 100 | 80 | 100 | 83 | 100 | 100 | 100 | 100 | N/A | 100 | 100 | N/A | N/A | N/A | N/A | | |
| Strong Positivity | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | N/A | 50 | 100 | 100 | N/A | N/A | | |

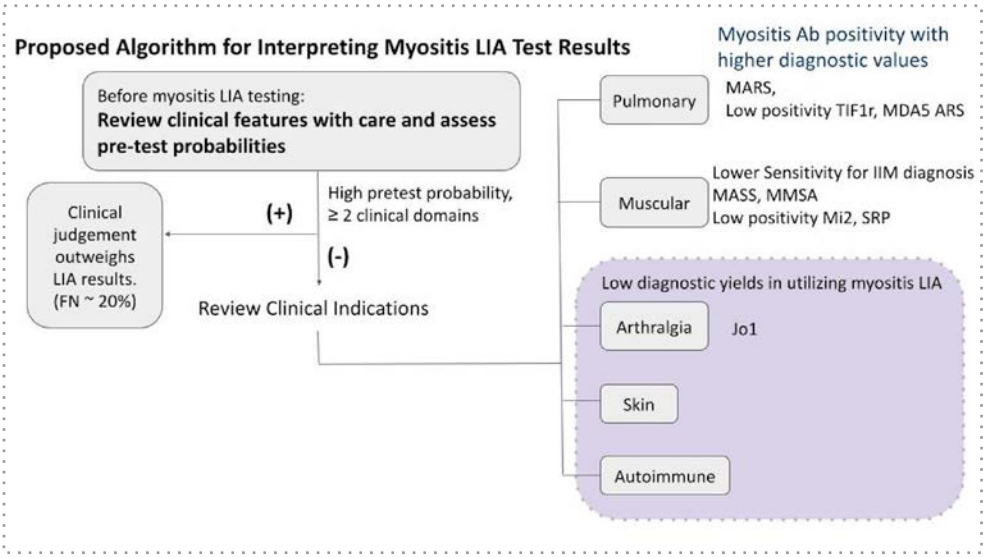
(2B)

| | TIF1r | Mi2 | MDA5 | Ku | PMScl | Jo1 | SRP | PL7 | SAE | OJ | Ro52 | EJ | NXP2 | PL12 | MASyS | MMSA | MMAA |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Overall PPV | 100 | 0 | 77.8 | 0 | 8.3 | 100 | 14.3 | 40 | 0 | 100 | 1.7 | 72.7 | 0 | 71.4 | 100 | 40 | 0 |
| Low Positivity | 100 | 0 | 50 | 0 | 14.2 | 100 | 0 | 14.3 | N/A | N/A | 0 | 25 | 0 | 50 | | | |
| Strong Positivity | N/A | 0 | 100 | 0 | 0 | 100 | 50 | 100 | 0 | 100 | 2.1 | 100 | 0 | 80 | | | |

(2C)

| | TIF1r | Mi2 | MDA5 | Ku | PMScl | Jo1 | SRP | PL7 | SAE | OJ | Ro52 | EJ | NXP2 | PL12 | MASyS | MMSA | MMAA |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Overall PPV | 0 | 0 | 0 | 0 | 0 | N/A | 0 | 33.3 | 0 | N/A | 0 | N/A | N/A | 0 | 0 | 0 | 0 |
| Low Positivity | 0 | 0 | 0 | 0 | 0 | N/A | N/A | 0 | N/A | N/A | 0 | N/A | N/A | 0 | | | |
| Strong Positivity | N/A | 0 | N/A | 0 | 0 | N/A | 0 | 50 | 0 | N/A | 0 | N/A | N/A | N/A | | | |

**Figure2**
**Myositis Autoantibody Positive Predictive Value (PPV), Stratified by LIA Testing Indications. Heatmap demonstrating PPV for individual autoantibody PPV(%) among the cases with LIA tested for (2A) Suspected IIM, (2B) Pulmonary manifestations and (2C) autoimmune work-ups.** Multiple autoantibody positivity was distinguished from single antibody positivity. Overall positivity PPV included PPV of both low and strong positivity. MMSA: presence of more than one myositis specific antibody. MASyS: presence of more than one anti-aminoacyl-tRNA synthetase antibody. MMAA: presence of more than one myositis associated antibody. N/A: non-applicable.




**igure 3. Proposed Algorithm for Interpreting Myositis LIA Test Results**

**海報摘要 TCR40**

**Loss of life expectancy and its determinants in newly diagnosed patients with polymyositis and dermatomyositis: a nationwide cohort study**

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**新診斷多發性肌炎與皮肌炎患者之預期壽命損失及其影響因素：全國性世代研究**

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**Background:** Polymyositis (PM) and dermatomyositis (DM) are rare systemic autoimmune diseases with notable morbidity and mortality. Despite advances in immunosuppressive therapy, the long-term survival impact across different age groups, sexes, and comorbid conditions remains underexplored. This study aimed to estimate the life expectancy (LE) and loss-of-LE in newly diagnosed PM/DM patients, and to examine the prognostic influence of comorbid cancer and interstitial lung disease (ILD).

**Methods:** We conducted a retrospective cohort study using Taiwan's National Health Insurance Database (2000–2022). Adults aged ≥20 years with newly diagnosed PM/DM confirmed by catastrophic illness certification were included. Cancer was defined by registry records within two years of diagnosis, and ILD by ≥3 outpatient visits or ≥1 hospitalization with relevant ICD codes. LE and loss-of-LE were estimated using a semi-parametric spline-based extrapolation method.

**Results:** A total of 3,296 patients were included. The overall LE was 13.0 years, and the loss-of-LE was 13.5 years. Patients aged 20–49 had the highest LE (33.99 years) but also the greatest loss-of-LE (11.03 years). Those with cancer had a markedly lower LE (11.21 years) and higher loss-of-LE (16.5 years) compared to non-cancer patients, with a mortality rate of 66.1%. Patients with ILD also had higher loss-of-LE (13.57 vs. 9.64 years).

**Conclusion:** PM/DM is associated with significant life expectancy loss. Younger patients, despite longer survival, experience substantial lifetime burden. Comorbid cancer and ILD are major predictors of worse prognosis, underscoring the need for early risk stratification and integrated multidisciplinary care to improve long-term outcomes.

**The key results as shown in the Table of next page:**

| | | n | no. of death (%) | Age (SD), year | LE (SE), year | loss of LE (SE), year |
|---|---|---|---|---|---|---|
| All | | | | | | |
| | 20-49 | 1435 | 284(19.79) | 38.64(8.03) | 33.99 (1.86) | 11.03 (1.88) |
| | 50-64 | 1244 | 475(38.18) | 56.77(4.26) | 16.26 (0.88) | 11.78 (0.88) |
| | >=65 | 617 | 414(67.10) | 72.14(5.61) | 6.78 (0.39) | 8.58 (0.39) |
| Male | | | | | | |
| | 20-49 | 425 | 111(26.12) | 38.69(8.11) | 28.21 (2.72) | 12.49 (2.78) |
| | 50-64 | 445 | 202(45.39) | 57.27(4.27) | 15.53 (1.06) | 9.22 (1.07) |
| | >=65 | 249 | 175(70.28) | 72.17(5.48) | 6.63 (0.67) | 7.06 (0.64) |
| Female | | | | | | |
| | 20-49 | 1010 | 173(17.13) | 38.62(8.00) | 35.36 (2.47) | 11.49 (2.48) |
| | 50-64 | 799 | 273(34.17) | 56.49(4.23) | 18.21 (1.15) | 11.73 (1.16) |
| | >=65 | 368 | 239(64.95) | 72.11(5.70) | 7.88 (0.69) | 8.56 (0.72) |
| ILD | | | | | | |
| | Y | 1058 | 402(38.00) | 52.63(12.40) | 18.6 (1.63) | 13.57 (1.64) |
| | N | 2238 | 771(34.45) | 51.34(15.03) | 23.8 (1.41) | 9.64 (1.39) |
| Cancer | | | | | | |
| | Y | 357 | 236(66.11) | 57.12(11.78) | 11.21 (1.53) | 16.5 (1.61) |
| | N | 2939 | 937(31.88) | 51.1(14.38) | 23.81 (1.1) | 9.91 (1.15) |

海報摘要 TCR41

**Antisynthetase Syndrome, a rare cause of Renal Failure**

**Y.C. Hsin, K.S. Tseng,**
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**抗合成酶抗體症候群患者併腎衰竭之案例分享**

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衛生福利部桃園醫院 風濕免疫過敏科

**Introduction:** Anti-synthetase syndrome is a rare autoimmune disorder characterized by autoantibodies against aminoacyl tRNA synthetases. Common features include interstitial lung disease, myositis, arthritis, and mechanics hands, while renal involvement is uncommon.

**Case presentation:** A 50-year-old man with hypertension, dyslipidemia, and prior stroke who presented with progressive myalgia, muscle weakness, dysphagia, and dyspnea. Laboratory findings showed elevated creatine kinase, positive ANA (AC-19, 1:160),anti-ENA and anti-EJ antibody. Despite initial steroid therapy, he developed aspiration pneumonia, acute kidney injury, and respiratory failure requiring intubation. Culture-confirmed CRAB infection prompted antibiotic adjustment. Despite intensive care and immunosuppressive treatment, the patient suffered upper gastrointestinal bleeding with hemorrhagic shock and died on June 4, 2025.formed nodules.

**Conclusion:** This case highlights a rare presentation of anti-synthetase syndrome with renal failure and rapid deterioration. Awareness of atypical organ involvement is essential for timely diagnosis and multidisciplinary management.

**海報摘要 TCR42**

**Abrocitinib use in refractory cutaneous manifestations of dermatomyositis: a case report**

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**Abrocitinib 用於皮肌炎難治性皮膚表現之個案報告**

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**Background**

Dermatomyositis (DM) is an idiopathic inflammatory myopathy with cutaneous, muscular, and internal organ involvement. Although standard treatment includes corticosteroids and immunosuppressants, some patients suffer from refractory skin manifestations. Janus kinase (JAK) inhibitors have emerged as potential off-label agents in these cases.

**Case :**

We present a 60-year-old male with TIF1-γ antibody-positive DM complicated by pulmonary hypertension and interstitial lung disease. Initial symptoms included proximal muscle weakness and dysphagia. Muscle and lung involvement improved with mycophenolate (720 mg/day), cyclosporin (50–100 mg/day), hydroxychloroquine, and corticosteroids (10–20 mg/day). However, his cutaneous disease remained active, with persistent erythema and severe pruritus over the malar area, forehead, V-area, and back, despite high-dose antihistamines (levocetirizine, ketotifen, doxepin), topical steroids, and calcineurin inhibitors. Skin biopsy confirmed dermatomyositis-related dermatitis.

Given the refractory nature of his dermatitis and steroid dependence, Abrocitinib 200 mg/day (self-paid) was initiated. Marked improvement in pruritus was observed within one week. Over 8 months of follow-up, corticosteroid and antihistamine dosages were tapered, and skin symptoms were largely controlled with minimal

residual erythema.

**Discussion:**

Although JAK inhibitors are not FDA-approved for DM, case reports have shown benefits with ruxolitinib, tofacitinib, upadacitinib, and baricitinib, particularly for cutaneous symptoms. Abrocitinib, a selective JAK1 inhibitor approved for atopic dermatitis, has not been widely studied in autoimmune diseases. In this case, Abrocitinib significantly improved refractory cutaneous manifestations in DM, allowing steroid tapering. To our knowledge, this is the first reported use of Abrocitinib in dermatomyositis, highlighting its potential in selected difficult-to-treat cases.

**海報摘要 TCR43**

**Incidence and mortality of primary Sjögren's syndrome in Taiwan: An updated population-based trends analysis**

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**台灣原發性修格蘭氏症的發生率與死亡率：最新全國人口趨勢分析**

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[2] 台南成功大學醫學院附設醫院內科部過敏免疫風濕科及國立成功大學醫學院

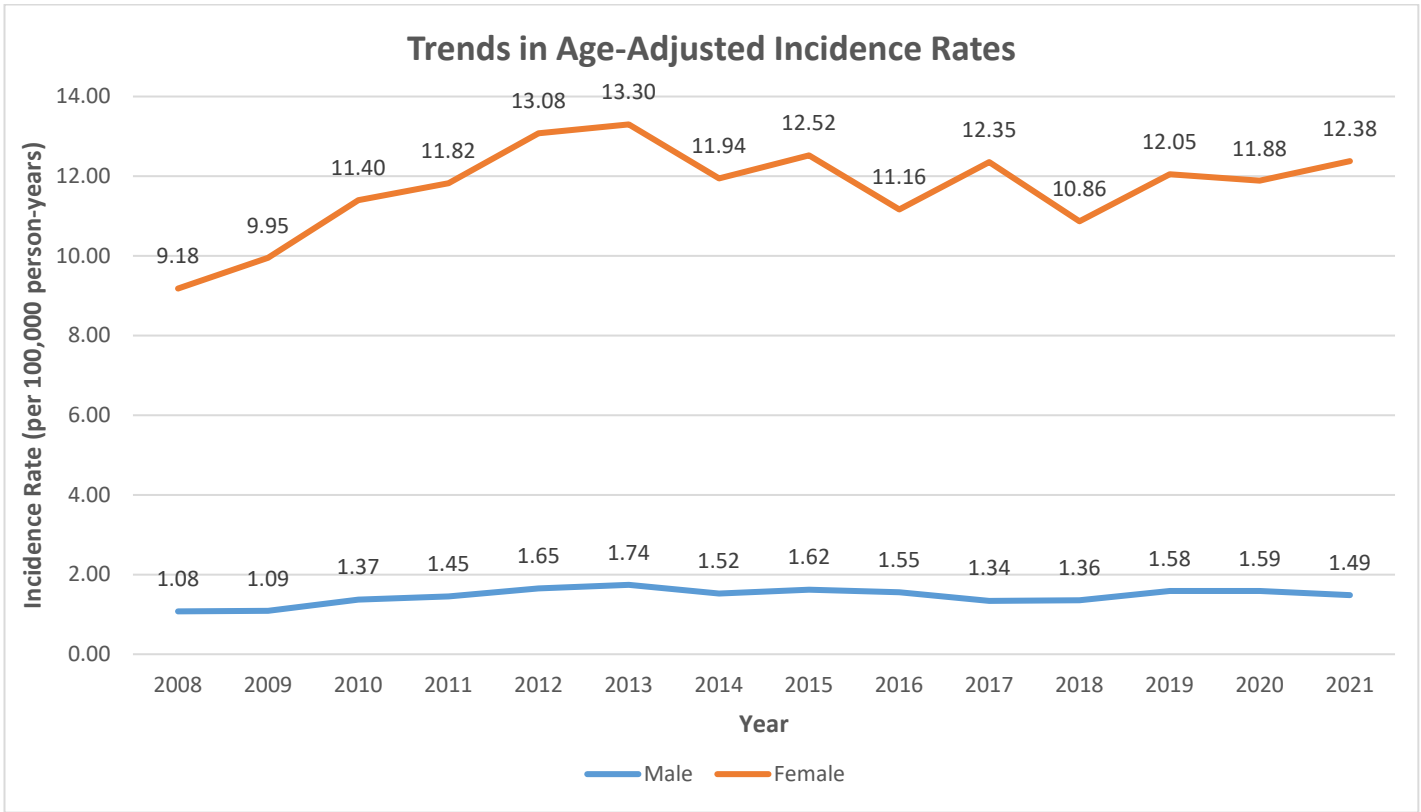
[3] 台灣國家衛生研究院高齡醫學與福利研究中心，雲林

**Background:** To update the population-based incidence and mortality rates of primary Sjögren's syndrome (pSS) by sex and age group in Taiwan from 2008 to 2021.

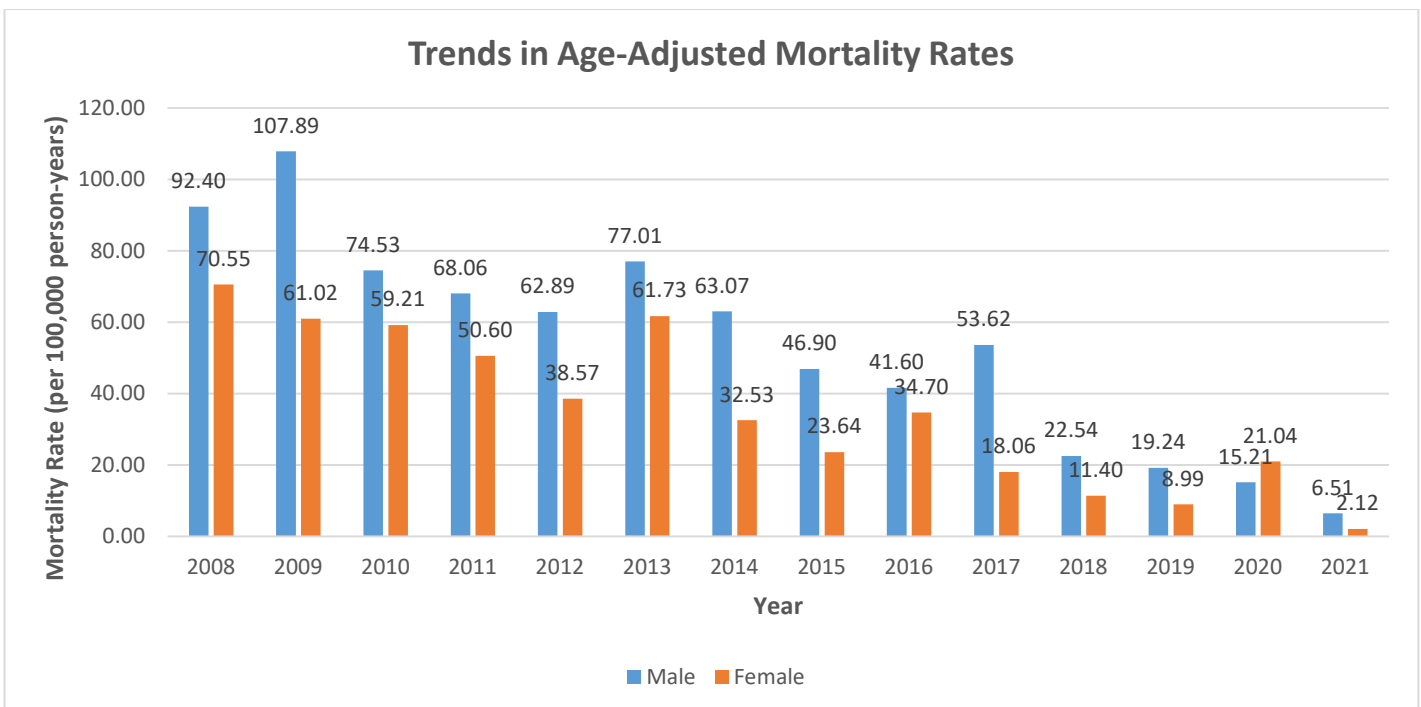
**Materials and Methods**: A retrospective analysis was conducted via Taiwan's National Health Insurance (NHI) claims database from 2008 to 2021. All newly diagnosed cases of pSS were included for calculated incidence and mortality rates by sex and age group.

**Results:** The incidence of pSS was significantly higher among women, with a total of 30,169 cases (89.13% women, 10.87% men), corresponding to an incidence rate of 7.89 per 100,000 person-years. The highest incidence rates for pSS were observed in the 55-64 age group (8,349 cases) and the 45-54 age group (7,229 cases). Unlike previous studies, this research noted an increase in the incidence rates of pSS, particularly among middle-aged and older women. Mortality analysis revealed that the main causes of death among pSS patients included tumors, hematologic, and immune system diseases, with a higher prevalence in women. Overall, the incidence of pSS showed an upward trend, peaking in 2013 at 7.64 per 100,000 person-years and reaching 7.04 in 2021. Mortality rates showed a declining trend, decreasing from 73.82 per 100,000 in 2008 to 2.99 in 2021, with higher mortality observed in males across all age groups.

**Conclusion:** This study reveals sex and age disparities in pSS incidence in Taiwan. An upward trend in pSS incidence and mortality necessitates targeted healthcare strategies for middle-aged and older women and targeted interventions to address these disparities and improve patient outcomes.




**Trends in Age-Adjusted Incidence Rates of Treated Primary Sjögren's Syndrome by Year in Taiwan (2008-2021)**




**Trends in Age-Adjusted Mortality Rates of Treated Primary Sjögren's Syndrome by Year in Taiwan (2008-2021)**

**Incidence rate of treated primary Sjögren's syndrome in Taiwan, 2008-2021**

| Age | Total | | | Male | | | Female | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | N | IR | 95% CI | N | IR | 96% CI | N | IR | 97% CI | Sex ratio |
| <25 | 488 | 0.55 | 0.53–0.58 | 62 | 0.13 | 0.12–0.15 | 426 | 1.00 | 0.95–1.05 | 7.45 |
| 25–34 | 1,721 | 3.48 | 3.39–3.56 | 117 | 0.47 | 0.42–0.51 | 1,604 | 6.58 | 6.41–6.74 | 14.12 |
| 35–44 | 3,883 | 7.32 | 7.20–7.43 | 327 | 1.24 | 1.17–1.31 | 3,556 | 13.29 | 13.07–13.52 | 10.7 |
| 45–54 | 7,229 | 14.11 | 13.94–14.28 | 547 | 2.16 | 2.06–2.25 | 6,682 | 25.85 | 25.53–26.16 | 11.99 |
| 55–64 | 8,349 | 19.4 | 19.18–19.61 | 866 | 4.12 | 3.98–4.26 | 7,483 | 33.95 | 33.55–34.34 | 8.23 |
| 65–74 | 5,531 | 22.65 | 22.34–22.95 | 812 | 7.06 | 6.81–7.30 | 4,719 | 36.54 | 36.01–37.07 | 5.18 |
| 75–84 | 2,570 | 19.55 | 19.16–19.94 | 460 | 7.67 | 7.31–8.03 | 2,110 | 29.53 | 28.88–30.17 | 3.85 |
| >84 | 398 | 8.87 | 8.42–9.31 | 90 | 4.38 | 3.92–4.84 | 308 | 12.65 | 10.6–11.4 | 2.89 |

Note: IR, incidence rate; CI, confidence interval; Sex ratio, Female/Male

**Mortality rate of treated primary Sjögren's syndrome by age in Taiwan, 2008–2021**

| Age | Total | | | Male | | | Female | | |
|---|---|---|---|---|---|---|---|---|---|
| | N | Mortality rate | 95% CI | N | Mortality rate | 95% CI | N | Mortality rate | 95% CI |
| <25 | 4 | 8.2 | 4.10–12.30 | 0 | — | — | 4 | 9.39 | 4.69–14.08 |
| 25–34 | 1,721 | 13.36 | 10.58–16.15 | 2 | 17.09 | 5.01–29.18 | 21 | 13.09 | 10.24–15.95 |
| 35–44 | 3,883 | 17.25 | 15.15–19.36 | 12 | 36.7 | 26.10–47.29 | 55 | 15.47 | 13.38–17.55 |
| 45–54 | 7,229 | 29.05 | 27.05–31.05 | 31 | 56.67 | 46.49–66.85 | 179 | 26.79 | 24.79–28.79 |
| 55–64 | 8,349 | 53.3 | 50.77–55.83 | 105 | 121.25 | 109.41–133.08 | 340 | 45.44 | 42.97–47.90 |
| 65–74 | 5,531 | 139.76 | 134.73–144.78 | 174 | 214.29 | 198.04–230.53 | 599 | 126.93 | 121.75–132.12 |
| 75–84 | 2,570 | 336.19 | 324.75–347.62 | 200 | 434.78 | 404.04–465.53 | 664 | 314.69 | 302.48–326.90 |
| >84 | 398 | 214.00 | 500.93–574.44 | 61 | 677.78 | 591.00–764.56 | 153 | 496.75 | 456.59–536.91 |

Note: CI, confidence interval

**海報摘要 TCR44**

**Hydroxychloroquine dose-dependently reduces the risk of incident diabetes in primary Sjögren syndrome patients on glucocorticoids: a nationwide population-based cohort study**

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**奎寧劑量依賴性地降低使用類固醇對原發性修格蘭氏症患者罹患糖尿病的風險：一項全國性的世代研究**

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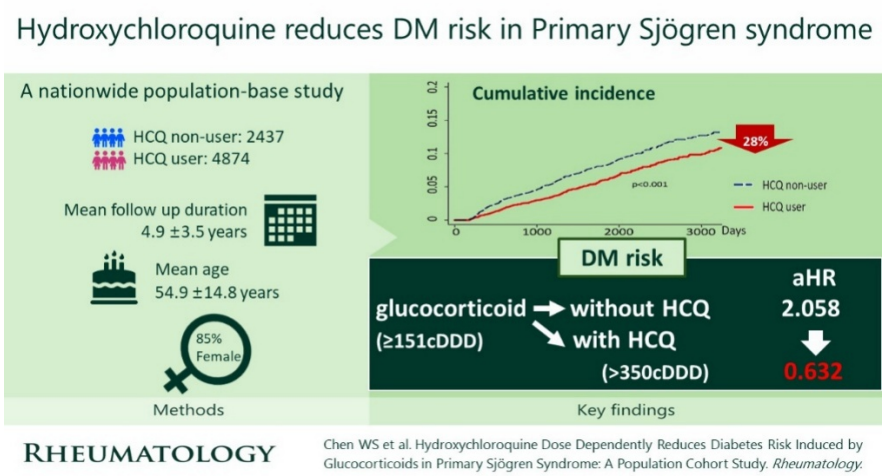
**Abstract**

**Background:** Hydroxychloroquine (HCQ) is commonly used to treat Sjögren syndrome (SS). Glucocorticoids, which are commonly applied for managing primary SS (pSS), can disrupt glucose metabolism and increase diabetes mellitus (DM) risk. HCQ reduces DM risk in systemic lupus erythematosus and rheumatoid arthritis.
Objective: This study aimed to investigate the relationship between HCQ and glucocorticoids in the incidence of new-onset diabetes in pSS.

**Methods:** This nationwide population-based cohort study identified patients diagnosed with pSS from the Taiwan's National Health Insurance Research Database from 2006 to 2015. Multivariate and stratified analyses, Kaplan–Meier method, and Cox proportional hazard regression were used to evaluate DM risk associated with the use of HCQ and glucocorticoid, both individually and in combination.

**Results:** Among pSS patients (4,874 HCQ users and 2,437 HCQ nonusers), 497 patients developed DM over an average follow-up of 4.89 years. Multivariate analysis revealed significantly lower adjusted hazard ratios (aHRs) for DM in HCQ users in the 151–350 cumulative defined daily dose (cDDD) and ≥351 cDDD subgroups (0.600, 95% CI: 0.454–0.794 and 0.326, 95% CI: 0.246–0.433, respectively) compared with HCQ nonusers. High-dose glucocorticoids (≥151 cDDD) were linked to increased DM risk (aHR: 1.833, 95% CI: 1.410–2.383). However, high-dose HCQ (>350 cDDD) mitigated this risk, even the risk caused by the use of high-dose glucocorticoids (≥151 cDDD) (aHR: 0.632, 95% CI: 0.421–0.948, P < 0.01).

**Conclusions:**Our study demonstrated that HCQ exposure significantly reduces the risk of developing diabetes in patients with pSS. While higher doses of glucocorticoids are associated with an increased diabetes risk, concurrent HCQ use mitigates this risk in a dose-dependent manner.



**海報摘要 TCR45**

**Subcutaneous panniculitis-like T cell lymphoma (SPTCL) responding to JAK inhibitors in patients initially diagnosed with primary Sjogren syndrome**

An 18-year-old girl initially presented with persistent fever and bilateral erythematous, tender subcutaneous nodules on the upper arms. She was diagnosed with primary Sjögren's syndrome based on positive anti-SSA antibodies and lymphoplasmacytic infiltration on a lip biopsy, and panniculitis proved by a skin biopsy. However, her fever persisted and the lesions responded poorly to hydroxychloroquine and prednisone (up to 15 mg/day). Notably, she had minimal sicca symptoms and was found to have hypogammaglobulinemia (IgG 300–400 mg/dL) upon seeking a second opinion in our hospital.

Further evaluation revealed intense FDG uptake in the upper arms on PET scan (Figure 1). Repeated skin biopsies eventually confirmed subcutaneous panniculitis-like T-cell lymphoma (SPTCL), which later spread to the face (Figure 2A). Given the unusual presentation of SPTCL and hypogammaglobulinemia, whole-exome sequencing was performed and identified a STAT3 gain-of-function (GOF) mutation.

The lymphoma was refractory to two cycles of R-CHOP chemotherapy, but responded dramatically to the JAK inhibitor baricitinib, with fading of the subcutaneous lesions (Figure 2B) and resolution of fever. A follow-up PET scan three months later confirmed complete remission.

STAT3 GOF mutations are known to cause combined immune dysregulation, including hypogammaglobulinemia, autoimmunity, and lymphoproliferative disease. While JAK inhibitors have shown efficacy in treating autoimmune symptoms in STAT3 GOF syndrome, to our knowledge, this is the first reported case of STAT3 GOF–associated lymphoma responding to JAK inhibition.

**Figure 1**

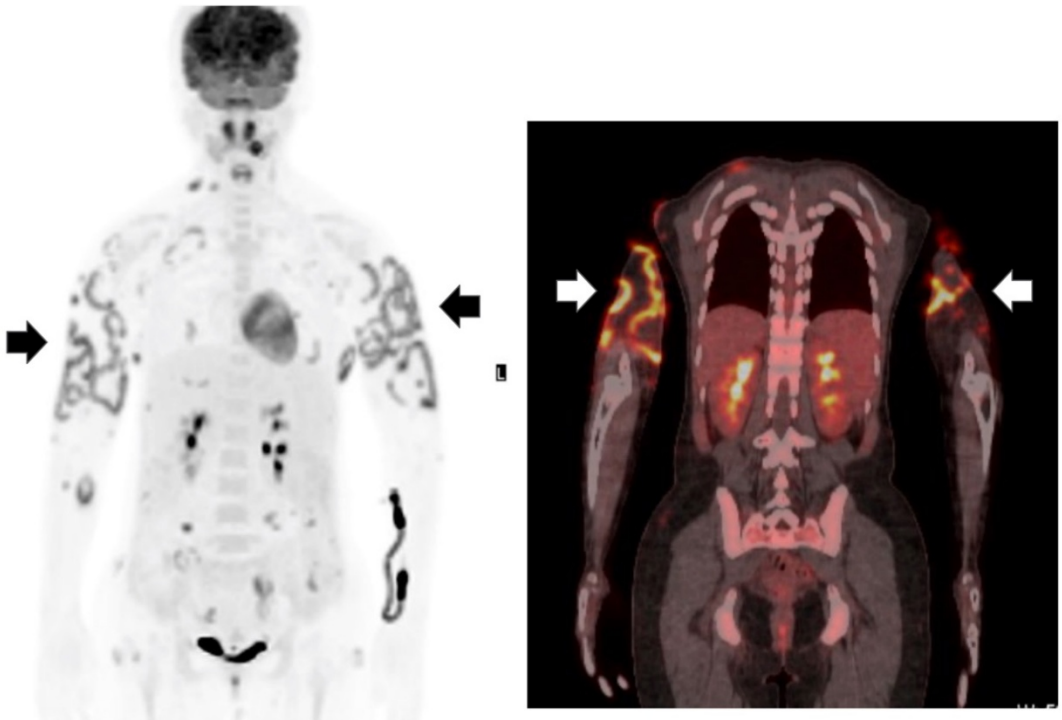
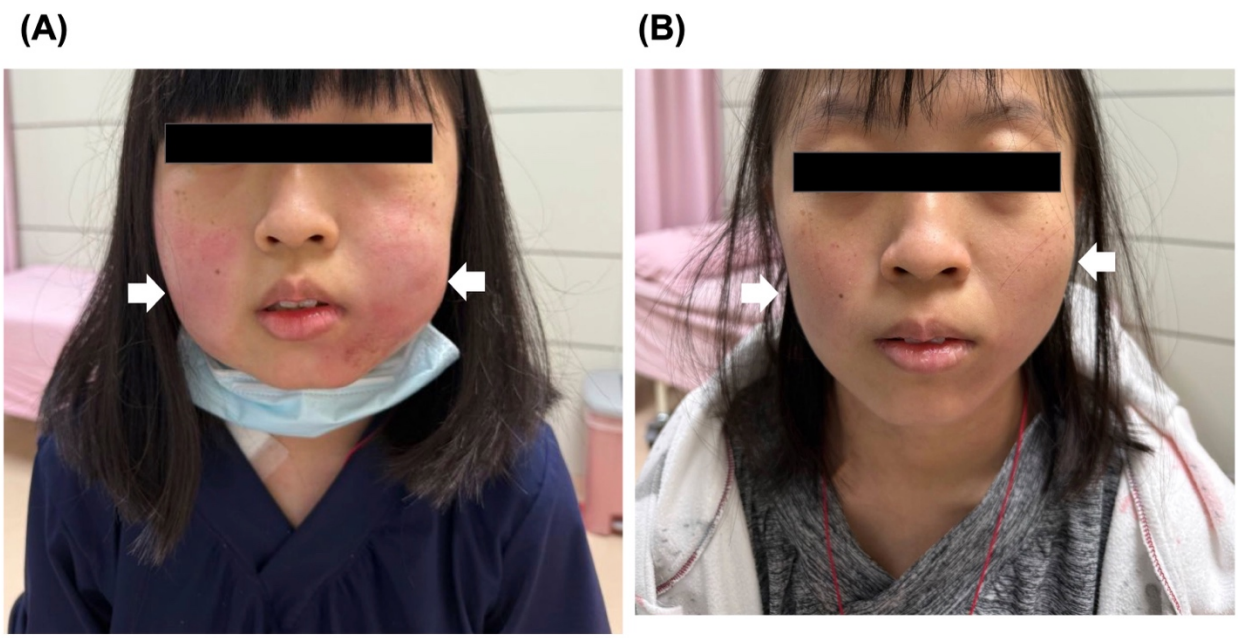


**Figure 2**




藍鼎淵 Ting-Yuan Lan [1], 李婉瑄 Wan-Hsuan Lee [2], 李岱儒 Tai-Ju Lee[1], 林冠言 Kuan-Yen Lin[1], 張庭暐 Ting-Wei Chang[1], 王恭宇 Kung-Yu Wang [1],吳政翰 Chang-Han Wu[1], 李克仁 Ko-Jen Li [3]

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**海報摘要 TCR46**

# Common variable immunodeficiency presenting as rheumatoid arthritis with Sjögren's syndrome

## 普通變異型免疫缺陷病以類風濕性關節炎併乾燥症為起始表現

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Several autoimmune diseases have been reported to be associated with common variable immunodeficiency disease (CVID), including rheumatoid arthritis and Sjögren's syndrome. On the other hand, approximately 20-30%of patients with rheumatoid arthritis develop secondary Sjögren's syndrome. A26-year-old woman had a 6-year history of chronic symmetric polyarthritis and 3-year history of sicca syndrome prior to admission for pneumonia. Rheumatoid arthritis with secondary Sjögren's syndrome had been diagnosed 1 year before. The patient had experienced 3 episodes of pneumonia during the previous 3 years. Markedly depressed serum immunoglobulin levels prompted a suspicion of common variable immunodeficiency, and the impression was confirmed after a series of examinations. Monthly administration of intravenous immunoglobulin (IVIG) alleviated the polyarthritis and improved the sicca syndrome. IVIG replacement therapy was ultimately successful in curing recurrent bacterial infections, chronic polyarthritis, and improving the severity of sicca syndrome.

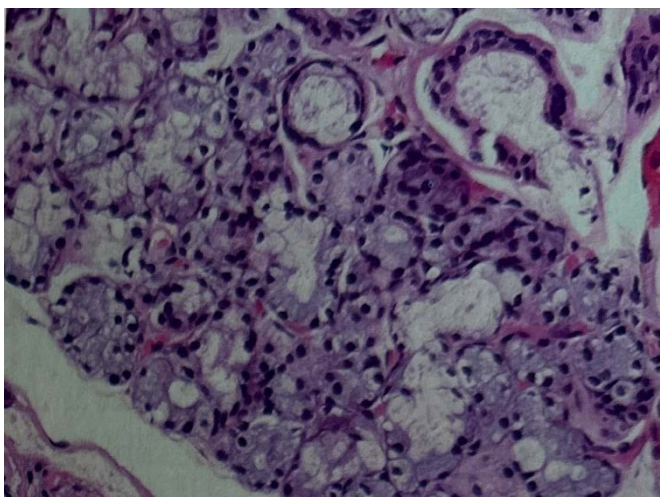

Fig. 1. Minor salivary gland biopsy from lower lip showed mild inflammatory change with some lymphocyte infiltration (hematoxylin and eosin stain, x 200).

**海報摘要 TCR47**

**Very-low-dose glucocorticoid lowers serious infection risk in ANCA-associated vasculitis: A network meta-analysis of randomized trials**

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**Background:** Serious infection is a significant issue for patients with ANCA-associated vasculitis on immunosuppressive therapy. Although achieving disease-free survival remains the primary goal, serious infections may undermine efficacy and lead to major morbidity and mortality. Therefore, comparing infection risk between regimens is essential.

**Methods:** PubMed, Cochrane Library, and ClinicalTrials.gov were systematically searched up to June 2025 for randomized controlled trials (RCTs) enrolling patients with microscopic polyangiitis or granulomatosis with polyangiitis, excluding eosinophilic granulomatosis with polyangiitis. Studies had to report serious infections during therapy. A random-effects network meta-analysis (NMA) was performed (reference: standard-dose glucocorticoids combined with cyclophosphamide (CYC) or rituximab (RTX). Odds ratios (ORs) and 95% confidence intervals (CIs) were calculated for serious infection risk.

**Results:** In this NMA including 18 RCTs (n = 2,423), therapies compared were: CYC or RTX with reduced glucocorticoid (PEXIVAS protocol), CYC or RTX + avacopan (with or without reduced-dose glucocorticoids), RTX + CYC + standard-dose glucocorticoid, CYC-AZA, methotrexate, mycophenolate mofetil. Only the very-low-dose glucocorticoid (LoVAS protocol) with CYC or RTX showed a significantly lower risk of serious infection (OR 0.25, 95% CI 0.10–0.65), while other regimens, including PEXIVAS protocol (OR 0.75, 95% CI 0.54–1.03), did not differ significantly from reference. Heterogeneity was low ($\tau^2 = 0.0851$, $I^2 = 15.1\%$, Q-test $p = 0.8874$), with no significant inconsistency.

**Conclusion:** Treatment with very-low-dose glucocorticoid plus CYC or RTX appears to lower serious infection risk versus both standard and reduced-dose (PEXIVAS protocol) regimens. Further prospective trials are warranted.

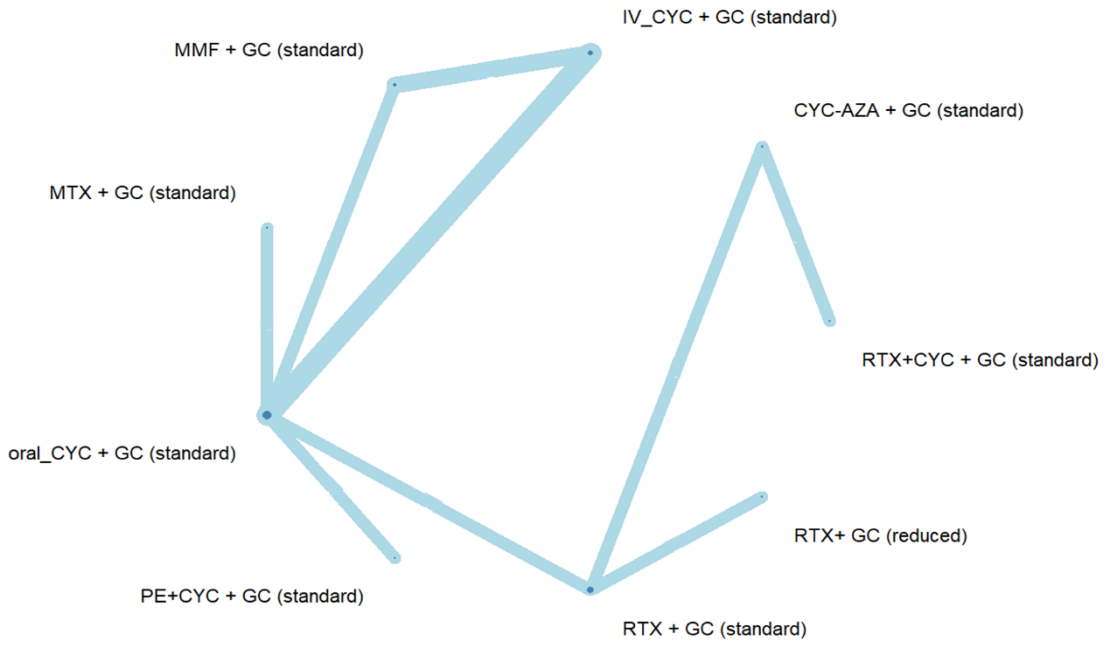


Fig. 1 Network plot for therapies assessed in the network meta-analysis.

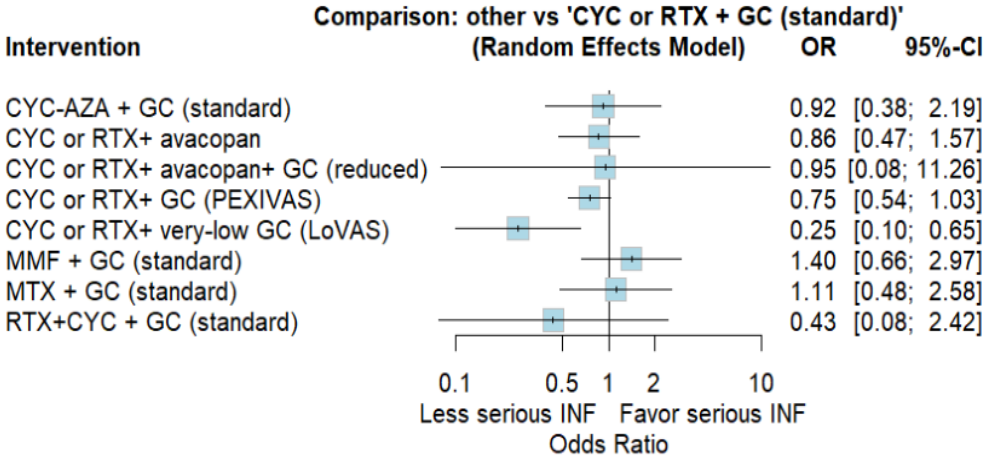


Fig. 2 Forest plot of serious infection risks across regimens in ANCA-associated vasculitis.

**海報摘要 TCR48**


文章標題中文　第二型冷凝球蛋白血症血管炎的個案報告
文章標題英文　Type 2 cryoglobulinemic vasculitis: A Case Report
文章作者中文　吳佩盈、鄭喬峯
文章作者英文　PEI-YING WU, CHIAO-FENG CHENG
服務機關單位中文　國立臺灣大學醫學院附設醫院
服務機關單位英文　National Taiwan University Hospital


**Case Presentation:**

An 82-year-old man with a history of metabolic syndrome and chronic kidney disease was admitted for evaluation of purpura. He was presented with recurrent episodes of multiple non-blanchable, palpable purpura, primarily involving the lower extremities and lower trunk (see figure 1). A skin biopsy obtained during one flare demonstrated features consistent with leukocytoclastic vasculitis (see figure 2). Laboratory evaluation revealed elevated erythrocyte sedimentation rate (ESR) >140 mm/hr, hypocomplementemia (low C3 and C4 levels) and disproportionately elevated IgM levels (ranging from 1160 to 1337 mg/dL), accompanied by mildly decreased IgG concentrations (<500 mg/dL) and normal IgA levels, suggestive of a selective overproduction of monoclonal IgM. Cryoglobulin testing was positive. Viral serologies for hepatitis B and C were negative. Serum protein electrophoresis (SPEP) identifies an IgM/kappa monoclonal gammopathy. Flow cytometry with a plasma cell dyscrasia (PCD) panel detected two small clonal B-cell populations. Bone marrow biopsy confirmed a diagnosis of immunoglobulin M monoclonal gammopathy of undetermined significance (IgM MGUS). Rheumatoid factor was markedly elevated (116 IU/mL), while anti-cyclic citrullinated peptide antibodies were negative, and the patient exhibited no clinical features of rheumatoid arthritis. Collectively, these findings were consistent with IgM MGUS–associated type II cryoglobulinemic vasculitis. The patient was subsequently treated with low-dose corticosteroids, hydroxychloroquine, and colchicine, resulting in complete resolution of the cutaneous vasculitis.

Figure 1

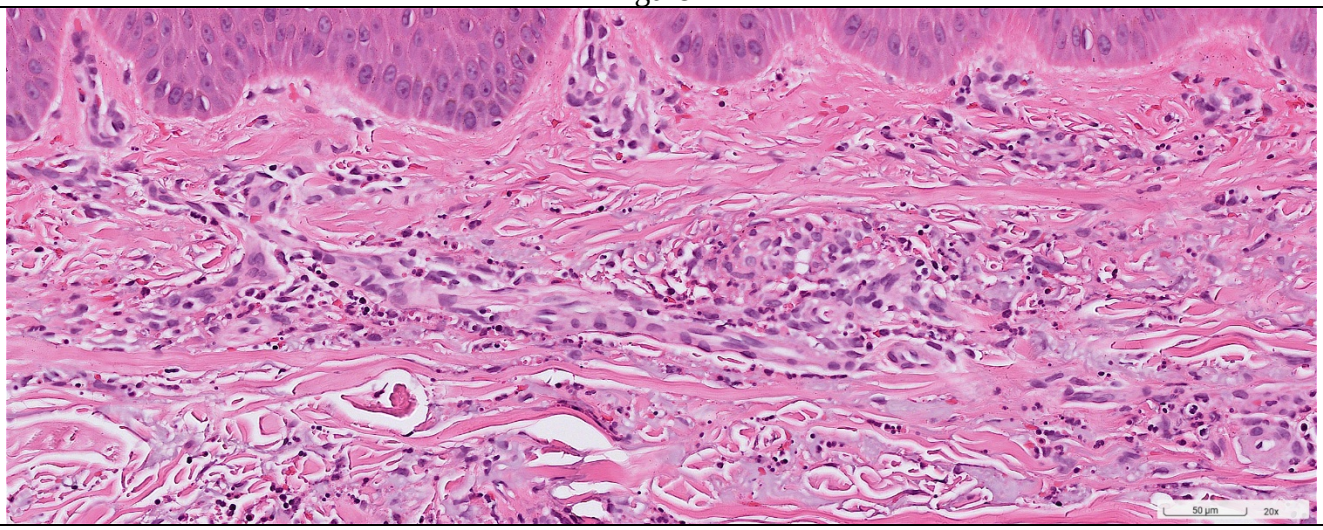


Figure 2

**海報摘要 TCR49**

**Case Report**

# Organizing pneumonia as initial presentation of anti-synthetase syndrome

# 以器質性肺炎為最初臨床表現之抗合成酶抗體症候群

孫俊明
SUN CHUN-MING
Yuan’s general hospital

**Abstract**:
This 45 y/o female is admitted via OPD due to swelling and pain over bilateral hands and dry cough for 1+ months. She has no fever, sorethroat, BW loss or muscle weakness. Sudden onset of severe SOB 2 days after admission. Chest CT reveals patchy consolidations and ground glass opacity over bilateral middle and lower lung fields. Autoimmune antibodies reveals negative ANA, RA factor and anti-CCP but positive anti-Jo1. Pulse steoid, cyclophosphamide and IVIG are administered. The condition is gradually improved during 3 weeks.

**海報摘要 TCR50**

**Signatures of Dysfunction and Senescence Distinct Natural Killer Cell Profiles in rheumatic diseases, including Systemic Lupus Erythematosus, Rheumatoid Arthritis, and Psoriatic Arthritis**

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[2] Department of Internal Medicine, Division of Rheumatology, Allergy and Immunology, Kaohsiung Chang Gung Memorial Hospital and Chang Gung University, Taiwan

**自然殺手細胞的功能失調與老化特徵：全身性紅斑狼瘡、類風濕性關節炎與乾癬性關節炎之異質性表現**

作者群：詹天明 [1], 許鐘元 [2], 蘇昱日 [2*]

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[2] 高雄長庚紀念醫院內科部風濕過敏免疫科及長庚大學

**Introduction**: Natural killer (NK) cell dysregulation is implicated in autoimmune rheumatic diseases, but direct comparisons across conditions are lacking. This study aimed to characterize and compare NK cell profiles in patients with systemic lupus erythematosus (SLE), rheumatoid arthritis (RA), and psoriatic arthritis (PsA) to identify disease-specific signatures.

**Methods:** In this cross-sectional study, peripheral blood was collected from 21 SLE, 24 RA, 10 PsA patients, and 65 healthy controls (HC), with all patients meeting established classification criteria. Using multi-color flow cytometry, NK cell populations were identified and quantified, focusing on the distribution of $CD56^{bright\ or\ dim}$ subsets and key markers of activation (NKp44) and terminal maturation (CD57). Non-parametric statistical tests were employed for analysis.

**Results:** A universal and significant reduction in the percentage of total NK cells was observed across all patient cohorts compared to HCs ($p<0.01$), with the most substantial decrease in SLE patients. Notably, the SLE cohort displayed a unique phenotype characterized by significantly higher percentages of cytokine-producing $CD56^{bright}$ cells ($p=0.03$) and activated NKp44+ cells ($p=0.01$). This was paradoxically contrasted by a profoundly lower proportion of terminally differentiated, senescent-like CD57+ NK cells ($p<0.01$). In contrast, the NK cell profiles in RA and PsA patients were less dramatically altered.

**Conclusion:** Patients with SLE, RA, and PsA share the feature of reduced circulating NK cells. However, SLE is uniquely defined by a dysfunctional profile suggesting cellular senescence (low CD57+) concurrent with chronic, skewed activation (high $CD56^{bright}$/NKp44+). This signature points towards an intrinsic defect in NK cell homeostasis that may distinctly contribute to SLE pathogenesis and represents a promising disease-specific biomarker for further investigation.

**NK cells with its surface markers in different diseases (systemic lupus erythematosus, SLE; rheumatoid arthritis, RA; psoriatic arthritis, PsA) and Healthy controls (HC).**

| Mean ± Std. Deviation | HC | SLE | RA | PsA | p value[1] |
|---|---|---|---|---|---|
| **n** | 65 | 21 | 24 | 10 | ND |
| **NK cell number study** | | | | | |
| **Lym%** | 31.52±5.988 | 24.91±8.525 | 31.92±10.04 | 26.49±9.045 | **0.04*** |
| **Total events** | 49761±1219 | 37231±17816 | 42127±14867 | 49769±447 | 0.09 |
| **CD56 bright** | 4.492±2.387 | 11.72±9.361 | 5.6±3.987 | 4.31±1.752 | **0.03*** |
| **CD16-CD56+ Immature NK** | 3.512±2.269 | 11.93±11.71 | 7.675±18.53 | 2.61±1.175 | **<0.01*** |
| **CD16-CD56- Immature NK** | 0.58±0.541 | 0.925±0.679 | 4.162±16.40 | 0.42±0.297 | 0.06 |
| **CD16+CD56+ Mature NK** | 93.25±3.845 | 83.6±13.63 | 82.25±26.06 | 93.39±3.253 | **<0.01*** |
| **CD94/NKG2D** | 48.79±15.55 | 57.19±20.81 | 46.05±11.89 | 47.27±12.65 | 0.14 |

| | | | | | |
|---|---|---|---|---|---|
| **CD16+CD56-** | x | 1.2±NaN | 2.871±1.15 | 3.638±3.367 | ND |
| **NK cell activation study** | | | | | |
| **WBC counts** | 6.66±1.73 | 6.64±3.47 | 7.25±2.06 | 7.22±2.29 | 0.46 |
| **Lym%** | 31.44±6.016 | 24.91±8.525 | 31.92±10.04 | 26.49±9.045 | **0.04*** |
| **Total events** | 49362.7±2762.78 | 35400.8±17872.4 | 43232.8±13991.2 | 49498.4±1192.96 | 0.87 |
| **NK cells** | 22.89±9.682 | 16.55±15.03 | 18.00±9.22 | 19.21±7.425 | **<0.01*** |
| **NKp30+** | 56.22±15.67 | 53.53±20.88 | 58.05±20.35 | 47.74±18.13 | 0.35 |
| **NKp44+** | 0.568±0.424 | 1.1±1.101 | 1.17±3.16 | 0.41±0.608 | **0.01*** |
| **NKp46+** | 46.76±16.25 | 48.79±19.67 | 49.73±21.62 | 44.45±17.25 | 0.75 |
| **CD57+** | 57.09±12.25 | 41.28±24.35 | 50.05±16.80 | 53.55±17.01 | **<0.01*** |
| **CD4: CD8 ratio** | ND | 1.23±0.81 | 1.54±0.60 | 1.33±0.65 | 0.16 |

**Dunn's Post Hoc Comparisons of lym% in NK cell activation study**

| **Comparison** | **z** | **$W_i$** | **$W_j$** | p value[2] |
|---|---|---|---|---|
| **SLE : RA** | -2.241 | 35.794 | 57.021 | **0.025*** |
| **SLE : PsA** | -0.521 | 35.794 | 42 | 0.602 |
| **SLE : HC** | -2.529 | 35.794 | 56.904 | **0.011*** |
| **RA : PsA** | 1.336 | 57.021 | 42 | 0.182 |
| **RA : HC** | 0.016 | 57.021 | 56.904 | 0.987 |
| **PsA : HC** | -1.445 | 42 | 56.904 | 0.149 |

**Dunn's Post Hoc Comparisons of NK cells in NK cell activation study**

| **Comparison** | **z** | **$W_i$** | **$W_j$** | p value[2] |
|---|---|---|---|---|
| **SLE : RA** | -1.333 | 39.875 | 53.917 | 0.182 |
| **SLE : PsA** | -1.297 | 39.875 | 57.35 | 0.195 |
| **SLE : HC** | -3.35 | 39.875 | 69.621 | **< .001*** |
| **RA : PsA** | -0.262 | 53.917 | 57.35 | 0.793 |
| **RA : HC** | -1.894 | 53.917 | 69.621 | 0.058 |
| **PsA : HC** | -1.04 | 57.35 | 69.621 | 0.299 |

**Dunn's Post Hoc Comparisons of NKp44+ in NK cell activation study**

| **Comparison** | **z** | **$W_i$** | **$W_j$** | p value[2] |
|---|---|---|---|---|
| **SLE : RA** | 1.685 | 77.7 | 60.021 | 0.092 |
| **SLE : PsA** | 3.104 | 77.7 | 36.05 | 0.002 |
| **SLE : HC** | 2.096 | 77.7 | 59.167 | **0.036*** |
| **RA : PsA** | 1.838 | 60.021 | 36.05 | 0.066 |
| **RA : HC** | 0.103 | 60.021 | 59.167 | 0.918 |
| **PsA : HC** | -1.966 | 36.05 | 59.167 | **0.049*** |

**Dunn's Post Hoc Comparisons of CD57+ NK cells in NK cell activation study**

| **Comparison** | **z** | **$W_i$** | **$W_j$** | p value[2] |
|---|---|---|---|---|
| **SLE : RA** | -1.695 | 37.425 | 55.271 | 0.09 |
| **SLE : PsA** | -1.898 | 37.425 | 63 | 0.058 |
| **SLE : HC** | -3.558 | 37.425 | 69.015 | **< .001*** |
| **RA : PsA** | -0.59 | 55.271 | 63 | 0.555 |
| **RA : HC** | -1.658 | 55.271 | 69.015 | 0.097 |
| **PsA : HC** | -0.51 | 63 | 69.015 | 0.61 |

p value[1], Kruskal-Wallis test, * indicates p<0.05

p value[2], Dunn's Post Hoc Comparisons tests, * p < .05, p < .01, * p < .001

ND, not done

**Altered NK cell subsets and activation markers in autoimmune rheumatic diseases.**

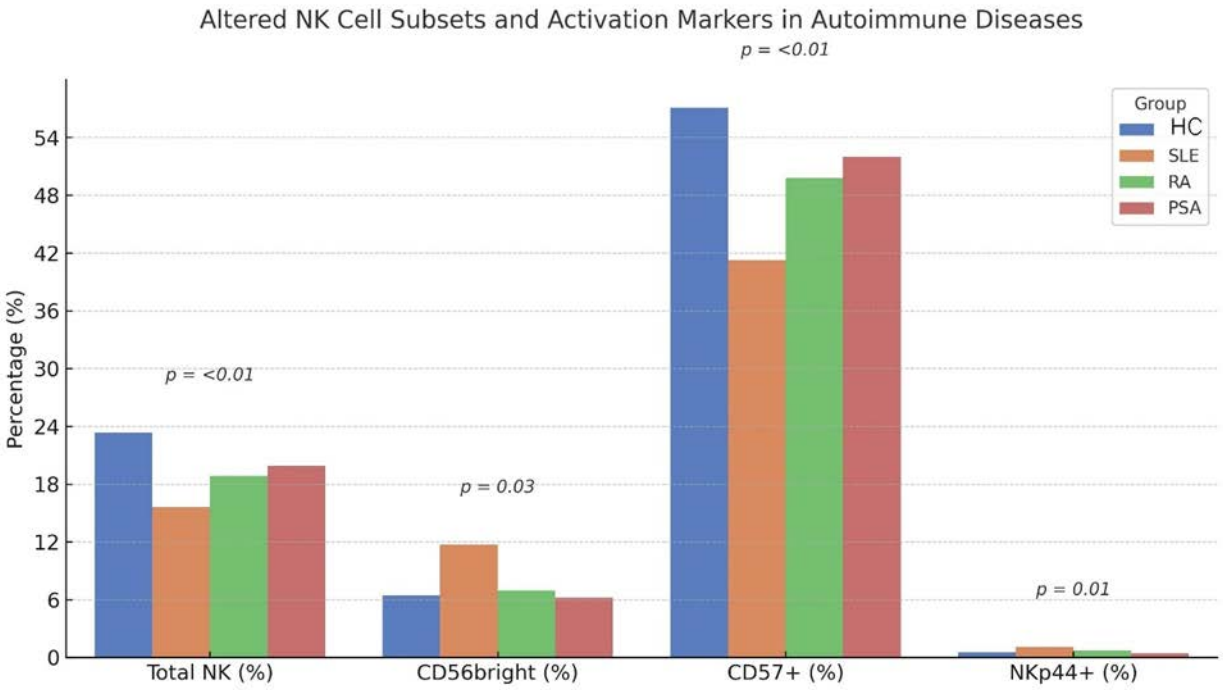


Figure 1. Bar plots comparing NK cell parameters among healthy controls (HC), systemic lupus erythematosus (SLE), rheumatoid arthritis (RA), and psoriatic arthritis (PSA). Displayed parameters include total NK cells, $CD56^{bright}$ NK cells, CD57+ NK cells, and NKp44+ NK cells. P-values, Kruskal-Wallis test.

**海報摘要 TCR51**

**“Effects of galactose related advanced glycation endproduct to immune system and inflammation response”**

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**半乳糖相關終期醣化產物對免疫系統及發炎反應影響**

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Introduction: Advanced glycation endproducts (AGEs), which formed via nonenzymatic glycation between reducing sugars and proteins, are implicated in the pathogenesis of diabetes mellitus, chronic inflammatory diseases, and age-related disorders. While glucose is the most studied element of AGEs, other mono-saccharide such as galactose also contribute to AGE formation and associated immune dysregulation. This study investigates the immunological effects galactose-derived AGE-modified human serum albumin (AGE-HSA) on immune cell responses.

Method: AGEs were synthesized by incubating HSA with galactose over 24 weeks. AGE formation was verified via fluorescence intensity, SDS-PAGE, and visual color change. Human immune cell lines, including Jurkat T cells and activated THP-1 macrophages, were exposed to 4 mg/mL of each AGE-HSA. Cell viability was measured using the WST-1 assay, and cytokine production was assessed post-stimulation. Signal pathway modulation was evaluated using specific inhibitors and western blot analysis of STATs and NF-κB pathways.

Results: We revealed that galactose-AGEs formed stronger fluorescence intensity, which suggest more deeply glycation. After stimulated with galactose-AGE, IL-2 production from Jurkat cells is lower comparing with glucose-derived AGEs and IL-8 production from activated THP-1 macrophages is higher than glucose-derived-AGE. These finding suggest a more prominent immune-modulatory effect of galactose-AGE. These findings support the hypothesis that non-glucose-derived AGEs, such as galactose-AGEs, may contribute to chronic inflammation and immune dysregulation. Further investigation into the underlying signaling pathways and in vivo relevance is warranted.

**海報摘要 TCR52**

**Elevated L5 in patients with antiphospholipid antibody syndrome**

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**抗磷脂抗體症候群患者的 L5 升高**

譚國棟, 林季千, 陳珠璜, 趙雅萱, 陳信華, 陳得源
台中榮總過敏免疫風濕科、中興大學生物醫學研究所、中國醫大附醫風濕免疫中心

**Objectives** L5 is the most negatively charged subfraction of low-density lipoprotein cholesterol (LDL-C) and strongly implicated in atherogenesis. However, circulating
L5 levels are not evaluated in patients with antiphospholipid antibody syndrome (APS).

**Method** We prospectively enrolled 28 APS patients, including 17 primary APS (pAPS) patients and 11 systemic lupus erythematosus (SLE) patients with antiphospholipid antibodies (aPL)/secondary APS, and 16 healthy subjects (HCs). The proportion of plasma L5 of LDL-C (L5%) were determined using anion-exchange purification with fast-protein liquid chromatography. We also measured markers of atherosclerosis, including carotid intima-media thickness (IMT) and brachial-ankle pulse wave velocity (baPWV), in comparison with a control group (59 individuals undergoing a health checkup examination). Multivariate logistic and linear regression were used to compare these groups of subjects.

**Results** After adjustments for age and sex in the multivariate linear regression, we observed an increase of L5% by 3.46% (95%CI: 1.88, 6.36) and of L5 level by 2.94 mg/dl (1.45, 5.93) in SLE patients with aPL/secondary APS and an increase of L5% by 2.29% (1.31, 4.01) in pAPS patients when compared with HCs. Besides, a higher proportion of pAPS patients had left carotid plaques when compared with the control group, with an odds ratio of 12.46 (95%CI: 2.60, 59.6). Bilateral IMT values were weakly correlated with the adjusted Global AntiPhospholipid Syndrome Score (aGPASS) scores.

**Conclusions** We found elevated plasma L5% in pAPS patients and SLE patients with aPL/secondary APS. The pathogenic role of L5 in atherosclerotic cardiovascular events in APS need to be explored.

**Table 1.** Multivariate analyses on the cardiovascular burden in study subjects.

| Multivariate analyses | SLE patients with aPL/APS | pAPS patients |
|---|---|---|
| **Linear regression, regression coefficient (95% confidence interval)** | | |
| Log (the percentage of L5)[a] | 1.24 (0.63, 1.85)** | 0.83 (0.27, 1.39)** |
| Log (the level of L5) | 1.08 (0.37, 1.78)** | 0.72 (0.05, 1.39)* |
| Log (left carotid IMT value)[b] | 0.06 (-0.07, 0.19) | 0.09 (-0.00, 0.18) |
| Log (right carotid IMT value)[b] | 0.14 (0.12, 0.27)* | 0.07 (-0.02, 0.17) |
| Log (left baPWV value) | 0.09 (-0.02, 0.19) | 0.00 (-0.07, 0.07) |
| Log (right baPWV value) | 0.06 (-0.05, 0.17) | 0.01 (-0.06, 0.08) |
| **Logistic regression, odds ratio (95% confidence interval)** | | |
| Left carotid plaque[c] | 2.90 (0.22, 38.2) | 12.46 (2.60, 59.6)** |
| Right carotid plaque[c] | 1.28 (0.11, 14.7) | 1.00 (0.24, 4.22) |

[a]The 16 healthy subjects as the reference group.
[b]The control group (n=59) as the reference group.
aPL, antiphospholipid antibodies; baPWV, brachial-ankle pulse wave velocity; IMT, intima-media thickness; pAPS, primary antiphospholipid antibody syndrome; SLE, systemic lupus erythematosus.*p<0.05; **p<0.01.
†p values of < 0.01 were regarded as statistically significant for multiple comparisons between SLE, pAPS patients and the controls in regards to the percentage and level of L5.

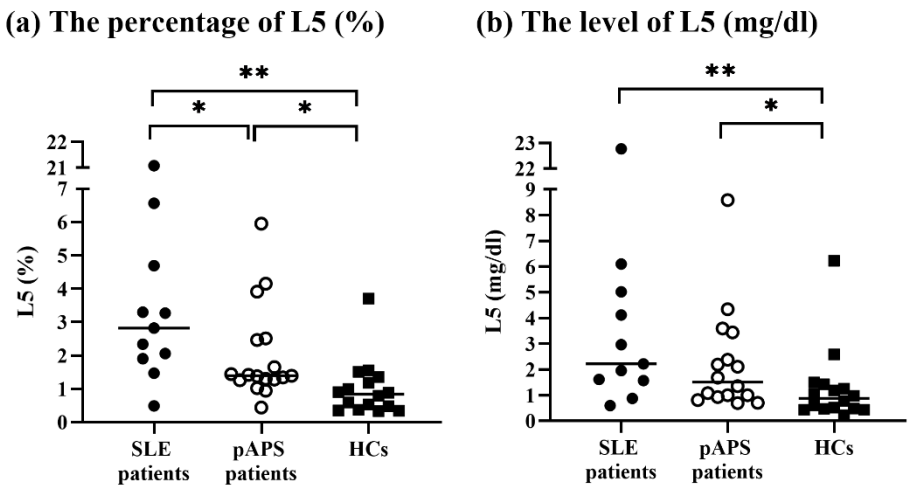


**Fig. 1** The (a) percentage and (b) level of L5 in SLE patients with aPL/APS and pAPS patients when compared with the healthy controls (HCs).
aPL, antiphospholipid antibody; pAPS, primary antiphospholipid antibody syndrome; SLE, systemic lupus erythematosus. $^{*}p<0.05$; $^{**}p<0.01$.

**海報摘要 TCR53**

**Risk of Allergic Diseases Following SARS-CoV-2 Infection in Children: A Population-Based Cohort Study**

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Abstract:
BACKGROUND:
Allergic diseases are common chronic conditions characterized by persistent inflammation and dysregulated immune responses. Coronavirus disease 2019 (COVID-19) is associated with a profound cytokine storm, marked by excessive activation of innate immunity and subsequent attenuation of type 1 immune responses. However, the relationship between COVID-19 and the subsequent development of allergic diseases remains incompletely understood.

METHOD:
We conducted a retrospective cohort study using the TriNetX Research Network, which provides access to real-world clinical data from multiple healthcare organizations across the United States. We identified two cohorts of patients under 18 years of age: one cohort with a positive SARS-CoV-2 PCR test and a propensity score–matched control cohort with negative test results. The primary outcome was the cumulative incidence of allergic diseases following COVID-19 infection, assessed using Kaplan–Meier survival analysis.

RESULTS:
The risk of incident allergic diseases was significantly higher in the COVID-19 cohort than in the control cohort. The hazard ratio (HR) for any allergic disease was 1.19 (95% confidence interval [CI], 1.17 to 1.21). The risk of asthma was increased (HR, 1.14; 95% CI, 1.12 to 1.17), as was the risk of allergic rhinitis (HR, 1.22; 95% CI, 1.20 to 1.25) and atopic dermatitis (HR, 1.25; 95% CI, 1.22 to 1.29).

CONCLUSION:
These findings suggest that SARS-CoV-2 infection is associated with an elevated risk of subsequent allergic diseases, including asthma, allergic rhinitis, and atopic dermatitis, in the pediatric population.
Keywords: Atopic dermatitis, Allergic rhinitis, Asthma, Allergic diseases, COVID-19

**海報摘要 TCR54**

**Artificial Intelligence in Stem Cell Therapy**

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**人工智慧應用於幹細胞治療**

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[2] 國防醫學院三軍總醫院

Artificial Intelligence (AI) is transforming rheumatology, with a myriad of studies aiming to improve diagnosis, prognosis, and treatment prediction, while also showing capacity to optimize the workflow, improve drug discovery and clinical trials.

Cell therapy is a promising field in regenerative medicine that involves the use of living cells to replace or repair damaged or diseased tissues and organs. One of the most promising areas of cell therapy is the use of stem cells.

Previously, we have demonstrated that TNF-α inhibitor reverse the effects of human umbilical cord-derived stem cells on experimental arthritis by increasing immunosuppression (1). Furthermore, CD146+ mesenchymal stem cells (MSC) display greater therapeutic potential than CD146- cells for treating collagen-induced arthritis in mice (2). In addition, our investigations demonstrated that the fetal MSC protected the CIA mice from cartilage damage and triggered an immunosuppressive response in them by increasing the number of CD14+ IL10+ cells (3).

However, it still faces significant challenges in identifying suitable cells, ensuring their safety, and optimizing their effectiveness.

These are where AI comes in. AI has the potential to revolutionize SC therapy by analyzing vast amounts of data (patient's genetic information and medical history), understanding the behavior of SCs, recognizing individual cell type before undergoing differentiation, characterization of SCs using mathematical models and prediction of mortality risk associated with SC transplantation.

One of the best benefits of using AI in SC therapy is its ability to help identify the best cells for a particular patient. This allows us to see the dawn in regenerative medicine.

References:

1. Deh-Ming Chang et al. Cell Immunol. 2012.
2. Deh-Ming Chang et al. Stem Cell Res Ther. 2016.
3. Deh-Ming Chang et al. Int J of Rheum Dis 2024.

**海報摘要 TCR55**

**Prevalence of Stroke, Heart Failure, and Myocardial Infarction Across ANA Patterns Compared to ANA-Negative Controls in Non-Autoimmune Individuals**

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**與非自體免疫疾病個體中抗核抗體陰性對照者相比，中風、心臟衰竭和心肌梗塞的不同抗核抗體模式盛行率**

林科名 [1], 詹天明 [2]

嘉義長庚紀念醫院風濕過敏免疫科

林口長庚紀念醫院風濕過敏免疫科

**Background:** The clinical relevance of antinuclear antibodies (ANAs) beyond autoimmune disease remains poorly defined. We evaluated the association between specific ANA patterns and the prevalence of major cardiovascular events, including heart failure, myocardial infarction, and stroke, in individuals without a diagnosed autoimmune condition.

**Methods:** We conducted a cross-sectional analysis of 38,572 patients from the Chang Gung Research Database who underwent ANA testing from 2019 to 2021. ANA patterns were classified via indirect immunofluorescence according to the International Consensus on ANA Patterns criteria. After excluding patients with diagnosed AID and those with inconsistent or mixed patterns, 29,295 individuals (26,124 ANA-negative, 3,171 ANA-positive) were included. CVD outcomes were identified using International Classification of Diseases codes.

**Results:** In the non-AID cohort, specific ANA patterns were linked to a higher prevalence of CVD compared to the ANA-negative group. The prevalence of heart failure was significantly higher in patients with Centromere (AC-3; OR 1.66, p=0.0369) and anti-mitochondrial antibody (AMA, AC-21; OR 2.04, p=0.0212) patterns. Conversely, no ANA pattern showed a significant association with the prevalence of myocardial infarction (p=0.9545). However, the risk of stroke was significantly elevated for multiple patterns, including Nuclear envelope (AC-11/12; OR 4.08, p=0.0058), AMA (AC-21; OR 2.27, p=0.0074), Golgi (AC-22; OR 4.63, p=0.0365), and Nucleolar (AC-10; OR 2.62, p=0.0353).

**Conclusion:** Even without clinical autoimmune disease, specific ANA patterns are independently associated with a higher prevalence of heart failure and stroke. These antibodies may be important subclinical biomarkers for identifying individuals at increased risk for cardiovascular events.

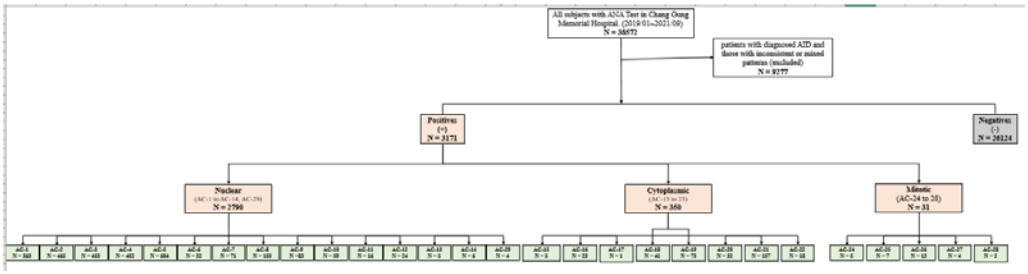


**Figure 1**. Flow chart of patient selection process

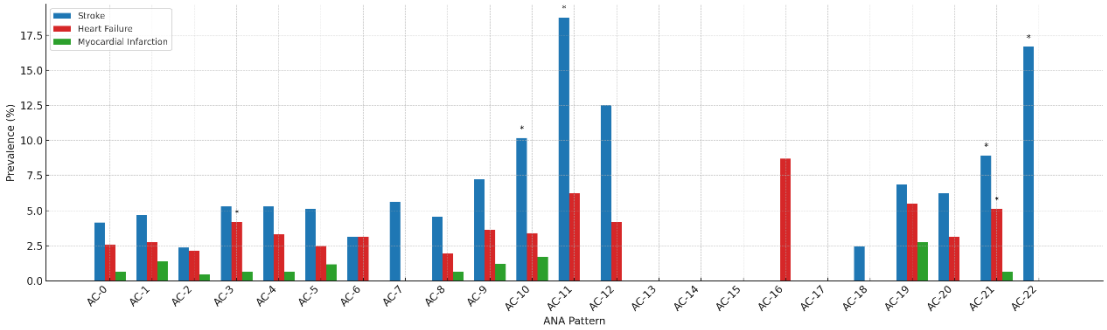


**Figure 2.** Prevalence of stroke, heart failure, and myocardial infarction across ANA staining patterns (AC-1 to AC-29) compared to ANA-negative controls (AC-0), in patients without autoimmune disease. Data represent the percentage prevalence of each cardiovascular outcome (stroke in blue, heart failure in red, myocardial infarction in green) by ANA pattern. Each bar corresponds to one ANA pattern as classified by the International Consensus on ANA Patterns (ICAP). The ANA-negative group (AC-0) serves as the reference for statistical comparisons. Asterisks (*) indicate statistically significant differences ($p < 0.05$) compared to AC-0, determined using Fisher's exact test.

**海報摘要 TCR56**

# First Clinical Report of Anti-RuvBL1/2 Autoantibody-positive Patients in Taiwan
# 台灣 Anti-RuvBL1/2 自體抗體陽性病患臨床報告

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5. 中國醫藥大學醫學系

**Background:**
The RuvBL1/2 complex plays key roles in chromatin remodeling, transcriptional regulation, DNA repair, and cell cycle control. Its dysregulation contributes to genomic instability and disrupted protein homeostasis, which have been implicated in malignancies, neurodegenerative disorders, and autoimmune diseases. Anti-RuvBL1/2 autoantibodies have recently been associated with scleromyositis, an autoimmune entity overlapping features of systemic sclerosis (SSc) and idiopathic inflammatory myopathy (IIM).

**Methods:**
We conducted a retrospective analysis of five Taiwanese patients positive for anti-RuvBL1/2 autoantibodies, confirmed by mass spectrometry, immunoblotting, and immunoprecipitation.

**Results:**
This is the first reported anti-RuvBL1/2-positive cohort in Taiwan. Four of the five patients were female (median age 56), differing from previous male-predominant reports. All had limited cutaneous SSc with puffy fingers and sclerodactyly. Raynaud's phenomenon occurred in three patients; one reported sicca symptoms. Four had gastrointestinal involvement. Two patients had mild myositis with CK levels ranging from 116 to 12,954 IU/L. ILD was found in all patients (four fNSIP, one CPFE), with reduced DLCO and FVC. ANA was positive in all, showing nuclear speckled pattern (AC-4).

**Conclusion:**
Anti-RuvBL1/2 autoantibody-positive patients in this Taiwanese cohort displayed a consistent clinical phenotype of limited cutaneous SSc with frequent ILD involvement and overlapping myositis. Unlike other SSc subsets, severe vasculopathy or major internal organ crises were absent. These findings support the existence of a distinct clinical subset within the scleromyositis spectrum, meriting further investigation into its pathogenic mechanisms and clinical implications.

海報摘要 TCR57

# Decoding the AC-4 ANA Pattern: A Distinct Serological Signal for Autoimmunity Risk Stratification

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# 解碼 AC-4 ANA 型態：自體免疫風險分層的獨特血清學訊號

詹天明[1]* 蕭朝陽[1], 黃思偉[1], 尤瀚華[1], 謝孟儒[1], 蔡昀臻[1], 李蒔青[2], 楊曜嘉[2],趙祥元[2], 許鐘元[3], 蘇昱日[3], 林科名[4], 林靖麒[4], 羅緯麟[5], 羅淑芬[1], 余光輝[1]

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## Abstract

### Background:

Antinuclear antibody (ANA) testing via indirect immunofluorescence (IIF) is a cornerstone in the diagnostic evaluation of autoimmune diseases (AIDs). Among the nuclear patterns, the fine speckled pattern (AC-4) has gained attention for its possible disease associations, yet its clinical implications compared to ANA-negative or other ANA-positive subtypes remain incompletely defined.

### Methods:

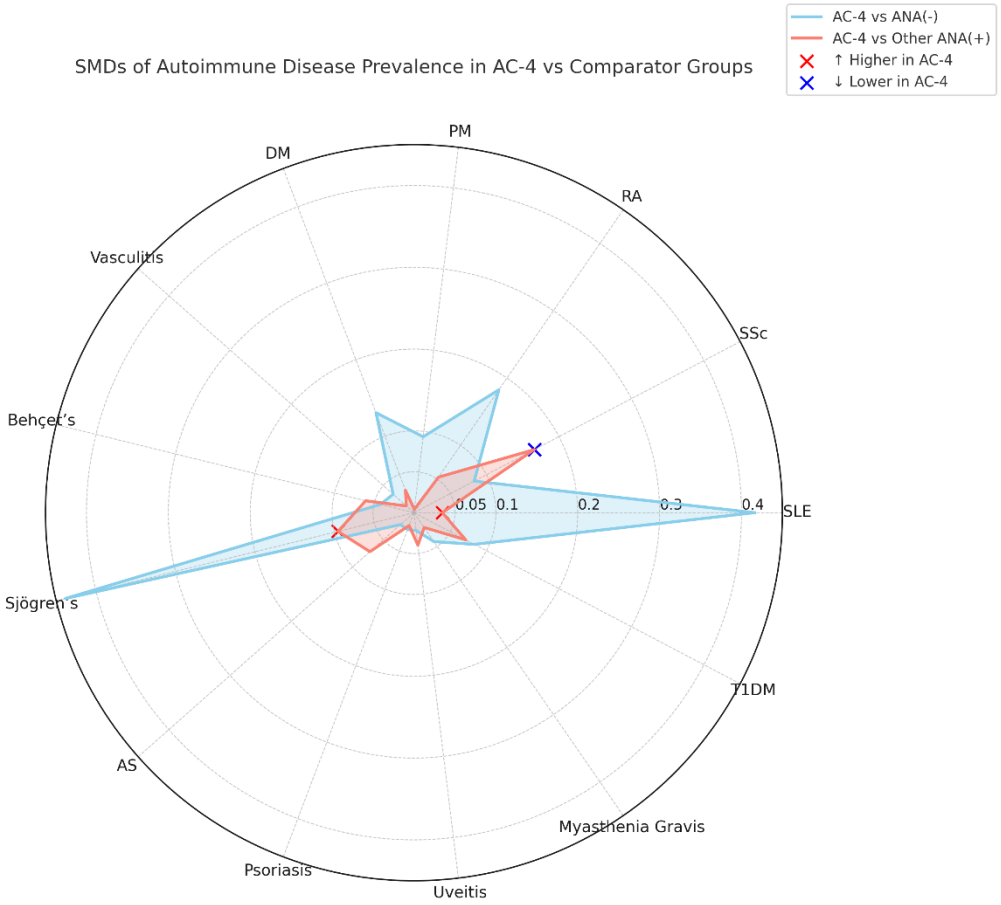
We conducted a retrospective cross-sectional study using the Chang Gung Research Database (CGRD), including 35,763 individuals who underwent ANA testing from January 2019 to September 2021. Subjects were categorized as AC-4 (n=670), ANA-negative (n=31,143), or other ANA-positive (n=3,950). Diagnoses of AIDs were confirmed through ICD-10 coding and Taiwan's catastrophic illness registry. Propensity score matching (1:5) was applied to adjust for confounders. Standardized mean differences (SMDs) were used to evaluate disease burden differences.

### Results:

Following PSM, the AC-4 group showed a significantly higher overall prevalence of autoimmune diseases than the ANA-negative group (32.54% vs. 16.12%, SMD=0.39). The most prominent differences were observed in Sjögren's syndrome (12.24% vs. 1.62%, SMD=0.43), systemic lupus erythematosus (8.66% vs. 0.52%, SMD=0.42), and rheumatoid arthritis (9.85% vs. 4.62%, SMD=0.20). When compared to other ANA-positive patients, most autoimmune conditions showed similar prevalence, except for systemic sclerosis, which was significantly less frequent in the AC-4 group (0.52% vs. 2.56%, SMD=0.17).

### Conclusion:

The pure AC-4 ANA pattern is associated with a distinctly elevated risk of specific autoimmune diseases, especially Sjögren's syndrome, SLE, and rheumatoid arthritis, compared to ANA-negative individuals. Interestingly, AC-4 appears to be inversely associated with systemic sclerosis relative to other ANA-positive patterns.




**SMDs of Autoimmune Disease Prevalence in AC-4 vs Comparator Groups**
Standardized mean differences comparing AC-4 with ANA-negative (blue) and other ANA-positive (red) patients after propensity score matching. Red markers indicate higher prevalence in AC-4 (e.g., SLE, Sjögren's syndrome), while the blue marker indicates lower prevalence (e.g., SSc). SMD > 0.1 denotes a meaningful difference.