The Clinical Significance of Rare Autoantibodies in the Prognosis of Systemic Lupus Erythematosus and Systemic Sclerosis

Ph.D. thesis

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1. INTRODUCTION

In systemic autoimmune diseases such as systemic lupus erythematosus (SLE) and systemic sclerosis (SSc), we often face symptoms that significantly affect both life expectancy and quality of life. Early recognition of specific organ manifestations is essential for timely therapy initiation and for the prevention of later complications. Due to the diverse organ involvement, comprehensive evaluation frequently includes invasive, time-consuming, burdensome, and costly procedures or interventions. Non-invasive risk assessment based on autoantibody profiles can help determine the necessity and frequency of further diagnostic evaluations.

Immunoserological results can provide substantial support in assessing disease activity and risk, and are also highly useful in differential diagnostics. Considering that autoimmunity often presents as a spectrum, atypical cases and overlap syndromes — in which multiple systemic autoimmune diseases coexist — may also occur. Autoantibodies play a crucial role in identifying such conditions, although a complete clinical assessment remains indispensable. Analyzing the association between autoantibodies and clinical manifestations, and applying the resulting conclusions in clinical practice, holds significant value. An increasing amount of data is being gathered about rare autoantibodies, which may further refine our understanding of disease prognosis and offer predictive insights into treatment effectiveness.

2. AIMS

The aim of this study was to investigate the prognostic role of rare autoantibodies in patients with systemic lupus erythematosus (SLE) and systemic sclerosis (SSc) under our care.

I. To determine the prevalence of scleroderma-specific anti-topoisomerase I (anti-topo I) and anticentromere antibodies (ACA) among patients with SLE, and to analyze the clinical characteristics of this patient subgroup (e.g. differences in organ manifestations, variation in disease onset).

II. To determine the prevalence of anti-cyclic citrullinated peptide (anti-CCP) antibodies — typically associated with rheumatoid arthritis (RA) — in the SLE patient cohort, and to

analyze their association with joint involvement. Additionally, we aimed to assess the diagnostic utility of anti-CCP antibodies in an unselected lupus population, including both patients with pure SLE and those with overlapping syndromes.

III. To analyze the association between SLE and neuromyelitis optica (NMO), with a focus on determining the co-prevalence of the two conditions. We also aimed to investigate the predictive role of autoantibodies in forecasting fluctuations in clinical disease activity. We hypothesized that B-cell dysfunction may lead to the production of potentially pathogenic autoantibodies in SLE-NMO cases, and that polyclonal activation may be associated with NMO relapses occurring in the context of SLE. To this end, our further objectives included:

- Investigating the correlation between AQP4 antibodies and SLE-specific autoantibodies.
- Measuring serum levels of cytokines and chemokines that reflect T-cell function.
- Analyzing the longitudinal relationship between NMO activity and autoantibody profiles.
- Identifying occult NMO cases among our SLE patient population.

IV. To examine the specificity of rarely occurring autoantibodies in systemic sclerosis and their association with organ manifestations.

- We aimed to determine the prevalence of rare autoantibodies.
- We sought to analyze the co-occurrence patterns of rare and classical autoantibodies, and to assess the overlap frequency of rare autoantibodies.
- Our goal was to evaluate the role of rare autoantibodies in association with specific organ manifestations.

3. PATIENTS AND METHODS

3.1. Investigation of Rare Autoantibodies in SLE

3.1.1. Association between SLE and anti-topoisomerase I, anti-centromere antibodies

We retrospectively analyzed data from patients treated for SLE between 2002 and 2012 at the Department of Rheumatology and Immunology, University of Pécs (PTE), to assess the presence of rare autoantibodies. The diagnosis of SLE was based on the decision of a specialist. We screened for the presence of scleroderma-specific anti-topoisomerase I (anti-topo I) and anti-centromere antibodies (ACA) using routinely performed ELISA tests (Hycor).

3.1.2. Investigation of Anti-CCP Antibodies in SLE

We conducted a cross-sectional analysis of 382 patients with SLE who had attended our clinic at least twice in 2020. The presence of anti-CCP antibodies was investigated, and overlapping syndromes were identified based on data recorded in patient documentation.

3.1.3. Investigation of AQP4 in SLE

As part of a multicenter study, we examined a total of 19 serum samples from 6 individuals diagnosed with both SLE and neuromyelitis optica (NMO). In addition, we analyzed serum from 11 healthy controls.

3.2. Systemic Sclerosis – Rare Autoantibodies

In our prospective study, 160 patients with systemic sclerosis under care at the Department of Rheumatology and Immunology, University of Pécs, were enrolled. Alongside recording epidemiological data, we documented clinical manifestations involving the skin, gastrointestinal tract, lungs, and cardiovascular system. We also analyzed the results of extended autoantibody testing, including NOR90, Th/T0, fibrillarin (U3RNP), Ku, and PmScl antibodies.

4. RESULTS

4.1.1 Clinical characteristics observed in SLE patients with anti-topoisomerase I and anti-centromere positivity

Anti-topo-I testing was performed in 343 patients, of which 10 tested positive, and 90.9% of them were female. No significant differences were observed in the distribution of organ involvement within this patient group.

Of the 222 patients tested for anti-centromere, 7 had positive results, all of whom were women. The average age at disease onset was 50 years, which showed a significant difference compared to the ACA-negative patient group (34.5 years, $p = 0.006$). No significant differences were observed regarding organ manifestations.

4.1.2 Analysis of the relationship between SLE and anti-CCP antibodies

Among the patients where joint involvement was suspected (331 patients), RF positivity was found in 48% (159/331), CCP positivity in 6.3% (21/331), and both antibodies were positive in 4.8% (16/331). In one-third of the patients studied, 138 (36%) had some form of overlap syndrome based on the clinician's assessment, and among these, 23 were diagnosed with coexisting RA.

The group of patients with overlap syndromes showed only a trend toward a higher frequency of anti-CCP positivity compared to patients with pure SLE (overlap: 11/138 patients [9%] vs. pure SLE: 10/244 patients [4%]). However, the less RA-specific RF was significantly more common among overlap patients than in those with pure SLE (Table 7).

When comparing pure SLE patients with those whose clinicians also suspected RA, the anti-CCP positivity rate differed significantly — this antibody was significantly more frequent among SLE-RA overlap patients compared to pure SLE patients (SLE-RA overlap: 9/23 patients [39.1%] vs. pure SLE: 9/244 patients [3.7%], $p < 0.0001$).

Among SLE-RA patients, anti-CCP positivity was also significantly more frequent compared to overlap patients whose lupus was not associated with RA (SLE-RA: 9/23 [39.1%] vs. SLE-other overlap: 2/115 [2%], $p < 0.0001$).

4.1.3 Results in patients with coexisting SLE and NMO

In all 6 SLE patients included in the study, the first symptoms of NMO/NMOSD appeared after the diagnosis of SLE.

4.1.3.1 Analysis of anti-AQP4 and anti-MOG antibodies

We examined 19 serum samples: 4 during NMO relapse and 15 following remission. AQP4-IgG1 antibodies were more frequently detected than IgM antibodies: IgM was present in 47% of samples, while IgG1 was found in 79%.

We also tested for the presence of anti-MOG IgG and IgM antibodies in the serum, none of which were positive in any of the samples.

Next, we assessed the relationship between anti-AQP4 serostatus and NMO and SLE activity. Although anti-AQP4 antibodies were detectable during remission, AQP4 seronegativity was observed only during NMO remission. However, in some cases, both IgG1 and IgM AQP4 antibodies were present during NMO remission.

We analyzed six samples taken during active SLE phases, three of which coincided with NMO relapses. Anti-AQP4 antibodies were present in all six samples, regardless of NMO relapse status.

4.1.3.2 Correlation of AQP4-IgG1 and IgM with SLE-associated antibodies in SLE-NMO patients

A significant correlation was found between AQP4-IgG1 and IgM antibodies ($p = 0.02$). Significant correlations were observed between AQP4-IgG1 and anti-nucleosome antibodies ($p = 0.04$), AQP4-IgM and anti-nucleosome antibodies ($p = 0.001$), as well as between AQP4-IgM and ANA ($p = 0.009$).

SLE-associated antibodies also showed correlations with each other: ANA and anti-dsDNA ($p = 0.01$), ANA and anti-nucleosome ($p < 0.001$), and anti-dsDNA and anti-nucleosome antibodies ($p = 0.001$).

4.1.3.3 Longitudinal analysis of autoantibodies in NMO/SLE patients

Three serum samples were available from the period prior to the first NMO attack, taken 1, 2, and 5 years before the initial relapse, respectively. All three samples tested positive for anti-AQP4 IgG1, and one also contained AQP4-IgM antibodies.

We also examined the relationship between NMO relapses and changes in anti-AQP4 antibody levels. AQP4-IgG1 was already detectable before the first relapse, and its levels further increased during relapse. AQP4-IgM was not detected in samples taken before the relapses, but IgM antibodies appeared during relapse. Both IgG1 and IgM antibody levels decreased during remission.

Subsequently, we analyzed SLE-associated antibodies in the same samples to determine

whether changes in their titers were associated with NMO relapses. ANA, anti-dsDNA, and anti-nucleosome antibody levels were elevated in all samples associated with NMO relapses.

4.1.3.4 Correlation between cytokine and chemokine concentrations and AQP4-IgG1/IgM titers in the serum of SLE/NMO patients

We analyzed the concentrations of cytokines and chemokines in serum samples that may be associated with NMO, and compared these levels to the AQP4-IgG1 and IgM titers.
Both AQP4-IgG1 and AQP4-IgM titers showed correlation with serum IFN-γ levels (R= 0.446, $p = 0.02$ and $p = 0.01$, respectively). AQP4-IgM levels also correlated with IL-17 ($p = 0.01$) and CXCL10/IP-10 (C-X-C motif chemokine ligand 10, also known as Interferon gamma-induced protein 10) levels (R= 0.460, $p = 0.03$).
We further examined the correlation between CXCL10/IP-10 and IFN-γ levels, which also proved to be significant (R= 0.448, $p = 0.05$). CCL11 (eotaxin) and CCL17 (TARC) did not show a correlation with AQP4 antibody levels in serum.

4.1.3.5 Longitudinal analysis of cytokines and chemokines in the serum of SLE/NMO patients

We assessed the longitudinal changes of IFN-γ, IL-17, CXCL10, and AQP4 antibody concentrations in relation to NMO clinical activity in SLE patients. In Patient 1, IFN-γ and IL-17 levels increased during an NMO relapse. Similar cytokine elevations were observed in two other relapses (Patients 3 and 4), followed by a decline in levels afterward.
CXCL10 (IP-10) concentrations closely followed NMO clinical activity and changes in antibody levels: its levels increased during relapse and decreased in serum collected during remission. CCL11 (eotaxin) and CCL17 (TARC) concentrations did not reflect NMO clinical activity, remaining relatively stable in peripheral blood.

4.2 Results of rare autoantibody testing in systemic sclerosis

Half of the examined patients (n = 81) tested negative for both ACA and anti-Topo-I antibodies. Among them, anti-RNA polymerase III positivity was observed in 16 cases.
In 65 patients (40%), none of the three most common SSc-specific autoantibodies were present. Among them, 11 patients were positive for PmScl antibodies.
Other rare autoantibodies were detected in 1–2 cases, often showing overlap with other diseases. Both ANA and SSc-specific autoantibodies were negative in 29 patients (18%). Among these, 2 tested positive for Ro-52, 9 for RF, and 3 for anti-CCP.

In our cohort, the frequency of rare autoantibodies was low, with positive blot results in 2–12% of cases.

In patients positive for anti-PmScl (n = 17, 10.6%), the diffuse form of the disease predominated in ¾ of the cases. No meaningful differences were observed regarding interstitial lung disease or cardiac involvement. The only noticeable trend was found in gastrointestinal involvement: anti-PmScl–positive patients showed fewer GI abnormalities according to the UCLA-GIT2 score.

Among anti-Ku–positive patients (n = 6, 3.7%), the ratio of limited to diffuse form was equal, and all affected were women. Interstitial lung disease was seen in 5 of 6 patients, and cardiac involvement in 4. Telangiectasia was significantly less frequent in anti-Ku–positive patients compared to those who were anti-Ku negative (anti-Ku+: 1/6 vs. 92/149, $p = 0.0380$).

In both patients with anti-U3RNP/fibrillarin positivity, gastrointestinal symptoms were observed (2/2 patient vs. 41/151 patient, $p = 0.0222$). Interstitial lung disease was also present, but no immunomodulatory treatment was administered in these cases.

Among anti-Th/To–positive patients (N = 6, 3.7%), the diffuse form was more prevalent (dcSSc: 5/lcSSc: 2). Joint contractures, interstitial lung disease, and cardiac abnormalities were more common, although not reaching statistical significance.

All 4 patients with anti-NOR90 positivity had the diffuse form (4/4 vs. 69/151, $p = 0.0471$), with a balanced male-to-female ratio. Gastrointestinal abnormalities were significantly more frequent in this group compared to anti-NOR90–negative patients (anti-NOR90+: 3/4 vs. 31/150, $p = 0.0339$).

5. DISCUSSION

5.1 Rare Autoantibodies in SLE

5.1.1 Prevalence of Anti-Topoisomerase I and Anti-Centromere Antibodies in SLE

In our SLE patient cohort, anti-topoisomerase I (anti-Topo I) antibody positivity was observed in 2.9% of patients, consistent with previously reported rates in the literature (4.1% – [50], 2.7% – [111]). No significant differences were found in organ involvement in the anti-centromere antibody (ACA)-positive subgroup; however, the average age at disease onset was significantly higher (50 years) compared to ACA-negative patients (34,5 years). This is consistent with other published data (ACA+: 47.5 years).

Despite the presence of scleroderma-specific autoantibodies, clinical signs of systemic sclerosis (SSc) were not observed in our patients, except in one case. A similar study involving a comparable SLE cohort (n=560) reported 11 ACA-positive patients, none of whom exhibited scleroderma manifestations. These findings further support the view that ACA positivity observed in SLE — in the absence of supporting clinical evidence — does not necessarily imply coexisting SSc, but may instead define a distinct lupus subphenotype.

5.1.2 Prevalence of Anti-CCP Antibody Positivity in SLE

Arthritis is a common clinical manifestation in SLE, observed in 331 out of 382 patients (86.6%) in our study. Although imaging findings were not analyzed, our serological data indicate that anti-CCP positivity, a marker considered specific for RA, did not significantly differentiate between pure SLE patients and those with overlap syndromes. However, the frequency of anti-CCP positivity was significantly higher among patients with SLE-RA overlap compared to those with overlap of SLE and other systemic autoimmune diseases. Therefore, although anti-CCP alone is not sufficient to distinguish SLE from SLE-RA, its presence can support a concurrent RA diagnosis in clinically suspected overlap cases.

5.1.3 Presence of Aquaporin-4 Autoantibodies in SLE

The precise prevalence of coexisting SLE and neuromyelitis optica (NMO) remains unclear, but is estimated at approximately 0.1%. In our cohort of 443 SLE patients, 4 cases (0.9%) met criteria for NMO. In 5 of these cases, SLE onset preceded NMO development, consistent with previous reports suggesting earlier onset and higher activity of SLE compared to NMO.

In this study, we examined AQP4-IgG1 and AQP4-IgM autoantibodies. While AQP4-IgG1 antibodies were present years before the first NMO-SD symptoms, AQP4-IgM antibodies

were not detectable prior to disease onset. The highest levels of AQP4-IgM were measured during active disease. Recent literature also supports that AQP4-IgM is associated with relapse phases.

We assessed the presence of various SLE-associated autoantibodies at the time of NMO symptom onset to investigate potential polyclonal B-cell activation. A positive correlation was found between AQP4-IgG and AQP4-IgM titers and anti-nucleosome antibodies, and between AQP4-IgM and ANA titers. These associations were also evident in longitudinal patterns corresponding to NMO clinical activity. The longitudinal behavior of anti-nuclear, anti-dsDNA, and anti-nucleosome antibodies mirrored that of AQP4-IgG1 during changes in disease activity. The correlation between SLE- and NMO-related antibody titers and their similar temporal patterns suggests that polyclonal B-cell activation may drive increased AQP4 antibody production in SLE-associated NMO.

To investigate the role of T-cell activation in clinical disease expression, we performed a longitudinal analysis of cytokine and chemokine levels in serum. Both AQP4-IgG and AQP4-IgM titers showed significant positive correlation with serum IFN-γ levels. AQP4-IgM also correlated with IL-17 concentrations, indicating that T-cell activation likely plays a key role in disease pathogenesis.

We also measured the longitudinal changes in selected chemokines. CXCL10 (IP-10) levels closely reflected NMO clinical activity and antibody titers: concentrations increased during relapse and decreased in remission. IP-10 levels also correlated with AQP4-IgM and IFN-γ, and may play an important role in SLE pathogenesis.

In addition to this Th1-associated chemokine, we also assessed two other Th2-related chemokines: CCL11 (eotaxin) and CCL17 (TARC). In contrast to IP-10, and similarly to eotaxin, TARC levels did not correlate with autoantibody titers and remained stable over time.

5.2. Rare Autoantibodies in Systemic Sclerosis

In our study, nearly half of the patients followed for systemic sclerosis (SSc) (70 out of 160) tested negative for both ACA and topo I antibodies. Among these patients, anti-RNA Pol III positivity was detected in 16 cases (10%), a proportion consistent with the findings of previously published data. The three most common SSc-specific autoantibodies proved negative in 65 cases, PmScl positivity was observed in 10, and other rare autoantibodies (anti-Ku, anti-U3RNP, anti-Th/To, anti-NOR90) were detected in 6 additional cases, aiding in the diagnostic process. In 29 cases (18%), both ANA and SSc-specific autoantibodies were negative. Among these, Ro-52 positivity was found in 2 patients, RF in 9, and anti-CCP in 3 of them. A German study found that in 10.5% of SSc cases (39/372), no SSc-specific autoantibodies were detectable, and in about one-third of these, ANA was also negative. Notably, significant overlap was observed among the rare autoantibodies, particularly in anti-PmScl–positive cases, where multiple antibody positivity occurred. This is important because, as also supported by our data, the three major, traditional SSc-specific autoantibodies rarely overlap within a single patient. In contrast, rare autoantibodies often do, a finding that has also been reported by other centers and can now be confirmed in our Hungarian patient population.

Despite this, rare autoantibodies were detectable only in a small proportion (2–12%) of our SSc cohort, thus associations with specific clinical manifestations should be interpreted with caution — an observation that aligns with current literature. A notable exception may be anti-PmScl, which showed the highest prevalence (12%) among our patients. It appeared to have a protective effect against gastrointestinal (GI) involvement, although this did not reach statistical significance, likely due to limited sample size. This observation is consistent with previous reports indicating lower esophageal involvement in anti-PmScl–positive patients. All four anti-NOR90–positive cases presented with diffuse cutaneous involvement and a balanced male-to-female ratio. Severe GI manifestations were noted in three cases, a finding that contrasts with some more recent, higher-sample studies.

6. Summary

In addition to well-established disease-specific autoantibodies, testing for rarer or less specific antibodies such as anti-centromere, anti-topoisomerase I, anti-CCP, and anti-AQP4 may be warranted in systemic lupus erythematosus (SLE). Based on our findings, the presence of SSc-specific autoantibodies alone does not necessarily indicate an overlap with systemic sclerosis; rather, they may define a distinct lupus subset. ACA-positive patients were found to be older, and the apparently milder clinical manifestations may not, for this reason alone, represent a distinct subgroup.

Based on our results, anti-CCP positivity by itself is insufficient to confirm overlap with rheumatoid arthritis (RA), but when accompanied by supportive clinical features, the likelihood of an SLE-RA overlap increases significantly. In fact, anti-CCP positivity can distinguish SLE-RA overlap patients from those with other SLE overlap syndromes.

The co-occurrence of SLE and neuromyelitis optica (NMO) was nearly 9 times more frequent in our cohort than previously estimated in the literature, with a prevalence of 0.9%. Therefore, testing for AQP4 antibodies should definitely be considered in SLE patients presenting with neurological symptoms. Investigation of AQP4 antibody serotypes and changes in antibody titers may contribute to a better understanding of NMO onset.

Based on our investigations in systemic sclerosis (SSc), we can state that rare scleroderma-specific autoantibodies show considerable overlap with each other — whereas among the classical SSc-specific autoantibodies, typically only one is present in a given patient. It is important to emphasize that rare autoantibodies may also be associated with the risk of internal organ involvement, thereby guiding our clinical assessments. Among the rare SSc autoantibodies, our results indicate that anti-PmScl is the most frequently detected, present in 12% of our patients. This antibody appears to have a protective effect in terms of gastrointestinal involvement. Based on these findings, the potential ‘protective’ or predisposing roles of individual autoantibodies in relation to specific organ manifestations remain unclear and require confirmation through larger, multicenter studies.

7. Summary of new findings

1. Anti-topoisomerase I, a systemic sclerosis–specific antibody, can rarely be detected in SLE patients; however, no scleroderma features were observed in these cases, and no significant difference in organ manifestations was found compared to anti-topo I–negative SLE patients.
2. Anti-centromere antibodies may occur in SLE. These patients tend to develop lupus at a later age, but central nervous system involvement was not observed in this subgroup.
3. In the case of SLE-NMO, AQP4 IgG antibody was detected before the symptoms of NMO, so screening of these antibodies is advisable for SLE patients without perception of NMO symptoms.
4. AQP4 antibody titers showed positive correlation with ANA, anti-dsDNA, and anti-nucleosome titers, as well as with IFN-γ and IL-17 levels, meaning that lupus activation may also play a role in the development of NMO activity.
5. The appearance and titer increase of AQP4 IgM was observed during NMO relapse, the examination of which may help to clarify the NMO/SLE origin of the CNS symptoms.
6. In SSc, the presence of rare autoantibodies (e.g., PmScl, Ku, NOR90, Th/To) can support diagnosis when classical SSc-specific antibodies (ACA, topo I, RNA Pol III) are absent.
7. Rare autoantibodies tend to show considerable overlap both with each other and, in some cases, with classical SSc-specific antibodies.
8. In relation to SSc, the presence of rare autoantibodies may direct the investigator's attention towards certain organ abnormalities (e.g., the rate of gastrointestinal involvement is lower with PmScl positivity, and higher with U3RNP positivity), however, based on the high rate of overlap and contradictory data, we do not yet have sufficient data to make a definitive assessment of these.

7.1. List of Publications

7.1.1. Publications Forming the Basis of the Thesis

1. **Kovács K. T., Nagy G., Halda-Kiss B., Kumánovics G.**
   Significance of autoantibody assays in systemic lupus erythematosus.
   Orvosi Hetilap. 2022 Oct 23;163(43):1695–1703.
   Hungarian. doi: 10.1556/650.2022.32599, PMID: 36273352.
2. **Csóka D. L., Kovács K. T., Kumánovics G.**
   A clinical picture of unselected patients with systemic lupus erythematosus in a tertiary Hungarian center—A spectrum ranging from pure lupus to overlap syndromes.
   Journal of Clinical Medicine. 2024 May 31;13(11):3251.
   doi: 10.3390/jcm13113251, PMID: 38892962; PMCID: PMC11172817.
3. **Kovács K. T., Kalluri S. R., Boza-Serrano A., Deierborg T., Csepány T., Simó M., Rókusz L., Miseta A., Alcaraz N., Czirják L., Berki T., Molnár T., Hemmer B., Illés Z.**
   Change in autoantibody and cytokine responses during the evolution of neuromyelitis optica in patients with systemic lupus erythematosus: A preliminary study.
   Multiple Sclerosis. 2016 Aug;22(9):1192–1201.
   doi: 10.1177/1352458515613165 Epub 2015 Oct 29. PMID: 26514978.
4. **Kéringer P, Kovács KT, Nagy G, Ágoston-Szabó Á, Filipánits K, Kiss FI, Szabó A, Kumánovics G.** Patient-reported gastrointestinal involvement is associated with reduced quality of life and disability in systemic sclerosis. J Scleroderma Relat Disord. 2025 Jun 5:23971983251345284. doi: 10.1177/23971983251345284. Epub ahead of print. PMID: 40488215; PMCID: PMC12141260.

7.1.2. Abstracts Forming the Basis of the Thesis

- **2011 – Hungarian Society of Rheumatology (MRE), Poster Presentation**
  Association of Neuromyelitis Optica and Systemic Autoimmune Diseases
  Dr. Katalin T. Kovács, Dr. Renáta Hóbor, Dr. Gábor Kumánovics, Prof. Dr. László Czirják, Prof. Dr. Zsolt Illés
- **2018 – Hungarian Society of Rheumatology (MRE), Oral Presentation**
  Anti-topoisomerase and anti-centromere antibodies in systemic lupus erythematosus
  Dr. Katalin T. Kovács, Dr. Éva Tuba, Dr. Diána Simon, Prof. Dr. Tímea Berki, Prof. Dr. László Czirják, Dr. Gábor Kumánovics
- **2023 – Hungarian Society of Rheumatology (MRE), Awarded Poster Presentation**
  Prevalence of rare scleroderma-specific autoantibodies in systemic sclerosis
  Dr. Katalin T. Kovács, Kristóf Filipanits, Prof. Dr. László Czirják, Dr. Gábor Kumánovics

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Impact Factor and Citation Metrics

- **Cumulative impact factor of first-author publications used in the thesis:** 5,44
- **Total impact factor of all publications used in the thesis (4 articles):** 9.840
- **Total impact factor of all publications by the author (6 articles):** 25.787
- **Author's h-index (HI):** 4
- **Total number of independent citations:** 88
