

Canadian Rheumatology Today

Canadian Rheumatology Today 2026 Rising Stars Conference

Event Summary

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Acronyms

AAV	ANTINEUTROPHIL CYTOPLASMIC ANTIBODY-ASSOCIATED VASCULITIS
ACR	AMERICAN COLLEGE OF RHEUMATOLOGY
ACR20	AMERICAN COLLEGE OF RHEUMATOLOGY 20% IMPROVEMENT RESPONSE
ANCA	ANTINEUTROPHIL CYTOPLASMIC ANTIBODY
APS	ANTIPHOSPHOLIPID SYNDROME
ASAS	ASSESSMENT OF SPONDYLOARTHRITIS INTERNATIONAL SOCIETY
axSpA	AXIAL SPONDYLOARTHRITIS
CK	CREATINE KINASE
CNI	CALCINEURIN INHIBITOR
CT	COMPUTED TOMOGRAPHY
DAS28-CRP	28-JOINT DISEASE ACTIVITY SCORE USING C-REACTIVE PROTEIN
ECP	EXTRACORPOREAL PHOTOPHERESIS
eGFR	ESTIMATED GLOMERULAR FILTRATION RATE
EMG	ELECTROMYOGRAPHY
ERS	EUROPEAN RESPIRATORY SOCIETY
ESKD	END-STAGE KIDNEY DISEASE
ESSDAI	EUROPEAN ALLIANCE OF ASSOCIATIONS FOR RHEUMATOLOGY SJÖGREN'S SYNDROME DISEASE ACTIVITY INDEX
EULAR	EUROPEAN ALLIANCE OF ASSOCIATIONS FOR RHEUMATOLOGY
FVC	FORCED VITAL CAPACITY
HR	HAZARD RATIO
IBS	IRRITABLE BOWEL SYNDROME
ICI	IMMUNE CHECKPOINT INHIBITOR
ICP	INTRAHEPATIC CHOLESTASIS OF PREGNANCY
IIM	IDIOPATHIC INFLAMMATORY MYOPATHY
ILD	INTERSTITIAL LUNG DISEASE

Acronyms con't

IV	INTRAVENOUS
IVIG	INTRAVENOUS IMMUNOGLOBULIN
JAK	JANUS KINASE
LN	LUPUS NEPHRITIS
MMF	MYCOPHENOLATE MOFETIL
MPAA	MYCOPHENOLIC ACID ANALOGUE
MRI	MAGNETIC RESONANCE IMAGING
NNT	NUMBER NEEDED TO TREAT
NPSLE	NEUROPSYCHIATRIC SYSTEMIC LUPUS ERYTHEMATOSUS
PFS	PROGRESSION-FREE SURVIVAL
PFT	PULMONARY FUNCTION TEST
PsA	PSORIATIC ARTHRITIS
QoL	QUALITY OF LIFE
RA	RHEUMATOID ARTHRITIS
RBC	RED BLOOD CELL
RCT	RANDOMIZED CONTROLLED TRIAL
RP-ILD	RAPIDLY PROGRESSIVE INTERSTITIAL LUNG DISEASE
SARD-ILD	SYSTEMIC AUTOIMMUNE RHEUMATIC DISEASE-ASSOCIATED INTERSTITIAL LUNG DISEASE
SLE	SYSTEMIC LUPUS ERYTHEMATOSUS
UACR	URINARY ALBUMIN-TO-CREATININE RATIO
UPCR	URINE PROTEIN-TO-CREATININE RATIO

Lupus Nephritis: Application of New Guidelines

DR. ANN CLARKE

LN develops in approximately 50% of people with SLE, usually within 5 years of diagnosis, and up to 20% of people with LN develop ESKD within 10 years. People with LN have sixfold higher mortality compared with the general population, and an increased risk of cardiovascular disease and diabetes compared with people with SLE who do not have LN. Fewer than 60% achieve complete clinical remission within 2 years, and flares after remission are common, underscoring the need for more effective treatment.

Because LN can be asymptomatic, Dr. Clarke stressed screening people with SLE every 3 to 6 months during the first 5 years after diagnosis. Those who have had no evidence of LN in the first 5 years or who are in clinical remission from LN can be screened every 6 to 12 months. Assessment should include urinalysis, eGFR, UPCR/UACR, and immune serology (anti-dsDNA and C3/C4). Biopsy should be considered when results show proteinuria ≥0.5 g/24 hours; urinary sediment abnormalities even without proteinuria; or an unexplained decline in eGFR. Dr. Clarke noted that classes III, IV, and V (focal, diffuse, and membranous) account for approximately 75% to 80% of LN cases.

Three trials have shaped the modern management of LN. In BLISS-LN, belimumab plus standard therapy produced a primary efficacy renal response in 43% of patients versus 32% with placebo plus standard therapy at 104 weeks; complete renal responses occurred in 30% of patients in the treatment arm versus 20% in the standard-of-care arm. Response curves separated after approximately 24 weeks.

In AURORA 1, voclosporin plus MMF and glucocorticoids produced complete renal responses

Clinical Pearls

For ACTIVE LN, triple is superior to dual therapy. Dual may be considered for:

- Mild LN
- Vulnerable patients, e.g., frail elderly
- Limited insurance coverage

BELIMUMAB has skin/joint efficacy, but is NOT recommended for:

- Aggressive LN
- Severe extra-renal manifestations
- Significant proteinuria
- Pure Class V

CNIs cause rapid protein reduction, but are NOT recommended for:

- Low eGFR (< 45 ml/min)
- Interstitial fibrosis/ tubular atrophy
- Extra-renal manifestations

1. Sammaritano LR, et al. 2024 American College of Rheumatology (ACR) Guideline for the Screening, Treatment, and Management of Lupus Nephritis. Arthritis Care Res. 2025; Mar.
2. Fanouriakis et al. EULAR recommendations for the management of systemic lupus erythematosus with kidney involvement: 2025 update. Ann Rheum Dis (2025), https://doi.org/10.1016/j.ard.2025.09.007

in 41% versus 23% with placebo plus standard-of-care therapy at 52 weeks. Proteinuria improved rapidly, reflecting the CNI's effects on podocyte stability as well as immunomodulation. While voclosporin is not expected to be available in Canada, Dr. Clarke explained that Canadian clinicians tend to extrapolate these findings to available CNIs, including tacrolimus and cyclosporine.

The phase 3 REGENCY trial comparing obinutuzumab plus MMF with placebo plus MMF demonstrated complete renal responses in approximately 46% versus 33% with placebo at 76 weeks. Biopsy findings also supported improvements in histological activity. Dr. Clarke cautioned against direct comparisons across trials because of differing durations and endpoints.

Treatment of LN has evolved from upfront dual therapy toward triple therapy. In 2024, the ACR released guidelines recommending incorporating high-dose corticosteroids with one of the following combinations: MPAA and belimumab, MPAA and a CNI, or low-dose cyclophosphamide and belimumab. The more recent 2025 EULAR recommendations incorporate MPAA and obinutuzumab into the initial treatment options.

For clinical practice, Dr. Clarke favoured upfront triple therapy, explaining that dual therapy may be appropriate for selected patients with very mild disease, a high risk of infection or limited insurance coverage. She recommended belimumab for patients with accompanying skin and joint disease but not for patients with aggressive LN, severe extrarenal manifestations, or significant proteinuria. CNIs can cause rapid protein reduction but should be avoided when eGFR falls below 45 mL/min/1.73 m^2, as well as in patients with interstitial fibrosis, tubular atrophy, or extrarenal manifestations. Obinutuzumab is most appropriate for patients with aggressive LN and severe extrarenal manifestations, but attention should be paid to infection risk. Finally, cyclophosphamide should be reserved for patients with very aggressive crescentic LN with declining eGFR, refractory LN, and severe, life-threatening extrarenal manifestations.

She concluded by emphasizing the importance of assessing patients' adherence to medication and ensuring supportive renal care, vaccination, appropriate infection prophylaxis, and pregnancy planning. She advised continuing treatment for a minimum of 3 to 5 years after remission, withdrawing treatment gradually, and maintaining hydroxychloroquine therapy for SLE unless contraindicated.

Clinical Pearls

B-CELL DEPLETING AGENTS, particularly Obinutuzumab, recommended for:

- Aggressive LN
- Severe extra-renal manifestations
- **BUT** higher infectious risk

CYCLOPHOSPHAMIDE reserved for:

- Very aggressive crescentic LN with declining eGFR
- Refractory LN
- Severe life-threatening extra-renal manifestations

REVIEW PATIENT ADHERENCE

- Variable compliance most common cause of flares and refractory disease

1. Sammaritano LR, et al. 2024 American College of Rheumatology (ACR) Guideline for the Screening, Treatment, and Management of Lupus Nephritis. Arthritis Care Res. 2025; Mar.
2. Fanouriakis et al. EULAR recommendations for the management of systemic lupus erythematosus with kidney involvement: 2025 update. Ann Rheum Dis (2025), https://doi.org/10.1016/j.ard.2025.09.007

Neuropsychiatric Lupus: Diagnosis and Management

DR. ALEXANDRA LEGGE

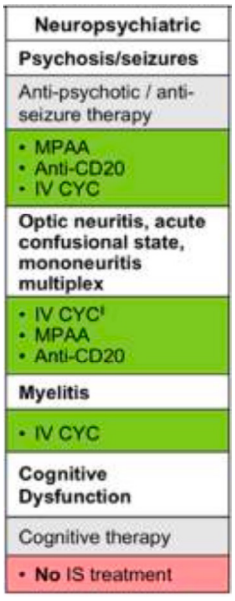
Dr. Legge outlined a systematic approach to the diagnosis and treatment of NPSLE, emphasizing the challenge of distinguishing lupus-related manifestations from neuropsychiatric events with other causes. In 1999, the ACR defined 19 neuropsychiatric syndromes, including 12 central nervous system and seven peripheral manifestations. Additional, increasingly recognized conditions include posterior reversible encephalopathy syndrome, neuromyelitis optica spectrum disorder, and small fibre neuropathy.

Because none of the included syndromes is specific to lupus, attribution models can support clinical judgment. The most recent attribution model, developed in Italy, provides a prediction score from 0 to 10 that estimates the probability that a neuropsychiatric event is attributable to SLE. This includes timing relative to SLE diagnosis, syndrome type, confounding factors, and favouring features (clinical or serological features that increase the probability of SLE being the cause). A score ≥7 demonstrated 88% sensitivity and 83% specificity, while scores <3 had a negative predictive value exceeding 90%.

A meta-analysis of 17 studies found that 56% of patients with SLE experienced a neuropsychiatric event during their disease course, usually within the first 5 years, although only about one-third of events are attributed to SLE. Mood disorders, seizures, and cerebrovascular disease were the most common neuropsychiatric events attributed to SLE. Although most events improve or resolve, recurrence occurs in 20% to 40% of patients and approximately 25% to 30% of patients experience residual neurological deficits. NPSLE has substantial effects on QoL and is associated with disability, high healthcare costs, and

2025 American College of Rheumatology (ACR) Guideline for the Treatment of Systemic Lupus Erythematosus

Severe neuropsychiatric syndromes: For Active lupus optic neuritis -OR- Lupus acute confusional state -OR- Active lupus mononeuritis multiplex: ...We conditionally recommend initial therapy with pulse/high-dose glucocorticoid taper plus immunosuppressive therapy with IV CYC, MPAA, or anti-CD20 therapy over pulse/high-dose glucocorticoid monotherapy alone.	Conditional	Very Low
For active lupus myelitis: ...We conditionally recommend initial therapy with pulse/high-dose glucocorticoid and IV CYC over pulse/high-dose glucocorticoid combined with other (non-CYC) immunosuppressive agents.	Conditional	Very Low
For active lupus psychosis: ...We conditionally recommend anti-psychotic therapy plus glucocorticoid, IV CYC, MPAA, or anti-CD20 therapy over anti-psychotic therapy alone.	Conditional	Very Low
Seizure: For seizures attributed to active SLE: ...We conditionally recommend anti-seizure medication plus glucocorticoid, CYC, MPAA, AZA, and/or anti-CD20 over anti-seizure medication alone.	Conditional	Very Low
Cognitive dysfunction: For isolated cognitive dysfunction attributed to SLE and documented by neuropsychological testing: ...We conditionally recommend *against* adding immunosuppressive therapy (including glucocorticoid) to cognitive therapy over using cognitive therapy alone.	Conditional Against	Very Low



Sammaritano LR, et al. *Arthritis Rheumatc*

greater organ damage over time.

Recognition remains a major barrier, with the INSPIRE project demonstrating that clinicians consistently underestimate neuropsychiatric symptoms among patients with systemic autoimmune rheumatic diseases: 74% of patients said they were never or rarely asked about neuropsychiatric experiences, although only 4% of clinicians agreed they never or rarely asked about neuropsychiatric symptoms. Dr. Legge advocated for proactive screening questionnaires, followed by comprehensive assessment to identify the syndrome and evaluate attribution to SLE. She recommended a laboratory assessment of SLE disease activity, cerebrospinal fluid examination, neuropsychiatric assessment, electrophysiological studies (electroencephalography, EMG/nerve conduction studies), neuroimaging, and a tissue biopsy, depending on the syndrome.

Discussing imaging, Dr. Legge explained that conventional MRI may be normal in NPSLE or show nonspecific abnormalities in patients without neuropsychiatric disease, limiting its diagnostic value.

Dr. Legge also highlighted the importance of identifying non-SLE-related contributors, including infection, metabolic abnormalities, and medication effects. For all patients, hydroxychloroquine is recommended and may be associated with a decreased risk of seizures and depressive symptoms, based on observational data. Additional management depends on the syndrome and may include cognitive rehabilitation, exercise programs and symptom-directed medications, including antidepressants, antiepileptic medications, or antipsychotic medications.

Dr. Legge distinguished inflammatory or immunothrombotic type 1 manifestations such as strokes, seizures, and cerebritis from more diffuse, chronic type 2 symptoms such as depression and anxiety. Escalating immunosuppression for isolated type 2 symptoms is generally ineffective, with treatment instead emphasizing symptom management and lifestyle interventions.

For type 1 NPSLE, treatment should be tailored according to the suspected inflammatory or ischemic mechanism, although some patients may need a combination of therapies targeting both mechanisms. Distinguishing active, potentially reversible disease from established neurological damage is essential to avoid unnecessary immunosuppression and inform prognosis. For antithrombotic management of APS, a vitamin K antagonist is generally recommended, with an international normalized ratio target of 2.0–3.0 or 3.0–4.0, depending on the risk profile. Direct oral anticoagulants are, however, discouraged in patients with definite APS with previous arterial events because of recurrent thrombosis risk (Celia et al., 2025).

RCT evidence for immunomodulatory therapy remains limited to a very small trial evaluating prednisone (Denburg et al., 1994) and a second trial of IV cyclophosphamide involving 32 patients with severe NPSLE. Dr. Legge highlighted 2025 ACR recommendations favouring immunosuppression for selected severe manifestations, while recommending against adding immunosuppression for isolated cognitive dysfunction. Emerging options include anti-CD20 therapies and anifrolumab, although evidence remains predominantly observational and case-based.



Update on ILD Guidelines in 2026

DR. SHANE SHAPERA

Dr. Shapera reviewed screening and treatment for SARD-ILD, comparing ACR and ERS/EULAR recommendations. He emphasized that key risk factors for ILD include older age, longer disease duration, disease-specific autoantibodies, increased inflammatory markers, and greater disease activity.

Dr. Shapera stressed that screening requires an ILD-protocol CT scan, with high-resolution cuts of approximately 1.5 mm or less and prone imaging to distinguish dependent atelectasis from ILD. He cited sensitivity of an ILD-protocol CT scan as 95% to 100%. Although ACR guidelines recommend both CT imaging and PFTs, Dr. Shapera questioned the incremental screening value of PFTs, which have low sensitivity, while emphasizing their use for characterizing physiology in established ILD. He also disagreed with the ACR recommendation for annual PFT screening and instead recommended repeat ILD-protocol CT scans every 3 to 5 years as a practical approach.

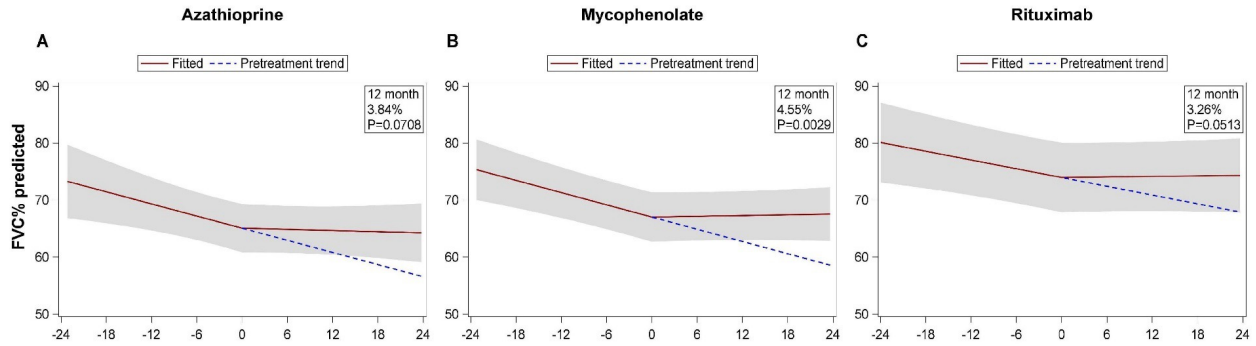
Turning to treatment, Dr. Shapera explained that mycophenolate is the preferred first-line option across many SARD-ILD presentations. In the SLS II trial, 142 patients received mycophenolate for 2 years or cyclophosphamide for 1 year, with FVC improving by approximately 2.2 and 2.9 percentage points, respectively. There was no significant difference between the two groups. Dr. Shapera explained that both medications are considered reasonable therapies because the natural history of the disease is to progress, and both groups saw slight improvements from baseline.

Dr. Shapera added that azathioprine is an alternative when cost or access limits mycophenolate, but mycophenolate is better tolerated. In a retrospective study of 97 patients, both treatments were associated with stable FVC over 4 years, while adverse effects led to discontinuation in 27% of patients receiving azathioprine versus 5% of patients receiving mycophenolate. Dr. Shapera said azathioprine is only very rarely prescribed in his clinic for ILD.

The RECITAL trial demonstrated similar FVC improvements with rituximab and IV cyclophosphamide, although neither treatment was superior. The EVER-ILD trial was highlighted as more relevant to outpatient practice, as it demonstrated that among 122 patients with nonspecific

What Is The "Best" First Line Therapy?

- Retrospective of 212 patients with RA-ILD starting therapy for ILD
  - AZA in 92 (43%), MMF in 77 (36%), and RTX in 43 (20%)
  - Similar attenuation in ΔFVC decline compared with pre-treatment in all 3 therapies



Matson SM, *et al.* Chest. 2023 Apr;163(4):861-869.

interstitial pneumonia due to SARD-ILD, interstitial pneumonia with autoimmune features, or idiopathic nonspecific interstitial pneumonia, rituximab plus mycophenolate improved FVC compared with mycophenolate alone over 6 months. FVC increased by 41 mL with combination therapy and declined by 59 mL with mycophenolate alone, while PFS also improved (HR 0.47). Nevertheless, a retrospective study of 212 patients did not establish one universally superior first-line agent (Matson et al., 2023), making toxicity, infection risk, cost, and patient characteristics important considerations.

Dr. Shapera explained that while cyclophosphamide remains effective, it is rarely used in his clinic because of toxicity. In the focuSSced trial, tocilizumab did not meet the primary skin-score endpoint but preserved lung function relative to placebo. Dr. Shapera explained that CNIs and JAK inhibitors are options in selected myositis-associated ILD, especially anti-MDA5-associated disease.

Discussing nintedanib as second-line therapy, Dr. Shapera explained that nintedanib slowed FVC decline in the INBUILD trial of more than 660 patients with progressive fibrotic ILD other than idiopathic pulmonary fibrosis. He emphasized that nintedanib can also be used as a first-line therapy in patients with RA-associated ILD with a fibrotic/usual interstitial pneumonia pattern. Evidence on whether radiographic scans predict response to nintedanib is conflicting, but Dr. Shapera recommended first-line antifibrotic therapy in selected patients with progressive pulmonary fibrotic disease.

Finally, he reviewed nerandomilast, a preferential PDE4B inhibitor with antifibrotic and anti-inflammatory effects. FIBRONEER-IPF and FIBRONEER-ILD enrolled 1,177 and 1,176 patients, respectively, comparing 9 mg or 18 mg nerandomilast twice daily with placebo. Both trials demonstrated attenuation of FVC decline, and FIBRONEER-ILD showed benefit with or without background nintedanib, supporting potential combination treatment. He highlighted an encouraging mortality signal while cautioning that mortality was not the primary endpoint.

Tolerability was promising: approximately one-quarter of patients receiving nerandomilast monotherapy experienced diarrhea, and pooled data showed diarrhea-related discontinuation in approximately 1.3% receiving the full monotherapy dose. Dr. Shapera contrasted these findings with the much higher rates of diarrhea associated with nintedanib. He anticipated that nerandomilast would substantially influence SARD-ILD management by expanding options for effective, better-tolerated antifibrotic treatment.



Sjögren's Syndrome in 2026: What You Need to Know

DR. ALICE MAI

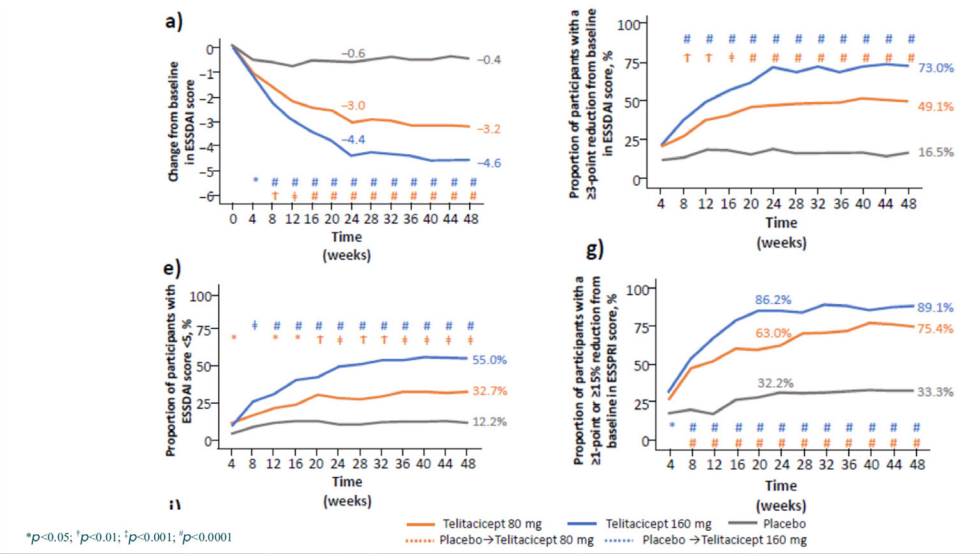
Dr. Mai reviewed the evidence supporting symptomatic and systemic treatment of Sjögren's syndrome. She emphasized that established treatment of dry eye and dry mouth symptoms remains symptomatic, while immunosuppressive therapy is reserved for moderate to severe extraglandular manifestations and prescribed off-label, largely on the basis of observational evidence.

For dry eyes, Dr. Mai recommended involving a cornea specialist and assessing patients for medications that may contribute to diminished tear production as well as differential diagnoses such as diabetes mellitus, eyelid deformities, ocular rosacea, and allergic conjunctivitis. Artificial tears are a foundational part of treatment. Dr. Mai strongly recommended preservative-free formulations when drops are needed more than 3 times daily because preservatives can damage the ocular surface. She also noted that dry eye often coexists with posterior blepharitis, which is treated with warm compresses and gentle eyelid cleansing. She recommended this hygiene regimen for all patients, as it can be difficult for rheumatologists to recognize blepharitis.

More severe ocular disease may require topical cyclosporine or lifitegrast, but cyclosporine use may be limited by burning and stinging. Short courses of topical corticosteroids lasting 2 to 4 weeks can be used as a bridge to longer-term therapy. Autologous serum drops offer another option for refractory disease, but can be difficult to access as they are only dispensed by specialty pharmacies. Moisture-chamber glasses, punctal plugs, and scleral lenses can also help to preserve ocular hydration.

For dry mouth, initial measures include frequent sips of water; avoiding irritants such as coffee, alcohol, and cannabis; reviewing medications; avoiding low-humidity environments; and using a nighttime humidifier targeting 40% to 50% humidity. Sugar-free gum or lozenges can stimulate residual salivary function, while mechanical stimulation with a button, for example, can help patients who want to avoid lozenges. Pilocarpine can be considered when patients have refractory dry eyes. A placebo-controlled RCT for pilocarpine demonstrated improved mouth dryness, nasal dryness, and ocular problems, although objective tear production did not improve. However, sweating and urinary frequency can affect tolerability. Dr. Mai also noted

Telitacicept



that artificial saliva is helpful in patients with no residual glandular function.

Dr. Mai highlighted dental decay, candidiasis, and recurrent acute parotitis as important complications. Caries may develop near the gingival margin and undermine crowns, making meticulous hygiene, fluoride, and frequent dental visits essential. Candidiasis may present as an erythematous, burning tongue rather than white plaques. Dr. Mai explained that sucrose-containing formulations warrant caution with recurrent use. Another complication is acute painful salivary gland swelling, which often reflects duct obstruction from salivary stasis; gland massage and salivary stimulation may help, and antibiotics should be reserved for associated infection.

Turning to treatment of extraglandular disease, Dr. Mai explained that ESSDAI scores of 5 to 13 indicate moderate activity and scores ≥14 indicate high activity. In the JOQUER trial, hydroxychloroquine did not improve the composite symptom endpoint of a reduction in pain, fatigue and dryness. However, a retrospective study of 221 patients suggested fewer extraglandular manifestations among hydroxychloroquine users, suggesting hydroxychloroquine may have a role in disease modification. Similarly, two rituximab RCTs failed to meet their oral dryness- and fatigue-focused endpoints, although Dr. Mai considered these findings insufficient to exclude benefit in systemic manifestations such as ILD or neuropathy. More encouragingly, a phase 2 trial of leflunomide plus hydroxychloroquine in 29 patients demonstrated a placebo-adjusted ESSDAI improvement of 4.35 points at 24 weeks.

Dr. Mai concluded by discussing positive phase 3 results for ianalumab and telitacicept, presented at ACR in 2025. In the NEPTUNUS-1 and NEPTUNUS-2 studies of patients with moderate to severe systemic Sjögren's syndrome, pooled results showed ESSDAI reductions of 6.5 versus 5.3 points with placebo at week 48, a statistically significant but modest difference. The salivary flow benefit appeared concentrated among patients with greater baseline residual function, suggesting a potential opportunity for earlier intervention.

Telitacicept was evaluated in 381 patients in China. At week 48, ESSDAI reductions were approximately 4.6 and 3.2 points with the higher and lower doses, respectively, versus 0.4 with placebo. Dr. Mai highlighted the markedly low placebo response in the telitacicept trial and noted that full publications could help clarify the data. Safety results for both medications were broadly reassuring, with serious adverse events and infections comparable between the treatment and placebo groups.



Updates on ANCA Vasculitis Assessment and Management in 2026

DR. PATRICK LIANG

Dr. Liang reviewed evolving approaches to AAV management. Guidelines consistently recommend induction with rituximab or cyclophosphamide for severe disease, with preference for rituximab, followed by rituximab maintenance therapy. Non-severe disease can be treated with glucocorticoids and methotrexate or other immunosuppressors (azathioprine, MMF).

Glucocorticoid exposure is a central concern with AAV, particularly the use of IV methylprednisolone pulses, which Dr. Liang pointed out are poorly supported by evidence. In a retrospective cohort of approximately 100 patients with severe renal involvement, pulse therapy offered no demonstrated survival or renal recovery advantage in addition to standard therapy (plasma exchange, cyclophosphamide, and high-dose oral corticosteroids). However, methylprednisolone pulses were associated with higher rates of infection and new-onset diabetes (Chanouzas et al., 2019).

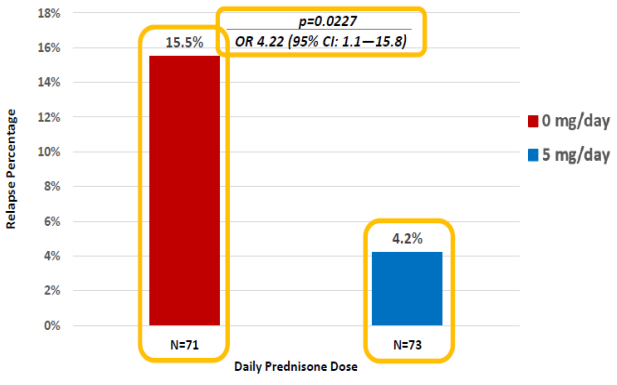
For less severe disease, the LoVAS trial supported lower starting doses of prednisone alongside rituximab. The trial enrolled more than 140 patients with newly diagnosed granulomatosis with polyangiitis or microscopic polyangiitis, without diffuse alveolar hemorrhage, and compared prednisone starting at 0.5 versus 1 mg/kg/day. In more severe AAV, the PEXIVAS trial established that a reduced-dose glucocorticoid regimen was noninferior to standard dosing for death or ESKD, with doses already halved by week 2 in the reduced-dose group. However, a subgroup analysis found patients receiving rituximab showed a nonsignificant signal towards worse outcomes with reduced-dose glucocorticoids.

A subsequent French retrospective study also found event-free survival was worse in patients who received reduced glucocorticoid dosing, especially among patients with creatinine above 300 µmol/L (Nagle et al., 2024). His interpretation was that the PEXIVAS taper is appropriate for most patients, but severe renal disease and diffuse alveolar

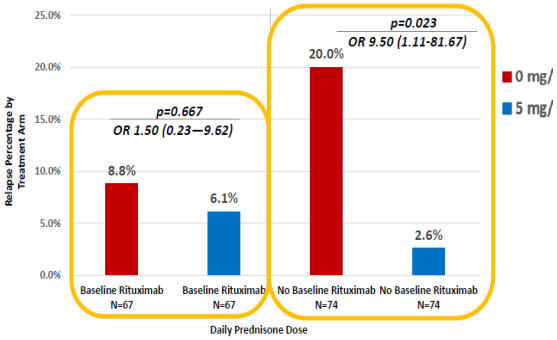
TAPIR study



TAPIR Results: Relapse by Month 6



TAPIR Results: Relapse by Month 6 Stratified by Use of Rituximab



DR. PATRICK LIANG

hemorrhage warrant slower steroid tapers.

Additional therapies may facilitate glucocorticoid withdrawal. The ADVOCATE trial compared an avacopan-based regimen with standard-dose prednisone and demonstrated similar remission rates at 6 months with approximately 65% less glucocorticoid exposure in the prednisone group. While avacopan is not currently available in Canada, Dr. Liang anticipated a future role for complement-targeting molecules. Short-course cyclophosphamide combined with rituximab is another strategy to accelerate glucocorticoid tapering. A small uncontrolled study demonstrated that rituximab combined with cyclophosphamide induced remission in more than 95% of patients with less than 2 weeks of oral glucocorticoids (Pepper et al., 2019). Dr. Liang also shared data from the TAPIR study showing that for patients in remission with rituximab maintenance, there is no value in continuing prednisone.

Relapse prediction can help to personalize steroid duration. RITAZAREM analyses suggested that persistent musculoskeletal symptoms and patient-reported outcomes offered predictive information during treatment, while rituximab therapy was protective. CD19 and ANCA measurements were not informative during active treatment, although returning CD19 levels, ANCA positivity, and inflammatory markers after treatment were associated with increased risk of relapse.

Turning to plasmapheresis, Dr. Liang described a shift towards selective use. The PEXIVAS study found no significant benefit for death or ESKD, but a subsequent meta-analysis suggested reduced ESKD risk, with an NNT as low as 7 among patients at the highest baseline creatinine levels. This benefit must be weighed against serious infections. A Bayesian analysis of PEXIVAS suggested a greater than 90% probability of both improved 1-year survival and increased serious infections in patients with diffuse alveolar hemorrhage, supporting individualized consideration in the most severe cases.

Finally, Dr. Liang shared data from the RITUXVAS and REASSESS studies showing that the combination of rituximab and cyclophosphamide may be valuable based on kidney histopathology features showing T cell mediated inflammation, and in the presence of the “crescentic” category as per the Berden classification. He noted the ENDURRANCE trial should help clarify the combination’s role.



Management of RA: New Drugs on the Horizon

DR. LIAM O'NEIL

Dr. O'Neil reviewed emerging approaches to RA treatment, emphasizing that substantial unmet needs remain despite numerous effective therapies. Approximately 40% of patients do not achieve low disease activity or remission, one in five patients meets criteria for difficult-to-treat RA, and drug-free remission remains uncommon. Many patients with RA also continue to suffer pain, fatigue, or disability.

New treatments face a high efficacy threshold and must show superiority to first- or second-line therapies. Dr. O'Neil noted that Bruton's tyrosine kinase inhibitors demonstrated efficacy but did not outperform adalimumab and therefore have not gained momentum for use in RA. Dr. O'Neil added that high placebo responses complicate interpretation of RA trials.

Discussing new targets, Dr. O'Neil explained that both otilimab, an anti-GM-CSF antibody, and peresolimab, a PD-1 agonist, failed. In contRAst 3, otilimab did not show improved ACR20 results versus placebo and did not establish noninferiority to sarilumab. The phase 2b study for peresolimab, meanwhile, was halted following safety concerns about malignancy and major adverse cardiovascular events.

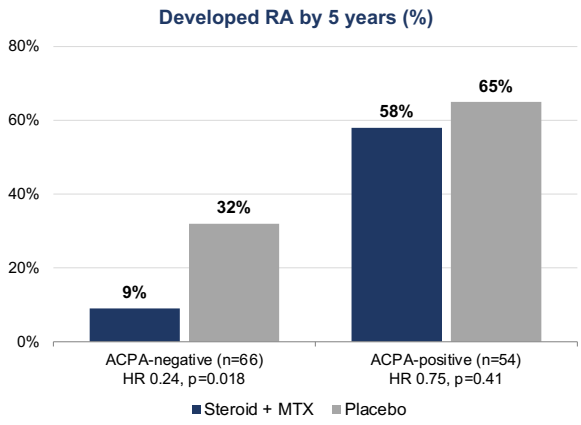
Turning to cellular therapies, Dr. O'Neil reviewed a phase 1 study of mivocabtagene autoleucel, a CD19 chimeric antigen receptor T-cell therapy, in six patients with severe, refractory ACPA-positive RA. Following lymphodepletion and treatment, all patients experienced reductions in DAS28-CRP, four achieved DAS28-CRP remission, and three achieved drug-free remission. However, all patients developed grade 1 or 2 cytokine release syndrome requiring tocilizumab and dexamethasone. Dr. O'Neil questioned whether these preliminary benefits justified the treatment intensity and risks.

Blinatumomab, a bispecific T-cell engager, was investigated in a small, open-label study in six patients with refractory RA. An IV infusion of 9 µg/day for 5 days, while methotrexate continued, demonstrated rapid B-cell depletion and clinical improvement with a median 28-joint Disease Activity Score of 2.28 (remission) at week 12. Longer follow-up in 15 patients showed ACR 50% and 70% improvement responses of 73% and 53%, respectively, but 14 patients subsequently flared after a mean of approximately 5 months. Interestingly, seven patients responded to previously unsuccessful therapies after relapse, suggesting

TREAT EARLIER: Methotrexate in At-Risk Arthralgia

- Clinically suspect arthralgia + MRI-detected subclinical inflammation (n=236); only ~23% ACPA+
  - Methylprednisolone 120 mg IM, then MTX (up to 25 mg/wk) for 1 year
  - Placebo injection + placebo tablets for 1 year
- **2 years: did not prevent arthritis** – 19% vs 18% (HR 0.81)
  - Improvement in HAQ, pain, morning stiffness and MRI inflammation
- **5 years** (increased-risk participants, n=120):
  - **ACPA-negative: RA in 9% vs 32%** (HR 0.24, NNT 4); HAQ benefit sustained
  - ACPA-positive: RA in 58% vs 65% (HR 0.75, NS); HAQ benefit lost
- Serious adverse events similar (11% in each group)

Takeaway: MTX may reduce progression to RA in ACPA-negative at-risk individuals



Krijbolder DI et al. *Lancet* 2022; van Mulligen E et al. *Lancet Rheumatol* 2026

that blinatumomab may induce an "immune reset." Dr. O'Neil cautioned, however, that the hypothesis requires further study.

Dr. O'Neil then discussed vagus nerve stimulation using an implanted device intended to activate the inflammatory reflex. In RESET-RA, 242 patients with prior biologic or targeted synthetic disease-modifying antirheumatic drug failure underwent active or sham stimulation. At 3 months, ACR20 responses were 35.2% in the active group versus 24.2% in the sham group, while DAS28-CRP low disease activity/remission occurred in 26.1% versus 15.4%. Adverse effects included voice hoarseness and stimulation-related discomfort. Although the study met its primary endpoint, Dr. O'Neil characterized efficacy as modest and questioned whether patients' awareness of stimulation may have compromised blinding.

Finally, Dr. O'Neil discussed the emerging idea of intervention before clinical RA develops. In APIPPRA, 12 months of abatacept reduced progression among autoantibody-positive individuals with arthralgia, with progression rates of 7% versus 31% during treatment and 25% versus 37% at 24 months, 12 months after the drug was stopped. However, the ALTO trial showed waning benefit after withdrawal, corresponding to approximately 5 additional arthritis-free months at 4 years among patients who received abatacept.

TREAT EARLIER evaluated 1 year of methotrexate following intramuscular methylprednisolone in individuals with clinically suspect arthralgia and MRI-detected inflammation. Results at 2 years did not demonstrate arthritis prevention overall, although symptoms and function improved. A subsequent analysis of ACPA-negative, high-risk participants found progression rates of 9% versus 32% at 5 years, with an NNT of 4. However, a similar benefit was not observed among ACPA-positive patients. Dr. O'Neil cautioned that this post hoc subgroup finding requires further confirmation. Dr. O'Neil summarized that better risk prediction and classification criteria for trials are needed to determine who can benefit from treatment to delay RA onset.



Radiologic Approach to Spondyloarthritis

DR. DENIS PODDUBNYY

Dr. Poddubnyy emphasized that effective axSpA management depends on an accurate diagnosis and confirmation that pain reflects inflammation. He introduced the revised classification criteria developed by ASAS and the Spondyloarthritis Research and Treatment Network, which require the exclusion of mimics, a history of chronic back pain beginning before age 45, and a combination of other axSpA features, as shown below. Dr. Poddubnyy explained that meeting the 11-point threshold is very unlikely without positive imaging. Crucially, MRI positivity now incorporates a global assessment of active inflammatory and structural lesions, rather than relying exclusively on bone marrow edema.

The 2026 EULAR imaging recommendations prioritize MRI of the sacroiliac joints when axSpA is suspected. If MRI is unavailable or contraindicated, radiography or preferably low-dose CT can be used. In a study of 163 patients assessed with radiography, MRI, and CT, radiography was positive in only 66% of patients with axSpA (Diekhoff et al., 2022). MRI sensitivity was approximately 82%, while CT also performed well, with a sensitivity of 72%, because many patients already have structural lesions by the time they present.

Dr. Poddubnyy highlighted that the current recommended imaging protocol for axSpA includes T2-weighted fat-suppressed sequences (short tau inversion recovery), T1-weighted sequences and the volumetric interpolated breath-hold examination sequence (or another three-dimensional gradient echo sequence for detecting erosions). The latter helps distinguish erosive damage from sclerosis. He added that contrast administration is generally unnecessary.

Bone marrow edema must be assessed according to its location, extent, and associated structural findings. In axSpA, inflammation typically involves subchondral bone in the midportion of the sacroiliac joint, whereas small or atypically located lesions have lower specificity. Sclerosis alone is nonspecific and may occur with mechanical conditions or condensing osteitis. Multiple erosions provide stronger evidence of inflammatory disease. Fat lesions become harder to interpret with age because normal marrow increasingly contains fat.

Dr. Poddubnyy then described the development of fat metaplasia in erosion cavities, termed backfill,

2025 ASAS-SPARTAN classification criteria for axSpA

Revised scoring framework

Eligible patients | Diagnosis of axial spondyloarthritis, with mimics excluded, plus back pain lasting ≥3 months and symptom onset before age 45.

Clinical and laboratory features	
HLA-B27	4 points
Inflammatory back pain[3]	3 points

Additional SpA features (1 point for each feature)
Psoriasis
Elevated CRP
Heel enthesitis
Acute anterior uveitis
Inflammatory bowel disease
Peripheral arthritis

Sacroiliac joint imaging	
Radiographic sacroiliitis[2]	7 points
OR	
MRI indicative of axSpA[1]	8 points

Classification threshold: total score ≥11 points

[1] MRI indicative of axSpA in the sacroiliac joints by global evaluation of all MRI sequences depicting active and structural lesions.
[2] According to modified New York criteria
[3] Ascertained by clinician global assessment

Adapted from: Maksymowych W et al. Arthritis Rheumatol. 2025;77(Suppl 9):0854.

followed by bone buds and ankylosis. He also shared images from a case illustrating the typical pattern of erosions and backfill.

Turning to the challenge of misdiagnosis in axSpA, Dr. Poddubnyy discussed the Improve-axSpA project, involving 969 patients from Germany and Austria. Expert central review reclassified 34% of locally diagnosed axSpA cases as not having axSpA. Among these discordant cases, 58% had active inflammatory sacroiliac joint MRI lesions, but none had active lesions judged indicative of axSpA, highlighting the consequences of interpreting nonspecific changes as disease-specific inflammation.

A German cohort study similarly demonstrated that bone marrow edema was present in approximately 30% of the general population. Female sex, increasing age, higher body mass index and occupational physical activity were associated with bone marrow edema, whereas recreational activity was associated with a lower likelihood.

Addressing the challenge of axial involvement in PsA, Dr. Poddubnyy presented the soon-to-be-published consensus definition developed by ASAS and the Group for Research and Assessment of Psoriasis and Psoriatic Arthritis, which requires at least one clinical item (chronic back pain, inflammatory back pain, good response of back pain to NSAIDs, and HLA-B27 positivity) and one imaging item (radiographic sacroiliitis, MRI indicative of axial involvement in the sacroiliac joints, presence of structural damage in an X-ray of the spine, or MRI indicative of axial involvement in the spine).



Updates on the Use of Checkpoint Inhibitors in Rheumatic Disease

DR. ALEXANDRA LADOUCEUR

Dr. Ladouceur reviewed ICI-associated myositis and inflammatory arthritis, followed by considerations for patients with pre-existing rheumatic disease. By blocking CTLA-4 or PD-1/PD-L1 pathways, ICIs enhance antitumour immunity but can also trigger immune-related toxicities. Proposed mechanisms include activation of self-reactive T and B cells, effects on regulatory T cells, shared antigens between tumours and healthy tissues, altered cytokine signalling, and microbiome effects.

ICI-associated myositis occurs in approximately 0.4% of treated patients. It is more frequent with PD-1/PD-L1 inhibitors and combination immunotherapy, as well as among men and patients older than 65 years. The median onset is 21 days after ICI initiation. Presentations include weakness, myalgia, and possible signs of rhabdomyolysis. Ptosis, diplopia, dysphagia, dysphonia, and dyspnea should prompt assessment for severe disease. Historical mortality estimates approach 20% for myositis and 50% in patients with myositis and myocarditis, although Dr. Ladouceur suggested that earlier recognition and improved treatment may have reduced the mortality rate.

Investigations include laboratory tests, EMG, MRI, muscle biopsy, as well as respiratory function tests. Respiratory muscle testing is particularly important in hospitalized patients. The differential includes ICI-associated hypothyroidism or adrenal insufficiency, neurological complications, paraneoplastic myositis, medication toxicity, and metabolic abnormalities.

Corticosteroids remain first-line treatment, with dose and urgency determined by severity. Additional options include MMF, methotrexate, IVIG, plasmapheresis, biologics, and JAK inhibitors. Dr. Ladouceur emphasized balancing toxicity control against potential interference with cancer therapy. Observational studies associate corticosteroid initiation earlier in the course of immunotherapy and higher peak doses with poorer cancer outcomes,

Diagnosis

Investigations	
Labs	CK, aldolase, LDH, ALT, AST, anti-AchR antibodies, ANA, myositis panel, troponines I, T, BNP
EMG	Myopathic changes
MRI	Edema c/w inflammation
Muscle bx	Endomysial infiltrate, T cells and macrophages +/- necrosis
Respiratory function tests	MIPs, MEPs

supporting the lowest effective dose without delaying treatment of life-threatening complications. Retrospective arthritis data similarly suggest faster arthritis control with tumour necrosis factor or interleukin-6 inhibitors than methotrexate, but shorter cancer PFS. For severe myocarditis, often overlapping with myositis, a French study reported a dramatic reduction in mortality after introducing a strategy incorporating respiratory screening, abatacept, and ruxolitinib.

Discussing JAK inhibitors, Dr. Ladouceur shared early trials suggesting that selected JAK inhibitor–ICI combinations may enhance tumour responses, although these findings remain preliminary.

ICI-associated inflammatory arthritis affects approximately 5% of patients, typically beginning around 4 months after treatment initiation. Common presentations resemble RA or polymyalgia rheumatica, although patients are frequently seronegative and may have normal CRP. Distinguishing inflammatory arthritis from activated osteoarthritis helps avoid unnecessary immunosuppression. Approximately 50% of patients who developed ICI-associated inflammatory arthritis still have active arthritis 6 months after ICI discontinuation. However, this persistence is associated with favourable tumour responses.

Treatment prioritizes function and QoL while limiting immunosuppression, rather than pursuing conventional treat-to-target remission. Mild residual symptoms or synovitis can be treated with nonsteroidal anti-inflammatory drugs, glucocorticoid injections or prednisone, with an accepted target of 10 mg/day. Conventional synthetic disease-modifying antirheumatic drugs are used as second-line therapy while biologics are third-line therapy. Decisions about continuing ICIs require coordination with oncology.

Finally, Dr. Ladouceur explained that pre-existing rheumatic disease should not automatically exclude patients from immunotherapy. A Dutch melanoma registry involving 4,367 patients treated with ICIs, including 415 with autoimmune disease, found no overall survival difference between those with and without pre-existing autoimmune disease. Canadian recommendations suggest considering tapering or stopping immunosuppression in patients with low-activity or inactive RA, PsA, or spondyloarthritis and targeting prednisone below 10 mg/day in patients with polymyalgia rheumatica while minimizing other immunosuppression. Baseline immunosuppression should be continued for systemic sclerosis, lupus, myositis, vasculitis, and Behçet's disease, while sarcoidosis treatment may be reduced unless the disease is organ-threatening. Dr. Ladouceur highlighted that these recommendations rest on very low-quality evidence, reinforcing individualized planning and close multidisciplinary follow-up.



Nociplastic Pain for the Rheumatologist in 2026

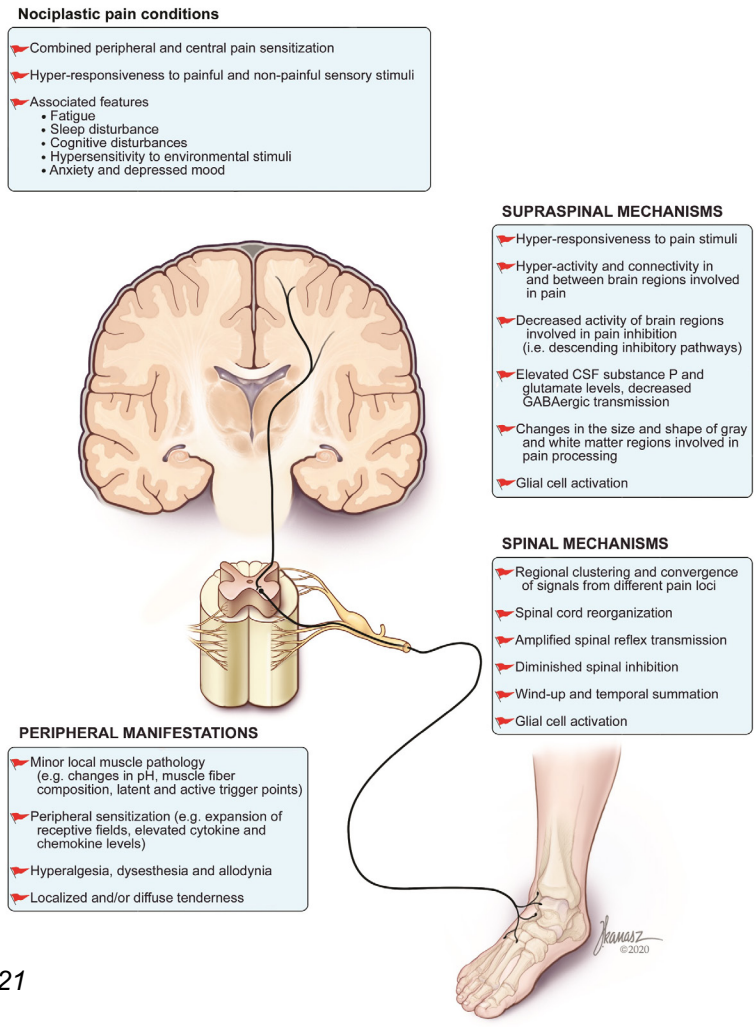
DR. MARY-ANN FITZCHARLES

Nociplastic pain is an important contributor to persistent symptoms in rheumatic disease and often coexists with nociceptive pain. Unlike nociceptive pain arising from tissue injury or neuropathic pain associated with nervous system lesions, nociplastic pain cannot be fully explained by structural abnormalities. Dr. Fitzcharles explained that the mechanism behind nociplastic pain is an amplified nervous system that clinically manifests as pain hypersensitivity.

Nociplastic pain sensitization occurs at peripheral, spinal, and cerebral levels, and is often accompanied by fatigue, cognitive difficulties, sleep disturbance, and mood symptoms. Approximately one in five patients with common rheumatologic diseases, including RA, PsA, and osteoarthritis, have a nociplastic component. Nociplastic pain affects QoL, physical function, and surgical outcomes. Dr. Fitzcharles explained that fibromyalgia is the prototype of nociplastic pain, and studies in fibromyalgia have greatly informed understanding of nociplastic pain.

Although validated questionnaires help to characterize nociplastic pain, Dr. Fitzcharles

Nociplastic pain is a fired up nervous system with sensitization at multiple levels



Fitzcharles et al. Lancet 2021

explained that clinical recognition relies primarily on history and examination. Clues include disproportionately severe, widespread, or fluctuating pain; sensitivity to environmental changes such as weather changes and stress; and accompanying fatigue or sleep disturbance. Migraine, IBS, and bladder symptoms may coexist. Pressure or gentle brushing may provoke allodynia or unpleasant sensations, and this sensation may persist after contact ends.

Turning to mechanisms, Dr. Fitzcharles reviewed evidence of persistent antigenic stimulation, glial activation, and the gut–immune–brain axis, adding that the risk of nociplastic pain is influenced by genetic and psychological factors.

Discussing immunological contributions, Dr. Fitzcharles discussed low-dose naltrexone, which may modulate neuroinflammation. A 12-week Danish RCT involving 99 patients with fibromyalgia showed no overall benefit with low-dose naltrexone. In another RCT of 28 patients with this condition, daily pain sensitivity decreased by 29% in the active arm versus 18% in the control arm over 12 weeks. In her own practice, Dr. Fitzcharles initiates a trial of naltrexone at 1 mg nightly, gradually increasing to 4.5 mg (available at compounding pharmacies). She recommended waiting 4 to 6 weeks to assess benefit, and noted vivid dreams and headaches as possible adverse effects. In her experience, some patients report substantial nociplastic pain improvements with the therapy.

As brain imaging demonstrates altered connectivity and impaired descending pain modulation in fibromyalgia, serotonin–norepinephrine reuptake inhibitor therapies have also been explored for nociplastic pain. A 14-week RCT of low-dose cyclobenzaprine in 503 patients with fibromyalgia demonstrated improved pain, sleep, and QoL. She described positive outcomes with a 5 mg evening dose of oral cyclobenzaprine in selected patients with persistent inflammatory arthritis symptoms.

The gut–immune–brain axis offers another potential mechanism for nociplastic pain. Research in this area has been especially focused on butyrate-producing organisms that support the intestinal lining. Faecal transplantation from patients with fibromyalgia to mice induced pain and associated behavioural changes, which were reversed following fecal transplantation with microbiota from healthy controls. Dr. Fitzcharles recommended a neolithic diet containing lentils, legumes, persimmons, potato skins, brown rice, and other whole grains (cooled rice has a higher resistant starch content). Studies also support plant-based diets as well as supplementation with marine oils, omega-3 fats, antioxidant-containing fruits, and turmeric.

Finally, genetics is emerging as an important player in the pathogenesis of nociplastic pain. Past studies have reported on familial aggregation in fibromyalgia, with first degree relatives having an eight-fold increased risk. A recent large multi-ancestry genome-wide association study has identified 26 risk loci for fibromyalgia, with loci similar to those identified for other chronic pain conditions such as low back pain, irritable bowel syndrome, and psychiatric conditions, leading the authors to state that there is now robust genetic evidence for fibromyalgia as a nervous system disorder. Although genes cannot be changed, epigenetic effects modulated by stress, early childhood experiences, diet, exercise, and exposure to toxins can modulate gene expression and thereby influence expression of conditions such as fibromyalgia.

Currently, lifestyle interventions are more effective than medications. Dr. Fitzcharles recommended education, physical activity, and stress reduction, emphasizing that telling patients that their pain is “real” and driven by an amplified nervous system can be reassuring for them. Dr. Fitzcharles also discussed cannabis, but noted that studies in this area are small and inconclusive, and that emerging long-term adverse effects, including potential cardiovascular effects, warrant caution.

Among nonpharmacological approaches, a trial of 384 participants with fibromyalgia found that transcutaneous electrical nerve stimulation plus physiotherapy produced a ≥30% reduction in movement-evoked pain in 41% of patients versus 13% with physiotherapy alone, with effectiveness persisting to 180 days. Transcranial stimulation also shows promise, although study limitations remain.

Dr. Fitzcharles stressed that there is currently no single treatment intervention that is a gold standard or can be universally recommended to treat nociplastic pain. Once non-pharmacologic measures have been tried, clinicians may consider a trial of drug therapy informed by evidence from treatments for fibromyalgia. She noted that most medications provide only modest effect and must always be assessed from a risk versus benefit standpoint.

The Myositis Maze: Finding Your Way Through Challenging Cases

DR. EUGENE KRUSTEV

Dr. Krustev presented six subtypes of IIM, noting that many patients previously labelled as polymyositis fit into more recently defined phenotypes. He focused on anti-MDA5 dermatomyositis.

Anti-MDA5 dermatomyositis accounts for approximately 30% of dermatomyositis cases in Japan and 5% to 15% in North American cohorts. It often presents amyopathically, although hypomyopathic and overtly myopathic presentations also occur. Other clinical manifestations include inflammatory arthritis, myocarditis, and spontaneous pneumomediastinum.

A French cohort identified three phenotypic clusters of MDA5 myositis: severe lung disease (18% of patients), predominantly rheumatoid or dermatorheumatologic disease (55%), and vasculopathic disease (27%). He added that RP-ILD develops early and is associated with risk factors including older age, Asian ethnicity, impaired PFT results, extensive CT abnormalities, lymphopenia, anti-Ro52 co-positivity, high anti-MDA5 titres, and elevated or rising ferritin.

For the treatment of anti-MDA5-related RP-ILD, ERS guidelines recommend glucocorticoids plus 2 additional agents, such as rituximab and a CNI. JAK inhibitors, IVIG, or plasmapheresis can also be considered, based on progression risk. In Canada, treatments vary regionally, as shown below.

The first case involved a previously healthy 43-year-old woman of Southeast Asian descent who developed a widespread rash, followed by dyspnea and dry cough, then fulminant respiratory failure requiring intubation. Skin biopsy showed interface dermatitis, while investigations identified strongly positive anti-MDA5 antibodies, anti-Ro52 co-positivity, ferritin of 2,600, and extensive pulmonary disease, supporting anti-MDA5 dermatomyositis with RP-ILD.



He emphasized the importance of early transplant referral, ideally before patients become critically ill. A review of 11 published transplant cases reported favourable outcomes in nine patients, supporting lung transplantation as a viable option in patients with MDA5 dermatomyositis.

Following double lung transplantation, the patient described above developed recurrent cutaneous disease requiring additional treatment, followed by persistent cytomegalovirus viremia during intensive immunosuppression. Dr. Krustev described his experience using experimental ECP for the patient to provide immunomodulation while reducing immunosuppressive therapy. After 49 cycles, the patient remained in remission with good lung function, and ECP frequency was being reduced.

The second case involved a 31-year-old woman with mild proximal weakness, CK of 2,800 U/L, low-titre anti-OJ and anti-PM/Scl-75 antibodies, muscle edema on MRI and a non-irritable myopathy on EMG. Despite escalating treatment with glucocorticoids, azathioprine, IVIG, rituximab, MMF, and tofacitinib, the patient's CK levels rose to 17,000 U/L. Muscle biopsy had suggested active and chronic necrotizing myopathy, yet her persistent biochemical abnormalities prompted diagnostic reconsideration. Neuropathology raised dysferlinopathy as an alternative diagnosis, and additional staining demonstrated a complete absence of dysferlin. Dysferlinopathy commonly presents between adolescence and the 30s and can resemble inflammatory myopathy. CK often remains above 10 times the upper limit of normal and rarely normalizes. Dysferlinopathy has no cure and is managed with rehabilitation, mechanical and respiratory aids, and surveillance. Dr. Krustev concluded that in patients with a diagnosis of IIM who are not responding to therapeutic escalation, the diagnosis should be reconsidered, as dysferlinopathy can mimic IIM both clinically and on biopsy.



Pregnancy in Rheumatic Disease in 2026

DR. ÉVELYNE VINET

Beginning with SLE in pregnancy, Dr. Vinet opened with the case of a 25-year-old woman who was in remission on hydroxychloroquine, azathioprine, and belimumab when she became pregnant. During pregnancy, azathioprine metabolite testing revealed increasing shunting towards 6-methylmercaptopurine. Dr. Vinet recommended splitting the dose of azathioprine to reduce shunting. However, at almost 25 weeks of pregnancy, the patient developed ICP, prompting azathioprine discontinuation and treatment with ursodeoxycholic acid. At 30 weeks, the patient's pruritus resolved, her bile acids were normal, she had normal renal and liver function, and UPCR was normal through pregnancy. In this case, Dr. Vinet decided to continue hydroxychloroquine and belimumab without adding tacrolimus.

The patient delivered at 36 weeks and 4 days and the infant's hemoglobin was normal. However, 2 months later, the infant presented at the emergency department with lethargy, respiratory distress, shock, and profound anemia, with hemoglobin of 18 g/L. Investigations identified parvovirus B19 infection, followed by persistent viremia, and aregenerative anemia. The case raises questions about infection susceptibility in infants following intrauterine belimumab exposure.

Turning to guidelines for SLE in pregnancy, Dr. Vinet emphasized achieving remission or low disease activity before conception and postponing pregnancy when disease remains active. The ACR strongly recommends low-dose aspirin for SLE because of the increased preeclampsia risk, and conditionally recommends low-dose aspirin for patients with aPL positivity alone. Treatment should begin early in pregnancy, ideally before 12 weeks, and generally continue until delivery.

The 2020 ACR guidelines recommend continuing low-dose glucocorticoids when appropriate (to the equivalent of ≤10 mg/day prednisone) and tapering patients on high-dose glucocorticoids (to the equivalent of ≤20 mg/day prednisone) while considering a pregnancy-compatible immunosuppressive. The 2024 EULAR recommendations favour maintenance doses of prednisone ≤5 mg/day, with withdrawal when possible in a treat-to-target approach.

The ACR and EULAR guidelines consider

Treatment of LN Class III/IV +/- V or V in Pregnancy

HCQ + ASA(hold if bx)
AND

GC pulse/oral GC >20 mg/day to taper to ≤5 mg/day at 3 months
AND

AZA + TAC +/- BEL

Consider RTX if inadequate response

Consider CYC or MMF if no response with ↓ renal function and if 2nd/3rd trimester

Adjunct Treatment

No RAAS-I but optimize BP

If nephrotic syndrome and albumin ≤20-25, prophylactic LMWH

PJP prophylaxis + Ca/vit D + PPI

hydroxychloroquine, azathioprine, tacrolimus, cyclosporine, and colchicine compatible with pregnancy and lactation. Hydroxychloroquine should be continued or initiated in pregnant patients with SLE and is also recommended for all patients who are positive for anti-Ro/SSA antibodies.

Methotrexate, MMF, and cyclophosphamide are teratogenic and should be discontinued at least 4 weeks, 6 weeks and 3 months before conception, respectively. In the case of accidental exposure, Dr. Vinet recommended a referral to a fetal medicine specialist. Cyclophosphamide may be considered during the second or third trimester when there is no other option for life- or organ-threatening disease. Additionally, the 2024 EULAR guidelines allow use of low-dose methotrexate while breastfeeding, if there is no alternative, while MMF and cyclophosphamide should not be used during breastfeeding.

Placental transfer of biologics is absent during the first trimester but increases later in pregnancy. The 2024 EULAR guidelines permit belimumab and rituximab when needed for maternal disease control, while anifrolumab should only be used in the absence of an alternative medication. Dr. Vinet explained that rituximab is associated with low or absent B lymphocyte levels in newborns who were exposed during the second or third trimester, with B-cell recovery occurring within 6 months of birth.

Moving to LN, Dr. Vinet summarized the treatment guidelines in the algorithm.

Preconception planning requires replacing effective MMF maintenance, typically with azathioprine. Dr. Vinet recommended thiopurine metabolite monitoring, however. Levels of 6-methylmercaptopurine above 5,700 pmol/(8 × 10^8 RBCs) indicate increased hepatotoxicity risk, while 6-thioguanine levels above 400 pmol/(8 × 10^8 RBCs) are associated with higher risk of myelotoxicity without increased efficacy. “Shunting” is defined as a 6-methylmercaptopurine to 6-thioguanine ratio ≥20 with high 6-methylmercaptopurine and low 6-thioguanine. In an SLE cohort of 127 patients, ICP occurred in approximately 17% of azathioprine-exposed patients versus 2.5% of unexposed pregnancies, with an adjusted HR of 12.1 (95% confidence interval 2.4–59.7). All four ICP cases with available metabolite data showed second-trimester shunting. These findings support counselling about pruritus and suggest a role for metabolite monitoring before and during pregnancy in patients exposed to azathioprine.





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