Rheumatology RESEARCH REVIEW™

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Issue 67 – 2026

In this issue:

Abbreviations used in this issue

ACPA = anti-citrullinated protein antibodies
ASDAS = axSpA Disease Activity Score
ASyS = antisynthetase syndrome
axSpA = axial spondyloarthritis
BASDAI = Bath Ankylosing Spondylitis Disease Activity Index
BASFI =Bath Ankylosing Spondylitis Functional Index
cDAPSA = Clinical Disease Activity Index for PsA
GCA = giant cell arteritis
GCAPS = Southend GCA Probability Score
(HR-)QoL = (health-related) quality of life
ILD = interstitial lung disease
IL-17A = interleukin-17A
IPTW = inverse probability of treatment weighting
NSAID = nonsteroidal anti-inflammatory drug
PsA = psoriatic arthritis
RA = rheumatoid arthritis
SSc = systemic sclerosis
THC = delta-9-tetrahydrocannabinol
TNF = tumour necrosis factor

Welcome to this issue of Rheumatology Research Review.

We begin with a double-blind Danish trial which found that cannabidiol was less effective than placebo for pain relief in fibromyalgia syndrome. This is followed by a 10-year analysis of French data which revealed that physical function and disease activity were both longitudinal determinants of HR-QoL in patients with axSpA. The next study provides evidence of differences between ACPA-positive versus ACPA-negative RA, by showing that work-related physical stress was only associated with increased joint inflammation among those with ACPA-positive RA. We also review an article which describes the performance of the Southend GCA Probability Score (GCAPS) among patients with suspected GCA within a fast-track pathway in Auckland, NZ. Our final study reports on a small yet exciting proof-of-concept case series describing the use of blinatumomab (CD19×CD3 T-cell engager) in five patients with ASyS, and teclistamab (BCMA×CD30 T-cell engager) in five patients with SSc.

We hope you enjoy this update in rheumatology research and find it valuable for the lives of your patients. Your comments and feedback are always appreciated.

Kind regards,

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Professor Simon Stebbings
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Cannabidiol versus placebo in patients with fibromyalgia

Authors: Rasmussen MU et al.

Summary: Although there is limited evidence on the efficacy of cannabidiol for pain in fibromyalgia, it is used by many patients as an analgesic supplement. In this double-blind trial from a single centre in Denmark, outpatients with fibromyalgia were randomly assigned 1:1 to receive 50mg plant-derived cannabidiol (n=100) or placebo tablets (n=100). At a follow-up of 24 weeks, patients who received cannabidiol experienced significantly smaller improvements in pain versus placebo (–0.4 vs. –1.1 points; 95% CI –1.2 to –0.25; p=0.0028). Adverse events were relatively mild and balanced between treatment arms.

Comment (AH): The pathological mechanisms of aberrant nociception in fibromyalgia syndrome are poorly understood, and pharmacological treatments, including antidepressants and anticonvulsants, provide limited benefit. Medicinal cannabis products, including THC and cannabidiol, are used in the treatment of fibromyalgia despite the lack of good quality evidence for benefit. The investigators of this study set out to test the hypothesis that cannabidiol would reduce pain scores in patients with fibromyalgia syndrome. Patients meeting the American College of Rheumatology criteria for fibromyalgia syndrome were recruited from specialist clinics, and randomised to receive either cannabidiol 50mg daily or placebo, 100 in each arm. The main outcome was change in a 0–10-point pain scale. There were secondary outcome measures of sleep, activities of daily living, quality of life and fatigue. Analysis of the data did not show that cannabidiol was superior to placebo for the primary outcome measure of reduction in pain, but it showed a statistically significant (p=0.0028), but not clinically significant, difference favouring placebo over cannabidiol. QoL improved more in the placebo group than in the cannabidiol group (EQ5D global VAS p=0.012). Adverse events were similar between the two groups; however, there were more cases of influenza in the cannabidiol group than placebo (7 vs. 23). This study does not support the use of cannabidiol for the treatment of fibromyalgia syndrome. The dose used is lower than in other studies, and it is possible that there may be benefit at a higher dose; however, the lack of any signal for a benefit at 50mg daily makes this less likely.

Reference: Ann Rheum Dis. 2026;85(3):566–74
Abstract

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Final validation of the hierarchical framework for outcomes in axial spondyloarthritis

Authors: Ortolan A et al.

Summary: The aim of this study was to examine the long-term associations between disease activity, physical function and health-related quality of life (HR-QoL) among patients with axSpA. The analysis included 663 patients (mean age 33.5 years; 46% men) from the DESIR cohort in France, with 10 years of follow-up. HR-QoL was significantly impacted by disease activity (ASDAS) across both physical components (–2.93; 95% CI –3.28 to –2.58) and mental components (–2.38; 95% CI –2.91 to –1.86). Function (BASFI) also had a significant impact on HR-QoL (physical –2.13; 95% CI –2.31 to –1.96; mental –2.38; 95% CI –2.91 to –1.86). While both function and disease activity were found to be longitudinal determinants of HR-QoL, function appeared to have a greater influence.

Comment (SS): axSpA is associated with a significant negative impact on QoL. A study 20 years ago, prior to the widespread availability of biologic therapies, showed that axSpA patients had worse QoL across all standard domains when compared with patients with heart disease, diabetes, chronic obstructive pulmonary disease and cancer (Rheumatology [Oxford]. 2007;46[6]:999–1004). This study used data from the French DESIR cohort, a 10-year prospective study of patients with early axSpA. Outcomes were collected regularly over 10 years. HR-QoL was assessed using SF-36 and ASQoL. Comparisons were made in a large cohort of 663 patients between HR-QoL and standard outcomes: disease activity (measured by ASDAS, BASDAI and C-reactive protein) and physical function (BASFI). Statistical models were also used to assess temporal relationships, adjusting for HR-QoL. Significant associations were noted between ASDAS, BASFI and HR-QoL; with a larger influence of BASFI on HR-QoL, compared with ASDAS. HR-QoL appears to reflect the cumulative impact of axSpA on an individual over time, and is mostly influenced by modifiable disease-related factors, which highlights the importance of sustained disease control. Factors such as coping strategies and personality traits, such as resilience, were not captured in the dataset, and these are likely to have an influence on HR-QoL. Overall, the findings emphasise that achieving and maintaining low disease activity is key to optimising HR-QoL in axSpA.

Reference: Ann Rheum Dis. 2026;85(2):308–18
Abstract

INDEPENDENT COMMENTARY BY
Professor Simon Stebbings

Simon Stebbings qualified from University College London. He is a Consultant Rheumatologist at Dunedin Hospital and Professor at Dunedin School of Medicine, University of Otago. His research interests include the pathogenesis of ankylosing spondylitis and the development of outcome measures in rheumatic disease.

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Work-related physical strain as novel risk factor for the severity of joint inflammation at diagnosis of anti-citrullinated protein antibodies-positive rheumatoid arthritis

Authors: Ton DA et al.

Summary: This observational cohort study from the Netherlands explored whether work-related strain was associated with joint inflammation severity at the time of RA diagnosis in ACPA-positive (n=197) and ACPA-negative (n=220) patients. While work-related physical strain was not associated with joint inflammation in ACPA-negative RA, APCA-positive patients with higher levels of occupational physical strain had more swollen joints (IRR 1.07; 95% CI 1.02–1.12) and elevated MRI joint inflammation scores (IRR 1.05; 95% CI 1.01–1.09), with raised osteitis scores (IRR 1.07; 95% CI 1.01–1.13). The authors noted that these data provide evidence for pathophysiological differences between patients with ACPA-positive and ACPA-negative RA.

Comment (AH): RA that is positive for anti-citrullinated protein antibodies (anti-CCP antibodies; ACPA) differs from ACPA-negative RA in respect to the influence of certain environmental risk factors through genetically-determined pathophysiological mechanisms. There is also limited published evidence that mechanical forces can influence the development of RA. Prolonged, repetitive, physical work is associated with an increased risk of RA, but these studies have not examined the role of ACPA in inflammatory responses to work-related physical stress. In this study, consecutive newly-diagnosed RA patients were assessed for the extent of work-related physical stress at the time of diagnosis, and the inflammatory burden, measured by physical examination (SJC44) and MRI, was compared in 197 ACPA-positive and 220 ACPA-negative RA patients. Work-related physical stress was associated with increased numbers of swollen joints in ACPA-positive RA, with each 10% increase in stress resulting in a 7% increase in SCJ44. This association was not seen in ACPA-negative RA, and subset analysis showed that this was only seen in the hands and wrists. Work-related physical stress was also associated with higher amounts of MRI-detected joint inflammation (5% increase per 10% increase in stress). Work-related physical stress was found to increase MRI-detected synovitis, tenosynovitis and osteitis, but not intermetatarsal bursitis. This study provides further evidence for a role for physical factors in ACPA-positive RA pathogenesis, and provides further evidence of differences between ACPA-positive and ACPA-negative RA.

Reference: Rheumatology (Oxford). 2026;65(2):keag009
Abstract

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Reference: 1.Nucala Data Sheet. GlaxoSmithKline New Zealand; December 2024. Available at www.medsafe.govt.nz Last Accessed December 2025.

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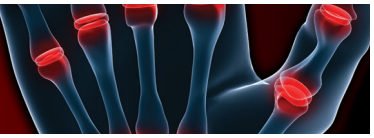

Survivorship of modern total hip replacement to 30 years

Authors: Pentland V et al.

Summary: This systematic review and meta-analysis of eight joint registries (n=1,899,034) and 29 clinical studies (n=5203) examined the longevity of contemporary total hip replacements using modern ceramics or crosslinked polyethylene. Pooled results revealed that the all-cause survivorship of hip implants was 97% (95% CI 0.96–0.98). According to registry data, the estimated survivorship was 93.6% at 20 years (95% CI 92.3–94.7), 92.8% at 25 years (95% CI 91.2–94.2) and 92.1% at 30 years (95% CI 90.1–93.7).

Comment (SS): Total hip replacement is one of the most successful surgical interventions in medicine. John Charnley in the UK evolved the procedure in the 1960s and developed a metal alloy femoral head articulating with a polyethylene acetabular component. Although incremental improvements have occurred in the last 50 years, the basic procedure remains fundamentally similar. Over time, aseptic loosening can occur in the total hip replacement prosthesis due to particulate wear debris, which causes inflammation and bone loss leading to implant failure. This was common in the past due to traditional ultra-high molecular weight polyethylene (UHMWPE) used for the acetabular cup, and in particular with metal-on-metal prostheses. Metal-on-metal prostheses peaked in 2008, but now represent <1% of procedures. The development of newer materials in the late 1990s, specifically highly crosslinked polyethylene and advanced ceramic on ceramic bearings, has greatly reduced implant wear and complications. As a result, this study set out to determine the survivorship of contemporary total hip replacements and bearing materials. The study interrogated clinical studies and joint registries as noted above, with a very large case number. The study shows that modern primary total hip replacements have excellent long-term durability, with survivorship of 93.6% at 20 years, and in excess of 92.1% at 30 years – substantially higher than found in a previous study in 2019 which reported survival at 25 years of 57.9%, and included older UHMWPE and ceramic surfaces. The improvement is, therefore, largely due to advances in bearing materials, particularly highly crosslinked polyethylene and modern ceramics, which have much lower wear rates and fewer complications than older materials. These materials are used overwhelmingly in contemporary clinical practice. Registry data formed much of the analysis, which has limitations, including under-reporting of complications. The study highlights major improvements in implant longevity, and should help rheumatologists provide up-to-date patient counselling for patients considering a total hip replacement.

Reference: Lancet. 2026;407(10531):855–66
Abstract

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Performance of the Southend Giant Cell Arteritis Probability Score in a single-centre New Zealand fast-track pathway

Authors: Okamura-Kho A et al.

Summary: These researchers evaluated the performance of the Southend GCA Probability Score (GCAPS) as a tool to identify patients at low risk of GCA within a fast-track pathway in Auckland, NZ. The analysis included 137 patients (mean age 78 years; 73% women; 75% European; 15% Asian; 4% Māori; 4% Pasifika) who were assessed between 2021–24 for suspected GCA in the Te Toka Tumai Auckland Rheumatology fast-track pathway. Overall, 61 patients (45%) were diagnosed with GCA. Other diagnoses included primary headache disorder (26%), infections (8%; i.e. dental, viral, sinusitis), mechanical issues (8%; i.e. temporomandibular dysfunction, cervical spondylosis, osteoarthritis) and polymyalgia rheumatica (7%). At referral, 127 patients (93%) commenced glucocorticoids. While 94% were assessed within 3 weeks, 82% were assessed within 2 weeks, 45% within 1 week and 16% within 72 hrs. Delayed assessment had a substantial impact on test performance: among those with GCA, eight had negative biopsy/imaging, and five of these had delayed imaging at more than 3 weeks after initiating glucocorticoids. Furthermore, ultrasound had a sensitivity of 92% within 3 weeks, versus 50% after 3 weeks. The GCAPS showed good discriminatory performance in identifying patients who were at low risk of GCA (AUC 0.91; 95% CI 0.86–0.96).

Comment (AH): GCA has the potential to cause permanent ischaemic damage to the eye, the brain and other structures. It is a medical emergency when it occludes vessels, as rapid treatment can restore blood flow. Fast-track pathways reduce the time to diagnosis and treatment, but they are time-intensive and can impeded by inappropriate referrals. The GCAPS has become a standard feature of fast-track pathways, as it has a high negative-predictive value below a score of 10/20. As different health services have variable access to diagnostic techniques such as biopsy, ultrasound and axial imaging, the performance of the GCAPS is specific to the context in which it is used and the means by which it is validated. This study was undertaken at a single NZ centre with good access to in-house ultrasound undertaken by experienced rheumatologist sonographers. Of 137 patients referred with suspected GCA, 45% were classified as GCA, and 55% not GCA. This allowed an evaluation of the performance of the GCAPS data, which had 100% sensitivity at a score of ≥9. Specificity rose steeply above that score to reach 100% at ≥15. This demonstrates the value of a low GCAPS for excluding an eventual diagnosis of GCA. The paper examined a number of other diagnostic features in this sample. C-reactive protein was >11 in 90% of cases, but not abnormal in all cases. Other symptoms that discriminated between GCA and not GCA included polymyalgia, fever, cranial or limb claudication, scalp tenderness, but not headache, visual disturbances, or any temporal artery signs. This study provides a useful stocktake of the performance of clinical and laboratory diagnostic features, and their value in determining the need for imaging and biopsy.

Reference: Int J Rheum Dis. 2026;29(2):e70562
Abstract

The impact of nonsteroidal anti-inflammatory drugs on radiographic spinal progression in patients with axial spondyloarthritis

Authors: Torgutalp M et al.

Summary: To assess the longitudinal impacts that NSAIDs had on spinal progression among patients with axSpA, these investigators analysed data from 252 patients (radiographic axSpA n=139; non-radiographic axSpA n=113) from the GESPIC cohort in Germany. Most patients (80.0%) were using NSAIDs at baseline (62% non-selective NSAIDs; 18% COX-2 inhibitors; mean intake score 38.3). Throughout 10 years, a higher intake of NSAIDs (10-point increase) was associated with a slowing of radiographic progression (β −0.052; 95% CI −0.097 to −0.007), particularly among patients with radiographic axSpA (β −0.077; 95% CI −0.152 to −0.00). Investigators noted that COX-2 inhibitors had a greater impact on slowing radiographic progression versus non-selective NSAIDs (point estimate β −0.061 vs. −0.045), although this did not reach statistical significance.

Comment (SS): In 2005, a study by Wanders et al. showed through cumulative probability plots that continuous NSAID use over 2 years reduced radiographic progression in axSpA (Arthritis Rheum. 2005;52[6]:1756–65). Subsequent studies and a 2020 meta-analysis have failed to reproduce this finding, leading to ongoing controversy. This study investigated different NSAID groups—both COX-2 selective inhibitors and non-selective NSAIDs—and divided patients with axSpA into radiographic and non-radiographic subgroups for analysis of radiographic progression in a modest-sized German cohort, with long-term follow-up of 10 years. The study found that a higher total intake of NSAIDs (i.e. higher doses and more frequent use, collected using the ASAS NSAID score) was associated with a modest but consistent slowing of radiographic spinal progression. A 10-point increase in NSAID intake corresponded with small reductions in structural damage progression, with the effect more pronounced in patients with established radiographic disease (radiographic axSpA). Surprisingly, selective COX-2 inhibitors appeared to have a stronger protective effect than non-selective NSAIDs. The authors discussed the mixed findings of previous studies noted above, which is likely due to differences in study design, populations, treatment regimens and methodologies. By including both radiographic and non-radiographic axSpA populations and comparing different NSAID types, this study helps to address some of the heterogeneity in previous study populations, which may have led to the previous conflicting data. There are compelling mechanisms by which NSAIDs may slow radiographic progression; in particular, the Wnt/DKK-1 pathway, where Wnt stimulates excessive bone formation, with inflammation and prostaglandin production increasing Wnt levels. Reduced prostaglandin production due to continuous NSAID use may, therefore, reduce prostaglandins and Wnt stimulated new bone formation. Although the observed effects seen in this study are small, cumulative effects over time may become clinically meaningful, suggesting NSAIDs, particularly COX-2 inhibitors, could have effects beyond symptom control. Overall, the study supports a modest protective role of NSAIDs in slowing disease progression, especially with COX-2 inhibitors in patients with established radiographic axSpA.

Reference: Arthritis Rheumatol. 2026;78(3):582–91
Abstract

Development and validation of a risk score for serious infection in patients with rheumatic diseases receiving prolonged high-dose glucocorticoids

Authors: Choi SR et al.

Summary: This team analysed data from two cohorts of South Korean patients with autoimmune inflammatory rheumatic diseases to develop a scoring system for infection risk among those receiving prolonged (≥4 weeks) high-dose glucocorticoid treatment (≥30mg/day prednisone equivalent). The scoring system was named the CORAL score (0–20), and infection risk was based on clinical factors such as older age, anaemia, albumin level, renal function, ILD, vasculitis, and concomitant cyclophosphamide/rituximab. CORAL achieved good predictive performance for serious infections (AUROC 0.726; 95% CI 0.681–0.771), and patients identified as high-risk (score >6) had substantially elevated risks of all-cause mortality (HR 4.55; 95% CI 2.91–7.11) and serious infection (HR 4.85; 95% CI 3.53–6.67) compared to low-risk patients (score ≤6). The accuracy of CORAL was consistent in the external validation cohort.

Comment (AH): Glucocorticoids are fast-acting, broad-spectrum immunosuppressive drugs that remain an essential component of treatment for autoimmune inflammatory rheumatic diseases. Glucocorticoids are useful for remission induction, which often requires high doses in the case of life- or organ-threatening disease. In this situation the risk of infection is increased, and where appropriate, mitigated by prophylactic antimicrobial therapies or early treatment in established infection. It would therefore be helpful to have a tool that accurately predicts risk of infection in the presence of glucocorticoids. This study from Korea recruited patients with autoimmune inflammatory rheumatic diseases treated with high-dose (≥30mg/day prednisone equivalent) glucocorticoids. There was an initial derivation cohort (n=1635) used to determine factors contributing to risk, and a validation cohort (n=672) used to assess the performance of the derived risk prediction tool (CORAL). The characteristics examined included demographic data, comorbidities, relevant laboratory data and concomitant treatments. Multivariate analysis was used to determine the factors that predicted risk, and these were given a weighting in the CORAL score. Cyclophosphamide, older age (≥60), rituximab, low serum albumin, and anaemia were weighted with 2 points; reduced renal function (eGFR <60) 4 points, interstitial lung disease (ILD) 5 points and ANCA-associated vasculitis 1 point. The components of the CORAL acronym (and mnemonic!) are underlined. The area under the ROC was 0.726, showing good predictive performance. A CORAL score of ≤6 was classed as low-risk, and >6 high-risk. Comparing the risk of serious infection in high-risk versus low-risk patients, the hazard ratio was 4.85 (95% CI 3.53–6.67), and for all-cause mortality the hazard ratio was 4.55 (95% CI 2.91–7.11). The predictive accuracy was confirmed in the validation cohort. This study underlines the contribution of ILD and renal impairment, and demonstrates potential value for the CORAL score in the management of infection risk in patients receiving high-dose glucocorticoids.

Reference: Arthritis Res Ther. 2026;28(1):72
Abstract

INDEPENDENT COMMENTARY BY

Associate Professor Andrew Harrison

Andrew Harrison is a rheumatologist based in Wellington, Associate Professor in Medicine at the University of Otago Wellington, and Medical Director of Arthritis New Zealand. He is an Otago graduate and obtained his PhD from the Royal Postgraduate Medical School in London. His research interests include the basic cellular and molecular mechanisms of inflammation, the genetics of gout and rheumatoid arthritis, and access to healthcare resources.

Current clinical practice trends in giant cell arteritis diagnosis and management

Authors: Ninan J et al.

Summary: Australian rheumatologists (n=52) and rheumatology trainees (n=6) completed an online survey to express their views and practices on the diagnosis and management of GCA. Most respondents reported using temporal artery biopsy alone (56%), while 19% used imaging alone, 15% used both and 10% used neither. Overall, 79% of respondents stated that they never or rarely diagnosed GCA without the use of temporal artery biopsy. There was uncertainty in the use of ultrasound, with only 40% of respondents feeling confident in using temporal artery ultrasound to diagnose GCA, even from their preferred radiology clinic. There was variation in management practices for GCA, and only 12% of respondents stated that more than half of their patients could successfully discontinue prednisolone within 12 months.

Comment (SS): There is a disparity in guidelines for investigating and diagnosing GCA, with EULAR advocating for imaging as first-line (especially ultrasound) and the ACR still recommending temporal artery biopsy. The American guidelines explicitly acknowledge this disparity, but note that the European guidelines assume widespread expertise in the use of ultrasound for diagnosis. In this study, Australian rheumatologists were asked about their practice in diagnosing and managing GCA. Surprisingly, diagnosis in Australia remains heavily reliant on temporal artery biopsy, with most clinicians (79%) rarely diagnosing GCA without it, reflecting alignment with ACR guidelines rather than EULAR recommendations. Although the use of ultrasound is increasing in Australia, scans are largely performed by ultrasonographers within radiology departments who may have little contact with clinicians. As a result, only ~30% of respondents felt confident in ultrasound results. In Germany and Scandinavia, rheumatologists perform most ultrasound scans themselves, with extensive ultrasound training. In Switzerland a mixed model is used, with most ultrasound scans performed by vascular ultrasonographers. This is a model increasingly adopted in NZ. Undoubtably, rheumatologists who are familiar with and use ultrasound in their own clinical practice are likely to be more confident to diagnose GCA on the basis of ultrasound alone, as this gives a them an understanding of the range of changes seen. Using ultrasound in combination with the GCAPS is also increasing in NZ (see above), and when used with ultrasound is a powerful additional diagnostic tool. It was surprising that the GCAPS was not included as an item of interest in questionnaires sent out to Australian Rheumatologists for this study. Treatment regimens varied widely. High-dose glucocorticoids were the standard treatment, but many clinicians are using steroid-sparing agents such as tocilizumab or methotrexate, often initiated at diagnosis or relapse. Tocilizumab is funded in Australia for a maximum of 12 months in GCA, unlike in NZ where access is variable via NPPA. Variation existed in how clinicians managed patients after the 12-month limit of funding, reflecting a lack of clear evidence or guidelines. Most patients remain on long-term steroids, with relatively few discontinuing treatment by 12 months. The study had a small sample size and a very poor response rate (14.5%), and also did not obtain multidisciplinary input from ophthalmologists and general physicians, which likely had a significant impact on the findings, as well as a likely strong effect of responder bias. However, good geographic coverage was obtained. This study suggests that NZ may be diverging from Australia with respect to the use of imaging and diagnosis of GCA. NZ seems to be following the European model and Australia the American – although these practices are likely to narrow over time. It would be interesting to undertake a similar study in NZ, and it would be extremely useful to establish an NZRA GCA registry.

Reference: Intern Med J. 2026;56(3):392–400
Abstract

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Cycling to TNFi vs. switching to IL-17Ai among patients with psoriatic arthritis and axial spondyloarthritis

Authors: Ogdie A et al.

Summary: There is a paucity of research on the efficacy of switching to an IL-17A inhibitor versus cycling to a second TNF inhibitor, among patients with PsA and axSpA following failure with TNF inhibitor therapy. This real-world analysis of CorEvitas PsA/SpA registry data found that among PsA patients, those who switched to an IL-17A inhibitor (n=194; 41%) achieved greater improvements in disease activity than those who cycled to a second TNF inhibitor (n=277; 59%) with regard to cDAPSA scores (–4.1; 95% CI –5.6 to –2.5). After IPTW, PsA patients who switched to an IL-17A inhibitor also achieved greater improvements in non-work activity impairment (– 4.7; 95% CI –9.2 to –0.2; p=0.039) and Physician Global Assessments of psoriasis and arthritis (–5.0; 95% CI –9.3 to –0.8; p=0.021). In the axSpA cohort, patients who switched to an IL-17A inhibitor (n=53; 31%) achieved greater improvements in disease activity versus TNF inhibitor cyclers (n=119; 69%) with regard to BASDAI scores (–0.7; 95% CI –1.2 to –0.2), and there were also improvements in fatigue after IPTW (– 9.4; 95% CI –15.8 to –3.1; p=0.004).

Comment (AH): TNF inhibitors and IL-17A inhibitors have proven efficacy in the treatment of PsA and axSpA. TNF inhibitors are commonly used before IL-17A inhibitors as they have been available for longer, and are often easier to access for first-line treatment. Following failure of a TNF inhibitor, the prescriber and patient have the option to cycle to another TNF inhibitor or to switch to an IL-17A inhibitor. This decision is not informed by strong evidence, and the authors of this study have undertaken a 'real-world' registry study to illuminate this question. The authors concluded that a switch to an IL-17A inhibitor is at least as effective, and in some measures more effective than cycling to a TNF inhibitor. IPTW methodology was used to minimise confounding due to baseline differences between cyclers and switchers. Nevertheless, there is a lingering impression that factors not fully addressed by the data may have influenced the cycle/switch decision. These include the tendency to switch rather than cycle when disease activity was high, the tendency to cycle for secondary failure and switch for primary failure, and guidelines set by funders to minimise treatment costs. Despite these limitations, the study shows that both cycling and switching are reasonable options, and there may be some outcomes that respond better to a change of biological target.

Reference: Adv Ther. Published online March 12, 2026
Abstract

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Bispecific T cell engagers for treatment-refractory autoimmune connective tissue diseases

Authors: Düsing C et al.

Summary: This case series describes five patients with treatment-refractory antisynthetase syndrome (ASyS) who were treated with blinatumomab (CD19×CD3 T-cell engager) and five patients with treatment-refractory systemic sclerosis (SSc) who received teclistamab (BCMA×CD3 T-cell engager). All patients had advanced, severe, progressive disease, and had failed to achieve disease control with ≥3 antifibrotic/immunomodulatory treatments. Among those with ASyS, blinatumomab led to reduced autoantibody titres, decreased target cells in affected muscles, improvements in markers of myositis and amelioration of ILD. In patients with SSc, teclistamab was associated with improvements in tendon friction rubs and skin fibrosis, and stabilisation of ILD. When rituximab was used as a subsequent maintenance therapy, patients demonstrated inhibited B-cell redifferentiation and extended disease control. There were no cases of immune effector cell-associated neurotoxicity syndrome, yet cytokine release syndrome occurred in all patients with SSc and two with ASyS. Overall, six patients developed respiratory tract infections and were treated with antibiotics.

Comment (SS): In a study by Müller in 2024 (N Engl J Med. 2024;390[8]:687–700), CAR T-cell therapy emerged as a potentially curative approach to treatment-resistant autoimmune disease by prolonged and profound B-cell depletion, allowing a reset and B-cell re-population after a single treatment. The need for ex-vivo cell manufacturing, however, severely limits this technology, which is prohibitively expensive and time-consuming. An alternative technology, T-cell engagers, act in a similar manner by offering sustained deep and targeted depletion of pathogenic B-cell populations, including plasma cells with simpler protocols, and a similar potential achieving sustained remission in treatment-refractory autoimmune disease. This paper reports a small but important proof-of-concept case series, exploring blinatumomab (CD19×CD3) and teclistamab (BCMA×CD30) in 10 patients with two types of refractory autoimmune diseases: ASyS and SSc. In these diseases, B-cells and autoantibodies are active mediators of disease through cytokine production, antigen presentation and tissue inflammation. Existing therapies like rituximab (anti-CD20) incompletely target the B-cell lineage, sparing plasmablasts and long-lived plasma cells, which continue producing pathogenic autoantibodies. In this study the use of bispecific T-cell engagers is important as a test-of-concept. Blinatumomab targets CD19, depleting a broad range of B-cells (but not plasma cells) and was used in patients with ASyS, whilst teclistamab targets BCMA and preferentially depletes plasma cells, and was used for patients with SSc. All 10 patients responded to treatment. In ASyS there was rapid normalisation of muscle enzymes, stabilisation of lung function and return of muscle strength. In SSc there were reduced mRSS skin scores and stabilisation of ILD. Autoantibody levels declined in both groups, especially in those treated with teclistamab. Rituximab was used to maintain benefit, but in one patient relapse occurred with B-cell return. Differences between the two agents were noted. Teclistamab caused profound antibody reduction, but there was a higher risk of infection and reduction in total immunoglobulins. Cytokine release syndrome also occurred in all patients. Macrophage activation occurs with teclistamab and leads to a cytokine storm, manifested particularly by IL-6 production, but this was less severe than seen in patients where the drug is used in cancer treatment. Intravenous immunoglobulins were required to support patients and reduce risks of infection, adding to costs. Although a small proof-of-concept study, the paper provides compelling evidence that deep, tissue-level depletion of B-cells and plasma cells using T-cell engagers can induce meaningful clinical and biologic remission in severe treatment-resistant autoimmune disease. The study supports a paradigm shift away from ideas of partial immunosuppression to immune "*resetting*" strategies. However, this approach carries significant risks for patients, specifically, cytokine release syndromes and profound immunosuppression, with very high risks of infection. Larger, controlled trials are needed to confirm efficacy, define optimal maintenance strategies, and better characterise long-term safety, but it promises a very exciting development in managing autoimmune diseases.

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Abstract

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