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The Impact of Chimeric Antigen Receptor (CAR) T Cell Therapy: Its Potential to Reshape Rheumatology Practice

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In recent years, genetically modified T cell therapy, using chimeric antigen receptor (CAR)-engineered T cells, has revolutionized the field of rheumatology. While CAR T cell therapy is approved by government agencies, including Health Canada, as a standard treatment for B cell lymphoproliferative malignancies, it has also shown remarkable efficacy in refractory cases of rheumatic diseases, including systemic lupus erythematosus, systemic sclerosis, idiopathic inflammatory myopathies, ANCA-associated vasculitis, and rheumatoid arthritis. A single infusion of CAR T cells has demonstrated the potential to induce long-term drug-free remission in most cases. This therapy achieves profound B cell depletion in both blood and tissues—an effect not typically observed with conventional antibody-based B cell-target therapies. Despite its transformative potential, several challenges remain, including questions around long-term safety, high costs, limited accessibility, and the absence of standardized guidelines, which complicate its broader application. Rheumatologists face practical uncertainties, such as determining the optimal timing for treatment, selecting suitable patients, and identifying which diseases might benefit the most from this therapy. This editorial explores the fundamental principles of CAR T cell therapy, highlights the unresolved challenges, and provides insights into how rheumatologists can optimize its use for managing rheumatic diseases. (Please note that this manuscript was written in April 2025. Given the rapid advancements and emerging evidence in this field, there may be updates by the time this article is published.)

Introduction

Since the first report of five refractory cases of systemic lupus erythematosus (SLE),[1] chimeric antigen receptor (CAR) T cell therapy targeting CD19-expressed B cells has garnered considerable attention as a potential treatment capable of inducing long-term remission for autoantibody-driven rheumatic diseases. According to the currently available literature, this therapy has been used in refractory cases of SLE,[1,2] systemic sclerosis (SSc),[3-5] idiopathic inflammatory myopathy (IIM),[5,6] ANCA-associated vasculitis (AAV), and rheumatoid arthritis (RA),[7] with additional reports likely to emerge for other rheumatic conditions. These patients had tried various currently available treatment options but failed to achieve disease control. Remarkably, all but one patient with IIM achieved drug-free remission without recurrence following a single infusion of CAR T cells, as reported in the latest conference update,[8] which included up to 3 years of follow-up in 30 patients with autoimmune diseases.

Although the remarkable effects of CAR T cell therapy have significantly impacted rheumatologists, they have also raised important considerations. Key questions include which types of rheumatic diseases, at what stages, and for which patients this therapy should be used. Additionally, safety concerns, cost, and available facilities remain major factors for many rheumatologists when contemplating the use of CAR T cell therapy in clinical practice.

This editorial provides insights into the fundamental aspects of CAR T cell therapy—such as its mechanism of action, efficacy, and safety—while also discussing its potential impact and practical considerations for rheumatologists to optimize patient outcomes with this groundbreaking treatment.

What is CAR T Cell Therapy and Why is it So Effective?

CAR T cells are genetically engineered by inserting CARs into T cells, typically using viral vectors. These T cells can be obtained from either the same patient (autologous)[1] or a donor (allogeneic).[5] CARs have various subtypes based on their targets and structural differences, with newer generations engineered to maximize their binding affinity to target cells.[4,9,10] In rheumatic diseases, where autoreactive B cell clones are primary targets, CARs targeting B cell surface markers such as CD19 or B cell maturation antigen (BCMA) have been used.[11,12] Once these CAR T cells are expanded and infused back into the patient, they recognize their target B cell lineages and eliminate the cells expressing these targets by releasing T cell-derived cytotoxic enzymes, such as granzymes and perforin.[9] Following administration, CAR T cells undergo dramatic expansion upon activation through interaction with targeted B cells, remain at high levels in the circulation for several weeks, and gradually decline over the next several months.[1,8] The key information on CAR T cells is provided in **Table 1**.

The efficacy of CAR T cells for autoimmune rheumatic diseases has been extensively discussed elsewhere,[1,13] and the results so far appear remarkably promising. Targeting B cells is not a new strategy in autoimmune-driven rheumatic diseases. Why, then, are CAR T cells so effective compared to other antibody-based B cell-targeted therapies, such as rituximab? The short answer lies in the ability of CAR T cell therapy to achieve deep depletion of B cells. As described below, several factors contribute to this superior efficacy.

First, as a "live-cell" therapy, CAR T cells possess the unique ability to migrate into tissues. Unlike rituximab, which primarily depletes circulating B cells, CAR T cells can infiltrate into major organs such as the kidneys, lungs, and even the brain by crossing the blood-brain barrier.[14] Once in the target tissues, CAR T cells bind to, and eliminate B cells, including autoreactive B cell clones, resulting in profound B cell depletion within these tissues. Indeed, a recent study demonstrated that while both CAR T cell therapy and rituximab effectively deplete B cells in the peripheral blood, only CAR T cell therapy achieves a visibly complete B cell depletion in tissues such as lymph nodes, colon, kidneys, and gallbladder.[15] This distinctive feature of CAR T cell therapy is particularly crucial in treating autoimmune diseases because it provides multiple benefits, including:

- Eliminating B cell maturation into plasmablasts or plasma cells that produce autoantibodies
- Reducing B cell-driven cytokine release
- Preventing B cell-mediated antigen presentation to naïve T cells, which could otherwise differentiate into effector T cells and contribute to tissue damage.

Questions	Answers based on currently available reports
What are the sources of CAR T cells?	Autologous (from the patient) or Allogenic (from healthy donors)
Which markers are targeted by CAR T cells?	CD19 and/or BCMA
How are CARs inserted?	Mostly virus-mediated insertion (e.g., lentiviral or retroviral vector)
How long does it take to expand CAR T cells before administration?	Several weeks. The CAR T cells are expanded using cocktails of cytokines, such as IL-2, IL-7, and IL-15
How soon are B cells eliminated from the circulation after CAR T cell infusion?	Within a week (between 3 and 7 days)
How long do CAR T cells remain in the body after infusion?	They are expanded over several weeks and gradually decreased over the following months
Which rheumatic diseases are the best targets for CD19- or BCMA-CAR T cell therapy?	B cell-driven autoimmune diseases, such as SLE, SSc, IIM, RA, SjS, and AAV
What are the common adverse events associated with CAR T cell therapy for rheumatic diseases?	CRS (grade 1*), neutropenia, lymphocytopenia, and infections (ICU cases are uncommon) The risks of ICANS and cancer appear to be very low
When does B cell reconstruction begin to be seen?	Approximately 3 months after CAR T cell infusion
Are newly emerging B cells pathogenic during reconstruction?	Based on currently available evidence, the new B cells appear to be healthy

Table 1. Ten common questions and answers regarding CAR T cell therapy for rheumatic diseases (based on evidence available as of January 2025); *courtesy of Akihiro Nakamura, MD, PhD.*

*Based on the Penn grading scale.[18]

Abbreviations: **AAV:** antineutrophil cytoplasmic antibodies (ANCA)-associated vasculitis, **BCMA:** B cell maturation antigen, **CAR:** chimeric antigen receptor, **CD19:** cluster differentiation-19, **CRS:** cytokine releasing syndrome, **ICANS:** immune-cell-associated neurotoxicity syndrome, **IIM**: idiopathic inflammatory myositis, **IL:** interleukin, **RA:** rheumatoid arthritis, **SjS:** Sjögren's syndrome, **SLE:** systemic lupus erythematosus, **SSc:** systemic sclerosis.

Another potential explanation for the deep B cell depletion achieved by CAR T cell therapy is its target, CD19 or BCMA. Unlike CD20, CD19 and BCMA are expressed on a broader range of more matured B cell lineage cells, including plasmablasts and some fractions of plasma cells. This is particularly critical, as they are major sources of autoantibodies. These plasmablasts and plasma cells do not predominantly express CD20 and can therefore persist in the circulation, tissues, or blood after anti-CD20 therapies such as rituximab and obinutuzumab.[15,16]

Another striking feature of CAR T cell therapy is its ability to induce B cell reconstruction. Within 3 to 7 days post-administration, B cells are usually completely eliminated from the circulating blood. However, approximately 3 months after CAR T cell infusion, B cells reemerge in the circulation with a phenotype similar to that of healthy individuals,[1,13] yet autoantibodies remain undetectable. This suggests a "reset" of the B cell population following CAR T cell administration. Importantly, this reset appears to be long-lasting, as patient observations over a span of 2 years have shown no reemergence of detectable autoantibodies.[13] Although the long-term outcomes beyond 2 years remain unknown and need to be monitored, the treatment has thus far produced remarkable results, including the regeneration of seemingly healthy B cells without pathogenic phenotypes.

Practical Considerations for Rheumatologists

Given such astonishing results, CAR T cell therapy holds significant potential for broader use in rheumatic diseases. However, there is currently no specific guidance or evidence for the optimal

use of CAR T cell therapy due to the limited number of cases in which it has been applied in rheumatic diseases. For rheumatologists, it is essential to address critical questions to optimize the treatment, including which diseases, at what time points, and for which patients CAR T cell therapy should be prioritized.

Although current evidence is limited, the nature of this therapy targeting B cells makes it clear that B cell-driven, autoantibody-mediated rheumatic diseases are the most suitable targets. In this context, SLE, SSc, IIM, AAV, and RA are reasonable candidates for CAR T cell therapy, as these diseases are driven by autoantibody-mediated inflammation that results in tissue damage. Importantly, recent omics-based phenotypic stratification has revealed substantial heterogeneity within the same disease, highlighting the need to identify patients with strong autoantibody signatures in their circulation and/or tissues. Other B cell-driven diseases, such as Sjögren's disease and IgG4-related disease, are also considered to potentially receive benefits from CAR T cell therapy. These diseases are currently being investigated in clinical trials, primarily based in China, as of January 2025 (**ClinicalTrials.gov** ID: NCT06497361 and NCT06056921). In contrast, diseases primarily driven by non-B cell populations (seronegative diseases), such as psoriatic arthritis and spondyloarthritis,[17] are unlikely to be suitable candidates for B cell-targeted CAR T cell therapy.

The timing of CAR T cell administration also requires discussion. Based on currently available reported cases, all patients have received the therapy after undergoing multiple treatments. This approach is logical at this point, as CAR T cell therapy is not yet a standard treatment for rheumatic diseases, largely due to the limited availability of facilities, its extremely high cost (approximately $350,000 to $500,000 USD per infusion),[9] and unconfirmed long-term safety. However, given the favourable outcomes observed over a period of up to 3 years,[8] and potentially beyond, rheumatologists may need to consider the optimal timing for CAR T cell administration to achieve the best treatment outcomes.

In this context, from my personal perspective, if no major barriers are identified in the future, earlier administration—before exhausting all other available treatments—may need to be considered, as treatment outcomes and prognosis primarily depend on minimizing permanent tissue and organ damage caused by long-term inflammation, vasculopathy, and fibrosis. To achieve this, it is crucial to risk-stratify patients based on various assessments, including the speed of disease progression, histological evaluation, imaging modalities, and functional studies. Once appropriate candidates are identified, a multidisciplinary team comprising of the rheumatologist and other specialists such as hematologists, nephrologists, and respirologists, along with an ethics board, should review the case and consider CAR T cell therapy if it is deemed the best option. Given the complexity of these cases and the need for facilities with access to a range of specialists within a multidisciplinary team, it is most practical to conduct CAR T cell therapy at tertiary academic centres. These centres often have hematologists experienced in using CAR T cell therapy for hematological cancers, therefore making them the most suitable option when Canadian rheumatologists begin using this therapy. Collaborating with hematologists also allows for the monitoring of hematological parameters, such as B cell reconstitution and potential hematological adverse events, after administration.

Safety

The safety of CAR T cell therapy, both in the short-term and long-term, is a major concern when applying it to rheumatic diseases. Given the limited number of cases in rheumatic diseases, any risks associated with CAR T therapies reported in hematological malignancy cases are considered conceivable. Although it is too early to draw definitive conclusions, the current safety profiles in rheumatic diseases appear to be milder than those in cancer treatments.

Major adverse events related to CAR T cell therapy include cytokine release syndrome (CRS), immune effector cell-associated neurotoxicity syndrome (ICANS: a condition caused by systemic inflammation and elevated cytokines, leading to CNS inflammation and can potentially life-threatening cerebral edema), macrophage activation syndrome (MAS), and hematological malignancies. CRS is commonly observed as an early adverse event following infusion, but most instances are grade 1 CRS (based on the Penn grading scale[18]), which is generally manageable with anti-pyretic treatments. Although grade 3 CRS and grade 4 ICANS were recently reported in a patient treated with CD19 CAR T cell therapy

for polyrefractory RA,[7] grade 2 or 3 CRS and other conditions, including ICANS and MAS, appear to be very uncommon based on currently available data.[8] This may be attributed to the smaller burden of B cells targeted by CAR T cells in rheumatic diseases compared to cancer, where a larger number of B cells are targeted and destroyed by the therapy. The destruction of B cells, especially in B cell lymphoma, continuously activates not only CAR T cells but also innate immune cells such as monocytes, macrophages, and neutrophils.[19] This activation leads to the release of cytotoxic or inflammatory cytokines that can cause tissue or organ damage. Furthermore, in cancer-related CAR T cell therapy, newly emerged rheumatic diseases have been reported, including RA, palindromic rheumatism, and inflammatory myositis.[20] This may be due to increased neoantigen (cancer cell-derived autoantigen) exposure from destroyed cancer cells to CD4+ T cells, triggering the activation and maturation of B cells that produce autoantibodies. Although new rheumatic diseases secondary to CAR T cell therapy have not been reported in patients with rheumatic diseases, this is theoretically possible. Therefore, careful monitoring—both during hospitalization following the infusion and during outpatient follow-up—is essential following therapy, particularly in distinguishing these conditions from the original rheumatic diseases.

Regarding the risk of infections, it is conceivable that CAR T cell therapy increases this risk. Indeed, severe infections have been reported in 7 out of 35 patients (20%), over an observation period of up to 3 years, including pneumonia related to COVID-19, cytomegalovirus (CMV), or respiratory syncytial virus (RSV).[8] In particular, due to common adverse events such as neutropenia and lymphocytopenia following lymphodepletion with cyclophosphamide and fludarabine prior to CAR T cell infusion, potential infectious risks should always be considered. However, during the observation period of up to 3 years, no cases requiring ICU admission were identified.[8] Additionally, since CAR T cell therapy has the potential to induce drug-free remission for years, the long-term risk of infections—particularly after B cell reconstruction following CAR T cell therapy—may decrease overall in patients with rheumatic diseases. Although infectious risks need to be firmly evaluated in clinical trials compared with control groups, CAR T cell therapy may ultimately reduce overall long-term infectious risks in rheumatic diseases. It is also crucial to maximize vaccination prior to CAR T cell therapy. The immunity acquired following vaccination is likely maintained even after CAR T cell-mediated elimination of CD19-expressing B cells, because long-term plasma cells generally do not express CD19, and they remain in the bone marrow for years.

In terms of malignancies, there are no reports of CAR T cell therapy causing malignancies in rheumatic diseases. Although the risk of T cell malignancy following CAR T cell therapy has raised concerns due to their nature of expansion after infusion, a recent analysis of over 3,000 pediatric and adult patients with hematologic malignancies (with a median observation period of up to 17.7 months) revealed only one case (0.03%),[21] indicating an extremely low risk. This risk appears to be similar for rheumatic diseases.

Future Directions and Conclusions

The excitement surrounding this innovative treatment, which offers the potential for long-term, drug-free remission and represents a significant step toward the ultimate goal of a ‘cure’ for autoimmune-driven rheumatic diseases, is already motivating rheumatologists to take the next steps. However, several critical challenges and uncertainties must be addressed to move forward. First, it is essential to confirm the efficacy and safety of this therapy beyond 3 years to ensure sustainable clinical outcomes without unforeseen adverse effects. As of April 2025, more than 80 clinical trials targeting rheumatic diseases are actively ongoing worldwide, according to the **ClinicalTrials.gov** registry. Most of these studies are being conducted in China, the United States, and various European countries. Currently, no trials are registered in Canada. However, given the approval of CAR T cell therapy for hematologic conditions, there is potential to initiate related clinical trials in Canada in the near future.

Second, as discussed in previous sections, rheumatologists must optimize patient selection and the timing of administration. Ideally, guidelines or recommendations for CAR T cell therapy will be developed in the future, but robust data is required to do so. While such data are unlikely

to be available in the next few years, preliminary guidance for clinicians could be developed in the future if the therapy comes to be regarded as a standard option for refractory patients. Regarding the types of diseases, testing this approach in other rapidly progressive and life-threatening B cell-driven diseases—such as anti-melanoma differentiation-associated protein 5 (MDA5)-positive dermatomyositis and catastrophic antiphospholipid antibody syndrome—should also be considered. This could broaden its applicability and address unmet needs in these critical conditions.

Finally, to achieve the best outcomes from this therapy, and minimize the risk of adverse events, such as graft-versus-host disease, further refinements in CAR structure (targeting different molecules beyond CD19 and BCMA), delivery systems (e.g., CRISPR technology), and exploration of alternative cell sources, such as γδ T cells and natural killer (NK) cells, are necessary. Additionally, employing allogeneic CAR T cells to bypass the need for T cell collection from patients, along with scaling production and marketing efforts, could significantly reduce costs and improve access to this transformative therapy.

While this therapeutic approach offers unprecedented promise, its widespread adoption and success will depend on addressing several challenges. Advances in technology, clinical validation of long-term safety, and innovative solutions to cost barriers will be critical in making this therapy a practical and sustainable option for patients with autoimmune rheumatic diseases. With continued research and collaboration, this treatment could pave the way for a new era in medicine, reshaping treatment strategies driven by rheumatologists.

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