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CAR-T CELL THERAPY IN ONCOLOGY AND RHEUMATOLOGY

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ABSTRACT

Background: CAR-T cell therapy is a highly personalized form of treatment. it involves modifying T cells extracted from the patients white blood cells during cell collection. Genetic modification of said T cells allows for the extraction of chimeric antigen receptor (CAR), which are later multiplied and infused into the recipients organism. The use of such a highly personalized method yields promising results in both oncology and hematology where relapses and the severity of conditions require precise therapy.
Aim: This narrative analysis aims to summarize and explore the present-day application of CAR-T cell therapy, currently held trials and possibilities for future therapy development in the fields of oncology and rheumatology.
Material and methods: A search of relevant research databases was conducted to identify eligible papers.
Results: CAR-T cell therapy shows promising results due to its mechanism of genetical modifications of patients' own T-cells, thus allowing them to recognize surface antigens present on cancer cells and reduce symptoms and long-term consequences of autoimmune diseases. However, its use still is limited, and more research needs to undergo to better utilize CAR-T cell therapy.
Conclusions: The use of CAR-T cell therapy in oncology and rheumatology is promising however, further studies are needed to assess its efficiency and safety.

KEYWORDS

CAR-T Cell Therapy, Hematologic Cancers, Solid Tumors, Systemic Lupus Erythematosus (SLE), Antisynthetase Syndrome

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Introduction

Chimeric Antigen Receptor T-cell therapy (CAR-T therapy) is one of the most advanced immunotherapy methods used in modern oncology. It involves genetically modifying the patient's own T cells to provide a chimeric antigen receptor (CAR). It enables them to recognize and destroy cancer cells without the major histocompatibility complex (MHC). As a result, T cells gain the ability to recognize surface antigens present on cancer cells and effectively induce their apoptosis (1, 2). At present, CAR-T therapy is most commonly used in treatment of recurrent hematological diseases including leukemia and lymphoma (3). The promising results in these cases where the basis for further evolution in solid tumors (4). CAR-T therapy could be an alternative treatment in patients with autoimmune diseases when existing therapeutic pathways, including corticosteroids and biologics, are insufficient (5). In some cases fewer pro-inflammatory cytokines were detected as well as lowered long-term symptoms and decreased antibody levels when CART-T therapy was introduced (6, 7).

1. Oncology

Preparation of CAR-T therapy consists of several stages. First, T cells are isolated from the patient through leukapheresis. These cells are then genetically modified, most often with lentiviral or retroviral vectors that insert a CAR-encoding gene into the genome. After the modified T cells have been multiplied in the laboratory, the patient undergoes lymphodepletion chemotherapy to facilitate the proliferation of the infused cells. The process concludes with a single intravenous infusion of the modified CAR-T cells into the patient (1, 2, 8).

CAR-T therapy has been most effective in treating hematologic cancers. Currently approved drugs are primarily used in patients with recurrent or resistant B-cell acute lymphoblastic leukemia (B-ALL), diffuse large B-cell lymphoma (DLBCL), mantle cell lymphoma (MCL), follicular lymphoma (FL), and multiple myeloma. In most cases, the receptors used target either CD19 or BCMA. Studies have shown that CAR-T therapy achieves high rates of complete remission even in patients who have received all available treatment options (9-13).

Despite great successes in the treatment of hematologic cancers, the use of CAR-T therapy in solid tumors remains limited. This is mainly due to the antigenic heterogeneity of tumors, impaired lymphocyte infiltration into the tumor, and an immunosuppressive tumor microenvironment. Research is focused on developing new CAR receptors capable of recognizing multiple antigens simultaneously, increasing lymphocyte resistance to immunosuppressive factors, and improving their ability to infiltrate tumor tissue. Current clinical trials include, among others, pancreatic cancer, ovarian cancer, breast cancer, lung cancer, and gliomas (2, 14-17).

Currently, the development of CAR-T therapies focuses on creating universal allogeneic "off-the-shelf" products, methods for directly modifying lymphocytes in vivo (in vivo CAR-T), and therapies using multispecific receptors. It is expected that the development of these technologies will increase treatment availability, shorten wait times for therapy, and improve the efficacy of treatment for solid tumors (2, 14, 15, 18).

CAR-T therapy is associated with characteristic adverse effects. The most common complication is cytokine release syndrome (CRS), which develops following massive activation of CAR-T cells upon recognition of a tumor antigen. The activated lymphocytes secrete numerous pro-inflammatory cytokines, including IFN-γ, TNF-α, GM-CSF, IL-2, IL-8 and IL-10, which stimulate monocytes, macrophages and dendritic cells to produce IL-6 and IL-1, leading to an intensified inflammatory response (19-22). Activation of the endothelium leads to increased vascular permeability, capillary leak syndrome, coagulation disorders and hemodynamic instability (21, 23). Clinically, CRS initially presents with fever, weakness, and muscle and joint pain; however, in more severe cases, it can lead to hypotonia, hypoxia, respiratory failure, shock and multi-organ failure (24-26).

The second key complication of CAR-T therapy is immune effector cell-associated neurotoxicity syndrome (ICANS). Symptoms may include speech disorders, confusion, tremors, aphasia, seizures and, in the most severe cases, cerebral oedema and coma. The mechanism underlying the development of ICANS has not yet been fully elucidated, although it is believed that damage to the blood–brain barrier and excessive activation of inflammatory cytokines play a significant role (21, 27, 28).

Common adverse reactions also include prolonged cytopenia, including neutropenia, thrombocytopenia and anemia. Severe cytopenia (grade ≥3) is common following CAR-T therapy, and cytopenia persisting for more than 30 days is observed in approximately 30% of patients treated with tisagenlecleucel or axicabtagene ciloleucel. The development of late-onset cytopenia is facilitated by prior intensive treatment, severe CRS and a history of hematopoietic stem cell transplantation, and results in an increased risk of severe infections and bleeding (9, 29, 30).

Patients treated with CD19-targeted CAR-T cells also frequently develop B-cell aplasia and hypogammaglobulinemia, which result from the elimination of normal B cells. Hypogammaglobulinemia may persist for many months, increasing susceptibility to infections; therefore, immunoglobulin replacement therapy is recommended for some patients – particularly children (31, 32).

Immunosuppression associated with the underlying disease, prior treatment, lymphodepletion, neutropenia and hypogammaglobulinemia means that patients who have undergone CAR-T therapy frequently develop bacterial and viral infections, and, less commonly, fungal infections and reactivation of latent viruses such as CMV, EBV or HBV (33, 34).

2. Rheumatology

2.1. Systemic lupus erythematosus

The first reported case of CAR-T therapy used to treat a rheumatic disease involved a young female patient with systemic lupus erythematosus (SLE). In this 20-year-old woman, the disease presented with multiple complications and was refractory to treatment (corticosteroids, hydroxychloroquine, cyclophosphamide, mycophenolate mofetil, tacrolimus, and biologics such as rituximab and belimumab had been used). Despite the short duration of the disease, the following conditions were observed, among others: WHO Class IIIA lupus nephritis, nephrotic syndrome, a history of Libman-Sacks endocarditis, pericarditis, pleuritis, and arthritis, as well as cutaneous lesions. Prior to CAR-T therapy, the previously administered medications were discontinued, and only prednisolone was continued. A comparison of results from days 3 and 9 after lymphocyte infusion revealed a significant increase in the number of CAR T-cells in vivo: 0.31% of the total pool of circulating T-cells on day 3 versus 27.69% on day 9, and their detectability persisted for 7 weeks. The effects of the treatment included not only significant clinical improvement and symptom reduction but also improvements in laboratory test results. A reduction in proteinuria and improved renal function was observed, along with a negative result for anti-dsDNA antibodies and simultaneous normalization of the complement components C3 and C4. Disease activity was assessed on the SLE Activity Scale as 0 points versus 16 points prior to CAR-T treatment (5). Results offered hope for the use of CAR-T therapy in the treatment of SLE in patients who had not responded to standard drug therapy. In 2022, an article was published presenting the findings from the use of CAR-T therapy in five additional patients. The results were unequivocal. Following the lymphocyte infusion, all patients achieved remission (a reduction in the disease activity score from 16 points to 0 points). In this case as well, normalization of autoantibodies against double-stranded DNA was observed. The patients were also weaned off immunosuppressive drugs, which are often associated with numerous side effects and significant reduction of patients' quality of life (35). The scientists also investigated whether autologous lymphocytes from SLE patients could be used for CAR-T therapy using the new CAR vector Hu19-CD828Z, which was provided by Kyverna Therapeutics. Unlike previously used vectors, it is characterized by the ability to maintain antigen-dependent cytotoxicity, reduced secretion of pro-inflammatory cytokines, and a limited activation-induced cell death effect. At the same time, the expression of Hu19-CD828Z ensures the production of a fully human CAR receptor targeting the CD19 antigen. The method used in this study was based on the utilization of several domains within the CAR molecule's structure. The levels of pro-inflammatory cytokines produced were assessed in vitro. It was found that in cultures of autologous primary B lymphocytes co-cultured with Hu19-CD828Z CAR T cells derived from SLE patients, fewer pro-inflammatory cytokines were detected compared to in vitro cultures using CAR T cells from healthy individuals. It was also demonstrated that autologous anti-CD19 CAR-T cells derived from SLE patients effectively eliminate CD19+ B cells and, in addition, secrete fewer pro-inflammatory cytokines than CAR-T cells derived from healthy donors (6). Recent studies have shown that it is possible to bypass the traditional

method of ex vivo cell modification and replace it with in vivo CAR-T technology that utilizes a specialized vector. In the case described, nearly complete depletion of CD19+ lymphocytes, normalization of C3 and C4 complement levels, and clinical improvement were achieved. However, further studies involving a larger group of patients are necessary (36).

2.2. Antisynthetase syndrome, systemic sclerosis, vasculitis, rheumatoid arthritis

Antisynthetase syndrome (ASS) – is an autoimmune disease characterized by the presence of anti-Jo-1 antibodies. In the treatment of refractory cases, CAR-T cell therapy can lead to remission, whether used as monotherapy or in combination therapy. It was noted that the use of CD19-targeted CAR-T cell therapy in these patients normalized the levels of pro-inflammatory cytokines, muscle enzymes and infiltrating CD8+ T cells. Additionally, the amount of anti-Jo-1 antibodies decreased. Patients also showed improvements in physical function, respiratory symptoms and non-muscle disease activity (7).

Another medical condition in which CAR-T cell therapy targeting CD19 may be effective is systemic sclerosis (SSc) - a chronic multiorgan autoimmune disease. The studies conducted to date have focused on the severe form of the disease, and the results have shown rapid improvement in cardiac and joint function, as well as skin symptoms. CAR-T therapy may also contribute to the inhibition of the progression of fibrotic changes in organs (7, 37, 38).

Other published studies have noted improvement in the severity of skin symptoms, specifically lesions with the number of ulcers decreased, the duration of Raynaud's attacks reduced, and pain alleviated. Patients have noted an improvement in hand function. In the studies, the number of B cells in the peripheral blood decreased temporarily, and the total of antinuclear antibodies also fell (38).

CAR-T cell therapy directed against CD19 is also being used in the treatment of (AAV) vasculitis associated with anti-neutrophil cytoplasmic antibodies (ANCA) which mainly affects kidneys and lungs. To date, this method has only been used in treatment of several patients with severe disease who have been unresponsive to treatment. In one patient who had received the treatment, there was a reduction in radiological lesions – granulomas – and a decrease in PR3-ANCA levels, and no new symptoms associated with AAV developed. Another patient with granulomatosis with polyangiitis (GPA) who received this treatment saw almost complete remission of the inflammatory changes in the lungs, despite previous treatment with, amongst other things, rituximab, cyclosporine A and prednisone. In contrast to the previous patient, this patient did not develop cytokine Release Syndrome (CRS). Another patient who received CAR-T cell therapy was suffering from AAV with renal involvement – glomerulonephritis with crescents. Studies observed a reduction in proteinuria and stabilization of renal function (39, 40).

In seropositive patients with rheumatoid arthritis (RA) – a condition characterized by inflammation and destruction of the synovial membrane in the joints – a reduction in disease activity, including the resolution of symptoms and remissions, was observed following treatment with CD19-targeted CAR-T cells. A decrease in the number of CD19+ B cells was observed in the peripheral blood. CRS was observed in isolated cases (7, 41, 42).

Conclusions

In oncology the application of CAR-T therapy primary focuses on treatment of hematologic cancers, solid tumor therapy is limited with trials on pancreatic cancer, breast cancer and others still in clinical stages. However, CAR-T therapy complications including cytokine release syndrome (CRS) and immune effector cell-associated neurotoxicity syndrome (ICANS) are challenging in terms of more widespread use of this therapeutic method.

Researches in order to enhance efficacy are developing new generations of CAR-T therapy such as capable of recognizing multiple antigens simultaneously (dual- and multi-target CAR-T cells), allogeneic ('off-the-shelf') constructs (4).

In the rheumatology current trials are focusing on finding more suitable targets for CAR-T therapy, improving its structure and efficiency are main goals in improving safety as well as availability (3). Most notably currently run trials focus on systemic lupus erythematosus, which could greatly improve treatment and offer further possibility of treatment for patients who are not as responsive to more traditional methods of treatment. The aforementioned therapy allowed to gain better results in laboratory tests and alleviated symptoms of the disease. CAR-T may be a promising method of treatment for patients suffering from rheumatological diseases who do not respond well to the standard treatment and combination therapies. This novel therapeutic agents may be important also in the rare conditions such as ASS.

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