American Journal of Case Reports

e-ISSN 1941-5923
© Am J Case Rep, 2026; 27: e952997
DOI: 10.12659/AJCR.952997

Received: 2026.01.31
Accepted: 2026.06.15
Available online: 2026.06.22
Published: 2026.XX.XX

CD7 CAR-T Cell as Bridging Therapy for Successful Allogeneic Hematopoietic Stem Cell Transplantation in Relapsed/Refractory T Lymphoblastic Leukemia/Lymphoma: A Case Report and Literature Review

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

BCD 1 Junying Yao*
F 2 Haiguo Zhang*
F 2 Yongtian Zhang*
F 2 Hongjing Zhou*
B 2 Yongqin Zhao
DE 2 Hongli Zhu
E 3 Junjun Meng
ABCEG 2 Meng Xiao
ABC 4 Shuguo Li

1 Shandong Second Medical University, Weifang, Shandong, PR China
2 Department of Hematology, Jining No. 1 People's Hospital, Jining, Shandong, PR China
3 Translational Pharmaceutical Laboratory, Jining No. 1 People's Hospital, Jining, Shandong, PR China
4 Department of Urology, Jining No. 1 People's Hospital, Jining, Shandong, PR China

* Junying Yao, Haiguo Zhang, Yongtian Zhang and Hongjing Zhou contributed equally to this work

Corresponding Authors: Meng Xiao, Department of Hematology, Jining No. 1 People's Hospital, Jining 272100, China, Phone: +86 537 2313703, e-mail: xiaomeng0702@163.com; Shuguo Li, Department of Urology, Jining No. 1 People's Hospital, Jining 272100, China, Phone: +86 537 2313703, e-mail: lishuguo0827@163.com

Financial support: The work founded by the Key R&D Program of Jining (Number: 2023YXNS096)
Conflict of interest: None declared

Patient: Male, 51-year-old
Final Diagnosis: T-ALL
Symptoms: Lymphadenopathy
Clinical Procedure: —
Specialty: Hematology
Objective: Unusual clinical course

Background: Relapsed/refractory T-cell acute lymphoblastic leukemia/lymphoma (R/R T-ALL/LBL) carries an exceptionally poor prognosis. Allogeneic hematopoietic stem cell transplantation (allo-HSCT) after achieving minimal residual disease (MRD)-negative complete remission (CR) offers the only possibility of long-term remission. However, conventional salvage chemotherapy rarely achieves MRD negativity. Thus, novel bridging strategies to eliminate MRD and enable transplant are urgently needed.

Case Report: We report the case of a 51-year-old man with T-ALL/LBL who remained MRD-positive despite multiple lines of salvage therapy. As a bridge to transplant, he received donor-derived CD7 chimeric antigen receptor T cells (CAR-T) from his 9/10 HLA-matched son after lymphodepletion. Grade 3 cytokine release syndrome occurred on day 3 and resolved with management; no neurotoxicity was observed. Bone marrow evaluation on day 15 confirmed morphological complete remission and MRD negativity, which proved sustained. The patient then underwent myeloablative haploidentical HSCT from the same donor. Neutrophil engraftment occurred on day +14 and platelet engraftment on day +45. No acute or chronic graft-versus-host disease developed. Serial monitoring through day +150 demonstrated sustained MRD-negative remission and donor chimerism.

Conclusions: Sequential CD7 CAR-T therapy followed by consolidative allo-HSCT is a promising curative approach for high-risk R/R T-ALL/LBL patients, enabling MRD clearance not achievable with chemotherapy alone. This case highlights the feasibility and efficacy of donor-derived CD7 CAR-T as a bridge to transplant, with manageable toxicity. Prospective studies with larger cohorts are needed to further validate this strategy and optimize timing and conditioning.

Keywords: Allogeneic Hematopoietic Stem Cell Transplantation • Case Reports • Chimeric Antigen Receptor T-Cell Therapy • T-Lymphoblastic Leukemia

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/952997

2702 — 3 34

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

Introduction

T-cell acute lymphoblastic leukemia/lymphoma (T-ALL/LBL) is a highly aggressive malignancy originating from immature T cell precursors, characterized by the abnormal proliferation and infiltration of lymphocytes in the bone marrow, peripheral blood, or extramedullary sites [1].

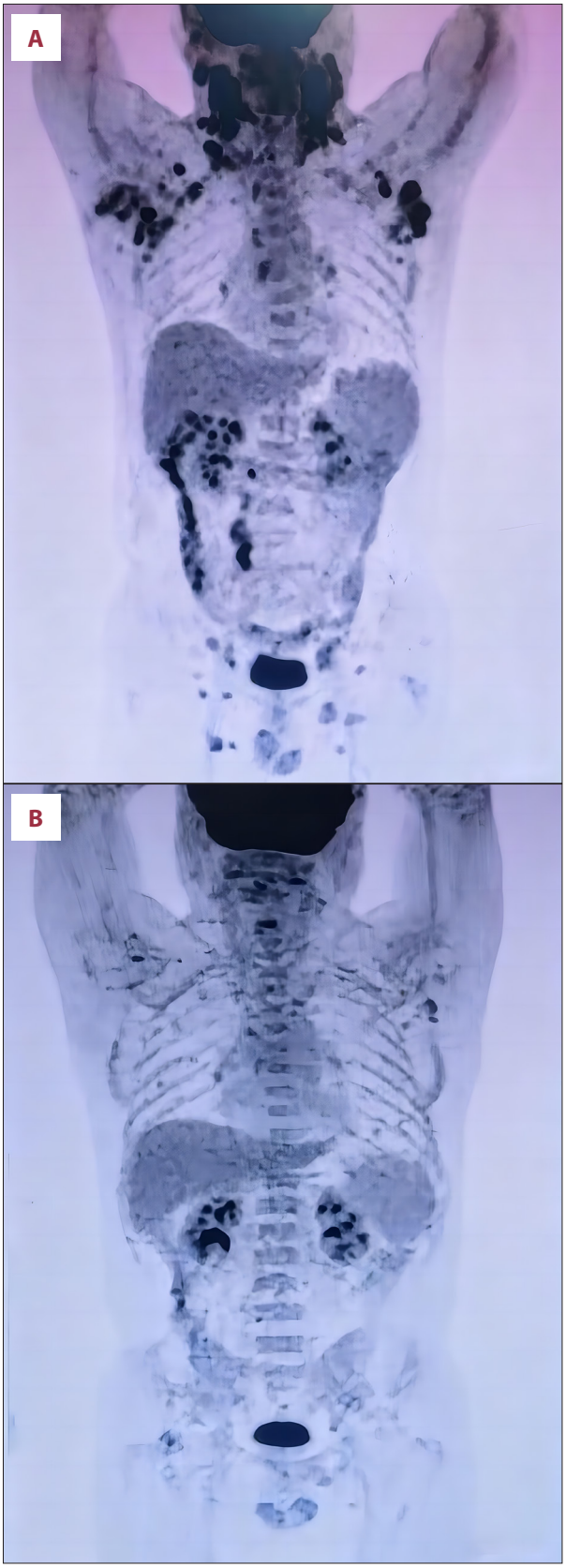
Although the incidence in adult is relatively low, patients with relapsed / refractory (R/R) T-ALL/LBL face a dismal prognosis. Following failure of standard chemotherapy, the 5-year overall survival (OS) rate is less than 20%. Minimal residual disease (MRD) status is a critical prognostic factor in ALL. Among patients who achieve complete remission (CR) after intensive chemotherapy, persistent MRD is observed in approximately 30% to 50% of cases and is the leading determinant of relapse [2]. For patients with R/R T-ALL/LBL, salvage treatment options remain limited. Nelarabine, the approved agent for this population, yields an overall response rate (ORR) of 50% and a complete remission (CR) rate of 36% in real-world studies, with 40% of patients subsequently undergoing allogeneic hematopoietic stem cell transplantation (allo-HSCT). After transplant, the 2-year and 5-year OS rates are 46% and 38%, respectively, whereas patients who do not undergo transplantation have a dismal 5-year OS of less than 10% [3]. More recently, BCL-2 inhibitors such as venetoclax have shown promise in combination regimens, achieving MRD-negative CR in selected R/R T-ALL/LBL cases. However, despite these encouraging results, the overall response rate for R/R T-ALL/LBL patients remains suboptimal, with reported CR rates ranging from 20% to 40% in this challenging population, and many responders eventually experience relapse, underscoring the limited durability of even novel targeted combination strategies 4]. Given these limitations, CD7-targeted chimeric antigen receptor T-cell (CAR-T) therapy specifically targets T-ALL/LBL cells and is a promising therapeutic avenue. CD7 is expressed on approximately 95% of T-cell leukemias and a subset of peripheral T-cell lymphomas, establishing it as an important target for CAR-T therapy in T-ALL/LBL. Reports on donor-derived CD7 CAR-T therapy as a bridge to allo-HSCT for R/R T-ALL/LBL are scarce. We retrospectively analyzed the case of a patient with R/R T-ALL/LBL who achieved MRD negativity following donor-derived CD7 CAR-T therapy, successfully bridged to allo-HSCT, and attained sustained remission. A review of the relevant literature is also provided. We present this article in accordance with the CARE reporting checklist.

Case Report

A 51-year-old man was hospitalized in Jining No. 1 People's Hospital on June 6, 2024, due to a right neck mass persisting for 1 month. The patient denied any significant past medical, family, or psychosocial history. No pertinent genetic history was noted. His past surgical and interventional history was noncontributory. Physical examination revealed multiple enlarged lymph nodes in the bilateral cervical, axillary, and inguinal regions, the largest measuring approximately 3 × 2 cm. An excisional biopsy of a right axillary lymph node was performed. Immunohistochemistry results were as follows: CD7(+), TDT(+); CD10(+), CD20(-), CD21(FDC+), CD23(FDC+), CD3(+), CD43(+), CD5(+), BCL-2(+), Bcl-6(-), CyclinD1(-), SOX-11(-), CD19(-), CD79a(-), LEF-1(+), CD4(-), CD8(-), CD56(-), CXCL-13(-), MPO(-), PD-1(-), granzyme B(-), TIA-1(-), CD2(partial+), CD34(partial+), CD117(-), CD30(-), ALK(-), and Ki67(~ 90%); in situ hybridization for EBER was negative. The histopathological findings were consistent with T lymphoblastic lymphoma (T-LBL). Bone marrow morphology revealed slightly decreased trilineage hematopoiesis, with immature lymphocytes accounting for 3%. Immunophenotyping by flow cytometry identified an abnormal population of precursor T lymphocytes comprising 44.57% of nucleated cells. These cells strongly expressed CD38m, CD3, cCD3, CD99, and CD7; partially expressed CD34 and CD2; weakly expressed CD10, CD5, and TDT; and were negative for CD117, CD33, CD14, CD19, CD16, CD13, CD1b, HLA-DR, CD71, CD36, CD11c, CD123, CD4, CD56, CD64, CD8, CD20, and CD1a. The immunophenotype supported a diagnosis of acute T lymphoblastic leukemia (T-ALL). Bone marrow biopsy showed approximately 50% cellularity, normal granulocyte-to-erythroid ratio, adequate megakaryocytes, increased lymphocytes, and myelofibrosis grade MF-1. Fusion gene screening was negative, and chromosomal karyotype analysis revealed no clonal abnormalities. PET-CT indicated metabolically active lymphadenopathy above and below the diaphragm, along with diffusely increased bone marrow metabolism, suggestive of lymphomatous involvement (**Figure 1A**). A final diagnosis of T-ALL/LBL was established.

On June 18, 2024, pre-phase therapy with the CP regimen (cyclophosphamide [CTX] 200 mg/m²and prednisone [Pre] 1 mg/kg for 5 days) was administered to reduce tumor burden. The HyperCVAD-A regimen was then initiated: CTX 200 mg/m^2 every 12 hours on days 1 to 3; vincristine (VDS) 4 mg on days 4 and 11; Epirubicin (EPI) 70 mg/m^2 on day 4; and dexamethasone (Dex) 40 mg on days 1 to 4 and 11 to 14. Bone marrow re-evaluation on July 21 showed trilineage hematopoiesis with immature lymphocytes at 28.0%. MRD analysis revealed 22.90% abnormal precursor T cells. On July 22, the HyperCVAD-B regimen was administered with methotrexate (MTX) 1g/m^2 on day 1 and cytarabine (Ara-c) 3g/m^2 every 12 hours on days 2 and 3. Bone marrow examination on August 24 demonstrated trilineage hematopoiesis with 5% immature lymphocytes; MRD decreased to 6.79%. Subsequently, the MA regimen combined with venetoclax (Ven) regimen was given: MTX 1g/m^2 on day 1; Ara-c 2.5g/m^2 every 12 hours on days 2 and 3; Ven 100 mg on day 1, 200 mg on day 2, and 400 mg on days 3 to

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]



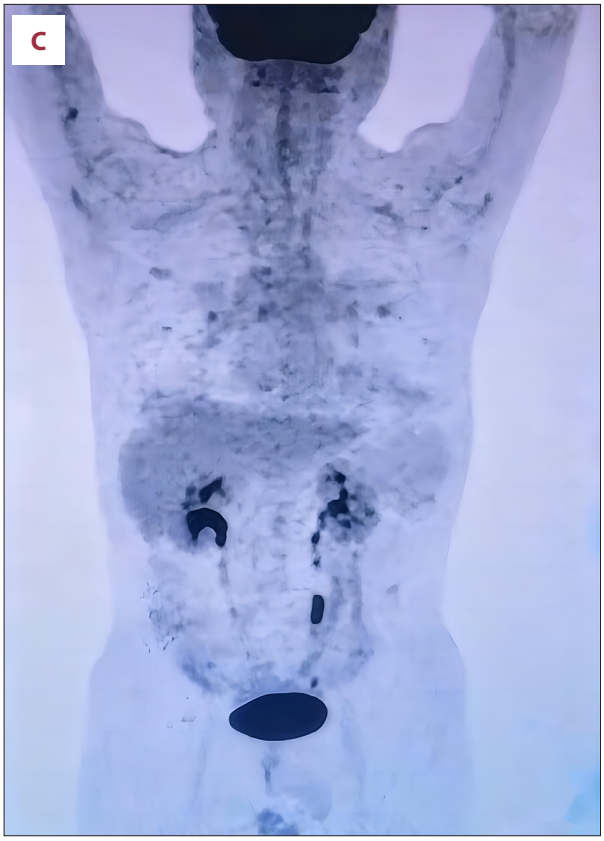


Figure 1. Results of PET-CT.(**A**) Results of PET-CT before initial treatment. Metabolically active lymphadenopathy above and below the diaphragm, along with diffusely increased bone marrow metabolism, suggestive of lymphomatous involvement. (**B**) Results of PET-CT before HSCT. Decreased size and metabolic activity in the supra- and infradiaphragmatic lymph node lesions relative to previous studies, with a Deauville score of 4. (**C**) Results of PET-CT at day +90 after HSCT. A complete metabolic response (CMR), with a Deauville score of 2. PET-CT, positron emission tomography-computed tomography; HSCT, hematopoietic stem cell transplantation.

14. On September 26, bone marrow was hypercellular with trilineage hematopoiesis; MRD was 8.61%. Due to persistent MRD positivity, the regimen was adjusted on September 27 to HyperCVD combined with azacitidine (AZA) and Ven chemotherapy: CTX 200 mg/m^2 every 12 hours on days 1 to 3, VDS 4 mg on days 4 and 11; Dex 40 mg on days 1 to 4 and 11 to 14, AZA 100 mg on days 4 to 10, and Ven 400 mg on days 1 to 7. Bone marrow assessment on October 30 showed trilineage hematopoiesis with 2% immature lymphocytes; MRD decreased to 4.31%. The MA plus Ven regimen was repeated. On December 4, bone marrow exhibited decreased cellularity with 1% immature lymphocytes; MRD further declined to 1.45%. On December 8, therapy was intensified to HyperCVD combined with AZA, Ven, and chidamide (Chi): CTX 200 mg/m^2 every 12

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

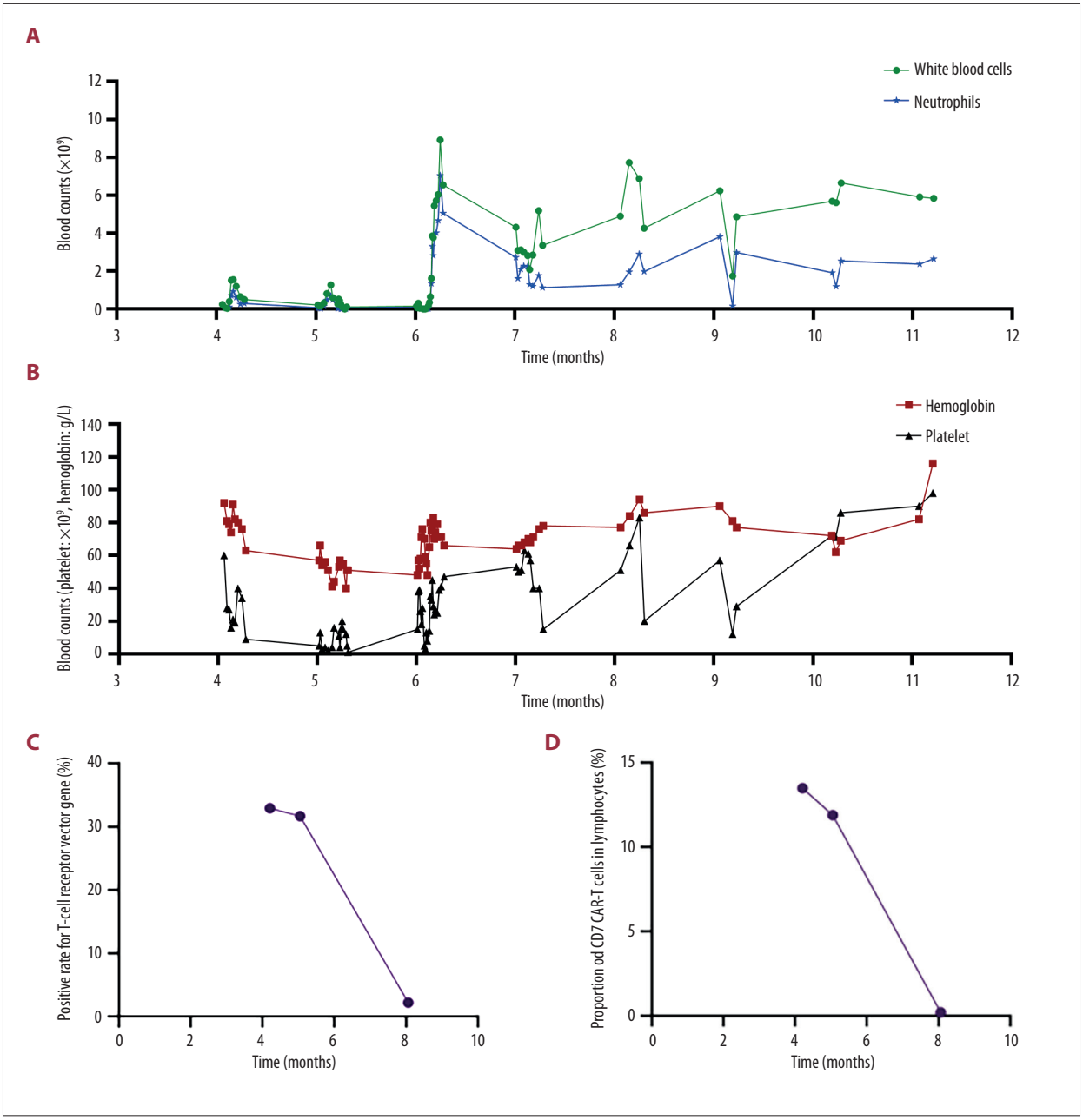


Figure 2. Dynamic monitoring of main indicators. (**A**) Changes of white blood cells and neutrophils counts in peripheral blood. (**B**) Changes of hemoglobin and platelet counts in peripheral blood. (**C**) Changes of positivity rate for T-cell receptor vector gene in peripheral blood. (**D**) Changes of proportion of CD7 CAR-T cells in lymphocytes in peripheral blood. CD7 CAR-T, CD7-targeted chimeric antigen receptor T-cell.

hours on days 1 to 3, VDS 4 mg on days 4 and 11, Dex 40 mg on days 1 to 4 and 11 to 14, AZA 100 mg on days 4 to 10, Ven 400 mg on days 1 to 7, and Chi 10 mg orally twice weekly for 2 weeks. On January 3, 2025, bone marrow showed 1% immature lymphocytes, but MRD increased to 6.32%. Given persistent MRD positivity, the regimen was changed on January 5 to GDP combined Ven and Chi: Gemcitabine 1 g/m^2 and cisplatin 75 mg/m^2 on day 1, Dex 40 mg on days 1 to 4, Ven 200mg on days 1 to 7, and Chi 10 mg orally twice weekly for 2 weeks. Bone marrow biopsy on February 5 revealed active trilineage hematopoiesis; MRD decreased to 3.04%. On February 8, the GDP combined with Ven and Chi regimen was repeated. On March 9, bone marrow examination revealed occasional naive lymphocytes. MRD increased to 11.35%. The failure of multiple intensive chemotherapy regimens combined with targeted agents to achieve MRD negativity indicated a poor prognosis.

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

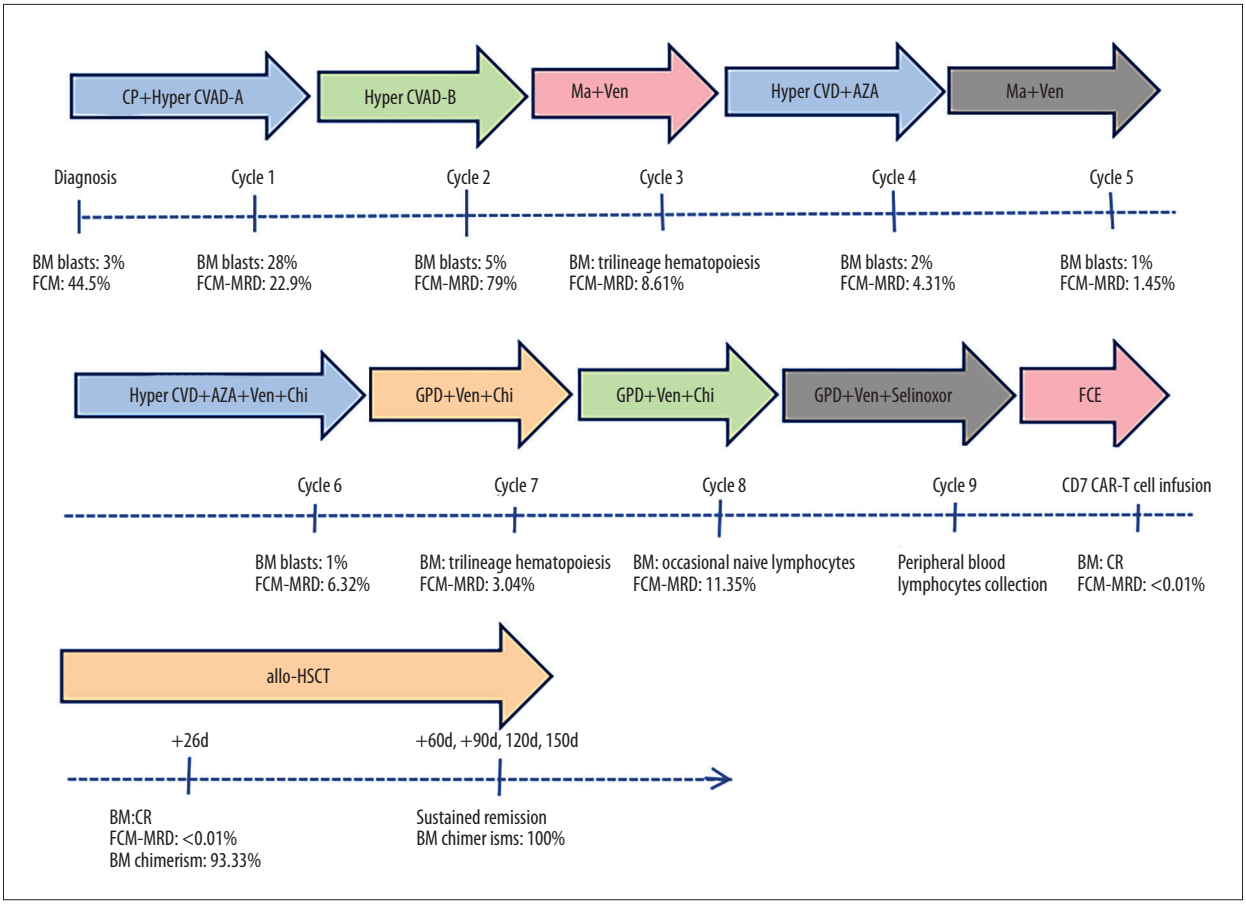


Figure 3. Display of the patient's systematic treatment process from diagnosis to allo-HSCT.CP: cyclophosphamide and prednisone; CVAD, cyclophosphamide, vincristine, epirubicin and dexamethasone; MA, methotrexate and cytarabine; Ven, venetoclax; AZA, azacitidine; Chi, chidamide; GDP, gemcitabine,cisplatin and dexamethasone; FCE, fludarabine, cyclophosphamide, and etoposide; allo-HSCT, allogeneic hematopoietic stem cell transplantation; BM, bone marrow; FCM, flow cytometry; MRD, minimal residual disease. CR, complete remission.

Salvage therapy with CD7 CAR-T cells followed by bridging to allo-HSCT was recommended. After obtaining informed consent, a reduced-dose GDP regimen combined with Ven and Selinexor was administered for tumor reduction treatment on March 11: Gemcitabine 0.6 g and Cisplatin 50 mg on day 1, Dex 20 mg on days 1 to 4, Ven 200mg on days 1 to 5, and Selinexor 20 mg orally twice weekly for 2 weeks. Concurrently, peripheral blood mononuclear cells were collected from the patient's son for the manufacture of donor-derived CD7 CAR-T cells. High-resolution HLA genotyping by polymerase chain reaction-sequence-based typing (PCR-SBT) revealed that the related donor (son) was a 9/10 HLA-matched donor (GVH and HVG direction) with a single allelic mismatch at HLA-B (donor B*15: 18 vs patient B*15: 01) and complete matching at HLA-A, C, DQB1, and DRB1. From March 31, an intensified lymphodepleting regimen consisting of fludarabine (30 mg/m^2), CTX (300 mg/m^2), and etoposide (100 mg) was administered for 5 consecutive days. The patient received infusions of donor-derived CD7 CAR-T cells (3.0 × 10^6/kg in total) on April 7th and 8th. On day 3 after infusion, the patient developed grade 3 cytokine release syndrome (CRS), which gradually improved after treatment with tocilizumab, Dex, and Dopamine to manage symptoms. No immune effector cell-associated neurotoxicity syndrome (ICANS) was observed. Bone marrow evaluation performed on day 15 after infusion demonstrated reduced cellularity with no evidence of abnormal blasts; MRD was negative (< 0.01%). The PET-CT reexamination on day 16 after infusion revealed decreased size and metabolic activity in the supra- and infradiaphragmatic lymph node lesions relative to previous studies, with a Deauville score of 4 (**Figure 1B**). Repeat bone marrow on day 39 after infusion confirmed complete morphological remission (CR) and sustained MRD negativity. No infections, clinically significant persistent cytopenias beyond the expected post-lymphodepletion nadir, or graft-versus-host disease (GVHD) were observed during the CAR-T cell treatment phase. On May 23, following myeloablative conditioning with busulfan (3.2 mg/kg/day, days -9 to -7), cyclophosphamide (40 mg/kg/day, days -6 to -5), and fludarabine (30 mg/m^2/day) plus

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

cytarabine (1 g/m^2/day, both days -4 to -2), the patient underwent haploidentical related-donor hematopoietic stem cell transplantation. Graft-versus-host disease (GVHD) prophylaxis consisted of rabbit anti-human thymocyte immunoglobulin (150 mg/day, days -4 to -2), cyclosporine in combination with mycophenolate mofetil (0.5 g/day, days +1 to +30), and short-course methotrexate (26 mg on day +1 and 17 mg on days +3, +6, and +11). Neutrophil engraftment was achieved on day +14 after transplantation, whereas platelet engraftment was delayed until day +45. Bone marrow evaluation on day +26 after HSCT showed donor-derived trilineage hematopoiesis with few megakaryocytes; MRD remained negative, and donor chimerism was 93.33%. Serial monitoring of blood counts (**Figure 2A, 2B**) and CAR-T cell expansion (**Figure 2C, 2D**) was performed. All assessments at days +60, +90, +120, and +150 after transplantation indicated sustained remission. PET-CT at day +90 after HSCT demonstrated a complete metabolic response (CMR), with a Deauville score of 2 (**Figure 1C**). The patient tolerated the procedure well and demonstrated excellent adherence. He had no major adverse events and remains under close follow-up. The disease treatment process is shown in **Figure 3**.

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Helsinki Declaration. Written informed consent for publication of this case report was not obtained from the patient or the relatives after all possible attempts were made.

Discussion

T-ALL/LBL is a highly aggressive and heterogeneous malignancy derived from immature T-cell precursors, frequently associated with chemotherapy resistance and poor outcomes. T-ALL accounts for 20% to 30% of adult ALL, while T-LBL accounts for 3% to 4% of adult non-Hodgkin lymphomas. Currently, T-ALL and T-LBL are considered the same disease with different clinical manifestations and at different stages of progression [5]. Conventional chemotherapy forms the mainstay of initial treatment; however, sustained remission rates are low [6]. Achieving early MRD negativity is a critical prognostic factor, with recurrence rates escalating when MRD levels exceed $\geq 10^{-4}$ [7]. Allo-HSCT remains the only potentially curative treatment for R/R T-ALL/LBL, although outcomes remain suboptimal in a subset of patients; early transplantation during remission is recommended for eligible individuals with high-risk features [8,9]. For R/R T-ALL/LBL, salvage multi-agent chemotherapy offers limited benefit, underscoring the urgent need for more effective therapies [10].

Therapeutic advances for R/R T-ALL/LBL have been modest. Investigational agents include histone deacetylase (HDAC) inhibitors, EZH1/2 inhibitors, Bcl-2 inhibitors, NOTCH1 inhibitors, CD38 monoclonal antibodies, and Cladribine, yet none have yielded transformative improvements [11,12]. Inspired by the success of CAR-T therapy in B-cell malignancies, CD7-targeted CAR-T therapy has emerged as a promising strategy for T-cell malignancies, given the high prevalence of CD7 expression [13,14]. Studies have shown that CD7 CAR-T cell therapy has significant clinical advantages in the treatment of R/R T-ALL/LBL. CD7 CAR-T cells exhibit a high response rate in eliminating tumor cells, with a complete remission rate reaching 90% to 95% [15,16]. Compared to conventional chemotherapy, it offers superior efficacy and serves as an effective bridge to subsequent allo-HSCT, potentially reducing relapse risk and improving long-term survival. However, in the application of CD7 CAR-T therapy, CRS and other immune related adverse effects need to be recognized and managed promptly [17,18].

Our patient presented with widespread lymphadenopathy and high tumor burden. After initial combination chemotherapy, MRD remained > 10^{-4}, indicating poor response to conventional agents. Subsequent regimens incorporating venetoclax, azacitidine, and chidamide-selected based on BCL-2 overexpression and epigenetic regulatory changes in T-ALL [19-26] also failed to achieve MRD negativity. Given the critical importance of MRD-negative status prior to transplantation, immunotherapy was pursued. Tumor cells expressed CD7, prompting the use of donor-derived CD7 CAR-T cells [27]. Crucially, post-CAR-T bone marrow evaluation confirmed sustained MRD negativity with complete remission—a deep response unattainable with prior therapies. This enabled successful bridging allo-HSCT after myeloablative conditioning. After transplant, the patient achieved sustained donor chimerism and remained in remission throughout follow-up. This sequential strategy underscores the unique ability of CD7 CAR-T therapy to convert MRD-positive refractory disease to a transplant-eligible state. Prospective trials across the Asia-Pacific region and Western countries confirm that CD7 CAR-T therapy achieves high rates of MRD-negative remission in R/R T-ALL/LBL, and consolidative allo-HSCT substantially improves survival. A phase II donor-derived CD7 CAR-T trial reported an 89% best overall response rate and superior long-term outcomes in transplanted patients [28]. Similarly, the NS7CAR trial achieved a 94.4% deep bone marrow complete remission rate, with a 1-year PFS of 67.2% versus 15.0% in transplanted versus non-transplanted responders [29]. A Western phase I/II trial of off-the-shelf WU-CART-007 demonstrated an 72.7% composite complete remission rate [30]. These data collectively support CD7 CAR-T followed by consolidative HSCT as a viable therapeutic strategy for high-risk T-ALL/LBL.

T-ALL has multiple antigens co-expressed by tumor cells and normal T cells, which may lead to a “fratricide” phenomenon where CAR-T cells attack each other during the manufacturing

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

process, thus limiting the therapeutic effect [31]. Researchers have developed various anti-CD7 CAR-T cell products to address this challenge. Oh et al [32] used an anti-CD7 protein expression blocker (PEBL) to inhibit CAR-T cell fratricide, enabling patients to achieve a high remission rate quickly, with strong antitumor activity and good safety. Zhang et al [33] utilized nanobody-derived CD7 CAR-T cells that persisted in the body for up to 270 days and showed significant efficacy, indicating a lasting therapeutic effect. Although CAR-T cell therapy shows good efficacy, safety issues cannot be ignored. Ma [34] pointed out that severe immune deficiency and hematotoxicity after CD7 CAR-T cell therapy were the major adverse events, and were accompanied by difficulties in immune reconstitution. The vast majority of R/R T-ALL patients should undergo bridging transplantation as soon as possible after CAR-T treatment because tumor and normal T cells are cleared after CAR-T cells infusion, leaving patients in an immunodeficient state. Timely bridging transplantation after disease remission can rebuild hematopoietic and immune function and effectively eliminate CAR-T cells, avoiding the occurrence of long-term immune deficiency.

Conclusions

This case demonstrates that donor-derived CD7 CAR-T therapy effectively induces deep remission—specifically converting MRD-positive refractory disease to sustained MRD negativity—and serves as a successful bridge to allo-HSCT in R/R T-ALL/LBL. These findings provide important evidence supporting the clinical application of CD7 CAR-T therapy, highlight its unique role in achieving MRD clearance that is unattainable with conventional regimens, and underscore the necessity of integrating immunotherapy with subsequent transplantation. Despite favorable outcomes, risks such as CRS, ICANS, immune dysfunction, and prolonged cytopenias require careful management. Larger, multicenter studies are warranted to optimize protocols and explore personalized strategies.

Department and Institution Where Work Was Done

Department of Hematology, Jining No. 1 People's Hospital, Jining, Shandong, PR China.

Patient Consent Statement

Written informed consent for publication was not obtained from the patient or the relatives after all possible attempts were made. The institutional ethics committee of Jining No. 1 People's Hospital approved this publication and waived the requirement for informed consent (Approval No. 2026-IIT-110).

Availability of Data and Materials

The datasets analyzed during this study are available from the corresponding author upon reasonable request.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

References:

1. Baig MU, Rytting M, Roth M, et al. Venetoclax and decitabine in pediatric refractory T-cell lymphoblastic lymphoma. J Pediatr Hematol Oncol. 2021;43(7):e991-e96
2. Abou Dalle I, Jabbour E, Short NJ. Evaluation and management of measurable residual disease in acute lymphoblastic leukemia. Ther Adv Hematol. 2020;11:2040620720910023
3. Candoni A, Lazzarotto D, Ferrara F, et al. Nelarabine as salvage therapy and bridge to allogeneic stem cell transplant in 118 adult patients with relapsed/refractory T-cell acute lymphoblastic leukemia/lymphoma. A CAMPUS ALL study. Am J Hematol. 2020;95(12):1466-72
4. Zhou M, Yang Y, Zhang X, et al. Combination of venetoclax with CHG regimen in refractory/relapsed T-lymphoblastic lymphoma/acute lymphoblastic leukemia: A case series and literature review. Discov Oncol. 2025;16(1):1202
5. Hematological Oncology Committee of China Anti-Cancer Association, Chinese Society of Hematology, Chinese Medical Association Chinese Working Group for T Cell Lymphoma, Hematological Oncology Committee of China Anti-Cancer Association. The consensus of the diagnosis and treatment of adult T-cell lymphoblastic lymphoma in China (2023). Chin J Hematol. 2023;44(5):353-58
6. Si Lim SJ, Ford JB, Hermiston ML. How I treat newly diagnosed and refractory T-cell acute lymphoblastic lymphoma in children and young adults. Blood. 2023;141(25):3019-30
7. Petit A, Trinquand A, Chevret S, et al. Oncogenetic mutations combined with MRD improve outcome prediction in pediatric T-cell acute lymphoblastic leukemia. Blood. 2018;131(3):289-300
8. Utsunomiya A. Progress in allogeneic hematopoietic cell transplantation in adult T cell leukemia lymphoma. Front Microbiol. 2019;10:2235
9. Inoue Y, Fuji S, Tanosaki R, et al. Prognostic importance of pretransplant disease status for post-transplant outcomes in patients with adult T cell leukemia/lymphom. Bone Marrow Transplant. 2018;53(9):1105-15
10. Gökbuget N, Stanze D, Beck J, et al. Outcome of relapsed adult lymphoblastic leukemia depends on response to salvage chemotherapy, prognostic factors, and performance of stem cell transplantation. Blood. 2012;120(10):2032-41
11. Utsunomiya A, Izutsu K, Jo T, et al. Oral histone deacetylase inhibitor tucidinostat (HBI 8000) in patients with relapsed or refractory adult T cell leukemia/lymphoma: Phase IIb results. Cancer Sci. 2022;113(8):2778 -87
12. Stuver R, Horwitz SM, Epstein Peterson ZD. Treatment of adult T cell leukemia/lymphoma: Established paradigms and emerging directions. Curr Treat Options Oncol. 2023;24(8):948-64
13. Semchenkova A, Mikhailova E, Demina I, et al. Analysis of antigen expression in T-cell acute lymphoblastic leukemia by multicolor flow cytometry: Implications for the detection of measurable residual disease. Int J Mol Sci. 2025;26(5):2002
14. Huang YH, Wan CL, Dai HP, Xue SL. Targeted therapy and immunotherapy for T cell acute lymphoblastic leukemia/lymphoma. Ann Hematol. 2023;102(8):2001-13
15. Visser N, González-Corrales M, Álvarez-Freile J, et al. K12-ligand-based CAR T cell therapy for CD7-positive T cell malignancies. Mol Ther Oncol. 2025;33(2):200988

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

16. Pan J, Tan Y, Wang G, et al. Donor-derived CD7 chimeric antigen receptor T cells for T-cell acute lymphoblastic leukemia: First-in-human, phase I trial. J Clin Oncol. 2021;39(30):3340-51
17. Delgado J. InsT-ALLing CD7 chimeric antigen receptors before transplantation. Br J Haematol. 2024;204(6):2155-56
18. Xiao J, Liu Y, Shen Y, et al. Clinical outcomes of CD7 CAR-T cell therapy in relapsed or refractory T-cell acute lymphoblastic leukemia and lymphoblastic lymphoma patients. Hematol Oncol. 2025;43(3):e70081
19. Richard-Carpentier G, Jabbour E, Short NJ, et al. Clinical experience with venetoclax combined with chemotherapy for relapsed or refractory T-cell acute lymphoblastic Leukemia. Clin Lymphoma Myeloma Leuk. 2020;20(4):212-18
20. Arora S, Vachhani P, Bachiashvili K, Jamy O. Venetoclax with chemotherapy inrelapse/refractory early T-cell precursor acute lymphoblastic leukemia. Leuk Lymphoma. 2021;62(9):2292-94
21. Kong J, Chen N, Li M, et al. Venetoclax and decitabine in refractory TP53-mutated early T-cell precursor acute lymphoblastic leukemia. Ann Hematol. 2022;101(3):697-99
22. Wan CL, Zou JY, Qiao M, et al. Venetoclax combined with azacitidine as an effective and safe salvage regimen for relapsed or refractory T-cell acute lymphoblastic leukemia: A case series. Leuk Lymphoma. 2021;62(13):3300-3
23. Cao HY, Zhang H, Zhang Y, et al. Venetoclax plus azacitidine in relapsed or refractory T-cell acute lymphoblastic leukaemia: A multicentre, single-arm, phase 2 trial. Lancet Haematol. 2025;12(12):e946-e55
24. Chi Z, Gao H, Liu H, et al. Chidamide induces necroptosis via regulation of c-FLIPL expression in Jurkat and HUT-78 cells. Mol Med Rep. 2020;21(2):936-44
25. Xi M, Guo S, Bayin C, et al. Chidamide inhibits the NOTCH1-MYC signaling axis in T-cell acute lymphoblastic leukemia. Front Med. 2022;16(3):442-58
26. Guan W, Jing Y, Dou L, et al. Chidamide in combination with chemotherapy in refractory and relapsed T lymphoblastic lymphoma/leukemia. Leuk Lymphoma. 2020;61(4):855-61
27. Hu Y, Zhang M, Yang T, et al. Sequential CD7 CAR T-cell therapy and allogeneic HSCT without GVHD Prophylaxis. N Engl J Med. 2024;390(16):1467-80
28. Pan J, Zhao L, Zhang Y, et al. Donor-derived CD7 CAR T cells for pediatric and adult relapsed/refractory T-ALL/LBL: A phase 2 trial. Blood. 2025;146(23):2745-57
29. Zhang X, Yang J, Li J, et al. Analysis of 60 patients with relapsed or refractory T-cell acute lymphoblastic leukemia and T-cell lymphoblastic lymphoma treated with CD7-targeted chimeric antigen receptor-T cell therapy. Am J Hematol. 2023;98(12):1898-908
30. Ghobadi A, Aldoss I, Maude SL, et al. Phase 1/2 trial of anti-CD7 allogeneic WU-CART-007 for patients with relapsed/refractory T-cell malignancies. Blood. 2025;146(10):1163-73
31. Oh BLZ, Vinanica N, Wong DMH, et al. Chimeric antigen receptor T-cell therapy for T-cell acute lymphoblastic leukemia. Haematologica. 2024;109(6):1677-88
32. Oh BLZ, Shimasaki N, Coustan-Smith E, et al. Fratricide-resistant CD7-CAR T cells in T-ALL. Nat Med. 2024;30(12):3687-96
33. Zhang M, Chen D, Fu X, et al. Autologous nanobody-derived fratricide-resistant CD7-CAR T-cell therapy for patients with relapsed and refractory T-cell acute lymphoblastic leukemia/lymphoma. Clin Cancer Res. 2022;28(13):2830-43
34. Ma JF, Yan CL, Jia X, et al. Clinical outcomes and safety of CAR-T cells in treatment of T-Cell acute lymphoblastic leukemia/lymphoma. Ann Hematol. 2025;104(1):57-63

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]