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Teclistamab for BCMA-positive relapsed/refractory B acute lymphoblastic leukemia

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Running head: Teclistamab for ALL

Relapsed B-cell acute lymphoblastic leukemia (B-ALL) remains a major therapeutic challenge. Despite the success of chimeric antigen receptor T-cell (CAR T-cell) therapy and bispecific T-cell engagers, many patients eventually relapse through the loss of target antigens such as CD19 and CD22. This highlights the need to identify alternative targets and develop novel therapeutic approaches.In addition to multiple myeloma, B-cell maturation antigen (BCMA) is expressed on malignant cells in several hematologic malignancies, including chronic lymphocytic leukemia, non-Hodgkin lymphoma, ALL, Hodgkin lymphoma, Waldenstrom macroglobulinemia, and acute myeloid leukemia[1]. In a study evaluating BCMA expression by flow cytometry in patients with B-ALL (n=23) and cell lines (n=6), BCMA was detected in a subset of patients (mean percentage of positive cells 26.6%, range 1.3–96.7%), whereas all cell lines were negative (Maia S, PlosOne2011). However, clinical experience with BCMA-directed therapies outside of multiple myeloma remains limited. A phase I study is currently evaluating a dual CD19/BCMA-targeted CAR T-cell therapy in patients with relapsed/refractory aggressive B-cell Lymphoma[2]. To our knowledge, BCMA-directed therapy has not previously been reported in ALL.

Ph-like B-ALL is characterized by a gene expression profile similar to that of Philadelphia chromosome-positive ALL but lacks the BCR::ABL1 fusion. It is often associated with a poor prognosis and resistance to conventional chemotherapy.

A 45-year-old male presented with a ninth relapse of Ph-like B-ALL characterized by an EPOR rearrangement, after exhausting all approved therapeutic options for B-ALL. His clinical course was marked by extreme chemoresistance and multiple relapses. This case report was conducted in accordance with the ethical requirements applicable in Israel.

He was diagnosed in June 2015 at the age of 35. Initial treatment followed a pediatric-inspired protocol, leading to a first matched unrelated donor allogeneic hematopoietic cell transplantation (MUD allo-HCT) in 2016. Following a relapse in 2017, he failed to respond to blinatumomab but achieved remission with inotuzumab ozogamicin, which served as a bridge to CD19-targeted CAR T-cell therapy in early 2018, followed by a second MUD allo-HCT from a different donor.

The disease relapsed again in late 2018 with CD19-negative, CD22-positive leukemic blasts.Mini-Hyper-CVAD plus inotuzumab ozogamicin was initiated but discontinued because of severe neutropenia. Measurable residual disease (MRD) negativity was subsequently achieved and maintained with a combination of vincristine, asparaginase, venetoclax, and navitoclax. This regimen was continued for 27 months before being discontinued because of persistent thrombocytopenia. In late 2021, a rising MRD level was treated with donor lymphocyte infusion.

In January 2022, the patient relapsed again. Remission was achieved with vincristine, bortezomib, and peg-asparaginase, followed by a third allo-HCT from his haploidentical sister in June 2022. This resulted in a remission lasting approximately two years. In April 2024, the patient relapsed and received

vincristine, bortezomib, and peg-asparaginase. Because of severe polyneuropathy, therapy was subsequently switched to gemcitabine plus polatuzumab vedotin, leading to a complete remission with persistent MRD positivity. Dasatinib maintenance therapy, administered to inhibit presumed ABL-pathway activation, failed to eradicate low-level MRD.

By late 2024, while the patient was receiving ruxolitinib to control mild chronic GVHD, he developed progressive disease. Flow cytometry revealed negative expression of CD10, CD19, CD20, CD22, CD56, CD117 and CD38 and positive for CD34 and HLA-DR and TdT. At that stage, a bone marrow sample was sent for a dedicated flow-cytometric assessment of BCMA expression, which was weakly positive.. Given the lack of remaining conventional therapeutic options and the documented BCMA expression, subcutaneous Teclistamab, a BCMA-directed CD3 T-cell engager, was administered on a compassionate-use basis.

Treatment with Teclistamab was initiated in January 2025. Remarkably, after only four doses, the patient achieved a morphologic remission in the bone marrow, accompanied by significant clinical improvement and recovery of peripheral blood counts (WBC 8.3 × 10^9/L, ANC 5.5 × 10^9/L, hemoglobin 11.8 g/dL, and platelet count 337 × 10^9/L). However, MRD remained positive. This response was achieved in a heavily pretreated setting in which prior immunotherapies and three previous transplants had failed. The patient remained in remission for several months before a subsequent relapse was documented in May 2025. No cytokine release syndrome, immune effector cell-associated neurotoxicity syndrome, or other Teclistamab-specific toxicities were observed. During treatment, the patient developed grade 4 sepsis, from which he subsequently recovered. In March 2026, the patient died from sepsis in the setting of active disease (Figure 1).

This case demonstrates the potential activity of Teclistamab as a salvage strategy for relapsed/refractory B-ALL. Although BCMA is traditionally regarded as a plasma cell marker, its expression on leukemic blasts in this patient provided a therapeutic target after failure of both CD19- and CD22-directed therapies. The rapid achievement of morphologic remission after only four doses of Teclistamab suggests that BCMA-directed T-cell engagement may retain activity even in highly refractory Ph-like ALL.

Although the response was temporary, this report provides preliminary clinical evidence supporting the potential role of BCMA-targeted therapy in ALL. The case highlights the importance of comprehensive immunophenotyping in heavily pretreated patients, after conventional treatment options have been exhausted,the as identification of non-canonical targets such as BCMA may reveal otherwise unavailable therapeutic options. Further evaluation of BCMA-directed approaches in relapsed/ refractory B-ALL is warranted.

References:

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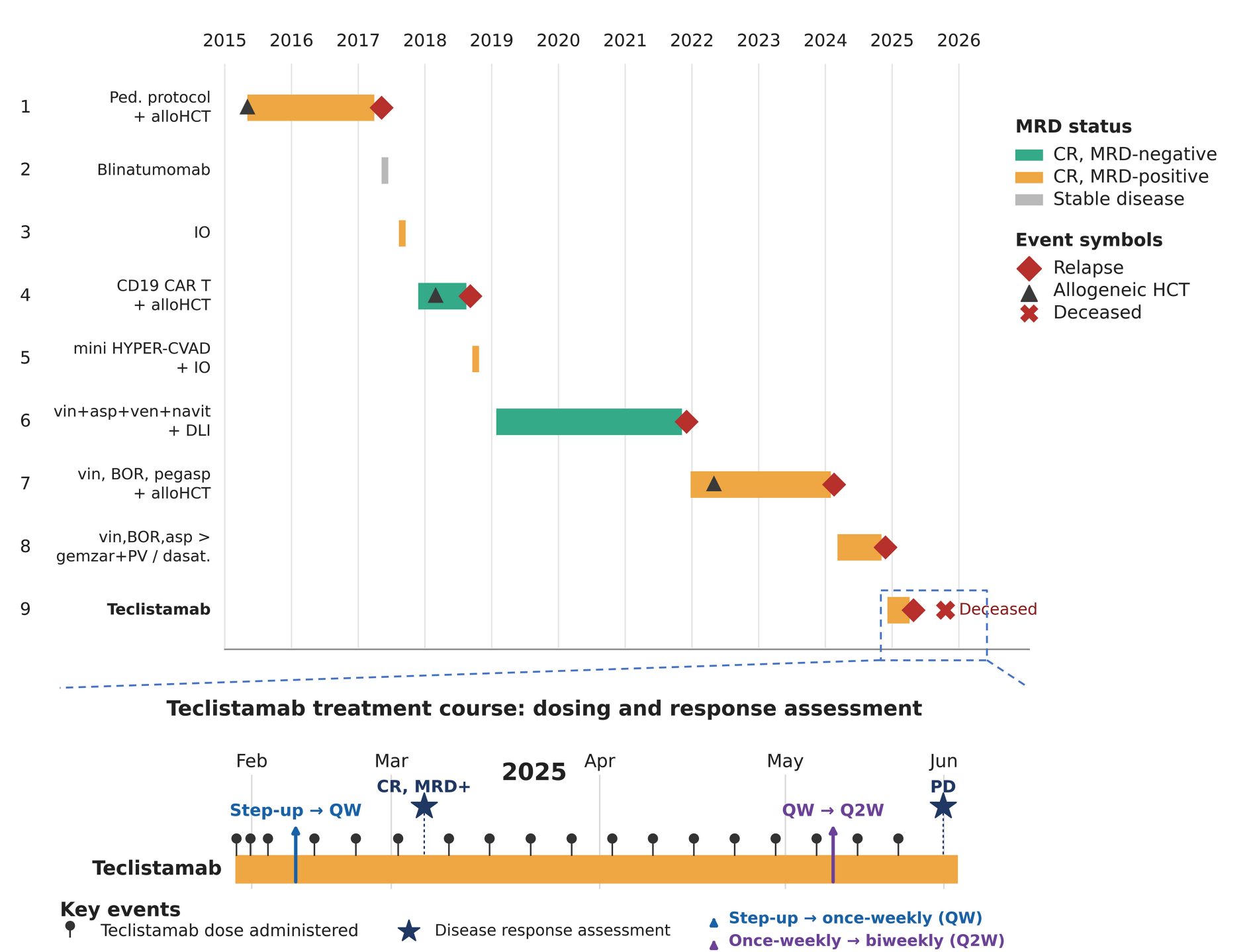
Legends:

Figure 1. Timeline of the patient's treatment course.

Abbreviations:
alloHCT: allogeneic hematopoietic stem cell transplantation
asp: asparaginase
BOR: bortezomib
CAR T: chimeric antigen receptor T-cell
CR: complete response
dasat: dasatinib
DLI: donor lymphocyte infusion
IO: inotuzumab ozogamicin
MRD: measurable residual disease
navit: navitoclax
Ped: pediatric
pegasp: pegasparaginase
PV: polatuzumab vedotin
SD: stable disease
ven: venetoclax
vin: vincristine


2015
2016
2017
2018
2019
2020
2021
2022
2023
2024
2025
2026
1
Ped. protocol
+ alloHCT
2
Blinatumomab
3
IO
4
CD19 CAR T
+ alloHCT
5
mini HYPER-CVAD
+ IO
6
vin+asp+ven+navit
+ DLI
7
vin, BOR, pegasp
+ alloHCT
8
vin,BOR,asp >
gemzar+PV / dasat.
9
Teclistamab
Deceased
MRD status
CR, MRD-negative
CR, MRD-positive
Stable disease
Event symbols
Relapse
Allogeneic HCT
Deceased
Teclistamab treatment course: dosing and response assessment
Feb
Mar
2025
Apr
May
Jun
CR, MRD+
PD
Step-up → QW
QW → Q2W
Teclistamab
Key events
Teclistamab dose administered
Disease response assessment
Step-up → once-weekly (QW)
Once-weekly → biweekly (Q2W)