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Review Article

Bispecific T-Cell Engager Therapy in Relapsed Acute Lymphoblastic Leukemia: A Review of Published Cohorts

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Summary

A bispecific antibody that redirects the patient's own T cells against CD19 doubled complete remission rates against chemotherapy in relapsed disease. The more useful finding is buried in the burden data: the same agent clears residual disease in four fifths of patients but produces remission in fewer than half of those with overt marrow involvement.

Objectives: To review the published evidence on bispecific T-cell engager therapy in relapsed and refractory B-cell precursor acute lymphoblastic leukaemia: what the randomised comparison established, how response varies with disease burden, what is achieved in the molecular disease setting, what toxicity occurs in routine use, and how the agent should be sequenced.

Material and Methods: Narrative review of randomised trials, single-arm registration studies, real-world multicentre series and regulatory assessments indexed in PubMed and the major haematology journals. Studies were eligible if they reported efficacy or toxicity of bispecific T-cell engager therapy in a defined B-cell precursor acute lymphoblastic leukaemia population. No new patient data were generated and no patients were recruited; every estimate quoted is one published by the original investigators.

Results: In the randomised comparison against standard-of-care chemotherapy in Philadelphia-negative relapsed or refractory disease, median overall survival was 7.7 against 4.0 months (HR 0.71, $p = 0.012$) and complete remission 34 against 16 per cent. In Philadelphia-positive disease, a single-arm study reported complete remission in 31 per cent. An earlier phase II study achieved complete remission or complete remission with partial haematological recovery in 43 per cent after two cycles, with median relapse-free survival of 5.9 months and overall survival of 6.1 months. Where only molecular disease remained, 16 of 21 patients cleared residual disease, 12 of those having been molecularly refractory to prior chemotherapy, with relapse-free survival of 78 per cent at a median follow-up of 405 days. Across 39 children and adolescents treated in routine practice, complete remission reached 46 per cent among those with 5 per cent or more blasts against 81 per cent residual disease clearance among those below that threshold, with grade 3 or higher events in 34.8 per cent, no cytokine release syndrome and no toxic deaths.

Conclusion: Efficacy scales inversely with disease burden, and the licensed indication describes the setting in which the agent performs least well. Median survival of 7.7 months in overt relapse is a real improvement over 4.0 and remains under a year.

Abbreviations: ALL – Acute Lymphoblastic Leukaemia, BCP-ALL – B-Cell Precursor ALL, BiTE – Bispecific T-Cell Engager, R/R – Relapsed or Refractory, Ph – Philadelphia Chromosome, CR – Complete Remission, CRh – Complete Remission with Partial Haematological Recovery, MRD – Measurable Residual Disease, RFS – Relapse-Free Survival, OS – Overall Survival, CRS – Cytokine Release Syndrome, allo-HSCT – Allogeneic Haematopoietic Stem Cell Transplantation, CAR – Chimeric Antigen Receptor.

Keywords: *Acute Lymphoblastic Leukaemia; Blinatumomab; Bispecific T-Cell Engager; Measurable Residual Disease; Relapsed Disease*

Introduction

Front-line therapy cures the large majority of children with B-cell precursor acute lymphoblastic leukaemia, with five-year survival above 90 per cent under current risk-adapted protocols, yet 15 to 20 per cent still relapse [1,2]. Five-year survival after first relapse is around 50 per

cent, and primary refractory disease or second relapse carries a dismal outlook even with intensive chemotherapy and transplantation [3,4].

Adults fare considerably worse. Outcome after relapse depends on response to salvage chemotherapy and on whether transplantation can be performed [5], and conventional salvage in a heavily pretreated patient rarely produces durable benefit [6,7].

Bispecific T-cell engagers address this by a different mechanism. A single molecule carrying two single-chain variable fragments binds CD3 on T cells and CD19 on B-lineage cells simultaneously, forming a synapse that redirects cytotoxic T cells against the leukaemic clone without requiring antigen presentation [8,9]. The approach needs no cell manufacture and no lymphodepletion [10].

This review sets out what the randomised evidence established, how response varies with the amount of disease present, what is achieved when only molecular disease remains, what toxicity occurs outside trials, and where the agent sits among the alternatives.

Material and Methods

Search strategy: PubMed and the archives of the major haematology journals were searched for studies of bispecific T-cell engager therapy in lymphoblastic leukaemia. Search terms combined acute lymphoblastic leukaemia with blinatumomab, bispecific T-cell engager, CD19-directed, relapsed refractory, measurable residual disease and real-world outcomes. Reference lists of retrieved trials and regulatory assessments were screened by hand.

Eligibility: Studies were included if they reported efficacy or toxicity outcomes for bispecific T-cell engager therapy in a defined B-cell precursor acute lymphoblastic leukaemia population. Studies of chimeric antigen receptor T-cell therapy are cited where they bear on sequencing decisions and are identified as such.

Data extraction and handling: Design, cohort size, disease burden at entry, response criteria and reported outcomes were extracted as published. Disease burden at entry is stated alongside every response figure, because the central finding of this review is that response varies with it and figures from different settings are not comparable. No pooling or re-analysis was performed and no patient-level data were obtained. Figures 1 to 4 and Tables 1 to 3 reproduce published values only. This is a narrative synthesis and was not registered as a systematic review.

Results

What the randomised trial established: A phase III trial randomised patients with primary refractory or relapsed Philadelphia-negative disease to continuous-infusion bispecific antibody, given in four-week cycles followed by two weeks off, or to investigator's choice of standard chemotherapy [11,12]. Median overall survival was 7.7 months against 4.0 months, with a hazard

ratio of 0.71 ($p = 0.012$), and complete remission was achieved in 34 against 16 per cent (Figure 1) [11,13].

This was the first immunotherapy to demonstrate an overall survival benefit in this population [12,13], and the finding supported conversion from accelerated to regular approval [13]. A parallel single-arm study in Philadelphia-positive disease reported complete remission in 31 per cent with a comparable adverse event profile, broadening the licensed population to both [13].

The earlier phase II study that led to these trials enrolled patients with early relapse, defined as within twelve months of first remission or of transplantation, or failure of salvage. After two cycles, 43 per cent achieved complete remission or complete remission with partial haematological recovery; among responders median relapse-free survival was 5.9 months (95% CI 4.8-8.3) and median overall survival 6.1 months (95% CI 4.2-7.5) [14].

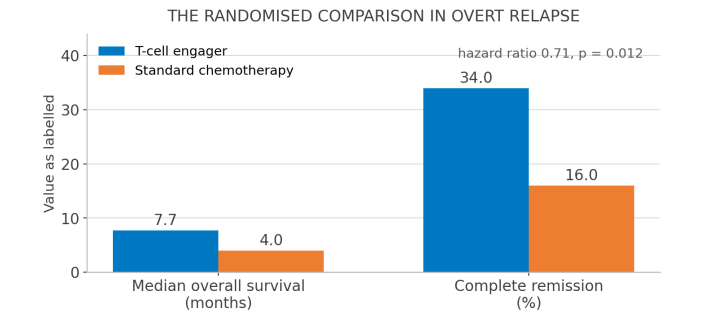


Figure 1: Survival and remission in the randomised comparison against chemotherapy [11,13]

Study	n	Setting	Principal reported finding
Topp et al. 2011 [15]	21	Molecular disease	16 cleared residual disease; 78% relapse-free
Phase II salvage [14]	—	Early relapse	43% remission after two cycles
Kantarjian et al. 2017 [11]	Randomised	Ph-negative relapse	Survival 7.7 against 4.0 months; HR 0.71
Martinelli et al. 2017 [16]	Single arm	Ph-positive relapse	Complete remission in 31 per cent
von Stackelberg et al. 2016 [17]	Phase I/II	Paediatric relapse	Dose and activity established in children
AIEOP series [18]	39	Routine paediatric use	Response 46% or 81% by disease burden

Table 1: Principal studies reviewed, with setting and the finding as reported by the original investigators

Response scales inversely with disease burden: The clearest demonstration comes from a single real-world cohort in which both settings were treated. Thirty-nine patients aged 0 to 21 with relapsed

or refractory disease received the agent across seven centres between 2015 and 2020, thirteen with the aim of inducing remission and twenty-six with the aim of reducing residual disease [18].

Among the thirteen patients with 5 per cent or more marrow blasts, complete remission was achieved in 46 per cent. Among the twenty-six with fewer than 5 per cent blasts, 81 per cent achieved residual disease negativity by polymerase chain reaction or flow cytometry (Figure 2) [18]. The same drug, in the same cohort, performed almost twice as well against the smaller disease burden.

That gradient is mechanistically coherent. The agent depends on the patient's own effector T cells forming synapses with target cells, and a high blast burden both dilutes the effector-to-target ratio and increases the cytokine load released on lysis [8,9,19]. Where few cells remain, the available T-cell compartment is sufficient.

The two groups were not randomly assigned. Patients received the agent with the aim of inducing remission or of reducing residual disease according to clinical judgement, so the comparison is between two populations that differed in more than blast count [18]. The gradient is nonetheless consistent with what the molecular disease studies report independently [15,23].

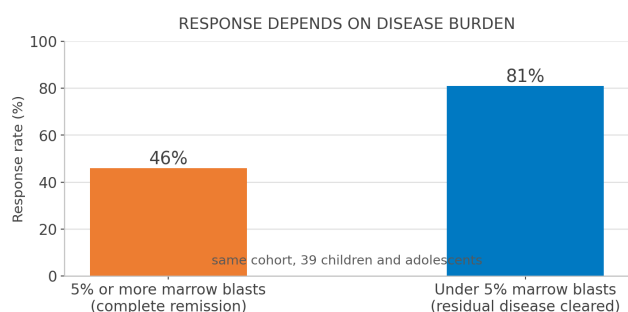


Figure 2: Response by marrow blast percentage within a single cohort of 39 patients [18]

What is achieved against molecular disease alone: The phase II study in this setting treated 21 adults with persistent or recurrent residual disease after induction and consolidation, using a four-week continuous infusion. Sixteen became residual disease negative as assessed by quantitative polymerase chain reaction for immunoglobulin or T-cell receptor rearrangements or for specific genetic aberrations [15].

Two features of that result deserve emphasis. Twelve of the sixteen responders had been molecularly refractory to previous chemotherapy, so the agent cleared disease that cytotoxic treatment could not [15]. And probability of relapse-free survival was 78 per cent at a median follow-up of 405 days (Figure 3) [15], which is a different order of durability from the 5.9 months seen in overt relapse [14].

Residual disease is the dominant prognostic variable in this disease [20,21], and an agent capable of converting a positive result

to a negative one addresses the variable that matters most [22]. That logic supported extension of the licence into the molecular disease setting [15,23].

Whether that durability reflects the agent or the population is not settled by a single-arm study. Patients with molecular disease alone have by definition responded to prior therapy sufficiently to reach that state, so they differ from patients in overt relapse in ways beyond burden [15,20]. The finding that most responders were molecularly refractory to chemotherapy nonetheless argues against pure selection [15].

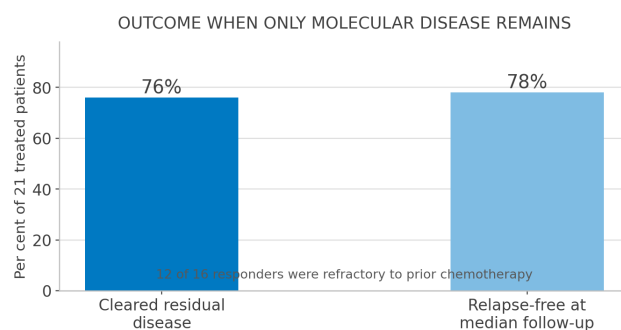


Figure 3: Outcome in 21 patients treated for molecular disease alone [15]

Toxicity in routine use: The characteristic toxicities are cytokine release syndrome and neurological events, the latter including tremor, encephalopathy, peripheral neuropathy and depression, all observed more frequently than with chemotherapy [13]. Depression emerged as a rare but potentially severe neurological event [13].

Real-world paediatric experience was more favourable than trial reporting would suggest. Across the 39-patient series, 34.8 per cent of adverse events were grade 3 or higher, no cytokine release syndrome occurred and there were no treatment-related deaths (Figure 4) [18]. That profile reflects a population treated largely for low disease burden, which is consistent with burden driving cytokine release [8,18].

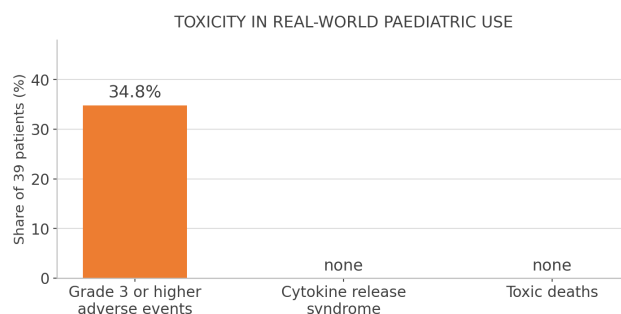


Figure 4: Toxicity across 39 children and adolescents treated in routine practice [18]

Setting	Reported response	Durability
Molecular disease only	16 of 21 cleared	78% relapse-free at 405 days [15]
Under 5% marrow blasts	81% cleared	Not separately reported [18]
5% or more marrow blasts	46% remission	Not separately reported [18]
Overt relapse, randomised	34% remission	Median survival 7.7 months [11]
Early relapse, phase II	43% remission	Median relapse-free 5.9 months [14]

Table 2: Reported response and durability by disease burden at treatment

Moving the agent earlier: If efficacy scales inversely with burden, the logical response is to use the agent before burden accumulates, and two randomised trials tested exactly that in first relapse. Consolidation with the bispecific agent rather than chemotherapy after reinduction improved disease-free survival in children, adolescents and young adults [29], and a parallel trial reported improved event-free survival in high-risk first relapse [28].

Both trials placed the agent where residual disease rather than overt leukaemia was the target, which is the setting the burden gradient predicts it should work best [15,18]. Both also used it as a bridge to transplantation rather than as definitive therapy [28,29].

Age and lineage biology shape how these results transfer. Adolescents and young adults occupy a boundary between paediatric and adult protocols [31,32], and the genetic landscape of the disease differs materially between children and adults [30], so a result established in one age group should not be assumed in the other.

Trial setting	Comparison	Reported outcome
Molecular disease after consolidation	Single arm	16 of 21 cleared residual disease [15]
High-risk first relapse, paediatric	Against chemotherapy	Improved event-free survival [28]
First relapse consolidation	Against chemotherapy	Improved disease-free survival [29]
Relapsed or refractory, adult	Against chemotherapy	Survival 7.7 against 4.0 months [11]

Table 3: Where the agent has been tested, and what each setting reported

Discussion

The randomised result is genuine and modest. Median survival of 7.7 months against 4.0 is a hazard ratio of 0.71 in a population with almost no other options [11], and it was the first immunotherapy to show a survival benefit here [12]. It is also less than a year.

The burden data suggest why. In one cohort treated at a single set of centres, response was 46 per cent above the 5 per cent blast

threshold and 81 per cent below it [18]. In the molecular disease setting, 16 of 21 patients cleared and 78 per cent remained relapse-free beyond a year [15]. The licensed indication for relapsed disease describes the setting in which the agent works least well.

Why burden should matter mechanistically

The molecule works by bringing an effector T cell into contact with a target cell [8,9]. Two consequences follow from a high blast burden. The ratio of available effectors to targets falls, and the cytokine released on lysis rises with the number of cells killed, which is the proximate cause of the syndrome that limits dosing [8,19].

The absence of cytokine release syndrome in a real-world cohort treated predominantly for low burden supports that reading [18], as does the standard practice of stepped dosing during the first cycle [10,13]. If burden is the constraint, then debulking before treatment is a rational strategy rather than a delay.

Sequencing against cellular therapy

Both this agent and CD19-directed chimeric antigen receptor T cells target the same antigen, and the order in which they are given is unresolved [24,25]. Whether prior exposure to one compromises the other through competitive inhibition, antigen escape or T-cell exhaustion has been raised as an open question rather than answered [26].

Case-level evidence exists in both directions. Remission has been reported with the bispecific agent after failure of cellular therapy [26], and the bispecific agent is widely used as a bridge to cellular therapy or to transplantation because it does not require manufacture time [10,24]. Antigen loss remains the shared vulnerability of both approaches [25,27].

Study limitations

The limitations belong to the underlying literature. The randomised trial enrolled Philadelphia-negative adults, and the Philadelphia-positive evidence is single-arm [11,13,16]. Response criteria differ between studies, with complete remission, complete remission with partial recovery, and molecular clearance all reported as response [14,15,18]. The burden comparison derives from one retrospective series of 39 patients treated in a compassionate or off-label setting, in which allocation to the two aims was by clinician judgement rather than randomisation [18]. Real-world cohorts exclude patients enrolled in trials, so they are selected in ways that are not documented [18]. And follow-up in the molecular disease study reached a median of 405 days, which is short relative to the relapse risk in this population [15,20].

Two gaps deserve naming. No trial has randomised treatment by disease burden, so the recommendation to debulk before treatment rests on observed gradients rather than on comparison [15,18]. And no study reviewed here establishes the optimal sequence relative to

cellular therapy, despite both targeting the same antigen [24,26].

Conclusion

Bispecific T-cell engager therapy improved median overall survival from 4.0 to 7.7 months and doubled complete remission from 16 to 34 per cent against chemotherapy in relapsed or refractory B-cell precursor acute lymphoblastic leukaemia, the first immunotherapy to show a survival benefit in this setting. Efficacy scales inversely with disease burden: in one cohort, 46 per cent achieved remission with 5 per cent or more marrow blasts against 81 per cent clearing residual disease below that threshold, and where only molecular disease remained 16 of 21 patients cleared, 12 of them refractory to prior chemotherapy, with 78 per cent relapse-free beyond a year. Toxicity in routine paediatric use was modest, with no cytokine release syndrome and no toxic deaths in a low-burden population. The agent performs best where least disease remains, which is not the setting its principal indication describes. Randomised trials moving it into first relapse consolidation, where residual disease rather than overt leukaemia is the target, report improved event-free and disease-free survival, which is what the burden gradient would predict [28,29].

The practical inference for a patient in overt relapse is that reducing burden before treatment is a rational step rather than a delay, though no trial has randomised that question [15,18]. And the sequence relative to cellular therapy, which targets the same antigen, remains unresolved despite both being in routine use [24,26].

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Conflict of Interest

The authors declare no competing interests.

Ethics Statement

This article reviews previously published studies only. No new patient data were collected and no participants were recruited, so ethical approval was not required.

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