

Bispecific Antibodies and CAR-T cell Therapy in Multiple Myeloma

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Abstract

Triple class exposed patients with multiple myeloma (MM) represent an unmet need with outcomes remaining poor with those with relapsed/refractory multiple myeloma (RRMM) having a mere 29.8% overall response rate and 12.4-month median overall survival. We explore emerging strategies focusing on bispecific antibodies and CAR-T cell therapies for treatment of refractory myeloma.

Talquetamab (Talq), is a bispecific antibody directed against GPRC5D which has been approved for patients with refractory/relapsed MM who have progressed on at least 4 prior lines of therapy. Patients were treated with Talquetamab as bridge therapy, 77% were treated every 2 weeks and 23% every 4 weeks. Ultimately 119 patients (89%) successfully proceeded to CAR T cell therapy. Teclistamab is the first bispecific antibody designed to redirect T cells by binding CD3 on T-lymphocytes and B-cell maturation antigen (BCMA) on myeloma cells. Most patients who initiated Teclistamab in the outpatient, (54.2%) completed step up dosing using the 3-day dose interval. Elranatamab (PF-06863135) is a humanized bispecific antibody designed to target myeloma cells by simultaneously binding both CD3 receptors T cells and BCMA (on myeloma cells) at which T cells induce a cytotoxic T- cell response. The phase 2 MagnetisMM-3 trial reached its primary endpoint with a confirmed objective response rate (ORR) of 61.0% including complete response of 35%.

The landscape of relapsed/refractory multiple myeloma (RRMM) treatment has been revolutionized by bispecific antibodies and CAR-T cell therapies, offering unprecedented efficacy for heavily pretreated patients.

Keywords: Bispecific antibodies, Chimeric antigen receptor T-cell therapy, Multiple myeloma, Stem cell transplantation

Introduction

Multiple myeloma (MM) represents the second most common hematologic cancer in adults, with increasing rates reported over recent years. As per the SEER database the number of incident cases between 2014–2018 was 47,966 [1]. Introduction of agents such as proteasome inhibitors (PIs), anti-CD38 therapy, and immune modulators (IMiDs) in various combinations has improved progression free survival and overall survival for patients with multiple myeloma who are no longer candidates for autologous transplantation. Patients who are fit for autologous transplantation (ASCT) or CAR-T cell therapy are also treated with the various combined therapies for three to six months as a bridge prior to ASCT or CAR-T cells.

Despite these advances, MM remains incurable which is largely attributed to the genetic and epigenetic evolution of

malignant plasma cells wherein copy number modifications accumulate, secondary mutations are acquired, and epigenetic changes occur. These occurrences create intratumor heterogeneity, resulting in multiple clonal subpopulations with various resistance mechanisms [2].

Patients whose disease becomes refractory after prior treatment with anti-CD38 monoclonal antibodies, two proteasome inhibitors (PIs), and two immunomodulatory drugs (IMiDs) generally have poor outcomes [3]. Fortunately, treatment options have expanded with the promising emergence of bispecific antibodies that redirect physical targeting of cytotoxic effector T cells toward myeloma cells. Through this process, these bispecific antibodies act as dual antigen targeting constructs between these T cells and myeloma cells including B-cell membrane antigen (BCMA), G-protein-coupled receptor family C group 5 member D

(GPRC5D), and Fc receptor-homolog 5 (FcRH5) [4]. These agents have been proven to induce deep and durable responses in patients who have been heavily pre-treated.

BCMA is a cell membrane type III non-tyrosine kinase receptor glycoprotein [5,6]. Its N-terminal section receptor contains six motifs which categorizes BCMA as part of the tumor necrosis factor receptor superfamily 17 (TNFRSF17)/CD269 [7]. Members of the TNF and TNF receptor family are vital in immune function [8]. These proteins are solely present on plasma blasts and plasma cells (PC) which make them attractive therapeutic targets. In contrast, G protein-coupled receptor, class C group 5 member D (GPRC5D) serves as a type C 7-pass transmembrane receptor protein, and is expressed at significantly higher level, at least 500-fold greater, on both malignant and normal plasma cells in the bone marrow compared to circulating plasma cells. Among normal tissues, only hair follicles express GPRC5D [9,10].

Triple class exposed patients represent an unmet need with outcomes remaining poor with those with relapsed/refractory multiple myeloma (RRMM) having a mere 29.8% overall response rate and 12.4-month median overall survival [11].

In this review, we explore emerging strategies focusing on bispecific antibodies and CAR-T cell therapies for treatment of refractory myeloma. We also provide an overview of development as well as a discussion of mechanisms of action.

Talquetamab (Talq)

Talquetamab (Talq), is a bispecific antibody directed against GPRC5D which has been approved for patients with refractory/relapsed MM who have progressed on at least 4 prior lines of

therapy [12]. The use of Talq is based on a pivotal study that showed 70% overall response rate (ORR) and 10.2 months median duration of response (DOR) (MonumentAL-1) [12]. Talquetamab is associated with side effects such as dysgeusia (60%), weight loss (30%) and skin changes (70%).

How does Talquetamab fit in treatment sequencing?

We suggest Talquetamab as a therapeutic bridge to CAR-T therapy because it can achieve significant disease debulking prior to BCMA CAR-T and provides an opportunity for dual targeting.

Safety and efficacy if Talquetamab as bridging therapy was studied in a retrospective multi-institutional cohort of 134 patients of which 43% had extramedullary disease, 45% had high risk cytogenetics, 10% had prior BCMA therapy, and patients had received a median of 5 prior treatment lines [13]. For patients treated with Talquetamab as bridge therapy, 77% were treated every 2 weeks and 23% every 4 weeks. Ultimately, 119 patients (89%) successfully proceeded to CART-cell therapy. The distribution of CAR-T therapies included idecabtagene vicleucel (Ide-cel) in 18% and ciltacabtagene autoleucel (Cilta-cel) in 82%.

Overall, the data suggests manageable toxicities with bridging therapy that allows most patients to proceed to CAR-T. In patients who received Talq as a bridge to CAR-T, 49% of patients had a deep response, 46% had unchanged response. Median PFS was 13 months (IQR 10-NE). Median follow up time from Talquetamab first dose was 7.5 months (IQR 5.4-10.4). Following CAR-T median follow up was 4.9 months (IQR 3.6-6.9). Main toxicities are summarized below in

Table 2.

Table 1. Currently approved bispecific antibodies with trial data.

Drug	Target	Pivotal Trial	Overall Response Rate (ORR)	Median Duration of Response (DOR)	Notable Toxicities
Talquetamab	GPRC5D	MonumentAL-1 [12]	70%	10.2 mo	CRS (78%), dysgeusia, skin/nail changes
Teclistamab	BCMA	MajesTEC-1 [14]	63%	18.4 mo	CRS (72%), neurotoxicity, infections
Elranatamab	BCMA	MagnetisMM-3 [17,18]	61%	Not reached by 14 months follow up	CRS (58%), cytopenias, infections
Linvoseltamab	BCMA	LINKER-MM1 [19]	71%	Data maturing	CRS, cytopenias, infections

Table 2. Toxicities for Talquetamab and CAR-T.

Toxicity	Talquetamab (Talq)	CAR-T
Cytokine release syndrome (CRS)	68% (grade 1–2 only; no grade ≥3 reported)	72%
Immune effector cell-associated neurotoxicity (ICANS)	10%	10%
Skin toxicities	42%	—
Oral toxicities	56%	—

Toxicity	Talquetamab (Talq)	CAR-T
Delayed neurotoxicity	—	1.5%
Infections	—	27%
Secondary malignancies	—	1.50%
New Guillain-Barré syndrome	0%	0%
Parkinsonian symptoms	0%	0%

Ultimately, 85% of patients proceeded to CAR-T therapy, achieving a complete or stringent complete response (CR/sCR) in 52%, very good partial response (VGPR) in 71%, and an overall response rate (ORR) of 95%, with most patients deepening their response.

Teclistamab

Teclistamab is the first bispecific antibody designed to redirect T cells by binding CD3 on T-lymphocytes and B cell maturation antigen (BCMA) on myeloma cells. It has a weight-based dosing system that delivers sustained significant responses for patients with RRMM. Following promising data reported in Majestic-1 trial boasting a 63% ORR and 18.4-month median duration of response [14]. Given toxicities and need for close monitoring, step up dosing of Teclistamab is commonly done in the inpatient setting.

Is administration of Teclistamab feasible in the outpatient setting?

A retrospective study was conducted to answer this question by evaluating successful dose escalation (step-up dosing, SUD) of Teclistamab from 2022 to 2023 with 57 (89.2%) patients who initiated Teclistamab in the outpatient setting [15]. Most patients (54.2%) completed step up dosing using the 3-day dose interval. Of these patients, 18 (31.6%) developed CRS (including one patient with Grade 4 CRS), and all required hospital admission. Tocilizumab required as treatment in 6 patients, but it was not given prophylactically. Median CRS-related hospital stay was 2 days. These rates and healthcare utilization burden are low, and in fact interestingly lower than those reported in Majestic-1. However, this may be due in part to limitations from underreporting in the outpatient setting and retrospective data review. Overall, outpatient administration of Teclistamab appears feasible.

Should age preclude administration of Teclistamab?

A retrospective multicenter study evaluated two cohorts, patients with RRMM younger than 75 years old and older than 75 years old who received standard of care Teclistamab [16]. There were 83 patients in the cohort of patients older than 75 years. The older age patient group had a lower rate of high-risk cytogenetics (45%) relative to the younger age group (58%). In terms of efficacy, the overall response rate in

the older group was 62% compared to 53% in the younger age group.

In terms of toxicity, there were not statistically significant differences in the rates of ICANS (19% vs 13%) or CRS (59% vs 52%) between the older and younger groups. There were no grade 4 events in the older group. Time to CRS onset and maximum CRS did not differ significantly. Time to ICANS onset was shorter in the older group at 3 days, compared with the younger group at 4 days, but not statistically significant. Infection rate was higher among the older group at 25% versus 10% in the younger group.

The results of this study were similar to those observed in Majestic-1 in elderly patients with RRMM treated with Teclistamab in the real-world setting showing good efficacy without increased toxicity in older patients. Based on these results, we suggest that age should not preclude the use of Teclistamab treatment.

Elranatamab

Elranatamab (PF-06863135) is a humanized bispecific antibody designed to target myeloma cells by simultaneously binding both CD3 receptors T cells and BCMA (on myeloma cells) at which T cells induce a cytotoxic T cell response. As demonstrated in the phase 2 MagnetisMM-3 trial [17], patients with relapsed or refractory multiple myeloma received subcutaneous Elranatamab once weekly after two step-up priming doses. The trial reached its primary endpoint with a confirmed objective response rate (ORR) of 61.0% including complete response of 35%. Among the responders, 80% achieved improved or sustained response for ≥ 6 months. At a 14.7 month median follow-up time, measures such as median duration of response, progression-free survival and overall survival had not yet been reached with rates of 71.5%, 50.9% and 56.7%, respectively. Frequently reported adverse events (any grade; grade 3–4) included infections (69.9%, 39.8%), cytokine release syndrome (57.7%, 0%), anemia (48.8%, 37.4%), and neutropenia (48.8%, 48.8%) [18].

Ultimately, 85% of patients proceeded to CAR-T therapy, achieving a complete or stringent complete response (CR/sCR) in 52%, very good partial response (VGPR) in 71%, and an overall response rate (ORR) of 95%, with most patients deepening their response [17,18].

Emerging Agents

Accelerated approval was granted by the FDA to Linvoseltamab in July 2025 based on promising ORR 70.9% and Kaplan-Meier estimated-DOR 29.4 months in LINKER-MM1 [19]. Low grade CRS was also reported in a minority of patients. Additional data from longer term follow up is still pending which will help to decide how linvoseltamab will fit into the sequence landscape of emerging treatments for RRMM.

CAR-T Therapy

Approval of CAR-T agents began with the approval of Ide-cel in 2022 and followed by the approval of Cilta-cel in 2022, both for RRMM with progression on at least four prior lines of therapy. In 2024 the approval for Ide-cel was expanded to include patients with progression on two prior lines of therapy and Cilta-cel was expanded to include lenalidomide refractory patients with progression on at least one prior line of therapy.

Ide-cel

Idecabtagene vicleucel is an autologous T-cell therapy that has been genetically engineered to be transduced with a lentiviral vector (LVV) to express a chimeric antigen receptor (CAR) that targets B-cell maturation antigen (BCMA) expressed on myeloma cells. The receptor is comprised of a murine extracellular single-chain variable fragment (scFv) specific for BCMA [20]. The subsequent binding of the anti-BCMA receptor to the target cells expressing BCMA triggers a signaling cascade through the CD3 ζ and 4-1BB domains and CAR-T cell activation. This action propagates further Car T-cell proliferation coupled with cytokine production and activity against BCMA- expressing cells.

In the single arm trial with idecabtagene vicleucel, 100 RRMM patients [21], were given one infusion after receiving lymphodepleting chemotherapy with cyclophosphamide and fludarabine. ORR was 72% (95% CI: 62, 81) with stringent CR rate of 28% (95% CI: 19, 38) (**Table 3**). After median follow-up of 10.7 months, median DOR was 11 months (95% CI: 10.3, 11.4) in responders (PR or better) and 19 months (95% CI: 11.4 months, not estimable [NE]) in patients who achieved stringent CR.

Table 3. Approved CAR-T with trial data.

Drug	Pivotal Trial	ORR (%)	Median DOR	Notable toxicities
Idecabtagene vicleucel (Ide-cel)	KarMMa-1 (21)	72%, 28% sCR	11 months	CRS: ~84%, Neurotoxicity:~ 28%, Cytopenias
Ciltacabtagene autoleucel (cilta-cel)	CARTITUDE-1 (24)	97%, sCR 82.5%	14 months	CRS: ~95%, Neurotoxicity ~20%,

Cilta-cel

Ciltacabtagene autoleucel (cilta-cel) is a second generation genetically engineered autologous CAR T-cell therapy that uses single-domain antibodies against 2 separate epitopes on the BCMA antigen [22]. This agent is the latest authorized therapy that can be offered as an alternative approach for RRMM patients. Ciltacabtagene autoleucel is the sixth CAR T-cell agent that has been FDA approved for B cell malignancies (after tisa-cel, axi-cel, brexu-cel, liso-cel, and ide-cel) as well as the second approved agent in its class that can be used in the treatment of multiple myeloma (after ide-cel).

The phase 1 Legend-2 trial in China as explained in Zhao *et al.* (2018) was a single-arm open-label multicenter study that initially evaluated cilta-cel [23]. This study assessed the safety and efficacy of cilta-cel in 57 RRMM patients. Ultimately, the study results demonstrated that one cilta-cel infusion resulted in a prompt, significant and persistent response while also maintaining an acceptable safety profile in heavily pretreated RRMM patients. Overall response rates (ORR) were 88%, including 68% complete responses (CR), 5% of very good partial responses (VGPR), and 14% partial responses (PR), with 63% of patients achieving minimal residual disease (MRD) negativity at the 10⁻⁵ threshold [25]. The median time to the first response was 1 month, with median progression-free survival and duration of response were 15 months and 14 months, respectively, at a median follow-up of 8 months. The median OS was not reported. However, by 2 years follow up ORR was 97.9% and 82.5% of patients achieved stringent complete response [24].

How does safety and efficacy of Cilta-cel compare to Ide-cel?

A multicenter retrospective study compared safety outcomes and efficacy of Cilta-cel and Ide-cel CAR T-cell therapies for patients with RRMM treated with at least 4 prior lines of therapy [25]. Ide-cel treated patients had an ORR of 79% vs. Cilta-cel had ORR of 89%. Best CR was 47% for patients treated with Ide-cel and 70% in patients treated with Cita-cel. There was also superior PFS and OS for Cilta-cel. However, patients treated with Cilta-cel experienced a statistically significant increase in grade ≥ 3 CRS compared to those treated with Ide-cel. Delayed neurotoxicity and infections were also statistically significantly higher in the cohort treated with Cilta-cel.

Overall, Cilta-cel boasts greater efficacy although at the expense of greater toxicity as well. The limitations of this study include retrospective design and potential for biases inherent to real-world data.

Conclusion

The landscape of relapsed/refractory multiple myeloma (RRMM) treatment has been revolutionized by bispecific antibodies and CAR-T cell therapies, offering unprecedented efficacy for heavily pretreated patients. Bispecific antibodies including Talquetamab, Teclistamab, and Elranatamab have demonstrated robust responses with manageable toxicity profiles that can be used safely across age groups. Talquetamab's successful use as bridging therapy exemplifies the potential for sequential immunotherapy approaches. CAR-T therapies have set new efficacy benchmarks, with ciltacabtagene autoleucel and idecabtagene vicleucel achieving high ORRs, though Cilta-cel's superior efficacy comes with increased toxicity. The recent expansion of these approvals to earlier lines of therapy reflects growing evidence of their transformative potential.

These combinations of bispecifics and CAR-T are promising in patients with RRMM. Moving forward, key priorities include optimizing treatment sequences, developing predictive biomarkers for patient selection, and managing unique toxicity profiles while expanding access to these therapies. The current era represents a paradigm shift toward achieving deeper, more durable responses in RRMM, with the potential to transform multiple myeloma from a uniformly fatal disease to one with significantly improved survival outcomes and quality of life for patients.

Abbreviations

ASCT: Autologous Stem Cell Transplantation; BCMA: B-cell Membrane Antigen; CART: Chimeric Antigen Receptor T-cell Therapy; CR: Complete Response; CRS: Cytokine Release Syndrome; DOR: Duration of Response; GPRC5D: G-protein-Coupled Receptor Family C Group 5 Member D; IMiDs: Immune Modulators; MM: Multiple Myeloma; ORR: Overall Response Rate; OS: Overall Survival; PFS: Progression Free Survival; PI: Proteasome Inhibitors; RRMM: Relapsed/Refractory Multiple Myeloma; sCR: Stringent Complete Response; TNFRSF17: Tumor Necrosis Factor Receptor Superfamily 17; VGPR: Very Good Partial Response

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Conflict of Interest Statement

There is no conflict of interest.

Ethical Statement

The study was reviewed and approved by the institutional review boards (IRBs) of participating sites.

Data Availability Statement

The data that support the findings of this study are available from the corresponding author [MN], upon reasonable request.

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