Updated results from a matching-adjusted indirect comparison of efficacy outcomes for ciltacabtagene autoleucel in CARTITUDE-1 versus idecabtagene vicleucel in KarMMa for the treatment of patients with relapsed or refractory multiple myeloma

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ABSTRACT

Objective: This study used the latest available data cuts from the CARTITUDE-1 and KarMMa clinical trials to update previously published matching-adjusted indirect treatment comparisons (MAICs) assessing the comparative efficacy of ciltacabtagene autoleucel (cilta-cel) versus the FDA-approved idecabtagene vicleucel (ide-cel) dose range of 300 to 450 x 10^6 CAR-positive T-cells in the treatment of patients with relapsed or refractory multiple myeloma (RRMM) who were previously treated with a proteasome inhibitor, an immunomodulatory drug, and an anti-CD38 monoclonal antibody (i.e. triple-class exposed).

Methods: MAICs were performed with the latest available individual patient data for cilta-cel (CARTITUDE-1) and published summary-level data for ide-cel (KarMMa). The analyses included treated patients from CARTITUDE-1 who satisfied the eligibility criteria for KarMMa. The MAIC adjusted for unbalanced baseline covariates of prognostic significance identified in the literature and by clinical expertise. Comparative efficacy was assessed for overall response rate (ORR), complete response or better (>CR) rate, duration of response (DoR), progression-free survival (PFS), and overall survival (OS).

Results: Cilta-cel was associated with statistically significantly improved ORR (odds ratio [OR]: 94.93 [95% confidence interval [CI]: 21.86, 412.25; p < .0001]; RR: 2.23), DoR (hazard ratio [HR]: 0.52 [95% CI: 0.30, 0.88; p = .0152]), PFS (HR: 0.38 [95% CI: 0.24, 0.62; p < .0001]), and OS (HR: 0.43 [95% CI: 0.22, 0.88; p = .0200]) compared with ide-cel.

Conclusions: These analyses demonstrate improved efficacy with cilta-cel versus ide-cel for all outcomes over longer follow-up and highlight its therapeutic potential in triple-class exposed RRMM patients.

Introduction

Multiple myeloma (MM) is a highly heterogenous cancer characterized by an excess of monoclonal (M) proteins and rapid growth of malignant plasma cells. Despite many advancements in therapy with improved responses and survival, MM remains an incurable malignancy and most patients develop relapsed or refractory myeloma (RRMM). To address the need for new, efficacious treatment options, two novel chimeric antigen receptor T-cell (CAR-T) products have been introduced for the treatment of patients with RRMM.

Ciltacabtagene autoleucel (cilta-cel; JNJ-68284528) and idecabtagene vicleucel (ide-cel; bb2121) are two B-cell maturation antigen (BCMA)-targeted CAR-T therapies that have been approved by the Food and Drug Administration (FDA) and European Medicine Agency (EMA) in the treatment of RRMM. Ide-cel is a second-generation autologous CAR-T therapy with an extracellular anti-BCMA single-chain variable fragment, a 4-1BB costimulatory domain, and an anti-CD38 monoclonal antibody (MoAB) (i.e. triple-class exposed), with cilta-cel being evaluated in the...
CARTITUDE-1 trial\(^5\) and ide-cel being evaluated in the KarMMa trial\(^6\). However, these treatments have not been compared directly in any prospective clinical trial.

Previously, results of matching-adjusted indirect treatment comparisons (MAICs) assessing the comparative efficacy of cilta-cel versus ide-cel (FDA-approved dose range of 300 to 450×10\(^6\) CAR-T cells) were published that demonstrated improved efficacy with cilta-cel versus ide-cel.\(^7\) However, new data cuts have since become available for both CARTITUDE-1 and KarMMa. Herein, we present updated MAIC results of cilta-cel versus ide-cel using longer-term follow-up data.

### Methods

#### Data sources

Details of the CARTITUDE-1 and KarMMa clinical trials have been previously published in the original publication,\(^9\) however, the current MAICs used updated data cuts from both studies (Table 1). The treated population of CARTITUDE-1 consisted of 97 patients who received infusions of cilta-cel (median [range] dose: 0.71×10\(^6\) [0.51–0.95×10\(^6\)] CAR-T cells/kg) after lymphodepletion.\(^8\) The data cut-off for CARTITUDE-1 was January 2022 and the median follow-up duration was 27.7 months. The comparator population of interest for the present study consisted of 124 patients from KarMMa who underwent leukapheresis for the 300 to 450×10\(^6\) CAR-positive T-cells cohort (median dose: 0.71×10\(^6\) [0.51–0.95×10\(^6\)] CAR-T cells/kg) after lymphodepletion.\(^8\) Although an additional four patients were treated in the 150 to 450×10\(^6\) CAR-T cells cohort, this dose level has not been approved by the FDA and was therefore not included in the main analysis. The latest available data were used for the FDA-approved dose cohort (300 to 450×10\(^6\) CAR-T cells; \(N=124\)) of KarMMa depending on the outcome (Table 1). For overall response rate (ORR) and complete response or better (≥CR) rate, the data cut-off was 21 December 2020 for which the median follow-up was 24.8 months.\(^10\) For duration of response (DoR) and progression-free survival (PFS), the data cut-off was 7 April 2020 with median follow-up duration of 15.4 months.\(^13\) For overall survival (OS), the data cut-off was January 2020, with median follow-up of 13.3 months.\(^9\) Given that DoR, PFS, and OS were not available for the FDA-approved doses from the latest published data cut for KarMMa, sensitivity analyses were performed for these outcomes using the latest available data (median follow-up 24.8 months) from the “all doses” cohort (150 to 450×10\(^6\) CAR-T cells; \(N=128\)).

### Outcomes

Five efficacy outcomes were assessed in this analysis: ORR, ≥CR rate, DoR, PFS, and OS. For detailed outcome definitions, please refer to the original publication.\(^7\)

### Statistical analyses

#### Matching-adjusted indirect comparison

For details on the MAIC methods please refer to the original publication.\(^7\) In summary, individual patient data from CARTITUDE-1 and published summary-level data from KarMMa were used to estimate the relative effect of cilta-cel versus ide-cel.\(^9\) To adjust for imbalances in patient characteristics of prognostic significance, infused patients of CARTITUDE-1 who satisfied the eligibility criteria of KarMMa were reweighted to match the distribution of prognostic factors in KarMMa. To reflect the impact of weighting, the effective sample size (ESS) was calculated.\(^16\)

Comparative efficacy for cilta-cel versus ide-cel was estimated for ORR and ≥CR rate with odds ratios (ORs) and 95% confidence intervals (CIs) using weighted logistic regression. Relative risks (RRs) were also calculated as the ratio of response rates for cilta-cel versus ide-cel. For DoR, PFS, and OS, hazard ratios (HRs) and 95% CIs were estimated using a weighted Cox proportional hazards model.\(^17\) All analyses were conducted based on the methods developed by Signorovitch et al.\(^18\) and were in line with the National Institute of Health and Care Excellence (NICE) Evidence Synthesis Technical Support Document Series.\(^19\)

### Identification and rank ordering of prognostic factors

As in the original publication, the base case scenario adjusted for refractory status, cytogenetic profile, revised International Staging System (R-ISS) stage, and all plasmacytomas.\(^1\) For details on the identification and ranking of prognostic factors, please refer to the original publication.\(^7\)

Additional sensitivity analyses were performed, which adjusted for all commonly available rank-ordered variables (adding number of prior LOTs, time since MM diagnosis, age, prior stem cell transplant, ECOG status, and sex).

### Table 1. Availability of data from CARTITUDE-1 and KarMMa by outcome.

| Data cut                   | Median follow-up | ORR | ≥CR | DoR | PFS | OS |
|----------------------------|------------------|-----|-----|-----|-----|----|
| **CARTITUDE-1 mITT**       |                  |     |     |     |     |    |
| January 2022 data cut      | 27.7 months      | ✓   | ✓   | ✓   | ✓   | ✓  |
| KarMMa FDA-approved dose cohort (300 to 450×10\(^6\) CAR-T cells) |                  |     |     |     |     |    |
| 21 December 2020 data cut  | 24.8 months      | ✓   | ✓   | X\(^a\) | X\(^a\) | X\(^a\) |
| 7 April 2020 data cut      | 15.4 months      | ✓   | ✓   | ✓   | ✓   | X  |
| 14 January 2020 data cut   | 13.3 months      | ✓   | ✓   | ✓   | ✓   | ✓  |

Abbreviations. CR, complete response; DoR, duration of response; FDA, Food and Drug Administration; mITT, modified intention-to-treat; ORR, overall response rate; OS, overall survival; PFS, progression-free survival.

\(^a\)Results were available for the “all doses” cohort (150 to 450×10\(^6\) CAR-T cells).

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**Table 1. Availability of data from CARTITUDE-1 and KarMMa by outcome.**
Research ethics statement

The CARTITUDE-1 trial protocol was reviewed and approved by an independent ethics committee/institutional review board (IEC/IRB) at all participating sites. All patients participating in the trial provided written informed consent. Similarly, the KarMMa trial protocol was approved by local or independent IRBs or ethics committees at participating sites and all patients provided written informed consent.9 The current analyses were conducted in accordance with a protocol and statistical analysis plan developed prior to the start of data analysis.

Results

Population alignment

The baseline characteristics of the 97 treated patients in CARTITUDE-1 and the 124 patients in KarMMa who underwent infusion for both dose cohorts remained unchanged from the original publication7. For details of adjustment and the resulting ESS in each population, please refer to Supplementary Table A.1, adapted from the original publication7.

Overall response rate and \( \geq \) complete response rate

For both the unadjusted and adjusted comparisons, the observed response rates, and the relative treatment effect estimates (RRs and ORs) are summarized in Table 2.

Prior to adjustment, 98% of patients who received cilta-cel responded and 83% achieved \( \geq \) CR (Figure 1). In comparison, 74% of patients treated with ide-cel responded and 33% achieved \( \geq \) CR (Figure 1). After base case adjustment, patients treated with cilta-cel were 1.3-fold more likely to respond (OR: 94.93 [95% CI: 21.86, 412.25; \( p < .0001 \); RR: 1.34) and 2.2-fold more likely to achieve \( \geq \) CR (OR: 5.65 [95% CI: 2.51, 12.69; \( p < .0001 \); RR: 2.23) compared to patients treated with ide-cel.

Duration of response

For both the unadjusted and adjusted comparisons, the relative treatment effect estimates (HRs) for DoR are summarized in Table 3.

The unadjusted HR for DoR for cilta-cel versus ide-cel was 0.35 (95% CI: 0.23, 0.54; \( p < .0001 \)) (Figure 2). After base case adjustment, DoR was significantly longer for cilta-cel compared to ide-cel (HR: 0.52 [95% CI: 0.30, 0.88]; \( p = .0152 \)).

The Grambsch-Therneau test20 for violation of the proportional hazards assumption was non-significant for the base case adjusted analysis (\( p = .69 \)), indicating that the proportional hazards assumption was not violated.

Progression-free survival and overall survival

For both the unadjusted and adjusted comparisons, the relative treatment effect estimates (HRs) for PFS and OS are...
summarized in Table 3. PFS data were available for all 124 patients in KarMMa who underwent infusion for the $300 \times 10^6$ and $450 \times 10^6$ CAR+T cells dose cohorts, whereas OS data were only available in the public domain for 123 of these patients (the available data removed one patient who received a non-conforming product that did not meet the product release specifications for ide-cel)\(^{14}\).

The unadjusted HR for cilta-cel versus ide-cel was 0.27 (95% CI: 0.18, 0.41; \(p < .0001\)) for PFS (Figure 3) and 0.31 (95% CI: 0.18, 0.52; \(p < .0001\)) for OS (Figure 4). After base case adjustment, cilta-cel was statistically significantly longer compared to ide-cel for PFS (HR: 0.38 [95% CI: 0.24, 0.62; \(p < .0001\)]) and for OS (HR: 0.43 [95% CI: 0.22, 0.88; \(p = .0200\)])

The Grambsch-Therneau test\(^{20}\) for violation of the proportional hazards assumption was non-significant for both outcomes in the base case adjusted analysis (PFS: \(p = .63\); OS: \(p = .07\)), indicating that the proportional hazard assumption was not violated.

**Sensitivity analyses**

The sensitivity analyses of the time-to-event outcomes using the “all doses” cohort from the latest data cut for KarMMa showed superior outcomes for cilta-cel versus ide-cel over longer follow-up and were consistent with the base case comparative estimates presented in Table 3 for the FDA-approved doses. Comparative efficacy estimates for the “all doses” comparison were as follows: DoR: HR = 0.51 (95% CI: 0.32, 0.83; \(p = .0063\)); PFS: HR = 0.38 (95% CI: 0.25, 0.60; \(p < .0001\)); OS: HR = 0.58 (95% CI: 0.34, 1.00; \(p = .049\)).

The sensitivity analyses that incrementally adjusted for additional baseline characteristics (for details, please refer to the original publication\(^7\)) showed superior outcomes for cilta-cel versus ide-cel. Comparative efficacy estimates for the all variables adjusted comparison were as follows: ORR: OR = 4.47 (95% CI: 1.95, 10.26; \(p = .0004\)); RR = 2.08; DoR: HR = 0.59 (95% CI: 0.34, 1.02; \(p = .0610\)); PFS: HR = 0.43 (95% CI: 0.26, 0.71; \(p = .0010\)); and OS: HR = 0.49 (95% CI: 0.23, 1.04; \(p = .0638\)).

**Discussion**

Despite an ever-changing treatment landscape, with several new drugs being approved in the past decade\(^{2,21-25}\), MM remains a difficult to treat and incurable disease. Patients who relapse after receiving IMiDs, PI, and anti-CD38 MoABs (i.e. triple-class exposed patients) have a particularly high unmet need for new safe and effective treatments. Given that head-to-head clinical trials evaluating the efficacy and safety of cilta-cel and ide-cel have not been conducted, MAICs are a valid alternative to estimate the relative treatment effect. Previous analyses have been published and showed improved efficacy with cilta-cel compared with ide-cel; however, follow-up data were limited to 18 months for cilta-cel and 13.3 months for ide-cel\(^7\). Since the publication of previous MAIC, longer-term follow-up data \(>2\) years from CARTITUDE-1 and KarMMa became available, allowing an updated assessment of the comparative efficacy of cilta-cel and ide-cel to better inform healthcare decisions for physicians, patients, and policy makers.

The results from this updated analysis using longer-term follow-up data showed that cilta-cel continues to provide superior efficacy compared with ide-cel beyond the follow-up period included in the original MAIC publication. While the updated estimates were consistent with those in the original publication, the use of more mature data resulted in more precise confidence intervals around these estimates. A summary of the base case results from both the original publication and the present analyses is presented in Supplementary Table B.1. The base case analyses adjusting for the most clinically important factors (i.e. refractory status, cytogenetic profile, R-ISS stage, and all plasmacytomas)
Table 3. Estimated medians and comparative efficacy for cilta-cel versus ide-cel.

| Base case adjusted comparison using FDA-approved doses cohort (sensitivity) | All unadjusted comparison using all doses | Base case adjusted comparison using all doses | Base case adjusted comparison using all doses (sensitivity) |
|---|---|---|---|
| **Table 3** | **Unadjusted comparison** | **Base case adjusted comparison** | **Base case adjusted comparison using all doses** |
| **Unadjusted comparison** | **Base case adjusted comparison using FDA-approved doses cohort (sensitivity)** | **Base case adjusted comparison using all doses** | **Base case adjusted comparison using all doses (sensitivity)** |
| **Median, months (95% CI) HRc** | **Median, months (95% CI) HRc** | **Median, months (95% CI) HRc** | **Median, months (95% CI) HRc** |
| **Duration of response** | **Progression-free survival** | **Overall survival** | **Overall survival** |
| **Patients treated with cilta-cel were 1.3-fold more likely to respond (ORR) and 2.2-fold more likely to achieve a ≥CR than patients treated with ide-cel.** | | | |
| **Abbreviations. CI, confidence interval; FDA, Food and Drug Administration; HR, hazard ratio; NR, not reached.** | | | |

**Notable strengths of these analyses have been discussed previously** and remain applicable herein. However, an additional strength of these updated analyses is the use of the most contemporary data cut available for each outcome.
from both CARTITUDE-1 and KarMMa. The use of the most mature data available for the comparators lends additional credibility to the findings. Furthermore, although IPD was not available for KarMMa, reliable reconstruction of the observed IPD was possible through the simulation of IPD from published Kaplan–Meier plots for DoR, PFS, and OS using the validated Guyot method. The Kaplan–Meier curves developed from simulated IPD were nearly identical to the published Kaplan–Meier curves, with potential slight discrepancies in the exact timings of censoring, which are expected to have only a minimal impact on the estimated HRs.

As in any comparison using non-randomized data, the potential for residual confounding cannot be disregarded. Furthermore, some clinically relevant prognostic factors were not collected in CARTITUDE-1 (e.g. 1q abnormalities) or reported in KarMMa (for details, please refer to the discussion in the original publication) however, the most
important factors, as identified in close consultation with multiple independent clinical experts, were accounted for. It should also be noted that while 45.2% of 73 patients who received bridging therapy in CARTITUDE-1 had a transient decrease in tumor burden (defined as change in serum M-protein, urine M-protein, or difference between involved and uninvolved free light chain) prior to cilta-cel infusion, a decrease in tumor burden does not equate to being a responder per IMWG criteria. No subjects in CARTITUDE-1 achieved CR while on bridging therapy. In the KarMMa study, the 4% of patients who received bridging therapy and had a confirmed partial or very good partial response as per investigator assessment also did not achieve a CR. Therefore, there is no indication that patients in CARTITUDE-1 had a different disease biology than those in KarMMa. Furthermore, as per trial protocol, bridging therapy must have been previously received in a prior line of therapy and therefore all patients who received bridging therapy had either progressed or been non-responsive to those same agents prior to enrollment in CARTITUDE-1.

Since the focus of the present study was to update previously published comparative efficacy analyses using the latest published data with longer follow-up from both studies, safety outcomes were not assessed. Future studies may be conducted including real-world data to address this data gap.

These updated analyses using the latest published data with longer follow-up from both studies were consistent with previously published comparative efficacy analyses in favor of cilta-cel compared with ide-cel in the treatment of triple-class exposed patients with RRMM, specifically in terms of response rates and survival outcomes. Future studies using real-world data may be used to fill this evidence gap and validate the findings from the current analyses.

**Conclusion**

The results of this updated MAIC analysis using longer follow-up data showed that the superior efficacy of cilta-cel compared with ide-cel was maintained for all outcomes studied (ORR, ≥CR rate, DoR, PFS, and OS). Overall, the results indicate that cilta-cel offers substantial clinical benefits for patients with triple-class exposed RRMM, greater than those provided by ide-cel.

**Transparency**

**Declaration of funding**

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**Declaration of financial/other relationships**

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**Author contributions**

All authors were responsible for study conception and design. JD, SVS were responsible for acquisition and analysis. All authors were responsible for drafting or reviewing the manuscript and approving the final version. All authors were responsible for interpretation of data and revising it critically.

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**Data availability statement**

Requests for access to the CARTITUDE-1 trial study data may be submitted through the Yale Open Data Access (YODA) Project site at http://yoda.yale.edu. The data sharing policy of Janssen Pharmaceutical Companies is available at https://www.janssen.com/clinical-trials/transparency. For KarMMa study, a data sharing statement is available with the full text of the primary publication at NEJM.org.12

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