Belantamab mafodotin does not induce B-cell maturation antigen loss or systemic immune dysfunction in multiple myeloma

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Abstract

Various drug classes target B-cell maturation antigen (BCMA) including chimeric antigen receptor T-cell (CAR T) therapies, bispecific antibodies (bsAb), and antibody-drug conjugates (ADC). Outcomes with CAR T and bsAb therapies in multiple myeloma (MM) have been affected by T-cell exhaustion, and abrogated expression/mutation of the BCMA target has been observed with anti-BCMA therapies. Optimal anti-BCMA sequencing strategies are needed to improve long-term clinical outcomes. We used data from multiple clinical studies of the ADC belantamab mafodotin (as monotherapy and combination regimens) to explore its impact on BCMA levels and binding (using electrochemiluminescence methodology) and T-cell/natural killer (NK) cell fitness (including cell counts, expression of functional markers), to determine whether belantamab mafodotin could be sequenced ahead of other BCMA-targeting therapies for MM. Levels of free soluble BCMA (sBCMA), measured at the best-confirmed response (BCR) and at progression, dropped at BCR but returned to near baseline at time of disease progression. There was no apparent impact on the binding epitope of BCMA, as indicated by the retention of belantamab mafodotin binding to sBCMA. No significant changes in cell counts or expression of T-cell exhaustion markers (PD-1, TIGIT, TIM-3 [except NK cells], or CTLA-4) and co-stimulatory markers (ICOS [except $CD4^+$ T cells], OX40, 4-1BB) were observed at relevant time points (up to 4 or 21+ months depending on the marker). No negative impact was observed on expression of proliferation (Ki67) and antitumor activity (granzyme B, CD107a) markers. Pending confirmatory studies, our results indicate potential for utilizing belantamab mafodotin ahead of other anti-BCMA therapies in MM.

Introduction

Multiple myeloma (MM) is a hematological cancer that follows a relapsing course.[1,2] The treatment landscape has advanced with novel therapies that improve outcomes; however, most patients continue to relapse and remissions become shorter as lines of therapy (LOT) progress.[1,2] Moreover, with the various drug classes available for MM treatment and the heterogeneity of the patient population with MM, the choice of therapy and sequencing of LOT has become more complex.[1,2] The use of multi-drug regimens for first-line therapy further complicates treatment decisions in the relapsed/refractory MM (RRMM) setting, with retreatment with the same drug/drug class becoming more prevalent and producing limited clinical benefit.[3] Therefore, effective treatments with novel mechanisms of action are needed after relapse. Several B-cell maturation antigen (BCMA)-targeting treatment modalities have shown high clinical benefit and/or activity in RRMM,[4] which may help address the issue of retreatment and reduced efficacy in this setting.

BCMA is overexpressed on MM plasma cells, and its activation leads to cell survival and proliferation.[4,5] When membrane-bound BCMA (mBCMA) is shed, soluble BCMA (sBCMA) is circulated (and can be used as a surrogate for mBCMA).[4,6] Higher levels of sBCMA are inversely correlated

with clinical outcomes and response to future treatment in MM,[4] suggesting that sBCMA, like M-protein, is a potential prognostic factor and biomarker for MM disease burden.[4,5] Minimal BCMA expression on tissues other than plasma cells makes it an ideal target for MM treatment.[7] BCMA-targeting agents available or being evaluated for MM include chimeric antigen receptor T-cell (CAR T) therapies, bispecific antibodies (bsAb), and antibody-drug conjugates (ADC).[5]

The anti-BCMA ADC belantamab mafodotin has been investigated as monotherapy, demonstrating consistent overall response rates (ORR) of 32-41% at 2.5 mg/kg in heavily pretreated RRMM, and in combination regimens for MM, showing significant progression-free survival (DREAMM-7 and DREAMM-8 studies) and overall survival (DREAMM-7) benefits in the combination regimens.[8] Belantamab mafodotin comprises a humanized, afucosylated, immunoglobulin (Ig)G antibody conjugated to the microtubule inhibitor monomethyl auristatin F, and its antitumor effects occur through immunogenic cell death, direct cytotoxicity, antibody-dependent cellular cytotoxicity, and antibody-dependent cellular phagocytosis.[7,9] With the emergence of BCMA-targeting CAR T, bsAb, and ADC therapies, it is imperative to understand factors that can impact their efficacy, including whether the use of a given anti-BCMA drug class impacts the use of subsequent alternative anti-BCMA drug classes.

All three anti-BCMA treatment modalities rely on target presence; however, outcomes with both CAR T and bsAb therapies are also dependent on baseline immune fitness.[10-13] That is, patients with higher levels of naïve/effector T cells and lower levels of regulatory T cells and inhibitory receptors/T-cell exhaustion markers (programmed cell death protein-1 [PD-1], T-cell immunoreceptor with immunoglobulin and tyrosine-based inhibitory motif [TIGIT], T-cell immunoglobulin and mucin domain 3 [TIM-3], and cytotoxic T-lymphocyte-associated protein 4 [CTLA-4]) have better outcomes.[10-13] Conversely, reduced immune fitness and the emergence of T cells with an exhausted phenotype, potentially due to chronic activation, characterize non-responders in patients treated with teclistamab[11] and are common features of patients previously treated with CAR T or bsAb.[10,13-15] Although infrequent, antigen escape can occur after BCMA-directed treatment, resulting in the emergence of MM clones with target-antigen loss/mutation and reduced responses to subsequent anti-BCMA therapies.[12,16-18] Studies have examined response rates of patients exposed to >1 BCMA-targeting modality. Responses have been observed with teclistamab or ciltacabtagene autoleucel treatment in patients previously exposed to BCMA-directed CAR T or bsAb therapies, or belantamab mafodotin.[19,20] Of note, the evidence available regarding exposure to ≥1 anti-BCMA agent is limited by small patient numbers, late LOT, and use of other LOT between the BCMA therapies, all of which complicate interpretation.

Potential resistance to anti-BCMA therapies over time highlights a need for BCMA-directed treatments that do not impact immune fitness or the ability of subsequent anti-BCMA therapies to bind to BCMA on MM cells. Additionally, there is a need to explore sequencing to determine if there is benefit to multiple BCMA-targeting therapy lines, whether they can be used consecutively, and what the optimal sequence would be. We assessed the impact of belantamab mafodotin on the target and immune fitness, and thus its potential to influence subsequent BCMA-targeting modalities by using translational data to evaluate BCMA levels and immune cell function during and after belantamab mafodotin treatment in patients with MM.

Methods

Studies

This is a *post hoc* analysis of data from studies of belantamab mafodotin as monotherapy (phase I DREAMM-1 [*clinicaltrials gov. Identifier: NCT02064387*], phase II DREAMM-2 [*clinicaltrials gov. Identifier: NCT03525678*], phase III DREAMM-3 [*clinicaltrials gov. Identifier: NCT04162210*], phase I DREAMM-12 [*clinicaltrials gov. Identifier: NCT04398745*], phase II DREAMM-14 [*clinicaltrials gov. Identifier: NCT05064358*]) or combinations (phase I/II DREAMM-5 [belantamab mafodotin with nirogacestat; *clinicaltrials gov. Identifier: NCT04126200*], phase I/II DREAMM-6 [belantamab mafodotin with lenalidomide and dexamethasone or with bortezomib and dexamethasone; *clinicaltrials gov. Identifier: NCT03544281*], phase III DREAMM-7 [belantamab mafodotin with bortezomib and dexamethasone; *clinicaltrials gov. Identifier: NCT04246047*], phase III DREAMM-8 [belantamab mafodotin with pomalidomide and dexamethasone; *clinicaltrials gov. Identifier: NCT04484623*]) in patients with RRMM and in patients with newly-diagnosed MM (phase I DREAMM-9 [belantamab mafodotin with bortezomib, lenalidomide, and dexamethasone; *clinicaltrials gov. Identifier: NCT04091126*]).[21-29] All studies complied with the Declaration of Helsinki and Good Clinical Practice guidelines. Trial protocols/amendments were approved by appropriate ethics bodies at participating institutions, and patients provided written informed consent.

Target antigen expression and binding

Free and complexed soluble B-cell maturation antigen

Free and complexed sBCMA were analyzed using electrochemiluminescence methodology (*Online Supplementary Materials*), to evaluate belantamab mafodotin binding to sBCMA and determine whether the binding epitope is altered at progression. Software/calculations used for analyses of free/complexed sBCMA are described in the *Online Supplementary Materials*.

Membrane-bound B-cell maturation antigen

Formalin-fixed and paraffin-embedded bone marrow biopsy samples were examined for BCMA and CD138 expression. Hematoxylin and eosin-stained tissue was used to identify

areas with malignant plasma cells for immunohistochemistry (IHC)-based assays, at Mosaic laboratories (Lake Forest, CA) in accordance with Mosaic Laboratories' standard operating procedures. Slides were scanned using an Aperio AT Turbo system (Aperio, Vista, CA); staining was evaluated by a pathologist. Evaluation of reactivity involved the pathologist tumor cell score indicating the level of cellular staining present and H-Score indicating intensity of staining (scoring described in the *Online Supplementary Appendix*).

Binding of belantamab mafodotin monoclonal antibody and teclistamab

Qualitative biolayer interferometry (BLI) was used to assess binding and pairing of GSK2857914 (belantamab mafodotin without the cytotoxic payload) and teclistamab with BCMA antigens (*Online Supplementary Methods; Online Supplementary Table S1*).

Immune fitness

Neutrophil-to-lymphocyte ratio (NLR) and absolute lymphocyte counts (ALC) were analyzed using complete blood count and differential counts (calculations described in the *Online Supplementary Appendix*).

T cells and natural killer (NK) cells were analyzed using flow cytometry as described in the *Online Supplementary Appendix (Online Supplementary Table S2; Online Supplementary Figure S1*). T-cell, B-cell, monocyte, and NK cell activation markers in whole blood were also evaluated using two separate flow cytometry assays (antibodies and fluorochromes presented in *Online Supplementary Table S2*). Levels (median fluorescence intensity and percent expression) of T-cell functional markers were evaluated. These included the T-cell suppression markers PD-1, TIGIT, TIM-3, and CTLA-4, the co-stimulatory receptors inducible T-cell co-stimulator (ICOS), OX40, and 4-immunoglobulin and cytokine receptor BB (4-1BB),[30,31] and the intracellular and intranuclear activation markers Ki67 (proliferation marker), granzyme B (marker of T-cell and NK cell antitumor activity), and CD107a (NK cell degranulation marker).[32] Sample preparation, cell surface and intracellular staining, and flow cytometry analysis were performed as described in the *Online Supplementary Appendix/Online Supplementary Figure S2*. Generalized least-squares or mixed models were used to model longitudinal data for flow cytometry. Samples were taken from patients across all studies assessed.

Results

Impact of belantamab mafodotin on target antigen expression

As BCMA expression is essential to successful treatment with anti-BCMA therapies, levels of mBCMA prior to treatment with belantamab mafodotin were assessed (Figure 1) and showed that mBCMA was consistently measurable independent of number of prior LOT (N=492 across 1 to ≥7 prior LOT [*P*=0.5965]). Baseline mBCMA levels were not significantly different between LOT. This suggests that BCMA is a good target in patients with MM, regardless of the therapy line.

In addition to evaluating BCMA levels prior to belantamab mafodotin treatment, we evaluated sBCMA levels (a surrogate for BCMA expression) during treatment and after progressive disease (PD) to determine if there was BCMA loss, which could impact subsequent anti-BCMA agents. Due to the competitive binding of belantamab mafodotin

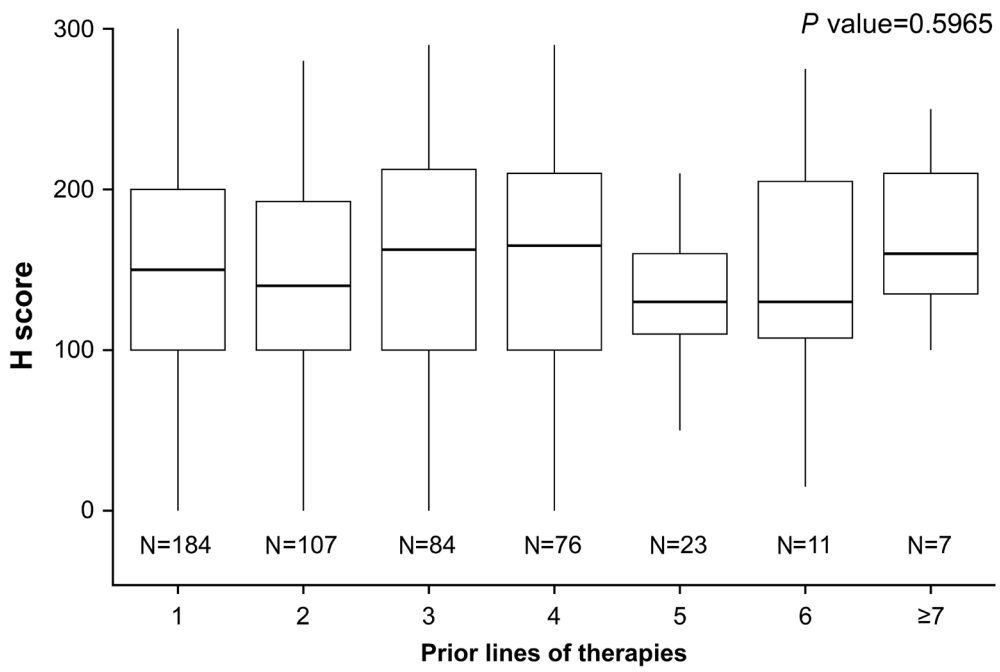

Figure 1. Membrane-bound B-cell maturation antigen levels at baseline across lines of therapy.* *Includes third-line-or-later monotherapy from DREAMM-2, DREAMM-3, DREAMM-12, and DREAMM-14; second-line-or-later combinations from DREAMM-6, DREAMM-7, and DREAMM-8; and a first-line combination from DREAMM-9. The black center line within the box represents the median value, the lower limit of the box represents the 25th percentile, the upper limit of the box represents the 75th percentile, whiskers represent the 5th and 95th percentiles.

with the reagents used to assess free sBCMA, selected time points were prior to dosing or >4 days post-dose. Among patients who had progressed at the time of this analysis, sBCMA was detectable at baseline in nearly all monotherapy-treated patients (100% [N=75] in DREAMM-1 and 99.1% [N=213] in DREAMM-2) and in all combination therapy-treated patients in DREAMM-7 and DREAMM-8 (N=32 each); at PD, 51 (98.0%) DREAMM-1, 183 (98.9%) DREAMM-2, 32 (100%) DREAMM-7, and 32 (100%) DREAMM-8 patients had detectable sBCMA (*Online Supplementary Table S3*). Among patients who had progressed at the time of this analysis and had sBCMA levels available at baseline, best confirmed response, and progression, sBCMA levels dropped at the time of response to belantamab mafodotin monotherapy (N=63) and the belantamab mafodotin, bortezomib, and dexamethasone (BVd) combination (N=32) but remained measurable and returned to near baseline at the time of PD (P=0.0169 [monotherapy] and P=0.1582 [BVd] for comparison between baseline and progression levels, Figure 2; *Online Supplementary Figure S3*). Similarly, sBCMA was measurable at progression in patients who had progressive disease by the time of this analysis after receiving belantamab mafodotin, pomalidomide, and dexamethasone (BPd), and the median change from baseline was similar to that of the comparator (pomalidomide, bortezomib, and dexamethasone [PVd]) (*Online Supplementary Figure S4*). Optional progressive disease bone marrow samples were only successfully collected from three patients across the studies. IHC images of BCMA/CD138 expression from these three patient samples showed that mBCMA levels were measurable at complete response (CR) and at PD in BPd-treated patients (N=2) and PVd-treated patients (N=1) (Figure 3).

Overall, the sBCMA and mBCMA results together indicate essentially no BCMA target loss after belantamab mafodotin monotherapy or combination therapy, with mBCMA expression and median change in sBCMA levels after BPd comparable to the standard of care therapy (PVd). Furthermore, sBCMA levels followed a similar trend as mBCMA, remaining measurable after belantamab mafodotin treatment.

Impact of belantamab mafodotin on target binding site

Distinct mechanisms underlying MM antigen escape post-anti-BCMA therapy are slowly emerging.[16,17] Not only is the presence of the target fundamental to BCMA-targeting therapies, but binding capacity and potential impact of mutations on the target are important to understand. We explored the impact of belantamab mafodotin on its binding epitope by generating complexed sBCMA (belantamab mafodotin bound to sBCMA) concentration data. Complexed sBCMA levels increased markedly and were maintained throughout belantamab mafodotin treatment (with and without nirogacestat [N=3-35 across time points]; Figure 4A), including up to the time point at which progressive disease was observed (N=20 patients who progressed; Figure 4B), and regardless of response status. Data suggest that there is no apparent impact on the belantamab mafodotin BCMA binding epitope upon PD.

To inform whether the apparent lack of impact on the belantamab mafodotin BCMA binding epitope translates into lack of impact on the binding epitope of the bsAb teclistamab, binding analysis was performed. Binding experiments showed that both GSK2857914 and teclistamab independently bind to histidine-tagged BCMA (His-BCMA). Pairing experiments showed that a saturated biotinylated GSK2857914:BCMA complex was formed for both His-BCMA and recombinant human BCMA (rhBCMA) over the second loading period. Further exposure of these immobilized complexes to teclistamab indicated little to no interaction (*Online Supplementary Figure S5*). This suggests that GSK2857914 and teclistamab share similar or overlapping epitopes on the BCMA antigen. Together with the complexed

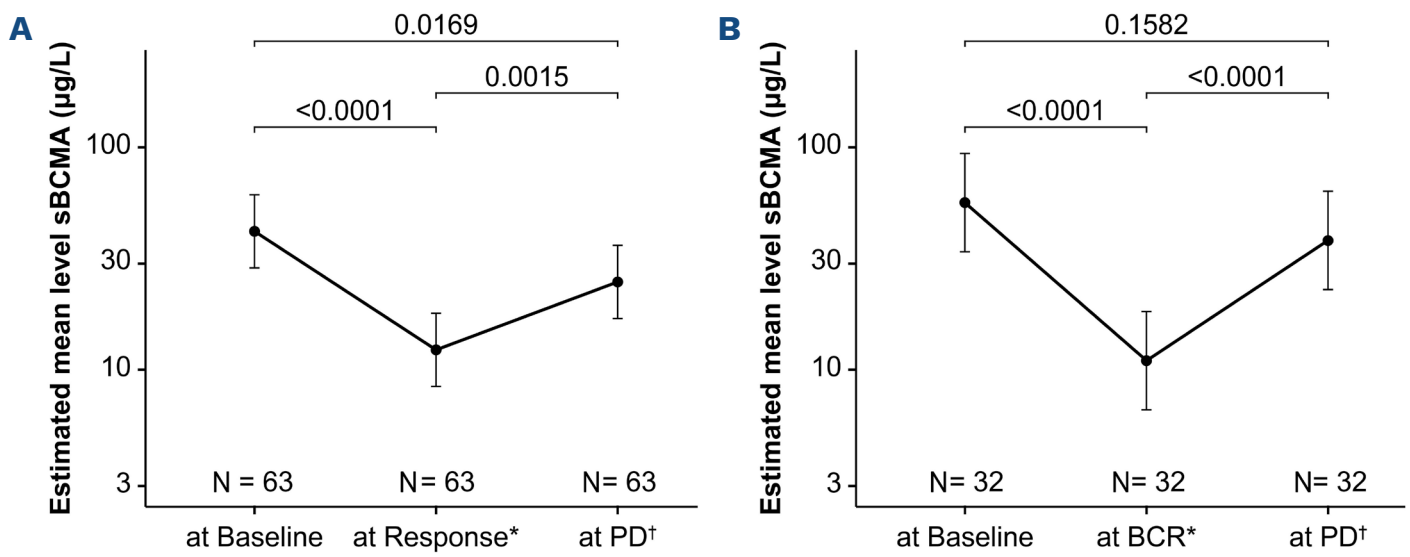

Figure 2. B-cell maturation antigen levels. B-cell maturation antigen levels at baseline, at best response, and at disease progression in patients receiving belantamab mafodotin as (A) monotherapy in DREAMM-2 or (B) in the belantamab mafodotin, bortezomib, dexamethasone regimen in DREAMM-7. *At response, minimum within best achieved response; †at progressive disease, latest recorded measure at progressive disease or later. Error bars represent 95% confidence intervals. BCR: best confirmed response; BVd: belantamab mafodotin, bortezomib, and dexamethasone; PD: progressive disease; sBCMA: soluble B-cell maturation antigen.

sBCMA data that showed belantamab mafodotin binding across time points, these data support that teclistamab would likely not be impacted by prior belantamab mafodotin treatment.

Impact of belantamab mafodotin on immune fitness

Immune fitness is fundamental to the success of CAR T and bsAb therapies;[10-13] as such, it is essential to determine what impact belantamab mafodotin has on the immune

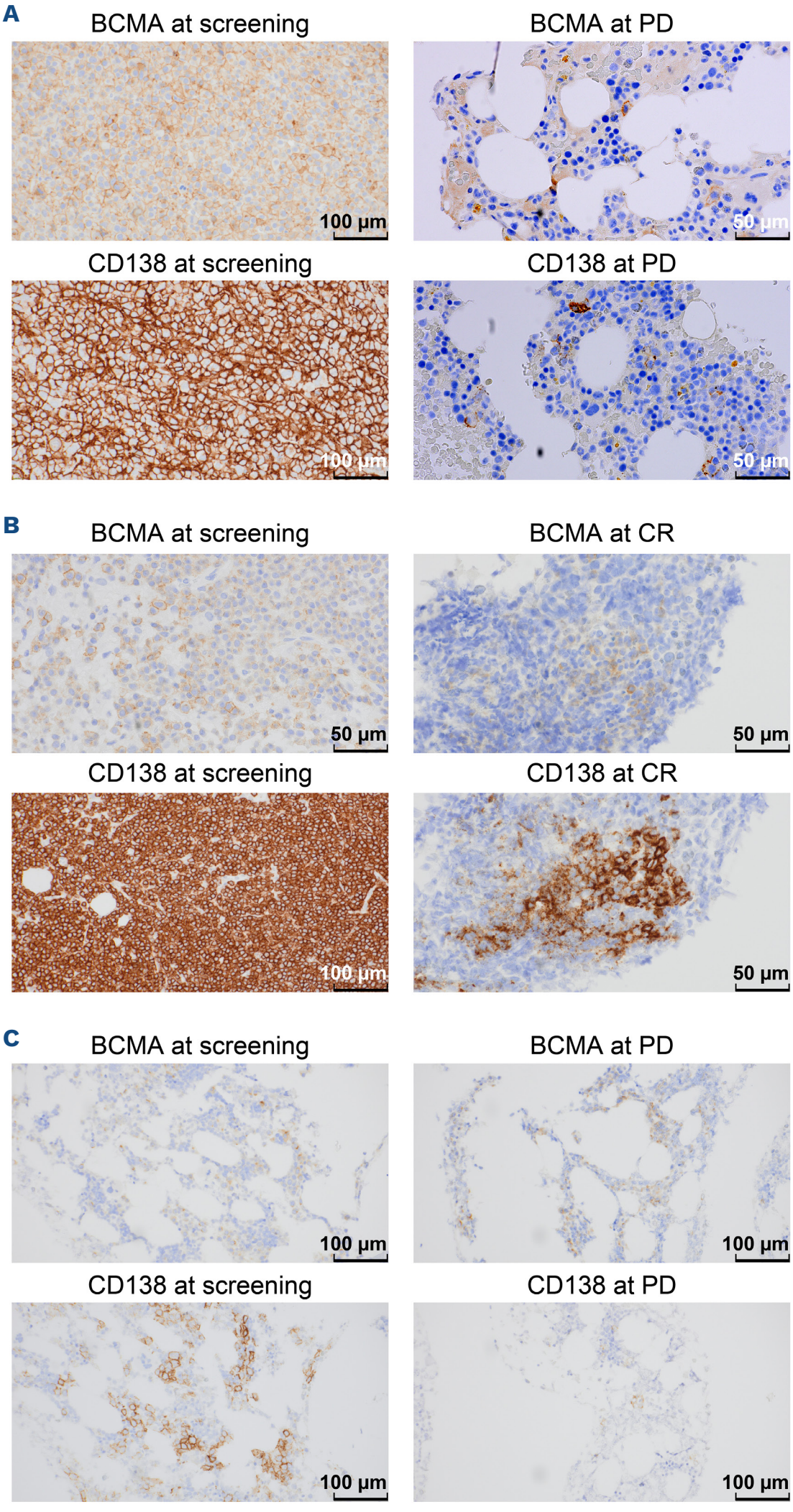

Figure 3. Membrane-bound B-cell maturation antigen and CD138 levels at screening and at response or disease progression in patients receiving belantamab mafodotin with pomalidomide and dexamethasone or pomalidomide with bortezomib and dexamethasone in DREAMM-8. (A) Patient receiving belantamab mafodotin with pomalidomide and dexamethasone (BPd), measured at screening and disease progression. (B) Patient receiving BPd, measured at screening and complete response. (C) Patient receiving pomalidomide with bortezomib and dexamethasone (PVd), measured at screening and disease progression. BCMA: B-cell maturation antigen; BPd: belantamab mafodotin, pomalidomide, and dexamethasone; CR: complete response; mBCMA: membrane-bound B-cell maturation antigen; PD: progressive disease; PVd: pomalidomide, bortezomib, and dexamethasone. Images were magnified at the following objectives: (A) BCMA and CD138 at screening 20x, BCMA and CD138 at PD 40x; (B) BCMA at screening, BCMA at CR, and CD138 at CR 40x, CD138 at screening 20x; (C) all images 20x.

cells that drive their mechanisms of action. Initially, global immune fitness was examined. Complete blood counts with differential showed that ALC and NLR were not significantly altered throughout 73 weeks of treatment with belantamab mafodotin monotherapy in DREAMM-2 (N=2-208 across time points; *P*=0.306 for ALC and *P*=0.898 for NLR; Figure 5A, B), and when evaluated by responders (N=2-77 across time points) and non-responders (N=2-131), there was no difference in ALC or NLR (*P*=0.836 for ALC and *P*=0.997 for NLR for responders *vs.* non-responders). In addition, $CD8^+$ (*P*=0.284) and the ratio of $CD4^+$ to $CD8^+$ cells (*P*=0.103) was not impacted by belantamab mafodotin treatment (N=10-139 across time points); $CD4^+$ counts were significantly increased after 21+ months of therapy (*P*=0.031) (Figure 5C-E). Next, we focused directly on factors affecting peripheral immune profiles of $CD4^+$ T cells, $CD8^+$ T cells, and NK cells, which can impact CAR T and bsAb mechanisms of action. $CD4^+$/$CD8^+$ T-cell profiles were not significantly altered over the course of belantamab mafodotin treatment when evaluated using data from DREAMM-5 (combination; N=4-11 across time points) and DREAMM-14 (monotherapy; N=13-142 across time points). At relevant time points (i.e., the final time points assessed), these cells showed no increase in percent expression from baseline in the T-cell exhaustion markers PD-1, TIGIT, TIM-3, or CTLA-4, nor any decrease in co-stimulatory markers ICOS, OX40, or 4-1BB, with the exception of a significant increase in TIM-3 expression on NK cells (*P*<0.001; median at baseline, 44%; median at 21+ months, 71%) and a significant decrease in ICOS expression on $CD4^+$ cells (*P*<0.001; median at baseline, 22%; median at 21+ months, 18%) (Figures 6 and 7; *Online Supplementary Figures S6-11*). There was also no significant increase in the regulatory T-cell population (*Online Supplementary Figure S12*). $CD4^+$ T cells, $CD8^+$ T cells, and NK cells retained their immune cell activity during treatment with belantamab mafodotin, as evidenced by no significant changes in percent expression of granzyme B in all three cell types and by no significant change in CD107a expression in NK cells (Figure 8). Both $CD4^+$ and $CD8^+$ T cells retained their proliferative capacity, with the percentage of cells expressing Ki67 remaining similar to baseline throughout treatment. Taken together, the immune fitness data demonstrate that belantamab mafodotin generally has no impact on total lymphocyte and T-cell numbers, CD4/CD8 T-cell ratio, or expression of phenotypic markers associated with poor responses to CAR T and bsAb therapies.

Discussion

BCMA-targeting CAR T, bsAb, and ADC treatments have fundamentally different mechanisms of action, and studies suggest that the effects of anti-BCMA therapies on target expression and immune cell composition may impact the efficacy of subsequent BCMA-targeting agents.[12,16,17] Therefore, it is important to consider sequencing of these treatments to optimize patient outcomes. Using data from multiple DREAMM studies, we examined the impact of belantamab

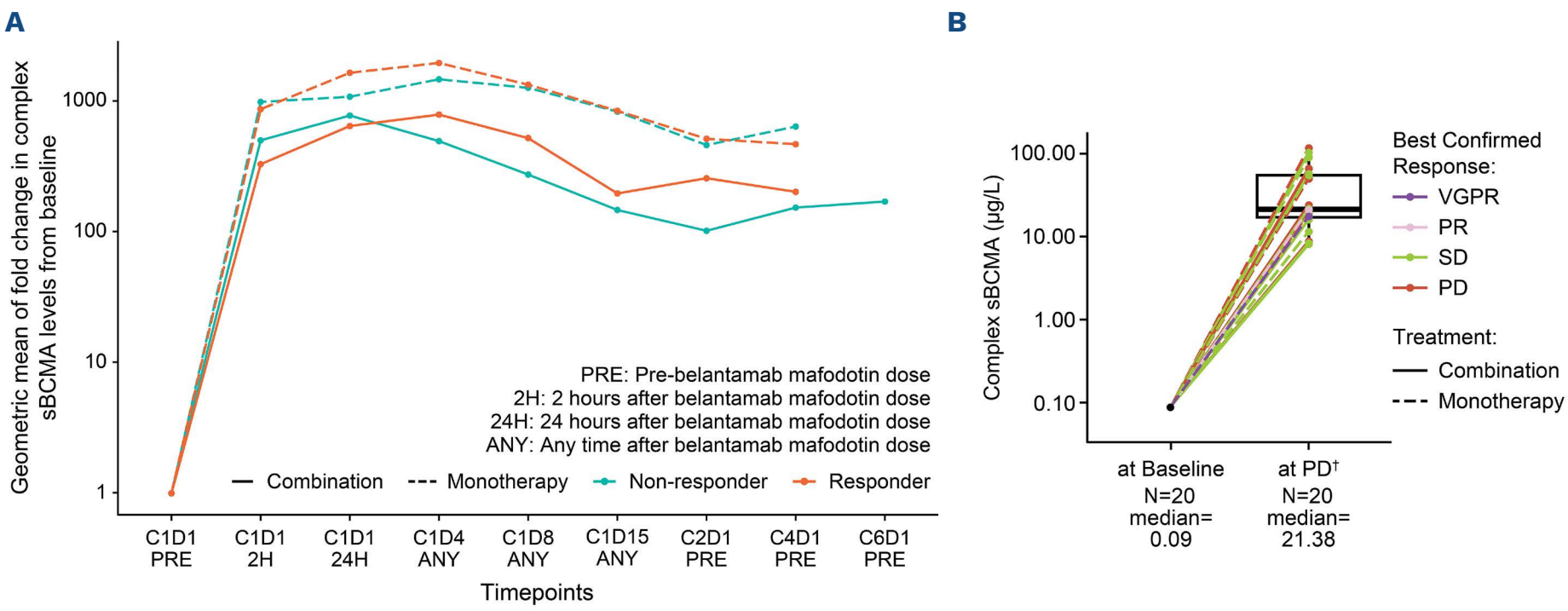

Figure 4. Belantamab mafodotin soluble B-cell maturation antigen binding. (A) Geometric mean of fold-change in complex soluble B-cell maturation antigen (sBCMA) from baseline. (B) Complex sBCMA concentration among patients who experienced disease progression.* *Data are from the DREAMM-5 study of belantamab mafodotin with and without nirogacestat, and included are time points with 3 or more patients per response status and treatment arm. †At progressive disease, samples taken within 4 weeks of clinical confirmation of progressive disease. In panel (B), baseline sBCMA levels for 2 patients with complex sBCMA exceeding the non-quantifiable threshold were adjusted to 0.09. Monotherapy: C1D1 N=35; C2D1 N=30; C4D1 N=11; C6D1 N=3. Combination: C1D1 N=29; C2D1 N=27; C4D1 N=17; C6D1 N=7. BCMA: B-cell maturation antigen; C1D1: cycle 1, day 1; PD: progressive disease; PR: partial response; PRE: pre-dose; sBCMA: soluble B-cell maturation antigen; SD: stable disease; VGPR: very good partial response.

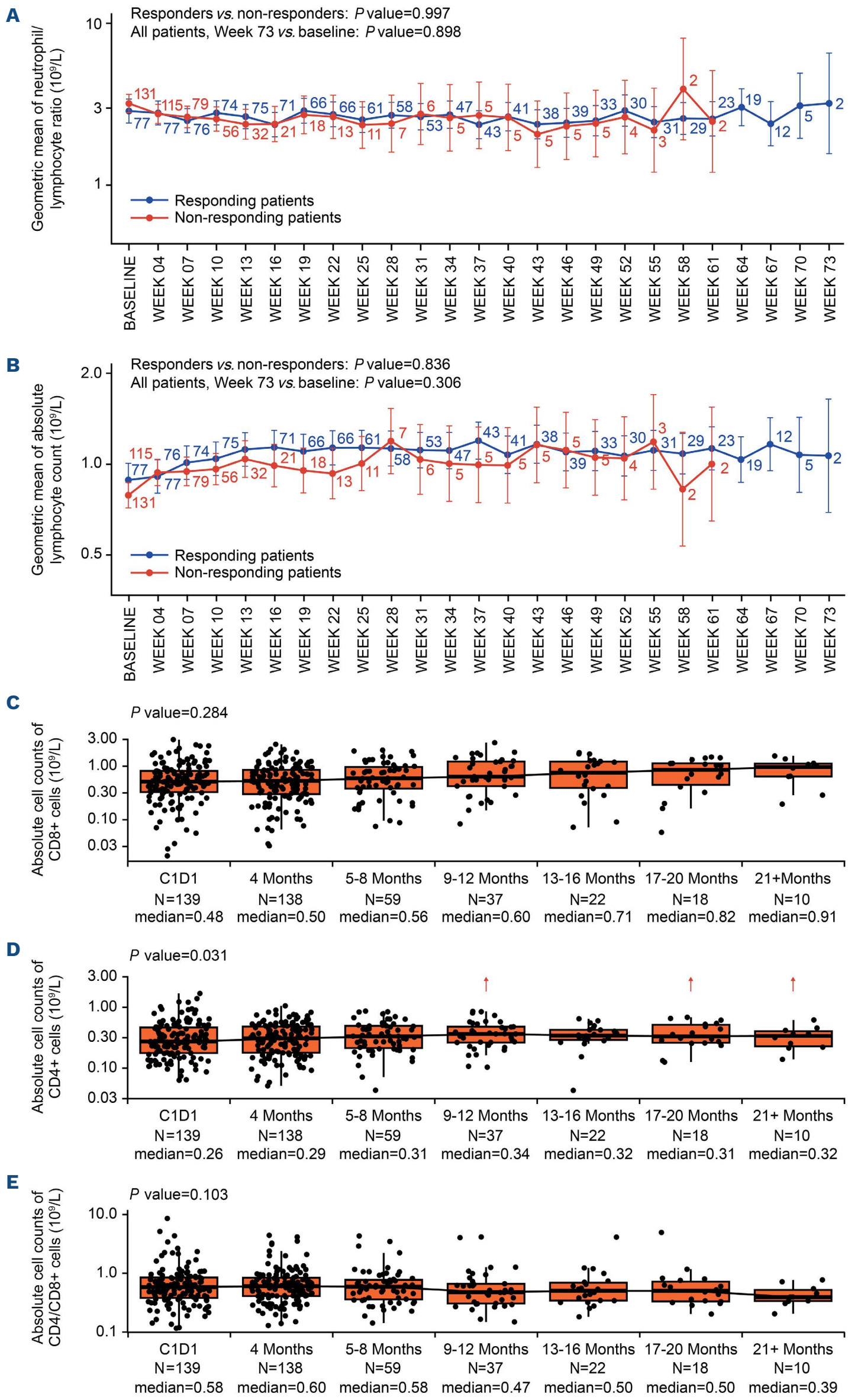

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Figure 5. Impact of belantamab mafodotin on cell counts. (A) Neutrophil-to-lymphocyte ratio, (B) absolute lymphocyte count, (C) $CD8^+$ counts, (D) $CD4^+$ counts and (E) $CD4/CD8^+$ counts over time.* *Neutrophil-to-lymphocyte ratio and absolute lymphocyte count data from the DREAMM-2 study of belantamab mafodotin monotherapy, and $CD4/CD8^+$ counts are from the DREAMM-14 study of belantamab mafodotin monotherapy. Patients who were not evaluable for response were excluded from the analyses of neutrophil-to-lymphocyte ratio and absolute lymphocyte count. C1D1: cycle 1, day 1.

mafodotin on the BCMA target and immune cells during and after treatment in patients who received belantamab mafodotin, to explore factors that might impact sequencing of belantamab mafodotin prior to bsAb or CAR T-cell therapy.

Several studies have shown that treatment with BCMA-directed T-cell-engaging therapies may affect BCMA expression through genomic deletion or loss of functional recognition and binding;[17,33,34] this may particularly be an issue after bsAb therapy, where loss or alteration of BCMA was noted in 43% of progressing patients in a recent study.[17] In contrast, genomic changes resulting in BCMA loss after belantamab mafodotin have been reported in only a single patient to date.[16] In the current study with a larger patient population, we evaluated the impact of belantamab mafodotin on BCMA levels using reagents that compete for the BCMA binding site with belantamab mafodotin, and found that sBCMA levels were reduced at best confirmed response, likely reflecting the reduction of malignant plasma cells, and returned to near-baseline levels at PD. sBCMA levels at PD may not have fully returned to baseline due to lower tumor burden or early detection of progression. The recovery to near-baseline sBCMA levels indicates that belantamab mafodotin treatment does not result in complete loss of target, that complete target loss is unlikely to be the primary mechanism of escape from belantamab mafodotin, and consequently, that other BCMA-directed therapies will still be able to bind BCMA on MM cells after treatment with belantamab mafodotin; however, further analyses are required to determine whether mechanisms exist that would lead to downregulation of BCMA in a subclonal population of cells in patients treated with belantamab mafodotin. In addition to no complete loss of target, we found evidence that belantamab mafodotin binds to sBCMA throughout the course of treatment, including when patients progress, suggesting that there was no apparent impact on the binding site of belantamab mafodotin. As the greatest selective pressure on MM cell clones would be expected at the belantamab mafodotin binding epitope during treatment, the apparent lack of change detected in this region suggests that the binding sites of other anti-BCMA therapies are unlikely to be impacted following belantamab mafodotin treatment.[33-36] While we also found that BPd-treated patients had measurable mBCMA levels at CR and at PD, it is important to acknowledge the potential presence of BCMA mutations that could impact belantamab mafodotin binding.[17,34] As such, the reliance on belantamab mafodotin binding to sBCMA and IHC samples from a small number of patients is a limitation of this analysis, and sequencing or structural modeling analyses of BCMA are required to confirm whether belantamab mafodotin induces genomic changes on the target that could impact binding sites of other BCMA-targeting agents. Analyses of complexed sBCMA levels of other anti-BCMA therapies following belantamab mafodotin relapse are currently ongoing. The preliminary epitope binding data reported here showed that belantamab mafodotin monoclonal antibody and teclistamab share a similar BCMA binding epitope; therefore, no impact on teclistamab binding would be expected following belantamab mafodotin treatment.

Reduced immune fitness and T-cell exhaustion related to prior treatment can negatively impact outcomes with CAR T and bsAb therapies, and may even be caused by these therapies themselves,[10,11,13-15] which may limit the clinical utility of sequencing these agents as first BCMA-targeting therapy. In our study, immune system impairment was not observed with belantamab mafodotin, as evidenced by no significant changes in ALC or NLR, and at relevant time points, no increase in expression of exhaustion markers (PD-1, TIGIT, TIM-3 [except NK cells], CTLA-4), and no decrease in expression of co-stimulatory markers (ICOS [except $CD4^+$ T cells], OX40, 4-1BB), activation markers (granzyme B, CD107a), or proliferation markers (Ki67) on $CD4^+/CD8^+$ T cells and/or NK cells. This suggests that cell counts remain stable during belantamab mafodotin treatment, and cells retain their proliferative and cytolytic capacity; however, additional studies would be beneficial to demonstrate that these remain stable over longer time frames, including at PD, as would be expected for patients treated in earlier LOT. TIM-3 was the only T-cell exhaustion marker that significantly increased at the final available assessment. While TIM-3 is thought to have an immunosuppressive role in various cancer types,[37] in patients with acute myeloid leukemia, its expression on NK cells was associated with improved effector functions and improved disease outcomes.[38] Further research is required to determine the impact of TIM-3 expression in MM. $CD4^+$ to $CD8^+$ T-cell ratio is an important predictor of MM prognosis, with lower ratios associated with poorer survival.[39] Higher $CD4^+$ to $CD8^+$ ratios are required in the leukapheresis product of CAR T therapies to optimize CAR T expansion and response.[40] In our analysis, the $CD4^+$ to $CD8^+$ T-cell ratio was not impacted by belantamab mafodotin treatment, indicating that belantamab mafodotin may not affect CAR T treatment outcomes. Similarly, the lack of belantamab mafodotin effect on T-cell expression and exhaustion suggests that belantamab mafodotin would not impact subsequent bsAb treatment.[11]

Previous studies have examined the clinical impact of

sequencing alternative BCMA-targeting agents following belantamab mafodotin or other anti-BCMA therapy, with varying outcomes reported. In these studies, CAR T or bsAb treatment after belantamab mafodotin or other anti-BCMA therapy demonstrated potential for response in most patients, but the reported ORR, PFS, and/or duration of responses were generally lower than that seen in patients without prior anti-BCMA therapy.[19,20,41-45] These studies were limited by small sample sizes, heterogenous and heavily pretreated patient populations, high rates of extramedullary disease in the patient populations studied, and/or multiple LOT and variable intervals between anti-BCMA treatments, leading to difficulty drawing definitive conclusions. Shorter duration of prior anti-BCMA treatment and a longer interval between the therapies were associated with improved outcomes with CAR T and teclistamab.[19,20,41,44,46] Specifically, an interval of ≥9 months between prior anti-BCMA therapies and teclistamab resulted in greater response rates

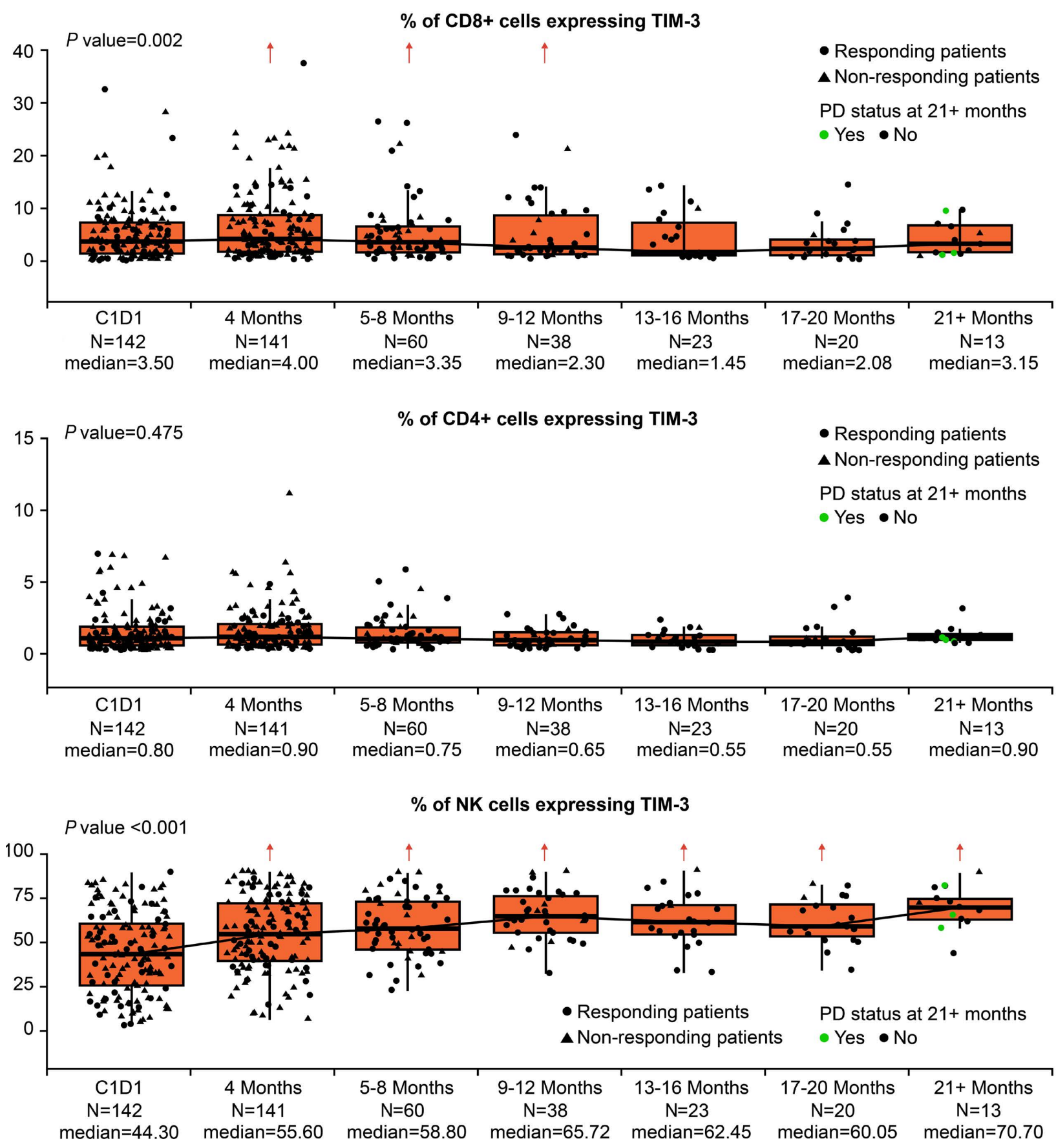

Figure 6. Impact of belantamab mafodotin on T-cell exhaustion marker TIM-3.* *TIM-3 data are from the DREAMM-14 study of belantamab mafodotin monotherapy. C1D1: cycle 1, day 1; NK: natural killer; PD: disease progression; TIM-3: T-cell immunoglobulin and mucin domain 3.

and PFS than shorter intervals,[45,46] while patients recently treated with belantamab mafodotin (regardless of the timing of prior belantamab mafodotin exposure) had similar teclistamab response rates to patients without prior anti-BCMA therapies.[46] Due to shedding of mBCMA and the half-life of belantamab mafodotin (~14 days), it is not believed that long-term belantamab mafodotin binding to mBCMA is responsible for the lower efficacy observed with anti-BCMA treatment following belantamab mafodotin in some studies.[4,47] It is currently unknown whether there are resistance mechanisms to belantamab mafodotin that may impact outcomes with subsequent anti-BCMA therapies,[8] and future studies investigating resistance mechanisms are required. It is also important to note that CAR T therapies can potentially be impacted by treatment status at the time of CAR T manufacturing, which may have affected previous CAR T sequencing reports. A study indicated that compared to newly-diagnosed patients, CAR T treatments in patients who relapsed at the time of manufacturing had reduced proliferative and antitumor capacity.[10]

Data from the DREAMM-7 and DREAMM-8 studies indicate that belantamab mafodotin has high efficacy in combination regimens in the second-line-or-later setting.[22,24,48] DREAMM-7 showed a robust PFS benefit (23-month increase in PFS with BVd *vs*. daratumumab plus bortezomib and dexamethasone), with maintenance of the PFS benefit in the next LOT (median PFS2 hazard ratio=0.56 [95% confidence interval: 0.41-0.76]) and a subsequent OS benefit.[48] DREAMM-8 also showed a significant PFS benefit for BPd *versus* PVd, which was maintained into the next line of therapy (hazard ratio=0.61 [95% confidence interval: 0.43-0.86]); follow-up for OS is ongoing in DREAMM-8.[22] The long-term effects demonstrated with BVd could indicate that immune-mediated effects of belantamab mafodotin are carried into the next LOT.[8]

Belantamab mafodotin represents a highly accessible anti-BCMA option due to its potential for wide availability and ease of administration.[49] Given the efficacy and manageable

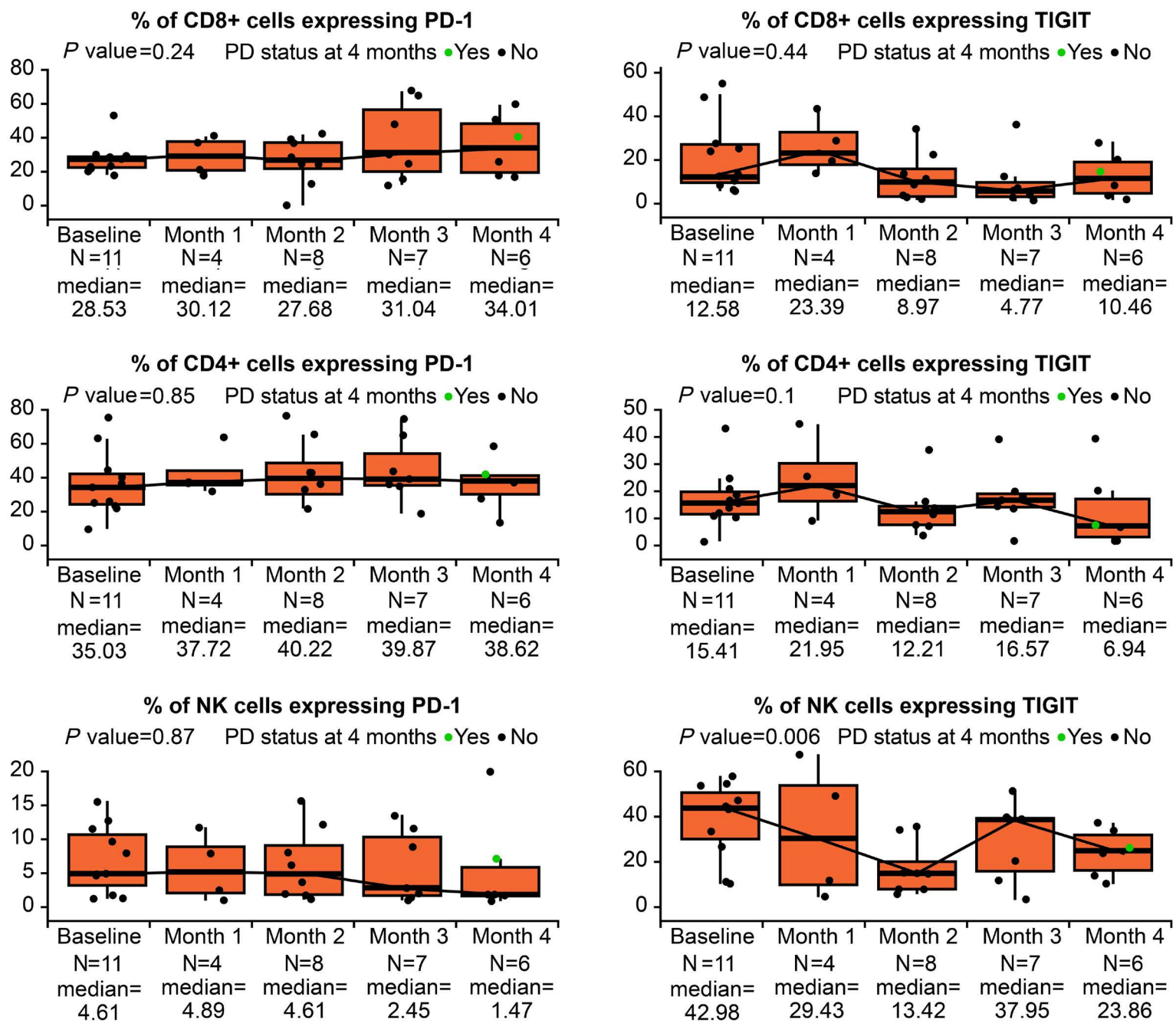

Figure 7. Impact of belantamab mafodotin on T-cell exhaustion markers PD-1 and TIGIT.* *PD-1 and TIGIT data are from the DREAMM-5 study of belantamab mafodotin with nirogacestat. NK: natural killer; PD-1: programmed cell death protein 1; PD: disease progression; TIGIT: T-cell immunoreceptor with immunoglobulin and ITIM domains.

safety profile of belantamab mafodotin combination regimens shown in clinical trials,[22,24,48] and the lack of changes in target expression/binding and immune cell composition demonstrated in our analysis, sequencing belantamab mafodotin as the first BCMA-targeting treatment may offer high clinical efficacy without compromising the use of subsequent BCMA-targeting therapies; however, rigorous prospective clinical studies of anti-BCMA efficacy after belantamab mafodotin treatment are required to support these findings in matched patient populations.

Limitations of our study include small sample sizes for IHC analysis of mBCMA and PD-1/TIGIT flow cytometry data and the lack of data on T-cell function. The immune fitness data may be limited by the use of data from three different trials, which included different doses and schedules of belantamab mafodotin, monotherapy and combination regimens, and differing follow-up periods; subgroup analyses in larger study populations and in the frontline setting would be valuable to further examine the impacts of belantamab mafodotin. PD was reached in few patients at the time of this analysis due to the long PFS and duration of response with BVd and BPd,[22,48] thereby limiting the number of PD samples collected for analysis of sBCMA levels.

Follow-up clinical data to determine the impact of belantamab mafodotin on subsequent exposure to a BCMA-directed CAR T/bsAb therapy are limited. However, a recent study examining subsequent therapies after BVd treatment in DREAMM-7 found that the median time from the start of bsAb exposure (including teclistamab as 4th to 7th LOT) to progression or death was 11.7 months.[50] This is comparable to the median PFS seen with teclistamab in the MajesTEC-1 study,[51] which was 11.4 months at a median follow-up of 30.4 months. However, as noted, this dataset is limited and further, more extensive, follow-up clinical studies of belantamab mafodotin in RRMM are required.

To our knowledge, this is the first study to analyze the

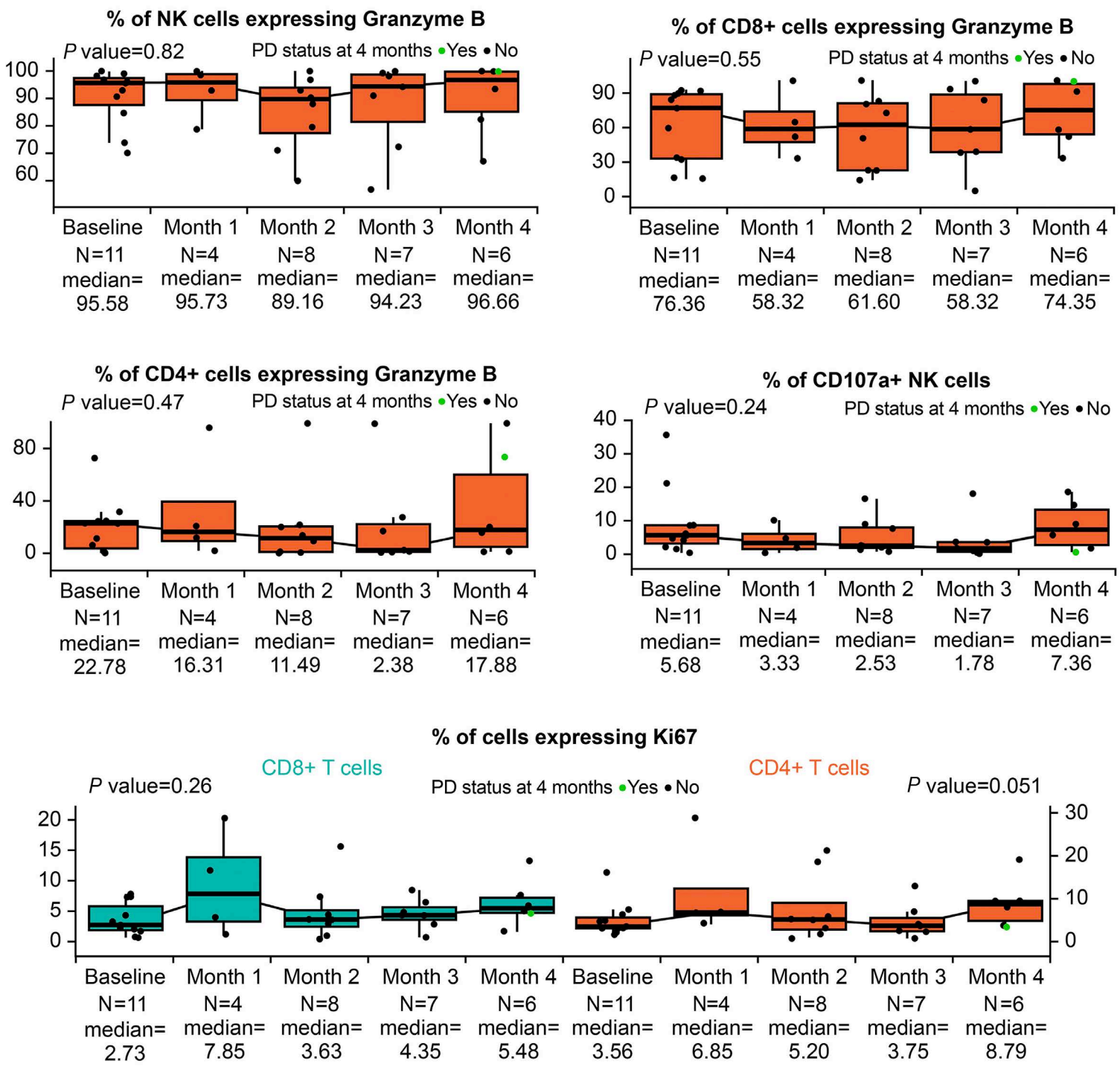

Figure 8. Impact of belantamab mafodotin on immune cell activity (granzyme B, CD107a) and proliferation (Ki67).* *Data are from the DREAMM-5 study of belantamab mafodotin with nirogacestat. NK: natural killer; PD: disease progression.

effects of belantamab mafodotin monotherapy and combination treatments on sBCMA and immune cell profiles across multiple studies in MM. Belantamab mafodotin provides durable and sustained benefit to patients with MM,[22,24] without impairing BCMA expression or binding, nor immune cell composition. Collectively our data suggest that belantamab mafodotin may not directly impact subsequent alternative BCMA-targeting therapies although confirmatory clinical studies are needed.

Disclosures

HM, TM-D, GF-B, QH, YM, SP, SS, XZ, and DEL are employees of and hold financial equities in GSK. MM has received payment for expert testimony from GSK, AbbVie, and Janssen; received honoraria from Abbvie, Amgen, BMS, Celgene, GSK, Janssen (to Institution [Inst.]), Janssen-Cilag, Merck, Novartis, Oncopeptides, Sanofi. ST received honoraria from Amgen, BMS GmbH & Co. KG, GSK, Janssen Oncology, Pfizer, Roche, and Sanofi; had a consulting or advisory role with AstraZeneca, BMS, Janssen, GSK, Pfizer, Sanofi; received grants from BMS, Janssen, GSK, K36, and Pfizer; and received research funding from Amgen (Inst.), BMS GmbH & Co. KG (Inst.), Genentech (Inst.), GSK (Inst.), Janssen (Inst.), Pfizer (Inst.), and Roche (Inst.). KW received honoraria from Abbvie, Adaptive Biotechnologies, Amgen, BMS, Celgene, GSK, Janssen (Inst.), Janssen-Cilag, Karyopharm Therapeutics, Menarini, Novartis, Oncopeptides, Pfizer, Roche, Sanofi, Stemline Therapeutics, and Takeda; had a consulting or advisory role with Adaptive Biotechnologies, Amgen, BMS, Celgene, GSK, Janssen-Cilag, Karyopharm Therapeutics, Menarini, Oncopeptides, Roche, Sanofi, and Takeda; received research funding from Abbvie (Inst.), Amgen (Inst.), BMS/Celgene (Inst.), Celgene (Inst.), GSK (Inst.), Janssen-Cilag (Inst.), and Sanofi (Inst.); and received travel, accommodations, or expenses from Amgen, BMS, Celgene, GSK, Janssen-Cilag, Menarini, and Takeda. PGR had a consulting or advisory role with BMS/Celgene, GSK, Karyopharm Therapeutics, Oncopeptides, and Sanofi; and received research funding from BMS/Celgene (Inst.), Karyopharm Therapeutics (Inst), and Oncopeptides (Inst.). ADC had a consulting or advisory role with Abbvie, Arcellx, BMS, GSK, Ichnos Sciences, ITeos Therapeutics, Janssen Oncology, Novartis, Pfizer, Roche/Genentech, Legend, Kite, Regeneron, Sanofi, Prothena, and Moderna; received research funding from Genentech/Roche (Inst.), GSK (Inst.), Janssen Oncology (Inst.), and Novartis (Inst.); has patents related to CAR T cells and biomarkers of Cytokine Release Syndrome; and received travel, accommodations, or expenses from Abbvie, BMS, Ichnos Sciences, GSK, and Janssen Oncology.

Contributions

HM participated in conceptualization, data acquisition, data analysis, and data interpretation. MM participated in data interpretation. ST participated in data interpretation. KW participated in data interpretation. TM-D participated in data acquisition and data analysis. GF-B participated in data acquisition and data analysis. QH participated in data acquisition and data analysis. YM participated in data acquisition and data analysis. SP participated in data acquisition and data analysis. SS participated in data acquisition and data analysis. XZ participated in data acquisition, data analysis, and data interpretation. PGR participated in data interpretation. ADC participated in data interpretation. DEL participated in conceptualization, data acquisition, data analysis, and data interpretation. All authors reviewed and revised the manuscript, approved the final version, and agreed to submit the manuscript for publication.

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Data-sharing statement

Information about GSK's data sharing commitments and access requests to anonymized individual participant data and associated documents can be requested for further research from https://www.gsk-studyregister.com/en/.

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