DREAMM-9: Phase I Study of Belantamab Mafodotin Plus Standard of Care in Patients With Transplant-Ineligible Newly Diagnosed Multiple Myeloma

S. Z. USMANI, MD[1], M. MIELNIK, MD[2], Y. KOH, MD[3], A. ALONSO ALONSO, MD[4], X. LELEU, MD, PHD[5], H. QUACH, MD[6], C. MIN, MD[7], W. JANOWSKI, MD[8], A. ABDALLAH, MD, PHD[9], M. GARG, MD, FRCP, FRCPATH[10], I. SANDHU, MD, FRCPC[11], E. M. OCIO SAN MIGUEL, MD, PHD[12], A. ORIOL, MD[13], P. RODRIGUEZ-OTERO, MD, PHD[14], K. RAMASAMY, MBBS, MRCP, FRCPATH, PHD[15], K. WEISEL, MD[16], B. BESEMER, MD[17], M. CAVO, MD[18], X. L. ZHOU, MD, PHD[19], M. C. KAISERMANN, MD, PHD[20], C. M. BEGO MARQUES[21], D. WILLIAMS, MSC[20], F. CARRENO, PHD[20], B. E. KREMER, MD, PHD[20], I. V. GUPTA, MD[20], M. HUS, MD, PHD[2]

1 Memorial Sloan Kettering Cancer Center, New York, USA | 2 Department of Hematooncology and Bone Marrow Transplantation, Medical University of Lublin, Poland | 3 Seoul National University Hospital, Seoul, South Korea | 4 Hospital Quirón Madrid, Madrid, Spain | 5 CHU de Poitiers, Poitiers, France | 6 St Vincent's Hospital Melbourne, Melbourne, Australia | 7 The Catholic University of Korea Seoul St. Mary's Hospital, Seoul, South Korea | 8 Calvary Mater Newcastle, Newcastle, Australia | 9 University of Kansas, Kansas City, KS, USA | 10 Leicester Royal Infirmary, Leicester, UK | 11 University of Alberta, Edmonton, Canada | 12 Hospital Universitario Marqués de Valdecilla (IDIVAL), Universidad de Cantabria, Santander, Spain | 13 Institut Català d'Oncologia and Institut Josep Carreras - Hospital Universitari Germans Trias i Pujol (HUGTP), Badalona, Spain | 14 Department of Hematology, Clínica Universidad de Navarra, Pamplona, Spain | 15 Churchill Hospital, Headington, Oxford, UK | 16 University Medical Center Hamburg-Eppendorf, Hamburg, Germany | 17 University of Tübingen, Tübingen, Germany | 18 IRCCS Azienda Ospedaliero-Universitaria di Bologna, Istitutodi Ematologia "Seràgnoli", Universitàdegli Studi di Bologna, Bologna, Italy | 19 GlaxoSmithKline, Waltham, MA, USA | 20 GlaxoSmithKline, Upper Providence, PA, USA | 21 GlaxoSmithKline, Barcelona, Spain

INTRODUCTION

The combination of bortezomib, lenalidomide and dexamethasone (VRd) as a standard of care (SOC) for transplant-ineligible newly diagnosed multiple myeloma (TI NDMM) has been further supported by the Phase 3 SWOG S0777 trial in patients with TI NDMM where a ≥VGPR of 75% and ORR of 90% were observed (with 7 years of follow-up).[1,2]

Ongoing development of novel therapies and combinations strive to improve survival outcomes beyond what is expected from SOC (median progression-free survival of 43 months with VRd).[1]

Belantamab mafodotin (belamaf) has a multimodal mechanism of action that eliminates multiple myeloma cells via direct cytotoxicity as well as by a systemic anti-MM tumour immune response.[3-5]

Enhanced anti-myeloma activity has been demonstrated in preclinical work when belamaf has been combined with bortezomib or lenalidomide, providing rationale for this treatment combination.[3,6]

An earlier presentation given at the American Society of Hematology Annual Meeting 2021 of data from the DREAMM-9 study of belamaf combined with VRd in 36 patients with TI NDMM showed encouraging efficacy, with deep responses of ≥VGPR achieved in at least half of all patients in each cohort (50-100%).[7]

OBJECTIVES

The aim of this study is to further evaluate the safety and tolerability of belamaf combined with VRd in up to 8 cohorts and up to 160 patients with TI NDMM, and to establish the recommended Phase III dose. We report the preliminary findings of belamaf + VRd for 5 cohorts with different dosing regimens.

METHODS

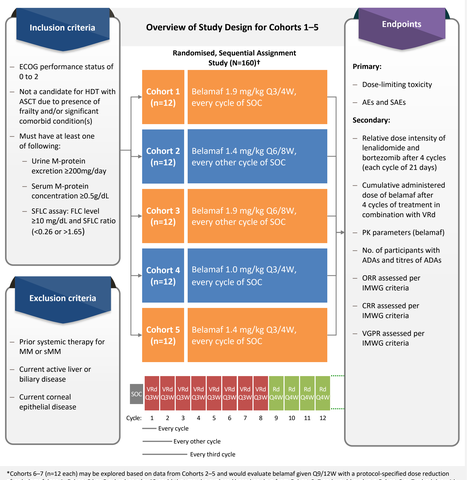
Patient Population

DREAMM-9 (NCT04091126) is an ongoing Phase I, open-label, randomised, dose and schedule evaluation study of belamaf + VRd in patients with TI NDMM. A detailed study design for Cohorts 1–5 is shown in **Figure 1**; data for Cohorts 6–8* are not yet mature.

Belamaf is given with VRd Q3/6W until Cycle 8, and with Rd Q4/8W thereafter.

After evaluation of safety data for Cohort 1, Cohorts 2–5 were opened in parallel and enrolled patients were randomised 1:1:1:1.

Figure 1. Study Design



*Cohorts 6–7 (n=12 each) may be explored based on data from Cohorts 2–5 and would evaluate belamaf given Q9/12W with a protocol-specified dose reduction after belamaf dose 1. Cohort 8 has 3 subcohorts (n=12 each) that may be explored based on data from Cohort 6–7 and would evaluate Cohort 6 or 7 schedules with dose reduction after belamaf dose 2 or initiate therapy at a reduced dose and Q12W schedule. †Total population of 160 patients reflects enrolment across all 8 cohorts, of which Cohorts 1–5 are shown here.
ADA, anti-drug antibodies; AEs, adverse events; ASCT, autologous stem cell transplant; belamaf, belantamab mafodotin; CRR, complete response rate; ECOG, Eastern Cooperative Oncology Group; FLC, free light chain; HDT, high-dose chemotherapy; IMWG, International Myeloma Working Group; MM, multiple myeloma; ORR, overall response rate; PK, pharmacokinetics; Q3W, every 3 weeks; Q4W, every 4 weeks; Q6W, every 6 weeks; Q8W, every 8 weeks; Q9W, every 9 weeks; Q12W, every 12 weeks; SAE, serious adverse events; SFLC, serum FLC; sMM, smouldering MM; SOC, standard of care; VGPR, very good partial response; VRd, bortezomib, lenalidomide and dexamethasone.

RESULTS

Patient Population

At data cut-off, 7 December 2021, 64 patients with TI NDMM have been randomised in this study; 12 patients in each of Cohorts 1–3, 15 in Cohort 4, and 13 in Cohort 5. Demographic data for these patients are presented in **Table 1**.

- 55% (n=35) of the total study population was male, and patients had a median age of 73 years.
- 33% of patients (n=21) in the total study population were International Staging System (ISS) stage I and 59% of patients (n=38) were stage II or III.
- 17% of patients (n=11) in the total study population had high-risk cytogenetics (consisting of one or more of the following cytogenetic abnormalities: t(4;14), t(14;16), del17p).

Table 1. Demographics, Baseline Disease, and Clinical Characteristics for Patients Treated with Belamaf + VRd

	Cohort 1 belamaf 1.9 mg/kg Q3/4W +VRd n=12	Cohort 2 belamaf 1.4 mg/kg Q6/8W +VRd n=12	Cohort 3 belamaf 1.9 mg/kg Q6/8W +VRd n=12	Cohort 4 belamaf 1.0 mg/kg Q3/4W +VRd n=15	Cohort 5 belamaf 1.4 mg/kg Q3/4W +VRd n=13	Total population N=64
Age, median (range), years	72.5 (63–77)	73.0 (69–80)	73.0 (69–78)	73.0 (51–85)	74.0 (65–88)	73.0 (51–88)
18 to <65 years, *n* (%)	2 (17)	0	0	2 (13)	0	4 (6)
65 to <75 years, n (%)	5 (42)	8 (67)	8 (67)	10 (67)	7 (54)	38 (59)
≥75 years, n (%)	5 (42)	4 (33)	4 (33)	3 (20)	6 (46)	22 (34)
Sex, n (%)						
Male	8 (67)	6 (50)	6 (50)	7 (47)	8 (62)	35 (55)
Race, n (%)						
White	10 (83)	10 (83)	8 (67)	12 (80)	11 (85)	51 (80)
Black/African American	0	0	0	1 (7)	0	1 (2)
Asian	2 (17)	2 (17)	4 (33)	2 (13)	2 (15)	12 (19)
ISS disease stage, n (%)						
Stage I	2 (17)	3 (25)	4 (33)	6 (40)	6 (46)	21 (33)
Stage II	6 (50)	5 (42)	6 (50)	7 (47)	3 (23)	27 (42)
Stage III	3 (25)	4 (33)	1 (8)	1 (7)	2 (15)	11 (17)
Unknown	1 (8)	0	1 (8)	1 (7)	2 (15)	5 (8)
Genetics, Amp (1q), *n* (%)	1 (8)	1 (8)	3 (25)	4 (27)	4 (31)	13 (20)
Cytogenetic abnormalities, n (%)						
High risk[a]	4 (33)	1 (8)	2 (17)	1 (7)	3 (23)	11 (17)
Other	8 (67)	11 (92)	10 (83)	14 (93)	10 (77)	53 (83)
Myeloma immunoglobulin, n (%)						
IgA	5 (42)	3 (25)	0	4 (27)	4 (31)	16 (25)
IgG	7 (58)	8 (67)	12 (100)	11 (73)	7 (54)	45 (70)
None	0	1 (8)	0	0	2 (15)	3 (5)
Light chain, n (%)						
Kappa	7 (58)	6 (50)	10 (83)	11 (73)	9 (69)	43 (67)
Lambda	3 (25)	5 (42)	2 (17)	4 (27)	4 (31)	18 (28)
Extramedullary disease, *n* (%)						
Yes	3 (25)	1 (8)	1 (8)	0	0	5 (8)
Median number of belamaf cycles (range)	6.0 (2–11)	3.0 (1–5)	3.5 (2–6)	4.5 (1–11)	5.0 (1–7)	NA
Median belamaf dose intensity, mg/kg/cycle (range)	0.54 (0.3–1.9)	1.40 (1.3–1.6)	1.72 (1.1–1.9)	1.0 (1.0–1.0)	1.19 (1.0–1.4)	NA

Data cut-off 7 December 2021. Baseline characteristics are shown for all patients who were randomised and does not exclude patients who were randomised but not treated.
[a]High-risk cytogenetics defined as t(4;14), t(14;16), del17p.
Belamaf, belantamab mafodotin; Ig, immunoglobulin; ISS, International Staging System; NA, not available; Q3/4W, every 3 weeks for Cycle 1–8 and every 4 weeks afterwards; Q6/8W, every 6 weeks for Cycle 1–8 and every 8 weeks afterwards; SOC, standard of care: bortezomib, lenalidomide, and dexamethasone in Cycle 1–8 followed by lenalidomide and dexamethasone from Cycle 9 onwards. VRd, bortezomib, lenalidomide, and dexamethasone.

Safety

There were no new safety signals reported for belamaf in combination with VRd.

Across Cohorts 1–4, all patients experienced adverse events (AEs) related to study treatment (**Table 2**).

Table 2. Safety Data* and Duration of Follow-Up for Patients Treated With Belamaf + VRd Across Cohorts

AEs, *n* (%), unless otherwise stated	Cohort 1 Belamaf 1.9 mg/kg Q3/4W +VRd n=12	Cohort 2 belamaf 1.4 mg/kg Q6/8W +VRd n=12	Cohort 3 belamaf 1.9 mg/kg Q6/8W +VRd n=12	Cohort 4 belamaf 1.0 mg/kg Q3/4W +VRd n=14	Cohort 5 belamaf 1.4 mg/kg Q3/4W +VRd n=13
Any AEs	12 (100)	12 (100)	12 (100)	14 (100)	13 (100)
Thrombocytopenia	7 (58)	7 (58)	5 (42)	6 (43)	6 (46)
Constipation	8 (67)	5 (42)	3 (25)	6 (43)	5 (38)
Diarrhoea	6 (50)	3 (25)	3 (25)	4 (29)	4 (31)
Peripheral sensory neuropathy	2 (17)	7 (58)	7 (58)	1 (7)	2 (15)
Treatment-related AEs	12 (100)	12 (100)	12 (100)	14 (100)	11 (85)
Grade ≥3 AEs	12 (100)	12 (100)	11 (92)	11 (79)	9 (69)
Grade 3 or 4 AEs related to belamaf	8 (67)	5 (42)	3 (25)	5 (36)	4 (31)
AEs leading to permanent discontinuation of study treatment[†]	3 (25)	3 (25)	2 (17)	3 (21)	1 (8)
AEs leading to dose reduction	12 (100)	10 (83)	10 (83)	7 (50)	4 (31)
AEs leading to reduction of belamaf	4 (33)	0	2 (17)	0	2 (15)
AEs leading to reduction of bortezomib	12 (100)	8 (67)	8 (67)	4 (29)	4 (31)
AEs leading to reduction of lenalidomide	8 (67)	4 (33)	7 (58)	4 (29)	2 (15)
AEs leading to reduction of dexamethasone	5 (42)	6 (50)	4 (33)	4 (29)	3 (23)
AEs leading to dose delay/interruption	12 (100)	12 (100)	11 (92)	13 (93)	11 (85)
AEs leading to delay of belamaf	10 (83)	5 (42)	3 (25)	7 (50)	7 (54)
AEs leading to delay of bortezomib	11 (92)	8 (67)	7 (58)	8 (57)	7 (54)
AEs leading to delay of lenalidomide	11 (92)	6 (50)	8 (67)	6 (43)	5 (38)
AEs leading to delay of dexamethasone	7 (58)	8 (67)	3 (25)	3 (21)	5 (38)
Any SAEs	11 (92)	6 (50)	7 (58)	4 (29)	4 (31)
Treatment-related SAEs	6 (50)	1 (8)	2 (17)	2 (14)	3 (23)
Fatal SAEs	1 (8)	1 (8)	1 (8)	0	0
Treatment-related fatal SAEs	0	0	0	0	0
Duration of follow-up (months), median (range; IQR)	17.4 (7.3–23.1; 15.2–19.3)	5.9 (4.8–11.3; 5.1–7.6)	6.1 (4.4–11.5; 5.2–7.5)	4.7 (0.1–11.7; 3.2–6.6)	5.8 (0.8–9.7; 4.3–7.5)

*Safety data excluding ocular AEs which are presented separately in Table 3. †One patient in Cohort 1 withdrew from study due to an AE of lung adenocarcinoma; 2 patients from Cohorts 1 and 2 had a fatal SAE caused by COVID-19 infection and 1 patient from Cohort 3 had a fatal SAE caused by non-MM cancer; 1 patient in Cohort 3 withdrew from study due to an AE of pancreatic adenocarcinoma; and 1 patient in Cohort 5 experienced a fall and withdrew the study due to deteriorating condition related to SAE.
AE, adverse event; belamaf, belantamab mafodotin; IQR, interquartile range; Q3/4W, every 3 weeks for Cycle 1–8 and every 4 weeks afterwards; Q6/8W, every 6 weeks for Cycle 1–8 and every 8 weeks afterwards; SAE, severe adverse event; SOC, standard of care: bortezomib, lenalidomide and dexamethasone in Cycle 1–8 followed by lenalidomide and dexamethasone from Cycle 9 onwards; VRd, bortezomib, lenalidomide, and dexamethasone.

The most common AEs leading to dose modification were thrombocytopenia, neutropenia and ocular events.

Grade ≥3 ocular events per the Keratopathy and Visual Acuity (KVA) scale ranged from 50–83% across Cohorts (**Table 3**).

Ocular AEs related to belamaf led to dose delays in ≥40% of patients across all cohorts and dose reductions in at most 1 (8%) patient per cohort (**Table 3**).

No patients had a permanent treatment discontinuation related to belamaf-induced ocular AEs.

Table 3. Summary of Ocular Events Across Cohorts

Ocular events	Cohort 1 belamaf 1.9 mg/kg Q3/4W n=12	Cohort 2 belamaf 1.4 mg/kg Q6/8W n=12	Cohort 3 belamaf 1.9 mg/kg Q6/8W n=12	Cohort 4 belamaf 1.0 mg/kg Q3/4W n=14	Cohort 5 belamaf 1.4 mg/kg Q3/4W n=13
Any event, n (%)*	12 (100)	12 (100)	12 (100)	12 (86)	12 (92)
Grade ≥3 ocular events per KVA scale[‡], n (%)	10 (83)	7 (58)	6 (50)	7 (50)	9 (69)
Grade ≥3 keratopathy	9 (75)	4 (33)	3 (25)	7 (50)	5 (39)
Grade ≥3 visual acuity	10 (83)	7 (58)	4 (33)	3 (21)	6 (46)
Grade ≥3 ocular AEs leading to dose reduction of belamaf	1 (8)	0	1 (8)	0	1 (8)
Grade ≥3 ocular AEs leading to belamaf dose delay	10 (83)	7 (58)	5 (42)	7 (50)	9 (69)
Median time to onset of Grade ≥3 ocular event (range), days	67.5 (39–106)	106.0 (64–197)	114.0 (84–246)	72.0 (42–145)	62.0 (22–113)
Worse case post baseline, n (%)					
≥3 line decline in BCVA (better eye)	6 (50)	3 (25)	0	1 (7)	3 (23)
≥3 line decline in BCVA (worse eye)	9 (75)	5 (42)	3 (25)	2 (14)	5 (38)

*There were no permanent discontinuations of belamaf due to ocular AEs. [‡]KVA scale for ocular events uses both ophthalmic examination findings and visual acuity for grading of events.
AE, adverse event; BCVA, best corrected visual acuity; belamaf, belantamab mafodotin; ISS, International Staging System; KVA, Keratopathy and Visual Acuity; Q3/4W, every 3 weeks for Cycle 1–8 and every 4 weeks afterwards; Q6/8W, every 6 weeks for Cycle 1–8 and every 8 weeks afterwards; SOC, standard of care: bortezomib, lenalidomide, and dexamethasone in Cycle 1–8 followed by lenalidomide and dexamethasone from Cycle 9 onwards.

Efficacy

Preliminary data on efficacy of belamaf combination treatments are encouraging.

ORR ranged from 80–100% across cohorts, with at least 67% of patients in each cohort achieving ≥VGPR (**Table 4**).

Table 4. Efficacy Data for Patients Treated With Belamaf + VRd

Clinical response	Cohort 1 belamaf 1.9 mg/kg Q3/4W +VRd n=12	Cohort 2 belamaf 1.4 mg/kg Q6/8W +VRd n=12	Cohort 3 belamaf 1.9 mg/kg Q6/8W +VRd n=12	Cohort 4 belamaf 1.0 mg/kg Q3/4W +VRd n=15	Cohort 5 belamaf 1.4 mg/kg Q3/4W +VRd n=13
ORR, n (%; 95% CI)	12 (100; 73.5–100)	11 (92; 61.5–99.8)	12 (100; 73.5–100)	12 (80; 57.2–98.2)	12 (92; 64.0–99.8)
sCR, n (%)	6 (50)	1 (8)	0	3 (20)	2 (15)
CR, n (%)	3 (25)	0	2 (17)	2 (13)	1 (8)
VGPR, n (%)	2 (17)	9 (75)	7 (58)	5 (33)	8 (62)
Median time (months) to VGPR (95% CI)	2.8 (0.7–3.5)	2.1 (0.8–4.9)	2.9 (0.8–3.5)	2.7 (1.4–3.3)	2.2 (0.8–3.0)
≥VGPR[†], n (%; 95% CI)	11 (92; 61.5–99.8)	10 (83; 51.6–97.9)	9 (75; 42.8–94.5)	10 (67; 41.9–91.6)	11 (85; 54.6–98.1)
MRD negativity[‡], n (%; 95% CI)	10 (91; 58.7–99.8) 10 patients tested	2 (20; 2.5–55.6) 6 patients tested	1 (11; 0.3–48.2) 4 patients tested	2 (20; 2.5–55.6) 4 patients tested	2 (18; 2.3–51.8) 3 patients tested
PR, n (%)	1 (8)*	1 (8)	3 (25)	2 (13)	1 (8)
SD, n (%)	0	1 (8)	0	0	0
NE, n (%)	0	0	0	1 (7)	1 (8)

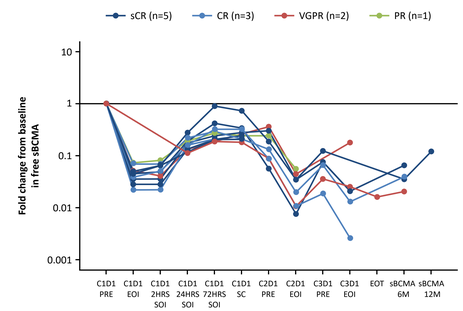
*Patient later achieved best confirmed response by the investigator of VGPR based on the real-time clinical database. [†]sCR+CR+VGPR. [‡]MRD negativity rates were only assessed in patients with best response of ≥VGPR; not all eligible patients had been assessed for MRD status at the time of data cutoff and repeat testing had not been conducted in Cohorts 2–5.
CI, confidence interval; CR, complete response; ORR, overall response rate; PR, partial response; Q3/4W, every 3 weeks for Cycle 1–8 and every 4 weeks afterwards; Q6/8W, every 6 weeks for Cycle 1–8 and every 8 weeks afterwards; sCR, stringent complete response; SD, stable disease; SOC, standard of care: bortezomib, lenalidomide and dexamethasone in Cycle 1–8 followed by lenalidomide and dexamethasone from Cycle 9 onwards; NE, not evaluable; VGPR, very good partial response; VRd, bortezomib, lenalidomide, and dexamethasone.

Impact of Belamaf on sBCMA Levels (Cohort 1 Only, Data Cutoff 11 August 2021)

All patients in Cohort 1 (belamaf 1.9 mg/kg Q3/4W + VRd) with evaluable data exhibited a decrease in circulating free (s)BCMA serum levels at the end of infusion compared to pre-infusion levels observed in time points assessed; sBCMA is plotted in logarithmic scale (**Figure 2**).

- Data for Cohorts 2–5 are currently not available.

Figure 2. Fold Change from Baseline in Free sBCMA Concentration Across Time by Patient and Best Confirmed Response in Cohort 1



Includes only patients with baseline and post-baseline sBCMA measurements. Excludes one patient with best confirmed response by investigator of sCR but without any available measurements for sBCMA. sBCMA assessment time points: C1D1-PRE, within 30 min prior to start of infusion on Cycle 1 Day 1; C1D1 EOI, within 15 min after end of infusion on Cycle 1 Day 1; C1D1-2hrs SOI, 2 hours (± 15 min) after start of infusion on Cycle 1 Day 1; C1D1-24hrs SOI, 24 hours after start of infusion on Cycle 1 Day 1; C1D1-72hrs SOI, 72 hours after start of infusion on Cycle 1 Day 1; C1D1-SC, Cycle 1 Day 1 scheduled collection (Day 8–Day 15); C2D1-PRE, within 30 min prior to start of infusion on Cycle 2 Day 1; C2D1-EOI, within 15 min after end of infusion on Cycle 2 Day 1; C3D1-PRE, within 30 min prior to start of infusion on Cycle 3 Day 1; C3D1-EOI, within 15 min after end of infusion on Cycle 3 Day 1; EOT, end of treatment; sBCMA- 6M, 6 months after achieving ≥VGPR; sBCMA-12M, 12 months after achieving ≥VGPR. CR, complete response; EOI, end of infusion; SC, scheduled collection; sCR, stringent complete response; SOI, start of infusion; PR, partial response; Rd, lenalidomide and dexamethasone; sBCMA, soluble B-cell maturation agent; VGPR, very good partial response.

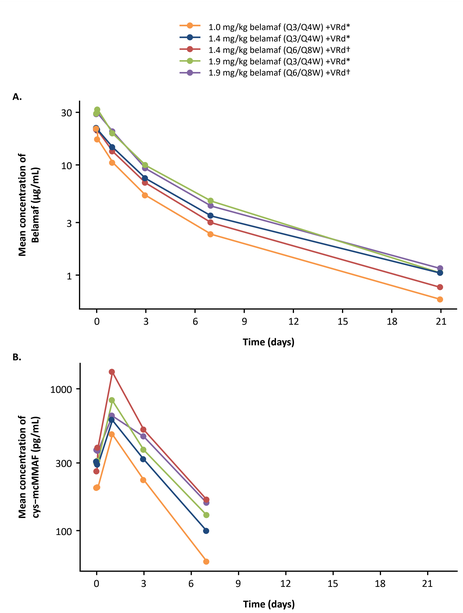
Belamaf Pharmacokinetics

First-cycle pharmacokinetics of belamaf and cys-mcMMAF were explored in all cohorts (**Figure 3**).

Lower belamaf and cys-mcMMAF first-cycle exposures were observed at lower doses.

Belamaf pharmacokinetics in 1L NDMM patients receiving belamaf + VRd were as anticipated from monotherapy belamaf pharmacokinetics, accounting for differences in baseline patient characteristics.[8,9]

Figure 3. Pharmacokinetics of Belamaf and cys-mcMMAF Across Cohorts



*VRd administered at every treatment cycle. †VRd administered at every other treatment cycle.
Belamaf, belantamab mafodotin; cys-mcMMAF, cysteine-maleimidocaproyl monomethyl auristatin F; h, hours; Q3/4W, every 3 weeks for Cycle 1–8 and every 4 weeks afterwards; Q6/8W, every 6 weeks for Cycle 1–8 and every 8 weeks afterwards; VRd, bortezomib, lenalidomide and dexamethasone.

CONCLUSIONS

Preliminary data suggest the combination of belamaf and VRd in patients with TI NDMM resulted in a safety profile consistent with the known profile of belamaf in patients with RRMM.[3,4]

- Lower rates of Grade ≥3 ocular events (KVA scale) were observed in the cohorts with extended dose schedules and lower doses, while maintaining high ORR.

Belamaf + VRd demonstrated high response rates in patients with TI NDMM, with deep responses of ≥VGPR achieved in more than half of all patients in each cohort (67–92%).

- All 10 patients with ≥VGPR who were tested for MRD negativity in Cohort 1 were MRD-negative.

Belamaf PK profile was similar to that observed in patients with RRMM taking into consideration baseline patient characteristics.

Additional patients will be recruited for Cohorts 6–8, and further follow-up is anticipated for Cohorts 1–5, to confirm safety and efficacy of belamaf + VRd in patients with TI NDMM.[10]

ACKNOWLEDGEMENTS

GSK (Study 209664); Drug linker technology licensed from Seagen Inc; Belamaf monoclonal antibody produced using POTELLIGENT Technology licensed from BioWa.

The authors thank Ling Du, PhD (GSK, Waltham, MA) for MRD data and Dawson Knoblock, PhD (GSK, Upper Providence, PA) for sBCMA data.

On behalf of all authors, and with their permission, an audio recording of this poster was prepared by Saad Usmani, who did not receive any payment for this recording.

Writing support provided by Sharon Bryant, DPT, and Taylor Sells, MS, of Fishawack Indicia Ltd, part of Fishawack Health, and funded by GSK.

DISCLOSURES

SU received consulting fees/honoraria from Celgene, Amgen, Janssen Oncology, Seagen Inc, Takeda, GSK, Karyopharm Therapeutics, AbbVie and Skyline Diagnostics; research funding from Celgene, Array BioPharma, Janssen Oncology, Pharmacyclics, Sanofi, BMS, Amgen and Seagen Inc. **MM**, **AAA**, **CKM**, **AOA**, **DW**, and **MH** report no disclosures. **YK** received consulting fees from Janssen, served as an advisor/expert for Pfizer, and received honoraria from Janssen, AstraZeneca, Novartis, GSK, Roche, Pfizer and Takeda. **XL** received honoraria from Amgen, BMS/Celgene, Janssen, Takeda, Novartis, Sanofi, Merck, Oncopeptide, Karyopharm, Roche, Abbvie, Carsgen, GSK and Harpoon Therapeutics. **HQ** received consulting fees/honoraria from GSK, Karyopharm, Amgen, Celgene and Janssen Cilag; grant funding from Celgene, Sanofi and Amgen. **WJ** received consulting fees from Celgene, AstraZeneca, Janssen and Amgen. **MG** received honoraria from Amgen, Janssen, Novartis, Sanofi and Takeda; travel support/conference fees from Amgen, BMS, Janssen, Sanofi and Takeda. **IS** received honoraria from Celgene/BMS, Janssen, Amgen, Takeda and Sanofi. **EMO** received consultancy fees/honoraria from Janssen, BMS, Sanofi, GSK, Oncopeptides, Amgen, Takeda, Secura-Bio, MSD, Pfizer and Karyopharm. **AO** served as an advisor/expert for Amgen, BMS/Celgene, Sanofi and GSK; received consulting fees from Amgen, BMS/Celgene, Sanofi, Janssen and GSK. **PRO** served as an advisor/expert for Amgen, BMS/Celgene, Sanofi and GSK; received honoraria from Janssen, Celgene, Oncopetides, Sanofi, Abbvie, GSK and Kite Pharma. **KR** served as an advisor/expert for Janssen, Celgene, Amgen, Takeda, Oncopeptides, Sanofi, AbbVie, GSK, Karyopharm, Pfizer and adaptive Biotech; received honoraria from Janssen, Celgene, Amgen, Takeda, Oncopeptides, Sanofi, AbbVie, GSK, Karyopharm, Pfizer, adaptive Biotech; research funding from Janssen, Celgene, Amgen, Takeda and GSK. **KW** received consulting fees/honoraria from Adaptive Biotec, Amgen, BMS, Celgene, GSK, Janssen, Karyopharm, Takeda and Sanofi; research funding from Amgen, Celgene, Janssen and Sanofi. **BB** received honoraria from GSK, BMS and Janssen. **MC** received consultancy fees/honoraria from Janssen, Celgene, BMS, Sanofi, Amgen, and GSK. **XZ**, **MCK**, **CB**, **DW**, **FC** and **BEK** are employees of GSK. **IVG** is an employee of GSK and owns stock/shares in GSK and Novartis.

REFERENCES

1. Durie BGM, et al. *Lancet.* 2017;389:519–27.
2. Durie BGM, et al. *Blood Cancer J.* 2020;10:53.
3. Tai YT, et al. *Blood.* 2014;123:3128–38.
4. Tai YT, Anderson KC. *Immunotherapy.* 2015;7:1187.
5. Montes de Oca R et al. *Mol Cancer Ther.* 2021;20:1941–55.
6. GSK data on file.
7. Usmani S, et al. Presented at American Society of Hematology Meeting 2021. Poster #2738.
8. Rathi C, et al. *CPT Pharmacometrics Syst Pharmacol.* 2021:851–63.
9. Ferron-Brady G, et al. *Clin Pharmacol Ther.* 2021:1281–92.
10. ClinicalTrials.gov. NCT04091126. https://clinicaltrials.gov/ct2/show/NCT04091126. Accessed May 9, 2022.

CONTACT INFORMATION

usmanis@mskcc.org