Idecabtagene vicleucel versus standard regimens in patients with triple-class-exposed relapsed and refractory multiple myeloma: a KarMMa-3 analysis in high-risk subgroups

MM-388

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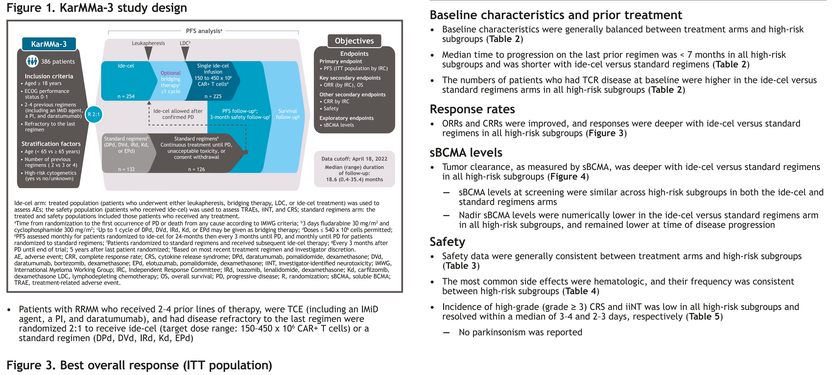
Introduction

- Despite improvements in the treatment landscape of relapsed and refractory multiple myeloma (RRMM) with the use of combination therapy, the introduction of immunomodulatory (IMiD®) agents, proteasome inhibitors (PIs), and anti-CD38 monoclonal antibodies in frontline and early relapse settings results in patients becoming triple-class-exposed (TCE) earlier in their treatment course[1-3]
- Outcomes in patients with high-risk disease characteristics such as cytogenetic abnormalities, advanced disease stage, high tumor burden, presence of extramedullary plasmacytoma (EMP), and triple-class-refractory (TCR) disease remain poor[4]
- Idecabtagene vicleucel (ide-cel), a B-cell maturation antigen (BCMA)–directed chimeric antigen receptor (CAR) T cell therapy, significantly improved median progression-free survival (PFS; 13.3 vs 4.4 months; HR, 0.49; 95% CI, 0.38-0.65; *P* < 0.001) and overall response rate (ORR; 71% vs 42%; *P* < 0.001) versus standard regimens in the overall population of patients with TCE RRMM in KarMMa-3 (NCT03651128)[5]
- Here, we assess the efficacy and safety of ide-cel versus standard regimens in patients with high-risk disease characteristics in KarMMa-3

Methods

- The full details of the KarMMa-3 study design have been previously presented[6] (**Figure 1**)

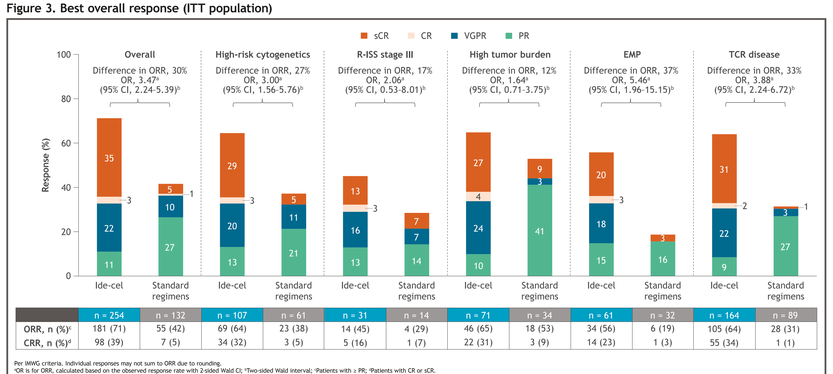
Figure 1. KarMMa-3 study design



Ide-cel arm: treated population (patients who underwent either leukapheresis, bridging therapy, LDC, or ide-cel treatment) was used to assess AEs; the safety population (patients who received ide-cel) was used to assess TRAEs, IINT, and CRS; standard regimens arm: the treated and safety populations included those patients who received any treatment.
*Time from randomization to the first occurrence of PD or death from any cause according to IMWG criteria; †3 days fludarabine 30 mg/m² and cyclophosphamide 300 mg/m²; ‡Up to 1 cycle of DPd, DVd, IRd, Kd, or EPd may be given as bridging therapy; §Doses ≥ 540 x 10⁶ cells permitted; ¶PFS assessed monthly for patients randomized to ide-cel for 24 months then every 3 months until PD, and monthly until PD for patients randomized to standard regimens; #Patients randomized to standard regimens and received subsequent ide-cel therapy: every 2 months after PD until end of trial; $ years after last patient randomized; ‖Based on most recent treatment regimen and investigator discretion.
AE, adverse event; CRR, complete response rate; CRS, cytokine release syndrome; DPd, daratumumab, pomalidomide, dexamethasone; DVd, daratumumab, bortezomib, dexamethasone; EPd, elotuzumab, pomalidomide, dexamethasone; IINT, Investigator-identified neurotoxicity; IMWG, International Myeloma Working Group; IRC, Independent Response Committee; IRd, ixazomib, lenalidomide, dexamethasone; Kd, carfilzomib, dexamethasone; LDC, lymphodepleting chemotherapy; OS, overall survival; PD, progressive disease; R, randomization; sBCMA, soluble BCMA; TRAE, treatment-related adverse event.

- Patients with RRMM who received 2-4 prior lines of therapy, were TCE (including an IMiD agent, a PI, and daratumumab), and had disease refractory to the last regimen were randomized 2:1 to receive ide-cel (target dose range: 150-450 x 10⁶ CAR+ T cells) or a standard regimen (DPd, DVd, IRd, Kd, EPd)

Figure 3. Best overall response (ITT population)



	Overall		High-risk cytogenetics		R-ISS stage III		High tumor burden		EMP		TCR disease	
	n = 254	n = 132	n = 107	n = 61	n = 31	n = 14	n = 71	n = 34	n = 61	n = 32	n = 164	n = 89
ORR, n (%)ᶜ	181 (71)	55 (42)	69 (64)	23 (38)	14 (45)	4 (29)	46 (65)	18 (53)	34 (56)	6 (19)	105 (64)	28 (31)
CRR, n (%)ᵈ	98 (39)	7 (5)	34 (32)	3 (5)	5 (16)	1 (7)	22 (31)	3 (9)	14 (23)	1 (3)	55 (34)	1 (1)

Per IMWG criteria. Individual responses may not sum to ORR due to rounding.
ªOR is for ORR, calculated based on the observed response rate with 2-sided Wald CI; ᵇTwo-sided Wald interval; ᶜPatients with ≥ PR; ᵈPatients with CR or sCR.
CR, complete response; OR, odds ratio; PR, partial response; sCR, stringent complete response; VGPR, very good partial response.

- In this analysis, efficacy and safety were assessed in high-risk subgroups (**Table 1**)
- An analysis was also performed to determine the sBCMA levels in high-risk subgroups at screening, at nadir, and at disease progression

Table 1. High-risk subgroup criteria

High-risk characteristic	High-risk cytogenetics		R-ISS stage III disease		High tumor burden		EMP		TCR disease	
Patients	Ide-cel (n = 107)	Standard regimens (n = 61)	Ide-cel (n = 31)	Standard regimens (n = 14)	Ide-cel (n = 71)	Standard regimens (n = 34)	Ide-cel (n = 61)	Standard regimens (n = 32)	Ide-cel (n = 164)	Standard regimens (n = 89)
Definition	Del(17p), t(4;14), or t(14;16)		Serum B2-microglobulin ≥ 5.5 mg/L and either high-risk chromosomal abnormalities by FISH OR high LDH[a]		≥ 50% CD138+ plasma cells in bone marrow		Soft-tissue-only and soft-tissue bone-related plasmacytomas		Refractory to ≥ 1 each of an IMiD agent, a PI, and an anti-CD38 antibody	

FISH, fluorescence in situ hybridization; LDH, lactate dehydrogenase; R-ISS, revised International Staging System.

Results

Baseline characteristics and prior treatment

- Baseline characteristics were generally balanced between treatment arms and high-risk subgroups (**Table 2**)
- Median time to progression on the last prior regimen was < 7 months in all high-risk subgroups and was shorter with ide-cel versus standard regimens (**Table 2**)
- The numbers of patients who had TCR disease at baseline were higher in the ide-cel versus standard regimens arms in all high-risk subgroups (**Table 2**)

Response rates

- ORRs and CRRs were improved, and responses were deeper with ide-cel versus standard regimens in all high-risk subgroups (**Figure 3**)

sBCMA levels

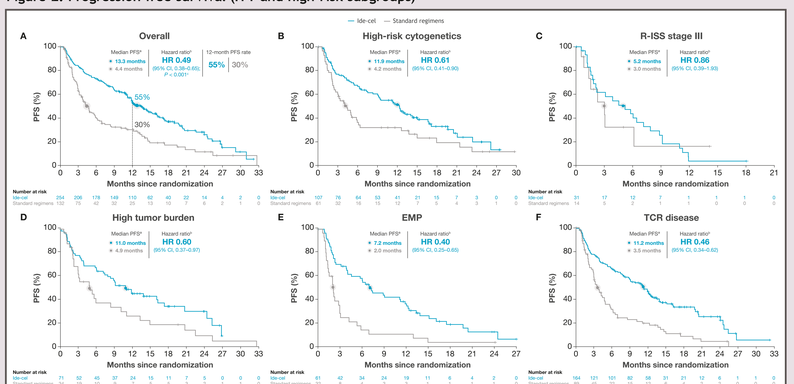
- Tumor clearance, as measured by sBCMA, was deeper with ide-cel versus standard regimens in all high-risk subgroups (**Figure 4**)
  - sBCMA levels at screening were similar across high-risk subgroups in both the ide-cel and standard regimens arms
  - Nadir sBCMA levels were numerically lower in the ide-cel versus standard regimens arm in all high-risk subgroups, and remained lower at time of disease progression

Safety

- Safety data were generally consistent between treatment arms and high-risk subgroups (**Table 3**)
- The most common side effects were hematologic, and their frequency was consistent between high-risk subgroups (**Table 4**)
- Incidence of high-grade (grade ≥ 3) CRS and iINT was low in all high-risk subgroups and resolved within a median of 3-4 and 2-3 days, respectively (**Table 5**)
  - No parkinsonism was reported

Median PFS was longer in patients treated with ide-cel versus standard regimens in the overall population and high-risk subgroups; interpretation in patients with R-ISS stage III disease was limited due to small subgroup size (Figure 2)

Figure 2. Progression-free survival (ITT and high-risk subgroups)



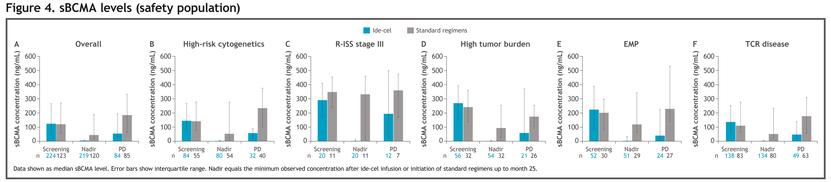
PFS was analyzed in the ITT population of all randomized patients in both arms and included early PFS events occurring between randomization and ide-cel infusion.
PFS based on IMWG criteria per IRC. ªBased on Kaplan-Meier approach; ᵇUnstratified HR based on univariate Cox proportional hazard model. CI is 2-sided; ᶜBased on stratified log-rank test.

Table 2. Baseline demographics and disease characteristics

	Overall[a]		High-risk cytogenetics		R-ISS stage III		High tumor burden		EMP		TCR disease	
	Ide-cel (n = 254)	Standard regimens (n = 132)	Ide-cel (n = 107)	Standard regimens (n = 61)	Ide-cel (n = 31)	Standard regimens (n = 14)	Ide-cel (n = 71)	Standard regimens (n = 34)	Ide-cel (n = 61)	Standard regimens (n = 32)	Ide-cel (n = 164)	Standard regimens (n = 89)
Median age, years (range)	63.0 (30-81)	63.0 (42-83)	61.0 (34-81)	63.0 (42-83)	63.0 (48-72)	63.5 (51-82)	62.0 (30-76)	63.0 (46-83)	62.0 (34-78)	59.0 (42-82)	62.0 (30-78)	62.0 (42-83)
Male, n (%)	156 (61)	79 (60)	63 (59)	39 (64)	14 (45)	12 (86)	41 (58)	21 (62)	37 (61)	20 (63)	97 (59)	55 (62)
Median time since diagnosis, years (range)	4.1 (0.6ᵇ-21.8)	4.0 (0.7ᵇ-17.7)	3.6 (0.8-13.1)	3.8 (1.1-12.4)	3.8 (1.2-8.8)	3.2 (0.7-9.1)	3.8 (0.6-15.3)	3.6 (0.7-12.1)	3.4 (0.8-8.1)	3.3 (0.7-11.4)	3.9 (0.6-21.8)	4.0 (0.7ᵇ-17.7)
High tumor burden, n (%)	71 (28)	34 (26)	40 (37)	21 (34)	17 (55)	8 (57)	71 (100)	34 (100)	16 (26)	8 (25)	53 (32)	22 (25)
Extramedullary disease, n (%)	61 (24)	32 (24)	24 (22)	14 (23)	10 (32)	5 (36)	16 (23)	8 (24)	61 (100)	32 (100)	48 (29)	25 (28)
ECOG performance status, n (%)[b]												
0	120 (47)	66 (50)	50 (47)	30 (49)	9 (29)	4 (29)	28 (39)	19 (56)	18 (30)	14 (44)	77 (47)	41 (46)
1	133 (52)	62 (47)	56 (52)	28 (46)	22 (71)	8 (57)	43 (61)	14 (41)	43 (71)	17 (53)	86 (52)	44 (49)
R-ISS stage, n (%)[c]												
I	50 (20)	26 (20)	0 (0)	0 (0)	0 (0)	0 (0)	7 (10)	1 (3)	5 (8)	6 (19)	24 (15)	19 (21)
II	150 (59)	82 (62)	84 (79)	48 (79)	0 (0)	0 (0)	46 (65)	24 (71)	42 (69)	18 (56)	108 (66)	53 (60)
III	31 (12)	14 (11)	20 (19)	12 (20)	31 (100)	14 (100)	17 (24)	8 (24)	10 (16)	5 (16)	23 (14)	10 (11)
Unknown	23 (9)	10 (8)	3 (3)	1 (2)	0 (0)	0 (0)	1 (1)	1 (3)	4 (7)	3 (9)	9 (6)	7 (8)
High-risk cytogenetics, n (%)[d]	107 (42)	61 (46)	107 (100)	61 (100)	20 (65)	12 (86)	40 (56)	21 (62)	24 (39)	14 (44)	77 (47)	40 (45)
del(17p)	66 (26)	42 (32)	66 (62)	42 (69)	11 (35)	9 (64)	23 (32)	14 (41)	14 (23)	11 (34)	47 (29)	27 (30)
t(4;14)	43 (17)	18 (14)	43 (40)	18 (30)	5 (16)	4 (29)	15 (21)	6 (18)	11 (18)	2 (6)	31 (19)	12 (14)
t(14;16)	8 (3)	4 (3)	8 (7)	4 (7)	5 (16)	0 (0)	6 (8)	1 (3)	2 (3)	0 (0)	6 (4)	3 (3)
1q gain/amplification, n (%)	125 (49)	51 (39)	63 (59)	28 (46)	17 (55)	8 (57)	49 (69)	17 (50)	30 (49)	10 (31)	87 (53)	35 (39)
Median (range) number of prior regimens	3 (2-4)	3 (2-4)	3 (2-4)	3 (2-4)	3 (2-4)	3 (2-4)	3 (2-4)	3 (2-4)	3 (2-4)	3 (2-4)	3 (2-4)	3 (2-4)
Previous autologous HSCT, n (%)	214 (84)	114 (86)	85 (79)	53 (87)	22 (71)	11 (79)	57 (80)	29 (85)	51 (84)	27 (84)	135 (82)	74 (83)
1 transplantation	167 (66)	87 (66)	65 (61)	39 (64)	16 (52)	10 (71)	46 (65)	20 (59)	41 (67)	23 (72)	102 (62)	58 (65)
> 1 transplantation	47 (19)	27 (20)	20 (19)	14 (23)	6 (19)	1 (7)	11 (15)	9 (26)	10 (16)	4 (13)	33 (20)	16 (18)
Median (range) time to progression on last prior antimyeloma therapy, months	7.1 (0.7-67.7)	6.9 (0.4-66.0)	5.5 (1.0-46.5)	6.9 (0.4-66.0)	3.9 (1.1-18.2)	3.5 (1.0-40.6)	5.1 (1.0-42.6)	6.2 (1.1-44.6)	5.1 (1.0-44.0)	5.1 (0.4-27.0)	5.6 (1.0-42.6)	5.8 (0.4-66.0)
Prior radiation therapy, n (%)	90 (35)	46 (35)	39 (36)	20 (33)	12 (39)	5 (36)	22 (31)	8 (24)	33 (54)	20 (63)	64 (39)	33 (37)
Refractory status, n (%)												
IMiD agent refractory	224 (88)	124 (94)	96 (90)	59 (97)	26 (84)	13 (93)	66 (93)	32 (94)	54 (89)	31 (97)	164 (100)	89 (100)
PI refractory	189 (74)	95 (72)	84 (79)	43 (70)	29 (94)	11 (79)	58 (82)	25 (74)	56 (92)	26 (81)	164 (100)	89 (100)
Daratumumab refractory[e]	242 (95)	123 (93)	104 (97)	56 (92)	30 (97)	12 (86)	68 (96)	29 (85)	59 (97)	30 (94)	164 (100)	88 (99)
Double-class refractory[f]	169 (67)	91 (69)	78 (73)	42 (69)	24 (77)	11 (79)	55 (77)	24 (71)	50 (82)	25 (78)	164 (100)	89 (100)
Triple-class refractory[g]	164 (65)	89 (67)	77 (72)	40 (66)	23 (74)	10 (71)	53 (75)	22 (65)	48 (79)	25 (78)	164 (100)	89 (100)
Penta-class refractory[h]	15 (6)	5 (4)	8 (7)	4 (7)	0 (0)	0 (0)	3 (4)	1 (3)	3 (5)	2 (6)	15 (9)	5 (6)

ªThe minimum value shown represents the second lowest value in the database; the lowest value was determined to be a data entry error that was corrected in the database after database lock. The patient whose data was incorrectly entered has a time from initial diagnosis to screening of 3.2 years, which was incorrectly reported as 0.2 years; ᵇAll patients had score 0 or 1 at screening but may be > 1 at baseline; ᶜISS was calculated using baseline values of albumin and β2-microglobulin. R-ISS was derived using baseline ISS stage, cytogenetic abnormality, and serum LDH; ᵈIncluded del(17p), t(4;14), or t(14;16); ᵉ1 patient in each arm was refractory to isatuximab; ᶠRefractory to ≥ 1 each of an IMiD agent and a PI; ᵍRefractory to ≥ 1 each of an IMiD agent, a PI, and an anti-CD38 antibody; ʰRefractory to lenalidomide, pomalidomide, bortezomib, carfilzomib, and daratumumab.
HSCT, hematopoietic stem cell transplantation.

Figure 4. sBCMA levels (safety population)



Data shown as median sBCMA level. Error bars show interquartile range. Nadir equals the minimum observed concentration after ide-cel infusion or initiation of standard regimens up to month 25.

Table 3. Safety summary (treated population)

	Overall[a]		High-risk cytogenetics		R-ISS stage III		High tumor burden		EMP		TCR disease	
	Ide-cel (n = 250)	Standard regimens (n = 126)	Ide-cel (n = 104)	Standard regimens (n = 56)	Ide-cel (n = 29)	Standard regimens (n = 12)	Ide-cel (n = 68)	Standard regimens (n = 33)	Ide-cel (n = 59)	Standard regimens (n = 31)	Ide-cel (n = 160)	Standard regimens (n = 85)
Patients with any-grade AE	248 (99)	123 (98)	102 (98)	56 (100)	28 (97)	12 (100)	67 (99)	32 (97)	59 (100)	29 (94)	158 (99)	83 (98)
Patients with grade 3/4 AE	233 (93)	94 (75)	96 (92)	45 (80)	26 (90)	12 (100)	63 (93)	27 (82)	57 (97)	21 (68)	148 (93)	61 (72)
Patients with grade 5 AE	36 (14)	8 (6)	20 (19)	4 (7)	6 (21)	2 (17)	13 (19)	2 (6)	11 (19)	2 (6)	24 (15)	7 (8)
Patients with any-grade serious AE	130 (52)	48 (38)	62 (60)	22 (39)	17 (59)	9 (75)	37 (54)	12 (36)	38 (64)	12 (39)	89 (56)	32 (38)

Data are n n (%).

Table 4. Grade 3/4 AEs (treated population)

	Overall[a]		High-risk cytogenetics		R-ISS stage III		High tumor burden		EMP		TCR disease	
Grade 3/4 AEs occurring in ≥ 10% of patients, n (%)[b,c]	Ide-cel (n = 250)	Standard regimens (n = 126)	Ide-cel (n = 104)	Standard regimens (n = 56)	Ide-cel (n = 29)	Standard regimens (n = 12)	Ide-cel (n = 68)	Standard regimens (n = 33)	Ide-cel (n = 59)	Standard regimens (n = 31)	Ide-cel (n = 160)	Standard regimens (n = 85)
Any grade 3/4 event	233 (93)	94 (75)	96 (92)	45 (80)	26 (90)	12 (100)	63 (93)	27 (82)	57 (97)	21 (68)	148 (93)	61 (72)
Hematologic	218 (87)	75 (60)	89 (86)	36 (64)	24 (83)	9 (75)	59 (87)	22 (67)	54 (92)	17 (55)	134 (84)	46 (54)
Neutropenia	189 (76)	50 (40)	74 (71)	23 (41)	16 (55)	4 (33)	48 (71)	15 (45)	45 (76)	9 (29)	106 (66)	26 (31)
Anemia	127 (51)	23 (18)	58 (56)	8 (14)	8 (28)	5 (42)	41 (60)	7 (21)	4 (7)	3 (10)	14 (9)	5 (6)
Thrombocytopenia	106 (42)	22 (17)	44 (42)	12 (21)	12 (41)	5 (42)	37 (54)	7 (21)	22 (37)	4 (13)	54 (34)	13 (15)
Leukopenia	71 (28)	11 (9)	26 (25)	4 (7)	6 (21)	2 (17)	21 (31)	3 (9)	21 (36)	1 (3)	42 (26)	5 (6)
Lymphopenia	70 (28)	23 (18)	20 (19)	8 (14)	6 (21)	3 (25)	15 (22)	8 (24)	15 (25)	4 (13)	39 (24)	10 (12)
Non-hematologic												
Infections and infestations	61 (24)	23 (18)	10 (10)	6 (11)	2 (7)	3 (25)	18 (26)	7 (21)	4 (7)	3 (10)	18 (11)	7 (8)
Hypophosphatemia	50 (20)	3 (2)	5 (5)	0	1 (3)	0	13 (19)	2 (6)	2 (3)	0	8 (5)	2 (2)
Hypertension	22 (9)	4 (3)	3 (3)	2 (4)	0	0	9 (13)	2 (6)	2 (3)	1 (3)	3 (2)	2 (2)
CRS	10 (4)	0	2 (2)	0	0	0	7 (10)	0	0	0	4 (3)	0
Acute kidney injury	8 (3)	3 (2)	3 (3)	2 (4)	1 (3)	0	3 (4)	2 (6)	2 (3)	0	3 (2)	1 (1)
Atrial fibrillation	5 (2)	3 (2)	2 (2)	1 (2)	3 (10)	1 (8)	1 (1)	1 (3)	1 (2)	0	0	1 (1)

[a]In either treatment arm of any high-risk subgroup. In treated population (ide-cel arm: patients who underwent either leukapheresis, bridging therapy, LDC, or ide-cel treatment; standard regimens arm: patients who received any dose of study treatment). In standard regimens arm, for the 69 patients who underwent leukapheresis in preparation for planned ide-cel treatment upon documented PD on standard regimen, only AEs before leukapheresis were included. All AEs were assessed from collection of informed consent for a minimum of 6 months after initiation of study treatment; grade ≥ 3 events continued to be recorded from month 7 until 28 days after PFS discontinuation visit, or 28 days after end of treatment visit for patients not continuing in PFS follow-up; [b]AEs were coded using Medical Dictionary for Regulatory Activities version 24.1 and graded using Common Terminology Criteria for Adverse Events version 4.03.

Table 5. CRS and iINT in patients treated with ide-cel (safety population)

	Overall[a] (n = 225)	High-risk cytogenetics (n = 84)	R-ISS stage III (n = 20)	High tumor burden (n = 56)	EMP (n = 53)	TCR disease (n = 139)
CRS, n (%)[b]						
Any grade	197 (88)	75 (89)	19 (95)	54 (96)	47 (89)	126 (91)
Grade 3	6 (3)	3 (4)	1 (5)	4 (7)	3 (6)	5 (4)
Grade 4	3 (1)	2 (2)	0	2 (4)	0	3 (2)
Grade 5	2[c] (1)	0	0	2 (4)	0	1 (1)
Median (range) time to first onset, days[c]	1.0 (1-14)	1.0 (1-6)	1.0 (1-6)	1.0 (1-6)	1.0 (1-14)	1.0 (1-5)
Median (range) duration, days[c]	3.5 (1-51)	3.0 (1-30)	3.0 (1-24)	4.0 (1-51)	4.0 (1-16)	4.0 (1-51)
Medications used for CRS management, n (%)						
Tocilizumab	161 (72)	64 (76)	15 (75)	47 (84)	37 (70)	106 (76)
Siltuximab	3 (1)	1 (1)	0	2 (4)	2 (4)	3 (2)
Anakinra	8 (4)	3 (4)	1 (5)	6 (11)	2 (4)	6 (4)
Steroids[d]	64 (28)	26 (31)	2 (10)	20 (36)	17 (32)	39 (28)
iINT, n (%)[e]						
Any grade	34 (15)	17 (20)	3 (15)	11 (20)	7 (13)	25 (18)
Grade 3	5 (2)	4 (5)	0	1 (2)	0	3 (2)
Grade 4	2 (1)	0	0	2 (4)	0	2 (1)
Grade 5	0	0	0	0	0	0
Median (range) time to first onset, days[c]	3.0 (1-317[f])	4.0 (1-317[f])	4.0 (1-5)	4.0 (1-22)	3.0 (1-6)	3.0 (1-22)
Median (range) duration, days[c]	2.0 (1-37)	3.0 (1-30)	2.0 (1-2)	3.0 (1-37)	2.0 (2-14)	2.0 (1-37)
Medications used for iINT management, n (%)						
Steroids	15 (7)	12 (14)	2 (10)	6 (11)	4 (8)	13 (9)

[a]CRS was graded according to modified Lee's criteria[10]; maximum-grade events are reported. patients could have > 1 event; [b]One of the grade 5 CRS events occurred after a decline in organ function (acute myocardial infarction potentially related to anemia, and rapid atrial flutter; the patient died on day 61 and 1 from concomitant grade 5 *Candida* sepsis; [c]Time to first onset of CRS or iINT · first start date of CRS or iINT · infusion date + 1; [d]Algorithm for duration: if the gap between 2 events is ≤ 1 day, then these 2 events are considered as 1 event regardless of grade change, drug relationship change, or severity change; "Steroid use was not mandated in the protocol and was used based on clinical judgement; [e]iINT includes immune effector cell-associated neurotoxicity syndrome reported by investigator as a neurologic toxicity AE; [f]Grade 2 encephalopathy, unrelated to ide-cel, was reported in 1 patient 317 days after ide-cel infusion, and was considered by the investigator to be related to worsening pneumonia and *C. difficile* colitis, not ide-cel.

Conclusions

- The KarMMa-3 study enrolled a high-risk TCE and highly refractory population of patients with RRMM
- In this subgroups analysis, a single infusion of ide-cel treatment consistently demonstrated clinically meaningful improvement in PFS and ORR versus standard regimens regardless of baseline high-risk disease characteristics
- Nadir levels of sBCMA, a pharmacodynamic marker of tumor clearance, were lower in the ide-cel arm versus the standard regimens arm in all high-risk subgroups
- The toxicity profile of ide-cel in high-risk subgroups was manageable and consistent with the overall population and previous studies,[11,12] and no parkinsonism was reported
- The benefit of ide-cel versus standard regimens observed in KarMMa-3 was maintained across patient populations with difficult-to-treat high-risk RRMM[6]

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