ALMA MATER STUDIORUM
UNIVERSITÀ DI BOLOGNA

Highlights from IMS 20th Meeting 2023

CAR-T anti-BCMA dopo almeno 1-4 precedenti terapie

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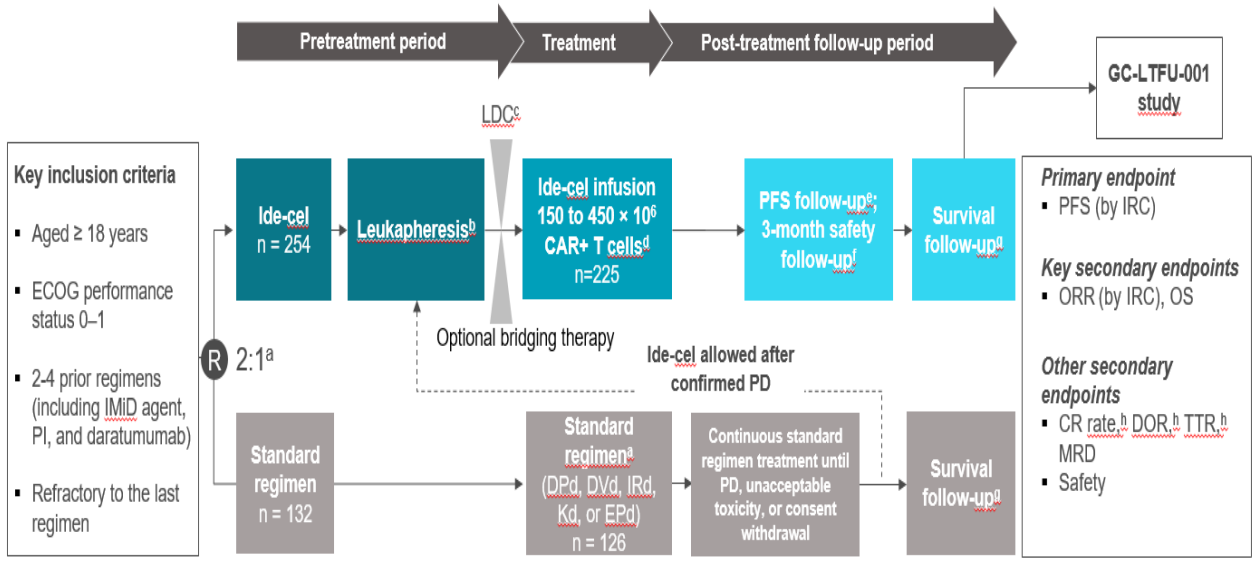
Disclosures: Michele Cavo

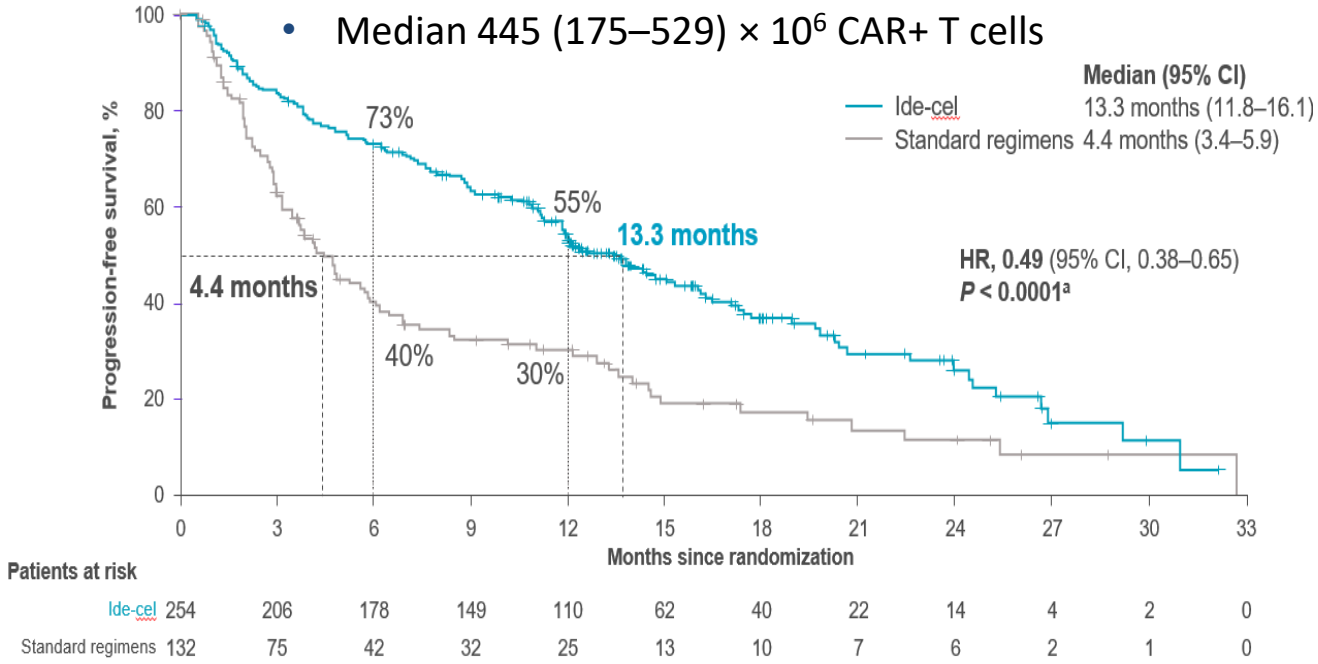
Company name	Research support	Employee	Consultant	Stockholder	Speakers bureau	Advisory board	Other
GlaxoSmithKline			×			×	Honoraria
Janssen			×		×	×	Honoraria
Sanofi			×		×	×	Honoraria
Roche			×			×	Honoraria
Amgen			×			×	Honoraria
Takeda			×			×	Honoraria
AbbVie			×			×	Honoraria
Bristol Myers Squibb			×		×	×	Honoraria
Celgene			×		×	×	Honoraria

KarMMa-3: design, baseline characteristics and PFS



Characteristic	Ide-cel (n = 254)	Standard regimens (n = 132)
Median (range) age, years	63 (30–81)	63 (42–83)
Sex, male, n (%)	156 (61)	79 (60)
Median (range) time from diagnosis to screening, years	4.1 (0.2–21.8)	4.0 (0.7–17.7)
High tumor burden, n (%)[a]	71 (28)	34 (26)
Extramedullary disease, n (%)[b]	61 (24)	32 (24)
Treatment	**Ide-cel (n = 254)**	**Standard regimens (n = 132)**
Median (range) number of prior regimens	3 (2–4)	3 (2–4)
Median (range) time to progression on last prior antimyeloma therapy, months	7.1 (0.7–67.7)	6.9 (0.4–66.0)
Refractory status, n (%)		
IMiD agent refractory	224 (88)	124 (94)
PI refractory	189 (74)	95 (72)
Daratumumab[a]	242 (95)	123 (93)
Double-class refractory[b]	169 (67)	91 (69)
Triple-class refractory[c]	164 (65)	89 (67)

- Median 445 (175–529) × 10^6 CAR+ T cells



Of 213 patients who received bridging therapy, 200 were evaluable for change in disease burden before ide-cel

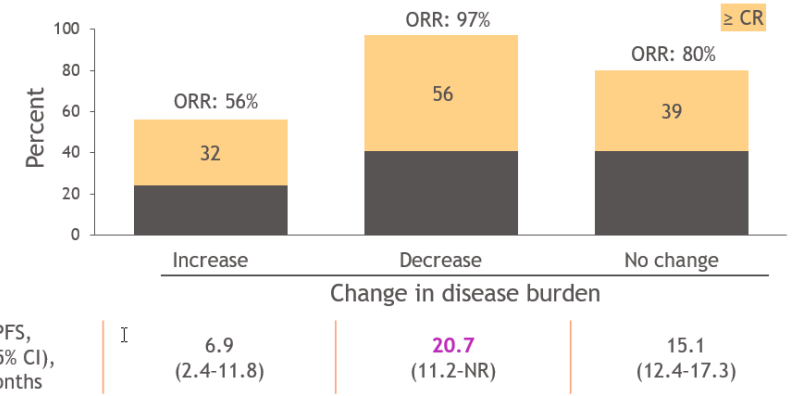
- DPd: 45 patients
- Kd: 26
- DVd: 20
- Epd: 59
- Ird: 25
- Other: 25

No. of cycles, median:
1 for all bridging therapy regimens

Change in disease burden, %		
Increase	Decrease	No change
28	15	51

A higher proportion of patients with disease burden increase, had EMD, high-risk cytogenetics and TCR disease at baseline versus those with disease burden decrease or no change

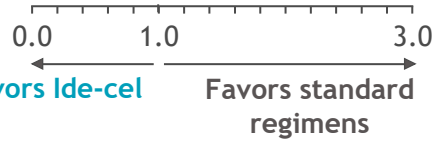
ORR and mPFS were numerically higher in patients with decrease versus increase or no change, with deeper, more durable responses



Rodriguez-Otero P. et al, N Engl J Med 2023; 388:1002-1014;
Einsele H, et al. IMS 2023 encore Poster P008.

PFS: subgroup analysis (ITT population)

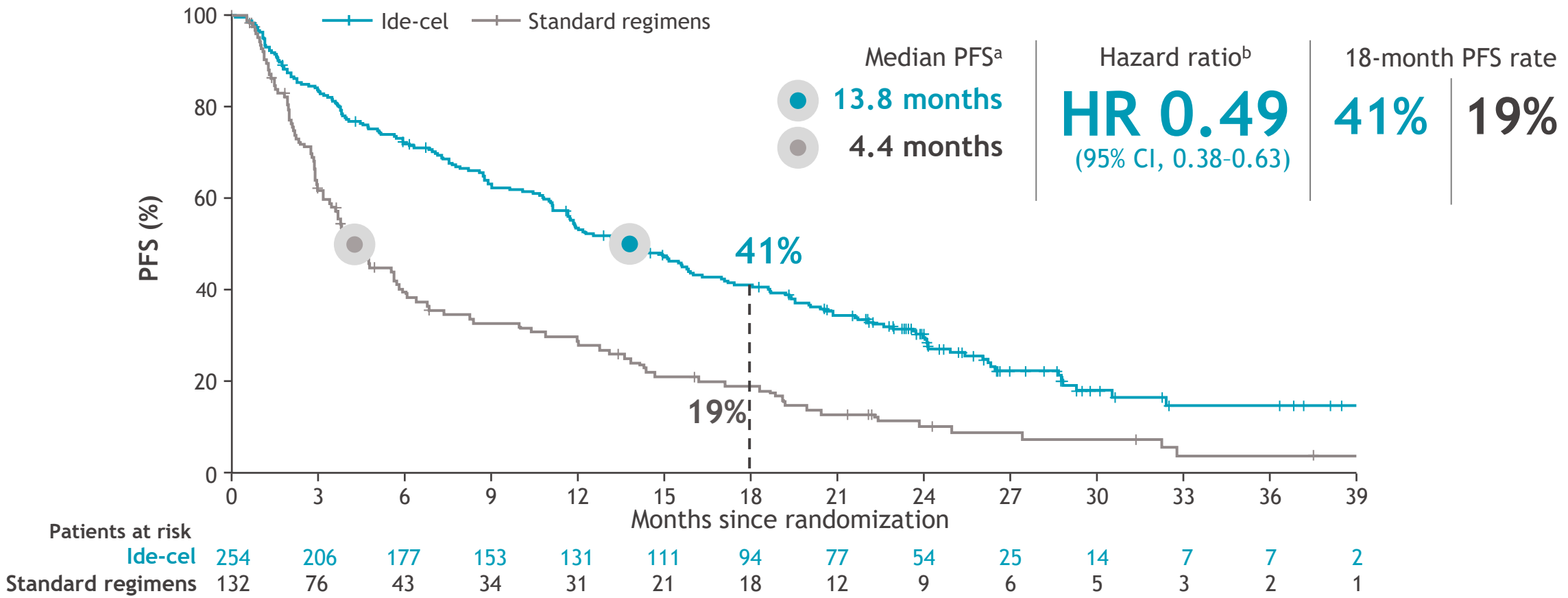
	Ide-cel (n = 254)	Standard regimens (n = 132)	HR
All	149	93	0.51
Sex			
Male	92/156	55/79	0.53
Female	57/98	38/53	0.47
Age group			
< 65	93/150	51/78	0.57
65-74	49/92	36/45	0.42
75-84	7/12	6/9	0.59
Race			
White	101/172	54/78	0.52
Asian	4/7	1/5	NC
Black or African American	8/18	13/18	0.50
Other	2/3	4/4	NC
Region			
North America	84/144	60/82	0.50
Europe	63/106	32/45	0.44
Japan	2/4	1/5	NC
R-ISS stage at baseline			
I or II	113/200	78/108	0.48
III	27/31	8/14	0.86
Tumor burden[a]			
< 50%	99/172	60/90	0.47
≥ 50%	44/71	28/34	0.60
High-risk cytogenetic abnormalities[b]			
Yes	65/107	42/61	0.61
No	84/147	51/71	0.44
Extramedullary plasmacytoma			
Yes	48/61	28/32	0.40
No	100/192	65/100	0.51
Daratumumab refractory			
Yes	143/242	88/123	0.51
No	6/12	5/9	0.40
Double-class refractory			
Yes	106/169	72/91	0.47
No	43/85	21/41	0.65
Triple-class refractory[c]			
Yes	103/164	70/89	0.46
No	46/90	23/43	0.65
Penta-drug refractory[d]			
Yes	12/15	3/5	0.63
No	137/239	90/127	0.49
No. of prior antimyeloma regimens			
2	41/78	26/39	0.51
3	57/95	37/49	0.44
4	51/81	30/44	0.58



The PFS benefit of ide-cel was consistently observed across multiple patient subgroups

Adapted from Rodríguez-Otero P, et al. Ide-cel or standard regimens in relapsed and refractory multiple myeloma. *N Engl J Med* 2023;388:1002–14.
Per IRC based on IMWG criteria. Assumption of proportional hazards was assessed using a treatment*log(time) interaction term in each model. [a]Determined by the higher value between bone marrow aspirate and bone marrow biopsy CD138+ plasma cell. Low: < 50%, High: ≥ 50%; [b]Defined as t(4;14), t(14;16), or del(17p); [c]Refractory to ≥ 1 each of an IMiD, a PI, and an anti-CD38 antibody; [d]Refractory to lenalidomide, pomalidomide, bortezomib, carfilzomib, and daratumumab.
NC, not computed.

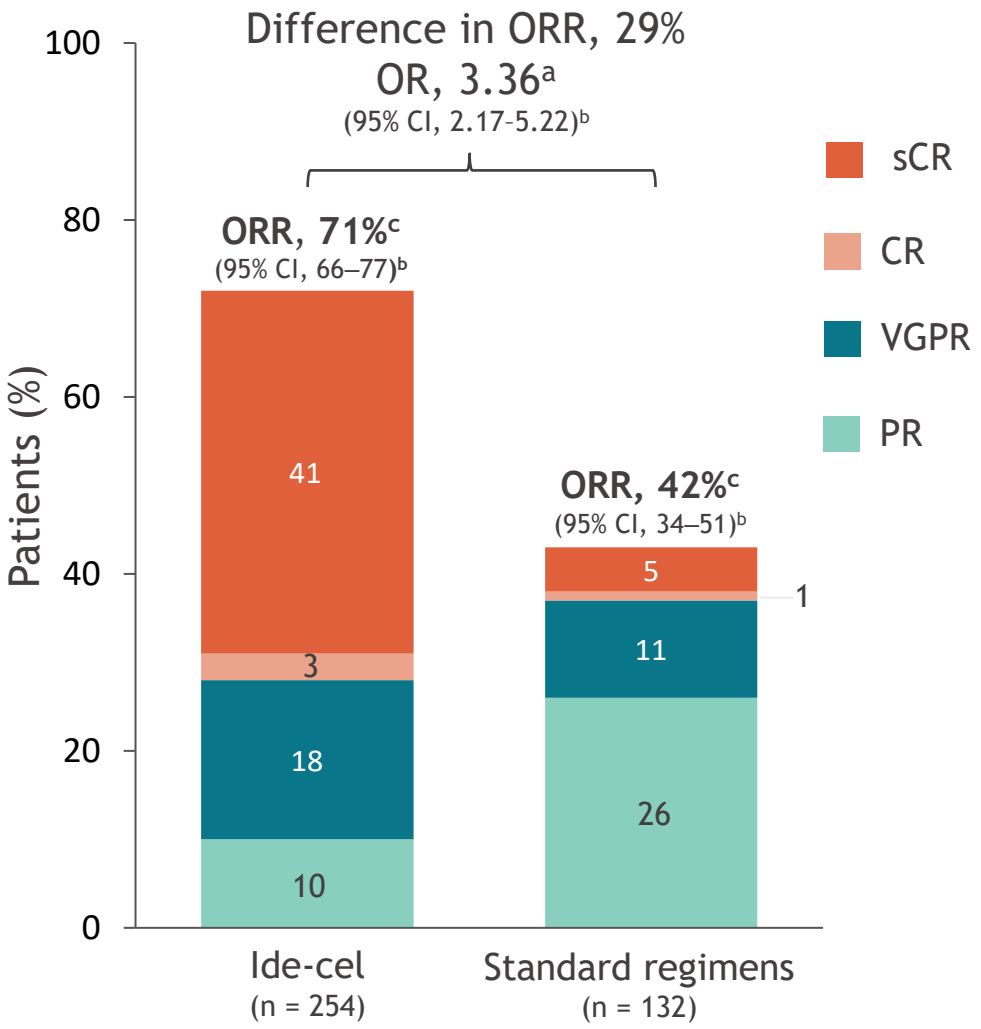
Final PFS analysis at 30.9 months median follow-up



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. [a]Based on Kaplan–Meier approach; [b]Stratified HR based on univariate Cox proportional hazard model. CI is 2-sided.

HR, hazard ratio; ide-cel, idecabtagene vicleucel; IMWG, International Myeloma Working Group; ITT, intent-to-treat; PFS, progression-free survival.

Deep and durable responses with ide-cel



Secondary endpoint	Ide-cel (n = 254)	Standard regimens (n = 132)
CR rate (95 % CI), %[d]	44 (38-50)	5 (2-9)
MRD-negative CR rate, n/N (%) (95% CI)[e]	57/163 (35) (28-42)	1/54 (2) (0-5)
Median (95% CI) DOR, months	16.6 (12.1-19.6)	9.7 (5.5-16.1)
Median PFS2, months	23.5	16.7
HR (95% CI)	0.79 (0.60-1.04)	

Patient disposition

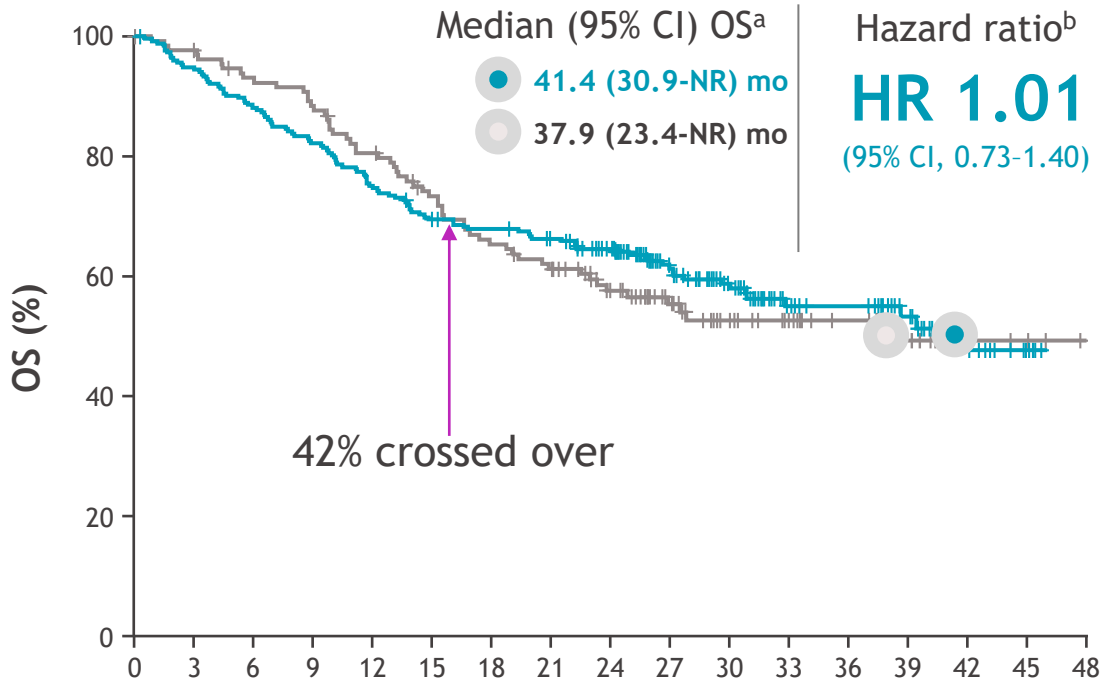
Patients, n (%)	Ide-cel (n = 254)	Standard regimens (n = 132)	Crossover from standard regimens to ide-cel[a] (n = 82)
ITT population[b]	**254 (100)**	**132 (100)**	-
Underwent leukapheresis	249 (98)	-	82 (62)
Received bridging therapy	212 (83)	-	68 (52)
Did not receive allocated study treatment	29 (11)	6 (5)	8 (6)
Treated population[c]	**225 (89)**	**126 (95)**	**74 (56)**
Ongoing in study	**136[d] (54)**	**10 (8)**	**52[e] (39)**
Ongoing for PFS	53 (21)	7 (5)	NA
Survival follow-up	83 (33)	3 (2)	50[f] (38)

[a]Following IRC-confirmed PD. Percentages used the standard regimens ITT population (n = 132) as the denominator; [b]All randomized patients; [c]Patients who received the study treatment to which they were randomly assigned (identical to the previously reported safety population), percentage calculated based on ITT population; [d]Included 3 patients ongoing in survival follow-up who received leukapheresis but did not receive ide-cel infusion; [e]Included 2 patients who received leukapheresis but not ide-cel infusion; [f]2 patients are also ongoing in the pretreatment period. ITT, intent-to-treat; NA, not applicable.

Rodríguez-Otero P, et al. ASH 2023 Abstract 1028

OS analysis confounded by substantial crossover

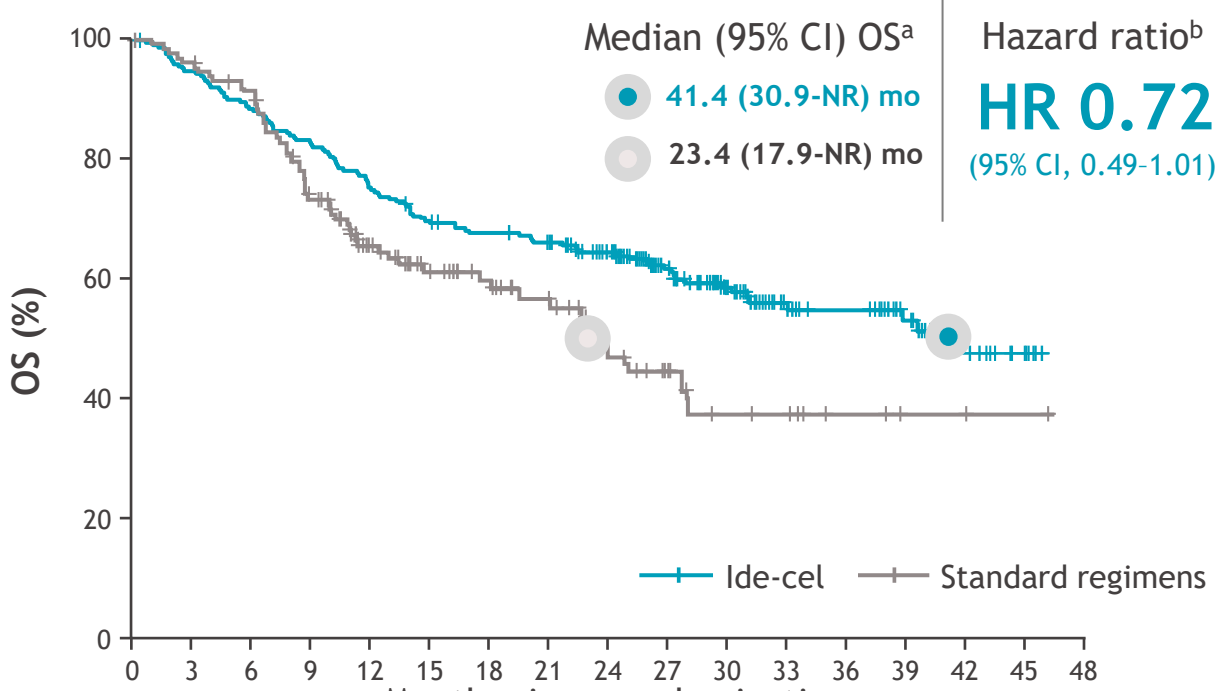
ITT population



Patients at risk

Ide-cel	254	240	223	208	190	175	169	161	143	103	75	48	44	30	13	4	0
Standard regimens	132	128	120	114	103	91	81	75	59	45	32	24	18	11	4	3	0

Sensitivity analysis adjusted for crossover[c]

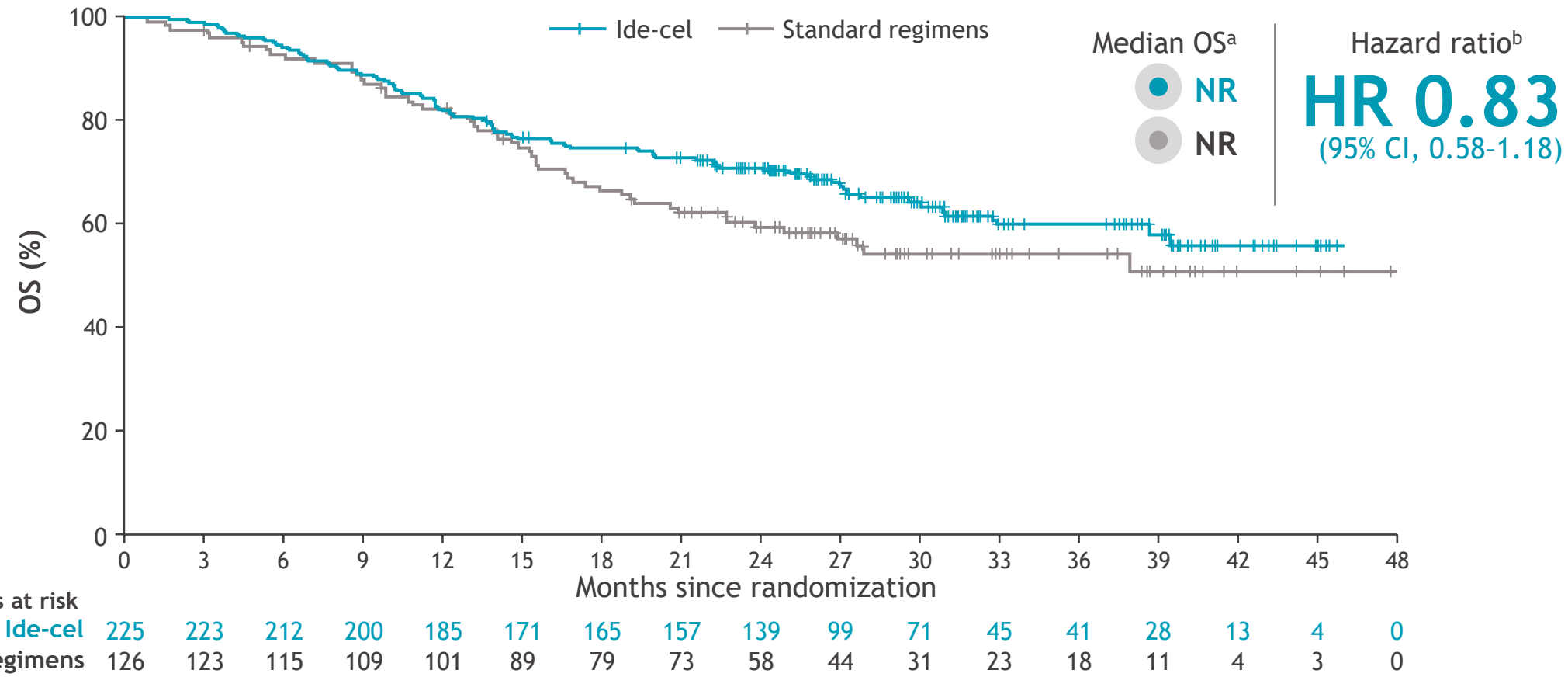


Ide-cel	254	240	223	208	190	175	169	161	143	103	75	48	44	30	13	4	0
Standard regimens	132	126	118	93	67	50	42	34	21	14	9	8	4	2	1	1	0

More than half of patients in standard regimens arm received ide-cel as subsequent therapy upon confirmed PD and the majority received ide-cel within 3-16 months of randomization

Prespecified crossover-adjusted analysis shows OS benefit of ide-cel

Trend of OS benefit with ide-cel among treated patients



- This is an exploratory analysis of the treated population without adjusting for crossover

Safety profile of ide-cel remained consistent

Treated population, n (%)	Ide-cel (n = 225)	Standard regimens (n = 126)
Any-grade AE	225 (100)	124 (98)
Serious AE	105 (47)	52 (41)
ITT population, n (%)	**Ide-cel (n = 254)**	**Standard regimens (n = 132)**
Overall deaths	106 (42)	58 (44)
Cause of death		
Disease progression	64 (25)	37 (28)
AEs	17 (7)	8 (6)
Other causes	23 (9)	12 (9)
SPMs[a]	2 (1)	1 (1)

Treated population, n (%)	Ide-cel (n = 225)
CRS[b]	
Any grade	197 (88)
Grade 3/4	9 (4)
iiNT[c]	
Any grade	34 (15)
Grade 3/4	7 (3)
Infections	
Any grade	125 (56)
Grade 3/4	50 (22)

- There were no new CRS or iiNT events with ide-cel since the interim analysis[1] and no parkinsonism or Guillain-Barré syndrome were reported
- **No SPMs of T-cell origin were reported in the ide-cel arm**
- No new safety signals

[a]Deaths due to SPMs in the ide-cel arm were leukemia (n = 1) and pancreatic adenocarcinoma (n = 1); death due to SPMs in the standard regimens arm was malignant neoplasm of unknown primary site (n = 1); [b]CRS was graded according to modified Lee's criteria;[2] maximum-grade events are reported, patients could have >1 event; [c]Includes immune effector cell-associated neurotoxicity syndrome reported by investigator as a neurologic toxicity.

AE, adverse event; CRS, cytokine release syndrome; ide-cel, idecabtagene vicleucel; iiNT, investigator-identified neurotoxicity; ITT, intent-to-treat; SPM, second primary malignancy.

1. Rodríguez-Otero P, et al. *N Engl J Med* 2023;388:1002–1014; 2. Lee DW, et al. *Blood*. 2014;124:188–195.

The NEW ENGLAND JOURNAL of MEDICINE

NEJM, January 24, 2024

Perspective

Secondary Cancers after Chimeric Antigen Receptor T-Cell Therapy

Nicole Verdun, M.D., and Peter Marks, M.D., Ph.D.

Since the first such product was approved in 2017, chimeric antigen receptor (CAR) T-cell therapies have become important treatments for relapsed or refractory hematologic cancers, and the six products involving autologous CAR T cells that have been approved in the United States now cover a range of indications spanning relapsed or refractory B-cell acute lymphoblastic leukemia, B-cell non-Hodgkin's lymphomas, and multiple myeloma (see table). In addition, numerous autologous and allogeneic CAR-T products are in development. Manufacturers of these next-generation products are seeking to improve on the efficacy and safety profile of existing therapies for hematologic cancers and to target solid tumors. CAR T cells are also under investigation for the treatment of nonmalignant conditions, such as autoimmune diseases.[1]

The demonstrated efficacy of the current generation of approved CAR-T products comes along with several well-described safety concerns that are noted in the products' labeling, including risks of cytokine release syndrome, immune effector cell–associated neurotoxicity syndrome, various forms of cytopenia, and hypogammaglobulinemia. Better understanding of some of these risks has led to improved outcomes, such as for patients who develop cytokine release syndrome.[2]

All currently approved CAR-T products employ T cells that are produced by using viral transduction to transfer the genetic construct. Given the relatively recent deployment of these therapies, the Food and Drug Administration (FDA) has issued draft guidance recommending that people who receive CAR T cells engineered with integrating vectors be monitored for extended periods for adverse events, including cancers.[3] Although CAR-T products have to date been associated with fewer cancers than products made with the previous generation of viruses used for gene therapy transduction, the potential for oncogenesis caused by genomic integration or other mechanisms still exists with the current generation of retroviral vectors. For instance, the lentiviral vector constructs, despite integrating in a semirandom fashion into the genome, have affinity for areas of the genome in which active gene expression is taking place, which may pose a risk for insertional oncogenesis.[4]

As of December 31, 2023, the FDA had become aware of 22 cases of T-cell cancers that occurred after treatment with CAR-T products. Such cancers have included T-cell lymphoma, T-cell large granular lymphocytosis, peripheral T-cell lymphoma, and cutaneous T-cell lymphoma. Among the 14 cases for which adequate data are currently available, the cancers have manifested within 2 years after administration of CAR T cells (range, 1 to 19 months), with roughly half occurring within the first year after administration. Cases have been reported in conjunction with five of the six available CAR-T products, but the small number of cases and variation in product use preclude conclusions about the strength of an association with any specific product. Some of these cases are still under investigation.

In three cases for which genetic sequencing has been performed to date, the CAR transgene has been detected in the malignant clone, which indicates that the CAR-T product was most likely involved in the development of the T-cell cancer. With more than 27,000 doses of the six approved products having been administered in the United States, the overall rate of T-cell cancers among people receiving CAR-T therapies appears to be quite low, even if all reported cases are assumed to be related to treatment. But relying on postmarketing reporting may lead to underestimates of such cases.

The FDA is attempting to gather as much information as possible on each of the reported cases, but in many instances, adequate samples of the lymphomas have not been retained for testing by means of polymerase chain reaction or genome sequencing. Determination of whether the T-cell cancer is associated with the CAR construct therefore most likely won't be possible for every case reported to date. The FDA plans to provide updates as substantive new information becomes available.

It is important for clinicians caring for people who have received CAR T cells to report the occurrence of any new cancer. At this time, we recommend that patients and clinical trial participants who receive treatment with these products be monitored for new cancers throughout their lives, since — owing to the relatively recent widespread introduction of CAR-T products into clinical care — we don't yet know how long after treatment people remain at risk for these adverse events. If a new cancer occurs after treatment with one of these products, clinicians should contact the manufacturer to report the event and obtain instructions on the collection of patient samples for testing for the presence of the CAR transgene. Clinicians are also encouraged to report such T-cell cancers to the FDA by contacting us at 1-800-FDA-1088 or visiting the website for our medical product safety reporting program (http://www.fda.gov/medwatch).

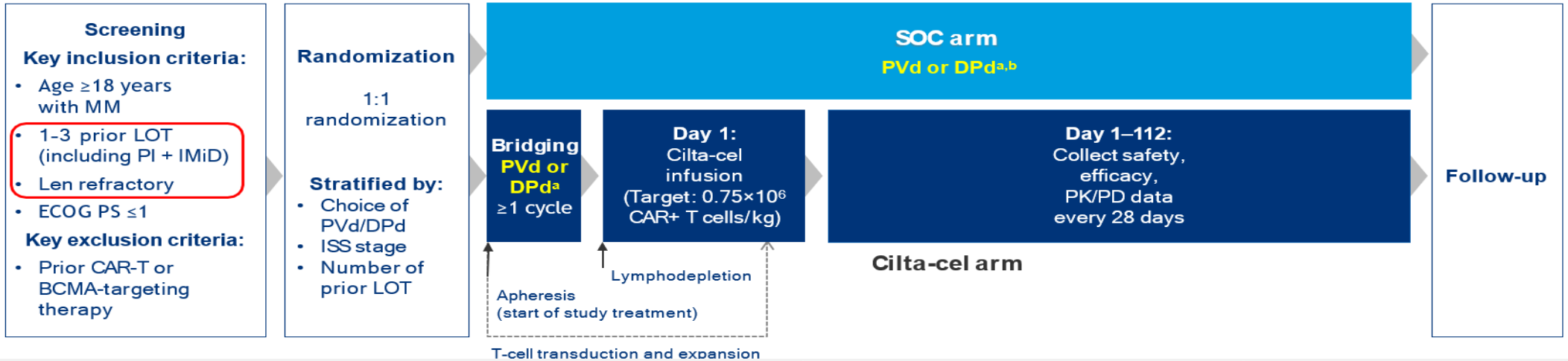
Moving forward, particularly as the use of CAR T cells for indications outside hematology and oncology is considered, new strategies involving targeting insertion of the CAR construct to specific loci might help reduce the risk of cancers due to integration of the CAR construct at oncogenic loci within the genome.[5] Comprehensive tumor-testing strategies might also generate information on the risk for and nature of these cancers and provide additional mechanistic insights. For now, second-

Chimeric Antigen Receptor T-Cell Products Approved in the United States.*

Brand Name	Generic Name	Manufacturer	Year Initially Approved	Indications (Relapsed or Refractory Disease)
Kymriah	Tisagenlecleucel	Novartis Pharmaceuticals	2017	Pediatric or young-adult B-cell ALL, large B-cell lymphoma, follicular lymphoma
Yescarta	Axicabtagene ciloleucel	Kite Pharma	2017	Large B-cell lymphoma
Tecartus	Brexucabtagene autoleucel	Kite Pharma	2020	B-cell ALL, mantle-cell lymphoma
Breyanzi	Lisocabtagene maraleucel	Juno Therapeutics/Bristol Myers Squibb	2021	Large B-cell lymphoma, primary mediastinal large B-cell lymphoma, follicular lymphoma
Abecma	Idecabtagene vicleucel	Celgene/Bristol Myers Squibb	2021	Multiple myeloma
Carvykti	Ciltacabtagene autoleucel	Janssen Biotech	2022	Multiple myeloma

* ALL denotes acute lymphoblastic leukemia.

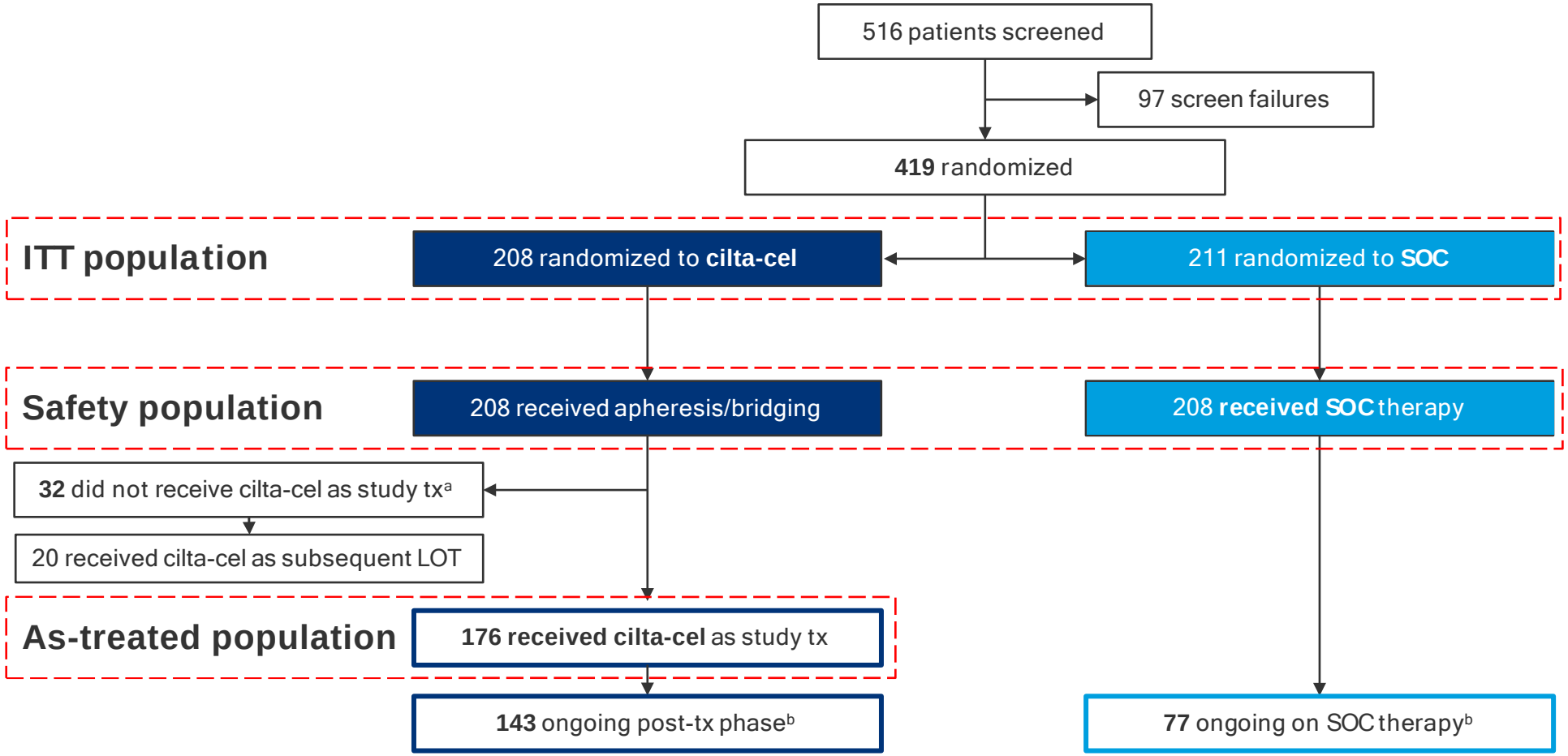
CARTITUDE-4: study design and baseline characteristics



Baseline characteristic	ITT population	
	Cilta-cel (n=208)	SOC (n=211)
Age, median (range), years	61.5 (27-78)	61.0 (35-80)
Male, n (%)	116 (55.8)	124 (58.8)
White, n (%)	157 (75.5)	157 (74.4)
ECOG PS ≤1, n (%) [a,b]	207 (99.5)	210 (99.5)
ISS stage, n (%)		
I	136 (65.4)	132 (62.6)
II	60 (28.8)	65 (30.8)
III	12 (5.8)	14 (6.6)
Bone marrow plasma cells ≥60%,[c] n (%)	42 (20.4)	43 (20.7)
Presence of soft tissue plasmacytomas,[d] n (%)	44 (21.2)	35 (16.6)
Years since diagnosis, median (range)	3 (0.3-18.1)	3.4 (0.4-22.1)
Prior LOT, median (range)	2 (1-3)	2 (1-3)
1 prior LOT, n (%)	68 (32.7)	68 (32.2)
2 or 3 prior LOT, n (%)	140 (67.3)	143 (67.8)

Baseline characteristic	ITT population	
	Cilta-cel (n=208)	SOC (n=211)
Cytogenetic high risk, n (%)[e]	123 (59.4)	132 (62.9)
del(17p)	49 (23.7)	43 (20.5)
t(14;16)	3 (1.4)	7 (3.3)
t(4;14)	30 (14.5)	30 (14.3)
gain/amp(1q)	89 (43.0)	107 (51.0)
2 or more high-risk cytogenetic features	43 (20.8)	49 (23.3)
del(17p), t(14;16), or t(4;14)	73 (35.3)	69 (32.9)
Triple-class[f] exposed, n (%)	53 (25.5)	55 (26.1)
Penta-drug[g] exposed, n (%)	14 (6.7)	10 (4.7)
Refractory status, n (%)		
Triple-class refractory[f,h]	30 (14.4)	33 (15.6)
Bortezomib	55 (26.4)	48 (22.7)
Pomalidomide	8 (3.8)	9 (4.3)
Daratumumab	48 (23.1)	45 (21.3)
Any PI	103 (49.5)	96 (45.5)

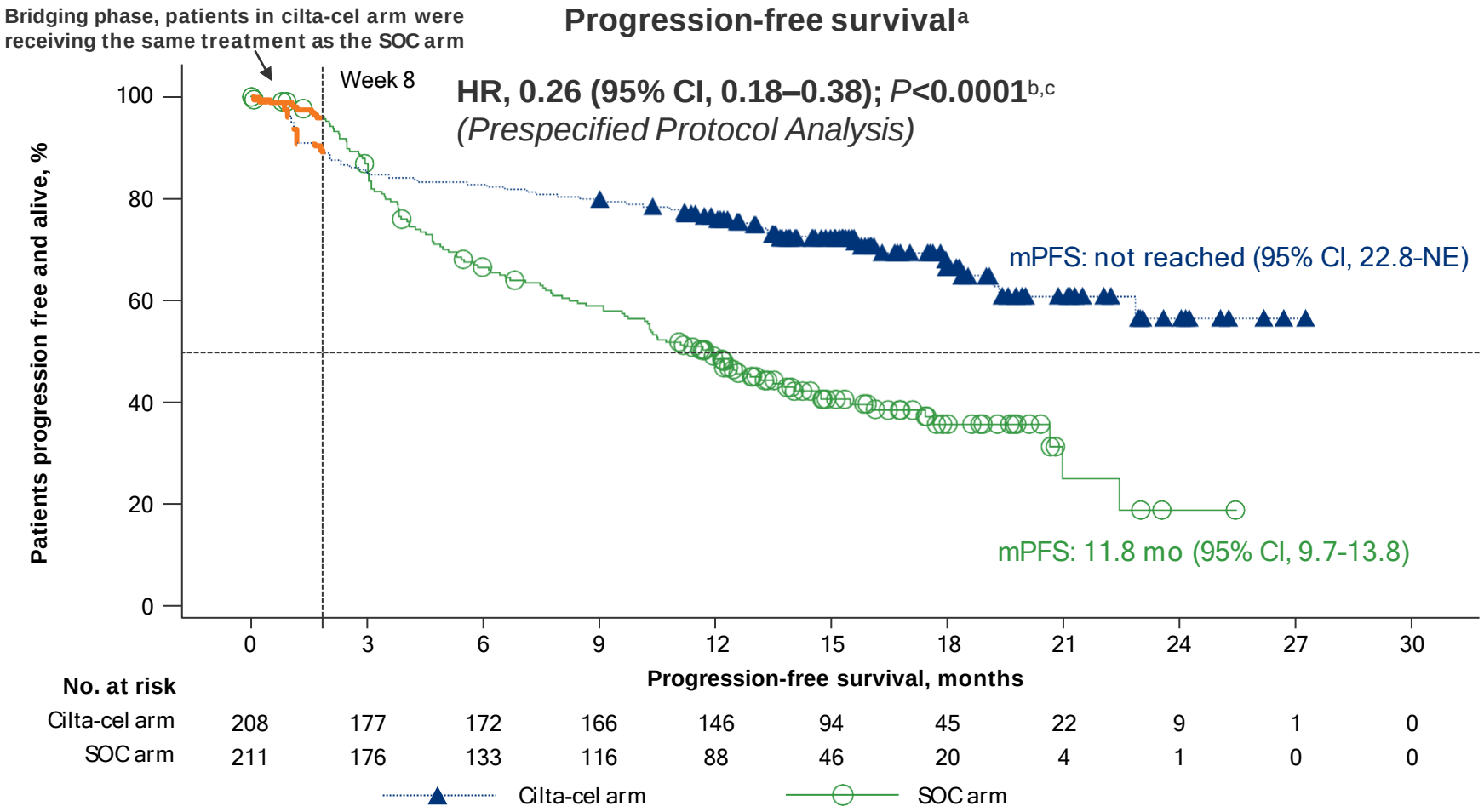
CARTITUDE-4: Patient Population and Follow-Up



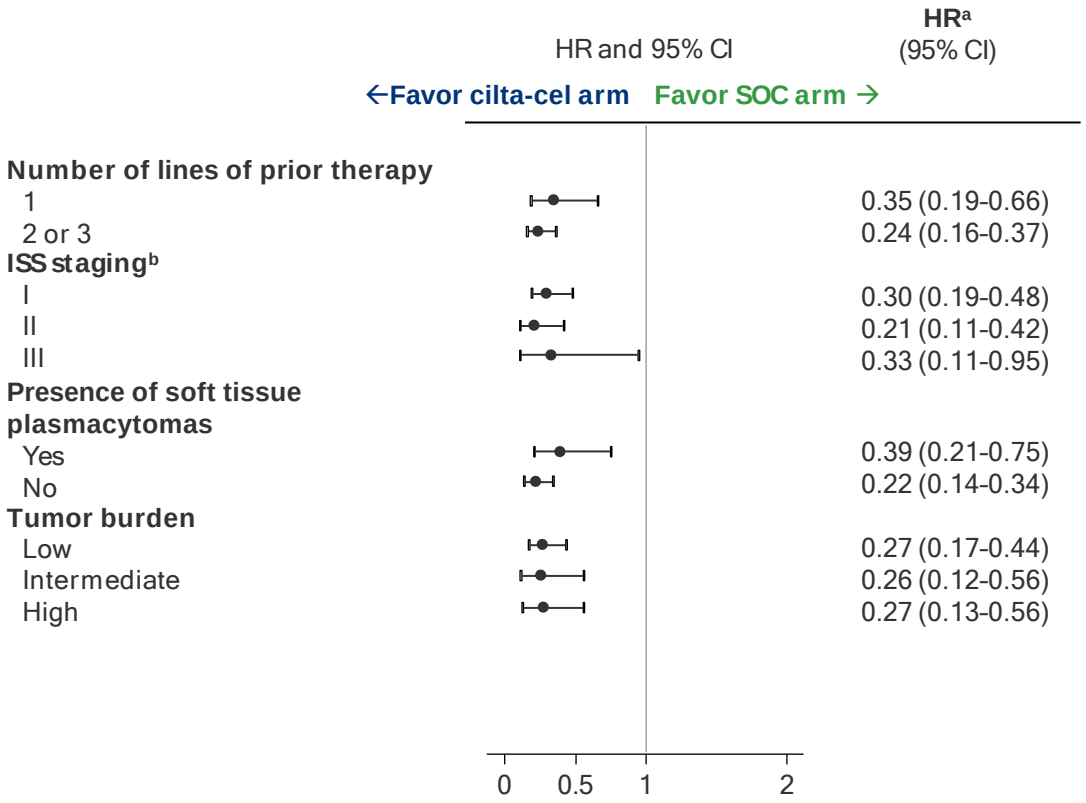
- At November 1, 2022, data cut-off, **median follow-up was 15.9 months** (range, 0.1-27)
- First patient randomized on July 10, 2020, and last patient randomized on November 17, 2021
- **Median time from first apheresis to cilta-cel infusion was 79 days**

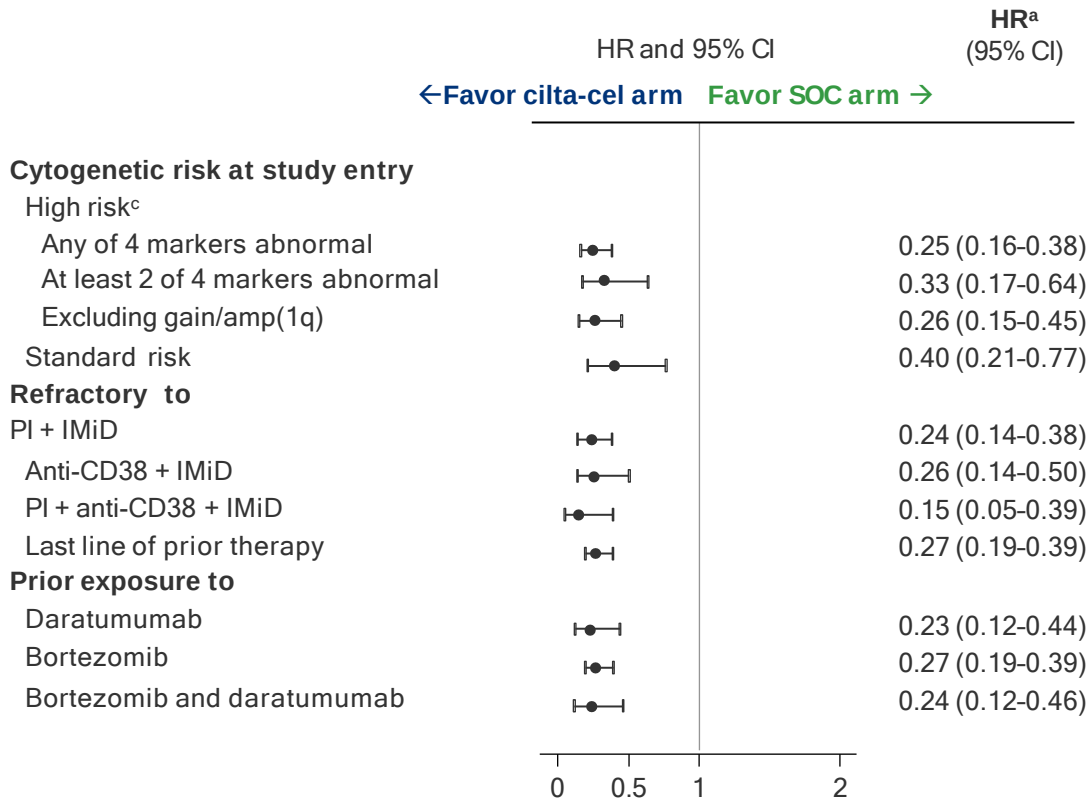
[a]Due to disease progression (n=30) or death (n=2) during bridging therapy/lymphodepletion. [b]Have not progressed.
cilta-cel, ciltacabtagene autoleucel; ITT, intent-to-treat; LOT, line of therapy; SOC, standard of care; tx, treatment.

CARTITUDE-4: PFS (ITT Population)

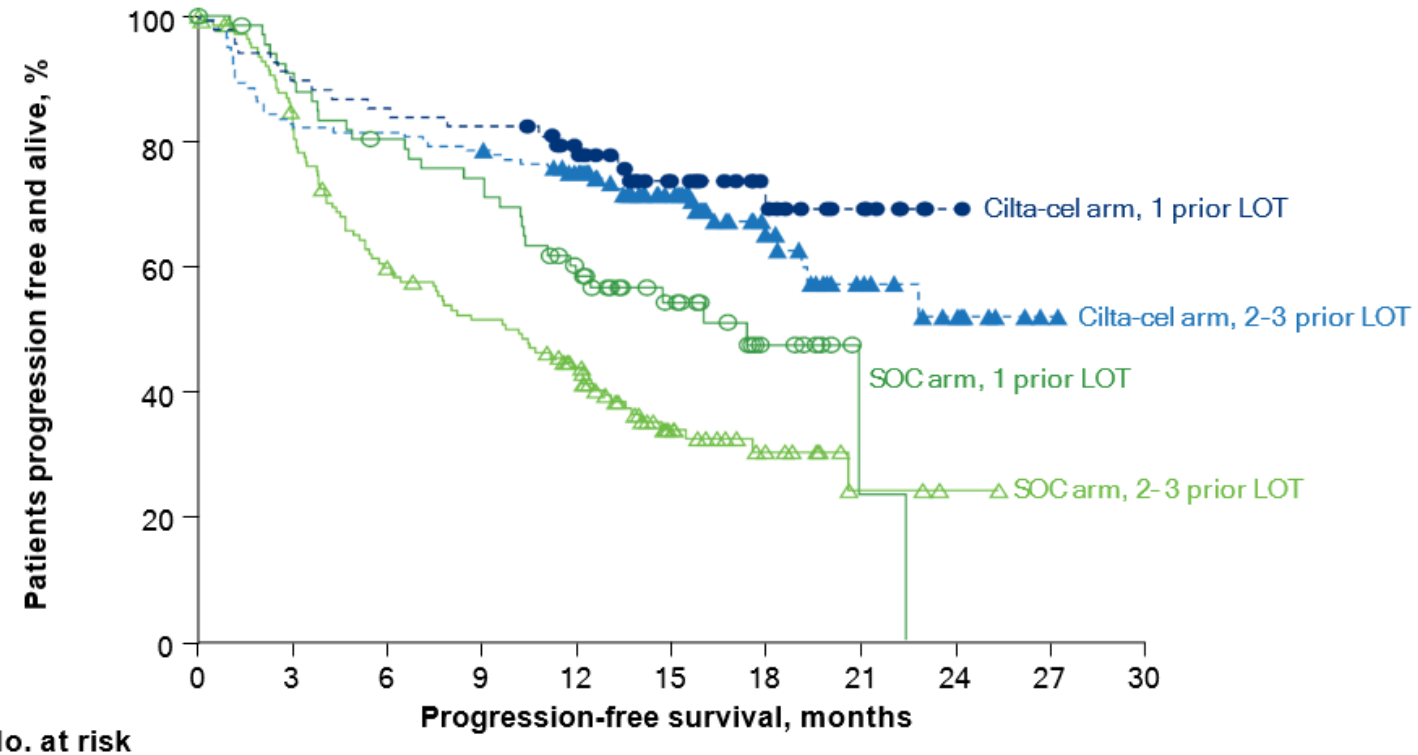


PFS: Key Subgroup Analysis (ITT)



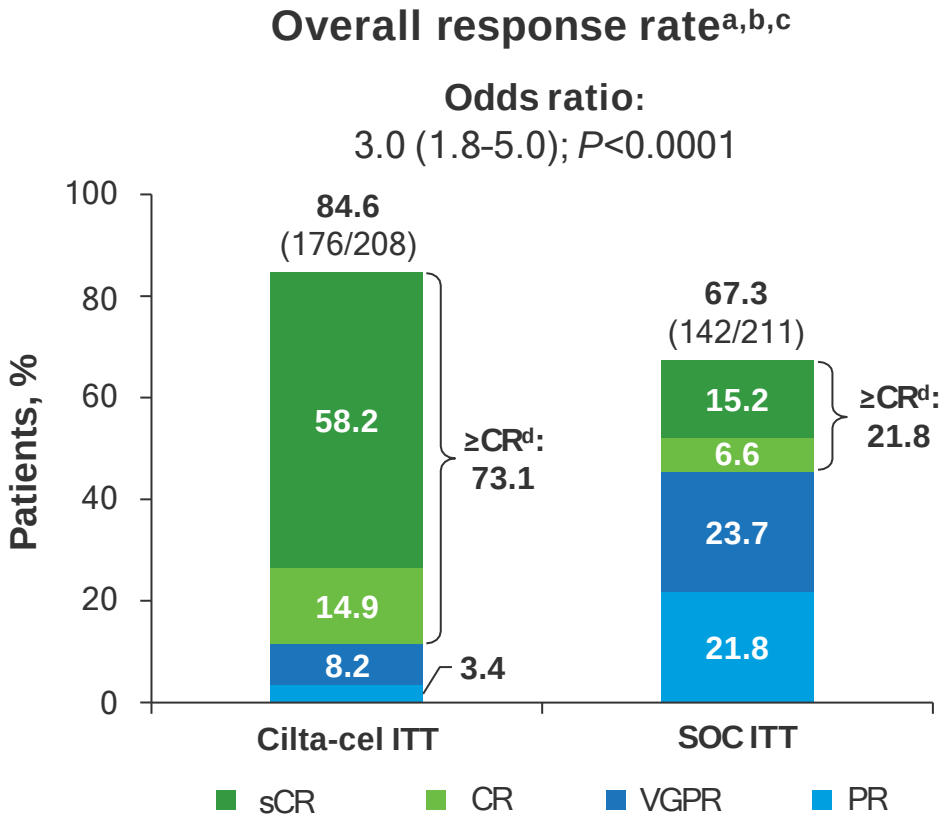


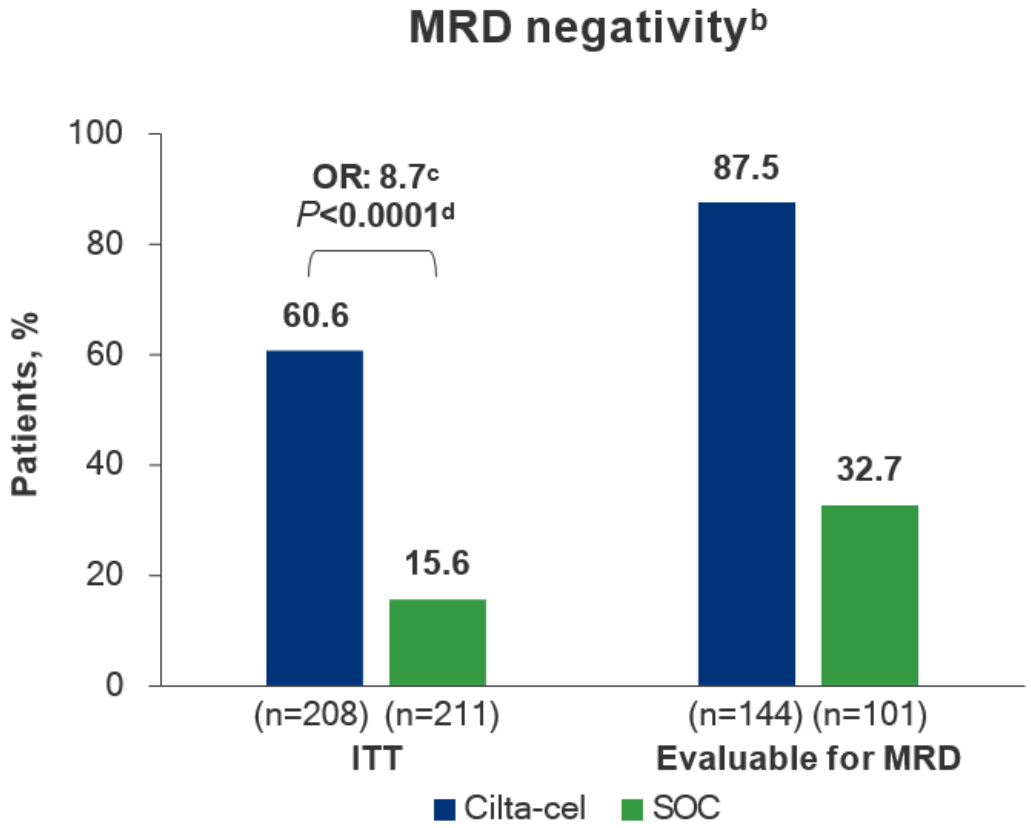
CARTITUDE-4: PFS by prior LoT



No. at risk											
Cilta-cel arm, 1 prior LOT	68	61	58	56	48	28	16	8	1	0	0
Cilta-cel arm, 2-3 prior LOT	140	116	114	110	98	66	29	14	8	1	0
SOC arm, 1 prior LOT	68	60	52	48	35	22	8	1	0	0	0
SOC arm, 2-3 prior LOT	143	116	81	68	53	24	12	3	1	0	0

ORR and MRD negativity rates





TEAEs, CRS and CAR-T-Related Neurotoxicity

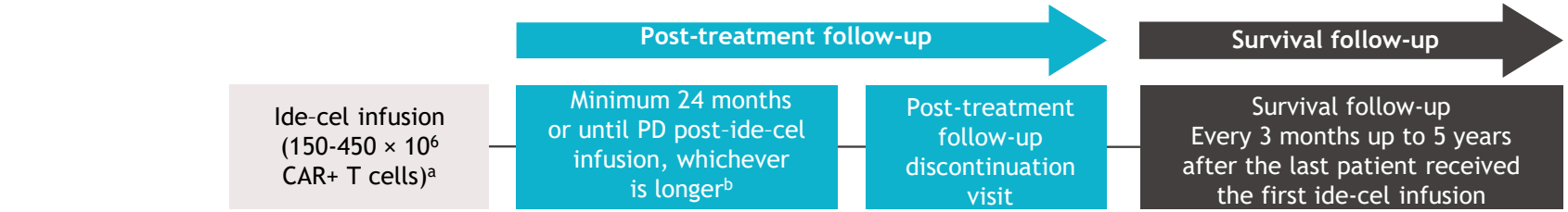
Select TEAE ≥15%, n (%)	Safety population			
	Cilta-cel (n=208)		SOC (n=208)	
	Any grade	Grade 3/4	Any grade	Grade 3/4
Any AE	208 (100)	201 (96.6)	208 (100)	196 (94.2)
Serious AE	92 (44.2)	67 (32.2)	81 (38.9)	70 (33.7)
Hematologic	197 (94.7)	196 (94.2)	185 (88.9)	179 (86.1)
Neutropenia	187 (89.9)	187 (89.9)	177 (85.1)	171 (82.2)
Anemia	113 (54.3)	74 (35.6)	54 (26.0)	30 (14.4)
Thrombocytopenia	113 (54.3)	86 (41.3)	65 (31.3)	39 (18.8)
Lymphopenia	46 (22.1)	43 (20.7)	29 (13.9)	25 (12.0)
Infections	129 (62.0)	56 (26.9)	148 (71.2)	51 (24.5)
Upper respiratory tract[a]	39 (18.8)	4 (1.9)	54 (26.0)	4 (1.9)
Lower respiratory tract[b]	19 (9.1)	9 (4.3)	36 (17.3)	8 (3.8)
COVID-19[c]	29 (13.9)	6 (2.9)	55 (26.4)	12 (5.8)

AEs, n (%)	As-treated patients (n=176)				
	Any grade	Grade 3/4	Median time to onset, days	Median duration, days	Resolved, n
CRS	134 (76.1)	2 (1.1)	8	3	134
Neurotoxicity[a]	36 (20.5)	5 (2.8)			
ICANS	8 (4.5)	0[b]	10	2	8
Other[c]	30 (17.0)	4 (2.3)			
Cranial nerve palsy[d]	16 (9.1)	2 (1.1)	21	77	14
Peripheral neuropathy	5 (2.8)	1 (0.6)	63	201	3
MNT	1 (0.6)	0	85	-	0

In the cilta-cel as-treated population:

- 30 patients had non-ICANS neurotoxicities[c]
  - 16 cranial nerve palsies (14 recovered)
  - 5 peripheral neuropathies
  - 1 MNT (grade 1)
- **Lower incidence and severity of CRS, ICANS, MNTs, and some cytopenias[e] observed with CARTITUDE-4 vs CARTITUDE-1**
  - Cilta-cel may be better tolerated when used earlier in treatment
  - Effective bridging therapy enables better control of tumor burden prior to CAR-T infusion
  - MNTs were lower likely related to patient management strategies implemented to mitigate this risk

KarMMa-2 cohort 2: ide-cel for “functional” HR MM



Cohort 2 (N = 99)
Clinical high-risk MM (1 regimen)

Cohort 2a (n = 37)
Early relapse (PD < 18 months from front-line therapy including ASCT)

Cohort 2b (n = 31)
Early relapse (PD < 18 months from front-line therapy without ASCT)

Cohort 2c (n = 31)
Key inclusion criteria
- **Inadequate response (< VGPR; excluding PD) 70-110 days post-ASCT (single/tandem)**
- ≥ 18 years of age
- Measurable disease[c]
- Received ≥ 3 cycles of induction therapy[d]
- ECOG performance status score ≤ 1

Primary endpoint
Cohort 2c: **CRR (CR and sCR;** by investigator per IMWG criteria)

Secondary endpoints
Cohort 2c: ORR, VGPR rate, TTR, DOR, PFS, TTP, OS, safety, PK, immunogenicity (anti-CAR antibody response), HRQoL

Exploratory endpoints
Cohort 2c: MRD, biomarkers

- Efficacy and safety were analyzed in all patients who received ide-cel

[a]After lymphodepletion (cyclophosphamide 300 mg/m^2 + fludarabine 30 mg/m^2 × 3), patients received a single infusion of ide-cel at a range of 150–450 × 10^6 CAR+ T cells (up to an additional 20%; 20% over the protocol-specified dose constituted overdose); [b]**At investigator discretion, patients could receive maintenance treatment post-infusion;** [c]Measurable disease determined by M protein (serum protein electrophoresis ≥ 0.5 g/dL or urine protein electrophoresis ≥ 200 mg/24 hours) and/or light chain MM without measurable disease in serum or urine (serum immunoglobulin free light chain ≥ 10 mg/dL and abnormal serum immunoglobulin κ:λ free light chain ratio); [d]Must contain a PI, an IMiD® agent, and dexamethasone.

ASCT, autologous stem cell transplantation; CAR, chimeric antigen receptor; CR, complete response; CRR, complete response rate; DOR, duration of response; ECOG, Eastern Cooperative Oncology Group; HRQoL, health-related quality of life; ide-cel, idecabtagene vicleucel; IMiD, immunomodulatory drug; IMWG, International Myeloma Working Group; MM, multiple myeloma; MRD, minimal residual disease; ORR, overall response rate; OS, overall survival; PD, progressive disease; PFS, progression-free survival; PI, proteasome inhibitor; PK, pharmacokinetics; sCR, stringent complete response; TTP, time to progression; TTR, time to response; VGPR, very good partial response.

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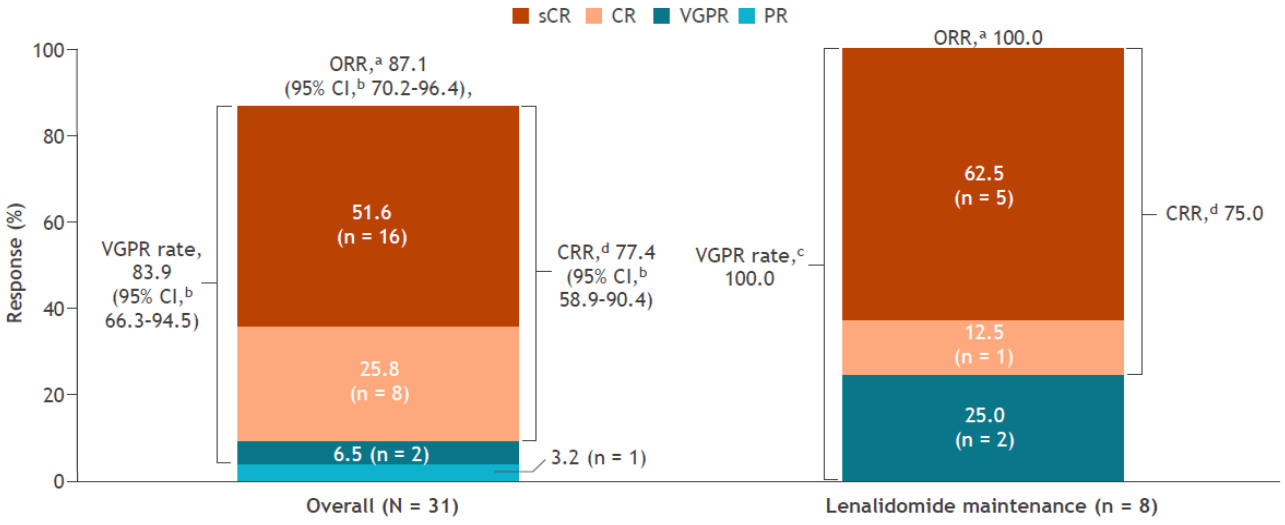
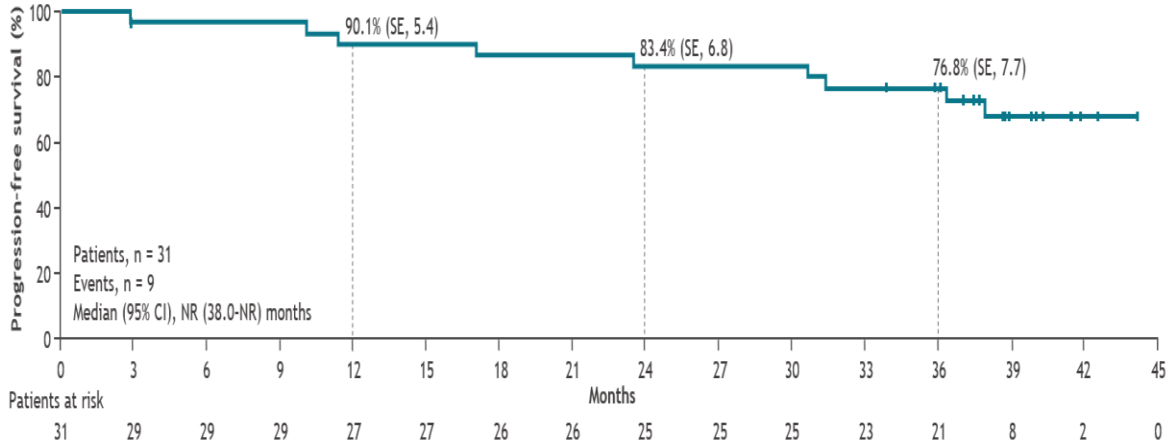
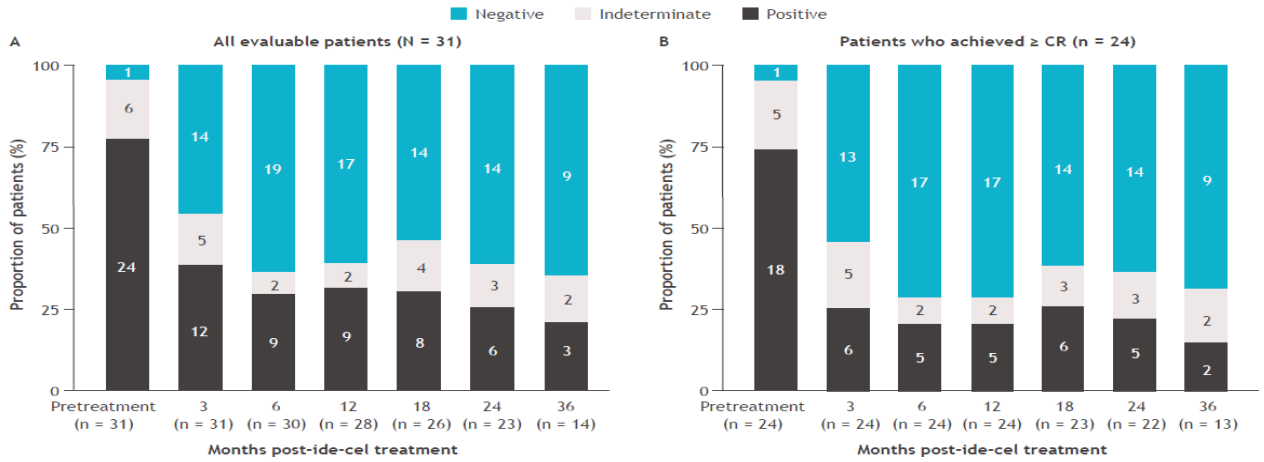
KarMMa-2 cohort 2c: clinical outcomes

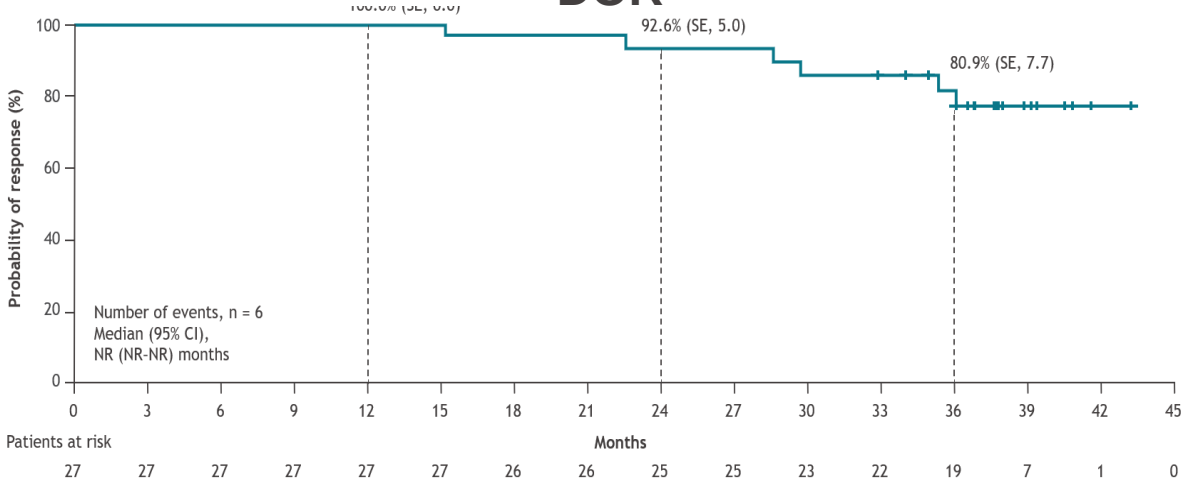


PFS





DOR



Summary

- **Ide-cel and cilta-cel significantly improved PFS vs SOC in patients with early lines of RRMM**
  - ✓ PFS benefit across many prespecified subgroups
- **Both ide-cel and cilta-cel significantly increased the ORR and depth of response vs SOC**
- **Relevance of the most effective bridging therapy**
- **The safety profile of ide-cel and cilta-cel was manageable and consistent with prior studies in later LoT**