Final Survival Analysis of Daratumumab Plus Lenalidomide and Dexamethasone Versus Lenalidomide and Dexamethasone in Transplant-ineligible Patients With Newly Diagnosed Multiple Myeloma (NDMM): MAIA Study

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HG has served in a consulting/advisory role for Adaptive Biotechnologies, Amgen, BMS, Celgene, Janssen-Cilag, Sanofi, and Takeda; received honoraria from Amgen, BMS, Celgene, Chugai Pharma, GSK, Janssen-Cilag, Novartis, and Sanofi; received research funding from Amgen, BMS, Celegen, Chugai Pharma Europe, Incyte, Janssen, Molecular Partners, MSD, Mundipharma, Novartis, and Takeda; received travel and accommodation funding Janssen-Cilag, Sanofi; other relationships with Amgen, Celgene/BMS, Chugai Pharma Europe, Janssen, and Sanofi**. TF** has nothing to disclose**. SKK** has served in a consulting/advisory role for Abbvie, Amgen, Antengene, Astra-Zeneca, Beigene, Bluebird Bio, BMS, Janssen, Kite, Oncopeptides, and Roche-Genentech; received honoraria from Antengene; and received research funding from Abbvie, Amgen, Astra-Zeneca, BMS, Carsgen, Janssen, Kite, Merck, Novartis, Roche-Genentech, Takeda, and Tenebio. **RZO** has served in a consulting/advisory role for and received honoraria from Amgen, BMS, Celgene, Kite Pharma, Sanofi, and Takeda; and received research funding from BioTheryX and Spectrum Pharma. **NB** has served in a consulting/advisory role for Amgen, Celgene, Janssen, Karyopharm Therapeutics, Pfizer, Sanofi, and Takeda; received honoraria from AbbVie, Amgen, Celgene, Genentech/Roche, GSK, Janssen, Karyopharm Therapeutics, Sanofi, and Takeda; and received research funding from Celgene, Janssen, and Pfizer. **PM** has served in a consulting/advisory role for and received honoraria from AbbVie, Amgen, Celgene, GlaxoSmithKline, Janssen, Oncopeptides, and Sanofi**. SB** has nothing to disclose. **CH** has served in a consulting/advisory role for Celgene; and received honoraria from Abbvie, Amgen, Celgene, and Janssen**. SZ** has served in a consulting/advisory role for and received research funding from Celgene, Janssen, and Takeda**. KW** has served in a consulting/advisory role for Adaptive Biotechnologies, Amgen, BMS, Celgene, GSK, Janssen-Cilag, Karyopharm Therapeutics, Oncopeptides, Roche, and Sanofi, Takeda; received honoraria from AbbVie, Adaptive Biotechnologies, Amgen, BMS, Celgene, GSK, Janssen-Cilag, Karyopharm Therapeutics, Novartis, Oncopeptides, Pfizer, Roche/Genentech, Sanofi, and Takeda; received research funding from Amgen, BMS, Celgene, GSK, Janssen-Cilag, and Sanofi; and received travel and accommodation funding from Amgen, BMS, Celgene, GSK, Janssen-Cilag, and Takeda**. AP has** served in a consulting/advisory role for and received honoraria from AbbVie, Amgen, BMS, Janssen, Pfizer, Sanofi, and Takeda**. CJ** has nothing to disclose. **NR** has nothing to disclose. **SM** has served in a consulting/advisory role for Adaptive Biotechnologies, Amgen, BMS, Celgene, Janssen, Pfizer, Regeneron, Roche/Genentech, and Sanofi**. AC** has served in a consulting/advisory role for Amgen, Genzyme, Janssen, Karyopharm Therapeutics, Oncopeptides, and Seattle Genetics**. MH** has nothing to disclose**. MMo** has received honoraria and research funding from Jazz Pharmaceuticals. **MD** has served as a consultant or advisor for Amgen, Bristol-Myers Squibb, GlaxoSmithKline, Janssen, Sanofi, Stemline Therapeutics, and Takeda. **TP** has served in a consulting/advisory role for Abbvie, Celgene, CSL-Behring, Genentech, Janssen, and Oncopeptides; and received research funding from Abbvie, Celgene, Genentech, Genmab, Janssen, Oncopeptides, and Takeda**. GC** has served in a consulting/advisory role for and received research funding from Amgen, BMS, Janssen, Karyopharm, Pfizer, Roche, Sanofi, and Takeda**. XL** has nothing to disclose**. HQ** has served in a consulting/advisory role for Celgene, CSL Behring, GSK, Janssen-Cilag, and Karyopharm Therapeutics. **CPV** has received honoraria from Amgen, Janssen, and Takeda; and received research funding from Amgen, Celgene**. MT** has nothing to disclose. **MMa** has received honoraria from BMS, Celgene, GSK, Janssen, Sanofi, and Takeda; received research funding from Janssen and Takeda; and received travel and accommodation funding from Janssen and Takeda**. LF** has nothing to disclose. **GW, HP, KB, RC** and **FB** are employees/hold stock and other ownership interests in Janssen. **SZU** has served in a consulting/advisory role for AbbVie, Amgen, BMS, Celgene, Genentech, Gilead Sciences, GSK, Janssen, Karyopharm Therapeutics, Merck, and Takeda; and received research funding from Amgen, Array BioPharma, BMS, Celgene, GSK, Merck, Pharmacyclics, Sanofi, Seattle Genetics, and SkylineDx.

MAIA: Introduction

- DARA is a human IgGκ monoclonal antibody targeting CD38 with a direct on-tumor[1-4] and immunomodulatory[5-7] MOA, demonstrating greater cytotoxicity toward MM cells ex vivo vs analogs of other CD38 antibodies[8]
- In the primary analysis of the phase 3 MAIA study, D-Rd significantly improved PFS compared with Rd alone in TIE patients with NDMM at median follow-up 28.0 months[9]
  - At median follow-up 64.5 months, D-Rd demonstrated
    - Significant OS benefit vs Rd (median, NR vs 65.5 months; HR, 0.66; 95% CI, 0.53–0.83; *P*=0.0003)
    - Continued PFS benefit vs Rd (median, 61.9 vs 34.4 months; HR, 0.55; 95% CI, 0.45–0.67; *P* <0.0001)[10]
  - Clinical benefit was even more pronounced among patients aged <70 years (OS: HR, 0.50; 95% CI, 0.27–0.90; *P*=0.0179 and PFS: HR, 0.35; 95% CI, 0.21–0.56; *P*<0.0001)[11]
  - Rate of sustained MRD negativity (10^{-5}) ≥18 months was 16.8% with D-Rd vs 3.3% with Rd (*P*<0.0001)[12]
- DARA is approved in combination with other standard-of-care regimens in NDMM[13]
  - DARA has been used in >518,000 patients worldwide[14] and consistently demonstrated efficacy in pivotal clinical trials[15-18]
- We present updated OS for D-Rd vs Rd and new data on subsequent antimyeloma therapies, with median follow-up ~7.5 years

CI, confidence interval; DARA, daratumumab; D-Rd, daratumumab, lenalidomide, and dexamethasone; HR, hazard ratio; Ig, immunoglobulin; MOA, mechanism of action; MM, multiple myeloma; MRD, minimal residual disease; NDMM, newly diagnosed multiple myeloma; NR, not reached; OS, overall survival; PFS, progression-free survival; Rd, lenalidomide and dexamethasone; TIE, transplant-ineligible.

1. de Weers M, et al. *J Immunol* 2011;186:1840-8. 2. Lammerts van Bueren J, et al. *Blood* 2014;124:3474. 3. Overdijk MB, et al. *MAbs* 2015;7:311-21. 4. Overdijk MB, et al. *J Immunol* 2016;197:807-13. 5. Krejcik J, et al. *Blood* 2016;128:384-94. 6. Adams HC III, et al. *Cytometry A* 2019;95:279-89. 7. Casneuf T, et al. *Leukemia* 2021;35:573-84. 8. Kinder M, et al. *Haematologica* 2021;106:2004-8. 9. Facon T, et al. *N Engl J Med* 2019;380:2104-15. 10. Weisel K, et al. *HemaSphere* 2023;7(suppl 2):14-15. 11. Facon T, et al. Presented at ASH; December 10–13, 2022; New Orleans, LA, USA. Poster 4553. 12. Kumar SK, et al. Presented at ASH; December 10–13, 2022; New Orleans, LA, USA. Poster 4559. 13. DARZALEX® (daratumumab) injection, for intravenous use [package insert]. Janssen Biotech, Inc.; 2023. 14. Data on file. 15. Mateos MV, et al. *Lancet* 2020;395:132-41. 16. Moreau P, et al. *Lancet Oncol* 2021;22:1378-90. 17. Voorhees PM, et al. *Lancet Haematol* 2023;10:e825-37. 18. Sonneveld P, et al. *N Engl J Med* 2024;390:301-13.

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MAIA: Study Design

- In MAIA (NCT02252172), patients with NDMM who were ineligible for high-dose chemotherapy and autologous stem cell transplant (due to age ≥65 years or the presence of comorbidities) were randomized 1:1 to receive D-Rd or Rd
- Patients received 28-day cycles of Rd (R: 25 mg orally once daily on days 1–21; d: 40 mg orally on days 1, 8, 15, and 22) with or without DARA (16 mg/kg intravenously weekly during cycles 1 and 2, every 2 weeks during cycles 3–6, and every 4 weeks thereafter) until disease progression or unacceptable toxicity

DARA, daratumumab; D-Rd, daratumumab, lenalidomide, and dexamethasone; NDMM, newly diagnosed multiple myeloma; Rd, lenalidomide and dexamethasone.

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MAIA: Assessments

- The primary endpoint was PFS; key secondary endpoints presented here include OS and time to subsequent antimyeloma therapy
- Time-to-event endpoints were compared between treatment groups using a stratified log-rank test
  - The Kaplan-Meier method was used to estimate distributions
  - For the whole ITT population, HRs and 95% CIs were estimated using a stratified Cox regression model with treatment as the sole variable and stratified with the following randomization stratification factors:
    - ISS disease stage (I vs II vs III), region (North America vs other), and age (<75 years vs ≥75 years)
  - For subgroups of patients in the ITT population, HRs and 95% CIs were estimated using a nonstratified Cox regression model with treatment as the sole variable
- Data on classes of subsequent therapies, subsequent regimens, rate of study treatment discontinuation, and causes of death were reported descriptively

CI, confidence interval; HR, hazard ratio; ISS, International Staging System; ITT, intent-to-treat; OS, overall survival; PFS, progression-free survival.

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MAIA: Demographic and Baseline Characteristics of the ITT Population[a]

- In total, 737 patients were randomized in MAIA (D-Rd, n=368; Rd, n=369)
- Baseline patient characteristics were balanced between groups; median age was 73 years (range 45–90), with 43.6% of patients aged ≥75 years

Characteristic	D-Rd (n=368)	Rd (n=369)
Median age, years (range)	73 (50-90)	74 (45-89)
Age ≥75, n (%)	160 (43.5)	161 (43.6)
Male, n (%)	189 (51.4)	195 (52.8)
ECOG PS, n (%)		
0	127 (34.5)	123 (33.3)
1	178 (48.4)	187 (50.7)
≥2	63 (17.1)	59 (16.0)
ISS disease stage, n (%)		
I	98 (26.6)	103 (27.9)
II	163 (44.3)	156 (42.3)
III	107 (29.1)	110 (29.8)
Type of measurable disease, n (%)		
IgG	225 (61.1)	231 (62.6)
IgA	65 (17.7)	66 (17.9)
Other[b]	9 (2.4)	10 (2.7)
Detected in urine only	40 (10.9)	34 (9.2)
Detected as serum FLC only	29 (7.9)	28 (7.6)
Cytogenetic risk,[c] n (%)		
n	319	323
Standard risk	271 (85.0)	279 (86.4)
High risk	48 (15.0)	44 (13.6)

[a]The ITT population included all randomized patients. [b]Inclusive of IgD, IgE, IgM, and biclonal disease. [c]Cytogenetic risk was based on fluorescence in situ hybridization or karyotype analysis; patients who had a high-risk cytogenetic profile had ≥1 of the following high-risk abnormalities: del(17p), t(14;16), or t(4;14).
D-Rd, daratumumab, lenalidomide, and dexamethasone; ECOG PS, Eastern Cooperative Oncology Group performance status; FLC, free light chain; Ig, immunoglobulin; ISS, International Staging System; ITT, intent-to-treat; Rd, lenalidomide and dexamethasone.

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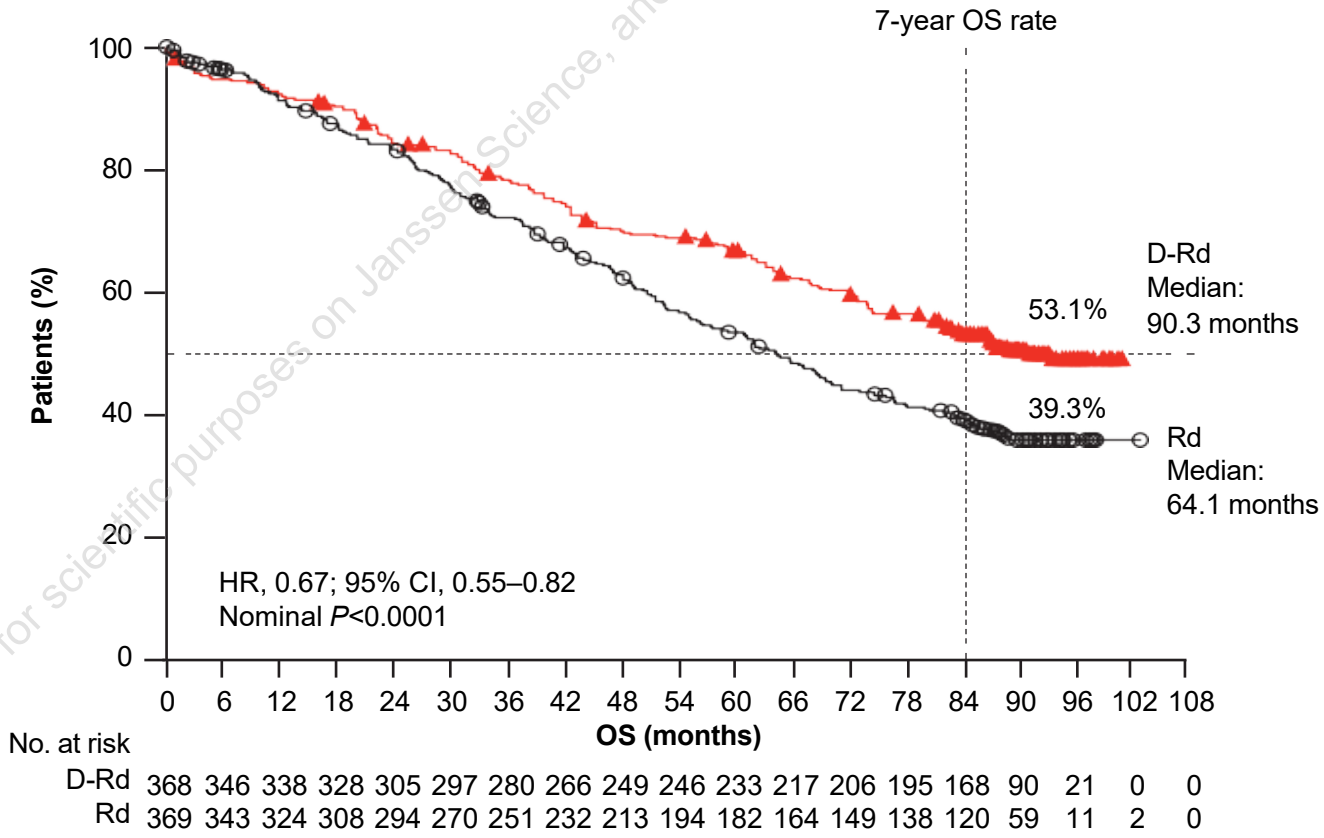
MAIA: OS

- With a median (range) follow-up of 89.3 (0–102.2) months, a 33% reduction in the risk of death was observed with D-Rd versus Rd
- Median OS was reached for the D-Rd group and was prolonged for patients in the D-Rd group versus those in the Rd group (90.3 vs 64.1 months, respectively)

OS with D-Rd and Rd in the ITT population[a]



[a]The ITT population included all randomized patients.
CI, confidence interval; D-Rd, daratumumab, lenalidomide, and dexamethasone; HR, hazard ratio; ITT, intent-to-treat; OS, overall survival; Rd, lenalidomide and dexamethasone.

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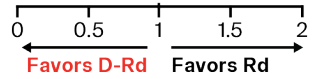
MAIA: OS in Prespecified Patient Subgroups

- Additionally, the OS benefit with D-Rd vs Rd was generally consistent across prespecified patient subgroups

OS in prespecified patient subgroups[a]

	D-Rd		Rd		HR (95% CI)[b]
	n/N	*Median OS (mo)*	*n/N*	*Median OS (mo)*	
Sex					
Male	95/189	82.5	120/195	60.6	0.72 (0.55-0.94)
Female	80/179	NE	98/174	67.8	0.66 (0.49-0.89)
Age					
<75 years	84/208	NE	107/208	79.6	0.69 (0.52-0.92)
≥75 years	91/160	72.3	111/161	54.8	0.67 (0.51-0.88)
Race					
White	161/336	92.7	197/339	65.5	0.71 (0.57-0.87)
Other	14/32	90.3	21/30	49.1	0.50 (0.25-0.99)
Region					
North America	46/101	92.7	64/102	54.8	0.57 (0.39-0.83)
Other	129/267	90.3	154/267	66.8	0.74 (0.58-0.93)
Baseline renal function (CrCl)					
>60 mL/min	99/206	92.7	123/227	69.9	0.78 (0.60-1.01)
≤60 mL/mln	76/162	90.3	95/142	54.4	0.57 (0.42-0.77)
Baseline hepatic function					
Normal	156/335	NE	203/340	63.8	0.65 (0.53-0.80)
Impaired	19/31	63.5	15/29	87.4	1.31 (0.66-2.58)
ISS disease stage					
I	34/98	NE	42/103	NE	0.79 (0.50-1.24)
II	77/163	92.7	95/156	61.7	0.63 (0.46-0.85)
III	64/107	65.2	81/110	47.3	0.68 (0.49-0.95)
Type of multiple myeloma					
IgG	111/225	87.2	132/231	69.3	0.78 (0.60-1.00)
Non-IgG	35/74	86.4	49/76	53.7	0.58 (0.37-0.89)
Cytogenetic risk at study entry[c]					
High risk	31/48	55.6	36/44	42.5	0.65 (0.40-1.06)
Standard risk	122/271	NE	160/279	65.5	0.66 (0.52-0.84)
ECOG PS					
0	48/127	NE	56/123	NE	0.76 (0.52-1.12)
1	86/178	92.7	118/187	58.3	0.64 (0.48-0.84)
≥2	41/63	62.8	44/59	39.0	0.68 (0.44-1.04)

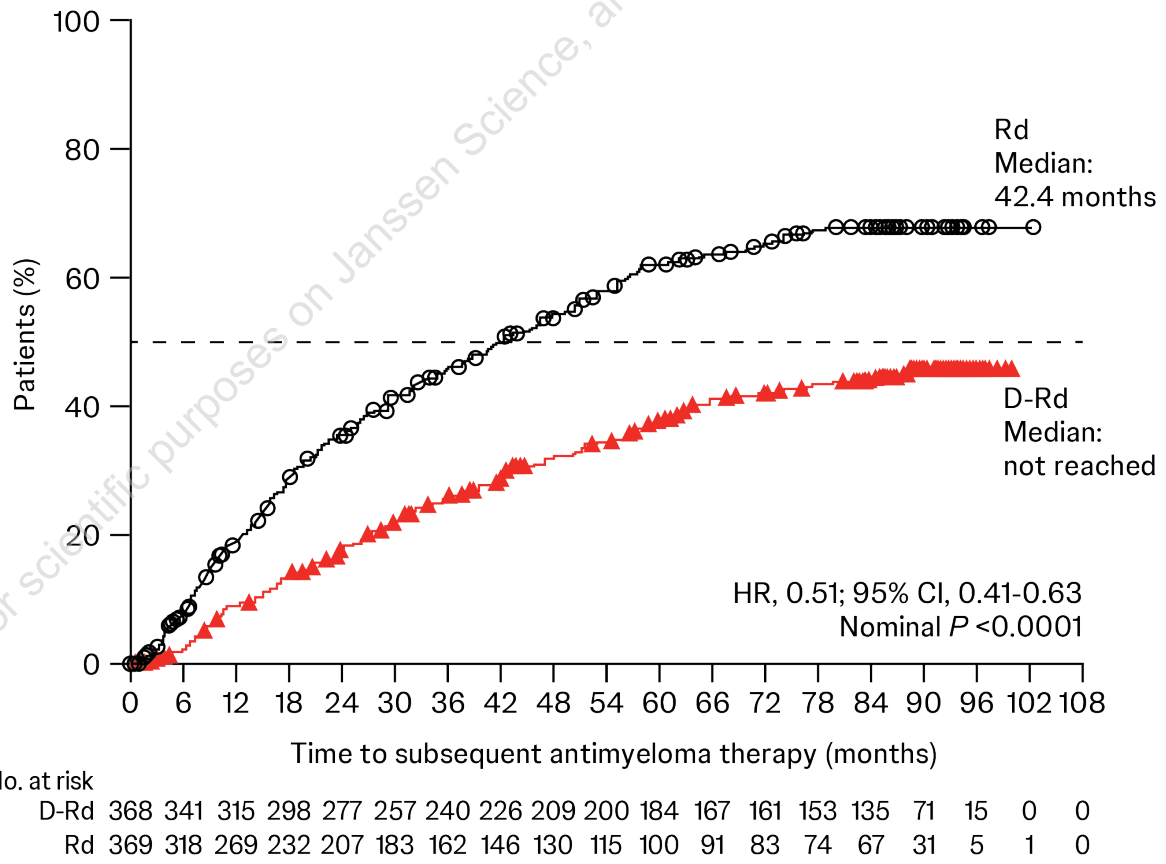


[a]In the ITT population, which included all randomized patients. [b]HRs and 95% CIs were from a Cox proportional hazards model with treatment as the sole explanatory variable. HRs <1 indicate an advantage for D-Rd. [c]Cytogenetic risk was based on fluorescence in situ hybridization or karyotype analysis; patients who had a high-risk cytogenetic profile had ≥1 of the following high-risk abnormalities: del(17p), t(14;16), or t(4;14).
CI, confidence interval; CrCl, creatinine clearance; D-Rd, daratumumab, lenalidomide, and dexamethasone; ECOG PS, Eastern Cooperative Oncology Group performance status; HR, hazard ratio; Ig, immunoglobulin; ISS, International Staging System; ITT, intent-to-treat; NE, not estimable; OS, overall survival; Rd, lenalidomide and dexamethasone.

MAIA: Time to Subsequent Antimyeloma Therapy

- Median time to subsequent antimyeloma therapy was not reached in the D-Rd group vs 42.4 months in the Rd group

Time to subsequent antimyeloma therapy in the ITT population[a]



[a]The ITT population included all randomized patients.
CI, confidence interval; D-Rd, daratumumab, lenalidomide, and dexamethasone; HR, hazard ratio; ITT, intent-to-treat; Rd, lenalidomide and dexamethasone.

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MAIA: Time to Subsequent Antimyeloma Therapy

- Among treated patients, 140/364 (38.5%) in the D-Rd group and 201/365 (55.1%) in the Rd group received ≥1 subsequent antimyeloma therapy
  - Across subsequent therapy lines, the most common antineoplastic agents after D-Rd and Rd, respectively, were bortezomib (27.7% vs 41.9%), DARA (6.3% vs 28.8%), and carfilzomib (7.7% vs 12.3%)
  - No patient in either group reported the use of BCMA- or GPRC5D-targeted therapy
  - 2 patients in each group received investigational drugs in subsequent therapy lines
- PI-based therapy was the most common first subsequent therapy class in both the D-Rd and Rd groups (69/140 [49.3%] and 101/201 [50.2%], respectively)
- DARA-containing regimens were received by 15/140 (10.7%) and 49/201 (24.4%) patients in the D-Rd and Rd groups, respectively, as their first subsequent therapy
- Among patients in the D-Rd and Rd groups who were evaluable for their best response to first subsequent antimyeloma therapy:
  - 6/130 (4.6%) and 8/193 (4.1%), respectively, achieved ≥CR
  - 18/130 (13.8%) and 46/193 (23.8%) achieved ≥VGPR

BCMA, B-cell maturation antigen; CR, complete response; DARA, daratumumab; D-Rd, daratumumab, lenalidomide, and dexamethasone; GPRC5D, G protein–coupled receptor class C group 5 member D; PI, proteasome inhibitor; Rd, lenalidomide and dexamethasone; VGPR, very good partial response.

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MAIA: First Subsequent Antimyeloma Therapy (Safety Population[a])

n (%)	D-Rd	Rd
Patients who received subsequent therapy, n	140	201
First subsequent therapy class[b,c]		
PI only	69 (49.3)	101 (50.2)
IMiD only	22 (15.7)	25 (12.4)
PI + IMiD	25 (17.9)	16 (8.0)
DARA monotherapy or combination	15 (10.7)	49 (24.4)
Other	9 (6.4)	10 (5.0)
Most common first subsequent therapy regimens[b,d]		
Bortezomib/cyclophosphamide/dexamethasone	19 (13.6)	29 (14.4)
Bortezomib/dexamethasone	20 (14.3)	28 (13.9)
Bortezomib/melphalan/prednisone	14 (10.0)	28 (13.9)
DARA/bortezomib/dexamethasone	4 (2.9)	27 (13.4)
Lenalidomide/dexamethasone	13 (9.3)	16 (8.0)
Bortezomib/pomalidomide/dexamethasone	9 (6.4)	3 (1.5)
Bortezomib/lenalidomide/dexamethasone	8 (5.7)	3 (1.5)
DARA/lenalidomide/dexamethasone	4 (2.9)	6 (3.0)
Pomalidomide/dexamethasone	2 (1.4)	6 (3.0)

[a]The safety population included all randomized patients who received ≥1 dose of study treatment. [b]Percentages were calculated with the number of patients who received subsequent therapy in each treatment group as the denominator. [c]Therapy classes are mutually exclusive. Patients in any therapy class subgroup may have received additional agents (other than PI, IMiD, or DARA), such as dexamethasone. [d]Regimens received by ≥3% of patients in either treatment group.
DARA, daratumumab; D-Rd, daratumumab, lenalidomide, and dexamethasone; IMiD, immunomodulatory drug; PI, proteasome inhibitor; Rd, lenalidomide and dexamethasone.

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MAIA: Safety and Tolerability

- Among the safety population, 285 (78.3%) and 345 (94.5%) patients in the D-Rd and Rd groups, respectively, discontinued study treatment
  - Mostly due to progressive disease (32.7% and 38.6%, respectively)
  - 16.5% and 25.8%, respectively, discontinued study treatment due to AEs
- Deaths were reported for 173 (47.5%) patients in the D-Rd group and 218 (59.7%) patients in the Rd group, most frequently due to disease progression

Summary of death and causes of death in the safety population[a]

n (%)	D-Rd (n=364)	Rd (n=365)
Total number of patients who died during the study	173 (47.5)	218 (59.7)
Primary cause of death		
Disease progression	76 (20.9)	88 (24.1)
AEs	44 (12.1)	40 (11.0)
Related to study treatment[b]	14 (3.8)	10 (2.7)
Unrelated to study treatment	28 (7.7)	29 (7.9)
Other[c]	53 (14.6)	90 (24.7)
Infections/infestations	9 (2.5)	30 (8.2)
General disorders/administration site conditions[d]	11 (3.0)	5 (1.4)
Neoplasms (benign, malignant, or unspecified)	11 (3.0)	4 (1.1)
Cardiac disorders	1 (0.3)	8 (2.2)
Nervous system disorders	3 (0.8)	5 (1.4)
Unknown	13 (3.6)	27 (7.4)
Deaths within 30 days of last study treatment dose	31 (8.5)	35 (9.6)
Primary cause of death		
Disease progression	1 (0.3)	1 (0.3)
AEs	29 (8.0)	32 (8.8)
Related to study treatment[b]	11 (3.0)	10 (2.7)
Unrelated to study treatment	18 (4.9)	22 (6.0)
Other[e]	1 (0.3)	2 (0.5)

[a]The safety population included all randomized patients who received ≥1 dose of study treatment. [b]Adverse events were related to ≥1 of the 3 components of study treatment: DARA, lenalidomide, and dexamethasone. [c]Other reasons were reported in ≥1% of patients in either treatment group. [d]All events were related to the general health condition of the patient. [e]Includes a nervous system disorder in 1 patient in the D-Rd group and a blood and lymphatic system disorder and general disorder/administration site condition in 1 patient each in the Rd group.
AE, adverse event; DARA, daratumumab; D-Rd, daratumumab, lenalidomide, and dexamethasone; Rd, lenalidomide and dexamethasone.

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MAIA: Conclusions

- In this final analysis of the MAIA study, median OS was finally reached in the D-Rd group after a median follow-up of approximately 7.5 years
- D-Rd continued to demonstrate a clinical OS benefit vs Rd alone in TIE patients with NDMM
- D-Rd also prolonged the median time to subsequent antimyeloma therapy
  - 28.8% of patients treated with Rd received DARA-based regimens as subsequent antimyeloma therapy, further emphasizing DARA as a standard of care for TIE patients with MM

With long-term follow-up in the MAIA study, the OS benefit observed with the addition of DARA to the Rd standard-of-care regimen continues to support the frontline use of D-Rd to maximize survival in TIE patients with NDMM

DARA, daratumumab; D-Rd, daratumumab, lenalidomide, and dexamethasone; MM, multiple myeloma; NDMM, newly diagnosed multiple myeloma; OS, overall survival; Rd, lenalidomide and dexamethasone; TIE, transplant-ineligible.

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