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A real-world comparative analysis of carfilzomib and other systemic multiple myeloma chemotherapies in a US community oncology setting

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Abstract

Background: Most multiple myeloma (MM) patients ultimately progress, with remission duration decreasing after first relapse. Recently, novel agents have been approved for the treatment of relapsed MM. There is a paucity of real-world data on these treatments. We sought to compare time to next treatment (TTNT) in MM patients in their second line of therapy (LOT2), treated with common proteasome inhibitor (PI)-based triplets.

Methods: Adult MM patients who received carfilzomib (K) between 1 November 2013 and 29 February 2016 at US Oncology Network (USON) clinics utilizing iKnowMed™ electronic health records (EHRs) were identified. Patients were included if they were ⩾18 years of age, not enrolled in clinical trials, had ⩾2 visits at a USON clinic and received LOT2 regimens consisting of: K+lenalidomide with steroid (KRd), bortezomib+lenalidomide with steroid (VRd), or bortezomib+cyclophosphamide with steroid (VCyd). TTNT was estimated from LOT2 initiation to LOT3 initiation using the Kaplan–Meier method, and hazard ratios (HRs) were estimated using Cox modeling.

Results: A total of 718 patients received a K-containing regimen sometime during their MM treatment (LOT1 to LOT5). Of these, 156 patients received: KRd ($n = 112$; 71.8%), VRd (n =27; 17.3%), or VCyd ($n = 17$; 10.9%). Baseline characteristics were similar between groups (mean age: 64.8 years; 58% male). Median TTNT was longest for KRd [25.3 months; 95% confidence interval (CI): 19.71–NR], *versus* VRd or VCyd (VRd median TTNT: 10.2 months, 95% CI: 4.24–12.71; VCyd: 6.5 months, 95% CI: 3.02–12.78; log-rank $p < 0.0001$). The adjusted HR for KRd was 0.19 (95% CI: 0.11–0.37), compared with VRd.

Conclusions: Considering the real-world nature of these data, the median TTNT observed with KRd was relatively consistent, with progression-free survival (PFS) for KRd observed in the phase III ASPIRE trial (median PFS: ITT population = 26.3 months; LOT2 = 29.6 months). Patients who received KRd at first relapse had significantly longer TTNT, compared with those on VRd or VCyd, confirming the value of KRd as an important treatment option for relapsed MM.

Keywords: carfilzomib, comparative effectiveness, multiple myeloma, relapse, second-line of therapy

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Introduction

Multiple myeloma (MM) is an incurable blood cancer of plasma cells that accumulate in bone marrow, leading to bone destruction, marrow failure, and end organ failure. In 2017, it was estimated that there will be 30,280 new cases and 12,790 deaths due to MM in the US.[1]

MM treatment has improved rapidly with the introduction of new classes of drugs in recent years, including immunomodulatory drugs (IMiDs), proteasome inhibitors (PIs) and monoclonal antibodies.[2] Among newly diagnosed MM patients, response rates have risen from ~30% with single agents to ~90% with triplet combination regimens.[3] However, while the disease responds to a variety of treatments, it is generally considered incurable and responses are often not sustained, with most patients ultimately experiencing relapsed or refractory disease (RRMM) and requiring subsequent therapy. Real-world claims analyses have shown that an estimated 37–61% of the patients with MM in the US progress following first-line therapy and move to a subsequent line of therapy (LOT), while other patients either die or are censored prior to receipt of subsequent treatment or are lost to follow up.[4–6] Further, about 22–43% patients require third and later LOTs.[4,6,7]

In relapsed/progressive MM, additional genetic mutations or alterations are acquired that render the disease more resistant, leading to progressively shorter durations of remission or response to each salvage therapy, and the ultimate development of refractory disease, which is known to be associated with poor clinical outcomes and overall survival (OS).[8,9] Many of the new drugs in MM have single-agent activity, however the duration of response, depth of response and proportion of patients achieving minimal residual disease negativity are limited when these drugs are used alone. In recent years, use of these drugs in triplet combinations has increased with triplet regimens demonstrating improved outcomes relative to single- or double-drug combinations.[2,10]

PIs, such as carfilzomib (K) and bortezomib, are frequently used in combination with IMiDs, such as lenalidomide, as second-line (2L) therapy, in combination with corticosteroids (dexamethasone and prednisone).[11] The most common triplet regimens for RRMM include K + lenalidomide with dexamethasone [KRd; National Comprehensive Cancer Network (NCCN)-preferred category 1 regimen], bortezomib + lenalidomide with dexamethasone (VRd; NCCN category 2A), and bortezomib + cyclophosphamide with dexamethasone (VCyd; also commonly referred to as CyBord; NCCN category 2A).[2]

PIs currently form the backbone of MM treatment, as they specifically target the 20S proteasome, which is central to proliferation of malignant plasma cells.[12] Myeloma cells are heavily dependent on the proteasome for clearing abnormal or cytotoxic proteins, and thus, are more susceptible to PIs than nonmalignant cells.[13–15]

K is an irreversible and selective inhibitor of the chymotrypsin-like (CT-L) activity of the 20S immunoproteasome. It is indicated for use as a single agent or in combination with dexamethasone (Kd dose: 56 mg/m^2) or with lenalidomide plus dexamethasone (KRd dose: 27 mg/m^2) for the treatment of patients with RRMM who have previously received one to three LOT.[16] Bortezomib (V) is a first-in-class PI that was initially approved in 2003 for use as a single agent in patients with RRMM who had received two prior therapies and were progressing on their most recent therapy. Recent data provide support for K as the more potent PI compared to V.[17,18] Specifically, data from the phase III ENDEAVOR trial comparing Kd *versus* Vd have shown the superior efficacy of Kd, which has been associated with longer OS and progression-free survival (PFS), as well as improved response rates and health-related quality of life.[17,19] Compared with V, K demonstrated improved OS [Kd group, median OS: 47.6 months, 95% confidence interval (CI): 42.5–NR; *versus* Vd group, median OS: 40.0 months, 95% CI: 32.6–42.3] and PFS (Kd group, median PFS: 18.7 months, 95% CI: 15.6–NR; *versus* Vd group, median PFS: 9.4 months, 95% CI: 8.4–10.4).[17,19]

Even with recent advances in treatment, achieving a sustained response to treatment with an acceptable level of toxicity remains a challenge, as most patients with MM will eventually experience relapse and relatively quickly exhaust available therapeutic options.[2,20] There are limited data comparing the effectiveness of these PI-based triplet regimens in the real-world setting. Thus, the objective of this study was to examine the time to next treatment (TTNT) as well as an exploratory analysis of OS for MM patients treated with the most common PI-based triplet regimens at first relapse (receiving LOT2 treatment) in a US community oncology setting.

Patients and methods

This retrospective cohort study evaluated patient characteristics, treatment patterns and clinical outcomes among adult (⩾18 years of age) RRMM patients who received a PI-based triplet regimen in combination with lenalidomide or cyclophosphamide plus dexamethasone. We identified our sample from a population of patients exposed to K-based regimens in any

LOT (1–5) between 1 November 1 2013 and 29 February 2016. Of these patients, a cohort of patients initiating LOT2 therapy with a PI-based triplet regimen were identified.

The population of patients was limited to those with exposure to K (pre- or post-LOT2 exposure to PIs), because the first indication for K was for the treatment of RRMM,[21] and a majority of K-exposed patients in the real world represented those who were intolerant or refractory to V and lenalidomide, and therefore constituted a distinct population. These patients were identified from the larger pool of MM patients treated in the US Oncology Network's (USON) nationwide clinics during the study period.

Patients were also required to have at least two office visits (in order to allow for calculation of follow-up time for time-to-event outcomes) during the study observation period at USON sites using the full iKnowMed (iKM)™ electronic health record (EHR) capacities at the time of treatment. Clinical trial participants were excluded.

Data source

iKM is an oncology-specific EHR system that captures outpatient practice encounter history for patients who receive care within the USON, including, but not limited to laboratory tests, diagnosis, therapy administration, LOT, cancer stage, comorbidities and performance status. iKM captures data on outpatient medical oncology care for patients treated across the US (19 states). Overall, the iKM EHR system captures data on approximately 10% of newly diagnosed cancer patients in the US. Because the study derived data mainly from the iKM database to meet the objectives, an intent-to-treat perspective was applied.

To supplement the data available in iKM on vital status and dates of death, the Social Security Death Index (SSDI) was used. The SSDI, which is maintained by the Social Security Administration, includes records of deaths reported by family members, funeral homes, hospitals, financial institutions, and federal agencies for individuals who have a social security number. The SSDI is updated monthly, but information on deaths occurring after March 2014 are limited due to regulatory restrictions on the release of new deaths for 3 years.[22] Persons never issued a social security number are not represented in the SSDI.

Exposure assessment

For an NCCN-recommended PI-based triplet regimen to be included in the analysis, at least 15 patients should have received that regimen in LOT2. Use of either steroid, dexamethasone or prednisone, was considered appropriate for the definition of the triplet-based regimens.

The baseline period consisted of a 60-day period prior to and 10-day period after initiation of LOT2 treatment. Patients had varying lengths of follow up depending on each patient's LOT2 initiation date and the last documented contact date, date of death, or the end of the study observation period; whichever occurred first. Study variables and outcomes were assessed regardless of minimum follow up using data available until the end of the study observation period, 29 February 2016.

To classify each treatment regimen into the appropriate LOT, duration of use and treatment administration dates, as well as provider-assigned LOT numbers (e.g. LOT2, LOT3, etc.), were considered. Treatment sequencing rules were created to define the order of the LOTs and to decide which regimens would be considered LOT1 line treatment, LOT2 line treatment, and so on. For these LOT assignments, documentation of disease progression was not considered. Instead, a regimen was assigned the next sequential LOT number (considered an advancement in LOT) if: a drug was added to an existing regimen (e.g. the addition of R to LOT1 Vd would be considered an advancement of a LOT, i.e. LOT1 Vd followed by LOT2 VRd); or a regimen that was administered for at least 30 days was followed by another nonoverlapping regimen with a minimum duration of 30 days (e.g. $\geqslant$30 days of Vd as LOT1 followed by $\geqslant$30 days of Kd, which would then be considered LOT2). If the next sequential regimen was the same as the previous combination therapy but a drug was dropped from the combination, it was considered the be the same LOT (no advancement in LOT number).

Ethical board approval

Institutional Review Board and Compliance/Privacy approval was gained prior to initiation of this study. Since this project involved the analysis of existing data and records, study information was analyzed in such a manner that research participants could not be directly identified. Thus, exemption status and a waiver of informed consent were approved. Data were handled in compliance with Health Insurance Portability

and Accountability Act and Health Information Technology for Economic and Clinical Health.

Statistical analysis

Descriptive analyses were conducted to assess demographic, clinical and treatment characteristics of the overall study cohort and stratified by LOT2 triplet-regimen use. Variables with missing values for greater than 35% of the study population were not considered in the analysis (cytogenetic data, transplant status and consolidative treatment are not presented for this reason). Continuous variables were described by mean, standard deviation, median and range. Categorical variables were described as frequency and percentage. To make statistical comparisons between the subgroups, Pearson χ^2 or Fisher's exact test were used to analyze categorical variables and Kruskal–Wallis tests were conducted for continuous variables.

Kaplan–Meier (KM) methods were used to examine time-to-event endpoints (both TTNT and an exploratory analysis of OS) with log-rank tests to compare LOT2 PI-based triplet-regimen groups. TTNT was estimated from the date of LOT2 initiation (first administration date of LOT2 regimen) through the date of initiation of the next LOT or death. Both advancement to the next LOT and death were considered failure events, and patients without failure events were censored. Note, the line assignment rules specified advancement in LOT would not occur for patients who discontinued one agent in combination regimen.

As an exploratory analysis, descriptive statistics on death (i.e. death rates and KM survival probabilities) were calculated. OS was estimated from the date of LOT2 initiation through date of death or censor. However, as survival data were not mature (i.e. majority of patients censored on the study end date), survival curves and detailed OS results are not shown.

Multivariable Cox regression modeling on TTNT was conducted. The variables considered for inclusion in the regression models were: baseline age, sex, race, geographic region of the practice, number of comorbidities, stage, body mass index, prior cancer history, performance status, and serum creatinine. Final models were constructed using a stepwise model building strategy (p value for inclusion = 0.25; p value for retention = 0.15). All analyses were conducted in SAS version 9.2 (The SAS Institute, Cary, NC).

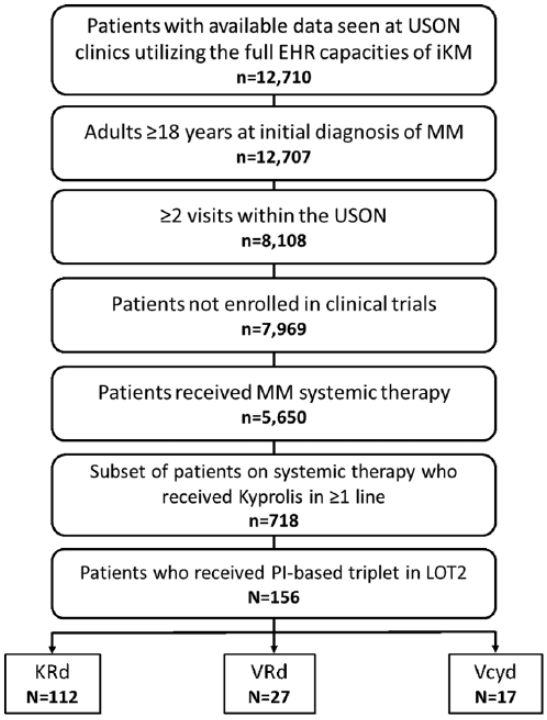

Figure 1. Eligibility and attrition table.
EHR, electronic health record; iKM, iKnowMed; LOT, lines of treatment; MM, multiple myeloma; PI, proteasome inhibitor; USON, US Oncology Network.

Results

Patient characteristics and demographics

A total of 12,707 adult patients with MM who had at least two office visits at a USON clinic were identified (Figure 1). Of these, 718 patients were treated with a K-containing regimen at some point during the study period and met other eligibility criteria. Among the K-treated population, 156 patients received a LOT2 PI-containing triplet regimen: KRd (n = 112; 71.8%), VRd (n = 27; 17.3%), and VCyd (n = 17; 10.9%). As the minimum number of patients required for inclusion was 15, not enough patients received KCyd in LOT2 for inclusion in our study.

The mean [± standard deviation (SD)] age of the study population was 64.8 (±11.3) years. In the KRd group, mean age was 64.7 (±11.6) years, whereas in the VRd and VCyd groups, mean age was 62.4 (±10.9) and 69.1 (±9.1), respectively (Table 1). Overall, 57.7% of the study population was male: 64.3% of the KRd group compared with 48.1% of the VRd group, and 29.4% of the VCyd group were male (p = 0.0138). No other significant demographic and clinical differences

Table 1. Baseline demographic and clinical characteristics of patients who received a PI-based LOT2 triplet regimen.

Characteristic	Overall	KRd	VRd	VCyd	*p* value
	***n* = 156**	***n* = 112**	***n* = 27**	***n* = 17**	
Age, years					0.2419
Mean (SD)	64.8 (11.3)	64.7 (11.6)	62.4 (10.9)	69.1 (9.1)	
Median (min, max)	67 (33.90+)	67 (33.90+)	64 (35.80)	67 (54.86)	
Age group, %					0.3222
<65 years	65 (41.7)	47 (42.0)	14 (51.9)	4 (23.5)	
65–75 years	59 (37.8)	40 (35.7)	10 (37.0)	9 (52.9)	
>75 years	32 (20.5)	25 (22.3)	3 (11.1)	4 (23.5)	
Sex, %					**0.0138**
Female	66 (42.3)	40 (35.7)	14 (51.9)	12 (70.6)	
Male	90 (57.7)	72 (64.3)	13 (48.1)	5 (29.4)	
Number of comorbidities, %					0.9223
1 comorbid condition	43 (27.6)	30 (26.8)	7 (25.9)	6 (35.3)	
⩾2 comorbid conditions	70 (44.9)	52 (46.4)	12 (44.4)	6 (35.3)	
None reported	43 (27.6)	30 (26.8)	8 (29.6)	5 (29.4)	
Stage at diagnosis, %					0.9890
I	36 (23.1)	26 (23.2)	7 (25.9)	3 (17.6)	
II	41 (26.3)	29 (25.9)	7 (25.9)	5 (29.4)	
III	51 (32.7)	37 (33.0)	9 (33.3)	5 (29.4)	
No information	28 (17.9)	20 (17.9)	4 (14.8)	4 (23.5)	
Baseline BMI category*, %					0.5976
Underweight	2 (1.3)	2 (1.8)	0 (0.0)	0 (0.0)	
Normal	42 (26.9)	29 (25.9)	7 (25.9)	6 (35.3)	
Overweight	58 (37.2)	40 (35.7)	11 (40.7)	7 (41.2)	
Obese	42 (26.9)	31 (27.7)	9 (33.3)	2 (11.8)	
No information	12 (7.7)	10 (8.9)	0	2 (11.8)	
History of prior cancer, %					0.1591
No	138 (88.5)	101 (90.2)	21 (77.8)	16 (94.1)	
Yes	18 (11.5)	11 (9.8)	6 (22.2)	1 (5.9)	

(Continued)

Table 1. (Continued)

Characteristic	Overall	KRd	VRd	VCyd	*p* value
	***n* = 156**	***n* = 112**	***n* = 27**	***n* = 17**	
ECOG at baseline, %					0.8443
0	15 (9.6)	13 (11.6)	2 (7.4)	0 (0.0)	
1	80 (51.3)	58 (51.8)	13 (48.1)	9 (52.9)	
2	18 (11.5)	13 (11.6)	3 (11.1)	2 (11.8)	
3	6 (3.8)	6 (5.4)	0 (0.0)	0 (0.0)	
No information	37 (23.7)	22 (19.6)	9 (33.3)	6 (35.3)	
Serum creatinine (mg/dl), %					0.7809
⩽2	120 (76.9)	86 (76.8)	21 (77.8)	13 (76.5)	
>2	16 (10.3)	11 (9.8)	4 (14.8)	1 (5.9)	
No information	20 (12.8)	15 (13.4)	2 (7.4)	3 (17.6)	

Bold numerals indicate statistical significance.
Baseline was defined as the period up to 60 days prior to or 10 days following initiation of a carfilzomib-containing regimen.
*Defined per standard definitions: underweight: BMI < 18.5 kg/m^2; normal: BMI ⩾ 18.5 kg/m^2 and <25.0 kg/m^2; overweight: BMI ⩾ 25.0 kg/m^2 and ⩽29.9 kg/m^2; obese: BMI ⩾ 30.0 kg/m^2.
BMI, body mass index; ECOG, Eastern Cooperative Oncology Group (disease-progression stage); KRd, carfilzomib+lenalidomide with dexamethasone or prednisone; LOT, lines of treatment; PI, proteasome inhibitor; SD, standard deviation; VCyd, bortezomib+cyclophosphamide with dexamethasone or prednisone; VRd, bortezomib+lenalidomide with dexamethasone or prednisone.

were noted between these PI-based regimens, including number of comorbidities, stage at diagnosis, Eastern Cooperative Oncology Group (ECOG) stage and serum creatinine levels (although information on these variables was missing for up to 23% of the patient population). Among the LOT2 patients, the three most common prior regimens (LOT1) were VRd (n = 41; 26.3%), Vd (n = 22; 14.1%), and VCyd (n = 21; 13.5%), but prior regimen data were missing for 53 patients (34.0%).

Clinical outcomes

Median TTNT was 25.3 months for the KRd group (95% CI: 19.71–NR), compared with 10.2 months in the VRd group (95% CI: 4.24–12.71) and 6.5 months in the VCyd group (95% CI: 3.02–12.78; log-rank $p < 0.0001$; Figure 2).

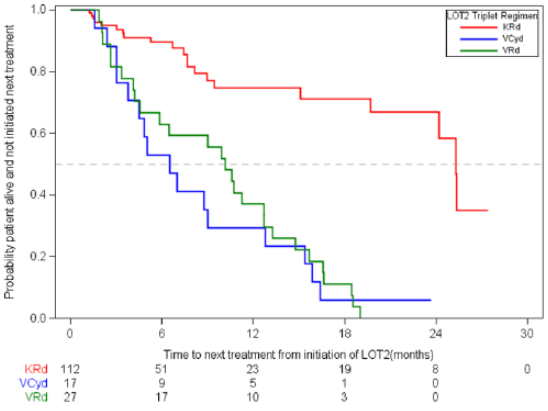

Figure 2. Kaplan–Meier curve of TTNT by LOT2 triplet regimens.
KRd, carfilzomib+lenalidomide with dexamethasone or prednisone; LOT, lines of treatment; VCyd, bortezomib+cyclophosphamide with dexamethasone or prednisone; VRd, bortezomib+lenalidomide with dexamethasone or prednisone.

In the exploratory survival analyses, we observed that median OS for the KRd group was not reached. Of the 112 KRd patients, 14.3% were deceased by the end of the study period (16 deaths), compared with 29.6% of the VRd patients (8 deaths among 27 patients) and 47.1% of the VCyd patients (8 deaths among 17 patients). Among the KRd group, 24-month survival was 71.8% (95% CI: 54.4–83.5), whereas 24-month survival was lower among the VRd

Table 2. Cox regression modeling: crude and final TTNT models.

Covariate	Level	Event frequencies			Hazard ratios			*p* value	
		Total	Censored	Event	Point estimate	95% lower limit	95% upper limit	Effect	Type 3
Crude TTNT model									
LOT2 regimen (with steroid)	VRd (reference)	27	0	27					**<0.0001**
	VCyd	17	1	16	1.116	0.599	2.078	0.7299	
	KRd	112	91	21	0.197	0.107	0.362	**<0.0001**	
Final Stepwise TTNT Model									
Stage at diagnosis	I (reference)	36	25	11					**0.0255**
	II	41	23	18	1.301	0.610	2.776	0.4960	
	III	51	31	20	1.421	0.676	2.988	0.3545	
	NA	28	13	15	3.138	1.404	7.012	**0.0053**	
LOT2 regimen (with steroid)	VRd (reference)	27	0	27					**<0.0001**
	VCyd	17	1	16	0.997	0.531	1.874	0.9938	
	KRd	112	91	21	0.182	0.097	0.342	**<0.0001**	

Bold numerals indicate statistical significance.
KRd, carfilzomib+lenalidomide with dexamethasone or prednisone; LOT, lines of treatment; PI, proteasome inhibitor; VCyd, bortezomib+cyclophosphamide with dexamethasone or prednisone; VRd, bortezomib+lenalidomide with dexamethasone or prednisone.

(64.7%; 95% CI: 39.2–81.7) and VCyd (35.7%; 95% CI: 10.3–62.8) groups. However, the majority of patients were censored because they survived beyond the study end date (among KRd patients, $n = 78$, 60.64% censored; among VRd, $n = 15$, 55.56 %; and among VCyd, $n = 5$, 35.29%).

Cox regression modeling: TTNT full and final model results. The unadjusted TTNT hazard ratio (HR) for KRd was 0.20 (95% CI: 0.11–0.36; Table 2). Despite adjusting for all the variables considered for inclusion in the TTNT model, there was no change in HR for KRd: 0.19 (95% CI: 0.11–0.37; compared with VRd as referent). Only disease stage and triplet regimen remained in the final stepwise TTNT model. The HR for KRd (final model HR: 0.18; 95% CI: 0.10–0.34) indicated an 80% lower risk of progression to next treatment or death among KRd patients, compared with those who received VRd (referent group) in LOT2. Patients receiving VCyd had a similar adjusted risk for progression to next treatment or death, as patients receiving VRd (HR: 1.00, 95% CI: 0.53–1.87).

Discussion

Published real-world data on the association between PI-based triplet regimens and clinical outcomes among RRMM patients are limited.[11,21] However, in this study, patients with RRMM who received KRd in LOT2 had significantly improved TTNT, compared with those who received VRd or VCyd.

Recently, Chari and colleagues utilized EHR data from 2008 to 2016 to compare outcomes in RRMM patients treated in second through fourth LOTs, between VRd, KRd, and ixazomib+Rd (IRd).[23] In contrast with this study, Chari and colleagues found that median TTNT for KRd was lower than that of VRd (8.7 *versus* 12.9 months, respectively). We believe that the

difference between TTNT in KRd *versus* VRd may have been confounded by LOT, as a large proportion of the KRd patients in their study were in more advanced lines of therapy (i.e. LOT3+), which would likely result in a shorter TTNT. Only 58% of KRd patients in their study were in LOT2, whereas substantially more of the VRd patients were LOT2 (76%) in the current study; all evaluated patients were receiving treatment. It is possible that patients receiving later LOTs have a more advanced disease status and a higher toxicity burden, which contributed to a shortened TTNT.

Although our available survival data were not yet mature (i.e. the majority of patients censored on the study end date), patients who received KRd had a lower mortality rate and higher 24-month survival than those who received VRd or VCyd. In fact, the 24-month survival in the KRd group in our study (71.8%; 95% CI: 54.4–83.5) is very consistent with that reported in the ASPIRE trial (73.3%; 95% CI: 68.6–77.5).[24] Because K is a more potent PI than V, the superior 24-month survival among those who received KRd compared with the V-based regimens is not unexpected, and is also consistent with the ENDEAVOR trial, in which K patients demonstrated better OS compared with V patients (Kd group, median OS: 47.6 months, 95% CI 42.5–NR; *versus* Vd group, median OS: 40.0 months, 95% CI: 32.6–42.3).[17] However, as our survival analysis results are exploratory, these mortality rates should be interpreted with caution.

This study has some limitations inherent to non-randomized EHR-based retrospective observational research studies.[25] Available data were limited to information in each patient's medical record. As such, there was the potential for documentation bias. Algorithm-based assignment of LOT sequences is another limitation of our real-world data, given that LOT numbers could not be validated. In addition, several key variables, including prior treatment regimen, cytogenetic risk, transplant status and consolidation therapy, had a high proportion of missing values in the structured EHR data, which prevented them from being included in the model and therefore, limited the conclusions that could be drawn. Likewise, prior treatment history was unavailable for patients who initiated treatment outside of the USON. These prior treatments, such as prior V among VRd and VCyd patients, may have influenced the results.

As in all observational studies, our results may be impacted by confounding (e.g. confounding by indication, incomplete control for confounding due to missing data/variables, and other residual confounding). Lastly, eligibility criteria for this study may have limited the number of K patients identified during the study identification period. In particular, patients who received care at USON facilities that did not utilize the full capacities of the EHR were excluded from the analysis to optimize the comprehensiveness of the dataset.

The iKM database is a rich resource of community-based oncology practice and patient data, and was likely one of the best resources available to study real-world treatment patterns. Major strengths of iKM include data on ~10% of newly diagnosed cancer patients in the US from 19 states. Although all regions of the country received some coverage in iKM, results may not be generalizable to all community-based oncology practices, as iKM only captures data from clinics that are part of the USON.

Conclusion

This real-world study provides insight into treatment patterns and outcomes for RRMM patients receiving systemic therapy in a US community oncology setting. Findings indicate that MM patients who received KRd, an NCCN category-1-preferred RRMM treatment, in LOT2 had significantly better TTNT, compared with those who received VRd or VCyd. Our findings confirm the value of KRd as an important therapeutic option for patients with MM at first relapse and contribute new evidence to the existing literature supporting the use of KRd over V-based triplets for the treatment of relapsed disease.

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Conflict of interest statement

The authors declare that there is no conflict of interest.

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