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Original Article

Comparative efficacy and safety of bispecific antibody teclistamab versus CAR-T cell therapies in relapsed/refractory multiple myeloma: a retrospective evaluation

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Abstract: To compare the clinical efficacy, safety, and cost-effectiveness of bispecific antibody teclistamab and chimeric antigen receptor T-cell (CAR-T) therapy in relapsed/refractory multiple myeloma (RRMM) to guide individualized treatment. This retrospective study enrolled 67 RRMM patients (excluded 6 of 73) hospitalized at Xinxiang Central Hospital (December 2024-May 2025), divided into teclistamab (n=32) and CAR-T (n=35) groups. Primary outcomes included overall response rate (ORR) and progression-free survival (PFS). Secondary outcomes comprised complete response rate (CRR), duration of response (DOR), minimal residual disease (MRD) negativity rate, overall survival (OS), adverse events (AEs), hospital stays, direct medical costs, and cost-effectiveness ratio (CER). The CAR-T group showed higher CRR (P=0.011), ORR (P=0.029), MRD negativity rate (P=0.027), longer median DOR [HR: 3.35 (1.838, 6.10), P<0.001], PFS [HR: 4.407 (1.994, 9.74), P<0.001], and better OS (HR: 3.204 (1.015, 10.1), P=0.021) than the teclistamab group. However, the CAR-T group had higher incidences of cytokine release syndrome (P=0.033) and hematological AEs (P=0.040), longer hospital stays, higher direct costs, and higher CER (all P<0.001). Prior treatment lines were independent prognostic factors (P=0.036). CAR-T therapy outperforms teclistamab in efficacy and survival outcomes but has higher AEs and costs. Teclistamab demonstrates superior safety and shorter hospital stays, supporting individualized clinical selection.

Keywords: Relapsed/refractory multiple myeloma, bispecific antibody, teclistamab, chimeric antigen receptor T cell, efficacy

Introduction

Multiple myeloma (MM), a malignant clonal disease originating from plasma cells, is characterized by both abnormal proliferation of plasma cells in bone marrow and extensive secretion of monoclonal immunoglobulins. This leads to a range of clinical symptoms, specifically bone destruction, anemia, and other complications [1-3]. The incidence of MM is increasing globally, making it one of the most common hematological malignancies. Its incidence is approximately 2 to 4 cases for every 100,000 per year. In China, with the acceleration of population aging, its incidence continues to rise. Relapsed/refractory multiple myeloma (RRMM) refers to cases where the disease progresses after achieving remission with initial treatment, or fails to respond to at least two different treatment regimens [4]. While notable progress has been made in treatment for RRMM recently, managing this disease remains challenging [5].

The therapeutic landscape of MM has evolved in several phases. Initially, chemotherapy was the cornerstone of therapy, with drugs such as melphalan and cyclophosphamide playing key roles. However, chemotherapy induces severe adverse effects, which impairs patients' quality of life [6, 7]. The combined use of proteasome inhibitors and immunomodulatory drugs has significantly improved response rates, as well as extends patients' progression-free survival (PFS) and overall survival (OS) clinically. However, the majority of patients will still eventually experience disease relapse or progression [8].

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In light of these challenges, novel immunotherapies have emerged as new options to clinical practice. Among these, bispecific antibodies and chimeric antigen receptor T-cell (CAR-T) therapy have become focal points in recent years [1].

Bispecific antibodies are a class of antibody molecules capable of simultaneously recognizing and binding to two different antigens. Thanks to innovative molecular design, the antigen-binding regions of two monoclonal antibodies are combined into a single molecular structure, conferring these antibodies with unique biological functions.

In the treatment of MM, bispecific antibodies act like precise molecular bridges. It attaches to specific antigens on the surface of myeloma cells while simultaneously linking to markers on T cells. This dual binding is similar to giving immune cells a built-in navigation system, which effectively activates T cells, guides them directly to tumor cells, and lets them wipe out malignant cells with precision. This innovative treatment method, with its strong targeting ability and promising clinical outcomes, has brought new hope to patients with MM [9]. Teclistamab is a bispecific antibody that targets two molecules, B-cell maturation antigen (BCMA) on myeloma cells and CD3 on T cells. In several clinical studies focused on RRMM, this drug has demonstrated promising therapeutic results. For example, in a clinical trial enrolling RRMM patients [10], nearly two-thirds of those treated with teclistamab obtained satisfactory disease control, with 39.4% achieving deep remission.

CAR-T cell therapy serves as an adoptive cellular immunotherapy approach. The basic principle involves the extraction of T cells from the patient's autologous body, amending them using genetic engineering techniques to express chimeric antigen receptors that can specifically recognize antigens adhering to the surface of tumor cells, followed by administering via infusion the altered T cells back into the patient to produce an anti-tumor effect. As part of the therapeutic approach for MM, CAR-T cells usually target BCMA, as high expression of BCMA is observed on the surface of myeloma cells but has low expression in normal tissues, resulting in high specificity. Several BCMA-targeted CAR-T cell products have shown promising results in clinical trials [11]. For example, in a clinical trial [12], cilta-cel achieved an ORR of up to 84.6% and a complete response rate (CRR) of 73.1% in RRMM patients, significantly extending median PFS and offering hope for long-term survival. As highlighted above, the bispecific antibody teclistamab and CAR-T cell therapy, as two novel therapeutic approaches, have provided new hope to patients with RRMM.

Although both bispecific antibodies and CAR-T cell therapy have demonstrated advantages in the treatment of RRMM, most existing studies focus on evaluating the efficacy of individual therapies, with limited systematic comparative analysis of the two therapies across multiple dimensions. In clinical practice, key questions such as differences in the applicable populations of the two therapies, stability of efficacy, and characteristics of long-term benefits remain unanswered, resulting in obvious gaps in research in this field. By conducting a comprehensive comparison of the core indicators between the two therapies, this study aims to provide high-quality comparative data to inform the RRMM immunotherapy field, enhance the theoretical framework for multiple myeloma treatment, and offer important references and research directions for future studies.

Materials and methods

Patient selection

We screened the medical records of patients with RRMM who were diagnosed and treated at Xinxiang Central Hospital from December 2024 to May 2025. **Figure 1** illustrates the study flow diagram. A total of 73 patients were initially screened. After exclusions, 71 patients remained. Two patients were excluded due to abnormal data, and another two were lost during follow-up. Finally, 67 patients were included in the retrospective comparative analysis. The patients were divided into two groups based on the treatment they received. The teclistamab group (32 patients) received teclistamab, while the CAR-T therapy group (35 patients) received CAR-T therapy.

Inclusion criteria: (1) Meeting the relevant diagnostic criteria outlined in the MMEHA-ESMO Clinical Practice Guidelines for Diagnosis, Treatment, and Follow-Up of Multiple Myeloma [13]; (2) Having received at least one frontline

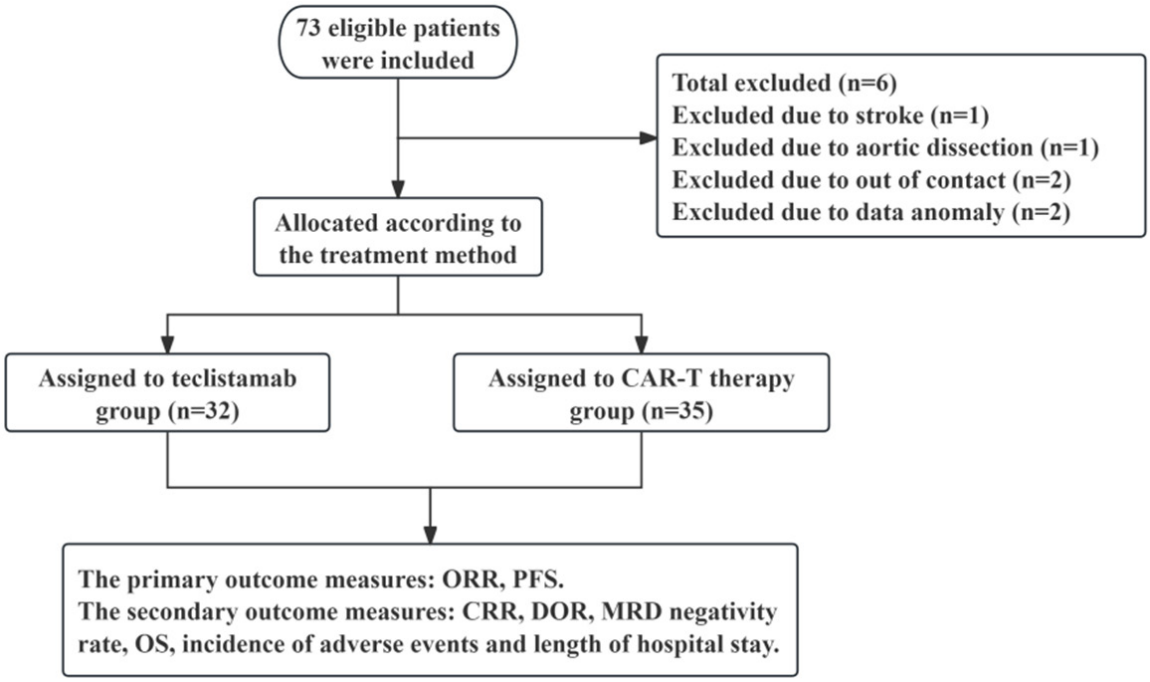

Figure 1. Research flowchart. Note: A total of 73 patients were initially screened in this study. After exclusion, 71 cases were included, among which 2 were lost to follow-up and 2 cases had data anomaly. Ultimately, a total of 67 cases were analyzed, with 32 in the teclistamab group and 35 in CAR-T therapy group. ORR: overall response rate; PFS: progression-free survival; CRR: complete response rate; DOR: duration of response; MRD: minimal residual disease; OS: overall survival; CAR-T: chimeric antigen receptor T-cell.

treatment regimen, with clinical relapse meeting one or more of the following criteria: development of new bone lesions or soft tissue plasmacytomas (excluding osteoporotic fractures); a confirmed increase (a ≥50% rise in the summation of the outputs of measurable lesions' maximum vertical diameters, alongside an absolute value of ≥1 cm) in existing plasmacytomas or bone lesions; hypercalcemia (>2.75 mmol/L); a decrease in hemoglobin ≥20 g/L (unrelated to treatment or non-MM factors); an increase in serum creatinine ≥176.8 μmol/L (2 mg/dl) since the initiation of MM treatment, with the increase being MM-related; serum M protein-associated hyperviscosity syndrome [14]; (3) Eastern Cooperative Oncology Group (ECOG) performance status score of 0-2; (4) Age ≥18 years; (5) No history of receiving other immunotherapies within the past 3 months; (6) Provision of written informed consent to participate in this study.

Exclusion Criteria: (1) Allergy to the study drugs or relevant adjuvant drugs; (2) Presence of severe liver or kidney disease; (3) Have participated in other clinical trials or received other treatments within 3 months; (4) Pregnant or lactating women; (5) Presence of mental illness or cognitive impairment; (6) Presence of uncontrolled active infection; (7) Presence of severe cardiovascular, cerebrovascular, or pulmonary disease; (8) History of other malignant tumors [15].

Data extraction

The extracted data included patients' baseline characteristics, efficacy metrics, safety, and hospitalization-related information. To ensure data reliability, two researchers independently extracted and cross-checked the data.

Treatment regimens

The treatment regimens in this study were determined based on actual clinical treatment decisions, and patients were grouped according to the treatment methods documented in their medical records.

Patients assigned to the teclistamab group received therapy with teclistamab Injection (TECVAYLI, Johnson and Johnson, U.S.), admin-

istered via subcutaneous injection. The first dose was 0.06 mg/kg, the second dose was 0.3 mg/kg, and the third therapeutic dose was 1.5 mg/kg. These three doses were administered at intervals of 2 to 4 days, with all three doses completed within approximately 7 to 10 days. Subsequent maintenance doses of 1.5 mg/kg were administered weekly [16]. If complete response (CR) or partial response (PR) was achieved during treatment, the same regimen continued. If there was no response or insufficient response, the treatment should be switched to CAR-T therapy, an antibody-drug conjugate (ADC), or an XPO1 inhibitor.

Patients in the experimental cohort received Ciltacabtagene Autoleucel Injection (Carvykti, Legend Biotech, China). A reduced-dose VDT-PACE regimen was used for one cycle of chemotherapy to reduce the patient's tumor burden. After the tumor burden was reduced, peripheral blood was collected from the patient to prepare BCMA-targeted CAR-T cells, which took approximately 2 weeks. Before CAR-T therapy, blood biochemical tests and serum M-protein detection were performed, followed by chemotherapy using the FC regimen. Specific administration: from Day 1 to Day 3, fludarabine phosphate (50 mg) and cyclophosphamide (600 mg) were administered via intravenous infusion. From Day 5, BCMA-CAR-T cells were intravenously infused. The infusion dose of CAR-T cells was 2.42×10^6 to 2.09×10^7 cells per kg of body weight, and the infusion was completed in 1 to 3 divided doses [17]. If CR or PR was achieved during treatment, the same regimen continued. If no response or relapse occurred during treatment, the patient should be switched to teclistamab, an ADC, or an XPO1 inhibitor.

In the treatment of RRMM with teclistamab or CAR-T therapy, the management of adverse events was as follows: Cytokine release syndrome (CRS) management: Grade 1: Fluid resuscitation, antipyretic therapy, and close monitoring were recommended as core interventions. Grade 2: Tocilizumab was added to the supportive care regimen; if no response was achieved or symptoms progressed, glucocorticoids were incorporated into the treatment. Grades 3-4: Patients were transferred to the ICU, where high-dose glucocorticoid therapy and advanced life support were promptly initiated.

Immune effector cell-associated neurotoxicity syndrome (ICANS) management: Grade 1: Monitoring was emphasized, and the avoidance of sedative drugs was recommended. Grade 2: Glucocorticoids were administered. Grades 3-4: Patients were transferred to the ICU for intensive treatment; tocilizumab was suspended, other etiologies were actively investigated, and symptomatic support was provided.

Hematological adverse events management: Neutropenia: Granulocyte colony-stimulating factor was used, and prophylactic antibiotics were administered as needed. Thrombocytopenia: Platelet transfusion was performed. Anemia: Red blood cell transfusion was performed.

Outcome measures

Primary outcome measures

ORR: ORR refers to the proportion of patients who achieve one of the three response statuses - CR, very good partial response (VGPR), or PR - among all treated patients. According to the Chinese Guidelines for the Diagnosis and Treatment of Multiple Myeloma (2024 Revision) [14]: (1) CR is defined as negative serum and urine immunofixation electrophoresis, disappearance of soft tissue plasmacytomas, and <5% plasma cells in the bone marrow; (2) VGPR is defined as undetectable M protein by serum protein electrophoresis, or ≥90% reduction in M protein, plus urine M protein <100 mg/24 h, with serum and urine immunofixation electrophoresis remaining positive; (3) PR is defined as ≥50% reduction in serum M protein, and either ≥90% reduction in 24-hour urine M protein or a decrease to <200 mg/24 h. If serum and urine M proteins are undetectable, a ≥50% reduction in the difference between involved and uninvolved serum free light chains (FLCs) is required. If serum M protein, urine M protein, and serum FLCs are all unmeasurable, and the baseline bone marrow plasma cell proportion is ≥30%, a ≥50% reduction in the number of bone marrow-resident plasma cells is required. In addition, if soft tissue plasmacytomas are present at baseline, a ≥50% reduction in the sum of the products of the maximum vertical diameters of measurable lesions is required. All serological and urine M protein indicators must be evaluated twice consecutively, with no evidence of new bone lesions or progression of existing bone lesions.

PFS: PFS is specified as the time interval from the date of treatment initiation to the first occurrence of disease progression (PD) or the patient's death.

The main criteria for PD are: (1) A ≥10% increase in the proportion of clonal plasma cells in the bone marrow compared with the post-treatment nadir, reaching an absolute value of ≥60%; confirmation of new bone marrow plasmacytomas via aspiration and biopsy with a plasma cell proportion of ≥10%; (2) A ≥25% increase in serum M protein from the post-treatment nadir; a ≥25% increase in urine M protein; if serum or urine M protein becomes undetectable levels after treatment, the reappearance of detectable M protein (positive immunofixation electrophoresis) is considered PD; (3) Development of new osteolytic lesions, enlargement of existing osteolytic lesions (≥50% increment in maximum diameter); occurrence of pathological fractures; a ≥50% increase in the sum of maximum diameters of existing soft tissue plasmacytomas, or development of new soft tissue plasmacytomas. New-onset or worsening anemia, hypercalcemia, or renal impairment also qualify as PD [14].

Patient death refers to death from any cause, regardless of its relationship to RRMM, and is confirmed based on the patient's medical records.

Secondary outcome measures

CRR: CRR refers to the proportion of patients who achieve CR among all treated patients.

Duration of response (DOR): DOR is the time interval from the date when RRMM patients first achieve any level of response (CR, VGPR, PR) after treatment to the first occurrence of PD or death [18].

Minimal residual disease (MRD) negativity rate: The MRD negativity rate refers to the proportion of RRMM patients with no detectable clonal plasma cells or myeloma cells, as assessed by next-generation sequencing (NGS), among all treated patients [14].

OS: OS is the time interval from the first day of treatment to the patient's death or the follow-up cutoff date (August 31, 2025). It is the only definitive endpoint that directly reflects patient survival [19].

Incidence of adverse events: Adverse events associated with teclistamab or CAR-T therapy include CRS, ICANS, and hematological adverse events [20, 21]. Incidence is calculated as the proportion of patients experiencing these three types of adverse events among the total number of patients.

Length of hospital stay: For RRMM patients undergoing treatment, the length of hospital stay refers to the continuous interval from the date of admission to the hospital for receiving teclistamab or CAR-T therapy or managing treatment-related adverse events [22] to the date when the patient meets the discharge criteria and completes the discharge procedures. The duration was obtained from the patient's medical records.

Direct medical costs and cost-effectiveness ratio (CER): Direct medical costs refer to the expenses directly paid to the hospital for receiving teclistamab or CAR-T therapy or managing treatment-related adverse events. These costs were obtained from the patient's medical records. CER is defined as the direct medical cost required to extend PFS by one month.

Statistical analysis

Statistical analysis was performed using SPSS 27 software. Normally distributed continuous variables were described as mean ± standard deviation (SD) and compared using independent samples t-test. Non-normal distribution data were expressed as median (IQR), and inter-group comparisons were performed using the *Mann-Whitney U* test. For categorical variables, data were presented as n (%) and compared using the *Chi-Square* test. Survival curves were plotted using the Kaplan-Meier method with GraphPad Prism 9.5 software, and intergroup comparisons were performed using the log-rank test. Risk factor analysis was performed using the Cox proportional hazards model. A two-tailed *P*-value <0.05 was considered statistically significant.

Results

Comparison of patients' baseline characteristics

Baseline information was collected from patients' medical records, including age, body mass index (BMI), gender, ECOG PS score, smok-

Table 1. Comparison of baseline data [mean ± SD, n (%)]

Variables	Teclistamab group (n=32)	CAR-T therapy group (n=35)	95% CI	P	Effect size (Cohen'D/Phi/Cramer'V)
Age (years)	46.97±8.59	48.51±8.39	-5.69, 2.60	0.459	-0.182
BMI (kg/m^2)	23.52±1.32	23.79±1.40	-0.94, 0.39	0.417	-0.200
Gender					
Male	18 (56.3)	19 (54.3)	-	0.872	0.02
Female	14 (43.8)	16 (45.7)	-		
ECOG PS score					
0	12 (37.5)	10 (28.6)	-	0.676	0.108
1	14 (43.8)	16 (45.7)			
2	6 (18.8)	9 (25.7)			
Smoking	18 (56.3)	20 (57.1)	-	0.941	-0.009
Drinking	20 (62.5)	21 (60.0)	-	0.834	0.026
R-ISS stage					
I	6 (18.8)	7 (20.0)		0.964	0.065
II	16 (50.0)	18 (51.4)			
III	7 (21.9)	6 (17.1)			
Unknown	3 (9.4)	4 (11.4)			
PCL	3 (9.4)	4 (11.4)		0.784	-0.034
EMP	4 (12.5)	5 (14.3)		0.830	-0.026
Previous treatment					
PI	30 (93.8)	32 (91.4)		0.718	0.044
IMiD	28 (87.5)	33 (94.3)		0.331	-0.119
Anti-CD38 antibody	26 (81.3)	30 (85.7)		0.622	-0.06
Double-refractory	27 (84.4)	28 (80.0)		0.641	0.057
Triple-refractory	30 (93.8)	27 (77.1)		0.057	0.233
Penta-refractory	26 (81.3)	24 (68.6)		0.234	0.146
CAR T-cell	25 (78.1)	27 (77.1)		0.923	0.012
Unknown	4 (12.5)	5 (14.3)		0.830	-0.026

Note: BMI: body mass index; ECOG PS: eastern cooperative oncology group performance status; R-ISS: revised international staging system; PCL: plasma cell leukemia; EMP: extramedullary plasmacytoma; PI: protease inhibitor; IMiD: immunomodulatory drugs; CAR-T: chimeric antigen receptor T-cell; SD: standard deviation; CI: confidence interval.

ing history, drinking history, R-ISS stage, presence of plasma cell leukemia or extramedullary plasmacytoma, and previous treatment regimens. No statistically significant disparities were noted in the aforementioned baseline characteristics (all $P>0.05$), indicating that the two groups of patients were comparable and appropriate for the comparison of outcome measures. See **Table 1**.

Multivariable regression analysis controls for confounding variables

Multivariable regression analysis revealed that, after adjustment for age, sex, BMI, ECOG PS score, smoking and drinking history, R-ISS stage, and previous treatment regimens, the results demonstrated that the survival benefits brought by CAR-T and teclistamab were attributed to the therapies themselves ($P=0.011$), rather than differences in other factors. See **Table 2**.

Comparison of ORR, CRR, and MRD negativity rate

In the teclistamab group, 20 patients achieved CR (62.5%), 26 achieved an overall response (OR; 81.3%), and 7 achieved MRD negativity (21.9%). In the CAR-T group, 30 achieved CR (85.7%), 34 achieved OR (97.1%), and 16 achieved MRD negativity (45.7%). The CAR-T therapy group had significantly higher CRR, ORR, and MRD negativity rate than the teclis-

Table 2. Multivariate regression analysis in patients with RRMM

Variable (n=67)	Beta Coefficient	Standard Error	P	OR	OR 95% CI
Treatment method (teclistamab/CAR-T)	1.382	0.543	0.011	3.983	1.374, 11.548
Age (years)	-0.002	0.028	0.957	0.998	0.945, 1.055
Gender (Male/Female)	-0.247	0.193	0.201	0.781	0.535, 1.140
BMI (kg/m^2)	-0.096	0.594	0.871	0.908	0.284, 2.907
ECOG PS score (0/1/2)	0.374	0.361	0.300	1.453	0.716, 2.949
Smoking	-0.637	0.590	0.280	0.529	0.166, 1.680
Drinking	-0.224	0.497	0.653	0.799	0.302, 2.119
R-ISS stage (I/II/III/Unknown)	-0.013	0.308	0.966	0.987	0.540, 1.805
PCL	-0.356	0.873	0.684	0.701	0.127, 3.878
EMP	0.937	0.911	0.304	2.551	0.428, 15.207
Previous treatment					
PI	-1.082	1.452	0.456	0.339	0.020, 5.838
IMiD	1.124	1.338	0.401	3.078	0.223, 42.416
Anti-CD38 antibody	-1.358	1.116	0.224	0.257	0.029, 2.292
Double-refractory	-1.058	1.034	0.306	0.347	0.046, 2.634
Triple-refractory	1.569	1.442	0.277	4.803	0.284, 81.146
Penta-refractory	1.183	0.909	0.193	3.264	0.550, 19.376
CAR T-cell	-0.131	0.945	0.890	0.877	0.138, 5.586
Unknown	-0.757	1.062	0.476	0.469	0.058, 3.763

Note: BMI: body mass index; ECOG PS: eastern cooperative oncology group performance status; R-ISS: revised international staging system; PCL: plasma cell leukemia; EMP: extramedullary plasmacytoma; PI: protease inhibitor; IMiD: immunomodulatory drugs; CAR-T: chimeric antigen receptor T-cell; RRMM: Relapsed and Refractory Multiple Myeloma; OR: odds ratio; CI: confidence interval.

Table 3. Comparison of CRR/ORR/MRD negativity [n (%)]

Variables	CR	OR	MRD negativity
Control group (n=32)	20 (62.5)	26 (81.3)	7 (21.9)
Experimental group (n=35)	30 (85.7)	34 (97.1)	16 (45.7)
P	0.029	0.034	0.040
Effect size (Phi)	-0.266	-0.260	-0.251

Note: ORR: overall response rate; CRR: complete response rate; MRD: minimal residual disease; CR: complete response; OR: overall response.

tamab group (*P*=0.029, *P*=0.034, *P*=0.040, respectively). From the above results, it can be concluded that for addressing RRMM, CAR-T therapy demonstrates superiority over the bispecific antibody teclistamab therapy in terms of patients' OR, CR, and MRD status. See **Table 3**.

Comparison of PFS, DOR, and OS

Consistently, the CAR-T group outperformed the teclistamab group in PFS, DOR, and OS (all P<0.05; **Figure 2**). For PFS, the teclistamab group had a median PFS of 9.9 months with a PFS rate of 40.6%, while 71.4% of patients in the CAR-T group remained progression-free, and the CAR-T group had significantly longer PFS (HR=4.017, P<0.001). For DOR, the median DOR was 8 months in the teclistamab group versus 10 months in the CAR-T group, and the CAR-T group had longer DOR (HR=2.618, P<0.001). Regarding OS, median OS was not reached in either group, but the CAR-T group had a higher OS rate (82.9% vs. 75%), with statistically significant difference (P=0.028).

Comparison of the incidence of adverse events

Patients receiving CAR-T therapy experienced significantly higher rates of CRS and blood-related side effects (P=0.033; P=0.040). The risk of ICANS was pretty similar between the two groups (P=0.495) (**Table 4**). These findings suggested that CAR-T therapy has a unique toxicity pattern, mainly marked by more frequent CRS and hematological adverse events.

Comparison of length of hospital stay

The mean length of hospital stay was 3.38±1.68 days in the teclistamab group and 21.26±2.56

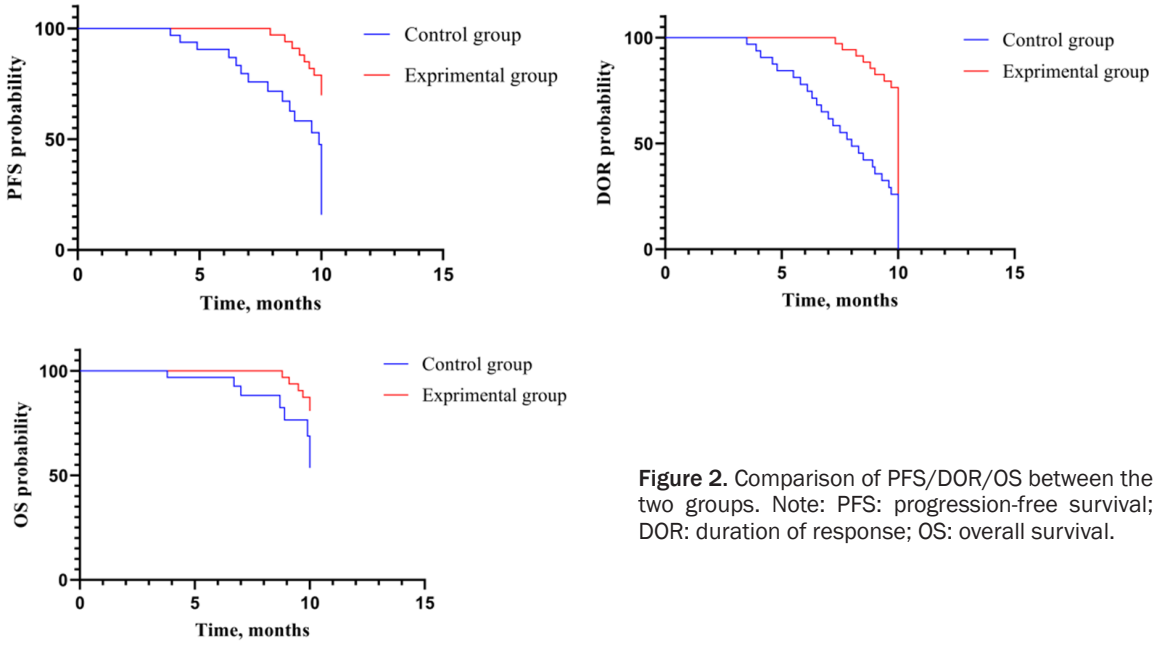

Figure 2. Comparison of PFS/DOR/OS between the two groups. Note: PFS: progression-free survival; DOR: duration of response; OS: overall survival.

Table 4. Comparison of adverse events [n (%)]

Variables	CRS	ICANS	Blood			Total
			Neutropenia	Anemia	Thrombocytopenia	
Teclistamab group (n=32)	19 (59.4)	6 (18.8)	16 (50.0)	11 (34.4)	8 (25.0)	17 (53.1)
CAR-T therapy group (n=35)	29 (82.9)	9 (25.7)	26 (74.3)	21 (60.0)	17 (48.6)	27 (77.1)
P	0.033	0.495	0.040	0.036	0.046	0.039
Effect size (Phi)	-0.26	-0.083	-0.251	-0.256	-0.243	-0.253

Note: CRS: cytokine release syndrome; ICANS: immune effector cell-associated neurotoxicity syndrome; CAR-T: chimeric antigen receptor T-cell.

Table 5. Comparison of length of hospital stay [mean ± SD, days/year]

Variables	Length of hospital stay
Control group (n=32)	3.38±1.68
Experimental group (n=35)	21.26±2.56
P	<0.001
95% CI	-18.95, -16.82
Effect size (Cohen's D)	-8.186

Note: SD: standard deviation.

days in the CAR-T therapy group. The results indicated that the length of hospital stay for patients treated via CAR-T therapy exceeded that for patients treated with teclistamab by a significant margin (*P*<0.001, *95% CI*: -18.95, -16.82) (**Table 5**).

Comparison of direct medical costs and CER

Both direct medical costs and CER of patients treated with CAR-T therapy were significantly higher than those of patients treated with teclistamab (*P*<0.001, *95% CI*: -1106612.12, -1092696.99; CER: *P*<0.001, *95% CI*: -111348.379, -100249.106). See **Table 6**.

Cox regression analysis of independent risk factors

Cox regression analysis revealed that, after adjustment for age, gender, BMI, ECOG PS score, smoking and drinking history, R-ISS stage, and number of prior treatment lines, the Cox regression model indicated that the number of prior treatment lines was an independent prognostic factor (*P*=0.036). See **Table 7**.

Discussion

MM is a malignant proliferative disease of plasma cells. Although traditional therapies have significantly improved the prognosis of newly diagnosed patients, more than 80% of patients still progress to RRMM within 3-5 years after

Table 6. Comparison of direct medical costs and CER [mean ± SD]

Variables	Direct medical costs (yuan)	CER (yuan/month of PFS)
Control group (n=32)	249796.88±7328.05	34454.35±11082.16
Experimental group (n=35)	1349451.43±18941.53	140253.10±1962.44
P	<0.001	<0.001
95% CI	-1106612.12, -1092696.99	-111348.379, -100249.106
Effect size (Cohen's D)	-75.297	-9.312

Note: CER: cost-effectiveness ratio; SD: standard deviation.

Table 7. Cox analysis of influencing factors in patients with RRMM

Variable (n=67)	Beta Coefficient	Standard Error	P	OR	OR 95% CI
Age (years)	0.000	0.026	0.996	1.000	0.951, 1.052
BMI (kg/m^2)	-0.229	0.144	0.112	0.795	0.600, 1.055
Gender (Male/Female)	-0.081	0.454	0.858	0.922	0.379, 2.244
ECOG PS score (0/1/2)	0.144	0.275	0.600	1.155	0.674, 1.981
Smoking	-0.536	0.431	0.214	0.585	0.251, 1.362
Drinking	-0.271	0.441	0.538	0.762	0.321, 1.808
R-ISS stage (I/II/III/Unknown)	0.342	0.274	0.212	1.408	0.823, 2.410
PCL	0.376	0.728	0.606	1.456	0.350, 6.062
EMP	0.479	0.744	0.520	1.614	0.376, 6.937
Previous Lines of Therapy	0.192	0.092	0.036	1.211	1.012, 1.450

Note: BMI: body mass index; ECOG PS: Eastern Cooperative Oncology Group Performance Status; R-ISS: Revised International Staging System; PCL: plasma cell leukemia; EMP: extramedullary plasmacytoma; RRMM: Relapsed and Refractory Multiple Myeloma; OR: odds ratio; CI: confidence interval.

treatment. In RRMM, the difficulty of treatment increases significantly owing to previous exposure to multiple lines of therapeutic intervention and the formation of drug-resistant clones of tumor cells. Thus, there remains an urgent clinical need for more effective novel therapeutic approaches [23]. In recent years, the bispecific antibody teclistamab and CAR-T therapy have emerged as major research hotspots in the field of RRMM treatment, thanks to their advantages of strong targeting and significant efficacy. However, their clinical positioning, differences in efficacy, and safety profiles in the treatment of RRMM have not yet been clarified, posing challenges to the selection of clinical regimens. This study's findings suggest that CAR-T therapy achieved notably higher CRR and ORR than teclistamab. A potential mechanism underlying this advantage is that CAR-T cell therapy uses genetic engineering technology to introduce BCMA-targeting chimeric antigen receptors into autologous T cells derived from the patient. After infusion of these modified CAR-T cells, they can continuously clear myeloma cells through a closed-loop process of antigen recognition, activation and proliferation, and specific killing. As a "living cell drug", this therapy can penetrate the bone marrow microenvironment and eliminate residual lesions that with traditional treatments rarely reach [24]. The elevated CRR observed in the CAR-T therapy group in this study directly reflects this potent cytotoxic capacity. Previous clinical studies have confirmed that the magnitude of disease response in RRMM patients is significantly associated with long-term prognosis. That is, the higher the CRR, the lower the hazard of subsequent disease progression and the longer the survival time of patients [25, 26]. For example, in the CARTITUDE-1 study, RRMM patients treated with cilta-cel achieved a CRR of over 70%, with a 3-year PFS rate of 50%-far higher than that of non-CR patients [27]. Our findings correspond with the conclusions from these prior findings, suggesting that CAR-T therapy provides a better survival foundation by improving CRR. In contrast, teclistamab, as a bispecific antibody, exerts effects relying on the transient interaction of bridging T cells and myeloma cells. The drug binds to myeloma cells via its BCMA-targeting end and to T cells via its CD3-targeting end, thereby activating the killing

function of T cells. However, this effect requires sustained drug exposure to maintain and is limited by the activity and quantity of T cells in the peripheral blood. When the drug concentration decreases, or T cell exhaustion occurs, the killing effect weakens, leading to obstacles that impede its realization complete clearance of myeloma cells [28]. This explains the teclistamab group's CRR and ORR being lower than those of the CAR-T therapy group. Although Tadao et al. [29] confirmed that the ORR of teclistamab (76.9%) is significantly superior to that of traditional chemotherapy, it cannot compete with CAR-T in terms of deep response, which further accounts for its inferior performance in PFS and OS as opposed to the group administered CAR-T therapy. Our results also showed that the DOR of teclistamab was shorter than that of CAR-T. The core reason for this difference lies in whether the two therapies can induce immune memory, which is crucial for achieving sustained treatment-free remission over the long term in RRMM. A primary merit of CAR-T cell therapy resides in its ability to form a pool of immune memory cells in the patient's body. After CAR-T cell infusion, some CAR-T cells differentiate into memory T cells, which can survive in the body for a long time. When myeloma cells reappear, these memory cells can be quickly activated and initiate a killing response, thereby maintaining remission state [30]. The longer DOR in the CAR-T therapy group in this study is a manifestation of this immune memory effect. For instance, in the LEGEND-2 study, RRMM patients receiving BCMA-targeted CAR-T therapy achieved a median DOR of over 30 months, and some maintained remission for more than 5 years, proving the long-term protective effect of immune memory [31]. In contrast, the DOR of teclistamab depends on continuous drug administration. Patients need weekly subcutaneous injections to maintain the blood drug concentration. Once treatment stops, the drug is metabolized and cleared within a few weeks; the cytotoxic effect of T cells disappears accordingly, and myeloma cells are prone to re-proliferation, leading to the loss of remission. The shorter DOR observed in this study aligns with the results of the MajesTEC-1 study (median DOR: 18.4 months) [32], suggesting that the durability of its remission is limited by the pharmacokinetic properties of the drug and cannot form long-term immune protection. In clinical practice, this means that patients receiving teclistamab therapy need to adhere to long-term administration and face the risk of disease progression after drug withdrawal, whereas patients receiving CAR-T therapy do not require subsequent treatment after achieving remission, significantly improving their quality of life. Additionally, the outcomes of this research indicated that a higher proportion of patients in the CAR-T therapy group achieved MRD negativity. MRD negativity is an important marker of functional cure in RRMM patients, and its rate is directly associated with long-term relapse-free survival; if MRD is positive, the likelihood of recurrence remains notably high. The higher MRD-negativity rate with CAR-T therapy reflects its superior ability to eradicate microresidual disease. CAR-T cells can penetrate bone marrow stromal barrier, recognize and kill BCMA-expressing minimal lesions, and their immune memory effect can continuously monitor and eliminate newly emerging myeloma cells, thereby maintaining MRD negativity [33]. To illustrate, in the CARTITUDE-1 study [26], RRMM patients treated with cilta-cel achieved an MRD negativity rate of 83%, and 70% maintained MRD negativity after 3 years, corresponding to a 5-year OS rate of over 60%. The results of this study are congruent with these findings, indicating that CAR-T therapy reduces the risk of recurrence for patients by increasing the MRD negativity rate. The lower MRD negativity rate in the teclistamab group is mainly due to its inability to penetrate bone marrow microenvironment and completely clear microlesions. The drug exerts its effect relying on peripheral blood circulation and is difficult to penetrate the protective barrier formed by bone marrow stromal cells; moreover, it lacks sustained killing ability, leading to the survival and gradual proliferation of some minimal residual lesions [34].

The findings of this study suggest that the longer PFS in the CAR-T group stems from the deep and durable elimination of myeloma cells. Higher CRR and MRD-negative rates translate to lower tumor burden and reduced progression risk in patients. The immune memory effect further delays disease progression, enabling patients to maintain a progression-free state for an extended period. Moreover, OS was superior in the CAR-T group, likely due to a reduced risk of disease progression and fewer requirements for subsequent therapies, there-

by lowering treatment-related risks. Collectively, these factors contribute to the survival advantage provided by CAR-T therapy. CAR-T therapy presents a distinct safety concern, it causes notably more cases of CRS and blood-related side effects than teclistamab. The core reason for the difference in CRS lies in the fact that CAR-T cells can proliferate 100-1000 folds in vivo, which is far higher than the count of T cells activated via teclistamab. Consequently, the total cytokines released is greater, resulting in an elevated risk of CRS. Prior to CAR-T therapy, lymphodepleting preconditioning is required, whose purpose is to clear the patient's own lymphocytes and create space for the proliferation of CAR-T cells. However, this preconditioning chemotherapy can damage bone marrow hematopoietic stem cells, resulting in severe and prolonged myelosuppression after treatment. The most common manifestations of myelosuppression include neutropenia, anemia, and thrombocytopenia [35]. In contrast, teclistamab does not require preconditioning chemotherapy. Its hematological adverse events mainly stem from the mild impact of T-cell activation on hematopoietic function. Although T cells activated by teclistamab release a small amount of cytokines, the damage to bone marrow hematopoietic stem cells is limited. Therefore, both the severity and duration of myelosuppression are milder [36]. In this study, length of hospital stay for patients receiving CAR-T therapy was significantly longer than that for those treated with teclistamab. Differences in hospitalization duration affect not only the consumption of medical resources but also are directly related to patients' quality of life and treatment accessibility. For patients in the CAR-T therapy group, the first hospitalization lasting up to one month and the possible subsequent re-hospitalizations will significantly increase medical costs and severely disrupt patients' daily lives. In contrast, the short hospitalization associated with teclistamab greatly reduces the time cost and economic burden on patients.

Study limitations

This study has several key limitations. As a single-center retrospective analysis, all data were extracted from patients' medical records, introducing potential selection and information biases. Only patients who received one of the two treatment regimens were included in the study, which may overestimation therapeutic efficacy compared with real-world clinical settings. Additionally, the documentation of information in medical records relies on the subjective descriptions of healthcare providers, which may introduce deviations in the assessment of long-term outcome indicators and reduce the accuracy of the results. This study included only 67 patients from a single institution. The small sample size undermines the statistical reliability of the findings, particularly when calculating the incidence of adverse reactions. Furthermore, the baseline characteristics of patients from a single center may be associated with regional or institutional-specific factors, which limits the generalizability of the study results. Third, although the International Myeloma Working Group criteria were referenced, methodological inconsistencies in efficacy and safety assessments, and some delayed adverse events may have been missed. Fourth, potential confounding factors were not fully considered, such as differences in patients' subsequent treatment regimens and varying intensities of medical management, which may have obscured the true impact of the initial treatment on survival and safety. In the future, multi-center, prospective head-to-head studies are required to validate these findings, explore the optimal treatment sequence for the two therapies, and provide more precise treatment strategies for patients with RRMM.

Conclusions

This study demonstrates that BCMA-targeted CAR-T therapy induces deep and long-term remission in RRMM patients, with teclistamab superior in safety and convenience. Clinically, individualized treatment plans can be formulated based on the specific conditions of patients.

Disclosure of conflict of interest

None.

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