Relapsed/refractory multiple myeloma with extramedullary plasmacytomas successfully rechallenged with humanized B-cell maturation antigen-directed chimeric antigen receptor T-cell immunotherapy in an advanced disease stage: A case report

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Abstract. Multiple myeloma (MM) is a plasma cell malignancy, and extramedullary plasmacytoma (EMP) is associated with an aggressive biology and poor outcomes. B-cell maturation antigen (BCMA)-directed chimeric antigen receptor T-cell (CAR-T) therapy has shown substantial activity in relapsed/refractory MM (RRMM); however, evidence in patients with EMP, particularly after prior BCMA CAR-T exposure, remains limited. The present study describes the case of a 38-year-old man with RRMM and extensive cranial EMP who progressed after multiple prior therapies, including proteasome inhibitor- and immunomodulatory drug-based regimens, anti-CD38 therapy, carfilzomib-based therapy and a previous non-humanized BCMA CAR-T product. Due to rapidly progressive disease, the patient received bridging cytoreduction followed by fully humanized BCMA CAR-T therapy (equecabtagene autoleucel). After infusion, serum and urine M-protein levels rapidly declined and the craniofacial mass regressed clinically and radiographically. The patient achieved stringent complete response with durable control of extramedullary disease during follow-up. The present case suggests that fully humanized BCMA CAR-T therapy may remain effective in selected patients with RRMM and EMP, even after prior non-humanized BCMA CAR-T failure. These findings support further investigation into retreatment strategies and post-CAR-T maintenance approaches.

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Introduction

Multiple myeloma (MM) is a malignant plasma cell disorder characterized by clonal proliferation of terminally differentiated B cells in the bone marrow and by end-organ damage related to monoclonal protein production, osteolytic bone disease, anemia, hypercalcemia, renal injury or a combination of these manifestations (1,2). Although therapeutic outcomes have improved substantially with the introduction of proteasome inhibitors (PIs), immunomodulatory drugs (IMiDs), anti-CD38 monoclonal antibodies, autologous stem cell transplantation, bispecific antibodies and cellular therapies, MM remains largely incurable once patients enter the relapsed/refractory phase (1-4).

Extramedullary plasmacytomas (EMP), particularly soft-tissue lesions that are biologically independent of the bone marrow microenvironment, represent one of the most challenging manifestations of MM; ~7% of patients have extramedullary disease at diagnosis, and an additional 6-20% develop it later during the disease course (5,6). EMP is typically associated with high tumor burden, clonal evolution, treatment resistance and inferior survival, and it is often regarded as a marker of biologically aggressive disease (5,6). In the setting of relapsed/refractory MM (RRMM), durable disease control becomes especially difficult when extramedullary lesions coexist with acquired high-risk cytogenetic abnormalities or prior exposure to multiple major drug classes.

B-cell maturation antigen (BCMA) is an established therapeutic target in RRMM because it is preferentially expressed on malignant plasma cells and plays an important role in plasma cell survival. Current BCMA-directed approaches include antibody-drug conjugates, bispecific antibodies and chimeric antigen receptor T-cell (CAR-T) therapy (3,4). Among these, CAR-T therapy can induce deep responses in heavily pretreated patients; however, outcomes remain less favorable in patients with EMP, and relapse after BCMA CAR-T therapy remains a clinically important problem (4,7,8). Potential mechanisms of resistance include insufficient CAR-T expansion or persistence,

T-cell exhaustion, antigen modulation and the immunosuppressive tumor microenvironment (7,9). Retreatment after failure of an earlier BCMA-directed CAR-T product is not yet standardized. Nevertheless, it is biologically plausible that a fully humanized CAR construct could retain activity in selected patients previously exposed to a non-humanized product, particularly if reduced immunogenicity improves expansion and persistence after reinfusion (7,8,10,11). In parallel, the role of post-CAR-T maintenance therapy in MM is under ative investigation, especially for patients with residual radiographic disease or high-risk clinical features (12-15).

The present case report describes the clinical course of a patient with advanced RRMM and bulky cranial EMP who had previously received multiple lines of therapy, including bortezomib, lenalidomide, pomalidomide, ixazomib, carfilzomib, daratumumab, radiotherapy, autologous stem cell transplantation and a prior non-humanized BCMA CAR-T product. The aim was to characterize the clinical response and safety following eque-cel treatment and subsequent pomalidomide maintenance.

Case report

A 38-year-old male patient was admitted to Peking Union Medical College Hospital (Beijing, China) in January 2017 with chest pain and a parasternal plasmacytoma and was subsequently diagnosed with MM. Positron emission tomography/computed tomography (PET/CT) demonstrated multiple metabolically active osteolytic lesions, consistent with disseminated myeloma bone disease, together with the parasternal soft-tissue lesion. Serum protein electrophoresis showed an M-protein concentration of 36.4 g/l (normally undetectable in healthy individuals), indicating a substantial circulating monoclonal immunoglobulin (Ig) burden, and serum immunofixation electrophoresis identified an IgG monoclonal band, confirming secretory IgG myeloma. Bone marrow morphology showed 9% plasma cells. Fluorescence *in situ* hybridization (FISH) analysis of CD138-sorted plasma cells did not reveal high-risk cytogenetic abnormalities (data not shown). The patient was diagnosed with MM IgG, Durie-Salmon stage IIIA, International Staging System (ISS) stage II and Revised-ISS (R-ISS) stage II (16,17).

From February 2017, the patient received five cycles of first-line VRd induction therapy consisting of bortezomib 2.7 mg intravenously on days 1, 8, 15 and 22, lenalidomide 25 mg orally on days 1-21 and dexamethasone 40 mg intravenously on days 1, 8, 15 and 22. This treatment was selected because the patient was young and transplant-eligible, and a PI/IMiDs-based triplet is a standard frontline approach in newly diagnosed transplant-eligible MM. A very good partial response (VGPR) was achieved, with resolution of the sternal mass. In September 2017, the patient underwent autologous stem cell transplantation and achieved a stringent CR (sCR). They then received 4 additional cycles of VRd consolidation and later maintenance with lenalidomide 25 mg every other day plus dexamethasone 20 mg orally on days 1, 8, 15 and 22 for ~2 years. In November 2020, IgGλ positivity recurred on serum immunofixation electrophoresis, indicating biochemical relapse.

In January 2021, VPd (bortezomib 2.7 mg intravenously on days 1, 8, 15 and 22, pomalidomide 4 mg orally on days 1-21, and dexamethasone 40 mg intravenously on days 1, 8, 15 and 22) was selected as second-line therapy because the patient had relapsed after lenalidomide maintenance and remained a candidate for a bortezomib-containing regimen, while pomalidomide is active in lenalidomide-exposed disease. After nine cycles, the best response was VGPR. However, by January 2022, the serum M-protein level had risen to 5.88 g/l, indicating disease progression with a progression-free survival (PFS) of 12 months. The third-line regimen was switched to eight cycles of IPd (ixazomib 4 mg on days 1, 8 and 15, pomalidomide 4 mg orally on days 1-21, and dexamethasone 40 mg on days 1, 8, 15 and 22) as an all-oral salvage regimen to maintain proteasome inhibition while changing the PI backbone after progression on bortezomib-based treatment.

In February 2022, a right frontoparietal extramedullary mass measuring 3 cm in diameter developed clinically, and subsequently progressed to involve the right frontoparietal and right temporoparietal regions, enlarging to ~10 cm. PET/CT demonstrated right frontoparietal bone destruction measuring ~107x42 mm with an adjacent soft-tissue mass (SUVmax 9.6). Bone marrow aspiration revealed 5% plasma cells. At this time, FISH detected newly acquired high-risk cytogenetic abnormalities, including IgH/fibroblast growth factor receptor 3 (FGFR3) translocation [t(4;14)] and 1q21 amplification. These abnormalities are considered high risk because t(4;14) is associated with dysregulated FGFR3/multiple myeloma SET domain signaling and inferior survival, whereas 1q21 gain/amplification is linked to copy-number-driven upregulation of genes such as cyclin-dependent kinase regulatory subunit 1B and myeloid cell leukemia 1, promoting proliferation, treatment resistance and poor prognosis (18,19).

The patient was then enrolled in the IBI346 clinical trial of BCMA-targeted CAR-T therapy in October 2022. BCMA is a surface antigen highly expressed on malignant plasma cells. The infused product was a non-humanized CAR-T construct. During CAR-T manufacturing, a new extramedullary mass emerged in the left mandibular area. After infusion, both the left mandibular and right frontoparietal masses continued to grow, and the serum M protein level increased from 32.8 g/l in December 2022 to 38.5 g/l in January 2023, confirming progression after non-humanized BCMA CAR-T therapy.

In March 2023, because the disease was rapidly progressing after non-humanized BCMA CAR-T therapy and urgent cytoreduction was required while other immunotherapeutic options were being arranged, the patient started fifth-line D-KPD (daratumumab 1,200 mg intravenously on days 1, 8, 15 and 22, carfilzomib 30 mg intravenously on days 1 and 2 and then 120 mg intravenously on days 8 and 15 in cycle 1, pomalidomide 4 mg orally on days 1-4 and then every other day on days 8-21 in cycle 1, and dexamethasone 40 mg intravenously on days 1, 8, 15 and 22) treatment and received radiotherapy (planning gross tumor volume 45 Gy/30 fractions) for the left mandibular mass. After five cycles, the M-protein level rose from 11.97 g/l in July 2023 to 24 g/l in August 2023, indicating relapse after a best response of partial response (PR). Pomalidomide was then replaced with 2 cycles of oral selinexor (D-KXD, selinexor 60 mg on days 1, 8, 15 and 22), but neutropenia and thrombocytopenia developed during this sixth-line therapy.

In December 2023, the patient received eque-cel, a fully humanized BCMA CAR-T product approved in China for patients with RRMM after at least three prior lines of therapy. Only fever consistent with grade 1 cytokine release syndrome (CRS) occurred within 2 weeks of infusion.

After 1 month of infusion, the serum M-protein level decreased to 10.32 g/l (January 2024), and the 24-h urine M-protein level became undetectable. After 2 months, the serum M-protein level further declined to 5.18 g/l (February 2024), and by 6 months it had fallen to 0.53 g/l (June 2024). Clinically, the extramedullary masses became softer and smaller within 13 days of eque-cel infusion. Serial MRI imaging documented continuous shrinkage of the right parietal lesion with reduction of the extraosseous soft-tissue component (Figs. 1 and 2). Because residual extramedullary disease persisted radiographically, pomalidomide maintenance was initiated in March 2024 at 4 mg orally every other day on days 1-21 of each cycle, with the aim of consolidating response and potentially supporting CAR-T persistence. At the 6-month follow-up, the serum M-protein level remained at 0.53 g/l. By October 2024, serial serum and urine immunofixation electrophoresis confirmed CR. MRI showed sustained reduction of the right parietal lesion (131.7x50.3 to 133x37 mm), corresponding to a ~20% decrease in the sum of product diameters for extramedullary disease (Fig. 2). CAR-T kinetics showed peak peripheral blood expansion (22.78% CAR^+ cells) at day 11 after initial CAR-T therapy induction. CAR transgene copies in peripheral blood genomic DNA were quantified using a commercial TaqMan real-time PCR assay, the Blood/Cell CAR Copy Number Detection Kit (cat. no. 102100; Shanghai Xingwan Biotechnology Co., Ltd.). The CAR, universal sequence (UNI) and single-copy gene (SCG) primer/probe sets were supplied as a proprietary CAR primer/probe premix included in this kit, with no separate catalogue numbers or oligonucleotide sequences disclosed by the manufacturer. The assay targets the CAR transgene, a UNI and a human SCG, with SCG serving as the endogenous loading control. The reporter channels were FAM for CAR, Cy5 for UNI and VIC for SCG, and CAR copy number was calculated according to the manufacturer's formula: CAR copy number/cell=2 x CAR/SCG. The CAR transgene copy number peaked at $7.13x10^3$ copies/μg DNA, and durable engraftment remained detectable at day 90 ($2.02x10^2$ copies/μg DNA). On the basis of clinical, laboratory and imaging findings, the treatment was considered effective. At the latest follow-up, 19 months after infusion, the patient remained on pomalidomide maintenance therapy with sustained hematologic CR. The timeline of interventions and responses is summarized in Table I.

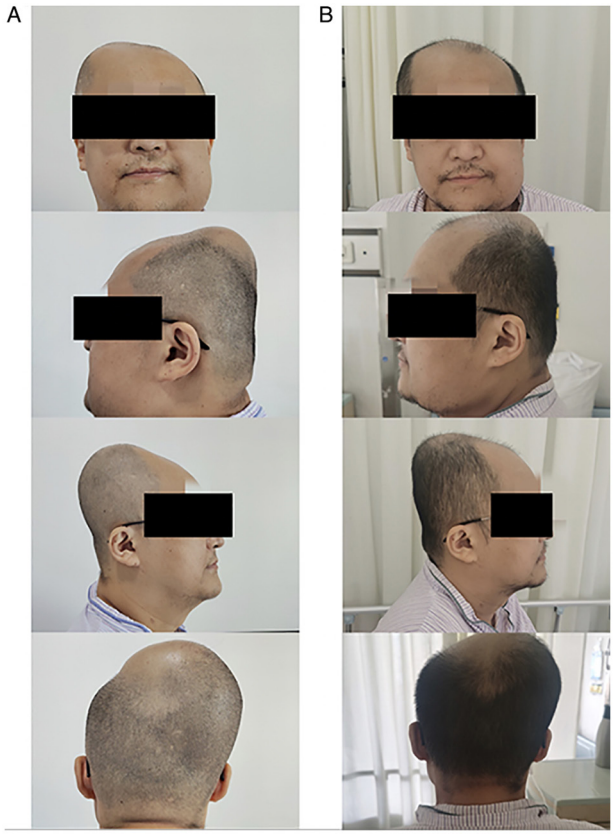

Figure 1. Serial craniofacial photographs taken before and after fully humanized B-cell maturation antigen chimeric antigen receptor T-cell infusion. (A) Baseline images before equecabtagene autoleucel infusion (B) Photographs obtained 13 days after infusion showing visible regression of the craniofacial extramedullary mass.

Discussion

The present study describes the case of a young patient with R-ISS stage II who developed paraosseous extramedullary disease and subsequently became refractory to multiple treatment lines, including PIs (bortezomib, ixazomib and carfilzomib), IMiDs (lenalidomide and pomalidomide), anti-CD38 therapy (daratumumab), autologous transplantation, radiotherapy and a prior non-humanized BCMA CAR-T product. During disease evolution, newly acquired t(4;14) and 1q21 amplification emerged, consistent with clonal selection toward a more aggressive, treatment-resistant phenotype. After progression following non-humanized BCMA CAR-T therapy, additional chemotherapy was not considered contradictory to the immunotherapeutic strategy; rather, it served as necessary bridging cytoreduction to control the rapidly progressive bulky EMP while access to a second BCMA-directed cellular product was being secured. The subsequent hematologic CR and durable disease control achieved with fully humanized BCMA CAR-T therapy suggests that this approach can still be clinically meaningful, even after prior BCMA CAR-T exposure in selected high-risk patients.

The FUMANBA-1 study is the most relevant clinical context for the present case because it evaluated the same fully human BCMA-directed CAR-T product, eque-cel in adults with RRMM (8). In FUMANBA-1, eque-cel produced a high overall response rate (ORR) of 96.0%, with 74.3% of patients achieving CR or better, and the median PFS had not been reached at the reported follow-up, with a 12-month PFS rate of 78.8% (8). The present patient achieved a similarly deep hematologic response, with a sustained sCR and ongoing control of extramedullary disease at 19 months after infusion. However, this patient differed from the general FUMANBA-1 population by having bulky cranial EMP, newly acquired high-risk cytogenetic abnormalities and prior failure of a non-humanized BCMA CAR-T product. Therefore, the durability observed in the present case is clinically notable. The present case does not prove superiority of fully humanized over non-humanized

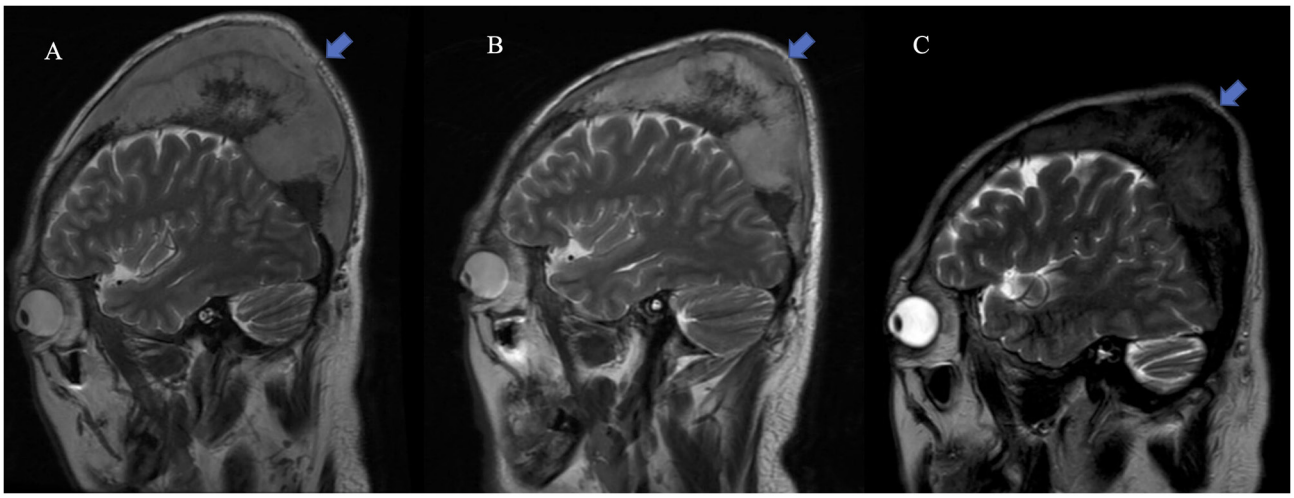

Figure 2. Serial MRI monitoring of the right parietal extramedullary lesion after fully humanized B-cell maturation antigen chimeric antigen receptor T-cell infusion. Closely matched sagittal cutting planes are shown across time points. (A) Baseline, (B) day 13 after infusion and (C) 9 months after infusion, demonstrating progressive reduction of the extraosseous soft-tissue component.

BCMA CAR-T therapy, but it does support the clinical plausibility that reducing immunogenicity and improving *in vivo* persistence may recapture antimyeloma activity after failure of a prior non-humanized construct. This interpretation is biologically reasonable because antigen loss, limited CAR-T expansion/persistence and T-cell exhaustion are recognized mechanisms of relapse after BCMA CAR-T therapy (7).

Although non-humanized BCMA CAR-T therapy had previously failed in the present patient, meaningful efficacy was still achieved after fully humanized BCMA CAR-T treatment. One possible explanation is that non-humanized antigen-recognition domains can induce host anti-CAR immune responses, which may shorten persistence on repeated exposure. By contrast, fully humanized or fully human constructs are designed to reduce immunogenicity while preserving antigen binding, thereby favoring expansion and persistence (10,11). In the present case, durable CAR transgene detection up to day 90 was consistent with this concept, although mechanistic confirmation was not available.

EMP remains an important adverse factor for CAR-T efficacy and survival. In MM, extramedullary spread reflects biological escape from bone marrow dependence, and is associated with altered adhesion and chemokine signaling, spatial clonal heterogeneity, high tumor burden and a more immunosuppressive microenvironment, all of which may impair CAR-T trafficking, expansion and durable tumor control (5,8,9). Consistent with this, the FUMANBA-1 trial reported a 12-month PFS rate of 60.6% in patients with EMP compared with 81.8% in patients without EMP (8). Similarly, a retrospective analysis of commercially available BCMA CAR-T products showed substantially lower ORR, PFS and overall survival in the EMP subgroup compared with in patients without EMP (20). In the present patient, durable control beyond 19 months despite bulky EMP and prior BCMA exposure suggests that fully humanized BCMA CAR-T therapy may still provide a clinically relevant benefit, particularly when followed by carefully selected consolidation or maintenance treatment.

Compared with CARTITUDE-1 and KarMMa, the present case shows both shared and distinct features in terms of response depth, durability and safety. In CARTITUDE-1, ciltacabtagene autoleucel (cilta-cel) produced an ORR of 97.9% and a sCR rate of 82.5%, with the median PFS not reached and a 27-month PFS rate of 54.9% after a median follow-up of 27.7 months (21). In KarMMa, idecabtagene vicleucel (ide-cel) achieved an ORR of 73%, a CR-or-better rate of 33%, and a median PFS of 8.8 months (22). The present patient achieved sustained hematologic sCR with ongoing extramedullary disease control at 19 months after eque-cel infusion, which is directionally consistent with the deep responses observed in pivotal BCMA CAR-T studies. However, unlike most patients in these studies, the present patient had bulky cranial EMP, newly acquired high-risk cytogenetic abnormalities and prior failure of a non-humanized BCMA CAR-T product. With regard to safety, only grade 1 CRS and no immune effector cell-associated neurotoxicity syndrome occurred in this case, whereas CRS and neurotoxicity were reported more frequently in the pivotal CAR-T trials (21,22). Therefore, the present case does not establish superiority of eque-cel over cilta-cel or ide-cel, but suggests that fully humanized BCMA CAR-T therapy may remain active in selected high-risk patients after prior non-humanized BCMA CAR-T failure.

Beyond cellular therapy, the therapeutic landscape of RRMM now also includes BCMA-directed bispecific antibodies, which act as off-the-shelf T-cell engagers and can be administered without individualized cell manufacturing (4). This practical advantage may be particularly important when rapid treatment initiation is required. By contrast, CAR-T therapy requires leukapheresis, individualized manufacturing and often bridging disease control, but it can produce very deep and durable responses in heavily pretreated RRMM (8,21,22). In patients with aggressive EMP, such as the present case, the potential for profound tumor reduction and sustained cellular persistence may be clinically relevant, whereas bispecific antibodies remain an important alternative when immediate access or manufacturing feasibility limits CAR-T treatment. These modalities should therefore be considered complementary

Table I. Clinical timeline of interventions, responses and disease milestones.

Time point	Treatment line	Intervention	Outcome	Extramedullary disease
January 2017	Diagnosis			Sternal EMP
February-August 2017	1st	Bortezomib, lenalidomide and dexamethasone	VGPR	Mass disappeared
September 2017	Consolidation	Autologous stem cell transplantation	sCR	
October 2017-November 2020	Maintenance	Lenalidomide	sCR to PD (41 month PFS)	
January-December 2021	2nd	Bortezomib, pomalidomide and dexamethasone	VGPR to PD (12 month PFS)	
February-August 2022	3rd	Ixazomib, pomalidomide and dexamethasone	SD to PD (4 month PFS)	Frontoparietal EMP (3 cm)
December 2022	4th	Murine B-cell maturation antigen chimeric antigen receptor T-cell therapy	PD	Left mandibular EMP
March-August 2023	5th	Daratumumab, carfilzomib, pomalidomide and dexamethasone + radiotherapy	PR to PD (5 month PFS)	Persistent EMP
August-December 2023	6th	Daratumumab, carfilzomib, selinexor and dexamethasone	Intolerance (Grade 4 cytopenia)	
December 2023	7th	Equecabtagene autoleucel	Cytokine release syndrome (Grade 1)	
January 2024			VGPR	EMP reduction
March 2024-ongoing	Maintenance	Pomalidomide		
Last follow up (19 months)			sCR	Ongoing response

EMP, extramedullary plasmacytoma; VGPR, very good partial response; sCR, stringent complete response; SD, stable disease; PR, partial response; PD, progressive disease; PFS, progression-free survival.

rather than mutually exclusive, and the movement of CAR-T therapy to earlier lines is supported by KarMMa-3 and CARTITUDE-4 (23,24).

Maintenance therapy remains a central strategy in MM, but its role after CAR-T therapy is still being defined. In the present patient, pomalidomide was chosen as maintenance therapy because residual EMP persisted radiographically, the disease had previously shown sensitivity to pomalidomide-containing regimens, and IMiDs may enhance CAR-T function and persistence (12-15). Recent clinical data have suggested that long-term pomalidomide treatment after BCMA CAR-T therapy is feasible and may deepen or maintain remission, particularly in patients with residual disease or extramedullary involvement (15). Mechanistically, lenalidomide-related studies have shown that IMiDs can enhance anti-BCMA CAR-T activity by promoting T-cell activation/function (13,14), whereas a pomalidomide-specific BCMA CAR-T study reported reduced CAR-T cell apoptosis and enhanced antigen-specific cytotoxicity, supporting improved CAR-T survival/persistence and antimyeloma activity (15). Accordingly, maintenance therapy may have contributed to the durability of response in the present case, although the independent effect of pomalidomide cannot be separated from that of CAR-T itself.

In conclusion, fully humanized BCMA CAR-T therapy provided durable clinical benefit in a heavily pretreated patient with RRMM, EMP, prior non-humanized BCMA CAR-T therapy failure and newly acquired high-risk cytogenetic abnormalities. The present case supports the feasibility of fully humanized BCMA CAR-T as a salvage option after prior BCMA exposure and suggests that maintenance therapy may help sustain response in selected patients. The main limitations of the present study are that this is a single case, the patient also received prior radiotherapy and subsequent pomalidomide maintenance and no serial analyses of BCMA expression, anti-CAR immunity or T-cell phenotype were available; therefore, the precise mechanism of response cannot be established.

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Availability of data and materials

The data generated in the present study are included in the figures and tables of this article.

Authors' contributions

RZ and LC conceptualized the study and managed clinical care. RZ collected and analyzed patient data and interpreted laboratory/imaging results. RZ, JL and LZ supervised CAR-T cell therapy administration, monitored adverse events and contributed to clinical data validation. RZ contributed to the original draft. JZ contributed to the conception and design of the study, interpretation of the clinical data, critical revision of the manuscript for important intellectual content, and provided senior oversight. RZ and LC confirm the authenticity of all the raw data. All authors read and approved the final version of the manuscript.

Ethics approval and consent to participate

The present study was approved by the Ethics Committee of the Peking Union Medical College Hospital (approval no. I-24PJ1292).

Patient consent for publication

Written informed consent was obtained from the patient for publication of the case details and accompanying images.

Competing interests

The authors declare that they have no competing interests.

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