

Talquetamab in Relapsed/Refractory Multiple Myeloma: A First Gulf Experience and Case Series Following BCMA CAR-T Therapy and Relapsed Refractory Extramedullary Disease

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Case Report

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Statements and Declarations

1. Author Contributions

The manuscript has been read and approved by all the authors. All authors meet the authorship requirements outlined in the journals' document and each author affirms that the manuscript represents honest work.

2. Funding statement

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3. Conflict of Interest

The authors declare that there is no conflict of interest in this study.

4. Ethics and Informed Consent

This study was conducted in accordance with the principles of the Declaration of Helsinki. As a retrospective case series describing standard-of-care clinical management, formal ethics committee approval was not required; however, institutional review board oversight was maintained in accordance with institutional policy. Written informed consent for publication of clinical details and imaging was obtained from both patients prior to submission.

5. Data availability statement

Data sharing is not applicable to this article as no datasets were generated or analyzed during this study.

6. Declaration of generative AI use

Generative AI tools were used during the preparation of this manuscript to assist with language editing, structural revision, and word count optimization. All clinical content, data, interpretations, and conclusions were generated by the authors. The authors reviewed and took full responsibility for the final submitted manuscript.

Abstract (220 words)

Relapsed or refractory multiple myeloma (RRMM) following BCMA-directed cellular therapy and aggressive extramedullary disease represent high-risk clinical scenarios with limited effective treatment options. Talquetamab, a first-in-class GPRC5D×CD3 bispecific T-cell redirecting antibody, offers a mechanistically distinct therapeutic approach targeting an antigen preserved after BCMA-directed therapy. We report two cases of heavily pretreated relapsed or refractory multiple myeloma treated with talquetamab in the Gulf Cooperation Council (GCC) region. In the first case, a 70-year-old male with IgG lambda myeloma who relapsed following multiple prior lines including BCMA-directed CAR-T cell therapy achieved a sustained and progressively deepening biochemical response across six cycles, with no cytokine release syndrome or neurotoxicity and manageable on-target adverse effects including dysgeusia, xerosis, and nail changes. In the second case, a 58-year-old male with IgG kappa myeloma complicated by acute spinal cord compression and aggressive extramedullary disease refractory to three prior regimens achieved complete metabolic resolution of all FDG-avid lesions after one cycle of talquetamab, enabling successful bridging to CAR-T cell therapy. To our knowledge, this represents the first documented clinical experience with talquetamab in the Gulf Cooperation Council region. These cases demonstrate that GPRC5D-directed bispecific antibody therapy is clinically active and well tolerated in heavily pretreated myeloma, supporting its role as both a salvage strategy following CAR-T cell therapy failure and an effective bridging approach in rapidly progressive extramedullary disease.

Keywords (5x)

Multiple myeloma, Talquetamab, GPRC5D, Bispecific antibody, CAR-T cell therapy

Background

Multiple myeloma (MM) is the second most common hematologic malignancy worldwide, accounting for approximately 1% of all cancers [1,2]. Its burden has risen in Gulf Cooperation Council (GCC) countries over the past decade [3,4]. Despite therapeutic advances—including proteasome inhibitors (PIs), immunomodulatory drugs (IMiDs), anti-CD38 monoclonal antibodies, and autologous stem cell transplantation—many patients develop triple-class refractory disease, which carries a median overall survival of less than one year [5,6].

B-cell maturation antigen (BCMA)-directed therapies, including bispecific antibodies and chimeric antigen receptor T-cell (CAR-T) therapies, have further expanded options for heavily pretreated MM. However, relapse following BCMA-directed therapy is increasingly encountered, driven by immune escape and treatment resistance, necessitating alternative therapeutic targets [7].

G protein-coupled receptor class C group 5 member D (GPRC5D) is selectively expressed on malignant plasma cells and preserved following BCMA-directed therapy, making it an attractive therapeutic target. Talquetamab, a first-in-class GPRC5D×CD3 bispecific antibody, received FDA approval in 2023 for adults with RRMM after at least four prior lines of therapy [8–10]. In patients with aggressive extramedullary disease or those requiring bridging to CAR-T therapy, achieving rapid disease control remains a critical challenge [11,12].

Here, we report two cases of RRMM treated with talquetamab in the Gulf region—the first documented clinical experience with this agent in the GCC. The first case describes talquetamab use following relapse after BCMA-directed CAR-T therapy; the second illustrates its role as bridging therapy in aggressive extramedullary disease prior to CAR-T infusion.

Case presentation #1

A 70-year-old male was diagnosed with IgG lambda multiple myeloma in 2015. He received the RAD regimen (lenalidomide, doxorubicin, dexamethasone) followed by lenalidomide maintenance and autologous stem cell transplantation (ASCT) in 2017. Disease progression in 2018, with identified ixazomib resistance, prompted transition to daratumumab, pomalidomide, and dexamethasone (DPd).

Between 2019 and 2021, he received multiple salvage regimens with only transient responses. In July 2021, further relapse was treated with cyclophosphamide, bortezomib, and dexamethasone (CyBorD). He subsequently underwent BCMA-directed CAR-T cell therapy (idecabtagene vicleucel) in February 2022, achieving remission for approximately three years.

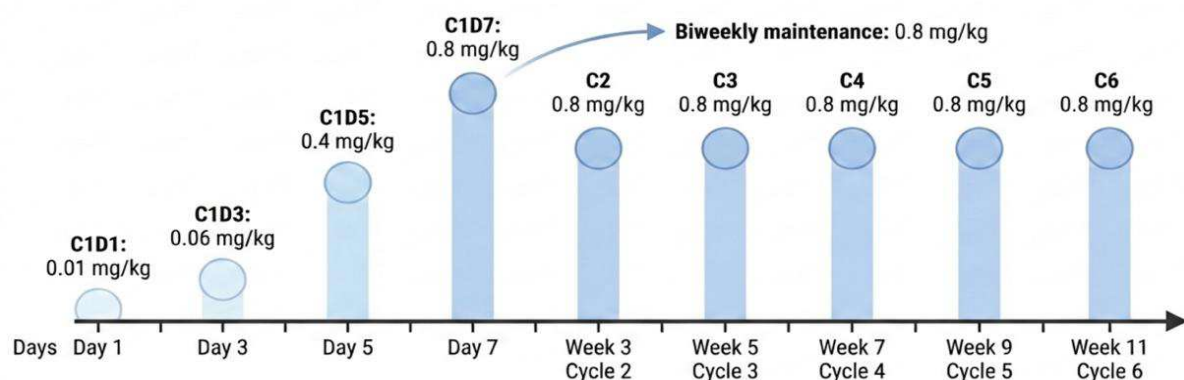
In July 2025, he presented with biochemical relapse and intermittent fevers. Laboratory evaluation demonstrated an M-spike of 3.3g/dL, IgG of 5,648mg/dL, and elevated lambda free light chains (λ 338.40mg/L; κ/λ ratio 0.05), with urine studies confirming free lambda chain excretion. β 2-microglobulin was elevated at 3.93mg/L with mild anemia and preserved renal function. Positron emission tomography/computed tomography (PET/CT) revealed diffuse mixed lytic and sclerotic osseous involvement with multiple compression deformities. Bridging therapy with cyclophosphamide and high-dose dexamethasone provided temporary stabilization, and talquetamab was initiated in September 2025 following progression through multiple prior lines including CAR-T.

Baseline assessment confirmed ongoing clonal activity. Cardiac evaluation demonstrated preserved biventricular systolic function (LVEF $60\pm 5\%$, normal strain parameters) with grade I diastolic dysfunction. Hepatitis B core antibody positivity was identified and entecavir prophylaxis was initiated.

Talquetamab was administered using an accelerated inpatient step-up dosing protocol to monitor for cytokine release syndrome (CRS) and immune effector cell–associated neurotoxicity syndrome (ICANS), with daily immune effector cell encephalopathy (ICE) scoring throughout. Step-up dosing was delivered subcutaneously on Days 1, 3, 5, and 7 at 0.01 mg/kg, 0.06 mg/kg, 0.4 mg/kg, and 0.8 mg/kg, respectively. All doses were well tolerated without clinical or biochemical evidence of CRS or ICANS, and ICE scores remained stable at 7–8/10, consistent with baseline cognitive function.

Following the full dose on Day 7, the patient was observed for an additional 48 hours without evidence of delayed toxicity and discharged on day 9. He transitioned to biweekly maintenance dosing at 0.8mg/kg per the recommended regimen, with cycles 2 through 6 administered as scheduled without dose reductions, delays, or interruptions [13] (**Figure 1**).

Figure 1 – Talquetamab treatment cycles and dosages.



Follow-up and Treatment-Emergent Adverse Effects

The patient demonstrated excellent tolerability throughout treatment, with no episodes of CRS, ICANS, or dose-limiting toxicities. All doses were administered as scheduled without modification, delay, or interruption.

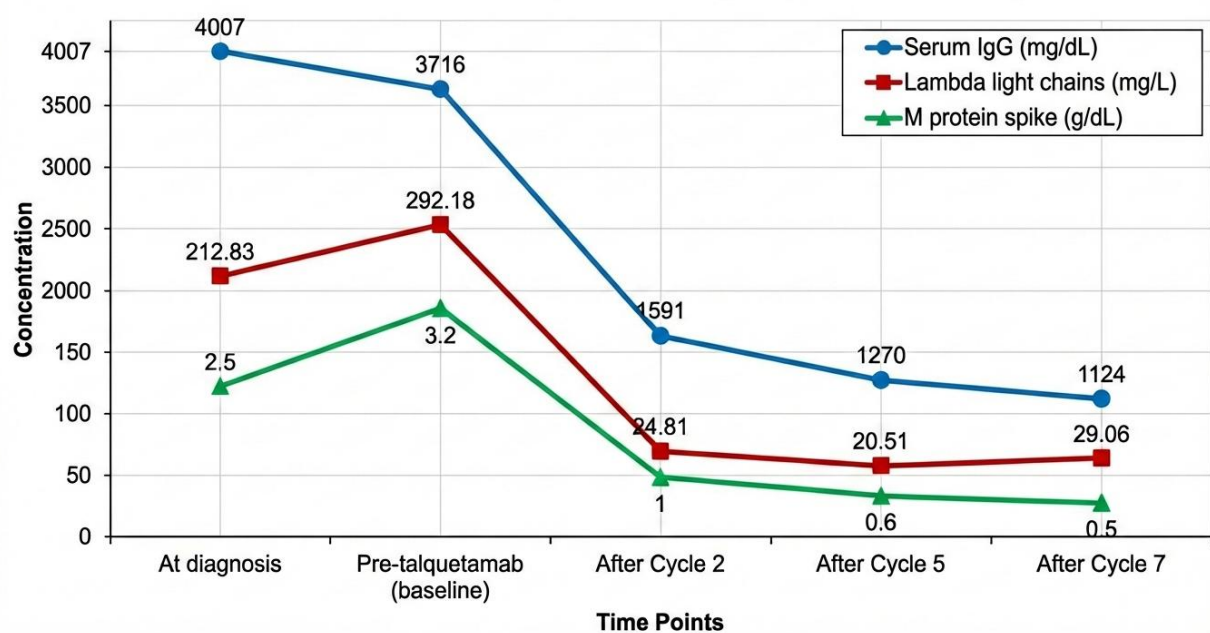
Adverse effects were consistent with the known on-target off-tumor toxicity profile of GPRC5D-directed therapy and were managed conservatively. Xerosis and localized cutaneous irritation were controlled with ceramide-based moisturizers and petrolatum-based emollients, with no secondary infection. Dysgeusia developed early in the treatment course and improved with zinc sulfate supplementation, preserving oral intake. Onychodystrophy was noted and managed conservatively without medical intervention. Insomnia was treated with low-dose quetiapine and anorexia with mirtazapine, resulting in improved sleep quality and stabilization of nutritional intake.

Supportive care included acyclovir prophylaxis against herpes simplex and varicella-zoster virus, monthly inhaled pentamidine for *Pneumocystis jirovecii* prophylaxis, allopurinol for tumor lysis prevention during early treatment, and pantoprazole for gastrointestinal protection. Serum immunoglobulin levels were monitored serially; intravenous immunoglobulin replacement was not required. Chronic comorbidities were managed with metformin and atorvastatin, and bone health was supported with denosumab. These measures collectively enabled uninterrupted biweekly dosing, reinforcing the favorable tolerability profile of talquetamab in a heavily pretreated patient.

Response assessment

Treatment response was assessed per International Myeloma Working Group (IMWG) criteria, incorporating serial measurements of serum and urine M-protein, free light chains, quantitative immunoglobulins, β 2-microglobulin, lactate dehydrogenase, and standard laboratory parameters, with PET/CT performed as clinically indicated [14]. Serial assessments demonstrated a sustained and progressively deepening biochemical response. By cycle 2, M-protein fell from 3.2g/dL to 1.0g/dL, with normalization of free light chains (κ/λ ratio 0.95) and IgG (1,591mg/dL). By cycle 5, M-protein further declined to 0.6g/dL (IgG 1,270mg/dL; κ/λ ratio 1.11), with β 2-microglobulin near normal at 2.91mg/L. Serum immunofixation continued to demonstrate a diminishing IgG lambda monoclonal band throughout. At cycle 6, the patient remained clinically stable with ongoing disease control. Following multidisciplinary review, dosing was extended to every four weeks, supported by emerging MonumenTAL-1 dose-spacing data demonstrating maintained efficacy at reduced dosing intensity [15] (**Figure 2**).

Figure 2 – Serial biochemical response during talquetamab therapy.



Patient perspective

Patient reported subjective improvement in energy levels and overall functional status during the course of treatment and expressed satisfaction with the tolerability of therapy relative to prior treatment lines.

Case presentation #2

A 58-year-old male with hypertension presented in March 2025 with progressive lower back pain and rapidly evolving bilateral lower extremity weakness, culminating in acute paraplegia and urinary retention. Magnetic resonance imaging (MRI) of the thoracic spine revealed a large extradural lytic lesion arising from the left T8 transverse process, extending into the spinal canal at T7–T9 (6.3×3×1.2cm) with severe cord compression. Urgent decompressive laminectomy and tumor resection was performed; histopathology confirmed a plasma cell neoplasm consistent with plasmacytoma per WHO Classification of Hematolymphoid Tumours, 5th edition (2022) [16].

Staging PET/CT demonstrated extensive FDG-avid multifocal lytic bone lesions, including a large destructive left iliac soft tissue mass (~8cm; **Figure 3A**). Serum IgG was elevated at 2,020mg/dL with suppression of IgA and IgM. Bone marrow biopsy confirmed IgG kappa multiple myeloma, with 15% kappa light chain–restricted plasma cells.

Systemic therapy was initiated with cyclophosphamide, bortezomib, and dexamethasone, followed by transition to lenalidomide, bortezomib, and dexamethasone (RVd), discontinued after one completed cycle due to rapid progression. Repeat PET/CT demonstrated new hepatic lesions and recurrent epidural soft tissue at T8; thoracic spine radiotherapy resulted in neurological improvement. Treatment was escalated to daratumumab, cyclophosphamide,

bortezomib, and dexamethasone (Dara-CyBorD) for four cycles (June–September 2025), with concurrent denosumab and left pelvic radiotherapy. Follow-up imaging showed a mixed response with hepatic and new osseous progression, consistent with evolving extramedullary disease (**Figure 3B**).

Therapy was further intensified with daratumumab, carfilzomib, pomalidomide, and dexamethasone (Dara-KPd). A left soft palate lesion on examination raised concern for additional extramedullary involvement (**Image 1**). Liver biopsy confirmed CD138-positive, kappa-restricted plasma cell infiltration with diffuse CD56 expression. After two cycles, interim PET/CT demonstrated mixed metabolic response with progression involving the ribs, lumbar vertebrae, and proximal femur (**Figure 3C**). Given relapsed/refractory disease with extensive extramedullary involvement, the patient was planned for BCMA-directed CAR-T cell therapy with ciltacabtagene autoleucel (cilta-cel); as this was unavailable locally, arrangements were made for treatment abroad. Talquetamab was initiated as bridging therapy to maintain disease control pending CAR-T infusion

Figure 3 – PET/CT pre and post talquetamab therapy

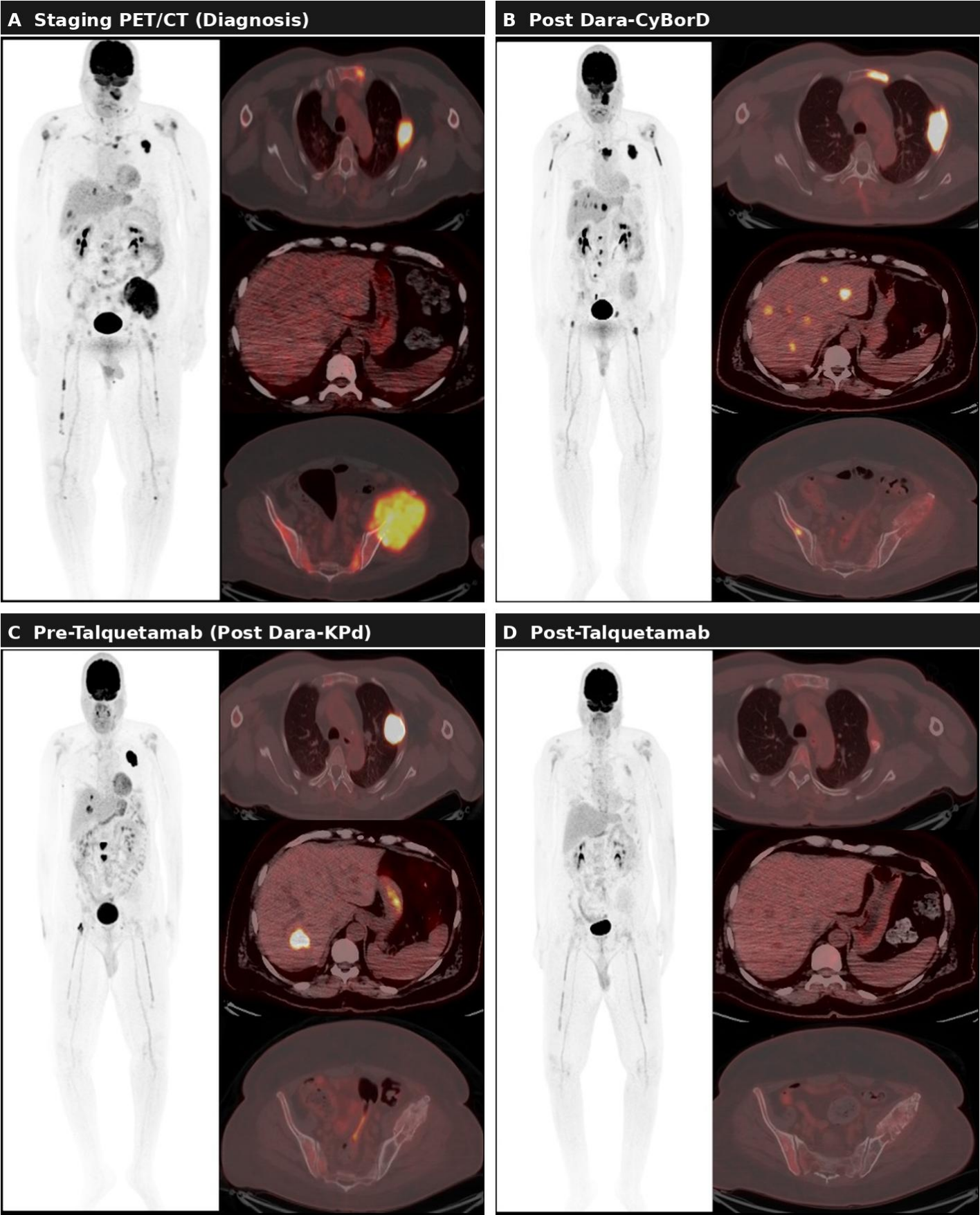


Image 1 – Left soft palate lesion concerning for extramedullary involvement.



Outcome and Follow-up

Talquetamab step-up dosing (days 1, 3, 5, and 7) was administered with prophylactic tocilizumab and completed without CRS or ICANS. The patient developed dysgeusia and a diffuse erythematous rash, managed with antihistamines and a short course of systemic corticosteroids. Transient neutropenia required granulocyte colony-stimulating factor (G-CSF) support.

Following one cycle of talquetamab and concurrent radiotherapy, repeat PET/CT demonstrated complete metabolic resolution of all previously FDG-avid lesions with no new disease sites, consistent with a marked radiographic and metabolic response (**Figure 3D**). The patient subsequently travelled abroad to undergo BCMA-directed CAR-T cell therapy, with talquetamab having served as effective bridging therapy.

Patient perspective

The patient reported marked improvement in lower extremity strength and pain control following talquetamab initiation and expressed willingness to proceed with subsequent CAR-T cell therapy.

Discussion and literature review

Relapsed/refractory multiple myeloma remains biologically complex and therapeutically challenging, particularly in patients who have progressed through proteasome inhibitors, immunomodulatory drugs, anti-CD38 monoclonal antibodies, and BCMA-directed therapies [7,8]. With successive lines of treatment, clonal evolution, immune escape, and cumulative toxicities contribute to progressively shorter remissions and shrinking therapeutic options [1,2]. Relapse following BCMA-directed CAR-T therapy, as illustrated in our first case, represents a particularly high-risk scenario with substantial unmet need, while aggressive extramedullary disease, as in the second case, defines an additional subgroup with poor prognosis, rapid progression, and limited effective salvage strategies.

Several real-world studies and case series have reported on talquetamab outside the clinical trial setting, supporting its generalizability. A multi-institutional retrospective analysis of 114 heavily pretreated RRMM patients demonstrated an overall response rate of 73% with manageable toxicity [17]. A German multicentre real-world analysis of 138 patients confirmed an overall response rate of 65% in a population enriched for high-risk features including extramedullary disease [18]. Of particular relevance to our second case, Dhakal et al. reported that among 134 patients receiving talquetamab as bridging therapy prior to BCMA-directed CAR-T cell therapy, 119 (88%) achieved rapid disease control and proceeded to CAR-T infusion — directly paralleling the outcome observed in our second case [19].

Talquetamab offers a mechanistically distinct approach by targeting GPRC5D, an antigen selectively expressed on malignant plasma cells and preserved following BCMA-directed therapy, making it an attractive option in both clinical contexts [9–11]. In the phase 1/2 MonumenTAL-1 program, subcutaneous talquetamab demonstrated overall response rates of 69–74% in heavily pretreated RRMM, with maintained activity in patients previously exposed

to T-cell–redirecting therapies, including BCMA-directed CAR-T and bispecific antibodies [8,20]. This was reflected in our cases: a sustained and progressively deepening biochemical response following post–CAR-T relapse in the first, and rapid complete metabolic response when used as bridging therapy in aggressive extramedullary disease in the second.

The tolerability profile observed across both cases is consistent with clinical trial and real-world data. Talquetamab's safety profile is characterized by expected hematologic toxicities alongside on-target, off-tumor effects arising from GPRC5D expression in keratinized tissues and taste receptors, including dysgeusia, xerosis, xerostomia, and nail changes; most events are low grade and infrequently treatment limiting [13,15,20,21]. In our first case, dermatologic and mucosal toxicities were mild and managed conservatively with emollients and zinc supplementation. In the second case, dysgeusia and an erythematous rash were similarly manageable with antihistamines and a brief course of corticosteroids, with no treatment discontinuation required in either patient. Real-world evidence further supports the feasibility of proactive, anticipatory supportive care strategies—including structured skin care regimens, nutritional interventions for dysgeusia, and close monitoring—as key enablers of treatment continuity [21].

The selection of talquetamab in these cases reflected a deliberate consideration of efficacy and tolerability relative to other available options. BCMA-directed bispecific antibodies, while effective, carry higher rates of profound hypogammaglobulinemia and clinically significant infections, which are particularly concerning in heavily pretreated patients with pre-existing immune dysfunction, as in our first case following CAR-T therapy [13,15,20]. Belantamab mafodotin was considered less suitable given its characteristic keratopathy, requiring frequent ophthalmologic monitoring that may limit feasibility in routine clinical practice [9]. By contrast, talquetamab's mucocutaneous toxicity profile is predictable and manageable, making

it a pragmatic choice both as salvage therapy following CAR-T failure and as bridging therapy in rapidly progressive extramedullary disease.

Careful monitoring during step-up dosing remains essential to detect CRS and ICANS. Both cases employed an accelerated inpatient step-up protocol (days 1, 3, 5, and 7), consistent with FDA and EMA label recommendations, allowing safe administration without immune-mediated complications [10,13]. Prophylactic tocilizumab was administered in the second case; emerging evidence from clinical trial analyses and real-world studies supports pre-emptive IL-6 blockade in reducing CRS incidence with bispecific antibodies without apparent efficacy loss and may facilitate a transition to outpatient step-up dosing in carefully selected patients [22,23]. However, its protective effect against ICANS remains inconsistent, and continued vigilance with standardized monitoring protocols is warranted.

Together, these cases demonstrate the versatility of GPRC5D-directed therapy across distinct high-risk RRMM scenarios and support the role of talquetamab as both an effective salvage and bridging therapeutic strategy.

Conclusion

These cases illustrate the clinical utility of talquetamab across two high-risk relapsed or refractory multiple myeloma scenarios encountered in routine practice. In the first case, GPRC5D-directed therapy produced a sustained biochemical response with excellent tolerability following relapse after BCMA-directed CAR-T cell therapy; a setting with very limited salvage options. In the second, a single cycle achieved complete metabolic remission in aggressive extramedullary disease, enabling successful bridging to CAR-T cell therapy. The predictable on-target toxicity profile, including dysgeusia, xerosis, and nail changes, was effectively managed with proactive supportive care in both patients, enabling uninterrupted

treatment delivery without dose modification. The use of an accelerated inpatient step-up protocol with prophylactic tocilizumab provides a practical framework that may inform institutional administration pathways. As myeloma therapy evolves toward sequential immunotherapy strategies, non-BCMA-directed agents such as talquetamab will be essential to address post-BCMA relapse and serve as effective bridging strategies during the coordination period preceding cellular therapy. To our knowledge, this represents the first documented clinical experience with talquetamab in the Gulf Cooperation Council region, contributing real-world evidence from an underrepresented setting and supporting the broader adoption of GPRC5D-directed therapy in clinical practice.

References

1. van de Donk NWCJ, Pawlyn C, Yong KL. Multiple myeloma. *Lancet* 2021;397:410–27.
2. Malard F, Neri P, Bahlis NJ, et al. Multiple myeloma. *Nat Rev Dis Primers* 2024;10:45.
3. Mafra A, Laversanne M, Marcos-Gragera R, et al. The global multiple myeloma incidence and mortality burden in 2022 and predictions for 2045. *J Natl Cancer Inst* 2025;117:907–14.
4. Ibrahim A, Chamseddine N, El-Cheikh J, et al. Management of multiple myeloma in the Middle East: unmet needs, challenges and perspective. *Clin Hematol Int* 2022;4:127–32.
5. Palumbo A, Anderson K. Multiple myeloma. *N Engl J Med* 2011;364:1046–60.
6. Costa LJ, Hungria V, Mohty M, Mateos MV. How I treat triple-class refractory multiple myeloma. *Br J Haematol* 2022;198:244–56.
7. Munshi NC, Anderson LD Jr, Shah N, et al. Idecabtagene vicleucel in relapsed and refractory multiple myeloma. *N Engl J Med* 2021;384:705–16.
8. Chari A, Minnema MC, Berdeja JG, et al. Talquetamab, a T-cell–redirecting GPRC5D bispecific antibody for multiple myeloma. *N Engl J Med* 2022;387:2232–44.

9. Rodriguez-Otero P, van de Donk NWCJ, Pillarisetti K, et al. GPRC5D as a novel target for the treatment of multiple myeloma: a narrative review. *Blood Cancer J* 2024;14:24.
10. US Food and Drug Administration. FDA approves talquetamab-tgvs for relapsed or refractory multiple myeloma. 2023.
11. Rajkumar SV, Harousseau JL, Durie B, et al. Consensus recommendations for the uniform reporting of clinical trials: report of the International Myeloma Workshop Consensus Panel 1. *Blood* 2011;117:4691–5.
12. Bladé J, Beksac M, Caers J, et al. Extramedullary disease in multiple myeloma: a systematic literature review. *Blood Cancer J* 2022;12:45.
13. Gong J, Zhou J, Yuan D, et al. Recommended phase II doses of talquetamab in patients with relapsed/refractory multiple myeloma from MonumenTAL-1: clinical pharmacology results. *Clin Pharmacol Ther* 2025;118:954–66.
14. Kumar S, Paiva B, Anderson KC, et al. International Myeloma Working Group consensus criteria for response and minimal residual disease assessment in multiple myeloma. *Lancet Oncol* 2016;17:e328–46.
15. Chari A, Oriol A, Krishnan A, et al. Efficacy and safety of less frequent or lower-intensity dosing of talquetamab in relapsed/refractory multiple myeloma: results from the phase 1/2 MonumenTAL-1 study. *Blood* 2023;142:1010.
16. Khoury JD, Solary E, Abla O, et al. The 5th edition of the World Health Organization Classification of Haematolymphoid Tumours: myeloid and histiocytic/dendritic neoplasms. *Leukemia* 2022;36:1703–19.
17. Multicenter Real-World Study Group. Talquetamab in relapsed refractory multiple myeloma: multi-institutional real-world study. *Blood Cancer J*. 2025. <https://doi.org/10.1038/s41408-025-01386-7>

18. Frenking J, et al. A German multicenter real-world analysis of talquetamab in 138 patients with relapsed/refractory multiple myeloma. *HemaSphere*. 2025. <https://doi.org/10.1002/hem3.70114>
19. Dhakal B, Akhtar OS, Cowan AJ, et al. Sequential targeting in multiple myeloma: talquetamab, a GPRC5D bispecific antibody, as a bridge to BCMA CAR-T therapy. *Blood*. 2025;146(17):2063-2072. <https://doi.org/10.1182/blood.2025029773>
20. Chari A, Touzeau C, Schinke C, et al. Safety and activity of talquetamab in patients with relapsed or refractory multiple myeloma (MonumenTAL-1): a multicentre, open-label, phase 1–2 study. *Lancet Haematol* 2025;12:e269–81.
21. Labanca C, Martino EA, Vigna E, et al. Talquetamab in multiple myeloma: efficacy, safety, and future directions. *Eur J Haematol* 2025;114:386–99.
22. Korst CLBM, Groen K, Bosman PWC, et al. Prophylactic tocilizumab reduces the incidence of cytokine release syndrome in relapsed/refractory myeloma patients treated with teclistamab: implications for outpatient step-up dosing. *HemaSphere* 2024;8:e132.
23. Kowalski A, Lykon J, Diamond B, et al. Tocilizumab prophylaxis for patients with multiple myeloma treated with bispecific antibodies. *Blood Adv* 2025