Bridging Innovation in Multiple Myeloma Care: A Review of Novel Therapies

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Authors' disclosures of conflicts of interest are found at the end of this article.

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https://doi.org/10.6004/jadpro.2026.17.7.25

Abstract

Multiple myeloma (MM) is a plasma cell malignancy that impairs immune regulation, leading to increased infection risk and disease-related complications. While advances in therapy have improved survival, MM remains incurable, with most patients eventually relapsing. T-cell–based immunotherapies, such as chimeric antigen receptor (CAR) T-cell therapy and bispecific antibodies (bsAbs), have emerged as powerful treatment options for relapsed/refractory MM (RRMM). CAR T-cell therapy involves complex manufacturing and requires administration in specialized centers due to the risk of severe toxicities such as cytokine release syndrome (CRS) and immune effector cell–associated neurotoxicity syndrome (ICANS). These therapies demand trained personnel, hemodynamic monitoring, and access to intensive care. Bispecific antibodies, in contrast, are off-the-shelf agents that redirect T cells to malignant plasma cells and offer more flexible, outpatient-friendly administration. While bsAbs can also cause CRS and neurotoxicity, these events are typically milder and more manageable. Despite their promise, bsAb therapies still require infrastructure for toxicity management, monitoring of prolonged cytopenias, and infection prevention. Prophylactic strategies, such as early tocilizumab use, may facilitate safer outpatient delivery. Expanding access to bsAbs, especially in community settings, requires 24/7 clinical support, caregiver engagement, and training for local providers. Advanced practice providers, with their advanced clinical skills and holistic patient care approach, are well-positioned to lead in patient education, early identification, and care coordination. Their involvement is essential to promoting equitable access to novel therapies and improving outcomes. Broader implementation of bsAbs will require investment in infrastructure, education, and interprofessional collaboration to ensure safe, equitable, and effective MM treatment.

J Adv Pract Oncol 2026

Multiple myeloma (MM) is a hematologic malignancy characterized by the clonal proliferation and accumulation of malignant plasma cells in the bone marrow, leading to complications such as anemia, osteolytic bone lesions, renal impairment, and hypercalcemia (Cowan et al., 2022). Multiple myeloma accounts for approximately 10% of all bone marrow-derived cancers (Rajkumar, 2019). Its cause is unknown, although several studies have evaluated potential risk factors for this disease (Kumar et al., 2017a). Despite significant advances in treatment over the past decades resulting in improved response rates and prolonged survival, MM remains an incurable disease (Sammartano et al., 2023; Kakiuchi-Kiyota et al., 2022), with most patients ultimately experiencing relapse at some point in the disease process (Rodríguez-Otero et al., 2021). Historically, 10% to 20% of patients succumbed to the disease within 2 to 3 years of diagnosis (Sonneveld et al., 2016). However, the introduction of novel therapeutic agents has markedly improved patient outcomes, increasing the 5-year overall survival (OS) rate from approximately 30% to 60% (Ozaki et al., 2014). More recently, immunotherapeutic strategies, including chimeric antigen receptor (CAR) T-cell therapies and bispecific antibodies (bsAbs), have emerged as promising treatment modalities, with the potential to significantly reshape the treatment landscape for patients with MM (Engelhardt et al., 2024). Together, these immunotherapies represent a significant advancement in the management of relapsed/refractory multiple myeloma (RRMM), offering promising alternatives for patients who have exhausted conventional treatment options.

Standard treatment options for MM include proteasome inhibitors (PIs), anti-CD38 monoclonal antibodies, and immunomodulatory drugs (IMiDs), all of which have significantly advanced the treatment of the disease (Moreau et al., 2022). These agents have contributed to a substantial improvement in patient outcomes, with median survival increasing from approximately 3 to 4 years in the 1990s to around 8 years in the current era (Podar & Leleu, 2021). In addition, high-dose chemotherapy followed by autologous stem cell transplant (ASCT) remains a cornerstone of frontline therapy for transplant-eligible patients and is widely regarded as the standard of care in this population (Al Hamed et al., 2019; Cavo et al., 2020; Ozaki et al., 2014). Achieving minimal residual disease (MRD) negativity following ASCT has been strongly associated with prolonged progression-free survival (PFS) and OS, underscoring its importance as a prognostic indicator and treatment goal (de Tute et al., 2022).

However, in the context of disease relapse and/or refractoriness, clinical outcomes tend to be poor due to decreased performance status and progressively diminished response duration with successive lines of treatment (Kumar et al., 2017b). Patients who are triple-class refractory (to IMiDs, PIs, and monoclonal antibodies) and experience relapse after standard treatment typically have a poor prognosis, with a response rate of about 30% and a median OS of 12.4 months (Mateos et al., 2022). Furthermore, once relapse occurs, the management of MM becomes increasingly complex and challenging due to its biological heterogeneity (Bhatt et al., 2023). The treatment options for RRMM depend on various factors, including therapy-related aspects (responses, toxicity, refractoriness to other therapies, eligibility for autologous stem cell transplant), disease characteristics (genetic variations, extent of prior remission, presence of extramedullary disease, elevated tumor burden, speed of disease progression, and end-organ dysfunction), and patient factors (performance status, comorbidities, treatment preferences, adherence to the treatment plan, access to specialized centers, and out-of-pocket costs; Podar & Leleu, 2021).

The development and clinical implementation of novel T-cell–based immunotherapies, such as CAR T-cell therapy and bsAbs, have changed the treatment landscape for patients with RRMM. CAR T-cell therapy involves harvesting a patient's own T cells, genetically engineering them ex vivo to express receptors that specifically recognize antigens on malignant plasma cells, and reinfusing them into the patient. Once administered, these modified T cells undergo an in vivo expansion phase, the duration of which varies depending on the product used. This approach has shown remarkable efficacy, particularly in targeting B-cell maturation antigen (BCMA), a protein highly expressed on MM cells (Mohyuddin et al., 2021). The

most common and serious adverse events include cytokine release syndrome (CRS) and immune effector cell–associated neurotoxicity syndrome (ICANS). Early recognition and prompt management of these toxicities are essential to ensure patient safety and optimize treatment outcomes.

In contrast to CAR T-cell therapy, bsAbs offer an off-the-shelf immunotherapeutic approach by simultaneously binding to CD3 on T cells and a tumor-associated antigen on myeloma cells, thereby redirecting and activating T cells to mediate targeted cytotoxicity (Betts & van der Graaf, 2020). Bispecific antibodies are specifically engineered to recognize two distinct epitopes or antigens, typically CD3 on T cells and a surface antigen on malignant plasma cells (Wang et al., 2019), enabling the formation of an immunological synapse that triggers T-cell activation (Omer et al., 2023). A notable subgroup, bispecific T-cell engagers (BiTEs), is designed to efficiently facilitate this interaction, enhancing the T-cell–mediated immune response against cancer (Pancholi, 2023). In addition to promoting cytotoxicity, bsAbs have also been shown to induce T-cell activation and proliferation, further contributing to their antitumor efficacy (Cho et al., 2022).

Despite notable advancements in the treatment of RRMM, numerous barriers continue to limit patient access to these innovative therapies, even among those who are clinically eligible. Key challenges include geographic distance from specialized cancer centers, lack of reliable caregiving support, and substantial out-of-pocket expenses. Additionally, limited knowledge or understanding of CAR T-cell therapy among health-care providers in community settings can result in delayed referrals, while some institutions may lack the necessary infrastructure and trained personnel to administer and manage these advanced therapies. Consequently, both patients and clinicians may remain unaware of emerging treatment options or be unable to navigate the complex logistical, financial, and social requirements associated with care. These systemic barriers contribute to significant health disparities and suboptimal patient outcomes, specifically as they relate to RRMM.

This review aims to explore the future applications of novel therapeutic agents in the management of RRMM. It underscores the critical importance of early identification and timely referral of eligible patients, along with the need to educate health-care providers on the expanding range of treatment options and the management of associated toxicities. Furthermore, the review highlights the necessity of establishing appropriate infrastructure and ensuring the availability of trained personnel to safely deliver these advanced therapies. Expanding access to care while maintaining high standards of patient safety is essential to optimizing outcomes and reducing disparities in treatment delivery.

BCMA-TARGETING AGENTS

BCMA is a transmembrane protein expressed on the surface of both normal and malignant plasma cells (Carpenter et al., 2013). It has emerged as a highly promising therapeutic target in the treatment of RRMM (Moreau et al., 2022; Mei et al., 2021; Wei et al., 2019). BCMA plays a critical role in regulating B-cell proliferation, survival, and differentiation, particularly in the progression from normal to malignant plasma cells (Cho et al., 2018). Several BCMA-targeted therapies have been developed and are currently available, including antibody-drug conjugates (ADCs), CAR T-cell therapies, and bsAbs, as illustrated in Figure 1. These approaches have demonstrated clinical efficacy and durable responses in heavily pretreated RRMM patient populations (Xu et al., 2019; Zhao et al., 2018; Raje et al., 2019).

Belantamab Mafodotin

Belantamab mafodotin-blmf (Blenrep) is a first-in-class ADC-targeting BCMA, composed of a humanized anti-BCMA monoclonal antibody linked to the cytotoxic agent monomethyl auristatin F (MMAF; Yu et al., 2020). Upon binding to BCMA on myeloma cells, the ADC is internalized, releasing MMAF intracellularly, which induces cell death by disrupting microtubule dynamics (Yu et al., 2020). In the phase II DREAMM-2 trial, belantamab mafodotin demonstrated overall response rates (ORR) of 32% and 35% in the 2.5 mg/kg and 3.4 mg/kg dosing cohorts, respectively. The median PFS was 2.8 months for the 2.5 mg/kg cohort and 3.9 months for the 3.4 mg/kg cohort (Nooka et al., 2023).

Unlike CAR T-cell therapies and bsAbs, belantamab mafodotin is not associated with CRS or

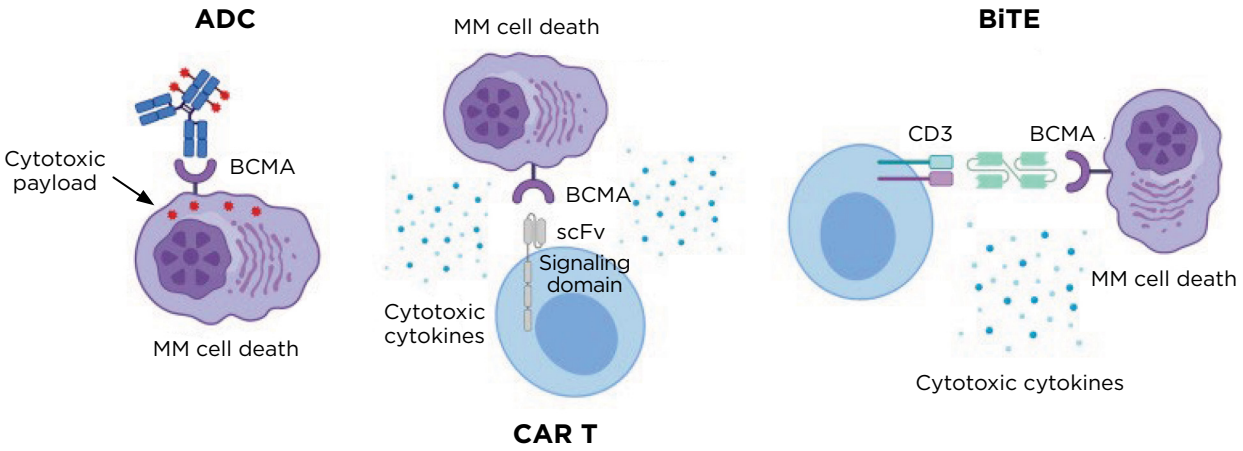

Figure 1. BCMA-targeted immunotherapies. Reproduced from Yu et al. (2020).

ICANS. However, it has a distinct safety profile characterized primarily by ocular toxicity, particularly keratopathy. Keratopathy, involving changes to the corneal epithelium, was the most common and serious adverse event, occurring in approximately 71% to 72% of patients. Grade 2 keratopathy was reported in about 25% of cases, while grade 3 occurred in 45% (GlaxoSmithKline LLC, 2025; Nooka et al., 2023; Sheikh et al., 2020). These ocular effects often led to blurred vision, dry eyes, and decreased visual acuity, necessitating regular ophthalmologic monitoring and frequently resulting in dose delays, reductions, or treatment discontinuation (Sheikh et al., 2020). Other notable adverse events included thrombocytopenia, which occurred in approximately 38% of patients, with 21% experiencing grade 3 or higher severity, and anemia, reported in 28% of patients, with 21% experiencing grade 3 or higher (Nooka et al., 2023). The overall infection rate ranged from 43% to 55%, with grade 3 or higher infections occurring in 20% and 44% of patients in the 2.5 mg/kg and 3.4 mg/kg cohorts, respectively (Nooka et al., 2023).

Despite its initial US Food and Drug Administration (FDA) approval in 2020, the confirmatory phase III DREAMM-3 trial failed to demonstrate a significant improvement in PFS compared to standard therapies, leading to the withdrawal of belantamab mafodotin from the U.S. market in 2023 (FDA, 2023a). However, on October 23, 2025, the FDA reapproved the drug for the treatment of adults with RRMM, but only as part of a combination regimen with bortezomib and dexamethasone after at least two prior lines of therapy, including a PI and an IMiD. This approval was primarily based on the results of the phase III DREAMM-7 trial, which demonstrated significant improvements in both PFS and OS compared with the standard regimen of daratumumab, bortezomib, and dexamethasone (FDA, 2025a). This combination achieved a median PFS of 36.6 months, surpassing the 13.4 months observed with standard regimens like daratumumab, bortezomib, and dexamethasone (Hungria et al., 2024).

Idecabtagene Vicleucel and Ciltacabtagene Autoleucel

The first BCMA-targeted CAR T-cell therapy approved for RRMM was idecabtagene vicleucel (ide-cel; Abecma), which received FDA approval in 2021 (Munshi et al., 2021). This was followed by the approval of ciltacabtagene autoleucel (cilta-cel; Carvykti) in 2022 for patients with RRMM who had received at least four prior lines of therapy. On April 5, 2024, the FDA expanded cilta-cel's indication to include adult patients with RRMM who had received at least one prior line of therapy and were refractory to lenalidomide. This expanded approval was based on results from the phase III CARTITUDE-4 trial, which demonstrated a significant improvement in PFS compared to standard treatment options (Alsdorf et al., 2024).

Both ide-cel and cilta-cel are engineered to target BCMA on malignant plasma cells and require preconditioning with lymphodepleting chemotherapy (LDC). The standard LDC regimen consists of cyclophosphamide 300 mg/m² IV and fludarabine 30 mg/m² IV, administered daily for 3 consecutive days (Chekol Abebe et al., 2022). This preparatory chemotherapy enhances the expansion, persistence, and efficacy of adoptively transferred CAR T cells (Turtle et al., 2016). Following a single infusion of the CAR T-cell product, patients require close monitoring in both inpatient and outpatient settings because of the risk of severe toxicities such as CRS and ICANS. These conditions can be life-threatening and necessitate prompt intervention to ensure patient safety.

Cytokine release syndrome is a potentially life-threatening systemic inflammatory response caused by the rapid and excessive release of cytokines (immune-signaling proteins) into the bloodstream. Although cytokines are essential for coordinating immune responses, their dysregulated overproduction can lead to widespread inflammation, capillary leak, organ dysfunction, and, in severe cases, multiorgan failure. Cytokine release syndrome is a well-documented complication of CAR T-cell therapies and typically presents with a constellation of symptoms, including fever, hypotension, hypoxia, myalgias, fatigue, shock, and, in severe instances, end-organ damage (Frey & Porter, 2019; Neelapu et al., 2018). In addition to CRS, another serious toxicity associated with CAR T-cell therapy is ICANS. This condition can range from mild cognitive disturbances and encephalopathy to severe neurologic complications such as aphasia, focal motor deficits, seizures, cerebral edema, and, in rare cases, death (Rubin et al., 2019; Karschnia et al., 2019). Notably, cilta-cel is also associated with non-ICANS neurotoxicities that are typically delayed in onset. These may include cranial nerve palsies (Richards et al., 2023; Patrick et al., 2023), as well as acute and chronic polyneuropathies (Ellithi et al., 2025).

When comparing the two BCMA-targeting CAR T-cell therapies, ide-cel and cilta-cel, key differences emerge in terms of both efficacy and safety profiles. In the phase II KarMMa trial, ide-cel demonstrated an ORR of 73%, with a complete response (CR) rate of approximately 33% (Munshi et al., 2021). Cytokine release syndrome occurred in 84% of patients, although grade ≥ 3 CRS was relatively uncommon at 5%. The median time to CRS onset was typically within 1 day post-infusion. Immune effector cell–associated neurotoxicity syndrome was reported in 18% of patients, with severe (grade ≥ 3) events in 3%; the median onset was around day 2 (Munshi et al., 2021). Infections were also frequent, with 70% of patients experiencing infections of any grade, and 23% developing grade ≥ 3 infections (Celgene Corporation, 2025). In contrast, cilta-cel has demonstrated superior efficacy, achieving an ORR of 97% and a stringent complete response (sCR) rate of 67% in early studies (Berdeja et al., 2021). However, its safety profile is similarly complex. Cytokine release syndrome occurred in approximately 95% of patients, with grade ≥ 3 events in 4%. Unlike ide-cel, the median time to CRS onset was later at day 7, with a median duration of 4 days (Janssen Biotech, Inc., 2025a). Immune effector cell–associated neurotoxicity syndrome was observed in 21% of patients, with severe cases in 10%, typically occurring around day 8 and lasting a median of 6 days (Chekol Abebe et al., 2022). Overall infections were reported in 59% of patients, with 23% experiencing grade ≥ 3 infections, similar to ide-cel (Janssen Biotech, Inc., 2025a).

Health-care facilities offering CAR T-cell therapy must ensure they have the necessary infrastructure and trained personnel to manage patients throughout the entire treatment process. This includes establishing specialized infusion centers equipped with advanced medical equipment such as hemodynamic monitoring systems, emergency resuscitation tools, and access to intensive care units for immediate intervention if severe toxicities occur. Additionally, facilities should have laboratory support for cytokine level testing and rapid processing of specimens. Education should focus on training health-care providers, such as physicians, advanced practice providers (APPs), nurses, and pharmacists, in the recognition and management of CAR T-cell toxicities such as CRS and ICANS, including the use of cytokine blockade therapies (e.g., tocilizumab) and corticosteroids. Staff should also be knowledgeable about patient selection criteria, eligibility assessments, and post-infusion follow-up protocols

to ensure comprehensive safety monitoring and optimal treatment outcomes.

Importantly, CAR T-cell therapy presents limitations beyond its acute toxicities. One of the most critical challenges is the issue of limited CAR T-cell expansion and persistence, which are essential for sustained antitumor activity. Inadequate expansion or early loss of CAR T cells following infusion has been identified as a major contributor to non-responsiveness and early relapses in patients with RRMM (Bao et al., 2025; Sterner & Sterner, 2021). Additionally, prolonged immunosuppression and delayed immune reconstitution remain well-recognized concerns. Beyond the intended depletion of B cells and plasma cells (an on-target, off-tumor effect), CAR T-cell therapy is frequently associated with persistent cytopenias, which may last for weeks to months post-infusion (Wudhikarn & Perales, 2022). These complications increase vulnerability to infections and further complicate recovery.

Given the complexity of CAR T-cell therapy, a multidisciplinary care team is essential. Optimal care requires close collaboration between oncologists, APPs, nurse coordinators, social workers, case managers, financial counselors, cellular therapy lab staff, and pharmacists to ensure safety, adherence to protocols, and appropriate post-treatment follow-up. Logistical challenges further complicate the patient journey. These include coordination of leukapheresis, administration of bridging therapy during manufacturing delays, and ensuring the patient's physical proximity to the infusion center. Until recently, the FDA-mandated REMS (Risk Evaluation and Mitigation Strategy) program required that patients remain within 2 hours of the treatment center and have a 24/7 caregiver for immediate support (FDA, n.d.). However, this requirement was officially lifted in June 2025, reflecting growing familiarity with the management of toxicities and efforts to broaden access to therapy (FDA, 2025b). Despite this regulatory shift, post-infusion monitoring and transitions of care remain complex. Communication between specialized cancer centers and local oncologists is not yet standardized and must be tailored based on each patient's clinical status, toxicity profile, and support network.

On the other hand, bsAbs offer advantages over CAR T-cell therapies. First, bsAbs do not require apheresis or LDC prior to their administration. Their shorter half-lives enable prompt therapy discontinuation without long-term side effects. Additionally, opportunistic infections associated with therapy and resultant immunosuppression are generally milder (Kegyes et al., 2022). Bispecific antibodies are readily available off-the-shelf for eligible patients, in contrast to CAR T-cell therapy, which requires 6 to 8 weeks for manufacturing (Narkhede et al., 2021). This waiting period can be challenging for patients in urgent need of treatment. Therefore, immediate access to bsAbs could greatly impact the patient care journey. Furthermore, T-cell redirecting bsAbs tend to exhibit lower grades of CRS and neurotoxicity compared to existing CAR T-cell therapies, making them particularly appealing for outpatient treatment (Lutfi et al., 2023). While most bsAbs necessitate hospitalization for step-up dosing, the overall duration of hospitalization is less than that required for CAR T-cell therapies. In fact, many specialized centers in the United States are now successfully delivering bsAbs entirely in the outpatient setting, provided there is close patient monitoring and the proper infrastructure in place. According to a roadmap published by experts involved in international workshops in 2024, bsAbs, including teclistamab-cqyv (Tecvayli), talquetamab-tgvs (Talvey), and elranatamab-bcmm (Elrexfio), can be safely administered in outpatient and community-based settings with clearly documented algorithms (Garfall et al., 2025). A real-world study at Mayo Clinic involving teclistamab demonstrated that 58 out of 65 patients were able to initiate step-up dosing in the outpatient setting. Patients received remote monitoring of vital signs, were seen daily during the dosing period, and only a small proportion (32%) experienced CRS requiring admission (Olivieri & Banerjee, 2025).

Teclistamab

The first bsAb approved for the treatment of RRMM was teclistamab-cqyv, a bsAb that simultaneously targets BCMA on malignant plasma cells and CD3 on T cells (Kang, 2022). This dual-targeting mechanism facilitates T-cell redirection and activation,

enabling cytotoxic T cells to recognize and eliminate myeloma cells. While this targeted immune engagement produces robust antimyeloma responses, it also carries a risk of immune-mediated toxicities, particularly CRS and ICANS, especially during early treatment cycles, necessitating close clinical monitoring. In the MajesTEC-1 clinical trial, teclistamab demonstrated an ORR of 65%, with 58% of patients achieving a very good partial response (VGPR) or better (Usmani et al., 2021). Cytokine release syndrome occurred in 70% of patients treated at the recommended phase II dose (n = 40) and in 58% of all patients (n = 157), with the majority of events classified as grade 1 or 2 (Usmani et al., 2021). Grade 3 CRS was reported in only 0.6% of cases, and no grade 4 or fatal CRS events were observed (Martin et al., 2023). The median time to CRS onset was approximately 2 days following the last step-up dose, with a median duration of 2 days (Martin et al., 2023; Janssen Biotech, Inc., 2026). Immune effector cell–associated neurotoxicity syndrome was observed in 6% of patients, all presenting with grade 1 or 2 symptoms, most commonly confusional state and dysgraphia. Onset typically occurred around 2 days post-dose, with a median duration of 3 days (Janssen Biotech, Inc., 2026). In addition to ICANS, other neurologic toxicities were reported in 57% of patients, including headache (25%), motor dysfunction (16%), and sensory neuropathy (15%; Janssen Biotech, Inc., 2026). Infectious complications were also prevalent, occurring in 80% of patients, with 55% experiencing grade 3 or 4 infections (Nooka et al., 2024). The most common types of infections included respiratory infections (56%), COVID-19 (21.2%), and gastrointestinal infections (10%; Nooka et al., 2024).

Elranatamab

In August 2023, the FDA granted accelerated approval for elranatamab-bcmm (FDA, 2023b). Like teclistamab, elranatamab-bcmm is a bsAb targeting both BCMA and CD3 on T cells. This treatment is indicated for adults with RRMM who have undergone at least four prior lines of therapy, including a PI, IMiDs, and an anti-CD38 monoclonal antibody (Pfizer Inc., 2026; Karwacz et al., 2020). In the MagnetisMM-3 phase II trial, it achieved an OR rate of approximately 61% in heavily pretreated patients, with 35% achieving a CR or better (Lesokhin et al., 2023). However, immune-mediated toxicities were common, with CRS occurring in about 58% of patients, although primarily grade 1, and ICANS occurring in approximately 3% (Lesokhin et al., 2023). Neurologic toxicity occurred in 59% of the patients, manifested by headache (24%), encephalopathy (15%), motor dysfunction (17%), sensory neuropathy (13%), and Guillain-Barré syndrome (0.5%; Pfizer Inc., 2026). Infections were frequent, affecting 70% of patients, with grade 3 or higher infections in 7% (Lesokhin et al., 2023). Hematologic toxicities were also prominent, with high rates of anemia (68%), neutropenia (62%), and thrombocytopenia (61%; Pfizer Inc., 2026). Despite these adverse effects, elranatamab-bcmm shows promising potential as a non-cellular, off-the-shelf therapy for difficult-to-treat RRMM.

Linvoseltamab

Linvoseltamab (Lynozyfic), a bsAb targeting both BCMA on MM cells and CD3 on T cells, received FDA approval on July 2, 2025, for the treatment of RRMM. This therapy facilitates precise T-cell redirection toward malignant plasma cells, enabling targeted immune-mediated cytotoxicity. In the pivotal phase I/II LINKER-MM1 trial, linvoseltamab demonstrated promising efficacy in a heavily pretreated population (patients who had received four or more prior lines of therapy). Among participants in the 200-mg dosing cohort (n = 117), the therapy achieved an ORR of 71%, with ≥ CR observed in over 50% of patients, depending on the length of follow-up. Furthermore, linvoseltamab at a dose of 200 mg showed greater efficacy compared to the 50-mg dose, particularly in patients with a high disease burden (Li et al., 2023). Notably, the median duration of response was 29.4 months, underscoring the durability of response with this agent, even in a heavily pretreated setting (Bumma et al., 2024). Linvoseltamab is also distinct in its route and frequency of administration. Unlike other BCMA × CD3 bsAbs such as teclistamab (administered subcutaneously once weekly) and elranatamab (given subcutaneously every 1–2 weeks), linvoseltamab is administered intravenously (IV) and has the potential for once-every-4-weeks maintenance dosing, offering a more convenient schedule (Waldschmidt et al., 2025).

In terms of safety, CRS occurred in 46% of patients, with the majority being grade 1 to 2. The median onset of CRS was 11 hours post-dose, and symptoms typically resolved within 15 hours. Neurotoxicity events were reported in 54% of patients, with 8% experiencing grade ≥ 3 events, most of which occurred during the step-up dosing phase and resolved within a median of 2 days. Common neurologic symptoms included confusion, depressed level of consciousness, and lethargy (Regeneron Pharmaceuticals, 2025). Hematologic toxicities were also frequently observed, including anemia (38%) and grade 3 to 4 neutropenia (42%). Infections were reported in 74% of patients, with 33% classified as grade ≥ 3, highlighting the need for careful infection monitoring and prophylaxis during treatment (Bumma et al., 2024).

GPRC5D-TARGETING AGENT

Talquetamab

Talquetamab-tgvs (Talvey) is a bsAb that targets G protein–coupled receptor, family C, group 5, member D (GPRC5D), a novel antigen highly expressed on malignant plasma cells in MM (Smith et al., 2019) but minimally expressed on normal tissues (Labanca et al., 2025). Talquetamab has one arm that binds GPRC5D on the myeloma cell and another that binds CD3 on T cells, bringing them together to stimulate T-cell activation and trigger immune-mediated killing of the cancer cells (Chari et al., 2022). In the MonumenTAL-1 phase I/II clinical trial, talquetamab demonstrated promising activity in RRMM, especially in heavily pretreated patients. Overall response rate exceeded 70% (Labanca et al., 2025), showing clinical benefit even in patients previously treated with BCMA-targeted therapies (Chari et al., 2022; Holstein et al., 2023). Like other T-cell redirecting therapies, talquetamab carries the risk of CRS, which was reported in approximately 77% to 80% of patients depending on dosing regimen, with all cases being grade 1 or 2 (Labanca et al., 2025). The median time for CRS onset was 27 hours after the last dose, with a median duration of 17 hours (Labanca et al., 2025; Janssen Biotech, Inc., 2025b). Immune effector cell–associated neurotoxicity syndrome occurred in about 9% of patients, with grade 3 or 4 neurotoxicity observed in 6% (Janssen Biotech, Inc., 2025b). The median onset and duration were 2.5 days following the most recent dose and 2 days, respectively (Janssen Biotech, Inc., 2025b).

Patients receiving talquetamab also experienced unique toxicities, including skin-related and nail changes, as well as taste disturbances and oral toxicities related to the high level of GPRC5D expression in epithelial and keratinized tissues (Chari et al., 2024). Rash toxicities were observed in 57 patients (39.9%) in the 0.4 mg/kg once-a-week cohort, compared to 43 patients (29.7%) in the 0.8 mg/kg every-2-weeks cohort (Labanca et al., 2025). Rash-related toxicities included maculopapular rash, erythematous rash, and erythema (Catamero et al., 2024). Nail toxicity ranged from discoloration to more severe conditions like onychomadesis and onycholysis, nail dystrophy, and ridging, all considered grade 1 (Labanca et al., 2025). Oral toxicities were common, affecting approximately 80% of patients, with symptoms like dysgeusia, dry mouth, and dysphagia (Chari et al., 2022). Weight loss was observed in 62% of patients, with nearly 30% experiencing a loss of 10% or more (Chari et al., 2022). In the MonumenTAL-1 trial, the incidence of infections was observed in 58.7% of patients receiving the 0.4 mg/kg dose, which accounted for 84 individuals. In comparison, the 0.8 mg/kg cohort experienced a higher infection rate of 66.2%, involving 96 patients (Labanca et al., 2025). The predominant severe infections identified included COVID-19, pneumonia, and urinary tract infections, all occurring within the first 100 days following the initiation of treatment (Chari et al., 2024). Opportunistic infections were identified in five patients (3.5%) within the cohort receiving a dosage of 0.4 mg/kg on a weekly basis, while eight patients (5.5%) presented with such infections in the cohort administered 0.8 mg/kg biweekly (Labanca et al., 2025). Hematologic toxicities included neutropenia grade 3 or 4 in 35% and thrombocytopenia in 22% (Janssen Biotech, Inc., 2025b). Overall, 9% of patients discontinued treatment due to adverse events, highlighting the need for close monitoring and supportive care during therapy (Janssen Biotech, Inc., 2025b). Talquetamab-related toxicity management strategies are in Table 1.

Table 1. Key Side Effects of Talquetamab With Clinical Relevance and Management Approaches

Side effects	Description	Frequency	Management strategies
Rash	Maculopapular rash, xerosis, palmar-plantar keratoderma	Common (≥ 20%)	Moisturizers, gentle cleansers, topical steroids, antihistamines if pruritic, ammonium lactate cream, triamcinolone cream, and Vaseline
Dry mouth (xerostomia)	Salivary gland toxicity; contributes to swallowing and taste issues	Common	Frequent sips of water, sugar-free gum/lozenges, saliva substitutes and rinses, avoid caffeine/alcohol
Dysphagia (difficulty swallowing)	Often linked to mucosal and salivary changes	Common	Soft/pureed diet, swallow therapy, topical/oral analgesics, nutritional support
Hair loss (alopecia/thinning)	Usually mild thinning, not universal	Less common	Gentle hair care, avoid harsh chemicals/heat, scalp cooling (if available), reassurance
Dysgeusia (taste disturbance)	Altered or loss of taste; major contributor to weight loss	Very common (up to 70%)	Flavor enhancers (lemon, spices, marinades), metal-free utensils, referral to dietitian, nutritional supplements, high-caloric shakes
Nail dystrophy	Brittle nails, dystrophy, sometimes painful nail loss	Common and clinically significant	Keep nails short, moisturizers, topical antibiotics if secondary infection, vitamin E oil, systemic hydration, biotin, triamcinolone 0.025% ointment, and protective nail coverings
Mucositis	Oral ulcerations, inflammation, pain	Less common	Oral hygiene, salt/soda rinses, avoid spicy/acidic foods, topical anesthetics, cryotherapy (ice chips)
Hand erythema/ hand-foot syndrome	Palmar-plantar keratoderma, erythema, discomfort	Common	Emollients, avoid friction/heat, dose adjustment if severe, Vanicream, ammonium lactate 12% cream, triamcinolone 0.1% cream
Pruritus	Often accompanies rash and xerosis	Common, mild-moderate	Antihistamines, topical corticosteroids, moisturizers, oatmeal baths

Note. Information from Mancia et al. (2021); Chari et al. (2024).

NEW PRODUCTS/TARGETS

Other bsAbs targeting BCMA and additional novel antigens are being actively investigated, with studies exploring their potential use in earlier lines of therapy. Cevostamab, a bsAb directed against Fc receptor-homolog 5 (FcRH5) and CD3, is under evaluation in patients with RRMM who have previously received anti-BCMA therapy (Zhao et al., 2023). By redirecting T cells to selectively eliminate myeloma cells, cevostamab leverages the near-universal expression of FcRH5 on plasma cells, supporting its potential efficacy in this patient population (Li et al., 2017). Additionally, F182112 is a bsAb that targets BCMA and CD3, redirecting $CD3^+$ T cells to recognize and lyse multiple myeloma cells expressing BCMA (D'Souza et al., 2022). In the NTP-F182112-001 (NCT04984434) trial, 22 patients received F182112 across eight dose levels, with an overall response rate of 45%, which was highest in the 10 and 20 µg/kg groups. Most experienced treatment-related adverse events, mainly CRS (72.7%), lymphopenia (68.2%), neutropenia (54.6%), leukopenia (50%), and anemia (31.8%), with all CRS events being grade 1 or 2 (Wang et al., 2024).

Alnuctamab is a humanized, trivalent bispecific antibody that binds bivalently to BCMA and monovalently to CD3 (Klein et al., 2019; Costa et al., 2019). In a phase I trial (NCT03486067), intravenous administration in patients with RRMM showed a median response duration of 33.6 months but was associated with CRS in 76% of patients. Switching to subcutaneous (SC)

dosing improved safety while maintaining efficacy: among 73 patients treated, the ORR was 54% across all doses, increasing to 63% at the target dose (≥ 30 mg), with all assessable patients achieving MRD negativity. Cytokine release syndrome was most frequent after the first incremental dose (40%) and decreased to < 1% per subsequent dose, demonstrating that SC alnuctamab combines promising antitumor activity with a favorable safety profile (Wong et al., 2022; Bar et al., 2023). In January 2024, a phase III study of alnuctamab was added to ClinicalTrials.gov, aiming to enroll 466 patients to evaluate whether the drug could improve PFS in individuals with RRMM. However, within just a few months of the trial's initial filing, the pharmaceutical company decided to abandon the study. According to the sponsor, this decision was driven by a shift in the company's business objectives. Additionally, slow patient enrollment and increased competition in the BCMA-targeting space, especially from late-stage CAR T-cell therapies, contributed to the trial's discontinuation (Quistgaard, 2024).

ABBV-383 is a fully human IgG4 bispecific antibody developed using knob-in-hole technology (Voorhees et al., 2022). In a phase I study (NCT03933735), ABBV-383 showed promising activity in patients with RRMM. Updated dose-expansion results demonstrated deep and durable responses at 40 mg and 60 mg, with a median PFS of 13.7 and 11.2 months, respectively, and 12-month duration of response rates of 70% and 66%. Cytokine release syndrome and ICANS occurred in 50% to 71% and 3% to 5% of patients across doses, respectively (Vij et al., 2023). Further clinical evaluation of ABBV-383 is ongoing.

RO7297089 is a bispecific antibody targeting BCMA and CD16a (Plesner et al., 2023). In a phase I study (NCT04434469), weekly intravenous doses ranging from 60 to 1,850 mg were well tolerated in patients with RRMM. However, the single-agent efficacy of RO7297089 was modest compared with that observed with BCMA-targeted bispecific antibodies (Usmani et al., 2021).

WVT078 is a potent anti-BCMA × anti-CD3 bsAb. Monotherapy with WVT078 has demonstrated a manageable level of toxicity in an ongoing study (NCT04123418), with an ORR of 38.5% at doses ranging from 48 to 250 μg/kg and 75% at the highest tested dose. BCMA is cleaved by gamma-secretase to produce soluble BCMA (sBCMA), which can bind to BCMA-targeting drugs and interfere with their activity (Wang et al., 2024). Overall, bsAbs show promising efficacy results with generally manageable AEs. A key difference between CAR T-cell therapy and bsAbs is their administration: CAR T-cell therapy is typically a one-time infusion, whereas bsAbs require repeated weekly or biweekly doses due to their shorter half-life. This difference allows bsAbs to be more accessible in community centers and suitable for frail or disadvantaged patients. Refer to Table 2 for the schedule, dosing options, and route of administration of bispecific antibodies.

Multiple myeloma is considered a disease of the immune system that causes dysfunction in immune regulation of plasma cells, leading to impaired immune responses, increased susceptibility to infections, and a higher risk of complications (Raje et al., 2023). Contributing factors include prolonged cytopenias, hypogammaglobulinemia, defects in T-cell and B-cell immunity, and issues with BCMA signaling. As a result, patients eligible for bsAbs are often immunocompromised due to prior treatments and generally experience significant therapy-related complications. Clinical trials have shown an increased risk of infections, such as *Pneumocystis jirovecii* pneumonia (PJP), cytomegalovirus (CMV) reactivation, and hepatitis B virus (HBV). Current guidelines recommend monitoring, prophylactic measures, and treatment strategies, such as antiviral and PJP prophylaxis, to prevent infections and improve care access in the community. Administration of intravenous immunoglobulin (IVIG) is also indicated when IgG levels drop below 400 mg/dL or if recurrent infections occur, as low IgG levels significantly increase the risk of severe infections and poor outcomes (Los-Arcos et al., 2021). Refer to Table 3 for the common antimicrobial prophylaxis strategies used in patients receiving bsAbs.

ASSESSING SUITABILITY FOR OUTPATIENT ADMINISTRATION OF bsAbs IN RRMM

T-cell engagers represent some of the most potent single-agent therapies ever developed for

Table 2. Bispecific Antibodies in Multiple Myeloma: Indications, Administration, and Dosing

Agent	Indication	Route of administration	Schedule	Dosing
Talquetamab	RRMM after ≥ 4 prior lines of therapy, including a PI, IMiDs, and anti-CD38 moAb	SC	Weekly, biweekly	Recommended weekly schedule: Step-up dosing includes Day 1: 0.01 mg/kg, Day 4: 0.06 mg/kg, Day 7: 0.4 mg/kg, then maintenance with 0.4 mg/kg once weekly (at least 6 days apart). Biweekly schedule: Step-up dosing includes Day 1: 0.01 mg/kg, Day 4: 0.06 mg/kg, Day 7: 0.4 mg/kg, Day 10: 0.8 mg/kg, then maintenance: 0.8 mg/kg every 2 weeks (minimum 12 days apart).
Linvoseltamab	RRMM after ≥ 4 prior lines of therapy, including a PI, IMiDs, and anti-CD38 moAb	IV	Weekly, biweekly, monthly	Recommended administration includes step-up doses of 5 mg, 25 mg, and 200 mg, followed by 200 mg weekly for 10 doses, followed by 200 mg biweekly. In patients who have achieved and maintained a VGPR or better at or after week 24 and received at least 17 doses of 200 mg, the dosing frequency is decreased to 200 mg every 4 weeks.
Teclistamab	RRMM after ≥ 4 prior lines of therapy, including a PI, IMiDs, and anti-CD38 moAb	SC	Weekly, biweekly	Recommended teclistamab dose is 0.06 mg/kg SC on Day 1, 0.3 mg/kg on Day 4, and 1.5 mg/kg on Day 7, followed by 1.5 mg/kg once weekly until POD or unacceptable toxicity. Patients who achieve and maintain CR or better for a minimum of 6 months can receive 1.5 mg/kg biweekly.
Elranatamab	RRMM after ≥ 4 prior lines of therapy, including a PI, IMiDs, and anti-CD38 moAb	SC	Weekly, biweekly	Step-up dosing (week 1): Day 1 (12 mg), Day 4 (32 mg) (at least 2 days after Day 1), Day 8 (76 mg) (at least 3 days after Day 4), then weekly maintenance (weeks 2–24) with 76 mg (minimum 6 days between doses). Extended interval dosing (responders only): Every 2 weeks (76 mg) starting week 25, for patients with PR or better maintained for ≥ 2 months. Every 4 weeks (76 mg) from Week 49 onward, for those who sustain response on biweekly dosing.

Note. RRMM = relapsed/refractory multiple myeloma; PI = proteasome inhibitor; IMiDs = immunomodulatory agents; moAb = monoclonal antibody; SC = subcutaneously; IV = intravenously; VGPR = very good partial response; POD = progression of disease; PR = partial response. Information from Janssen Biotech, Inc. (2025b); Pfizer Inc. (2026); Janssen Biotech, Inc. (2026); Regeneron Pharmaceuticals (2025).

RRMM, offering convenient formulation and straightforward administration. These features confer significant advantages for patients, improve treatment accessibility, and support broader implementation in community-based settings (Pelland et al., 2025). However, their use outside of specialized centers presents several challenges. These include the need for ongoing monitoring of acute toxicities, particularly CRS and neurotoxicity; the management of prolonged cytopenias, which elevate the risk of infections; and the requirement for robust healthcare infrastructure and trained clinical staff to ensure safe delivery and prompt management of adverse events (Pelland et al., 2025).

A recent review by the Mayo Clinic involving 57 patients who received step-up dosing of T-cell engagers in an outpatient setting demonstrated that this approach is both safe and feasible, while also potentially reducing the unnecessary utilization of hospital resources (Sandahl et al., 2025). This outpatient model incorporates elements of hospital-level care at home, including remote monitoring, digital health technologies, and coordinated medical oversight, enabling timely management of adverse events and supporting broader

Table 3. Common Antimicrobial Prophylaxis Strategies for Patients Receiving Bispecific Antibodies

Type of prophylaxis	Medication/approach	Indications
Antiviral	Acyclovir or valacyclovir	Prevent HSV and VZV reactivation during therapy
Antibacterial	Levofloxacin or equivalent	For prolonged neutropenia (ANC < 500 for 7 days or greater), risk-based use
Antifungal	Fluconazole (or posaconazole if high risk)	Prolonged neutropenia or history of fungal infections, risk-based use
PJP	Trimethoprim-sulfamethoxazole or alternatives (dapsone, atovaquone, inhaled or intravenous pentamidine)	Prolonged corticosteroids, T-cell suppression, or low CD4 count. Continue for at least 3 months post last bispecific antibody dose.
Immunoglobulin replacement	IVIG	IgG levels < 400 mg/dL (and/or recurrent infections)
Vaccinations	Inactivated vaccines (e.g., influenza, COVID-19)	Before and during therapy; avoid live vaccines

Note. HSV = herpes simplex virus; VZV = varicella-zoster virus; ANC = absolute neutrophil count; PJP = *Pneumocystis jirovecii* pneumonia; IVIG = intravenous immunoglobulin. Information from Lancman et al. (2023); Raje et al. (2023).

implementation outside of tertiary centers (Pelland et al., 2025). See Figure 2 for patient eligibility factors for outpatient bsAb administration.

Additionally, emerging real-world data suggest that prophylactic tocilizumab may reduce the incidence of CRS without compromising the efficacy of bsAbs. In a study by Kowalski et al. (2025), 119 patients received a single dose of tocilizumab prior to the first dose of a bispecific antibody targeting either BCMA × CD3 or GPRC5D × CD3. The ORR was 65.7% (95% CI = 55.8%–74.7%), indicating preserved clinical activity. The overall CRS rate was low at 10.1% (95% CI = 5.3%–17%), with drug-specific rates of 8.9% for teclistamab, 12.5% for elranatamab, 0% for linvoseltamab, and 13% for talquetamab. Most CRS events (10 of 12) were grade 1, with no grade 3 events, and no additional doses of tocilizumab or corticosteroids were required. Rates of ICANS were also low (5.9%, 95% CI = 2.4%–11.7%) and comparable to those observed without prophylactic intervention (Kowalski et al., 2025). In the MajesTEC-1 trial, the use of prophylactic tocilizumab prior to teclistamab administration was associated with a significantly reduced incidence of CRS. Cytokine release syndrome occurred in 25% of patients in the prophylactic cohort, compared to 72% in the non-prophylactic group, with all events limited to grade 1 or 2 severity (Moreau et al., 2022). These results further support the role of prophylactic tocilizumab as a preventive strategy to mitigate CRS in patients receiving bispecific antibodies, thereby enhancing the safety profile of these therapies and improving the feasibility of outpatient administration.

Expanding access to bispecific antibodies in MM requires a strong emphasis on education, infrastructure, and coordination, particularly in the outpatient setting. Educating both patients and community-based health-care providers is essential to ensure that these therapies are delivered safely and effectively outside of academic centers. Early referral to specialized cancer centers remains critical for initial evaluation and initiation, but with proper training and protocols, ongoing care can often be transitioned to the community. Equipping community clinicians with the knowledge and tools to recognize and manage toxicities, such as CRS, neurotoxicity, and prolonged cytopenias, is vital. This includes familiarity with step-up dosing, monitoring protocols, and access to emergency interventions like tocilizumab. Ensuring that appropriate resources, including trained staff, real-time monitoring systems, and clear referral pathways, are in place allows for safe outpatient administration, even in lower-resource or rural settings. This approach supports the broader, equitable adoption of bispecific therapies, helps reduce disparities in access to care, and promotes a more integrated health-care network that delivers innovative treatment to patients regardless of

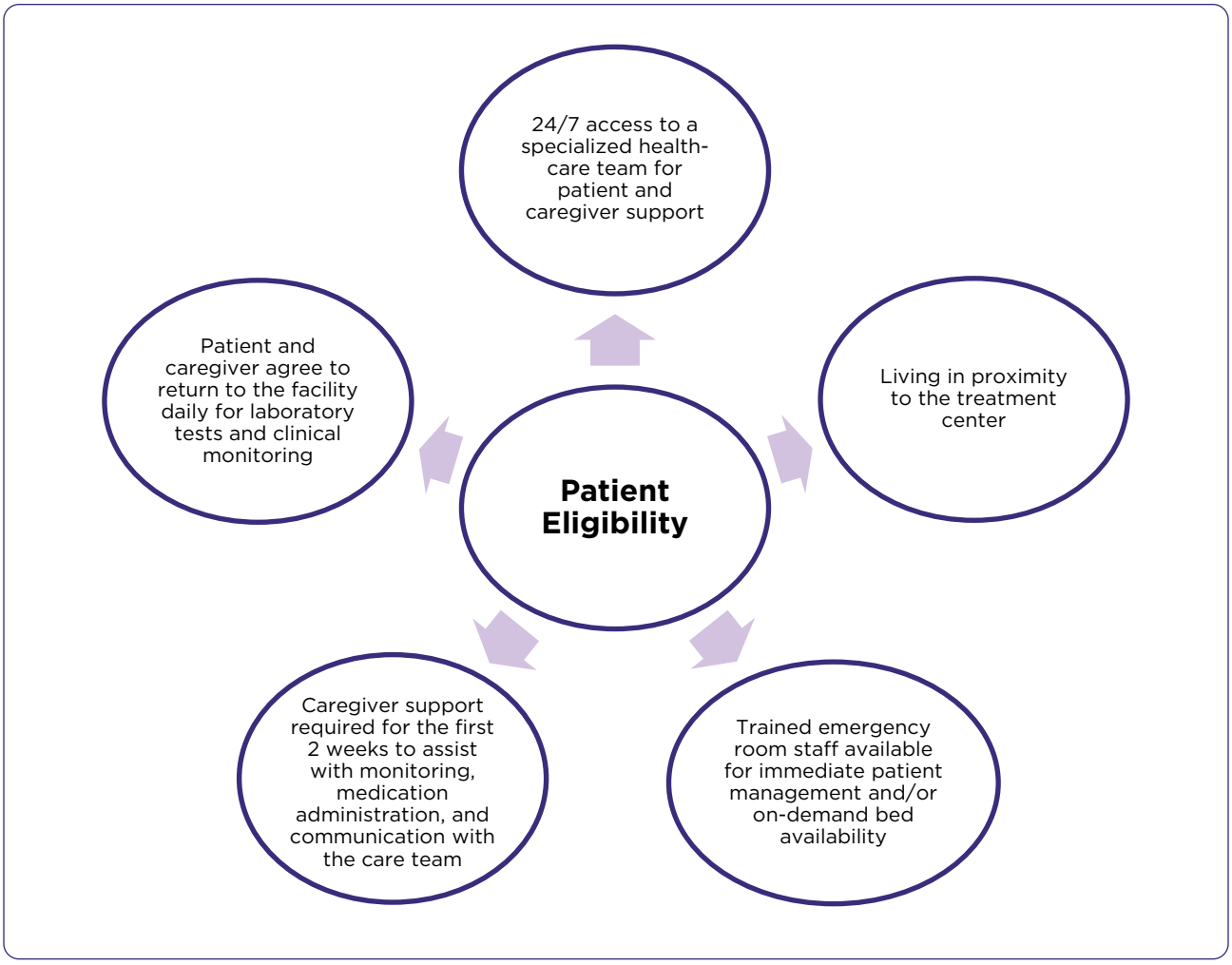

Figure 2. Defining patient eligibility for outpatient bispecific antibody therapy.

geography or socioeconomic status. Ultimately, building community capacity for safe outpatient delivery of bsAbs is key to unlocking their full potential in real-world practice.

CONCLUSION

Although MM remains incurable, recent therapeutic advances have significantly improved survival and clinical outcomes for patients with relapsed/refractory disease. These off-the-shelf, readily administered therapies hold great promise for broader use beyond academic centers. However, to safely expand access in community settings, several barriers must be addressed.

Ensuring equitable access to bsAbs requires educating and training local oncologists and multidisciplinary teams to recognize eligible patients early and initiate timely referrals to specialized centers when needed. As more patients begin bsAb therapy closer to home, robust infrastructure must be in place to manage potential treatment-related toxicities such as CRS, neurotoxicity, and cytopenias. This includes access to trained staff, 24/7 clinical support, coordinated care plans, and caregiver engagement during the initial treatment phase. With the right resources and support systems, safe outpatient administration of bsAbs in community settings is achievable, helping to reduce geographic and socioeconomic disparities in care. Empowering local providers through education and collaboration with specialized centers will be critical to realizing the full potential of bsAbs and improving outcomes for patients across all care environments.

IMPLICATIONS FOR PRACTICE AND FUTURE DIRECTIONS

As trusted providers on the front lines, APPs are uniquely positioned to influence patient outcomes through their advanced knowledge, clinical skills, and holistic approach to care. In our rapidly evolving health-care landscape, it is essential for these health-care providers to stay current with the latest evidence-based practices, particularly regarding novel cancer treatments. This expertise enables APPs to advocate for equitable access to innovative therapies for all eligible patients, irrespective of social or financial barriers. Ensuring high-quality care is a shared responsibility that demands collaboration among health-care professionals, organizations, and society as a whole. This underscores the importance of ongoing education, advocacy, and public engagement by informing patients, educating peers, and influencing policy to promote health-care equity, expand access, and improve health outcomes across diverse populations. ●

Disclosures

The authors have no conflicts of interest to disclose.

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