EJPMR, 2026, 13(4), 281-286 **Review Article** **Impact Factor: 8.158**

EUROPEAN JOURNAL OF PHARMACEUTICAL AND MEDICAL RESEARCH

www.ejpmr.com

ISSN (O): 2394-3211
ISSN (P): 3051-2573
Coden USA: EJPMAG

ADVANCES IN DIAGNOSIS AND TREATMENT OF MULTIPLE MYELOMA: A MOLECULAR AND THERAPEUTIC UPDATE

Gamal Abdul Hamid[*1,2], Ivan Saleh[1]

[1]National Oncology Center, Aden.
[2]Department of Hematology, Faculty of Medicine Health Sciences, University of Aden.

***Corresponding Author: Gamal Abdul Hamid**
National Oncology Center, Aden.

DOI: https://doi.org/10.5281/zenodo.19415294

How to cite this Article: Gamal Abdul Hamid[*1,2], Ivan Saleh[1]. (2026). Advances In Diagnosis and Treatment of Multiple Myeloma: A Molecular and Therapeutic Update. European Journal of Pharmaceutical and Medical Research, 13(4), 281–286.

Article Received on 05/03/2026 **Article Revised on 25/03/2026** **Article Published on 04/04/2026**

ABSTRACT
Multiple myeloma (MM) is a clinically and biologically heterogeneous plasma cell malignancy, representing the second most common hematologic cancer. Recent years have witnessed transformative advances in molecular diagnostics, risk stratification, and therapeutic modalities, leading to significantly improved survival outcomes. This review provides a comprehensive update on the epidemiology, molecular pathogenesis, diagnostic criteria, staging systems, and contemporary treatment paradigms of MM. Emphasis is placed on the integration of next-generation sequencing, fluorescence in situ hybridization (FISH), and minimal residual disease (MRD) monitoring into clinical practice. The advent of proteasome inhibitors, immunomodulatory drugs, monoclonal antibodies, chimeric antigen receptor (CAR) T-cell therapy, and bispecific antibodies has redefined MM management. Despite these advances, high-risk cytogenetic subgroups continue to pose therapeutic challenges. This manuscript highlights the shift toward personalized medicine in MM, discusses current clinical challenges, and outlines future research directions aimed at achieving durable remissions and potential cure.

KEYWORDS: Multiple myeloma, MGUS, cytogenetics, proteasome inhibitors, immunomodulatory drugs, CAR-T therapy, minimal residual disease, personalized medicine.

1. INTRODUCTION
Multiple myeloma (MM) is a clonal plasma cell malignancy characterized by the uncontrolled proliferation of malignant plasma cells within the bone marrow, the production of monoclonal immunoglobulins, and associated end-organ dysfunction.[1] Despite accounting for only 1.8% of all new cancer diagnoses, MM is the second most prevalent hematologic malignancy and contributes to 2.1% of cancer-related deaths in the United States.[2] The disease evolves along a clinical continuum, beginning with an asymptomatic premalignant phase termed monoclonal gammopathy of undetermined significance (MGUS), progressing to smoldering multiple myeloma (SMM), and culminating in active symptomatic MM marked by hypercalcemia, renal failure, anemia, and bone lesions (CRAB criteria).[3]

Historically, MM was associated with a poor prognosis, with a median survival of less than three years in the pre-novel therapy era. However, the past two decades have witnessed a paradigm shift in MM management, driven by profound insights into its molecular pathogenesis, the advent of high-throughput genomic technologies, and the development of targeted therapeutic agents.[4] The introduction of proteasome inhibitors (e.g., bortezomib), immunomodulatory drugs (e.g., lenalidomide), monoclonal antibodies (e.g., daratumumab), and, more recently, cellular therapies such as chimeric antigen receptor (CAR) T-cells, has dramatically improved patient outcomes, transforming MM into a chronic, manageable condition for many.[5]

Nevertheless, MM remains biologically heterogeneous and genetically complex. High-risk cytogenetic abnormalities, including del(17p), t(4;14), t(14;16), and amplification of 1q, continue to confer poor prognosis

and suboptimal responses to conventional therapies.[6] Furthermore, the emergence of clonal evolution and therapy-resistant subclones poses significant challenges to achieving durable remissions.[7] In this context, the integration of advanced molecular diagnostics—such as next-generation sequencing (NGS), fluorescence in situ hybridization (FISH), and minimal residual disease (MRD) monitoring—has become indispensable for risk stratification, treatment personalization, and prognostication.[8]

This review synthesizes the latest advancements in the diagnosis, risk stratification, and treatment of MM, with a particular emphasis on molecular innovations, emerging therapeutic modalities, and the evolving role of personalized medicine in improving clinical outcomes and overcoming therapeutic resistance.

2. EPIDEMIOLOGY

The global incidence of MM varies significantly by geographic region and ethnicity. The annual age-standardized incidence rate is approximately 5–7 per 100,000 in Western countries, with higher rates reported among African Americans (approximately 15 per 100,000) and lower rates in Asian populations.[9] MM is predominantly a disease of older adults, with a median age at diagnosis of 69 years. Only 2% of patients are diagnosed under the age of 40.[10]

Male gender, advancing age, African ancestry, family history of MM or MGUS, and obesity are established risk factors.[11] Environmental exposures, such as agricultural chemicals, benzene, and ionizing radiation, have also been implicated, though evidence remains inconclusive.[12] Importantly, nearly all cases of MM are preceded by MGUS, which progresses to MM at a rate of about 1% per year.[13]

3. CLASSIFICATION AND STAGING

3.1. Disease Classification

The International Myeloma Working Group (IMWG) criteria classify plasma cell disorders into:

- **MGUS:** Serum M-protein <3 g/dL, bone marrow plasma cells <10%, absence of CRAB features or myeloma-defining events.[14]
- **SMM:** Serum M-protein ≥3 g/dL and/or bone marrow plasma cells 10–60%, absence of CRAB features or myeloma-defining events.[15]
- **Active MM:** Clonal bone marrow plasma cells ≥10% or biopsy-proven plasmacytoma, plus one or more myeloma-defining events (CRAB features: hypercalcemia, renal failure, anemia, bone lesions; or biomarkers of malignancy: clonal bone marrow plasma cells ≥60%, serum free light chain ratio ≥100, >1 focal lesion on MRI).[16]

3.2. Staging Systems

- **Durie-Salmon Staging System:** An older system based on hemoglobin, calcium, M-protein levels, and bone lesions.[17]
- **International Staging System (ISS):** Based on serum β2-microglobulin and albumin levels.[18]
- **Revised ISS (R-ISS):** Incorporates ISS, LDH levels, and high-risk cytogenetic abnormalities [del(17p), t(4;14), t(14;16)].[19]
- **R2-ISS:** A newly proposed model that integrates gain/amplification of chromosome 1q to better stratify intermediate-risk patients.[20]

4. MOLECULAR BIOLOGY AND CYTOGENETICS

MM is characterized by complex genomic alterations that drive pathogenesis, progression, and therapy resistance.[21]

4.1. Primary Cytogenetic Abnormalities

These are early, foundational events

- **Hyperdiploidy (HRD):** Present in ~45% of cases, involves trisomies of odd-numbered chromosomes (3, 5, 7, 9, 11, 15, 19, 21). Associated with favorable prognosis.[22]
- **IgH Translocations:** Occur in ~40% of cases, place oncogenes under the control of the IgH enhancer.[23]
  - **t(11;14)(q13;q32):** Most common (15–20%), leads to CCND1 (cyclin D1) overexpression; generally favorable risk.[24]
  - **t(4;14)(p16;q32):** 10–15% of cases, involves FGFR3 and NSD2 (MMSET); considered high-risk.[25]
  - **t(14;16)(q32;q23) & t(14;20)(q32;q11):** Rare (<5%), involve MAF and MAFB oncogenes; associated with poor prognosis and renal involvement.[26]

4.2. Secondary Cytogenetic Abnormalities

Acquired during disease progression, associated with advanced disease and resistance.[27]

del(17p)/TP53 loss: High-risk marker, associated with extramedullary disease and poor survival.[28]

- **Gain/amplification of 1q:** Present in 30–40%, correlated with proliferative disease and inferior outcomes.[29]
- **RAS/MAPK pathway mutations:** NRAS, KRAS (40%), BRAF (7%).[30]
- **MYC rearrangements:** Occur in 20–50% of advanced MM.[31]
- **NF-κB pathway dysregulation:** Mutations in TRAF3, NFKB2, etc., promote survival and therapy resistance.[32]

4.3. The Bone Marrow Microenvironment

MM cells interact with stromal cells, osteoblasts, osteoclasts, and immune cells via cytokines (IL-6, VEGF, IGF-1) and adhesion molecules (VLA-4, VCAM-1), creating a pro-tumorigenic niche that supports growth, angiogenesis, bone destruction, and drug resistance.[33]

5. DIAGNOSIS

The diagnostic workup for MM is multidisciplinary and includes.[34]

1. **Laboratory Studies**
   - Complete blood count, creatinine, calcium, albumin, β2-microglobulin, LDH.
   - Serum protein electrophoresis (SPEP) and immunofixation (IFE) to quantify and type M-protein.
   - Serum free light chain assay (FLC) for ratio assessment.
   - 24-hour urine protein electrophoresis and immunofixation.

2. **Bone Marrow Examination**
   - Aspirate and biopsy for morphology, immunophenotyping (CD138+), and plasma cell percentage.
   - Cytogenetics (karyotyping) and FISH for high-risk abnormalities.
   - Next-generation sequencing for mutational profiling.

3. **Imaging**
   - Whole-body low-dose CT, MRI (spine/pelvis), or PET-CT to detect lytic lesions, extramedullary disease, and assess treatment response.

Minimal Residual Disease (MRD) assessment via next-generation flow cytometry or next-generation sequencing is increasingly used to evaluate depth of response and guide therapy.[35]

6. CLINICAL MANIFESTATIONS

Clinical features result from plasma cell infiltration, paraprotein effects, and immune dysfunction.[36]

- **Skeletal:** Bone pain, pathologic fractures, lytic lesions, hypercalcemia.
- **Renal:** Acute kidney injury (light-chain cast nephropathy), chronic kidney disease.
- **Hematologic:** Anemia, neutropenia, thrombocytopenia.
- **Infectious:** Recurrent bacterial infections due to hypogammaglobulinemia.
- **Neurologic:** Peripheral neuropathy, spinal cord compression.
- **Constitutional:** Fatigue, weight loss, hyperviscosity syndrome.

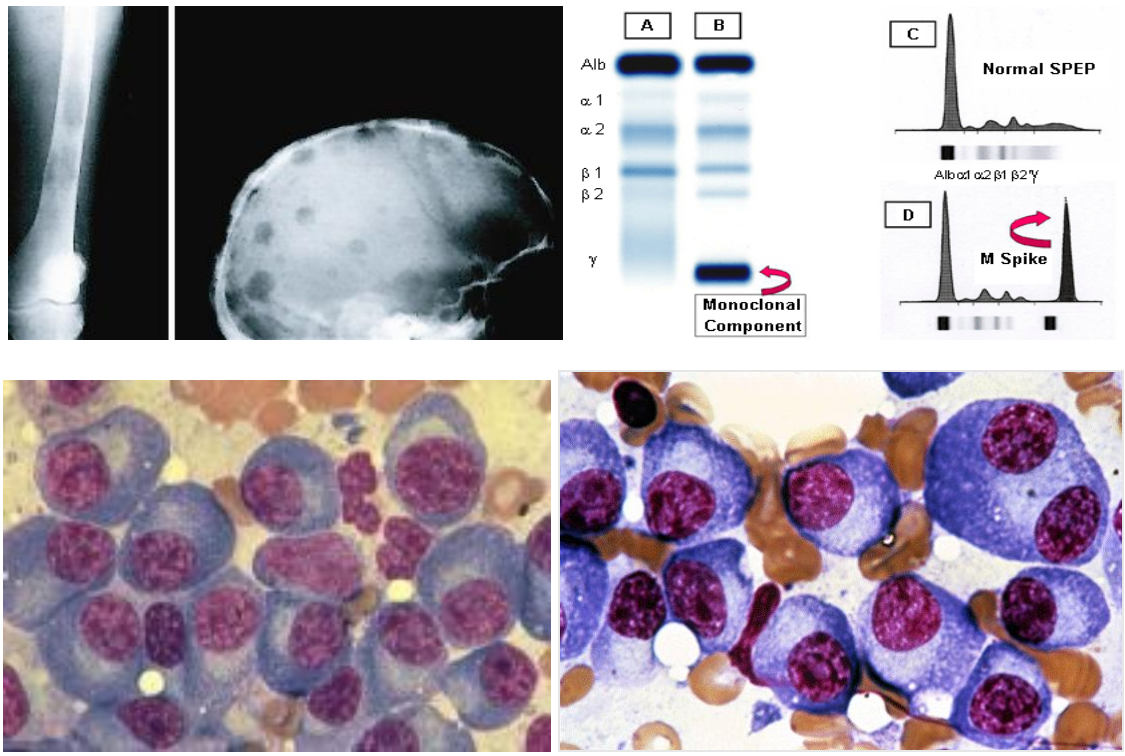

Figure 1. Osteolytic lesions, Nornal and pathological monoclonal component in SPEP and bone marrow plasma cells.

7. TREATMENT ADVANCES

Treatment strategies are tailored based on transplant eligibility, age, performance status, and cytogenetic risk.[37]

7.1. Newly Diagnosed Transplant-Eligible Patients

- **Induction Therapy:** Triplet regimens such as bortezomib/lenalidomide/dexamethasone (VRd) or carfilzomib/ lenalidomide/ dexamethasone (KRd) for 3–4 cycles.[38]

- **Autologous Stem Cell Transplantation (ASCT):** High-dose melphalan followed by ASCT remains standard for eligible patients.[39]
- **Consolidation/Maintenance:** Post-ASCT maintenance with lenalidomide improves progression-free and overall survival. Proteasome inhibitor-based maintenance is considered for high-risk disease.[40]

7.2. Newly Diagnosed Transplant-Ineligible Patients

- **First-line Therapy:** Daratumumab/lenalidomide/dexamethasone (DRd) or bortezomib/melphalan/prednisone (VMP) are preferred.[41]
- **Continuous Therapy:** Until progression or intolerance.

7.3. Relapsed/Refractory Multiple Myeloma (RRMM)

The treatment landscape has expanded dramatically.[42]

- **Monoclonal Antibodies**
  - Daratumumab and isatuximab (anti-CD38).[43]
  - Elotuzumab (anti-SLAMF7).[44]
- **CAR T-Cell Therapies**
  - Idecabtagene vicleucel (anti-BCMA).[45]
  - Ciltacabtagene autoleucel (anti-BCMA).[46]
- **Bispecific T-Cell Engagers (BiTEs)**
  - Teclistamab (BCMAxCD3).[47]
  - Talquetamab (GPRC5DxCD3).[48]
- **Novel Small Molecules**
  - Selinexor (XPO1 inhibitor).[49]
  - Venetoclax (BCL2 inhibitor; particularly effective in t(11;14) MM).[50]
  - Melflufen (peptide-drug conjugate).[51]

7.4. Supportive Care

- Bisphosphonates (zoledronic acid) or denosumab for bone health.[52]
- Erythropoiesis-stimulating agents, transfusions for anemia.
- Infection prophylaxis (antivirals, antibiotics, IVIg in selected cases).
- Management of treatment-related adverse effects (peripheral neuropathy, cytopenias, cardiac toxicity).

8. DISCUSSION

The management of MM has evolved from palliation to a precision medicine approach, driven by deep molecular profiling and biomarker-guided therapy. The integration of FISH, NGS, and MRD monitoring has refined risk stratification and treatment personalization.[53] Despite remarkable progress, several challenges persist:

- **High-Risk MM:** Patients with del(17p), amp(1q), or dual-hit genetics continue to have poor outcomes despite novel therapies.[54]
- **Treatment Resistance:** Clonal evolution and microenvironment-mediated resistance lead to relapse.[55]
- **Access and Cost:** Advanced therapies (CAR-T, bispecifics) are expensive and not universally accessible.[56]
- **Long-Term Toxicity:** Prolonged therapy can lead to secondary malignancies, organ toxicity, and diminished quality of life.[57]

Future directions include

- Development of next-generation immunotherapies (allogeneic CAR-T, NK cell therapies).[58]
- Targeting the bone marrow niche and immune microenvironment.[59]
- Epigenetic therapies and combination regimens to overcome resistance.[60]
- Universal MRD-driven treatment algorithms to guide therapy duration and escalation.[61]

9. CONCLUSION

Multiple myeloma remains a complex and heterogeneous disease, but unprecedented advances in molecular diagnostics and targeted therapeutics have transformed its prognosis. The shift toward personalized, risk-adapted therapy has improved survival and quality of life for many patients. Ongoing research into disease biology, resistance mechanisms, and novel immunotherapies holds promise for further improving outcomes, particularly in high-risk disease. A multidisciplinary, patient-centered approach, incorporating the latest diagnostic and therapeutic innovations, is essential for optimizing MM care in the modern era.

Conflicts of Interest: The author declares no conflicts of interest.

Funding: None.

Ethical

Approval: Not applicable.

ACKNOWLEDGMENTS: None.

10. REFERENCES

1. Rajkumar SV. Multiple myeloma: 2023 update on diagnosis, risk-stratification, and management. Am J Hematol. 2023; 98(7): 1086-107.
2. Siegel RL, Miller KD, Wagle NS, Jemal A. Cancer statistics, 2023. CA Cancer J Clin. 2023; 73(1): 17-48.
3. Kyle RA, Larson DR, Therneau TM, et al. Long-term follow-up of monoclonal gammopathy of undetermined significance. N Engl J Med. 2018; 378(3): 241-9.
4. Hamid GA, Clinical hematology 2013. https://doi.org/10.13140/RG.2.1.1477.1683
5. Kumar SK, Rajkumar SV, Dispenzieri A, et al. Improved survival in multiple myeloma and the

impact of novel therapies. Blood., 2008; 111(5): 2516-20.
6. Sonneveld P, Avet-Loiseau H, Lonial S, et al. Treatment of multiple myeloma with high-risk cytogenetics: a consensus of the International Myeloma Working Group. Blood. 2016; 127(24): 2955-62.
7. Bolli N, Avet-Loiseau H, Wedge DC, et al. Heterogeneity of genomic evolution and mutational profiles in multiple myeloma. Nat Commun. 2014; 5: 2997.
8. Avet-Loiseau H, Bene MC, Wuilleme S, et al. Minimal residual disease negativity using deep sequencing is a major prognostic factor in multiple myeloma. Blood. 2018; 132(23): 2456-64.
9. Cowan AJ, Allen C, Barac A, et al. Global burden of multiple myeloma: a systematic analysis for the Global Burden of Disease Study 2016. JAMA Oncol. 2018; 4(9): 1221-7.
10. Howlader N, Noone AM, Krapcho M, et al. SEER Cancer Statistics Review, 1975-2017. National Cancer Institute, 2020.
11. Waxman AJ, Mink PJ, Devesa SS, et al. Racial disparities in incidence and outcome in multiple myeloma: a population-based study. Blood. 2010; 116(25): 5501-6.
12. Landgren O, Kyle RA, Hoppin JA, et al. Pesticide exposure and risk of monoclonal gammopathy of undetermined significance in the Agricultural Health Study. Blood. 2009; 113(25): 6386-91.
13. Abdul Hamid G., Abbas RY. Clinical Profile of Multiple Myeloma in National Oncology Center, Aden, Yemen; Asian Hematology Research Journal; 2023; 6(3): 129-142. Article no. AHRJ 98983.
14. Rajkumar SV, Dimopoulos MA, Palumbo A, et al. International Myeloma Working Group updated criteria for the diagnosis of multiple myeloma. Lancet Oncol. 2014; 15(12): e538-48.
15. Mateos MV, Hernández MT, Giraldo P, et al. Lenalidomide plus dexamethasone for high-risk smoldering multiple myeloma. N Engl J Med., 2013; 369(5): 438-47.
16. Kumar SK, Callander NS, Adekola K, et al. Multiple myeloma, version 3.2021, NCCN Clinical Practice Guidelines in Oncology. J Natl Compr Canc Netw., 2020; 18(12): 1685-717.
17. Durie BG, Salmon SE. A clinical staging system for multiple myeloma. Correlation of measured myeloma cell mass with presenting clinical features, response to treatment, and survival. Cancer. 1975; 36(3): 842-54.
18. Greipp PR, San Miguel J, Durie BG, et al. International staging system for multiple myeloma. J Clin Oncol. 2005; 23(15): 3412-20.
19. Palumbo A, Avet-Loiseau H, Oliva S, et al. Revised International Staging System for multiple myeloma: a report from International Myeloma Working Group. J Clin Oncol. 2015; 33(26): 2863-9.
20. D'Agostino M, Cairns DA, Lahuerta JJ, et al. Second revision of the International Staging System (R2-ISS) for overall survival in multiple myeloma: a European Myeloma Network (EMN) report within the HARMONY project. J Clin Oncol. 2022; 40(29): 3406-18.
21. Fonseca R, Bergsagel PL, Drach J, et al. International Myeloma Working Group molecular classification of multiple myeloma: spotlight review. Leukemia. 2009; 23(12): 2210-21.
22. Chng WJ, Dispenzieri A, Chim CS, et al. IMWG consensus on risk stratification in multiple myeloma. Leukemia. 2014; 28(2): 269-77.
23. Bergsagel PL, Kuehl WM. Chromosome translocations in multiple myeloma. Oncogene. 2001; 20(40): 5611-22.
24. Fonseca R, Blood E, Rue M, et al. Clinical and biologic implications of recurrent genomic aberrations in myeloma. Blood. 2003; 101(11): 4569-75.
25. Keats JJ, Reiman T, Maxwell CA, et al. In multiple myeloma, t(4;14)(p16;q32) is an adverse prognostic factor irrespective of FGFR3 expression. Blood. 2003; 101(4): 1520-9.
26. Avet-Loiseau H, Attal M, Moreau P, et al. Genetic abnormalities and survival in multiple myeloma: the experience of the Intergroupe Francophone du Myélome. Blood. 2007; 109(8): 3489-95.
27. Lohr JG, Stojanov P, Carter SL, et al. Widespread genetic heterogeneity in multiple myeloma: implications for targeted therapy. Cancer Cell. 2014; 25(1): 91-101.
28. Chng WJ, Price-Troska T, Gonzalez-Paz N, et al. Clinical significance of TP53 mutation in myeloma. Leukemia. 2007; 21(3): 582-4.
29. Shah V, Sherborne AL, Walker BA, et al. Prediction of outcome in newly diagnosed myeloma: a meta-analysis of the molecular profiles of 1905 trial patients. Leukemia. 2018; 32(1): 102-10.
30. Lohr JG, Stojanov P, Carter SL, et al. Widespread genetic heterogeneity in multiple myeloma: implications for targeted therapy. Cancer Cell. 2014; 25(1): 91-101.
31. Walker BA, Boyle EM, Wardell CP, et al. Mutational spectrum, copy number changes, and outcome: results of a sequencing study of patients with newly diagnosed myeloma. J Clin Oncol. 2015; 33(33): 3911-20.
32. Annunziata CM, Davis RE, Demchenko Y, et al. Frequent engagement of the classical and alternative NF-kappaB pathways by diverse genetic abnormalities in multiple myeloma. Cancer Cell. 2007; 12(2): 115-30.
33. Hideshima T, Mitsiades C, Tonon G, Richardson PG, Anderson KC. Understanding multiple myeloma pathogenesis in the bone marrow to identify new therapeutic targets. Nat Rev Cancer. 2007; 7(8): 585-98.
34. Hamid G.A, Hanbala N.. Comparison of bone marrow aspiration and bone marrow biopsy in neoplastic diseases Gulf J Oncol 2009; 41-44.

35. Munshi NC, Avet-Loiseau H, Anderson KC, et al. A large meta-analysis establishes the role of MRD negativity in long-term survival outcomes in patients with multiple myeloma. Blood Adv. 2020; 4(23): 5988-99.
36. Kyle RA, Gertz MA, Witzig TE, et al. Review of 1027 patients with newly diagnosed multiple myeloma. Mayo Clin Proc. 2003; 78(1): 21-33.
37. Moreau P, Kumar SK, San Miguel J, et al. Treatment of relapsed and refractory multiple myeloma: recommendations from the International Myeloma Working Group. Lancet Oncol. 2021; 22(3): e105-18.
38. Richardson PG, Weller E, Lonial S, et al. Lenalidomide, bortezomib, and dexamethasone combination therapy in patients with newly diagnosed multiple myeloma. Blood. 2010; 116(5): 679-86.
39. Attal M, Lauwers-Cances V, Hulin C, et al. Lenalidomide, bortezomib, and dexamethasone with transplantation for myeloma. N Engl J Med. 2017; 376(14): 1311-20.
40. McCarthy PL, Holstein SA, Petrucci MT, et al. Lenalidomide maintenance after autologous stem-cell transplantation in newly diagnosed multiple myeloma: a meta-analysis. J Clin Oncol. 2017; 35(29): 3279-89.
41. Facon T, Kumar S, Plesner T, et al. Daratumumab plus lenalidomide and dexamethasone for untreated myeloma. N Engl J Med., 2019; 380(22): 2104-15.
42. Rajkumar SV. Multiple myeloma: 2023 update on diagnosis, risk-stratification, and management. Am J Hematol. 2023; 98(7): 1086-107.
43. Lokhorst HM, Plesner T, Laubach JP, et al. Targeting CD38 with daratumumab monotherapy in multiple myeloma. N Engl J Med., 2015; 373(13): 1207-19.
44. Lonial S, Dimopoulos M, Palumbo A, et al. Elotuzumab therapy for relapsed or refractory multiple myeloma. N Engl J Med., 2015; 373(7): 621-31.
45. Munshi NC, Anderson LD Jr, Shah N, et al. Idecabtagene vicleucel in relapsed or refractory multiple myeloma. N Engl J Med., 2021; 384(8): 705-16.
46. Berdeja JG, Madduri D, Usmani SZ, et al. Ciltacabtagene autoleucel, a B-cell maturation antigen-directed chimeric antigen receptor T-cell therapy in patients with relapsed or refractory multiple myeloma (CARTITUDE-1): a phase 1b/2 open-label study. Lancet, 2021; 398(10297): 314-24.
47. Moreau P, Garfall AL, van de Donk NW, et al. Teclistamab in relapsed or refractory multiple myeloma. N Engl J Med., 2022; 387(6): 495-505.
48. 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(24): 2232-44.
49. Chari A, Vogl DT, Gavriatopoulou M, et al. Oral selinexor-dexamethasone for triple-class refractory multiple myeloma. N Engl J Med., 2019; 381(8): 727-38.
50. Moreau P, Chanan-Khan A, Roberts AW, et al. Promising efficacy and acceptable safety of venetoclax plus bortezomib and dexamethasone in relapsed/refractory MM. Blood, 2017; 130(22): 2392-400.
51. Richardson PG, Oriol A, Larocca A, et al. Melflufen and dexamethasone in heavily pretreated relapsed and refractory multiple myeloma. J Clin Oncol, 2021; 39(7): 757-67.
52. Terpos E, Morgan G, Dimopoulos MA, et al. International Myeloma Working Group recommendations for the treatment of multiple myeloma-related bone disease. J Clin Oncol, 2013; 31(18): 2347-57.
53. Perrot A, Corre J, Avet-Loiseau H. Risk stratification and targets in multiple myeloma: from genomics to the bedside. Am Soc Clin Oncol Educ Book, 2018; 38: 675-80.
54. Neben K, Lokhorst HM, Jauch A, et al. Administration of bortezomib before and after autologous stem cell transplantation improves outcome in multiple myeloma patients with deletion 17p. Blood, 2012; 119(4): 940-8.
55. Maura F, Bolli N, Angelopoulos N, et al. Genomic landscape and chronological reconstruction of driver events in multiple myeloma. Nat Commun, 2019; 10(1): 3835.
56. Fonseca R, Abouzaid S, Bonafede M, et al. Trends in overall survival and costs of multiple myeloma, 2000-2014. Leukemia, 2017; 31(9): 1915-21.
57. Musto P, Anderson KC, Attal M, et al. Second primary malignancies in multiple myeloma: an overview and IMWG consensus. Ann Oncol, 2017; 28(2): 228-45.
58. Shah N, Chari A, Scott E, Mezzi K, Usmani SZ. B-cell maturation antigen (BCMA) in multiple myeloma: rationale for targeting and current therapeutic approaches. Leukemia, 2020; 34(4): 985-1005.
59. Gulla A, Anderson KC. Multiple myeloma: the (r)evolution of current therapy and a glance into the future. Haematologica, 2020; 105(10): 2358-67.
60. Moreau P, Touzeau C. Multiple myeloma: from front-line to relapsed therapies. Am Soc Clin Oncol Educ Book, 2015; 35: e504-11.
61. Paiva B, van Dongen JJ, Orfao A. New criteria for response assessment: role of minimal residual disease in multiple myeloma. Blood. 2015; 125(20): 3059-68.