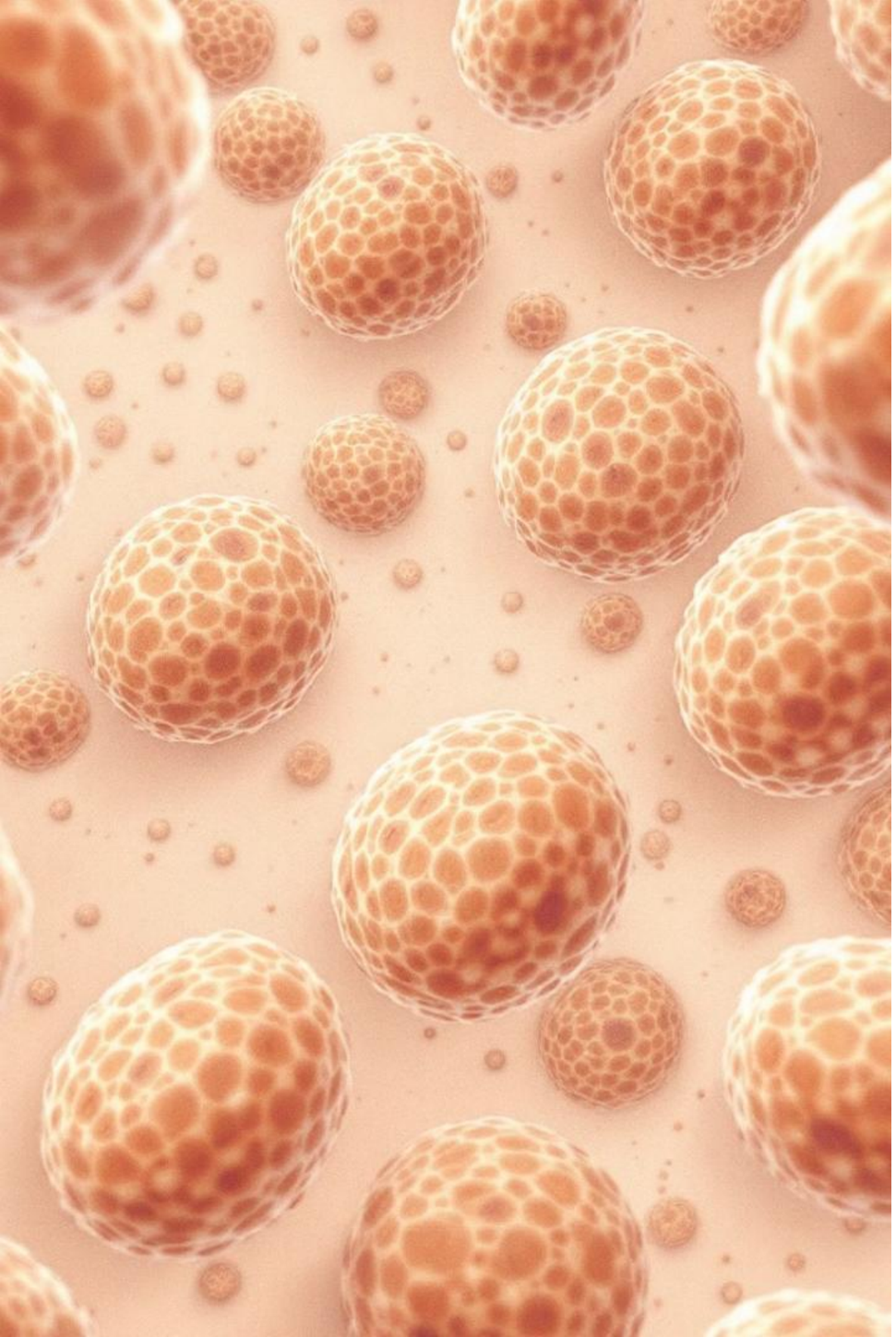


סדנת מחלות תאי פלזמה למתמחים בהמטולוגיה
24-11-2025
קראון פלאזה, עזריאלי תל אביב

Supportive Care and AE Management in Multiple Myeloma

Multiple myeloma is a complex hematological malignancy that requires comprehensive supportive care to manage the disease and its associated complications. This presentation will explore evidence-based strategies for managing adverse events, renal and bone disease, peripheral neuropathy, and the unique toxicities of novel therapies like CAR-T and BiTE.

Dr. Ory Rouvio,
Head, Internal Medicine A
Multiple Myeloma Clinic, Hematology Institution
Soroka University Medical Center
Beer- Sheva Israel

Evidence-Based Data: Clinical Trials and Real-World Outcomes

Clinical Trials

Landmark studies have demonstrated the efficacy of various drug regimens in treating multiple myeloma and managing associated complications.

Real-World Data

Post-approval surveillance and registry studies provide valuable insights into the real-world safety and effectiveness of myeloma therapies.

Bridging the Gap

Integrating clinical trial evidence with real-world data can guide personalized supportive care strategies for myeloma patients.

Topics to be discussed:

- Bone disease in MM
- Renal disease in MM
- Peripheral Neuropathy
- Infections
- Anemia
- Medications in MM: Mechanisms of Action and Safety Profiles
- CAR-T and BiTE Therapy: Adverse Events and Management

Bone Disease in Multiple Myeloma: Pathophysiology and Treatment

Bone Remodeling Imbalance

Myeloma cells disrupt the normal bone remodeling process, leading to osteolytic lesions and fractures.

Bisphosphonate Therapy

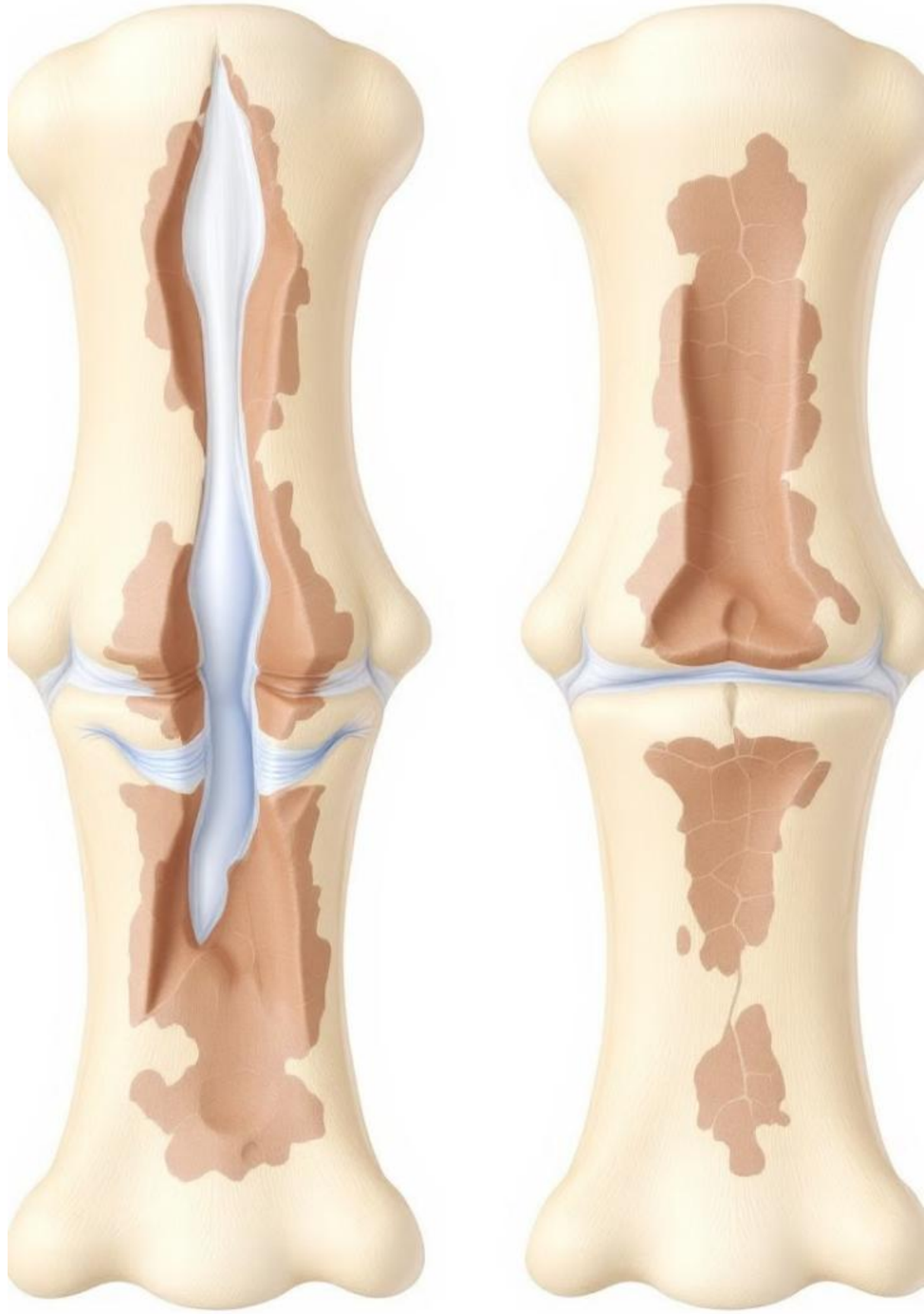
Bisphosphonates help reduce the risk of skeletal-related events and bone pain in myeloma patients.

Novel Bone-Targeting Agents

Emerging treatments like denosumab and radiopharmaceuticals offer additional options for managing myeloma bone disease.

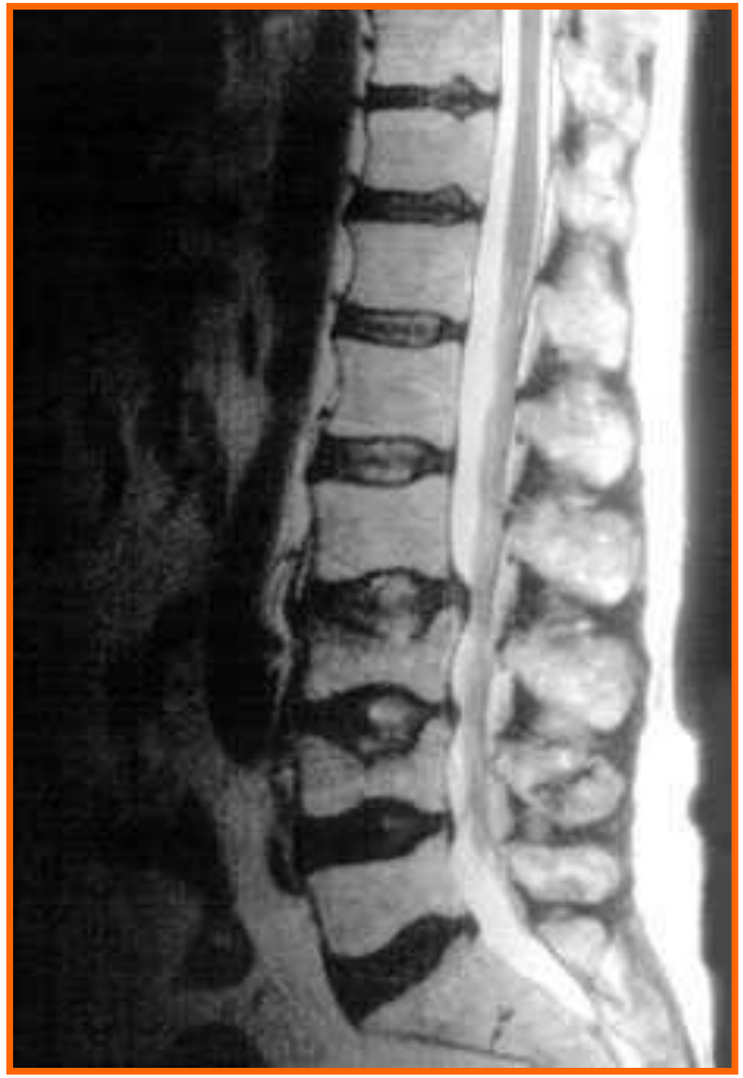
Supportive Measures

Orthopedic interventions, physical therapy, and pain management are crucial components of comprehensive bone care.



Bone Disease in Multiple Myeloma

- A burdensome and frequent complication in MM
  - **Present in up to 80% of patients at diagnosis**
- Characterized by osteolytic bone lesions secondary to increased bone resorption and impaired bone formation
- Sequelae
  - Pathological fractures
  - Osteoporosis
  - Hypercalcemia
  - Bone pain
  - Spinal cord compression



Bone Disease in MM

- **Almost 80% of patients have abnormalities in bone radiographs at diagnosis[1]**
- **Up to 90% of patients develop lytic lesions over the disease course[2,3]**

Sites commonly affected by lytic bone disease[2]	
Vertebrae	65%
Ribs	45%
Skull	40%
Shoulders	40%
Pelvis	30%
Long bones	25%

Lytic lesions in the femur[4]

Myeloma bone disease can lead to skeletal complications, including skeletal-related events, bone pain, and hypercalcemia[3]

1. Kyle RA, et al. *Mayo Clin Proc*. 2003;78:21-23. **2.** Dimopoulos M, et al. *Leukemia.* 2009;23;1545-1556. **3.** Terpos E, et al. *J Clin Oncol.* 2011;29:1907-1915. **4.** Terpos E, et al. *Am Soc Clin Oncol Educ Book.* 2016;35:e407-e417. image courtesy of Prof E. Terpos.

THE LANCET
Oncology

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Treatment of multiple myeloma-related bone disease: recommendations from the Bone Working Group of the International Myeloma Working Group

Prof Evangelos Terpos, MD • Elena Zamagni, MD • Prof Suzanne Lentzsch, MD • Matthew T Drake, MD • Ramón García-Sanz, MD • Prof Niels Abildgaard, MD • et al. Show all authors

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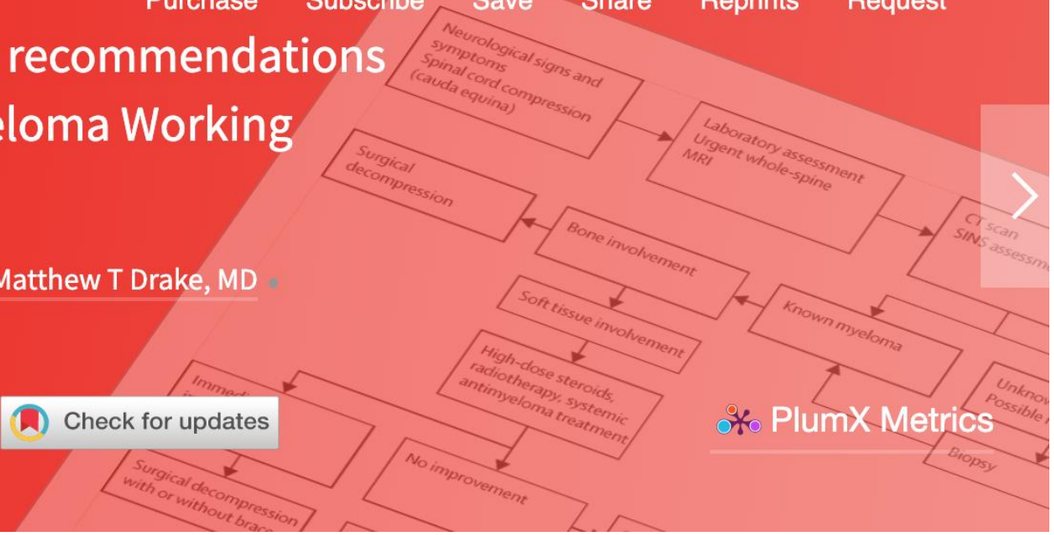
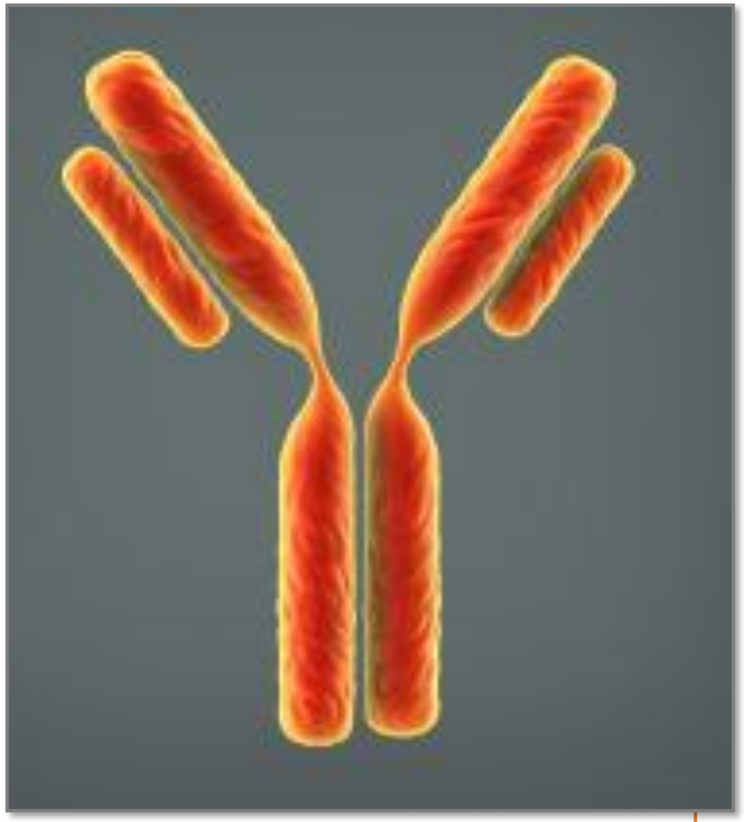


The Bone Working Group of the IMWG recommendations

- Grading recommendations using the Grading of Recommendations, Assessment, Development, and Evaluations (GRADE) method
- Zoledronic acid as the preferred bone-targeted agent for patients with newly diagnosed multiple myeloma, with or without multiple myeloma-related bone disease.
- Once patients achieve a very good partial response or better, after receiving **monthly** zoledronic acid for at least **12 months**, the treating physician can consider decreasing the frequency of or discontinuing zoledronic acid treatment.

Denosumab 120 mg SC Q4W is Approved for the Prevention of SREs in Adults With Advanced Malignancies Involving Bone



Denosumab is a human monoclonal antibody that binds to RANKL to inhibit osteoclast-mediated bone destruction[1]

Denosumab can be administered regardless of renal function and does not need to be dose adjusted, unlike bisphosphonates[2]

Patients with CrCl < 30 mL/min were excluded from the denosumab trials[3]

SC, subcutaneously; Q4W, once every 4 weeks.

1. XGEVA® Summary of Product Characteristics, Amgen. **2.** Raje N, et al. Presented at: American Society of Clinical Oncology 2017 Annual Meeting; June 2–7, 2017; Chicago, IL. **3.** XGEVA® (denosumab) EPAR, Amgen.

The Bone Working Group of the IMWG recommendations

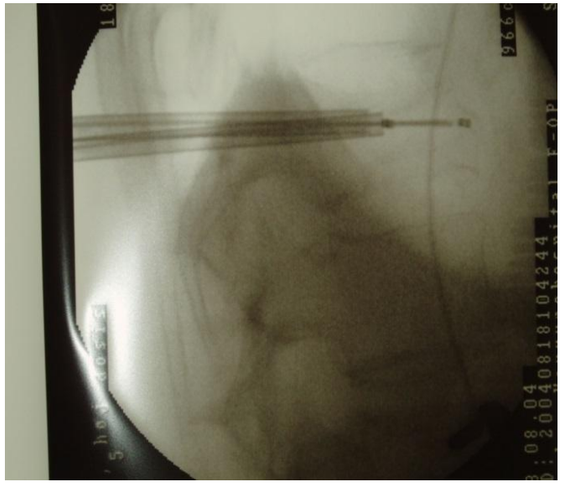
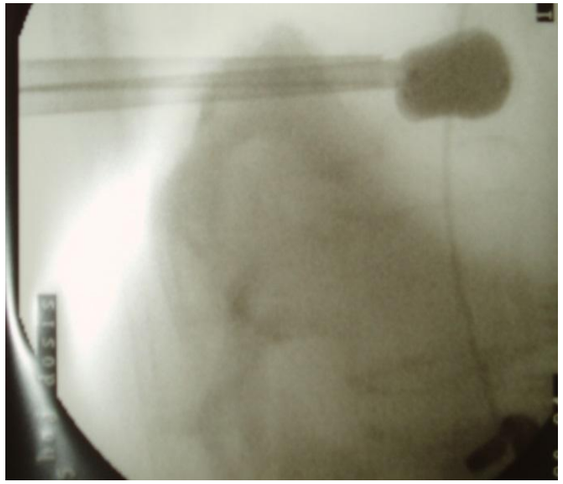
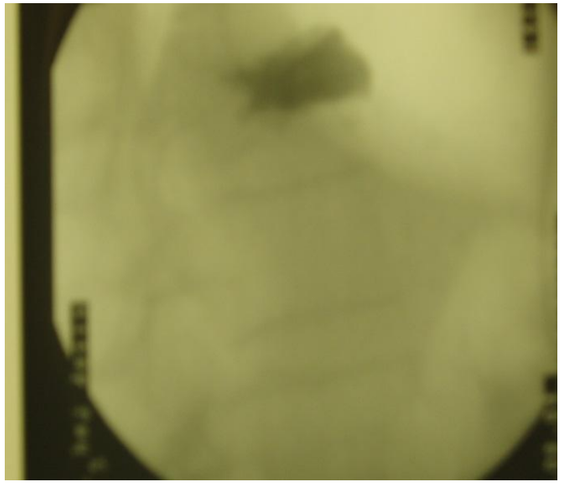
- **Denosumab** can also be considered for the treatment of multiple myeloma-related bone disease, particularly in patients with renal impairment.
- **Denosumab** might prolong progression-free survival in patients with newly diagnosed multiple myeloma who have multiple myeloma-related bone disease and who are eligible for autologous stem-cell transplantation.
- Denosumab discontinuation is challenging due to the rebound effect.

The Bone Working Group of the IMWG recommendations

- The Bone Working Group of the International Myeloma Working Group also found **cement augmentation** to be effective for painful vertebral compression fractures.
- **Radiotherapy** is recommended for uncontrolled pain, impending or symptomatic spinal cord compression, or pathological fractures. **Surgery** should be used for the prevention and restoration of long-bone pathological fractures, vertebral column instability, and spinal cord compression with bone fragments within the spinal route.

IMWG guidelines: Kyphoplasty

- Balloon kyphoplasty should be considered for symptomatic vertebral compression fractures and is the procedure of choice to improve quality of life in patients with painful vertebral compression fractures (grade A).
- Balloon kyphoplasty or vertebroplasty can relieve the pain of approximately 80% of patients unresponsive to medicinal treatment

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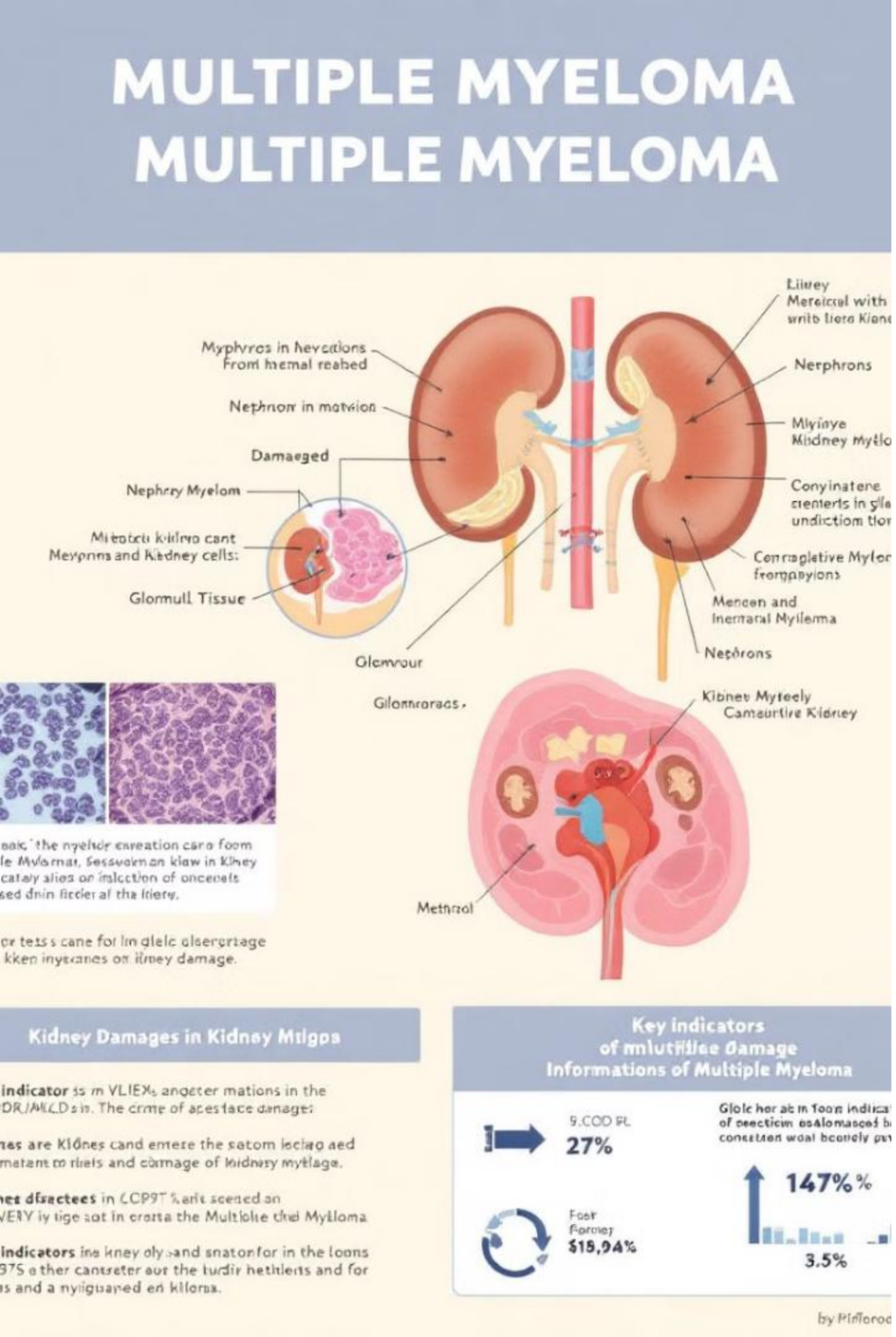


Renal Disease in Multiple Myeloma: Causes, Diagnosis, and Management

1

Renal Complications

Myeloma-related kidney injury can be caused by factors like cast nephropathy, light chain deposition, and amyloidosis.

2

Early Diagnosis

Prompt detection of renal impairment through lab tests and imaging is crucial for effective management.

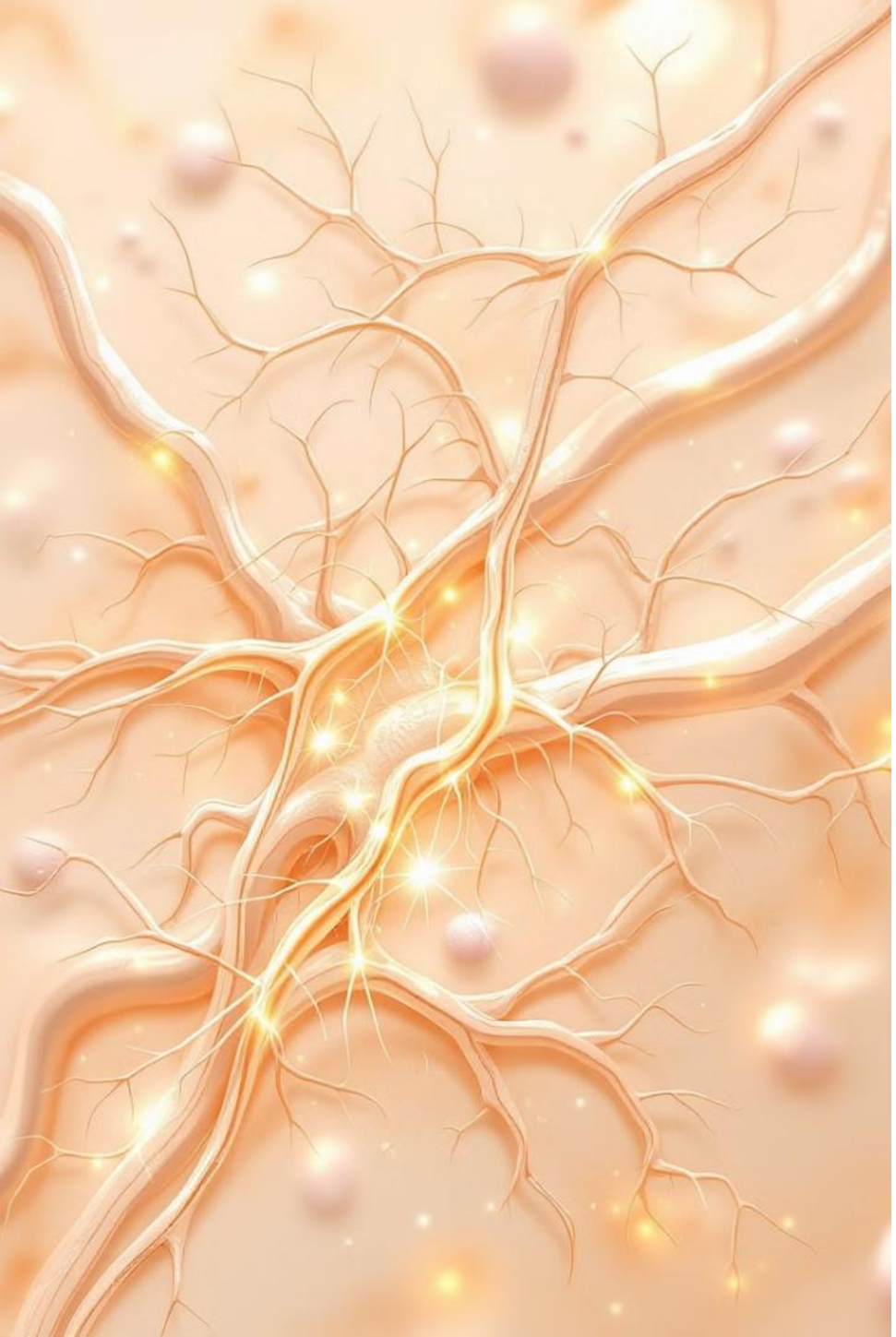
3

Supportive Strategies

Treatment approaches include hydration, diuretics, dialysis, and targeted therapies to address the underlying causes.

Supportive and Therapeutic Strategies

- **Prompt anti-myeloma therapy – (e.g. Velcade based)**
- Acute severe renal injury- Hemodialysis
- High-cutoff hemodialysis and bortezomib-based chemotherapy in multiple myeloma patients with acute kidney injury have a tendency to improve renal outcomes, but more RCTs based on novel drugs are warranted
- Avoid: NSAIDS, nephrotoxic medications, contrast-induced nephropathy
- Dose adjustment of anti-myeloma drugs to kidney function/GFR



Peripheral Neuropathy in Multiple Myeloma: Etiology and Symptom Management

1

Myeloma-Induced

Myeloma cells can directly infiltrate and damage peripheral nerves, leading to neuropathic symptoms.

2

Treatment-Induced

Certain myeloma therapies, such as bortezomib and thalidomide, can also cause peripheral neuropathy.

3

Comprehensive Management

Effective management involves pain medications, neuroprotective agents, and dose adjustments of neurotoxic drugs.

Peripheral Neuropathy

- Common complication in MM
- Two major causes:
  1. Pressure on nerve roots secondary to MM lytic lesions and vertebra collapse
  2. Neurotoxicity of anti myeloma drugs esp. proteosome inhibitors (Velcade , Kyprolis) and Thalidomide.
- Treatment:
  - Orthopedic intervention (Kyphoplasty, disc internal fixation)
  - Vitamins (complex of Vitamin B1, B6, B12)
  - Lyrica or Cymbalta
  - Medical Canabis

Infection in Multiple Myeloma and Therapy

Immunosuppression

Patients with multiple myeloma are at increased risk of infections due to immunosuppression from the disease itself and its treatments.

Impaired Antibody Production

Myeloma can also impair antibody production, leaving patients more susceptible to infections.

Immune System Damage

Chemotherapy treatments can further damage the immune system, increasing the risk of infections.

Respiratory Infections

Common types of infections in myeloma patients include respiratory infections like pneumonia and bronchitis.

Urinary Tract Infections

Urinary tract infections (UTIs) are another common infection in myeloma patients.

Skin Infections

Skin infections are also a concern for myeloma patients.

Bloodstream Infections

Bloodstream infections, such as bacteremia and sepsis, can be life-threatening in myeloma patients.

Antibiotics, Antivirals, and Antifungals

Treatment for infections in myeloma patients may include antibiotics, antivirals, or antifungals to target the specific pathogen.

Supportive Care

Supportive care, such as intravenous fluids and oxygen, may also be necessary.

Adjustments to Myeloma Therapy

Adjustments to myeloma therapy may be needed to reduce immunosuppression and improve infection control.

Intravenous Immunoglobulin (IVIG) Therapy

Intravenous immunoglobulin (IVIG) therapy can boost antibody levels to help fight infections.

Infections

- Infections continue to be one of the main causes of morbidity and mortality in multiple myeloma (MM) patients. Compared to the general population, MM patients have a 7-fold increased risk of bacterial infections and 10-fold risk of viral infections.
- Due to immune deficiency especially in the humoral arm, MM patients are more susceptible to capsulated bacteria infections (e.g. *Strep. Pneumonia).*
- Early and empiric antibiotic treatment is advised in case of fever and infection.
- MM patients receiving proteosome inhibitors (Velcade/ Carfilzomib), Daratumumab and patients receiving autologous SCT are prone to Herpes Zoster reactivation, and should receive prophylaxis (e.g. T. Acyclovir 400mg X2/d)
- PCP prophylaxis after ASCT, Daratumumab/BITE therapy (Trimethoprim-sulfamethoxazole X3/week)

IVIG replacement therapy for the following patients:

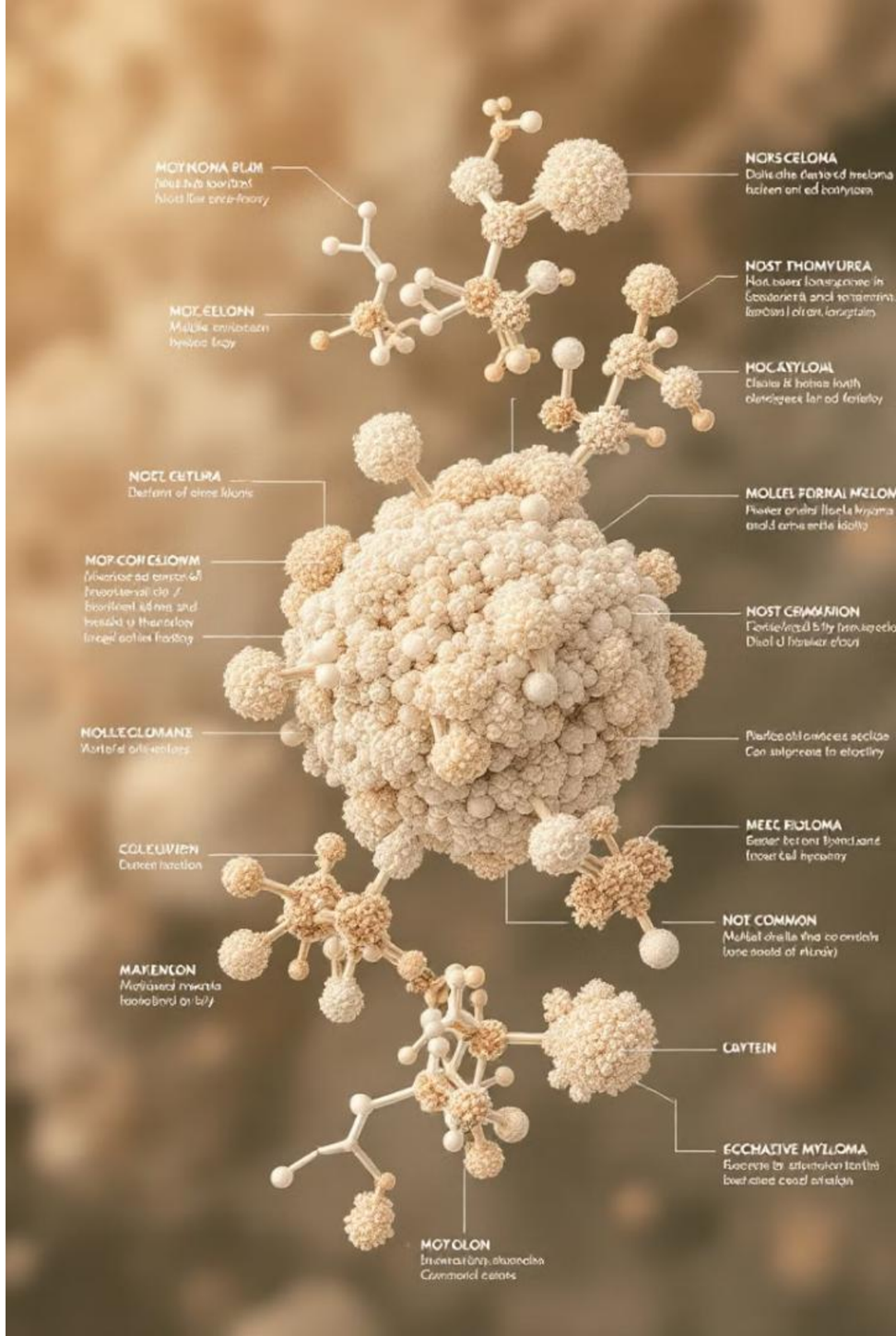
- Patients whose IgG levels <400 mg/dl (level IIC)
- Patients who have experienced ≥2 severe recurrent infections by encapsulated bacteria, regardless of IgG level (level IIC)
- Patients with a life-threatening infection (level III)
- Patients with documented bacterial infection with no or insufficient response to antibiotic therapy (level IIC)

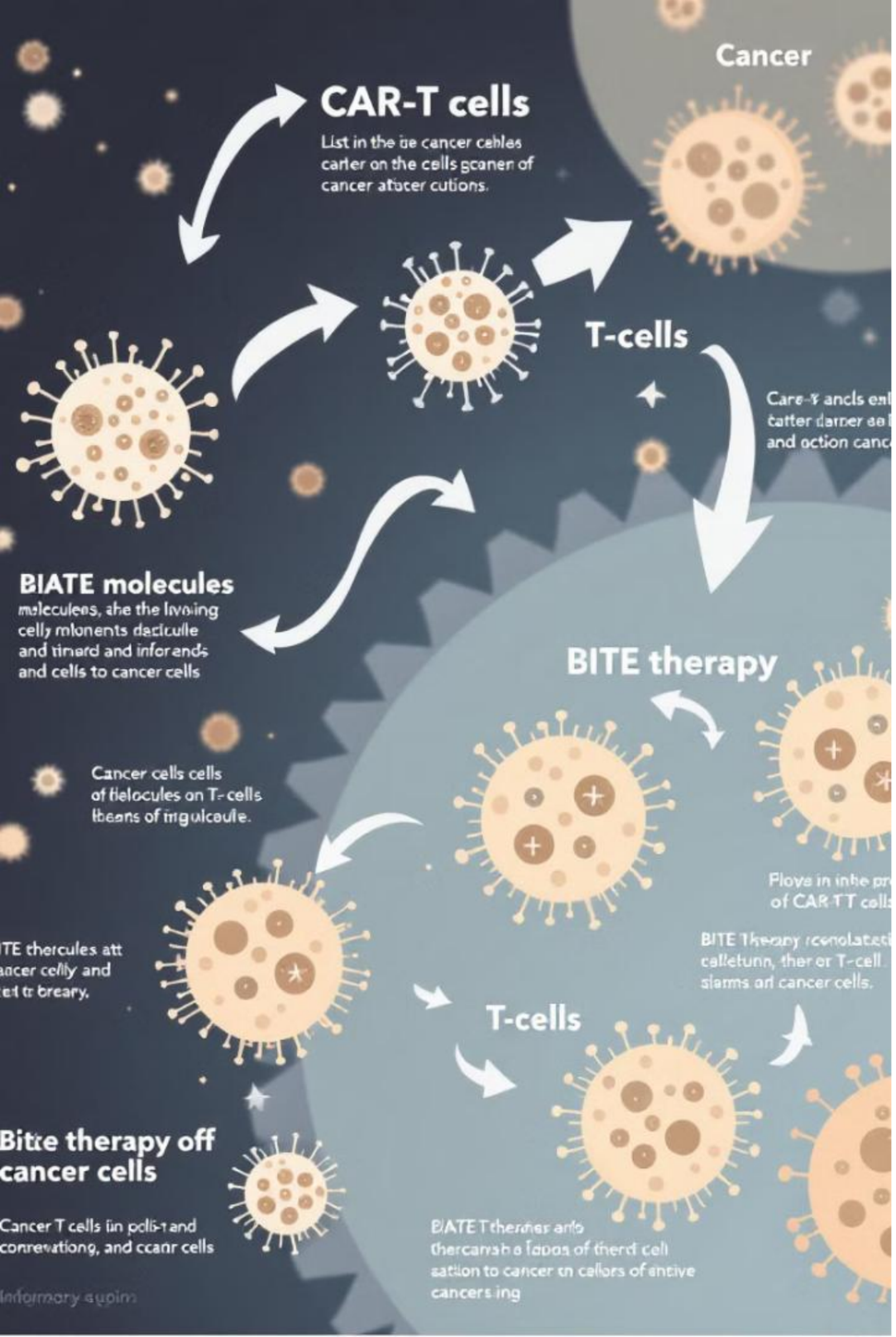
Anemia

- Several causes for anemia in MM:
  - Bone marrow infiltration with plasma cells
  - Secondary to kidney injury
  - Secondary to anti-myeloma drugs
- Treatment accordingly:
  - Iron/Vit B12/ Folic acid supplementation
  - Dose adjustment of drugs related to the anemia (e.g. Lenalidomide)
  - Treat Ac/Chr kidney injury
  - Addition of EPO (e.g. Binocrit)

Medications in Multiple Myeloma: Mechanisms of Action and Safety Profiles

Drug Class	Mechanism of Action	Adverse Events
Proteasome Inhibitors	These drugs target the proteasome, a cellular machinery responsible for degrading proteins. By inhibiting the proteasome, proteasome inhibitors disrupt protein homeostasis in myeloma cells, leading to their death. Commonly used proteasome inhibitors include bortezomib (Velcade), carfilzomib (Kyprolis), and ixazomib (Ninlaro).	Peripheral neuropathy, thrombocytopenia (low platelet count), gastrointestinal toxicity (nausea, vomiting, diarrhea), and fatigue are common side effects.
Immunomodulatory Drugs (IMiDs)	IMiDs, such as thalidomide (Thalomid), lenalidomide (Revlimid), and pomalidomide (Pomalyst), modulate the immune system to target myeloma cells. They act by stimulating the immune system to recognize and destroy myeloma cells, while also inhibiting the growth and survival of these cells.	Common adverse events include thromboembolism (blood clots), fatigue, rash, and birth defects.
Monoclonal Antibodies	These antibodies are specifically designed to target myeloma cells or components of the tumor microenvironment. They can directly kill myeloma cells, block their growth, or enhance the activity of the immune system. Examples include daratumumab (Darzalex) and elotuzumab (Empliciti).	Infusion reactions, infections, neutropenia (low white blood cell count), and fatigue are common side effects.
Anti-CD38 Antibodies	These antibodies specifically target the CD38 protein, which is found on the surface of myeloma cells. By binding to CD38, these antibodies trigger the immune system to attack and destroy myeloma cells. A notable example is daratumumab (Darzalex).	Similar to other monoclonal antibodies, anti-CD38 antibodies can cause infusion reactions, neutropenia, fatigue, and other side effects.





CAR-T and BiTE Therapy: Adverse Events and Management

Cytokine Release Syndrome

Potentially life-threatening hyperinflammatory response requiring close monitoring and targeted interventions.

Neurotoxicity

Neurological complications, such as confusion and seizures, that necessitate prompt diagnosis and management.

Immune Suppression

Increased risk of infections due to profound and prolonged T-cell depletion, requiring prophylaxis.

blood® Editorial

Introduction to a How I Treat series on emergent CAR T-cell toxicities

When the Food and Drug Administration (FDA) approved the first 2 chimeric antigen receptor T-cell therapy (CAR-T) products in 2017, the agency deemed that both required risk evaluation management strategies because of significant toxicities including cytokine release syndrome (CRS) and immune cell–associated neurological syndrome (ICANS). Several articles were published at that time, developing grading systems and standards for administration of immune effector cells and providing guidance on management of these toxicities.[1-5] Since then, 4 additional CAR-T products have been approved by the FDA[6] (Table 1), all with risk evaluation management strategies requirements, and there are many more in clinical development. A significant amount of experience has been obtained from real-world use of these products in which the patient populations may have more comorbidities than those treated in clinical trials.[6-8]

The American Society of Hematology Subcommittee on Emerging Gene and Cell Therapies therefore felt it was timely to develop a How I Treat series focusing on management of complications associated with CAR-T. The articles in this series discuss several types of complications (Figure 1), review the current understanding of etiologies, and provide guidance on therapies.

- Michael D. Jain, Melody Smith, and Nirali N. Shah, "How I treat refractory CRS and ICANS after CAR T-cell therapy"
- Bianca D. Santomasso, Juliane Gust, and Fabiana Perna, "How I treat unique and difficult-to-manage cases of CAR T-cell therapy–associated neurotoxicity"
- Cristina Gutierrez, Tomas G. Neilan, and Natalie S. Grover, "How I approach optimization of patients at risk of cardiac and pulmonary complications after CAR T-cell therapy"
- Tania Jain, Timothy S. Olson, and Frederick L. Locke, "How I treat cytopenias after CAR T-cell therapy"

Although the incidence of severe CRS and ICANS has decreased with broader use of preemptive or earlier interventions, persistent or progressive CRS and ICANS remain

Table 1. Approved CAR-T products in the United States as of March 2023

Generic name	Trade name	Target	Indication
Tisagenlecleucel	Kymriah	CD19	B-cell acute lymphoblastic leukemia among those aged <26 with refractory or multiply relapsed disease. Adult patients with relapsed or refractory large B-cell or follicular lymphoma after ≥2 lines of therapy.
Axicabtagene ciloleucel	Yescarta	CD19	Adult patients with relapsed or refractory large B-cell or follicular lymphoma after ≥2 lines of therapy. Adult patients with large B-cell lymphoma that is refractory to first-line chemoimmunotherapy or that relapses within 12 months of first-line chemoimmunotherapy.
Lisocabtagene maraleucel	Breyanzi	CD19	Adult patients with relapsed or refractory large B-cell lymphoma after ≥2 lines of therapy or that is refractory to first-line therapy or that relapses within 12 mo of first-line therapy.
Brexucabtagene autoleucel	Tecartus	CD19	Adult patients with relapsed or refractory mantle-cell lymphoma. Adult patients with relapsed or refractory B-cell precursor acute lymphoblastic leukemia
Idecabtagene vicleucel	Abecma	BCMA	Relapsed or refractory multiple myeloma after ≥4 prior lines of therapy.
Ciltacabtagene autoleucel	Carvykti	BCMA	Relapsed or refractory multiple myeloma after ≥4 prior lines of therapy.

BCMA, B-cell maturation antigen.

Submitted 6 March 2023; accepted 6 March 2023; prepublished online on *Blood* First Edition 16 March 2023. https://doi.org/10.1182/blood.2023020228.

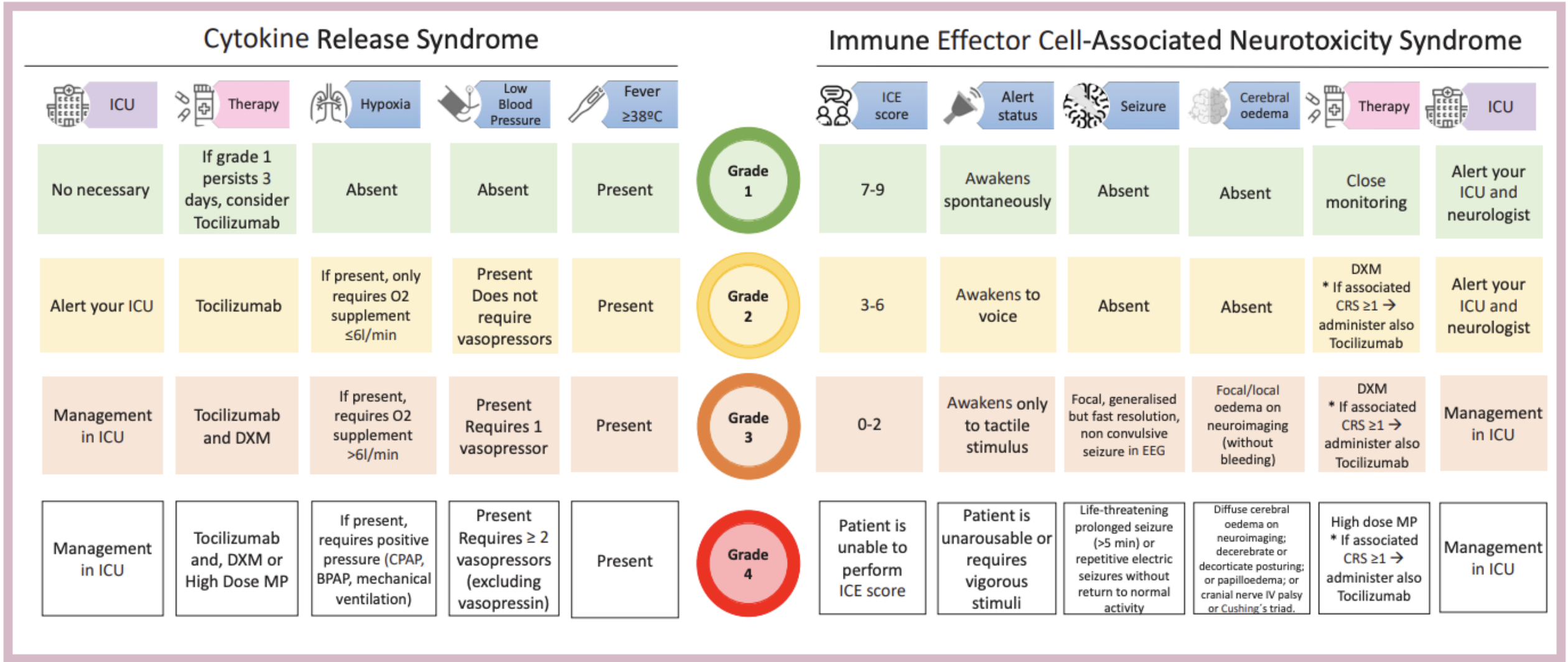
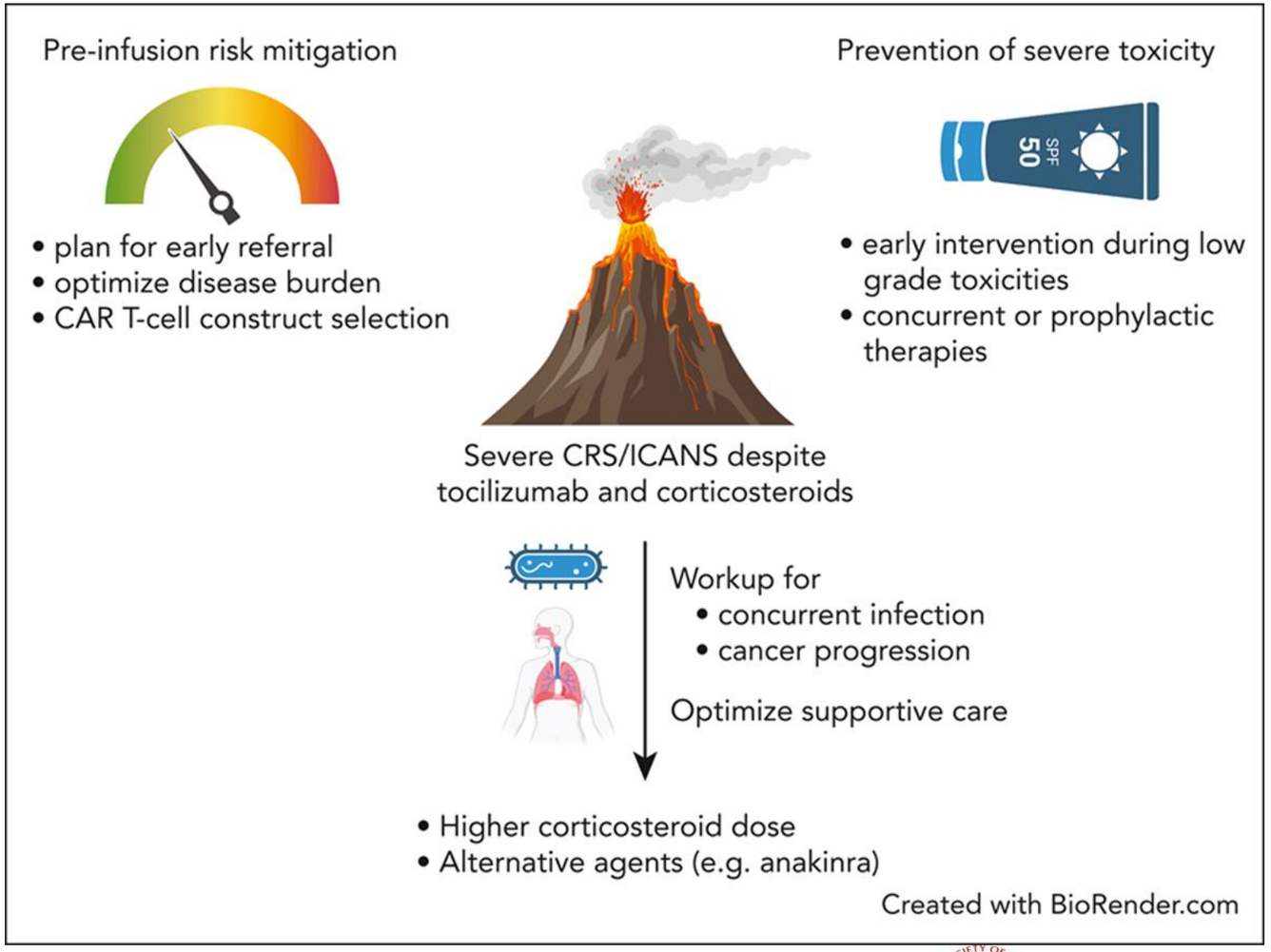


Figure 1 The American Society for Transplantation and Cellular Therapy (ASTCT) consensus grading and recommended management for cytokine release syndrome (CRS) and neurological toxicity associated with immune effector cells (ICANS). DXM, Dexamethasone; ICE, immune effector cell-associated encephalopathy; ICU, intensive care unit; MP, Methylprednisolone.

How I treat refractory CRS and ICANS after CAR T-cell therapy



cancers

MDPI

Review

T-Cell Redirecting Therapies in Multiple Myeloma: Pathogenesis and Management of Toxicities Beyond CRS and ICANS

Katia Mancuso [1,2], Marco Talarico [1,2], Enrica Manzato [1,2], Simona Barbato [1,2], Paola Tacchetti [1], Elena Zamagni [1,2,*] and Michele Cavo [2]

Cancers 2025, 17, 1514

Table 2. Incidence of cytopenias, hypogammaglobulinemia and infections in pivotal trials.

	Ide-Cel		Cilta-Cel		Teclistamab	Elranatamab	Talquetamab	
	KarMMa [1]	KarMMa-3 [2,18]	CARTITUDE-1 [25–27]	CARTITUDE-4 [3,28]	MajesTEC-1 [4,29,30]	MagnetisMM-3 [5]	MonumenTAL-1 [6,31,32]	
							405 µg QW	800 µg Q2W
Neutropenia								
All grade	91%	78%	96%	90%	72%	49%	67%	36%
Grade ≥ 3	89%	76%	95%	90%	65%	49%	60%	32%
Persistent * grade ≥ 3	41%	40%	30%	26%	/	/	/	/
Thrombocytopenia								
All grade	63%	54%	79%	54%	42%	31%	37%	23%
Grade ≥ 3	52%	42%	60%	41%	23%	24%	23%	11%
Persistent * grade ≥ 3	48%	37%	41%	26%	/	/	/	/
Anaemia								
All grade	70%	66%	81%	54%	55%	49%	60%	42%
Grade ≥ 3	60%	51%	68%	36%	38%	37%	30%	23%
Hypogammaglobulinemia								
All grade §	21%	11%	12%	42%	74.5%	75.5%	87%	71%
Infections								
All grade	69%	58%	58%	62%	80%	70%	58%	65%
Grade ≥ 3	22%	24%	20%	27%	55%	40%	22%	16%
Grade 5	UR	4%	4%	6%	13%	6.5%	<1.5%	
IEC-HS								
	3%		1%			No cases reported		

* Persistent: >1 month. § Definition of hypogammaglobulinemia varies across different trials. Abbreviations: QW = every week; Q2W = every other week; UR = unreported; IEC-HS = immune-effector cell-associated hemophagocytic lymphohistiocytosis-like syndrome.

Table 3. Summary of recommendations for cytopenias and hypogammaglobulinemia (references in text).

CAR-T-Cell Recipients	BsAbs Recipients
Anaemia	
Transfusion of packed RBC concentrates as per institutional standards Erythropoiesis stimulating agents if long lasting/severe anemia as per institutional guidelines	Transfusion of packed RBC concentrates if G ≥ 3 or symptomatic patient Erythropoiesis stimulating agents
Thrombocytopenia	
Transfusion of PLT concentrates as per institutional standards Thrombopoiesis stimulating agents if long lasting/severe thrombocytopenia as per institutional guidelines	Transfusion of PLT concentrates if G4 without bleeding or G3 with bleeding Hold BsAb administration until PLT > 50,000
Neutropenia	
Prophylactic G-CSF from day +2 if high risk of severe ICAHT (high HT score) Therapeutic G-CSF if ANC < 500/µL If G-CSF refractory: autologous or allogeneic HCB and TPO-RA If no response: allo-HCT	G-CSF if G ≥ 3 neutropenia (to be avoided when pts are at high risk for CRS) If G4 or febrile neutropenia: BsAb discontinuation until resolution
Hypogammaglobulinemia	
IgRT (400–500 mg/kg every 4 weeks) if serum IgG < 400 mg/dL or if severe and/or recurrent infections	

Abbreviations: CAR-T = chimeric antigen receptor T-cells; BsAbs = bispecific antibodies; RBC = red blood cell; G = grade; PLT = platelet; G-CSF = granulocyte colony-stimulating factor; ICAHT = immune effector cell associated hematotoxicity; HT = CAR-HEMATOTOX; ANC = absolute neutrophil count; HCB = haematopoietic cell boost; TPO-RA = thrombopoietin receptor agonist; allo-HCT = allogeneic haematopoietic stem cell transplantation; CRS = cytokines release syndrome.

Table 4. Summary of recommendations for antimicrobial prophylaxis (references in text).

CAR-T-Cell Recipients	BsAbs Recipients
Herpes prophylaxis	
Aciclovir or valaciclovir: all pts from LD until 1 yr after infusion and/or CD4+ count > 200/μL.	Aciclovir or valaciclovir: all pts until 3 mos off-therapy and CD4+ count > 200/μL.
PJP prophylaxis	
Co-trimoxazole: all pts from LD until 1 yr after infusion and/or CD4+ count > 200/μL.	Co-trimoxazole: all pts throughout treatment period until CD4+ count > 200/μL.
Antibacterial prophylaxis	
Specific drug based upon local bacterial epidemiology: high risk of severe ICAHT and ANC < 500/μL.	Specific drug based upon local bacterial epidemiology: first months of therapy; ANC < 500/μL; prolonged neutropenia; high risk of bacterial infections
Antifungal prophylaxis	
Specific drug based upon individual risk: high risk of severe ICAHT and ANC < 500/μL; high risk of fungal infections (concomitant steroids, prior allo-HCT, prior invasive aspergillosis)	Specific drug based upon individual risk: high risk of fungal infections (prolonged and severe neutropenia, prolonged and/or high dose corticosteroids, prior fungal infection)
CMV prophylaxis	
Not recommended	Not recommended
HBV prophylaxis	
Entecavir or tenofovir: HBV carriers or previous HBV infection	Entecavir or tenofovir: HBV carriers or previous HBV infection
Recommended vaccinations	
- Influenza: ≥2 weeks before LD, booster 3–6 mos after infusion, then annually - SARS-CoV-2: ≥2 weeks before LD, then full vaccination starting 3 mos after infusion - VZV recombinant adjuvanted 2–12 mos after infusion (particularly if seropositive, history of chickenpox or shingles) - HBV > 6 mos after infusion if lacking seroprotection - PCV20: > 6 mos after infusion - DTap > 6 mos after infusion, with 2 DT further doses over 6–12 mos - Hib and meningococcal if additional risk factors (e.g., functional asplenia)	- Influenza (yearly prior to onset of winter) - SARS-CoV-2 - PCV20 - VZV recombinant adjuvanted (particularly if seropositive, history of chickenpox or shingles) - Hib and meningococcal if additional risk factors (e.g., functional asplenia and hypogammaglobulinemia)

Figure 1 Map of GPRC5D protein expression by immunohistochemistry in tissues with GPRC5D-positive gene expression.
Abbreviations: GPRC5D = G protein–coupled receptor family C group 5 member D.

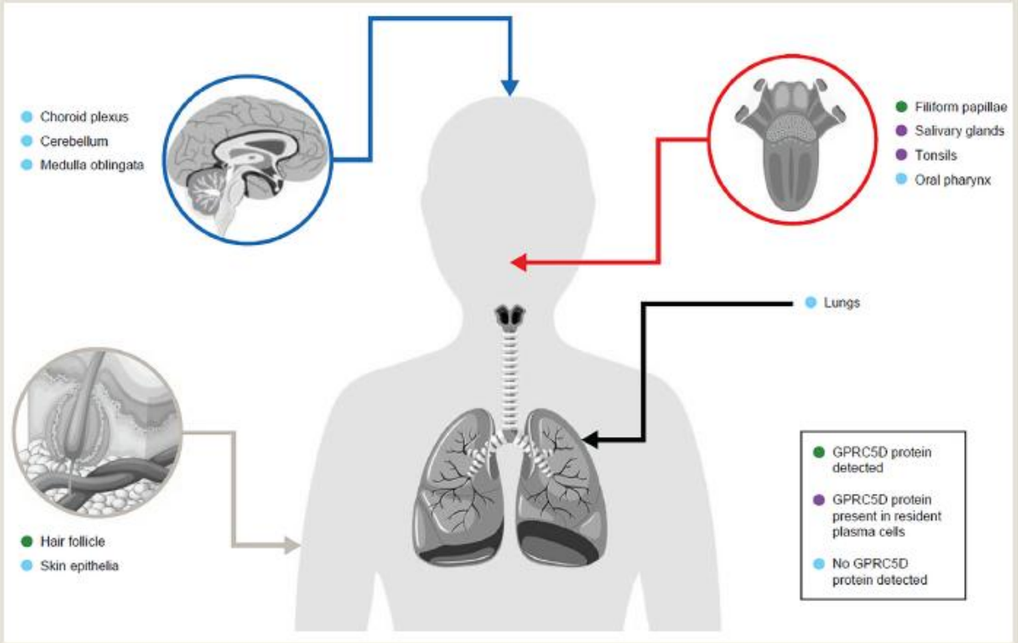
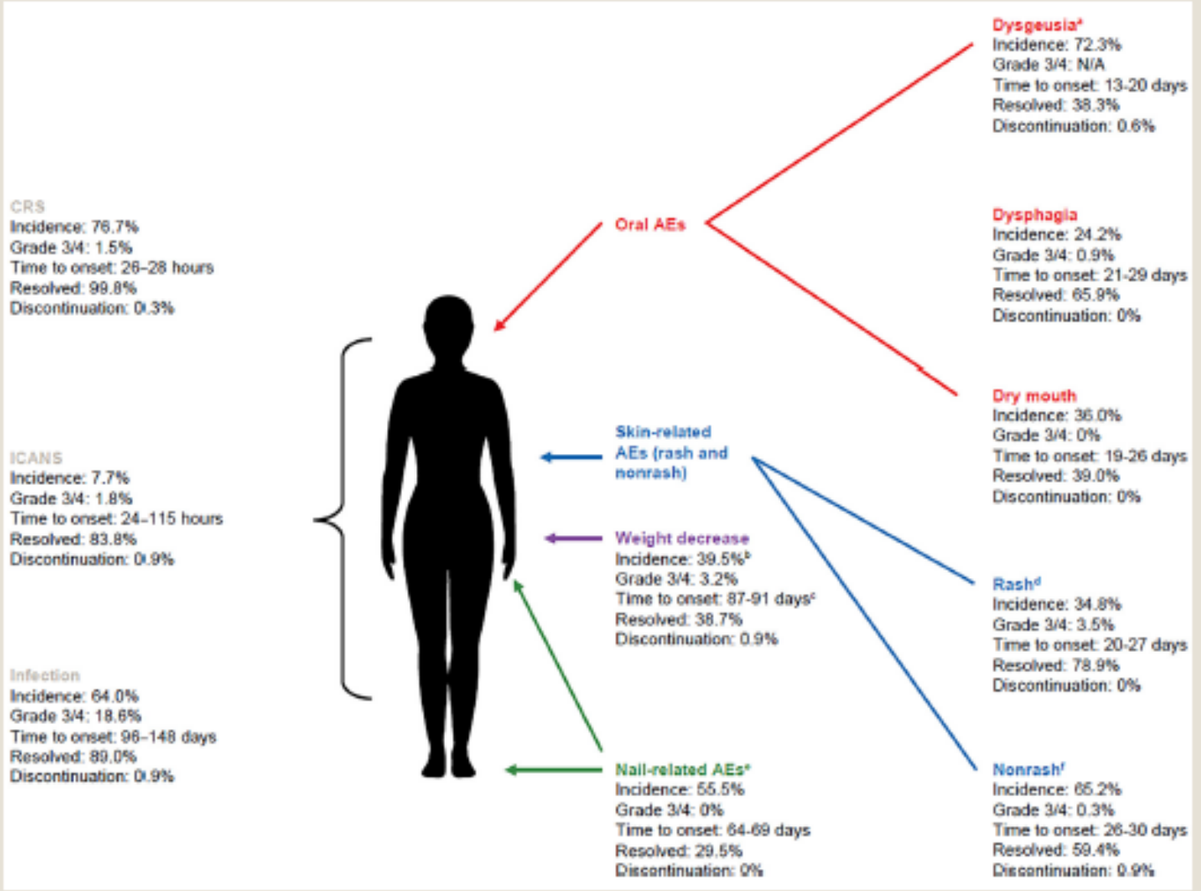
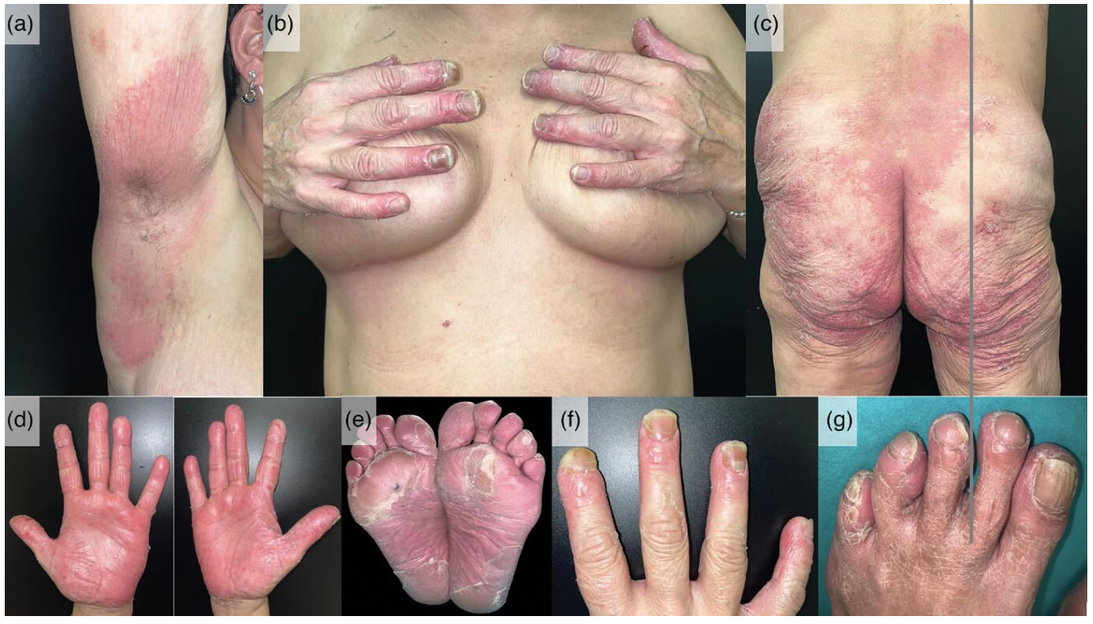


Figure 2 Summary of key AEs associated with talquetamab.
Abbreviations: AE = adverse event; CRS = cytokine release syndrome; ICANS = immune effector cell-associated neurotoxicity syndrome; QW = weekly; Q2W = every other week.
[a]Includes dysgeusia, ageusia, hypogeusia, and general taste disorders.
[b]The number of patients with a ≥ 10% decrease in weight from baseline in the 0.4 mg/kg QW, 0.8 mg/kg Q2W, and prior T-cell redirection cohorts was 37.1%, 32.4%, and 29.4%, respectively.
[c]Time to onset for weight loss is reported for patients with a ≥ 10% decrease in weight from baseline.
[d]Includes rash, maculopapular rash, erythematous rash, and erythema.
[e]Includes nail discoloration, nail disorder, onycholysis, onychomadesis, onychoclasis, nail dystrophy, nail toxicity, and nail ridging.
[f]Includes skin exfoliation, dry skin, pruritus, and palmar-plantar erythrodysesthesia syndrome.





Adverse Event	Time to Onset (days)	Time to Resolution (days)	Management
Xerostomia (dry mouth)	19–26	57–89	- Hydration - Artificial saliva (saliva substitutes, sialogogues) - Sugar free chewing gums, hard candies, ice chips - Limit intake of caffeine and alcohol.
Dysgeusia	13–20	95–130	- Baking soda mouth rinses - Experiment with food of - Different textures and tastes - Enhance the taste of food with addition of spicy, sour or other aromatic flavor additives
Dysphagia	21–29	73–174	- Small bites (sips of liquid between bites) - Frequent small meals - Avoid dry food - Soft or mashed food - Sitting upright when eating
Nail-related (discoloration, onycholysis, dystrophy)	64–69	74–122	- Emollients - Nail hardeners - Vitamin E oil - Biotin supplements
Skin-related - Rash - Non-rash (dry skin, pruritus, skin exfoliation)	20–27 26-30	15–28 32-39	- Hydration - Emollients - Topical/oral antihistamines and/or steroids.
Weight loss (≥10% decrease from baseline)	87–91	50–403	- Nutrient dense food - Calorie boosting food - Appetite stimulants

Conclusion: Optimizing Supportive Care for Improved Patient Outcomes

1 Comprehensive Approach

Effective supportive care requires a multidisciplinary team to address the diverse complications of multiple myeloma.

2 Personalized Management

Tailoring supportive interventions to individual patient needs and disease characteristics is key to optimizing outcomes.

3 Ongoing Monitoring

Vigilant monitoring and early intervention are crucial for preventing and managing adverse events.