

# Multiple Myeloma

One clone of plasma cells has broken free and is flooding the blood with one antibody (the M-protein) while crowding out healthy blood cells, dissolving bone, and clogging the kidneys.

## THE STORY IN ONE SENTENCE

One clone of plasma cells has broken free and is flooding the blood with one antibody (the M-protein) while crowding out healthy blood cells, dissolving bone, and clogging the kidneys.

“Multiple myeloma is one type of plasma cell (an antibody-making white blood cell) taking over the bone marrow, breaking down bone, and squeezing out normal blood production.”

## Normal anatomy — the body's job

Plasma cells live in the bone marrow and make many different antibodies, each shaped to grab a different germ. The marrow balances the birth of red blood cells (oxygen carriers), white blood cells (infection fighters), and platelets (clotting helpers). At the same time, bone is constantly remodeled: osteoblasts build new bone, and osteoclasts break down old bone to keep it strong and fresh. Picture that balanced system—because every weird finding in myeloma is a change from this normal job.

## BODY'S JOB

Plasma cells are antibody factories. Each healthy plasma cell makes one type of antibody against one germ, and together thousands of different plasma cells cover thousands of germs. The marrow keeps red cells, white cells, and platelets in balance. Bone builders and bone breakers work together to keep bone strong.

1. A germ (bacteria or virus) enters the body.
2. B cells (a type of white blood cell) meet the germ and learn its shape.
3. The B cell that matches the germ becomes a plasma cell.
4. That plasma cell makes antibodies shaped to grab that one germ.
5. Thousands of different plasma cells make thousands of different antibodies, covering the whole germ world.

6. The marrow also makes red blood cells (to carry oxygen), neutrophils (to eat bacteria), and platelets (to clot blood)—all at once, in balance.
7. Osteoblasts (builder cells) lay down new bone; osteoclasts (breaker cells) dissolve old bone; the cycle keeps bone strong and repairs tiny cracks.
8. Healthy plasma cells live a few days, make their antibodies, and then die—making room for new ones.

## What breaks

---

One plasma cell breaks the rules, copies itself endlessly, and takes over the marrow. It makes one antibody (the M-protein) in huge amounts, signals osteoclasts to dissolve bone, and crowds out red cells, white cells, and platelets.

### CLINICAL TERM

A single plasma cell undergoes a malignant transformation (acquires mutations that let it ignore normal growth limits). The clone expands unchecked in the bone marrow, secretes a monoclonal immunoglobulin (M-protein) and free light chains, and secretes cytokines—especially RANKL (receptor activator of nuclear factor kappa-B ligand)—that activate osteoclasts and suppress osteoblasts. The expanding tumor burden crowds normal hematopoietic precursors, causing cytopenias (low blood counts). Light chains are filtered by the kidneys and can precipitate in the renal tubules, causing cast nephropathy (myeloma kidney). The break is upstream: one plasma cell that will not stop dividing.

## How the body compensates

---

The body tries to keep oxygen delivery, blood pressure, and calcium balance steady even as the marrow fills with cancer, bone dissolves, and the kidneys fail. Vital signs can look normal for a while—that hidden middle is the most dangerous time, because the team may not see the cliff ahead. Watch the trend, not just one snapshot.

1. {'what': 'The heart beats faster and harder to deliver oxygen when red blood cells drop (anemia).', 'why': 'Less red cells mean less oxygen per heartbeat, so the heart pumps faster (tachycardia) and harder (increased stroke volume) to keep oxygen delivery up.', 'limit': 'Once hemoglobin drops below ~7 g/dL or the heart cannot keep up (especially in older patients or those with heart disease), fatigue, shortness of breath, and heart failure appear.'}

2. {'what': 'The kidneys try to hold onto water and salt to keep blood pressure up when the patient is dehydrated from high calcium (hypercalcemia).', 'why': 'High calcium (from dissolving bone) makes the kidneys lose water (calcium blocks the kidney's ability to concentrate urine), leading to dehydration; the kidneys respond by activating the renin-angiotensin-aldosterone system to hold onto salt and water.', 'limit': 'If calcium keeps rising or light chain casts block the tubules, the kidneys fail (acute kidney injury), and the body cannot balance fluids, potassium, or acid anymore.'}
  3. {'what': 'The bones try to repair lytic lesions, but osteoblasts are blocked by myeloma signals.', 'why': 'Normally osteoblasts would fill in holes, but myeloma cells secrete DKK1 (Dickkopf-1), which shuts down osteoblast activity—so bone dissolves but is not rebuilt.', 'limit': 'This leads to pathologic fractures (bones breaking from normal activity or minor bumps), especially in the spine (vertebral compression fractures), ribs, and long bones.'}
  4. {'what': 'The body tries to buffer high calcium by moving it into cells and excreting it in urine.', 'why': 'High serum calcium is toxic to the heart, brain, and kidneys; the body tries to lower it by shifting calcium into bone and muscle and increasing kidney excretion.', 'limit': 'Once calcium rises above ~12 mg/dL, confusion, weakness, constipation, and life-threatening arrhythmias (heart rhythm problems) appear (hypercalcemic crisis).'}
5. {'what': 'The immune system tries to fight infections with fewer tools.', 'why': 'Normal plasma cells are suppressed, so the body has fewer antibodies; neutrophils (bacteria-eating white cells) are also low from marrow crowding.', 'limit': 'Recurrent infections—especially pneumonia (*Streptococcus pneumoniae*, *Haemophilus influenzae*) and urinary tract infections—become common and life-threatening.'}
  6. {'what': 'Vital signs (blood pressure, heart rate, oxygen saturation) can stay normal even as the marrow fills, bones dissolve, and kidneys fail—this is the dangerous middle.', 'why': 'Compensation buys time, but it hides how sick the patient really is. A stable blood pressure today does not mean the patient is stable if calcium is rising, creatinine is climbing, or hemoglobin is falling.', 'limit': 'Once compensation fails, the crash is sudden: hypercalcemic crisis, acute kidney injury requiring dialysis, spinal cord compression from vertebral fracture, or sepsis. Watch the trend—calcium climbing from 10 to 11 to 12, creatinine rising from 1.0 to 1.5 to 2.5, new back pain—and act before the cliff.'}

## Domino effect

---

1. One plasma cell breaks free and copies itself endlessly in the bone marrow. — Genetic mutations (such as t(4;14), t(14;16), deletion 17p, or gain of chromosome 1q) let one plasma cell ignore normal stop signals and divide without limit—this is the single upstream break that starts everything.

2. The malignant clone floods the blood with one antibody (the M-protein) and free light chains. — Unlike healthy plasma cells that make many different antibodies, the myeloma clone makes only one type (IgG, IgA, or light chain only) in huge amounts—this monoclonal protein shows up as an M-spike on serum protein electrophoresis and free light chains in urine (Bence Jones protein).
3. The clone secretes signals (RANKL and DKK1) that activate osteoclasts and block osteoblasts, dissolving bone. — RANKL tells osteoclasts (bone-eating cells) to work overtime; DKK1 blocks osteoblasts (bone-building cells)—so bone is dissolved but not rebuilt, leaving punched-out lytic lesions (visible as holes on X-ray) and releasing calcium into the blood (hypercalcemia).
4. Bones hurt and break from normal activity or minor bumps (pathologic fractures). — Dissolved bone is weak and full of holes; even small stress—lifting a bag, coughing, rolling over in bed—can break ribs, vertebrae (causing back pain and risk of spinal cord compression), or long bones.
5. Calcium rises in the blood (hypercalcemia), causing confusion, weakness, constipation, and kidney damage. — Dissolved bone dumps calcium into the bloodstream; high calcium slows the heart and brain (causing arrhythmias, confusion, and coma) and makes the kidneys lose water (leading to dehydration and worsening kidney injury).
6. The expanding plasma cell mass crowds the marrow, squeezing out red cell, white cell, and platelet production. — The marrow has limited space; as the myeloma clone fills it, there is less room for normal blood cell precursors—red blood cells drop (anemia, causing fatigue and shortness of breath), neutrophils drop (raising infection risk), and platelets drop (raising bleeding risk, though severe bleeding is less common).
7. Free light chains (pieces of the M-protein) are filtered by the kidneys and jam the renal tubules, causing cast nephropathy (myeloma kidney). — Light chains pass through the glomerulus (the kidney's filter), travel down the tubule, and bind to Tamm-Horsfall protein, forming dense casts that block the tubule, trigger inflammation, and scar the kidney—leading to acute kidney injury and, if untreated, permanent kidney failure.
8. The immune system weakens, and infections become frequent and severe. — The myeloma clone suppresses normal plasma cells (so the body cannot make a full range of antibodies), and marrow crowding lowers neutrophils—leaving the patient vulnerable to bacterial infections, especially pneumonia (*Streptococcus pneumoniae*, *Haemophilus influenzae*) and urinary tract infections.

## Symptoms · what the patient feels, and why

---

- Bone pain, especially in the back or ribs: The cancer cells crowd into the bone marrow and release chemicals (cytokines like IL-6 and TNF- $\alpha$ ) that wake up osteoclasts (the cells that break down bone). These osteoclasts chew away bone faster than the body can rebuild it, leaving weak spots that hurt and can crack easily.
- Getting tired easily and looking pale (anemia): The myeloma cells pack the bone marrow so tightly that there's no room left for the stem cells that normally make red blood cells. Fewer red blood cells means less oxygen reaches your muscles and brain, so you feel worn out and your skin looks pale.
- Breaking a bone from a tiny bump or fall (pathologic fracture): Because the osteoclasts have eaten away so much bone, the skeleton becomes brittle—like a cracker instead of a strong beam. Even a small knock or lifting something light can snap a rib or crack a vertebra in the spine.
- Getting infections often, like pneumonia or urinary tract infections: Myeloma cells are abnormal plasma cells that crowd out the healthy plasma cells, which are the body's antibody factories. Without enough normal antibodies (immunoglobulins), your immune system can't tag and kill germs as well, so bacteria and viruses take hold more easily.
- Feeling confused, very thirsty, or urinating a lot (high calcium): When osteoclasts break down bone, calcium floods into the bloodstream (hypercalcemia). High calcium slows down nerves and muscles, making you feel foggy or weak, and it also makes your kidneys dump extra water into urine to try to wash the calcium out, leaving you thirsty and peeing constantly.
- Foamy urine or swelling in the legs (kidney trouble): The myeloma cells dump huge amounts of abnormal antibody pieces (light chains, also called Bence Jones protein) into the blood. These light chains clog the tiny filters in the kidneys (the tubules), damaging them so they leak protein into the urine (making it foamy) and can't balance fluids well (causing swelling).
- Nosebleeds or easy bruising: The bone marrow is so packed with myeloma cells that it can't make enough platelets (the sticky blood cells that plug leaks). With fewer platelets, even a small scratch or bump can bleed longer than normal.
- High total protein on a blood test, but albumin is normal: Myeloma cells churn out massive amounts of one kind of abnormal antibody (a monoclonal immunoglobulin, or M protein). That flood of protein makes the total protein number climb, but albumin—the liver's normal protein—stays steady, creating a telltale gap that doctors spot on the lab report.

## Focused exam

---

- Back pain or rib pain that does not go away, gets worse when you press on it or move: The myeloma plasma cells tell osteoclasts (bone-breaking cells) to chew up bone faster than osteoblasts (bone-building cells) can fix it. Pain marks the spot where bone is dissolving. This is not muscle soreness — it points straight to the bone problem.
- Pale skin, pale nail beds, and feeling tired even after rest: The marrow is packed with myeloma plasma cells, so it cannot make enough red blood cells. Fewer red cells means less oxygen reaches the body. Paleness and tiredness are the body showing that oxygen delivery is falling.
- Bone breaks from a light bump, a sneeze, or even a cough (called a pathologic fracture): Osteoclasts have dissolved so much bone that it snaps under normal stress. A fracture from almost nothing is multiple myeloma showing how weak the bones have become.
- Swollen legs or puffiness around the eyes, or dark urine: The M-protein and light chains made by myeloma plasma cells clog and damage the kidney tubes. Swelling and dark urine mean the kidneys are failing. Rising creatinine is a danger sign in multiple myeloma.
- Confusion, extreme thirst, or nausea that does not go away: Dissolved bone releases calcium into the blood. High calcium (hypercalcemia) confuses the brain, makes the person very thirsty, and causes nausea. In multiple myeloma, the calcium comes from bone breaking down, not from diet or the parathyroid.
- Fever or signs of infection (cough, chills, pain when urinating) in an older patient: The myeloma plasma cells make only one antibody (the M-protein) and crowd out the cells that make all the other antibodies. The patient's immune system cannot fight off common germs. Infection risk climbs. In multiple myeloma, watch for sepsis.

## Diagnostics · every test answers a question

---

- Serum protein electrophoresis (SPEP) and immunofixation → Is there a large spike of one type of antibody (M-protein) in the blood?
- Serum free light chains (kappa and lambda) and the kappa/lambda ratio → Are free light chains out of balance, showing one plasma cell type is overproducing?
- Bone marrow biopsy with flow cytometry and cytogenetics → Are plasma cells taking over the marrow, and do they carry high-risk genetic changes?
- Complete blood count (CBC) → Is the marrow so crowded with myeloma cells that normal blood cells cannot be made?
- Serum creatinine and estimated glomerular filtration rate (eGFR) → Are the kidneys being damaged by light chains and M-protein?

- Serum calcium (corrected for albumin) → Is bone dissolving so fast that calcium is flooding the blood?
- Skeletal survey (X-rays of skull, spine, ribs, pelvis, long bones) or whole-body low-dose CT or PET-CT → Are there holes in the bones (lytic lesions) or fractures from bone breakdown?
- Beta-2 microglobulin and albumin (for staging) → How much myeloma burden is in the body, and what is the prognosis?

## Treatment logic

---

- Triplet induction chemotherapy (proteasome inhibitor + immunomodulatory drug + corticosteroid, e.g. bortezomib + lenalidomide + dexamethasone, called VRd) → Attacks the myeloma plasma cells three ways at once. The proteasome inhibitor (bortezomib) stops the cells from breaking down old proteins, so trash piles up inside and the cell dies. The immunomodulatory drug (lenalidomide) wakes up the immune system's T cells and natural killer cells to recognize and kill myeloma cells, and also stops new blood vessels from feeding the tumor. The corticosteroid (dexamethasone) directly kills plasma cells and calms inflammation. Together, they shrink the myeloma clone so normal marrow cells can grow back, red blood cells rise, and symptoms ease.
- Bisphosphonates (zoledronic acid 4 mg IV every 3–4 weeks, or pamidronate) → Blocks osteoclasts (bone-breaking cells) from dissolving more bone. It poisons the osteoclasts so they cannot respond to the myeloma's bone-breaking signals. This stops new lytic lesions from forming, lowers the risk of fractures, and reduces bone pain. Also lowers high calcium by stopping calcium release from bone.
- Aggressive IV hydration (normal saline, often 200–500 mL/hour if the heart can handle it) and avoid nephrotoxic drugs (NSAIDs, contrast dye when possible) → Dilutes the M-protein and light chains in the blood so they are less likely to clog the kidney tubes. Keeps urine flowing fast to flush light chains out before they can settle and cause damage. Also helps lower high calcium by flushing it out through the kidneys.
- Autologous stem cell transplant (after 3–4 cycles of induction therapy, in patients younger than 65–70 and fit enough) → High-dose melphalan (a chemotherapy drug) wipes out the marrow, including the myeloma plasma cells. Then the patient's own stem cells (collected before the big chemo dose) are put back in to rebuild healthy marrow. This pushes the myeloma deeper into remission and makes people live longer without the cancer coming back.
- Maintenance therapy after transplant or induction (lenalidomide, continued for years) → Keeps the immune system turned on against any leftover myeloma cells. By blocking new blood vessel growth and keeping T cells active, it holds the myeloma in check longer and delays relapse. Extends the time people live without the cancer growing back.

- Supportive care: red blood cell transfusions (if hemoglobin < 7–8 g/dL and symptomatic), erythropoiesis-stimulating agents (if appropriate), infection prevention (vaccinations for pneumococcus, influenza, COVID-19; avoid live vaccines; preventive (prophylactic) antibiotics or antivirals if risk is high) → Transfusions raise oxygen delivery quickly when anemia is severe and the patient feels dizzy or short of breath. Erythropoiesis-stimulating agents (like epoetin alfa) can help the marrow make more red cells if the marrow still has room and the patient is not getting transfusions often. Vaccinations and preventive (prophylactic) antimicrobials lower the risk of pneumonia, shingles, and other infections because the myeloma has wiped out normal antibody-making cells.
- Radiation therapy to a painful bone lesion or a fracture site → Kills myeloma plasma cells in one small area of bone. Stops the pain quickly (often within days) and can strengthen the bone enough to prevent a fracture or help one heal. Useful when one spot is causing urgent trouble, like pressing on the spinal cord.

## Medications

---

### **Bortezomib (Velcade) · Proteasome inhibitor**

Bortezomib blocks the proteasome (the cell's trash compactor that breaks down old proteins). When myeloma cells can't get rid of their protein junk, the trash piles up inside them until they die. Normal cells handle this stress better than cancer cells, so myeloma cells die off more.

1. Bortezomib enters the myeloma cell.
2. It locks onto the proteasome and blocks its cutting blades.
3. Misfolded and damaged proteins pile up inside the cell.
4. The cell's alarm systems trigger cell death (apoptosis).
5. The myeloma cell dies and the tumor shrinks.

#### **SAFETY WATCH**

Nerve damage (peripheral neuropathy): tingling, numbness, or burning pain in hands and feet—usually reversible if caught early · Low platelet count: higher bleeding or bruising risk · Shingles reactivation: antiviral prevention (prophylaxis) (acyclovir or valacyclovir) is often given to prevent this · Fatigue and low blood counts

### **Lenalidomide (Revlimid) · Immunomodulatory drug (IMiD)**

Lenalidomide has three main jobs. It wakes up the immune system's T cells (infection fighters) and natural killer cells so they attack myeloma cells. It also blocks new blood vessels from

feeding the tumor. Finally, it directly damages the myeloma cells' DNA and protein-making machinery so they can't grow.

1. Lenalidomide binds to cereblon (a protein inside cells that tags other proteins for destruction).
2. This causes myeloma cells to destroy two key growth proteins (Ikaros and Aiolos).
3. Without those proteins, myeloma cells stop dividing and die.
4. At the same time, T cells and NK cells become more active and start killing myeloma cells.
5. New blood vessel growth slows, starving the tumor of nutrients.

#### **SAFETY WATCH**

Blood clots (deep vein clot formation (thrombosis) or pulmonary embolism (a clot lodging in a lung artery)): patients often take aspirin or a blood thinner to prevent clots · Low blood counts (white cells, red cells, platelets): regular blood tests are needed · Birth defects: this drug is a cousin of thalidomide and causes severe birth defects—patients must use reliable birth control and follow strict pregnancy-prevention rules · Increased infection risk · Rash, diarrhea, fatigue

### **Dexamethasone · Corticosteroid**

Dexamethasone is a strong steroid that kills myeloma cells directly and calms down inflammation. It slips into myeloma cells, turns on genes that trigger cell death, and shuts down survival signals. It also quiets the immune overreaction that makes patients feel sick.

1. Dexamethasone enters the myeloma cell.
2. It binds to glucocorticoid receptors (steroid switches) inside the cell.
3. These receptors move into the cell's control center (nucleus) and flip genetic switches.
4. Survival genes turn off and death genes (like caspases) turn on.
5. The myeloma cell shrinks and dies (apoptosis).

#### **SAFETY WATCH**

High blood sugar: can unmask or worsen diabetes · Mood swings, trouble sleeping, or feeling 'wired' · Increased appetite and weight gain · Weak bones with long-term use (osteoporosis) · Higher infection risk because it dampens the immune system · Stomach irritation: often given with a stomach protector · Muscle weakness

### **Daratumumab (Darzalex) · Monoclonal antibody (anti-CD38)**

Daratumumab is a lab-made antibody that sticks to CD38 (a protein badge on the surface of myeloma cells). Once it locks on, it marks the cancer cell for destruction. The body's immune

system sees the tag and kills the myeloma cell in three ways: direct cell death, calling in immune attack cells, and punching holes in the cancer cell's wall.

1. Daratumumab binds tightly to CD38 on the myeloma cell surface.
2. The antibody flags the cell for attack by the immune system.
3. Natural killer (NK) cells and macrophages (scavenger immune cells) recognize the tag and kill the myeloma cell.
4. Complement proteins (a cascade of blood proteins) punch holes in the myeloma cell membrane, causing it to burst.
5. The myeloma cell dies and the tumor shrinks.

#### **SAFETY WATCH**

Infusion reactions (especially the first dose): fever, chills, cough, shortness of breath, throat tightness—patients get pre-medications (antihistamines, acetaminophen, steroids) to prevent this · Low blood counts (especially neutrophils and platelets) · Increased infections, especially respiratory infections · Interferes with blood type testing: tell the blood bank you are on daratumumab before transfusions · Fatigue, nausea, back pain

### **Carfilzomib (Kyprolis) · Proteasome inhibitor (second-generation)**

Carfilzomib blocks the proteasome (the cell's protein trash compactor) more tightly than bortezomib and does not let go. Myeloma cells drown in their own protein junk and die. Because it binds irreversibly (permanently), it may work even when bortezomib stops working.

1. Carfilzomib enters the myeloma cell.
2. It binds irreversibly to the proteasome's active site, shutting it down permanently.
3. Damaged and misfolded proteins pile up inside the cell.
4. The cell's stress alarms trigger programmed cell death (apoptosis).
5. The myeloma cell dies and the tumor shrinks.

#### **SAFETY WATCH**

Heart problems: high blood pressure, heart failure, or rarely heart attack—monitor blood pressure and watch for shortness of breath or swelling · Kidney damage: patients need good hydration before and after infusions · Low platelet count: bleeding or bruising risk · Infusion reactions: fever, chills, shortness of breath · Less nerve damage than bortezomib, but still possible · Fatigue

## **Zoledronic acid (Reclast, Zometa) · Bisphosphonate**

Zoledronic acid slows down osteoclasts (the cells that chew up bone). In myeloma, the cancer cells tell osteoclasts to dig too many holes in the bone. Zoledronic acid poisons the osteoclasts so they stop breaking down bone, which prevents fractures, eases bone pain, and lowers dangerous calcium levels in the blood.

1. Zoledronic acid is given by IV and travels through the blood to bones.
2. It sticks to bone mineral in areas where bone is being broken down.
3. Osteoclasts eat the bone mineral and take in the drug.
4. Inside the osteoclast, zoledronic acid blocks an enzyme (farnesyl pyrophosphate synthase) needed to build the cell skeleton.
5. The osteoclast dies and bone breakdown slows, reducing fractures and lowering blood calcium.

### **SAFETY WATCH**

Kidney damage: check kidney function before every dose and keep patients well hydrated · Low calcium, magnesium, or phosphate: supplement calcium and vitamin D · Jaw bone death (osteonecrosis of the jaw): see a dentist before starting and avoid tooth extractions or invasive dental work while on the drug · Flu-like symptoms after infusion: fever, muscle aches, chills—usually improve after the first dose · Atypical fractures with very long-term use (rare)

## **Pomalidomide (Pomalyst) · Immunomodulatory drug (IMiD)**

Pomalidomide is a stronger cousin of lenalidomide. It wakes up the immune system (especially T cells and NK cells) to attack myeloma, blocks new blood vessels from feeding the tumor, and directly kills myeloma cells by destroying key growth proteins inside them.

1. Pomalidomide binds to cereblon (a protein tagging machine inside cells).
2. This causes the cell to destroy Ikaros and Aiolos (two proteins myeloma cells need to survive).
3. Without those proteins, myeloma cells stop dividing and die.
4. T cells and NK cells become more active and kill myeloma cells.
5. New blood vessel growth slows, starving the tumor.

**SAFETY WATCH**

Blood clots: patients often take aspirin or a blood thinner · Low blood counts (especially neutrophils): regular blood tests needed · Severe birth defects: strict pregnancy prevention required (same as lenalidomide) · Increased infections · Fatigue, rash, constipation, nerve damage (less common than with lenalidomide)

## Red flags

---

- New fracture or severe bone pain in the spine (could mean the vertebra is collapsing and pressing on the spinal cord)
- Sudden confusion, extreme thirst, or barely urinating (signs of dangerously high calcium or failing kidneys)
- Fever plus shaking chills in someone with known myeloma (weakened immune system makes infections life-threatening fast)
- Numbness, tingling, or weakness in the legs (could mean spinal cord compression from a collapsed vertebra)

## Nursing priorities

---

1. Run IV normal saline at 200 to 300 mL per hour to keep urine output above 100 mL per hour — Aggressive hydration flushes light chains and calcium out through kidneys before they cause permanent tubular damage (blocked kidney filters); dehydration is the top cause of acute kidney injury in myeloma
2. Hold all NSAIDs like ibuprofen and all contrast dye procedures until renal function is checked — Nephrotoxic drugs (medicines that poison kidneys) plus light chains cause sudden irreversible kidney failure; even one dose of ibuprofen or CT contrast can push borderline kidneys into dialysis need
3. Check calcium level immediately if patient has confusion, severe constipation, or drowsiness — High calcium (Hypercalcemia) from bone breakdown causes life-threatening cardiac arrhythmias (irregular heartbeats) and coma; calcium above 12 mg/dL needs emergency treatment with IV fluids, calcitonin, and bisphosphonates within one hour
4. Use gentle handling during all care and log-roll for position changes; get x-ray before moving patient with new pain — Lytic bone lesions leave bones fragile as eggshells; rough turning or lifting can snap ribs, spine, or long bones causing pathologic fractures, spinal cord compression, and permanent paralysis

5. Monitor temperature every 4 hours and start neutropenic fever protocol immediately for temp above 100.4 F — Immunoparesis means patients cannot fight bacteria; fever in myeloma is infection until proven otherwise and needs blood cultures and broad-spectrum antibiotics like cefepime within one hour to prevent septic shock
6. Teach patient to report any new back pain, leg weakness, or trouble urinating right away — Spinal cord compression (tumor or broken vertebra squeezing spinal cord) causes permanent paralysis within hours; early radiation or surgery in first 24 hours can save ability to walk
7. Give bisphosphonates like zoledronic acid slowly over at least 15 minutes and check creatinine before each dose — Bisphosphonates stop osteoclasts (cells that eat bone) but infusing too fast causes acute tubular tissue death (necrosis) (sudden kidney death); giving with poor kidney function doubles the damage
8. Assess for peripheral neuropathy by testing light touch and asking about numbness before each chemotherapy dose — Bortezomib and thalidomide damage peripheral nerves; catching early neuropathy allows dose adjustment before patients lose ability to button shirts or walk safely, which is often irreversible

## Sources

---

- International Myeloma Working Group (IMWG) 2014 diagnostic criteria
- NCCN Clinical Practice Guidelines in Oncology: Multiple Myeloma, Version 3.2025
- American Society of Hematology (ASH) guidelines
- UpToDate / Cochrane systematic reviews on multiple myeloma

Last reviewed: 2026-07-07