

MS • Multiple Sclerosis

A plain-English guide for you and your family

WHAT IS HAPPENING

In multiple sclerosis, your immune system makes a mistake and attacks myelin, the fatty insulation that wraps around nerve fibers in your brain and spinal cord. Think of myelin like the rubber coating on electrical wires—it keeps signals moving fast and clear. When T cells (a type of white blood cell that normally fights germs) break through the blood-brain barrier and attack myelin, the signals slow down or stop completely, like a frayed wire sparking instead of carrying electricity smoothly. The damage happens in scattered patches all over the central nervous system, so symptoms depend on which nerves lose their coating. You might have blurred vision if the optic nerve is hit, weak legs if motor pathways are damaged, numbness in your hands if sensory tracts are attacked, trouble holding urine if bladder control fibers are inflamed, or brain fog if patches form in thinking areas. Symptoms often come in relapses—flare-ups lasting days to weeks—then partly or fully fade as inflammation calms, but each attack can leave scar tissue (sclerosis) that adds up over time. Heat makes symptoms worse (called Uhthoff phenomenon) because warmth slows conduction even more in damaged nerves. Disease-modifying therapies (DMTs) are medicines that quiet the immune attack and slow new damage, giving your nervous system the best chance to keep working well for years.

What you might feel

- Vision loss or blurry vision in one eye
- Double vision or eyes that jump around
- Numbness or tingling in the arms, legs, or face
- Weakness in one or more limbs
- Trouble walking or keeping balance
- Feeling exhausted even after rest
- Electric-shock feeling down the spine when bending the neck forward (Lhermitte sign)
- Bladder problems — urgency, leaking, or trouble emptying
- Heat makes symptoms worse (Uhthoff phenomenon)

Call the doctor right away if...

- Vision loss in one eye — may be optic neuritis (swelling of the optic nerve), not just pink eye or simple blurriness; needs urgent imaging and neurology evaluation.
- Sudden weakness or numbness on one side of the body, especially with trouble speaking or drooping face — could be a stroke instead of MS; call 911 immediately.
- Trouble breathing, swallowing, or moving all four limbs — suggests a large lesion in the brainstem or high cervical cord; this is a medical emergency requiring hospital care right away.
- New severe headache, fever, neck stiffness, or confusion — may indicate infection (like meningitis) or another serious brain problem, not just an MS relapse; get emergency evaluation.

What the care team will do

- Check airway, breathing, and swallowing ability every shift during acute flare.
- Monitor for high blood sugar and mood changes during IV methylprednisolone (steroid) treatment.
- Assess fall risk and place the call bell within reach at all times.
- Check bladder fullness by palpating (feeling) the lower abdomen and measure post-void residual (leftover urine after urination) if patient reports incomplete emptying.
- Keep the room cool and offer cold drinks or cooling vests if available.
- Monitor infusion site during IV methylprednisolone and flush line well after each dose.
- Screen for depression and refer to social work or counseling if patient expresses hopelessness or reports isolation.
- Teach the patient to use assistive devices like walkers or braces and consult physical therapy early in admission.

Medicines you may be given — and why

Methylprednisolone (IV)

During an MS flare-up (also called a relapse), the immune system is actively attacking the myelin coating on nerves in the brain and spinal cord, causing inflammation and swelling that blocks signals. Methylprednisolone is a powerful steroid that calms down this immune attack very quickly. It tells the immune cells to stop releasing inflammatory chemicals and helps the swelling go down so nerve signals can travel again. This drug is used only for short bursts during a flare to speed up recovery; it does NOT stop the disease long-term or prevent future attacks.

Ocrelizumab

MS happens when the immune system mistakenly attacks myelin. One key player in this attack is a type of immune cell called a B cell. B cells help other immune cells (T cells) find and destroy myelin, and they also make antibodies that can damage nerves. Ocrelizumab is an antibody drug that locks onto a marker called CD20 on the surface of B cells and wipes them out. With fewer B cells around, the immune attack on myelin slows way down, so there are fewer relapses, less new damage on MRI, and slower worsening of disability. This drug is approved for both relapsing MS and primary progressive MS.

Natalizumab

In MS, immune cells (especially T cells) travel from the blood into the brain and spinal cord, cross through the walls of blood vessels, and attack myelin. To cross into the brain, these immune cells use a sticky protein on their surface called alpha-4-integrin, which acts like a key to unlock the blood-brain barrier. Natalizumab is an antibody that blocks alpha-4-integrin, so immune cells lose their key and cannot get into the brain and spinal cord to cause damage. This dramatically reduces relapses and new brain lesions. It is one of the most effective MS drugs but carries a serious infection risk.

Ofatumumab

Like ocrelizumab, ofatumumab targets and destroys B cells, which play a major role in the immune attack on myelin in MS. B cells help organize the attack and make antibodies that can damage nerves. Ofatumumab locks onto the CD20 marker on B cells and wipes them out, sharply reducing inflammation in the brain and spinal cord. The key difference from ocrelizumab is that ofatumumab is given as a self-injection under the skin at home once a month, making it more convenient. It is approved for relapsing forms of MS.

Siponimod

Immune cells (especially T cells and B cells) normally move back and forth between lymph nodes (where they are trained and activated) and the bloodstream (where they can travel to the brain to attack myelin). A chemical signal called sphingosine-1-phosphate (S1P) acts like a 'come out' signal that tells immune cells to leave the lymph nodes and enter the blood. Siponimod blocks the S1P receptor on immune cells, so they never get the 'come out' signal. The cells stay trapped in the lymph nodes and cannot travel to the brain and spinal cord to attack myelin. This reduces relapses and slows disability progression. Siponimod is approved for relapsing MS and also for secondary progressive MS with active relapses.

Fingolimod

Fingolimod works the same way as siponimod: it blocks the S1P receptor on immune cells, trapping them in lymph nodes so they cannot travel through the blood to the brain and spinal

cord to attack myelin. With fewer immune cells reaching the central nervous system, inflammation drops and relapses are reduced. Fingolimod was the first oral disease-modifying therapy approved for MS and is used for relapsing forms of the disease.

Dimethyl fumarate

In MS, immune cells attack myelin, and the inflammation creates harmful molecules called reactive oxygen species (like rust inside cells) that damage nerves even more. Dimethyl fumarate activates a protein inside cells called Nrf2, which turns on the cell's antioxidant defense system to neutralize these harmful molecules. It also shifts the immune system away from the inflammatory type of immune cells (Th1 and Th17 cells that attack myelin) toward a calmer, regulatory type. This reduces both the immune attack and the toxic byproducts of inflammation, leading to fewer relapses and less new damage on MRI. It is a moderate-efficacy oral drug used for relapsing MS.

Glatiramer acetate

Glatiramer acetate is a mixture of four amino acids arranged in a way that resembles myelin, the coating on nerves that the immune system attacks in MS. When injected under the skin, it acts as a decoy: immune cells (especially T cells) that were trained to attack myelin see glatiramer instead and latch onto it. This shifts the immune response away from a destructive inflammatory type (Th1 cells) toward a calmer, regulatory type (Th2 cells and regulatory T cells) that produce anti-inflammatory signals. It also may help protect nerves by encouraging the release of growth factors. Glatiramer reduces relapses and new MRI lesions modestly and is considered a moderate-efficacy, well-tolerated option, especially for people who prefer injections over pills or infusions.

Interferon beta-1a / Interferon beta-1b

Interferons are naturally occurring proteins that the body makes to fight viruses and regulate the immune system. In MS, the immune system is overactive and inflamed, attacking myelin. Interferon beta medications are lab-made versions of the body's own interferon that are injected to dial down the inflammatory immune response. They reduce the activity of inflammatory T cells and B cells, decrease the production of pro-inflammatory chemicals (cytokines), and help keep the blood-brain barrier more stable so fewer immune cells cross into the brain and spinal cord. This leads to fewer relapses and less new damage on MRI. Interferons were among the first disease-modifying therapies for MS and are still used, especially in people who prefer injectables or have milder disease, but they are now considered moderate-efficacy drugs.

Dalfampridine

In MS, the myelin coating on nerves is damaged, and signals traveling down the nerve slow down or get blocked, especially in the spinal cord pathways that control leg movement.

Dalfampridine blocks certain potassium channels on the surface of damaged nerve fibers. Normally, when a nerve signal travels, potassium leaks out through these channels and the signal weakens. By blocking the leak, dalfampridine helps the nerve signal travel farther and more reliably, which can improve walking speed and leg strength in some people with MS. It does NOT change the disease itself, repair myelin, or prevent relapses — it only improves nerve conduction in already-damaged nerves to help with walking.

Oxybutynin / Tolterodine (anticholinergics) or Mirabegron (beta-3 agonist)

MS can damage nerves that control the bladder, leading to overactive bladder symptoms: urgent need to urinate, frequent urination, and sometimes leakage (urgency incontinence). The bladder muscle (detrusor) contracts too often or at the wrong times. Anticholinergic drugs (oxybutynin, tolterodine) block the acetylcholine signal that tells the bladder muscle to squeeze, so the bladder stays relaxed and can hold more urine without sudden urges. Mirabegron works differently: it stimulates beta-3 receptors in the bladder wall, which relax the bladder muscle, allowing it to fill more fully. These drugs do NOT treat MS itself — they only manage the bladder symptom.

Vitamin D supplementation

Vitamin D is not a drug, but a nutrient that the body makes when skin is exposed to sunlight, and it can also be taken as a supplement. Vitamin D plays a role in regulating the immune system, and low levels of vitamin D have been linked to a higher risk of developing MS and possibly more active disease. While vitamin D does NOT treat or cure MS, many guidelines recommend that people with MS keep their vitamin D levels in the normal range (usually aiming for a blood level of 30–40 ng/mL or higher) because it may support overall immune health and bone health (important since some MS drugs and reduced mobility can weaken bones). The evidence that vitamin D supplementation slows MS progression is not strong, but correcting a deficiency is safe, inexpensive, and may have modest immune and bone benefits.

Where this information comes from

- American Academy of Neurology (AAN) MS guideline 2018
- McDonald criteria 2017 (International Panel on MS Diagnosis)
- National Multiple Sclerosis Society clinical recommendations
- Lancet Neurology MS review 2018

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