THE Sumaira FOUNDATION

What to KNOW about MOGAD

The information on this page is designed to provide a patient-friendly overview about myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD).

MOGAD is a relatively recently recognized condition, and our understanding of the disease continues to evolve. The development of reliable MOG-IgG antibody testing - and its commercial availability beginning in 2015 (Europe) and 2017 (US) - was a major milestone, enabling more patients to be accurately identified and diagnosed. Today, the number of neurologists specializing in MOGAD is growing, while researchers continue to work to better understand the disease, improve diagnostic testing and develop targeted treatments. In 2023, the publication of the first international diagnostic criteria for MOGAD marked another significant step forward in recognizing and accurately diagnosing this condition.

Below, you will find a list of important questions and answers with the goal of informing you and your loved ones about this condition. This guide includes important information covering diagnosis, symptoms, and available treatments. Please be aware that the questions below are answered with general information about MOGAD and may not fit your individual situation.

Information on this page is not intended to be used as a substitute for medical care and should not be relied upon for the diagnosis or treatment of MOGAD. If you have questions or concerns regarding your health, please contact your healthcare provider.

What is MOGAD?

Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) is a condition where the body's immune system, which normally fights infections, mistakenly attacks a protein called MOG (myelin oligodendrocyte glycoprotein) that sits on the surface of myelin, the protective coating around nerve fibers in the brain, spinal cord, and optic nerves. This attack causes inflammation and damage to the central nervous system (CNS).

MOGAD is considered rare. **Roughly 1 to 3 people per 100,000 are living with MOGAD**. Unlike many other autoimmune diseases, MOGAD affects men and women roughly equally. It can start at any age, with an **average age of onset around 30 years, and approximately 30% of cases begin in childhood.**

What are the common symptoms of MOGAD?

MOGAD attacks the central nervous system and can affect the optic nerves, spinal cord, and brain. Symptoms depend on which part of the nervous system is inflamed:

Eyes (optic neuritis) — the most common presentation in adults:

- Pain with eye movement, often severe
- Vision loss, which can be sudden and significant, sometimes affecting both eyes at the same time (up to 50% of cases)
- Swelling of the optic disc (papilledema), which is often prominent
- Vision typically recovers well with treatment, though repeated attacks can cause lasting damage

Spinal cord (transverse myelitis):

- Weakness, numbness, or tingling in the arms and/or legs
- Difficulty walking
- Bladder or bowel problems
- Lesions in the spinal cord are often long (spanning three or more vertebral segments), a pattern called longitudinally extensive transverse myelitis (LETM)

Brain (acute disseminated encephalomyelitis — ADEM):

- The most common presentation in young children
- Widespread inflammation in the brain causing confusion, drowsiness, irritability, or difficulty walking
- Often preceded by a viral illness or infection
- Usually recovers well, especially in children

Brainstem and cerebellum:

- Dizziness, difficulty with balance and coordination

- Double vision, difficulty swallowing, or facial weakness

Cerebral cortical encephalitis:
- Seizures
- Fever
- Confusion or altered consciousness
- A less common but recognized presentation of MOGAD

Unlike MS, **MOGAD does not typically cause a slow, progressive worsening of symptoms over time**. Instead, disability in MOGAD comes from the damage caused by individual attacks, which is why preventing attacks and treating them quickly is so important.

What is the cause of MOGAD?

Like other autoimmune diseases, the immune system makes a mistake and attacks the body's own healthy tissue. In MOGAD, the immune system produces antibodies (called MOG-IgG) that target the MOG protein on the surface of myelin. This leads to inflammation and demyelination: damage to the protective coating of nerve fibers.

Attacks are often preceded by an infection (reported in about 20–40% of cases), suggesting that infections may sometimes trigger the immune system to mistakenly attack MOG. However, **MOGAD is not an infection and is not contagious.**

What antibodies are associated with MOGAD?

When a demyelinating disease is suspected, blood tests for specific antibodies help determine which condition you have. Three antibodies are important to know about:

MOG-IgG (the MOGAD antibody):
This antibody targets myelin oligodendrocyte glycoprotein (MOG), a protein on the surface of the protective coating around nerve fibers. A positive MOG-IgG test is required for a MOGAD diagnosis. **Key points:**
- Must be tested using a specialized method called a cell-based assay — older testing methods are not reliable
- Levels tend to be highest during or shortly after an attack, so timing of the blood draw matters
- Levels can change over time — patients whose MOG-IgG becomes negative may have a lower risk of future attacks
- A high-positive result is very reliable; a low or borderline result needs to be interpreted carefully alongside your symptoms and MRI findings

AQP4-IgG (the NMOSD antibody):
This antibody targets aquaporin-4, a water channel protein in the brain and spinal cord. It is the hallmark of a different condition called neuromyelitis optica spectrum disorder (NMOSD). MOGAD and NMOSD can look similar, but they are different diseases requiring different treatments. MOG-IgG and AQP4-IgG almost never occur together in the same patient. When one is suspected, both are typically tested at the same time.

Multiple sclerosis (MS) — no specific antibody
Unlike MOGAD and NMOSD, there is no blood antibody test for MS. This is one reason antibody testing is so valuable — a positive MOG-IgG or AQP4-IgG result helps steer doctors toward the correct diagnosis, which is critical because the treatments are very different.

How is MOGAD diagnosed?

MOGAD is diagnosed by a neurologist, ideally one who specializes in neuroimmunology or demyelinating diseases. According to the 2023 International MOGAD Panel diagnostic criteria, three requirements must be met:

1. **A compatible clinical attack:** One of six recognized types of CNS inflammation; optic neuritis, myelitis, ADEM, cerebral monofocal or polyfocal deficits, brainstem/cerebellar deficits, or cerebral cortical encephalitis.
2. **Positive MOG-IgG antibodies:** Detected in the blood (serum) using a specialized test called a cell-based assay (CBA). This is the key diagnostic test. If the antibody level is low or borderline, additional supporting clinical or MRI features are required to confirm the diagnosis.
3. **Exclusion of other diagnoses:** Particularly multiple sclerosis, which can sometimes look similar.

Important notes about testing:

- **MOG-IgG should be tested using a cell-based assay**; older testing methods are not reliable enough
- **Testing should ideally be done during or shortly after an attack**, when antibody levels are highest
- **Not everyone with CNS inflammation should be tested for MOG antibodies;** indiscriminate testing in patients with a low likelihood of MOGAD can lead to false positive results
- Antibody levels can fluctuate over time and may become negative, especially in patients with monophasic disease

Other tests that may be performed:

- **MRI of the brain, spinal cord, and orbits:** To look for patterns of inflammation characteristic of MOGAD (such as long optic nerve enhancement, longitudinally extensive spinal cord lesions, or the "H-sign" on axial spinal cord images)
- **Lumbar puncture (spinal tap):** To analyze the cerebrospinal fluid. Unlike MS, MOGAD patients usually do not have oligoclonal bands in their spinal fluid
- **Blood tests:** To check for AQP4 antibodies (to rule out NMOSD) and other conditions

How is MOGAD different from multiple sclerosis (MS) and neuromyelitis optica spectrum disorder (NMOSD)?

MOGAD was only recently recognized as its own distinct disease. For many years, **patients with MOGAD were often misdiagnosed with MS or NMOSD because the symptoms can look similar**; all three conditions can cause optic neuritis (inflammation of the optic nerve), myelitis (inflammation of the spinal cord), and brain lesions.

However, MOGAD is now understood to be a separate condition with its own:

- **Antibody**: MOG-IgG (different from the AQP4 antibody in NMOSD; MS has no specific antibody)
- **Disease behavior**: MOGAD can be monophasic (a single episode that never returns), which is uncommon in MS or NMOSD
- **MRI patterns**: MOGAD tends to show different patterns on brain and spinal cord MRI than MS or NMOSD
- **Treatment response**: MOGAD responds differently to treatment — importantly, we do not know if medications approved for MS or NMO would be helpful. Currently, treatments used for MOGAD are off-label, meaning they have not been tested in clinical trials or approved by the FDA.
- **Prognosis**: Overall, MOGAD tends to have a more favorable long-term outcome than NMOSD, with more than 80% of patients having no or mild disability at last follow-up

This distinction matters because the right diagnosis leads to the right treatment.

How does MOGAD present differently in children versus adults?

The way MOGAD appears depends significantly on age:

Children (especially under age 11):

- **ADEM** is the most common first presentation, occurring in nearly half of pediatric cases
- **Children are more likely to have a monophasic** (one-time) course — more than 70–80% of children may never have a second attack

- Recovery is generally excellent, with most children returning to normal or near-normal function
- Children who become MOG-antibody negative over time are less likely to relapse

Adults:

- **Optic neuritis is the most common presentation** (about 55–67% of cases), followed by transverse myelitis
- **Adults have a higher risk of relapse than children**
- **Adults are more likely to have residual disability after attacks** compared with children

What does a MOGAD attack look like?

A MOGAD attack (also called a relapse or exacerbation) is a new episode of CNS inflammation. It typically comes on over hours to days and may include:

- New or worsening vision loss or eye pain
- New weakness, numbness, or tingling in the arms or legs
- Difficulty walking or with balance
- Confusion, drowsiness, or seizures (especially in children)
- New difficulty with bladder or bowel control

Attacks are often preceded by an infectious illness (such as a cold, sinus infection, or stomach bug). Unlike the daily fluctuation seen in some other neurological conditions, MOGAD attacks represent distinct episodes of new inflammation.

Can MOGAD attacks cause lasting damage?

Yes. **While MOGAD generally has a more favorable prognosis than NMOx=SD, this does not mean everyone recovers completely**. About half of MOGAD patients are left with some form of lasting deficit after their attacks. Disability in MOGAD comes from the damage caused by individual attacks, not from slow, progressive worsening between attacks, which is why preventing attacks and treating them quickly is so critical.

The specific lasting effects depend on where the attack occurred:

- **After optic neuritis:** Most patients recover vision well, but some are left with reduced color vision, blind spots, difficulty with contrast, or, in about 5–14% of cases, significant permanent vision loss. Repeated attacks increase the risk of cumulative damage.
- **After transverse myelitis:** This is the attack type most likely to cause lasting disability. Residual effects can include weakness (hemiparesis or paraparesis), gait instability, chronic neuropathic pain (burning, stabbing, tingling, or electric shock sensations),

numbness, spasticity, and fatigue. Bladder, bowel, and sexual dysfunction are strikingly common, studies show 44–75% of patients who have had myelitis are left with permanent bladder problems, even when walking ability recovers well. Over half of myelitis patients report chronic pain. These "hidden" symptoms are real, common, and often underappreciated by standard disability measures.

- **After brain attacks (ADEM):** Most patients, especially children, recover well. However, some may experience lasting cognitive difficulties (attention, memory, processing speed), fatigue, depression, or anxiety.

Will I have another attack?

This is one of the most common and important questions after a first MOGAD attack. The honest answer is: it is difficult to predict.

- **Monophasic disease (single attack, no relapses):** Occurs in approximately 30–50% of adults and 50–72% of children. Many patients will only ever have one attack.
- **Relapsing disease:** Occurs in approximately 40–80% of cases overall. Relapses are most likely to happen in the first two years after the initial attack, with the risk declining significantly after that.

Factors that may help predict the disease course:

- Patients whose MOG-IgG antibodies become negative over time appear to have a lower risk of relapse
- Early relapses (within the first year) are associated with a higher risk of long-term relapsing disease
- Persistently high MOG-IgG titers are associated with a higher likelihood of relapse

Because it is not possible to reliably predict who will relapse after a first attack, ongoing follow-up with a neurologist is essential.

How are MOGAD attacks treated?

Acute attack treatment:

The first-line treatment for a MOGAD attack is high-dose intravenous (IV) corticosteroids, typically IV methylprednisolone given for 3–5 days. MOGAD attacks are generally very responsive to steroids.

If steroids do not lead to adequate improvement, the next step is usually:

- **Plasma exchange (PLEX/plasmapheresis)**: A procedure that filters harmful antibodies out of the blood

Intravenous immunoglobulin (IVIg) may also be used as an alternative acute treatment.

Important — do not stop steroids too quickly:

A key lesson learned about MOGAD is that tapering steroids too rapidly after an attack can trigger a relapse. Studies have shown that a prolonged oral steroid taper (at least 12 weeks or longer) after the initial IV pulse is associated with a lower risk of relapse. Your neurologist will create a tapering schedule tailored to your situation.

Does delayed diagnosis or treatment worsen the prognosis?

Yes. Early treatment of acute attacks is strongly associated with better recovery. One large study found that patients treated within 5 days of their first attack had dramatically lower relapse rates at 10 years (41%) compared with those treated later or not at all (87%). For patients who go undiagnosed for months or years — as can happen when MOGAD is mistaken for MS, untreated attacks may cause damage that could have been reduced or prevented with timely therapy.

When should I seek emergency care?

Go to the nearest emergency room or call 911 if you experience:

- Sudden severe vision loss in one or both eyes
- Sudden inability to walk or move your arms or legs
- Difficulty breathing
- Seizures or sudden confusion
- Loss of bladder or bowel control

Make sure the emergency doctors know you have MOGAD so they can consult a neurologist promptly.

What are the long-term treatment options for MOGAD?

Should everyone with MOGAD be on long-term treatment?

Not necessarily. Because a significant proportion of patients (especially children) will only ever have one attack, many neurologists wait to see if a second attack occurs before starting long-term preventive treatment. However, long-term treatment may be started after a first attack if:

- Recovery from the first attack is incomplete
- The first attack was particularly severe
- MOG-IgG antibodies remain persistently positive at high levels

Preventive (maintenance) treatments:

There are currently no FDA-approved treatments specifically for MOGAD, so all maintenance therapies are used "off-label." The main options include:

- **Intravenous immunoglobulin (IVIg)**: Given as regular infusions (typically monthly). Evidence suggests IVIg may be the most effective maintenance therapy for preventing MOGAD relapses, with a favorable safety profile. It is often considered a first-line option.
- **Oral corticosteroids (prednisone):** Effective at preventing relapses but long-term use carries risks including weight gain, bone thinning, diabetes, cataracts, and mood changes. Doctors try to use the lowest effective dose.
- **Rituximab**: An IV medication that targets B cells (immune cells that produce antibodies). It reduces relapse rates in MOGAD, though relapses can still occur despite B-cell depletion — it may not be as effective in MOGAD as it is in NMOSD.
- **Azathioprine**: A daily pill that broadly suppresses the immune system. It can take several months to become fully effective and requires regular blood monitoring.
- **Mycophenolate mofetil (CellCept):** Another daily immunosuppressant pill. It appears to have a more modest effect on MOGAD relapses compared with IVIg or azathioprine.
- **Tocilizumab**: An IL-6 receptor blocker given as an IV infusion or injection. Increasingly used in MOGAD, particularly in refractory cases.

Medications to avoid:

Standard MS disease-modifying therapies, such as interferon beta, glatiramer acetate, natalizumab, and fingolimod, have not been tested for MOGAD.

Can treatment ever be stopped?

This is an active area of research. Unlike NMOSD, where lifelong treatment is generally recommended, the natural history of MOGAD, with its tendency toward fewer relapses over time, raises the possibility that treatment may eventually be safely discontinued in some patients.

Factors that may support considering treatment discontinuation include:

- Several years of relapse-free stability on treatment
- MOG-IgG antibodies becoming negative
- A declining relapse rate over time

However, this decision should always be made **carefully with a neurologist**, as relapses can still occur after stopping treatment.

What does the future look like for MOGAD treatment?

MOGAD research is advancing rapidly. The publication of the first international diagnostic criteria in 2023 was a major milestone. Clinical trials specifically designed for MOGAD are now underway, testing targeted therapies that may be more effective and have fewer side effects than current off-label treatments. Scientists are also working to better understand why some patients have a single attack while others relapse, which could lead to more personalized treatment approaches in the future.

A closer look at two current clinical studies:

For the first time, large, rigorously designed clinical trials are testing whether specific medications can prevent MOGAD relapses. Two of the most important are listed below. Both focus on people with relapsing MOGAD (those who have had more than one attack) and both are studying whether a targeted medication can lower the risk of future attacks compared with a placebo. Enrollment in these particular studies has closed, but they represent a major step toward the first treatments approved specifically for MOGAD.

Rozanolixizumab study (NCT05063162): This Phase 3 study is testing rozanolixizumab, an "FcRn antagonist." This type of medication works by speeding up the body's removal of harmful antibodies (like the MOG antibody) from the bloodstream. The study is enrolling adults (ages 18–89) who have MOGAD, are MOG-antibody positive, AQP4-antibody negative, and have had at least one relapse in the past year. This study is now closed to new participants.

Satralizumab study (NCT05271409): This Phase 3 study is testing satralizumab, a medication that blocks a signal in the immune system called interleukin-6 (IL-6), which is involved in driving inflammation. Notably, this study includes not only adults but also adolescents (ages 12 and older). It is designed to measure how long patients go before their next relapse, and participants may be able to continue on the medication afterward in an extension phase. This study has completed and reported positive results. The FDA is currently evaluating the trial results.

Why this matters: Because there are currently no FDA-approved treatments made specifically for MOGAD, all current preventive medications are used "off-label." Trials like these are how new, targeted therapies eventually become approved and widely available. If you are interested in learning whether a clinical trial might be an option for you, talk with your neurologist, and always discuss any trial with your care team before enrolling.

How does MOGAD affect pregnancy?

MOGAD frequently affects women of childbearing age, so pregnancy planning is important.

Key things to know:

- Relapse risk appears to decrease during pregnancy, particularly in the third trimester. In studies, no relapses were observed during pregnancy in most patients.
- The postpartum period (the months after delivery) carries a higher risk of relapse. Close monitoring during this time is important.
- About 12.5% of women with MOGAD experience their first symptoms during pregnancy or in the year after delivery.
- Some women with previously monophasic disease may transition to a relapsing course postpartum.

Treatment during pregnancy:

- Acute attacks during pregnancy can be treated with IV methylprednisolone, with plasma exchange reserved for severe or steroid-resistant cases
- Longer steroid tapers after an attack during pregnancy may help reduce relapse risk
- The decision to continue or start preventive treatment during pregnancy should be individualized, some treatments may be continued, while others should be avoided
- IVIg is generally considered safe during pregnancy

Always discuss pregnancy planning with your neurologist well in advance so that medications can be adjusted and a monitoring plan can be put in place

How can I manage MOGAD in my daily life?

- **Know your symptoms:** Learn to recognize the early signs of a new attack (changes in vision, new weakness or numbness, balance problems) so you can seek treatment quickly. Early treatment of attacks leads to better outcomes.
- **Keep a symptom diary:** Track any new or changing symptoms to share with your neurologist.
- **Stay up to date on infections:** Since infections can trigger attacks, practice good hygiene, stay current on recommended vaccinations (discuss with your neurologist first, as some vaccines may need to be avoided depending on your treatment), and seek prompt treatment for infections.
- **Attend regular follow-up appointments:** Even if you feel well, regular check-ups with your neurologist are important to monitor antibody levels, assess for subtle changes, and adjust treatment if needed.
- **Communicate with all your doctors:** Make sure every healthcare provider you see knows you have MOGAD. This is especially important if you are ever prescribed a new medication, as standard MS treatments can be harmful in MOGAD.
- **Stay connected:** Patient organizations and support groups for MOGAD and related conditions can provide valuable information, emotional support, and community.

Living with residual symptoms

If you are living with lasting effects from a MOGAD attack, you are not alone. Talk to your neurologist about all of your symptoms — not just the ones that happen during attacks. Depending on your needs, your care team may recommend physical therapy, occupational therapy, medications for neuropathic pain (such as gabapentin or pregabalin), urology referral for bladder issues, and mental health support. Many residual symptoms can be improved with the right interventions.

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