Fact Sheet

MOG Antibody Disease

MOGAD

Revised 4/12/2021 | This information sheet has been reviewed and approved by members of SRNA’s Medical and Scientific Council.
MOG antibody disease (MOGAD) is a recently coined neuro-inflammatory condition that preferentially causes inflammation in the optic nerve but can also cause inflammation in the spinal cord and brain. Myelin oligodendrocyte glycoprotein (MOG) is a protein that is located on the surface of myelin sheaths in the central nervous system.\(^1,2\) While the function of this glycoprotein is not exactly known, MOG is a target of the immune system in this disease.\(^3\) The diagnosis is confirmed when MOG antibodies in the blood are found in patients who have repeated inflammatory attacks of the central nervous system.\(^4\)

Those with MOG antibody disease may previously have been diagnosed with neuromyelitis optica spectrum disorder (NMOSD), transverse myelitis (TM), acute disseminated encephalomyelitis (ADEM), optic neuritis (ON), or multiple sclerosis (MS) because of the pattern of inflammation it causes including brain, spinal cord and optic nerve damage. Patients with persistently positive antibodies are at risk for recurrent events. Those with MOG antibody disease do not test positive for the NMO antibody called aquaporin 4 (AQP-4). MOG antibody disease and AQP-4 positive NMOSD are thought to have distinct immunological mechanisms.\(^4\) Furthermore, those with MOG antibody disease seem to be less likely to have other autoimmune disorders (such as rheumatoid arthritis, Hashimoto’s thyroiditis, etc.) than those with AQP-4 positive NMOSD.\(^4\)
Epidemiology

Among patients with AQP-4 seronegative NMOSD, the frequency of a positive MOG antibody test ranges between 7.4% and 39%. Studies have indicated that between 40% and 58% of children diagnosed with ADEM are positive for the anti-MOG antibody. While there is significant overlap between MOGAD, NMOSD, and ADEM, it appears that MOGAD is a unique immunological condition.

Some studies have shown that those with MOG antibody disease are on average younger and are likely to be male compared to those with AQP-4 positive NMOSD, but other studies have shown no age differences and varying gender distributions. One study revealed a higher proportion of those of Caucasian ethnicity among MOG patients, while others have not shown this difference.

Signs and Symptoms

MOG antibody disease preferentially causes inflammation in the optic nerve, but can also cause inflammation in the spinal cord, brain, and brainstem. Symptoms can include:

- Loss or blurring of vision in one or both eyes
- Loss of color vision
- Paralysis (no motor function) of a limb or limbs
- Paraparesis (weakness) of a limb or limbs
- Loss of sensation
- Loss of bladder or bowel control
- Profound bladder retention
- Seizures

Those with MOG antibody disease are more likely to have both optic nerves affected at the same time, and if the symptoms are in only one eye, the other optic nerve may show subclinical atrophy.

Children can be found to have the MOG antibody in the setting of ADEM; however, a positive MOG antibody test in the setting of ADEM does not necessarily imply a course of MOGAD. In many kids, the MOG antibody disappears within 1 year, and relapses do not occur. In some, the MOG antibody persists, and relapses may occur. When a relapse occurs, the diagnosis of MOGAD is confirmed.

MOG antibody disease can also occur in relation to another condition called anti-N-methyl-D-aspartate (NMDA) receptor encephalitis. NMDA receptor encephalitis is an autoimmune encephalitis that can cause psychosis, issues with memory and language, and seizures.

One study described symptoms and MRI findings of MOG antibody disease in children under the age of 7 that were similar to leukodystrophies. Leukodystrophies are genetic rare diseases that affect the central nervous system by disrupting myelination.
Diagnosis

There are blood tests that can test for MOG antibodies. Only cell-based assays are considered reliable for the diagnosis of MOGAD because of the improved specificity over older ELISA tests. CSF analysis from a lumbar puncture may show increased white blood cell counts in some patients during a relapse, and oligoclonal bands are not usually found.

Unlike anti-AQP4 antibodies, anti-MOG antibodies may decrease over time, and may not be detectable early in the disease process or during remission, and this is especially the case for MOG antibody disease associated ADEM. Those with persistent detection of anti-MOG may be more likely to have a relapsing rather than monophasic disease course.

There appears to be no overlap between individuals with anti-MOG positivity and AQP-4 positivity, although there have been some isolated cases reported using the older ELISA assay.

MRI findings are similar to those with MS and NMOSD, but there may be some differences. MOG antibody disease optic neuritis seems to predominantly affect the retrobulbar region, while AQP-4-associated optic neuritis is found intracranially. Furthermore, MOGAD lesions in the brain can look like lesions seen in those with ADEM.

Acute Treatments

Treatment guidelines for MOG antibody disease have not been established. The following are possible treatments in the management of an acute event.

Intravenous Steroids

Although there are no clinical trials that support a unique approach to treat patients with MOG antibody disease, it is well recognized as a standard of care to give high-dose intravenous methylprednisolone for suspected acute myelitis or optic neuritis, generally for 3 to 5 days, unless there are compelling reasons not to. The decision to offer continued steroids or add a new treatment is often based on the clinical course and MRI appearance at the end of 5 days of steroids. Those with MOG antibody disease seem to respond well to steroids. An oral steroid taper may be helpful to prevent steroid-withdrawal relapses.

Plasma Exchange (PLEX)

PLEX is believed to work in autoimmune CNS diseases through the removal of specific or nonspecific soluble factors likely to mediate, be responsible for, or contribute to inflammatory-mediated organ damage. PLEX is often recommended for moderate to aggressive forms of TM and ON, as is very often the case with MOG antibody disease, if there is not much improvement after being treated with intravenous steroids. If presenting symptoms are severe, PLEX may be initiated concurrently with steroids. There have been no prospective clinical trials that prove PLEX’s effectiveness in MOG antibody disease, but retrospective studies of TM treated with IV steroids followed by PLEX have shown a beneficial outcome. PLEX also has been shown to be effective in other autoimmune or inflammatory central nervous system disorders. Early treatment is beneficial – PLEX is typically started within days of administering steroids, very
... Acute Treatments

often before the course of steroids has finished. Particular benefit has been shown if started within the acute or sub-acute stage of the myelitis or if there is continued active inflammation on MRI.

**Intravenous Immunoglobulin (IVIG)**

Another option for treating anti-MOG associated acute inflammation is intravenous immunoglobulin (IVIG). Immunoglobulin comes from pooled blood that is donated from thousands of healthy people. As the name suggests, IVIG is given intravenously. IVIG is generally well-tolerated. Potential adverse reactions are uncommon, but usually occur during or immediately after an infusion and include headache, nausea, muscle pain, fever, chills, chest discomfort, skin and anaphylactic reactions. Reactions after an infusion can be more serious and include migraine headaches, aseptic meningitis, renal impairment and blood clots. Like corticosteroids and PLEX, there are no data confirming the value of IVIG in the setting of acute events. While most studies support the use of corticosteroids and/or PLEX in acute demyelinating syndromes, IVIG can be considered in certain circumstances.

**Other Acute Treatments**

In cases of no response to either steroids or PLEX therapy and continued presence of active inflammation in the spinal cord, other forms of immune-based interventions may be required. The use of immunosuppressants or immunomodulatory agents may be considered in some cases. Initial presentation with aggressive forms of myelitis, or if particularly refractory to treatment with steroids and/or PLEX, aggressive immunosuppression is considered. Individuals should be monitored carefully as potential complications may arise from immunosuppression. As with all medications, risks versus benefits of aggressive immunosuppression need to be considered and discussed with the clinical care team.

**Prognosis and Management**

Initially, the presence of anti-MOG was thought to be associated with fewer relapses and better outcomes than those with AQP-4 positive NMOSD, but studies with longer follow-up times indicate higher relapse rates than previously reported.

A cohort study from 2016 found that 80% of those in the cohort had a multiphasic disease and an annualized relapse rate (AAR) of 0.9. They found that one third of patients with optic neuritis and around half of patients with spinal cord inflammation made a full recovery. In contrast, two other studies showed that the retinal neuro-axonal damage found after an acute attack of optic neuritis was as severe among anti-MOG positive individuals as individuals with AQP-4 positive NMOSD.

Those with MOG antibody disease should consider ongoing treatment with medications that suppress the immune system. There are no FDA-approved medications for maintenance in MOG antibody disease, so anything prescribed is done off-label. The primary therapies used in the US are mycophenolate mofetil (CellCept), rituximab (Rituxan), azathioprine (Imuran), and repeated IVIG infusions or subcutaneous immunoglobulin. Some studies from the United Kingdom have supported the use of IVIG to prevent relapses.
Prognosis and Management

Some patients presenting with optic neuritis or transverse myelitis who also test positive for the MOG antibody may start treatment after the initial event if the attack was severe and the individual does not want to risk a relapse.

All of these medications carry a risk of infections, particularly upper respiratory infections and urinary tract infections (UTIs). Good hygiene and hand washing are important if on immunosuppressants, as is having a good urologist if at risk for UTIs. There is also the risk with any of these medications of the development of a rare brain infection called progressive multifocal leukoencephalopathy, or PML. PML is an infection caused by the reactivation of a virus, called the JC virus, which lives in the kidney. In someone who is immunosuppressed, this virus can escape the kidney, cross the blood-brain barrier, and enter the brain, causing profound inflammation. Although it can be treated, it is very devastating and sometimes fatal. It is important to know that exposure to these medications in MOG antibody disease has not led to a known case of PML. The known rate of incidence of PML if on Rituxan is estimated at 1 in 25,000 and the rate in CellCept is estimated at 1 in 6,000 based on data from use of these medications for immunosuppression for other purposes. The manufacturer of Imuran cautions about a risk of PML with Imuran as well, but the incidence of PML on Imuran is not documented. Clinical diligence and early intervention are important if PML is suspected.

Chronic immunosuppression requires regular skin exams with a dermatologist since our immune system is our best defense against cancer cells developing, and any of these treatments can interfere with its normal functioning.

Mycophenolate mofetil and azathioprine are both twice daily pills which broadly suppress the immune system. Both medications were originally FDA approved for organ transplant rejection prophylaxis, although azathioprine now is indicated in rheumatoid arthritis, and both have been widely used in several autoimmune disorders. These medications require frequent blood draws upfront, then generally twice yearly to monitor for liver toxicity and to ensure optimal immunosuppression (absolute lymphocyte count around 1 and total white blood cell count between 3 and 4).

Azathioprine

Azathioprine is the medication that has been around the longest. However, while the AAR seems to be low on azathioprine, one complication with this medication is that some are not able to stay in remission on azathioprine alone and have to also be on steroids (complications of steroids will be discussed below). Additionally, a long-term study of azathioprine found that the risk of lymphatic-proliferative cancers was reported to be 3%. A common side effect includes gastrointestinal upset, and this may manifest as bloating, constipation, nausea, diarrhea, and may vary throughout the course of one’s time on the medication. Azathioprine is contraindicated in pregnancy, so pregnancy planning is very important. It is FDA Category D (which means don’t take this drug during pregnancy unless it’s lifesaving) and is associated with an increased risk of miscarriages, 7% rate of congenital problems, and high rate of bone...
... Prognosis and Management

marrow suppression that recovers after birth. It is the cheapest of the medications.
One study among those with MOG antibody disease found that the mean ARR for
azathioprine was 0.99, with 41% of the attacks occurring during the first 6 months,
and most of these early attacks were in those who were not also being treated with
corticosteroids.\textsuperscript{3,13}

**Mycophenolate mofetil**

Mycophenolate mofetil has a similar effect on the gastrointestinal system, though
many report that the symptoms are milder with mycophenolate as compared with
azathioprine. Additionally, some complain of headaches with mycophenolate,
particularly in the beginning; these tend to wane with ongoing use. Lymphoma may be
a risk of this medication; however, there have been no cases reported in MOG antibody
disease patients while on this medication, so the risk is likely low. Mycophenolate is
also contraindicated in pregnancy, so, again, planning is imperative. It is also an FDA
Category D (don't take this drug during pregnancy unless it's lifesaving) and carries
a 45% chance of miscarriage. Of those that do not miscarry, 22% have congenital
defects mostly in the face (mouth, ears).

**Rituximab**

Rituximab is an intravascular infusion which works differently from the other two
agents listed above. Rather than being a broad immunosuppressant, rituximab
completely depletes one particular type of white blood cell called B-cells, which has
downstream effects on the rest of the immune system. Though protocols are slightly
different, in general, it is given two times twice a year (4 infusions total) and is given
in an outpatient infusion center. This is because of a 30% risk of an infusion reaction
without pre-medication with some cocktail of methylprednisolone, diphenhydramine
and perhaps acetaminophen. The medication is quite well-tolerated. There are
generally no side effects to the medication. There is no lymphoma risk with this
medication. There is a monthly blood test to monitor the B-cell CD20 expression.
Rituximab is safer in pregnancy than the other two previously described, (Category
C; may be toxic in animals or no human data) — there are no official FDA reports of
birth defects in cases of pregnancy with rituximab, but babies are born with no CD20
cells. It does not appear to increase risk of infection in babies as the cells re-populate
within 6-18 months. In monkey studies performed by the manufacturer, there was
no toxicity on the fetus, and monkey babies were born with no CD20 cells, again with
no infection risks. In the largest case series published in February 2011, out of 153
women who became pregnant on rituximab, there were 4 post-natal infections and
two congenital abnormalities (1 club foot, 1 heart defect), but these women were
also on other immunosuppressant medications during the pregnancy, including
azathioprine and mycophenolate. They concluded that rituximab does not increase the
risk of congenital malformations above the natural rate of 1-2%. Planned pregnancy
is still recommended. A study looking at rituximab among those with MOG antibody
disease found that three out of nine patients experienced a decline in the ARR, and
most relapses occurred either soon after an infusion or at the end-of-dose period.\textsuperscript{13}

**Prednisone**

Low-dose prednisone is used as well, more often outside of the United States. As
noted above, some clinicians also use it in combination with azathioprine for those
Long-Term Care

After the acute phase, rehabilitative care to improve functional skills and prevent secondary complications of immobility involves both psychological and physical accommodations. There is very little written in the medical literature specifically dealing with rehabilitation after MOGAD. However, much has been written regarding recovery from spinal cord injury (SCI), in general, and this literature applies. The physical issues include bowel and bladder management, sexual dysfunction, maintenance of skin integrity, spasticity, activities of daily living (i.e., dressing), mobility, and pain.

It is important to begin occupational and physical therapies early during the course of recovery to prevent the inactivity-related problems of skin breakdown and soft tissue contractures that lead to a decreased range of motion. Assessment and fitting for splints designed to passively maintain an optimal position for limbs that cannot be actively moved is an important part of the management at this stage.

The long-term management of MOGAD requires attention to a number of issues. These are the residual effects of any spinal cord injury or injury to the optic nerve. In addition to chronic medical problems, there are the ongoing issues of ordering the appropriate equipment, reentry into school, re-socialization into the community, and coping with the psychological effects of this condition by the patients and their families. During the early recovery period, family education is essential to develop a strategic plan for dealing with the challenges to independence following return to the community.
**Visual Issues**  
For patients who have had optic nerve inflammation, residual vision loss can be experienced. Patients can possibly note blurred vision, loss of color vision, difficulty with depth perception and glares or halos around lights at night. Furthermore, patients who fully recover vision after optic neuritis may experience transient returns of blurred vision during times of stress, exertion or heat exposure.

**Bladder Function**  
When the spinal cord is damaged, two general problems can affect the bladder. The bladder can become overly sensitive and empty after only a small amount of urine has collected, or relatively insensitive, causing the bladder to become over extended and overflow. An overly distended bladder increases the likelihood of urinary tract infections and, in time, may threaten the health of the kidneys. Depending on the dysfunction, treatment options include timed voiding, medicines, external catheters for males (a catheter connected to a condom), padding for women, intermittent internal self-catheterization, an indwelling catheter or electrical stimulation. Surgical options may be appropriate for some people. Common bladder problems include incontinence, frequency, nocturia (frequent urination at night), hesitancy, and retention. Treating incontinence, frequency, and nocturia is often easier than treating hesitancy and retention, where clean intermittent urinary catheterizations are the basic component to success. Working with a good urologist is imperative to prevent potential serious complications, particularly those who understand spinal cord disease. Urodynamic testing is necessary to determine urine retention to check risk for urinary tract infections, particularly if there is a history of UTIs to guide the urologist in terms of the best management.

**Bowel Function**  
Another major area of concern is effective management of bowel function. A common problem in spinal cord injury is difficulty with evacuation of stool, although fecal incontinence can also occur. The neurologic pathways for defecation are similar to those of the bladder. Many lacking voluntary control of the bowel may still be able to achieve continence by diet, strategic use of stool softeners and fiber, and the technique of rectal stimulation. Other aids include suppositories, anal irrigation, and oral medications. A high-fiber diet, adequate and timely fluid intake, and medications to regulate bowel evacuations are the basic components of success. Regular evaluations by medical specialists for adjustment of the bowel program are recommended to prevent potentially serious complications. There are some surgical options, although this is rarely necessary.

**Sexual Dysfunction**  
Sexual dysfunction involves similar innervation and analogous syndromes as those found in bladder dysfunction. Treatment of sexual dysfunction should take into account baseline function before the onset of MOGAD. Of the utmost importance is adequate education and counseling about the known physical and neurologic changes that MOGAD has on sexual functioning. Because of the similarities in innervation between sexual and bladder function, patients with sexual dysfunction should be encouraged to empty their bladders before sexual stimulation to prevent inopportune incontinence. The mainstays of treatment of erectile dysfunction in
... Long-Term Care

men are inhibitors of cGMP phosphodiesterase, type 5, which will allow most men with spinal cord damage to achieve adequate erections for success in intercourse through a combination of reflex and/or psychogenic mechanisms. Although less effective in women, these same types of medications have been shown capable of enhancing a woman's sexual functioning. The most commonly used oral erectile dysfunction drugs are Viagra (sildenafil), Levitra (vardenafil), and Cialis (tadalafil). Although sexual experience is impacted by spinal cord injury, sensual experience and even orgasm are still possible. Lubricants and aids to erection and ejaculation (for fertility) are available. Adjustment to altered sexuality is aided by an attitude of permissive experimentation, as the previous methods and habits may no longer serve.

Skin Breakdown

Skin breakdown occurs if the skin is exposed to pressure for a significant amount of time, without sensation or the strength to shift position as necessary. Sitting position should be changed at least every 15 minutes. This can be accomplished by standing, by lifting the body up while pushing down on armrests, or by just leaning and weight shifting. Wheelchairs can be supplied with either power mechanisms of recline or tilt-in-space to redistribute weight bearing. A variety of wheelchair cushions are available to minimize sitting pressure. Redness that does not blanch when finger pressure is applied may signal the beginning of a pressure ulcer. Good nutrition, vitamin C, and avoidance of moisture all contribute to healthy skin. Pressure ulcers are much easier to prevent than to heal.

Spasticity

Spasticity means stiffness or muscle spasms and is often a very difficult problem to manage. Some stiffness in our muscles is necessary in order to control our movement, but when they become too tight, the result can range from slightly bothersome stiffness (particularly upon wakening) to uncontrollably painful spasms. When the latter occurs, small triggers such as changes in position, temperature, humidity, or presence of infections can cause this painful spasticity. The key goal is to remain flexible with exercise, a daily stretching routine, and a bracing program with splints, as needed. These splints are commonly used at the ankles, wrists or elbows. Also recommended are appropriate strengthening programs for the weaker of the spastic muscles acting on a joint and an aerobic conditioning regimen. These interventions are supported by adjunctive measures that include antispasticity drugs (e.g., diazepam, baclofen, dantrolene, tizanidine), therapeutic botulinum toxin injections, and serial casting. In cases where spasticity is severe, a baclofen pump, which provides the medication directly to the spinal cord, may be considered. The therapeutic goal is to improve function in performing specific activities of daily living (i.e., feeding, dressing, bathing, hygiene, mobility) through improving the available joint range of motion, teaching effective compensatory strategies, and relieving pain. Left untreated, severe spasticity can lead to shortening of the affected muscle or joint called contractures, further impacting mobility, rehabilitation, and independence.

Pain

Changes in sensation often occur and can manifest as lack of sensation, or numbness, as well as painful sensations called neuropathic pain. This pain is described in many different ways, including burning, squeezing, stabbing, or tingling. Having the
sensation of pain means the nerve signal is getting through, but in an inappropriate way. While this can get better over time, there is a long list of medications to treat these symptoms. The same medication doesn't work for everyone, so the trial and error of finding the right medication can be frustrating. Alternative therapies such as acupuncture and meditation have also been utilized, with varying success.

While the body is constantly working toward repair, once damage is done to the central nervous system, there will always be evidence of this damage, usually evidenced on an MRI. Clinical fluctuations of old symptoms, particularly in the setting of infection, stress, heat (Uthoff’s phenomenon), menstrual cycle, or anything that increases core body temperature or throws the body off of its normal course are also possible. It is important to note that this is not inflammatory driven and therefore in no way represents worsening of the condition.

The first step in treating pain effectively is obtaining an accurate diagnosis. Unfortunately, this can be very difficult. Causes of pain include muscle strain from using the body in an unaccustomed manner, nerve compression (i.e., compression of the ulnar nerve at the elbow due to excessive pressure from resting the elbow on an armrest continuously) or dysfunction of the spinal cord from the damage caused by the inflammatory attack. Muscle pain might be treated with analgesics, such as acetaminophen (Tylenol), non-steroidal, anti-inflammatory drugs such as naproxen or ibuprofen (Naprosyn, Aleve, Motrin), or modalities such as heat or cold. Nerve compression might be treated with repositioning and padding (i.e., an elbow pad for an ulnar nerve compression).

Nerve pain can be a significant challenge to find effective treatment. Nerve messages traveling through the damaged portion of the spinal cord may become scrambled and misinterpreted by the brain as pain. Besides the treatments listed above, certain antidepressants such as amitriptyline (Elavil), or anticonvulsants, such as carbamazepine, phenytoin, or gabapentin (Tegretol, Dilantin, Neurontin) may be helpful. Stress and depression should also be addressed since these conditions make pain harder to tolerate.

**Depression**

Individuals with MOGAD should be educated about the effect of MOGAD on mood regulation and routinely screened for the development of symptoms consistent with clinical depression. Warning signs that should prompt a complete evaluation for depression include failure to progress with rehabilitation and self-care, worsening fixed low mood, pervasive decreased interest, and/or social and professional withdrawal. A preoccupation with death or suicidal thoughts constitutes a true psychiatric emergency and should lead to prompt evaluation and treatment. Depression is not due to personal weakness or the inability to “cope.” It can have devastating consequences; not only can depression worsen physical disability (such as fatigue, pain, and decreased concentration) but it can have lethal consequences.

During the early recovery period, family education is essential to develop a strategic plan for dealing with the challenges to independence following a return to home.
Ongoing problems typically include ordering the appropriate equipment, dealing with re-entry into school, work, and community, and coping with the psychological effects of this condition on both those diagnosed with MOG antibody disease and their families. Being saddened or demoralized by the diagnosis of MOG antibody disease is appropriate. The inability to move past this grief in a reasonable period of time such that it interferes with relationships and functional living needs to be addressed and treated. Many fear that depression reflects on oneself as an inadequate ability to cope with their diagnosis and feel weak. But it is not a personal strength issue, and depression is very much a physiological manifestation and treatable. Both talking to a psychiatrist/psychologist and medication management can be beneficial, and some studies indicate a synergistic effect of combining the two. Depression can rebound and can at times become more resistant to treatment.

**Fatigue**

Fatigue is the lack of mental and/or physical energy. Fatigue can be a direct result of a disease process (primary fatigue) or an indirect result (secondary fatigue). In MOG antibody disease, fatigue is more often thought to be a result of secondary fatigue. Examples of secondary fatigue include fatigue from medications, depression, stress, poor sleep patterns, infections, or changes in walking, which increase energy requirements. The key is to try to identify the underlying cause of the fatigue – for example, if one is not sleeping well because of pain, bladder dysfunction, or depression, this needs to be identified and addressed; not getting consistent sleep will worsen every other aspect of MOG antibody disease! If too much energy is exerted due to changes in walking, physical therapy can help identify better body mechanics that will help conserve energy. When nothing else can be identified as contributing to fatigue, REST is recommended!Conserving energy such that activities are planned and paced can allow for these activities to be more enjoyable rather than stressful. Also, reorganizing home and office can help to reduce the amount of wasted energy exerted so that energy can be saved up for activities that are enjoyable. Also, exercise routines incorporated in the day can actually help build stamina and reduce fatigue in the long-run – it’s also a great stress reducer! Pilates, yoga, and swimming are great, but the key is to find something enjoyable and not overdo it.

**Rehabilitation and Activities of Daily Living**

An appropriate strengthening program and an aerobic conditioning regimen are recommended. The effects on mobility as a result of MOGAD can vary widely, however, from paralysis to mild weakness. Either way, physical therapy is instrumental in returning function. Because physical therapists deal with many different types of injuries and diseases, it is ideal to work with one who has a particular interest in spinal cord rehabilitation when possible. Assistive devices may be necessary for weakness – it can be difficult and oftentimes humbling to take the necessary step of using an assistive device, but when faced with the alternative of broken hips, heads, and the downstream effects of lost wages or jobs, it is an important and sometimes indispensable step in maintaining independence. It is also always very important to remember to exercise, as tolerated, in order to maintain physical health and stamina.
**Long-Term Care**

Individuals with MOGAD may find ordinary tasks such as dressing, bathing, grooming, and eating very difficult. Many of these obstacles can be mastered with training and specialized equipment. For example, long handled sponges can make bathing easier as can grab bars, portable bath seats and hand-held shower heads. For dressing, elastic shoelaces can eliminate the need to tie shoes while other devices can aid in donning socks. Occupational therapists are specialists in assessing equipment needs and helping people with limited function perform activities of daily living. A home assessment by an experienced professional is often helpful.

Physical therapists assist with mobility. Besides teaching people to walk and transfer more easily, they can recommend mobility aids. This includes everything from canes (single point vs. small quad cane vs. large quad cane) to walkers (static vs. rolling vs. rollator) and braces. For a custom-fabricated orthotic (brace), an orthotist is necessary. Careful thought should go into deciding whether the brace should be an ankle-foot orthosis, whether it should be flexible or stiff, and what angle the foot portion should be in relationship to the calf portion. Some will benefit by a knee-ankle foot orthosis. Each person should be evaluated individually. The best results occur when a physician coordinates the team so that the therapists and orthotists are united on what is to be achieved. The physician best trained to take this role is the physiatrist.

---

**Additional Resources**

**Myelitis Helpline**

srna.ngo/helpline

For questions about our organization and rare neuroimmune disorders, visit the Myelitis Helpline, an online tool developed by SRNA.

**Resource Library**

srna.ngo/resources

To access up-to-date resources on rare neuroimmune disorders, which include symposium videos, magazines, podcast recordings, published research summaries, information sheets and relevant external resources, visit our Resource Library.

---

**References**


