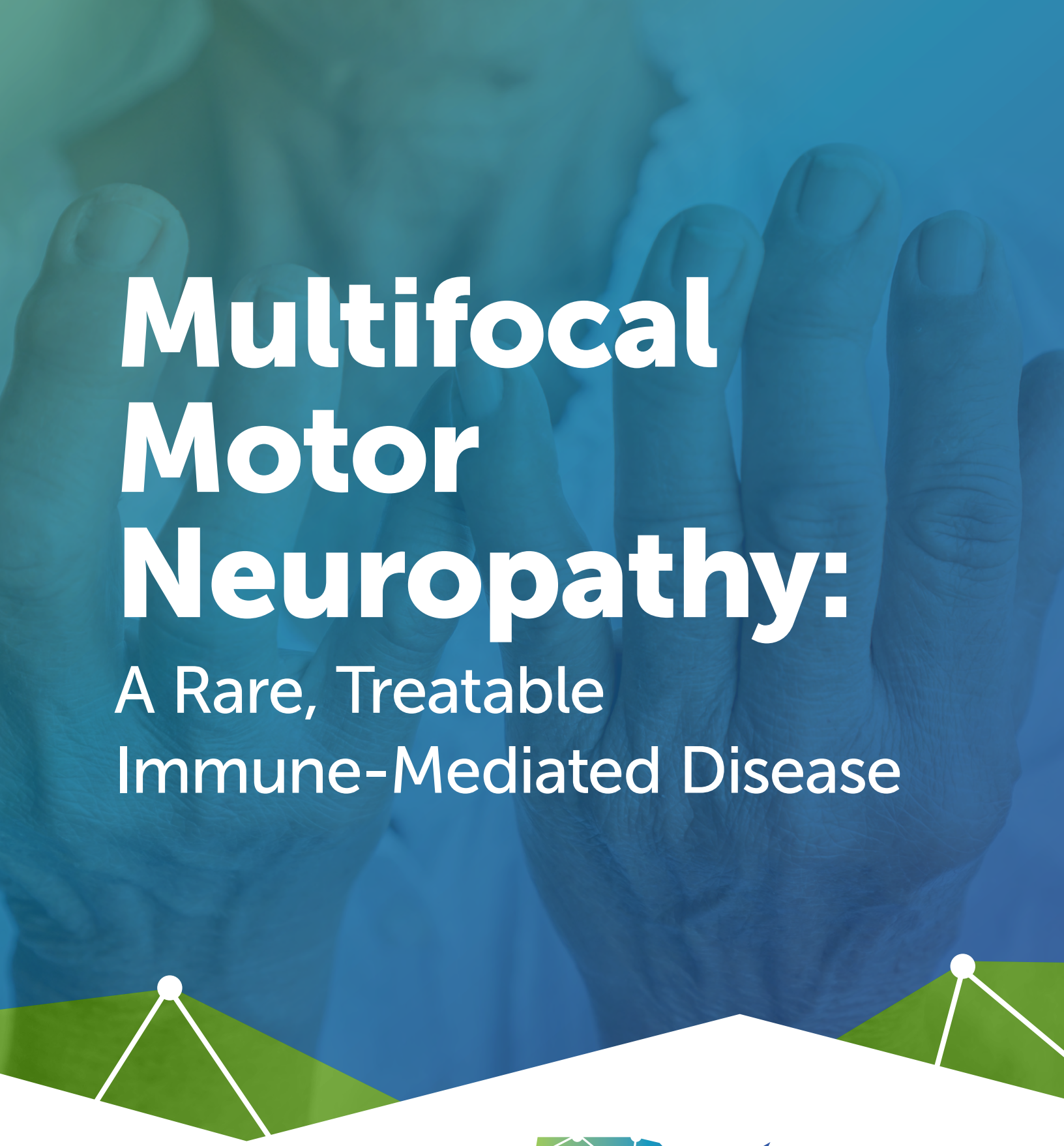

Multifocal Motor Neuropathy:

A Rare, Treatable Immune-Mediated Disease

A Clinical Proceedings White Paper

July 2026

Clinical Neurological Society of America

CNSA's Clinical Proceedings

The Clinical Neurological Society of America has more than 50 years of experience bringing together leading experts and clinical neurologists for educational programming. CNSA's Clinical Proceedings—a white paper series—are informational resources intended to raise awareness and address unmet needs in neurology. CNSA recognizes the expert panel members who contributed to the development of this white paper.

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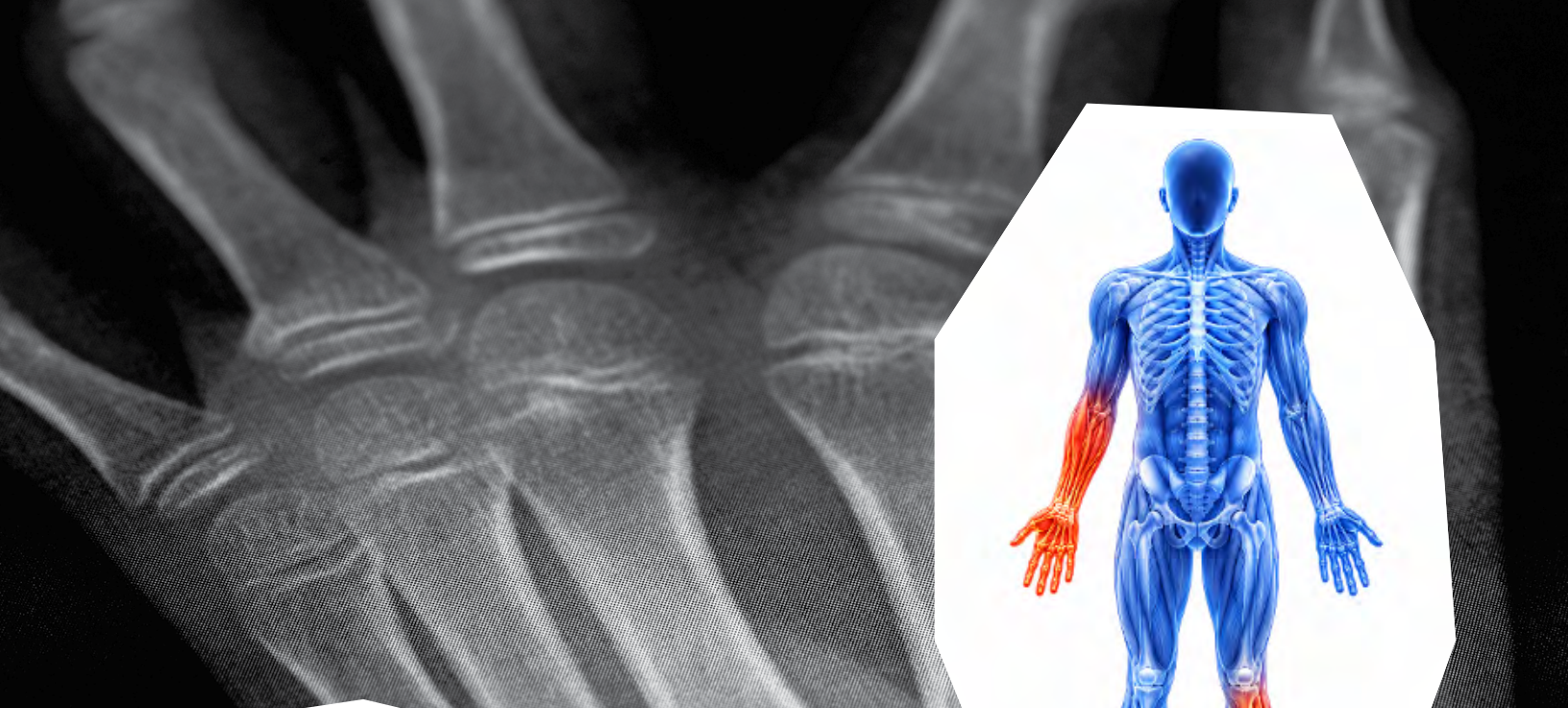
Introduction

Multifocal motor neuropathy (MMN) is a rare, immune-mediated disease characterized by muscle weakness that preferentially affects distal upper limb muscles. Patients often experience difficulties with activities like drawing, buttoning shirts, and gripping objects, all of which require fine motor skills.

Multifocal motor neuropathy affects fewer than 1 person in 100,000 and is more common in men than women by a ratio of about 2 or 3 to 1.[1-3] Symptoms often appear around age 45, but can begin as early as the teenage years or as late as the 60s.[2,4] There is no cure for MMN and in most cases long-term immunotherapy is needed to prevent or minimize disease worsening.



Clinical Signs and Symptoms

Symptoms and signs of MMN first appear in distal limbs, preferentially affecting the upper extremity

Approximately 60-80% of people with MMN first experience asymmetric weakness in the distal upper limb, whereas others rarely first experience weakness in the distal lower limb.[4] The disease is progressive and eventually affects the distal upper limb of nearly all patients. Finger extensor muscles are typically more affected than finger flexor muscles. Cold paresis (worsening of strength in cold environments) is a common complaint in patients affected by MMN.[4]

Fasciculations have been documented in about a quarter of patients and cramps in about 40%.[5] Cramps and fasciculations are usually restricted to clinically weakened limbs. Deep tendon reflexes are often reduced in the affected limbs but may be normal in limbs that are not affected by weakness. Patients with MMN do not experience sensory symptoms such as paresthesia and pain, although approximately 20% of patients exhibit a mildly impaired abnormal vibration sense typically in the distal lower limbs, particularly with longer duration of disease.[3]

As MMN progresses, patients can experience further reductions in strength and an increase in self-reported symptoms and number of affected muscle groups.[6] Muscle atrophy is an eventual complication in nearly all cases, but in the early disease stages, weakness is often disproportionate to atrophy—a potential diagnostic clue.[4]

Etiology and Pathophysiology

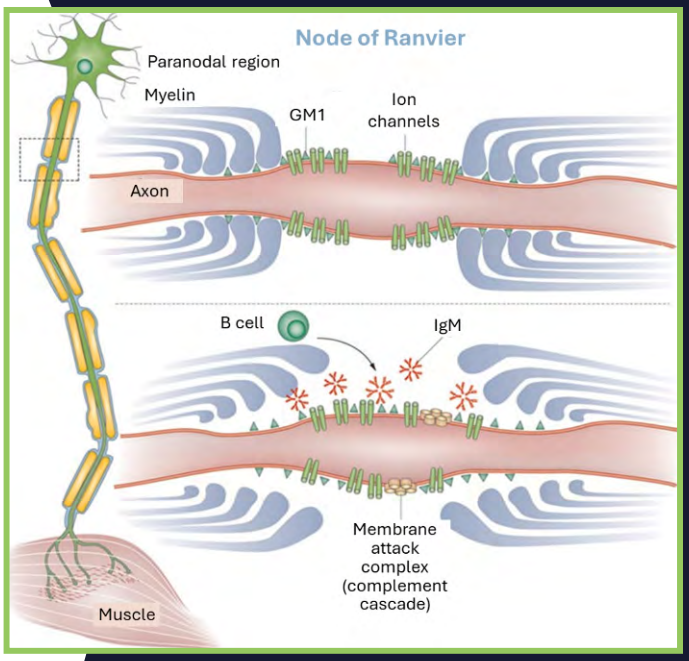
Autoimmune targeting of motor nerves

Multifocal motor neuropathy is an immune-mediated peripheral nerve disorder.[7] Approximately 30-50% of people with MMN have serum antibodies against GM1 ganglioside.[5,8] However, the percentage of patients who are positive for GM1 varies based on the sensitivity of the test and can be 75% or higher with specialized assays.[9] Conversely, GM1 antibodies, typically in low titers, can rarely be detected in disorders other than MMN, including amyotrophic lateral sclerosis (ALS).

> “Tests for the detection of GM1 antibodies have different sensitivities. Just because patients do not test positive for anti-GM1 antibodies doesn’t mean that you aren’t dealing with MMN.”
>
> **–Karissa Gable, MD, FAAN**

Anti-GM1 antibodies can damage motor nerves directly by disrupting ion channel function and signaling pathways, as well as indirectly through activation of the complement pathway.[11] The final stage in complement activation is a membrane attack complex that disrupts ion channel clustering and the nodal/paranodal structure of motor neurons; these actions interrupt nerve signaling and result in muscle weakness.

Autoimmune targeting of motor nerves in multifocal motor neuropathy.
Multifocal motor neuropathy is believed to result from damage to the nodal and paranodal regions of motor neurons, areas at which ion channels and GM1 gangliosides are concentrated. In at least a portion of people with MMN, B cells produce IgM antibodies against GM1 that can initiate a complement cascade, resulting in a membrane attack complex that damages axons.



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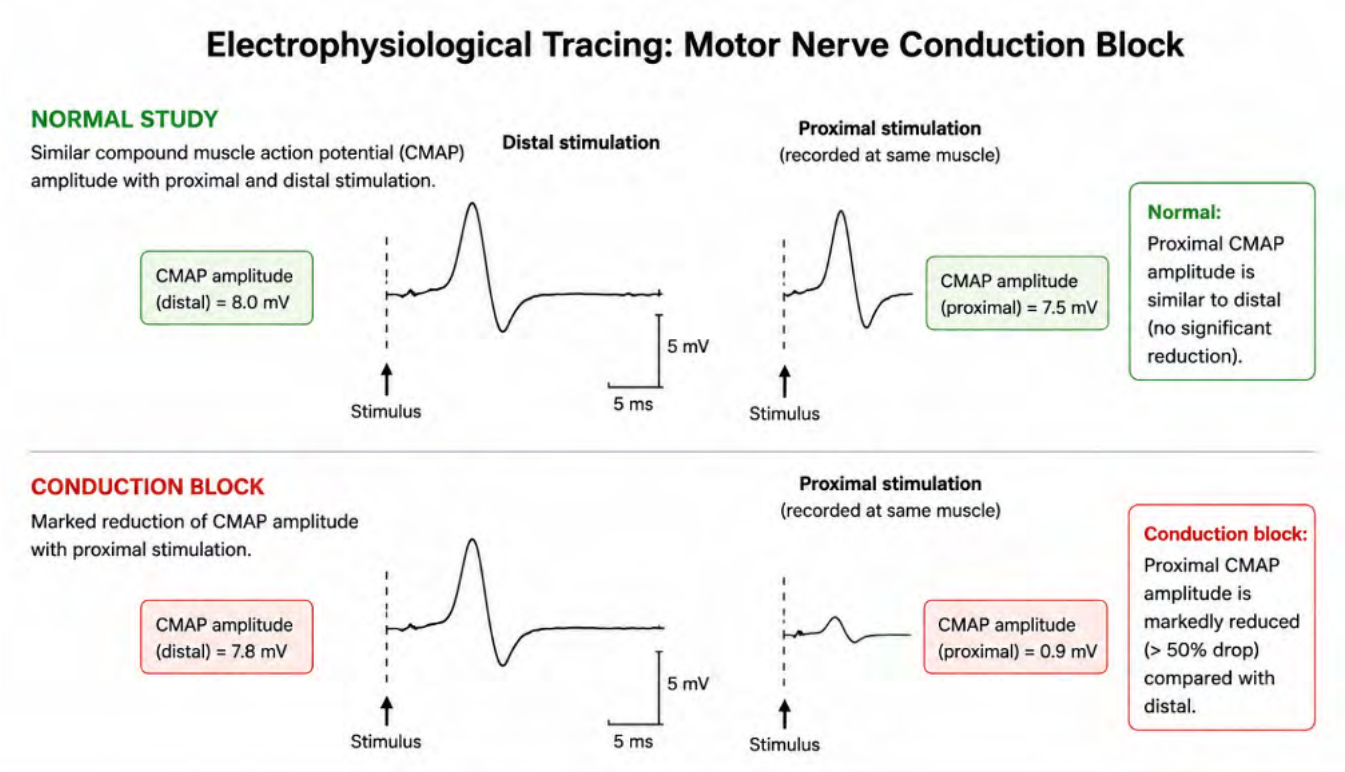
The presence of anti-GM1 antibodies and their consequences for motor nerve function are the leading hypothesis for MMN pathophysiology. However, it should be noted that anti-GM1 antibodies are somewhat non-specific and can be detected in a portion of people who are neurologically normal or have been diagnosed with ALS or other neuropathies.[11] Thus, the exact nature of the link between GM1 antibodies and MMN is not fully understood.

Motor nerve conduction block

Anti-GM1 antibodies and the subsequent complement cascade are believed to result in motor nerve conduction block, a key electrophysiological feature of MMN.[11] Motor nerve conduction block is an electrophysiological finding in which proximal nerve stimulation produces a much smaller electrical response than distal nerve stimulation—for instance, stimulation of a nerve at the elbow (proximal) may lead to a reduced electrophysiological response compared with stimulation at the wrist (distal) when recorded at the same muscle. Specific electrophysiological criteria must be met to be considered conduction block.[12]

> “Chronic conduction block in MMN is an important consideration because it can lead to axonal degeneration.”
>
> **–Mamatha Pasnoor, MD, FAAN**



Diagnosis and Mimics

Diagnostic criteria

Multifocal motor neuropathy was first recognized as a separate diagnostic entity in the 1980s.[13,14] Diagnostic criteria from the European Federation of Neurological Societies/Peripheral Nerve Society updated in 2010 require the following:[15]

(1) slowly progressive or stepwise progressive, focal, asymmetric limb weakness in the motor nerve distribution of at least 2 nerves for more than 1 month, usually more than 6 months; and

(2) no objective sensory abnormalities, except for minor vibration sense abnormalities in the lower limbs.

Supportive clinical criteria are predominant upper limb involvement, decreased or absent tendon reflexes in the affected limb, absence of cranial nerve involvement, cramps and fasciculations in the affected limb, and response to immunomodulatory treatment. Multifocal motor neuropathy is excluded as a diagnosis in the presence of upper motor neuron (UMN) signs, marked bulbar involvement, marked sensory loss, and diffuse symmetric weakness during the initial weeks of disease.[15]

Most people with MMN exhibit nerve conduction block.[16] Conduction block is highly supportive of an MMN diagnosis but may be difficult to identify under some conditions. If the location of the conduction block is in a nerve segment that is not assessable with a nerve conduction study (such as a very proximal segment), it may be difficult to detect. Nerves with severe axon loss may also fail to demonstrate conduction block. The electrophysiologic guidelines recommend at least 50% proximal to distal conduction block for definite MMN and at least 30% for probable conduction block for MMN in a non-compressible site. Importantly, sensory nerve conductions are normal in the limb with conduction block.[15]

> “Conduction block can help confirm a diagnosis of MMN. Detection can be tricky, for instance, if the conduction block is very proximal. We now know that conduction block can’t be proven in everyone with MMN, but it definitely assists with the diagnosis.”
>
> **–Kyle Ruffing, MD, FAAN**

Differential diagnosis

Differential diagnosis of MMN involves exclusion of the following conditions:[4,17]

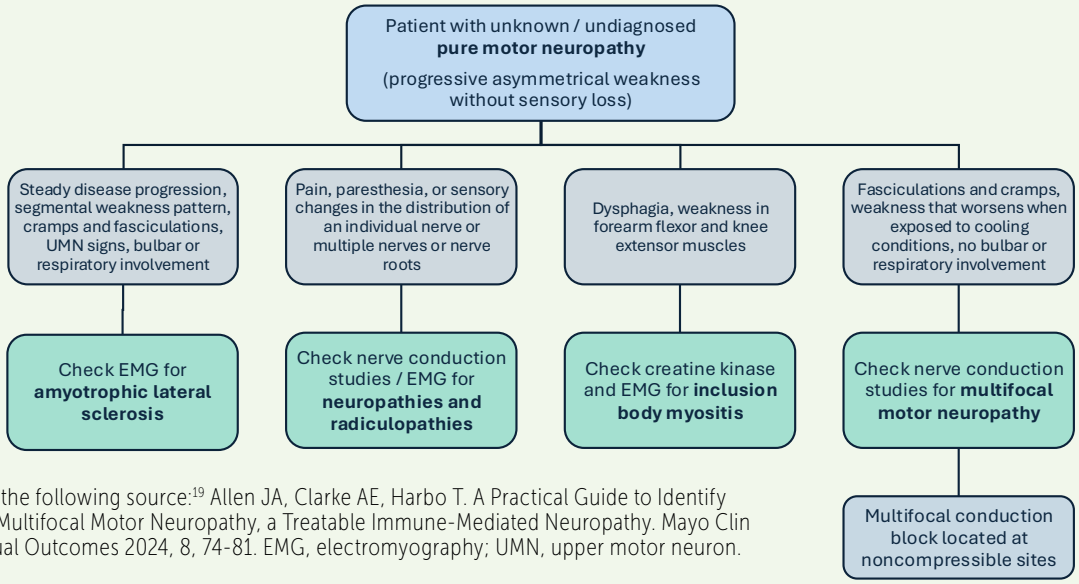
- **Amyotrophic lateral sclerosis (ALS):** Distinguished by the presence of upper motor neuron signs, usually more prominent muscle atrophy, absence of conduction block
- **Chronic inflammatory demyelinating neuropathy (CIDP):** Distinguished by sensory involvement and symmetric weakness, although multifocal CIDP has asymmetric presentation of weakness
- **Spinal muscular atrophy (SMA):** Has an established genetic cause, symmetric weakness, proximal more than distal involvement
- **Inclusion body myositis (IBM):** Typically affects quadriceps and finger flexors; knee extension weakness and dysphagia are common, absence of conduction block, shows characteristic pathology upon muscle biopsy, needle electromyography is myopathic

In a survey of 55 patients with MMN, initial misdiagnosis was common, led by ALS (25%) and compression neuropathies such as carpal tunnel syndrome and ulnar neuropathy (25%).[18]

> "As a general neurologist, I've seen three patients with MMN in over 30 years. One of my patients was misdiagnosed with ALS—a mimic that still fools neurologists."
>
> **–Christina Mayville, MD**

Other conditions that should be considered in the differential diagnosis of MMN are peripheral nerve compression or entrapment, vasculitic neuropathy, toxic/paraneoplastic neuropathy, Hirayama disease, and hereditary neuropathy with liability to pressure palsies (HNPP).[4] Neuralgic amyotrophy may be considered in the initial stages of disease when patients present with the involvement of only one nerve; as other nerves become involved, a diagnosis of MMN becomes more obvious.

General approach for identification of patients with motor neuropathy.



Adapted from the following source:[19] Allen JA, Clarke AE, Harbo T. A Practical Guide to Identify Patients With Multifocal Motor Neuropathy, a Treatable Immune-Mediated Neuropathy. Mayo Clin Proc Innov Qual Outcomes 2024, 8, 74-81. EMG, electromyography; UMN, upper motor neuron.

Impact and Burden

Multifocal motor neuropathy is a lifelong disease with no cure and although it is not typically a fatal condition, it has a substantial negative impact on patients' lives. At least half of those afflicted report activity limitations.[20] These often involve fine motor skills of the hand, such as buttoning clothes, writing, typing, using tools, and manipulating small objects. Patients are often unable to turn keys, open jars, carry or lift objects, or grip utensils due to reduced grip strength and weakness. In a study of 88 patients with a mean disease duration of 11 years, 82% reported moderate or severe impairment of the arms as assessed by Overall Disability Sum Scores (ODSS).[3]

Distal lower limb weakness can limit walking ability, ranging from an abnormal gait to reliance on a wheelchair.[3,21] Fatigue can be debilitating for many patients and quality of life is significantly reduced.

Multifocal motor neuropathy often progresses over time, with patients showing a decline in muscle strength and increased muscle atrophy and disability.[6,22]

Patients

A major problem encountered in MMN is lack of timely diagnosis. Patients often visit multiple physicians and undergo numerous medical tests for years before receiving a diagnosis of MMN.[18,23] Additionally, patients report that their symptoms have been dismissed by physicians who lacked sufficient understanding and knowledge of their condition.[23]

The impairment associated with MMN leads to reduced quality of life, and chronic disability reduces mental wellbeing and contributes to the psychological burden.[24] The disease particularly interferes with the ability to function physically and socially, with patients often foregoing regular social activities.[18,24]

Multifocal motor neuropathy also impacts the ability to work. In a survey of 214 patients with MMN, half indicated that the disease often or always interferes with their employment.[25] In

another survey of 55 patients with MMN, nearly all (95%) indicated that their symptoms interfered with work, with muscle weakness (57%) and fatigue (33%) having the greatest impact.[18] Patients missed an average of 87 days of work over the past year due to their disease.

Notably, MMN usually develops during the middle years of life when patients are working, taking care of children, and managing family and household roles—a life stage with peak occupational productivity and responsibilities.

Families and caregivers

As MMN progresses, patients may become more functionally disabled and reliant on family members and caregivers. This can lead to caregiver strain, including psychological and physical fatigue.[26]

Patients' reduced ability to work can lead to financial strain and may necessitate changes in the home environment to accommodate the disease. Frequent physician visits, medication costs, emergency department visits, and hospitalizations are an additional burden on families.[18,27]

Healthcare system and society

Although MMN is a rare disease, its persistent and lifelong course contributes to an important impact on the healthcare system and society. A large study using a retrospective database of healthcare claims compared costs for MMN with those of MMN mimics such as ALS, carpal tunnel syndrome, spinal muscle atrophy, neuralgic amyotrophy, and others.[27] In the year before diagnosis, the MMN cohort had significantly higher MMN-related healthcare costs than the MMN-mimic cohort, costing more than $23,000 per person on average ($23,625 MMN vs $12,890 MMN-mimic). In the year after diagnosis, the MMN cohort was associated with significantly higher healthcare costs than the non-MMN cohort, reaching an average of nearly $75,000 per person ($74,187 MMN vs $50,652 MMN-mimic). Thus, MMN is an expensive disease, even compared to other similar neurological conditions—which are themselves expensive.

Barriers to Timely Diagnosis

As noted previously, many patients ultimately diagnosed with MMN experience diagnostic delays of a year or more.[18,23] As a rare disease, MMN is not top of mind for general practitioners or neurologists. Additionally, many patients lack access to a specialist or even a neurologist who has encountered MMN in their practice, particularly in rural areas. Physician education related to MMN may be beneficial in both residency and continuing education programs to help increase awareness of the disease and ensure that MMN is part of the differential diagnosis.

An important impediment to the timely diagnosis of MMN is the lack of a biomarker or clinical assessment that reliably distinguishes MMN from other related diseases. The symptoms and signs of MMN overlap with a variety of conditions, and supportive features such as the presence of motor nerve conduction block and the presence of GM1 antibodies are not definitive or exclusive. Emerging diagnostic tools such as neuromuscular ultrasound and magnetic resonance imaging (MRI) may be used to detect nerve enlargement or swelling.[11] However, no algorithm is yet established for using these tools to reliably distinguish MMN from other conditions.

Updated diagnostic guidelines for MMN would also be beneficial. The most recent clinical diagnostic guidelines for MMN are from 2010[15] and the electrophysiological guidelines are from 2003.[16] The European Academy of Neurology and Peripheral Nerve Society have indicated their intention to update diagnostic guidelines,[28] which would be welcome to further differentiate MMN from other conditions, clarify the role and interpretation of conduction block, and incorporate other diagnostic tools.[11]

Treatment and Monitoring

Intravenous immunoglobulin (IVIG)

The primary treatment for MMN is intravenous immunoglobulin (IVIG), which is approved by the United States Food and Drug Administration (US FDA) as a first-line therapy. Approximately 70-94% of MMN patients treated with IVIG show improved muscle strength that lasts about 4-6 weeks.[4,11,29] Per professional treatment guidelines, maintenance doses of IVIG of 1 g/kg may be given every 2 to 4 weeks or 2 g/kg every 1 to 2 months, guided by patient responses.[15]

Although IVIG can improve muscle strength and disability, it does not necessarily prevent disease progression.[22] Over a period of years, some nerves show improvements such as reinnervation and disappearance of conduction block, whereas others show demyelination and new conduction block. Clinical studies have found that more severe disability or weakness is associated with axon loss and years untreated.[3,21] Consequently, treatment of MMN and appropriate adjustments during the disease course should begin early to help prevent axonal loss and minimize disability.[11]

> “Although patients do improve with treatment, most still experience functional decline and progression of disability. This is frustrating to patients who view their disease as treatable. MMN can still be a progressive disease even though it responds to treatment.”
>
> **–Kyle Ruffing, MD, FAAN**

As MMN progresses, patients may require more frequent and higher doses of IVIG, although the treatment regimen varies based on individual patient needs.[30] It is extremely important to monitor each patient over time so that treatment can be adjusted as needed.

Monitoring Progression of MMN

To help prevent axon loss and minimize disability, MMN patients should be regularly monitored so that treatment can be adjusted as needed. In addition to regular clinical exams, patients should undergo the following tests:

- Grip strength using a hand dynamometer that provides an objective reading that can be compared over time AND
- MMN-RODS (Multifocal Motor Neuropathy Rasch-built Overall Disability Scale), a scale specifically designed to assess MMN-related function and impact on daily life.

> Treatment timing is variable among patients. Some patients already have axonal damage by the time they reach our office, and that is difficult to reverse. But the more recent weakness often can be improved with more frequent IVIG treatments."
>
> **–Mamatha Pasnoor, MD, FAAN**

> Patients need to be followed regularly and monitored closely over time, not only via clinical exam but also using other outcome measures such as grip strength. In the absence of serologic biomarkers to assess disease activity, close monitoring is essential to re-assess outcomes and modify treatment as needed."
>
> **–Karissa Gable, MD, FAAN**

Adverse effects of IVIG can include headache, chills, nausea, malaise, and myalgia.[4] Rare, serious side effects may also occur, including aseptic meningitis, acute renal failure, congestive heart failure, and thromboembolic events.

Although IVIG improves the symptoms of MMN in most patients, it can be incredibly time consuming. Each infusion may last 3 hours or more every 2 to 4 weeks. Many patients are able to receive IVIG treatments at home, but others must travel to infusion centers weekly or monthly.[18] This means that life must be planned around MMN therapy.

> The treatment itself is a complicated undertaking for patients—it's not like swallowing a pill. For some patients the stress of the needles and procedures can be emotionally daunting and takes some adjustment."
>
> **–Irina Rozenfeld, ANP-BC**

Subcutaneous immunoglobulin (SCIG)

For patients who are responsive to IVIG but seek a more convenient treatment, subcutaneous immunoglobulin (SCIG) is often recommended, although it is not formally approved by the FDA for MMN. The benefits of SCIG are similar to IVIG with potentially better tolerability.[4] An important advantage of one type of SCIG is that patients do not need to go to an infusion center or depend on a visit from an infusion nurse, allowing them more independence. However, not all patients will want to administer their own injections. There is another option for SCIG that can be infused by a nurse if assistance is needed for administration. Like IVIG, SCIG does not suppress the underlying disease process or lead to remission of MMN.

> Some patients prefer subcutaneous treatment because it allows them more independence. But most formulations of SCIG require self-administration and patient education."
>
> **–Irina Rozenfeld, ANP-BC**

Other existing therapies

Patients who do not respond to IVIG or SCIG have few other treatment options. Neither steroids nor plasmapheresis is effective and may actually worsen MMN. Cyclophosphamide and rituximab have been evaluated for MMN but lack strong evidence for efficacy and both therapies carry their own risks.[4,15]

Future Therapies

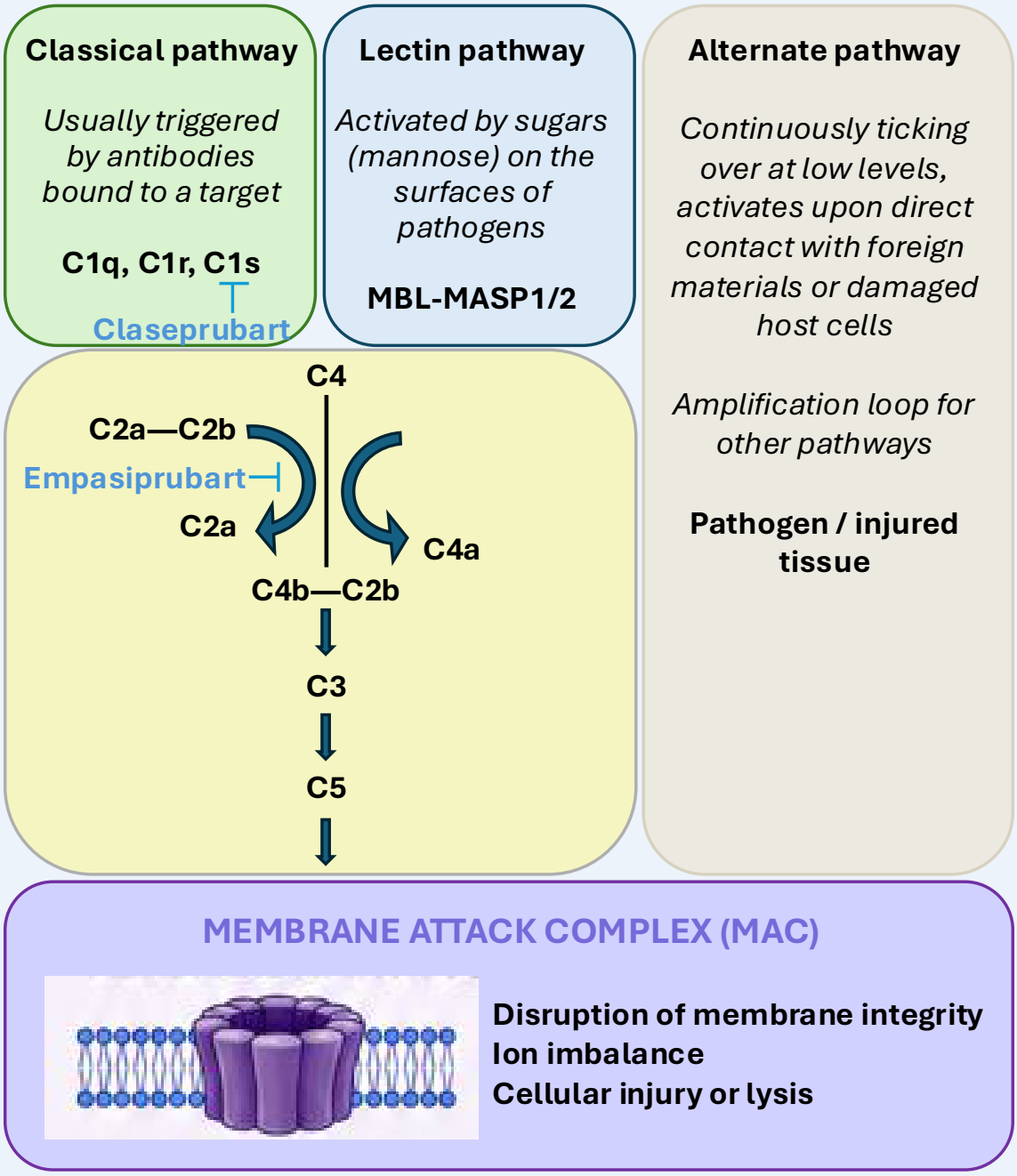
Given that treatment with IVIG and SCIG do not halt progression of MMN, new therapies are needed. Several medications currently in development focus on inhibiting the complement pathway due to its role in axonal damage. The complement system is involved in the body's immune defense against microbes and inhibiting this system may increase the risk of infection, although this is likely to depend on the particular step in the complement pathway targeted by the medication.[31] Whether vaccinations and immune monitoring are needed with complement therapies will be determined by future research.

Empasiprubart (ARGX-117)

Empasiprubart is a humanized monoclonal antibody that binds to C2—a protein in the complement pathway that has been associated with nerve damage in MMN. Empasiprubart has been found to reduce C2 levels in humans.[32] In a randomized, controlled, phase 2 study of MMN patients, intravenous administration of empasiprubart substantially improved muscle strength, grip strength, and patient disability compared with placebo and reduced the need for IVIG re-treatment.[33] Most adverse events were mild or moderate in severity and a phase 3 study comparing empasiprubart to IVIG is currently underway.

Claseprubart (DNTH103)

Claseprubart is a monoclonal antibody that binds to activated C1—another protein in the complement pathway that has been associated with nerve damage in MMN. Intravenous administration of claseprubart is being studied in a randomized, controlled phase 2 study for the treatment of MMN.[34]



Overview of Complement Pathways.

The complement system is a vital part of the immune system that aids in clearing microbes and damaged cells. It can be activated through three distinct mechanisms as shown in this diagram. Claseprubart inhibits C1s in the classical pathway and empasiprubart inhibits C2, part of both the classical and lectin pathways. These medications likely do not affect the alternative pathway, which does not appear to be as involved in MMN but is important for maintaining some residual immune function. For details see Van de Walle et al, 2025[32] and Burgelman et al, 2023.[35]

Importance of Multidisciplinary Therapy

As a chronic neurological disease that affects many aspects of life, MMN is best treated from a multidisciplinary perspective.[4,8,11,28] The following professionals are ideally involved, although in reality many healthcare systems and practices lack access to a full multidisciplinary team.

Neurologist:
provides diagnosis; prescribes and adjusts medical therapy; monitors disease progression

Physical therapist:
engages patients in strengthening and range of motion exercises; helps prevent contracture and preserve functional mobility

Occupational therapy:
trains patients on tasks that help maintain fine motor skills (self care, activities of daily living)

Orthotist:
designs and fits splints and braces to prevent joint deformities and stabilize muscles

Pharmacist:
assists with obtaining insurance coverage for IVIG; helps navigate home versus hospital infusions

Nurse:
administers IVIG at home or in the clinic and monitors tolerability/adverse events; provides patient education; covers many other tasks in practices that lack other professionals, including navigating the healthcare system and coping with the disease and its treatment

Neuropsychologist:
helps with fatigue, mood, anxiety, and coping strategies

Social worker:
helps navigate the healthcare system, insurance, and home support

Conclusion

Multifocal motor neuropathy is a rare immune-mediated disease that is difficult to diagnose but important to identify and treat early to help prevent the loss of motor axons and minimize disability. Current treatment with IVIG or SCIG is effective but does not prevent disease progression, and patients benefit from lifelong care from a multidisciplinary team of healthcare professionals. Several new therapies are in the pipeline for MMN that will hopefully reduce the burden of disease and enable patients to participate more fully in their lives.

About the Clinical Neurological Society of America

Established in 1974, the Clinical Neurological Society of America is a non-profit 501(c)(6) made up of neurologists and other healthcare professionals practicing in clinical and academic settings. CNSA's mission is to improve clinical practice and patient care through education and thought leadership. CNSA is led by a volunteer board of directors who, like the society's members, hail from across the country and treat patients with a range of neurologic conditions.

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