What is Chronic Inflammatory Demyelinating Polyneuropathy (CIDP)?

Chronic Inflammatory Demyelinating Polyneuropathy is a rare disorder of the peripheral nerves characterized by gradually increasing sensory loss and weakness associated with loss of reflexes.

While GBS and CIDP share many features, one that separates them is the onset: in GBS the onset to maximum weakness occurs in under 30 days and in most people in under 14 days, while in CIDP the sensory loss and weakness progress beyond those times. The incidence of new cases of CIDP is about 1-4 per million people but as the disease can be present in any one person for a long time, the prevalence may be as high as 9 per 100,000.

Like GBS, CIDP is caused by damage to the covering of the nerves, called myelin. It can start at any age and is more frequent in men than women. Unlike GBS, the active phase of CIDP is not limited to less than a month. Although in about 1/3rd of patients the disease can go into a stage of remission where no immune treatments are needed, most with CIDP experience slow progression or relapses over years or more. Left untreated, 30% of CIDP patients will progress to wheelchair dependence. Early recognition and proper treatment can avoid a significant amount of disability.

MISSION STATEMENT

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- We fund **research** through grants, establishing fellowships and other appropriate avenues to identify the causes of and discover treatments;
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MORE INFORMATION

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CIDP

Chronic Inflammatory Demyelinating Polyneuropathy

SUPPORT EDUCATION RESEARCH ADVOCACY

Working for a future where every person affected by GBS, CIDP, MMN or a related variant, will have access to early and accurate diagnosis, appropriate treatment and knowledgeable support services.

WHAT CAUSES CIDP?

Current theory holds that the body's immune system, which normally protects itself, perceives myelin as foreign and attacks it. Myelin is an important part of the peripheral nervous system. It wraps around the nerve axon (the long, wire-like part of a nerve cell) much like insulation around an electrical wire. The nerves extend from the spinal cord to the rest of the body, stimulating muscle contraction and transmitting sensory information back to the nervous system from receptors in the skin and joints. This insulation (myelin) allows electrical impulses to efficiently travel along the nerve axon. When myelin is damaged or removed, these electrical impulses are slowed or lost, and messages transmitted from the brain are disrupted and may never make it to their final destination. What causes this process is not yet clear.

HOW IS CIDP DIAGNOSED?

Diagnosis of CIDP is based on the symptoms of the patient:

- Symptoms such as loss of sensation (numbness), abnormal sensation (tingling and pain), loss of reflexes, and weakness (difficulty walking, foot drop)
- Tests such as nerve conduction and EMG (usually showing a demyelinating neuropathy), spinal fluid analysis (usually showing elevated protein with normal cell count), blood and urine tests (to rule out other disorders that may cause neuropathy and to look for unusual proteins)

HOW IS CIDP TREATED

There are three standard or first line treatments in CIDP:

- **Corticosteroids** (Prednisone, Prednisolone) are similar to naturally occurring anti-inflammatory hormones made by the body, and can be used as an initial treatment. Corticosteroids often improve strength, are conveniently taken by mouth, and are inexpensive. Side effects however can limit long-term use.
- **High dose Intravenous Immune Globulins** (IVIG) is the only drug that has FDA, Canadian, and European approval for treatment of CIDP. IVIG contains naturally occurring antibodies obtained from healthy volunteers. IVIG is given through a vein over the course of several hours. Newer preparations of higher concentrations that can be given under the skin (subcutaneous) are currently being tested in controlled trials in CIDP patients.
- **Plasma Exchange (PE), or Plasmapheresis** (PLEX), is a process by which some of the patient's blood is removed and the blood cells returned without the liquid plasma portion of the patient's blood. It may work by removing harmful antibodies contained in the plasma.
- **Subcuteaneous Immune Globulins** (SCIg) SCIg is commonly used in patients with immunodeficiency. SCIg is administered by patients themselves at home. Infusions are generally given in the fat under the skin in the stomach or thighs. It is approved by the FDA in the US for treatment in CIDP.

There are a large number of so-called second line drugs used to treat CIDP. These are used when the above standard treatments fail, cause significant side-effects, or the clinical response is not optimal. These drugs are largely not tested in randomized controlled trials, but their use is supported by case series from the medical literature.

There are a number of so-called third line treatments, usually chemotherapy drugs, but these should be given only in selected circumstances and by those with extensive experience in their use.

There are also ongoing research studies (see www.clinicaltrials.gov)

Centers of Excellence

Treatment of CIDP is an art. An experienced doctor is more likely to have good outcomes than someone treating their first case as is true throughout medicine. That is why we have set up the Centers of Excellence program. If treated early, most CIDP patients respond well to therapy that can limit the damage to peripheral nerves and contribute to improved function and quality of life and at times can cure the disorder altogether. Please visit gbs-cidp.org/support/centers-of-excellence for more information.

Although CIDP can affect children and adults of any age, the peak period of life during which patients typically develop this disorder is between 50 to 60 years of age. It is more common in men than women.

NEED HELP?

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What is Multifocal Motor Neuropathy (MMN)?

Multifocal Motor Neuropathy (MMN) is an acquired, chronic but treatable condition affecting multiple motor peripheral nerves that connect the spinal cord with the muscles. Damage to these nerves by the patient's immune system results in muscle weakness most often in the arms with minimal or no sensory changes. It develops in a chronically progressive or step-wise manner and over time leads to wasting or atrophy of the muscles controlled by the involved nerve.

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MMN

Multifocal Motor Neuropathy

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WHAT ARE THE SYMPTOMS OF MMN?

Males are affected with MMN almost three times as frequently as females and experience an earlier onset with a peak incidence between 50 and 60 years of age. Onset can occur between 20 and 60 years of age. The disease affects one to two people per 100,000 population.

Symptoms progress in a slow or stepwise fashion over 20 or more years. In the beginning, the weakness is most common in the hands as opposed to the legs and asymmetric — or not the same — on each side of the body. The hand weakness can result in significant disability for patients by interfering with their ability to write, button a shirt, handle a fork and knife or turn a key in a lock. Even if symptoms initially occur in the legs, over time the arms become more involved. During the disease course, patients may require the use of a cane or brace but generally do not become wheelchair dependent even late in life. Most patients can continue their jobs, unless the job is physically demanding. Muscles that are used for swallowing, speech and chewing are not involved.

Involved muscles may develop wasting (thinning) or atrophy over time and be associated with twitching of muscle fibers that can be seen under the skin. Patients, particularly at disease onset, have normal sensory function and can feel pain, cold or hot sensations and touch. In rare cases, some sensory loss may occur over time.

WHAT ARE THE CAUSES OF MMN?

The cause of MMN is not fully known. Evidence, however, supports that damage by the immune system to multiple, focal areas in peripheral nerves underlies this disorder and that immune treatment improves neurological function.

Peripheral nerves carry electrical signals from the spinal cord to muscle. The motor nerve cell or neuron in the spinal cord extends a fiber or axon which, like an electrical wire, is covered by segments of insulation or myelin that abut one to another. In MMN focal areas of myelin and axon damage block the transmission of signals from the brain to the muscle and result in muscle weakness.

At least 30% to 50% of MMN patients have proteins in their blood called antibodies that bind large lipid/fat molecules, GM1, located primarily on the surface of motor axons in focal areas not covered by the myelin segments (nodes of Ranvier). Whether GM1 antibodies participate in nerve damage is not known, although they are useful markers for MMN and help to support the diagnosis. At this time there is no evidence to support that MMN is inherited from parent to child.

HOW IS MMN DIAGNOSED?

The diagnosis of MMN is based on a combination of the patient's clinical findings and electrical testing of peripheral nerve function.

Motor nerves are stimulated with a small amount of electricity at two or more sites (for example the wrist and elbow) and the resulting muscle movement (for example at the base of thumb) is measured. From this study, it can be determined whether the nerve can conduct an electrical impulse and if so, how rapidly. In MMN the conduction of the electrical impulse is blocked in a focal segment of at least two or more nerves while conduction of the impulse in sensory fibers in the same nerves is normal. Such findings are diagnostic of MMN and help to distinguish this treatable disorder from a form of motor neuron disease known as amyotrophic lateral sclerosis (ALS) or Lou Gehrig's disease in which the immune system has little involvement and for which no effective therapy exists. This test also helps distinguish MMN from other forms of acute and chronic inflammatory neuropathies including Guillain-Barré Syndrome and chronic inflammatory demyelinating polyneuropathy.

HOW IS MMN TREATED?

Almost 85% of MMN patients show short and long term improvement with immunoglobulin (Ig), administered intravenously (IVIg) or subcutaneously (SCIg), making it the best first line of treatment. Patients typically respond to a course of immunoglobulin within hours to days, with improved strength that may last three to six weeks to months. Repeated doses of immunoglobulin are required in most patients to maintain the improvement although there may be gradual deterioration over years. Other therapies such as corticosteroids are not as effective and in some MMN patients can lead to worsening.

Immunoglobulin is the only agent shown to be beneficial in randomized and controlled trials. Immunosuppressant drugs including cyclophosphamide and azathioprine may be effective in individual patients but have not been tested in controlled trials. However, side effects such as cancer of white blood cells associated with long-term use of these drugs limit their benefit in a chronic disorder like MMN.

Physical and occupational therapy are helpful to maintain activities of daily living through exercise as are aids that facilitate specific function such as a hook used to button a shirt. Excessive exercise may result in increased muscle pain and poorer muscle function if not monitored properly.

NEED HELP?

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What is Miller Fisher Syndrome (MFS)?

Miller Fisher Syndrome (MFS), also called Fisher's syndrome, usually begins with the rapid development, over days, of 3 problems:

1. weak eye muscles, with double or blurred vision, and often drooping eyelids with facial weakness;
2. poor balance and coordination with sloppy or clumsy walking; and
3. on physical examination, loss of deep tendon reflexes, such as the knee and ankle jerk.

MFS is named after Dr. C. Miller Fisher who described it in 1956 as a limited variant of ascending paralysis, Guillain-Barré Syndrome (GBS).

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MFS

Miller Fisher Syndrome

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HOW IS MILLER FISHER SYNDROME DIAGNOSED?

Patients typically seek medical attention because of a rapid decrease in vision over days and/or difficulty walking. These changes are frequently preceded by a viral or diarrheal illness 1 to 4 weeks earlier. Slurred speech, difficulty swallowing and abnormal facial expression with inability to smile or whistle may also occur. Examination shows poor balance and coordination of the hands as well as loss of deep tendon reflexes and eye muscle weakness. Facial weakness, enlarged or dilated pupils and a decreased gag reflex on stimulation of the throat can be present in some patients. Tests of nerve conduction may show diminished activity of nerves that carry sensory information to the spinal cord and brain.

Magnetic resonance (MRI) or other imaging of the brain and/or spinal cord are usually normal. Spinal fluid protein is often elevated.

Pure Miller Fisher syndrome is uncommon, with many patients going on to develop the prominent widespread weakness of GBS.

HOW IS MILLER FISHER SYNDROME TREATED?

Fortunately, this disorder is often short lived, progressing for only a few weeks and then improving. MFS symptoms can signal the beginning of GBS, with breathing difficulties, so patients are often hospitalized for observation.

In pure MFS, a near full recovery typically occurs within 2-3 months. In rare cases when symptoms substantially impair function, various treatments that limit or neutralize immune system activity may be considered. These include high dose immune globulins or plasma exchange.

WHAT CAUSES MILLER FISHER SYNDROME?

The cause(s) of Miller Fisher Syndrome is not completely understood. The waddling, duck-like gait is likely due to the loss of a fat rich insulating material called myelin around nerves, designated as 1A, that innervate the major sensory organ

of muscle called the muscle spindle. These fibers send information to the spinal cord about the speed and extent of muscle stretch without which skeletal muscles can not properly function. As the clinical course progresses, other sensory fibers can be involved as well as motor and autonomic fibers that respectively innervate muscles that move the eyes and face and control function of the eye, pupil and the bladder.

Multiple lines of evidence support an auto-immune mechanism in which the preceding/triggering infection stimulates production of an antibody that reacts to a sugar found on both the surface of infectious organism and the peripheral nerve causing demyelination and loss of function of the nerve.

WWW.GBS-CIDP.ORG

Working for a future when no one afflicted with Guillain-Barré syndrome (GBS), chronic inflammatory demyelinating polyneuropathy (CIDP) or variants suffers alone and every patient has a full recovery.

NEED HELP?

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What is Guillain-Barré Syndrome (GBS)

Guillain-Barré (Ghee-YAN Bah-RAY) **Syndrome**, also called acute idiopathic polyneuritis and Landry's ascending paralysis, is an inflammatory disorder of the peripheral nerves, those outside the brain and spinal cord. It is characterized by the rapid onset of weakness and often, paralysis of the legs, arms, breathing muscles and face. Abnormal sensations often accompany the weakness.

Many patients require an intensive care unit during the early course of their illness, especially if support of breathing with a machine is required or if swallowing is involved. Although most people recover, this can take months, and some may have long-term disabilities of varying degrees. Mortality rate is less than 5 percent. GBS can develop in any person at any age, regardless of gender.

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GBS
Guillain-Barré Syndrome

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WHAT CAUSES GBS?

The cause is unknown. We do know that about 50% of cases occur shortly after a microbial infection (viral or bacterial), some as simple and common as the flu or food poisoning. Some theories suggest an autoimmune trigger, in which the patient's defense system of antibodies and white blood cells are called into action against the body, damaging myelin (nerve covering or insulation), leading to numbness and weakness.

HOW IS GBS DIAGNOSED?

Quite often, a patient's symptoms and physical exam are sufficient to indicate the diagnosis. The rapid onset of (ascending) weakness, frequently accompanied by abnormal sensations that affect both sides of the body similarly is common.

Loss of deep tendon reflexes, such as the knee jerk, are often found. To confirm the diagnosis, a lumbar puncture to find elevated fluid protein and electrical tests of nerve and muscle function may be performed.

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HOW IS GBS TREATED?

GBS in its early stages is unpredictable, so except in very mild cases, most newly diagnosed patients are hospitalized. Usually, a new case of GBS is admitted to ICU (Intensive Care) to monitor breathing and other body functions until the disease is stabilized. Plasma exchange (a blood "cleansing" procedure) and high dose intravenous immune globulins are often helpful to shorten the course of GBS. The acute phase of GBS typically varies in length from a few days to months, with over 90% of patients moving into the rehabilitative phase within four weeks. Patient care involves the coordinated efforts of a team such as a neurologist, physiatrist (rehabilitation physician), internist, family physician, physical therapist, occupational therapist, social worker, nurse, and psychologist or psychiatrist. Some patients require speech therapy if speech muscles have been affected.

THE GBS|CIDP FOUNDATION INTERNATIONAL

The organization was founded in 1980 by Estelle and Robert Benson to help others deal with this frightening and potentially catastrophic disorder from which recovery may not always be complete. The Foundation has over 182 chapters in the North America, Europe, the near and middle East, Africa, Australia, New Zealand and The Netherlands.

Its goals are to support you, the GBS patient and family. The Foundation is proud to have on its medical advisory board some of the world's leading experts on GBS, as well as physicians who themselves have had the disorder.

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