Miller Fisher Syndrome

To obtain more information, contact the GBS/CIDP Foundation International

The Holly Building
104 1/2 Forrest Avenue
Narberth PA 19072
1-866-224-3301   (610) 667-0131
Fax: (610) 667-7036
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Supported by an educational grant from Talecris Biotherapeutics, Center for Science and Education.
**What Is Miller Fisher Syndrome?**

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).

**How Is MF 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.

**How is MFS Treated?**

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

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 Fisher Syndrome?**

The cause(s) of Fisher’s 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.

**The GBS/CIDP Foundation International**

The Foundation was founded in 1980 by Robert and Estelle Benson to help others with this family of frightening and potentially catastrophic disorders. The Foundation has over 160 chapters around the world. Its goals are to support you, the patient and family. Our medical advisory board includes some of the world’s leading experts and some who themselves have had GBS and variants.

**Need Help?**

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