CLINICAL FINDINGS AND REHABILITATION TREATMENT IN MULTIPLE SCLEROSIS

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ABSTRACT
Multiple sclerosis is the most common cause of non-traumatic disability affecting young adults. Providers of care to persons with multiple sclerosis have recently begun to recognize the value of rehabilitation management techniques aside from the pharmacologic treatment because rehabilitation is the only way to improve function and quality of life in these patients affected by multiple sclerosis. Therefore rehabilitative management continues to play a vital role in the care of multiple sclerosis patients and will continue to do so into the future.

Keywords: multiple sclerosis, rehabilitation

INTRODUCTION
Multiple sclerosis (MS) is an inflammatory disorder with consequent damage primarily to myelin sheaths and oligodendrocytes and less to axons and nerve cells in the central nervous system (1).
Its prevalence is estimated to 2 million worldwide. The disease usually manifests between the ages of 20 to 40 years, is twice more often in women than in men, and is more frequent in the white race.
There are four common clinical courses in MS (1):
Relapsing-remitting MS in which patients have episodes of acute worsening of neurologic function followed by periods of remission, and can remain with deficits after the acute episode. This is the most frequent form of evolution.
Secondary progressive MS in which the patient has a relapsing-remitting course initially followed by progression with or without additional episodes of exacerbation and improvement. The majority of patients have the transition to this disease course.
Primary progressive MS in which the patient has a relentless progression of symptoms from the start.
Progressive-relapsing MS in which patients have a baseline progressive course with episodes of acute relapses followed by a return to the baseline progressive course.
The positive diagnosis is made if there is evidence of two attacks separated by at least 1 month with clinical, laboratory or imaging evidence of at least 2 lesions in the brain or spinal cord. Evidence may be obtained from clinical findings, MRI, cerebrospinal fluid analysis or visual evoked potentials.

CLINICAL FINDINGS IN MS
The symptoms of MS usually involve multiple systems (2).
Motor symptoms include weakness and spasticity, present up to 85% of patients and as many as a third can be affected by severe spasticity severe enough to diminish their quality of life. Also many patients present with progressive paraparesis in association with sphincter dysfunction (3).
Sensory symptoms can appear in various body parts, most often affecting the trunk and including paresthesia in 50% of patients, being described as abnormal sensation, as pain, pins and needles, or tingling and most commonly is neuropathic. The Lhermitte sign may occur in up to 40% of patients, being an electric-shock like sensation that radiates down the spine to the legs when the neck is flexed.
Visual symptoms may include optic neuritis from inflammation of the optic nerves and is manifested as retro-orbital pain or painful eye movements. Visual deficits, scotoma and ocular motor deficits are manifested as diplopia, blurry vision and reading fatigue.

Cerebellar symptoms include tremor at rest or with purposeful actions, involving various parts of the body, including the head, trunk or limbs.

Constipation and bowel incontinence may occur in half of patients due to slowed colonic transit, decreased pelvic muscle function, sensory deficits and adverse effects of medication.

Bladder dysfunction occurs in more than 80% of patients and are caused by lesions in the spinal cord with small spastic bladder due to detrusor over-activity and is manifested by urinary urgency, frequency, voiding of small amounts of urine and incontinence. This is often associated with urinary tract infections that can worsen MS symptoms.

Involvement of cranial nerves VII, IX, X and XII result in dysphagia or swallowing difficulties, manifested as coughing, complaints of food remaining in the throat, weight loss, weak voice or even aspiration pneumonia.

Fatigue is very common, up to 90% of patients, being the most disabling symptom, described as a “feeling of tiredness in those who have done little and are not depressed”.

Cognitive deficits are present in 50% of patients, manifested as problems with memory, planning, concentration, judgment, problem solving and processing speed. The patients frequently report heat intolerance with exacerbation of symptoms in warm or humid environments.

FUNCTIONAL LIMITATIONS IN MS

The deficits that appear in MS lead to difficulties with activities of daily living (ADL) and mobility. Weakness, incoordination, spasticity and sensory deficits may raise the risk of falling during ambulation. Decreased mobility leads to further weakness, decreased endurance and less independence for the patient. There may appear problems with feeding and self-care, resulting in need for personal care attendance.

Bowel and bladder dysfunction can isolate the patients from the community and social life.

Depression, insomnia and fatigue can contribute to activity intolerance. Visual deficits may limit activities such as driving, reading and ambulation, thus limiting participation in working and recreation.

PHARMACOLOGIC TREATMENT OF MS

Patients with MS require a multidisciplinary approach that can identify the symptoms and formulate a treatment plan (4).

Education of the patient and family should be included in the initial treatment, with information about a balanced diet, adequate fluid intake, weight control and appropriate exercise. The patient is encouraged to continue working and participating in recreational activities for as long as possible.

High-dose methylprednisolone for 3 to 5 days is an effective treatment of acute relapses and then continued with tapering doses of oral prednisolone. The medications approved by the Food and Drug Administration as first-line treatment to decrease the relapse rate in relapsing-remitting MS include interferon beta-1a and interferon beta-1b.

Spasticity management is complex, because some of the patients use their spasticity to assist to transfers or gait, so it must be treated only if interferes with mobility or ADL. Oral baclofen is the usual first-line treatment, starting with 5 mg two or three times per day up to a maximum of 80 mg in divided doses. The newer antiepileptic gabapentin has antispastic effects, being usually started at 100 mg three times a day and highered up to 3600 mg in divided doses. Sedation is the most common side effect. Intractable spasticity may be managed with muscle or nerve blocks or intrathecal administration of baclofen (8).

Bladder dysfunction can be assessed with a urodynamic study. Non-pharmacologic interventions include timed voiding, minimizing the intake of bladder irritants (such as caffeine) and regulation of fluid intake. Detrusor hyperactivity may respond to anticholinergic drugs such as oxybutynin 2.5 to 5 mg three times a day or tolterodine 2 mg twice per day, which has fewer anti-cholinergic side effects. Detrusor under-activity may respond to cholinergic agents such as bethanechol 10 to 50 mg titrated up to four times a day. Detrusor-sphincter dyssynergia may respond to botulinum toxin type A injections in the urethral sphincter.

Bowel dysfunction manifested as constipation is treated by establishing a bowel program with adequate fluid intake, fiber ingestion (fruits, vegetables, whole grains, nuts and seeds).

Fatigue should be treated first by non-pharmacologic interventions such as treating underlying factors that exacerbate fatigue (correction of sleep disturbances, treatment of depression), improving physical fitness by aerobic exercises, improving mobility by physical and occupational therapy,
teaching energy conservation with timed rest periods and work simplification techniques, teaching cooling techniques by avoiding the heat.

Cognitive impairment can be assessed during interactions with family, speech-language pathologists, physical therapists and occupational therapists. Antidepressant medication and counseling can improve the patients quality of life.

Acute visual deficits due to inflammation may be treated with high-dose intravenous methylprednisolone. Prisms lenses may help compensate for double vision. The patient with visual deficits needs special treatment from the ophtalmologist (5).

THE REHABILITATION TREATMENT OF MS

Physical therapy procedures to decrease spasticity include range of motion exercises, stretching, positioning, aerobic exercises and relaxation techniques.

Mobility can be improved by training the patient to use various assistive devices to compensate for weakness and fatigue. Pain management can be treated by using transcutaneous electrical stimulation (TENS). Aerobic exercises prevent deconditioning, improve endurance and delay or minimize the effects of fatigue (6). Weakness that can occur in demyelinated nerves in MS may be made worse if the patient exercises to the point of fatigue so the exercise programs should be individualized and updated at the present condition of the patient (7).

Occupational therapy can help minimize the effects of fatigue by teaching energy conservation and work simplification through the use of devices and techniques. Ataxia and tremor can benefit of assistive devices and weights on the distal limbs that lessen the effect of the tremor (9).

A vocational rehabilitation counselor can play an important role in integrating the disabled patient with MS back into the workplace as the majority of them are adult working patients.

CONCLUSION

Patients with MS live with their disease for the rest of their life so the better they can preserve and improve their general health, the better they will be. Because MS is a lifetime disease, lifetime management and rehabilitation must be used.

REFERENCES