

Rehabilitation Strategies in Neurodegenerative Diseases: Case Reports in ALS and MS

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INTRODUCTION

Multiple sclerosis

Multiple Sclerosis is a chronic, immune-mediated disorder of the central nervous system (CNS), primarily affecting the brain, spinal cord, and optic nerves. It is characterized by inflammation, demyelination, and subsequent neurodegeneration. The course may be relapsing-remitting or progressive. Lesions in the CNS occur at different times and in different CNS locations. Because of this, multiple sclerosis lesions are sometimes said to be "disseminated in time and space." The clinical course of the disease is quite variable ranging from stable chronic disease to a rapidly evolving and debilitating illness^(*).

* Michael J. Olek. Multiple Sclerosis

Amyotrophic lateral sclerosis

Amyotrophic Lateral Sclerosis, also known as Lou Gehrig's disease, is a progressive neurodegenerative disorder that affects motor neurons in the brain and spinal cord. Unlike MS, ALS does not involve demyelination but instead leads to the degeneration and eventual death of upper and lower motor neurons. This neuronal loss results in muscle weakness, atrophy, and spasticity, eventually leading to paralysis. Early symptoms often include limb weakness or difficulty speaking or swallowing. As the disease advances, respiratory muscles become involved, which is the most common cause of death^(*).

* Brotman RG, Moreno-Escobar MC, Joseph J, et al. Amyotrophic Lateral Sclerosis.

Diagnosis:

Multiple sclerosis

- Clinical Evaluation:
 - History of neurological episodes (sensory loss, weakness)
 - Objective neurological deficits on examination
- MRI: Detects demyelinating lesions in characteristic CNS regions (periventricular, juxtacortical, infratentorial, spinal cord)
- Cerebrospinal Fluid Analysis: Presence of abnormal amount of white blood cells, proteins and other abnormalities that are secondary to MS.

Amyotrophic lateral sclerosis

- Clinical Examination:
 - Upper motor neuron signs: spasticity, hyperreflexia, Babinski sign
 - Lower motor neuron signs: muscle atrophy, weakness, fasciculations
 - Progressive spread of symptoms across body regions
- Electromyography:
 - Shows evidence of active and chronic denervation

Differential Diagnosis

Multiple sclerosis

- Neuromyelitis Optica Spectrum Disorder: severe optic neuritis + longitudinal spinal cord lesions, often more aggressive than MS, associated with AQP4 antibodies
- Acute Disseminated Encephalomyelitis: usually monophasic, more common in children
- Systemic Lupus Erythematosus: CNS involvement can mimic MS, systemic signs (rash, arthritis, antibodies)

Amyotrophic lateral sclerosis

- Cervical Spondylotic Myelopathy: Can produce UMN + LMN signs, has neck pain and sensory symptoms
- Spinal Cord Tumor: progressive deficits, sensory level or pain
- Primary Lateral Sclerosis: only upper motor neuron signs, no muscle atrophy or fasciculations
- Progressive Muscular Atrophy: only lower motor neuron signs, no spasticity or Babinski sign
- Spinal Muscular Atrophy: genetic disorder, usually earlier onset
- Multifocal Motor Neuropathy
- Myasthenia Gravis

Treatment

Multiple sclerosis

- Corticosteroids
- Disease-Modifying Therapies
 - Interferon beta
 - Fingolimod
 - Monoclonal antibodies
- Symptomatic Treatment:
 - Baclofen
 - Gabapentin
- Rehabilitation

Amyotrophic lateral sclerosis

- Disease-Modifying Therapies:
 - Riluzole
 - Edaravone
- Symptomatic Management:
 - Baclofen
 - Anticholinergic medications
- Respiratory Support
- Rehabilitation

Complications

Multiple sclerosis

- Progressive motor weakness
- Cognitive decline
- Visual impairment
- Spasticity
- Ataxia

Amyotrophic lateral sclerosis

- Progressive muscle weakness leading to paralysis
- Muscle atrophy (amyotrophy)
- Spasticity and painful cramps
- Loss of mobility leading to wheelchair dependence
- Progressive respiratory muscle weakness
- Hypoventilation (especially at night)
- Ineffective cough leading to secretion retention and aspiration pneumonia

Prognosis

Multiple sclerosis

- Most patients have a near-normal life expectancy, especially with modern treatment and lifestyle adjustments
- Accumulated disability can occur due to axonal loss
- Poor quality of life.

Amyotrophic lateral sclerosis

- Median survival: ~3–5 years after symptom onset
- Death usually results from respiratory failure
- Disability
- Supportive care can extend life and improve quality of life

Case presentation

- This paper presents the case of two patients admitted to our Neuromuscular Rehabilitation Clinical Division. The first patient presenting with:
 - Male, 51 years old;
 - Triparetic motor deficit with predominance of left hemiparesis
 - Bilateral knee pain, mixed character (VAS = 7)
 - Neurogenic bladder in remission
 - Difficult gait

Medical history

Martie 2016 – Onset:
paresthesia in the left lower
limb and exertional fatigability

Iunie 2016 – Episode of right optic
neuritis; brain MRI revealed
demyelinating lesions; CSF positive for
oligoclonal bands → diagnosis of
relapsing-remitting multiple sclerosis
(RRMS)

2017–2018 – Recurrent
episodes of fatigue and mild
weakness in the left lower
limb, with partial remission
under treatment

Septembrie 2019 – First
significant motor episode: mild
left hemiparesis, onset of
spasticity

Ianuarie 2021 – Development of
sphincter disturbances → diagnosis
of neurogenic bladder, later
improved under treatment

Noiembrie 2022 – Progressive
worsening of motor deficit and
reduced walking distance →
transition to secondary progressive
form; initiation of treatment with
Siponimod

Medical history

May 2023 – Need to use a cane for long-distance ambulation; increased fatigability.

February 2024 – Onset of bilateral knee pain, in the context of gait disturbances

September 2025 – Progressive worsening of motor deficit, with severe limitation of walking, development of triparesis with left predominance

January 2026 – Hospital admission for: triparetic motor deficit, bilateral knee pain, neurological and functional reassessment

Medical history

- Allergies: None reported
- Personal pathological history
 - Thrombophilia with heterozygous mutation of PAI-1 and MTHFR genes
 - Anxious-depressive disorder
- Family medical history
 - Grandfather – multiple sclerosis
 - Father - thrombophilia

Medication

1. Aspenter 75mg, 1cp/zi po, 0-1-0
2. Lioresal 25 mg, 1cpx2/zi po, 1 -0-1
3. Sortis 20 mg, 1cp/zi po, 0-0-1
4. Vigantoleten 200 UI 1-0-0
5. Milgamma N 200 mg, 1cpx2/zi po, 0-2-0
6. Esentiale forte
7. Zeliftan 6/0,4 mg
8. Siponimod la 2 zile 0-1-0
9. Anxiar 1 mg 1/2cp/zi po, 0-1/2
10. Pantoprazol 20 mg 1cp/zi po, 1-0-0

Working Conditions/Standard of Living

- Middle-aged adult
 - Former civil engineer (construction site manager)
 - Lives with his wife (accountant), in an urban apartment
 - Has 3 grandchildren
-
- Denies smoking
 - Occasional alcohol consumption

Diagnosis

- Central motor neuron syndrome with triparesis, predominantly left hemiparesis, in the context of Multiple Sclerosis
- Bilateral knee algofunctional syndrome, caused by biomechanical gait imbalance in the context of Multiple Sclerosis

General examination

- Conscious, cooperative, oriented (time, space, person)
- General condition: good
- Height: 1.81 m
- Weight: 87 kg
- Blood pressure: 131/90 mmHg
- Heart rate: 85 bpm
- Respiratory, digestive, and renal systems: balanced

NEURO-MIO-ARTHRO-KINETIC EXAMINATION

- Cranial nerves: within normal limits
- Motor deficit:
 - Triparesis with predominance of left hemiparesis
- Muscle strength (MRC scale):
- Right upper limb: 5/5
- Left upper limb: 4/5
- Lower limbs (bilateral): 4/5
- Spasticity: left lower limb (MAS 2)
- Plantar reflex: indifferent bilaterally
- Deep tendon reflexes: symmetrical

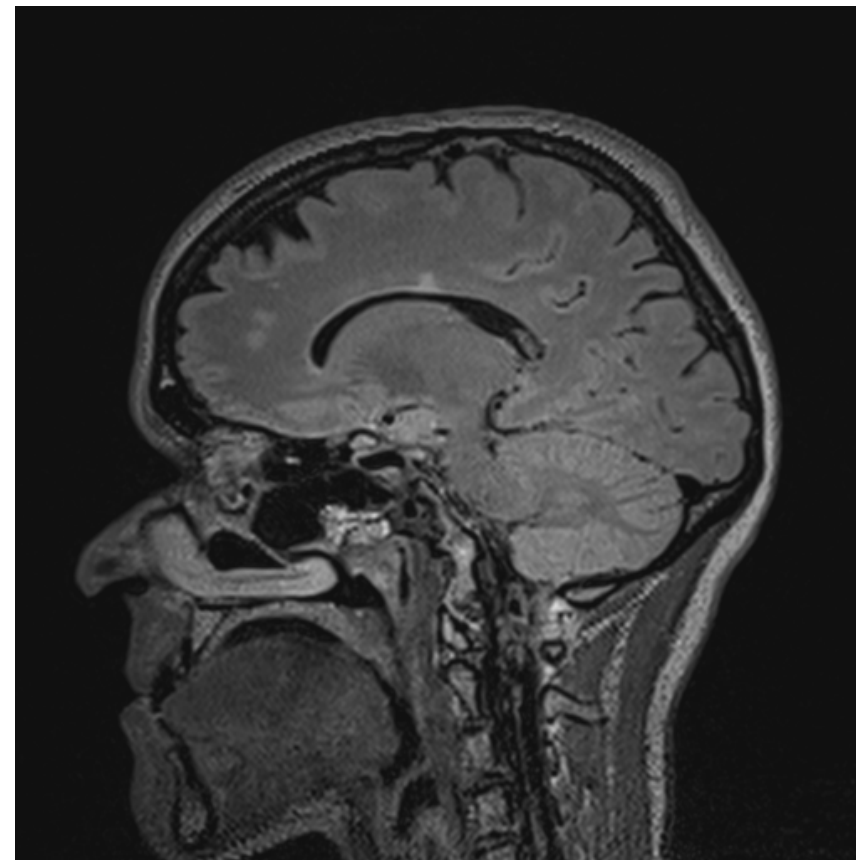
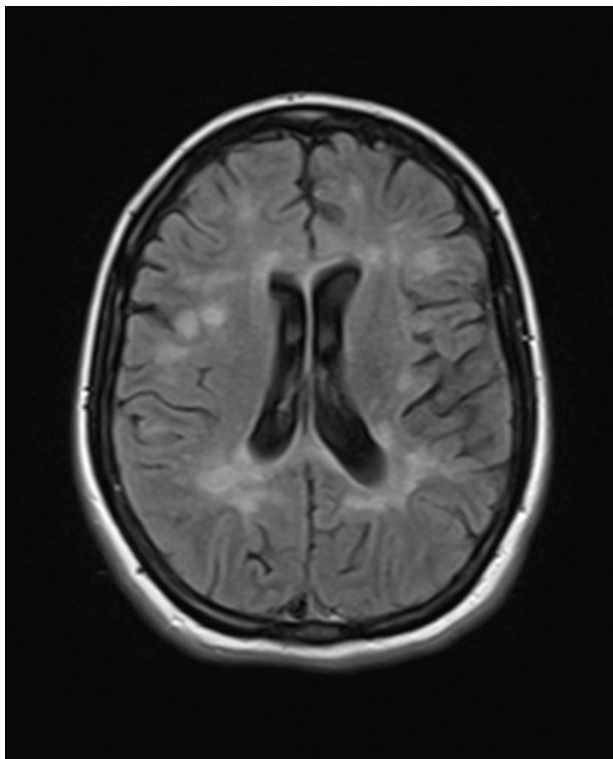
Functionality

- Independent in bed mobility and transfers
- Able to walk short distances without support (under supervision), walk medium distances with tetrapod cane support

LABORATORY FINDINGS

- Physiological spinal curves preserved
- ECG: sinus rhythm, HR 60 bpm, normal QRS axis, no ST-T changes
- Laboratory Findings:
 - Complete blood count: within normal limits
 - Biochemistry: within normal limits
 - Lipid profile: within cardiovascular risk targets
 - Urinalysis: normal

Paraclinic



FUNCTIONAL ASSESSMENT

- EDSS – 6,5/10
- Barthel – 60/100
- Ashworth Scale – frequency: 2, severity: 2
- FAC – 2/5 (able to walk or balance with occasional/intermittent support)

ICF-DH

Health Condition:
Multiple Sclerosis

Body Functions & Structures:

- Weakness
- Spasticity
- Ataxia
- Fatigue

Activities:

- Walking limited
- Needs assistance
- Difficulty stairs

Participation:

- Reduced work
- Limited social life
- Reduced independence

Environmental Factors:

- + Family support
- + Therapy
- Barriers

Personal Factors:

- Motivation
- Anxiety
- Active lifestyle

Objectives

- Improvement of pain symptoms
- Increase in muscle strength
- Reduction of paresthesias
- Improvement of gait pattern and reduction of fall risk
- Increase in autonomy (independence)

Rehabilitation program

- Objectives:
 - Correction of hip and knee flexion contracture
 - Restoring arm swing in gait
 - Reduction of pain
 - Increase muscle strength
 - Decrease paresthesia
 - Improve gait pattern and reduce fall risk
 - Increase independence

Physical therapy

- Methodes:
 - Passive/active hamstring stretching exercises
 - Postural exercises in extension
 - Strengthening
 - Isometric strengthening with progressive resistance of: gluteal muscles, quadriceps, paravertebral muscles, abdominal muscles
 - Gait, balance, and proprioception exercises with visual/sensory feedback
 - Walking on varied surfaces

Evolution

- The patient benefited from a complex neuro-muscular rehabilitation program, having a favourable evolution with:
 - Reduction in pain and spasticity
 - Improved muscle strength and coordination
 - Better gait stability and reduced fall risk
 - Increased tolerance to physical effort
- The patient with Multiple Sclerosis typically demonstrates a favorable functional evolution during inpatient rehabilitation, with improvement in motor function, balance, gait, and autonomy. However, given the chronic and potentially progressive nature of the disease, long-term outcomes depend on disease course, therapeutic adherence, and ongoing multidisciplinary management.

Case presentation

- The second patient is:
- Female, 66 years old
- Motor deficit: distal brachial diparesis, right > left
- Balance disorders
- Mixed-type vertebral pain

Medical history

- Allergies: None reported
- Family medical history: Non-significant
- Personal pathological history:
 - Hypertensive cerebral microangiopathy
 - Essential hypertension (Grade II)
 - Bilateral carotid atherosclerosis (without hemodynamic significance)
 - Dyslipidemia (hypercholesterolemia)
 - Cervico-dorso-lumbar spondylodiscoarthrosis
 - Multifactorial vertigo syndrome

Medication

- Norvasc 5 mg 0-0-1
- Nolet 5 mg 1-0-0
- Atoris 40 mg 0-0-1
- Controloc 20 mg 1-0-0
- Riluzol 50 mg 1-0-1
- Betaloc Zok 50 mg if AV >90 bpm
- Cipralex 10 mg 0-0-1
- Anxiar 1 mg 0-0-1
- Enerion 200 mg
- Naldorex 550 mg if pain

Working Conditions/Standard of Living

- Retired (age-related retirement)
- Former accountant
- Lives with husband (urban environment, 1st-floor apartment)
- Denies alcohol consumption
- Smoking history: ~10 pack-years

Medical history

June 2024

- Insidious, progressive onset
- Difficulty performing fine motor tasks (buttoning shirt, writing)
- No sensory symptoms

August 2024

- Weakness gradually worsens
- Visible muscle wasting in hand
- Appearance of fasciculations
- Grip strength decreases

November 2024

- Weakness spreads to right forearm → arm → contralateral limb
- Difficulty lifting objects
- Slight dysarthria
- Dysphagia

January 2025 – Hospital admission for biparetic motor deficit, balance disturbances, and mixed-type pain of the vertebral column

Diagnosis

- Peripheral Motor Neuron Syndrome with biparesis predominantly right more than left through ALS
- Bilateral algofunctional syndrome of the hands, caused by biomechanical imbalance in the context of Amyotrophic Lateral Sclerosis

General examination

- Patient conscious, cooperative, oriented to time, space, and person
- General condition: satisfactory
- Height: 1.61 m
- Weight: 58 kg
- Blood pressure: 128/78 mmHg
- Heart rate: 71 bpm
- Respiratory, digestive, and renal systems: balanced
- Spine:
 - Cervical straightening
 - Right dorsal scoliosis
 - Paravertebral muscle contracture (cervico-dorso-lumbar spine)

NEURO-MIO-ARTHRO-KINETIC EXAMINATION

- Mild bilateral hypoacusis
- Mild dysarthria, dysphagia
- Motor deficit: brachial diparesis (bilateral upper limb weakness)
- Dysmetria on finger-to-nose test (right upper limb)
- Muscle Strength (MRC scale):
- Upper limbs: 3/5 proximally, 2/5 distally (bilateral)
- Lower limbs: 4/5 proximally, distally slightly reduced
- “Simian hand” deformity: right > left
- Deep tendon reflexes: exaggerated
- Babinski sign positive bilaterally

Functionally

- Unable to perform pincer grasp
- Limited mobility in activities of daily living (ADLs)
- Functionally:
 - Able to walk with small, unsteady steps, over moderate distances

LABORATORY FINDINGS

- Cardiovascular & General Examination
- Physiological spinal curves preserved
- ECG: sinus rhythm, HR 60 bpm, normal QRS axis, no ST-T changes
- Laboratory Findings
 - Mild anemia
 - Biochemistry within normal limits
 - Lipid profile: hypercholesterolemia
 - Impaired glucose tolerance

FUNCTIONAL ASSESSMENT

- Barthel – 55/100
- ADL – 3/6
- IADL – 6/8
- ALSFRS-R – 26/48
- FAC – 3/5 (No physical help, just supervision for safety)

Diagnosis

- Upper Motor Neuron (UMN) degeneration:
 - Positive Babinski sign
- Lower Motor Neuron (LMN) degeneration:
 - Muscle weakness
 - Fasciculations
 - Atrophy
- Progressive spread of symptoms:
 - From one region to another

ICF-DH

Health Condition:
Amyotrophic lateral sclerosis

Body Functions & Structures

- Generalized muscle weakness
- Muscle atrophy
- Fasciculations
- Spasticity (upper motor neuron signs)
- Dysarthria (speech difficulties)
- Dysphagia

Activities

- Walking is limited → needs assistance or mobility aid
- Difficulty climbing stairs
- Needs help with dressing, bathing, and personal care
- Impaired hand function (difficulty handling objects, writing, utensils)
- Speech is slow and sometimes unintelligible
- Fatigue limits daily activities

Participation

- Unable to maintain full-time work (may stop working)
- Reduced social participation and leisure activities
- Dependent on caregiver and family support
- Limited community mobility and social roles
- Impact on family and caregiver burden

Environmental Factors

- Family support and involvement
- Multidisciplinary healthcare team
- Physical barriers at home and in the community

Personal Factors

- High motivation and willingness to adapt
- Awareness of disease and emotional impact
- Anxiety and uncertainty about the future
- Coping strategies and resilience

Objectives

- Maintaining functional independence as long as possible
- Prevent deconditioning
- Maintain available strength with **low–moderate intensity exercise**
- Avoid fatigue and overexertion
- Prevent joint contractures → stretching, positioning
- Avoid pressure ulcers
- Optimize respiratory function
- Improve quality of life

Kinesiotherapy Program

- Mobility Exercises
 - passive mobilizations, active-assisted exercises, active exercises (for unaffected muscle groups)
 - stretching to prevent contractures
- Strengthening Exercises (with caution)
 - low-resistance exercises with few repetitions, avoiding muscle overfatigue
- Functional Training
 - gait training and training of activities of daily living (ADLs)- use of assistive devices

Kinesiotherapy Program

- Respiratory Physiotherapy
 - diaphragmatic breathing exercises
 - thoracic expansion exercises
- Balance and Coordination Exercises
 - exercises in sitting and standing
 - proprioceptive training
 - fall prevention strategies
- Postural Management
 - proper positioning in bed and chair
 - frequent position changes

Evolution

- The patient benefited from a complex neuro-muscular rehabilitation program focused on maintaining function, comfort, and quality of life having a slightly improved evolution.
- Hand functionality: can perform hand grips with low muscle strength
- ALS progression is inevitable but variable
- Rehabilitation helped:
 - Slow functional decline impact
 - Prevent secondary complications
 - Improved quality of life

CONCLUSION

- Neurorehabilitation plays a central role in both Multiple sclerosis and Amyotrophic lateral sclerosis, but its objectives and expected outcomes differ because of the underlying disease mechanisms.
- A multidisciplinary approach is essential, integrating physical, occupational, speech, and psychological therapies.
- Rehabilitation should be individualized and adaptive, evolving with disease progression and patient needs.
- Early initiation of rehabilitation leads to better long-term functional outcomes and quality of life.
- Patient education and caregiver involvement are critical components of effective care.
- The ultimate goal is not only physical function but also participation, autonomy, and well-being

CONCLUSION

- In multiple sclerosis, rehabilitation benefits from the disease's variability and potential for remission. Targeted interventions can lead to meaningful functional recovery or stabilization, especially after relapses. Fatigue management, spasticity control, and gait training often translate into improved independence and participation. Neuroplasticity plays an important role here, allowing patients to regain or compensate for lost functions.
- In amyotrophic lateral sclerosis, neurorehabilitation has a more supportive and palliative trajectory. Because ALS is relentlessly progressive, rehabilitation focuses on preserving function for as long as possible, preventing complications (such as contractures or pressure ulcers), and maintaining quality of life. Assistive technologies, respiratory support, and communication aids become increasingly important as the disease advances.

THANK YOU!