Impact of Structured Neurorehabilitation on Functional and Respiratory Outcomes in Bulbar Motor Neuron Disease: A Case Study

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ABSTRACT

Background: Motor Neuron Disease (MND) represents a group of progressive neurodegenerative disorders characterized by selective degeneration of upper and lower motor neurons. Among its clinical phenotypes, Amyotrophic Lateral Sclerosis (ALS) is the most prevalent. Bulbar-onset disease, often referred to as Progressive Bulbar Palsy, presents predominantly with dysarthria and dysphagia and carries a comparatively poorer prognosis due to early respiratory involvement.

Objective: To evaluate the therapeutic impact of structured, personalized physiotherapy intervention on functional, respiratory, and swallowing outcomes in an elderly patient with bulbar MND.

Methods: An 85-year-old male diagnosed with bulbar-onset MND underwent comprehensive physiotherapy for 8 weeks. Outcome measures included Manual Muscle Testing (MMT), Visual Analog Scale (VAS), Dysphagia Outcome Severity Scale (DOSS), Functional Oral Intake Scale (FOIS), and House–Brackmann Scale (HBS).

Results: Post-intervention analysis revealed improvements in muscle strength (3+/5 to 4–5/5), swallowing safety (DOSS 2 to 5), oral intake (FOIS 3 to 5), and pain reduction (VAS 7 to 3). Respiratory endurance and cough efficiency improved significantly.

Conclusion: Targeted physiotherapy significantly improved functional outcomes and quality of life in bulbar MND, reinforcing the importance of early multidisciplinary neurorehabilitation.

Keywords: Motor Neuron Disease, Amyotrophic Lateral Sclerosis, Bulbar Palsy, Dysphagia, Neurorehabilitation, Respiratory Therapy.

INTRODUCTION: Motor Neuron Disease (MND) comprises a heterogeneous group of progressive, long-term neurological disorders characterized by selective degeneration of upper and lower motor neurons[1]. The disease leads to progressive muscle weakness, atrophy, spasticity, and ultimately respiratory failure. Prognosis depends on age at onset, pattern of neuronal involvement, and rate of progression.

The most common phenotype of MND is Amyotrophic Lateral Sclerosis (ALS), which involves combined upper motor neuron (UMN) degeneration in the motor cortex and corticospinal tracts, and lower motor neuron (LMN) degeneration in the anterior horn cells and cranial nerve nuclei. Clinically, this results in muscle wasting, fasciculations, hyperreflexia, spasticity, dysarthria, dysphagia, and progressive respiratory compromise.[1,2]

Four principal phenotypes are recognized based on the site of onset and pattern of involvement amyotrophic lateral sclerosis (ALS), primary lateral sclerosis (PLS), progressive muscular atrophy (PMA), and progressive bulbar palsy (PBP). Bulbar-onset disease accounts for approximately 20–30% of cases and initially presents with slurred speech due to tongue weakness, fasciculations, and dysphagia.[3,4] Emotional lability (pseudobulbar affect) is common due to corticobulbar tract involvement. In rare cases, respiratory muscle weakness may precede limb symptoms.[4]

Globally, the incidence of MND is approximately 1–2 per 100,000 population per year, with a prevalence of 5–7 per 100,000. The condition is slightly more common in males (male: female ratio ≈1.5:1). Familial ALS represents 5–10% of cases, often inherited in an autosomal dominant pattern, while 90–95% are sporadic.[5]

Motor neurons are specialized cells responsible for voluntary movement, speech, swallowing, and respiration. Upper motor neurons (UMN) originate in the motor cortex and descend via the corticospinal (pyramidal) tract to the brainstem and spinal cord and Lower motor neurons (LMN) arise from anterior horn cells of the spinal cord or cranial nerve nuclei and directly innervate skeletal muscles.[6,7] Degeneration of UMNs leads to spasticity and hyperreflexia, while LMN degeneration causes muscle wasting, weakness, and fasciculations. The corticospinal tract plays a major role in voluntary motor control, Lateral corticospinal tract- fine distal limb movements and Anterior corticospinal tract- proximal muscle control.[7]

Motor cortex areas (Brodmann area 4 – primary motor cortex; area 6 – premotor cortex; areas 44 & 45 – Broca's area) coordinate movement initiation, planning, and speech production, explaining the bulbar manifestations seen in ALS.[7]

The exact etiology of MND remains unclear. Proposed mechanisms include, Genetic mutations (e.g., SOD1, C9orf72), Glutamate-mediated excitotoxicity, Oxidative stress, Neuroinflammation, Environmental toxin exposure. Pathologically, there might be a Thinning of anterior spinal roots, Loss of motor neurons in the cortex, brainstem, and anterior horns, Degeneration of corticospinal and corticobulbar tracts, Absence of significant inflammatory response.[8]

Clinical Features, At Early Stage includes Limb weakness, Slurred speech, Fasciculations, Fatigue, Dysphagia, Emotional lability. At Progressive Stage includes Muscle atrophy, Respiratory difficulty, Drooling, Joint stiffness, Weight loss. And at the Advanced Stage, Severe respiratory insufficiency, Complete functional dependence, Ventilatory support requirement in which the Respiratory failure remains the most common cause of mortality. Diagnosis is primarily clinical, supported by Electromyography (EMG) and nerve conduction studies, MRI (to exclude other pathologies), Blood tests to rule out differential diagnoses, Lumbar puncture (when indicated).[9]

There is no cure for MND; management focuses on symptom control and functional preservation through multidisciplinary care. The Symptomatic management includes- Anticholinergics for drooling, Baclofen or tizanidine for spasticity, SSRIs for emotional lability, Mucolytics for secretions, Laxatives for constipation, Non-invasive ventilation for respiratory compromise.[9,10]

Rehabilitation plays a central role, with low-to-moderate intensity strengthening, stretching, respiratory training, and energy conservation strategies improving quality of life and delaying functional decline.[11]

Aim of the study is to Evaluate the effectiveness of a personalized physiotherapy program in improving motor function, respiratory efficiency, speech and swallowing abilities, and overall quality of life in a patient diagnosed with bulbar motor neuron disorder (MND). And Objectives of the study is To assess the baseline physical, respiratory, and functional impairments in a patient with bulbar motor neuron disease. To design and implement an individualized physiotherapy regimen focusing on respiratory training, muscle strengthening, and posture correction. To evaluate changes in muscle strength, speech and swallowing function, and pain levels before and after physiotherapy intervention. To analyze the impact of physiotherapy on the patient's

mobility, endurance, and quality of life. To emphasize the importance of a multidisciplinary approach (physiotherapy, respiratory therapy, and speech therapy) in managing bulbar MND.

An 85-year-old male was admitted to the neurology department in October 2025 with complaints of progressive difficulty in swallowing solid food and mild discomfort while drinking liquids. The patient had a significant past medical history of Ischemic stroke (April 2016), COVID-19 infection requiring hospitalization (2020), post-COVID pulmonary changes, Long-standing hypertension, Diagnosed bulbar-onset Motor Neuron Disease for 1.5 years.

Following detailed neurological evaluation and investigations, a diagnosis of progressive bulbar involvement was confirmed, and comprehensive physiotherapy was recommended. On Clinical Findings, General Examination, Conscious, cooperative, and well-oriented, Mesomorphic body build, Vital signs stable. Cranial Nerve and Bulbar Assessment- Weak facial musculature, Difficulty smiling and tightly closing eyes, Slurred speech, Tongue deviation with visible wasting, Dysphagia for solids > liquids.

On Motor Examination- Manual Muscle Testing (Medical Research Council Scale)**,** Reduced strength in both upper and lower limbs (average 3+/5), N[illegible]ved deep tendon reflexes. Respiratory Status- History of post-COVID pulmona[illegible]athlessness**,** No acute respiratory distress at presentation.

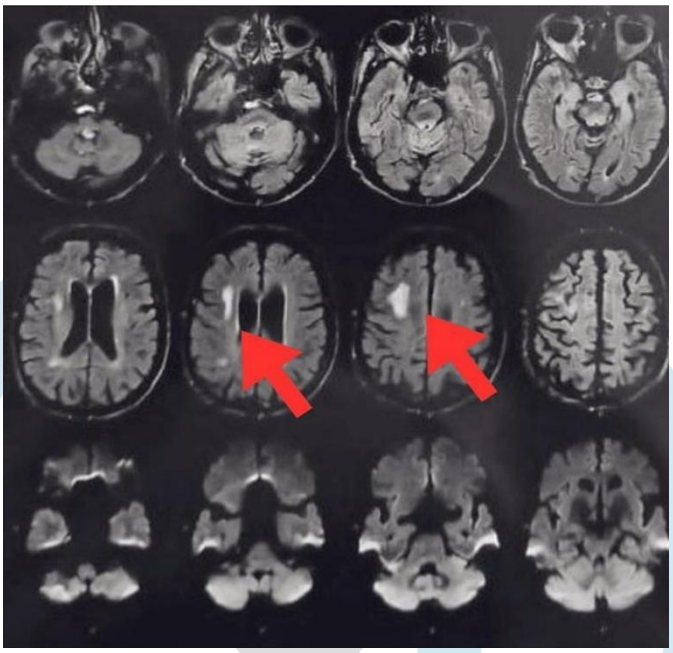
Clinical Photograph Showing Tongue Deviation

Radiological and Diagnostic Investigations

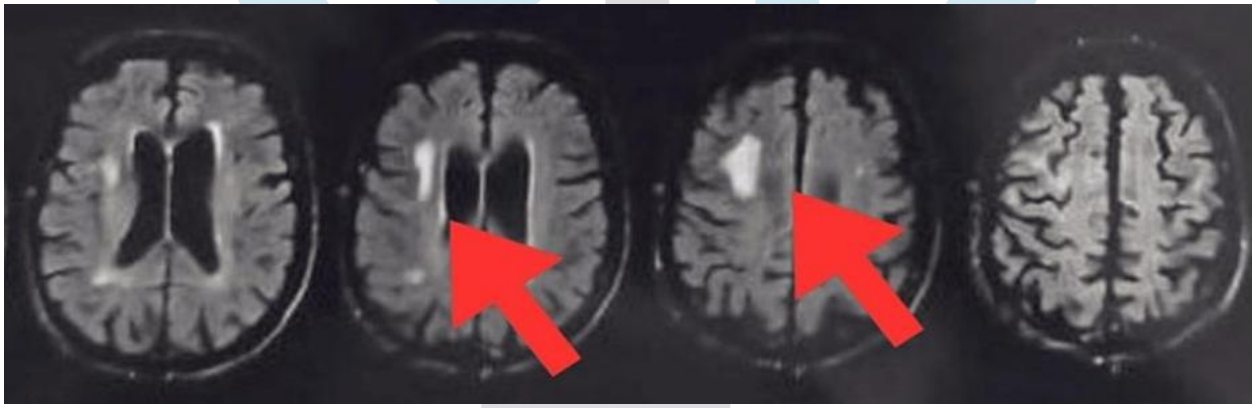
MRI Brain Findings

T2-weighted and FLAIR axial sequences demonstrated hyperintense lesions.

MRI Brain Axial T2/FLAIR with Red Arrow Markings



Ventricular Level – Corpus Callosum, Centrum Semiovale)



Findings by Anatomical Level Basal Level (Brainstem & Cerebellum**)-** Symmetrical structures, No abnormal hyperintensities. Thalamic & Basal Ganglia Level- Periventricular white matter hyperintensity (left side), Suggestive of chronic small vessel ischemic change. Ventricular Level (Corpus Callosum / Centrum Semiovale)- Left periventricular chronic lesion, right deep white matter focal hyperintensity (possible acute/subacute ischemia). High Cortical Level- No focal abnormalities. Summary of MRI Abnormalities.

Table – 1: Radiological Summary

Location	Radiological Finding	Interpretation
Left periventricular white matter	Hyperintensity	Chronic ischemic change
Right deep white matter	Focal hyperintensity	Acute/subacute ischemia
Other regions	Normal	—

Pulmonary Imaging (Post-COVID, 2020)- High-Resolution CT (HRCT) Thorax showed, Multiple patchy ground-glass opacities, Interlobular septal thickening, Fibrotic bands, Bilateral lung involvement.

Electromyography (EMG) and Nerve Conduction Velocity (NCV) revealed -Active and chronic partial denervation in tongue muscles, Chronic denervation in bilateral first dorsal interosseous, Left tibialis anterior involvement, right medial gastrocnemius involvement, Absent F response in peroneal nerves, Absent sensory response in sural nerves. These findings were consistent with lower motor neuron degeneration.

A comprehensive, individualized Physiotherapy Rehabilitation protocol was implemented targeting bulbar dysfunction, respiratory compromise, limb weakness, and fatigue.

Table – 2: Comprehensive Rehabilitation Protocol

Component Affected	Goal	Physiotherapy Intervention
Speech & Swallowing	Improve bulbar control	Swallow exercises, oromotor training
Respiratory muscles	Enhance ventilation	Diaphragmatic breathing, assisted cough
Facial muscles	Improve expressions	Facial strengthening exercises
Mobility & posture	Maintain independence	ROM exercises, gait training
Fatigue	Improve endurance	Energy conservation strategies
Pain	Reduce discomfort	Joint mobilization
Flexibility	Prevent contractures	Passive & active stretching
Strength	Maintain muscle function	Low-to-moderate resistance training
Emotional well-being	Improve coping	Relaxation techniques

Follow-up and outcome measure the patient underwent 8 weeks of supervised physiotherapy.

Table – 3: Pre- and Post-Rehabilitation MMT Scores

Joint	Pre (R/L)	Post (R/L)
Shoulder	3+/5 – 3/5	4/5 – 4/5
Elbow	3+/5 – 3+/5	5/5 – 5/5
Wrist	3+/5 – 3+/5	4/5 – 4/5
Hip	3/5 – 3/5	4/5 – 4/5
Knee	3/5 – 3/5	4/5 – 4/5
Ankle	3+/5 – 3+/5	4/5 – 4/5

Table – 4: Outcome Measure Comparison

Outcome Tool	Pre-Treatment	Post-Treatment
VAS	7	3
DOSS	Level 2	Level 5
FOIS	3	5
HBS	Grade 4	Grade 3

MMT – Manual Muscle Testing, VAS – Visual Analog Scale, DOSS – Dysphagia Outcome Severity Scale, FOIS – Functional Oral Intake Scale, HBS – House–Brackmann Score

Clinical Outcome Summary After 8 weeks of structured physiotherapy The patient demonstrated measurable functional gains despite progressive bulbar Amyotrophic Lateral Sclerosis pathology. Which includes Muscle strength improved in all major groups, swallowing safety improved significantly (DOSS Level 2 → 5), Oral intake progressed from modified diet to functional intake, Facial symmetry improved, Pain reduced substantially, Respiratory endurance improved.

DISCUSSION: Bulbar-onset Motor Neuron Disease presents significant rehabilitation challenges due to early involvement of cranial motor nuclei, progressive dysphagia, dysarthria, and respiratory compromise. The primary objective of physiotherapy in such patients is not curative but functional optimization — preserving independence, preventing secondary complications, and enhancing quality of life despite ongoing neurodegeneration.[12]

In the present case, the rehabilitation strategy was structured around a multidomain impairment model addressing bulbar dysfunction, respiratory decline, generalized limb weakness, fatigue, and psychosocial adaptation. The individualized approach allowed targeted intervention while accommodating the progressive nature of the disorder.

Bulbar involvement significantly impairs speech articulation, swallowing coordination, and facial expression. In this patient, repetitive task-specific oromotor exercises were implemented to strengthen residual neuromuscular control and delay deterioration of swallowing safety.[13] The strategy incorporated- Structured tongue and lip resistance training, Controlled swallow maneuvers, Postural adjustments (chin-tuck strategy), Adaptive communication techniques.

This approach aligns with principles of activity-dependent neuroplasticity, where repetitive activation of surviving motor units may enhance synaptic efficiency and optimize residual motor output. Although motor neuron loss in Amyotrophic Lateral Sclerosis is irreversible, compensatory reorganization within intact neural circuits may improve functional coordination. Importantly, therapy was adjusted dynamically to match disease progression, maintaining present function while preparing for anticipated decline.[14]

Respiratory muscle weakness is a major determinant of morbidity and mortality in bulbar MND. The patient's prior COVID-related pulmonary fibrosis compounded neuromuscular respiratory compromise, necessitating proactive intervention. Respiratory rehabilitation focused on, Diaphragmatic breathing training, Inspiratory muscle strengthening, Assisted cough techniques, Airway clearance strategies. These interventions aim to preserve forced vital capacity, improve secretion clearance, and reduce risk of aspiration pneumonia.[15] Early respiratory physiotherapy has been shown to delay ventilatory dependence and improve symptom burden in ALS populations. The integration of breathing retraining also enhanced voluntary respiratory control, reducing fatigue during speech and functional tasks.

Although MND is characterized by progressive motor neuron degeneration, surviving neurons can exhibit adaptive sprouting and compensatory reinnervation.[16] Motor relearning strategies in this patient were grounded in the concept of optimizing neural efficiency rather than restoring lost neurons.

Mechanisms of Motor Relearning in This Case, Task-Specific Training Functional activities such as sit-to-stand, reaching, and gait training promoted coordinated motor patterns rather than isolated strengthening. Repetition and Graded Challenge. Structured repetition reinforced correct movement patterns while gradually increasing task complexity to prevent overwork weakness.[17] Mirror Feedback and Visual Cueing Visual feedback improved motor awareness and minimized compensatory asymmetry in facial and limb movements. Facilitation Techniques Proprioceptive and tactile cues enhanced muscle activation in weak motor units.[17,18]

Through repeated activation, synaptic strengthening and improved motor unit recruitment may occur in remaining neural circuits. While disease progression continues, improved motor efficiency enables better performance of daily activities with reduced energy expenditure.[19]

Generalized limb weakness significantly compromises independence and increases fall risk. In this case, low-to-moderate intensity resistance training was implemented cautiously to avoid overwork fatigue, a known concern in ALS management. Preservation of joint range of motion, Prevention of contractures, Functional strength training, Balance and gait re-education. This functional rehabilitation model prioritizes meaningful activity over isolated muscle strengthening, contributing to improved participation in activities of daily living.[18,19]

Fatigue is a pervasive symptom in MND, often multifactorial in origin, including neuromuscular inefficiency, respiratory compromise, and psychological burden. Energy conservation techniques, pacing strategies, and structured rest intervals were incorporated into the rehabilitation plan. By optimizing task sequencing and reducing unnecessary muscular effort, the patient demonstrated improved endurance and reduced perceived exertion.[19,20]

A key component of successful management in this case was regular reassessment and collaborative care involving neurologists, physiotherapists, and speech therapists. The progressive nature of bulbar MND necessitates continual adaptation of therapeutic goals. This coordinated approach ensured by Early detection of functional decline, Timely modification of exercises, Prevention of secondary complications.[20]

Despite advanced age and multiple comorbidities, including prior stroke and post-COVID pulmonary fibrosis, the patient demonstrated measurable improvements in muscle strength, swallowing safety, pain levels, and respiratory endurance following 8 weeks of structured physiotherapy. This reinforces, Rehabilitation in MND should begin early, Motor relearning principles remain valuable even in degenerative conditions, Functional gains are achievable despite progressive pathology, Multidomain intervention improves overall quality of life.

Although MND remains incurable, targeted physiotherapy plays a critical role in maximizing residual function and slowing functional decline.

CONCLUSION: This case report demonstrates that personalized physiotherapy plays a pivotal role in the comprehensive management of bulbar-onset Motor Neuron Disease. Through targeted and adaptable interventions addressing respiratory function, speech and swallowing coordination, muscle strength, mobility, fatigue management, and emotional well-being, the patient achieved measurable functional improvements despite the progressive nature of the disease. The findings reinforce the importance of an individualized, multidisciplinary rehabilitation model involving physiotherapists, respiratory specialists, speech therapists, and neurologists to ensure holistic and patient-centered care. By optimizing residual motor function, minimizing secondary complications, and promoting independence in daily activities, physiotherapy significantly enhances quality of life in patients with bulbar Amyotrophic Lateral Sclerosis. Therefore, structured neurorehabilitation should be considered a cornerstone of long-term management in individuals affected by this complex neurodegenerative condition.

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