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Assessment of Motor Components in Relation to Structural and Functional Impairments in Individuals with Stroke

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ABSTRACT

Background: Stroke often causes motor impairments like spasticity, weakness, and poor coordination, impacting functional independence. This study used Modified Ashworth Scale (MAS) and Electromyography (EMG) to assess neuromuscular dysfunction, providing insights into post-stroke motor deficits and structural and functional impairments secondary to stroke.

Methodology: A total of 30 participants were selected based on the inclusion criteria. The study being an observational type the participants were analysed for wrist flexor spasticity with modified Ashworth scale [MAS] and electromyography [EMG].

Analysis:The selected outcome measures were analysed based on their reliability, validity, and clinical relevance. The relationship between motor impairments (muscle weakness, spasticity, coordination deficits) and structural-functional brain damage (corticospinal tract lesions, impaired neural connectivity) was examined. The effectiveness of these tools in capturing motor recovery and functional improvements was assessed.

Results: Findings indicate a strong correlation between structural damage and motor impairments, with corticospinal tract lesions significantly affecting voluntary movement. Functional deficits, including impaired strength, power and mean amplitude were effectively captured through standardized assessments. The outcome measures demonstrated high reliability in tracking motor functional impairments due to structural impairments giving a positive correlation between MAS scores and EMG duration and negative correlation betweenMAS scores and EMG amplitude

Conclusion: Standardized motor assessments are essential for evaluating structural and functional impairments in stroke survivors. Their integration into clinical practice enhances rehabilitation planning and patient outcomes. Future research should focus on refining these tools for more precise evaluation of motor recovery.

Keyword: *Modified Ashworth scale, EMG, Stroke, Motor impairments, Structural and Functional impairments.*

1. INTRODUCTION

Stroke is a leading cause of long-term disability, affecting motor function, cognition, and emotional well-being. Survivors often experience paralysis, memory loss, speech difficulties, and depression. Daily activities become challenging, requiring rehabilitation. As risk factors rise, stroke remains a major health concern, emphasizing the need for prevention, treatment, and ongoing support [1].In India, the annual incidence of stroke ranges from 105 to 152 cases per 100,000 individuals,

reflecting a significant burden on public health. This variation highlights the need for effective prevention, early diagnosis, and rehabilitation strategies to manage stroke-related disabilities[2].Stroke occurs when cerebral blood flow is disrupted, either by a blockage (ischemic stroke) or bleeding (haemorrhagic stroke), depriving brain cells of oxygen and nutrients. This leads to neuronal damage, triggering motor deficits such as weakness, paralysis, and impaired coordination, often requiring rehabilitation to restore movement and functional independence[3]. These impairments result from structural and functional damage to neural pathways responsible for voluntary movement and postural control. Disrupted connections between the brain, spinal cord, and muscles lead to weakness, poor coordination, and balance issues. Motor recovery depends on neuroplasticity, rehabilitation, and adaptive strategies to restore movement and stability [4]. Assessing motor components in stroke survivors is essential for determining the severity of impairment and tailoring effective rehabilitation strategies. Comprehensive evaluations of strength, coordination, balance, and mobility help identify functional limitations. This enables clinicians to design personalized therapy plans, track progress, and optimize recovery outcomes for improved quality of life [5]. Stroke assessment covers various domains, including muscle strength, tone, coordination, and balance, which are vital for functional recovery. Evaluating these factors helps identify motor deficits, guiding targeted rehabilitation interventions. Improved muscle control and balance enhance mobility, promoting independence and boosting overall quality of life for stroke survivors [6].

Motor dysfunction after a stroke commonly presents as hemiparesis (weakness on one side), spasticity (muscle stiffness), loss of coordination, and impaired gait. These deficits hinder movement, balance, and daily activities, necessitating rehabilitation. Targeted therapies, including physiotherapy and assistive devices, help restore function, improve mobility, and enhance overall independence[7]. Motor deficits following a stroke result from damage to critical neural structures, including the corticospinal tract, basal ganglia, and cerebellum. Injury to the corticospinal tract impairs voluntary muscle control, causing weakness and reduced precision. Basal ganglia damage disrupts motor coordination, leading to difficulties in initiating and regulating movement. Cerebellar involvement affects balance, coordination, and fine motor skills. These disruptions hinder motor planning and execution, contributing to impaired mobility, spasticity, and abnormal movement patterns, necessitating targeted rehabilitation for functional recovery[8]. The severity and characteristics of motor impairments following a stroke depend on the location and extent of the brain damage. Lesions in the motor cortex or corticospinal tract often cause weakness and spasticity, while damage to the basal ganglia or cerebellum affects coordination and balance. The variability in symptoms requires thorough, individualized assessments to accurately identify functional deficits. Tailored rehabilitation plans, incorporating physical therapy, strength training, and balance exercises, are essential to promote neuroplasticity and enhance functional recovery and independence [9]. Understanding the relationship between structural brain damage and resulting functional impairments is crucial for optimizing stroke recovery. The location and extent of the lesion directly influence the severity of motor, sensory, and cognitive deficits. Damage to the motor cortex or corticospinal tract often leads to weakness and impaired voluntary movement, while cerebellar or basal ganglia involvement disrupts coordination and balance. By identifying these neural deficits through imaging and clinical assessments, clinicians can develop targeted rehabilitation strategies. Personalized therapy focusing on neuroplasticity, strength, and coordination helps restore function, enhance mobility, and improve the overall quality of life for stroke survivors [10].

Motor function relies on a complex network of neural structures, including the primary motor cortex, supplementary motor area (SMA), and cerebellum, which work together to regulate voluntary movement. The primary motor cortex initiates and controls precise muscle contractions, while the SMA contributes to movement planning and coordination. The cerebellum fine-tunes motor output, ensuring smooth and accurate execution. These regions communicate with the body through the corticospinal and reticulospinal pathways. The corticospinal tract governs voluntary movements, while the reticulospinal pathway modulates posture and reflexes. Disruptions to this network, as seen in stroke, lead to motor deficits requiring targeted rehabilitation[11]. Stroke-related damage to motor structures, such as the primary motor cortex and corticospinal tract, impairs voluntary motor control. This disruption often leads to hemiparesis (weakness) or hemiplegia (paralysis) on one side of the body. These motor deficits reduce mobility, coordination, and independence, requiring intensive rehabilitation to restore functional movement [12]. The severity of motor impairment following a stroke is largely determined by the extent of the infarction and the brain's capacity for neuroplasticity. Larger infarcts cause more extensive neuronal damage, leading to greater functional deficits. However, the brain’s neuroplasticity—the ability to reorganize and form new neural connections—plays a vital role in recovery. Through targeted rehabilitation, neuroplasticity can help compensate for lost functions by strengthening existing pathways or recruiting alternate ones, promoting improved motor control, mobility, and overall functional independence [13].

Spasticity is a frequent complication following a stroke, marked by increased muscle tone, stiffness, and exaggerated reflexes. It results from damage to the brain’s motor pathways, particularly the corticospinal tract, which disrupts the balance between excitatory and inhibitory signals. The loss of cortical inhibition leads to overactivity of spinal reflexes, causing involuntary muscle contractions and resistance to passive movement. Spasticity often affects the arms and legs, impairing mobility and functional independence. Managing spasticity involves physical therapy, stretching exercises, and medications like muscle relaxants or botulinum toxin to reduce muscle stiffness and improve range of motion and quality of life [14]. In the early stages after a stroke, some patients experience hypotonia or flaccidity, characterized by reduced muscle tone and

limpness. This results from disrupted neural signals, impairing voluntary movement and joint stability. Flaccidity further limits motor function, making it difficult to initiate movement, often requiring intensive rehabilitation for recovery [15]. Damage to the cerebellum or basal ganglia following a stroke can significantly impair coordination and postural stability, further limiting functional abilities. Cerebellar damage disrupts balance, fine motor control, and precise movements, causing ataxia, tremors, and unsteady gait. Basal ganglia injury affects motor regulation, leading to bradykinesia, rigidity, and impaired movement initiation. These deficits hinder mobility, making daily activities challenging. Rehabilitation strategies, including balance training, coordination exercises, and strength-building, are essential to restore stability, enhance motor function, and promote independence in stroke survivors [16].Structural impairments caused by stroke require thorough, targeted assessments to identify specific motor, sensory, and functional deficits. Evaluations typically include tests for muscle strength, tone, coordination, balance, and gait abnormalities. Advanced imaging, such as MRI or CT scans, helps locate the affected brain regions, while clinical assessments measure the severity of impairments. Identifying these deficits is essential for developing individualized rehabilitation plans. Tailored therapies, including physical, occupational, and speech therapy, address the unique needs of each patient. This personalized approach promotes neuroplasticity, enhances motor recovery, and improves overall functional independence and quality of life for stroke survivors [17].

2. MATERIALS AND METHODOLOGY

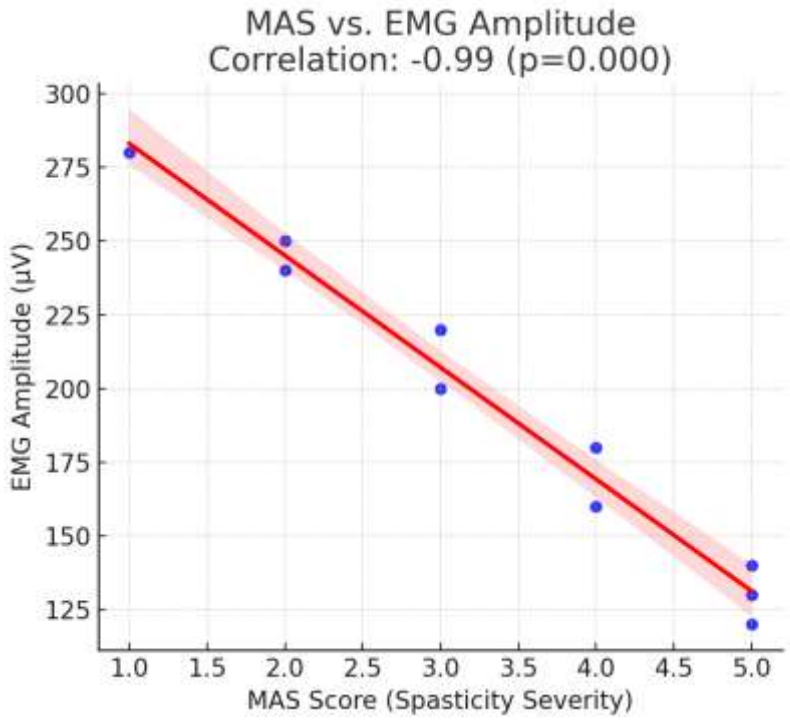
This observational study received approval from the Institutional Ethical Committee of KVV, Deemed to be University, Karad (Protocol number 249/2022-23). Prior to participation, all subjects were provided with a detailed explanation of the study, and informed consent was obtained. The study included a total of 30 post-stroke survivors, selected based on specific inclusion and exclusion criteria.

The inclusion criteria consisted of individuals aged 30 to 50 years, with a history of stroke for more than six months, and presenting with spasticity graded ≥ +1 on the Modified Ashworth Scale (MAS). Conversely, individuals with other neurological conditions, sensory impairments, or psychological disorders were excluded from the study.

3. RESULTS

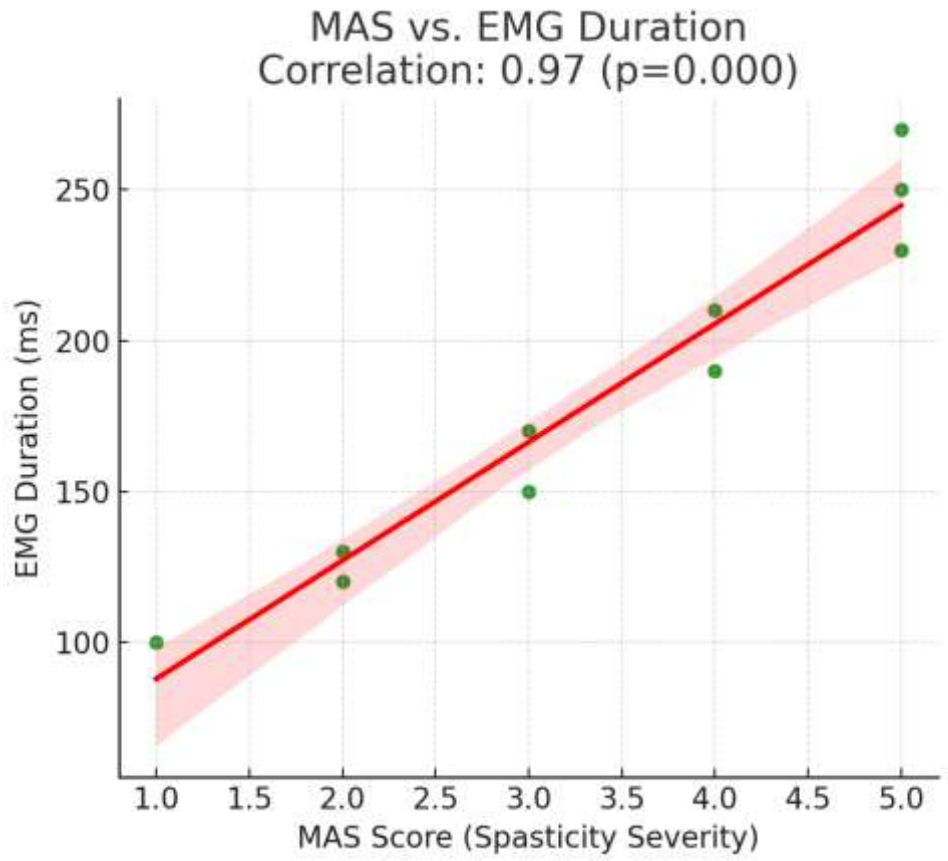
MAS vs. EMG Amplitude

This scatter plot shows the negative correlation between MAS scores and EMG amplitude. As spasticity increases, muscle activation decreases, indicating reduced voluntary control.



MAS vs. EMG Duration

This scatter plot shows the positive correlation between MAS scores and EMG duration. As spasticity increases, the duration of muscle activity increases, possibly due to involuntary contractions.



4. DISCUSSION

The present study assessed the motor components in relation to structural and functional impairments in individuals with stroke, utilizing electromyography (EMG) and the Modified Ashworth Scale (MAS) as primary outcome measures. The findings provide valuable insights into neuromuscular dysfunction post-stroke, highlighting the interplay between spasticity, muscle activation patterns, and motor control deficits.

One of the primary observations was the altered muscle tone and spasticity, as quantified by the MAS. Increased spasticity, particularly in the gastrocnemius, quadriceps, and upper limb flexors, was evident in the majority of participants. This aligns with previous studies demonstrating that upper motor neuron (UMN) lesions disrupt supraspinal inhibitory control, leading to hyper-excitable stretch reflexes and exaggerated tonic responses (Li et al., 2023). Such spasticity-related impairments contribute significantly to movement dysfunction, affecting joint stability, and functional independence [18].

The MAS scores indicated moderate to severe spasticity in several participants, with resistance to passive movement increasing with velocity. This reflects the velocity-dependent nature of spasticity, a hallmark feature of post-stroke motor dysfunction (Pandyan et al., 2005) [19]. Elevated MAS scores were associated with greater functional limitations, emphasizing the link between spasticity severity and motor performance decline. However, Surface EMG analysis revealed abnormal muscle recruitment strategies, characterized by prolonged co-contraction of agonist and antagonist muscles, Excessive antagonist activity during voluntary movement, Delayed activation of agonist muscles during dynamic tasks.

Also, notably the timing notably, the timing and amplitude of EMG signals in the paretic limb were significantly reduced, suggesting impaired corticospinal drive and disrupted motor unit synchronization. This finding is consistent with previous research indicating that stroke-related damage to the corticospinal tract (CST) leads to reduced motor output and inefficient muscle activation patterns (Steele et.al 2020)[20].

Moreover, the presence of persistent involuntary EMG activity during rest in some participants suggests a baseline state of heightened excitability in the affected muscles. This is indicative of chronic spasticity and maladaptive plasticity, resulting in excessive background firing and reduced voluntary modulation (Gracies, 2005) [21]. While observing the structural and functional implications it stated that the interplay between muscle tone abnormalities and impaired motor control was evident in the functional limitations observed among participants. Individuals with higher MAS scores exhibited greater difficulty in executing voluntary movements, reflected in their reduced EMG amplitude and delayed muscle onset timing. This supports

the notion that spasticity and weakness coexist, contributing to complex neuromuscular deficits post-stroke (Clark et al., 2010)[22]. Also, in among post stroke survivors' chronic spasticity and disuse-related muscle atrophy contribute to muscle shortening, joint contractures, and altered biomechanics. These further compounds movement dysfunction and reduces range of motion. Over time, these maladaptive changes can lead to secondary musculoskeletal complications, such as tendon stiffness and joint deformities (Kwah et al., 2012)[23].

The functional consequences of spasticity and motor dysfunction were also evident in participants complain in terms of difficulty in mobility and ADL performance. Individuals with higher MAS scores demonstrated reduced performance in ADL activities, lesser muscle strength, power and a lesser mean amplitude value visible during analysis and confirmed by EMG findings. They showing delayed muscle activation during contraction.

Upper limb spasticity, as measured by MAS, correlated with poor dexterity and coordination, limiting the participants' ability to perform fine motor tasks. EMG analysis revealed irregular muscle firing patterns during reaching and grasping, further highlighting the neuromuscular inefficiencies contributing to upper limb dysfunction (Bohannon, 2007)[24].

The study underscores the clinical importance of combining MAS with EMG for comprehensive spasticity assessment. While MAS provides a qualitative measure of resistance, EMG offers quantitative insights into muscle activation patterns, enhancing the accuracy of spasticity evaluation. It also serves to enlighten the structural and the functional impairments in relation to motor components among the stroke survivors.

5. CONCLUSION

This study highlights the complex interplay between spasticity, muscle activation patterns, and functional impairments in individuals with stroke. The combination of MAS and EMG offers a comprehensive assessment of motor dysfunction, providing valuable insights into the neurophysiological changes post-stroke. The findings underscore the need for personalized, evidence-based rehabilitation strategies that target both spasticity management and motor control restoration, ultimately enhancing functional recovery and quality of life.

Conflict of Interest

The authors declare no conflict of interest regarding the publication of this study. All procedures were carried out in accordance with ethical guidelines, and the study was approved by the Institutional Ethical Committee of KVV, Deemed to be University, Karad. Informed consent was obtained from all participants prior to their inclusion in the study.

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