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Original Research Article

Impact of Calf Muscle Spasticity on Tibialis Anterior Muscle Activation in Chronic Stroke Patients: A Cross-Sectional Study

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ABSTRACT

Background: Calf muscle spasticity is a common consequence after stroke and can impede the functional activation of antagonist muscles such as the tibialis anterior, affecting gait and mobility.

Objective: To determine the relationship between calf muscle spasticity severity (via Modified Ashworth Scale, MAS) and tibialis anterior activation (via RMS, surface EMG) in chronic stroke patients.

Methods: Cross-sectional study on 108 chronic stroke patients aged 35–65 years. Variables included Age, Gender, Side, MAS for calf, and tibialis anterior RMS. Spearman correlation and Kruskal-Wallis group analysis were applied.

Results: There was a significant negative correlation between MAS and RMS (ñ = –0.42, p < 0.001). RMS of tibialis anterior decreased with increasing calf muscle spasticity.

Conclusion: Greater calf muscle spasticity is associated with reduced tibialis anterior activation, with important implications for gait rehabilitation and physiotherapy planning.

KEYWORDS: Stroke, Spasticity, Tibialis Anterior, Surface Electromyography, Modified Ashworth Scale, Foot Drop, Chronic Stroke.

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INTRODUCTION

Stroke remains one of the leading causes of mortality and long-term disability worldwide, with a rising burden particularly in low- to middle-income countries (1). Globally, millions of individuals experience a first-ever stroke each year, and the resulting physical, cognitive, and psychological impairments place a substantial strain on patients, families, and healthcare systems (2). A critical aspect of post-stroke disability is impaired mobility, most commonly due to motor dysfunction and muscle tone abnormalities (1).

Spasticity, defined as a velocity-dependent increase in muscle tone with exaggerated tendon jerks, develops in around 19–43% of stroke survivors, often within weeks to months following the index event (3,4).

Post-stroke spasticity most frequently affects the lower limbs, with the calf musculature (gastrocnemius and soleus) being particularly vulnerable (5,6).

Calf muscle spasticity is clinically important as it contributes to equinovarus deformity, impedes dorsiflexion, and leads to substantial gait disturbances such as reduced step length, decreased walking speed, and increased risk of falls (7,8).

The tibialis anterior muscle is the primary ankle dorsiflexor and is crucial for foot clearance in the swing phase and controlled foot placement during stance (9). Diminished activation of the tibialis anterior, whether due to direct weakness or reciprocal inhibition arising from spastic antagonists, leads to gait abnormalities such as foot drop and compensatory hip/knee movements (10,11). Such functional deficits have direct implications for independence and quality of life in stroke survivors.

Understanding the interplay between calf muscle spasticity and tibialis anterior activation is thus essential for targeted rehabilitation (12). Insights into this relationship can inform physiotherapy and management strategies, especially when planning interventions aimed at improving ambulation, safety, and functional outcomes. While EMG techniques provide objective quantification of muscle activation, and standardized clinical scales such as the Modified Ashworth Scale (MAS) reliably assess spasticity severity, there remains a gap in research directly linking these parameters in chronic stroke populations (13,14).

Therefore, the objective of this study is to assess the effect of calf muscle spasticity on tibialis anterior activation in chronic stroke patients, hypothesizing that increased spasticity is associated with reduced antagonist muscle activity.

MATERIALS AND METHODS

Study Design and Setting: This study was designed as a cross-sectional observational analysis conducted at S.S. Agrawal institute of physiotherapy and Medical Care Education, Navsari. The study protocol adhered to ethical standards and was approved by the institutional review board. Written informed consent was obtained from all participants prior to inclusion.

Participants: A total of 108 chronic stroke patients were enrolled according to the following criteria:

Inclusion Criteria: Adults aged 35 to 65 years, diagnosed with unilateral chronic stroke (post-onset duration greater than 6 months), who demonstrated calf muscle spasticity and were able to participate in the study assessments.

Exclusion Criteria: Patients with neuromuscular or musculoskeletal conditions other than stroke, orthopedic deformities affecting lower limbs, cognitive impairments preventing informed consent or cooperation, or those receiving treatments influencing muscle tone (e.g., botulinum toxin) within the last 3 months were excluded.

Sample Size Calculation

Sample size was computed to detect a moderate correlation (r = 0.4) between the Modified Ashworth Scale (MAS) score and tibialis anterior electromyographic activation with 80% power and a two-sided significance level of 0.05. Using the following formula:

$$n = \left(\frac{Z_{1-\alpha/2} + Z_{1-\beta}}{0.5 \times \ln\left(\frac{1+r}{1-r}\right)} \right)^2 + 3$$

where $Z_{1-\alpha/2}$ = 1.96 for α = 0.05 and $Z_{1-\beta}$ = 0.84 for β= 0.20, the minimum required sample size was approximately 47 participants (15,16). To enhance the robustness of findings, 108 patients were ultimately recruited

Variables and Outcome Measures

Demographic Variables: Age, Gender, Height, Weight, and Affected Side.

Independent Variable: Calf muscle spasticity quantified using the Modified Ashworth Scale (MAS), a widely accepted clinical tool for grading spasticity (13).

Dependent Variable: Tibialis anterior muscle activation quantified by root mean square (RMS) amplitude obtained through surface electromyography (Semg). sEMG recordings were performed using standard sensor placement guidelines to ensure reliability and validity (14).

Data Collection Procedure

Spasticity was evaluated clinically using the MAS by an experienced physiotherapist blinded to the EMG results. The MAS grades used were 1, 1+, and 2, consistent with established post-stroke spasticity assessments.

For sEMG, electrodes were placed over the belly of the tibialis anterior on the involved side following SENIAM (Surface ElectroMyoGraphy for the Non-Invasive Assessment of Muscles) recommendations. The RMS of the muscle activity was recorded during a controlled dorsiflexion task.

Statistical Analysis

Descriptive statistics were computed for demographic and clinical variables, presented as mean ± standard deviation for continuous variables and frequency (%) for categorical variables.

Normality of RMS data was assessed using Shapiro-Wilk and Kolmogorov-Smirnov tests. Due to non-normal distribution ($p < 0.001$), nonparametric statistical methods were employed.

Spearman's rank correlation coefficient was used to examine the relationship between MAS scores and tibialis anterior RMS amplitude. Group-wise comparisons of RMS values across MAS categories (1, 1+, and 2) were conducted using the Kruskal-Wallis test. Statistical significance was set at $p < 0.05$.

RESULTS

Descriptive Statistics: The study included 108 chronic stroke patients with a mean age of **52.77 ± 9.76 years** (range 35–65 years). The mean height was **162.32 ± 9.45 cm**, mean weight was **72.20 ± 13.49 kg**, and mean tibialis anterior root mean square (RMS) electromyographic activity was **51.53 ± 26.66 µ V** (range 4.95–130.80µV).

Table 1: Descriptive Characteristics of Participants.

z	N	Minimum	Maximum	Mean	SD
Age (years)	108	33	66	52.77	9.76
Height (cm)	108	139	182	162.32	9.45
Weight (kg)	108	42	108	72.2	13.5
RMS PRE (µV)	108	4.95	130.8	51.53	26.66

Demographic Distribution: Among the participants, **63.9% were male** and **36.1% female**. The majority had involvement on the **right side (60.2%)**, while the remaining **39.8% had left-sided involvement**.

Table 2: Distribution by Sex and Involved Side.

Variable	Category	Frequency	Percentage
Sex	Male	69	63.90%
	Female	39	36.10%
Involved side	Right	65	60.20%
	Left	43	39.80%

Modified Ashworth Scale (MAS) Distribution: The MAS for calf muscle showed that **46.3% of patients had grade 2 spasticity**, **39.8% had grade 1+**, and **13.9% had grade 1**.

Table 3: Distribution of MAS Scores.

MAS PRE	Frequency	Percentage
1	15	13.90%
1+	43	39.80%
2	50	46.30%

Normality Testing

The Shapiro–Wilk and Kolmogorov–Smirnov tests indicated that RMS values were **not normally distributed** ($p < 0.001$). Hence, nonparametric tests were applied for further analysis.

Correlation Between Spasticity and Tibialis Anterior Activation

Spearman's rho analysis revealed a **significant negative correlation** between MAS and RMS values (**$\rho = -0.420$, $p < 0.001$**). This suggests that **increasing calf spasticity is associated with reduced tibialis anterior activation**.

Table 4: Spearman's Correlation Between MAS and RMS.

Variable	Correlation (ρ)	p-value
MAS PRE vs RMS PRE	–0.420**	<0.001

($p < 0.01$, significant)

Group-Wise Comparison of RMS by MAS Grade

The Kruskal–Wallis test showed a **statistically significant difference** in RMS across MAS categories ($\chi^2 = 19.498$, df = 2, $p < 0.001$).

Patients with **MAS = 1** had the highest RMS mean rank (**80.47**).

Those with **MAS = 1+** had moderate activation (**59.92**).

Patients with **MAS = 2** had the lowest RMS mean rank (**42.05**).

This confirms that **as spasticity severity increases, tibialis anterior activation decreases significantly**.

Table 5: Kruskal–Wallis Test for RMS Across MAS Grades.

MAS PRE	N	Mean Rank
1	15	80.47
1+	43	59.92
2	50	42.05
Total	108	–

Test Statistics: $\chi^2 = 19.498$, df = 2, $p < 0.001$

Interpretation

The majority of patients had moderate to severe spasticity (MAS 1+–2).

Tibialis anterior RMS activity progressively decreased with increasing calf spasticity.

The negative correlation and significant group differences strongly support the hypothesis that **calf muscle spasticity interferes with tibialis anterior activation in chronic stroke patients**.

DISCUSSION

This study examined the relationship between calf muscle spasticity and tibialis anterior muscle activation in chronic stroke patients, demonstrating a significant negative correlation between spasticity severity (measured by MAS) and antagonist muscle activation (measured by tibialis anterior RMS via sEMG). Our findings align with previous reports highlighting the detrimental impact of increased muscle tone on reciprocal muscle activity, which is essential for coordinated and efficient gait post-stroke (10,11).

The inverse relationship observed suggests that as calf spasticity worsens, functional activation of the tibialis anterior diminishes. This reduced dorsiflexor activation likely contributes to common post-stroke gait abnormalities including foot drop, impaired foot clearance, and compensatory gait patterns (10,17). Such impairments increase the risk of falls and reduce mobility, impairing independence and quality of life (18,19).

Pathophysiologically, spasticity results from upper motor neuron lesions causing disordered reflex circuits and excessive excitability of stretch reflexes, which can inhibit antagonist muscle groups through reciprocal inhibition mechanisms (12). The gastrocnemius-soleus complex's spasticity may specifically limit tibialis anterior recruitment during dorsiflexion tasks; an effect reflected in the reduced RMS amplitudes recorded here (6,7).

The use of surface EMG in this study provides objective quantitative data on muscle activation changes post-stroke, supplementing clinical assessments of spasticity such as the Modified Ashworth Scale (20–22). EMG metrics allow for nuanced evaluation of motor system impairments, informing rehabilitation strategies to optimize motor recovery. The MAS, despite some limitations, remains a clinically practical and validated tool widely used in stroke spasticity evaluation.

Contrasting with some literature suggesting variable co-activation patterns depending on stroke chronicity and severity (23), our results indicate a predominant inhibitory effect of spasticity on antagonist muscle function in a chronic stroke cohort. This emphasizes the importance of comprehensive assessment of both agonist and antagonist muscles when designing rehabilitation programs, highlighting physiotherapy approaches targeting spasticity reduction and tibialis anterior strengthening to restore gait efficiency (24,25).

Strengths : This study's strengths include a relatively large sample size for sEMG studies, standardized measurement protocols, and the application of both clinical and objective assessments. The focus on chronic stroke patients adds valuable insight into long-term motor sequelae beyond acute phases.

Limitations

However, several limitations warrant consideration. The cross-sectional design precludes causal inferences between spasticity and muscle activation. The focus on a single antagonist muscle and measurement during isolated dorsiflexion tasks may limit generalizability to complex functional movements like walking. Effects of prior therapy, medications, or stroke lesion characteristics were not controlled, potentially confounding results.

Future Recommendations

Future studies should include longitudinal designs to assess temporal changes in muscle activation with rehabilitation, incorporate multi-muscle EMG analyses during dynamic gait tasks, and explore interventions directly targeting spasticity and antagonist activation interplay. Inclusion of neuroimaging or neurophysiological correlates could elucidate underlying mechanisms and guide personalized therapy.

CONCLUSION

In conclusion, this study demonstrates that greater calf muscle spasticity is associated with decreased tibialis anterior activation in chronic stroke patients. These findings support integrative rehabilitative approaches combining spasticity management and antagonist muscle strengthening to improve gait function and patient outcomes.

List of Abbreviations

MAS – Modified Ashworth Scale
RMS – Root Mean Square
sEMG – Surface Electromyography
TA – Tibialis Anterior
SD – Standard Deviation

Authors' Contributions

JP: Conceived the study, collected data, performed statistical analysis, interpreted results, and drafted the manuscript. **DS:** Contributed to study design, supervised the research, critically revised the manuscript, and approved the final version.

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