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ORIGINAL ARTICLE

Exploring the Relationship Between Muscle Tone, Echotexture, and Walking Speed in Chronic Stroke Patients: A Pilot Analysis

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ABSTRACT

Background: Spasticity and changes in muscle structure are known to influence walking ability after stroke. While the Modified Ashworth Scale (MAS) and Modified Heckmatt Scale (MHS) are commonly used in clinical practice, their relationship to functional outcomes such as walking speed remains unclear.

Objective: This pilot study explored the relationship between spasticity, muscle echotexture, and gait speed in patients with chronic stroke.

Methods: Eight ambulatory patients, 8 – 24 months post first stroke, were assessed at a neurorehabilitation clinic. Muscle tone was measured using MAS, muscle echotexture using MHS, and walking speed derived from the 6-minute walk test. Data were analyzed using Spearman's rank correlation.

Results: The mean gait speed was 0.82 ± 0.21 m/s, mean MAS score was 2.1 ± 0.6, and mean MHS score was 2.7 ± 0.5. MAS and MHS showed weak to moderate correlations (ρ = 0.067–0.417), while MHS and gait speed demonstrated very weak negative associations (ρ = –0.126 to –0.206). None of the associations reached statistical significance ($p > 0.05$).

Conclusion: Although no significant correlations were found, these findings provide valuable early insights. Larger and more diverse studies, particularly involving subacute stroke patients and stratification by Brunnstrom stage, are needed to clarify how spasticity and muscle quality contribute to mobility outcomes in stroke rehabilitation.

Keyword: *chronic stroke, muscle tone, echotexture, walking speed, ultrasonography*

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INTRODUCTION

Stroke continues to pose a major global health challenge, ranking among the foremost causes of long-term disability and affecting more than 12 million individuals annually.[1] One of the most prevalent sequelae of stroke is spasticity, a motor disorder characterized by velocity-dependent hypertonia resulting from exaggerated stretch reflexes. This condition often contributes to functional impairment, especially in gait and balance, when affecting the lower limbs.[2]

The Modified Ashworth Scale (MAS) has long been the most frequently employed clinical instrument for assessing spasticity. Its widespread use sterms from its practicality: the tool is rapid to administer, requires no specialized equipment, and can be applied even in resource-constrained clinical environments. Moreover, MAS demonstrates adequate sensitivity to change, allowing clinicians to track improvements following interventions such as botulinum toxin injections or physical therapy. Despite these strengths, the MAS is not without limitations. Its subjective scoring and mode's inter-rater reliability often draw criticism, and importantly, the scale does not discriminate between neural factors (such as reflex hyperexcitability) and structural contributors (such as soft tissue stiffness) that both influence muscle tone. [2,3]

In addition to neural hyperexcitability, stroke induces progressive alterations in skeletal muscle morphology on both the affected and unaffected sides. Documented changes include reductions in cross-sectional area, accumulation of intramuscular fat, fiber-type transformation towards type IIx, and diminished capillary density. These changes are aggravated by inactivity and impaired central motor drive, and typically evolve within weeks pf the initial event, continuing into the chronic phase. Such remodeling contributes not only to weakness and fatigability but also to impaired motor coordination. The gastrocnemius-soleus complex is particularly vulnerable to these alterations, which substantially compromise propulsive power during gait. However, these structural changes often remain undetected by clinical scales that primarily evaluate tone. [6,8]

The Modified Heckmatt Scale (MHS) is a sonographic grading system that evaluates muscle echogenicity as a reflection of pathological changes like fibrosis and fatty degeneration. [9,10] This scale has demonstrated reliability in quantifying chronic muscle changes in spastic patients, and recent improvements in its grading via Rasch analysis have enhanced its utility in research and clinical settings. [11] However, there is still limited evidence regarding how well sonographic muscle characteristics correlate with traditional spasticity scores or functional measures like gait speed in stroke populations. [6]

Walking speed is increasingly recognized as a robust marker of post-stroke recovery, with strong associations to community ambulation, independence, and long-term outcomes.[6] although spasticity has traditionally been viewed as a primary determinant of gait performance, recent studies suggest that muscle degeneration and disuse may exert greater influence than previously understood.[5,12,13] In particular, studies utilizing ultrasound have revealed function-dependent changes in spastic muscles, underscoring the complex relationship between spasticity, structural muscle integrity, and mobility. [12,14] Given these considerations, this pilot study was designed to explore the correlation between muscle tone, muscle echotexture (evaluated through MHS), and walking speed in patients with chronic stroke. Understanding these relationships may enhance clinical assessment and guide more targeted rehabilitation strategies.

METHODS

This research was designed as a pilot study approach with a cross-sectional design, with the primary goal of testing feasibility and identifying trends that may guide future, larger-scale studies. The research was carried out at the outpatient clinic of Physical Medicine and Rehabilitation of Hasan Sadikin Hospital, Bandung, Indonesia. Residents of Physical Medicine and Rehabilitation did physical examinations, spasticity assessment, and functional tests. To minimize variability in MAS scoring, all assessors underwent training under supervision of a senior neurorehabilitation consultant. While inter-rater reliability was not formally testes, calibration sessions were conducted to improve consistency, recognizing that MAS is known for its subjectivity. For the MHS, ultrasound images were analyzed by a single specialist with over 5 years of experience. Although this ensured technical expertise, the absence of blinded re-analysis

represents a limitation. Recent refinements of the Heckmatt Scale using Rasch analysis may further enhance its reliability in future research.

Participant

Eight ambulatory individuals with chronic stroke and spastic hemiplegia were consecutively recruited between December 2024 and May 2025. Eligibility criteria included**:** *(i)* aged ≥18 years, (*ii)* first-ever hemorrhagic or ischemic stroke confirmed by computed tomography (CT) or magnetic resonance imaging (MRI), *(iii)* stroke duration >6 months, *(iv)* presence of spasticity in the gastrocnemius-soleus complex of the affected lower limb, and *(v)* have brunnstrom stage of at least 4. Patients were excluded if they demonstrated inability to follow instructions, unstable medical conditions, history of botulinum toxin injection within three months, neuromuscular or peripheral musculoskeletal disorders, or undergone surgical intervention in the lower limb.

Ultrasound of selected muscle

Muscle echotexture was examined using a Clarius L15 HD wireless ultrasound system (Clarius Mobile Health, Vancouver, Canada) equipped with a high-frequency linear-array transducer operating within a 5 – 15 MHz frequency range (38 mm footprint, maximum depth 7 cm). Imaging focused on the gastrocnemius-soleus complex, including the lower limb (LL) muscles included the soleus (S), lateral gastrocnemius (LG), medial gastrocnemius (MG), and ankle plantar flexors (AP). Both the affected and unaffected sides were evaluated for muscle thickness and echogenicity.

All assessments were conducted by a neurorehabilitation specialist with over 5 years of experience in stroke rehabilitation and musculoskeletal ultrasound, ensuring consistency and reliability in image acquisition and interpretation. Participants were examined in a comfortable supine position, with the lower limb slightly externally rotated and fully supported to maintain relaxation of the support to maintain relaxation of the gastrocnemius-soleus muscle group. Prior to scanning, the skin over the calf was cleaned, and ultrasound gel was applied to facilitate optimal acoustic coupling and to minimize friction. The transducer was positioned transversely at approximately 30% of this distance from the knee crease, corresponding to the proximal third of the muscle belly. Both affected (spastic), and unaffected (contralateral) sides were examined in the same manner. Real-time B-mode images were acquired for each muscle and saved in high-resolution digital format for subsequent offline analysis. Muscle echogenicity was assessed using the Modified Heckmatt Scale (MHS), a four-grade ordinal scale evaluating the degree of echogenicity and visibility of the underlying bone, where: Grade 1 indicates normal muscle echogenicity with clear bone outline, Grade 2 shows increased muscle echogenicity with preserved bone definition, Grade 3 demonstrates marked echogenicity with partially obscured bone structures, and Grade 4 reflects severe echogenicity with complete loss of bone shadow, indicative of advanced fibrotic or fatty infiltration.

Spasticity

Spasticity of gastrocnemius-soleus complex was assessed using the Modified Ashworth Scale (MAS). Raw scores (0, 1, 1 +, 2, 3, 4) were converted into a six-point ordinal scale (0, 1, 2, 3, 4, 5) for statistical purposes.

Functional Assessment

Gait performance was evaluated using walking speed derived from the 6-Minute Walking Distance (6MWD). Participants were instructed to walk in a straight line at their normal pace at a safe and comfortable 12-meter path. The path was marked start and stop with distance 12-meter, and mark per 1-meter. The test was recorded in 6 minutes, the distance that subjects get in meters then divided by 360 second so that gait speed is described as m/s.

Statistical analysis

Descriptive statistics summarized participant characteristics. Paired *t*-tests compared MHS values between paretic and non-paretic sides. Repeated-measures ANOVA was used for muscle thickness comparisons. Non-parametric ANOVA tested differences in MHS scores. Spearman's rank correlation and Pearson correlation coefficients explored associations between MAS, MHS, and gait speed. Linear regression analyses were also conducted. A significance threshold was set at $\alpha = 0.05$.

Ethical approval

This study followed the principles outlined in the Declaration of Helsinki. Ethical approval was granted by the ethical committee of Hasan Sadikin Hospital, Bandung, Indonesia (Approval No. DP.04.03/D.XIV.6.5/493/2025). All participants were

informed of the objectives and procedures of the study and provided written informed consent prior to enrollment. Confidentiality and anonymity of all participant data were strictly maintained throughout the research process.

RESULTS

Participants characteristics

A total of eight individuals with chronic stroke were included, with a mean age 54 (SD ± 6.84) participated in the study. The majority presented with left-sided hemiparesis (75%) and ischemic stroke (75%). Lesions were predominantly both subcortical and cortical (62,5%). Stroke onset was under 12 months in 62,5% of cases. Brunnstrom staging indicated equal distribution between stage 5 and 6 (50% each). Regarding recent interventions, balance-oriented physiotherapy was the most common (37,5%), followed by ESWT to the gastrocsoleus (25%). Other approaches interventions (12,5% each) included home-based walking, occupational therapy for ADLs, and stretching with leg ergometer-based aerobic exercise.

Ultrasonography assessment of spastic muscles

Spearman correlation analysis revealed on table 2 show that a very weak positive correlation between MAS and MHS in the medial gastrocnemius (p = 0.067), a weak correlation in the lateral gastrocnemius (p = 0.269), and a moderate correlation in the soleus (p = 0.417). None of these were statistically significant (p > 0.05).

For gait speed, negative and very weak correlations were found with MHS scores: medial gastrocnemius (p = –0.126), lateral gastrocnemius (p = –0.206), and soleus (–0.169), with no statistical significance observed as stated in table 3. Whereas spearman correlation analysis based on table 3 revealed a moderate correlation between MAS ankle plantarflexors and gait speed (p = 0.132).

According to Table 4, linear regression analysis confirmed these findings. The regression model for predicting MAS from MHS scores (MG, LG, and S) was not statistically significant (R^2 = 0.208, F = 0.350, p = 0.793). Similarly, the model predicting gait speed from MHS was also non-significant (R^2 = 0.062, F = 0.088, p = 0.963). No individual predictor showed statistical significance in either model. Table 5 showed that paired t-tests revealed significant differences in muscle thickness between MHS of the paretic and non-paretic sides in gastrocsoleus muscles. The paretic (affected) side showed greater values in all muscles assessed with MG (p = < 0.001), LG (p = 0.002), and soleus muscle (p = < 0.001). Scatter plot residuals shown on Fig 1 and Fig 2 have no specific pattern, supporting assumptions of normality and homoscedasticity. However, a few high leverage values suggest potential outliers or influential cases. Overall, this pilot study found no significant association between MAS, MHS, and gait speed in chronic stroke patients.

DISCUSSION

This pilot study set out to examine the interplay between spasticity, muscle echotexture, and gait speed in individuals living with chronic stroke. Although weak to moderate correlations were observed between the Modified Ashworth Scale (MAS) and the Modified Heckmatt Scale (MHS), as well as between MHS and gait speed, none of these reached statistical significance. Several insights emerge from these findings that may contribute to a broader understanding of post-stroke neuromuscular adaptations.

Spasticity and muscle degeneration

The limited correlation between MAS and MHS indicates that elevated muscle tone does not necessarily reflect structural muscle alterations. This reinforces earlier reports emphasizing that MAS primarily reflects neural excitability and velocity-dependent resistance, while being less sensitive to chronic tissue remodelling.[2,3,17] Moreover, MAS has limited ability to detect long-term muscular changes such as fibrosis or fatty infiltration, which are better assessed using imaging modality like ultrasound.[4,9] Ultrasound-based echogenicity scoring via MHS offers a window into the chronic morphologic degeneration of muscle tissue, including fiber loss, increased collagen deposition, and sarcopenia changes that are often missed by clinical palpation or passive movement tests.[8] The gastrocsoleus complex is particularly vulnerable to these changes due to its postural function and fiber composition, especially the

soleus, which showed the most consistent pattern in this study. In the present study, the soleus muscle displayed the strongest association between MAS and MHS, consistent with its postural role and susceptibility to chronic spasticity-related remodeling.[8,18]

Asymmetry between the affected and unaffected muscles

Significant difference in echogenicity and thickness between the paretic and non-paretic gastrocnemius-soleus muscles underscore the role of disuse and spasticity in shaping muscle quality. These findings align with prior evidence showing increased echo intensity and reduced bulk in affected muscle post-stroke.[12] Spasticity in the gastrocnemius-soleus complex has been associated with impaired gait performance, notably diminished push-off, and uneven walking patterns.[16] The identified asymmetries support the involvement of both neurological and muscular factors in gait dysfunction, emphasizing the relevance of integrating clinical scales such as the MAS with imaging-based measures such as the MHS for thorough comprehensive profile of motor impairment.[9]

Muscle quality affects gait speed

Gait speed is an integrated functional outcome influenced by muscle strength, coordination, energy efficiency, and spasticity. Our finding of a very weak negative correlation between MHS and gait speed support the hypothesis that higher muscle echogenicity is associated with slower ambulation, though the association did not reach statistical significance. Prior studies have highlighted that stroke-related sarcopenia and chronic disuse may exert greater influence on mobility than hypertonia alone.[6,7,21] This pathophysiological distinction is critical: a patient may exhibit minimal spasticity on MAS yet still suffer from poor gait due to underlying myopathic processes that degrade functional output.[10] Furthermore, recent revisions of the MHS using Rasch analysis have increased its sensitivity and reliability in detecting such muscle degeneration in both clinical and research settings.[11]

Influence of sample characteristics

Several characteristics of the study sample may further explain the absence of significant association between spasticity (MAS), muscle echogenicity (MHS), and gait speed.

Most participants in this study presented with mixed cortical and subcortical lesions. The anatomical distribution introduces heterogeneity in motor impairment profiles. Cortical lesions primarily disrupt motor planning and sensorimotor integration, while subcortical lesions impair direct descending pathways such as the corticospinal tract.[27] In the chronic phase of stroke recovery, neural reorganization and compensatory pathways may emerge differently depending on lesion location. Such variability may lead to inconsistent muscle recruitment and activation patterns across subjects, weakening the expected correlation between muscle tone, structure, and function. Previous neuroimaging studies have confirmed that lesion topography is a significant determinant of both spasticity severity and motor recovery potential.[27] A more homogeneous sample such as patients with surely subcortical infarcts might yield more interpretable and consistent results. Stratification based on lesion location in future studies is recommended to clarify these anatomical-functional relationships.

In this study, all participants were in Brunnstrom stages 5 and 6, reflecting the stage of motor recovery where voluntary movement is reemerging and synergistic patterns are dissolving, relatively high motor recovery and likely contributing to ceiling effect. At this point, many individuals regain functional movement sufficient for walking and the influence of spasticity and muscle degeneration on gait speed may diminish. This “ceiling effect” in motor recovery likely attenuates the variability in functional outcomes across the sample.[28,30] Consequently, correlations between clinical impairments and gait performance become harder to detect statistically. Previous research has indicated that patients in earlier Brunnstrom stages exhibit stronger dependence on neural deficits for ambulation, with greater contributions from tone and weakness.[30] Future studies involving a wider Brunnstrom stage range (e.g., stages 2-6) may reveal more distinct trends and relationships.

All participants fell within a narrow and relatively older adult age range, which may confound results due to age-related neuromuscular adaptations. Sarcopenia, fiber-type shifting, and reduced neuromuscular activation are known to occur naturally with aging and can influence muscle echogenicity, even in the absence of neurological injury.[7,8,12]

Thus, the echogenic changes observed in ultrasound may not be exclusively attributable to post-stroke myopathy, potentially diluting the association between MHS and functional performance. Monjo et

al. demonstrated that stroke survivors exhibit greater echo intensity than age-matched controls, but the overlap with natural aging processes remains significant, particularly beyond age 50.[12] Including a wider age range, or comparing to younger stroke patients, could help isolate stroke-specific effects from baseline age-related degeneration.

All subjects in this study were in the chronic phase of stroke (7 – 24 months post onset), which presents its own implications. In chronic stages, neuromuscular and functional systems often have minimal neuroplasticity. Adaptive changes such as increased contraction, postural compensation, and habitual movement strategies may stabilize or mask the acute dynamics of muscle degeneration and tone alteration.[5,8] Consequently, relationships between spasticity, muscle quality, and mobility become less dynamic and harder to detect statistically. In earlier phases such as subacute stroke these changes are still unfolding and may present with clearer linear associations. Including patients at multiple time points post stroke would allow for a more comprehensive understanding of the temporal evolution of spasticity, muscle integrity, and function.[5,7,8]

Clinical implications

These findings suggest that neither MAS nor MHS, when used in isolation, appears sufficient to fully explain gait performance in chronic stroke. MAS captures neural aspects of spasticity but no long-term muscle degeneration, while MHS provides insight into muscle quality without reflecting motor coordination. Combining these tools in clinical practice could enrich assessment and support more personalized rehabilitation strategies. For example, ultrasound-detected increases in muscle echogenicity may signal the need for early resistance training, neuromuscular electrical stimulation, or interventions targeting sarcopenia.[6,7,10]

From rehabilitation perspective, these results support a paradigm shift from spasticity centric models toward multidimensional approaches that integrate muscle preservation, motor learning, and functionally relevant training.[5,6] For example, interventions targeting sarcopenia and fatty infiltration such as resistance training or neuromuscular electrical stimulation may offer greater functional benefits than ant spasticity treatment alone.[6,7]

The moderate correlation between MAS of the ankle plantar flexors and gait speed found in this study is consistent with findings from Park et al., who identified clinically meaningful MAS thresholds linked with ambulation potential.[15] However, as other studies show, spasticity's impact on gait is often nonlinear and influenced by coexisting motor and sensory impairments.[16]

Strength and Limitations

A key strength of this study is the combined use of clinical, imaging, and functional assessments to explore multidimensional outcomes in chronic stroke. It also contributes to the emerging body of evidence supporting ultrasound as a complementary tool in neurorehabilitation practice.[20] Nonetheless, several limitations warrant discussion. The small sample size limits generalizability and statistical power, and outliers observed in regression residuals may reflect heterogeneity in stroke characteristics, chronicity, or therapy history. Although ultrasound evaluation was performed by an experienced specialist, inherent subjectivity in interpreting MHS scores remains a possible source of bias.[9,11] Moreover, gait speed while widely validated is only one dimension of mobility. Other parameters such as gait symmetry, balance, or endurance may be more sensitive to subtle muscular or neural changes.[23,24,25]

Future directions

Future research should aim to recruit larger and more diverse groups of patients, which would improve both the reliability and the generalizability of the findings. Including individuals in the subacute phase of stroke recovery (1 – 6 months post-onset) could be especially valuable, as this is a critical period when neuromuscular changes and neuroplastic processes are still actively unfolding. It would also be important to analyze outcomes according to Brunnstrom stage, examining patients in stage 3 separately from those in stages 4 and 5, since each stage represents a different level of motor recovery and may influence the relationship between spasticity, muscle degeneration, and function in distinct ways. Finally, broadening outcome measures beyond gait speed—such as incorporating gait symmetry, balance, and endurance—would provide a more comprehensive picture of how muscle tone and echotexture contribute to mobility in stroke survivors.

Conclusion

This pilot investigation did not demonstrate statistically significant associations among spasticity, muscle echogenicity, and gait speed in patients with chronic stroke. Although weak to moderate

correlations were observed, the findings suggest that neither clinical spasticity scales nor sonographic measures of muscle quality, when considered independently, adequately explain gait performance in this population. The relatively preserved functional status and homogeneity of the sample likely contributed to the absence of stronger relationships. These results emphasize the complexity of post stroke motor recovery, where neural tone, muscle degeneration, and compensatory straegies interact in mutifaceted ways. For clinical practice, the study highlights the value of combining sructural and functional assessments rather than relying on a single measure. Future studies with larger and more diverse cohorts, spanning a wider range of Brunnstrom stages and recovery phases, are warranted to clarify the contributions of spasticity and muscle integrity to gait dysfunction. Incorporating advanced assessment tools may also deepen understanding and guide the development of more targeted rehabilitation strategies.

Table 1. Subject Characteristics

	Number of Subjects n (%)
Subjects, *n*	8
Age, years, mean (SD)	54 (6.84)
Hemiparesis, n (%)	
Right hemiparesis	2 (25.0)
Left hemiparesis	6 (75.0)
Stroke, n (%)	
Ischemic stroke	6 (75.0)
Haemorrhagic stroke	2 (25.0)
Lession, n (%)	
Subcortical	2 (25.0)
Cortical	1 (12.5)
Both subcortical and cortical	5 (62.5)
Onset, n (%)	
< 12 months	5 (62.5)
≥ 12 months	3 (37.5)
Brunnstrom stage (LE), n (%)	
Stage 5	4 (50.0)
Stage 6	4 (50.0)
Last 3-month therapy, n (%)	
ESWT a.r gastrocsoleus muscle	2 (25.0)
Home programme and ground walking	1 (12.5)
Occupational therapy (ADL)	1 (12.5)
Physiotherapy balance training	3 (37.5)
Physiotherapy (Stretching and aerobic exercise using leg ergometer)	1 (12.5)

Table 2. Spearman correlation between MHS and MAS

	MAS		Strength of Correlation
	***p*-value**	**Coefficient**	
MHS affected side MG*	0,875	0.067	Very weak correlation
MHS affected side LG*	0,519	0,269	Weak correlation
MHS affected side S*	0,304	0,417	Moderate correlation

Note: *MG: medial gastrocnemius, LG: lateral gastrocnemius, S: soleus

Table 3. Spearman correlation between MHS, MAS, and gait speed

	Gait speed		Strength of Correlation
	***p*-value**	**Coefficient**	
MHS affected side MG	0,766	−0.126	Very weak correlation
MHS affected side LG	0,624	−0.206	Weak correlation
MHS affected side S	0,689	−0.169	Very weak correlation
MAS ankle plantarflexors	0,132	−0.580	Moderate correlation

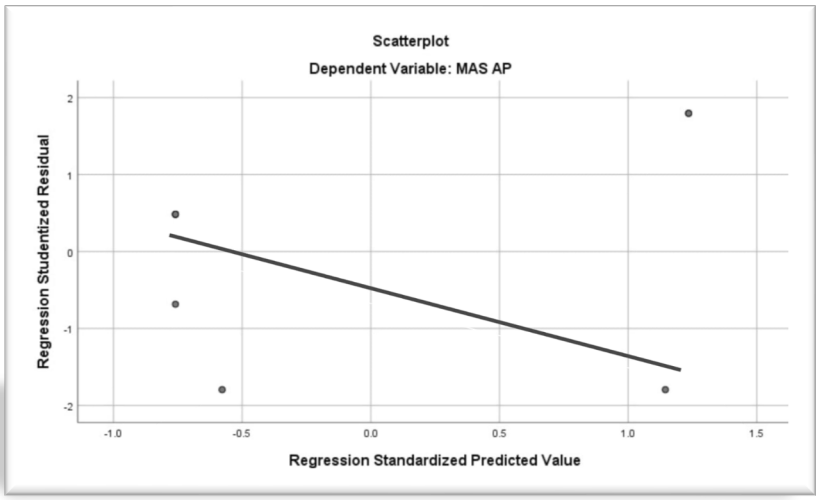
Table 4. Regression linear analysis correlation between MHS, MAS, and gait speed gastrocnemius-soleus muscle**

	MAS				Gait Speed			
	R^2	**P**	***p*-value**	**β**	**R^2**	**P**	***p*-value**	**β**
MHS affected side MG			0,970	0.019			0,907	0.064
MHS affected side LG	0,208	0,793	0,970	−0.026	0.062	0,963	0,709	−0.287
MHS affected side S			0,515	0,470			0,890	0,106

Note : **MHS gastrocnemius-soleus as independent variable

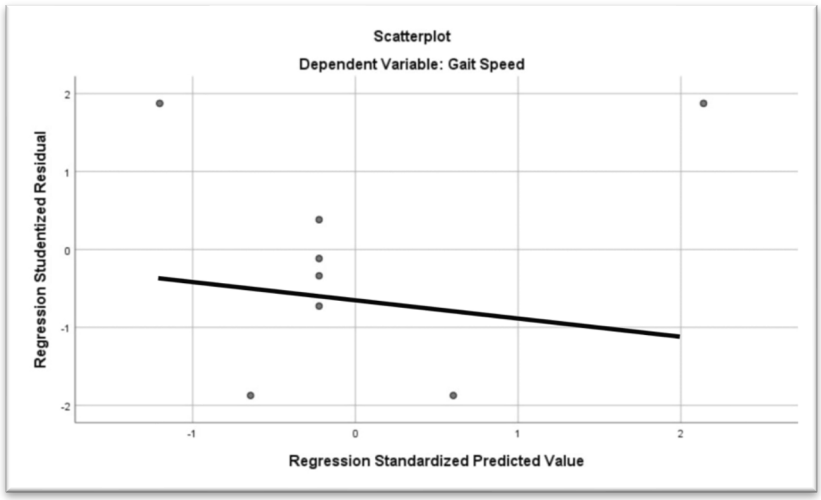
Table 5. Paired T-test comparison between MHS affected and unaffected side gastrocnemius-soleus muscles

Muscles side	***p*-value**
Medial gastrocnemius (MG)	< 0.001
Lateral gastrocnemius (LG)	0.002
Soleus (S)	< 0.001



Note: X = MAS AP, Y = MHS

Fig 1. Relationship between MHS and MAS gastrocnemius-soleus muscle showing a negative linear trend (r = 0.34, p = 0.66).



Note : X = gait speed, Y = MHS

Fig 2. Relationship between MHS and gait speed showing a negative linear trend (r = 0.5, p = 0.56)

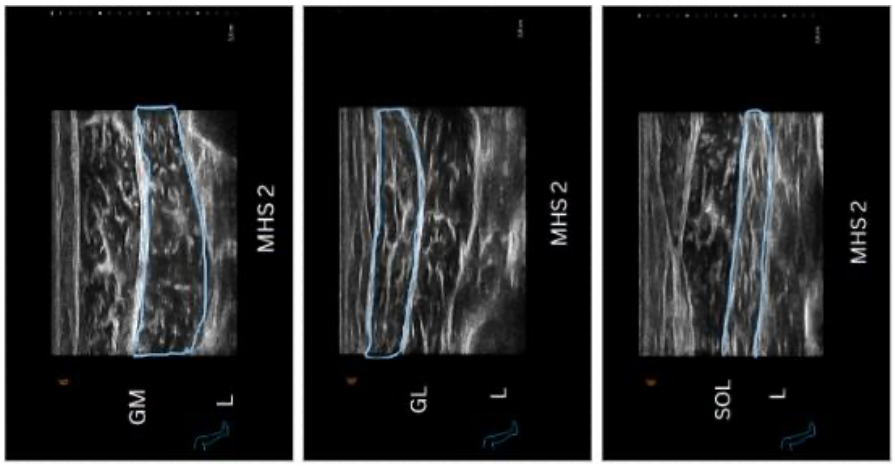


Fig 3. Traced gastrocsoleus muscles using Clarius L15 HD wireless ultrasound device

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