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DRY NEEDLING WITH INTRAMUSCULAR ELECTRICAL STIMULATION IMPROVES UPPER-LIMB IMPAIRMENTS AFTER CHRONIC ISCHEMIC STROKE: A RANDOMIZED CONTROLLED TRIAL

A PUNÇÃO SECA COM ESTIMULAÇÃO ELÉTRICA INTRAMUSCULAR MELHORA AS DEFICIÊNCIAS DOS MEMBROS SUPERIORES APÓS ACIDENTE VASCULAR CEREBRAL ISQUÊMICO CRÔNICO: UM ENSAIO CLÍNICO RANDOMIZADO CONTROLADO

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Abstract
Background: Dry needling is increasingly being used to manage post-stroke upper-limb spasticity, but evidence remains limited for protocols that deliver electrical stimulation through dry-needling. Objective: The study determined whether adding dry needling with intramuscular electrical stimulation to conventional physical therapy improves spasticity and motor outcomes in patients with chronic ischemic stroke. Methods: In this assessor-blinded randomized controlled trial, 64 participants were allocated to DN+ES+CPT or CPT alone for 6 weeks. Spasticity (Modified Ashworth Scale) was the primary outcome. Secondary outcomes were upper-limb motor impairment (Fugl–Meyer Assessment Scale), H-reflex latency, and the Hmax/Mmax ratio.

Resumo
Contexto: O agulhamento seco tem sido cada vez mais utilizado para o tratamento da espasticidade do membro superior pós-AVC, mas as evidências sobre protocolos que aplicam estimulação elétrica por meio do agulhamento seco ainda são limitadas. Objetivo: Este estudo determinou se a adição de agulhamento seco com estimulação elétrica intramuscular à fisioterapia convencional melhora a espasticidade e os resultados motores em pacientes com AVC isquêmico crônico. Métodos: Neste ensaio clínico randomizado e controlado, com avaliação cega, 64 participantes foram alocados para o grupo DN+ES+CPT ou para o grupo CPT isoladamente, durante 6 semanas. A espasticidade (Escala de Ashworth Modificada)

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Assessments were conducted at baseline, week 3, and week 6. Results: By week 6, the DN+ES+CPT group achieved greater motor improvement than CPT (FMAS Δ=+8.95 vs +4.60; p=0.002; d=0.73) and reduction in spasticity (MAS Δ=−1.56 vs −0.80; p<0.001; d=−1.63). Although, Hmax/Mmax ratio decreased more in the intervention arm, but between-group differences were not significant (p=0.090). Latency also improved similarly in both groups (between group p=0.581). Conclusion: Adding dry needling with electrical stimulation to CPT produced superior reductions in spasticity and greater motor recovery than CPT alone in chronic ischemic stroke. The combined approach appears feasible and clinically useful.

Keywords: Stroke. Muscle Hypertonia. Spasticity. Electrical Stimulation Therapy. Dry Needling.

foi o desfecho primário. Os desfechos secundários foram o comprometimento motor do membro superior (Escala de Avaliação de Fugl-Meyer), a latência do reflexo H e a razão Hmax/Mmax. As avaliações foram realizadas no início do estudo, na 3ª semana e na 6ª semana. Resultados: Na 6ª semana, o grupo DN+ES+CPT apresentou maior melhora motora do que o grupo CPT (FMAS Δ=+8,95 vs +4,60; p=0,002; d=0,73) e redução da espasticidade (MAS Δ=−1,56 vs −0,80; p<0,001; d=−1,63). Embora a razão Hmax/Mmax tenha diminuído mais no grupo de intervenção, as diferenças entre os grupos não foram significativas (p=0,090). A latência também apresentou melhora semelhante em ambos os grupos (p=0,581 entre os grupos). Conclusão: A adição de agulhamento seco com estimulação elétrica à CPT produziu reduções superiores na espasticidade e maior recuperação motora do que a CPT isoladamente em pacientes com AVC isquêmico crônico. A abordagem combinada parece viável e clinicamente útil.

Palavras-chave: *AVC. Hipertonia Muscular. Espasticidade. Terapia de Estimulação Elétrica. Agulhamento a Seco.*

1 INTRODUCTION

Stroke remains a major cause of long-term disability worldwide and continues to place a real burden on patients, families, and healthcare systems [1,2]. A large proportion of stroke survivors develop chronic spasticity, typically described as a velocity-dependent increase in muscle tone resulting from upper motor neuron lesion [3]. In practice, spasticity is rarely limited to increased tone alone but it often presents alongside pain, abnormal limb posturing, reduced selective motor control, and difficulty performing everyday tasks. These effects hinders community participation, increase dependence on caregivers, and contribute to higher healthcare utilization [1]. When spasticity is not recognized and managed effectively, secondary complications, including soft tissue shortening, joint contracture, and skin breakdown, may develop over time, compounding impairment and further diminishing quality of life [4]. Accordingly, improving spasticity management remains a central priority in stroke rehabilitation.

Conventional management strategies include therapeutic exercise and task-specific training, stretching and positioning programmes, and adjunctive modalities delivered within routine physical therapy; pharmacologic treatment with agents such as baclofen or tizanidine; focal chemodenervation using botulinum toxin or phenol; and in selected cases, invasive approaches such as intrathecal baclofen therapy [5,6]. Although these approaches may reduce muscle hypertonia and improve comfort, real-world effectiveness is often constrained by limited duration of benefit, dose-limiting adverse effects [e.g., sedation, generalized weakness], and logistical barriers including cost, repeated procedures, and access to specialized services [5,7]. These limitations have increased interest in adjunctive, non-pharmacological interventions that are feasible within outpatient rehabilitation, acceptable to patients, and capable of improving both muscle tone and functional performance.

Dry needling [DN] is a minimally invasive technique in which a fine filiform needle is inserted into hypertonic muscle tissue or myofascial trigger points without injecting medication [8–10]. Although originally used in myofascial pain syndromes, DN has been increasingly applied in neurorehabilitation. Proposed mechanisms include modulation of dysfunctional motor end-plate activity, alteration of nociceptive and proprioceptive afferent input, and a reduction in passive muscle stiffness, sometimes accompanied by local twitch responses [11]. Clinical studies have reported short-term reductions in post-stroke spasticity following DN, but the magnitude of improvement is variable and effects may attenuate over time, particularly in patients with established chronic hypertonia [12]. This suggests that DN alone may be insufficient to induce durable neurophysiological change in some stroke populations.

Neuromuscular electrical stimulation [ES] is another widely used approach in stroke rehabilitation. By activating paretic muscles and/or their antagonists, ES may facilitate reciprocal inhibition, influence spinal reflex excitability, and support motor relearning through repetitive activation and sensory feedback [13,14]. Regular ES sessions have been associated with improvements in spasticity scores and functional outcomes, in part by maintaining neuromuscular activity and mitigating disuse-related weakness [15]. Nevertheless, responses to ES vary, and improvements may be modest or temporary, especially in patients with pronounced or longstanding spasticity [14]. These

observations provide a physiological rationale for combining interventions that target both peripheral muscle properties and central reflex pathways.

A combined strategy using DN together with ES may offer complementary effects. Dry needling may reduce local muscle hyperactivity and alter afferent signaling from the treated muscle, potentially influencing spinal excitability [16]. Electrical stimulation may further modulate reflex circuits and contribute to activity-dependent neuroplasticity through repeated sensorimotor input [17,18]. Delivering ES through or in conjunction with the needled tissue [often described as intramuscular electrical stimulation] may therefore engage both peripheral and central mechanisms potentially producing greater and more sustained improvements than either modality alone.

Early clinical evidence has suggested that electrical stimulation delivered via inserted needles or applied alongside DN may enhance neuromodulatory effects and improve clinical outcomes [19]. Small clinical studies and preliminary reports in chronic stroke populations have described improvements in upper-limb tone and motor performance following DN+ES, but the available literature remains limited by small sample sizes, heterogeneous protocols, and incomplete reporting of neurophysiological correlates [13,20,21]. Importantly, there remains a lack of adequately powered randomized controlled trials evaluating whether DN combined with ES provides clinically meaningful advantages over conventional physical therapy alone, particularly when both clinical measures and neurophysiological indices are assessed in parallel.

To address this gap, we conducted a randomized controlled trial comparing dry needling combined with intramuscular electrical stimulation plus conventional physical therapy [DN+ES+CPT] versus conventional physical therapy alone [CPT] in patients with chronic ischemic stroke. We hypothesized that DN+ES+CPT would produce greater reductions in spasticity, measured using the Modified Ashworth Scale, and more substantial improvements in upper-limb motor function and neurophysiological measures than CPT alone.

2 MATERIALS AND METHODS

2.1 Study design, setting, ethics, and registration

This study was a single-center, parallel-group, assessor-blinded randomized controlled trial conducted at a community rehabilitation center in Lahore, Pakistan, from August 2024 to July 2025. Ethical approval was obtained from the Institutional Review Board of the University of Lahore. The trial was prospectively registered with the Pan African Clinical Trials Registry (PACTR202411566651896). All procedures conformed to the Declaration of Helsinki. Written informed consent was obtained from all participants prior to enrollment. Participant confidentiality was protected by assigning coded identifiers, and only de-identified data were used for analysis. Participants were counseled regarding expected and potential adverse effects, including post-needling soreness, transient bruising, and mild discomfort related to electrical stimulation. Adverse events were actively monitored throughout the intervention period and recorded at each visit.

2.2 Participants: eligibility, recruitment, and screening

Adults aged 35–65 years were eligible if they had a clinically and neuroimaging-confirmed diagnosis of ischemic stroke and were in the chronic phase (≥3 months post-event). Additional eligibility criteria included upper-limb spasticity of at least mild severity (Modified Ashworth Scale [MAS] ≥1) and adequate cognitive function to follow instructions (Mini-Mental State Examination [MMSE] ≥18). Patients were excluded if they had severe cognitive impairment (MMSE <18), active malignancy, uncontrolled cardiovascular disease, major psychiatric illness, open wounds or local infection at the treatment area, known bleeding disorders, or intolerance/hypersensitivity to electrical stimulation. To avoid confounding by recent spasticity interventions, participants were also excluded if they had received botulinum toxin or phenol injection in the preceding months or had recent changes in antispasticity medication, as verified by medical records and participant report. Recruitment occurred through hospital referrals, outpatient clinics,

and community outreach. Eligibility was confirmed through standardized clinical examination and medical record review.

2.3 Sample size determination

A total sample size of 64 participants (32 per group) was planned to detect a between-group difference of 0.6 points on MAS, assuming a standard deviation of 0.8, 80% power, and a two-sided alpha of 0.05. A 10% allowance was incorporated for potential attrition.

$$n = \frac{2\sigma^2(Z_{1-\alpha/2}+Z_{1-\beta})^2}{\Delta^2}, \qquad (1)$$

where:

n is the sample size per group,
$\sigma$is the assumed standard deviation of the primary outcome (MAS),
Δ is the expected between-group mean difference,
$\alpha$is the type-I error (two-sided),
and $1-\beta$ is statistical power. Using $\Delta = 0.6$, $\sigma = 0.8$, $\alpha = 0.05$, and 80% power, a total sample of 64 participants (32 per group) was planned, with allowance for potential attrition.

2.4 Randomization, allocation concealment, and blinding

Participants were randomized in a 1:1 ratio to either (1) dry needling with intramuscular electrical stimulation plus conventional physical therapy (DN+ES+CPT) or (2) conventional physical therapy alone (CPT). The allocation sequence was computer-generated by an independent biostatistician. Allocation was concealed using sequentially numbered, opaque, sealed envelopes prepared in advance. After completion of baseline assessments, a research coordinator not involved in outcome assessment opened the next envelope in sequence and assigned the participant to the indicated group. Due to the nature of the interventions, treating physiotherapists and participants could not be blinded. Outcome assessors remained blinded to group assignment, and participants were

instructed not to discuss treatment details with assessors. Data analysis was performed with group identity masked until primary analyses were finalized.

All interventions were delivered in person on a one-to-one basis in the outpatient neurorehabilitation setting. The treatment period was 6 weeks. Participants allocated to DN+ES+CPT received dry needling with intramuscular electrical stimulation twice weekly for 6 weeks, in addition to conventional physical therapy three times weekly.

Dry needling procedure: Certified physiotherapists used sterile, single-use filiform needles (0.25 mm diameter; 20–40 mm length). Needling targeted spastic forearm flexor muscles commonly involved in the post-stroke upper-limb flexor pattern: flexor carpi radialis (FCR), flexor carpi ulnaris (FCU), flexor digitorum superficialis (FDS), and flexor digitorum profundus (FDP). Needling sites were selected based on palpation of taut bands (firm, rope-like structures within the muscle) and clinical hypertonia, irrespective of pain complaints. Needles were inserted perpendicularly into identified taut bands, with the intent to elicit local twitch responses where present. Needles were retained in situ during electrical stimulation. Intramuscular electrical stimulation parameters: Electrical stimulation was delivered through the inserted needles using a biphasic waveform at 2–10 Hz, with pulse width 0.2–0.3 ms. Intensity was titrated to participant tolerance and adjusted within an approximate range of 1–10 mA to produce a visible muscle twitch without undue discomfort. Stimulation duration was 10–15 minutes per session.

Participants allocated to CPT-only received conventional physical therapy three times weekly for 6 weeks, delivered with the same session length and components used in the DN+ES+CPT group. Each CPT session lasted approximately 45–60 minutes and included: (1) passive stretching using 30-second holds for 3–5 repetitions for relevant upper-limb musculature; (2) progressive strengthening emphasizing antagonists (e.g., wrist extensors) using isometric and concentric exercises with resistance bands or free weights; and (3) task-specific functional training (e.g., grasp-and-release practice, reaching tasks, and fine motor activities such as buttoning). A home exercise program was prescribed to all participants. Attendance at supervised sessions and adherence to the home program were monitored using session logs and participant report.

2.5 Treatment fidelity and adherence monitoring

To maintain consistent treatment delivery, all treating physiotherapists attended a pre-trial orientation that covered the intervention steps and the required documentation. Fidelity was reinforced using standardized session checklists and periodic supervisory review. Any missed visits, departures from the planned procedures, and reasons for non-attendance were documented throughout the trial. No protocol amendments were introduced after the study commenced.

Outcome assessments were completed at three prespecified time points: baseline (pre-intervention), mid-intervention (week 3), and immediately after the intervention period (week 6). All assessments were performed by trained evaluators who were blinded to group allocation.

2.6 Primary outcome

Spasticity was assessed using the Modified Ashworth Scale (MAS). Assessments targeted the affected upper-limb flexor synergy, and analysis, these MAS ratings were summarized into a single composite index of upper-limb flexor tone to reduce multiplicity and to reflect the overall flexor spasticity pattern.

2.7 Secondary outcomes

Motor impairment was assessed using the Fugl–Meyer Assessment for the upper extremity (FMAS). Pain intensity was measured using a visual analogue scale (VAS), where applicable. Segmental spinal reflex excitability was evaluated using H-reflex measures including H-reflex latency and the Hmax/Mmax ratio. All measures were obtained using consistent procedures across time points.

2.8 H-reflex acquisition protocol

H-reflexes were recorded from the flexor carpi ulnaris (FCU) to assess segmental spinal excitability. The participants were seated (elbow ~90°, forearm supinated), with

surface electrodes placed over the FCU belly (active), ulnar styloid (reference), and dorsal forearm (ground) [22,23]. The ulnar nerve stimulated at the medial epicondyle (0.5–1.0 ms pulses, 0.5–1.0 Hz) to get H- and M-wave recruitment and see Hmax and Mmax, confirming H-reflex attenuation at higher intensities due to antidromic collision [24]. Signals were then amplified and band-pass filtered (10–1,000 Hz), latencies, and Hmax/Mmax was calculated with standardized positioning [25,26].

2.9 Statistical analysis

All analyses were conducted using R (v4.2.1). Participant characteristics at baseline were summarized using means±standard deviations for continuous variables and frequencies (percentages) for categorical variables. Baseline between-group comparisons were reported descriptively (independent samples t-tests for continuous variables and chi-square or Fisher's exact tests for categorical variables, as appropriate) and were not used to determine trial validity or to select covariates post hoc.

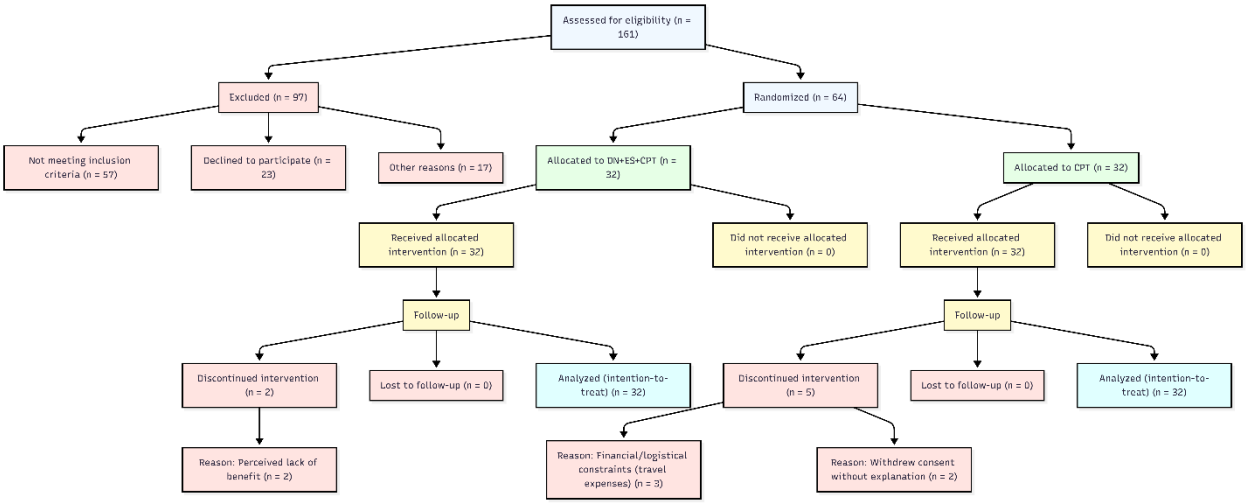
Primary analyses followed the intention-to-treat principle. Missing outcome data were minimal (<5%). Longitudinal analyses used mixed-effects modeling, which provides valid estimates under a missing-at-random assumption by using all available observations without listwise deletion. Outcomes measured over time (baseline, week 3, week 6) were modeled with fixed effects for group, time, and the group×time interaction, and a subject-specific random intercept to account for within-participant correlation. More complex random-effects structures were considered but were not retained when they resulted in convergence instability given the sample size.

Because MAS is an ordinal outcome, spasticity was analyzed using an ordinal mixed-effects model (cumulative link mixed model). Continuous outcomes (FMAS, latency, and Hmax/Mmax ratio) were analyzed using linear mixed-effects models. Prespecified covariates were included to improve precision: baseline outcome value, age, sex, body mass index, affected side, and time since stroke onset. Continuous covariates were grand mean-centered to aid interpretability and reduce multicollinearity. Model fit and assumptions for continuous outcomes were evaluated using residual diagnostics. Influential observations were screened using standard influence diagnostics; no cases warranted exclusion based on prespecified criteria.

Effect sizes were reported for key treatment effects. For mixed-model contrasts, standardized mean differences (Cohen's d) were derived for principal between-group comparisons at post-intervention. Where appropriate, omnibus effect sizes (e.g., partial eta-squared) were reported for overall effects, with 95% confidence intervals estimated using bootstrap resampling (5,000 iterations). Post hoc pairwise comparisons were conducted using estimated marginal means. To control family-wise error for secondary outcomes, Holm adjustment was applied for FMAS, MAS, and Hmax/Mmax ratio comparisons, and Bonferroni adjustment was applied for latency comparisons due to the smaller set of planned contrasts. Statistical significance was set at $p<0.05$ (two-sided).

Figure 1

CONSORT flow diagram of participant recruitment, allocation, follow-up, and analysis.



3 RESULTS

Sixty-four participants were enrolled and randomized to DN+ES+CPT (n=32) or CPT-only (n=32). All participants completed baseline assessments and were included in the intention-to-treat analyses. Baseline demographic and clinical characteristics were comparable between groups, with no clinically meaningful imbalances (Table 1). The mean age was 50±6 years in the DN+ES+CPT group and 51±7 years in the CPT group. Females comprised 22/32 (69%) of the intervention group and 19/32 (59%) of the control group. Body mass index, hand dominance, vascular comorbidities, affected side, stroke

recurrence, pre-stroke physical activity level, and NIHSS severity distribution were similar across groups.

Baseline clinical outcome measures were also comparable (Table 1). Mean MAS was 2.7±0.5 in the DN+ES+CPT group and 2.5±0.6 in the CPT group. Baseline Hmax/Mmax ratio was 0.47±0.17 versus 0.48±0.18, and baseline latency was 15.0±1.7 ms versus 14.4±1.6 ms, respectively. These data support baseline equivalence prior to the intervention period.

Table 1

Baseline Characteristics of Participants by Group (N=64)

Variable	DN+ES+CPT (n=32)	CPT (n=32)	p-value	Test
Demographics				
Age, years	50 ± 6	51 ± 7	0.41	t-test
Height, cm	171 ± 9	171 ± 10	0.89	t-test
Weight, kg	79 ± 14	81 ± 14	0.58	t-test
BMI, kg/m²	27 ± 5	28 ± 5	0.29	t-test
Female sex	22 (69%)	19 (59%)	0.30	χ^2
Right-hand dominant	30 (94%)	29 (91%)	0.47	χ^2
Socioeconomic status				
Low	14 (44%)	18 (56%)	0.16	χ^2
Middle	17 (53%)	11 (34%)		
High	1 (3%)	3 (9%)		
Education level*				
Primary	0 (0%)	14 (44%)	0.74	χ^2
Secondary	9 (28%)	7 (22%)		
Higher	13 (41%)	11 (34%)		
Other/Not reported*	10 (31%)	0 (0%)		
Occupation*				
Employed	7 (22%)	9 (28%)	0.64	χ^2
Unemployed	21 (66%)	19 (59%)		
Other/Not reported*	4 (12%)	4 (13%)		
Clinical characteristics				
Hypertension	13 (41%)	18 (56%)	0.07	χ^2
Diabetes	8 (25%)	8 (25%)	1.00	χ^2
Hyperlipidemia	13 (41%)	13 (41%)	1.00	χ^2
Coronary artery disease	10 (31%)	13 (41%)	0.12	χ^2
Side affected				
Left	18 (56%)	15 (47%)	0.74	χ^2
Right	13 (41%)	17 (53%)		
Bilateral	1 (3%)	0 (0%)		
Stroke recurrence				
First-ever stroke	27 (84%)	25 (78%)	0.34	χ^2
Recurrent stroke	5 (16%)	7 (22%)		
Pre-stroke physical activity				
Sedentary	19 (59%)	15 (47%)	0.71	χ^2
Active	13 (41%)	17 (53%)		
NIHSS severity				

Variable	DN+ES+CPT (n=32)	CPT (n=32)	p-value	Test
Mild	9 (28%)	8 (25%)	0.69	χ^2
Moderate	20 (63%)	21 (66%)		
Severe	3 (9%)	3 (9%)		
Time since stroke (months)†				
>3<6 months	29 (91%)	29 (91%)	1.00	χ^2
>6 months	3 (9%)	3 (9%)		
Baseline outcome measures				
MAS (baseline)	2.7 ± 0.5	2.5 ± 0.6	0.24	t-test
Hmax/Mmax ratio (baseline)	0.47 ± 0.17	0.48 ± 0.18	0.84	t-test
Latency (ms, baseline)	15.0 ± 1.7	14.4 ± 1.6	0.30	t-test

Abbreviations: DN+ES+CPT = dry needling + electrical stimulation + conventional physical therapy; CPT = conventional physical therapy; BMI = body mass index; NIHSS = National Institutes of Health Stroke Scale; MAS = Modified Ashworth Scale.

Upper-limb motor recovery, assessed using the Fugl–Meyer Assessment Scale (FMAS), improved over time in both groups; however, improvements were greater in the DN+ES+CPT group (Table 2). Model-derived estimated marginal means (EMMs) in the DN+ES+CPT group increased from 20.10±1.00 at baseline to 26.45±1.00 at week 3 and 29.05±1.00 at week 6. Corresponding within-group changes were +6.35±1.02 points at week 3 (95% CI: 4.31–8.39) and +8.95±1.02 points at week 6 (95% CI: 6.91–10.99). In the CPT-only group, FMAS increased from 19.90±1.00 at baseline to 23.00±1.00 at week 3 and 24.50±1.00 at week 6, with a total improvement of +4.60±1.02 points at week 6 (95% CI: 2.56–6.64).

Between-group comparisons favored DN+ES+CPT at week 3 (difference=−3.45±1.41; p=0.017; d=0.51) and week 6 (−4.55±1.41; p=0.002; d=0.73). Mixed-effects modeling demonstrated significant effects of group (p=0.044), time (p<0.001), and a group×time interaction (p=0.022), indicating a differential improvement trajectory over the 6-week intervention.

3.1 Spasticity (Modified Ashworth Scale)

Spasticity outcomes, assessed using MAS, improved substantially in the DN+ES+CPT group relative to CPT alone (Table 2). In the DN+ES+CPT group, MAS decreased from 2.66±0.08 at baseline to 1.78±0.08 at week 3 and 1.09±0.08 at week 6, corresponding to a week 6 change of −1.56±0.11 (95% CI: −1.77 to −1.35). In the CPT-only group, MAS decreased from 2.50±0.05 at baseline to 2.44±0.05 at week 3 and 1.70±0.05 at week 6 (Δ week 6=−0.80±0.08; 95% CI: −0.95 to −0.65).

Between-group differences were significant at both week 3 (difference=−0.66±0.09; p<0.001; d=−1.76) and week 6 (−0.61±0.09; p<0.001; d=−1.63), favoring DN+ES+CPT. Model-level testing showed significant effects of group, time, and a strong group×time interaction (all p<0.001), consistent with a larger and sustained reduction in upper-limb flexor tone in the intervention arm.

3.2 Neurophysiological measures (Hmax/Mmax Ratio)

The Hmax/Mmax ratio decreased over time in both groups (Table 2), with a numerically greater reduction observed in the DN+ES+CPT group but without statistically significant between-group differences. In the DN+ES+CPT group, the ratio declined from 0.473±0.031 at baseline to 0.332±0.021 at week 6 (Δ=−0.141±0.038; 95% CI: −0.214 to −0.067). In the CPT-only group, the ratio decreased from 0.482±0.031 at baseline to 0.393±0.028 at week 6 (Δ=−0.089±0.042; 95% CI: −0.171 to −0.007).

Between-group contrasts were not significant at week 3 (p=0.283) or week 6 (p=0.090). Mixed-effects modeling indicated a significant main effect of time (p<0.001) but no significant group×time interaction (p=0.621), suggesting longitudinal improvement in reflex excitability without clear evidence of a differential treatment effect.

3.3 H-reflex latency

Latency increased across the intervention period in both groups (Table 3). At week 6, latency increased by +1.53 ms in the DN+ES+CPT group and by +1.31 ms in the CPT-only group. The between-group difference at week 6 was not significant (difference=+0.22±0.31; p=0.581; d=0.13). Mixed-effects modeling demonstrated a strong main effect of time (p<0.001), with no significant group or interaction effects.

3.4 Pain outcome (VAS)

Pain was prespecified as a secondary outcome but was not included in inferential analyses due to extremely low prevalence and a clear floor effect. At baseline, only 3 of

64 participants (4.7%) reported VAS pain scores >1/10, while the remaining participants reported no pain. Given the lack of variability and limited sensitivity to change, pain outcomes were not analyzed further. This is acknowledged as a feasibility-related deviation from the prespecified secondary outcomes and was decided prior to outcome modeling.

Table 2

Estimated Marginal Means and Within-Group Changes for FMAS, MAS, and Hmax/Mmax Ratio

Measure	Group	Baseline (EMM ± SE, 95% CI)	Week 3	Week 6	Δ Week 3–Baseline	Δ Week 6–Baseline	Δ Week 6–Week 3
FMAS	G1	20.10 ± 1.00 (18.10–22.10)	26.45 ± 1.00 (24.45–28.45)	29.05 ± 1.00 (27.05–31.05)	+6.35 ± 1.02 (4.31–8.39)	+8.95 ± 1.02 (6.91–10.99)	+2.60 ± 1.02 (0.56–4.64)
	G2	19.90 ± 1.00 (17.90–21.90)	23.00 ± 1.00 (21.00–25.00)	24.50 ± 1.00 (22.50–26.50)	+3.10 ± 1.02 (1.06–5.14)	+4.60 ± 1.02 (2.56–6.64)	+1.50 ± 1.02 (–0.54–3.54)
MAS	G1	2.66 ± 0.08 (2.51–2.81)	1.78 ± 0.08 (1.63–1.93)	1.09 ± 0.08 (0.94–1.24)	–0.87 ± 0.11 (–1.09 to –0.66)	–1.56 ± 0.11 (–1.77 to –1.35)	–0.69 ± 0.11 (–0.90 to –0.48)
	G2	2.50 ± 0.05 (2.39–2.61)	2.44 ± 0.05 (2.33–2.54)	1.70 ± 0.05 (1.60–1.81)	–0.06 ± 0.08 (–0.21–0.09)	–0.80 ± 0.08 (–0.95–0.65)	–0.73 ± 0.08 (–0.88–0.59)
Hmax/Mmax	G1	0.473 ± 0.031 (0.412–0.533)	0.378 ± 0.023 (0.334–0.423)	0.332 ± 0.021 (0.290–0.374)	–0.094 ± 0.038 (–0.169–0.019)	–0.141 ± 0.038 (–0.214–0.067)	–0.047 ± 0.031 (–0.108–0.015)
	G2	0.482 ± 0.031 (0.420–0.543)	0.414 ± 0.023 (0.368–0.460)	0.393 ± 0.028 (0.338–0.448)	–0.068 ± 0.039 (–0.144–0.009)	–0.089 ± 0.042 (–0.171–0.007)	–0.021 ± 0.037 (–0.093–0.051)

Table 3

Between-Group Differences in Estimated Marginal Means at Each Time Point

Measure	Time Point	G1 EMM ± SE (95% CI)	G2 EMM ± SE (95% CI)	Difference (G1 – G2) ± SE (95% CI)	p-value	Effect Size (d)
FMAS	Baseline	20.10 ± 1.00 (18.10–22.10)	19.90 ± 1.00 (17.90–21.90)	–0.20 ± 1.41 (–3.00–2.60)	.887	0.02
	Week 3	26.45 ± 1.00 (24.45–28.45)	23.00 ± 1.00 (21.00–25.00)	–3.45 ± 1.41 (–6.25––0.65)	.017	0.51

Measure	Time Point	G1 EMM ± SE (95% CI)	G2 EMM ± SE (95% CI)	Difference (G1 – G2) ± SE (95% CI)	p-value	Effect Size (d)
	Week 6	29.05 ± 1.00 (27.05–31.05)	24.50 ± 1.00 (22.50–26.50)	–4.55 ± 1.41 (–7.35––1.75)	.002	0.73
MAS	Baseline	2.66 ± 0.08 (2.51–2.81)	2.50 ± 0.05 (2.39–2.61)	0.16 ± 0.09 (–0.03–0.34)	.094	0.42
	Week 3	1.78 ± 0.08 (1.63–1.93)	2.44 ± 0.05 (2.33–2.54)	–0.66 ± 0.09 (–0.84––0.47)	<.001	–1.76
	Week 6	1.09 ± 0.08 (0.94–1.24)	1.70 ± 0.05 (1.60–1.81)	–0.61 ± 0.09 (–0.79––0.43)	<.001	–1.63
Hmax/Mmax	Baseline	0.473 ± 0.031 (0.412–0.533)	0.482 ± 0.031 (0.420–0.543)	–0.009 ± 0.044 (–0.095–0.077)	.838	–0.05
	Week 3	0.378 ± 0.023 (0.334–0.423)	0.414 ± 0.023 (0.368–0.460)	–0.035 ± 0.033 (–0.099–0.029)	.283	–0.27
	Week 6	0.332 ± 0.021 (0.290–0.374)	0.393 ± 0.028 (0.338–0.448)	–0.061 ± 0.035 (–0.130–0.008)	.090	–0.43
Latency	Baseline	14.40 ± 0.26 (14.11–14.69)	13.93 ± 0.37 (13.20–14.66))	0.47 ± 0.45 (–0.41–1.35)	.30	0.36
	Week 3	15.26 ± 0.30 (14.67–15.85)	15.08 ± 0.28 (14.52–15.63)	0.18 ± 0.30 (–0.42–0.78)	.637	0.10
	Week 6	15.93 ± 0.31 (15.31–16.54)	15.71 ± 0.29 (15.13–16.30)	0.22 ± 0.31 (–0.38–0.82)	.581	0.13

Table 4

Estimated Marginal Means and Within-Group Changes for Latency

Group	Baseline (EMM ± SE, 95% CI)	Week 3	Week 6	Δ Week 3–Baseline	Δ Week 6–Baseline	Δ Week 6–Week 3
G1	14.40 ± 0.29 (14.11–14.69)	15.26 ± 0.30 (14.67–15.85)	15.93 ± 0.31 (15.31–16.54)	+0.86 ± 0.08 (0.69–0.99)	+1.53 ± 0.08 (1.20–1.50)	+0.67 ± 0.08 (0.52–0.82)
G2	13.93 ± 0.37 (14.11–14.69)	15.08 ± 0.28 (14.52–15.63)	15.71 ± 0.29 (15.13–16.30)	+0.68 ± 0.08 (0.53–0.83)	+1.31 ± 0.08 (1.16–1.46)	+0.63 ± 0.08 (0.47–0.79)

Table 5

Model-Level Statistics for Group, Time, and Group × Time Effects

Measure	Effect	F Value	p-value	Partial η²
FMAS	Group	4.22	.044	0.068
	Time	8.77	<.001	0.131
	Group × Time	3.95	.022	0.064
MAS	Group	19.62	<.001	0.253
	Time	45.41	<.001	0.439
	Group × Time	22.14	<.001	0.292
Hmax/Mmax	Group	2.61	.108	0.014
	Time	9.85	<.001	0.096
	Group × Time	0.48	.621	0.005
Latency	Group	2.20	.138	0.012
	Time	304.63	<.001	0.621
	Group × Time	2.48	.115	0.015

Footnotes

Abbreviations: FMAS = Fugl-Meyer Assessment Scale; MAS = Modified Ashworth Scale; Hmax/Mmax = H-reflex to M-wave ratio; EMM = estimated marginal mean; SE = standard error; CI = confidence interval. Statistical notes: Bolded p-values indicate significance at $p < .05$. All models except latency were adjusted for baseline score, age, sex, BMI, side affected, and stroke duration. Latency analyzed separately due to normal residuals and no baseline imbalances. Missing data <5% handled with full-information maximum likelihood (FIML).

3.5 Adverse events

No serious adverse events were reported during the intervention period. Minor transient soreness and mild bruising at the needling site were reported by four participants in the DN+ES+CPT group and resolved spontaneously. No participant discontinued treatment due to adverse effects.

4 DISCUSSION

This randomized controlled trial investigated whether adding dry needling [DN] with intramuscular electrical stimulation [ES] to conventional physical therapy [CPT] provides additional benefit for upper-limb impairments after chronic ischemic stroke. The principal finding is that DN+ES+CPT produced substantially greater improvements in both motor impairment [FMAS] and spasticity [MAS] than CPT alone over six weeks. Neurophysiological measures showed time-related changes in both groups, with a directional trend toward greater reduction in spinal reflex excitability in the DN+ES+CPT arm, although between-group differences for Hmax/Mmax did not reach statistical

significance. Overall, the results support the clinical utility and feasibility of integrating DN with ES as an adjunct to standard neurorehabilitation in patients with upper-limb hypertonia.

Motor recovery, quantified by FMAS, improved in both groups, consistent with the expected response to structured task-oriented rehabilitation and strengthening in stroke survivors [4,7]. However, gains were larger in the DN+ES+CPT group, with an adjusted improvement of 8.95 points at week 6 and a significant between-group difference at week 6 [p=0.002; d=0.73]. This magnitude of change exceeds improvements reported in some prior DN-based protocols, including studies that used fewer sessions or DN without concurrent stimulation [12,21]. For example, a prior report observed a smaller FMAS gain following DN with stimulation [21], and pooled estimates from DN-focused systematic reviews generally indicate moderate standardized effects on motor outcomes [9,12]. The larger effect observed in the present trial may reflect the combined neuromodulatory and training-related mechanisms of a multimodal regimen delivered repeatedly over six weeks. DN may reduce reflex-mediated resistance and passive stiffness, potentially enabling more effective practice of task-specific movements during CPT, while ES may amplify afferent input and facilitate motor relearning through repeated sensorimotor activation [16–18]. In this model, the added benefit is not solely attributable to symptom reduction [tone], but also to improved capacity for motor practice and recruitment during therapy sessions.

Spasticity outcomes showed an even clearer separation between groups. MAS decreased by 1.56 points in the DN+ES+CPT group compared with 0.80 points with CPT alone [both clinically meaningful; between-group $p<0.001$; $d=-1.63$]. The magnitude and direction are consistent with the literature indicating that DN can reduce post-stroke spasticity and improve range of movement and motor performance [9,12]. Importantly, the effect observed here appears larger than pooled MAS reductions typically reported for DN interventions [12]. A plausible explanation is the combined application of DN and intramuscular ES, which may strengthen inhibitory modulation at the spinal level while simultaneously altering local muscle properties. DN is thought to influence motor endplate activity and peripheral afferent signaling, while ES may further promote reciprocal inhibition and suppress hyperexcitable spinal motor circuits [13–15]. Repeated dosing may also contribute to sustained tone reduction compared with single-session

effects reported in mechanistic studies [27]. Clinically, the reduction in upper-limb flexor tone may have improved the biomechanical environment needed for functional practice [e.g., opening the hand, reaching, and controlled release], thereby contributing to the parallel improvement in FMAS.

Neurophysiologically, the Hmax/Mmax ratio decreased over time in both groups, indicating time-related modulation of spinal excitability. The DN+ES+CPT group demonstrated a numerically greater reduction [week 6 change −0.141] than the CPT group, but the between-group difference did not meet statistical significance [p=0.090]. While this should be interpreted cautiously, the effect sizes at weeks 3 and 6 suggest a possible treatment-related trend. This observation is relevant because objective neurophysiological endpoints can help clarify whether clinical changes in tone are accompanied by measurable changes in segmental spinal circuitry. Prior work has reported immediate or short-term neurophysiological changes after DN or DN with electrical stimulation, but many studies were limited by lack of control conditions, single-session designs, or short follow-up windows [20,21,27]. The present trial extends this evidence by evaluating changes across three time points within a controlled design. Nevertheless, the absence of statistical significance may reflect limited power for electrophysiological endpoints, inter-individual variability in H-reflex measurement post-stroke, and the potential that clinical tone improvements partly reflect peripheral mechanical changes rather than purely spinal inhibitory effects [22–26].

Latency increased over time in both groups without between-group differences. This aligns with the expectation that DN and ES interventions primarily influence excitability and reflex behavior rather than peripheral nerve conduction velocity [27]. The stability of between-group latency contrasts supports the interpretation that the observed clinical gains are unlikely to be explained by changes in peripheral conduction and instead may reflect modulation within spinal or supraspinal pathways combined with improved motor practice and functional training [16–18].

Safety findings were reassuring. No serious adverse events occurred, and minor transient soreness or bruising was self-limited and did not lead to discontinuation. This is consistent with the broader safety literature on DN in neurological populations when performed by appropriately trained clinicians using sterile technique [10]. Although the

present study was not designed to detect rare complications, the observed tolerability supports feasibility in outpatient neurorehabilitation settings.

This study has several strengths: randomized allocation, blinded outcome assessment, standardized intervention dosing, and inclusion of both clinical and neurophysiological outcomes, allowing evaluation of functional change alongside mechanistic signals. Limitations should also be considered. First, the study was conducted at a single center, which may limit generalizability. Second, the follow-up period ended at week 6, so durability of effects beyond the intervention window remains unknown. Third, although protocol fidelity procedures were applied, some inter-therapist variability cannot be excluded. Finally, electrophysiological outcomes may require larger sample sizes and repeated standardized acquisition to detect modest between-group differences. Future trials should incorporate longer-term follow-up, multicenter recruitment, stratified randomization [e.g., by severity or chronicity], and expanded neurophysiological endpoints [e.g., reciprocal inhibition indices or EMG-based reflex measures]. Mechanistic studies combining electrophysiology with neuroimaging or high-density EMG may further clarify whether DN+ES primarily acts through peripheral tissue mechanisms, spinal inhibitory modulation, cortical plasticity, or combined pathways.

From a clinical standpoint, the size of the improvement in both spasticity and motor impairment suggests that DN with intramuscular electrical stimulation can be a useful addition to conventional therapy for people living with chronic stroke. These techniques are generally accessible and relatively inexpensive, and they can be delivered in routine outpatient rehabilitation. The present results indicate that, when integrated into a structured physiotherapy program, DN+ES may translate into meaningful functional gains. Future studies should refine key practical questions—such as the most effective dosing schedule, which muscles should be prioritized, and which patient subgroups are most likely to respond—and should test the approach across different clinical settings to strengthen generalizability.

5 CONCLUSION

Among patients with chronic ischemic stroke, adding dry needling with intramuscular electrical stimulation to conventional physical therapy produced greater

improvements in upper-limb spasticity and motor impairment than conventional therapy alone over six weeks and intervention was well tolerated. Although between-group differences in H-reflex excitability were not statistically significant, the direction of change was consistent with a neuromodulatory effect. Overall, these findings support DN+ES as a feasible adjunct to standard rehabilitation and therefore, a larger, multicenter trials with longer follow-up should be conducted in future to confirm durability and better define underlying mechanisms.

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Authors' Contribution

All authors contributed equally to the development of this article.

Data availability

All datasets relevant to this study's findings are fully available within the article.

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