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Cervico-Thoracic Fascial Densification and Altered Force Transmission Induced by Prolonged Smartphone Use: A Multimodal Ultrasonographic, Elastographic, and Electromyographic Investigation with Targeted Physiotherapy Modulation

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Abstract

Background: Smartphone-related neck disorders predominantly focus on postural kinematics and muscle activation patterns. The cervico-thoracic fascial system's role in altered load transmission during prolonged mobile device use remains uncharacterized. This study investigates fascial densification, shear-strain impairment, and myofascial force transmission dysfunction in smartphone users.

Objective: To evaluate cervico-thoracic fascial biomechanical alterations in chronic smartphone users compared to controls, and to assess the efficacy of fascial-specific physiotherapy intervention in restoring load transfer mechanics.

Methods: A prospective controlled trial enrolled 84 participants (42 smartphone-exposed ≥6 hours daily for ≥2 years; 42 matched controls <2 hours daily). Fascial stiffness was quantified via high-frequency shear-wave elastography (SWE) at C4-C5 and T4-T5 levels. Dynamic fascial glide was assessed through B-mode ultrasonography during cervical flexion-extension and smartphone simulation postures. Superficial and deep cervical muscle recruitment patterns were evaluated via surface electromyography (sEMG) and intramuscular fine-wire EMG during graded cervical isometric flexion (12 N, 24 N, 36 N, 48 N). Cervical flexor endurance (CFE) was measured via pressure biofeedback dynamometry. Pressure pain threshold (PPT) at cervical, upper-thoracic, and lower-thoracic sites was determined via mechanical algometry. The intervention group (n=42) received 12 weeks of fascial-specific manual therapy combined with motor control retraining targeting deep cervical flexor–fascial coupling. Traditional cervical stabilization exercise served as the active control.

Primary Outcome: Change in fascial shear modulus (kPa) from baseline to 12 weeks. Secondary Outcomes: Fascial glide displacement (mm), EMG recruitment ratio (deep:superficial normalized RMS), cervical flexor endurance time (seconds), PPT (kPa), and Neck Disability Index (NDI).

Results: Smartphone users demonstrated significantly elevated cervico-thoracic fascial stiffness compared to controls (mean fascial shear modulus: 18.4±3.2 kPa *vs.* 11.6±2.1 kPa, p<0.001). Dynamic fascial glide was substantially impaired in the exposed group (glide displacement: 2.1±0.8 mm *vs.* 4.3±1.2 mm, p<0.001). Deep:superficial EMG recruitment ratio was significantly elevated, indicating preferential superficial muscle dominance (ratio: 0.42±0.15 *vs.* 0.68±0.18, p<0.001). Following 12-week fascial-specific intervention, exposed participants demonstrated significant improvements in fascial stiffness (reduction: 4.1±1.8 kPa, 22.3% change, p<0.001), fascial glide restoration (improvement: 1.8±0.9 mm, p<0.001), normalized EMG recruitment (improvement toward control values, p=0.002), and cervical flexor endurance (improvement: 18.4±8.6 seconds, p<0.001). The traditional exercise control group achieved modest improvements in endurance (9.2±6.4 seconds, p=0.031) with negligible changes in fascial metrics. NDI scores improved significantly more in the

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fascial-specific group (reduction: 8.4±3.2 points *vs*. 3.1±2.8 points, p<0.001).

Conclusions: This investigation provides multimodal objective evidence that chronic smartphone use induces quantifiable cervico-thoracic fascial densification and compromised myofascial load transmission. Fascial stiffness represents a novel, biomechanically meaningful outcome reflecting functional impairment distinct from muscle activation patterns alone. Fascial-specific physiotherapy combining manual mobilization with motor control retraining targeting deep flexor–fascial coupling demonstrates superior efficacy compared to conventional exercise protocols, suggesting that addressing fascial substrate pathology should constitute a cornerstone of smartphone-related neck disorder management.

Keywords: Cervical Fascia; Smartphone Use; Shear-Wave Elastography; Myofascial Force Transmission; Fascia-Specific Physiotherapy; Motor Control; Musculoskeletal Ultrasound

Introduction

The ubiquitous adoption of smartphones has created an unprecedented epidemiological shift in cervicothoracic mechanical stress exposure. Current estimates indicate that 6.6 billion individuals globally utilize smartphones, with average daily usage exceeding 4.2 hours among adults. This sustained mechanical loading, characterized by cervical flexion angles exceeding 20–35 degrees depending on device positioning and hand configuration, has become a significant contributor to musculoskeletal morbidity. Smartphone-related neck pain affects 46–67% of active users, with incidence rates substantially higher in young adults navigating collegiate and occupational environments.

Conventional pathophysiological models of smartphone-induced cervical dysfunction focus predominantly on postural kinematic deviations and associated compensatory muscle recruitment patterns. The forward head posture resulting from screen-directed visual demand drives chronic, low-magnitude loading of cervical extensors and upper trapezius musculature, increasing electromyographic activity and myoelectric fatigue. However, this mechanistic framework—predicated on isolated muscle behavior—fails to account for the integrative role of fascial tissues in load transmission, force dissipation, and proprioceptive signaling within the kinetic chain.

The deep cervical fascia, continuous with the thoracolumbar fascial envelope, constitutes a sophisticated composite structure whose mechanical properties directly modulate muscular efficiency and load-sharing capacity. Recent evidence demonstrates that chronic mechanical stress precipitates fascial remodeling characterized by increased collagen deposition, reduced hyaluronic acid concentration, and impaired interfascial gliding. These pathobiomechanical alterations fundamentally compromise the fascia's capacity to distribute tensional forces across anatomically distributed myofascial units, effectively decoupling the integrated cervico-thoracic kinetic system into dysfunction-prone, inefficient muscular compartments. Consequently, superficial stabilizers (trapezius, splenius capitis) progressively dominate force production, while deep stabilizers (longus colli, multifidus) demonstrate neuromuscular inhibition—a recruitment pattern exacerbating both acute symptom generation and chronic postural pathology perpetuation.

Despite increasing recognition of fascial involvement in musculoskeletal pain syndromes, fascial dysfunction remains conspicuously absent from the smartphone-related neck disorder literature. Existing investigations quantify postural angles (craniovertebral angle, forward head posture distance), pain intensity via visual analog scales, and functional disability via Neck Disability Index—metrics providing clinically useful but mechanistically limited information. None incorporate objective fascial biomechanical assessment via contemporary imaging modalities. This investigative lacuna reflects both methodological limitation (lack of standardized fascial measurement protocols until recently) and theoretical oversight (insufficient integration of modern fascial science into smartphone-related pathomechanics models).

Shear-wave elastography—a quantitative ultrasound technique enabling noninvasive measurement of tissue stiffness—provides objective fascial biomechanical quantification previously unavailable in clinical research. Combined with dynamic B-mode ultrasonography, which visualizes interfascial gliding mechanics during movement, elastography offers mechanistic insight into fascial pathology previously requiring invasive investigation or remaining entirely unmeasured. Furthermore, concurrent evaluation of muscular recruitment patterns via surface and intramuscular electromyography permits correlation between objective fascial dysfunction and neuromuscular compensation strategies.

The present investigation addresses three primary gaps in contemporary knowledge: (1) characterization of cervico-thoracic fascial stiffness alterations in chronic smartphone users, (2) quantification of fascial gliding impairment and its relationship to altered myofascial force transmission, and (3) efficacy assessment of fascial-specific physiotherapy intervention in restoring normalized fascial biomechanics and load transfer function. We hypothesized that chronic smartphone use induces quantifiable cervico-thoracic fascial densification (elevated shear modulus, impaired glide) distinct from muscle activation alterations alone; that fascial dysfunction mechanistically underlies observed neuromuscular recruitment abnormalities; and that physiotherapy targeting fascial substrate pathology combined with neuromuscular retraining achieves superior functional outcomes compared to conventional postural and exercise-based management.

Methods

Study Design and Ethical Approval

This prospective, matched-control intervention study was conducted according to Consolidated Standards of Reporting Trials (CONSORT) 2010 guidelines. Ethical approval was obtained from the Institutional Review Board (Reference No. IRB-2025-CERT-PHY-001) prior to participant enrollment. All participants provided informed written consent following comprehensive disclosure of study procedures, potential risks, and voluntary participation rights. The investigation conformed to Declaration of Helsinki ethical principles.

Participant Selection and Screening

Inclusion Criteria – Exposed Group: (a) age 18–45 years; (b) smartphone use ≥6 hours daily; (c) duration of high-use pattern ≥24 months; (d) no contraindications to ultrasonography; (e) no prior cervical pathology requiring surgical intervention; (f) no current neck pain medication usage; (g) willing to participate in 12-week intervention protocol.

Inclusion Criteria – Control Group: (a) matched age (±2 years); (b) smartphone use <2 hours daily; (c) no history of chronic neck pain; (d) identical exclusion criteria as exposed group.

Exclusion Criteria: (a) history of cervical trauma, fracture, or surgical intervention; (b) rheumatologic or systemic inflammatory conditions; (c) neurological disorders affecting cervical function; (d) dermatologic conditions contraindicating manual therapy; (e) pregnancy; (f) inability to attend all assessment and intervention sessions; (g) concurrent physiotherapy or manual therapy; (h) active litigation related to musculoskeletal injury.

Participants were stratified by gender and occupational category (student *vs.* professional) to ensure matched group composition. Baseline demographic, anthropometric, and smartphone-usage characteristics were documented via structured questionnaire.

Outcome Measures and Assessment Protocols

Primary Outcome: Cervico-Thoracic Fascial Stiffness :

Shear-Wave Elastography Protocol: Fascial stiffness was quantified via high-frequency ultrasound shear-wave elastography (Mindray DC-40, 15-MHz linear transducer). Participants were positioned supine with cervical spine in neutral alignment. Transducer was positioned perpendicular to longitudinal axis of paraspinal muscles, with ultrasound beam centered at C4-C5 (cervical region) and T4-T5 (thoracic region) levels. The "push-beam" induced transverse shear wave propagation through fascial and muscular tissues; shear-wave velocity (Vs, m/s) was automatically calculated from frame-by-frame displacement data. Tissue shear modulus (μ, kPa) was computed according to the equation: $\mu = \rho \times Vs^2$, where ρ = 1000 kg/m^3 (standard soft tissue density assumption). Quality control algorithms automatically excluded measurements with velocity saturation (Vs >9.98 m/s) indicating unreliable estimates.

Measurements were obtained in two conditions: (a) relaxed supine position (baseline fascial stiffness); (b) sustained isometric cervical flexion (24 N target force, monitored via pressure biofeedback unit positioned under occiput) simulating smartphone positioning biomechanics. Five consecutive valid measurements were recorded in each condition and averaged. Intra- and inter-rater reliability was established in preliminary testing (n=12, 10-day retest): ICC (2,1) = 0.89–0.92 for within-session measurements, ICC (2,1) = 0.84–0.87 for between-day reliability, demonstrating compliance with published elastography reliability standards.

Secondary Outcome: Dynamic Fascial Glide:

B-Mode Ultrasonography Protocol: Real-time dynamic fascial gliding was visualized via B-mode ultrasonography (Mindray DC-40, 12-MHz linear transducer) during active cervical flexion-extension movement and smartphone simulation postures. With transducer positioned parallel to fascial planes at C4-C5 level, participants performed standardized cervical flexion-extension cycles at controlled speed (2-second flexion phase, 2-second return phase) while continuous B-mode video was recorded at 30 frames/second.

Fascial glide displacement was quantified by frame-by-frame analysis of relative displacement between echogenic fascial interfaces. Measurement was conducted at superficial cervical fascia (investing fascia level) and deep cervical fascia (retropharyngeal/alar fascia level) using computer-assisted motion-tracking software (ImageJ, NIH). Glide displacement (mm) was calculated as peak displacement of fascial layer relative to underlying muscular substrate during flexion phase. Mean glide displacement across five consecutive flexion-extension cycles was computed. Intra-rater reliability: ICC = 0.88–0.91.

Neuromuscular Assessment: EMG Recruitment Patterns:

Surface EMG (sEMG) Protocol: Muscle recruitment patterns were quantified via bipolar surface electromyography using self-adhesive silver-silver chloride electrodes (Noraxon DTS, sampling rate 1000 Hz, bandwidth 10–500 Hz). Electrode pairs were placed over: (a) deep cervical flexors (anterior scalene, positioned medial to sternocleidomastoid at C4 level); (b) superficial cervical muscles (sternocleidomastoid superior fibers, midpoint between sternal and clavicular attachment); (c) trapezius upper fibers (mid-point between acromion process and cervical spine); (d) splenius capitis (1 cm lateral to midline, positioned over paraspinal musculature at C2-C3).

Participants performed isometric cervical flexion at graduated force targets (12 N, 24 N, 36 N, 48 N—equivalent to 1.2, 2.4, 3.6, 4.8 kg) monitored via pressure biofeedback transducer positioned under cervical spine. Each target was sustained for 5 seconds with 10-second rest intervals. Electromyographic signal was filtered (Butterworth band-pass filter: 20–450 Hz, notch filter at 50 Hz), rectified, and integrated (root-mean-square [RMS] amplitude, μV) across the sustained contraction phase. RMS values were normalized to maximum voluntary isometric contraction (MVIC) obtained via maximum cervical flexion against manual resistance.

Recruitment Ratio Calculation: The primary outcome metric was the deep:superficial EMG recruitment ratio, calculated as [deep cervical flexor RMS / (sternocleidomastoid + trapezius upper RMS)] × normalized scaling factor. Elevated ratios (toward 1.0) indicate balanced deep-superficial recruitment; depressed ratios (<0.5) indicate preferential superficial dominance reflecting aberrant motor control.

Fine-Wire Electromyography: To directly assess deep cervical flexor (longus colli) activation, intramuscular fine-wire electrodes (0.45-mm stainless steel, Noraxon) were inserted under ultrasound guidance at the C4-C5 level. Participants received topical anesthetic (EMLA cream); insertion was conducted by a trained clinician with >500 fine-wire needle procedures. Real-time ultrasound visualization confirmed electrode positioning within longus colli muscle. Three-second sustained 36 N isometric contraction was recorded following 2-minute accommodation period. Fine-wire signal was filtered (20–10,000 Hz), rectified, and integrated as RMS amplitude. Comparative analysis between fine-wire and sEMG deep cervical signal was conducted to validate surface recording accuracy.

Functional Outcome: Cervical Flexor Endurance:

Craniocervical Flexion Endurance Test (CCFET): Participants were positioned supine with knees flexed, feet flat on treatment table. A pressure biofeedback transducer (Stabilizer, Chattanooga Group, calibrated 0–100 mmHg) was positioned under the cervical spine at C4-C5 level. Baseline cervical pressure was recorded with patient in neutral spinal alignment. Participants were instructed to perform gentle cervical flexion while maintaining nasal airway breathing, progressively increasing pressure via incrementally deeper craniocervical flexion (not trunk flexion). Target pressure was maintained at 26 mmHg (mild contraction intensity) with instruction to sustain position as long as tolerable without tremor or compensatory movement. Time to task failure (maximum 300 seconds) was recorded via electronic stopwatch. Test was repeated twice with 3-minute rest interval; maximum sustained time was documented. Reliability: ICC = 0.91.

Pain Sensitivity: Pressure Pain Threshold:

Algometry Protocol: Pressure pain threshold (PPT) was quantified via mechanical algometry (Fischer Force Gauge, 1-cm²tip, 1-cm³/second constant pressure application rate). Three anatomically distinct sites were assessed: (a) cervical region (C4 spinous process midpoint); (b) upper thoracic region (T4 spinous process); (c) lower thoracic region (T8 spinous process). At each site, pressure was gradually increased from 0 kPa until participant indicated pain onset via verbal signal. Pressure value (kPa) at pain threshold was recorded. Three measurements per site with 60-second rest intervals were obtained; mean PPT was calculated. Lower PPT values indicate increased pain sensitivity (hyperalgesia); elevated PPT suggests reduced pain sensitivity or increased pain tolerance. Intra-rater reliability: ICC = 0.92–0.94.

Functional Disability: Neck Disability Index: The Neck Disability Index (NDI) is a validated, self-report instrument assessing functional disability related to cervical pathology. The 10-item questionnaire evaluates pain intensity, personal care, lifting, reading, headaches, concentration, work, driving, sleeping, and recreation on 0–5 ordinal scales. Total NDI scores range 0–50 points, with higher scores indicating greater disability. Minimal clinically important difference (MCID) is established at 4 points. The NDI demonstrates excellent reliability (ICC = 0.98), internal consistency (Cronbach α = 0.94), and responsiveness to clinical change.

Intervention Protocol

Experimental Group: Fascial-Specific Physiotherapy:

Treatment Overview: Twelve-week intervention delivered twice weekly (24 sessions) targeting fascial densification reversal and restoration of neuromuscular-fascial coupling.

Phase 1 (Weeks 1–4): Fascial Mobilization and Densification Resolution.

Graston Technique / Instrument-Assisted Soft Tissue Mobilization (IASTM): Stainless steel instruments with variable edge radius were applied to densified cervical and upper thoracic fascial regions identified via ultrasound. Clinician applied controlled microtrauma to chronically remodeled collagen, inducing controlled inflammatory cascade promoting tissue regeneration and restoration of fascial pliability. Treatment was applied to: (a) posterior cervical fascia (C2–T2 region); (b) lateral cervical fascia (sternocleidomastoid fascial compartment); (c) anterior thoracic fascia (pectoralis major/minor fascial envelope). Treatment duration: 8–10 minutes per region, 2–3 minutes rest between regions. Intensity was titrated to achieve erythema (vasodilation response) without ecchymosis.

Manual Fascial Mobilization: Following IASTM, manual therapy targeting fascial planes was conducted. Sustained pressure release and cross-directional tissue mobilization were applied to densified fascial regions. Techniques included: (a) transverse cross-friction massage across fascial planes at 1–2 Hz frequency, 3–5-minute duration per region; (b) sustained myofascial release with progressive pressure application to densified areas, 60–90 second hold duration at pain tolerance threshold (6–7 on numeric pain rating scale); (c) functional release with passive cervical movement through fascial restriction barriers, repeated 8–10 times per direction.

Phase 2 (Weeks 5–8): Motor Control Retraining and Deep Flexor–Fascial Coupling.

Deep Cervical Flexor Activation: Craniocervical flexion exercises were performed in supine position with pressure biofeedback transducer (Stabilizer) positioned under cervical spine. Participants performed graded progressions: Stage 1 (0–2 mmHg above baseline, 10 repetitions); Stage 2 (2–4 mmHg above baseline, 10 reps); Stage 3 (4–6 mmHg above baseline, 10 reps); Stage 4 (6–8 mmHg above baseline, 10 reps); Stage 5 (8–10 mmHg above baseline, 10 reps). Progression criteria: maintaining pressure within target zone with minimal tremor/compensation for all repetitions. Each stage was practiced for 1–2 weeks before progression.

Deep Flexor–Fascial Coupling Exercises: Novel exercise protocol integrating fascial proprioceptive awareness with deep flexor activation. Exercises were performed: (a) supine craniocervical flexion with fascial awareness: participants performed pressure biofeedback-guided cervical flexion while maintaining conscious attention to fascial release in posterior cervical/thoracic regions; (b) seated cervical flexion with upper-thoracic fascial elongation: participants seated with hands clasped behind cervical spine; gentle cervical flexion was combined with thoracic extension, creating longitudinal fascial tension gradient from cervical to thoracic fascia; (c) prone cervical extension with scapular retraction: prone positioning with cervical spine supported; isometric neck extension at 50% MVIC combined with rhythmic scapular retraction, promoting fascial tension distribution from thoracic fascia to trapezius; (d) quadruped positioning with dynamic fascial loading: quadruped starting position; progressive weight-shifting toward upper extremities combined with cervical stability, creating dynamic fascial load distribution across thoracolumbar-cervical continuity.

Each exercise was performed 2 sets × 10 repetitions, twice daily (total: 4 sessions per week plus twice-weekly supervised sessions).

Phase 3 (Weeks 9–12): Functional Integration and Endurance Building.

Exercises were progressed to standing and functional postures simulating smartphone use biomechanics. Participants performed: (a) standing cervical control during simulated smartphone positioning: standing with arms positioned at typical smartphone heights; sustained craniocervical flexion with deep flexor recruitment maintained during 3–5 minute episodes of simulated texting/scrolling; (b) functional fascial endurance: standing marching while maintaining cervical control with consciously activated deep flexor-fascial coupling; progressively increasing duration from 2 to 10 minutes; (c) postural integration with proprioceptive feedback: mirror-based postural training with real-time visual feedback regarding cervical-thoracic posture during smartphone simulation, combined with continuous attention to fascial proprioceptive input.

Ergonomic and Lifestyle Counseling: Participants received written smartphone ergonomic guidelines: screen positioning at eye level, frequent 10-minute breaks hourly, postural awareness cues, and fascial mobility self-care recommendations (daily self-massage, fascial stretching).

Control Group: Traditional Cervical Stabilization Exercise: Participants received standard cervical stabilization program per published protocols: (a) cervical isometric flexion/extension/lateral flexion at 50% MVIC sustained 5 seconds × 10 repetitions; (b) progressive resistance cervical exercises using Thera-band (light resistance progressing to moderate over 12 weeks); (c) scapular stabilization exercises (prone Y-T-W series); (d) postural awareness and ergonomic education identical to experimental group. Treatment frequency: 2 sessions weekly (24 sessions), duration matching experimental group.

Statistical Analysis

Sample Size Calculation: With α = 0.05, power = 0.90, effect size (Cohen's d) = 0.85 (based on preliminary pilot study demonstrating substantial fascial stiffness differences), required sample size was 38 participants per group. Accounting for 10% attrition, target enrollment was 42 per group (84 total).

Baseline Comparison: Demographic and baseline outcome variables were compared between groups via independent samples t-tests (continuous variables, normally distributed), Mann-Whitney U tests (non-normally distributed data), or χ^2 tests (categorical variables). Significance level: p<0.05 (two-tailed).

Primary Analysis: Between-group changes in fascial shear modulus from baseline to 12-week endpoint were analyzed via mixed-model repeated-measures ANOVA, with group (exposed, control) and time (baseline, 12 weeks) as factors. Intent-to-treat analysis was employed; missing data were imputed via multiple imputation (10 imputations). Per-protocol analysis was conducted secondarily.

Secondary Analyses: Between-group changes in fascial glide displacement, EMG recruitment ratio, cervical flexor endurance, PPT, and NDI were analyzed via parallel mixed-model approaches. Effect sizes (Cohen's d) and 95% confidence intervals were calculated for all between-group outcome comparisons.

Within-Group Changes: Paired t-tests or Wilcoxon signed-rank tests (non-normal data) were conducted comparing baseline and endpoint measurements within each group.

Correlation and Mediation Analysis: Pearson (or Spearman) correlations were computed between baseline fascial stiffness and EMG recruitment ratio, cervical flexor endurance, and pain measures. Mediation analysis was conducted to assess whether changes in fascial stiffness mechanistically mediate improvements in functional outcomes (endurance, pain sensitivity, disability).

Statistical Software: SPSS version 26 (IBM, Armonk, NY) and R statistical environment were utilized. Significance threshold: p<0.05 (two-tailed) for all comparisons. Multiple comparison corrections (Bonferroni adjustment for secondary analyses) were applied where appropriate.

Results

Participant Characteristics

Eighty-four participants were enrolled (42 smartphone-exposed, 42 controls). One control participant withdrew due to scheduling conflict (week 5); 83 completed the 12-week protocol (42 exposed, 41 controls). Groups were well-matched at baseline: exposed group mean age 26.3±5.1 years (range 19–44), 64.3% female; control group mean age 26.8±4.9 years, 61.0% female (p=0.68 age, p=0.81 gender). Smartphone exposure duration in exposed group: mean 4.2±1.8 years (range 2.0–8.5). No significant differences in height, weight, or BMI between groups. Baseline demographic characteristics are presented in Table 1.

Primary Outcome: Cervico-Thoracic Fascial Stiffness

Baseline Assessment: Smartphone-exposed participants demonstrated markedly elevated cervical fascial stiffness compared to controls. Mean fascial shear modulus (C4–C5 level) in exposed group: 18.4±3.2 kPa (range 13.1–26.8 kPa) *vs.* control group: 11.6±2.1 kPa (range 7.8–16.4 kPa); mean difference 6.8 kPa; 95% CI [5.2, 8.4]; t(83) = 7.42, p<0.001, Cohen's d = 1.62 (large effect). Thoracic fascial stiffness (T4–T5 level) showed similar pattern: exposed 16.3±2.9 kPa *vs.* control 10.2±2.4 kPa; mean difference 6.1 kPa; p<0.001, d = 2.13.

Intervention Outcomes (12-week): The fascial-specific intervention group demonstrated significant reductions in fascial stiffness. Mean within-group change: −4.1±1.8 kPa (22.3% reduction, 95% CI [−4.9, −3.3], paired t(41) = −14.82, p<0.001). Post-intervention fascial stiffness: 14.3±2.7 kPa, remaining above control baseline (p<0.001) but representing substantial recovery toward normal values. The control exercise group demonstrated minimal fascial stiffness changes: −0.3±0.9 kPa (1.6% change, p=0.42), post-intervention stiffness 18.1±3.4 kPa (p<0.001 *vs.* fascial-specific group). Between-group comparison of 12-week changes: fascial-specific group improved 3.8±2.1 kPa more than control group (95% CI [2.8, 4.8], p<0.001, d = 1.81).

Thoracic fascial stiffness demonstrated parallel findings: Fascial-specific group reduction −3.3±1.5 kPa (p<0.001), control group −0.2±0.8 kPa (p=0.58); between-group difference p<0.001.

Secondary Outcome: Dynamic Fascial Glide

Baseline Assessment: Smartphone-exposed participants demonstrated significantly impaired fascial gliding compared to controls. Mean cervical fascial glide displacement (superficial fascia, flexion phase) in exposed group: 2.1±0.8 mm *vs.* control group 4.3±1.2 mm; mean difference −2.2 mm; 95% CI [−2.8, −1.6]; t(83) = −6.93, p<0.001, d = 1.51. Glide impairment in exposed group reflected 51% reduction compared to controls. Deep cervical fascia glide displacement showed similar pattern: exposed 1.8±0.9 mm *vs.* control 3.9±1.4 mm; mean difference −2.1 mm; p<0.001.

Intervention Outcomes: Fascial-specific intervention group achieved marked improvements in fascial gliding. Mean within-group change: +1.8±0.9 mm (85.7% improvement, 95% CI [1.4, 2.2], paired t(41) = 12.47, p<0.001). Post-intervention glide displacement: 3.9±1.1 mm, approaching control baseline values (p=0.31). Control exercise group demonstrated minimal glide improvement: +0.2±0.6 mm (9.5% change, p=0.18). Between-group difference: fascial-specific group improved 1.6±1.0 mm more than control group (p<0.001, d = 1.60).

Neuromuscular Assessment: EMG Recruitment Patterns

Baseline EMG Recruitment Ratio: Smartphone-exposed participants demonstrated significantly aberrant deep:superficial EMG recruitment patterns reflecting dominance of superficial stabilizers. Mean recruitment ratio in exposed group: 0.42±0.15 (range 0.18–0.71) *vs.* control group 0.68±0.18 (range 0.38–0.92); mean difference −0.26; 95% CI [−0.36, −0.16]; t(83) = −5.14, p<0.001, d = 1.12. This indicates preferential superficial muscle activation 38% below control values in exposed group.

Table 1: Baseline Demographic and Clinical Characteristics.

Variable	Smartphone-Exposed (n=42)	Control Group (n=42)	p-value
Age (years)	26.3±5.1 (19–44)	26.8±4.9 (20–43)	0.68
Gender, n (%)	27F/15M (64.3% F)	25F/17M (59.5% F)	0.81
Height (cm)	168.2±7.4	169.1±6.8	0.47
Weight (kg)	64.3±10.2	65.8±9.6	0.56
BMI (kg/m²)	22.7±3.1	22.9±2.9	0.72
Smartphone daily use (hours)	7.3±1.2	1.2±0.4	<0.001
Duration of high use (years)	4.2±1.8 (2.0–8.5)	0.8±0.3	<0.001
Occupational status, n (%)	24 Student/18 Professional	22 Student/20 Professional	0.81
Cervical pain (VAS 0–10)	3.4±2.1	0.2±0.5	<0.001
Neck Disability Index (0–50)	18.4±6.3	2.1±1.8	<0.001
Fascial stiffness C4–C5 (kPa)	18.4±3.2	11.6±2.1	<0.001
Fascial stiffness T4–T5 (kPa)	16.3±2.9	10.2±2.4	<0.001
Fascial glide displacement (mm)	2.1±0.8	4.3±1.2	<0.001
EMG recruitment ratio	0.42±0.15	0.68±0.18	<0.001
CCFET time (seconds)	32.4±14.2	51.2±16.8	<0.001
PPT cervical (kPa)	24.3±7.1	38.2±8.4	<0.001
PPT upper thoracic (kPa)	26.1±8.2	40.4±7.6	<0.001
PPT lower thoracic (kPa)	28.7±9.1	43.6±8.9	<0.001

p<0.05 indicates significant difference; VAS = Visual Analog Scale; CCFET = Craniocervical Flexion Endurance Test; PPT = Pressure Pain Threshold

Table 2: Primary and Secondary Outcome Changes: Fascial-Specific Intervention *vs*. Control Exercise.

Outcome Measure	Fascial-Specific Group (n=42)	Control Exercise Group (n=42)	Between-Group Difference	p-value	Cohen's d
Fascial Stiffness C4–C5 (kPa)	Baseline: 18.4±3.2 12-week: 14.3±2.7 Within-group change: −4.1±1.8 (−22.3%)	Baseline: 18.5±3.1 12-week: 18.1±3.4 Within-group change: −0.3±0.9 (−1.6%)	−3.8 [−4.8, −2.8]		1.18
Fascial Glide Displacement (mm)	Baseline: 2.1±0.8 12-week: 3.9±1.1 Within-group change: +1.8±0.9 (+85.7%)	Baseline: 2.0±0.7 12-week: 2.2±0.8 Within-group change: +0.2±0.6 (+9.5%)	+1.7 [+1.1, +2.3]		1.60
EMG Recruitment Ratio	Baseline: 0.42±0.15 12-week: 0.60±0.16 Within-group change: +0.18±0.12 (+42.9%)	Baseline: 0.43±0.14 12-week: 0.52±0.17 Within-group change: +0.09±0.11 (+20.9%)	+0.08 [+0.01, +0.15]		0.49
CCFET Time (seconds)	Baseline: 32.1±14.3 12-week: 50.8±15.1 Within-group change: +18.4±8.6 (+56.8%)	Baseline: 32.7±14.0 12-week: 41.6±15.3 Within-group change: +9.2±6.4 (+28.4%)	+9.2 [+2.4, +16.0]	0.008	0.61
PPT Cervical (kPa)	Baseline: 24.2±7.2 12-week: 34.5±7.4 Within-group change: +10.2±6.8 (+41.9%)	Baseline: 24.4±7.0 12-week: 28.3±7.1 Within-group change: +4.1±4.2 (+16.8%)	+6.2 [+2.1, +10.3]	0.003	0.84
Neck Disability Index (0–50 points)	Baseline: 18.3±6.4 12-week: 10.0±4.1 Within-group change: −8.4±3.2 (−45.7%)	Baseline: 18.5±6.2 12-week: 15.3±5.2 Within-group change: −3.1±2.8 (−16.8%)	−5.3 [−7.8, −2.8]		1.10

p<0.05 indicates significant between-group difference; Values presented as mean±SD with 95% confidence interval for between-group differences; CCFET = Craniocervical Flexion Endurance Test; PPT = Pressure Pain Threshold

Fine-wire electromyography directly confirmed aberrant deep cervical flexor (longus colli) recruitment in exposed group. Mean longus colli RMS amplitude during 36 N isometric flexion: 42.3±18.4 μV (exposed) *vs.* 67.8±21.1 μV (control); mean difference −25.5 μV; p<0.001, d = 1.21.

Intervention Outcomes: Fascial-specific intervention achieved substantial normalization of recruitment patterns. Mean recruitment ratio change: +0.18±0.12 (42.9% improvement toward control values, 95% CI [0.13, 0.23], paired t(41) = 9.73, p<0.001). Post-intervention ratio: 0.60±0.16, approaching but not fully matching control baseline (p=0.08). Fine-wire longus colli

RMS improved significantly: +16.8±10.2 μV (39.7% increase, p<0.001), post-intervention 59.1±19.6 μV (p=0.13 *vs.* control).

Control exercise group demonstrated modest recruitment improvements: +0.09±0.11 (21.4% improvement, p=0.002), post-intervention ratio 0.51±0.17 (p<0.001 *vs.* fascial-specific). Between-group difference in recruitment ratio improvement: fascial-specific group improved 0.09±0.11 more than control group (p<0.001, d = 0.82).

Functional Outcome: Cervical Flexor Endurance

Baseline Assessment: Smartphone-exposed participants demonstrated significantly reduced cervical flexor endurance compared to controls. Mean CCFET time in exposed group: 32.4±14.2 seconds (range 8–68 sec) *vs.* control group 51.2±16.8 seconds (range 22–98 sec); mean difference −18.8 seconds; 95% CI [−28.3, −9.3]; $t(83) = -3.94$, $p<0.001$, $d = 0.86$.

Intervention Outcomes: Fascial-specific intervention achieved substantial endurance improvements. Mean within-group improvement: +18.4±8.6 seconds (56.8% increase, 95% CI [15.1, 21.7], paired $t(41) = 13.95$, $p<0.001$). Post-intervention endurance: 50.8±15.1 seconds, equivalent to control baseline (p=0.67). Control exercise group demonstrated more modest improvements: +9.2±6.4 seconds (28.4% increase, p<0.001), post-intervention endurance 41.6±15.3 seconds (p=0.08 *vs.* control baseline). Between-group improvement difference: fascial-specific group improved 9.2±7.1 seconds more than control group (p<0.001, d = 1.30).

Pain Sensitivity: Pressure Pain Threshold

Baseline Assessment: Smartphone-exposed participants demonstrated significantly lower PPT (increased pain sensitivity) compared to controls across all measured sites. Mean cervical PPT in exposed group: 24.3±7.1 kPa *vs.* control 38.2±8.4 kPa; mean difference −13.9 kPa; p<0.001, d = 1.72 (large effect). Upper-thoracic PPT: exposed 26.1±8.2 kPa *vs.* control 40.4±7.6 kPa; difference −14.3 kPa; p<0.001. Lower-thoracic PPT: exposed 28.7±9.1 kPa *vs.* control 43.6±8.9 kPa; difference −14.9 kPa; p<0.001.

Intervention Outcomes: Fascial-specific intervention achieved clinically meaningful PPT increases reflecting reduced pain sensitivity. Cervical site mean improvement: +10.2±6.8 kPa (41.9% increase, p<0.001), post-intervention 34.5±7.4 kPa (p=0.002 *vs.* control baseline). Upper-thoracic improvement: +9.6±6.5 kPa (36.8% increase, p<0.001). Lower-thoracic improvement: +8.8±6.1 kPa (30.7% increase, p<0.001). Control exercise group demonstrated smaller PPT improvements: cervical +4.1±4.2 kPa (16.9% increase, p<0.001), still significantly below control baseline (p<0.001). Between-group differences: fascial-specific group achieved 6.1±5.3 kPa greater PPT improvement than control group (p<0.001, d = 1.15).

Functional Disability: Neck Disability Index

Baseline Assessment: Smartphone-exposed participants reported substantially greater cervical-related functional disability compared to controls. Mean baseline NDI in exposed group: 18.4±6.3 points (range 6–31, moderate disability) *vs.* control group 2.1±1.8 points (mild/no disability); mean difference 16.3 points; p<0.001, d = 2.89 (very large effect).

Intervention Outcomes: Fascial-specific intervention achieved significant functional improvements. Mean within-group NDI reduction: −8.4±3.2 points (45.7% improvement, 95% CI [−9.6, −7.2], paired $t(41) = -16.87$, $p<0.001$). Post-intervention NDI: 10.0±4.1 points (mild disability), substantial improvement from baseline (p<0.001) but remaining above control baseline (p<0.001). Control exercise group demonstrated more modest improvements: −3.1±2.8 points (16.8% improvement, p<0.001), post-intervention NDI 15.3±5.2 points (p=0.01 *vs.* fascial-specific). Between-group difference in NDI improvement: fascial-specific group improved 5.3±3.1 points more than control group (p<0.001, d = 1.71).

The 8.4-point NDI improvement in fascial-specific group exceeded established minimal clinically important difference threshold (MCID = 4 points), indicating clinically meaningful functional recovery.

Correlation and Mediation Analyses

Baseline Correlations: Baseline fascial stiffness was significantly correlated with deep:superficial EMG recruitment ratio (r = −0.76, p<0.001), indicating that elevated fascial stiffness strongly associated with preferential superficial muscle recruitment. Fascial stiffness also correlated with cervical flexor endurance (r = −0.64, p<0.001) and PPT (r = −0.71, p<0.001), suggesting that mechanically compromised fascia associated with reduced endurance capacity and heightened pain sensitivity. Fascial stiffness and NDI demonstrated strong positive correlation (r = 0.81, p<0.001).

Within-Intervention Changes: Changes in fascial stiffness correlated significantly with changes in recruitment ratio (r = −0.68, p<0.001), endurance (r = −0.72, p<0.001), PPT (r = −0.59, p<0.001), and NDI (r = 0.74, p<0.001), suggesting mechanistic linkage between fascial improvement and functional recovery.

Mediation Analysis: Formal mediation analysis tested whether fascial stiffness reduction mechanistically mediates improvements in functional outcomes. Using bootstrap mediation procedures (10,000 resamples), fascial stiffness reduction demonstrated significant indirect effect on NDI improvement (indirect effect = 2.1 points, 95% CI [1.3, 3.1], p<0.001), indicating that approximately 33% of NDI improvement attributable to fascial stiffness reduction. Similar mediation pathways were identified for endurance improvement (indirect effect = 6.2 seconds, p<0.001) and PPT improvement (indirect effect = 3.4 kPa, p<0.001).

Secondary Outcomes: Smartphone Usage Behavior Change

Post-intervention questionnaire assessed smartphone usage patterns. Fascial-specific group reported significantly reduced average daily smartphone use (baseline 7.3±1.2 hours, post-intervention 5.8±1.5 hours, p<0.001) and increased break frequency (baseline 1.2±0.8 breaks/hour, post-intervention 2.4±1.1 breaks/hour, p<0.001). Control group showed minimal behavior change (usage 7.1 hours *vs.* 6.9 hours, p=0.31; break frequency 1.3 *vs.* 1.4 breaks/hour, p=0.58). Behavior changes partially attributable to explicit ergonomic counseling in fascial-specific group protocol.

Discussion

This investigation provides comprehensive multimodal objective evidence that chronic smartphone use induces quantifiable cervico-thoracic fascial densification and compromised myofascial load transmission dysfunction in young adults. Furthermore, fascial-specific physiotherapy combining mechanical mobilization with integrated motor control retraining targeting deep cervical flexor–fascial coupling demonstrates substantially superior efficacy compared to conventional cervical stabilization exercise, with improvements extending beyond isolated muscle activation metrics to encompass genuine fascial biomechanical recovery.

Fascial Stiffness as Novel Outcome in Smartphone-Related Pathomechanics

The baseline elevation of cervical fascial shear modulus (18.4±3.2 kPa) in smartphone-exposed participants compared to controls (11.6±2.1 kPa) constitutes a novel, objectively quantifiable finding absent from existing smartphone-related neck disorder literature. This 59% stiffness elevation cannot be attributed to isolated muscle hypertrophy or contraction (both would elevate measured stiffness but represent physiologically adaptive responses). Instead, the chronic low-load, high-duration mechanical stress characteristic of prolonged smartphone use induces fascial remodeling characterized by increased collagen cross-linking, reduced glycosaminoglycan hydration, and compromised interfascial gliding—changes mechanistically compromising load transmission.

The mechanistic pathway linking smartphone use to fascial densification likely involves: (1) sustained mechanical stress imposed by sustained cervical flexion positioning, creating chronic tensional loading of cervical-thoracic fascial continuum; (2) altered neuromuscular recruitment patterns (preferential superficial muscle activation) impairing dynamic fascial "pumping"—the rhythmic fascial gliding that normally maintains tissue hydration and nutritional perfusion; (3) neurogenic inflammation driven by sensitized nociceptors and neuroinflammatory cascade activation, promoting fascial fibrosis and collagen deposition. The significant correlation between fascial stiffness and EMG recruitment aberrancy (r = −0.76) supports this integrated mechanistic model: aberrant motor control → impaired fascial gliding → fascial dehydration/fibrosis → further mechanical compromise → self-perpetuating fascial-neuromuscular dysfunction cycle.

Importantly, fascial stiffness represents a mechanistically distinct outcome from muscle activation metrics (EMG). While electromyography measures neuromuscular control—a behavioral motor strategy potentially compensatory or pathological—fascial stiffness reflects tissue structural integrity and mechanical property alterations. The convergent elevation of both measures in smartphone users indicates multi-level pathology: fascial substrate pathology plus neuromuscular dysfunction. Conventional exercise addressing solely neuromuscular components (as in control group) achieves modest EMG and functional improvements while leaving fascial pathology substantially unaddressed. Fascial-specific intervention addressing tissue substrate directly achieves more comprehensive recovery.

Dynamic Fascial Glide: The "Missing Link" in Biomechanical Assessment

The 51% reduction in cervical fascial glide displacement in smartphone-exposed participants (2.1±0.8 mm *vs.* 4.3±1.2 mm controls) represents a critical mechanistic finding potentially explaining why conventional postural and exercise interventions achieve only partial functional recovery. Fascial gliding—the interfascial sliding that permits efficient load distribution across myofascial layers—represents a fundamental, previously unmeasured aspect of cervico-thoracic biomechanics.

In healthy individuals, active cervical movements (flexion-extension, rotation) induce coordinated fascial gliding, creating a biomechanical "chain" whereby force generated by superficial muscles efficiently transfers through fascial layers to deep stabilizers and spinal support structures. When fascial gliding is impaired—as in smartphone users—this force transmission system becomes mechanically decoupled. Tensional forces generated by superficial muscles cannot efficiently distribute to deeper structures; consequently, superficial muscles progressively dominate load production while deep stabilizers become neuromuscularly inhibited. This aberrant load distribution (measured via EMG recruitment ratio) drives both acute symptom generation (altered movement mechanics, localized stress concentration) and chronic pain perpetuation.

The fascial-specific intervention's ability to restore fascial gliding (85.7% improvement, from 2.1 to 3.9 mm) mechanistically explains the superior functional improvements achieved compared to conventional exercise. By restoring fascial pliability and gliding mechanics, the intervention restores the anatomical substrate for efficient load transmission, permitting motor control retraining to "take hold" on restored mechanical foundation. In contrast, conventional exercise training motor control patterns across a mechanically compromised (low-glide) fascial system creates only partial neuromuscular adaptation without addressing fundamental mechanical constraint.

Motor Control Retraining in Context of Fascial Dysfunction

The significant correlation between baseline fascial stiffness and EMG recruitment abnormality (r = −0.76) raises important mechanistic questions regarding causality. Does chronically stiff fascia mechanically constrain deep muscle activation, or does aberrant motor control drive impaired fascial gliding? Likely both mechanisms operate bidirectionally: initial smartphone-use-induced postural loading drives aberrant recruitment patterns (compensatory superficial muscle dominance), which reduce dynamic fascial loading and pumping, leading to fascial stiffness; fascial stiffness then mechanically constrains deep muscle activation, perpetuating aberrant recruitment.

The mediation analyses demonstrating that 33% of NDI improvement is mediated by fascial stiffness reduction (independent of EMG recruitment ratio change) suggests that fascial properties constitute partially independent pathological contributors not fully captured by neuromuscular metrics. Conventional motor control training (control group) achieves modest EMG normalization (+0.09 recruitment ratio improvement) and functional gains; however, without addressing fascial substrate, functional improvement plateaus. Fascial-specific intervention's ability to improve recruitment ratio by 0.18—double the conventional approach—likely reflects mechanically restored gliding permitting more complete neuromuscular adaptation.

Importantly, the fascial-specific group's recruitment ratio improvement (+0.18) still did not fully normalize toward control values (final 0.60 *vs.* control 0.68, p=0.08). This persistent mild recruitment abnormality despite substantial fascial improvement and clinical recovery suggests that 12-week intervention duration may be insufficient for complete neuromuscular-fascial reorganization, or that chronic smartphone use produces some degree of "permanent" neural adaptations requiring longer intervention duration or additional neuromodulatory approaches (e.g., motor imagery, virtual reality feedback) to fully normalize.

Pain Sensitivity Changes: Nociceptive *vs.* Neuropathic Mechanisms

The baseline PPT elevation in smartphone-exposed participants (24.3±3.1 kPa cervical site *vs.* 38.2±8.4 kPa controls)—indicating 36% reduced pain threshold, or heightened pain sensitivity—reflects likely central and peripheral sensitization mechanisms. Peripherally, chronically densified and mechanically compromised fascia demonstrates altered nociceptor expression and inflammatory mediator production, lowering pain activation threshold.

Centrally, chronic mechanical stress and associated psychosocial factors (disability, activity avoidance) promote spinal cord wind-up and descending pain facilitation.

The fascial-specific intervention achieved 41.9% PPT improvement (10.2 kPa increase, post-intervention 34.5±7.4 kPa), substantially exceeding control group improvement (4.1 kPa, 16.9% improvement). This disproportionate pain improvement attributable to fascial intervention—beyond what would be expected from endurance or functional improvements alone—suggests that mechanical restoration of fascial tissue directly ameliorates peripheral nociceptor sensitization. Fascial mobilization may desensitize mechanoreceptors and nociceptors through: (1) mechanical release of tissue adhesions reducing nociceptor irritation; (2) reduction of pro-inflammatory cytokine production as mechanical stress resolves; (3) restoration of normal fascial fluid dynamics improving nutrient diffusion to nociceptor-rich fascial regions.

Importantly, the persistent 14.9% PPT deficit in fascial-specific group post-intervention (34.5 kPa *vs.* control 38.2 kPa, p=0.002) despite substantial mechanical recovery suggests ongoing central sensitization component not fully resolved by peripheral tissue intervention. Clinical management of smartphone-related pain may benefit from integrating central sensitization-addressing approaches (cognitive-behavioral therapy, pain neuroscience education, graded activity progression) alongside fascial intervention.

Neck Disability Index: Integration of Structural and Functional Improvement

The marked NDI improvements in fascial-specific group (8.4±3.2 point reduction, 45.7% improvement) substantially exceeded control group (3.1±2.8 points, 16.8% improvement), with fascial-specific final NDI (10.0 points) approaching control baseline (2.1 points) and exceeding MCID threshold. This recovery trajectory indicates that restoring fascial biomechanics produces genuine functional improvement, not merely reduced symptom perception.

The mediation analysis demonstrating that 33% of NDI improvement attributable to fascial stiffness reduction indicates a direct mechanistic pathway: improved fascial mechanics → enhanced load distribution → reduced muscular fatigue and pain → improved functional tolerance. The remaining 67% of NDI improvement mediated through other pathways (EMG recruitment normalization, increased endurance, pain sensitivity reduction, psychological factors) reflects the multifactorial nature of functional disability.

Interestingly, post-intervention fascial-specific group NDI (10.0 points) remained elevated compared to control baseline (2.1 points), despite substantial improvements. This persistent mild disability may reflect: (1) psychological factors (fear-avoidance, self-efficacy deficits) not fully addressed by mechanical intervention; (2) residual fascial stiffness (post-intervention 14.3 kPa *vs.* control 11.6 kPa) not completely normalized; (3) incomplete neuromuscular-fascial reorganization within 12-week timeframe; (4) ongoing smartphone use behavior requiring sustained ergonomic modification. Longitudinal follow-up beyond 12 weeks would clarify whether continued functional improvement trajectory or stabilization occurs.

Neurophysiological Implications: Fascial Proprioception and Neuromuscular Control

Contemporary fascial science increasingly recognizes fascia as an integrated sensorimotor organ, not merely passive load-bearing tissue. The deep fascia contains extensive mechanoreceptor networks (Ruffini endings, Lamellar corpuscles, free nerve endings) embedding fascial afferent input into proprioceptive control systems. Chronic fascial stiffness and reduced gliding mechanically alter mechanoreceptor loading patterns, impairing proprioceptive feedback and contributing to neuromuscular control dysfunction independent of direct neuromuscular injury.

The fascial-specific intervention's explicit incorporation of "fascial proprioceptive awareness"—training participants to consciously attend to fascial release and gliding sensations during motor control exercises—represents a neurorehabilitation innovation potentially optimizing proprioceptive re-education. By training motor cortex and cerebellar circuits to integrate restored fascial proprioceptive input, motor control retraining achieves greater neuromuscular-sensorimotor integration compared to conventional approaches operating without fascial proprioceptive emphasis.

This mechanistic insight suggests that future smartphone-related neck disorder management should emphasize "sensorimotor restoration" rather than isolated "postural correction" or "muscle strengthening." Interventions specifically targeting fascial proprioceptive re-education, body awareness, and movement quality may achieve superior outcomes compared to conventional strength-oriented approaches.

Study Limitations and Methodological Considerations

Study Limitations: (1) Relatively short intervention duration (12 weeks); longer follow-up periods would clarify whether improvements sustain and whether additional recovery occurs. (2) No long-term follow-up data (6–12 months post-intervention); determining whether functional improvements maintain after supervised intervention termination requires extended assessment. (3) Single-site study; multicenter replication would strengthen generalizability. (4) Smartphone use quantification relied on self-report; objective usage tracking (accelerometer, app-based monitoring) would provide more precise exposure measurement. (5) Cross-sectional baseline comparison lacks temporal causality evidence; prospective cohort study following incident smartphone users would clarify causality *vs.* association. (6) Fine-wire EMG limited to longus colli; sampling additional deep muscles (rectus capitis anterior, posterior cervical intervertebral muscles) would provide more comprehensive motor control assessment. (7) No control for psychological factors (anxiety, depression, catastrophization); inclusion of standardized psychological measures would clarify psychosocial contributions.

Methodological Strengths: (1) Multimodal outcome assessment (elastography, ultrasound, EMG, functional tests, pain measures, disability scales) providing comprehensive mechanistic evaluation. (2) Standardized, reliably measured outcomes with established validity and responsiveness. (3) Matched control group design with baseline equivalence in demographics and confounders. (4) Intent-to-treat analysis with imputation for missing data. (5) Mediation analysis clarifying mechanistic pathways. (6) Intervention fidelity: standardized protocols with clinician training and supervision ensuring consistent delivery.

Clinical Implications and Future Research Directions

Clinical Practice Implications: The findings establish fascial assessment—via elastography and dynamic ultrasound—as clinically valuable adjunct to conventional musculoskeletal evaluation in smartphone-related neck disorder management. Clinicians encountering patients with persistent neck symptoms despite conventional exercise-based management should consider fascial dysfunction assessment. Fascial-specific intervention, particularly when combined with traditional motor control training, offers mechanistically superior approach compared to exercise-alone management.

Future Research Directions: (1) Comparison of intervention components: dismantling studies isolating fascial mobilization, manual therapy, and motor control retraining contributions to overall efficacy. (2) Dosage optimization studies: determining whether different treatment frequencies or durations produce superior outcomes. (3) Neuroimaging investigation: functional MRI assessment of motor cortex and cerebellar reorganization during fascial intervention and motor control retraining. (4) Psychological intervention integration: testing combined fascial-specific physiotherapy with cognitive-behavioral pain management. (5) Prevention studies: evaluating whether early fascial intervention arrests smartphone-use-related pathology before symptoms develop. (6) Longitudinal mechanistic studies following incident smartphone users to clarify causality and temporal progression of fascial changes. (7) Comparative effectiveness: head-to-head trials comparing fascial-specific physiotherapy to emerging interventions (e.g., dry needling, radiofrequency ablation, neural mobilization).

Conclusion

This investigation provides robust multimodal evidence that chronic smartphone use induces quantifiable cervico-thoracic fascial densification and compromised myofascial load transmission representing novel, mechanistically distinct pathology absent from existing literature. Fascial stiffness elevation (59% above controls) and dynamic glide impairment (51% reduction) mechanistically contribute to observed neuromuscular recruitment dysfunction and functional disability. Fascial-specific physiotherapy combining mechanical mobilization with integrated motor control retraining targeting deep cervical flexor–fascial coupling achieves substantially superior outcomes compared to conventional cervical stabilization exercise, with improvements encompassing genuine fascial biomechanical recovery, normalized load transmission, and clinically meaningful functional restoration exceeding MCID thresholds.

The research fills critical investigative gaps by: (1) introducing objective fascial biomechanical assessment to smartphone-related pathology literature; (2) mechanistically linking fascial dysfunction to neuromuscular and functional impairment; (3) demonstrating that tissue-specific intervention outperforms general motor control training; (4) establishing fascial substrate pathology as essential treatment target in smartphone-related neck disorder management.

These findings fundamentally reframe understanding of smartphone-related cervical pathomechanics from isolated postural-muscular dysfunction to integrated fascial-neuromuscular system pathology. Clinical management optimizing outcomes requires addressing fascial substrate directly, not merely correcting posture or strengthening muscles across mechanically compromised tissue.

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