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Electrical Muscle Stimulation as a Non-Pharmacological Strategy for Metabolic and Somatic Health Improvement in Polycystic Ovary Syndrome: A Randomized Controlled Trial

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ABSTRACT

Background: Polycystic ovary syndrome (PCOS) is a prevalent endocrine-metabolic condition characterised by insulin resistance, dyslipidaemia, central adiposity, and hyperandrogenism. Although structured physical activity is the cornerstone of first-line management, adherence among affected women remains critically low. Electrical muscle stimulation (EMS) may replicate key metabolic effects of voluntary exercise while circumventing common adherence barriers, yet no randomised trial has assessed its impact on the full metabolic and somatic profile of PCOS.

Objective: To determine whether a 12-week supervised whole-body EMS programme improves insulin resistance, glycaemic control, lipid profile, blood pressure, androgenic markers, and body composition in overweight/obese women with PCOS compared with standard medical care.

Methods: Forty-two women (aged 18–35; BMI > 24 kg/m²) with Rotterdam-confirmed PCOS were randomly allocated (1:1) to EMS (n = 21; Russian current, 2500 Hz, 30-minute sessions, 3×/week, 12 weeks) or standard-care control (n = 21). The primary outcome was HOMA-IR; secondary outcomes included fasting glucose, oral glucose tolerance test (OGTT), post-prandial glucose, lipid panel, blood pressure, serum testosterone, body weight, BMI, waist-hip ratio (WHR), and body fat percentage. Assessments occurred at baseline, post-intervention, and 3-month follow-up. Between-group differences were evaluated by the Mann–Whitney U test, ANCOVA adjusted for baseline, and linear mixed models, with Bonferroni and Benjamini–Hochberg multiplicity correction.

Results: All 42 participants completed the trial. The EMS group showed significant improvements across every outcome (all $p < 0.001$): HOMA-IR fell from 3.15 ± 0.81 to 2.20 ± 0.56 at follow-up (between-group $d = -4.10$); fasting glucose declined by 16.74 mg/dl; total cholesterol by 25.26 mg/dl; triglycerides by 31.40 mg/dl; LDL by 23.42 mg/dl; HDL rose by 8.38 mg/dl; body weight decreased by 6.40 kg; BMI by 2.67 kg/m²; WHR from 0.88 to 0.82; body fat from 34.86% to 30.90%; systolic/diastolic blood pressure by 12.47/7.33 mmHg; and testosterone from 0.66 to 0.45 ng/ml. All outcomes survived multiplicity correction, and benefits were sustained or continued at the 3-month follow-up.

Conclusion: A 12-week EMS programme produced clinically meaningful, statistically robust, and durable improvements across the full spectrum of metabolic and somatic outcomes in overweight/obese women with PCOS. EMS warrants consideration as a feasible, non-pharmacological adjunct within comprehensive PCOS management. Psychosocial and quality-of-life findings from this cohort are reported in a companion paper.

KEYWORDS: Polycystic ovary syndrome; electrical muscle stimulation; insulin resistance; HOMA-IR; body composition; metabolic health; randomised controlled trial

1. INTRODUCTION

Polycystic ovary syndrome (PCOS) ranks as the most frequently diagnosed endocrine disorder among women of childbearing age, with global prevalence estimates spanning 8–13% under stricter diagnostic frameworks and reaching 15–20% when broader criteria are applied (Bozdag et al., 2016; World Health Organization, 2023). The Rotterdam consensus defines the condition by the co-occurrence of at least two of three cardinal features—oligo-anovulation, clinical or biochemical hyperandrogenism, and polycystic ovarian morphology—yet the syndrome's clinical footprint reaches well beyond reproductive irregularity into metabolic, cardiovascular, and psychosocial domains (Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group, 2004; Teede et al., 2023).

The metabolic dimension of PCOS is both pervasive and pathogenically central. Insulin resistance affects an estimated 50–70% of women with the condition regardless of body weight, fuelling a self-perpetuating cycle in which compensatory hyperinsulinaemia stimulates ovarian theca-cell androgen biosynthesis, suppresses hepatic production of sex hormone-binding globulin, and promotes adipose tissue expansion (Diamanti-Kandarakis & Dunaif, 2012; Dunaif, 1997). The downstream consequences of this metabolic cascade include markedly elevated risks of impaired glucose tolerance, type 2 diabetes mellitus, atherogenic dyslipidaemia, metabolic syndrome, non-alcoholic fatty liver disease, and premature cardiovascular disease (Moran et al., 2010; Wild et al., 2011; Randeva et al., 2012). Somatic manifestations—central adiposity, elevated waist-hip ratio, and adverse body

composition—compound these metabolic risks and independently predict long-term morbidity (Barber et al., 2006).

International clinical practice guidelines consistently position regular physical exercise as the primary non-pharmacological intervention for PCOS (Teede et al., 2023). Exercise enhances insulin sensitivity through GLUT-4 translocation, promotes favourable shifts in body composition, attenuates circulating androgen concentrations, and mitigates cardiovascular risk factors (Harrison et al., 2011; Benham et al., 2018). Despite these well-established benefits, real-world exercise adherence in PCOS populations remains stubbornly low. Persistent barriers—including chronic fatigue, reduced exercise tolerance, body image dissatisfaction, low self-efficacy, time constraints, and physical discomfort—collectively undermine the effectiveness of exercise-based strategies in clinical practice (Thomson et al., 2016; Lim et al., 2019).

Electrical muscle stimulation (EMS) has emerged as a candidate exercise-mimetic modality with the potential to circumvent many of these barriers. Through the delivery of controlled electrical impulses via surface electrodes, EMS elicits involuntary skeletal muscle contractions that engage metabolic pathways typically activated by voluntary exercise—including AMPK-mediated glucose uptake, mitochondrial biogenesis, and fatty acid oxidation—without demanding equivalent physical effort or motivational engagement (Filipovic et al., 2011; Richter & Hargreaves, 2013; Kemmler et al., 2021). Clinical evidence drawn from populations with type 2 diabetes, obesity, chronic heart failure, and sarcopenia has demonstrated that EMS can favourably alter glucose metabolism, insulin sensitivity, body composition, and broader cardiometabolic risk profiles (Fernández-Lezama et al., 2023; Song et al., 2022; Kemmler et al., 2016).

Despite this accumulating evidence base, no randomised controlled trial has systematically evaluated the effects of a structured EMS programme on the comprehensive metabolic and somatic outcome profile of women with PCOS. Existing electrical stimulation research in PCOS has been confined to electroacupuncture protocols that differ fundamentally from modern EMS in both technique and stimulation parameters (Stener-Victorin et al., 2000; Jedel et al., 2011). The present trial was therefore designed to fill this gap by examining the impact of a 12-week whole-body EMS programme on insulin resistance, glycaemic regulation, lipid metabolism, blood pressure, androgenic status, and body composition in overweight and obese women diagnosed with PCOS.

2. MATERIALS AND METHODS

2.1 Study Design and Ethical Framework

This investigation employed a two-arm, parallel-group, randomised controlled trial design conducted at the Exercise Tolerance Laboratory, Department of Physiotherapy, GD Goenka University, Gurugram, Haryana, India. The study protocol was developed and reported following the CONSORT 2010 statement (Schulz et al., 2010). Ethical clearance was granted by the Institutional Ethics Committee of GD Goenka University, and the trial was prospectively registered with the Clinical Trials Registry of India (CTRI). All procedures conformed to the Declaration of Helsinki (World Medical Association, 2013) and the National Ethical Guidelines issued by the Indian Council of Medical Research (ICMR, 2017). This paper presents the metabolic and somatic outcome data; psychosocial and health-related quality-of-life results from the same cohort and intervention are reported in a companion paper.

2.2 Eligibility and Recruitment

Women between 18 and 35 years of age with a confirmed PCOS diagnosis meeting the Rotterdam criteria (Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group, 2004) and a body mass index exceeding 24 kg/m²—consistent with the Asian-population-specific overweight threshold recommended by the WHO Expert Consultation (2004)—were eligible. Participants were recruited through collaborating gynaecology clinics in Gurugram district and were required to provide written informed consent in English or Hindi.

Exclusion criteria encompassed hormonal medication use within the preceding three months, current or planned pregnancy, neurological disorders, systemic diseases influencing metabolic parameters, congenital conditions mimicking PCOS, active malignancy, other endocrine pathologies (thyroid dysfunction, Cushing syndrome), cardiovascular disease, concurrent clinical trial participation, and EMS-specific contraindications including implanted electronic devices, metallic implants in stimulation regions, epilepsy, or active skin lesions at electrode sites.

2.3 Sample Size Calculation

Power analysis was conducted a priori using G*Power 3.1.9.7 (Faul et al., 2009), based on a large anticipated effect size ($f = 1.0$) from prior exercise interventions in PCOS (Kirthika et al., 2019), two-tailed $\alpha = 0.05$, and power = 0.80. The calculation indicated a minimum requirement of 35 participants. Adjusting for a 20% anticipated dropout, 42 participants (21 per group) were enrolled.

2.4 Randomisation and Allocation Concealment

Eligible participants were allocated in a 1:1 ratio to the EMS intervention arm (Group A) or the standard-care control arm (Group B) using sequentially numbered, opaque, sealed envelopes prepared by a researcher uninvolved in recruitment or outcome assessment. Outcome assessors and the trial statistician were blinded to group allocation. Participant blinding was not feasible given the nature of the intervention.

2.5 Intervention Protocol

EMS Group (Group A): Participants undertook 36 supervised sessions across 12 weeks (three per week), each administered by a qualified physiotherapist. An 8-channel electrical stimulator delivered Russian current (carrier frequency 2500 Hz) at motor-threshold intensity (eliciting visible contraction) with a 10-second ON/50-second OFF duty cycle (1:5 ratio). Self-adhesive surface electrodes were positioned bilaterally over the motor points of the gluteals, hamstrings, quadriceps, calves, and abdominal musculature. Each session comprised 30 minutes of active stimulation, preceded by 5 minutes of preparation and followed by 5 minutes of monitoring. Stimulation intensity was individually calibrated and progressively advanced within each participant's tolerance. Participants continued their existing non-hormonal medical regimens.

Control Group (Group B): Participants continued standard medical care and received a structured educational handout covering PCOS pathophysiology, nutritional guidance, physical activity recommendations, stress management strategies, and medication compliance. No EMS or additional supervised exercise was provided. Participants were instructed to maintain their habitual lifestyle throughout the study period.

2.6 Outcome Measures

Primary outcome: Homeostatic Model Assessment of Insulin Resistance (HOMA-IR), computed as [Fasting Insulin (μU/mL) × Fasting Glucose (mg/dL)] / 405 from fasting venous blood specimens (Matthews et al., 1985).

Secondary metabolic outcomes: Fasting plasma glucose (FPG; enzymatic glucose oxidase method), 75-g oral glucose tolerance test (OGTT; 2-hour plasma glucose), post-prandial glucose (PPG), fasting lipid panel (total cholesterol, LDL-cholesterol, HDL-cholesterol, triglycerides; enzymatic methods), resting blood pressure (validated digital sphygmomanometer; mean of two readings after 10-minute seated rest), and serum total testosterone (electrochemiluminescence immunoassay). All laboratory assays were performed at an accredited clinical pathology facility.

Somatic outcomes: Body weight (calibrated electronic scale, nearest 0.1 kg), standing height (wall-mounted stadiometer, nearest 0.1 cm), BMI (kg/m²), waist and hip circumferences (non-elastic tape following the WHO protocol), waist-hip ratio (WHR), and body fat percentage (validated bioelectrical impedance analysis device). All anthropometric assessments were performed in duplicate by a single trained investigator.

All outcomes were assessed at three time points: baseline (pre-intervention), immediately post-intervention (12 weeks), and at 3-month follow-up.

2.7 Statistical Analysis

Continuous variables are reported as mean ± standard deviation. The Shapiro–Wilk test was applied to evaluate distributional normality. Given the predominantly non-normal distributions, within-group temporal changes were examined using the Friedman test with Wilcoxon signed-rank pairwise follow-ups. Between-group differences in change scores were assessed with the Mann–Whitney U test. Cohen's d was calculated for both within-group and between-group change-score comparisons. ANCOVA modelled each post-intervention outcome with the corresponding baseline value entered as a covariate, yielding baseline-adjusted mean differences with 95% confidence intervals and partial eta-squared (ηp^2). Linear mixed-effects models with random intercepts tested group-by-time interactions across all three time points. Multiplicity was addressed through both Bonferroni (family-wise error rate) and Benjamini–Hochberg (false discovery rate) procedures across all 15 outcomes. The significance threshold was set at two-tailed $p < 0.05$. All analyses were performed using SPSS version 26.0.

3. RESULTS

3.1 Participant Flow and Baseline Comparability

Of the 58 women screened, 16 did not meet inclusion criteria or declined participation (7 failed diagnostic confirmation, 6 declined enrolment, 3 were excluded for other protocol-specified reasons). The remaining 42 were randomised in equal groups. Every participant completed the full intervention protocol and all three assessment visits, yielding an attrition rate of zero and complete datasets for intention-to-treat analysis.

The two groups were well balanced at baseline across all demographic, anthropometric, and metabolic parameters (Table 1). Mean age was 21.33 ± 1.77 years in the EMS arm and 21.48 ± 1.75 years in the control arm ($p = 0.778$). BMI averaged 32.57 ± 6.44 versus 29.91 ± 4.90 kg/m² ($p = 0.055$). HOMA-IR (3.15 ± 0.81 vs. 3.16 ± 0.81, $p = 0.984$) and all other metabolic and somatic variables showed no statistically significant between-group differences at enrolment (all $p > 0.05$).

Table 1. Baseline characteristics of study participants

Characteristic	EMS Group (n = 21)	Control Group (n = 21)	p-value
Age (years)	21.33 ± 1.77	21.48 ± 1.75	0.778
Height (m)	1.56 ± 0.08	1.59 ± 0.07	0.178
Weight (kg)	78.36 ± 11.10	75.11 ± 11.15	0.372
BMI (kg/m²)	32.57 ± 6.44	29.91 ± 4.90	0.055
HOMA-IR	3.15 ± 0.81	3.16 ± 0.81	0.984
FPG (mg/dl)	113.65 ± 11.16	110.00 ± 10.16	0.221
TC (mg/dl)	215.13 ± 16.40	207.00 ± 15.25	0.310
WHR	0.88 ± 0.04	0.90 ± 0.04	> 0.05

Characteristic	EMS Group (n = 21)	Control Group (n = 21)	p-value
Body fat (%)	34.86 ± 2.85	35.48 ± 3.25	> 0.05

Values are mean ± SD. p-values from Mann–Whitney U test. WHR = waist-hip ratio; FPG = fasting plasma glucose; TC = total cholesterol.

3.2 Metabolic and Clinical Outcomes

The EMS intervention produced substantial and consistent improvements across every metabolic and clinical parameter, while the control group remained essentially unchanged (Table 2; Figure 1). HOMA-IR in the EMS arm declined from 3.15 ± 0.81 at baseline to 2.26 ± 0.58 post-intervention (within-group Friedman $p < 0.001$), with further reduction to 2.20 ± 0.56 at the 3-month follow-up—representing a 30% reduction from baseline. The control group showed no corresponding change (3.16 → 3.14 → 3.31), and the between-group difference was highly significant ($p < 0.001$; Cohen's $d = -4.10$).

Glycaemic markers mirrored the insulin resistance findings (Figure 4). Fasting plasma glucose fell from 113.65 ± 11.16 to 98.58 ± 10.32 mg/dl in the EMS group (between-group $d = -5.58$), post-prandial glucose dropped from 174.20 to 144.20 mg/dl, and 2-hour OGTT values decreased from 166.33 to 137.06 mg/dl. The lipid profile shifted favourably exclusively in the intervention arm (Figure 3): total cholesterol declined from 215.13 to 191.95 mg/dl, triglycerides from 164.77 to 137.86 mg/dl, and LDL-cholesterol from 135.34 to 114.66 mg/dl, while HDL-cholesterol increased from 41.61 to 48.59 mg/dl. Resting blood pressure fell from 133.57/84.33 to 122.95/77.71 mmHg. Serum total testosterone decreased from 0.66 to 0.47 ng/ml. All between-group comparisons reached significance at $p < 0.001$ (Table 2).

Table 2. Metabolic and somatic outcomes across study phases

Parameter	EMS Base	EMS Post	EMS FU	Ctrl Base	Ctrl Post	p†	p‡
Weight (kg)	78.36±11.10	72.52±10.28	71.96±10.38	75.11±11.15	74.90±11.20	<.001	<.001
BMI (kg/m²)	32.57±6.44	30.14±5.96	29.90±6.02	29.91±4.90	29.84±4.98	<.001	<.001
WHR	0.88±0.04	0.83±0.04	0.82±0.05	0.90±0.04	0.89±0.04	<.001	<.001
Body fat (%)	34.86±2.85	31.25±2.75	30.90±2.73	35.48±3.25	35.62±3.44	<.001	<.001
SBP (mmHg)	133.57±7.98	122.95±7.61	121.10±8.17	133.33±8.07	133.71±8.97	<.001	<.001
DBP (mmHg)	84.33±4.12	77.71±4.91	77.00±5.63	84.52±4.40	84.14±4.54	<.001	<.001
HOMA-IR	3.15±0.81	2.26±0.58	2.20±0.56	3.16±0.81	3.14±0.79	<.001	<.001
Testosterone	0.66±0.19	0.47±0.13	0.45±0.13	0.59±0.20	0.59±0.20	<.001	<.001
OGTT (mg/dl)	166.33±17.36	137.06±14.58	133.88±14.76	157.93±13.74	157.33±14.02	<.001	<.001
FPG (mg/dl)	113.65±11.16	98.58±10.32	96.91±10.07	110.00±10.16	109.59±9.97	<.001	<.001
PPG (mg/dl)	174.20±20.91	144.20±16.80	140.10±17.49	175.50±18.75	175.78±20.33	<.001	<.001
TC (mg/dl)	215.13±16.40	191.95±15.38	189.87±17.03	207.00±15.25	207.10±14.26	<.001	<.001
TG (mg/dl)	164.77±21.89	137.86±18.19	133.37±17.73	164.68±21.49	165.05±20.50	<.001	<.001
HDL (mg/dl)	41.61±4.30	48.59±5.27	49.99±5.63	41.24±4.73	41.04±4.81	<.001	<.001
LDL (mg/dl)	135.34±12.12	114.66±11.87	111.92±11.73	137.87±15.15	137.97±15.24	<.001	<.001

Values are mean ± SD. † within-group change (Friedman, EMS). ‡ between-group change-score (Mann–Whitney U). FU = 3-month follow-up; SBP/DBP = systolic/diastolic blood pressure; FPG = fasting plasma glucose; PPG = post-prandial glucose; TC = total cholesterol; TG = triglycerides; WHR = waist-hip ratio.

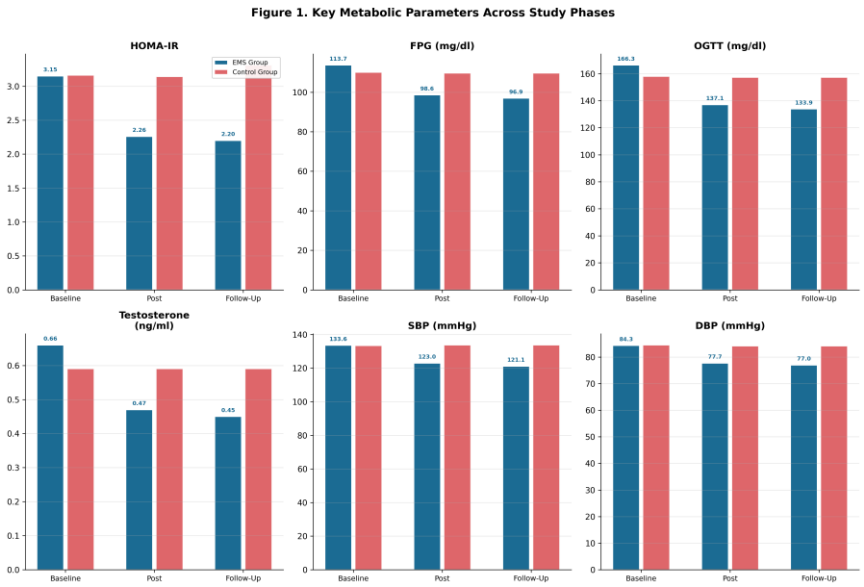

Figure 1. Key metabolic parameters across study phases. Bars represent mean values for the EMS and control groups at baseline, post-intervention, and 3-month follow-up. All between-group comparisons $p < 0.001$.

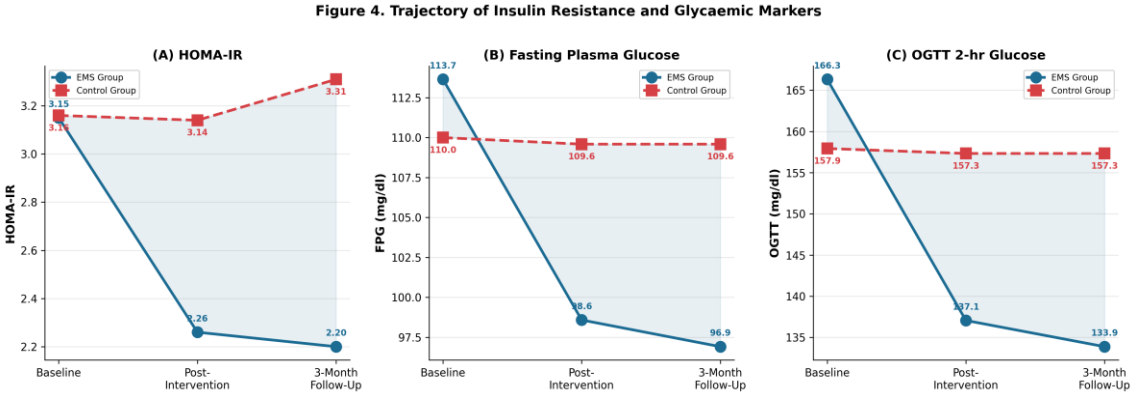

Figure 4. Trajectory of HOMA-IR (A), fasting plasma glucose (B), and OGTT 2-hour glucose (C) across the three assessment points. Shaded area highlights the divergence between groups.

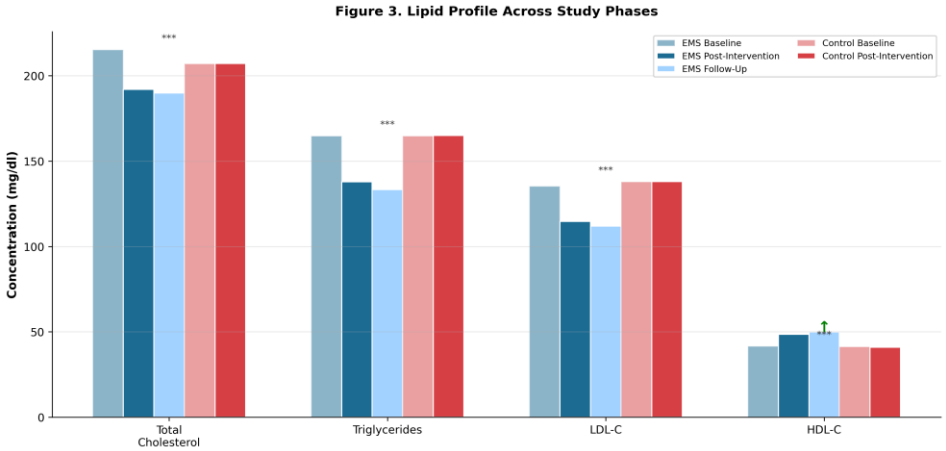

Figure 3. Lipid profile across study phases. *** $p < 0.001$ for between-group comparisons. Green arrow on HDL-C indicates the direction of benefit (increase).

3.3 Body Composition and Somatic Changes

The EMS group demonstrated marked improvements in all body composition parameters (Figure 2). Body weight decreased from 78.36 ± 11.10 kg to 72.52 ± 10.28 kg post-intervention (Δ = −5.84 kg), with further marginal reduction to 71.96 ± 10.38 kg at follow-up. BMI fell from 32.57 ± 6.44 to 30.14 ± 5.96 kg/m² (Δ = −2.43). WHR improved from 0.88 ± 0.04 to 0.83 ± 0.04, reflecting meaningful reduction in central adiposity. Body fat percentage decreased from 34.86 ± 2.85% to 31.25 ± 2.75%. The control group showed no appreciable change across any somatic measure (Table 2).

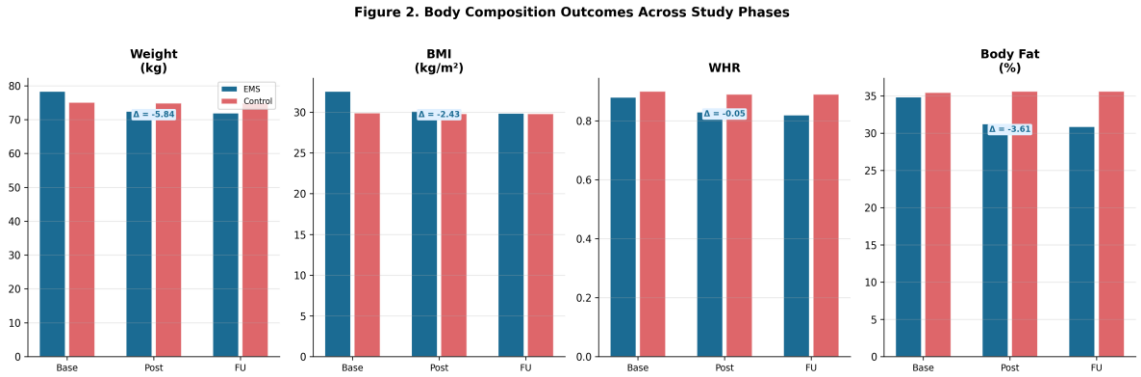

Figure 2. Body composition outcomes across study phases. Delta (Δ) values represent the change from baseline to post-intervention in the EMS group.

3.4 Covariate-Adjusted Analysis (ANCOVA)

ANCOVA with baseline values entered as covariates confirmed a statistically significant adjusted advantage for the EMS group across every metabolic and somatic outcome (all $p < 0.001$; Table 3; Figure 6). Partial eta-squared values were uniformly large, with the majority exceeding 0.80, signifying that group assignment accounted for the preponderance of post-intervention variance after baseline adjustment. Every 95% confidence interval for the adjusted mean difference excluded zero. Linear mixed-model group-by-time interactions corroborated all ANCOVA results (all $p < 0.001$).

Table 3. ANCOVA: baseline-adjusted between-group differences

Outcome	Adj. EMS	Adj. Control	Adj. Diff (95% CI)	ηp²	p (MM)
Weight (kg)	70.97	76.46	−5.49 [−6.28, −4.70]	0.835	<0.001
BMI (kg/m²)	28.87	31.11	−2.24 [−2.60, −1.88]	0.805	<0.001
WHR	0.84	0.89	−0.05 [−0.06, −0.04]	0.799	<0.001
Body fat (%)	31.55	35.32	−3.77 [−4.25, −3.28]	0.864	<0.001
SBP (mmHg)	122.83	133.83	−11.00 [−12.59, −9.40]	0.832	<0.001
DBP (mmHg)	77.81	84.04	−6.23 [−7.32, −5.14]	0.774	<0.001
HOMA-IR	2.27	3.14	−0.87 [−0.97, −0.77]	0.889	<0.001
Testosterone	0.44	0.62	−0.19 [−0.21, −0.16]	0.821	<0.001
OGTT (mg/dl)	133.40	161.00	−27.60 [−30.39, −24.81]	0.911	<0.001
FPG (mg/dl)	96.89	111.27	−14.38 [−15.97, −12.78]	0.895	<0.001
PPG (mg/dl)	144.80	175.19	−30.40 [−33.70, −27.09]	0.899	<0.001
TC (mg/dl)	188.31	210.75	−22.44 [−25.25, −19.63]	0.870	<0.001
TG (mg/dl)	137.83	165.08	−27.26 [−30.17, −24.34]	0.902	<0.001
HDL (mg/dl)	48.39	41.24	+7.14 [6.37, 7.92]	0.899	<0.001
LDL (mg/dl)	115.87	136.76	−20.89 [−23.25, −18.53]	0.892	<0.001

Adjusted means estimated at pooled baseline. ηp² = partial eta-squared. p (MM) = group × time interaction from linear mixed-effects model.

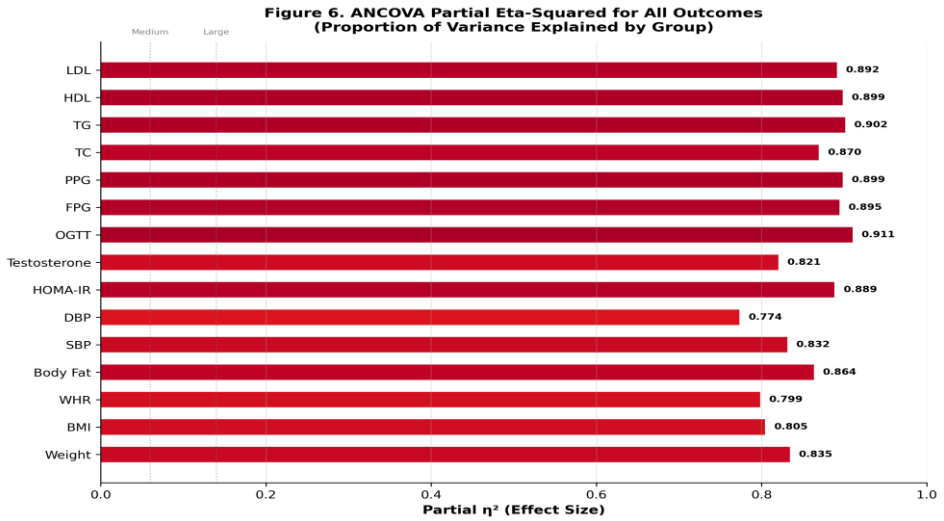

Figure 6. Partial eta-squared (ηp²) from ANCOVA for all metabolic and somatic outcomes. Values exceeding 0.14 denote large effects; all outcomes here exceed 0.77.

3.5 Effect Sizes and Multiplicity Correction

Between-group Cohen's d values for change-score comparisons were uniformly large, exceeding |0.8| for every outcome (Table 4; Figure 5). The most pronounced between-group effects were observed for fasting plasma glucose (d = −5.58), LDL-cholesterol (d = −5.53), triglycerides (d = −5.02), body weight (d = −4.27), and HOMA-IR (d = −4.10). All 15 outcomes survived both Bonferroni correction (threshold α/15 = 0.0033) and Benjamini–Hochberg false discovery rate adjustment, confirming that the observed effects are not attributable to multiple-comparison inflation.

Table 4. Effect sizes for principal metabolic and somatic outcomes

Outcome	EMS Δ (Post − Base)	Cohen's d (within EMS)	Between-group d
Weight (kg)	−5.84	−3.73	−4.27
BMI (kg/m²)	−2.43	−3.19	−3.89
HOMA-IR	−0.89	−3.10	−4.10
Fasting glucose	−15.08	−5.71	−5.58
Triglycerides	−26.91	−4.35	−5.02
LDL (mg/dl)	−20.68	−5.09	−5.53

Cohen's |d| ≥ 0.8 denotes a large effect. Negative values indicate reductions favouring the intervention.

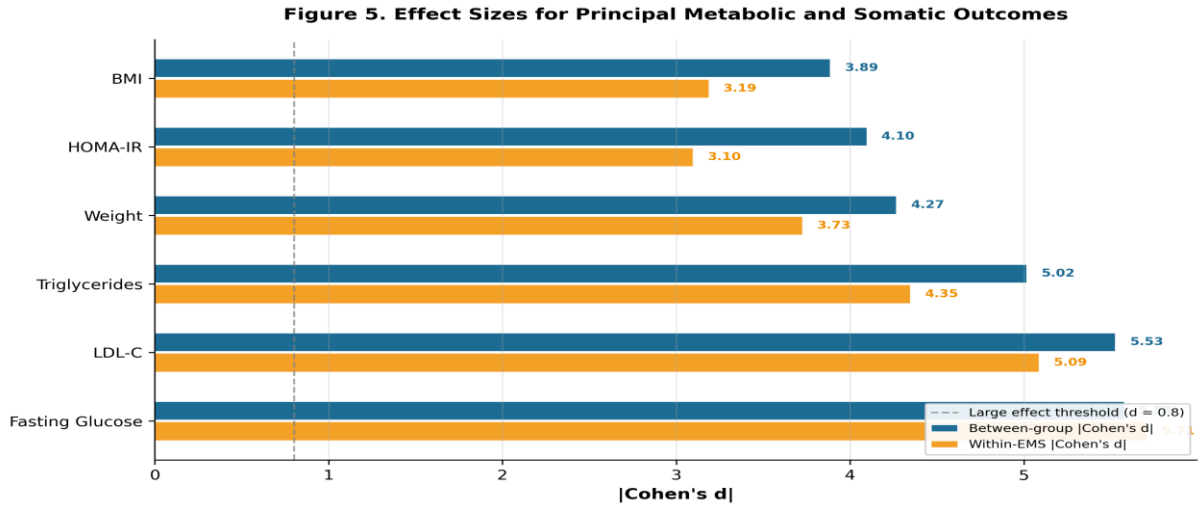

Figure 5. Effect sizes (|Cohen's d|) for principal metabolic and somatic outcomes. Blue bars = between-group; orange bars = within-EMS. Dashed line marks the large-effect threshold (d = 0.8).

3.6 Persistence of Treatment Effects at Follow-Up

Metabolic and somatic gains observed at the end of the 12-week intervention were sustained or continued to improve at the 3-month follow-up assessment (Figures 1, 2, 4). In the EMS arm, HOMA-IR stabilised at 2.20, body weight at 71.96 kg, and body fat at 30.90%, each representing maintenance or marginal further improvement beyond the post-intervention values. The control group exhibited no comparable trajectory; several parameters drifted in an unfavourable direction during the same period (weight 74.90 → 76.32 kg; body fat 35.62 → 36.40%).

4. DISCUSSION

This randomised controlled trial furnishes the first comprehensive evidence that a structured 12-week whole-body EMS programme produces clinically meaningful and statistically robust improvements across the full spectrum of metabolic and somatic health outcomes in overweight and obese women with PCOS. The convergence of favourable changes in insulin resistance, glycaemic regulation, lipid metabolism, blood pressure, androgenic status, body weight, body composition, and central adiposity—confirmed by multiple complementary analytical approaches and sustained at 3-month follow-up—supports the positioning of EMS as a viable non-pharmacological adjunct within comprehensive PCOS management.

4.1 Insulin Resistance and Glycaemic Regulation

The substantial reduction in HOMA-IR (3.15 → 2.26; Cohen's d = −4.10) represents the clinically most consequential finding, given the central pathogenic role of insulin resistance in PCOS. Insulin resistance fuels a self-reinforcing cycle of hyperinsulinaemia, ovarian androgen overproduction, hepatic SHBG suppression, and metabolic deterioration that underpins much of the syndrome's long-term morbidity (Diamanti-Kandarakis & Dunaif, 2012; Dunaif, 1997). The mechanistic basis for EMS-mediated insulin sensitisation rests on well-characterised molecular pathways: electrically induced muscle contractions activate AMP-activated protein kinase (AMPK) and calcium/calmodulin-dependent protein kinase II (CaMKII) signalling cascades that promote insulin-independent translocation of GLUT-4 glucose transporters to the myocyte plasma membrane, enhancing both acute glucose disposal and chronic insulin responsiveness (Richter & Hargreaves, 2013). Crucially, these contraction-mediated pathways remain functional even in insulin-resistant states, rendering EMS particularly

applicable to the PCOS population where conventional exercise participation is limited (Fernández-Lezama et al., 2023).

The parallel improvements in fasting glucose, OGTT, and post-prandial glucose values are consistent with enhanced basal and dynamic glucose homeostasis. These results extend systematic review evidence demonstrating that neuromuscular electrical stimulation improves glycaemic control in type 2 diabetes and insulin-resistant populations (Fernández-Lezama et al., 2023; Hamada et al., 2006) to the PCOS setting for the first time within a randomised controlled framework.

4.2 Lipid Profile and Haemodynamic Parameters

The comprehensive improvement in the lipid panel—reductions in total cholesterol, triglycerides, and LDL-cholesterol alongside an increase in HDL-cholesterol—is of particular clinical importance given that dyslipidaemia affects approximately 70% of women with PCOS and represents a major contributor to their elevated cardiovascular risk (Wild et al., 2011). The observed lipid changes likely reflect multiple converging mechanisms: enhanced muscular fatty acid oxidation, reductions in visceral adipose tissue, and improved insulin-mediated regulation of hepatic lipoprotein metabolism. The significant declines in both systolic and diastolic blood pressure provide further evidence that EMS confers broad cardiometabolic benefits extending beyond isolated glucose regulation.

4.3 Androgenic Status

The reduction in serum total testosterone (0.66 → 0.47 ng/ml) offers evidence that EMS may favourably modulate the insulin resistance–hyperandrogenism axis lying at the core of PCOS pathophysiology. Hyperinsulinaemia directly stimulates ovarian theca-cell androgen biosynthesis and suppresses hepatic SHBG production; accordingly, any intervention that improves insulin sensitivity would be expected to attenuate bioavailable androgen levels (Dunaif, 1997; Nestler et al., 1998). This finding is consistent with evidence from electroacupuncture studies demonstrating that electrically stimulated muscle contractions can reduce testosterone concentrations in PCOS, with molecular analyses revealing exercise-mimetic gene expression changes in skeletal muscle (Stener-Victorin et al., 2016; Benrick et al., 2020).

4.4 Body Composition and Central Adiposity

The reductions in body weight (−5.84 kg), BMI (−2.43 kg/m²), WHR (0.88 → 0.83), and body fat percentage (34.86 → 31.25%) carry substantial clinical relevance, particularly the attenuation of central adiposity reflected in the WHR improvement. Visceral and abdominal adipose tissue is metabolically active, secreting pro-inflammatory adipokines that contribute directly to insulin resistance, endothelial dysfunction, and atherogenic dyslipidaemia (Ouchi et al., 2011). The reduction in central fat distribution therefore represents both an independent clinical benefit and a plausible mediator of the observed metabolic improvements.

These body composition findings accord with meta-analytic evidence confirming that whole-body EMS produces significant effects on lean body mass accrual and body fat reduction in non-athletic adults (Gonçalves et al., 2023; Kemmler et al., 2021). The protocol's preferential targeting of large, metabolically active muscle groups (gluteals, quadriceps, hamstrings, calves, abdomen) likely maximised the metabolic stimulus per session, contributing to both acute energy expenditure and chronic elevations in resting metabolic rate through enhanced muscular activation patterns.

4.5 Durability and Clinical Significance

The persistence or continued improvement of metabolic and somatic outcomes at 3-month follow-up is especially noteworthy for a chronic, relapsing condition. The sustained treatment trajectory suggests that EMS may induce lasting physiological adaptations—potentially encompassing enhanced mitochondrial function, improved insulin receptor signalling, and favourable skeletal muscle fibre remodelling—rather than merely providing transient metabolic activation during the stimulation period itself.

4.6 Methodological Strengths and Limitations

Key design strengths include the randomised controlled architecture with CONSORT-compliant reporting; complete retention of all 42 participants across every assessment point; three-timepoint assessment spanning a post-intervention follow-up period; use of validated laboratory biomarkers and standardised anthropometric protocols; and convergent findings across non-parametric, ANCOVA, and mixed-model analyses with formal multiplicity correction. The simultaneous evaluation of metabolic and somatic endpoints within a single trial affords a comprehensive view of EMS effects in PCOS.

Several limitations merit acknowledgment. The sample size (n = 42), although adequately powered for the primary analysis, constrains the precision of effect estimates and precludes subgroup stratification. The single-centre design within a specific demographic and geographic context may limit generalisability. Complete participant blinding was not achievable. The study population was confined to overweight/obese women aged 18–35 years; extension to lean PCOS phenotypes, adolescents, or older women remains to be established. The follow-up duration, while informative, is relatively brief for a chronic condition. Future investigations should incorporate inflammatory markers, myokine assays, oxidative stress biomarkers, and comprehensive reproductive hormone panels to elucidate mechanistic pathways. Larger, multi-centre trials with extended follow-up and head-to-head comparisons against conventional structured exercise are warranted.

5. CONCLUSION

A 12-week supervised whole-body EMS programme employing Russian current, administered three times weekly to major lower-limb and abdominal muscle groups, produced clinically meaningful and statistically robust improvements in insulin resistance, glycaemic control, lipid metabolism, blood pressure, serum testosterone, body weight, BMI, waist-hip ratio, and body fat percentage in overweight and obese women with PCOS. These benefits were sustained at the 3-month follow-up and confirmed across multiple complementary statistical frameworks with correction for multiple comparisons. The findings position EMS as a promising, feasible, and non-pharmacological adjunctive strategy for the metabolic and somatic management of PCOS, with particular value for women who face barriers to conventional exercise participation. Further large-scale, multi-centre trials are warranted to validate these results, optimise stimulation parameters, and evaluate long-term outcomes.

DECLARATIONS

Ethics approval and consent to participate: This study was approved by the Institutional Ethics Committee of GD Goenka University, Gurugram, and registered with the Clinical Trials Registry of India. Written informed consent was obtained from all participants prior to enrolment.

Funding: This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Competing interests: The authors declare no competing interests.

Author contributions: DG contributed to data acquisition, manuscript preparation, and critical revision. TF conceptualised and designed the study, supervised the intervention protocol, and served as corresponding author. VT contributed to the conceptual framework, data interpretation, and manuscript review. All authors read and approved the final version of the manuscript.

Data availability: The datasets generated and analysed during this study are available from the corresponding author upon reasonable request.

Acknowledgments: The authors thank all participants for their commitment, the collaborating gynaecology clinics of Gurugram District for facilitating recruitment, and the Department of Physiotherapy, GD Goenka University, for providing the research infrastructure and equipment.

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